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Genetics of stripe rust (*Puccinia striiformis* Westend.) resistance in the soft white spring wheat, Owens.

BY

Joanna Pinto

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

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IN

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled **Genetics of stripe rust (*Puccinia striiformis* Westend.) resistance** submitted by **Joanna Pinto** in partial fulfillment of the requirements for the degree of **Master of Science in Plant Breeding.**

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..Robert Conner.....

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Date.....

ABSTRACT

Stripe rust (yellow rust), caused by *Puccinia striiformis* Westend. is a major disease of wheat and barley, particularly in cool, moist growing seasons. The Canadian soft white spring wheat, Owens, is known to possess resistance to the stripe rust race SRC 4-84. The aim of this project was to study the genetics of resistance in Owens. Several Kenyan wheat cultivars were also tested for their response to the Canadian stripe rust races.

The cultivar Owens posesses one, homozygous dominant gene for seedling resistance against SRC 4-84. This gene is not Yr1, Yr3a, Yr3b, Yr3c, Yr4a, Yr4b, Yr5, Yr6, Yr7, Yr8 or Yr10. It is unlikely to be Yr9 or Yr15, as neither gene is indicated in the Owens pedigree. The remaining, untested seedling genes are Yr2 and YrA. It is also possible that the resistance in Owens is conferred by a gene that has not been described.

The yellow rust differential cultivar Hybrid 46 exhibits a recessive resistance to race SRC 4-84, which is either being conferred by Yr3b or a previously undescribed gene in this cultivar.

Eight Kenyan lines (Kenya Chiriku, Kenya Mlembe, Kenya Popo, Kenya Kulungu, Kenya Nyumbu, Pasa, Kwale, Paa) were resistant to the Canadian stripe rust race SRC 4-84. Based on already published data and the findings of this study, it was determined that the effective resistance in Kenya Kulungu against race SRC 4-84 is being conferred by Yr3b or a previously unidentified gene. Low levels of residual resistance were detected in seven, older cultivars (Kenya Tembo, Kenya Swara, Kenya Leopard, Kenya Page, Bounty, Romany, Africa Mayo).

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1. GENERAL INTRODUCTION

Stripe rust (yellow rust), caused by *Puccinia striiformis* Westend., is a major disease of wheat, barley and other grasses (Mulder and Booth, 1971). It is mainly a problem in areas with cool, moist growing seasons since fungal growth is limited by nighttime temperatures above 20°C (Park, 1990; Rapilly, 1979). Such conditions occur in temperate climates or at higher elevations (2400-3000m) in tropical climates (Tanner and Van Ginkel, 1988). Under a severe infection, yield losses due to this disease have ranged as high as 79% in Canada (Conner and Kuzyk, 1988). The rust is capable of overwintering as mycelium in infected leaves and is spread by uredospores (Martens *et al.*, 1985). Although yield loss estimates are not available from Kenya, stripe rust is known to be an economically serious disease (Tanner and Van Ginkel, 1988).

Stripe rust is more strongly influenced by environmental variation than any other cereal rust, which makes it difficult to evaluate genotypic resistance in the host (Robbelen and Sharp, 1978). Resistance conferred by minor genes is particularly sensitive to changes in the environment (Lewellen *et al.*, 1967).

Besides the use of fungicides (Brown *et al.*, 1986; Conner and Kuzyk, 1988), genetic diversity can be managed to reduce rust severity (Knott, 1989). Most of these methods require a knowledge of the resistance genes present in the breeding material. The purpose of this study was to identify the gene(s) conferring stripe rust resistance to race SRC 4-84 in the Canadian soft white wheat cultivar Owens. An additional investigation was conducted to learn more about stripe rust resistance in some Kenyan cultivars, by testing them with two Canadian races.

2. LITERATURE REVIEW

2.1 ECONOMIC IMPORTANCE

Stripe (yellow) rust, caused by *Puccinia striiformis* Westend., is a major rust diseases of wheat and barley (Mulder and Booth, 1971) and is primarily found at high elevations and in areas with cool, moist growing conditions. Temperatures above 20°C prevent rust buildup (Newton and Johnson, 1936; Park, 1990; Rapilly, 1979; Tolenaar and Houston, 1967).

In Canada, the 1990/91 seeded acreage of wheat was 34.8 million acres (Canadian Wheat Board, December 1990) of which 220,000 acres was soft white spring wheat which is particularly susceptible to stripe rust. Yield losses in soft white spring wheat have ranged as high as 40%, and grain from severely infected fields can also be downgraded because of kernel shrivelling (Conner and Kuzyk, 1988). In Canada, this disease is primarily a problem in southern Alberta and British Columbia (Martens *et al.*, 1985). In Montana, yield losses up to 30% in soft white spring wheat and up to 60% in some susceptible winter wheats have been reported (McNeal and Sharp, 1963). Doling and Doodson (1968) estimated yield loss in Britain by comparing varieties in yield trials subjected to severe natural infection with those in uninfected trials, from 1956-1966. Both spring and winter wheats had similar losses that ranged between 8-20% at stripe rust foliar infections of 15-61%. They speculated that higher losses reported elsewhere in the literature might have been due to the infection having spread to the glumes. Yield losses in excess of 50% have been reported in the USSR (Domashova, 1959) and on susceptible wheat cultivars in Australia (Park *et al.*, 1988; Smith *et al.*, 1986).

Stripe rust has its centre of origin in Transcaucasia, from where it spread to other parts of the world (Stubbs, 1988). The rust moved into Europe where it subsequently evolved into distinct populations within the continent. Stripe rust is also distributed through several zones in Asia, and primarily along the western mountain ranges of North and South America (Stubbs, 1988). In 1979, stripe rust first reached Australia and is now a very serious disease, particularly since the low yield potential in many wheat-growing areas precludes the economical use of chemical control (Wellings and McIntosh, 1990). In Kenya, stripe rust is a chronic disease problem, though precise yield loss estimates are not available. Both wheat and rust survive year-round in the cool, humid ecological niches of the upland areas around Mt. Kenya and on the Mau Escarpment (Bonthuis, 1985). The close relationship between changes in the host and pathogen in these perennial reservoirs is thought to foster the local evolution of new virulence (Bonthuis, 1985).

2.2 TAXONOMY

According to a review by Zadoks (1961), Schmidt described yellow rust in 1818 under the name *Uredo glumarum*. In 1854, Westendorp described yellow rust under the name *Puccinia striaeformis*; Fuckel in 1860 gave it the name *Puccinia straminis*. The name *Puccinia glumarum* was introduced in 1896 by Eriksson and Henning and used commonly until the late 1950's and early 1960's when the name *Puccinia striiformis* was revived in the literature. This is now the accepted name for the yellow rust (stripe rust) fungus (Zadoks, 1961).

Puccinia striiformis belongs to the class Basidiomycetes. The classification is as follows;

Subdivision	Basidiomycotina
Class	Basidiomycetes
Subclass	Teliomycetes
Order	Uredinales
Family	Pucciniaceae
Genus and species	*Puccinia striiformis*

2.3 LIFECYCLE

The basic rust lifecycle consists of five spore stages although many variations occur. A rust that has all five spore stages is referred to as macrocyclic; demicyclic or microcyclic if it has fewer than five stages, depending on which stages have been omitted (Laundon, 1973). A rust is heteroecious if it requires an alternate host in its lifecycle, and autoecious if it can complete its lifecycle on one host. Stripe rust is a hemiform rust (Muldur and Booth, 1971), described as one in which only uredospores and teliospores are found (Petersen, 1974). These are sometimes thought of as imperfect rusts in which other stages will eventually be found. Although teliospores of *P. striiformis* can be easily germinated, there is no evidence that basidiospores occur in the natural lifecycle of the rust (Wright and Lennard, 1978). Because of their shortened lifecycles, hemiform rusts do not have an alternate host. The alternate host for stripe rust may only have existed in the centre of origin (Stubbs, 1985), which still contains the greatest number of physiological races of the rust (Leppik, 1970). The regenerative part of the stripe rust lifecycle consists of repeated cycles of the asexual uredial stage. The rust is capable of overwintering as mycelium in the tissue of

living plants, or as uredospores (Martens *et al.*, 1985). Teliospores of yellow rust are non-functional since they are incapable of reestablishing the rust. Stripe rust is assumed to be heterothallic (Little and Manners, 1969b; Wright and Lennard, 1980). Based on studies of seven *Puccinia* species, McGinnis (1956) reported that homothallic rusts had the chromosome number n=4 and heterothallic rusts had chromosome numbers of n=3 or n=6. Haploid chromosome numbers of three (Wright and Lennard, 1978) and six (Goddard, 1976a) have been reported for *P. striiformis*, which suggests that this fungus is heterothalic.

2.4 VARIATION WITHIN THE PATHOGEN

2.4.1 Formae speciales

Puccinia striiformis infects wheat, barley and other grasses. The fungus has not been reported to attack oats (*Avena sativa* L.), rice (*Oryza sativa* L.) or maize (*Zea mays* L.) (Stubbs, 1988). Rye (*Secale cereale* L.) is only rarely infected (Stubbs, 1985). The species has been divided into more specialized categories, designated special forms or formae speciales. In 1894, Eriksson distinguished five formae speciales of *Puccinia glumarum* Ericks. et Henn., namely, f.sp. *tritici*, *hordei*, *elymi*, *secalis* and *agropyri* (Stubbs, 1985). Tollenaar and Houston (1967) criticised this subdivision system since they found that when uredospores collected off several different, naturally-infected wild grasses were cross-inoculated, they produced identical reaction types to those caused by uredospores collected from wheat. They concluded that, in California, the yellow rust occuring on *Agropyron* spp., *Bromus* spp., *Elymus* spp and *Hordeum* spp. all belonged to *P.* striiformis f.sp. *tritici*. By comparison, an isolate from Kentucky blue grass, *Poa pratensis* L., was distinguishable in that it only produced uredia on *Poa* spp. and had different physiologically

optimum temperatures than *P. striiformis* f.sp. *tritici*. However, the isolate *P. striiformis* f.sp. *poae* did not merit varietal rank. The designation of variety names to formae speciales requires that they also be morphologically distinct (Tolenaar, 1967). The differences in size of f.sp. *poae* uredospores were insufficient to provide the necessary distinctiveness, given that spore dimensions of *P. striiformis* are highly variable and governed as much by position on the host plant and host plant genus as by fungal genotype (Tolenaar, 1967). The stripe rust form infecting *Dactylis glomerata* L., however, was given varietal rank; *P. striiformis* var. *dactylidis* (Manners, 1960). The final, commonly acknowledged distinction at the formae speciales level is barley stripe rust, *P. striiformis* f.sp. *hordei* (Stubbs, 1985), which was relatively unknown in the Americas until the mid 1970's when it was apparently introduced by accident from Europe (Dubin and Stubbs, 1986) and spread to epidemic proportions on the barley crop. *P. striiformis* f.sp. *hordei* is specific to barley and is thus distinguishable from *P. striiformis* f.sp. *tritici*. In addition, there are consistent differences in isozyme and dsRNA (double-stranded RNA) phenotypes between f.sp. *tritici* and f.sp. *hordei*, indicating that these are two discrete populations (Newton *et al.*, 1985). However, because of the few cultivars susceptible to both formae speciales there is the possibility of an exchange of genetic material.

2.4.2 Physiological races

Authors have used various definitions for the terms 'pathogenicity', 'virulence' and 'aggressiveness'. The terms are used here in the sense defined by Green (1975). 'Pathogenicity' refers to the ability to cause disease on a host species. 'Virulence' is a relative term referring to the specific relationship between a host variety and a rust culture. It is commonly described as the pattern of infection of pathogen isolates on a set of host cultivars carrying different resistance genes. 'Aggressiveness' describes the relative ability of

pathogen isolates to cause different amounts of disease on a susceptible host. It includes those characteristics which contribute to vigorous pathogenic behaviour.

For the rust fungi, the term 'physiological races' refers to 'subdivisions within a formae speciales, differing in virulence'. By definition, a more appropriate term would be 'pathogenic races' (Browder, 1971). A pathogenic race is described by a specific combination of virulence on a defined set of differentials (Knott, 1989). The race may consist of a number of genotypes, and is subject to a potentially endless number of further subdivisions as supplemental differentials are added to the set. Genetic markers that distinguish one race from another are relatively rare. Newton *et al.* (1985) found no differences in isozyme phenotype among 29 diverse isolates of *P. striiformis* f.sp. *tritici*. However, subsequent studies (Dickinson and Pryor, 1989; Dickinson *et al.*, 1990) with different races of *P. striiformis* detected variation in dsRNA phenotype both between races and between isolates within races. The dsRNA of *P. striiformis* is probably contained in virus-like particles within the cytoplasm, as has been demonstrated for other rusts (Dickinson and Pryor, 1989; Lawrence *et al.*, 1988). These dsRNA phenotypes, however, appear to be independent of the nuclear encoded pathogenicity phenotypes (Dickinson *et al.*, 1990; Pryor *et al.*, 1990). The usefulness of 'races', as defined, has been questioned from a number of different perspectives. The level of information conveyed by a race designation is limited by the extent to which the genetic makeup of the differential set used is known (Browder, 1971). Vanderplank (1985) considered 'races' to be artifacts in that a race designation would change with the selection of differential cultivars. Individual genes for virulence, and not arbitrarily chosen gene combinations, are the segregating units of interest in a population (Browder, 1971; Day, 1974). In England, the physiological race 104E137 (as defined by the World and European sets of differential cultivars) was found to consist of four significantly different isolate types

(Johnson and Taylor, 1972; Priestley and Doling, 1974). The isolates were collected from varieties Joss Cambier and Champlein, and differed in aggressiveness as measured by the amount of pustule development on leaves at different growth stages following a point inoculation. Samples of Joss Cambier that were two generations advanced from Basic Seed were significantly more susceptible than Basic Seed to the most aggressive isolate PS4, possibly reflecting the fact that the isolate may have been naturally selected under field conditions for the ability to attack field populations of Joss Cambier (Priestley and Doling, 1974). However, until a more precise system of virulence formulae is agreed upon, the 'race' concept provides a means to track gross changes in virulence of a pathogen population (Knott, 1989).

2.5 ORIGINS OF PATHOGENIC VARIABILITY

In those fungi with a sexual stage, meiosis promotes genetic recombination through random assortment of chromosomes into haploid sets and through crossing over. However, in areas where the alternate host for a sexual cycle does not occur, or in the case of a hemiform rust like *P. striiformis* which has no known alternate host, there are other mechanisms to generate variability (Wellings and McIntosh, 1990).

2.5.1 Mutation

It is generally assumed that host genes for resistance in wheat are dominant while rust genes for virulence are recessive (Roelfs, 1988). Mutations from a dominant avirulence to a recessive virulent condition are expected to occur more frequently than vice versa in the pathogen (Knott, 1989; Ellingboe, 1982); for stem rust the rate of mutation to increased virulence has been estimated at 8.3×10^{-6} per uredinial generation at a locus

heterozygous for a dominant avirulence (Schafer and Roelfs, 1985). If the gene at that locus was homozygous for a dominant avirulence, a double mutation would be needed since the rusts are dikaryotic, so it's frequency would be of the order 6.9×10^{-11}. However rust uredospores are produced in vast quantities. The number of leaf rust uredospores produced by a hectare of wheat with 1% of the leaf area occupied by uredia is estimated at 10^{11} spores per day (Parlevliet and Zadoks, 1977). A daily production of 3,000-35,000 uredospores per cm^2 for infection types 3-9, respectively, has been reported for stripe rust (Young, 1977). A virulent spore landing on a resistant cultivar would have enough of a selective advantage over the avirulent majority to promote its rapid increase (Day, 1974). In their review of the history of yellow rust in Australia, Wellings and McIntosh (1990) demonstrated the sequential detection of races varying only in single attributes of virulence phenotype from a previously existing race. They proposed that the most plausible explanation for the origin of pathogenic variability in *P. striiformis* since its introduction to Australia in 1979, was mutation. Mutation was also used to explain changes observed in stem rust and leaf rust races in Australia where, in the absence of sexual recombination, dramatic changes in virulence have occurred through sequential single-gene mutations (Burdon *et al.*, 1982; Watson, 1981).

It is also possible to have mutations for virulence in a rust population where the virulence is not strictly necessary for the fungus' survival, i.e. there is no selection pressure from the growing of cultivars having the corresponding gene for resistance. In the North American *P. graminis* f.sp. *tritici* race group 15B-1L, virulence has evolved through a series of apparently random changes to virulence or avirulence rather than a progressive increase in virulence as found in Australia (Green, 1975). This difference was ascribed to a lack of host selection pressure. Pathotypes showing virulence on the host resistance genes Yr5, Yr8, and the unidentified resistance gene(s) in Spaldings Prolific, were detected in

Australia (Wellings & McIntosh, 1990), despite the fact that these resistance genes are not present in Australian wheats. Usually these pathotypes occur at low frequencies and may not show up in subsequent rust surveys, as was the case for the Australian isolate 360E137A+ with virulence for Yr5. Mutation may be an even more powerful source of pathogenic variation than first thought, if the mutations from avirulence to virulence do not occur independently. Given that the specificity in host-parasite systems is controlled by genes for avirulence and that virulence results from a loss of function (Damman, 1987; Ellingboe, 1979), then a single deletion mutation that affected more than one avirulence locus could result in simultaneous, dependent mutations from avirulence to virulence (Gabriel and Rolfe, 1990). The genetics of virulence has not been thoroughly explored in *P. striiformis*, but some of the work on other *Puccinia* species (Jones, 1988; Samborski and Dyck 1968; Mundt, 1990) would suggest that mutations to virulence at different loci are not necessarily independent.

2.5.2 Nuclear Reassortment

If mutation was the main source of variation in *P. striiformis* populations, then cultures of single races could reasonably be expected to produce mutations as frequently as those of mixed races, but this is not always the case. Cultures have been maintained for as long as 16 years without any change in virulence (Little and Manners, 1969a). For asexually-reproducing rusts there is another means of genetic recombination. When the uredospores of different clones of a fungus are mixed on a susceptible host, fusion of hyphae can occur followed by the reassortment of parent nuclei into recombinant dikaryons. Nuclear reassortment was first identified in stripe rust by Little and Manners (1969a), who produced two new races by inoculating two existing races on a cultivar susceptible to both, and making single spore cultures from the resulting infection. Although several genetical interpretations

of these results were considered, the mechanism considered most plausible was the reassortment of whole nuclei. Goddard (1976a) and Wright and Lennard (1980) also produced nuclear recombinants. Though the frequency of recombinants was surprisingly high, ranging from 6-10% in both experiments, only one recombinant type was isolated where two had been expected. Certain recombinants seem more likely to arise than others. Goddard (1976a), Taylor (1976) and Wright and Lennard (1980) all recorded the genesis of the same recombinant race 105E137 although thay started with different sets of parental biotypes. Although race 105E137 differed in virulence from the parental type 104E137 only on a single differential, it is unlikely to have arisen by mutation in the frequencies at which it was detected. Furthermore the parental isolate of 104E137 was a white spore mutant and, since all recombinant isolates were yellow-spored, a change in virulence would have to have been accompanied by a simultaneous change in the factor controlling uredospore colour (Wright and Lennard, 1980) showing that the changes were likely due to nuclear reassortment. Trinucleate heterokaryons were detected by Wright and Lennard (1980) at a 2% frequency, as well as by Goddard (1976a) but these proved unstable and dissociated quickly.

2.5.3 Parasexual Cycle

The parasexual cycle as described by Pontecorvo (1956) begins with the fusion of dissimilar nuclei to form diploids in the heterokaryon. Subsequently, mitotic recombination and haploidization create new races. In fungi the diploid condition is usually indicated by a larger nucleus, but this condition has never been reported in cytological studies of yellow rust (Goddard, 1976a; Little and Manners, 1969b). Until a diploid phase is

conclusively identified in *P. striiformis*, the existence of a parasexual cycle cannot be confirmed.

2.5.4 Cytoplasmic Inheritance

Just as different nuclei in a heterokaryon can be partitioned in separate uredospores during vegetative propogation, so may cytoplasmic determinants undergo reassortment. Goddard (1976b) reported the production of an isolate, MB-5, with spores that were significantly larger than either of the parent races. Since the nuclei of this race were found to be no larger than those of its parents, the possibility of it being a diploid stage was discarded. Although some of the stripe rust isolates showed variation in growth rate and germination, there is insufficient evidence to conclude the presence of extra-chromosomal inheritance. Recent work (Dickinson *et al.*, 1990) has detected differences in the cytoplasmic double-stranded RNA phenotypes within the *P. striiformis* population, but this variation does not appear to be connected to variation in virulence.

2.6 EFFECT OF ENVIRONMENT

Stripe rust is more strongly influenced by the environment than any other cereal rust. Spore germination is reduced by increasing concentrations of naturally occurring, intermediate-sized (mobility 0.03 - 0.274 cm^2 $volt^{-1}$ sec^{-1}) atmospheric ions (Sharp, 1967). The data were calculated on the basis of negative ion concentrations, but positive ions were reported to be at a similar concentration. Any meteorological factors which enhance the accumulation of atmospheric pollutants will exacerbate this effect. However, stripe rust cultures from northwest Europe have a higher frequency of spore germination when exposed

to atmospheric pollution than do cultures from the Andes, East Africa or Asia, indicating that adaptation to pollution is possible (Stubbs, 1985).

The most critical factor is temperature, both the temperature in which the host plant was growing prior to inoculation as well as the post-inoculation and incubation temperatures. Sharp (1965) reported that the wheat variety Rego exhibited a resistant (Type 0) infection type when grown at 2/18°C, but a susceptible (Type 3) reaction when grown under a 15/24°C regime. When seedlings were transferred from the low to the high temperature regime at different times before and after inoculation, varietal differences in infection type were apparent. Electrophoretic analyses of plants grown at both temperature regimes (Strobel and Sharp, 1965) showed that the variety Rego resolved at least two protein bands, B and C, when it was susceptible (15/24°C) that were not produced when expressing resistance (2/18°C). The same was true for variety Nord Deprez, which behaves similarly to Rego in its reaction to the fungus at different temperatures. The proteins were suggested to have been formed as a result of the activation of certain genes by the specific temperature profile in which the plants were growing. They could be involved in the production of compounds necessary for the growth of *P. striiformis*, or perhaps be acting to destroy compounds otherwise inhibitory to the fungus (Strobel and Sharp, 1965). Another explanation is based on the theory that a host plant produces a gene product which reacts with a corresponding gene product from the pathogen to determine an incompatible reaction (Ellingboe, 1979). This gene product could be a protein that functions normally at 2/18°C but is ineffective at 15/24°C. The protein bands isolated from susceptible Rego could be breakdown products of the denaturation process. Notwithstanding these results, the general tendency in most stripe rust race-differential combinations is for decreased compatibility (and reduced spore production) with increasing temperatures between 7°C and 24°C (McGregor and

Manners, 1985; Sharp, 1965; Stubbs, 1980; Zadoks, 1961). The most commonly used temperature regime for the artificial culture of stripe rust is 15°C ± 3°C (Rapilly, 1979; Zadoks, 1961), except in the 48 hours after inoculation when the optimum temperature for infection to occur is between 7°C and 10°C (Sharp, 1965; Dennis, 1987). The use of a diurnal temperature profile in greenhouse screenings to simulate natural diurnal fluctuations has been shown to encourage susceptible reactions (Brown and Sharp, 1969).

Relative humidity (RH) is usually maintained around 80% except in the 48 hours after inoculation, when an RH of 100% is critical for infection (Zadoks, 1961). Free water on a lesion will prevent sporulation (Zadoks, 1961).

The effect of light on the development of stripe rust symptoms has been investigated primarily in growth cabinets under controlled conditions. Thus, it is often possible to make a distinction between the effects of light intensity and photoperiod. Bever (1934), working with the stripe rust susceptible barley variety Pannier, observed no difference in the expression of infection type between a 6-hour and a 12-hour daylength period, although the shorter day increased the incubation period by 9 days. However, when the daylength exceeded 12 hours there was a definite decrease in infection type from a susceptible type 4 to a resistant type 0. This observation was confirmed in wheat by Manners (1950) who also reported that lengthening the duration of light, from 12 to 18 hours/day, increased resistance in both Holzapfels Fruh and Carstens Dickkopf V.

There have been conflicting reports about the effect of light intensity on infection type. Reducing light intensity reduced total spore production in the cultivars Maris Beacon, Maris Huntsman and Maris Nimrod, due to reductions in both pustule density and spore production per pustule (McGregor and Manners, 1985). However, reducing light intensities had little effect on the infection type except for a reduction at the very low light

levels (less than 5 Watts/m^2). Higher light intensities can shorten the incubation period of the rust (Bever, 1934) and increase sporulation rates (McGregor and Manners, 1985). Conversely, other studies have indicated that low light intensities encourage susceptible reactions while high light intensities produce resistant reactions (Manners, 1950; Stubbs, 1967; 1980). The mycelium density tends to decrease with higher light intensities which results in fewer sporulating lesions. It has been speculated that the quality, as well as the intensity and duration of light, is probably important (Manners, 1950). The genetic basis of resistance could also be important. Phenotypes conferred by the YrA resistance vary when exposed to different light intensities in the post-inoculation phase (Wellings *et al.*, 1988).

A study investigating the performance of stripe rust under natural conditions at different locations in the world (Stubbs, 1980), also concluded that the observed change from susceptibility to resistance had been induced by a recorded increase in radiation, possibly in combination with relatively high nighttime temperatures. Stripe rust spores have been found to be three times more susceptible to ultraviolet radiation than stem rust spores (Maddison and Manners, 1972) so it is possible that the quality of natural light might have been less than ideal for infection. In experiments with forage grasses, Deinum (1966) found that higher light intensities decreased the nitrate content in plants and increased the water-soluble-carbohydrate content, under both greenhouse and field conditions. If this relationship holds true for wheat as well, and given that stripe rust is an obligate parasite, the nutritional status of a host plant might be sufficiently compromised by higher light intensities to explain the reduced rust infection.

2.7 INHERITANCE OF RESISTANCE

2.7.1 Gene-for-Gene Hypothesis

Flor (1942) was the first to study the genetics of virulence in a pathogen, in conjunction with the inheritance of resistance in the host. From his observations on flax rust (*Melampsora lini* Desm.) and its host flax (*Linum usitatissimum* L.), he noted that for every gene conditioning resistance in the host, there was a corresponding gene for virulence in the pathogen. The two genotypes function together to produce the phenotype of the association. Thus a gene for resistance in the host has no selective advantage in the absence of the corresponding gene for avirulence in the pathogen. In the case of the cereal rusts, resistance and avirulence are commonly (though by no means exclusively) dominant conditions, which suggests that they are determined by active gene products (Ellingboe, 1982). It is generally believed that the fungal avirulence allele determines a product which betrays the pathogen's presence to the host whose recognition facility is conferred by the corresponding gene for resistance i.e. a recognition for resistance or incompatibility (Browder and Eversmeyer, 1986; Damann, 1987; Ellingboe, 1982). Vanderplank (1984) proposed a theory of recognition for susceptibility, based on his observation that a genotype possessing a dominant resistance gene (R) can only be parasitized by a pathogen having the corresponding virulence gene (v). However, this theory does not account for the susceptible reactions observed in two other host-parasite combinations, rrVV and rrvv. A gene-for-gene relationship has been demonstrated in several host-parasite systems (Day, 1974 p. 96-97).

Direct proof for the existence of a gene-for-gene relationship is usually obtained by making crosses between isolates of the pathogen, but since a sexual cycle has not been discovered for *P. striiformis*, such a system can only be hypothesised (Lewellen *et al.*, 1967; Line *et al.*, 1970; Zadoks, 1961).

Table 1. A quadratic check showing a gene-for-gene interaction between a resistance gene (R) and its corresponding avirulence gene (V) in the pathogen.

		Pathogen genotype	
		V_	vv
Host genotype	R_	I	C
	rr	C	C

I = incompatible reaction /resistance
C = compatible reaction / susceptibility

Based on the gene-for-gene theory, Loegering *et al.* (1971) developed a system for analyzing sets of infection type data and postulating genotypes of both host and pathogen. As diagrammed above (Table 1), for a single pair of corresponding genes with resistance dominant and virulence recessive, there are four possible interactions; R_/V_ = I, R_/vv = C, rr/V_ = C and rr/vv = C. Whenever a compatible reaction occurs it means that the host/pathogen combination must be R_/V_; this is referred to by Loegering as the definitive phenotype. Other, slightly different methods for processing infection type data have been developed to identify a resistance gene (Browder and Eversmeyer, 1980) and are summarised in Knott (1989 pp. 91-95). Genetic analyses based on infection type data are usually supported by additional information such as the pedigrees of the genotypes being studied (Dubin *et al.*, 1989; Vallavieille-Pope *et al.*, 1990). However, care must be taken in the interpretation of infection type data, as illustrated in an example by Johnson *et al.* (1987). They re-examined data from Kirmani *et al.* (1984) in which 40 *Puccinia striiformis* races of diverse origin were used to produce specific infection types on 11 cultivars of unknown genotype. Because all of the races which lacked virulence for Yr8 were also avirulent on the 11 unknown cultivars, the authors concluded that all 11 cultivars carried Yr8. This conclusion was shown to be extremely unlikely in subsequent tests with different races, and

by examination of the varietal pedigrees. The resolving power of such analyses is limited by the range of variation represented in the set of rust isolates used in the test. One example is the difficulty in distinguishing between the resistances at the Yr3 and Yr4 loci. Most races of *P. striiformis* are characterized on the World and European sets of differentials, in which Vilmorin 23 contains both Yr3a and Yr4a, and Hybrid 46 contains both Yr3b and Yr4b respectively. The presence of either one of the genes in these pairs can only be unambiguously shown in tests with a race possessing virulence to one but not the other Yr gene of the pair. In tests with 19 different isolates of yellow rust on bread wheat varieties from the Ethiopian breeding programme, Badebo *et al.* (1990) postulated that the Kenyan variety, Kenya Kulungu (= Har 472), had the gene composition Yr4b+ (as represented by the differential Hybrid 46) with or without Yr2 (present in Heines VII) and/or Yr3 (present in Vilmorin 23). The + signs indicate the presence of other, perhaps unknown, genes for resistance in the differentials named. The presence or absence of Yr2 and/or Yr3 could not be proven because all races virulent on Yr4+ were also virulent on those genes.

Another drawback associated with using infection-type data to postulate resistance genes is that the expression of an infection type can be quite variable. Temperature may influence some infection types perhaps by affecting the expression of a particular gene (Dubin *et al.*, 1989; Vallavielle-Pope *et al.*, 1990). The genetic background in which a gene is expressed can also have an effect on the infection type (Dyck and Samborski, 1968; Labrum, 1980; Johnson, 1981). When a cultivar has several genes for resistance to a specific disease it is usually assumed that each acts independently, but this is not always the case. Interactions between genes can produce misleading infection types or mask the presence of resistance genes. To cite just a few examples; Dyck and Samborski (1982) reported complementary gene action between LrT2 and LrT3 conferring leaf rust resistance to several

breadwheat accessions; a residual expression of mild resistance can be conferred by 'defeated' stem rust genes Sr6, Sr8, and Sr9a when confronted by virulent races of stem rust (Brodny *et al.*, 1986); the variety Thatcher reportedly possesses a dominant inhibitor of the Yr5 stripe rust resistance in *T. spelta* (Wolfe, 1984). The varieties PI 178383 and Chinese 166 were each found to have an incompletely dominant major gene for resistance to stripe rust, as well as an undetermined number of minor genes that modified the action of the major genes (Lewellen *et al.*, 1967).

In addition, unrecognized virulence in the races used could confuse the interpretation of results based on known genes for resistance, as suggested by Vallavielle-Pope *et al.*, (1990) to explain the unlikely gene postulations for the variety Darius. It should be noted, though, that difficulties of undetected variation in the races used can also lead to misinterpretation of data from standard genetic studies. The precise identification of genes for race-specific resistance requires crosses and genetic analysis based on the classification of segregating generations into phenotypes, based on the reaction of plants to specific pathogen races (Dyck and Kerber, 1985).

2.7.2 Major Gene Resistance

Stripe rust was the first disease in which host resistance was shown to be inherited in a Mendelian manner, by Biffen in 1906 (Roelfs and Bushnell, 1985). Following Biffen's report, much work was done on stripe rust resistance in wheat but it was not until much later that the formal nomenclature of Yr genes was proposed by Lupton and Macer (1962). A list of gene designations and the varieties they occur in, is updated at regular intervals (McIntosh, 1988).

Two types of specific resistance to stripe rust have been recognized (Zadoks, 1961). The first type of resistance is evident at the seedling stage and lasts for the life of the plant, while the second type is apparent only in the adult plant. In certain wheat-growing areas, the host plant can show morphological differences in resistance between the leaves and the head (Stubbs, 1980). This condition appears to be an environmental effect, possibly caused by variations in light, and its genetic control has yet to be studied. Eleven seedling resistance factors have been genetically analyzed and are designated genes. In an evaluation of F1, F2, BCF1 and F3 progeny from intervarietal crosses between six lines, using 4 races of stripe rust, Lupton and Macer (1962) identified the genes Yr1 (in Chinese 166), Yr2 (in Heines VII), Yr3 and Yr4 (in Capelle-Deprez and Hybrid 46). A subsequent study (Macer, 1966) identified three more genes, Yr5 (in *Triticum spelta* L.), Yr6 (in Heines Peko) and Yr7 (in Thatcher). Each of these loci had only one allele except Yr3 and Yr4 which had three and two resistance-determining alleles, respectively (Macer, 1972). The gene Yr8 was transferred to wheat chromosome 2D from chromosome 2M of *Aegilops comosa* Sibth. and Sm., by genetically induced homeologous recombination (Riley *et al.*, 1968), to form the interspecific hybrid cultivar Compair. This was achieved through an intermediate 'bridging cross' between *Triticum aestivum* L. em. Thell. and *Aegilops speltoides* Tausch. The *A. speltoides* genotype inhibits the Ph1 locus on chromosome 5BL in wheat hybrids. Consequently, the genetic suppression of homoeologous recombination normally exercised by the Ph1 gene in the wheat genome was circumvented. By deduction from differing resistance patterns, Macer (1975) identified the resistance gene Yr9 and Yr10 in Riebesel 47/51 and Moro, respectively. The gene Yr9 was found to be on the wheat-rye translocated chromosome 1BL/1RS (Mettin *et al.* 1973; Riley and Macer, 1966; Zeller, 1973). This gene was closely linked to the genes controlling resistance to leaf rust (Lr26), stem rust (Sr31) and to W-secalins (Sec1), which

controls the deleterious trait of dough stickiness. All four genes are located on a very small segment of chromatin on the short arm of rye chromosome 1R (Singh *et al.*, 1990). The gene Yr10 was found to be associated with brown glume colour in PI 178383 (Metzger and Silbaugh, 1970). The gene Yr15 is derived from *Triticum dicoccoides* Koern. var. *aaronsohni* and confers a seedling resistance that is effective over a wide range of stripe rust races (Gerechter-Amitai *et al.*, 1989).

Table 2.
The identified genes for stripe rust resistance (reviewed in Knott, 1989)

Gene	Common source	Infection type	Chromosome location
Yr1	Chinese 166, Heines 110, Maris Templar	00	2A
Yr2	Heines VII, Soissonais-Deprez, Hustler	0-2	7B
Yr3a	Cappelle-Deprez, Hobbit, Maris Templar	0-1+	-
Yr3b	Hybrid 46	0-1+	-
Yr3c	Minister	0-1+	-
Yr4a	Cappelle-Deprez, Hobbit, Maris Templar	0-1+	-
Yr4b	Hybrid 46	0-1+	-
Yr5	*Triticum spelta* A.	00-0	2BL
Yr6	Heines Kolben, Peko	0-2+	7BS
Yr7	Thatcher, Lee, Reichersberg 42	0N-1+	2BL
Yr8	Compair (from *Aegilops comosa*)	00	2D
Yr9	1BL/1RS wheat-rye translocations, Veery	00-0	1BL/1RS
Yr10	Moro, PI 178383	00	1BS
Yr11	Joss Cambier	A	-
Yr12	Mega, Armada	A	-
Yr13	Maris Huntsman, Maris Nimrod, Hustler	A	-
Yr14	Maris Bilbo, Hobbit, Avalon	A	-
Yr15	Hexaploid derivatives of *T. diccocoides* G-25	00	1B*
Yr16	Cappelle-Deprez	A	2DS

A = adult plant resistance
* R.A. McIntosh personal communication, unpublished

The resistant alleles of Yr1, Yr2, Yr5, Yr6, Yr7, Yr8, Yr9 and Yr10 are usually dominant although Yr2 and Yr6 react recessively in some cases (Labrum, 1980; Macer, 1966). Allelic series have been proposed at the Yr3 and Yr4 loci (Lupton and Macer, 1962). The cultivar Cappelle Desprez was postulated to carry Yr3a and a recessive allele Yr4a, while two dominant alleles Yr3b and Yr4b were present in Hybrid 46 (Lupton and Macer, 1962). Resistance in the cultivar Minister was postulated to be due to the allele Yr3c

(Lupton and Macer, 1962), but this has been questioned (Knott, 1989) in light of the fact that susceptible F2 progeny were recorded from crosses between Hybrid 46 and Minister.

Four race-specific factors for adult-plant resistance to stripe rust in British varieties have been described (Taylor *et al.*, 1981) and designated (McIntosh, 1988); Yr11 in Joss Cambier, Yr12 in Mega, Yr13 in Maris Huntsman and Yr14 in Hobbit. Through the development of homozygous recombinant lines between the wheat variety Capelle-Desprez and a substitution line for chromosome 2D, the gene Yr16 for adult-plant resistance was identified (Worland and Law, 1986). Because of the supposed homoeology between chromosomes 2BL and 2DS, on which are located genes Yr5/Yr7 and Yr16 respectively, a possible commonality of action between the two homoeologous resistances was suggested (Worland and Law, 1986).

Many other examples of race-specific resistance to stripe rust have been observed and may be controlled by genes other than those already designated. In western Europe some of these resistance factors are named after the reference varieties in which they were found (Stubbs, 1985) e.g. Strubes Dickkopf (SD), Suwon 92/Omar (Su), Carstens V (CV) and Spaldings Prolific (Spa). Similarly, the resistance factor designated YrA was named after Avocet, the Australian cultivar in which it was first identified (Wellings *et al.*, 1988).

2.7.3 Minor Gene Resistance

In addition to the major gene resistance described above, there have been numerous reports of minor genes conferring noticeable levels of resistance. The presence of an undetermined number of minor genes has been reported in wheat varieties grown in the US Pacific Northwest (Henriksen and Pope, 1971; Lewellen *et al.*, 1967; Milus and Line, 1986) and Europe (Hyde and Elahinia, 1989; Wallwork and Johnson, 1984) and in *T. dicoccoides* (Gerechter-Amitai and van Silfhout, 1989; Grama *et al.*, 1984). In general the resistance

conditioned by these minor genes behaves either as a partially (Milus and Line, 1986) or completely recessive trait (Krupinsky and Sharp, 1978; Wallwork and Johnson, 1984). They also commonly display an additive gene action, expressed in the form of transgressive segregation for resistance (Grama *et al.*, 1984; Lewellen *et al.*, 1967; Milus and Line, 1986; Pope, 1968; Wallwork and Johnson, 1984).

Most minor genes for stripe rust resistance in wheat are temperature-sensitive, frequently conditioning an increased resistance with temperatures above 15°C (Gerechter-Amitai and van Silfhout, 1989; Lewellen *et al.*, 1967; Milus and Line, 1986). However, the reverse effect has also been reported (Brown and Sharp, 1969). Grama *et al.* (1984), working with *T. dicoccoides*, reported that additive resistance detected at the high temperature profile of 15/24°C was also expressed at 15°C. In a follow-up experiment, out of 24 lines of *T. dicoccoides* possessing temperature-sensitive minor genes, three entries showed a shift towards increased resistance at higher temperatures (15/24°C) with some isolates but towards susceptibility with others (Gerechter-Amitai and van Silfhout, 1989). The authors suggested the possibility of two different kinds of temperature-sensitive genes in these entries, supporting the opinions of Robbelen and Sharp (1978) who commented that "differences in sensitivity to temperature [were] due to the diverse physiological functions of the minor genes concerned". The physiology of the response conditioned by these minor genes to stripe rust closely resembles that of several other host-rust combinations (Mares, 1979) but the exact mode of action of each of the individual resistance genes has yet to be elaborated.

Factors affecting the rate of disease progression may operate before or after penetration of the leaf. Reduced spore deposition because of the waxiness of a leaf or steeper leaf angle, and reduced spore germination have been suggested as possible components of a disease-escape tendency (Russell, 1976; 1978). However, the majority of factors probably

operate after penetration and these could involve effects on spore receptivity (the production of fewer lesions), latent period, spore production and the length of the infectious period (Parlevliet, 1985). In the cultivars Little Joss and Nord Desprez, a non-hypersensitive cell browning response, rather than cell collapse, severely hampers the pathogen's ability to produce a normal pustule (Cartwright and Russell, 1980). The cultivar Nord Desprez, exhibited a moderately resistant reaction at temperatures of 20-24°C against a race to which it was susceptible at lower temperatures (Cartwright and Russell, 1980). Since Nord Desprez is thought to possess minor genes for resistance (Wallwork and Johnson, 1984), this may indicate that some minor genes act in a non-hypersensitive manner. Treatment of adult plants of these cultivars with KCl or sucrose solutions (through the roots and leaves, respectively) tended to increase the natural incidence of this mode of resistance suggesting, perhaps, the alteration of osmotic equilibria of host and fungal cells (Cartwright and Russell, 1980).

The question of whether minor genes for resistance to stripe rust are race-specific or non race-specific, is also contentious. The common assumption is that resistance conferred by minor genes is non race-specific (Line, 1980; Quayoum and Line, 1985; Robbelen and Sharp, 1978; Sharp and Volin, 1970) but this assumption can never be conclusively proven since it would hold true for any resistance factor until new virulent races appear. From crosses between Joss Cambier x Nord Desprez that showed transgressive segregation for resistance, Wallwork and Johnson (1984) discovered a previously unreported, race-specific component of the adult plant resistance in Nord Desprez. One of the minor genetic components of resistance in Joss Cambier, affecting fungal sporulation, was shown to be race-specific as well (Johnson and Taylor, 1972). Differences were observed in aggressiveness of isolates of *P. striiformis* on field populations of Joss Cambier (Priestley and Doling, 1974). This was attributed to the erosion of that variety's adult plant resistance after

several generations of natural selection under field conditions, and further indicated race-specificity. Gerechter-Amitai and van Silfhout (1989) tested 24 accessions of *T. dicoccoides* with 15 isolates of *P. striiformis* for the occurrence of significant shifts in resistance from one temperature regime to another. They reported that many of the temperature-sensitive, minor genes were race-specific, as shown by differential reactions to the isolates within accessions. The considerable variation in the magnitude of these shifts was postulated to indicate the presence of two or more 'minor-effect' genes, each reacting specifically to the different test races. Nonetheless, the fact that some minor genes can be overcome by certain races does not completely invalidate the possible existence of non race-specific components of minor gene resistance.

2.8 GENE IDENTIFICATION

In identifying a single, unknown, race-specific gene for resistance, some useful information can be obtained by testing the line with a number of races (Dubin *et al.*, 1989; Vallavielle-Pope *et al.*, 1990). The infection type can then be compared with those of a set of lines carrying known genes of resistance. However, this procedure has a number of limitations in the stripe rust-*Puccinia striiformis* system, as described previously.
Some alternative procedures include:

1. The line carrying the unknown gene can be crossed with as complete a set as possible of lines carrying the known genes for resistance (a differential series)(Lupton and Macer, 1962). The F2 progeny from a cross between two resistant lines is then tested with a race to which both parents are resistant (Singh and Johnson, 1989). If the cross segregates producing susceptible plants, then the lines have no effective resistance genes to that race in common; if there are no susceptible F2 progeny, and provided the number of plants tested is

large (i.e exceeding 400), then the resistant gene in the unknown is either the same as, allelic or tightly linked to the gene in the tester (Dyck *et al.*, 1985).

A test for allelism involves inoculating progeny from crosses between the unknown cultivar and a standard cultivar, with two different races. The two races should separately possess virulence for each one of the resistance genes in the F1 heterozygote (Flor, 1965). If resistance in the unknown cultivar is allelic to that in the standard, then the single-virulence races will produce 3:1 F2 ratios, with none of the individual plants susceptible to one race being susceptible to the other as well. The test of mutual exclusivity of resistance alleles is to obtain recombinant progeny. A very large sample of F2 plants is required to distinguish a tight linkage since the frequency of crossing-over is low (Hanson, 1959).

2. Another method for studying an unknown gene is to conduct a cytogenetic analysis using monosomic lines (Macer, 1966). The progeny should be tested with a race to which the monosomic lines are susceptible, and the cultivar with the unknown gene is the only one bearing resistance in order to simplify the expected ratios. For a dominant resistance, F2 populations grown from monosomic F1 plants will segregate in a 3:1 ratio except in the critical family which will have no (or very few) susceptible plants (Kush, 1973). Once the unknown resistance has been located, tests can be done to determine possible linkage relationships with genes controlling other traits that are known to be located on that chromosome (Singh *et al.*, 1990b).

3. Molecular genetic techniques can also be used to help locate an unknown gene. Some of the techniques include the use of RFLPs (Young *et al.*, 1988), isozyme markers (Gale and Sharp, 1988) or bulked segregant analysis (Michelmore *et al.*, 1991). Major genes can also be cloned for further study (Ellis *et al.*, 1988).

2.9 DIFFERENTIAL SERIES

Sets of near-isogenic lines carrying individual genes at most of the known loci are already available for leaf and stem rust. The situation for stripe rust is different in that the differential sets are not near-isogenic lines but consist of collections of varieties established by convention and reviewed periodically (Zadoks, 1961). The first consistent system of differentials was initiated at a meeting of the foremost European stripe rust research scientists (Johnson *et al.* 1972) with the aim of developing a universally understood nomenclature for stripe rust races. A set of differential varieties was selected, placed in a fixed linear order, and then assigned decanery values that correspond to an ascending binary notation (Table 2). The sum decanery value of all the varieties in the set which are susceptible to any given race is used as the race designation. A primary set of varieties was selected to test the broad characteristics of virulence over the world, and subsidiary sets were proposed to better describe the variation in virulence within a region. Rust populations can differ substantially from one part of the world to the next, and it is of use to breeders to have specific information about rust virulence on key varieties grown in their area. The prefix E was chosen for the number derived from the European set.

Table 3.
List of differential varieties selected for the World and European sets, with their decanery and binary values (Johnson *et al.* 1972)

Decanery value	Binary value	World varieties	European varieties
1	1	Chinese 166	Hybrid 46
2	10	Lee	Reichersberg 42
4	100	Heines Kolben	Heines Peko
8	1000	Vilmorin 23	Nord Desprez
16	10000	Moro	Compair
32	100000	Strubes Dickkopf	Carstens V
64	1000000	Suwon 92/ Omar	Spaldings Prolific
128	10000000	—	Heines VII

Both differential sets are open-ended to allow for the addition of differential varieties as new virulences arise in the pathogen population or new resistance genes are identified. The variety Riebesel 47-51 which possesses the resistance gene Yr9 was subsequently added to the world set (Stubbs, 1985). Standard growth room conditions have been suggested for the testing of virulence formulae; in Europe, 16 h. light period at 18°C and 8 h. dark period at 11°C and in North America, 12 h. light period at 18°C and 12 h. dark period at 2°C to better reflect the indigenous conditions (Johnson *et al.*, 1972). Line *et al.* (1970) proposed a North American set of numbered differentials for the United States (Table 3.), and an accompanying system of classifying races using a code that lists the numbers of those differentials the race is avirulent on, followed by a list of numbers for the differentials the race is virulent on. The two number strings are listed consecutively and separated with a slash (/) e.g. the race PNW-3 is coded 2,5,6,7/1,3,4.

Table 4.
Differential cultivars for identifying races of *Puccinia striiformis* in the United States (adapted from Line *et al.*, 1970)

Differential No.	Cultivar	Subsequent additions (Line *et al.*, 1988)
1	Lemhi	Fielder
2	Chinese 166	Produra
3	Heines VII	Yamhill
4	Moro	Stephens
5	Paha	Lee
6	Druchamp	Tyee
7	Riebesel 47-51	

There are presently regional sets of differentials for India, China and Australia as well (Stubbs, 1985 p.75-80).

All varieties in the differential sets were originally selected for consistency of their differential response, but subsequent studies using different 'foreign' races have revealed an array of resistant genes in the background of some differential varieties. The cultivar Heines Peko was known to possess Yr6 from its parent Heines Kolben but it has been speculated that it probably contains Yr2 as well, though in a genetic background that inhibits its expression in seedlings (Johnson *et al.*, 1986). The variable expression of Yr2 in different genetic backgrounds has previously been reported (Labrum, 1980; Singh *et al.*, 1990a). Likewise, in a study using a foreign race 6E16 and the Indian variety Kalyansona, Singh and Johnson (1989) concluded that the differential variety Heines VII possessed an unidentified gene for intermediate resistance to stripe rust, in addition to Yr2. There have been similar suggestions of multiple genes for resistance in *T. spelta*, Moro, Riebesel 47-51 (Luthra *et al.*, 1989) and Lovrin 13 (Liu, 1988). When tested with a Kenyan race, Riebesel 47-51 was resistant and Clement was susceptible although both carry Yr9 (Vallavielle-Pope and Line, 1990).

The ideal differentials would be a set of near-isogenic lines each carrying a single gene of resistance. Griffey and Allan (1986) produced 21 families of backcross-derived Lemhi 53 near-isogenic lines, representing eight different genes or alleles for resistance. They reported differences in yield potential (based on mean yields of triadimefon protected plots) and percent yield loss both among and within families. However, many of the lines had resistance genes in common and some had adult plant resistance as well (Griffey and Allan, 1988). Tests to further elucidate the genetic basis of resistance in these lines would have to be conducted before they can be used effectively in a genetic study.

2.10 GEOGRAPHICAL DISTRIBUTION OF PATHOGENICITY

The proposed centre of origin for the cultivated wheats is in the Transcaucasian mountain region of the Middle East. This area contains the greatest number of physiologic races of *P. striiformis*, presumably since both host and parasite have long been associated as reciprocal selective factors in evolution (Leppik, 1970).

Elsewhere in the world, stripe rust has evolved in different directions. A new gene for virulence in one region may be spread to other regions by dispersal of uredospores, either naturally or through human intervention. Stripe rust in Australia was first detected in 1979 (race 104E137) and is thought to have been an unintentional introduction from Europe, where this race is common (Wellings and McIntosh, 1990). It is also possible for the same virulence to arise independently in more than one region, as was the case for the emergence of virulence on Yr10 in both the eastern Mediterranean region and in North America (Stubbs, 1985).

Virulence on Chinese 166 (Yr1) occurs worldwide, and is especially common in the cultivar's Asian centre of origin (Stubbs, 1985) and in Europe, where Chinese 166 was

a commonly used source of resistance in early breeding programmes (Robbelen and Sharp, 1978). The frequent use of Heines VII (Yr2), Vilmorin 23 (Yr3a, Yr4a) and Hybrid 46 (Yr3b, Yr4b) as sources of resistance worldwide (Lupton and Macer, 1962; Stubbs, 1988; Zadoks, 1961), has resulted in the common occurrence of virulence for these genes. Virulence for *T. spelta* (Yr5) is still rare, and but has been reported at very low frequencies in India (Stubbs, 1985) and Australia (Wellings and McIntosh, 1990). The genes Yr6 (Heines Kolben) and Yr7 (Thatcher) have been in regular use for many years (Robbelen and Sharp, 1978). This probably explains the global distribution of corresponding virulences. In the past, races of southwestern Europe were characterized by an absence of Yr7 virulence, although this is no longer the case since the arrival of race 6E16 from northwestern Africa (Stubbs, 1988). Yr7 is believed to have originated from Iumillo durum and is tightly linked to Sr9g for resistance to stem rust (McIntosh *et al.*, 1981). More recently, Yr7 has been spread around the world in the form of the cultivar Thatcher which is used in many breeding programmes (Stubbs, 1985). The parental source for the gene Yr8, *Ae. comosa*, is native in the Middle East. Consequently, stripe rust virulence on Compair (Yr8) arose rapidly in that area and spread into Africa (Badebo *et al.*, 1990; Prescott *et al.*, 1985; Stubbs, 1985). Virulence for Yr9 (obtained in a wheat-rye cross through a 1B/1R translocation) (Mettin *et al.*, 1973) seems to arise relatively quickly in the presence of the corresponding gene for resistance. In South America this virulence was detected in a trap nursery before any known use of the resistance in commercial cultivars (Stubbs, 1988). Virulence for Yr9 has been detected in East Africa (Badebo *et al.*, 1990; Stubbs, 1985). The resistance Yr10 was derived from PI 178383 of Turkish origin and subsequently incorporated into the American cultivar Moro (Lewellen *et al.*, 1967). The corresponding virulence is indigenous to the so-called Levantine zone of the eastern Mediterranean, but absent elsewhere in Europe (Stubbs, 1988).

In America, an independently arising virulence was detected shortly after the gene Yr10 was introduced into commercial cultivars (Beaver and Powelson, 1969).

In Kenya the evolution of virulence in relation to host resistance has been studied by Bonthuis (1985). He identified a zone within the wheat-growing area where the conditions of temperature and moisture allowed for a permanent reservoir of stripe rust, encouraged by a continuous wheat cropping system. These reservoirs were postulated to be the source for local evolution of virulence within the rust population. However, the possibility of outside introduction cannot be ruled out particularly when the ITCZ (Inter-Tropical Convergence Zone) is south of the Equator and the prevailing winds come from Ethiopia (Grace and Lyamchai, 1991). In Ethiopia, races carrying virulence for Yr1, Yr2, Yr3a, Yr4a, Yr6, Yr7, Yr8, Yr9, Yr10 and/or YrA have been identified (Badebo *et al.*, 1990). Although that study did not identify virulence for the gene Yr4b (in Hybrid 46), it has been noted to occur at a low frequency in the East Mediterranean and East Africa zone (Stubbs, 1985). Already in Eastern and Central Africa quite complex races occur that combine virulence for all of the above resistance genes (Badebo *et al.*, 1990).

Stripe rust probably entered the American continent by way of the Aleutians and Alaska (Stubbs, 1988). This formae specialis of *P. striiformis* attacks wheat and barley in the United States and Canada, primarily in the cooler growing areas which includes regions of Idaho, Oregon, Washington and western Montana. In Canada it is most commonly found in Alberta and British Columbia (Conners, 1967; Sanford and Broadfoot, 1933). Virulences for Yr1, Yr2, Yr3a, Yr4a, Yr6, Yr7 and Suwon 92/Omar are present in both North American and European races (Stubbs, 1985; Vallavielle-Pope and Line, 1990). Virulence for Yr3b and Yr4b (Hybrid 46) is uncommon in North American races, while the virulence on Moro (Yr10) is well established in N. America but has not been detected in Europe. Virulence is

either absent or very rare in North America for Yr5, Yr8 and Yr9 (Vallavielle-Pope and Line, 1990). Because of the extreme winter temperatures in Canada, stripe rust seldom survives and infections in the spring are usually initiated by inoculum from the Pacific Northwest of the United States. However, the fungus is capable of enduring a mild winter as mycelium in a fall-infected, winter wheat crop (Conner *et al.*, 1988; Sanford and Broadfoot, 1933). Newton and Johnson (1936) identified only two 'physiologic forms' (8 and 13) in all the samples they collected off cultivated cereals and wild grasses in Canada, one of which (13) was far more prevalent than the other.

2.11 MANAGING GENETIC DIVERSITY TO CONTROL RUSTS

2.11.1 Single Gene Resistance

The resistant alleles of most genes for specific stripe rust resistance are often dominant (Lupton and Macer, 1962; Macer, 1966) and can consequently be manipulated fairly easily. The transfer of a monogenic resistance is most efficiently achieved by backcrossing (Briggs and Allard, 1953). It is important to select a recurrent parent that will not become obsolete over the time it takes to complete a backcross programme (Briggs and Allard,1953). However, if the donor parent is reasonably well adapted, even material having 75% or 87.5% of the recurrent parent's genes (i.e after fewer backcrosses) in addition to rust resistance would be an improvement over a rust-susceptible line (Knott, 1989). The most serious disadvantage of single gene resistance is its tendency to destabilize pathogen populations (Parlevliet, 1981). If a host crop is immune to stripe rust, a tremendous selection pressure is imposed on the pathogen to adapt (Robbelen and Sharp, 1978). As well, any virulent spore will survive unchallenged and, presumably, multiply proportionately. Once the latest resistance gene is defeated, breeders often replace it with another single gene and the cycle

continues. In New Zealand, the original stripe rust races were avirulent to Yr7 but virulence developed within two years (Welling and McIntosh, 1990). A similarly rapid breakdown of resistance occurred for Yr10 in the United States (Beaver and Powelson, 1969). Both cases of increased virulence in the pathogen populations followed soon after those specific resistances went into popular cultivation. However, not all race-specific single genes are defeated at the same rate. Virulence on Yr1, Yr2 and Yr3 is found worldwide while virulence on Yr5 is still rare (Stubbs, 1985).

2.11.2 Gene Pyramiding

Gene pyramiding is the incorporation of more than one resistance gene into a single cultivar (Green and Campbell, 1979). In principle, a breakdown in resistance would only result from simultaneous mutations at all the matching virulence loci in the pathogen. This hypothesis assumes that mutations to virulence at different loci are independent, and that the probability of mutation to virulence at multiple loci is the product of the rates of mutation to virulence for each gene locus. Mundt (1990) questions this assumption, and proposes that the probability of a mutation to multiple virulence may be less important than the effect of mutation to multiple virulence on the fitness of the rust. If some virulence combinations are more debilitating on rust fitness than others, then natural selection should stabilise the frequency of virulence genes in the pathogen population, eliminating those unnecessary for survival. In some instances (Taylor *et al.*, 1981), gene pyramids have retained a measure of useful resistance even after all the identified race-specific components have been overcome.

2.11.3 Multilines

Multilines can be two types of mixtures (Groenewegen, 1977);

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1. Mixtures of closely related lines produced by 3-7 backcrosses and having 90-99% of their genes in common, usually selected for diverse resistance to only one disease.

2. Mixtures of moderately related lines with 50-90% of their genes in common, selected for diverse resistance to one target disease, but may also contain resistance to other diseases depending on the choice of parents and level of selection.

The original concept of the multiline included lines that would be free of all rust and thus 'clean' (Borlaug, 1958; Jensen, 1952). If the resistance of a component broke down because of the appearance of a new pathogenic race, it was replaced by a resistant alternative. A major problem with this approach was the finite supply of resistance factors (Parlevliet, 1979b).

Fortunately, a moderate amount of rust particularly later in the season, can be tolerated by the crop and it is not necessary for every line in the mixture to be resistant to all the prevailing races. The premise of the 'dirty multiline approach' is that since each component of the multiline can be attacked by only a few races, the remaining lines act as a physical barrier reducing the spread of disease on the crop (Wolfe, 1985). The 'dirty multiline' approach has a couple of advantages. First it significantly delays the within-crop buildup of disease. The extent of the delay will vary depending on how many genotypes are present in the mixture, what proportion of the component lines have functioning resistance, the genotype unit area, and several other factors (Mundt *et al.*, 1986; Mundt and Brophy, 1988). The second advantage of the 'dirty crop' approach is that since many different races have an opportunity to survive on the multiline and since each host component carries only a single resistance gene, selection against unnecessary virulence genes in the pathogen will ensure that simple races dominate the pathogen population. This concept was first proposed as 'stabilizing selection' (Vanderplank, 1963) and it is still keenly debated. It has been shown in other

pathosystems that complex races will not necessarily be less fit (Grant and Archer, 1983); recessive genes for virulence may be deleterious but they are only one of many factors affecting the pathogen's fitness. Large numbers of apparently unnecessary virulence genes were identified in a study of 94 *Rhyncosporium secalis* (Oud.) Davis isolates from American populations (McDonald *et al.*, 1989). Bronson and Ellingboe (1986) studied two isolates of *Erysiphe graminis* D.C. f.sp. *tritici* E. Marchal that differed by at least four unlinked genes for virulence, and found that the race with increased virulence was less fit. However, when the F1 progeny of crosses between these two races was examined under the same conditions, unnecessary virulence and reduced fitness segregated from each other and thus could not be causally related.

It has been suggested (Grant and Archer, 1983; Groth, 1976) that a combination of multiple genes for resistance in a single multiline might force the evolution of a superrace capable of attacking all the resistance genes. Simulation models have been developed in an attempt to predict the likelihood of a superrace developing (reviewed in Marshall, 1989). A key component of all these models is the level of selection pressure against unnecessary virulence, a character which is very difficult to measure in the field. It is influenced by factors such as whether or not pathogen generations are discrete or overlapping, and at what stage in the lifecycle of the pathogen this selection pressure is assumed to act (Marshall *et al.*, 1986). One mechanism possibly involved in the heightened resistance of multilines is induced resistance. Inoculations of wheat seedlings with an avirulent race of *P. striiformis* delayed the onset of sporulation of a virulent race and also decreased the spore mass produced (Johnson and Allen, 1975). In a multiline, any race that is avirulent on some of the component lines could induce resistance. Physiological resistance induced in this manner can develop in a matter of days and is thought to be non race-specific, but it does not

usually become systemic in the plant which necessitates heavy doses of inducing inoculum to obtain significant levels of resistance.

A multiline approach had been tried toward breeding for stable stripe rust resistance. The winter wheat Tumult was released in the Netherlands with stripe rust resistance from seven sources including *Triticum spelta* (Groenewegen, 1977). In the Pacific Northwest, the first multiline cultivar released was Crew (Allan *et al.*, 1983), a soft white winter wheat with stripe rust resistance from 10 closely related lines. Subsequent multilines developed from this programme have been shown to yield competitively with successful pureline cultivars, but probably because of improved grain yield potential rather than the result of superior rust resistance (Allan, 1988).

2.11.4 Cultivar Mixtures

A mixture is composed of existing cultivars, which makes additional breeding unnecessary. In addition, a broad range of cultivars differing in resistance to several diseases can be considered for inclusion. As with multilines, it is expected that the spread of rust in a mixture will be slower than in a pure stand (Buiel *et al., 1989;* Wolfe, 1985). In studies using crown rust of oats, Mundt and Browning (1985) demonstrated that the size and distribution of the genotype units (i.e. the ground area occupied by an individual) is also important. Not all mixtures are equivalent in yielding ability or disease protection (Wolfe, 1985). It is possible to conduct mixing ability analyses based on the standard combining ability analysis used in plant breeding (Knott and Mundt, 1990).

2.11.5 Interfield Diversity and Regional Deployment of Genes

Several countries have schemes to encourage cultivar diversity, for the purposes of slowing the erosion of resistance. For example, in Britain wheat cultivars are classified into six diversification groups, according to genetic similarity for resistance to yellow rust (Beaton, 1989). In a few of the groups all varieties are susceptible and, with the current complex race situation that exists in Britain, there is some risk that the rust may spread between groups. Aside from the breeding difficulties this system poses, growers have in general not readily implemented it. Obviously many factors are considered before making a decision on which varieties to seed (Priestley and Bayles, 1980a, 1980b). In Britain, a new royalty system was set up in which individual varieties attract different payments set by the breeder (Beaton, 1989), and this is yet another decision-making factor in the grower's choice of variety.

The regional deployment of resistance is intended to work against those diseases that spread over a wide area in a stepwise manner e.g. the wheat rusts. It can be of particular value in areas where the rust is not endemic, and new infections are initiated each season from outside inoculum e.g. the Puccinia Pathway for stem rust and crown rust of oats in North America (Browning *et al.*, 1969). In the course of their wind-assisted migration northward, rust spores may fall on a resistant crop and be unable to multiply further. Those that survive one resistance barrier are met with a different one further along the pathway. The end result of passage through this 'selection sieve' (Knott, 1989) is that the initial inoculum is reduced to a point where it is unable to cause a rust epidemic. The principle of stabilizing selection (Vanderplank, 1968), which predicts that complex races will be relatively less fit to survive on simple host genotypes, discourages the formation of a 'superrace' that would be unaffected by gene deployment (Nelson, 1984). Gene deployment requires the

cooperation of all breeders along an infection pathway. It has not been used in stripe rust breeding because the stripe rust pathogen is endemic in most areas (Rapilly, 1979).

2.11.6 Teliospore Induction

Since the primary infectious stage in the stripe rust lifecycle is the uredial stage, any condition that hastens the pathogen's advancement to the non-infectious telial stage can be considered a form of passive resistance. The exact conditions that induce teliospore formation are not well understood. In India, teliospores form commonly at all growth stages of the host, sometimes as early as two weeks after appearance of the rust (Prasada, 1948). In the same study it was also found that stripe rust teliospores are formed more readily when the weather is warm and unfavourable for the propogation of uredospores. Telia arise on mature leaves at cell sap pH 6.0 or more for most of the major rusts, and redox potentials in tissues within the plant host were found to vary with pH (Benada, 1966). Teliospores arise earlier on the outside of the leaf sheath than on the inside, and on the adaxial side of a leaf rather than the abaxial (Benada, 1966).

The induction of teliospores by other fungi has been reported in *Puccinia recondita* Rob. ex Desm. f. sp. *tritici* through interaction with *Septoria nodorum* Berk. (Van der Wal *et al.*, 1970) and in several other rusts by the hyphomycetous fungus *Aphanocladium album* Preuss. (Biali *et al.*, 1972). The hyperparasite *Verticillium lecanii* (Zimm.) has been observed to grow within and around uredospores of stripe rust in sori on wheat leaves (Mendgen, 1981). It did not penetrate the sporogenous tissue of the rust or the leaf tissue, but did attack spore walls and contents. However, *V. lecanii* does not induce the formation of teliospores directly. The effect of a plant's nutrient status on the formation of teliospores is not known; teliospores could be induced either by the exhaustion of nutrients from the host

tissues, or by the drying of host tissue and subsequent intensive accumulation of nutrients in the mycelium (Benada, 1966).

2.12 DURABLE RESISTANCE

Durable resistance is defined as that which remains effective over a long period of time in widely-grown cultivars (Johnson, 1981). This definition implies nothing about the genetic control of durable resistance, its degree of expression or its race-specificity. It is possible that one or a few major, race-specific genes could confer durable resistance, if the corresponding mutation for virulence in the pathogen is so disadvantageous as to reduce the pathogen's fitness (Vanderplank, 1975). In stem rust, the gene Sr2 in Selkirk has been described as durable (Hare and McIntosh, 1979) as well as other combinations like Sr6 and SrTt1 (Mundt, 1990). In stripe rust, the resistance of Yr5 from *T. spelta* is still effective in most areas (Stubbs, 1985) although it is possible that this resistance has not yet been employed widely enough to confirm its durability.

Durability of resistance has often been associated with slow rusting or partial resistance. Slow rusting is a characteristic of disease development that operates after penetration of the host plant (Parlevliet, 1979a). The components most frequently studied are receptivity, length of the latent period, spore production and infectious period. Slow rusting can be due to at least three possible causes (Johnson, 1988): 1) race-specific, adult plant resistance or temperature-sensitive resistance; 2) a low frequency of virulence for a particular gene in a mixed population of races, so that cultivars having this gene have a low incidence of matching infection; 3) durable, apparently non race-specific rusting. Slow rusting is often considered to be non race-specific (Parlevliet and Zadoks, 1977), and conferred by minor genes inherited in an additive manner (Henriksen and Pope, 1971; Johnson and Taylor, 1972; Milus and Line,

1986). Partial resistance is difficult to identify from infection types because stripe rust shows a continuous spectrum of resistance ranging from a hypersensitive, low infection type reaction to a high infection type reaction (Parlevliet, 1988). The durably resistant cultivar Litle Joss exhibits both a hypersensitive as well as a 'non-immediate', non-necrotic type of reaction (Cartwright and Russell, 1980). Hyde and Elahinia (1989; 1990) reported that the durable resistance in Hybride de Bersee involved components similar to those controlled by Yr14, such as an increase in latent period, and was correlated with uredospore production and colony length.

It is often suggested or implied that partial resistance is likely to be more durable than higher levels, on the grounds that there is less of a selection pressure towards a mutation for virulence. However, Johnson and Taylor (1980) presented data to the contrary. Based on repeated screenings between 1970 and 1977 of the cultivars Maris Nimrod, Maris Huntsman, Maris Bilbo and Hobbit they observed no relationship between level of resistance and its durability. Durable resistance to stripe rust is not self-evident but can only be determined in retrospect. Because of the difficulties in identifying breeding material with durable resistance to stripe rust, old cultivars that have retained a high level of resistance over a period of extensive cultivation are especially valuable as sources of durable resistance (Johnson, 1988). Various levels of durable resistance have been identified in the Kenyan cultivars Bounty, Africa Mayo and Kenya Leopard (Danial, personal communication).

2.13 THE CANADIAN SITUATION

Canada western soft white spring wheat (SWS) is a low protein soft wheat used primarily for the manufacture of cookies, cakes, crackers, flatbread and pastry (Loving and Brenneis, 1981). Soft white spring wheat was introduced to Canada around 1925 through

contracts issued by Ellison Milling in Lethbridge (Canada Grains Council, 1982). Dicklow was the first variety introduced in Alberta but it proved to be weak-strawed and shattering susceptible (Thomas, 1984), as well as being susceptible to rusts and smuts (Canada Grains Council, 1982). It was followed by Federation, which had a problem of yellow flour colour. Both were supplanted by the Idahoan variety Lemhi, which was superseded in the early 70's by the first semi-dwarf soft white spring wheat, Springfield (Thomas, 1984). Springfield proved to be susceptible to powdery mildew and was replaced by Fielder (Sunderman and Bruinsma, 1975) which had a higher yield and testweight, and was resistant to powdery mildew and ergot. Unfortunately it had high protein levels and was susceptible to kernel black point (Conner and Thomas, 1985). When it was first released, Fielder was resistant to stripe rust (Sunderman and Bruinsma, 1975) but a subsequent change in the fungus rendered Fielder susceptible. Yield losses as high as 79% have been reported on Fielder (Conner and Kuzyk, 1988). The stripe rust resistant cultivar, Owens, was given a temporary license in Canada in 1984 (Thomas and Conner, 1985) but did not receive widespread acceptance by growers because of problems with lodging and kernel shape (Conner and Kuzyk, 1988). It also yielded less than Fielder in areas of low stripe rust risk (Thomas, 1984). The most recent soft white spring wheat registered in Alberta is SWS-52, which was developed at the Agriculture Canada Research Station, Lethbridge (Sadasivaiah and Thomas, 1991a; 1991b). Besides having good resistance to lodging and a slightly higher average yield than either Owens or Fielder, SWS-52 is resistant to a new race of stripe rust that was first observed on Owens in 1989 (Conner, personal communication). However, SWS-52 has only been issued an interim registration because of its marginal cookie quality (Sadasivaiah and Thomas, 1991a, 1991b).

Stripe rust has been a recurring problem in irrigated soft white spring wheat since the early 80's. Typically the initial inoculum blows in as uredospores from the Pacific Northwest every spring, although in a mild winter it is possible for the fungus to survive as mycelium on fall infected leaves of winter wheat (Conner *et al.*, 1988). Because of this epidemiology, any resistance present in widely grown US varieties is at risk in Canada too, should there be a change of virulence in the fungus (Thomas, 1984). The incorporation of stripe rust resistance into new Canadian varieties will continue to be a major breeding objective, and knowledge of the genetics of stripe rust resistance in breeding lines will continue to be highly useful.

2.14 WHEAT BREEDING IN KENYA

Wheat was introduced in Kenya towards the end of the 19th Century, and yielded well at higher altitudes until the appearance of stem rust and stripe rust in the early 1900's (Thorpe, 1958). Stem rust soon spread throughout the wheat-growing area in Kenya, leading to rapid changes in the cultivars grown (Martens, 1975; Thorpe, 1958). In consequence, breeding for resistance to stem rust was accorded first priority since about 1950 during which period a strong resistance was secured by pyramiding major stem rust genes within locally adapted lines (Pinto and Hurd, 1970). Baking quality improved gradually until 1940 (Pinto and Hurd, 1970), but unfortunately good stem rust resistance was closely associated with poor quality (Thorpe, 1958). The Canadian cultivars Regent and Ceres R64 were introduced as parents to improve baking quality (Thorpe, 1957). More recently, stripe rust has caused significant yield losses especially in the medium to high elevation wheat-growing areas (2000m and above) (Torres *et al.*, 1988). In response to the stripe rust threat there has been a shift in emphasis towards breeding for resistance (Tanner and van Ginkel,

1988). The stripe rust pathogen has evolved to produce slight differences in each sub-zone within the East and Central Africa region (Stubbs, 1988; Torres *et al.*, 1988). In Kenya, races have moved toward increased virulence partly because of the succesive release of closely related cultivars (Bonthuis, 1985).

PART 1. Genetics of stripe rust resistance in Owens.

3. INTRODUCTION

In Canada, stripe rust occurs most commonly in the interior of British Columbia and southern Alberta (Conners, 1967). The disease reduces yield by affecting grain weight (Gaunt and Cole, 1991; Sunderman and Wise, 1964) and losses can range as high as 40% in soft white spring wheat (Conner and Kuzyk, 1988). Although stripe rust can be controlled quite effectively with the use of systemic foliar fungicides (Brown *et al.*, 1986; Conner and Kuzyk, 1988), the most efficient means of control is through the use of resistant cultivars.

The stripe rust resistant cultivar Owens was given temporary registration in Canada in 1984 (Thomas and Conner, 1985). It remained resistant to the prevailing races of *P. striiformis* until 1989 when a new virulent race appeared (Conner, unpublished data) and was used extensively in breeding programmes for resistance (Conner, personal communication). The gene(s) involved in the stripe rust resistance of Owens have not previously been characterized. Although the genetic identification of a source of resistance is not essential prior to its use, this information is important for the strategic deployment of race specific genes. In addition, comparative genetic analysis involving known genes for resistance avoids wasteful duplication in the use of identical types of resistance (Dyck and Kerber, 1985).

The aim of this study was to identify the gene(s) conferring stripe rust resistance to race SRC 4-84, in the Canadian soft white spring wheat cultivar Owens.

4.1 Cultivars and Crosses

This study was carried out at the Agriculture Canada Research Station in Lethbridge, Alberta. The cultivars used in this genetic study and the genes of resistance they possess are listed in Table 5. Their response to the two races used in this study (Conner, unpublished data) is listed alongside.

The soft white spring wheat cultivars, Fielder and Owens, were intercrossed and were also crossed to six of the standard cultivars which have been shown to carry different Yr genes for seedling resistance, and which were uniformly resistant to SRC 4-84. Crosses to the susceptible cultivar Fielder were used to determine the number of resistance genes in each cultivar. The six cultivars used in crosses were Chinese 166, Hybrid 46, *Triticum spelta*, Thatcher, Compair and Moro. In this set, all but Thatcher, Fielder and Owens were winter wheats which necessitated a 9-week period of vernalisation of parents and progenies to ensure heading. Seed of the differential cultivars was obtained from Dr. R. Johnson of the JI Centre for Plant Science Research in Norfolk, UK. and from Plant Gene Resources of Canada, Ottawa, Ontario. Only uniformly resistant lines were used in the crosses. The cultivars Cappelle Desprez, carrying genes Yr3a and Yr4a, Minister, carrying Yr3c (Lupton and Macer, 1962) and Heines Kolben, carrying Yr6 (Johnson *et al.*, 1986) were uniformly susceptible to both races and were not used in further crossing. The cultivars Heines VII and Lovrin 13, carrying Yr2 (Lupton and Macer, 1962) and Yr9 (Mettin *et al.*, 1978) respectively, were not used in crosses because they were still segregating for stripe rust reaction after two generations of selfing.

Table 5. Reaction of some standard cultivars to the Canadian stripe rust isolates SRC 4-84 and SRC-89, and the resistance genes they possess.

Variety	Resistance genes	Stripe rust isolate SRC 4-84	SRC 89
Chinese 166	Yr1 [A]	Resistant	Resistant
Heines VII	Yr2 [A]	Segregating	Susceptible
Soissonais Desprez	Yr2 [A]	Resistant	Susceptible
Cappelle-Desprez	Yr3a, Yr4a [A]	Susceptible	Susceptible
Hybrid 46	Yr3b, Yr4b [A]	Resistant	Resistant
Minister	Yr3c [A]	Susceptible	Susceptible
Opal	Yr4b [A]	Susceptible	Susceptible
Triticum spelta A.	Yr5 [B]	Resistant	Resistant
Heines Kolben	Yr6 [B]	Susceptible	Susceptible
Thatcher	Yr7 [B]	Resistant	Susceptible
Compair	Yr8 [C]	Resistant	Resistant
Lovrin 13	Yr9 [D]	Segregating	Segregating
Clement	Yr9 [E]	Resistant	Resistant
Moro	Yr10 [F]	Resistant	Resistant
Fielder	Yr6 [G]	Susceptible	Susceptible
Owens	Unknown	Resistant	Susceptible

(Conner, unpublished data)

A = Lupton and Macer, 1962
B = Macer, 1966
C = Riley *et al.*, 1968
D = Mettin *et al.*, 1978
E = McIntosh, 1988
F = Macer, 1975
G = Vallavielle-Pope and Line, 1990

The cultivars Owens and Fielder (Sunderman and Bruinsma, 1975) originated from the USDA-ARS Experimental Station in Aberdeen, Idaho.

With the exception of the Fielder x Hybrid 46 cross, eight resistant F1 plants from each cross were backcrossed to the susceptible cultivar Fielder. The F1, F2 and BCF1 generations were tested from each of the crosses and its reciprocal. No fewer than 400 plants from 8 F2 families and no fewer than 50 plants from 4 BCF1 families were tested. The plants were seeded in Hillson-style (4 x 8) rootrainer trays (Spencer-Lemaire Industries, Edmonton, Alberta) containing Cornell mix (Boodley and Sheldrake, 1973), two to a cavity, and grown in controlled environment growth chambers. The day/night temperature regime was 16 hours of light at 15°C and 8 hours of darkness at 8°C (Newton and Johnson, 1936). Diurnal temperature regimes were used to simulate natural conditions (Brown and Sharp, 1969). Owens and Fielder were seeded in each rootrainer as a resistant and susceptible check, respectively.

4.2 Pathogenic Races

One pathogenic race collected in southern British Columbia was used. Race SRC 4-84 was first collected at Creston in 1984 from leaves of the cultivar Fielder, and was continuously cultured on pots of susceptible cultivars under the diurnal temperature regime described above. Precautions were taken to ensure that no cross-contamination of races occurred. Races were cultured in separate growth cabinets, and spore harvests of different races were carried out at least a day apart. The harvested uredospores were put in 1 mL cryogenic vials and stored at -196°C in liquid nitrogen.

4.3 Inoculation procedure

The seedlings were inoculated at the 2-3 leaf stage by atomizing a suspension of stripe rust spores in a light, non-phytotoxic mineral oil, Dustrol (Ciba-Geigy Canada Ltd., Regina, Saskatchewan) onto the leaves with a pressurised aerosol sprayer (Browder, 1971). After the oil dried, the inoculated seedlings were sprayed with a fine mist of water containing a few drops of the surfactant Tween 20 (polyoxyethylene sorbitan monolaurate) and then incubated in the dark for 48 hours at 10°C in plastic bags in order to maintain a film of free water on the leaves and a relative humidity of close to 100%. After incubation the seedlings were returned to their former growth cabinet. Disease observations were made 14-17 days after inoculation.

4.4 Classification system

The seedlings were rated for disease using the 0-4 scale (Volin and Sharp, 1973) where infection types 0-1 are considered resistant, infection type 2 is an intermediate reaction but still considered resistant, and infection types 3-4 are susceptible.

Although the existence of extra-chromosomal (maternal) inheritance has never been reported for stripe rust resistance, it was considered prudent to make reciprocal crosses for each varietal pairing in this study. Data from reciprocal crosses were tested for homogeneity of variances using the chi-square test for homogeneity (Snedecor and Cochrane, 1976) and, where appropriate, the frequencies were combined into a generation total. A chi-square test for goodness of fit of various genetic ratios was used to analyze all F2 and backcross data (Steel and Torrie, 1980). SAS (SAS Institute Inc., Cary, NC. USA) computer programmes, CHISQ and CHIRATIO, were used for both these statistical analyses (Statistics Unit, Lethbridge Research Station).

5. RESULTS

5.1 SCREENING OF CULTIVARS

Thatcher, Heines VII and Soissonais-Desprez were the only cultivars, in addition to Owens, which were resistant to race SRC 4-84 but susceptible to the new race, SRC-89 (Conner, unpublished data). There were no cultivars that were susceptible to race SRC 4-84 and resistant to race SRC-89. The cultivar Fielder occasionally shows plants with resistant infection types when screened with SRC-89 under growth cabinet conditions. Further tests were initiated with race SRC-89 but are not included in this report (Conner, unpublished data). The cultivars Cappelle-Desprez, Minister, Opal and Heines Kolben were susceptible to both races, while the cultivars Chinese 166, Hybrid 46, *Triticum spelta*, Compair, Clement and Moro were resistant to both races. The cultivar Heines VII segregated for its reaction to race SRC 4-84, and cultivar Lovrin 13 segregated in its response to both races.

5.2 SCREENING OF SEGREGATING GENERATIONS

Fielder x Owens

In crosses between Owens and the susceptible cultivar, Fielder, all the F1 progeny were resistant (Table 6). The F2 generation from both reciprocal crosses fit a 3:1 ratio of resistant to susceptible plants, indicating that the resistance in Owens to race SRC 4-84 is conferred by a single, dominant gene. This conclusion was further supported in the Fielder/Owens backcross, which fit the expected 1:1 ratio, but not in its reciprocal. The ratio 13:3 for the segregation of one dominant and one recessive gene produced a better fit of the Owens/Fielder F2 data but a poorer fit overall.

Table 6.
Stripe rust response of F1, F2 and BCF1 progeny of crosses between Owens and Fielder screened with SRC 4-84.

Cross and Generation			Observed		Expected [a]		Ratio	Prob.
			R	S	R	S	R:S	
Checks	Owens		20	0				
	Fielder		0	24				
Owens/Fielder		F1	40	0				
		F2	77	19	72	24	3:1	0.238
			77	19	78	18	13:3	0.794
		BCF1	29	11	20	20	1:1	0.004
Fielder/Owens		F1	40	0				
		F2	70	25	71	24	3:1	0.767
			70	25	77	18	13:3	0.059
		BCF1	23	16	19	19	1:1	0.262
Combined		F2	147	44	143	48	3:1	0.531
			147	44	156	36	13:3	0.129
		BCF1	52	27	39	40	1:1	0.005

a Expected frequencies rounded off to nearest whole number.

Fielder x Chinese 166

The F1 progeny from the cross between Chinese 166 and the susceptible cultivar Fielder were uniformly resistant, suggesting that the gene in Chinese 166 is dominant (Table 7). The frequencies of resistant and susceptible plants in the F2 and BCF1 generations, fit a 3:1 and a 1:1 ratio, respectively, indicating that the resistance in Chinese 166 to race SRC 4-84 is conferred by a single, dominant gene, probably Yr1 (Lupton and Macer, 1962).

Table 7.
Stripe rust response of F1, F2 and BCF1 progeny of crosses between Fielder and Chinese 166 screened with race SRC 4-84.

Cross and Generation		Observed		Expected [a]		Ratio	Prob.
		R	S	R	S	R:S	
Checks	Owens	63	0				
	Fielder	1	63				
	Chinese 166	21	0				
Fielder/Chinese 166	F1	36	0				
	F2	341	94	326	109	3:1	0.102
	BCF1	32	39	35	36	1:1	0.406
Chinese 166/Fielder	F1	35	0				
	F2	350	93	332	111	3:1	0.051
	BCF1	45	37	41	41	1:1	0.377
Combined	F2	691	187	658	220	3:1	0.011
	BCF1	77	76	76	77	1:1	0.936

a Expected frequencies rounded off to nearest whole number.

Owens x Chinese 166

In crosses between Chinese 166 and Owens, the F2 generation fit a 15:1 ratio of resistant to susceptible plants in one direction of the reciprocal cross. Nevertheless, the reciprocals were statistically homogeneous and the combined F2 population also fit a 15:1 ratio, indicating the segregation of two dominant genes (Table 8). This result was supported by a good fit for a 3:1 segregation ratio of resistant to susceptible plants in the BCF1 generation.

Table 8.
Stripe rust response of F1, F2 and BCF1 progeny of crosses between Owens and Chinese 166 screened with race SRC 4-84.

Cross and Generation		Observed R	Observed S	Expected [a] R	Expected [a] S	Ratio R:S	Prob.
Checks	Owens	55	0				
	Fielder	0	57				
	Chinese 166	21	0				
Owens/Chinese 166	F1	37	0				
	F2	400	39	412	27	15:1	0.023
	BCF1	23	5	21	7	3:1	0.383
Chinese 166/Owens	F1	40	0				
	F2	408	29	410	27	15:1	0.739
	BCF1	63	20	62	21	3:1	0.849
Combined	F2	808	68	821	55	15:1	0.064
	BCF1	86	25	83	28	3:1	0.547

a Expected frequencies rounded off to nearest whole number.

Fielder x Hybrid 46

The F1 progeny of crosses between Fielder and Hybrid 46 were susceptible to stripe rust (Table 9), indicating that the effective resistance factor in Hybrid 46 was homozygous recessive. The single resistant F1 plant observed was probably an escape. The F2 generation segregated in a 1:3 ratio of resistant to susceptible plants, suggesting the action of a single recessive gene in Hybrid 46. Backcrosses to the recessive parent Hybrid 46 were precluded by the fact that seed of that cultivar had not been vernalised. This pairing would be necessary to confirm the observed F2 ratios.

Table 8.
Stripe rust response of F1, F2 and BCF1 progeny of crosses between Owens and Chinese 166 screened with race SRC 4-84.

Cross and Generation		Observed		Expected [a]		Ratio	Prob.
		R	S	R	S	R:S	
Checks	Owens	55	0				
	Fielder	0	57				
	Chinese 166	21	0				
Owens/Chinese 166	F1	37	0				
	F2	400	39	412	27	15:1	0.023
	BCF1	23	5	21	7	3:1	0.383
Chinese 166/Owens	F1	40	0				
	F2	408	29	410	27	15:1	0.739
	BCF1	63	20	62	21	3:1	0.849
Combined	F2	808	68	821	55	15:1	0.064
	BCF1	86	25	83	28	3:1	0.547

a Expected frequencies rounded off to nearest whole number.

Fielder x Hybrid 46

The F1 progeny of crosses between Fielder and Hybrid 46 were susceptible to stripe rust (Table 9), indicating that the effective resistance factor in Hybrid 46 was homozygous recessive. The single resistant F1 plant observed was probably an escape. The F2 generation segregated in a 1:3 ratio of resistant to susceptible plants, suggesting the action of a single recessive gene in Hybrid 46. Backcrosses to the recessive parent Hybrid 46 were precluded by the fact that seed of that cultivar had not been vernalised. This pairing would be necessary to confirm the observed F2 ratios.

Table 9.
Stripe rust response of F1 and F2 progeny from crosses between Fielder and Hybrid 46 screened with SRC 4-84.

Cross and generation		Observed R	Observed S	Expected [a] R	Expected [a] S	Ratio R:S	Prob.
Checks	Owens	60	1				
	Fielder	0	64				
	Hybrid 46	11	0				
Fielder/Hybrid 46	F1	1	37				
	F2	58	160	55	163	1:3	0.584
Hybrid 46/Fielder	F1	0	39				
	F2	63	174	59	178	1:3	0.574
Combined	F2	121	334	114	341	1:3	0.432

a Expected frequencies rounded off to nearest whole number.

Owens x Hybrid 46

All of the F1 progeny of crosses between Owens and Hybrid 46 were resistant, as expected for the segregation of the dominant gene in Owens (Table 10). The F2 generation, however, produced many more resistant plants than were required to fit the expected 13:3 ratio for the segregation of one dominant gene and one recessive gene. The only ratio that fit the data was 55:9, for the segregation of one dominant gene and two recessive genes. The backcross data for both reciprocals was also disproportionate but in the opposite direction. The recessive gene in Hybrid 46 would not be expressed in this backcross but assuming segregation of at least the one dominant gene from Owens, there was a great excess of susceptible plants. The F2s and backcrosses were screened several months apart and it is possible that there were differences in growth cabinet environment between the two tests. This possibility is discussed

in Section 6. Although the results from F2 and BCF1 generations appear conflicting, they both suggest that Owens and Hybrid 46 do not share any resistance genes in common.

Table 10.
Stripe rust response of F1, F2 and BCF1 progeny from crosses between Owens and Hybrid 46 screened with SRC 4-84.

Cross and generation		Observed		Expected [a]		Ratio	Prob.
		R	S	R	S	R:S	
Checks	Owens	60	0				
	Fielder	0	64				
	Hybrid 46	11	0				
Owens/Hybrid 46	F1	36	0				
	F2	377	53	370	60	55:9	0.300
	BCF1	27	57	42	42	1:1	0.001
Hybrid 46/Owens	F1	38	0				
	F2	379	48	367	60	55:9	0.094
	BCF1	22	62	42	42	1:1	0.000
Combined	F2	756	101	736	121	55:9	0.055
	BCF1	49	119	84	84	1:1	0.000

a Expected frequencies rounded off to nearest whole number.

Fielder x *Triticum spelta*

In the crosses between Fielder and *Triticum spelta* all the F1 progeny were resistant (Table 11). The F2 progeny segregated in a 3:1 ratio and the BCF1 generation segregated in a 1:1 manner indicating the presence of a single dominant gene for resistance to SRC 4-84 in *T. spelta*, possibly Yr5 (Macer, 1966).

Table 11.
Stripe rust response of F1, F2 and BCF1 progeny of crosses between Fielder and *Triticum spelta* screened with SRC 4-84.

Cross and Generation		Observed		Expected [a]		Ratio	Prob.
		R	S	R	S	R:S	
Checks	Owens	57	0				
	Fielder	0	62				
	T. spelta	18	0				
Fielder/*T. spelta*	F1	22	0				
	F2	295	91	289	97	3:1	0.518
	BCF1	38	46	42	42	1:1	0.383
T. spelta/Fielder	F1	24	0				
	F2	295	96	293	98	3:1	0.838
	BCF1	56	56	56	56	1:1	1.000
Combined	F2	590	187	583	194	3:1	0.548
	BCF1	94	102	98	98	1:1	0.568

a Expected frequencies rounded off to nearest whole number.

Owens x *T. spelta*

The F1 progeny of crosses between Owens and *T. spelta* were uniformly resistant (Table 12). However, the F2 and BCF1 generations segregated to fit 3-gene ratios of 63:1 and 15:1 respectively, indicating a possible suppression of crossing over in this cultivar pairing.

Table 12.
Stripe rust reaction of F1, F2 and BCF1 progeny of crosses between Owens and *Triticum spelta* screened with SRC 4-84.

Cross and Generation			Observed		Expected [a]		Ratio	Prob.[b]
			R	S	R	S	R:S	
Checks	Owens		64	0				
	Fielder		1	63				
	T. spelta		18	0				
Owens/*T. spelta*		F1	29	0				
		F2	364	3	361	6	63:1	0.250
		BCF1	98	13	104	7	15:1	0.017
T. spelta/Owens		F1	17	0				
		F2	329	5	329	5	63:1	0.923
		BCF1	107	3	103	7	15:1	0.127
Combined		F2	693	8	690	11	63:1	0.369

a Expected frequencies rounded off to nearest whole number.
b Probability for calculated expected frequencies (to three decimals).

Fielder x Thatcher

The F1 generation of the crosses between Fielder and Thatcher was uniformly resistant (Table 13). The F2 generation segregated in a 3:1 ratio of resistant to susceptible plants, indicating a single dominant gene, Yr7, in Thatcher (Macer, 1966). This conclusion was further supported by a 1:1 segregation ratio in three out of the four backcrosses. There was an excess of resistant plants segregating in the P/Fielder backcross.

Table 13.
Stripe rust reaction of F1, F2 and BCF1 progeny of crosses between Fielder and Thatcher screened with SRC 4-84.

Cross and Generation			Observed R	Observed S	Expected [a] R	Expected [a] S	Ratio R:S	Prob.
Checks	Owens		16	0				
	Fielder		0	28				
	Thatcher		85	0				
Fielder/Thatcher (P)		F1	32	0				
		F2	89	32	91	30	3:1	0.713
Fielder/ P		BCF1	29	31	30	30	1:1	0.796
P /Fielder		BCF1	41	18	29	29	1:1	0.003
Thatcher/Fielder		F1	31	0				
		F2	151	59	157	53	3:1	0.300
		*BCF1	63	56	59	59	1:1	0.521
Combined		F2	240	91	248	83	3:1	0.295

a Expected frequencies rounded off to nearest whole number.
* Reciprocal backcrosses combined.
P Abbreviation for Fielder/Thatcher cross.

Owens x Thatcher

In crosses between Owens and Thatcher, the F1s were resistant and the F2s segregated in a 15:1 ratio, suggesting that the single dominant gene in Owens is different from the one in Thatcher (Table 14). The BCF1 generation from the Thatcher/Owens cross barely fit the expected 3:1 ratio, while the BCF1 from the reciprocal Owens/Thatcher cross did not fit. However, neither cross fit a 1:1 ratio either.

Table 14.
Stripe rust response of F1, F2 and BCF1 progeny of crosses between Owens and Thatcher screened with SRC 4-84.

Cross and Generation		Observed R	Observed S	Expected [a] R	Expected [a] S	Ratio R:S	Prob.
Checks	Owens	22	0				
	Fielder	1	23				
	Thatcher	85	0				
Owens/Thatcher	F1	31	0				
	F2	422	23	417	28	15:1	0.346
	BCF1	99	20	89	30	3:1	0.039
Thatcher/Owens	F1	31	0				
	F2	350	24	351	23	15:1	0.894
	BCF1	80	38	88	30	3:1	0.071
Combined	F2	772	47	768	51	15:1	0.546

a Expected frequencies rounded off to nearest whole number.

Fielder x Compair

All the F1 progeny from the Fielder/Compair cross and its reciprocal were resistant indicating that the resistance in Compair, probably Yr8 (Riley *et al.*, 1968), is dominant. The F2 generation did not produce a good fit of a 3:1 ratio of resistant to susceptible plants due to a lack of sufficient susceptible progeny (Table 15). A better fit was observed for a 13:3 ratio, suggesting the presence of one dominant and one recesssive gene in Compair. The backcross generations segregated as expected, in a 1:1 ratio.

Table 15.
Stripe rust response of F1, F2 and BCF1 progeny of crosses between Fielder and Compair screened with SRC 4-84.

Cross and Generation			Observed		Expected [a]		Ratio	Prob.
			R	S	R	S	R:S	
Checks	Owens		122	0				
	Fielder		6	122				
	Compair		28	0				
Fielder/Compair		F1	29	0				
		F2	226	51	208	69	3:1	0.011
					225	52	13:3	0.885
		BCF1	30	26	28	28	1:1	0.593
Compair/Fielder		F1	30	0				
		F2	253	67	240	80	3:1	0.093
					260	60	13:3	0.316
		BCF1	69	71	70	70	1:1	0.866
Combined		F2	479	118	448	149	3:1	0.003
					485	112	13:3	0.525
		BCF1	99	97	98	98	1:1	0.886

a Expected frequencies rounded off to nearest whole number.

Owens x Compair

The F1 generation of crosses between Owens and Compair were uniformly resistant and the F2 progeny fit a 15:1 ratio as expected for the segregation of two dominant genes (Table 16). The suggestion that Compair might possess more than one resistance gene was not supported by appropriate segregation in this cross (Table 19). The expected 3:1 ratio of resistant to susceptible plants in the BCF1 generation was observed in the Owens/Compair cross. Although the BCF1 progeny of the reciprocal Compair/Owens cross produced a poor fit of the expected 3:1 ratio due to an excess of susceptible progeny, the results also did not fit a 1:1 ratio, indicating that more than one gene was segregating.

Table 16.
Stripe rust response of F1, F2 and BCF1 progeny of crosses between Owens and Compair screened with SRC 4-84.

Cross and Generation			Observed R	Observed S	Expected [a] R	Expected [a] S	Ratio R:S	Prob. [b]
Checks	Owens		96	0				
	Fielder		5	87				
	Compair		47	0				
Owens/Compair		F1	29	0				
		F2	336	25	338	23	15:1	0.596
		BCF1	125	42	125	42	3:1	0.964
Compair/Owens		F1	30	0				
		F2	374	31	380	25	15:1	0.243
		BCF1	128	67	146	49	3:1	0.003
Combined		F2	710	56	718	48	15:1	0.225
		BCF1	253	109	271	91	3:1	0.025

a Expected frequencies rounded off to nearest whole number.
b Probability for calculated expected frequencies (to three decimals).

Fielder x Moro

All F1 progeny from crosses between Fielder and Moro were resistant, and the F2 generati fit a 3:1 ratio of resistant to susceptible plants (Table 17). The BCF1 progeny from each of the reciprocal crosses fit a 1:1 ratio but the two data sets were not homogeneous enough to combine into a generation total. These results indicate that resistance to SRC 4-84 in Moro conditioned by a single, dominant gene, probably Yr10 (Macer, 1975).

Table 17.
Stripe rust response of F1, F2 and BCF1 progeny of crosses between Fielder and Moro screened with SRC 4-84.

Cross and Generation			Observed		Expected [a]		Ratio	Prob.
			R	S	R	S	R:S	
Checks	Owens		56	0				
	Fielder		0	63				
	Moro		74	0				
Fielder/Moro		F1	24	0				
		F2	325	111	327	109	3:1	0.825
		BCF1	50	65	58	57	1:1	0.162
Moro/Fielder		F1	30	0				
		F2	324	118	331	111	3:1	0.410
		BCF1	111	84	97	98	1:1	0.053
Combined		F2	649	229	658	220	3:1	0.459

a Expected frequencies rounded off to nearest whole number.

Owens x Moro

Crosses between Owens and Moro segregated in a 15:1 ratio of resistant to susceptible plants in the F2 generation, indicating that resistance in each variety is controlled by a different gene (Table 18). The BCF1 progeny fit the expected 3:1 ratio further indicating the segregation of two different genes.

Table 18.
Stripe rust response of F1, F2 and BCF1 progeny of crosses between Owens and Moro screened with SRC 4-84.

Cross and Generation			Observed R	Observed S	Expected [a] R	Expected [a] S	Ratio R:S	Prob.
Checks	Owens		62	0				
	Fielder		1	62				
	Moro		93	0				
Owens/Moro		F1	30	0				
		F2	410	31	413	28	15:1	0.499
		BCF1	141	55	147	49	3:1	0.322
Moro/Owens		F1	26	0				
		F2	407	34	413	28	15:1	0.205
		BCF1	98	41	104	35	3:1	0.221
Combined		F2	817	65	827	55	15:1	0.170
		BCF1	239	96	251	84	3:1	0.122

a Expected frequencies rounded off to nearest whole number.

Alternate hypotheses

In the Owens/Fielder crosses, a 13:3 ratio would also fit the observed F2 data which could mean that Owens has two resistance genes, one dominant and one recessive (Table 19). The expected F2 ratio for subsequent crosses to cultivars with a dominant, monogenic resistance would be 61:3. This ratio only fit F2 progeny of the Owens/Thatcher cross. In crosses to cultivars with a recessive gene the F2 ratio would be 55:9, for the segregation of one dominant and two recessive genes. Progeny from the Owens/Hybrid 46 cross fit this ratio (Table 19). In all other crosses the results did not fit the ratios expected if Owens carried one dominant and one recessive gene for resistance.

Table 19.
Stripe rust response of F2 progeny from crosses between Owens and other cultivars, screened with SRC 4-84.

Cross	Gene	Observed		Expected		Ratio	Prob.
		R	S	R	S	R:S	
Owens/Chinese 166	Yr1	400	39	418	21	61:3	0.000
Chinese 166/Owens		408	29	417	20	61:3	0.054
Combined		808	68	835	41	61:3	0.000
Owens/Hybrid 46	Yr3b	377	53	370	60	55:9	0.300
Hybrid 46/Owens	Yr4b	379	48	367	60	55:9	0.094
Combined		756	101	736	121	55:9	0.055
Owens/*T. spelta*	Yr5	364	3	350	17	61:3	0.000
T. spelta/Owens		329	5	318	16	61:3	0.006
Combined		693	8	668	33	61:3	0.000
Owens/Fielder	Yr6	77	19	78	18	13:3	0.794
Fielder/Owens		70	25	77	18	13:3	0.059
Combined		147	44	156	35	13:3	0.129
Owens/Thatcher	Yr7	422	23	424	21	61:3	0.631
Thatcher/Owens		350	24	356	18	61:3	0.114
Combined		772	47	781	38	61:3	0.155
Owens/Compair	Yr8	336	25	344	17	61:3	0.044
Compair/Owens		374	31	386	19	61:3	0.005
Combined		710	56	730	36	61:3	0.001
Owens/Moro	Yr10	410	31	420	21	61:3	0.020
Moro/Owens		407	34	420	21	61:3	0.003
Combined		817	65	841	41	61:3	0.000

Homogeneity chi-square values for the crosses listed above (from top to bottom) are; 0.284, 0.214, 0.623, 0.398, 0.444, 0.699, 0.699.

6. DISCUSSION

From crosses between the susceptible cultivar Fielder and the resistant cultivar Owens, it was determined that Owens possesses one dominant gene against the race SRC 4-84. Although the F2 data from these same crosses also fit a 13:3 ratio, the presence of two genes in Owens (one dominant and one recessive) is not indicated in the majority of other crosses involving Owens (Table 19). A more definitive way to distinguish between these two ratios would be to test F3 progeny from susceptible F2 plants. If Owens does possess one dominant and one recessive gene, then 2/3 of the susceptible F2s will produce a 1:3 ratio of resistant to susceptible plants in the F3 generation. If Owens has only one dominant gene, all the susceptible F2s will produce completely susceptible F3 families. This study demonstrated that the homozygous dominant gene in Owens, effective against SRC 4-84, is definitely not Yr1 (Chinese 166), Yr5 (*T. spelta*), Yr7 (in Thatcher), Yr8 (Compair) or Yr10 (Moro). In crosses with Owens, all five cultivars produced F2 ratios which showed the segregation of at least two genes.

While the cultivar *T. spelta* seemed to possess only one resistance gene when crossed to the susceptible Fielder, when crossed to Owens it appeared to be segregating for at least two genes besides the one in Owens. In tests with other stripe rust races, *T. spelta* has been reported to display complex patterns of resistance (Robbelen and Sharp, 1978; Griffey and Allan, 1988; Luthra *et al.*, 1989). Resistance from *T. spelta* (Yr5) is suppressed in crosses with the cultivar Thatcher, which is thought to possess a dominant inhibitor of Yr5 (Wolfe, 1984). An appreciable disturbance at meiosis has been reported to occur within the A and B genomes in crosses between *Triticum durum* (tetraploid) and *T. spelta* (hexaploid)(Lapochkina and Pukhalski, 1983) although not in any crosses with *Triticum aestivum* (hexaploid). Nonetheless, since the gene Yr5 is located in the B genome, on

chromosome arm 2B (Law, 1975) this could be a possible explanation for the reduced crossing-over with *T. spelta*. It is possible that the unknown resistance in Owens is also located on chromosome 2B (Conner, unpublished data). If the gene in Owens is linked to Yr5 in *T. spelta* there could be suppression of crossing over between the two cultivars, resulting in fewer susceptible plants than expected.

The segregation of resistant and susceptible plants in the F2 generation of crosses between Thatcher and Owens showed that the two cultivars do not share a common resistance. Although the backcross generation did not provide a perfect fit of the expected 3:1 ratio for the segregation of two genes, the results clearly did not fit a three-gene (15:1) ratio.

The resistance in Owens is also not the same as that in Hybrid 46 since a great many susceptible plants were observed in the segregating generations. However, the exact pattern of genetic interaction between these two cultivars is not clear. The results indicate that in Hybrid 46, which carries Yr3b and Yr4b (Lupton and Macer, 1962), the effective gene against SRC 4-84 is at the Yr3 locus, since Yr4b (in Opal) is susceptible to that race. Furthermore, the resistance in Hybrid 46 to this race is recessive, since the F1 progeny from crosses between Hybrid 46 and the susceptible variety Fielder were uniformly susceptible. The discovery of a recessive resistance was unexpected so vernalised plants of Hybrid 46 were not immediately available for backcrossing to the susceptible F1s. A backcross with Hybrid 46 would further help determine the number of genes in Hybrid 46. Results from screening crosses between Owens and Hybrid 46 (Table 10) are consistent with the presence of a dominant gene in Owens, as the F1 progeny were resistant. Since the resistance in Hybrid 46 is recessive, it would not be expressed in the backcross to a susceptible cultivar like Fielder. The best explanation for the segregation of resistant plants in

the BCF1 generation is that Owens possesses a dominant gene. Additional crosses should be made to clarify the genetics of resistance in Hybrid 46. The original F2 data of Lupton and Macer (1962), who first designated the genes Yr3b and Yr4b as dominant in Hybrid 46, would also support the alternate hypothesis of Yr3b being recessive. Confirmation of the ratio would require further F2 screening to increase population sizes, or testing of F3 progeny. Recessive genes controlling resistance to stripe rust have been reported previously (Lupton and Macer, 1962; Stubbs *et al.*, 1984). In host-parasite relationships, the dominance or recessiveness of a resistance gene is not an absolute attribute but rather the phenotypic expression of a specific interaction between a plant and a pathogen (Robbelen and Sharp, 1978). The gene Yr6 in Heines Peko was reported to be dominant when screened with race 2B but recessive when screened with race 8B (Macer, 1966). Lupton and Macer (1962) postulated a reversal of dominance for the genes Yr3a and Yr4a in Cappelle Deprez and Yr3c in Minister, in which the resistance genes reacted dominantly against the less aggressive races 5 and 8, and recessively against the more aggressive races 2B and 8B. They proposed a dosage effect to explain this observation. In the heterozygous state, a single allele might express resistance against a less aggresssive race but not against a more aggressive one. The phenomenon of dominance reversal has also been attributed to the interaction of two alleles of the same gene in different parents (Lupton and Macer, 1962), chromosome instability in one of the parents, or the influence of the genetic background (Robbelen and Sharp, 1978). Since virulence is usually recessive, an apparent reversal of dominance can be caused by the existence of heterozygous virulence loci in the pathogen (Roelfs, 1988). The possibility cannot be ruled out that the recessive resistance in Hybrid 46 is a third, previously unidentified gene.

There are four known genes for seedling resistance to stripe rust that were not tested. The gene Yr15 is derived from *Triticum dicoccoides* Koern. (Gerechter-Amitai *et al.*, 1989) and is unlikely to be in the Owens pedigree, although there may be more than one source of this gene. Resistance genes Yr2 and Yr9 in cultivars Heines VII and Lovrin 13, respectively, were still heterozygous for stripe rust resistance even after two generations of selection. Uniformly resistant lines of Heines VII and Lovrin 13 have recently been identified, and will be used in further crosses to identify the resistance gene in Owens. A cross between Owens and the cultivar Clement (also possessing Yr9) is also currently underway. Based on screenings with the two Canadian stripe rust races (Table 5), it is unlikely that the resistance in Owens is being conferred by Yr9 since Lovrin 13 (with Yr9) was resistant to the 'new' race SRC-89 while Owens was susceptible. Owens also lacks the sticky dough trait that is known to be tightly linked to Yr9 on the short arm of chromosome 1R (Singh *et al.*, 1990). Nonetheless, the cross will be made to investigate the possibility that the gene in Owens is a different allele of Yr9.

The uncertain genetic background of Heines VII has been mentioned previously in the literature review (Section 2.9). Heines VII possesses at least Yr2 and possibly one or two other resistance genes (Singh and Johnson, 1989). A further complication is that Yr2 has been reported to act recessively in some genetic backgrounds (Labrum, 1980). It was hoped that the European cultivar Soissonais-Desprez could be substituted in crosses to Fielder and Owens as a source of Yr2 but this cultivar, too, possesses at least one additional gene for resistance to certain races (Singh and Johnson, 1989). Moreover, at least one of these additional genes is common to both Heines VII and Soissonais-Desprez (Singh and Johnson, 1989). Since both cultivars have shown similar disease responses to that of Owens, in response to infection by the two Canadian races, it is

possible that the gene in Owens conferring resistance to SRC 4-84 could be Yr2. Tests to identify the seedling resistance YrA, first identified in Avocet (Wellings *et al.*, 1988), will be initiated when seed of this Australian cultivar is available. The YrA resistance, either alone or in conjunction with Yr6, was present in several Australian and exotic spring wheats. Its resistance response was shown to be influenced by light intensity, displaying a greater susceptibility at low light intensities (Wellings *et al.*, 1988). In addition, genetic heterogeneity within cultivars with respect to YrA was quite common. Both these latter observations are similar to some aspects of Owens' response to SRC 4-84, but until the Canadian stripe rust races are characterised with respect to their virulence/avirulence on YrA, the possible presence of this resistance in Owens cannot be investigated. The resistance factor YrA has yet to be fully characterized and may be any one of the previously identified Yr genes. Finally, the possibility cannot be ruled out that the gene in Owens is one that was never described.

There were instances where the data from reciprocal crosses were not homogeneous, mostly in the BCF1 generation where population sizes were small. None of the Owens and Fielder crosses which showed heterogeneity of response between reciprocals in the BCF1, showed a similar heterogeneity in the corresponding F1 or F2 generations. There was no consistent evidence for the presence of exta-chromosomal inheritance within any cross.

Stripe rust is more strongly influenced by the environment than any other cereal rust (Stubbs, 1985). In this study, temperature, humidity, and air quality were kept constant by conducting the disease screenings in growth cabinets. Minor differences in light intensity were noted between cabinets, in a preliminary check on light levels, but the overall range was still optimum for infection development. There were a few resistant plants in the susceptible check Fielder, and a single susceptible plant in the resistant check Owens.

Barring these exceptions, the check varieties performed as expected. However, the environment may have had more of an effect on other genotypes in the test than it did on the checks.

7. CONCLUSION

It was determined that the soft white spring wheat cultivar Owens possesses one effective, dominant gene against the race SRC 4-84. This homozygous dominant gene is definitely not Yr3a (Cappelle-Desprez), Yr3c (Minister), Yr4a (Cappelle-Desprez), Yr4b (Opal) or Yr6 (Heines Kolben), all of which are susceptible to race SRC 4-84. It is also not Yr1 (Chinese 166), Yr3b (Hybrid 46), Yr5 (*T. spelta*), Yr7 (Thatcher), Yr8 (Compair) or Yr10 (Moro) since the F2 progeny from crosses between these cultivars and Owens showed the clear segregation of two or more separate genes. The effective resistance factor against SRC 4-84 in the cultivar Hybrid 46 (Yr3b, Yr4b) was determined to be acting recessively. It could be either Yr3b or a previously undetected gene in Hybrid 46. The resistance in Owens is unlikely to be Yr9 (Lovrin 13), which shows a different infection-type response with the Canadian races than Owens does, although it could possibly be a previously unreported allele of Yr9. The gene Yr15 (identified in *T. dicoccoides*) is not indicated in the Owens pedigree. The untested resistance factors that could possibly be present in Owens are Yr2 (Heines VII) or YrA (Avocet). The possibility cannot be ruled out that the gene in Owens is one that has never been described.

PART 2. Screening of Kenyan lines with Canadian races of stripe rust.

8. INTRODUCTION

Stripe rust is a recurring problem on wheat in Kenya especially in the cool, highland areas where conditions are ideal for infection. Recently, the threat posed by stripe rust has resulted in a shift of emphasis towards breeding for resistance (Tanner and Van Ginkel, 1988). It is likely that some of the existing stripe rust genes were introduced fortuitously along with genes for stem rust resistance or improved quality. However, this resistance is often short-lived (Annual Report NPBRC, 1983). The hypersensitive reaction produced on many resistant cultivars suggests the action of major genes, but little is known about the genetics of stripe rust resistance in Kenyan cultivars. At present there are no adequate facilities for working with different races at the National Plant Breeding Research Centre (NPBRC), Njoro, Kenya.

The aim of this study was to learn more about the nature of stripe rust resistance in Kenyan cultivars, by testing them with two Canadian races that have previously been classified for their reaction to the major resistance genes.

9. MATERIALS AND METHODS

The Kenyan cultivars used in this study and information on their pedigrees were provided by the National Plant Breeding Research Centre in Njoro, Kenya. The cultivar Bounty was obtained from Plant Gene Resources in Ottawa, Canada.

The two stripe rust isolates that were used, SRC 4-84 and SRC-89, have been described earlier in this manuscript (Section 4.2).

The Kenyan cultivars were double-seeded in random order within 8x4 rootrainer trays (Spencer-Lemaire Industries, Edmonton, Alberta) and replicated three times. There were eight plants of each Kenyan entry in each replicate. The check cultivars used in tests with isolate SRC 4-84 were Owens and Fielder, with the addition of SWS 52 in tests with SRC-89. The cultivar SWS 52 was developed at the Agriculture Canada Research Station in Lethbridge, Alberta from an F4 bulk of unknown crosses obtained from Aberdeen, Idaho (Sadasivaiah and Thomas, 1991a, 1991b). The inoculation procedure and classification system used were identical to those described previously.

Table 20.
Infection type response of Kenyan cultivars, and Owens and Fielder checks, screened with stripe rust races SRC 4-84 and SRC-89 (Springfield).

Cultivar	Year of release *	Infection type SRC 4-84	SRC-89
Kenya Chiriku	1989	22/22 Ty0 **	22/22 Ty0
Kenya Mlembe	1989	8/8 Ty0	24/24 Ty0
Pasa	1989	23/23 Ty0	21/21 Ty0
Kenya Tausi	1987	24/24 Ty0	19/24 Ty0; 5/24 Ty3
Mbuni	1987	23/23 Ty0	19/20 Ty3; 1/20 Ty0
Kwale	1987	24/24 Ty0	24/24 Ty0
Kenya Popo	1982	23/23 Ty0	24/24 Ty0
Kenya Kulungu	1982	24/24 Ty0	21/21 Ty0
Kenya Nyumbu	1982	22/22 Ty0	23/23 Ty0
Paa	1981	21/21 Ty0	21/21 Ty0
Kenya Nungu	1975	22/22 Ty4	14/22 Ty4;8/22 Ty2-3
Kenya Tembo	1975	22/22 Ty3-4	20/22 Ty3-4; 2/22 Ty1
Kenya Swara	1972	22/24 Ty3-4; 2/24 Ty1	20/21 Ty3-4; 1/21 Ty1
Bounty	1967	20/21 Ty3-4; 1/21 Ty0	20/21 Ty3-4; 1/21 Ty0
Kenya Leopard	1966	20/24 Ty3-4; 4/24 Ty0	24/24 Ty3-4
Romany	1966	21/21 Ty3-4	13/22 Ty3;9/22 Ty0-1
Kenya Page	1963	20/24 Ty3-4; 4/24 Ty0	21/23 Ty3-4; 2/23 Ty1
Africa Mayo	1960	18/24 Ty3-4; 6/24 Ty1	8/24 Ty3;16/24 Ty0-1
Fielder	1975	23/23 Ty4	17/23 Ty3; 6/23 Ty0
Owens	1984	24/24 Ty0-1	23/23 Ty4
SWS 52	1991	32/32 Ty0	32/32 Ty0

* Annual Reports NPBRC
** Number of plants with a specific infection type/ total number of plants

The only Kenyan cultivar for which the infection type changed significantly between screenings with the different races was Mbuni, which was resistant to SRC 4-84 but susceptible to SRC-89. Kenya Tausi was also resistant to SRC 4-84 but showed some susceptibility to SRC-89. The cultivars Kenya Nungu and Kenya Tembo were unambiguously

susceptible to SRC 4-84, but showed a few intermediate and resistant infection types when screened with SRC-89. Kenya Swara, Bounty, Kenya Leopard, Romany, Kenya Page and Africa Mayo all showed a few resistant plants among the predominantly susceptible infection types. All other cultivars tested were uniformly resistant to both stripe rust isolates.

The Kenyan cultivars released before 1975 (Table 20) were more susceptible to both Canadian races of stripe rust than those released later, possibly because they were not bred with any major gene resistance to stripe rust.

There are three sets of sister lines among the Kenyan cultivars tested: Kenya Tembo and Kenya Nungu, Kenya Kulungu and Kenya Nyumbu, and Kenya Chiriku and Kenya Mlembe (M. Kinyua, personal communication). Within pairs of sister lines, the three sets had similar stripe rust ratings against both Canadian isolates.

11. DISCUSSION

The cultivar Mbuni has remained moderately resistant to stripe rust in Kenya since its release in 1987 (Danial, personal communication). The genes Yr9 (from the 1B/1R wheat-rye translocation) and Yr2 (from Kalyansona) are known to be present in much of the CIMMYT derived germplasm, from which Mbuni was selected (Rajaram *et al.*, 1983). However, since neither of the Canadian races is virulent on Yr9, it appears that Mbuni lacks Yr9. Further evidence to support this conclusion, is that Mbuni also lacks the sticky dough trait caused by the secalin gene(s) that are known to be tightly linked to Yr9 on the 1RS chromosome (Singh *et al.*, 1990). From previous screenings of differential cultivars (Table 5) it was determined that the two Canadian races differ for virulence on Yr2 and Yr7 with SRC-89 being virulent on both genes, and SRC 4-84 virulent on neither. It follows that the effective resistance of Mbuni to Canadian stripe rust race SRC 4-84 is probably being conferred by Yr2 and/or Yr7, or a previously unidentified resistance gene. In addition to Yr2 or Yr7, the cultivar Mbuni may also carry resistance genes to which both Canadian races are virulent e.g. Yr3a, Yr4a or Yr6. Since virulence for Yr2 and Yr7 was present in Kenya when Mbuni was released (Stubbs, 1988), the cultivar is probably protected against Kenyan stripe rust races by other genes in addition to Yr2 and Yr7. Only a few plants of Kenya Tausi were susceptible to race SRC-89 so it could be protected by several resistance factors, some of which are still segregating. The susceptible plants could also possibly be off-types resulting from seed mixture. Kenya Tausi shows a low incidence of infection in Kenya which suggests either that it has some partial resistance, or that the virulent inoculum is still building up. Since the cultivars were tested at the seedling stage, the expression of adult plant resistance is not expected.

Based on the information from this study, it is also possible to speculate on the resistance of another Kenyan cultivar, Kenya Kulungu. According to Badebo *et al.* (1990), who screened a number of Ethiopian lines and cultivars with 19 different isolates of stripe rust from an international collection, the cultivar Kenya Kulungu (= Har 472) was only susceptible to races having virulence for Yr4b+ (in the differential Hybrid 46). The + sign was used to indicate the presence of other, unidentified resistance genes in the differential cultivar. Hybrid 46 also possesses the gene Yr3b (Lupton and Macer, 1962). The added presence of Yr2 (in the differential Heines VII) and/or Yr3a and Yr4a (in Vilmorin 23) could not be unambiguously proven or disproved because all races tested with virulence for Yr4b+ were also virulent on those genes (Badebo *et al.*, 1990). The rust isolates with virulence on Hybrid 46 were from the Netherlands. Race 108E141 with virulence for Yr2, 3a, 3b, 4a, 4b, and 6 was virulent on Kenya Kulungu suggesting that the remaining genes Yr1, 5, 7, 8, 9, 10 and A are not present in the genetic composition of Kenya Kulungu. Race 232E137 does not have virulence for Yr6 but it was virulent on Kenya Kulungu, so Kulungu does not possess Yr6. The remaining possibilities for the resistance factor in Kenya Kulungu are Yr2, 3a, 3b, 4a and 4b, or some combination thereof.

However, the Canadian isolate SRC-89(Springfield) was virulent on Yr2, Yr3a, Yr3c, Yr4a, Yr4b, Yr6 and Yr7 and avirulent on Yr1, Yr3b, Yr5, Yr8, Yr9 and Yr10. It is also avirulent on Kenya Kulungu. Therefore the Yr2 resistance, in Heines VII, and the Yr3a and Yr4a, in Capelle Deprez, are ineffective against race SRC-89. The Yr4b resistance, in Opal, is ineffective as well. Based on this information and the results of Badebo *et al.* (1990), the resistance in Kenya Kulungu against Canadian isolates of stripe rust is conferred either by Yr3b or a previously unidentified gene. Whether or not Kenya Kulungu also possesses Yr4b cannot be proven until the races used by Badebo *et al.* (1990) have been

tested on lines bearing Yr3b and Yr4b separately. Because isolate SRC-89 is also virulent on Yr2 and Yr4a, it is still not possible to discount the hypothetical existence of these genes in Kenya Kulungu. Their ineffectual presence would be masked behind the reaction of the effective resistance gene. Only one of the Yr4 alleles would be present. Among the Ethiopian stripe rust isolates, only the virulences in Hybrid 46 (Yr3b, Yr4b) and in *Triticum spelta (Yr5)* remain undetected (Badebo *et al.*, 1990).

The cultivars Kenya Tembo, Kenya Swara, Bounty, Leopard, Romany and Kenya Page all scored a few resistant plants. This could be the result of heterogeneity or, possibly, heterozygosity of resistance loci. Cultivars may be heterogeneous for several reasons including admixing or segregation from the original cross, particularly in populations that were bulked at an early generation (Harrington, 1929). There could also be a persistent low level of heterozygosity caused by segregation from outcrossing (Heyne and Smith, 1967) or chromosomal irregularities (Riley and Kimber, 1961). Alternatively, the presence of residual levels of stripe rust resistance could be another reason why a few resistant plants screened out from the cultivars listed above (Brodny *et al.*, 1986). The cultivar Bounty has been reported to possess the race specific gene Yr1 as well as a gene for adult-plant resistance Yr13 (Taylor *et al.*, 1981). Adult-plant resistance is not expected to be a factor in seedling tests, but even the postulation of Yr1 is inconsistent with the observation that Bounty was mostly susceptible to both Canadian races although neither race has virulence for the gene Yr1 (in Chinese 166). It is possible that the cultivar Bounty used in tests by Taylor *et al.* (1981) was a different genotype from the Kenyan cultivar Bounty (Zeven and Zeven-Hissink, 1976). In the cultivar Africa Mayo very few Type 0 or Type 4 plants were recorded, an observation that was masked by the compilation of data into resistant (Type 0-1) and susceptible (Type 3-4) infection types (Table 20). This intermediate reaction in Africa Mayo

could be due to the presence of some level of durable resistance that persisted even after the cultivars race specific resistance had been defeated. It could also be due to a race-specific gene conditioning a low infection type (Type 2), possibly effective in the latter stages of pustule formation.

Stripe rust symptoms under growth cabinet conditions, particularly those of the Canadian race SRC-89 (Springfield) are strongly influenced by light parameters. In addition, some cultivars appear to be more sensitive to the light environment than others (R. Conner, personal communication). The cultivar Fielder, used as a susceptible check, has no effective resistance genes to race SRC-89 and yet it consistently produces at least a few plants with Type 0 reactions. The progeny from these plants have been shown to be susceptible in subsequent tests. For this reason, the occurrence of a small number of resistant plants in screenings with either race, but especially with SRC-89, could be an environmentally induced escape.

12. CONCLUSION

Eight of the eighteen Kenyan cultivars tested were uniformly resistant to both Canadian races. The cultivar Mbuni was resistant to race SRC 4-84 but mostly susceptible to race SRC-89, so its effective resistance against SRC 4-84 is probably being conferred by Yr2 and/or Yr7. Kenya Tausi was resistant to SRC 4-84 and showed only a few plants that were susceptible to SRC-89, so it is probably protected by several genes. Based on information from this study and on the data of Badebo *et al.* (1990) who screened a number of East African cultivars with international races, effective resistance in the cultivar Kenya Kulungu is being conferred by Yr3b or a previously unidentified gene. The cultivars Kenya Tembo, Kenya Swara, Bounty, Kenya Leopard, Romany, Kenya Page and Africa Mayo scored a few resistant plants among their predominantly susceptible responses. This could be the result of heterogeneity in the cultivar, or residual levels of resistance, or perhaps a race-specific gene conditioning a partly resistant response.

13. GENERAL CONCLUSION

The soft white spring wheat cultivar, Owens, possesses one effective, dominant gene of resistance against the Canadian stripe rust race SRC 4-84. This homozygous resistance is effective at the seedling stage. The gene in Owens is not Yr1, Yr3a, Yr3b, Yr3c, Yr4a, Yr4b, Yr5, Yr6, Yr7, Yr8 or Yr10. It is unlikely to be Yr9 or Yr15. The remaining, untested seedling resistance factors are Yr2 and YrA. The possibility cannot be dismissed that the gene in Owens is one that has never been described. The operative resistance against race SRC 4-84 in Hybrid 46 (Yr3b, Yr4b) was found to be acting recessively. This gene is most likely to be Yr3b or a previously undetected gene in the cultivar.

Eight of the Kenyan cultivars (K. Chiriku, K. Mlembe, Pasa, Kwale, K. Popo, K. Kulungu, K. Nyumbu and Paa) tested with stripe rust races SRC 4-84 and SRC-89 were uniformly resistant to both races. The cultivar K. Nungu was susceptible to both races. Resistance against SRC 4-84 in the cultivar Mbuni is probably conferred by Yr2 and/or Yr7. The cultivar Kenya Kulungu is protected against race SRC 4-84 by Yr3b, or a previously unidentified gene. The reaction of seven, older cultivars (K. Tembo, K. Swara, Bounty, K. Leopard, Romany, K. Page and Africa Mayo) to both races rated primarily susceptible but with a few resistant plants, a response that suggests the presence of residual resistance.

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