THE INHERITANCE OF RESISTANCE TO PUCCINIA RECONDITA ROB. EX. DESM. IN HEXAPLOID TRITICALE

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INTRODUCTION

Since Biffen (8) first reported on the genetic nature of plant resistance in 1905, the production of resistant crop cultivars has prevented huge crop losses. In the process, considerable understanding has been gained into the nature of host resistance, variability of plant pathogens, and on the interrelationships of host-pathogen-environment interactions (27, 32, 56, 74, 88). However, not all the problems have been solved. The control of some plant diseases through the utilization of host resistance has not been completely achieved (87). Plant pathologists and breeders have learned not to expect newly released resistant cultivars to remain resistant over long periods of time. The pathogenic population has proven very versatile in overcoming the obstacles provided by the growing of resistant cultivars. Through sexual and parasexual mechanisms of variation as well as mutation, pathogenic fungi have been able to overcome previously resistant cultivars.

Intensive cropping and the extensive use of single crop varieties over large areas is a feature of today's agriculture. This has, however, eliminated the intraspecific and intravarietal diversity that was so valuable in stabilizing the population of plant pathogens (13, 36, 41). As a result, new virulent pathogenic strains are endowed with a selective advantage which allows them to spread unchecked over a continuous host genotype over large areas. The occurrence of catastrophic epidemics especially in the small cereal grains clearly illustrates this point (13).

For these reasons, new emphasis has been placed on finding more profitable ways of using traditional types of resistance, i.e. specific resistance (13, 56, 74), and efforts are now also being directed at ex-

ploring the potential of other types of resistance, i.e. general resistance in controlling plant pathogens.

It is the general consensus that plant pathogens may never be exterminated, but losses caused by destructive epiphytotics can and must be avoided. A better understanding of the mechanisms of resistance in plant populations, of the exact nature of host-pathogen interactions, and of the mechanisms through which new pathogenic forms arise and become predominant will undoubtedly help in bringing about better crops and more food for a fast rising world population.

LITERATURE REVIEW

Specific Resistance

Although resistance of this sort has mainly been studied in connection with pathogenic fungi, it is now well established that it also operates against bacteria, viruses, insects and nematodes (1, 39, 44, 45, 91).

Specific resistance has been described as the type of resistance that protects the host from certain strains, races, or populations of a pathogen, but not from others (16, 36, 74). A multitude of names have been used for describing specific resistance including vertical resistance, seedling resistance, oligogenic resistance, hypersensitivity, protoplasmic resistance, and physiologic resistance among others (16, 36, 74).

Genetic Nature of Resistance

Specific resistance is usually conditioned by one or a few genes whose genetic behavior can be fully studied and described in so far as their spectrum of resistance, allelic relationship, linkage, pleiotropy, epistacy, and the effect of the environment or specific modifiers are concerned.

Biffen (8) was the first to recognize the genetic nature of resistance in 1905. He reported that resistance to Puccinia striiformis West., the causal agent of the stripe rust disease in wheat, followed Mendel's laws of heredity, and that it was governed by a single recessive gene pair. Subsequent studies on the nature of resistance have corroborated this fact and further extended our knowledge on the essence of host-pathogen interactions.

Host plants possessing specific resistance genes usually react

hypersensitively when attacked by incompatible strains or races of a pathogen. Stakman (86) first described the hypersensitive reaction of several cereal species when inoculated with non-compatible cultures of Puccinia graminis sp. The hypersensitive reaction was characterized in the host by a pronounced chlorosis or necrosis followed by death of the infected tissue which was triggered soon after the pathogen made physiological contact with the host plant. Death of infected host cells and immediately adjacent tissue was thought to be due to the action of the pathogen. Now, however, tissue necrosis is considered to be more of a host response to invasion by any incompatible disease inciting organism (88). The invading organism is thus isolated from the living host tissue and eventually degenerates. This type of resistance is frequently referred to as hypersensitivity, and it characterizes most of the genes that condition specific resistance. Not all resistance genes, however, induce this type of host reaction (74).

When a pathogen is comprised of several separable and stable races, specific host resistance is likely to be effective against only a few of those races. Other races of the pathogen will be characterized by their virulence against host specific resistance genes (97).

Host-Pathogen Interactions

The exact nature of the host-pathogen interaction that leads to host resistance or susceptibility was not fully understood until the work of Flor (26, 27), which led him to postulate the gene-for-gene theory. Working with flax, Linum usitatissimum L. and the causal agent of flax rust Melampsora lini Desm., Flor realized that the type of infection in a given flax host plant was the result of an interaction between two complementary genetic systems, one in the host and the other

in the pathogen. He tested F_2 populations of flax crosses, with F_2 progeny from crossed rust races and found that: when flax F_2 populations segregated for one dominant gene for resistance, the F_2 rust population segregated for one recessive gene for virulence; F_2 flax crosses segregating for two resistance genes, characterized populations of the rust pathogen with two recessive genes for virulence; three dominant genes for resistance in the host differentiated rust races with three recessive genes for virulence. This equivalence of the number of loci governing virulence in the pathogen, with the number of loci controlling resistance in the host led him to propose that during the correlative evolution of host and pathogen a complementary genetic system developed in such a way that each specific locus governing resistance in the host could be counteracted by a corresponding related locus for virulence in the parasite. Thus, a host variety carrying genes for specific resistance could only be rendered susceptible by rust races possessing genes for virulence to those specific resistance genes.

In his elucidation of the mechanisms that contribute to the evolution of host-pathogen systems Person (71) recognized that during the development of such a system two mutational events have evolutionary significance. These are: 1) mutations to resistance in the host, and 2) mutations to virulence in the parasite. Other mutations, such as host mutations to susceptibility and parasite mutations to avirulence, although expected to occur, have no selective advantage and do not persist. Person (71) pointed out that mutations for resistance in the host, or for virulence in the parasite, are not limited to any specific locus, though when a mutation for virulence occurs in the parasite its genetic expression is related to the specific resistance gene that is rendered

ineffective.

The gene-for-gene theory is generally accepted in all cases where the pathogenic population consists of several races and specific resistance is operative in the host population. This concept has stimulated research towards a better understanding on the nature of host-pathogen interactions.

Breeding Behavior of Specific Resistance Genes

Following Biffen's (8) demonstration that genes for specific resistance were inherited in a mendelian fashion, many reports followed which indicated that segregation for resistance usually fitted simple genetic ratios. There are situations, however, where resistance is attributed to more complex genetic interactions.

Allelism for Resistance. - Production of crop cultivars with useful combination of resistance genes has often been hindered because some genes occur at a single locus comprising an allelic series for resistance (21, 23, 27, 28, 33, 53, 83).

In wheat, Green et al. (33) reported that two alleles for rust resistance at the Sr7 locus could be distinguished. One allele, Sr7a, was present in the variety Kenya Farmer, and the second gene, Sr7b, was described in Marquis and Hope (53). They also reported alleles at the Sr9 locus.

Some of the genes conditioning resistance to races of P. recondita Rob. ex Desm. f.sp. tritici also consist of an allelic series at a given locus. Soliman et al. (85) recognized three alleles for rust resistance, at the Lr2 locus. Haggag (35) found four alleles or very closely linked resistance genes at the Lr3 locus. Dyck and Samborski (23) reported the presence of two alleles at the Lr14 locus.

Multiple alleles for rust resistance have also been reported in other crop species. Flor (27) found 5 loci controlling resistance to races of M. lini in flax. In maize, five loci are also known to occur for resistance to P. sorghi Schw., (83) among which 14 alleles at the Rp1 locus have been distinguished.

Unexpectedly low frequencies of recombination have sometimes been observed to occur between genes for rust resistance originally described as alleles (23, 28, 83). Conceivably, such alleles characterize a group of genes originally derived from one ancestral gene for rust resistance. By gradual differentiation they have evolved specificity of reaction to races of the corresponding rust pathogen. Recombination can be explained by assuming that such alleles belong to a complex locus governing rust reaction. In the locus the genes are arranged in tandem fashion (83). If this assumption is valid more than one allele in the coupling phase could be incorporated in the complex locus, and thus a wider spectrum of resistance could be introduced into a commercial cultivar.

Not all loci conditioning rust resistance are as complex and in some of them only two alleles, one for resistance and one for susceptibility, have been recognized. In wheat, at least 17 loci for stem rust resistance have been detected and conceivably the number will increase as more emphasis is placed on the identification of specific resistance genes. About 18 wheat leaf rust resistance loci have also been identified in wheat (22).

Dominance. - The majority of the genes conditioning specific resistance which have been investigated exhibit complete dominance (5, 6, 27, 36, 74). Normally a single gene provides resistance to a wide spectrum of races of a pathogen. Many of the new crop cultivars are known to

possess several dominant genes for rust resistance which usually act independently. The wheat cultivars Manitou and Timgalen are examples of such cultivars.

Most of the dominant rust resistance genes are usually expressed in the seedling stage, although this resistance is normally operative throughout the life of the host plant. Some cultivars, however, are susceptible in the seedling stage, but resistant at later stages of plant development. Such is the case in the mature-plant resistance to some races of stem rust carried by the bread wheat cultivars Hope, H-44 and some of their derivatives (6). Adult-plant resistance to leaf rust is reported in the cultivars Exchange, Frontana, and hybrids derived from them (24).

Some situations are known where a single resistance gene exhibits complete dominance to one rust race, but behaves as recessive to another race. This is true of the stem rust resistance gene Sr6, to races 56 and 15B, respectively (47), and of the Yr3 alleles for stripe rust resistance carried by some European wheat cultivars (57). A resistance gene may occasionally behave either as a dominant or as a recessive to the same rust race, depending upon the genetic background of the host into which the gene was incorporated (21). Resistance genes may exhibit complete dominance in one background but only incomplete dominance in another (3, 21).

Recessiveness. - Recessive resistance genes are less common. Resistance to P. striiformis has been found to be mainly controlled by recessive genes or by a polygenic system (5, 57, 64, 82). Recessive genes for rust resistance are also known to operate in corn against P. sorghi (59) and in wheat to P. recondita (25, 105).

Complementary Gene Action. - The term complementary gene action is used here to describe the relationship of two or more genes, both or all of which are necessary for the full expression of resistance. Resistance due to a complementary effect of two or more genes against some races of P. graminis Pers. f.sp. tritici Erikss. & Henn. was found in crosses with the wheat cultivars Kenya 58, Kenya 117A, Egypt NA95 (4), Gabo, Lee and Timstein (47). In oats, resistance to some crown rust races in seedlings of the cultivar Victoria is dependent on two linked dominant complementary genes (94). In corn, three recessive complementary genes are reported to condition resistance to P. sorghi (59).

Linkage and Independent Assortment. - If two or more genes for resistance are located on the same chromosome, they will segregate independently of one another only if they are 50 or more cross-over units apart. If they do not segregate at random but assort together more often than they do apart, the genes are said to be linked. If two linked genes can frequently be separated by recombination, the linkage is regarded as loose. If only a small percentage of recombinants are recovered, the genes are considered tightly linked.

Linkage of genes governing resistance to certain rust races has been detected in corn against P. sorghi (34) and P. polysora Underw. (90); in oats against P. coronata Corda f.sp. avenae Erikss. (94); in bread wheat to P. graminis tritici (56) and P. recondita (25, 105) and in flax to some races of M. lini (27). In some instances, genes for resistance to two rust species are reported to exhibit linkage (55, 56, 62, 95).

Epistasis. - When a gene at one locus masks or inhibits the expression of a gene at another locus, the first gene is considered epis-

tatic over the second. Resistance genes that condition the broadest spectrum of resistance are commonly epistatic to those conditioning a narrower spectrum of resistance (36, 83). Rust resistance genes expressed in the seedling stage normally mask the effect of adult-plant resistance genes (24).

Factors that Affect Host-Pathogen Interactions

A disease, when caused by a microbial organism, is the result of an interaction between the genetic system of the host and the genetic system of the pathogen as affected by the environment (27). When a pathogen infects a host plant, an interaction is initiated which may lead to host resistance or susceptibility. The phenotypic expression of the disease is described as "infection type" and is a measure of the host-pathogen interaction. Resistance or susceptibility are expressions of host reactions to specific races of a pathogen and are genetically controlled. By the same token avirulence or virulence are pathogenicity characters and are under genetic control in the pathogen. The phenotypic expression of host characters, resistance or susceptibility, and the phenotypic expression of pathogen characters, avirulence or virulence, cannot yet be separately determined and are therefore measured through the observation of their interaction as described by the infection type and as affected by the environment (50, 52).

Role of Temperature. - Variations in temperature are known to affect the expression of "infection types". According to Johnson (40), Waterhouse first reported that seasonal variations in climate, mainly variations in temperature and light, had a marked influence on the rust reactions of wheat, oats and barley seedlings when tested in the greenhouse. Johnson (40) also noted that fluctuations in temperature may

profoundly affect the expression of rust reaction on some host varieties. He noted that temperature exerted its effect not on the host or on the pathogen but on the "host-parasite complex". Studies by Newton and Johnson (67) on the effect of temperature and light on the reactions to P. recondita of the wheat leaf rust differential hosts showed variations in infection type in different varieties.

Specific rust resistance genes have more recently been identified and transferred singly into a common susceptible background. This has facilitated studies on the effect of environmental factors upon the expression of such genes. Loegering (50) investigated the effect of different temperatures on the host-pathogen interaction involving the Sr6 alleles in the host and the P6 alleles in the pathogen. He found a resistant reaction when the Sr6 allele interacted with the P6 allele at 60°F temperature; at 80°F, however, a susceptible reaction was obtained. Other specific rust resistance genes are also known to be temperature sensitive. Such is the case with the Lr17 and Lr18 genes for leaf rust resistance; the level of effectiveness is decreased with increased temperatures (22).

Other Environmental Factors. - Variations in the expression of infection types can also be caused by fluctuations in light intensity and duration, although the effect of light alone is difficult to separate from that of temperature. Newton and Johnson (67) mentioned work carried out by Haussebrauk in 1939 on the effect of light on the reaction of the leaf rust differential hosts when inoculated with several races of leaf rust. Haussebrauk found that reduction of light either had no effect at all, or increased resistance. The cultivars Carina and Brevit, however, varied in the degree and direction of reaction change, depending

upon specific races to which they were tested. To certain races they were more resistant under conditions of reduced light, whereas to other races they were more susceptible under the same conditions. Newton and Johnson (67) also investigated the effect of light duration and intensity on the reaction to leaf rust of the differential wheat cultivars. They showed that plants giving an intermediate reaction (moderate resistance) were more sensitive to changes than highly resistant or highly susceptible varieties. In general, they reported that there was a tendency towards susceptibility as the duration of light was longer and the intensity was higher.

Differences in soil fertility may also influence the expression of the infection type in host-pathogen interactions. According to Chester (18), Gassner and Haussebrauk were the first to attempt an experimental approach to this problem in 1934. They demonstrated that some of the leaf rust differential wheat cultivars, when grown on increased levels of nitrogen, changed the reaction types from resistant to susceptible. Some cultivars, like Malakof, however, were quite stable under all levels of nitrogen.

Modifying Genes. - Some resistance genes are altered by the presence or absence of other host genes, which by themselves have no noticeable effect on host reaction. Such modifying genes have been noticed in connection with rust resistance (3, 21, 33, 35). The effect of a modifying gene may be towards increased resistance or in the direction of susceptibility.

The effect of modifying genes can best be studied when specific resistance genes have been incorporated singly into a common susceptible background, and then compared with the original host reaction (33).

Knott and Anderson (47) reported that the resistance to race 15B of stem rust conditioned by the incompletely dominant Sr7 gene could be enhanced in the presence of genes Sr9 or Sr10, which by themselves are ineffective against race 15B. Green et al. (33) found that stem rust resistance genes Sr7 and Sr10 showed a decreased level of effectiveness when transferred by backcrossing to the variety Marquis. They attributed this loss of resistance to loss of modifiers.

The effect of modifying factors in the expression of leaf rust resistance genes has also been observed. Anderson (3) studied the breeding behavior of the Lr2a allele present in the variety Webster. He reported that this gene, when transferred into the background of Prelude, exhibited complete dominance, while when transferred into Thatcher, behaved as an incompletely dominant gene. He postulated that Thatcher carries one or more modifying genes which suppress the full dominance of Lr2a. He also found that Thatcher suppressed the level of resistance of the Bagè (Lr3) gene.

Dyck and Samborski (21) clarified the behavior of the alleles for leaf rust resistance at the Lr2 locus. They showed that the resistance conditioned by the Lr2a allele, as reported by Anderson (3), was partially inhibited in the Thatcher background. The Lr2c allele carried by the variety Carina, behaved in an almost identical manner. That is, it acted as a dominant gene for leaf rust resistance in the Prelude background and in crosses with Red Bobs, but was only partially dominant in crosses with Thatcher. The clearest demonstration of the effect of modifiers, was noted with the Lr2b allele carried by the variety Loros. Lr2b in a Prelude background behaved as a dominant gene; in crosses with Red Bobs it exhibited incomplete dominance; and in crosses with Thatcher

it behaved as a recessive gene. This reversal of dominance was explained by assuming that the expression of the resistance gene, when in a heterozygous state, was controlled by the genetic background of the susceptible parent used in the cross.

Dyck et al. (24) also found that the level of resistance of the Lr12 and Lr13 genes for adult plant leaf rust resistance carried by the varieties Exchange and Frontana, respectively, could be enhanced by the presence of modifying genetic factors.

Use of Genetic Resistance in Breeding Crop Cultivars

Since Biffen (8) first demonstrated that resistance to plant pathogens was genetically controlled, the production of resistant cultivars has been strongly advocated by plant pathologists and breeders as the least expensive way of combatting plant diseases. Specific resistance genes have frequently been used in producing resistant cultivars. Nevertheless, this kind of resistance has frequently proven unreliable because plant pathogens have great plasticity and are able to produce new virulent strains identifiable by their virulence on previously resistant cultivars. As a consequence, a repetitious cycle of producing a resistant variety to prevalent races of a pathogen and the discovery sooner or later of new virulent races that can attack the previously resistant variety, has developed (41, 87). Yet, the combination of more and more genes for resistance into newer cultivars has become a common procedure.

Cabbage is perhaps one of the first cultivated species where specific resistance genes were incorporated through plant breeding. According to Stakman and Harrar (88), Jones and his associates undertook the task of incorporating resistance to Fusarium oxysporum f. conglutinans, the causal agent of cabbage yellows, into cabbage in 1909. They started

by selecting plants that survived in heavily infested soil and by a program of cross pollination and progeny testing on infested soil they eventually obtained several lines that possessed a suitable resistance to the pathogen. In 1916 they released the cabbage variety Wisconsin Hollander descended from these lines. The resistance of this variety was found to be unstable, particularly at high soil temperatures (97). Studies on the nature of resistance indicated that this cultivar was made up of at least two genotypes for resistance. Some lines possessed a single dominant resistance gene (96), while others carried more complex factors, probably a system of minor genes (2). Continuous selection by breeders, has resulted in more stable resistance by the incorporation of both resistance systems in the same genotype. Wisconsin Hollander is still grown successfully in some areas of the U.S.A. (97).

In potatoes, Solanum tuberosum L., specific resistance genes (R genes) to Phytophthora infestans (Mont.) de Bary were first introduced from the Mexican wild potato S. demissum Lindl., about 1926 (80). Previously, all potato varieties belonged to the species tuberosum proper and lacked genes for specific resistance. Hence, all were susceptible to P. infestans (30, 63), although some cultivars possessed a degree of so called "field resistance" which will be discussed later in connection with general resistance.

The incorporation of R-gene specific resistance through breeding, was followed by a period of great expectations. In greenhouses and experimental nurseries, R resistant plants reacted hypersensitively when the foliage was inoculated with the prevalent population of P. infestans. The infected tissue became necrotic and the pathogen was prevented from becoming established. The first potato cultivars that were released

carrying such resistance proved almost immune when exposed to the then prevalent population of the pathogen, probably races 0 and 4 according to Van der Plank (74). After a blight-free period of 2 to 3 years, it was discovered, however, that R resistant cultivars became susceptible to new strains of P. infestans possessing genes for virulence previously undetected in the pathogenic population (10, 11, 74).

The first R resistant cultivars possessed the R_1 resistance gene and the first P. infestans race identified by its virulence on R_1, was named race 1, according to the system for naming races proposed by Black et al. (12), where a race is named after the R specific gene it overcomes. Race 2 appeared when potato cultivars possessing the R_2 gene were released and so on.

Potato breeders soon noticed the extreme and unexpected versatility of the pathogen. When cultivars with only one R gene were released, the first races of P. infestans to appear possessed only one gene for virulence that was specifically related to the host resistance gene that was overcome. When cultivars with two R resistance genes were produced the pathogen counteracted with races possessing the two related genes for virulence, and cultivars with three genes for resistance have been rendered susceptible by races possessing the three corresponding genes for virulence (30, 74).

Studies on the breeding behavior of the R resistance genes have shown that such genes exhibit complete dominance and assort independently (9, 60). A total of nine independently inherited R resistance genes have been described from S. demissum and one from S. stoloniferum Schlecht. (29). Most of the pathogenic races that are able to render ineffective these resistances have already been identified (29, 37, 74).

This situation illustrates a fact already noted by Black (10) that resistance through hypersensitivity may never give adequate protection to potato cultivars, due to the rapidity with which the pathogen changes its races when under selective pressure created by the R resistant host population.

In wheat, Triticum aestivum and T. turgidum spp., resistance to the three major pathogens P. graminis tritici, P. recondita and P. striiformis, depends mainly on specific resistance genes, a fact already discussed.

Incorporation of specific resistance genes to P. graminis tritici has absorbed much of the effort in wheat breeding programs in Australia, North America (Mexico, U.S.A., Canada), Kenya, and practically in every country where wheat is an important crop (42) and stem rust is a threat.

Serious attempts to produce varieties resistant to P. graminis were undertaken in the U.S.A. after the 1916 stem rust epiphytotic that destroyed the wheat crop in the cereal belt of U.S.A. and Canada. The cultivar Ceres, released in 1926, was a direct product of such efforts (88). This cultivar replaced Marquis until the stem rust epidemic of 1935 caused by race 56. The cultivars Thatcher was released in 1934 (88) and it carried the genes Sr5 and Sr16 (32, 46) for specific resistance which provided good protection to race 56. Those genes were ineffective against a strain of race 15B, first isolated in 1939, but Thatcher remained free of rust until 1950 when race 15B suddenly became prevalent and attacked it. In 1953 and 1954 this same race nearly destroyed the bread wheat (Thatcher) and durum wheat (Stewart) crop of Minnesota, Dakotas, and the Canadian prairie provinces (19, 32). In 1954, the cultivar Selkirk containing resistance gene Sr6 was released

by the Canadian breeding program, and it eventually became the most important bread wheat cultivar in the spring wheat area of U.S.A. and Canada (51). The Sr6 gene provided good protection against the then prevalent races of P. graminis despite the isolation of races 15B-3 CAN. and 15B-5 CAN. with virulence on Selkirk. For some unknown reason these strains never became prevalent (32). Selkirk also carried resistance to leaf rust in genes Lr10 and Lr16 and it was the breakdown of this resistance, coupled with the fact that agronomically better varieties became available, that prompted its retirement from cultivation.

Selkirk is one of the few varieties in which specific resistance genes provided effective protection to a cultivar over a relatively long period of time. Eventually it was replaced in Canada by the cultivars Manitou and Neepawa, without ever having sustained any losses from stem rust. The persistence of the Selkirk resistance has been attributed to a combination of the specific resistance of the Sr6 gene, and general resistance, perhaps derived from H-44 through its parent Redman (16). Green (32), however, argued that this is not the case, and he noted that Selkirk, in addition to the Sr6 gene, also carried some specific adult-plant resistance derived from H-44. In Canada, the currently grown bread wheat cultivars, Manitou and Neepawa, are becoming increasingly susceptible to some strains of leaf rust and eventually they will need to be replaced by newer resistant cultivars; as we noted earlier, the never-ending vicious circle continues.

Wheat breeding in Australia has followed a similar pattern to that in North America and elsewhere. Prior to 1938, the cultivated wheat varieties were susceptible to some of the then prevalent "wild type" strains of P. graminis though they possessed some specific resistance

(56). In 1938 the variety Eureka containing gene Sr6, was released and proved highly resistant to race 126-6,7 of P. graminis. This race had prevailed in Australia and New Zealand for about 15 years (56). A few years after the release of Eureka, a new form of race 126-6,7 was identified by its ability to attack this cultivar. This new form was designated race 126-1,6,7 according to the system of race classification developed by Watson and Luig (103). The cultivar Gabo containing gene Sr11, followed Eureka, and in turn was followed by the detection of a new strain of race 126 that had virulence against Sr11, designated race 126-2,6,7. Eureka and Gabo were followed by other cultivars, all of which carried different single dominant genes for resistance. In each case, the pathogen developed strains that were virulent to the specific genes used in the breeding program (56, 103).

In 1964, cultivars with more than one gene for resistance, such as Mendos which contains genes SrTt, Sr11, and Sr17, began to be released (56). But again the pathogen evolved new virulence and the resistance of Mendos was overcome with the appearance of race 21-2,3,4,5,7. Since then varieties with four or five genes for specific resistance such as Gamut containing genes Sr6, Sr9b, Sr11, SrG5, and Timgalen containing genes Sr5, Sr6, Sr8, SrTt, and SrT have been developed. At the present time no strains of P. graminis virulent on these cultivars have been isolated from field collections, although laboratory cultures have been obtained to which Timgalen is susceptible (56).

Genes for specific resistance have also been used in oats, Avena sativa L., against its two major pathogens P. graminis avenae and P. coronata avenae (13, 14). The release of resistant oat cultivars has likewise been paralleled by the appearance of races of the pathogen

carrying genes for virulence that match the specific genes for resistance possessed by the cultivars (14, 61).

The list of crop species where genetically controlled disease resistance has been incorporated through breeding is very great. This kind of resistance has lessened considerably the risks of catastrophic epiphytotics. Nevertheless, the pathogens are constantly evolving in response to the selection pressures imposed by resistant cultivars.

Recently, attention has been turned towards another kind of resistance which operates against the entire pathogenic population, and although it does not confer immunity to a host, it does limit damage caused by the parasite by restricting its development in space or time. This type of resistance has been described as general or generalized resistance (10, 16, 36, 63, 74, 92).

General Resistance

By definition, general or generalized resistance, affords the host protection against all races or populations of a pathogen (16, 36, 74). General resistance does not involve a differential interaction between varieties of the host and races of the pathogen as is the case with specific resistance. As a rule, cultivars possessing general resistance are scored as susceptible when tested in the greenhouse, but when grown in the field they show some degree of resistance. This kind of resistance has been described as "field resistance" in potatoes and it operates against the entire pathogenic population of P. infestans (30, 63). Other names that have been applied to general resistance are: horizontal resistance, partial resistance, nonhypersensitive resistance, nonspecific resistance, minor gene resistance, and quantitatively inherited resistance (16, 30, 36, 74, 92).

Nature of General Resistance

The exact mechanism of general resistance has never been fully understood. Stevenson et al. (89), postulated that in potatoes it is governed by multiple recessive genes. Caldwell (16) stated that this type of resistance may be a manifestation of few or several interacting plant characters. Van der Plank (74) advanced the theory that it is the interaction of genes not particularly concerned with the resistance mechanism per se, that is, of genes that govern ordinary processes in healthy plants. It is known, for instance, that cultivars of S. tuberosum which do not possess any R-gene specific resistance and therefore are not immune to blight, differ significantly in the degree of resistance they possess (49, 63, 74, 92). This variation is not found in the infection type but rather in the time of appearance of disease symptoms and the speed of disease development to epidemic proportions. Muller and Haigh (63) reported that potato varieties which possessed high levels of field resistance had a lower "probability of becoming infected per unit area of foliage" when compared with susceptible varieties. The probability of a leaf becoming infected depends upon the: source and amount of inoculum, persistence of water on the leaf surface, and the susceptibility of the leaf per se (49). Lapwood (49) reported that the potato varieties Majestic and Arran Viking, when grown in the field, blighted slower than Up to Date and King Edward cultivars. This was mainly due to fewer spores produced per lesion on the former cultivars, rather than to greater resistance of leaves to infection or longer generation time by the fungus.

In oats, Krull et al. (48) reported that some cultivars bear significantly smaller uredia when attacked by P. graminis avenae under field

conditions. They suggested that such varieties possess some "partial resistance" to the pathogen. Some varieties of corn that are susceptible in the seedling stage to P. polysora and/or P. sorghi possess general resistance (74). However, since corn is an outbreeder, it is much more difficult to evaluate resistance owing to the greater genotypic heterogeneity present in this crop.

General resistance may or may not occur alone in crop cultivars. In potato varieties derived from S. tuberosum proper, general resistance was the only protective defensive mechanism possessed by some cultivars (63). When the R-gene for specific resistance was incorporated into S. tuberosum from S. demissum, the two kinds of resistance were likely brought together in some of the initial crosses. However, general resistance was masked by the effect of the R-genes which are epistatic. In many instances general resistance was lost when breeders selected strongly in favor of phenotypes that were immune or hypersensitive to attack by P. infestans. In corn, the two kinds of resistance may naturally occur together since resistance to P. sorghi and/or P. polysora is controlled by specific resistance genes as well as by some degree of general resistance (36, 74).

Factors that Influence the Expression of General Resistance

Plant Age. - Crop cultivars possessing some degree of general resistance and devoid of genes for specific resistance, are normally susceptible when naturally exposed or artificially inoculated in the seedling stage. This has been reported in potatoes (31), corn (74) and perhaps in some wheat cultivars (16). However, as the plants grow older, they become more resistant up to about the flowering stage when resistance is at its peak. After flowering, resistance slowly declines and

the plant becomes susceptible at the onset of senescence.

Nutrition. - High levels of nitrogen in the soil condition susceptibility in crop cultivars with general resistance (92), perhaps as a consequence of the more luxurious growth that plants attain at high levels of nitrogen. Main and Gallegly (58) reported that potato varieties with general resistance appeared fully susceptible when grown in soil high in nitrogen, whereas the same varieties showed resistance under normal growing conditions. Lowings and Acha (54), nevertheless, noted that under certain conditions, high levels of nitrogen increased resistance in some potato cultivars, perhaps by delaying the onset of senescence.

Temperature. - Little is known about the effect of temperature on the expression of general resistance. Walker (97) reported that there are two types of resistance to cabbage yellows caused by F. oxysporum f. conglutinans. One type is of the specific kind and is controlled by a single dominant gene. The second type is polygenic in nature and possibly a kind of general resistance. This type of resistance is not fully expressed in young cabbage seedlings but it does appear in older plants, and it is suppressed at soil temperatures of 25°C or higher. In general, it is possible that high temperatures affect general resistance in the same manner as specific resistance, that is, at higher temperatures the host tends to become more susceptible.

Variability in Plant Pathogens

The extreme variability of plant pathogens is clearly exemplified by the many reports in the literature on the appearance of new pathogenic races that render previously resistant crop varieties susceptible. In addition to variation resulting from sexual recombination, the origin

of new races in plant pathogenic fungi has resulted from mutation, heterokaryosis and parasexual recombination.

Mutation. - Mutation, the ultimate source of variability in living organisms provides a constant flow of new genes into the species gene pool. Mutations have greater selective advantage in organisms that normally undergo sexual reproduction, because the mutated genes can then be reassorted in new combinations which can be fixed by selection (72). In organisms devoid of sexual reproduction, the capacity to store genetic variability is limited, but alternative mechanisms including heterokaryosis, parasexuality, and to a limited extent cytoplasmic inheritance, maintain genetic variability (38, 72).

Many pathogenic fungi mutate abundantly when cultured in artificial media and there is evidence that mutations also occur in nature (88). In nature, any mutation that places the parasite at a competitive advantage, such as a mutation to virulence towards a previously resistant host population, will ultimately determine the survival of the mutated form, provided such mutation does not impair the overall fitness of the mutant to survive.

A change from avirulence to virulence in plant pathogenic fungi may be explained by a one-step mutation alone if: the pathogenic phase of the parasite occurs in the haploid condition and virulence is governed by a single gene; or if the parasite is dikaryotic, but heterozygous at the locus for avirulence. Virulence as a rule is expressed as a recessive trait, and homozygosity is necessary to overcome the related host gene for resistance.

Changes in the race composition of P. infestans have been attributed to mutations (37). Reddick and Mills (77) reported that when iso-

lates of this fungus were passed through a series of host varieties with increased levels of resistance, new and more virulent races were recovered, by a process they called "adaptive parasitism". Subsequently, however, Peterson and Mills (73) referred to new races arising from repeated passage through resistant foliage as mutant forms, thereby ruling out adaptive parasitism as a mechanism through which new races of this fungus may appear.

Mutation is considered an important contributor to the origin of new races of rust fungi. Newton and Johnson (66) obtained mutants for virulence in P. graminis tritici, and Watson (98) isolated three greenhouse mutants of P. graminis different from the parental races by their ability to attack single gene lines of the host which the parental races were unable to attack. Watson and Luig (104) suggested that new Australian races of P. graminis arise by "progressive increase in virulence", brought about by a mutation in one of the dikaryotic nuclei resulting in a change from the homozygous avirulent to the heterozygous semivirulent condition. In other rust fungi, reports of mutation of loci governing pathogenicity are presented by Flor (27) for M. lini, and by Samborski (81) for P. recondita and many others.

Heterokaryosis. - Heterokaryosis in fungi is defined as the state in which two or more nuclei with different genetic factors share a common cytoplasm (20, 70).

The establishment of the heterokaryotic condition may come about through: mutation; fusion of vegetative cells carrying unlike nuclei; or inclusion of non-identical nuclei in a single spore.

The potential of heterokaryosis as a mechanism of natural variation especially in fungi pathogenic to plants has been widely discussed (15,

17, 20, 70), and divergent opinions exist concerning what constitutes evidence for the occurrence and role of heterokaryosis in natural populations of fungi. The heterokaryotic mechanism has frequently been used to explain variability in plant pathogens, especially those fungi in which the sexual stage is unknown.

Most plant pathologists are of the opinion that heterokaryosis is a common phenomenon in natural populations of fungi pathogenic to plants. Little, however, is known of the extent heterokaryosis alone can affect the pathogenic capabilities of a parasite, and under what circumstances heterokaryosis operates in nature. Furthermore, much of the evidence presented for heterokaryosis in plant pathogens is derived from laboratory studies with auxotrophic mutants under circumstances unlikely to be found in nature. Any extrapolation made from such studies to explain heterokaryosis in wild populations of fungi should be taken with caution (17, 70). Heterokaryosis is extremely important for the initiation of the parasexual cycle in both perfect and imperfect fungi, and hence for the release of genetic variability through somatic recombination.

The importance of heterokaryosis is best demonstrated in the heterothallic basidiomycetes. In both saprophytic and pathogenic fungi, the dikaryon represents a special kind of heterokaryon which is very stable. In saprophytic fungi the heterokaryon as well as the homokaryon are capable of indefinite vegetative growth, while in some pathogenic basidiomycetes such as the smuts, the dikaryon is pathogenic while the homokaryon is saprophytic. In the rust fungi, the dikaryon as well as the homokaryon can be either pathogenic on the same host species as in the autoecious rusts, or the heterokaryon can be pathogenic on one group of hosts while the homokaryon infects the alternate hosts as in the

heteroecious rusts.

Exchange and regrouping of nuclei between dikaryons has been suggested in some rust fungi, possibly as a mechanism for the evolution of new races, (27, 65, 99). Nelson et al. (65) mixed urediospores of races 38 and 56 of P. graminis tritici and used the mixture to inoculate a large number of susceptible wheat seedlings. The first generation of urediospores harvested from those susceptible seedlings were used to inoculate the resistant emmer wheat Khapli, and they isolated a new form that was virulent on this cultivar. Many of the urediospores of this new rust isolate had three or four nuclei. This heterokaryon was unstable, and after 25 uredial generations could no longer be maintained on Khapli. Watson (99) found that new heterokaryons could be produced as a result of hyphal fusion and nuclear exchange between two or more races of P. graminis tritici. But Watson and Luig (101) stated that although nuclear exchange is relatively frequent in the rust fungi, such an event seldom contributes to the formation of new races. They considered other asexual systems of variation such as parasexuality to have more significance than nuclear exchange by itself.

Parasexual Recombination. - Genetic recombination in the absence of meiosis was described by Pontecorvo (75, 76) as the parasexual cycle. Parasexuality was first reported in Aspergillus and has since been demonstrated in many fungal species. It is thought to be of general occurrence, especially in fungi lacking a perfect stage. This or a similar process has been thought to operate in several plant pathogenic fungi (15, 70, 102). The parasexual cycle consists of: heterokaryosis; fusion of unlike nuclei in the hyphal cell; recombination of genetic factors at mitosis, and segregation without going through sexual reproduction. New patho-

genic forms assumed to be derived by parasexual recombination have been reported by Buxton (15), and Singh and Hoffmann (84) in Fusaria; by Tinline (93) in Helminthosporium sativum; and in Ustilago zeae by Rowell (79).

Variation in some rust fungi has been explained at least in part by somatic recombination. Watson (99, 101) and Watson and Luig (102) studied the occurrence of somatic recombinants in P. graminis from uredospore mixtures of different races after being passed through a susceptible host. From such mixtures they obtained a diversity of new forms that could not be accounted for by nuclear exchange alone and they suggested that somatic recombination occurred. Parasexual recombination for factors conditioning virulence was also proposed to occur in a mixture of two races of P. coronata avenae by Bartos et al. (7).

Sexual Recombination. - Pathogenic variability through sexual reproduction is a direct consequence of the reassortment of genes at loci controlling virulence.

Flor (27) has shown that in the autoecious fungus M. lini, recombination of genes controlling virulence takes place during sexual reproduction. He reported the isolation of 64 races from the F_2 progeny of a cross between race 22 from South America and race 24 from U.S.A. Of these, 62 were previously unidentified races and some possessed more virulence than either parent. Variations of P. graminis tritici due to reassortment of genes for virulence during sexual reproduction have been reported by Craigie (19), Newton et al. (69), Roane et al. (78), Stakman and Harrar (88), and many others. Of special interest is the report of Roane et al. (78) who studied the appearance of new races arising from barberry as a result of sexual reproduction under natural conditions.

In a four year period, they isolated 42 races and subraces from cereal fields in the area of barberry bushes. The virulence potential of these 42 races was so broad that no commercial wheat cultivar in the United States would have effective resistance if all races were to become prevalent.

Changes in Race Distribution Due to Differences in Virulence and Aggressiveness. - The race composition of many pathogenic fungi fluctuates (32, 43, 100), partly as a direct response to man-made alterations in the host population, brought about by the production of resistant crop cultivars (41). However, variation in the prevalence of races occurs even when the host population remains undisturbed (32, 43). Alterations in race prevalence not directly related to changes in host cultivars may be due to the interaction of environmental factors with genotypic characters of the pathogen other than virulence, mainly aggressiveness. Virulence and aggressiveness are unrelated pathogen characters, but both are under genotypic control (100). Virulence relates the ability of a pathogenic race to overcome specific host resistance genes, while aggressiveness describes the aptitude of a race to become predominant when in competition with other equally virulent races of the pathogen (43, 100).

Changes in the race composition of P. graminis tritici have been observed which cannot be explained by variation in the virulent capabilities of the races involved. Luig and Watson (56) reported that the elimination of the Australian-New Zealand wild-type races of stem rust were not directly related to the growing of resistant wheat cultivars, but to their inadequate fitness to meet the competition from race 126-6,7 when it appeared in that area. This race, first isolated in 1925

(56), proved to be highly aggressive and soon overran the wild-type races. Selective pressure caused by the growing of resistant wheat cultivars produced changes to race 126-1,6,7, and later to race 126-2,6,7. These new races adquired by mutation the ability to attack cultivars which previously were resistant to strain 126-6,7. At this point, specific changes for virulence related to the corresponding genes for resistance in the cultivars, became reassociated with characters for survival and aggressiveness (56). Strains of race 126 and of a closely related race 222 became predominant from 1938 to 1954. According to Luig and Watson (56) the race 126-222 complex would have continued to predominate had it not been for the rise to prominence of more aggressive and previously unrecorded variants of races 21 and 34. The prevalence of races 21 and 34 has been explained on the basis of characters associated with aggressiveness (56, 100).

In the spring wheat belt of the U.S.A. and Western Canada, races 56 and 15B have predominated for the last 35 years although a great many other races have been identified in trace amounts during rust surveys (32). Race 56, first identified in Canada in 1931 (68), became predominant and caused the epiphytotic of 1935 which nearly destroyed the variety Ceres (88). This race did not become predominant on account of its virulence on Ceres, since this cultivar was also known to be susceptible to other contemporary races such as 11 and 34 (100), but because of its greater aggressiveness. This race continued to prevail despite the fact that cultivars resistant to it were planted after 1935 (43). In 1950, race 56 was displaced by the less aggressive but more virulent race 15B, mainly due to the fact that 15B possessed virulence to cultivars to which race 56 was avirulent. Race 15B caused the epidemics of 1950, 1953, and 1954 which prompted the release of Selkirk which is re-

sistant to both races 15B and 56. When host varieties with resistance to both races were grown, race 15B soon declined and race 56 once more rose to predominance. Race 56 again was later displaced by the more virulent and probably aggressive race 15B-1L CAN. This in turn was superseded by the still more virulent strain 15B-1LX CAN which still predominates (32).

The prevalence of these races in Western Canada when all cultivars presently grown are resistant, has been in part explained by taking into consideration the prevailing environmental conditions and host populations under which these rust races overwinter. It is known that the primary environmental conditions and host populations that influence the predominance of stem rust races in northern U.S.A. and Western Canada, are not found in those areas, but in Mexico and in the south of the United States where the rust population overwinters. Stem rust of wheat does not survive the winter in Western Canada and rust development is initiated every spring from inoculum blown in from the south. It appears clear that races found in Canada, are those that predominated and proved more aggressive in the south, and were able to overcome the obstacles of unfavorable climate and varieties in their annual migration to the north (32). It has been suggested that pathogens possessing unnecessary genes for virulence are less fit to survive and would be displaced by natural selection in favor of forms carrying the minimum number of genes for virulence (74). According to Green (32), this hypothesis does not apply to the present pathogenic rust population prevailing in Western Canada, where rust races with a large number of unused genes for virulence predominate. Green (32) and Luig and Watson (56) have presented ample evidence which shows that the prevalence of rust races depends not only on the number of

genes for virulence, but in addition, on other characters directly related to aggressiveness.

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SECTION TWO - TRITICALE, A MAN-MADE NEW BOTANICAL GENUS.

INTRODUCTION AND LITERATURE REVIEW

Since the prime topic of this thesis is concerned with the mode of inheritance of genes conditioning resistance to P. recondita tritici in hexaploid Triticale, the following section is devoted to a review of the literature related to this amphiploid.

Triticale is a new plant genus produced artificially by man. It results from crossing either hexaploid or tetraploid wheat (Triticum sp.) with diploid rye species (Secale sp.) followed by the doubling of the chromosome complement of the sterile F_1 hybrid. The name Triticale has been coined from the prefix of Triticum and the suffix of Secale, the parental genera (17). According to O'Mara (13), Wilson in 1876 was the first to obtain and describe a sterile F_1 triticale hybrid, although the first fertile triticale was not reported until 1888 by a German worker, Rimpau. Presumably, the fully fertile amphiploid was derived via spontaneous chromosome doubling within a natural population. Since then, many new triticales have been produced artificially.

In general, most of the early triticales were the subject of study by botanists and cytogeneticists who were mainly concerned with the taxonomic and evolutionary aspect of intergeneric hybrids (4, 6, 13). It was not until the early 1930's that triticale began to be evaluated as a potentially new field crop (10, 11).

Ploidy Level of Triticale

Two polyploid forms can commonly be produced in triticale, namely, hexaploid (2N = 6x = 42) and octoploid (2N = 8x = 56). Hexaploid triticales result from hybridizing any of the wheat cultivars belonging to the tetraploid species T. turgidum L.[1/] with any of the diploid species

[1/]The wheat nomenclature system as proposed by Morris and Sears (9) is used throughout.

of Secale sp. followed by doubling of the chromosome number of the resulting sterile hybrid. Likewise, an octoploid triticale is derived by doubling the hybrid produced from crosses between any of the hexaploid cultivars of T. aestivum L. em Thell. and diploid rye.

Cytogenetics and Fertility of Primary Triticales

Triticale combines the full chromosome complement of both wheat and rye. Wheat-rye crosses are relatively easy to perform, although the percentage seed-set obtained is normally not high. However, by excising the embryo from the seed and culturing it in synthetic media (2, 16) relatively good success in rearing the hybrid seedlings can be achieved. These F_1 hybrids are sterile due to chromosomal imbalance of the gametes, however, with the use of certain drugs, e.g. colchicine, their complete chromosome complement can be doubled (2). This results in the production of balanced gametes and consequently a fertile amphiploid. Triticales produced by this means are usually referred to as raw or primary triticales in contrast to the so-called "secondary" triticales which are obtained by recombination due to crossing and selection between two or more primary triticales.

Primary triticales are characterized by their partial fertility which has been observed in both the hexaploid and octoploid types (6, 10, 13). The reason for such infertility is yet not fully understood although Müntzing and his coworkers (10, 12) observed the meiotic chromosome behavior of octoploid triticales to be highly irregular and suggested that this in part was the cause of infertility. He proposed that this anomalous meiotic behavior resulted from inbreeding depression of the rye component when incorporated into an otherwise inbreeding species. O'Mara (13), however, disagreed with this interpretation on the basis of

his findings that hybrids between octoploid triticales - in which there would not be inbreeding depression - were sometimes more meiotically irregular and less fertile than the parental lines. Riley and Chapman (17) proposed that cytological instability and low fertility of triticale were two unrelated phenomena and that selection for improved fertility would not automatically result in increased meiotic regularity. They contended that infertility was the result of a general incompatibility between the genotypes of inbreeders such as wheat and the genotype of rye which is an outbreeder. Riley and Bell (16) showed that there was little correlation between the frequency of univalents and fertility in several artificially produced amphiploids. Müntzing (11), and Müntzing et al. (12) noted that most chromosomes observed as univalents in metaphase I of triticale were rye chromosomes, a condition particularly true in octoploid triticales in which reversion to pure wheat due to loss of the rye chromosomes is sometimes observed to occur.

Cytological instability and partial fertility is also known to exist in primary hexaploid triticale (6, 18). Sanchez-Monge (18) reported that the frequency of univalents observed in metaphase I is of the same magnitude as that found in the octoploid forms. Moreover, he expressed the opinion that such univalents were rye chromosomes. More recently, however, Shigenaga and Larter (19, 20) reported that in the hexaploid cultivar Rosner, both wheat and rye chromosomes contribute to the aneuploid condition.

It remains, however, that the fertility of both octoploid and hexaploid forms of primary triticales, has been improved over the years as a result of selection, although none has been found with fertility equal to that of cultivated wheat. Further progress in the improvement of

fertility has been made through the utilization of primary strains in a hybridization and selection program.

Improvement of Triticale Through Breeding

Low seed yield of triticale in general has been correlated with one or more of the following characters:

1) Reduced fertility,
2) Shrivelling of seed resulting from incomplete endosperm development and consequently, low test weight,
3) Weak straw, which results in early lodging,
4) Lateness, which becomes a limiting factor under conditions of soil moisture stress or risk of frost.

In addition, Zillinsky and Borlaug (24) mentioned the following factors that affect yield stability of triticale:

5) Genetic sensitivity to day-length; some triticales require long days in order to flower, therefore, they behave poorly under short-day conditions,
6) Restricted adaptation, and
7) Disease problems.

a) Octoploid Triticales - Müntzing (10) in 1934 was first to engage in a program to improve triticale through hybridization and selection. The first strains evaluated by him, yielded only 50 percent of the yield of a wheat cultivar included as a standard. By 1963, however, the yield had been raised to 97 percent of the wheat standard. Müntzing (11) attributed this success to the improvement of seed type and increased test weight. He noted, that although fertility of these triticales was improved relative to the strains initially tested, it never approached that of the wheat cultivar.

b) Hexaploid Triticales - Recently attention has been shifted towards the hexaploid forms of triticale as a crop species. Sanchez-Monge (18) postulated that the optimum chromosome number of triticale was at the hexaploid level and that emphasis should be placed on the investigation of strains of this particular type. He noted that primary hexaploid (6x) triticales had similar incomplete fertility to that found in the octoploid (8x) forms. However, phenotypically the 6x triticale was far more vigorous and tillered more than the 8x types. Pissarev (14) reported that almost complete fertility could be achieved by crossing octoploid with hexaploid triticales and then selecting for highly fertile recombinants. Kiss (4) has also followed a similar approach with some success.

The most intensive breeding programs geared to develop hexaploid triticale as a new field crop are those being conducted by the University of Manitoba in Canada and by the Centro de Mejoramiento de Maiz y Trigo (CIMMYT) in Mexico (6, 24).

The Canadian program was initiated in 1954 when the University of Manitoba obtained a large number of primary triticales from co-operators throughout the world (6). These triticales were initially evaluated in the field and the most promising strains saved for further improvement through hybridization. Simultaneously, the production of new primary triticales was undertaken. According to Larter et al. (6), the first lines derived from intercrossing primary triticales were excessively tall and not suited for immediate use as a cereal crop. They observed, however, that hexaploid triticale was higher in fertility and developed better seed than the octoploid forms.

In 1963, the University of Manitoba initiated a winter triticale

nursery in Northwest Mexico, with the aim of obtaining two plant generations each year, one in Canada in the summer months, and one in Mexico during the winter. This move proved highly rewarding to the breeding program in that it permitted breeding materials to be evaluated in a new environment where strains with broader adaptation, earliness and insensitive to day-length could be selected (6).

Yield tests from 26 localities across western Canada showed that some of the most recent triticale selections compared favorably with wheat cultivars in regard to plant height, maturity, straw strength, and resistance to stem rust (6). It was observed, however, that triticale was narrowly adapted particularly under conditions of high moisture stress where considerable sterility and thus low yields were recorded (6). However, the overall yield of the best triticale strain equalled that of the standard wheat cultivar.

The Mexican triticale program carried out by CIMMYT is a direct outgrowth of a cooperative breeding program established in 1964 by this institution and the University of Manitoba (24). These two centers freely exchange breeding materials and information which has led to a rapid improvement of hexaploid triticale. The following are some of the most outstanding improvements of triticale achieved by this combined breeding effort (6, 24):

1) The development of lines having complete fertility thereby removing the most important limiting factor in raising yields.
2) The selection of fertile lines in which seed shrivelling has been eliminated.
3) Selection of lines carrying genes for light insensitivity, which permit the evaluation of breeding material under either

short or long day conditions.

4) Improvement of yield - a direct response to the overall upgrading of plant type, seed weight, and fertility.

The solution to these problems has greatly enhanced the possibilities of triticale becoming a new cereal crop. However, many other problems still remain to be solved among which the following are given priority by CIMMYT'S breeders:

a) The introduction of genes for dwarfism to reduce crop losses due to lodging.

b) The fixation of genotypes that condition broader adaptation and stability under many different environment and soil conditions.

c) An understanding of the mechanisms of disease resistance.

d) The search for triticale lines possessing high levels of grain protein and lysine, and thus, the production of triticale cultivars with high nutritive value.

Potential Utilization of Triticale

Pissarev (14) reported that octoploid triticales could produce a higher protein content and still retain the bread-making quality of hexaploid wheats. Müntzing (11) has also found that octoploid triticales have in general a higher protein content than wheat, together with superior gluten and bread-making properties.

Hexaploid triticales do not possess the same bread-making characteristics present in the octoploid forms (21). However, Larter et al. (6) do not consider this to be a serious drawback since hexaploid triticale could likely find its place in those countries where unleavened bread is commonly used. They also reported that some triticale lines

have been experimentally processed as breakfast foods with excellent results. Other possible uses of triticale are in the distilling and brewing industry and as a feed for livestock consumption (6).

Protein content in the grain of hexaploid triticales is reported to be of the same level as that found in bread wheats with the amino-acid balance comparing favorably with that of either wheat or barley (6, 22), particularly the essential amino acids threonine and lysine.

Studies made at CIMMYT (22, 24) have shown that both protein and lysine content of the grain are greatly affected by the environment and that there is an inverse relationship between protein content and percentage of lysine.

Some recent studies on the nutritive value of triticale proteins in feeding trials using the meadow vole Microtus pennsylvanicus have shown that considerable diversity exists in the efficiency with which such proteins increase body weight (24). Protein efficiency ratings as reported by Elliott (see 24) ranged from values approaching zero, to values equal to egg protein. These findings have further enhanced the possibility of hexaploid triticale becoming an established crop species of importance, especially if plant breeders could produce cultivars with high nutritional value.

Disease Problems in Triticale

Octoploid as well as hexaploid forms of triticale are reported to be susceptible to many of the same microbial pathogens that attack both wheat and rye (24). Susceptibility to ergot (Claviceps purpurea) is a serious problem in most rye cultivars and it is also found in some triticales (6, 24). This disease is endemic in temperate zones where rye is commonly cultivated and could seriously restrict triticale production

in these areas unless resistance is found. Bacterial blight presumably caused by Xanthomonas translucens (24) also is found in many experimental lines of triticale.

The cereal rusts (Puccinia sp.) are considered to be the most serious threat to triticale production. It is generally believed that wheat is resistant to the rust forms which attack rye, and that rye is not attacked by rusts specialized to wheat (8, 23). Consequently, triticale which combines the full chromosome complement of both wheat and rye could be expected to be resistant to both rusts forms. In practice this expectation has not been realized and several rust species particularly those having wheat as a host infect triticale (3, 7, 15). Nevertheless, variation in infection types similar to that found in wheat also occurs in triticale.

Chester (3) reported that leaf rust reactions of several triticales tested by him were the same as those of the wheat parent, i.e. susceptibility to wheat leaf rust (P. recondita tritici) and resistance to rye leaf rust P. dispersa (= P. recondita secalis). Larter et al. (6) recognized that resistance to P. recondita tritici was lacking in Canadian triticales. They also suggested that resistance to the existing races of P. graminis tritici in western Canada, was conditioned by genes for resistance contributed by the rye parent.

Lopez (7) screened many lines of octoploid and hexaploid triticales in the seedling stage with several stem rust isolates collected from triticale, wheat and rye. He reported that it was mainly the wheat stem rust pathogen that was virulent on triticales, although a few rye stem rust isolates were also found to attack the same triticales. Rajaram et al. (15) recorded the reactions of several lines of hexaploid and

octoploid triticales to leaf rust collected from hexaploid wheats. They reported that some lines had seedling resistance. In addition, some of the seedling susceptible triticales, when tested in the adult-plant stage, showed some resistance.

Taxonomic Problems in Triticale

According to Baum (1), Wittmack in 1899 first applied the name Triticosecale to intergeneric fertile hybrids resulting from the cross Triticum aestivum x Secale cereale. This same class of hybrids were later referred to as Triticale (10) and O'Mara (13) suggested that this name be conserved to describe all allopolyploids resulting from crossing wheat with rye. The name has been widely accepted and used in connection with both octoploid and hexaploid forms. Baum (1) proposed that Triticale should be reserved as the valid generic name in view of its extensive use in the literature, notwithstanding the earlier use of the term Triticosecale.

In regard to the species name, some disagreement exists. Larter et al. (5), proposed the scientific name Triticale hexaploide Lar. for the hexaploid forms. However, their proposal has not been accepted by Baum (1) on the grounds that it does not conform to regulations of the International Code of Botanical Nomenclature. Baum (1) in turn proposed a species name in accordance with the parental wheat and rye species involved in the synthesis of the amphiploid. Thus, Triticale turgidocereale would describe the hybrid from the cross T. turgidum x Secale cereale, Triticale dicoccocereale describes the amphiploid T. dicoccum x S. cereale and so on. This system of naming would have some merit only if we were to restrict the naming of triticales to the primary forms. In addition, all of these "species" are fully interfertile and extensively used in

hybridization programs. Some system of naming hybrids such as Baum's Triticale turgidocereale x Triticale duro-montanum or Triticale dicocco-cereale x Triticale persicocereale or any other hybrid combination for that matter would have to be developed. Clearly, Baum's proposal lacks the flexibility needed in such a system.

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SECTION THREE - THE INHERITANCE OF RESISTANCE TO PUCCINIA RECONDITA ROB. EX. DESM. IN HEXAPLOID TRITICALE.

THE INHERITANCE OF RESISTANCE TO PUCCINIA RECONDITA ROB. EX. DESM. IN HEXAPLOID TRITICALE

M. A. Quinones

ABSTRACT

The inheritance of resistance to wheat leaf rust caused by P. recondita Rob. ex. Desm. f.sp. tritici was studied in the hexaploid triticales 6A-190, Rosner, Armadillo, Bronco, and Toluca 160.

Resistance in the triticales studied was monogenically inherited and each line carried a single dominant gene. The genes conditioned resistance to races 15 and 30, and were given the following temporary designations: Gene A (6A-190), B (Rosner), C (Armadillo), D (Bronco), and E (Toluca 160). Gene A was linked in repulsion with gene C with a cross over value of 44.75 ± 1.44 percent and assorted independently from the other genes. Genes B and C were also independently inherited. No recombinants were recovered from the cross between Bronco x Toluca 160 suggesting that genes D and E were identical or closely linked. The parental triticale lines Bronco and Toluca 160 (genes D and E, respectively) appeared to carry in addition to the resistance gene, modifying genes which inhibited the expression of resistance in certain crosses.

The results obtained indicated that the genes governing resistance were derived from the wheat parental species and that resistance to wheat leaf rust carried by the rye parent was not expressed in the triticale amphiploid. It was also found that genes conditioning resistance to wheat leaf rust were equally effective in conditioning resistance to rye leaf rust.

INTRODUCTION

During the last two decades, considerable breeding efforts have been channeled into the development of hexaploid Triticale as a commercial crop species (11, 12, 24). During this short period of time tremendous progress has been made in the improvement of agronomic characteristics such as fertility, seed type, earliness, plant type, and yield. However, other problems still remain to be solved, among which, the clarification of the mechanisms of disease resistance is of considerable importance.

The cereal rusts (Puccinia sp.) are considered to be the most serious threat to triticale production, especially leaf rust caused by P. recondita Rob. ex Desm. f.sp. tritici which has been found to be particularly virulent on many of the experimental triticale lines (12, 24). To date there have been no studies on the inheritance of resistance in triticales to cereal rusts. The present study was undertaken with the purpose of gaining some insight into the genetic mechanisms that contribute to leaf rust resistance in hexaploid triticale.

MATERIALS AND METHODS

The hexaploid triticale lines used as progenitors in this study, their parentage, origin, and reaction to several races of wheat leaf rust and to one isolate of rye leaf rust are given in Table I.

Diallel crosses were made between the 6 lines 6A-190, Rosner, Armadillo, Bronco, Toluca 160 and Accession No. 6239. All of these lines were backcrossed to Acc. 6239 which was used as a common susceptible parent. In addition, other susceptible lines were used in crosses and backcrosses in the following manner: Rosner with Rosner "S" and Acc. 6507; Armadillo with line 6A 276; Bronco with Bronco "S" and Acc. 6507; and Toluca 160 with Toluca 160 "S". Rosner "S", Bronco "S", and Toluca

TABLE I. Progenitors, their parentage, origin and seedling reaction to the indicated races of *Puccinia recondita tritici* and *P. recondita secalis*.

Parentage	Name or * Accession No.	Origin	Wheat leaf rust Race 9	Race 15	Race 30	Race 161	Rye leaf rust isolate RLR-1/71
(*T. turgidum* var. *durum* cv. Stewart x *S. cereale*)	6A-190	U. of M.	;1^+	;1^+	;	;1	;1^-
[(*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)]	Rosner	U. of M.	;1^-	;1	;1^-	;1^-	;1^-
Rosner "S"	Rosner "S"	U. of M.	4	4	4	4	3
Rosner x [(*T. turgidum* var. dicoccoides x *S. cereale*) x *T. turgidum* var. *persicum* x *S. cereale*)] X-308-14Y-1M-0Y-1W-0W	Armadillo	CIMMYT	;1^-	;1^-	;1^-	;1^-	;1^-
[(*T. turgidum* var. *durum* x *S. cereale* cv. Petkus) x (*T. turgidum* var. *persicum* x *S. cereale*)] x [(*T. turgidum* var. *durum* cv. Ghiza x *S. cereale*) x (*T. turgidum* var. *durum* x *S. cereale*)] X-224-15Y-2M-1Y-2M-0W	Bronco	CIMMYT	1^+ 2	1^+ 2^+	;1^+ 2	1^+ 2	;1^+ 2^+
Bronco "S" X-224-40Y-1M-2Y-1M-0W	Bronco "S"	CIMMYT	3^+	4	4	3	3^+ 4

TABLE I. continued

Parentage	Name or * Accession No.	Origin	Wheat leaf rust Race 9	Race 15	Race 30	Race 161	Rye leaf rust isolate RLR-1/71
[(T. turgidum var. durum x S. cereale) x (T. turgidum var. persicum x S. cereale)] x [(T. turgidum var. persicum x S. cereale) x (T. turgidum var. durum cv. Stewart x S. cereale)] X-298-21Y-2M-1Y-1M-OW	Toluca 160	CIMMYT	;1^+	;1^+ 2^+	;1^+ 2^+	1^+ 2	1^+ 2
Toluca 160 "S" X-298-14Y-1M-1Y-2M-OW	Toluca 160 "S"	CIMMYT	4	4	4	4	4
(T. turgidum var. persicum x S. cereale)	6A-276	U. of M.	4	4	4	4	3 4
(T. turgidum var. durum cv. Ghiza x S. cereale) x (T. turgidum var. dicoccoides x S. cereale)	Acc. 6239	U. of M.	4	4	4	4	3 4
[(T. turgidum var. durum cv. Ghiza x S. cereale) x (T. turgidum var. durum cv. Carleton x S. cereale)] x [(T. turgidum var. durum cv. Stewart x S. cereale cv. Prolific) x (T. turgidum var. dicoccum x S. cereale)]	Acc. 6507	U. of M.	4	4	4	4	3 4

*Accession number given by the Department of Plant Science, University of Manitoba.

160 "S", are susceptible isogenic lines of Rosner, Bronco, and Toluca 160, respectively.

To minimize outcrossing, two heads per plant were bagged at flowering, which provided enough seed for the rust tests. Greenhouse rust reactions were scored on seedlings of F_2 backcross lines and on F_3 families. Homozygous seedling susceptible F_2 backcross lines or F_3 families, were planted in the field for an evaluation of their adult plant leaf rust reaction as scored at flowering time.

The F_2 and F_3 lines to be tested for seedling reaction were grown in pots in the greenhouse or growth chamber under conditions normally used for such studies. Enough seed of each F_2 backcross line or F_3 family were sown to obtain 15 - 20 seedlings in the susceptible x resistant crosses, and from 30 - 60 seedlings in the resistant x resistant F_3 families. Parental material was included in all tests. Race 15, isolate 20/68 of P. recondita tritici, was used (hereafter referred as race 15) to test the rust reactions in all crosses. In addition, some of the lines, whose reactions to race 15 were known, were inoculated with race 30 of wheat leaf rust and with isolate RLR-1/71 of rye leaf rust (P. recondita secalis).

For each inoculation, potted seedlings at the 1 to 1½ leaf stage were placed in an incubation chamber and dusted with a mixture of talc and rust spores of one of the races used. After 18 to 24 hours incubation the pots were removed to a bench in the greenhouse. Rust readings were taken 10 - 12 days after inoculation according to the system described by Stakman, et al. (22).

Linkage intensities were calculated on the basis of the following formula of Mode and Schaller (16):

$$P = \frac{N - \sqrt{N^2 - 2nN}}{N} \qquad (1)$$

where n is the number of segregating families and N is the total number of families observed.

In the absence of segregation in the cross Bronco x Toluca 160, it was possible that two linked loci were involved. Therefore, an estimate was made of the maximum amount of recombination p, which might exist between the two loci each controlling resistance to the same race without observing a single recombinant type in the population tested. The formula used was outlined by Hanson (7) as follows:

$$P_{RC} = 1 - \sqrt[n]{P} \qquad (2)$$

where P_{RC} is the probability of obtaining the recombinant types, n is the number of families tested, and P is the probability of being wrong.

RESULTS

Susceptible x Resistant Crosses

A complete summary of F_2 backcross data and F_3 data is given in Tables II and III, respectively. For the sake of clarity, each cross is presented separately under the heading of the resistant parent used.

6A-190

This strain is a primary triticale that has been subjected to selection for a period of more than 20 plant generations. It was included in this study to determine if genetic studies could be carried out using primary triticales in which fertility is incomplete.

6A-190 was crossed and backcrossed to the susceptible triticale Acc. 6239. A total of 101 F_2 backcross lines and 71 F_3 families were analyzed for their reaction to race 15 of wheat leaf rust. The F_2 back-

cross lines gave a satisfactory fit to a ratio of 1 segregating:1 susceptible (Table II). Within each of the segregating lines a 3:1 ratio was obtained, indicating that a single dominant gene for resistance is present in 6A-190. Data from F_3 families of this same cross segregated in the expected 1:2:1 ratio and substantiated the above hypothesis (Table III). These results indicate that genetic studies can be carried out using primary triticale strains. The gene in 6A-190 has temporarily been designated as gene A and controls seedling resistance to leaf rust races 15, 30, and possibly 9, and 161 (Table I). In addition, this same gene appears to confer resistance to the rye leaf rust isolate RLR-1/71, a conclusion based upon the following observations: 1) The cultivar Prolific which contributed the rye genome (R) to the 6A-190 triticale is susceptible to this particular isolate, thus, resistance to rye leaf rust in 6A-190 is not conditioned by the rye parent; and 2) F_3 families with known reactions to race 15 were inoculated twice on the same plants, first at the 1-leaf stage with rye leaf rust (RLR-1/71) and 6 days later at the two-leaf stage with wheat leaf rust race 30. Families resistant to race 15 were also found resistant to rye leaf rust and to race 30. Similarly families either susceptible or segregating to race 15 were also susceptible or segregating respectively to the other two races. Most important, within a segregating family, seedlings resistant to RLR-1/71 were also resistant to race 30, and seedlings susceptible to one were also susceptible to the other race. This clearly showed that the same gene conditioned resistance to all races tested. Families scored as homozygous resistant exhibited a ;1 to 1^+ type of reaction, a level of resistance equal to that of 6A-190. In the segregating families, however, the resistant seedlings ranged from ;1 to 2^+ in reaction type indicating that

TABLE II. Segregation of reaction to race 15 of leaf rust in F_2 lines from backcrosses to the indicated susceptible parent.

Backcross combination		Number of lines		Expected ratio	P.
		Segregating	Susceptible		
Acc. 6239[2] x 6A-190	F_2	47	54	1:1	.50-.30
Rosner "S"[2] x Rosner	F_2	70	62	1:1	.50-.30
Acc. 6239[2] x Rosner	F_2	33	39	1:1	.50-.30
Acc. 6507[2] x Rosner	F_2	12	9	1:1	.50-.30
6A 276[2] x Armadillo	F_2	24	33	1:1	.30-.20
Acc. 6239[2] x Armadillo	F_2	12	9	1:1	.50-.30
Bronco "S"[2] x Bronco	F_2	18	23	1:1	.50-.30
Acc. 6239[2] x Bronco	F_2	3	16	1:1	$<$.01
Acc. 6507[2] x Bronco	F_2	8	30	1:1	$<$.01
Toluca 160 "S"[2] x Toluca 160	F_2	39	42	1:1	.70-.50
Acc. 6239[2] x Toluca 160[2]	F_2	12	43	1:1	$<$.01

the resistance gene when in the heterozygous condition was reduced in its overall effectiveness as influenced by the genetic background of the susceptible variety.

Rosner

Rosner is the first Canadian hexaploid triticale cultivar to be licensed (11).

Rosner was crossed and backcrossed to three different susceptible triticales, namely, Rosner "S", Acc. 6239, and Acc. 6507, and a total of 225 F_2 backcross lines and 144 F_3 families were tested with race 15. Disease reactions and segregation among F_2 and F_3 lines were the same in crosses involving the 3 susceptible parents (Tables II and III). The resulting segregation indicated that Rosner carries a single dominant gene, tentatively designated as gene B, which confers resistance to wheat leaf rust races 15, 30, and possibly 9, and 161 (Table I). In addition, some families with known reactions to race 15 were also inoculated with rye leaf rust (RLR-1/71) and wheat leaf rust race 30 in the same manner as described above in the discussion of 6A-190 triticale. The results indicated that gene B also conditioned resistance to race 30 and to the rye leaf rust isolate. All seedlings scored as resistant exhibited the same level of resistance as that of Rosner, indicating that gene B conditioned complete dominance.

Armadillo

Armadillo is an advanced line produced by the CIMMYT Mexican triticale program. It is one of the first light-insensitive, fully fertile lines to be developed and has been used extensively in hybridization programs to introduce the two above mentioned characters into other triticales (24).

TABLE III. Segregation of reaction to race 15 of leaf rust in F_3 families derived from resistant x susceptible crosses.

Cross combination	Number of families			Expected ratio	P.
	Resistant	Segregating	Susceptible		
Acc. 6239 x 6A-190	12	39	20	1:2:1	.50-.25
Rosner "S" x Rosner	20	45	27	1:2:1	.75-.50
Acc. 6239 x Rosner	15	27	10	1:2:1	.20-.10
6A 276 x Armadillo	24	55	38	1:2:1	.20-.10
Acc. 6239 x Armadillo	27	54	21	1:2:1	.75-.50
Bronco "S" x Bronco	33	54	37	1:2:1	.50-.25
Acc. 6239 x Bronco	8	22	34	1:2:1	$<$.01
Toluca 160 "S" x Toluca 160	37	92	30	1:2:1	.25-.10
Acc. 6239 x Toluca 160	6	45	35	1:2:1	$<$.01

This strain was crossed with two different susceptible parents, 6A-276 and Acc. 6239. A total of 78 F_2 backcross lines and 219 F_3 families were scored for leaf rust reaction to race 15. The segregation among the F_2 backcross lines and F_3 families was the same in crosses involving both susceptible parents (Tables II and III). A ratio of 1 segregating:1 susceptible was obtained with the F_2 backcross lines, indicating that a single dominant gene was segregating in this cross. F_3 data supported this assumption. This gene was provisionally designated as gene C and confers seedling resistance to races 15, 30, and possibly 9, and 161 (Table I). F_3 families inoculated with isolate RLR-1/71 of rye leaf rust and race 30 of wheat leaf rust, as previously described, segregated only for gene C which suggested that this gene also conditions resistance to rye leaf rust. This gene exhibited complete dominance as observed by the ;1 to 1^+ type of reaction scored in all resistant seedlings.

Bronco

Bronco is another advanced line obtained from the CIMMYT triticale program. Its fertility is acceptable although lower than that of Rosner or Armadillo. It was chosen in the present study because its type of reaction to leaf rust clearly differed from that of the other resistant lines indicating that a different gene may be involved.

The results obtained from studies with this line were difficult to interpret. The parental line proved highly heterogeneous in its rust reaction ranging from ;1^- to 2^+ and selection of the most resistant plants over two plant generations failed to yield a progeny with a stable type of reaction. Nevertheless, three susceptible lines were used in crosses with Bronco. The results are discussed separately in view of the anoma-

lous segregation obtained in the progeny from certain of these crosses (Tables II and III).

A total of 41 F_2 backcross lines and 124 F_3 families of the cross Bronco "S" x Bronco were tested with race 15. From the F_2 backcross lines, a 1 segregating:1 susceptible ratio was obtained indicating monogenic inheritance (Table II). Within segregating lines a 3:1 ratio was observed. The segregation among the F_3 families from this same cross fitted a 1:2:1 ratio supporting the assumption that only one gene was present (Table III). The resistant seedlings exhibited the same variation in type of reaction (;1^- to 2^+) as that of the resistant parent. This gene was temporarily designated as D and conditions the same type of seedling reaction to races 15, 30, and possibly 9, and 161 (Table I). This gene was also found to condition resistance to the RLR-1/71 isolate.

Backcross lines and F_3 families from crosses involving the other susceptible parents behaved differently. Of 19 F_2 backcross lines of the cross Acc. 6239^2 x Bronco, only 3 segregated for resistance and 16 were scored as susceptible. These results significantly deviate ($P < .01$) from the expected 1:1 ratio (Table II). Moreover, segregation within the 3 segregating lines did not give a satisfactory fit to a ratio of 3 resistant:1 susceptible as would normally be expected for a single dominant gene, but rather the anomalous ratio of 1/3 resistant:2/3 susceptible. Similarly, when 64 F_3 families of the same cross were scored for their reaction to race 15, a significant deviation from the expected 1:2:1 ratio was observed, the majority of families being susceptible (Table III). In addition, of the 22 segregating families, 15 gave a good fit to a ratio of 3 resistant:1 susceptible, but the remaining 7 families had a significantly higher number of susceptible seedlings. This segregation suggested

the presence of a second gene which modifies, in the direction of susceptibility, the expression of gene D. This modifying factor appears to be carried by the resistant parent as suggested by the fact that distorted ratios were observed in some other crosses involving this line.

In the combination Acc. 6507^2 x Bronco, 38 F_2 backcross lines were available for study. This cross also deviated significantly from the expected ratio of 1 segregating:1 susceptible (Table II). Only 8 F_2 backcross lines segregated, while 30 were scored as susceptible. Within each of the segregating lines 3 segregated in a ratio of 3 resistant:1 susceptible, but the remaining five had a significantly higher number of susceptible seedlings. This cross further indicated that a modifying gene inhibiting resistance was segregating.

Toluca 160

Some difficulty was encountered in the testing of this line. Its type of reaction to the races tested varied from ;1^+ to 2^+ and attempts to select a stable line were unsuccessful.

Toluca 160 was crossed with two different susceptible lines. Each cross is presented separately in view of the different results obtained.

A total of 81 F_2 backcross lines of the cross Toluca 160 "S" x Toluca 160 were tested with race 15. The segregation observed fitted the 1:1 ratio expected on the assumption that a single gene was operating (Table II). This was later confirmed by testing 159 F_3 families of the same cross (Table III). Within segregating F_2 backcross or F_3 lines, a good fit to a ratio of 3 resistant:1 susceptible was obtained. The type of reaction on the resistant seedlings varied from ;1^- to 2^{++} which closely approximated that of the resistant parent. This gene was provisionally designated as gene E and confers the same type of seedling reaction to

races 15, 30, and possibly 9, and 161 (Table I). In addition, a selected number of F_3 families were tested to rye leaf rust in the manner previously described, and the results indicated that this gene also conditioned resistance to rye leaf rust.

In the cross Acc. 6239^2 x Toluca 160, a total of 55 F_2 backcross lines were tested with race 15. The results obtained deviated significantly from the expected 1:1 ratio (Table II). Only 12 lines segregated whereas the remaining 43 were susceptible. In addition, within the segregating lines only five gave a normal ratio of 3 resistant:1 susceptible, while the remaining 7 lines had a significantly higher proportion of susceptible seedlings. Similarly, when 86 F_3 families of the same cross were tested with race 15, the segregation among the families deviated significantly from the 1:2:1 ratio normally expected for a single gene (Table III). This anomaly suggested that a modifier gene was involved, particularly in view of the fact that among the 45 segregating F_3 families, 30 segregated within each family in a ratio of 3 resistant:1 susceptible, while the remaining 15 families carried a significantly higher number of susceptible seedlings. This hypothesis, however, fails to explain the reduced number of resistant families that were observed.

Resistant x Resistant Crosses

The relationship among the genes present in the resistant parents used in this study was investigated by intercrossing them in diallel. F_3 families of each cross were tested with race 15 in the seedling stage.

From the results presented in Table IV it is observed that gene A present in 6A-190 was independently inherited from genes B (Rosner), D (Bronco), and E (Toluca 160), and was linked in repulsion with gene C (Armadillo) with a cross over value of 44.75 ± 1.4 percent.

TABLE IV. Segregation of reaction to race 15 of leaf rust in F_3 families derived from resistant x resistant crosses.

Cross	Genes involved	Number of families			P. for 15:1
		Resistant	Segregating	Susceptible	
6A-190 x Rosner	A x B	18	32	7	.10-.05
6A-190 x Armadillo	A x C	140	76	3	<.01
6A-190 x Bronco	A x D	60	10	2	.30-.20
6A-190 x Toluca 160	A x E	26	27	5	.75-.50
Rosner x Armadillo	B x C	61	7	1	.10
Rosner x Bronco	B x D	24	45	11	<.01
Rosner x Toluca 160	B x E	38	75	16	<.01
Armadillo x Bronco	C x D	35	84	20	<.01
Armadillo x Toluca 160	C x E	32	27	4	.99
Bronco x Toluca 160	D x E	94	0	0	-

Gene B carried in Rosner appears to assort independently from gene C (Armadillo). However, only a limited number of segregating families was observed. These results at the moment cannot be clearly explained unless the segregation of 61:7:1 is an extremely poor fit to the modified two factor ratio of 11:4:1. Another possible explanation is to assume that genes B and C belong to the same locus and the 7 segregating and 1 susceptible families resulted from outcrossing or seed mixtures. In the crosses involving Rosner x Bronco (B x D) or Rosner x Toluca 160 (B x E), the results obtained have not been satisfactorily explained (Table IV). It is clear that gene B of Rosner is different from either genes D or E. However, an excess of susceptible families were recovered in the two crosses. Moreover, 11 of the 45 segregating families of the cross Rosner x Bronco gave within each family an equal or higher number of susceptible seedlings than resistant ones. Likewise, in the Rosner x Toluca 160 cross 14 out of 75 segregating families presented a similar anomalous ratio. These results suggested that in addition to the resistancegenes, an inhibitor factor was also segregating.

In the cross Armadillo x Bronco (C x D) a similar distorted ratio was observed (Table IV). An excessive number of susceptible families were recovered, and of the 84 segregating families 20 showed within a family a significantly higher number of susceptible seedlings, indicating that a modifying gene was also segregating. In the cross of Armadillo x Toluca 160 (C x E), the data obtained indicated that these two genes assorted independently (Table IV).

When the cross Bronco x Toluca 160 (D x E) was studied, no recombinants were identified (Table IV). This indicates that either only one locus is involved and genes D and E are the same or alleles, or the two

genes if different are closely linked. If two linked genes are involved, they would have to be separated by less than 3.14 cross-over units (see formula 2, Materials and Methods).

Evidence gathered during the course of this study indicated that all the resistance genes that were identified were contributed by the wheat parental species and none by the rye parent. To test this hypothesis, wheat-rye crosses were made with three tetraploid wheat cultivars and three rye cultivars and the resulting polyhaploid F_1 hybrids were inoculated with race 15 of wheat leaf rust and with the rye leaf rust isolate RLR-1/71.

From the results (Table V) it can be observed that resistant reactions are obtained only when the wheat parent carries some genes for resistance, otherwise the hybrid is susceptible. In other words, the rye parent did not contribute to resistance of the F_1 hybrid.

An event that could have extraordinary significance in the mechanics of the synthesis of triticale in the future was noted during the rust studies carried out with the polyhaploid F_1 sterile hybrids. It was observed that approximately 35 percent of the hybrid plants (normally completely sterile) exhibited some degree of fertility indicating that chromosome doubling had occurred. The percentage of doubling was too high to be explained on the basis of spontaneous doubling. The F_1 hybrid seedlings were grown during the rust test in a growth chamber operating at $20^{o}C \pm 1$ and 16 hours light. Inadvertently these hybrids when in the 1 to $2\frac{1}{2}$ leaf stage, were subjected to a high concentration of ammonia gas in the chamber's atmosphere following a heavy application of commercial fertilizer and it is suspected that this gas may in part be responsible for the occurrence of partially fertile hybrids. The validity of this

TABLE V. Seedling reaction of wheat-rye F_1 hybrids and their parents to race 15 of wheat leaf rust and to the rye leaf rust isolate RLR-1/71.

Material tested	No. of plants tested	Leaf rust reactions Race 15	RLR-1/71
T. turgidum v. durum cv. Ghiza	20	2^{++} 3^{+}	0;
(T. turgidum v. durum cv. Ghiza x S. cereale cv. Argentina) F_1	8	2^{++} 4	;1^{+}
S. cereale cv. Argentina	20	0;	4
T. turgidum v. durum cv. Ghiza	20	2^{++} 3^{+}	0;
(T. turgidum v. durum cv. Ghiza x S. cereale Acc. 2D-285*) F_1	3	3^{+} 4	0 1^{+}
S. cereale Acc. 2D-285	20	0;	4
T. turgidum v. durum cv. Ghiza	20	2^{++} 3^{+}	0;
(T. turgidum v. durum cv. Ghiza x S. cereale Acc. OD-289) F_1	2	3^{+} 4	;1^{++}
S. cereale Acc. OD-289	20	0;	4
T. turgidum v. durum cv. Stewart 63	20	;1^{+}	0;
(T. turgidum v. durum cv. Stewart 63 x S. cereale cv. Argentina) F_1	5	;1^{+}	;
S. cereale cv. Argentina	20	0;	4
T. turgidum v. durum cv. Stewart 63	20	;1^{+}	0;
(T. turgidum v. durum cv. Stewart 63 x S. cereale Acc. OD-289) F_1	3	;1^{+}	;
S. cereale Acc. OD-289	20	0;	4
T. turgidum v. durum cv. Carleton	20	;1^{-}	0
(T. turgidum v. durum cv. Carleton x S. cereale Acc. OD-289) F_1	2	;1	;
S. cereale Acc. OD-289	20	0;	4

*Accession numbers used by the Department of Plant Science, University of Manitoba.

assumption awaits cytological verification, meanwhile, the process is being tested further in view of its practical significance.

Field Studies

F_2 backcross lines and F_3 families that were homozygous susceptible in the seedling stage were planted in the field to determine whether additional factors for adult plant resistance were present. The nursery was inoculated about 4 weeks before heading with a mixture of the leaf rust races which are presently prevalent in Western Canada (20). Rust reactions were recorded after heading.

Some difficulties were encountered in classifying individual plants for rust reaction. Most of the material was susceptible in the field and among the few lines that were segregating a continuous variation from resistance to susceptibility was observed. Rosner appears to carry an incompletely dominant gene for adult plant leaf rust resistance, but no conclusions could be reached with regard to the other lines.

DISCUSSION

This is the first study specifically directed at an investigation of the genetic factors that contribute to leaf rust resistance in hexaploid triticale. The triticale material used in the present study was selected mainly on the basis of fertility, and attempts were made to include progenitors which were as unrelated as possible.

As a working hypothesis for this study, it was assumed that resistance of hexaploid triticale to wheat leaf rust was genetically complex. In other words, genes for leaf rust resistance from both the wheat and rye parents were expected to be expressed in the triticale amphiploid. This assumption was based on the observation that both wheat and rye, the parental genera, exhibit high levels of resistance to this rust

pathogen (15, 18). Genetic complexity in the amphiploid was further anticipated since the number of interacting genes for leaf rust resistance from both parents was also unknown.

The genetic studies were carried out with race 15 of wheat leaf rust in view of its widespread distribution in Western Canada (20). However, any other race would have been as appropriate because the resistant lines exhibited the same type of reaction to all races tested. Similarly the susceptible parents were susceptible to all of these races.

The results suggest that resistance in each triticale studied is conditioned by a single gene. This finding indicates the desirability of conducting genetic studies to aid breeding programs. Different sources of resistance could then be identified and combined in a single cultivar. Based on experience with other crop cultivars resistance is generally conditioned by a single gene and it is often ephemeral (2, 4, 9, 14, 17). This is because the pathogen requires but a single mutation from avirulence to virulence to overcome each specific host resistance gene.

The combination in a single triticale cultivar of genes A, B, C, D, and E identified in this study would be difficult to achieve at the present time, since no rust culture is available to differentiate these genes. However, triticale lines carrying two resistance genes at a time could be developed. These lines in turn could be employed in a double cross program to combine four different resistance genes. Large F_2 populations derived from such crosses could be grown and many resistant plants selected in order to obtain recombinants homozygous for the four resistance genes. Test crosses could be used to identify such plants. Unfortunately, this method is time consuming and impractical since breeders are concerned not only with disease resistance but must also select for

other agronomic traits. Thus, F_2 populations must be disproportionately large in order to obtain a single recombinant homozygous at all the required resistance loci. The scarcity of recombinants containing the desired genes for rust resistance would greatly reduce any opportunity for selections based on other agronomic characters.

The finding that in triticale the resistance genes were only contributed by the wheat parental species was unexpected. Rye is reported to be resistant to wheat leaf rust (15) and the consensus among most plant breeders is that this resistance also operates in the amphiploid. However, as it turned out, the rye resistance genes appeared to be hypostatic to the susceptibility carried into triticale by the wheat parent. If this is a prevalent phenomenon among all triticales, resistance to wheat leaf rust would depend solely on resistance genes from wheat and not from rye as was previously expected. In addition, the results obtained indicated that the wheat resistance genes were effective in providing protection against rye leaf rust.

The gene A ascribed to the 6A-190 triticale conditioned almost complete dominance. However, when in the heterozygous state, this gene appeared to be slightly affected by the total genetic background of the susceptible line used. In the present study this was noted by minor modifications on the type of reaction observed when this gene was segregating. Since in the triticales studied, resistance to wheat leaf rust was under the genetic control of the wheat parent exclusively, it can be inferred that the durum cultivar Stewart 63 - the parental wheat in the 6A-190 triticale - carries only one gene for seedling resistance to leaf rust.

The results obtained with the cultivar Rosner indicated that a com-

pletely dominant gene designated gene B was operative in the seedling stage. In addition, Rosner carries a gene for adult-plant resistance. This gene conditioned a lower level of resistance and was independently inherited from gene B.

Likewise, the Armadillo triticale line was found to possess a single dominant gene designated gene C.

In the study with the line Bronco, the results obtained cannot satisfactorily be explained. Bronco was crossed with three susceptible lines; Bronco "S", Acc. 6239, and Acc. 6507. In the cross Bronco "S" x Bronco, the data indicated monogenic inheritance and the resistance gene was designated gene D. However, when the other two susceptible lines were crossed with Bronco, the results obtained did not fit the expected ratio for a single gene. Most of the F_2 backcross lines or F_3 families from these two crosses were found to be susceptible. In addition, when segregating families were tested, a few gave a 3 resistant:1 susceptible ratio, but most had significantly higher numbers of susceptible seedlings. Distorted ratios were also observed in other crosses involving the line Bronco.

These anomalous results can in part be explained if it is assumed that modifier genes were also operating. The expression of the resistance gene in the heterozygous condition may be inhibited in the presence of such modifier genes. In this manner, a higher percentage of susceptible seedlings in the segregating lines can occur. However, this cannot explain the excessive number of susceptible lines observed unless the relatively low number of seedlings tested per line failed to reveal the distorted segregation. If this interpretation is correct, the normal segregation obtained in the cross Bronco "S" x Bronco can be explained

by assuming that the modifying genes do not operate in the Bronco genetic background. Similar modifying genes have also been reported in a number of studies of resistance to leaf rust in cultivars of common wheat (1, 3, 5, 6, 8).

Other alternative hypotheses which could also help explain the distorted ratios are: 1) aneuploidy, and 2) the presence of a pollen-killing gene linked with the resistance gene.

The meiotic behavior of hexaploid triticales is not completely stable (12, 21) and some aneuploids usually occur even in the progeny of highly fertile triticales such as Rosner (21). If the chromosome carrying the resistance gene is preferentially eliminated in some of the crosses, then distorted ratios and a high number of susceptible families would be obtained. Preliminary cytological observations indicated that the triticales included in this study exhibited a high degree of meiotic instability. Nevertheless, their fertility was acceptable. If aneuploidy was in fact responsible for the distorted ratios, then, this situation would be unique to certain crosses but would not occur in others.

Deviations from expected ratios have also been reported due to the presence of a pollen-killing gene in some studies of rust resistance (13). This possibility is worthy of further investigation since it may help explain part of the anomalous results found in the present study.

Similar results to those obtained with the Bronco crosses were found in the study of Toluca 160. This line gave a normal segregation for one resistance gene in the cross Toluca 160 "S" x Toluca 160, and the gene was designated gene E. However, when the cross Acc. 6239 x Toluca 160 was studied, a deviation from the expected monogenic segregation was observed. This result cannot be clearly explained and the possibility of

either modifier genes, aneuploidy, or the presence of a pollen-killing gene is again suggested.

In North America, evolution of virulence in the wheat leaf rust organism has taken place primarily on hexaploid wheat cultivars. As a result, all tetraploid durum cultivars presently grown in North America are reported to be resistant (18, 20, 23). Results obtained in this study and elsewhere (19), demonstrated that when a triticale was resistant to one leaf rust race, it was also resistant to all other races tested. Conversely, a susceptible triticale appeared susceptible to all races which suggested the total absence of wheat leaf rust resistance genes. No indication of a differential interaction among triticales to leaf rust races was found. This phenomenon can be explained by postulating that the leaf rust pathogen in North America has not yet evolved virulence towards any of the resistance hexaploid triticales whose resistance genes are derived from the tetraploid wheats.

Genetic studies of rust resistance using F_2 lines derived from backcrosses to a susceptible parent are often preferable to studies using F_3 families (10). Fewer lines are required for the backcross analysis, in addition if more than one gene is present in a cultivar, F_2 backcross lines provide an easier method for isolating lines that segregate for only one gene. Regardless of the method that is followed, i.e. F_2 backcrosses or F_3 families, a study of segregation among families rather than within families is more dependable.

Results obtained from field observations indicated that Rosner carries an additional gene for adult-plant resistance which has already been described. In all the other resistant triticale lines it was not possible to determine if adult-plant resistance governed by major genes was pre-

sent in addition to the seedling genes that were identified. However, the presence of minor genes contributing to rust resistance in adult plants was suggested by the observed continuous variation in rust reaction from moderate resistance to susceptibility among F_2 backcross and F_3 families which lacked seedling resistance genes.

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SECTION FOUR - GENERAL DISCUSSION.

GENERAL DISCUSSION

During the process of developing triticale into a crop species of commercial value, several factors which could potentially restrict its improvement have been encountered. Among these factors, reduced yield due to restricted adaptation, weak straw, and disease problems are of primary importance.

Little is known concerning the genetic factors that contribute to disease resistance in triticale. Reports found in the literature are mainly related to observations of resistance to several pathogens, but no genetic studies of disease resistance have been undertaken (3, 11, 12, 19, 20, 22, 26).

It was the object of the present investigation to study the inheritance of leaf rust resistance in hexaploid triticale. As a working hypothesis it was assumed that the resistance of triticale was under the genetic control of both wheat and rye, the parental genera. Since wheat is resistant to the rust forms which attack rye, and rye is reported to be resistant to the rusts specialized on wheat (14, 25), a phenomenon of cross-protection against the rust pathogens was envisaged.

The results of this investigation suggest that the resistance to leaf rust carried by the rye parent was not expressed in the triticales studied. This finding was unexpected since resistance to leaf rust has been transferred from rye to wheat by translocating a small segment of the rye chromosome carrying the resistance gene (5). It appears, however, that when the complete genomes of wheat and rye are combined in the amphiploid, complex genetic interactions take place which inhibit the expression of certain genes including those from rye governing leaf rust resistance. Further studies in this field are needed to gain a better

understanding of the kind of intragenomic relationships that exist in the amphiploid. It is of the upmost importance to determine whether some triticale lines exist where the wheat and rye resistance are expressed as they may provide by cross-protection a long-standing barrier to the evolution of virulent races of the pathogen.

The results obtained indicated that resistance to leaf rust was under genetic control and dependent upon the wheat genes introduced into triticale. Resistance in the lines studied was found to be monogenically inherited. The resistance to leaf rust governed by a single gene has also been reported in several cultivars of common wheat (1, 4, 15, 23).

However, the production of cultivars with single resistance genes has limited value in controlling rust diseases. New pathogenic races are continuously evolving which can overcome monogenically resistant cultivars, by means of asexual and/or sexual variation (13, 16, 18). Recently, however, the combination of several genes for resistance into single crop cultivars has become a common practice (7, 13, 16). This procedure is also suggested for triticale if a broad and lasting resistance is to be obtained.

It was observed that triticale seedlings lacking genes for wheat leaf rust resistance were susceptible when inoculated with some strains of rye leaf rust. However, this rust form was found to be less aggressive than wheat leaf rust. This was indicated by a longer generation period and by the reduced number of sporulating pustules produced per leaf, even when a heavy concentration of spores was used. The reduced number of lesions per leaf may be due to an inadequate incubation period, which permitted only a limited number of spores to germinate and become established. If this is true, rye leaf rust would require a longer incubation period than that

ordinarily given to wheat leaf rust, i.e. more than 20 hours under high relative humidity after inoculation. The lack of aggressiveness on the part of the rye leaf rust partly explains the fact that under field conditions only wheat leaf rust is collected from susceptible triticales, even when rye rust is present in noticeable quantities on rye cultivars.

Evidence obtained in this study and elsewhere (19), showed that the resistance genes present in hexaploid triticale confer protection to all the leaf rust races so far investigated. This can be explained by assuming that such resistance genes have not been used in North America in breeding wheat cultivars, and consequently the pathogen population has not evolved virulence to those genes. It is very likely, however, that as triticale is cultivated on a commercial scale, new races of the pathogen would be identified with virulence on triticale genes for resistance.

The extremely high potential for variation found in the cereal rusts makes it necessary for the breeder to investigate all possible sources of host resistance. The general type of resistance reported in other crop species (2, 9, 10, 17, 24) deserves special consideration because it affords protection against the entire pathogen population. Limited field evidence was obtained during the course of this investigation which suggested that general resistance based on a polygenic system may be operative in triticale. Slow rusting and relatively light infections were observed in several segregating families which did not carry seedling resistance genes. Similar observations were reported by Zillinsky and Borlaug (26). No information is available to indicate whether this type of resistance is under the genetic control of wheat, rye, or both, and further work is required on this problem. The production of crop cultivars with combined general and specific resistance is of unquestion-

able value. Unfortunately, a breeding method has yet to be devised to allow the incorporation of these two kinds of resistance in the same cultivar.

The isolation of single resistance genes in a common susceptible background by means of backcrossing has been carried out in several host-parasite systems (1, 8). These isogenic lines would be useful to characterize resistance genes in the same background and in the absence of modifiers. They would also be valuable in race identification by relating the number of virulent genes in the pathogen to the number of host resistance genes (1, 8, 21). In view of its practical value it is recommended that a similar approach be undertaken in triticale.

Further investigations are needed to determine the complete spectrum of resistance afforded by each gene identified in this study. If races of leaf rust possessing virulence to some genes are found, a study on the inheritance of virulence on the part of the pathogen could be carried out to determine whether or not the gene-for-gene theory as proposed by Flor (6) is also applicable to triticale and wheat leaf rust.

This is the first study dealing specifically with genetic mechanisms of disease resistance in hexaploid triticale. The information gained should provide the necessary basis from which additional investigations could be undertaken.

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APPENDIX

TABLE A.1 - Seedling reaction to race 30 of wheat leaf rust and to rye leaf rust isolate RLR-1/71 of a selected number of backcross lines, which previously were scored as homozygous susceptible to race 15 of wheat leaf rust.

Backcross combination	Number of lines	Leaf rust reactions		
		Race 15	Race 30	isolate RLR-1/71
Acc. 6239[2] x 6A-190	23	4	4	4
Rosner "S"[2] x Rosner	30	4	4	4
6A-276[2] x Armadillo	16	4	4	4
Bronco "S"[2] x Bronco	13	4	4	4
Toluca 160 "S"[2] x Toluca	25	4	4	4

TABLE A.2 - Segregation of reaction to race 30 of wheat leaf rust, and to the rye leaf rust isolate RLR-1/71 in F_3 families derived from resistant x susceptible crosses.

	Number of families					
	Race 30			isolate RLR-1/71		
Cross combination	Resistant	Susceptible	Segregating	Resistant	Susceptible	Segregating
Acc. 6239 x 6A-190	5	9	7	5	9	7
Rosner "S" x Rosner	5	14	6	5	14	6
Acc. 6239 x Armadillo	4	13	8	4	13	8
Bronco "S" x Bronco	13	7	9	13	7	9
Toluca 160 "S" x Toluca 160	12	20	8	12	20	8

TABLE A.3 - Fertility of the hexaploid triticales used in the present study, and of three cultivars of durum wheat.

Name or Accession No.	No. of spikes examined	Ave. No. of spikelets	Ave. No. of seeds/spike	Ave. No. of florets/spike	% of florets with seed	Ave. No. of seeds/spikelets
GROUP 1						
Hexaploid triticales						
6A-190	10	11.7	55.6	79.8	69.6	2.3
Rosner	10	9.8	75.0	91.4	82.0	3.8
Rosner "S"	10	11.8	82.2	104.2	78.8	3.4
Armadillo	10	9.1	66.8	77.0	86.7	3.6
Bronco	10	9.7	65.8	79.8	82.4	3.3
Bronco "S"	10	9.7	56.8	70.4	80.6	2.9
Toluca 160	10	9.7	64.8	76.6	84.5	3.3
Toluca 160 "S"	10	8.9	49.6	62.6	79.2	2.7
6A-276	10	12.0	64.8	88.8	72.9	2.7
Acc. 6239	10	13.1	71.8	90.4	79.4	2.7
Acc. 6507	10	10.0	65	83.2	78.1	3.2
GROUP 2						
Durum wheat cultivars						
Carleton	10	8.8	53.8	59.6	90.2	3.0
Ghiza	10	8.0	51.4	59.6	86.2	3.2
Stewart 63	10	8.9	57.6	65.8	87.5	3.2