Sources of and genetic action of resistance in barley to different virulence types of Pyrenophora teres, the causal organism of net blotch
by Michael Eidis Bjarko

A thesis submitted in partial fulfillment of the requirements for the degree of MASTER OF SCIENCE in Plant Pathology
Montana State University
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Abstract:
Research was initiated in February, 1977 to study the reactions of different barley lines to isolates of Pyrenophora teres.

Studies revealed that maximum sporulation of P. teres in culture could be obtained using V8 juice as a growth medium while incubating the cultures under an eight hour photoperiod of 17-18° C.

Twenty-six isolates of P. teres, fifteen from the Middle East and eleven from Montana, were separated into different virulence types based on the differential reactions of different barley lines. Seven distinct Middle East virulence types and five Montana virulence types could be distinguished.

Nine of these virulence types were used to screen 147 barley lines for resistance to P. teres. Nine lines were found to contain resistance to all nine virulence types. These lines were C.I. 1615, C.I. 4207, C.I. 5298, C.I. 5401, C.I. 5845, C.I. 7208, C.I. 9768, C.I. 13262 and Unitan. An additional twenty-two lines were found to contain resistance to eight of the nine virulence types of P. teres.

Crosses involving resistant and susceptible barley lines were made, and the resulting F2 populations were tested with different isolates of P. teres. It was found that at least five major genes exist in barley for resistance to P. teres. Four lines, C.I. 5791, C.I. 9819, Unitan and Steptoe each contain at least three genes for resistance.

Evidence for additive resistance to P. teres was found. Crosses between susceptible parents produced F2 seedlings which were more resistant than either parent to the isolate of P. teres used in the test.

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THE CAUSAL ORGANISM OF NET BLOTCH

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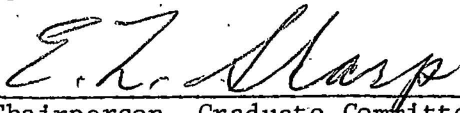
Approved:



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Bozeman, Montana

March, 1979

ACKNOWLEDGMENT

I express my deepest appreciation and thanks to the following people:

Dr. E.L. Sharp for his support, encouragement, and guidance while serving as my major professor.

Dr. T.W. Carroll, Dr. D.E. Mathre and Professor R.F. Eslick for their help and advice while serving on my graduate committee.

Dr. H.E. Bockelman for his patience, advice and encouragement throughout my graduate career.

My fellow graduate students for their understanding and support.

My parents, Albert and Marian, and my sisters, Ann, Mary Jo, Allison and Melissa, whose constant love and encouragement meant so much.

US-AID for financial support.

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ABSTRACT

Research was initiated in February, 1977 to study the reactions of different barley lines to isolates of *Pyrenophora teres*.

Studies revealed that maximum sporulation of *P. teres* in culture could be obtained using V8 juice as a growth medium while incubating the cultures under an eight hour photoperiod of 17-18° C.

Twenty-six isolates of *P. teres*, fifteen from the Middle East and eleven from Montana, were separated into different virulence types based on the differential reactions of different barley lines. Seven distinct Middle East virulence types and five Montana virulence types could be distinguished.

Nine of these virulence types were used to screen 147 barley lines for resistance to *P. teres*. Nine lines were found to contain resistance to all nine virulence types. These lines were C.I. 1615, C.I. 4207, C.I. 5298, C.I. 5401, C.I. 5845, C.I. 7208, C.I. 9768, C.I. 13262 and Unitan. An additional twenty-two lines were found to contain resistance to eight of the nine virulence types of *P. teres*.

Crosses involving resistant and susceptible barley lines were made, and the resulting F_2 populations were tested with different isolates of *P. teres*. It was found that at least five major genes exist in barley for resistance to *P. teres*. Four lines, C.I. 5791, C.I. 9819, Unitan and Steptoe each contain at least three genes for resistance.

Evidence for additive resistance to *P. teres* was found. Crosses between susceptible parents produced F_2 seedlings which were more resistant than either parent to the isolate of *P. teres* used in the test.

INTRODUCTION

Net blotch of barley, caused by *Pyrenophora teres*, is a disease common on all commercial varieties. It is found wherever barley is grown, and is especially severe under cool, humid conditions.

Changes in cultural practices, such as the extensive use of irrigation, minimum tillage, continuous cropping and early sowing of barley, and restrictions on chemical treatment, especially the mercury seed treatments, have produced an environment conducive to the growth and spread of this organism. The sowing of susceptible barley varieties then provides a potential for severe disease epidemic.

Net blotch, along with barley scald (*Rhyncosporium secalis*), has produced detrimental effects on the yield and malting quality of barley grown on the Fairfield Bench in Northcentral Montana, where yield losses in 1973 were estimated to be as high as 50% in some fields. During the 1977-78 growing season in California, infection by *P. teres* was so severe that fields were burned rather than harvested.

This study was initiated in order to find and evaluate sources of resistance to *P. teres*. Also, the relationship of different isolates of *P. teres* to different sources of resistance, and the genetics of resistance to *P. teres* was studied.

LITERATURE REVIEW

Pyrenophora teres Drechs. is the causal organism of net blotch of barley. The imperfect stage of this fungus, *Helminthosporium teres* Sacc., syn. *Drechslera teres* Ito., is placed in the class Hyphomycetes, based on the fact that the conidia are not borne in any form of pycnidium or acervulus (Talbot, 1971). The pigmentation of the mucelium and conidia place it in the family Dematiaceae. The genus *Helminthosporium* is recognized chiefly by the presence of brown, septate conidiophores and large, brown, phragmoid conidia (Talbot, 1972). The binomial *Helminthosporium teres* was first applied by Saccardo to a fungus collected on leaves of barley in 1881 (Drechsler, 1923). The fungus was described as having 3-septate condiophores arising in a group of five from a green substratum and bearing at the tip a single conidium. The conidia were described as dark green structures, thick walled, 4-5 septate, ellipsoidal or subcylindrical and tapering perceptibly toward the rounded ends.

The original genus *Helminthosporium* was extremely heterogeneous. Because of this, groups of species have been segregated from it and placed into more discriminating genera. The net blotch organism was placed by Ito in the genus *Drechslera*, based primarily on the origin, development and morphology of the condiophores and conidia (Schoemaker, 1962; Talbot, 1973). *Drechslera* differs from *Helminthosporium* in that the conidia develop at the tips of successive lateral proliferations

of the conidiophores and are pseudopleurogenous. They are phragmoid, cylindrical and subhyaline to pale brown. Conidial germtubes are lateral, usually amphigenous, and may arise from intercalary as well as terminal cells (Luttrell, 1977; Talbot, 1973; Schoemaker, 1962).

The conidiophores of *Pyrenophora teres* occur singly or in groups of two to three. They are straight to flexous, often swollen at the base and are pale to mid-brown or olivaceous in color. They measure 120-200 x 7-11 μm. The conidia are straight, cylindrical, with rounded apical cells and constricted septa. The basal cell is inflated, producing a subglobose appearance. The conidia are 1-11 septate, 30-175 x 15-23 μm and subhyaline to green fuligenous in color. They are sessile and germinate from any or all cells (Drechsler, 1923; Luttrell, 1951; Ellis, 1971). Among the Pyrenophora species, the inflated basal cell and constricted septa are unique to P. *teres*.

The perfect, or sexual, stage of this organism produces asci and ascospores, placing it in the subdivision Ascomycotina. The asci are bitunicate, placing it in the class Loculoascomycetes and the presence of pseudoparaphyses places it in the order Pleosporales (Talbot, 1971). Drechsler (1923) was the first to describe the ascigerous stage of *Pyrenophora teres*. He placed it in the genus *Pyrenophora*, based on the presence of setae on the perithecial surface.

The asci of P. *teres* are club shaped or subcylindrical, rounded at the apex and with a short stalk at the base. They are bitunicate,

and 180-274 x 30-61 µm. The ascospores are light brown, 18-28 x 43-61 µm, ellipsoidal, with three transverse septa and one or two longitudinal septa in the median cells, but not in the terminal cells. The spores are much constricted at the septa, with germtubes forming from any or all cells (Drechsler, 1923; Smedegard-Petersen, 1972).

Pyrenophora teres also produces pycnidia on host tissue and in culture. They are globose to pear-shaped, 64-172 µm in diameter, with a thin fragile wall. They are yellow to brown in color, depending on age. The spores are hyaline, spherical or ellipsoidal, non-septate and 1.0-1.9 x 1.4-3.2 µm (Smedegard-Petersen, 1972). The function of these pycnidiospores is not known, although it is thought they may act as spermatia (McDonald, 1963; Smedegard-Petersen, 1972).

The heterothallic nature of *P. teres* has been shown (McDonald, 1963; Smedegard-Petersen, 1972). Monoconidial or monoascosporic isolates did not produce fertile perithecia when paired with other than their opposite mating types. Mature perithecia of *P. teres* were produced by growing mixed suspensions of two compatible single-spore isolates. A 1:1 ratio of the mating types was obtained when ascospore progeny from perithecia were backcrossed to parental isolates (McDonald, 1963).

P. teres produces three types of spores; ascospores, conidia and pycnidiospores. The ascospores and conidia will readily infect susceptible host tissue. Suspensions of pycnidiospores have never

produced symptoms (Smedegard-Petersen, 1972). All three spore types will germinate and grow on artificial media, although the growth of pycnidiospores is initially very slow. Once mycelium has been established, growth of cultures from pycnidiospores is normal. Suspensions of homogenized mycelium from cultures originating from any of the three spore types will readily infect barley leaf tissue. Conidiophores and conidia are produced from the symptomatic tissue.

The fungus overwinters as mycelium in infested plant debris and infected seed. It also overwinters as perithecia on infested straw.

Although seedling infection resulting from seed is known to occur, the extent to which this happens is unknown and appears to be affected very much by the environment. In Denmark, Smedegard-Petersen (1974) found 18.5% and 13.8% of the seedlings of the susceptible barley cultivars Wing and Lauda, respectively, showing symptoms due to infected seed. Piening (1968), in Canada, found only 0.5% to 1.5% of barley seedlings showing symptoms resulting from seed infection. Seedling infection from infected seed occurs at 10-15° C, but not above 20° C (Drechsler, 1923). Lesions may develop on the coleoptile as a result of infected seed, but more often it is the first, or plumular, leaf which is attacked. Kenneth (in Shipton, et al., 1973) has observed that infection of the hull is inconsequential. For seedling infection to occur the caryopsis itself must be infected.

Even with low percentages (0.5-1.5%) of the plants infected

via seed-borne inoculum, Piening (1968) observed that where leaves with primary infection were not removed, twice as many plants were infected twenty-eight days after emergence as on plots where infected leaves were removed as soon as they were noticed. It therefore appears that the chief danger of seed-borne inoculum is in providing a potential for disease spread throughout the growing season and the ultimate production of contaminated plant debris which could provide abundant inoculum the following year.

Infected straw is of major importance as a source of primary inoculum. Piening (1968) observed 42% of the plants infected with *P. teres* in a field where the previous year the straw and stubble were lightly disced, whereas only 8% of the plants were infected in a field where the straw and stubble were ploughed under during the previous year.

Early in the growing season, when environmental conditions are favorable, conidia are produced on infested plant debris. In addition, perithecia release asci and ascospores. The perithecia are also often densely covered with conidia which form on the tips of setae and conidiophores between the setae. The relative importance of the spore types as primary inoculum is unknown and appears to vary with geographic location and environment (Shipton, 1966; Smedegard-Petersen, 1972; Shipton, et al., 1973; Piening, 1968).

The conidia and ascospores will both germinate readily on host

tissue. The germtubes give rise to appressoria-like structures, and infection may occur after a moist period of as few as five hours, although 10-30 hours of moisture appears necessary for maximum infection (Shipton, et al., 1973). The penetration hyphae pass through the epidermal cells and enlarge slightly upon passing through the inner cell wall. Development of the hyphae is then intercellular, with cell death occurring in advance of the fungus. The respiration rate of susceptible barley leaves increases, significantly exceeding that of non-infected leaves after sixteen hours. The respiration rate continues to increase, reaching a maximum rate coinciding with the appearance of visible necrosis, after which the rate sharply decreases. Externally, symptoms may be evident within two days of infection (Shipton, et al., 1973; Smedegard-Petersen, 1976, 1977b).

Conidiophores arise directly from between epidermal cells or stomata and produce conidia on the surface of primary lesions. These conidia then cause secondary infection of the developing foliage. This process is repeated throughout the growing season, as long as conditions remain favorable. Complete necrosis of the leaves may occur by flowering time (Schaller, 1955; Shipton, et al., 1973).

Symptoms induced by P. *teres* occur on the blades and sheaths of the leaves and may extend to the flowers and grains. The initial lesions appear as minute spots or streaks which increase in size to form narrow, dark brown, longitudinal and transverse streaks,

producing a net-like pattern. This netting appearance is characteristic of *P. teres* and it is from this appearance that the disease gets its common name of net blotch (Drechsler, 1923; Dickson, 1956).

Isolates of *P. teres* have been collected which do not produce the characteristic netting symptoms on host tissue, but instead produce spot lesions very similar to those produced by *Pyrenophora japonicum*. These isolates were considered to be *P. teres* rather than *P. japonicum* because they could be crossed with isolates of *P. teres* that produce the netting symptoms (McDonald, 1967). Smedegard-Petersen (1971) suggested the designation *Pyrenophora teres* Drechs. forma *maculata* forma nova for the spot type of *P. teres*, and *Pyrenophora teres* Drechs. forma *teres* for the net type.

The symptoms induced by the spot form of *P. teres* also occur on the blades and sheaths of the leaves and consist of dark brown, elliptical or fusiform lesions measuring up to 3 x 6 mm in size and surrounded by a chlorotic zone of varying width (Smedegard-Petersen, 1971).

The spot form of *P. teres* has been found to be more prevalent in Denmark than the net form (Smedegard-Petersen, 1972). Tekauz and Buchannon (1977) have observed the spot form in the Winnipeg, Mannitoba region and believe that its distribution indicates this biotype may have been introduced into the region on contaminated barley seed. In crosses between spot and net types of *P. teres*, it was found that

symptom expression is controlled by two independent allelic pairs of genes (Smedegard-Petersen, 1976).

Pyrenophora teres has been found to produce two toxins, both low molecular weight peptides (Smedegard-Petersen, 1976, 1977a). Except for the netting or spotting lesions, both toxins incite most of the symptoms incited by the pathogen itself, i.e.; chlorosis, necrosis and increased respiration. The two toxins do not seem to determine pathogenicity, however, but rather they contribute to the virulence of the organism (Smedegard-Petersen, 1976, 1977a, 1977b).

Under field conditions, the netted lesions of *P. teres* will coalesce with time, forming longitudinal stripes. These stripes can extend the length of the blade and into the leaf sheath. The stripes resemble those normally produced by *Pyrenophora graminea*, the causal organism of barley stripe disease. Often the symptoms of the two diseases are virtually indistinguishable. The morphology of both the sexual and asexual stages of these two species is also very similar, although there are some differences. The ascospores of *P. graminea* are larger than those of *P. teres*, and only four are produced in each ascus (Schoemaker, 1962). The conidia of *P. teres* are constricted at the septa and have an enlarged basal cell. Those of *P. graminea* do not have an enlarged basal cell and are not constricted at the septa. The conidia of *P. graminea* measure 31-111 x 11-24 μm and are 1-7 septate, and are smaller than those of *P. teres* (Drechsler, 1923;

Luttrell, 1951). *P. teres* also sporulates more readily in culture than *P. graminea*.

The variability in these morphological characteristics makes it difficult to distinguish between the two species using the morphological differences as a key (Smedegard-Petersen, 1976; Metz, 1978). The problem is further complicated by the fact that isolates of *P. teres* and *P. graminea* can be crossed in culture and perithecia containing viable ascospores can be obtained (Smedegard-Petersen, 1976). The most reliable method of distinguishing between the two species is in their mode of infection. *P. teres* will cause infection and symptom development when sprayed on the foliage of susceptible barley, while *P. graminea* cannot infect barley in this manner. *P. graminea* is a floral infecting pathogen and becomes systemic, while *P. teres* does not systemically infect barley.

Net blotch is found wherever barley is grown. It is of economic importance in North and South America, Europe, Australia, the Middle East and Africa (Shipton, et al., 1973). The effect on grain yield and quality has frequently been studied. Stakman (1922) estimated losses of 5% and 3% statewide for Iowa and South Dakota, respectively. Shipton (1966) observed a 17.4% decrease in yield in control plots as compared to plots protected with the fungicide Maneb. He also observed a significant reduction in malting quality. Piening and Kaufmann (1969) measured yield losses of 53.3% and a marked reduction in

kernel number per spike, indicating that the effect began in the early stages of plant growth. Caddel and Wilcoxson (1975) reported yield losses of 26% due to *P. teres*. Smedegard-Petersen (1974) reported yield losses of 9.0% and 11.1% in the barley cultivars Wing and Lauda, respectively, due mainly to reduction in grain size and weight.

Cultural changes have led to a recent increase in the prevalence of net blotch. Changes resulting in an increase in overwintering inoculum, such as minimum tillage and continuous cropping, and changes which have helped produce an environment more conducive to disease spread, i.e., irrigation, early sowing, and the planting of susceptible varieties have all helped to produce a situation which lends itself to potential disease epidemics (Smedegard-Petersen, 1971; Shipton, et al., 1973).

These cultural practices are more beneficial than harmful. Control of this disease must therefore utilize other methods. Foliar sprays are effective, but the cost is prohibitive. Seed treatment is helpful, but will not completely control the disease. The use of resistant varieties is the most efficient and desirable means of controlling net blotch. Resistant varieties, in addition to suffering less yield loss, also slow the spread of disease during the growing season and provide less infected plant debris, thus reducing the source of primary inoculum for the following year.

Resistant varieties apparently have no effect on spore germination,

the number of germtubes produced, the length and branching of germtubes or the number of penetrations (Keeling and Banttari, 1975). Inhibition of hyphal growth is pronounced after penetration and many infections do not progress beyond the penetrated cell. The organism therefore causes fewer and smaller lesions on resistant plants than on susceptible plants. Sporulation of the fungus on excised leaves of resistant plants was also found to be less than on leaves of susceptible plants (Keeling and Banttari, 1975). Tekauz and Buchannon (1977) found that the time required for sporulation of conidia on excised leaves varied from less than twenty-four hours with susceptible varieties to ninety-six hours for resistant varieties.

The first extensive screening of barley lines for sources of resistance was done by Schaller and Wiebe (1952). A total of 4,526 barley lines was tested to California isolates of *P. teres*. Twenty-five highly resistant lines were found, with the greatest percentage of these originating in Manchuria. Buchannon and McDonald (1965) tested 6,174 barley lines for resistance to net blotch. Of these they found forty lines, seventeen originating in Ethiopia, which were resistant in the seedling stage to isolates of *P. teres* from Canada, Mexico and the United States. Khan and Boyd (1969a) used seventeen Australian isolates to screen 142 barley lines which has previously been reported to be resistant to net blotch. Twelve highly resistant and twenty-two resistant lines, from both Manchuria and Ethiopia,

were found, with the Ethiopian lines generally exhibiting a higher degree of resistance than the Manchurian lines. Eighty barley lines, out of 2,608 lines tested, were found by Caddel and Wilcoxson (1975) to be resistant to Moroccan isolates of *P. teres*. Metcalfe, et al. (1978), evaluated 226 Ethiopian barley lines and found this region to be a good source of resistance to isolates of *P. teres* from Canada.

Isolates from various geographic areas differ significantly in pathogenicity. The physiologic specialization of *P. teres* became apparent with the screening of different barley lines. The lines found by Schaller and Wiebe (1952) to be resistant to California isolates of *P. teres* were susceptible to Canadian isolates (Buchannon and Mc-Donald, 1965). Most barley lines resistant to *P. teres* elsewhere in the world proved to be susceptible or moderately susceptible to Mor-occan isolates (Caddel and Wilcoxson, 1975). In general, the spot form of *P. teres* found in Canada is less virulent than the net types when lesion characteristics and extent of chlorosis are compared (Tekauz and Mills, 1974). This is not the case in Denmark, where the spot form is more prevalent than the net form (Smedegard-Petersen, 1971, 1976). Khan and Boyd (1969a) separated isolates of *P. teres* from Western Australia into three different virulence types using two differential varieties.

The first study to determine the genetics of resistance to *P. teres* was done by Schaller (1955). He found that Tifang contains a single

incompletely dominant gene for resistance effective against a California isolate. The symbol Pt was suggested for this gene. Mode and Schaller (1958) designated the resistance gene in Tifang Pt_1. They found C.I. 4797, C.I. 739 and C.I. 4929 contain a single gene for resistance, designated Pt_2, and C.I. 2750 and C.I. 4922 contain two genes for resistance, designated Pt_2 and Pt_3. It was found that Pt_1 and Pt_2 are closely linked, with a 2.57% recombination, Pt_3 is independent of Pt_1 and Pt_2. Khan and Boyd (1969c) found C.I. 2330, C.I. 4797 and Tifang to contain a single gene for resistance to a Western Australian isolate of *P. teres*. C.I. 9819 and C.I. 5791 contained two dominant genes for resistance to this isolate. All five varieties have a common gene for resistance to this isolate of *P. teres*.

Tifang and C.I. 4797 carry linked genes, Pt_1 and Pt_2, respectively, for resistance to a California isolate of *P. teres*. These two lines also carry an allelic gene for resistance to isolates of *P. teres* from Western Australia. Since neither Tifang nor C.I. 4797 carry the gene Pt_3, the common gene effective against the Australian isolate would need to be a fourth gene. This gene was designated Pt_a by Khan and Boyd (1969c).

Bockleman, et al. (1977), utilizing trisomic analysis, found Tifang to contain a single dominant gene for resistance effective against a Tunisian isolate of *P. teres*. The gene was located on chromosome 3. C.I. 7584 also contains a single dominant gene for

resistance, located on chromosome 2, effective against this isolate. C.I. 9819 contains two dominant genes, on chromosomes 3 and 5, effective against this isolate of *P. teres*.

Environment appears to affect the expression of resistance to *P. teres*. High inoculation and post-inoculation temperatures bring about a breakdown in resistance (Khan and Boyd, 1969; Tekauz and Buchannon, 1977). Light intensity, high pre-inoculation temperatures and spore concentration will also affect the expression of resistance in some varieties (Khan and Boyd, 1969b).

Consistency of expression of resistance is also a varietal characteristic and is dependent on the genetic background of the host. This is especially important when lines are crossed. The penetrance and expressivity of the resistance genes are highly dependent upon the genetic background of the parents used in the cross, even the background of a susceptible parent (Khan, 1969; Khan and Boyd, 1969b, 1969c). Khan (1969), using C.I. 5791 as a resistant parent and Atlas and Dampier as susceptible parents showed that F_1 host reaction and F_2 segregation patterns are considerably influenced by the choice of susceptible parent, with less resistance being expressed in the Atlas x C.I. 5791 cross than the Dampier x C.I. 5791 cross.

Chapter 1

THE EFFECT OF DIFFERENT GROWTH MEDIA ON THE SPORULATION OF *Pyrenophora teres*

For all inoculations done in this study, condial suspensions were used as inoculum. Since an adequate concentration of conidia in the suspension is needed to obtain a satisfactory infection, and hence good symptom development, it is important that the cultures of *P. teres* produce adequate amounts of conidia. In this experiment, different types of growth media were tested to determine which type would be most favorable for sporulation of *P. teres*.

Materials and Methods

One isolate of *P. teres*, Pt S (Sidney, Mt.), was used in the experiment. Eight different types of growth media were tested, V8 juice agar, Potato dextrose agar (Difco), Modified Eckert's Medium, Czapeck's agar (Difco), Barley leaf extract agar, Mycophil agar (BBL), Lima bean agar (Difco), and Rice cereal agar.[1]

Six replications of the experiment were carried out. For the initial transfer of *P. teres* to the different agar types, single spore transfers from leaf tissue were made. Transfers for subsequent replications were done as mass spore transfers using a dissecting needle, and

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[1]Recipes of these agar types are included in the appendix.

transfering spores to four spots on each petri plate. Transfers to fresh plates were made using spores only from plates of the same agar type. Spores were transferred to four plates of each agar type, three of these to be used for counting and the fourth as a source for the next transfer.

Spores were counted seven days after transferring. Counts were made by flooding the plates with distilled water, scraping the spores loose with a microscope slide and straining the suspension through three layers of cheesecloth. A total of 65 ml of distilled water was used per three plates. The spore concentration of the resulting suspension was then measured using a Howard Mold Counting Chamber (Hausser Scientific). Three counts were made, with the average of these used as the spore concentration for that agar type.

The cultures were grown in an incubation chamber with a 12 hour photoperiod and a temperature of 17-18° C.

Results and Discussion

Table 1 shows the spore concentrations for each agar type at each transfer and also the average number of spores per ml for the different agar types. An analysis of variance was performed on the data in this table, as shown below.

SOURCE	SS	DF	MS	F	$F_{.05}$
Total	244.293×10^8	47	----	----	----
Agar Type	132.745×10^8	7	18.964×10^8	6.8	2.25
Error	111.548×10^8	40	2.789×10^8	----	----

The results indicate that there is a significant difference in the sporulation of *P. teres* with respect to agar type.

To determine which averages were significantly different, a Duncan's Multiple Range test was utilized. The results of this test are indicated in Table 1.

Lima bean agar (LBA), V8 juice agar (V8), Mycophil agar (MyA) and Barley leaf extract agar (BLEA) all allowed abundant sporulation of *P. teres*. Potato dextrose agar (PDA), Modified Eckert's Medium (MEM) and Rice cereal agar (RCA) were less favorable for sporulation, while Czapeck's agar (CzA) was least suitable as a growth medium.

The results indicate that the type of growth medium used has a significant effect on the sporulation of *P. teres*. *P. teres* sporulates well enough on LBA, V8, BLEA and MyA that any of these should be adequate as a growth medium for this organism.

Table 1-1. Differential sporulation (spores/ml) of *P. teres* on eight types of growth media.

MEDIUM	1st transfer	2nd transfer	3rd transfer	4th transfer	5th transfer	6th transfer	average (spores/ml)
LBA[1]	59,081.9	49,928.4	21,828.6	52,207.7	62,388.1	49,819.9	49,200.8 A[2]
V8	66,100.9	47,649.1	33,285.6	48,951.5	54,161.5	42,330.6	48,746.5 A
MyA	57,634.7	32,923.8	28,461.6	34,117.7	78,510.6	10,492.2	40,356.8 A
BLEA	41,245.2	62,844.7	8,321.4	27,750.1	73,807.2	26,664.7	40,105.6 A
PDA	47,395.8	23,046.7	12,060.0	19,899.0	75,145.9	27,967.1	34,253.9 AB
MEM	20,875.9	8,321.4	7,839.0	25,579.3	6,982.7	22,069.8	15,278.0 BC
RCA	18,922.1	13,922.1	7,839.0	10,962.5	33,647.4	3,256.2	14,687.1 BC
CzA	3,002.9	1,085.4	1,688.4	1,447.2	1,085.4	361.8	1,445.2 C

1) LBA: Lima bean agar, V8: V8 juice agar, MyA: Mycophil agar, BLEA: Barley leaf extract agar, PDA: Potato dextrose agar, MEM: Modified Eckert's Medium, RCA: Rice cereal agar, CzA: Czapeck's agar

2) Averages followed by different letters differ significantly at P = .05 probability level.

Chapter 2

EFFECT OF PHOTOPERIOD AND TYPE OF GROWTH MEDIUM ON THE SPORULATION OF *P. teres*

In the previous experiment different types of growth media were tested for their effectiveness in inducing the sporulation of *P. teres*. Based on the amount of sporulation, four agar types were found to be particularly effective.

In this experiment, these four agar types, Mycophil agar (BBL), Lima bean agar (Difco), V8 juice agar and Barley leaf extract agar, were again used. These were tested over a range of photoperiods in an effort to determine an optimum photoperiod and agar type.

Materials and Methods

The same isolate used previously, Pt S (Sidney, MT), was used in this experiment. The four types of growth media were prepared following the procedures in Appendix A. Seven different photoperiods were used; no light, 8 hr., 12 hr., 16 hr., 20 hr., 22 hr., and 24 hr. of light. Temperature in the growth chamber was maintained at 17-18° C, with a photoperiod of 24 hr. of light. Cultures grown under shorter photoperiods were placed in cardboard boxes and wrapped in aluminum foil during the dark period. All four agar types were tested at each photoperiod. Spores were initially transferred from leaf tissue to plates of the four agar types. These cultures were then grown at the different photoperiods for seven days. The amount of sporulation was then

determined in the manner described previously. Four successive replications were run.

Results and Discussion

Table 2-1 (a-g) shows the sporulation of *P. teres* on the different agar types at each photoperiod. The values were used to determine the average sporulation at each photoperiod over all agar types (Table 2-2) and for each agar type over all photoperiods (Table 2-3). An analysis of variance test was then computed to determine which averages were significantly different (Table 2-4). Because of the extremely low sporulation of *P. teres* under conditions of no light, the date in Table 2-1 (a) were not included.

The results of the analysis of variance test indicate that the differences within replications, photoperiods, replications x photoperiod interaction and replication x agar type interaction were statistically significant, while the differences within agar types and agar type x photoperiod interaction were not statistically significant. These results indicate that the different agar types did not significantly affect the sporulation of *P. teres*, while the photoperiod under which the cultures of *P. teres* were grown did have a significant effect upon sporulation.

A Duncan's Multiple Range Test was utilized to determine which photoperiods differed significantly (Table 2-2). The results of this test show that the sporulation of *P. teres* under a photoperiod of eight hours

was significantly higher than the sporulation under any other photoperiod. The results also indicate that sporulation increases significantly as the photoperiod decreases. Because of the very low sporulation with no light, it is evident that some light is necessary. The results obtained in this test indicate that of the seven different photoperiods tested, an eight hour photoperiod is best for sporulation of *P. teres*. These results also indicate that it would be worthwhile to test the sporulation of *P. teres* grown under photoperiods shorter than eight hours.

There is a paucity of studies involving the effect of environment on sporulation of *P. teres*. Onesirosan and Banttari (1969) studied the effect of light and temperature on sporulation. Using photoperiods of no light, 12 hr. light and 24 hr. light, they found a 12 hr. photoperiod most conducive to sporulation. They also studied the effect of light quality, and found that ultraviolet light with wavelengths of 310-355 µm was necessary for conidiophore formation. Conidia production was most favored in the absence of light or in the absence of light of wavelengths 355-495 µm. It appears that stimulation of conidiophore production is critical, and once this is accomplished, production of conidia is not light dependent. One might be able to obtain maximum sporulation by growing the cultures in only ultraviolet light.

Although not significantly so, the sporulation of *P. teres* on mycophil agar was higher than on the other agar types. For this

reason the entire collection was transferred to plates of mycophil agar. This resulted in a differential range of reactions of the isolates to this agar type. It appeared that certain isolates grew and sporulated well on it while others sporulated very poorly. Since all isolates sporulated reasonably well on V8 juice agar and since the sporulation of *P. teres* on the two agars in this test was not significantly different, it was decided that the cultures would be maintained on V8 juice agar.

Using V8 juice agar as a growth medium, and using a photoperiod of 8 hr. produces highly sporulating cultures of *P. teres*. Adequately high concentrations of conidia are obtained to produce readable, differentiating symptoms on barley plants.

Table 2-1. Sporulation (spores/ml) of *P. teres* on different agar types at different photoperiods.

2-1(a); No light

Agar type[1]	1st transfer	2nd transfer	3rd transfer	4th transfer	average (spores/ml)
V8	0.0	108.5	108.5	0.0	54.3
LBA	0.0	470.3	0.0	108.5	144.7
MyA	0.0	253.3	470.3	242.4	241.5
BLEA	0.0	108.5	1,193.9	1,085.4	597.0

[1] V8 = V8 juice agar, LBA = Lima bean agar, MyA = Mycophil agar, BLEA = Barley leaf extract agar

2-1(b); 8 Hours of light

Agar type	1st transfer	2nd transfer	3rd transfer	4th transfer	average (spores/ml)
V8	361.8	62,591.4	45,104.4	79,133.6	46,792.8
LBA	5,427.0	55,234.8	58,129.2	79,234.2	49,506.3
MyA	2,062.3	99,615.8	78,402.1	70,792.2	62,718.1
BLEA	6,150.6	47,395.8	41,607.0	91,656.0	46,702.4

2-1(c); 12 Hours of light

Agar type	1st transfer	2nd transfer	3rd transfer	4th transfer	average (spores/ml)
V8	46,057.1	17,366.4	5,897.3	64,641.6	33,490.6
LBA	40,159.8	17,619.7	9,298.3	61,867.8	32,236.4
MyA	16,642.8	11,577.6	361.8	84,781.8	28,333.5
BLEA	68,870.7	8,936.5	723.6	42,330.6	30,215.4

2-1(d); 16 Hours of light

Agar type	1st transfer	2nd transfer	3rd transfer	4th transfer	average (spores/ml)
V8	34,732.8	5,427.0	14,363.7	36,059.4	22,645.7
LBA	39,327.7	10,492.2	7,344.5	34,853.4	23,054.5
MyA	19,537.2	13,133.3	3,509.5	86,349.6	30,632.4
BLEA	51,013.8	3,979.8	5,427.0	31,476.6	22,974.3

2-1(e); 20 Hours of light

Agar type	1st transfer	2nd transfer	3rd transfer	4th transfer	average (spores/ml)
V8	21,961.3	7,489.3	4,956.7	20,140.2	13,636.9
LBA	15,448.9	12,771.5	9,298.3	41,848.2	19,841.7
MyA	832.1	30,282.7	3,364.7	60,420.6	23,725.0
BLEA	54,378.5	3,364.7	1,700.5	37,386.0	24,207.4

2-1(f); 22 Hours of light

Agar type	1st transfer	2nd transfer	3rd transfer	4th transfer	average (spores/ml)
V8	25,217.5	20,069.8	20,622.6	11,577.6	19,371.9
LBA	14,725.3	6,620.9	10,854.0	10,612.8	10,703.3
MyA	2,170.8	12,301.2	20,875.9	32,320.8	16,917.2
BLEA	61,614.5	6,512.4	16,642.8	11,577.6	24,086.8

2-1(g); 24 Hours of light

Agar type	1st transfer	2nd transfer	3rd transfer	4th transfer	average (spores/ml)
V8	15,810.7	17,113.1	13,278.1	3,738.6	12,485.1
LBA	6,982.7	5,173.7	8,212.9	12,180.6	8,137.5
MyA	1,193.9	10,492.2	17,728.2	18,451.8	11,966.5
BLEA	5,318.5	12,301.2	4,088.3	13,024.8	8,683.2

Table 2.2 Average sporulation (spores/ml) of *P. teres* at each photoperiod.

Photo period	1st transfer	2nd transfer	3rd transfer	4th transfer	average	Duncan's Multiple Range Test P=.05*	P=.01**
8 hr	3,500.4	66,209.4	55,810.4	80,199.0	51,429.8	A	A
12 hr	42,932.6	13,875.1	4,070.3	63,405.5	31,070.9	B	B
16 hr	36,152.9	8,258.1	7,661.2	47,184.8	24,814.3	BC	BC
20 hr	23,155.2	13,477.1	4,830.1	39,948.8	20,352.8	CD	BCD
22 hr	25,932.0	11,376.1	17,248.8	16,522.2	17,769.8	CDE	CD
24 hr	7,326.5	11,270.1	10,826.0	11,849.0	10,318.1	E	D

Table 2-3. Average sporulation (spores/ml) of *P. teres* on each agar type.

Agar type	1st transfer	2nd transfer	3rd transfer	4th transfer	average	Duncan's Multiple Range Test P=.05*
MyA	7,073.2	29,567.1	20,717.0	58,852.8	29,052.5	A
LBA	20,345.2	17,985.5	17,189.5	40,099.5	23,904.9	A
V8	24,023.5	21,676.2	17,370.5	35,878.5	24,737.2	A
BLEA	41,224.4	13,748.4	11,698.2	37,908.6	26,144.9	A

*, ** Those values not followed by similar letters differ significantly at probability P=.05, or P=.01, respectively.

Table 4. Analysis of Variance Test.

	DF	SS	MS	F value
Reps (R)	3	1,000,120.0	333,374.0	27.135+
Photoperiod (P)	5	1,631,030.0	326,207.0	26.551+
R x P	15	2,029,070.0	135,271.0	11.010‡
Agar (A)	3	36,718.4	12,239.5	0.996‡
R x A	9	627,475.0	69,719.5	5.675‡
P x A	15	129,328.0	8,261.84	0.702‡
RPA (Error)	45	552,863.0	12,285.9	-- +
	95	6,006,610.0		

+ Significant at P=.01

‡ Non-significant at P=.05 or P=.01

Chapter 3

DETERMINATION OF DIFFERENT VIRULENCE TYPES OF *Pyrenophora teres*

In the spring of 1977, when testing of barley varieties for their reaction to net blotch was first initiated, seven different isolates of *Pyrenophora teres* were available. Four of these isolates were from Montana and three had been collected in the Middle East. By the summer of 1977, twenty-six isolates were available, fifteen originating in the Middle East and eleven from various parts of Montana.

It was possible to test a limited number of varieties to all twenty-six isolates, but when a screening test was set up to screen large numbers of barley lines to *P. teres*, limits on space and time made it necessary to use fewer isolates. To insure that the reduced number of isolates would be representative of the entire collection, the collection was separated into different virulence types.

Materials and Methods

The twenty-six isolates were separated into different virulence groups based on the differential reactions of fifteen barley varieties which had been tested to the isolates during the summer of 1977.

The varieties were grown in metal flats, 14"x10"x3", with twelve varieties planted per flat. Ten seeds of each variety were planted in six rows across the width of the flat, with two varieties per row. The plants were grown in a growth chamber with a 15°/24°C dark/light temperature, and a 12 hour photoperiod.

The isolates of *P. teres* were grown on V8-juice agar, in an incubation chamber with a temperature of 17-18°C and a 12 hour photoperiod. The isolates were subcultured utilizing the mass spore transfer method mentioned previously. Isolates to be used as inocula were transferred on the same day the barley to be inoculated was planted. The barley was then inoculated seven days later with the seven day old cultures. The inoculum consisted of a suspension of conidia and some mycelial fragments. As a surfactant, a solution of 1% Tween 20 at approximately 0.1 ml of surfactant to 100 ml of inoculum (0.1%) was used. The inoculum was applied using an atomizer driven by compressed air. Approximately 15-18 ml of inoculum were used per flat. A minimum of 10^4 spores/ml seemed necessary to obtain adequate infection. Spore concentrations of 20-30 x 10^3 spores/ml were not difficult to obtain. No standardization of the spore concentration was attempted. with concentrations approaching 80-90 x 10^3 spores/ml used in the inoculations.

Inoculated plants were incubated in a dew chamber at 100% relative humidity in darkness for 24 hours. The temperature of the water bath was 35°C and the wall temperature of 7°C. The resulting air temperature was 24-27°C.

Readings on plant infection were made seven days after inoculation. Reaction types were based on a scale of 0-4:

0: no observable infection

1: pin-point to very slightly elongated lesions (1-2 mm) with no observable chlorosis

2: slightly elongated lesions, with no netting symptoms, limited amounts of chlorosis

3: elongated lesions forming netting symptoms, easily observable chlorosis, no necrosis

4: well developed, netted lesions, with extensive chlorosis and necrosis of leaf tissue

The isolates were then grouped, based on the similarities and differences of the reactions of the barley varieties and C.I. numbers. The isolates and their origin are listed in Table 3-1. The barley varieties utilized as differentials are listed in Table 3-2.

Results and Discussion

The Middle Eastern isolates were separated from the Montana isolates, since the two groups as a whole produced different reactions on several varieties. For example, Shabet, Firlbecks III, Betzes, and Ingrid were susceptible to all Montana isolates, yet contained resistance to several Middle Eastern isolates. C.I. 7584, C.I. 9776 and Steptoe were resistant to all Montana isolates, yet were susceptible to some Middle Eastern isolates. Because of the nondiscriminatory reactions of these varieties and lines to the Montana isolates, they were of little use as differentials. Hence, it was necessary to utilize other varieties

in order to determine virulence types of the Montana isolates.

Table 3-3 shows the Middle Eastern isolates grouped into their respective virulence types, based on the reactions of fourteen barley lines. Virulence type A contains five isolates, the other types contain from one to three isolates. The reactions of those varieties and lines which are most useful in separating the isolates into the different virulence types are circled. For example, C.I. 9776 is susceptible to type B, while Shabet, Betzes and Ingrid are all resistant. Type D is the only virulence type to which Mona-Arivat is resistant. Both C.I. 7584 and Tifang are susceptible to type E, while type F is the only virulence type to which C.I. 7584, C.I. 9776 and Steptoe are all susceptible and both Shabet and Ingrid are resistant. Arimont is resistant only to virulence type G.

Table 3-4 shows the virulence types of the Montana isolates of *P. teres*, based on the reactions of eight barley varieties and lines. Here again, those lines and varieties most useful in separating the different virulence types are circled. Tifang is susceptible only to type C. C.I. 9819 exhibits type 4 symptoms only to those isolates in type D. Both Lud and Piroline are intermediate in reaction to type E, while Lud is resistant to intermediate in reaction to type B and Piroline is susceptible.

Using differential varieties a dichotomous key was set up with which it was possible to distinguish the virulence types of the Middle

Eastern isolates (Table 3-5). All seven virulence types could be separated using five differential barley lines; C.I. 9776, C.I. 7584, Arimont, Mona-Arivat and Betzes.

Another dichotomous key, utilizing a different set of differentials, was set up for determining the virulence types of the Montana isolates of *P. teres* (Table 3-5). The five Montana virulence types could be distinguished using four differential barley lines; Lud, Piroline, Tifang and C.I. 9819.

The separation of the isolates into virulence types was done primarily to obtain isolates representative of the entire collection. It was determined that nine isolates of *P. teres* could be efficiently used in a screening test. Nine isolates were therefore chosen, each from a different virulence type. Because the isolates had been separated into more than nine virulence types, the isolates were chosen from the nine most virulent groups. These groups are indicated in Tables 3-5 and 3-6 by asteriks.

This work is not viewed as a final conclusion in dealing with the virulence types of these isolates. It is useful as a method for selecting representative isolates from a large collection for use in screening tests. Knowledge of the different virulence types occurring in an organism is valuable in that it provides a tool by which broad based sources of resistance can be selected.

Table 3-1. Isolates of *P. teres* included in the collection and the place of origin of each.

Isolate	Origin
Pt 8	Tunisia
Pt 8orig	"
Pt 10	Lebanon
Pt 10orig	"
Pt T-1	Tunisia
Tunis 1	"
Tunis 75	"
Mor 1	Morocco
Mor 14	"
Mor 15	"
Mor 18	"
Pt 6	Turkey
ELS 77-5	Morocco
ELS 77-6	Jordan
ELS 77-9	Egypt
Mt 77-1	Fairfield, Mt.
Mt 77-2	"
Mt 77-3	Fort Benton, Mt.
Mt 77-4	Creston, Mt.
Mt 77-5I	Sidney, Mt.
Mt 77-6	Moccassin, Mt.
Mt 77-7	Huntley, Mt.
Pt S	Sidney, Mt.
Pt S-2	"
Pt R	Rudyard, Mt.
Pt B	Baker, Mt.

Table 3-2. Varieties and C.I. lines used in determining the different virulence types of *P. teres*.

Unitan	Tifang
Steptoe	C.I. 5791
Shabet	C.I. 9819
Dekap	C.I. 9776
Ingrid	C.I. 7584
Betzes	Arimont
Firlbecks III	Mona-Arivat
Horsford	Larker
Lud	Piroline
Palliser	Hypana

Table 3-3. Virulence types of the Middle East isolates of *P. teres*.

	Variety or C.I. Number													
Isolate[1]	7584	9776	9819	5791	Tifang	Steptoe	Unitan	Dekap	Firlbecks III	Shabet	Betzes	Ingrid	Ari-mont	Mona-Arivat
Type A														
Pt 8	1,2	1	2,3	2,1	2,1	1,2	2,1,3	4	4,3	4,3	4	4	4,3	4,3
Pt 8orig*	1,2	1,2	2,3	2,1,3	1,2	1,2	2,1	4	3,4	4,3	4,3	4,3	4	3,4
Pt 10	1,2	1,2	2,1	2,1	2	2	1,2	4,3	3,4	4	4	4	4	4,3
Pt 10orig	2,3,1	2,1	3,2	3,2,4	1,2	1	2,1	4	3,4	4,3	4,3	4,3	4,3	2,3,4
Pt T-1	1,2	1,0	2,1	1,2	1,2	1,2	1,2	4,3	4,3	4	4	4,3	4,3	3,2
Type B														
Mor 15*	2,3	4,3[2]	1,2	1,2	2,1	2,1,3	2,1	4,3	3,2,4	2,3,1	1,2,3	1,2	4,3	4,3
Mor 18	1,2	3,4	2,1	1,2	1,3,2	2,1	2	4,3	2,4,3	2,3	1,2,3	1,2	4,3	4
Type C														
ELS 77-6*	1,2	2,1	1,3,2	1,3,2	1,2	3,2	3,2	3,4	3	3,2,4	1,3,2	2,1	4,3	3,4,2
ELS 77-9	2,1	2,3	2,1,3	1,2	2,1	1,2	3,2,4	3,4	2,3	---	2,1	2,1,3	3,2,4	3,2
Type D														
Tunis	2,3,1	2,1	2,1	1,2	1,3,2	2,1	2,1	4,3	2,3	3,4	3,4,2	1,2	4,3	2,1,3
Type E														
Mor 1*	4,3	1,2	2,1	1	4,3	2,3	2,1	4	4,3	4,3	3,2	4,3	4	4
Type F														
Mor 14*	4,3	4,3	2,1	2,1,3	3,2	3,4,2	3,2	4	4	2,1	3,2,4	1	4,3	4
Type G														
Pt6	3,4	1,2	2,3	1,2	3,2,4	2,3	1,2	3	2	2,1,3	3,2,4	2,1	2,1,3	3,4
ELS 77-5	2	2,1	3,2	2,1	2,3	2,3	2,3	3,4,2	2,3,1	2,3	3,2	2,3	2,1	3,4
Tunis 75	3,2	2,1	3,2	2,1	2,3	2,3	1,2	3,2	2,3	2,1	2,1	2,1	1,2	3,2

1. Those isolates followed by an asterik were used in the screening of barley lines for resistance to *P. teres* (Chapter 4).
2. Those reactions circled were especially useful in determining the different virulence types of the Middle East isolates of *P. teres*.

Table 3-4. Virulence types of the Montana isolates of *P. teres*.

	Variety or C.I. Number							
Isolate[1]	C.I. 9819	C.I. 5791	Tifang	Unitan	Larker	Lud	Piroline	Dekap
Type A								
Pt R*	2,1	1,2	3,2	2,1	4,3	4	4,3	4
Mt 77-2	2,1,3	1,2	1,3	2,1	3,4	4,3	4	4
Type B								
Pt S	2,3,1	2,1	3,2	2,3	4,3	1,2,3[2]	3,4	4
Pt S-2	1,2	2,1,3	1,2	2,1	3,4	2,1,3	4,3	4
Pt B*	2,1,3	2,1	1,2	2,1	3,2,4	3,2	3,4	4
Type C								
Mt 77-1	1,2	2,3	4	2,3	3,4	3,2,4	3,4	4,3
Mt 77-5I*	2,1	2,1	4	2,1	4,3	3,4,2	4	4
Type D								
Mt 77-3*	2,4,3	3,4	1	2,1	2,3,4	3,2,4	2,3	4
Mt 77-4	3,2,4	2,3	---	2,1	2,3	3,4	3,4	4,3,2
Type E								
Mt 77-6	2,3	2,3	1,2	2,3	3,2	2,3	2,3	4
Mt 77-7	2,3	2,3,1	3	3,2	3	2,3	2,3	4,3

1. Those isolates followed by an asterik were used in the screening of barley lines for resistance to *P. teres* (Chapter 4).
2. Those reactions circled were especially useful in determining the different virulence types of the Montana isolates of *P. teres*.

Table 3-5. Key for identifying the virulence types of the Middle Eastern isolates.

Host Reaction	Virulence type
C.I. 9776 susceptible	
C.I. 7584 susceptible.	Type F
C.I. 7584 resistant.	Type B
C.I. 9776 resistant	
C.I. 7584 susceptible.	Type E
C.I. 7584 resistant	
Arimont resistant.	Type G
Arimont susceptible	
Mona-Arivat resistant.	Type D
Mona-Arivat susceptible	
Betzes susceptible.	Type A
Betzes resistant.	Type C

Table 3-6. Key for identifying the virulence types of the Montana isolates.

Host Reaction	Virulence type
Lud resistant	
Piroline resistant	Type E
Piroline susceptible	Type B
Lud susceptible	
Tifang susceptible	Type C
Tifang resistant	
C.I. 9819 susceptible	Type D
C.I. 9819 resistant	Type A

Chapter 4

SCREENING OF BARLEY LINES FOR RESISTANCE TO *Pyrenophora teres*

The virulence types previously designated for isolates of *Pyrenophora teres* were utilized in this experiment to screen barley lines and varieties for resistance to net blotch. It was anticipated that the testing of lines against nine separate isolates, each representing a different virulence type, would result in the discovery of lines which contain resistance to a wide range of virulence types.

Materials and Methods

One hundred and forty-seven lines of barley were screened in this test. These lines consisted of three major groups. The largest group included 78 lines previously found to be resistant to Moroccan isolates of *P. teres* (Caddel and Wilcoxson, 1975). Another group of forty lines had previously been found to be resistant in Canada to North American isolates of *P. teres* (Buchannon and McDonald, 1965). The third group consisted of 23 varieties commercially grown in the state of Montana and not previously screened for resistance to *P. teres*. Seed of these lines was obtained either from seed stocks on hand or from Dr. J. C. Craddock, Beltsville, Md.

The lines were planted in metal flats and grown under conditions previously described. Eleven lines plus the susceptible check, Dekap, were planted in each flat.

A representative isolate was chosen from each of the nine virulence

types indicated in the previous experiment. These isolates were:

Pt R (Rudyard, Mt.)	Mor 1 (Morocco)
Pt B (Baker, Mt.)	Mor 14 (Morocco)
Mt 77-3 (Fort Benton, Mt.)	Mor 18 (Morocco)
Mt 77-5I (Sidney, Mt.)	ELS 77-6 (Jordan)
Pt 8orig (Tunisia)	

The isolates were cultured in the same manner described previously, except that a photoperiod of 8 hr., rather than 12 hr., was used. Inoculation procedures and recording of the disease reactions were also performed as previously described.

The disease reactions of the barley lines were separated into three groups:

Resistant: Lines exhibiting type 1 or type 2 reactions, or a combination of type 1 and type 2 reactions.

Intermediate: Lines exhibiting a combination of type 2 and type 3 reactions.

Susceptible: Lines exhibiting type 3 or type 4 reactions, or a combination of type 3 and type 4 reactions.

Results and Discussion

The results of the screening test are shown in Tables 4-1 and 4-2. The barley lines are grouped in two different ways. In Table 4-1, the lines are grouped according to the number of virulence types against which they are resistant. In Table 4-2, they are grouped according to

the number of virulence types against which they exhibit a resistant or an intermediate type of reaction.

Of the 147 lines tested, only three were resistant to all nine virulence types (Table 4-1). Four more lines were resistant to eight of the nine virulence types. Thirty lines, 20.4% of the total, were resistant to six or more virulence types. Considering both resistant and intermediate reactions in one class, nine lines were effective against all nine virulence types (Table 4-2). An additional twenty lines showed resistant or intermediate reactions to eight virulence types.

As stated earlier, the lines involved in this screening test consist mainly of three major groups. Of those lines found to be resistant to North American isolates of *P. teres*, seventeen lines, 42.5% of the total, were resistant to six or more virulence types (Table 4-3). Of those lines previously found resistant to Moroccan isolates of *P. teres*, 10 lines, 12.8% of the total, were resistant to at least six virulence types. Only two of the 23 commercial varieties were resistant to six or more virulence types. Twenty of the twenty-three varieties were resistant to three or fewer of the virulence types.

The lines reported to be resistant in Canada had been tested in the greenhouse to isolates from Canada, Mexico and the U.S. The resistance found to be effective against those isolates appeared to also be relatively effective against the isolates used in this screening test.

Thirty-one lines, 77.5% of the total, showed resistant or intermediate reactions to at least six of the virulence types (Table 4-4).

In Morocco, natural infection in the field was relied upon to produce readable symptoms on the plants. The resistance found effective in Morocco was less effective against the wider range of virulence types used in this test. Twnety-nine lines, 37.2% of the total, were resistant or intermediate in reaction to six or more virulence types. Overall, these lines are relatively less effective than those tested in Canada, but are considerably more resistant than the commercial varieties which had not previously been screened for net blotch resistance (Table 4-4). These results indicate the value of using a wide range of virulence types when screening for disease resistance.

The barley lines exhibiting resistant or intermediate reactions to eight or nine virulence types were crossed into a male sterile facilitated recurrent selection population being developed for net blotch resistance. It is anticipated that these lines will provide a source of broad-based resistance effective against a wide range of virulence types of this organism.

Table 4-1. Relative resistance of various barley lines to different isolates of *Pyrenophora teres*.

Barley line	Pt R	Pt B	Mt 77-3	Mt 77-5I	Pt 8orig	Mor 1	Mor 14	Mor 18	ELS 77-6
Lines resistant to nine virulence types									
C.I. 1615 (C)	R	R	R	R	R	R	R	R	R
C.I. 5845 (C)	R	R	R	R	R	R	R	R	R
C.I. 9768 (C)	R	R	R	R	R	R	R	R	R
Lines resistant to eight virulence types									
C.I. 5791 (C)	R	R	S	R	R	R	R	R	R
C.I. 6388 (C)	R	R	S	R	R	R	R	R	R
C.I. 12821 (M)	R	R	R	S	R	R	R	R	R
C.I. 14023 (C)	S	R	R	R	R	R	R	R	R
Lines resistant to seven virulence types									
C.I. 4207 (M)	I	R	R	R	R	R	I	R	R
C.I. 5401 (C)	I	R	R	R	R	R	R	I	R
C.I. 6475 (C)	R	R	I	R	R	R	R	R	S
C.I. 7208 (C)	I	R	I	R	R	R	R	R	R
C.I. 7447 (M)	R	R	R	R	R	R	S	S	R
C.I. 9669 (M)	S	R	I	R	R	R	R	R	R
C.I. 9751 (M)	R	S	R	S	R	R	R	R	R
C.I. 9819 (C)	R	R	S	R	I	R	R	R	R
Lines resistant to six virulence types									
C.I. 2235 (C)	R	I	R	I	R	S	R	R	R
C.I. 5276 (M)	I	R	R	R	R	R	S	S	R
C.I. 5298 (M)	I	R	R	R	R	R	I	R	I
C.I. 5404 (C)	I	R	R	R	R	I	R	R	S

1) Those lines follwed by (C) were tested to North American isolates, those followed by (M) were tested to Moroccan isolates, those followed by (Mt.) are the Montana grown varieties, those not followed by any letter are not involved with any of the three major groups.

Table 4-1 (cont.)

Barley line	Pt R	Pt B	Mt 77-3	Mt 77-5I	Pt 8orig	Mor 1	Mor 14	Mor 18	ELS 77-6
	Isolates								
Lines resistant to six virulence types (cont.)									
C.I. 5822 (C)	I	R	R	R	R	R	R	I	I
C.I. 7504 (C)	I	R	I	R	R	R	R	R	S
C.I. 7584 (C)	R	R	R	S	R	S	S	R	R
C.I. 9776 (C)	R	R	R	I	R	R	S	S	R
C.I. 9825 (C)	I	I	R	R	R	R	R	R	S
C.I. 9827 (M)	S	S	R	R	R	I	R	R	R
C.I. 10379 (M)	S	R	R	R	R	I	S	R	R
C.I. 12860 (M)	S	R	R	R	R	I	I	R	R
Steptoe (Mt.)	R	R	R	R	R	I	S	R	I
Tifang	I	R	R	S	R	S	R	R	R
Unitan (Mt.)	R	R	R	R	R	I	I	R	I
Lines resistant to five virulence types									
C.I. 7272 (C)	R	R	R	I	I	R	I	S	R
C.I. 7744 (M)	S	R	S	R	R	R	I	R	I
C.I. 8332 (C)	I	R	R	R	R	R	S	S	I
C.I. 8333 (C)	R	R	R	R	R	S	S	I	S
C.I. 9647 (C)	S	R	S	R	R	I	R	R	I
C.I. 9648 (C)	S	R	R	R	R	I	I	R	S
C.I. 10903 (M)	S	R	R	S	R	R	S	R	I
C.I. 11631 (M)	I	R	I	R	R	R	S	R	S
Lines resistant to four virulence types									
C.I. 1064 (M)	S	I	I	R	R	R	R	I	I
C.I. 4373 (M)	S	S	I	S	R	R	I	R	R
C.I. 5810 (C)	R	R	S	I	R	I	S	R	S
C.I. 6496 (M)	S	I	I	R	R	R	I	R	I
C.I. 9408 (M)	S	S	R	I	R	R	I	S	R
C.I. 9440 (C)	I	R	R	R	I	I	R	I	S
C.I. 9505 (C)	I	I	R	R	R	S	S	R	I
C.I. 9758 (M)	R	S	R	S	I	R	I	R	I
C.I. 10070 (M)	S	R	S	S	I	S	R	R	R
C.I. 10778 (M)	S	R	S	S	R	S	S	R	R

Table 4-1 (cont).

Barley line	Pt R	Pt B	Mt 77-3	Mt 77-51	Pt 8orig	Mor 1	Mor 14	Mor 18	ELS 77-6
				Isolates					
Lines resistant to four virulence types (cont.)									
C.I. 10781 (M)	S	R	S	S	R	I	I	R	R
C.I. 13244 (M)	S	I	I	S	S	R	R	R	R
C.I. 13727 (M)	I	R	R	S	R	S	R	I	I
C.I. 14127 (M)	I	---	S	S	R	R	R	S	R
C.I. 14265 (M)	R	R	S	S	I	R	S	I	R
Freja (Mt.)	S	S	S	S	S	R	R	R	R
Lines resistant to three virulence types									
C.I. 1261 (M)	S	R	I	R	I	R	S	S	S
C.I. 3430 (C)	S	R	R	S	R	I	S	I	I
C.I. 4428 (C)	S	I	R	S	R	S	I	S	R
C.I. 4544	S	S	S	S	R	S	R	S	R
C.I. 5196 (M)	S	S	S	R	R	R	S	I	I
C.I. 5199 (M)	S	S	R	R	R	I	S	S	I
C.I. 6868 (M)	S	R	S	I	R	R	S	I	S
C.I. 7296 (M)	S	I	R	R	I	R	I	I	I
C.I. 7297 (C)	I	R	I	I	R	S	R	I	I
C.I. 7324	S	S	S	S	S	R	R	I	R
C.I. 7339 (M)	S	S	S	R	S	R	I	R	I
C.I. 8868 (M)	S	I	S	S	R	R	S	S	R
C.I. 9214 (C)	S	I	S	R	R	S	R	S	S
C.I. 9215 (M)	S	R	I	R	R	I	S	I	S
C.I. 9263 (C)	I	R	S	I	R	R	I	S	S
C.I. 9567 (M)	S	R	S	I	I	R	S	S	R
C.I. 9698 (M)	S	R	I	I	R	R	I	I	I
C.I. 9741 (M)	S	S	I	R	R	R	I	S	S
C.I. 10075 (M)	S	R	I	S	S	I	R	R	I
C.I. 11977 (M)	S	S	S	S	S	I	R	R	R
C.I. 11978 (M)	R	S	I	R	S	I	R	---	S
C.I. 12875 (M)	I	R	S	S	I	R	S	R	S
C.I. 13262 (M)	R	I	I	I	I	I	R	R	I
C.I. 13749 (M)	R	---	S	R	I	S	R	S	S
C.I. 14103 (M)	R	---	R	S	R	S	I	S	S
C.I. 14295 (M)	I	R	R	S	R	I	S	S	S
Erbet (Mt.)	S	S	S	S	S	S	R	R	R

Table 4-1 (cont.)

Barley line	Pt R	Pt B	Mt 77-3	Mt 77-5I	Pt 8orig	Mor 1	Mor 14	Mor 18	ELS 77-6
			Isolates						
Lines resistant to three virulence types (cont.)									
Ingrid (Mt.)	S	S	S	S	S	S	R	R	R
Klages (Mt.)	S	S	S	S	I	S	R	R	R
Manker (Mt.)	S	I	S	S	S	R	R	R	S
Lines resistant to two virulence types									
C.I. 4638 (C)	S	I	R	S	R	I	I	I	S
C.I. 5108 (C)	S	I	S	R	S	S	R	S	I
C.I. 8270 (C)	S	R	R	I	S	S	S	S	I
C.I. 9584 (C)	S	R	S	R	I	S	I	I	S
C.I. 9592 (C)	S	S	S	I	R	I	R	S	S
C.I. 9693 (C)	S	S	S	R	I	R	S	I	S
C.I. 9702 (M)	S	I	S	I	I	R	R	I	I
C.I. 9831 (C)	I	I	I	I	R	S	I	R	I
C.I. 9881 (M)	I	S	I	S	R	R	S	I	I
C.I. 10786 (M)	S	S	I	S	R	S	S	R	S
C.I. 11056 (M)	S	R	S	S	R	S	S	S	S
C.I. 11121 (M)	S	S	S	I	R	S	S	R	S
C.I. 11546 (M)	I	R	S	S	R	I	S	I	S
C.I. 11578 (M)	I	S	S	S	R	I	I	R	S
C.I. 12949 (M)	S	R	S	S	I	S	S	S	R
C.I. 13797 (M)	S	---	R	S	S	S	R	S	I
C.I. 14033 (M)	R	---	R	S	I	S	I	S	I
Georgie (Mt.)	S	S	S	S	S	S	R	R	I
Lud (Mt.)	S	I	S	S	S	S	R	R	I
Piroline (Mt.)	S	S	I	S	S	I	R	R	S
Lines resistant to one virulence type									
C.I. 1197	I	I	I	I	R	S	---	I	I
C.I. 2216 (M)	S	S	I	S	S	R	S	I	S
C.I. 2330	S	S	S	S	I	S	R	S	S
C.I. 5057 (C)	S	S	S	R	S	I	S	I	S
C.I. 5172 (M)	S	S	S	I	I	I	S	I	R
C.I. 5376 (M)	S	S	S	S	I	I	S	S	R
C.I. 5809 (C)	S	R	S	I	I	I	I	I	S
C.I. 8952 (M)	S	I	I	S	I	I	S	S	R

Table 4-1 (cont.)

Barley line	Pt R	Pt B	Mt 77-3	Mt 77-5I	Pt 8orig	Mor 1	Mor 14	Mor 18	ELS 77-6
				Isolates					
Lines resistant to one virulence type (cont.)									
C.I. 9327 (M)	I	S	S	S	R	S	S	S	S
C.I. 9569 (M)	S	S	S	S	S	I	S	R	I
C.I. 9669 (C)	S	R	S	I	I	I	S	S	S
C.I. 10615 (M)	S	S	S	I	S	S	S	S	R
C.I. 10624 (M)	S	I	S	S	R	S	S	I	S
C.I. 10905 (M)	S	R	S	S	I	S	S	I	S
C.I. 12034 (M)	S	S	S	S	S	I	S	I	R
C.I. 12674 (M)	S	S	I	S	S	S	I	R	I
C.I. 12894 (M)	I	R	S	S	S	S	S	S	S
C.I. 14218 (M)	S	S	R	S	I	S	S	S	S
C.I. 14273 (M)	S	S	R	S	I	S	I	S	S
Betzes (Mt.)	S	S	S	S	S	S	S	S	R
Hector (Mt.)	S	S	S	S	S	S	S	I	R
Horsford (Mt.)	S	S	S	S	I	R	S	S	S
Hypana (Mt.)	S	I	I	R	S	S	S	S	I
Palliser (Mt.)	S	S	S	S	I	R	S	S	S
Shabet (Mt.)	S	S	S	S	S	S	R	I	S
Lines with no resistance to any of the virulence types									
C.I. 4179 (M)	S	S	S	S	S	I	S	S	S
C.I. 5187 (M)	S	S	I	S	I	S	S	S	I
C.I. 7725 (M)	S	S	S	S	S	I	S	S	S
C.I. 9345 (M)	S	S	S	I	I	I	S	S	S
C.I. 9350 (M)	S	S	I	S	I	I	S	S	S
C.I. 10132 (M)	S	S	I	S	S	S	S	S	S
C.I. 10364 (M)	S	S	S	S	S	I	S	S	I
C.I. 10367 (M)	S	I	S	S	I	I	S	S	S
C.I. 14093 (M)	I	---	S	S	I	S	S	S	S
C.I. 14354 (M)	S	---	S	S	I	S	S	S	S
Arimont (Mt.)	S	S	S	S	S	S	S	S	S
Compana (Mt.)	S	S	S	S	S	S	S	S	S
Dekap (Mt.)	S	S	S	S	S	S	S	S	S
Firlbecks III	S	S	S	S	S	S	S	S	S
Karl (Mt.)	S	S	S	S	S	S	I	I	S

Table 4-1 (cont.)

Barley line	Pt R	Pt B	Mt 77-3	Mt 77-5I	Pt 8orig	Mor 1	Mor 14	Mor 18	ELS 77-6
Lines with no resistance to any of the virulence types (cont.)									
Larker (Mt.)	S	S	S	S	S	I	S	I	S
Mona-Arivat	S	S	S	S	S	S	S	S	S
Vanguard (Mt.)	S	S	S	S	S	S	I	I	I

Table 4-2. Relative resistance of barley lines to various isolates of *Pyrenophora teres* (resistant and intermediate reactions combined).

Resistant or intermediate to nine virulence types

C.I. 1615 (C)
C.I. 4207 (M)
C.I. 5298 (M)
C.I. 5401 (C)
C.I. 5845 (C)
C.I. 7208 (C)
C.I. 9768 (C)
C.I. 13262 (M)
Unitan (Mt.)

Resistant or intermediate to eight virulence types

C.I. 1064 (M)
C.I. 2235 (C)
C.I. 5404 (C)
C.I. 5791 (C)
C.I. 5822 (C)
C.I. 6388 (C)
C.I. 6475 (C)
C.I. 6496 (M)
C.I. 7272 (C)
C.I. 7296 (M)
C.I. 7297 (C)
C.I. 7504 (C)
C.I. 9440 (C)
C.I. 9698 (M)
C.I. 9699 (M)
C.I. 9819 (C)
C.I. 9825 (C)
C.I. 9831 (C)
C.I. 12821 (M)
C.I. 12860 (M)
C.I. 14023 (C)
Steptoe (Mt.)

Resistant or intermediate to seven virulence types

C.I. 1197
C.I. 5276 (M)
C.I. 7447 (M)
C.I. 7744 (M)
C.I. 8332 (C)
C.I. 9505 (C)
C.I. 9647 (C)
C.I. 9648 (C)
C.I. 9702 (M)
C.I. 9751 (M)
C.I. 9758 (M)
C.I. 9776 (C)
C.I. 9827 (M)
C.I. 10379 (M)
C.I. 11631 (M)
C.I. 13727 (M)
Tifang

Resistant or intermediate to six virulence types

C.I. 3430 (C)
C.I. 4373 (M)
C.I. 4638 (C)
C.I. 5809 (C)
C.I. 5810 (C)
C.I. 7584 (C)
C.I. 8333 (C)
C.I. 9215 (M)
C.I. 9263 (C)
C.I. 9408 (M)
C.I. 9881 (M)
C.I. 10075 (M)
C.I. 10781 (M)
C.I. 10903 (M)
C.I. 13244 (M)
C.I. 14265 (M)

Resistant or intermediate to five virulence types

C.I. 1261 (M)
C.I. 4428 (C)
C.I. 5172 (M)
C.I. 8952 (M)
C.I. 9567 (M)
C.I. 9584 (C)
C.I. 11978 (M)
C.I. 12875 (M)
C.I. 14033 (M)

Table 4-2 (cont.)

Resistant or intermediate to five virulence types (cont.)

C.I. 5196 (M)
C.I. 5199 (M)
C.I. 6868 (M)
C.I. 7339 (M)
C.I. 9741 (M)
C.I. 10070 (M)
C.I. 11546 (M)
C.I. 11578 (M)
C.I. 14127 (M)
C.I. 14295 (M)

Resistant or intermediate to four virulence types

C.I. 5108 (C)
C.I. 7324
C.I. 8270 (C)
C.I. 8868 (M)
C.I. 9214 (C)
C.I. 9592 (C)
C.I. 9669 (C)
C.I. 9693 (C)
C.I. 10778 (M)
C.I. 11977 (M)
C.I. 12674 (M)
C.I. 13749 (M)
C.I. 14103 (M)
Freja (Mt.)
Hypana (Mt.)
Klages (Mt.)
Lud (Mt.)
Manker (Mt.)
Piroline (Mt.)

Resistant or intermediate to three virulence types

C.I. 2216 (M)
C.I. 4544
C.I. 5057 (C)
C.I. 5187 (M)
C.I. 5376 (M)
C.I. 9345 (M)
C.I. 9350 (M)
C.I. 9569 (M)
C.I. 10367 (M)
C.I. 10624 (M)
C.I. 10786 (M)
C.I. 10905 (M)
C.I. 11121 (M)
C.I. 12034 (M)
C.I. 12949 (M)
C.I. 13797 (M)
C.I. 14273 (M)
Erbet (Mt.)
Georgie (Mt.)
Ingrid (Mt.)
Vanguard (Mt.)

Resistant or intermediate to two virulence types

C.I. 2330
C.I. 9327 (M)
C.I. 10364 (M)
C.I. 10615 (M)
C.I. 11056 (M)
C.I. 12894 (M)
C.I. 14093 (M)
C.I. 14218 (M)
Hector (Mt.)
Horsford (Mt.)
Karl (Mt.)
Larker (Mt.)
Palliser (Mt.)
Shabet (Mt.)

Resistant or intermediate to one virulence type

C.I. 4179 (M)
C.I. 7725 (M)
C.I. 10132 (M)
C.I. 14354 (M)
Betzes (Mt.)

Table 4-2 (cont.)

Susceptible to all nine virulence types

Arimont (Mt.)
Compana (Mt.)
Erbet (Mt.)
Firlbecks III (Mt.)
Mona-Arivat

Table 4-3. Relative resistance of barley lines to net blotch, in relation to previous screening tests.

Resistant to	Lines Previously Tested to Moroccan Isolates[1]	Lines Previously Tested to N. American Isolates[2]	Montana Grown Varieties	Others	Total
Nine types	0	3 (7.5%)	0	0	3 (2.0%)
Eight types	1 (1.3%)	3 (7.5%)	0	0	4 (2.7%)
Seven types	4 (5.1%)	4 (10.0%)	0	0	8 (5.4%)
Six types	5 (6.4%)	7 (17.5%)	2 (8.7%)	1	15 (10.2%)
Five types	3 (3.8%)	5 (12.5%)	0	0	8 (5.4%)
Four types	12 (15.4%)	3 (7.5%)	1 (4.3%)	0	16 (10.9%)
Three types	19 (24.4%)	5 (12.5%)	4 (17.4%)	2	30 (20.4%)
Two types	10 (12.8%)	7 (17.5%)	3 (13.0%)	0	20 (13.6%)
One type	14 (17.9%)	3 (7.5%)	6 (26.1%)	2	25 (17.0%)
None	10 (12.8%)	0	7 (30.4%)	1	18 (12.2%)
	78 (100%)	40 (100%)	23 (100%)	6	147 (100%)

[1]Caddel and Wilcoxson, 1975.
[2]Buchannon and McDonald, 1965.

Table 4-4. Relative resistance of barley lines to net blotch, in relation to previous screening tests (resistant and intermediate reactions combined).

Resistant or Intermediate to	Previously Tested to Moroccan Isolates	Previously Tested to N. American Isolates	Montana Grown Varieties	Others	Total
Nine types	3 (3.8%)	5 (12.5%)	1 (4.3%)	0	9 (6.1%)
Eight types	7 (9.0%)	14 (35.0%)	1 (4.3%)	0	22 (15.0%)
Seven types	10 (12.8%)	5 (12.5%)	0	2	17 (11.6%)
Six types	9 (11.5%)	7 (17.5%)	0	0	16 (10.9%)
Five types	17 (21.8%)	2 (5.0%)	0	0	19 (12.9%)
Four types	6 (7.7%)	6 (15.0%)	6 (26.1%)	1	19 (12.9%)
Three types	15 (19.2%)	1 (2.5%)	4 (17.4%)	1	21 (14.3%)
Two types	7 (9.0%)	0	6 (26.1%)	1	14 (9.5%)
One type	4 (5.1%)	0	1 (4.3%)	0	5 (3.4%)
None	0	0	4 (17.4%)	1	5 (3.4%)
	78 (100%)	40 (100%)	23 (100%)	6	147 (100%)

Chapter 5

TESTING OF F_2 POPULATION FOR RESISTANCE TO DIFFERENT ISOLATES OF *Pyrenophora teres*

Once sources of resistance are identified, it is useful to obtain information on the genetics of resistance involved in these sources of resistance. Information concerning the number of resistance genes available, lines with common genes for resistance and the mode of action of these genes is necessary to efficiently exploit these potential sources of resistance.

Crosses were made between resistant lines, between resistant and susceptible lines and between susceptible lines. F_2 populations of these crosses were tested to different isolates of *Pyrenophora teres* in an effort to obtain information regarding the genetics of resistance involved in these crosses.

Materials and Methods

In the summer of 1977, the commercial varieties, Unitan, Steptoe, Firlbecks III, Hypana and Georgie were crossed in all possible combinations with each other. Each of these varieties was also crossed with the varieties Betzes and Tifang and the lines, C.I. 9819, C.I. 9776, C.I. 7584 and C.I. 5791. The commercial varieties were chosen on the basis of earlier tests which indicated these varieties contained resistance to *P. teres*. Ten F_1 seeds of each of these crosses were then sent to Arizona and grown during the winter of

1977-78. The four most vigorous F_1 plants were chosen, where possible, for harvesting F_2 seed.

F_2 seed of crosses made by Dr. Harold Bockelman during the summer of 1976 were also tested.

These crosses were:

Tifang X C.I. 4544	C.I. 5791 X Tifang	C.I. 9819 X C.I. 1197
Tifang X C.I. 2330	C.I. 7584 X Tifang	C.I. 9819 X C.I. 5791
Tifang X C.I. 5791	C.I. 9819 X Tifang	C.I. 7584 X C.I. 9819
Tifang X C.I. 7584	C.I. 7584 X C.I. 5791	C.I. 7584 X C.I. 1197
Tifang X C.I. 9819	C.I. 9819 X C.I. 2235	C.I. 7584 X C.I. 9776
Tifang X C.I. 9776	C.I. 2235 X C.I. 9819	
C.I. 2235 X Tifang	C.I. 9819 X C.I. 9776	

Testing of the F_2 seed was performed in the greenhouse. Twenty-five F_2 seeds were planted in each of six rows across the width of metal flats 14" x 10" x 3", with half rows planted with ten seeds of each parent, a susceptible check (Dekap), and, when available, F_1 seed. Four flats of each cross were planted for each isolate to which the cross was tested, with each flat containing F_2 seed from one of the four F_1 plants harvested in Arizona. The isolates of *P. teres* used in the tests were grown on V8 juice agar in an incubation chamber at 17-18° C, with an eight hour photoperiod. Cultures were transferred on the same day the seed was planted. Inoculation took place when the plants were 7-8 days old. Conidial suspensions were used

as inoculum with 0.1 ml of 1% Tween 20 added per 100 ml of inoculum. The inoculum was sprayed on the plants using an atomizer driven by compressed air. Fifteen to eighteen ml of inoculum were used per flat. The spore concentration was not standardized, with concentrations ranging from 10,000 - 90,000 spores per ml. Inoculated plants were placed in a dew chamber at 100% relative humidity and no light. The wall temperature was kept at 7° C, and the water bath at 35 C, with a resulting air temperature of 24 -27° C. After 20-24 hours, the plants were removed from the dew chamber and placed on benches in an isolation greenhouse. Disease symptoms were read 7-8 days after inoculation.

Results and Discussion

Table 5-1 lists the results of the tests of the crosses to different isolates. The tests are grouped on the basis of the isolate used. Therefore, crosses are listed more than once, with the reactions of the parents, F_1 generation (whenever available) and F_2 generation to the different isolates listed accordingly. The different reaction types of the parents and F_1 generations are listed. The reaction types of the F_2 generations are separated into resistant and susceptible types, with the reaction types 1, 2 and 3 considered resistant and reaction type 4 considered susceptible.

Barley lines with genes in common. The determination of common genes for resistance in different barley lines is relatively straightforward. The F_2 generation of a cross between two resistant barley lines should contain no susceptible plants if the two parents share a common gene for resistance. Because of the possibility of mixed seed, the occurrence of a small amount of outcrossing, and the appearance of offtypes, susceptible plants can occur in crosses between lines sharing a common gene for resistance. However, the percentage of susceptible plants in the F_2 generation should be low enough that the ratio of resistant to susceptible plants will not easily fit the expected ratio of any probable gene models.

Of the crosses tested in this study, four appeared to share a common gene for resistance against every isolate of *P. teres* to which they were tested. These crosses were: Unitan x Steptoe, C.I. 5791 x Unitan, C.I. 5791 x Steptoe and Tifang x C.I. 9819 (Table 5-2).

The results of these tests indicate that Unitan, Steptoe and C.I. 5791 contain much the same genetic information for resistance to *P. teres*. There is evidence that Steptoe and C.I. 5791 do not contain identical genes for resistance, though. When the crosses C.I. 9819 x C.I. 5791 and C.I. 9819 x Steptoe were tested to Mor 15, C.I. 9819 and C.I. 5791 appeared to share a common gene for resistance, while the F_2 generation of the cross C.I. 9819 x Steptoe segregated in a 15:1 ratio of resistant to susceptible plants (Table 5-1).

This indicated that C.I. 9819 and Steptoe each contain a single different dominant gene for resistance. This is also evidence that C.I. 5791 and Steptoe do not contain the same genes for resistance. The cross Unitan x Steptoe was tested to Mor 15 and there is evidence that these two varieties share a common gene effective against this isolate (Table 5-1). Since both Unitan and Steptoe appear to contain only one dominant gene for resistance to Mor 15, this suggests that Unitan and C.I. 5791 also do not contain identical genes for resistance.

The cross Tifang x C.I. 9819 and its reciprocal, C.I. 9819 x Tifang, also appeared to have common genes for resistance to three isolates of *P. teres*. Tifang is susceptible to several isolates, for example Mt 77-1 and Mt 77-51, while C.I. 9819 is resistant, indicating that C.I. 9819 contains additional genes for resistance other than those it shares with Tifang. Other crosses showed evidence for sharing genes for resistance to some, but not all, of the isolates to which they were tested, indicating the lines involved in these crosses did not contain identical genes for resistance.

In several crosses, such as Steptoe x C.I. 9776 and Unitan x C.I. 9776, the number of susceptible plants that appeared in the F_2 generation produced a ratio which fit a gene model consisting of three dominant genes. Because the parents do contain more than one gene for resistance, this type of gene model is very possible. The gene

ratio for three dominant genes and a situation in which a few susceptible plants appear in the F_2 generation of a cross in which the parents share a common gene for resistance cannot readily be distinguished without testing F_3 lines. Therefore, crosses of this type are listed as having possible genes in common (Table 5-2).

Number of genes for resistance to *P. teres*. It was possible, from the data, to determine a minimum number of genes for resistance in each line, but due to the possibility, and probable occurrence, of genes for resistance being effective against more than one isolate of *P. teres*, a definite number of genes could not be determined (Table 5-3).

Steptoe. Crosses with susceptible varieties indicated that Steptoe contains two dominant genes for resistance to several isolates, including Mt 77-3 and Mt 77-6. There is also evidence that Steptoe shares a common gene for resistance with Betzes effective against Mor 18. Since Betzes is susceptible to Mt 77-6 and Mt 77-3, this common gene must be different than the two genes effective against these isolates. Therefore, Steptoe probably contains at least three dominant genes for resistance to *P. teres*.

Unitan. Unitan also contains two dominant genes for resistance effective against several isolates, including Pt S-2 and Mt 77-3, based on crosses with susceptible varieties. Unitan also probably shares with Betzes a common gene for resistance effective against Mor 18. Since Betzes is susceptible to Pt S-2 and Mt 77-3, Unitan, like Steptoe,

probably contains at least three dominant genes for resistance.

<u>C.I. 9819</u>. C.I. 9819 shares common genes for resistance to Pt S-2 with Tifang and Unitan. It also shares a gene for resistance effective against Mor 1 with Tifang, but not Unitan. This gene effective against Mor 1 is therefore different than the one effective against Pt S-2. C.I. 9819 is resistant to Mt 77-1, an isolate to which Tifang is susceptible. The gene in C.I. 9819 effective against Mt 77-1 is therefore different than either the gene for resistance to Mor 1 or the gene effective against Pt S-2. C.I. 9819 therefore contains at least three dominant genes for resistance.

<u>C.I. 5791</u>. C.I. 5791 shares a common gene for resistance with C.I. 7584 effective against Mor 18. C.I. 5791 also contains two dominant genes for resistance to Mor 1. C.I. 7584 is susceptible to Mor 1. Therefore, the gene for resistance shared by C.I. 5791 and C.I. 7584 is different from the two genes located in C.I. 5791 which are effective against Mor 1. C.I. 5791, then, contains at least three genes for resistance to *P. teres*.

<u>Tifang</u>. Tifang and C.I. 7584 have a gene in common effective against Pt S-2. They each carry single different genes for resistance to Pt 8. A third gene cannot be positively separated from these two genes. Tifang contains at least two genes for resistance.

<u>C.I. 7584</u>. C.I. 7584 contains a gene in common with Tifang for resistance to Pt S-2, plus a single dominant gene effective against Pt 8

different than that in Tifang. C.I. 7584 contains at least two genes for resistance.

C.I. 1197. C.I. 1197 and C.I. 9819 share a common gene for resistance to Mor 15, yet have different genes for resistance to Mt 77-1. C.I. 1197 and C.I. 7584 have a gene in common for resistance to Aust 1, but are dissimilar in resistance to Mor 15. From this it can be concluded that C.I. 1197 has at least two genes for resistance.

C.I. 9776. C.I. 9776 and C.I. 7584 share a common gene for resistance to Pt S-2. C.I. 9776 is resistant to Mor 1 and C.I. 7584 is susceptible. C.I. 9776 must therefore contain another gene for resistance other than the one it shares with C.I. 7584. Hence, C.I. 9776 has at least two genes for resistance to *P. teres*.

C.I. 2235. Crosses with C.I. 9819 tested to Pt T-1 and Mt 77-1 indicate that C.I. 2235 has at least one dominant gene for resistance to *P. teres*.

Betzes. Betzes shares a common gene for resistance to Mor 18 with Steptoe and Unitan. Betzes also has a gene in common with Georgie effective against ELS 77-6. It cannot be determined whether or not this is the same gene or different genes. Betzes contains at least one gene for resistance to *P. teres*.

Georgie. Georgie has at least one gene for resistance in common with Betzes, effective against ELS 77-6.

Firlbecks III. Firlbecks III contains at least one dominant gene

effective against Mor 18, different than that found in Betzes.

C.I. 2330 and C.I. 4544. C.I. 2330 and C.I. 4544 each share a common gene for resistance with Tifang effective against Pt S-2. This is the only isolate to which these lines were tested.

Total number of resistance genes. The total number of different resistance genes available in these barley lines cannot be determined from these data. A minimum number of resistance genes can be distinguished, however. Unitan, C.I. 9819 and C.I. 5791 all have at least three genes for resistance. Indirect evidence suggests that while C.I. 9819 and C.I. 5791 share a common gene for resistance to Mor 15, C.I. 5791 and Unitan do not. C.I. 5791 and Unitan probably do share a common gene for resistance to Mor 1, while C.I. 9819 and Unitan do not (Table 5-1). This suggests that the resistance in C.I. 9819 effective against Mor 1 is different than the resistance in C.I. 5791 effective against this isolate.

There exists, then, three barley lines, each with at least three different genes for resistance, yet none of the three are identical in resistance. For example, Unitan may contain three genes, Pt a, Pt b and Pt c, with gene Pt a effective against Pt S-2, gene Pt b effective against Mor 1 and gene Pt c effective against Mor 15. C.I. 5791 could then contain Pt a and Pt b since it has genes in common with Unitan for resistance to Pt S-2 and Mor 1, but it must have a different gene, gene Pt d, effective against Mor 15. C.I. 9819 contains

genes Pt a and Pt d, since it shares a common gene for resistance to Pt S-2 with Unitan and to Mor 15 with C.I. 5791, but it must have another different gene effective against Mor 1, gene Pt e.

One may then conclude that at least five genes for resistance exist, with Unitan containing genes Pt a, Pt b, and Pt c, C.I. 5791 containing genes Pt a, Pt b, and Pt d, and C.I. 9819 containing the genes Pt a, Pt d and Pt e. The designation of different letters to each gene is for the sake of explanation only.

The evidence for the occurrence of five genes is indirect, yet this explanation is plausible. Further tests, especially tests involving F_3 lines from these crosses, must be performed before more conclusive information is obtained. It must be stressed that these five genes are the minimum number that could be distinguished and it is possible that with further testing more genes will be differentiated.

It was not possible to confidently distinguish between a situation involving three dominant genes and one involving a common gene for resistance when low percentages of susceptible plants appeared. The possibility of linkage also exists. Further studies involving F_3 families are necessary in order to make any conclusions involving these possibilities.

Tifang was found to have a single, incompletely dominant gene for resistance to a California isolate of *P. teres* (Schaller, 1955).

This gene was found to be linked to a resistance gene in C.I. 4797 and C.I. 2750 with a recombination percentage of 2.57 (Mode and Schaller, 1958). Khan and Boyd (1969b) found Tifang and C.I. 4797 to have a common gene for resistance to an Australian isolate of *P. teres*. From this the inference can be made that Tifang has two genes for resistance to *P. teres*. Khan and Boyd (1969b) also found that Tifang, C.I. 2330, C.I. 4797, C.I. 9819 and C.I. 5791 all share a common gene for resistance to this Australian isolate. The results of the present study indicate two genes for resistance in Tifang with Tifang having genes in common with C.I. 2330, C.I. 9819 and possibly C.I. 5791.

Bockelman, et al. (1977), found Tifang to contain a single dominant gene for resistance to Pt 8, located on chromosome 3, and C.I. 7584 to contain a single gene for resistance to Pt 8 located on chomosome 2. The data from the present study indicate that Tifang and C.I. 7584 contain single, different dominant genes for resistance to Pt 8.

Expression of genetic resistance. In several instances, the reaction of a barley line to a particular isolate varied from test to test. This was especially evident with Tifang, but also occurred with C.I. 7584. Tifang varied from an intermediate (2,3) to a totally susceptible (4) reaction in response to the isolate Pt R. It also varied from an intermediate (2,3) to a susceptible (4,3) reaction in response

to Mor 1. The reaction of C.I. 7584 varied from a moderately resistant (2) reaction to a susceptible (4,3) reaction when tested with Mor 15 and from a resistant (1) to a variable (2,4,3) type of reaction in response to Pt R.

The cause of this variation could not be determined. Khan and Boyd (1969c) observed a breakdown in resistance of C.I. 2330 with increasing spore concentration, but not with Tifang. There was no correlation between spore concentration and resistance in the tests performed during the present study. Khan (1969), and Khan and Boyd (1969c) also found the temperature during the pre-inoculation period, the incubation period and the post inoculation period to have an effect on the resistance of some varieties, but not others. These temperatures were not recorded for each test, so their effects can be neither discounted nor confirmed. The possibility of changes in the virulence of the isolates used must also be considered.

The variability of the parents extended to the F_2 generation. For example, the cross C.I. 7584 x Tifang was tested twice to Pt R. In one test C.I. 7584 showed a resistant (1,2) reaction, Tifang was intermediate (2,3) and the F_2 generation showed a total of 346 resistant and 58 susceptible plants. In the other test C.I. 7584 was variable (2,4,3) in reaction, Tifang was susceptible (4) and the F_2 generation had 72 resistant and 304 susceptible plants.

That the two parents reacted similarly in both tests indicates

that the factors causing a breakdown in resistance are probably the same for both lines. Because of the change of the F_2 reaction, there is a possibility that radically different conclusions concerning the type of resistance involved could be drawn.

Khan (1969) found that variability of host reaction in the progeny of resistant varieties was influenced by the genetic background of the susceptible parent used. By using the resistant line C.I. 5791 and the susceptible lines Dampier and Atlas, he showed that the F_2 generation of Dampier x C.I. 5791 expressed a higher degree of resistance than the F_2 generation of Atlas x C.I. 5791. The F_1 generation of the cross with Dampier also showed a higher degree of resistance than the F_1 of the cross with Atlas.

In the present study, evidence was found supporting the differential effects of susceptible varieties on the expression of resistance. The susceptible varieties Hypana and Georgie were crossed with the resistant varieties Steptoe and Unitan and the resistant line C.I. 7584. The F_1 and F_2 generations were tested to the isolate Pt R. The crosses involving Hypana showed a higher degree of resistance in both the F_1 and F_2 generations than the crosses involving Georgie (Table 5-4). These results indicate that the genetic background of Hypana is more conducive to the expression of resistance than the genetic background of Georgie.

Possible additive resistance in barley to *P. teres*. Five crosses

were tested, using isolates to which both parents in each cross were susceptible. It would be expected that a cross involving two susceptible parents would result in a susceptible F_2 population. However, in the F_2 populations of these crosses there appeared plants showing a higher degree of resistance than either parent (Table 5-5).

This observed transgressive segregation suggests the possibility of additive resistance to *P. teres*. It is possible that the susceptible lines contain genes which are not effective by themselves, but when combined produce an observable resistant reaction. It must be pointed out that each of the lines used as parents in these crosses contains at least one gene for resistance effective against other isolates of *P. teres*. These genes appear to have a major effect against the isolates to which they are effective. If these major effect genes are different, then it is possible that when they are combined they will have an additive effect against isolates to which each alone is ineffective.

Nelson, et al. (1970), reported that genes for vertical resistance in maize not only govern qualitative resistance to some isolates of *Trichometasphaeria turcica* (*Helminthosporium turcicum*), the causal organism of leaf blight disease of maize, but also quantitatively condition the amount of disease.

It is also possible that there are present in these lines minor effect genes that, when combined, produce an observable increase in

resistance. Parlevliet (1976) reported the occurrence in barley of minor effect genes with additive resistance to leaf rust (*Puccinia hordei*).

Lewellen and Sharp (1968) investigated the inheritance of minor effect genes in wheat for resistance to stripe rust (*Puccinia striiformis*). These genes are temperature sensitive with a group of genes in the variety Rego effective at a low temperature profile and another group in the line P.I. 178383 effective at a high temperature profile. When these two groups of genes are combined there is an additive interaction resulting in greater resistance at both temperature profiles.

Sharp, et al. (1976), showed that varieties of wheat susceptible to strip rust could be crossed and, after several generations of selection, produce progeny with high levels of resistance.

Since additive resistance has been shown to result from the combination of genes with both major and minor effects, and since it has been shown that susceptible lines can be combined to produce resistant progeny, it is desirable to investigate further the possibility of additive resistance to *P. teres* in barley.

Table 5-1. Reactions of F_2 populations tested to different isolates of *P. teres*.

Cross	F_1 reaction	F_2 reaction[1] Res.	Susc.	Possible Gene Model	Chi Square Value
Crosses tested to Pt T-1					
Tifang (1)[2] x C.I. 9819 (1,2)	no F_1	396	4	three dominant genes	.50 - .30
				same gene	----
C.I. 9819 (1,2) x Tifang (1)	no F_1	209	2	three dominant genes	.50 - .30
				same gene	----
C.I. 9819 (1,2) x C.I. 2235 (1,2,3)	no F_1	278	17	two dominant genes	.75 - .70
				two dominant, one recessive gene	.50 - .30
C.I. 9819 (1) x C.I. 2235 (1,2)	no F_1	299	8	two dominant, one recessive gene	.70 - .50
Unitan (1,2) x C.I. 9776 (1)	F_1; 1	316	3	same gene	----
				three dominant genes	.50 - .30
Steptoe (1,2) x C.I. 9776 (1)	no F_1	93	3	same gene	----
				two dominant, one recessive gene	.50 - .30
				two or three dominant genes	.25 - .20
C.I. 5791 (1,2) x Steptoe (1)	F_1; 1	394	0	same gene	----
C.I. 7584 (1) x Unitan (1,2)	F_1; 1	335	0	same gene	----
C.I. 2235 (1) x Tifang (1)	no F_1	96	1	same gene	----
				three dominant genes	.70 - .50
Crosses tested to Pt 8					
Tifang (1,2) x C.I. 7584 (1,2)	no F_1	144	8	two dominant genes	.70 - .50
				two dominant, one recessive gene	.70 - .50
Tifang (2,1) x C.I. 7584 (1,2)	no F_1	295	13	two dominant genes	.20 - .10
				two dominant, one recessive gene	.75 - .70
C.I. 7584 (2,1) x Tifang (2,1)	no F_1	345	35	two dominant genes	.025 - .01
C.I. 7584 (2,1) x Tifang (1,2)	no F_1	369	18	two dominant genes	.20 - .10
				two dominant, one recessive gene	.99 - .95

[1]Reaction types 1, 2 and 3 considered resistant, reaction type 4 considered susceptible.

[2]Parental reaction types in parentheses.

Table 5-1 (cont.)

Cross	F_1 reaction	F_2 reaction		Possible Gene Model	Chi Square Value
		Res.	Susc.		
Crosses tested to Pt 10					
Betzes (4) x Hypana (-)	no F_1	96	264	one recessive gene	.50 - .30
Betzes (4) x Hypana (2)	F_1; 1,2,4	105	305	one recessive gene	.80 - .75
Betzes (4) x Unitan (1,2)	F_1; 1,2	342	82	one recessive, one dominant gene	.80 - .75
Tifang (2,3) x C.I. 5791 (3,2)	no F_1	169	59	one dominant gene	.80 - .75
C.I. 7584 (-) x C.I. 5791 (-)	no F_1	63	2	same gene	----
				three dominant genes	.50 - .30
				two dominant genes	.30 - .25
Crosses tested to Mt 77-1					
C.I. 9776 (1) x Georgie (4,3)	F_1; 1	196	80	one dominant gene	.20 - .10
Betzes (4) x Steptoe (1,2)	F_1; 1,2	159	26	one dominant, two recessive genes	.995 - .990
C.I. 9819 (1,2) x C.I. 5791 (1,2)	no F_1	207	3	same gene	----
				three dominant genes	.90 - .80
C.I. 9819 (1,2) x C.I. 2235 (1,2)	no F_1	396	19	two dominant genes	.20 - .10
				two dominant, one recessive gene	.95 - .90
C.I. 9819 (1,2) x C.I. 2235 (1,2)	no F_1	301	3	same gene	----
				three dominant genes	.50 - .30
C.I. 7584 (2,1) x C.I. 9819 (1)	no F_1	203	7	two dominant genes	.10 - .05
				two dominant, one recessive gene	.50 - .30
C.I. 9819 (1,2) x C.I. 1197 (2,1)	no F_1	309	67	one dominant, one recessive gene	.70 - .50
C.I. 9819 (1,2) x Steptoe (1,2)	F_1; 1	394	0	same gene	----
Tifang (4) x Georgie (4)	F_1; 4	25	158	both parents susceptible	----

Table 5-1 (cont.)

Cross	F_1 reaction	F_2 reaction Res.	F_2 reaction Susc.	Possible Gene Model	Chi Square Value
Crosses tested to Mt 77-2					
C.I. 9819 (1,2) x Firlbecks III (3,2,4)	F_1; 1,2,3	328	105	one dominant gene	.75 - .70
C.I. 5791 (1) x Georgie (4,3)	F_1; 1,2	348	36	two dominant genes	.025 - .01
C.I. 5791 (1,2) x Unitan (1,2)	F_1; 1,2	207	6	same gene	----
				three dominant genes	.20 - .10
				two dominant, one recessive gene	.20 - .10
C.I. 5791 (1,2) x Steptoe (1)	F_1; 1	376	0	same gene	----
C.I. 9819 (1) x Unitan (1,2)	F_1; 1	80	0	same gene	----
Crosses tested to Mt 77-3					
Firlbecks III (4,2,3) x Steptoe (1,2)	F_1; 1,2	163	13	two dominant genes	.70 - .50
Hypana (4) x Unitan (1,2)	F_1; 2,3	265	18	two dominant genes	.95 - .90
Tifang (1) x Unitan (1,2)	F_1; 1	191	1	same gene	----
Crosses tested to Mt 77-5I					
C.I. 5791 (1) x Firlbecks III (3,4)	F_1; 1,2	386	37	two dominant genes	.05 - .025
C.I. 9819 (1,2) x Firlbecks III (4,3)	F_1; 1,2,3	348	42	one dominant, two recessive genes	.10 - .05
Tifang (4,3) x Firlbecks III (3,4)	F_1; 3,2	209	182	both parents susceptible	----
Crosses tested to Mt 77-6					
Firlbecks III (4,2,3) x Steptoe (1)	F_1; 1	210	13	two dominant genes	.90 - .80
Betzes (4) x Georgie (3,4)	F_1; 3,2	47	254	both parents susceptible	----
Tifang (1,2) x Steptoe (1)	F_1; 1	119	3	three dominant genes	.50 - .30
				same gene	----
C.I. 7584 (1) x Unitan (1,2)	F_1; 1	242	0	same gene	----
Georgie (3,4) x Firlbecks III (4,3)	F_1; 3,2,4	216	155	both parents susceptible	----

Table 5-1 (cont.)

Cross	F_1 reaction	F_2 reaction Res.	Susc.	Possible Gene Model	Chi Square Value
<u>Crosses tested to Mt 77-7</u>					
C.I. 9776 (1,3,2) x Georgie (3,4,2)	no F_1	70	37	one dominant gene	.025 - .01
C.I. 9776 (-) x Firlbecks III (-)	F_1; 2,1	158	5	three dominant genes	.20 - .10
				two dominant genes	.10 - .05
Betzes (4) x Firlbecks III (4,3,2)	F_1; 4,3	41	328	both parents susceptible	----
<u>Crosses tested to Aust 1</u>					
C.I. 7584 (1) x C.I. 1197 (1)	no F_1	86	0	same gene	----
Firlbecks III (2,4) x Unitan (1,0)	F_1; 1	233	6	three dominant genes	.25 - .20
				same gene	----
<u>Crosses tested to ELS 77-6</u>					
Bestzes (1,2) x Georgie (2,3)	no F_1	179	3	same gene	----
				three dominant genes	.95 - .90
<u>Crosses tested to Mor 1</u>					
Unitan (2,1) x C.I. 9776 (1)	F_1; 1	296	6	three dominant genes	.70 - .50
				same gene	----
C.I. 5791 (1) x Unitan (1,2)	no F_1	325	2	same gene	----
				three dominant genes	.20 - .10
C.I. 9819 (1) x Unitan (3,2)	F_1; 1	193	13	two dominant genes	.95 - .90
Steptoe (-) x C.I. 9776 (-)	no F_1	97	3	same gene	----
				two dominant, one recessive gene	.50 - .30
				two or three dominant genes	.25 - .20

Table 5-1 (cont.)

Cross	F_1 reaction	F_2 reaction Res.	Susc.	Possible Gene Model	Chi Square Value
Crosses tested to Mor 1 (cont.)					
C.I. 5791 (1) x Steptoe (2)	F_1; 1	340	1	same gene	----
C.I. 7584 (3,4) x C.I. 5791 (1,2)	no F_1	65	7	two dominant genes	.25 - .20
C.I. 7584 (4,3) x C.I. 9776 (1)	no F_1	379	6	three dominant genes	.995 - .990
C.I. 5791 (1,2) x Tifang (4,3)	no F_1	195	25	one dominant, two recessive genes	.25 - .20
Tifang (3,2) x Firlbecks III (3,2)	F_1; 2	305	90	one dominant gene	.50 - .30
Tifang (4,3) x C.I. 9776 (1,2)	no F_1	200	51	one dominant gene	.10 - .05
C.I. 9819 (1) x Tifang (2,3)	no F_1	429	0	same gene	----
Tifang (2,3,4) x C.I. 9819 (1,2)	no F_1	228	2	same gene three dominant genes	---- .50 - .30
Tifang (2,3) x C.I. 9819 (1)	no F_1	286	1	same gene	----
Crosses tested to Mor 14					
C.I. 9819 (1,2) x Steptoe (3,2)	F_1; 2,1	361	51	one dominant, two recessive genes	.20 - .10
Crosses tested to Mor 15					
C.I. 7584 (2,3) x Unitan (1,2)	F_1; 2	303	29	two dominant genes	.05 - .025
Betzes (-) x Unitan (-)	no F_1	368	50	one dominant, two recessive genes	.25 - .20
Firlbecks III (4,1) x Unitan (1,2)	F_1; 1,2	274	9	two dominant, one recessive gene	.25 - .20
Tifang (1,2) x Unitan (1)	F_1; 2,1	179	8	two dominant, one recessive gene two dominant genes	.80 - .75 .30 - .25
Unitan (1,2,3) x Steptoe (1,2)	no F_1	88	1	same gene three dominant genes	---- .75 - .70