EPIDEMIOLOGY OF THE SEPTORIA DISEASE COMPLEX OF WHEAT: EFFECT OF CULTIVAR, CROP ROTATION AND WEATHER ON DISEASE DEVELOPMENT

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ABSTRACT

The complex of the septoria diseases of wheat, septoria nodorum blotch, septoria tritici blotch and septoria avenae blotch, is economically important in the Parkland region of Saskatchewan. Cultural practices are the only economic means of control. Thus, the objectives of this study were to examine the epidemiology of the septoria disease complex on spring wheat grown in the Parkland region of Saskatchewan and to determine the effects of cultivar, crop rotation and weather conditions on disease development.

Cultivars used in this study possessed only low levels of resistance to the septoria complex. However some cultivars were more resistant than others. HY320 and Oslo had the highest levels of resistance, while Park, Kenyon and Roblin had the lowest. Katepwa, Neepawa, Pembina and Columbus had intermediate levels of resistance.

Weather conditions were favorable for disease development in July and August of 1987 and severe septoria epidemics occurred. A rotation with one year of summerfallow between wheat crops showed little reduction of disease development compared to continuous wheat. However, a rotation of two years between wheat crops significantly reduced the area under the disease progress curve (ADPC) and

significantly increased the time to 50% disease severity (T_{50}) of all cultivars.

In 1988, weather conditions were much less favorable for disease development and septoria epidemics were light to moderately severe. Under this lower disease pressure a rotation of one year between wheat crops adequately controlled the disease complex. Crop rotation had a small and inconsistent effect on the apparent infection rates of cultivars in both years.

The *Septoria* species involved in the disease complex were examined by identifying and counting lesions produced by each pathogen. Lesions of *S. tritici* were rarely observed on the cultivar Oslo indicating that Oslo possesses a fairly high level of resistance to this pathogen. A rotation of two years between wheat crops significantly reduced the number of lesions of *S. nodorum*. The relative occurrence of *S. nodorum* and *S. tritici* varied with location and year. Few lesions of *S. avenae* f.sp. *triticea* were observed.

Grain yield and kernel weight at the continuous wheat site was lower than that at the barley or canola stubble sites in 1987. In 1988, the low level of disease and the drought conditions at Shellbrook resulted in little useful yield and kernel weight data. At Weirdale in 1988, a significant yield loss of 12 to 19% occurred with a rotation

of continuous wheat or one year between wheat crops. No significant yield loss occurred at the site with a rotation of two years between wheat crops. Kernel weight loss at the Weirdale sites did not appear to be related to disease.

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TABLE OF CONTENTS

LIST OF TABLES

Table

LIST OF FIGURES

FIGURE

1. INTRODUCTION

In recent years the complex of the septoria diseases of wheat has become increasingly evident in Saskatchewan. Wheat production practices, such as increased use of nitrogen fertilizer, continuous cropping and reduced tillage result in greater amounts of plant debris left on the soil surface following harvest. Because infected wheat debris is a major source of primary inoculum, these practices also lead to a higher incidence and severity of septoria epidemics (Eyal, 1981). Furthermore, the development of wheat cultivars with resistance to major leaf diseases (e.g. the stem and leaf rusts) has shifted plant pathologists' and plant breeders' attention to the less obvious, but potentially destructive, septoria diseases.

The septoria diseases of wheat are economically important in all wheat producing areas of the world. Yield losses of 30 to 50% have been reported in West Germany, the United Kingdom and Brazil (Jones, 1985; King et al. 1983). Locally, moderate septoria epidemics have caused an average yield loss of 15% (G.R. Hughes, unpublished data).

Disease surveys have shown that the septoria disease complex is widespread throughout the Parkland region of Saskatchewan (unpublished data). Severe epidemics occurred in 1982 and 1983 when *S. nodorum* was the predominant species (G.R. Hughes, personal communication).

Control measures need to be implemented to reduce the economic impact of this disease complex. Host resistance is the most desirable means of control, but resistant cultivars are not yet available in Saskatchewan. Fungicide application has given successful control on winter wheat in Europe (Ferhmann, 1985), but would probably not be cost effective on spring wheat in Saskatchewan because of low yields and depressed grain prices. The only practical means of control available at present are through cultural practices.

Crop rotation is a standard cultural practice in the Parkland region of the province, but its effectiveness in controlling the septoria disease complex of wheat under Saskatchewan conditions has not been determined. This study was conducted with the following objectives:

1. to monitor the natural progress of the septoria disease complex of wheat on several spring wheat cultivars in the Parkland region of Saskatchewan;
2. to determine the effect of crop rotation on this disease progression;
3. to determine the involvement of the different *Septoria* species in the disease complex; and
4. to monitor weather conditions and to investigate general relationships between weather and development of the septoria epidemics.

2. LITERATURE REVIEW

2.1 Disease complex

2.1.1 Causal fungi

Three *Septoria* species incite leaf spotting diseases of wheat in Saskatchewan. *S. nodorum* (Berk.) Berk. (teleomorph: *Leptosphaeria nodorum* Muller) is the causal agent of septoria nodorum blotch of wheat (syn. glume blotch of wheat). *S. tritici* Rob. ex Desm. (teleomorph: *Mycosphaerella graminicola* (Fuckel) Schroeter) is the causal agent of septoria tritici blotch of wheat (syn. septoria leaf blotch, speckled leaf blotch of wheat) and *S. avenae* Frank f.sp. *triticea* T. Johnson (teleomorph: *Leptosphaeria avenaria* Weber f.sp. *triticea* T. Johnson) is the causal agent of septoria avenae blotch of wheat (syn. septoria leaf blotch of wheat).

2.1.2 Host range

The pathogens causing the septoria disease complex infect a number of different graminaceous hosts (Shipton et al., 1971; King et al., 1983). *S. nodorum* colonizes the widest range of grass genera and is thought, therefore, to be less specialized than the other septoria pathogens (Williams and Jones, 1973; Ao and Griffith, 1976). *S. nodorum* has been isolated from species of 17 genera (Shipton et al., 1971) including *Triticum* spp., *Secale cereale* L., *Poa pratensis* L. (Weber, 1922), *Lolium multiflorum*, *Phleum*

pratense, *Bromus sterilis* (Harrower, 1977), *Hordeum vulgare* (Holmes and Colhoun, 1970; Cunfer and Youmans, 1983), species of *Agropyron*, *Elymus*, *Bromus*, *Festuca*, *Hystrix*, (Rufty et al., 1981b; Krupinsky, 1985), *Hordeum jubatum* (Krupinsky, 1985), and *Avena sativa* (Johnston and Scott, 1988).

Beach (1919) suggested that *S. tritici* was capable of attacking 15 genera of grasses, but Weber (1922) and Sprague (1944) were unable to confirm these reports. Weber (1922) found that isolates taken from wheat, rye and *Poa pratensis* were only able to infect *Triticum* spp., rye and *P. pratensis*, while Sprague (1944) found that isolates from wheat attacked *Triticum* spp. and *Poa* spp.

Isolates of *S. avenae* f.sp. *triticea* from wheat, rye and barley were found to infect each of these hosts (Shearer and Wilcoxson, 1977). Oat-adapted isolates from Canada produced larger lesions on detached oat leaves, but were able also to infect detached wheat and barley leaves (Johnston and Scott, 1988).

2.1.3 Pathogen specialization

Host pathogen specialization has been reported for both *S. nodorum* and *S. tritici*. Virulence of a *S. nodorum* wheat isolate decreased on wheat and increased on barley after passage through barley three times. The reverse response was obtained for a barley isolate passed through wheat (Rufty et al., 1981b). Fitzgerald and Cooke (1982) and

Sharma et al. (1982) found similar results for barley isolates passed through wheat, but no change in virulence was observed for wheat isolates passed through barley. Cunfer and Youmans (1983) reported *S. nodorum* isolates from barley and wheat to be highly virulent on their original host, but weakly virulent on the opposite host in reciprocal inoculations. When cultured on oxgall agar, the isolates from wheat fluoresced under near-ultraviolet light, whereas most isolates from barley did not. These nonfluorescing barley isolates possessed colony characteristics that were different from the wheat isolates and were considered, therefore, to be biotypes of *S. nodorum*. A further study found that the barley biotype could be recovered from single spore isolates of the wheat biotype after 2 to 6 passages through wheat (Cunfer et al., 1984).

After successive passages of a *S. nodorum* wheat isolate through *L. multiflorum*, *P. pratense* and *B. sterilis* an increase in virulence on these grass hosts was reported (Harrower, 1977a). As in previous reports, a decrease in virulence on the original host was observed. Ao and Griffths (1976) found that virulence of wheat isolates of *S. nodorum* and *S. tritici* changed following a single passage through an alternative grass host and that the change was similar in direction and magnitude for both species. Cooley et al. (1988) recently developed *S. nodorum* isolates carrying nitrate non-utilizing mutations as genetic markers. Osbourn

et al. (1987) used the isolates to examine virulence changes of *S. nodorum* following passage through barley or wheat. They concluded that the change in virulence resulted not from the isolate adapting to the host, but from cross contamination of isolates followed by selection of the better adapted contaminants.

Specific host-pathogen interactions have been reported for both *S. nodorum* and *S. tritici* (Eyal et al., 1987). However, the occurrence of physiologic specialization remains to be proven. In Florida, researchers found 282 single spore *Septoria nodorum* isolates to have 253 distinct resistance patterns (Allingham and Jackson, 1981). While these results suggest physiologic specialization, they did not permit conventional race differentiation. Rufty et al. (1981a) tested the pathogenicity of nine isolates of *S. nodorum* on four wheat cultivars. A significant cultivar X isolate interaction was obtained, but differences in host response were not great and may have been influenced by the environment. Cultivar X isolate interactions were found among 14 different cultures of *S. nodorum* on 10 spring and winter wheats (Scharen and Eyal, 1983). However, because the magnitude of host response was low and since no isolate was found to be virulent on the uniformly resistant cultivars, pathogenicity patterns could not be classified into defined physiologic races. Virulence frequencies of 33 isolates of *S. nodorum* from eight countries were evaluated on 38 wheat

and triticale cultivars (Scharen et al., 1985). Although cultivar X isolate interactions were not significant, it was estimated that 21 different genes were operative among the cultivars when a gene-for-gene relationship was assumed.

Studies on physiologic specialization in *S. tritici* have given conflicting results. Generally, researchers have been unable to distinguish physiologic races, although, as with *S. nodorum*, isolates may differ in aggressiveness. Contrary to previous reports, Eyal et al. (1973 and 1985b) found that *S. tritici* isolates from Israel display differential interactions on both common wheat and durum. This suggested physiological specialization in the conventional sense. Virulence patterns of 97 *S. tritici* isolates from 22 countries were evaluated on seedlings of 35 wheat and triticale cultivars (Eyal et al., 1985a). Significant cultivar X isolate interactions indicated the existence of specific virulence genes among isolates. Thirteen durum wheats were tested for reaction to 34 isolates of *S. tritici* from seven countries in the Mediterranean area (Van Ginkel and Scharen, 1988). Differences due to cultivars and isolates were highly significant, but the cultivar X isolate interaction was small and not significant. The researchers proposed that isolates varied in aggressiveness and cultivars in horizontal resistance. Van Silfout (1989) tested 48 *S. tritici* isolates collected from *T. aestivum* and *T. durum* in

Asia, Africa, America, and Europe on *T. aestivum* and *T. durum*. Analysis of percent pycnidial coverage showed highly significant differences due to cultivars and isolates. A significant cultivar X isolate interaction was found also, but only 2% of the variance was explained by the interaction.

Physiologic specialization occurs in *S. avenae*. Johnson (1947) separated *S. avenae* f.sp. *triticea* from the morphologically similar *S. avenae* on the basis of its ability to infect and reproduce on wheat and barley.

2.1.4 Morphology

The morphology of the sexual and asexual structures of the three septoria species have been described by Sutton and Waterston (1966a, 1966b), Shipton et al. (1971), Sivanesan (1971), Sanderson, (1972), Wiese (1977), Berggren (1981) and Scharen and Sanderson (1985). A comparison of the structures is given in Table 2.1.

S. nodorum

Ascocarps are immersed, globose, finally depressed, mid-brown to black, 120-200 µ in diameter, with the ostiole slightly papillate. Asci are clavate, cylindrical or curved, shortly stipitate, 8-spored, 47.5-65 x 8-10 µ; the ascus wall is thick and bitunicate. Ascospores are fusoid, subhyaline to pale brown, 3-septate, 20-32 x 4-6 µ, constricted at the septa with the penultimate cell swollen. Pycnidia are immersed, globose, honey brown to dark brown,

Table 2.1 Descriptive comparison of the septoria complex pathogens

	Mycosphaerella graminicola	*Leptosphaeria nodorum*	*Leptosphaeria avenaria f.sp. triticea*
	Teleomorph		
Ascocarp	70-100[1]	120-200	60-130
Ascospore	10-15 x 2-3	20-32 x 4-6	19-28 x 4-6
Number of septa	1	3	3
	Anamorph		
Pycnidium	60-200	140-210	90-150
Conidium	35-98 x 1-3	15-32 x 2-4	26-42 x 2-3
Number of septa	2-5	0-3	3-4

[1]All measurements except number of septa are in microns.

epiphyllous, 140-210 µ in diameter, with the ostiole slightly papillate. Conidia are curved (S-shaped), 0-3 septate, 15-32 x 2-4 µ and rounded on the ends.

S. tritici

Ascocarps are superficially immersed, globose becoming laterally compressed, dark brown, 76-80 x 70-100 µ in diameter, with the ostiole papillate. Asci are obpyriform, bitunicate, 34-41 x 11-13 µ with eight spores that are biseriate and irregularly arranged. No paraphyses are present. Ascospores are two-celled, hyaline, elliptical, with one cell slightly larger and measure 10-15 x 2-3 µ. Pycnidia are immersed, globose to elliptical, honey brown, turning black at maturity, amphigenous, often aggregated and arranged longitudinally between the veins. They are 60-200 µ in diameter, and the ostiole is neither papillate nor protruding. Conidia are hyaline and filiform with 2-5 septa. Typically, the conidia are curved, tapering gradually to an acute apex, measure 35-98 x 1-3 µ and have an obtuse base.

S. avenae **f.sp.** *triticea*

Ascocarps are immersed, globose to subglobose, black, and are 60-130 µ in diameter. The ostiole is round, but not protruding. Asci are narrowly clavate with a rounded apex, 8-spored, and measure 30-100 x 10-18 µ. Ascospores are fusoid, straight or curved, light yellow to olivaceous, 3-septate, measure 19-28 x 4-6 µ and have a swollen penultimate cell. The pycnidia are immersed, globose to

subglobose, brown to black, measure 90-150 µ in diameter and have a rounded ostiole that is slightly elevated. Conidia are hyaline, cylindrical, straight, rounded on the ends, 3-4 septate and measure 26-42 x 2-3 µ.

The three *Septoria* species may be distinguished by their conidial morphology. *S. nodorum* generally has smaller conidia with fewer septa than *S. avenae* f.sp. *triticea*. However, because the ranges of conidial characteristics do overlap, some confusion does occur. *S. tritici* is readily distinguished by its long narrow conidia.

2.1.5 Taxonomy

Taxonomy of the septoria pathogens has been reviewed by Shipton et al. (1971) and Berggren (1981).

The anamorphic forms are classified among the Sphaeropsidales in the Fungi Imperfecti (Deuteromycetes). Conidia or pycnidiospores are formed in ostiolate globose bodies called pycnidia. The teleomorphs or sexual states of these fungi are classified in the Loculoascomycetes. The sexual fruiting bodies, pseudothecia, contain ascospores within bitunicate asci.

Holm (1957) revised the genus *Leptosphaeria* and placed *Leptosphaeria nodorum* and *Leptosphaeria avenaria* f.sp. *triticea* into the genus *Phaeosphaeria*. Based on conidial characters *S. avenae* and *S. nodorum* have been placed in the genus *Stagonospora* (Bissett, 1982a; 1982b).

In an effort to avoid confusion, participants in the

3rd International Workshop on the Septoria Diseases of Cereals (1983) agreed that nomenclature should be based on the teleomorphs, and that commonly used taxa should be maintained, namely *Leptosphaeria nodorum* Muller, *L. avenaria* Weber f.sp. *triticea* T. Johns., and *Mycosphaerella graminicola* (Fuckel) Schroeter. It was also proposed that the common names of the diseases be septoria nodorum blotch, septoria avenae blotch and septoria tritici blotch, that a lower case "s" be used for "septoria", and that septoria nodorum blotch etc. should not be italicized.

2.1.6 Disease symptoms

Symptoms of the septoria diseases of wheat have been reviewed by Weber (1922), Shipton et al. (1971), Wiese (1977) and Zillinsky (1983).

S. nodorum

Lesion development occurs on the glumes and rachis, on leaf blades, leaf sheaths and nodes. Symptoms usually appear first on the lower leaves. Lesions are initially yellowish to tan-brown, oval or lens-shaped spots with darker borders. As the disease develops, lesions enlarge, coalesce and become golden to dark brown or, rarely, grey colored. At this stage light brown pycnidia develop randomly within the necrotic tissue of the lesions. The lesions may eventually cause the death of the entire leaf. The glume blotch stage, if present, generally occurs as the crop approaches maturity. Lesion development starts at the tips of the

glumes and proceeds downward. The lesions, initially brown, eventually become greyish. Pycnidia often develop before the blotch covers the top 1/3 of the glume. Pseudothecia may develop on senescent leaves and/or glumes.

S. tritici

Symptoms of septoria tritici blotch usually develop on leaves and culms, but may occur occasionally on the rachis and the glumes. Lesions generally appear first on the lower leaves as small light-green chlorotic areas which enlarge into elongated, yellowish to light-brown necrotic spots. Lesion growth is restricted by the leaf veins and, therefore, lesions tend to develop longitudinally. The narrow lesions are often used as a diagnostic feature of *S. tritici*. Under suitable environmental conditions lesions extend, coalesce and take on an ash-gray to light-grey color. Pycnidia appear as tiny dark specks embedded in the diseased tissue. Lesions may eventually cover the entire leaf blade, resulting in complete necrosis. Ascocarps may develop in the necrotic tissues of the leaf sheaths and blades.

S. avenae **f.sp.** *triticea*

The symptoms of this pathogen are restricted mainly to the leaf blades and sheaths. Lesions appear first on the lower leaves as small, chocolate-brown spots. Further symptom development is very similar to, and may be confused with, that of *S. nodorum*. Conidial morphology must be used

to distinguish these two pathogens. Ascocarps and pycnidia develop on senescent leaves within the borders of old lesions.

2.2 Disease cycle

The disease cycle of the three septoria pathogens is very similar. Infected crop residue is the most important source of primary inoculum, although infected seed and alternative hosts may also play a role in the epidemiology of these diseases.

The asexual stages of the septoria fungi overwinter on crop residue (Holmes and Colhoun, 1974; Brokenshire, 1975b) with little or no loss of viability (Weber, 1922). Von Wechmar (1966) recovered pycnidiospores from inoculated straw stored outside for eight months and reported a loss of spore viability (due to increased microbial activity) when infected debris was buried below the soil surface for one month. Harrower (1974) found that spores from buried debris showed less than 10% viability after 50 days, while those from surface debris showed 70% viability.

At the beginning of the growing season, during humid or wet conditions, spores within a pycnidium are extruded in a spore mass (cirrus) through the ostiole. Spores in the spore mass are dispersed by rain-splash from the infected residue onto developing plants. In the presence of free water the spores germinate and penetrate the host to initiate infection. Generally, the lower leaves are the first to be

infected. Under suitable environmental conditions, secondary inoculum produced within lesions on the lower leaves is splash dispersed to leaves higher up the plant. Several cycles of inoculum production allow the progression to successive leaf layers (ladder effect) until the flag leaf and, in the case of *S. nodorum*, possibly the head become infected (Shipton et al., 1971; Shaner, 1981; King et al., 1983).

In some areas of the world, the teleomorph may be important in the initiation of infection by *Septoria* spp. Ascocarps may be found on necrotic tissue as the crop approaches or has reached maturity (Hosford et al., 1969; Mehta, 1975; Sanderson et al., 1985; Scott and Sanderson, 1988; Kemp et al., 1989; Mehta, 1989). Ascospores of *L. nodorum* are liberated from pseudothecia on wheat debris throughout most of the season in France (Rapilly et al., 1973). In New Zealand, ascospores of *L. nodorum* and *M. graminicola* were trapped from late autumn to early spring and are considered to be the primary source of inoculum (Sanderson et al., 1985). A similar situation has been described for *M. graminicola* in Australia (Brown et al., 1978). In the United Kingdom, Shaw and Royle (1989a), concluded that primary infection of winter wheat by *S. tritici* occurred in the fall and was the result of evenly dispersed inoculum, probably ascospores.

Ascospores are released in the presence of free water

resulting from rain, dew, fog, and thaw after frost (Sanderson et al., 1985). They may be dispersed over long distances by the wind (Sanderson, 1978).

Occasionally, seed-borne inoculum is an important cause of primary infection (Cunfer, 1981). Seed samples from Georgia had up to 59% infection (Babadoost and Hebert, 1984). Similar tests on seed samples in North Carolina revealed 98.5% infection (Cunfer, 1978). Using the oxgall agar technique (Mathur and Lee, 1978), Le Roux (1989) found 0 to 48% *S. nodorum* seed infection in 207 seed samples collected from six major wheat producing areas in South Africa.

Disease frequency in seedlings from naturally infected seed was closely correlated with the frequency of seed infection (Hewett, 1975). However, subsequent foliar infection was not related to frequency of seed infection (Bateman, 1977; Hewett, 1975; Le Roux, 1989). Although *S. nodorum* survived in seed stored for up to seven years, viability was reduced rapidly after four years (Machacek and Wallace, 1952).

Seed-borne inoculum of *S. tritici* occurs, but it is believed not to be significant in the epidemiology of septoria tritici blotch (King et al., 1983). Seed infection by *S. avenae* f.sp. *triticea* has not been reported.

Epidemics of the septoria diseases have been observed, early in the season, in wheat crops following non-cereal crops, indicating overwintering on alternative hosts (Jenkyn

and King, 1988). Hilu and Bever (1957) and Jenkyn and King (1988) have suggested that *S. nodorum* and *S. tritici* can overwinter on alternative hosts, particularly on grasses. Others (Williams and Jones, 1973; Brokenshire, 1975a; Harrower, 1977; Krupinsky, 1985) have reported the ability of both pathogens to infect wild and forage grass species. Thus, in areas where wheat has not been grown for a number of years, alternative hosts may be important epidemiologically. This aspect of inoculum carryover requires further investigation.

2.3 Role of the environment

The environment plays a major role in the initiation and development of the septoria disease complex. Environmental conditions affect pathogen survival, inoculum production, spore release and dispersal, infection and symptom expression.

Crop residue is the major source of primary inoculum of the septoria species attacking wheat. When infected stubble was alternately wetted and dried, new pycnidia of *S. nodorum* formed (Harrower, 1974; Scharen, 1964; Von Wechmar, 1966) and existing pycnidia produced from six to eight cycles of spores (Scharen, 1964). If stubble remained continuously wet no new pycnidia formed (Scharen, 1964). Pycnidia of *S. tritici* also produced up to 12 cycles of spores, but this fungus did not form new pycnidia while in the saprophytic state of growth (Brokenshire, 1975). Cool

weather extended the period of viability of spores within pycnidia (Shaner, 1981).

Dispersal, both vertical and horizontal, is predominantly by rain-splash. Pycnidiospores of *S. nodorum* were detected in funnel-type spore traps on each of 22 days when rain fell during a two-month period (Griffith and Ao, 1976), but spores were not caught in traps more than 40 cm above ground level. When simulated rain droplets were directed onto straw pieces infected with *S. nodorum*, one drop was estimated to generate 25 spore-carrying droplets, each containing 30 spores (Brennan et al., 1985a). The maximum height reached by spore-carrying splash droplets was 50 cm; simulated wind at 4 m/sec carried such droplets up to 2 m downwind (Brennan et al., 1985b). Shaw (1987) using a splash-meter monitored the upward movement of splash droplets under natural and artificial conditions. The height at which half the area of the rain-splash receptor was covered with dye was strongly dependent on the size of the rain drops. The splash-meter indicated that a sudden outbreak of *S. tritici* in the field coincided with an unusually large amount of rain-splash, caused by a heavy rain three weeks previous.

Royle et al. (1986) observed two patterns of development of *S. nodorum* and *S. tritici* on the foliage of winter wheat. Lesions appeared either simultaneously (sudden outbreaks) on the upper leaf layers or appeared gradually on

successive leaf layers (ladder effect). Sudden outbreaks of disease were attributed to short, heavy rain storms in which inoculum was splashed directly from the basal leaves to the flag or penultimate leaves. The ladder effect, whereby inoculum was splash dispersed vertically by lighter rains, required more time for the upper leaves to become diseased.

Release of ascospores of *L. nodorum* occurred within several minutes of the commencement of rain (Shaner, 1981). Ascospores of *M. graminicola* were discharged within several hours after the commencement of a light rain or at cessation of heavy rain (Brown et al. 1978; Sanderson and Hampton, 1978). Release of ascospores of *M. graminicola* was promoted by dew, fog, or thaw following frost (Sanderson et al., 1985), but more were released following rain (Sanderson and Hampton, 1978).

Relative humidity, temperature, and light affect the infection process and symptom expression. Germination of *S. nodorum* pycnidiospores occurred between 10 and 28°C (Shipton et al., 1971) and relative humidity exceeding 98 per cent was required, but free water was not (Rapilly and Skajennikoff, 1974). Spores within a cirrus gel were protected from desiccation for up to a year (Griffiths and Peverett, 1980), but once free from the gel, spores lost viability rapidly. Pycnidiospores of *S. tritici* germinated at 2 to 37°C, with an optimum at 10 to 30°C (Gheorghies, 1974). Germination time increased as light intensity was

increased from 500 to 10,000 lux, but decreased under a light intensity of 24,000 lux (Benedict, 1971).

Germination of ascospores of *L. nodorum* required free water and temperatures between 0 and 31°C, with an optimum above 25°C (Rapilly et al., 1973). Ascospores of *M. graminicola* germinated on water agar at 19°C and remained viable for up to two weeks, but were quickly killed by bright sunlight (Brown et al., 1978).

Both free moisture and high humidity appear to be essential for infection. However, the moist period required by *S. nodorum* appears to be shorter than that required by *S. tritici*. Only 3 h of high humidity were required for *S. nodorum* to infect mature wheat plants, compared to 20 h for *S. tritici* (Holmes and Colhoun, 1974). Longer moist periods resulted in more lesions and increased disease severity (Holmes and Colhoun, 1974; Eyal et al.,1977; Tomerlin, 1985). The length of the moist period required for a given severity depended on temperature (Holmes and Colhoun, 1974; Hess and Shaner, 1983).

Infection by *S. tritici* has been reported to occur between 16 and 21°C (Morales, 1958), with an optimum at 21°C (Renfro and Young, 1956). No infection occurred at 7°C (Renfro and Young, 1956; Holmes and Colhoun, 1971). Shaw (1989) observed infection by *S. tritici* within the temperature range of 5 to 18°C following lengthy periods of 100% relative humidity. He also observed that interruption

of the high humidity period reduced infection more on resistant than on susceptible cultivars and that the effect increased as the relative humidity during the interruption period decreased.

Jeger et al. (1981) described the minimum criteria for *S. nodorum* infection to occur in the field as:

1) a relative humidity of more than 63% at inoculation; and
2) in the 24 h period following inoculation, a minimum temperature not less than 6°C, at least 4 h with relative humidity greater than 90%, and no more than 4 h with a relative humidity less than 60%.

Shearer and Zadoks (1972) found that *S. nodorum* required a 24 h moist period to induce pycnidial formation following lesion development. They found the latent period of *S. nodorum* to be 10 days when wheat plants were exposed to 12 h of 100% relative humidity alternated with 12 h of 85 to 90% relative humidity following inoculation at 20°C. In a continuously saturated atmosphere at 22°C the latent period was reduced to 6 days (Shearer and Zadoks, 1972).

Pycnidia of *S. nodorum* and *S. tritici* formed in necrotic lesions 10 to 20 days after infection (Hampton et al., 1978; Hilu and Bever, 1957). Near-ultra-violet light promoted pycnidial formation on leaves (Hampton et al., 1978).

Several studies have investigated the relationship

between disease severity and weather parameters. The vertical progression of *S. tritici* from lower to higher leaves and the severity of disease symptoms were correlated with the number of rainy and dewy days (Bahat et al., 1980). The number of septoria lesions also increased with rising rainfall intensity (Jeger et al., 1981). Thomas et al. (1989) identified critical periods suitable for *S. tritici* infection of winter wheat. The periods were identified by occurrences of at least 10 mm of rain on one day or a total of 10 mm or more on two or three consecutive days. Shaw and Royle (1989b) predicted that the final disease levels on winter wheat were strongly dependent on the amount of rain during emergence of the top two leaves.

2.4 Distribution and yield loss

The septoria diseases of wheat have a world-wide distribution (Shipton et al., 1971; King et al., 1983; Eyal et al., 1987), although countries and even localities may differ considerably in the extent to which different *Septoria* species are dominant. In West Germany, *S. nodorum* was reported as the dominant septoria pathogen and has reduced yields by as much as 25-30% (Jones, 1985). In Australia, New Zealand and Brazil, *S. tritici* has occurred most frequently (Shipton et al., 1971; King et al., 1983) and has been associated with yield losses of 19-30% in Australia, 9% in New Zealand (Shipton et al., 1971) and 50% in Brazil (Jones, 1985). In Bulgaria, *S. tritici* was

considered the most prevalent species followed by *S. avenae* f.sp. *triticea* and finally *S. nodorum* (Rodeva, 1989). *S. avenae* f.sp. *triticea* was reported as the predominant septoria pathogen in North Dakota (Hosford et al., 1969).

Since more than one *Septoria* species often occur together, yield reduction must be attributed to the disease complex caused by these pathogens. All three species occur in the United Kingdom. *S. nodorum* has been considered the major pathogen in this area (Jones, 1985), but recent studies indicate that *S. tritici* is now becoming the predominant species (Shaw and Royle, 1989b). Surveys of winter wheat in England and Wales have estimated annual losses, due to *Septoria* spp., of up to 7.4% (King, 1975). The septoria complex may also be important in the Prairie provinces of Canada, where severe epidemics occurred in 1982 and 1983 (G.R. Hughes, personal communication). *S. nodorum*, *S. tritici* and possibly *S. avenae* f.sp. *triticea* were involved.

Yield loss caused by the septoria diseases is mainly due to a reduction in kernel size and weight (shrivelling), and in the number of kernels per head. Grain quality is also lowered as a result of shrivelled kernels and poor grain color (Shipton et al., 1971).

Relationships between disease development and yield have been reported. In the United Kingdom a yield loss of 1.011% has been estimated for each 1% increment of severity

on the flag leaf (at Zadoks GS 75), and a loss of 0.551% for each 1% increment on the penultimate leaf (King et al., 1983). Yield loss caused by *S. tritici* in spring wheat was related to disease severity averaged over the three upper leaves (X) at GS 71 (Ziv and Eyal, 1978); percentage loss in yield was equal to 0.7111X. Thomas et al. (1989) related the area under the disease progress curve (ADPC) for each of the top three leaves of several winter wheat cultivars to yield loss. The ADPC for leaf 2 (flag-1) or leaf 3 (flag-2) was found to be the best indicator of yield loss. King et al. (1983) reported that the greatest yield loss occurred when the flag leaf and the head became infected before the end of grain-filling.

2.5 Methods of control

Strategies used to control the septoria diseases of wheat involve measures based on cultural practices, host resistance and chemical treatments.

2.5.1 Cultural practices

Infected crop residue appears to be the major source of primary inoculum. Therefore, cultural practices that eliminate residue from the soil surface may be effective in reducing disease. Such practices include tillage, crop rotation and burning.

The incorporation of crop residues into the soil increases the rate at which they break down, thus decreasing substrate availability. In addition, burying straw creates a

physical barrier that interferes with the spread of propagules to the above-ground plant parts (Tekauz and Howard, 1988). Tillage operations also eliminate volunteer wheat and alternative hosts that may act as inoculum reservoirs. Harrower (1974) found that pycnidiospores of *S. nodorum* from wheat debris rapidly lost their viability when buried in soil 10 cm below ground level. He proposed ploughing down infected debris soon after harvest as an effective control measure.

Crop rotations that include non-host species also extend the time period for infected residues to decompose. Because of the ability of the *Septoria* species to survive on wheat residue for long periods of time, rotations of three years (two years between wheat crops) or longer have been recommended (Shipton et al., 1971; Luke et al., 1983).

Burning facilitates quick removal of surface debris and thereby reduces the amount of primary *Septoria* inoculum (Brokenshire, 1975a; Harrower, 1974). However, in the light of current soil conservation practices this is no longer recommended in western Canada.

2.5.2 Host resistance

Most of the wheat cultivars grown today are susceptible to septoria diseases (Eyal et al., 1987). Since host resistance is the preferred means of control, the incorporation of resistance into desirable cultivars is of primary importance.

Resistance to the septoria pathogens is more widely distributed among winter than spring wheat cultivars (Eyal, 1981). Early maturing, short strawed cultivars are usually more susceptible than later, taller types (Nelson et al., 1974; Scott, 1973; Tavella, 1978). However, some resistance to both *S. nodorum* and *S. tritici* has been detected in early maturing, short strawed breeding lines (Scott et al., 1982).

Most genetic studies have reported that resistance to *S. nodorum* in adult wheat plants is inherited polygenically (Fried and Meister, 1987). However, two studies have reported genetic control of resistance to be the result of a single gene (Frecha, 1973; Wong and Hughes, 1989). Monogenic, oligogenic and polygenic inheritance have all been reported for resistance to *S. tritici* (Wilson, 1979; Scott et al., 1982; Dannon and Eyal, 1989; Shaner and Buechley, 1989; D.A. Potts, personal communication).

2.5.3 Chemical treatments

Two strategies involving chemical control of the septoria diseases are commonly used. In the first, fungicide protection is used only when specific disease problems arise. This strategy secures high yields from susceptible cultivars. In the second, chemical control is part of a crop management system to control several foliar pathogens (Carmi et al., 1985). The traditional approach has been to use protectant fungicides, but recently, systemic fungicides with curative properties have been developed for

use on wheat.

Dithiocarbamates (maneb, manzate, mancozeb and zineb) are protectant fungicides. This group of fungicides has been effective in controlling septoria diseases, but repeated applications are required at 10-14 day intervals (Eyal et al., 1987). In Florida, a spray program using mancozeb (dithane M-45 or manzate 200) to control septoria nodorum blotch was profitable if begun before full emergence of the flag leaf (Kucharek, 1983). In Germany, captafol is the most widely used protectant fungicide to control septoria nodorum blotch attacking the heads (Fehrmann, 1985). The key to success in using captafol, and other protectant fungicides, is the critical timing of application. If applied late, protectant fungicides are not effective.

Systemic fungicides provide a longer time period of protective action than do protectants. Effective control of the septoria diseases has been reported with benomyl (Benlate), prochloraz (Sportak), triadimefon (Bayleton) and propiconazole (Tilt) (Fehrmann, 1985; Nelson and Philley, 1985; Carmi et al., 1985).

There are two major groups of systemic fungicides: the methylbenzimidazole carbamates (e.g. benomyl) and the ergosterol-biosynthesis inhibitors (e.g. prochloraz, triadimefon, propiconazole and cyproconazole). In New Zealand, a single spray of benomyl at the 4-5 leaf stage was adequate to control septoria tritici blotch (Eyal et al.,

1987). Methylbenzimidazole carbamate (MBC) fungicides are used in Europe to control *Septoria nodorum* on the heads (Eyal et al., 1987). However, there is evidence that their use may lead to the selection of resistance to this group of fungicides. Resistance to benzimidazole in *S. tritici* has been reported in Israel (Zelikovitch et al., 1986).

The effect of the ergosterol-biosynthesis inhibitors (EBI) is to increase the latent period of the pathogen and inhibit pycnidial production. Application of EBI fungicides have been effective in decreasing disease severity and increasing yields. In Germany, propiconazole was found to control septoria nodorum blotch and increase yields (Fehrmann, 1985). In Israel, control of septoria tritici blotch was achieved with two early applications of propiconazole (Carmi et al., 1985). When the fungicide was applied only once, an early application was more effective than a late one.

Mixing protectant and systemic fungicides has been shown to give good control of several foliar pathogens including *Septoria* species (Eyal et al., 1987). This strategy also probably decreases the selection pressure favoring fungicide resistant isolates.

The effects of seed treatment on subsequent disease development have been equivocal. Jenkyn and King (1977) reported improved seedling emergence, decreased septoria nodorum blotch severity on the flag leaf and higher yield

following treatment of seed with mercury compounds. In more recent work, seed treatment with mercury or MBC-type fungicides improved seedling emergence, but had no effect on final disease severity or yield (Fehrmann, 1985).

2.6 Disease development

2.6.1 Disease assessment

The different methods used to assess the level of disease caused by the *Septoria* species has been reviewed by Eyal et al. (1987). The extent of damage was usually evaluated on the basis of the amount of tissue that was affected. These estimates were made in three ways:

1) estimating (counting) the number of pycnidia per unit area,
2) determining the area of diseased tissue, and
3) determining the non-green leaf area, or the remaining green leaf area.

In some cases all three estimates have been combined.

Pycnidial density has been estimated by comparing wheat leaves with standard diagrams (Eyal and Brown, 1976).

Severity of septoria nodorum blotch has been evaluated using a leaf and head infection evaluation scale (Bronnimann, 1968). This method uses standard area diagrams to estimate the percentage of necrotic and chlorotic tissue. James (1971), using an electronic scanner, also developed standard area diagrams for assessing the severity of leaf diseases including those caused by *Septoria* species.

The Horsfall and Barratt (1945) scale has also been used to assess disease severity. This is a logarithmic scale, consisting of 12 grades, that can be fast and accurate.

The Saari-Prescott 0-9 scale (Saari and Prescott, 1975) was developed for foliar diseases, such as the septoria diseases, that "climb up" the plant. The 0-9 scale estimates the extent of infection by measuring the height of disease on the plant. This scale has recently been improved by adding a second digit (also 0-9) which estimates disease severity on the infected leaves (Eyal et al., 1987).

Rosielle (1972) developed a six-point scale to determine resistance of a host to *S. tritici*. The scale ranges from 0 (immune) to 5 (very susceptible).

2.6.2 Analysis of disease progression

2.6.2.1 Disease progress curves

Disease progress curves summarize the effects of host, pathogen and environment on epidemic development, and are frequently investigated with linear regression analysis (Madden, 1986). Because this analysis requires a linear relationship between disease severity and time, transformation to linearize the curves obtained from most disease assessment methods is usually required prior to analysis.

The septoria diseases of wheat are polycyclic in nature i.e. several infection cycles occur during the disease cycle. Disease progress curves are, therefore,

generally sigmoid. Two biological models are frequently used to describe sigmoid disease progress curves, the logistic and the gompertz.

Biological models are based on prior assumptions about the mechanisms of disease increase. For example, the logistic model is based on the assumption that the absolute rate of disease increase is proportional to the level of disease severity and the level of available healthy tissue (Madden, 1986). Further assumptions of the logistic model are:

1) a constant environment,
2) uniformly distributed inoculum,
3) neither pathogen nor host change with time or age, and
4) lesion growth either does not occur or is not an important factor in the increase of diseased tissue.

The biological rationale of the gompertz model is less clear, but may be stated as: relative to plant growth, the pathogen loses equal proportions of its power to increase in equal small intervals of time (Waggoner, 1986).

Some or all of the underlying assumptions are violated in most applications of the logistic equation (Rouse et al., 1981) and other problems have been encountered. For example, the model does not always fit disease progress data, an instantaneous latent period is assumed and the apparent infection rate is difficult to interpret biologically (Jeger, 1985).

In instances where the underlying assumptions of a biological model are violated more emphasis may be placed on statistical (empirical) models. Statistical models are based on the observed set of data with few assumptions about the mechanisms of disease increase. Campbell (1986) stated that "if the principle task is to simply describe disease progression, the model that best fits the observed data is sufficient". In other words statistical models are valid if their purpose is comparative analysis of factor or treatment effects on disease progression. Biological models may also be used in a statistical sense as by Stack (1980) using the logistic model to describe progression of common root rot of wheat.

The logistic model is described by the equation

$$dY/dt = rY(1-Y) \qquad (2.1)$$

and the gompertz model is described by the equation

$$dY/dt = rY(\ln Y) \qquad (2.2)$$

where Y is the disease severity and r is the proportionality constant.

In this form these models describe epidemics that reach 100% severity or have an asymptote that is fixed at 1 or 100. The linear transformation formula for the logistic function is $\ln(Y/(1-Y))$ and for the gompertz function is $-\ln(-\ln(Y))$. Following transformation, the slope and intercept of the line can be used to estimate the rate and the level of disease at time zero, respectively.

The rate parameter is an estimate of the apparent infection rate (AIR) or the speed of the epidemic process measured in units per day (Vanderplank, 1963). If Y is measured as the percentage of diseased tissue and t is time in days, then an AIR of 0.19 indicates that for every percent of diseased tissue there is potential for 0.19 percent more to become diseased per day subject to the availability of healthy tissue.

The logistic model describes a sigmoid curve that is symmetrical about its inflection point (Y=0.5 or 50%), while the gompertz model describes a positively skewed sigmoid disease progress curve (e.g. skewed due to host growth) with an inflection point at Y=0.37 or 37%.

Comparisons of disease progress curves have been made using the standard deviations of the estimated parameters (paired t-test), analysis of variance or multivariate analysis of variance of the parameter estimates for each replication of each treatment (Madden, 1986).

The Horsfall-Barratt scale is a pre-transformed scale that linearizes symmetrical, sigmoid disease progress (Horsfall and Cowling, 1978). If Horsfall-Barratt ratings plotted against time (disease progress curves) are linear, then regression analysis can be conducted on the raw data. If the plot is not linear, then Horsfall-Barratt ratings must be converted into percent disease severity values prior to further analysis.

The progress of foliar diseases is often examined using the logistic and gompertz models. However, few studies have reported using the logistic model to describe progression of the septoria diseases and none using the gompertz model.

Forrer and Zadoks (1983) used the logistic model to transform disease severity of *S. tritici* on winter wheat. No difference in apparent infection rate was found among plots inoculated with different concentrations of conidia. However, a significant difference in the apparent infection rate was detected between inoculated and uninoculated control plots. Furthermore, the time to 50% disease severity significantly increased with decreasing inoculum concentrations.

Burleigh and Loubane (1984) studied the progress of septoria tritici blotch using infected stubble as the source of inoculum. They compared epidemic development in plots of three different sizes, 40 x 40 m, 20 X 20 m and 10 x 10 m. Apparent infection rates, calculated from the logistic model, ranged from 0.159 to 0.067, but were not significantly affected by plot size.

In the United Kingdom, Jenkyn and King (1988) tested the effects of various treatments on perennial ryegrass on development of *Septoria* spp. in a subsequent crop of winter wheat. Percentage disease severity on the flag and flag-1 leaves was assessed once during the 1980 growing season. Prior to statistical analysis the severity values were

transformed to logits. Septoria tritici blotch was most severe in winter wheat after fallow and least severe in wheat that had been direct-drilled after ryegrass. Symptoms of septoria nodorum blotch occurred on the ryegrass, but disease in the subsequent wheat crop was light. Septoria nodorum blotch was more severe on the flag leaves of wheat following grass that had been inoculated with *S. nodorum* conidia than on wheat following grass sprayed with captafol.

2.6.2.2 Model selection

Models used to linearize disease progress curves should be selected on the basis of prior biological knowledge of the host-pathogen system and on how well the data fit the model. Important statistical evaluation criteria include independence of the residuals, realistic and significant estimated parameters, the coefficient of determination (R^2) and the standard deviation about the regression line (S) (Madden, 1986).

It is recognized that disease progress data often violate some of the assumptions of ordinary least squares regression i.e. residuals being normally and independently distributed random variables with mean zero and constant variance. Regression results are not very sensitive to lack of normality or constant variance, but if the variance is highly dependent on the level of disease severity, results may be erroneous (Madden, 1986).

With disease progression data the level of disease at

successive times is often correlated and, therefore, independence of the residuals should always be evaluated. Independence of the residuals can be determined by plotting the residuals (Draper and Smith, 1966) and by estimation of the autocorrelation parameter (P). A model is considered inappropriate if a pattern (e.g. U-shaped or inverted U-shaped) is readily discernible in plots of residuals versus predicted values . The autocorrelation parameter measures correlation of the residuals between successive time periods and varies between -1 and +1 (Madden, 1986). If P does not equal 0 then autocorrelation exists and methods other than ordinary least squares regression should be used to analyze the data. Durbin and Watson (1951) developed a statistical procedure to test the null hypothesis H_o: P=0. Madden (1986) suggested that if the Durbin-Watson statistic is significant and P>0.5, then autocorrelation should be accounted for in the data analysis.

When determining the fit of a disease progression model the R^2 and S values are examined. However, where different transformations have been used on the dependent variable, the R^2 and S values for fitted linear models cannot be directly compared. Comparison of the S values can be made if the observations and corresponding predicted values for each fitted linear model are converted to an equivalent scale (e.g. percent) prior to calculation of the residuals (Waggoner, 1986).

Using these criteria the best fitting model will have realistic and significant estimated parameters, a high coefficient of determination and the lowest S (calculated on an equivalent scale). The parameter estimates are considered accurate if the Durbin-Watson statistic is not significant and the autocorrelation parameter is not greater than 0.5.

2.6.2.3 Area under the disease progress curve

In crops such as wheat where yield is not related to the leaf area at a single time, but rather to the integral of leaf area over time (leaf area duration), area under the disease progress curve (ADPC) may be closely related to yield (Waggoner, 1986). ADPC is usually calculated by trapezoidal integration. A disadvantage of this method is that it does not weight disease development in relation to time. For example, this method may not differentiate light early epidemics from heavy late epidemics, i.e. dissimilar epidemics could have similar ADPCs.

This method of measuring disease progress has been used in several studies of septoria diseases. Fried and Meister (1987) used ADPC to measure disease progress in a study of the inheritance of leaf and head resistance of winter wheat to *S. nodorum*. Plot size effects on the disease progress of *S. tritici* was examined by comparing the ADPC of natural epidemics (Burleigh and Loubane, 1984). The ADPCs from plots 20 x 20 m and 10 x 10 m were similar and significantly less than those from plots 40 x 40 m. In the same study, plot size

had no effect on the apparent infection rates.

Spadafora et al. (1987) studied the effects of septoria nodorum blotch on yield of soft red winter wheat in Pennsylvania. The ADPC was significantly correlated with the yield of cultivars Hart and Tyler in 1985, but not in 1986. Thomas (1989) related yield loss in winter wheat to the area under the *S. tritici* disease progress curve. Regression models incorporating ADPC for any of the top three leaves as independent variables satisfactorily explained yield loss at four sites where disease severity was high.

3. MATERIALS AND METHODS

3.1 Disease development

The objectives of the study were to determine the effects of crop rotation, different cultivars and weather conditions on the natural progression of the septoria disease complex of wheat. Field experiments were conducted in the 1987 and 1988 growing seasons at several locations in the Parkland region of Saskatchewan. Data collected from a 1986 experiment, conducted prior to commencement of this study, were also available for analysis.

1986

Data were available from a single experiment conducted at Weirdale in 1986. The spring wheat cultivars HY320, Columbus, Neepawa, Katepwa, Pembina and Park were seeded into wheat stubble (Table 3.1) with a small-plot seeder on May 26. Plots were 6.1 m x 4.9 m, with 15.2 cm between rows, in a randomized complete block with six replications. The seeding rate was 100 kg/ha and fertilizer was applied according to soil test recommendations. Interplot interference was minimized by maintaining a 2.4 m cultivated strip between experimental units.

1987

Six experiments were conducted in 1987. The cultivars Katepwa, Kenyon, Pembina and Oslo were seeded into wheat stubble at Paddockwood and Weirdale (Table 3.1). At

Table 3.1 Location and cropping history of experimental sites, 1986-1988

Location	Year	Site: Previous crop	Site: Rotation	Site: Years out of wheat
Paddockwood	1987	wheat	W-W[1]	0
Shellbrook	1987	wheat	W-W	0
		fallow	W-SF-W	1
		barley	W-B-W	1
		canola	W-SF-C-W	2
	1988	wheat	W-W	0
		fallow	W-SF-W	1
		canola	W-SF-C-W	2
Weirdale	1986	wheat	W-W	0
	1987	wheat	W-W	0
	1988	wheat	W-W	0
		peal	W-P-W	1
		pea2	W-C-P-W	2

[1]W=spring wheat, B=barley, SF=summerfallow, C=canola, and P=pea. Peal and pea2 indicate a one and two year rotation between wheat crops, respectively.

Shellbrook, the same cultivars were seeded into wheat, barley and canola stubble, and into summerfallow. The sites at Shellbrook were selected to allow comparisons of the following crop rotations: continuous wheat, one year out of wheat, and two years out of wheat (Table 3.1).

The experimental design, plot size and seeding methods were the same as in 1986. Seeding dates were May 15 and 20 for the Paddockwood and Weirdale experiments, respectively. At Shellbrook, the seeding dates were May 26, 27 and June 1 for the summerfallow, barley and canola stubble, and wheat stubble experiments, respectively. All experiments were fertilized according to soil test recommendations and those at Shellbrook were brought to the same fertility level to minimize site effects and allow yield comparisons. At maturity, a 1.2 m strip was harvested from the left side of each plot, using a Hege small-plot combine.

1988

Six experiments were conducted in 1988. Cultivars Kenyon and Pembina were excluded from these experiments to reduce time and labor inputs. Robin, an early maturing cultivar, had not been tested for resistance to the septoria diseases and was therefore included in two of the 1988 trials.

Spring wheat cultivars Katepwa and Oslo were seeded into wheat and canola stubble at Shellbrook and into wheat stubble and pea stubble (pea1) at Weirdale (Table 3.1). In

addition, experiments including cultivars Katepwa, Oslo and Roblin were seeded into summerfallow at Shellbrook and into pea stubble (pea2) at Weirdale. The sites at each location were selected to allow comparisons of three crop rotations: continuous wheat, one year out of wheat, and two years out of wheat (Table 3.1).

Seeding methods were as described for 1986. Seeding dates were May 22 for all Shellbrook experiments and May 27 and 28 for the Weirdale pea and wheat stubble experiments, respectively. As in 1987, the experimental plots at each location were brought to the same fertility level, on the basis of soil tests, to allow yield comparisons.

Fungicide control plots were included in the 1988 experiments to permit a more accurate measurement of disease-related yield loss. The centre 3 m of each control plot was sprayed with propiconazole (Tilt, CIBA GEIGY) at the rate of 125 g ai/ha in 200 L of water at 14-day intervals, from Zadoks GS 14 to GS 77. Fungicide was applied with a model D-201 hand-held sprayer (R & D Sprayers Inc., Opelousas, LA, USA), pressurized with CO_2 at 207 kPa, with a 1.5 m boom fitted with 4 nozzles (model LF3-80^0) 48 cm apart. At maturity, a 2.4 m strip was harvested from the left side of each untreated plot and from the center of each treated plot using a Wintersteiger small-plot combine.

Plot size and row spacing were the same as in 1986. Interplot interference was minimized by seeding wheat into a

2.4 m strip between each experimental unit. These strips were mowed on a regular basis. Each experiment was a split-plot with cultivars as the whole-plot treatments and fungicide application as the sub-plot treatments in a six-replicate randomized complete block design.

3.1.1 Disease assessment

Random 10-plant samples were collected weekly from the centre 2.4 m strip of each untreated plot in 1986 and 1987 and from the right 2.4 m strip of each untreated plot in 1988. Samples were collected between Zadoks GS 14 and senescence of the flag leaf. Occasionally, wet field conditions prevented sampling. Plots were not sampled in the first and third weeks of June in 1986 and in the first week of August at Shellbrook in 1988. Samples were collected, at GS 71 and 75, from the centre 2.4 m strip of each fungicide-treated plot in 1988. To avoid edge effects, plants on the edges of plots were not sampled. After sampling, each plot sample was placed in a plastic bag. The bags were packed in ice and returned to Saskatoon where they were stored at 3° C until the plants were rated for disease severity.

Disease severity was determined by estimating percent necrosis of each of the upper four leaves of each main culm using the Horsfall-Barratt (HB) scale (Horsfall and Barratt, 1945). Leaf layers were designated 1, 2, 3 and 4 for the flag, flag-1, flag-2 and flag-3 leaves, respectively.

The basis of the HB scale is the Weber-Fechner law,

which states that visual acuity is proportional to the logarithm of the intensity of the stimulus. The scale is symmetrical about 50% disease severity and consists of 12 classes:

0	=			0%	diseased tissue
1	=	0	to	3%	diseased tissue
2	=	3	to	6%	diseased tissue
3	=	6	to	12%	diseased tissue
4	=	12	to	25%	diseased tissue
5	=	25	to	50%	diseased tissue
6	=	50	to	75%	diseased tissue
7	=	75	to	88%	diseased tissue
8	=	88	to	94%	diseased tissue
9	=	94	to	97%	diseased tissue
10	=	97	to	100%	diseased tissue
11	=			100%	diseased tissue

Diseased tissue was assessed up to 50% disease severity and healthy tissue was assessed above 50% disease severity.

Horsfall-Barratt values were averaged over the 10 culms to give a mean value for each replicate of each of the four leaf positions within a plot. At each rating, plant developmental stage was assessed on the Zadoks growth scale (Zadoks et al., 1974).

3.1.2 Disease progress

Two approaches were taken in analysing the progression of the septoria disease complex: calculation of the area

under the disease progress curve (ADPC), and curve-fitting. In neither case were the data for the fungicide-treated cultivars in 1988 included because of an insufficient number of disease assessments.

The ADPC was used to examine the progress of disease up the plant (vertical disease progression) and disease progression within each experimental plot. Mean HB values were transformed into percent disease severity values using equations (Table A.1) modified from the conversion equations given by Redman et al. (1969). After transformation, the ADPC was calculated for each replication of each leaf position within a cultivar as:

$$ADPC = \sum_{i=1}^{n} [(Y_{i+1}+Y_i)/2][t_{i+1}-t_i] \qquad (3.1)$$

where Y_i is disease severity at the ith observation, t_i is time (days) at the ith observation, and n is the total number of observations (Shaner and Finney, 1977).

The effect of cultivar on the ADPC was examined on individual leaf layers by analysis of variance. Effects of leaf layer and cultivar on disease progression at each experimental site were examined by repeated measures analysis of variance (Madden, 1986). The effect of rotation on disease progression at Shellbrook in 1987 and at Shellbrook and Weirdale in 1988 was examined by a fixed model combined analyses of variance.

In the curve-fitting approach, the progress of the septoria disease complex on whole plants was examined.

Therefore, mean HB values were averaged over the upper four leaf layers to give a single HB plot mean.

The HB scale gives a very poor estimate of disease when the percentage of diseased tissue is less than 3 or greater than 97 (unpublished data). Furthermore, the larger error associated with such estimates may be magnified by some disease progression models. For example, an attribute of the logistic and gompertz models is that small differences in the level of disease severity below approximately 2% disease produce large differences in the logits and gompits (Rouse et al., 1981). To minimize the risk of using poor disease severity estimates, HB plot means were examined at each sample date. If more than three of the replicates of a cultivar had HB values less than 1 or greater than 10, the six replicates were excluded from any further curve fitting analysis.

Following this selection process the data were fitted to three disease progression models: the statistical Horsfall-Barratt (HB) model, and the biological logistic (Vanderplank, 1963) and gompertz models (Berger, 1981). Since the raw data were HB values, no further data transformation was required for the HB model. The logistic and the gompertz models require disease severity values measured in percent. Therefore, HB plot means were transformed into percent (Table A.1), then logits and

gompits were calculated using the following equations:

$$\text{logit} = \ln(Y/(1-Y)) \quad (3.2)$$

$$\text{gompit} = -\ln(-\ln(Y)) \quad (3.3)$$

where Y is the proportion of disease severity.

Ordinary least squares regression of each of the three dependent variables (HB, logit and gompit) on time (days after June 1) was conducted for each cultivar rotation combination. Subsequently, the following steps were carried out for each model:

1) The slope and location parameters were estimated and tested for significance.
2) The autocorrelation parameter (P) was determined.
3) The Durbin-Watson statistic was calculated and tested for significance.
4) The adjusted coefficient of determination (R^2), the standard deviation about the regression line (S) and the percent standard deviation about the regression line (S%) were calculated.
5) Plots of residuals against predicted values were examined.

Using the criteria presented in section 2.6.2.2, the most appropriate model was chosen to estimate the slope and intercept for each replication of each cultivar. The slope parameter is an estimate of the apparent infection rate (Vanderplank, 1963), while the intercept is the estimate of disease severity at time zero. However, it is difficult to

visualize differences in the intercept estimates measured in HB values, logits or gompits. Therefore, the regression equation was used to determine the location on the time axis where the epidemic reached 50% disease severity. This value is called the T_{50} (Shaner and Finney, 1977) and is calculated from the equation

$$T_{50}=(HB-a)/AIR \qquad (3.4)$$

where HB is the value of 5.5 (50% disease severity), a is the intercept and AIR, the apparent infection rate, is the regression coefficient.

The effect of cultivar on the AIR and the time to 50% disease severity (T_{50}) at each experimental site was examined by analysis of variance. The effect of crop rotation on AIR and T_{50} was examined by combined analyses of variance (fixed model).

3.1.3 Monitoring weather variables

In 1987 and 1988, model CR21, CR21X and CR10 microloggers (Campbell Scientific Canada Inc., Edmonton Alta., Canada) were used to monitor weather variables at all locations. Atmospheric (air) temperature and relative humidity were measured 1.5 m above ground level within a Stevenson screen. Precipitation was measured with a tipping bucket raingauge measuring 0.254 mm per tip (Sierra-Misco Inc. Berkeley, CA, USA). Thermistors, placed at 40 cm above ground level and shielded from direct sunlight, measured canopy temperature. Leaf wetness was measured with 6 x 8 cm

electrical impedance grids coated with two thin layers of white latex paint (Gillespie and Kidd, 1978). The short axis of the sensor surface was rotated approximately 15° from horizontal with the tilted top facing north. The sensors were adjusted weekly to flag leaf height. Leaf wetness sensors were considered wet when they gave a reading of 75% or greater. All sensor readings were recorded at 15 min intervals.

Temperature data were summarized as monthly average minimum temperature and monthly average maximum temperature for both the atmosphere and the crop canopy. Precipitation data were summarized as total monthly precipitation, and monthly frequency of precipitation and leaf wetness data as the duration (in hours) of each leaf wetness period.

3.1.4 Effect of weather on disease development

The effect of precipitation and canopy temperature on progression of the septoria complex on each of the upper four leaf layers of Katepwa was determined. HB data from the wheat stubble sites at each location in 1987 and 1988 were used. Mean HB values for each leaf layer of Katepwa were averaged over the six replicates and were plotted against time (Julian days). These disease progress curves were then related to daily precipitation and daily maximum and minimum canopy temperatures.

3.2 Relative occurrence of the septoria pathogens

The relative occurrence of the septoria pathogens was examined to determine the importance of each pathogen in the disease complex at the different locations in 1987 and 1988. Two methods were used.

3.2.1 Single plot sampling

Samples, consisting of 10 randomly selected main culm leaves, were collected from each plot in the wheat stubble trials at Paddockwood and Shellbrook in 1987. Leaves 3, 2 and 1 (flag-2, flag-1 and flag) were collected from Paddockwood at Zadoks GS 59, 73 and 77, respectively. At Shellbrook, leaf 2 samples were collected at GS 53 and 65, and leaf 1 was collected at GS 77.

In 1988, the wheat and canola stubble trials at Shellbrook and the wheat and pea2 stubble trials at Weirdale were sampled. Leaves 3, 2 and 1 were collected from both locations at GS 59, 73 and 77, respectively.

Leaves from each sample were incubated at room temperature, with a 12 hour photoperiod of cool white fluorescent light, for five days on two layers of moist filter paper in 15 cm diameter petri dishes. Lesions on each leaf were counted and causal fungi were identified by pycnidiospore morphology. The effect of cultivar and pathogen species on lesion number at each location was determined by analysis of variance.

3.2.2 Combined plot sampling

Samples consisting of one main culm leaf were collected from each plot and bulked. In 1987, the barley and canola stubble and summerfallow trials at Shellbrook, and the wheat stubble trial at Weirdale were sampled. Leaves 2 and 1 were collected from Shellbrook at Zadoks GS 59 and 73, respectively. Flag leaves (leaf 1) were collected from Weirdale at GS 67 and 83. The procedures of the single plot sampling method were used for lesion counts and identification of causal fungi. The effect of rotation and pathogen species on lesion number was determined by comparing means and standard errors.

3.3 Grain Yield

After harvest the grain from each plot was air-dried, cleaned and weighed to determine grain yield. Three samples of 250 kernels from each plot were weighed and mean kernel weight determined. The effect of crop rotation and cultivar on kernel weight and yield was determined by analysis of variance.

In 1988, percent yield reductions for each cultivar were calculated as

$$\frac{\text{fungicide check treatment - untreated treatment}}{\text{fungicide check}} \times 100.$$

3.4 Statistical considerations

Homogeneity of error variance was tested prior to performing each combined analysis of variance using

the Bartlett test (Steel and Torrie, 1980). In cases where error variances were not homogeneous, the data were transformed. The Bartlett test was then conducted on the transformed data. The transformation was considered to be adequate if it resulted in a nonsignificant chi-square value.

In those analyses of variance where significant differences were detected between main factors, means separation was based on Duncan's new multiple range test (Steel and Torrie, 1980). Analyses were performed with the ANOVA, GLM and REG procedures of the SAS statistical package (SAS Institute, 1985).

4. RESULTS AND DISCUSSION

4.1 Progress of the septoria disease complex

4.1.1 Area under the disease progress curve

The area under the disease progress curve (ADPC) was calculated for each of the upper four leaves of cultivars in the 1986, 1987 and 1988 experiments.

4.1.1.1 1986 Trial

The area under the disease progress curve was significantly different among cultivars and among leaf layers in 1986 (Table A.2). A highly significant cultivar X leaf layer interaction was also detected, indicating a differential response of cultivars over leaf layer.

The significant cultivar X leaf layer interaction resulted, mainly, from a change in the ranking of cultivars on leaf 4. The biological significance of this interaction seems questionable.

The ADPC was greatest on Park and least on HY320 for all four leaves (Table 4.1). In general, the ranking of cultivars for increasing ADPC was HY320, Columbus, Neepawa, Katepwa, Pembina and Park.

The vertical progression of the disease complex was similar for all cultivars. The ADPC was greatest on leaf 4 and decreased with each successively higher leaf layer (Table 4.1).

Table 4.1 Area under the septoria disease progress curve ($x10^{-2}$) on the upper four leaves of spring wheat cultivars grown on wheat stubble at Weirdale in 1986

	Cultivar						
Leaf[1]	Park	Pembina	Katepwa	Neepawa	Columbus	HY320	Mean
4	34.3a[2]	34.0a	33.5a	34.1a	32.1b	29.1c	31.1A
3	29.5a	27.8b	27.1b	26.6b	25.1d	17.3d	21.0B
2	24.8a	20.4b	19.4bc	18.9c	15.5d	11.7e	11.6C
1	16.2a	10.8b	10.4bc	9.3cd	8.3d	5.8e	2.5D
Mean	26.2a	23.2b	22.6b	22.2c	20.3d	16.0e	

[1] 1=flag leaf, 2=flag-1, etc.
[2] Means followed by the same lower case letter in a row or upper case letter in a column are not significantly different (P=0.05) according to Duncan's new multiple range test.

4.1.1.2 1987 Trials

The area under the disease progress curve differed significantly among cultivars and among leaf layers at all sites in 1987 (Table A.3). The vertical progress of the disease complex was similar on cultivars at all sites; as in 1986, the ADPC was greatest on leaf 4 and decreased with each successively higher leaf (Table 4.2). The leaf layer X cultivar interactions were highly significant. These interactions were a result of either a change in the rank of cultivars or a greater difference among cultivars on leaves 4, 3 and 2 than on leaf 1. When a change in cultivar rank occurred, the difference was significant only at Paddockwood.

The leaf layer X cultivar interaction at Paddockwood was due to a change in the ranking of cultivars on leaf 1. On leaves 4, 3 and 2 the ADPC was greatest for Kenyon and least for Oslo (Table 4.2). However, Oslo had the greatest ADPC on the flag leaf. The ADPC of Pembina was similar to Katepwa on all leaves.

Kenyon had a significantly greater ADPC on leaves 2 and 1 than the other cultivars at Weirdale (Table 4.2). Oslo showed the lowest ADPC on all four leaf layers. Kenyon and Pembina had similar ADPCs on leaf 4. The ADPC of Katepwa was lower than that of Pembina on leaf 4, but the two cultivars had a similar ADPC on the upper three leaves.

The ADPC of Katepwa, Kenyon and Pembina was similar

Table 4.2 Area under the septoria disease progress curve ($x10^{-2}$) on the upper four leaves of spring wheat cultivars grown in different crop rotations at three locations in 1987

Rotation	Leaf[1]	Cultivar: Kenyon	Pembina	Katepwa	Oslo	Mean	SE[2]
Paddockwood							
W-W[3]	4	36.8a[4]	32.4b	31.0b	24.3c	31.1A	0.7
	3	23.9a	21.3b	21.0b	17.7c	21.0B	0.8
	2	14.6a	11.5b	10.3b	10.0b	11.6C	0.7
	1	2.7a	2.1b	1.9b	3.2a	2.4D	0.3
Weirdale							
W-W	4	39.2a	38.6a	37.9b	32.2c	37.0A	0.3
	3	34.8a	32.8ab	31.7b	23.6c	30.7B	0.5
	2	21.7a	18.5b	17.7b	13.8c	17.9C	0.5
	1	10.6a	6.6bc	8.0b	5.8c	7.8D	0.4
Shellbrook							
W-W	4	29.2a	29.7a	28.8a	22.8b	27.6A	0.8
	3	22.4a	22.8a	20.8a	18.5c	21.1B	0.7
	2	15.8a	15.9a	14.5a	10.3c	14.1C	0.6
	1	3.2a	3.4a	4.3a	3.0c	3.7D	0.5
W-SF-W	4	32.1a	29.6b	31.0ab	21.6c	28.6A	0.5
	3	26.2a	21.3b	20.9b	14.4c	20.7B	0.7
	2	17.8a	15.8b	14.8b	6.6c	13.7C	0.5
	1	5.6a	3.6b	3.9b	1.6c	3.7D	0.5
W-B-W	4	31.7a	29.8b	27.8b	20.5c	27.4A	0.6
	3	23.5a	20.4b	20.1b	14.5c	19.6B	0.7
	2	14.2a	12.2ab	10.7b	6.3c	10.9C	1.0
	1	3.2a	2.3ab	1.4bc	1.3c	2.0D	0.3
W-SF-C-W	4	29.2a	23.4b	22.5b	13.1c	22.1A	0.9
	3	18.5a	15.4ab	14.3b	5.8c	13.5B	1.1
	2	7.3a	6.1ab	5.2b	2.0c	5.2C	0.6
	1	2.9a	1.9ab	1.8b	1.0c	1.9D	0.3

[1] 1=flag, 2=flag-1, etc.
[2] SE=standard error.
[3] W=wheat, SF=summerfallow, B=barley and C=canola.
[4] Means followed by the same lower case letter in a row or upper case letter in a column are not significantly different (P=0.05) according to Duncan's new multiple range test.

on each leaf at the Shellbrook wheat stubble site (Table 4.2). As at Weirdale, Oslo had the lowest ADPC on all leaf layers. At the Shellbrook summerfallow, barley and canola stubble sites, the ADPC on the four leaves was greatest for Kenyon and least for Oslo, but the differences were significant in only 6 of the 12 comparisons. The ADPC for Katepwa and Pembina was similar and intermediate between the other cultivars.

Rotation effects on the ADPC were examined by a combined analysis of variance (Gomez and Gomez, 1981) of the data from the Shellbrook sites in 1987 (Table 4.3). The ADPC differed significantly among rotations, cultivars and leaf layers. The cultivar X rotation, leaf layer X rotation, leaf layer X cultivar and leaf layer X cultivar X rotation interactions were highly significant.

The cultivar X rotation interaction resulted from crop rotation having opposite effects on the ADPC of Kenyon and Oslo. Compared to continuous wheat, a rotation of summerfallow between wheat crops reduced the ADPC of Oslo, but increased that of Kenyon (Table 4.4). The leaf layer X cultivar interaction reflected the change in the ranking of Pembina and Katepwa. On leaf 2 the ADPC for Katepwa, averaged over rotations, was greater than for Pembina (means not presented), while on leaf 1 the reverse occurred. However, no significant differences in ADPC were detected between the two cultivars on any of the leaves.

Table 4.3 Combined analysis of variance for area under the septoria disease progress curve on the upper four leaves of spring wheat cultivars grown in different crop rotations at Shellbrook in 1987

Source	df	MS ($x10^{-2}$)
Rotations(R)	3	76580**
Replicates/Rotations	20	1005
Cultivars(C)	3	97737**
C X R	9	2579**
Error A	60	582
Leaf layers(L)	3	991397**
L X R	9	5920**
L X C	9	7275**
L X C X R	27	782**
Error B	240	196

** Significant at P = 0.01.

Table 4.4 Area under the septoria disease progress curve ($x10^{-2}$) for spring wheat cultivars grown in different crop rotations at Paddockwood, Shellbrook and Weirdale in 1987

	Cultivar				
Rotation	Kenyon	Pembina	Katepwa	Oslo	Mean
		Paddockwood			
W-W[1]	19.5a[2]	16.8b	16.1b	13.8c	16.5
		Weirdale			
W-W	26.6a	24.1b	23.8b	18.9c	23.4
		Shellbrook			
W-W	17.6a	17.9a	17.1a	13.6b	16.6A
W-SF-W	20.4a	17.6b	17.6b	11.1c	16.7A
W-B-W	18.1a	16.2b	15.0b	10.6c	15.0B
W-SF-C-W	14.5a	11.7b	10.9b	5.5c	10.6C
Mean	17.7a	15.1b	15.2b	10.2c	

[1]W=wheat, SF=summerfallow, B=Barley, C=canola.
[2]Average of the four upper leaf layers. Means followed by the same lower case letter in a row or upper case letter in a column are not significantly different (P=0.05) according to Duncan's new multiple range test.

The leaf layer X rotation interaction did not involve a change in the ranking of leaf means, but resulted from a lower magnitude of differences at the canola stubble site than at the other sites (Table 4.2).

The analysis also indicated a significant leaf layer X cultivar X rotation interaction. On leaf 4, Pembina had a greater ADPC than Katepwa at all sites, except at the summerfallow site where the ranking was reversed (Figure 4.1). On leaf 1, the ADPC of Katepwa was greater than that of Pembina and Kenyon at the wheat stubble site. However, the ADPC of Katepwa was less than that of Pembina at the barley and canola stubble sites, and was less than that of Kenyon at all three remaining sites (Figure 4.1 and Table 4.2). Furthermore, the effect of a rotation of summerfallow between wheat crops had opposite effects on the ADPCs of Oslo and Kenyon.

The ADPC within leaf layers showed the same trends as the ADPC averaged over leaf layer and cultivar (Table 4.4). The ADPC was greatest at sites with continuous wheat or summerfallow between wheat crops. A one year rotation with barley between wheat crops reduced the ADPC and the lowest ADPC occurred with a rotation of two years out of wheat. Among the cultivars, ADPC was greatest on Kenyon. No difference was found between the intermediate ADPC values of Katepwa and Pembina; Oslo had the lowest ADPC.

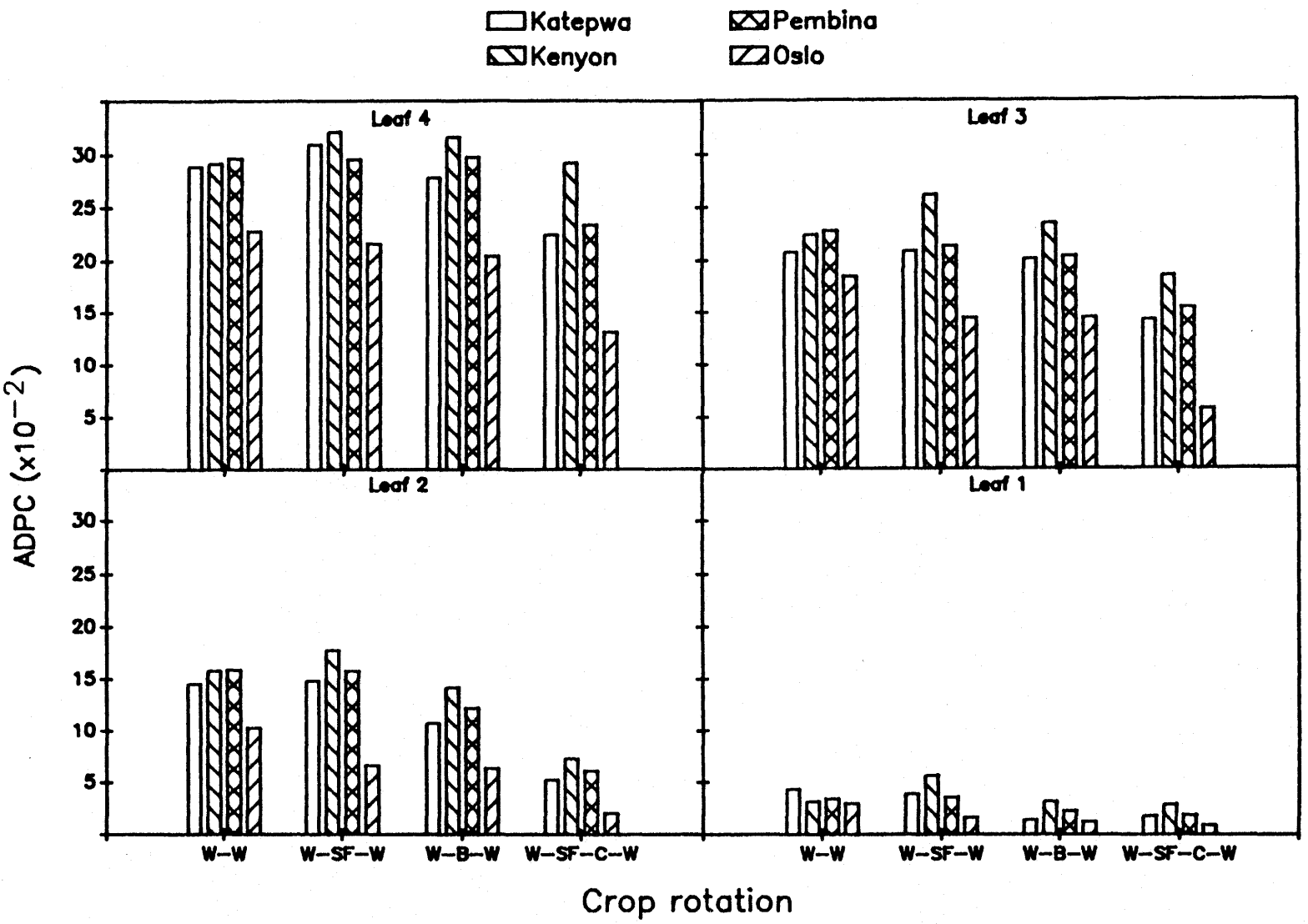

Figure 4.1 The effect of crop rotation on the area under the septoria disease progress curve on the upper four leaves (1=flag, 2=flag-1, etc.) of spring wheat cultivars at Shellbrook in 1987. W=wheat, SF=summerfallow, B=barley and C=canola.

4.1.1.3 1988 Trials

The area under the disease progress curve was significantly different among leaf layers, but not among cultivars at the Shellbrook wheat stubble site in 1988 (Table A.4). Significant differences were detected among cultivars and among leaf layers at the Shellbrook summerfallow and canola stubble sites (Table A.4). The leaf layer X cultivar interactions were significant at all three test sites.

The ADPC on leaves 4 and 3 was greater for Katepwa than Oslo at all three Shellbrook sites, but only three of the six comparisons were significant (Table 4.5). The reverse was true on leaves 2 and 1, although only two of the six comparisons were significant. This change in cultivar ranking from leaf 4 to leaf 1 was responsible for the significant leaf layer X cultivar interactions. At the summerfallow site, the ADPC of Roblin was significantly greater than Katepwa on leaves 4, 2 and 1 and was significantly greater than Oslo on leaves 4 and 3.

The vertical progression of the disease complex was similar to the pattern of development observed in 1986 and 1987.

The effect of rotation on the ADPC was examined by a combined analysis of variance of the Shellbrook data. To maintain a balanced design Roblin was not included in the analysis. The Bartlett test showed the whole unit error

Table 4.5 Area under the septoria disease progress curve ($x10^{-2}$) on the upper four leaves of spring wheat cultivars grown in different crop rotations at two locations in 1988

Rotation	Leaf[1]	Cultivar: Katepwa	Oslo	Roblin	Mean[2]	SE
Shellbrook						
W-W[3]	4	24.8a[4]	20.1b	---	22.4A	0.3
	3	14.9a	13.3a	---	14.1B	1.1
	2	7.1a	8.3a	---	7.7C	0.8
	1	3.9b	7.1a	---	5.5D	0.7
W-SF-W	4	19.6b	18.1b	29.0a	18.9A	1.0
	3	10.2ab	8.5b	11.7a	9.4B	0.7
	2	6.3b	6.7ab	7.6a	6.5C	0.3
	1	2.5b	6.0a	7.3a	4.3D	0.6
W-SF-C-W	4	21.9a	13.1b	---	17.5A	1.2
	3	11.3a	8.2b	---	9.8B	0.8
	2	5.7a	5.9a	---	5.8C	0.4
	1	0.8a	2.3a	---	1.5D	0.7
Weirdale						
W-W	4	27.8a	19.2b	---	22.7A	0.4
	3	19.2a	17.5b	---	15.5B	0.4
	2	9.9a	4.3b	---	7.1C	0.4
	1	2.9a	1.8a	---	2.3D	0.3
W-P-W	4	21.0a	10.4b	20.7a	15.7A	0.4
	3	13.4a	4.7b	13.1a	9.1B	0.3
	2	4.2b	2.0c	5.4a	3.1C	0.2
	1	1.0b	0.9b	4.1a	1.0D	0.1
W-C-P-W	4	22.3a	12.7b	---	17.5A	0.8
	3	13.3a	5.0b	---	9.2B	0.6
	2	3.6a	1.8a	---	2.7C	0.1
	1	0.8a	0.8a	---	0.8D	0.1

[1] 1=flag, 2=flag-1, etc.
[2] Mean of Katepwa and Oslo only. SE=standard error.
[3] W=wheat, SF=summerfallow, C=canola and P=pea.
[4] Means followed by the same lower case letter in a row or upper case letter in a column are not significantly different (P=0.05) according to analysis of variance or Duncan's new multiple range test.

variances to be heterogeneous. An adequate transformation of the data was not found the analysis was therefore conducted on the untransformed data. Significant differences were detected among rotations, cultivars and leaf layers (Table 4.6). The cultivar X rotation interaction was not significant, but the leaf layer X rotation, leaf layer X cultivar and leaf layer X cultivar X rotation interactions were significant.

The leaf layer X rotation interaction did not involve a change in the ranking of the leaf layer means. However, differences among leaf layers were greater at the wheat stubble site than at the other sites. The change in cultivar ranking mentioned previously explains the significant leaf layer X cultivar and leaf layer X cultivar X rotation interactions (Figure 4.2). The latter interaction also resulted from the opposite effect of rotation on cultivars on leaf 4. A rotation of two years out of wheat significantly increased the ADPC of Katepwa and significantly decreased the ADPC of Oslo.

A rotation of one year out of wheat decreased the ADPC on all four leaves of both cultivars (Figure 4.2). A similar result was observed when the ADPC was averaged over leaf layers and cultivars (Table 4.7). A rotation of two years out of wheat further reduced the ADPC on leaves 4 and 1 of Oslo and on leaf 1 of Katepwa (Figure 4.2).

Significant differences in ADPC were found among

Table 4.6 Combined analysis of variance for area under the septoria disease progress curve on the upper four leaves of spring wheat cultivars grown in different crop rotations at two locations in 1988

		MS ($x10^{-2}$)	
Source	df	Shellbrook	Weirdale
Rotations(R)	2	18107**	32911**
Replicates/Rotations	15	920	105
Cultivars(C)	1	3274*	108199**
C X R	2	2457	443
Error A	15	728	174
Leaf layers(L)	3	171637**	213479**
L X R	6	1275**	2357**
L X C	3	9996**	18066**
L X C X R	6	776*	383**
Error B	90	283	76

*, ** Significant at P=0.05 and P=0.01, respectively.

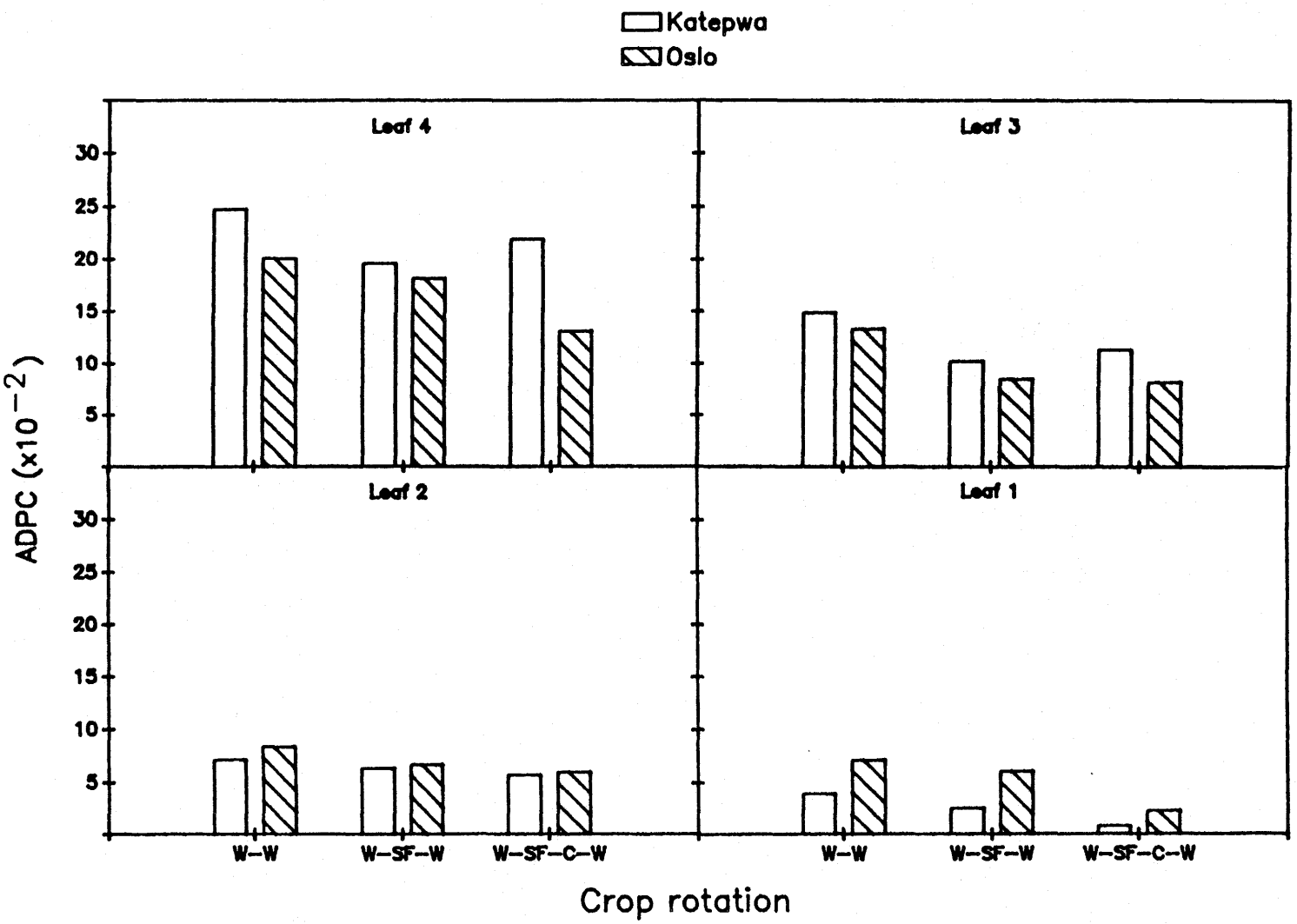

Figure 4.2 The effect of crop rotation on the area under the septoria disease progress curve on the upper four leaves (1=flag, 2=flag-1, etc.) of spring wheat cultivars at Shellbrook in 1988. W=wheat, SF=summerfallow and C=canola.

Table 4.7 Area under the septoria disease progress curve ($x10^{-2}$) for spring wheat cultivars grown in different crop rotations at Shellbrook and Weirdale in 1988

Rotation	Cultivar			
	Katepwa	Oslo	Roblin	Mean[2]
		Shellbrook		
W-W[3]	12.7a[4]	12.2a	---	12.4A
W-SF-W	9.7b	9.8b	13.9a	9.7B
W-SF-C-W	9.9a	7.4b	---	8.7B
Mean	10.8a	9.8b		
		Weirdale		
W-W	15.0a	8.8b	---	11.9A
W-P-W	9.9b	4.5c	10.8a	7.5B
W-C-P-W	10.0a	5.1b	---	7.2B
Mean	11.6a	6.1b		

[1]Average of the upper four leaf layers
[2]Mean of Katepwa and Oslo only.
[3]W=wheat, SF=summerfallow, C=canola and P=pea
[4]Means follwed by the same lower case letter in a row or upper case letter in a column are not significantly different (P=0.05) according to analysis of variance or Duncan's new multiple range test.

cultivars and among leaf layers at the three Weirdale sites (Table A.5). As found at Shellbrook, the cultivar X leaf layer interactions were highly significant.

The ADPC on leaves 4, 3 and 2 was greater for Katepwa than for Oslo at the three Weirdale sites; eight of the nine comparisons were significant (Table 4.5). Katepwa and Oslo had similar ADPCs on leaf 1. At the pea1 stubble site, the ADPC on leaves 4 and 3 was similar for Katepwa and Roblin, however, Roblin had the greatest ADPC on leaves 2 and 1. The ADPC for Katepwa and Oslo, averaged over leaf layers, showed the same trends as the ADPC within leaf layers (Table 4.7). The ADPC of Katepwa was significantly greater than that of Oslo at all three sites, at the pea1 stubble site Roblin had the highest ADPC.

The pattern of vertical progression of the septoria complex was similar to previous experiments (Table 4.5).

When the effect of rotation on ADPC was examined, significant differences were found among rotations, cultivars and leaf layers. The leaf layer X rotation, leaf layer X cultivar and leaf layer X cultivar X rotation interactions were highly significant also (Table 4.6).

The leaf layer X rotation interaction resulted from a larger magnitude of difference among leaf layer means at the wheat site than at the other two sites (means not presented). The leaf layer X cultivar interaction involved a greater difference between cultivars on leaves 4 and 3 than

on leaves 2 and 1 (Table 4.5) and the three-way interaction was a combination of these effects. The difference between cultivars was greater on leaves 4 and 3 than on leaves 2 and 1 and the difference between cultivars on leaves 2 and 1 was greater at the wheat stubble site than at the other two sites (Figure 4.3). None of these interactions involved change in rank of the means.

Compared to continuous wheat, a rotation of one year out of wheat reduced the ADPC, while a rotation of two years out of wheat had no further effect (Table 4.7 and Figure 4.3).

4.1.1.4 Discussion

Examination of the vertical progression of the septoria disease complex required calculation of the area under the disease progress curve (ADPC) for each of the upper four leaves. Vertical progression of the disease complex was similar on all cultivars in all years of the study. Disease always occurred on the lowest leaf first and then spread upward to each successively higher leaf layer. Similar results were obtained for septoria tritici blotch and septoria nodorum blotch by Royle and Shaw (1986).

With the exception of Paddockwood in 1987 and Shellbrook in 1988, analyses of ADPC data indicated that the contribution of the interactions to the total variability was small compared to that of the main effects, leaf layer, cultivar and rotation. However, the interactions did show

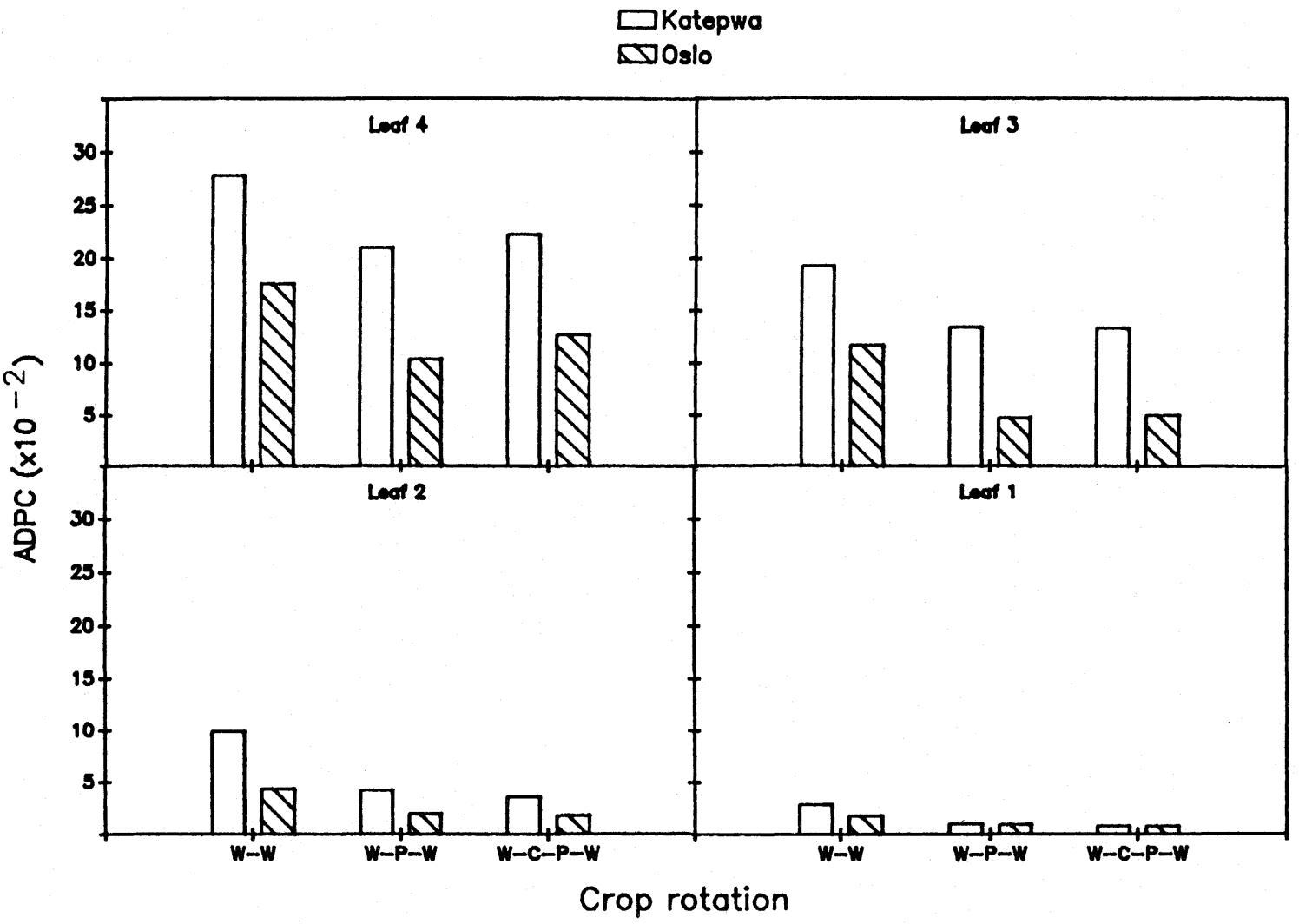

Figure 4.3 The effect of crop rotation on the area under the septoria disease progress curve on the upper four leaves (1=flag, 2=flag-1, etc.) of spring wheat cultivars at Weirdale in 1988. W=wheat, P=pea and C=canola.

that cultivar differences were greater at higher levels of disease. The exception to this trend occurred at the Shellbrook wheat stubble site in 1987 where disease was severe, but few differences were found among cultivars. It is possible that once some threshhold in the level of disease is exceeded the low levels of resistance possessed by the cultivars used in this study are no longer expressed.

In the case of Paddockwood in 1987 and Shellbrook in 1988, meaningful interactions did occur. On the two lower leaf layers the level of disease was greater for Katepwa than Oslo, but the reverse occurred on the flag leaf. Several factors could explain this change in cultivar response. Bahat et al. (1980) and Scott et al. (1985) determined that plant stature and canopy structure had an effect on disease development. For example, semidwarf wheats have shorter distances between leaves and more dense canopies than standard height wheats. Consequently, inoculum is more effectively dispersed from a lower leaf to the next higher leaf, and the microclimate is more favorable for the infection process to occur, on semidwarf compared to standard height wheats. However, if plant stature and canopy structure were responsible for the change in cultivar response observed in this study, then similar changes should have occurred at all or most of the experimental sites.

A more plausible explanation for the change in cultivar response is the difference in time to maturity.

Disease severity was measured as the percentage of necrotic leaf tissue, a method of disease assessment which confounds disease severity with natural senescence. Generally, this is not a problem early in the growing season because leaves die of the disease before they senesce naturally. However, late in the season natural senescence of the flag leaf can occur before the leaf is completely diseased.

Oslo is earlier maturing than Katepwa. Therefore, the risk of confounding natural senescence with disease severity is greater for Oslo than for Katepwa. Plants matured first at Paddockwood and Shellbrook in 1987 and 1988, respectively. Natural senescence may have caused overestimation of the actual disease severity on the flag leaf of Oslo at these two sites, thus resulting in the interactions observed.

All cultivars possessed only low levels of resistance to the septoria disease complex, but some cultivars were more resistant than others. The semidwarf wheats HY320 and Oslo showed the highest level of resistance, while Park, Kenyon and Roblin were the least resistant. Katepwa, Neepawa, Pembina and Columbus were intermediate in resistance.

Analysis of the combined data did not permit separation of rotation effects from other site effects e.g. soil moisture, soil structure, microclimate, etc. However, soil characteristics likely have little effect on the development of foliar diseases, such as the septoria diseases, and sites

within a location were selected as close to one another as possible to minimize climatic variation. Therefore, the major effect on disease was considered to be crop rotation. The similarity of the results from experiments at Shellbrook and Weirdale in 1987 and 1988 support this conclusion.

Rotation had a significant effect on disease development. The most severe septoria epidemics occurred in 1987 and summerfallow between wheat crops did not reduce the level of disease below that of continuous wheat. However, a one year rotation of barley between wheat crops did reduce disease. The greatest reduction in disease was achieved with a two year rotation between wheat crops. In 1988, disease was less severe than in 1987 and a rotation of one year out of wheat substantially reduced the level of disease compared to continuous wheat. A rotation of two years out of wheat had no further effect on the level of disease.

Crop rotation is a means of reducing the level of initial inoculum. This occurs when infected wheat debris becomes buried during subsequent tillage operations, the debris decomposes, or the pathogens are unable to survive the period between host plantings. Trash cover was observed to be heavy at the summerfallow site in 1987. This probably resulted in a large amount of initial inoculum and could be one reason why septoria epidemics were more severe at this site than at the barley stubble site. Also, at the barley stubble site barley debris was partially covering the

remaining wheat debris. This would create a physical barrier that would hinder rainsplash dispersal of conidia and reduce the level of initial infection. Finally, barley is an alternate host of *S. nodorum* and previous studies (Rufty et al., 1981b; Cunfer and Youmans, 1983) have observed a reduction in the virulence of this pathogen on wheat following passage through barley. Therefore, the lower level of disease observed at the barley stubble site may have resulted from a reduction in the virulence of *S. nodorum*.

The greatest reduction in disease resulted from the longest rotation out of wheat. This indicates that under Saskatchewan conditions septoria inoculum survives in wheat debris for a period of one year but is greatly reduced after a period of two years. Similar findings were reported in South Africa where *S. nodorum* survived on surface debris for eight months (Von Wechmar, 1966), and in the United Kingdom where the survival period was up to a year (Harrower, 1974).

When conditions are favorable for disease, as in 1987, a large reduction in initial inoculum is required to obtained some level of control. However, if conditions are unfavorable, as in 1988, weather becomes the major limiting factor in the increase of disease and a smaller reduction in initial inoculum is required for control. For this reason, the level of disease was low at the one and two year rotations sites in 1988, even though the level of inoculum may have been different.

Luke et al. (1983) and Jenkyn and King (1988) also found that under conditions favorable for disease a one year rotation between wheat crops was ineffective in reducing the level of septoria disease.

4.1.2 Rate of disease increase

4.1.2.1 Model selection

The autocorrelation parameter (P) ranged from -0.34 to 0.40, -0.31 to 0.39 and -0.45 to 0.49 for the Horsfall-Barratt (HB), logistic and gompertz models, respectively (Table A.6). Several cultivar and rotation combinations also had a significant Durbin-Watson statistic. However, in all instances of a significant Durbin-Watson statistic the autocorrelation parameter was less than 0.50. Therefore, corrections for autocorrelation were not required.

All parameter estimates were highly significant. The adjusted percentage coefficient of determination (R^2) ranged from 76.8 to 98.9, 72.4 to 98.1 and 58.9 to 98.1 for the HB, logistic and gompertz models, respectively (Table A.6).

The standard deviation about the regression line, was less for the HB model in 23 of the 44 cultivar and rotation combinations (Table A.6). It was less in 10 of the combinations for both the logistic and gompertz models. Occasionally, plots of residuals versus predicted values revealed U-shaped or inverted U-shaped patterns. However, the HB and logistic models produced random patterns more often than the gompertz model.

Because the purpose of the model is to allow a comparative analysis of factor or treatment effects on disease progression, greater emphasis was placed on the statistical fit of the model. The HB model gave the best overall fit to the data and was selected for all further curve-fitting analyses. Using the HB model resulted in realistic and significant estimated parameters, a high R^2, and most often the lowest S%. The logistic model fitted the data adequately, but the underlying assumptions of the model were violated. For example, the environment was not constant, lesion growth could not be discounted as a factor in disease increase and the species composition of the pathogen population was not constant at all sites during the growing season. The gompertz model gave the poorest fit of the three models.

4.1.2.2 Apparent infection rate

The apparent infection rate was determined for each cultivar in the 1986, 1987 and 1988 experiments.

4.1.2.2.1 1986 Trial

Highly significant differences in apparent infection rate (AIR) were found among cultivars in 1986 (Table A.7). The AIR was slowest on the semidwarf HY320, while the remaining cultivars had similar and higher rates (Table 4.8).

4.1.2.2.2 1987 Trials

The AIR differed significantly among cultivars at all

Table 4.8 Apparent infection rate (AIR) and time to 50% disease severity (T_{50}) for the septoria complex on spring wheat cultivars grown in wheat stubble at Weirdale in 1986

	Cultivar					
Parameter[1]	Park	Pembina	Katepwa	Neepawa	Columbus	HY320
AIR	0.188a	0.191a	0.200a	0.191a	0.194a	0.157b
T_{50}	41.9d	45.7c	47.2c	47.3c	49.8b	54.6a

[1]AIR is the regression coefficient of the linear regression of Horsfall-Barratt (HB) values on time. The T_{50}, in days after June 1, was calculated by solving the regression equation T_{50}=(HB-a)/AIR when HB is the value 5.5 (50% disease severity), a is the intercept and AIR is the regression coefficient.

[2]Means followed by the same letter in a row are not significantly different (P=0.05) according to Duncan's new multiple range test.

sites in 1987, except at the Shellbrook wheat and barley stubble sites (Table A.8). Oslo had the fastest AIR at Paddockwood and Weirdale (Table 4.9). At Paddockwood the AIR was significantly slower on Kenyon than on Katepwa or Pembina, but not at Weirdale where the AIR on these cultivars was similar.

Few significant differences in AIR were detected at the Shellbrook sites (Table 4.9). At the summerfallow site, the AIR was slowest on Oslo and similar on Katepwa, Kenyon and Pembina. The fastest AIR at the canola stubble site occurred on Pembina and Katepwa, a significantly slower AIR occurred on Kenyon and Oslo had the slowest rate.

Rotation effects on apparent infection rate were examined by a combined analysis of variance of the Shellbrook data. Heterogeneous error variances were detected by the Bartlett test. Transformation of the data did not result in homogeneous error variances, therefore, analysis was conducted on the untransformed data. The AIR did not vary significantly among rotations (Table 4.10). However, the rotation X cultivar interaction was significant indicating that the AIR on cultivars differed over rotation. For example, the AIR on Oslo was slower at the canola stubble site than at the barley stubble site (Figure 4.4). Conversely, the AIR on Katepwa was faster at the canola stubble site than at the barley stubble site. Rotation did not have an effect on the AIR of Kenyon or Pembina.

Table 4.9 Apparent infection rate (AIR[1]) for the septoria disease complex on the upper four leaves of spring wheat cultivars grown in different crop rotations at three locations in 1987

	Cultivar				
Rotation	Kenyon	Pembina	Katepwa	Oslo	Mean
			Paddockwood		
W-W[2]	0.193c[3]	0.218b	0.218b	0.247a	0.219
			Weirdale		
W-W	0.187b	0.176b	0.188b	0.205a	0.189
			Shellbrook		
W-W	0.242a	0.246a	0.256a	0.258a	0.251A
W-SF-W	0.246a	0.254a	0.245ab	0.235b	0.245A
W-B-W	0.234a	0.239a	0.229a	0.256a	0.240A
W-SF-C-W	0.228b	0.262a	0.265a	0.167c	0.230A
Mean	0.237b	0.250a	0.249a	0.229b	

[1]AIR is the regression coefficient of the linear regression of Horsfall-Barratt values on time.
[2]W=wheat, SF=summerfallow, B=barley and C=canola.
[3]Means followed by the same lower case letter in a row or upper case letter in a column are not significantly different (P=0.05) according to Duncan's new multiple range test.

Table 4.10 Combined analysis of variance for apparent infection rate (AIR) and time to 50% disease severity (T_{50}) for spread of the septoria complex on the upper four leaves of spring wheat cultivars grown in different crop rotations at Shellbrook in 1987

		MS	
Source	df	AIR (x1000)	T_{50}
Rotations(R)	3	1.77	434.1**
Replicates/Rotations	20	0.87	5.9
Cultivars(C)	3	2.40**	450.4**
C X R	9	3.85**	34.8**
Error	60	0.34	4.5

** Significant at P = 0.01.

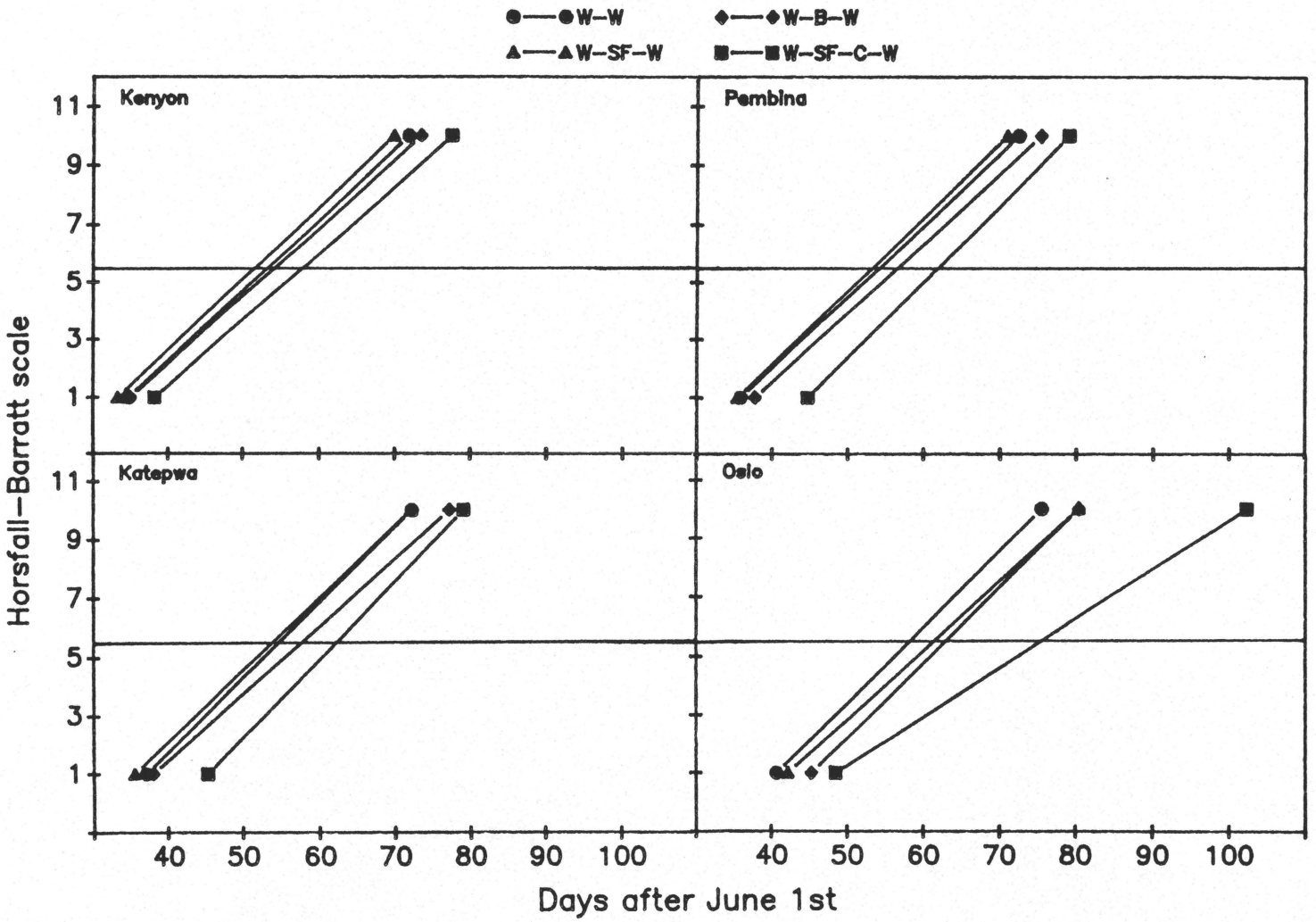

Figure 4.4 Effect of crop rotation on the rate of increase of the septoria complex and time to 50% disease severity (T_{50}) on several spring wheat cultivars at Shellbrook in 1987. The T_{50} is indicated by the point of intersection between the regression lines and the horizontal reference line. W=wheat, SF=summerfallow, B=barley and C=canola.

4.1.2.2.3 1988 Trials

Significant differences in AIR were detected among cultivars at all Shellbrook sites in 1988 (Table A.9). The AIR was much faster on Oslo than on Katepwa at the wheat stubble and canola stubble sites (Table 4.11). However, at the summerfallow site, the AIR was fastest on Katepwa. Roblin showed the slowest rate.

Rotation effects on AIR were examined by a combined analysis of variance of the Shellbrook data (Roblin data excluded). Differences in AIR were highly significant among rotations and cultivars (Table 4.12). The rotation X cultivar interaction was also highly significant. The AIR on cultivars differed over rotations. The AIR on Katepwa was significantly greater at the summerfallow site than at the wheat stubble site, while that on Oslo was significantly less at the summerfallow site than at the wheat stubble site (Figure 4.5 and Table 4.11).

Analysis of the Weirdale data showed significant differences among cultivars at all sites (Table A.10). As observed at the Shellbrook sites, the apparent infection rate was faster on Oslo than on Katepwa at the wheat and pea2 stubble sites (Table 4.11). However, at the pea1 stubble site, Roblin had the fastest AIR, while the AIR on Katepwa was not significantly different from Oslo.

A combined analysis of variance of the Weirdale data (Roblin data excluded) showed significant differences in AIR

Table 4.11 Apparent infection rate (AIR[1]) for the septoria disease complex on the upper four leaves of spring wheat cultivars grown in different crop rotations at Shellbrook and Weirdale in 1988

	Cultivar			
Rotation	Katepwa	Oslo	Roblin	Mean[2]
		Shellbrook		
W-W[3]	0.274b[4]	0.417a	--	0.345A
W-SF-W	0.329a	0.287b	0.248c	0.308B
W-SF-C-W	0.289b	0.339a	--	0.314B
Mean	0.297a	0.348a	--	
		Weirdale		
W-W	0.271b	0.334a	--	0.302A
W-P-W	0.287b	0.279b	0.314a	0.283B
W-C-P-W	0.262b	0.285a	--	0.274B
Mean	0.274b	0.299a	--	

[1]AIR is the regression coefficient of the linear regression of Horsfall-Barratt values on time.
[2]Mean of Katepwa and Oslo only.
[3]W=wheat, SF=summerfallow, C=canola and P=pea.
[4]Means followed by the same lower case letter in a row or upper case letter in a column are not significantly different (P=0.05) according to analysis of variance or Duncan's new multiple range test.

Table 4.12 Combined analysis of variance for apparent infection rate (AIR) and time to 50% disease severity (T_{50}) for the septoria disease complex on the upper four leaves of spring wheat cultivars grown in different crop rotations at Shellbrook and Weirdale in 1988

		MS			
		Shellbrook		Weirdale	
Source	df	AIR (x1000)	T_{50}	AIR (x1000)	T_{50}
Rotations(R)	2	4.85**	74.6**	2.56**	137.2**
Replicates/Rotations	15	0.72	2.2	0.20	0.4
Cultivars(C)	1	22.77**	0.1	5.90**	377.3**
C X R	2	25.85**	6.8	3.79**	1.9
Error	15	0.93	2.1	0.20	0.8

** Significant at P=0.01.

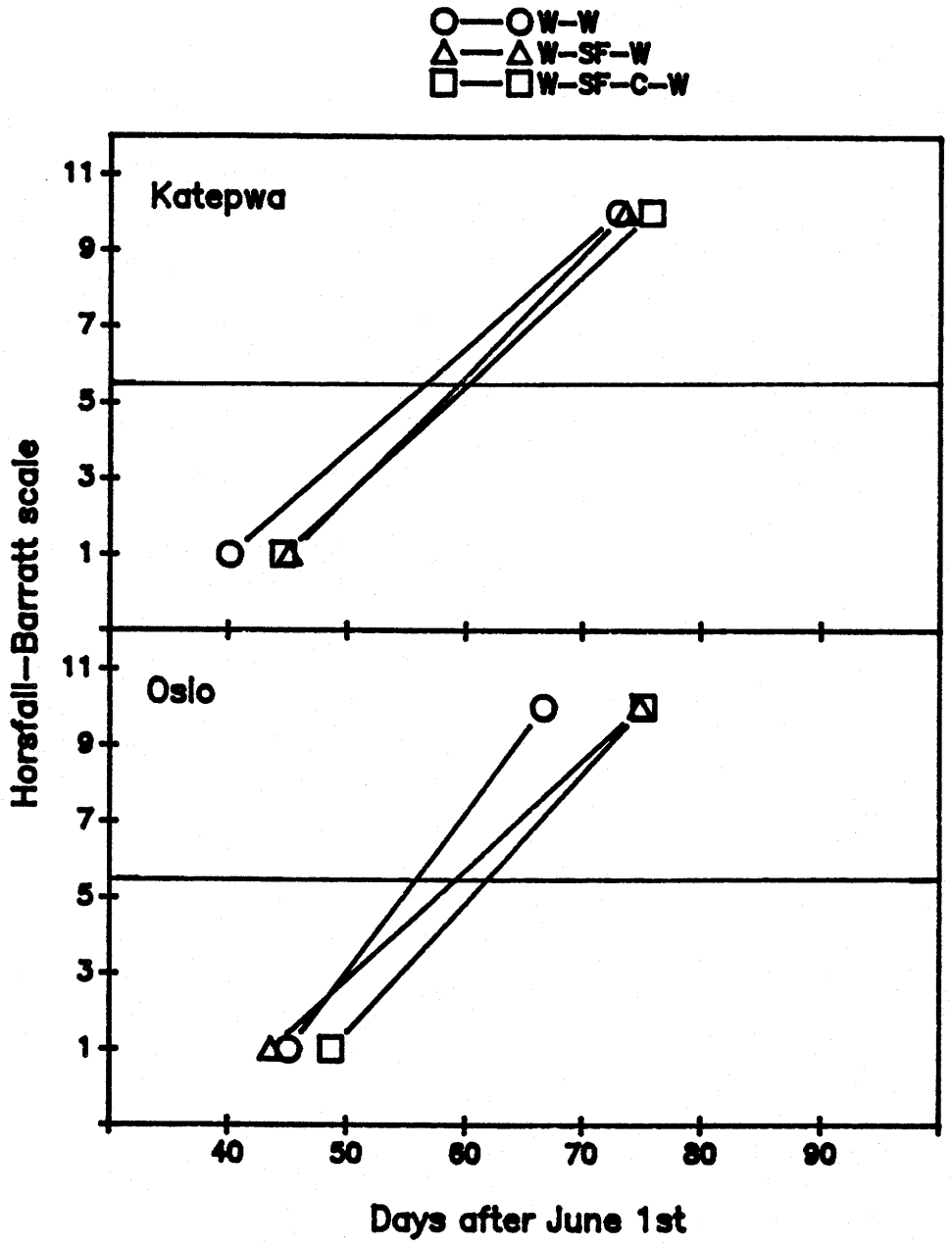

Figure 4.5 Effect of crop rotation on the rate of increase of the septoria complex and time to 50% disease severity (T_{50}) on two spring wheat cultivars at Shellbrook in 1988. The T_{50} is indicated by the point of intersection between the regression lines and the horizontal reference line. W=wheat, SF=summerfallow and C=canola.

among rotations and among cultivars (Table 4.12). In addition, the rotation X cultivar interaction was highly significant.

The AIR was greater on Katepwa at the pea1 stubble site than at the wheat stubble site (Figure 4.6 and Table 4.11). Conversely, the rate of disease increase on Oslo was significantly slower at the pea1 stubble site than at the wheat stubble site. These results were similar to those obtained at Shellbrook.

4.1.2.3 Time to 50% disease severity

4.1.2.3.1 1986 Trial

Significant differences in the time to 50% disease severity (T_{50}) were detected among cultivars in 1986 (Table A.7). The T_{50} was longest for HY320, 55 days, and shortest for Park, 42 days (Table 4.8). The T_{50} for Pembina, Katepwa and Neepawa was similar, while the T_{50} for Columbus was greater than for Neepawa.

4.1.2.3.2 1987 Trials

The T_{50} differed significantly among cultivars at all sites in 1987 and was always significantly longer for Oslo than for the other cultivars tested (Table A.8). The T_{50} for Kenyon was the shortest at all sites and, at most sites, was significantly shorter than Pembina (Table 4.13). Katepwa and Pembina had a similar T_{50}.

Rotation effects on T_{50} at Shellbrook were examined by a combined analysis of variance. Homogeneity of error

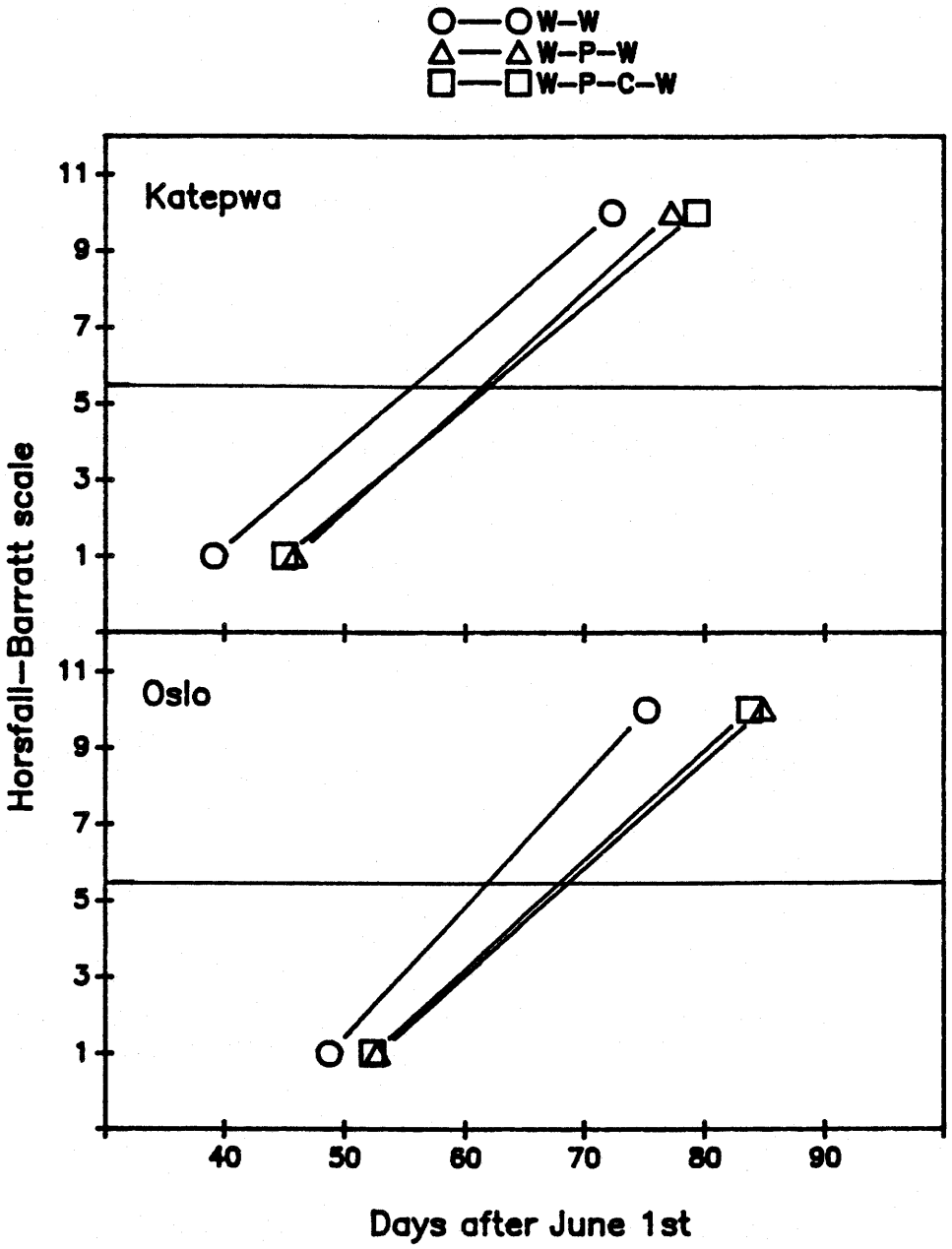

Figure 4.6 Effect of crop rotation on the rate of increase of the septoria complex and time to 50% disease severity (T_{50}) on two spring wheat cultivars at Weirdale in 1988. The T_{50} is indicated by the point of intersection between the regression lines and the horizontal reference line. W=wheat, P=pea and C=canola.

Table 4.13 Time to 50% disease severity (T_{50}[1]) for the septoria disease complex on the upper four leaves of spring wheat cultivars grown in different crop rotations at three locations in 1987

	Cultivar				
Rotation	Kenyon	Pembina	Katepwa	Oslo	Mean
			Paddockwood		
W-W[2]	42.9c[3]	46.7b	47.0b	49.0a	46.4
			Weirdale		
W-W	41.7c	44.7b	45.3b	50.4a	45.5
			Shellbrook		
W-W	53.5c	54.2bc	54.8b	58.1a	55.1C
W-SF-W	51.5c	54.6b	54.7b	61.3a	55.5C
W-B-W	54.4c	56.5b	57.5b	62.8a	57.8B
W-SF-C-W	57.7b	62.0b	62.3b	75.4a	64.3A
Mean	54.3c	56.8b	57.3b	64.4a	

[1]The T_{50}, measured in days after June 1, was calculated by solving the regression equation $T_{50}=(HB-a)/AIR$ where HB is the Horsfall-Barratt value of 5.5 (50% disease severity), a is the intercept and AIR is the regression coefficient.
[2]W=wheat, SF=summerfallow, B=barley and C=canola.
[3]Means followed by the same lower case letter in a row or upper case letter in a column are not significantly different (P=0.05) according to Duncan's new multiple range test.

variances was rejected by the Bartlett test. For the same reason as indicated earlier the analysis was conducted on the untransformed data. Highly significant differences were found among rotations and cultivars and the cultivar X rotation interaction was also significant (Table 4.10). This interaction resulted from a differential response of cultivars over rotations. Kenyon had a shorter T_{50} at the summerfallow site compared to the wheat stubble site, while the reverse was true for Oslo (Table 4.13). Also, the differences among cultivars were greater at the canola stubble site than at the other three sites.

A rotation of barley between wheat crops significantly increased the T_{50} by an average of 2.7 days compared to a rotation of summerfallow between wheat crops or continuous wheat (Table 4.13). A rotation of two years out of wheat resulted in an even further increase in the T_{50}, a 9 day increase over that for continuous wheat.

4.1.2.3.3 1988 Trials

Significant differences in the T_{50} were not found between cultivars at the Shellbrook wheat and canola stubble sites in 1988 (Table A.9). However, significant differences in T_{50} did occur among cultivars at the summerfallow site. The T_{50} was least on Roblin, while Katepwa and Oslo had a similar T_{50} (Table 4.14).

A combined analysis of variance was conducted on the Katepwa and Oslo data from Shellbrook. As in 1987, the error

Table 4.14 Time to 50% disease severity (T_{50}[1]) for the septoria disease complex on the upper four leaves of spring wheat cultivars grown in different crop rotations at Shellbrook and Weirdale in 1988

	Cultivar			
Rotation	Katepwa	Oslo	Roblin	Mean[2]
		Shellbrook		
W-W[3]	56.6a[4]	55.8a	--	56.2C
W-SF-W	60.0a	59.2a	54.2b	59.6B
W-SF-C-W	60.2a	62.0a	--	61.1A
Mean	58.9a	59.0a	--	
		Weirdale		
W-W	55.7b	62.3a	--	59.0B
W-P-W	61.5b	68.7a	58.7c	65.1A
W-C-P-W	62.1b	67.9a	--	64.6A
Mean	59.7b	66.1a	--	

[1] The T_{50}, measured in days after June 1, was calculated by solving the regression equation $T_{50}=(HB-a)/AIR$ where HB is the Horsfall-Barratt value of 5.5 (50% disease severity), a is the intercept and AIR is the regression coefficient.
[2] Mean of Katepwa and Oslo only.
[3] W=wheat, SF=summerfallow, C=canola and P=pea.
[4] Means followed by the same lower case letter in a row or upper case letter in column are not significantly different (P=0.05), according to analysis of variance or Duncan's new multiple range test.

variances were not homogeneous, but for the same reason, the untransformed data were used in the analysis. The analysis showed that the T_{50} differed significantly among rotations, but differences among cultivars were not significant and no cultivar X rotation interaction was detected (Table 4.12). A rotation of one year out of wheat increased the T_{50} by an average of 3.4 days (Table 4.14 and Figure 4.5). A further increase of 1.5 days was achieved with a rotation two years out of wheat.

The T_{50} differed significantly among cultivars at the three Weirdale sites (Table A.10). Oslo had a longer T_{50} than Katepwa at all sites (Table 4.14). Roblin had the shortest T_{50} at the peal stubble site.

The effect of rotation on the T_{50} for Katepwa and Oslo was examined by a combined analysis of variance of the Weirdale data (Table 4.12). Significant differences in the T_{50} were found among rotations and cultivars. The cultivar X rotation interaction was not significant.

Fewer days were required to reach the T_{50} when cultivars were grown in a continuous wheat rotation (Table 4.14 and Figure 4.6). A rotation of one year out of wheat significantly delayed the epidemic development (increased the T_{50}) by an average of 6.1 days. A crop rotation of two years out of wheat had no further effect.

4.1.2.4 Discussion

The apparent infection rate (AIR) of the septoria

complex was determined from the slope of the regression line fitted to Horsfall-Barratt values averaged for the four upper leaves and plotted against time. Linear regression gave a good fit to the data. The adjusted coefficient of determination was greater than 90% for 38 of the 44 cultivar-rotation combinations.

In the three years of this study, the AIR was found to be significantly different among cultivars at all the test sites, except the 1987 Shellbrook wheat and barley stubble sites. In 1986, HY320 had a slower AIR than all other cultivars, indicating a higher level of resistance to the septoria disease complex. In 1987, Oslo had the fastest rate at Paddockwood and Weirdale, but had the slowest rate at the Shellbrook summerfallow and canola stubble sites. The AIR of Kenyon, Pembina and Katepwa was similar at four of the six test sites. Kenyon had a significantly slower AIR than Katepwa and Pembina at Paddockwood and at the Shellbrook canola stubble site. In 1988, the AIR of Oslo was significantly greater than that of Katepwa at four of the test sites. The opposite result was observed at the remaining two sites. Roblin had the slowest rate at the Shellbrook summerfallow site and the fastest rate at the Weirdale peal stubble site.

The increased rate of disease development on Oslo may have resulted from more favorable environmental conditions or from a larger amount of host area available for

infection. The T_{50} data indicated that epidemics occurred later on Oslo than on the other cultivars. Therefore, the different epidemic phases occurred later on Oslo than on the other cultivars and possibly under more favorable environmental conditions. Also, these phases would have occurred during later plant developmental stages and more host tissue would have been available for infection.

Analysis of data sets combined over rotations did not permit separation of the rotation effect from the other site effects. These other effects may have been larger on the AIR data because there was little response to crop rotation. However, the reasoning presented previously does apply to the T_{50} data and the major site effect was considered to be crop rotation.

Crop rotation did not have a significant effect on the AIR in 1987, but a cultivar X rotation interaction was detected. Compared to a one year rotation of barley between wheat crops, a rotation of two years out of wheat increased the AIR of Katepwa, but slowed the AIR of Oslo. As indicated earlier, a change in the virulence of *S. nodorum* could have reduced the AIR on Katepwa at the barley stubble site.

The substantially slower AIR observed for Oslo at the Shellbrook canola stubble site was likely not a result of a reduction in inoculum because a slower AIR was not detected for any of the other cultivars. However, the response may indicate resistance of Oslo to one of the *Septoria* species.

In 1988, rotation had a significant effect on the AIR at both Shellbrook and Weirdale and significant cultivar X rotation interactions were detected at both locations. At Shellbrook and Weirdale a rotation of one year out of wheat increased the AIR of Katepwa and decreased the AIR of Oslo compared to the continuous wheat rotation. The reason for this differential response of cultivars is not known. The unusually high rates of disease development on Katepwa and Oslo at Shellbrook likely resulted from the dry conditions at this test site. Assessing disease severity as the percentage of necrotic tissue confounds senescent tissue with diseased tissue. The heat and drought stress resulting from the weather conditions at Shellbrook caused both cultivars to mature early, especially Oslo. Therefore, the increase in the AIR may have been a measurement of the rate of senescence, due to the effects of drought, and not disease.

The effect of cultivar and crop rotation on the apparent infection rate was not as large as on the area under the disease progress curve and was not consistent from one test site to the next. The smaller effect of cultivar and rotation on AIR resulted in fewer significant differences between treatments than analyses of ADPC.

The time required for the epidemic to reach 50% disease severity (T_{50}) embodies both position and slope of the linear regression of HB values on time. A longer T_{50} would

be associated with a treatment that delays epidemic development. The T_{50} was found to be more consistent and more effective in distinguishing significant differences between treatments than the apparent infection rate. Crop rotation is a form of sanitation that results in a reduction in the initial level of disease (Y_o). Vanderplank (1963) stated that reducing Y_o would delay disease development, but would not affect the AIR. This hypothesis would explain why crop rotation had a small effect on AIR and a large effect on T_{50} in this study.

Analyses of the T_{50} gave results that were similar to the ADPC analyses. In 1986 and 1987, the disease complex reached the T_{50} fastest on Park and Kenyon and slowest on HY320 and Oslo. The T_{50} occurred 12.7 days earlier on Park than on HY320 in 1986. In 1987, the T_{50} occurred 6.1 and 8.7 days earlier on Kenyon than on Oslo at Paddockwood and Weirdale, respectively. At Shellbrook, the T_{50} for Kenyon, averaged over the four test sites, was reached 10.1 days earlier than that of Oslo.

In 1988, the T_{50} was not significantly different among cultivars at the Shellbrook wheat and canola stubble sites. At the summerfallow site, no difference in the T_{50} was detected between Katepwa and Oslo, but the T_{50} for Roblin was significantly shorter, by approximately 5 days, than for the other cultivars. As mentioned earlier, natural senescence probably caused overestimation of disease

severity on Oslo. This would result in a shorter T_{50} and would obscure the differences between Oslo and Katepwa. At Weirdale, the T_{50} was significantly longer for Oslo than for Katepwa at all sites. When averaged over sites the T_{50} occurred 6.4 days earlier on Katepwa than on Oslo.

Cultivar resistance to disease has been expressed as a longer T_{50} (Shaner and Finney, 1977). Therefore, based on the T_{50} values obtained in this study, the most resistant cultivars tested were the semidwarf wheats HY320 and Oslo, although the level of this resistance was quite low. The least resistant cultivars were Park, Kenyon and Roblin. Pembina, Katepwa, Neepawa and Columbus had a level of resistance to the septoria complex that was intermediate between the two previous groups of cultivars.

Crop rotation was found to have a significant effect on T_{50}. Compared to continuous wheat, a rotation of summerfallow between wheat crops in 1987 had no effect on the T_{50} of Pembina and Katepwa, lengthened the T_{50} of Oslo and shortened the T_{50} of Kenyon. A rotation of barley between wheat crops delayed epidemic development by 2.7 days and a rotation of two years between wheat crops resulted in a 9.2 day delay. In 1988, conditions were less favorable for the development of the septoria complex and a one year rotation between wheat crops was more effective in increasing the T_{50} than in 1987. A one year rotation delayed epidemic development an average of 3.4 days at Shellbrook

and 6.1 days at Weirdale. A two year rotation between wheat crops increased the T_{50} a further 1.5 days at Shellbrook, but had no further effect on the T_{50} at Weirdale.

Both the ADPC and the curve-fitting approaches were effective in determining the effects of cultivar and crop rotation on disease progression. Each method detected a similar number of significant differences between treatments. However, the information provided by these methods is not the same. The ADPC approach gives a measurement that is the integral of the amount of disease the crop is exposed to over a given period of time. Curve-fitting provides both an apparent infection rate and the time required for the epidemic to reach a given disease severity. If the purpose of the disease assessment method is strictly treatment comparison, ADPC is likely a better method to use because it is easier to calculate and gives a single measure of disease stress. However, the curve-fitting approach provides a greater amount of information and has proven useful in predicting the severity of epidemics and the effect of disease control practices (Berger, 1988). For example, in a uniform environment, if an average epidemic rate has been determined for a host-pathogen system a measure of the level of disease during the development of an epidemic can be used to predict future levels of disease.

4.1.3 Effect of weather on the septoria complex

4.1.3.1 Weather summary

The effect of weather on the development of the septoria complex was investigated by monitoring environmental conditions at the the experiment locations in 1987 and 1988. Temperature and precipitation were monitored and compared to 30 year averages (1951 to 1980) obtained from the Environment Canada weather station at Prince Albert. These values were determined to be the best available long-term estimates for the Paddockwood, Shellbrook and Weirdale locations.

Precipitation in June of 1987 was below average at Paddockwood and Shellbrook and above average at Weirdale (Table 4.15). In July and August of 1987, precipitation was far above average at all locations. Both Shellbrook and Weirdale received far below average precipitation in June of 1988, but in July of 1988 precipitation was below average at Shellbrook and was well above average at Weirdale. However, Shellbrook received three times the average precipitation in August of 1988.

The frequency of precipitation recorded at the experiment locations in 1987 and 1988 is presented in Table 4.15. In both years, the frequency of precipitation was below average in June, particularly at Weirdale in 1988, and above average in August. In July of 1987, the frequency of rainfall was higher than average, but lower in July of 1988.

Table 4.15 Precipitation amounts and frequencies at all experiment locations in June, July and August of 1987 and 1988 and averages at Prince Albert for the period of 1951-1980

		Precipitation					
		June		July		August	
Year	Location	mm	Days	mm	Days	mm	Days
1987	Paddockwood	52	11	121	13	72	19
	Shellbrook	49	9	99	15	58	18
	Weirdale	89	10	113	15	65	15
1988	Shellbrook	31	10	42	9	153	14
	Weirdale[1]	8	6	95	10	57	12
	Weirdale[2]	7	7	83	10	86	11
Average	Prince Albert	69	12	65	12	52	11

[1] Weirdale wheat stubble site
[2] Weirdale canola stubble site

The monthly average high and low atmospheric temperatures were above average in June of 1987 at all locations (Table 4.16), but below average in July and August of 1987. The monthly average high and low atmospheric temperatures were well above average in June of 1988. In July and August of 1988, the temperatures were close to the long term average.

Canopy temperatures showed the same trends as air temperature only to greater extremes (Table 4.16). Canopy temperatures of the standard height wheats and the semidwarf wheat were similar.

The duration of leaf wetness periods at the flag leaf height was monitored in 1987 and 1988. In both years, the frequency of leaf wetness periods greater than 10 h in duration was higher in July and August than in June (Table 4.17).

Leaf wetness periods greater than 15 h in duration occurred at Paddockwood and Weirdale in all three months of 1987. At Shellbrook in 1987, periods of leaf wetness 15 h or greater in duration occurred in June and July, but not in August. In 1988, leaf wetness periods at least 15 h in duration occurred at Weirdale in July and at both locations in August (Table 4.17). No 15 h leaf wetness periods were recorded in June.

A greater number of leaf wetness periods 10 h or longer in duration occurred in the semidwarf wheat than in the

Table 4.16 Monthly average high and low canopy temperatures (^{o}C) in standard height and semidwarf spring wheat and monthly average high and low atmospheric temperatures (^{o}C) at all experimental locations in June, July and August of 1987 and 1988 and monthly averages at Prince Albert for the period of 1951-1980

		June			July			August		
		Canopy			Canopy			Canopy		
Year	Location	Sh[1]	Sd	Atm	Sh	Sd	Atm	Sh	Sd	Atm
Average high										
1987	Paddockwood	26.6	26.8	23.3	25.1	25.2	21.6	22.2	23.3	19.6
	Shellbrook	27.1	27.8	23.7	27.2	25.4	21.8	23.4	22.9	19.0
	Weirdale	26.3	--	23.3	24.8	--	21.5	23.4	--	18.9
1988	Shellbrook	28.5	28.5	--	29.2	28.5	--	25.8	24.6	--
	Weirdale	29.3	29.5	25.5	27.7	27.1	24.0	24.4	24.4	22.2
Average Prince Albert				21.5			24.2			22.8
Average Low										
1987	Paddockwood	8.2	8.2	9.0	9.0	9.0	8.4	4.5	4.6	5.5
	Shellbrook	8.6	9.1	10.9	9.3	9.4	11.1	5.4	5.5	6.7
	Weirdale	8.7	8.5	10.3	8.6	8.5	9.2	2.6	2.5	6.4
1988	Shellbrook	10.2	10.1	--	8.4	8.3	--	7.5	7.5	--
	Weirdale	9.4	9.4	12.0	8.0	7.8	10.8	7.3	7.1	10.4
Average Prince Albert				7.7			10.6			8.9

[1]Sh=standard height, Sd=semi-dwarf and Atm=atmospheric.

Table 4.17 Frequency of the duration of leaf wetness periods at the flag leaf height of standard height (Sh) and semidwarf (Sd) spring wheat at Paddockwood, Shellbrook and Weirdale in June, July and August of 1987 and 1988

Duration (h)	1987						1988					
	June		July		August		June		July		August	
	Sh[1]	Sd	Sh	Sd	Sh	Sd	Sh	Sh	Sd	Sh	Sd	Sh
Paddockwood												
1 - 10	18	24	19	28	19	12	--	--	--	--	--	--
11 - 20	2	2	14	16	20	20	--	--	--	--	--	--
21 - 30	1	1	1	1			--	--	--	--	--	--
Total >10	3	3	15	17	20	20	--	--	--	--	--	--
Total >15	3	3	3	1	5	4	--	--	--	--	--	--
Shellbrook												
1 - 10	14	13	30	26	25	20	18	19	20	24	19	20
11 - 20	1	2	7	7	7	13	1	1	2	2	10	9
21 - 30	1	1	1	1							1	3
Total >10	2	3	8	8	7	13	1	1	2	2	11	12
Total >15	1	2	3	3	0	0	0	0	0	0	3	4
Weirdale												
1 - 10	17	--	21	--	27	--	11	--	19	--	21	--
11 - 20	2	--	15	--	5	--	1	--	6	--	11	--
21 - 30		--		--		--		--	1	--	1	--
Total >10	2	--	15	--	5	--	1	--	7	--	12	--
Total >15	2	--	3	--	1	--	0	--	0	--	4	--

standard height wheats (Table 4.17). The number of leaf wetness periods 15 h or greater in duration was similar for both standard height and semidwarf wheats.

4.1.3.2 Effect of weather on disease progression

Figures 4.7 and 4.8 show the effect of precipitation on the development of the septoria complex on Katepwa. The pattern of vertical disease progression is also evident. As found with analysis of the area under the disease progress curve data, the disease was initiated on leaf 4 first and on each successively higher leaf layer as time progressed.

The level of disease on the upper four leaves was related to the precipitation that occurred 8-12 days earlier. For example, at Paddockwood in 1987, disease on leaf 4 was initiated 11 days after a rain (Figure 4.7). Rain on days 167, 168 and 171 occurred 9-12 days prior to an increase in disease on leaf 4 and the initiation of the epidemic on leaves 3 and 2. Initial disease on leaves 3 and 2 occurred 11 days after a 12 mm rainfall at Weirdale in 1987 (Figure 4.7). The appearance of disease on the flag leaf followed a series of rains 9-12 days earlier. Similar events were observed at Shellbrook in 1987 (Figure 4.7). A disease increase on leaf 4 and initial disease on leaves 3 and 2 occurred 11 days after a 5 mm rainfall. Rain on days 184 and 185 occurred 10-11 days prior to an increase in disease on leaves 3 and 2.

The conditions in May, June and July of 1988 were not

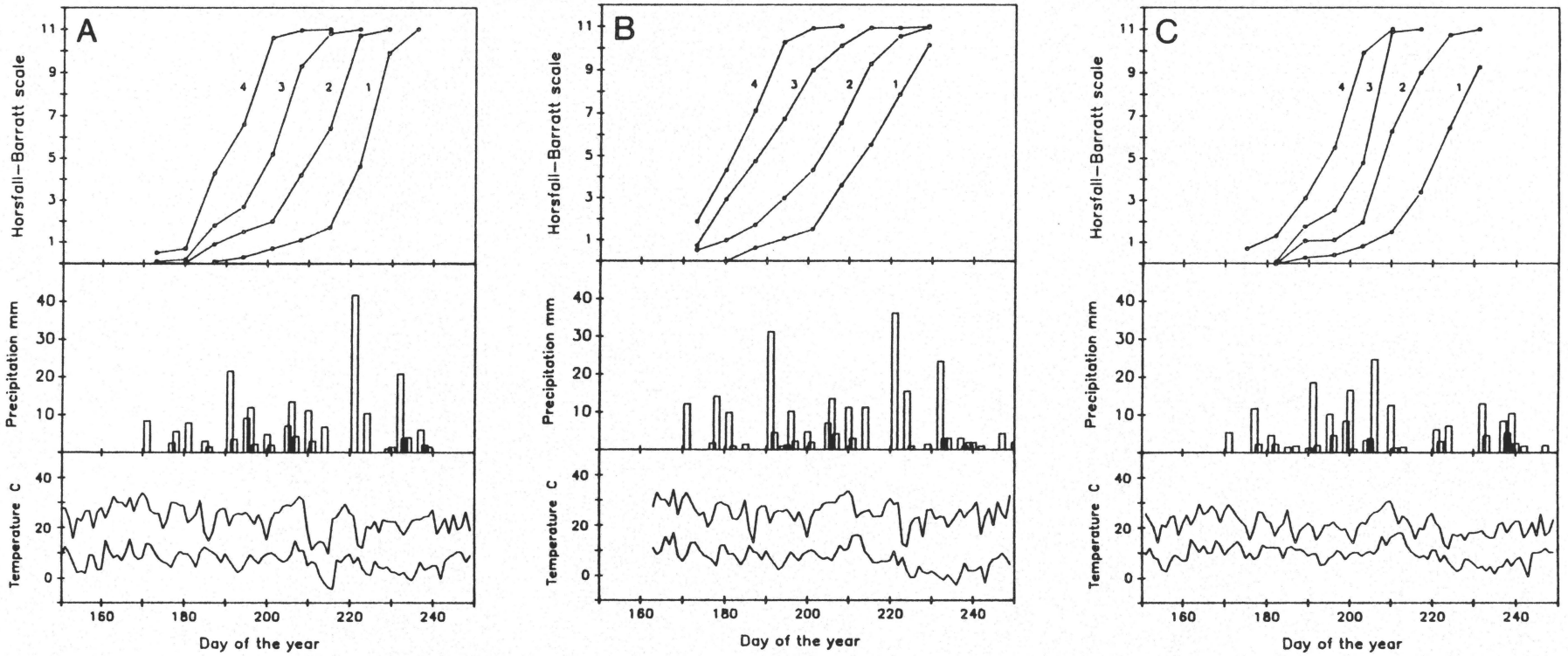

Figure 4.7 Disease progression of the septoria disease complex on the upper four leaf layers (1=flag, 2=flag-1, etc.) of Katepwa, precipitation and high and low canopy temperatures at the wheat stubble test sites in 1987. Bars represent precipitation 10 days prior to the indicted day of the year. **A**) Paddockwood, **B**) Weirdale, **C**) Shellbrook.

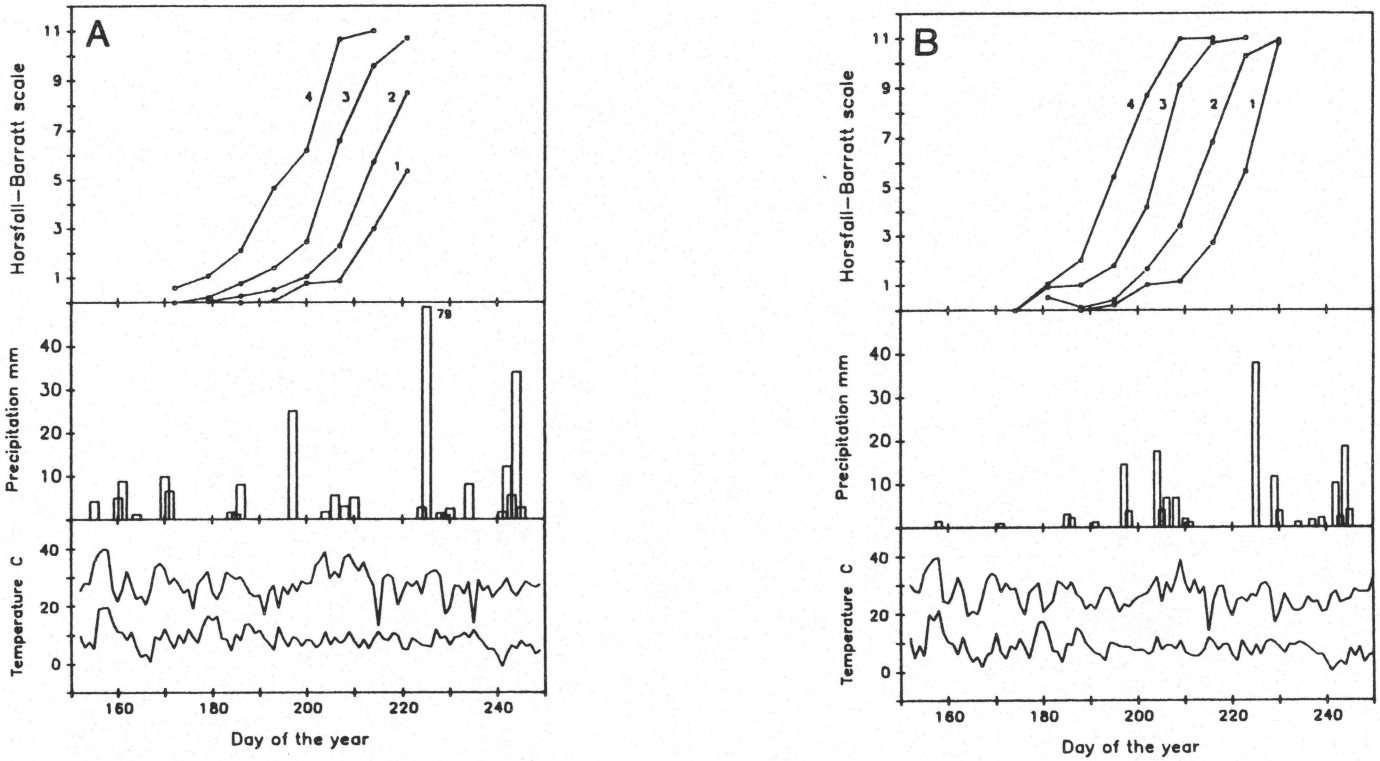

Figure 4.8 Disease progression of the septoria disease complex on the upper four leaf layers (1=flag, 2=flag-1, etc.) of Katepwa, precipitation and high and low canopy temperatures at the wheat stubble test sites in 1988. Bars represent precipitation 10 days prior to the indicted day of the year. **A**) Shellbrook, **B**) Weirdale.

as favorable as in 1987 for the development of the septoria disease complex. Rainfall was below average, especially at Shellbrook (Table 4.15), and canopy temperatures rose to 40°C (Figure 4.8). These conditions not only delayed disease development, but probably confounded disease severity with tissue damage due to heat and drought stress. Therefore, the relationship between precipitation and disease development is not as clear as in 1987.

An increase in disease severity on leaf 4 occurred 10-11 days after two consecutive days of rain (Julian day 160 and 161) at Shellbrook (Figure 4.8) and initial disease on leaf 3 was observed after the same rainfall. A rainfall on day 187 preceded an increase in disease severity on leaves 3 and 2. Disease on the flag leaf reached a plateau between day 200 and 207, even though a 23 mm rain occurred 12 days earlier. This plateau region coincided with a 3 day period where canopy temperatures rose above 35°C.

Initial disease on leaves 4 and 3 occurred following a rainfall of only 1 mm 11 days earlier at Weirdale (Figure 4.8). Rainfall on day 186 and 187 preceded an increase in disease on the penultimate and flag leaves by 9 days. As at Shellbrook, the progress of disease on the flag leaf reached a plateau between day 201 and 209. This plateau also coincided with a period of high canopy temperature.

4.1.3.3 Discussion

Weather conditions were abnormal in 1987 and 1988. In both years, the growing season began with very hot and dry conditions. At most locations, the lower temperatures and higher precipitation that are normally experienced in June occurred in July. However, conditions at the experimental sites in both years were sufficient for the initiation and development of the septoria disease complex, except at Shellbrook in 1988 where dry conditions resulted in poor plant growth and poor disease development.

Disease development on individual leaf layers was influenced by precipitation of at least 1 mm that occurred 8 to 12 days earlier and to canopy temperature. Precipitation is essential for dispersal of pycnidiospores. The results from this study suggest that inoculum dispersal occurred during a rainfall of only 1 mm. This observation supports Shaw's (1987) finding that raindrop size is more important for pycnidiospore dispersal than total amount of rainfall. He measured a mean rainsplash height of 23 cm during a 1 mm rainfall.

The higher frequency of precipitation in 1987 allowed disease increase to proceed on all four leaf layers without interruption. In 1988, fewer rainfall events occurred delaying initial disease development. Disease increase on the flag leaf was delayed for approximately 7 days as a result of a period high canopy temperature. The delay

occurred even though rain fell 12 days earlier, indicating that high temperature was the dominant weather variable. precipitation.

Temperature has an effect on the infection process and on symptom expression of the septoria diseases. In previous studies of pycnidiospore germination, those of *S. nodorum* germinated between 10 and 28°C (Shipton et al., 1971) and those of *S. tritici* between 2 and 37°C (Gheorghies, 1974). Infection of these two pathogens did not occur at temperatures below 6 to 7°C (Renfro and Young, 1956; Holmes and Colhoun, 1971; Jeger et al., 1981).

The monthly average high and low canopy temperatures in June, July and August of 1987 were within the range required for spore germination and infection by both pathogens. However, in June the daily high temperature exceeded 30°C on several days. These temperatures were high enough to prevent germination of *S. nodorum* pycnidiospores and likely resulted in a delay in the epidemic development. In 1988, the monthly average high canopy temperature exceeded 28°C at both locations in June and at Shellbrook in July. These temperatures may have been adequate for the development of septoria tritici blotch, providing sufficient moisture was available, but were unfavorable for the development of septoria nodorum blotch. The extremely high canopy temperatures recorded on Julian days 154 to 158 and 200 to 210 likely prevented spore germination and the infection

process of both pathogens. This would explain the plateau in the development of the disease complex on the flag leaf.

Postinoculation leaf wetness is a critical factor in the establishment of septoria infection (Scharen, 1964; Eyal et al., 1977; Hess and Shaner, 1987). Infection of susceptible spring wheats by *Septoria nodorum* required only 7 h of postinoculation leaf wetness (Eyal et al., 1977), while *S. tritici* required at least 15 h (Renfro and Young, 1956). Leaf wetness in the field is generally a result of precipitation and dew.

Leaf wetness periods of a duration sufficient for infection of wheat by *S. nodorum* occurred in June, July and August of 1987 and 1988. Leaf wetness periods long enough for *S. tritici* to infect wheat occurred in all three months of 1987, but occurred only in August in 1988.

During the 1987 and 1988 growing seasons, precipitation in July appeared to be critical for disease development. Rainfall in July of 1987 resulted in severe epidemic development. Hot and dry conditions in July of 1988 at Shellbrook prevented epidemic development, while cooler temperatures and greater rainfall at Weirdale resulted in moderately severe epidemics.

Under average Saskatchewan conditions, both the amount and frequency of precipitation would be higher in June and less in July and August than observed at most locations in 1987 and 1988. This would probably result in earlier and

more severe epidemics. In the United Kingdom, the critical period for septoria development on winter wheat is mid- to late-May (Tyldesley and Thompson, 1980; Shaw and Royle, 1989b). This period corresponds to the emergence of the upper two leaf layers. Spring wheat is the major crop in Saskatchewan and emergence of the upper two leaves occurs from mid to late June. Therefore, in Saskatchewan, mid to late June may be a critical period of development for the septoria diseases.

4.2 Relative occurrence of the septoria pathogens

4.2.1 Single plot sampling

4.2.1.1 1987 Trials

Pathogens were identified and lesions counted on several leaf layers of cultivars grown in the continuous wheat rotation. Since the leaf layers were sampled at different stages of crop development, the data from each sampling were analyzed separately.

At GS 59, at the Paddockwood location, few *Septoria nodorum* lesions were present on leaf 2 of any of the four cultivars, but by GS 73 a substantial increase in the number of lesions had occurred on all cultivars (Table 4.18). By growth stage 77 the flag leaf of all four cultivars was moderately infected by *S. nodorum*. There were no significant differences in the number of *S. nodorum* lesions on leaf 2 of any of the cultivars at either growth stage or

Table 4.18 Mean number of *Septoria nodorum* (Sn) and *S. tritici* (St) lesions on the flag-1 and flag leaf of spring wheat cultivars grown on wheat stubble at Paddockwood in 1987

	Leaf and Sampling time[1]					
	2 (GS 59)		2 (GS 73)		1 (GS 77)	
Cultivar	Sn	St	Sn	St	Sn	St
Kenyon	8.1a[2]	0	59.8a	6.2a[3]	22.6a	15.8a[3]
Pembina	6.5a	0	53.3a	5.9a	25.3a	16.6a
Katepwa	5.0a	0	47.4a	4.8a	23.1a	15.9a
Oslo	3.8a	0	58.7a	0.5b	28.1a	0.3b

[1] 1=flag, 2=flag-1 and GS=Zadoks growth stage.
[2] Means followed by the same letter in a column are not significantly different (P=0.05) according to Duncan's new multiple range test.
[3] Data transformed to X'=log(X+1) before statistical analysis.

on leaf 1 (Table A.11).

At Paddockwood, lesions caused by *S. tritici* were found on leaf 2 at GS 73 and leaf 1 at GS 77 (Table 4.18). Oslo had significantly fewer *S. tritici* lesions than Kenyon, Pembina or Katepwa (Tables A.11 and 4.18). The number of lesions caused by *S. nodorum* was greater than the number caused by *S. tritici* on both leaf 1 and leaf 2 (Table 4.18).

Lesions caused by *S. avenae* f.sp. *triticea* and *Pyrenophora tritici-repentis*, the tan spot pathogen, were enumerated at all sites in 1987. Since, rarely was more than one lesion identified per leaf; neither pathogen was considered to be important at any of the experimental sites.

Analyses of the Shellbrook data showed no significant differences among cultivars in the number of *S. nodorum* lesions on leaves 3 and 2 (Table A.12 and Table 4.19). However, on leaf 1 at GS 77 there was a significantly greater number of *S. nodorum* lesions on Oslo than on Kenyon, Pembina and Katepwa.

Lesions caused by *S. tritici* were first recorded on leaf 2 at GS 65. On leaf 2 and leaf 1, there were significantly fewer lesions on Oslo than on Kenyon, Pembina or Katepwa (Table 4.19). The largest number of *S. tritici* lesions on the flag leaf was recorded on Pembina and Katepwa.

Table 4.19 Mean number of *Septoria nodorum* (Sn) and *S. tritici* (St) lesions on the upper three leaves of spring wheat cultivars grown on wheat stubble at Shellbrook in 1987

	Leaf and Sampling time[1]					
	3 (GS 53)		2 (GS 65)		1 (GS 77)	
Cultivar	Sn	St	Sn	St	Sn	St
Kenyon	11.0a[2]	0	38.6a	27.5a[3]	27.7b	29.1b[3]
Pembina	8.3a	0	44.8a	24.9ab	36.3b	38.4a
Katepwa	7.2a	0	31.9a	19.9b	29.9b	36.2a
Oslo	6.9a	0	31.2a	0.2c	45.5a	0.2c

[1] 1=flag, 2=flag-1, 3=flag-2 and GS=Zadoks growth stage.
[2] Means followed by the same letter in a column are not significantly different (P=0.05) according to Duncan's new multiple range test.
[3] Data transformed to X'=log(X+1) prior to statistical analysis.

The number of *S. nodorum* lesions on leaf 2 of all cultivars was greater than the number of *S. tritici* lesions (Table 4.19). The number of *S. tritici* lesions increased markedly relative to the number of *S. nodorum* lesions on the flag leaf of all cultivars except Oslo.

4.2.1.2 1988 Trials

In 1988, *Septoria* lesions were counted on three leaf layers of Katepwa and Oslo grown both in a continuous wheat rotation and in a rotation two years out of wheat. No data were obtained for leaves 3 and 2 collected from the Shellbrook canola stubble trial or the Weirdale pea2 stubble trial because the level of disease at GS 59 and 73 was extremely low at both sites. Small lesions were observed on leaves at these sampling times, but attempts to induce the formation of diagnostic pycnidia failed.

The difference in the number of *S. nodorum* lesions was not significant between cultivars on leaves 3, 2 and 1 at Shellbrook (Table A.13). The number of *S. nodorum* lesions was much greater on leaf 2 at GS 73 and leaf 1 at GS 77 than on leaf 3 at GS 59 (Tables 4.20 and 4.21).

No *S. tritici* lesions on leaves 3 and 2 and only few lesions on the flag leaf were identified at both Shellbrook sites (Tables 4.20 and 4.21). Analysis of the flag leaf data indicated that the difference in the number of *S. tritici* lesions between cultivars was not significant at the wheat stubble site (Table A.13).

Table 4.20 Mean number of *Septoria nodorum* (Sn) and *S. tritici* (St) lesions on the flag-2 and flag-1 leaves of spring wheat cultivars grown on wheat stubble at Shellbrook and Weirdale in 1988

	Leaf and Sampling time[1]			
	3 (GS 59)		2 (GS 73)	
Cultivar	Sn	St	Sn	St
Shellbrook				
Katepwa	0.6a[2]	0	27.8a	0
Oslo	0.5a	0	34.8a	0
Weirdale				
Katepwa	2.4a	0	75.1a	0
Oslo	0.6b	0	45.0b	0

[1] 2=flag-1, 3=flag-2 and GS=Zadoks growth stage.
[2] Means followed by the same letter in a column are not significantly different (P=0.05) according to analysis of variance.

Table 4.21 Mean number of *Septoria nodorum* (Sn) and *S. tritici* (St) lesions on the flag leaves, sampled at Zadoks GS 77, of spring wheat cultivars grown in two different crop rotations at Shellbrook and Weirdale in 1988

	Shellbrook					Weirdale				
	S. nodorum			S. tritici		S. nodorum			S. tritici	
Cultivar	W-W[1]	W-SF-C-W	Mean	W-W	W-SF-C-W	W-W	W-C-P-W	Mean	W-W	W-C-P-W
Katepwa	23.9a[2]	7.2a	16.3a	0.1a	1.1	40.6a	11.7a	27.5a	0.5	0.3
Oslo	19.4a	12.0a	16.1a	0.1a	0	30.9b	5.7b	19.4b	0	0
Mean	21.7A	9.6B		0.1	0.5	35.8A	8.6B		0.3	0.2

[1]W=wheat, SF=summerfallow, C=canola and P=pea.
[2]Means followed by the same lower case letter in a column or upper case letter in a row are not significantly different (P=0.05) according to analysis of variance.

As in 1987, only a few lesions caused by *S. avenae* f.sp. *triticea* or *P. tritici-repentis* were found at both locations in 1988. Again, these pathogens were considered not to be a problem.

The effect of rotation on the number of *S. nodorum* lesions was examined by a combined analysis of variance of the Shellbrook flag leaf data. The GLM procedure of the SAS statistical package (SAS Institute, 1985) was used to accommodate the unbalanced experimental design. Significant differences were found between rotations, but not between cultivars (Table 4.22). The cultivar X rotation interaction was also significant.

Compared to the continuous wheat rotation, the rotation of two years out of wheat resulted in 70% and 38% fewer *S. nodorum* lesions on the flag leaf of Katepwa and Oslo, respectively (Table 4.21). The significant cultivar X rotation interaction resulted from a greater effect of rotation on lesion number for Katepwa than for Oslo.

At Weirdale, significant differences in *S. nodorum* lesion number were found between cultivars on leaves 3, 2 and 1 at the wheat stubble site and on leaf 1 at the pea2 stubble site (Table A.14). Very few *S. nodorum* lesions were observed on leaf 3 at GS 59 at the wheat stubble site, but the number of lesions increased on leaves 2 and 1 and was significantly greater for Katepwa than Oslo on all leaves at both sites (Tables 4.20 and 4.21).

Table 4.22 Combined analyses of variance for the number of *S. nodorum* lesions on the flag leaves of spring wheat cultivars grown in two crop rotations at Shellbrook and Weirdale in 1988

		MS	
Source	df	Shellbrook	Weirdale
Rotations(R)	1	795.2**	4017.5**
Replicates/Rotations	9	38.0	10.2
Cultivars(C)	1	0.1	343.7**
C X R	1	116.6*	16.8
Error	9	17.0	15.5

*, ** significant at P = 0.05 and P = 0.01, respectively.

No *S. tritici* lesions were detected on leaves 3 and 2 and only few were detected on leaf 1 of Katepwa (Tables 4.20 and 4.21). No *S. tritici* lesions were observed on any leaves of cultivar Oslo. Statistical analyses were not conducted on the *S. tritici* data from Weirdale because of the high number of zero values.

The effect of rotation on the number of *S. nodorum* lesions was examined using the same analysis as used for the Shellbrook data. A highly significant difference in lesion number was found between crop rotations and between cutivars, but the cultivar X rotation interaction was not significant (Table 4.22). Thus, the effect of rotation on lesion number was similar for both cultivars.

As found at Shellbrook, a crop rotation with two years out of wheat resulted in a substantial reduction in the number of *S. nodorum* lesions compared to continuous wheat (Table 4.21). The number of *S. nodorum* lesions on the flag leaf was reduced by 71% on Katepwa and 82% on Oslo.

4.2.2 Combined plot sampling

In the combined plot sampling method, one leaf per plot was collected and the leaves were bulked. The number of lesions of *S. nodorum* and *S. tritici* was compared by calculating and comparing means and standard errors from the data of each site (Table 4.23). Combined plot sampling was conducted only in 1987.

Table 4.23 Mean and standard error of the number of *Septoria nodorum* and *S. tritici* lesions on the bulked upper two leaves of cultivars grown in different crop rotations at Shellbrook and Weirdale in 1987

Rotation	Time of sampling	*Septoria nodorum*	*Septoria tritici*	Number of leaves sampled
		Shellbrook Leaf 2		
W-SF-W[1]	59[2]	16.8 ±3.5	2.1 ±0.2	22
W-B-W	59	36.0 ±3.1	2.1 ±0.6	23
W-SF-C-W	59	2.8 ±1.2	2.1 ±0.7	21
		Shellbrook Leaf 1		
W-W	77	34.8 ±2.0	25.9 ±1.7	24
W-SF-W	77	26.2 ±3.1	10.1 ±1.9	24
W-B-W	77	17.7 ±1.7	19.5 ±3.2	24
W-SF-C-W	77	14.7 ±2.1	30.6 ±4.6	23
		Weirdale Leaf 1		
W-W	63	12.0 ±5.4	1.0 ±0.3	24
W-W	83	69.8 ±4.3	4.2 ±1.2	13

[1] W=wheat, SF=summerfallow, B=barley and C=canola.
[2] Zadoks growth stage.

On leaf 2, sampled at growth stage 59, the number of *S. nodorum* lesions was greater than the number of *S. tritici* lesions at the Shellbrook summerfallow and barley stubble sites (Table 4.23). However, the number of lesions of the two pathogens was similar at the Shellbrook canola stubble site.

The Shellbrook wheat stubble site was not sampled using the combined plot method. Therefore, flag leaf data from the single plot sampling method was used. At GS 77, the number of *S. nodorum* and *S. tritici* lesions on the flag leaf was similar at the wheat stubble and barley stubble sites (Table 4.23). *S. nodorum* produced a greater number of lesions at the summerfallow site, while *S. tritici* produced the greatest number of lesions at the canola stubble site.

A dramatic increase in the number of *S. nodorum* lesions occurred between GS 63 and 83 on the flag leaves of all cultivars at the Weirdale wheat stubble site (Table 4.23). The number of *S. tritici* lesions increased only slightly during the same period.

The effect of rotation on lesion number at Shellbrook is evident in Figure 4.9. Leaf 2 was not sampled at the wheat stubble site. A rotation of two years out of wheat substantially reduced the number of *S. nodorum* lesions on leaf 2 compared to a rotation of one year out of wheat. Rotation did not affect the number of *S. tritici* lesions which were already very low in number. Barley between wheat

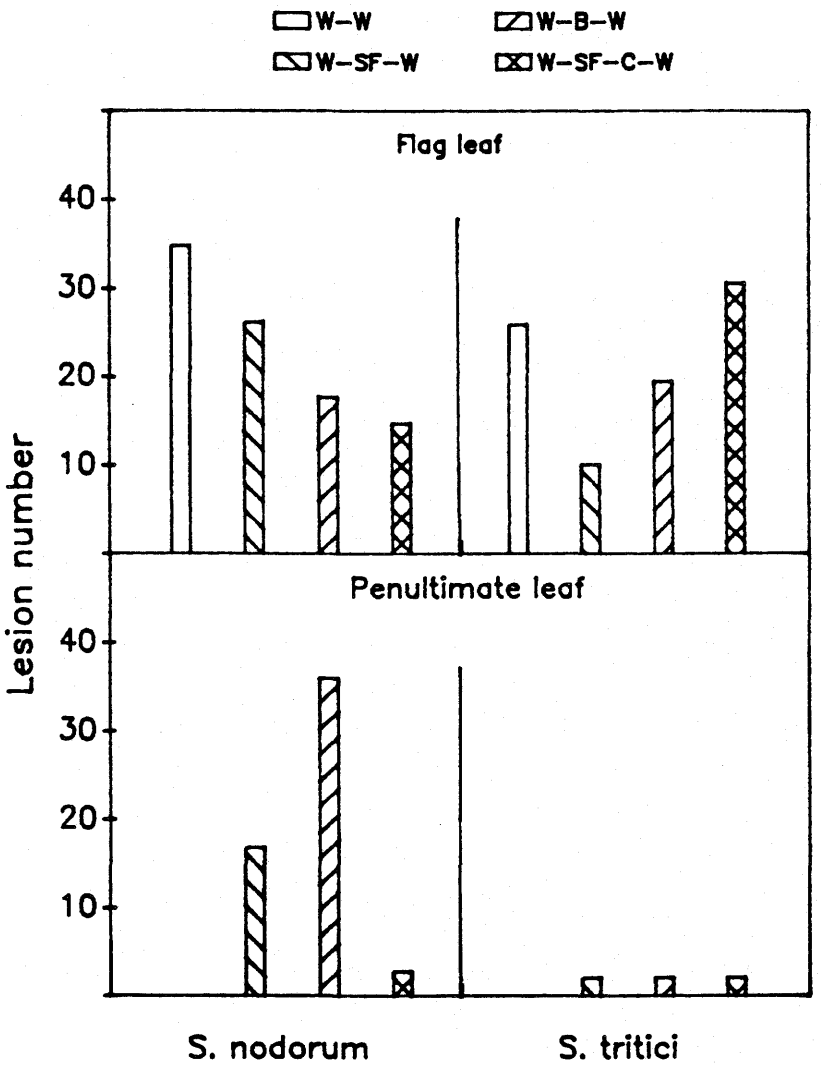

Figure 4.9 Mean number of lesions on the flag and penultimate leaves averaged for spring wheat cultivars grown in four crop rotations at Shellbrook in 1987. The penultimate leaf was not sampled at the W-W site. W=wheat, SF=summerfallow, B=barley and C=canola.

crops resulted in significantly fewer *S. nodorum* lesions on the flag leaf than continuous wheat, but the same result was not found when summerfallow occurred between wheat crops. The lowest number of *S. nodorum* lesions on the flag leaf occurred when cultivars were grown in a rotation of two years out of wheat.

Crop rotation did not have a consistent effect on lesions of *S. tritici*. A rotation of summerfallow between wheat crops resulted in fewer *S. tritici* lesions than a rotation of continuous wheat or barley between wheat crops (Table 4.23 and Figure 4.9). However, the number of *S. tritici* lesions on the flag leaf was similar for cultivars grown in a continuous wheat rotation and a rotation of two years out of wheat.

4.2.3 Effect of weather on the occurrence of *Septoria* pathogens

In 1987 and 1988, *S. nodorum* produced lesions with mature pycnidia on seedlings within three weeks of emergence, while *S. tritici* did not produce lesions bearing pycnidia until early to mid-July. It is not known if this is the typical pattern of development for the septoria diseases in the Parkland region of Saskatchewan since weather conditions in this area in 1987 and 1988 were far from average. However, by comparing the occurrence of the septoria pathogens to local weather conditions some relationships between disease and weather may be

determined.

S. tritici blotch was more prevalent at Shellbrook than at Paddockwood or Weirdale in 1987. Shellbrook had the lowest precipitation amounts and frequencies (Table 4.15), but the frequency of the duration of leaf wetness periods was similar at all three locations (Table 4.17). The monthly average high and low temperature was greater at Shellbrook in June and July (Table 4.16). This suggests that precipitation and the frequency of duration of leaf wetness periods did not limit the development of septoria tritici blotch at Paddockwood or Weirdale. However, the lower average monthly maximum and minimum temperatures may have constrained the increase in the prevalence of *S.tritici* at these two sites.

Severe septoria nodorum blotch epidemics occurred at all three locations in 1987. This suggested that temperature is not as critical for the development of this disease as it is for septoria tritici blotch.

In 1988, weather conditions were unfavorable for the development of disease. The level of septoria nodorum blotch was much lower than in 1987 and septoria tritici blotch was barely detectable. Precipitation amounts and frequencies in June of 1988 were well below normal (Table 4.15), while average monthly maximum and minimum temperatures were well above normal (Table 4.16). These conditions resulted in less frequent leaf wetness periods of

shorter duration. No leaf wetness periods greater than 15 h in duration occurred in June or July at Shellbrook and only one such period occurred at Weirdale (Table 4.17).

4.2.4 Discussion

Identifying and counting lesions permitted examination of the components of the septoria disease complex. It was possible to determine which species were involved and the extent of this involvement.

The relative occurrence of *Septoria nodorum* and *S. tritici* varied with location and year. In 1987, *S. nodorum* was the predominant pathogen at Paddockwood and Weirdale throughout the season. At Shellbrook, *S. nodorum* was predominant early in the season, but by GS 77 *S. nodorum* and *S. tritici* were equally prevalent at the wheat and barley stubble sites, while at the summerfallow site *S. nodorum* became prevalent and at the canola stubble site *S. tritici* became prevalent. In 1988, *S. nodorum* was the predominant pathogen at both locations. *S. tritici* did not appear until late in the season and produced few lesions.

These patterns of development are in contrast to those observed in the United Kingdom. Jenkins and Jones (1981) stated that septoria tritici blotch occurred at the earlier growth stages of wheat crops and caused less damage as the plants approached maturity. *S. nodorum* rarely produced symptoms on young plants, but caused severe infections on the flag leaf.

The limited development of *S. tritici* at Paddockwood and Weirdale, in 1987, may have resulted from the lower maximum and minimum temperatures that occurred at both sites. A decrease in temperature, especially minimum temperature, can extend the latent period of *S. tritici*, resulting in a slower infection cycle and ultimately slower disease progress. Holmes and Colhoun (1971) and Renfro and Young (1956) observed inhibition of *S. tritici* infection during two days with a minimum temperature of 7°C or less. In this study, more days with a minimum temperature of 7°C or less occurred at Paddockwood and Weirdale than at Shellbrook (Figure 4.7).

The occurrence of *S. nodorum* and *S. tritici* in 1988 was related to the duration of leaf wetness periods. *S. nodorum* apparently infected both cultivars during leaf wetness periods less than 15 h in duration and *S. tritici* did not. These results support the finding of Renfro and Young (1956) that *S. tritici* required leaf wetness periods longer than 15 h in duration for infection to occur. The results also agree with Shaner (1981) who observed that infection by *S. nodorum*, as determined by lesion counts, required shorter leaf wetness periods than infection by *S. tritici*.

Under high disease pressure, in 1987, the cultivars Kenyon, Pembina and Katepwa were equally susceptible to both *Septoria* pathogens. However, *S. tritici* was isolated only rarely from Oslo suggesting that Oslo possesses resistance

to this pathogen. This finding may account for the significant cultivar X rotation interaction found in the apparent infection rate data (Table 4.10). Lesion counts showed that *S. tritici* was the predominant pathogen at the canola stubble site. Therefore, the large decrease in the apparent infection rate of Oslo compared to the other cultivars may have resulted from this cultivar's resistance to *S. tritici*.

At Shellbrook, in 1987, a significantly greater number of *S. nodorum* lesions was found on Oslo than on the other cultivars. This may mean that Oslo is more susceptible to *S. nodorum* than these other cultivars. However, it may also reflect the lack of competition for available healthy tissue resulting from Oslo's apparent resistance to *S. tritici*.

The lower disease pressure experienced in 1988 caused a greater difference in the number of *S. nodorum* lesions on Katepwa and Oslo. Under this low disease pressure Oslo indeed appeared to be more resistant to *S. nodorum* than Katepwa. This low level of resistance was not apparent in 1987 when weather conditions were more favorable for disease development.

Crop rotation was more effective in reducing the number of lesions of *S. nodorum* than *S. tritici*. The longer the break between wheat crops, the greater the reduction in the number of *S. nodorum* lesions. A reduction in lesions caused by *S. tritici* occurred with a rotation of summerfallow

between wheat crops in 1987, but did not with a rotation of two years between wheat crops, even though summerfallow was practiced prior to the canola crop. The reason for this latter observation is not known, but volunteer wheat in the canola crop may have provided a source of *S. tritici* inoculum. Eyal (1981) reported a reduction in the incidence of septoria tritici blotch with crop rotations of 3 to 5 years out of wheat.

Lesion counts were not as effective in determining treatment differences as area under the disease progress curve or time to 50% disease severity. This was probably because disease severity is not only a function of lesion number but also of lesion size. In addition, coalescence of lesions on the more susceptible cultivars may result in an underestimation of the actual number of lesions.

4.3 Effect of the septoria complex on grain yield and kernel weight

4.3.1 1987 Trials

Significant differences in grain yield and kernel weight were found among cultivars at all Shellbrook sites in 1987, except the wheat stubble site (Table A.15).

At the Shellbrook wheat stubble site there were no significant differences between cultivars for yield, although Oslo had the highest yield. At the other Shellbrook sites, Oslo yielded significantly more than Kenyon, Pembina and Katepwa (Table 4.24).

Table 4.24 Grain yield and kernel weight of cultivars grown in different crop rotations at Shellbrook in 1987

Rotation	Cultivar				
	Kenyon	Pembina	Katepwa	Oslo	Mean
	Yield (kg/ha)				
W-W[1]	2142a[2]	2175a	2355a	2749a	2355B
W-SF-W	2356b	2584b	2606b	3305a	2713A
W-B-W	2640b	2766b	2733b	3081a	2805A
W-SF-C-W	2478b	2547b	2892b	3182a	2775A
Mean	2404c	2518bc	2647b	3079a	
	Kernel weight (mg)				
W-W	30.7a	31.8a	32.7a	31.8a	31.8C
W-SF-W	33.5c	35.7a	35.0ab	34.0bc	34.6B
W-B-W	32.5b	34.0a	33.2ab	32.0b	32.9C
W-SF-C-W	33.2b	34.0b	35.8a	36.8a	35.0A
Mean	32.5b	33.9a	34.2a	33.7a	

[1]W=wheat, SF=summerfallow, B=barley and C=canola.
[2]Means followed by the same lower case letter in a row or upper case letter in a column are not significantly different (P=0.05) according to Duncan's new multiple range test.

Kernel weight was similar for all cultivars at the Shellbrook wheat stubble site (Table 4.24). At the summerfallow site, the kernel weights of Kenyon and Oslo were lower than those of Pembina and Katepwa. Pembina and Katepwa had the highest kernel weights at the barley stubble site, but the kernel weight of Katepwa was not significantly different from that of Kenyon or Oslo. Kenyon and Pembina had the lowest kernel weights at the canola stubble site, while the kernel weights of Katepwa and Oslo were similar.

The effect of rotation on grain yield and kernel weight at Shellbrook was examined. Cultivar and crop rotation had significant effects on grain yield, but the cultivar X rotation interaction was not significant (Table 4.25).

Crop rotations excluding wheat for one year or more significantly increased grain yield. Yield at the wheat stubble site was approximately 15% lower than at the barley and canola stubble sites, and 13% lower than at the summerfallow site (Table 4.24).

Kernel weight also differed significantly among rotations and among cultivars at Shellbrook (Table 4.25). The cultivar X rotation interaction was also significant, indicating that the kernel weight of cultivars was not the same over sites.

The significant cultivar X rotation interaction resulted from the differential response of Pembina and Oslo at the summerfallow and canola stubble sites. The kernel

Table 4.25 Combined analysis of variance for grain yield and kernel weight of spring wheat cultivars grown in different crop rotations at Shellbrook in 1987

Source	df	MS	
		Yield	Kernel weight
Rotations(R)	3	1039094**	52.9**
Replicates/Rotations	20	130345	1.7
Cultivars(C)	3	2094437**	13.5**
C X R	9	81395	5.9**
Error	60	87996	1.4

** Significant at P = 0.01.

weight of Pembina was greater at the summerfallow site than at the canola stubble site (Table 4.24). Conversely, the kernel weight of Oslo was greater at the canola stubble site than at the summerfallow site.

The difference in disease severity at the Shellbrook sites was reflected in the kernel weights. The lowest kernel weight was obtained from cultivars grown in a continuous wheat rotation where disease was most severe (Table 4.24). A significant increase in the kernel weight of all cultivars occurred with a rotation of summerfallow between wheat crops and a rotation of two years out of wheat. Compared to the canola stubble site, kernel weight was reduced by 6% and 11% on barley and wheat stubble, respectively.

4.3.2 1988 Trials

Severe drought conditions developed at Shellbrook in 1988 and only light septoria epidemics occurred at this site. Heat and drought stress appeared to have a greater effect on plant health than disease.

Significant differences in grain yield were found among cultivars at the Shellbrook wheat stubble and summerfallow sites in 1988 (Table A.16), but not at the canola stubble site or among fungicide treatments at any of the sites. None of the cultivar X fungicide interactions were significant.

The yield of Katepwa was significantly higher than that of Oslo at the wheat stubble and summerfallow test sites (Table 4.26). The grain yield was similar for both cultivars

Table 4.26 Grain yield and kernel weight of propiconazole (Tilt) treated and untreated cultivars grown in different crop rotations at Shellbrook in 1988

Rotation	Treatment	Cultivar			
		Katepwa	Oslo	Roblin	Mean
		Yield (kg/ha)			
W-W[1]	NT[2]	2074A[3]	984A	--	1529A
	T	1823A	1129A	--	1476A
	Mean	1948a	1057b		
W-SF-W	NT	2723A	1832A	2085A	2213A
	T	2694A	2078A	2149A	2307A
	Mean	2709a	1955b	2117b	
W-SF-C-W	NT	3746A	3817A	--	3782A
	T	3659A	3320A	--	3489A
	Mean	3702a	3568a		
		Kernel weight (mg)			
W-W	NT	34.8A	39.9B	--	37.4B
	T	34.7A	42.8A	--	38.7A
	Mean	34.8b	41.4a		
W-SF-W	NT	33.8A	45.0A	36.6A	38.4A
	T	34.8A	45.6A	36.9A	39.1A
	Mean	34.3c	45.3a	36.7b	
W-SF-C-W	NT	34.7A	38.1B	--	36.4B
	T	35.9A	41.4A	--	38.6A
	Mean	35.3b	39.8a		

[1] W=Wheat, SF=summerfallow, C=canola.
[2] NT=no Tilt and T=Tilt.
[3] Means followed by the same lower case letter in a row or upper case letter in a column are not significantly different (P=0.05) according to analysis of variance or Duncan's new multiple range test.

at the canola stubble test site. Katepwa had a significantly higher yield than Roblin at the summerfallow site, but the yields of Oslo and Roblin were not significantly different.

In 1988, yield loss was measured for Oslo at the Shellbrook wheat stubble and summerfallow sites, while a yield gain occurred for Katepwa at the wheat stubble site (Table 4.27). However, the effect of fungicide on grain yield was not significant at any of the Shellbrook sites.

Analysis of the Shellbrook kernel weight data showed significant differences among cultivars and fungicide treatments at the wheat and canola stubble sites (Table A.16). Differences among cultivars were also significant at the summerfallow site, but differences among fungicide treatments were not. The cultivar X fungicide interaction was significant at the wheat stubble site only.

Oslo had the highest kernel weight at all Shellbrook sites (Table 4.26). At the summerfallow site Roblin had a greater kernel weight than Katepwa. Fungicide application did not have a significant effect on the kernel weights of Katepwa at any site or of Oslo and Roblin at the summerfallow site. Fungicide application did significantly increase the kernel weight of Oslo at the wheat and canola stubble sites (Table 4.26). However, the greatest loss in kernel weight occurred at the canola stubble site (Table 4.27); the site with the lowest level of disease. This suggests that the loss in kernel weight observed was not a

Table 4.27 Mean percentage loss or gain of yield and kernel weight of cultivars grown in different crop rotations at Shellbrook and Weirdale in 1988

Rotation	% Loss[1]					
	Katepwa		Oslo		Roblin	
	Yield	Kernel weight	Yield	Kernel weight	Yield	Kernel weight
Shellbrook						
W-W[2]	-13.8[3]	-0.6	12.8	6.8*	--	--
W-SF-W	-1.1	2.9	11.8	1.3	3.0	0.8
W-SF-C-W	-2.4	3.3	-15.0	8.0*	--	--
Weirdale						
W-W	19.0*	8.8	12.4*	5.5	--	--
W-P-W	17.6*	3.0	10.4*	4.6	16.7*	11.5*
W-C-P-W	-2.5	8.1*	7.9	5.6*	--	--

*Loss or gain is significant (P=0.05).
[1] %loss=[(fungicide check - untreated treatment)/ fungicide check].
[2] W=wheat, SF=summerfallow, C=canola and P=pea.
[3] A negative loss in yield or kernel weight represents a gain in yield or kernel weight.

result of disease.

The effect of cultivar on grain yield was significant at the pea1 stubble site, but not at the wheat or pea2 stubble sites at Weirdale (Table A.17). Fungicide application significantly increased grain yield at the wheat and pea1 stubble sites, but not at the pea2 stubble site.

The yield losses for Oslo were 12% at the wheat stubble site and 10% at the pea1 stubble site. The yield loss of Katepwa was 19% in a continuous wheat rotation and 18% in a rotation one year out of wheat. No significant yield loss was measured with a rotation two years out of wheat (Table 4.27).

Significant differences for kernel weight were found among cultivars and between fungicide treatments at all sites at Weirdale (Table A.17).

Oslo had a significantly higher kernel weight than Katepwa at each Weirdale site (Table 4.28). When averaged over cultivars, the fungicide-treated plots had a higher kernel weight than untreated plots at each site (Table 4.28). The greatest loss in the kernel weight of Katepwa occurred at the wheat stubble and pea2 stubble sites, whereas the the kernel weight loss of Oslo was approximately 5% at all sites (Table 4.27).

4.3.3 Discussion

In 1987, the yield of Oslo was more variable than the other cultivars. Little difference was detected among the

Table 4.28 Grain yield and kernel weight of propiconazole (Tilt) treated and untreated cultivars grown in different crop rotations at Weirdale in 1988

		Cultivar			
Rotation	Treatment	Katepwa	Oslo	Roblin	Mean
		Yield (kg/ha)			
W-W[1]	NT[2]	1994B[3]	2124B	--	2059B
	T	2461A	2427A	--	2444A
	Mean	2227a	2275a		
W-P-W	NT	2459B	2556B	2189B	2401B
	T	2983A	3061A	2629A	2891A
	Mean	2721a	2808a	2409b	
W-C-P-W	NT	1663A	1524A	--	1594A
	T	1621A	1655A	--	1638A
	Mean	1642a	1590a		
		Kernel weight (mg)			
W-W	NT	30.9A	36.4A	--	33.6B
	T	33.9A	38.5A	--	36.2A
	Mean	32.4b	37.5a		
W-P-W	NT	35.7A	41.8A	33.2B	36.9B
	T	36.8A	43.8A	37.5A	39.3A
	Mean	36.2b	42.8a	35.3b	
W-C-P-W	NT	35.3B	40.8B	--	38.1B
	T	38.4A	43.2A	--	40.8A
	Mean	36.8b	42.0a		

[1] W=Wheat, C=canola and P=pea.
[2] NT=No Tilt and T=Tilt.
[3] Means followed by the same lower case letter in a row or upper case letters in a column are not significantly different (P=0.05) according to analysis of variance or Duncan's new multiple range test.

yields of Kenyon, Pembina and Katepwa. In 1988, Katepwa yielded more than Oslo under drought conditions, but the yield of the two cultivars was similar when growing conditions were more favorable.

In 1987, Kenyon had the lowest kernel weight, while little difference was detected among the kernel weights of Pembina, Katepwa and Oslo. The large increase in the kernel weight of Oslo at the canola stubble site likely resulted from this cultivar's resistance to the predominant pathogen, *S. tritici*. In 1988, the kernel weight of Oslo was consistently greater than the kernel weight of Katepwa.

Crop rotation appeared to have a large effect on grain yield and kernel weight in 1987. Site effects other than that of crop rotation may have had an effect on both yield and kernel weight. However, the increase in the grain yield of cultivars grown in a rotation two years out of wheat, compared to a rotation of continuous wheat, reflected the decrease in disease severity. No equivalent relationship was found for the rotation of one year out of wheat. At the summerfallow site, disease was severe yet yields were high. This response was likely a result of increased soil moisture resulting from the summerfallow year. Disease was moderate at the barley stubble site, but yield was not significantly different from that at the summerfallow site. This may have been due partly to the slower apparent infection rate observed at this site (Table 4.11), the slower rate

significantly delayed the epidemic by 2.7 days.

In 1988, the disease pressure was low and loss in kernel weight was not disease related. Kernel weight loss was similar for cultivars grown in different rotations regardless of the level of disease. The lack of differences in kernel weight may have resulted from fungicide application. Propiconazole (Tilt) is classified as a triazole derivative. These chemicals may possess cytokinin-like antisenescent activity. King et al. (1983) reported that wheat sprayed with fungicides, including benomyl (another triazole derivative), decreased percent senescence compared to the unsprayed plants. Delaying senescence extends the grain-filling period and may result in increased kernel weight and/or grain yield.

Crop rotation had little effect on yield, in 1988, at Shellbrook. This was considered to be due to drought conditions and the low level of disease at that site. Growing conditions were better at Weirdale and rotation did affect yield. In a continuous wheat rotation at Weirdale, yield loss for Katepwa and Oslo was 19% and 12.4%, respectively. When these cultivars were grown in a rotation two years out of wheat, no significant yield loss was measured for either cultivar. Similar results were obtained in 1987. The greatest yields occurred at the site with a rotation of two years out of wheat.

A one year rotation between wheat crops at Weirdale was not as effective in reducing yield loss as a two year rotation. The area under the disease progress curve, and the time to 50% disease severity were similar for cultivars grown in both rotations and did not explain this difference in yield loss (Tables 4.7 and 4.14). The apparent infection rate for Oslo was also similar at both sites (Table 4.11). However, Katepwa had a greater apparent infection rate at the one year rotation site than the two year rotation site, resulting in higher disease severity during the latter half of the epidemic (Table 4.11 and Figure 4.6). The higher disease severity at the pea1 stubble site during grain-filling may have resulted in a lower yield.

5. SUMMARY OF RESULTS

Both the area under the disease progress curve and the curve-fitting methods were useful in describing disease progression. The effect of cultivar and crop rotation was larger on the ADPC and T_{50} values than AIR values. Thus, analysis of the ADPC and T_{50} data detected a greater number of significant differences between treatments than AIR.

Vertical progression of the septoria disease complex was similar for all cultivars in 1986, 1987 and 1988. Disease occurred on the lowest leaves first and then spread upward to each successively higher leaf layer.

Analysis of ADPC and T_{50} data indicated differences among cultivars in levels of resistance to the septoria disease complex. HY320 and Oslo were the most resistant of the cultivars tested. This higher level of resistance was expressed as a lower ADPC or a longer T_{50} value. Katepwa, Neepawa, Pembina, and Columbus gave intermediate ADPC and T_{50} values. Park and Kenyon had the least resistance of all the cultivars. Although some cultivars showed greater resistance to the septoria complex than others, none were truly resistant types.

Data on lesion number were less effective in identifying differences in resistance levels among cultivars than were ADPC and T_{50} values, but such data did allow examination of resistance to each pathogen separately. Few

S. tritici lesions occurred on the leaves of cultivar Oslo, indicating that Oslo possesses resistance to this pathogen. In 1987, significant variation in the number of *S. nodorum* lesions was found only on the flag leaf (at Shellbrook). The cultivar Oslo had a higher number of lesions than the other cultivars, but this likely resulted from the lack of competition between the two pathogens rather than differences in the level of resistance. In 1988, the disease pressure was low and *S. tritici* was almost absent. Katepwa had a significantly higher number of *S. nodorum* lesions than Oslo, indicating that Katepwa was less resistant to this pathogen than Oslo.

Resistance of HY320 to the septoria complex was identified by a significantly slower AIR in 1986. However, the relationship between AIR and cultivar resistance was not as clear in 1987 and 1988. In these two years, the more resistant Oslo had the fastest AIR at most of the test sites. The exception was at the Shellbrook canola stubble site in 1987, where Oslo's resistance to the predominant pathogen, *S. tritici*, markedly slowed the AIR. No significant difference in AIR was detected among Kenyon, Katepwa and Pembina at four of the six test sites in 1987. At the remaining two sites, Kenyon had the slowest AIR. In 1988, Roblin had the slowest AIR at Shellbrook but the fastest AIR at Weirdale.

Crop rotation was effective in controlling the septoria disease complex. In 1987, under high disease pressure, summerfallow between wheat crops had no effect on the ADPC or T_{50} compared to continuous wheat. Barley between wheat crops did result in a significant reduction in the ADPC and significantly increased the the T_{50} by an average of 2.7 days. However, the greatest reduction in disease or the longest delay in epidemic development (9.2 days) was achieved with a rotation of two years between wheat crops.

The number of septoria lesions observed on the flag leaf revealed that crop rotation was more effective in reducing septoria nodorum blotch than septoria tritici blotch in 1987. Summerfallow between wheat crops significantly reduced the number of *S. tritici* lesions compared to continuous wheat, but had little effect on the number of *S. nodorum* lesions. Barley between wheat crops had little effect on the number of *S. tritici* lesions, but significantly reduced those of *S. nodorum*. A rotation of two years out of wheat resulted in the greatest reduction in *S. nodorum* lesions, but a large number of *S. tritici* lesions were observed at this site. Volunteer wheat in the previous canola crop may have provided a source of *S. tritici* inoculum.

Under the low disease pressure of 1988, a rotation of one year out of wheat significantly reduced the ADPC and increased the T_{50} by 3.4 and 6.1 days at Shellbrook and

Weirdale, respectively, compared to continuous wheat. No further reduction in disease or delay in disease development occurred with a two year rotation out of wheat at Weirdale. At Shellbrook, a two year rotation did result in a further decrease in the ADPC and increased the T_{50} an additional 2.8 days for Oslo, but not for Katepwa. A rotation of two years between wheat crops caused a significantly lower number of *S. nodorum* lesions on the flag leaf of Oslo and Katepwa at both the Shellbrook and Weirdale locations.

Crop rotation did not have a consistent effect on the AIR. Compared to continuous wheat a rotation of summerfallow between wheat crops had no effect on the AIR of cultivars in 1987. Barley between wheat crops had little effect on the AIR of Kenyon, Pembina or Oslo, but significantly reduced the AIR on Katepwa. A rotation of two years between wheat crops affected only the AIR on Oslo, however, the reduction was likely a result of Oslo's resistance to *S. tritici* not crop rotation. In 1988, a two year rotation out of wheat had little effect on the AIR of cultivars. A one year rotation between wheat crops hastened the AIR of Katepwa and slowed that of Oslo.

The relative occurrence of the septoria pathogens was found to vary with location and year. In 1987, *S. nodorum* was the predominant pathogen throughout the growing season at Paddockwood and Weirdale. At Shellbrook, *S. nodorum* was predominant early in the season, but by GS 77 *S. nodorum*

and *S. tritici* had become equally prevalent at the wheat and barley stubble sites, *S. nodorum* was prevalent at the summerfallow site and *S. tritici* was the predominant pathogen at the canola stubble site. In 1988, *S. nodorum* was the predominant pathogen throughout the season at both locations.

Weather conditions in the 1987 and 1988 growing seasons were abnormal. In both years the month of June was unusually hot and dry. At all locations in 1987 and at Weirdale in 1988, July and August received above average precipitation. At Shellbrook in 1988, the hot and dry conditions persisted until August. Septoria epidemics at the wheat stubble sites were severe at all locations in 1987, moderate at Weirdale in 1988, and light at Shellbrook in 1988.

The following general relationships between weather and septoria disease development were observed. Initiation of disease and disease increase occurred 8 to 12 days following precipitation of at least 1 mm or after precipitation on two or three consecutive days. Canopy temperatures in excess of 35°C delayed epidemic development. Development of septoria nodorum blotch occurred under drier conditions than that of septoria tritici blotch. *S. tritici* was not detected during those months of the growing season when few or no leaf wetness periods of 15 h in duration occurred.

The major emphasis of this study was on the epidemiology of the septoria disease complex and experimental design was

not selected for determining relationships between disease development and yield or kernel weight loss. However, grain yield and kernel weight was determined in 1987 and 1988. Crop rotation appeared to have an effect on grain yield and kernel weight in 1987. Yield at the continuous wheat site was approximately 15% lower than at the stubble sites with at least a one year rotation out of wheat. Compared to the two year rotation site, kernel weight was reduced by 6 and 11% with a rotation of barley between wheat crops and continuous wheat, respectively. A rotation of summerfallow between wheat crops resulted in high yields and kernel weights, even though disease was severe. This was likely a result of the higher soil moisture associated with summerfallowing.

The low level of disease and the drought conditions at Shellbrook in 1988 resulted in little useful yield or kernel weight data. At Weirdale, yield loss ranged from 12 to 19% with a continuous wheat rotation or a rotation of one year out of wheat. Kernel weight loss was similar for cultivars grown in different crop rotations regardless of the level of disease and was, therefore, considered not to be a result of disease.

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APPENDIX A. TABLES

LIST OF APPENDIX TABLES

Table A.1 Equations to convert Horsfall-Barratt (HB) values to percent disease severity (DS) values

HB Range	Conversion equation
0 - 5	$\%DS = 75(0.5)^{[(5-HB)+1]}$
>5 - <6	$\%DS = 37.5+(HB-5)25$
6 - 11	$\%DS = 100-75(0.5)^{[(HB-6)+1]}$

Table A.2 Repeated measures analysis of variance for area under the septoria disease progress curve of each of the upper four leaves of different spring wheat cultivars grown on wheat stubble at Weirdale in 1986

Source	df	MS ($x10^{-2}$)
Replicates(R)	5	568
Cultivars(C)	5	27980**
C X R (Error A)	25	200
Leaf layers(L)	3	340488**
L X C	15	1523**
Error B	90	97

** Significant at P=0.01.

Table A.3 Repeated measures analyses of variance for area under the septoria disease progress curve of each of the upper four leaves of different spring wheat cultivars grown at Paddockwood, Weirdale and Shellbrook in 1987

		MS (x100)					
		Paddockwood	Weirdale	Shellbrook			
Source	df	W-W[1]	W-W	W-W	W-SF-W	W-B-W	W-SF-C-W
Replicates(R)	5	671	1721	2531	453	456	581
Cultivars(C)	3	13232**	24981**	9588**	37722**	24081**	34081**
C X R (Error A)	15	370	476	460	363	568	937
Leaf layers(L)	3	364100**	409972**	256402**	267954**	288735**	196065**
L X C	9	3207**	1519**	1136**	1945**	1957**	4585**
Error B	60	213	125	222	177	173	213

** Significant at P=0.01.
[1]W=wheat, SF=summerfallow, B=barley and C=canola.

Table A.4 Repeated measures analyses of variance for area under the septoria disease progress curve of each of the upper four leaves of different spring wheat cultivars grown at Shellbrook in 1988

		MS ($x10^{-2}$)			MS ($x10^{-2}$)
Source	df	W-W[1]	W-SF-C-W	df	W-SF-W
Replicates(R)	5	1267	1338	5	314
Cultivars(C)	1	247	7909*	2	13825**
C X R (Error A)	5	682	983	10	4754
Leaf layers(L)	3	69165**	55429**	3	106034**
L X C	3	3480**	6351**	6	4141**
Error B	30	334	257	45	270

*, ** Significant at P=0.05 and P=0.01, respectively.
[1] W=wheat, SF=summerfallow and C=canola.

Table A.5 Repeated measures analyses of variance for area under the septoria disease progress curve of each of the upper four upper leaves of different spring wheat cultivars grown at Weirdale in 1988

		MS ($x10^{-2}$)			MS ($x10^{-2}$)
Source	df	W-W[1]	W-C-P-W	df	W-P-W
Replicates(R)	5	203	78	5	69
Cultivars(C)	1	44989**	28979**	2	28054**
C X R (Error A)	5	143	308	10	49
Leaf layers(L)	3	97365**	68365**	3	87264**
L X C	3	4514**	6700**	6	4073**
Error B	30	69	92	45	62

** Significant at P=0.01.
[1] W=wheat, C=canola and P=pea.

Table A.6 Adjusted coefficient of determination (R^2), standard deviation about the regression line in percent (S%), autocorrelation parameter (P), and the Durbin-Watson statistic (D) for linear regression of Horsfall-Barratt values, logits and gompits plotted on time for each cultivar-rotation combination at test locations in 1986, 1987 and 1988.

		Horsfall-Barratt				Logistic				Gompertz				
Rotation	Cultivar	R^2	S%	P	D	R^2	S%	P	D	R^2	S%	P	D	df
						Weirdale 1986								
W-W[1]	Park	94.7	5.53	0.04	1.49	89.5	5.75	0.05	1.69	89.7	5.02	0.07	1.77	28
	Pembina	97.2	3.74	-0.11	2.16	92.4	4.67	-0.15	2.28	91.2	3.45	-0.06	2.12	28
	Katepwa	97.4	3.61	-0.09	2.15	96.6	5.16	-0.03	2.05	97.7	2.80	-0.04	2.07	28
	Neepawa	96.4	3.73	-0.29	2.55	96.2	5.12	-0.22	2.40	96.7	2.95	-0.30	2.59	28
	Columbus	94.4	6.46	-0.24	2.47	94.5	6.82	-0.18	2.34	94.3	4.09	-0.24	2.45	28
	HY320	77.9	19.37	-0.34	2.68*	76.2	18.67	-0.31	2.61	73.4	16.15	-0.45	2.89**	28
						Paddockwood 1987								
W-W	Kenyon	96.0	8.67	0.22	1.55	92.7	8.69	0.20	1.58	89.7	10.62	0.21	1.56	40
	Pembina	98.3	5.08	0.36	1.26*	96.2	5.49	0.12	1.73	92.4	8.79	0.22	1.53	40
	Katepwa	98.3	4.11	-0.07	2.13	96.4	4.65	-0.05	2.07	92.3	7.04	0.17	1.63	40
	Oslo	97.4	6.30	0.08	1.83	95.6	6.88	0.09	1.79	90.1	12.20	0.31	1.34*	40
						Weirdale 1987								
W-W	Kenyon	96.5	7.40	0.24	1.08**	95.3	7.35	0.25	1.03**	98.1	5.95	0.36	1.03**	34
	Pembina	97.0	5.87	0.02	1.82	96.7	5.85	0.09	1.68	97.8	5.09	-0.02	1.97	34
	Katepwa	97.8	4.51	-0.11	2.13	97.4	4.92	0.02	1.88	97.0	5.55	-0.14	2.24	34
	Oslo	94.3	10.54	0.40	1.14**	93.7	10.55	0.37	1.24*	88.9	13.83	0.38	1.17**	34

						Shellbrook 1987								
W-W	Kenyon	95.2	7.43	0.18	1.77	94.3	8.24	0.12	1.73	94.4	9.01	-0.16	2.31	34
	Pembina	94.4	7.62	0.03	1.88	93.6	8.62	-0.02	1.98	93.3	10.58	-0.14	2.22	34
	Katepwa	92.8	6.84	0	1.97	94.2	8.65	-0.06	2.10	92.5	11.08	-0.07	2.12	34
	Oslo	93.9	10.35	-0.26	2.49	92.7	10.60	-0.27	2.50	89.5	12.45	-0.08	2.12	34
W-SF-W	Kenyon	95.1	5.97	0.34	1.20**	94.4	6.34	0.39	1.11**	97.2	4.47	0.08	1.81	34
	Pembina	96.1	5.82	0.17	1.62	95.3	6.94	0.10	1.78	94.5	8.85	-0.09	2.15	34
	Katepwa	95.4	6.97	0.03	1.90	94.9	8.14	-0.02	2.00	93.4	10.34	-0.12	2.20	34
	Oslo	94.0	7.45	0.36	1.23*	92.7	8.21	0.37	1.22*	84.9	12.48	0.49	0.99**	34
W-B-W	Kenyon	95.6	6.20	-0.01	2.00	95.8	6.70	0.01	1.97	95.9	7.29	-0.23	2.42	34
	Pembina	94.4	9.22	0.04	1.90	94.1	9.88	-0.01	2.02	91.5	10.83	-0.07	2.13	34
	Katepwa	94.8	9.12	-0.16	2.31	94.3	9.72	-0.20	2.38	92.3	9.57	-0.30	2.58	34
	Oslo	94.5	6.78	0.12	1.56	94.2	7.07	0.11	1.62	87.4	9.50	0.23	1.30*	34
W-SF-C-W	Kenyon	91.5	9.43	0.13	2.09	92.7	9.22	-0.09	2.04	88.1	9.87	-0.05	1.97	28
	Pembina	92.4	12.85	0.03	1.78	92.3	11.86	0.05	1.79	85.1	11.59	0.13	1.67	28
	Katepwa	90.0	15.14	0.03	1.82	89.6	13.92	0.09	1.74	81.2	14.14	0.11	1.71	28
	Oslo	76.8	14.29	0.08	1.67	72.4	14.68	0.06	1.69	58.9	15.23	0.01	1.69	28
						Shellbrook 1988								
W-W	Katepwa	93.3	7.74	0.08	1.79	93.2	8.09	0.07	1.83	88.5	10.46	0.24	1.43	28
	Oslo	89.9	10.45	-0.30	2.59	89.7	9.99	0.32	2.63	88.3	13.78	0.47	2.94**	22
W-SF-W	Katepwa	98.9	3.11	-0.04	2.05	96.8	3.06	0.21	1.56	92.7	7.35	0.14	1.71	22
	Oslo	88.0	6.89	-0.01	1.97	86.5	8.40	-0.02	2.00	80.5	16.62	-0.05	2.06	22
	Roblin	86.1	12.80	0.19	1.56	85.2	11.70	0.19	1.56	77.1	19.10	0.13	1.67	22
W-SF-C-W	Katepwa	97.1	4.94	0.15	1.65	97.6	4.60	0.15	1.66	96.9	6.66	0.01	1.90	22
	Oslo	93.7	5.22	0.28	1.38	88.1	5.47	0.11	1.77	81.3	9.43	0.06	1.88	22

						Weirdale 1988								
W-W	Katepwa	98.4	4.71	0.07	1.83	97.8	5.54	-0.06	2.08	97.3	6.25	-0.15	2.26	28
	Oslo	96.0	9.79	-0.02	1.95	95.6	9.03	0.08	1.75	88.4	11.91	-0.02	1.92	22
W-P-W	Katepwa	97.4	5.69	-0.16	2.24	98.1	4.87	-0.02	1.96	95.6	6.24	-0.03	1.86	22
	Oslo	86.3	9.25	-0.04	2.01	83.0	11.17	-0.06	2.03	73.2	14.77	-0.10	2.11	22
	Roblin	95.9	4.24	-0.18	2.17	96.1	3.95	-0.08	1.98	88.7	9.47	0.16	1.53	28
W-C-P-W	Katepwa	97.3	8.45	-0.07	2.10	97.5	7.59	-0.02	2.02	95.6	8.98	0.15	1.61	28
	Oslo	94.5	6.45	0.26	1.45	93.3	7.78	0.22	1.52	85.1	13.48	0.27	1.40	28

*, ** indicates significant at P=0.05 and P=0.01, respectively.
[1]W=wheat, SF=summerfallow, B=barley, C=canola and P=pea.

Table A.7 Analyses of variance for apparent infection rate (AIR) and time[1] to 50% disease severity (T_{50}) for spring wheat cultivars grown in wheat stubble at Weirdale in 1986

		MS	
Source	df	AIR (x1000)	T_{50}
Replicates	5	0.21	1.6
Cultivars	5	1.35**	108.2**
Error	25	0.18	1.9

** Significant at P=0.01.
[1]Days after June 1.

Table A.8 Analyses of variance for apparent infection rate and time to 50% disease severity for spring wheat cultivars grown in different crop rotations at Paddockwood, Weirdale and Shellbrook in 1987

		MS					
		Paddockwood	Weirdale	Shellbrook			
Source	df	W-W[1]	W-W	W-W	W-SF-W	W-B-W	W-SF-C-W
		Apparent infection rate (x1000)					
Replicates	5	0.11	0.22	0.60	0.66	0.46	1.76
Cultivars	3	3.00**	0.87**	0.39	0.36*	0.83	12.37**
Error	15	0.14	0.10	0.17	0.07	0.35	0.76
		Time to 50% disease severity					
Replicates	5	1.9	4.1	4.6	1.7	2.7	14.4
Cultivars	3	39.6**	78.8**	24.5**	101.7**	76.9**	351.7**
Error	15	1.7	1.6	0.4	1.3	1.8	14.4

*, ** Significant at P=0.05 and P=0.01, respectively.
[1]W=wheat, SF=summerfallow, B=barley and C=canola.

Table A.9 Analyses of variance for apparent infection rate and time to 50% disease severity for spring wheat cultivars grown in different crop rotations at Shellbrook in 1988

Source	df	MS W-W[1]	MS W-SF-C-W	df	MS W-SF-W
Apparent infection rate (x1000)					
Replicates	5	1.22	0.36	5	0.51
Cultivars	1	61.65**	7.46**	2	9.84**
Error	5	1.78	0.47	10	0.34
Time[2] to 50% disease severity					
Replicates	5	2.0	3.6	5	0.5
Cultivars	1	1.9	9.9	2	59.7**
Error	5	3.0	1.9	10	1.3

** Significant at P=0.01.
[1]W=wheat, SF=summerfallow, C=canola.
[2]Days after June 1.

Table A.10 Analyses of variance for apparent infection rate and time to 50% disease severity for spring wheat cultivars grown in different crop rotations at Weirdale in 1988

		MS			MS
Source	df	W-W[1]	W-C-P-W	df	W-P-W
Apparent infection rate (x1000)					
Replicates	5	0.11	0.28	5	0.21
Cultivars	1	11.71**	1.57**	2	2.07**
Error	5	0.24	0.08	10	0.23
Time[2] to 50% disease severity					
Replicates	5	0.5	0.4	5	0.2
Cultivars	1	132.7**	94.1**	2	158.0**
Error	5	0.6	1.2	10	0.4

** Significant at P=0.01.
[1]W=wheat, C=canola and P=pea.
[2]Days after June 1.

Table A.11 Analyses of variance for the number of *Septoria nodorum* and *S. tritici* lesions on different leaf layers of spring wheat cultivars grown on wheat stubble at Paddockwood in 1987

		MS		
Source	df	Leaf 2	Leaf 2	Leaf 1
		S. nodorum		
Replicates	5	7.0	477.8	72.7
Cultivars	3	20.9	194.9	38.1
Error	15	15.3	67.7	42.6
		S. tritici [2] **(x100)**		
Replicates	5	--	5.5	0.8
Cultivars	3	--	63.3**	186.5**
Error	15	--	3.9	1.3

*, ** Significant at P=0.05 and P=0.01, respectively.
[1] 2=flag-1, 1=flag sampled at Zadoks GS 59 and 73, and 77, respectively.
[2] The data were transformed to X'= log(X+1) before analysis.

Table A.12 Analyses of variance for the number of *Septoria nodorum* and *S. tritici* lesions on different leaf layers of spring wheat cultivars grown on wheat stubble at Shellbrook in 1987

		MS		
Source	df	Leaf 3[1]	Leaf 2	Leaf 1
		S. nodorum		
Replicates	5	101.0	398.8	229.0
Cultivars	3	20.7	248.2	384.1**
Error	15	46.6	119.4	51.8
		***S. tritici*[2] (x100)**		
Replicates	5	--	3.1	2.1
Cultivars	3	--	260.8**	326.9**
Error	15	--	0.8	0.7

** Significant at P=0.01.
[1] 3=flag-2, 2=flag-1 and 1=flag, sampled at Zadoks GS 53, 65 and 77, respectively.
[2] The data were transformed to X' = log(X+1) before analysis.

Table A.13 Analyses of variance for the number of *Septoria nodorum* and *S. tritici* lesions on the flag-1 and flag-2 leaves of spring wheat cultivars grown in different crop rotations at Shellbrook in 1988

			MS		MS	
Rotation	Source	df	Leaf 3[1]	df	Leaf 2	Leaf 1
			S. nodorum			
W-W[2]	Replicates	4	0.30	5	198.3	63.4
	Cultivars	1	0.03	1	149.0	59.9
	Error	4	0.05	5	153.8	24.1
W-SF-C-W	Replicates	-	--	4	--	6.3
	Cultivars	-	--	1	--	57.1
	Error	-	--	4	--	8.2
			***S. tritici* (x100)**			
W-W	Replicates	-	--	5	--	2.2
	Cultivars	-	--	1	--	0.1
	Error	-	--	5	--	4.8

[1] 3=flag-2, 2=flag-1 and 1=flag, sampled at Zadoks GS 59, 73 and 77, respectively.
[2] W=wheat, SF=summerfallow and C=canola.

Table A.14 Analyses of variance for the number of *Septoria nodorum* lesions on different leaf layers of spring wheat cultivars grown in two crop rotations at Weirdale in 1988

		MS[1]		MS		MS
Source	df	Leaf 3[2]	df	Leaf 2	df	Leaf 1
			W-W[3]			
Replicates	5	0.038	4	437.0	5	7.4
Cultivars	1	0.273*	1	2255.3*	1	281.8*
Error	5	0.024	4	292.0	5	24.3
			W-C-P-W			
Replicates	-	--	-	--	4	13.7
Cultivars	-	--	-	--	1	95.6**
Error	-	--	-	--	4	4.6

*, ** Significant at P=0.05 and P=0.01, respectively.
[1]The data were transformed to X'= log(X+1) before analysis.
[2]3=flag-2, 2=flag-1 and 1=flag.
[3]W=wheat, P=pea and C=canola.

Table A.15 Analyses of variance for yield and kernel weight of spring wheat cultivars grown in several crop rotations at Shellbrook in 1987

Source	df	MS			
		W-W	W-SF-W	W-B-W	W-SF-C-W
		Yield			
Replicates	5	98475	17836	390774	14296
Cultivars	3	466346	1012531**	220663*	639081**
Error	15	150096	66259	60040	75588
		Kernel weight			
Replicates	5	1.60	2.24	1.47	1.44
Cultivars	3	4.06	5.71*	4.50*	16.82**
Error	15	1.89	1.31	0.87	1.42

*, ** Significant at P=0.05 and P=0.01, respectively.
W=wheat, SF=summerfallow, B=barley and C=canola.

Table A.16 Split-plot analyses of variance for yield and kernel weight of spring wheat cultivars grown in different crop rotations at Shellbrook in 1988

		MS			MS
Source	df	W-W[1]	W-SF-C-W	df	W-SF-W
		Yield			
Replicates(R)	5	324476	1229815	5	473659
Cultivars(C)	1	4770645*	107749	2	1887636**
Error A	5	424509	3604908	10	72898
Fungicide(F)	1	16946	514475	1	79407
C X F	1	236461	250539	2	58542
Error B	10	59984	596416	15	52191
		Kernel weight			
Replicates(R)	5	5.5	6.2	5	1.6
Cultivars(C)	1	262.7**	121.5**	2	399.1**
Error A	5	10.2	2.5	10	1.0
Fungicide(F)	1	10.9*	30.8**	1	3.7
C X F	1	13.5*	6.8	2	0.5
Error B	10	2.2	1.7	15	1.8

*, ** Significant at P=0.05 and P=0.01, respectively.
[1]W=wheat, SF=summerfallow and C=canola.

Table A.17 Split-plot analyses of variance for yield and kernel weight of spring wheat cultivars grown in different crop rotations at Weirdale in 1988

		MS			MS
Source	df	W-W[1]	W-C-P-W	df	W-P-W
		Yield			
Replicates(R)	5	54798	46800	5	235921
Cultivars(C)	1	13755	16449	2	529187*
Error A	5	93803	89030	10	75486
Fungicide(F)	1	890903**	11952	1	2156950**
C X F	1	40937	44682	2	5695
Error B	10	34903	32499	15	35910
		Kernel weight			
Replicates(R)	5	9.0	0.2	5	4.6
Cultivars(C)	1	154.5**	159.1**	2	199.2**
Error A	5	0.8	1.1	10	3.1
Fungicide(F)	1	39.3*	43.2**	1	53.5**
C X F	1	0.9	0.7	2	7.7
Error B	10	8.1	0.6	15	2.7

*, ** Significant at P=0.05 and P=0.01, respectively.
[1]W=wheat, P=pea and C=canola.