INHERITANCE OF TAN SPOT RESISTANCE IN HEXAPLOID, TETRAPLOID, AND DIPLOID WHEAT

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Ellen Elizabeth Sykes

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GENERAL ABSTRACT

Sykes, Ellen Elizabeth. Ph.D., The University of Manitoba, August, 1989.
Inheritance of tan spot resistance in hexaploid, tetraploid, and diploid wheat.
Major Professor: Dr. C.C. Bernier.

The inheritance of resistance to one Manitoban isolate (ASC1) of the tan spot fungus (Pyrenophora tritici-repentis (Died.) Drechs.; anamorph, Drechslera tritici-repentis (Died.) Shoem.) was studied in month-old (Feekes 4-5) plants of hexaploid, tetraploid, and diploid wheat under growth room conditions. In contrast to previous inheritance studies of tan spot resistance, a qualitative disease assessment criterion, lesion morphology or type (described by Lamari and Bernier, 1989), was applied. A qualitative genetic approach (Chi-square goodness of fit tests to expected F_2 genetic ratios) was used. Qualitative inheritance of tan spot resistance was observed within all three ploidy levels.

For the hexaploid inheritance study, F_1 results of 28 crosses and reciprocals among eight parents (ranging from resistant to susceptible) indicated that tan spot resistance may appear completely dominant, incompletely dominant, or recessive, depending on the genetic background involved. Of the 28 crosses, 18 were selected for further study in the F_2 generation. Qualitative inheritance of tan spot resistance was observed in several crosses. Although variability in the F_2 data was observed, the results indicated that the resistant parents, Salamouni, Carifen12, and Erik, likely had in common one recessive gene pair for resistance. BCF_1 results of three resistant x susceptible or moderately susceptible crosses involving Salamouni (Salamouni with Columbus, Glenlea, and HY320) further supported the F_2 results under the assumption of incomplete dominant gene action. BCF_1

results of the resistant x moderately resistant cross, Salamouni with BH1146, indicated that the two parents differed for resistance gene(s), in agreement with the F_2 results. BCF_1 results of the resistant x resistant cross, Salamouni with Carifen12, indicated that these resistant parents shared the same gene(s) for resistance, also in agreement with the F_2 results. F_3 family data for the resistant x moderately resistant cross, Salamouni with BH1146, suggested that Salamouni and BH1146 differed for resistance genes. F_3 results of the resistant x moderately susceptible cross, Salamouni with Glenlea, indicated that Salamouni possessed one gene for resistance, likely incompletely dominant. Results of all other crosses were inconclusive; genetic heterogeneity in the parents, growth stage effects, incomplete dominant gene action, or modifier genes may have been responsible. Surprisingly, in a cross between the moderately susceptible parent, HY320 and the susceptible parent, Columbus, a few resistant progeny were obtained. It is likely that the two "susceptible" parents of the cross differed for minor genes for resistance and that the resistant progeny arose from an accumulation of these minor genes. In addition, an apparent reciprocal difference was noted in this cross, but further testing is needed to verify this result. Reciprocal differences were absent in the F_2 generation of the other 17 crosses studied.

Ten crosses (including reciprocals) among five parents, ranging from moderately resistant to susceptible, comprised the tetraploid inheritance study. Qualitative inheritance for resistance was detected although suspected genetic heterogeneity in the parents, the use of parents with only moderate resistance, incompletely dominant gene action (suggested by the F_1 data), and small population sizes made interpretation difficult. F_2 results

for both the 2-leaf and 4-6 leaf growth stages indicated that the moderately resistant parent, 4B175 possessed one recessive gene pair for resistance. F_2 results were variable for crosses involving the other moderately resistant parents, 4B233 and 4B242, but suggested that they shared the resistance gene(s) of 4B175. Based on F_1 and F_2 data, no reciprocal differences were noted in the tetraploid crosses.

For the diploids, results indicated only qualitative inheritance of tan spot resistance. F_1 data indicated that resistance was recessive and F_2 results confirmed this. F_2 data demonstrated that the resistant parents, 2B27 and 2B53, each carried one recessive gene pair for resistance which may or may not be common to both. The resistant parents, 2B27, 2B26, and 2B7 were shown to possess the same resistance gene or genes.

GENERAL INTRODUCTION

Tan spot or yellow spot, is an important foliar disease of wheat worldwide. Significant yield losses (Luz and Hosford, 1980; Rees et al., 1981; Rees et al., 1982; Tekauz et al., 1982), reduced test weight (Sharp et al., 1976), and a pink discolouration of infected kernels known as "red smudge" resulting in reduced quality (Vanterpool, 1963; Lyster, 1987) have been reported. Tan spot is presently a major foliar disease of wheat in North Dakota (Hosford, 1982) and the Canadian prairies (Tekauz et al., 1982).

Tan spot is caused by the ascomycetous fungus, Pyrenophora tritici-repentis (Died.) Drechs. (anamorph, Drechslera tritici-repentis (Died.) Shoem.). The fungus has a broad host range, including more than 33 grass species (Hosford, 1982). Wheat is a susceptible host, but barley, oats, and rye are highly resistant (Wiese, 1977; Larez et al., 1986).

In the past, various quantitative criteria such as lesion numbers (Nagle et al., 1982; Raymond et al., 1985), lesion size (Cox and Hosford, 1982; Raymond et al., 1985), and disease severity (which may take leaf age and growth stage into account) (Misra and Singh, 1972; Luz and Hosford, 1980; Gough and Johnston, 1982; Raymond et al., 1985; Cox and Hosford, 1987) have been used to assess resistance/susceptibility of wheat to the tan spot fungus. These quantitative criteria have been applied in inheritance studies of the host and in virulence studies of the pathogen.

Few studies of the inheritance of resistance to tan spot of wheat have been conducted. Using the quantitative criterion, disease severity (based on %leaf area affected), Nagle et al. (1982) examined six hexaploid wheat crosses for resistance to the PyW17 isolate. For each cross, F_2 and BC_1F_1 results did not fit expected monogenic or digenic ratios. Tan spot

resistance was therefore considered a quantitatively inherited trait. Hence, a quantitative genetic approach was begun; Griffing's F_1 diallel analysis of a 10-parent diallel was undertaken. General combining ability (GCA) and specific combining ability (SCA) were significant for two quantitative disease assessment criteria, %leaf area affected and number of lesions/cm.^{2}. GCA significantly exceeded SCA for both criteria, suggesting that additive effects were much more important than nonadditive effects (i.e. dominance and epistasis) in determining tan spot reaction.

Using the quantitative disease assessment criterion, %leaf area affected, lack of fit to expected monogenic or digenic ratios in F_2's and BC_1F_1's of five tetraploid crosses led to the conclusion that tan spot resistance within tetraploid wheat is also inherited quantitatively (Nagle et al., 1982). A quantitative genetic approach was followed by Cantrell et al. (1985). They obtained low to moderate heritability estimates from testing F_4 and F_5 lines of two resistant x susceptible tetraploid crosses. A quantitative genetic approach was also used by Elias et al. (1989). They observed significant additive genetic variance in the F_5 generation of one tetraploid cross, and obtained a high narrow sense heritability estimate.

Quantitative disease assessment criteria have also been used in virulence studies of the pathogen. In a study from India, three isolates of the tan spot fungus were reported to differ in virulence since they exhibited differential reactions across groups of wheat lines (assessed on the basis of %leaf area affected) (Misra and Singh, 1972). In an American study, 40 P. tritici-repentis isolates from the Central Plains were separated into 12 different virulence groups by six wheat cultivars (assessed on the basis of %leaf area affected) (Luz and Hosford, 1980).

In contrast to these earlier studies, a qualitative disease assessment criterion, lesion type, has recently been used successfully to measure genotypic differences among wheat lines and to differentiate virulence groups of the pathogen (Lamari and Bernier, 1989). A rating scale comprised of five lesion types was used: Type 1 were described as small dark spots lacking surrounding chlorosis or tan necrosis (resistant); Type 2 lesions were similar to Type 1 but had margins comprised of a little chlorosis and/or tan necrosis (moderately resistant); Type 3 (moderately resistant to moderately susceptible), Type 4 (moderately susceptible), and Type 5 (susceptible) lesions differed in amount of surrounding chlorosis and/or tan necrosis and in degree of lesion coalescence. Using this qualitative disease assessment criterion, genotypic differences for reaction to one isolate of the tan spot fungus were detected among 695 accessions of diploid, tetraploid, hexaploid, and octoploid wheat. Lesion type has previously been used successfully in the Pyrenophora teres (Drechs.)-barley system (Tekauz, 1985).

The qualitative criterion, lesion type, was also used to separate 92 isolates of the tan spot fungus from western Canada into three virulence groups (pathotypes) (Lamari, 1988). An additional lesion type, characterized by extensive chlorosis, was important in differentiating the three pathotypes. Pathotype 1 (nec^+ ch^+) induced tan necrosis or extensive chlorosis on appropriate wheat hosts; pathotype 2 (nec^+ ch^-) induced only tan necrosis; pathotype 3 (nec^- ch^+) induced only extensive chlorosis on appropriate hosts. In addition, isolates capable of inducing necrosis (nec^+ types) were found to produce a toxin in vitro.

Toxin production by the tan spot fungus was reported only recently (Tomas et al., 1986). A relationship between host susceptibility to the

fungus and sensitivity to toxin has been shown (Tomas and Bockus, 1987; Lamari, 1988). Lamari (1988) reported that one dominant gene controlled both susceptibility to the fungus and toxin sensitivity. The toxin is a single protein of 9,300 molecular weight and is considered to function as a pathogenicity factor (Lamari et al., in press). Although a toxin may play a role in determining resistance/susceptibility at the cellular level, the factors responsible for the expression of resistance at this level (i.e. restriction of intercellular mycelial growth) are presently unknown (Larez et al., 1986; Loughman and Deverall, 1986; Lamari, 1988).

There has been only one published report of qualitative inheritance of tan spot resistance. The resistance of the hexaploid winter wheat cultivar, Carifen12, was attributed to one pair of recessive genes (Lee and Gough, 1984). Tan spot reaction was assessed on a qualitative basis; plants were classified as either resistant or susceptible.

The primary objective of this study was to further investigate the inheritance of qualitative resistance to tan spot (assessed on the basis of lesion type) recently found in hexaploid, tetraploid, and diploid wheat (Lamari and Bernier, 1989). A qualitative genetic approach (involving Chi-square goodness of fit tests to expected F_2 ratios) was followed.

LITERATURE REVIEW

1. Introduction to tan spot of wheat

1.1 History

The foliar disease, tan spot (also known as yellow spot), was first reported in 1902 on grass hosts in Germany (Hosford, 1982). In 1928, it was first identified on wheat in Japan. Tan spot is presently an important disease of wheat throughout the world. In the early 1980's, tan spot was reported as the major foliar disease of wheat in North Dakota (Hosford, 1982) and the Canadian prairies (Tekauz et al., 1982).

Tan spot is caused by an ascomycete fungus (Hosford, 1982). Previously, the teleomorph had been described as a member of Sphaeria, Pleospora, and Pyrenophora genera. The anamorph had been placed into the genera, Helminthosporium and Drechslera. The tan spot fungus is presently known as Pyrenophora tritici-repentis (Died.) Drechs. (anamorph, Drechslera tritici-repentis (Died.) Shoem.).

The tan spot fungus has a broad host range, and is known to infect more than 33 species of grasses (Hosford, 1982). Wheat is a susceptible host, but barley, oats, and rye are highly resistant (Wiese, 1977; Larez et al., 1986).

1.2 Symptoms and effects of the disease

The term "tan spot" aptly describes the visible symptoms of this foliar disease. Typical lesions on a susceptible host are elongated light brown spots (approximately 0.5 cm. x 1.0 cm.) of tan necrotic tissue containing a central small dark spot; the lesions are bounded by a zone of yellow chlorotic tissue (Lamey and Hosford, 1982). Lesions can coalesce over time

resulting in eventual death of the entire leaf. Infection of the seed may also occur, sometimes causing a pinkish discolouration known as "pink smudge" or "red smudge" (Vanterpool, 1963; Lyster, 1987).

Tan spot has caused significant yield losses in many countries throughout the world (Hosford, 1982) including the U.S.A. and Canada (Luz and Hosford, 1980; Tekauz, 1976; Tekauz et al., 1983; Wiese, 1984). Losses due to tan spot in both common and durum wheat have been increasing since the 1970's in the Central Plains of the U.S.A. and Canada (Luz and Hosford, 1980; Cantrell, 1982; Tekauz et al., 1982). The increase in minimum tillage practices, which favours overwintering of the fungus, may be partly responsible (Cantrell, 1982; Gough and Johnston, 1982; Sebesta, 1982; Tekauz et al., 1982). In North Dakota, average annual losses of 7.4% with losses of 30-40% in individual fields have been reported (Hosford, 1982). Yield losses from all foliar diseases including tan spot were estimated at 3-4% in western Canada (Tekauz, 1976). Lower test weight has also been reported due to tan spot infection (Sharp et al., 1976). Reduced seed grade due to pink smudge symptoms is a concern particularly in durum wheat (Vanterpool, 1963; Lyster, 1987).

1.3 Disease cycle

The disease cycle of the tan spot fungus is comprised of a sexual phase and an asexual phase. With regard to the sexual phase, the fungus is considered homothallic (Hosford, 1971). The pathogen is classified ecologically and nutritionally as a necrotroph, causing extensive tissue damage to the host in its parasitic phase, and confined largely to dead or dying host plant tissue in its nonparasitic phase (Webster, 1980).

In North America, the tan spot fungus overwinters in pseudothecia (small, black sexual fruiting bodies) on grass and wheat straw and stubble. In the spring, ascospores (and perhaps some conidiospores) are released and can infect young wheat plants nearby (Lamey and Hosford, 1982). Infected seed has been reported as a source of primary infection in Kenya, but is likely not an important initiator of the disease in North America (Hosford, 1982). The fungus sporulates in the lesions and releases conidiospores (asexual spores). Air currents can carry the conidiospores to neighbouring plants and distant fields (Platt and Morrall, 1980; Lamey and Hosford, 1982). Mycelial fragments may also become windborne and serve as inoculum (Hosford, 1972). Infection occurs on wheat plants wet with dew or rain (Lamey and Hosford, 1982). Lesions resulting from conidiospore infection support further conidiospore production and the disease progresses. Cool damp weather favours development of the epidemic. As the host matures, pseudothecia form on dead or dying host tissue.

1.4 Factors affecting development of the fungus and the disease

Both in vitro and in vivo studies have identified factors which affect the development of the tan spot fungus. The effect of nutritional factors on the asexual stage has been studied in vitro. The fungus can be grown and maintained on potato dextrose agar (PDA) medium (Hosford, 1982). For the production of conidiospores, V-8 medium is normally required (McDonald, 1963, cited by Hosford, 1982). To facilitate inoculum production, a modified medium comprised of wedges of one-fourth strength PDA in V-8 agar medium was utilized by Raymond and Bockus (1982). A V-8/PDA blend medium (850 mls. distilled water, 150 mls. V-8 juice, 10 g. PDA, 10 g. agar, 3 g. calcium

carbonate per liter) was developed by Lamari (1988) and used to promote sporulation in many tan spot fungus isolates.

The duration of the light/dark period, light intensity, relative humidity (R.H.), and temperature have all been found to affect conidiation. Both light and darkness are necessary for asexual reproduction; light was found to initiate conidiophore production and darkness was found to promote conidiospore development (Khan, 1971). A light intensity of 13.3 W./m.^{2} supported maximum conidiospore production; relative humidities of 83-85% allowed some condiospore production, but maximum conidiation occurred at 100% R.H. (Platt and Morrall, 1980). Conidiophores were produced over a temperature range of 10-31^{o}C and conidiospores at 10-25^{o}C, with an optimum temperature for conidiospore production of 21^{o}C (Platt et al., 1977). Temperature was also found to affect the sexual stage; cool temperatures (15^{o}C or less) for approximately 8 weeks were required for ascospore maturation (Odvody et al., 1982).

The spread of tan spot disease and the development of epidemics are affected by factors such as relative humidity, wind speed, and light/dark period. Relative humidities of 31-100% with a wind speed of 3.3 m./s. brought about almost complete liberation of conidiospores (Platt and Morrall, 1980). The effects of the light/dark period on conidiospore production were believed to underlie the observed diurnal rhythm (peaking at 1200 hours) in numbers of airborne conidiospores detected over native grasslands in the Canadian prairies (Morrall and Howard, 1975).

1.5 Disease control

Cultural methods, chemicals, and resistant cultivars can be used in the control of tan spot of wheat. Cultural methods such as crop rotation and cultivation to bury infected straw and stubble have been recommended in the U.S.A. (Lamey and Hosford, 1982), Australia (Rees and Platz, 1980), and Canada (M.A.S.C.C., 1986). Application of high levels of nitrogen has been reported effective in reducing tan spot severity (Huber et al., 1987).

Chemical methods for tan spot control rely on fungicides. Mancozeb (Dithane M-45, Manzate 200) has been used in North Dakota and western Canada (Tekauz et al, 1983; Lyster, 1987) and triadimefon (Bayleton) is registered in Europe (Tekauz et al., 1983) and western Canada (Manitoba Agriculture, 1988). Because both are foliar protectants, more than one application may be required, thus crop yields must justify the costs of the chemicals. A foliar-applied fungicide with systemic properties, propiconazole (Tilt), has been registered in western Canada recently and may prove to be more cost-effective (Manitoba Agriculture, 1988). In North Dakota, a systemic seed dressing, triadimenol, provided up to 35 days of early season control of tan spot in field trials (Luz and Bergstrom, 1986).

Growing genetically resistant cultivars is often the most effective and economical means of disease control. In the U.S.A., a number of cultivars with some degree of resistance to the tan spot fungus are available (Wiese, 1977; Gough and Johnston, 1982; Cox and Hosford, 1987). Resistant cultivars are presently unavailable in Australia (Rees and Platz, 1980) and western Canada (M.A.S.C.C., 1986).

2. Tan spot disease reaction: resistance and susceptibility

2.1 Definitions

Disease resistance has been defined in numerous ways. Resistance in the tan spot-wheat system was defined in genetic terms by Nagle et al. (1982). Resistance governed by one or two genes was termed qualitative and resistance controlled by more than two genes was referred to as quantitative; in this study, qualitative refers to control by one to several genes. Nelson (1973) defined resistance in a non-genetic sense and stressed that resistance and susceptibility were relative terms, used to describe the amount of disease sustained by a plant or the amount of damage resulting from the disease. In the tan spot-wheat system, resistance has generally been taken to mean "less disease", i.e. resistant lines display less diseased, damaged, necrotic and/or chlorotic tissue.

2.2 Assessment of tan spot disease reaction

Various quantitative criteria have been used to assess resistance/susceptibility to the tan spot fungus: lesion number, lesion size, %leaf area affected, and disease severity (which may take into account leaf age or growth stage of the plant). In comparison with susceptible lines, resistant lines have been described as having fewer lesions/cm.^{2} (Nagle et al., 1982; Raymond et al., 1985), smaller-sized lesions (Raymond et al., 1985; Cox and Hosford, 1987), less %leaf area affected (Nagle et al., 1982), and lower disease severity ratings as measured by various rating scales (Misra and Singh, 1972; Luz and Hosford, 1980; Gough and Johnston, 1982; Raymond et al., 1985; Cox and Hosford, 1987). Disease reaction has been assessed in a similar manner in other necrotrophic systems, such as the

septoria diseases and helminthosporium leaf blight of wheat (Cunfer, 1987).

In contrast, a qualitative criterion, lesion morphology or type, has recently been used to assess disease reaction in the P. tritici-repentis-wheat system (Lamari, 1988). Five lesion types, ranging from resistant to susceptible, were defined by Lamari and Bernier (1989). An additional susceptible lesion type, characterized by extensive chlorosis, was useful in differentiating different pathotypes of the fungus (Lamari, 1988). Lesion type was used to differentiate resistance and susceptibility in the closely related Pyrenophora teres (Drechs.)-barley system (Tekauz, 1985). In this latter case, resistance measured by lesion type could be related to reduced conidiospore production (Keeling and Banttari, 1975).

Although measurement of the amount of diseased tissue (by either quantitative or qualitative criteria) has been the most common method of assessing tan spot disease reaction, other criteria have also been used to characterize resistance and susceptibility. Disease progress has been measured and resistant lines were found to exhibit slower rates of disease progress (Raymond et al., 1985). Percentage yield loss due to disease has also been used as an evaluation criterion; resistant lines had expressed lower %yield loss than susceptible lines (Rees and Platz, 1983; Raymond et al., 1985).

2.3 Factors which affect tan spot disease expression

Factors other than the genetic nature of the host and the pathogen have been shown to affect tan spot disease expression. Duration of the leaf wetness period, ambient temperature, age of the inoculated leaf, growth stage of the plant, and fertilizer status have all been reported to affect disease

expression in the tan spot-wheat system.

Several studies have reported that the duration of the leaf wetness period following inoculation had an effect on the expression of resistance/susceptibility. When assessed by a rating scale based on the quantitative criterion, %leaf area affected, disease symptoms developed in susceptible lines after relatively short leaf wetness periods (<24 h.), but resistant lines required longer leaf wetness periods (48-54 h.) before becoming diseased (Hosford, 1971; Hosford, 1982; Hosford et al., 1987). In a similar study recently conducted at the University of Manitoba, in which disease reaction was assessed by the qualitative criterion, lesion type, no change in disease reaction was observed in seedlings of 66 lines subjected to 48, 54, 60, and 72 h. leaf wetness periods following inoculation with the ASC1 isolate of the tan spot fungus (Lamari and Bernier, 1989). The different criteria used to evaluate disease reaction likely contributed to the different results of this study and the earlier ones.

Post-inoculation temperature has been shown to affect tan spot disease expression, and at lower temperatures, symptoms developed more slowly (Luz and Bergstrom, 1986; Hosford et al., 1987). In one study, 18-20^{o}C was reported as the optimum temperature range for disease expression and development, with slightly different optima for different cultivars (Luz and Bergstrom, 1986).

Growth stage and leaf age have also been found to affect disease expression. Quantitative tan spot ratings of 14 resistant winter wheat cultivars were more severe on seedlings, on average, than on adult plants (Cox and Hosford, 1987). Regardless of growth stage, tan spot disease expression has always been less on the youngest leaf (Raymond et al., 1985;

Cox and Hosford, 1987). Various quantitative rating scales have been devised to incorporate the effect of leaf age on disease expression (Hosford, 1982; Raymond et al., 1985; Cox and Hosford, 1987).

The addition of fertilizer may also affect disease expression. Tan spot severity under field conditions was reduced by high rates of applied nitrogen (Huber et al., 1987).

2.4 The physiological basis of tan spot resistance

Microscopy studies of the infection process have served as a starting point for investigations into the physiological basis of tan spot resistance. Resistant and susceptible wheat lines did not differ greatly in the major events of the infection process (i.e. conidiospore germination, appressorial formation, penetration of the epidermis, formation of intracellular vesicles, and intracellular hyphal growth) (Larez et al., 1986; Loughman and Deverall, 1986; Lamari, 1988). The high level of resistance of Lodi oats was attributed to greater papillae production (Larez et al., 1986), but differences in papillae production were not significant between resistant and susceptible wheat cultivars (Larez et al., 1986; Loughman and Deverall, 1986). Tan spot resistance in wheat was apparently not expressed until the fungus reached the intercellular spaces of the mesophyll. Resistant hosts restricted intercellular mycelial growth to a greater extent than susceptible hosts. The factors responsible for the restriction of growth are presently unknown, (Larez et al., 1986; Loughman and Deverall, 1986; Hosford et al., 1987; Lamari, 1988). A cultivar-specific toxin produced by the fungus may be an important determiner of resistance/susceptibilty (Tomas et al., 1986; Tomas and Bockus, 1987; Lamari, 1988).

2.5 The role of toxin in tan spot disease

The first report of toxin produced by the tan spot fungus was by Tomas et al. in 1986. In a subsequent study, all nine isolates of the tan spot fungus tested were found to produce filtrates toxic to the wheat host (Tomas and Bockus, 1987). When the cell-free toxic filtrates were applied to a tan spot susceptible cultivar, typical tan spot symptoms were induced. Although indisputable evidence for a causal role of toxin in the disease syndrome was not established, a high correlation between suceptibility to the pathogen and sensitivity to the toxic filtrates was observed among the 10 wheat cultivars tested.

In a similar study, Lamari (1988) obtained toxic filtrates from a number of tan spot fungus isolates. The same relationship between pathogen susceptibility and toxin sensitivity was observed. However, all isolates in this study did not produce toxic filtrates -- isolates capable of inducing necrosis in host lines (nec^+ types) produced toxin in culture, while isolates which induced only extensive chlorosis (nec^- ch^+ types) did not. F_2 and BCF_1 genetic data indicated that one dominant gene controlled both susceptibility to the fungus and sensitivity to toxin. However, the cultivar Columbus was found to be comprised of toxin sensitive and toxin insensitive components, but both were susceptible to the tan spot fungus. Although confirmatory genetic studies would be required, perhaps toxin sensitivity/insensitivity and fungus susceptibility/resistance may be governed by a minimum of two individual but closely linked genes. The toxin discovered by Lamari (1988) is considered a pathogenicity factor by Lamari and Bernier (1989). It is now known to be a protein of 9,300 molecular weight (Lamari et al., in press).

3. Variability in the pathogen

3.1 Variability for virulence

In a study from India, the quantitative criterion, %leaf area affected, was used to assess the reaction of 50 wheat lines to three isolates of the tan spot fungus (Misra and Singh, 1972). The three isolates used resulted in differential reactions in the wheat lines, and were thus reported to differ in virulence.

The quantitative criterion, %leaf area affected, was also used in an American study to assess the reaction of six wheat host differentials to tan spot (Luz and Hosford, 1980). Forty isolates of the tan spot fungus from the Central Plains could be separated into 12 different virulence groups by the six wheat cultivars.

In contrast, the qualitative criterion, lesion type, was used in a recent Canadian study to separate 92 isolates from western Canada into three virulence groups or pathotypes (Lamari, 1988). Wheat lines which exhibited extensive chlorosis, a previously unreported lesion type, were useful in differentiating the pathotypes. Pathotype 1 (nec^+ ch^+) was defined as having the capacity to induce tan necrosis or extensive chlorosis on differential wheat lines; pathotype 2 (nec^+ ch^-) could induce only tan necrosis; pathotype 3 (nec^- ch^+) could only induce extensive chlorosis. The ability of isolates to induce necrosis was found to be related to their capacity for producing toxin.

3.2 Variability for aggressiveness

There is apparently only one report in the literature describing variability for aggressiveness in the tan spot fungus. Hunger and Brown

(1987) measured average lesion length to detect differences in aggressiveness among nine isolates originating from single ascospores. All isolates were virulent on the wheat cultivar TAM 101, but two "white" isolates and one "cream-coloured" isolate produced the longest lesions on average, i.e. were more aggressive than the others.

4. Variability in the host and inheritance studies of tan spot resistance

4.1 Genotypic variability in the host

Genotypic variability for tan spot reaction in wheat has been observed although different isolates, inoculation methods, and quantitative disease assessment criteria have been used in the various studies (Misra and Singh, 1972; Cantrell, 1982; Gough and Johnston, 1982; Nagle et al., 1982; Sebesta, 1982; Tekauz et al., 1982; Lee and Gough, 1984; Cantrell et al., 1985; Claude, 1985; Cox and Hosford, 1987; Wong and Hughes, 1987).

In a recent Manitoban study, the qualitative disease assessment criterion, lesion type, was used to demonstrate genotypic variability among 695 wheat accessions for reaction to one isolate of the tan spot fungus (Lamari and Bernier, 1989). High levels of resistance were identified within diploid, tetraploid, hexaploid, and octoploid wheat.

4.2 Inheritance of tan spot resistance in hexaploid wheat

Very few inheritance studies of the resistance to tan spot in wheat have been conducted. Nagle et al. (1982) examined six hexaploid wheat crosses for resistance to the PyW17 isolate of the tan spot fungus. The susceptible line, ND495, was crossed to six resistant lines, Eklund, ND7716, Sundance, ND7575, Saratovskaja 29, and BH1146. The quantitative criterion, disease

severity (based on %leaf area affected), was used to assess resistance/susceptibility. For each cross, the F_2's did not fit expected monogenic or digenic ratios, rather, a continuous distribution of tan spot ratings between the two parents was observed. For each cross, BC_1F_1 results also differed from monogenic or digenic expectations. It was concluded that the inheritance of tan spot resistance was controlled by more than one or two genes, and was therefore considered to be quantitative.

As part of the same study, a diallel was conducted in order to investigate further the apparent quantitative nature of resistance. Ten parents, ranging from highly resistant to highly susceptible, ND7575, Eklund, Sundance, BH1146, ND7716, Saratovskaja 29, ND7611, Len, Coteau, and ND495 were intercrossed without reciprocals. Griffing's F_1 diallel analysis indicated significant general combining ability (GCA) and significant specific combining ability (SCA) for two quantitative disease assessment criteria, %leaf area affected and number of lesions/cm.^{2}. GCA exceeded SCA significantly for both criteria, suggesting that additive effects were much more important than nonadditive effects (dominance and epistasis) in determining tan spot reaction. Individual gca effects indicated that the six resistant parents differed in their ability to transmit resistant factors to their progeny. ND7716, Eklund, BH1146, and ND7575 performed well in hybrid combination and were identified as good parents for use in breeding for tan spot resistance.

Quantitative inheritance has been reported in wheat for resistance to other necrotrophic pathogens. Resistance to Leptosphaeria nodorum E. Muller (anamorph, Septoria nodorum (Berk.) Berk.) is apparently quantitative (Cunfer, 1987). The most important types of resistance to Mycosphaerella

graminicola (Fuckel) Schroeter (Septoria tritici Rob. & Desm.) are also inherited quantitatively (Cunfer, 1987).

There is only one report in the literature of qualitative inheritance of tan spot resistance. The hexaploid winter wheat cultivar, Carifen12, was found to carry one recessive gene pair for resistance to the PYOK-2 isolate (Lee and Gough, 1984). Four F_1 plants of the cross, Carifen12 x TAM W-101, were the source of 141 F_3 families used in the study. Ten to 30 seedlings per F_3 family were inoculated and rated as either resistant or susceptible. A good fit to the expected ratio of 1 homozygous resistant:3 segregating and homozygous susceptible F_3 families was found. Some conflicting evidence was noted however; 30 of the 97 segregating F_3 families segregated in a 3:1 ratio, indicative of dominant resistance. In the same study, Carifen12 was also found to carry a single dominant gene for resistance to a mixture of two isolates of the septoria leaf blotch fungus (Septoria tritici). Chi-square tests indicated a probable association between the septoria leaf blotch and tan spot resistance genes.

4.3 Inheritance of tan spot resistance in tetraploid wheat

Only quantitative inheritance for tan spot resistance has been reported in tetraploid wheat. The inheritance of resistance to the PyW17 isolate of the tan spot fungus was studied in crosses between a susceptible line, Rolette, and five resistant lines, Wells, PI194526, PI166308, D6761, and D6876 (Nagle et al., 1982). The quantitative criterion, disease severity (based on %leaf area affected), was used to assess resistance/susceptiblity. For all crosses, F_2 and BC_1F_1 results did not correspond with expectations for monogenic or digenic inheritance. Tan spot resistance was therefore

considered a quantitative trait.

Advanced generations (F_4 and F_5) of two tetraploid crosses were studied in a quantitative genetic approach by Cantrell et al. (1985). The resistant line PI184526 was crossed to two susceptible wheat cultivars, Edmore and Calvin. Plants were inoculated in the greenhouse and in the field. Parent-offspring regression (F_4-F_5) estimates of heritability ranged from 0.35-0.44 under greenhouse conditions. Under field conditions, higher values of 0.63-0.79 were obtained using variance component estimates on an F_4 progeny mean basis. The low to moderate heritability estimates obtained suggested that breeding for tan spot resistance in tetraploid wheat would be difficult, but possible.

In a similar study, F_5 progeny of the cross, Calvin (susceptible) x PI184526 (resistant) were evaluated for resistance to the PyD7 isolate of the tan spot fungus and rated based on the quantitative disease assessment criterion, lesion size (Elias et al., 1989). A high narrow sense heritability estimate was obtained (H = 0.73), and additive genetic variance was significant. The results suggested that breeding for tan spot resistance would be possible in this material.

4.4 Inheritance of tan spot resistance in diploid wheat

There are no reports of the inheritance of tan spot resistance in diploid wheat. However, screening of 8 diploid and 10 polyploid species of the related wild wheat species Aegilops spp. against an isolate of the tan spot fungus from the U.S.A. has recently been carried out using a rating scale based on a combination of lesion type and %leaf area affected (Alam and Gustafson, 1988). A number of resistant accessions were identified.

Screening of diploid wheat accessions from the University of Manitoba collection using the qualitative disease assessment criterion, lesion type, has revealed genotypic variability for resistance to the ASC1 isolate of the tan spot fungus (Lamari and Bernier, 1989). Diploid lines with high levels of resistance were identified.

The inheritance of resistance to the ASC1 isolate of the tan spot fungus (Pyrenophora tritici-repentis) in hexaploid, tetraploid, and diploid wheat.

ABSTRACT

The inheritance of resistance to the ASC1 isolate of the tan spot fungus was investigated in hexaploid, tetraploid, and diploid wheat in growth room studies. Plants were inoculated at the 4-6 leaf growth stage (Feekes 4-5) with the ASC1 isolate. Resistance/susceptibility was assessed on the basis of lesion type, a qualitative disease assessment criterion described by Lamari and Bernier (1989). Qualitative inheritance of tan spot resistance was detected within all ploidy levels.

Qualitative inheritance of tan spot resistance was indicated in several of the 18 crosses (and reciprocals) of the hexaploid inheritance study. In the F_1 generation, tan spot resistance appeared dominant (in crosses with TpxTm), incompletely dominant (in crosses with the resistant parents, Salamouni and Erik, and moderately resistant parent, BH1146), and recessive (in crosses with the resistant parent, Carifen12). F_2 data was variable, but suggested that the resistant parents, Salamouni, Carifen12, and Erik, likely had in common one recessive gene pair for resistance. F_2 results also indicated that the moderately resistant parent, BH1146 differed from the three resistant parents for resistance gene(s). BCF_1 and F_3 results provided further support for these conclusions.

F_2 results of the remaining crosses of the hexaploid inheritance study, involving the moderately resistant parent, BH1146, and moderately susceptible parents, HY320 and Glenlea, were inconclusive. Suspected genetic heterogeneity in the parents likely contributed to the inconclusive results. Difficulty in classification due to the use of moderate levels of resistance

and susceptibility, growth stage or leaf age effects, and incomplete dominant gene action may also have contributed. The F_2 results suggested that BH1146 possessed at least one gene for resistance which differed from the resistance gene of the resistant parents, Salamouni, Carifen12, and Erik. In the cross of the moderately susceptible parent, HY320 with the susceptible parent, Columbus, a few resistant progeny were unexpectedly obtained. The "susceptible" parents likely differed for minor genes for resistance, which, when combined in the progeny, gave an increased level of resistance. Of the 18 crosses studied in the F_2 generation, reciprocal effects were absent with the possible exception of the above cross.

Qualitative inheritance of tan spot resistance was detected in the tetraploid inheritance study comprised of 10 crosses and reciprocals. F_2 data for both the 2-leaf and 4-6 leaf growth stages revealed that the moderately resistant parent, 4B175 possessed one recessive gene pair for resistance. F_2 results for the other moderately resistant parents, 4B233 and 4B242 were variable, but suggested that they shared the resistance gene(s) of 4B175. Suspected genetic heterogeneity of the parents, the use of moderately resistant parents, small population sizes, and possible incomplete dominant gene action (suggested by the F_1 data) made interpretation of the tetraploid inheritance study difficult. As in the moderately susceptible with susceptible hexaploid cross, evidence of the accumulation of minor genes for resistance was found in the moderately susceptible with susceptible tetraploid cross, Arcola with Coulter. No reciprocal differences were noted in the F_1 or F_2 generation of the 10 tetraploid crosses.

Qualitative inheritance of tan spot resistance in diploid wheat was observed in two resistant x susceptible and two resistant x resistant

crosses. F_2 data revealed that the resistant parents, 2B27 and 2B53, each carried one recessive gene pair for resistance, which may or may not be common to both. F_2 data also indicated that the resistant parents, 2B27, 2B26, and 2B7, possessed the same resistance gene or genes.

INTRODUCTION

Tan spot or yellow spot is a foliar disease of wheat caused by the fungus Pyrenophora tritici-repentis (Died.) Drechs. (anamorph, Drechslera tritici-repentis (Died.) Shoem.). The disease has caused significant yield losses ranging from 3 to 40% in the Central Plains of the U.S.A. and Canada since the 1970's (Tekauz, 1976; Luz and Hosford, 1980; Tekauz et al., 1982). Due to the increasing use of minimum tillage practices in spring wheat and zero tillage in winter wheat production which favours overwintering of the fungus in the crop residue, losses due to tan spot may be expected to rise. A reduction in test weight (Sharp et al., 1976), and a pink discolouration of the kernels resulting in reduced grade are also consequences of tan spot infection (Vanterpool, 1963; Lyster, 1987).

P. tritici-repentis has a broad host range and is known to infect more than 33 species of grasses (Hosford, 1982), although barley, oats, and rye are highly resistant (Wiese, 1977; Larez et al., 1986). Tan spot resistance in wheat has generally been described as reduced amounts of diseased tissue. Various quantitative disease assessment criteria, such as lesion frequency (Nagle et al., 1982; Raymond et al., 1985), lesion size (Raymond et al., 1985; Cox and Hosford, 1987), %leaf area affected (Nagle et al., 1982), and disease severity (which may take into account leaf age and growth stage) (Misra and Singh, 1972; Luz and Hosford, 1980; Gough and Johnston, 1982; Raymond et al., 1985; Cox and Hosford, 1987) have been used to measure tan spot resistance/susceptibility. More recently, a qualitative criterion, lesion morphology or type, was used to detect differences in tan spot reaction among 695 accessions of diploid, tetraploid, hexaploid, and octoploid wheat (Lamari and Bernier, 1988). Within each ploidy level, many

lines were identified which expressed an environmentally stable, high level of resistance, characterized by small dark brown or black spots without chlorosis or tan necrosis components (lesion type 1). Lesion type had been used to differentiate between resistant and susceptible barley lines in the closely related Pyrenophora teres (Drechs.)-barley system (Tekauz, 1985).

Variability for virulence has been reported among isolates of the tan spot fungus although various quantitative criteria have been used to assess the differential reactions on the host plants (Misra and Singh, 1972; Luz and Hosford, 1980; Hosford, 1982). Using the qualitative criterion, lesion type, isolates from western Canada have been clearly divided into three virulence groups (pathotypes) based on their ability to induce tan necrosis and/or extensive chlorosis (a previously unreported susceptible symptom type) on differential wheat hosts (Lamari, 1988). Additionally, isolates capable of inducing necrosis were found to produce toxin in vitro.

Toxin production by the tan spot fungus has been recognized only very recently (Tomas et al., 1986; Tomas and Bockus, 1987; Lamari, 1988). A relationship between susceptibility to the fungus and sensitivity to toxin has been demonstrated (Tomas and Bockus, 1987; Lamari, 1988). Lamari (1988) reported that one dominant gene controlled both susceptibility to the fungus and toxin sensitivity. The toxin discovered by Lamari is a single protein of 9,300 molecular weight (Lamari et al., in press). Although this toxin is currently considered a pathogenicity factor and may play an important role in determining resistance/susceptibility at the cellular level, tan spot resistance has been attributed to unknown factor(s) which restrict intercellular mycelial growth of the fungus (Larez, Hosford, and Freeman, 1986; Loughman and Deverall, 1986; Lamari, 1988).

Few studies of the inheritance of resistance to tan spot have been published. At the hexaploid level, quantitative inheritance for resistance to one isolate of the tan spot fungus, evaluated on the basis of the quantitative criteria, %leaf area affected and lesion size, has been reported (Nagle et al., 1982). Qualitative inheritance within hexaploid wheat has also been indicated -- Carifen12 was found to carry one recessive gene pair for resistance to one isolate of the tan spot fungus (Lee and Gough, 1984). At the tetraploid level, only quantitative inheritance for tan spot resistance, assessed by the quantitative criterion, %leaf area affected, has been reported (Nagle et al., 1982; Cantrell et al., 1985). Low to high heritability estimates have been reported in tetraploid wheat crosses (Cantrell et al., 1985; Elias et al., 1989). There have been no reports of the inheritance of tan spot resistance in diploid wheat, although some screening has been carried out (Alam and Gustafson, 1988; Lamari and Bernier, 1989).

The objective of this study was to investigate the inheritance of resistance to the ASC1 isolate of the tan spot fungus within hexaploid, tetraploid, and diploid wheat using the qualitative disease assessment criterion, lesion type (described by Lamari and Bernier, 1989). A qualitative genetic approach (involving Chi-square tests of expected F_2 genetic ratios) was used.

MATERIALS AND METHODS

1. Plant material

Eighteen wheat lines (eight hexaploids, five tetraploids, and five diploids) from among a large number of lines studied by Lamari and Bernier (1989) were selected as parents to study the inheritance of resistance to the ASC1 isolate of the tan spot fungus. The species, origin, and tan spot rating of the parents are shown in Table 1. The tan spot ratings reported are in agreement with those of Lamari and Bernier (1989) with the exception of Carifen12; a resistant component of Carifen12 was inadvertently selected and used in this study. All except Carifen12 were spring types, and ploidy levels were confirmed by root tip chromosome counts.

Eight parents, four resistant (TpxTm, Salamouni, Carifen12, and Erik), one moderately resistant (BH1146), two moderately susceptible (HY320 and Glenlea), and one susceptible (Columbus) were used in the hexaploid inheritance study (Table 1). All possible resistant x susceptible, resistant x resistant, and susceptible x susceptible crosses among the parents were made in reciprocal. Crosses were made using bulked pollen obtained from a number of "male" parent plants which had been previously inoculated and shown to exhibit the tan spot reaction characteristic of that parent. F_1's of the 28 crosses and reciprocals were tested for tan spot reaction. Eighteen crosses and their reciprocals (ten resistant x susceptible, six resistant x resistant, and two susceptible x susceptible) were selected for further study in the F_2 generation. The F_1 plants were bagged to ensure purity of the F_2 seed. Four F_1-derived F_2 populations per cross were evaluated. In addition, BCF_1 populations of three resistant with susceptible or moderately susceptible crosses (Salamouni with Columbus, Glenlea, and HY320,

respectively) and two resistant with resistant or moderately resistant crosses (Salamouni with Carifen12 and BH1146, respectively) were tested. Reciprocal BCF_1's were derived from approximately 10 F_1 plants (as female parents) per cross and included some or all of the F_1 plants used to provide F_2 population data. Bulked pollen from previously inoculated parent plants was used in crossing. F_3 families of the crosses, Salamouni with Glenlea, and Salamouni with BH1146, were also grown and tested. For these crosses, F_3 families were derived from four and six F_1 plants, respectively, and 100 F_3 families of approximately 12 plants per family were evaluated. Due to poor experimental conditions, the results obtained for the F_2's derived from the same F_1 plants as the F_3's were discounted and another set of F_1's were used to obtain F_2 data for these crosses.

Parents, F_1, and F_2 populations were tested to investigate the inheritance of tan spot resistance in tetraploid wheat. Five parents, three moderately resistant (4B175, 4B233, and 4B242), one moderately susceptible (Arcola), and one susceptible (Coulter) were used (Table 1). As with the hexaploids, all possible resistant x susceptible, resistant x resistant, and susceptible x susceptible crosses were made in reciprocal. As with the hexaploids, crosses were made using bulked pollen obtained from a number of previously inoculated "male" parent plants which exhibited a tan spot reaction characteristic of that parent. F_1's of the 10 crosses and reciprocals were grown and tested for tan spot reaction. For each cross, four F_1 plants, different from those tested for F_1 tan spot reaction, were bagged to produce four F_1-derived F_2 populations which were evaluated.

Parents, F_1, and F_2 populations were used to investigate the inheritance of tan spot resistance in diploid wheat. As with the hexaploids and

tetraploids, bulked pollen obtained from a number of "male" parent plants which were previously inoculated and displayed a tan spot reaction characteristic of that parent, was used in crossing. Also, F_1 plants were bagged to ensure purity of the F_2 seed. Two resistant x susceptible (2B27 x 2B37 and 2B53 x 2B37) and two resistant x resistant crosses (2B27 x 2B26 and 2B26 x 2B7) were evaluated.

All seed was pre-germinated prior to planting in soiless mix (Metro-Mix 200, W.R. Grace and Co., Ajax, Ont.). Hexaploid and diploid F_1 plants were grown in clay pots (15 cm. diameter); tetraploid F_1's and all other plants were grown individually in 500 ml. milk cartons. Plants were kept in a growth room at 20°C day/15°C night temperatures with a 16 h. photoperiod, and fertilized approximately once a week with 20:20:20 (N:P:K) fertilizer.

2. Inoculation

Plants were inoculated at the 4-6 leaf growth stage or at Feekes growth stages 4 to 5 (Large, 1954) with the ASC1 isolate of the tan spot fungus (obtained from Dr. Lamari, University of Manitoba, Winnipeg, Man.). All F_2's of three tetraploid crosses were inoculated at the 2-leaf growth stage (Feekes growth stages 1 to 2) as well as at the 4-6 leaf stage.

For inoculum production sufficient to inoculate four F_1-derived F_2 populations (one week of inoculation), a single conidiospore of ASC1 was obtained from sporulating leaf tissue using a sterile needle and grown on 9 cm. petri plates of potato dextrose agar (PDA) medium at 15-20°C for 7 days, as described previously by Lamari (1988). Agar plugs (0.5 $cm.^3$) of the fungus were removed and placed on 9 cm. petri plates of V-8/PDA medium (850 mls. distilled water, 150 mls. V-8 juice, 10 g. PDA, 10 g. agar, 3 g. calcium

carbonate per liter). After 5 days at 15-20°C in the dark, the fungus thallus was moistened with sterile distilled water, flattened with a sterile glass test tube, and then placed under fluorescent lights (suspended approximately 50 cm. above the plates) at room temperature for 24 hours of continuous illumination. The plates were then transferred to a dark incubator at 15°C for a further 24 hours. The conidia were harvested by flooding the fungal surface with sterile distilled water and sweeping gently with a sterile inoculation loop. Half of the aqueous suspension obtained was placed on ice in a coldroom at 4°C for use the next day. The remainder was adjusted to 3000 conidia/ml., Tween 20 (polyoxyethylene sorbitan monolaurate, Fisher Scientific) was added at 1 drop/100 mls. of inoculum, and was applied to the leaf surfaces until runoff using a small paint sprayer at 10-15 p.s.i. pressure. Inoculated plants were placed in a clear polyethylene-covered humidity chamber contained in the growth room for 24 hours. The humidity chamber was equipped with an ultrasonic humidifier to maintain approximately 100% relative humidity. Due to limited space, only one F_1-derived F_2 population or a portion of BCF_1 or F_3 populations could be inoculated on one day. Testing proceeded for a period of two years. Plants were rated for tan spot reaction 7 days after inoculation using the qualitative criterion, lesion type. Lesion type, as described by Lamari and Bernier (1989) (Table 2) was recorded for the two uppermost inoculated leaves of the main culm. A modified 1-5 rating scale which took both leaves into consideration (Table 3) was used for the analyses. The modified 1-5 rating scale was used because it provided a better assessment of tan spot reaction at the 4-6 leaf growth stage for the parents used in this study, especially the moderately resistant and moderately susceptible parents.

3. Statistical analyses

Chi-square goodness of fit tests to expected genetic ratios were performed on the hexaploid, tetraploid, and diploid F_2 data. The modified 1-5 scale (Table 3) was used to separate plants into resistant or susceptible classes in dependence on the tan spot ratings of the parents and the bimodal groupings in the F_2 generation of the cross. Two classifications of resistance, $R=1+1^+$ and $R=1+1^++2$, were used in this study; all plants with ratings greater than the resistance classification used were considered susceptible. F_2 results were pooled for crosses in which Chi-square tests for homogeneity indicated pooling was appropriate (Cochran and Cox, 1957). A description of the digenic genotypic ratios used in the Chi-square tests is presented in Table 4.

RESULTS

1. Hexaploids

1.1 F_1's

Tan spot ratings of the F_1's (Table 4) may be compared with those of the parents (Table 1). With the exception of crosses involving the most consistently resistant parent, TpxTm, considerable variability in tan spot rating was observed in the F_1's. Of the 28 crosses studied, reciprocal differences were not evident in the F_1 generation.

An indication of type of gene action can be obtained from a consideration of the F_1's of the resistant with susceptible crosses (TpxTm, Salamouni, Carifen12, and Erik, respectively with Columbus) and moderately resistant with susceptible cross (BH1146 with Columbus). Dominant resistance was indicated in the cross with TpxTm since the F_1's were predominantly rating 1, similar to TpxTm. Incomplete dominance was implicated by the range of tan spot ratings in the F_1's of the crosses involving Salamouni, Erik, and BH1146. For the cross involving Carifen12, F_1 tan spot ratings were similar to the susceptible parent, Columbus, suggesting that resistance was recessive.

For the resistant x moderately susceptible crosses involving the resistant parents, TpxTm, Salamouni, and Erik, tan spot ratings of the F_1's were predominantly rating 1, similar to the resistant parents. In contrast, for those crosses involving the resistant parent, Carifen12, predominant tan spot ratings of the F_1's were characteristic of the moderately susceptible parent. For the F_1's of the moderately resistant x moderately susceptible crosses, tan spot ratings ranged from 1^+ to 3, similar to the moderately resistant parent.

F_1's of the resistant x resistant crosses had tan spot ratings of 1 or 1^+, the same as the resistant parents, with the exception of one F_1 plant with a rating of 3 in the cross, Erik x Carifen12.

Considering the moderately resistant x resistant crosses, all F_1's of the crosses involving the resistant parents, TpxTm and Erik, had tan spot ratings of 1 or 1^+, similar to the resistant parents. For the other two crosses involving the resistant parents, Salamouni and Carifen12, the predominant F_1 ratings were similar to the moderately resistant parent, BH1146.

All F_1's of the moderately susceptible x susceptible crosses (Glenlea with Columbus, and HY320 with Columbus) had tan spot ratings similar to the susceptible parent, Columbus. Tan spot ratings of the intercross between the two moderately susceptible parents were within the range of the parents.

1.2 F_2's

For the 18 crosses, the range of tan spot ratings observed in the F_2's and their parents is shown in Table 6. Over the course of F_2 testing, the three resistant parents, Salamouni, Carifen12, and Erik were fairly consistent, displaying predominantly rating 1 or 1^+ reactions. The susceptible parent, Columbus, consistently exhibited high tan spot ratings of 3 to 5 (rarely 2). However, considerable variability was noticeable in the moderately resistant parent, BH1146; tan spot reaction ranged from rating 1, characteristic of the resistant parents, to rating 3, typical of the moderately susceptible parents. Likewise, the reaction of the moderately susceptible parents, HY320 and Glenlea, ranged from rating 1^+, observed in the resistant parents to rating 4, commonly found in the susceptible parent,

Columbus.

The variability in the parental reactions suggests that they may be genetically heterogeneous, which would contribute to inconsistency in observed F_2 genetic ratios. As well, lack of conclusiveness in F_2 results may arise from difficulties in classification due to incompletely dominant gene action indicated by the F_1 data for the resistant parents, Salamouni and Erik and the moderately resistant parent, BH1146. These two factors, possible genetic heterogeneity in the parents and incompletely dominant gene action should be kept in mind in the following discussion of the F_2 results. Descriptions of the digenic genotypic ratios used in the Chi-square tests are found in Table 4.

1.2.1 Resistant x susceptible crosses

For the three resistant x susceptible crosses studied, tan spot ratings ranged from 1 to 5 in the F_2 populations (Table 6). Bimodality was evident in the F_2 distributions with the division between the two groups at rating 2. On the basis of the bimodal groupings and the parental reactions (the resistant parents, Salamouni, Carifen12, and Erik typically had 1 or 1^+ ratings), F_2 plants with ratings of 1 or 1^+ were considered resistant ($R=1+1^+$ classification of resistance) and all others were considered susceptible in the Chi-square tests (Table 7). Although Chi-square tests of expected F_2 ratios were inconsistent within each of the crosses, the 1:3 (resistant:susceptible) and 1:15 (resistant:susceptible) ratios obtained indicated the presence of one recessive gene pair or two recessive complementary genes for tan spot resistance (Table 7). For the three resistant x susceptible crosses, the most common ratio obtained was a 1:3 ratio, suggesting that as little as one gene may give resistance to tan spot.

Evidence that Erik possessed duplicate recessive genes was indicated in one population of the cross, Erik with Columbus, where a 7:9 (resistant:susceptible) ratio fit the observed data.

1.2.2 Resistant x resistant crosses

No segregation was noted in the F_2 generation of intercrosses between the three resistant parents, Salamouni, Carifen12, and Erik, indicating that they had the same gene or genes for resistance (Table 6). The three resistant parents characteristically had tan spot ratings of 1 or 1^+; all F_2's of the intercrosses had ratings of 1 or 1^+.

1.2.3 Moderately resistant x susceptible cross

As with the resistant x susceptible crosses, tan spot ratings ranging from 1 to 5 were observed in the F_2 populations of the moderately resistant x susceptible cross, BH1146 with Columbus (Table 6). The F_2 distributions also appeared bimodal with the division between the two groups at rating 2. Considering the predominant tan spot ratings of the moderately resistant parent (Tables 1 and 6), and the division of the bimodal distribution at rating 2, F_2 plants with ratings of 1, 1^+, or 2 ($R=1+1^+ +2$) were considered resistant, and all others were classified as susceptible in the Chi-square tests (Table 7). Although the Chi-square ratios were inconsistent, 1:3, 7:9, and 9:7 ratios were obtained. This suggested that the moderately resistant parent, BH1146 possessed either one recessive gene pair or possibly two gene pairs (either duplicate recessive or dominant complementary) for tan spot resistance.

1.2.4 Resistant x moderately susceptible crosses

Tan spot ratings ranging from 1 to 4 were observed in the F_2 populations of the five crosses involving the resistant parents, Salamouni, Carifen12,

and Erik and the moderately susceptible parents, Glenlea and HY320 (Table 6). Similar to the resistant x susceptible crosses, most of the F_2 distributions of the resistant x moderately susceptible crosses were bimodal with the division at rating 2. Since the resistant parents, Salamouni, Carifen12, and Erik had tan spot ratings of 1 or 1^+, predominantly, and since the bimodality suggested separation at rating 2, the R=1+1^+ classification of resistance was used in the Chi-square tests (Table 7). Although Chi-square ratios within the crosses were inconsistent, ratios indicative of one or two gene pairs with various types of gene action were obtained. For all five crosses, a 9:7 or 7:9 ratio predominated; perhaps the moderately susceptible parents, Glenlea and HY320 possess a minor resistance gene different from the major resistance gene of the resistant parents and which interacts in either a dominant complementary or duplicate recessive manner.

1.2.5 Moderately resistant x moderately susceptible cross

Tan spot ratings ranged from 1 to 4 in the F_2 of the cross of the moderately resistant parent, BH1146 with the moderately susceptible parent, Glenlea. The F_2 distributions also appeared bimodal with the division between the two groups at rating 2. On the basis of the bimodal groupings and predominant tan spot ratings of the parents, the R=1+1^++2 classification of resistance was used in the Chi-square tests (Table 7). Given the considerable variability for tan spot rating within the parents, the inconsistency in the F_2 ratios observed was not unexpected. F_2 ratios of 3:1, 7:9, and 9:7 were obtained, indicating that the two parents differed by one dominant gene or two genes (either duplicate recessive or dominant complementary) for tan spot resistance.

1.2.6 Moderately resistant x resistant crosses

F_2 distributions from crosses of the moderately resistant parent, BH1146 with the three resistant parents, Salamouni, Carifen12, and Erik are presented in Table 6. Given the wide range of tan spot ratings in the moderately resistant parent, BH1146 (rating 1 to 3), it was difficult to determine whether segregation had or had not occurred in the F_2 generation of these crosses. There was no apparent segregation in the F_2 of the cross, BH1146 with Carifen12, since the F_2's had tan spot ratings ranging from 1 to 3, within the range of the parents. This suggested that BH1146 and Carifen12 did not differ for resistance gene(s). However, F_2 plants with rating 4 were detected in one population of the cross, BH1146 with Salamouni, and in all populations of the cross, BH1146 with Erik, indicating that segregation had occurred, and that BH1146 differed from Salamouni and Erik for resistance gene(s). Since the three resistant parents have been shown to possess the same resistance gene(s), it is likely that genetic heterogeneity in BH1146 was responsible for the different results of these three crosses. Perhaps only some of the BH1146 plants used in these crosses possessed all the resistance genes of the resistant parents.

1.2.7 Moderately susceptible x susceptible crosses

For the moderately susceptible x susceptible cross, Glenlea with Columbus, all F_2's had tan spot ratings within the range of the parents (Table 6).

In contrast, for the other cross, HY320 with Columbus, reciprocal differences and segregation beyond the parents were noted in the F_2 generation (Table 6). Although tan spot ratings of the moderately susceptible parent, HY320, ranged from 1^+ to 4 and the susceptible parent,

Columbus had ratings of 2 to 5, a small number of F_2 plants with a rating of 1 (typical of the resistant parents in this study) were obtained from two populations in which HY320 was the female parent. The recovery of resistant F_2 plants from a cross between "susceptible" parents likely represents an accumulation of minor genes for resistance for which the parents differed. A greater number of populations need to be tested to confirm the apparent reciprocal effect observed in this cross. Since reciprocal differences were not observed in the F_2 generation of the other 17 hexaploid crosses studied, nor in the F_1 generation of the 28 hexaploid crosses, it is unlikely that this observed reciprocal difference is a true difference.

1.3 BCF_1's

The distribution of tan spot ratings in the parents and BCF_1 plants from five crosses (Salamouni (resistant) with Columbus (susceptible), Glenlea (moderately susceptible), HY320 (moderately susceptible), BH1146 (moderately resistant), and Carifen12 (resistant), respectively) are presented in Table 8.

For the cross of the resistant parent, Salamouni with the susceptible parent, Columbus, the BCF_1's to the resistant parent had tan spot ratings of 1 or 1^+, similar to the resistant parent, with the exception of two plants with rating 3 (Table 8). BCF_1's to the susceptible parent had predominantly susceptible tan spot ratings although several plants with ratings of 1 or 1^+ were detected. The backcross results did not conform to that expected for one recessive gene pair or two recessive complementary gene pairs for resistance, as suggested by the 1:3 and 1:15 F_2 ratios obtained for this cross. However, the F_1 data suggested that the resistance gene(s) were not

fully recessive (incomplete dominant gene action). If this was the case, BCF_1 plants heterozyous for as few as one resistance gene could appear "resistant" due to incomplete dominant gene action; this could account for the resistant BCF_1's in the backcross to the susceptible parent and the mainly resistant BCF_1's in the backcross to the resistant parent in the observed results. Modifier genes may also have affected the BCF_1 results.

Backcross results of the crosses of the resistant parent, Salamouni with the moderately susceptible parents, Glenlea and HY320, resembled results of the cross, Salamouni with Columbus; a number of resistant plants were found in the susceptible parent backcross and the majority of the BCF_1 plants from the resistant parent (Salamouni) backcross were resistant (Table 8). These results reinforced the Salamouni with Columbus results and further indicated that the resistance of Salamouni may be due to as few as one incompletely dominant gene pair.

For the cross of the resistant parent, Salamouni with the moderately resistant parent, BH1146, many plants with ratings of 1 or 1^+, similar to the resistant parent, were detected in both backcrosses (Table 8). However, segregation (plants with rating 4) was also noticed in both sets of BCF_1's. This indicated that the resistant parent, Salamouni, and moderately resistant parent, BH1146 did not have the same gene(s) for tan spot resistance, in agreement with some of the F_2 results of this cross (Table 6).

For the cross between the two resistant parents, Salamouni and Carifen12, all BCF_1 plants had tan spot ratings of 1 or 1^+, the same as the parents (Table 8). This indicated that Salamouni and Carifen12 possessed the same gene(s) for resistance. These backcross results are in agreement with and further support the F_2 results of this cross.

1.4 F_3's

Over the course of F_3 testing for the cross, Salamouni with BH1146, parental ratings ranged from 1 to 1^+ for Salamouni (resistant) and from 1 to 3 for BH1146 (moderately resistant). Considering the reaction of the resistant parent, the R=1+1^+ classification of resistance was used to categorize the F_3 families as 65 homozygous resistant: 35 segregating :0 homozygous susceptible. Six of the 35 segregating F_3 families contained plants with ratings of 4, segregants for susceptibility. This suggested that Salamouni and BH1146 did not have the same gene(s) for resistance. The F_2 results and BCF_1 results also suggested that Salamouni and BH1146 differed for resistance gene(s).

For the cross of the resistant parent, Salamouni, with the moderately susceptible parent, Glenlea, the R=1+1^+ classification of resistance was used to categorize the F_3 families as 25 homozygous resistant:75 segregating:0 homozygous susceptible. If the segregating and homozygous susceptible classes were grouped, the observed data fit a 1 homozygous resistant:3 segregating or homozygous susceptible ratio with no significant heterogeneity (Table 9). This indicated that Salamouni possessed one gene for resistance. The ratios within the segregating F_3 families indicated that the resistance gene may be dominant or incompletely dominant. Dominant resistance was also indicated in two of the four F_2 populations of this cross (Table 7).

2. Tetraploids

2.1 F_1's

Tan spot ratings of F_1's of the 10 tetraploid crosses are shown in Table

10, and can be compared with characteristic parental reactions shown in Table 1. Considerable variability in tan spot ratings was noticed. Reciprocal differences were not observed except possibly in the cross, 4B233 with Arcola; when 4B233 was used as the female parent, F_1 tan spot ratings ranged from 2 to 4 with predominantly rating 4, but when Arcola was used as the female parent, the F_1's ranged from 1^+ to 4 and predominantly rating 1^+ plants were obtained. Although the sample size of five F_1 plants was small, evidence to suggest that the inheritance of tan spot resistance was incompletely dominant was found in the range of tan spot ratings in the moderately resistant x susceptible crosses.

2.2 Inoculation at the 2-leaf stage vs. 4-6 leaf stage

The F_2's of two moderately resistant x susceptible crosses (4B175 with Coulter, and 4B242 with Coulter) and the intercross of the two moderately resistant parents (4B175 with 4B242) were inoculated at both the 2-leaf and 4-6 leaf growth stages. For all three crosses, a narrower range of tan spot ratings and a greater proportion of rating 1 plants were identified at the 2-leaf stage (Table 11) compared to the 4-6 leaf stage (Table 12). For the crosses, Coulter with 4B175, and Coulter with 4B242, no rating 1 plants were found at the 4-6 leaf stage. For the cross, 4B242 with 4B175, fewer rating 1 plants were detected at the 4-6 leaf stage, but all F_2 ratings were within the range of the parents for both growth stages.

2.3 F_2's

The range of tan spot ratings observed for F_2's and their parents at the 4-6 leaf growth stage are presented in Table 12. With the exception of the

susceptible parent, Coulter, considerable variability in tan spot rating was recorded for the parents. Over the course of F_2 testing at the 4-6 leaf stage, ratings for the moderately resistant parents, 4B175, 4B233, and 4B242, ranged from rating 1 or 1^+, predominantly to 3. For the moderately susceptible parent, Arcola, ratings ranged from rating 1^+ to 4, with a rating of 3 most common.

2.3.1 Moderately resistant x susceptible crosses

F_2 distributions of the two moderately resistant x susceptible crosses (4B175 with Coulter and 4B242 with Coulter) tested at the 2-leaf stage are shown in Table 11. For both crosses, the F_2 distributions ranged from rating 1 to 3, with a low frequency of plants of rating 1 to 2 and a high frequency of plants of rating 3. The distibutions were thus considered bimodal with division between the two groups at rating 2. On the basis of the parental reactions and the bimodal groupings, plants with ratings of 1 to 2 were considered resistant ($R=1+1^+$+2 classification of resistance) and plants with ratings greater than 2 were classified as susceptible in the Chi-square tests (Table 13). For the cross, 4B175 with Coulter, all F_2 populations tested and the pooled population fit a 1:3 (resistant:susceptible) ratio with no evidence of heterogeneity (Table 15). For the cross, 4B242 with Coulter, a 1:3 ratio also fit two F_2 populations, but a 7:9 ratio was obtained in the other two F_2 populations tested. These results indicated that the moderately resistant parent, 4B175 possessed one recessive gene pair for resistance exhibited at the 2-leaf stage, and that the moderately resistant parent, 4B242, also possessed one recessive gene pair or possibly two duplicate recessive genes. Genetic heterogeneity within the moderately resistant parent, 4B242, may have been responsible for the inconsistency in the F_2

ratios observed.

F_2 distributions of the three moderately resistant x susceptible crosses (4B175, 4B233, and 4B242, respectively with Coulter) tested at the 4-6 leaf stage are presented in Table 12. For the crosses, 4B175 with Coulter, and 4B233 with Coulter, a bimodal distribution with division at rating 2 was apparent. Since over the course of F_2 testing, tan spot ratings of the three moderately resistant parents ranged from 1 to 2, predominantly, to 3 and since the bimodality observed suggested division between rating 2 and 3, the $R=1+1^{+}+2$ classification of resistance was used in the Chi-square tests (Tables 14 and 15).

For the cross, 4B175 with Coulter, tested at the 4-6 leaf stage, all F_2 populations and the pooled population fit a 1:3 (resistant:susceptible) ratio with no evidence of heterogeneity (Table 15). These results are in agreement with the results from the 2-leaf stage testing, and indicated that the moderately resistant parent, 4B175 possessed one recessive gene pair for resistance.

For the cross, 4B233 with Coulter, a 9:7 ratio fit three of the four F_2 populations and a 1:15 ratio fit the remaining population of plants tested at the 4-6 leaf stage (Table 14). The results suggested that the moderately resistant parent, 4B233 was genetically heterogeneous and that this parent possessed two complementary resistance genes (either dominant or recessive). This cross was not tested at the 2-leaf stage.

For the cross, 4B242 with Coulter, tested at the 4-6 leaf stage, a 7:9 ratio fit three of the four F_2 populations and a 9:7 ratio fit the fourth F_2 population (Table 14). When tested at the 2-leaf stage, a 7:9 ratio was obtained for two of the four F_2 populations. The results indicated that the

moderately resistant parent, 4B242 may be genetically heterogeneous and that duplicate recessive genes likely governed resistance.

2.3.2 Moderately resistant x moderately susceptible crosses

For the three crosses involving the moderately resistant parents, 4B175, 4B233, and 4B242 and the moderately susceptible parent, Arcola, tan spot ratings ranged from 1 to 4 or 5 in the F_2 generation (Table 12). Due to the great overlapping of the ranges of tan spot ratings of the moderately resistant parents (rating 1 to 3) and the moderately susceptible parent (rating 1^+ to 4), Chi-square tests of expected F_2 genetic ratios were not calculated. Segregation was detected in two F_2 populations of the cross, 4B233 with Arcola, and in one F_2 population of the cross, 4B242 with Arcola, in which rating 5 plants were obtained. This suggests that the moderately resistant parents, 4B233 and 4B242, and the moderately susceptible parent, Arcola, differed for resistance gene(s).

2.3.3 Moderately resistant x moderately resistant crosses

When evaluated at the 2-leaf stage, all F_2's of the cross, 4B242 with 4B175 had tan spot ratings of 1 or 1^+, within the range of the parents. This lack of segregation in the F_2 suggested that the moderately resistant parents, 4B175 and 4B242 had the same resistance gene(s) in common. Considering the 4-6 leaf stage, all F_2's from intercrosses among the moderately resistant parents, 4B175, 4B233, and 4B242 also had tan spot ratings within the range of the parents (rating 1 to 3) (Table 12). The lack of segregation in the F_2 suggested that 4B175, 4B233, and 4B242 shared the same genetic mechanism for resistance. However, the variability for tan spot rating within the parents indicated that such a conclusion should only be tentative. Lack of segregation was most strongly indicated in the cross,

4B175 with 4B242, in which only rating 1 and 1^+ F_2 plants were observed.

2.3.4 Moderately susceptible x susceptible cross

Resistant segregants were unexpectedly detected in F_2's of the cross between the moderately susceptible parent, Arcola and the susceptible parent, Coulter (Table 12). Although the two parents, Arcola and Coulter, had tan spot ratings of 1^+ to 4 and 3 to 4, repectively, a number of plants with a rating of 1, characteristic of a resistant plant, were obtained. Perhaps Arcola and Coulter have different minor genes for resistance which when combined resulted in an increased level of resistance. The considerable variability within the moderately susceptible parent, Arcola suggested that this parent may be genetically heterogeneous.

3. Diploids

3.1 F_1's

In contrast to the hexaploid and tetraploid inheritance studies, there was very little variability in tan spot reaction within the diploid parents and among the F_1's of a cross (Table 16). For the two resistant x susceptible crosses studied, 2B27 x 2B37 and 2B53 x 2B37, the F_1 plants were susceptible (rating 4 or 5), suggesting that resistance was recessive. F_1's from the two resistant x resistant crosses were resistant (rating 1).

3.2 F_2's

The range of tan spot ratings observed in the diploid parents and their F_2's is presented in Table 17. In contrast to the hexaploid and tetraploid parents, there was very little variability in tan spot rating within the diploid parents. The four resistant parents, 2B7, 2B26, 2B27, and 2B53 had

tan spot ratings of 1, predominantly, or 1^+. The susceptible parent, 2B37, displayed susceptible ratings of 4 or 5.

3.2.1 Resistant x susceptible crosses

Tan spot ratings ranged from 1 to 5 in the F_2 populations of the two resistant x susceptible crosses, 2B27 x 2B37 and 2B53 x 2B37 (Table 17). Bimodality was also apparent with division at rating 2. On the basis of the parental reactions and the bimodal groupings, F_2 plants with ratings of 1 or 1^+ were considered resistant and all others were considered susceptible ($R=1+1^+$ classification of resistance) in the Chi-square tests (Table 18).

For the cross, 2B53 x 2B37, a 1:3 (resistant:susceptible) ratio was obtained in the F_2 generation. This indicated that the resistant parent, 2B53 possessed one recessive gene pair for resistance. Unfortunately, only one F_2 population of this cross could be tested due to poor viability of the F_1 seed. Testing of additional populations of this cross are needed to confirm this result.

For the cross, 2B27 x 2B37, all F_2 populations and the pooled population fit a 1:3 ratio with no evidence of heterogeneity (Table 18). This indicated that the resistant parent, 2B27 possessed one recessive gene pair for resistance, which may or may not be the same as that identified in 2B53.

3.2.2 Resistant x resistant crosses

No segregation was detected in the F_2 of the two resistant x resistant crosses, 2B27 x 2B26, and 2B26 x 2B7; all F_2's had tan spot ratings of 1 or 1^+, as did the parents (Table 17). This lack of segregation in the F_2 indicated that the resistant parents, 2B27, 2B26, and 2B7 shared the same gene(s) for resistance.

Table 1. Species, origin, and tan spot rating of the 18 wheat parents.

Line	Species	Origin	Rating[1]
Hexaploids			
TpxTm	T. persicum/ T. monococcum	Georgia, U.S.A. (Morey, 1966)	1,1+
Salamouni	T. aestivum	Lebanon (Tell-Amara, 1957)	1,1+
Carifen12	T. aestivum	Chile (Gough, 1982)	1,1+,2,3
Erik	T. aestivum	U.S.A.	1,1+,2
BH1146	T. aestivum	Brazil (Hosford, 1982)	1,1+,2,3
HY320	T. aestivum	Swift Current, SK (Agr. Canada)	1+,2,3
Glenlea	T. aestivum	Winnipeg, MB (U. of Manitoba)	3,4
Columbus	T. aestivum	Winnipeg, MB (Agr. Canada)	3,4,5
Tetraploids			
4B175	T. durum var. melanopus	U.S.S.R. (Zhukovsky, 1957)	1,1+,2,3
4B233	T. durum var. Moghraby el Karak	Lebanon (Tell-Amara, 1957)	1,1+,2,3
4B242	T. orientale var. Gigante ingles	U.S.A. (USDA, 1955)	1,1+,2,3
Arcola	T. durum	Saskatoon, SK (U. of Sask.)	1,1+,2,3
Coulter	T. durum	Winnipeg, MB (Agr. Canada)	3,4
Diploids			
2B7	T. aegilopoides var. larionowii	England (Percival, 1955)	1
2B26	T. monococcum var. flavescens	England (Percival, 1955)	1
2B27	T. monococcum var. flavescens	England (Percival, 1955)	1,1+
2B53	T. monococcum var. flavescens	Japan (Matsumura, 1957)	1
2B37	T. monococcum var. macedonicum	Germany (Max-Planck, 1956)	4,5

1 Tan spot rating on the modified 1-5 scale for two leaves (Table 3), modified from Lamari and Bernier (1989); rating reported here is a range obtained from 4 replicates of 1 to 8 plants per line at the 4-6 leaf stage with the predominant (most frequently observed) rating underlined.

Table 2. Descriptive 1-5 tan spot rating scale (Lamari and Bernier, 1989).

Lesion Type	Description
1	Small dark brown to black spots without any surrounding chlorosis or tan necrosis (resistant)
2	Small dark brown to black spots with very little chlorosis or tan necrosis (moderately resistant)
3	Small dark brown to black spots completely surrounded by a distinct chlorotic or tan necrotic ring; lesions generally not coalescing (moderately resistant to moderately susceptible)
4	Small dark brown or black spots completely surrounded with chlorotic or tan necrotic zones; some of the lesions coalescing (moderately susceptible)
5	The dark brown or black centres may or may not be distinguishable; most lesions consist of coalescing chlorotic or tan necrotic zones (susceptible)

Table 3. The modified 1-5 tan spot rating scale for two leaves (modified from Lamari and Bernier, 1989).

Rating	Lesion Type on Upper Leaf/Lower Leaf[1]
1	1/1
1^+	1/2
2	2/2
3	1/3 or 2/3 or 3/3
4	1/4 or 2/4 or 3/4 or 4/4
5	2/5 or 3/5 or 4/5

[1] Upper leaf/lower leaf applies to the two uppermost inoculated leaves of plants at the 4-6 leaf stage; when rated, only the top-half of the upper leaf had been inoculated whereas the entire lower leaf was inoculated.

Table 4. Description of the digenic genotypic ratios used in the Chi-square tests.

Phenotypic Ratio (R:S)[1]	Genotypic Ratio	Descriptive Name
9:3:3:1	9 A_B_:3 A_bb:3 aaB_:1 aabb	two independent dominant genes
9:7	9 A_B_:7 [3 A_bb:3 aaB_:1 aabb] where A,B = resistant and a,b = susceptible alleles	two dominant complementary genes
7:9	7[3 A_bb:3 aaB_:1 aabb]:9 A_B_ where a,b = resistant and A,B = susceptible alleles	duplicate recessive genes
1:15	1 aabb: 15 [9 A_B_:3 A_bb:3 aaB_] where a,b = resistant and A,B = susceptible alleles	two recessive complementary genes

[1] The three epistatic digenic ratios described here are derived from the 9:3:3:1 ratio characteristic of two independent dominant genes.

Table 5. Tan spot ratings of hexaploid F_1's from 28 crosses involving resistant, moderately resistant, moderately susceptible, and susceptible parents.

	Cross		Reciprocal	
	No.	Rating[1]	No.	Rating[1]
Resistant x Susceptible Crosses				
TpxTm x Columbus	18	1,1+,3,4	13	1
Salamouni x Columbus	14	1,3,4	11	1,2,3,4
Carifen12 x Columbus	10	3,4,5	10	3,4,5
Erik x Columbus	10	1+,2,3,4,5	10	2,3,4,5
Moderately Resistant x Susceptible Cross				
BH1146 x Columbus	10	1,1+,3	10	1+,2,3
Resistant x Moderately Susceptible Crosses				
TpxTm x Glenlea	19	1,3	8	1
Salamouni x Glenlea	14	1,2,4	14	1,3,4
Carifen12 x Glenlea	8	1+,3,4	10	1,1+,3,4
Erik x Glenlea	10	1+,2,3,4	10	1+,4
TpxTm x HY320	18	1	26	1
Salamouni x HY320	6	1,1+,3,4	6	1,3,4
Carifen12 x HY320	10	3,4	8	2,3,4,5
Erik x HY320	10	1,1+,3	6	1,1+,3
Moderately Resistant x Moderately Susceptible Crosses				
BH1146 x Glenlea	10	1+,2,3	10	1+,2,3
BH1146 x HY320	10	1+,3	7	1+
Resistant x Resistant Crosses				
Salamouni x TpxTm	16	1	20	1
Carifen12 x TpxTm	15	1	16	1
Erik x TpxTm	2	1	10	1
Carifen12 x Salamouni	11	1,1+	12	1,1+
Erik x Salamouni	10	1,1+	9	1,1+
Erik x Carifen12	9	1,1+,3	9	1,1+
Moderately Resistant x Resistant Crosses				
BH1146 x TpxTm	3	1	17	1
BH1146 x Salamouni	14	1,2,3	14	1,2,3
BH1146 x Carifen12	10	1,1+,3,4	9	1,1+,3,4
BH1146 x Erik	10	1,1+	10	1,1+
Moderately Susceptible x Susceptible Crosses				
Glenlea x Columbus	10	3,4	8	3,4,5
HY320 x Columbus	8	3,4	9	3,4,5
Moderately Susceptible x Moderately Susceptible Cross				
HY320 x Glenlea	9	1+,2,3	10	1+,3

[1] Tan spot rating on the modified 1-5 tan spot rating scale (modified from Lamari and Bernier, 1989); the predominant ratings observed are underlined.

Table 6. Frequency distribution of tan spot ratings of hexaploid F_2's and their parents.

Cross	Code[1]	1	1+	2	3	4	5	Total
Resistant x Susceptible Crosses								
Salamouni x Columbus	6-9	6	17	1	53	38	4	119
Salamouni x Columbus	6-12	14	22	3	64	17		120
Columbus x Salamouni	5-7	6	10	1	53	47	2	119
Columbus x Salamouni	5-8	5	9	1	64	35	6	120
Carifen12 x Columbus	8-2		2		12	24	16	54
Carifen12 x Columbus	8-6	6	20	1	30	51	42	150
Columbus x Carifen12	7-1	4	13	3	32	60	16	128
Columbus x Carifen12	7-2	8	10	5	17	36	6	82
Erik x Columbus	18-4	7	30		49	32	1	119
Erik x Columbus	18-7	11	15	4	56	30	4	120
Columbus x Erik	17-1	11	35	4	44	25	1	120
Columbus x Erik	17-6	8	10	4	44	50	4	120
Moderately Resistant x Susceptible Cross								
BH1146 x Columbus	10-4	14	25		32	29		100
BH1146 x Columbus	10-7	36	33		33	18		120
Columbus x BH1146	9-1	8	25	3	37	40	1	114
Columbus x BH1146	9-7	21	35		35	20	1	112

[1] Code refers to cross-F_1 plant number.

Cross	Code[1]	1	1+	2	3	4	5	Total
Resistant x Moderately Susceptible Crosses								
Salamouni x Glenlea	4-1	25	21		50	15		111
Salamouni x Glenlea	4-6	67	35		16			118
Glenlea x Salamouni	3-2	80	33		5			118
Glenlea x Salamouni	3-5	15	33	1	58	12		119
Carifen12 x Glenlea	26-1	27	21	22	32	2		104
Carifen12 x Glenlea	26-8	21	31	11	15	5		83
Glenlea x Carifen12	25-1	20	8	8	13			49
Glenlea x Carifen12	25-6	38	36	10	28	1		113
Erik x Glenlea	32-6	54	22	16	21	7		120
Erik x Glenlea	32-9	59	14	3	35	8		119
Glenlea x Erik	31-5	13	39	6	42	19		119
Glenlea x Erik	31-8	21	27	27	30	7		112
Salamouni x HY320	38-3	63	30	1	16			110
Salamouni x HY320	38-5	42	27	3	38	8		118
HY320 x Salamouni	37-1	48	21	1	30	10		110
HY320 x Salamouni	37-3	30	33	1	40	15		119
Erik x HY320	34-2	25	52	26	15	1		119
Erik x HY320	34-3	52	30	29	7			118
HY320 x Erik	33-1	53	38	6	6			103
HY320 x Erik	33-6	43	28	7	9			87

[1] Code refers to cross-F_1 plant number.

Cross	Code[1]	1	1+	2	3	4	5	Total
Moderately Resistant x Moderately Susceptible Cross								
BH1146 x Glenlea	30-8	15	37		59	9		120
BH1146 x Glenlea	30-10	32	32	6	42	5		117
Glenlea x BH1146	29-1	16	35	2	32	4		89
Glenlea x BH1146	29-2	23	42	2	32	20		119
Resistant x Resistant Crosses								
Carifen12 x Salamouni	11-5	134	4					138
Carifen12 x Salamouni	11-9	121	7					128
Salamouni x Carifen12	12-1	129	10					139
Salamouni x Carifen12	12-3	83	16					99
Erik x Salamouni	15-6	117	2					119
Erik x Salamouni	15-7	84	6					90
Salamouni x Erik	16-4	90	25					115
Salamouni x Erik	16-8	87	13					100
Erik x Carifen12	22-5	76	27					103
Erik x Carifen12	22-7	62	17					79
Carifen12 x Erik	21-1	60	36					96
Carifen12 x Erik	21-7	73	40					113

[1] Code refers to cross-F_1 plant number.

Cross	Code[1]	1	1+	2	3	4	5	Total
Moderately Resistant x Resistant Crosses								
BH1146 x Salamouni	1-4	73	30	2	15			120
BH1146 x Salamouni	1-11	98	16		6			120
Salamouni x BH1146	2-1	99	19		2			120
Salamouni x BH1146	2-4	66	30	1	18	4		119
BH1146 x Carifen12	24-3	81	26					107
BH1146 x Carifen12	24-4	78	19	1	6			104
Carifen12 x BH1146	23-5	85	27		3			115
Carifen12 x BH1146	23-6	56	15		3			74
BH1146 x Erik	19-4	18	23	1	26	18		86
BH1146 x Erik	19-6	37	43	1	37	20		138
Erik x BH1146	20-6	46	48	7	27	12		140
Erik x BH1146	20-7	39	35		47	21		142
Moderately Susceptible x Susceptible Crosses								
Glenlea x Columbus	28-2		2	9	56	50	1	118
Glenlea x Columbus	28-3		1	9	61	48	1	120
Columbus x Glenlea	27-5		1	14	38	66	1	120
Columbus x Glenlea	27-6			6	49	65		120
HY320 x Columbus	36-1	5	16	3	65	28		117
HY320 x Columbus	36-5	4	24	1	46	30		105
Columbus x HY320	35-2		6	1	30	73	9	119
Columbus x HY320	35-3		3	1	30	72	11	117

[1] Code refers to cross-F_1 plant number.

Parent	1	1^+	2	3	4	5	Total
Resistant							
Salamouni	90	22		3			115
Carifen12	42	17					59
Erik	75	32	4				111
Moderately Resistant							
BH1146	42	40		10			92
Moderately Susceptible							
HY320		23	4	21	4		52
Glenlea		8	12	55	23		98
Susceptible							
Columbus			4	12	75	22	113

Table 7. Chi-square tests of expected F_2 ratios of hexaploid crosses inoculated with the tan spot fungus.

Cross	Code[1]	F2 Plants R	F2 Plants S	Ratio (R:S)	Chi-square	P
Resistant x Susceptible Crosses						
R = 1 + 1$^+$						
Salamouni x Columbus	6-9	23	96	1:3	2.042	0.50>P>0.10
Salamouni x Columbus	6-12	36	84	1:3	1.600	0.50>P>0.10
Columbus x Salamouni	5-7	16	103	1:3	8.473	0.01>P>0.001
				1:15	10.515	0.01>P>0.001
Columbus x Salamouni	5-8	14	106	1:3	11.378	P<0.001
				1:15	6.009	0.05>P>0.01
R = 1 + 1$^+$						
Carifen12 x Columbus	8-2	2	52	1:15	0.598	0.50>P>0.10
Carifen12 x Columbus	8-6	26	124	1:3	4.702	0.05>P>0.01
				1:15	31.447	P<0.001
Columbus x Carifen12	7-1	17	111	1:3	9.375	0.01>P>0.001
				1:15	10.800	0.01>P>0.001
Columbus x Carifen12	7-2	18	64	1:3	0.406	0.90>P>0.50
R = 1 + 1$^+$						
Erik x Columbus	18-4	37	82	1:3	2.356	0.50>P>0.10
Erik x Columbus	18-7	26	94	1:3	0.711	0.50>P>0.10
Columbus x Erik	17-1	46	74	7:9	1.431	0.50>P>0.10
Columbus x Erik	17-6	18	102	1:3	6.400	0.05>P>0.01
				1:15	15.680	P<0.001
Moderately Resistant x Susceptible Cross						
R = 1 + 1$^+$ + 2						
BH1146 x Columbus	10-4	39	61	7:9	0.917	0.50>P>0.10
BH1146 x Columbus	10-7	69	51	9:7	0.076	0.90>P>0.50
Columbus x BH1146	9-1	36	78	1:3	2.632	0.50>P>0.10
Columbus x BH1146	9-7	56	56	7:9	1.778	0.50>P>0.10
				9:7	1.778	0.50>P>0.10

[1] Code refers to cross-F_1 plant number.

Cross	Code[1]	F2 Plants R	F2 Plants S	Ratio (R:S)	Chi-square	P
Resistant x Moderately Susceptible Crosses						
$R = 1 + 1^+$						
Salamouni x Glenlea	4-1	46	65	7:9	0.240	0.90>P>0.50
Salamouni x Glenlea	4-6	102	16	3:1	8.237	0.01>P>0.001
				15:1	10.759	0.01>P>0.001
Glenlea x Salamouni	3-2	113	5	15:1	0.816	0.50>P>0.10
Glenlea x Salamouni	3-5	48	71	7:9	0.564	0.50>P>0.10
$R = 1 + 1^+$						
Carifen12 x Glenlea	26-1	48	56	7:9	0.244	0.90>P>0.50
Carifen12 x Glenlea	26-8	52	31	9:7	1.382	0.50>P>0.10
Glenlea x Carifen12	25-1	28	21	9:7	0.016	0.90>P>0.50
				7:9	3.571	0.10>P>0.05
Glenlea x Carifen12	25-6	74	39	9:7	3.918	0.05>P>0.01
				3:1	5.454	0.05>P>0.01
$R = 1 + 1^+$						
Erik x Glenlea	32-6	76	44	9:7	2.446	0.50>P>0.10
Erik x Glenlea	32-9	73	46	9:7	1.255	0.50>P>0.10
Glenlea x Erik	31-5	52	67	7:9	0.000	P = 1.00
Glenlea x Erik	31-8	48	64	7:9	0.036	0.90>P>0.50
$R = 1 + 1^+$						
Salamouni x HY320	38-3	93	17	3:1	5.345	0.05>P>0.01
				15:1	15.905	P<0.001
Salamouni x HY320	38-5	69	49	9:7	0.237	0.90>P>0.50
HY320 x Salamouni	37-1	69	41	9:7	1.875	0.50>P>0.10
HY320 x Salamouni	37-3	63	56	9:7	0.529	0.50>P>0.10
$R = 1 + 1^+$						
Erik x HY320	34-2	77	42	9:7	3.458	0.10>P>0.05
Erik x HY320	34-3	82	36	3:1	1.910	0.50>P>0.10
HY320 x Erik	33-1	91	12	3:1	9.790	0.01>P>0.001
				15:1	5.127	0.05>P>0.01
HY320 x Erik	33-6	71	16	3:1	2.027	0.50>P>0.10

[1] Code refers to cross-F_1 plant number.

Cross	Code[1]	F2 Plants R	S	Ratio (R:S)	Chi-square	P
Moderately Resistant x Moderately Susceptible Cross						
$R = 1 + 1^{+} + 2$						
BH1146 x Glenlea	30-8	52	68	7:9	0.008	0.95>P>0.90
BH1146 x Glenlea	30-10	70	47	3:1	0.609	0.50>P>0.10
Glenlea x BH1146	29-1	53	36	9:7	0.394	0.90>P>0.50
Glenlea x BH1146	29-2	67	52	9:7	0.000	P = 1.00

[1] Code refers to cross-F_1 plant number.

Table 8. Frequency distribution of tan spot ratings of the parents and BCF_1 plants of five hexaploid crosses.

Parent or Cross[1]	1	1+	2	3	4	5	Total
Salamouni	24	7					31
Columbus				3	19	4	26
BCF1(Salamouni)	54	7		2			63
BCF1(Columbus)	2	6	3	83	116	1	211
Salamouni	24	7					31
Glenlea				7	17	2	26
BCF1(Salamouni)	40	12		8			60
BCF1(Glenlea)	5	19	20	95	154	11	304
Salamouni	8	6					14
HY320		5	1	8			14
BCF1(Salamouni)	58	15		15			88
BCF1(HY320)	18	33		46			108
Salamouni	24	7					31
BH1146	3	20	1	2			26
BCF1(Salamouni)	34	10	1	9	4		58
BCF1(BH1146)	110	112	9	116	48		396
Salamouni	3	2					5
Carifen12	4	1					5
BCF1(Salamouni)	51	5					56
BCF1(BH1146)	32	4					36

[1] For each cross, distributions of tan spot ratings for the parents and the BCF_1's are shown; the female backcross parent is indicated by parentheses.

Table 9. Chi-square test for homogeneity for tan spot reaction of four F_1-derived F_3 populations of the cross, Salamouni with Glenlea.

Cross	Code[1]	F3 Families[2] R	S+S	Ratio R:S+S	Chi-square	P
R = 1 + 1$^+$						
Salamouni x Glenlea	4-8	4	21	1:3	1.213	0.50>P>0.10
Salamouni x Glenlea	4-10	9	16	1:3	1.613	0.50>P>0.10
Glenlea x Salamouni	3-10	3	22	1:3	2.253	0.50>P>0.10
Glenlea x Salamouni	3-11	9	16	1:3	1.613	0.50>P>0.10
Total					6.692	0.50>P>0.10
Pooled		25	75	1:3	0.000	P = 1.00
Heterogeneity					6.692	0.10>P>0.05

1 Code refers to cross-F_1 plant number.

2 F3 families were classified as R = homozygous resistant or S+S = segregating or homozygous susceptible.

Table 10. Tan spot ratings of tetraploid F_1's from 10 crosses involving moderately resistant, moderately susceptible, and susceptible parents.

	Cross[1]		Reciprocal[1]	
	No.	Rating	No.	Rating
Moderately Resistant x Susceptible Crosses				
4B175 x Coulter	5	1,1$^+$,2,4	5	1$^+$,2,3
4B233 x Coulter	5	2,3	5	2,3,4
4B242 x Coulter	5	1,1$^+$,3	5	1$^+$,2
Moderately Resistant x Moderately Susceptible Crosses				
4B175 x Arcola	5	1,1$^+$,2	5	1,1$^+$
4B233 x Arcola	5	2,3,4	5	1$^+$,2,4
4B242 x Arcola	5	2,3,4	5	1,3,4
Moderately Resistant x Moderately Resistant Crosses				
4B233 x 4B175	5	1$^+$,3	5	1,1$^+$,3,4
4B242 x 4B175	5	1$^+$,3	5	1,3
4B233 x 4B242	5	1,1$^+$,2	5	1,1$^+$
Moderately Susceptible x Susceptible Cross				
Arcola x Coulter	5	1$^+$,2,3,4	5	3,4

[1] Tan spot rating on the modified 1-5 tan spot rating scale (modified from Lamari and Bernier, 1989); the predominant ratings observed are underlined.

Table 11. Frequency distribution of tan spot ratings of tetraploid F_2's and their parents inoculated at the 2-leaf growth stage.

Cross or Parent	Code[1]	1	1+	2	3	4	5	Total
Moderately Resistant x Susceptible Crosses								
4B175 x Coulter	4-3	3	8	10	78			99
4B175 x Coulter	4-4	5	9	12	78			104
Coulter x 4B175	3-3	3	17	6	57			83
Coulter x 4B175	3-7	3	7	13	54			77
4B242 x Coulter	2-1	6	6	22	85			119
4B242 x Coulter	2-7	6	1	15	33			55
Coulter x 4B242	1-4	9	8	16	39			72
Coulter x 4B242	1-5	3	2	9	33			47
Moderately Resistant x Moderately Resistant Cross								
4B242 x 4B175	5-7	89	2					91
4B242 x 4B175	5-8	73	3					76
4B175 x 4B242	6-4	60	25					85
4B175 x 4B242	6-6	42	2					44
Parents								
4B175		27	5					32
4B242		20	10	6				36
Coulter					26			26

[1] Code refers to cross-F_1 plant number.

Table 12. Frequency distribution of tan spot ratings of tetraploid F_2's and their parents inoculated at the 4-6 leaf growth stage.

Cross	Code[1]	1	1+	2	3	4	5	Total
Moderately Resistant x Susceptible Crosses								
4B175 x Coulter	4-3		14	14	70	1		99
4B175 x Coulter	4-4		6	19	76	3		104
Coulter x 4B175	3-3		12	12	59			83
Coulter x 4B175	3-7		8	16	50	3		77
4B233 x Coulter	14-3		28	16	35	1		80
4B233 x Coulter	14-6	6	11	3	19	1		40
Coulter x 4B233	13-3		6		51	3		60
Coulter x 4B233	13-5	1	13	4	11			29
4B242 x Coulter	2-1		9	45	64	1		119
4B242 x Coulter	2-7		1	32	22			55
Coulter x 4B242	1-4		2	30	39	1		72
Coulter x 4B242	1-5		3	19	24	1		47
Moderately Resistant x Moderately Susceptible Crosses								
4B175 x Arcola	10-1	7	16	7	47	1		78
4B175 x Arcola	10-7		16	11	57	4		88
Arcola x 4B175	9-8	6	14	4	62	3		89
Arcola x 4B175	9-10	8	23	7	31			69
4B233 x Arcola	16-3	28	25	4	23			80
4B233 x Arcola	16-4	24	15	7	21		1	68
Arcola x 4B233	15-1	14	26	14	20			74
Arcola x 4B233	15-4	10	25	1	47		1	84

[1] Code refers to cross-F_1 plant number.

Parent or Cross	Code[1]	1	1+	2	3	4	5	Total
4B242 x Arcola	8-1	3	19	33	33	4		92
4B242 x Arcola	8-9	10	36	25	46			117
Arcola x 4B242	7-7	2	15	14	61	24	4	120
Arcola x 4B242	7-9	20	28	29	28	2		107
Moderately Resistant x Moderately Resistant Crosses								
4B233 x 4B175	17-6	5	17	12	36			70
4B233 x 4B175	17-8	1	12		7			20
4B175 x 4B233	18-4	8	24	4	55			91
4B175 x 4B233	18-6	46	56	1	5			108
4B242 x 4B175	5-7	21	65	5				91
4B242 x 4B175	5-8	6	66	4				76
4B175 x 4B242	6-4	7	64	14				85
4B175 x 4B242	6-6	3	24	17				44
4B233 x 4B242	19-1	1	16	4	48			69
4B233 x 4B242	19-4	8	34	11	36			89
4B242 x 4B233	20-1		12	6	57			75
4B242 x 4B233	20-6	33	30	2	1			66
Moderately Susceptible x Susceptible Cross								
Arcola x Coulter	12-12	1	11	5	50			67
Arcola x Coulter	12-15		9	6	79	4		98
Coulter x Arcola	11-13	1	11	1	40	2		55
Coulter x Arcola	11-15		4		47	2		53

[1] Code refers to cross-F_1 plant number.

Parent	1	1^+	2	3	4	5	Total
Moderately Resistant							
4B175	21	35	11	3			70
4B233	13	29	4	6			52
4B242	8	29	26	11			74
Moderately Susceptible							
Arcola		6	6	55	2		69
Susceptible							
Coulter				35	8		43

Table 13. Chi-square tests of expected F_2 genetic ratios for tetraploid crosses inoculated with the tan spot fungus at the 2-leaf growth stage.

Cross	Code[1]	F2 Plants R	F2 Plants S	Ratio (R:S)	Chi-square	P
Moderately Resistant x Susceptible Crosses						
R = 1 + 1$^+$ + 2						
4B175 x Coulter	4-3	21	78	1:3	0.758	0.50>0>0.10
4B175 x Coulter	4-4	26	75	1:3	0.000	P = 1.00
Coulter x 4B175	3-3	26	57	1:3	1.771	0.50>P>0.10
Coulter x 4B175	3-7	23	54	1:3	0.974	0.50>P>0.10
R = 1 + 1$^+$ + 2						
4B242 x Coulter	2-1	34	85	1:3	0.809	0.50>P>0.10
4B242 x Coulter	2-7	22	33	7:9	0.314	0.90>P>0.50
Coulter x 4B242	1-4	33	39	7:9	0.127	0.90>P>0.50
				9:7	3.175	0.10>P>0.05
Coulter x 4B242	1-5	14	33	1:3	0.574	0.50>P>0.10

[1] Code refers to cross-F_1 plant number.

Table 14. Chi-square tests of expected F_2 genetic ratios for tetraploid crosses inoculated with the tan spot fungus at the 4-6 leaf growth stage.

Cross	Code[1]	F2 Plants R	F2 Plants S	Ratio (R:S)	Chi-square	P
Moderately Resistant x Susceptible Crosses						
R = 1 + 1$^+$ + 2						
4B175 x Coulter	4-3	28	71	1:3	0.569	0.50>P>0.10
4B175 x Coulter	4-4	25	79	1:3	0.051	0.95>P>0.90
Coulter x 4B175	3-3	24	59	1:3	0.679	0.50>P>0.10
Coulter x 4B175	3-7	24	53	1:3	1.563	0.50>P>0.10
R = 1 + 1$^+$ + 2						
4B233 x Coulter	14-3	44	36	9:7	0.051	0.95>P>0.90
4B233 x Coulter	14-6	20	20	7:9	0.635	0.50>P>0.10
				9:7	0.635	0.50>P>0.10
Coulter x 4B233	13-3	6	54	1:15	1.440	0.50>P>0.10
Coulter x 4B233	13-5	18	11	9:7	0.399	0.90>P>0.50
R = 1 + 1$^+$ + 2						
4B242 x Coulter	2-1	54	65	7:9	0.128	0.90>P>0.50
4B242 x Coulter	2-7	33	22	9:7	0.314	0.90>P>0.50
Coulter x 4B242	1-4	32	40	7:9	0.014	0.95>P>0.90
Coulter x 4B242	1-5	22	25	7:9	0.179	0.90>P>0.50
				9:7	1.702	0.50>P>0.10

[1] Code refers to cross-F_1 plant number.

Table 15. Chi-square tests for homogeneity of expected F_2 genetic ratios of one tetraploid cross, 4B175 with Coulter, inoculated with the tan spot fungus at the 2-leaf and 4-6 leaf growth stages.

Cross	Code[1]	F2 Plants R	F2 Plants S	Ratio (R:S)	Chi-square	P
R = 1 + 1$^+$ + 2 (2-leaf stage)						
4B175 x Coulter	4-3	21	78	1:3	0.758	0.50>P>0.10
4B175 x Coulter	4-4	26	78	1:3	0.000	P = 1.00
Coulter x 4B175	3-3	26	57	1:3	1.771	0.50>P>0.10
Coulter x 4B242	3-7	23	54	1:3	0.974	0.50>P>0.10
Total					3.503	0.50>P>0.10
Pooled		96	267	1:3	0.405	0.90>P>0.50
Heterogeneity					3.098	0.50>P>0.10
R = 1 + 1$^+$ + 2 (4-6 leaf stage)						
4B175 x Coulter	4-3	28	71	1:3	0.569	0.50>P>0.10
4B175 x Coulter	4-4	25	79	1:3	0.051	0.95>P>0.90
Coulter x 4B175	3-3	24	59	1:3	0.679	0.50>P>0.10
Coulter x 4B242	3-7	24	53	1:3	1.563	0.50>P>0.10
Total					2.862	0.90>P>0.50
Pooled		101	262	1:3	1.544	0.50>P>0.10
Heterogeneity					1.318	0.90>P>0.50

[1] Code refers to cross-F_1 plant number.

Table 16. Tan spot ratings of diploid parents and F_1's from two resistant x susceptible and two susceptible x susceptible crosses.

	No. of Plants	Tan Spot[1] Rating
Resistant x Susceptible Crosses		
2B27 x 2B37	6	4,5
2B53 x 2B37	1	5
Resistant x Resistant Crosses		
2B27 x 2B26	5	1
2B26 x 2B37	1	1
Parents		
2B7	3	1
2B26	6	1
2B27	5	1
2B53	3	1
2B37	8	5

[1] Tan spot rating on the modified 1-5 tan spot rating scale (modified from Lamari and Bernier, 1989); the predominant rating observed is underlined.

Table 17. Frequency distribution of tan spot ratings of diploid F_2's and their parents.

Parent or Cross	Code[1]	1	1+	2	3	4	5	Total
Resistant x Susceptible Crosses								
2B27 x 2B37	1-1	17	18	11	12	37	22	95
2B27 x 2B37	1-2	18	5	7	10	32	30	102
2B27 x 2B37	1-3	24	5	4	8	44	16	101
2B27 x 2B37	1-4	21	3	4	4	39	9	80
2B53 x 2B37	2-1	5		1	1	11	2	20
Resistant x Resistant Crosses								
2B27 x 2B26	3-1	104	5					109
2B27 x 2B26	3-2	98	4					102
2B27 x 2B26	3-3	89	8					97
2B27 x 2B26	3-4	85	12					97
2B26 x 2B7	4-1	82	3					85
Parents								
Resistant								
2B7		20	1					21
2B26		20	1					21
2B27		31	11					42
2B53		13						13
Susceptible								
2B37						3	1	4

[1] Code refers to cross-F_1 plant number.

Table 18. Chi-square tests of expected F_2 ratios of resistant x susceptible diploid crosses inoculated with the tan spot fungus.

Cross	Code[1]	F2 Plants R	F2 Plants S	Ratio (R:S)	Chi-square	P
$R = 1 + 1^+$						
2B27 x 2B37	1-1	25	70	1:3	0.088	0.90>P>0.50
2B27 x 2B37	1-2	23	79	1:3	0.327	0.90>P>0.50
2B27 x 2B37	1-3	29	72	1:3	0.743	0.50>P>0.10
2B27 x 2B37	1-4	24	56	1:3	1.067	0.50>P>0.10
Total					2.225	0.90>P>0.50
Pooled		101	277	1:3	0.596	0.50>P>0.10
Heterogeneity					1.629	0.90>P>0.50
$R = 1 + 1^+$						
2B53 x 2B37	2-1	5	15	1:3	0.000	P = 1.00

[1] Code refers to cross-F_1 plant number.

DISCUSSION

1. Hexaploids

This is the first report of a detailed inheritance study in which tan spot reaction was assessed on the basis of the qualitative criterion, lesion type, described by Lamari and Bernier (1989). Assessment of tan spot reaction on the basis of lesion type facilitated the detection of qualitative inheritance of resistance to the ASC1 isolate of the tan spot fungus in this study. To date, there is only one published report of qualitative inheritance of tan spot resistance; the resistance of the hexaploid winter wheat cultivar, Carifen12 to the PYOK-2 isolate was attributed to one recessive gene pair (Lee and Gough, 1984).

In this study, F_2 data from crosses of the resistant parents, Salamouni, Carifen12, and Erik with the susceptible parent, Columbus indicated that the three resistant parents possessed one or possibly two recessive gene pairs for tan spot resistance. Inconsistency in the F_2 ratios may have resulted from genetic heterogeneity in the parents or misclassification due to growth stage and leaf age effects or incomplete dominant gene action (as suggested by the F_1 data. BCF_1 results of the cross, Salamouni with Columbus, and the crosses of Salamouni with the two moderately susceptible parents, Glenlea with HY320, differed from expectations for a one or two recessive resistance gene model, but were in agreement with predictions for one resistance gene with incomplete dominant gene action. F_3 results of the cross, Salamouni with the moderately susceptible parent, Glenlea, indicated that Salamouni possessed one dominant or incompletely dominant gene for resistance. F_2 results from intercrosses among the resistant parents indicated that Salamouni, Carifen12, and Erik had the same gene or genes for resistance.

BCF_1 results of the cross, Salamouni with Carifen12 provided additional support for this conclusion. Considering the F_2, BCF_1, and F_3 results together, it is likely that Salamouni, Carifen12, and Erik have in common one gene for tan spot resistance which exhibits recessive or incomplete dominant gene action. It is possible that one or more modifier genes may affect the expression of this resistance gene under different growth conditions or in different genetic backgrounds.

The results of this study are in agreement with those of Lamari (1988) who concluded that Salamouni possessed one recessive gene pair for resistance to the ASC1 isolate. Although inconsistency in F_2 ratios were not observed, Lamari studied only two populations. As well, the inheritance of resistance was studied at the 2-leaf stage rating only one leaf, which may also have reduced the potential for variability. In this study, growth stage effects were found to have the potential to alter genetic interpretation of F_2 results; in three tetraploid crosses inoculated at both the 2-leaf and 4-6 leaf stage, fewer resistant F_2 plants were identified at the 4-6 leaf stage than at the 2-leaf stage. The results of the present study are also in agreement with the additional findings of Lamari (1988) who presented results of two 2-leaf stage BCF_1 populations, (Erik x Columbus) x Erik and (Erik x Celtic) x Erik which indicated that the resistant parent, Erik, possessed one recessive gene pair for resistance to the ASC1 isolate.

In the present study, F_2 results were variable for the crosses which involved the moderately resistant parent, BH1146, or moderately susceptible parents, HY320 and Glenlea. Suspected genetic heterogeneity in the parents likely contributed to the inconclusive F_2 results. The failure to obtain

conclusive results may also have resulted from the difficulty in classification due to the use of moderate levels of resistance and susceptibility, possible growth stage or leaf age effects, additional modifier genes, and incomplete dominant gene action (suggested by the F_1 data) on the expresssion of resistance. Despite such difficulties, some tentative conclusions may be drawn from the results.

F_2 results of the crosses of the moderately resistant parent, BH1146 with the moderately susceptible parent, Glenlea, and susceptible parent, Columbus, suggested that BH1146 may possess one or two genes for resistance (with various types of gene action). Previous inheritance studies involving BH1146 reported a lack of fit to expected monogenic or digenic ratios and therefore concluded that the resistance of BH1146 was quantitative (Nagle et al., 1982). In this study, F_2 results of intercrosses of BH1146 with the three resistant parents, Salamouni, Carifen12, and Erik suggested that BH1146 was genetically heterogeneous and that some BH1146 genotypes possessed a different resistance gene or genes. BCF_1 and F_3 results of the cross, Salamouni with BH1146, provided further evidence to suggest that BH1146 and Salamouni (and thus the other resistant parents) had different resistance genes.

F_2 results of the moderately susceptible x susceptible crosses were as expected for the cross, Glenlea with Columbus; all F_2's had tan spot ratings within the range of the parents. However, resistant segregants were observed for the cross, HY320 with Columbus. It is likely that the moderately resistant parent, HY320 and the susceptible parent, Columbus differed for minor genes for resistance resulting in the recovery of F_2 plants with a tan spot rating lower than that of the parents (rating 1). Resistance which is

based on the accumulation of several minor genes has been observed in other host-pathogen systems: wheat-Puccinia striiformis, wheat-Puccinia graminis tritici, wheat-Puccinia recondita, oats-Puccinia graminis avenae, and oats-Puccinia coronata (Krupinski and Sharp, 1978; Knott, 1982; Samborski and Dyck, 1982; Martens et al., 1981, cited by Harder and McKenzie, 1984). An apparent reciprocal effect was also detected in the cross, HY320 with Columbus, but further testing is needed to verify this effect. Since reciprocal differences were not detected in the F_2 populations of any of the other 17 hexaploid crosses tested nor in the F_1 generation of the 28 crosses studied, it is unlikely that the apparent reciprocal effect of this study is real. Reciprocal differences had not been tested in previous inheritance studies of tan spot resistance.

2. Tetraploids

As with the hexaploids, evaluation of tan spot reaction on the basis of the qualitative criterion, lesion type, enabled the detection of qualitative inheritance of resistance. To date, there have been no reports of qualitative inheritance of tan spot resistance in tetraploid wheat. However, further testing to confirm the results of this study is advisable since genetic heterogeneity in all parents except the susceptible parent, Coulter, was suspected given the considerable variability in tan spot ratings observed. Small population sizes, growth stage or leaf age effects and the effects of incompletely dominant gene action (suggested by the F_1 data) on the expression of resistance may have contributed to the inconsistency of the F_2 results.

Evidence of qualitative inheritance of resistance was obtained from the

moderately resistant x susceptible crosses. For the cross, 4B175 with Coulter, F_2 data for both the 2-leaf and 4-6 leaf growth stages indicated that 4B175 possessed one recessive gene pair for resistance. F_2 results of the other two moderately resistant x susceptible crosses (4B233 with Coulter, and 4B242 with Coulter) were inconsistent, but evidence that 4B233 and 4B242 possessed either one or two genes for resistance (with various types of gene action) was obtained. However, F_2 results of the intercrosses among the resistant parents suggested that they shared the same gene(s) for resistance.

These results are in general agreement with the results of Lamari (1988) who reported that one recessive gene pair accounted for the resistance of 4B242 in one F_2 population of the cross, 4B242 x Coulter, inoculated at the 2-leaf stage with the ASC1 isolate.

The lack of conclusive results in the other tetraploid crosses may have been due to many factors, such as genetic heterogeneity in the parents, small population sizes, and growth stage or leaf age effects. Although further studies of growth stage effects are needed, the ability of growth stage effects to potentially alter genetic ratios was revealed in F_2 results of three tetraploid crosses inoculated at both the 2-leaf and 4-6 leaf stages; for all three crosses, fewer resistant plants were detected at the 4-6 leaf stage.

Another possible explanation for the lack of conclusive results is that additional minor resistance genes may be operative in these crosses. Tan spot resistance in tetraploid wheat has previously been thought to be under the control of more than one or two genes (Nagle et al., 1982; Cantrell et al., 1985; Elias et al., 1989). As with the hexaploids, evidence of minor tan spot resistance genes was found in the detection of resistant (rating 1)

segregants within F_2's of the moderately susceptible x susceptible cross, Arcola with Coulter. Perhaps the "susceptible" parents differed for a number of minor resistance genes, which when combined, resulted in a few F_2 plants with tan spot ratings lower than that of the parents. Resistance attributed to the accumulation of several minor genes has been reported in other host:pathogen systems (Harder and McKenzie, 1984). Although such quantitative resistance may be more difficult to manipulate in a breeding program, it is encouraging to know that resistant selections may be recovered from crosses of susceptible material. As well, such resistance is considered more complex and is thus expected to be more long-lasting or durable than simply, qualitatively inherited genes (Dyck and Kerber, 1982).

As with the hexaploids, there was no evidence of reciprocal differences in the F_1 generation of the 10 tetraploid crosses tested. Likewise, reciprocal differences were not apparent in the F_2 generation of these crosses. Reciprocal differences have not been tested in previous tan spot inheritance studies in tetraploid wheat.

3. Diploids

In contrast to the hexaploid and tetraploid inheritance studies, results of the diploid inheritance study were very clearcut. Qualitative inheritance of tan spot resistance was clearly established at the diploid level. The lack of variability in the parents and the use of parents representing extremes of the range of tan spot reaction likely contributed to the clarity of the results. The F_1 data suggested that resistance was recessive and the F_2 results confirmed this. F_2 results indicated that the resistant parents, 2B27 and 2B53, each carried one recessive gene pair for resistance which may

or may not be common to both. As well, lack of segregation in the F_2 indicated that the resistant parents, 2B27, 2B26, and 2B7, possessed the same gene or genes for resistance. At the present time, there are no reports in the literature of the inheritance of tan spot resistance in diploid wheat with which to compare the results of this study, although there are reports of screening for tan spot resistance in diploid wheat (Lamari and Bernier, 1989) and in the related wild wheat, Aegilops spp. (Alam and Gustafson, 1988).

Additional inheritance studies are needed to determine whether the single resistance genes identified in the diploid, tetraploid, and hexaploid studies are the same, and thus reside on one of the chromosomes of the A genome. Searching for additional simply inherited tan spot resistance genes at the diploid level may be necessary if such genes cannot be easily identified at the hexaploid and tetraploid level. The identification of simply inherited tan spot resistance genes at all ploidy levels should be welcomed by breeders.

GENERAL DISCUSSION

In this study, use of the qualitative disease assessment criterion, lesion type, described by Lamari and Bernier (1989), facilitated the detection of qualitative inheritance of resistance to the ASC1 isolate of the tan spot fungus in hexaploid, tetraploid, and diploid wheat. This is the first detailed inheritance study concerning tan spot of wheat in which numerous examples of qualitative inheritance were obtained. This study confirmed and expanded the conclusions of Lamari (1988), in which qualitative inheritance of resistance to the ASC1 isolate was first observed. The only published report of qualitative inheritance of tan spot resistance was that of Lee and Gough (1984) who found that the winter wheat cultivar, Carifen12 had one recessive gene pair for resistance to the PYOK-2 isolate. In this study, the hexaploid results indicated that Carifen12 and the other resistant parents, Salamouni and Erik, likely have in common one recessive gene pair for resistance to the ASC1 isolate. It would be interesting to determine whether the resistance gene identified by Lee and Gough (1984) which gives resistance to the PYOK-2 isolate is the same as that which conditions resistance to the ASC1 isolate.

In addition, further studies are needed to determine whether the resistance gene effective against the ASC1 isolate provides resistance to other isolates of the tan spot fungus. From a local breeding perspective, the resistance gene identified in Salamouni, Carifen12, and Erik needs to be shown to be effective against the tan spot isolates commonly found in western Canada. The ASC1 isolate originated in Manitoba and is believed to be representative of the isolates found in this area.

The ASC1 isolate used in this inheritance study is also known to belong

to the nec^+ virulence group which brings about necrosis on susceptible hosts (Lamari and Bernier, 1989). Another virulence group, denoted nec^- ch^+, is capable of causing extensive chlorosis on susceptible hosts. Perhaps the inheritance of resistance to an isolate of the nec^- ch^+ virulence group could be studied.

Results of this study also indicated qualitative inheritance of resistance to the ASC1 isolate in tetraploid wheat. One recessive gene pair was found to condition resistance in the moderately resistant parent, 4B175, when tested at both the 2-leaf and 4-6 leaf growth stages. Evidence was found to suggest that the other moderately resistant parents, 4B233 and 4B242, shared the same gene(s) for resistance as 4B175, but the considerable variability in the parental reactions made such a conclusion only tentative. Further studies are needed to clarify and confirm these results. As with the hexaploids, it would be useful to determine whether these tetraploid parents are resistant to additional isolates of the tan spot fungus, in particular those typically found in Manitoba. Further testing and inheritance studies of additional resistant tetraploid germplasm would be of benefit to breeders.

Qualitative inheritance of resistance was clearly evident in the diploid study. The resistant parents, 2B27 and 2B53, were found to possess one recessive gene pair for resistance to the ASC1 isolate. Additional study involving an intercross between the parents is needed to determine whether they have the same resistance gene. The resistant parents, 2B7 and 2B26, were found to possess the same resistance gene(s) as 2B27. Further studies at the diploid level are needed to determine whether new genes for tan spot resistance exist within diploid germplasm. Diploid wheat may provide a valuable source of tan spot resistance genes for breeding purposes if such

genes are either not available or not easily identifiable in hexaploid and tetraploid wheat. Wild wheat species are presently considered a valuable germplasm reserve for rust resistance genes.

Although monogenic or digenic qualitative inheritance was detected within all three ploidy levels in this study, some evidence to suggest that the inheritance of tan spot resistance was more complex appeared in the hexaploid and tetraploid studies. For the hexaploid cross involving the moderately susceptible parent, HY320 and susceptible parent, Columbus, several resistant segregants appeared in the F_2 generation. Similarly, in the tetraploid cross of the moderately susceptible parent, Arcola with the susceptible parent, Coulter, resistant segregants were detected in the F_2 populations. In each case, it is likely that the "susceptible" parents differed for a number of minor genes for resistance. These minor resistance genes may provide an additional source of tan spot resistance and may be valuable in producing cultivars with a broad genetic base of resistance. This resistance may prove more durable than resistance based on one major, qualitatively inherited gene (Dyck and Kerber, 1985). The accumulation of minor genes in wheat for resistance to stripe rust, stem rust, and leaf rust has been noted in various studies (Krupinski and Sharp, 1978; Knott, 1982; Samborski and Dyck, 1982, cited by Harder and McKenzie, 1984).

In this study, the discovery that tan spot resistance is qualitatively inherited should be welcomed by breeders. In addition, the lack of reciprocal differences in the hexaploid and tetraploid crosses (with the possible exception of one hexaploid cross) is encouraging from a breeding perspective; direction of the cross need not be a constraint in breeding for tan spot resistance. This is the first study of the inheritance of tan spot

resistance in wheat in which reciprocal crosses were tested.

Although evidence of qualitative inheritance of tan spot resistance was obtained in this study, the results of some hexaploid and tetraploid crosses were inconclusive. It is likely that genetic heterogeneity of the parents (in particular, the moderately resistant parents, BH1146, 4B175, 4B233, and 4B242, and the moderately susceptible parents, HY320, Glenlea, and Arcola) contributed to the inconclusive results. This study could have been greatly improved by ensuring genetic homogeneity of the parents before crossing was begun, but time constraints did not permit such testing.

Another likely contributor to the inconclusive results was the effect of incompletely dominant gene action (suggested by the F_1 hexaploid and tetraploid data) on the phenotypic expression of resistance. Incompletely dominant gene action obscures the resistant reaction and makes classification much more difficult. Testing in the F_3 generation using larger F_3 family sizes would have improved the ease of classification. Similarly, evaluation of the BCF_2 generation rather than BCF_1 plants would likely have improved the study. Unfortunately, time and space constraints did not allow such testing.

In this study, use of the qualitative disease assessment criterion, lesion type (described by Lamari and Bernier, 1989) facilitated the detection of qualitative inheritance of tan spot resistance within hexaploid, tetraploid, and diploid wheat. The use of lesion type for the assessment of tan spot reaction is recommended for future tan spot studies due to the ease of rating and reproducibility of results (providing the lines are homogeneous and appropriate experimental conditions are used). Testing at the 2-leaf stage and rating only one leaf would have simplified and greatly

increased the efficiency of the study although the younger growth stage used may have altered the genetic interpretation. In this study, results of three tetraploid crosses indicated growth stage effects could potentially alter genetic ratios. More work is needed to understand the effect of growth stage on the expression of tan spot reaction. Further research is also needed to reveal the epidemiological relationship between lesion type and resistance/susceptibility. Such studies are particularly important for those lines which express moderately resistant or moderately susceptible reactions.

Overall, this study represents the first detailed inheritance study of tan spot of wheat in which numerous examples of qualitative inheritance were found. This is the first report of the inheritance of tan spot resistance in diploid wheat. As well, this is the first tan spot inheritance study in which reciprocal crosses were tested. Additional research is needed to increase our understanding of tan spot resistance in wheat, but hopefully this study has contributed in some part toward this goal.

REFERENCES

Alam, K.B. and Gustafson, J.P. 1988. Tan-spot resistance screening of Aegilops species. Plant Breeding. 100:112-118.

Briggs, F.N. and Knowles, P.F. 1967. Introduction to plant breeding. Reinhold Publ. Corp., U.S.A.

Cantrell, R.G. 1982. Selecting and breeding durum wheat for resistance to tan spot. [in: Tan Spot of Wheat and Related Diseases Workshop. Hosford, R.M. Jr. (ed.), North Dakota State Univ., Fargo, N.D.].

Cantrell, R.G., Elias, E. and Hosford, R.M. Jr. 1985. The inheritance of resistance to tan spot in durum wheat. Agron. Abstr. 77:50 (Abstr.).

Claude, P.P. 1985. The evaluation of 391 spring wheat introductions for resistance to stem rust, leaf rust, loose smut and tan spot. M.Sc. thesis, Dept. of Plant Science, University of Manitoba, Winnipeg, Man.

Cochran, W.G. and Cox, G. 1957. Experimental designs. 2nd ed. John Wiley & Sons, Inc. pp. 376-395.

Cowling, E.B. and Horsfall, J.G. 1980. Prologue: how plants defend themselves [in: Plant Disease: An Advanced Treatise. Vol. V. Horsfall, J.G. and Cowling, E.B. (eds.), Academic Press, New York].

Cox, D.J. and Hosford, R.M. Jr. 1987. Resistant winter wheats compared at differing growth stages and leaf positions for tan spot severity. Plant Dis. 71: 883-886.

Cunfer, B.M. 1987. Bacterial and fungal blights of the foliage and heads of wheat. [in: Wheat and Wheat Improvement. 2nd ed. Heyne, E.G. (ed.), ASA-CSSA-SSA, Madison, Wisc. pp. 528-541].

Day, P.R. 1974. Genetics of host-parasite interaction. Freeman and Co., San Francisco.

Dyck, P.L. and Kerber, E.R. 1985. Resistance of the race-specific type. [in: The Cereal Rusts. Vol. II. Academic Press, New York, pp. 469-500.]

Elias, E., Cantrell, R.G., and Hosford, R.M. 1989. Heritability of resistance to tan spot in durum wheat and its association with other agronomic traits. Crop Sci. 29:299-304.

Frohberg, R.C. 1982. Breeding hard red spring wheat for resistance to tan spot. [in: Tan Spot of Wheat and Related Diseases Workshop. Hosford, R.M. Jr. (ed.), North Dakota State Univ., Fargo, N.D. p. 48].

Gough, F.J. and Johnston, R.A. 1982. Observations on Septoria leaf blotch and Pyrenophora tan spot in Oklahoma in 1981. [in: Tan Spot of Wheat and Related Diseases Workshop. Hosford, R.M. Jr. (ed.), North Dakota State Univ., Fargo, N.D. pp. 95-100].

Griffing, B. 1956. Concept of general and specific combining ability in relation to diallel crossing systems. Aust. J. Biol. Sci. 9:463-493.
Hosford, R.M. Jr. 1971. A form of Pyrenophora trichostoma pathogenic to wheat and other grasses. Phytopathology 61:28-32.

Harder, D.E. and McKenzie, R.I.H. 1984. Complex additive-type of resistance to Puccinia coronata in Avena sterilis. Can. J. Plant Pathol. 6:135-138.

Hosford, R.M. Jr. 1972. Propagules of Pyrenophora trichostoma. Phytopathology. 62:627-629.

Hosford, R.M. Jr. 1982. Tan spot. [in: Tan Spot of Wheat and Related Diseases Workshop. Hosford, R.M. Jr. (ed.), North Dakota State Univ., Fargo, N.D. pp. 1-24].

Hosford, R.M. Jr. and Busch, R.H. 1974. Losses in wheat caused by Pyrenophora trichostoma and Leptosphaeria avenaria f.sp. triticea. Phytopathology 64:184-187.

Hosford, R.M. Jr., Larez, C.R. and Hammond, J.J. 1987. Interaction of wet period and temperature on Pyrenophora tritici-repentis infection and development in wheats of differing resistance. Phytopathology. 77:1021-1027.

Huber, D.M., Lee, T.S., Ross, M.A. and Abney, T.S. 1987. Amelioration of tan spot-infected wheat with nitrogen. Plant Dis. 71:49-50.

Hunger, R.M. and Brown, D.A. 1987. Colony color, growth, sporulation, fungicide sensitivity, and pathogenicity of Pyrenophora tritici-repentis. Plant Dis. 71:907-910.

Keeling, B.L. and Bantarri, E.E. 1975. Factors associated with the resistance of barley to Helminthosporium teres. Phytopathology. 65:464-467.

Khan, T.N. and Boyd, W.J.R. 1969. Environmentally induced variability in the host reaction of barley to net blotch. Aust. J. Biol. Sci. 22:1237-1244.

Krupinsky, J.M. 1982. Observations on the host range of isolates of Pyrenophora trichostoma. Can. J. Plant Pathol. 4:42-46.

Lamari, L. 1988. Tan spot of wheat, caused by Pyrenophora tritici-repentis: host and pathogen variability, host-pathogen interactions and involvement of toxin in disease. Ph.D. thesis, Dept. of Plant Science., University of Manitoba, Winnipeg, Man.

Lamari, L. and Bernier, C.C. 1989. Evaluation of wheat lines and cultivars to tan spot (Pyrenophora tritici-repentis) based on lesion types. Can. J. Plant Pathol. (in press)

Lamey, H.A. and Hosford, R.M. Jr. 1982. Tan spot of wheat. Coop. Ext. Serv., North Dakota State Univ., Fargo, N.D., USDA Circ. PP-766.

Larez, C.R., Hosford, R.M. Jr. and Freeman, T.P. 1986. Infection of wheat and oats by Pyrenophora tritici-repentis and initial characterization of resistance. Phytopathology. 76:931-938.

Large, E.E. 1954. Growth stages in cereals -- illustrations of the Feekes scale. Plant Pathol. 3:128-129.

Lee, T.S. and Gough, F.J. 1984. Inheritance of Septoria leaf blotch (S. tritici) and Pyrenophora tan spot (P. tritici-repentis) resistance in Triticum aestivum cv. Carifen 12. Plant Dis. 68:848-851.

Loughman, R. and Deverall, B.J. 1986. Infection of resistant and susceptible cultivars of wheat by Pyrenophora tritici-repentis. Plant Pathol. 35:443-450.

Luz, da W.C, and Bergstrom, G.C. 1986. Evaluation of triadimenol seed treatment for early season control of tan spot, powdery mildew, spot blotch and Septoria nodorum spot on spring wheat. Crop Prot. 5:83-87.

Luz, da W.C. and Hosford, R.M. Jr. 1980. Twelve Pyrenophora trichostoma races for virulence to wheat in the Central Plains of North America. Phytopathology 70:1193-1196.

Lyster, B. 1987. Do fungicides pay in spring wheat? Country Guide (Western Edition) 106:34-34B.

M.A.S.C.C. 1986. Field crop production recommendations for Manitoba. Manitoba Agriculture, Manitoba Agricultural Services Coordinating Committee.

Manitoba Agriculture. 1988. Field crop production guide for Manitoba 1988-90. Manitoba Agriculture Publ., Winnipeg, Man.

Morrall, R.A.A. and Howard, R.J. 1975. The epidemiology of leaf spot disease in a native prairie. II. Airborne spore populations of Pyrenophora tritici-repentis. Can. J. Bot. 53:2345-2353.

Misra, A.P. and Singh, R.A. 1972. Pathogenic differences amongst three isolates of Helminthosporium tritici-repentis and the performance of wheat varieties against them. Indian Phytopathol. 25:350-353.

Nagle, B.J., Frohberg, R.C. and Hosford, R.M. Jr. 1982. Inheritance of resistance to tan spot of wheat. [in: Tan Spot of Wheat and Related Diseases Workshop. Hosford, R.M. Jr. (ed.), North Dakota State Univ., Fargo, N.D. pp. 40-45].

Nelson, R.R. 1973. Breeding plants for disease resistance -- concepts and applications. Penn. State Univ. Press, Pennsylvania.

Odvody, G.N., Boosalis, M.G., and Watkins, J.E. 1982. Development of pseudothecia during progressive saprophytic colonization of wheat straw by Pyrenophora trichostoma. [in: Tan Spot of Wheat and Related Diseases Workshop. Hosford, R.M. Jr. (ed.), North Dakota State Univ., Fargo, N.D. pp. 33-35].

Pfender, W.F. and Wootke, S.L. 1987. Production of pseudothecia and ascospores by Pyrenophora tritici-repentis in response to macronutrient concentrations. Phytopathology 77:1213-1216.

Platt, H.W. and Morrall, R.A.A. 1980. Effects of light intensity and relative humidity on conidiation in Pyrenophora tritici-repentis. Can. J. Plant Pathol. 2:53-57.

Platt, H.W., Morrall, R.A.A. and Gruen, H.E. 1977. The effects of substrate, temperature, and photoperiod on conidiation of Pyrenophora tritici-repentis. Can. J. Bot. 55:254-259.

Raymond, P.J. and Bockus, W.W. 1982. An in vitro technique for profuse sporulation of Drechslera tritici-repentis. Phytopathology. 72:934 (Abstr.).

Raymond, P.J., Bockus, W.W. and Norman, B.L. 1985. Tan spot of winter wheat: procedures to determine host response. Phytopathology. 75:686-690.

Rees, R.G., Mayer, R.J. and Platz, G.J. 1981. Yield losses in wheat from yellow spot: a disease-loss relationship derived from single tillers. Aust. J. Agric. Res. 32:851-859.

Rees, R.G. and Platz, G.J. 1980. The epidemiology of yellow spot of wheat in Southern Queensland. Aust. J. Agric. Res. 31:259-267.

Rees, R.G. and Platz, G.J. 1983. Effects of yellow spot on wheat: comparison of epidemics at different stages of crop development. Aust. J. Agric. Res. 34:39-46.

Rees, R.G., Platz, G.J. and Mayer, R.J. 1982. Yield losses in wheat from yellow spot: comparison of estimates derived from single tillers and plots. Aust. J. Agric. Res. 33:899-908.

Sebesta, P.G. 1982. Breeding hard red winter wheat for resistance to tan spot. [in: Tan Spot of Wheat and Related Diseases Workshop. Hosford, R.M. Jr. (ed.), North Dakota State Univ., Fargo, N.D. p. 50].

Sharp, E.L., Sally, B.K. and McNeal, F.H. 1976. Effect of Pyrenophora wheat leaf blight on the thousand kernel weight of 30 spring wheat cultivars. Plant Dis. Reptr. 60:135-138.

Snedecor, G.W. and Cochran, W.G. 1980. Statistical methods. 7th ed. Iowa State Univ. Press, Ames, Iowa.

Tekauz, A. 1976. Distribution, severity, and relative importance of leaf spot diseases of wheat in western Canada in 1974. Can. Plant Dis. Survey. 56:36-40.

Tekauz, A. 1985. A numerical scale to classify reactions of barley to Pyrenophora teres. Can. J. Plant Pathol. 7:181-183.

Tekauz, A., Platford, R.G. and English, N.C. 1982. Tan spot of wheat. [in: Manitoba Agronomists, Ann. Conf., Tech. and Sci. Papers, Dec. 15-16, pp. 60-65].

Tekauz, A., Samborski, D.J., Rourke, D.S.R. and Iverson, A.T. 1983. Diseases of winter wheat in Manitoba in 1983. [in: Manitoba Agronomists, Ann. Conf., Tech. and Sci. Papers, Dec. 14-15. pp. 63-68].

Tomas, A. and Bockus, W.W. 1987. Cultivar-specific toxicity of culture filtrates of Pyrenophora tritici-repentis. Phytopathology. 77:1337-1340.

Tomas, A., Bockus, W.W., and Leach, J.E. 1986. Involvement of a toxin in the symptomatology of tan spot of wheat. Phytopathology. 76:1125 (Abstr.).

Vanterpool, T.C. 1963. Pink and smudge-pink discoloration of wheat seed associated with the yellow leaf spot disease (Drechslera tritici-repentis). Proc. Can. Phytopathol. Soc. 30:19-20.

Webster, J. 1980. Introduction to fungi. 2nd ed. Cambridge Univ. Press, Cambridge, Eng.

Wiese, M.V. 1977. Compendium of wheat diseases. Am. Phytopathol. Soc.

Wiese, M.V. 1984. Impact of diseases on wheat yields in Idaho's Kootenai valley in 1981. Plant Dis. 68:421-424.

Wong, L.S.L. and Hughes, G.R. 1987. Evaluation of resistance to Leptosphaeria nodorum and Pyrenophora tritici-repentis in wheat. Can. J. Plant Pathol. 9:287 (Abstr.).