AN ABSTRACT OF THE THESIS OF

Hafiz Uddin Ahmed for the degree of Doctor of Philosophy in Botany and Plant Pathology presented on November 1, 1993.

Title: Pathogenic Variability and Adaptation of *Septoria tritici* to Different Wheat Cultivars.

Redacted for Privacy

Abstract approved: _____
Stella M. Coakley

Redacted for Privacy

_____
Christopher C. Mundt

Experiments were done to determine pathogenic variability and pathogenic adaptation of *Septoria tritici* to different wheat cultivars. Fifteen *S. tritici* isolates from California, Oregon, and Texas were evaluated on seedlings of two sets of geographically diverse wheat cultivars under greenhouse conditions. Significant isolate effects, cultivar effects, and isolate X cultivar interactions were found, though the interaction terms were very small compared to the main effects of isolate and cultivar. All except one isolate were virulent to two sets of cultivars, and the virulence patterns varied among the isolate-cultivar combinations. Variability in virulence among the *S. tritici* isolates within and between locations also was observed. The isolates

were usually more virulent to the cultivars of the same geographic origin than cultivars of other locations, and this demonstrates location-specific adaptation of *S. tritici*.

Four populations of *S. tritici*, secured from four winter wheat cultivars grown in a field experiment, were evaluated on seedlings of the same wheat cultivars under greenhouse conditions. Significant spore population differences, cultivar differences, and spore population X cultivar interactions were obtained. The interaction term was fairly large compared to the main effect of spore population, and it is likely that the significant interaction was due to increased virulence of *S. tritici* to its own cultivar of origin. The "own" spore population (inoculation with spore population obtained from the same cultivar) produced significantly larger area under disease progress curve on the cultivar of origin than the "others" (inoculation with spore populations obtained from other cultivars), suggesting cultivar-specific adaptation of *S. tritici*. Spore populations obtained from the two susceptible cultivars indicated general adaptation to all of the test cultivars, whereas, the spore population secured from the most resistant cultivar showed specific adaptation to its cultivar of origin. Consequences of pathogenic variability, increased virulence, and adaptation of *S. tritici* to wheat cultivars are discussed.

Pathogenic Variability and Adaptation of *Septoria tritici* to Different Wheat Cultivars

by

Hafiz Uddin Ahmed

A THESIS

submitted to

Oregon State University

in partial fulfillment of
the requirements for the
degree of

Doctor of Philosophy

Completed November 1, 1993

Commencement June, 1994

APPROVED:

Redacted for Privacy

Co-Major Professor of Botany and Plant Pathology

Redacted for Privacy

Co-Major Professor of Botany and Plant Pathology

Redacted for Privacy

Head of Department of Botany and Plant Pathology

Redacted for Privacy

Dean of Graduate School

Date thesis is presented November 1, 1993

Thesis typed by Hafiz Uddin Ahmed for Hafiz Uddin Ahmed

TABLE OF CONTENTS

LIST OF TABLES

Pathogenic Variability and Adaptation of *Septoria tritici* to Different Wheat cultivars.

CHAPTER I

INTRODUCTION

To feed the ever growing human population, scientists and policy makers around the globe are focusing on all possible methods of increasing the world food supply. These methods include expansion of crop area, improved methods of cultivation, increased fertilization, use of improved cultivars of crops, increased irrigation, and improved crop protection (Agrios, 1988). Wheat is the number one food crop among the cereals, and feeds the majority of the world population. However, there are many constraints to growing wheat, including the Septoria tritici blotch (STB) disease.

Occurrence, distribution and economic importance of STB

Septoria tritici blotch of wheat (*Triticum aestivum* L.) is caused by the ascomycetous pseudothecial fungus *Mycosphaerella graminicola* (Fuckel) J. Schrot. in Cohn (anamorph= *Septoria tritici* Roberge in Desmaz.). It is a major wheat disease in all wheat growing areas of the world and causes severe yield loss (Coakley et al., 1985; Dubin, 1985; Eyal et al., 1973; Eyal et al., 1987; King et al., 1983; Schuh, 1990; Zelikovitch & Eyal, 1991). Yield losses

from slight to 60% have been attributed to natural infection (Camacho-Casas, 1986; Eyal, 1972; Eyal, 1981; Jenkins & Morgan, 1969; Shipton et al., 1971; Van Ginkel & Scharen, 1987; Ziv & Eyal, 1978). Annual average reduction in potential wheat yield due to STB and Septoria nodorum blotch (SNB) (caused by *Septoria nodorum* Miller) was estimated as 1% for the United States (Anonymous, 1965; Coakley, et al., 1985) and estimated in 1982 at about 9 million metric tons worldwide with a value of over $ 1 billion (Eyal et al., 1987; Scharen & Sanderson, 1985).

In the humid areas east of the Mississippi River in the U.S., the average constraint to yield due to STB is probably higher than in other parts of the U.S. (Coakley et al., 1985), and the importance of this disease has increased in Indiana (Coakley et al., 1985; Shaner & Finney, 1976). Septoria tritici blotch is also one of the most important limiting factors to wheat yield in California (Madariaga et al., 1989), western Oregon and Washington, and Texas (Personal communications with Warren Kronstad, Oregon State University and Bruce McDonald, Texas A & M University, respectively). Camacho-Casas (1986) reported that STB is an endemic foliar disease of high rainfall, wheat growing areas of the Pacific Northwest and mentioned that several severe epidemics were observed during the first half of the 1980's in the Willamette Valley of Oregon. Average yield loss

caused by this disease in five soft-white cultivars in the Willamette Valley was estimated at 24.1% in the absence of fungicide. Early reports during the 1940's and 1950's also indicate that STB outbreaks occurred in Missouri, Kansas, Washington, Oregon and Northern California, and sometimes in the Mid-Atlantic and Mid-West States (Shipton et al., 1971).

The disease severity of STB may have increased with the replacement of tall, late-maturing cultivars by short, early maturing cultivars grown at high nitrogen levels (Eyal, 1981; Shaner et al., 1975). Early maturity, short stature, high nitrogen levels, and changing husbandry practices apparently all favor STB (King et al., 1983; Narvaez, 1957; Shipton, 1966; Shipton et al., 1971). Septoria tritici blotch is a serious threat to wheat production in Mediterranean countries, where it dramatically gained importance with the introduction of nitrogen-responsive, high yielding semi-dwarf cultivars from Mexico (Eyal et al., 1973; Eyal, 1981; Saadaoui, 1987; Saari & Wilcoxson, 1974).

Although the increased severity of Septoria epidemics is often associated with semi-dwarf wheats, there is evidence that the introduction of the semi-dwarf wheats did not create the Septoria menace in the Mediterranean zone. The disease had been reported from Mediterranean countries many decades ago (Eyal et al., 1973; Eyal, 1981; Saadaoui,

1987; Saari & Wilcoxson, 1974) and it has been suggested that the semi-dwarf wheat merely drew attention to Septoria disease. Semi-dwarf wheat, unlike land races, are usually grown at higher densities with high amounts of fertilizer, both of which induce microclimates favoring Septoria epidemics (Fellows, 1962; Saadaoui, 1987; Saari & Wilcoxson, 1974).

Reports of increased occurrence and severity of STB have lead to a prominent place for *S. tritici* in a number of research and crop improvement programs worldwide (Bayles et al., 1985; Dubin & Rajaram, 1981; Krupinsky et al., 1977; Van Ginkel & Scharen, 1987). Severe epidemics in the late 1960's and early 1970's have caused the disease to be considered one of prime importance in national wheat improvement programs in several North African and Middle Eastern countries bordering the Mediterranean Sea (Djerbi et al., 1974; Saari, 1974; Stewart et al., 1972; Van Ginkel & Scharen, 1987).

Host resistance, pathogenicity, virulence and aggressiveness

Van der Plank (1968) proposed that there are two types of disease resistance. Vertical resistance, controlled by major genes, is characterized by differential interaction between cultivars of the host and races of the pathogen. In

horizontal or minor gene resistance there is no differential interaction. Further, Van der Plank (1968) proposed that there are two sorts of pathogenic races that do and do not interact differentially with cultivars of the host plants. He proposed that races which do not interact differentially with cultivars be said to vary in aggressiveness. Correspondingly, races which interact differentially with cultivars were said to vary in virulence (Van der Plank, 1968). Differentially interacting races are of concern in problems of resistance and breeding of resistant cultivars.

Analysis of variance is one way of testing for differential interaction. Different cultivars and different races may differ significantly from each other but there may be no significant interaction between cultivars and races. In this case, cultivars differ in resistance horizontally but not vertically, and the races differ in aggressiveness but not in virulence (Van der Plank, 1968). Van der Plank (1968) also proposed that if the order of ranking of disease severity for all isolate X cultivar combinations are the same, the host-pathogen interaction is not differential. Correspondingly, if they differ, host-pathogens are interacting differentially.

In contrast, Parlevliet and Zadoks (1977) provide an integrated concept of disease resistance which suggests that

all genes for true resistance in the host population, whether they are major or minor genes, are considered to interact in a gene-for-gene way (interaction model) with virulence genes, either major or minor, in the pathogen population. In support of this concept, differential interactions involving polygenic resistance have been found in some host-pathogen systems (Caten, 1974; Clifford & Clothier, 1974; Hamid et al., 1982; Parlevliet, 1977).

Other definitions regarding pathogen virulence have also been utilized. Agrios (1988) defines pathogenicity as the ability of a pathogen to cause disease, and he considered virulence to be the degree of ability of a pathogen to cause disease. Shaner et al., (1992) consider virulence as the relative disease-evoking capacity of an isolate compared to other isolates. In this thesis, I define pathogenicity as the ability of a pathogen to cause disease and virulence as the relative amount of disease produced by an isolate of a pathogen compared to other isolates.

Resistance to STB

The use of resistant cultivars is the most economical, easiest, safest, and most effective means of controlling plant diseases in crops. Cultivation of resistant cultivars not only reduces losses from diseases, but also eliminates expenses for sprays and other methods of disease control,

and avoids contamination of the environment with toxic chemicals. Cultivar resistance, chemical control, and suitable cultural practices are the major control measures for Septoria diseases. Breeding for resistance has been the most widely used method of disease control for the foliar diseases of wheat, and the Septoria diseases have been no exception (Nelson & Marshall, 1990). The most important objective of plant breeders and plant pathologists is to develop resistance that is stable over time, easy to transfer across genotypes, easy to identify in segregating generations, effective under disease conducive conditions, and nondetrimental to yield potential under disease-free conditions (Nelson & Marshall, 1990). Resistance to *S. tritici* has been reported by several researchers (Brokenshire, 1976; Camacho-Casas, 1989; CIMMYT, 1972; El Ahmed et al., 1984; Eyal, 1981; Eyal et al., 1987; Hilo & Bever, 1957; Krupinsky et al., 1977; Morales, 1957; Rosielle, 1972; Tyagi et al., 1970; Yechilevich-Auster et al., 1983).

Different modes of inheritance of resistance to *S. tritici* have been reported among common and durum wheats. These range from simple Mendelian inheritance to quantitative inheritance, with the genetic variation involving both nonadditive and additive gene action (Camacho-Casas, 1989; Van Ginkel & Scharen, 1987). It has

been reported that resistance to STB is controlled by a single dominant gene (Narvaez & Caldwell, 1957; Rillo & Caldwell, 1966; Rosielle & Brown, 1979), two additive genes (Narvaez & Caldwell, 1957), and at least three recessive genes (Rosielle & Brown, 1979).

Host range, *forma speciales* and physiologic specialization

The concept of host specialization was introduced by Eriksson (1894) when he demonstrated the existence of specialized forms (*formae speciales*) of *Puccinia graminis* Pers. on various hosts. Further specialization into physiologic races within a *forma speciales* was shown in 1916 (Stakman & Piemseisel, 1917). The rust infection types on wheat and other cereals are subject to environmental modification, but not to the same extent as *Septoria-Triticum* combinations (Shipton et al., 1971). *S. tritici* has been recorded on non-cereal hosts, but the evidence for existence of specialized forms is conflicting (Shipton et al., 1971). *S. nodorum* and *S. tritici* can colonize grasses, and *S. tritici* is more specialized than *S. nodorum* in terms of host range (Ao & Griffiths, 1976). A cross inoculation experiment was done to establish the existence of specialized forms in *S. tritici* and the results showed that the form occurring on *Triticum* species was host-specific and other forms must therefore be present to explain the wide host range of the pathogen (Shipton et al., 1971). Three

specialized forms, other than the one which occurs on wheat, have been documented. These forms are *S. tritici* f.sp. *avenae*, *S. tritici* f. sp. *holci* and *S. tritici* f. sp. *lolicola*, and are apparently restricted to species and subspecies of *Avena; Holcus;* and *Lolium multiflorum* and *L. perenne*, respectively (Shipton et al., 1971).

Most researchers of *S. tritici* have been unable to distinguish physiologic races, although the isolates differ in virulence, and pathogenicity tests on a range of resistant and susceptible wheat cultivars indicated that all of the isolates belonged to the same pathogenic races (King et al., 1983). Four isolates of *S. tritici* from various localities in the United States were examined on 16 wheat cultivars, but no evidence of physiologic specialization was demonstrated (Morales, 1958). Twenty-eight western Australian isolates were tested on five wheat cultivars, but no evidence of physiologic specialization was obtained (Shipton et al., 1971). However, in Israel, Morocco, and at other sites in the United States researchers found evidence of differential interactions with wheat cultivars, suggesting physiologic specialization in *S. tritici* wheat combinations in the true sense (Eyal et al., 1973; Eyal, 1981; King et al., 1983; Saadaoui, 1987).

Eyal et al. (1985) evaluated virulence patterns, in terms of ability to incite compatible disease reaction, for 97 isolates of *S. tritici* on seedlings of 35 wheat and triticale cultivars. Significant isolate X cultivar interactions indicated the existence of specific virulence genes among isolates, and the number of genes for resistance in the cultivars was estimated based on the assumption of a gene-for-gene relationship. Eyal et al. (1985) designated 28 hypothesized complementary genes in the 97-isolate X 35-cultivar matrix. On the basis of these hypothesized genes, the virulence frequencies and distribution of *S. tritici* genotypes were determined. Virulence frequencies varied considerably among regions and within countries. Latin America, represented by Mexico and Uruguay, had the highest overall virulence frequency. Isolates from Latin America were more virulent, having more compatible reactions with cultivars originating from Latin America. Isolates obtained from tetraploid wheats in Syria and Tunisia were more virulent to tetraploid cultivars than to hexaploid cultivars. Isolates from some areas, including Oregon in the United States, were virulent to a larger number of differentials. Four isolates secured from the Oregon State University Hyslop Farm were highly virulent, especially to the Russian winter wheat derivatives. They also stated that the differences in geographic distribution of virulences of *S. tritici* within regions and countries suggest the

feasibility of strategies for cultivar deployment for resistance to STB.

Marshall (1985) found marked variation in aggressiveness, measured as area under disease progress curves (AUDPC), of *S. tritici* populations collected from three locations in California and 13 locations in nine other States. *S. tritici* populations from central and north central California and northern regions of Indiana and Ohio were more aggressive, while those from northern Alabama, Mississippi, eastern Arkansas, southern Nebraska, and central Kansas were less aggressive. He also found greater variability in aggressiveness in the highly aggressive *S. tritici* populations than in the less aggressive ones.

Genetic variability among *S. tritici* populations

McDonald & Martinez (1990) used RFLP analysis on 93 isolates of *S. tritici,* using eight probes. Single locus and multilocus analysis of RFLP data indicated a high level of genetic variability within this population. It was suggested that the majority of genetic variation in *S. tritici* may be distributed on a local scale rather than on a macrogeographical scale. The presence of genetic variability among *S. tritici* populations would allow for rapid selection of clones that are fungicide-resistant or virulent to wheat cultivars that have pathotype-specific resistance. McDonald

& Martinez (1990) stated that deletion and insertion events are common in *S. tritici* nuclear DNA among geographically diverse groups of isolates and that genome rearrangements occur at a high frequency.

Although secondary cycles occur via splash dispersal of pycnidiospores, the ascospores of *M. graminicola*, are the airborne source of primary inoculum (Shaw & Royle, 1989). These have been shown to occur in wheat stubble in New Zealand (Sanderson, 1972), Victoria, Australia (Brown et al., 1978), Chile (Madariaga, 1986), the eastern UK (Scott et al., 1988), Brazil (Mehta, 1989); California, USA (Madariaga et al., 1989); the Netherlands (Shaw & Royle, 1989), and Oregon (personal observation ,1990). This sexual stage provides the opportunity for increased genetic variation within the *S. tritici* population.

Pathogen selection

Since there is evidence of genetic variability in populations of *S. tritici*, there is likely selection of host specific, virulent races adapted to the wheat cultivars grown over years in a certain location. Parlevliet (1981) suggested that the majority of our crops co-evolved with pathogens over a long span of time. Thus, mixtures of crop genotypes developed which were not exceptionally susceptible to locally occurring pests and diseases, allowing

coexistence of host and parasite. Resistance genes with large effect have often been incorporated into modern crop cultivars to control diseases. However, these resistance genes were only temporarily effective in many host-pathogen systems due to increase in the frequencies of pathogen genotypes with matching virulence genes to the introduced resistance genes (Parlevliet, 1981). For example, there is evidence that race 1 of *Xanthomonas oryzae* pv. *oryzae*, was predominant in the Philippines before the wide cultivation of improved, modern rice cultivars, and was virulent to rice cultivars with resistance genes *Xa*-10, *Xa*-11, *Xa*-14. When cultivars with the *Xa*-4 gene were introduced, race 1 declined but races 2 and 3, which can infect cultivars with the *Xa*-4 gene, appeared throughout the country (Mew et al., 1992).

If pathogen populations are variable, increased aggressiveness (an increase in the extent of disease in a compatible host) could be selected for over time (Alexander et al., 1985; Caten, 1974; James & Fry, 1983; Latin, et al., 1981) and hence it is likely that the durability of resistance will be reduced (Clifford & Clothier, 1974; Latin et al., 1981; Newton, 1989).

Many researchers have investigated whether selection for increased aggressiveness/virulence occurred after

several cycles of pathogen passage through the same host (Alexander et al., 1985; Clifford & Clothier, 1974; James & Fry, 1983; Jinks & Grindle, 1963; Latin et al., 1981; Leonard, 1969; Osburn et al., 1986; Osburn et al., 1987). It is reported that, when isolates of *Phytophthora infestans* were cycled several times through the same host, these isolates were more aggressive to the potato cultivars from where they were isolated but there was no evidence of increased aggressiveness after cycling (Caten, 1974; Jefferey et al., 1962; Jinks & Grindle, 1963; Latin et al., 1981), and a similar result was obtained with *Hordeum vulgare-Puccinia hordei* pathosystem (Clifford and Clothier, 1974). However, James & Fry (1983) found no evidence of increased aggressiveness of *Phytophthora infestans* isolates to the potato cultivar from which they were isolated, nor evidence of selection after repeated passage through that cultivar. On the other hand, Leonard (1969) reported that, after passage of *Puccinia graminis* f. sp. *avenae* through two oat (*Avena sativa*) cultivars, each subpopulation became more virulent to their "own" (the cultivar from which they were isolated) than to their "others" (cultivars from which they were not isolated).

Adaptation of Septoria to its host

The complete adaptation of a pathogen population to a newly introduced vertical resistance was suggested to involve a

two-stage process consisting of the acquisition of the matching virulence, followed by a recovery in general fitness (Parlevliet, 1981). King et al. (1983) reviewed several studies which indicated adaptation of both *S. nodorum* and *S. tritici* isolates to the host. After three cycles of passage through barley, a wheat isolate of *S. nodorum* showed increased virulence to barley (Rufty et al., 1981) and passages of a barley isolate through wheat resulted in greater sporulation than after passage through barley. Successive passage of a wheat isolate of *S. nodorum* through the grass hosts *Bromus sterilis*, *Lolium multiflorum*, and *Pheleum pratense* resulted in increased virulence to grass and a decrease in virulence to the original wheat cultivar from which it was isolated (Harrower, 1977). On the other hand, Ao & Griffiths (1976) reported that, after passage through a number of alternate hosts, including grasses, both *S. nodorum* and *S. tritici* from wheat were less virulent to the original host than isolates obtained directly from wheat. Osburn et al. (1986 & 1987)) found some changes in host preference when barley-adapted and wheat-adapted *S. nodorum* isolates were serially cycled through both barley and wheat, but concluded that it was due to cross-contamination with subsequent selection.

The inconsistent results regarding adaptation of *S. tritici* to its host make it imperative to further study

variability in virulence and adaptation in *S. tritici* populations. Since there exists genetic variability in *S. tritici* populations, the introduction and extensive cultivation of resistant wheat cultivars might select matching virulent pathogen genotypes to overcome that resistance or for specific adaptation of the pathogen to that cultivar. An adequate knowledge of pathogenic variability and adaptation of a pathogen population is a prerequisite to planning breeding programs and deployment of resistant genotypes for disease management.

In this study, experiments were conducted to determine pathogenic variability in virulence and adaptation of *S. tritici* populations secured from different locations using two sets of cultivars in two experiments. In another experiment, four *S. tritici* populations were tested on their "own" host, from which they were isolated, and cross-inoculated on "others", from which they were not obtained, to determine if there is variability in virulence among the populations and specific adaptation to the cultivar of origin.

REFERENCES

Agrios, GN, 1988. *Plant Pathology*, 3rd. edn. Academic Press, Inc.

Alexander, HM, Groth, JV, Roelfs, AP, 1985. Virulence changes in *Uromyces appendiculatus* after five asexual generations on a partially resistant cultivar of *Phaseolus vulgaris*. *Phytopathology* 75, 449-453.

Anonymous, 1965. *Losses in Agriculture*. Washington: United States Department of Agriculture. Handbook no. 291.

Ao, HC, Griffiths E, 1976. Change in virulence of *Septoria nodorum* and *Septoria tritici* after passage through alternative hosts. *Transactions of the British Mycological Society* 66, 219-224.

Bayles, RA, Parry, VWL, Clark, T, 1985. Resistance of winter wheat varieties to *Septoria tritici*. *Journal of National Institute of Agricultural Botany* 17(1), 21-26.

Brokenshire, T, 1976. The reaction of wheat genotypes to *Septoria tritici*. *Annual Applied Biology* 82, 415-423.

Brown JS, Kellock AW, Paddick RG, 1978. Distribution and dissemination of *Mycosphaerella graminicola* (Fuckel) Schroeter in relation to the epidemiology of speckled leaf blotch of wheat. *Australian Journal of Agricultural Research* 29, 1139-1145.

Camacho-Casas, MA, 1989. *Inheritance of Septoria Leaf Blotch Resistance and the Possible Influence of Heading Date and Plant Height on the Disease Expression in a Winter Wheat Cross*. Oregon: Oregon State University.

Camacho-Casas, MA, 1986. *Yield Loss Assessment in Non-protected Winter Wheat Varieties*. Oregon: Oregon State University.

Caten, CE 1974. Intra-racial variation in *Phytophthora infestans* and adaptation to field resistance for potato blight. *Annals of Applied Biology* 77, 259-270.

Centro Internacional de Mejoramiento de Maiz y Trigo (CIMMYT), 1972. *Annual Report* 1970-71. 114pp.

Clifford, BC, Clothier RB, 1974. Physiologic specialization of *Puccinia hordei* on barley hosts with nonhypersensitive resistance. *Transactions of the British Mycological Society* 63(3), 421-430.

Coakley, SM, McDaniel, LR, Shaner, G, 1985. Model for predicting severity of *Septoria tritici* blotch on winter wheat. *Phytopathology* 75, 1245-1251.

Djerbi, M, Ghodbane, A, Daaloul, A, Varughese, G, 1974. Identification de sources de resistance a la septoriose du ble (*Septoria tritici* Rob. et Desm.). *Annals of Phytopathology* 6, 495-496.

Dubin, HJ, 1985. *Occurrence and importance of* Septoria nodorum *and* Septoria tritici *in the Andean countries of South America*. Beltsville, Md: USDA Agriculture Research Service no.12.

Dubin, HJ, Rajaram, S, 1981. The strategy of the International Maize and Wheat Improvement Center (CIMMYT) for breeding disease resistant wheat: An international approach. In: Jenkyn JF, Plumb RT, eds. *Strategies for the Control of Cereal Disease*. Oxford: Blackwell Scientific Publications, 27-35.

El Ahmed, A, Maamouri, AR, Deghaies, M, 1984. Wheat breeding for septoria resistance in Tunisia: Identification of resistance genotypes. *Rachis* 3(2), 20-23.

Eyal, Z, 1972. Effect of Septoria leaf blotch on the yield of spring wheat in Israel. *Plant Disease Reporter* 56, 983-986.

Eyal, Z, 1981. Integrated control of Septoria disease of wheat. *Plant Disease* 65, 763-768.

Eyal, Z, Amiri, Z, Wahl, I, 1973. Physiologic specialization of *Septoria tritici*. *Phytopathology* 63, 1087-1091.

Eyal, Z, Scharen, AL, Huffman, MD, 1985. Global insights into virulence frequencies of *Mycosphaerella graminicola*. *Phytopathology* 75, 1456-1462.

Eyal, Z, Scharen, AL, Prescott, JM, Van Ginkel, M, 1987. *The Septoria Disease of Wheat: Concept and Methods of Disease Management*. Mexico, D. F.: CIMMYT.

Eriksson, J, 1894. Ueber die Specialisurung des Parasitimus bei den Getreiderostpilzen. *Ber. Dt. Bot. Ges.* 12, 292-331.

Fellows, H, 1962. Effect of light, temperature and fertilizer on infection of wheat leaves by *Septoria tritici*. *Plant Disease Reporter* 46, 846-848.

Hamid, AH, Ayers, JE, Hill, RR, 1982. Host isolate interactions in corn inbreds inoculated with *Cochliobolus carbonum* race 3. *Phytopathology* 72, 1169-1173.

Harrower, KM, 1977. Specialization of *Leptosphaeria nodorum* to alternative graminacious hosts. *Transactions of the British Mycological Society* 65, 101-103.

Hilo, HM, Bever, WM, 1957. Inoculation, oversummering, and suscept-pathogen relationship of *Septoria tritici* on *Triticum* species. *Phytopathology* 47, 474-480.

James, RV, Fry, WE, 1983. Potential for *Phytophthora infestans* populations to adapt to potato cultivars with rate reducing resistance. *Phytopathology* 73, 984-988.

Jefferey, SIB, Jinks, JL, Grindle, M, 1962. Intraracial variation in *Phytophthora infestans* and field resistance to potato blight. *Genetica* 32, 323-338.

Jenkins, JEE, Morgan, W, 1969. The effect of Septoria diseases on the yield of winter wheat. *Plant Pathology* 18, 152-156.

Jinks, JL, Grindle, M, 1963. Changes induced by training in *Phytophthora infestans*. *Heredity* 8(3), 245-264.

King, JE, Cook, RJ, Melville, SC, 1983. A review of septoria diseases of wheat and barley. *Annals of Applied Biology* 103, 345-373.

Krupinsky, JM, Craddock, JC, Scharen, AL, 1977. Septoria resistance in wheat. *Plant Disease Reporter* 61, 632-636.

Latin, RX, Mackenzie, DR, Cole, HJr., 1981. The influence of host and pathogen genotypes on the apparent infection rates of potato late blight epidemics. *Phytopathology* 71, 82-85.

Leonard, KJ, 1969. Selection in heterogenous populations of *Puccinia graminis* f. sp. *avenae*. *Phytopathology* 59, 1851-1857.

Madariaga RB, 1986. Presentia en Chile de *Mycosphaerella graminicola* (Fuckel) Schroeter, estado sexuada de *Septoria tritici* Rob. ex. Desm. *Agricultura Tecnica* 46, 209-211.

Madariaga, RB, Gilchrist, DG, Martensen, AN, 1989. Presence and role of *Mycosphaerella graminicola* in California wheat. *Phytopathology* 79, 1141.

Marshall, D, 1985. Geographic distribution and aggressiveness of *Septoria tritici* on wheat in the United States. *Phytopathology* 75, 1319.

McDonald, BA, Martinez, JP, 1990. DNA restriction fragment length polymorphisms among *Mycosphaerella graminicola* (anamorph *Septoria tritici*) isolates collected from a single wheat field. *Phytopathology* 80, 1368-1373.

Mehta, YR, 1989. Occurrence of *Septoria tritici* and its perfect state in Brazil. *Proceedings of the Third International Workshop on Septoria Diseases of Cereals*, 1989. Swiss Federal Research Station for Agronomy, Zurich, 34-35.

Mew, TW, Vera Cruz, CM, Medalla, ES 1992. Changes in race frequency of *Xanthomonas oryzae* pv. *oryzae* in response to rice cultivars planted in Philippines. *Plant Disease* 76, 1029-1032.

Morales, IN, 1958. Studies on *Septoria* leaf blotch of wheat. *Dissertation Abstracts* 18, 357-358.

Morales, IN, 1957. *Studies on Septoria Leaf Blotch of Wheat.* Indiana: Purdue University.

Narvaez, I, 1957. *Studies of Septoria leaf blotch of wheat.* Indiana: Purdue University.

Narvaez, I, Caldwell, RM, 1957. Inheritance of resistance of leaf blotch of wheat caused by *Septoria tritici*. *Phytopathology* 47, 529.

Nelson, LR, and Marshall, D, 1990. Breeding wheat for resistance to *Septoria nodorum* and *Septoria tritici*. *Advances in Agronomy* 44, 257-277.

Newton, AC, 1989. Genetic adaptation of *Erysiphe* f. sp. *hordei* to barley with partial resistance. *Journal of Phytopathology* 126, 133-148.

Osburn, AE, Caten, CE, Scott, PR, 1987. Adaptation of *Septoria nodorum* to wheat or barley on detached leaves. *Plant Pathology* 36, 565-576.

Osburn, AE, Scott, PR, and Caten, CE, 1986. The effect of host passaging on the adaptation of *Septoria nodorum* to wheat or barley. *Plant Pathology* 35, 135-145.

Parlevliet, JE, 1977. Evidence of differential interaction in the polygenic *Hordeum vulgare-Puccinia hordei* relation during epidemic development. *Phytopathology* 67, 776-778.

Parlevliet, JE, 1981. Stabilizing selection in crop pathosystems: an empty concept or a reality? *Euphytica* 30, 259-269.

Parlevliet, JE, Zadoks, JC, 1977. The integrated concept of disease resistance; a new view including horizontal and vertical resistance in plants. *Euphytica* 26, 5-21.

Rillo, AO, and Caldwell, RM, 1966. Inheritance of resistance to *Septoria tritici* in *Triticum aestivum* subs. *vulgare* Bulgaria 88. *Phytopathology* 56, 897.

Rosielle, AA, 1972. Sources of resistance in wheat to speckled leaf blotch caused by *Septoria tritici*. *Euphytica* 21, 152-161.

Rosielle, AA, Brown, AG, 1979. Inheritance, heritability and breeding behaviour of three sources of resistance to *Septoria tritici* in wheat. *Euphytica* 28, 385-392.

Rufty, RC, Herbert, TT, Murphy, CF, 1981. Variation in virulence in isolates of *Septoria nodorum*. *Phytopathology* 71, 593-596.

Saadaoui, EM, 1987. Physiologic specialization of *Septoria tritici* in Morocco. *Plant Disease* 71, 153-155.

Saari, EE, 1974. Results of studies on *Septoria* in the Near East and Africa In: *Proceedings of the Fourth FAO/Rockefeller Foundation Wheat Seminar*, 1974. Tehran, Iran, 441.

Saari, EE, and Wilcoxson, RD, 1974. Plant disease situation of high yielding dwarf wheats in Asia and Africa. *Annual Review of Phytopathology* 12, 49-68.

Sanderson, FR, 1972. A Mycosphaerella species as the ascogenous state of *Septoria tritici* Rob. and Desm. *New Zealand Journal of Botany* 10, 707-710.

Scharen, AL, Sanderson, FR, 1985. Identification, distribution and nomenclature of the *Septoria* species that attack cereals. In: Scharen AL, ed. *Septoria of Cereals. Proceedings of Workshop*, 1983. Bozeman, Montana, United States Department of Agriculture, 37-41.

Schuh, W, 1990. Influence of tillage systems on the disease intensity and spatial pattern of Septoria leaf blotch. *Phytopathology* 80, 1337-1340.

Scott, PR, Sanderson, FR, and Benedikz, PW, 1988. Occurrence of *Mycosphaerella graminicola*, teleomorph of *Septoria tritici* on wheat in U.K. *Plant Pathology* 37, 285-290.

Shaner, G, and Finney, RE, 1976. Weather and epidemics of Septoria leaf blotch of wheat. *Phytopathology* 66, 781-785.

Shaner, G, Finney, RE, and Patterson, FL, 1975. Expression and effectiveness of resistance in wheat to Septoria leaf blotch. *Phytopathology* 65, 761-766.

Shaner, G, Stromberg EL, Lacy GH, Barker, KR, Pirone, TP, 1992. Nomenclature and concepts of pathogenicity and virulence. *Annual Review of Phytopathology* 30, 47-66.

Shaw, MW, Royle, DJ, 1989. Airborne inoculum as a source of *Septoria tritici* (*Mycosphaerella graminicola*) infections in the winter wheat crops in the UK. *Plant Pathology* 38, 35-43.

Shipton, WA, 1966. Septoria leaf and glume blotch of wheat. *Journal of Agriculture, Western Australia* 7, 160-163.

Shipton, WA, Boyd, WJR, Rosielle, AA, Shearer, BL, 1971. The common septoria diseases of wheat. *Botanical Review* 37, 231-262.

Stakman, EC, Piemseisel, FJ, 1917. A new strain of *Puccinia graminis*. *Phytopathology* 7, 73.

Stewart, DM, Hafiz, A, Abdel Hak, T, 1972. Disease epiphytotic threats to high-yielding and local wheats in the Near East. FAO Plant Protection Bulletin 20, 50-57.

Tyagi, PD, Yoshi, LM, Renfro, BL, 1970. Reaction of wheat varieties to *Septoria tritici* and report of an epidemic in north-western Punjab. *Indian Phytopathology* 22, 175-178.

Van der Plank, JE, 1968. *Disease Resistance in Plants*, 2nd edn. New York: Academic Press.

Van Ginkel, M, and Scharen, AL, 1987. Generation mean analysis and heritabilities of resistance to *Septoria tritici* in durum wheat. *Phytopathology* 77, 1629-1633.

Yechilevich-Auster, M, Levi, E, Eyal, Z, 1983. Assessment of interactions between cultivated and wild wheats and *Septoria tritici*. *Phytopathology* 73, 1077-1083.

Zelikovitch, N, Eyal, Z, 1991. Reduction in pycnidial coverage after inoculation of wheat with mixtures of isolates of *Septoria tritici*. *Plant Disease* 75, 907-910.

Ziv, O, Eyal, Z, 1978. Assessment of yield component losses caused in plants of spring wheat cultivars by selected isolates of *Septoria tritici*. *Phytopathology* 68, 791-796.

Host-Pathogen Relationship of Geographically Diverse Isolates of *Septoria tritici* and Wheat Cultivars.

CHAPTER II

SUMMARY

Pathogenic variability of 15 *Septoria tritici* isolates from different locations in California, Oregon, and Texas was determined on seedlings of two sets of geographically diverse wheat cultivars under greenhouse conditions. Significant isolate effects, cultivar effects, and isolate X cultivar interactions were found, though the interaction terms were very small compared to the main effects of isolate and cultivar. All except one isolate were virulent to two sets of cultivars, and the virulence patterns varied among the isolate-cultivar combinations. Variability in virulence among the *S. tritici* isolates within and between locations also was observed. The isolates were usually more virulent to the cultivars of the same geographic origin than cultivars of other locations. Linear contrasts between all "own" (isolate X cultivar combination of same location) and all "others" (isolate X cultivar combination of different locations) indicated that all own produced significantly more disease than all others. The observed amount of disease was higher than the expected amount of disease on each isolate X cultivar combination of the same location. These

results demonstrate location-specific adaptation of *S. tritici*. Implications of pathogenic variability and adaptation in *S. tritici* are discussed.

INTRODUCTION

Septoria tritici blotch (STB) of wheat (*Triticum aestivum* L.) caused by the ascomycetous pseudothecial fungus *Mycosphaerella graminicola* (Fuckel) J. Schrot. in Cohn (anamorph= *Septoria tritici* Roberge in Desmaz.) is a major disease in all wheat growing areas of the world and causes severe yield loss (Camacho-Casas, 1986; Coakley et al., 1985; Dubin, 1985; Eyal, 1972; Eyal et al., 1973; Eyal, 1981; Eyal et al., 1987; Jenkins & Morgan, 1969; King et al., 1983; Madariaga et al., 1989; Schuh, 1990; Shaner & Finney, 1976; Shipton et al., 1971; Van Ginkel & Scharen, 1987; Zelikovitch & Eyal, 1991; Ziv & Eyal, 1978). Annual average reduction in potential wheat yield due to STB and Septoria nodorum blotch (SNB) (caused by *Septoria nodorum* Miller) was estimated as 1% for the United States (Anonymous, 1965; Coakley, et al., 1985) and estimated in 1982 at about 9 million metric tons worldwide with a value of over $ 1 billion (Eyal et al., 1987; Scharen & Sanderson, 1985).

The disease severity of STB may have increased with the replacement of tall, late-maturing cultivars by short, early maturing cultivars grown at high nitrogen levels (Eyal, 1981; Shaner et al., 1975). Early maturity, short stature, high nitrogen levels, and changing husbandry practices

apparently all favor STB (King et al., 1983; Narvaez, 1957; Shipton, 1966; Shipton et al., 1971).

Resistance to *S. tritici* has been reported by several researchers (Brokenshire, 1976; CIMMYT, 1972; Hilo & Bever, 1957; Morales, 1957; Nelson & Marshall, 1990; Rosielle, 1972; Tyagi et al., 1970; Yechilevich-Auster, 1983). Different modes of inheritance of resistance to *S. tritici* have been reported among common and durum wheats. These range from simple Mendelian inheritance to quantitative inheritance, with the genetic variation involving both nonadditive and additive gene action (Camacho-Casas, 1989). It has been reported that resistance to STB is controlled by a single dominant gene (Narvaez & Caldwell, 1957; Rillo & Caldwell, 1966; Rosielle & Brown, 1979), two additive genes (Narvaez & Caldwell, 1957), and at least three recessive genes (Rosielle & Brown, 1979).

Existence of specialized forms in *S. tritici* have been documented (Shipton et al., 1971) but many researchers have been unable to distinguish physiologic races within this *formae specialies*, although the isolates differ in degree of virulence (King et al., 1983). However, in Israel, Morocco, and the United States researchers found evidence of differential interactions with wheat cultivars, suggesting

physiologic specialization in *S. tritici* (Eyal et al., 1973; Eyal, 1981; King et al., 1983; Saadaoui, 1987).

Eyal et al. (1985) evaluated virulence patterns, in terms of ability to incite compatible disease reaction, for 97 isolates of *S. tritici* on seedlings of 35 wheat and triticale cultivars. Significant isolate X cultivar interactions indicated the existence of specific virulence genes among isolates. Virulence frequencies varied considerably among regions and within countries. Eyal et al. (1985) indicated that *Septoria* isolates were more virulent to the cultivars from where the isolates were obtained. Isolates from some areas, including Oregon in the United States, were more virulent to a larger number of differentials than isolates from other areas.

Marshall (1985) found marked variation in aggressiveness, measured as area under disease progress curves (AUDPC), of *S. tritici* populations collected from 16 locations of ten states in the U.S.A. *S. tritici* populations of central and north central California and northern regions of Indiana and Ohio were more aggressive, while those from northern Alabama, Mississippi, eastern Arkansas, southern Nebraska, and central Kansas were least aggressive. He also found greater variability in aggressiveness in the highly

aggressive *S. tritici* populations than in the less aggressive ones.

The sexual stage of *S. tritici* has been shown to occur in wheat stubble in New Zealand (Sanderson, 1972), Victoria, Australia (Brown et al., 1978), Chile (Madariaga, 1986), the eastern UK (Scott et al., 1988); Brazil (Mehta, 1989); California, USA (Madariaga et al., 1989); the Netherlands (Shaw & Royle, 1989), and Oregon (personal observation, 1990). The sexual stage provides the opportunity for increased genetic variation within the *S. tritici* population. In fact, McDonald & Martinez, (1990) quantified a high level of genetic variability within *S. tritici* populations using restriction fragment length polymorphosim (RFLP) analysis. They suggested that this genetic variability among *S. tritici* populations would allow for rapid selection of clones that are fungicide-resistant or virulent to wheat cultivars that have pathotype-specific resistance. McDonald & Martinez also found that deletion and insertion events are common in *S. tritici* nuclear DNA among geographically diverse groups of isolates, and that genome rearrangements occur at a high frequency.

Since there is evidence of high genetic variability in populations of *S. tritici*, there might be selection of host-specific, virulent races depending on the wheat cultivar

grown in a certain location. Thus, there is need for more research on the pathogenic variability and adaptation of *S. tritici*, and this information is a prerequisite for planning breeding programs and deployment of resistant cultivars to combat STB. The present study was undertaken to determine the pathogenic variability of *S. tritici* isolates from different locations using host differentials from different geographic regions, and also to determine if there is specific adaptation of the pathogen to host cultivars grown within the same geographic region.

MATERIALS AND METHODS

Host

For the first experiment, five Oregon winter wheat cultivars were used (Table II.1). For the second experiment, 12 cultivars from California, the UK, Indiana, Oregon, and Texas were evaluated (Table II.2 & II.3). The plants were raised under artificial lighting in the greenhouse in 10 cm pots filled with pot mixture (1 part peat; 1 part sand; 1 part loam soil; 2 parts # 8 pumice with no fines). Approximately 15 seeds of a cultivar were sown in each pot. At seven days after seeding, the plants were thinned to ten per pot. The plants were fertilized twice with 50 ml/pot of Miracle-Gro (ammoniacal nitrogen 6.8%; urea nitrogen 8.2%; available phosphoric acid P_2O_5 30%; soluble potash K_2O 15%; boron 0.02%; copper 0.07%; iron 0.15%; manganese 0.05%; molybdenum 0.0005% and zinc 0.06%) at the rate of 15g/L of water after two weeks and five weeks of seeding. The plants were sprayed and/or fumigated with insecticide (insecticidal soap 15ml/L and/or nicotine smoke) as necessary to control aphids and fumigated weekly with fungicide (fenarimol) to control powdery mildew. Greenhouse temperature was maintained at 20 ± 5°C.

Pathogen

Fifteen monopycnidial isolates of *S. tritici* from three different locations were obtained from Bruce McDonald of Texas A & M University. Six isolates were of California origin, secured from the cultivar Yolo; six isolates were from Oregon, isolated from the cultivar Stephens; and three isolates were obtained from unknown wheat cultivars grown in the Texas area (personal communication with Bruce McDonald) (Table II.1). The isolates were maintained on refrigerated silica gel in cryovials. Before plant inoculation, crystals of silica gel were plated onto potato dextrose agar (PDA) plates fortified with 50 mg/L of streptomycin. After three-to-four days, the blastospores of the fungus grew on and around the crystals of silica gel and were streaked with a bacteria transfer loop onto the surface of the same PDA plates. Two days after streaking, the fungus was transferred onto PDA slants and spread uniformly with a bacteria transfer loop. After three days, the fungus was used for inoculations. About two-to-three ml of sterile water was poured into each slant, and then scraped with a fungal transfer needle to suspend the spore mass.

Inoculation

At twenty-one days after seeding, the plants were sprayed with a hand sprayer using 25 ml (first experiment) and 50 ml

(second experiment) per pot of a suspension of 10^6 spores/ml. These amounts were sufficient to inoculate the plants to run-off. After inoculation, the plants were kept in a moist chamber covered with a polyethylene sheet for 96 hours. High humidity (95% or above) was maintained using a humidifier inside the moist chamber. A temperature of 20 ± 5°C was maintained inside the moist chamber and greenhouse; this was possible since the experiments were conducted during the period from November to March (1991-1993). The pots were subsequently placed on a greenhouse bench in a randomized order until scoring.

Disease assessment

Percent diseased leaf area (% DLA) of the second leaf from the base of each of the ten plants in each pot were scored visually. In addition, % DLA on a per pot basis was estimated four times for the second experiment at two-day intervals beginning 13 days after inoculation when some isolate/host combinations showed visible disease lesions. The area under disease progress curve (AUDPC) was then calculated to summarize disease severity over time for each pot using the equation described by Shaner & Finney (1977).

Statistical Analysis

Due to space and labor limitations, each of the four replications of each experiment was done at different times. Data were analyzed as a factorial analysis of variance for a randomized complete block design using SAS (1988). The sources of variance were replications, cultivars, isolates and cultivar X isolate interaction. Fisher's protected LSD (P= 0.05) was used for making multiple comparisons among treatment means. For the second experiment, the data were also pooled in a separate analysis of variance according to location (California, Oregon, and Texas) from where the isolates originated. The Indiana and UK cultivars were excluded from this analysis, since no isolates from these locations were used in this study. Linear contrasts were used to compare all "own" isolates (isolates tested on cultivars from the regions where the pathogen was isolated) vs all "others" (isolates tested on cultivars from outside the region of isolation) of pooled % DLA and AUDPC to determine if there is location-specific variability and adaptation of *S. tritici*. Expected % DLA and AUDPC (isolate mean for a location X cultivar mean for isolates from that location/grand mean) were calculated for each isolate X cultivar combination. These expected values were compared to the observed values for both "own" and "other" combinations to test the presence of location-specific adaptation of *S. tritici*.

RESULTS

All but one isolate of *S. tritici* were pathogenic to the two sets of cultivars, but their degree of pathogenicity, i.e, virulence, varied among the isolate/cultivar combinations. Reaction patterns of the 15 isolates on five Oregon winter wheat cultivars indicated that Oregon isolates caused more diseased leaf area than the California and Texas isolates (Table II.1). However, California and Texas isolates were highly virulent to the cultivar Madsen. There was also variability in virulence among isolates of the same location. California isolates ST36 and ST47 and Texas isolates BB1 and BB2 were more virulent to Madsen than were other isolates. Among all of the isolates, Texas isolate BB1 incited the highest diseased leaf area on Madsen. There was no virulence to the cultivar Gene among the isolates studied. The Texas isolate 83.22 lacked virulence to all of the cultivars. The order of ranking of % DLA differed among isolate X cultivar combinations. Analysis of variance (Table II.2) of experiments one and two indicated significantly large cultivar differences, moderate isolate differences, and small but highly significant isolate X cultivar interactions.

On the basis of disease reactions over all isolates, the five Oregon wheat cultivars fell into three distinct groups: resistant (Gene), intermediate (Hill, Malcolm, and

Stephens) and susceptible (Madsen) (Table II.1); although when considering only the Oregon isolates, Madsen was less susceptible than Malcolm or Stephens. Disease reactions over all cultivars for experiment one showed that Oregon isolates were more virulent than California and Texas isolates (Table II.1). Results with the set of 12 cultivars for both % DLA and AUDPC (Table II.3 & II.4) showed significant differences among the cultivars, and the 12 cultivars fell into nine groups (Table II.3 & II.4). On the basis of disease reaction over the set of 12 cultivars, the groupings of the isolates are different from the previous experiment which utilized only the Oregon cultivars. However, in this experiment, Texas isolate 83.22 was again the least virulent (Table II.3 & II.4).

Analysis of variance of pooled data (Table II.5) across isolate X cultivar combinations of locations California, Oregon, and Texas also showed highly significant cultivar effects, isolate effects, and significant isolate X cultivar interactions. Means comparison of % DLA pooled across isolates and cultivars indicated that Texas isolates were most virulent, followed by California and Oregon isolates, and that Texas cultivars were most susceptible, followed by Oregon and California cultivars (Table II.6).

Significant differences were obtained when contrasts were made between all "own" (isolate X cultivar combinations of same location) and all "others" (isolate X cultivar combinations of different locations) (Table II.5), and all "own" produced more disease than the all "others". It is also evident that "observed" % DLA and AUDPC values were always higher than "expected" values for isolate X cultivar combinations of the same location (Table II.6).

Analysis of variance of AUDPC data of the 12 cultivar X 15 isolate combinations indicated highly significant differences in cultivars, isolates, and isolate X cultivar interaction (Table II.2). The isolate X cultivar interaction was small relative to main effects of isolate and of cultivar. Cultivar and isolate groupings based on AUDPC were similar to those based on % DLA taken once at 21 days after inoculation (Tables II.3 & II.4).

DISCUSSION

To formulate a breeding program for disease resistance, it is a prerequisite to have comprehensive knowledge of pathogenic variability or physiologic specialization of the pathogen. Many researchers have been unable to identify physiologic specialization in *S. tritici*, but found differences in virulence (relative disease evoking capacity) among isolates of this pathogen (Eyal et al., 1973), as indicated by lack of a isolate X cultivar interaction and constant ranking of isolates across cultivars. Our results showed highly significant isolate X cultivar interactions, that isolates of the pathogen varied in the levels of disease they caused, and that ranking of the cultivars depended on the isolate used. These pathogenic characteristics would indicate vertical pathogenicity *sensu* Van der Plank (1968) or the existence of "physiologic races" *sensu* Stakman et al. (1962). Evidence of physiologic specialization in the *Septoria-Triticum* pathosystem has also been reported from Israel, Morocco, and the U.S.A. (Eyal et al., 1973; King et al., 1983; Saadaoui, 1987). Saadaoui (1987) found a highly significant isolate X cultivar interaction, but did not note the relative amount of variation explained by this interaction as compared to main effects.

In our study, the amount of variation explained by isolate X cultivar interactions, though highly significant statistically, was very small compared to main effects of isolate and cultivar (Table II.2). Eyal et al. (1985) reported similar relative amounts of variation explained by main effect and interaction terms, as did Rufty et al. (1981) and Scharen & Eyal (1983). Rufty et al. (1981) indicated that the magnitude of interactions was small, and therefore, classification of the isolates into physiologic races was inappropriate. It should be noted, however, that resistance genes have not been as widely deployed in wheat cultivars against *Septoria* as in *Puccinia-Triticum* or *Pyricularia-Oryza* pathosystems (personal communication with Christopher Mundt, Oregon State University). Thus, it is less likely that gene-for-gene interaction would lead to the evolution of physiologic races in the *S. tritici* system.

Parlevliet & Zadoks (1977) provide an integrated concept of disease resistance which holds that all genes for true resistance in the host population, whether they are major or minor genes, interact in a gene-for-gene way with virulence genes, either major or minor, in the pathogen population. Evidence of differential interaction in the polygenic *Hordeum vulgare-Puccinia hordei* relation has been reported (Parlevliet, 1977).

Marshall (1985) found wide variation for aggressiveness of *S. tritici* as measured by disease severity and pycnidial density, but no isolate X cultivar interactions. Van Ginkel and Scharen (1988) suggested that *S. tritici* isolates have species specificity and were specifically adapted to either bread or durum wheat, but that cultivar specificity was not significant. In both experiments, in our study, isolates markedly varied in virulence over cultivars. In experiment one, on the average, the Oregon isolates were more virulent to Oregon cultivars than the California and Texas isolates were. This result suggests that there is selection of virulence factor(s) for Oregon isolates against Oregon wheat cultivars. However, two California and two Texas isolates were more virulent to Madsen than the Oregon isolates were; this result deserves further research. Cordo et al. (1989) reported that *S. tritici* isolates secured from wheat leaves are heterogeneous for virulence. It has been reported that certain *S. tritici* isolates were able to overcome the resistance of wheat cultivars, and that these isolates must have one or more genes for specific virulence that do not occur in other isolates (Van Shilfhout et al., 1989).

Analysis of variance, along with the contrast of pooled data across isolates and cultivars of each location, showed that isolates are significantly more virulent to cultivars from their region of origin than from other regions. The

"observed" values for each isolate X cultivar combination of the same location is always higher than the "expected" values. These results support those of Eyal et al., (1985), who found that isolates from Latin America were more virulent to cultivars originating from Latin America and that isolates obtained from tetraploid wheat from Syria and Tunisia were more virulent to tetraploid than to hexaploid wheat cultivars. Thus, it may be common for *S. tritici* populations to become adapted to the host cultivars grown in a certain location. Means comparison of % DLA and AUDPC across all isolates and cultivars (Table II.6) indicated that Texas isolates were most virulent, and that Texas cultivars were most susceptible, suggesting that isolates from the most susceptible cultivars were more virulent. This conclusion is supported by data presented in chapter III of this thesis.

Extensive research has been taken to incorporate resistance gene(s) into agronomically adapted cultivars. Once such cultivars with specific resistance gene(s) against *S. tritici* have been deployed, it is likely that corresponding virulent races would evolve to overcome that resistance. For example, resistance to bacterial leaf blight of rice was once thought to be race non-specific (Mew, 1987). Race 1 of *Xanthomonas oryzae* pv. *oryzae* was predominant in the Philippines before the extensive

cultivation of improved modern rice cultivars, and was virulent to rice cultivars with *Xa*-10, *Xa*-11, *Xa*-14 genes. When cultivars with the *Xa*-4 gene were introduced, race 1 declined, but races 2 and 3, which can attack cultivars with the *Xa*-4 gene, appeared throughout the country (Mew et al., 1992). Therefore, research is needed to fix a set of common differentials for the *S. tritici-Triticum* pathosystem to monitor changes in populations of *S. tritici*.

REFERENCES

Anonymous, 1965. *Losses in Agriculture*. Washington: United States Department of Agriculture. Handbook no. 291.

Brokenshire, T, 1976. The reaction of wheat genotypes to *Septoria tritici*. *Annals Applied Biology* 82, 415-423.

Brown JS, Kellock AW, Paddick RG, 1978. Distribution and dissemination of *Mycosphaerella graminicola* (Fuckel) Schroeter in relation to the epidemiology of speckled leaf blotch of wheat. *Australian Journal of Agricultural Research* 29, 1139-1145.

Camacho-Casas, MA, 1989. *Inheritance of Septoria Leaf Blotch Resistance and the Possible Influence of Heading Date and Plant Height on the Disease Expression in a Winter Wheat Cross*. Oregon: Oregon State University.

Camacho-Casas, MA, 1986. *Yield Loss Assessment in Non-protected Winter Wheat Varieties*. Oregon: Oregon State University.

Centro Internacional de Mejoramiento de Maiz y Trigo (CIMMYT), 1972. *Annual Report* 1970-71. 114pp.

Coakley, SM, McDaniel, LR, Shaner, G, 1985. Model for predicting severity of *Septoria tritici* blotch on winter wheat. *Phytopathology* 75, 1245-1251.

Cordo, CA, Perello, A, Arriaga, H, Pascar A, 1989. Study of the stability of *Mycosphaerella graminicola* (*Septoria tritici*) isolates.In Fried, MF, eds. *Proceedings of the Third International Workshop on Septoria Diseases of Cereals*, 1989. Swiss Federal Research Station for Agronomy, Zurich, 55-58.

Dubin, HJ, 1985. *Occurrence and importance of* Septoria nodorum *and* Septoria tritici *in the Andean countries of South America*. Beltsville, Md: USDA Agriculture Research Service no.12.

Eyal, Z, 1972. Effect of Septoria leaf blotch on the yield of spring wheat in Israel. *Plant Disease Reporter* 56, 983-986.

Eyal, Z, 1981. Integrated control of Septoria disease of wheat. *Plant Disease* 65, 763-768.

Eyal, Z, Amiri, Z, Wahl, I, 1973. Physiologic specialization of *Septoria tritici*. *Phytopathology* 63, 1087-1091.

Eyal, Z, Scharen, AL, Huffman, MD, 1985. Global insights into virulence frequencies of *Mycosphaerella graminicola*. *Phytopathology* 75, 1456-1462.

Eyal, Z, Scharen, AL, Prescott, JM, Van Ginkel, M, 1987. *The Septoria Disease of Wheat: Concept and Methods of Disease Management*. Mexico, D. F.: CIMMYT.

Hilo, HM, Bever, WM, 1957. Inoculation, oversummering, and suscept-pathogen relationship of *Septoria tritici* on *Triticum* species. *Phytopathology* 47, 474-480.

Jenkins, JEE, Morgan, W, 1969. The effect of Septoria diseases on the yield of winter wheat. *Plant Pathology* 18, 152-156.

King, JE, Cook, RJ, Melville, SC, 1983. A review of septoria diseases of wheat and barley. *Annals of Applied Biology* 103, 345-373.

Madariaga RB, 1986. Presentia en Chile de *Mycosphaerella graminicola* (Fuckel) Schroeter, estado sexuada de *Septoria tritici* Rob. ex. Desm. *Agricultura Tecnica* 46, 209-211.

Madariaga, RB, Gilchrist, DG, Martensen, AN, 1989. Presence and role of *Mycosphaerella graminicola* in California wheat. *Phytopathology* 79, 1141.

Marshall, D, 1985. Geographic distribution and aggressiveness of *Septoria tritici* on wheat in the United States. *Phytopathology* 75, 1319.

McDonald, BA, Martinez, JP, 1990. DNA restriction fragment length polymorphisms among *Mycosphaerella graminicola* (anamorph *Septoria tritici*) isolates collected from a single wheat field. *Phytopathology* 80, 1368-1373.

Mehta, YR, 1989. Occurrence of *Septoria tritici* and its perfect state in Brazil. *Proceedings of the Third International Workshop on Septoria Diseases of Cereals*, 1989. Swiss Federal Research Station for Agronomy, Zurich, 34-35.

Mew, TW, 1987. Current status and future prospects of research on bacterial blight of rice. *Review of Phytopathology* 25, 359-382.

Mew, TW, Vera Cruz, CM, Medalla, ES, 1992. Changes in race frequency of *Xanthomonas oryzae* pv. *oryzae* in response to rice cultivars planted in Philippines. *Plant Disease* 76, 1029-1032.

Morales, IN, 1957. *Studies on Septoria Leaf Blotch of Wheat*. Indiana: Purdue University.

Narvaez, I, 1957. *Studies of Septoria leaf blotch of wheat*. Indiana: Purdue University.

Narvaez, I, Caldwell, RM, 1957. Inheritance of resistance of leaf blotch of wheat caused by *Septoria tritici*. *Phytopathology* 47, 529.

Nelson, LR, and Marshall, D, 1990. Breeding wheat for resistance to *Septoria nodorum* and *Septoria tritici*. *Advances in Agronomy* 44, 257-277.

Parlevliet, JE, 1977. Evidence of differential interaction in the polygenic *Hordeum vulgare-Puccinia hordei* relation during epidemic development. *Phytopathology* 67, 776-778.

Parlevliet, JE, Zadoks, JC, 1977. The integrated concept of disease resistance; a new view including horizontal and vertical resistance in plants. *Euphytica* 26, 5-21.

Rillo, AO, and Caldwell, RM, 1966. Inheritance of resistance to *Septoria tritici* in *Triticum aestivum* subs. *vulgare* Bulgaria 88. *Phytopathology* 56, 897.

Rosielle, AA, 1972. Sources of resistance in wheat to speckled leaf blotch caused by *Septoria tritici*. *Euphytica* 21, 152-161.

Rosielle, AA, Brown, AG, 1979. Inheritance, heritability and breeding behaviour of three sources of resistance to *Septoria tritici* in wheat. *Euphytica* 28, 385-392.

Rufty, RC, Herbert, TT, and Murphy, CF, 1981. Variation in virulence in isolates of *Septoria nodorum*. *Phytopathology* 71, 593-596.

Saadaoui, EM, 1987. Physiologic specialization of *Septoria tritici* in Morocco. *Plant Disease* 71, 153-155.

Sanderson, FR, 1972. A Mycosphaerella species as the ascogenous state of *Septoria tritici* Rob. and Desm. *New Zealand Journal of Botany* 10, 707-710.

SAS Institute, Inc., 1988. SAS/STAT User's Guide, Release 6.03 edition. SAS Institute, Inc., Cary, NC.

Scharen, AL, and Eyal, Z, 1983. Analysis of symptoms on spring and winter wheat cultivars inoculated with different isolates of *Septoria nodorum*. *Phytopathology* 73, 143-147.

Scharen, AL, Sanderson, FR, 1985. Identification, distribution and nomenclature of the *Septoria* species that attack cereals. In: Scharen AL, ed. *Septoria of Cereals*. *Proceedings of Workshop*, 1983. Bozeman, Montana, United States Department of Agriculture, 37-41.

Schuh, W, 1990. Influence of tillage systems on the disease intensity and spatial pattern of Septoria leaf blotch. *Phytopathology* 80, 1337-1340.

Scott, PR, Sanderson, FR, and Benedikz, PW, 1988. Occurrence of *Mycosphaerella graminicola*, teleomorph of *Septoria tritici* on wheat in U.K. *Plant Pathology* 37, 285-290.

Shaner, G, and Finney, RE, 1976. Weather and epidemics of Septoria leaf blotch of wheat. *Phytopathology* 66, 781-785.

Shaner, G, Finney, RE, 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. *Phytopathology* 67, 1051-1056.

Shaner, G, Finney, RE, and Patterson, FL, 1975. Expression and effectiveness of resistance in wheat to Septoria leaf blotch. *Phytopathology* 65, 761-766.

Shaw, MW, Royle, DJ, 1989. Airborne inoculum as a source of *Septoria tritici* (*Mycosphaerella graminicola*) infections in the winter wheat crops in the UK. *Plant Pathology* 38, 35-43.

Shipton, WA, 1966. Septoria leaf and glume blotch of wheat. *Journal of Agriculture, Western Australia* 7, 160-163.

Shipton, WA, Boyd, WJR, Rosielle, AA, Shearer, BL, 1971. The common septoria diseases of wheat. *Botanical Review* 37, 231-262.

Stakman, EC, Stewart, DM, Loegering, WQ, 1962. *Identification of physiologic races of* Puccinia graminis var. tritici. United States Department of Agriculture. Agriculture Research Service. e617. pp 53.

Tyagi, PD, Yoshi, LM, Renfro, BL, 1970. Reaction of wheat varieties to *Septoria tritici* and report of an epidemic in north-western Punjab. *Indian Phytopathology* 22, 175-178.

Van der Plank, JE, 1968. *Disease Resistance in Plants*, 2nd edn. New York: Academic Press.

Van Ginkel, M, and Scharen, AL, 1987. Generation mean analysis and heritabilities of resistance to *Septoria tritici* in durum wheat. *Phytopathology* 77, 1629-1633.

Van Ginkel, M, Scharen, AL, 1988. Host-pathogen relationships of wheat and *Septoria tritici*. *Phytopathology* 78, 762-766.

Van Shilfhout, CH, Arama, PF, Kema, GHJ, 1989. International survey of virulence of *Septoria tritici*. In Fried, MF, eds. *Proceedings of the Third International Workshop on Septoria Diseases of Cereals*, 1989. Swiss Federal Research Station for Agronomy, Zurich, 36-38.

Yechilevich-Auster, M, Levi, E, Eyal, Z, 1983. Assessment of interactions between cultivated and wild wheats and *Septoria tritici*. *Phytopathology* 73, 1077-1083.

Zelikovitch, N, Eyal, Z, 1991. Reduction of pycnidial coverage after inoculation of wheat with mixtures of isolates of *Septoria tritici*. *Plant Disease* 75, 907-910.

Ziv, O, Eyal, Z, 1978. Assessment of yield component losses caused in plants of spring wheat cultivars by selected isolates of *Septoria tritici*. *Phytopathology* 68, 791-796.

Table II.1. Percent diseased leaf area of five Oregon wheat cultivars caused by 15 *S. tritici* isolates tested in the greenhouse.

Isolate	Origin[+]	Cultivars					
		Gene	Hill	Madsen	Malcolm	Stephens	Mean
1. BB1	TX	1.0	3.1	41.1	2.1	1.2	9.7 de[*]
2. BB2	TX	1.0	1.7	21.4	1.0	1.3	5.3 fgh
3. 83.22	TX	1.1	1.1	1.1	1.1	2.6	1.4 i
4. ST5	CA	1.1	1.3	10.2	5.5	1.7	3.9 ghi
5. ST36	CA	2.7	1.6	26.6	3.7	6.4	8.2 ef
6. ST47	CA	1.0	1.1	24.9	3.5	2.0	6.5 fg
7. ST60	CA	1.0	1.0	4.2	1.3	1.2	1.7 i
8. ST66	CA	1.5	1.8	9.0	2.3	2.8	3.5 hi
9. ST85	CA	1.0	1.1	12.3	1.6	3.5	3.9 ghi
10. 11-2A.2	OR	1.0	21.8	11.0	22.3	15.9	14.4 bc
11. 11-3B.7	OR	1.1	19.2	12.9	13.2	12.1	11.7 cd
12. 11-3B.17	OR	1.4	30.1	11.7	20.7	20.5	16.9 b
13. 11-4A-14	OR	1.6	17.5	13.1	19.7	17.1	13.8 c
14. 15-3B.4	OR	1.3	26.8	9.3	17.9	14.1	13.9 bc
15. 15-4A.20	OR	3.1	27.9	22.3	20.9	33.2	21.5 a
Mean		1.4	10.5	15.4	9.1	9.0	
		C[*]	B	A	B	B	

Table II.1 Footnotes

*Means followed by the same letter are not significantly different at P= 0.05 according to Fisher's protected LSD. Capital letters are for comparison of cultivar means and lower case are for comparison of isolate means.
+CA= California, OR= Oregon, and TX= Texas.

Table II.2. Analysis of variance of percent diseased leaf area (%DLA) and area under disease progress curve (AUDPC) of five (exp. 1) and 12 (exp. 2) wheat cultivars of different geographic origin caused by 15 *S. tritici* isolates.

	Df			Mean Square			Pr>F		
	Exp.1	Exp.2		Exp.1	Exp.2		Exp.1	Exp.2	
Source	%DLA	%DLA	AUDPC	%DLA	%DLA	AUDPC	%DLA	%DLA	AUDPC
Model	77	182	182	380.6	31050.8	36840.5	0.0001	0.0001	0.0001
Error	222	6817	537	23.2	424.8	3200.5			
Rep	3	3	3	111.0	24229.6	169644.3	0.0030	0.0001	0.0001
Isolate	14	14	14	735.9	94609.8	123814.7	0.0001	0.0001	0.0001
Cultivar	4	11	11	1514.6	244482.6	261499.2	0.0001	0.0001	0.0001
Isolate* Cultivar	56	154	154	225.2	10055.3	10299.6	0.0001	0.0001	0.0001

Table II.3. Percent diseased leaf area of 12 wheat cultivars caused by 15 *S. tritici* isolates tested in the greenhouse.

Isolate (Origin)*	Cultivars# (Origin)*												
	1 (IN)	2 (UK)	3 (IN)	4 (TX)	5 (OR)	6 (CA)	7 (UK)	8 (OR)	9 (IN)	10 (TX)	11 (CA)	12 (TX)	Mean
1 (TX)	78.1	26.0	56.5	43.5	0.2	56.6	2.0	69.5	97.9	83.0	10.5	88.4	51.4 ab*
2 (TX)	80.5	35.7	38.0	46.0	0.1	57.9	1.8	65.6	93.5	82.7	7.1	87.2	49.7 b
3 (TX)	0.3	0.1	0.2	0.1	0.1	0.3	0.5	0.4	4.2	0.3	0.1	0.1	0.5 h
4 (CA)	8.9	38.9	61.4	17.8	0.3	41.3	61.3	26.0	91.0	19.0	0.1	44.0	34.0 cd
5 (CA)	76.8	73.9	57.4	57.8	0.9	48.8	67.5	54.5	91.2	51.0	1.0	58.2	53.3 a
6 (CA)	17.8	23.6	14.2	10.3	0.6	19.3	36.7	19.1	72.5	23.5	0.1	22.9	21.8 g
7 (CA)	36.1	46.5	39.1	23.2	0.2	37.6	44.7	37.4	96.0	35.1	0.1	39.2	36.1 c
8 (CA)	30.5	37.7	40.5	30.2	0.4	36.9	25.3	23.4	85.2	31.4	0.1	28.4	30.8 e
9 (CA)	30.7	43.6	10.5	13.9	0.1	25.8	28.9	12.3	67.7	12.3	0.1	16.6	21.8 g
10 (OR)	16.0	15.0	44.4	16.6	0.1	5.7	56.8	20.4	77.2	37.6	0.1	24.7	26.2 f
11 (OR)	36.2	45.8	18.4	11.1	0.1	12.3	70.6	53.7	89.6	26.1	0.1	4.8	30.5 e
12 (OR)	14.7	40.5	41.3	19.9	0.1	3.4	64.1	55.8	77.4	43.5	0.1	25.6	31.9 de
13 (OR)	12.5	25.6	59.4	19.2	0.1	5.5	67.8	48.5	90.5	25.6	0.1	14.6	30.7 e
14 (OR)	6.3	27.0	39.5	10.6	0.1	5.3	63.7	33.7	70.6	19.7	0.2	2.2	23.4 g
15 (OR)	62.7	58.5	59.4	26.4	5.6	47.0	78.7	55.6	89.3	54.6	6.3	72.9	51.4 ab
Mean	34.1	35.5	38.7	23.1	0.6	26.7	44.7	38.4	79.8	36.9	1.7	35.4	
	E*	DE	C	G	H	F	B	C	A	CD	H	DE	

Table II.3 Footnotes

*CA= California, IN= Indiana, OR= Oregon, TX= Texas, and UK= United Kingdom.
#Cultivars one through 12 are, respectively, Auburn, Avalon, Clark, Collin, Gene, Inia 66R, Longbow, Madsen, Monon, Siouxland 89, Tadinia, and Tam 105.
†Means followed by the same letters are not significantly different at P= 0.05 according to Fisher's protected LSD test. Capital letters are for comparison of cultivar means and lower case are for comparison of isolate means.

Table II.4. Area under disease progress curve of 12 wheat cultivars caused by 15 *S. tritici* isolates tested in the greenhouse.

Isolate (Origin)*	Cultivar* (Origin)*												
	1 (IN)	2 (UK)	3 (IN)	4 (TX)	5 (OR)	6 (CA)	7 (UK)	8 (OR)	9 (IN)	10 (TX)	11 (CA)	12 (TX)	Mean
1 (TX)	295.5	121.4	110.5	89.5	0.6	136.2	15.0	159.7	391.2	267.7	20.9	342.0	162.5 ab*
2 (TX)	311.5	118.5	91.7	127.5	5.4	162.7	13.8	166.5	421.0	310.0	18.8	321.5	172.4 a
3 (TX)	0.3	0.3	0.3	0.3	0.3	1.3	0.8	2.8	9.5	2.3	0.3	0.3	1.6 h
4 (CA)	21.9	72.9	122.5	33.7	0.7	92.4	123.8	49.6	249.5	41.2	1.9	104.6	76.2 cde
5 (CA)	250.6	199.5	132.1	119.5	3.8	128.3	133.5	83.5	340.3	172.8	3.6	112.3	140.0 b
6 (CA)	46.0	55.4	21.0	22.0	0.7	28.0	55.0	43.6	224.3	62.3	0.8	49.2	50.7 fg
7 (CA)	85.8	82.8	62.8	59.8	0.8	56.3	82.8	44.8	328.3	64.3	1.0	46.1	76.7 cde
8 (CA)	85.1	111.4	95.9	87.5	3.4	96.4	65.5	53.7	271.3	62.8	0.8	65.1	83.2 c
9 (CA)	67.4	70.7	14.2	33.5	0.7	38.9	47.0	19.5	188.8	16.3	0.6	19.2	43.1 g
10 (OR)	25.7	26.9	103.3	46.3	1.3	26.6	104.1	64.5	243.3	109.5	3.2	58.5	67.8 cdef
11 (OR)	44.5	56.5	31.4	29.8	1.1	33.0	121.0	71.0	254.8	63.5	0.6	16.2	60.2 defg
12 (OR)	23.4	51.8	68.3	40.2	0.8	18.2	109.0	89.5	252.0	94.5	1.1	49.5	66.5 cdef
13 (OR)	22.7	54.7	98.5	39.6	3.8	38.0	169.0	159.6	243.0	78.7	1.3	41.0	79.1 cd
14 (OR)	9.4	43.3	41.6	24.7	0.8	21.8	155.3	83.3	228.3	60.3	1.3	4.9	56.2 efg
15 (OR)	220.3	204.5	176.0	104.1	26.3	138.0	207.0	141.3	338.5	258.8	13.5	219.3	170.6 a
Mean	100.7	84.7	78.0	57.2	3.4	67.7	93.5	82.2	265.6	111.0	4.6	96.6	
	BC*	CDE	DE	F	G	EF	BCD	CDE	A	B	G	BCD	

Table II.4 Footnotes

*CA= California, IN= Indiana, OR= Oregon, TX= Texas, and UK= United Kingdom.
#Cultivars one through 12 are, respectively, Auburn, Avalon, Clark, Collin, Gene, Inia 66R, Longbow, Madsen, Monon, Siouxland 89, Tadinia, and Tam 105.
†Means followed by the same letters are not significantly different at P= 0.05 according to Fisher's protected LSD test. Capital letters are for comparison of cultivar means and lower case are for comparison of isolate means.

Table II.5. Analysis of variance of percent diseased leaf area (% DLA) and area under disease progress curve (AUDPC) of wheat cultivars and *S. tritici* isolates of three locations.

		Mean Square		Pr > F	
Source	Df	% DLA	AUDPC	% DLA	AUDPC
Model	11	419.2	5978.8	0.0001	0.0001
Error	24	52.8	196.2		
Rep	3	44.2	3644.7	0.4868	0.0001
Isolate	2	588.7	8268.5	0.0004	0.0001
Cultivar	2	1189.9	13698.9	0.0001	0.0001
Isolate*Cultivar	4	230.4	2724.4	0.0086	0.0001
All own[a] vs others[b]	1	858.6	9295.0	0.0005	0.0001

[a]own indicates isolate-cultivar combinations of same location.
[b]others indicates cultivars from other locations.

Table II.6. Observed and expected percent diseased leaf area (% DLA) and area under disease progress curve (AUDPC) caused by *S. tritici* on cultivars of the same and different origin.*

Isolate origin	Cultivar origin: California (CA)*		Oregon (OR)		Texas (TX)		Mean	
	%DLA	AUDPC	%DLA	AUDPC	%DLA	AUDPC	%DLA	AUDPC
CA*	17.12	37.40	14.55	25.38	28.22	65.10	19.96	42.63
	13.06+	**27.34**	**17.14**	**31.06**	**29.68**	**69.50**	B	Y
OR	7.11	24.60	22.50	53.59	24.21	74.39	17.94	50.86
	11.74	**32.61**	**15.41**	**37.06**	**26.67**	**82.91**	B	Y
TX	20.84	56.72	22.07	55.90	49.96	162.30	30.96	91.64
	20.26	**58.76**	**26.59**	**66.77**	**46.04**	**149.39**	A	X
Mean	15.02b	39.57y	19.71b	44.96y	34.13a	100.60x	22.95	61.71

	% DLA	AUDPC
All "own"	43.19**	84.45**
All "others"	19.5	50.36

Table II.6 Foot notes

*California and Oregon each include six isolates and two wheat cultivars and Texas includes three isolates and three cultivars.
†Bold figures are expected values calculated as isolate mean for a location X cultivar mean for isolates from that location/grand mean.
[a]"own" indicates isolate-cultivar combination of same location.
[b]"others" indicates isolate-cultivar combination of different locations.
Figures followed by the same letter are not significantly different at P= 0.05 according to Fisher's protected LSD test. Capital letters are for comparison of isolate means and lower case are for comparison of cultivar means.
**indicates significantly different at P <0.01 level by linear contrast.

Adaptation of *Septoria tritici* to Four Winter Wheat Cultivars

CHAPTER III

SUMMARY

Four populations of *Septoria tritici*, secured from four winter wheat cultivars grown in a field experiment, were evaluated on seedlings of the same wheat cultivars under greenhouse conditions. Significant cultivar effects, spore population effects and cultivar X spore population interactions were demonstrated. The interaction term was fairly large compared to the main effect of spore population, and it is likely that the significant interaction was due to increased virulence of *S. tritici* to its cultivar of origin. Linear contrast of "own" spore population (inoculation with spore population obtained from the same cultivar) versus "others" (inoculation with spore populations obtained from other cultivars) showed that own produced significantly more disease on the cultivar of origin than the others. The observed amount of disease was always higher than the expected amount of disease for each spore population on its cultivar of origin. These results indicate cultivar-specific adaptation of *S. tritici*. Spore populations obtained from the two susceptible cultivars

indicated general adaptation to all of the test cultivars, whereas the spore population secured from the most resistant cultivar showed specific adaptation to its cultivar of origin. Consequences of increased virulence and adaptation of *S. tritici* to wheat cultivars are discussed.

INTRODUCTION

Septoria tritici blotch (STB) of wheat (*Triticum aestivum* L.), caused by the fungus *Mycosphaerella graminicola* (Fuckel) J. Schrot. in Cohn (anamorph= *Septoria tritici* Roberge in Desmaz.), is a serious wheat disease in all wheat growing areas of the world and causes severe yield losses (Coakley et al., 1985; Dubin, 1985; Eyal et al., 1973; Eyal et al., 1987; King et al., 1983; Schuh, 1990; Shipton et al., 1971; Van Ginkel & Scharen, 1987; Zelikovitch & Eyal, 1991; Ziv & Eyal, 1978). Early maturity, short stature, high nitrogen levels and changing husbandry practices apparently all favor STB (King, et al., 1983; Narvaez, 1957; Shipton, 1966; Shipton et al., 1971), and the introduction of short, early maturing cultivars grown at high nitrogen levels may have predisposed wheat to STB (Eyal, 1981; Shaner, et al., 1975).

Pathogenic variability in virulence (King et al., 1983; Shipton et al., 1971) and physiologic specialization (Eyal et al.,1973; Eyal, 1981; King et al., 1983; Saadaoui, 1987) have been documented in populations of *S. tritici*. Eyal et al. (1985) reported significant isolate X cultivar interactions, indicating the existence of specific virulence genes among isolates, and that virulence frequencies varied considerably among regions and within countries. They also noted that *Septoria* isolates were more virulent to cultivars

originating from the same location, and that isolates secured from tetraploid wheats were more virulent to tetraploid cultivars than to hexaploid cultivars.

Genetic variability in *S. tritici* may evolve through sexual recombination, since the teleomorph of the fungus has been reported from many countries (Brown et al., 1978; Madariaga, 1986; Madariaga et al., 1989; Mehta, 1989; Scott et al., 1988; Sanderson, 1972; Shaw & Royle, 1989). A high level of genetic variability within *S. tritici* populations has been reported, which may allow for rapid selection of clones that are virulent to wheat cultivars with pathotype-specific resistance (McDonald & Martinez, 1990).

Since there is evidence of genetic variability in populations of *S. tritici,* there might be selection of one or more host-specific, virulent pathotype(s) depending on the wheat cultivar grown over time in a given location. Resistance genes with large effect have often been incorporated into modern crop cultivars to control diseases. However, these resistance genes were only temporarily effective in many host-pathogen system due to increases in the frequency of pathogen populations with matching virulence genes corresponding to introduced resistance genes (Parlevliet, 1981). Further, many researchers have suggested that if pathogen populations are variable, increased

aggressiveness (an increase in the extent of disease in a compatible host) could be selected for over time (Alexander et al., 1985; Caten, 1974; James & Fry, 1983; Latin, et al., 1981), thus reducing the stability of resistance (Clifford & Clothier, 1974; Latin et al., 1981; Newton, 1989).

Several authors have studied whether selection for increased aggressiveness/virulence occurred after several cycles of pathogen reproduction on the same host (Alexander et al., 1985; Clifford & Clothier, 1974; James & Fry, 1983; Jinks & Grindle, 1963; Latin et al., 1981; Leonard, 1969; Osburn et al., 1986; Osburn et al., 1987). It was reported that *Phytophthora infestans* isolates were more aggressive to the potato cultivars from which they were isolated, but they did not show evidence of increased aggressiveness after passage through the same host genotype several times (Caten, 1974; Jefferey et al., 1962; Jinks & Grindle, 1963; Latin et al., 1981). A similar result was obtained in the *Hordeum vulgare-Puccinia hordei* pathosystem (Clifford and Clothier, 1974). James & Fry (1983) also worked on potato late blight, but found no evidence for increased aggressiveness of the pathogen to the potato cultivar from which the isolates were obtained, nor any evidence of selection after several cyclings through the host. However, Leonard (1969) found that, after cycling of *Puccinia graminis* f. sp. *avenae*

through two oat (*Avena sativa*) cultivars, each subpopulation became more virulent to their "own" cultivar than the "other".

King et al. (1983) reviewed several studies which indicated adaptation of both *S. nodorum* and *S. tritici* isolates to their host species. After three cycles of passage of *S. nodorum* isolates through barley, a wheat isolate showed increased virulence to barley (Rufty et al., 1981) and passage of a barley isolate through wheat resulted in greater sporulation than after passage through barley. Successive passage of a wheat isolate of *S. nodorum* through a grass host led to increased virulence to several grass species and a decrease in virulence to the original wheat cultivar from where it was isolated (Harrower, 1977). On the other hand, Ao & Griffiths (1976) reported that, after passage through grasses, both *S. nodorum* and *S. tritici* from wheat were less virulent to the original host than isolates obtained immediately from wheat. Osburn et al. (1986 & 1987) found some changes in host preference when barley-adapted and wheat-adapted *S. nodorun* isolates were serially cycled through both barley and wheat, but concluded that the result was due to cross-contamination, with subsequent selection.

Our study presented in chapter II indicated that *S. tritici* isolates were more virulent to wheat cultivars at

the same location from where the isolates were obtained, which suggests selection of location-specific virulent pathotypes. It is also important to know if there are cultivar-specific pathotypes among pathogen populations in a certain location before planning breeding programs for disease resistance and deployment of resistant cultivars. The present study was undertaken to determine pathogenic variability of four populations of *S. tritici* secured from four wheat cultivars and to determine if there is specific adaptation of *S. tritici* populations to the cultivars from which they were isolated.

MATERIALS AND METHODS

Host

Four winter wheat cultivars (Gene, Madsen, Malcolm and Stephens) were raised in 10 cm pots filled with pot mixture (1 part peat; 1 part sand; 1 part loam soil; 2 parts # 8 pumice with no fines) under artificial lighting in the greenhouse. Approximately 15 seeds of a cultivar were sown in each pot. At seven days after seeding, the plants were thinned to ten per pot. The plants were fertilized twice with 50 ml/pot of Miracle-Gro (ammoniacal nitrogen 6.8%; urea nitrogen 8.2%; available phosphoric acid P_2O_5 30%; soluble potash K_2O 15%; boron 0.02%; copper 0.07%; iron 0.15%; manganese 0.05%; molybdenum 0.0005% and zinc 0.06%) at the rate of 15g/L of water after two weeks and five weeks of seeding. The plants were sprayed and/or fumigated with insecticide (insecticidal soap 15ml/L and/or nicotine smoke) as necessary to control aphids and fumigated weekly with fungicide (fenarimol) to control powdery mildew. Greenhouse temperature was maintained within a range of 20-25°C.

Pathogen

Randomly chosen, diseased leaf samples were collected from each of the four plots of each wheat cultivar planted in a randomized complete block design with four replications at

the Oregon State University Botany and Plant Pathology Field Laboratory. Each plot was 4.9 X 1.5 m.

Leaf sections of about 5 cm with distinct disease lesion(s) were washed with sterilized water, and then surface sterilized with 5% sodium hypochlorite solution for about 30 seconds. After sterilization, the leaf sections were pressed with blotting paper to remove excess water. Two sterilized leaf sections were then taped on a sterilized glass slide. This slide with leaf sections was then placed inside a petri dish previously mounted with blotting paper and soaked with sterile water, and was subsequently placed on a laboratory bench overnight under ambient conditions. On the following day, using a stereoscope, cirrhi from individual pycnidia were transferred with a needle onto a PDA plate fortified with 50mg/L streptomycin solution. After two days of fungal growth, a loopful of sterile water was added with a bacterial transfer loop to the fungal spore mass and streaked onto another PDA plate with the same bacterial transfer loop. After three-to-four days, single pycnidiospore isolates were obtained from an individual colony and transferred onto PDA slants. About five-to-seven isolates were secured from each cultivar per replication, and each isolate was from a different pycnidium. Each isolate was maintained on silica gel in a refrigerated cryovial.

Twenty isolates were selected from each cultivar, representing all replications. Before plant inoculation, crystals of silica gel were plated onto potato dextrose agar (PDA) plates fortified with 50 mg/L of streptomycin. After three-to-four days, the blastospores of the fungus grew on and around the crystals of silica gel and were streaked with a bacterial transfer loop onto the surface of the same PDA plates. Two days after streaking, each individual isolate was transferred on a separate PDA slant and spread uniformly with a bacterial transfer loop. After three days, the fungus was used for inoculations. The blastospores of all 20 isolates of the fungus from each cultivar were mixed before inoculation and the spore concentration was adjusted to 10^6/ml.

Inoculation

At twenty-one days after seeding, 16 pots of plants (four cultivars X four replications) were sprayed with a hand sprayer using 75 ml of spore suspension. These amounts were sufficient to inoculate the plants to run-off. After inoculation, the plants were kept in a moist chamber covered with a polyethylene sheet for 96 hours. High humidity (95% or above) was maintained using a humidifier inside the moist chamber. A temperature of 20 ± 5°C was maintained inside the moist chamber and greenhouse; this was possible since the experiments were conducted during the period from November

to March (1991-1993). The pots were subsequently placed on a greenhouse bench in a randomized complete block design until disease scoring.

Disease assessment

Three weeks after inoculation percent diseased leaf area (% DLA) of the second leaf from the base of each of ten plants in each pot was estimated visually. In addition, % DLA per pot was estimated at two-day intervals beginning 13 days after inoculation, when some spore population/cultivar combinations showed visible disease lesions. Area under disease progress curve (AUDPC) was then calculated following the method described by Shaner & Finney (1977).

Statistical Analysis

Data were analyzed as a factorial analysis of variance for a randomized complete block design using the General Linear Models procedure of the SAS statistics package (SAS Institute, 1988). The sources of variance were replications, cultivars, spore populations and cultivar X spore population interactions. Fisher's protected LSD (P= 0.05) was used for making multiple comparisons among cultivar and pathogen population means. Linear contrasts were performed to determine if there was cultivar-specific adaptation of *S. tritici* populations. For each cultivar, a

contrast of "own" (spore population originally isolated from that cultivar) versus "others" (mean of spore populations isolated from the other three cultivars) was done. In addition, a linear contrast of all own cultivar/spore population combinations versus all other cultivar/spore population combinations was performed. For the purpose of comparison, expected % DLA (spore population mean for acultivar X cultivar mean for spore population from that cultivar/grand mean) for own and others was calculated to investigate adaptation of *S. tritici* to source cultivars.

RESULTS

Analysis of variance of both % DLA and AUDPC indicated statistically significant differences among *S. tritici* populations and among cultivars. Cultivar X spore population interaction was also highly significant, though a small amount of variation is attributed to the interaction term compared to combined main effects of cultivar and spore populations (Tables III.1 & III.2). Means comparison showed that the cultivars Malcolm and Stephens are most susceptible, Madsen is moderately susceptible and Gene is resistant (Tables III.3 & III.4; Figure III.1). *S. tritici* populations obtained from the cultivars Malcolm and Stephens were more virulent, followed by populations isolated from the cultivars Gene and Madsen (Tables III.3 & III.4; Figure III.1).

S. tritici populations obtained from Malcolm and Stephens were significantly more virulent to their respective own cultivars than the others for both methods of disease assessment. *S. tritici* populations secured from the resistant cultivar Gene and moderately susceptible cultivar Madsen revealed no significant differences between own and others, although disease severity on the cultivar Gene was always higher for the own versus the other combinations (Tables III.1-4). When all own versus all others were compared, all own produced more disease than the all others,

although this contrast was statistically significant for AUDPC but not for % DLA, taken once at 21 days after inoculation (Tables III.1-4).

For both disease assessment procedures, the observed values were always higher than the expected values for each own spore population X cultivar combination (Tables III.3 & III.4).

DISCUSSION

Linear contrast analysis showed that spore populations obtained from Malcolm and Stephens were more virulent to their respective hosts than the other spore populations. In addition, the mean of all own spore populations produced significantly more disease on their own cultivars than was produced by the mean of all other spore populations for AUDPC, and the observed value for each own cultivar X spore population combination was always higher than the expected value. These results suggest that *S. tritici* populations were specifically adapted to their cultivar of origin. Similarly, Eyal et al. (1985) reported that *S. tritici* isolates obtained from tetraploid wheats were more virulent to tetraploid wheats than hexaploid wheats. Our results of a previous study presented in chapter II also showed that *S. tritici* isolates were more adapted to the cultivars of the same geographic origin than those from other locations.

It has been suggested that the presence of cultivar X isolate interaction in a host-pathogen system is an indication of cultivar-specific adaptation (Latin et al., 1981). Host-pathogen interactions in this study were highly significant, though the amount of variation explained by the interaction terms were small compared to the main effects of cultivar. But, for both methods of disease assessment, the

amounts of variation attributed to the interaction term relative to the main effect of spore population were fairly large.

It is likely that the spore population X cultivar interaction in our study was due to increased virulence of the spore populations to their cultivar of origin. Similarly, Clifford & Clothier (1974) found significant host-pathogen interaction with *Puccinia hordei* and barley with nonhypersensitive resistance, and this interaction term was attributed to increased aggressiveness of the pathogen to its cultivar of origin. However, significant host-pathogen interactions have been demonstrated by several researchers (Knott & Mundt, 1991; Krupinsky, 1977; Kuhn et al., 1978; Latin et al., 1981; Milus & Line, 1980; Newton, 1989; Parlevliet, 1977; Whitney & Mackey, 1989) when the cause of interaction could not be attributed to an increase in aggressiveness/virulence of pathogens on their cultivars of origin.

S. tritici populations isolated from the susceptible cultivars Malcolm and Stephens were more virulent compared to the populations obtained from the resistant cultivar Gene and the moderately susceptible cultivar Madsen. Similarly, we found in our previous study (Chapter II) that *S. tritici* isolates and wheat cultivars collected from Texas were,

respectively, more virulent and susceptible than the isolates and wheat cultivars of other locations. It is likely that *S. tritici* populations isolated from Malcolm and Stephens have general adaptation to all of the test cultivars, whereas the *S. tritici* population obtained from Gene has specific adaptation to its cultivar of origin. Knott and Mundt (1991) indicated that significant differences in wheat leaf rust populations were due to the selective effects of source cultivars.

James & Fry (1983) pointed out that expression of pathogen adaptation would depend on the diversity in the pathogen population, and this diversity could be increased by using different single isolates, populations derived from a mixture of isolates, or populations resulting from mutagenesis. Our pathogen populations were each a mixture of twenty isolates which were collected from a randomized and replicated field experiment. The sexual stage of the pathogen is very active in Oregon, and restriction fragment length polymorphism analysis indicates a high degree of genetic variation in populations of *S. tritici* (McDonald & Martinez, 1990). Thus, we used diverse pathogen populations which allowed *S. tritici* to become specifically adapted to their own cultivars. It is likely that *S. tritici* populations used in the present study were previously selected for adaptation to the test cultivars, since these

cultivars have been grown at the same location for several years. In addition, we made isolations late in the season when there would have been opportunity for selection of *S. tritici* populations adapted to their own cultivars. Further, Knott and Mundt (1991) suggested that the source cultivars had different selective effects on wheat leaf rust populations, which might have changed the composition of the pathogen population.

As our study indicates adaptation of *S. tritici* to the cultivars of origin, the long-term use of a resistant cultivar would be endangered. Thus, gene rotation and cultivar mixtures might be useful for STB disease management to sustain the stability of resistance.

REFERENCES

Alexander, HM, Groth, JV, Roelfs, AP, 1985. Virulence changes in *Uromyces appendiculatus* after five asexual generations on a partially resistant cultivar of *Phaseolus vulgaris*. *Phytopathology* 75, 449-453.

Ao, HC, Griffiths E, 1976. Change in virulence of *Septoria nodorum* and *Septoria tritici* after passage through alternative hosts. *Transactions of the British Mycological Society* 66, 219-224.

Brown JS, Kellock AW, Paddick RG, 1978. Distribution and dissemination of *Mycosphaerella graminicola* (Fuckel) Schroeter in relation to the epidemiology of speckled leaf blotch of wheat. *Australian Journal of Agricultural Research* 29, 1139-1145.

Caten, CE, 1974. Intra-racial variation in *Phytophthora infestans* and adaptation to field resistance for potato blight. *Annals of Applied Biology* 77, 259-270.

Clifford, BC, Clothier RB, 1974. Physiologic specialization of *Puccinia hordei* on barley hosts with nonhypersensitive resistance. *Transactions of the British Mycological Society* 63(3), 421-430.

Coakley, SM, McDaniel, LR, Shaner, G, 1985. Model for predicting severity of *Septoria tritici* blotch on winter wheat. *Phytopathology* 75, 1245-1251.

Dubin, HJ, 1985. Occurrence and importance of *Septoria nodorum* and *Septoria tritici* in the Andean countries of South America. Beltsville, Md: USDA Agriculture Research Service no. 12.

Eyal, Z, 1981. Integrated control of Septoria disease of wheat. *Plant Disease* 65(9), 763-768.

Eyal, Z, Amiri, Z, Wahl, I, 1973. Physiologic specialization of *Septoria tritici*. *Phytopathology* 63, 1087-1091.

Eyal, Z, Scharen, AL, Huffman, MD, 1985. Global insights into virulence frequencies of *Mycosphaerella graminicola*. *Phytopathology* 75, 1456-1462.

Eyal, Z, Scharen, AL, Prescott, JM, and van Ginkel, M, 1987. The septoria disease of wheat: Concept and methods of disease management. Mexico, D. F. : CIMMYT.

Harrower, KM, 1977. Specialization of *Leptosphaeria nodorum* to alternative graminacious hosts. *Transactions of the British Mycological Society* 65, 101-103.

James, RV, Fry, WE, 1983. Potential for *Phytophthora infestans* populations to adapt to potato cultivars with rate reducing resistance. *Phytopathology* 73, 984-988.

Jefferey, SIB, Jinks, JL, Grindle, M, 1962. Intraracial variation in *Phytophthora infestans* and field resistance to potato blight. *Genetica* 32, 323-338.

Jinks, JL, Grindle, M, 1963. Changes induced by training in *Phytophthora infestans*. *Heredity* 8(3), 245-264.

King, JE, Cook, RJ, Melville, SC, 1983. A review of septoria diseases of wheat and barley. *Annals of Applied Biology* 103, 345-373.

Knott, EA, Mundt, CC, 1991. Latent period and infection efficiency of *Puccinia recondita* f. sp *tritici* populations isolated from different wheat cultivars. *Phytopathology* 81, 435-439.

Krupinsky, JM, 1989. Variability in *Septoria musiva* in aggressiveness. *Phytopathology* 79, 413-416.

Kuhn, RC, Ohm, HW, Shaner CE, 1978. Slow leaf-rusting resistance in wheat against twenty-two isolates of *Puccinia recondita*. *Phytopathology* 68, 652-656.

Latin, RX, Mackenzie, DR, Cole, HJr, 1981. The influence of host and pathogen genotypes on the apparent infection rates of potato late blight epidemics. *Phytopathology* 71, 82-85.

Leonard, KJ, 1969. Selection in heterogenous populations of *Puccinia graminis* f. sp. *avenae*. *Phytopathology* 59, 1851-1857.

Madariaga RB, 1986. Presentia en Chile de *Mycosphaerella graminicola* (Fuckel) Schroeter, estado sexuada de *Septoria tritici* Rob. ex. Desm. *Agricultura Tecnica* 46, 209-211.

Madariaga, RB, Gilchrist, DG, Martensen, AN, 1989. Presence and role of *Mycosphaerella graminicola* in California wheat. *Phytopathology* 79, 1141.

McDonald, BA, Martinez, JP, 1990. DNA restriction fragment length polymorphisms among *Mycosphaerella graminicola* (anamorph *Septoria tritici*) isolates collected from a single wheat field. *Phytopathology* 80, 1368-1373.

Mehta, YR, 1989. Occurrence of *Septoria tritici* and its perfect state in Brazil. *Proceedings of the Third International Workshop on Septoria diseases of Cereals*, 1989. Swiss Federal Research Station for Agronomy, Zurich, 34-35.

Milus, EA, Line, RF, 1980. Characterization of resistance to leaf rust in Pacific Northwest wheat lines. *Phytopathology* 70, 167-172.

Narvaez, I, 1957. *Studies of Septoria leaf blotch of wheat.* Indiana: Purdue University, West Lafayette.

Newton, AC, 1989. Genetic adaptation of *Erysiphe* f. sp. *hordei* to barley with partial resistance. *Journal of Phytopathology* 126, 133-148.

Osburn, AE, Caten, CE, Scott, PR, 1987. Adaptation of *Septoria nodorum* to wheat or barley on detached leaves. *Plant Pathology* 36, 565-576.

Osburn, AE, Scott, PR, Caten, CE, 1986. The effect of host passaging on the adaptation of *Septoria nodorum* to wheat or barley. *Plant Pathology* 35, 135-145.

Parlevliet, JE, 1977. Evidence of differential interaction in the polygenic *Hordeum vulgare-Puccinia hordei* relation during epidemic development. *Phytopathology* 67, 776-778.

Parlevliet, JE, 1981. Stabilizing selection in crop pathosystems: an empty concept or a reality? *Euphytica* 30, 259-269.

Rufty, RC, Herbert, TT, Murphy, CF, 1981. Variation in virulence in isolates of *Septoria nodorum*. *Phytopathology* 71, 593-596.

Saadaoui, EM, 1987. Physiologic specialization of *Septoria tritici* in Morocco. *Plant Disease* 71, 153-155.

Sanderson, FR, 1972. A Mycosphaerella species as the ascogenous state of *Septoria tritici* Rob. and Desm. *New Zealand Journal of Botany* 10, 707-710.

SAS Institute, Inc., 1988. SAS/STAT User's Guide, Release 6.03 edition. SAS Institute, Inc., Cary, NC.

Schuh, W, 1990. Influence of tillage systems on the disease intensity and spatial pattern of Septoria leaf blotch. *Phytopathology* 80, 1337-1340.

Scott, PR, Sanderson, FR, Benedikz, PW, 1988. Occurrence of *Mycosphaerella graminicola*, teleomorph of *Septoria tritici* on wheat in U.K. *Plant Pathology* 37, 285-290.

Shaner, G, Finney, RE, 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. *Phytopathology* 67, 1051-1056.

Shaner, G, Finney, RE, and Patterson, FL, 1975. Expression and effectiveness of resistance in wheat to Septoria leaf blotch. *Phytopathology* 65, 761-766.

Shaw, MW, and Royle, DJ, 1989. Airborne inoculum as a source of *Septoria tritici* (*Mycosphaerella graminicola*) infections in the winter wheat crops in the UK. *Plant Pathology* 38, 35-43.

Shipton, WA, 1966. Septoria leaf and glume blotch of wheat. *Journal of Agriculture, Western Australia* 7, 160-163.

Shipton, WA, Boyd, WJR, Rosielle, AA, and Shearer, BL, 1971. The common septoria diseases of wheat. *Botanical Review* 37, 231-262.

Van Ginkel, M, Scharen, AL, 1987. Generation mean analysis and heritabilities of resistance to *Septoria tritici* in durum wheat. *Phytopathology* 77, 1629-1633.

Whitney, ED, Mackey, BE, 1989. Differences in aggressiveness of *Erwinia caratovora* subsp. *betavasculorum* strains and their reactions to sugar beet cultivars. *Plant Disease* 73, 220-222.

Zelikovitch, N, Eyal, Z, 1991. Reduction in pycnidial coverage after inoculation of wheat with mixtures of isolates of *Septoria tritici*. *Plant Disease* 75, 907-910.

Ziv, O, Eyal, Z, 1978. Assessment of yield component losses caused in plants of spring wheat cultivars by selected isolates of *Septoria tritici*. *Phytopathology* 68, 791-796.

Table III.1. Analysis of variance of percent diseased area of four wheat cultivars caused by four populations of *S. tritici* tested in the greenhouse.

Source	Df	Mean Square	Pr > F
Model	18	964.64	0.0001
Error	45	67.29	
Replication	3	60.02	0.4526
SPOP[#]	3	806.99	0.0001
CVAR[$]	3	4228.48	0.0001
SPOP * CVAR	9	230.79	0.0027
SPOP1[@] vs others[*] (on Gene)	1	0.13	0.9655
SPOP2 vs others (on Madsen)	1	62.34	0.3410
SPOP3 vs others (on Malcolm)	1	848.40	0.0009
SPOP4 vs others (on Stephens)	1	1113.61	0.0002
All own[+] vs all others	1	77.76	0.2881

[#]SPOP= a mixture of 20 isolates from one of four cultivars grown in the field.
[$]CVAR= cultivars.
[@]SPOP1, SPOP2, SPOP3, and SPOP4 were obtained from the cultivars Gene, Madsen, Malcolm, and Stephens, respectively
[*]Others indicates populations which were isolated from cultivars other than the one indicated in parentheses.
[+]All own indicates mean of all isolates tested on cultivars from which they were isolated.

Table III.2. Analysis of variance of area under disease progress curve of four wheat cultivars caused by four populations of *S. tritici* tested in the greenhouse.

Source	Df	Mean Square	Pr > F
Model	18	8180.22	0.0001
Error	45	300.91	
Replication	3	1308.97	0.0122
SPOP[#]	3	5808.21	0.0001
CVAR[$]	3	37696.10	0.0001
SPOP * CVAR	9	1422.68	0.0004
SPOP1[@] vs others[*] (on Gene)	1	0.41	0.7518
SPOP vs others (on Madsen)	1	1392.13	0.0216
SPOP3 vs others (on Malcolm)	1	7363.13	0.0002
SPOP4 vs others (on Stephens)	1	8008.33	0.0001
All own[+] vs all others	1	4804.20	0.0015

[#]SPOP= a mixture of 20 isolates from one of four cultivars grown in the field.
[$]CVAR= cultivar.
[@]SPOP1, SPOP2, SPOP3, and SPOP4 were obtained from the cultivars Gene, Madsen, Malcolm, and Stephens, respectively.
[*]Others indicates populations which were isolated from cultivars other than the one indicated in parentheses.
[+]All own indicates mean of all isolates tested on cultivars from which they were isolated.

Table III.3.Observed and expected percent diseased leaf area caused by four populations of *S. tritici* on their "own"* and "other"+ host cultivars.

Cultivar	SPOP1[$]	SPOP2	SPOP3	SPOP4	Mean[@]	Own	Others
Gene	0.49	0.39	0.13	0.34	0.34c	0.49[ns]	0.29
	0.28[++]	**0.24**	**0.43**	**0.41**			
Madsen	24.26	27.49	41.25	25.35	29.66b	27.49[ns]	30.29
	24.61	**20.65**	**37.64**	**35.34**			
Malcolm	31.38	20.18	49.62	44.13	36.45a	49.62[**]	31.90
	30.24	**25.37**	**46.26**	**43.44**			
Stephens	27.05	22.11	36.05	50.65	33.88a	50.65[**]	28.40
	28.11	**23.58**	**43.00**	**40.37**			
Mean[@]	20.69B	17.36C	31.65A	29.72A	24.94	32.06[ns]	22.72

[++]Bold figures are the expected values, which were calculated as spore population mean for a cultivar X cultivar mean for spore population from that cultivar/grand mean.
*"Own" indicates disease reaction of *S.tritici* populations on cultivars from which the isolates were obtained.
+"Other" indicates disease reactions of *S. tritici* populations on the cultivars from which the isolates were not obtained.
[@]Figures followed by the same letter are not significantly different at P= 0.05 according to Fisher's protected LSD test.
[**]Significant at P= < 0.01 by linear contrast.
[ns]Non-significant at P= 0.01 by linear contrast.
[$]SPOP1, SPOP2, SPOP3, and SPOP4 were obtained from the cultivars Gene, Madsen, Malcolm, and Stephens, respectively.

Table III.4. Observed and expected area under disease progress curve caused by four populations of *S. tritici* on their "own"* and "other"+ host cultivars.

	SPOP1[$]	SPOP2	SPOP3	SPOP4	Mean[@]	Own	Others
Gene	2.60	2.68	1.30	2.72	2.32d	2.60[ns]	2.23
	2.04[++]	**1.54**	**3.00**	**2.69**			
Madsen	55.88	43.50	86.75	52.50	59.66c	43.50[ns]	65.04
	52.71	**39.50**	**77.27**	**69.17**			
Malcolm	100.50	75.63	149.25	123.00	112.09a	149.25[**]	99.71
	99.04	**74.22**	**145.17**	**129.95**			
Stephens	79.00	56.50	111.50	134.00	97.75b	134.00[**]	82.33
	86.37	**64.72**	**126.58**	**113.33**			
Mean[@]	59.49B	44.58C	87.20A	78.06A	67.33	82.34[**]	62.33

[++]Bold figures are the expected values, which were calculated as spore population mean for a cultivar X cultivar mean for spore population from that cultivar/grand mean.
*"Own" indicates disease reaction of *S.tritici* populations on cultivars from which the isolates were obtained.
+"Other" indicates disease reactions of *S. tritici* populations on the cultivars from which the isolates were not obtained.
[@]Figures followed by the same letter are not significantly different at P= 0.05 according to Fisher's protected LSD test.
[**]Significant at P= < 0.01 by linear contrast.
[ns]Non-significant at P= 0.01 by linear contrast.
[$]SPOP1, SPOP2, SPOP3, and SPOP4 were obtained from the cultivars Gene, Madsen, Malcolm, and Stephens, respectively.

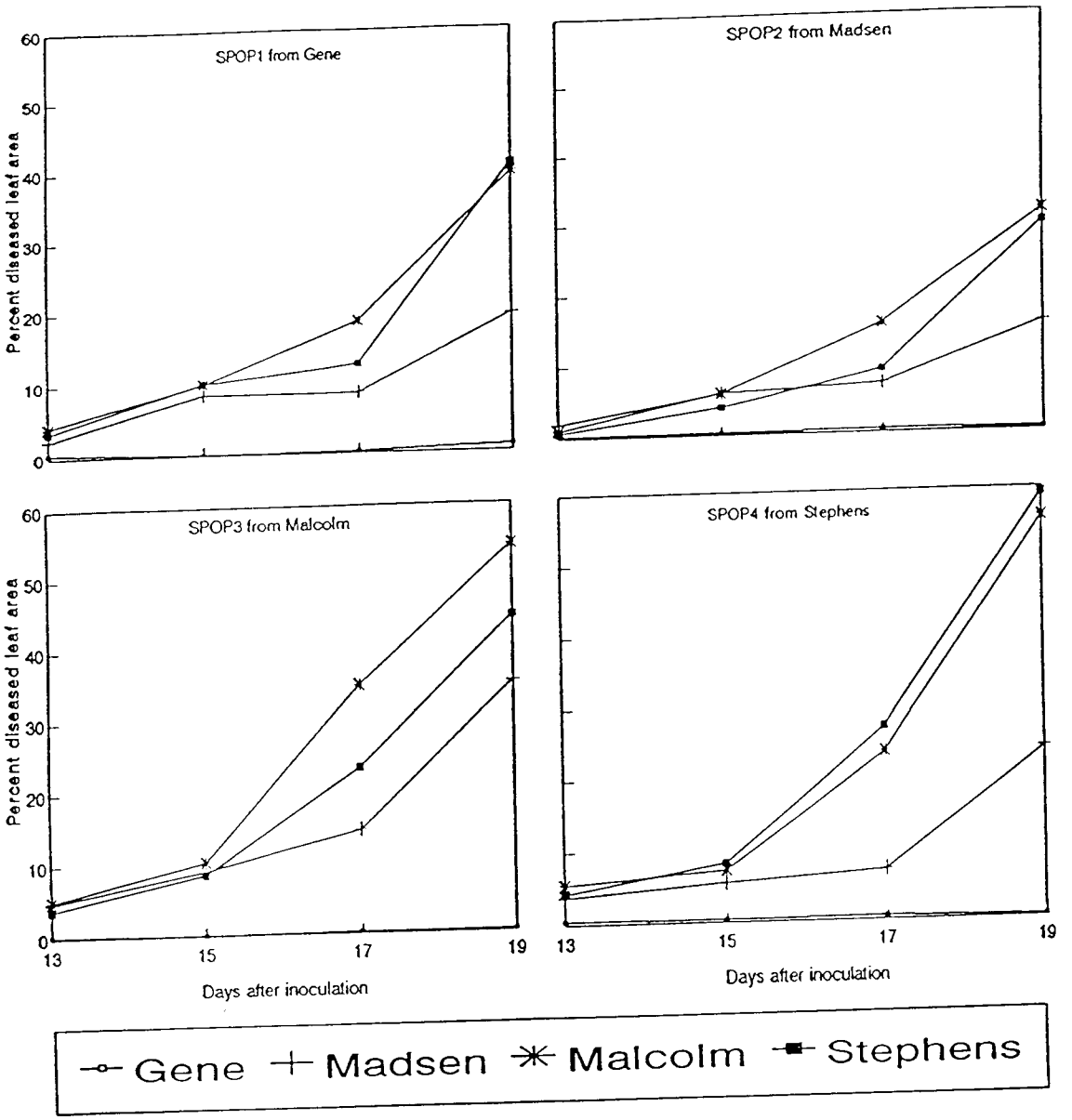

Fig. III.1.Disease progress curves of four winter wheat cultivars caused by four populations of *S. tritici* on their "own"* and "other"* host cultivars in the greenhouse."Own" indicates disease reaction of *S. tritici* populations on cultivars from which the isolates were obtained."Other" indicates disease reaction of *S. tritici* populations on cultivars from which the isolates were not obtained. SPOP = Spore population.

BIBLIOGRAPHY

Agrios, GN,1988. *Plant Pathology*, 3rd. edn. Academic Press, Inc.

Alexander, HM, Groth, JV, Roelfs, AP,1985. Virulence changes in *Uromyces appendiculatus* after five asexual generations on a partially resistant cultivar of *Phaseolus vulgaris*. *Phytopathology* 75, 449-453.

Anonymous, 1965. *Losses in Agriculture*. Washington: United States Department of Agriculture. Handbook no. 291.

Ao, HC, Griffiths E, 1976. Change in virulence of *Septoria nodorum* and *Septoria tritici* after passage through alternative hosts. *Transactions of the British Mycological Society* 66, 219-224.

Bayles, RA, Parry, VWL, Clark, T, 1985. Resistance of winter wheat varieties to *Septoria tritici*. *Journal of National Institute of Agricultural Botany*. 17(1), 21-26.

Brokenshire, T, 1976. The reaction of wheat genotypes to *Septoria tritici*. *Annals Applied Biology* 82, 415-423.

Brown JS, Kellock AW, Paddick RG, 1978. Distribution and dissemination of *Mycosphaerella graminicola* (Fuckel) Schroeter in relation to the epidemiology of speckled leaf blotch of wheat. *Australian Journal of Agricultural Research* 29, 1139-1145.

Camacho-Casas, MA, 1989. *Inheritance of Septoria Leaf Blotch Resistance and the Possible Influence of Heading Date and Plant Height on the Disease Expression in a Winter Wheat Cross*. Oregon: Oregon State University.

Camacho-Casas, MA, 1986. *Yield Loss Assessment in Non-protected Winter Wheat Varieties*. Oregon: Oregon State University.

Caten, CE, 1974. Intra-racial variation in *Phytophthora infestans* and adaptation to field resistance for potato blight. *Annals of Applied Biology* 77, 259-270.

Centro Internacional de Mejoramiento de Maiz y Trigo (CIMMYT), 1972. *Annual Report* 1970-71. 114pp.

Clifford, BC, Clothier RB, 1974. Physiologic specialization of *Puccinia hordei* on barley hosts with nonhypersensitive resistance. *Transactions of the British Mycological Society* 63(3), 421-430.

Coakley, SM, McDaniel, LR, Shaner, G, 1985. Model for predicting severity of *Septoria tritici* blotch on winter wheat. *Phytopathology* 75, 1245-1251.

Cordo, CA, Perello, A, Arriaga, H, Pascar A, 1989. Study of the stability of *Mycosphaerella graminicola* (*Septoria tritici*) isolates.In Fried, MF, eds. *Proceedings of the Third International Workshop on Septoria Diseases of Cereals*, 1989. Swiss Federal Research Station for Agronomy, Zurich, 55-58.

Djerbi, M, Ghodbane, A, Daaloul, A, Varughese, G, 1974. Identification de sources de resistance a la septoriose du ble (*Septoria tritici* Rob. et Desm.). *Annals of Phytopathology* 6, 495-496.

Dubin, HJ, 1985. *Occurrence and importance of* Septoria nodorum *and* Septoria tritici *in the Andean countries of South America*. Beltsville, Md: USDA Agriculture Research Service no.12.

Dubin, HJ, Rajaram, S, 1981. The strategy of the International Maize and Wheat Improvement Center (CIMMYT) for breeding disease resistant wheat: An international approach. In: Jenkyn JF, Plumb RT, eds. *Strategies for the Control of Cereal Disease*. Oxford: Blackwell Scientific Publications, 27-35.

El Ahmed, A, Maamouri, AR, Deghaies, M, 1984. Wheat breeding for septoria resistance in Tunisia: Identification of resistance genotypes. *Rachis* 3(2), 20-23.

Eyal, Z, 1972. Effect of Septoria leaf blotch on the yield of spring wheat in Israel. *Plant Disease Reporter* 56, 983-986.

Eyal, Z, 1981. Integrated control of Septoria disease of wheat. *Plant Disease* 65, 763-768.

Eyal, Z, Amiri, Z, Wahl, I, 1973. Physiologic specialization of *Septoria tritici*. *Phytopathology* 63, 1087-1091.

Eyal, Z, Scharen, AL, Huffman, MD, 1985. Global insights into virulence frequencies of *Mycosphaerella graminicola*. *Phytopathology* 75, 1456-1462.

Eyal, Z, Scharen, AL, Prescott, JM, Van Ginkel, M, 1987. *The Septoria Disease of Wheat: Concept and Methods of Disease Management*. Mexico, D. F.: CIMMYT.

Eriksson, J, 1894. Ueber die Specialisurung des Parasitimus bei den Getreiderostpilzen. *Ber. Dt. Bot. Ges.* 12, 292-331.

Fellows, H, 1962. Effect of light, temperature and fertilizer on infection of wheat leaves by *Septoria tritici*. *Plant Disease Reporter* 46, 846-848.

Hamid, AH, Ayers, JE, Hill, RR, 1982. Host isolate interactions in corn inbreds inoculated with *Cochliobolus carbonum* race 3. *Phytopathology* 72, 1169-1173.

Harrower, KM, 1977. Specialization of *Leptosphaeria nodorum* to alternative graminacious hosts. *Transactions of the British Mycological Society* 65, 101-103.

Hilo, HM, Bever, WM, 1957. Inoculation, oversummering, and suscept-pathogen relationship of *Septoria tritici* on *Triticum* species. *Phytopathology* 47, 474-480.

James, RV, Fry, WE 1983. Potential for *Phytophthora infestans* populations to adapt to potato cultivars with rate reducing resistance. *Phytopathology* 73, 984-988.

Jefferey, SIB, Jinks, JL, Grindle, M, 1962. Intraracial variation in *Phytophthora infestans* and field resistance to potato blight. *Genetica* 32, 323-338.

Jenkins, JEE, Morgan, W, 1969. The effect of Septoria diseases on the yield of winter wheat. *Plant Pathology* 18, 152-156.

Jinks, JL, Grindle, M, 1963. Changes induced by training in *Phytophthora infestans*. *Heredity* **8(3),** 245-264.

King, JE, Cook, RJ, Melville, SC, 1983. A review of septoria diseases of wheat and barley. *Annals of Applied Biology* 103, 345-373.

Knott, EA, Mundt, CC, 1991. Latent period and infection efficiency of *Puccinia recondita* f. sp *tritici* populations isolated from different wheat cultivars. *Phytopathology* 81, 435-439.

Krupinsky, JM, Craddock, JC, Scharen, AL, 1977. Septoria resistance in wheat. *Plant Disease Reporter* 61, 632-636.

Kuhn, RC, Ohm, HW, Shaner CE, 1978. Slow leaf-rusting resistance in wheat against twenty-two isolates of *Puccinia recondita*. *Phytopathology* 68, 652-656.

Latin, RX, Mackenzie, DR, Cole, HJr., 1981. The influence of host and pathogen genotypes on the apparent infection rates of potato late blight epidemics. *Phytopathology* 71, 82-85.

Leonard, KJ, 1969. Selection in heterogenous populations of *Puccinia graminis* f. sp. *avenae*. *Phytopathology* 59, 1851-1857.

Madariaga RB, 1986. Presentia en Chile de *Mycosphaerella graminicola* (Fuckel) Schroeter, estado sexuada de *Septoria tritici* Rob. ex. Desm. *Agricultura Tecnica* 46, 209-211.

Madariaga, RB, Gilchrist, DG, Martensen, AN, 1989. Presence and role of *Mycosphaerella graminicola* in California wheat. *Phytopathology* 79, 1141.

Marshall, D, 1985. Geographic distribution and aggressiveness of *Septoria tritici* on wheat in the United States. *Phytopathology* 75, 1319.

McDonald, BA, Martinez, JP, 1990. DNA restriction fragment length polymorphisms among *Mycosphaerella graminicola* (anamorph *Septoria tritici*) isolates collected from a single wheat field. *Phytopathology* 80, 1368-1373.

Mehta, YR, 1989. Occurrence of *Septoria tritici* and its perfect state in Brazil. *Proceedings of the Third International Workshop on Septoria Diseases of Cereals*, 1989. Swiss Federal Research Station for Agronomy, Zurich, 34-35.

Mew, TW, 1987. Current status and future prospects of research on bacterial blight of rice. *Review of Phytopathology* 25, 359-382.

Mew, TW, Vera Cruz, CM, Medalla, ES, 1992. Changes in race frequency of *Xanthomonas oryzae* pv. *oryzae* in response to rice cultivars planted in Philippines. *Plant Disease* 76, 1029-1032.

Milus, EA, Line, RF, 1980. Characterization of resistance to leaf rust in Pacific Northwest wheat lines. *Phytopathology* 70, 167-172.

Morales, IN, 1957. *Studies on Septoria Leaf Blotch of Wheat*. Indiana: Purdue University.

Morales, IN, 1958. Studies on *Septoria* leaf blotch of wheat. *Dissertation Abstract* 18, 357-358.

Narvaez, I, 1957. *Studies of Septoria leaf blotch of wheat*. Indiana: Purdue University.

Narvaez, I, Caldwell, RM, 1957. Inheritance of resistance of leaf blotch of wheat caused by *Septoria tritici*. *Phytopathology* 47, 529.

Nelson, LR, and Marshall, D, 1990. Breeding wheat for resistance to *Septoria nodorum* and *Septoria tritici*. *Advances in Agronomy* 44, 257-277.

Newton, AC, 1989. Genetic adaptation of *Erysiphe* f. sp. *hordei* to barley with partial resistance. *Journal of Phytopathology* 126, 133-148.

Osburn, AE, Caten, CE, Scott, PR, 1987. Adaptation of *Septoria nodorum* to wheat or barley on detached leaves. *Plant Pathology* 36, 565-576.

Osburn, AE, Scott, PR, and Caten, CE, 1986. The effect of host passaging on the adaptation of *Septoria nodorum* to wheat or barley. *Plant Pathology* 35, 135-145.

Parlevliet, JE, 1977. Evidence of differential interaction in the polygenic *Hordeum vulgare-Puccinia hordei* relation during epidemic development. *Phytopathology* 67, 776-778.

Parlevliet, JE, 1981. Stabilizing selection in crop pathosystems: an empty concept or a reality? *Euphytica* 30, 259-269.

Parlevliet, JE, Zadoks, JC, 1977. The integrated concept of disease resistance; a new view including horizontal and vertical resistance in plants. *Euphytica* 26, 5-21.

Rillo, AO, and Caldwell, RM, 1966. Inheritance of resistance to *Septoria tritici* in *Triticum aestivum* subs. *vulgare* Bulgaria 88. *Phytopathology* 56, 897.

Rosielle, AA, 1972. Sources of resistance in wheat to speckled leaf blotch caused by *Septoria tritici*. *Euphytica* 21, 152-161.

Rosielle, AA, Boyd, WJR, 1985. *Genetics of host- pathogen interactions to the Septoria species of wheat*. Beltsville, Md.: USDA Agriculture Research Service no. 12.

Rosielle, AA, Brown, AG, 1979. Inheritance, heritability and breeding behaviour of three sources of resistance to *Septoria tritici* in wheat. *Euphytica* 28, 385-392.

Rufty, RC, Herbert, TT, Murphy, CF, 1981. Variation in virulence in isolates of *Septoria nodorum*. *Phytopathology* 71, 593-596.

Saadaoui, EM, 1987. Physiologic specialization of *Septoria tritici* in Morocco. *Plant Disease* 71, 153-155.

Saari, EE, and Wilcoxson, RD, 1974. Plant disease situation of high yielding dwarf wheats in Asia and Africa. *Annual Review of Phytopathology* 12, 49-68.

Saari, EE, 1974. Results of studies on *Septoria* in the Near East ant Africa In: *Proceedings of the Fourth FAO/Rockefeller Foundation Wheat Seminar*, 1974. Tehran, Iran, 441.

Sanderson, FR, 1972. A Mycosphaerella species as the ascogenous state of *Septoria tritici* Rob. and Desm. *New Zealand Journal of Botany* 10, 707-710.

SAS Institute, Inc., 1988. SAS/STAT User's Guide, Release 6.03 edition. SAS Institute, Inc., Cary, NC.

Scharen, AL, and Eyal, Z, 1983. Analysis of symptoms on spring and winter wheat cultivars inoculated with different isolates of *Septoria nodorum*. *Phytopathology* 73, 143-147.

Scharen, AL, Sanderson, FR, 1985. Identification, distribution and nomenclature of the *Septoria* species that attack cereals. In: Scharen AL, ed. *Septoria of Cereals. Proceedings of Workshop*, 1983. Bozeman, Montana. United States Department of Agriculture, 37-41.

Schuh, W, 1990. Influence of tillage systems on the disease intensity and spatial pattern of Septoria leaf blotch. *Phytopathology* 80, 1337-1340.

Scott, PR, Sanderson, FR, and Benedikz, PW, 1988. Occurrence of *Mycosphaerella graminicola*, teleomorph of *Septoria tritici* on wheat in U.K. *Plant Pathology* 37, 285-290.

Shaner, G, and Finney, RE, 1976. Weather and epidemics of Septoria leaf blotch of wheat. *Phytopathology* 66, 781-785.

Shaner, G, Finney, RE, 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. *Phytopathology* 67, 1051-1056.

Shaner, G, Finney, RE, and Patterson, FL, 1975. Expression and effectiveness of resistance in wheat to Septoria leaf blotch. *Phytopathology* 65, 761-766.

Shaner, G, Stromberg EL, Lacy GH, Barker, KR, Pirone, TP, 1992. Nomenclature and concepts of pathogenicity and virulence. *Annual Review of Phytopathology* 30, 47-66.

Shaw, MW, Royle, DJ, 1989. Airborne inoculum as a source of *Septoria tritici* (*Mycosphaerella graminicola*) infections in the winter wheat crops in the UK. *Plant* Pathology 38, 35-43.

Shipton, WA, 1966. Septoria leaf and glume blotch of wheat. *Journal of Agriculture, Western Australia* 7, 160-163.

Shipton, WA, Boyd, WJR, Rosielle, AA, Shearer, BL, 1971. The common septoria diseases of wheat. *Botanical Review* 37, 231- 262.

Stakman, EC, Piemseisel, FJ, 1917. A new strain of *Puccinia graminis*. *Phytopathology* 7, 73.

Stewart, DM, Hafiz, A, Abdel Hak, T, 1972. Disease epiphytotic threats to high-yielding and local wheats in the Near East. FAO Plant Protection Bulletin 20, 50-57.

Tyagi, PD, Yoshi, LM, Renfro, BL, 1970. Reaction of wheat varieties to *Septoria tritici* and report of an epidemic in north-western Punjab. *Indian Phytopathology* 22, 175-178.

Van der Plank, JE, 1968. *Disease Resistance in Plants*, 2nd edn. New York: Academic Press.

Van Ginkel, M, and Scharen, AL, 1987. Generation mean analysis and heritabilities of resistance to *Septoria tritici* in durum wheat. *Phytopathology* 77, 1629-1633.

Van Ginkel, M, Scharen, AL, 1988. Host-pathogen relationships of wheat and *Septoria tritici*. *Phytopathology* 78, 762-766.

Van Shilfhout, CH, Arama, PF, Kema, GHJ, 1989. International survey of virulence of *Septoria tritici*. In Fried, MF, eds. *Proceedings of the Third International Workshop on Septoria Diseases of Cereals*, 1989. Swiss Federal Research Station for Agronomy, Zurich, 36-38.

Whitney, ED, Mackey, BE, 1989. Differences in aggressiveness of *Erwinia caratovora* subsp. *betavasculorum* strains and their reactions to sugar beet cultivars. *Plant Disease* 73, 220-222.

Yechilevich-Auster, M, Levi, E, Eyal, Z, 1983. Assessment of interactions between cultivated and wild wheats and *Septoria tritici*. *Phytopathology* 73, 1077-1083.

Zelikovitch, N, Eyal, Z, 1991. Reduction in pycnidial coverage after inoculation of wheat with mixtures of isolates of *Septoria tritici*. *Plant Disease* 75, 907-910.

Ziv, O, Eyal, Z, 1978. Assessment of yield component losses caused in plants of spring wheat cultivars by selected isolates of *Septoria tritici*. *Phytopathology* 68, 791-796.