Interaction of genes and environment conditioning inheritance of stripe rust resistance of wheat
by Robert Thomas Lewellen

A thesis submitted to the Graduate Faculty in partial fulfillment of the requirements for the degree of DOCTOR OF PHILOSOPHY in Genetics
Montana State University
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Abstract:
Seedling infection types of four wheat varieties and their F1, F2, F3, testcross, and testcross-F2 distributions were infected with a single unidentified race of Puccinia striiformis and used to study the inheritance of stripe rust resistance genes, their interaction with each other, and with temperature. Two rigidly controlled environment chambers were used, temperature being the important variable. Diurnal temperature profiles with gradual inclines and declines were used which simulated spring and fall conditions, 2 C night/18 C day, and early to mid-summer conditions, 15 C night/24 C day.

Wheat varieties P. I. 178383 (ovo) and Chinese 166 (ovo) had dominant genes for resistance which conditioned a ovo infection type. The heterozygote of these factors conditioned a oo to 0 infection type. The double heterozygote conditioned a ovo infection type. In addition, these varieties had minor genes which conditioned 0, 1, 2, and 3 infection types in certain additive combinations.

Rego (0 at 2/18 and 3 at 15/24) had a pair of dominant complementary genes which conditioned a 3 infection type at both profiles. However, at 2/18 Rego had temperature sensitive minor genes which conditioned a O infection type in combination with the two complementary genes.

Lemhi (4) was considered to be completely void of resistance genes.

Distributions of segregating progenies showed a significant interaction with the temperature profile. The minor factors from either P. I. 178383 or Chinese 166 conditioned greater resistance at 15/24 than at 2/18 and had just the opposite response of Rego's temperature sensitive factors. Combinations of factors from these parents were additive at both temperature profiles. These minor factors were not entirely in common and transgressive segregation was observed for increased resistance.

The minor factors were also effective in causing the major factors to condition greater resistance, particularly for the heterozygote of either major gene.

The infection types used, ovo, oo, 0-, 0, 1, 2, 3, and 4 were found to have a genetic basis and were good indications of the genotype at the controlled conditions. The infection type distribution for each cross at each temperature was unique as to proportion of different types and range of expression of various factors or combinations of factors.

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A thesis submitted to the Graduate Faculty in partial fulfillment of the requirements for the degree

of

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in

Genetics

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ABSTRACT

Seedling infection types of four wheat varieties and their F_1, F_2, F_3, testcross, and testcross-F_2 distributions were infected with a single unidentified race of Puccinia striiformis and used to study the inheritance of stripe rust resistance genes, their interaction with each other, and with temperature. Two rigidly controlled environment chambers were used, temperature being the important variable. Diurnal temperature profiles with gradual inclines and declines were used which simulated spring and fall conditions, 2 C night/18 C day, and early to mid-summer conditions, 15 C night/24 C day.

Wheat varieties P. I. 178383 ($\overset{v}{oo}$) and Chinese 166 ($\overset{v}{oo}$) had dominant genes for resistance which conditioned a $\overset{v}{oo}$ infection type. The heterozygote of these factors conditioned a oo to 0 infection type. The double heterozygote conditioned a $\overset{v}{oo}$ infection type. In addition, these varieties had minor genes which conditioned 0, 1, 2, and 3 infection types in certain additive combinations.

Rego (0 at 2/18 and 3 at 15/24) had a pair of dominant complementary genes which conditioned a 3 infection type at both profiles. However, at 2/18 Rego had temperature sensitive minor genes which conditioned a 0 infection type in combination with the two complementary genes.

Lemhi (4) was considered to be completely void of resistance genes.

Distributions of segregating progenies showed a significant interaction with the temperature profile. The minor factors from either P. I. 178383 or Chinese 166 conditioned greater resistance at 15/24 than at 2/18 and had just the opposite response of Rego's temperature sensitive factors. Combinations of factors from these parents were additive at both temperature profiles. These minor factors were not entirely in common and transgressive segregation was observed for increased resistance. The minor factors were also effective in causing the major factors to condition greater resistance, particularly for the heterozygote of either major gene.

The infection types used, $\overset{v}{oo}$, oo, 0-, 0, 1, 2, 3, and 4 were found to have a genetic basis and were good indications of the genotype at the controlled conditions. The infection type distribution for each cross at each temperature was unique as to proportion of different types and range of expression of various factors or combinations of factors.

INTRODUCTION

Since the beginning of agriculture, man has selected and reseeded the most acceptable forms of a particular crop, including types resistant to disease. As a result of planting disease resistant types, genes which condition resistance have been accumulated into the present gene pool. The pathologist and plant breeder uses this gene pool to incorporate the desired disease resistance into agronomically desirable crop varieties.

In order for these factors to be efficiently and effectively employed in a breeding program, it would be ideal to have the resistance genes cataloged; that is, to know where these genes are located, how many there are, how they are inherited, what relationships and interactions they have with each other and possibly with other factors, and how the environment influences their potential expression of resistance.

Various aspects of disease resistance were studied in this investigation with the emphasis placed upon the influence of controlled temperature regimes upon the reaction of segregating wheat populations to stripe rust (Puccinia striiformis West). Two diurnal temperature profiles, 2 C night/18 C day and 15 C night/24 C day, were used to study the inheritance of stripe rust resistance. These two profiles simulated the two important environmental patterns present in areas where stripe rust is an economically important disease. The first profile, 2/18, is similar to the spring and fall conditions in which primary and early secondary infections occur. The second profile, 15/24, is similar to conditions in early or mid-summer in which economic losses caused by this disease would be greatest.

The influence of environment on the reaction of cereal varieties to rust fungi is well known. However, most disease resistance studies have been carried out in the field or greenhouse with little or no control of environmental factors, except possibly a constant temperature regime. When constant temperatures are used, an unnatural situation is created for both the host and pathogen. By using controlled environment chambers with simulated diurnal profiles for temperature, light, and relative humidity, the infection type conditioned in response to a specific host-pathogen interaction should be accurately expressed. Sharp (1965) has determined that the response of a host-pathogen complex at a constant temperature was different from the response at a diurnal temperature profile for stripe rust. He observed that the simulated diurnal profile matched the response of certain varieties to field conditions, whereas constant temperatures did not necessarily do so.

The use of broad response classifications for a host-parasite interaction has been a common practice in disease resistance inheritance studies. For example, in the rust diseases of cereals infection types 1 through 2 are generally considered resistant and infection types 3 and 4 are considered susceptible, regardless of the cross or situation. In the present study the infection types conditioned by each cross were investigated at both temperature profiles in an attempt to determine the association between the same genotypes at the two profiles.

REVIEW OF LITERATURE

Stripe rust is most commonly found in areas of the world that have a northwest marine or similar climate. This is the most destructive wheat disease in Europe (Lupton and Macer, 1962) and in the highlands of Northern India (Bahl and Kohli, 1960). It is found in cool intermountain areas (Orjuela, 1956) and the cool southern plains (Vallega, 1955) of South America, in the mountainous areas of Japan and China (Kajiwara, Ueda, and Iwata, 1964), and in the coastal and intermountain valleys of northwestern North America. A general discussion of the distribution of this disease and the favorable climatic conditions was given by Dickson (1956).

The causal organism of stripe rust is Puccinia striiformis West. The life cycle of this basidiomycete is similar to the organism that causes black stem rust of wheat (Puccinia graminis f. sp. tritici Eriks. & Henn.) except that the aecial stage is unknown. The life cycle of P. striiformis was studied in the United States by Hungerford (1923). He concluded that the fungus overwinters in the uredial stage, or may overwinter as mycelium in cereal or grass seedlings, especially when there is sufficient snow cover. He also determined that the hot, dry months of July and August were critical periods for the survival from one crop year to the next and that fall infection was important for heavy epiphytotics to occur the following year. Sharp and Hehn (1963) studied the manner in which this organism survived the winters in the Intermountain Valleys of Montana. They concluded that it overwintered as dormant mycelium in live leaves of winter wheat seedlings and that the native grasses were probably of little importance.

Just as the other rust fungi exhibit specialized forms and physiological races, so does P. striiformis. Hungerford and Owens (1923) found that there were probably two distinct forms, tritici and hordei, in the Northwest.

Within the form tritici many races have been defined. Hungerford and Owens (1923) found that the urediospores collected on one cereal crop, cereal variety, or grass would not always infect different cereal crops, cereal varieties, or grasses. Gassner and Straib (1932) and Straib (1937) found many races in Germany and Europe. Vallega (1955) reported that the races of Chile were different from those found in Argentina or Europe. Bever (1934) found that there were at least two races in the United States. Kajiwara et al (1964) showed that different races occurred on the several islands of Japan and that these races were generally different from those found on the China mainland.

Sharp (1965) reported that only a single race occurred in Montana when the standard differentials were used. However, Purdy and Allan (1963b) gave evidence for several races in Washington. Fuchs (1966) determined that the spore samples of Montana and California gave different infection patterns. Race 13 which is virulent on Chinese 166 was positively identified, but the predominant race appeared to be different and needed more experimentation before description.

Using two resistant and two susceptible varieties of both wheat and barley, Bever (1937) indicated that stripe rust reduced the yield of grain,

stunted root size, and influenced the water efficiency of both susceptible varieties of wheat and barley.

Allan and Vogel (1962) obtained a highly significant correlation of -0.494 between yield and stripe rust reaction when they used near isogenic lines of wheat. Allan, Vogel, and Purdy (1963) showed that yield and test weight losses caused by stripe rust were greater in susceptible short-strawed varieties than in standard-height varieties.

McNeal and Sharp (1963) used moderately resistant Thatcher spring wheat as a check and obtained a highly significant correlation coefficient of -0.839 between yield and the August coefficient of infection. Lemhi 53 sufferred a 34.4% loss, while other susceptible spring wheat varieties suffered losses in excess of 23%. They attributed no reduction in test weight to the epidemic of stripe rust.

However, Pope, Sharp, and Fenwick (1963) estimated yield losses as high as 60% and test weights as low as 48 pounds per bushel for Lemhi in Southern Idaho. In the mountain valleys of Montana, they estimated winter wheat losses from 25 to 60% on susceptible varieties.

Sunderman and Wise (1964) stated that stripe rust lowered the yield of susceptible plants by 37.5%, of which 19% was due to lower kernal weight and 17.6% was due to fewer seeds. The grain from these plants also had 1.7% lower protein content, and several test values used to measure the quality of flour were affected. They suggested that plant breeders should be cognizant of the effect of stripe rust on varieties in evaluation of nurseries, otherwise erroneous conclusions may be reached about

the milling and baking data.

Shortly after the rediscovery of Mendel's Laws in 1900, Biffen (1905) demonstrated that resistance to stripe rust was inherited in a Mendelian fashion. This was the first instance in which disease resistance was shown to segregate in a Mendelian ratio. In this classic experiment the very susceptible Red King (Triticum vulgare) was crossed with resistant Rivet (T. turgidum) to produce susceptible F_1 plants. In the F_2 there was clearly a one resistant to three susceptible segregation. In the F_3 the resistant plants remained resistant and the susceptible plants segregated for resistance and susceptibility. This clearly indicated that one recessive gene conditioned resistance. In a later experiment, Biffen (1907) showed that the immunity of American Club was also inherited as a single Mendelian recessive in a cross with susceptible Michigan Bronze.

Nilsson-Ehle (1911) was the next to show that resistance to stripe rust in cereal varieties was inherited in a Mendelian manner. Using parents that were different from those used by Biffen, he obtained data which indicated multigenic rather than monogenic inheritance for resistance.

Despite these three early demonstrations of genes for stripe rust resistance, the inheritance studies for this disease have lagged behind those for most of the other important cereal diseases. This may have been partially due to the sporadic occurance of stripe rust, or to the fact that several races have complicated the inheritance patterns, or to the difficulty in handling the inoculum.

Unlike stripe rust resistance studies, many cases have been reported for resistance to stem rust and leaf rust of wheat and barley and crown rust of oats. The early studies were summarized by Ausemus (1943). Active groups have continuously studied resistance to these diseases in most countries where they are prevalent.

Armstrong (1922) was next to report a factor for resistance to P. striiformis in wheat. In a cross of Wilhelmina x American Club, he also obtained a three to one segregation for susceptible to immune plants in the F_2 of adult plants.

Rudorf (1929) was the first to use seedling plants and a greenhouse to carry out resistance studies to stripe rust. He showed that resistance at the seedling stage was simply inherited and defined a number of resistant varieties. He showed that the hypersensitive reaction of Chinese 166 was unaffected by environmental conditions, and he was the first to demonstrate that this reaction was inherited as a dominant.

Straib (1934; 1937) also demonstrated that Chinese 166's immunity was inherited as a single dominant character to all 18 physiological races to which it was subjected. Other resistant varieties were also shown to be controlled by one or more dominant genes to various races.

In a study to determine if the factors that conditioned stem rust reaction were the same as those that conditioned stripe rust reaction in several crosses, Neatby (1936) found that the seedling reaction to P. striiformis was closely associated with the mature plant reaction to P.

graminis, and he concluded that the relationships were probably due to a plieotropic effect of the genes concerned rather than to genetic linkage.

Bever (1938) studied the varietal reaction to stripe rust of 317 American wheat varieties including common, club, durum, emmer, poulard, and polonicum wheats and 1284 foreign introductions. He found that the common white group was more susceptible than either the soft red winter, hard red winter, or durum groups. The durum group was the most resistant. The club wheats, as a whole, were the most susceptible, there being only one resistant variety in the entire group.

Pal (1951) and Pal, Sikka, and Rao (1956) made a number of crosses between varieties resistant to the Indian races and found that single dominant genes were responsible for the resistance of Chinese 166, Spalding's Prolific and Carsten's V, and they found that there was a trifactorial inheritance with the resistant Frondoso with susceptible NP. 789.

Favret and Vallega (1953) reported that Chinese 166 had one dominant factor for resistance to the Argentina races. Later Vallega (1955) indicated that the races of Chile were able to overcome the resistance of this variety. Fuchs (1966) reported that race 13 of the United States was also virulent on Chinese 166. Kajiwara et al (1964) also showed that the races of Japan were virulent to Chinese 166.

Konzak et al (1956) irradiated the susceptible Gabo variety of wheat and induced a resistant true-breeding line. The mutation was inherited as a recessive. The Gabo parent had a 4 infection type while the mutant

possessed a 1 infection type.

Allan and Vogel (1961) showed that one dominant gene controlled adult resistance of Suwon 92 to the prevalent race that occurred in Eastern Washington at that time.

Singh and Dhillon (1963) found that the resistance of Rio Negro, Centaria, and La Prevision was controlled by a single dominant gene and that resistance in Frontana was controlled by two dominant genes.

Gosh, Sikka, and Rao (1958) found a single dominant gene governing resistance of Cometa Klein to the most prevalent Indian races.

Bahl and Kohli (1960) reported the results of two crosses using two resistant parents, Cometa Klein and Rio Negro, and two races of P. striiformis, 13 and H. The resistance of Cometa Klein against both races was controlled by two pair of complementary genes, both of which are contributed by Cometa Klein. A nine to seven ratio was obtained with seedling F_2 plants. The genetic constitution of Cometa Klein was proposed to be $R_1R_1R_2R_2$ and that of the susceptible parent as $r_1r_1r_2r_2$. R_1 and R_2 were independent, and both were required to condition resistance. Although this cross fit nine to seven ratios for both races, the genes contributing to resistance appeared to be different on the basis of a chi-square test of independence on 97 F_3 families.

In the second cross reported by Bahl and Kohli, the inheritance of resistance of Rio Negro to race 13 in a cross with Pb. C. 518 revealed that interaction of a gene for resistance and an inhibitory gene was

involved. Rio Negro contributed the dominant gene R for resistance while Pb. C. 518 contributed the dominant inhibitory gene, I. The presence of R and the absence of I were necessary for resistance. The resistant parent then had the genotype RRii and the susceptible parent the genotype rrII. There was an F_2 ratio of 3 resistant to 13 susceptible. Infection types were read according to the scheme of Gassner and Straib (1932). Infection types 0, 1, and 2 were considered resistant, and types 3 and 4 were considered susceptible. The actual infection types of the parents, F_1, or F_2 seedling plants were not given.

In a later paper by Nambisan and Kohli (1961), Cometa Klein was again reported to have two dominant complementary genes for seedling resistance to races 13 and H. They also showed that the resistant variety La Prevision had a dominant gene which was inhibited by a dominant gene of Selection NP. 770.

The earlier results of Singh and Swaminathan (1959) were not entirely in agreement with the later findings of Bahl and Kohli (1960) and Nambisan and Kohli (1961). Using monosomic analysis, they identified three recessive genes on three chromosomes of Cometa Klein that conditioned resistance to P. striiformis. They reported that recessive factors which controlled resistance to race H were located on chromosomes IV and VI and one recessive gene which controlled resistance to race 13 was located on chromosome IX. In this monosomic study Cometa Klein was crossed with the 21 monosomes of Chinese Spring. All 21 F_1 lines were susceptible. The

F_2 deviations from the expected were used to locate the chromosomes carrying resistant factors. With race H, recessive complementary genes were expected to condition resistance, or there should have been a seven resistant to nine susceptible ratio. With race 13, a single homozygous recessive gene was expected to condition resistance, or the F_2 data were fit to a one resistant to three susceptible ratio. These results were nearly opposite those obtained by Bahl and Kohli (1960).

These deviations are unexplained unless the infection types that were considered resistant or susceptible varied. For this study, susceptible infection types were considered to be 3 and 4 types while resistant types were considered to be 0, 1, and 2 types. Plants that were between types 2 and 3 were grouped separately. The manner in which these separate studies were classified into response groups may then account for the differences in the proposed genetic systems involved. Since it is also known that some varieties respond differently at different temperatures (Sharp, 1965), there may have been enough temperature variation to significantly change the apparent results between the two studies.

Up to the time of Lupton and Macer's (1962) report, there had not been a published account of the relationship between the various factors that condition resistance to P. striiformis. No deliberate effort had been made to find the commonness of factors between the resistant varieties or the allelism that may occur for resistance to different races. Also, except for the account of Singh and Swaminathan (1959), the linkage

relationships were not known.

To determine the relationship between genes for resistance in some of the important wheat varieties grown in England, Lupton and Macer made a diallel set of crosses between seven varieties. In the F_1, F_2, and F_3 of these crosses, the rust reaction to four physiological races was used to identify seven genes conferring resistance at four loci. In general, they found resistance to be inherited as a dominant character, although cases of recessive resistance to very aggressive races of the rust organism occurred.

In this study, seedling plants were divided into susceptible and re-resistant groups. Plants having infection types 0 or 1 were classified resistant, and those having infection types 2, 3, or 4 were classified susceptible for all crosses. Gassner and Straib's (1932) scheme was used. The plants were grown in greenhouses and, except for one year, were subjected to wide temperature variations. The greenhouse was maintained at a constant 60 $\pm$ 5 F during the last year of the experiment. The results of chi-square tests of goodness of fit to many of the F_2 ratios were very poor.

They found that Chinese 166 possessed a single gene, Yr_1, for resistance to all races. Heine VII and Soissonais possessed the same gene, Yr_2, for resistance to three of the four races. Holdfast was the susceptible variety and possessed no genes for resistance. Cappelle had a third dominant gene Yr_{3a}, for resistance to two of the races, and two recessive

genes yr_{3a} and yr_{4a}, for resistance to a third race. Hybrid 46 carried two dominant factors for resistance, Yr_{3b} and Yr_{4b}, one being allelic to the Cappelle factor, Yr_{3a}. Hybrid 46 may also have had an allele of the second Cappelle locus, Yr_{4b}.

Lupton and Macer stated, "Resistance is thus determined by a relatively simple major gene system, similar to that shown by Knott and Anderson (1956) to determine the reactions of wheat to stem rust."

Even after this account, the interactions between resistant loci in the same plant were very meagerly known. The majority of papers reported one, two, or three dominant or recessive factors for resistance. Simple cases of interallelic action were reported by Bahl and Kohli (1960), Nambison and Kohli (1961), and Singh and Swaminathan (1959). Lupton and Macer (1962) also noted gene dosage effects between two dominant genes to give greater resistance. However, all of these reports supposedly dealt with major genes, and resistance or susceptibility fell into qualitative classes. The influence of minor or modifying genes that more accurately fall into a quantitative system were not discussed.

The first possible mention of quantitative inheritance of stripe rust resistance was by Allan et al (1963). Studying the influence of stripe rust on yield of closely related lines, they noted that some fourth-generation backcross lines of (Norin 10 - Brevor 14) x Burt were more resistant than the susceptible parents in 1962. This moderate level of field resistance indicated that complementary gene action was involved in

the expression of resistance between susceptible or moderately susceptible varieties and that, in effect, transgressive segregation for greater adult field resistance had occurred.

Purdy and Allan (1963a) found that seedling reactions were generally indicative of the mature plant reaction, although the adult reaction of certain semi-dwarf selections could not be reliably predicted at the seedling stage.

In a later study, Allan et al (1966) determined the relationship between seedling and adult resistance. They found that an association existed between adult and seedling reactions for every cross studied. There were two crosses, however, Itana x Spinkcota and Dickson 114 x Itana, that had major discrepancies between field and greenhouse reactions. Generally, more lines were resistant as adult plants than as seedlings. They also encountered transgressive segregation for adult resistance to stripe rust in the Itana x Burt cross. Simple monogenic inheritance occurred in crosses between C. I. 13431 x Golden, C. I. 13431 x Itana, and Spinkcota x Itana. Two complementary loci, or epistasis between loci in the greenhouse seedling stage, were found for Norin 10 - Brevor 14 x Nord Desprez, Spinkcota x Golden, and Dickson 114 x Itana. In all of the Dickson 114 crosses there were two or three loci involved in the seedling stage.

Using field observations on pure-line wheat varieties and their segregating progeny, Pope and Henriksen (1964; 1965), Henriksen and Pope (1965), and Pope (in press) found that in all wheat varieties observed, only Lemhi had no genes for resistance. All varieties with near total

susceptibility contributed a heritable resistance to stripe rust by transgressive segregation in appropriate crosses. Varieties that were intermediate for stripe rust reaction showed either no segregation (genes in common) or transgressive segregation, suggesting that all gene combinations were more or less additive. Reactions with the tested wheat varieties suggested at least 20 different resistance genes to P. striiformis. They stated that these genes were apparently recessive.

Using a controlled diurnal temperature profile of 15/24 C, Lewellen, Sharp, and Hehn (1965) showed that P. I. 178383 possessed a major dominant gene and several recessive genes for stripe rust resistance in seedling plants. By selecting F_2 plants that had an infection type 4 and growing out F_3 lines, they demonstrated that the minor factors in P. I. 178383 conditioned various levels of resistance and were probably additive.

Metzger and Silbaugh (1966), using monosomic analysis, indicated that P. I. 178383 carried a single dominant locus for resistance and that this gene is linked with the gene for brown glume color in P. I. 178383 with two crossover units distance. McCallum, Welsh, and Sharp (Personal communication, 1965) located this gene on chromosome 2B.

All of the investigations with stripe rust have been carried out under field conditions or in greenhouses where temperatures were either not controlled or were controlled at a single level. However, various investigations have shown that the resistance of some wheat varieties to P. striiformis is quite variable under changing environmental conditions. Gassner and Straib (1932) indicated that varieties resistant to P. glumarum

(P. striiformis) at 20 C were severely rusted at slightly lower temperatures. Newton et al (1933) found that all the wheat varieties tested were resistant to stripe rust at 78 F, but that many varieties were susceptible at 54 F.

Generally it was found and observed that increasing temperatures increased resistance, but Manners (1950) showed that high temperatures increased the susceptibility of some differential hosts to certain stripe rust races but decreased that of others. Sharp (1962a,b) indicated that the amount of infection or severity varied with different host-parasite combinations, but was usually greater when plants were grown at lower temperatures prior to inoculation. He suggested that physiological changes that influence the reaction of a particular race with a particular variety must take place before as well as after inoculation.

The influence of temperature upon the expression of resistance of wheat varieties to P. striiformis has been most accurately studied by Sharp (1965). The host plants were grown in walk-in environment chambers rigidly controlled and programmed for temperature, relative humidity, and light. Both constant and diurnal temperature profiles were investigated. In the latter, declines and inclines from extreme temperatures were gradual. A single unidentified race of P. striiformis was used for all inoculations.

Sharp found that the wheat varieties Westmont, Omar, Lemhi, and Idaed were all highly susceptible at a constant 15 C. As temperatures were

increased to 24 C, all varieties became resistant with Lemhi remaining the most susceptible at all temperatures. Using the diurnal temperature profiles, Sharp found that P. I. 178383 and Westmont showed no shift, and this tended to be the case for all varieties which were either highly resistant (P. I. 178383) or highly susceptible (Westmont). Turkey-1 and Idaed showed decreasing compatibility with increasing temperature profiles, whereas Rego reacted in just the opposite manner. He showed that the temperature during the dark period was most important.

Investigations to determine the inheritance of the temperature sensitive factors have not been reported in the literature. However, an active project is underway in Dr. Sharp's laboratory at Montana State University to determine the same as well as to more accurately determine the inheritance of the recessive minor factors which appear to be additive in conditioning stripe rust resistance.

Investigations to determine the quantitative nature of disease resistance and the influence of environment have been carried out for several other plant diseases. However, the general procedure has been to account for those resistances caused by major genes which are not influenced by the environment. The gene-for-gene systems first described by Flor (1956) for flax rust and later found to occur in late blight of potatoes (Toxopeus, 1956), powdery mildew of barley (Moseman, 1959), stem rust of wheat (Loegering and Powers, 1962; Williams, Gough, and Rondon, 1966), and theoretically for any disease (Person, 1959) have been based entirely upon these major dominant factors for resistance. As an example, in the recent studies

by Berg, Gough, and Williams (1963), Knott and Anderson (1956), Knott (1962; 1956), Rondon, Gough, and Williams (1966), and Sunderman and Ausemus (1963) with stem rust resistance only the major, dominant genes were described.

Green et al (1960) and Green and Knott (1962) have shown that minor or modifying genes were involved in the expression of stem rust resistance in both seedling and adult plants. In these studies, resistance genes were transferred into a completely susceptible background using substitution lines. They stated:

> "The reaction of the substitued lines and the Marquis lines carrying the same genes were similar, although the substituted lines were more susceptible to some races. With the Sr_{10} gene for resistance, resistance was lost with each backcross and the gene became increasingly difficult to detect. This gene was variable with temperature change and resistance was diminished at high temperatures."

Knott (1957a; 1957b) encountered the "modifier effect" in earlier work and explained:

> "The 'modifier effect' probably explains many of the difficulties encountered in maintaining full resistance while backcrossing to produce rust resistant varieties. While the genes reported determine whether a variety is or is not resistant to races of 15B and 56, the degree of resistance they condition is variable in different crosses, and it is not known whether the difference is due to specific modifiers or to the genetic environment in general. For example, Thatcher's resistance to race 56 is probably due to modifiers that this variety apparently possesses. These modifiers are necessary in connection with other major genes in order to get good resistance to race 15B."

Schafer et al (1963) used varieties with less than immune reactions to leaf rust of wheat (which, they stated, has generally not been done for

any rust disease) and showed that they could get better resistance from true breeding F_3, F_4, or F_5 lines than was apparent in either parent. All combinations of the moderately resistant parents yielded some lines with better resistance than either parent, even though the actual genetic constitutions of the parents were not known. They suggested that these factors may provide a better source of long term resistance than single major gene resistances that can be overcome by a single mutation in the pathogen.

A similar interaction of resistance factors was reported earlier by Finkner (1954) for crown rust of oats.

Diseases caused by rust fungi are not the only ones that respond in this manner. For example, Wasuwat and Walker (1961) reported that the degree of resistance to cucumber mosaic virus controlled by a major gene may be increased or reinforced by an undetermined number of modifying genes.

In the review paper by Wingard (1941) and the book by Walker (1957), the effects of environment upon the level of resistance in various host-pathogen interactions were discussed. However, few of these related to the rust diseases of cereals and most related to the diseases in which the pathogens were soil borne. There have been only a few accounts of the influence of environment on the cereal rusts since.

In a study to determine at which constant temperature a temperature sensitive variety changes response to several races of P. graminis, Bromfield (1961a) found that the breakdown period for seven sensitive

varieties was between 70 F and 77 F. In general, the varieties were resistant at 70 F, mixed in reaction at 72 F to 74 F, and susceptible at 76 F. In a later study Bromfield (1961b) determined that the temperature sensitive wheat varieties Kenya N. B. 263 and Lerna 52 were resistant to P. graminis at 70 F and when transferred to either a constant 77 F or 85 F did not retain the resistance. Conversely, varieties that were grown at constant 77 F or 85 F and moved to 70 F did not retain the susceptibility. He concluded that the effect of temperature was most likely on the host-parasite complex and not on either of the components independently.

The implication of results from Green and Johnson (1955) would be that specific temperature sensitive genes occur in the host, the parasite or both. In this study the reaction of adult plants of ten wheat varieties to ten races of stem rust at temperatures of about 60 F and 80 F was investigated. The temperature at which resistance or susceptibility for certain combinations of host variety and rust race broke down was specific. If a one to one genic relationship is valid for host-parasite interactions, then the differences observed by different combinations would be due to specific temperature sensitive factors present in one or both of the component parts.

The classic example of the influence of temperature upon disease expression is the reaction of cabbage to Fusarium oxysporium f. sp. conglutinans (Wr.) Snyder & Hansen (Walker and Smith, 1930; Blank, 1937; Walker, 1963). Two types of resistance were shown to exist in cabbage. Type A

resistance was shown to be monogenic, dominant, and stable to temperature changes. The type B resistance was shown to be multigenic, only partially dominant, and varied with the temperature. At a low soil temperature the type B conditioned resistance equal to that of type A, however, as soil temperatures increased the plants with type B resistance became more and more susceptible. It was shown that the more type B genes possessed by a variety of cabbage, the higher the soil temperature needed to be to break down the resistance.

Walker (1963) stated the same type of A and B resistances occur for fusarium wilts of tomato varieties and for some potato varieties resistant to some races of Phtyphthora infestans (Mont.) DeBy.

This second type of resistance (type B) is probably what accounts for field resistance in many varieties of agronomic and horticultural crops.

MATERIALS AND METHODS

Parent varieties used were P. I. 178383, Chinese 166, Lemhi (C. I. 11415), and Rego (C. I. 13181).

P. I. 178383 was screened from the world collection as a source of resistance to several races of dwarf bunt, Tilletia controversa Kühn, and it was subsequently found to possess resistance to stripe rust. It has been used as a parent in several breeding programs specifically designed to obtain an agronomically acceptable wheat that is stripe rust and/or bunt resistant. It was used as a parent in the new stripe rust resistant variety 'Moro'. Except for resistance to smut and rust, P. I. 178383 has little agronomic value because of undesirable field characteristics.

Chinese 166 has been the most commonly used variety for stripe rust inheritance studies. It was included by Gassner and Straib (1932) as one of the standard differentials. Lupton and Macer (1962) give the history of this variety, which was collected in Western China early in the 20th century. Chinese 166 is a winter wheat with soft, red kernels and has been used both as a variety and parent in Western Europe.

Lemhi is the most susceptible variety known to prevalent Northwestern races of P. striiformis (Pope, 1965; Sharp, 1965) and, therefore, it made an ideal susceptible parent for inheritance studies. It is a soft, white, spring wheat most commonly grown in the irrigated valleys of Southeastern Oregon, Southern Idaho, Nevada, and Utah. Lemhi was selected from a cross between Federation and Dicklow and released in 1939 by the Idaho Agricultural Experiment Station.

Rego was selected for this study because of its unusual temperature response in regard to stripe rust infection. Sharp (1965) showed this variety to be more incompatible to P. striiformis at low temperatures than at high temperatures. This hard, red, winter variety was selected from a Yogo x Rescue cross as a solid-stemmed, sawfly resistant variety by the Montana Agricultural Experiment Station and released in 1956. Since 1962 it has been grown on some Montana acreage because of its moderate resistance to stripe rust.

Originally, a diallel set of crosses was to be made with these varieties, with each F_1 being backcrossed to the parents, except where one of the parents was immune to P. striiformis. The Rego x Chinese 166 cross was missed. The parents were grown in a crossing block in a greenhouse bench in the spring and summer of 1963. The parents were grown from selfed plants which had previously been checked for uniform rust reactions.

The parents, F_1, F_2, F_3, testcross, and testcross-F_2 seedlings were used to determine the inheritance of resistance. All seedling plants checked for their infection type to stripe rust were grown in one of two controlled environment chambers described earlier by Sharp (1965).

> "The chambers were rigidly controlled and programmed for temperature, relative humidity, and light. Two diurnal temperature profiles were used in which declines and inclines from extreme temperatures were gradual. The low temperature profile was maintained at cyclical temperatures of 2/18 (2 C at night to 18 C during the day), and the high temperature profile was maintained at 15/24 (15 C at night to 24 C during the day). Relative humidity was about 95% during the dark period and 65% during the light. Photoperiods were 12 hours. Light intensities

were increased stepwise from 300 to 1,800 to a maximum of 3,500 ft-c at the middle of the photoperiod and then decreased through a similar range."

The same collection of inoculum was used for the entire experiment. The inoculum was collected in the nursery plots west of Bozeman in August, 1963. After germination tests, the inoculum was stored by lyophilization in a manner previously discussed by Sharp (1965) and Sharp and Smith (1952). Samples of this inoculum and cultures developed from single pustules and single spores of the collection were tested on both the standard differentials of Gassner and Straib (1932) and on supplemental wheat varieties and gave no evidence for more than one physiologic race (Sharp, 1965). This race is believed to be the same race which Fuchs (1966) was unable to describe and is probably still the predominant race in Montana.

The infection types used are basically those of Gassner and Straib (1932) with some modifications and additions. Gassner and Straib defined six infection types: i = immune, no evidence of infection; 0 = chlorosis or necrosis without pustules; 1 = chlorosis or necrosis with few very small pustules; 2 = chlorosis or necrosis with small pustules; and 3 = chlorosis without necrosis; and 4 = normal pustulation without chlorosis.

Instead of the i infection type, the $\overset{v}{o}o$ infection type was substituted. When even the most resistant plants were inoculated and given a proper dew period, there appeared very minute chlorotic flecks as evidence that the pathogen had penetrated but had very quickly been destroyed by the host tissue. The next most resistant infection type used was designated oo. The characteristic of this infection type was a larger,

symetric fleck, chlorotic or necrotic, about one to two times as large as the period at the end of this sentence. The 0- infection type was the next most resistant averaging midway between the oo and 0 types. This infection type was less symetric, generally necrotic, and larger than the oo, but did not span across the entire leaf. The 0 infection type is much as described earlier, but encompassed more of the leaf than any of the other previously mentioned types. These types, $\overset{y}{o}o$, oo, 0-, and 0, represent varying degrees of host-parasite hypersensitivity, with the $\overset{y}{o}o$ being so hypersensitive that immediate death occurs to the pathogen and probably to the layer of cells surrounded by the penetration tube in the host. Hypersensitivity then decreases to the 0 type in which death to both the host and pathogen cells are delayed, and a very incompatible interaction is established, but death eventually occurs to the parasite without pustulation occuring.

From the 0 type through the 4 type compatibility increases, until the most compatible relationship or interaction possible between a host and parasite is established in the 4 infection type. The infection types 1, 2, 3, and 4 were used much as defined by Gassner and Straib (1932). The 1 infection type was divided into 1- and 1 types. With some plants that first appear to be 0 types, a few pustules eventually form on the perimeters of the necrotic host tissue. These plants are described as having the 1- types. The 2 type was used as described. The 3 type was split into 3 and 3- types. With some plants, particularly those derived from Rego parentage, the decision to call the infection type 2 or 3 was

questionable, because of the nature of the chlorosis or necrosis. These plants were given the 3- infection type. Plants that were very susceptible were called 4 types. From observing the infection types 0 through 4, one begins to see reactions that no longer appear to be hypersensitive *per se* but begin to take on the appearance of a "nutritional basis" of incompatibility or compatibility, with a struggle being waged by the host, pathogen, or both to maintain themselves in a beneficial equilibrium.

Unlike most other rust fungi, *P. striiformis* grows vegetatively throughout a compatible host leaf by runner hyphae. Many infections are not necessary, because one infection will soon cover most of a leaf. The infection types were the only disease readings used. Severity (average per cent of leaf area infected) and prevalence (percentage of plants showing infection) were not used. Plants that were definite escapes were not counted.

Generally, twenty seeds were planted in a row across a four-inch clay pot containing Gallatin sandy loam with one-fourth part peat moss. The pots were subirrigated in galvanized steel trays. The seedlings were inoculated in the first leaf stage just as the secondary leaf began to appear. The leaves were bound in a horizontal position by the use of metal backboards and rubber bands. Inoculations were made in a modified settling tower (Sharp, 1965) in which a CO_2 pistol was used to disperse the urediospores and to allow an even distribution on the adaxil surface of the leaves. Two lyophilized tubes, each containing 40 mg of spores,

were used for each set of 15 to 20 pots. After allowing four minutes for the spores from the first tube to settle on the plants below, the pots were rotated 180° and the second tube was fired.

After the backboards were removed, the pots were placed in a dark dew chamber which is a small chamber within a larger, walk-in chamber. The air temperature of the walk-in chamber was maintained at a constant 2 C. In the small chamber, the water bath temperature was 10 C and the air temperature was 5 C, allowing ample dew to form on the leaves. After forty-eight hours the plants were returned to their original environment chamber. To check spore germination, either a collodion strip or a polyethylene membrane was used (Sharp, 1965).

The plants were scored for rust reaction when the pustules on the susceptible check were adequately developed. This required 14 to 16 days at 15/24 and 21 to 23 days at 2/18. Before reactions were read, the secondary and tertiary leaves were removed. The plants were scored twice to insure accurate readings. Plants that germinated late were generally removed and not counted.

Because the plants scored as seedlings were used to produce the following generation, specific seedling plants were marked with paper tags and saved. All unwanted plants were removed from the pot. The selected plants were moved to a vernalization chamber and maintained at a diurnal cycle of 2/10 C for six to eight weeks. The plants were then transplanted into eight-inch pots which were placed on greenhouse benches.

Because the top florets of the heads appeared to be male sterile, all heads were covered with glycine bags prior to anthesis to insure only selfed seed. Standard procedures were used to make the desired crosses. Morphological checks were made on all progenies to insure that the proper crosses had been made. The harvested seed from each crossed head or selfed plant was maintained separately. These lots of seed were considered a random sample for that particular experiment and were pooled for analysis.

Two chi-square tests were employed to analyze the data. Chi-square tests of goodness of fit were used to analyze data from segregating populations for one, two, or three factors. Chi-square tests of independence were used to test the hypothesis of the independence of two segregating distributions, either of the same cross at different temperature profiles or of different crosses at the same temperature profile. This test is also called a test of interaction and is described by Steel and Torrie (1960).

> "The hypothesis of independence implies that the ratio of numbers of plants emerging from treatment to treatment is the same, within random sampling errors. If there is no independence, there is said to be interaction. In the case of interaction, the ratio from treatment to treatment is dependent on the variable, i. e., the variables are not independent. No assumptions need be made about the true ratios for either variable, either within any category of the other variable or over all categories. If there is a significant chi-square, the table of contributions would supply information on possible causes."

An analysis of variance and Duncan's New Multiple Range Test were used to draw conclusions about mean infection types for the Rego x Lemhi cross.

EXPERIMENTAL RESULTS

I. Lemhi x P. I. 178383

Lemhi x P. I. 178383 at 2/18 The infection types of the parents, F_1, F_2, and Lemhi testcross distributions are presented in Table I. The oo type for the F_1 plants indicated that the gene or genes in P. I. 178383 were partially dominant.

The F_2 and the Lemhi testcross are shown giving the total number of plants observed for each infection type. The F_2 infection types were divided into three classes in which resistance was equal to P. I. 178383, $\overset{v}{o}o$, resistance was equal or slightly more susceptible than the F_1, oo, O-, and O, and susceptibility was nearly equal to the Lemhi parent, 3 and 4.

When the F_2 segregation was subjected to a chi-square test for either a 3:1 or 1:2:1 ratio, a good fit was obtained. This indicated either one completely dominant gene for resistance or one partially dominant gene for resistance depending upon which infection types were included in the various classifications.

In the testcross segregation the $\overset{v}{o}o$ infection type was absent. The absence of this type plus a good fit to a chi-square test of a 1:1 ratio further indicated that P. I. 178383 contained one major gene for resistance and that it expressed itself as being partially dominant.

Thus, the homozygous dominant and heterozygous condition of the major gene were distinguishable. The heterozygote apparently conditioned a oo to O infection type range at this temperature profile. Table II demonstrates that F_2 plants that were $\overset{v}{o}o$ types in the F_2 remain homozygous for approximately P. I. 178383's resistance, F_2 plants

Table I. The infection types of the parents, P. I. 178383 and Lemhi, the F_1, the F_2, and the testcross distributions at 2/18 and results of chi-square tests for goodness of fit.

Parents and progeny	Infection types and observed no. of plants									Total	Ratio*	X^2 value	P value
	v oo	oo	0-	0	1-	1	2	3	4				
P. I. 178383	all												
Lemhi									all				
F_1		all											
F_2	30	39	13	20				1	35	138			
		92					36				3:1	.09	.75-.90
	30		72				36				1:2:1	.78	.50-.75
(Lemhi x 178383) x Lemhi		4	13	5				5	28	55			
			22				33				1:1	2.20	.10-.25

* The 3:1 includes types v oo, oo, 0-, 0 to types 1-, 1, 2, 3, 4. The 1:2:1 includes type v oo to types oo, 0-, 0 to types 1-, 1, 2, 3, 4.

that were oo, O-, or O types in the F_2 segregated in a similar manner as did the $F_{2'}$ and F_2 plants that were 4 types segregated from 0 to 4 types (see Table I and Table II in the appendix).

Table II also presents the results of the F_3 segregation of forty-five randomly selected F_2 plants. If P. I. 178383 contains only a single major gene, then there should be a 1:2:1 ratio in the segregation patterns produced by these F_3 lines. A satisfactory fit to this ratio by a chi-square test confirmed this prediction.

To determine if the oo and O types were conditioned by variations of the same genotype or whether they were the result of a heterozygous major gene and differences in minor modifying genes, the F_3 lines that were selected from each of these infection types were pooled and compared. The individual F_3 lines of each infection type were tested for homogeneity and a non-significant value was obtained.

Because the oo and O lines are conditioned by a heterozygous major gene, these pooled F_3 lines should still segregate for either a 1:2:1 or 3:1 ratio. When the pooled oo lines were tested for a fit to these ratios, significance occurred (see Table III). The lack of fit to a 3:1 may have resulted from a number of escapes occurring and being called $\overset{v}{oo}$ types. These families were checked in January when Sharp and Pool (1965) showed that the germination of P. striiformis spores may be very low. When the pooled O lines were checked for goodnes of fit to a 1:2:1 or 3:1 ratio, a good fit was obtained only for the 3:1 ratio.

Although neither of the combined families segregated for a

Table II. Seedling infection types of F_3 lines from randomly selected F_2 plants from Lemhi x P. I. 178383 at 2/18.

F_2 infection type at 2/18	F_3 distribution of infection types from randomly selected F_2 plants								No. of F_3 lines	Obs. no.	Exp. no.	P value*	Probable F_2 genotype
v o o	v o o								8				AA
	v o o	oo							3				AA
	v o o	oo	0-						2	13	15		AA
oo, 0-, 0	v o o	oo	0-	0	1	2	3	4	1				Aa
	v o o	oo	0-	0		2	3	4	1				Aa
	v o o	oo	0-	0			3	4	6				Aa
	v o o	oo	0-	0				4	7				Aa
	v o o	oo	0-			2	3	4	3				Aa
	v o o	oo	0-				3	4	6				Aa
	v o o	oo				2	3	4	6				Aa
	v o o	oo					3	4	3	33	30		Aa
4				0	1	2	3	4	2				aa
					1	2	3	4	1				aa
						2	3	4	6				aa
							3	4	3				aa
	died								2	14	15	.50-.75	aa

* Chi-square test for goodness of fit to a 1:2:1

Table III. F_3 distributions of pooled $\overset{y}{oo}$, oo, 0, and 4 lines from Lemhi x P. I. 178383 and the goodness of fit of the pooled oo and 0 lines to ratios of 3:1 and 1:2:1 when tested at 2/18.

F_2 infection types at 2/18	Pooled F_3 infection types and observed no. of plants								Total	Ratio*	X^2 value	P value
	$\overset{y}{oo}$	oo	0-	0	1	2	3	4				
Pooled $\overset{y}{oo}$ lines	516	35	3	2					556			
Pooled oo lines	423	288	44	5	1	19	48	123	951			
		760				191				3:1	12.25	<.005
	423		337			191				1:2:1	192.14	<.005
Pooled 0 lines	59	82	81	53			14	66	355			
		275				80				3:1	1.15	.25-.50
	59		216			80				1:2:1	19.18	<.005
Pooled 4 lines				2	5	48	77	411	543			

* Ratios include same infection types as Table I.

1:2:1 ratio, they appeared to deviate from this ratio for different reasons. The oo lines had too many $\overset{v}{o}o$ types in relation to oo, O-, and O types while the O lines had too few $\overset{v}{o}o$ types in relation to oo, O-, and O types. The pooled $\overset{v}{o}o$ types also presented in Table III indicated that plants that contained the homozygous major gene still segregated for types more susceptible than a $\overset{v}{o}o$ type at this temperature profile. The pooled 4 type lines given in the same table also showed that a few O types were conditioned in plants wtih a homozygous recessive major gene.

A chi-square test for independence was run to compare the segregation of the pooled oo and O lines to determine if they were conditioned by different genotypes. A significant chi-square value indicated that the two F_3 distributions were different. The greatest deviations occurred in the excess numbers of $\overset{v}{o}o$ and oo types for the oo lines and the excess of O- and O types for the O lines (see Table IV). There were then different genotypes that conditioned the expression of the F_2 oo and O types, and it was possible to read these infection types as different responses to this disease. These factors would appear to be minor genes that modify the expression of the heterozygous major gene. The effect of these factors could then be responsible for the lack of fit to a 1:2:1 ratio.

A comparison was also made between the parts of the pooled oo and O lines that have a homozygous recessive major gene (i.e., types 1, 2, 3, and 4) to determine if these minor factors have any influence without the presence of the heterozygous major gene. Table V presents the

Table IV. Chi-square test for independence of F_2 infection types of Lemhi x P. I. 178383 using the pooled F_3 lines selected as oo and 0 types at 2/18 in the F_2.

F_3 infection types at 2/18		Distribution of pooled F infection types from: oo F_2 plants	0 F_2 plants	Totals
oo	Observed	423	59	482
	Expected	351.0	131.0	
	Deviation	+72.0	-72.0	
	X^2	14.8	39.4	
oo	Observed	288	82	370
	Expected	269.4	100.6	
	Deviation	+18.6	-18.6	
	X2	1.3	3.4	
0-	Observed	44	81	125
	Expected	91.0	34.0	
	Deviation	-47.0	+47.0	
	X^2	24.3	65.0	
0	Observed	5	53	58
	Expected	42.2	15.8	
	Deviation	-37.2	+37.2	
	X^2	32.8	87.6	
1-4	Observed	191	80	271
	Expected	197.3	73.7	
	Deviation	-6.3	+6.3	
	X^2	.2	.5	
Totals		951	355	1306

X^2 = 269.3**. Significant at the 1% level.

Table V. Chi-square test for independence of F_2 infection types from Lemhi x P. I. 178383 comparing types 1, 2, 3, and 4 from the pooled oo and O F_3 lines grown at 2/18.

F_3 infection types at 2/18		Distribution of pooled F_3 infection types from: oo F_2 plants	O F_2 plants	Totals
1	Observed	1		1
	Expected	.7	.3	
	Deviation	+.3	-.3	
	X^2	.1	.3	
2	Observed	19		19
	Expected	13.4	5.6	
	Deviation	+5.6	-5.6	
	X^2	2.3	5.6	
3	Observed	48	14	62
	Expected	43.7	18.3	
	Deviation	+4.3	-4.3	
	X^2	.4	1.0	
4	Observed	123	66	189
	Expected	133.3	55.8	
	Deviation	-10.3	+10.2	
	X^2	.8	1.9	
Totals		191	80.0	271

X^2 = 12.4**. Significant at the 1% level.

distributions for these infection types. A significant chi-square value indicated that the distribution of this part of the F_3 segregation was also dependent upon the F_2 infection types. The pooled oo types had proportionally more resistant types (1, 2, and 3) than lines selected as 0 types. The distribution suggested that the factors that modified the expression of the heterozygous major gene were also responsible for the increased resistance of that part of the population containing the homozygous recessive major genes.

Lemhi x P. I. 178383 at 15/24 The infection types of the parents, F_1, F_2, and Lemhi testcross distributions are presented in Table VI. As at 2/18, the F_1 plants were oo types.

Chi-square tests on the F_2 segregation again indicated that there was one major factor involved, and, as was the case at 2/18, there was a good fit to either a 3:1 ratio (types $\overset{y}{o}o$, oo, 0-, and 0 to types 1-, 1, 2, 3, and 4) or 1:2:1 ratio (type $\overset{y}{o}o$ to types oo, 0-, and 0 to types 1-, 1, 2, 3, and 4). The Lemhi testcross at this temperature profile again supported the presence of one major factor. The lack of $\overset{y}{o}o$ types indicated that this factor was partially dominant as it was at 2/18.

When F_2 plants with known infection types were selfed, the $\overset{y}{o}o$ types remained homozygous for P. I. 178383's resistance, the oo, 0-, and 0 types segregated into approximately 3:1 ratios, and the 1-, 1, 2, 3, and 4 types remained or segregated from 0 to 4 types (see Table II in the appendix).

To determine if certain infection types were the result of biological variation within a particular genotype or whether they were

Table VI. The infection types of the parents, P. I. 178383 and Lemhi, the F_1, the F_2, and the testcross distribution at 15/24 and the results of chi-square tests for goodness of fit.

Parents and progeny	Infection types and observed no. of plants									Total	Ratio*	X^2 value	P value
	vo	oo	o-	o	1-	1	2	3	4				
P. I. 178383	all												
Lemhi									all				
F_1		all											
F_2	73	113	23	10	1	2	9	13	45	289			
		219					70				3:1	.09	.75-.90
	73		146				70				1:2:1	.10	.90-.95
(Lemhi x P. I. 178383) x Lemhi		3	6	15				10	20	54			
		24					30				1:1	.67	.25-.50

* Ratios include same infection types as Table I.

conditioned by different genotypes, F_2 plants with specific infection types were selected and selfed to produce F_3 lines. The combined F_3 lines from plants which had the same F_2 infection type are presented in Table VII.

A chi-square test was subjected upon the F_3 distribution of the pooled oo and O F_3 lines. Both the pooled oo and O lines segregated for one gene in a 3:1 ratio. However, only the pooled O lines fit a 1:2:1 ratio. The lines selected as O types apparently lacked the modifying factors that caused the selected oo types to express more $o^v o$ types.

As was done for the pooled F_3 distributions at 2/18, the F_3 distributions for the pooled oo and O lines were compared to determine if it was possible to distinguish the oo type from the O type at this temperature profile (15/24) and to show that these infection types were conditioned by different genotypes. A chi-square test for independence was highly significant indicating that the segregation of the pooled F_3 lines was dependent upon the infection type at which they were selected (see Table VIII).

Proportionally, there were more $o^v o$ types in the pooled oo F_3 distribution than the pooled O F_3 distribution. At this temperature, just as at 2/18, there must be modifying factors that condition higher levels of resistance than are conditioned by the heterozygous major alone, and more of these favorable modifying factors must be present in the selected oo types than the selected O types of the F_2 segregation.

If P. I. 178383 had only a single gene, then the homozygous

Table VII. F_3 distributions of pooled $\overset{v}{oo}$, oo, 0 and 4 lines from Lemhi x P. I. 178383 and the goodness of fit of the pooled oo and 0 lines to ratios of 3:1 and 1:2:1 when tested at 15/24.

F_2 infection types at 2/18	Pooled F_3 infection types and observed no. of plants								Total	Ratio*	X^2 value	P value
	$\overset{v}{oo}$	oo	0-	0	1	2	3	4				
Pooled $\overset{v}{oo}$ lines	526	58	1						585			
Pooled oo lines	598	378	58	24	17	52	104	137	1368			
		1058				310				3:1	268.00	< .005
	598		460			310				1:2:1	3.99	.05
Pooled 0 lines	123	142	48	57	1	3	34	76	484			
		370				114				3:1	.44	.50-.75
	123		247			114				1:2:1	.53	.25-.50
Pooled 4 lines				2	10	57	225	442	736			

* Ratio includes same infection types as Table I.

Table VIII. Chi-square test for independence of F_2 infection types of Lemhi x P. I. 178383 using the pooled F_3 lines selected as oo and 0 types at 15/24 in the F_2.

	Distribution of pooled F_3 infection types at 15/24					Totals
	oo	oo	0-	0	1-4	
Pooled F_3 lines from oo F_2 plants	598 +	378 -	58 -	24 -	310 -	1386
Pooled F_3 lines from 0 F_2 plants	123 -	142 +	48 +	57 +	114 +	484
Totals	721	520	106	81	424	1852

$X^2 = 133.1^{**}$. Significant at the 1% level.

Table IX. Chi-square test for independence of F_2 infection types from Lehmi x P. I. 178383 comparing types 1, 2, 3, and 4 from the pooled oo and 0 F_3 lines grown at 15/24.

	Distribution of pooled F_3 infection types at 15/24				Totals
	1	2	3	4	
Pooled F_3 lines from oo F_2 plants	17 +	52 +	104 +	137 -	310
Pooled F_3 lines from 0 F_2 plants	1 -	3 -	34 -	76 +	114
Totals	18	55	138	213	424

$X^2 = 25.7^{**}$. Significant at the 1% level.

recessive condition should condition only those infection types that were as susceptible as Lemhi (type 4). Furthermore, the F_1 of Lemhi x P. I. 178383 (oo) should result in only three infection types when selfed, i.e., $\frac{o}{o}$, oo, and 4 types. However, in the F_2 distribution of of Lemhi x P. I. 178383 at 15/24 there were a number of more resistant types than Lemhi that were classified earlier in that portion of the ratios that contain the homozygous recessive major factor.

To determine if all 4 types were the same genotype, F_2 plants with this infection type were selected and selfed. The segregation of these F_3 lines are given in Table X. It can be seen that there were several patterns of segregation apparently caused by different combinations of minor factors.

Therefore, it is apparent that in addition to the major dominant gene, P. I. 178383 possesses a number of minor or modifying factors that can condition levels of resistance better than that of the very susceptible Lemhi parent. When certain F_2 plants with a 4 infection type were selfed, resistance as good as a 0 was observed. There must be several combinations of factors that can condition this infection type in this cross with at least one of the combinations containing the heterozygous major gene and one of them containing the homozygous recessive major gene with minor genes.

What are the relationships between these factors that condition the 1-, 1, 2, and 3 reactions and those that modify the expression of the heterozygous major gene? To investigate this relationship a comparison between the infection types that range from 1 through 4 was

Table X. Distribution patterns of F_3 lines of Lemhi x P. I. 178383 tested at 15/24 selected from F_2 seedlings with infection type 4.

Selection number	F_3 distribution at 15/24						Total
	0	1-	1	2	3	4	
42	2	5	9	11	42	14	83
105	2	4	7	13	21	6	53
113	3	1	4	8	10	20	46
174	5	1	7	9	13	4	39
310	1		1	10	17	30	59
314	1	1	1	12	14	31	60
35		1	2	5	41	37	86
34			1	1	20	65	87
271			1	11	10	54	76
299			3	15	21	16	55
306			3	5	28	44	80
43				3	54	29	86
103				4	14	29	47
115				6	5	45	56
265				1	15	18	34
270				1	12	44	57
308				2	43	12	57
32					8	60	68
173					10	34	44
274					33	47	80
276					17	42	59
277					6	53	59
281					9	51	60

made. It is noted in Table IX that the pooled oo families have a higher proportion of 1, 2, and 3 types than the pooled 0 families. A chi-square test for independence was used, and these pooled families were significantly different or there was a significant interaction between the genotypes of the plants of the two pooled families and P. striiformis which resulted in different proportions of infection types being produced. The families with the greatest level of resistance or incompatibility to the fungus were those selected as oo types. It would thus appear that the factors or part of the factors which modify the expression of the heterozygous major gene in response to P. striiformis at this temperature profile are the same as those that condition greater incompatibility than Lemhi's genotype.

Interaction of Lemhi x P. I. 178383 with the temperature profiles 2/18 and 15/24 When a comparison was made between the F_2 distributions of Lemhi x P. I. 178383 at both temperature profiles (see Table XI), it was observed that at 15/24 there were 1-, 1, and 2 types and a higher proportion of oo types than at 2/18. To determine if these differences were independent or dependent upon the temperature profile, a test of independence was made. The two F_2 distributions were highly significantly different at the two profiles. There was then an interaction between the temperature and the random genotypes present in the F_2 distributions.

Although this test does not state which types caused the significance, the magnitude of the individual cell chi-square values suggested that most of the significance was a result of more oo and fewer 0 types

at 15/24 than at 2/18. The lack of 1, 2, and 3 types at 2/18 also contributed to the significant value.

In order to determine if there was a difference in any part of the F_2 distribution, the 1-, 1, 2, 3, and 4 types were compared. Within this part of the distribution there was also a significant interaction between the genotypes and the temperature profile (see Table XII). Most of the difference was again in the larger proportion of 1, 2, and 3 types and smaller proportion of 4 types at 15/24 as compared to 2/18. These significant differences would indicate that besides the major gene in P. I. 178383 there are a number of temperature sensitive factors that modify the expression of the heterozygous major factor to condition increased resistance and that also condition greater levels of resistance in combination with the homozygous recessive major at 15/24 than at 2/18.

Are these temperature sensitive factors the same factors that were responsible for the differences in the expression of the heterozygous major gene at the same temperature profile? By comparing the pooled F_3 lines grown at 2/18 and 15/24 that were produced from the same F_2 plants with the same infection type, it should be possible to determine if there is a difference in the two distributions at the two temperature profiles. If there is no difference, then the factors that modify the heterozygous major gene to condition a oo type are probably the same factors that appear to be temperature sensitive in the absence of the dominant form of the major gene. If there is a significant difference within the pooled F_3 families grown at different profiles, then

Table XI. Chi-square test for interaction of F_2 distributions of the Lemhi x P. I. 178383 cross at the two temperature profiles.

	F_2 distributions and observed no. of plants[1]									Totals
	oo	oo	0-	0	1-	1	2	3	4	
At 2/18	30	39	13	20	0	0	0	1	35	138
	-	-	+	+	-	-	-	-	+	
At 15/24	73	113	23	10	1	2	9	13	45	289
	+	+	-	-	+	+	+	+	-	
Totals	103	152	36	30	1	2	9	14	80	427

$X^2 = 34.5^{**}$. Significant at the 1% level.

[1] The + or - sign indicates whether this infection type was proportionally larger or smaller than expected.

Table XII. Chi-square test of interaction between the infection types from 1- through 4 in the F_2 distribution of Lemhi x P. I. 178383 and the temperature profiles.

	F_2 distributions and observed no. of plants					Totals
	1-	1	2	3	4	
At 2/18	0	0	0	1	35	36
	-	-	-	-	+	
At 15/24	1	2	9	13	45	70
	+	+	+	+	-	

$X^2 = 14.2^{**}$. Significant at the 1% level.

these factors are probably not entirely in common with the factors that modify the heterozygote to condition either a oo or 0 infection type. A second possibility would be that the factors are mostly in common but vary in their ability to condition various levels of resistance at the two temperature profiles.

Tables XIII and XV show the comparisons between the pooled oo lines at 2/18 and 15/24 and between the pooled 0 lines at 2/18 and 15/24. For the oo lines there was a significant difference at the 5% level or an interaction of these genotypes and temperature. For the 0 lines there was a highly significant difference, or there was apparently greater interaction between the genotypes and the temperature. The 0 lines, therefore, tended to display greater temperature sensitivity than the oo lines, although this may have been simply due to the fact that the pooled oo lines had already attained a higher level of resistance and the temperature sensitive effect was masked to a certain extent. In fact, a reversal occurred for the oo lines with greater resistance at 2/18 than at 15/24.

By making similar comparisons for the part of the distributions that contained the homozygous recessive major gene, it was determined that temperature sensitivity was also expressed for this part of the distribution. For this part of the pooled oo lines there was a highly significant difference with proportionally more resistant types at 15/24 than at 2/18 (see Table XIV). For the pooled 0 lines there was no difference (see Table XVI). The interaction of genotypes and the temperature profile was then greater for the oo lines than for the 0 lines

Table XIII. Chi-square test of interaction between the pooled oo F_3 lines of Lemhi x P. I. 178383 and the temperature profiles.

	Pooled oo F_3 infection types and observed number of plants					Totals
	o%	oo	0-	0	1-4	
At 2/18	423 +	288 +	44 +	5 -	191 -	951
At 15/24	598 -	378 -	58 -	24 +	310 +	1368
Totals	1021	666	102	29	501	2319

X^2 = 10.2*. Significant at the 5% level.

Table XIV. Chi-square test of interaction between the infection types from 1 through 4 of the pooled oo F_3 lines of Lemhi x P. I. 178383 and the temperature profiles.

	Pooled oo F_3 infection types and observed number of plants				Totals
	1	2	3	4	
At 2/18	1 -	19 -	48 -	123 +	191
At 15/24	17 +	52 +	104 +	137 -	310
Totals	18	71	152	260	501

X^2 = 19.6**. Significant at the 1% level.

Table XV. Chi-square test of interaction between the pooled 0 F_3 lines of Lemhi x P. I. 178383 and the temperature profiles.

	Pooled 0 F_3 infection types and observed number of plants					Totals
	o͡o	oo	0-	0	1-4	
At 2/18	59 -	82 -	81 +	53 +	80 -	355
At 15/24	123 +	142 +	48 -	57 -	114 +	484
Totals	182	224	129	110	194	839

X^2 = 34.2**. Significant at the 1% level.

Table XVI. Chi-square test of interaction between the infection types from 1 through 4 of the pooled 0 F_3 lines of Lemhi x P. I. 178383 and the temperature profiles.

	Pooled 0 F_3 infection types and observed number of plants				Totals
	1	2	3	4	
At 2/18	0 -	0 -	14 -	66 +	80
At 15/24	1 +	3 +	34 +	76 -	114
Totals	1	3	48	142	194

X^2 = 7.2 Probability = .05-.07. Not significant.

Table XVII. Chi-square test of interaction between the pooled 4 F_3 lines of Lemhi x P. I. 178383 and the temperature profiles.

	Infection types of pooled 4 lines and observed number of plants					Totals
	0	1	2	3	4	
At 2/18	2	5	48	77	411	543
	+	-	+	-	+	
At 15/24	2	10	57	225	442	736
	-	+	-	+	-	
Totals	4	15	105	302	853	1279

$X^2 = 48.1^{**}$. Significant at the 1% level.

in this portion of the pooled F_3 distributions.

The results of these four comparisons suggested that the minor factors carried in the oo lines that modified the expression of the heterozygous major gene to condition greater resistance were probably the same as the temperature sensitive factors.

To further check for temperature sensitivity, F_2 plants that conditioned 4 infection types were selfed to produce F_3 seed. This seed was randomly divided, part of it grown at 2/18 and part at 15/24. The families grown at the same temperature profile were then pooled, and a comparison was made between the two distributions produced at the two profiles. A significant chi-square value indicated that the F_3 distributions were dependent upon temperature (see Table XVII).

II. Chinese 166 x Lemhi

Chinese 166 x Lemhi at 2/18 The infection types of the parents and F_2 distribution are given in Table XVIII. The F_2 distribution fits a one gene ratio of 3:1 with types $\overset{v}{o}o$, oo, 0-, 0 and 1 included in one class and types 1, 2, 3, and 4 in the other. At this temperature profile there was also a satisfactory fit to a 1:2:1 ratio with the $\overset{v}{o}o$ type comprising the first class in this ratio.

These data indicated that Chinese 166 had also one major factor for resistance to P. striiformis and that this major gene was also partially dominant. Thus, it is possible at this profile to distinguish the homozygous major from the heterozygous major factor.

Chinese 166 x Lemhi at 15/24 The infection types of the parents and F_2 distribution are presented in Table XIX. When the F_2 was

Table XVIII. The infection types of the parents, Chinese 166 and Lemhi, and the F_2 distribution at 2/18 and the results of chi-square tests for goodness of fit.

Parents and progeny	Infection types and observed no. of plants									Total	Ratio*	X^2 value	P value
	$\overset{v}{oo}$	oo	0-	0	1-	1	2	3	4				
Chinese 166	all												
Lemhi									all				
F_2	40	37	45	31	3			23	37	216			
			156					60			3:1	.89	.25-.50
	40		116					60			1:2:1	4.89	.05-.10

* The 3:1 ratio includes types $\overset{v}{oo}$, oo, 0-, 0, 1- to types 1, 2, 3, 4. The 1:2:1 ratio includes type $\overset{v}{oo}$ to types oo, 0-, 0, 1- to types 1, 2, 3, 4.

subjected to a chi-square test for one gene, there was a good fit to a 3:1 ratio where types $\overset{v}{o}o$, oo, and 0- were included in the first class of the ratio and 0, 1-, 1, 2, 3, and 4 types in the second class. There was a rather poor fit to a 1:2:1 ratio where the $\overset{v}{o}o$ type comprised the first class of the ratio. At this profile it was more difficult to distinguish the homozygous major factor pair from the heterozygous major factor.

At this temperature profile a second factor pair appeared to segregate that was hypostatic to the major dominant gene. When a 12:1:3 ratio was subjected to a chi-square test, there was a very good fit. This ratio included types $\overset{v}{o}o$, oo and 0- to types 0, 1-, 1, and 2 to types 3 and 4, respectively. This ratio suggested that there was a recessive factor in Chinese 166 that conditioned infection types 0, 1-, 1, and 2.

However, there was also a good fit to a 12:3:1 ratio where types $\overset{v}{o}o$, oo, and 0- made up the first class of the ratio, types 0, 1-, 1, 2, and 3 the second class, and type 4 the third. This ratio suggested that this second factor in Chinese 166 had dominant gene action and the dominant state conditioned types 0 through 3. A third possibility exists with partial dominance being present and the 3 type the result of the heterozygous condition of this factor pair (see Table XIX). Because F_2 plants with specific infection types were not saved to determine if these division in the F_2 segregation are correct, Chinese 166 will be considered to have two independent factor pair conditioning resistance.

Interaction of Chinese 166 x Lemhi with the temperature profiles 2/18 and 15/24 The results of the F_2 distributions at the two temperature profiles suggested that there was a temperature response to this cross. At 2/18 there appeared to be a single dominant gene and possibly several minor genes. However, at 15/24 there appeared, in addition to the dominant major gene, convincing evidence for a second favorable factor that conditioned intermediate infection types. At 15/24 the range of the expression of the heterozygous major gene appeared to be less (i.e., from oo to 0- as compared to oo, 0-, 0, and 1- at 2/18). This difference may have resulted from the direct influence of temperature or from the modification brought about by the second favorable factor which clearly appeared to be temperature or environmental sensitive.

When the two F_2 segregations were subjected to a chi-square test for independence of temperature, there was a highly significant value. The two F_2 distributions would then clearly be dependent upon the temperature profile at which they were tested. These results indicated a definite genotype by temperature interaction (see Table XX).

The individual cell chi-square values suggested that the greatest differences lay in three areas of the distributions: the proportion of 4 types, of 1 and 2 types, and oo, 0-, and 0 types. It appeared that at 15/24 the increased numbers of 1 and 2 types came at the expense of the 4 types, and the increased numbers of $\overset{v}{oo}$ and oo types came at the expense of the 0 and 0- types.

Table XIX. The infection types of the parents, Chinese 166 and Lemhi, and the F_2 distribution at 15/24 and the results of chi-square tests for goodness of fit.

Parents and progeny	Infection types and observed no. of plants									Total	Ratio*	X^2 value	P value
	v oo	oo	0-	0	1-	1	2	3	4				
Chinese 166	all												
Lemhi									all				
F_2	130	186	11	2	3	13	13	48	34	440			
		327				113					3:1	.11	.50-.75
	130	197				113					1:2:1	6.12	.04-.05
		327			31			82			12:1:3	.48	.75-.90
		327			79			34			12:3:1	1.71	.40-.50

* The ratios include the following infection type classes: the 3:1 includes types v oo, oo, 0- to types 0, 1-, 1, 2, 3, 4; the 1:2:1 includes type v oo to types oo, 0- to types 0, 1-, 1 2, 3, 4; the 12:1:3 includes types v oo, oo, 0- to types 0, 1-, 1, 2 to types 3, 4; the 12:3:1 includes types v oo, oo, 0- to types 0, 1-, 1, 2, 3 to type 4.

Table XX. Chi-square test of independence of F_2 distributions of the Chines 166 x Lemhi cross at the two temperature profiles.

	F_2 distribution and observed no. of plants									Totals
	$\overset{v}{oo}$	oo	0-	0	1-	1	2	3	4	
At 2/18	40	37	45	31	3	0	0	23	37	216
	-	-	+	+	+	-	-	-	+	
At 15/24	130	186	11	2	3	13	13	48	34	440
	+	+	-	-	-	+	+	+	-	
Totals	170	223	56	33	6	13	13	71	71	656

X^2 = 171.8**. Significant at the 1% level.

At 15/24 the temperature sensitive factor or factors not only increased incompatibility within the most susceptible types of the distribution, but have also caused the heterozygous major factor to produce mostly oo types and possibly even $\overset{v}{o}o$ types. The lack of a good fit to a 1:2:1 ratio at this temperature profile also supports this interpretation.

III. Chinese 166 x P. I. 178383

Chinese 166 x P. I. 178383 at 2/18 The infection types of the parents , F_1, and F_2 distributions are presented in Table XXI. The F_1 had the same infection type as the parents. The F_2 segregation very closely approached a 15:1 segregation for two dominant major genes and a very good fit was obtained from a chi-square test. Within the grouping conditioned by at least one dominant factor were infection types $\overset{v}{o}o$, oo, O-, and O, and the homozygous recessive grouping contained infection types 1-, 1, 2, 3, and 4.

From the F_2 distribution grown at this temperature profile plants with known infection types were selected and selfed to produce F_3 lines. These F_2 plants were selected randomly within any particular infection type, but the several infection types were not randomly selected and were not represented in the F_3 in the same ratio as in the F_2. The segregation of the selected F_3 lines are given in Table III of the appendix.

Of the 33 F_3 lines saved that were $\overset{v}{o}o$ in the F_2, 23 did not segregate for infection types that were more susceptible than the O type. Ten of the $\overset{v}{o}o$ types segregated for infection types more

Table XXI. The infection types of the parents, Chinese 166 and P. I. 178383, the F_1, and the F_2 distribution at 2/18 and the results of chi-square tests for goodness of fit.

Parents and progeny	Infection types and observed no. of plants									Total	Ratio*	X^2 value	P value
	o̊o	oo	0-	0	1-	1	2	3	4				
Chinese 166	all												
P. I. 178383	all												
F_1	all												
F_2	139	17	1	5			1	2	7	172			
		162					10				15:1	.06	.75-.90
	139		23				10				11:4:1	13.00	<.005

* The ratios include the following types: the 15:1 ratio includes types o̊o, oo, 0-, 0 to types 1-, 1, 2, 3, 4; the 11:4:1 ratio includes type o̊o to types oo, 0-, 0 to types 1-, 1, 2, 3, 4.

susceptible than the 0 type. At 2/18 these ten pooled lines had a good fit to a chi-square test for a 15:1 ratio (see Table XXII).

These data suggested that both Chinese 166 and P. I. 178383 have a major gene for stripe rust resistance and that these genes were independently inherited. Both genes appeared to condition a $\overset{v}{o}o$ infection type when homozygous dominant. Results of the F_1 and these 10 pooled F_3 lines indicated that the double heterozygous phase also conditioned a $\overset{v}{o}o$ infection type.

If the locus of the major gene from P. I. 178383 is assigned the letter A and that from Chinese 166 the letter B, then the F_1 would be AaBb. Table XXII gives the probable F_2 genotypes determined for the F_3 lines.

The 23 lines selected as $\overset{v}{o}o$ types that did not segregate probably had at least one pair of major factors homozygous dominant and, therefore, did not segregate to the more susceptible types. In Table XXII a dash indicates that the condition of the allele at this locus can not be determined. For example, for these 23 $\overset{v}{o}o$ lines the genotypes could be: AAbb, AABb, AABB, AaBB, or aaBB.

For the ten $\overset{v}{o}o$ lines that did segregate in a 15:1 ratio, the probable genotype is AaBb. This is the same genotype for these plants as would be present in the F_1, which was a $\overset{v}{o}o$ and which did segregate for a dihybrid ratio of 15:1.

Because the $\overset{v}{o}o$ types were selected randomly, and if the proposed genotypes for the F_2 plants with the $\overset{v}{o}o$ infection types are correct, there should be a ratio of seven lines that remained resistant to

Table XXII. The infection types of pooled F_3 lines of Chinese 166 x P. I. 178383 tested at 2/18 and the results of chi-square tests for goodness of fit.

F_2 infection types of pooled F_3 lines	No. of lines	Infection types and observed number in pooled F_3 lines									Total	Ratio*	X^2 value	P value	Probable F_2 genotype**
		ṽo	oo	0-	0	1-	1	2	3	4					
ṽo	23	437	16		2										AA-- or --BB
ṽo	10	138	38	10	3		1		4	3	197				AaBb
				189				8				15:1	1.60	.10-.25	
oo,0	13	91	85	15	8	5	8	7	22	18	259				Aabb or aaBb
				199				60				3:1	.46	.50	
2	1		1		1		1	3	7	6	20				aabb
3	2		1		3	3	5	2	17	9	40				aabb
4	5						2	4	34	59	99				aabb

* The 15:1 and 3:1 ratios include types ṽo, oo, 0-, 0 to types 1-, 1, 2, 3, 4.

** The dash indicates either allele at this locus could be present.

four lines that segregated for susceptibility. A chi-square test to check this ratio for the 33 F_3 families had a probability of .25-.50.

The pooled F_3 segregation for the 13 F_3 families selected as oo or 0 infection types are also presented in Table XXII. These pooled lines segregated for one resistant gene in a 3:1 ratio. A probability of .25-.50 was obtained when this ratio was tested with a chi-square test for goodness of fit. The probable genotype of these lines would be either Aabb or aaBb. Because the F_2 plants were selected at 2/18 and all of the oo and 0 lines segregated from $\overset{v}{o}o$ to 4 infection types, the F_3 data supported the previously assigned grouping of the F_2 distribution.

At 2/18 the following genotypes could then condition the assigned infection types:

$\overset{v}{o}o$		oo, 0-, or 0	1-, 1, 2, 3, or 4
AABB	AaBB	aaBb	aabb
AABb	aaBB	Aabb	
AAbb	AaBb		

The assignment of these infection types to these genotypes was not necessarily supported by the F_2 ratio at 2/18. The chi-square test for goodness of fit to an F_2 ratio of 11:4:1 had a probability of less than 0.005 (see Table XXI). However, the variations in infection types within any of these genotypes may be attributed to the previously demonstrated minor factors in Chinese 166 and P. I. 178383. For example, the lack of sufficient oo, 0-, and 0 types in the F_2 is probably due to the minor factors modifying the aaBb and Aabb genotypes to condition a $\overset{v}{o}o$ reaction. The differences obtained

with a specific genotype such as aabb was probably due to these minor genes (see the pooled 2, 3, and 4 lines in Table XXII).

Because the $\overset{v}{oo}$ types that segregated with a 15:1 segregation were supposedly like the F_1 for the major genes, these ten lines should have also segregated for a 11:4:1 ratio. When the F_3 distribution from these ten pooled lines was tested with a chi-square test there was a very good fit with a probability of .25-.50. These pooled F_3 lines then supported the assigned genotypes.

The segregation of the F_3 lines selected as 2, 3, and 4 infection types is given in Table XXII. It was observed that the F_2 plants that had the best resistance generally produced the F_3 lines with the best resistance. Since these F_3 lines have relatively low numbers and the second favorable factor in Chinese 166 and some of the minor factors in P. I. 178383 are temperature sensitive and not as strongly expressed at this temperature profile, the different possible genotypes for these lines were not determined.

Chinese 166 x P. I. 178383 at 15/24 The parents, F_1, and F_2 distributions are given in Table XXIII. The F_1 was as resistant as the parents with a $\overset{v}{oo}$ infection type. The F_2 distribution fit a chi-square test for a 15:1 segregation with a probability of .50-.75. This distribution was different from the F_2 distribution at 2/18, because infection type 0 was included with the susceptible group and there were no 0- types. The results of the F_3 lines discussed later supported this grouping.

Because the second favorable factor of Chinese 166 was opera-

Table XXIII. The infection types of the parents, Chinese 166 and P. I. 178383, the F_1, and the F_2 distribution at 15/24 and the results of chi-square tests for goodness of fit.

Parents and progeny	Infection types and observed no. of plants									Total	Ratio*	X^2 value	P value
	$\overset{v}{oo}$	oo	0-	0	1-	1	2	3	4				
Chinese 166	all												
P. I. 178383	all												
F_1	all												
F_2	128	19		3			3	1	1	155			
		147				8					15:1	.31	.50-.75
		147		3			5				60:1:3	.86	.50-.75
	128		19			8					11:4:1	14.67	<.005

* The 15:1 includes types $\overset{v}{oo}$, oo, 0- to types 0, 1-, 1, 2, 3, 4. The 60:1:3 includes types $\overset{v}{oo}$, oo, 0- to types 0, 1-, 1 to types 2, 3, 4. The 11:4:1 includes type $\overset{v}{oo}$ to types oo, 0- to types 0, 1-, 1, 2, 3, 4.

tive at this temperature profile, there should be a three gene segregation in this F_2. There was a good fit to a 60:1:3 ratio.

Fifty-four F_3 lines were also grown at this temperature profile and are presented in Table III of the appendix. Seed for these lines came from the same F_2 plants that were selected at 2/18 and were used to produce the F_3 lines grown at 2/18. Of the 33 plants that had a $\overset{v}{o}o$ infection type, again ten of them segregated for susceptibility to fit a ratio of 7:4. These ten pooled lines are presented in Table XXIV.

These ten pooled lines had a rather poor fit to a 15:1 ratio with a probability of .01-.02. As with the F_2 distribution at this temperature, the 0 types were included with more susceptible group, and this may have caused the poor fit. Part of the 0 types observed may have been due to a heterozygous major gene. Because 0 types from the F_2 grown at 15/24 were not selected to produce F_3 lines, it was not possible to be sure how many were due to a heterozygous major gene and how many were due to a favorable combination of minor genes.

The results of the F_3 distributions from the oo and 0 lines and the 2, 3, and 4 lines supported the placement of the majority of the 0 types in the more susceptible group (see Table XXIV). Of the thirteen F_3 lines that were selected as oo and 0 types and pooled, there was a good fit to a 3:1 ratio. The 0 types were placed in the susceptible fourth of the distribution. From these data it would appear that the genotypes for the major genes assigned to the

Table XXIV. The infection types of pooled F_3 lines of Chinese 166 x P.I. 178383 tested at 15/24 and the results of chi-square tests for goodness of fit.

F_2 inf. types	No. of lines	Inf. types and obs. no. in pooled F_3 lines								Tot.	Ratio*	X^2 value	P value	Prob. F_2 genotype**
		v oo	oo	0-	0	1-	1	2	3	4				
v oo	23	430	18		3						451			AA---- or --BB--
v oo	10	157	17	1	9		1	3	6		194			AaBb--
			175				19					15:1	4.16	.01-.02	
oo, 0	13	127	56	6	18	2	3	10	19	13	254			Aa---- or --Bb--
			189				65					3:1	.05	.80-.90	
Infection type of F_2	Selection number													
2	206				3	2	2	3	10		20	1:3	6.67	.01	aabbCc
3	230				12	2	3		3		20	-	-	-	aabbCC
3	234				3	2	2	3	9	1	20	1:3	6.67	.01	aabbCc
4	201				1		1	3	9	6	20	1:3	.00	1.00	aabbCc
4	212				6	1	1	7	2	3	20	1:3	26.67	<.005	aabbCc
4	250					1	2	12	3	2	20	1:3	26.67	<.005	aabbCc
4	252								2	18	20	-	-	-	aabbcc
4	238								10	10	20	-	-	-	aabbcc
2,3,4	8				25	8	11	28	48	40	160				
						72			88			3:5	3.84	.05	

* The 15:1 and 3:1 include types v oo, oo, 0- to types 0, 1-, 1, 2, 3, 4. The 3:5 and 1:3 include types 0-, 1-, 1, 2 to types 3, 4.

** The dash indicates that either allele at this locus could be present. The capital C represents the allele that conditions resistance regardless of gene action.

infection types at 2/18 are the same except that the aabb group probably includes those plants classified as 0 infection type.

The F_2 segregation does not closely fit a 11:4:1 ratio. The ten pooled $\overset{v}{oo}$ F_3 lines also did not support the assigned genotypes as they did at 2/18. These discrepancies are again believed to be due to the temperature sensitive modifying factors.

The segregation of each F_3 line that was selected in the F_2 as a 2, 3, or 4 type is presented in Table XXIV. The lines that were selected for the most resistance generally showed the most resistant F_3 segregation. Because these types were selected randomly without particular knowledge of the second favorable factor in Chinese 166, this factor should segregate in a random manner in these lines. If this is the case, there should have been a 3:5 segregation in the pooled F_3 lines. Of the 160 plants observed, 72 had infection types 0, 1-, 1, and 2 while 88 plants had infection types 3 and 4. The chi-square test for goodness of fit for these types to the 3:5 ratio had a probability of .05. The larger number of 0, 1-, 1, and 2 types than expected may be due to the temperature sensitive factors from P. I. 178383 conditioning some of these types and thus reducing the number of 3 and 4 types.

If the second locus in Chinese 166 is assigned the letter C, then the F_3 lines selected as 2, 3, and 4 types can be assigned genotypes for the F_2 plant from which they were derived. Because of the unknown influence of minor factors from P. I. 178383, these assigned genotypes may not necessarily fit a chi-square test (see

Table XXIV).

Interaction of Chinese 166 x P. I. 178383 with the temperature profiles 2/18 and 15/24 To determine if the two F_2 distributions of this cross were temperature sensitive and dependent upon the temperature profile at which they were evaluated, a chi-square test of interaction was used (see Table XXV). The chi-square value of 7.1 with 6 degrees of freedom had a probability of greater than 0.25 indicating that these two distributions were not dependent upon the temperature profile, despite the fact that similar tests showed that both Lemhi x P. I. 178383 and Chinese 166 x Lemhi were dependent upon the temperature profile and that both resistant parents have temperature sensitive factors that express themselves at 15/24.

Since only one-sixteenth of the sample shows the greatest amount of temperature sensitivity, the sample size may have been too small to detect any real differences. The 0 infection types were also shown to be conditioned by different genotypes, but in this comparison they were directly compared.

When only the infection types conditioned by the double homozygous recessive condition of the major genes were considered, there was a significant difference at the 5% level (see Table XXVI). The 0 types were included at 15/24, but not a 2/18.

As a further check on the extent of temperature sensitive factors and the genotype by temperature interaction, comparisons were also made between the ten pooled $\frac{v}{oo}$ lines that segregated 15:1,

Table XXV. Chi-square test of interaction between the F_2 distributions of Chinese 166 x P. I. 178383 and the temperature profiles.

	F_2 distributions and observed no. of plants									Totals
	oo	oo	0-	0	1-	1	2	3	4	
At 2/18	139	17	1	5	0	0	1	2	7	172
	-	-	+	+			-	+	+	
At 15/24	128	19	0	3	0	0	3	1	1	155
	+	+	-	-			+	-	-	
Totals	267	36	1	8			4	3	8	327

X^2 = 7.1. Probability = .25-.30. Not significant.

Table XXVI. Chi-square test of interaction between the infection types not conditioned by a dominant major gene for the Chinese 166 x P. I. 178383 cross and the temperature profiles.

	F_2 distributions and observed no. of plants				Totals
	0	2	3	4	
At 2/18	0	1	2	7	10
	-	-	+	+	
At 15/24	3	3	1	1	8
	+	+	-	-	
Totals	3	4	3	8	18

X^2 = 9.0*. Probability = .02-.04. Significant at the 5% level.

between the pooled oo and 0 lines, and between the pooled 2, 3, and 4 lines. Any differences observed in these comparisons were probably due to the temperature profile interacting with the certain genotypes, because the same F_2 plants were used to produce the F_3 plants checked at the two temperature profiles.

With all three comparisons there was a significant interaction (see Table XXVII). As was observed with both the Lemhi x P. I. 178383 and Chinese 166 x Lemhi crosses, at 15/24 there were more o$\breve{o}$ types than at 2/18 and fewer oo and 0- types. There were also more 0, 1-, 1, and 2 types at 15/24 than at 2/18 and fewer 3 and 4 types. With this cross, then, there was also a temperature by genotype interaction that caused at least some of the genotypes to condition higher levels of resistance at the higher temperature profile, 15/24.

IV. Interaction of factors from P. I. 178383 and Chinese 166

In order to obtain additional information on the minor genes of P. I. 178383 and Chinese 166, the F_2 segregations of these parents were compared. Three separate comparisons were made at each temperature profile. The comparisons involved these parents crossed with susceptible Lemhi and with each other.

From these comparisons it was hoped to obtain information on the commonness of the minor factors and to observe the type of gene action conditioned by these minor factors when crossed together into a common F_2 distribution. The emphasis was placed on that portion of the F_2 distributions conditioned by the homozygous recessive

Table XXVII. Chi-sqaure test of interaction between the pooled F_3 lines with infection types $\overset{v}{oo}$, oo and 0, and 2, 3, and 4 at the two temperature profiles, 2/18 and 15/24, for the Chinese 166 x P. I. 178383 cross.

	Infection type distribution of pooled F_3 lines									Totals
	$\overset{v}{oo}$	oo	0-	0	1-	1	2	3	4	
1. Pooled $\overset{v}{oo}$ lines that segregated 15:1. $X^2 = 26.1^{**}$										
At 2/18	138	38	10	3	0	1	0	4	1	197
	-	+	+	-			-	-	+	
At 15/24	157	17	1	9	0	1	3	6	0	194
	+	-	-	+			+	+	-	
Totals	295	55	11	12	0	2	3	10	3	391
2. Pooled oo and 0 lines. $X^2 = 24.5^{**}$										
At 2/18	91	85	15	8	5	8	7	22	18	259
	-	+	+	-	+	+	-	+	+	
At 15/24	127	56	6	18	2	3	10	19	13	254
	+	-	-	+	-	-	+	-	-	
Totals	218	141	21	26	7	11	17	41	31	513
3. Pooled 2, 3, and 4 lines. $X^2 = 40.8^{**}$.										
At 2/18		2		4	3	8	9	58	74	158
		+		-	-	-	-	+	+	
At 15/24				25	8	11	28	48	40	160
				+	+	+	+	-	-	
Totals		2		29	11	19	37	106	114	318

** Significant at the 1% level.

major gene.

Interaction at 2/18 When the F_2 of Lemhi x P. I. 178383 was compared with the F_2 of Chinese 166 x Lemhi at 2/18, there was a highly significant difference. When only infection types 1, 2, 3, and 4 were compared there was still a highly significant difference (see Table XXVIII). This indicated that the two resistant parents had different minor factors. The greatest difference in the distributions occurred in the larger proportion of 3 types for the F_2 of Chinese 166 x Lemhi.

When types 1, 2, 3, and 4 of the F_2 of Lemhi x P. I. 178383 and the F_2 of Chinese 166 x P. I. 178383 were compared, there was a significant difference at the 5% level. The F_2 of Chinese 166 x P. I. 178383 had a higher proportion of more resistant types.

When the third comparison was made with the 1, 2, 3, and 4 types of the F_2 distributions of Chinese 166 x Lemhi and Chinese 166 x P. I. 178383, there was a significant difference at the 5% level. To determine if the difference was due to the greater number of 2 or 4 types in the Chinese 166 x P. I. 178383 cross, only types 2 and 3 were compared. The significance was found to lie in the greater proportion of 2 than 3 types.

The results of these three comparisons indicated that the F_2 of Chinese 166 x P. I. 178383 had better resistance than either of the resistant parents crossed with Lemhi. The first comparison indicated that the minor factors of P. I. 178383 and Chinese 166 were not completely common, and the next two comparisons with Chinese 166 x P. I.

Table XXVIII. Chi-square test of independence to compare the F_2 infection type patterns for types 1, 2, 3, and 4 produced at 2/18 by Chinese 166 x Lemhi, Lemhi x P. I. 178383, and Chinese 166 x P. I. 178383.

	Distribution of infection types 1	2	3	4	Totals
1. Lemhi x P. I. 178383 vs Chinese 166 x Lemhi. X^2 = 15.2**					
Lemhi x P. I. 178383			1 -	35 +	36
Chinese 166 x Lemhi			23 +	37 -	60
Totals			24	72	96
2. Lemhi x P. I. 178383 vs Chinese 166 x P. I. 178383. X^2=8.0* P=.01-.02					
Lemhi x P. I. 178383		0 -	1 -	35 +	36
Chinese 166 x P. I. 178383		1 +	2 +	7 -	10
Totals		1	3	42	46
3. Chinese 166 x Lemhi vs Chinese 166 x P. I. 178383. X^2=7.1* P=.02-.03					
Chinese 166 x Lemhi		0 -	23 +	37 -	60
Chinese 166 x P. I. 178383		1 +	2 -	7 +	10
Totals		1	25	44	70

* Significant at the 5% level.

** Significant at the 1% level.

178383 suggested that a combination of minor factors from these resistant parents conditioned better resistance than was obtained in the F_2 of either resistant parent times Lemhi.

From these results it could be stated that the minor factors have additive gene action. An example of this action was observed in the F_3 lines of Chinese 166 x P. I. 178383 in which oo types were conditioned without the influence of a major gene (see line 206 and line 230 in appendix Table III).

Interaction at 15/24 When the F_2 distributions of Lemhi x P. I. 178383 and Chinese 166 x Lemhi were compared at 15/24, the two distributions were again found to be highly significant indicating that the minor factors in these two parents were not completely common. (For the comparisons at this temperature profile, the 1-, 1, 2, 3, and 4 types will be used for the Lemhi x P. I. 178383 cross, and the 0, 1-, 1, 2, 3, and 4 types will be used for the Chinese 166 x Lemhi and Chinese 166 x P. I. 178383 crosses (see Table XXIX).)

The comparison between F_2 distributions of Lemhi x P. I. 178383 and Chinese 166 x P. I. 178383 was highly significant as was the comparison between F_2 distributions of Chinese 166 x Lemhi and Chinese 166 x P. I. 178383. In both of these comparisons there was a larger proportion of 0 types in the Chinese 166 x P. I. 178383 cross than in the crosses between the resistant parents and Lemhi.

The results of these three comparisons were nearly identical to those at 2/18. Again, the first comparison showed that the F_2 distributions of either resistant parent crossed with Lemhi were not the

Table XXIX. Chi-square test of independence to compare the F_2 infection type patterns conditioned without the influence of a dominant major gene produced at 15/24 by Chinese 166 x Lemhi, Lemhi x P. I. 178383, and Chinese 166 x P. I. 178383.

	Distribution of infection types						Totals
	0	1-	1	2	3	4	
1. Lemhi x P. I. 178383 vs Chinese 166 x Lemhi. $X^2 = 24.7^{**}$							
Lemhi x P. I. 178383	0 (-)	1 (-)	2 (-)	9	13 (-)	45 (+)	70
Chinese 166 x Lemhi	2 (+)	3 (+)	13 (+)	13	48 (+)	34 (-)	113
Totals	2	4	15	22	61	79	183
2. Lemhi x P. I. 178383 vs Chinese 166 x P. I. 178383. $X^2 = 37.6^{**}$							
Lemhi x P. I. 178383	0 (-)	1 (+)	2 (+)	9 (-)	13 (+)	45 (+)	70
Chinese 166 x P. I. 178383	3 (+)	0 (-)	0 (-)	3 (+)	1 (-)	1 (-)	8
Totals	3	1	2	12	14	46	78
3. Chinese 166 x Lemhi vs Chinese 166 x P. I. 178383. $X^2 = 31.7^{**}$							
Chinese 166 x Lemhi	2 (-)	3 (+)	13 (+)	13 (-)	48 (+)	34 (+)	113
Chinese 166 x P. I. 178383	3 (+)	0 (-)	0 (-)	3 (+)	1 (-)	1 (-)	8
Totals	5	3	13	16	49	35	121

** Significant at the 1% level.

same and that different minor factors were present in the two parents.

The next two comparisons indicated that the combination of minor factors in the F_2 of Chinese 166 x P. I. 178383 conditioned a higher number of resistant types than either parent alone in crosses with Lemhi. This is further evidence of interaction of additive gene action between the minor factors from both parents to condition a more resistant F_2 distribution. Although these results suggested that some of the minor factors were different, they did not indicate how many factors were involved in P. I. 178383 or Chinese 166.

V. Rego x Lemhi

Rego x Lemhi at 15/24 The infection types of the parental, F_1, F_2, and testcross distributions are given in Table XXX. The Rego check plants demonstrated a slight variability in infection types from type 3- to type 3. The Lemhi plants were uniformly type 4. The thirteen F_1 plants indicated that Rego's reaction at this temperature profile was conditioned by dominant gene action.

Of the 153 seedling plants checked in the F_2, two were read to be more resistant than Rego, ninety-two had infection types equal to Rego, and fifty-nine had infection types that were more susceptible than Rego. Of these fifty-nine plants, fifty-one were as susceptible as Lemhi while eight appeared to be slightly more incompatible than Lemhi and were designated as 4- types. The 3 types were also divided into a 3- type. However, due to the difficulty of accurately reading these differences, the 4- and 4 types were considered a single class, as were the 3- and 3 types.

Table XXX. The infection types of the parents, Rego and Lemhi, the F_1, the F_2, and the Rego and Lemhi testcross distribution at 15/24 and the results of chi-square tests for goodness of fit.

Parents and progeny	Infection types and observed no. of plants									Total	Ratio*	X^2 value	P value
	0		1-	1	2	3-	3	4-	4				
Rego					1	20	16			37			
Lemhi									all				
F_1						3	10			13			
F_2					2	4	88	8	51	153			
				94					59		9:7	1.67	.10-.25
				94					59		3:1	15.01	<.005
Rego^2 x Lemhi						1	32	8	3	44			
				33					11		1:0	>10.00	<.005
Rego x Lemhi^2							8	7	25	40			
					8				32		1:3	.53	.25-.50
					8				32		1:1	144.00	<.005

* All of the ratios included types 0, 1-, 1, 2, 3-, 3 to types 4-, 4.

The F_2 distribution fits a two gene segregation of 9:7 for Rego types to Lemhi types with a probability of .10-.25. This ratio suggested that Rego has two dominant factors for resistance and that both factors are necessary in the dominant condition for F_2 plants to have the resistance of Rego.

The F_2 results were substantiated by the Lemhi testcross. This testcross should segregate for 1 Rego type to 3 Lemhi types. For this testcross there were 8 Rego types and 32 plants with infection types more susceptible than Rego to satisfactorily fit a ratio of 1:3 with a probability of .25-.50.

A discrepancy to this proposed system occurred with the Rego testcross. In the Rego testcross there should not have been a segregation for types 4- and 4. However, of the forty-four plants observed, eleven plants were more susceptible than Rego. A testcross ratio of 3:1 is difficult to explain when Rego's level of resistance appeared to be conditioned by dominant gene action.

Rego x Lemhi at 2/18 The infection types of the parental, F_1, F_2, and testcross distributions are presented in Table XXXI. Rego showed a temperature sensitive response that was just the opposite of the minor factors of P. I. 178383 and Chinese 166. At this temperature profile the majority of the plants had a 0 infection type. The F_1, however, did not demonstrate this temperature response, and all the F_1 plants were type 3-, or very similar to the F_1 response at 15/24. The F_1 plants suggested that the 3 infection type conditioned at both temperature profiles was conditioned by dominance and that the temperature

Table XXXI. The infection types of the parents, Rego and Lemhi, the F_1, F_2, and the Rego and Lemhi testcross distributions at 2/18, and the results of chi-square tests for goodness of fit.

Parents and progeny	Infection types and observed no. of plants								Total	Ratio*	X^2 value	P value
	0	1-	1	2	3-	3	4-	4				
Rego	28	7	7	5					47			
Lemhi								all				
F_1					all							
F_2	8	10	35	87	51	120	18	121	450			
			311				139			9:7	30.24	<.005
			311				139			3:1	8.32	<.005
Rego^2 x Lemhi	3	8	8	37	23	31	1		111			
			110				1			1:0	-	-
Rego x Lemhi^2						11	6	15	32			
			11				21			1:3	1.50	.20-.25
			11				21			1:1	3.13	.05-.10

* All of the ratios include types 0, 1-, 1, 2, 3 to types 4-, 4.

response of the Rego parent at 2/18 was due to recessive factors, and/or the influence of lower temperatures directly upon the dominant factors to condition great incompatibility.

The results with the F_2 did not directly indicate the presence of two dominant genes as it did at 15/24. The probability of a 9:7 ratio was less than 0.005 for a chi-square test when 0, 1-, 1, 2, and 3 types were placed in one class and the 4 types in the other. However, neither did the F_2 fit a 3:1 ratio. There were too many 4 types to fit a one gene segregation and too few to fit a 9:7 ratio for a two gene segregation. Since the ratio falls between a one and two gene segregation there may be a two gene segregation for dominant factors with temperature sensitivity causing some plants that would have been 4 types at 15/24 to be more resistant and thus fall into infection types of higher resistance. This hypothesis was supported by F_3 data in which plants selected as 4 types in the F_2 segregated to 2 and 3 types, apparently as a result of the recessive temperature sensitive factors (see Appendix Table IV).

The Rego testcross segregated from infection types 0 through 3 (one plant out of 111 was a 4-). This testcross indicated that dominant gene action occurred to condition at least the 3 type level of resistance.

Of the thirty-two plants observed in the Lemhi testcross, eleven were 3 types and twenty-one were 4 types. This segregation fit the expected 1:3 ratio for two dominant factors.

Interaction of Rego x Lemhi with the temperature profiles of 2/18 and 15/24 The F_2 distributions of Rego x Lemhi were compared using a chi-square test of interaction. Assuming that the two F_2 distributions contained a random assortment of the possible genotypes, the effect of temperature could be observed by determining which infection types contributed to the significant chi-square value. The comparison between these two F_2's is presented in Table XXXII.

The overall picture presented by these results indicates that part of the genotypes producing the 4, and particularly the 3 types, at 15/24 were responsive to a lower temperature profile to condition greater resistance. However, this comparison did not show the relationship between a particular type at 15/24 and what it would have been at 2/18. Ideally, to determine what infection type each plant would have produced at the two temperature profiles, the same plant would be tested at both. Because the primary leaves of seedling plants were used, this was not done.

By examining the F_3 distributions produced from selected F_2 plants, additional information on the relationship of the infection types produced at the two temperature profiles was obtained. F_2 plants were selected at 2/18 and selfed to produce F_3 seed. The seeds from each F_2 plant were randomly split and F_3 lines were grown at both 2/18 and 15/24. The infection types of the selected F_2 plants and their F_3 distributions are given in Table IV of the Appendix. Table XXXIII gives the selected F_3 lines that illustrated the various types of segregation observed at both temperature profiles.

Table XXXII. Chi-square test for interaction of the F_2 distribution of the Rego x Lemhi cross at the two temperature profiles.

	F_2 infection types and observed number of plants						Total
	0	1-	1	2	3	4	
At 15/24	0	0	0	2	92	59	153
	-	-	-	-	+	+	
At 2/18	8	10	35	87	171	139	450
	+	+	+	+	-	-	
Total	8	10	35	89	263	198	603

$X^2 = 54.2^{**}$ Significant at the 1% level.

Table XXXIII. Selected F_3 lines that illustrated the various types of distributions that occurred with the Rego x Lemhi cross at 2/18 and 15/24* and the comparison between these distributions at the two profiles.

Selection number	F_2 infection type at 2/18	F_3 infection types and observed number of plants								Total
		0	1-	1	2	3-	3	4-	4	
Rego	-	28	7	7	5					47
					1	20	16			37
317	1-	44	11	3						58
					5	11	4			20
328	2	11	10	1						22
					1	10	9			20
341	1	11	9	10	5	1				36
						6	11	1		18
355	2+	1	1	7	20	3	1			33
						3	14	2		19
351	3	2	1	4	7	9	7	1	7	37
						2	14	3	2	21
316	4					1	4	23	27	55
									20	20
343	4							7	27	34
								4	16	20

* The distribution tested at 2/18 is listed first.

Of the 56 F_3 lines checked, only selection 317 and 328 were not statistically different from the Rego check at both temperature profiles. Selection 317 was selected as a 1- and 328 as a 2. Although the plants used to produce the F_3 were not randomly selected, it was possible to use these two plants to estimate the number of factors involved in conditioning Rego's resistance at 2/18. From the particular F_2 distribution from which these plants were selected, all plants with infection types 1-, 1, and 2 were saved. (There were no 0 types.) Selecting all the most resistant plants then assured that any plant with Rego's genotype would be saved. In this particular F_2 distribution there were 173 plants. This would give an approximate ratio of one plant with Rego's genotype to eighty-six plants with other genotypes, or suggested that at least three factor pairs were involved.

There were several selections which were quite similar to Rego at both 2/18 and 15/24 but which were distributed over a wider range than Rego, particularly at 2/18; e.g., selection numbers 341 and 362 which were selected as 1 types and 320 and 344 which were selected as 2 types. These lines suggested that one of the temperature sensitive factors was segregating.

Selection 335 demonstrated segregation from 0 to 3 at 2/18 and was similar to Rego a 15/24, but the distribution centered around the 2 type which made it different from the above examples. Lines of this type were usually selected as 2 types in the F_2 and were probably heterozygous for several factors involved in Rego's temperature

response, because there were very few 4 types produced at either temperature.

Part of the lines selected as 2 types and most of the lines selected as 3 types were distributed from 0 to 4 at 2/18 and 3- to 4 at 15/24, e.g., selection 351 in Table XXXIII. These F_2 plants segregated for at least one of the factors that were necessary to condition a 3 infection type at 2/18 or 15/24.

A variety of F_3 distributions occurred in which there were no 0 types and few 1- or 1 types, but the number of 4 types was greater than in the previous example. Most of these distributions occurred from plants selected as 3 or 4 types. These plants probably had both temperature sensitive and dominant factors segregating.

There were several plants selected as 4 types that did not segregate in the F_3 at 15/24 but did segregate to more resistant types at 2/18, e.g., number 316. These plants were apparently homozygous recessive for the dominant factors but still segregated for the temperature sensitive factors. At 15/24 these factors had little influence, but at 2/18 they caused some of the plants to be 2 and 3 types.

The last type of F_3 lines produced were those that remained 4 types at both 2/18 and 15/24. All of these lines were grown from plants selected as 4 types in the F_2. However, these lines demonstrated more incompatibility than Lemhi to the race of P. striiformis used and were partially read as 4- types. There were no F_2 plants selected which passed the compatibility of the very susceptible Lemhi to all of the plants checked in the F_3 distribution.

From these selected lines it was observed that several genotypes can condition the various infection types, or that there was an overlap in the expression of a particular genotype. For example, some of the plants that were selected as either 2 or 3 types segregated the same. These lines also indicated that the factors that conditioned the 3 infection type at 15/24 were not necessarily the same as those that conditioned the temperature response. The lines that segregated for the 3 type but not for temperature sensitivity and, conversely, the types that segregated for temperature sensitivity but not the 3 type would support this interpretation.

Since more than one infection type can be conditioned by a single genotype, the F_3 lines that were derived from F_2 plants with the same infection types were compared using mean infection types to determine the reliability of using infection types as a measure of the genotype in this type of gene system. The mean F_3 values were also used to suggest the relationship between the same genotypes and the levels of resistance at the two profiles. Tables XXXIV and XXXV give the analysis of variance for the four infection type classes used. The F_3 lines derived from 1- and 1 types were placed together in a single class as were the 3- and 3 types. The highly significant F-value at both temperature profiles indicated that the F_3 mean values were significantly different and were dependent upon the infection type of the F_2.

Duncan's New Multiple-range Test was used to determine which of the means were significantly different from each other. The F_3 mean

Table XXXIV. Analysis of variance for the mean infection types of the F_3 lines of Rego x Lemhi at 2/18 on the basis of their F_2 infection types.

Source of variation	Degrees of freedom	Mean square	F value
Infection type	3	15.07	44.3**
Error	52	.34	

** Significant at the 1% level.

Table XXXV. Analysis of variance for the mean infection types of the F_3 lines of Rego x Lemhi at 15/24 on the basis of their F_2 infection types.

Source of variation	Degrees of freedom	Mean square	F value
Infection type	3	1.81	36.2**
Error	52	.05	

** Significant at the 1% level.

Table XXXVI. Mean F_3 infection types for the F_3 lines derived from F_2 plants with the same infection types.

F_2 infection types	1	2	3	4
F_3 infection type means at 2/18	.82	1.81	2.58	3.58
F_3 infection type means at 15/24	2.88	3.34	3.54	3.89

Means underlined by the same line are not significantly different at the 1% level.

infection types are listed in Table XXXVI. At 2/18 all means were different at the 1% level. At 15/24 all means were different at the 1% level except for the means derived from the 2 and 3 lines.

When the means at both temperature profiles were ranked, they fell in the same order as the infection types. At 2/18 this suggested that the F_2 infection types were quite reliable in determining the genotypes which conditioned the various levels of resistance, expecially since the mean values of the F_3 lines were nearly the same as the F_2 infection types.

Since the F_2 plants were selected at 2/18, the relationship between the mean F_3 values and the F_2 values at 15/24 was not determined. These data would suggest, however, that an infection type of 0 to 1 at 2/18 would be a 3- at 15/24, that infection types of 2 and 3 at 2/18 would be a 3 to 3+ at 15/24, and that a 4 at 2/18 would also be a 4 at 15/24.

The means of the 1 type at 15/24 and 2/18 were statistically more resistant than the other means. These means suggested that the high level of resistance conferred by these lines was effective at both temperature profiles. The F_2 plants that contributed to these lines were apparently more homozygous for factors that condition resistance and possessed most of the factors found in the Rego parent. They suggested that both the dominant and temperature sensitive factors were required to condition the Rego level of resistance at both temperature profiles.

VI. Rego x P. I. 178383

Rego x P. I. 178383 at 15/24 The infection types of the parental, F_1, F_2, and testcross distributions are presented in Table XXXVII. Again, the F_1 seedlings from this cross lacked the very high level of resistance of P. I. 178383 and were recorded as oo types.

Of the 175 plants checked, fifty-two were $\overset{v}{o}o$, seventy-two were oo or O-, and fifty-one were O through 4 types. This segregation had a poor fit to a 1:2:1 ratio. When the $\overset{v}{o}o$, oo, and O- types were combined, there was a satisfactory fit to a 3:1 ratio. When the Rego testcross was subjected to a chi-square test for a one gene segregation of 1:1, there was a very good fit. A second testcross of (Rego x P. I. 178383) x Lemhi also was made. It had a good fit for a 1:1 ratio.

From the F_2 and testcrosses grown at this temperature profile, specific plants were selected on the basis of their infection type. These plants and the F3 distributions they produced at the two profiles are listed in Table V of the Appendix. Because of space limitations, only a small sample of plants were grown to produce F_3 seed.

From the F_2 at 15/24 there were twelve plants selected of which there were four $\overset{v}{o}o$, two oo, one O-, one O, one 2, and three 3 types. Three of the four $\overset{v}{o}o$ types segregated, and all three satisfactorily fit a 3:1 ratio, but none fit a 1:2:1 ratio. Very few of the F_3 plants were of the 4 type, verifying that there were other factors for resistance present. This indicated that with the help of minor genes, some plants with a heterozygous major gene were as resistant

Table XXXVII. The infection types of the parents, P. I. 178383 and Rego, the F_1, F_2, and the testcross distributions at 15/24 and the results of chi-square tests for goodness of fit.

Parents and progeny	Infection types and observed no. of plants									Total	Ratio*	X^2 value	P value
	$\overset{v}{oo}$	oo	0-	0	1-	1	2	3	4				
P. I. 178383	all												
Rego							1	36		37			
F_1		all											
F_2	52	62	10	1	1	2	14	24	9	175			
		124					51				3:1	1.60	.10-.25
	52	72					51				1:2:1	5.55	.05-.10
$Rego^2$ x P. I. 178383		15	5	2			1	13	4	40			
		22					18				1:1	.40	.50-.75
(Rego x P. I. 178383) x Lemhi		12	13	5			1	20	11	62			
		30					32				1:1	.06	.75-.90

* The 3:1 ratio includes types $\overset{v}{oo}$, oo, 0- to 0, 1-, 2, 3, 4. The 1:2:1 ratio includes type $\overset{v}{oo}$ to types oo, 0- to types 0, 1-, 1, 2, 3, and 4. The 1:1 ratios include types oo, 0-, 0 to types 1-, 1, 2, 3, 4.

as plants with a homozygous dominant major gene.

The plants selected as oo and 0- types produced F_3 plants that segregated for the major gene, and two of the three lines fit both 3:1 and 1:2:1 segregations. The F_2 plants that were oo and 0- types then lacked the modifying help from certain minor factors that the three $\overset{v}{oo}$ lines possessed.

The plant selected as a 0 type did not segregate for $\overset{v}{oo}$, oo, or 0- types. This was the only 0 type in this F_2 distribution. It suggested that the 0 type was conditioned by minor factors and not the heterozygous major gene as in the Lemhi x P. I. 178383 cross. The plants selected as 2 and 3 types produced F_3 plants that were mostly centered around the 2 and 3 infection types.

Of the nine plants selected from the Rego testcross, two were 0 types. These were the only two 0 types in this testcross. Both of these plants yielded lines which segregated for the heterozygous major gene. In this testcross the 0 types were included with the other plants that were suspected of being conditioned by the heterozygous major gene. The fact that the 0 types from the F_2 and Rego testcross did not segregate in the same manner demonstrated that several different genotypes can condition this reaction. The lines produced from F_2 plants selected as 3 and 4 types would support this, because these lines segregated for resistance as well as a 0 infection type. The lines that were selected for greater resistance than a 0 segregated for a heterozygous major factor, and those more susceptible than a 0 segregated for only the minor factors.

From the Lemhi testcross, seven plants were selected and selfed. The 0 types produced F_3 lines that segregated for the heterozygous major gene. The most noticeable observations made on these testcross lines, as compared to the F_2 or Rego testcross lines, were that the majority of the susceptible types occurred as 4 reactions, and there was a higher proportion of 0- and 0 types. The extra dose of Lemhi had a very definite effect upon the number of minor factors from both parents, and this resulted in more susceptible plants being produced with and without the heterozygous major factor.

Rego x P. I. 178383 at 2/18 The infection types of the parental, F_1, F_2, and testcross distributions are presented in Table XXXVIII. The F_1 plants were read as oo- types. Of the 160 F_2 plants scored for infection types, 69 were o$\overset{v}{o}$, 53 were oo or 0-, and 38 were 0 through 4 types. This F_2 segregation does not fit a 1:2:1 segregation but has a good fit to a 3:1 segregation. At this temperature profile, then, it was impossible to clearly distinguish the heterozygous major gene from the homozygous dominant major gene for this cross.

The Rego testcross distribution also fit a 1:1 ratio when the distribution was divided between the 0- and 0 types. The Lemhi testcross distribution also fit a 1:1 ratio. It may be observed in Table XXXVIII that the distributions of these two testcrosses were quite different, even though both did fit the same ratio. With the Rego testcross there were mostly 0, 1-, 1, and 2 types in the half of the distribution conditioned by the homozygous recessive major gene, while there were mostly 3 and 4 types with the Lemhi testcross. The

Table XXXVIII. The infection types of the parents, Rego and P. I. 178383, the F_1, the F_2, and the testcross distributions at 2/18 and the results of chi-square tests for goodness of fit.

Parents and progeny	Infection types and observed no. of plants									Total	Ratio*	X^2 value	P value
	$\overset{v}{oo}$	oo	0-	0	1-	1	2	3	4				
P. I. 178383	all												
Rego				28	7	7	5			47			
F_1		all oo-											
F_2	69	45	8	15	4	3	6	4	6	160			
			122			38					3:1	.13	.50-.75
	69		53			38					1:2:1	30.23	<.005
Rego^2 x P. I. 178383	11	28	4	9	5	9	7	5		78			
			43			35					1:1	.82	.25-.50
(Rego x P. I. 178383) x Lemhi	1	29	6	4			3	15	12	70			
			40				30				1:1	1.43	.10-.25

* The ratios of the 3:1 and 1:1 of the Rego testcross include types $\overset{v}{oo}$, oo, 0- to types 0, 1-, 1, 2, 3, 4. The 1:2:1 ratio includes type $\overset{v}{oo}$ to types oo, 0- to types 0, 1-, 1, 2, 3, 4. The 1:1 ratio of the Lemhi testcross includes types $\overset{v}{oo}$, oo, 0-, 0 to types 1-, 1, 2, 3, 4.

difference would be due to Rego factors. Another difference apparently due to Rego factors was the presence of $\overset{v}{oo}$ types with the Rego testcross. These factors must be responsible for modifying the heterozygous major enough to condition $\overset{v}{oo}$ types that were equivalent to the infection type conditioned by the homozygous dominant major gene of P. I. 178383.

Specific plants were selected from the F_2 and both testcross segregations and selfed to determine how the various infection types were inherited. These plants were selfed and the seed was divided to grow F_3 lines at both temperature profiles. The F_3 distributions for each selected plant are given in Table V in the Appendix. Again, because of limited facilities, only a few plants were selected.

The F_3 lines were indicative of the difficulty encountered in distinguishing the homozygous dominant major from the heterozygous major when the factors from Rego were present. From the F_2 fourteen lines were tested of which four were $\overset{v}{oo}$ types. Of these four lines only one remained homozygous for the $\overset{v}{oo}$ reaction. The other three segregated 3:1 for a heterozygous major.

Five of the fourteen lines were selected as 0 types, and none of these 0 types segregated for the major. The 0 type F_2 plants were then placed within that portion of the ratio conditioned by the homozygous recessive major factor. One oo plant was saved, and it segregated for a heterozygous major factor.

Of the two 0- types selected, one segregated for the major factor and one did not. The one that did segregate for the major factor

produced 4 plants out of 45 that were also the 0- type at 2/18. For this cross at 2/18, the dividing point between the influence of the heterozygous and the homozygous recessive of the major factor of P. I. 178383 would be within the 0- types. This division point was further demonstrated by the Rego testcross.

The 0- selected in the Rego testcross did not segregate for the major. Of twenty-two plants checked at 2/18, two came back as 0- types. Since there could not be a homozygous dominant factor in the Rego testcross, the selected $\overset{v}{o}o$ types should segregate 3:1. Both of the $\overset{v}{o}o$ types saved did. It was observed that of the two testcrosses, only the Rego testcross at 2/18 conditioned a significant number of $\overset{v}{o}o$ types.

Only five of the eleven Lemhi testcross plants lived to produce F_3 seed. Of these there was only one plant each of the $\overset{v}{o}o$, oo, 0-, 0, and 3 types. The $\overset{v}{o}o$, oo, 0-, and 0 plants all produced F_3 lines that segregated for the major gene. The 3 type produced F_3 plants that were mostly 4 types at both temperature profiles. Just as at 15/24, the heterozygous major conditioned the 0- and 0 types, and the majority of the plants with a homozygous recessive major were 4 types. The influence of the minor factors in Rego and P. I. 178383 versus Lemhi was clearly demonstrated by these selected lines.

Interaction of Rego x P. I. 178383 with the temperature profiles 2/18 and 15/24 Both Rego and P. I. 178383 were shown to contain temperature sensitive factors that conditioned greater resistance at

one temperature profile than the other. The greatest difference, however, was in the direction in which these factors operate. P. I. 178383 was shown to have greater resistance at 15/24, and Rego was shown to have greater resistance at 2/18. How these temperature sensitive factors operate when combined was the next topic investigated. In this section the F_2's, F_3's, testcrosses, and testcross-F_2's will be compared for interactions to temperature. In the next section the various aspects of this cross compared with Rego x Lemhi and Lemhi x P. I. 178383 will be given.

The F_2 distributions of Rego x P. I. 178383 at the two temperature profiles are presented in Table XXXIX. Since these two distributions were produced from randomly selected seed, they should form the same distributions unless there was a significant interaction with temperature. The interaction chi-square value was highly significant, revealing that the frequencies of the various infection types were different and that there was a genotype by temperature interaction. It appeared that some 4, 3, and 2 types at 15/24 would be 1, 1-, or 0 types at 2/18, and part of the 0- and oo types at 15/24 would be $\overset{v}{o}o$ types at 2/18. These data disclosed that the Rego temperature sensitive factors interact with the temperature profile to a greater degree than do the P.I. 178383 temperature sensitive factors.

If just that part of the F_2 and testcross distributions at both temperatures were considered that were without the influence of the major dominant factor from P. I. 178383, there would be a one gene

Table XXXIX. Chi-square test for interaction[1] of F_2 distributions of Rego x P. I. 178383 at the two temperature profiles.

	F_2 infection types and observed number of plants									Total
	V oo	oo	0-	0	1-	1	2	3	4	
At 2/18	69 +	45 -	8 -	15 +	4 +	3 +	6 -	4 -	6 -	160
At 15/24	52 -	62 +	10 +	1 -	1 -	2 -	14 +	24 +	9 +	175
Total	121	107	18	16	5	5	20	28	15	335

[1] $X^2 = 36.07^{**}$ Significant at the 1% level.

segregation for 0, 1-, 1, 2, and 3 types to 4 types. If the possible effects of P. I. 178383's minor factors were ignored, these segregations would indicate that Rego possesses only one dominant gene that conditions the 3 or better reactions. However, since the effects of P. I. 178383's minor genes are not precisely known, there could be two dominants as indicated in the Rego x Lemhi crosses.

When the Rego testcross distributions produced at both temperature profiles were tested for interactions, there was a highly significant difference. This difference was expected, however, because in this distribution there were three times as many Rego factors as P. I. 178383 factors, and the temperature sensitive influence of Rego was even more completely expressed. The comparison showed that there was nearly a complete void of 3 and 4 types at 2/18 and nearly a complete void of 0-, 1-, 1, and 2 types at 15/24. The impact of temperature differences or interactions was clearly demonstrated.

When the Lemhi testcross distributions were tested for independence at the two temperature profiles, there was a significant difference only at the 5% level. The elimination of infection type oo showed that the significance occurred for this type and not for the types conditioned solely by the minor factors. Because Rego and P. I. 178383 would have each contributed only one-fourth of the factors in this testcross, the interaction with temperature was not expected as it was for the F_2 or Rego testcross. All factors in this testcross would have been heterozygous, and this not only strengthened the argument for at least one dominant in Rego to condition infection

type 3, but would also have further suggested that the temperature sensitive factors are recessive.

The selected F_3 lines can also be used to demonstrate the temperature sensitive factors of both parents. Some F_3 lines were more resistant at 15/24 than 2/18, e.g., selection numbers 3 and 10 (see Appendix Table V). Some F_3 lines were just the opposite being more resistant at 2/18, e. g., selection numbers 14 and 18. There was a third situation present in which the distributions were similar and not statistically different, e.g., selection number 4.

The F_3 lines also demonstrated the influence of combining the minor factors from both parents. Line 76 which was selected as a 0- at 2/18 is an example. This line was more resistant than the Rego parent at both 2/18 and 15/24, thus both factors from Rego and P. I. 178383 were involved in this line. Many of the lines (e.g., 70) were equal to Rego at 2/18 but were more resistant than Rego at 15/24.

These F_3 lines revealed that the temperature sensitive factors from the two parents were not in common.

VII. Interaction of factors from P. I. 178383 and Rego

Although the comparison between the Rego x P. I. 178383 cross gave some indication of the relative effectiveness of the two opposing types of temperature sensitive factors by comparing the distributions at different temperature profiles, it did not show what possible interactions these combined factors had at the same temperature profile. By comparing the F_2 distributions of Rego x Lemhi, Lemhi x P. I. 178383, and Rego x P. I. 178383 at both 2/18 and 15/24,

it should be possible to determine if significant interactions occurred for increased resistance.

Interaction at 15/24 In the comparison between the three crosses presented in Table XL, the primary emphasis was placed upon that portion of the distributions which did not contain the homozygous dominant or heterozygous dominant factor from P. I. 178383.

When the F_2 of Rego x Lemhi was compared with the F_2 of Lemhi x P. I. 178383 for infection types 1- through 4, the chi-square test for independence was significant. Since this comparison involved Rego or P. I. 178383 crossed with the highly susceptible parent Lemhi, the differences in distributions must be caused by different factors in the Rego and P. I. 178383 parents. At this temperature profile, Lemhi x P. I. 178383 had a greater proportion of more resistant types than Rego x Lemhi.

In the comparison between the F_2 of Rego x Lemhi and the F_2 of Rego x P. I. 178383, the significant difference showed that the distributions were dependent upon the different genotypes of each F_2. The F_2 distributions of Rego x P. I. 178383 had the greatest number of resistant types.

The third comparison at this temperature involved the F_2 distributions of Rego x P. I. 178383 and Lemhi x P. I. 178383. There was again a significant difference disclosing that the F_2 distributions were composed of different genotypes that interacted with the pathogen in ways to cause different proportions of infection types. The data suggested that the F_2 of Rego x P. I. 178383 had a higher number of

Table XL. Chi-square tests of independence to compare the F_2 infection type patterns for types 0, 1-, 1, 2, 3, and 4 produced at 15/24 by Lemhi x P. I. 178383, Rego x Lemhi, and Rego x P. I. 178383.

	F_2 infection types and observed no. of plants						Total
	0	1-	1	2	3	4	
1. Lemhi x P. I. 178383 vs Rego x Lemhi. $X^2 = 44.01^{**}$							
Lemhi x P. I. 178383	0	1 +	2 +	9 +	13 -	45 +	70
Rego x Lemhi	0	0 -	0 -	2 -	92 +	59 -	153
Total	0	1	2	11	105	104	223
2. Rego x Lemhi vs Rego x P. I. 178383. $X^2 = 51.97^{**}$							
Rego x Lemhi	0 -	0 -	0 -	2 -	92 +	59 +	153
Rego x P. I. 178383	1 +	1 +	2 +	14 +	24 -	9 -	51
Total	1	1	2	16	116	68	204
3. Rego x P. I. 178383 vs Lemhi x P. I. 178383. $X^2 = 27.04^{**}$							
Rego x P. I. 178383	1 +	1 +	2 +	14 +	24 +	9 -	51
Lemhi x P. I. 178383	0 -	1 -	2 -	9 -	13 -	45 +	70
Total	1	2	4	23	37	54	121

** Significant at the 1% level.

genotypes that conditioned infection types 0, 1-, 1, 2, and 3 than Lemhi x P. I. 178383 which conditioned mostly 4 types.

From the comparisons of these three F_2 distributions, the following relationships were established for the ability of these crosses to condition the greatest resistance in the F_2:

Rego x P. I. 178383 > Lemhi x P. I. 178383 > Rego x Lemhi.

These comparisons pointed out that the minor factors were not in common for each parent. Since the Rego x P. I. 178383 cross conditioned greater resistance than either the Rego x Lemhi or Lemhi x P. I. 178383 cross, there must have been an interaction between the Rego factors and the P. I. 178383 factors to condition greater resistance than either could alone. This observation of transgressive segregation would fit a multigenic situation in which the addition of every additional favorable allele causes the plant to be that much more resistant. In other words, there would appear to be additive gene action operating for the minor factors of Rego and P. I. 178383 at this temperature profile.

Interaction at 2/18 The same three comparisons between the F_2 distribution were made at this temperature profile (see Table XLI).

When the F_2 distributions of Lemhi x P. I. 178383 and Rego x Lemhi were compared, there was a very definite proportion of Rego x Lemhi plants that showed greater resistance than the Lemhi x P. I. 178383 plants. Only the 4 types of Lemhi x P. I. 178383 were proportionally larger. This comparison contrasted the abilities of different genotypes to respond to temperature changes, for at the

Table XLI. Chi-square test of independence to compare the F_2 infection type patterns for types 0, 1-, 1, 2, 3, and 4 produced at 2/18 by Lemhi x P. I. 178383, Rego x Lemhi, and Rego x P. I. 178383.

	F_2 infection types and observed no. of plants						Total
	0	1-	1	2	3	4	
1. Lemhi x P. I. 178383 vs Rego x Lemhi. $X^2 = 71.02^{**}$							
Lemhi x P. I. 178383	0 -	0 -	0 -	0 -	1 -	35 +	36
Rego x Lemhi	8 +	10 +	35 +	87 +	171 +	139 -	450
Total	8	10	35	87	172	174	486
2. Rego x Lemhi vs Rego x P. I. 178383. $X^2 = 123.20^{**}$							
Rego x Lemhi	8 -	10 -	35 -	87 +	171 +	139 +	450
Rego x P. I. 178383	15 +	4 +	3 +	6 -	4 -	6 -	38
Total	23	14	38	93	175	145	488
3. Rego x P. I. 178383 vs Lemhi x P. I. 178383. $X^2 = 50.31^{**}$							
Rego x P. I. 178383	15 +	4 +	3 +	6 +	4 +	6 -	38
Lemhi x P. I. 178383	0 -	0 -	0 -	0 -	1 -	35 +	36
Total	15	4	3	6	5	41	74

** Significant at the 1% level.

other temperature profile the opposite results were obtained. The environment in studies involving disease resistance and inheritance can then be a major factor in the expression of infection types and can influence the ratios obtained from one test to the next.

In the next comparison there was also an interaction of genotypes, even though both crosses contained Rego. The Rego x P. I. 178383 cross produce more 0 and 1- types than did the Rego x Lemhi cross.

To complete this series of comparisons, the F_2 of Rego x P. I. 178383 was compared with the F_2 of Lemhi x P. I. 178383. The Rego x P. I. 178383 progeny again conditioned greater resistance.

Just as with the three comparisons at 15/24, the crosses can be placed in order by the increased proportion of resistant plants occurring in each cross. The relationship for these three crosses is:

Rego x P. I. 178383 > Rego x Lemhi > Lemhi x P. I. 178383.

Because the Rego x P. I. 178383 F_2 plants were significantly more resistant than either the Rego x Lemhi or Lemhi x P. I. 178383 F_2 plants, there must again have been an interaction between the factors from P. I. 178383 and Rego that conditioned greater resistance than either parent in a cross with highly susceptible Lemhi. When a wider array of minor factors for resistance were brought together, transgressive segregation and additive gene action were again implicated at this temperature profile.

DISCUSSION

In the early 1960's when stripe rust reappeared in the Northwest as an epiphytotic, it caused extensive reduction in wheat yield. Although this disease was probably endemic in the Northwest, there was very limited knowledge about the inheritance and sources of host resistance. As is the case with other cereal rusts, the most practical method of control is with resistant varieties.

Early studies in Europe and later studies in this country revealed that the pathogen and host-pathogen complex were very sensitive to environmental conditions. This disease was apparently much more sensitive to the environment than had been reported for the other rust diseases. Preliminary studies by Sharp (1962a, b; 1965) soon after this disease devastated susceptible wheat varieties in Western Montana showed that, in general, there were four responses of this disease to changing environmental conditions, particularly temperature. He observed that highly resistant varieties retained their resistance across temperature changes, that highly susceptible varieties remained highly susceptible, that most moderately resistant varieties became more resistant as temperatures increased, and that a few moderately resistant varieties became more susceptible as temperatures increased.

These response types were first suspected of being changes in race patterns or were attributed to so called "mature plant resistance." Sharp (1965) eliminated the first of these possible explanations by showing that only a single race was involved. He also found that seedling plants underwent corresponding changes with changing temperatures. However, further investigation was needed, and inheritance studies were

initiated to determine the nature of disease resistance for these various response groups.

Three types of resistance factors were found to condition stripe rust resistance in the four varieties reported in this investigation. Single dominant genes which controlled non-temperature sensitive resistant reactions were found in P. I. 178383 and Chinese 166. Recessive factors which were temperature sensitive were found in P. I. 178383, Chinese 166, and Rego. Dominant factors which were relatively non-temperature sensitive but which conditioned only moderate incompatibility were found in Rego.

The temperature insensitive, dominant factors of P. I. 178383 and Chinese 166 were called major genes. Although these genes were independently inherited, they were nearly alike in action. When either was homozygous dominant, they conditioned a $o^{v}o$ infection type. Dominance was not complete, however, and the heterozygote conditioned infection types from oo to O. The double heterozygote was equivalent to either factor being homozygous dominant. There was thus a dosage effect for these factors, and any combination of two dominants resulted in the same infection type.

These major factors probably correspond to the major genes found in flax on which a gene-for-gene system was hypothesized for the flax--*Melampsora lini* interaction (Flor, 1956). However, to determine this, crosses between virulent and avirulent rust races would need to be made for a particular major factor and a one-to-one relationship established. Because *P. striiformis* has no known sexual cycle, this gene-for-gene interaction can only be speculated upon.

The physiological basis for resistance due to major genes is not well

understood. Tomiyama (1963) reviewed the physiological nature of disease resistance and summarized the findings. He suggested that this type of resistance was due to hypersensitivity of host cells which caused death to the intruding fungus. The earlier the death of the host cells took place, the more resistant were the plants.

The dosage effect of the major genes found in P. I. 178383 and Chinese 166 must be involved with the speed at which host cell death occurs. The difference between the $o^{v}o$ and oo to O infection types would then be due to the speed of reaction, with the greatest speed occurring for the $o^{v}o$ type and actually resulting in the least damage to the host.

The second and temperature sensitive type of resistance in the host varieties was apparently due to recessive gene action. Rather than initiating violent hypersensitive reactions within the host, this type of resistance appeared to change the degree of compatibility of the host-pathogen complex.

Tomiyama (1963) also reviewed the physiological aspects of this type of disease resistance. In field resistance, or the type of resistance conditioned by minor genes, any change in physiological factors may result in changes of disease resistance. Factors such as toughness of the cell wall, which restricts or delays penetration by the parasite, the nutrition in the host which may be unfavorable to the parasite, and certain toxins or metabolic analogs which affect metabolism may be involved.

The influence of different environments in some manner is involved with the expression of these and probably other physiological factors which

change the balance or interaction between the host-parasite components so that either increased or decreased resistance occurs. The level of metabolism at which temperature has its greatest influence is not known, i.e., the effect of temperature could occur anywhere from the genic level to the level of the final products. The influence of temperature is well-known for enzymatic reactions, and it could be related to the amount of product produced. If this type of resistance were nutritional, then in most cases it would seem that increased temperatures would cause greater susceptibility, or compatibility, as is the case with Rego. However, if resistance were based upon toxins produced by the host, then there could be greater resistance at higher temperatures. It is doubtful that only one system would be involved and that the compatibility of host-parasite systems depends simply upon the supply or amount of certain materials, either nutritional or toxic.

Strobel and Sharp (1965) demonstrated that several proteins in several varieties showed qualitative differences concomitant with changes in the environment in which the plants were grown. Rego was one of these varieties. Just how this protein difference was involved in changing the reaction of Rego to this parasite was only speculated upon and is being further investigated.

The use of a diurnal temperature was shown by Sharp (1965) to create natural responses for wheat varieties to stripe rust. Smith (1966) showed that the use of diurnal cycles with several amplitudes of temperature change, significantly changed the expression of disease resistance in

charcoal root disease of pine. Since the difference between diurnal or constant temperatures could affect the metabolism, use, and accumulation of certain products, the use of a simulated cycle in disease investigations would generally increase the accuracy of the experiment.

Increased resistance in adult plants or the so-called mature plant resistance may partially be due to a temperature response. In the field, the mature plants would generally be grown under relatively higher temperatures, and increased resistance is most often noted with higher temperatures.

To this point, the primary concern has been with the effects of temperature on the host. Temperature may also influence the speed of certain parasite reactions and its relationship to the host.

The operation of an additive gene system in the host could be explained in terms of these same physiological factors. For example, if a fungus were grown upon a complete media (or a very susceptible host variety) at a set temperature profile, it would achieve a certain growth rate. If one of the necessary nutrients were reduced one-half, the growth rate would probably be reduced also. If a second factor were cut in half (or a less susceptible host variety) or the same factor further reduced, then the growth rate would be decreased even more until the fungus was no longer able to grow or starvation occurred. The act of reducing the required nutrients would then be additive in decreasing the rate of growth. The same situation could be visualized for the addition of toxic materials or metabolic analogs.

What the pathogen does to the host is probably of equal importance

in the establishment of compatibility, but since the same race was always used in this study, this aspect will not be discuessed further.

The third type of resistance, such as the factors that condition the 3 infection type in Rego, is probably much the same as the previous type, except it is not as likely to change under different environments and it appears to act as a dominant. In P. I. 178383 or Chinese 166, all minor factors were probably temperature sensitive, but the factors in Rego conditioning the 3 infection type were probably not entirely in common with certain other temperature sensitive factors.

Within a single plant several of these types of resistances are possible, and P. I. 178383 and Chinese 166 were shown to have both major and minor temperature sensitive factors. Apparently these factors do not react as separate entities but act to reinforce each other. For example, the heterozygous major gene would probably condition a 0 reaction by itself. However, with the influence of these minor, modifying factors, it conditioned a oo type in the F_1 of Lemhi x P. I. 178383 and Chinese 166 x Lemhi and was able to modify the heterozygote to condition a $o^v o$ in some instances, for example, the F_1 of Rego x P. I. 178383 at 2/18. It is possible that the homozygous dominant major gene may result in an infection type higher than a $o^v o$ in the absence of all minor genes.

These several types of resistance are probably similar to the factors observed in Fusarium yellows in cabbage which were called type A and type B (Blank, 1937). Here the type A resistance would be the major factors, and the type B resistance would be the temperature sensitive minor factors.

The maximum resistance conditioned by minor genes in either P.I.

178383 or Chinese 166 varieties were not exactly determined because of the epistatic major genes, however, F_3 segregation patterns suggested that both would condition at least a 0 infection type. In fact, when Race 13 (virulent to the major gene in Chinese 166) was checked against Chinese 166, a 0 to 2 infection type was obtained at 15/24 (Sharp, personal communication). The maximum level of resistance possible with just minor factors from two parents was not determined, but F_3 segregation for Rego x P. I. 178383 suggested that transgressive segregation occurred to produce 0- to oo infection types. Studies underway at Montana State University have demonstrated that at least the oo infection type can be obtained with minor factors alone.

From the results of this study, all the infection types used would appear to have a genotypic basis. In fact there were probably more infection types than the eye was able to distinguish. The use of set types as $\overset{v}{oo}$,oo, 0-, 0, 1, 2, 3, and 4 and plus and minus types made a good system to study disease resistance, but as more minor factors became involved, the differences between types became less discrete. There was probably a continuous variation within parts of the infection type range, depending upon the cross. However, the division between the influence of the major and minor genes can be very discrete, for example, the Lemhi x P. I. 178383 cross at 2/18.

For the Rego x Lemhi cross at 2/18 and for the oo and 0 types of the Lemhi x P. I. 178383 cross at 2/18 and 15/24, the F_2 infection types were shown to be good indicators of the genotypes. Under these controlled environmental conditions, the heritability of resistance was high, even

for the minor factors.

Some of the infection types were conditioned by quite different genotypes. Either of the heterozygous major genes, with certain minors or certain combinations of minors, were shown to be able to condition infection types 0-, 0, and 1-. Depending upon the cross, the number of minor factors, and the temperature profile, each situation was somewhat different as to which types were conditioned by the major genes and which were conditioned by the minor genes.

Major genes are preferred for use in breeding programs when they are known. The reasons are fairly simple. A single major gene usually conditions a high level of resistance, and it could be easily followed in a breeding program. However, the use of major genes only has had its drawbacks, as has been painfully experienced by many breeders. Where a one-to-one relationship exists for this type of resistance between the host and parasite, a single mutation or recombination in the parasite may be all that is required to have a change from an avirulent pathogen to a virulent one.

Several schemes have been devised using major genes to keep the pathogen under control. One is to employ several major genes in the same variety. This scheme is based on the tenet that the probability of two mutations or recombinations occurring at the same time is the product of the individual probabilities, and the chance of a change in the pathogen to virulence is reduced. A second scheme involves the use of isogenic varieties with different genes for resistance. Then, as the race patterns change, a new resistant isogenic could be used. However, these schemes

could eventually cause problems. As the pathogen is able to combine or mutate to virulent races, the virulent genes could be maintained in one race until a universal virulent race is evolved. It would then become more and more difficult to develop and retain resistant varieties.

From the plant breeders' standpoint, the quantitative forms of resistance are less desirable because of the increased problems of incorporating them into agronomically acceptable varieties. However, this type of resistance has been used successfully where major genes are not available. Since this type of resistance may not have a one-to-one relationship between the host and pathogen, and because a desirable level of resistance may result from several factors, this resistance may be more stable to race changes than the resistance conditioned by major genes. In certain situations this type of resistance could then be of value.

In crops where major genes are known, the minor factors may be used to greatest advantage to reinforce resistance. For example, a variety that had a single major gene could be protected against a race change by use of a moderate level of resistance from minor genes. Then if a mutation occurred that was virulent to the major gene, this new race would still have little selective advantage. By using major and minor factors for resistance the plant breeder would utilize further built-in protection against a possible race change that would impair the usefulness of his variety. He would also have resistance to a wider array of races, and would probably increase the resistance to individual races. This built-in protection would be particularly useful for stripe rust resistance, because mutations are the only known way for race changes to

occur (not considering race introductions).

At present, investigations are being conducted at Montana State University to: (1) determine the inheritance and number of minor factors involved, (2) determine on which chromosomes these factors are located, and (3) determine the physiological nature of the temperature sensitive resistance for several wheat varieties. These investigations combined with this study will provide a much better understanding of the inheritance of stripe rust resistance and of the nature of temperature sensitivity.

In this investigation Rego's inheritance of rust resistance and its temperature sensitivity were not thoroughly understood. Several studies could be devised to better understand the nature of this variety's unusual temperature response and how the several minor factors might be related to this response. Lower temperature profiles could be investigated to see if Rego's infection type could be maintained at a 0, and if so, how this might influence the distribution of segregating types. A series of chromosome substitutions from Rego into a monosomic could provide evidence on the inheritance of stripe rust resistance and possibly on how these factors influence the response of the substituion lines.

The hypothesis is proposed that major factors in this study had avirulent complementary or corresponding genes in the rust race while the minor factors did not and were not part of a strict gene-for-gene system. To determine if this were true for the interaction between the minor genes and the pathogen, several races could be checked against these varieties to see if the level of incompatibility varied and to see if temperature sensitivity was affected. This check would also serve as a test to

determine if the temperature sensitivity was a characteristic of the host, the rust race, or the interaction between the two. For example, it would be of interest to know if Rego or Chinese 166 (without the major gene) responded the same for the two temperature profiles to all races of the parasite.

The Rego x P. I. 178383 cross indicated that both types of temperature response could be obtained in the same F_3 line. It would be informative to know at what level both responses could be incorporated into the same true breeding line. For example, if the factors of Rego and Chinese 166 (not the major) were incorporated into the same line, then a source of resistance would be available that would condition resistance over wider temperature ranges. This line would further provide possible evidence on the factors that actually condition temperature response and how the opposing types operate within a single plant.

SUMMARY AND CONCLUSIONS

In this investigation the inheritance of resistance to stripe rust of four wheat varieties was studied at two controlled diurnal temperature profiles, 2/18 and 15/24. The parents were selected because they yielded the most information. Both Chinese 166 and P. I. 178383 gave a $\overset{v}{oo}$ infection type at both profiles. Rego had predominantly a 0 infection type at 2/18 and a 3 infection type at 15/24. Lemhi was very susceptible at both profiles and gave a 4 infection type.

The F_1, F_2, F_3, testcross, and testcross-F_2 segregations from a diallel set of crosses (except Chinese 166 x Rego) were used to determine the inheritance of stripe rust resistance and the interaction of the temperature profile with resistance genes. Progenies from selected plants were used to interpret the range of expression of particular genes or gene combinations and to determine the reliability of using infection types as an indication of the genotype.

From the results of this study, the following conclusions can be made:

1. P. I. 178383 and Chinese 166 possess different major, partially dominant genes for resistance, which are independently inherited. When homozygous dominant, either of these factors condition a $\overset{v}{oo}$ infection type, or condition a oo to 0 infection type when heterozygous. The double heterozygote conditions a $\overset{v}{oo}$ infection type to demonstrate a dosage effect for these major factors.
2. P. I. 178383 has a number of temperature sensitive minor factors that condition greater resistance at 15/24 than 2/18. There is

a significant interaction between these factors and the temperature profile. In a cross with Lemhi, these factors show additive gene action and, in certain combinations, can condition at least a 0 infection type.

3. Chinese 166 has at least one temperature sensitive factor, of which the predominant one may be either recessive or partially dominant. This factor conditions greater resistance at 15/24 than at 2/18. At 15/24 a 0 to 2 infection type is conditioned.
4. The minor factors of P. I. 178383 and Chinese 166 interact with the major genes to condition increased resistance. There is a greater interaction at 15/24 than at 2/18.
5. The temperature sensitive minor factors of P. I. 178383 are responsible for the difference between infection types oo and 0. These minor factors are the same as those that express themselves in the absence of the major gene to condition infection types 0, 1, 2, and 3.
6. The minor factors of P. I. 178383 and Chinese 166 are not entirely in common. These factors show additive gene action when brought together at both temperature profiles. The additivity is greater at 15/24. The combination of these factors conditions greater resistance than either set alone.
7. Rego shows just the opposite temperature sensitivity of the minor factors of P. I. 178383 and Chinese 166. Rego does not possess a dominant, major gene.
8. Rego possesses two pair of complementary dominant genes that

condition a 3 infection type at both temperature profiles. In addition to these factors, Rego probably has several other temperature sensitive minor genes. The combination of all these factors are needed to condition a 0 reaction at 2/18.

9. The temperature sensitive factors of Rego condition two significantly different F_2 distributions at the two temperature profiles, i. e., there is a significant genotype by temperature interaction. The factors that condition the 0 to 1, 2, 3, and 4 infection types at 2/18 condition 3-, 3, 3, and 4 infection types at 15/24, respectively. The infection types are a good indication of the genotype at 2/18.

10. The minor, temperature sensitive factors of P. I. 178383 and Rego are not entirely in common. When these factors are crossed together, additive gene action and transgressive segregation for increased resistance occur at both temperature profiles. Both directions of temperature response are maintained in such a hybrid segregation.

11. The factors of Rego are able to modify the expression of the major gene in P. I. 178383 to increased resistance, particularly at 2/18.

12. The interaction of both minor genes and temperature can cause significant differences in the segregation distributions. These interactions cause the distributions of each cross at each temperature profile to be singularly different. For genetic studies of stripe rust resistance, these factors should be taken into

consideration instead of arbitrarily calling certain infection types resistant and certain infection types susceptible.

APPENDIX

Appendix Table I. Infection type distributions of randomly selected Lemhi x P. I. 178383 F_3 lines grown at 2/18 and 15/24* that were selected from F_2 seedlings grown at 2/18.

Selection number	F_2 infec. type	F_3 infection type and observed no. of plants									Total
		;oo	oo	0-	0	1-	1	2	3	4	
255	;oo	42	7								49
		38									38
256	oo	6	4		1					8	19
		10	2	5	1				2	18	38
257	;oo	58	1								59
		39									39
258	oo	14	19					1	5	5	44
		35	8		2			4	5	4	58
259	;oo	39									39
		39	1								40
260	oo	11	22	5	1			1	2	8	50
		18	23	1	3		1	4	1	8	59
261	;oo	40									40
		39									39
262	oo	35	16						4	4	59
		36	11					1	5	7	60
263	;oo	60									60
		39									39
264	0	3	4	4	2					7	20
		5	6	5	6				4	6	32
265	4							2	3	14	19
								1	15	18	34
266	oo	10	22	5	1					9	47
		24	22	1	2				3	6	58

* For each F_3 line, the distribution tested at 2/18 is listed first and the distribution grown at 15/24 is listed second.

Appendix Table I. (cont.)

		♂♀	♂♂	0-	0	1-	1	2	3	4	
267	4	Died									
268	♂♂	17						1	2		20
		16					2	3	3		35
269	0	9	11	12	4					5	41
		23	14	3	4	1			3	8	56
270	4								2	50	52
								1	12	44	57
271	4				1			6	9	56	72
							1	11	10	54	76
272	0	6	12	12	6				6	9	51
		21	17	6	2		1	1	8	3	59
273	0	15	12	7	3				1	7	45
		19	21	6	2			1	3	8	60
274	4						1	22	19	33	75
									33	47	80
275	♂♂	10	13	7				1	2	8	41
		22	13	6				3	8	8	60
276	4							2	4	41	47
									17	42	59
277	4								1	37	38
									6	53	59
278	♂♂	12	21	8						8	49
		8	21	7	6			2	6	10	59
279	0		6	11	9	1			3	10	40
		10	26	8	3			1	1	10	59
280	♀♂	38									38
		40									40
281	4							1	3	46	50
									9	51	60

Appendix Table I. (cont.)

		V oo	oo	0-	0	1-	1	2	3	4	
282	0	9	11	9	9				2	7	47
		10	15	4	17				4	11	61
283	V oo	38	1		1						40
		51	9								60
284	oo	19	14	1					1	5	40
		23	12	7	2			1	7	5	57
285	V oo	37	2	1							40
		38	1								39
286	4	Died									
287	oo	25	9							5	39
		29	22	1	2			2	2	2	60
288	oo	16	15	3	1				3	2	40
		22	22				1	4	7	4	60
289	oo	24	7					3	2	4	40
		26	15	4	3		1	2	6	1	58
290	0	5	13	9	3					10	40
		16	10	5	14				3	13	61
291	0	7	6	9	8	1			3	4	38
		11	24	4	4				4	12	59
292	oo	18	15	1			1	1	2	2	40
		23	25	1					2	8	59
293	oo	22	4						1	8	35
		28	17					3	9	3	60
294	V oo	28	9								37
		34	27								61
295	oo	26	5	1					5	3	40
		31	12	2			2	2	8	2	59
296	oo	19	12	1					5	3	40
		33	14	1	1		2	1	7	1	60

Appendix Table I. (cont.)

		V oo	oo	0-	0	1-	1	2	3	4	
297	V oo	35									35
		37									37
298	oo	23	7	1				3	5	1	40
		36	9				1	7	5	2	60
299	4				1	1	3	7	7	21	40
							3	15	21	16	55
300	oo	12	11	1	1				1	9	35
		23	15	5					2	14	59
301	oo	16	10	7					1	5	39
		21	17	8	1				5	7	59
302	oo	16	14	1					2	6	39
		22	17	3						12	54
303	0	5	7	8	7					6	33
		8	9	7	4				4	5	37
304	oo	21	8					3	1	7	40
		27	18	1				3	4	6	59
305	oo	17	9	1				3		8	38
		25	23	1	1			1	1	7	59
306	4							3	8	25	36
							3	2	28	44	80
307	oo	16	15	1				1	1	3	37
		31	14	1			3	5	3	2	59
308	4								13	26	39
								2	43	12	57
309	V oo	37									37
		56	2	1							59
310	4							3	2	25	40
					1		1	10	17	30	59
311	V oo	23	15	2	1						41
		45	16								61

Appendix Table I. (cont.)

		$\overset{v}{oo}$	oo	0-	0	1-	1	2	3	4	
312	oo	18	16					1	3	2	40
		29	17	1			4	5	3		59
313	$\overset{v}{oo}$	41									41
		38	2								40
314	4							2	6	27	35
					1	1	1	12	14	31	60

Appendix Table II. Infection type distributions of non-randomly selected Lemhi x P. I. 178383 F_3 lines and Lemhi testcross-F_2 lines at 2/18 and 15/24* that were selected from F_2 and testcross seedlings grown at both 2/18 and 15/24 as designated.

Selection number**	F_2 infec. type	F_3 or TC-F_2 infection types and observed no. of plants									Total
		⁰ᵛo	oo	0-	0	1-	1	2	3	4	
Selected from F_2 grown at 15/24											
29	0	3	6	7						4	20
		15	5	5	26				2	16	69
30	4									21	21
									1	49	50
32	4									21	21
									8	60	68
33	oo	4	10	2					2	2	20
		24	18	15	6			2	11	9	85
34	4									21	21
							1	1	20	65	87
35	4								2	20	22
						1	2	5	41	37	86
36	⁰ᵛo	12	3					1	2	3	21
		37	17	1	1		5	4			65
37	⁰ᵛo	22									22
		41									41
38	oo	7	6	1	1			1	1	3	20
		23	10	1	1			1	2	4	42
39	⁰ᵛo	23									23
		60									60

* When F_3 lines were tested at both temperature profiles, the line tested at 2/18 is listed first, and the line grown at 15/24 is listed second. When there is only one profile used, it is 15/24. Only selection numbers 29 through 41 were tested at 2/18.

** When selection numbers are missing, the selected plant died before producing F_3 seed.

Appendix Table II. (cont.)

		$\overset{V}{oo}$	oo	0-	0	1-	1	2	3	4	
41	oo	8	6	2					3	3	22
		31	18	1	6	1	2	3	15	3	80
42	4				2	5	9	11	42	14	83
43	4							3	54	29	86
44	0	16	18	2	13	1	2	4	3	3	62
45	0-	8	12	5	8		2	1	2	5	43
46	oo	35	22	3	5	1	3	6	10	3	88
47	$\overset{V}{oo}$	17									17
Selected from Lemhi testcross grown at 15/24											
48	4								5	31	36
49	3								13	82	95
50	0-	22	12	5	5				5	10	59
51	0	13	1	8	17				8	9	58
52	4								16	61	77
53	4								9	51	60
54	4					1	3	6	24	49	83
55	0	23	7	3	13					10	56
56	4								17	60	77
57	0	17	10	5	18				1	11	62
58	4							1	10	41	52
Selected from F_2 grown at 2/18											
102	oo	19	11	4	1	1	1		2	2	41
103	4							4	14	29	47

Appendix Table II. (cont.)

		V oo	oo	0-	0	1-	1	2	3	4	
105	4				2	4	7	13	21	6	53
106	oo	25	6		5		3	2	1		42
109	V oo	19	1								20
111	oo	26	5	1	2	2	2	1		3	42
112	0	20	4	3	6				5	8	46
113	4				3	1	4	8	10	20	46
115	4							6	5	45	56
118	0	9	6	6	7				2	11	41
173	4				1				10	34	45
174	4				5	1	7	9	13	4	39
175	0-	15	13	3				1	2	5	39
176	oo	22	9	3	1	1			3	4	43
Selected from Lemhi testcross grown at 2/18											
123	4								11	31	42
126	0-	12	5	2	14					9	42
127	4								3	38	41
128	4								5	32	37
Selected from Lemhi testcross grown at 15/24											
154	0-	23	8	8	10			1	2	6	58
155	3								10	66	76
156	3								31	68	99
157	0-	17	16	6	3				1	16	59

Appendix Table II. (cont.)

		V oo	oo	0-	0	1-	1	2	3	4	
158	oo	22	21	5	4				3	6	41
159	oo	22	23	10	6			1	9	13	84

Appendix Table III. Infection type distributions of selected Chinese 166 x P. I. 178383 F_3 lines grown at 2/18 and 15/24* that were selected from F_2 seedlings grown at 2/18.

Selection number	F_2 infec. type	F_3 infection types and observed no. of plants									Total
		voo	oo	0-	0	1-	1	2	3	4	
201	4						1	1	6	11	19
					1		1	3	9	6	20
202	voo	19									19
		20									20
203	oo	7	10	1						2	20
		10	2	1					3	4	20
204	voo	20									20
		20									20
205	voo	17	1	1						1	20
		18			2						20
206	2		1		1		1	3	7	6	19
					3	2	2	3	10		20
207	voo	20									20
		19									19
208	oo	1	8	6		1	1		3		20
		12			5		1	1	1		20
209	oo	9	5		1	1	2	1	1		20
		13	3		2			1	1		20
210	voo	14	6								20
		19									19
211	oo	5	8						3	3	19
		8	4	3					2	3	20
212	4						1	3	12	4	20
					6	1	1	7	2	3	20

* For each F_3 line, the distribution tested at 2/18 is listed first, and the distribution tested at 15/24 is listed second.

Appendix Table III. (cont.)

		V oo	oo	0-	0	1-	1	2	3	4	
213	oo	2	11	3			3	1			20
		9	8			1		1	1		20
214	V oo	4	13				1		1		19
		14	3		1				2		20
215	V oo	20									20
		20									20
216	V oo	20									20
		19	1								20
217	V oo	20									20
		20									20
218	V oo	20									20
		19	1								20
219	V oo	19									19
		19									19
220	oo	5	7	1	1		1		3	1	19
		5	9	1	1			1	2	1	20
221	V oo	16	2		1				1		20
		17			3						20
222	V oo	20									20
		20									20
223	V oo	15	2	2					1		20
		19									19
224	0	9	5				1	2	1	2	20
		10	4	1					2	1	18
225	V oo	20									20
		20									20
226	V oo	18	2								20
		19									19
227	V oo	11	7		2						20
		14	1					2	1		18

Appendix Table III. (cont.)

		V oo	oo	0-	0	1-	1	2	3	4	
228	0	7	2	2	4	3		1	1		20
		13	2		3		1		1		20
229	V oo	19									19
		17	2		1						20
230	3		1		3	3	5	1	6	1	20
					12	2	3		3		20
231	V oo	17	1								18
		18			1				1		20
232	V oo	18	2								20
		10	7		1						18
233	V oo	13	5		2						20
		18	2								20
234	3							1	11	8	20
					3	2	2	3	9	1	20
235	V oo	14	1						5		20
		11	3		4			1		1	20
236	V oo	19									19
		19			1						20
237	V oo	20									20
		20									20
238	4									20	20
									10	10	20
239	V oo	12	4	3						1	20
		13	2		1		1	1	1		19
240	V oo	15	3						1		19
		12	6	1	1						20
241	oo	8	8						1	3	20
		15	2		1			2	1		21
242	oo	9	6		1			1	3		20
		11	1		1			2	2		17

Appendix Table III. (cont.)

		V oo	oo	0-	0	1-	1	2	3	4	
243	V oo	20									20
		19									19
244	V oo	20									20
		19									19
245	V oo	20									20
		20									20
246	V oo	17	2	1							20
		13	2		1				1		17
247	oo	7	10	1						2	20
		6	9						2	3	20
248	V oo	14	3	2						1	20
		19	1								20
249	V oo	20									20
		19									19
250	4								12	8	20
						1	2	12	3	2	20
251	oo	8	4	1	1			1	1	5	21
		4	9		1	1	1	1	1		18
252	4								4	16	20
									2	18	20
253	V oo	20									20
		21									21
254	V oo	18	1								19
		14	5								19

Appendix Table IV. Infection type distributions of non-randomly selected F_3 lines of Rego x Lemhi grown at 2/18 and 15/24* that were selected from F_2 seedlings grown at 2/18 and the mean infection types of each line.

Selection number	F_2 infec. type	F_3 infection type and observed no. of plants							Total	Mean infec. type**
		0	1-	1	2	3-	3	4		
Rego check		28	7	7	5				47	.5
					1	20	16		37	3.0
315	3		1	4	11	16	12	8	52	2.8
						1	9	9	19	3.5
316	4					1	4	50	55	3.9
								20	20	4.0
317	1-	44	11	3					58	.2
					5	11	4		20	2.8
318	1	7	10	18	10				45	1.1
						8	10	1	19	3.1
319	3	1	1	6	9	4	12	15	48	2.8
						3	3	14	20	3.7
320	2	15	15	9	7	3	1		50	1.0
					4	12	4	1	21	2.9
321	2+			1	7	8	20	6	42	2.9
						1	6	13	20	3.7
322	4				2		3	44	49	3.9
								20	20	4.0
323	3				1		14	11	26	3.4
							8	12	20	3.6
324	3		1	3	5	6	6	11	32	2.9
						3	9	7	19	3.4
325	4				1	1	5	18	25	3.7
							1	19	20	4.0

* For each F_3 line, the distribution grown at 2/18 is listed first.

** The mean infection type for each line was computed on the basis that infection type 0 = 0, 1- and 1 = 1, 2 = 2, 3- and 3 = 3, and 4 = 4.

Appendix Table IV. (cont.)

		0	1-	1	2	3-	3	4		
326	2	2	1	2	19	7	1		32	2.0
						9	7	3	19	3.2
327	3	5	2	3	9	1	2	3	25	1.8
						2	7	11	20	3.6
328	2	11	10	1					22	.5
					1	10	9		20	3.0
329	4						6	18	24	3.8
							2	18	20	3.9
330	2		1	5	10	2	8		26	2.2
							20		20	3.0
331	3		1	2	13	5	1	7	29	2.6
						3	8	10	21	3.5
332	3+	3	3	2	3	4	7	12	34	2.7
							14	6	20	3.3
333	3	4	2	7	4	2	5	4	28	1.9
						4	9	7	20	3.4
334	4			1	1	2	8	3	15	3.0
							4	15	19	3.8
335	2+	1	1	7	20	3	1		33	1.8
						3	14	2	19	3.1
336	2	8		3	9	3		7	30	1.9
							9	11	20	3.6
337	4							17	17	4.0
								15	15	4.0
339	2	12	4	6	3	4	2	5	32	1.3
							11	6	21	3.3
340	4	1		4	6	5	9	8	33	2.7
							12	9	21	3.4
341	1	11	9	10	5	1			36	.9
						6	11	1	18	3.1

Appendix Table IV. (cont.)

		0	1-	1	2	3-	3	4		
342	4						5	36	41	3.9
								19	19	4.0
343	4							34	34	4.0
								20	20	4.0
344	2	14	5	7	10		1		37	.9
					2	5	13		20	2.9
345	4	7		2	11	3	10	5	38	2.2
							7	13	20	3.7
346	4							26	26	4.0
								19	19	4.0
347	3					8	4	11	23	3.5
							9	11	20	3.6
348	2		2	9	13	5	3	5	37	2.3
							6	14	20	3.7
349	4						3	50	53	3.9
							2	18	20	3.9
350	2	12	1	5	8	4	1	3	34	1.4
						1	8	11	20	3.6
351	3	2	1	4	7	9	7	8	38	2.6
						2	14	5	21	3.2
352	1	9	4	16					29	.7
					1	6	10	2	19	3.1
353	4		1		7	6	13	9	36	3.0
						3	4	14	21	3.7
354	2	2	5	8	11	5	4	5	40	2.1
						2	4	14	20	3.7
355	2	11	3	6	6	1		5	32	1.4
						2	10	8	20	3.4
356	4						1	19	20	4.0
							1	18	19	4.0

Appendix Table IV. (cont.)

		0	1-	1	2	3-	3	4		
357	3	5	1	6	7	5	3	5	32	2.0
						1	6	13	20	3.7
358	2	1	2		8	5	1	12	29	2.9
					1	2	11	6	20	3.3
360	3	4	1	5	4	6	7	8	35	2.4
						1	12	7	20	3.4
361	4-	2	1	5	7	6	10	6	37	2.5
							7	14	21	3.7
362	1+	13	5	8	9	2			37	1.0
					6	9	5		20	2.7
363	2	1	3	1	11	3	13	5	37	2.5
					1		3	16	20	3.7
364	1	6	12	15	6				39	1.0
					11	6	4		21	2.5
365	4						3	31	34	3.9
								20	20	4.0
366	4						1	37	38	4.0
								19	19	4.0
367	3-	4	3	3	7	5	2	3	27	2.0
							7	12	19	3.6
368	3-	2	2	2	14	5	3	8	36	2.4
							8	11	19	3.6
369	3		1	2	6	6	11	8	34	2.9
							5	14	19	3.7
370	3	2	1	7	4	3	11	7	35	2.5
						1	9	9	19	3.8
372	4							40	40	4.0
							1	18	19	4.0
373	3		3	4		7	3	9	35	2.6
						1	6	13	20	3.6

Appendix Table V. Infection type distributions of non-randomly selected Rego x P. I. 178383 F_3 lines and Rego and Lemhi testcross-F_2 lines at 2/18 and 15/24* that were selected from F_2 and testcross seedlings grown at both 2/18 and 15/24 as designated.

Selection number	F_2 infec. type	F_3 and testcross-F_2 segregation									Total
		$\overset{v}{oo}$	oo	0-	0	1-	1	2	3	4	
F_3 lines selected at 15/24 in the F_2											
1	0-	2	8	4	1		4	2			21
		9	22	2	2		1	4			40
2	oo	9	6	1	3			2	1		22
		21	7	1					11		40
3	2						3	19			22
					1	9	12	16	2		40
4	$\overset{v}{oo}$	8	7				1	5		1	22
		19	10	1				5	4		39
5	$\overset{v}{oo}$	6	13	1		1	1	1		1	24
		21	9		1		2	2	5		40
6	3-			1	4	3	5	3	1	5	22
							11	9	19	1	40
7	$\overset{v}{oo}$	9	7		2	3	1				22
		17	11	1			1	7	2	1	40
8	oo	6	13				1	2			22
		10	18	2	2	3	2	2			39
9	$\overset{v}{oo}$	24									24
		40									40
10	0					8	9	4			21
					12	6	16	5		1	40
11	3					2	9	3		8	22
								4	33	5	42

* For each F_3 and testcross-F_2 line, the distribution tested at 2/18 is listed first.

Appendix Table V. (cont.)

		V oo	oo	0-	0	1-	1	2	3	4	
12	3				6	3	7	4	2		22
						2	3	11	13	10	39
(Rego^2 x P. I. 178383) - F_2 lines selected at 15/24											
13	4				1	6	5	8	1		21
					1	1	6	11	22	1	42
14	3						8	13			21
								7	35		42
15	oo	8	7			2		2		3	22
		8	19	2	1			5	5	3	43
16	0-	7	9	1		1	4				22
		6	23				2	2	8	1	42
17	oo-	1	14	2	3		1	1			22
		18	15				1	2	5		41
18	3				3	7	11	1			22
								4	37		41
19	4-					2	13	7			22
								12	35	1	38
20	0	5	13			1	2	1			22
		8	13	2	1			4	13		41
21	0	3	12			1	3	5			24
		7	20	2				2	6	6	43
(Rego x P. I. 178383) x Lemhi-F_2 lines selected at 15/24											
22	4									22	22
							1		7	33	41
23	0	2	3	6	8					3	22
		4	20	4	5				1	9	43
24	0-	2	4	10	3			1	1		21
		9	16	8	2	1	1			3	40
25	0	5	2	2	10					1	20
		8	10	12	9			2	2	1	44

Appendix Table V. (cont.)

		$\overset{v}{oo}$	oo	0-	0	1-	1	2	3	4	
26	4							3	1	17	21
									4	39	43
27	3+								1	21	22
									9	35	44
28	0	5	3	3	8	1			1	1	22
		10	5	7	7				5	5	39
F_3 lines selected at 2/18 in the F_2											
64	$\overset{v}{oo}$	13	17	4	4	1	2	3			44
		27	8				2	4	4		45
65	$\overset{v}{oo}$	9	13	3	4			2	7	6	44
		30	18					3	7	8	66
66	1-				5	3	10	14	2	7	41
					1		6	24	12	2	45
67	0		4	9	18	15	7	6	7		66
				4	20	6	8	5			43
68	0-	18	13	2	7		5				45
		23	9	1		1		3	4	3	44
69	$\overset{v}{oo}$	21									21
		41									41
70	0				33	8	3				44
				1	11	4	11	19	17		63
71	0				12	3	24	18	6	3	66
							1	7	24	11	43
73	$\overset{v}{oo}$	19	15				1	2	5	1	43
		23	9				1	1	3	6	43
74	0				4	5	10	12	11		42
							10	15	17		42
75	0				12	7	20	10	12	6	67
					1		1	11	28	1	42

Appendix Table V. (cont.)

		V oo	oo	0-	0	1-	1	2	3	4	
76	0-			4	28	9	4				45
					12	9	26	9	4		62
77	oo	14	21		3	3	4	1			46
		7	21		1	1	2	5	3	2	42
78	1				4	7	23	5			39
					1		2	20	17	2	42
Rego^2 x P. I. 178383-F_2 selected at 2/18											
79	V oo	10	8		2		2				22
		6	23	1				4	4	3	41
80	0			1	12	6	3				22
					5	1	4	9	22	1	42
81	1-				5	5	5	4	2	1	22
					2		2	8	11	19	42
82	oo	14	4			1			1	1	21
		9	17	4	1				5	4	40
83	1+			2	13	4	1				20
								15	25		40
84	V oo	3	12					2	3	1	21
		12	17						2	8	39
85	0-			2	6	4	9	1			22
								17	24		41
86	0				3		7	4	5	2	22
								3	33	5	41
87	oo	5	11	1	1	2	1	1			22
		7	22	4				1	6	1	41
88	2				5	3	4	3		5	20
								3	29	10	42
89	1				2		15	5			22
								6	32	2	40

Appendix Table V. (cont.)

		$\overset{v}{oo}$	oo	0-	0	1-	1	2	3	4	
90	0						3	12	6	1	22
								4	26	12	42
(Rego x P. I. 178383) x Lemhi-F_2 selected at 2/18											
91	0-		6	5	5			1	3	1	21
		3	8	9	6				3	10	39
92	0	1	4	4	5		3	3	1		21
		9	14	8	3				2	2	38
93	oo	7	12	1						2	22
		13	21	4						3	41
94	3							1		21	22
								4	7	50	61
100	$\overset{v}{oo}$	4	3	8	1			3	2	1	22
		7	19	14	14			3	3	8	60

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