EXPRESSION AND GENETICS OF RESISTANCE TO *SEPTORIA TRITICI* IN WHEAT

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ABSTRACT

This study of resistance in wheat (*Triticum aestivum*) to the leaf blotch pathogen *Septoria tritici* (anamorph of *Mycosphaerella graminicola*) was conducted to provide information on the inheritance and heritability of resistance, the occurrence of physiologic specialization in the pathogen, the components of resistance and association of resistance with undesirable agronomic traits.

The inheritance and heritability of resistance to a Saskatchewan isolate of *S. tritici* was studied in seedling tests. Resistance was controlled by a single incompletely dominant gene in cultivars Oasis, French Peace and Frontana, a single partially recessive gene in Bunyip and by two independent genes, one dominant and one recessive, in Lacos III 262 sel. no. 166-82 (Lacos). There was some evidence that genes with minor effects modified the expression of resistance in all crosses. Allelism tests showed that with the exception of the resistance genes in Frontana and Bunyip, genes controlling resistance were at different loci. Heritability of resistance ranged from 38 to 62%.

Physiologic specialization was studied in a controlled environment by inoculating nine cultivars with nine *S. tritici* isolates from Saskatchewan, one from England and one from Indiana. A test for crossover interactions indicated that there were cultivar-specific differences in pathogenicity between isolates from Saskatchewan and those from England and Indiana, but not among Saskatchewan isolates.

Components of resistance were studied on seedlings and adult plants in controlled environment experiments. Significant differences among cultivars were found for all components and components tended to be associated within cultivars. The cultivar Lacos exhibited the longest latent period, lowest infection frequency, lowest spore production and lowest rate of lesion expansion.

To study association of resistance with heading time and height, progeny of crosses between resistant and susceptible parents were studied in the field. The association between resistance and winter habit in Oasis was studied in a growth room experiment.

No association was found between disease reactions in growth room tests and any of the agronomic traits. Resistance rating in the field was moderately associated with late heading and to a lesser extent with tallness in the French Peace and Frontana crosses. However, the relationship was probably not strong enough to prevent selection of lines combining resistance with early maturity and short height.

Simple genetic control of resistance and the association of resistance components within cultivars are factors which should facilitate breeding for resistance. Moderate heritability of resistance and, in some crosses, association of resistance with undesirable traits are factors which indicate that large population sizes will be required to enable effective selection for resistance. The presence of physiologic specialization, although not detected at the regional level, suggests that several different sources of resistance should be used in a breeding program.

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1.0 Introduction

Septoria tritici Rob. ex. Desm. (teleomorph: *Mycosphaerella graminicola* (Fückel) Schroeter) is a fungal pathogen of wheat (*Triticum aestivum* L.). It often occurs in association with other *Septoria* species that are pathogenic on wheat, *Septoria nodorum* Berk. (teleomorph: *Leptosphaeria nodorum* Müller) and *Septoria avenae* f.sp. *triticea* T. Johnson (teleomorph: *Leptosphaeria avenae* f.sp. *triticea* T. Johnson).

The relative prevalence of these pathogens and the damage they cause varies throughout the world. However, *Septoria tritici* and *S. nodorum* are generally considered to be more important than *S. avenae* f.sp. *triticea.* Yield losses in a severe epidemic can reach 50%, but probably average 2% on a world-wide basis (Scharen and Sanderson, 1985). Yield losses in the northern cropping area of Saskatchewan have been estimated to be 15% (unpublished data).

Hard red spring wheat is the most important crop grown in Saskatchewan, occupying approximately 60% of the seeded acreage. Thus, even a small percentage loss due to disease is important economically. Given the low input - low return farming system common in western Canada, genetic and cultural disease control methods are the most feasible.

None of the hard red spring wheat cultivars currently grown in western Canada have any appreciable resistance to *S. tritici.* Therefore this study was initiated to provide information that would facilitate breeding for resistance to *S. tritici.* The specific objectives were:

1. to determine the inheritance and heritability of resistance in selected sources of resistance;
2. to determine if physiologic specialization occurs in the pathogen population;
3. to characterize resistance by quantifying the components of resistance that affect epidemic development; and
4. to determine if resistance is associated with undesirable agronomic traits.

2.0 Literature Review

2.1 Overview of *Septoria tritici*

Early research done on septoria diseases was reviewed by Weber (1922) and, more recently, Shipton et al. (1971) and King et al. (1983a). A review by Eyal et al. (1987) emphasized methods used in the research and control of septoria diseases.

2.1.1 Taxonomy and morphology

Within the Deuteromycetes (Fungi Imperfecti), *Septoria* fungi are classified in the order Sphaeropsidales, which is characterized by conidia in the form of pycnidiospores, contained within fruiting structures called pycnidia (Scharen and Sanderson, 1985).

Sanderson (1972) found the sexual state of *S. tritici* to be *Mycosphaerella graminicola*. *M. graminicola* is an ascomycetous fungus within the subclass Loculoascomycetes, order Dothideales, family Dothideaceae (Scharen and Sanderson, 1985). Müller (1989) classifies *Mycosphaerella* in the family Mycosphaerellaceae.

Septoria nodorum and *S. avenae* have been placed in the genus *Stagonospora*, and their teleomorph states have been classified in the genus *Phaeosphaeria*, family Phaeosphaeriaceae, order Pleosparales (Müller, 1989). At the second International *Septoria* Workshop it was decided to continue the use of the *Leptosphaeria* classification and to refer to the diseases caused by their *Septoria* anamorph names; septoria tritici blotch, septoria nodorum blotch and septoria avenae blotch (Scharen and Sanderson, 1985). Septoria tritici blotch has been known also as "speckled leaf blotch" and septoria nodorum blotch as "glume blotch" (Weber, 1922).

Table 2.1 shows the dimensions of the fruiting structures and spores of the three major *Septoria* species, as reported by Scharen and Sanderson (1985). In both the sexual and asexual states, the spores of *S. tritici* are quite distinct from those of the other *Septoria* species.

Table 2.1. Dimensions of *Septoria* spores and their fruiting bodies

Asexual state	Pycnidium (µm)	Pycnidiospore (µm)	Septa
S. tritici	60 - 200	35 - 98 x 1 - 3	3 - 5
S. nodorum	160 - 210	15 - 32 x 2 - 4	0 - 3
S. avenae	90 - 140	26 - 42 x 3 - 4	3 - 4
Sexual state	**Pseudothecium (µm)**	**Ascospore (µm)**	**Septa**
M. graminicola	70 - 100	10 - 15 x 2 - 3	1
L. nodorum	120 - 200	23 - 32 x 4 - 6	3
L. avenae	100 - 220	19 - 25 x 4 - 6	3

It has been reported that *S. tritici* also produces micro-pycnidiospores (9 x 0.9 um) in micro-pycnidia (Harrower, 1976, cited in King et al., 1983a) and in normal pycnidia (Rodeva, 1989). Another type of micro-conidium (1.5 - 4.0 x 8.8 - 26.5 um) is produced in culture (Weber, 1922; Hilu and Bever, 1957) and on leaves (Djerbi et al., 1972) by the budding of pycnidiospores. In culture this budding process gives rise to yeast-like colonies.

2.1.2 Host range

Septoria tritici is considered to be a more specialized pathogen than *S. nodorum* (Williams and Jones, 1973). Beach (1919) found that *S. tritici* isolated from *T. aestivum* would not sporulate on other *Triticum* spp., rye (*Secale cereale*), or *Poa pratensis*. However, Weber (1922) reported that *S. tritici* isolated from wheat, rye or *P. pratensis* was able to attack a number of *Triticum* species, including *T. aestivum*, *T. durum*, *T. compactum*, *T. dicoccum*, *T. monococcum*, *T. polonicum*, *T. spelta* and *T. turgidum*, as well as rye and *P. pratensis*. No barley (*Hordeum vulgare*) variety was infected.

Hilu and Bever (1957) inoculated 11 grass species with *S. tritici*, but only obtained

infection on *T. aestivum*, *T. durum* and *T. monococcum*. Brokenshire (1975a) observed pycnidial development on eight of 12 grass species, including some barley cultivars.

Isolates from one species may be less pathogenic on another species. Williams and Jones (1973) found that five grass species inoculated with *S. tritici* from wheat did not show any symptoms before leaves began to senesce, but pycnidia formed after senescing leaves were placed in a mist chamber. Isolates collected from 11 grass species inoculated with an isolate from wheat seemed less pathogenic on wheat than the isolate obtained from wheat (Ao and Griffiths, 1976).

2.1.3 Epidemiology

2.1.3.1 Disease cycle

Infected crop residue is an important source of primary inoculum (Weber, 1922; Hilu and Bever, 1957; Wenham, 1959; Brokenshire, 1975b). Between wheat crops, pycnidiospores survive in pycnidia during winter (Weber, 1922) and summer (Hilu and Bever, 1957). Ascospores from pseudothecia on crop debris are an important source of primary inoculum in several countries (Brown et al., 1978; Sanderson and Hampton, 1978; Shaw and Royle, 1989a).

Infection in fields that contain little or no wheat debris is more difficult to explain. Weber (1922) suggested that debris containing pycnidia may be blown to other fields. Holmes and Colhoun (1975) speculated that water droplets containing pycnidiospores might be carried by the wind. Brown et al. (1978) could not detect any wind-blown pycnidiospores or debris, but trapped large numbers of air-borne ascospores. Shaw and Royle (1989a) found evidence that widespread, evenly distributed primary infections in England were caused by air-borne ascospores.

Seed infection is another possible source of inoculum, but although seed infection by *S. tritici* has been found, transmission to seedlings has not been observed (Brokenshire, 1975c).

Alternative hosts have been suggested as possible sources of primary infections (Williams and Jones, 1973; Brokenshire, 1975a), especially where wheat crops are widely separated in time and in space (Mamluk and Naimi, 1989).

Secondary infection is caused by pycnidiospores spread by rain splash from pycnidia in the original lesions on lower leaves to the upper leaves as they emerge (Eyal, 1981). Thus, the disease progresses upwards in the crop canopy, a phenomenon known as the "ladder effect" (Bahat et al., 1980). The disease can progress quickly to the top leaves if there is a heavy rainfall that is effective in transporting spores vertically (Royle et al., 1986). Since the number of infection cycles within any leaf layer is limited due to the long latent period of *S. tritici*, a severe epidemic will occur only if there is either substantial infection of top leaves during stem extension or early infection of top leaves (Shaw and Royle, 1989b).

2.1.3.2 Environmental effects

Moisture is important in the dissemination and infection process of *S. tritici* (Holmes and Colhoun, 1974; Shaner and Finney, 1976; Hess and Shaner, 1987a,b). Spores are released from pycnidia when the relative humidity (RH) is near 100% (Eyal, 1971) and from pseudothecia when they have been moist for 30 minutes (Brown et al., 1978). Rain was more effective than dew or fog in liberating ascospores (Sanderson and Hampton, 1978). Spores in a cirrhus can remain viable for at least 15 days at 35 to 85% RH and 25°C (Eyal, 1971).

Pycnidiospores are transported vertically and horizontally in the plant canopy by rain splash. Rain with large, widely dispersed drops is most effective for vertical transport of spores (Shaw, 1987). Working with *S. nodorum*, Brennan et al. (1985) found that a wind of 4 m sec^{-1} would carry spore containing droplets at least 2 m downwind.

High RH at the leaf surface is critical for infection. In greenhouse studies, 72 h of high RH following inoculation was required to achieve maximum infection (Hess and

Shaner, 1987a). Hilu and Bever (1957) found that spores on a moist leaf could germinate in 12 hours and penetrate stomata within 24 hours. The length of the moist period required for germination and penetration depends on temperature (Holmes and Colhoun, 1974). Conidia germinated from 2 to 37°C with an optimum of 20 to 25°C (Eyal et al., 1987). Shaner and Finney (1976) found that minimum temperatures of less than 7°C retarded epidemic development. Light intensity also affects disease development. Benedict (1971) found that 8000 to 12,000 lux were optimal for spore germination and mycelial growth, while maximum pycnidial formation occurred at 2000 lux.

The optimal conditions for *S. tritici* infection have been summed up as cloudy and rainy and a temperature between 20 and 25°C (Eyal et al., 1987).

The relationship between epidemics of septoria tritici blotch and weather conditions has prompted efforts to forecast epidemics from weather data. Shaner and Finney (1976) and Coakley et al. (1985) used long term weather data and disease ratings in Indiana to find meteorological variables most closely correlated to disease ratings. Coakley et al. (1985) concluded that the number of days without rain and the number of days where the temperature exceeded 25°C were the best predictors of final disease severity.

A model (EPIPRE) has been designed in the Netherlands to indicate how often farmers should apply fungicides to control septoria diseases (Zadoks, 1989). This model uses the number of rainy days, total precipitation and other variables such as varietal tolerance, disease incidence and the amount of nitrogen fertilizer and growth regulators used.

2.1.4 Infection process and symptom expression

Detailed histological studies of the infection process of *S. tritici* have been carried out by Weber (1922) and Hilu and Bever (1957). Disease symptoms have been described in several reviews (Shipton et al., 1971; Wiese, 1977; Zillinsky, 1983).

Hilu and Bever (1957) observed that pycnidiospores began to germinate about 12

hours after inoculation and penetrated the leaf, usually through stomata, after about 24 hours. On the other hand, Weber (1922) claimed that germ tubes almost always penetrated the cuticle.

Following penetration, the hyphae grew between the cells in all directions, but were limited by the bundle sheaths (Hilu and Bever, 1957). The sclerenchyma cells of the bundle sheath appeared to protect the vascular bundles from attack. In a susceptible cultivar, the hyphae grew extensively for about nine days before there was enough cell death to cause macroscopic symptoms. Pycnidia formed from hyphae that branched and fused extensively under stomata. Very few hyphae were observed within dead or living plant cells. In resistant cultivars, stomatal penetration was as efficient as in the susceptible, but the hyphae did not become established as often. When hyphae did become established, they grew slower and the pycnidia produced were smaller than in susceptible cultivars.

The macroscopic symptoms begin as irregular brown lesions (Zillinsky, 1983), or as light green chlorotic spots (Weber, 1922), that develop longtitudinally, restricted by leaf veins. The centres of the lesions often become ash coloured and dotted with black pycnidia, which tend to occur in rows because they develop in the sub-stomatal cavities. The lesions may eventually coalesce and cover the entire leaf blade (Zillinsky, 1983). Septoria tritici blotch usually occurs on the leaf and culm, but it has also been observed on the head of the plant (Jones and Cooke, 1970).

Weber (1922) noted that, in some cases, areas of the leaf that contained pycnidia remained green longer than other parts of the leaf, creating a "green island" effect. Rosielle (1972) reported that some genotypes exhibited a high degree of necrosis in response to *S. tritici*, but very few pycnidia were produced.

2.1.5 Disease assessment and relationship to yield loss

Methods of assessment of septoria disease severity have been reviewed by Eyal et al.

(1987). Disease ratings are usually based on necrotic leaf area, pycnidial density or a combination of the two. To assess the percent area affected, standard area diagrams have been developed (James, 1971). Shaner et al. (1975) used a quantified five-point scale to estimate pycnidial density, while Eyal and Brown (1976) used a television scanner to quantify pycnidial coverage on leaves.

Diseases such as septoria tritici blotch, that begin at the base of the plant and progress upwards, can be rated according to the height that they reach on the plant. Saari and Prescott (1975) developed a scale that takes into account the height of the disease and the severity at that level. Eyal and Ziv (1974) used a measure of disease height divided by plant height, which they called the "septoria progress coefficient".

Rosielle (1972) developed a six-point scale that combines pycnidial density and the amount of necrosis. This scale, or a variation of it, has been used as a basis for classification of plants as resistant or susceptible prior to Mendelian analysis (Wilson, 1979, 1985; Shaner and Buechley, 1989). Eyal et al. (1985) used cluster analysis of percent necrosis data to classify cultivar-isolate interactions as being of the resistant or susceptible type.

In working with *S. nodorum*, several workers have measured specific components of resistance to assess cultivar reactions (Baker and Smith, 1979; Jeger et al., 1983; Griffiths and Jones, 1987). Brokenshire (1976) used the parameters, incubation period, latent period, disease and sporulation levels and 1000 kernel weight, to characterize reactions to *S. tritici*. Other methods that have been suggested for disease assessment include measuring pycnidiospore production (Gough, 1978) and bioassays for chitin (Harrower, 1977) or ergosterol (Griffiths, 1985).

The relationship between disease and yield loss has been studied by several workers. This is usually done by generating different disease levels through the use of fungicides or artificial inoculations.

Eyal (1972) and King et al. (1983b) used regression analysis to describe the

relationship between percent necrosis and yield loss. When septoria diseases were assessed at Zadoks' growth stage 75 (Zadoks et al., 1974), King et al. (1983b) found that percent yield loss was approximately equal to percent necrosis on the flag leaf or to approximately half the magnitude of percent necrosis on the penultimate leaf. Similarly, Thomas et al. (1989) reported that yield loss could be predicted from disease ratings on any of the top three leaves.

Forrer and Zadoks (1983) found a good relationship ($r^2 = 0.795$) when kernel weight was regressed on area under the disease progress curve. Yield loss was also highly correlated ($r = 0.92 - 0.96$) with cumulative leaf area affected (Eyal and Ziv, 1974).

The relationship between disease severity and yield loss may be cultivar dependent. Ziv and Eyal (1976) suggested that the cultivar Miriam possessed tolerance to septoria tritici blotch because it suffered a relatively small yield loss compared to other cultivars with similar levels of disease.

2.1.6 Control

2.1.6.1 Resistance

Resistance to *S. tritici* is not as common in *T. aestivum* as in some other *Triticum* spp. (Brokenshire, 1976; Krupinsky et al., 1977; Eyal, 1981; Yechilevich-Auster et al., 1983). Resistance in *T. aestivum* has been associated with winter growth habit, tallness and late maturity (Eyal, 1981). The lack of information concerning inheritance, and the possibility that physiologic races of the pathogen exist, are other obstacles to resistance breeding efforts (Eyal 1981).

Despite these problems there has been some success in the last 20 years in incorporating resistance into commercial cultivars in the U.S.A. (Patterson et al., 1975), in the CIMMYT program (van Ginkel and Rajaram, 1989), in Australia (Ballantyne, 1989a) and in England (Griffiths and Jones, 1987).

2.1.6.2 Chemical control

Foliar fungicides to control septoria diseases are used where agronomic and weather conditions are conducive to disease development and where the potential economic value of the crop makes fungicide use economically justifiable (Eyal et al., 1987). In the United Kingdom over 90% of the cereal crops received at least one fungicide spray in 1987, while 64% received two or more sprays (Thomas et al., 1989).

Protectant fungicides such as maneb and mancozeb are effective against septoria tritici blotch, but require repeated applications at 10 to 14 day intervals (Eyal and Wahl, 1975). However, systemic fungicides such as benomyl, prochloraz and propiconazole have some curative properties and longer protective action than the protectants (Eyal et al., 1987). Two early applications of systemic fungicides have been found to be effective in controlling septoria tritici blotch (Carmi et al., 1985; Lockley, 1989).

In western Canada Dithane M-45 (mancozeb) and Tilt 250E (propiconazole) have been registered for control of septoria diseases on some classes of wheat (Anonymous, 1989). Although it has been demonstrated that wheat yields can be increased when septoria diseases are controlled by fungicides, the relatively low yield potential and low grain prices prevalent in Western Canada since the registration of the fungicides have limited their use.

2.1.6.3 Cultural control

The largest reservoir of primary inoculum of *S. tritici* is infected wheat stubble and debris left on the soil surface. Therefore, cultural practices that reduce the amount of infected residue in fields planted to wheat should help to control septoria diseases (Eyal, 1981).

Crop rotation is one method of reducing primary inoculum. A 3 to 5 year rotation decreased the incidence of septoria tritici blotch in Israel, but even a 6 to 8 year interval between wheat crops did not completely eliminate outbreaks (Eyal, 1981). Pedersen and

Hughes (1989) found that excluding wheat for one or two years reduced the number of lesions and disease severity of both *S. tritici* and *S. nodorum* in Saskatchewan. Wheat yields were higher in fields where wheat was not planted into wheat stubble.

Burning stubble and deep ploughing are also methods of reducing the amount of wheat residue on the surface (Wenham, 1959). However, these practices are discouraged because they are causes of soil degradation and erosion.

Other aspects of crop management, such as nitrogen fertilization, row spacing and seeding rate, may have an influence on the severity of septoria diseases through their effects on microclimate and crop architecture (Broscious et al., 1985). Fellows (1962) and Broscious et al. (1985) reported increased severity of septoria diseases with increased nitrogen fertilization. Increased seeding rate was associated with greater septoria severity in Saskatchewan (Tompkins et al., 1989) and in Pennsylvania (Broscious et al., 1985). However, Eyal (1981) stated that less dense canopies have higher disease levels because of enhanced rain splash.

Wolfe and Barrett (1980) suggested using mixtures of cultivars as a disease control method. Jeger et al. (1981) found that a cultivar mixture could be effective in reducing severity of a non-specialized pathogen such as *S. nodorum*, as long as the cultivars differed in their components of resistance. Dinoor and Pnuel (1989) reported that in mixtures of three cultivars, none of which was immune, septoria disease level was reduced to near that of the most resistant cultivar.

2.1.6.4 Biological control

For some diseases, lack of adequate resistance in commercial cultivars and concerns about possible harmful health effects of fungicides have prompted research into using other micro-organisms as bio-control agents. The problems and potential of using biological agents to control pathogens on leaf surfaces has been reviewed by Blakeman and Fokkema (1982).

Fokkema et al. (1979) attempted to enhance the growth of saprophytic yeast mycoflora on leaf surfaces by applying suspensions of yeast with or without nutrients. The severity of septoria nodorum blotch was reduced on plants with added yeasts and nutrients for three weeks after treatment, but eventually the saprophytic population and the septoria severity on the control plants was equal to that on the treated ones.

In a field experiment in Oklahoma, Gough et al. (1986) found that applications of streptomycin resulted in a 51% increase in the number of *S. tritici* lesions. Unsprayed plots were found to have populations of *Bacillus subtilis* and *Pseudomonas fluorescens* that were inhibitory to *S. tritici*. In Israel, Levy et al. (1988) discovered that two fluorescent pseudomonads isolated from soil were inhibitory to *S. tritici* both in vitro and on seedlings. The area covered by pycnidia was reduced by 72 to 92% in seedling tests. Gough and El-Nashaar (1989) found that wheat cultivars differ in their ability to support a bacterial antagonist of *S. tritici*.

2.2 Resistance to *Septoria tritici*

2.2.1 Inheritance of resistance

The first reported study of inheritance was conducted by Mackie (1929). He found that resistance in an unidentified cultivar was due to a single recessive gene. Since that time most workers have reported simple genetic control of resistance, most often a single dominant gene. Narvaez and Caldwell (1957) found that resistance in Nabob was controlled by two independent, partially dominant genes with additive effects. Resistance in Lerma and P14 was governed by a single dominant gene in each. Similarly, Rillo and Caldwell (1966) reported that resistance in Bulgaria 88 was due to a single dominant gene, although the resistance level in heterozygotes declined over time. Resistance from Bulgaria 88 was transferred to 'Arthur' type wheats to create the soft red winter wheat cultivar Oasis (Patterson et al., 1975).

Rosielle and Brown (1979) studied the inheritance of resistance in Veranopolis,

Seabreeze and IAS 20 in the field. There were no clear discontinuities in the F_2 distributions, but classification of F_3 families suggested that resistance in Veranopolis and IAS 20 could be monogenic, while resistance in Seabreeze was controlled by at least three recessive genes. Wilson (1979) confirmed that a single dominant gene controlled resistance in Veranopolis and Bulgaria 88 and also in Israel 493. Segregation for resistance in crosses between resistant cultivars indicated that the gene in Veranopolis is different from those in Bulgaria 88 or Israel 493. Subsequently it was shown that the genes in Bulgaria 88 and Israel 493 are also independent (Wilson, 1985).

Wilson (1985) crossed and backcrossed 28 different sources of resistance to a common susceptible cultivar and evaluated segregation in several generations. In 22 of the crosses a single dominant gene model provided the best fit to the data. The remaining crosses fitted single incomplete dominant, single recessive, two dominant and two complementary gene models. However, for some crosses, not all of the generations studied fitted the same model.

Shaner and Buechley (1989) studied the inheritance of resistance to *S. tritici* in two cultivars, Oasis and Sullivan and one breeding line (72626). All are believed to carry resistance from Bulgaria 88. They also studied breeding lines that carry resistance from South American cultivars Sao Sepe and Sudeste. In lines with resistance derived from Bulgaria 88, resistance was controlled by a single gene, completely dominant in Sullivan and 72626 and partially dominant in Oasis. In lines with resistance derived from Sao Sepe, resistance was controlled also by a single dominant gene. The resistance from Sudeste may be due to a single gene, but occasional susceptible reactions recorded on the breeding line carrying the resistance made interpretation difficult.

The gene-for-gene model has been used to identify hypothetical resistance genes in experiments where a number of cultivars have been inoculated with a series of isolates of *S. tritici* (Yechilevich-Auster et al., 1983; Eyal et al., 1985; van Ginkel and Scharen, 1988). In these experiments, cut-off points are established in order to classify each

cultivar-isolate combination as being either resistant or susceptible. Unique combinations of resistance/susceptibility exhibited by cultivars in response to the isolates are attributed to hypothetical resistance genes. Eyal et al. (1985) identified 28 different hypothetical genes in 35 wheat and triticale (X *Triticosecale*) cultivars inoculated with 97 isolates of *S. tritici*.

Quantitative analysis methods have been used by some workers. Ziv et al. (1981) selected for tolerance to *S. tritici* by selecting for the maintenance of thousand kernel weight under disease pressure. From the rapid response achieved, they speculated that tolerance was controlled by a few additive loci. Danon et al. (1982) estimated the number of genes controlling resistance in 34 crosses, using Burton's (1951) formula, to be 0.1 to 4.3 with a mean of 2.0. Shaner and Finney (1982) reported that some breeding lines had partial resistance to septoria tritici blotch, although the parents had no obvious resistance. They suggested that this resistance was due to additive or complementary gene action.

Van Ginkel and Scharen (1987) used generation means analysis to study the genetics of resistance in 13 durum wheat cultivars. Twenty-nine of 65 crosses studied showed no significant genetic effects. Approximately 50% of the crosses had significant additive effects, while one-third had significant dominance effects. Epistasis was not common. Estimates of gene number ranged from 0 to 3,029 genes. Thirty-two of the 63 crosses had gene number estimates of more than five, while only eight of the crosses produced estimates of two or less.

2.2.2 Heritability of resistance

Rosielle and Brown (1979) used correlation between severity of septoria tritici blotch on F_2 plants and mean severity on their F_3 progeny to estimate "standard unit heritability". In crosses with the susceptible cultivar Gamenya, heritability was estimated to be 68% for Seabreeze, 64% for Veranopolis and 57% for IAS 20.

Ziv et al. (1981) used high thousand kernel weight (MKW) under disease pressure as

an indication of tolerance to *S. tritici* and selected for high MKW in F_3 and F_4 populations from crosses involving the tolerant cultivar Miriam. Realized heritability values (Falconer, 1981) ranged from 50 to 74%. However, selection in populations protected with fungicide was just as effective in raising mean MKW, suggesting that the heritability measured related to MKW, not tolerance. Estimates of heritability in 65 crosses among 13 durum cultivars that varied in resistance to *S. tritici*, ranged from 0 to 78%, with a mean of 38% (van Ginkel and Sharen, 1987).

2.2.3 Components of resistance

Parlevliet (1979) defined incomplete resistance as all resistance that allows some spore production by the pathogen. Incomplete resistance gives an epidemic rate (k) that is lower than that of a susceptible cultivar. If resistance is complete or nearly complete, the k value is zero. Contrary to Vanderplank's views (1963; 1968), Parlevliet (1979) points out that all resistances are rate-reducing whether they are race-specific or non race-specific. Inheritance of incomplete resistance is often polygenic (Nelson, 1978), but monogenic and oligogenic control is also common (Parlevliet, 1979).

Parlevliet (1979) identified four resistance components that can reduce the rate of an epidemic. Infection frequency is the proportion of spores that result in sporulating lesions, latent period is the time from infection to spore production, spore production is the number of spores produced per lesion or per unit time and infectious period is the time period over which the diseased tissue supports sporulation.

The components of resistance to *S. tritici* have not been specifically studied, although Brokenshire (1976) estimated incubation period, latent period and pycnidial coverage on seedling and adult plants. Hess and Shaner (1987a) noted that the resistant cultivar Auburn had fewer, smaller lesions and smaller pycnidia than susceptible cultivars.

More detailed studies have been conducted on the components of resistance to *S. nodorum*. Jeger et al. (1983) measured 11 components on 41 randomly chosen wheat

genotypes to determine whether or not the components were associated. No clear evidence of association among the components was found. Lancashire and Jones (1985) studied components of resistance on ten cultivars that had similar field ratings for septoria nodorum blotch. They found significant differences between cultivars for some components, and used cluster analysis to group cultivars on the basis of their components of resistance.

Factor analysis (Jeger et al., 1983) and principal components analysis (Lancashire and Jones, 1985) both identified four underlying factors that contributed to the variation in the components measured. This was interpreted as suggesting that four genes may be involved (Jeger et al., 1983).

Shaner and Hess (1978) pointed out that the effects of a component on an epidemic in the field are difficult to determine, because the components interact and their effects are cumulative. To relate components of resistance to field results, equations that integrate the components can be used (Shaner and Hess, 1978; Lancashire and Jones, 1985), or a simulation model can be developed (Rapilly et al., 1977).

Lancashire and Jones (1985) combined the components of resistance in a mathematical model that produced a resistance index number for each cultivar. Rapilly et al. (1977) used a simulation model to investigate the effects of incubation period, latent period and rate of lesion expansion on a septoria nodorum blotch epidemic. The rate of lesion expansion was predicted to be the most important parameter in determining the extent of an epidemic.

A simple regression model was used to determine those components of resistance that could be used to predict field resistance (Griffiths and Jones, 1987). Sporulation and incubation period were found to be the best predictors of field resistance.

2.2.4 Pathogenic variation

Specific resistance refers to resistance that is effective against some, but not all,

isolates of a pathogen. Physiologic specialization is the pathogenic counterpart to specific resistance.

Flor (1955) developed the gene-for-gene hypothesis to explain specificity of reactions in the flax (*Linum usitatissimum*)-flax rust (*Melampsora lini*) system. Vanderplank (1968) differentiated between "vertical resistance", which is effective against some pathogen genotypes, and "horizontal resistance", which is equally effective against all pathogen genotypes. He suggested that if a number of cultivars were evaluated with a series of pathogen isolates, specific resistance could be detected as a significant cultivar x isolate interaction in an analysis of variance. Parlevliet and Zadoks (1977) demonstrated that if resistance and pathogenicity are determined by a large number of genes with small but specific effects, the interaction term in the analysis of variance may not be significant. Vanderplank (1988) later suggested using non-parametric ranking tests to analyze interactions, since parametric analyses are sensitive to the way in which disease is assessed.

Jenns et al. (1982) proposed a method of analyzing host-pathogen interactions that is similar to the stability analysis of Eberhart and Russell (1966). They regressed the disease severity on a cultivar for each isolate against the average disease severity caused by those isolates. The slope of the regression line was considered to indicate the degree of sensitivity to general pathogenicity (aggressiveness), while deviations from the regression line indicated specific reactions. Subsequently, Jenns and Leonard (1985) suggested a simpler analysis in which cultivars are inoculated with a series of isolates and the magnitude of the variance of disease severity for each cultivar is considered to be a measure of the specificity of the resistance of the cultivar.

There have been conflicting reports regarding pathogenic specialization in *S. tritici* (Eyal et al., 1987). Eyal et al. (1973) were the first to report physiologic specialization in *S. tritici*. When durum and bread wheat cultivars were inoculated with isolates obtained from both types of wheat, differential reactions were found. The main differences

between the isolates were in their reactions on the different wheat species. Prestes (1976) found that some genotypes that appeared resistant when tested in the greenhouse, were susceptible in the field. He attributed this to the existence of different pathogenic types in the State of Washington.

Many workers have inoculated a series of cultivars with a series of isolates and used analysis of variance to determine whether or not significant cultivar x isolate interactions exist (Yechilevich-Auster et al., 1983; Eyal et al., 1985; Eyal and Levy, 1987; Saadaoui, 1987; van Ginkel and Scharen, 1988; van Silfhout et al., 1989; Perello et al., 1989). Except for van Ginkel and Scharen (1988), these workers have reported significant cultivar x isolate interactions, although the interaction is small relative to the main effects of cultivars and isolates.

In several studies, the most important source of interaction was the differential response of wheat species to isolates collected from other wheat species (Eyal et al., 1973; Yechilevich-Auster et al., 1983; Eyal and Levy, 1987; Saadaoui, 1987; van Silfhout et al., 1989). Van Ginkel and Scharen (1988) suggested that the lack of interaction in their study may have been due to the exclusive use of durum wheat cultivars.

Area of origin has also been reported to be an important source of variation among isolates on both a world-wide (Eyal et al., 1985; van Silfhout et al., 1989) and a regional basis (Eyal and Levy, 1987).

Person's (1959) reaction matrix has been used to assign hypothetical resistance genes to cultivars and hypothetical virulence genes to isolates (Yechilevich-Auster et al., 1983; Eyal et al., 1985; Eyal and Levy, 1987; van Ginkel and Scharen, 1988). For this type of analysis reactions are classified as being either resistant or susceptible. The cut-off point used to classify the reactions is established using cluster analysis (Eyal et al., 1985).

Ranking of cultivars according to their reactions to a series of isolates has also been used to determine if physiologic specialization exists. In Argentina, Perello et al. (1989) concluded that true physiologic races of *S. tritici* did not exist, while in Australia,

Ballantyne (1989b) reported reverse ranking of cultivars indicating true physiologic specialization.

Zelikovitch et al. (1986) have reported that isolates not only differ in their virulence, but that an avirulent isolate can inhibit a virulent isolate if they are applied on a cultivar at the same time.

2.2.5 Association of resistance with agronomic traits

Increase in septoria diseases coincided with the introduction of short, early maturing cultivars in many countries (Eyal, 1981). A positive correlation was observed also between resistance and winter growth habit (Eyal, 1981). The possible association of resistance with tallness, late maturity and winter growth habit could create problems for breeders interested in incorporating resistance into short, early maturing, spring wheat cultivars. Eyal (1981) and Tavella (1978) found high correlations between tallness, late maturity and resistance in cultivar trials in Israel and Uruguay, respectively.

Shaner et al. (1975) observed a type of field resistance that reduced the rate of disease development without greatly reducing the pycnidial density. Within crosses where this type of resistance was evident, correlations of -0.35 to -0.77 were found between heading date and septoria tritici blotch severity. Resistance derived from Bulgaria 88 did not appear to be related to late maturity, although Rosielle and Brown (1979) and Danon et al. (1982) have reported difficulty in incorporating this resistance into early maturing spring types.

Rosielle and Brown (1979) found significant negative genetic correlations between septoria tritici blotch severity and heading date and height in three crosses. However, they felt that these correlations were not high enough to prevent selection of desirable types.

Danon et al. (1982) studied the relationship between resistance, heading time and height in F_1 and F_2 generations of crosses between resistant and susceptible parents. The correlations between disease severity and height were generally low and non-significant,

while the negative correlations between septoria tritici blotch and heading time were usually greater. Resistance was controlled by a few genes in these crosses. The authors concluded that linkage caused the significant correlations. They considered that a few genes acting with pleiotropic effects would have resulted in higher correlations.

Scott et al. (1982), believed that the association between tallness and resistance to *S. nodorum* could be due to chance, linkage or pleiotropy. They found that in F_3 lines, tallness and resistance tended to segregate together, whereas late heading and resistance were less closely associated. There was substantial variation in resistance that was not associated with either trait. Assuming that resistance was controlled by many genes, they considered pleiotropy to be the most likely cause of the association between resistance and height. In a subsequent experiment, Scott et al. (1985) found that tall cultivars tended to have a micro-climate that was less favourable to septoria nodorum blotch development than did short cultivars.

Scott and Benedikz (1985) reported that the major dwarfing gene *Rht-2* was not associated with resistance to septoria nodorum blotch, but minor genes affecting height were. The authors hypothesized that genes which gave the plant greater vigour had positive effects on both height and resistance.

3.0 Inheritance and heritability

3.1 Materials and methods

3.1.1 Plant material

Five sources of resistance and two susceptible cultivars were used in this study (Table 3.1).

Table 3.1. Pedigrees of wheat cultivars used in studying resistance to *Septoria tritici*

Cultivar	Pedigree	Septoria tritici blotch rating
Oasis[a]	Arthur 71/5/Arthur*3/3/Purdue 6028// Riley*2/Riley 67*2/4/Arthur*2/3/ Riley 67*2//Riley/Bulgaria 88	resistant
Lacos III 262 sel.no. 166-82[b]	Pavon/Manella	resistant
French Peace[c]	unknown	resistant
Frontana[d]	Fronteira/Mentana	resistant
Bunyip[d]	Rymer/Maffra	moderately resistant
Park[d]	Mida/Cadet//Thatcher	susceptible
Conway[e]	Neepawa/Opal//Siete Cerros/Chris	susceptible

[a]Patterson et al. (1975).
[b]A.L. Scharen, pers. comm.
[c]Knott, (1983).
[d]Zeven and Zeven-Hissink, (1976).
[e]G.R. Hughes, pers. comm.

Oasis is a soft red winter wheat bred specifically for resistance to *S. tritici* (Patterson et al., 1975). Its resistance is derived from Bulgaria 88. Frontana is an old cultivar from Brazil and has been used as a source of leaf rust (*Puccinia recondita*) resistance. Several lines derived from Frontana have been reported to possess resistance to *S. tritici* (Narvaez and Caldwell, 1957; Rosielle and Brown, 1979; Danon and Eyal, 1989). Lacos III 262 selection number 166-82 (Lacos) is a semi-dwarf line from Chile that was obtained from the screening nursery of Dr. A.L. Scharen at Montana State University. French Peace and

Bunyip are old cultivars that were obtained from the wheat collection of the Crop Science and Plant Ecology Department, University of Saskatchewan. The susceptible cultivar Park is a hard red spring wheat registered in Canada in 1963. It is early maturing, and is still grown on a limited acreage in western Canada. Conway is a hard red spring wheat registered in Canada in 1986.

All experiments on inheritance and heritability were conducted in a walk-in growth room with a 16 h photoperiod and 22°C day:16°C night temperatures. Plants were grown individually in 6 cm diameter pots containing a 1:1:1 mixture of soil, peat moss and vermiculite. The pots were placed in metal trays and watered from the bottom, with 20:20:20 fertilizer added as required. To achieve even emergence, seeds were germinated in petri dishes before planting in soil.

Reciprocal crosses were made between resistant and susceptible cultivars. Some F1 plants were backcrossed to both parents, while others were selfed to produce F2 seed. After being rated for disease reaction, some F2 plants were randomly selected and selfed to produce F3 families.

Crosses were also made between resistant cultivars and F2 populations produced and tested for disease resistance. Susceptible F2 plants were selfed to produce F3 families, which were tested to verify the F2 ratings.

For crosses between resistant and susceptible cultivars, data were collected in three series of experiments. Preliminary experiments included parental, F2 and a small number of F1 and backcross plants. F3 families were produced from the F2 individuals in these tests. A larger experiment with parental, F1, F2 and backcross generations was conducted also for each cross. The third type of experiment consisted of inoculation tests of F3 families with parental checks included.

A completely randomized design was used in all experiments. In experiments with F3 families, randomization was both within and among families.

3.1.2 Pathogen culture and inoculation

Single-pycnidial isolates of *S. tritici* were obtained from infected leaves and grown on an agar medium containing 4 g of yeast extract, 4 g of malt extract, 4 g of dextrose and 15 g agar per litre of distilled water. Spores placed on this medium multiplied to form yeast-like colonies. After several days, colonies were transferred to 150 mL bottles containing 100 mL of a liquid medium containing 2 g malt extract and 0.5 g yeast extract per litre of distilled water. Liquid cultures were shaken by hand twice daily. Cultures were maintained by pouring approximately 10 mL of liquid culture into a test tube containing sterile soil and storing these test tubes in a refrigerator.

To produce inoculum, a small amount of saturated soil was removed from a test tube and placed in each bottle of liquid medium. Cultures were maintained under ordinary room conditions and shaken twice daily for seven days. Spore concentration was estimated using a haemocytometer and adjusted if necessary to give a concentration of approximately 1.0×10^6 spores per mL.

A single isolate (Sask2) was used in the inheritance and heritability studies. It was isolated from cv. Katepwa, grown at Tarnapol, Saskatchewan in 1985.

Inoculation was carried out when the plants had three completely unfolded leaves. The plants were placed in a mist chamber and sprayed with the liquid culture spore suspension. Approximately 1200 mL of inoculum was used to inoculate 450 plants. The inoculated plants were placed in a mist chamber at 100% RH and after three days were returned to the growth room bench. The third leaf of each plant was rated for septoria tritici blotch reaction 17 days after inoculation using a 0-5 scale similar to that described by Rosielle (1972). This scale varied somewhat according to the severity of disease in an experiment, but it was generally as illustrated in Figure 3.1. Percent leaf necrosis was also estimated.

3.1.3 Statistical analyses

Transformation was required to achieve homogeneity of variances when analyzing

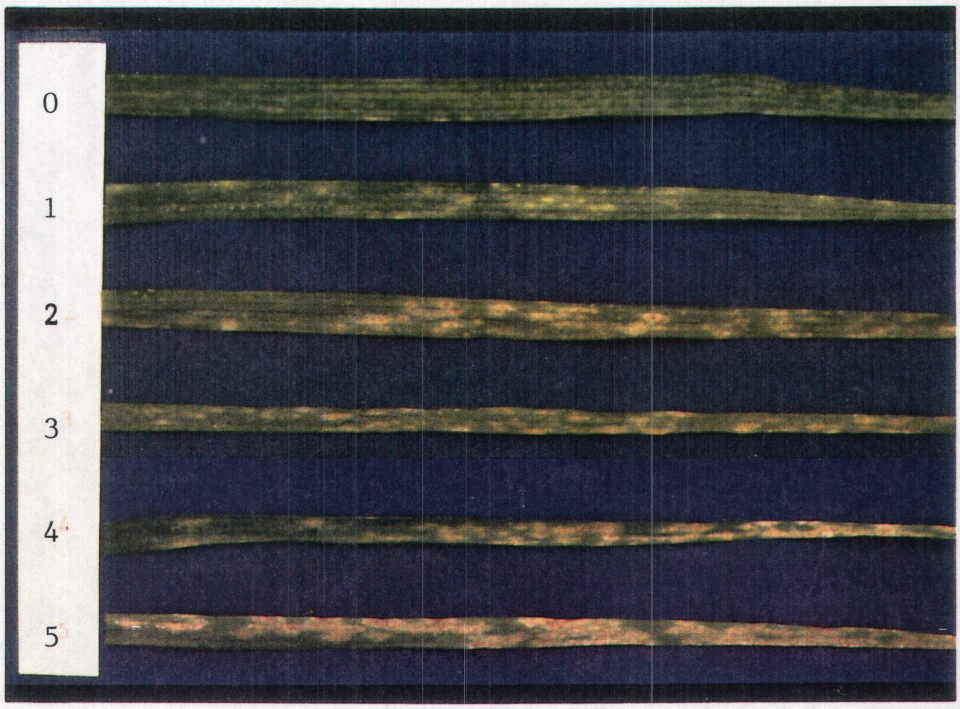

Figure 3.1. Scale used in rating severity of septoria tritici blotch. Rating was based on the number and size of lesions and the density of pycnidia within the lesions

percent leaf necrosis, but not when using the 0-5 scale. Since the 0-5 scale takes both pycnidial density and the amount of necrosis into account, it was considered a better indicator of relative resistance than percent necrosis. This scale was used for all analyses and was treated as a continuous scale.

For Mendelian analysis of the data, the scale was divided into resistant and susceptible categories, the division being based on parental reactions. Before rating each experiment, the parental checks were observed to assess the variation in disease within the parental classes. The categories of the rating scale were then adjusted so that the susceptible parent would not receive a rating of less than '3'. Plants were then classed as resistant if they rated 0-2, and susceptible if they rated 3-5. Yates correction for continuity was used when there was only one degree of freedom in the chi-square test (Steel and Torrie, 1980).

A problem arose in some crosses where the F1 distribution overlapped that of both of the parents. Since the resistant parents varied from 0-2, and the susceptible parents varied from 3-5, there was no way of identifying an intermediate class. The result was that the F1 generation of some crosses had some plants rated as resistant and some as susceptible. The apparent "segregation" of the F1 generation was caused by the inability of the rating scale to consistently distinguish heterozygotes from either of the parents. This made traditional Mendelian analysis of segregating generations difficult, because the expected ratios were unclear.

To test whether or not the data were compatible with a single gene hypothesis in crosses where Mendelian analysis could not be used, a procedure developed by Elston (1966) was used. This procedure has been used to test for one-locus control of resistance to common root rot caused by *Cochliobolus sativus* (Cohen et al., 1969). The Elston procedure uses a minimum chi-square method to estimate the probability of a genotype responding to a stimulus, in this case, the probability of being rated as susceptible. Table 3.2 shows the hypothetical expected genotypic constitution and the probability of a

susceptible response for the seven generations studied.

Table 3.2. Genotypic constitution and probability of a susceptible response for a one-locus hypothesis[a]

Class	Genotypic constitution	P(S)[b]	Number rated	Observed S[c]
P1	AA	α_0	n_0	x_0
BC1	1/2(AA+AB)	$1/2(\alpha_0+\alpha_1)$	n_1	x_1
F1	AB	α_1	n_2	x_2
F2	1/4(AA+2AB+BB)	$1/4(\alpha_0+2\alpha_1+\alpha_2)$	n_3	x_3
F3	1/8(3AA+2AB+3BB)	$1/8(3\alpha_0+2\alpha_1+3\alpha_2)$	n_4	x_4
BC2	1/2(AB+BB)	$1/2(\alpha_1+\alpha_2)$	n_5	x_5
P2	BB	α_2	n_6	x_6

[a]Adapted from Elston (1966).
[b]Probability of being rated as susceptible.
[c]Observed number rated as susceptible.

In many cases, α_0 is assumed to be equal to 0 and α_2 is assumed to be equal to 1. The minimum chi-square estimation of α_1 can be found by solving Eq. (3.1),

$$2(2m_1+m_3)\alpha_0 + 4(m_1+4m_2+m_3+.25m_4+m_5)\alpha_1 + 2(m_3+.75m_4+2m_5)\alpha_2 = 8(z_1+2z_2+z_3+.5z_4+z_5) \quad (3.1)$$

where:

$$z_i = n_i^2/(n_i - x_i) \quad \text{and} \quad (3.2)$$

$$m_i = n_i z_i / x_i \quad (3.3)$$

In the cross Bunyip x Park, α_0 cannot be assumed to equal 0 since three Bunyip plants were rated as susceptible and so Eq. (3.4) must be solved simultaneously with Eq. (3.1).

$$(16m_0+4m_1+m_3+2.25m_4)\alpha_0 + 2(2m_1+m_3+.75m_4)\alpha_1 + (m_3+2.25m_4)\alpha_2 = 4(4z_0+2z_1+z_3+1.5z_4) \quad (3.4)$$

To use information from the F3 generation, equations (7) and (6) of Elston (1966) have been modified to form Equations (3.1) and (3.4), respectively. Once the α values

have been found, the P(S) for any generation is a simple function of the genotypic constitution of that generation (Table 3.2). For every parameter (α_i) that was estimated from the data, one degree of freedom was lost. Therefore, chi-square tests are not carried out on each generation, but the chi-square values are summed and a total chi-square test is carried out. In this test the degrees of freedom are equal to the number of generations in the test minus the number of parameters estimated.

The Kolmogorov-Smirnov two-sample test was used to test for major gene effects (Mode and Gasser, 1972). A random sample was drawn from the parental and F1 generations to create an expected frequency distribution for the F2 generation. The sample size was set as the number of individuals in the class with the least number. The expected F2 frequency was compared to the observed, and the maximum difference was compared to a critical value. The critical value was calculated according to Steel and Torrie (1980). The Kolmogorov-Smirnov method has the advantage of not requiring the division of the F2 range into discrete categories, and thus it may be useful in confirming or rejecting a single gene hypothesis suggested by another model (Mulitze, 1983).

Tests using F3 family segregation data were carried out also. Approximately 50 families from a cross were tested at one time, usually with eight individuals per family. To identify the segregating families derived from heterozygous F2 plants, an adequate number of individuals must be tested in each F3 family. If a single dominant gene is involved, then 11 individuals per F3 family must be tested to reduce the probability of failure in identifying a segregating F3 family to less than 0.05 (Hanson, 1957).

The binomial distribution can be used to find the probability of any segregation ratio within a F3 family. For example, the probability of all eight progeny of a heterozygous individual being resistant when resistance is controlled by a single dominant gene is $(0.75)^8 = 0.10$. In other words, there is a 1/10 chance that the progeny of a heterozygote will be misclassified as being homozygous resistant if eight individuals are tested. Since the probability of misclassification is known, the expected number of families classed as

homozygous resistant, segregating and homozygous susceptible can be adjusted. This adjustment was made before chi-square analysis was carried out on F3 segregation ratios.

To determine if quantitative analysis would give the same results as the chi-square tests, estimates of gene number were made. The basic formula used was developed by Castle (1921) and Wright (1934). Mulitze (1983) stated that if the following are assumed:

1. there are no allelic interactions,
2. there are equal additive effects at all k loci,
3. one parent (P2) is homozygous for all plus alleles at k loci, and the other parent is homozygous for all minus alleles at k loci,
4. there is no linkage,
5. no selection, and
6. normal diploid meiosis,

then $k_{cw} = D^2/(8(VF2 - Ve))$ (3.5)

or $k_{cw} = D^2/[4/((2^{n-1}-1)/(2^{n-1}))(VFn - Ve)]$ (3.6)

where k_{cw} is the estimated number of genes, D is the genetic range, n is the filial generation, V is the variance and e is environment.

The Castle-Wright formula estimates the number of genes by squaring an estimate of the genotypic range (usually estimated from parental means) and then dividing by a constant x the estimated genetic variance. For any given genotypic range, the estimated number of genes will depend on the genetic variance, with a small number of genes producing a larger genetic variance than a large number of genes.

The formula suggested by Burton (1951) differs from the Castle-Wright formula in that it takes dominance into account, with the assumption of equal dominance at all loci. In the F2 generation the formula is:

$$k_b = 0.25D^2(0.75 - h + h^2)/(VF2 - Ve) \quad (3.7)$$

where k_b is the number of loci, h = (F1 - P1)/(P2 - P1) and P2 is the parent with the largest mean.

To obtain a gene estimate in the F3 generation using the Burton formula, Eq. (3.7) was modified to:

$$k_b = (3.75 - 3h + 3h^2)D^2/(16(VF3 - Ve)) \quad (3.8)$$

Variation in disease rating within parental and F1 classes indicates that environment was influencing disease expression. Therefore heritability was calculated to estimate the proportion of the total phenotypic variation due to genetic causes and to provide an indication of how effective selection would be in the early generations.

Regression of F3 family means on F2 individual values was used to estimate heritability. According to Cahaner and Hillel (1980), the slope of the regression line is:

$$b = CovF3F2/VF2 \quad (3.9)$$

which is equal to

$$(Va + 1/2Vd)/(Va + Vd + Ve) \quad (3.10)$$

where Va, Vd and Ve are the variances due to additive, dominance and environmental effects, respectively, in the F2 generation. Since the numerator contains a portion of the variance due to dominance, this estimate of heritability is between a narrow and a broad-sense estimate.

Broad-sense heritability was estimated by dividing the genetic variance by the phenotypic variance. The total F2 generation variance was used as the estimate of phenotypic variance. Genetic variance was estimated by subtracting the mean variance of the parental and F1 generations from the F2 variance.

3.2 Results and discussion

Bartlett's test for homogeneity conducted on parental and F1 generations indicated that variances were homogeneous on the 0-5 scale for all crosses.

Analysis of variance showed no reciprocal differences in the F1 and F3 generations of the five crosses (Table A.1). This indicated that cytoplasmic genes were not involved in determining disease reaction and that accidental selfing during crossing was not a serious

problem.

3.2.1 Frequency distributions

The relative frequency distribution of plant disease ratings is given in Tables 3.3 to 3.7 for the crosses Oasis x Park, Lacos x Conway, French Peace x Park, Frontana x Park and Bunyip x Park, respectively.

With the exception of Bunyip, all the resistant parents were rated in the range 0-2, and the susceptible parents were rated in the range 3-5 in all experiments. The resistance of Bunyip is moderate compared to that of the other resistant parents used, consequently the difference between Bunyip and the susceptible parent was not always clear and three Bunyip plants were rated susceptible.

The F1 distributions were intermediate and skewed towards susceptibility in all crosses except Bunyip x Park where resistance was partially recessive. Except for the Oasis x Park cross, the F1 distributions overlapped those of the parents. In the Oasis x Park cross the average rating of F1 plants was higher than that of Oasis, but no plant was rated as susceptible (Table 3.3). There was minimal overlap in the Lacos x Conway cross (Table 3.4). This overlap was due to the inability to distinguish consistently the heterozygote phenotype from either of the parental phenotypes.

An example of a F1 reaction that is intermediate to those of the the parents is shown in Figure 3.2. Lee and Gough (1984) and Rillo and Caldwell (1966) have also noted intermediate reactions in F1 plants, with the level of resistance declining with time after inoculation.

Backcrosses to the resistant parents (BC1) produced distributions that were intermediate between those of the resistant parents and of the F1 generations. The BC1 distribution overlapped the susceptible parent distribution in all crosses except Lacos x Conway (Table 3.4). This overlap might be expected since there was an overlap in the F1 generation and a proportion of BC1 plants will have the same genotype with respect to

Table 3.3. Relative frequency distribution of plant ratings of septoria tritici blotch resistance in the cross Oasis x Park tested at the three-leaf stage under controlled environmental conditions

	Disease rating class						
Generation	0	1	2	3	4	5	Plants rated
Oasis	.28	.52	.20				50
BC[a](Oasis) F1	.10	.62	.25	.03	.01		73
F1		.39	.61				28
F2	.05	.41	.30	.20	.03	.01	269
F3	.06	.26	.28	.32	.07	.02	399
BC(Park) F1		.20	.31	.27	.15	.07	95
Park				.11	.53	.36	47

[a]Backcross to the parent in parentheses.

Table 3.4. Relative frequency distribution of plant ratings of septoria tritici blotch resistance in the cross Lacos x Conway tested at the three-leaf stage under controlled environmental conditions

	Disease rating class						
Generation	0	1	2	3	4	5	Plants rated
Lacos	.55	.42	.03				65
BC[a](Lacos) F1	.18	.66	.16				73
F1	.04	.42	.49	.04			45
F2	.10	.44	.26	.09	.08	.03	284
F3	.16	.42	.20	.12	.07	.03	399
BC(Conway) F1		.13	.34	.13	.25	.16	77
Conway				.05	.39	.56	64

[a]Backcross to the parent in parentheses.

Table 3.5. Relative frequency distribution of plant ratings of septoria tritici blotch resistance in the cross French Peace x Park tested at the three-leaf stage under controlled environmental conditions

	Disease rating class						
Generation	0	1	2	3	4	5	Plants rated
French Peace	.38	.52	.10				69
BC[a](Fr.P.) F1	.31	.54	.12	.02			90
F1	.12	.42	.39	.07			59
F2	.07	.39	.26	.13	.10	.05	348
F3	.04	.28	.31	.24	.09	.05	407
BC(Park) F2	.01	.10	.22	.29	.30	.09	237
Park				.10	.41	.49	70

[a]Backcross to the parent in parentheses.

Table 3.6. Relative frequency distribution of plant ratings of septoria tritici blotch resistance in the cross Frontana x Park tested at the three-leaf stage under controlled environmental conditions

	Disease rating class						
Generation	0	1	2	3	4	5	Plants rated
Frontana	.30	.59	.11				37
BC[a](Front.) F1	.21	.43	.27	.05	.04		81
F1	.04	.06	.58	.31			48
F2	.05	.30	.34	.14	.12	.04	291
F3	.01	.28	.30	.16	.15	.11	407
BC(Park) F1		.16	.18	.23	.25	.19	80
Park				.14	.52	.34	29

[a]Backcross to the parent in parentheses.

Table 3.7. Relative frequency distribution of plant ratings of septoria tritici blotch resistance in the cross Bunyip x Park tested at the three-leaf stage under controlled environmental conditions

	Disease rating class						
Generation	0	1	2	3	4	5	Plants rated
Bunyip	.08	.35	.52	.06			52
BC[a](Bny.) F1	.08	.34	.28	.30			53
F1		.01	.25	.50	.21	.03	68
F2	<.01	.12	.25	.31	.22	.10	424
F3	.01	.11	.28	.27	.24	.09	788
BC(Park) F1		.02	.08	.41	.37	.13	63
Park				.21	.39	.39	56

[a]Backcross to the parent in parentheses.

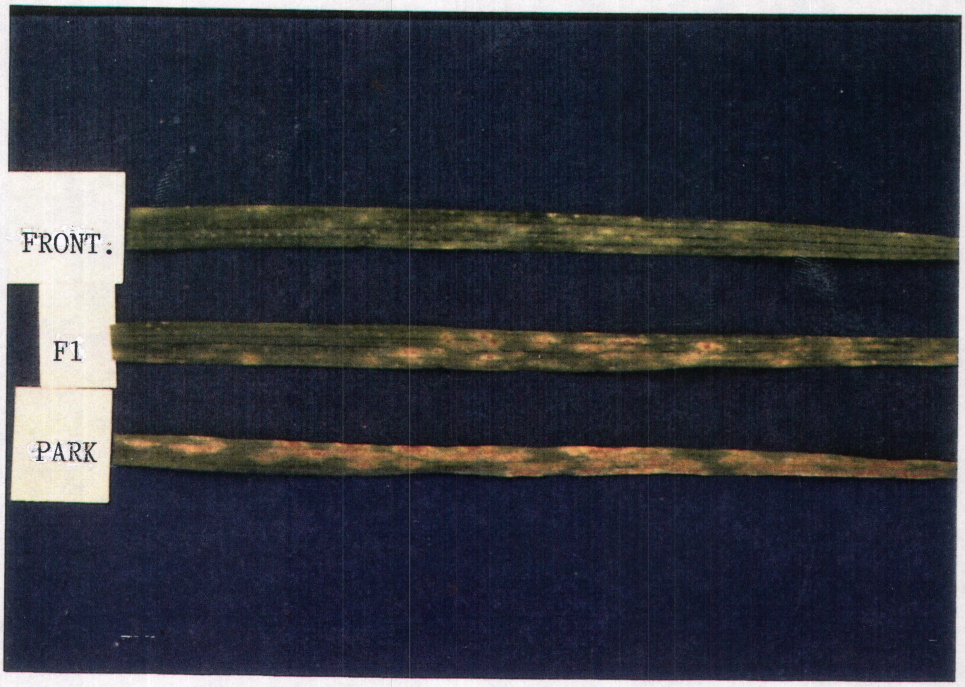

Figure 3.2. Typical disease reaction on parental and F1 generations of the cross Frontana x Park

resistance as the F1 generation. The overlap was minimal in the Oasis x Park (Table 3.3) and French Peace x Park (Table 3.5) crosses, but moderate in the Frontana x Park cross (Table 3.6) and substantial in the Bunyip x Park cross (Table 3.7). The high number of BC1 plants within the range of the susceptible parent in the Bunyip x Park cross supports the conclusion that resistance is recessive in this cross.

The backcross to the susceptible parent (BC2) produced distributions that were intermediate between those of the F1 generation and of the susceptible parent (Tables 3.3 - 3.7). With the exception of the Bunyip x Park cross, a substantial number of BC2 individuals fell within the range of the resistant parent. This indicated that resistance was dominant or partially dominant in all crosses except Bunyip x Park.

The frequency distribution of individuals in the F2 and F3 generations covered the range 0-5 in all crosses. The F2 and F3 distributions were unimodal and, except for the Bunyip x Park cross, skewed towards the susceptible parent. Unimodal distributions could be caused by incomplete dominance at one or more loci controlling resistance. There was a tendency for a high frequency of intermediate values and few extreme values, especially extreme susceptibility. This suggests that there may be more than one gene controlling resistance, because more intermediate values and fewer extreme values are likely if more than one gene is segregating.

The Kolmogorov-Smirnov test was used to determine whether the observed F2 cumulative relative frequency distribution was consistent with the distribution expected from the F1 and parental generations, assuming a single gene model (Table 3.8). There were fewer extreme values and more intermediate than expected in all crosses, but in no case was the difference large enough to cause a one gene model to be rejected ($P = 0.05$).

3.2.2 Mendelian analysis and one-locus tests

The F1 distribution of crosses Oasis x Park and Lacos x Park showed little or no overlap with the range of the susceptible parent (Tables 3.3, 3.4). Chi-square tests of

Table 3.8. Kolmogorov-Smirnov two-sample tests to determine if the F2 distributions of septoria tritici blotch ratings are compatible with a one-gene hypothesis

Cross	Number sampled[a]	Number of F2 plants	Maximum difference[b]	Critical value[c]
Oasis x Park	28	269	.16	.27
Lacos x Conway	45	284	.09	.22
French Peace x Park	59	348	.09	.19
Frontana x Park	29	291	.18	.26
Bunyip x Park	52	424	.06	.20

[a]The number sampled from the parental and F1 generations to produce an expected cumulative relative frequency curve.
[b]The maximum interval between the expected and observed cumulative relative frequency curves.
[c]Maximum differences exceeding this value are significant at P = 0.05.

simple Mendelian ratios were carried out using the parental ranges to categorize individuals in segregating generations as resistant or susceptible.

The observed segregation ratios for the F2, F3 and BC2 generations of the cross Oasis x Park fit a single dominant gene hypothesis (Table 3.9). The F1 generation data showed that resistance is partially dominant (Table 3.3). Single, partially dominant gene control of resistance in Oasis has been reported also by Shaner and Buechley (1989).

Segregation in the F2 and BC2 generations of the Lacos x Conway cross suggested a single dominant gene hypothesis, but this model was rejected in the chi-square test of the F3 generation ($P < 0.01$). Segregation among individuals in the F3 generation fit a hypothesis of two independent genes controlling resistance, one dominant and one recessive (Table 3.10). The segregation ratio in the F2 generation also fit this model better than a single-gene model.

In the crosses involving French Peace, Frontana and Bunyip, the F1 distribution overlapped that of both the parents, so the procedure suggested by Elston (1966), outlined in Section 3.1.3, was used instead of Mendelian analysis (Tables 3.11 - 3.13). The probability of being rated as susceptible (P(S)) for heterozygotes was calculated using Eq. (3.1) to be 0.057, 0.198 and 0.750 for the French Peace, Frontana and Bunyip crosses, respectively. Since three plants of the resistant parent Bunyip were rated as susceptible, the probability of a homozygous resistant genotype being rated as susceptible was calculated for that cross using Eq. (3.4) and found to be 0.06. The probability of individuals in other generations being rated as susceptible was calculated as outlined in Table 3.2.

In the French Peace x Park cross five generations were involved in the chi-square test (Table 3.11). The low number of F1 plants that were rated as susceptible was reflected in the low P(S) value for heterozygous plants. The expected ratios for the F2, F3 and BC2F1 generations were similar to those used in Mendelian analysis. The small probability of a heterozygous plant being rated as susceptible served to lower the expected ratio of

Table 3.9. Chi-square tests of segregation for resistance to septoria tritici blotch in the cross Oasis x Park tested at the three-leaf stage under controlled environmental conditions

Generation	Resistant	Susceptible	Ratio tested	P
Oasis	50	0	-	-
BC[a](Oasis) F1	70	3	-	-
F1	28	0	-	-
F2	205	64	3:1	0.50 - 0.75
F3	236	163	5:3	0.10 - 0.25
BC(Park) F1	48	47	1:1	1.0
Park	0	47	-	-

[a]Backcross to the parent in parentheses.

Table 3.10. Chi-square tests of segregation for resistance to septoria tritici blotch in the cross Lacos x Conway tested at the three-leaf stage under controlled environmental conditions

Generation	Resistant	Susceptible	Ratio tested	P
Lacos	65	0	-	-
BC[a](Lacos) F1	73	0	-	-
F1	43	2	-	-
F2	226	58	13:3	0.50 - 0.75
F3	312	87	49:15	0.25 - 0.50
BC(Conway) F1	36	41	1:1	0.50 - 0.75
Conway	0	64	-	-

[a]Backcross to the parent in parentheses.

Table 3.11. Test of a one-locus hypothesis for control of resistance to septoria tritici blotch in the cross French Peace x Park tested at the three-leaf stage under controlled environmental conditions

Generation	P(S)[a]	Plants rated	Resistant	Susceptible	X^2
French Peace	0	69	69	0	-
BC[b](Fr.P.) F1	0.029	90	88	2	0.14
F1	0.057	59	54	5	0.80
F2	0.279	348	252	96	0.02
F3	0.389	407	256	151	0.55
BC(Park) F1[c]	0.529	50	22	28	0.18
Park	1.0	70	0	70	-
Total $X^2_{(4)} = 1.69$		P = 0.75 - 0.90			

[a]Probability of being rated as susceptible. See Section 3.1.3 in text.
[b]Backcross to the parent in parentheses.
[c]Based on progeny classification.

Table 3.12. Test of a one-locus hypothesis for control of resistance to septoria tritici blotch in the cross Frontana x Park tested at the three-leaf stage under controlled environmental conditions

Generation	P(S)[a]	Plants rated	Resistant	Susceptible	X^2
Frontana	0	37	37	0	-
BC[b](Front.) F1	0.099	81	74	7	0.14
F1	0.198	48	33	15	3.97
F2	0.349	291	201	90	2.03
F3	0.425	407	237	170	0.09
BC(Park) F1	0.599	80	27	53	1.35
Park	1.0	29	0	29	-

Total $X^2_{(4)} = 7.58$ P = 0.10 - 0.25

[a]Probability of being rated as susceptible. See Section 3.1.3 in text.
[b]Backcross to the parent in parentheses.

Table 3.13. Test of a one-locus hypothesis for control of resistance to septoria tritici blotch in the cross Bunyip x Park tested at the three-leaf stage under controlled environmental conditions

Generation	P(S)[a]	Plants rated	Resistant	Susceptible	X^2
Bunyip	0.060	52	49	3	0
BC[b](Bny.) F1	0.405	53	37	16	2.36
F1	0.750	68	18	50	0.08
F2	0.640	424	156	268	0.12
F3	0.585	788	311	477	1.34
BC(Park) F1	0.875	63	6	57	0.52
Park	1.0	56	0	56	-
Total $X^2_{(4)} = 4.42$		P = 0.25 - 0.50			

[a]Probability of being rated as susceptible. See Section 3.1.3 in text.
[b]Backcross to the parent in parentheses.

resistant to susceptible plants. The total chi-square test did not reject a one-locus hypothesis for this cross (P = 0.75 - 0.90).

In the Frontana x Park cross the relatively high P(S) value (0.198) for heterozygotes resulted in a substantial adjustment to the expected number of susceptible plants in segregating generations (Table 3.12). Five generations were used in the chi-square analysis and the one-locus hypothesis was not rejected (P = 0.10 - 0.25). The greatest contribution to the chi-square value came from the F1 generation, in which there were more susceptible plants than expected.

In the Bunyip x Park cross six generations were used in the chi-square test, but since a P(S) value had to be calculated for both the resistant parent genotype and the heterozygote genotype, the degrees of freedom for the chi-square test remained at four (Table 3.13). The one-locus hypothesis was not rejected (P = 0.25 - 0.50).

As with the Kolmogorov-Smirnov tests, the one-locus tests suggested that single genes control resistance in French Peace, Frontana and Bunyip. However, as Elston (1966) pointed out, the one-locus test can reject a one-locus hypothesis but it cannot prove that only one locus is involved, because more complex models can usually be formulated that will explain the results equally well.

Analysis of F3 family segregation ratios was used to verify the results based on individual plants. Since the number of plants in each family was relatively small (8-9), the expected segregation ratio was adjusted by the method outlined in Section 3.1.3. This adjustment results in more families expected to be homozygous and fewer segregating than would normally be the case.

With a single dominant gene hypothesis, it is extremely unlikely (P < 0.001) for a family to segregate in the ratio of 1R:7S. Therefore in crosses involving Oasis, French Peace and Frontana, a single resistant plant in an otherwise susceptible family was considered to be either an escape or a misclassification. That family was considered homozygous susceptible.

In the Oasis x Park cross the observed F3 family segregation data fit the adjusted ratio expected for segregation of a single gene, but the chi-square value was close to the rejection level of P = 0.05 (Table 3.14). Only six homozygous susceptible families were observed, although the expected number was 12.5. However, it was observed that only six of the 50 F2 plants used to produce the F3 families had been rated as susceptible. The unexpectedly low number of homozygous susceptible families may have been due to non-representative sampling of the F2 population.

The F3 family data for the Lacos x Conway cross supported the hypothesis that one dominant and one recessive gene independently confer resistance (Table 3.15). The large number of homozygous resistant families and the small number of homozygous susceptible families fit a two-gene model (P = 0.5 - 0.75), but not a single-gene hypothesis (P < 0.01).

The F3 family segregation in crosses involving French Peace (Table 3.16) and Frontana (Table 3.17) fit single dominant gene hypotheses, although there were fewer than expected homozygous families in each case.

In the Bunyip x Park cross the F3 family segregation indicated a single recessive gene model (Table 3.18). Since Bunyip itself was occasionally classed as susceptible, families with one or two susceptible individuals were classed as homozygous resistant.

Tests of F3 family segregation supported the results of Mendelian analysis and the one-locus tests in all crosses. Family segregation data in Oasis, French Peace, Frontana and Bunyip crosses fit single-gene models, although there were more segregating and fewer homozygous families than expected in every case. A two-gene model provided the best fit in the Lacos x Conway cross.

3.2.3 Allelism

To determine if the resistant parents possessed different genes for resistance to *S. tritici*, F2 populations from crosses between resistant parents were studied (Table 3.19).

Table 3.14. F3 family segregation for resistance to septoria tritici blotch in the cross Oasis x Park

	Homozygous resistant	Segregating	Homozygous susceptible	Total families
Observed[a]	14	30	6	50
Expected[b]	15	22.5	12.5	50

$X^2_{(2)} = 5.95$ P = 0.05 - 0.10

[a]Families with only one individual rated as resistant are considered to be homozygous susceptible.
[b]Based on a hypothesis of a single dominant gene conferring resistance and adjusted for a family size of eight.

Table 3.15. F3 family segregation for resistance to septoria tritici blotch in the cross Lacos x Conway

	Homozygous resistant	Segregating[a]		Homozygous susceptible	Total families
		R≥S	R<S		
Observed	27	15	3	5	50
Expected[b]	24.9	15.8	5.6	3.7	50

$X^2_{(3)} = 1.9$ P = 0.50 - 0.75

[a]Segregating families with more resistant than susceptible, or equal numbers of each (R≥S), and families with fewer resistant than susceptible (R<S).
[b]Based on a hypothesis of two independent genes, one dominant and one recessive, and adjusted for a family size of eight.

Table 3.16. F3 family segregation for resistance to septoria tritici blotch in the cross French Peace x Park

	Homozygous resistant	Segregating	Homozygous susceptible	Total families
Observed[a]	12	24	10	46
Expected[b]	13.2	21.3	11.5	46

$X^2_{(2)} = 0.65$ P = 0.50 - 0.75

[a]Families with only one individual rated as resistant are considered to be homozygous susceptible.
[b]Based on a hypothesis of a single dominant gene conferring resistance and adjusted for a family size of nine.

Table 3.17. F3 family segregation for resistance to septoria tritici blotch in the cross Frontana x Park

	Homozygous resistant	Segregating	Homozygous susceptible	Total families
Observed[a]	23	57	20	100
Expected[b]	30	45	25	100

$X^2_{(2)} = 5.83$ P = 0.05 - 0.10

[a]Families with only one individual rated as resistant are considered to be homozygous susceptible.
[b]Based on a hypothesis of a single dominant gene conferring resistance and adjusted for family size of eight.

Table 3.18. F3 family segregation for resistance to septoria tritici blotch in the cross Bunyip x Park

	Homozygous resistant	Segregating	Homozygous susceptible	Total families
Observed[a]	21	55	24	100
Expected[b]	25	45	30	100

$X^2_{(2)} = 4.06$ P = 0.10 - 0.25

[a]Families with less than three individuals rated as susceptible are considered to be homozygous resistant.
[b]Based on a hypothesis of a single recessive gene controlling resistance and adjusted for a family size of eight.

Table 3.19. Chi-square tests for allelism of genes for resistance to septoria tritici blotch in five wheat cultivars

Cross[a]	Resistant	Susceptible	Ratio[b] tested	P[c]
Oasis x Lacos	168	7	61:3	0.75 - 0.90
Oasis x Frontana	168	10	15:1	0.75 - 0.90
Lacos x Frontana	155	5	61:3	0.25 - 0.50
Lacos x French Peace	108	8	61:3	0.25 - 0.50
Lacos x Bunyip	152	25	55:9	1.0
Frontana x French Peace	102	10	15:1	0.25 - 0.50
Frontana x Bunyip	160	0	13:3	<.005
French Peace x Bunyip	76	20	13:3	0.50 - 0.75

[a]Oasis x French Peace (Fr.P.) and Oasis x Bunyip crosses were not tested.
[b]Based on results of crosses between resistant and susceptible cultivars.
[c]Probability of a larger X^2.

The crosses Oasis x French Peace and Oasis x Bunyip were not tested because of sterility of the F1 hybrids. Only in the Frontana x Bunyip cross were no susceptible plants observed. Park, a susceptible cultivar, was included in these tests to determine the cut-off point. The observed F2 segregation ratios fit those expected, assuming that Oasis, French Peace and Frontana have single dominant genes, Bunyip has a single recessive and Lacos has one dominant and one recessive gene controlling resistance.

In the Frontana x Bunyip cross, no F2 plant was rated as being more susceptible than Bunyip. This suggested that their genes for resistance to *S. tritici* may be at or near the same locus. The small difference between reactions on Bunyip and on Park in this experiment, coupled with the lack of extremely susceptible F2 plants that has been noted in other crosses, may have prevented identification of segregation for resistance in this cross.

These tests indicated that, with the possible exception of Frontana and Bunyip, the resistant parents possess different genes for resistance to *S. tritici*. Combining these genes may provide a greater level of resistance. Rigorous testing would be required to identify plants possessing both resistance genes where each resistance gene already provides a good level of resistance.

If physiologic specialization exists in the pathogen, the different resistance genes may be required to provide protection from different pathogenic races. In this case, testing with different physiologic races would provide a means of identifying plants carrying more than one gene for resistance.

The data presented support hypotheses of simple genetic control of resistance to *S. tritici*. This finding agrees with most other workers who have studied inheritance of resistance to *S. tritici* (Mackie, 1929; Narvaez and Caldwell, 1957; Rillo and Caldwell, 1966; Rosielle and Brown, 1979; Wilson, 1979, 1985; Shaner and Buechley, 1989). Specifically, the conclusion that a single, partially dominant gene controls resistance in Oasis agrees with the results of Shaner and Buechley (1989). The conclusion that a single,

incompletely dominant gene governs resistance in Frontana is supported indirectly by evidence that P14, a derivative of Frontana, possesses an incompletely dominant gene for resistance (Narvaez and Caldwell, 1957).

3.2.4 Gene number estimates

The low frequency of very high or very low plant disease ratings in the F2 populations and the general occurrence of a smaller than expected number of homozygous F3 families suggest that other genes may be involved, perhaps in a modifying role.

Danon et al. (1982) and van Ginkel and Scharen (1988) used a biometrical formula to estimate the number of genes controlling reaction to *S. tritici*. The formula used was reported by Burton (1951) and is derived from the Castle-Wright formula.

Using these formulae, the estimated number of genes controlling resistance to *S. tritici* in the five crosses in this study, ranged from 1.1 to 3.0 (Table 3.20). Estimates made using the Burton formula were slightly higher than the Castle-Wright estimates where dominance was involved. Estimates using F3 population data agreed well with estimates based on F2 population data in crosses involving Oasis, Lacos and Frontana, but were somewhat larger in crosses involving French Peace and Bunyip.

Whereas qualitative analysis had suggested single gene control of resistance in the Oasis, French Peace and Frontana crosses, these biometric formulae usually gave estimates of two or three genes. The low frequency of extremely low or extremely high values in the F2 and F3 generations resulted in reduced estimates of genetic variance and in gene number estimates of more than one. The biometric gene number estimates suggest that the number of genes influencing the reaction to septoria tritici blotch may be greater than one, but is still quite small.

The gene number estimates reported here are similar to those reported by Danon et al. (1982). Van Ginkel and Scharen (1988) obtained some very large gene number estimates

Table 3.20. Estimates of number of genes conditioning resistance to septoria tritici blotch in the F2 and F3 generations of five wheat crosses

	Estimation formula and generation used			
	Castle-Wright[a]		Burton[b]	
Cross	F2	F3	F2	F3
Oasis x Park	2.4	2.4	2.8	2.7
Lacos x Conway	2.2	2.4	2.6	2.7
French Peace x Park	1.5	2.7	1.8	3.0
Frontana x Park	1.4	1.6	2.1	2.0
Bunyip x Park	1.1	1.7	1.1	1.7

[a]Number of genes = $(P1 - P2)^2/(\text{constant})Vg$, where Vg = total genetic variance, constant = 8 for F2 and 16/3 for F3.

[b]Number of genes = $.25(.75 - h + h^2)(P1 - P2)^2/Vg$ in the F2 and $(3.75 - 3h + 3h^2)(P1 - P2)^2/(16Vg)$ in the F3, where Vg = total genetic variance and h = (F1 - P1)/(P2 - P1) and P1 is the smaller parental mean.

in their durum wheat crosses. However, van Ginkel and Scharen (1988) modified the formula by using the F2 generation to estimate the genotypic range. Mulitze and Baker (1985) have pointed out that using a phenotypic range to estimate a genotypic range can cause an upward bias in the gene number estimate if the heritability is less than 1.0. The average heritability in the crosses analyzed by van Ginkel and Scharen (1987) was 38%.

Castle-Wright and Burton estimates are considered to be minimal estimates because departures from the assumptions result in downward bias (Mulitze, 1983). However, there may be some systematic upward bias in these estimates. If the parental plants are recognized when rating, there may be an unconscious tendency to give low values to the resistant parent and high values to the susceptible. This will increase the estimate of genotypic range, resulting in a higher gene number estimate.

The Kolmogorov-Smirnov, chi-square, one-locus and F3 family segregation analyses all supported the hypothesis of single gene control of resistance in crosses involving Oasis, French Peace, Frontana and Bunyip and one or two gene control of resistance in the Lacos x Conway cross. Paradoxically, the gene number estimates were all greater than one. The tendency towards intermediate rating values that was noted in the frequency distributions was not severe enough to cause one-locus hypotheses to be rejected using a Kolmogorov-Smirnov test, but it reduced estimates of genetic variance enough to cause gene number estimates to be greater than one. Apparently the Kolmogorov-Smirnov test, with the sample size used in these experiments, is not as sensitive as the biometric formulae to departures from the expected frequency distribution. The Kolmogorov-Smirnov test even accepted a one-gene model in the Lacos X Conway cross, where Mendelian analysis suggested that two genes were involved.

Classification of plants as resistant or susceptible resulted in acceptance of simple genetic hypotheses, suggesting that resistance mechanisms are under single gene control. Tendency towards intermediate values in frequency distributions, fewer than expected homozygous F3 families and the gene number estimates suggest that other genes are at

least modifying the expression of resistance.

3.2.5 Heritability

Although disease ratings were categorized as resistant and susceptible, it was obvious from the variation within the non-segregating generations that environment played a role in producing the disease reaction phenotype. Narrow-sense heritability estimates ranged from 38 to 62% with a mean of 47% (Table 3.21). As would be expected in crosses where dominance was detected, broad-sense heritability estimates were larger with a mean of 65.4%. The cross with the widest range between parental means (Lacos x Conway) produced the largest heritability estimates, and the cross with the narrowest parental range (Bunyip x Park) produced the smallest estimates.

The heritability estimates indicate the effectiveness of selection of F2 individuals. Where resistance is dominant, selection against susceptible F2 individuals would be more effective than selection for resistant individuals. However, heritability estimates derived from growth-room seedling tests are only valid for predicting the effectiveness of selection carried out under similar conditions.

The heritability estimates for the crosses in this study are quite similar in magnitude to the standard unit heritability estimates reported by Rosielle and Brown (1979), but are larger, on average, than the estimates reported by van Ginkel and Scharen (1987). Many of the parents used by van Ginkel and Scharen (1987) had similar disease reactions. Small heritability estimates could be expected in many cases since 20 out of 65 crosses they studied showed no significant genetic effects according to generation means analyses.

Table 3.21. Estimates of broad and narrow-sense heritability of resistance to septoria tritici blotch in five crosses

Cross	Broad-sense heritability[a]	Narrow-sense heritability[b]	
Oasis x Park	0.60	0.49	(0.27 - 0.71)[c]
Lacos x Conway	0.72	0.62	(0.46 - 0.78)
French Peace x Park	0.70	0.45	(0.28 - 0.62)
Frontana x Park	0.69	0.41	(0.19 - 0.63)
Bunyip x Park	0.56	0.38	(0.21 - 0.56)

[a]Heritability = (VF2 - (VP1 + VP2 + VF1)/3)/VF2. [b]Heritability = Regression coefficient where F3 family means are regressed on F2 individual values.
[c]95% confidence interval.

4.0 Pathogenic variation

4.1 Materials and methods

4.1.1 Plant material

The cultivars used in this study represented a wide range of disease reaction to isolate Sask2 of *S. tritici* (Table 4.1).

Two experiments, each with 50 plants of each cultivar were conducted. The plants were grown as described in Section 3.1.1.

Table 4.1. Disease reaction to isolate 'Sask2' of *Septoria tritici* of cultivars used in the pathogenic variation experiments

Cultivar	Species	Experiment	Disease reaction
HY320	*T. aestivum*	1 + 2	intermediate
Oslo	*T. aestivum*	1 + 2	resistant
Lacos	*T. aestivum*	1 + 2	resistant
Du75	*X Triticosecale*	1 + 2	resistant
Oasis	*T. aestivum*	1	resistant
Arthur 71	*T. aestivum*	1	unknown
Frontana	*T. aestivum*	2	resistant
Maris Butler	*T. aestivum*	2	unknown
Sceptre	*T. durum*	1 + 2	unknown
Park	*T. aestivum*	1 + 2	susceptible
Benito	*T. aestivum*	1 + 2	susceptible

4.1.2 Pathogen culture and inoculation

Nine of the isolates used in this study were collected from widely dispersed locations

within the northern grain-belt of Saskatchewan. One isolate was obtained from England and another from Indiana. The geographic origin of the isolates is listed in Table 4.2.

Isolation of the fungus and production of liquid cultures of each isolate were performed as described in Section 3.1.2. Single spore cultures of each isolate were produced by flooding yeast-malt agar plates with liquid cultures containing low spore concentrations. Re-isolation was made from individual colonies, each of which was believed to have been derived from a single spore.

To produce inoculum, yeast-malt agar plates were flooded with approximately 0.75 mL of liquid culture. Spores were harvested by washing 4-day-old plate cultures with

Table 4.2. Origin and designation of isolates of *Septoria tritici* used in pathogenic variation experiments

Place of origin	Isolate designation	Year isolated	Experiment
England	Engl	1988	1 + 2
Battleford, Sask.	Sask1	1985	1 + 2
Tarnapol, Sask.	Sask2	1985	1 + 2
Fosston, Sask.	Sask3	1985	1 + 2
Saskatoon, Sask.	Sask4	1987	1 + 2
Weirdale, Sask.	Sask5	1988	1 + 2
Shellbrook, Sask.	Sask6	1988	1 + 2
Weirdale, Sask.	Sask7	1985	1
Naicam, Sask.	Sask8	1988	1 + 2
Tisdale, Sask.	Sask9	1988	2
Indiana	Ind1	1988	1 + 2

distilled water. Spore concentrations were checked using a haemocytometer and adjusted to 1.0×10^6 mL^{-1}. Care was taken not to mix cultures of different isolates.

Before inoculation, plants were removed from the growth room and laid flat on lab benches. The third leaf of each plant was held in place by taping the tip of the leaf to the bench. Three holes, approximately 4 cm apart, were poked in each leaf using a dissecting needle and a 10 µL drop of inoculum was placed on each hole. This inoculation method was suggested by G. Buechley and Dr. G. Shaner (Purdue University, Indiana). Five plants of each cultivar-isolate combination were inoculated in each experiment. When the drops had dried, the plants were returned to the growth room and arranged in a completely randomized design.

Inoculated leaves were removed from the plants 14 days after inoculation and examined under a dissecting microscope. Lesion length was measured and the number of pycnidia within each lesion counted.

4.1.3 Statistical analysis

Analysis of variance of each variable was performed in each experiment. Analyses of variance were also carried out on the combined experiments, using those cultivars and isolates common to both experiments. Before analysis, pycnidial counts (X) were transformed to $X' = \log_{10}(X + 1)$.

Where cultivar x isolate interactions were significant, a Gail-Simon test for crossover interactions was conducted (Gail and Simon, 1985). This test was used to determine if cultivar x isolate interactions involved significant changes in rank. Interactions that involve change in rank are referred to as "crossover" or "qualitative" interactions. As used in this study, this test compares all possible pairs of isolates to determine if one isolate produces significantly greater disease on some host genotypes, while producing significantly less disease on others. Plant means were used for all analyses, and the standard error of mean difference used in the Gail-Simon test was calculated using the

pooled error variance in each experiment. The critical values for this test were obtained from Table 1 of Gail and Simon (1985). This procedure was proposed by Baker (1988) as a test for identifying meaningful genotype x environment interactions.

4.2 Results and discussion

In this thesis, "virulence" refers to the ability of a pathogen genotype to successfully infect a particular host genotype. "Pathogenicity" will be used as an inclusive term that can refer to both qualitative and quantitative differences in infection.

The pin-hole method of inoculation allowed for complete randomization of cultivar-isolate combinations with little risk of cross-infection. It also enabled accurate, objective measurement of disease symptoms. Lesion length and the number of pycnidia within each lesion were measured in both experiments. This method produced more severe symptoms on some host genotypes than the usual spray inoculation method. Pycnidia formed on Lacos, Oslo and Oasis within 14 days, whereas pycnidia seldom developed within 17 days when these cultivars were spray-inoculated with the same fungal isolate. Contrarily, the method of inoculation had no effect on the resistance of the triticale cultivar Du75. The lesions on this cultivar were scarcely larger than the pin-holes themselves (Tables 4.3, 4.5), and no pycnidia were found in any lesion in either experiment (Tables 4.4, 4.6). Since disease symptoms were qualitatively different on Du75 than on other cultivars, and showed little or no variation among isolates, it was not included in analyses of variance or tests for qualitative interactions.

Cultivars Oasis and Arthur 71 were not included in Experiment 2. These were the only winter wheat types in the experiment and the leaves of the unvernalized seedlings appeared to senesce faster than those of the spring types. In the first experiment, Arthur 71 showed disease symptoms that were similar to those on Oasis, even when inoculated with the isolate from Indiana (Tables 4.3, 4.4). This was unexpected since Oasis possesses a resistance gene that is not found in Arthur 71 (Patterson et al., 1975). This suggests that

Table 4.3. Mean lesion length (mm) for cultivars, *Septoria tritici* isolates and cultivar x isolate combinations used in Experiment 1

	Cultivar									
Isolate	HY320	Oslo	Lacos	Du75	Oasis	Arthur71	Sceptre	Park	Benito	Mean
Engl	6.0	7.7	8.6	1.3	5.8	6.6	10.4	8.1	10.1	7.3
Sask1	6.5	5.5	4.0	1.3	3.9	3.2	5.9	7.5	9.0	5.2
Sask2	7.3	7.6	6.0	1.4	4.4	5.2	8.9	8.2	10.8	6.7
Sask3	8.6	7.3	6.6	1.5	4.3	4.1	5.7	11.4	11.4	7.0
Sask4	9.3	7.6	4.7	1.5	4.7	5.7	6.3	10.6	10.9	6.8
Sask5	9.6	5.2	6.5	1.2	6.2	4.6	5.2	9.3	10.3	6.5
Sask6	8.4	7.1	4.9	1.3	4.6	3.9	5.7	8.4	9.8	6.0
Sask7	8.1	4.5	3.6	1.2	3.8	3.3	4.1	7.7	9.4	5.1
Sask8	8.1	4.5	4.1	1.1	3.7	4.9	6.0	7.9	9.1	5.5
Ind1	9.9	9.9	8.7	1.4	5.1	5.3	8.3	8.3	10.1	7.6
Mean	8.2	6.8	5.8	1.3	4.9	4.9	6.7	8.7	10.1	

Standard error of difference for cultivar x isolate means = 0.93

Table 4.4. Mean pycnidia per lesion (log10 (x + 1)) for cultivars, *Septoria tritici* isolates and cultivar x isolate combinations used in Experiment 1

	Cultivar									
Isolate	HY320	Oslo	Lacos	Du75	Oasis	Arthur71	Sceptre	Park	Benito	Mean
Eng1	1.01	1.27	1.63	0.00	1.15	1.09	1.67	1.86	1.85	1.28
Sask1	0.86	0.55	0.48	0.00	0.68	0.98	0.78	1.66	1.76	0.86
Sask2	0.92	0.72	1.01	0.00	0.93	1.10	1.22	1.82	2.02	1.08
Sask3	1.44	0.88	1.14	0.00	1.14	1.14	0.92	2.17	2.22	1.23
Sask4	1.58	1.08	0.94	0.00	1.09	1.32	1.21	2.00	2.01	1.23
Sask5	1.64	0.82	1.19	0.00	1.50	1.25	0.94	2.10	2.16	1.29
Sask6	1.55	0.93	0.64	0.00	1.02	1.00	1.04	1.99	2.19	1.14
Sask7	1.05	0.38	0.50	0.00	0.84	0.67	0.64	1.75	1.88	0.84
Sask8	1.47	0.43	0.71	0.00	0.95	0.97	1.23	1.83	2.05	1.07
Ind1	1.74	1.81	1.36	0.00	1.24	1.32	1.54	1.69	1.80	1.39
Mean	1.33	0.89	0.96	0.00	1.05	1.08	1.12	1.89	1.99	

Standard error of difference between cultivar x isolate means = 0.17

Table 4.5. Mean lesion length (mm) for cultivars, *Septoria tritici* isolates and cultivar x isolate combinations used in Experiment 2

	Cultivar									
Isolate	HY320	Oslo	Lacos	Du75	Frontana	M. Butler	Sceptre	Park	Benito	Mean
Engl	5.5	7.1	7.6	1.3	5.0	5.9	8.0	6.2	8.2	6.1
Sask1	3.5	1.8	2.1	1.5	1.4	2.0	2.9	6.8	7.0	3.2
Sask2	5.2	4.0	2.1	1.2	1.3	3.2	7.2	8.4	7.6	4.4
Sask3	5.2	3.7	2.5	1.2	1.6	2.5	6.0	9.0	9.1	4.4
Sask4	5.9	3.4	2.5	1.2	1.7	3.9	5.7	7.2	6.7	4.2
Sask5	4.5	2.6	1.5	1.1	1.7	4.0	4.6	7.3	8.5	4.0
Sask6	4.8	2.7	2.0	1.1	3.7	3.1	4.7	7.9	7.7	4.2
Sask9	4.5	3.3	3.0	1.1	2.1	3.1	5.1	8.8	8.1	4.3
Sask8	5.9	2.3	1.8	1.0	2.7	2.5	4.4	7.9	8.3	4.1
Ind1	7.6	6.5	3.1	1.2	5.4	2.4	7.9	5.0	6.3	5.1
Mean	5.3	3.8	2.8	1.2	2.6	3.2	5.6	7.4	7.8	

Standard error of difference between cultivar x isolate means = 0.71.

Table 4.6. Mean pycnidia per lesion (log10 (x + 1)) for cultivars, *Septoria tritici* isolates and cultivar x isolate combinations used in Experiment 2

	Cultivar									
Isolate	HY320	Oslo	Lacos	Du75	Frontana	M. Butler	Sceptre	Park	Benito	Mean
Engl	0.84	1.07	1.38	0.00	0.57	0.96	1.13	0.87	1.29	0.89
Sask1	0.75	0.36	0.35	0.00	0.00	0.28	0.57	1.45	1.49	0.58
Sask2	0.94	0.75	0.39	0.00	0.00	0.56	0.85	1.57	1.62	0.72
Sask3	1.16	0.83	0.73	0.00	0.24	0.41	0.85	1.59	2.01	0.85
Sask4	1.16	0.64	0.39	0.00	0.00	0.78	0.77	1.69	1.62	0.78
Sask5	0.91	0.58	0.42	0.00	0.00	0.81	0.69	1.85	2.08	0.83
Sask6	1.32	0.68	0.49	0.00	0.37	0.56	0.69	1.91	1.90	0.87
Sask9	0.99	0.71	0.79	0.00	0.26	0.53	0.74	1.82	1.79	0.82
Sask8	1.21	0.61	0.33	0.00	0.26	0.50	0.86	1.74	1.86	0.82
Ind1	1.50	1.49	0.66	0.00	0.86	0.05	1.03	1.37	1.51	0.95
Mean	1.07	0.77	0.59	0.00	0.26	0.54	0.81	1.60	1.72	

Standard error of difference between cultivar x isolate means = 0.15.

the cultivar designation of the seed may have been incorrect.

Differences among isolates in the spore concentration used, or differences in the ability of isolates to produce viable spores in culture, could cause isolates to differ in their apparent pathogenicity. Isolate Sask7 was not used in the second experiment because it tended to produce fewer spores in culture than other isolates.

In analyses of variance for lesion length and pycnidial count in Experiment 1 (Table A.2) and for the same variables in Experiment 2 (Table A.3), the cultivar x isolate interaction was highly significant, although small relative to the main effects. For pycnidial count, the interaction mean square was 2.4% of the total variance in Experiment 1 and 2.5% of the total variance in Experiment 2. For lesion length, the interaction mean square accounted for 3.0% of the total variance in both experiments.

Combined analyses of variance using only those cultivars and isolates that were used in both experiments were conducted (Table 4.7). For both lesion length and pycnidial count the experiment x cultivar x isolate interaction was small but significant. This indicated that at least some of the cultivar x isolate interactions were experiment specific. Failure of cultivar x isolate interactions to be consistent in different environments has been discussed by Kulkarni and Chopra (1982). In the present study the most likely causes of change in cultivar x isolate interactions would be changes in spore concentration or spore viability from one experiment to the other.

The cultivar x isolate interactions for lesion length and pycnidial count were significantly larger than the three-way interactions. This indicates that some of the cultivar x isolate interactions were consistent in the two experiments.

The Gail-Simon test can be used to identify which isolates and cultivars are involved in crossover interactions and whether or not they are the same in each experiment. When two isolates are compared in a Gail-Simon test, a significant result indicates that one isolate produced significantly larger lesions or more pycnidia on one or more cultivars, while the other isolate produced larger lesions or more pycnidia on one or more other

Table 4.7. Analysis of variance of lesion length (mm) and pycnidia per lesion (log10 (x + 1) transformation) for Experiments 1 and 2 combined[a]

Source of variation[b]	Degrees of freedom	Mean square	
		Lesion length	Pycnidia
Experiment (E)	1	787.62**	12.656**
Cultivar (C)	5	244.64**	18.083**
Isolate (I)	8	43.83**	1.315**
E x C	5	16.27**	0.185*
E x I	8	5.32**	0.161**
C x I	40	13.26**	0.633**
E x C x I	40	3.47**	0.092*
Error	422	1.95	0.062

*,** Significant at P = 0.05 and 0.01, respectively.
[a]Includes only those cultivars and isolates used in both trials.
[b]Experiment effects are considered random, those of cultivar and isolate fixed.

cultivars. In Experiment 1, 13 interactions for either lesion length or pycnidial count were significant (Table 4.8). Eleven of these were comparisons involving either isolate Eng1 or isolate Ind1 and a Saskatchewan isolate. However, since this test was done on all 45 possible isolate comparisons, the experimentwise error protection was poor and some interactions may have been falsely declared significant.

The results of the Gail-Simon test on Experiment 2 confirmed, in general, the results of Experiment 1; 26 of the 28 significant interactions involved isolates Eng1 or Ind1 (Table 4.9). The significant interactions involving only Saskatchewan isolates were not the same as those in Experiment 1. This suggests these interactions may have been due to chance.

On some genotypes, isolate Eng1 produced larger lesions, but fewer pycnidia, than some other isolates (Tables 4.5, 4.6). This resulted in more significant crossover interactions involving Eng1 for pycnidial count than for lesion length, particularly in Experiment 2 (Table 4.9).

Examination of the tables of cultivar-isolate means will reveal which cultivars reacted differently to different isolate pairs, thus causing significant crossover interactions (Tables 4.3 - 4.6). Oslo, a cultivar that has been observed to be resistant in the field in Saskatchewan, and Lacos, highly resistant in growth room tests, were both more susceptible to isolate Eng1 than to Saskatchewan isolates. The Saskatchewan isolates tended to be more pathogenic on Park, Benito and HY320, cultivars that have been grown in western Canada. Sceptre, also a western Canadian cultivar, was consistently more susceptible to isolate Eng1 than to the Saskatchewan isolates.

Isolate Ind1 was more pathogenic on most cultivars than the Saskatchewan isolates (Tables 4.3 - 4.6). Ind1 appeared to be particularly pathogenic on Oslo, a cultivar developed in the U.S.A.. Qualitative interactions involving Saskatchewan isolates could be ascribed to lower pathogenicity of Ind1 on Park and Benito, and on Maris Butler in Experiment 2. Ind1 differed from Eng1 in its pathogenicity on HY320 and Maris Butler.

Table 4.8. Results of the Gail-Simon test[a] for crossover interactions between isolates of *Septoria tritici* for pycnidial count and lesion length on eight wheat cultivars in Experiment 1

		Sask1	Sask2	Sask3	Sask4	Sask5	Sask6	Sask7	Sask8	Ind1
Eng1	b	-	-	**	**	***	**	-	*	-
	c	-	-	***	***	**	-	-	-	-
Sask1			-	-	-	-	-	-	-	-
			-	-	-	-	-	-	-	-
Sask2				-	-	-	-	-	-	-
				**	-	**	-	-	-	-
Sask3					-	-	-	-	-	**
					-	-	-	-	-	**
Sask4						-	-	-	-	-
						-	-	-	-	-
Sask5							-	-	-	**
							-	-	-	-
Sask6								-	-	-
								-	-	-
Sask7									-	-
									-	-
Sask8										-
										-

*,**,*** Significant at P = 0.05, 0.025 and 0.001 respectively.
[a]Gail and Simon (1985).
[b]Pycnidia per lesion, transformed by $\log_{10}(x + 1)$.
[c]Lesion length (mm).

Table 4.9. Results of the Gail-Simon test[a] for crossover interactions between isolates of *Septoria tritici* for pycnidial count and lesion length on eight wheat cultivars in Experiment 2

		Sask1	Sask2	Sask3	Sask4	Sask5	Sask6	Sask9	Sask8	Ind1
Engl	b	**	***	***	***	***	***	***	***	***
	c	-	*	**	-	-	-	**	-	-
Sask1			-	-	-	-	-	-	-	-
			-	-	-	-	-	-	-	-
Sask2				-	-	-	-	-	-	**
				-	-	-	**	-	-	***
Sask3					-	*	-	-	-	***
					-	-	-	-	-	***
Sask4						-	-	-	-	***
						-	-	-	-	**
Sask5							-	-	-	***
							-	-	-	***
Sask6								-	-	***
								-	-	***
Sask9									-	***
									-	***
Sask8										***
										***

*,**,*** Significant at P = 0.05, 0.025 and 0.001 respectively.
[a]Gail and Simon (1985).
[b]Pycnidia per lesion, transformed by $\log_{10}(x + 1)$.
[c]Lesion length (mm).

The near absence of crossover interactions among Saskatchewan isolates indicated that cultivar resistance ranking was similar for these isolates (Tables 4.8, 4.9). However, isolate Sask2 may be behaving somewhat differently, since three out the four significant interactions between Saskatchewan isolates involved Sask2. This isolate produced relatively large lesions on the durum cultivar Sceptre.

Although the pattern of cultivar ranking was similar for all of the Saskatchewan isolates, not all of them produced significant crossover interactions with the Eng1 and Ind1 isolates. The overall pathogenicity of the Saskatchewan isolates was variable, and consequently some of the less pathogenic isolates did not produce significantly larger lesions or more pycnidia than Eng1 or Ind1 on any cultivar. This was particularly evident in Experiment 1 (Tables 4.3, 4.4). These differences in pathogenicity of the Saskatchewan isolates may be due to genetic differences, but could also be due to undetected differences in spore concentration or differential ability to produce viable spores in culture.

The presence of crossover interactions indicated that at least some of the genes controlling pathogenicity are specific to certain host genotypes. If virulence is defined as the ability to reproduce on a certain host genotype, then all isolates were virulent on all the cultivars except Du75, and in some cases, Frontana (Tables 4.4, 4.6). In some instances two isolates reproduced on a cultivar and therefore both would be considered virulent. However, substantial differences in the cultivar's reaction to the two isolates would cause the cultivar to be rated as resistant to one but susceptible to the other. For example, Lacos might be considered to be resistant to Saskatchewan isolates but susceptible to isolate Eng1. In some cases the specific reactions did not involve such differences, but rather the differences were in degree of resistance or susceptibility. In Experiment 2, for example, all isolates produced fewer pycnidia on Frontana than on Benito, yet the ranking of the isolates on the two cultivars varied considerably (Table 4.6).

Assuming that these differences in the degree of resistance have a genetic basis, then

there must be minor genes affecting the expression of resistance. These relatively minor, but cultivar specific, differences among isolates might be regarded as adaptation to particular hosts.

Genes that have minor effects on resistance are often assumed to be non-specific (Robinson, 1976). However, significant cultivar x isolate interactions have been reported in several cases where resistance is believed to be polygenically controlled (Caten, 1974; Parlevliet, 1976; Milus and Line, 1980; Kolmer and Leonard, 1986).

Van Silfhout et al. (1989) concluded that genes for both general and specific pathogenicity exist in *S. tritici* because both the isolate and the cultivar x isolate mean squares were significant in an analysis of variance. However, if significant interactions do exist, a significant main effect due to isolates does not provide proof that differences in general pathogenicity exist. The isolate means will depend upon the host genotypes used in the test. If it is established that there are no crossover interactions among the isolates in a test, then general pathogenicity of the isolates can be compared. The Saskatchewan isolates used in these experiments could be compared in this way. However, the main differences between the Saskatchewan isolates appear to be related to the age of the culture, with the older cultures generally being less pathogenic. For this type of study, all isolations should be made from plant material at approximately the same time. Comparisons of the pathogenicity of isolates are also subject to the assumption that the isolate producing the most pathogenic inoculum in culture will be the most pathogenic in the field.

Although a limited number of isolates were used in this study, the results agree with Eyal et al. (1985) and van Silfhout et al. (1989) in that isolates from different parts of the world differ in their pathogenicity. Eyal and Levy (1987) also reported differences in virulence among isolates from different regions and different locations within regions in Israel. Although the Saskatchewan isolates in this study came from locations dispersed over a fairly large area, there were no consistent qualitative differences between them.

Reports of cultivar x isolate interactions have often been based primarily on differences between host species, and isolates collected from different species (Eyal et al., 1973; Yechilevich-Auster et al., 1983; Saadaoui, 1987; van Silfhout et al., 1989). The presence of species other than *T. aestivum* in this study was probably not critical in the detection of cultivar x isolate interactions. The triticale cultivar Du75 reacted uniformly to all isolates, while the durum cultivar Sceptre was one of several cultivars that were more susceptible to the isolates from England and Indiana than those from Saskatchewan. The possible adaptation of *S. tritici* to different host species was not fully investigated in this study, since all isolates are believed to have come from *T. aestivum* cultivars.

The high level of resistance exhibited by Du75 was also noted by Eyal et al. (1985). In their experiments, Du75 was considered to be resistant to all 97 isolates used.

The statistical method used in this study to test for cultivar x isolate interactions is different from those used previously. Other authors working with *S. tritici* have usually used the interaction term in the analysis of variance to indicate whether or not specific cultivar x isolate interactions exist (Eyal et al., 1985; van Ginkel and Scharen, 1988; van Silfhout et al., 1989). Reactions have also been classified according to reaction type, so that differential reactions could be studied (Eyal et al., 1973). Where quantitative measures of disease have been made, cluster analysis has been used to categorize reactions as being resistant or susceptible (Eyal et al., 1985; van Ginkel and Scharen, 1988).

The Gail-Simon test has an advantage over the analysis of variance in that it only considers crossover type interactions. In this aspect it is similar to ranking tests (Vanderplank, 1968; Parlevliet, 1976). It has a possible advantage over ranking tests in that changes in rank have to be of a significant magnitude using the original scale before they are declared significant.

Where the distinction between a resistant and a susceptible reaction is clear, and can be applied to all cultivar-isolate combinations, a traditional differential reaction analysis

can be easily used. However, when disease is measured quantitatively and a cut-off point is established somewhat arbitrarily, a differential reaction analysis could give misleading results. For example, in the experiments of Eyal et al. (1985) the cut-off point was set at 16.6% necrotic leaf area. Using this criterion it would be concluded that a cultivar with a mean necrotic leaf area of 16% when inoculated with a certain isolate, possesses a gene for resistance that a cultivar with a necrotic leaf area of 17% does not. Error involved in measurement has been ignored. The Gail-Simon test does not require any division of reactions into resistant or susceptible classes.

The Gail-Simon test also indicates which isolates are involved in significant cultivar x isolate interactions. If cultivars are considered to be the factor of interest, then the test could also be used to indicate cultivars that are useful in differentiating isolates.

There are some disadvantages to using this test. Like other tests, the results are dependent upon the cultivars chosen to differentiate the isolates. Since it sums the differences between isolates over all cultivars in the test, and the critical value increases with the number of cultivars used, a differential reaction involving only two of the cultivars may not be large enough to be declared significant. Another drawback of the Gail-Simon test is that the number of significant interactions will depend on the significance level chosen, and when many isolates are compared, the overall Type I error rate protection is poor.

The Gail-Simon test also identifies some crossover interactions that are not very important in terms of resistance and susceptibility. For example, the reduced pathogenicity of isolate Engl on Benito contributes to the significant crossover interaction between that isolate and several of the Saskatchewan isolates, but Benito would probably not be considered to be resistant to the Engl isolate.

Despite the limitations of the Gail-Simon test, it can be useful in determining those isolates and cultivars involved in significant interactions. In this study isolates from England and Indiana were found to differ from isolates from Saskatchewan.

5.0 Components of resistance

5.1 Materials and methods

5.1.1 Experiments I and II

Twelve cultivars, 11 common wheat and one triticale, were used in these experiments (Table 5.1). Previous growth room tests indicated that these cultivars represented a range of reactions to *S. tritici*.

Table 5.1. Cultivars used in Experiments I and II, their countries of origin and their reactions to a mixture of isolates of *Septoria tritici* from Saskatchewan

Cultivar	Country of origin	Reaction to *Septoria tritici*
Benito	Canada	susceptible
Conway	Canada	susceptible
Park	Canada	susceptible
Klein Aniversario	Argentina	intermediate
Poso	U.S.A.	intermediate
Centrifen	Chile	intermediate
Fortuna	U.S.A.	intermediate
HY320	Canada	intermediate
Frontana	Brazil	resistant
Lacos	Chile	resistant
Du75 (triticale)	Tunisia	resistant
French Peace	unknown	resistant

The experiments were carried out in a growth cabinet with a photoperiod of 16 hours and a 22°C day/16°C night temperature. Plants were grown in 15 cm diameter pots, with three plants per pot. A mixture of soil:peat:vermiculite in a 1:1:1 ratio was used and 20-20-20 fertilizer was applied every two weeks. In both experiments, four pots of each cultivar were included and a completely randomized design was used.

Plants were inoculated after the third leaf had fully emerged by spraying them until run-off with a liquid culture containing 10^6 spores mL^{-1} of *S. tritici* isolate Sask2. Inoculum was prepared as described in Section 3.1.2. Plants were kept in a mist chamber at 100% RH for three days following inoculation.

In Experiment I measurements were taken on: incubation period (INC), the number of

days from inoculation until symptoms were first observed; latent period (LP), the number of days from inoculation until pycnidia were observed; total spore production (TSP), the number of spores exuded from pycnidia on diseased leaves at the end of the experiment; percent diseased leaf area 11 days after inoculation (DLA1); and percent diseased leaf area 18 days after inoculation (DLA2). All measurements were made on the third leaf.

In Experiment II, only two pots of each cultivar were inoculated, using the same inoculation method as in Experiment I. Uninoculated plants were included to monitor natural leaf senesence and physiologic leaf spotting.

The percent diseased leaf area was estimated every four days. From these estimates, the rate at which lesion area expanded (LER) and the area under the disease progress curve (AUDPC) were calculated. LER was considered to be the slope of the regression of disease over time, after the increase in disease over time had been linearized by the transformation

$\log_e$(proportion diseased / (1 - proportion diseased))

This transformation, commonly called the "logistic" transformation, was suggested by Vanderplank (1963) as a means of linearizing disease progress in a polycyclic epidemic. Rates were also calculated using untransformed values and the "simple interest" transformation suggested by Vanderplank (1963) for monocyclic diseases. Using r^2 values as criteria, the logistic transformation was found to be the best method of linearizing disease progress over time, even though this was a monocyclic disease progress experiment.

AUDPC was calculated using the formula (5.1) suggested by Shaner and Finney (1977)

$$\text{AUDPC} = \sum_{i=1}^{n} [(Y_{i+1} + Y_i)/2] [X_{i+1} - X_i] \qquad (5.1)$$

where Y_i = septoria tritici blotch severity at the i^{th} observation, X_i = time (days) at the i^{th}

observation and n = the total number of observations.

Analysis of variance was carried out for each variable to determine if differences existed among cultivars. Correlation analysis was conducted using cultivar means to examine association among components of resistance.

5.1.2 Experiments III to VI

Six cultivars used in the first two experiments were chosen for more detailed study. Of the six cultivars chosen, two (Benito, Park) were considered to be susceptible, two (Lacos, French Peace) resistant and two (Fortuna, HY320) intermediate. Greater replication was used in these experiments to achieve more precise estimates of the resistance components. Ten pots of each cultivar were included in each experiment, two of which were uninoculated controls. Plants were grown as described in Section 5.1.1, except that the experiments were carried out in a growth room instead of a growth cabinet.

Measurements in Experiments III and IV were made on the third leaf, while measurements in Experiments V and VI were made on the flag leaves of most cultivars. Lacos and HY320 were slower in development than the other cultivars and so measurements were made on penultimate leaves of these cultivars. Despite this, these experiments will be referred to as having been conducted at the flag leaf stage.

In Experiment III, two pots of each cultivar were inoculated by placing 20 μL drops of inoculum with a concentration of 1.0×10^6 spores mL^{-1} on the leaves and spreading the inoculum by stroking the leaves between two fingers. The number of spores applied on a leaf was estimated from the number of spores in a drop and the number of drops used on a leaf. This inoculation procedure was used so that infection frequency could be estimated. The remaining six pots of each cultivar were inoculated as described in Section 5.1.1. Plants in Experiments IV to VI, except controls, were inoculated in the same manner. In all experiments plants were kept in a mist chamber for three days following

inoculation.

Several of the variables measured in Experiments I and II could be described as "measures" of disease severity rather than as components that determine the rate of an epidemic. In Experiments III to VI only four components were measured, infection frequency (INF), latent period (LP), spore production per unit diseased (SP) and lesion expansion rate (LER).

INF was calculated as the number of lesions on a leaf divided by the number of spores applied. Before analysis, INF was coded by multiplying by 100,000 and then transformed by log10 to achieve homogeneity of variance. LP was considered to be the number of days from inoculation until pycnidia were just visible.

SP was expressed as the number of spores produced per cm^2 diseased leaf area. To estimate diseased leaf area, an estimate was made at the end of each experiment of the proportion of the leaf affected by septoria tritici blotch, discounting necrosis and chlorosis believed due to natural senescence. The length and width of each leaf was measured and the leaf area estimated by equation 5.2:

$$\text{leaf area } (cm^2) = \text{length} * \text{width} * 0.80 \qquad (5.2)$$

This was multiplied by the proportion leaf area diseased to give the diseased leaf area in cm^2. The number of spores produced on a leaf was estimated by removing the leaves when they were fully necrotic, soaking them in a known quantity of distilled water for 12 hours, then estimating the spore concentration in the water using a haemocytometer.

LER was calculated as described for Experiment II. To avoid confusion with natural senescence, data collected after the leaves of the uninoculated control plants began to senesce were not used.

Analysis of variance for each variable was based on pot means. Correlation analysis of cultivar means was used to evaluate association among resistance components.

5.1.3 Field experiments

To relate resistance components measured in controlled environment experiments to disease progress in the field, three field experiments were established, two in 1988 at Saskatoon and Shellbrook and one in 1989 at Shellbrook.

The growing seasons of 1988 and 1989 were hot and dry and the level of septoria tritici blotch remained very low in both years even on susceptible cultivars. Consequently, the amount of infection observed in the field experiments was considered to be too low to be useful in relating disease progress to resistance components and the experiments were discarded.

5.2 Results and discussion

5.2.1 Experiments I and II

Highly significant differences were found among cultivars for all components measured in Experiments I and II (Table A.4).

Cultivars that were regarded as susceptible (Table 5.1) combined short LP, high LER, high TSP and high values for DLA1, DLA2 and AUDPC. However, with the occasional exception for INC, cultivars that were considered resistant (Table 5.1) exhibited favourable levels of the variables measured (Table 5.2).

The remaining cultivars produced intermediate values for most variables (Table 5.2). The LP of these cultivars was significantly longer and, except for cultivar Poso, the TSP values were significantly smaller than those of the fully susceptible cultivars. Significant differences in LER between the intermediate and susceptible cultivars were not as common. Cultivars Poso, Klein Aniversario and Fortuna produced LER values that were not significantly smaller than those of the susceptible types.

Except for INC, correlations among resistance components were highly significant (Table 5.3). INC was probably not a very reliable measure of disease, since small leaf spots due to other causes can easily be confused with early disease symptoms. The lack of

Table 5.2. Means for components of resistance to septoria tritici blotch measured on the third leaf of cultivars tested under controlled environmental conditions

	Experiment I					Experiment II	
Cultivar	Inc[a]	DLA1	DLA2	LP	TSP	LER	AUDPC
Benito	7.7 bc[b]	8.3 a	73.9 b	11.5 a	20.08 a	0.47 a	353.8 a
Conway	7.0 ab	8.7 a	77.3 ab	11.5 a	18.66 a	0.42 a	274.8 b
Park	8.4 bc	8.8 a	85.8 a	11.8 a	20.78 a	0.33 ab	213.6 bc
Klein Aniversario	6.9 ab	5.4 b	42.7 c	14.0 b	3.28 b	0.29 abc	170.9 c
Poso	10.0 de	1.2 cd	50.9 c	13.8 b	8.82 a	0.36 a	148.2 c
Centrifen	7.1 ab	3.0 c	18.4 de	14.9 bc	1.22 cd	0.11 cd	51.0 d
Fortuna	5.8 a	5.2 b	17.3 de	17.3 de	0.65 de	0.34 a	21.2 d
Frontana	10.9 e	0.7 d	12.4 ef	17.9 ef	0.93 d	0.16 bcd	42.7 d
HY320	8.8 cd	1.8 cd	23.1 d	16.3 cd	2.45 bc	0.14 cd	37.1 d
Lacos	11.2 e	0.8 d	5.0 f	18.9 fg	0.31 e	0.12 cd	14.1 d
Du75	7.4 bc	1.2 cd	2.6 f	20.7 h	0.10 f	0.02 d	26.0 d
French Peace	8.0 bc	1.3 cd	4.1 f	19.9 gh	1.06 cd	0.12 cd	7.0 d

[a]INC = incubation period (days), DLA1 = diseased leaf area (%) 11 days after inoculation, DLA2 = diseased leaf area (%) 18 days after inoculation, LP = latent period (days), TSP = total spore production (million spores per leaf), LER = lesion expansion rate (regression of $\log_e$(diseased proportion/(1 - diseased proportion))/time and AUDPC = area under the disease progress curve (% disease x days).

[b]Means within a column followed by the same letter are not significantly different at P = 0.05, according to Duncan's new multiple range test.

Table 5.3. Simple correlations among components of resistance to septoria tritici blotch measured in Experiments I or II

	DLA1[a]	DLA2	LP	TSP	LER	AUDPC
INC	-0.54	-0.19	0.22	-0.13	-0.27	-0.18
DLA1		0.86**	-0.81**	0.84**	0.78**	0.81**
DLA2			-0.94**	0.97**	0.84**	0.93**
LP				0.86**	-0.83**	-0.94**
TSP					0.79**	0.87**
LER						0.87**

** Significant at P = 0.01.

[a]DLA1 = diseased leaf area (%) 11 days after inoculation, DLA2 = diseased leaf area (%) 18 days after inoculation, LP = latent period, TSP = total spore production, LER = lesion expansion rate, AUDPC = area under the disease progress curve and INC = incubation period.

correlation between INC and other measures was probably due to physiologic leaf spotting on cultivars that were rated resistant by other measures. An example of this is the triticale cultivar Du75, which had a low value for INC but otherwise was highly resistant (Table 5.2).

Some of the correlations might be expected because of obvious relationships among the variables. It is not surprising that DLA1 is positively correlated with DLA2 and AUDPC (Table 5.3). Likewise, TSP is highly correlated with measures of diseased leaf area. It would require large differences in size or density of pycnidia to cause a non-significant or negative correlation between TSP and diseased leaf area. To make spore production independent of total diseased area, it was expressed as spore production per cm^2 diseased leaf area in subsequent experiments.

LP is negatively correlated with AUDPC (Table 5.3), but a short latent period is not a direct cause of high AUDPC values in these experiments as it would be in a field experiment. Here it is assumed that spores produced at the end of the latent period do not cause new infections and therefore do not contribute to an increase in disease measures such as AUDPC. It is more likely that LP and disease measures such as DLA1, DLA2 and AUDPC are correlated because they all depend on the ability of the pathogen to spread through the host.

Despite the generally high correlations among components, there were some exceptions. A significant positive correlation existed between TSP and LER (Table 5.3). However, comparison of Fortuna and Park showed that the LER for Fortuna is as high as the LER for Park, but the TSP value for Fortuna is much lower than that of Park (Table 5.2). Eyal et al. (1985) reported that Fortuna may develop large necrotic areas containing few pycnidia and other lines that show necrosis but reduced pycnidial coverage have been noted (Rosielle, 1972; Shaner and Finney, 1982). *Septoria tritici* is known to produce a toxin (Malcolm, 1978). It is possible that host genotypes which restrict hyphal growth, but are sensitive to the toxin, may have extensive necrosis with relatively few pycnidia.

The close association among resistance components in this study contrasts with the relatively weak association of components of resistance to *S. nodorum* reported by Jeger et al. (1983) and Lancashire and Jones (1985). These studies suggested that different cultivars often exhibit different resistance components. In both *S. nodorum* studies, principal components analysis suggested that four different principal components were important in determining disease reaction. Lancashire and Jones (1985) felt this may represent four different physiologic processes involved in resistance. These could be combined by breeding or through cultivar mixtures. In addition, Jeger et al. (1981) found that disease severity could be reduced by mixing a cultivar that exhibited low infection frequency with a cultivar that caused reduced sporulation.

In contrast, this study indicates that important components of resistance to *S. tritici* are often combined in single cultivars. There was little evidence that different physiologic processes were involved. The possible exception was cultivars that had large lesions but restricted pathogen reproduction.

The different results of the *S. nodorum* and these studies may be due to differences in choice of experimental material. Jeger et al. (1983) used 41 randomly chosen host genotypes and Lancashire and Jones (1985) used 10 cultivars that possessed similar field ratings to *S. nodorum*. In this study several cultivars were known to be either resistant or susceptible. This may have increased the probability that resistance components would be associated. However, no prior detailed observations on resistance components had been made on these cultivars.

This study also provided a comparison of different measures of disease severity that could be used in screening for disease resistance. The usual procedure has been to assess disease severity approximately 17 days after inoculation, a measure corresponding closely to the DLA2 measurement in Experiment I. The high correlations between DLA2 and other disease measures (Table 5.3) indicates that DLA2 is a good measure of disease severity, and it is much easier to measure than LP, TSP, LER or AUDPC.

5.2.2 Experiments III to VI

The components studied in these experiments were chosen because they are likely to be important determinants of epidemic severity, and they are mathematically, although not necessarily biologically, independent.

A separate analysis of variance was done for each experiment because of heterogeneous error variances among experiments (Tables A.5, A.6). Since each experiment was conducted on only one growth stage, and substantial differences between experiments existed, no attempt was made to quantitatively measure the effect of growth stage on the components.

The shortest latent period (LP) on each cultivar was recorded in an experiment conducted at the three-leaf stage (Experiment III). However, there was no consistent growth stage effect on LP, as values in Experiment IV were not shorter than those in experiments conducted at the flag leaf stage (Table 5.4).

Cultivars Benito and Park had the shortest LP values in all experiments (Table 5.4). The LP for Benito was significantly shorter than that for Park in those experiments performed at the flag leaf stage. Lacos had the longest LP in every experiment and, on average, the LP for Lacos was twice as long as that for Benito. This difference in LP is much larger than that commonly reported by investigators working with "slow-rusting" wheats (Shaner et al., 1978). Since a slightly longer latent period is considered to be an important component of partial resistance to leaf rust, the differences in LP recorded in these experiments should have a considerable impact on an epidemic development. This is particularly likely if many infection cycles occur during the growing season.

Brokenshire (1976) reported a similar range in magnitude of LP (13.0 to 25.0 days) on seedlings and flag leaves of 29 wheat cultivars inoculated with *S. tritici*.

Benito and Park had significantly higher infection frequency (INF) values than the other cultivars, except in Experiment III, where they were not significantly different from Fortuna (Table 5.5). INF on Lacos was significantly lower than that on other cultivars,

Table 5.4. Mean latent period (days) for wheat cultivars inoculated with *Septoria tritici* at two growth stages in controlled environment studies

	Growth stage and experiment number				
	Three-leaf stage		Flag leaf stage		
Cultivar	III	IV	V	VI	Mean
Benito	11.4 a[a]	15.0 a	15.5 a	12.3 a	13.6
Park	11.6 a	16.5 a	18.6 b	14.9 b	15.4
Fortuna	13.5 b	22.9 b	24.2 c	18.0 c	19.7
HY320	14.3 b	25.3 c	28.3 d	23.1 e	22.8
French Peace	18.4 c	24.4 bc	25.8 c	20.4 d	22.3
Lacos	20.7 d	26.2 c	30.7 d	31.8 f	27.4

[a]Means followed by the same letter within a column are not significantly different at P = 0.05, according to Duncan's new multiple range test.

Table 5.5. Mean infection frequencies (lesions/spores applied) x (10^4) on wheat cultivars inoculated with *Septoria tritici* at two growth stages in controlled environment studies

	Growth stage and experiment number				
	Three-leaf stage		Flag leaf stage		
Cultivar	III	IV	V	VI	Mean
Benito	6.0 a[a]	5.8 a	5.4 a	6.0 a	5.8
Park	5.0 a	5.4 a	5.4 a	5.6 a	5.3
Fortuna	3.7 ab	2.5 b	1.3 c	2.0 b	2.4
HY320	2.7 b	1.7 c	1.9 b	1.6 b	2.0
French Peace	2.1 bc	1.7 c	1.0 c	1.7 b	1.6
Lacos	1.3 c	1.2 d	0.5 d	0.4 c	0.9

[a]Means followed by the same letter within a column are not significantly different at P = 0.05, according to Duncan's new multiple range test.

except in Experiment III, where it was not significantly different from French Peace.

Averaged over all experiments, INF ranged from 0.87 x 10^{-4} on Lacos to 5.80 x 10^{-4} on Benito (Table 5.5). Even on a susceptible cultivar under favourable conditions for infection, over 1,000 spores were applied for every lesion that was counted. Since several infection sites may combine to form a lesion, INF may be underestimated because counting lesions may underestimate the number of infection sites. It is also possible that pycnidiospores produced under natural conditions have a greater infection and survival ability than spores produced in culture.

Lancashire and Jones (1985) reported a range for INF of 4.8 x 10^{-4} to 13.9 x 10^{-4} for *S. nodorum* on wheat cultivars with partial resistance. The INF values for *S. tritici* on Benito and Park were within this range (Table 5.5). The mean INF value for Lacos was 0.87 x 10^{-4}. Probably no cultivar studied by Lancashire and Jones (1985) had resistance to *S. nodorum* that was as effective as the resistance of Lacos to *S. tritici*.

The number of successful leaf penetrations was assessed indirectly by counting lesions. Before a lesion becomes visible, some pathogen growth within the host must take place. Therefore, this measure of INF includes the ability of the pathogen to become established in the host as well as the ability of a spore to germinate and penetrate the leaf. An association between INF and LER might be expected. Hilu and Bever (1957) found that there was no difference between a resistant and a susceptible cultivar in the number of successful leaf penetrations, but fewer hyphae became established in the resistant cultivar.

Differences in mean spore production (SP) among cultivars appeared to be due to differences in density or size of pycnidia. Benito and Park had the highest SP values in tests at the three-leaf stage (Table 5.6). Lacos had significantly lower SP values than all other cultivars at the flag leaf stage, while the SP values for French Peace, HY320 and Fortuna were not significantly lower than those of Park.

The difference in sporulation between Fortuna and Park in Experiment I (Table 5.2)

Table 5.6. Mean spore production (thousand spores per cm^2 diseased leaf area) for wheat cultivars inoculated with *Septoria tritici* at two growth stages in controlled environment studies

	Growth stage and experiment number				
	Three-leaf stage		Flag leaf stage		
Cultivar	III	IV	V	VI	Mean
Benito	2871 a[a]	1011 b	2276 a	1907 a	2016
Park	2309 a	1790 a	1640 b	1318 b	1764
Fortuna	952 b	618 c	1762 ab	1374 b	1177
HY320	1040 b	555 c	1492 b	1294 b	1095
French Peace	1087 b	464 c	1629 b	939 b	1030
Lacos	899 b	312 c	913 c	277 c	600

[a]Means followed by the same letter within a column are not significantly different at P = 0.05, according to Duncan's new multiple range test.

was evident only in experiments carried out at the three-leaf stage (Table 5.6). Because the upper leaves did not become necrotic as quickly, experiments at the flag leaf stage were almost twice the duration of experiments at the three-leaf stage. This may have allowed enough time for pycnidia to develop on cultivars Fortuna, HY320 and French Peace, thus reducing the difference in SP values between these cultivars and the susceptible cultivars Park and Benito. SP values of Lacos appeared to be unaffected by growth stage.

SP values were based on estimates of leaf area, percent disease and number of spores produced. Errors associated with these estimates caused coefficient of variation values to be large (36-50%).

Gough (1978) studied spore production of *S. tritici* by estimating the number of spores produced per pycnidium. Combining his estimates of pycnidial density with the number of spores per pycnidium provides an estimated SP of 320,000 per cm^2 for the resistant cultivar Oasis and an estimated SP of 1,833,000 spores per cm^2 for the susceptible cultivar Improved Triumph. The estimated SP for Oasis is within the range of the estimates for Lacos, while the estimate for Improved Triumph is within the range calculated for the susceptible cultivar Benito (Table 5.6). Thus the range of SP values was similar, even though the methods used to calculate them were quite different.

For nearly all cultivars the highest lesion expansion rates (LER) occurred in experiments conducted at the three- leaf stage. Fortuna was the exception with a higher LER in Experiment VI than in Experiment IV. The very high LER rates recorded in Experiment III may have been due to the spray inoculation method used.

LER on cultivar Lacos was significantly lower than that on Park and Benito in all experiments (Table 5.7). There were no other consistent significant differences among cultivars, although LER on HY320 and French Peace was lower than that on Benito and Park in all experiments. As noted for the SP data, coefficient of variation values for LER were quite high (20-33%).

Table 5.7. Mean lesion expansion rate[a] for wheat cultivars inoculated with *Septoria tritici* at two growth stages in controlled environment studies

	Growth stage and experiment number				
	Three-leaf stage		Flag leaf stage		
Cultivar	III	IV	V	VI	Mean
Benito	0.49 a[b]	0.35 a	0.12 b	0.23 ab	0.30
Park	0.47 a	0.28 ab	0.20 a	0.22 ab	0.30
Fortuna	0.47 a	0.21 bc	0.09 b	0.26 a	0.26
HY320	0.44 a	0.20 c	0.10 b	0.16 c	0.22
French Peace	0.28 b	0.18 c	0.10 b	0.17 bc	0.18
Lacos	0.20 b	0.18 c	0.05 c	0.12 c	0.14

[a]Calculated as the regression coeffient of $\log_e$(proportion diseased/(1 - proportion diseased))/days after inoculation.
[b]Means followed by the same letter within a column are not significantly different at P = 0.05, according to Duncan's new multiple range test.

Whereas some diseases increase over time almost entirely through increase in lesion number, lesion expansion can also be an important component of disease increase (Berger and Jones, 1985). Morrall and Verma (1981) stated that lesion expansion could cause a pathogen with a monocyclic disease cycle to exhibit logistic disease progress. In these monocyclic infection experiments with *S. tritici*, it has been found that disease increase due to lesion expansion was best described by a logistic equation.

A biological rationale can be found for the logistic increase of disease due to expansion of lesions on a single leaf. According to the logistic equation (5.3), absolute increase in disease (dy/dt) is determined by three terms: y, k and 1 - y, where y = the proportion diseased and k = the intrinsic rate of increase.

$$dy/dt = y*k*(1 - y) \qquad (5.3)$$

If it is assumed that y reflects the number of hyphae in the leaf, k is the growth rate of the hyphae and 1 - y reflects the limit to hyphal growth imposed by limited host tissue, then lesion area will increase logistically as the number of hyphae increase at an intrinsic rate, with the increase being limited by the amount of host tissue available.

In most cases the resistance components studied were significantly correlated (Table 5.8). The only exception was the correlation between LER and SP, which was non-significant in all four experiments. For these two components, Lacos consistently had the lowest and Park and Benito had the highest values. The other cultivars showed variable values within and among experiments (Tables 5.6, 5.7). For example, Fortuna had relatively high LER and low SP values in Experiments III and IV but not in Experiments V or VI. The relatively low correlation between these two components may be due to an actual disassociation between the components, but also could reflect the high error involved in measuring these variables.

Association of resistance components in a single cultivar makes it an attractive source of resistance for a breeding program. Such a cultivar is Lacos, which had the most desirable mean value for all components. To a lesser extent similar associations occurred

Table 5.8. Simple correlations among components of resistance to septoria tritici blotch measured on wheat cultivars in Experiments III to VI

	Experiment number	SP[a]	INF	LER
LP	III	-0.71	-0.97**	-0.98**
	IV	-0.86*	-0.90*	-0.70
	V	-0.83*	-0.99**	-0.97**
	VI	-0.91*	-0.98**	-0.84*
SP	III		0.81*	0.58
	IV		0.75	0.43
	V		0.86*	0.69
	VI		0.89*	0.76
INF	III			0.93**
	IV			0.82*
	V			0.94**
	VI			0.74

*,** Significant at P = 0.05 and 0.01, respectively.
[a]SP = spore production, LP = latent period, INF = infection frequency and LER = lesion expansion rate.

also in French Peace and HY320. Even Fortuna, which is used as a susceptible check in Montana (Eyal et al., 1985), appears to have a favourable combination of resistance components relative to Benito and Park.

Association among resistance components may be due to a single physiologic process that affects them all. Retarding hyphal growth could cause a lower LER, a longer LP, fewer and smaller pycnidia and slower appearance of lesions. Hilu and Bever (1957) observed that the difference between a resistant and a susceptible cultivar was that hyphae grew more slowly in the resistant cultivar, with few hyphae becoming established and fewer and smaller pycnidia being formed.

The mean value of each resistance component for each cultivar, relative to that of Benito, is presented in Table 5.9. The cultivar ranking is the same for all resistance components, except for the higher relative LP value of HY320 compared to that of French Peace. Park has mean values for INF and LER that are almost equal to those of Benito. These two components are the ones that determine the diseased leaf area in a monocyclic experiment. Park has a longer LP and lower SP than Benito. This should reduce disease severity on Park relative to Benito in the field. Differences in disease progress due to differences in LP and SP will be greatest if there are several infection cycles. It is not known how many infection cycles of *S. tritici* occur during the growing season in Saskatchewan.

The other cultivars showed substantial differences relative to Benito for all resistance components (Table 5.9). These experiments do not indicate whether the effects of the resistance components are equal or how the components will interact to reduce an epidemic. Monocyclic infection experiments are useful in measuring components of resistance, but the effects of the components on an epidemic in the field are difficult to predict, since the components will interact and their effects will be cumulative (Shaner and Hess, 1978).

The relative importance of the resistance components in determining the severity of

Table 5.9. Mean value of components of resistance to septoria tritici blotch relative to those of cultivar Benito

Cultivar	INF[a]	LP	SP	LER
Benito	1.00	1.00	1.00	1.00
Park	0.92	1.13	0.88	0.99
Fortuna	0.41	1.45	0.58	0.87
HY320	0.34	1.68	0.54	0.75
French Peace	0.28	1.64	0.51	0.62
Lacos	0.15	2.01	0.30	0.47

[a]INF = infection frequency, LP = latent period, SP = spore production and LER = lesion expansion rate.

an epidemic will depend partially on the number of infection cycles. To determine the relative importance of the components of resistance in field experiments, lines which are similar in maturity and growth habit, but which have different combinations of resistance components, must be available (Shaner and Hess, 1978). Lines that fit these requirements have not been found in these experiments.

The effects of resistance components can also be assessed by developing equations that integrate the components of resistance (Shaner and Hess, 1978) or by the use of simulation models (Rapilly et al., 1977). The measurements taken in these experiments may provide useful information in developing equations or a simulation model. A simple model, which combines estimates of resistance components to give a prediction of the relative severity of septoria tritici blotch in the field would be useful. At the present time such a model would be speculative, since field data adequate for model testing are lacking.

6.0 Association of resistance with agronomic traits

6.1 Materials and methods

6.1.1 Plant material

Populations derived from crosses between resistant and susceptible cultivars were used to test for association of resistance with agronomic traits. All resistant cultivars possess traits that are undesirable in a hard red spring wheat grown in Saskatchewan. Cultivars French Peace and Frontana are tall and late maturing, Lacos is also late maturing and Oasis has a winter growth habit.

F2 populations of each cross were grown and tested for reaction to *S. tritici* as described in Section 3.1. F3 populations of crosses involving French Peace and Frontana, were tested in a growth room experiment. After rating for disease reaction, plants that were used to produce seed for field experiments were transferred to 10 cm square pots and grown to maturity. To test for association between winter habit and resistance in the Oasis x Park cross, plants of the F1, F2 and parental generations were grown to the heading stage. No plants were vernalized.

To test for association of resistance with heading time and plant height, populations from crosses involving French Peace, Frontana and Lacos were grown at the North Seed Farm, University of Saskatchewan, Saskatoon in 1987 and 1988.

In 1987, F4 families of crosses French Peace x Park and Frontana x Park and F3 families of Lacos x Conway were seeded in 1.8 m rows with 30 cm between rows. Every third row was planted to the susceptible cultivar Kenyon. Within a population, families and parental checks were completely randomized. A single plant from each of approximately 50 rows of each population was harvested to provide seed for the 1988 experiment. In 1988 two rows of each F4 or F5 family of the three crosses and the parental checks were seeded in a completely randomized design with Kenyon planted in every third row.

In 1987 the French Peace x Park F4 population was grown on barley stubble, while

the other populations were grown on wheat stubble. In 1988 all populations were grown on fallow. Water was supplied by a mist irrigation system. Plant populations were grown between pipes 21 m apart in 1987 and 18 m apart in 1988. Sprinklers were staggered on the two pipes and were approximately 21 m apart on each pipe.

6.1.2 Pathogen culture and inoculation

Procedures used in the growth room were as described in Section 3.1.2.

For inoculations in the field in 1987, liquid cultures of isolate Sask2 were prepared as described in Section 3.1.2. When cultures reached a spore concentration of approximately 1.0×10^6 mL^{-1}, 100 mL of liquid culture was poured into 4.5 L bottles containing 3 L of liquid yeast-malt medium. These bottles were kept on a lab bench and shaken by hand twice daily for 4 to 8 days.

In 1988 inoculum was produced by flooding yeast-malt agar plates with 0.75 mL of a liquid culture of *S. tritici*. After approximately five days, spores were removed from the plates by washing with water and rubbing gently. Enough spores were produced on two plates to produce 1 L of inoculum with a spore concentration of 1.0×10^6 mL^{-1}.

Approximately 45 L of inoculum were prepared for each inoculation. Inoculum was applied using a hand-held compressed air sprayer. Inoculations were carried out in the evening or in the afternoon if the sky was cloudy on days when the weather forecast was favourable i.e. cloudy, cool, rainy weather predicted. All plants in both years were inoculated twice, the first inoculation was about 10 days before heading of the earliest lines and the second when heading of all lines was nearly complete.

Plots were irrigated intermittently during the night for at least three nights following an inoculation.

6.1.3 Rating and analysis

Rating for septoria tritici blotch severity on plants in the growth room was as

described in Section 3.1.2.

Rating for septoria tritici blotch in the field was done approximately two weeks after anthesis on a row basis. A 0 to 5 scale was used, where 0 represents no disease symptoms and 5 represents extensive damage of upper leaves caused by *S. tritici*. In 1987 only the French Peace x Park F4 population was rated for septoria tritici blotch. Extensive septoria nodorum blotch on the other populations made rating for septoria tritici blotch unreliable.

Heading date was considered to be the day on which one-half of the heads on primary tillers in a row were fully emerged. Height was recorded as the average height of plants in a row, excluding awns, recorded approximately 10 days after anthesis was complete.

The growing seasons of 1987 and 1988 were hot and dry, particularly in the early part of the season. Inoculation and irrigation ensured disease development, but uneven irrigation patterns caused marked differences in plant growth throughout the experimental area. To reduce these effects, plant height and days until heading were adjusted by subtracting the corresponding value of the neighbouring check row.

Correlation analysis was based on individual plant values for the growth room data, single row values in the 1987 field experiments and family mean values in the 1988 experiments. Analysis of variance was conducted on the 1988 data.

To test for association between resistance and growth habit in the Oasis x Park cross, plants were classified as winter type or spring type according to their heading date. All plants including the winter wheat parent eventually headed, but some not until up to 190 days after seeding. F1 generation plants had heading times similar to those of the spring wheat parent, with a maximum of 60 days until heading. Therefore F2 plants that headed more than 60 days after seeding were classified as winter types. Seedling classification for disease reaction was as described in Section 3.1.3. Association between resistance and growth habit was tested using a contingency chi-square analysis.

6.2 Results and discussion

The results of a chi-square test for association between resistance and winter growth habit in the cross Oasis x Park are presented in Table 6.1. Column and row totals were used to calculate the expected values in this test. Although there were fewer resistant spring types and fewer susceptible winter types than expected, the test did not indicate that growth habit and reaction to septoria tritici blotch were associated. There may be a trend towards association between these traits in this cross, but it was not strong enough to prevent identification of resistant spring types.

Analysis of variance of 1988 field data indicated that there were significant differences among families for all traits measured (Table A.6).

Correlations among disease severity and agronomic measurements for the Lacos x Conway cross are presented in Table 6.2. The simple and partial correlations between growth room and field disease ratings were highly significant. No other correlations were significant except for heading time in the F3 and F4 generations. Negative associations were observed between disease severity and late heading, but none were significant. Correlations between disease severity and plant height were very low. Tall plant height is often associated with low disease severity (Bahat et al., 1980), but in this case the resistant parent was the shortest. Thus, tallness was more likely to have been associated with high disease severity. In this cross, the low correlations between resistance and agronomic traits suggested that under the conditions of these experiments, heading time and plant height have little or no effect on disease severity.

Correlations between measurements of a trait on parents and their offspring in two different environments is an indication of the heritability of the trait (Frey and Horner, 1957). The correlation between the F2 rating for septoria tritici blotch and the rating on the derived F4 families (0.55) is similar in magnitude to the heritability estimate reported earlier (Table 3.21).

Correlations among disease ratings and agronomic traits for the Frontana x Park cross

Table 6.1. Contingency test for association between growth habit and reaction to *Septoria tritici* in F2 population of Oasis x Park tested under controlled environmental conditions

Disease reaction		Growth habit		
		Spring	Winter	Total
Resistant	Observed	85	42	127
	Expected	89.2	37.8	
	X^2	0.20	0.47	
Susceptible	Observed	33	8	41
	Expected	28.8	12.2	
	X^2	0.61	1.45	
	Total	118	50	168
	Total X^2(1df) = 2.73		P = 0.05 - 0.10	

Table 6.2. Simple[a] and partial[b] correlations among septoria tritici blotch severity, days until heading and plant height in populations derived from the cross Lacos x Conway

Trait	Number	(1)	(2)	(3)	(4)	(5)
Stb[c] F2 (1)	51		.55**	-.11	-.05	-.13
Stb F4 (2)	51	$.54_3$**		-.23	.01	-.19
Head F4 (3)	51		$-.25_4$		.31*	.25
Height F4 (4)	51		$.09_3$			.11
Head F3 (5)	51					

*,** Significant at P = 0.05 and 0.01 respectively.
[a]Simple correlations are above the diagonal.
[b]Partial correlations are below the diagonal. The subscript indicates the variable that was held constant in the partial correlation.
[c]Septoria tritici blotch severity rating.

are presented in Table 6.3. Growth room disease rating of F3 plants was correlated with disease severity on F5 families in the field, but not with plant height or days to heading. Disease severity on F5 families was negatively correlated with late heading and tallness in the F4 generation, but only the correlation with heading was significant in the F5 generation. A stronger relationship with heading time than with height was evident when partial correlations were considered. The correlation between disease severity and heading (height held constant) was highly significant, while the correlation between disease severity and height (heading held constant) was low. Correlations among heading time and height were positive and significant in both F4 and F5 populations.

The correlation between disease severity of F3 plants in growth room tests and of F5 families in the field (Table 6.3) is similar to the heritability estimate obtained previously (Table 3.21).

A negative correlation was observed between disease severity in the field and days to heading, but not between disease severity in the growth room and days to heading (Table 6.3). This suggests that linkage between genes controlling the two traits is not the primary reason for the association, since linkage would cause disease severity measured in the growth room to be correlated also with heading.

Correlations among disease severity ratings and agronomic traits for the French Peace x Park cross are presented in Table 6.4. Disease severity in the growth room was correlated with disease severity in the field, but not with days to heading or with plant height. Simple correlations of disease severity of F4 families with heading time and with height were significant, but the corresponding partial correlations were not. There was a significant negative association between disease severity and days to heading among F5 families. Although the simple correlation was not significant, there was a significant positive partial correlation between disease severity and plant height (heading time held constant) in the F5 population of this cross.

The maximum association observed between septoria tritici blotch and an agronomic

Table 6.3. Simple[a] and partial[b] correlations among septoria tritici blotch severity, days until heading and plant height in populations derived from the cross Frontana x Park

Trait	Number	(1)	(2)	(3)	(4)	(5)	(6)
Stb[c] F3 (1)	147		.39**	-.06	-.01	-.12	.05
Stb F5 (2)	50	$.41_3$**		-.47**	-.25	-.35*	-.34*
Head F5 (3)	50		$-.42_4$**		.36*	.70**	.49**
Height F5 (4)	50		$-.11_3$			.39**	.36*
Head F4 (5)	145						.69**
Height F4 (6)	147						

*,** Significant at P = 0.05 and 0.01, respectively.
[a]Simple correlations are above the diagonal.
[b]Partial correlations are below the diagonal. The subscript indicates the variable that was held constant in the partial correlation.
[c]Septoria tritici blotch severity rating.

Table 6.4. Simple[a] and partial[b] correlations among septoria tritici blotch severity, days until heading and plant height in populations derived from the cross French Peace x Park

Trait	Number	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Stb[c] F3 (1)	176		.44**	.30*	-.10	-.15	-.01	.04
Stb F4 (2)	167			.50**	-.18	.09	-.23**	-.21**
Stb F5 (3)	46	$.32_4$*	$.48_4$**		-.39**	.06	.07	.06
Head F5 (4)	46			$-.51_5$**		.55**	.63**	.41**
Height F5 (5)	46			$.36_4$**			.26	.26
Head F4 (6)	142		$-.16_7$					.55**
Height F4 (7)	167		$-.11_6$					

*,** Significant at P = 0.05 and 0.01, respectively.
[a]Simple correlations are above the diagonal.
[b]Partial correlations are below the diagonal. The subscript indicates the variable that was held constant in the partial correlation.
[c]Septoria tritici blotch severity rating.

trait in any cross was the correlation of -0.47 between disease severity and days to heading in the Frontana x Park F5 population. Although this value was highly significant, it indicates that heading time accounts for less than 25% of the variation in septoria tritici blotch rating. There is substantial variation in disease severity that is independent of heading time.

Association between traits in these experiments may be due to pleiotropy, linkage or environmental correlations. Differences in environment within an experiment (such as the amount of irrigation water received) could have similar or opposite effects on the traits measured. Making the measurements relative to those on the nearest check should reduce the effect of environment, but it will not eliminate it. Error mean squares and mean cross-products were used to calculate environmental correlations between traits in the 1988 field experiments. None of these were significant (P = 0.05), but the highest environmental correlation was a positive correlation of 0.26 between disease severity and plant height in the French Peace x Park F5 population. A possible explanation for this is that rows which received more water grew taller and had greater disease development. This may have been the cause of the positive partial correlation between these traits (Table 6.4).

Interpretation of association between disease severity and agronomic traits depends on the experimental material and the way in which data are gathered. Scott et al. (1982) suggested that association between septoria nodorum blotch and plant height may be due to chance (as when cultivar means are used in correlation analysis), or due to pleiotropy or linkage. Possible reasons for pleiotropy between tallness and resistance to septoria nodorum blotch are (Scott et al., 1985):

1) there are greater distances between leaves, thus hindering spore dispersal;

2) tall plants create a less favourable micro-climate for disease,

3) short plants may be earlier maturing and more susceptible at any given time; and

4) long straw and resistance may both be influenced by genes that control plant vigour.

The experimental methods used in this study reduced or eliminated some of the possible causes of association between disease severity and height. Chance association should not have been a factor since segregating lines were studied rather than cultivars. Inoculation of plants by spraying from above eliminated the need for upward movement of spores and so there was no "ladder effect" to cause an association between disease severity and height. The use of single rows spaced 30 cm apart probably limited the differences due to micro-climate that may occur in large plots. Associations between height and disease severity in crosses involving French Peace and Frontana may have been reduced by the elimination of the ladder effect and by the reduction of micro-climate effects. The factors which affect association between traits are dependent upon the experimental circumstances and therefore the correlations found in this study relate only to selection for resistance under similar conditions.

Shaner et al. (1975) speculated that a positive association between early maturity and disease severity may be due to more favourable weather for disease development early in the season, linkage or a stronger expression of resistance genes in late-maturing wheat.

In all three crosses, association between disease severity and heading was generally stronger than association with height. As stated earlier, the lack of significant correlation between growth room disease rating and days until heading, suggests that the genes controlling the two traits are not linked. Danon et al. (1982) observed a mean correlation of -0.29 between severity of septoria tritici blotch and heading time in 13 crosses. They reasoned that this correlation must be due to linkage rather than pleiotropy since "if the same genes controlled both traits the correlation should be very high". Danon et al. (1982) did not seem to consider the possibility that a gene which influenced maturity might have an indirect, modifying effect on the expression of resistance. This would result in a moderate correlation between the traits.

There are several possible causes of pleiotropy between resistance and late maturity in these experiments. Plants were at slightly different growth stages when they were

inoculated and when they were rated for disease severity. Faster growing families would have had more leaves exposed at the time of the first inoculation and this could cause a greater proportion of the leaf surface to become diseased. In addition, early lines were close to the time of normal leaf senescence when they were rated for disease. Mistaking normal senescence for septoria tritici blotch, and possibly the partial breakdown of resistance as leaves begin to senesce, could also have resulted in an association between resistance and late heading.

From these experiments, it appears that selection for resistance in the growth room will be independent of the effects of height and maturity, but that, for some crosses, selection in the field may not be. Selection for resistance in the field without consideration of maturity or plant height may lead to a higher than desired proportion of tall and late-maturing lines. Selection for resistance in the field should be carried out on agronomically desirable lines. Selection for resistance to septoria tritici blotch in a short, early-maturing spring wheat genotype is possible as demonstrated by the cultivar Oslo, which combines all three attributes (Pedersen and Hughes, 1989).

7.0 General discussion

The main objective of this study was to provide information needed for breeding for resistance to *S. tritici*. Inheritance and heritability were studied to determine the genetic control of resistance in selected sources and the expected efficiency of selection for resistance in early generations.

When plants were categorized as resistant or susceptible, major gene control of resistance was found. Single genes with incomplete dominance were found in crosses involving Oasis, French Peace and Frontana, a single partially recessive gene was identified in Bunyip and two independent genes, one dominant and one recessive, conferred resistance in Lacos. The frequency distributions of disease ratings suggested that other genes were modifying the expression of resistance in all crosses, but not enough to prevent identification of the major gene effects. Allelism tests indicated that with the exception of the Frontana and Bunyip genes, the resistance genes identified are at different loci.

Simple genetic control of resistance suggests that transfer of resistance into adapted cultivars could be accomplished by backcrossing. Nevertheless, the possibility that minor genes are involved and the low to moderate heritability of resistance indicates that large population sizes and perhaps a generation of selfing between backcrosses may be necessary to ensure that the level of resistance of the resistant parent is maintained. Screening of breeding material under controlled environmental conditions where disease severity can be maximized should give the best results.

Although inheritance studies were conducted on seedlings in a growth room, it is believed that the resistance is also expressed on adult plants in the field. Correlations between disease ratings on seedlings and those on field-grown progeny were not substantially different from heritability estimates in tests where parents and progeny were both rated for disease reaction in growth room seedling tests. As well, there were few changes in cultivar ranking for resistance components between experiments

conducted at different growth stages. Brokenshire (1976) also reported good association between seedling and adult plant disease ratings.

Pathogenic variation was studied to determine if physiologic specialization occurs in the pathogen population. No clear differences among isolates from Saskatchewan in their ability to infect different cultivars were found, but there were differential reactions between isolates from Saskatchewan and those from England and Indiana. Where significant crossover interactions occurred, they appeared to result from the superior ability of foreign isolates to infect cultivars resistant to Saskatchewan isolates, while the Saskatchewan isolates sometimes had greater pathogenicity on cultivars grown in western Canada.

The differential reactions appeared to involve both major and minor genes affecting resistance. In some cases the difference in pathogenicity between two isolates on a particular cultivar would make the difference between that cultivar being classed as resistant or as susceptible. In other cases the difference in reactions, while statistically significant, only involved differences in degree of resistance or susceptibility. This suggests that the minor, modifying genes hypothesized in the inheritance study may be isolate-specific and that there are genes in the pathogen population which confer increased ability to infect particular cultivars.

In breeding for resistance to *S. tritici*, there must be an awareness of the possibility that the pathogen population might become more pathogenic on cultivars that are currently resistant. Because of this, strategies such as combining resistance genes from different sources in a single cultivar or use of several sources of resistance in a breeding program are desirable. This study suggests that several different resistance genes are available. Combining genes from different sources in a single cultivar may also serve to increase the overall level of resistance. However, despite the evidence from this study and several others (Eyal et al., 1973; Eyal and Levy 1987; van Silfhout et al., 1989) that physiologic specialization exists in *S. tritici*, the single gene resistance derived from

Bulgaria 88 has remained effective in Indiana since 1973 (Shaner and Buechley, 1989) and resistance in breeding lines in Australia has remained effective for 22 years (Ballantyne, 1989a). This suggests that some factor, such as limited dispersal ability of the pathogen, is acting to prevent or retard the replacement of non-pathogenic races with pathogenic ones.

The superior resistance of the triticale cultivar Du75, originally observed by Eyal et al. (1985), was noted also in this study. No isolate used in this study was able to reproduce on this cultivar.

The components of resistance to *S. tritici* were commonly found to be associated e.g. cultivar Lacos combines a long latent period with low infection frequency, low spore production and a low rate of lesion expansion. However, a relatively weak association was found between spore production and lesion expansion rate. The cultivar Fortuna exhibits relatively rapid spread of necrosis while restricting pathogen reproduction. This type of reaction has been noted by Rosielle (1972) and Shaner and Finney (1982). Shaner and Finney (1982) speculated that cultivars which restrict pathogen reproduction will perform better in the field than in greenhouse tests. This does not appear to be true for Fortuna, which is considered to be susceptible in Montana (Eyal et al., 1985).

This association of components of resistance to *S. tritici* eliminates the need to combine the components through breeding or cultivar mixtures, as has been suggested in the case of *S. nodorum* (Jeger et al., 1983).

On the basis that single genes with major effects determine resistance, it is concluded that one gene affects all the components of resistance. Although the exact mechanism is not known, Hilu and Bever (1957) noted a general slowing of hyphal growth in resistant cultivars. A single gene which has the effect of retarding hyphal growth would likely influence all of the components and thereby result in the observed association among the components.

Although French Peace and Lacos are both considered to be resistant to *S. tritici*,

measurements of components of resistance showed that Lacos was consistently superior. This could be attributed to unequal effects of their respective resistance genes, or by an epistatic effect between resistance genes in Lacos.

Association between resistance and agronomic traits was studied to determine the difficulty of incorporating resistance from tall, late-maturing or winter-type cultivars into spring wheat cultivars adapted to Saskatchewan conditions.

No association was found between resistance and agronomic traits when the resistance rating was based on seedling reaction in a growth room. However, some negative associations were found in crosses involving French Peace or Frontana when resistance rating was conducted in the field. The strongest associations were between resistance and late-heading, although they were probably not strong enough to prevent selection of early, resistant lines. Although Lacos heads later than either French Peace or Frontana, no association between late-heading and resistance was detected in the Lacos x Conway cross. This may be caused by the maintenance of resistance in Lacos as the plant ages, while resistance in French Peace and Frontana may deteriorate as the leaves begin to senesce. Spore production on French Peace was significantly higher than on Lacos at the flag leaf stage, but not at the three-leaf stage.

Several areas for further research are suggested by the results of this study.

The possibility of physiologic specialization in the pathogen deserves continued monitoring. More isolates should be tested and cultivars which may serve as good differentials should be identified. The possibility that the pathogen can adapt to a particular cultivar should also be tested using repeated inoculation and isolation from a resistant cultivar.

The high degree of resistance of Du75 makes it a desirable source of resistance and studies on the feasibility of transferring this resistance to common wheat are desirable. The clear expression of resistance in this cultivar makes it valuable for study of the mechanisms of resistance.

At present, the level of resistance needed to adequately suppress a septoria tritici blotch epidemic in Saskatchewan is unknown. More field data for septoria tritici blotch would be useful in determining if the moderate resistance exhibited by a cultivar such as HY320 in growth room tests is adequate under field conditions.

Given the low severity of septoria tritici blotch in field experiments in 1988 and 1989, it may be questioned whether breeding for resistance to *S. tritici* in wheat is a worthwhile objective. However, there are several reasons why this should remain an important breeding objective in Saskatchewan. A return to cooler weather with more moisture would most likely bring a resurgence of septoria tritici blotch. The use of fungicides to control leaf blotch diseases in Saskatchewan remains uneconomical, and while cultural control practices can reduce the effects of these diseases, they are not as effective as genetic resistance. As well, it is increasingly difficult to develop hard red spring wheat cultivars in western Canada that are higher yielding than the cultivars presently used, yet have the necessary quality. Incorporating resistance to septoria tritici blotch into a hard red spring wheat cultivar may be one way of maintaining the yield potential of a cultivar without sacrificing quality.

8.0 Conclusions

The main conclusions drawn from this study are:

1. Resistance to *S. tritici* in Oasis, French Peace, Frontana and Bunyip is controlled by single genes. Two independent genes control resistance in Lacos.
2. Genes with minor effects modify the expression of resistance.
3. With the exception of the genes in Frontana and Bunyip, resistance genes identified are at different loci.
4. Heritability of resistance is low to moderate.
5. Cultivar-specific differences exist in pathogenicity between isolates of *S. tritici* from Saskatchewan and those from other areas, but not among Saskatchewan isolates.
6. Genes which control both major and minor differences in pathogenicity between isolates are cultivar-specific.
7. The triticale cultivar Du75 is highly resistant to all isolates studied.
8. Significant differences for latent period, infection frequency, spore production and lesion expansion rate exist among cultivars.
9. Components of resistance are usually associated within cultivars.
10. There is no significant association of resistance measured in the growth room with winter growth habit, heading time or plant height.
11. In some crosses, resistance is associated with late heading and to a lesser extent with height, when disease rating is conducted in the field.

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APPENDIX TABLES

Table A.1. Analyses of variance of septoria tritici blotch ratings for the effect of cytoplasm in crosses between resistant and susceptible cultivars

Cross	Generation	Number[a]	Mean square (cytoplasm)
Oasis x Park	F3	50	0.82 ns[b]
Lacos x Conway	F1	45	0.27 ns
	F3	50	0.36 ns
French Peace x Park	F1	49	0.25 ns
	F3	46	0.96 ns
Frontana x Park	F1	48	0.84 ns
	F3	50	0.00 ns
Bunyip x Park	F1	43	0.52 ns
	F3	50	0.57 ns

[a]Number of F1 plants or F3 families, approximately equal number with each cytoplasm.
[b]Non-significant at P = 0.05.

Table A.2. Analysis of variance of lesion length (mm) and pycnidia per lesion (log10(x + 1)) in Experiment 1

Source of variation	Degrees of freedom	Mean square	
		Lesion length	Pycnidia
Cultivar (C)	7	174.53**	8.747**
Isolate (I)	9	36.21**	1.604**
C x I	63	6.51**	0.257**
Error	315	2.18	0.069

** Significant at P = 0.01.

Table A.3. Analysis of variance of lesion length (mm) and pycnidia per lesion (log10(x + 1)) in Experiment 2

Source of variation	Degrees of freedom	Mean square	
		Lesion length	Pycnidia
Cultivar (C)	7	198.76**	12.675**
Isolate (I)	9	26.86**	0.507**
C x I	63	6.98**	0.336**
Error	311	1.27	0.056

** Significant at P = 0.01.

Table A.4. Analyses of variance for resistance components measured in Experiments I and II

Source	Degrees of freedom	Mean square
Incubation period		
Cultivar	11	11.25**
Error	36	0.92
Diseased leaf area (Day 11)		
Cultivar	11	42.49**
Error	36	1.82
Diseased leaf area (Day 18)		
Cultivar	11	3763.9**
Error	36	47.3
Latent period		
Cultivar	11	43.04**
Error	36	1.09
Total spore production		
Cultivar	11	295.4**
Error	36	12.9
Rate of lesion expansion		
Cultivar	11	0.0419**
Error	12	0.0060
Area under disease progress curve		
Cultivar	11	104901**
Error	12	3037

** Significant at P = 0.01

Table A.5. Analyses of variance for components of resistance[a] to septoria tritici blotch measured in Experiments III to VI

Source	Degrees of freedom	Mean square		
		LP	SP	LER
Experiment III				
Cultivar	5	85.02**	4288306.3**	0.0857**
Error	30	1.90	589685.7	0.0058
Experiment IV				
Cultivar	5	181.68**	2350006.0**	0.0394**
Error	42	3.27	113984.3	0.0058
Experiment V				
Cultivar	5	267.75**	1549218.0**	0.0209**
Error	42	5.82	291976.7	0.0011
Experiment VI				
Cultivar	5	381.96**	2353939.7**	0.0212**
Error	42	1.78	185825.5	0.0032

** Significant at P = 0.01.
[a]LP = latent period, SP = spore production and LER = lesion expansion rate.

Table A.6. Analyses of variance for infection frequency of *Septoria tritici* on six wheat cultivars in Experiments III to VI

Source	Degrees of freedom	Mean square
Experiment III		
Cultivar	5	0.122**
Error	6	0.011
Experiment IV		
Cultivar	5	0.638**
Error	42	0.011
Experiment V		
Cultivar	5	1.300**
Error	42	0.015
Experiment VI		
Cultivar	5	1.489**
Error	42	0.018

** Significant at P = 0.01.

Table A.7. Analyses of variance for septoria tritici blotch severity, days until heading and plant height among families of three crosses grown at the North Seed Farm, Saskatoon in 1988

Source	Degrees of freedom	Mean square		
		Disease severity	Days to heading	Height
French Peace x Park				
F5 Family	45	3.30**	51.88**	57.64**
Error	46	0.52	3.92	14.63
Frontana x Park				
F5 Family	49	3.29**	56.31**	44.88**
Error	50	0.53	5.67	17.50
Lacos x Conway				
F4 Family	50	2.63**	14.77**	59.73**
Error	51	0.30	3.12	13.26

** Significant at P = 0.01.