THE GENETICS OF RESISTANCE TO *PUCCINIA GRAMINIS TRITICI* IN HEXAPLOID TRITICALE

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"THE GENETICS OF RESISTANCE TO

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FOREWORD

This thesis is written in paper format in order that the presentation of the author's research data would be in publishable form. The thesis is subdivided as follows:

SECTION I: General Introduction and General Literature Review. (Pages 1 - 44).

SECTION II: The paper form of the thesis, including Abstract, Introduction, Materials and Methods, Results and Discussion. (Pages 45 - 90).

SECTION III: General Discussion, Suggestions for Further Research and Summary and Conclusions (Pages 91 - 107).

SECTION IV: List of References and Appendix. (Pages 108 - 134).

It is intended that a somewhat modified version of Section II [including only divisions (i) to (vi) of the Results] be submitted for publication to the Canadian Journal of Genetics and Cytology.

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ABSTRACT

MORRISON, ROBERT JOHN. Ph.D. The University of Manitoba, October, 1976. THE GENETICS OF RESISTANCE TO *PUCCINIA GRAMINIS TRITICI* IN HEXAPLOID TRITICALE.

MAJOR PROFESSOR: E. N. Larter.

The inheritance of resistance to wheat stem rust, *Puccinia graminis* Pers. f.sp. *tritici* Eriks. and E. Henn., was studied in nine hexaploid triticale lines. Genetic studies were carried out using stem rust races C17 and C33; in addition, races C35, C10, C27, 111 and a rye stem rust isolate were used for further testing the spectrum of resistance present in the nine triticales. Field resistance was also studied. Several triticales were synthesized to determine the source of resistance.

Resistance was monogenically inherited in 70HN458, 6TA204, 6A413, and 6A250, digenically inherited in Rosner, MT36-1, Beaver, and 6A406, and trigenically inherited in 6A190. Resistance was dominant (with the possible exception of 6A406, in which partial dominance appeared to be operating) and the genes conditioning a fleck infection type to C17 and C33 were epistatic to those conditioning a type (2) infection type. No genic interaction was observed except in the case of 6A406 in which the resistance appeared to be due to at least two complementary genes. There appeared to be at least eight different resistance genes in these cultivars, five of which appeared to condition resistance to races C17, C33, C35, C10, C27, and 111, although this was not conclusively proven for all these races. The resistance conditioned by these five genes was clearly expressed in the field in the adult plant stage as well as in the greenhouse in the seedling stage. Of the remaining three genes,

one from 6A190 conditioned resistance to only C17, C35, C27 and race 111, but not C33 and C10. The two complementary genes in 6A406 conditioned resistance in the field to race mixtures, and in the greenhouse to all races tested except C10, while individually, the genes in 6A406 appeared to condition a mesothetic resistance to C17 and C33. In field tests, no additional adult plant resistance appeared to be operating in these seedling resistant cultivars (however, 6A20, a seedling-susceptible line used in the crosses, was resistant in the field).

The resistance genes appeared to be distributed into at least four linkage groups, the genes conditioning a fleck infection type from Rosner, 70HN458 and 6A190 appearing closely linked, allelic or identical, similarly, the genes conditioning a (2) infection type from Rosner, 6TA204, and 6A250. The two nondifferential genes of MT36-1 and 6A190 conditioning a moderate resistance each segregated independently (linkage relationships were not determined for the resistance genes of 6A406 and the differential gene in 6A190). Rosner, MT36-1, 6A413 and Beaver carried identical genes conditioning a (;) infection type; Beaver and MT36-1 carried identical genes conferring a moderate resistance.

The genes were temporarily designated genes 1 [Rosner, MT36-1, 6A413, Beaver, (;)], 2 [70HN458, 6A190, (;) (1^{-})], 3 [6TA204, 6A250, Rosner, (2)], 4 [MT36-1, Beaver, (2)], 5 [6A190 (2)], 6 [6A190 (2) differential], 7 and 8 [6A406, (;)].

The isolate of rye stem rust used was avirulent on the triticales and durums tested. The synthesis of new triticales indicated that both durum wheat and rye can contribute resistance to triticale, the level of the triticale resistance being equal to that of the more resistant parent. The results from the use of several different durums indicated

that the durums could contribute both wide-spectrum and narrow-spectrum resistance to triticale, while the one rye source of resistance used, Centeno, contributed only wide-spectrum resistance.

SECTION I

INTRODUCTION

AND

LITERATURE REVIEW

1. INTRODUCTION

After two decades of intensive research and development, hexaploid triticale, *X Triticosecale* (Wittmack), has progressed from the plant breeders' plots to commercial farm production. At present, it is being tested in many areas around the world, including North America, Europe, North and East Africa, India, Iran and Chile. Most of the disease observations on triticale have come from small plots and greenhouse experiments. Triticale is also being grown commercially in large areas in a few scattered locations in the U.S.A. and Europe. However, the acreages and time period are still too limited to allow adequate observations on epidemic development within the crop and on the longevity of triticale resistance (Richardson and Waller, 1974).

From small plot observations, researchers have observed in triticale good resistance to wheat stem rust, *Puccinia graminis* Pers. f.sp. *tritici* Eriks. and E. Henn., even in East Africa where stem rust is a severe problem in wheat (Pinto, 1974; Wabwoto, 1974). This is in contrast to the situation for leaf rust, *Puccinia recondita* Rob. ex. Desm., which appears to be a more serious problem in triticale than in wheat (Zillinsky, 1974b).

Certain raw amphidiploid triticales and advanced lines are susceptible to stem rust, however. Lopez (1971) attempted to identify the *formae speciales* of stem rust able to attack triticale. The isolates which were virulent on certain triticales appeared characteristic of wheat stem rust. Several triticales tested by Lopez, however, exhibited good resistance to all the stem rust isolates that he collected from wheat, rye and triticale in Mexico.

Both the tetraploid wheats and diploid rye have contributed

important stem rust resistance genes to bread wheat in breeding programs in Europe and North America (Allard, 1960; Zeller, 1973). Combined into triticale, the tetraploid wheat and rye resistances should be able to reinforce each other. It has been pointed out that the use of several otherwise unused resistance genes in a cultivar presents a difficult barrier for the pathogen to overcome (Watson and Singh, 1952; Knott, 1972). Green (1971a) has pointed out that varieties derived from interspecific crosses may redirect pathogen evolution towards more non-aggressive forms, because of the difficulty of broadening the host range while maintaining the destructiveness.

Because stem rust is such a destructive pathogen and because it is able to evolve new virulent forms fairly easily, it is important to understand whether or not triticale resistance is complex. It can be expected that extensive use of triticale will shape evolution of the pathogen and in this case the plant breeder must be able to anticipate what will be the most effective way to use triticale resistance.

The present study attempts to clarify the inheritance, nature, variety and source of triticale resistance through determining the number of genes per variety and the genes common to several varieties, the range of the resistance and the contributions of the rye and wheat progenitors. It is hoped that such information combined with an understanding of present knowledge on host-parasite systems can indicate some of the problems that may be encountered with stem rust when triticale is grown over large acreages.

2. LITERATURE REVIEW

2.1. The Nature of the Host, Triticale, and its Progenitors

2.1A. Triticale

(i) Genetic Constitution

Triticale is the common name of *X Triticosecale* (Wittmack), the amphidiploid between wheat and rye. This genus, originally found in nature only as an occasional sterile polyhaploid hybrid but now being domesticated as a crop plant through artificial doubling, crossing and selection, has been under scientific investigation for over a century. Intensive efforts towards development as a crop species did not occur until the latter half of this period, however. The earlier efforts centred on the study of octoploid forms of triticale, but the development of hexaploids shifted the emphasis, as these appeared to present more potential as a crop species.

Present-day varieties of triticale emerging from breeding programs are generally hexaploid. The main species presently being used for hexaploid triticale synthesis are the tetraploid wheats, particularly *Triticum durum* L., and the diploid ryes, *Secale cereale* L. and *S. montanum* Guss. (Scoles and Kaltsikes, 1974). Considerable introgression of germ plasm from hexaploid wheat (*T. aestivum* L. em. Thell.) into hexaploid triticale has occurred in breeding programs (Merker, 1975). Octoploid triticales are derived from *T. aestivum* and *Secale* spp.

Hexaploid triticales may be classified as being either primary or secondary hexaploids. Primary hexaploid triticales are derived from tetraploid wheat x rye crosses through direct synthesis ("raw" amphiploids) and subsequent interbreeding and selection (advanced

triticales). Their genomic constitution is AABBRR. Secondary triticales are derived from either octoploid triticale x hexaploid triticale crosses or hexaploid triticale x hexaploid wheat crosses. Secondary triticales include a range of genetic types, from lines with a complete AABBRR constitution — i.e., a full rye complement — to lines with as few as one pair of rye chromosomes, in which case D genome chromosomes substitute for the missing rye chromosomes (segmental or substitutional polyploids).

In the case of secondary triticales, the triticale morphology is carried mainly on the two rye chromosomes B (3R)* and F (5R) (Merker, 1975). When both are present, the plant is a typical triticale. The presence of either one alone produces a wheaty-type triticale. Their absence leads to classification as wheat [wheat lines carrying one or two other pairs of rye chromosomes are used in the Mexican and European wheat breeding programs (Mettin *et al.*, 1973; Zeller, 1973)]. Also, certain wheat backgrounds suppress the rye characters of chromosomes B and F, leading to classification as wheat.

Nevertheless, the appropriate classification of hexaploid lines with three or four pairs of rye chromosomes is still unclear; no scheme exists for classifying them as *Triticum* or *Triticosecale* (Merker, 1975).

The lines termed Armadillo have been the foundation of the CIMMYT triticale program (Zillinsky, 1974a) and these appear to carry a 2D-2R substitution (Gustafson and Zillinsky, 1973; Merker, 1975). Merker (1975) further detected the loss of 2R in Beaver and Rosner, the latter

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* Chromosome identification according to Merker (1975) and Gustafson (personal communication).

hitherto thought of as being a pure triticale. Merker studied about 50 lines from the CIMMYT breeding program and found that both rye chromosomes A (2R) and D (4R) were commonly lacking as well as a third rye chromosome in many cases. Most of the lines he studied seemed to have retained chromosomes E (6R) and G (1R). The German wheats described by Mettin *et al.* (1973) and Zeller (1973) carry 1R-1B substitutions or translocations.

(ii) Agronomic Characteristics

A typical primary hexaploid triticale such as Rosner is characterized by bearded, rough-awned mid-dense spikes, with long-beaked, acuminate chaff and pubescent necks, and by wrinkled kernels (Larter *et al.*, 1970). Merker (1975) points out that the typical triticale characteristics are carried mainly by two rye chromosomes, B (3R) and F (5R).

Early primary triticales were also characterized as being tall, long-spiked, only partially fertile, late and daylight-sensitive (Larter, 1968; Larter *et al.*, 1968; Quinones, 1972). With the introgression of germplasm from bread wheat, especially D genome chromosomes (via cultivars such as Armadillo), and the loss of some rye chromosomes (eg. the 2D-2R substitution of Armadillo), secondary triticales were developed with reduced stature, improved fertility, improved seed type, earliness, and daylight-insensitivity (Zillinsky, 1974a). Dwarf durums and ryes are also being used to improve triticale (Larter, 1974b).

Although synthesis of triticale has the effect of converting the heterozygous outbreeding rye component to a homozygous inbreeding one, hexaploid triticale has retained some of the outcrossing characteristics. Hexaploid triticale tends to exhibit a greater degree of anther extrusion

than wheat, a higher pollen count per anther, and also a longer flowering period. Rosner exhibits approximately 5% outcrossing at the University of Manitoba (Yeung and Larter, 1972).

During early breeding programs, triticale was found to exhibit some sensitivity to stress. In testing a number of primary triticale lines, Larter *et al.* (1968) reported a decrease in meiotic stability, with increase in temperature. Hot, dry conditions during flowering in the prairie region of Canada induced considerable sterility, and thus yield reductions, in triticales grown in 1967 (Larter, 1968). The early Armadillo lines appeared to flower and mature too quickly when stressed by hot dry conditions early in the season. Efforts are being made in the CIMMYT and University of Manitoba programs, however, to improve the adaptability of triticales (Zillinsky, 1974a,b) and average yields have been steadily increasing (Larter, 1974a).

At present, advanced lines of triticale appear to be performing best under areas of stable moderate climate with adequate rainfall or irrigation, including marginal soil areas and environments favourable for disease. Often, these are areas where wheat and barley do poorly. Thus, Kiss (1974) reported triticales doing well in Hungary on the intergrade sandy soils between the heavy wheat soils and poor light rye soils (rye normally lodges on the intergrade soils due to luxuriant growth); Srivastava (1974) suggested a potential usefulness of triticale on Indian foothill soils with low productivity due to pH problems; Wabwoto (1974) indicated that triticale can be grown successfully in the areas of severe rust epidemics in E. Africa.

Zillinsky (1974a,b) made the observation that triticales were competitive with other cereals in three main areas, i.e., regions with:

(1) sandy soils/moderate rainfall: E. and W. Europe and Mexico (on rye soils).

(2) high elevation/high to moderate rainfall: Himalayan foothills, E. Africa, Columbia, Mexico.

(3) low growing temperatures: central Europe, south U.S.A. (late fall and winter crops).

(iii) Disease Resistance

Larter (1974b) has pointed out that diseases have not yet been limiting to triticale production, though most wheat diseases can attack some triticales. In Hungary, where significant acreages of triticale are grown, the leaf diseases (*Septoria* spp., and *Helmintho-sporium* spp.), ergot [*Claviceps purpurea* (Fr.) Tul.] and Fusarium wilts are of importance in triticale, whereas the leaf and stem rusts, powdery mildew and smuts are unimportant under Hungarian conditions (Kiss, 1974). In western Canada, leaf rust (*Puccinia recondita* Rob. ex. Desm.) and ergot have been the principal problems (Larter *et al.*, 1968).

Lopez (1971) reported that wheat stem rust (*P. graminis* Pers. f.sp. *tritici* Eriks. and E. Henn.) and rye stem rust (*P. graminis secalis*) could attack certain triticales, but Larter (1968) pointed out that most strains of triticale are resistant to stem rust.

Though leaf rust in triticale is more of a problem than stem rust, adequate resistance is available (Larter, 1975b). Ergot has been a very serious problem for triticale in some areas because of the susceptibility of triticale and toxicity of the sclerotia produced by the fungus. This disease has been dealt with mainly through improvements

in fertility, although attempts are presently being made to transfer resistance from *T. timopheevi* Zhuk., one of the few good sources of resistance (Larter, 1974b).

Triticale has also been found to be parasitized by stripe rust [*P. glumarum* (Schm.) Eriks. and E. Henn.], bacterial blight [*Xanthomonas translucens* (Jones, Johnson, and Reddy) Dowson], snow mold (*Fusarium nivale* (Fr.) Ces.), loose smut [*Ustilago tritici* (Pers.) Rostr.] and barley yellow dwarf virus. Triticale tends to be resistant to powdery mildew (*Erysiphe graminis* DC.) (Larter, 1974b) and the more recent triticales from CIMMYT have stripe rust resistance (Zillinsky and Borlaug, 1971). Resistance is available against bacterial blight but the situation is unclear for snow mold (Larter, 1974b). Triticale is generally resistant to loose smut (Nielsen, 1973). Gardner *et al.* (1969) report a wide spectrum of resistance to wheat streak mosaic virus and Qualset *et al.* (1973) report a wide range of susceptibility to barley yellow dwarf virus, but neither disease appears to be a problem (Larter, 1974b). Triticale appears to exhibit resistance to *Septoria* diseases in North Africa and South America, where *Septoria* can be a severe problem on wheat (Richardson and Waller, 1974).

Sanchez-Monge (1959) pointed out that triticale usually seems to express the level of resistance of the more resistant of the wheat and rye parents. It would seem that the resistance of triticale to disease will prove significant in its potential utilization, as a single rye chromosome substitution or translocation is already providing important resistance in a number of European wheat varieties to stem rust, leaf rust, stripe rust, and powdery mildew (Mettin *et al.*, 1973; Zeller, 1973).

(iv) Chromosomal Behavior

Mitosis. Shkutina and Khvostova (1971) have reported mitotic irregularities in triticale; fragments, lagging chromosomes, micronuclei, bridges and tri-polar spindles were observed in root tip cells of both hexaploid and octoploid triticale. Few other mitotic studies have been done with this species. Scoles and Kaltsikes (1974) suggested that it is unlikely that triticale differs from wheat in the degree of mitotic irregularity.

Meiosis. Some degree of meiotic irregularity is present in most triticales (Scoles and Kaltsikes, 1974). The meiotic instability is expressed as the appearance of univalents at metaphase, and subsequent loss of these to produce aneuploid megaspores (and microspores, although these cannot compete with euploid microspores). As a result a considerable proportion of triticale progeny may be aneuploid, the fertility and vigour of these plants often being reduced. Tsuchiya (1974) reported the average proportion of aneuploids as 40% in euploid octoploid progenies and 10% in euploid hexaploid progenies, with bulk populations averaging 63% and 15% aneuploids for octoploids and hexaploids, respectively. For hexaploids, Tsuchiya cited figures of 24-47% normal sporocytes, with an average of approximately 1-3 univalents/PMC (with a range of 0-28), for euploid plants.

In octoploids, it is predominantly the rye chromosomes that form univalents. Octoploids tend to revert to bread wheat. In hexaploids, both wheat and rye chromosomes are lost, in proportion to their numbers (Scoles and Kaltsikes, 1974).

Scoles and Kaltsikes cited figures of 16-83% of euploid octoploid progenies being aneuploid, with 38-100% disturbed pollen mother cells.

For euploid hexaploids, they cited a range of 1.5-17% aneuploid progeny, with up to 57% of some bulk populations being aneuploid. They cited ranges of disturbed cells at metaphase of 30-70% for primary and advanced hexaploid triticales and 8-22% for secondary hexaploid triticales, with the range of univalents per cell being 0-14, and the average approximately 2 univalents/cell for primary and advanced triticales.

The cultivar Rosner averages approximately 2 univalents per cell and approximately 50% abnormal metaphase cells (Scoles and Kaltsikes, 1974).

It appears that univalent formation in hexaploids is due to a gene interaction between the wheat and rye genomes, while in octoploids it appears due to meiosis occurring too rapidly for the rye chromosomes to synapse properly (Scoles and Kaltsikes, 1974).

No correlation exists between fertility and meiotic stability in triticale, due to the high survival rate of aneuploid megaspores (Scoles and Kaltsikes, 1974).

Various workers, including Müntzing (1959), have reported that F_1 hybrids between triticales exhibit more meiotic irregularities than their parents.

2.1B. Wheat

As wheat contributes germplasm to triticale and will no doubt be grown in close proximity to triticale in many areas, useful examination may be made of the distribution of wheat disease resistance between wheat classes, and in turn, the geographical distribution of these classes.

T. durum L. has been one of the main contributors of germ plasm to triticale, with a considerable amount of infusion of *T. aestivum* germ plasm as well. Vavilov (1914) generalized that the monococcums were the most resistant to the rusts and powdery mildew, durums less so and aestivums most susceptible. The durums are grown in a number of areas where triticale could potentially be grown.

(i) Durum Distribution

Durum wheat is grown mainly in north central U.S.A. and adjacent areas of Canada, around the Mediterranean (Spain, Italy, France, the E. Mediterranean area, and N. Africa), Iran, Turkey, Ethiopia, the Soviet Union, India and Argentina. Durum predominates over hexaploid wheat in Tunisia, Algeria, Morocco, S. India and Turkey, and is important in the Middle East and E. Mediterranean countries and Ethiopia. It is unimportant in China, Pakistan, Egypt, Kenya, S. Africa and Australia (Reitz, 1967; Mangelsdorf, 1953).

About 1/5 of the spring-sown wheat in the U.S.S.R. is durum. American durum production has varied between 3-9% of the American wheat acreage between 1919 and 1964. 85% of American durum is grown in N. Dakota, the other main areas being S. Dakota, Minnesota, and Montana. Canada's durum production is mainly in Saskatchewan (Kipps, 1970). The durums are generally grown in the drier areas of the U.S.A. spring wheat area (Dickson, 1956) and are able to grow well under dry conditions (Morris and Sears, 1967).

(ii) Durums and Stem Rust

The durums (and tetraploid wheats in general) have been important in N. America in dealing with stem rust. Iumillo durum, and Vernal emmer provided important resistance to N. American wheat varieties — Iumillo to Thatcher, and Vernal emmer to Stewart and Carleton durums (Allard, 1960). Iumillo durum retained its resistance for 50 years in the U.S.A. and Vernal emmer 75 years before succumbing to race 15B (Martin and Salmon, 1953). The stem rust resistant durums widely replaced N. American hexaploid wheat (Marquis) from 1916-1923 due to the evolution of races able to attack Marquis (Stakman and Harrar, 1957).

However, the stem rust pathogen in N. America has evolved virulence on the durums several times. The first successful American durum, Mindum, succumbed to stem rust upon the appearance of races 17 and 38. Stewart and Carleton (Mindum derivatives) then were released in 1943 and grown until the appearance of race 15B in 1953 and 1954, which devastated the durum crop because the later maturation of the durum crop allowed greater rust development (65-75% of the American durum crop was lost). Resistance from Ethiopian durum St. 464 was incorporated into Stewart, resulting in the release of Stewart 63 in Canada. In the U.S.A., Langdon, released in 1956, replaced Stewart but succumbed to a new biotype of 15B. Wells and Lakota then were released in 1960 and replaced Langdon but virulent races then evolved for these varieties. In Canada present durums include Hercules, Macoun, Wakooma and Wascana (Stakman and Harrar, 1957; Loegering *et al.*, 1967; Horsfall *et al.*, 1972; Buchanan *et al.*, 1974).

(iii) Durums and Leaf Rust

Chester (1946) pointed out that the durums were generally resistant to leaf rust and the bread wheats generally susceptible. Quinones (1972) pointed out that evolution of the leaf rust pathogen in N. America appears to have been mainly on hexaploid wheats, so that present N. American durums are resistant (Rajaram and Sartari, 1971). Dickson (1956), as well, pointed out that nearly all durums are resistant to leaf rust.

In the south of France, the durums grown do not carry sufficient leaf rust resistance (Grignac, 1974). The old cultivated Italian durums are very susceptible to stem and leaf rust (Zitelli, 1973). However, although many are susceptible to stem and leaf rust, a number of Iberian durums do represent important sources of resistance (Salazar *et al.*, 1973).

(iv) Bread Wheats, Stem Rust and Leaf Rust

In winter wheat areas in the U.S.A., varieties resistant to stem rust are not particularly needed. Rust damage is minimal due to early maturity of the crop and the existence of unfavourable conditions for rust development (stem rust is most adapted to hot weather). However, race 56 did cause heavy damage to winter wheats in 1961 and 1962 (Dickson, 1956; Loegering *et al.*, 1967).

The spring wheats initially introduced into N. America had little stem rust resistance and as new resistance genes were incorporated into the spring wheats, the stem rust pathogen evolved to overcome them (Stakman and Harrar, 1957). This same pattern has occurred in other areas such as Australia (Allard, 1960).

Chester (1946) pointed out that the bread wheats were generally susceptible to leaf rust.

(v) Infection Chimaeras in Wheat

In disease studies in bread wheat, the mitotic loss of a chromosome has been postulated as the cause of the occasional chimaeral reaction to a disease. McIntosh and Baker (1969) noted three examples in the progeny of an F_1 hybrid monosomic for a chromosome carrying gene *Pm3a* (from the cross Chinese Spring 1A x Asosan) conditioning resistance to powdery mildew. These three plants had leaves with adjacent sectors of resistant and susceptible tissue, with the midribs appearing as the division line between the two classes. It was assumed that the monosomic resistance-carrying chromosome was lost early in the embryonic development of the plant.

Baker (Day, 1974) observed a similar situation for *P. graminis tritici* when testing Gabo wheat (2n = 43) carrying a single *Agropyron elongatum* (Host) Beauv. chromosome conditioning resistance, the *Agropyron* univalent presumably having been lost through misdivision.*

2.1C. Rye

Since rye is a parent of triticale, a review of rye's geographical distribution and disease reaction was deemed necessary.

(i) Distribution

Rye, primarily a winter crop and once the most common bread grain of Europe and Russia, is now a minor crop, exceeding wheat production only in Poland (Scoles, 1975). It is thought to have been originally introduced to Europe and Russia as a weed in wheat and barley, gradually becoming

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* Burrows (1970) also noted leaf chimaeral reactions to crown rust, *P. coronata* Cda, in trisomic oat plants carrying the gene *Pc15* for resistance.

domesticated and grown widely (Halbæk, 1971). Its acreage declined after the 17th century, due to several factors; increased affluence was one important factor contributing to the shift to the preferred wheat bread (Horsley, 1969).

The U.S.S.R. is presently the major rye producer, Europe being second, and the U.S.A., Argentina and Turkey producing small amounts. In 1961-63, nearly 90% of world rye production was in the Soviet Union, Poland, W. Germany, E. Germany and Czechoslovakia (Martin and Leonard, 1967). In 1969, 39% of world of rye production was in the U.S.S.R. and 37% in Eastern Europe (Carmichael and Norman, 1970).

In Canada, approximately 50% of the total rye production is in Saskatchewan. 89% of Canada's rye production in 1969-70 was from the Prairie Provinces. 85% of the rye grown in Canada is fall rye (Carmichael and Norman, 1970).

In the U.S.A. rye is grown primarily in the area just north of the winter wheat area, mainly in the Dakotas and Nebraska. Small amounts are also grown for pasture in the east and southeast U.S.A. (Martin and Leonard, 1967; Kipps, 1970).

Rye is generally more productive than other grains on infertile, sandy or acid soils and thus tends to be grown on marginal land (Carmichael and Norman, 1970).

(ii) Disease Resistance

The limited acreage of rye in most areas as well as rye's early maturity tend to minimize its disease problems. Ergot has been the most serious disease of rye, while the various leaf diseases are common in the humid winter rye areas. Leaf and stem rusts present only an occasional problem.

P. graminis secalis generally causes little damage to rye, but in Germany economic loss from this rust is often serious (Arthur, 1929). In the U.S.A. stem rust damage on rye is rare (except for the occasional infestation near barberry) because of rye's earliness and the lack of rye in the south where inoculum could build up (Martin and Salmon, 1953). Some grain and pasture ryes in the U.S.A. do carry resistance, however, such as Gator rye in Florida (Martin and Leonard, 1967). Stewart *et al.* (1968) have pointed out that Prolific spring rye is very susceptible to rye stem rust.

Leaf rust of rye is abundant in N. America and Europe and occasionally causes losses (Arthur, 1929). The early maturity of rye does tend to minimize these losses, however. Rye leaf rust overwinters on winter rye as dormant mycelium and causes losses most often in the southern range of American rye culture, where the fungus overwinters in greater abundance (Dickson, 1956; Martin and Leonard, 1967). It may occasionally become sufficiently abundant on fall rye pasture crops to cause winter killing (Martin and Salmon, 1953). The varieties Gator (in Florida) and Explorer (in the southern U.S.A.) carry leaf rust resistance (Martin and Leonard, 1967).

2.2. The Nature of the Pathogen, Stem Rust

Craigie (1957) has noted that of the cereal rusts, stem rust is the most dreaded, the losses from it far exceeding that from any of the others. The fungus interferes with the main translocation system of the plant for water and nutrients, disrupts protective and productive surface tissues and weakens the straw, resulting in reduced yields and quality. The disruptive effect on individual plants is compounded by the pathogen's adaptations enabling several cycles of spore production in a season and widespread dissemination by the wind. Widespread monoculture of the host, combined with the ability of the pathogen to evolve virulence on widely-used resistance genes, hampers efforts to block the pathogen (Craigie, 1957).

To become widespread, isolates of the pathogen must acquire fitness or aggressiveness characteristics, as well as virulence. Fitness attributes such as a short incubation time, the capacity to infect and sporulate under a broad range of climatic conditions, high rate of spore production and a high rate of infection contribute to an increased rate of disease spread (Day, 1974).

In many countries, *P. graminis* is able to cycle throughout the year by asexual reproduction on cultivated cereals and wild grasses (the uredial stage), occasionally going through sexual recombination on barberry, *Berberis vulgaris* L. (aecial stage). The uredial stage has become specialized into a number of varieties, or *formae speciales*, restricted to particular hosts. Thus, *P. graminis tritici* is found mainly on wheat, *P. graminis secalis* on rye and *P. graminis avenae* on oats (Craigie, 1957).

P. graminis tritici and *secalis* appear to be closely related, as they both can attack cultivated and wild barley (*Hordeum vulgare* L. and *Hordeum jubatum* L., respectively) and certain wheat and rye lines, and can hybridize fairly easily (Watson and Luig, 1962; Green, 1971a; Lopez, 1971). However, most wheats are resistant to rye stem rust and most ryes to wheat stem rust. *P. graminis secalis* also differs in that it is common on couch or quack grass [*Agropyron repens* (L.) Beauv.].

2.2.A. Different Host Species - Pathogen Variety Interactions

(i) Wheat Stem Rust on Wheat

Wheat stem rust is prevalent on wheat and often causes serious damage, although numerous sources of resistance exist. Useful control has been achieved through the use of resistant varieties but breakdowns in this resistance have been costly (Robinson, 1971). Craigie (1957), Hooker (1967) and Nelson (1973) have reviewed much of the information on this area.

(ii) Wheat Stem Rust on Rye

Wheat stem rust has occasionally been observed on rye, especially when it is adjacent to heavily rusted wheat fields (Cereal Rust Laboratory, 1972), However, wheat stem rust is generally avirulent on rye (Craigie, 1957). Stewart *et al.* (1968) found Prolific rye to be highly resistant to the three races of wheat stem rust they used. Green (1971a) pointed out that Rosen rye usually exhibits type (1) infection* with wheat stem rust and type (4) infections with rye stem

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* See Appendix III for classification system.

rust. Lopez (1971) obtained three different isolates representing three races of wheat stem rust which were generally virulent on the wheats that he tested and which were able to attack certain ryes. One race attacked an Explorer rye inbred line and Prolific, another attacked Gator rye and the third, a Gator rye x Wrens hybrid selection. A different Explorer rye inbred was susceptible to all three wheat stem rust races.

(iii) Wheat and Rye Stem Rust on Barley

Most barleys are susceptible to wheat and rye stem rust but these are not a threat because barley matures early and because they are not aggressive on barley (Johnson and Buchannon, 1954; Green, 1971a). A few barley varieties have resistance to wheat stem rust (e.g. Peatland, Conquest) but no commercial barley varieties are resistant to rye stem rust (Green, 1971a, 1975). Genes *T* and *T2* in barley condition resistance to some races of *P. graminis tritici* but not to *P. graminis secalis* (Roane, 1973). Rye stem rust has never seriously damaged barley in western Canada (Green, 1971a).

(iv) Rye Stem Rust on Wheat

Rye stem rust is generally avirulent on wheat (Craigie, 1957). Lopez (1971) tested 10 rye stem rust isolates on a large number of wheats and generally obtained at most a fleck infection type and occasionally a type (1) infection. An exception was found in that one isolate could attack one durum wheat, one bread wheat (Yalta) and seven of the bread wheat differentials. Luig *et al.* (1973) reported that the seedling resistance [infection type (2)] of Little Club wheat to *P. graminis secalis* is due to a single factor. Little Club wheat generally exhibits a type (4) to wheat stem rust.

Watson and Luig (1962) were able to obtain rye stem rust strains virulent on wheat by selfing rye stem rust cultures. In their experiments, Little Club wheat exhibited a type (2≡) infection to rye stem rust, other wheats mostly fleck infection types, with an occasional type (2^-) infection and a rare type (3^-) infection. Through selfing, they obtained a culture which could produce a type (3^-) infection on several wheats and a type (3^+) infection on Little Club. The culture, however, was more vigorous on rye, especially when temperatures were low.

Sanghi and Luig (1971) mention that certain wheats (e.g. W2691) are moderately susceptible (2^{++}3c) to rye stem rust. In studying the genetics of wheat resistance to rye stem rust, they found that *Sr8* in Mentana wheat conditions a type (2) infection to both wheat and rye stem rust, while *Sr11* in Yalta conditions a type (;)(2≡) infection to both wheat and rye stem rust. As well, Mentana carried three other genes conditioning resistance to only rye stem rust and a fifth gene conditioning resistance to two avirulent *secalis-tritici* hybrids and race 111 of wheat stem rust but not rye stem rust. Yalta also carried another gene for resistance to rye stem rust but not wheat stem rust, as well as two more genes for resistance to an avirulent *tritici-secalis* hybrid and an avirulent wheat stem rust race. However, the *Sr11* gene proved ineffective against two hybrid stem rusts and an avirulent wheat stem rust (Race 111).

(v) Rye Stem Rust on Rye

Rye stem rust is generally virulent on most ryes. Lopez (1971) tested ten rye stem rust isolates on 31 ryes and detected only occasional

resistance, this being for only a few isolates. Prolific, Gator and Explorer inbreds were generally susceptible. Stewart *et al.* (1968) likewise found Prolific very susceptible to rye stem rust. Green (1971a) noted that Rosen rye generally exhibits an infection type (4) to rye stem rust.

Nevertheless, resistance has been noted in rye to rye stem rust. Mains (1926b) noted stem rust resistance in Abruzzes rye. Levine and Stakman (1923) reported that Rosen, Swedish, and Prolific varied in their degree of susceptibility to different isolates of rye stem rust. Cotter and Levine (1932) noted that Rosen, Swedish, Prolific, Dakold, Colorless, and Giant Winter ryes differed in their resistance. Using these varieties as differentials they were able to distinguish 13 races in the U.S.A., two of which were widely distributed across the northern U.S.A. over a period of 9 years. These races were avirulent on the wheat differentials.

In Canada, rye stem rust is widely distributed, often appearing on rye in rust detection nurseries and sometimes in barley (Green, 1972). However, its prevalence varies considerably from year to year,especially on the prairies where barberry is unavailable. The earliness of rye, and limited acreage, have prevented serious damage in Canada (Johnson and Buchannon, 1954). Rye stem rust tends to be more widespread in eastern than western Canada.

On the Canadian prairies, overwintering of the uredial stage on couch grass is the most likely source of annual infection (Johnson and Buchannon, 1954). Under Australian conditions, rye stem rust is of little agricultural importance, although it is widespread on the common *Agropyron repens* (Watson and Luig, 1962). In Germany, where large

acreages of rye are grown, severe infestations have been known to occur (Arthur, 1929). In the U.S.A. severe attacks may occasionally occur near barberry (Martin and Salmon, 1953) but damage is generally rare due to the early maturity of rye.

(vi) Stem Rust on Triticale

Lopez (1971) collected[1] stem rust from triticale, bread wheat and rye and increased the isolates for screening tests. The rust attacking triticale in the field was clearly *P. graminis tritici*. Nine out of 10 isolates of rye stem rust could not attack triticale or wheat. A tenth isolate of rye stem rust was able to attack one durum wheat, one bread wheat variety, seven of the wheat differentials, nine of the 38 hexaploid triticales and none of the octoploid triticales tested. It was suggested that the isolate was a recombinant between *tritici* and *secalis*.

The three races of wheat stem rust Lopez obtained from wheat were virulent on most of the 14 wheats tested, on two to three of the five ryes tested, on most of the six octoploid triticales, and only on a minority of the hexaploid triticales. The behaviour on wheat and rye was typical of that established for *P. graminis tritici* by Stakman (1917) and Stakman *et al.* (1918). For the six octoploid triticales*, five were susceptible to two of these races and two of these five were susceptible to the third race. Of the 31 hexaploid triticales screened, all were resistant to one race and all but five were resistant to the other two races.

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* Five of the six octoploids were derived from one wheat, Inia 66.

1 In Mexico.

The 19 isolates Lopez collected from triticale could be classified as wheat stem rust as well. The international bread wheat differentials were largely susceptible to these isolates. The five ryes tested were resistant to eight of these isolates but from 1-4 of these ryes were susceptible to each of the other 11 isolates. All of the octoploids could be attacked by several isolates. Twenty of the 31 hexaploid triticales tested were resistant to all triticale-derived isolates and the other 11 were susceptible to one to four of the isolates.

Of the 20 resistant hexaploid triticales mentioned, 15 were resistant to all isolates collected from wheat, rye and triticale (these 15 included 11 Armadillo selections). Of the remaining five, one could be attacked by one of the races collected from wheat and the other four (including one Armadillo) could be attacked by the widely virulent rye stem rust selection.

Of the ten isolates collected from rye, all were virulent on virtually all of 31 ryes, and nine were avirulent on the 12 differentials, 11 bread wheats, ten durums, 38 hexaploid triticales and 6 octoploid triticales. The tenth isolate, while still virulent on virtually all 31 ryes, was also virulent on several wheats and triticales.

2.2B. Hybridization

Because both *P. graminis secalis* and *tritici* are found on barberry, wild barley and cultivated barley, there is ample opportunity for hybridization through sexual or asexual means (hyphal anastomosis) (Watson and Luig, 1959). In Australia, what appear to be probable *tritici-secalis* hybrids are widely distributed on wild grasses (Sanghi and Luig, 1971; Luig and Watson, 1972).

By crossing *tritici* and *secalis* on barberry, Green (1971a) obtained F_1 and F_2 hybrid isolates, which were mostly avirulent on wheat or rye or both. Green ascribed this to (a) the recessive virulence genes being masked by dominant avirulence genes in the hybrid, and (b) a large number of factors governing virulence on wheat and rye, hence the difficulty in recovering a virulent combination in the F_2 generation. Although avirulent on wheat and rye, the F_1 and F_2 isolates were still able to infect barley.

The few of Green's hybrids which could infect both wheat and rye were intermediate in reaction type between the parent isolates. Thus, broader host range had been achieved at the expense of some degree of virulence on one of the hosts.

However, Sanghi and Luig (1971) found that certain *tritici-secalis* hybrids had acquired virulence against *Sr11* (although the hybrids were otherwise avirulent). They suggested that natural hybridization could potentially overcome wheat stem rust resistance in wheat, and also the resistance transferred from rye to wheat. Nevertheless, the probable hybrids they had observed on wild grasses were avirulent on wheat and rye.

Typical of the races recovered from *tritici* by *secalis* crosses was race 111 of *P. graminis tritici*, which will infect only Little Club wheat in the standard differential set (Johnson, 1949).

2.2C. Evolution of *Formae Speciales*

Watson and Luig (1959, 1962) suggested that rye stem rust is simply an avirulent strain of wheat stem rust, and pointed out that *secalis* harboured heterozygous recessive virulence genes on wheat which

could be revealed by selfing.

Green (1971a) suggested that these *formae speciales* evolved from unspecialized forms, by the accumulation of virulence genes for certain hosts at the expense of virulence on others. He suggested that the original ancestral forms had a wide host range but were low in virulence and aggressiveness, presumably because of a generalized resistance in the host population. Presumably, as the host population evolved and diverged, the rust pathogen specialized on the divergent parts of the population, developing high virulence and aggressiveness on a limited number of hosts by overcoming the vertical resistance genes that they had in addition to a generalized resistance.

Thus, present day *tritici-secalis* hybrids would be expected to resemble the ancestral type, with wide host range, low virulence, little specialization, and low aggressiveness (as with the barley-stem rust relationship).

Green further suggested that the evolution of the pathogen could be reversed to these nonaggressive avirulent forms by the use of broad combinations of resistance, i.e., interspecific and intergeneric crosses. The pathogen, in effect, would be forced to broaden its host range, and would likely have to sacrifice aggressiveness or virulence to do it.

2.2D. Leaf Rust in Comparison to Stem Rust

P. recondita clearly differs from *P. graminis* in a number of ways:

(a) wheat and rye stem rust share the alternate host, barberry, while wheat and rye leaf rust have two different alternate hosts. The leaf rust alternate hosts are meadow-rue (*Thalictrum* spp.) for *tritici* and bugloss (*Lycopsis* spp. and *Anchusa* spp.) for *secalis* (Arthur, 1929;

Johnson and Newton, 1946; Martin and Salmon, 1953).

(b) wheat and rye stem rust can both infect barley but leaf rust cannot (Johnston, 1936), to any significant extent.

(c) The stem rust sexual stage is important in epidemic development in some areas while the leaf rust sexual stage is inconsequential (Jackson and Mains, 1921).

(d) stem rust disrupts the plant more than leaf rust. Leaf rust damage is confined to the leaves whereas stem rust infects both stems and leaves (Loegering *et al.*, 1967).

(e) stem rust does not thrive as well as leaf rust in cooler temperatures (Loegering *et al.*, 1967).

The 68 rye lines and varieties that were tested by Mains (1923) were essentially susceptible to rye leaf rust. However, Mains (1926b) obtained rye plants from Abruzzes rye which were highly resistant to rye leaf rust. Mains (1926a) reported two physiologic races of rye leaf rust in the U.S.A. and Waterhouse (1939) two in Australia. Rye leaf rust is abundant in N. America and Europe and occasionally causes losses (Arthur, 1929). Gator and Explorer ryes in the southern U.S.A. carry leaf rust resistance (Martin and Leonard, 1967). Severe infections are largely confined to the southern range of rye culture in N. America, especially where rye is used for winter pasturage (Martin and Salmon, 1953; Dickson, 1956). The early maturity of rye tends to restrict damage.

Wheat leaf rust is more common and severe on bread wheat than other wheat species (Loegering *et al.*, 1967).

2.3. Host-Parasite Relationships and Resistance

Nelson (1973) classifies disease resistance as an active dynamic host response to a parasite, in which an incompatible interaction interferes with the pathogen's attempt to withdraw sustenance, resulting in a lower level of disease. Host resistance must be related to two pathogen characters, virulence and aggressiveness. Virulence is the ability of a particular pathogen strain to cause disease on a particular host genotype (Day, 1960). Virulence is measured by the amount of disease incited, e.g., relative size or number of pustules, by an isolate of the pathogen. Nelson (1973) considers aggressiveness to be the ability to develop on the host at a faster rate; thus, a more aggressive isolate incites the same amount of disease in less time. The ability to develop at a faster rate is associated with the following fitness characteristics (Nelson, 1973):

(a) ability to persist in the nonparasitic stage.

(b) ability to infect under a wide range of environments.

(c) ability to produce a large number of infection sites.

(d) ability to generate a large amount of inoculum per given area of infection site.

(e) ability to complete a cycle of spore production in a shorter time (reduced incubation period).

The ability of the pathogen to cause severe damage in epidemics is related to its virulence and aggressiveness. A pathogen must have virulence to be able to infect and must be aggressive in order to survive year-round and to spread quickly.

Race-specific resistance is used to counteract virulence. Non-specific resistance theoretically reduces the effective aggressiveness

of the pathogen.

Race-specific resistance has the following characteristics (Nelson, 1973):

(1) it conditions against the establishment of a successful infection site (i.e., halts colonization of tissue).

(2) it usually involves hypersensitivity; Muller (1959) defined this as the "premature dying off (necrosis) of the infected tissue as well as the inactivation and localization of the infectious agent."

(3) it is an all-or-nothing resistance, effective against some races and totally ineffective against others (i.e., a differential reaction).

(4) it is the result of a gene-for-gene relationship or interaction. "For each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite" (Flor, 1955, 1956, 1971). The host and parasite have presumably developed this complementary genic system during their co-evolution.

(5) A resistance gene will condition resistance to all current races if none carry the corresponding virulence allele.

(6) A single incompatible interaction at any locus is sufficient to induce resistance no matter how many compatible interactions there are at other loci (Day, 1974).

(7) the resistance gene apparently becomes ineffective when the pathogen gene product being interacted with is absent, undetectable or inactive (Knott, 1967).

Knott (1967) suggests that incompatibility and the resistant response are induced by the interaction between two biologically active (dominant) gene products; and compatibility and susceptibility result

when one of the gene products does not participate in an interaction. Thus, a recessive virulence locus is able to avoid inducing the resistant response so that infection can proceed uninhibited.

Day (1974) uses the gene regulation model of Britten and Davidson (1969) to suggest that race-specific resistance genes are sensor genes whose product binds with the product of a specific avirulence gene from the parasite and thereby initiates the activation of a series of producer genes, producing the changes in metabolism associated with resistance. Thus, the pathogen induces a resistant response (the host selectively responds to specific signals without having to acquire a permanently altered metabolism) (Day, 1974). In such a model, the change to virulence need only be alteration of the pathogen gene product so it is a different shape and unrecognizable, or absent (Day, 1974).

It has been suggested that resistance in natural plant populations is a combination of specific and non-specific resistance, and that through plant breeding, the specific resistance genes are stripped of the protective and complementary polygenic non-specific genes and are subsequently exposed to the pathogen over large acreages. Consequently, mutations at a single locus are all that is required to overcome resistance. Extensive use of the resistance-gene guarantees survival and multiplication of the virulent races without competition from other races. Thus, the gene-for-gene concept becomes an artifact of breeding for resistance (Day, 1974).

Non-specific resistance, on the other hand, may be treated as having the following characteristics (Nelson, 1973):

(1) it reduces disease development rate due to restricted colonization of tissue and restricted inoculum production over time.

(2) it is generally polygenic.

(3) it is stable (cannot be completely overcome by the pathogen in one jump).

(4) it is effective against all races, though not uniformly so, as the more aggressive races may still be able to spread relatively faster.

Non-specific resistance may be associated with increased incubation time, restriction of the amount of tissue invaded and reduction of inoculum production (Nelson, 1973).

Day (1974) suggests that a parasite must acquire many properties to assist in infection and their genetic basis must be complex. General or nonspecific resistance would then involve the systems that affect the balance between host and parasite metabolism. Genes of small effect would then cumulatively block the induced susceptibility.

If a large number of mutations of small effect are necessary in the pathogen to overcome nonspecific resistance, then this accounts for the stability of the resistance (Nelson, 1973). Single mutations will have little selective value and it would be difficult to accumulate all the mutations necessary to overcome the resistance.

In spite of the advantages of non-specific resistance, specific resistance is the primary type of resistance used by plant breeders because it is easily recognized and readily attained due to its simple inheritance (Nelson, 1975).

2.4. Genetics of Resistance

2.4A. Genetics of Rust Resistance

In Hooker's (1967) review of the genetics of rust resistance it was pointed out that rust resistance may be inherited as:

(1) a single gene — commonly dominant, much less commonly incompletely dominant, and occasionally recessive,

(2) two or more genes, acting independently (as many as six in a line have been detected),

(3) two or more genes, linked, either loosely or tightly (rust resistance genes usually assort independently rather than being linked),

(4) complementary genes: two or three genes, either dominant or recessive or a combination of both,

(5) modifier genes acting on major or minor resistance genes to enhance or reduce their resistance,

(6) a polygenic resistance — reports of this are rare (however, a polygenic stable resistance conditioning low intensity of infection against *P. sorghi* Schw. has been highly effective in the U.S.A.).

Hooker also points out that "genes that condition a higher level of rust resistance are commonly epistatic to those conditioning a less resistant reaction." Inhibitors of resistance have also been demonstrated.

Multiple alleles, each conditioning a different phenotype or spectrum of resistance, may exist for a particular resistance locus. Alleles are differentiated by spectrum of resistance and by the lack of crossover products in a few hundred meiotic products. Alleles may actually be tightly linked loci, with broad spectrum alleles being complex loci consisting of several linked genes (Hooker, 1967).

A resistance gene may be dominant to one race and recessive to another. As well, a resistance gene may be temperature-sensitive, operating in one environment but not another (Hooker, 1967).

2.4B. Genetics of Stem Rust Resistance in Durums

Heermann *et al.* (1957) found that St. 464, an Ethiopian durum which was later used to provide the 15B stem rust resistance of Stewart 63 (Knott, 1963), had two dominant genes for resistance to 15B, one conditioning a type (x) infection, the other a type (1) infection, and together a type (o)(;). These genes were also present in four other cultivars; and three other lines carried the second gene (Heermann, 1954). Kenaschuk *et al.* (1959) reported the same genes in four Ethiopian lines and one Portuguese line. Ataullah (1963), using three Australian races, discovered a third gene in St. 464 conditioning a differential reaction.

Kenaschuk (1959) also reported a moderate-resistance gene in three other varieties (from Arabia, Spain and S. Africa) and a different gene in another Portuguese variety.

2.4C. Genetics of Leaf Rust Resistance in Triticale

Quinones (1972) reported monogenic dominant resistance to *P. recondita tritici* in five triticales. Five different genes were designated, each conditioning resistance to the same four races; two of the genes were either closely linked or allelic. The susceptible lines used were susceptible to both wheat leaf rust and rye leaf rust. The wheat leaf rust resistance genes also conditioned resistance to rye leaf rust.

When synthesizing polyhaploid F_1 triticales, Quinones (1972) noted that both the *P. recondita tritici* and *secalis* resistance was derived from the durum parent, the rye parent contributing nothing to resistance.

He also obtained hybrids susceptible to wheat leaf rust and resistant to rye leaf rust (this pattern being derived from the durum parent). All three ryes used were susceptible to rye leaf rust and resistant to wheat leaf rust.

2.5. Disease Resistance Derived from Wide Crosses

Green (1971a) suggested that incorporating rust resistance genes from other species or genera might force the pathogen to evolve to a more primitive state, sacrificing destructiveness for broadened host range.

Considerable effort has been made to transfer resistance genes carried on alien chromosomes, especially *Agropyron* and rye resistance into wheat. However, the alien genes were generally found to be race-specific, though initially providing broad-spectrum resistance (Hooker, 1967).

2.5A. Tetraploid Wheat into Hexaploid Wheat

Iumillo durum stem rust resistance was used to produce the variety Marquillo, which later became one of the parents of Thatcher. Yaroslav Emmer (*T. dicoccum* L.) stem rust resistance was transferred to hexaploid wheat to produce Hope, H-44 and Selkirk varieties (Allard, 1960). Race 15B overcame the Thatcher and Hope resistance (Knott, 1967). Shands (1941) and Allard (1949) made transfers of *T. timopheevi* Zhuk. stem rust resistance to hexaploid wheat.

2.5B. *Agropyron* and *Aegilops* into Hexaploid Wheat

Alien transfers have been used to provide wheat with leaf rust resistance from *Aegilops umbellulata* Zhuk. (resulting in the cultivar Transfer), stem and leaf rust resistance from *Agropyron elongatum* Host (Beauv.) (the wheat cultivars Agatha and Agrus carry the same leaf rust resistance gene; Agent carries a different one), stripe rust resistance from *Aegilops bicornis* (Forsk.) Jaub. and Spach., and stem, stripe and

leaf rust resistance from *Agropyron intermedium* Host (Beauv.) (Sharma and Knott, 1966; Smith *et al.*, 1968; Horsfall *et al.*, 1972; Sears, 1972a,b, 1973; Cauderon *et al.*, 1973). Samborski (1963) was able to obtain a leaf rust culture virulent on the dominant resistance gene from Transfer, simply by selfing a culture able to infect Transfer in the form of a type (1^+) infection. The original culture was heterozygous for an incompletely dominant avirulence gene and selfing produced some homozygotes which produced either a (o)(;) or (4) type infection on Transfer. Samborski pointed out that widespread use of the Transfer gene alone would select for the heterozygote and increase the chance of a spontaneous mutation to virulence. By 1974, the Transfer resistance gene was reported to be ineffective in the south U.S.A. as a single source of resistance (Cereal Rust Laboratory, Report #1, 1974).

2.5C. Rye into Hexaploid Wheat

Leaf, stem and stripe rust resistance and powdery mildew resistance have been transferred from rye to wheat.

A number of current European cultivars possess leaf and stem rust resistance derived from rye through 1B/1R substitutions or translocations (Bartos *et al.*, 1973). The genes are tightly linked and provide resistance to a large number of stem and leaf rust races. Mettin *et al.* (1973) pointed out that these varieties also carry race-specific mildew and stripe rust resistance from the rye. The cultivar Petkus may have been one source of these genes (Zeller, 1973). Zeller (1973) suggested that widespread culture of the 1B/1R substitution lines would place considerable selection pressure on the pathogens, and that natural hybridization between *formae speciales* could result in races able to

overcome the rye resistance.

The cultivar Transec carries a translocation involving rye chromosome 2R, and derives its powdery mildew and leaf rust resistance from the rye (Driscoll and Anderson, 1967).

Jensen and Kent (1952) reported leaf rust resistance from Rosen rye in a winter wheat selection. In studying this leaf rust resistance, Driscoll and Jensen (1964) reported that the seedling reactions were classified as an (x) infection type on the first leaf and the leaves became progressively more resistant until a (1) infection type stabilized at the sixth leaf stage. F_1 hybrids took longer to stabilize and conditioned an (x) type reaction under field conditions compared to a (o) to (1) type reaction for two doses of the gene.

Stewart *et al.* (1968) reported that Acosta's (1963) translocation lines involving a portion of an Imperial rye chromosome (3R) in a Chinese Spring background (Bielig and Driscoll, 1973) were highly resistant to race 15B at various temperatures and also to a large number of other wheat stem rust races, including highly virulent Kenyan races. The lines were resistant to both *P. graminis tritici* and *secalis*.

In wheat-rye addition lines derived from a Holdfast-King II cross, Riley and Macer (1966) found rye chromosome V (1R) to carry resistance to powdery mildew and stripe rust.

In their study, Riley and Macer found that the susceptibility of King II rye to *P. recondita secalis* was not expressed in the addition lines, nor was the resistance of King II to *P. recondita tritici*. They found that one chromosome arm contributed the full King II stripe rust resistance to wheat to eight races. An additional chromosome contributed a differential race-specific stripe rust resistance. Four chromosome

arms contributed good resistance to *E. graminis tritici*. Holdfast proved susceptible to stem rust and the amphidiploid resistant, but no single addition line was resistant. Riley and Macer suggested either complementation or suppression were involved in the absence of resistance in addition lines to wheat stem and leaf rust.

Riley and Macer, in discussing the possible evolution of pathogens, along with the divergent evolution of wheat and rye from a single diploid population, suggested three possible evolutionary sequences leading to rye resistance to wheat pathogens:

(1) resistance evolving as a secondary activity of new genes acquired for other primary functions involved with divergence. This would be complex resistance.

(2) resistance being retained from the original diploid progenitor as a residual genotypic difference, e.g., rye evolving from a resistant portion of the population, wheat evolving from a susceptible portion. This would be a simply-inherited resistance.

(3) resistance evolving due to selection pressure by the wheat-attacking pathogens. (Riley and Macer suggest this is unlikely as resistance to wheat-attacking pathogens would be of marginal significance with virulent rye-attacking pathogens around.)

Riley and Macer conclude that rye resistance to wheat pathogens is fortuitous, appearing to fall in evolutionary sequence (1) for stem rust, and sequence (2) for stripe rust.

2.6. Host-parasite Systems

2.6A. Effectiveness of Vertical Resistance

Robinson (1971) discusses crop and pathogen factors which affect the usefulness of vertical or race-specific resistance. He suggests vertical resistance may be more useful if the pathogen:

(a) is a "simple interest disease", i.e., multiplies slowly (as opposed to a "compound interest disease").

(b) has low vertical mutability (i.e., a low mutation rate for virulence).

(c) is not transmitted by host propagating material (e.g., wheat and *Puccinia graminis tritici*, as opposed to *Phytophythora infestans* (Mont.) de Bary in potatoes), or, if it is, then the pathogen not be "compound interest" (so that seed inspection can be effective even if incomplete).

(d) is rendered less fit by the presence of unnecessary virulence genes.

He also suggests a number of crop factors which favour the usefulness of vertical resistance:

(a) annual growth habit.

(b) genetic non-uniformity.

(c) limited acreages.

(d) the presence of vertical resistance genes which select for unfit virulent strains.

(e) separation of the resistance in space (e.g., multilines or zones) for "compound interest" diseases.

(f) separation in time (through rotations or by off seasons) for "simple interest" diseases.

(g) effective seed-health certification possible (will be for simple interest but not compound interest diseases).

(h) complete vertical resistance, in which case the pathogen can develop new races only through parasitizing another host population (if incomplete, then the host carries the disease and greatly magnifies the chances for mutation to virulence).

(i) a closed season which results in the annual destruction of the local pathogen population (through severe heat or frost). In this case the annual infection is from inoculum from external sources.

(j) legislative control of crop varieties being possible, to control seed-health certification and regional deployment of resistance, and prevent both resistant and susceptible crops being grown (the occurrence of which would increase the chances of virulent mutants surviving and multiplying).

(k) reinforcement of vertical resistance by horizontal resistance.

(l) complex resistance (assuming complex races are less fit).

Robinson points out that vertical resistance is not useful against late blight because *P. infestans* is a compound interest disease with a high vertical mutability and is carried by host propagating material; also, the host is often genetically uniform for resistance.

In the case of wart disease (*Synchrytrium endobioticum* (Schilb.) Perc. of potato, vertical resistance can be highly effective and long-lasting because the disease is a simple interest disease, an obligate parasite and has low mutability. Thus, where legislative control has prevented the planting of susceptible varieties (e.g., W. Europe), the resistance has been effective, whereas in E. Europe and Newfoundland, both resistant and susceptible cultivars were grown and the resistance

broke down. A reservoir of the pathogen existed where mutations could occur, in this latter case.

In the case of stem rust of wheat, a diversity of resistance genotypes appears to have favoured the use of a vertical resistance, as useful control has been achieved. However, costly breakdowns have occurred because the disease is a compound interest disease, and the crop is genetically uniform, tending to be grown on large acreages with a single or few cultivars. In addition, the protection conferred is usually incomplete, the crop is often grown where there is no closed season (e.g., Kenya), and it is probable that the horizontal resistance has been lost. Deployment of resistance in space through legislated regional gene deployment and through multilines could improve the longevity of resistance, especially if virulent races tend to remain unfit (Knott, 1972).

Van der Plank (1968) suggests that simple races are more fit to survive than complex races (stabilizing selection), speculating that mutation to virulence requires the dismantling of normal efficient metabolic pathways. Therefore, simple races would predominate on simple cultivars and complex races would not develop to become virulent on all regionally deployed genes or components of a multiline. Thus, the bulk of inoculum produced on the inoculum source (with simple resistance) would be simple races unable to attack regionally deployed genes or other components of a multiline.

Nelson (1973) cites considerable evidence against stabilizing selection and feels the concept is not valid. He suggests that virulence and fitness are inherited independently and that complex fit races can evolve and persist and predominate, rendering regional deployment ineffective.

Where the race makeup of a pathogen is determined by the host commercial crop, the resulting races are necessarily virulent in order to survive. Where the race makeup of a pathogen is determined separately from the host crop, in a different environment, the development of new races attacking the commercial crop may be limited by the competition of more adapted "non-virulent" races. It may be difficult to combine virulence with competitiveness on both the overwintering and cultivated hosts. Thus, the initial inoculum source for *Fusarium* wilt is the saprophytic stage, for stripe rust in the Pacific Northwest of the U.S.A., wild grasses, and for rust on N. American spring wheats, winter wheat in the S. U.S.A.; the development of new virulent races in these situations is limited (Horsfall *et al.*, 1972).

The N. American spring wheat-winter wheat situation is a good example. Knott (1972) pointed out that the races virulent on *Sr6* in N. America do exist but have not become predominant because the overwintering area for stem rust does not have the *Sr6* gene for resistance which would selectively increase these virulent races if *Sr6* were there. However, when, through the variety Austin, resistance to race 56 was used in the overwintering area, the corresponding virulent race 15B was selectively increased, without competition from race 56, contributing to the devastating epidemic in spring wheat areas which carried the resistance gene (Stakman and Harrar, 1957; Knott, 1972).

In contrast, Robinson (1971) noted that the effectiveness of vertical resistance to stem rust in Kenya is limited. The rust survives year round on the host crop and thus maintains a large reservoir of initial inoculum, undermining the main effect of vertical resistance which is to limit the amount of effective initial inoculum and delay the onset of disease.

The Committee on Genetic Vulnerability of Major Crops (Horsfall *et al.*, 1972) has pointed out that "genetic uniformity is the basis of vulnerability to epidemics" and that "most major crops are impressively uniform genetically and impressively vulnerable." The widespread use of a single resistance gene greatly multiplies the chance that a new virulent mutation or recombinant will survive and also ensures rapid build-up of that genotype because there is a lack of competition from other genotypes of the disease and a lack of different resistant varieties to restrict the spread.

2.6B. Patterns of Rust Epidemics

The Great Plains of North America and the Indian subcontinent both have similar patterns for rust development and spread in that the rust is continuously growing year round, but for part of that year is restricted to a particular area. In North America, the rust overwinters in the winter wheat area of the southern U.S.A. and spreads northward in the spring to Canada. The inoculum generated in the northern areas provides subsequent infections of the following winter wheat crop (Craigie, 1957; Knott, 1972). In the Indian subcontinent, the rust is killed by the summer heat except in the northern foothills, where the rust persists and initiates the spread of epidemics in the subsequent cool season (Mehta, 1931).

However, Prasada and Sharma (1973) pointed out that new races of stem rust may blow into India from neighbouring West Asian and near African countries.

Year-round uredial persistence of stem rust occurs in the American Pacific coastal region, the southern plateau and West coast of Mexico, Kenya (due to the availability of higher cooler elevations), and

Australia (Craigie, 1957; Oggema, 1972).

Barberry-originated infections are important for stem rust in Europe and Eastern N. America, where the uredial stage is unable to overwinter (Craigie, 1957).

In general, although there may be locally initiated infestations, stem rust tends to spread from warmer areas where it can overwinter (e.g., south U.S.A.) or multiply earlier (e.g., N. Australia) or from areas of higher elevation where it can escape severe heat (e.g., N. India, parts of Kenya, and Ethiopia) (Craigie, 1939).

Rye stem rust differs from wheat stem rust in N. America in that the uredial stage can overwinter on couchgrass in the much cooler areas of the northern U.S.A. or the Canadian prairies. This local infection constitutes the main source of inoculum (Johnson and Buchannon, 1954; Craigie, 1957).

SECTION II

THE GENETICS OF RESISTANCE TO *PUCCINIA GRAMINIS TRITICI* IN HEXAPLOID TRITICALE

1. ABSTRACT

THE GENETICS OF RESISTANCE TO *PUCCINIA GRAMINIS TRITICI* IN HEXAPLOID TRITICALE

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The inheritance of resistance to wheat stem rust, *Puccinia graminis tritici*, was studied in nine hexaploid triticales, using races C33 and C17. Seedling resistance was monogenically inherited in 70HN458, 6TA204, 6A413 and 6A250, digenically inherited in Rosner, MT36-1, Beaver and 6A406, and trigenically inherited in 6A190. It was determined that in total the cultivars carried at least eight different resistance genes. Tests with several races indicated that five of these genes conditioned wide-spectrum resistance while a gene from 6A190 and the genes from 6A406 did not condition resistance to all races used.

The seedling resistance genes conferred resistance in both the seedling and adult stages. Genes conferring resistance only in the adult plant stage were apparently non-existent in the lines selected for genetic study. However, 6A20, a seedling-susceptible line, was resistant in the field.

Synthesis of new triticales indicated that both durum wheat (*Triticum turgidum* var. *durum*) and rye (*Secale cereale*) could contribute stem rust resistance to triticale.

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2. INTRODUCTION

After a brief period (two decades) of intensive development as a new commercial crop species, hexaploid triticale, *X Triticosecale* (Wittmack), is showing promise in some areas of the world. Plant breeders initiating work with this unfamiliar crop require information on its disease resistance. To date, few studies on triticale disease resistance have been done.

Leaf rust (*Puccinia recondita* Rob. ex. Desm. f.sp. *tritici*) has been one of the most troublesome diseases in triticale breeding programs in North America. Quinones (1972) investigated the inheritance of triticale leaf rust resistance. He found resistance to be controlled in each line he studied by a single dominant gene and also concluded that the resistance was derived only from the wheat parent.

Ergot [*Claviceps purpurea* (Fr.) Tul.] is also a critical problem in triticale in some areas and attempts are presently being made to incorporate resistance into triticale (Larter, 1974a,b).

Stem rust (*Puccinia graminis* Pers. f.sp. *tritici* Eriks. and E. Henn.) in hexaploid triticale has not been a problem of the same magnitude as leaf rust and ergot. Much of the breeding material is resistant. However, susceptibility often appears in segregating material and this has occasionally caused the elimination of some potentially high-yielding advanced lines in breeding programs. For this reason, also because of the ability of stem rust to overcome resistance barriers by evolution, thereby creating a potential for severe crop losses, it was felt necessary to undertake a study to clarify the genetic mechanisms of stem rust resistance in triticale.

3. MATERIALS AND METHODS

The hexaploid triticale lines used in this study are listed in Table 1, along with parentage, origin, and reaction to races C17 and C33 of wheat stem rust (*P. graminis tritici*) and isolate #224-73 of rye stem rust (*P. graminis secalis*). Triticales were chosen on the basis of an initial stem rust testing of 700 lines, with an attempt to include different reaction types, areas of origin, plant types, and degrees of improvement from the "raw" amphiploid level. Nine resistant lines were chosen for the study, and 5 of these, viz. Rosner, 70HN458, 6TA204, 6A190, and MT36-1, were used for a detailed genetic examination of their rust resistance.

All nine lines were crossed to the common susceptible parent, MT32-1. In addition, the five principal parents were crossed to susceptible line 6A20. All crosses were advanced in the greenhouse and field to obtain F_3 lines. As well, backcrosses were made involving the five principal parents and the two susceptible parents, and advanced to obtain BC_1F_2 lines.

Diallel crosses were made among the nine resistant parents to determine gene relationships. F_2 seed was derived from these crosses. Part of the F_2 seed from crosses involving the five principal parents only was also advanced to the F_3 generation. 6TA204, 6A250 and four moderately resistant lines with resistance derived from Rosner, MT36-1, Beaver and 6A190 were also intercrossed; these crosses were advanced to the F_2 generation for testing, again to determine gene relationships.

All F_1 plants were grown in the greenhouse and the heads were bagged to ensure selfing.

TABLE 1. Parents, Pedigree, Source and Seedling Infection Types[1] to Races C17 and C33 of *Puccinia graminis tritici* and to Isolate #224-73 of *Puccinia graminis secalis*.

Line	Pedigree	Source	Seedling reaction		
			Wheat stem rust		Rye stem rust
			C17	C33	
Rosner	[(*T. turgidum* var. *durum* cv. Ghiza x *S. cereale*) x (*T. turgidum* var. *durum* cv. Carleton x *S. cereale*)] x [(*T. turgidum* var. *persicum* x *S. cereale*) x (*T. turgidum* x *S. cereale* hybrid of unknown identity)]	U. of M.	;	;	;
70HN458 (Armadillo Selection)	Rosner x [(*T. turgidum* var. *dicoccoides* x *S. cereale* x *T. turgidum* var. *persicum* x *S. cereale*)] X-308-14Y-1M-0Y-1W-0W	Mexico	$;^{c}1^{=}$	$;^{c}1^{-}$	$1, 1$ to 3^{-}
6TA204	[*T. aestivum* "P4160E$_3$" x (*T. turgidum* var. *durum* x *S. cereale*)] x [(*T. aestivum* x *S. cereale*) x (*T. turgidum* var. *durum* x *S. cereale*)]	California	1 to 2^{-}	1 to $2^{=}$	$;1^{-}$
6A190	*T. turgidum* var. *durum* cv. Stewart x *S. cereale*	U. of M.	;	;	$;N1^{-}N$
MT36-1	Beaver 'S' x UM940†	U. of M.	;	;	;
Beaver	A selection from a bulk population which predominantly involved Armadillo	Mexico	;	;	;
6A413	*T. turgidum* var. *durum* (RD121-9) x *S. cereale*	U. of M.	;	;	;
6A250	*T. turgidum* var. *persicum* x *S. cereale*	U.S.S.R. (Pissarev)	1	2^{-}	$;1\equiv$
6A406	*T. turgidum* var. *durum* (4B909) x *S. cereale* (2D53)	U. of M.	$;1^{=}$	$;N1^{=}$	$;1^{-}N$
MT32-1	Armadillo 'S' x Rosner	Mexico	4	4	$1^{\pm}$, 1 to 3^{-}
6A20	*T. turgidum* var. *durum* cv. Carleton x *S. cereale*	U.S.A. (O'Mara)	3 to 3^{++}	3^{++}	;

[1] Classification according to Stakman *et al.* (1962).

† UM940 is derived from the cross [(*T. aestivum* cv. Prelude x *S. cereale* cv. Prolific) x (*T. turgidum* var. *persicum* x *S. cereale*)] x [(*T. turgidum* var. *durum* cv. Ghiza x *S. cereale*) x (*T. turgidum* var. *durum* cv. Carleton x *S. cereale*)].

The F_2 families and BC_1F_2 and F_3 lines were grown in pots for rust testing in the greenhouse. The screening procedure involved testing 20-25 seedlings per line from susceptible x resistant crosses or 50-60 seedlings per line from resistant x resistant crosses. Week old seedlings (at the 1 to 1½ leaf stage) were inoculated by spraying an oil suspension of spores on the leaves. The inoculated plants were then incubated in the dark in a moist chamber for 24 hours, then put through a 12 hour day/12 hour night cycle at controlled temperatures (24°C day; 21°C night). The plants were then moved to the greenhouse. Rust reactions were recorded 12-14 days after inoculation, using the system described by Stakman *et al.* (1962).

Segregating material was inoculated with races C17 (56), isolate 33-71, and C33 (15B-1L), isolate 42-71. These were chosen to represent the predominant races in Western Canada in the last 35 years, races which clearly differ in their characteristics yet which have caused serious epidemics (Green, 1971b). C33 has been the most prevalent race in Western Canada since 1970 (Green, 1973); C17 was the predominant race in the period 1934-1949 (Johnson and Green, 1957; Green, 1971b).

Parental material was included in all tests of segregating material. The parents were further tested as seedlings to a number of races to determine range of resistance; these races included C10 (15B-1), isolate #121-69, C35 (32-113), isolate #111-70, race 111 (111X36) WSR #2179, race C27 (59), isolate #43-71, rye stem rust isolate #224-73, and the 1975 wheat stem rust race mixture. As well, several "extracted" F_3 and F_4 lines exhibiting moderate resistance previously masked in the parental lines were tested with these races. A number of miscellaneous triticales, durums and ryes were included in these tests.

Tests were conducted to determine the effectiveness of the seedling resistance in later stages in the field. A mixture of races C17 and C33 was used for field testing at The University of Manitoba in 1974, while a mixture of many races was employed at the Canada Department of Agriculture Research Station rust nursery at Glenlea in 1973 and 1974.

Seedling susceptible F_3 and BC_1F_2 lines from susceptible x resistant crosses were also tested as adult plants in the field in 1974 to a mixture of races C17 and C33 to detect the presence of any independently segregating resistance genes expressed only in the adult stage. The F_2 progeny of 6A20 x MT32-1 was also included in this test. The seedling susceptible BC_1F_2 lines were also tested as seedlings with races C10, C35, C27, 111, and rye stem rust in the hope of detecting additional seedling resistance genes.

In order to determine the source of triticale resistance to wheat stem rust, hexaploid triticales were synthesized from several durums and ryes, and the resistances of the parents and amphiploids were compared. Susceptible and resistant durums and resistant ryes were readily available, but a susceptible spring rye line was obtained only after intercrossing several susceptible segregants from Centeno, Prolific and Argentina ryes. Crosses of durum and rye were made with the durum as the female plant. Fifteen-day-old hybrid embryos were artificially cultured and the resulting F_1 seedlings were tested for resistance using race C33. These polyploid seedlings were also treated with colchicine and doubled. The C_1 progenies of doubled F_1 plants were tested with race C33 and, subsequently, the parents and C_2 progenies were screened with several races. C_2 plants were also grown in the field under natural rust epidemic conditions in 1975.

For F_2, F_3 and BC_1F_2 data, the χ^2 test was used to test goodness of fit. In resistant x resistant crosses where few or no recombinants were obtained, estimates were made of linkage values according to Mode and Schaller (1958) or of maximum recombination values according to Hanson (1959). Estimation of linkage in susceptible x resistant crosses was according to Immer (1934).

4. RESULTS

(i) Susceptible x Resistant Crosses

Inheritance. Data on the mode of inheritance of resistance for the five principal parental lines used in this study were obtained from seedling reactions of F_3 and BC_1F_2 populations (Tables 2, 3, 4 and 5) involving susceptible lines 6A20 and MT32-1.

F_3 and BC_1F_2 populations involving Rosner were inoculated with race C17; the progenies obtained by backcrossing to MT32-1 were re-inoculated with race C33. The data indicate that Rosner carries two resistance genes, one conditioning a fleck infection type to both C17 and C33, the second conditioning an infection type (2) to both races. Rosner itself and the F_1's involving Rosner all exhibited a fleck reaction to C33, indicating dominance of the gene conferring a fleck reaction. An F_3 line (24-39) from the cross MT32-1 x Rosner carried the gene conditioning a type (2) infection, and when later crossed to the susceptible MT32-1, this gene also exhibited dominance.

A similar procedure was used for 70HN458 and 6TA204, except that the F_3 lines from MT32-1 x 6TA204 were also screened with race C33. 70HN458 was found to possess one dominant gene conditioning infection types fleck and (1) to both races C17 and C33, while the line 6TA204 also appeared to carry a single dominant gene, in this case conditioning an infection type (2) to both races. The segregation of the cross MT32-1^2 x 70HN458 to C33 was distorted from a 1:1 ratio. However, fewer lines were available for screening with C33 than with C17, as a number of C17-susceptible lines had insufficient seed for screening with C33 also. If these C17-susceptible lines were susceptible to C33, this would explain the distortion. Simply chance could account for the distortion.

TABLE 2. Segregation of Reaction to Races C17 and C33 among F_3 Lines Derived from Susceptible x Resistant Crosses.

Cross	Race	Number of lines			Expected ratio	P
		Resistant	Segregating	Susceptible		
MT32-1 x Rosner	C17	33	41	9	7:8:1	.30 - .20
6A20 x Rosner	C17	29	37	5	7:8:1	.90 - .70
MT32-1 x 70HN458	C17	24	45	24	1:2:1	1.0 - .95
6A20 x 70HN458	C17	23	29	13	1:2:1	.20 - .10
MT32-1 x 6TA204	C33	15	37	9	1:2:1	.20 - .10
MT32-1 x 6TA204	C17	16	44	25	1:2:1	.50 - .30
6A20 x 6TA204	C17	21	47	17	1:2:1	.70 - .50
MT32-1 x 6A190	C33	19	44	7	7:8:1	.05 - .01*
MT32-1 x 6A190	C17	43	36	2	37:26:1	.70 - .50
6A20 x 6A190	C17	31	19	1	37:26:1	.90 - .70
MT32-1 x MT36-1	C33	54	51	3	7:8:1	.30 - .20
MT32-1 x MT36-1	C17	72	77	6	7:8:1	.50 - .30
6A20 x MT36-1	C17	39	32	1	7:8:1	.10 - .05
MT32-1 x Beaver	C17	34	48	4	7:8:1	.70 - .50
MT32-1 x 6A413	C33	21	60	25	1:2:1	.50 - .30
MT32-1 x 6A250	C17	19	45	20	1:2:1	.90 - .70

* If ratio is adjusted for linkage, p = .10 - .05.

TABLE 3. Segregation of Reaction to Races C33 and C17 among BC_1F_2 Lines Derived from Susceptible[2] x Resistant Backcrosses.

Cross	Race	Number of lines		Expected ratio	P
		Segregating	Susceptible		
6A20[2]$_2$ x Rosner	C17	9	6	3:1	.30 - .20
MT32-1$_2^2$ x Rosner	C17	28	7	3:1	.70 - .50
MT32-1[2] x Rosner	C33	24	6	3:1	.70 - .50
6A20[2]$_2$ x 70HN458	C17	22	15	1:1	.50 - .30
MT32-1$_2^2$ x 70HN458	C17	22	18	1:1	.70 - .50
MT32-1[2] x 70HN458	C33	23	10	1:1	.05 - .01
6A20[2]$_2$ x 6TA204	C17	18	22	1:1	.70 - .50
MT32-1$_2^2$ x 6TA204	C17	23	21	1:1	.90 - .70
MT32-1[2] x 6TA204	C33	21	13	1:1	.30 - .20
6A20[2]$_2$ x 6A190	C17	5	4	7:1	.05 - .01
MT32-1$_2^2$ x 6A190	C17	23	6	7:1	.30 - .20
MT32-1[2] x 6A190	C33	17	10	3:1	.30 - .20
6A20[2]$_2$ x MT36-1	C17	26	15	3:1	.20 - .10
MT32-1$_2^2$ x MT36-1	C17	39	12	3:1	.95 - .90
MT32-1[2] x MT36-1	C33	36	10	3:1	.90 - .70

TABLE 4. Comparison of Reaction of Individual F_3 Lines to Races C17 and C33, in Populations Derived from Susceptible MT32-1 x Resistant Parent Crosses.

Resistant parent	Classification of lines*									Expected ratio	P
	Res. C17 Res. C33	Res. C17 Seg. C33	Res. C17 Sus. C33	Seg. C17 Res. C33	Seg. C17 Seg. C33	Seg. C17 Sus. C33	Sus. C17 Res. C33	Sus. C17 Seg. C33	Sus. C17 Sus. C33		
6TA204	8	-	-	-	25	-	-	-	9	1:2.1	.50 - .30
6A190	18	15	2	-	23	4	-	-	1	28:8:1:24:2:1	.05 - .01**
MT36-1	54	-	-	-	51	-	-	-	3	7:8:1	.20 - .10

* Res. = Resistant; Seg. = Segregating; Sus. = Susceptible.

** If ratio is adjusted for linkage, p = .20 - .10.

TABLE 5. Comparison of Reaction of Individual BC_1F_2 Lines to Races C17 and C33, in Populations Derived from Susceptible MT32-1 x Resistant Parent Crosses.

Resistant parent	Classification of lines*				Expected ratio	P
	Seg. C17 Seg. C33	Seg. C17 Sus. C33	Sus. C17 Seg. C33	Sus. C17 Sus. C33		
Rosner	24	-	-	6	3:1	.70 - .50
70HN458	21	-	-	10	1:1	.10 - .05
6TA204	21	-	-	13	1:1	.30 - .20
6A190	16	5	-	5	6:1:1	.30 - .20
MT36-1	36	-	-	7	3:1	.30 - .20

* Seg. = Segregating; Sus. = Susceptible.

F_3 and BC_1F_2 populations involving MT32-1 and the fourth principal parental line, 6A190, were inoculated with both races C17 and C33. In addition, F_3 and BC_1F_2 populations involving 6A20 and 6A190 were inoculated with C17. The F_3 lines, when screened with C17, clearly indicated the presence of three genes, one conditioning a fleck infection type, the other two each conditioning a (2) type reaction. The 6A20 backcross data did not support this, possibly because of inadequate population size and accidental selfing. Screening with race C33, however, produced a susceptible reaction on a portion of the F_3 lines which exhibited a type (2) infection with race C17. This suggested that only two of the genes conditioned resistance to both C17 and C33, with a third gene conditioning resistance to C17 only. The backcross lines screened with both C17 and C33, supported this supposition. However, the F_3 lines from MT32-1 x 6A190, when screened with C33, did not fit an expected segregation of 7:8:1 (resistant:segregating:susceptible), nor a 12:3:1 F_2 ratio [(;) reaction:(2) reaction:(4) reaction]. In the case of a hypothesized F_2 12:3:1 ratio, the F_2 segregation obtained by classifying the original F_2 plants on the basis of F_3 line reactions was 58 (;):5 (2):7 (4). Too few individuals in the second category and more than expected in the third indicated linkage. The two 6A190 genes conditioning resistance to C33 were calculated to be 23.62 ± 9.32 crossover units apart.

The two genes conditioning moderate resistance in 6A190 were each isolated separately in the homozygous condition in F_3 lines 27-44 [conditioning infection type (2) to both C17 and C33] and 27-99 [conditioning infection type (2) to C17 and an infection type (4) to C33]. In a cross to MT32-1, the resistance gene in line 27-44 was dominant. In F_3 lines segregating for the differential (resistant to C17 but not C33) gene only, a 3:1 ratio

was exhibited, again indicating dominance.

Using a similar procedure to that used for 6A190, MT36-1 was found to possess two genes, one conditioning a fleck type infection with both C17 and C33, the second conditioning a (2) infection type for both races. MT36-1 itself, and the F_1's of the susceptible parent x MT36-1 crosses, all exhibited a fleck infection type for C33, indicating dominance of the gene conferring the fleck type resistance. F_3 line 4-49 from the cross MT32-1 x MT36-1 carried only the gene conferring moderate resistance and was used in later crosses to MT32-1, in which dominance was also demonstrated.

The following three lines, Beaver, 6A413 and 6A250, were not tested as comprehensively as the above five lines, in that only F_3 populations from crosses to MT32-1 were analyzed. Segregation to race C17 within the F_3 population involving Beaver indicated that Beaver carried two genes conditioning resistance to C17, one of these conferring a fleck type resistance, the second conferring a (2) type resistance. Beaver itself, and the F_1 of the cross MT32-1 x Beaver, exhibited a fleck type reaction to C33, and segregation within F_3 lines screened with C17 further indicated both genes to be dominant. F_3 line 44-9 from the cross MT32-1 x Beaver carried the gene conditioning moderate resistance to race C33 and was used in later crosses to MT32-1, where dominance was also indicated. Time and space limitations prevented checking all the F_3 lines with C33 to determine if the two genes each conditioned resistance to both races. However, three F_3 lines were tested to both races and the reaction was consistent between races in that line 44-36 segregated for a fleck type reaction only, line 44-31 segregated for a type (2) reaction only, and line 44-9

appeared homozygous for a gene conditioning a type (2) reaction. (The gene conferring moderate resistance varied between a type (1) and (2) reaction, depending on growing conditions.)

F_3 lines derived from 6A413 crossed with susceptible MT32-1 indicated that 6A413 carries one dominant gene conditioning a fleck type resistance to C33. 6A413 itself, and the F_1 from the cross MT32-1 x 6A413, exhibited a fleck reaction to C33, further indicating dominance. Again, due to time and space limitations, no check was made to determine if the same gene conditioned resistance to C17.

Similarly, F_3 lines from the cross of susceptible MT32-1 x 6A250 were inoculated with C17 and this indicated that 6A250 carries one gene conditioning a moderate type (2) resistance to C17. 6A250 itself, and the F_1 of the cross MT32-1 x 6A250, exhibited a 2 infection type to C33, again indicating dominance. As well, dominance is indicated in the case of C17, as a 3:1 segregation occurred within F_3 lines screened with C17. Again, due to time and space limitations, no check was made to determine if the same gene conditioned resistance to C33.

Testing with further races. A total of 24 seedling susceptible BC_1F_2 lines from the 5 principal parents were further tested with four more races, C10, C35, C27, and 111, in the hope of detecting further resistance genes. However, none were expressed. The lines that were seedling susceptible to both C17 and C33 were also seedling susceptible to these four different races. Unfortunately, only three to six lines per parent were available for this testing, so this cannot be considered an exhaustive test for further genes.

(ii) Resistant x Resistant Crosses

F_2 populations from all resistant x resistant crosses were tested with C33 (Tables 7, 8, 9 and 10); as well, F_3 lines from the crosses involving the diallel of Rosner, 70HN458, 6TA204, 6A190 and MT36-1 were tested with C33 (Table 6). To determine the interrelationships between genes conditioning fleck infection types, the data from several multi-gene crosses were classified according to two-gene segregations. In these cases, under the assumption of linkage, moderately-resistant segregants were considered to be recombinants.

The differential gene of 6A190 (conditioning resistance to race C17 but not race C33) was not considered in resistant x resistant crosses since C33 was used as the screening race.

Although the inheritance of the resistance of Beaver and 6A250 was clarified for race C17 only, it was assumed that these lines carried two and one gene(s) respectively for resistance to race C33; this assumption was supported by the results of the resistant x resistant crosses (Table 8). Whether the Beaver and 6A250 resistance genes individually condition resistance to both race C17 and race C33, or whether several differential genes are involved was not determined in the present study.

The results indicate that the genes conditioning a fleck infection type in Rosner, MT36-1, 70HN458, 6A413, and Beaver are either identical, allelic, or very tightly linked, as no recombinants were obtained when these were intercrossed (Tables 6 and 7). (It should be noted that Beaver was crossed only to MT36-1). Rosner, MT36-1, 6A413 and Beaver have identical phenotypic reactions to stem rust, i.e., a minute fleck under certain conditions, or a (0) reaction under other conditions.

TABLE 6. Segregation of Reaction to Race C33 among F_3 Lines Derived from Resistant x Resistant Crosses and Classification as Nonrecombinant and Recombinant Lines.

Cross and parental rust reaction	Number of F_3 lines			Test ratio	P	Number of F_3 lines		Test ratio‡	P
	Res.[1]	Seg.	Sus.			Nonrecombinant*	Recombinant**		
6TA204 (2) x 70HN458 (;)	40	42	1	7:8:1	.20-.10	40	43	7:9	.50-.30
6TA204 (2) x MT36-1 (;)	74	33	0	37:26:1	.05-.01	74	33	37:27	.05-.01
6A190 (;) x 6TA204 (2)	69	30	0	37:26:1	.05-.01	69	30	37:27	.05-.01
6A190 (;) x Rosner (;)	82	2	0	175:80:1	< .001	81	3	7:9	< .001
6A190 (;) x MT36-1 (;)	74	4	0	175:80:1	< .001	71	7	7:9	< .001
6A190 (;) x 70HN458 (;)	93	3	0	37:26:1	< .001	93	3	7:9	< .001
Rosner (;) x 70HN458 (;)	59	0	0	37:26:1	< .001	59	0	7:9	< .001
70HN458 (;) x MT36-1 (;)	65	0	0	37:26:1	< .001	65	0	7:9	< .001
Rosner (;) x MT36-1 (;)	109	0	0	175:80:1	< .001	109	0	7:9	< .001
Rosner (;) x 6TA204 (2)	83	0	0	37:26:1	< .001	83	0	37:27	< .001

[1] Res. = Resistant; Seg. = Segregating; Sus. = Susceptible.

* Nonrecombinant = parental infection types.

** Recombinant = nonparental infection types.

‡ Simplified to a two-gene ratio for crosses in which both parents exhibited a fleck infection type, so that only the genes conditioning a fleck infection type are being considered.

TABLE 7. Nonrecombinants and Recombinants Observed within F_2 Progenies Derived from Resistant (;) x Resistant (;) Crosses Tested with Race C33.

Cross	Number of F_2 plants		P for
	Nonrecombinant*	Recombinant**	15:1 ratio***
6A190 x 6A413	335	0	.001
6A190 x Rosner	632	0	.001
6A190 x 70HN458	882	2	.001
6A413 x 70HN458	419	0	.001
Rosner x 70HN458	546	0	.001
70HN458 x MT36-1	858	0	.001
Beaver x MT36-1	960	0	.001
6A413 x MT36-1	230	0	.001
Rosner x MT36-1	1095	0	.001
Rosner x 6A413	296	0	.001

* Nonrecombinant = parental infection type, fleck.

** Recombinant = nonparental infection type, (1), (2), (3) or (4). The two observed recombinants exhibited a (2) and (4) infection type, respectively.

*** The 15:1 ratio is used under the assumption of independence when only the two genes conditioning a fleck infection type are being considered.

TABLE 8. Segregation of Reaction to Race C33 within F_2 Progenies from Resistant x Resistant Crosses Involving Fleck-type and Moderate-type Reactions.

(;) type parent involved in cross	Expected ratio	(2) type parent involved in cross					
		6TA204		P for Expected ratio	6A250		P for Expected ratio
		Number of F_2 plants in Cross			Number of F_2 plants in Cross		
		Resistant	Susceptible		Resistant	Susceptible	
6A413	15:1	368	11	.01-.001	143	12	.70-.50
70HN458	15:1	1221	53	.01-.001	131	16	.05-.01
6A190	63:1	1001	20	.50- .30	95	0	.50-.30
Rosner	63:1	716	0	.01-.001	253	4	.90-.70
MT36-1	63:1	379	5	.90- .70	231	4	.95-.90
Beaver	63:1	219	3	1.0 - .95	-	-	-

TABLE 9. Segregation of Reaction to Race C33 within F_2 Progenies Derived from Resistant x Resistant Crosses Involving only Moderate-type Reactions.

Cross	Number of F_2 plants		P for 15:1 ratio
	Resistant	Susceptible	
6A250 x 24-39	375	2	< .001
6A250 x 6TA204	314	0	< .001
6TA204 x 24-39	215	0	< .001
44-9 x 4-49	273	0	< .001
44-9 x 24-39	203	13	1.0
4-49 x 24-39	240	21	.30 - .20
6A250 x 44-9	206	39	< .001
6A250 x 4-49	174	18	.20 - .10
44-9 x 6TA204	196	8	.30 - .20
4-49 x 6TA204	217	34	< .001
27-44 x 24-39	224	49	< .001
27-44 x 6TA204	424	32	.70 - .50
6A250 x 27-44	197	35	< .001
44-9 x 27-44	202	26	.01 - .001
27-44 x 4-49	101	25	< .001

TABLE 10. Segregation of Reaction within F_2 Progenies Derived from Resistant x Resistant and Resistant x Susceptible Crosses Involving 6A406.

Cross and parental rust reaction	F_1 reaction to C33	Race for screening F_2's	Number of F_2's			Expected ratio Res.:Sus.*	P	Expected ratio (;):(2):(4)	P
			Resistant		Susceptible				
			(;)	(1) to (2)	(4)				
MT32-1 (4) x 6A406 (;)	$1^{+}N$	C17	28	69	21	13:3	.90-.70	3:10:3	.50-.30
6A406 (;) x MT32-1 (4)	-	C17	17	84	21	13:3	.90-.70	3:10:3	.30-.20
6A406 (;) x 6A250 (2)	1N	C33	23	93	28	51:13	.90-.70	12:39:13	.70-.50
6A406 (;) x 6TA204 (2)	$;_c 1^{=}$	C33	76	57	2	63:1	1.0	36:27:1	1.0 -.95
6A406 (;) x 70HN458 (;)	$;_c$	C33	121	10	32	51:13	.95-.90	48:3:13	.70-.50
6A406 (;) x 6A413 (;)	;	C33	100	10	24	51:13	.70-.50	48:3:13	.30-.20
6A406 (;) x Rosner (;)	;	C33	252	44	11	243:13	.30-.20	192:51:13**	.05-.01
6A406 (;) x MT36-1 (;)	;	C33	159	33	8	243:13	.70-.50	192:51:13	.50-.30
6A406 (;) x 6A190 (;)	;	C33	237	61	13	243:13	.70-.50	192:51:13	.90-.70

* Res. = Resistant; Sus. = Susceptible.

** 204:39:13 ratio, p = .50-.30. The better fit to this ratio indicates a similarity to the behaviour of the 6A406 x 6A250 cross.

TABLE 11. Summary of Relationships Between Genes Identified for Seedling Resistance to Races C33 and C17 of Wheat Stem Rust.

Linkage group	Gene	Infection type		Sources
		Race C17	Race C33	
I	1	0, ;	0, ;	Rosner, MT36-1, Beaver, 6A413
	2	$;_c$ to 1^-	$;_c$ to 1^-	70HN458, 6A190
II	3	1 to 2^-	1 to 2^-	Rosner (24-39), 6TA204, 6A250
III	4	1	2	MT36-1 (4-49), Beaver (44-9)
IV	5	2^-	2	6A190 (27-44)
?	6	2^-	4	6A190 (27-99)
?	7	2 or $x^{\pm}$	x^+ or 4	6A406
?	8	2 or $x^{\pm}$	x^+ or 4	6A406

After MT36-1 and Rosner were backcrossed three times to MT32-1, and 6A413 twice, the phenotypic reactions were still identical, although being then expressed as a clear, consistent fleck infection type. Since MT36-1 was originally derived from a cross involving Beaver, it would be expected that these carry the identical gene. Furthermore, MT36-1, Beaver, Rosner and 6A413 exhibit the same reaction to all races tested. Allelism is therefore ruled out unless a rust race can be found to differentiate them.

However, 70HN458 exhibits a distinctly different phenotype from Rosner, MT36-1, Beaver and 6A413. 70HN458 displays a distinct $(;)_c(1^-)$ infection type to C33, and after three backcrosses to MT32-1, a (1^-) type reaction to C33. It may be that 70HN458 carries a gene that is tightly linked to the Rosner gene conditioning a fleck infection type; and this gene of 70HN458 would have to be 4.55 crossover units or closer to the Rosner gene (since no recombinants were obtained in the 59 F_3 lines derived from the cross to Rosner and the 65 F_3 lines derived from the cross to MT36-1). The 70HN458 gene could also be allelic to the Rosner gene.

Another possibility is that the locus for the Rosner gene actually could consist of two tightly-linked genes. This would not be detectable in the susceptible x resistant crosses since there would be confusion with the independent Rosner gene conferring moderate resistance. Since 70HN458 is derived from a cross involving Rosner, it may be that this tight linkage was broken, leaving 70HN458 with the gene conferring the lesser resistance.

From phenotype, the 6A190 gene conferring a fleck-type reaction also appears different from the corresponding Rosner gene; segregants obtained in the F_3 populations derived from the crosses 6A190 x Rosner

and 6A190 x MT36-1 seem to support this distinction (Table 6). The Rosner gene is expressed as a minute fleck infection type, while the 6A190 gene is expressed as a chlorotic fleck, though more indistinct than the 70HN458 chlorotic fleck. Also, after 70HN458 and 6A190 were each backcrossed three times to MT32-1, they both exhibited a type (1^-) reaction to race C33. Although similar in phenotype to the 70HN458 gene, the 6A190 gene does not appear identical to it, as recombinants were obtained in both the F_2 and F_3 populations derived from 6A190 x 70HN458 (Tables 6 and 7). Recombinants were not obtained in the F_2 populations derived from 6A190 x 6A413 and 6A190 x Rosner, which could be attributed to inadequate population sizes.

Because of the different phenotypes, and recombinants, it can be concluded that the genes of 6A190 and Rosner conferring a fleck reaction are different but tightly linked. However, in considering the relationship between the genes from 6A190 and 70HN458 conferring an identical resistance phenotype, the supposed recombinants from 6A190 x 70HN458 could easily have been due to aneuploidy. Since 6A190 has a high rate of aneuploidy, the evidence does not appear sufficient to conclude that the fleck genes of 6A190 and 70HN458 are different.

The remaining genes involved in these resistant x resistant crosses all conferred a moderate type (2) resistance. 6A250, 6TA204 and the extracted lines each carrying a single moderate resistance gene from Rosner (24-39), MT36-1 (4-49), Beaver (44-9) or 6A190 (27-44) were intercrossed to provide additional data to that from the F_2 and F_3 populations from parental intercrosses (Table 9).

No segregants were obtained in the F_2 population derived from 44-9 x 4-49, indicating that Beaver and MT36-1 carry identical genes, as would

be expected with Beaver in the parentage of MT36-1.

No convincing segregants were obtained in the F_2 populations from intercrosses of 6TA204, 6A250 and 24-39. The 6TA204 gene appeared allelic or identical to the Rosner (24-39) and 6A250 moderate resistance genes, as no segregants were obtained. The F_2 and F_3 data from Rosner x 6TA204 supported this, as no segregants were derived from this cross (Tables 6 and 8). When 6A250 and 24-39 were intercrossed, however, two of the 377 F_2 plants derived from the cross appeared susceptible, indicating tight linkage (Table 9). The F_2 plants derived from 6A250 x Rosner also segregated, 253 resistant : 4 susceptible ($p = .90 - .70$ for a 63:1 ratio), (Table 8), but this indicated independence, which would be too inconsistent with the above data.

The "segregants" from 6A250 x 24-39 and from 6A250 x Rosner may be attributed to off-types of some kind. The two segregants from 6A250 x 24-39 exhibited the infection types ($3^{=}$) for one and (3) for the other, unlike the (4) infection type exhibited by the susceptible checks, and one of the discarded F_1 progenies from the cross segregated 20 (2):10 (4), indicating chromosome loss. One of the susceptible plants from 6A250 x Rosner appeared stunted and, therefore, possibly aneuploid. With the above mentioned abnormalities, it seems that the data cannot eliminate the possibility that the genes being considered in 6A250, 6TA204 and Rosner are identical or allelic.

When 6A250, 6TA204 and 24-39 were intercrossed with lines 4-49 and 44-9, independence was indicated, although two of the six F_2 cross populations did not fit a 15 resistant : 1 susceptible ratio due to a surplus of susceptible plants (Table 9). F_2 data from the populations derived from 6TA204 x MT36-1, 6TA204 x Beaver, and 6A250 x MT36-1 all

clearly fit 63 resistant : 1 susceptible ratios, further supporting the conclusion of independence (Table 8). The F_3 lines obtained from the cross 6TA204 x MT36-1 were categorized as 74 resistant : 33 segregating : 0 susceptible ($p = .05 - .01$ for a 37:26:1 ratio), again supporting the above observations, although there appears to be a surplus of resistant lines (perhaps due to outcrossing or insufficient sample size).

F_2 data from crosses of 27-44 with the intermediate-type lines indicated the 6A190 moderate resistance gene to be independent of the 6TA204 and MT36-1 loci conferring moderate resistance (Table 9). However, only the F_2 data from 27-44 x 6TA204 clearly fitted a 15 resistant: 1 susceptible ratio, as the other four crosses had a surplus of susceptible plants. Segregating F_3 lines obtained in the populations derived from crosses of 6A190 to MT36-1, Rosner and 6TA204 also indicated the 6A190 gene for moderate resistance to be different from the 6TA204 and MT36-1 genes conferring moderate resistance. The F_3 lines from 6A190 x 6TA204 did not segregate to fit a 37:26:1 ratio, due to a surplus of resistant lines, perhaps again because of outcrossing or too small a sample. The F_2 data from 6A190 x 6TA204 fitted a 63:1 ratio of resistant:susceptible ($p = .50 - .30$), further indicating independent segregation of the moderate resistance genes of 6A190 and 6TA204 (Table 8). The F_2 population from 6A250 x 6A190 was too small to ensure detection of susceptible plants from a normal three-gene segregation (Table 8).

The surplus of susceptible plants obtained on a number of the crosses of extracted lines is not readily explainable. The extracted lines were agronomically poor plants, however, and could easily have

been aneuploid which could account for some distortion of results.

The 6TA204 gene appeared to be loosely linked to the genes of 6A413 and 70HN458 conferring a fleck reaction, as crosses involving these parents failed to fit an F_2 segregation of 15 resistant:1susceptible due to a deficiency of susceptible plants (Table 8). The F_2 results for 6A250 crossed to 6A413 and 70HN458 were contradictory to those for 6TA204, as any distortion of the ratio was towards a surplus of susceptible plants (Table 8).

(iii) 6A406

The line 6A406 is discussed separately herein, combining data from both susceptible x resistant and resistant x resistant crosses (Table 10).

F_2 plants from the crosses between 6A406 and MT32-1 were highly infertile and adequate F_3 progenies were therefore not available.

The F_2 populations from various crosses involving 6A406 segregated in a manner suggesting that 6A406's resistance is controlled by two additive or complementary genes, each apparently conditioning a mesothetic reaction, the dominance relationship and exact reaction varying in different backgrounds and for different races.

6A406 exhibited a type (;)($1^{=}$) reaction to C33 and C17. When crossed to susceptible MT32-1, the F_1 heterozygote reaction was a type (1^{+})N to C33. The F_2 results from crosses between 6A406 and MT32-1 indicated the presence of a dominant and a recessive gene together conditioning a fleck reaction to C17, but individually conditioning a dominant and recessive type (1) to (2) reaction respectively, resulting in a 3(;):10(2):3(4) ratio.

Generally, when C33 was used to screen resistant x resistant cross F_2 populations involving 6A406, the 6A406 genes individually conditioned

no resistance but together conditioned a dominant-recessive complementary infection type 2. The resulting ratio of 3 resistant:13 susceptible was observed in populations derived from 6A406 crossed with 70HN458, 6A413, Rosner, MT36-1 and 6A190.

The 6A406 genes appeared to operate differently in crosses of 6A406 with 6TA204 and 6A250. Instead of an infection type 2, the 6A406 genes appeared to condition together a fleck infection type to race C33. Individually, the 6A406 genes appeared to condition dominant type 2 infections when involved with 6TA204 in crosses, and type 4 infections when involved with 6A250 in crosses. Also, the 6A406 genes appeared to be acting both as dominants in their combined effect in crosses with 6TA204, while in crosses with 6A250, one 6A406 gene operated as a dominant and one as a recessive in their combined effect. The F_1 plants of the cross 6A406 x 6TA204 exhibited a (;)c($1^{=}$) infection type to race C33, while the F_1 plants of the cross 6A406 x 6A250 exhibited a (1)N infection type, as would be expected with the dominance relationship changing in different backgrounds.

Thus, in the population involving 6TA204, three dominant genes, each conditioning a 2 infection type, appeared to be operating, with the two 6A406 genes retaining their dominance to condition a fleck infection type in combination. In the population involving 6A250, the 6A406 genes appeared to act in combination as a recessive and a dominant to confer a fleck infection type, but individually did not condition resistance.

One difficulty with the analysis is that the susceptible x resistant cross population involving 6A406 was not tested with race C33, which makes the explanation of the resistant x resistant cross populations screened with race C33 very difficult. The distinction between infection types was also often not clear. If the resistance conditioned by

individual genes tends to be mesothetic, as certain lines seemed to suggest, then there would be a good chance of misclassification. Irregular transmission of a gene could have also confused the results.

It should also be noted that two gene complementary systems for rust resistance have rarely been proposed and, if proposed, have rarely, if ever, been proved to be true over long periods (Green, personal communication).

The behaviour conditioned by 6A406 resistance genes in different crosses could be summarized as follows:

(1) With parents such as 70HN458 (race C33):

dominant-recessive complementary type (2) infections;

individually a type (4) infection.

(2) With 6TA204 (race C33):

dominant-dominant complementary fleck type infections;

individually a dominant type (2) infection.

(3) With 6A250 (race C33):

dominant-recessive complementary fleck type infections;

individually a type (4) infection.

(4) With MT32-1 (race C17):

dominant-recessive complementary fleck type infections;

individually a dominant and recessive type (2) infection.

It is possible that, in fact, the genes of 6A406 confer a mesothetic type of resistance which would partly account for the inconsistent results. Also, several F_3 lines and F_2 plants derived from MT32-1 x 6A406 exhibited a type (2) reaction to C17 and a susceptible reaction to C33. From this and from the 13:3 segregations for C17 and the 3:13 segregations for C33 suggested previously, it appears that individually the 6A406 genes are generally more effective against C17 than C33.

The F_1 of 6A406 and 6A20 exhibited an infection type ($2^{++}_{=}$) or (x^{-})

to C33, also suggesting that individually or together in the heterozygote, the 6A406 genes conditioned a mesothetic reaction, which could have been misclassified in the resistant x resistant crosses as susceptible.

Attempts were made to isolate the resistance genes of 6A406 by backcrossing to MT32-1 while screening with C33, but the (2) type reaction appeared very difficult to recover. Only a small proportion of backcrossed plants were of type (2), even when selfed BC_nF_2 progeny were tested: a (2) to (3^+) type reaction was most frequently obtained. Selfed progeny in these cases did sometimes exhibit a (1^-) type reaction, so it may be that only in the original 6A406 background was a fleck expressed. Irregular gene transmission may have complicated the results.

Overall, it may be that two complementary or additive semi-dominant genes conferring a mesothetic reaction are operating, each alone conditioning a (2) to (4) [C17] or (4) [C33] type infection, but together in a heterozygote, conditioning a (2) [C17] or (x^-) [C33] type infection, and in a homozygote a (1^-) reaction. The sensitivity of a mesothetic resistance to different genetic backgrounds would explain the different results obtained for different crosses. No evidence of linkage appeared in the crosses involving 6A406.

Limited time and space, the difficulty of working with mesothetic resistance, and the infertility of 6A406 prevented adequate clarification of the genetic picture for this line.

(iv) Field Tests

Tests of resistance in the field were conducted for seedling susceptible F_3 and BC_1F_2 lines, parents and moderately resistant extracted lines.

Of the 146 seedling susceptible F_3 and BC_1F_2 lines derived from all

susceptible x resistant crosses involving MT32-1, none exhibited any resistance in the adult plant stage in the field to a mixture of races C33 and C17. In addition, a total of 68 seedling susceptible lines from the crosses and backcrosses involving 6A20 and the five principal resistant parents were tested to a mixture of races C33 and C17. Again, no adult plant resistance originating from the principal parents could be detected over the reaction of 6A20; however, 6A20 itself proved to be resistant in the field (Appendix XIV). Though exhibiting susceptible pustules, 6A20 carried only 5% rust, usually as a 5M type reaction, whereas MT32-1 usually was classified highly susceptible (80S). Where there were sufficient lines from a particular cross, it appeared as if a single semi-dominant gene conditioned the field resistance of 6A20 (Appendix XV). In the F_2 population of the cross 6A20 x MT32-1, grown in the field, a semi-dominant gene seemed to be operating. However, the results are far from conclusive, especially in the F_2 population, as the division of resistant and susceptible was somewhat arbitrary due to an almost continuous variation in resistance. As well, there were too few F_3 and BC_1F_2 lines to clearly differentiate between possible expected genetic ratios.

In the field tests, 6A20 remained the main exception in correlating seedling reaction to adult plant reaction. The tests of resistant parents and extracted lines indicated the relative level of seedling resistance to be closely related to the relative level of field resistance. The resistance of the parents was consistent over three years and a wide variety of races.

6A406 again proved to be an anomaly. Although it exhibited high resistance (almost immunity) in two out of three field tests, in the

third field test, it clearly exhibited moderately susceptible (MS) type pustules above the nodes. The rust epidemic in that test was particularly severe; the plants were also stressed that summer by drought and high temperatures. The young undifferentiated tissue above the nodes is particularly sensitive tissue, in any case, and stress and mitotic instability may have combined to result in these susceptible pustules in this area.* A rare virulent race such as C10 (Green, 1971b, 1975) may also account for these occasional pustules.

(v) Synthesis of Raw Amphiploids to Determine Source of Resistance

Crosses of durum x rye were made in the following parental combinations: resistant x resistant, resistant x susceptible, susceptible x resistant, and susceptible x susceptible. Generally, both the resulting polyhaploid and its subsequently doubled raw amphiploid hexaploid triticale (C_1 and C_2 generations) were as resistant to race C33 as the most resistant parent (Table 12). The reactions of the most resistant parent, the polyhaploid, and the hexaploid were essentially identical, the variations readily attributable to environmental effects. When tested with five other wheat stem rust races (C17, C35, C10, C27, 111) and a race mixture, these results held, except in the case of resistant durum wheat 4B921 x susceptible rye, in which case the resistance of the hexaploid triticale appeared to be reduced, relative to the durum (Appendix VI). However, 4B921 was the only durum used that exhibited a differential reaction, and furthermore, its resistance often appeared as an (x^-) infection type. The resulting triticale also exhibited the differential

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* It is quite common to find a few pustules just above the nodes at times on almost any genotype of wheat (Knott, personal communication).

TABLE 12. Infection Types of Eight Triticales and Their Durum and Rye Parents to Two Races and a Race Mixture of Wheat Stem Rust and to a Rye Stem Rust Isolate, RSR-224-73.

Material tested	Rust Races			
	C33	C27	1975 Race Mixture	RSR-224-73
S. cereale cv. Centeno	1	;, 1^-, 2^+	$;1^=$	1, 2, 3, 4
S. cereale "WSR susceptible rye"*	4	4	2 to 3^+	$3^{\pm}$, 4
T. turgidum var. *durum* cv. Pelissier	4^+	4	3^- to 4	;
Pelissier x WSR susceptible rye	4	4	3^- to 4	$;1^-$
Pelissier x Centeno	2	2	1 to 2^-	$;1^-$
T. turgidum var. *durum* cv. Marracos	4^+	4	3 to $4^=$	$1^=$
Marracos x Centeno	1	2^-	1 to 2^-	1 to 2, 1 to 3
T. turgidum var. *durum* (4B925)	1	;	; to $2^=$	;
4B925 x WSR susceptible rye	1 to $2^=$	;	1^- to $2^=$	;
4B925 x Centeno	$;1^=$	-	-	-
T. turgidum var. *durum* (4B921)	1	4	$1^=$ to $3^=$	;
4B921 x WSR susceptible rye	2	4	$3^{\pm}$	-
T. turgidum var. *durum* cv. Hercules	2^-N	$;1^=$	;N to 2^-N	;
Hercules x WSR susceptible rye	1	$;1^-$	;N to 2^-N	1^-
T. turgidum var. *durum* (2106)	$2^=$	1^-	1^- to 2^-	;
2106 x WSR susceptible rye	1	1^-	1 to 2^-	;

* A wheat stem rust susceptible rye selection.

reaction, but the resistance tended to shift to the more susceptible end of the reaction range of 4B921.

The resistance of the tetraploid wheats, 4B925, Hercules and 2105, as expressed in the triticales synthesized from them, varied between fleck, (1) and (2) infection types, depending on the race used, but resistance was exhibited to all races tested. The resistance of Centeno rye, as expressed in the triticales derived from susceptible durum x Centeno crosses, was more uniform than the durum resistance in its reaction to the races tested, varying between a (1) and (2) type reaction, depending on the race used.

The synthesized triticales and parents were also tested with a rye stem rust isolate. All durums exhibited a high level of resistance [fleck or ($1^{=}$)] to a rye stem rust isolate, including Pelissier and Marracos, which were susceptible to wheat stem rust. Centeno rye segregated in reaction to the isolate, (1), (2) and (4) type infections being exhibited. The triticales derived from all durums except Marracos exhibited a high level of rye stem rust resistance, similar to the durum parent. Seed from 4B921 durum x rye was unavailable for this screening. The results from screening Marracos durum x Centeno with the isolate were unusual in that the triticale exhibited an (x) infection type although Marracos itself was very resistant [($1^{=}$)]. However, Marracos was the only durum tested which exhibited pustules rather than the necrotic or chlorotic cells of a fleck infection type to the rye stem rust isolate.

The field nursery of these synthesized triticales in 1975 was not designed as a rust screening test; however, a natural stem rust epidemic

was heavy enough to distinguish some differences fairly clearly. The resistance contributed by Centeno rye was clearly evident in the cross Pelissier durum x Centeno, which exhibited a reaction of 20MR compared to an adjacent row of Pelissier durum x susceptible rye which exhibited a 70S reaction. Pelissier is widely susceptible to most stem rust races (Newton *et al.*, 1940). Marracos durum x Centeno was also highly resistant, exhibiting a 5R reaction. Hercules durum x susceptible rye and 2105 durum x susceptible rye were also highly resistant. The crosses 4B921 durum x susceptible rye and 4B925 durum x susceptible rye, however, both were in the range of 10-20 MS, which could be expected from the seedling reactions. 4B925 durum x Centeno rye was resistant (1R) as expected.

(vi) Tests for Range of Resistance for Genes Identified

The parental triticales involved in susceptible x resistant crosses were tested to further races to determine their spectrum of resistance. Moderately resistant extracted lines were also included in these tests.

Rosner, MT36-1, Beaver, 6A413, 70HN458, 6A190, 6TA204, 6A250, 24-39, 4-49, 44-9, 27-44, MT32-1, and 6A20 were consistent in reaction to races C33, C10, C35, C17 and C27 (Appendix V). Avirulent race 111 generally produced a more resistant infection type, as expected [a (2) or (1) infection type tended to be replaced by a fleck infection type]. The line 27-99 derived from the cross MT32-1 x 6A190 was resistant to C35, C17, C27 and 111 and susceptible to C33 and C10 (both these being in the 15B race grouping). Carleton durum expressed a

resistance pattern similar to that of line 27-99 (Carleton is a sister selection of Stewart, from which 6A190 was derived). Line 6A20, though being derived from Carleton, did not express the Carleton resistance to C35, C17, and C27. The resistance of 6A406 was overcome by C10.

In general, rye stem rust could not attack most triticale lines. The extremes were MT32-1 and 70HN458 (both having Armadillo parentage) which tended to exhibit an (x^-) type infection, still good resistance.

(vii) Miscellaneous Observations

(a) Screening triticales and ryes. A number of interesting observations were made while screening the University of Manitoba triticale and rye germ plasm collections.

Of the 91 hexaploid triticale amphiploids screened with race C33, 12 were classified as susceptible, and of the 111 octoploid amphiploids screened, 33 were classified as susceptible (21 of these were classified as winter types and four were derived from Kharkov). This is at best a tentative classification, as the lines tested had been clearly exposed to outcrossing or mixing. In addition, a great many of the octoploid lines were quite probably regressing to the hexaploid condition through chromosome elimination, resulting in a possible loss of resistance.

Eleven selections of different field-susceptible triticale breeder's lines were tested in the greenhouse to a number of races to determine if any race-specific genes could be detected. Only MT73-14 showed any significant resistance, and this was an intermediate reaction to C17 and C27. Screening with race 111 resulted in the detection of

some (2) type or fleck type infections in a few lines. As with other triticales, the 11 lines tended to be resistant to rye stem rust, although three exhibited a mesothetic reaction that went as high as an (x^+) in 72HN415-1.

When inbred and bulk ryes were screened with race C33, they generally expressed (2) type or fleck type infections. None of the six rye varieties (Centeno, Prolific, Argentina, Marco Juarez, Snoopy and Gazelle) tested was susceptible, although susceptible segregants could be obtained by screening a large enough population. When four of the varieties were screened with rye stem rust (RSR), a high percentage of the plants were susceptible (50 - 100%) although some plants did express a moderate resistance, mainly those from Centeno rye. The wheat stem rust (WSR) susceptible rye bulk appeared to be susceptible to both WSR and RSR, although a few resistant plants appeared in the population when screened with either rust variety. Bulk Centeno rye appeared to be about 50% susceptible to RSR, but resistant to WSR, indicating the possibility of a separate mechanism of resistance for each rust.

During the synthesis of the raw amphiploids produced in this study, the cross MT32-1 x Centeno was made, and the resulting hybrid was tested in the undoubled state with race C33. A (1^+) infection type to C33 occurred in the hybrid, indicating that the Centeno resistance was still being expressed in the ABRR hybrid.

The rye UC90 appears to transmit a moderate-type resistance to triticale as a number of Chinese Spring x UC90 doubled hybrids obtained from the Department of Plant Science, University of Manitoba, exhibited a (2) type infection when screened with C33, while Chinese

Spring was susceptible.

When wheat stem rust seedling susceptible lines of rye were exposed to a field epidemic of C17 and C33, some lines exhibited a 20S reaction, and one line segregated into 20S and 70S reactions. Gazelle and Prolific ryes expressed a trMR and 3 MR reaction, respectively.

(b) Chimaeras. Half-leaf reactions or chimaeras were detected eight times in the present study. Lines 6A190, 6A406, MT36-1 and 6TA204 were the principal parents involved. Four of the eight chimaeras involved 6A190. In six of the eight cases, it appeared most possibly to be due to the loss of only one resistance gene; i.e., the chimaeral plants were from segragating material, involving only one resistance locus, in which heterozygotes were present. In the other two cases, which involved F_3 material derived from 6A190 x Rosner, the source lines appeared to have genes from both 6A190 (gene #2) and Rosner (gene #1) segregating. In this situation the chimaeras obtained could possibly have been due to the loss of only a single chromosome if,

(a) the chimaeral plant was originally aneuploid and hemizygous for the chromosome carrying resistance,

(b) genes #3 and #5 had already been lost through normal segregation and/or were linked to the genes conferring a fleck infection type and lost with them.

Further testing of later leaves of the chimaeral plant in two of the eight cases seemed to indicate the complete loss of the locus for resistance, and testing of the progeny in these cases confirmed this.

(c) Further information on 6TA204. Line 6TA204 appeared to exhibit occasionally an unusual variation of its (2) infection type to wheat stem rust. The typical reaction was a (1) to (2^-) infection type which often varied to almost a fleck, in which case it was often rated as a ($2\equiv$) reaction. Occasionally, a plant would appear exhibiting a ($3^=$) infection type, one of which when selfed produced progeny with ($2^=$) and (2^+) to ($3^=$) type reactions (18 and 13 seedlings respectively).

In the field under artificial epidemic conditions (races C33 and C17), an occasional 6TA204 plant exhibited a 10S reaction, unlike the normal MR reaction; the seed from an unbagged head of one of these produced only normally reacting seedlings, possibly because of outcrossing that could have occurred to resistant plants. A normally reacting seedling, when grown out and selfed, appeared to produce normally reacting seedlings in the next generation, although a sample of 19 plants would not be enough to pick up unusual segregants if rare. Aneuploidy or chimaera formation could be possible explanations, which would indicate again that the presence of susceptible-appearing segregants must be treated with caution.

(d) Rye stem rust. The rye stem rust isolate #224-73 seemed avirulent on the seven durums tested and on most of the triticales used in this study. However, some susceptibility, expressed as an (x) reaction, was observed in Marracos durum x Centeno rye, 70HN458, MT32-1, 72HN195-3, MT103, 72CB692 and Little Club wheat. The most susceptibility was expressed by MT103 which exhibited a (2) to (4^-) infection type. The latter five of this group of lines are completely susceptible to wheat

stem rust; the former two are resistant to all wheat stem rust races used. The Marracos durum x Centeno rye amphiploid exhibits the (x) infection type in spite of Marracos durum being quite resistant to rye stem rust and Centeno contributing resistance to wheat stem rust. A Pelissier durum x Centeno rye amphiploid, which possesses wheat stem rust resistance contributed only by the rye was quite resistant to rye stem rust, unlike the Marracos durum x Centeno rye amphiploid.

The rye Centeno exhibited both resistance and susceptibility to both rye stem rust and wheat stem rust; as already mentioned, a far greater percentage of the variety appears to be susceptible to rye stem rust than to wheat stem rust (wheat stem rust susceptible seedlings are rare in Centeno). The wheat stem rust susceptible selected bulk was also almost entirely susceptible to rye stem rust. Prolific, Argentina and Marco Juarez were predominantly susceptible to rye stem rust and resistant to wheat stem rust.

Ten wheat stem rust susceptible BC_1F_2 lines obtained from crosses of MT32-1 with Rosner, 6TA204, 70HN458, and MT36-1, all reacted to rye stem rust similarly to MT32-1 in having a range of reaction from type (1) to (3). No fleck type reaction was obtained from these lines. A larger number of lines would be necessary to determine whether the rye stem rust resistance was always lost when the wheat stem rust resistance was lost (which would indicate linkage).

MT32-1 may in fact be a bulk of two reaction types to rye stem rust, the predominant one a type (1±), the other a type (1) to (3^-), since the seed source was a bulk from several plants. However, the distinction between the two reaction types was not clear; the apparent

differences may be due to environmental effects rather than genetic differences.

5. DISCUSSION

Although the results appeared adequate to satisfy the main objectives of this study, the interpretations must be accepted with some caution. The difficulties that may appear can be illustrated by considering the observations from testing material derived from 6A190. Meiotic instability and mitotic instability in this material may have led to the production of recombinant-type lines. 6A190 is a highly unstable line, and has been known to produce at least 20% aneuploid progeny (Larter, personal communication). The recombinant-type lines may simply be derived from F_2 plants that have lost a chromosome. Furthermore, it is possible for a resistance-carrying chromosome to be lost in mitosis, so that a tissue-section, a half-leaf, a leaf, a plant, or the progeny from a plant appear susceptible (McIntosh and Baker, 1969; Burrows, 1970). Eight chimaeras (in which a leaf was half-resistant, half-susceptible, with the midrib forming the division between the two areas) were observed in the triticale segregating material; four of these involved 6A190 in crosses, and two of these four were in F_3 material from the cross 6A190 x Rosner (the other four examples involved 6A406, MT36-1, and 6TA204). It is possible that this mitotic loss could have occurred earlier in the development of the plant, resulting in seedlings being classified as susceptible. These difficulties indicate the problem with resistant x resistant crosses in at least some triticales; the large populations necessary to distinguish tightly-linked genes may also increase the probability of chromosome loss leading to the inappropriate identification of recombinants.

The present study leads to the consideration of the potential for triticale to offer significant resistance to stem rust in the future.

Stem rust may be able to attack triticale either because the resistance genes have been lost or because the pathogen has evolved virulence to otherwise resistant genes. Virulence could evolve easily enough if indeed all that is required is a mutation to an inactive gene (Knott, 1967). As well, widely virulent hybrids could evolve through assembly of virulence genes from both wheat stem rust and rye stem rust, through natural hybridization (the initially avirulent hybrids could be maintained on wild grasses and go through further intercrossing, backcrossing and selfing and eventually lose the dominant avirulence genes). If triticales with the appropriate combination of the corresponding rye and wheat resistance genes were available, then these hybrid pathogen isolates would be selectively increased and further reassortment thereby enhanced. The result could be a stem rust variety specialized on triticale, or widely virulent on wheat and triticale, or rye and triticale, or all three, depending on how widely virulent the original parental *secalis* and *tritici* populations were. Presumably, a widely virulent hybrid would have to acquire some fitness or aggressiveness characteristics before it could displace the predominant wheat stem rust races. On the other hand, if triticales were not grown in an area and the predominant available host (other than the wild grasses) was either wheat or rye (thus lending no selective advantage to hybrid races), reversion of the hybrids to wheat stem rust or rye stem rust would probably occur (although a few virulence genes on the other host might be retained).

Lopez (1971) obtained field isolates of rye stem rust with virulence on a few triticales and a few wheats, and wheat stem rust with virulence on certain triticales and ryes. The 19 isolates of stem rust he collected from triticale in the field appeared to be wheat stem rust, although 11 of them could attack a few ryes and when screened on a number of triticales, the isolates could attack some triticales. The broader host range of certain of these isolates appears due to additional virulence genes, which could have been acquired through hybridization of *tritici* with *secalis*.

In the present study, the ability of wheat stem rust to attack certain ryes and triticales appears to be due to a loss of host resistance, as the particular cultivars are widely susceptible. Such triticales should be screened out of breeding programs with relative ease. However, hybridization in breeding programs may lead to a loss of genes conditioning resistance to *P. graminis secalis*, as it is unlikely there will be an effective screening with rye stem rust. If most wheat genomes carry resistance to *P. graminis secalis*, however, this may be a rare occurrence. In any case, where rye and triticale acreages remain limited, rye stem rust epidemic development will also be limited.

If triticale becomes widely grown, it will undoubtedly shift the composition of stem rust populations by selectively increasing mutations and recombinants virulent on lines carrying resistance genes from rye, especially if the resistance on the A and B genomes is identical to that of the commonly grown wheats. This would create a threat to rye by increasing the potential inoculum load of virulent races. The chance of further hybridization of the hybrids with rye stem rust also would be increased, perhaps resulting in wheat stem rust races more tolerant of cooler

temperatures and more able to overwinter in cooler climates, thereby accelerating epidemic spread.

The use of hexaploid wheat in hexaploid triticale breeding may be building up common wheat resistance genes in triticale populations, making it much easier for the pathogen evolving on bread wheat to extend its range to triticale and rye. However, even if only durum-rye triticales were used in a bread wheat area, it is probable that the double insurance of having two distinctly different resistance sources from the predominant wheat grown would be lost, as it would be difficult to maintain both the durum and rye resistance in a breeding program if no races exist to detect them.

Alternatives to specific resistance may exist. Because the whole or major part of the rye genome is incorporated into triticale, the fine physiologic host-pathogen balance for wheat stem rust may be disrupted by a number of small gene effects, thereby rendering wheat stem rust less fit on triticale than on wheat. This would be a form of nonspecific resistance and could be useful in breeding programs if it could be detected.

SECTION III

GENERAL DISCUSSION

SUGGESTIONS FOR FURTHER RESEARCH

SUMMARY AND CONCLUSIONS

1. GENERAL DISCUSSION

1A. Difficulties of Genetic Studies with Triticale

In a genetic study of triticale, several problems can be expected, including aneuploidy, F_1 instability, chimaeras, infertility, outcrossing, abnormal reactions to stress, distorted segregations, problems with D genome substitutions, and heterozygosity.

Sampling from bulk seed sources may lead to sampling aneuploid seed, as 10 - 15% of the seed may be missing a chromosome (Tsuchiya, 1974). Furthermore, since there could be as many as 50% abnormal metaphase cells, the use of a resistant triticale as a female parent increases the risk of loss of a resistance-carrying chromosome. The use of triticale as a pollen parent lessens the problem of aneuploidy in that the certation effect results in predominantly "euploid" pollen being effective, lessening the probability of a loss of a resistance-carrying chromosome. However, an aneuploid F_1 or F_2 plant having lost a susceptible allele and being monosomic for a resistant allele, may produce an overabundance of resistant progeny because of the certation effect. The increased instability of F_1 plants may further increase the proportion of aneuploid progeny and of abnormal segregation in F_3 lines. As well, certain resistance genes may appear to segregate abnormally if located on particularly unstable chromosomes.

Although there may be a significant proportion of aneuploidy, chances would still be very small that the particular critical resistance-carrying chromosome would be lost. Any distortion of results would only become critical when screening larger populations in attempting to find rare recombinants. The larger populations increase both the probability

of detecting a true recombinant and the probability of detecting a false aneuploid "recombinant". Thus, resistant x resistant crosses would have definite limitations.

The presence of the occasional chimaera in which a resistant chromosome is lost mitotically would again critically affect resistant x resistant crosses. Mitotic loss would compound the effects of aneuploidy where meiotic loss of one resistance-carrying homologue was followed by mitotic loss of the other resistance-carrying homologue, leading to incorrectly classifying a plant as susceptible.

The use that was made of resistant parents as pollen parents in susceptible x resistant crosses in this study would mitigate against loss of a resistant chromosome. Pollen deficient for a resistance-carrying chromosome would tend to be ineffective, whereas deficient egg cells would tend to be effective in fertilization (Scoles and Kaltsikes, 1974). In backcrosses, also, the susceptible parent was used as a female and the resistant F_1 as the male. Thus, there was some assurance of retaining the resistance in crossing and backcrossing. However, if the susceptible homologue were lost in producing an F_1 or BC_1F_1, an excess of resistant progeny could result. Also, in a backcross in which a resistance-carrying chromosome had no homologue due to a D substitution in the other parent, then the resistance-carrying chromosome could be more frequently transmitted. However, if pollen carrying the D but not R homeologue was more competitive, there might then be an excess of susceptible progeny. Since the recurrent parent MT32-1 probably carried a D substitution[1] (Gustafson, personal communication),

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[1] The parents of MT32-1 both appear to carry a 2D-2R substitution (Merker, 1975).

and since the rye component seems important in contributing resistance, such a bias for particular chromosomes involved in resistance could conceivably occur. Such a situation might also arise in resistant x resistant crosses involving Beaver, MT36-1, Armadillo and the single-gene lines extracted from susceptible x resistant crosses involving MT32-1. This would distort the ratios, but probably not inferfere with the conclusion that the two sources of resistance carried different independently-segregating genes (the exception being that both the D and R homeologues might carry resistance and all F_2's would appear resistant, if there was a strong certation effect).

Aneuploidy in susceptible x resistant crosses can be partially counteracted by screening the plants used in crossing, and the F_1's to ensure the resistance is retained, and by replicating a particular cross using different resistant plants. A further precautionary measure would include keeping the individual F_1 plant progenies separate so as to be able to detect abnormal segregations, as was done in this study. This procedure would also assist in eliminating hybrids derived from resistant plants which were heterozygous due to outcrossing.

Stress such as heat or drought could increase the problem of aneuploidy, particularly if the F_1's are grown under such conditions. In the present study the F_1's were advanced under cool winter greenhouse conditions.

The infertility of triticale, which is increased by stress and aneuploidy, can limit the amount of seed available for sufficiently large F_2 populations and F_2 plant progenies for adequate sampling. As well, because of tricicale's tendancy to outcross, F_1's and F_2's may be contaminated by foreign pollen. In this study the F_1's were bagged,

but the F_2's were left unbagged so that the proportion of segregating F_3 lines might have increased from outcrossing at the expense of susceptible lines. The degree to which this occurs would vary greatly, and would be most critical in susceptible x resistant crosses in which both parents were infertile.

Aneuploidy and infertility should prove more critical in genetic studies of the more unstable octoploids, since rye chromosomes are preferentially lost (Scoles and Kaltsikes, 1974). This would be especially significant in that the stem rust resistance would be expected to be derived predominantly from the rye.

1B. Triticale Resistance

Previous studies have indicated that the rye resistance to wheat pathogens can be transferred to wheat. Several European wheats derive their leaf and stem rust resistance from rye chromosome 1R, possibly through Petkus rye. Acosta's wheat translocation lines derive stem rust resistance from Imperial rye (chromosome 3R), Transec derives leaf rust resistance from chromosome 2R of rye and Jensen and Kent (1952) reported in winter wheat an adult plant resistance to leaf rust which was conditioned by a semi-dominant gene derived from Rosen rye. Riley and Macer (1966) reported expression of rye resistance to stem rust in a Holdfast-King II amphidiploid but could not detect stem rust or leaf rust resistance derived from rye in any of the addition lines derived from this cross. Quinones (1972) reported that the three ryes he used in triticale synthesis did not contribute resistance to leaf rust. The present study demonstrated that rye can contribute resistance to wheat stem rust, although crosses obviously can occur in which no resistance is transferred,

due to the heterozygous nature of rye. The triticale 6A190 must also have stem rust resistance derived from rye, as its wheat progenitor Stewart is susceptible to 15B and 6A190 carries two genes for resistance to 15B.

It is curious that Riley and Macer, and Quinones did not detect any transfer of leaf rust resistance from rye to wheat. Several explanations may be offered for this:

(1) the rye lines they used could have been heterozygous and they sampled only the susceptible rye gametes,

(2) the resistance transferred may not have been detectable till a later stage in development,

(3) in Quinones' polyhaploids, the single dose of resistance may have been insufficient [in Jensen and Kent's (1952) study, there seemed to be a dosage effect],

(4) if a resistance gene is indeed a switch gene (i.e., activator of a metabolic pathway conditioning resistance), then certain resistance genes from rye may not be able to "switch on" the wheat pathways,

(5) in Quinones' study, only ryes susceptible to rye leaf rust were used. A rye resistant to both rye and wheat leaf rust may have given different results,

(6) the rye resistance to wheat leaf rust could be a nonspecific resistance, the wheat leaf rust being highly integrated to the wheat metabolic system but unadapted to the rye metabolic system. Such resistance might be hard to transfer, as interaction between the wheat and rye genomes might disrupt it or the wheat metabolic system might predominate. Leaf rust does appear to be more closely integrated to the wheat metabolic system than wheat stem rust, as wheat leaf rust

is more specialized (restricted to fewer species and fewer plant parts) and more aggressive (spreads under cooler temperature). Perhaps, the rye component of triticale does decrease the aggressiveness of wheat leaf rust, but this would be less easily detected than hypersensitive resistance.

As well, because the wheat stem rust pathogen is more critically damaging (i.e., to stem tissue) than wheat leaf rust, the selection pressure for hypersensitive specific resistance in rye may have been greater for stem rust. Wheat leaf rust may simply have lost the ability to attack rye by becoming too specialized.

In the present study, a mesothetic durum reaction to wheat stem rust was occasionally shifted to a more susceptible reaction upon synthesis into a triticale; in one case, an infection type ($1^{=}$) produced on the durum in response to rye stem rust shifted to a more susceptible infection type. One explanation is that the rye stem rust isolate is heterozygous for a semi-dominant avirulence and triticale synthesis "desensitizes" the host to the already decreased product of the avirulence gene, through dilution or disruption of the sensing process. Day (1974) suggests that mesothetic reactions result when the competition between induced resistance and induced susceptibility is equally likely to be resolved in either direction for each local interaction. This balance could be shifted or disrupted by triticale synthesis.

MT32-1 and 70HN458 also exhibited a mesothetic reaction to rye stem rust. This may result from decreased effectiveness of the durum resistance genes or from the loss of common durum resistance genes in the original wide cross from which Armadillo (and its derivative, MT32-1) was derived. However, the Armadillo lines Lopez (1971) tested

to rye stem rust were highly resistant. Different test environments and isolates might account for this discrepancy.

As has been mentioned, 6A190 appears to derive two resistance genes from rye. The third, race specific, gene then must apparently be the Vernal emmer gene that was incorporated into Mindum to give Stewart. 6A20, derived from Carleton, a sister selection of Stewart, should have the same gene, but apparently has derived no significant seedling resistance from either wheat or rye. 6A20 does exhibit a slight resistance in the form of a chlorotic (3^{++})-type pustule in seedlings; and in the field 6A20 is resistant to both 15B and 56. It would thus appear that the rye has contributed adult plant resistance and that the Vernal gene has been lost or rendered ineffective.

From Sanghi and Luig's (1971) investigations, it is apparent that wheat carries genes that condition resistance to both wheat and rye stem rust, and genes conditioning resistance to only rye stem rust or only wheat stem rust. In the present study there is some indication that the *P. graminis tritici* resistance genes in triticale may also condition resistance to rye stem rust.

In the synthesis of triticales in this study it was observed that the wheat stem rust susceptible durums carried resistance to rye stem rust. Furthermore, the ryes could carry resistance to both *secalis* and *tritici*, although usually only a small proportion of a rye bulk was resistant to *secalis*, while almost the entire bulk usually was resistant to *tritici*. This would seem to indicate that one or more widespread rye genes condition resistance to *tritici*, while others contribute resistance to *secalis* or both *secalis* and *tritici*. Durum genes and rye genes each contributing widespread resistance to

secalis and *tritici* may be the most valuable resistance source in triticale breeding. If the rye genes in triticale conditioning resistance to wheat stem rust are overcome by virulent rye stem rust genes, then it seems that widespread culture of triticale may assist in the transfer of those virulence genes to wheat stem rust. Thus, screening for *secalis*-resistant ryes may be useful.

Although transfer of virulence genes from *secalis* to *tritici* through hybridization may be possible, several factors do mitigate against this:

(1) *secalis-tritici* hybrids are avirulent and, therefore, are restricted to susceptible wild grasses and barley and must compete with more aggressive adapted isolates and with the large inoculum load emanating from the cultivated crops. (The large number of factors controlling avirulence result in most of the hybrid progenies being avirulent).

(2) Green (1971a) suggests that a widened host range is associated with a loss of aggressiveness (e.g., stem rust is widely virulent but not aggressive on barley). Hence, even if hybridization, selfing and backcrossing revealed enough genes to attack both wheat and rye, the hybrid isolates would still be unable to compete with the stem rust varieties specialized on these crops. However, the availability of triticales susceptible to the hybrids but resistant to the parental varieties would allow the hybrid rust isolates to increase without competition and there would then be more opportunity for further adaptation. Triticales with complex resistance different than the predominant wheats and ryes, or for which no virulence existed in natural populations, present a barrier against this evolution.

It would seem that if triticales with only one source of resistance are grown widely, then the stem rust hybrids could easily become specialized and aggressive on triticale. However, from Green's comments, it would seem unlikely that a rust could evolve with widespread virulence and aggressiveness on all three of wheat, rye and triticale. To evolve aggressiveness, it would seem necessary to specialize and adapt to a particular host's physiologic pathways. On the other hand, a wheat stem rust race could extend its virulence to a few triticales and ryes but remain aggressive on the wheat and unaggressive on the triticales and ryes. There would be little selective advantage to the organism in evolving widespread aggressiveness and virulence if triticale and rye remained minor crops.

Lopez (1971) suggested designating a new variety, *P. graminis triticalis*, to cover isolates with a broad host range on wheat, rye and triticale, but it would seem that the categories *secalis* and *tritici* would be adequate at present to classify his isolates.

The CIMMYT triticale breeding program is tending to produce a number of very wheat-like triticales, as the result of D substitutions, and these could well represent a host on which wheat stem rust could easily extend its virulence without loss of aggressiveness. Certainly, the more minor resistance or protective genes that are stripped away from the major resistance genes, the easier it will be for the pathogen to overcome the resistance. The European wheats with a translocated stem rust resistance gene from rye represent the extreme in this regard; if grown on wide acreages, they may very easily shift the rust population to a broader range of virulence. Examples of this process already exist, as with the breakdown of the *Aegilops* resistance in Transfer wheat

derivatives once they were used widely. Similarly, the emmer resistances widely used in both durums and bread wheats were broken down simultaneously by race 15B.

As triticale seems to be adapted to high to moderate rainfall, moderate climate areas, leaf rust may be more of a threat than stem rust. Nevertheless, the moisture levels will undoubtedly favour the spread of stem rust; and triticale is also being developed in such severe stem rust areas as Kenya. If triticale comes into use in the Mediterranean countries, the simultaneous widespread culture of durums in these areas may make it much easier for wheat stem rust evolving on durums to make the jump to triticale. In Europe and the U.S.S.R., where rye culture is widespread, large reservoirs of virulence on the rye resistance genes are likely to exist, so that triticale resistance may be threatened in these areas. If winter triticales come into widespread use in Texas, *secalis-tritici* hybridization and specialization may be encouraged, possibly increasing the aggressiveness of stem rust in cool weather on winter wheats, and possibly posing a threat to ryes grown north of the winter wheat area. As well, both rye and durum are commonly grown in the Dakotas, and triticale resistance in this area would be less secure. In other areas of the world, where durums and ryes are little used, triticale resistance may provide significant protection against stem rust.

However, the ease with which stem rust has evolved virulence in the past dictates caution in evaluating the effectiveness of triticale resistance. Triticale has the advantage of having potentially complex resistance derived from both highly resistant durums and ryes, and also the advantage that protective genes will not have been easily stripped

away, so that the pathogen may be rendered less aggressive. It may be wise to preserve some of the nonspecific resistance by maintaining the full rye complement, avoiding introgression of bread wheat germ plasm, and selecting deliberately for nonspecific as well as specific resistance. (It is tempting to suggest that the leaf rust problems in triticale are due to bread wheat introgression). Maintaining a high level of resistance in wheat, rye and triticale would minimize the rust population size and opportunities for mutation and evolution of virulence. In this regard, areas carrying a reservoir of inoculum on susceptible hosts should be minimized (particularly in overwintering or oversummering areas). As well, widespread use of a single triticale variety should be avoided; care must be taken to use different sources of resistance in different regions. As varieties are released, some effort should be made to ensure the resistance is unique and preferably complex; releasing varieties with single resistance genes should be avoided. It is important that the effectiveness of triticale resistance not be considered in isolation, but rather, in relation to crop patterns and the distribution of resistance.

1C. Evolution of Resistance and Virulence

Green (1971a) suggested that stem rust evolved from a form widely pathogenic and moderately virulent but non-aggressive on a number of gramineous hosts, into specialized forms with high virulence and aggressiveness on a limited number of hosts. This pattern of evolution of virulence on certain hosts presumably happened at the expense of virulence on other hosts.

Watson and Luig (1962) suggested that rye stem rust is simply an

avirulent form of wheat stem rust, being restricted to rye.

Such rust specialization on rye during evolution would have been encouraged because the earliness of rye would allow an aggressive pathogen to exist at high levels of virulence without seriously damaging rye. There would be little selection pressure for specific resistance to rye stem rust. On the other hand, since rye originally evolved as a weed in wheat fields, there would be considerable selection pressure for specific resistance against wheat stem rust, but little selective advantage for wheat stem rust to extend its virulence onto rye resistance genes.

Riley and Macer (1966) also suggested that as the original diploid progenitors of wheat and rye diverged, different resistance genes became fixed in the diverging populations, the differentiation of wheat and rye stem rust being determined by these original sources of resistance. They also suggested that the resistance of rye to wheat pathogens could also have come from the side-effects of genes with other functions which evolved during the divergence, or from response to selection pressure by the pathogen.

Recent evolution of rye may have been towards a loss of minor gene nonspecific resistance, as hybridization and selection broke up ancestral complexes of specific and nonspecific genes and artificial selection concentrated only on specific resistance.

2. SUGGESTIONS FOR FURTHER RESEARCH

Experiments on the aggressiveness of stem rust isolates on various triticales lacking specific resistance would be useful to determine if the addition of a whole rye genome to wheat renders the pathogen less aggressive through nonspecific resistance. The rapidity of spread from a central point in a small solid plot of triticale might be used as a measure of aggressiveness (Differences in maturity could complicate such an approach).

Synthesis of a number of triticales lacking specific resistance would also be useful in providing material for such an experiment.

Screening ryes for specific resistance to both wheat and rye stem rust could also be useful in identifying genes with wide-spectrum resistance that could be transferred to triticale.

A further investigation of the genetics of resistance of 6A20 is also necessary, as the actual number of genes controlling its resistance is still in doubt. Also, the resistance could be transferred to a line more useful for breeding purposes.

Synthesis of a number of triticales from susceptible durums and various resistant ryes could be useful in trying to identify a number of different rye genes for resistance.

It might be useful to determine whether the D chromosome substitutions carry any resistance, and if so, whether it is wide-spectrum resistance.

The identification of durums which have demonstrated world-wide resistance, and the subsequent incorporation of these resistance genes into triticales would be useful.

Further clarification of the rye contribution to triticale leaf rust resistance is necessary.

3. SUMMARY AND CONCLUSIONS

The factors investigated in this study included the inheritance of wheat stem rust seedling resistance in several triticales, the rye and durum contributions to triticale stem rust resistance, the range of triticale resistance using several races, and the expression of the resistance in adult plants under field conditions. F_2 populations and BC_1F_2 and F_3 lines were used to study inheritance. "Raw" amphiploid triticales were synthesized to determine the source of resistance. All these triticales were tested as seedlings with the two most important races in Western Canada in the last 50 years, 15B and 56 (Green, 1971b), as well as a number of other races including a rye stem rust isolate, from which a more complete picture of the range of resistance was determined. Finally, the parents (and derived lines carrying resistance) which underwent inheritance studies were grown under field conditions to relate seedling resistance to resistance in the mature plant.

The triticale resistance was mainly dominant (except for the partial dominance of 6A406) and the inheritance relatively simple (one to three genes); complementary resistance was possibly operating in one line. The genes identified conditioned a non-differential resistance to a number of races, there being one clear exception (the race-specific gene of 6A190). The resistance genes were distributed into at least four linkage groups, with two of these groups each including three tightly linked, allelic or identical genes. A number of lines or varieties had identical genes (four of the nine lines carried one particular gene). Most of the susceptible lines of

triticale tested were susceptible to all races used.

The resistance of triticale to wheat stem rust appeared to originate from both wheat and rye, the level of triticale resistance being that of the most resistant parent (when neither parent carried resistance, the resulting triticale was susceptible). The triticale resistance derived from Centeno rye appeared to operate against a wide variety of races. The triticale resistance derived from some durums operated against only certain races, while the resistance from other durums operated against a wide variety of races.

The seedling reaction of the triticales tested appeared to be a good indication of the resistance of adult plants. Adult-plant resistance genes (i.e., genes operating only in the adult stage) appeared to be absent in parental lines with seedling resistance genes. Seedling susceptible 6A20, however, exhibited resistance in the field, mainly through a restriction of the amount of rusted tissue occurring.

P. graminis secalis seemed avirulent on the durums and triticales; however, the mesothetic reaction of the Marracos durum x Centeno rye amphiploid indicated that the durum resistance to rye stem rust may be modified during triticale synthesis.

SECTION IV

LIST OF REFERENCES

APPENDIX

1. LIST OF REFERENCES

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A P P E N D I X

APPENDIX I

Formula Numbers, Virulence Formulas, Corresponding Physiologic Races and Isolate Numbers for the Main Races Used in the Study.

Formula number	Virulence formula (effective/ineffective host genes)	Physiologic race	Isolate used
C35 *	9d, 9e, 10, 11, 13, 17, 22, Tt2/5, 6, 7a, 8, 9a, 9b, 14, 15, 16	32-113	111-70
C33 **	6, 9a, 9b, 13, 15, 17, 22, Tt2/5, 7a, 8, 9d, 9e, 10, 11, 14, 16	15B-1L	42-71
C10 ***	6, 7a, 8, 22, GB/5, 9a, 9b, 9d, 9e, 10, 11, 13, 14, 15, 17, Tt2, 16	15B-1	121-69
C17 ****	6, 8, 9a, 9b, 9d, 9e, 11, 13, 17, 22, Tt2/5, 7a, 10, 14, 15, 16	56	33-71
C27 [1]	6, 8, 9e, 11, 17, Tt2/5, 7a, 9a, 10, 15, 16	33	43-71
111 (111 x 36)[2] WSR 2179	6, 8, 9a, 9b, 9d, 11, 14, Prelude, Marquis/5, 7, 10, 13, Little Club	111	--
RSR #224-73	--	--	224-73

* Moderately virulent on Manitou and Neepawa, but not sufficiently aggressive to cause important damage.

** Most prevalent race in Western Canada presently.

*** C10 is the original 15B subrace in Canada; it is now rare (Green, 1971b).

**** Present from 1931-1969; predominated from 1935-50, 1957-60; more aggressive and competitive than 15B at high but not low temperatures.

[1] Attacks most resistance genes, but not sufficiently prevalent to do harm.

[2] 111 x 36 No. 5-4-1-2-1-23. Derived from a cross between avirulent race 111 and widely virulent race 36 (Loegering and Powers, 1962; Kao and Knott, 1969).

APPENDIX II

Race Composition of the Mixtures of Wheat Stem Rust Used for Greenhouse Screening of Seedlings, or for Field Testing of Adult Plants at Glenlea, C.D.A.

Races	Race mixtures and composition			
	1972 Epidemic	1973 Epidemic	1974 Epidemic	1975 Epidemic
C 1 (17)	x			x
C 2 (17A)	x	x	x	x
C 5 (29-1)	x			
C 9 (15B-1L)				x
C10 (15B-1)	x	x	x	x
C11 (15B-4)	x			
C14 (38)		x	x	x
C17 (56)	x	x	x	x
C18 (15B-1L)	x	x	x	x
C20 (11)	x	x	x	x
C22 (32)	x	x	x	x
C25 (38)	x	x	x	x
C33 (15B-1L)	x	x	x	x
C35 (32-113)	x	x	x	x
C38 (15B-1L)	x	x	x	x
C41 (32-113)	x	x	x	x
C42 (15)				x
C44 (15B-1L)	x	x	x	
C46 (15B-1L)		x	x	x
C49 (15)				x
C52 (32-113)	x	x	x	x
C54 (38)			x	x
C56 (38-151)				x

APPENDIX III

Explanation of infection type symbols, according to Stakman *et al.* (1962).

Infection type[a]	Varietal Reactions and Reaction Classes
	Resistant
0	IMMUNE. No uredia nor other indications of infection.
0;	NEARLY IMMUNE. No uredia, but hypersensitive flecks present.
1	VERY RESISTANT. Uredia minute, surrounded by distinct necrotic areas.
2	MODERATELY RESISTANT. Uredia small to medium; chlorotic or necrotic halos surround green islands, in the centre of which the uredia are usually located.
	Susceptible
3	MODERATELY SUSCEPTIBLE. Uredia of medium size and usually separate. Necrosis absent but chlorotic areas may surround the uredia, especially under unfavourable conditions.
4	VERY SUSCEPTIBLE. Uredia large and often coalescing from large irregular pustules.
	Mesothetic
x	HETEROGENEOUS. Uredia variable, sometimes including all infection types.

[a] The symbols $^{-}$, ±, $^{+}$ indicate quantitative variations in types of uredial infection.

APPENDIX IV

Explanation of field classification of infection types,* according to the methods used by the U.S.D.A. International Rust Nursery.

0 = No visible infection on plants.

R = Resistant. Necrotic areas with or without minute uredia present.

MR = Moderately resistant. Small uredia present surrounded by necrotic areas.

M = Intermediate. Variable sized uredia, some with necrosis and/or chlorosis.

MS = Moderately susceptible. Medium uredia with no necrosis but possibly some distinct chlorosis.

S = Susceptible. Large uredia with no necrosis and little or no chlorosis present.

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* Field readings also include an estimate of the relative percentage of rust infection, with the maximum 37% of actual surface covered being assigned the figure 100% and with tr = trace.

APPENDIX V

Seedling Reactions of 16 Triticale Cultivars and Lines and One Durum Cultivar to Several Races of Wheat Stem Rust and to a Rye Stem Rust Isolate (RSR-224-73).

Gene being expressed	Line	Race and Seedling Reaction							
		C33	C10	C35	C17	C27	111	Race mixture 1975	RSR
I-1	Rosner	;	;	;	;	;	0	;	;
	MT36-1	;	;	;	;	;	0	;	;
	Beaver	;	;	;	;	0	0	;	;
	6A413	;	;	;	;	;	0	;	;
I-2	70HN458	;$_c1^-$	1^-	;1^-	;$_c1^=$	;	;	;$_c1^=$	1 to 3^-
I-2	6A190	;	;1^-	;	;	;	0	;$_c1^=$	;N 1-N
II-3	24-39	$2^=$	$2^=$	2	2^-	2^-	;1	2^-	$1^\pm$
II-3	6TA204	1 to $2^=$	$2^=$	$2^=$	1 to 2^-	1	1	$2^=$	;1^-
II-3	6A250	2^-	1 to 2≡	1 to 2≡	1	2^-	;N 1^-	$2^=$	;1≡
III-4	4-49	2	2^-	2	1	;1^-	;	2	;1
	44-9	2	2^-	2	1	1^-	;	2	;
IV-5	27-44	2	2	2^-	2^-	1	;	$2^\pm$	1 to 2
-6	27-99	4	4	2^-	2^-	$2^=$	;	2 to 4	-
-7, 8	6A406	;N $1^=$	3^+	;	;$1^=$	;	;	; to $3^=$	; 1^-N
-	MT32-1	4	4	4	4	4	2 to 3^+	4	1^- to $3^=$
-	6A20	3^{++}	4	$3^\pm$	3 to 3^{++}	2^{++} to $3^\pm$	;1^-	3^- to 4	;
-	Carleton durum	4	4	1^+N	1N	1N	;N	; to 4	;

APPENDIX VI

Seedling Reaction of Eight Triticales and Their Durum and Rye Parents to Several Races of Wheat Stem Rust and to a Rye Stem Rust Isolate (RSR-224-73).

Material tested	Race and Seedling Reaction*							
	C33	C17	C35	C10	C27	111	Race mixture	RSR-224-73
Centeno rye	1**	1**	;, ;$1^{=}$, 1, 2	1	;, 1^{-}, 2^{+}	;1^{-}	;$1^{=}$	1, 2, 3, 4
Susceptible rye	4	3, $3^{\pm}$, 4	3	4	4	2, 4	2 to 3^{+}	$3^{\pm}$, 4
Pelissier durum	4^{+}	4	4^{+}	4^{+}	4	3^{+}	3^{-} to 4	;
Pelissier x susceptible rye	4	4	4^{-}	3	4	3^{+}	3^{-} to 4	;1^{-}
Pelissier x Centeno	2	2^{-}	1-	1^{-}	2	2^{-}	1 to 2^{-}	;1^{-}
Marracos durum	4^{+}	4	4^{+}	4	4	3^{+} to 4	3 to $4^{=}$	$1^{=}$
Marracos x Centeno	1	1	1^{-}	1^{-}	2^{-}	2^{-}	1 to 2^{-}	1 to 2, 1 to 3
4B925 durum	1	;$1^{\equiv}$	;$1^{=}$	1, 1 to $3^{\equiv}$	* ;	;	; to $2^{=}$	;
4B925 x susceptible rye	1 to $2^{=}$	;$1^{\equiv}$	;1^{-}	* 1 to $2^{\pm}$	;	;	1^{-} to $2^{=}$	;
4B925 x Centeno	* ;$1^{=}$	-	;1	* 1	-	-	-	-
4B921 durum	1	1 to $3^{=}$	1	1^{+}, $3^{\equiv}$	4	1 to 2^{-}	$1^{=}$ to $3^{=}$	;
4B921 x susceptible rye	* 2	1 to 3	* 2 to 3	* $3^{\equiv}$	4	2 to 2^{+}	$3^{\pm}$	-
Hercules durum	2^{-}N	;N 1^{-}N	1	;N 1^{-}N	;$1^{=}$	;N $1^{=}$N	;N to 2^{-}N	;
Hercules x susceptible rye	1	;$1^{=}$	;1^{-}, 1	;1^{-}	;1^{-}	;$1^{=}$	;N to 2^{-}N	1^{-}
2106 durum	$2^{=}$	1	2^{-}	1^{-} to 1^{+}	1^{-}	;	1^{-} to 2^{-}	;
2106 x susceptible rye	* 1	1	1	* $1^{=}$ to 1^{-}	1^{-}	;	1 to 2^{-}	* ;

* The seedling reaction was generally based upon 5-20 seedlings/line, but out of the 122 screenings, in 10 cases, only 1-4 seedlings were tested due to a shortage of seed. Also, the testing period ranged over two months with fluctuating greenhouse conditions so that small differences between race reactions may be only apparent and not real.

** Selfed progeny of a rye plant used in crossing.

APPENDIX VII

Seedling Reactions of Several Selected Field-susceptible Triticale Lines and Little Club Wheat to Several Races of Wheat Stem Rust and a Rye Stem Rust Isolate (RSR-224-73).

Line	Source	Race and Seedling Reaction							
		C33	C10	C35	C17	C27	111	Race mixture	RSR
MT32-1	Armadillo x Rosner	4	4	4	4	4	2 to 3+	4	1^{-} to $3^{=}$
6A20	Carleton durum x *S. cereale*	3^{++}	4	3^{+}_{+}	3 to 3^{+}_{+}	2^{+}_{+} to 3^{+}_{+}	$;1^{-}$	3^{-} to 4	;
MT17-3	Beaver x Armadillo	4	4^{-}	4	4	4	2^{+}	$3^{\pm}$	1
MT16-4-2	Beaver x Armadillo	4	3^{++} to 4	4^{-}	4^{+}	4	2	3 to 4	$;1^{-}$
72HN195-3	(6A303 x 6A271) x Rosner	4	3^{++} to 4	4^{+}	4^{+}	4^{+}	$3^{\pm}$	3	1 to 3^{+}
72HN395-2	MT84	4	4	4^{+}	4^{-}	4^{+}	2 to 2^{+}_{+}	3^{-}	$1^{+}c$
72HN415-1	MT103	4	4	3^{++}	4	4	3^{-}	$3^{\pm}$	2 to 4^{-}
1972 Bulk 1-2-1	Bulk Selection 1972	4	2^{+}_{+} to 3	4	3^{++}	4	3^{-}	3	1N to 2N
72CB1611	MT90	4	4^{++}	4^{+}	3^{++}	4	1	$3^{\pm}$	;
72HN65-2	(Beaver x Armadillo) x 71CB61	4	4^{-}	4	3^{++}	4^{+}	2, 3	$4^{=}$	$;1^{=}$
72CB692	[UM940'S' (Tc1 MY64/Per-Dicds-Crlt)] x Bulk MY69	4^{+}	4^{+}	4	4^{+}	4	3 to 4	3^{+}	1N to $3^{=}$N
72CB1354	John x Rosner	4^{+}	3^{-} to 3	3^{++}	3^{++}	4	2	3^{+} to 4	;
MT73-14	S-1063	3	3^{++}	4	2^{+}	2	;1	3^{+}	$;1^{=}$
Little Club Wheat	*Triticum compactum*	4	4	4^{+}	4	4	4	4^{+}	1 to 3

APPENDIX VIII

Greenhouse Seedling Reactions and Adult Plant Field Reactions of Resistant and Susceptible Parents, for Two Years and Two Locations*, to Wheat Stem Rust.

Gene being expressed	Parental line	Rust race and Seedling reaction		Year, location, races used, and field reaction		
		C17	C33	1973 CDA Race Mixture	1974 CDA Race Mixture	1974 UM C17 + C33
I-1	Rosner	;	;	1R	5R	0
I-1	MT36-1	;	;	1R	trR	0
I-1	Beaver	;	;	1R	trR	0
I-1	6A413	;	;	3R	trR	0
I-2	70HN458	;$c1^{=}$	;$c1^{-}$	5R	trR	0
I-2	6A190	;	;	trR	0	trR
II-3	6TA204	1 to 2^{-}	1 to $2^{=}$	trR	trMR	3MR
II-3	6A250	1	2^{-}	20MR	20MR	30MR
- 7, 8	6A406	;$1^{=}$	1N $1^{=}$	trR	0	2MS***
-	MT32-1	4	4	80S	80S	80S
-	6A20	3 to 3^{++}	3^{++}	5M	0**	5M

* The locations were the C.D.A. (Canada Department of Agriculture) rust nursery at Glenlea, Manitoba, and the Plant Science research plots at University of Manitoba, Winnipeg, Manitoba.

** Infection was light in this nursery, causing a lower reading.

*** The MS pustules occurred above the nodes; otherwise, the plants were clean.

APPENDIX IX

Greenhouse Seedling Reactions and Adult Plant Field Reactions to Races C17 and C33 of Wheat Stem Rust, for a Number of F_3 Lines Which Appear Homozygous or Segregating for a Single Gene.

Gene	Line	Source	Race and seedling reaction		Year, location, races and field reaction
			C17	C33	1974 UM C17 + C33
I-1	24-15	MT32-1 x Rosner	;, 4	;, 4	0, 70S
	4-21	MT32-1 x MT36-1	;, 4	;, 4	0, 20MS
	44-36	MT32-1 x Beaver	;, 4	;, 4	0, 20S
I-2	27-84	MT32-1 x 6A190	;, ;$1^{=}$, 4	;, 4	0, 70S
II-3	24-39	MT32-1 x Rosner	2^{-}	$2^{=}$	20MR
III-4	4-49	MT32-1 x MT36-1	1	2	5MR
	44-9	MT32-1 x Beaver	1	2	3MR
IV-5	27-44	MT32-1 x 6A190	2^{-}	2	2MR
-6	27-68	MT32-1 x 6A190	$2^{=}$	3^{++}	50S
?	37-17	MT32-1 x 6A406	1^{-}, 1, 1^{+}	3^{+}, 4	40S
?	37-12	MT32-1 x 6A406	2^{-}	3^{+}, 4	30-40S
?	37-87	MT32-1 x 6A406	;1^{-}, 2^{-}, 4	3, 4	30S, 70S

APPENDIX X

A Comparison of Field Reaction to Stem Rust for a Number of Seedling Susceptible Lines of Triticale, and for Several Lines of Rye, and Little Club Wheat, Over Two Years and Two Locations.

Line	UM 1973 Natural Epidemic	UM 1974 C17 + C33	CDA 1973 Race Mixture	CDA 1974 Race Mixture
MT32-1	5-15S (ripened early)	70S	80S	80S
6A20	trS	5M	5M	1MS
MT17-3	10S	30S	-	70-80S
MT16-4	30S	40-50S	70S	70-80S
72HN195-3	30MS, 60S	70S	70MR	50MS
72HN395-2	60S	40S	-	60-70S
72HN415-1	5S	40S	-	40S
1972 Bulk Sel.	25S	50S	-	70-80S
72CB1611	15S	40S	-	80S
72HN65-2	30S	50S	40S	50S
72CB692	5-10S	60S	40S	70S
72CB1354	30S	60S	70S	80S
MT73-14	0	20S	70S	30S
S-533	10MS	40-60S	70S	90S
72HN196-1-1-1	30S	80S	90S	-
Little Club Wheat	-	50S	-	-
Susceptible rye				
Selection #1	-	40S	-	-
#2	-	20S	-	-
#3	-	10M	-	-
#4	-	20S	-	-
#5	-	20S, 70S	-	-
Gazelle rye	-	0, trMR	-	-
Prolific rye	-	3MR	-	-

APPENDIX XI

Data on Field and Seedling Reactions to Stem Rust of Several Durums, Ryes and Triticales Synthesized from These Durums and Ryes.

Line	Field Reaction Natural Epidemic UM 1975	Field Reaction Artificial Epidemic UM 1974 C33 + C17	Field Reaction Artificial Epidemic CDA 1974 Race Mixture	Seedling Reaction Race C33
Susceptible rye selection	-	20S, 70S	-	4
Centeno rye	-	-	-	1
Pelissier	-	30S	70S	4+
Pelissier x Centeno	20MR	-	-	4
Pelissier x susceptible rye	70S	-	-	2
Marracos	-	30S	90S	4+
Marracos x Centeno	5R	-	-	1
4B925	-	-	-	1
4B925 x Centeno	1R	-	-	;1=
4B925 x susceptible rye	20MS	-	-	1 to 2=
4B921	-	-	-	1
4B921 x susceptible rye	10MS	-	-	2
2105	-	-	-	2=
2105 x susceptible rye	0	-	-	1
Hercules	-	-	-	2-N
Hercules x susceptible rye	trMR	-	-	1
4B909	-	5MR, 5MS	-	2, 2 to 4
6A406	-	2MS	0	;N1=
RD121-9	-	60S	-	4
6A413	-	0	trR	;
Carleton	-	40S	15S - 50S	4
6A20	-	5M	0	3++

APPENDIX XII

Segregation of Reaction to Race C33 Among F_3 Lines Derived from Resistant x Resistant Crosses.

Cross and Reaction Type	Number of F_3 lines							Test Ratio	P
	R			Seg.			Sus.		
	(;)	(2)	(;)(2)	(;)(2)(4)	(;)(4)	(2)(4)	(4)	Res.:Seg.:Sus.	
Rosner (;) x 70HN458 (;)	59	0	0	0	0	0	0	37:26:1	< .001
Rosner (;) x 6TA204 (2)	31	23	29	0	0	0	0	37:26:1	< .001
6A190 (;) x Rosner (;)	81	0	1	1	1	0	0	175:80:1	< .001
Rosner (;) x MT36-1 (;)	109	0	0	0	0	0	0	175:80:1	< .001
6TA204 (2) x 70HN458 (;)	20	5	15	23	2	17	1	7:8:1	.20-.10
6A190 (;) x 70HN458 (;)	93	0	0	1	1	1	0	37:26:1	< .001
70HN458 (;) x MT36-1 (;)	65	0	0	0	0	0	0	37:26:1	< .001
6A190 (;) x 6TA204 (2)	25	16	28	18	3	9	0	37:26:1	.05-.01
6TA204 (2) x MT36-1 (;)	29	9	36	17	2	14	0	37:26:1	.05-.01
6A190 (;) x MT36-1 (;)	71	0	3	2	2	0	0	175:80:1	< .001

APPENDIX XIII

Screening Results for Several Races Inoculated onto the Seedling Susceptible Lines Originally Obtained by Screening the BC_1F_2 Lines with C17*.

Source of lines	Number of lines	Race and Number of Lines Tested**						Rust reaction
		C17	C33	C10	C35	C27	111	
MT32-1[2] x Rosner	6	6	6	6	6	4	4	all susceptible[1]
MT32-1[2] x 70HN458	4	4	4	4	4	4	4	all susceptible
MT32-1[2] x 6TA204	6	6	6	6	6	5	5	all susceptible
MT32-1[2] x 6A190	3	3	3	3	3	-	1	all susceptible
MT32-1[2] x MT36-1	5	5	5	5	5	5	4	all susceptible

* Only those C17 susceptible lines with sufficient seed for further screening were retained.

** Changes in numbers for a particular resistant parent are due to running out of seed.

[1] Susceptible here meaning equal to or more susceptible than the reaction of MT32-1 to a particular race.

APPENDIX XIV

Field Reaction of Seedling Susceptible F_3 and BC_1F_2 Lines to a Mixture of Races C17 and C33.

Resistant parent	Cross or line	Crosses involving MT32-1			Cross involving 6A20		
		Lines tested	Lines field susceptible	Infection rating	Lines tested	Lines field susceptible	Infection rating
Rosner	MT32-1 x Rosner	8	8	80 - 90S			
	6A20 x Rosner				2	2	40 - 50S
	MT32-1[2] x Rosner	6	6	80 - 90S			
	6A20[2] x Rosner				2	2	2M - 50S
	MT32-1 Checks			80S			80S
	6A20 Checks						2-5M
70HN458	MT32-1 x 70HN458	20	20	50MS - 90S			
	6A20 x 70HN458				7	7	30MS - 90S
	MT32-1[2] x 70HN458	6	6	80 - 90S			
	6A20[2] x 70HN458				10	10	5M - 80S
	MT32-1 Checks			70 - 90S			70 - 80S
	6A20 Checks						5M
6TA204	MT32-1 x 6TA204	21	21	40S - 90S			
	6A20 x 6TA204				14	14	5M - 60S
	MT32-1[2] x 6TA204	12	12	80 - 90S			
	6A20[2] x 6TA204				20	20	5M - 30S
	MT32-1 Checks			70 - 90S			70 - 90S
	6A20 Checks						5MR - 5M
6A190	MT32-1 x 6A190	1	1	60S			
	6A20 x 6A190				1	1	5MS
	MT32-1[2] x 6A190	7	7	50 - 80S			
	6A20[2] x 6A190				2	2	20MS - 80S
	MT32-1 Checks			60S			60S
	6A20 Checks						3-4M
MT36-1	MT32-1 x MT36-1	5	5	50 - 80S			
	6A20 x MT36-1				1	1	60S
	MT32-1[2] x MT36-1	9	9	60 - 80S			
	6A20[2] x MT36-1				9	9	20MR - 60S
	MT32-1 Checks			60 - 80S			80S
	6A20 Checks						10MR - 3M
6A413	MT32-1 x 6A413	25	25	70 - 90S			
	MT32-1 Checks			60S			
Beaver	MT32-1 x Beaver	4	4	70 - 80S			
	MT32-1 Checks			60S			
6A250	MT32-1 x 6A250	15	15	50 - 90S			
	MT32-1 Checks			60S			
6A406	MT32-1 x 6A406	7	7	60 - 80S			
	MT32-1 Checks			60S			

APPENDIX XV

Segregation of the Adult Plant Resistance Found in 6A20, to an Artificial Field Epidemic of Races C17 and C33 of Wheat Stem Rust, among Seedling Susceptible F_3 and BC_1F_2 Lines from Susceptible x Resistant Crosses.

Source of lines	Expected ratio	Number of lines		Reaction of segregating and susceptible lines and checks	P expected ratio
		Resistant (5M)	Segregating and Susceptible		
6A20 x 6TA204	1:3	4	10	10M - 60S	1.0
$6A20^2$ x 6TA204	1:1	11	9	10M - 40S	.90 - .70
$6A20^2$ x MT36-1	1:1	5	6	10M - 40S	1.0
6A20	-	-	-	3MR - 5M	-
MT32-1	-	-	-	60S - 90S	-

APPENDIX XVI

Half-leaf Chimaeral Reactions and the Crosses, Generations and Race Combinations Where They Occurred.

Cross	Race	Generation	Line	Line reaction	Chimaeral reaction
6A190 x Rosner	C33	F_3	3-2	$\frac{75}{76}$ (;), (;) (1^-)	½ (0) ½ (4)
6A190 x Rosner	C33	F_3	3-64	$\frac{80}{81}$ (0), (;)	½ (;) ($1^=$) ½ (4)
MT32-1[2] x 6A190	C17	BC_1F_2	47-10	$\frac{5}{45}$ (;) ($1^=$) $\frac{25}{45}$ (1), (2) $\frac{14}{45}$ (4)	½ (2^-) ½ (4)*
MT32-1[5] x 6A190	C33	BC_4F_1	-	$\frac{17}{19}$ (4) $\frac{1}{19}$ (2)	½ (2) ½ (4)
MT32-1 x [F_3 plant from MT32-1 x 6A406][1]	C33	F_1	-	$\frac{13}{22}$ (2±) $\frac{8}{22}$ (4)	½ (2) ½ (4)**
6A406 x 70HN458	C33	F_2	-	$\frac{24}{34}$ (;) $\frac{9}{34}$ (4)	½ (;) ½ (4)
MT32-1 x MT36-1	C17	F_3	4-38	$\frac{38}{52}$ (1) $\frac{13}{52}$ (4)	½ (1) ½ (3)
MT32-1 x 6TA204	C17	F_3	17-78	$\frac{21}{29}$ (1) $\frac{7}{29}$ (4)	½ (1) ½ (4)

[1] The resistant F_3 plant was from line 37-73 which was segregating for resistance to C17 and C33.

* The reaction on the 1st leaf also occurred on the 3rd leaf, but the 2nd and 4th leaves expressed only a susceptible reaction on both sides of the midrib. Selfing produced only 34 susceptible progeny.

** The second leaf of this plant was completely susceptible to C33. When selfed, the resulting F_2 plants were all susceptible and when backcrossed to MT32-1, the 8 BC_1F_1 plants were all susceptible.

APPENDIX XVII

CLARIFICATION OF DATA FROM SUSCEPTIBLE X RESISTANT CROSSES

[SECTION II-4(i)]

The backcrosses involving Rosner segregated in a ratio of 3 segregating lines: 1 susceptible with both races, indicating that two genes are involved (Table 3). This was confirmed by the F_3 lines which segregated in a 7 resistant:8segregating:1 susceptible ratio with race C17 (Table 2). The backcrosses to MT32-1 were tested with both races and each BC_1F_2 family was either segregating to both or susceptible to both (Table 5). Thus, each gene conditions resistance to both races. In the backcrosses and in the F_3 lines, some lines segregated only for a fleck infection type, some only for a type 2 infection and some for both. Thus, one gene controls a fleck infection type and the other a type 2 infection. The F_1 plants from the Rosner crosses exhibited a fleck infection type to race C33, indicating that the gene involved is dominant. From the cross MT32-1 x Rosner, an F_3 line (24-39) homozygous for the gene conditioning the 2 infection type, was selected. This line was crossed to MT32-1 and the F_1 plants exhibited a 2 infection type, showing that the second gene is also dominant.

The backcrosses involving 70HN458 segregated in a ratio of 1 segregating line:1 susceptible line with race C17, indicating that one gene was involved (Table 3). Fewer of these lines were available for testing with C33 than with C17, as a number of lines had insufficient seed for testing with both races. The backcross lines tested with C33 did not fit

a 1:1 ratio (Table 3), apparently due to chance; the lines lost due to insufficient seed were all susceptible to C17.

That one resistance gene was operating in 70HN458 against C17 was confirmed by the F_3 lines which segregated in a 1:2:1 ratio (Table 2). The BC_1F_2 lines that were tested to both races were either segregating to both or susceptible to both, in a 1:1 ratio (Table 5), indicating that a single gene conditions resistance to both races. The resistance was expressed basically as a fleck infection type. The F_1 plants from the 70HN458 crosses exhibited a fleck infection type, indicating that the gene involved is dominant.

The backcrosses involving 6TA204 segregated in a ratio of 1 segregating line:1 susceptible line with both races, indicating that a single gene is involved (Table 3). This was confirmed by the F_3 lines which segregated in a 1:2:1 ratio with both races (Table 2). The backcross lines and F_3 lines derived from the cross MT32-1 x 6TA204 were screened with both races, and each line was resistant to both, segregating to both or susceptible to both (Tables 4 and 5). Thus, the gene conditions resistance to both races. The resistance was expressed as an infection type 2. The F_1 plants from the 6TA204 crosses exhibited a type 2 infection, indicating that the gene involved is dominant.

The backcrosses involving MT32-1 and 6A190 segregated in a ratio of 7 segregating lines:1 susceptible with race C17, indicating that three genes are involved (Table 3). This was confirmed by the F_3 lines derived from MT32-1 x 6A190, which segregated in a 37:26:1 ratio with race C17 (Table 2). When testing with race C33, however, the backcrosses involving MT32-1 and 6A190 segregated in a 3:1 ratio, indicating that only two genes

are involved in conditioning resistance to race C33 (Table 3).

When testing the backcross lines involving 6A190 and MT32-1, it was observed that half of the ten lines which were susceptible to race C33 were segregating in reaction to race C17, indicating that one of the resistance genes of 6A190 conditioned resistance to only race C17 (Table 5). Similarly, for the seven C33-susceptible F_3 lines derived from MT32-1 x 6A190, 2/7 were resistant to race C17, 4/7 segregating and 1/7 susceptible, again indicating a gene conditioning resistance to C17 only (Table 4). The segregation of reaction of backcross lines to both races C17 and C33 was consistent with the assumption of two genes each conditioning resistance to both races and a third differential gene conditioning resistance to only race C17 (Table 5).

The F_3 and BC_1F_2 lines resistant or segregating to race C17 and susceptible to race C33 exhibited only type 2 infections to C17 on resistant plants, indicating that the differential gene conditions a type 2 infection with race C17. In the BC_1F_2 and F_3 lines screened with C33, some lines segregated only for a fleck infection type, some only for type 2 infection and some for both. Thus, one gene controls a fleck infection type and the other a type 2 infection.

The two genes in 6A190 conditioning moderate resistance to race C17 were each isolated separately in the homozygous condition in F_3 lines 27-44 (conditioning infection type 2 to both races C17 and C33) and 27-99 (conditioning infection type 2 to race C17 and infection type 4 to race C33). The F_1 involving MT32-1 and line 27-44 exhibited moderate resistance to race C33, indicating dominance. Within individual F_3 lines segregating for the differential gene only, a 3 resistant:1 susceptible ratio was observed for race C17, with resistant plants uniformly exhibiting

a type 2 infection, indicating dominance.

The F_1 involving MT32-1 and 6A190 exhibited a fleck infection type to race C33, indicating dominance for the remaining gene.

The nine backcross lines derived from 6A20 x 6A190 did not fit a 7 segregating:1susceptible ratio when tested with race C17 (Table 3), possibly because of outcrossing (6A20 exhibits a fair degree of partial sterility, which would facilitate outcrossing). Certainly, the data from this cross must be considered unreliable because of the low expected class numbers.

The segregation of F_3 lines derived from MT32-1 x 6A190 did not fit the expected 7:8:1 ratio for race C33 (Table 2) or 28:8:1:24:2:1 ratio for both C17 and C33 (Table 4). In the former case, there were too few of the moderately resistant lines (five out of 70 were homozygous moderately resistant or segregating for this resistance) and too many of the susceptible lines (seven out of 70) to expect independence; the expected ratio under independence would be 3:1 as compared to the actual 5:7. A linkage value of 23.62 ± 9.32 crossover units for the two genes from 6A190 conditioning resistance to race C33 was calculated from this data. When the F_3 ratios for race C33 and races C17 and C33 together were adjusted for linkage, an adequate fit was obtained (Tables 2 and 4).

The backcrosses involving MT36-1 segregated in a ratio of 3 segregating lines:1 susceptible with both races, indicating that two genes are involved (Table 3). This was confirmed by the F_3 lines which segregated in a 7:8:1 ratio with both races (Table 2). The backcross lines and the F_3 lines derived from the cross MT32-1 x MT36-1 were screened with both races, and each line was resistant to both, segregating to both or susceptible to both (Tables 4 and 5). Thus, each gene conditions

resistance to both races. In the backcrosses and in the F_3 lines, some lines segregated only for a fleck infection type, some only for a type 2 infection, and some for both. Thus, one gene controls a fleck infection type and the other a type 2 infection. The F_1 plants from the MT36-1 crosses exhibited a fleck infection type to race C33, indicating that the gene involved is dominant. From the cross MT32-1 x MT36-1, an F_3 line (4-49), homozygous for the gene conditioning a 2 infection type, was selected. This line was crossed to MT32-1 and the F_1 plants exhibited a 2 infection type, showing that the second gene is also dominant.

The F_3 lines derived from the cross MT32-1 x Beaver segregated in a ratio of 7 resistant lines:8 segregating lines:1 susceptible line with race C17, indicating that two resistance genes are involved (Table 2). Some F_3 lines segregated only for a fleck infection type, some only for a 2 infection type and some for both. Thus, one gene controls a fleck infection type and the other a 2 infection type. The F_1 plants of the cross MT32-1 x Beaver exhibited a fleck infection type to race C33, indicating that the gene conditioning a fleck infection type is dominant. Segregation of reaction within F_3 lines tested with race C17 supported this. From the cross MT32-1 x Beaver, an F_3 line (44-9), homozygous for the gene conditioning the 2 infection type, was selected. This line was crossed to MT32-1, and the F_1 plants exhibited a 2 infection type to race C33, indicating that the second gene is also dominant. Segregation of reaction within F_3 lines tested with race C17 also supported this.

Since all the F_3 lines from the cross MT32-1 x Beaver were not tested with both races C17 and C33, it is not clear whether the two genes conditioning resistance to race C17 also conditioned resistance to race C33. However, three of these F_3 lines were tested to both races

and the reaction was consistent between races in that line 44-36 segregated for a fleck infection type only, line 44-31 segregated for a type 2 infection only and line 44-9 appeared homozygous for a gene conditioning a type 2 infection.

The F_3 lines derived from the cross MT32-1 x 6A413 segregated in a ratio of 1 resistant:2 segregating:1 susceptible with race C33, indicating that one resistance gene is involved (Table 2). The F_1 plants of the cross MT32-1 x 6A413 exhibited a fleck infection type to race C33, indicating that the gene involved is dominant. No check was made to determine whether this gene conditioned resistance to both races C17 and C33.

The F_3 lines derived from the cross MT32-1 x 6A250 segregated in a ratio of 1 resistant:2 segregating:1 susceptible with race C17, indicating that one resistance gene is involved (Table 2). The F_1 plants of the original cross exhibited a fleck infection type to race C33, indicating that the gene involved is dominant. Segregation of reaction within F_3 lines tested with C17 supported this. No check was made to determine whether this gene conditioned resistance to both races C17 and C33.