EPIDEMIOLOGY OF *PYRENOPHORA TERES* AND ITS EFFECT ON GRAIN YIELD OF *HORDEUM VULGARE*

A Thesis

Submitted to the Faculty of Graduate Studies and Research

in Partial Fulfillment of the Requirements

for the Degree of

Doctor of Philosophy

in the

Department of Crop Science and Plant Ecology

University of Saskatchewan

Saskatoon

by

Cornelius Gerardus Jacobus van den Berg

July, 1988

ABSTRACT

Recent surveys in Saskatchewan have shown that the prevalence of Pyrenophora teres Drechs., the causal agent of net blotch of barley (Hordeum vulgare L.), has increased. Severe epidemics have been observed in several commercial fields. The major objectives of this study were the investigation of the epidemiology of net blotch and the determination of the effect of net blotch on barley production.

Conidium germination and time to infection were compared for two spot-type isolates (P. teres f. maculata) and one net-type isolate (P. teres f. teres) in a growth cabinet. Spot-type isolates germinated sooner than the net-type isolate between 10 and 20 ^{o}C. Time to infection was shorter for the spot-type isolates than for the net-type isolate at all temperatures.

The relationships among local weather conditions, incidence of airborne conidia, barley development and sporulation were investigated in field experiments with the susceptible cultivar Elrose at Shellbrook, Saskatchewan in 1986 and 1987. Airborne conidia were observed daily throughout the growing season. Their incidence showed a diurnal pattern with a peak between 12:00 and 16:00 h. At night, frequent dew formation favoured infection. Infection of lower leaves was due to infection by primary inoculum produced on crop debris. Infection of upper leaves was

due to primary inoculum and to secondary inoculum produced on the lower leaves.

The effect of net blotch on barley production was investigated in an experiment using Tilt (propiconazole) on Elrose in 1985 and 1986 and on the moderately resistant cultivar Argyle in 1986 at Saskatoon, Shellbrook and Medstead, Saskatchewan. Repeated application of Tilt reduced the rate of disease progress on Elrose, but not on Argyle, where resistance reduced the rate of disease progress. Resistance slowed disease progress more than repeated application of Tilt. On Elrose, Tilt increased grain yield and kernel weight, but had no effect on number of tillers and number of kernels per spike. On Argyle, Tilt had no effect on any trait.

The potential yield loss caused by net blotch was estimated at 50%. Infection of net blotch lowered grain grade through kernel discolouration and low test weight. Using the components of variance and covariance, strong correlations were obtained between treatment effects of grain yield and disease severity. However, no satisfactory critical point model could be developed to predict percentage yield loss from disease severity.

In Saskatchewan, infected crop debris is the most important source of primary inoculum of P. teres. Under favourable conditions with sufficient primary inoculum, severe epidemics can be expected on susceptible cultivars. Foliar fungicide application is not cost effective at

current grain prices, therefore extended rotations or the use of resistant cultivars are the only practical means to control net blotch in barley.

ACKNOWLEDGEMENTS

I wish to thank Dr. B.G. Rossnagel for his guidance and continuous support throughout this study; Drs. L.J. Duczek, B.L. Harvey, D.R. Knott and R.A.A. Morrall for their constructive criticism and suggestions; Mr. C. Brown and Miss J. Weller for technical assistance.

Financial support was provided by a Postgraduate Scholarship from the University of Saskatchewan, the R. Vandeveld postgraduate scholarship and the Western Grains Research Foundation.

Also, I would like to thank Dr. R.J. Baker for the stimulating discussions on statistics and the Department of Interlibrary Loans for the prompt delivery of many references.

Last, but not least I wish to thank Dr. L.S.L. Wong for her inexhaustible patience, her encouragement and support.

TABLE OF CONTENTS

ABSTRACT iv

ACKNOWLEDGEMENTS vii

TABLE OF CONTENTS viii

LIST OF FIGURES xi

LIST OF TABLES xiii

LIST OF ABBREVIATIONS xvi

1. INTRODUCTION 1

2. LITERATURE REVIEW 3

2.1. Pathosystem 3
2.1.1. Host 3
2.1.1.1. Morphology 3
2.1.1.2. Agronomy 4
2.1.2. Pathogen 5
2.1.2.1. Morphology 5
2.1.2.2. Taxonomy 6
2.1.2.3. Symptomatology 8
2.1.3. Distribution, prevalence and importance . 9
2.1.4. Disease cycle 10

2.2. Measurement and analysis of disease progress ... 13
2.2.1. Measurement 13
2.2.2. Analysis 16
2.2.3. Effect of fungicides on disease progress 20
2.2.4. Progress of net blotch 21

2.3. Effect of disease on plant production 23
2.3.1. Measurement of the effect on grain yield 23
2.3.2. Measurement of the effect on primary yield components 27
2.3.3. Effect of net blotch on barley production 30
2.3.4. Effect of net blotch on kernel quality .. 32

2.4. Relationship between grain yield and disease severity 33
2.4.1. Linear regression analysis 33
2.4.2. Critical point analysis 35
2.4.3. Analysis using area under the disease progress curve 37
2.4.4. Multiple point analysis 38

3. MATERIALS AND METHODS 40

3.1. Experiment I: Conditions for infection 40

3.2. Experiment II: Potential for disease establishment 45

3.3. Experiment III: Effect of fungicides on the pathosystem 52

4. RESULTS AND DISCUSSION 62

4.1. Conditions for infection 62
4.1.1. Conidium germination 62
4.1.2. Time to infection 72
4.1.3. Discussion 75

4.2. Potential for disease establishment in Saskatchewan 76
4.2.1. Microclimate 76
4.2.2. Development of barley 81
4.2.3. Progress of net blotch 81
4.2.4. Sporulation potential and latent period . 85
4.2.5. Incidence of airborne conidia 90
4.2.6. Discussion 96

4.3. Progress curves for net blotch 97
4.3.1. Fitting of disease progress curves 98
4.3.1.1. Estimation of parameters 98
4.3.1.2. Biological interpretation 99
4.3.2. Observed progression for net blotch 101
4.3.2.1. Percentage necrotic leaf area .. 101
4.3.2.2. Percentage affected leaf area .. 105
4.3.3. Discussion 110

4.4. Effect of net blotch on barley 111
4.4.1. Percentage affected leaf area 111
4.4.2. Grain yield 117
4.4.2.1. Effect of fungicide application 117
4.4.2.2. Estimation of potential yield loss 120
4.4.3. Primary yield components 124
4.4.3.1. Effect of fungicide application 124
4.4.3.2. Relationship between grain yield and yield components 128
4.4.4. Grain quality 129
4.4.5. Discussion 130

4.5. Relationship between disease severity and grain yield 133
4.5.1. Relationship between independently measured variables 133
4.5.2. Correlation 138
4.5.3. Regression 142
4.5.4. Discussion 146

5. GENERAL DISCUSSION 148

5.1. Infection cycle 148

5.2. Sources of inoculum 149

5.3. Effect of net blotch on barley 150

5.4. Disease control 151

5.5. Net blotch in Saskatchewan: Prospects 153

5.6. Suggestions for future research 155

REFERENCES 157

APPENDIX A. Tables 168

APPENDIX B. Figures 187

LIST OF FIGURES

Figure 2.1.1. Disease cycle of Pyrenophora teres on barley (Adapted from Mauler, 1983. p. 28) 11

Figure 2.2.1. Standard area diagrams for the assessment of severity of net-type symptoms of Pyrenophora teres on barley (Source Hampton and Arnst, 1978. p. 18-4) .. 15

Figure 2.2.2. Disease progress curves: 1 = linear, 2 = monomolecular, 3 = logistic (Source Hau and Kranz, 1977. p. 62) 18

Figure 2.3.1. Yield levels and associated yield losses (Adapted from Zadoks and Schein, 1979. p. 245) 24

Figure 2.3.2. Yield components in barley and their determination during crop development (Source Teng and Gaunt, 1980. p. 139) 29

Figure 3.1.1. Humidity box with inoculated leaves (modified from Ubels, 1979) 42

Figure 3.2.1. Standard area diagrams used to assess percentage necrotic leaf area (Adapted from Hampton and Arnst, 1978. p. 18-4.) 49

Figure 3.2.2. Standard area diagrams used to assess percentage green and chlorotic leaf area (Source James, 1971. p. 44) 50

Figure 4.1.1. Time course of conidium germination for three isolates of Pyrenophora teres at constant 10 °C regime. Vertical bars represent the standard deviation for each incubation period 65

Figure 4.1.2. Time course of conidium germination for three isolates of Pyrenophora teres at constant 15 °C regime. Vertical bars represent the standard deviation for each incubation period 66

Figure 4.1.3. Time course of conidium germination for three isolates of Pyrenophora teres at constant 20 °C regime. Vertical bars represent the standard deviation for each incubation period 67

Figure 4.1.4. Time course of conidium germination for three isolates of Pyrenophora teres at constant 25 °C regime. Vertical bars represent the standard deviation for each incubation period 68

Figure 4.1.5. Time course of conidium germination for three isolates of Pyrenophora teres at 20 decreased to 10 °C regime. Vertical bars represent the standard deviation for each incubation period 69

Figure 4.1.6. Time course of conidium germination for three isolates of Pyrenophora teres at 25 decreased to 10 °C regime. Vertical bars represent the standard deviation for each incubation period 70

Figure 4.2.1. Development of Elrose based on the Zadoks scale (Zadoks et al., 1974) at Shellbrook in 1986 and 1987 82

Figure 4.2.2. Green leaf area for five leaf positions and total green leaf area per main culm in Elrose on seven observation days at Shellbrook in 1986 83

Figure 4.2.3. Green leaf area for seven leaf positions and total green leaf area per main culm in Elrose on eight observation days at Shellbrook in 1987 84

Figure 4.2.4. Percentage necrotic leaf area for five leaf positions in Elrose infected with Pyrenophora teres f. maculata on six observation days at Shellbrook in 1986 86

Figure 4.2.5. Percentage necrotic leaf area for seven leaf positions in Elrose infected with Pyrenophora teres f. maculata on eight observation days at Shellbrook in 1987 87

Figure 4.2.6. Number of conidia of Pyrenophora teres trapped per day at Shellbrook in June 1986 91

Figure 4.2.7. Number of conidia of Pyrenophora teres trapped per day at Shellbrook in June 1987 92

Figure 4.2.8. Number of conidia of Pyrenophora teres trapped per day at Shellbrook in July 1986 93

Figure 4.2.9. Number of conidia of Pyrenophora teres trapped per day at Shellbrook in July 1987 94

Figure 4.2.10. Diurnal trend in incidence of airborne conidia of Pyrenophora teres at Shellbrook in June and July of 1986 and 1987. Percentages of conidia was calculated as the ratio of the observed number to the total number observed during the investigation period multiplied by 100 95

LIST OF TABLES

Table 2.3.1. Estimates of potential yield loss caused by net blotch in barley field experiments 31

Table 3.1.1. Analysis of variance for percentage germinated conidia for each combination of temperature and leaf wetness period 44

Table 3.1.2. Analysis of variance for time to infection 46

Table 3.2.1. Time table of field operations for experiment II in 1986 and 1987 47

Table 3.3.1. Time table of field operations for experiment III at three sites in 1985 and 1986 55

Table 3.3.2. Analysis of variance for parameters estimated for Elrose 58

Table 3.3.3. Analysis of variance for parameters estimated for Argyle 60

Table 3.3.4. Analysis of variance for traits measured for each combination of year and cultivar 61

Table 4.1.1. Mean squares for isolate and error effect for each combination of temperature and leaf wetness period in Experiment I 64

Table 4.1.2. Analysis of variance for time to infection 73

Table 4.1.3. Time to infection for three isolates and six temperature regimes 74

Table 4.2.1. Monthly average maximum temperature, monthly average minimum temperature, monthly precipitation at Shellbrook and Prince Albert in June and July of 1986 and 1987, and monthly normals for the period 1951 - 1980 at Prince Albert 78

Table 4.2.2. Frequency of the duration of leaf wetness periods at Shellbrook in June and July of 1986 and 1987 80

Table 4.2.3. Number of conidia per leaf obtained for each leaf position after one, three and five days of incubation for six sampling dates in 1987 89

Table 4.3.1. Parameters of the monomolecular curve fitted to the percentage necrotic leaf area for two treatments in Elrose in 1985 and 1986 102

Table 4.3.2. Parameters of the exponential curve fitted to the percentage necrotic leaf area for two treatments in Argyle in 1986 104

Table 4.3.3. Parameters of the logistic curve fitted to the percentage affected leaf area for two treatments in Elrose in 1985 and 1986 106

Table 4.3.4. Parameters of the logistic curve fitted to the percentage affected leaf area for two treatments in Argyle in 1986 107

Table 4.4.1. Percentage affected leaf area of penultimate leaves infected with Pyrenophora teres for five treatments in Elrose at three sites in 1985 112

Table 4.4.2. Percentage affected leaf area of penultimate leaves infected with Pyrenophora teres for five treatments in Elrose at three sites in 1986 114

Table 4.4.3. Percentage affected leaf area of penultimate leaves infected with Pyrenophora teres for five treatments in Argyle at three sites in 1986 116

Table 4.4.4. Analysis of variance for grain yield of Elrose at three sites in 1985 and 1986 and of Argyle at three sites in 1986 118

Table 4.4.5. Analysis of variance for grain yield for four fungicide treatments, forming a 2x2 factorial, based on data obtained from the literature for Pyrenophora teres on barley 121

Table 4.4.6. Mean grain yield for five treatments in Elrose at three sites in 1985 and 1986 and for five treatments in Argyle in 1986 122

Table 4.4.7. Mean number of tillers for five treatments in Elrose at three sites in 1985 125

Table 4.4.8. Mean number of kernels per spike for five treatments in Elrose at three sites in 1985 and 1986 and for five treatments in Argyle in 1986 126

Table 4.4.9. Mean kernel weight for five treatments in Elrose at three sites in 1985 and 1986 and for five treatments in Argyle in 1986 127

Table 4.4.10. Grain grade for five treatments in Elrose at three sites in 1985 and 1986 130

Table 4.4.11. Grain grade for five treatments in Argyle at three sites in 1986 132

Table 4.5.1. Analysis of variance and covariance for a randomized complete block design 136

Table 4.5.2. Analysis of variance and covariance for a randomized complete block design 137

Table 4.5.3. Correlation coefficients for treatment, replication and error effect between kernel weight, grain yield, and percentage affected leaf area, observed on different days, at Shellbrook in 1985 139

Table 4.5.4. Correlation coefficients for treatment, replication and error effect between kernel weight, grain yield and percentage affected leaf area observed on different days, at Shellbrook in 1986 140

Table 4.5.5. Regression coefficients and coefficients of determination for regression of treatment effects of percentage affected leaf area on percentage yield loss at Shellbrook in 1985 and 1986 143

Table 4.5.6. The actual and predicted percentage yield loss for four treatments in 1985 and 1986 145

LIST OF ABBREVIATIONS

A	Asymptote
a	Intercept
B	Location parameter
b	Regression coefficient
C	Rate parameter
cov	Covariance
df	Degrees of freedom
F	F-value
i	Counter for summations
ln	Natural logarithm
LSD	Least significant difference
MS	Mean square
n	Number of terms in summations
r	Correlation coefficient; Number of replications
r^2	Coefficient of determination
SD	Standard deviation
SS	Sum of squares
t	Time; Number of treatments
var	Variance
X	Disease severity
X'	Transformed value for disease severity
Y	Percentage yield loss

1. INTRODUCTION

Barley, Hordeum vulgare L., is the second most important cereal crop in Saskatchewan. Annually, approximately 1.2 million ha are planted to spring barley and the production is approximately 3.5 million tonnes (Saskatchewan Agriculture, 1986a).

Pyrenophora teres Drechs., causal agent of net blotch, is a common pathogen of barley, present wherever barley is grown (Commonwealth Mycological Institute, 1977). Recent disease surveys in Saskatchewan indicate that the prevalence of P. teres has increased (Tekauz, 1978) and severe epidemics have been observed in several commercial fields (B.G. Rossnagel, pers. comm.). Infection by P. teres may reduce grain yield by 11 to 40% (Martin et al., 1981; Smedegaard-Petersen, 1974). Reduced grain yield has generally been attributed to reduced kernel weight.

Two forms of P. teres have been described: P. teres f. teres that causes the classical net-type symptoms, and P. teres f. maculata that causes spot-type symptoms (Smedegaard-Petersen, 1971). Until 1974, only isolates causing net-type symptoms were observed in Saskatchewan (Tekauz and Buchannon, 1977). Since that time, isolates causing spot-type symptoms, have spread throughout Saskatchewan, apparently replacing the isolates causing net-type symptoms (Tekauz, 1978; B.G. Rossnagel, pers. comm.)

The epidemiology of net blotch has been investigated in

England (Jordan, 1981) and New Zealand (Sheridan et al., 1983). This information is not readily transferable to Saskatchewan, as growing conditions are markedly different.

Effective disease control for any plant disease is based on epidemiological principles (Berger, 1977). Three control strategies can be applied to minimize losses:

1. reduction of initial inoculum,
2. reduction of rate of disease progress and
3. reduction of the period the crop is exposed to the pathogen.

Initial inoculum is reduced through sanitation or rotation. The rate of disease progress may be reduced through the removal of diseased plants, the use of fungicides or by growing resistant cultivars. The period that the crop is exposed to the pathogen may be reduced by adjusting the seeding date or by using early-maturing cultivars. The effectiveness of these control measures depends on the disease in question. Reduction of initial inoculum is most effective against simple interest diseases (sensu Van der Plank, 1963). Reduction of the rate of disease progress is most effective against compound interest diseases.

The major objectives of this study were (1) the investigation of the epidemiology of spot-type net blotch in Saskatchewan and (2) the determination of the effect of spot-type net blotch on barley production.

2. LITERATURE REVIEW

2.1. Pathosystem

2.1.1. Host

2.1.1.1. Morphology

A barley plant is made up of several culms. Each culm is comprised of four organs: roots, stem, leaves and the spike (Reid, 1985; Reid and Wiebe, 1979). The roots are fibrous and can be classified as primary or secondary roots. The primary roots arise from the coleorhiza and the secondary roots from the crown. The stem is cylindrical with hollow internodes and usually has seven solid nodes. Leaves are borne alternately on opposite sides of the stem, arising at each node. Each leaf consists of a sheath, a ligule, a pair of auricles and a blade. The size of the leaf blade increases from the first to the penultimate leaf. The blade of the flag leaf is smaller than that of the penultimate leaf. The spike consists of spikelets, three of which are attached at each node of a flat zigzag rachis. Each spikelet consists of a single floret. In six-row barley all three spikelets produce a fertile floret, whereas in two-row barley only the central spikelet produces a fertile floret. In Western Canada all cultivars have awned lemmas (Saskatchewan Agriculture, 1986b).

2.1.1.2. Agronomy

Barley is used for both feed and malting. Two-row cultivars generally produce larger seeds than six-row cultivars and are preferred for feed purposes (Newman and McQuire, 1985). A large premium is paid for malting barley and in recent years has been higher for two-row than for six-row malting barley. Therefore, farmers usually prefer to grow two-row barley over six-row barley.

In Saskatchewan, more than 75% of the barley acreage is seeded in the northern part of the grainbelt (Saskatchewan Agriculture, 1986a). Common rotations in this area are canola-barley-wheat-wheat and barley-barley-wheat-wheat (Saskatchewan Agriculture, 1984). However, some farmers grow barley on a continuous basis to avoid admixtures caused by volunteer plants.

In Saskatchewan, soil tillage for barley production is often minimal. In the preceding fall, herbicides and fertilizer are incorporated into the soil. At seeding time, the soil is worked lightly with a cultivator. During the growing season chemical weed control is generally used. At harvest barley is swathed and combined. As part of this operation, the straw is chopped and spread evenly over the soil surface.

2.1.2. Pathogen

2.1.2.1. Morphology

Pyrenophora teres Drechsler (anamorph: Drechslera teres (Sacc.) Shoemaker; synonym: Helminthosporium teres Saccardo) is the causal agent of net blotch in barley. The anamorph is observed more frequently than the teleomorph, both in culture and in nature. The anamorph has been documented by Chidambaram et al. (1973), Ellis and Waller (1973) and Shoemaker (1962). Conidiophores are solitary, erect and pale to mid brown. Conidia are straight, cylindrical, rounded at the ends, subhyaline, smooth and have 0 to 10 (commonly 4 to 6) transverse septa. According to Shipton et al. (1973), the size of the conidia is variable. The minimum length ranges from 15 to 100 µm and the maximum length from 80 to 300 µm.

The teleomorph has been documented by Smedegaard-Petersen (1972) and Webster (1951). Mature pseudothecia are globose to elongated, measuring 300 - 700 x 200 - 400 µm and are covered with dark septate setae. Prior to maturation one to four beaks develop. The asci contain three to eight ascospores. The ascospores measure 34 - 67 x 13 - 26 µm and have 2 - 5 transverse and 0 - 3 longitudinal septa (Shipton et al., 1973). Under moist conditions the pseudothecia are densely covered with conidia on short conidiophores (Smedegaard-Petersen, 1972).

2.1.2.2. Taxonomy

Shoemaker (1962) published a key for the identification of species within the genus Drechslera, based on conidiophore morphology (width) and conidium morphology (ratio of the width of the apical septum to the width of the basal septum, formation of secondary conidiophores, conidium shape and conidium colour). He also considered the symptomatology, host range and characteristics of cultures on sucrose proline agar (formation of protothecia, pigmentation and formation of mycelial tufts).

In 1971, Smedegaard-Petersen described a new form of P. teres that causes spot-type symptoms on barley. He named this form P. teres forma maculata and named the form causing net-type symptoms on barley P. teres f. teres. Smedegaard-Petersen (1971) reported that the anamorph of P. teres f. maculata is identical to the anamorph of P. japonica Ito and Kuribayashi. Kenneth (1962) concluded that there are no morphological differences between the anamorph of P. teres f. teres and P. japonica. He accepted P. japonica as a separate species because it produces large, non-netted blotches on Japanese hull-less barley. McDonald (1967) considered P. japonica as a strain of P. teres. Shoemaker (1962) reported that D. tuberosa (Atk.) Shoemaker is identical to the anamorph of P. japonica. D. tuberosa causes a leaf spotting disease on a number of grasses including the genus Hordeum.

To resolve some of the taxonomic confusion, Smedegaard-

Petersen (1977) attempted to intermate P. teres f. teres, P. teres f. maculata and P. graminea (Died.) E. Mueller, the causal agent of leaf stripe on barley. In laboratory experiments isolates from all three groups intermated readily and produced viable and segregating progeny. Smedegaard-Petersen concluded that spot-type symptoms, net-type symptoms and leaf stripe symptoms are conditioned by three independent loci. He speculated that hybridization also occurs under natural conditions. If so, P. teres and P. graminea should be considered as forms of one biological species and a taxonomic review of the species within the genus Pyrenophora will be inevitable.

At present, classification of species in the genus Pyrenophora is based solely on their morphology. This method of classification can result in misclassification of the causal agents, as described by Drechsler (1923) for P. teres and P. graminea. A classification based on mating compatibility, morphology, host range and symptomatology would differentiate the causal agents, as well as the diseases. Differentiation among diseases at the level of forma and forma specialis would allow plant pathologists to determine the prevalence and importance of each disease, yet preserve the relationship of the causal agents (Kenneth, 1962; Shoemaker, 1981).

2.1.2.3. Symptomatology

The symptoms caused by P. teres are variable and depend on the host genotype and the pathogen strain (Barrault et al., 1982; Smedegaard-Petersen, 1971). Symptoms usually appear within two days of infection. Brown, pin point lesions develop on the leaves and sheaths. On resistant cultivars the lesions remain small and show little or no surrounding chlorosis (Keeling and Banttari, 1975; Tekauz, 1985). On susceptible cultivars the lesions increase in size and become surrounded by a yellow chlorotic margin (Smedegaard-Petersen, 1971; Tekauz, 1985). Pin point lesions can develop into one of two distinct type of lesions, depending on the isolate. With net-type isolates, the lesions increase in size and form narrow, dull brown, longitudinal and transverse streaks in a net-like pattern. With spot-type isolates, the lesions increase in size and form dark-brown, elliptical or fusiform lesions. These net-type and spot-type lesions are the extremes. Intermediate lesions can also be found (Barrault et al., 1982). The spot-type symptoms closely resemble the symptoms caused by Cochliobolus sativus (causal agent of spot blotch on barley and wheat). The spot-type symptoms caused by P. teres tend to be more regularly elliptical and have a slightly different shade of brown from the symptoms caused by C. sativus (Smedegaard-Petersen, 1971). Still, it is often necessary to confirm the diagnosis by microscopic examination of the conidia, which are readily distinguishable.

The chlorosis surrounding the necrotic lesions caused by

P. teres can expand until the entire leaf blade is affected, causing it to wither. Chlorosis is an important part of the disease syndrome (Tekauz, 1985). Disease severity is more dependent on the amount of chlorosis and withering than on the amount and size of the brown, necrotic area (Smedegaard-Petersen, 1971).

2.1.3. Distribution, prevalence and importance

Net blotch is a common disease of barley, occurring wherever barley is grown (Commonwealth Mycological Institute, 1977). However, little is known about its prevalence or importance in most countries. In Great Britain, New Zealand and Canada extensive surveys have been conducted.

In south-west Great Britain, three counties were surveyed at Feekes growth stage 11.1 and net blotch was found in 29, 90 and 78% of the fields in 1967, 1968 and 1969, respectively (Melville and Lanham, 1972). Disease severity on the two uppermost leaves ranged from 0.03 to 5.50% on a county basis. In these counties, mildew, scald and brown rust were more prevalent and more severe than net blotch.

In the Wairarapa district of New Zealand, disease surveys were conducted at Feekes growth stage 10 and net blotch was found in 63, 83 and 96% of the fields in 1973, 1975 and 1976, respectively (Matthews and Hampton, 1977; Sheridan, 1977). Disease severity on the two uppermost leaves was as high as 15% in a single field. The regional averages

were 0.3 and 3.6% in 1975 and 1976, respectively. In these surveys, net blotch was the most prevalent disease. Spot blotch and scald were less prevalent and occurred at lower levels of disease severity.

In the Manawatu-Wanganui district of New Zealand, disease surveys were conducted at Feekes growth stage 11.1 and net blotch was found in 52, 100 and 56% of the fields in 1982, 1983 and 1984, respectively (Sheridan and Grbavac, 1985). Disease severity was assessed on the two uppermost leaves. The regional averages were 7.9, 12.6 and 0.5% in 1982, 1983 and 1984, respectively.

In the Canadian Prairie provinces, net blotch was found in 27 and 76% of the fields surveyed in 1974 and 1976, respectively (Tekauz, 1978; Tekauz and Buchannon, 1977). Net-type isolates were found in 92 and 79% of the infected fields in 1974 and 1976, respectively. The frequency of spot-type isolates increased from 8 to 21% between 1974 and 1976.

2.1.4. Disease cycle

The disease cycle of *P. teres* on barley has been described by Jordan (1981), Khan (1968) and Mauler (1983), and is summarized in Figure 2.1.1. Primary infection occurs either via seed borne mycelium or as a result of the dispersal of conidia or ascospores from infected crop debris. Secondary infection is due to the dispersal of conidia produced on infected leaves. Lesions develop mainly on the leaf blades and sheaths, but also on the floral bracts and

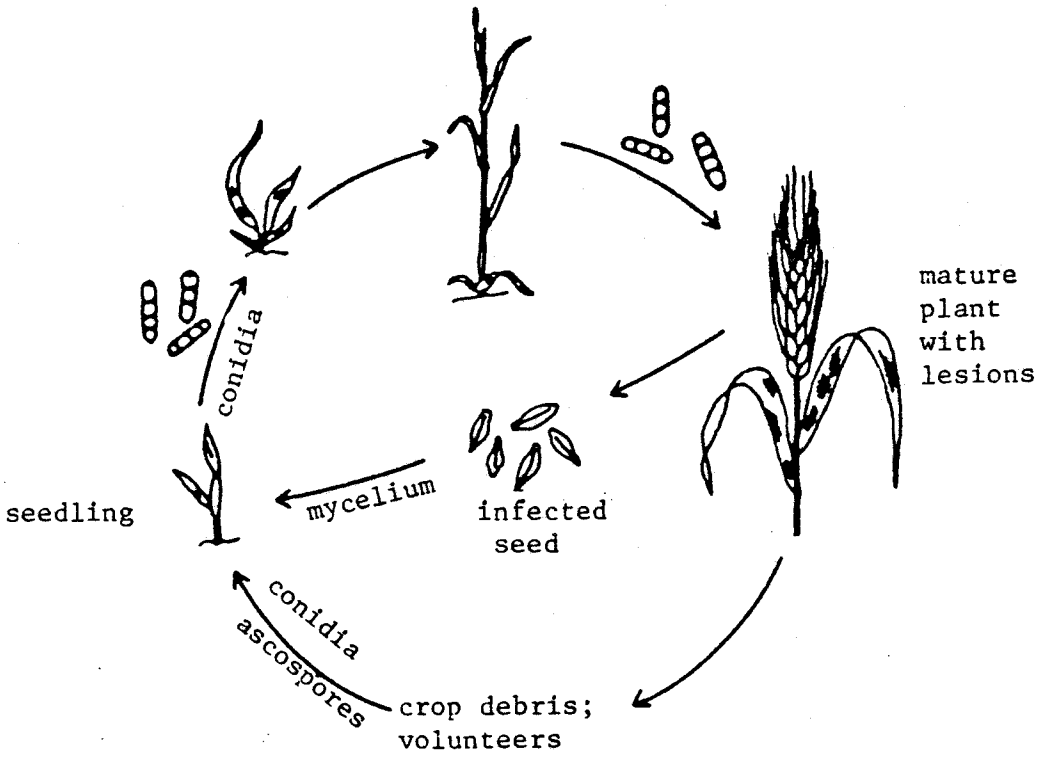

Figure 2.1.1. Disease cycle of Pyrenophora teres on barley (Adapted from Mauler, 1983. p. 28).

awns. Infection of the floral bracts leads to seed infection. Infected seed and infected crop debris allow pathogen survival and can initiate a new disease cycle. Due to the confusion surrounding the taxonomy of the pathogen, it is impossible to assess the importance of wild hosts in the epidemiology of net blotch on barley.

The presence of viable mycelium on the seed has been reported in Canada (Greaney and Machacek, 1946), the United States (Singh, 1962), New Zealand (Sheridan, 1977) and Great Britain (Hewett, 1975). The pathogen survives on the seed surface or beneath the pericarp (Singh and Chand, 1985). Jørgensen (1980) showed that seed infection can lead to infected seedlings in the field. Sheridan and Grbavac (1983) reported that seed treatment can control the spread of net blotch due to seed infection. Da Luz (1982) found that seed infection has no effect on seed germination.

The significance of stubble and plant debris as a source of primary inoculum has been reported by Jordan (1981), Piening (1968) and Singh (1962). Straw burning has been suggested as a means of field sanitation for disease control (Jordan and Allen, 1984). Mauler (1983) pointed out the importance of volunteer barley and the year-round presence of barley in the spread of the pathogen in West Germany. Jordan (1981) and Smedegaard-Petersen (1972) found pseudothecia on infected plant debris and suggested that ascospores are an important source of primary inoculum in the spring, even though most pseudothecia did not produce any ascospores.

Jordan and Allen (1984) reported that large numbers of conidia are released from infected stubble.

Secondary infection is the result of the dispersal of conidia from infected leaves. Only infected, senesced leaves produce conidia to infect later developing leaves (Jordan, 1981). The conditions for sporulation have not been investigated for P. teres. A related fungus, Setosphaeria turcica, causal agent of northern leaf blight on corn, sporulates only when the relative humidity is close to 100% and wind speed is below 0.3 m/s (Leach, 1985). Conidia of S. turcica are released violently at low relative humidity (15%) with high light intensity (Meredith, 1965). Conidia of S. turcica and P. teres can be released by air currents and water droplets (Kenneth, 1964). The release of conidia from P. teres follows a diurnal pattern with maximum release at 12:00 hours from dry leaves (Martin and Clough, 1984). Conidia cause infection at temperatures between 8 and 33 ^{o}C (Singh, 1963a) and at leaf wetness periods longer than three hours (Shaw, 1986; Singh, 1963b).

2.2. Measurement and analysis of disease progress

2.2.1. Measurement

Two parameters are used for the measurement of disease on plants: incidence and severity (or intensity). Disease incidence can be defined as the number of units infected, such as whole plants, leaves, heads or seeds. Disease severity can be defined as the area or volume of plant tissue

that is infected. For net blotch, disease incidence is commonly used to measure seed infection (Hewett, 1975), while disease severity is commonly used to measure foliar infection (Martin, 1985; Shaw and Royle, 1987). Disease severity is commonly assessed visually. Various scales have been used for assessing net blotch severity. Commonly used scales are standard area diagrams (Amelung, 1985; Hampton and Arnst, 1978) and the Horsfall-Barratt scale (Martin, 1985; Sutton and Steele, 1983).

Standard area diagrams must be developed prior to the experiment and can include as many classes as desired. This method provides an objective means to assess disease severity (James, 1971). An illustration of these diagrams developed for net-type symptoms is given in Figure 2.2.1. The disease severity of the sampled leaf is determined by assessing the amount of disease on the leaf in relation to the diagrams. The process of comparing the sampled leaves to the diagrams is time consuming (James, 1974).

Horsfall and Barratt (discussed in Horsfall and Cowling, 1978) suggested the use of a logarithmic scale for the assessment of disease severity. The use of a logarithmic scale is based on the Weber-Fechner law in physics, which states that visual precision is proportional to the logarithm of the intensity of the stimulus. The Horsfall-Barratt scale has 12 classes:

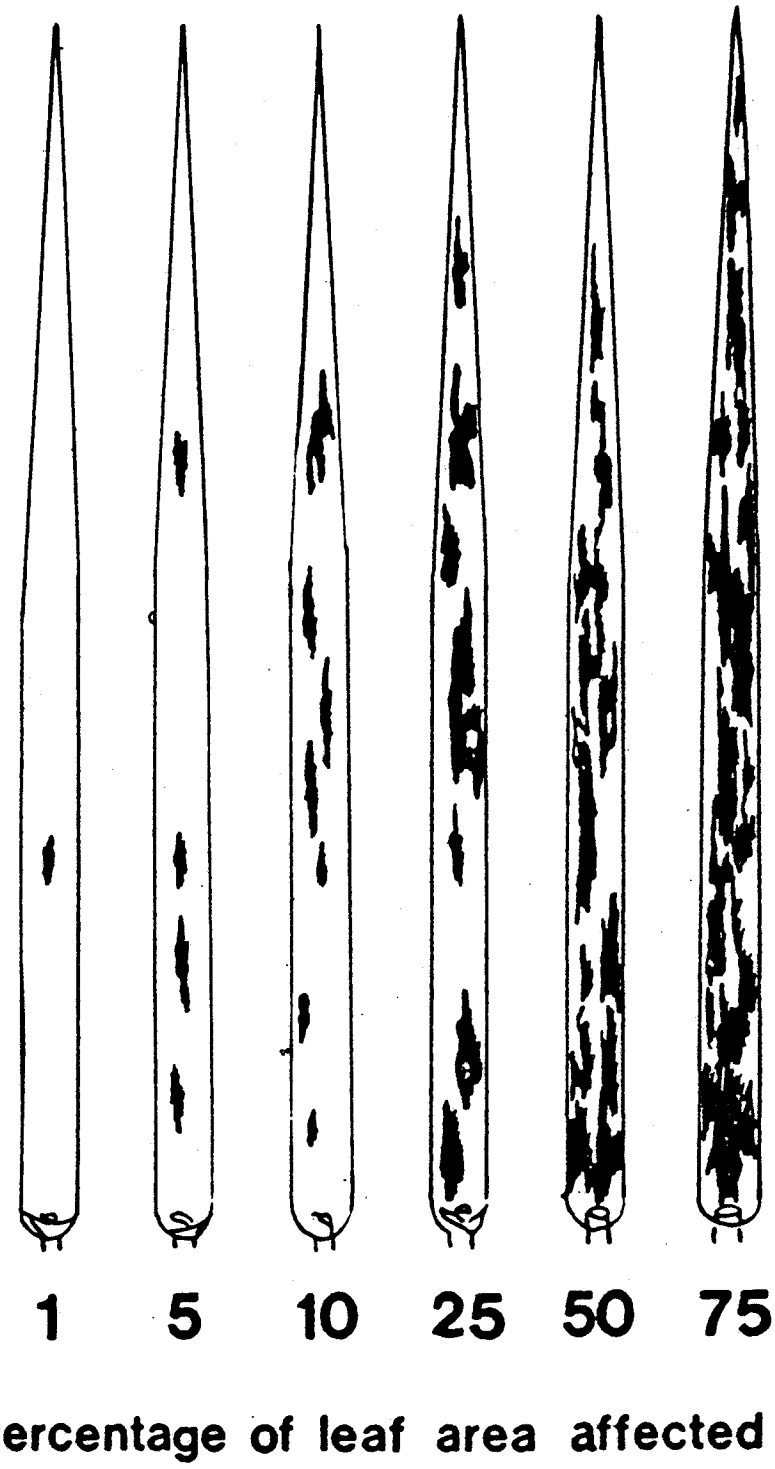

Figure 2.2.1. Standard area diagrams for the assessment of severity of net-type symptoms of Pyrenophora teres on barley (Source Hampton and Arnst, 1978. p. 18-4).

0 = 0% diseased leaf tissue

1 = 0 - 3% diseased leaf tissue

2 = 3 - 6% diseased leaf tissue

3 = 6 - 12% diseased leaf tissue

4 = 12 - 25% diseased leaf tissue

5 = 25 - 50% diseased leaf tissue

6 = 50 - 75% diseased leaf tissue

7 = 75 - 88% diseased leaf tissue

8 = 88 - 94% diseased leaf tissue

9 = 94 - 97% diseased leaf tissue

10 = 97 - 100% diseased leaf tissue

11 = 100% diseased leaf tissue.

The scale is symmetrical around 50% with the percentage diseased tissue assessed below 50% infection and the percentage healthy tissue assessed above 50% infection. The Horsfall-Barratt scale allows a rapid assessment of disease severity.

2.2.2. Analysis

Disease severity data can be analysed in two ways, depending on the purpose of the study. If the purpose of the study is to determine the effect of various treatments on disease severity, then the analysis of variance can be used to test for differences in disease severity among treatments for one observation day. If the purpose of the study is to investigate disease progress, then disease severity must be plotted against time. The curve obtained is called the

disease progress curve. The shape of the disease progress curve is frequently investigated with linear regression analysis.

Standard area diagrams provide values expressed in percentages. The analysis of variance can be performed on the data only if the range of observed values is less than 20%. If the range of observed values is larger than 20%, then heterogeneous error variances can be expected (Steel and Torrie, 1980). To obtain homogeneous error variances, a square root or arcsine transformation is recommended (Steel and Torrie, 1980). The Horsfall-Barratt scale is a pretransformed scale, therefore, the analysis of variance can be performed on the observed values (Little and Hills, 1978).

For linear regression analysis, the disease progress curve must be linear. Most disease progress curves obtained with standard area diagrams are not linear and thus require a transformation for linearization. The functions most frequently used to linearize non-linear curves, are the monomolecular (= multiple infection) function and the logistic function. The general shape for linear, monomolecular and logistic function are presented in Figure 2.2.2. The general formula for the monomolecular function (Park and Lim, 1985) is

$$X = A * (1 - e^{-(B+Ct)}) \qquad (2.1)$$

and the general formula for the logistic function (Kiyosawa, 1972) is

$$X = A / (1 + e^{-(B+Ct)}) \qquad (2.2)$$

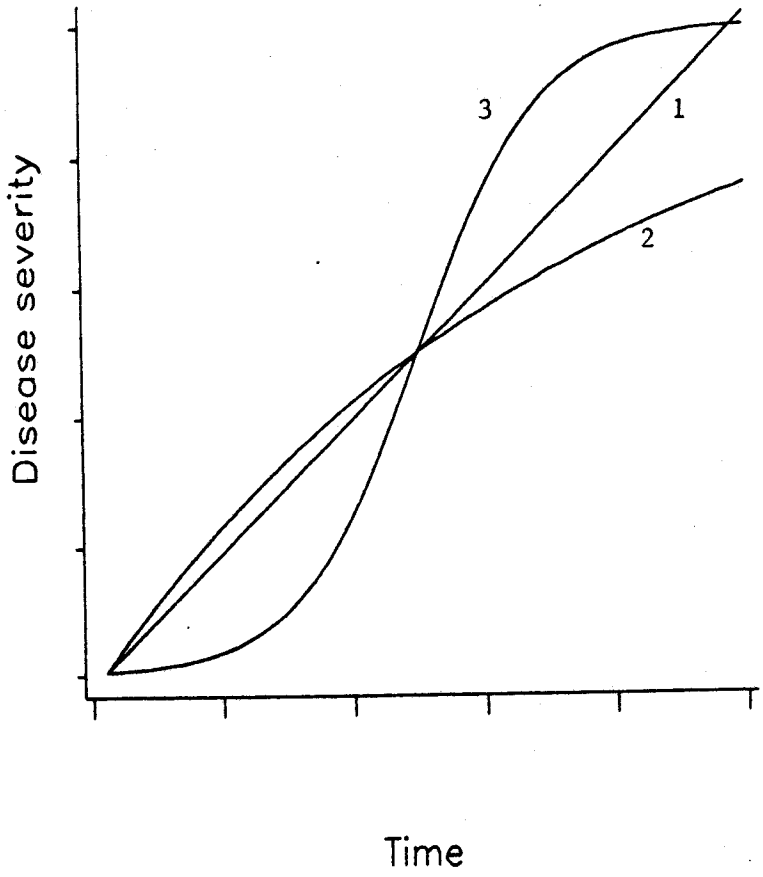

Figure 2.2.2. Disease progress curves: 1 = linear, 2 = monomolecular, 3 = logistic (Source Hau and Kranz, 1977. p. 62).

where A represents the asymptote, B the location parameter, C the rate parameter, X the disease severity and t the time. In epidemiological experiments, the asymptote is generally set at 1 or 100%, i.e. the entire leaf area is affected. In that case, the transformation formula for the monomolecular function is ln(1/(1-X)) and for the logistic function ln(X/(1-X)). The transformation used for a particular curve depends on the shape of the curve, as any transformation can linearize only curves with the appropriate shape (Hau and Kranz, 1977). After transformation, the rate parameter can be estimated as the slope and the location parameter as the intercept of the resulting linear curve. The rate parameter is also called the apparent infection rate (Van der Plank, 1963; Zadoks and Schein, 1979). It is always mentioned in the literature, whereas the location parameter is never mentioned.

The Horsfall-Barratt scale is a pretransformed scale, intended for a symmetrical, sigmoid curve (Horsfall and Cowling, 1978). If disease progress fits this type of curve, then linear regression analysis can be performed on the obtained (transformed) data. If the disease progress curve is not symmetrically sigmoid, then it is necessary to convert the obtained (transformed) data into percentages before further analysis can be done.

Linear regression analysis in combination with transformation can only be used for the analysis of regular disease progress curves. However, irregular disease progress curves are frequently observed. To analyse this type of

disease progress curves, the apparent infection rate for the period between two consecutive observation days or the area under the disease progress curve may be considered.

For the comparison of epidemics, the estimated parameters can be compared. Available methods for this comparison are: paired t-test, analysis of variance and multiple analysis of variance (Madden, 1986).

Van der Plank (1963) related the monomolecular function with simple interest epidemics and the logistic function with compound interest epidemics. This relationship holds only when the disease severity is directly related to the number of infection sites (Van der Plank, 1963). For leaf spotting diseases, such as net blotch, lesions expand until the entire leaf is affected and lesion growth has a large influence on disease severity (Berger, 1977; Tekauz, 1985) Therefore, observed disease severity is not directly related to the number of infection sites and disease progress curves should not be interpreted in terms of epidemic type (Huisman, 1982).

2.2.3. Effect of fungicides on disease progress

Fungicides can be applied as seed treatment or as foliar spray. Seed treatment is recommended when the causal agent is either seed or soil borne. The effect of seed treatment on leaf spotting diseases is characterized by a reduction in the number of infested seeds (Da Luz, 1982) and a reduction in the rate of disease progress (Martin, 1985). Non-systemic fungicides decrease the number of infested seeds, whereas

systemic fungicides decrease the number of infected seeds and the apparent infection rate during the effective period of the fungicide (Evans, 1977). Berger (1977) suggested that seed treatment provides effective disease control, when the number of infected seeds is reduced to a level so low that subsequent disease progress will not result in an appreciable crop loss.

The application of foliar fungicides decreases the apparent infection rate of epidemics caused by leaf spotting diseases. Following application, a substantial increase in disease severity can still be observed, due to enlargement of existing lesions, appearance of latent infections and incomplete coverage with the fungicide (Berger, 1977). Once the fungicide, either systemic or non-systemic, has lost its effectiveness, the apparent infection rate increases in the treated plots and can exceed the rate observed in untreated plots (Jeger, 1982; Sutton and Steele, 1983). This rapid disease progress can greatly reduce the beneficial effect of the fungicidal treatment (Berger, 1977). Berger (1977) recommended the crop be sprayed regularly until a subsequent increase in disease severity would no longer affect yield or quality.

2.2.4. Progress of net blotch

Numerous articles can be found on the effect of foliar fungicides or seed treatment on grain yield and net blotch severity. Observations of disease severity are usually taken

once or twice on a single leaf layer (Amelung, 1985; Jordan and Stinchcombe, 1986). In such studies, little attention has been given to the progress of epidemics. In the following, attempts to study the progress of net blotch are reviewed.

Burleigh and Loubane (1984) followed the progress of net blotch using infected stubble as the source of primary inoculum. Plots of three different sizes were used in one year. Disease severity was assessed with standard area diagrams on ten culms per plot. Three to six observations were made over a three month period. Results showed that the untransformed curves were quadratic to sigmoid. Asymptote values were between 50 and 90%. Apparent infection rates ranged from 0.065 to 0.135 units per day.

Sutton and Steele (1983) studied the progress of net blotch with infected seed as the source of primary inoculum at two sites in one year. Observations were made on 30 penultimate leaves per plot for four days. Disease severity was assessed according to the Horsfall-Barratt scale. They found that disease progress was erratic. Apparent infection rates were calculated between two consecutive observation days. The observed values ranged from 0.032 to 0.357 units per day for the untreated plots. The use of both foliar fungicides and seed treatment showed some degree of disease control. Initially, disease severity and apparent infection rate in the treated plots were lower than those in the untreated plots. However, later in the season, the fungicides lost their effectiveness and the apparent infection rate was

higher for treated plots than for untreated plots.

Martin (1985) studied the progress of net blotch with infected seed as the source of primary inoculum over three years. Disease severity was assessed on ten culms per plot, using the Horsfall-Barratt scale. Three to six observations were made on the penultimate, third and fourth leaf. Disease progress was erratic and the apparent infection rate between two consecutive observation days ranged from 0.125 to 1.156 units per day for untreated plots. Apparent infection rates were also calculated for the entire observation period. They ranged from 0.383 to 0.445 units per day for untreated plots. The apparent infection rate was higher for treated plots than for untreated plots. The use of seed treatment delayed the progress of net blotch.

2.3. Effect of disease on plant production

2.3.1. Measurement of the effect on grain yield

Extensive terminology has been developed to describe the effect of disease on grain yield (Chiarappa, 1981; Zadoks and Schein, 1979). The effect of disease can be described with three yield levels: attainable yield, actual yield and minimal yield (Figure 2.3.1). Attainable yield is the yield level obtained from a disease-free crop grown with current crop management (James and Teng, 1979). Actual yield is the yield level obtained from a diseased crop, using current crop management (Zadoks and Schein, 1979). Minimal yield is the yield level obtained from a severely infected crop grown in

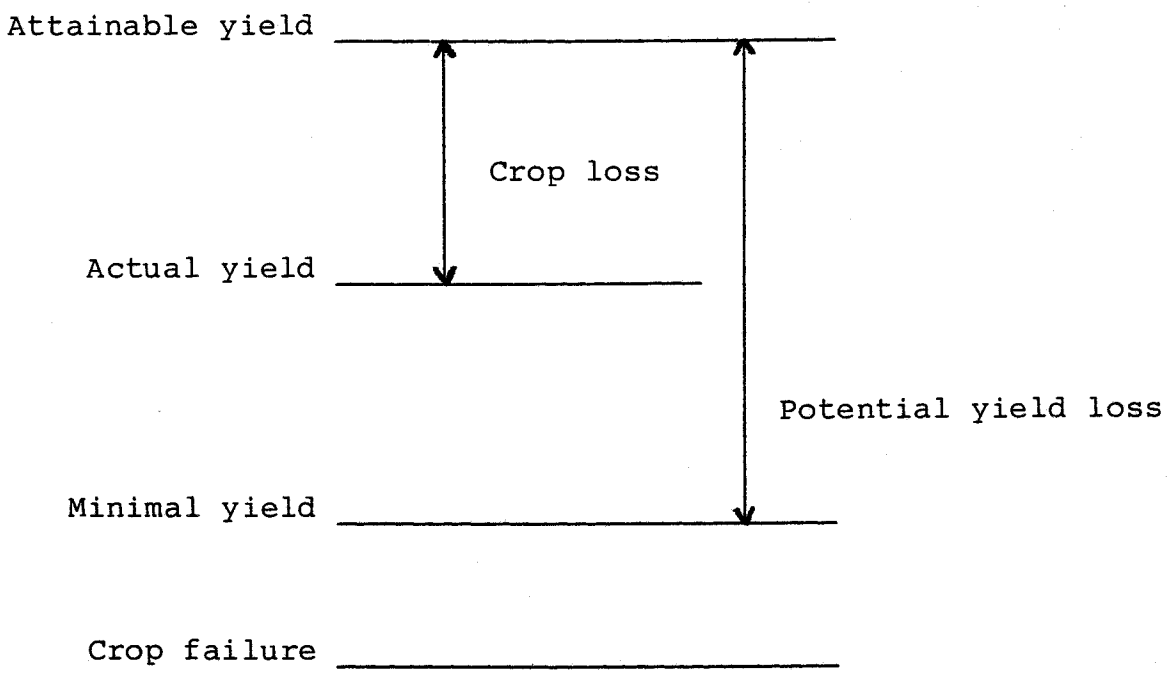

Figure 2.3.1. Yield levels and associated yield losses (Adapted from Zadoks and Schein, 1979. p. 245).

the absence of any disease control measures. Crop failure represents the lowest value for the minimal yield. The difference between attainable and actual yield is called crop loss. The difference between attainable yield and minimal yield is called potential yield loss.

Given these definitions, experiments must be properly designed to estimate defined yield losses. To obtain these estimates treatments must be established with different disease pressure. Disease pressure is generally expressed as disease severity at a specific growth stage or as a disease progress curve. In most experiments, a large number of treatments is included. For each treatment, disease severity at one or more growth stages and the associated crop loss are measured. Obtained data are generally analysed with linear regression analysis for the determination of the relationship between crop loss and disease severity, as discussed in Section 2.4. Relationships are then used for forecasting or region-wide crop loss estimates (James and Teng, 1979). Typically, three types of field experiments are employed to estimate the effect of disease on grain yield: fungicide experiments, cultivar experiments and experiments with isogenic lines (Van der Graaff, 1981; James, 1974).

In fungicide experiments, differences among treatments are generated by the use of fungicides. Utilizing differences in the timing of application and the rate and efficacy of fungicides, a large number of different treatments can be obtained in a single experiment. The potential yield loss is

estimated as the difference in grain yield between the disease-free and the most infected treatment. In practice, the interpretation of the data can be complicated, due to incomplete control of the disease by the fungicide treatments. As a result, no disease-free treatment is maintained in the experiment and the yield for the disease-free treatment must be estimated (James, 1974). In addition to fungicidal effects, fungicides may possess phytotoxic or phytotonic effects. The tonic effect can be the persistence of green tissue (Cook, 1981) or the initiation of tillers (Peat and Shipp, 1981). These effects would be confounded with the effect of fungicidal control, unless independent estimates are available.

In cultivar experiments, a set of cultivars differing in resistance to the disease investigated, but with comparable yield potential under disease-free conditions is used. To estimate the effect of disease on grain yield, the cultivars are grown in a set of environments that differ in disease conduciveness. The differences in yield between the immune and the least resistant cultivar are calculated for all environments. The potential yield loss is estimated as the largest difference in yield between these cultivars grown in disease-free and disease prone environments (McDonald and Buchannon, 1964). Interpretation of the data is based on the absence of tolerance and genotype x environment interactions. If tolerance is present, then there is no unique value for the crop loss associated with certain treatments. If a

genotype x environment interaction exists and is independent of the disease conduciveness, then the estimate of the crop loss is confounded with that interaction.

In experiments with isogenic lines, a set of isogenic lines is grown in an environment conducive to disease development. Potential yield loss is estimated as the difference in yield between the immune and the least resistant line. This method is the best available for estimating the effect of disease on grain yield, however, few sets of isogenic lines have been developed for this purpose.

Concerns have been expressed about the appropriateness of current field techniques (Jenkyn, 1981). Experiments are done to obtain the absolute values of the yield differences among treatments, not just the ranking of treatments. In this regard, plot size and plot shape are critical, due to potential interplot interference (James and Shih, 1973; James et al., 1976). Interplot interference is caused by differences in the dispersal of spores between neighbouring plots. As a result, differences in both disease severity and grain yield between treatments are reduced. A solution to this problem may be the use of buffer areas between plots.

2.3.2. Measurement of the effect on primary yield components

In addition to the effect of disease on grain yield, the effect of disease on primary yield components has been studied. The primary yield components are: number of tillers, spikelets per tiller, florets per spikelet and kernel weight

(Yoshida, 1972). Grain yield is the product of these components. Any limitation to plant production influences the grain yield through one or more of its primary yield components (Gaunt, 1980; 1987).

During the growing season, plants grow and develop. Development is the passage through the sequence of phenological stages from seed to seed. The scales commonly used for measuring development in cereals are the Feekes scale (Large, 1954) and the decimal code scale (Zadoks et al., 1974). The growth stage of a crop is defined as the growth stage of its main culms. Growth is the increase in quantity, such as area of green foliage, stem length, or dry matter content.

The potential and the realization of each primary yield component are determined during a certain period of crop development (Figure 2.3.2). The number of tillers is determined during the period from emergence to anthesis. The number of spikelets per tiller is determined during the period from tillering to anthesis. For barley the maximum number of fertile florets produced per spikelet is one (Reid and Wiebe, 1979). Kernel weight is determined in the period between anthesis and ripening. As most leaf spotting diseases can be present from the seedling stage onward, they have the capability to affect the potential and/or the realization of any primary yield component (Gaunt, 1987). If the effect of disease is studied in relation to the yield components, the relationship between disease severity and grain yield will be

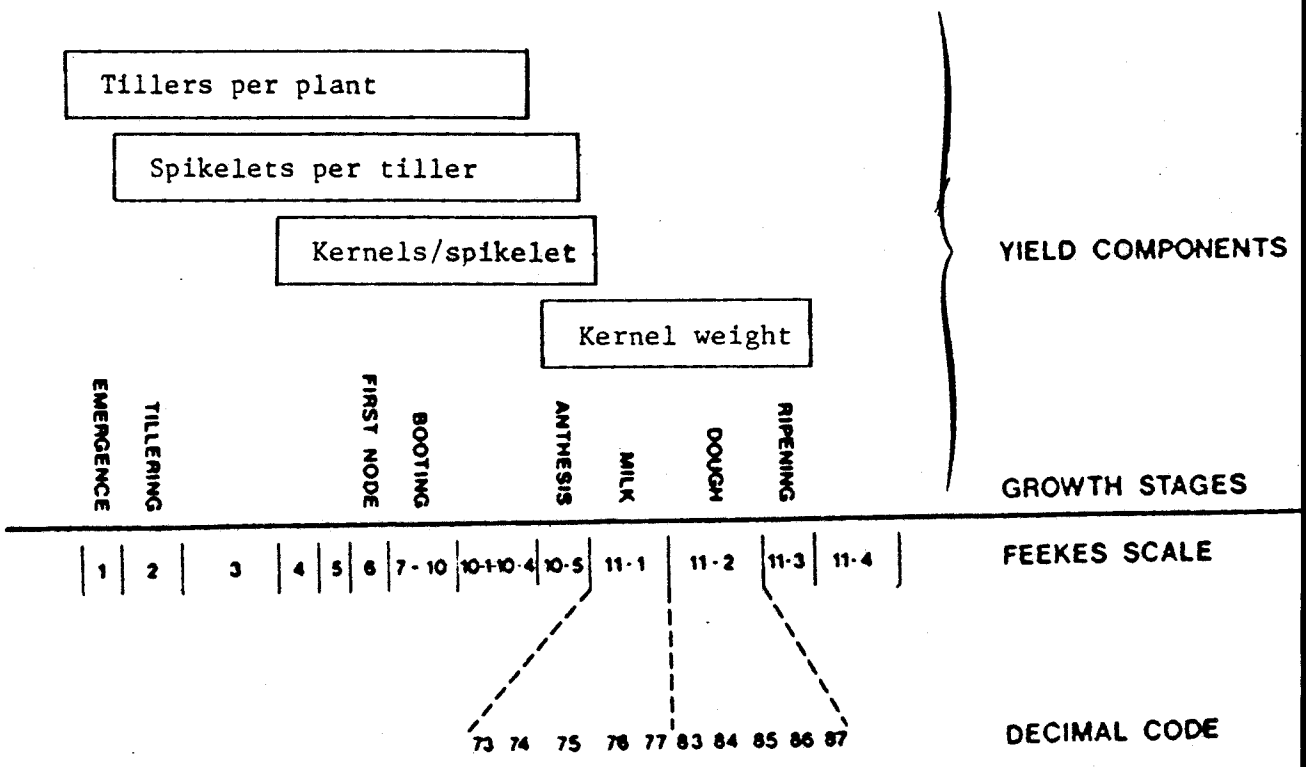

Figure 2.3.2. Yield components in cereals and their determination during crop development (Source Teng and Gaunt, 1980. p. 139).

better understood (Teng and Gaunt, 1980). The effect of disease on primary yield components can be determined with the same experiments used to determine the effect of disease on grain yield.

2.3.3. Effect of net blotch on barley production

Net blotch is seed or trash borne and can therefore be present during the entire growing season and affect all yield components. Early infection had no significant effect on the number of plants (Martin, 1985; Smedegaard-Petersen, 1974), the number of tillers (Martin, 1985; Rintelen, 1969; Sutton and Steele, 1983), or the number of seeds per spike (Neuhaus and Moritz, 1986; Sutton and Steele, 1983). The overall effect of net blotch was only reflected in kernel weight (Martin, 1985; Neuhaus and Moritz, 1986; Shipton, 1966; Sutton and Steele, 1983) and plumpness (Neuhaus and Moritz, 1986).

Several attempts have been made to estimate the effect of net blotch on grain yield (Table 2.3.1). Different treatments were generated by the use of fungicides or cultivars. Disease severity cannot be compared among these experiments, as it was assessed at different growth stages. Estimates of potential yield loss caused by net blotch ranged from 11 to 40%. In all reports, treated plots were slightly to moderately infected with net blotch. Therefore, a potential yield loss of 40% is likely a conservative estimate.

Table 2.3.1. Estimates of the potential yield loss caused by net blotch in barley field experiments.

Reference	Type[b]	Loss[c]	Disease severity[a]		
			Treated	Untreated	GS
North America					
Martin, 1985	F	18	I	I	-
Martin et al., 1981	F	40	5	46	51
McDonald and Buchannon, 1964	C	27	-	-	-
Sutton and Steele, 1983	F	16	10	41	77
Europe					
Neuhaus and Moritz, 1986	F	18	20	80	70
Smedegaard-Petersen, 1974	C+F	11	T	19	50
Australia					
Hampton and Arnst, 1978	F	30	5	40	75
Khan, 1987	F	33	I	I	-
Shipton, 1966	F	15	I	I	-

[a] GS = growth stage; I = infected; T = trace infection; - = not available.
[b] F = fungicide experiment; C = cultivar experiment.
[c] The yield for treated plots is 100%.

2.3.4. Effect of net blotch on kernel quality

Quality assessment is an area often neglected by plant pathologists (Zadoks and Schein, 1979). For barley, an extensive grading system has been established in many countries, often with substantial price differences among the grades.

In Western Canada, grades have been established for six-row and two-row barley (Agriculture Canada, 1986). Cultivars with long rachilla hairs are eligible for select (malting) and general purpose (feed) grades, whereas cultivars with short rachilla hairs are eligible for general purpose grades only. At the elevator, grain is graded based on test weight, size, foreign material and 'soundness'. Soundness includes the absence of diseased, weather-stained and discoloured kernels.

Barley is mainly used as feed. It is fed to swine, poultry and ruminants (Newman and McGuire, 1985). Although various components have been identified that are important for the feeding value, only test weight and the presence of diseased kernels are presently considered in the grading system.

The second largest use of barley is for malting. Once grain has received a select grade, it is offered to malting companies. These companies evaluate the grain for characteristics important in malting and brewing, such as percentage germination, dormancy, moisture content, protein content, microbial infection and malt extract yield (Burger

and LaBerge, 1985). The malting company will purchase the grain only when all requirements have been met.

Negative effects of net blotch on grain quality are expressed in kernel composition and malting quality. Net blotch reduced the carbohydrate content of the grain (Garg and Mandahar, 1976; Shipton, 1966). Nitrogen content did not show any significant increase (Shipton, 1966), but changes in the amino acid composition were observed (Garg and Mandahar, 1976). Also, net blotch infection reduced malt extract yield (Shipton, 1966).

2.4. Relationship between grain yield and disease severity

2.4.1. Linear regression analysis

The majority of the models describing the relationship between grain yield and disease severity are based on simple or multiple linear regression analysis (Teng, 1987). Grain yield is used as the dependent variable and disease severity as the independent variable. The general formula for the regression equation is

$$Y = a + b_1X_1 + \ldots + b_nX_n + e, \qquad (2.3)$$

where Y represents the percentage yield loss, X the n disease parameters, a the intercept, b the regression coefficient and e the error. The model is based on the following assumptions (Wetherill, 1986):

-The relationship is linear. If a non-linear relationship is observed, then transformation of the data may be necessary.

-The independent variable is not subject to error. If the independent variable is subject to error, then the method of Bartlett (1949) should be used.
-The errors are normally distributed.
-The errors are independent.
-The errors have a common variance (are homoscedastic).

As well, for proper interpretation, the data must meet the following requirements (Wetherill, 1986):

-All units of measurement must be independent. If the data have a special structure, such as replications, then the analysis of variance in combination with orthogonal contrasts is the only appropriate technique.
-Outliers must be absent. Outliers may lead to non-normality and heteroscedascity. They influence the estimates of the regression coefficients.
-Multicollinearity among the independent variables must be absent. Multicollinearity leads to spurious results, because the variance-covariance matrix of the independent variables is singular.
-The number of explanatory (independent) variables must be smaller than the number of observations for a numerical solution of the model.

The equations obtained are commonly evaluated with some of the following criteria (Teng, 1987; Teng and Gaunt, 1980):

-coefficient of (multiple) determination (r^2-statistic),
-(multiple) correlation coefficient (r-statistic),
-F-statistic for the analysis of variance (F-statistic),
-standard error of estimate of dependent variable (s-statistic) and
-significance of (partial) regression coefficient (t-statistic).

Teng and Gaunt (1980) cautioned against the evaluation of the equation using these statistics. Validation of the model should be performed with data collected independently from the data used to develop the equation. Linear regression analysis shows only the best fit model of an empirical relationship. The existence of a causal relationship cannot be assumed, but must be proven by other means (Box, 1966). Linear regression analysis is a descriptive technique, rather than an explanatory technique (Wetherill, 1986).

Linear regression analysis is the tool used to develop what are commonly known as critical point models, models using area under the disease progress curve and multiple point models. The difference among these models lies in the type and number of disease parameters used as independent variables.

2.4.2. Critical point analysis

Critical point analysis is based on the assumption that there is a single growth stage in the development of the crop, when it is particularly sensitive to the effects of

disease (Teng and Gaunt, 1980). The general formula for the yield loss equation is

$$Y = a + bX, \qquad (2.4)$$

where Y represents the percentage yield loss and X the disease severity at a specific growth stage. Disease severity can be expressed as a percentage or as a transformed value. Critical point models have been successfully applied to cereal diseases for epidemics occurring near the grain filling period. The critical time for disease observation was shown to occur approximately at the midpoint of the period of dry matter accumulation, i.e. between Feekes growth stages 10.5 and 11.3 (James, 1974).

James et al. (1968) developed a model to estimate the effect of scald (Rhynchosporium secalis) on barley. Crop loss associated with the disease severity on the two uppermost leaves was estimated with the following set of equations:

Y_1 = 2/3 * X on the flag leaf at Feekes 11.1 (2.5)

Y_2 = 1/2 * X on the penultimate leaf at Feekes 11.1 (2.6)

These estimates were combined into a single estimate of crop loss with the following equation:

$$Y = (Y_1 + Y_2) / 2 \qquad (2.7)$$

These equations have been used to estimate the crop loss using survey data (James, 1969).

Critical point models are simple and require only one disease assessment. However, they imply that both the rate of disease progress and the shape of the disease progress curve are not important for the determination of the yield loss

(James, 1974).

2.4.3. Analysis using area under the disease progress curve

Analysis using area under the disease progress curve has been developed to incorporate the shape of the disease progress curve in the yield loss model. This analysis is based on the assumption that the area under the disease progress curve can differentiate different kinds of epidemics. The area under the disease progress curve is generally calculated using rectangular integration of the disease severity over time with the following equation:

$$AUDPC = \sum_{i=2}^{n} (t_i - t_{i-1}) * (X_i + X_{i-1}) / 2 \qquad (2.8)$$

where t represents the time of observation and X the disease severity in percentage for n observation days (Shaner and Finney, 1977). The general formula for the yield loss equation is

$$Y = a + b * AUDPC, \qquad (2.9)$$

where Y represents the percentage yield loss and AUPDC the area under the disease progress curve.

Operational models using the area under the disease progress curve have not yet been developed for the estimation of yield loss. This kind of model has limited capability to distinguish between epidemics, since it is not possible to weigh disease development in relation to time or plant development (James, 1974; James and Teng, 1979). For instance, the area under the disease progress curve cannot distinguish between light early epidemics and severe late

epidemics, even though these epidemics can cause different crop losses.

2.4.4. Multiple point analysis

Multiple point analysis is based on the assumption that a combination of several disease observations can be used to estimate the yield loss. The general formula for the yield loss equation is

$$Y = a + b_1X_1 + \ldots + b_nX_n, \qquad (2.10)$$

where Y is the percentage yield loss and X represents the n disease parameters used to develop the model. Disease parameters can be in the form of actual disease readings at different observation days or disease increments between two observation days (James, 1974; Teng, 1987).

Teng et al. (1980) developed a multiple point model for the effect of leaf rust on barley. The equation included readings of the two uppermost leaves taken at four growth stages (Feekes 10.5, 10.5.2, 11.1 and 11.2) as independent variables. Models in this form can be regarded as critical point models with more than one critical point. There are some concerns about the negative sign of some partial regression coefficients, as this implies that disease at that growth stage increases grain yield (James, 1974; Gaunt, 1980).

James et al. (1972) developed a multiple point model for the effect of late blight on potatoes. The equation included disease increments from nine weekly periods as independent

variables. Partial regression coefficients allowed for the weighing of disease severity in relation to time or growth stage (James and Teng, 1979). Only positive partial regression coefficients were entered in the equation. This reflects the fact that an increase in disease severity is associated with a decreased grain yield (James and Teng, 1979).

Multiple point models allow for a description of the disease progress curve and the weighing of disease development in relation to time. The increased accuracy obtained with multiple point models comes at a considerable increase in labor and cost (James, 1974). The estimated (partial) regression coefficients can only be used for the region and cultivars used in the development of the equation. If the model is used outside these restrictions, then the (partial) regression coefficients must be adjusted (Zadoks and Schein, 1979). Critical point models may be the only models available to adequately estimate the crop loss caused by a pathogen over a large area at regular intervals.

3. MATERIALS AND METHODS

3.1. Experiment I: Conditions for infection

This experiment was conducted to determine the effects of temperature on the percentage germinated conidia and time to infection. Three isolates of Pyrenophora teres were used: WRS 858, M and S. Isolate WRS 858 causes net-type symptoms and was collected at Teulon, Manitoba in 1973 (Tekauz and Mills, 1974). Isolates M and S cause spot-type symptoms and were collected at Medstead and Shellbrook, Saskatchewan, respectively in 1985. The experiment was conducted in a Conviron model E15 growth cabinet with a CMP3023 control computer and M15 lighting. Lighting had 80% input wattage of Philips cool white fluorescent (F72T12/CW/SHO; 160 W; 1500 MA) and 20% input wattage of Philips incandescent light (60 W). Light duration was 16 h with an abrupt light-dark change. Air temperature was 20 $\pm$ 1 ^{o}C during the light period and 15 $\pm$ 1 ^{o}C during the dark period, unless stated otherwise. Air flow was 0.23 m/sec through the unifloor. Ten to 15 plants of the barley cultivar 'Elrose', which is susceptible to net blotch, were grown in 15 cm pots in a mixture of soil, vermiculite and peatmoss (2:1:1 by volume).

During the leaf wetness period, four constant and two decreasing temperature regimes were used in the dark in the growth cabinet. Temperature regimes were selected on the basis of meteorological data collected at Shellbrook, Saskatchewan in 1986, and the limits of the growth cabinet.

Constant temperature regimes were 10, 15, 20 and 25 ^{o}C. Decreasing temperature regimes were 25 decreased to 10 ^{o}C and 20 decreased to 10 ^{o}C. Temperature decreased at a constant rate for six hours. Each temperature regime was tested on a different day.

For each inoculation, conidia were obtained directly from infected leaves, which were previously inoculated with the specific isolate. To induce sporulation, infected leaves were placed in petri dishes with moist filter papers in the lid and put in a incubator (Hotpack Corp., Philadelphia, PA, USA) with cool white fluorescent light and a 12 h light-dark cycle at 19 ^{o}C. Approximately 24 h later, leaves were submersed in distilled water containing one drop of Tween 20 per 100 mL and washed for 15 sec on a wrist-action shaker. The conidium concentration was adjusted to 3,000/mL using a haemocytometer. Either third or fourth leaves were placed in a horizontal position in a humidity box (Figure 3.1.1) and about seven drops of the conidium suspension were applied with a Pasteur pipet. The humidity box was placed in the growth cabinet.

To determine percentage germinated conidia, leaves were removed hourly from the humidity box for six hours, creating six leaf wetness periods for all temperature regimes. Leaves were detached and air dried. Three to five sections (1 - 2 cm long) covered by the conidium suspension were cleared overnight in Carnoy solution (3 alcohol : 1 glacial acetic acid), as described by Shaw (1986). Cleared sections were

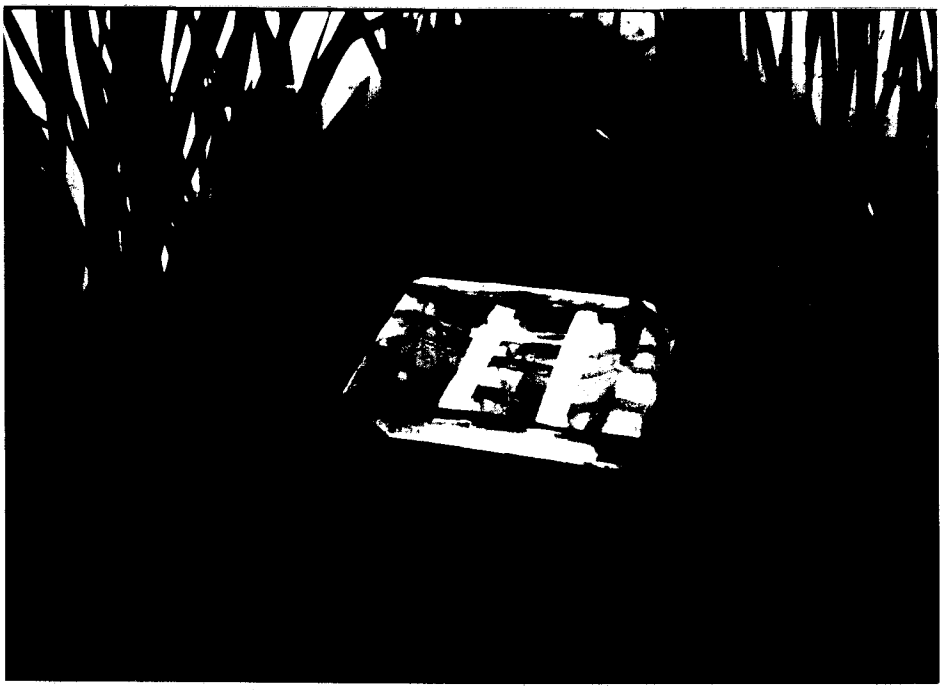

Figure 3.1.1. Humidity box with inoculated leaves (modified from Ubels, 1979).

mounted on a microscope slide in lactophenol-cotton blue. The total number of conidia deposited and the number of germinated conidia were counted at 100x magnification. A conidium was classified as germinated when a germ tube was visible. A minimum of 60 conidia was counted for each combination of temperature, leaf wetness period and isolate. Percentage germinated conidia was calculated as the number of germinated conidia divided by the total number of conidia, multiplied by 100. The experiment was conducted as a randomized complete block design with two replications in time. An analysis of variance for percentage germinated conidia was conducted for each combination of temperature and leaf wetness period (Table 3.1.1). Error variances were tested for homogeneity with the chi-square statistic developed by Bartlett (Steel and Torrie, 1980). The chi-square statistic was calculated as:

$$X^2 = \sum_{i=1}^{n} (edf) * \ln\left(\sum_{i=1}^{n} s^2/n\right) - \sum_{i=1}^{n} (edf * \ln(s^2)) \qquad (3.1)$$

with (n-1) degrees of freedom, where edf represents the error degrees of freedom, s^2 the error mean square for each combination and n the number of error variances.

To determine time to infection for each isolate, two inoculated leaves were removed hourly from the humidity box for eight hours at the constant 10 ^{o}C regime and for six hours for the other regimes, establishing eight and six leaf wetness periods, respectively. Leaves were left attached to the plant and air dried in the growth cabinet. The occurrence of visible lesions was recorded for the two leaves 7 - 10

Table 3.1.1. Analysis of variance for percentage germinated conidia for each combination of temperature and leaf wetness period.

Source	df	MS	F-test
Isolate	2	MS_1	MS_1/MS_3
Replication	1	MS_2	MS_2/MS_3
Error	2	MS_3	
Total	5		

days and 14 - 18 days after inoculation. The shortest leaf wetness period after which visible lesions developed was designated as the time to infection. The experiment was conducted as a completely randomized split-plot design with two replications in time. Temperature regimes were the whole plots; isolates were the sub-plots. For each combination of temperature and isolate two leaves were sampled. An analysis of variance for time to infection was conducted as described in Table 3.1.2.

3.2. Experiment II: Potential for disease establishment

The objective of this experiment was to investigate the relationship between developmental stages of barley, net blotch progress, sporulation on infected leaves, incidence of airborne conidia and local weather conditions. The experiment was conducted at Shellbrook in 1986 and 1987 in a plot of two-row barley cultivar 'Elrose' seeded into barley stubble infected with P. teres f. maculata. The plot was 15 x 30 m and seeded at 110 kg/ha with 15 cm row spacing. A time table of field operations for 1986 and 1987 is given in Table 3.2.1.

Net blotch severity was assessed on ten primary culms collected at 4 m intervals from the entire plot at 3 to 11 day intervals beginning June 30 in 1986 and at four to eight day intervals beginning at emergence in 1987 (Table 3.2.1). Leaves of these tillers were assessed for:

-% green = percentage of total leaf area that was green.

Table 3.1.2. Analysis of variance for time to infection.

Source	df	MS	F-test
Temperature	5	MS_1	MS_1/MS_2
Replication(Temperature)	6	MS_2	
Isolate	2	MS_3	MS_3/MS_5
Temperature x Isolate	10	MS_4	MS_4/MS_5
Error	12	MS_5	
Sampling error	36	MS_6	
Total	71		

Table 3.2.1. Time table of field operations for experiment II in 1986 and 1987.

Operation	1986	1987
Seeding	23/5	26/5
Equipment installation	2/6	26/5
Sampling		9/6
		17/6
		25/6
	30/6	30/6
		7/7
	11/7	14/7
	16/7	
	21/7	21/7
	24/7	28/7
	31/7	
	5/8	4/8
Equipment removal	11/8	11/8

-% necrotic = percentage of total leaf area that was covered with necrotic lesions.

-% chlorotic = percentage of total leaf area that was chlorotic due to disease, senescence and/or other damage.

-% affected = percentage of total leaf area that was affected by net blotch, calculated as the sum of percentage necrotic and percentage chlorotic, or as the difference between 100% and percentage green.

These traits were assessed by comparing the sampled leaves to standard area diagrams (Figures 3.2.1 and 3.2.2). Mean values were recorded for each leaf position. For each sampling date, leaf area was measured for each leaf position on ten tillers, using a Delta-T area meter (Delta-T Devices Ltd, Burwell, Cambridge, UK). Average leaf area was recorded for each leaf position. Plant development was assessed on the Zadoks scale (Zadoks et al., 1974).

The level of sporulation was determined for each leaf position at each sampling date in 1987 (Table 3.2.1). For each leaf position, ten leaves were incubated in petri dishes with moist filter paper (filter paper was wetted daily). After 1, 3 and 5 days, these leaves were submersed in 10 mL distilled water and washed for 15 sec on a wrist-action shaker. The concentration of conidia in the suspension was determined with a haemocytometer. The number of conidia produced by the leaves was calculated as the product of the

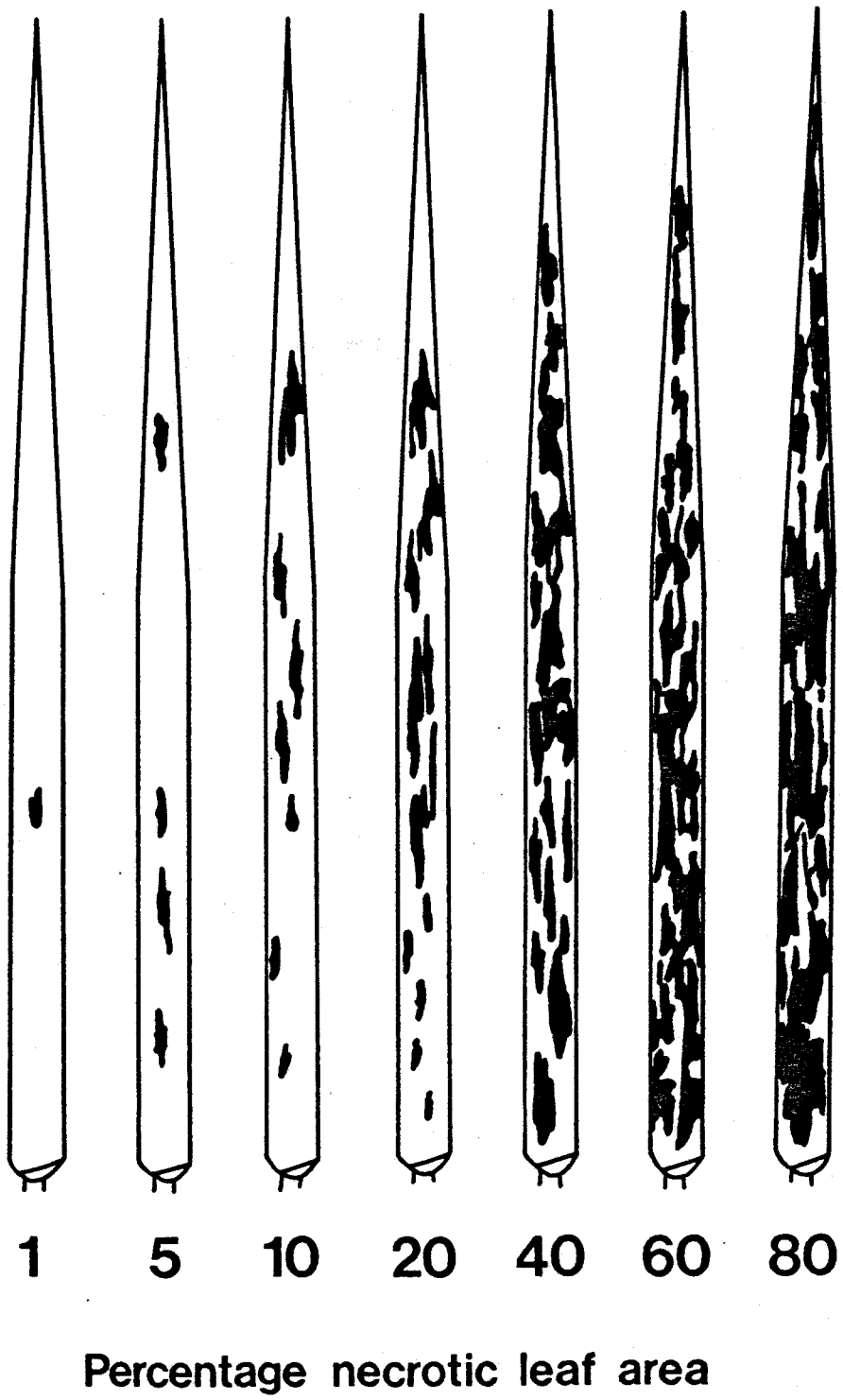

Figure 3.2.1. Standard area diagrams used to assess percentage necrotic leaf area (Adapted from Hampton and Arnst, 1978. p. 18-4).

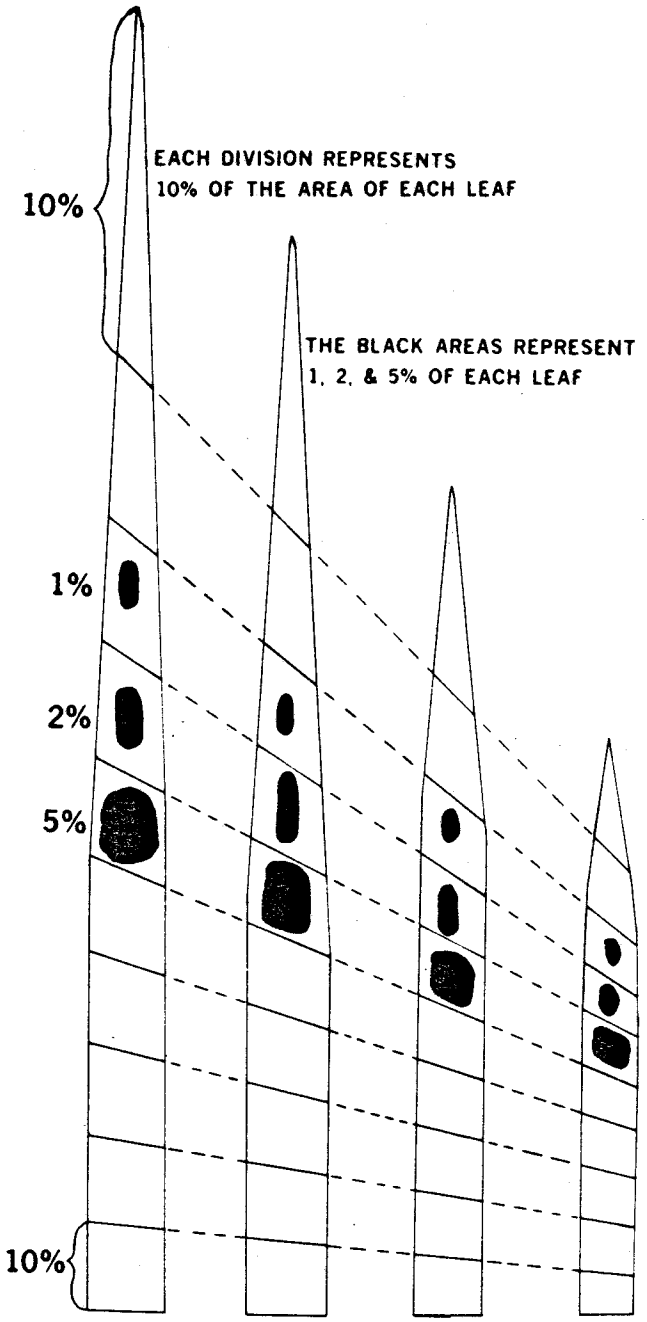

Figure 3.2.2. Standard area diagrams used to assess percentage green and chlorotic leaf area (Source James, 1971. p. 44).

concentration and the volume of the wash water. Similarly, sporulation was determined for the crop debris early in the season. Assessments were made on ten 5-cm long pieces of stem and on ten leaves.

A Burkard recording volumetric spore trap (Burkard Scientific Instruments, Rickmansworth, Herts., UK) was used to monitor the incidence of airborne conidia in 1986 and 1987 (Table 3.2.1). The spore trap was developed by Hirst (1952) and improved with a rotating drum by Kramer et al. (1976). The battery operated spore trap was placed in the plot with the air-intake orifice at 42 cm above ground level. The air intake was adjusted to sample 10 L of air per minute. Plants within 1 m of the sampler were cut regularly. To quantify the conidia, exposed tapes from the spore sampler were cut into 24 h segments and mounted on microscope slides in lactophenol-cotton blue. Conidia of P. teres trapped on the tapes were counted and quantified on an hourly basis.

Local weather conditions (microclimate) were monitored at 15 min intervals in 1986 and 1987 (Table 3.2.1). A Campbell CR21 micrologger (Campbell Scientific Canada Inc., Edmonton, Canada) was used to monitor temperature, precipitation and leaf wetness. Temperature was measured with two thermistors placed at 25 and 30 cm above ground level and shielded from direct sun light. Precipitation was measured with a tipping bucket raingauge, measuring 0.254 mm per tip (Sierra-Misco Inc. Berkeley, CA, USA). Leaf wetness was measured with two 6 x 8 cm electrical impedance grids coated

with two thin coats of latex paint (Gillespie and Kidd, 1978). One sensor was employed with a rotation of 15° along the short axis and the other with a rotation of 15° along the long axis. Both sensors were placed 50 cm above ground level with the tilted top facing north. For temperature and precipitation, collected data were summarized into monthly average minimum temperature, monthly average maximum temperature, monthly amount of precipitation and monthly frequency of precipitation. For leaf wetness, the duration was determined for each period. Leaves were considered to be wet when the sensor indicated a value above 75%.

3.3. Experiment III: Effect of fungicides on the pathosystem

In this experiment the effects of fungicide application on the progress of net blotch, grain yield and primary yield components were investigated. The data obtained were subjected to three analyses:

1. regression analysis for disease progress,
2. analysis of variance for each trait measured,
3. analysis of variance and covariance components between pairs of traits.

Each analysis is discussed separately in the Results and Discussion (Sections 4.3 to 4.5).

Experiments were conducted in 1985 and 1986 at three sites in Saskatchewan: Shellbrook, Medstead and Saskatoon. The experiments at Medstead and Shellbrook were conducted on barley stubble infected with *P. teres* f. *maculata*. The

experiments at Saskatoon were on fallow. The two-row barley cultivar 'Elrose' was grown at all sites in 1985. Elrose and the six-row barley cultivar 'Argyle' were grown at all sites in 1986. Elrose is susceptible (Tekauz, 1986) and Argyle is moderately resistant to spot-type isolates (J. Weller, pers. comm.).

At all sites, five application schedules of the fungicide Tilt (propiconazole) were employed: no application (untreated control), application at Zadoks growth stage 31 (early application), application at Zadoks growth stage 49 (late application), application at Zadoks growth stage 31 and 49 (double application) and application at 7 - 10 day intervals (repeatedly sprayed control). At Medstead the repeatedly sprayed control was omitted in 1986. Tilt 250E was supplied by Ciba-Geigy Canada Ltd. (Mississauga, Ont., Canada). It was applied at the recommended rate of 1 L in 200 L water per ha (125 g a.i./ha). A model D-201 hand sprayer (R & D Sprayers Inc., Opelousas, LA, USA) was operated at 140 kPa with a boom 1.5 m wide, fitted with six nozzles (model LF 3 nylon 80^{o}) at 30 cm spacing.

At each site the treatments were arranged in a randomized complete block design with six replications. Each experimental unit consisted of a plot 3 x 3.6 m, drill planted with 15 cm row spacing. The experimental units were bordered with 1.5 m wide plots of oat in 1985 and spring wheat in 1986 to reduce interplot interference, that could be caused by secondary spread of net blotch or fungicide drift.

The timing of field operations is shown in Table 3.3.1. Seeding was conducted with a small plot seeder. Seeding rate was 110 kg/ha. At all sites, fertilizer as 27-27-0 was placed with the seed at 112 kg/ha. At Medstead and Shellbrook fertilizer as 34-0-0 was also broadcast at 67 kg/ha at the seedling stage. Weeds were controlled with 3.5 L/ha Roundup (glyphosate) applied prior to seeding, and a tankmix of 1.0 L/ha Buctril-M (bromoxynil and MCPA) and 3.5 L/ha Avenge 200-C (difenzoquat) applied at the tillering stage. At Saskatoon, weeds were controlled with 1.0 L/ha Buctril-M applied at the tillering stage, and grasshoppers were controlled with an aerial application of 2 L/ha Sevin XLR (carbaryl) on August 4, 1985. Whole plots were harvested with a Hege small plot combine at all sites.

Severity of all diseases present was assessed on the leaves of ten main culms taken at random from each plot at each sampling date. The method of assessment was as described in Section 3.2. Mean values were recorded for each leaf position and in each plot. In 1985, disease assessment was conducted for all six replications. In 1986, disease assessment was conducted only on the first three replications at Medstead and Shellbrook and on the first two replications at Saskatoon.

In 1985, the number of tillers per metre squared was determined for each plot by counting the number of tillers in 1 m row at Zadoks growth stage 55 and multiplying by 6.67. In both 1985 and 1986, 10 spikes from primary tillers were taken

Table 3.3.1. Time table of field operations for experiment III at three sites in 1985 and 1986.

	Medstead		Shellbrook		Saskatoon	
Operation	1985	1986	1985	1986	1985	1986
Seeding	28/5	26/5	27/5	23/5	22/5	27/5
Fertilizer	13/6	10/6	3/6	2/6		
Herbicide	2/6	10/6	2/6	9/6	20/6	20/6
Sampling	20/6		20/6	30/6		
	2/7		3/7	4/7	7/7	
	10/7		10/7	11/7		
		16/7		16/7		
	20/7		20/7	21/7	21/7	
				24/7		
	29/7	31/7	29/7	31/7		
	6/8	7/8	6/8	5/8		4/8
	14/8			11/8	17/8	13/8
Fungicide						
Early	10/7	2/7	7/7	4/7	2/7	4/7
Late	20/7	16/7	20/7	16/7	22/7	14/7
Repeated	20/6	-	20/6	16/6	18/6	17/6
	2/7		4/7	23/6	27/6	27/6
	10/7		10/7	30/6	2/7	4/7
	20/7		20/7	11/7	12/7	14/7
	29/7		29/7	21/7	22/7	24/7
	6/8		6/8	31/7	1/8	4/8
				5/8		
Harvest	11/9	10/9	28/8	27/8	3/9	5/9

at random from each plot prior to harvest. Kernels from these spikes were counted and weighed to obtain mean number of kernels per spike and mean kernel weight. After harvesting, the grain was air dried, cleaned and weighed to determine grain yield. A representative sample of the harvested grain was taken from each treatment for official grading by the Inspection Division of the Canadian Grain Commission, Saskatoon.

Progress of percentage necrotic leaf area and percentage affected leaf area was analysed for the untreated control and the repeatedly sprayed control. Regular disease progress curves can be expected only for these two treatments (Berger, 1977). For Elrose, percentage necrotic leaf area and percentage affected leaf area were assessed in 1985 and 1986. The monomolecular curve was fitted to the percentage necrotic leaf area and the logistic curve to the percentage affected leaf area. For the percentage necrotic leaf area, the asymptote was estimated as the sum of the maximum observed value and 0.005. Data of percentage necrotic leaf area were transformed with the formula:

$$X' = \ln(A/(A-X)), \qquad (3.2)$$

where A represents the asymptote, X the observed value and X' the transformed value. The rate and location parameter were estimated with linear regression analysis on the transformed data. The estimated regression was:

$$X' = B + Ct, \qquad (3.3)$$

where B represents location parameter, C the rate parameter

and t the time expressed in days after seeding. For the percentage affected leaf area, the asymptote was set at 100%. Data of the percentage affected leaf area were transformed with the formula:

$$X' = \ln(X/(100-X)). \qquad (3.4)$$

The rate and location parameter were estimated with linear regression analysis on the transformed data (Equation 3.3). From the estimates of the location and rate parameter, the quotient was calculated as -B/C. An analysis of variance was performed for the estimates of the parameters (Table 3.3.2). Differences among means of each combination of year and treatment were tested for significance using the least significance difference test.

For Argyle, percentage necrotic leaf area and percentage affected leaf area were assessed only in 1986. The exponential curve was fitted to the percentage necrotic leaf area and the logistic curve to the percentage affected leaf area. Data of percentage necrotic leaf area were transformed with the formula:

$$X' = \ln(X), \qquad (3.5)$$

where X represents the observed value and X' the transformed value. The rate and location parameter were estimated with linear regression analysis on the transformed data (Equation 3.3). For the percentage affected leaf area, the asymptote was set at 100%. Data on percentage affected leaf area were transformed with Equation 3.4. The rate and location parameter were estimated with linear regression analysis on

Table 3.3.2. Analysis of variance for parameters estimated for Elrose.

Source	df	MS	F-test
Year	1	MS_1	MS_1/MS_2
Replication(Year)	7	MS_2	
Treatment	1	MS_3	MS_3/MS_5
Treatment x Year	1	MS_4	MS_4/MS_5
Error	7	MS_5	
Total	17		

the transformed data (Equation 3.3). The quotient was calculated as -B/C. An analysis of variance was performed for the estimates of these parameters (Table 3.3.3).

For each combination of site and cultivar, treatment and replication effects were determined for disease severity, grain yield, number of tillers, number of kernels per spike and kernel weight, as shown in Table 3.3.4. Four orthogonal contrasts were determined for grain yield:

C = repeatedly sprayed control minus the average of the other treatments.

E = the average of the early and double application minus the average of the late application and the untreated control.

L = the average of the late and double application minus the average of the early application and the untreated control.

ExL = the average of the untreated control and double application minus the average of the early and late application.

Error mean squares for the combination of site and cultivar were tested for homogeneity using Equation 3.1. Differences among treatment means within each combination of site and cultivar were tested for significance using Duncan's multiple range test. Analyses were performed with the procedures ANOVA, GLM and REG of the SAS statistical package (SAS Institute, 1985).

Table 3.3.3. Analysis of variance for parameters estimated for Argyle.

Source	df	MS	F-test
Treatment	1	MS_1	MS_1/MS_3
Replication	2	MS_2	MS_2/MS_3
Error	2	MS_3	
Total	5		

Table 3.3.4. Analysis of variance for traits measured for each combination of year and cultivar.

Source	df	Treatment[a] 1	2	3	4	5	Expected mean square
Treatment	4						$var(e_{ij}) + 1.5\ sum(T_i^2)$
C: Repeated vs others	1	1	1	1	1	-4	
E: Early application	1	-1	1	-1	1	0	
L: Late application	1	-1	-1	1	1	0	
ExL: Early x late interaction	1	1	-1	-1	1	0	
Replication	5						$var(e_{ij}) + 5\ var(R_j)$
Error	20						$var(e_{ij})$
Total	29						

[a] 1 = No application of fungicide; 2 = Application of Tilt at Zadoks growth stage 31; 3 = Application of Tilt at Zadoks growth stage 49; 4 = Application of Tilt at Zadoks growth stage 31 and 49; 5 = Application of Tilt at 7 to 10 day intervals.

4. RESULTS AND DISCUSSION

4.1. Conditions for infection

Epidemics are composed of infection cycles (Zadoks and Schein, 1979). A single infection cycle consists of several processes, such as infection, sporulation and dissemination. Each of these can be divided into subprocesses.

Infection can be subdivided into germination, penetration and colonization. Germination and penetration occur on the epidermis outside host tissue and can thus be affected by environmental conditions. Many conidia may germinate, but fail infect the host, thus limiting the epidemic (Shaw, 1986). The most important environmental conditions influencing infection are temperature and leaf wetness duration (Rotem, 1978). Leaf wetness may be caused by precipitation or dew formation. The effect of temperature and duration of leaf wetness on conidium germination and time to infection were investigated for three Western Canadian isolates of P. teres under controlled conditions (Experiment I).

4.1.1. Conidium germination

The percentage of conidia germinated ranged from 5 to 100. For this range of percentages, heterogeneous variances can be expected and the use of the arc sine transformation is recommended (Steel and Torrie, 1980). Individual analysis of variance, as described in Table 3.1.1, was performed for each

combination of temperature and leaf wetness period on untransformed data. Error variances obtained from these analyses were tested for homogeneity (Steel and Torrie, 1980). The chi-square test for homogeneity of variance yielded a value of 94.5. This value exceeded the 0.99 point of the chi-square distribution with 35 degrees of freedom (49.8). Individual analyses were also performed on arc sine transformed data. In this case, the obtained value for the chi-square test was 77.4, showing that the error variances remained heterogeneous after arc sine transformation. For ease of interpretation, individual analyses based on untransformed data are presented for each combination of temperature and leaf wetness period (Table 4.1.1).

With constant 10 and 15 ^{o}C regimes, some conidia germinated after one hour of leaf wetness and the standard deviation was small (Figures 4.1.1 and 4.1.2). The percentage of conidia germinated increased over the next three hours. At the same time, the standard deviation increased. Subsequently, the percentage of conidia germinated approached 100% at 6 h and the standard deviation decreased. With constant 20 and 25 ^{o}C regimes, and decreasing temperature regimes, the percentage of conidia germinated was less than 90% for the first three hours and the standard deviation was generally large (Figures 4.1.3 to 4.1.6). Subsequently, as the percentage of conidia germinated approached 100%, the standard deviation decreased.

With constant 10, 15, and 20 ^{o}C regimes, significant

Table 4.1.1. Mean squares for isolate and error effect for each combination of temperature and leaf wetness period in Experiment I.

Temperature	Incubation period	Mean square		
		Isolate	Replication	Error
°C	h			
10	1	25.2	24.0	6.00
	2	268.7*	42.7	8.67
	3	1513.2**	352.7*	15.17
	4	350.0	240.7	72.67
	5	25.2	1,204.2	186.17
	6	28.7	266.7+	16.67
15	1	157.0**	16.3*	0.64
	2	206.2	169.6	122.80
	3	1555.2*	522.7	73.17
	4	640.5	352.7	588.17
	5	155.2*	308.2**	2.17
	6	54.2	2.7	60.17
20	1	88.7+	150.0*	8.00
	2	360.7*	112.7	16.67
	3	114.0	2.7	64.67
	4	120.2**	6.0	1.50
	5	8.0	54.0+	6.00
	6	15.2	0.2	5.17
25	1	114.7	80.7	200.67
	2	516.2	20.2	155.17
	3	11.2	73.5	10.50
	4	40.7	66.7	8.67
	5	2.2	28.2*	1.67
	6	0.2	0.7	1.67
20 decreased to 10	1	1,514.0**	130.7**	0.67
	2	1,310.1*	16.7	28.17
	3	693.2	42.7	197.17
	4	546.0+	4.2	52.67
	5	433.5*	20.2	20.17
	6	126.2*	4.2	2.17
25 decreased to 10	1	714.7	28.2	384.67
	2	732.17+	4.2	43.17
	3	288.67	73.5	182.00
	4	313.2	112.7	161.17
	5	109.5	204.2+	16.17
	6	8.2	8.2	8.17

+, *, ** Significant at the 0.1, 0.05 and 0.01 probability level, respectively.

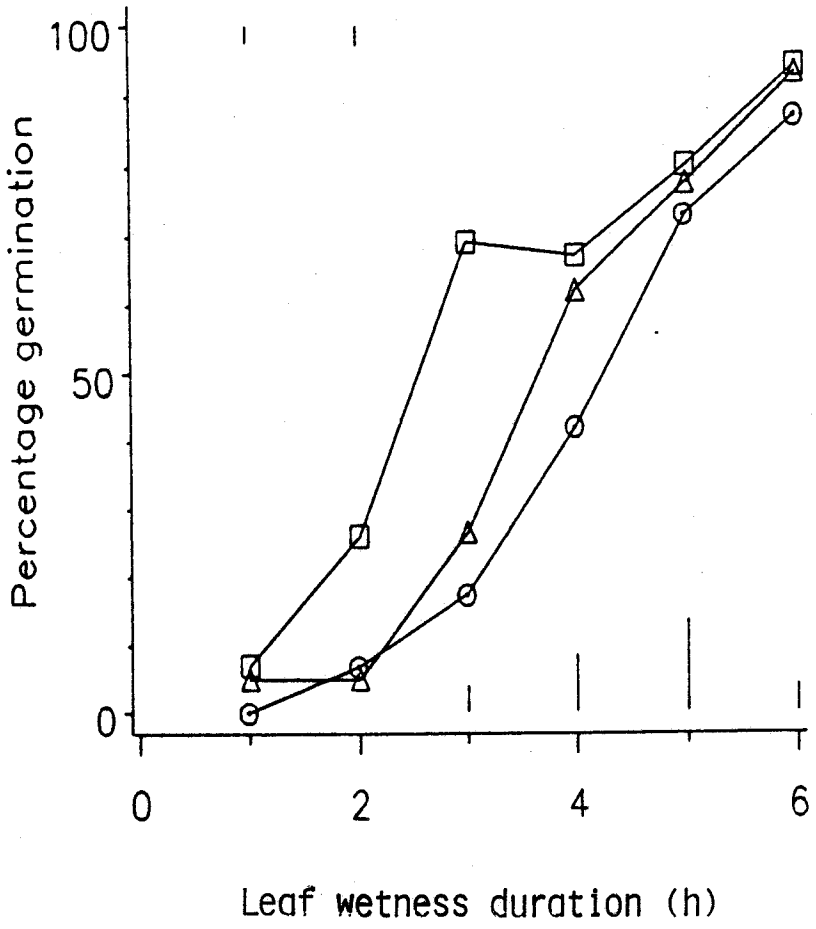

Figure 4.1.1. Time course of conidium germination for three isolates of Pyrenophora teres at constant 10 °C regime (○ = WRS 858; □ = M; △ = S). Vertical bars represent standard deviation for each incubation period.

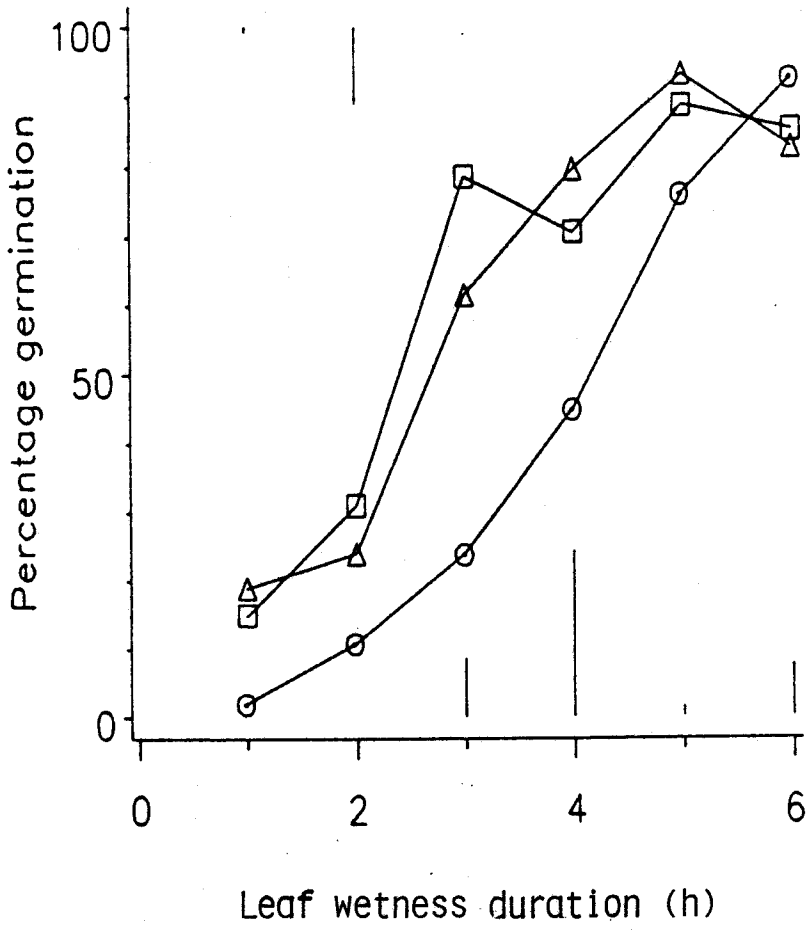

Figure 4.1.2. Time course of conidium germination for three isolates of Pyrenophora teres at constant 15 °C regime (○ = WRS 858; □ = M; △ = S). Vertical bars represent standard deviation for each incubation period.

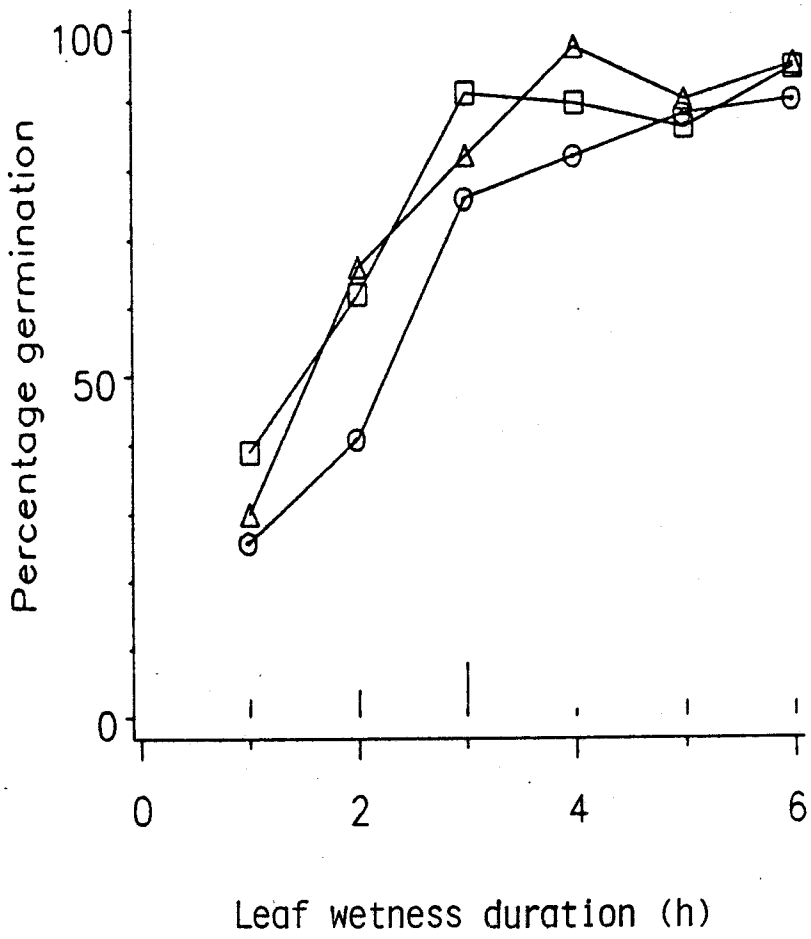

Figure 4.1.3. Time course of conidium germination for three isolates of Pyrenophora teres at constant 20 °C regime (○= WRS 858; □ = M; △ = S). Vertical bars represent standard deviation for each incubation period.

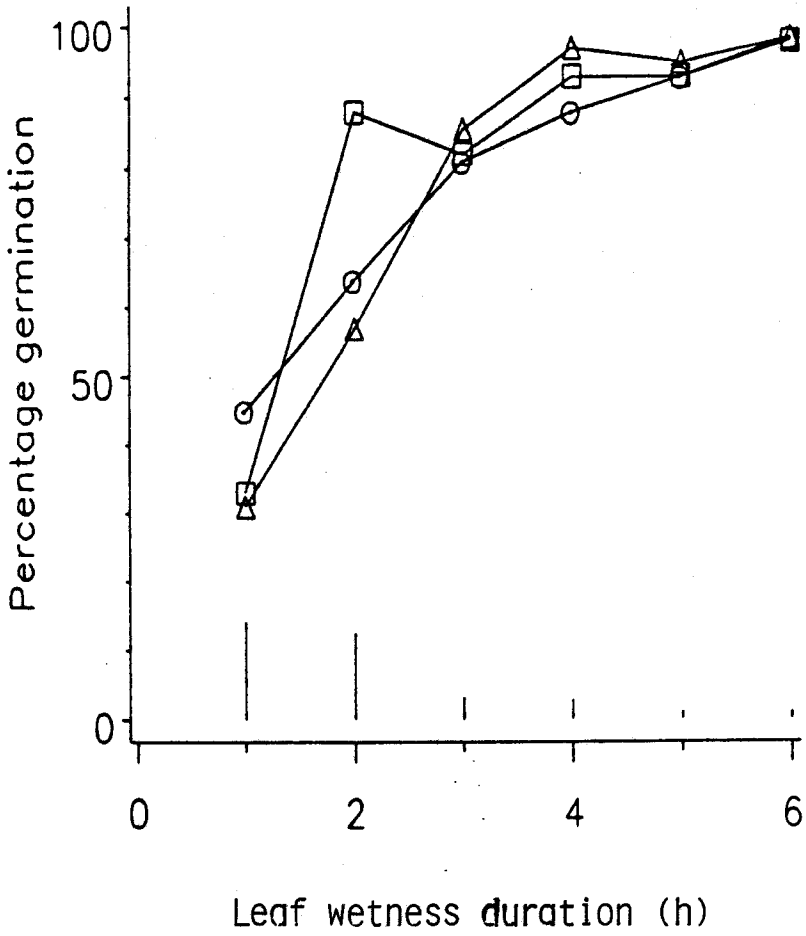

Figure 4.1.4. Time course of conidium germination for three isolates of Pyrenophora teres at constant 25 °C regime (○ = WRS 858; □ = M; △ = S). Vertical bars represent standard deviation for each incubation period.

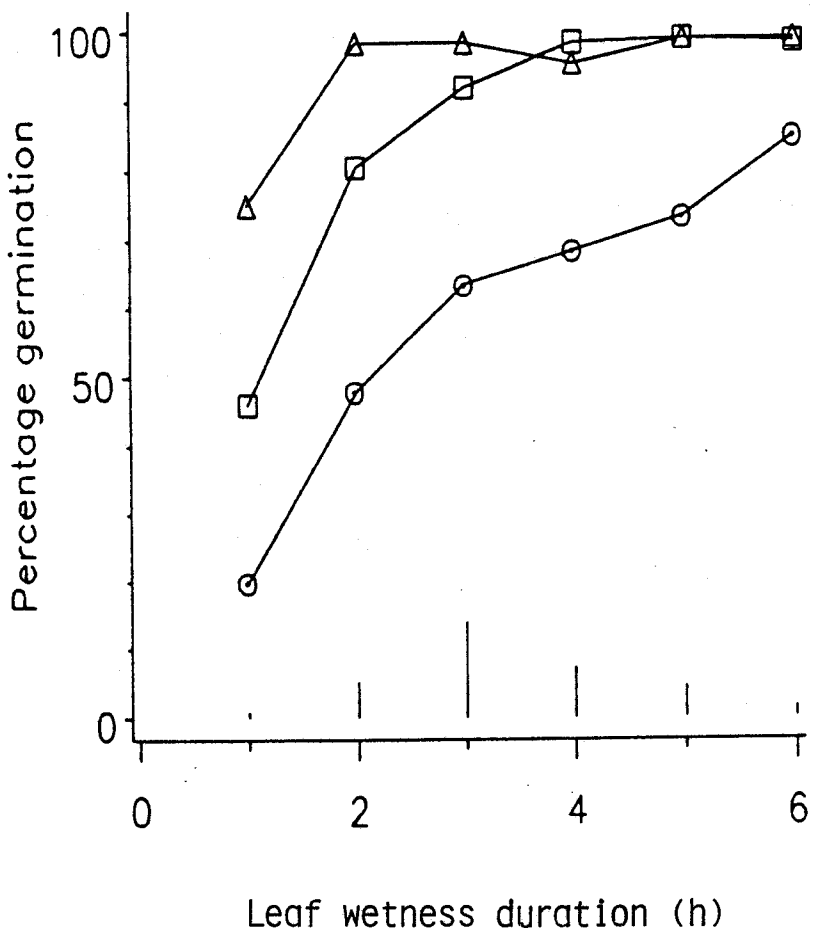

Figure 4.1.5. Time course of conidium germination for three isolates of Pyrenophora teres at 20 decreased to 10 °C regime (○ = WRS 858; □ = M; △ = S). Vertical bars represent standard deviation for each incubation period.

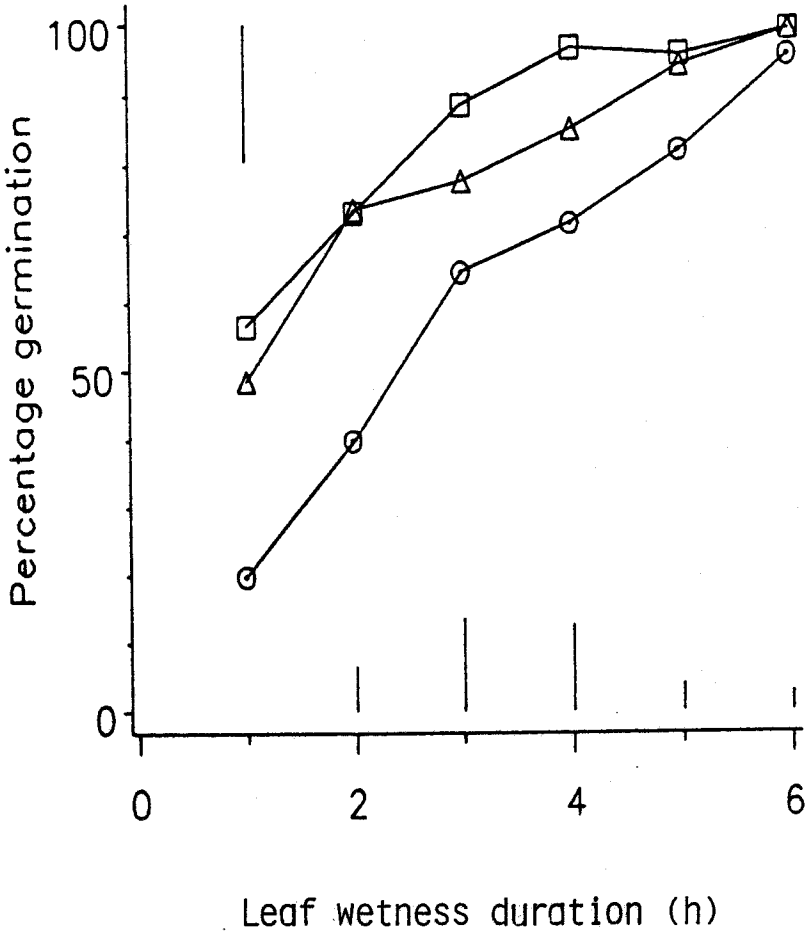

Figure 4.1.6. Time course of conidium germination for three isolates of Pyrenophora teres at 25 decreased to 10 °C regime (○ = WRS 858; □ = M; △ = S). Vertical bars represent standard deviation for each incubation period.

differences between the spot-type (M and S) and net-type (WRS 858) isolates could be observed during the first four hours of the leaf wetness period (Figures 4.1.1 to 4.1.6). Spot-type isolates M and S germinated sooner and usually reached higher percentages than the net-type isolate WRS 858. After four hours, germination of the net-type isolate reached levels similar to those of the spot-type isolates. With constant 25 ^{o}C, no differences were observed. With the 20 decreased to 10 ^{o}C regime, spot-type isolates had a higher germination percentage than the net-type isolate WRS 858 over the entire observation period. With the 25 decreased to 10 ^{o}C regime, no differences were observed. There were no differences between the two spot-type isolates at any temperature regime, with the exception of the constant 10 ^{o}C after 2 and 3 h, and 20 decreased to 10 ^{o}C after 1 h (Figures 4.1.1 and 4.1.5).

The percentage of conidia germinated at six hours ranged from 77 to 100 (Figures 4.1.1 to 4.1.6). This indicates that a leaf wetness period of six hours is sufficient for these isolates to reach their maximum percentage of germination. There were no differences among isolates after six hours at any temperature regime, except the 20 decreased to 10 ^{o}C regime (Table 4.1.1). This indicates that the isolates usually did not differ for maximum percentage of conidia germinated.

4.1.2. Time to infection

During the incubation in the humidity box, free water was present on the leaves. After the leaves were removed from the humidity box, the leaf surface dried in the growth cabinet within 10 to 20 min. As conidia of P. teres do not germinate on dry leaves (Shaw, 1986), it can be assumed that infection of host tissue only occurred as a result of conidium germination during the leaf wetness period. The minimum leaf wetness period necessary to establish infection is referred to as time to infection. The appearance of visible symptoms was assessed twice, because the symptoms appeared later when inoculation was done at 10 or 15 ^{o}C.

Analysis of variance was performed as described in Table 3.1.2. The temperature and isolate effect were highly significant (Table 4.1.2). The temperature x isolate interaction was significant at the 0.1 probability level. For all isolates, the time to infection decreased as the temperature increased from 10 to 20 ^{o}C (Table 4.1.3). Under all regimes the net-type isolate WRS 858 required more time to establish infection than the spot-type isolates M and S. The significant interaction indicates a differential response of the isolates to a change in temperature. An increase in constant temperature regime from 10 to 25 ^{o}C decreased the leaf wetness period required to establish infection from approximately eight to four hours for isolate WRS 858 and from six to three hours for isolates M and S.

The time to infection for the 20 decreased to 10 ^{o}C

Table 4.1.2. Analysis of variance for time to infection.

Source	df	SS	F
Temperature	5	101.78	370.11**
Replication(Temperature)	6	0.33	
Isolate	2	17.69	39.75**
Temperature x Isolate	10	4.97	2.23+
Error	12	2.67	
Sampling error	36	5.00	
Total	71	132.44	

+, ** Significant at the 0.1 and 0.01 probability level, respectively.

Table 4.1.3. Time to infection for three isolates and six temperature regimes.

Temperature	Isolate		
	WRS 858	M	S
°C	h		
10	7.8	5.6	6.0
15	6.0	5.0	4.3
20	4.0	3.3	3.0
25	4.3	3.3	3.5
20 decreased to 10	3.8	3.5	3.0
25 decreased to 10	3.8	3.0	3.0
LSD(0.05)		0.72	

regime and the 25 decreased to 10 °C regime did not differ from those for the constant 20 and 25 °C regimes, respectively (Table 4.1.3). Therefore, temperature regimes with decreasing temperature required a leaf wetness period similar to that of the starting temperature for establishing infection.

4.1.3. Discussion

The finding that a leaf wetness period of six hours was sufficient for maximum percentage of conidium germination at temperatures 10 °C and above, is similar to the results reported by Shaw (1986) for a net-type isolate collected in England. The maximum percentage of germinated conidia ranged from 77 to 100%.

With linear interpolation and extrapolation, time to infection for the net-type isolate WRS 858 with constant 7.5, 12.5 and 17.5 °C regimes was estimated to be 9, 7 and 5 hours, respectively. Similar values were reported by Shaw (1986), who found that time to infection was 8, 7 and 5 hours with constant 8, 13 and 18 °C regimes, respectively. Singh (1963a) observed infection after five hours at room temperature for five net-type isolates. In the current study, the net-type isolate established infection in four hours at the constant 20 °C regime.

Differences between spot-type isolates M and S and net-type isolate WRS 858 were observed for rate of conidium germination and time to infection. If typical, these

differences among isolates suggest that spot-type isolates have a competitive advantage over net-type isolates in disease establishment. Over time, spot-type isolates might replace net-type isolates. Spot-type isolates have increased in prevalence, as indicated by recent surveys (Tekauz, 1978; Tekauz and Buchannon, 1977). As well, due to the shorter time to infection, spot-type isolates have the potential to establish themselves in areas that were previously unfavourable for net-type isolates due to unfavourable combinations of temperature and leaf wetness period. This should increase the prevalence of net blotch in Saskatchewan.

4.2. Potential for disease establishment in Saskatchewan

The need for a susceptible host, a pathogen and favourable microclimate to establish a pathosystem is well-known. However, little is known about the net blotch-barley pathosystem (Shipton et al., 1973). Temperature, precipitation and dew formation are the most important microclimatic factors in disease establishment (Rotem, 1978). In experiment II, the net blotch-barley pathosystem was studied in relation to microclimatic factors.

4.2.1. Microclimate

Net blotch developed on barley leaves during June and July at Shellbrook in both 1986 and 1987. Temperature, precipitation and leaf wetness were recorded at the experimental site. Average monthly values for maximum

temperature, minimum temperature and precipitation are presented in Table 4.2.1. For comparison, average monthly values for maximum temperature, minimum temperature, precipitation and their normal values for the period 1951 to 1980 were obtained from the Environment Canada weather station in Prince Albert (44 km east of Shellbrook). These normals were assumed to be the best available long-term estimates for Shellbrook.

In both 1986 and 1987, the maximum and minimum temperature were above normal at Prince Albert for June. For July, the maximum temperature was below normal and the minimum temperature above normal. Generally, the monthly average maximum temperature was higher and the monthly average minimum temperature was lower at Shellbrook than at Prince Albert. At Shellbrook, temperature was recorded in the crop canopy, whereas at Prince Albert observations were taken under standardized meteorological conditions. In general, microclimatic conditions within the crop canopy tend to vary from the macroclimatic conditions, as measured under standard conditions (Coakley, 1985; Rotem, 1978).

In June of 1986 and 1987, precipitation was slightly below normal at Prince Albert and Shellbrook (Table 4.2.1). In July of 1986, precipitation was almost twice normal. In July of 1987, precipitation was slightly above normal at Shellbrook and almost three times normal at Prince Albert. In both years, precipitation was adequate for crop growth throughout the growing season.

Table 4.2.1. Monthly average maximum temperature, monthly average minimum temperature, monthly precipitation at Shellbrook and Prince Albert in June and July of 1986 and 1987 and monthly normals for the period 1951 - 1980 at Prince Albert.

	June		July	
Year	Shellbrook	Prince Albert	Shellbrook	Prince Albert
Maximum temperature (°C)				
Normal		21.5		24.2
1986	22.6	22.7	23.5	22.6
1987	25.6	24.5	25.8	22.9
Minimum temperature (°C)				
Normal		7.7		10.6
1986	4.5	7.9	8.4	11.1
1987	7.3	11.2	9.3	11.7
Precipitation (mm)				
Normal		69.1		65.3
1986	47.5	25.5	114.0	105.8
1987	55.6	62.6	72.6	175.8
Precipitation (days)				
Normal		12		12
1986	12	14	14	17
1987	10	12	18	19

The occurrence of free water on the leaf surface is required for infection of leaves by P. teres (Shaw, 1986). In the field, leaves are wetted by precipitation or dew formation. Precipitation was recorded for 12 and 10 days in June and for 14 and 18 days in July of 1986 and 1987, respectively (Table 4.2.1). Precipitation provided free water on the leaves, but the ensuing leaf wetness period was restricted. Dew formed frequently at night. The duration and frequency of leaf wetness periods, as a result of precipitation or dew formation, are presented in Table 4.2.2. In section 4.1, it was inferred that infection would occur after 9 hours of leaf wetness at 7.5 ^{o}C. Therefore, five and four periods in June of 1986 and 1987, respectively, were of sufficient duration for the establishment of infection (longer than 10 hours). During July, 18 and 11 periods in 1986 and 1987, respectively, were of sufficient duration for the establishment of infection. This number of periods sufficient for the establishment of infection is a conservative estimate. The effect of intermittant leaf wetness periods on the establishment of infection has not been investigated. It is possible that infection may be established during several short periods of leaf wetness, as shown for Stemphylium botryosum in irrigated tomato crops (Bashi and Rotem, 1974).

Table 4.2.2. Frequency of the duration of leaf wetness periods at Shellbrook in June and July of 1986 and 1987.

	1986		1987	
Duration	June	July	June	July
— h —				
1 - 5	4	4	10	4
6 - 10	11	4	7	8
11 - 15	4	9	4	5
16 - 20	1	7	0	4
21 - 30	0	1	0	1
> 30	0	1	0	1
Total	20	26	21	23

4.2.2. Development of barley

In 1986 and 1987, barley emerged in early June. An even stand was established on the barley stubble. Elrose reached equivalent developmental stages about one week earlier in 1987 than in 1986 (Figure 4.2.1). June 30 was the 38th and 35th day after seeding in 1986 and 1987, respectively. Concurrent with developmental stages, the five uppermost leaves appeared during June and early July in 1986 (Figure 4.2.2). All seven leaves appeared during June in 1987 (Figure 4.2.3). All leaves expanded to their maximal leaf area in about one week. Once maximum leaf area was reached, green leaf area declined due to disease, senescence and/or mechanical damage. Leaves were numbered from the flag leaf downward (i.e. leaf 1 = flag leaf, leaf 2 = penultimate leaf, etc.). In both 1986 and 1987, the lower leaves (5 to 7) lost their photosynthetic activity in late June. Leaf 4 lost its photosynthetic activity by the middle of July. The upper leaves (1 to 3) lost their photosynthetic activity in early August in 1986 and in late July in 1987, shortly after spike emergence. Leaf life span ranged from 20 - 30 days. Total green leaf area increased until the middle of July in 1986 and the end of June in 1987. Total green leaf area decreased rapidly after it reached its maximum.

4.2.3. Progress of net blotch

Spot-type net blotch (*P. teres* f. *maculata*) was the major pathogen observed at Shellbrook in 1986 and 1987. Net-

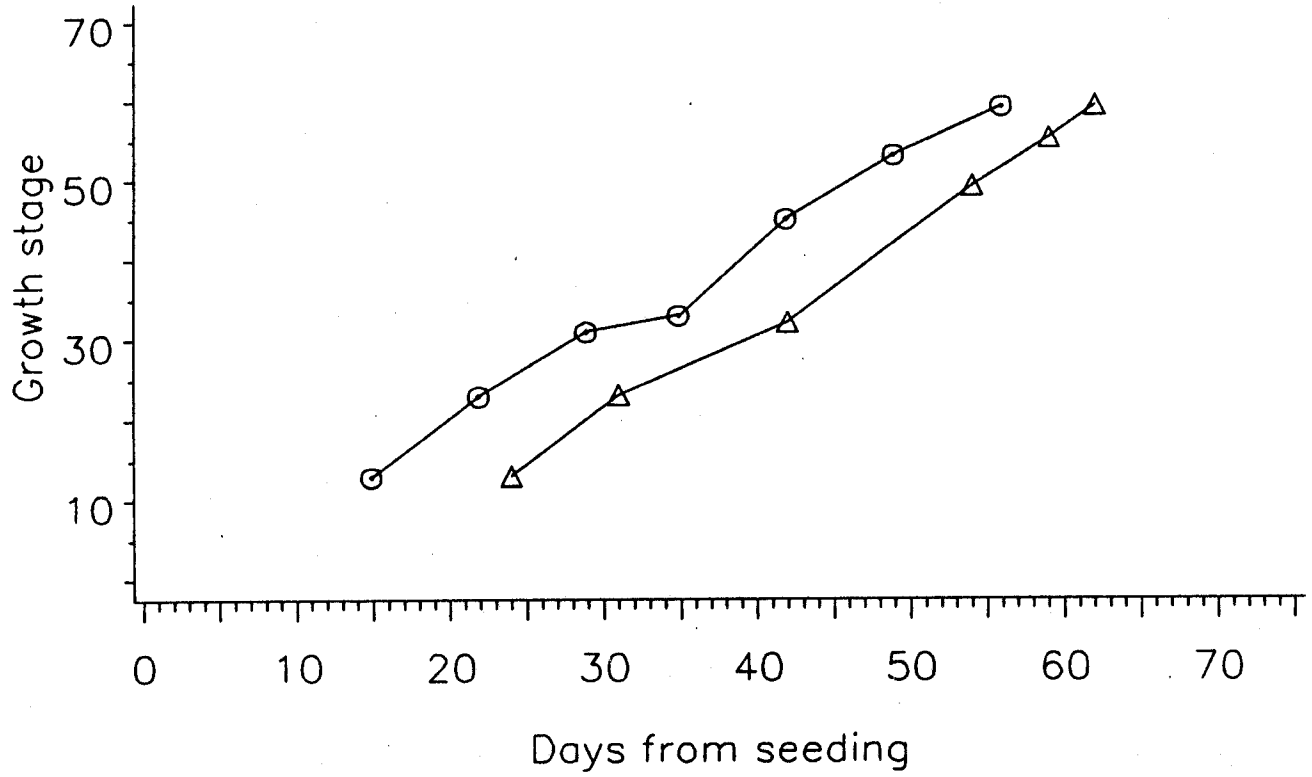

Figure 4.2.1. Development of Elrose based on the Zadoks scale (Zadoks et al., 1974) at Shellbrook in 1986 (△) and 1987 (○).

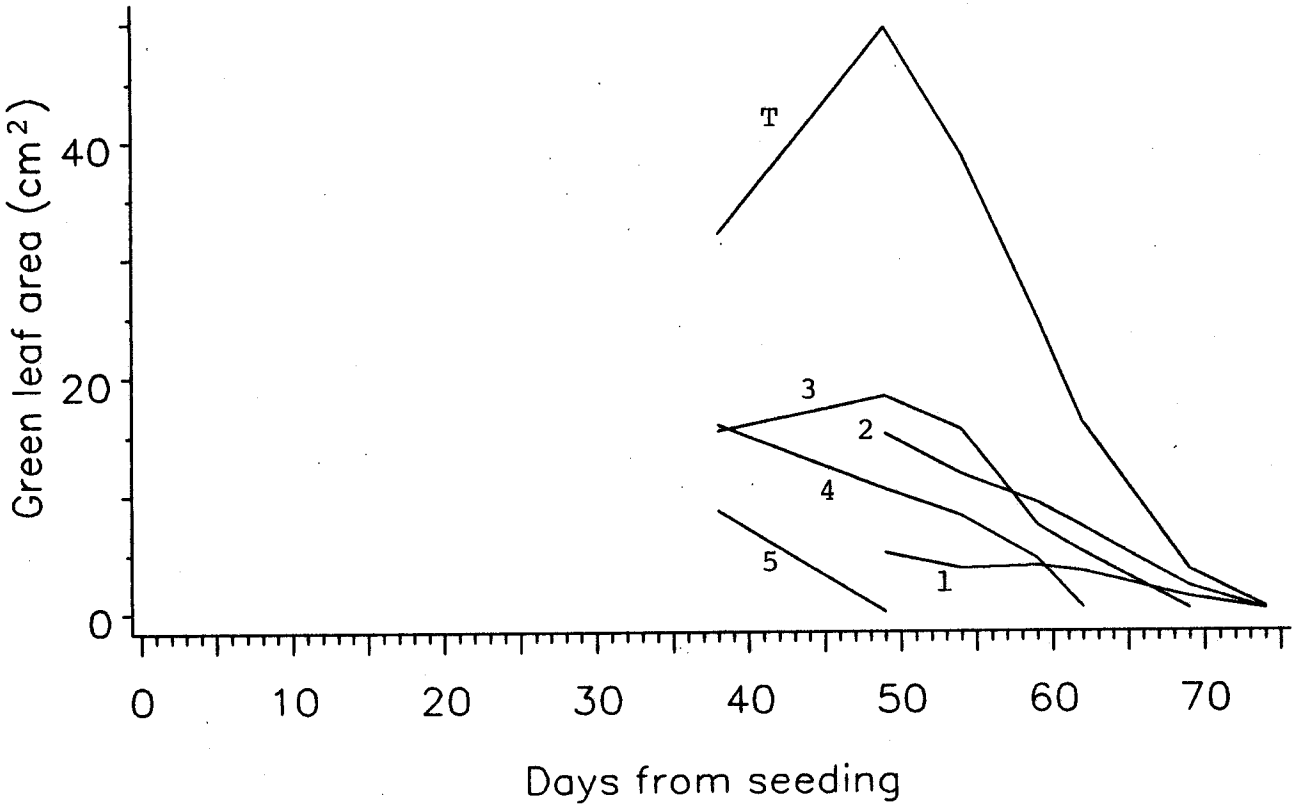

Figure 4.2.2. Green leaf area for five leaf positions (1-5) and total green leaf area (T) per main culm in Elrose on seven observation days at Shellbrook in 1986.

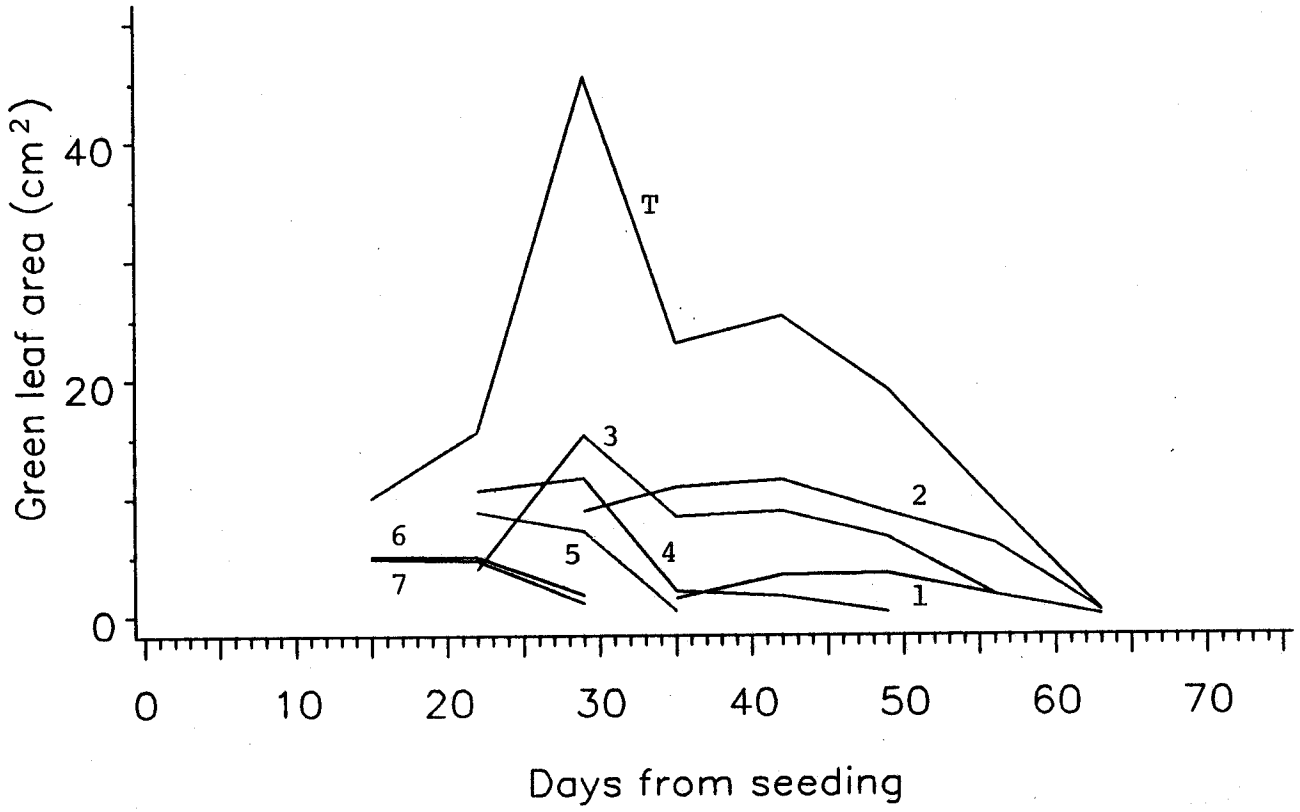

Figure 4.2.3. Green leaf area for seven leaf positions (1-7) and total green leaf area (T) per main culm in Elrose on eight observation days at Shellbrook in 1987.

type symptoms of net blotch were not observed. Spot blotch (Cochliobolus sativus) and scald (Rhynchosporium secalis) were also observed, but did not occur on more than 3% of the total leaf area. Spot-type net blotch was present from the seedling stage onward. Differences in barley development were reflected in the progress of net blotch for each leaf position. Severity of net blotch on Elrose in 1986 lagged about one week behind that in 1987.

On the lower leaves (numbers 5 to 7), net blotch developed slowly and caused relatively little necrosis (Figures 4.2.4 and 4.2.5). On the upper leaves (1 to 4), net blotch caused extensive necrosis. As well, net blotch lesions developed on the leaf sheaths, glumes and awns. Infection of the lower leaves was likely inhibited by the weather conditions in June. Only four or five nights provided conditions conducive for infection (Table 4.2.2). In July the weather was more favourable with 11 to 18 nights providing conditions conducive to infection.

4.2.4. Sporulation potential and latent period

Crop debris left from the previous season likely served as the source of primary inoculum. Based on sampling, the sporulation potential was 200 to 400 conidia per cm of stem and 700 to 900 conidia per leaf. It should be noted that these leaves were completely shrivelled and measured only a few square centimeters each. Pseudothecia-like bodies were observed on the leaves and straw, but no ascospores

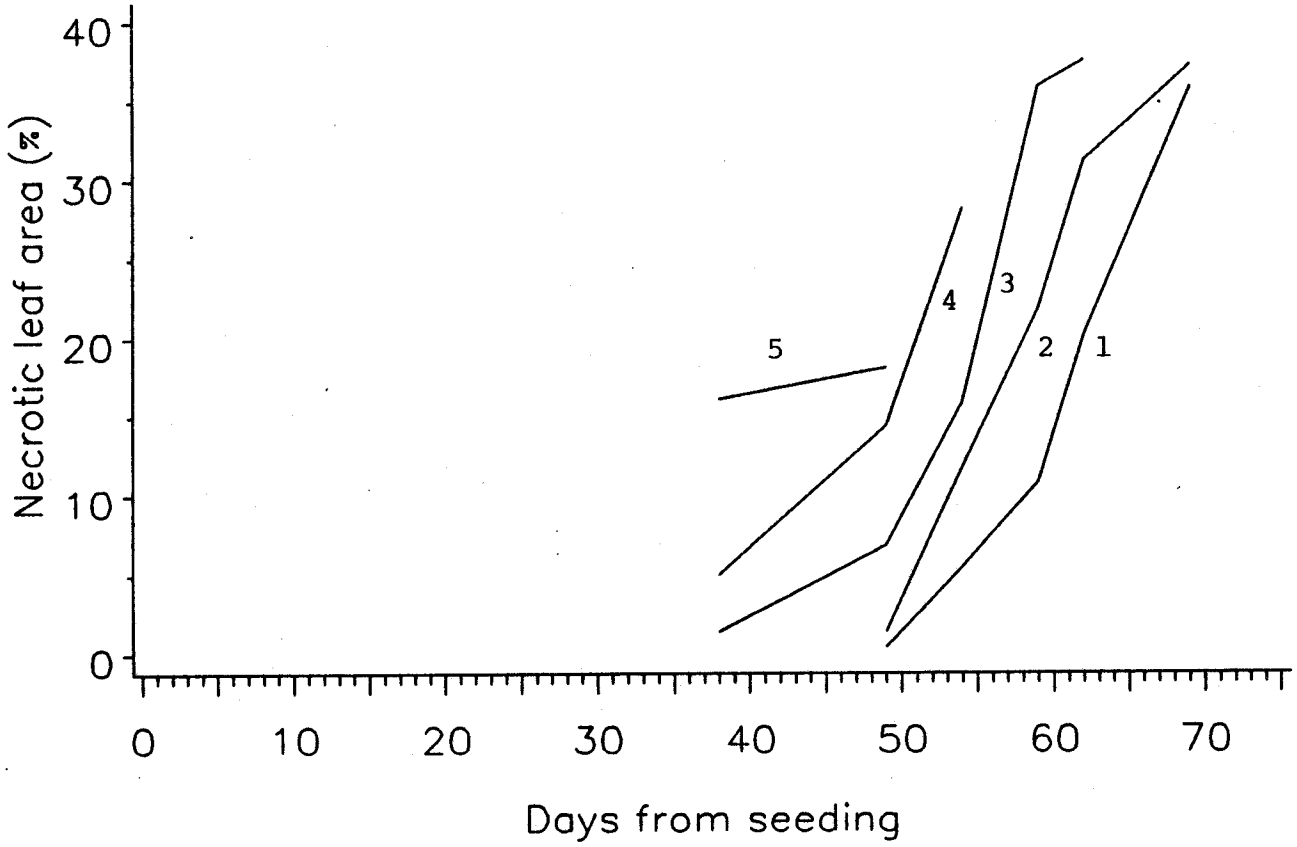

Figure 4.2.4. Percentage necrotic leaf area for five leaf positions (1-5) in Elrose infected with Pyrenophora teres f. maculata on six observation days at Shellbrook in 1986.

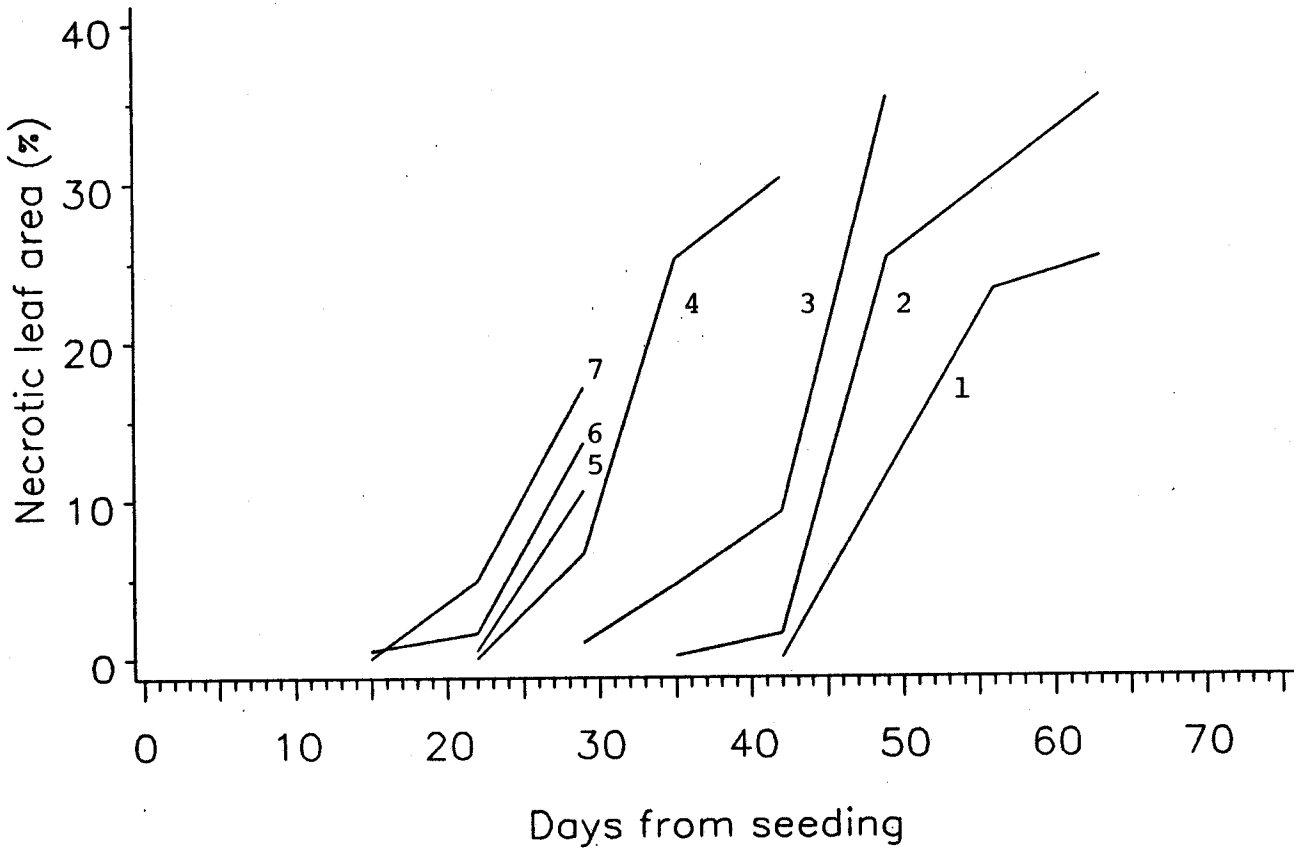

Figure 4.2.5. Percentage necrotic leaf area for seven leaf positions (1-7) in Elrose infected with Pyrenophora teres f. maculata on eight observation days at Shellbrook in 1987.

developed.

The number of conidia produced on infected leaves of the growing barley leaves is presented in Table 4.2.3. Sporulation occurred only on leaves with more than 50% senesced tissue. No sporulation was observed in the dark-brown necrotic spots. Sporulation was observed only on the chlorotic and necrotic areas surrounding the dark-brown spots. Sporulation was observed on the entire surface of completely senesced leaves. Completely senesced leaves were easily damaged by treatment with distilled water. On a few occasions leaves were discarded after the third day. Leaf 1, 2 and 3, sampled on July 14, 21, 24, and August 4, failed to produce spores within the first 24 hours of incubation.

The number of conidia produced per leaf was very high during the first two weeks following senescence (>10,000 conidia per rinse) (Table 4.2.3). The production of conidia decreased rapidly after this initial period. In this experiment conidia were produced continually and production did not cease after rinsing. In contrast, Shaw (1986) observed a one-time production of conidia on inoculated leaves.

The latent period is estimated as the period between the first appearance of net blotch symptoms and the onset of sporulation. In 1987, the latent period was 20 days for leaf 3 through 7. The latent period for leaf 1 and 2 was 14 days. These differences in latent period were probably due to the higher temperature in July.

Table 4.2.3. Number of conidia per leaf obtained for each leaf position after one, three and five days of incubation for six sampling dates in 1987.

Leaf position	Observation day	Days from seeding[a] 35	42	49	56	63	70
7	1	2110	.	.	.	.	.
	3	4830					
	5	7130					
6	1	950	1445	.	.	.	.
	3	3990	1840				
	5	10585	-				
5	1	.	2890	3440	.	.	.
	3		21890	3000			
	5		-	2890			
4	1	.	1720	2560	2780	.	.
	3		19055	8170	7000		
	5		-	11000	11280		
3	1	.	.	1000	6560	5330	0
	3			8780	16390	2890	2555
	5			17000	20610	3335	2165
2	1	.	.	0	0	8000	0
	3			1890	6220	6725	5670
	5			8330	14450	8275	8830
1	1	.	.	0	0	0	0
	3			4670	7890	8000	7670
	5			19220	21670	8890	9890

[a] Ten leaves were sampled on each date; Values represent the average number of conidia observed on each observation day;
- = number of conidia could not be determined;
. = leaves were not sampled.

4.2.5. Incidence of airborne conidia

During the investigation period, primarily conidia of *P. teres* were trapped. Conidia of *Alternaria spp.* and *Cladosporium spp.* and some unidentified genera were also observed along with pollen, small insects, soil particles and fragments of mycelium. No ascospores of *P. teres* were observed.

The number of conidia trapped was less than 100 per day during June of 1986 (Figure 4.2.6). In 1987, up to 350 conidia were trapped daily during June (Figure 4.2.7). During July of 1986 and 1987, the number of conidia increased to more than 500 per day, presumably because more leaves senesced (Figures 4.2.8 and 4.2.9).

The incidence of airborne conidia showed a diurnal pattern (Figure 4.2.10), which was largely independent of the observed weather parameters. During the morning the number of airborne conidia increased as more leaves dried. The highest incidence was observed between 12:00 and 16:00 h. The incidence of airborne conidia was high on days with extended dew periods (>10 h) and when the temperature difference on the preceding day exceeded 10 ^{o}C. Incidence was low when the maximum temperature exceeded 25 ^{o}C, or when the minimum temperature fell below 5 ^{o}C.

The incidence of airborne conidia showed similar increases later in the growing season in Eastern Canada (Martin and Clough, 1984) and in West Germany (Mauler, 1983). A similar diurnal pattern of conidia incidence was also

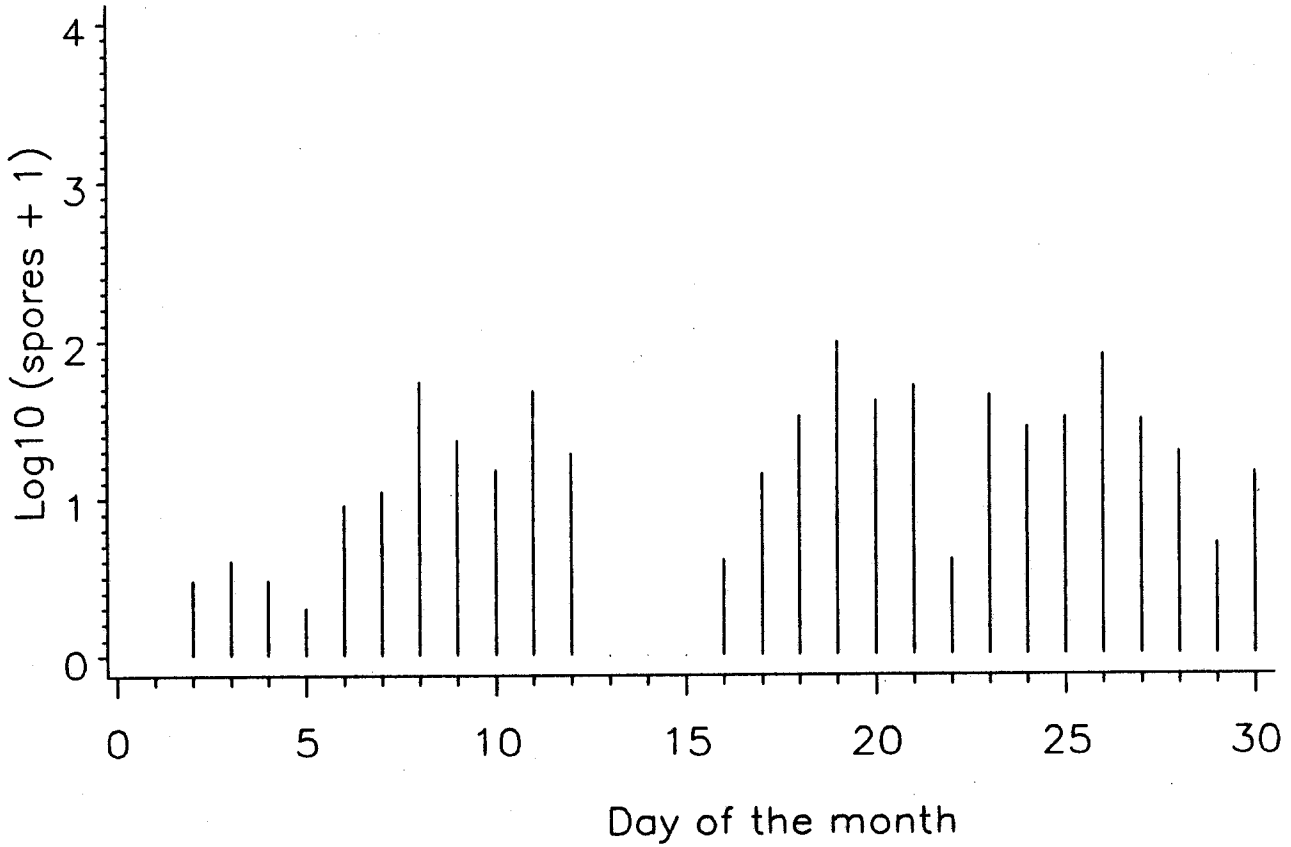

Figure 4.2.6. Number of conidia of Pyrenophora teres trapped per day at Shellbrook in June 1986.

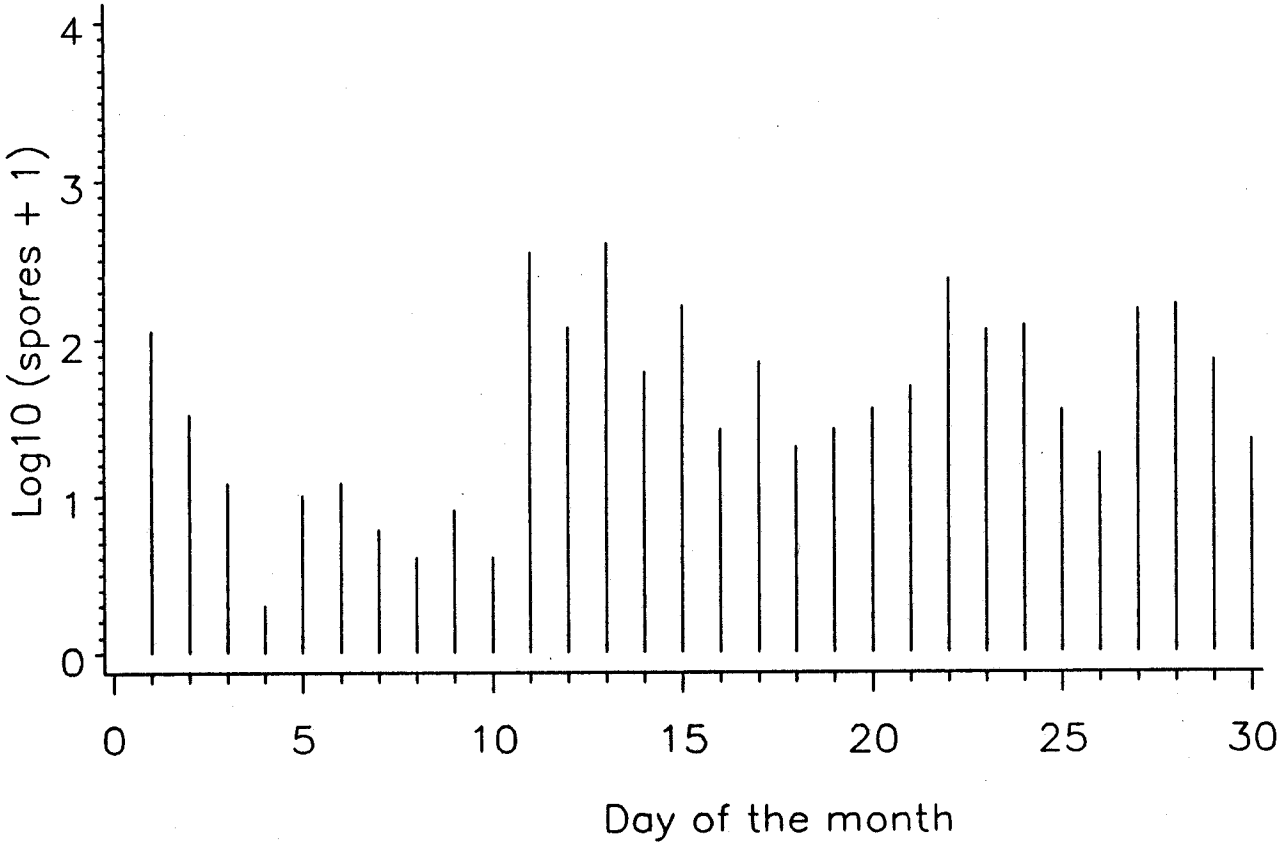

Figure 4.2.7. Number of conidia of Pyrenophora teres trapped per day at Shellbrook in June 1987.

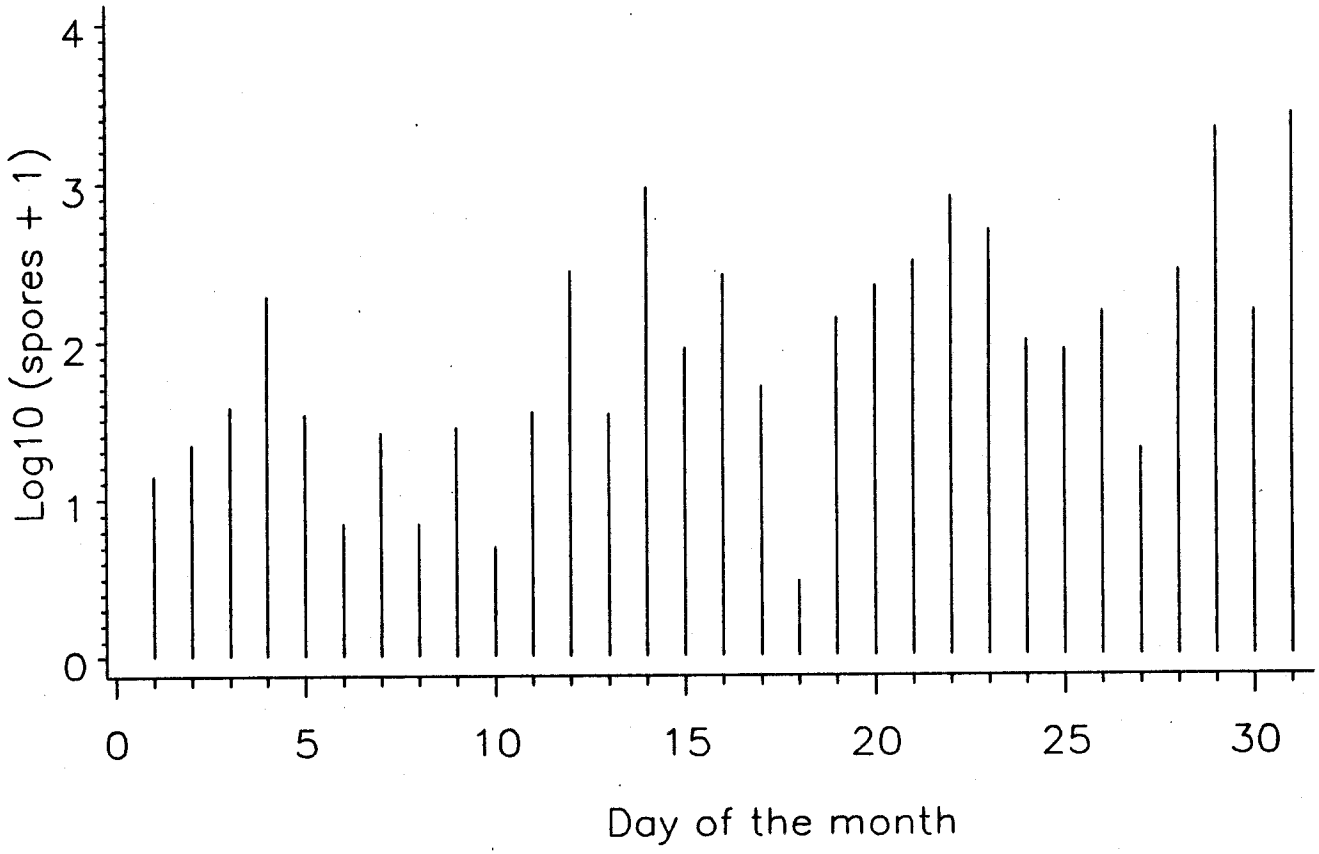

Figure 4.2.8. Number of conidia of Pyrenophora teres trapped per day at Shellbrook in July 1986.

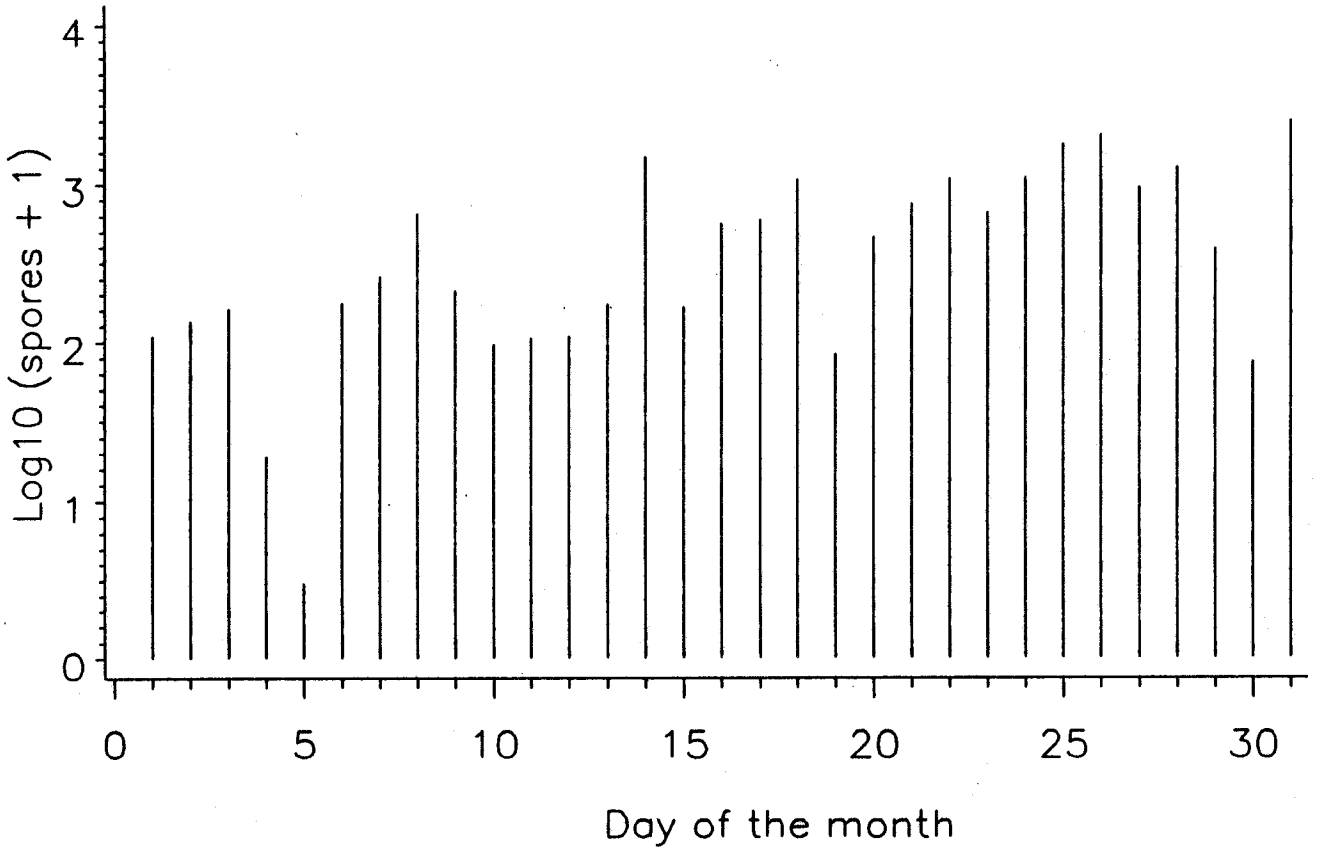

Figure 4.2.9. Number of conidia of Pyrenophora teres trapped per day at Shellbrook in July 1987.

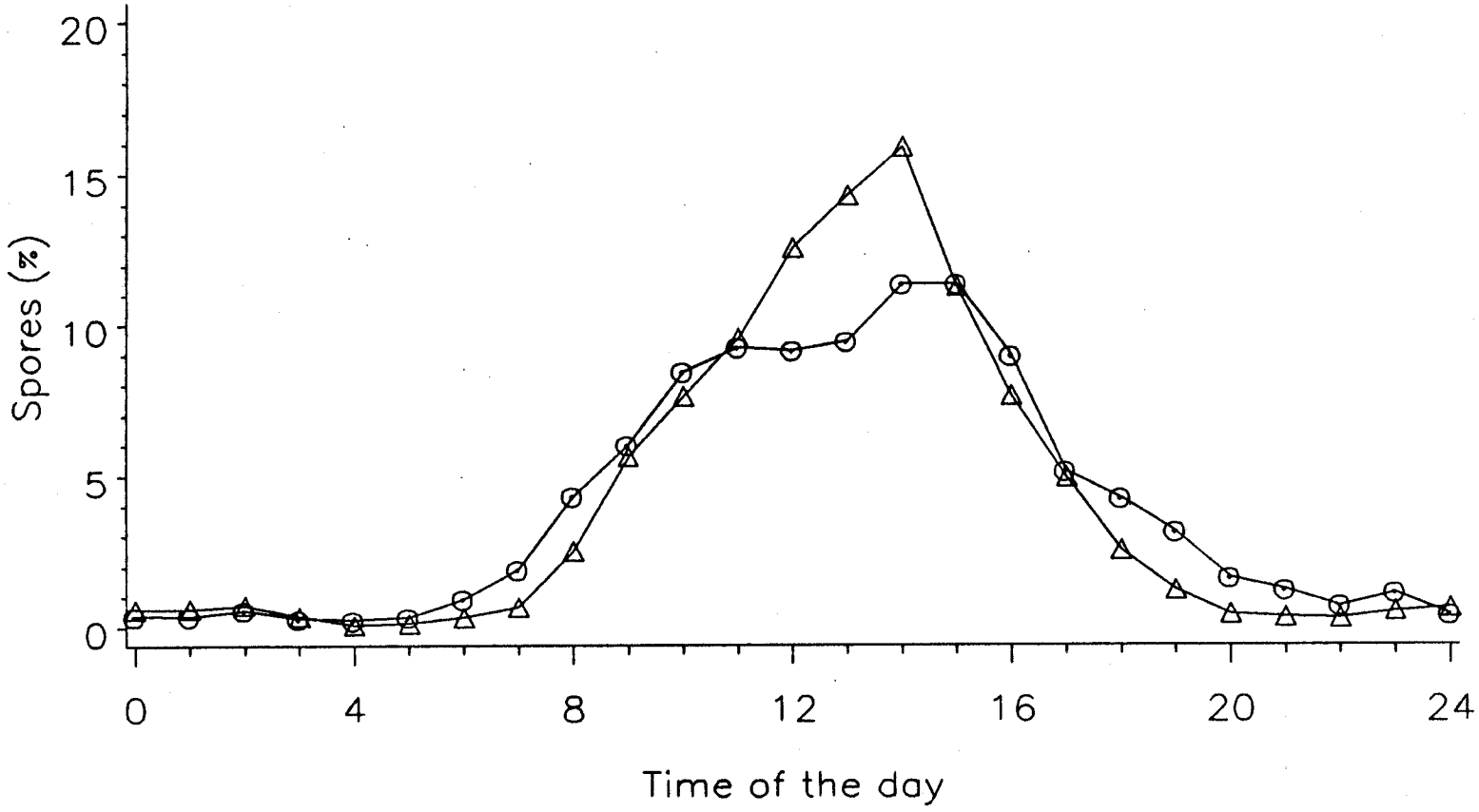

Figure 4.2.10. Diurnal trend in incidence of airborne conidia of Pyrenophora teres at Shellbrook in June and July of 1986 (Δ) and 1987 (O). Percentages of conidia were calculated as the ratio of the observed number to the total number observed during the investigation period multiplied by 100.

observed by Martin and Clough (1984) in Eastern Canada. The incidence of airborne conidia peaked at 12:00 h. Afterwards, the incidence decreased rapidly due to deposition and dispersal of conidia. Diurnal periodicities have also been reported for related pathogens, such as P. tritici-repentis (Morrall and Howard, 1975) and C. sativus (Couture and Sutton, 1978).

Airborne conidia were observed on most days. As well, conidia developed on senesced leaves when these were kept overnight in the laboratory (Section 4.2.4). This suggests that in the field, conidia were formed each night when the requirements for conidiation including high humidity were met (Oniserosan and Banttari, 1969; Shaw, 1986). Similarly, for other Western Canadian isolates, Tekauz and Buchannon (1977) observed sporulation within 24 h of incubation. In contrast, for a British isolate, Shaw (1986) reported that conidia were only produced after 42 h at 18 ^{o}C and after 68 h at 8 ^{o}C. Singh (1956) reported that Minnesota isolates did not sporulate below 10 ^{o}C. These observations are not consistent with the data obtained at Shellbrook, where conidia were formed during leaf wetness periods shorter than 20 h and at temperatures below 10 ^{o}C.

4.2.6. Discussion

Based on the data obtained, the following sequence of events is proposed. Conidia were produced daily on the crop debris and senesced leaves during the night. During the

day, conidia became airborne and some were deposited on healthy leaf tissue. Infection subsequently occurred during the next leaf wetness period of sufficient duration. In many instances this was the following night.

Barley emerged in early June and no sporulation was observed on the growing crop before June 30. Therefore, crop debris were likely the only source of inoculum for the infection of lower leaves (4 to 7). Crop debris and the senesced lower leaves were the sources of inoculum for the upper leaves (1 to 3). For each leaf position, infection was apparently due to external sources of inoculum. Therefore, P. teres completed only one infection cycle for each leaf position and behaved as a simple interest disease. On a crop basis, the number of infection cycles in Saskatchewan appears to be limited. This conclusion contrasts with the inferences made by Shaw (1986) in England. He reported latent periods of 8 - 12 days and a life span of 50 - 60 days for each leaf, and concluded that several infection cycles could occur on a single leaf.

4.3. Progress curves for net blotch

The progress of disease severity can be shown as a curve of disease severity against time, called the disease progress curve. These curves can be used for longitudinal comparison of disease development (Kranz, 1974). In this section, the monomolecular and logistic functions (described in Section 2.3; Equations 2.1 and 2.2) were fitted to disease readings

of the penultimate leaf for the untreated control and the repeatedly sprayed control (Experiment III).

4.3.1. Fitting of disease progress curves

4.3.1.1. Estimation of parameters

A complete description of the monomolecular and logistic functions requires three parameters: asymptote, location parameter, and rate parameter (Kiyosawa, 1972; Park and Lim, 1985). If sufficient observations are taken, then all parameters can be estimated with iterative curve fitting procedures for non-linear functions, such as NLIN of the SAS statistical package (SAS Institute, 1985). Usually, too few observations are taken to qualify for the use of this method and the parameters can only be estimated using linear regression analysis. In that case, the asymptote must be estimated empirically. This estimate is important, as it is used in transforming the data. The general formula of the logistic transformation is:

$$X' = \ln (X/(A-X)), \qquad (4.1)$$

where X represents the untransformed data, X' the transformed value and A the asymptote. The transformed data are then used to estimate the location and rate parameter (Equation 3.3). Although the asymptote has a profound effect on the estimates of other parameters, it is generally set at one. Experimental values smaller than one are frequently obtained (Shaner and Finney, 1977). The use of values other than one for the asymptote has been suggested by Kiyosawa (1972) and Zadoks

and Schein (1979). The use of an estimate larger than the true value for the asymptote causes an underestimation of the rate parameter (Park and Lim, 1985).

The monomolecular curve is also sensitive to overestimation of the asymptote, as the asymptote is used in transforming the observations. The general formula for the monomolecular transformation is:

$$X' = \ln (A/(A-X)). \qquad (4.2)$$

4.3.1.2. Biological interpretation

The transition from mathematical description to biological interpretation requires restrictions that accommodate the biological system investigated. The range of the function must be restricted to the interval [0,1] or [0,100%]. The measurement of disease severity must be positive and may not exceed the maximum leaf area available. As the maximum leaf area is not included in the function, it must be treated as a constant. Therefore, these functions should only be used for single leaf positions, when it can be assumed that the leaf area is constant.

The asymptote represents the maximum percentage of diseased leaf area. Its value is usually set at 1.00, but smaller values have been observed. An asymptote of 0.37 was observed for the percentage leaf area covered by rust pustules (Peterson et al., 1948). Reasons for asymptotes smaller than 1.00 have not been investigated, but Kiyosawa (1972) suggested they may be due to:

1. changes in environmental conditions or
2. increased resistance with plant age.

To accommodate these limitations, an asymptote based on leaf area may be replaced by an asymptote that describes the termination time of the epidemic.

The rate parameter describes the velocity of disease progress. The maximum velocity is A*C at X=0 and t=-B/C for the monomolecular function and A*C/4 at X=A/2 and t=-B/C for the logistic function. The maximum velocity of the monomolecular function is higher than that of the logistic function. The maximum velocity of the monomolecular function is obtained at the beginning of the epidemic. The monomolecular function has been used in situations where large amounts of initial inoculum are present (Jowett et al., 1974).

The location of the function is determined by the quotient of the location and rate parameter (-B/C). For the monomolecular function this quotient describes the location of the intercept or the start of the epidemic. For the logistic function this quotient describes the location of the point of inflexion or the point at which diseased leaf area reached 50% of its maximum. At these locations, the velocity of the epidemic is maximal for both functions.

4.3.2. Observed progression of net blotch

4.3.2.1. Percentage necrotic leaf area

For Elrose, the monomolecular function was fitted to percentage necrotic leaf area. The asymptote was estimated as the largest observed value plus 0.005. After transformation, the location and rate parameter were estimated. The quotient was determined as it describes the start of the epidemic. To determine the effect of fungicide application on a susceptible cultivar, parameter estimates for the untreated control and the repeatedly sprayed control of Elrose were compared. The asymptote for the untreated control was larger in 1986 than in 1985 (Table 4.3.1). The asymptote for the repeatedly sprayed control was similar in both years. In both years, the asymptote for the untreated control was larger than that for the repeatedly sprayed control. The location parameter for the untreated control was larger in 1985 than in 1986. The location parameter for the repeatedly sprayed control was similar in both years. The location parameter was larger for the repeatedly sprayed control than for the untreated control in both years. The rate parameter for the untreated control was larger in 1986 than in 1985. The rate parameter for the repeatedly sprayed control was larger in 1985 than in 1986. In both years, the rate parameter for the untreated control was larger than that for the repeatedly sprayed control. For both treatments, the quotient was larger in 1986 than in 1985. In 1985, the quotient was similar for both treatments. Thus, necrotic symptoms appeared on the same

Table 4.3.1. Parameters of the monomolecular curve fitted to the percentage necrotic leaf area for two treatments in Elrose in 1985 and 1986.

Year	Treatment[a]	Asymptote[b]	Location parameter	Rate parameter	Quotient
1985	Untreated	0.338b	-7.07b	0.156b	45.2c
1986	Untreated	0.382a	-10.96c	0.213a	51.6b
1985	Repeated	0.218c	-6.23a	0.135c	46.1c
1986	Repeated	0.215c	-6.29a	0.117d	53.9a

[a] Untreated = No application of fungicide; Repeated = Application of Tilt at 7 to 10 day intervals.

[b] Means within a column followed by the same letter are not significantly different from each other at the 0.05 probability level according to LSD test.

day in both treatments. In 1986, the quotient was larger for the repeatedly sprayed control than for the untreated control. Thus, necrotic symptoms appeared later on the repeatedly sprayed control than on the untreated control. In both treatments, the necrotic symptoms appeared later in 1986 than in 1985.

For Elrose, regular application of the fungicide Tilt decreased the value of the asymptote and the rate parameter and increased the value for the location parameter and the quotient (Table 4.3.1). This indicates that fungicide application slowed the progress of percentage necrotic leaf area and lowered the maximum percentage necrotic leaf area. Fungicide application delayed the appearance of necrotic symptoms in the repeatedly sprayed control.

For Argyle, the exponential function was fitted to percentage necrotic leaf area. After transformation, the location and rate parameter were estimated. To determine the effect of fungicide application on a resistant cultivar, parameter estimates for the untreated control and the repeatedly sprayed control of Argyle were compared. No differences were observed between the treatments for any parameter (Table 4.3.2). Therefore, fungicide application did not influence the progress of percentage necrotic leaf area on Argyle.

The monomolecular function was fitted to percentage necrotic leaf area on Elrose and the exponential function was fitted to percentage necrotic leaf area on Argyle. Because

Table 4.3.2. Parameters of the exponential curve fitted to the percentage necrotic leaf area for two treatments in Argyle in 1986.

Treatment[a]	Location parameter[b]	Rate parameter
Untreated	-2.16	0.102
Repeated	-3.41	0.090

[a] See Table 4.3.1.
[b] Means within a column do not differ significantly from each other at the 0.05 probability level.

different functions were used, no comparisons can be made between the treatments of susceptible Elrose and resistant Argyle.

4.3.2.2. Percentage affected leaf area

The sum of the percentage necrotic leaf area and percentage chlorotic leaf area is called percentage affected leaf area. The logistic function was fitted to observations of percentage affected leaf area. The asymptote was set at 1.00 in all cases. The location and rate parameter were estimated after transformation. The quotient was determined, as it describes time to 50% affected leaf area. Mean values for asymptote, location parameter, rate parameter, and quotient for Elrose are presented in Table 4.3.3 and for Argyle in Table 4.3.4.

To determine the effect of fungicide application on a susceptible cultivar, parameter estimates for the untreated control and the repeatedly sprayed control of Elrose were compared. In both years, the location parameter was larger for the repeatedly sprayed control than for the untreated control (Table 4.3.3). In both years, the rate parameter was larger for the untreated control than for the repeatedly sprayed control. The location and rate parameter did not differ within treatments. The quotient was smaller for the untreated control than for the repeatedly sprayed control in both years. For the untreated control, time to 50% affected leaf area occurred on day 58 (July 24) in 1985 and

Table 4.3.3. Parameters of the logistic curve fitted to the percentage affected leaf area for two treatments in Elrose in 1985 and 1986.

Year	Treatment[a]	Location parameter[b]	Rate parameter	Quotient
1985	Untreated	-15.84b	0.270a	58.7d
1986	Untreated	-18.11b	0.287a	63.2c
1985	Repeated	-11.57a	0.174b	66.6b
1986	Repeated	-13.37a	0.175b	76.6a

[a] See Table 4.3.1.
[b] Means within a column followed by the same letter are not significantly different from each other at the 0.05 probability level according to LSD test.

Table 4.3.4. Parameters of the logistic curve fitted to the percentage affected leaf area for two treatments in Argyle in 1986.

Treatment[a]	Location parameter[b]	Rate parameter	Quotient
Untreated	-10.09	0.099	102.4
Repeated	-10.37	0.113	92.6

[a] See Table 4.3.1.
[b] Means within a column do not differ significantly from each other at the 0.05 probability level.

day 63 (July 25) in 1986 for the untreated control. For the repeatedly sprayed control, time to 50% affected leaf area was delayed by eight and 13 days in 1985 and 1986, respectively, compared to the untreated control.

For Elrose, repeated application of the fungicide Tilt decreased the value of the rate parameter and increased the value of the location parameter and the quotient (Table 4.3.3). This indicates that application of Tilt slowed the progress of percentage affected leaf area in the repeatedly sprayed control and increased the time required to reach 50% affected leaf area. The latter effect was partly negated by an increase of the location parameter.

To determine the effect of fungicide application on a resistant cultivar, parameter estimates for the untreated control and the repeatedly sprayed control of Argyle were compared. No differences were observed between treatments for any parameter (Table 4.3.4). Therefore, application of the fungicide did not influence disease development on the resistant cultivar Argyle.

To determine the effect of resistance on disease progress, parameter estimates for 1986 of the untreated control of Argyle and Elrose were compared. The value of the location parameter was larger for Argyle than for Elrose (Tables 4.3.3 and 4.3.4). The value of the rate parameter for Argyle was smaller than that for Elrose. The quotient, time to 50% affected leaf area, was larger for Argyle than for Elrose. This indicates that the resistance in Argyle slowed

the progress of net blotch and lengthened time to 50% affected leaf area. The latter effect was partly negated by an increase of the location parameter.

To compare the effect of resistance and fungicide application on disease progress, parameter estimates for 1986 of the untreated control of Argyle and the repeatedly sprayed control of Elrose were compared. The value of the location parameter was smaller for the repeatedly sprayed control of Elrose than for the untreated control of Argyle (Tables 4.3.3 and 4.3.4). The value of the rate parameter was smaller for the untreated control of Argyle than for the repeatedly sprayed control of Elrose. The quotient, time to 50% affected leaf area, was larger for the untreated control of Argyle than for the repeatedly sprayed control of Elrose. To evaluate the effectiveness of fungicide application and resistance, time to 50% affected leaf area is compared to the harvest date (day 96; August 27, 1986). For Elrose, time to 50% affected leaf area was on day 63 (prior to harvest). For Argyle, time to 50% affected leaf area would be on day 93 (near harvest time), if the leaves had not senesced earlier. This indicates that the disease resistance in Argyle slowed the progress of percentage affected leaf area more than did the repeated application of Tilt on the susceptible cultivar Elrose.

4.3.3. Discussion

For percentage affected leaf area, the effect of fungicide application and resistance on disease progress were compared. Differences among curve parameters for the untreated control and the repeatedly sprayed control of Elrose showed that the fungicide slowed disease progress on a susceptible cultivar. Similar results were obtained in Ontario by Sutton and Steele (1983). In contrast, Metcalfe and Jones (1985) observed complete control of net blotch on winter barley with Tilt in England.

Differences among curve parameters for the untreated control of Argyle and Elrose showed that the resistance in Argyle slowed disease progress. Argyle has a moderate level of resistance to spot-type net blotch. Various levels of resistance, but no immune reaction, have been observed (Tekauz, 1985; 1986). The inheritance of the resistance to *P. teres* may be qualitative (Metcalfe et al., 1970) or quantitative (Douglas and Gordon, 1985).

The effectiveness of fungicide application and resistance was compared. Differences among curve parameters indicated that the resistance in Argyle was more effective than fungicide application on Elrose in slowing the progress of net blotch. Based on the data from this study, the best control of net blotch will be achieved through the use of resistant cultivars.

4.4. Effect of net blotch on barley

To measure the effect of net blotch on barley, treatments with different infection levels must be established. In this study, different infection levels were established by the use of various fungicide treatments and a resistant cultivar (Experiment III).

4.4.1. Percentage affected leaf area

In 1985, spot-type net blotch was the most prevalent foliar disease present on Elrose at Medstead and Shellbrook from the seedling stage onward. Traces of scald were observed at both sites. At Saskatoon, only traces of spot-type net blotch were observed. Net-type symptoms of net blotch were not observed at any site. Disease severity was assessed on the four uppermost leaves. Preliminary analysis indicated that the data collected for the penultimate leaf provided the best information for use in this study.

At Medstead and Shellbrook, the untreated control had the largest percentage affected leaf area on the penultimate leaf (Table 4.4.1). The repeatedly sprayed control had the lowest percentage affected leaf area. The double application of fungicide at growth stages 31 and 49 controlled net blotch almost to the same extent as the repeated application. For most observation days, the double application and the repeatedly sprayed control had similar percentage affected leaf area. The effect of a single fungicide application was variable. The early application did not differ from the

Table 4.4.1. Percentage affected leaf area of penultimate leaves infected with Pyrenophora teres for five treatments in Elrose at three sites in 1985.

Treatment[b]	Days from seeding[a]						
	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79
Medstead							
Repeated			4a		14c	23d		63c
Double			5a		16c	21cd		78b
Early			6a		21b	39b		96a
Late			7a		19bc	28c		79b
Untreated			6a		30a	64a		98a
Shellbrook								
Repeated	2a		12b		37b	66c		
Double	2a		14b		37b	73c		
Early	1a		14b		44b	91ab		
Late	2a		27a		42b	83b		
Untreated	2a		25a		61a	97a		
Saskatoon								
Repeated					0a			0a
Double					0a			0a
Early					0a			0a
Late					0a			0a
Untreated					0a			2a

[a] Means within a column and site followed by the same letter are not significantly different from each other at the 0.05 probability level according to Duncan's multiple range test.

[b] Repeated = Application of Tilt at 7 to 10 day intervals; Double = Application of Tilt at Zadoks growth stage 31 and 49; Early = Application of Tilt at Zadoks growth stage 31; Late = Application of Tilt at Zadoks growth stage 49; Untreated = No application of fungicide.

double application for the early readings of percentage affected leaf area. As the fungicide lost its effectiveness, percentage affected leaf area increased rapidly for the early application. The late application generally affected percentage affected leaf area to the same extent as the double application, and percentage affected leaf area was decreased, when compared to the untreated control. At Saskatoon, no differences were observed among the treatments for percentage affected leaf area.

In 1986, spot-type net blotch was the most prevalent foliar disease present on Elrose at Medstead and Shellbrook from the seedling stage onward. Traces of scald were observed at both sites. At Saskatoon, spot-type net blotch was observed from the elongation stage onward and stem rust severely affected Elrose after heading. Net-type symptoms of net blotch were not observed at any site.

Compared with 1985, the untreated control, the early, double and repeated fungicide application had similar effects on percentage affected leaf area (Table 4.4.2). The late application decreased percentage affected leaf area at Saskatoon, but not at Shellbrook and Medstead. At Shellbrook, the late application was made later than scheduled. At Medstead, differences may not have been significant due to insufficient replication.

At Medstead in 1986, spot-type net blotch was observed on Argyle from the seedling stage onward. After heading a severe scald epidemic developed. While traces of scald were

Table 4.4.2. Percentage affected leaf area of penultimate leaves infected with Pyrenophora teres for five treatments in Elrose at three sites in 1986.

Treatment[b]	Days from seeding[a]							
	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79
Medstead								
Repeated			-		-			
Double			2a		27b			
Early			1a		62a			
Late			3a		58a			
Untreated			6a		73a			
Shellbrook								
Repeated		1a	4c	5b	7c	13b	50c	
Double		1a	7bc	11b	14b	19b	69b	
Early		1a	5bc	12b	15b	27b	84ab	
Late		1a	14a	23a	33a	69a	97a	
Untreated		1a	12a	22a	39a	78a	98a	
Saskatoon								
Repeated						6b		41bc
Double						4b		29c
Early						1b		32c
Late						4b		57b
Untreated						15a		90a

[a] Means within a column and site followed by the same letter are not significantly different from each other at the 0.05 probability level according to Duncan's multiple range test.

[b] See Table 4.4.1.

observed at Shellbrook, spot-type net blotch was the most prevalent foliar disease present from the seedling stage onward. At Saskatoon, spot-type net blotch was the most prevalent disease from the elongation stage onward. Net-type symptoms of net blotch were not observed at any site. At all sites, no differences were observed among the treatments for percentage affected leaf area due to net blotch infection (Table 4.4.3).

The fungicide Tilt has been reported to cause a delay in the senescence of healthy leaves by decreasing the population of endophytic fungi in barley leaves (Riesen and Close, 1987; Tolstrup and Smedegaard-Petersen, 1984). In 1985, when very low levels of disease were observed at Saskatoon, no differences were observed among treatments in the senescence of leaves. Therefore, Tilt had no measurable effect on the senescence of healthy leaves under Saskatchewan conditions. At Medstead and Shellbrook, differences among treatments for leaf senescence were due to infection and disease control.

Application of Tilt provided only partial control of net blotch at Medstead and Shellbrook even when applied repeatedly (Tables 4.1.1 and 4.1.2). Sutton and Steele (1983) also observed only partial control, but Metcalfe and Jones (1985) reported complete control of net blotch with Tilt. These differences may be due to the use of winter barley grown in a different climate.

Table 4.4.3. Percentage affected leaf area of penultimate leaves infected with Pyrenophora teres for five treatments in Argyle at three sites in 1986.

Treatment[b]	Days from seeding[a]						
	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79
Medstead								
Repeated			-			-	-	
Double			1			5	10	
Early			1			7	12	
Late			1			9	11	
Untreated			1			9	21	
Shellbrook								
Repeated		0	1	2	2	3	7	23
Double		0	1	2	2	3	6	12
Early		0	1	2	2	3	7	14
Late		1	2	3	5	5	6	22
Untreated		1	2	3	4	6	13	26
Saskatoon								
Repeated						2		5
Double						1		8
Early						1		7
Late						2		5
Untreated						1		8

[a] Means within a column and site are not significantly different from each other at the 0.05 probability level.
[b] See Table 4.4.1.

4.4.2. Grain yield

4.4.2.1. Effect of fungicide application

Grain yield was determined for two cultivars and five spray schedules of the fungicide Tilt. For each combination of location, year and cultivar, an analysis of variance was performed and four contrasts were tested (Table 3.3.4). Mean squares and significances of contrasts are listed in Table 4.4.4. The value for the chi-square test for homogeneous error variances was 47.1. This value exceeded the right hand 0.95 point of the chi-square distribution with 8 degrees of freedom (15.5). Therefore, error variances were heterogeneous and the combined analysis of variance was not performed.

In 1985, the repeated fungicide application (C) had a major effect on grain yield of Elrose at Medstead and Shellbrook, but not at Saskatoon (Table 4.4.4). The early application (E) had an effect on grain yield only at Medstead. The late application (L) had a major effect on grain yield at Medstead and Shellbrook, but not at Saskatoon. The interaction between the early and late application (ExL) had no effect on grain yield at any site. The data show that in the absence of net blotch, the fungicide application did not result in a significant response in grain yield at Saskatoon. Thus, the effects on grain yield observed at Medstead and Shellbrook can be attributed to the control of net blotch by the fungicide.

In 1986, the repeated fungicide application had a large effect on grain yield of Elrose at Shellbrook, but not

Table 4.4.4. Analysis of variance for grain yield of Elrose at three sites in 1985 and 1986 and of Argyle at three sites in 1986.

Experiment	Mean square[a]				
	C	E	L	ExL	Error
1985 Elrose					
Medstead	1.372**	0.195*	0.483**	0.003	0.0394
Shellbrook	1.389**	0.061	0.433**	0.016	0.0228
Saskatoon	0.162	0.015	0.116	0.020	0.0455
1986 Elrose					
Medstead	-	0.866**	0.920**	0.001	0.0366
Shellbrook	5.307**	2.789**	0.308*	0.001	0.0398
Saskatoon	0.044	1.304**	1.110**	0.062	0.0198
1986 Argyle					
Medstead	-	0.088	1.761**	0.265	0.1449
Shellbrook	0.006	0.742*	0.046	0.059	0.1239
Saskatoon	0.002	0.058	0.135*	0.030	0.0247

*, ** Significant at the 0.05 and 0.01 probability level, respectively.

[a] C = Repeatedly sprayed control minus the average of the other treatments;
E = Average of the early application and double application minus the average of the late application and untreated control;
L = Average of the late application and double application minus the average of the early application and untreated control;
ExL = Average of the untreated control and double application minus the average of the early and late application.

at Saskatoon (Table 4.4.4). Both the early and late application had a major effect on grain yield at all three sites. The interaction between the early and late application had no effect on grain yield at all sites. At Saskatoon, effects of the early and late application were likely due to the presence of both net blotch and stem rust.

In 1986, a significant effect of the fungicide application on the grain yield of Argyle was obtained with the early application at Shellbrook and late application at Medstead and Saskatoon (Table 4.4.4). At Medstead, the fungicide was effective in controlling scald. At both Shellbrook and Saskatoon, the reason for the significant response in grain yield was not known as there were no differences among treatments for percentage affected leaf area.

At Saskatoon, none of the contrasts was significant for Elrose in 1985 (Table 4.4.4). As diseases were absent, differences in grain yield would reflect the effect of Tilt on healthy plants. The absence of significant treatment effects indicates that Tilt had no effect on grain yield. In healthy crops, Tilt increased grain yield significantly in Denmark (Tolstrup and Smedegaard-Petersen, 1984), but had no significant effect on grain yield in New Zealand (Riesen and Close, 1987).

No significance was observed for the interaction between the early and late application of Tilt (Table 4.4.4). This indicates that the effects of these applications are additive

in a statistical sense and that the yield increase associated with the application of Tilt at both stages is the sum of the yield increase associated with the application at either stage. The interaction between consecutive fungicide applications has received little attention in the literature. Very few studies possess the factorial design required for the analysis. Reported experiments for the net blotch-barley pathosystem that contained treatments forming a 2x2 factorial were reanalysed (Table 4.4.5). No significant interaction between consecutive applications of the fungicides propiconazole and prochloraz is evident. Therefore, there was no carry-over effect of the fungicides. The absence of any carry-over effect greatly simplifies the evaluation of spray schedules and the recommendation of spray schedules for disease control.

The treatment means are listed in Table 4.4.6 for each year, site and cultivar combination. The untreated control had the lowest yield at all sites in both years. Therefore, the fungicide increased grain yield for all significant contrasts.

4.4.2.2. Estimation of potential yield loss

In fungicide experiments, the potential yield loss is estimated as the difference between the disease-free treatment and the untreated control. In this study, the potential yield loss was estimated as the difference in grain yield between the repeatedly sprayed control and the

Table 4.4.5. Analysis of variance for grain yield for four fungicide treatments, forming a 2x2 factorial, based on data obtained from the literature for Pyrenophora teres on barley.

Source	Fungicide	Growth stage[b]	Mean square[a] E	L	ExL	Error	Error df
Meeus, 1982[c]	propiconazole+ carbendazim	32, 51	3.241+	14.902**	0.491	0.924	75
	prochloraz+ chlorothalonil	32, 51	2.370**	3.893**	0.273	0.142	80
Sutton and Steele, 1983	propiconazole+ seed treatment	21, 55	0.212+	2.496**	0.004	0.058[d]	15
Martin et al., 1981	propiconazole	24, 31	0.578**	1.649**	0.004	0.068[d]	30
		24, 39	0.536**	0.861**	0.008		
		24, 51	0.236+	1.166**	0.114		
		31, 39	1.508**	0.810**	0.014		
		31, 51	1.457**	1.631**	0.019		
		39, 51	0.412*	1.082**	0.143		

+, *, ** Significant at the 0.1, 0.05, 0.01 probability level, respectively.

[a] E = Average of the early and double application minus the average of the late application and untreated control; L = Average of the late and double application minus the average of the early application and untreated control; ExL = Average of the untreated control and double application minus the average of the early and late application.

[b] Growth stage, according to Zadoks et al. (1974) for fungicide application.

[c] Taken from Table VII and Table X, respectively.

[d] Mean square for error is estimated from differences among treatments.

Table 4.4.6. Mean grain yield for five treatments in Elrose at three sites in 1985 and 1986 and for five treatments in Argyle in 1986.

Treatment[b]	Elrose[a]						Argyle[a]		
	1985			1986			1986		
	Medst	Shell	Sask	Medst	Shell	Sask	Medst	Shell	Sask
	t/ha								
Untreated	1.85d	2.12d	4.47a	1.90c	2.89c	3.92c	3.28b	4.30b	4.63b
Early	2.00cd	2.27cd	4.58a	2.29b	3.56b	4.48b	3.61ab	4.79a	4.66b
Late	2.17bc	2.38bc	4.39a	2.30b	3.10c	4.45b	4.03a	4.49ab	4.71ab
Double	2.22b	2.58b	4.39a	2.67a	3.79b	4.81a	3.94a	4.75ab	4.88a
Repeated	2.60a	2.87a	4.64a	-	4.39a	4.51b	-	4.61ab	4.70ab

[a] Means within a column followed by the same letter are not significantly different from each other at the 0.05 probability level according to Duncan's multiple range test; Medst = Medstead; Shell = Shellbrook; Sask = Saskatoon.

[b] See Table 4.4.1.

untreated control of Elrose. At Shellbrook, the grain yield of the untreated control was 74 and 66% of the grain yield of the repeatedly sprayed control in 1985 and 1986, respectively (Table 4.4.6). At Medstead, the untreated control yielded 71% of the repeatedly sprayed control in 1985. Estimates of the potential yield loss were 26, 34 and 29% in 1985 at Shellbrook, in 1986 at Shellbrook and in 1985 at Medstead, respectively. These values are likely conservative, as the repeatedly sprayed control was infected with net blotch and probably yielded less than a healthy crop.

In cultivar experiments, the potential yield loss is estimated as the difference in grain yield between the immune cultivar and the susceptible cultivar. The grain yield of the resistant cultivar Argyle was calculated as the average of all treatments receiving at least one fungicide application. The yield of the untreated Elrose control was 62 and 49% of that of Argyle at Shellbrook and Medstead, respectively (Table 4.4.6). As Argyle yields 1% more than Elrose (Saskatchewan Agriculture, 1986b), estimates of the potential yield loss were 37 and 50% at Shellbrook and Medstead, respectively.

Estimates of the potential yield loss obtained from the fungicide experiments were similar to estimates reported by Khan (1987), Martin et al. (1981), and McDonald and Buchannon (1964). Estimates of the potential yield loss obtained from the cultivar experiments exceeded all previous estimates reported for field studies. If uncontrolled, infection with

net blotch can cause a yield loss of 50% on susceptible cultivars. This estimate indicates that net blotch can be a more serious problem than has previously been assumed.

4.4.3. Primary yield components

4.4.3.1. Effect of fungicide application

No differences among treatments were observed for the number of tillers at Medstead, Shellbrook and Saskatoon (Table 4.4.7). For the number of kernels per spike, significant differences were observed among treatments (Table 4.4.8), however, the differences were not consistent among treatments across sites and years. Significant differences among treatments were observed for kernel weight (Table 4.4.9).

For Elrose, differences among treatments were only due to net blotch at Medstead and Shellbrook in 1985 and 1986. At both sites in both years, the untreated control had the lowest kernel weight (Table 4.4.9). For the single early and late fungicide application, the kernel weight was higher than that for the untreated control. The double application generally increased kernel weight over that of a single application. The repeatedly sprayed control consistently had the highest kernel weight. The nature of the differences in kernel weight were similar to those for grain yield (Tables 4.4.6 and 4.4.9). Treatments with the highest grain yield had the highest kernel weight and treatments with a low grain yield had a low kernel weight.

Table 4.4.7. Mean number of tillers for five treatments in Elrose at three sites in 1985.

Treatment[b]	Site[a]		
	Medstead	Shellbrook	Saskatoon
	——— no./m^2 ———		
Untreated	644	598	927
Early	654	650	848
Late	642	649	849
Double	540	657	892
Repeated	680	707	828

[a] Means within a site are not significantly different from each other at the 0.05 probability level.
[b] See Table 4.4.1.

Table 4.4.8. Mean number of kernels per spike for five treatments in Elrose at three sites in 1985 and 1986 and for five treatments in Argyle in 1986.

	Elrose[a]						Argyle[a]		
	1985			1986			1986		
Treatment[b]	Medst	Shell	Sask	Medst	Shell	Sask	Medst	Shell	Sask
Untreated	21.3a	21.8a	18.7a	20.9a	21.5bc	21.6b	56.5ab	58.1a	58.2b
Early	20.8ab	21.8a	19.2a	20.2a	21.9ab	22.4ab	54.6b	59.5a	59.5ab
Late	19.7c	22.1a	18.7a	20.2a	21.0c	21.6b	58.3a	60.1a	60.1a
Double	20.0bc	22.2a	19.5a	21.2a	22.3a	22.6a	57.4ab	59.1a	59.1ab
Repeated	20.6abc	22.7a	18.8a	-	21.6abc	22.5a	-	59.3a	59.7ab

[a] Means within a column followed by the same letter are not significantly different from each other at the 0.05 probability level according to Duncan's multiple range test;
[b] Medst = Medstead; Shell = Shellbrook; Sask = Saskatoon.
See Table 4.4.1.

Table 4.4.9. Mean kernel weight for five treatments in Elrose at three sites in 1985 and 1986 and for five treatments in Argyle in 1986.

	Elrose[a]						Argyle[a]		
	1985			1986			1986		
Treatment[b]	Medst	Shell	Sask	Medst	Shell	Sask	Medst	Shell	Sask
	mg								
Untreated	26.3d	26.5d	49.4ab	24.8d	32.9e	34.9b	31.6c	39.4b	34.8a
Early	28.3c	29.1c	49.5a	26.2c	40.1c	36.3a	32.3bc	40.9a	34.5a
Late	29.6bc	29.9bc	48.4c	28.0b	35.4d	36.5a	33.5ab	40.4ab	34.4a
Double	30.3b	31.4b	48.5bc	29.5a	41.8b	36.5a	34.0a	40.9a	34.8a
Repeated	32.6a	34.0a	49.2abc	-	43.3a	35.8ab	-	40.9a	35.2a

[a] Means within a column followed by the same letter are not significantly different from each other at the 0.05 probability level according to Duncan's multiple range test; Medst = Medstead; Shell = Shellbrook; Sask = Saskatoon.

[b] See Table 4.4.1.

Martin (1985) observed differences in kernel weight only in one of his field experiments and differences in both kernel weight and number of tillers in another. Khan (1987) reported differences in number of tillers, number of kernels per spike and kernel weight in one field experiment. In contrast, Smedegaard-Petersen (1974) and Sutton and Steele (1983) reported that net blotch affected kernel weight only.

4.4.3.2. Relationship between grain yield and yield components

Grain yield can be expressed as a function of the primary yield components. The relationship between grain yield and the primary yield components is (Yoshida, 1972):

$$GY = NT * KS * KW, \qquad (4.4)$$

where GY represents the grain yield [mg/m^2], NT the number of tillers [N/m^2], KS the number of kernels per spike and KW the kernel weight [mg]. If grain yield is decreased due to disease by a factor W, then the product of the primary yield components is also decreased by the same factor W. If the number of tillers and the number of kernels per spike are constant, then differences in grain yield should be accompanied by similar differences in kernel weight. In formula form this is expressed as:

$$(W * GY) = (NT * KS) * (W * KW). \qquad (4.5)$$

At Shellbrook, differences in grain yield between the repeatedly sprayed control and the untreated control were 26 and 34% in 1985 and 1986, respectively, whereas differences in kernel weight were 19 and 24%, respectively

(Tables 4.4.6 and 4.4.9). At Medstead, the difference in grain yield between the repeatedly sprayed control and the untreated control was 29%, whereas the difference in kernel weight was 19% in 1985. This discrepancy may be due to the loss of small kernels during harvest, which may enlarge the differences in grain yield among treatments.

A discrepancy between the difference in grain yield and the difference in primary yield components has also been reported by other researchers for the effect of P. teres on barley. Sutton and Steele (1983) obtained differences in grain yield up to 26% and differences in kernel weight up to 11%. Neuhaus and Moritz (1986) obtained a difference in grain yield of 22% and a difference in kernel weight of 16%. In contrast, Khan (1987), Martin (1985) and Smedegaard-Petersen (1974) reported differences for grain yield and kernel weight of similar magnitude.

4.4.4. Grain quality

The assessment of grain quality was based on the grading system used by the Canadian Grain Commission. Grading is based on the visual appearance of the grain, its test weight, and the presence of admixtures and weed seeds (Agriculture Canada, 1986). Both Elrose and Argyle are eligible for malting grades. As shown in Table 4.4.10, all samples of Elrose were graded general purpose barley. The grain from Medstead and Shellbrook had stains on the hulls. These stains were likely due to infection with net blotch in 1985 and

Table 4.4.10. Grain grade for five treatments in Elrose at three sites in 1985 and 1986.

Treatment[a]	Grade	Test weight	Comments
1985 Medstead			
Untreated	2 CW	56.0	Light weight
Early	2 CW	57.3	Light weight
Late	1 CW	58.4	Stained
Double	1 CW	59.0	Stained
Repeated	1 CW	59.7	Stained
1985 Shellbrook			
Untreated	2 CW	57.7	Light weight
Early	1 CW	59.5	Stained
Late	1 CW	61.0	Stained
Double	1 CW	61.2	Stained
Repeated	1 CW	63.5	Stained
1985 Saskatoon			
Untreated	1 CW	73.8	Green
Early	1 CW	73.0	Green
Late	1 CW	74.0	Green
Double	1 CW	73.0	Green
Repeated	1 CW	73.0	Green
1986 Medstead			
Untreated	1 CW	58.8	Stained
Early	1 CW	58.8	Stained
Late	1 CW	61.0	Stained
Double	1 CW	63.3	Stained
1986 Shellbrook			
Untreated	1 CW	65.5	Stained
Early	1 CW	71.1	Stained
Late	1 CW	68.0	Stained
Double	1 CW	72.1	Stained
Repeated	1 CW	73.1	Stained
1986 Saskatoon			
Untreated	1 CW	68.6	Green
Early	1 CW	68.9	Green
Late	1 CW	68.9	Green
Double	1 CW	69.3	Green
Repeated	1 CW	68.5	Green

[a] See Table 4.4.1.

1986. In 1985, the untreated control at both Shellbrook and Medstead and the early fungicide application at Medstead had a test weight lower than 58.0 kg/hl and were thus graded 2 CW. The grain from Saskatoon contained green seeds which resulted from late tillers that were induced by heavy rainfall in late July.

Despite being mature, the grain of Argyle had a green colouration. For this reason it was assessed a general purpose grade (Table 4.4.11).

4.4.5. Discussion

Net blotch affected leaf blades, leaf sheaths, floral bracts and awns. On the susceptible cultivar Elrose, percentage affected leaf area increased rapidly in the untreated control. Repeated application of Tilt slowed progress of percentage affected leaf area, but did not provide complete disease control. The response to a single and double application of Tilt was usually significant, but showed a large temporal variation in percentage affected leaf area. Similar results were reported by Sutton and Steele (1983).

If uncontrolled, net blotch can cause losses in grain yield up to 50% in a susceptible cultivar. A single application of Tilt increased grain yield by 7 to 23% compared to the untreated control. Yield increases between 5 and 57% have been reported for a single application of Tilt (Martin et al., 1981; Sutton and Steele, 1983).

Table 4.4.11. Grain grade for five treatments in Argyle at three sites in 1986.

Treatment[a]	Grade	Test weight	Comments
Medstead			
Untreated	1 CW	61.7	Green
Early	1 CW	61.9	Green
Late	1 CW	62.3	Green
Double	1 CW	62.2	Green
Shellbrook			
Untreated	1 CW	66.6	Green
Early	1 CW	66.5	Green
Late	1 CW	67.0	Green
Double	1 CW	67.2	Green
Repeated	1 CW	67.7	Green
Saskatoon			
Untreated	1 CW	65.9	Green
Early	1 CW	65.8	Green
Late	1 CW	65.4	Green
Double	1 CW	66.0	Green
Repeated	1 CW	65.4	Green

[a] See Table 4.4.1.

Increased grain yield was associated with increased kernel weight. Differences in other yield components were not associated with fungicide treatments. Kernel infection and reduced test weight lowered grain grade and reduced the value of the harvested grain.

4.5. Relationship between disease severity and grain yield

The relationship between disease severity and grain yield has commonly been investigated with linear regression analysis. For this analysis it is assumed that the dependent variable is measured without error and that the data do not contain a special structure such as replication. In the literature, most experiments in which disease severity, grain yield and plant characters were assessed violated both assumptions. According to Wetherill (1986), the use of linear regression analysis is inappropriate when replications are present. Tukey (1951) recommended the analysis of variance and covariance components for this type of data. In this section, this method is used for the analysis of the relationship between disease severity and plant characters.

4.5.1. Relationship between independently measured variables

In section 4.4, all variables were analysed using a randomized complete block design. The linear model used was:

$$V_{ij} = \nu + \tau_i + \beta_j + \varepsilon_{ij}, \qquad (4.6)$$

where V_{ij} represents the observed value, ν the overall mean, τ_i the treatment effect, β_j the block effect, and ε_{ij} the

error effect. The mean for treatment i estimates the function: $\tau_i + \bar{\varepsilon}_{i.}$ and the mean for replication j estimates the function: $\beta_j + \bar{\varepsilon}_{.j}$. The mean square for the treatment effect is

$$r * var(\tau_i + \bar{\varepsilon}_{i.}) \tag{4.7}$$

and for the replication effect

$$t * var(\beta_j + \bar{\varepsilon}_{.j}). \tag{4.8}$$

For the analysis of variance it can be assumed that $cov(\tau, \varepsilon) = 0$ and $cov(\beta, \varepsilon) = 0$. Thus, the mean square for the treatment effect is

$$r * (var(\tau_i) + 1/r * var(\varepsilon_{ij})) \tag{4.9}$$

and for the replication effect

$$t * (var(\beta_j) + 1/t * var(\varepsilon_{ij})). \tag{4.10}$$

These formulas can be simplified to

$$var(\varepsilon_{ij}) + r * var(\tau_i) \text{ and} \tag{4.11}$$

$$var(\varepsilon_{ij}) + t * var(\beta_j), \tag{4.12}$$

respectively.

The analysis of variance and covariance components is an extension of the analysis of variance and analogous to the analysis of genetic variance components used in quantitative genetics. The theory will be developed for two variables (U and V). Based on the analysis of variance for a randomized complete block design, the linear model for U is:

$$U_{ij} - \mu = \theta_i + \rho_j + \eta_{ij}. \tag{4.13}$$

where U_{ij} represents the value for observation ij, μ the overall mean, θ_i the effect of treatment i, ρ_j the effect of replication j, and η_{ij} the error term associated with

observation ij. The linear model for V is:

$$V_{ij} - \nu = \tau_i + \beta_j + \varepsilon_{ij}. \qquad (4.14)$$

where V_{ij}, ν, τ_i, β_j and ε_{ij} are as described previously. From the linear models, a table of mean squares and mean products can be constructed (Table 4.5.1). The covariances between U and V are included for all sources of variation. These components are based on the usual assumptions of the analysis of variance, as well as the assumption that the treatment effect of Y is influenced only by the corresponding treatment effect of X. The latter must also apply to the replication and error effects. These assumptions set $\mathrm{cov}(\theta, \beta)$, $\mathrm{cov}(\theta, \varepsilon)$, $\mathrm{cov}(\rho, \tau)$, $\mathrm{cov}(\rho, \varepsilon)$, $\mathrm{cov}(\eta, \tau)$ and $\mathrm{cov}(\eta, \beta)$ at zero. Thus, only estimates of $\mathrm{cov}(\theta, \tau)$, $\mathrm{cov}(\rho, \beta)$ and $\mathrm{cov}(\eta, \varepsilon)$ are of interest. These components can be estimated from the equations derived from the mean squares and the mean products (Table 4.5.2). A regression coefficient and a coefficient of determination can be estimated for each source of variation, using the estimates for the variances and the covariance. The significance of these coefficients must be determined from their confidence interval (Tukey, 1951).

The separation of effects based on fixed and random model has no impact on the arithmetic of the estimation of (co)variance components in a randomized complete block design. $\mathrm{Var}(\theta_i)$, $\mathrm{cov}(\theta_i, \tau_i)$ and $\mathrm{var}(\tau_i)$ represent $\mathrm{sum}(\theta_i^2)/(t-1)$, $\mathrm{sum}(\theta_i * \tau_i)/(t-1)$ and $\mathrm{sum}(\tau_i^2)/(t-1)$, respectively in the case of a fixed model; σ_θ^2, $\sigma_{\theta\tau}$ and σ_τ^2

Table 4.5.1. Analysis of variance and covariance for a randomized complete block design.

Source	df[b]	Mean square/product[a] U,U	U,V	V,V
Treatment	t-1	$r*var(\theta_i+\overline{\eta}_{i.})$	$r*cov(\theta_i+\overline{\eta}_{i.},\tau_i+\overline{\varepsilon}_{i.})$	$r*var(\tau_i+\overline{\varepsilon}_{i.})$
Replication	r-1	$t*var(\rho_j+\overline{\eta}_{.j})$	$t*cov(\rho_j+\overline{\eta}_{.j},\beta_j+\overline{\varepsilon}_{.j})$	$t*var(\beta_j+\overline{\varepsilon}_{.j})$
Error	(t-1)(r-1)	$var(\eta_{ij})$	$cov(\eta_{ij}, \varepsilon_{ij})$	$var(\varepsilon_{ij})$

[a] θ_i = treatment effect for U; ρ_j = replication effect for U; η_{ij} = error effect for U; τ_i = treatment effect for V; β_j = replication effect for V; ε_{ij} = error effect for V.

[b] t = number of treatments; r = number of replications.

Table 4.5.2. Analysis of variance and covariance for a randomized complete block design.

Source	df[a]	Mean square/product[a]		
		U,U	U,V	V,V
Treatment	t-1	$\text{var}(\eta_{ij})+r*\text{var}(\theta_i)$	$\text{cov}(\eta_{ij},\varepsilon_{ij})+r*\text{cov}(\theta_i,\tau_i)$	$\text{var}(\varepsilon_{ij})+r*\text{var}(\tau_i)$
Replication	r-1	$\text{var}(\eta_{ij})+t*\text{var}(\rho_j)$	$\text{cov}(\eta_{ij},\varepsilon_{ij})+t*\text{cov}(\rho_j,\beta_j)$	$\text{var}(\varepsilon_{ij})+t*\text{var}(\beta_j)$
Error	(t-1)(r-1)	$\text{var}(\eta_{ij})$	$\text{cov}(\eta_{ij},\varepsilon_{ij})$	$\text{var}(\varepsilon_{ij})$

[a] See Table 4.5.1.

respectively in the case of a random model. The estimated values for the regression and correlation coefficient are the same for either model. The interpretation depends on the model used.

4.5.2. Correlation

The analysis of variance and covariance components described in section 4.5.1 was performed with the subroutine A2ALD (IMSL, 1987). The analysis was performed on the data collected at Shellbrook in 1985 and 1986 for kernel weight, grain yield and percentage affected leaf area showing significant treatment differences. The correlation coefficients for each source of variation can be separated into three groups:

1. a correlation coefficient between kernel weight and grain yield,
2. correlation coefficients between the percentages affected leaf area observed on different days and
3. correlation coefficients between kernel weight or grain yield and percentage affected leaf area observed on different days.

For the treatment effect, the correlation coefficient between kernel weight and grain yield was 0.99 in both 1985 and 1986 (Tables 4.5.3 and 4.5.4). This indicates that the treatments affected the kernel weight and grain yield in a similar manner in both years, confirming the results discussed in Section 4.4.3.

Table 4.5.3. Correlation coefficients for treatment, replication and error effect between kernel weight, grain yield, and percentage affected leaf area, observed on different days, at Shellbrook in 1985.

Trait[a]	Weight	Yield	Day54	Day63
Treatment				
Yield	0.99			
Day54	-0.78	-0.82		
Day63	-0.99	-0.93	0.95	
Day71	-1.01[b]	-0.99	0.70	0.91
Replication				
Yield	0.78			
Day54	-0.99	-1.09		
Day63	-0.30	-0.57	0.99	
Day71	-1.21	-1.10	1.60	0.56
Error				
Yield	0.18			
Day54	0.16	-0.05		
Day63	0.05	0.03	0.24	
Day71	-0.35	-0.32	-0.01	0.16

[a] Weight = Kernel weight; Yield = Grain yield; Day54, Day63, Day71 = Percentage affected leaf area, observed 54, 63 and 71 days after seeding, respectively.

[b] r-values larger than 1.00 indicate sampling fluctuation (Tukey, 1951).

Table 4.5.4. Correlation coefficients for treatment, replication and error effect between kernel weight, grain yield and percentage affected leaf area observed on different days, at Shellbrook in 1986.

Trait[a]	Weight	Yield	Day49	Day54	Day59	Day62	Day69
Treatment							
Yield	0.99						
Day49	-0.96	-0.96					
Day54	-1.02[b]	-0.96	0.77				
Day59	-1.08	-0.98	0.80	1.13			
Day62	-1.01	-0.98	0.94	0.99	1.08		
Day69	-1.04	-0.94	0.89	1.02	1.06	1.01	
Day74	-0.94	-1.00	0.90	0.86	0.99	0.91	0.91
Replication							
Yield	-[c]						
Day49	-	-					
Day54	-3.53	-	-				
Day59	-	-	-	-			
Day62	-	-	-	-	-		
Day69	-	-	-	-	-	-	
Day74	-	-	-	-	-	-	-
Error							
Yield	-0.60						
Day49	0.06	-0.44					
Day54	-0.07	-0.59	0.33				
Day59	0.18	-0.53	0.03	0.65			
Day62	-0.07	-0.50	0.57	0.70	0.02		
Day69	0.32	-0.80	0.55	0.37	0.32	0.52	
Day74	-0.36	-0.28	0.30	0.70	0.02	0.90	0.25

[a] Weight = Kernel weight; Yield = Grain yield; Day49, Day54, Day59, Day62, Day69, Day74 = Percentage affected leaf area, observed 49, 54, 59, 62, 69 and 74 days after seeding, respectively.

[b] r-values larger than 1.00 indicate sampling fluctuation (Tukey, 1951).

[c] - = Estimate of variance for one of the variables was negative.

For the treatment effect, the correlation coefficient between percentages affected leaf area observed on different days ranged from 0.70 to 1.13 (Tables 4.5.3 and 4.5.4). This indicates that the effect of treatments was reflected in the percentage affected leaf area observed on the penultimate leaf. The correlation coefficient between percentages affected leaf area observed on different days was rather variable because treatments with a single fungicide application were included. The effect of a single fungicide application on the percentage affected leaf area varied over observation days (Section 4.4.1).

For the treatment effect, the correlation coefficients between kernel weight and percentage affected leaf area ranged from -0.78 to -1.08 (Tables 4.5.3 and 4.5.4). The correlation coefficients between grain yield and percentage affected leaf area ranged from -0.82 to -1.00 (Tables 4.5.3 and 4.5.4). This inverse relationship indicates that a change in percentage affected leaf area was accompanied by a change in the opposite direction in grain yield or kernel weight. The range observed for the correlation coefficient indicates that percentage affected leaf area can be used to predict differences in kernel weight or grain yield.

For the replication effect, the correlation coefficients showed a pattern similar to that observed for the treatment effect in 1985 (Table 4.5.3). In 1986, only a few correlation coefficients could be estimated, because the estimates for many variance components were negative (Table 4.5.4). As the

source for the variation among replications is unknown, the correlation coefficients cannot be interpreted.

For the error effect, the correlation coefficients were generally smaller than 0.80 (Tables 4.5.3 and 4.5.4). This indicates that the error components of different variables were independent.

4.5.3. Regression

Yield loss-disease severity models are used to predict the percentage yield loss from the observed disease severity. Commonly used analyses to develop these models are the critical point and multiple point analysis. For practical reasons, critical point analysis is preferred (Section 2.4), but is inappropriate for data obtained from replicated experiments. In such instances, the analysis of variance and covariance components can be used as a modified critical point analysis. In this analysis, disease severity observed on one day is regressed on percentage yield loss using analysis of variance and covariance components.

In 1985, the coefficient of determination for the regression of percentage affected leaf area on percentage yield loss increased with days from seeding (Table 4.5.5). In 1986, the coefficient of determination showed little variation. This indicates that in 1985, the relationship between percentage yield loss and percentage affected leaf area was stronger later in the growing season. In 1986, the relationship between percentage yield loss and percentage

Table 4.5.5. Regression coefficients and coefficients of determination for regression of treatment effects of percentage affected leaf area on percentage yield loss at Shellbrook in 1985 and 1986.

Days from seeding	1985		1986	
	b	r^2	b	r^2
49	.	.	6.92	0.93
54	1.22	0.64	2.94	0.92
59	.	.	2.75	0.96
62	.	.	0.91	0.95
63	0.95	0.84	.	.
69	.	.	0.40	0.88
71	0.82	1.00	.	.
74	.	.	0.65	0.99

Within each replication grain yield of the repeatedly sprayed control was taken as 100%. For each treatment, percentage yield loss was determined for each replication. The obtained values were then used in the analysis of variance and covariance components.

affected leaf area was similar for all observation days.

In both 1985 and 1986, the regression coefficient for the regression of percentage affected leaf area on percentage yield loss decreased with days from seeding (Table 4.5.5). This decrease was due to an increase in the range observed for percentage affected leaf area on later observation days (Tables 4.4.1 and 4.4.2). The estimate for the regression coefficient showed little variation between 62 and 74 days after seeding. The average of these regression coefficients (0.93) may thus be used as an initial estimate for the regression coefficient for the regression of percentage affected leaf area on percentage yield loss. For each increase of 1% in disease severity measured as percentage affected leaf area, a corresponding increase in yield loss of 0.93% is expected.

The estimated regression coefficient (0.93) was used to evaluate the accuracy of the prediction of percentage yield loss based on the observed percentage affected leaf area (Table 4.5.6). Grain yield of the repeatedly sprayed control was taken as 100%. The actual percentage yield loss was calculated from the data on grain yield shown in Table 4.4.6. The predicted percentage yield loss was calculated as the difference in percentage affected leaf area between the particular treatment and the repeatedly sprayed control multiplied by 0.93. The data on percentage affected leaf area were taken from Tables 4.4.1 and 4.4.2. In 1985, the predicted value closest to the actual percentage yield loss

Table 4.5.6. The actual and predicted percentage yield losses in Elrose for four treatments in 1985 and 1986.

	1985			1986			
		Predicted[a]			Predicted[a]		
Treatment[b]	Actual	Day63	Day71	Actual	Day62	Day69	Day74
Untreated	26	22	29	34	30	60	45
Early	21	7	23	19	8	13	32
Late	17	4	16	20	26	52	44
Double	10	0	7	14	7	6	18

[a] The number of days after seeding
[b] Untreated = No application of fungicide; Early = Application of Tilt at Zadoks growth stage 31; Late = Application of Tilt at Zadoks growth stage 49; Double = Application of Tilt at Zadoks growth stage 31 and 49.

was obtained 71 days after seeding for all treatments. In 1986, the predicted value closest to the observed percentage yield loss was obtained 62 days after seeding for the untreated control and the late application, 69 days after seeding for the early application and 74 days after seeding for the double application. All these predicted values were relatively close to the observed values, indicating that 0.93 was a reasonable estimate for the prediction of percentage yield loss in those instances. However, in some instances, an estimate of 0.93 was not applicable. Therefore, caution should be taken in forecasting of percentage yield loss.

4.5.4. Discussion

The coefficient of determination indicated that a strong relationship exists between the treatment effect of percentage yield loss and percentage affected leaf area. Percentage affected leaf area may thus be used to predict percentage yield loss.

Yield loss-disease severity models are developed to estimate the yield loss caused by a pathogen in a regional survey (James, 1974). For this purpose, only critical point models can be employed due to excessive costs associated with the use of other models. A modified critical point model was developed. The relationship between the percentage yield loss and percentage affected leaf area was:

$$Y = 0.93 * X \text{ on the penultimate leaf} \qquad (5.15)$$

where Y represents the percentage yield loss and X the

percentage affected leaf area between 63 and 74 days after seeding.

Validation of the regression coefficient revealed discrepancies between observed and predicted values for percentage yield loss. The discrepancies were likely due to temporal variation in the rate of disease progress caused by single fungicide application. To reduce the temporal variation in the rate, fungicides should be applied on a regular basis. As critical point models have generally been developed using single or double fungicide applications (Khan, 1987; Martin, 1985), their applicability is probably limited.

5. GENERAL DISCUSSION

5.1. Infection cycle

In host-pathogen interaction, the infection cycle is defined as the process from sporulation through dissemination and infection to sporulation (Zadoks and Schein, 1979). For spot-type isolates of Pyrenophora teres, the periodicity of the infection cycle has not been reported previously. From this study, the periodicity of the infection cycle can be deduced. Sporulation occurs on infected stubble or infected leaves. Conidia are produced during the night and disseminated during the day. Infection then occurs during an ensuing period, when leaves are wetted by dew or precipitation. During the leaf wetness period, time to infection is six to eight hours at 10 ^{o}C. Following infection, symptoms develop and sporulation occurs once more than 50% of the leaf tissue has senesced. The period between the first appearance of visible symptoms and the onset of sporulation takes from 14 to 20 days. Since the life span of the leaves ranges from 20 to 30 days, only one infection cycle can be completed for each leaf.

At Shellbrook, in 1986 and 1987, the combinations of temperature and leaf wetness period were frequently suitable for infection. At night, temperatures ranged from 0 to 15 ^{o}C and leaf wetness periods regularly exceeded six hours. These conditions were likely sufficient for sporulation and

infection. These conditions are typical for most of the northern grainbelt in Saskatchewan and severe epidemics of net blotch can thus be expected throughout this area.

5.2. Sources of inoculum

Sources of primary inoculum of *P. teres* may be infected seed or infected crop debris. The incidence of seed infection generally appears to be low (Hewett, 1975; Machacek et al., 1951), but may be as high as 90% (Sheridan, 1977). The relationship between seed infection and seedling infection is not clear. Sometimes, incidence of seed infection and that of seedling infection are similar (Jørgensen, 1980), but at other times, incidence of seed infection is much higher than that of seedling infection (Machacek and Wallace, 1942). In New Zealand, infected seed is considered to be the most important source of primary inoculum (Sheridan et al., 1983).

Infected crop debris is an alternate source of primary inoculum. In Israel, the accumulation of infected crop debris increased net blotch severity (Kenneth, 1962). Under current cropping regimes in Saskatchewan, crop debris is abundant in farm fields. As most barley fields are infected with net blotch (Tekauz, 1978), crop debris is likely the most important source of primary inoculum in Saskatchewan.

In this study, infected crop debris was the major source of primary inoculum. Secondary inoculum was produced on infected leaves of the growing crop only when the leaves senesced. Infected crop debris was the source of inoculum for

the lower leaves. Infected crop debris and senesced lower leaves were the sources of inoculum for the upper leaves. Thus, net blotch can probably reach epidemic proportions only when large amounts of primary inoculum are present.

5.3. Effect of net blotch on barley

Net blotch reduces grain yield in barley, but crop failure has never been attributed to net blotch (Shipton et al., 1973). In a susceptible cultivar, severe epidemics caused by net-type isolates of P. teres have been reported to cause yield losses up to 27% in Western Canada (McDonald and Buchannon, 1964) and losses up to 40% elsewhere (Khan, 1987; Martin et al., 1981) . Estimates from the current study showed that severe epidemics caused by spot-type isolates can cause yield losses up to 50%. This estimate suggests that the newly established spot-type isolates can be a more important constraint to barley production than indigenous net-type isolates. However, in a moderately resistant cultivar, spot-type isolates had no effect on grain yield.

In a susceptible cultivar, reduced grain yield due to infection of P. teres was associated with reduced kernel weight. In addition, net blotch infection affected quality resulting in lower grain grade due to low test weight and discolouration. Lower grades result in a lower price and therefore, reduced producer income.

In this study, strong correlations were observed between treatment effects on percentage yield loss and disease

severity on the penultimate leaf for several observation days. Accordingly, a modified critical point model was developed for disease severity and percentage yield loss. The model provided an accurate prediction for about half of the estimates. However, some predicted values differed markedly from the actual values. This may have been due to the effect of a single fungicide application on the rate of disease progress. The rate of disease progress showed a large temporal variation due to a single fungicide application, as was also shown by Sutton and Steele (1983). To obtain a consistent response, fungicides should be applied at regular intervals. Different disease progress curves may be obtained by the use of different fungicide rates (Johnson and Beute, 1986). An estimate of the percentage yield loss from disease severity using the modified critical point model that was developed, would require a repeatedly sprayed control to be present as a reference point. This would make the model impractical.

5.4. Disease control

Net blotch of barley like many other diseases can be controlled by means of sanitation, rotation, fungicides or host resistance (Zadoks and Schein, 1979). Sanitation involves the removal of crop debris from the field. Burying of debris by deep ploughing and straw burning have been suggested to control net blotch (Jordan and Allen, 1984). However, these practices cannot be recommended for

Saskatchewan, as they dramatically increase the potential for soil erosion.

Rotation is effective as a means to avoid epidemics of many diseases, including net blotch (Zadoks and Schein, 1979). In Saskatchewan, narrow rotations are being used (Saskatchewan Agriculture, 1984) because only few crops are commonly grown. There is even a tendency towards monoculture, as cereal rotations may lead to undesirable levels of admixtures, which in turn lead to downgrading.

Control of net blotch through the use of fungicides has received considerable attention. Significant increases in grain yield have been obtained with seed treatment (Martin, 1985) and foliar application (Meeus, 1982; Sutton and Steele, 1983). Seed treatment controls net blotch when infected seed is the source of primary inoculum (Martin, 1985), but may not be effective when infected crop debris is the source of primary inoculum (Berger, 1977). Foliar fungicide application can control net blotch whether infected seed (Sheridan et al., 1983) or infected stubble is the source of primary inoculum. In Saskatchewan, where infected crop debris is the most important source of primary inoculum, only foliar fungicide application would likely provide adequate control. In this study, a single application of Tilt increased grain yield from 7 to 23%. At a price for barley of C$ 110.00 per tonne, this yield increase would not be sufficient to recover the cost of fungicide application in Saskatchewan (Van den Berg et al., 1986).

Resistance has long been the mainstay for disease control in cereals in Western Canada. In this study, the moderate level of resistance present in Argyle provided adequate protection against net blotch. At present, considerable effort is directed towards breeding for resistance to net blotch. The objective is to release only adapted cultivars that have at least some resistance to net blotch (MR-MS on the scale of Tekauz, 1985) (B.G. Rossnagel, pers. comm.).

5.5. Net blotch in Saskatchewan: Prospects

Soil erosion is a very important agricultural concern in Saskatchewan. To reduce soil erosion, practices such as reduced tillage, stubble cropping and continuous cropping have been introduced. These practices reduce soil erosion through the presence of crop debris on the soil surface. However, these practices have likely increased the potential for net blotch epidemics.

A large acreage in the northern grainbelt of Saskatchewan is planted to barley annually. Weather conditions are usually wet and cool, favouring the development of net blotch. Also, there are large amounts of primary inoculum present. As a result, significant yield losses can be expected whenever a susceptible cultivar is grown in a narrow rotation. To prevent severe epidemics, rotations should be extended wherever possible. Ideally, a 4-year rotation of barley-canola-wheat-flax/legume should be

used. At current grain prices, the use of foliar fungicides is not economically feasible. This particularly true, since the best fungicides currently available provide only partial control of net blotch and do not reduce the amount of primary inoculum, thereby perpetuating the problem. In this situation, the use of resistant cultivars is the best means to control net blotch.

In the southern part of the grainbelt of Saskatchewan, a smaller acreage is planted to barley. Weather conditions are usually warm and dry, and are therefore generally less favourable for the development of net blotch. No severe epidemics have been reported from this area and crop rotation may be sufficient to control net blotch. However, the data from this study show that time to infection is shorter for spot-type isolates than that of the net-type isolates. Therefore, the chances for the development of epidemics in this region may increase, because leaf wetness periods of shorter duration are sufficient for infection. Continued use of narrow rotations may increase the amount of primary inoculum to a level that may cause substantial damage to a susceptible cultivar. If so, rotations in this region should be extended and resistant cultivars should be used. In addition, spot-type isolates might establish themselves in areas that were previously unfavourable for the disease due to insufficient leaf wetness periods for net-type isolates, thereby increasing the prevalence of net blotch in Saskatchewan.

5.6. Suggestions for future research

Future research should focus on the topics of disease establishment, dispersal of conidia of *P. teres* and field resistance in barley. Disease establishment has received some attention (Shaw, 1986; Singh, 1963a; 1963b). Temperatures generally ranged from 15 to 35 ^{o}C and leaf wetness periods generally exceeded 24 hours in these studies. In Saskatchewan, infection was shown to occur mainly during the night when the temperature ranged from 0 to 15 ^{o}C and the leaf wetness period rarely exceeded 10 hours. Disease development under these conditions has not been reported previously. In order to evaluate disease progress in the field, more research is necessary using local conditions.

Airborne conidia of net-type isolates of *P. teres* can be transported over 30 m (Martin and Clough, 1984), but rarely travel more than 6 m (Piening, 1968). At Shellbrook, uniform infection with spot-type isolates was observed in a 20 m wide strip of barley that was adjacent to the infected stubble. At Medstead, uniform infection was also observed in a commercial field of Harrington barley seeded on fallow, but adjacent to a field with barley stubble. These observations suggest that inoculum from outside sources may be more important than suggested in the literature.

Breeding for resistance to net blotch has received extensive attention worldwide (Ghobrial et al., 1981; Khan, 1971; Metcalfe et al., 1978; Singh and Singh, 1978). The definition of resistance varied considerably among reports.

Resistance to net blotch appears to be based on reduced lesion growth or reduced sporulation (Keeling and Banttari, 1975). No systematic research has been conducted to determine the level of resistance that provides adequate protection in the field.

REFERENCES

Agriculture Canada. 1986. Grain grading handbook for Western Canada: Effective August 1, 1986. Agriculture Canada. Canadian Grain Commission. Winnipeg. Manitoba. Canada.

Amelung, D. 1985. Epidemiologie, Befallsentwicklung und Schadwirkung der Netzfleckenkrankheit der Gerste, verursacht durch Drechslera teres (Sacc.) Shoemaker. Nachrichtenbl. Pflanzenschutz DDR 39: 49-52.

Barrault, G., B. Al-Ali, M. Petitprez, and L. Albertini. 1982. Contribution à l'étude de l'activité toxique de l' Helminthosporium teres, parasite de l'orge (Hordeum vulgare). Can. J. Bot. 60: 330-339.

Bashi, E., and J. Rotem. 1974. Adaptation of four pathogens to semi-arid habitats as conditioned by penetration rate and germinating spore survival. Phytopathology 64: 1035-1039.

Bartlett, M.S. 1949. Fitting a straight line when both variables are subject to error. Biometrics 5: 207-212.

Berg, C.G.J. van den, G.R. Hughes, and B.G. Rossnagel. 1986. Economics of disease control in cereals. p. 79-89. In: Crop disease and insect update. November 4-6, 1986. Univ. of Saskatchewan. Saskatoon. Saskatchewan. Canada.

Berger, R.D. 1977. Application of epidemiological principles to achieve plant disease control. Annu. Rev. Phytopathol. 15: 165-183.

Box, G.E.P. 1966. Use and abuse of regression. Technometrics 8: 625-629.

Burger, W.C. and D.E. LaBerge. 1985. Malting and brewing quality. In D.C. Rasmusson (ed.). Barley. Agronomy 26: 367-402.

Burleigh, J.R., and M.J. Loubane. 1984. Plot size effects on disease progress and yield of wheat infected by Mycosphaerella graminicola and barley infected by Pyrenophora teres. Phytopathology 74: 545-549.

Chiarappa, L. 1981. Crop loss terminology. p. 115-123. In L. Chiarappa (ed.). Crop loss assessment methods. Supplement 3. FAO. Commonwealth Agricultural Bureaux. Farnham Royal. Slough. UK.

Chidambaram, P., S.B. Mathur, and P. Neergaard. 1973. Identification of seed-borne Drechslera species. Friesia 10: 165-207.

Coakley, S.M. 1985. Describing and quantifying the environment. Plant Dis. 69: 461-466.

Commonwealth Mycological Institute. 1977. Pathogen: Pyrenophora teres Drechsl. Distribution maps of plant diseases no. 364. 3rd edition. Commonwealth Mycological Institute. Kew. Surrey. UK.

Cook, R.J. 1981. Unexpected effects of fungicides on cereal yields. EPPO Bull. 11: 277-285.

Couture, L., and J.C. Sutton. 1978. Relation of weather variables and host factors to incidence of airborne spores of Bipolaris sorokiniana. Can. J. Bot. 56: 2162-2170.

Douglas, G.B., and I.L. Gordon. 1985. Quantitative genetics of net blotch resistance in barley. N. Z. J. Agric. Res. 28: 157-164.

Drechsler, C. 1923. Some graminicolous species of Helminthosporium. J. Agric. Res. 24: 641-740.

Ellis, M.B., and J.M. Waller. 1973. Pyrenophora teres (conidial state: Drechslera teres). Descriptions of pathogenic fungi and bacteria no. 390. Commonwealth Mycological Institute. Kew. Surrey. UK.

Evans, E. 1977. Efficient use of systemic fungicides. p. 198-212. In R.W. Marsh (ed.). Systemic fungicides. Longman Group Ltd. London. UK.

Garg, I.D., and C.L. Mandahar. 1976. Effect of net blotch infection on the physiology of barley: carbohydrate and nitrogen metabolism. Indian J. Exp. Biol. 14: 66-68.

Gaunt, R.E. 1980. Physiological basis of yield loss. p. 98-111. In P.S. Teng and S.V. Krupa (eds.). Crop loss assessment. Proc. E.C. Stakman Commemorative Symposium. Dept. of Plant Pathol. Univ. of Minnesota. Minn. USA.

Gaunt, R.E. 1987. A mechanistic approach to yield loss assessment based on crop physiology. p. 150-159. In P.S. Teng (ed.). Crop loss assessment and pest management. APS Press. St. Paul. MN. USA.

Ghobrial, E., I. Shafik, and A.M. Hammouda. 1981. Sources of resistance to barley helminthosporiosis. Barley Newsletter 25: 64-67.

Gillespie, T.J., and G.E. Kidd. 1978. Sensing duration of leaf moisture retention using electrical impedance grids. Can. J. Plant Sci. 58: 179-187.

Graaff, N.A. van der. 1981. Increasing reliability of crop loss information: the use of 'indirect' data. p. 65-72. In L. Chiarappa (ed.). Crop loss assessment methods. Supplement 3. FAO. Commonwealth Agricultural Bureaux. Farnham Royal. Slough. UK.

Greaney, F.J., and J.E. Machacek. 1946. The prevalence and control of seedborne diseases of cereals in Manitoba. Scientific Agriculture 26: 59-78.

Hampton, J.G., and B.J. Arnst. 1978. The relationship between net blotch and yield loss in spring barley. p. 18-1 to 18-4. In R.C. Close, I.C. Harvey, F.R. Sanderson, R.E. Gaunt, A.J. McCully, and P.S. Teng (eds.). 1978. Epidemiology and crop loss assessment. Proc. APPS Workshop. August 1977. Lincoln College. Canterbury. New Zealand.

Hau, B., and J. Kranz. 1977. Ein Vergleich verschiedener Transformationen von Befallskurven. Phytopathol. Z. 88: 53-68.

Hewett, P.D. 1975. A health survey of seed barley. Plant Pathol. 24: 229-232.

Hirst, J.M. 1952. An automatic volumetric spore trap. Ann. Appl. Biol. 39: 257-265.

Horsfall, J.G., and E.B. Cowling. 1978. Pathometry: the measurement of plant disease. p. 120-136. In J.G. Horsfall, and E.B. Cowling (eds.). Plant disease - an advanced treatise. Vol. II. How disease develops in populations. Acacemic Press. New York. NY. USA.

Huisman, O.C. 1982. Interrelations of root growth dynamics to epidemiology of root-invading fungi. Annu. Rev. Phytopathol. 20: 303-327.

IMSL. 1987. User's manual STAT/LIBRARY: Fortran subroutines for statistical analysis. IMSL Inc. Houston. TX. USA.

James, W.C. 1969. A survey of foliar diseases of spring barley in England and Wales in 1967. Ann. Appl. Biol. 63: 253-263.

James, W.C. 1971. An illustrated series of assessment keys for plant diseases: their preparation and usage. Can. Plant Dis. Surv. 51: 39-65.

James, W.C. 1974. Assessment of plant diseases and losses. Annu. Rev. Phytopathol. 12: 27-48.

James, W.C., J.E. Jenkins, and J.L. Jemmett. 1968. The relationship between leaf blotch caused by Rhynchosporium secalis and losses in grain yield of spring barley. Ann. Appl. Biol. 62: 273-288.

James, W.C., and C.S. Shih. 1973. Size and shape of plots for estimating yield losses from cereal foliage diseases. Exp. Agric. 9: 63-71.

James, W.C., C.S. Shih, W.A. Hodgson, and L.C. Callbeck. 1972. The quantitative relationship between late blight of potato and loss in tuber yield. Phytopathology 62: 92-96.

James, W.C., C.S. Shih, W.A. Hodgson, and L.C. Callbeck. 1976. Representational errors due to interplot interference in field experiments with late blight of potato. Phytopathology 66: 695-700.

James, W.C., and P.S. Teng. 1979. The quantification of production constraints associated with plant diseases. p. 201-267. In T.H. Coaker (ed.). Applied Biology. Vol. IV. Academic Press. London. UK.

Jeger, M.J. 1982. The relation between total, infectious, and postinfectious diseased plant tissue. Phytopathology 72: 1185-1189.

Jenkyn, J.F. 1981. Experimental design and interplot interference. p. 35-42. In L. Chiarappa (ed.). Crop loss assessment methods. Supplement 3. FAO. Commonwealth Agricultural Bureaux. Farnham Royal. Slough. UK.

Johnson, C.S., and M.K. Beute. 1986. The role of partial resistance in the management of Cercospora leaf spot of peanut in North Carolina. Phytopathology 76: 468-472.

Jordan, V.W.L. 1981. Aetiology of barley net blotch caused by Pyrenophora teres and some effects on yield. Plant Pathol. 30: 77-87.

Jordan, V.W.L., and E.C. Allen. 1984. Barley net blotch: influence of straw disposal and cultivation methods on inoculum potential, and on incidence and severity of autumn disease. Plant Pathol. 33: 547-559.

Jordan, V.W.L., and G.R. Stinchcombe. 1986. Interactions between fungicide, plant growth regulator, nitrogen fertilizer applications, foliar disease and yield of winter barley. Ann. Appl. Biol. 108: 151-165.

Jørgensen, J. 1980. Comparative testing of barley seed for inoculum of Pyrenophora graminea and P. teres in greenhouse and field. Seed Sci. Technol. 8: 377-381.

Jowett, D., J.A. Browning, and B.C. Haning. 1974. Non-linear disease progress curves. p. 115-136. In J. Kranz (ed.). Epidemics of plant diseases: Mathematical analysis and modeling. Springer-Verlag. New York. NY. USA.

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

Kenneth, R. 1962. On the taxonomy, morphology, and geographic origins of Pyrenophora teres Drechsler, and allied species. Bull. Res. Counc. Israel Sect. D Bot. 11: 55-82.

Kenneth, R. 1964. Conidial release in some Helminthosporia. Nature (London) 202: 1025-1026.

Khan, T.N. 1968. Studies in the breeding of resistance to net blotch of barley. Ph. D. thesis. University of Western Australia. Nedlands. Australia.

Khan, T.N. 1971. Turkish barley varieties as a source of resistance to net blotch. Euphytica 20: 292-298.

Khan, T.N. 1987. Relationship between net blotch (Drechslera teres) and losses in grain yield of barley in Western Australia. Aust. J. Agric. Res. 38: 671-679.

Kiyosawa, S. 1972. Mathematical studies on the curve of disease increase - a technique for forecasting epidemic development. Ann. Phytopathol. Soc. Japan 38: 30-40.

Kramer, C.L., M.G. Eversmeyer, and T.I. Collins. 1976. A new 7-day spore sampler. Phytopathology 66: 60-61.

Kranz, J. 1974. Comparison of epidemics. Annu. Rev. Phytopathol. 12: 355-374.

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

Leach, C.M. 1985. Effect of still and moving moisture-saturated air on sporulation of Drechslera and Peronospora. Trans. Br. Myc. Soc. 84: 179-183.

Little, T.M., and F.J. Hills. 1978. Agricultural experimentation - design and analysis. Wiley and Sons. New York. NY. USA.

Luz, W.C. da. 1982. Effets de fongicides systémiques utilisés en traitement des semences sur la rayure reticulée de l'orge. Fitopatol. Brasileira 7: 169-173.

Machacek, J.E., W.J. Cherewick, H.W. Mead, and W.C. Broadfoot. 1951. A study of some seed-borne diseases of cereals in Canada. II. Kinds of fungi and prevalence of disease in cereal seed. Scientific Agriculture 31: 193-206.

Machacek, J.E., and H.A.H. Wallace. 1942. Non-sterile soil as a medium for tests of seed germination and seed-borne disease in cereals. Can. J. Res. Sect. C. Bot. Sci. 20: 539-557.

Madden, L.V. 1986. Statistical analysis and comparison of disease progress curves. p. 55-86. In K.J. Leonard and W.E. Fry (eds.). Plant disease epidemiology: Population dynamics and management. MacMillan Publishing Co. New York. USA.

Martin, R.A. 1985. Disease progression and yield loss in barley associated with net blotch as influenced by fungicide seed treatment. Can. J. Plant Pathol. 7: 83-90.

Martin, R.A., and K.S. Clough. 1984. Relationship of airborne spore load of Pyrenophora teres and weather variables to net blotch development on barley. Can. J. Plant Pathol. 6: 105-110.

Martin, R.A., H.W. Johnston, and F.G. Dunnewold. 1981. Efficacy of timed applications of CGA64250 on net blotch, stem breakage, and yield in barley. Pestic. Res. Rep. Agric. Can. 1981: 274.

Matthews, D., and J.G. Hampton. 1977. A survey of New Zealand certified barley seed for the presence of pathogenic Drechslera species. N. Z. J. Exp. Agric. 5: 331-334.

Mauler, A. 1983. Netzfleckenkrankheit der Gerste - Krankheitsentwicklung, Bekaempfung und Ertragsgestaltung. Doktorarbeit. Georg-August Univ. Goettingen. FRG.

McDonald, W.C. 1967. Variability and the inheritance of morphological mutants in Pyrenophora teres. Phytopathology 57: 747-755.

McDonald, W.C., and K.W. Buchannon. 1964. Barley yield reductions attributed to net blotch infection. Can. Plant Dis. Surv. 44: 118-119.

Meeus, P. 1982. Essais de lutte chimique contre la maladie des taches brunes de l'escourgeon causées par Pyrenophora teres. Meded. Fac. Landbouwwet. Rijksuniv. Gent 47: 897-911.

Melville, S.C., and C.A. Lanham. 1972. A survey of leaf diseases on spring barley in South-west England. Plant Pathol. 21: 59-66.

Meredith, D.S. 1965. Violent spore release in Helminthosporium turcicum. Phytopathology 55: 1099-1102.

Metcalfe, D.R., K.W. Buchannon, W.C. McDonald, and E. Reinsberg. 1970. Relationships between the 'Jet' and 'Milton' genes for resistance to loose smut and genes for resistance to other barley diseases. Can. J. Plant Sci. 50: 423-427.

Metcalfe, D.R., A Chiko, J.W. Martens, and A. Tekauz. 1978. Reaction of Ethiopian barleys to Canadian barley pathogens. Can. J. Plant Sci. 58: 885-890.

Metcalfe, N.D.S., and D.R. Jones. 1985. Evaluation of fungicides for control of net blotch of winter barley. Ann. Appl. Biol. 106: (Suppl. 6: Tests of agrochemicals and cultivars) 42-43.

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

Neuhaus, W. and D. Moritz. 1986. Ergebnisse zur Bekaempfung der Netzfleckenkrankheit (Pyrenophora teres Drechsler) in Sommergerste. Nachrichtenbl. Pflanzenschutz DDR 40: 84-87.

Newman, C.W., and C.F. McGuire. 1985. Nutritional quality of barley. In D.C. Rasmusson (ed.). Barley. Agronomy 26: 403-456.

Onesirosan, P.T., and E.E. Banttari. 1969. The effect of light and temperature upon sporulation of Helminthosporium teres in culture. Phytopathology 59: 906-909.

Park, E.W., and S.M. Lim. 1985. Empirical estimation of the asymptote of disease progress curves and use of the Richards generalized rate parameters for describing disease progress. Phytopathology 75: 786-791.

Peat, W.E., and D.M. Shipp. 1981. The effects of Benomyl on the growth and development of wheat. EPPO Bull. 11: 287-293.

Peterson, R.F., A.B. Campbell, and A.E. Hannah. 1948. A diagrammatic scale of estimating rust intensity on leaves and stems of cereals. Can. J. Res. Sect. C Bot. Sci. 26: 496-500.

Piening, L.J. 1968. Development of barley net blotch from infested straw and seed. Can. J. Plant Sci. 48: 623-625.

Plank, J.E. van der. 1963. Plant diseases: Epidemics and control. Academic Press. New York. NY. USA.

Reid, D.A. 1985. Morphology and anatomy of the barley plant. In D.C. Rasmusson (ed.). Barley. Agronomy 26: 75-101.

Reid, D.A., and G.A. Wiebe. 1979. Taxonomy, botany, classification and world collection. p. 78-104. In Barley, origin, botany, culture, winterhardiness, genetics, utilization, pests. USDA Agricultural Handbook no. 338.

Riesen, T.K., and R.C. Close. 1987. Endophytic fungi in propiconazole-treated and untreated barley leaves. Mycologia 79: 546-552.

Rintelen, J. 1969. Untersuchungen ueber die Ertragsverluste durch die Netzfleckenkrankheit der Gerste (Erreger: Pyrenophora teres Drechsl., stat. con.: Helminthosporium teres Sacc.). Z. Pflanzenkr. Pflanzenpathol. Pflanzenschutz 76: 147-152.

Rotem, J. 1978. Climatic and weather influences on epidemics. In J.G. Horsfall, and E.B. Cowling (eds.). Plant disease - an advanced treatise. Vol. II. How disease develops in populations. Acacemic Press. New York. NY. USA.

SAS Institute. 1985. SAS user's guide: Statistics. Version 5 Edition. SAS Institute Inc. Cary. NC. USA.

Saskatchewan Agriculture. 1984. Guide to farm practice in Saskatchewan. Division of Extension and Community Relations. University of Saskatchewan. Saskatoon. Saskatchewan. Canada.

Saskatchewan Agriculture. 1986a. Agricultural Statistics 1985. Saskatchewan Agriculture, Economics Branch. Regina. Saskatchewan. Canada.

Saskatchewan Agriculture. 1986b. Varieties of grain crops for Saskatchewan 1986. Saskatchewan Agriculture, Plant Industry Branch. Regina. Saskatchewan. Canada.

Shaner, G., and R.E. Finney. 1977. The effect of nitrogen fertilization on the expression of slow-midewing in Knox wheat. Phytopathology 67: 1051-1056.

Shaw, M.W. 1986. Effects of temperature and leaf wetness on Pyrenophora teres growing on cv. Sonja. Plant Pathol. 35: 294-309.

Shaw, M.W., and D.J. Royle. 1987. A new method for disease-loss studies used to estimate effects of Pyrenophora teres and Rhynchosporium secalis on winter barley yields. Ann. Appl. Biol. 110: 247-262.

Sheridan, J.E. 1977. Drechslera spp. and other seed-borne pathogenic fungi in New Zealand cereals. N. Z. J. Agric. Res. 20: 91-93.

Sheridan, J.E., and N. Grbavac. 1983. Seed treatments for control of net blotch in barley. Proc. N. Z. Weed & Pest Control Conf. 36: 237-241.

Sheridan, J.E., and N. Grbavac. 1985. Cultivar differences in response to triadimenol seed treatment for control of barley net blotch caused by Pyrenophora teres. Plant Dis. 69: 77-80.

Sheridan, J.E., N. Grbavac, and L. Ballard. 1983. Strategies for controlling net blotch in barley. Proc. N. Z. Weed & Pest Control Conf. 36: 242-246.

Shipton, W.A. 1966. Effect of net blotch infection of barley on grain yield and quality. Aust. J. Exp. Agric. Anim. Husb. 6: 437-440.

Shipton, W.A., T.N. Khan, and W.J.R. Boyd. 1973. Net blotch of barley. Rev. Plant Pathol. 52: 269-290.

Shoemaker, R.A. 1962. Drechslera Ito. Can. J. Bot. 40:809-836.

Shoemaker, R.A. 1981. Changes in taxonomy and nomenclature of important genera of plant pathogens. Annu. Rev. Phytopathol. 19: 297-307.

Singh, A., and S. Singh. 1978. Resistant donors against Helminthosporium teres inciting net-blotch of barley. Indian Phytopathol. 31: 520-521.

Singh, M., and J.N. Chand. 1985. Studies on the survival of Helminthosporium teres incitant of net blotch of barley. Indian Phytopathol. 38: 659-661.

Singh, S. 1956. Physiology and epidemiology of Helminthosporium teres. Ph. D. thesis. Univ. of Minnesota. St. Paul. Minn. USA. (Diss. Abstr. 00-20563)

Singh, S. 1962. Biotic factors affecting barley net-blotch (Helminthosporium teres) epidemiology. Indian Phytopathol. 15: 195-202.

Singh, S. 1963a. Effect of temperature on secondary infection and development of net-blotch of barley (Helminthosporium teres Sacc.). Indian Phytopathol. 16: 94-97.

Singh, S. 1963b. Effect of moisture on the incidence of net-blotch of barley (Helminthosporium teres Sacc). Indian Phytopathol. 16: 128-132.

Smedegaard-Petersen, V. 1971. Pyrenophora teres f. maculata f. nov. and Pyrenophora teres f. teres in Denmark. K. Vet. Landbohosjk. Arsskr. 1971: 124-144.

Smedegaard-Petersen, V. 1972. The perithecial and pycnidial stages of Pyrenophora teres and Pyrenophora graminea in Denmark. Friesia 10: 61-85.

Smedegaard-Petersen, V. 1974. Reduction in yield and grain size of barley due to attack by the net blotch fungus Pyrenophora teres. K. Vet. Landbohosjk. Arsskr. 1974: 108-117.

Smedegaard-Petersen, V. 1977. Inheritance of genetic factors for symptoms and pathogenicity in hybrids of Pyrenophora teres and P. graminea. Phytopathol. Z. 89: 193-202.

Steel, R.G.D., and J.H. Torrie. 1980. Principles and procedures of statistics - a biometric approach. 2nd edition. McGraw-Hill. New York. NY. USA.

Sutton, J.C., and P. Steele. 1983. Effects of seed and foliar fungicides on progress of net blotch and yield in barley. Can. J. Plant Sci. 63: 631-639.

Tekauz, A. 1978. Incidence and severity of net blotch of barley and distribution of Pyrenophora biotypes in the Canadian prairies in 1976. Can. Plant Dis. Surv. 58: 9-11.

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

Tekauz, A. 1986. Effect of plant age and leaf position on the reaction of barley to Pyrenophora teres. Can. J. Plant Pathol. 8: 380-386.

Tekauz, A, and K.W. Buchannon. 1977. Distribution of and sources of resistance to biotypes of Pyrenophora teres in western Canada. Can. J. Plant Sci. 57: 389-395.

Tekauz, A, and J.T. Mills. 1974. New types of virulence in Pyrenophora teres in Canada. Can. J. Plant Sci. 54: 731-734.

Teng, P.S. 1987. Quantifying the relationship between disease intensity and yield loss. p. 105-113. In P.S. Teng (ed.). Crop loss assessment and pest management. APS Press. St. Paul. MN. USA.

Teng, P.S., M.J. Blackie, and R.C. Close. 1980. A comparison of models for estimating yield loss caused by leaf rust (Puccinia hordei) on Zephyr barley in New Zealand. Phytopathology 69: 1239-1244.

Teng, P.S., and R.E. Gaunt. 1980. Modelling systems of disease and yield loss in cereals. Agric. Syst. 6: 131-154.

Tolstrup, K., and V. Smedegaard-Petersen. 1984. Saprophytic leaf fungi on barley and their effect on leaf senescence and grain yield. Vaxtskyddsnotiser 48(3/4): 66-75.

Tukey, J.W. 1951. Components in regression. Biometrics 7: 33-69.

Ubels, E. 1979. A method to test wheat leaves for their reactions to inoculation with Septoria species. Neth. J. Plant Pathol. 85: 143-150.

Webster, J. 1951. Graminicolous Pyrenomycetes. II. The occurence of the perfect stage of Helminthosporium teres in Britain. Trans. Br. Myc. Soc. 34: 309-317.

Wetherill, G.B. 1986. Regression analysis with applications. Chapman and Hall. New York. NY. USA.

Yoshida, S. 1972. Physiological aspects of grain yield. Annu. Rev. Plant Physiol. 23: 437-464.

Zadoks, J.C., T.T. Chang, and C.F. Konzak. 1974. A decimal code for the growth stages of cereals. Weed Res. 14: 415-421.

Zadoks, J.C., and R.D. Schein. 1979. Epidemiology and plant disease management. Oxford University Press. Oxford. UK.

APPENDIX A. Tables

LIST OF APPENDIX TABLES

Table A.1. Percentage germinated conidia for three isolates for six leaf wetness periods at six temperature regimes 171

Table A.2. Development of Elrose, measured on the Zadoks scale at Shellbrook in 1986 and 1987 173

Table A.3. Total green leaf area and green leaf area for five leaf positions in 1986 and for seven leaf positions in 1987 at Shellbrook 174

Table A.4. Percentage necrotic leaf area for five leaf positions in 1986 and for seven leaf positions in 1987 at Shellbrook 175

Table A.5. Number of spores trapped per day at Shellbrook in June of 1986 176

Table A.6. Number of spores trapped per day at Shellbrook in June of 1987 177

Table A.7. Number of spores trapped per day at Shellbrook in July of 1986 178

Table A.8. Number of spores trapped per day at Shellbrook in July of 1987 179

Table A.9. Incidence of airborne conidia summed over the observation period for each hour of the day 180

Table A.10. Estimates of the parameters for the monomolecular function fitted to percentage necrotic leaf area and for the logistic function fitted to percentage affected leaf area of Elrose in 1985 and 1986, and for the exponential function fitted to percentage necrotic leaf area and for the logistic function fitted to percentage affected leaf area of Argyle in 1986 181

Table A.11. Analysis of variance for percentage affected leaf area of Elrose at three sites in 1985 and 1986 and of Argyle at three sites in 1986 182

Table A.12. Analysis of variance for grain yield of Elrose at three sites in 1985 and 1986 and of Argyle at three sites in 1986 183

Table A.13. Analysis of variance for number of tillers of Elrose at three sites in 1985 184

Table A.14. Analysis of variance for number of kernels per spike of Elrose at three sites in 1985 and 1986 and of Argyle at three sites in 1986 185

Table A.15. Analysis of variance for kernel weight of Elrose at three sites in 1985 and 1986 and of Argyle at three sites in 1986 186

Table A.1. Percentage germinated conidia for three isolates for six leaf wetness periods at six temperature regimes.

Leaf wetness period	Isolate WRS 858	M	S	SD
Constant 10 °C				
1	0.1	5.0	7.0	2.4
2	7.0	5.0	26.0	2.9
3	17.5	26.5	69.0	3.9
4	42.0	62.0	67.0	8.5
5	73.0	77.5	80.0	13.6
6	87.0	93.0	94.0	4.1
Constant 15 °C				
1	2.1	19.0	15.0	0.8
2	11.0	24.0	31.0	11.1
3	24.0	61.5	78.5	8.6
4	45.0	79.5	70.5	24.3
5	76.0	93.0	88.5	1.5
6	92.5	82.5	85.0	7.8
Constant 20 °C				
1	26.0	30.0	39.0	2.8
2	41.0	66.0	62.0	4.1
3	76.0	82.0	91.0	8.0
4	82.0	97.5	89.5	1.2
5	88.0	90.0	86.0	2.4
6	90.0	95.0	94.5	2.3
Constant 25 °C				
1	45.0	31.0	33.0	14.2
2	64.0	57.0	88.0	12.5
3	81.0	85.5	82.0	3.2
4	88.0	97.0	93.0	2.9
5	93.0	95.0	93.0	1.1
6	98.5	98.5	98.0	1.1
20 decreased to 10 °C				
1	20.0	75.0	46.0	0.8
2	48.0	98.5	80.5	5.3
3	63.5	98.5	92.0	14.0
4	68.5	95.5	98.5	7.3
5	73.5	99.0	99.0	4.5
6	85.0	99.0	98.5	1.5

... continued

Table A.1. (Continued)

Leaf wetness period	Isolate			
	WRS 858	M	S	SD
25 decreased to 10 °C				
1	20.0	48.5	56.5	19.6
2	40.0	73.5	73.0	6.6
3	64.5	77.5	88.5	13.5
4	71.5	85.0	96.5	12.7
5	82.0	94.0	95.5	4.0
6	95.5	99.0	99.0	2.9

Data points for Figures 4.1.1 to 4.1.6.

Table A.2. Development of Elrose, measured on the Zadoks scale at Shellbrook in 1986 and 1987.

Days from seeding	Growth stage 1986	1987
15	.	13
22	.	23
24	13	.
29	.	31
31	23	.
35	.	33
42	32	45
49	.	53
54	49	.
56	.	59
59	55	.
62	59	.

Data points for Figure 4.2.1.

Table A.3. Total green leaf area and green leaf area for five leaf positions in 1986 and for seven leaf positions in 1987 at Shellbrook.

Days from seeding	Leaf position[a] 7	6	5	4	3	2	1	Total
	cm^2							
1986								
38	.	.	8.54	15.71	15.21	.	.	31.8
49	.	.	0	10.28	18.08	14.98	4.97	49.1
54	.	.	.	8.00	15.23	11.52	3.66	38.4
59	.	.	.	4.45	7.25	9.09	3.83	24.6
62	.	.	.	0.33	4.98	7.11	3.40	15.8
69	.	.	.	.	0.21	2.08	1.21	3.5
74	.	.	.	.	.	0.19	0.13	0.31
1987								
15	4.85	5.03	.	.	.	.	.	9.9
22	4.66	4.99	8.74	10.53	3.93	.	.	15.5
29	1.02	1.70	7.13	11.55	15.20	8.83	.	45.3
35	.	.	0.41	2.02	8.36	10.81	1.44	23.0
42	.	.	.	1.65	8.77	11.42	3.37	25.2
49	.	.	.	0.25	6.58	8.64	3.49	19.0
56	.	.	.	.	1.73	6.06	1.67	9.5
63	.	.	.	.	.	0.31	0.02	0.33

[a] Data points for Figures 4.2.2 and 4.2.3.
Flag leaf is leaf position 1.

Table A.4. Percentage necrotic leaf area for five leaf positions in 1986 and for seven leaf positions in 1987 at Shellbrook.

Days from seeding	Leaf position[a] 7	6	5	4	3	2	1
1986							
38	.	.	16.0	5.0	1.4	.	.
49	.	.	18.0	14.3	6.8	1.4	0.4
54	.	.	.	28.0	15.8	11.7	5.4
59	.	.	.	.	35.7	21.7	10.7
62	.	.	.	.	37.3	31.0	20.0
69	.	.	.	.	.	37.0	35.6
1987							
15	0.0	0.5	.	.	.	.	.
22	4.8	1.6	0.5	0.0	.	.	.
29	17.0	13.5	10.5	6.5	1.0	.	.
35	.	.	.	25.0	4.6	0.1	.
42	.	.	.	30.0	9.1	1.5	0.0
49	.	.	.	.	35.0	25.0	11.5
56	.	.	.	.	.	30.0	23.0
63	.	.	.	.	.	35.0	25.0

[a] Data points for Figures 4.2.4 and 4.2.5.
Flag leaf is leaf position 1.

Table A.5. Number of spores trapped per day at Shellbrook in June of 1986.

Day of the month	log10(number of spores +1)	number of spores
1	-	-
2	0.47712	2
3	0.60206	3
4	0.47712	2
5	0.30103	1
6	0.95424	8
7	1.04139	10
8	1.73239	53
9	1.36173	22
10	1.17609	14
11	1.67210	46
12	1.27875	18
13	0.00000	0
14	0.00000	0
15	0.00000	0
16	0.60206	3
17	1.14613	13
18	1.50515	31
19	1.97772	94
20	1.60206	39
21	1.69897	49
22	0.60206	3
23	1.63347	42
24	1.43136	26
25	1.49136	30
26	1.89209	77
27	1.47712	29
28	1.27875	18
29	0.69897	4
30	1.14613	13

Data points for Figure 4.2.6.

Table A.6. Number of spores trapped per day at Shellbrook in June of 1987.

Day of the month	log10(number of spores +1)	number of spores
1	2.05308	112
2	1.51851	32
3	1.07918	11
4	0.30103	1
5	1.00000	9
6	1.07918	11
7	0.77815	5
8	0.60206	3
9	0.90309	7
10	0.60206	3
11	2.53148	339
12	2.06070	114
13	2.58433	383
14	1.77815	59
15	2.19590	156
16	1.41497	25
17	1.83885	68
18	1.30103	19
19	1.41497	25
20	1.54407	34
21	1.68124	47
22	2.35218	224
23	2.03342	107
24	2.06446	115
25	1.53148	33
26	1.25527	17
27	2.16137	144
28	2.19312	155
29	1.84510	69
30	1.34242	21

Data points used for Figure 4.2.7.

Table A.7. Number of spores trapped per day at Shellbrook in July of 1986.

Day of the month	log10(number of spores +1)	number of spores
1	1.14613	13
2	1.34242	21
3	1.57978	37
4	2.28556	192
5	1.53148	33
6	0.84510	6
7	1.41497	25
8	0.84510	6
9	1.44716	27
10	0.69897	4
11	1.54407	34
12	2.43457	271
13	1.53148	33
14	2.95424	899
15	1.94448	87
16	2.40312	252
17	1.69897	49
18	0.47712	2
19	2.12710	133
20	2.33244	214
21	2.48572	305
22	2.88705	770
23	2.67943	477
24	1.98227	95
25	1.92428	83
26	2.16435	145
27	1.30103	19
28	2.42160	263
29	3.30920	2037
30	2.17026	147
31	3.40261	2526

Data points for Figure 4.2.8.

Table A.8. Number of spores trapped per day at Shellbrook in July of 1987.

Day of the month	log10(number of spores +1)	number of spores
1	2.03342	107
2	2.12710	133
3	2.20683	160
4	1.27875	18
5	0.47712	2
6	2.23805	172
7	2.40824	255
8	2.80482	637
9	2.31597	206
10	1.97313	93
11	2.01703	103
12	2.02531	105
13	2.23045	169
14	3.15412	1425
15	2.20952	161
16	2.73239	539
17	2.75891	573
18	3.01072	1024
19	1.90849	80
20	2.64933	445
21	2.85974	723
22	3.01578	1036
23	2.80277	634
24	3.02612	1061
25	3.23096	1701
26	3.28780	1939
27	2.95713	905
28	3.08422	1213
29	2.56937	370
30	1.85733	71
31	3.37033	2345

Data points used in Figure 4.2.9.

Table A.9. Incidence of airborne conidia summed over the observation period for each hour of the day.

Hour of the day	1986		1987	
	Number	%[a]	Number	%[a]
0 = 24	119	0.60	105	0.38
1	122	0.61	100	0.37
2	146	0.73	156	0.57
3	69	0.35	77	0.28
4	11	0.06	63	0.23
5	22	0.11	89	0.33
6	64	0.32	254	0.93
7	132	0.66	500	1.9
8	504	2.5	1133	4.3
9	1108	5.6	1580	6.0
10	1521	7.6	2212	8.4
11	1891	9.5	2423	9.2
12	2484	12.5	2397	9.1
13	2827	14.2	2476	9.4
14	3148	15.8	2976	11.3
15	2222	11.2	2976	11.3
16	1514	7.6	2344	8.9
17	975	4.9	1343	5.1
18	493	2.5	1106	4.2
19	238	1.2	816	3.1
20	79	0.40	450	1.6
21	60	0.30	334	1.2
22	53	0.27	187	0.68
23	100	0.50	313	1.1
Total	19902		26410	

[a] Data points used in Figure 4.2.10.
% = percentage of Total.

Table A.10. Estimates of the parameters for the monomolecular function fitted to percentage necrotic leaf area and for the logistic function fitted to percentage affected leaf area of Elrose in 1985 and 1986, and for the exponential function fitted to percentage necrotic leaf area and for the logistic function fitted to percentage affected leaf area of Argyle in 1986.

		Percentage necrotic leaf area				Percentage affected leaf area		
Treatment	Rep	B	C	A	r^2	B	C	r^2
		1	1/day	%	1	1	1/day	1
Elrose 1985								
Repeated	1	-6.39	0.139	22.5	0.93	-11.31	0.172	0.99
	2	-6.18	0.136	24.5	0.96	-13.49	0.215	0.97
	3	-6.16	0.133	24.5	0.87	-11.55	0.176	0.98
	4	-6.21	0.133	24.5	0.88	-10.94	0.162	0.99
	5	-6.28	0.137	21.5	0.93	-9.99	0.148	0.98
	6	-6.14	0.133	21.5	0.94	-12.15	0.173	0.98
Untreated	1	-7.30	0.154	32.5	0.99	-16.63	0.292	0.98
	2	-6.65	0.161	32.5	0.96	-14.83	0.250	0.96
	3	-7.37	0.147	32.5	0.95	-14.13	0.240	0.95
	4	-7.33	0.163	33.5	0.96	-16.60	0.287	0.94
	5	-7.10	0.162	33.5	0.97	-15.55	0.264	0.99
	6	-6.18	0.151	35.5	0.87	-17.35	0.287	0.98
Elrose 1986								
Repeated	1	-6.18	0.115	16.5	0.64	-13.32	0.168	0.96
	2	-6.45	0.120	26.5	0.54	-13.39	0.180	0.85
	3	-6.24	0.115	21.5	0.54	-13.41	0.176	0.93
Untreated	1	-10.74	0.207	39.5	0.83	-17.36	0.276	0.99
	2	-10.16	0.214	41.5	0.83	-21.36	0.343	0.98
	3	-11.00	0.217	33.5	0.95	-15.61	0.242	0.97
Argyle 1986								
Repeated	1	-3.43	0.069	-	0.87	-10.32	0.092	0.95
	2	-3.94	0.097	-	0.85	-10.20	0.104	0.80
	3	-2.86	0.103	-	0.90	-9.75	0.144	0.94
Untreated	1	-0.97	0.101	-	0.98	-9.14	0.101	0.97
	2	-2.46	0.113	-	0.91	-9.72	0.102	0.85
	3	-3.06	0.093	-	0.94	-12.25	0.093	0.91

Table A.11. Analysis of variance for percentage affected leaf area of Elrose at three sites in 1985 and 1986 and of Argyle at three sites in 1986.

Days from seeding	SS			df[a]		
	Replication	Treatment	Error	Rep	Trt	Error
		Elrose 1985				
Medstead						
62	36	966**	283	5	4	20
70	203+	7560**	377	5	4	20
78	109	4886**	851	5	4	20
Shellbrook						
54	116+	1181**	192	5	4	20
62	769*	2654**	937	5	4	20
70	461	4095**	937	5	4	20
		Elrose 1986				
Medstead						
51	10	35	52	2	3	6
66	50	2602*	1238	2	3	6
Shellbrook						
54	27	248*	101	2	4	8
59	33	712*	238	2	4	8
62	3	2194**	82	2	4	8
69	148	10841**	1011	2	4	8
74	132	4630**	540	2	4	8
Saskatoon						
69	36*	217**	9	1	4	4
78	3	4878**	199	1	4	4
		Argyle 1986				
Medstead						
73	50	228+	101	2	3	6
Shellbrook						
74	30	92	70	2	4	8
80	119	428+	283	2	4	8

Analysis of variance is presented for observation days on which the difference between the highest and the lowest value exceeded 5%.

+, *, ** Significant at the 0.1, 0.05 and 0.01 probability level, respectively.

[a] Rep = Replication; Trt = Treatment.

Table A.12. Analysis of variance for grain yield of Elrose at three sites in 1985 and 1986 and of Argyle at three sites in 1986.

	SS			df[a]		
Location	Replication	Treatment	Error	Rep	Trt	Error
Elrose 1985						
Medstead	0.10	1.90**	0.91	5	4	20
Shellbrook	1.42**	2.05**	0.79	5	4	20
Saskatoon	0.22	0.31	0.91	5	4	20
Elrose 1986						
Medstead	0.27	1.79**	0.55	5	3	15[b]
Shellbrook	0.39	8.40**	0.80	5	4	20
Saskatoon	0.32*	2.52**	0.40	5	4	20
Argyle 1986						
Medstead	0.39	2.11*	2.17	5	3	15[b]
Shellbrook	0.57	0.87	2.35	5	4	20
Saskatoon	0.05	0.23+	0.49	5	4	20

+, *, ** Significant at the 0.1, 0.05 and 0.01 probability level, respectively.

[a] Rep = Replication; Trt = Treatment.

[b] Repeatedly sprayed control was not used.

[c] One plot was missing.

Table A.13. Analysis of variance for number of tillers of Elrose at three sites in 1985.

	SS			df[a]		
Location	Replication	Treatment	Error	Rep	Trt	Error
Medstead	17,127	6,518	117,250	5	4	20
Shellbrook	69,933	38,509	156,281	5	4	20
Saskatoon	79,129	35,784	513,834	5	4	20

[a] See Table A.12.

Table A.14. Analysis of variance for number of kernels per spike of Elrose at three sites in 1985 and 1986 and of Argyle at three sites in 1986.

	SS			df[a]		
Location	Replication	Treatment	Error	Rep	Trt	Error
Elrose 1985						
Medstead	2.76	10.43*	14.89	5	4	20
Shellbrook	7.28	3.32	13.26	5	4	20
Saskatoon	0.75	3.26	12.06	5	4	20
Elrose 1986						
Medstead	3.45	4.60	21.89	5	3	15[a]
Shellbrook	1.84	5.35*	6.88	5	4	20
Saskatoon	2.81	5.78*	9.25	5	4	20
Argyle 1986						
Medstead	11.16	45.50	92.09	5	3	15[a]
Shellbrook	26.70	12.45	72.64	5	4	20
Saskatoon	28.23*	12.60	33.98	5	4	20

* Significant at the 0.05 probability level.
[a] See Table A.12.

Table A.15. Analysis of variance for kernel weight of Elrose at three sites in 1985 and 1986 and of Argyle at three sites in 1986.

	SS			df[a]		
Location	Replication	Treatment	Error	Rep	Trt	Error
Elrose 1985						
Medstead	11.34	132.01**	33.87	5	4	20
Shellbrook	48.65**	185.96**	1.98	5	4	20
Saskatoon	4.00	5.85*	9.55	5	4	20
Elrose 1986						
Medstead	13.50+	76.43**	17.52	5	3	15[a]
Shellbrook	19.03*	463.20**	24.39	5	4	20
Saskatoon	19.41*	11.56+	21.66	5	4	20
Argyle 1986						
Medstead	16.42+	22.42**	19.14	5	3	15[a]
Shellbrook	1.58	10.61+	21.72	5	4	20
Saskatoon	8.64*	2.17	8.99	5	4	20

+, *, ** Significant at the 0.1, 0.05 and 0.01 probability level, respectively.

[a] See Table A.12.

APPENDIX B. Figures

LIST OF APPENDIX FIGURES

Figure B.1. Progress of percentage necrotic leaf area and percentage affected leaf area in the untreated control of Elrose in 1986 (Replication 3). Data were used to fit the monomolecular and logistic curve, respectively (See Table A.10) 189

Figure B.2. Progress of percentage necrotic leaf area and percentage affected leaf area in the untreated control of Argyle in 1986 (Replication 1). Data were used to fit the exponential and logistic curve, respectively (See Table A.10) 190

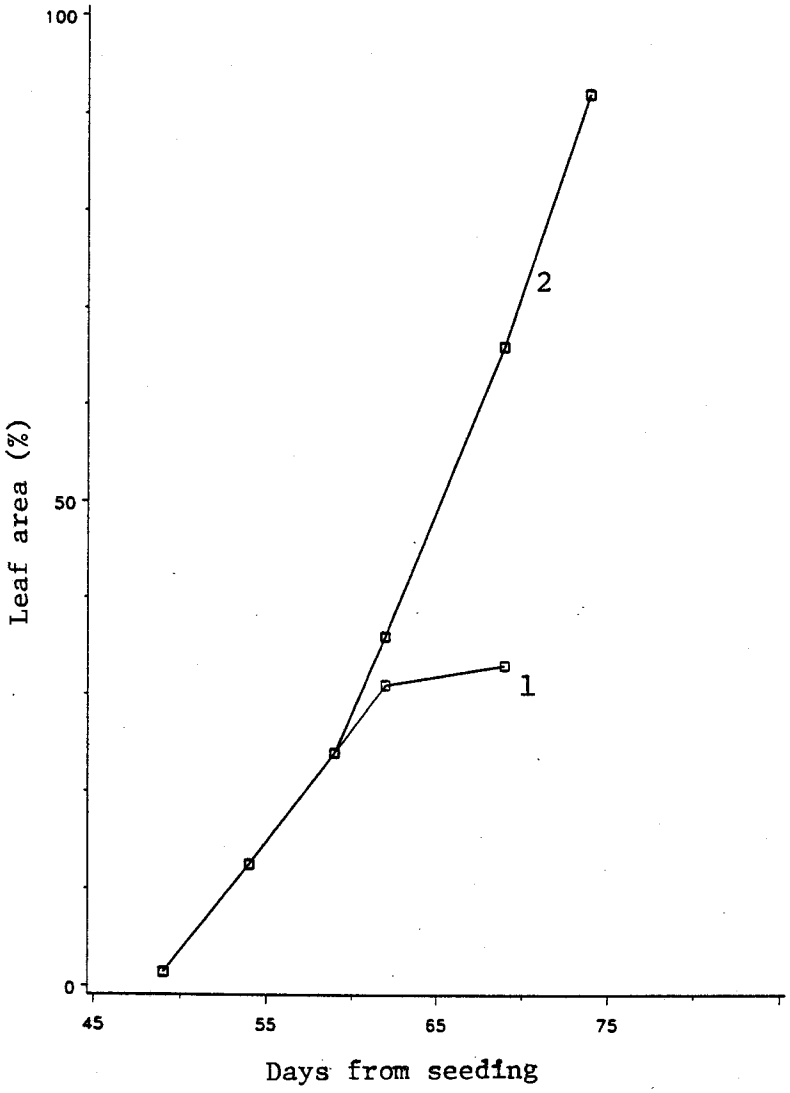

Figure B.1. Progress of percentage necrotic leaf area (1) and percentage affected leaf area (2) in the untreated control of Elrose in 1986 (Replication 3). Data were used to fit the monomolecular and logistic curve, respectively (See Table A.10).

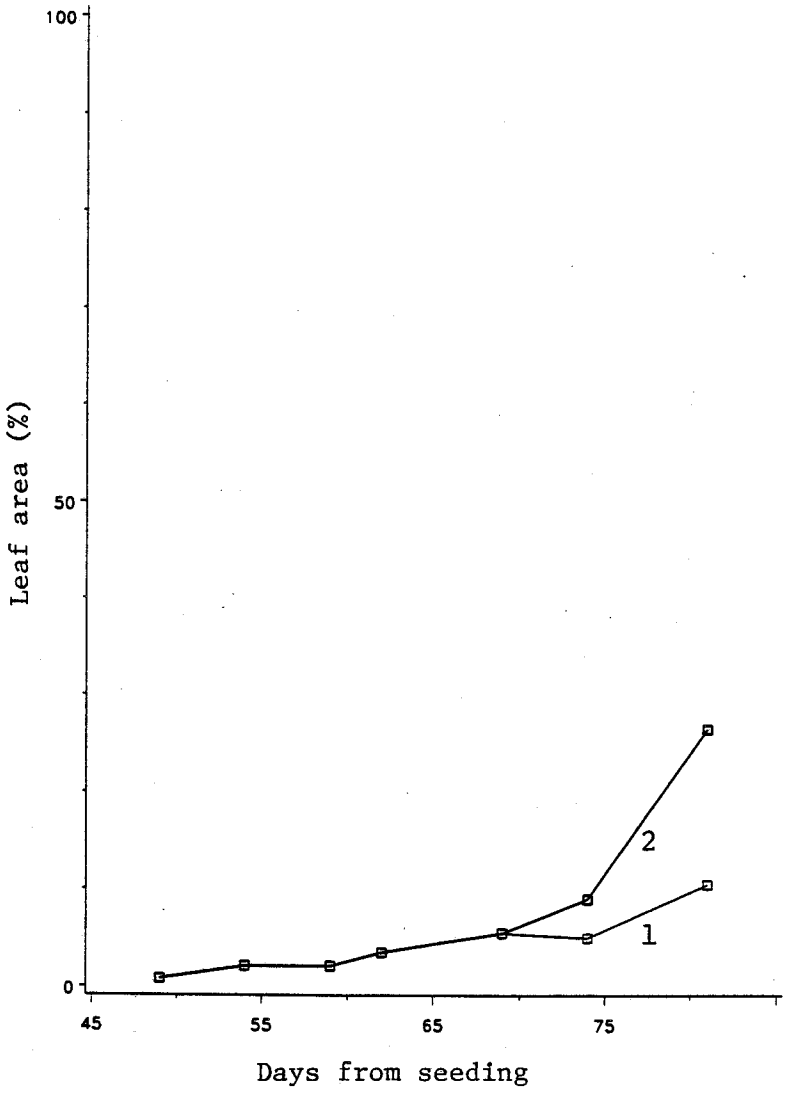

Figure B.2. Progress of percentage necrotic leaf area (1) and percentage affected leaf area (2) in the untreated control of Argyle in 1986 (Replication 1). Data were used to fit the exponential and logistic curve, respectively (See Table A.10).