THE EVALUATION OF 391 SPRING WHEAT INTRODUCTIONS FOR RESISTANCE TO STEM RUST, LEAF RUST, LOOSE SMUT AND TAN SPOT.

A Thesis

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by

Pierre-Philippe Claude

In Partial Fulfillment of the

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FOREWORD

This thesis is written in the ´thesis sytle´ specified in the 1976 Plant Science Thesis Preperation Guide. This format has been approved by the Master´s Thesis Examining Committee. A manuscript entitled ´The evaluation of 391 spring wheat introductions for resistance to stem and leaf rust´ will be derived from this thesis. It is intended that the manuscript be submitted for publication to the Canadian Journal of Genetics and Cytology.

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GENERAL ABSTRACT

Claude, Pierre-Philippe, The University of Manitoba,

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An Evaluation of 391 Spring Wheat Introductions for Resistance to Stem and Leaf Rust, Loose Smut and Tan Spot.

Three hundred ninety one (391) spring wheat introductions from Asian, Middle Eastern and Mediteranean areas were screened for resistance to races C17, C20, C25, C49, C53 and C57 of *Puccinia graminis tritici*; races 1, 5, 9, 15 and bulks 1, 4 and 10 of *Puccinia recondita*; races T2, T10, T13 and T39 of *Ustilago tritici* and to 6 isolates of *Drechslera tritici-repentis* prevalent in western Canada.

Of the 34 introductions resistant to *P. graminis tritici*, 15 were genetically studied using F2 segregation data derived from the progeny of the crosses involving resistant introductions, their corresponding near isogenic lines and stem rust universal suscepts. Eleven of these were found to carry single Sr genes for resistance, notably, *Sr30*, *Sr13* and *Sr15*. Of the 70 introductions resistant to *P. recondita*, 28 were studied and 9 were found to carry known Lr genes for resistance, notably *Lr10* and the genes present in RL6057 and RL6061. Twenty two introductions are believed to carry either

1 or 2 unidentified dominant, recessive, partially dominant and/or complementary genes for resistance to either stem or leaf rust.

Five introductions were immune and 6 highly resistant to the 4 races of U. tritici. Sixty-nine introductions were resistant to D. tritici-repentis. These were arbitrarily classified into 10 'phenotypic classes' according to their reactions to the 6 isolates used.

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INTRODUCTION

Centers of origin of cultivated crops as described by Vavilov (1949) have repeatedly been said to be excellent sources of genes for disease resistance (Dietel 1904; Vavilov 1939; D´Oliveira 1940, 1951, 1960; Wahl 1958; Leppick 1970). These regions often coincide with areas where large amounts of genetically varied forms exist (Vavilov 1926). Of particular interest to wheat scientists are the Central Asiatic, Near-Eastern, Mediterranean and Abyssinian centers as described by Vavilov (1949). Harlan (1971) modified Vavilovs´ proposals and described three centers of origin, two of which, the Near East and North Chinese centers are considered to be wheats´ centers of origin.

The spread of intensive agriculture and its´ emphasis on crop uniformity has led to the destruction of a sizable portion of the genetic variability in these regions. Bennett (1970) noted that;

> "... at the present time the loss of genetic resources is taking place so rapidly that there is grave concern for numerous crop races in the centers of diversity, - one might even say with some justification, centers of ´former´

> diversity... Reference to the collections described by Vavilov and his colleagues, and to more recent collections made in the Mediterranean, the Near East and in Central Asia, shows that many local races of wheat and other crops, described by earlier expeditions, have now disappeared from their native habitats or have become rare, and the same is true in other parts of the world... "

The value of the present world collections and their conservation, multiplication and evaluation thus becomes apparent.

Metcalfe et al (1978) reported that the evaluation of 226 barleys from Ethiopia for disease reaction to pathogens prevalent in Canada indicated that this region was a good source of disease resistance. Martens et al (1980) reported that 21 genes for resistance to Puccinia coronata avenae had been identified in oat materials from north African and Middle Eastern collections and had been transferred to cultivated oats. The authors noted that these genes were widely used in oat breeding programs in many parts of the world.

Bartos et al (1969) screened a number of European wheat varieties for resistance to leaf rust. In this particular study the results suggested that the rust resistance of many of these European varieties was controlled by genes that had

already been described in North America. This was not to be unexpected since North American varieties resistant to leaf rust have been used in many European wheat breeding programs and vice-versa. Bartos et al (1970) screened a number of European wheat varieties for resistance to stem rust. Again the results suggested that the European varieties carried several identified Sr genes already known to exist in North America. However, they were also shown to carry a few resistance genes not previously investigated. The use of European wheat cultivars as sources of new disease resistance for North America thus appears to be limited by the common origin of the breeding material used in developing them. Wild, non-improved and non-varietal wheat materials collected in areas corresponding approximately to Vavilovian centers may prove to be better sources of new genes for resistance.

The objective of the present study is to evaluate 391 spring wheat introductions (Triticum aestivum L.) from southern European, Asian, north African, Mediterranean and Middle Eastern areas for resistance to isolates of leaf rust (Puccinia recondita Rob. ex Desm.), stem rust (Puccinia graminis Pers.f.sp.tritici Eriks. and E.Henn), tan-spot (Drechslera tritici-repentis(Died.)Shoem.) and loose smut (Ustilago tritici Pers.). Also, the genetics of some of the introductions found to be rust resistant will be studied in the hope of identifying new genes for resistance to both stem and leaf rust of wheat.

LITERATURE REVIEW

General

The Rusts

The rust fungi are traditionally divided into two families; (1) Melampsoraceae and (2) Pucciniaceae. Puccinia graminis tritici Eriks. and Henn. (stem rust) and P. recondita Rob. ex. Desm. (leaf rust) are included in the latter. The leaf rust and stem rust fungi are heteroecious and macrocyclic Basidiomycetes belonging to the order Uredinales.

Control. Dickson (1959) mentioned phenolic, inorganic sulfur and organic sulfur componds along with some metals (Cu, Mg, B, Se, F, Fe, Li, Mn, Ni), antibiotics and growth regulators as possible chemical agents to control wheat rusts. He concluded his review by noting that the control of cereal rusts through the use of fungicides did not appear economical. According to Rowell (1968), one or two applications of the fungicides resulted in only partial control of cereal rusts and that the short duration of activity of these systemic and protectant fungicides limited their effectiveness. He also noted that the need for fungicidal activity to persist through the last month of

wheat development conflicted with the requirements for minimum residues on the harvested grain. The use of genetically controlled host resistance is by far the most common means of control of cereal rusts in the great plains region of North America (Agrios 1978). However, the higher yields and more intensive cereal management practices help make the chemical control of cereal rusts more economical in Europe.

Loose Smut.

Ustilago tritici Pers. is reponsible for the loose smut disease of wheat. It is a Basidiomycete, part of the order Ustilaginales. Resistance to infection is present in most cultivars recommended in Manitoba (Manitoba Agriculture 1985). Seed treatment with carboxin and other derivatives of 1,4-oxanthin and the use of disease free seed are also possible means of control (Agrios 1978).

Tan-Spot

The fungus responsible for the tan-spot disease of wheat is an Ascomycete whose asexual and sexual stages are known as Pyrenophora tritici-repentis Died. and Drechslera tritici-repentis (Died.) Shoem. respectively. It was first identified on grasses in Germany in 1902 and on wheat in Japan in 1928. It´s host range includes at least 33 cereal and grass species, including Agropyron, Elymus, Triticum,

Hordeum, Avena and Secale (Krupinsky 1981). Recently the disease has risen from a position of minor importance on wheat to that of high priority mainly because of changing cultivar genotypes (Cantrell 1982; Gough and Johnston 1982) and cultural practicies (Cantrell 1982; Gough and Johnston 1982; Rees 1982 and Watkins et al 1978).

Control. Biological anatagonists of the tan spot organism have been reported by Gough and Ghazanfari (1981). Some of these were cited as possibly becoming control agents in the future. Lamey (1981) presented an overview of minimum tillage and chemical control methods for tan spot. He indicated that under North Dakota conditions at the time, an economic return from spraying was possible. Hard red spring wheats have been bred for increased resistance (Frohberg 1982) and simple (Gough 1982; Frohberg 1982) and more complex (Nagle et al 1982) inheritance mechanisms have been reported.

A Few Definitions.

(1)Disease. Disease can be seen as a malfunctioning of a biological process or, in the words of Horsfall and Cowling (1977), "a malfunctioning process that is caused by continuous irritation". This "continous irritation" may be caused by biological and/or non-biological agents. Heat and water stress, air pollution and acid or alkali soils are example of non-biological agents. Biological agents are more relavent to this thesis. Rodents, insects, bacteria,

nematodes and fungi are all important biological agents. Plants can defend themselves from such continous irritation either by resistance, tolerance or both. An extreme form of resistance being 'immunity'.

(2)Disease Resistance. Disease resistance can be defined as the ability of a host to contain infection and colonization by a pathogen thus preventing sizable damage" (Cowling and Horsfall 1980). Cowling and Horsfall (1980) refer to resistance as "defense". They note that;

> "Both medieval castles and plant hosts are immobile; they have given up important options-the ability to move, to side step onrushing pathogens, to retreat. Both must stand and wait, and then have it out with their attackers. Thus their defenses must be even stronger than otherwise. Like a Castle, the defense by the host begins at the perimeter- at the outer wall and the gate."

In addition to the medieval castle analogy, the concept of "Aegricorpus" (Loegering 1966) helps understand the phenomenon of disease and disease resistance. According to Loegering (1966), once the plant is diseased, the plant and the organisms inhabiting it become one; the "Aegricorpus". Both plant and pathogen are altered. Gaumann (1950) noted three such pathogen-induced changes in the host plant; (1) biological predisposition, (2) induced antiinfectional

defense reactions and (3) induced tolerance. Disease resistance is included and partly defined by the second, "induced anti-infectional defense reactions". "Immunity" to disease represents an extreme case of resistance where the pathogen is contained almost immediatly after it´s contact with the host. Immunity nevertheless does allow for some very small lesion formation barely visible with the naked eye. Immunity can break down if these small lesions are numerous and coalesce into larger ones.

(3)Disease Tolerance. Disease resistance and immunity imply the exclusion or containment of the pathogen. If the pathogen is not fully contained or excluded and the host plant still manages to yield a crop comparable to that it would have yielded in the absence of the pathogen, the plant is said to possess "tolerance" to the pathogen. Simply stated, tolerance is the ability of plants to produce a good crop despite the insults of the pathogens (Mussell 1980).

Nature of Resistance.

The nature of resistance refers to the mechanisms in the host plant responsible for either physiological, morphological or biochemical changes hindering the progress of the invading pathogen. According to Chakravorty (1982),

> "...host parasite interactions trigger changes in the patterns of gene expression in the host plant

> at a very early stage. The direction and magnitude of these changes determine whether the host will be susceptible or resistant to the pathogen..."

The morphological and biochemical aspects of host resistance were reviewed by Heath, (1982). Notable aspects were;

> (1) cessation of fungal growth during the actual process of penetration (Sood and Sackston, 1970) due to the surrounding host cells secreting a constitutional or inducible toxin, (ie: phytoalexin),(Zimmer, 1965)
> (2) necrosis of haustorium-containing host cells
> (3) encasement of haustoria in callose containing material (Heath, 1971).

Physiological changes in the rusted host tissue partly responsible for the expression of either resistance or susceptibility are two fold. First, there is a ´juvenile host response´ (Bushnell 1967; Allen 1923) during which cells are kept physiologically young, the nuclei of the host cells increase in volume, there is an increase in the synthesis of nucleolar and extranucleolar RNA and a general increase in the size and number of organelles. These events are followed by an ´autolytic host response´ (Bushnell 1967; Allen 1923) when the volume of the host cell nuclei and organelles regresses and the cells may eventually become necrotic and

lyse. Once a host cell has lysed, an obligate parasite such as rust will be contained within that cell, unless it has had the time to infect neighboring cells prior to cell lyses. The faster the autolytic response occurs, the less time the pathogen will have to inflict damage on neighboring cells and the greater will be the level of resistance (Rowell 1981). Immunity is essentially due to an almost immediate autolytic response, and total suceptibility to a quasi non-existance of such an autolytic response.

Possible classifications of resistance.

(1)Host vs Nonhost Classification: Ward and Stoessel (1976) mentioned the concept of ´nonhost resistance´ and ´cultivar´ or ´host resistance´. The concept refers to the basic incompatibilities between a pathogen and a plant outside it´s host range (ie:nonhost resistance) and to the more specific incompatibilities between a pathogen and a plant within it´s host range (ie:cultivar resistance). According to Heath (1982) "the responses seen in nonhost plants have been suggested to be part of a battery of potential defense mechanisms possessed by every plant, of which one or more is non-specifically elicited by any plant pathogen for which the plant is not a host". Pathogenic fungi are those fungi which have been able to overcome these nonhost defense mechanisms (Heath 1974).

(2)Oligo- vs Polygenic Classification: The work of Barrus (1911), Stakman (1914) and Flor (1942, 1947, 1956, 1955) greatly contributed to the study of the genetics of resistance in plants. These investigations were well suited for the identification of host resistance controlled by one gene (monogenic) or a few gene (oligogenic). More complex modes of inheritance are often inferred and are said to involve more than a few genes (polygenic) (Ausemus 1946)

(3)Major vs Minor Gene Classification: Vanderplank (1968) discussed briefly the concept of major and minor gene resistance as a possible criterion for classifying types of resistance. He dismisses such a classification on the basis that strong (major) and weak (minor) genes do not always behave as their names would imply.

(4)Vertical vs Horizontal Classification: The concept of vertical and horizontal resistance was proposed by Vanderplank (1968) and reiterated by Robinson (1976). The classification is based on the idea that vertical resistance is very effective against one or a few races of a pathogen (ie: race-specific) while horizontal resistance is somewhat less effective against all races of the pathogen (ie: non race-specific). Horizontal resistance is supposedly more 'durable' than vertical resistance in that pathogens apparently fail to evolve exceptionally strong virulence against it.

Methods to Evaluate Resistance.

To measure the amount of disease four measurements can be made; incidence, severity, intensity and infection type (IT). These measurements can in turn give good indications as to the level of resistance present.

'Incidence' refers to the proportion of plants in a given area which are diseased. 'Severity' refers to the percentage (or proportion) of leaf area which is either covered or affected by the disease. 'Intensity' is the product of incidence and severity. 'Infection types' (ITs) as described by Stakman (1914) are of particular importance in rust research. IT readings for Puccinia graminis tritici and Puccinia recondita are made on a 1 - 4 scale according to Stakman (1914). With proper training, infection types can be classified fairly precisely and consistently, (Appendix C).

The determination of severity is more subjective than that of IT. Cobb (1892) drew sketches of rust infected leaves showing diagramatically five degrees of rust ranging from 1 to 50%. By comparing the sketches with real leaves he could derive a measure of severity. The Cobb Scale, one of the first disease assessment scales to be devised, was subsequently modified by Melchers and Parker (1922). This modified scale, although improved, did not fully account for the various shapes and sizes of rust pustules. A five percent coverage could be due to either many small pustules or a few larger pustules. The modified Cobb Scale did not allow for

such interpretations. In response to this situation, a series of standard diagrams were devised by Peterson et al (1948) (Appendix B). The different diagrams accounted for different pustule and leaf sizes encountered when determining the percentage surface area affected by rust.

Centers of Origin Vs Centers of Diversity.

Candolle in 1886 (Candolle 1959) was one of the first to discuss extensively phytogeography and crop origin and domestication. His work, while being of academic and intellectual interest, was limited by the scant archeological knowledge at the time. On the other hand, Vavilov (1949) had at his disposal an extensive network of scientific research centers and personnel. His work was consequently more exhaustive and conclusive. Vavilov (1926) proposed that centers of origin of crops could be reliably determined by the analyses of patterns of variations. The geographic region with the greatest genetic diversity of a crop plant was considered to be its center of origin. Based on the evidence he accumulated, Vavilov (1949) went on to propose 8 'centers of origin' of cultivated plants.

(1) The Chinese Center

(2) The Indian Center

(2a) The Indo-Malayan Center

(3) The Central-Asiatic Center

(4) The Near-Eastern Center

(5) The Mediterranean Center

(6) The Abyssinian Center

(7) The South Mexican and Central American Center

(8) The South American Center

(8a) The Chilean Center

(8b) The Brazilian-Paraguayan Center

Wheats apparently originated from centers 3, 4, 5 and 6.

Vavilovian centers are actually more like ´centers of diversity´ and not necessairly ´centers of origin´. These regions, as described by Vavilov (1949), do possess large amounts of genetic variability (Leppick 1970; Flor 1971; D´Oliveria 1940, 1951, 1960), but are not necessarily unique and do not always account for archeological and anthropological data. This discrepancy has been reported by various authors, (Harlan 1951, 1971; Zohary 1970). Harlan (1971) thus modified Vavilovs´ proposals accordingly and described three independant systems (A, B and C) each comprising a center of origin (A1, B1 and C1) and a ´noncenter´ or ´secondary center´ of origin (A2, B2 and C2);

A1: Near East Center

A2: African Non-Center

B1: North-Chinese Center

B2: South-East Asian and South Pacific Non-Centers

C1: Meso-American Center

C2: South American Non-Center

Cultivated crops are said to have originated in A1, B1 or C1 and later spread to A2, B2 and C2. Centers A1 and B1 correspond approximately to Vavilovian centres 4 and 5, that is wheats´ centers of origin.

Realizing that neither centers of origin as described by Vavilov (1949) or ´noncenters´ of origin as described by Harlan (1971) could account for the origin, diversity and distribution of all cultivated crops, Harlan (1975) set out to propose different ´evolutionary patterns´ applicable to different crops. The main patterns are classified as follows;

Endemic: applicable to crops that originated in a limited area and that did not spread appreciably

Semiendemic: applicable to crops that originated in a definable center and with a limited dispersal

Monocentric: applicable to crops with a definable center of origin and wide dispersal

Oligocentric: applicable to crops with a definable center of origin, a wide dispersal and one or more secondary centers (non-centers) of diversity

Noncentric: applicable to crops whose pattern of variation suggest domestication over a wide area

Regardless of pattern of evolution and, whether or not centers of origin and centers of diversity coincide, geographical regions containing much genetic variability do exist (Harlan 1975). These regions coincide approximately with Vavilovian and Harlanian centers of origin, and are presumably excellent sources of new resistant plant materials (Coons 1953; D´Oliveria 1940, 1951, 1960; Zhukovsky 1959; Flor 1971; Nelson 1978). As early as 1904, Dietel (1904) noted the co-evolution of plant pathogens and their hosts within these centers of diversity. Vavilov (1939) demonstrated that the "epicenters" of wheat corresponded to those of its most destructive diseases, notably, the rusts. Vavilovs´ demonstration was later supported by the studies of Wahl (1958), Zhukovsky (1959) and Urban (1980). Leppick (1970) reviewed literature concerning ´gene centers´ for resistance for a number of crops. ´Gene centers´ refer to the geographical origins of materials carrying genes responsible for the expression of particular traits such as disease resistance and free-threshability. Invariably, these ´gene centers´ coincided with Vavilovian centers of origins.

Landraces.

Wild hexaploid wheats as such do not exist. Hexaploid T. aestivum probably originated and entered cultivation only after the more or less simultaneous domestication of diploid and tetraploid forms (Feldman 1976). It was introduced into

the new world in 1529 when the Spaniards took it to Mexico and to Australia in 1789 (Feldman 1976). Within the T. aestivum species, 6 varieties are recognized by Feldman (1976), 3 are hulled (ie: varieties spelta, macha and vavilovii) and 3 are free-threshing (ie: varieties aestivum, compactum and sphareococcum). T. aestivum var. aestivum gave rise to T. aestivum var. compactum and T. aestivum var. sphareococcum through mutation (Feldman 1976). The free-threshing character of the latter makes them economically more advantageous than the hulled varieties. These free-threshing varieties have mainly existed in the form of 'landraces'. Landraces are essentially a mixture of homozygous lines differing in their degree of disease resistance, threshability, height and other qualitative and quantitative traits. Landraces are usually poor agronomically. Nevertheless, it is only in the last century or so that landraces have been replaced by uniform, true-breeding cultivars (Harlan 1975). Although between 14 000 (Zeven and Zeven-Hissink 1976) and 17 000 (Feldman 1976) different cultivars have so far been developed by plant breeders across the world, landraces are still grown wherever traditional agriculture is practiced. Landraces obtained from areas coinciding with wheats' centers of diversity should prove to be good sources of resistance genes. Harlan (1975) noted that;

> "... landrace populations are often highly variable in appearance but they are identifiable and usually have local names.... Genetic variation within a landrace may be considerable.... The great variability of landraces makes them good sources of genes for modern plant breeding.... "

Other than their use as gene sources, landraces are of little use in modern agriculture since they are not adapted to high fertility, high plant populations, or intensive production (Harlan 1975).

Genetics of Leaf and Stem Rust Resistance

Demonstration of Mendelian Inheritance

Biffen (1905, 1907, 1912) demonstrated that resistance to stripe rust of wheat, Puccinia striiformis West., was inherited as a single recessive Mendelian factor. Biffen observed a close adherence to a 3:1 segregation for susceptibility and resistance in F2 populations derived from crosses between rust-resistant Rivet and susceptible Red King. Hayes et al (1920) noted that while many observations had been made on the resistance of wheat varieties, only Biffen's (1907) and Nilsson-Ehle's (1911) were considered at the time carefully controlled studies which showed the mode of inheritance of stripe rust resistance. The authors concluded that the mode of inheritance of rust resistance

seemed entirely comparable with the general Mendelian manner of inheritance of botanical and morphological characters.

Genotypes of Stem (SR) and Leaf (LR) Rust Resistant Wheat Cultivars Grown in Canada.

Green and Campbell (1979) listed stem rust resistant spring wheat cultivars released for production in the rust area of western Canada. They estimated the annual value of wheat stem rust resistance in that area at $217 million. Cultivars released after 1951 and their corresponding Sr genotypes are listed below (Green and Campbell 1979);

Cultivar	Year licensed	Sr Genotype
Selkirk	1953	Sr6, 7b, 9d, 17, 23
Canthatch	1959	Sr5, 7a, 9g, 12, 16
Pembina	1959	Sr5, 6, plus
Manitou	1965	Sr5, 6, 7a, plus
Neepawa	1969	Sr5, plus
Pitic 62	1969	Sr8, Sr9
Napayo	1972	Sr5, 6, 7a, plus
Glenlea	1972	
Norquay	1974	
Sinton	1975	

The actual genotypes for most of the spring wheat cultivars grown in western Canada has not yet been determined. However,

since many of them are of a Thatcher background they probably have similar Sr/Lr genotypes, that is; Sr 5, 9g, 12, 16 (McIntosh et al 1981) and Lr22b (Dyck 1979). The recently introduced cultivars Benito has genes Lr1, 2a, 12 & 13 (Campbell and Czarnecki 1981), Columbus has Lr13 & 16 (Samborski and Dyck 1982) and Katepwa has Sr11 (A.B. Campbell, - unpublished data).

Sexual Recombination and Variability Within the Rust Fungi

Eriksson (1894) noted that although the morphology of the Puccinia graminis fungi attacking different cereal species were similar, there was a distinct specialization in parasitism. He subsequently identified "formae specialis" (specific forms) within the Puccinia graminis species. P. graminis f. sp. tritici, P. graminis f. sp. hordei, P. graminis f. sp. secalis and P. graminis f. sp. avenae were said to specifically parasitize wheat, barley, rye and oats respectively. The 'f.sp.' notation may be omitted, eg: P. graminis tritici.

Stakman (1914) and Stakman and Levine (1922) published studies on cereal rusts concerning the occurrence of 'physiologic races' of wheat stem rust. Using a set of 12 wheat varieties, which are now known as the Stakman Differentials, he showed that a number of stem rust isolates reacted differentially to the 12 wheat varieties. That is, their distinctive physiological natures enabled them to

attack certain varieties of wheat but not others. The specific reactions of these races on each of the 12 wheat differentials could be used as a means of classifying them.

Variability within the wheat stem and leaf rust fungi is in part attributable to sexual recombination. Craigie (1927) was the first to link the pycnia to sexual recombination in P. graminis tritici. Craigie (1931) developed a method by which cultures derived from single spores of the fungi could be hybridized. He also reported the origin of a 'new' race obtained through such hybridization.

New races of wheat rust may also arise through asexual means such as somatic hybridization between different formae speciales, mutations, physiological adaptations and progressive changes in virulence (Watson 1970). In a study by Luig and Watson (1972) it was shown that Australian native and cultivated grasses could play an effective part in the evolution of new strains of P. graminis. Agropyron scabrum and Hordeum leporinum appeared to be important as sources of somatic hybrids involving P. graminis tritici and P. graminis secalis. According to the authors, the principles governing hybridization between these two formae speciales may also be applicable to corresponding events among other rusts, notably, leaf and stem rust of wheat.

The Significance of Sexual and Asexual Recombination Within the Rust Fungi.

One of the immediate consequences of recombination within the rust fungus is the potential for new virulent races to arise. According to Vanderplank (1968), recombination within the rust fungus and the widespread use of resistant varieties carrying only one or a few resistance genes has led to a rapid selection and increase of isolates capable of attacking these resistant cultivars. Vanderplank (1982) describes two types of selection; (1) ´directional´, adapting the pathogen to the host and, (2) ´stabilizing´, hindering this adaptation. Directional selection is said to have two effects;

(1) avirulence becomes rare or rarer, because the avirulent pathogen is not adapted to the resistant host cultivars,
(2) virulence becomes more common (Vanderplank 1982).

Simultaneously, stabilizing selection is said to operate in an opposite manner, hindering this adaptation of the pathogen to the the resistant host. Vanderplank (1982) argues that the end result of these two opposite selection pressures acting upon a host-pathogen system is "a frequency pattern with rare extremes and abundant intermediates where much avirulence and much virulence are rare and intermediate, and presumably adequate, virulence is common".

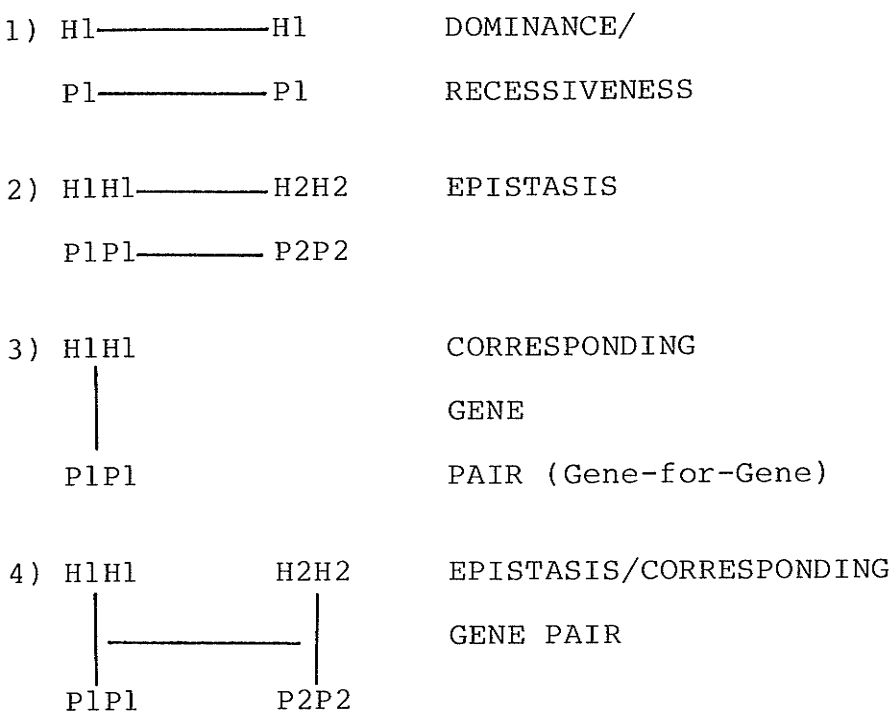
According to Vanderplank (1982), this type of equilibrated situation can be disturbed if either of the selection pressures is altered. The growing of large acreages of genetically uniform and true-breeding wheat cultivars, often carrying only a few genes for resistance, is seen as an increased stabilizing selection jeopardizing this homeostatic condition. This phenomemom is said to be responsible for the relatively rapid 'boom and bust' cycle involving resistant varieties in the major wheat growing regions of the developed world (Robinson 1976). Krivchenko and Tikhomirov (1981) noted that in all climatic zones of the USSR the composition of the Ustilago tritici and Puccinia spp. race populations are constantly changing as a result of the introduction of new wheat varieties and the instability of races in respect of virulence. Each race is said to consist of many intraracial units whose virulence differs from that of the race as a whole and can itself vary. The heterogeneity and heterozygosity of the pathogen in respect to virulence is said to enable new races and isolates to develop and allow the most virulent to be fixed in the fungal populations.

The whole concept of stabilizing selection acting upon pathogen populations as described by Vanderplank (1968, 1982) has been questioned. In a paper by Parlevliet (1981) entitled: 'Stabilizing Selection in Crop Pathosystems: an empty concept or a reality?', the whole concept as applied to cultivated crops and their pathogens was systematically

demolished. Parlevliet concluded that stabilizing selection as a general phenomenon does not exist and that it appeared to be an empty concept. He suggests that strategies for improving disease resistance on the basis of Vanderplanks' stabilizing selection concept should be reconsidered. Even Vanderplank (1982) noted that vertical resistance, supposedly very vulnerable to directional selection, has often been introduced into major crops and has remained effective since. According to the author, the position at the moment is that maize, wheat and other grains, sugarcane, beet and many other field crops are being grown with little or no protection by fungicide except for seed and planting material. Some of this resistance is said to be controlled by one or a few genes. The author also notes that millions of hectares of crops are presently being grown successfully under the protection of such simply inherited vertical resistance. In these instances resistance in the host plant has not brought about matching changes in the pathogen which allows for compatibility and the expression of susceptibility.

Host-Pathogen Genotypic Interactions

Loegering and Powers (1962) described four types of host--pathogen interactions possible at the genotypic level;



where H represents a host gene for resistance and P a pathogen gene for virulence.

Interaction number 3 is commonly refered to as the Gene-for-Gene hypothesis proposed by H.H. Flor (Flor 1955). The theory stipulates that for every resistance gene in the host population there is a corresponding matching gene for avirulence in the parasite population. Resistance is effective only if the host possesses a dominant allele for resistance and the pathogen possesses a dominant allele for avirulence at the corresponding locus (resistance and avirulence do not necessarily have to be dominant). Such a gene-for-gene relationship was demonstrated for wheat leaf rust by Samborski and Dyck (1968, 1976) and Bartos et al (1969_A) and for wheat stem rust by Green (1966) and Kao and Knott (1969).

Interaction and Enhancement of Resistance genes

Additional resistance genes introduced into cultivars often appear to interact synergically. The resulting resistance is often of a higher degree than that expressed by the individual genes in similar backgrounds. Luig and Watson (1970) noted that;

> "Asexual recombinants and mutants provide the variability which has enabled the rust to attack resistant varieties. As more genes are accumulated into the latter the rust adapts by gaining corresponding genes for virulence. There is strong evidence that a negative relationship exists between the number of genes for virulence and aggressiveness of the pathogen ... A broadly based specific resistance coupled with genetic diversity in the cultivars has protected the Australian wheat crop from stem rust for more than twenty years."

Samborski and Dyck (1982) reported that the resistance of the spring wheat cultivar Columbus to P. recondita was enhanced due to the interaction of genes Lr13 and Lr16 and that highly resistant selections were obtained from a cross involving four lines each with a single gene conditioning a lower level of resistance. Voronkova (1980) noted the

manifestation of a complex interaction of genes for resistance to P. recondita in wheat. It was stated that, "only an interaction between different genes can account for the fact that a varying percentage of highly resistant plants is obtained when one particular resistant variety is crossed with susceptible varieties." It was concluded that the uniting of different groups of genes is very desirable in creating a stronger rust barrier.

Adult Plant Resistance

Genes Lr22 (Dyck and Kerber, 1970), 12 and 13 (Dyck et al, 1966) and a gene present in RL5711 (E.R. Kerber, personal communication) are known as 'adult plant resistance genes'. Lr22 has been derived from Aegliops squarrosa and genes Lr12 and 13 from the common wheat varieties Exchange and Frontana. The gene in RL5711 was probably derived from Aegilops speltoides. When tested against particular, races these genes confer little or no resistance at the seedling stage and moderate to excellent resistance at the adult plant stage. To some races, Lr13 will confer resistance at both the seedling and adult plant stages. Sr2 (Knott 1968) has been the only gene identified for adult plant resistance to P. graminis tritici.

According to Dyck et al (1966) studies on the inheritance of adult plant resistance are difficult for several reasons. First, the presence of seedling resistance in most varieties

masks the expression of adult plant resistance. Second, modifying genes notably affect the expression of adult plant resistance and, finally, the effect of the environment on both adult plant genes and their modifiers is substanstial. These limitations, along with the fact that the inoculation and the scoring of adult plants is tedious and time consuming, may explain why the genetic nature of adult plant resistance is poorly understood.

Transgressive Segregation

Transgressive segregation refers to the appearance of plants in the F2 or later generations which are either more resistant or more susceptible than either of the original parents (Hooker 1967). Hooker (1967) noted that this phenomenom was not unusual with resistance to *P. sorghi* in maize. Wallwork and Johnson (1984) reported the occurrence of transgressive segregation for resistance to *P. striiformis* in F2 and F3 progeny from crosses involving moderately susceptible wheat cultivars. These F2 and F3 transgressive segregants were more resistant than either parents. The resistance was shown to be due to the accumulation of recessive factors and effective against a broad spectrum of races. The results suggested that observed transgressive segregation originated from a combination of genetic components from both parents. Nilsson-Ehle (1911), Pesola (1927), Pope (1965) and Krupinsky and Sharp (1979) also

reported transgressive segregation for adult plant resistance to yellow rust of wheat.

Reversal of Dominance

A resistance gene may behave as dominant with one physiological race and reccessive with another (Knott and Anderson 1956), dominant in one genetic background and reccessive in another (Sunderman and Ausemus 1963) or, recessive at the seedling stage and dominant at the adult stage and vice-versa, (Athwal and Watson 1954).

MATERIALS AND METHODS

Seed of the 391 spring wheat introductions was obtained from Dr. T.E. Miller at the Plant Breeding Institute in Cambridge, England. The introductions are part of the A.E. Watkins collection. According to Dr. Miller, the collection, accumulated in the late 1920's and early 1930's, has undergone considerable regeneration and is now substantially reduced in size, mainly as a result of poor storage conditions during World War II. The Watkins collection consists mostly of landraces collected from many of the wheat growing regions of the world. The collection is classified according to country of origin (see PBI 1983).

Puccinia graminis tritici (Stem Rust): (Figure 1)

All 391 spring wheat introductions were tested (see Appendix A for inoculation method) in the field at the adult stage, to a bulk inoculum consisting of isolates prevalent in western Canada and, in the greenhouse at the seedling stage to race C17. The severity of the rust infection in the field was rated according to a visual scale developed by Peterson et al (1948) (Appendix B). The infection types (ITs) in the greenhouse were evaluated according to a scale developed by Stakman (1914) (Appendix C). For the sake of discussion, the

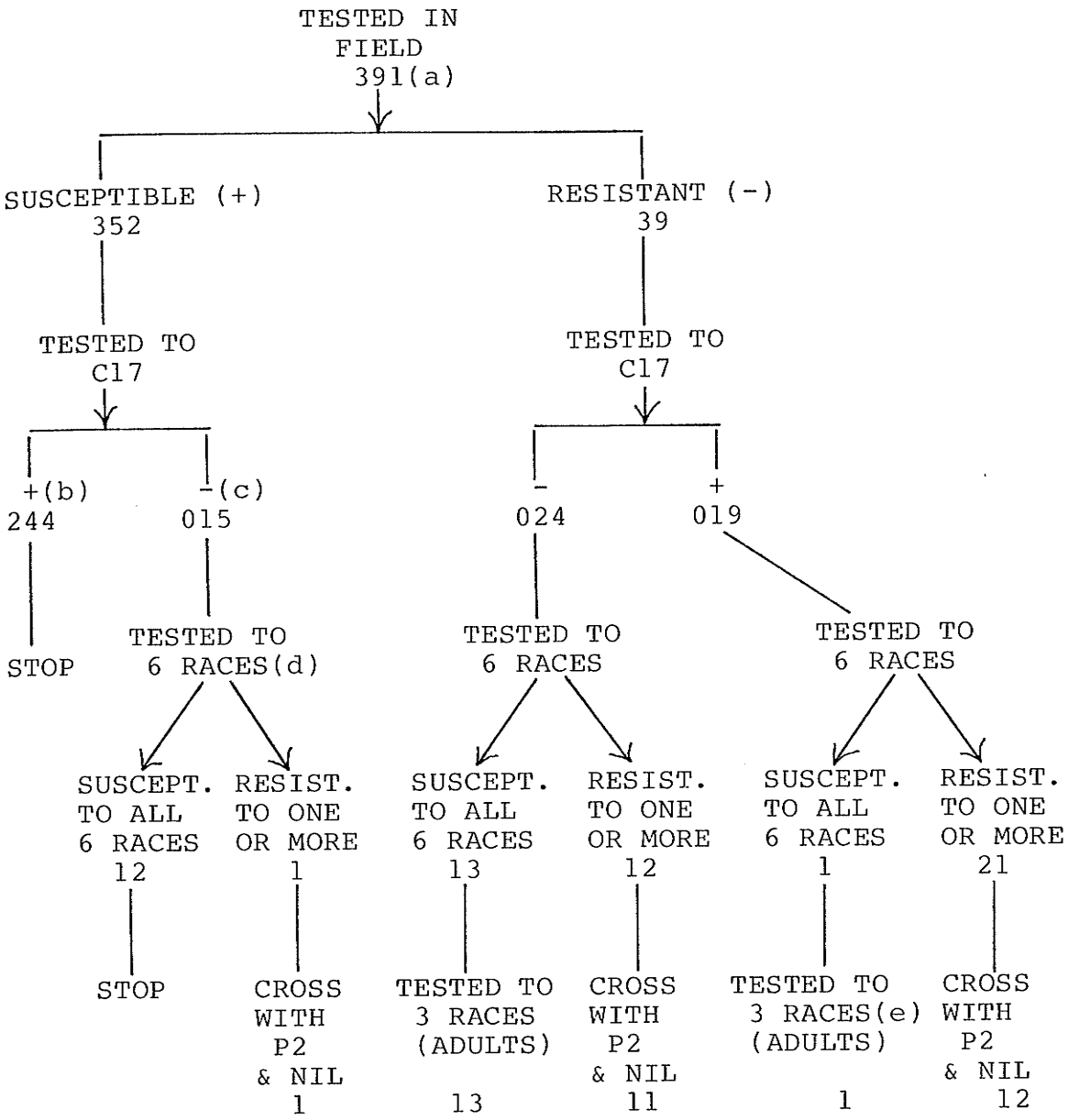


FIGURE 1. Flow-chart demonstrating the sequence of events during the screening of the 391 introductions for resistance to stem rust.

(a) number of introductions
(b) susceptible
(c) resistant
(d) C10, C20, C25, C49, C53, C57
(e) C10, C17, C53

terms 'infection type' and 'reaction' will be used synonymously through-out the text.

All introductions showing resistance to either or both the bulk inoculum and/or C17 were tested in the greenhouse at the seedling stage to a set of 6 races: C10, C20, C25, C49, C53 and C57. Only the introductions which showed resistance to at least one of these races was considered as resistant.

The patterns of infection types (IT) obtained from these tests allowed for the postulation of hypothetical Sr genotypes. This was done by comparing the IT patterns of the various introductions to those of the near isogenic lines (NILs) carrying single Sr genes. McVey and Roelfs (1973, 1975) postulated hypothetical genotypes for stem rust resistance in the entries of the 4th International Winter Wheat Nursery in essentially the same way. The resistant introductions were crossed with RL6071, a universal suscept, and with NILs carrying the Sr genes coresponding to their respective hypothetical genotypes. The crosses involving RL6071 will be referred to as 'susceptible by resistant' or 'SXR crosses' and those involving the NILs as 'resistant by resistant' or 'RXR crosses'.

Three to four F1 seeds from each of the SXR and RXR crosses were planted to produce F2 families. These F2 families were tested at the seedling stage to races known to be avirulent on the hypothetical Sr genotype. The F2 plants were then classified according to their ITs as either resistant or susceptible. The segregation for resistance and suscepti-

bility within the F2 families was noted and the occurrence of a particular theoretical segregation ratio was confirmed using a Chi-square test (Appendix F). Homogeneity of the F2 data obtained from different families of a cross was tested according to a method described by Mather (1957) (Appendix E). If the data proved to be homogeneous they were pooled to allow for larger F2 family sizes. Minimum F2 family sizes to allow for statistical inference were determined according to Hanson (1959) (Appendix D).

All introductions showing good resistance (50MS or better) to the bulk inoculum in the field at the adult stage and little or no resistance (3-4 ITs) to all of the six races used in the greenhouse at the seedling stage were said to possess 'adult plant' resistance. These introductions were tested at the adult stage in the greenhouse (Appendix A) to three races C10, C17 and C53.

Puccinia recondita tritici (Leaf Rust): (Figure 2)

A procedure similar to that of P. gramainis tritici was used. All introductions were tested in the field at the adult stage to a bulk inoculum consisting of isolates prevalent in western Canada. The introductions were then tested in the greenhouse at the seedling stage to race 1. Introductions resistant to race 1, regardless of their reactions in the field, were tested in the greenhouse at the seedling stage to a set of 6 isolates: race 5 (R5), race 9 (R9), race 15 (R15), isolate 1 (B1), isolate 4 (B4) and isolate 10 (B10).

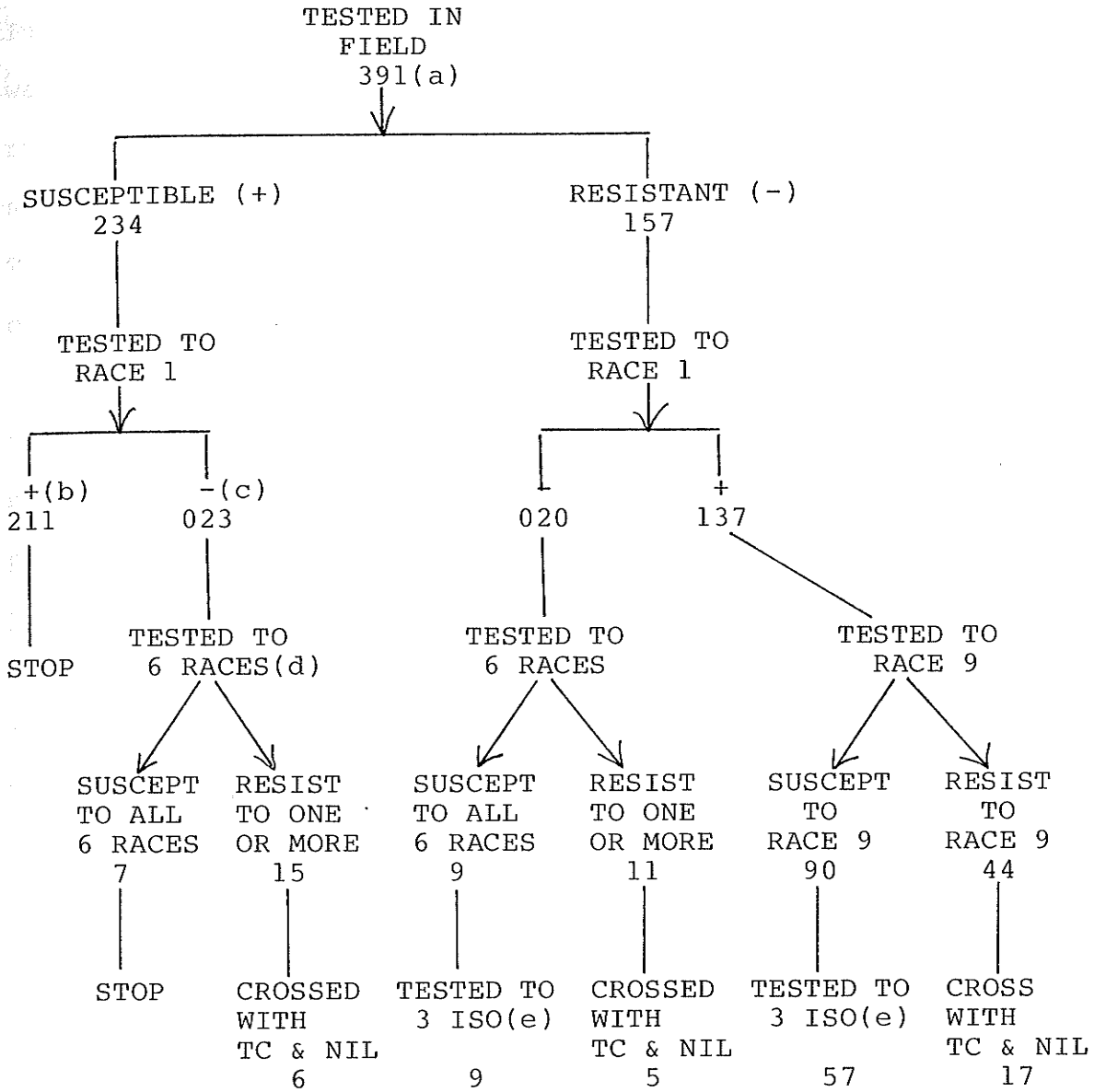


FIGURE 2. Flow-chart demonstrating the sequence of events during the screening of the 391 introductions for resistance to leaf rust.

(a) number of introductions
(b) susceptible
(c) resistant
(d) races 5, 9 & 15 and, bulks 1, 4 & 10
(e) isolates 114(76), B10(76) & 98(76)

Introductions resistant in the field but susceptible to R1 were tested solely to R9. Due to the large number of leaf rust resistant introductions, only part of these were selected to be studied genetically. Crosses were made with Thatcher (TC), a universal suscept, and with the corresponding Lr NILs.

Adult plant resistance was investigated using leaf rust isolates 114(76), B10(76) and 98(76) known to differentiate between genes Lr 12, 13, 22, T2 and T3 (RL6050) and the gene present in RL5711. Those introductions that were found to possibly carry Lr13 were tested at the seedling stage to isolate 32F derived from a cross between isolates 98 & 96. Pretorius et al (1984) reported that the resistance conferred by Lr13 could be expressed at the seedling stage thus eliminating the need for adult plant testing when studying this gene.

Ustilago tritici (Loose Smut)

All 391 spring wheat introductions were inoculated with a mixture of races T2 and T10. These races, in combination, carry the virulence genes Utv1, Utv2, Utv3, Utv4 and one unidentified gene (Dr. J. Neilson, personal communication; Neilson 1977, 1982). Those found to be immune or highly resistant to this mixture were then inoculated with a mixture of races T13 and T39 which have at least two additional unidentified virulence genes. The inoculations were made according to a method developed by Dr. Jens Neilson here at

the Agriculture Canada Research Station in Winnipeg, Manitoba. The method is based on that of Poelhman (1945) (Appendix G). Only introductions immune or highly resistant to all four races were reported.

Drechslera tritici-repentis (Tan Spot)

All 391 introductions were first tested to isolate CDA 1241 known to be prevalent in Manitoba (Dr. A. Tekauz, personal communication). Eight to 12 plants per introduction were tested using a 24 h incubation period and allowing ten days for symptom development. The inoculations for this first test were done using the 'Misting Method' (Appendix H). The introductions were scored according to their apparent degree of resistance (Appendix G).

All introductions immune after the first test were then tested to 6 isolates known to be prevalent in Manitoba (Dr A. Tekauz, personal communication), ie: CDA 1241, CDA 866, AT82-254-1, AT76-199-2, AT82-199-1, AT82-27-1. The inoculation method used for this second test differed from that of the first test (see Appendix J; the 'Dipping Method'). A 30 h incubation period was used followed by the usual 10 days to allow for symptom development. The introductions were put into ten arbitrary phenotypic groups according to the similarities in their reactions to the 6 isolates (Appendix K).

RESULTS

Stem Rust

The seedling screening results are presented in TABLE 1. Of the 391 introductions tested, only those resistant to one or more of the 7 races used are listed. These resistant introductions were grouped according to patterns of infection types corresponding to those of Sr NILs. The Sr29 and Sr30 groups were considered as one because Green (1981) reported that both genes apparently conferred resistance to all 7 races used. Actually, in the present study, C20 was found to be virulent on Sr30 and not Sr29, and could have served to differentiate between the two. Seventeen introductions were classified as 'unidentified'. Their IT patterns did not correspond to that of any of the various Sr NILs.

The F2 segregation ratios obtained from the various crosses involving the selected resistant introductions, RL6071 and their respective NILs are presented in TABLE 2. The data in TABLE 2 are arranged according to Sr groups as is TABLE 1. Tests for homogeneity of data in all instances suggested homogeneity. Thus, the heterogeneity P-values were omitted from TABLE 2.

Unfortunately, no infection was obtained when the introductions believed to possess adult plant resistance were

TABLE 1

Seedling infection types and adult plant reaction of stem rust resistant spring wheat introductions and near isogenic lines (NILs) to seven races and a bulk inoculum of *Puccinia graminis tritici*.

	races(a)							bulk(b)
INTRO	C10	C17	C20	C25	C49	C53	C57	FIELD
Sr7b group								
V499	2+	;/4	4	1+	2+	4	4	20MS
Sr13 group								
V289	3	2+	2+	4	2+	2+	3+	40MS
Sr15 group								
V578	3	2+	3+	4	2+	1+	4	40S
Sr17 group								
V275	2+	2+	4	3	2	3	3+	40MS
V285	3	3+	4	3+	2	2+	3+	40MS
V612A	2+/3	2+nc	4	3/4	2/2+	3	3+	10MR
Sr29, 30, group								
V226	2	2	4	1+	2+	1+	1+	
V314	2	;1+	3+	1+	1+	1+	1	10R
V320	2/2+	;1-	3+	1+	1+	1+	1+	50MR
V524	2+	2+	4	2/2+	2/2++	2+	2+	30MS
V519	2	;2	;/3	1+	2	1+	1+	20MR
V609	2+	2+/3	3+	2+	2+/3	2+	2+	30MR
V611	2+	2+/3	3+	2+	2+/3	2+	1+	10MR
V614	2+	2+	3+	2+	3	2+/3	2/2+	10MR
V619	-	;1/3	4	2+/3	2+	3	2+/3	20MR
Unidentified group								
V57	4	;3/4	2+	3/3+	4	4	1/3	5M-70S
V276	3	2+	4	3	3	2+	3+	
V278	2/3+	2+	3-/3+	3+	3-	2+/3	3+	
V310	2+/4	2+	3+	3	2	2/4	3+	60S
V317	3+	2+	4	2/2	2+/3+	2+	2	50S

(a) infection types (seedlings; Appendix C)
(b) field reactions (adults; Appendix B)

TABLE 1 (cont'd)

	races(a)							bulk(b)
INTRO	C10	C17	C20	C25	C49	C53	C57	FIELD
Unidentified group (cont'd)								
V322	2+	2	3+	2/3	3/3+	2+	2+	
V324	2+/3	2+	4	2	2+	2	1+	50MS
V326	3+	4	3+	3+	2/4	1+/4	;1/3	40MR
V327	4	3	4	1	2/3	1/3	;1/3	50MS
V418	2+	2+	3+	2+	3	2+	2+	30MS
V430	2/4	;1+/3+	2/3+	2/3+	4	3+	3+	70S
V455	2	2	3c/4	2+/3+	2+	4	4	20MS
V583	2+	2+/3	0	2+	2+	3	3	40MR
V594	2/4	2/2	3+	3+	2/4	2	4	20MS
V601	2	2/2+	3+	4	2/4	2	3+	30MS
V627	2+/4	;/3	4	-	4	2	2	70S
V642	2+	;1/3	4	3+	2+	2	3	50S
Near Isogenic Line Group								
Sr7b(NIL)+	+	+	-	+	+	+	70MS	
Sr13 "	+	-	-	+	-	-	+	60MS
Sr15 "	+	+	+	+	-	-	+	70S
Sr17 "	+	+	+	+	-	+	+	30S
Sr29 "	-	-	-	-	-	-	-	60S
Sr30 "	-	-	+	-	-	-	-	40MR
RL6071	+	+	+	+	+	+	+	70S

(a) infection types (seedlings; Appendix C)
(b) field reactions (adults; Appendix B)

inoculated with the 3 stem rust isolates. The experiment was discarded.

In the result section to follow, the segregation ratios mentioned will always refer to the number of resistant and susceptible plants respectively, eg: a 3:1 ratio implies 3 resistant plants for every susceptible plant observed.

Sr7b group

The F2 of cross V499 X RL6071 when tested with C25 segregated for reaction according to a 3:1 ratio indicating that V499 carried a dominant gene for resistance. Although this gene was thought to have been Sr7b, this hypothesis was proven false when susceptible plants were observed in the F2 of the cross V499 X Sr7b tested with C25.

Sr13 group

When tested with C17, the F2 of the cross V289 X RL6071 segregated for reaction according to a 1:3 F2 ratio suggesting that V289 may be carrying a reccessive gene. The presence of such a gene was confirmed when V289 was crossed to NIL Sr13 and no F2 plants susceptible to C17 were found.

Sr15 group

F2 plants derived from the cross between RL6071 and V578 segregated for reaction to C49 according to a 1:3 ratio indicating the presence of a single recessive gene in V578. When V578 was crossed with NIL Sr15, no F2 plants susceptible to C49 could be found. Thus, the sole introduction in this group, V578, was shown to carry a recessive gene, Sr15.

Sr17 group

All three intoductions, V275, V285 and V612A, were crossed to RL6071. The F2 populations derived from these crosses segregated for reaction to C49 according to 1:3 ratios. These ratios suggested that V289, V285 and V612A may be carrying single recessive genes. Plants susceptible to C49 were later found in all three F2 populations derived from crosses involving the above introductions and NIL Sr17. Thus, none of the introductions thought to carry Sr17 were shown to do so.

Sr29, Sr30 group

When introductions V314 and V320 were crossed with RL6071 the resulting F2 populations segregated for reaction to C17 according to 3:1 ratios. These ratios indicated that V314 and V320 may be carrying single dominant genes. The F2 populations of crosses involving introductions V524, V609, V611 and V619 with RL6071 segregated for reaction to C17 according to 1:3 ratios indicating that these introductions may be carrying single recessive genes. When V614 was crossed to RL6071, the resulting F2 population gave the best fit to a 9:7 segregation ratio, indicating the presence of 2 complementary genes. However, a partially dominant gene may have been involved. No seed set was obtained when V226 was crossed to RL6071.

All of the crosses involving the resistant introductions and NIL Sr30 gave rise to F2 populations free of plants susceptible to C17. In crosses with NIL Sr29, plants

TABLE 2

Segregation for reaction to races of *Puccinia graminis tritici* in F2 populations from crosses between susceptible and resistant parents and between resistant parents and near isogenic lines (NILs) with identified *Sr* genes

Cross	Race	Number of Plants Res.	Susc.	Expected Ratio	P-value
Sr7b group					
US(a)X V499	C25	218	60	3:1	.50-.25
V499 X Sr7b	C25	236	64	13:3	.50-.25
Sr13 group					
US X V289	C17	50	166	1:3	.90-.75
V289 X Sr13	C17	317	0	13:3	<.005
Sr15 group					
US X V578	C49	70	202	1:3	.90-.75
V578 X Sr15	C49	400	0	13:3	<.005
Sr17 group					
US X V275	C49	46	130	1:3	.90-.75
V275 X sr17	C49	172	258	7:9(b)	.50-.25
US X V285	C49	57	167	1:3	.95-.90
V285 X sr17	C49	163	195	7:9(b)	.75-.50
US X V612A	C49	61	175	1:3	.90-.75
V612A X sr17	C49	149	196	7:9(b)	.90-.75
Sr29-Sr30 group					
US X V226	C17	------ no seed ----------			
V226 X Sr29	C17	208	35	13:3	.25-.10
V226 X Sr30	C17	313	0	15:1	<.005
US X V314	C17	99	24	3:1	.25-.10
V314 X Sr29	C17	160	33	13:3	.75-.50
V314 x Sr30	C17	336	0	15:1	<.005
US X V320	C17	134	30	3:1	.10-.05
V320 X Sr29	C17	190	15	15:1	.75-.50
V320 X Sr30	C17	284	0	15:1	<.005
US X V519	C17	31	115	1:3	.75-.50
V519 X Sr29	C17	132	32	13:3	.90-.75
V519 x Sr30	C17	326	0	13:3	<.005
US X V524	C17	25	78	1:3	.95-.90
V524 X Sr30	C17	282	0	13:3	<.005

TABLE 2 (cont'd)

Cross	Race	Number of Plants Res.	Number of Plants Susc.	Expected Ratio	P-value
Sr29-Sr30 group (cont'd)					
US X V609	C17	40	130	1:3	.90-.75
V609 X Sr29	C17	118	40	13:3	.10-.05
V609 X Sr30	C17	212	0	13:3	<.005
US X V611	C17	41	121	1:3	.95-.90
V611 X Sr29	C17	123	43	13:3	.05-.025
V611 X Sr30	C17	319	0	13:3	<.005
US X V614	C17	103	65	9:7(c)	.50-.25
V614 X Sr30	C17	280	0	57:7(c)	<.005
US X V619	C17	24	117	1:3	.50-.25
V619 X Sr29	C17	150	36	13:3	.90-.75
V619 x Sr30	C17	297	0	13:3	<.005

(a) universal suscept (US); RL6071
(b) suggest the presence of two reccessive genes, one of which being the unidentified gene in introductions V275, V285 and V612A and the other, Sr17, known to be recessive
(c): suggest the possible presence of two reccessive genes, two complementary genes, linkage and/or partial dominance
: the F2 population size was not adequate to allow testing for the actual occurrence of the ratios

susceptible to C17 were invariably found in all F2 populations. Thus, all the introductions in the group carry Sr30 and not Sr29.

Leaf Rust

The screening results are presented in TABLE 3. Of the 391 introductions tested, only those resistant to at least one of the 7 differential races are included. The introductions are grouped according to similar phenotypic responses and hypothetical Lr genotypes. Those found to be susceptible to race 1, resistant to race 9 and resistant in the field were included in the RL6050 group. RL6050, or one of the genes present in RL6050, frequently gives a susceptible response to race 1 but still provides some resistance in the field. The NIL Lr11, RL6057, Lr21, Lr30 groups were combined because of their similar reactions to the races used. Three introductions were classified as 'unidentified' since the IT patterns of these introductions did not correspond to that of the NILs. Only the introductions marked with a '*' were crossed with TC and a corresponding NIL.

Introductions showing little or no resistance at the seedling stage (ie: 3-4 IT) but adequate resistance in the field (ie: 30MS or better) are listed in TABLE 4. The presence of Lr12 in V53, V289, V448, V459 and V641, Lr13 in V55, V464, V571 and V633 and Lr22 or the gene in RL5711 in V267, V584, V587, V634 and V638 could be hypothesized. All

TABLE 3

Seedling infection types and adult reaction of leaf rust resistant spring wheat introductions and Lr near isogenic lines (NILs) to races and a bulk inoculum of Puccinia recondita

	races(a)							bulk(b)
INTRO	R1	R5	R9	R15	B1	B4	B10	FIELD
RL6050 group								
V4	4		2					50MS
V9 *	3+		1					10MR
V51	4		2/2+					50MS
V53	4		2/2+					10MR
V55	4		2					10MS
V60	4		2					20MS
V61	4		2+					20MS
V63 *	4		;1+					10MR
V66	3+		1+/2+					TR
V72 *	3+		1+					10MR
V104	4		1+					50MS
V111*	3+		1+					40MR
V112*	4		1					10MR
V113*	3+		1+					20MS
V152*	4		2					10MR
V153	4		2/2+					20MS
V197	3+		2/2+					10MS
V201	3+		2					30MS
V214	3+		1+					50MS
V230	3		1+/2+					50MS
V237*	3+		;1					20MS
V241	4		;1					50MS
V278*$	4		1/1+					TR
V279*	3+		;1					5MR
V285 $	4		1					40MS
V291*	4		1+					TMR
V321*	3+		2					10MR
V336*			;					10MR
V499 $			;					10MS
V525	4		1+					30MS
V552	S		1+					15MS
V578 $	3		2+					50MS
V580	4		2c					10MS
V581			2+					30MS

(a) infection types (seedlings; Appendix C)
(b) field reactions (adults; Appendix B)

TABLE 3 (cont'd)

	race(a)							bulk(b)
INTRO	R1	R5	R9	R15	B1	B4	B10	FIELD
RL6050 group (cont'd)								
V600	3		;1-					50MS
V618*			;					5R
V628*			;					10MS
V634	4		2+					5MS
V635	4		;1+/2					15MS
V637*	3		1					5MS
V641	S		2+					30MR
V642*$	4		1+					30MR
V643	4		2+					30MR
V645	4		2					30MS
Lr10 group								
V6	2+-3	3+	3+	4	4	3+	-	60S
V7	;1+-3+	3	3+	3+	3+	3+	;1/4	80S
V27	;1	4	2+/3	4	3+	4	;1+	60S
V31	;1--4	4	3+	-	4	4	;1+/3+	60S
V34 *	;1+	4	3+	4	3+	3+	2	70S
V67	;1	4	4	4	4	3+	2+	40S
V68	;1-4	4	2+/3+	4	4	4	;1/3+	30MS
V127	;1	4	3+	4	4	4	2+	20MS
V146	1	4	3+	4	4	4	;1/3+	10MS
V154*	;1	4	2/2+	4	4	4	1+	60S
V156 #	1+	4	2+	4	4	4	2+	50MS
V158	;1-	4	3+	4	4	4	2/2+	50S
V160*	;1-	4	3+	4	4	4	2	70S
V187*	;1	4	3+	4	4	4	2	5MR
V267	;1-	;1/4	1	3+	3+	3+	;1	TMR/70S
V298*	;1-	4	3+	4	4	4	;1+	20MR
V418*$	R	4	3+	4	4	4	;1+	70S
V541	;1	4	3+	4	4	4	2+	60MS
V623*	;1	4	3	4	4	4	;1+	60MS
Lr11, RL6057, Lr21, Lr30 group								
V407*	;1-	2	;1	;1+	;1+	2-3+	;1+	20MR
Lr16 group								
V410*	2	-	;1-	3+	;1+	2+-3	;1+	30MS

(a) infection types (seedlings; Appendix C)
(b) field reactions (adult; Appendix B)

TABLE 3 (cont'd)

	races(a)							bulk(b)
INTRO	R1	R5	R9	R15	B1	B4	B10	FIELD
Lr3 group								
V10 *	;-3+	4	;1/3+	4	3+		;1/4	70S
RL6061 group								
V503*	R	2/3+	1	4	3+	3+	2	20MR
Unidentified group								
V411	-	4	3+	2+	4	4	4	70S
V540	;-4	4	2+	;1++	4	4	3+	TMR
V562	;1+	4	3+	-	2+	-	-	40S
Near Isogenic Line (NIL) group								
Lr3(NIL)	;1-	4	;1-	4	4	;1	4	70S
Lr10 "	;1-	4	4	4	4	4	;1	70S
Lr11 "	1+	1+	1+	1+	1+	1+	1+	60MS
Lr16 "	1	1+	1	4	1+	2+	1+	15MR
Lr21 "	;1	;1	;1-	;1	1+	;1+	;1+	TMR
Lr30 "	1+	1+	;1	1+	4	1+	1+	3MR
LrB "	2+	4	2+	4	4	4	2	60MS
RL6057	1+	1+	1+	1+	1+	2++	1+	10MR
RL6061	2	2	2+	4	4	4	2	60MS
RL6050	4	-	2	-	-	-	-	3MR
Thachter	4	4	4	4	4	4	4	70S

* crossed to TC and NIL
$ also resistant to stem rust
# also resistant to loose smut
(a) infection types (Appendix C)
(b) field reactions (adults; Appendix B)

TABLE 4

Adult plant infection types of 29 introductions of spring wheat to three isolates of Puccinia recondita, their reactions in the field to a bulk inoculum and their hypothetical Lr genotypes.

Introduction	Isol(a)	Iso2(b)	Iso3(c)	Field 1983	Field 1984	Hypoth. Genotype
V40	3+	1+	3+	15MS	60MS	??
V48	3+	1+-3+	4	30MR	10MS	??
V53	;	;-3+	4	10MR	5MR	Lr12
V55	4	3+	1+-3+	10MS	10MR	Lr13
V66	3	2	3+	TR	TMR	??
V68	4	1+	3+	30MS	10MR	??
V127	4	;1	4	20MS	TMR	??
V146	4	;	;1-3	10MS	--	??
V154	3	;-1+	;1-3	30MR	5MR	??
V165	2	3	2+	10MR	TMR	??
V187	Z	;	3+	5MR	TMR	??
V250	4	1+-4	3+	10MR	10MR	??
V267	;1c	;1c	;1+	TMR-70S	TMR	Lr22/RL5711
V289	;-;1	;	3+	20MR	20MR	Lr12
V298	3+-4	;1-	3+-4	20MR	5MR	??
V443	3	;-2	3	5MR	TR-5MR	??
V448	2-2+	2+	4	10MS	TMR	Lr12
V459	;1-1+	;1-3+	3-3+	TR-70S	10MR	Lr12
V464	4	Z	1-2	10MR	30MR	Lr13
V563	3+	;-4	;-3	5MR	TR	??
V571	4	X-4	;1-4	5MR	TR	Lr13
V580	2-4	;1+-3	;1-3+	10MS	30MS	??
V584	;1-3+	2-4	;-4	10MR	TR-40MR	Lr22/RL5711
V587	;1+	2+-3	2+-3	TR	TR-50MR	Lr22/RL5711
V601	;1-3+	2+-3+	3+	10MS	----	??
V633	4	4	2	10MR	50MS	Lr13
V634	2-;1	1	1+-2	5MS	TMR	Lr22/RL5711
V638	;1-1	;1	;1-1	TMR	TMR	Lr22/RL5711
V641	;1+	;	3+	30MR	20MR	Lr12
Lr12	;1	;1	4	----	5MR	
Lr13	4	4	;1+	----	20MS	
Lr22	1+	1+	1	----	TMR	
RL5711	1+	2	1+	----	TMR	

(a) Isol => isolate 114(76)
(b) Iso2 => isolate B10(76)
(c) Iso3 => isolate 98(76)

TABLE 5

Segregation for reaction to avirulent races of *Puccinia recondita* in F2 seedling populations derived from crosses between susceptible and resistant *Triticum aestivum* parents and between resistant parents and *Lr* NILs

Cross	Race	Number of Plants Res.	Susc.	Expected Ratio	P-value
RL6050 group					
TC(a)X V9	9	153	50	3:1	.95-.90
V9 X RL6050	9	284	18	15:1	.90-.75
TC X V63	9	24	78	1:3	.90-.75
V63 X RL6050	9	291	21	15:1	.75-.50
TC X V72	9	72	25	3:1	.95-.90
V72 X RL6050	9	184	14	15:1	.75-.50
TC X V111	9	113	70	9:7(b)	.50-.25
V111 X RL6050	9	----- no seed -----			
TC X V112	9	40	122	1:3	.975-.95
V112 X RL6050	9	235	42	13:3	.25-.10
TC X V113	9	42	140	1:3	.90-.75
V113 X RL6050	9	246	37	13:3	.05-.025
TC X V152	9	121	57	3:1	.10-.05
V152 X RL6050	9	279	15	15:1	.50-.25
TC X V237	9	132	70	3:1	<.005
V237 X RL6050	9	176	13	15:1	.75-.50
TC X V278	9	122	48	3:1	.50-.25
V278 X RL6050	9	261	19	15:1	.50-.25
TC X V279	9	117	47	3:1	.50-.25
V279 X RL6050	9	195	20	15:1	.10-.05
TC X V291	9	49	156	1:3	.90-.75
V291 X RL6050	9	243	58	13:3	.90-.75
TC X V321	9	123	55	3:1	.25-.10
V321 X RL6050	9	167	3	63:1	.75-.50
TC X V336	9	216	12	15:1	.75-.50
V336 X RL6050	9	299	4	255:1(c)	.90-.75
TC X V618	9	192	8	15:1	.25-.10
V618 X RL6050	9	326	3	255:1(c)	.95-.90
TC X V628	9	194	7	15:1	.25-.10
V628 X RL6050	9	187	7	255:1(c)	.95-.90
TC X V637	9	151	50	3:1	.975-.95
V637 X RL6050	9	297	16	15:1	.50-.25
TC X V642	9	---- self ----			
V642 X RL6050	9	283	42	13:3	.025-.01

(a) Thatcher, a universal suscept
(b) suggest the presence of either two complementary genes, two reccessive genes, partial dominance or likage
(c) suggest the presence of 4 dominant genes, two of which could be the two unidentified dominant genes in introductions V336, V618 and V628

TABLE 5 (cont'd)

Cross	Race	Number of Plants Res.	Susc.	Expected Ratio	P-value
Lr10 group					
TC X V34	1	119	42	3:1	.90-.75
V34 X Lr10	1	294	0	15:1	<.005
TC X V154	1	130	53	3:1	.50-.25
V154 X Lr10	1	316	0	15:1	<.005
TC X V160	1	160	37	3:1	.10-.05
V160 X Lr10	1	330	0	15:1	<.005
TC X V187	1	131	54	3:1	.50-.25
V187 X Lr10	1	291	0	15:1	<.005
TC X V298	1	133	35	3:1	.75-.50
V298 X Lr10	1	311	0	15:1	<.005
TC X V418	1	157	54	3:1	.90-.75
V418 X Lr10	1	340	0	15:1	<.005
TC X V623	1	146	42	3:1	.50-.25
V623 X Lr10	1	349	0	15:1	<.005
Lr11, RL6057, Lr21, Lr30 group					
TC X V407	1	159	50	3:1	.90-.75
V407 X Lr11	1	273	25	15:1	.25-.10
V407 X RL6057	1	331	0	15:1	<.005
V407 X Lr21	1	42	0	15:1	<.005
V407 X Lr30	1	281	45	13:3	.05-.025
Lr16 group					
TC X V410	1	150	51	3:1	.95-.90
V410 X Lr16	1	308	18	15:1	.75-.50
Lr3 group					
TC X V10	1	---- self ----------			
V10 X LrB	1	164	38	13:3	.99-.975
RL6061 group					
TC X V503	1	147	36	3:1	.25-.10
V503 X RL6061	1	317	0	15:1	<.005

four introductions believed to carry Lr13 (ie: V55, V464, V571 and V633) were susceptible to isolate 32F.

The segregation for reaction to leaf rust races 1 and 9 of F2 populations derived from crosses between selected resistant introductions and TC (SXR crosses) and between resistant introductions and their corresponding NILs (RXR crosses) is shown in TABLE 5. As with the stem rust data, tests for homogeneity of data were in all instances positive. The P-values associated with these tests were omitted from TABLE 5.

RL6050 group

The F2 of crosses involving V9, V72, V152, V237, V278, V279, V321 and V637 with TC segregated for reaction to race 9 according to 3:1 ratios indicating the presence of single dominant genes. The F2 from crosses involving V63, V112, V113 and V291 with TC segregated for reaction according to 1:3 ratios which suggested the presence of single recessive genes. The F2 of crosses involving V336, V618 and V628 with TC segregated according to 15:1 ratios indicating the presence of 2 dominant genes. Finally, the F2 of cross V111 with TC segregated according to a 9:7 ratio indicating the presence of two complementary dominant genes.

All the crosses involving the introductions in this group and RL6050, gave F2 populations with plants susceptible to race 9. Thus, on the basis of these seedling tests none of these introductions can be said to carry the RL6050 type resistance as was originally hypothesized.

Lr10 group

The segregation for reaction to race 1 of the F2 from crosses involving introductions V34, V154, V160, V187, V418 and V623 with TC fitted 3:1 ratios suggesting that these introductions carry single dominant genes. All F2 populations derived from crosses involving these introductions with NIL Lr10 were free of plants susceptible to race 1. Thus, all the introductions in this group have Lr10.

Lr11, RL6057, Lr21, Lr30 group

The F2 population derived from the cross between V407 and TC segregated for reaction to race 1 according to a 3:1 ratio indicating the presence of a single dominant gene in V407. The F2 progenies derived from crosses involving V407 and NIL Lr11 and NIL Lr30 segregated for reaction to race 1 suggesting that these two genes were not present in V407. On the other hand, the F2 progenies derived from crosses involving V407 with RL6057 and NIL Lr21 were free of plants susceptible to race 1. Since gene Lr21 is of interspecific origin (Rowland of Kerber 1974) and the F2 of V407 X Lr21 had only 42 plants it is unlikely that V407 has gene Lr21. V407 must carry the gene in RL6057.

Lr16 group

The sole introduction in this group, V410, when crossed to TC, produced an F2 population which segregated for reaction to race 1 according to a 3:1 ratio. Since plants susceptible to race 1 were found in the F2 populations derived from a cross between V410 and NIL Lr16, V410 does not carry Lr16.

Lr3 group

V10 does not carry Lr3 since F2 plants susceptible to race 1 were observed amongst the progeny derived from the cross V10 X NIL Lr3. The cross TC X V10 was selfed.

RL6061 group

The cross TC by V503 yielded an F2 populations which segregated for reaction to race 1 according to a 3:1 ratio suggesting the presence of a dominant gene in V503. When V503 was later crossed to RL6061, no F2 plant susceptible to race 1 were observed. This indicated that the gene in RL6061 is also carried by V503.

Smut

Of the 391 introductions inoculated with a mixture of races T2 and T10, only 61 were found to be immune or highly resistant. Of these, only 5 were immune and 6 highly resistant to a mixture of races T13 and T39. These 11 introductions are reported in TABLE 6. Immune introductions showed 0% infection and highly resistant introductions showed less that 5% infection (ie: 1 plant out of 20). The remaining introductions showed more than 5% infection and were considered as susceptible.

TABLE 6

Reaction of immune and highly resistant spring wheat introductions to two mixtures of Ustilago tritici (loose smut): T2/T10 and T13/T39.

Introduction	race mixture T2/T10 (+:-)(a)	race mixture T13/T39 (+:-)	IM/HR(b)
V21	0:8	0:13	IM
V156	0:12	0:14	IM
V483	0:20	0:15	IM
V523	0:12	0:15	IM
V649	0:24	0:15	IM
V20	1:30	2:10	HR
V52	0:6	1:20	HR
V602	0:14	1:20	HR
V609	1:12	1:10	HR
V627	0:20	2:10	HR
V635	0:11	2:10	HR

(a) + => susceptible/ - => resistant
(b) IM => immune/ HR => highly resistant

Tan Spot

Of the 391 introductions initially tested for resistance to isolate CDA 1241, 69 were considered as resistant. The reactions of these 69 resistant introductions to 4 isolates of tan spot prevalent in Manitoba are listed in TABLE 7. Isolates 3 and 6, despite their ability to grow profusely on artificial medium failed to infect any of the 69 introductions and were omitted from TABLE 7.

The ´Dipping Method´ (Appendix I) allowed for much more severe symptom development than the ´Misting Method´ (Appendix H). Apart from the fact that the inoculum suspension may have caused some leaf damage, possibly through the clogging of stomata, the ´Dipping Method´ is favored because of its higher culling rate.

Symptoms on the first, second and third leaves differed from those on the fourth and fifth leaves. The symptoms on the older leaves (1-3) were generally heavier. However, plants showing resistance on any of the five leaves were considered resistant. Resistant plants developed dark brown or grey flecks surrounded by chlorotic bands similar to those reported by Lee and Gough (1984). Lesions on susceptible plants were spreading and coalesced into chlorotic-necrotic spots. The leaves were also subject to ´tip-necrosis´ (Lee and Gough 1984) which often extended to most of the leaf surface. This necrosis appeared to be a form of mechanical damage due to the inoculums´ suspension medium rather than pathogenic in nature.

TABLE 7

Disease reactions(a) of 69 spring wheat introductions to 4 isolates of Drechslera tritici-repentis (tan spot) prevalent in Manitoba inoculated using the 'Dipping Method' and their designated phenotypic groups(b)

		isolates			
Intro	Phenotype	Isol(c)	Iso2(d)	Iso4(e)	Iso5(f)
V627	1	5/7	4/9	5/9	5/8
V557	2	4/9	3/7	5/9	6/9
V637	2	5/7	4/9	4/7	7/8
V648	2	-/4	-/-	5/9	8/9
V523	3	-/9	5/5	2/9	4/7
V525	3	7/7	3/9	3/9	5/9
V72	4	5/7	4/7	7/7	7/9
V163	4	5/7	2/7	9/9	7/9
V216	4	4/7	3/7	6/6	9/9
V217	4	4/7	3/7	6/7	9/9
V291	4	5/7	3/9	7/9	6/8
V317	4	4/7	-/5	6/7	8/9
V426	4	4/9	4/7	-/9	9/9
V579	4	-/5	2/7	-/7	9/9
V562	4	-/5	3/7	-/7	9/9
V479	5	-/9	-/7	4/7	3/7
PLT2	5	9/9	7/7	5/9	5/9
V63	6	-/9	4/7	5/7	7/9
V87	6	-/7	4/7	5/7	6/8
V112	6	-/9	4/7	5/7	7/9
V552	6	9/9	5/5	5/9	7/9
V640	6	-/7	1/9	5/9	7/9
V52	7	-/5	-/7	9/9	7/9
V126	7	-/5	7/7	7/9	9/9
V635	7	4/7	-/7	7/9	7/8
V639	7	4/7	-/5	7/9	8/9
V12	8	9/9	3/3	7/9	7/9
V13	8	7/7	4/4	7/9	6/9
V20	8	9/9	-/5	6/9	7/9
V50	8	-/9	4/4	6/9	7/9
V81	8	7/7	4/7	-/7	7/9
V136	8	-/7	4/7	7/9	9/9
V184	8	7/7	1/7	7/9	7/9
V188	8	-/7	3/7	6/7	7/9
V189	8	7/7	3/7	6/8	7/9

TABLE 7 (cont'd)

		isolates			
Intro	Phenotype	Isol(c)	Iso2(d)	Iso4(e)	Iso5(f)
V192	8	-/9	4/7	9/9	9/9
V219	8	-/9	1/7	7/9	7/9
V267	8	-/9	3/9	7/9	9/9
V275	8	9/9	5/9	9/9	-/9
V278	8	7/7	3/7	9/9	7/9
V301	8	7/7	3/9	6/7	6/7
V342	8	-/9	-/5	7/7	7/9
V371	8	-/9	2/7	7/7	7/9
V399	8	-/9	-/4	6/9	7/9
V407	8	-/9	-/4	8/8	9/9
V438	8	-/9	-/5	-/7	6/7
V472	8	-/9	-/5	-/7	7/9
V487	8	-/7	5/9	7/9	6/9
V494	8	-/9	5/7	6/9	6/9
V507	8	7/7	-/5	-/6	7/9
V532	8	9/9	4/7	7/9	9/9
V534	8	9/9	5/5	7/9	7/9
V551	8	-/7	2/7	7/9	7/9
V593	8	-/7	5/5	7/9	7/9
V605	8	-/7	-/5	7/9	7/9
V620	8	-/7	4/9	7/9	7/9
V631	8	-/7	4/9	-/9	7/8
V165	8	-/7	7/7	5/7	9/9
V177	9	7/7	7/7	5/7	6/8
V309	9	-/9	-/7	5/7	7/9
V473	9	7/9	-/7	5/7	-/-
V483	9	7/7	6/6	5/7	7/9
V604	9	7/7	-/7	4/7	7/9
V157	10	-/7	7/7	9/9	5/7
V227	10	-/9	6/7	9/9	4/7
V232	10	-/9	6/7	-/9	5/7
V354B	10	-/9	6/6	-/8	3/7
V443	10	-/-	-/-	-/-	4/7
V619	10	-/9	-/9	7/9	4/7

(a) see Appendix K for evaluation procedure
(b) see Appendix K for description of classes
(c) CDA 1241
(d) CDA 866
(e) AT76-199-2
(f) AT82-27-1

DISCUSSION

Rusts

Seedling Tests.

The individual races used in these tests were chosen mainly because of their ability to differentiate between a number of Sr and Lr genes. On the other hand, the *P. graminis tritici* and *P. recondita* races present in the bulk inoculum are meant to represent races prevalent in Canada (Dr. D.J. Samborski, personal communication). Although resistance to individual races can be of academic interest, further genetic analyses should be mainly directed towards introductions possessing good field resistance to the bulk inoculum.

Some of the limiting factors in the present study were;

(1) the difficulty in classifying seedlings and adult plants as either resistant or susceptible
(2) the presence of partial dominance which led to the frequent misclassification of the heterozygotes

The classification of infected plants into discrete resistant and susceptible groups according to their ITs and field reactions can be fairly subjective, especially if the level of resistance is not exceptionally high as was the case

with most of stem rust resistant introductions. The partial dominance of a gene for rust resistance could lead to F2 segregation ratios which deviate significantly from those expected when fully dominant or fully recessive genes are involved. The presence of partial dominance will increase the frequency of intermediate types which, due to the subjectivity of the scoring scheme, can frequently be misclassified and thus lead to erroneous F2 segregation ratio.

Introductions shown to carry identified resistance genes have been listed in TABLE 8. The remainder of the resistant introductions studied were shown not to carry the proposed resistance genes and are listed in TABLE 9. The rust resistance of these introductions must be due to either other identified genes or new as-of-yet unidentified genes. These introductions need to be retested to a broader spectrum of races and further genetic studies conducted.

On the basis of their rust reactions, it was originally believed that the introductions listed in TABLE 8 carried single resistance genes. In the case of V407, four distinct hypothetical genotypes had to be proposed. These four genes, that is Lr11, Lr21, Lr30 and RL6057, produce similar IT patterns to the races used. Of these, Lr30 and Lr11 were shown not to be present in V407. Either of the remaining two genotypes, Lr21 or the gene present in RL6057, could be present. To determine which of the two is present, larger F2

TABLE 8

Spring wheat introductions carrying known genes for resistance to either stem rust (*Sr* genes) or leaf rust (*Lr* genes)

Introduction	Resistance Gene	Origin
V226	Sr30	Egypt
V289	Sr13	Canary Islands
V314	Sr30	Crete
V320	Sr30	Turkey
V519	Sr30	Poland
V524	Sr30	Sardinia
V578	Sr15	Tunis
V609	Sr30	Greece
V611	Sr30	Greece
V614	Sr30	Greece
V619	Sr30	Syria
V34	Lr10	India
V154	Lr10	China
V160	Lr10	Iraq
V187	Lr10	India
V298	Lr10	Spain
V407	RL6057	Iran
V418	Lr10	Spain
V503	RL6061	Afghanistan
V623	Lr10	Iran

TABLE 9

Spring wheat introductions not carrying the originally hypothesized genes for resistance to either stem rust (Sr genes) or leaf rust (Lr genes) and the probable number of unidentified genes and their possible genetic nature

Introduction	Number of Unidentified Genes	Origin	Field Reaction
	Sr genes		
V275	1 R	India	40MS
V285	1 R	Morocco	40MS
V612A	1 R	Greece	10MR
	Lr genes		
V9	1 D	Portugal	10M
V10	1 D	India	70S
V63	1 R	Spain	10MR
V72	1 D	Spain	10M
V111	2 C, 2R or PD	France	40M
V112	1 R	France	10M
V113	1 R	France	20MS
V152	1 D	France	10M
V237	1 D	Tunis	20MS
V278	1 D	Algeria	TR
V279	1 D	Morocco	5MR
V291	1 R	Canary	TMR
V321	1 D	Turkey	10M
V336	2 D	Persia	10M
V410	1 D	Persia	30MS
V618	2 D	Turkey	5R
V628	2 D	Persia	10MS
V637	1 D	China	5MS
V642	1 R	Portugal	30M

R-> reccessive gene
D-> dominant gene
C-> complementary genes
PD-> partially dominant gene

populations or F3 lines should be tested. In fact, the F2 population of V407 X NIL Lr21 was too small (only 42 plants) to allow for significant statistical inference. Also, Lr21 has only recently been introduced into wheat from Aegilops squarrosa (Rowland and Kerber 1974), while RL6057 is a back cross line derived from a hexaploid spring wheat introduction collected in China; PI58548 (Dyck 1977). The same gene has also been found in introductions PI268454 and PI268316 from Afghanistan and Iran, respectively (Dyck 1977). Since V407 also originated in the Persian gulf area, it is likely that it to possesses the resistance gene present in RL6057, rather than Lr21.

Introduction V614 is also of special interest. The F2 population derived from the cross between V614 and RL6071 apparently segregated for rust reaction according to a 9:7 ratio. When V614 was crossed to Sr30, no susceptible F2 plants were observed, demonstrating the presence of Sr30 in V614. Thus it can be postulated that V614 carries either;

(1) two complementary genes, one of which is Sr30

(2) one partially dominant gene which happens to be Sr30

(3) one domiant gene (Sr30) which is subjected to an inhibitor carried by RL6071.

Adult Plant Tests.

Many of the introductions resistant to leaf rust at the adult plant stage listed in TABLE 4 gave no indication of carrying identified adult plant resistance genes. The other introductions however, apparently carried either Lrl2, Lrl3, Lr22 or the gene present in RL5711. The presence of these adult plant resistance genes could then be confirmed using conventional F2 data and monosomic analyses. The presence of either Lr22 or the gene present in RL5711 is unlikely. Both genes are of interspecific origin (from Aegilops) and their presence in unimproved landraces due to natural introgression is doubtful.

Pretorius (1984) suggested that the presence of Lrl3 could be detected at the seedling stage. This would eliminate the need for adult plant testing when working with Lrl3. Unfortunately, this expression of Lrl3 at the seedling stage was not observed in the present study. This non-expression may have been due to the cool ambient temperature in the greenhouse. Pretorius (1984) noted that Lrl3 was detected in seedlings at 25.5 C but not at 18.1 C. Lrl3 is valuable because it conditions resistance against many races in adult plants and is particulary effective in combinations with other genes (Samborski 1984).

Vavilovian Centers of Diversity.

The introductions studied originated in areas which coincide with wheats' Vavilovian centers of origin. It was then to be expected that a good proportion of these introductions would carry good resistance to at least one of the four diseases studied. Twenty seven percent or 104 of the 391 introductions showed some resistance to at least one of the 7 races used. Many of these introductions also expressed good resistance in the field to the bulk inoculum, with the notable exceptions being introductions in the Lr10 group. These results compare well with results of other studies using materials of similar origins. Shirokov and Chmut (1981) found that 680 (29%) of the 2350 hexaploid spring wheat introductions screened showed resistance to either P. graminis tritici and/or P. recondita. Tverdokhled and Goncharova (1981) showed that 33% of the 390 spring wheat introductions tested had resistance to either P. graminis tritici and/or P. recondita. However, Shevchenko et al (1981) reported that only 3% of the introduction they tested had resistance to P. recondita. In the present study, 18% were resistant to P. recondita.

Assuming that wheats' Vavilovian centers of origin do yield large amounts of resistant materials, the question is; why? Vavilov (1949) attempted to answer this by enouncing two rules (laws?) which take into consideration the individuality of both pathogen and host;

(1) the weaker the expression of specialization of parasite on genera and species of host plants, the less the chance for existence, and consequently of finding, immune varieties

(2) the distribution of immunity in narrowly specialized parasites to a great extent is associated with the genetic differentiation of varieties.

In other words, the fact that much genetic variabilty exist in these centers of origin and that these centers coincide with areas where variability in the rust fungi is high (Dietel 1904; Wahl 1958; Zhukovsky 1959; Urban 1980 and Leppick 1970) may account for the evolution of a large number of resistant forms of wheat.

Transgressive Segregation.

The results of the present experiment yielded some evidence suggesting the presence of transgressive segregation. In the case of leaf rust, transgressive segregation was not detected because the level of resistance, in both the resistant introductions and the near isogenic lines was of a ; or;l type making the detection of notable improvments unlikely. The resistance expressed by the stem rust resistant introductions and their corresponding isogenic lines being of a lesser degree made the detection of transgressive segregation more likely. Only a few introductions showed any indication of this (TABLE 10);

TABLE 10
Evidence for Transgressive Segregation

Cross	Best Parental Phenotype	Best F2 Phenotype
333 X Sr9a	1-1+	1
574 X Sr9b	2	1
589 X Sr29	2++	1+
594 X Sr9b	2	1+

F2 plants slightly more resistant than either parents were considered to be transgressive segregants.

Reversal of Dominance.

Evidence for reversal of dominance was found in 3 introductions; V314, V407 and V499 (TABLE 11).

TABLE 11

Evidence for reversal of dominance in three rust resistant introductions

	F2 Ratios	
Introduction	SXR Cross	RXR Cross
V314	3:1(RL6071)	13:3(Sr29)
V407	3:1(TC)	13:3(Lr30)
V499	3:1(RL6071)	13:3(Sr7b)

Introductions V314, V407 and V499 when crossed to their corresponding universal suscepts gave rise to F2 populations which segregated for rust reaction according to 3:1 ratios suggesting the presence of single dominant genes. When the same introductions were crossed with their corresponding NILs, the resulting F2 populations segregated for phenotype according to 13:3 ratios suggesting the presence of two genes, one dominant and the other recessive. In the absence

of reversal of dominance, the RXR F2s would be expected tofit 15:1 ratios and not 13:3 ratios as observed. Either the genes carried by the NILs or by the introductions may have been affected.

Smut

Of the races of *Ustilago tritici* used in the present study, only T10 is prevalent in western Canada (Dr. J. Nielson, personal communication). Race T2 is almost non-existent in western Canada because most of the cultivars grown there are resistant to it (Nielson, personal communication). Race T13 and T39 are foreign to Canada. Race T13 is from the USSR and Race T39 is from the eastern USA (Nielson, personal communication). The main reason for having used these 4 races rather than 4 other more prevalent races is that the spectrum of virulence obtain when combining these races is most complete. That is, no other combination of four races carries as many virulence genes, notably *Utv1*, *Utv2*, *Utv3* and *Utv4*. Only one of the known genes for virulence in *U. tritici*, *Utv5*, is absent in this race combination.

The screening for resistance to *U. tritici* is hampered by three major constraints;

(1) the need for growing out the progeny of an inoculated plant to determine its reaction to the fungus

(2) the poor resolution of the differentials used, that is, they are not all necessarily isogenic

(3) the sizable effect of the environment on the differentials which is of particular importance when these are used in third world countries under extreme light and heat conditions
(4) the necessity to use a mixture of races, rather than pure cultures, which may lead to the suppression of the virulence of any one race in the mixture (Neilson 1977).

Since wheat is self pollinated, it can be assumed that the introductions are homozygous at most loci, including those responsible for resistance to U. tritici. This assumption is of particular importance when screening for resistance. The U. tritici resistant materials identified in this study are intended for use as sources of resistance in breeding programs and, since infection of a host leads to a total destruction of its grain yield, full immunity is desired. However, if the introductions are not homozygous, which is unlikely with a self pollinated crop such as wheat, low level of infection may be due to host heterozygosity. It is also possible that the introductions are susceptible and that the low level of infection is due to a poor inoculation technique. Of greater importance is the degree of heterogeneity of the landrace population represented by the introduction. Since probably only a few heads of the landrace were initially collected and only a few heads per introduction were inoculated, the infection data obtained is not necessarily representative of the variability that may exist within the landrace population. Anderson (1961b)

encountered a similar situation, He reported that;

> "...if a variety was heterogeneous for resistance, and an immune genotype was selected by chance, the variety was considered immune. Heterogeneity for loose smut resistance may be more widespread than is usually realized... In many tests for cultivar resistance, some cultivars are immune, others carry trace to 30% smut, while still others are completely or nearly completely susceptible. The question is whether all those in the trace-to-30% group are susceptible, or whether only a portion of the population is susceptible..."

Introductions immune to all four U. tritici races used in this study should prove to carry stable loose smut resistance and thus be of use in future breeding programs. However, due to the heterogeneous natures of the introductions, one should not expect all selections from these introductions to be equally smut resistant.

Three percent of the 391 introductions were designated as being either immune or highly resistant. In a similar study performed by Neilson (1983), 8% of the 2644 introductions showed either immunity or a high degree of resistance. These figures are in accord with the 5-10% resistant introductions found among landraces obtained from the International Wheat Nursery and tested by Dr. J. Neilson at the Agriculture Canada Research Station, Winnipeg, Manitoba over a 20 year period, (Neilson, personal communication).

Tan-Spot

The isolates used in this screening experiment were representative of those prevalent in Manitoba (Dr. A. Tekauz, personal communication). Resistance to them, notably CDA 1241, should prove useful.

Studies comparable to the present, that is, involving materials from similar origins, could not be found in the literature. Reported results were of the cultivar trial type involving lines and cultivars of domestic and foreign origins (Hosford 1981).

The scale presented by Hosford (1981) for evaluating tan spot infection was meant to be used for quasi-adult plants in the field. Since it was inadequate for scoring seedlings grown in the greenhouse a new scheme had to be developed. Due to time and space limitations, this scheme (Appendix G) was not evaluated further and could probably be improved.

Luz and Hosford (1980) mentioned that physiological specialization in *D. tritici-repentis* led to a wide range of races virulent on wheat. This is most probably due to the occurrence of the sexual stage of the fungus on the North American continent. Under these circumstances monogenic resistance mechanisms would probably not endure. Simply inheritated resistance has been reported by Lee and Gough (1984), Frohberg (1982) and Gough (1982). More complex resistance such as that reported by Nagle et al (1982) should be sought for or possibly developed by introducing a number of single genes into a cultivar.

SUMMARY AND CONCLUSIONS

Spring wheat introductions from northern Africa, Asia, the Middle East and the Medeterranean Sea and Persian Gulf areas were screened for resistance to races of Puccinia graminis tritici (stem rust), Puccinia recondita (leaf rust), Ustilago tritici (loose smut) and Drechslera tritici-repentis (tan spot). Twenty-seven percent (102) of the 391 introductions expressed resistance to either stem or leaf rust. This figure is comparable to that of other studies using materials of similar origins. Only 3% of the introductions were found to be either immune or highly resistant to U. tritici. This percentage is also similar to that obtained in similar screening reports. Sixty-nine, or 18% of the introductions, were resistant to at least one of the four D. tritici--repentis isolates used.

Some of the plants believed to possess adult plant resistance to leaf rust were assigned hypothetical Lr genotypes, notably Lr12, Lr13, Lr22 or the gene in RL5711. The detection of Lr13 in the seedling stage, as described by Pretorius (1984), was possibly hampered by the cool ambient temperature in the greenhouse where the seedlings were grown. Hypothetical Sr genotypes could not be assigned to introductions believed to carry adult plant resistance to stem rust due to the lack of infection.

The inheritance of resistance to stem and leaf rust was studied in 42 of the 102 resistant introductions. Evidence for the occurrence of transgressive segregation and reversal of dominance was observed in a number of introductions. Eleven of the stem rust resistant introduction and 9 of the leaf rust resistant introductions were found to carry identified resistance genes, notably, Sr30 and Lr10. The remaining 22 introductions were shown not to carry identified resistance genes. These introductions could ultimately prove to be sources of new genes for resistance to stem and/or leaf rust. These possibly new genes could be identified, located and designated through the use of either a broader spectrum of differential races, genetic studies and/or monosomic analysis. Such efforts should be directed mainly to those introductions showing good field resistance.

Seventeen stem rust resistant introductions and 3 leaf rust resistant introductions could not be assigned any Sr/Lr hypothetical genotypes and the genetic nature of their resistance remains unknown. These introductions cannot as of now be considered as carriers of new genes. Many of the ITs observed on these introductions, especially those resistant to stem rust, were intermediate and could easily be reclassified as either resistant or susceptibile. These introductions should be retested to the same and additional differential races to ascertain the unidentified character of their Sr/Lr genotypes.

A good proportion of the 391 introductions possessed disease resistance with approximately 40% of the introductions expressing resistance to at least one disease. This high percentage was anticipated since most of the introductions originated from areas coinciding with Vavilovian centers of origin. These centers, known to contain much genetic variability, had previously been reported to yield large amounts of resistant materials.

Though the merits of polygenetically controlled rust resistance have been noted in the past, wheat materials carrying single genes for resistance should not be neglected. The trend in the recent past of developing wheat cultivars with "broadly based specific resistance" has proven successfull (Luig and Watson 1970; Green and Campbell 1979). The availability of wheat materials possessing single and easily transferable genes for resistance has been in part responsible for this development. The search for such new genes for disease resistance should allow for the continued development of disease resistant wheat cultivars.

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APPENDICES

Appendix A: Methods for stem and leaf rust inoculations

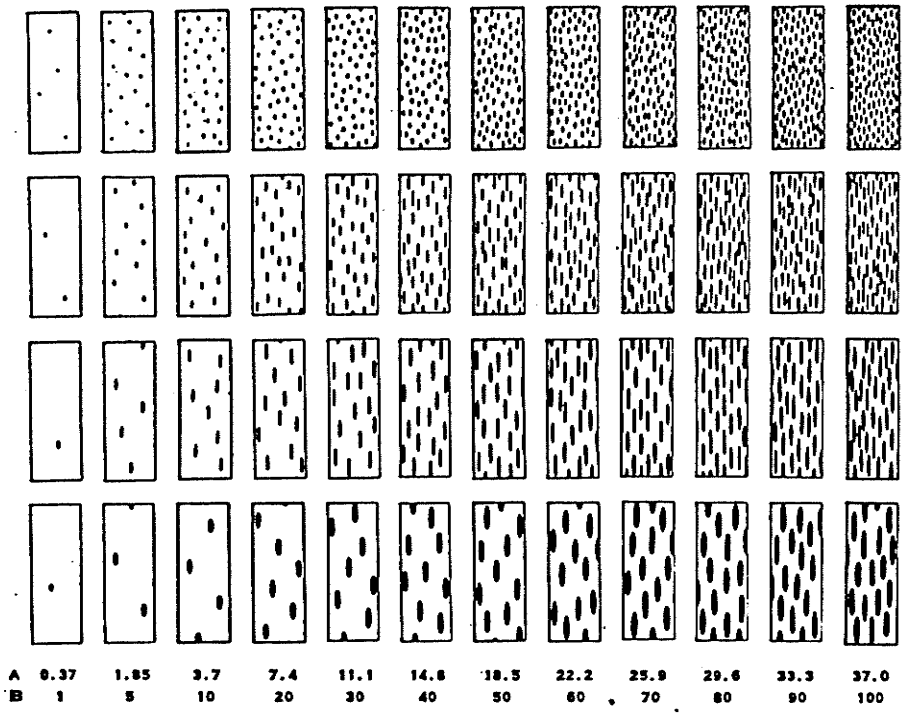
a) in the field: Inoculum in the field were generated by inoculating susceptible border rows at the 5 to 6 leaf stage using a mixture of talc and rust spores. The mixture was dusted onto the border rows at dusk to take advantage the dew formation. Secondary inoculum produced by these border rows was responsible for the infection of the introductions within the plots.

b) in the greenhouse at the seedling stage: Seedling were grown to the 1 1/2 leaf stage, sprayed with Tween 20 and water and then dusted with a mixture of talc and rust spores. The seedlings were immediately put into an incubation chamber for 24 h. For stem rust inoculations, the spores were suspended in oil and sprayed onto the leaf surface with an atomiser.

c) in the greenhouse at the adult stage: Plant were grown to heading, sprayed with Tween 20 and inoculated. Only the flag leaf was inoculated. Due to the poor infection obtained with the dusting of the spores and talc onto the leaves, individual flag leaves were inoculated by the application of spores directly on the surface of the leaf using the thumb and index finger. The plants were then put into the inoculation chamber for 24 h.

Appendix B: Scale used for scoring rusted plants in the field. (A) refers to actual percentage of area occupied by rust pustules and, (B) refers to the standard rust readings according to Peterson and al 1948). This type of scoring may be supplemented with infection type characterization as described in appendix C.

eg: 70% coverage with "moderately susceptible" pustules (MS) => 70MS.



Appendix C: Infection type (IT) characterizations applicable to seedlings and adult plants of wheat. These ITs can be produced by either Puccinia gaminis tritici or P. recondita (see Stakman and Levine 1922).

Types of Infection

0 - IMMUNE[0]: no uredinia developed; hypersensitive flecks usually present, but sometimes there is absolutely no trace of mycelial invasion in the host tissue

1 - VERY RESISTANT[R]: uredinia minute and isolated; surrounded by sharp, continuous, hypersensitive, necrotic areas

2 - MODERATELY RESISTANT[MR]: uredinia isolated and small to medium in size; hypersensitive areas present in the form of necrotic halos or circles; pustules often in green, but slightly chlorotic, islands

3 - MODERATELY SUSCEPTIBLE[MS]: uredinia medium in size; development of rust somewhat subnormal; true hypersensitiveness absent; chlorotic areas, however, may be present

4 - VERY SUSCEPTIBLE[S]: uredinia large, numerous and confluent; true hypersentiveness entirely absent, but chlorosis may be present when cultural conditions are unfavorable

X - HETEROGENEOUS: uredinia very variable, apparently including all types and degrees of infection on the same blade; no mechanical seperation possible; on reinoculation small uredinia may produce large one, and vice versa; infection ill defined

Z - HETEROGENEOUS: same as ´X´ except that infection types tend to become progressively more severe towards the tip of the leaf blade

Y- HETEROGENEOUS: same as ´Z´ except that infection types tend to become progressively more severe towards the base of the leaf blade.

Degrees of Infection

(=) - TRACE[T]: uredinia very few in number and covering a limited surface; development of rust generally poor and decidedly subnormal

(-) - SLIGHT: rust development below normal, but somewhat better than trace

(+-) - MODERATE: variation in rust development from "slight" to "considerable"; when infection is uniform but only medium in quantity the symbol is omitted

(+) - CONSIDERABLE: infection better than normal; uredinia fairly numerous and scattered

(++) - ABUNDANT: luxuriant development of rust; uredinia very many, covering large area of affected host

Miscellaneous Symbols

(;) - hypersensitive flecks

(.) - necrotic lesions

Appendix D: Minimum family sizes required to differentiate between 2 expected proportions, a1 and b1, based on .05 and .025 levels of probability, (according to Hanson 1959).

$$\sqrt{n} = t\left[\frac{(a_1a_2)^{\frac{1}{2}} + (b_1b_2)^{\frac{1}{2}}}{b_1 - a_1}\right],$$

Where;

a1 is the expected proportion of the population having a certain character and

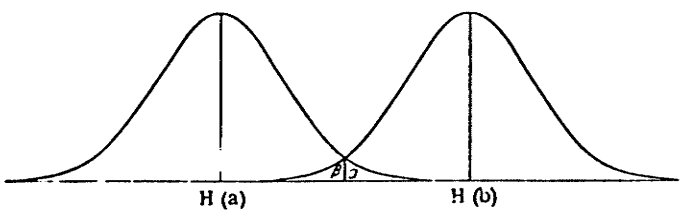
a2 is the expected proportion of the population not having the character for a given hypothesis (a1 + a2 = 1)

b1 and b2 have similar interpretation for the alternative genetic hypothesis

"The problem then is to select n, the family size of such magnitude that one can identify a1 from the alternative hypothesis, that b1 is the expected proportion (or vice versa) with a reasonable degree of assurance".

Different values of n for various a1/b1 hypotheses are tabulated below;

a_1 \ b_1	.500	.525	.550	.575	.600	.625	.650	.675	.700	.725	.750	.775	.800	.850	.900	.950
.500	=	4330	1080	476	265	168	118	83	63	48	38	31	25	17	11	7
	=	6140	1530	675	377	239	164	117	89	69	54	43	34	23	16	10
.525		=	4310	1010	471	262	163	113	82	60	47	37	29	19	13	9
		=	6110	1520	669	372	235	160	116	86	66	52	42	27	18	11
.550			=	4260	1060	464	257	162	110	79	59	54	35	22	15	9
			=	6050	1500	658	365	230	182	112	84	64	50	32	20	13
.575				=	4200	1040	454	251	158	107	76	57	43	26	17	10
				=	5960	1480	645	357	223	152	105	80	61	37	23	14
.600					=	4110	1020	442	244	152	103	78	54	32	19	12
					=	5840	1440	628	346	216	146	104	77	45	27	16
.625						=	4000	982	427	235	146	98	70	38	22	13
						=	5680	1400	607	333	207	139	99	54	40	18
.650							=	3870	947	411	225	139	94	48	27	15
							=	5500	1350	583	319	197	132	67	38	21
.675								=	3720	907	381	213	131	61	32	17
								=	5280	1280	535	302	186	86	45	24
.700									=	3550	860	389	200	80	39	20
									=	5040	1230	524	283	114	56	29
.725										=	3330	808	345	112	50	24
										=	4760	1150	490	159	79	34
.750											=	3140	751	169	85	25
											=	4450	1070	240	92	41
.775												=	2900	299	90	36
												=	4110	410	127	51
.800													=	796	133	46
													=	881	189	66
.850														=	468	90
														=	664	128
.900															=	291
															=	413
.950																=
																=



Appendix E: Chi-Square calculation as a measure of homogeneity of data from different F2 families (from Mather 1957)

$$X^2homo = [X^2fam1 + X^2fam2 + \ldots + X^2famn] - X^2$$

Where;

X^2homo is a measure of the homogeneity of the data from different families

X^2famn is a measure of the discrepency between the observed and expected ratios (see Appendix I) within each of the n F2 family

X^2 is the Chi-Square value for the F2 population as a whole as calculated in Appendix I

with n-1 degrees of freedom

Appendix F: Chi-Square calculation for F2 population as a whole.

$$X^2 = \frac{(\text{Observed} - \text{Expected})^2}{(\text{Expected})}$$

Where;

OBSERVED -> observed proportion of plants being either resistant or susceptible

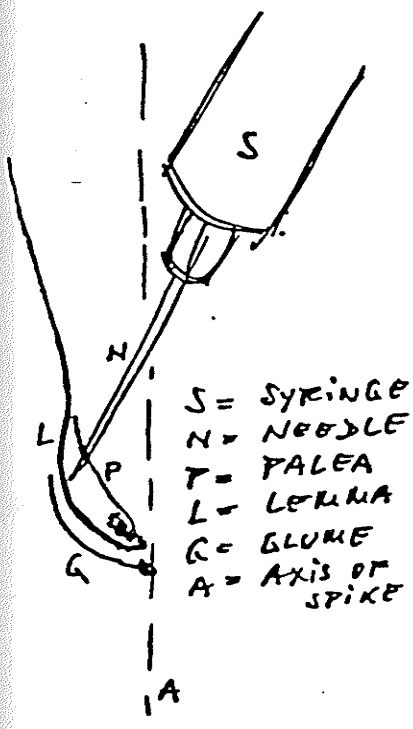
EXPECTED -> theoretical or expected proportion of plants being either resistant or susceptible

with one degree of freedom

Appendix G: Test in the greenhouse and in the field for reaction of wheat introductions to loose smut (Ustilago tritici) (adapted from Poehlman 1945).

Inoculum. Two mixtures of races were used; T2/T10 and T13T39. To maintain or increase these races, a susceptible check should be inoculated seperately. After several years, a fresh supply of spores of the two races should be obtained to prevent the testing with only one of them. At room temperature, spores will lose their viability within 2-3 months, whereas in a fridge at 2-5 C they will stay viable for many years.

The inoculum should have a concentration of approx. 1g spores/ 1 water/ race. Too high a concentration is detrimental. Since somewhat lower concentration have only minimal effect on % infection, there is no need to waste time on fine adjusting the suspension. To prepare it, a piece of infected spike is broken off and the spores rubbed off under water into a pertri plate. To remove particles that would not pass through the needle at inoculation the suspension is screened through a dense nylon mesh, 90/cm, which is folded twice, wetted before screening, and held over a beaker in shape of a funnel.



Inoculation. Two spikes per introduction were inoculated at anthesis. The optimal time for inoculation is when the anthers extruded by the florets in the middle of the spike begin to turn white. Most of the anthers of the upper and lower spikelets will still be yellow, and the most distal florets may not yet have dehisced.

A 5 or 10 ml syringe is used for injection of the inoculum; with a gauge 22 or 24 hypodermic needle, 1/2 or 3/4 inch long. The syringe is held at an angle of about 10 degree to the rachis. The needle is inserted into the floret by piercing through the upper third of the palea. When a slight resistance is felt due to the needle reaching the tougher lemma, the plunger is press momentarily to inject a drop of inoculum. There will be a change of hue of color of the lemma as the floret fills with inoculum. The lowest florets on one side of the spikelet and in one row should be inoculated first.

Evaluation. Once the inoculated spikes are mature, harvested and threshed, at least 30 kernels from each spike are seeded in the greenhouse. After heading, the infected and total number of plants are counted and percent infection is established.

Appendix H: ´Misting Method´ for Drechslera tritici-repentis inoculations.

This method was developed by Dr. Andy Tekaus, here at the Agriculture Canada Research Station in Winnipeg, Manitoba.

Single spores of isolate CDA 1241 were transfered onto a few (5-6) 12 cm PDA filled petri-plates and allowed to grow for ten days at 20 degrees celcius under a 12-12 h light-dark regime. At that time small 0.5 cm2 squares of PDA and mycelium from the periphery of the plate were transferred to 60 V8-agar slants. The slants were incubated for ten days at 20 degrees Celcius under a 12-12 h light-dark regime to permit extensive mycelial growth. The slants were then filled with sterile distilled water and vigorously shaken. The water and mycellium were poured into 20 cm petri-plates filled with V8-agar. These plates were incubated for 6 days at 20 degrees Celcius under a 12-12 light-dark regime. The resulting conidia were vigorously shaken from the plates, sieved using cheese cloth and diluted to approximately 10 000 spores/ml. Ten drops of Tween 20/l are added to the inoculum. The method gave one litre of inoculum and which was sufficient to inoculate over 3000 seedlings using a small atomizer and compressor. The inoculated seedlings were then incubated in the darkness for 24 h at 100% RH and ambient temperature.

Appendix I: Evaluation of Drechslera tritici-repentis Infections Following Inoculation by the 'Misting Method' (Appendix H).

The following scale, developed by Dr. Andy Tekaus of the Agriculture Canada Research Station, Winnipeg, Manitoba, was used to evaluated infections obtained by the 'Misting Method' using data from all 4 incubation periods.

Symptoms After	Severity	Reaction
06 h incub.	>trace	VS
06 h incub.	trace	S
12 h incub.	>light	MS-S
12 h incub.	trace or light	MS
* 24 h incub.	>light	MR-MS
* 24 h incub.	trace or light	MR
48 h incub.	>light	R-MR
48 h incub.	trace or light	R
no symptoms	------	VR

Appendix J: ´Dipping Method´ for D, tritici repentis inoculations.

This method is that of Dr. R. M. Horsford, Jr. (1982). The method goes as follows;

> Laboratory grown conidia are produced from strains from single conidia, ascospores or leaf spots. Each strain is grown on 48 petri plates of PDA for 10 days at 23-25 degree Celcius, until the mycelium grows within 0.5 cm of the sides of the plates. Then 1 cm-diameter disks of mycelium and agar from the young light grey mycelium near the sides of the plates are placed 10 to a plate on modified V8-agar. Forty eight of these inoculated V8-agar plates are placed at 19-22 degreee Celcius for 24 h in the light followed by 18 h in the dark. The resultant conidia are vigorously shaken from the disks in 300ml of water, sieved, diluted to 1200-2000 conidia per ml in a final volume of 1l. Five drops of Tween 20 are added to the solution. A variation in the method that produces good inoculum when conidiation is poor, is to blend the 480 disks of conidia, conidiophores, mycelium and PDA for 20 sec in 1 liter of water and then add 5 drops of Tween 20. Within one hour the upper leaves of 16 headed plants or 120 seedling are dipped and shaken for one minute in the mixture and the excess mixture is discarded.

Appendix K: Evaluation of *D. tritici repentis* infections following inoculations by the 'Dipping Method', (Appendix J).

The following scheme was developed by myself:

Readings were taken on fourth and fifth leaves and, on the first to third leaves. A scale of 1 to 9 was used. A 1 score indicating almost complete immunity and 9 indicating almost complete susceptibility. Scores of 1 to 5 were considered as resistant (R). Scores 6 to 9 were considered as susceptible (S). Since the level of infection on the fourth and fifth leaves varied from that on the first, second and third leaves, the plants were classified as resistant (R) or susceptible (S) according to the following convention;

fourth+fifth	leaves	R	/	first-third	leaves	R	=>	R
"	"	"	R	/	"	"	"	S	=>	R
"	"	"	R	/	"	"	"	-	=>	R
"	"	"	-	/	"	"	"	R	=>	R
"	"	"	S	/	"	"	"	R	=>	R
"	"	"	S	/	"	"	"	S	=>	S
"	"	"	S	/	"	"	"	-	=>	S
"	"	"	-	/	"	"	"	S	=>	S

The plants were then arbitrarily classified according to their phenotypes.

Phenotype	Iso1	Iso2	Iso3	Iso4	Iso5	Iso6
1	R	R	-	R	R	-
2	R	R	-	R	S	-
3	S	R	-	R	R	-
4	R	R	-	S	S	-
5	S	S	-	R	R	-
6	S	R	-	R	S	-
7	R	S	-	S	S	-
8	S	R	-	S	S	-
9	S	S	-	R	S	-
10	S	S	-	S	R	-

Iso1 -> CDA 1241
Iso2 -> CDA 866
Iso3 -> CDA AT82 - 254 -1
Iso4 -> CDA AT76 - 199 -2
Iso5 -> CDA AT82 - 199 -1
Iso6 -> CDA AT82 - 27 -1

Appendix L: A Brief Description of Sr and Lr Genes Mentioned in This Study

Genes for Stem Rust Resistance

Sr7b,(Chinese Spring X Hope): Sears and Loegering (1961) note a factor in the cultivar Hope which conditioned a reduction of the size of the pustule without the development of necrosis. The degree to which the effect is produced varies with the culture, but the infection type was reported to be in the 3 class. It was demonstrated that the resistance present was dominant and fairly simply inherited, prehaps due to a single gene.

Green et al (1960) found that Sr7a was allelic or closely linked with a gene responsible for a type 2 infection on the cultivar Marquis. The gene was designated Sr7b. The gene is said to have been originally derived from Red Fife and then transferred to many cultivars, notably Hope, H-44 and their derivatives.

Sr13,(Prelude X Sr13): Sr13 was first reported present in the cultivar Khapstein, (Knott 1962). Khapstien was found to carry gene Sr7 which controls resistance to C10 and two additional genes, one of which was designated Sr13. The latter conditions a 2+-3 reaction to C10 and a type 2 infection to C17. McIntosh (1972) located Sr13 on the beta arm of chromosome 6A, 0.54 +/- 0.07 map units from the centromere.

Sr15,(Prelude X Sr15): The gene was first reported in the cultivar Norka and is said to be effective only at temperatures below 21 C, (Watson and Luig 1966). The authors also located Sr15 on the long arm of chromosome 7A and reported that it was closely linked with genes controling resistance to strains of organisms causing leaf rust and powdery mildew.

Sr17,((Prelude X Mg) X Esp.): The gene was first reported present in the cultivar Hope, (McIntosh et al 1967). The authors noted that the gene was operative in both seedling and adult stages and recessive and thus designated sr17. sr17 is apparently ineffective in conferring resistance to North American stem rust races. According to the authors, it's incorporation into Hope, H-44 and their derivatives presumably resulted from it's linkage with genes for resistance to other diseases. It was located on the long arm of chromosome 7B by McIntosh et al (1967).

Sr29,((Prelude X Mq) X Etoile de Choisy): Sr29 was first reported in the cultivars Etiole de Choisy by McIntosh et al (1974). Dyck and Kerber (1977) located it on the beta arm of chromosome 6D. The gene was previously designated SrEC, (Dyck and Kerber 1977).

Sr30,(Webster): Knott and McIntosh (1978) were the first to report the gene (tentatively designated SrW) on the cultivar Webster. their monosomic analysis indicated that Sr30 was located on the long arm and independant of the centromere of chromosome 5D.

Genes for Leaf Rust Resistance

Lr3,(TC X Democrat, RL6002): Its presence was first noted by Mains et al (1926), designated Lr3 by Ausemus et al (1946) and located on the long arm of chromosome 6B by Sears and Loegering (1961).

Lr10,(TC X Exchange, RL6004): The presence of the factor was first reported and designated Lr10 by Chaudhuri (1958) and located on chromosome 1A by Dyck and Kerber (1971).

Lr11,(TC X Hussar, RL6053): The Variety Hussar was found to carry a major dominant gene for leaf rust resistance to physiological race 15 on chromosome 2B, (Soliman et al 1963). This gene was designated Lr11.

Lr12,(Exchange X TC , RL6011): Two independantly inheritated genes were identified and isolated one from each of two varieties, (Dyck et al 1966). The gene isolate from the variety Exchange was designated Lr12. it conditioned a type 2 reaction to race 5 in the adult stage, is partially dominant and located on 4A, (Dyck and Kerber 1971).

Lr13,(Manitou, 81P T Inc 36): The gene isolated from the cultivar Frontana (Dyck et al 1966) was designated Lr13. It is partially dominant but when transferred to Manitou it behaves as a reccessive gene, (Dyck et al 1966).

Lr16,(TC X Exchange, RL6005): The gene was initially reported as LrE or ´E gene´ by Anderson (1961a). The symbol Lr16 was later assigned by Dyck and Samborski (1968b). Lr16 and Lr12 were both located on chromosome 4A using the Rescue monosomic series, (Dyck and Kerber 1971). However, the genes, being more than 50 map units apart, segregated independantly.

Lr21,(TC X RL6054, RL6043): Lr21 was introduced into synthetic hexaploid wheat (2n=42=AABBDD) from Aegilops squarrosa (RL5289), (Rowland and Kerber 1974), and later located on chromosome 1D.

Lr22,(TC X RL5406, RL6044): Following monosomic analysis, Rowland (1972) associated the adult plant resistance found in the resistant lines RL5404 and RL5406 to a gene he designated as Lr22, introduced into hexaploid wheat from Ae. squarrosa (RL5829) and located on chromosome 2D. Lr22 was later located

on the alpha arm of 2D 63.6 +/- 4.8% map units from the centromere by Rowland and Kerber (1974).

Lr30,(TC X Terenzio, RL6049): LrT (re-designated Lr30) present in the backcross line RL6049 and originally derived from the cultivar Terenzio was located on the long arm of chromosomr 4B, 2.9 +/- 1.3 map units from the centromere, (Dyck and Kerber 1981).

RL5711, (Marquis X RL5347). The adult leaf rust resistamce gene present in RL5711 was apparently derived from Aegilops speltoides via the tetraploid RL5347 (Ae. speltoides X T. monococcum; 2n = 28 = SSAA) (E.R. Kerber, personal communication). RL5711 also possesses one gene for seedling stem rust resistance which is closely linked to the adult leaf rust resistance gene (E.R. Kerber, personal communication).

RL6057,(TC X PI58548): Plant introductions PI268454a, PI58548 and PI268316 were reported having a common gene conferring a 1+ type of resistance tentatively designated as ´1+gene´ by Dyck (1977). A backcross line, RL6057 (Tc*6/PI58548) was developed by the author.

RL6061,(TC X PI268316): Gene C was isolated from plant introduction PI268316 which has 3 interacting genes for resistance, one giving a 1+ reaction (ie: 1+gene), one similar to LrB (probably LrB) and a third, tentatively designated Gene C, giving a 2+ reaction, (Dyck 1977). A backcross line, RL6061 (Tc*6/PI268316) was developed by the author.

RL6050,(LrT2 and LrT3): Introductions Terenzio, Lageadinho, Frontana, 72 Hills 175, PI321999, PI197249, CRIC26809-68 and CIRC32125 have in common two complementary genes, LrT2 and T3, that give a variable type of resistance, (Dyck and Samborski 1982). A backcross line, RL6050, was developed by Dyck and Samborski (1982).