STUDIES ON BLACKLEG DISEASE OF OILSEED RAPES: GERMPLASM EVALUATION, VARIATION FOR VIRULENCE AND YIELD LOSS/DISEASE RELATIONSHIPS

BY

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ABSTRACT

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Resistance of oilseed rape and oilseed turnip rape (Brassica napus L. and B. rapa L. respectively) to infection by Leptosphaeria maculans (Desm.) Ces. et de Not., has become an important consideration in breeding programs due to the rapid spread of the pathogen throughout western Canada. Cultivars of B. rapa currently registered do not contain acceptable levels of resistance to the pathogen. Evaluation for blackleg resistance of 232 accessions of B. rapa from the University of Manitoba germplasm collection was performed in the greenhouse at cotyledon and adult stages with one isolate of L. maculans. Accessions were also evaluated in a field disease nursery. No accessions were highly resistant at either stage. Nine accessions in the cotyledon stage and 7 accessions in the adult stage were moderately resistant. In field tests, variation for resistance was found among accessions. There was a weak correlation between results of the cotyledon and adult test. However no significant correlations were found between other tests, although there were individual accessions which had a low disease severity rating in more than one test.

Variation for virulence in isolates of L. maculans from two locations in western Canada and from Western Australia over a range of resistant to susceptible brassica lines was investigated. Virulence tests were conducted at the cotyledon and adult stages under controlled conditions, using isolates derived from single ascospores. Double haploid B.

napus lines, inbred B. juncea lines and lines of the cross-pollinated species B. rapa, were used as hosts. There were highly significant differences between isolates within sites as well as among sites. There were highly significant line x isolate interactions, an indication of differential interactions. The lines R8314.47 and UM3132 were found to differentiate isolates at the cotyledon stage while R8314.47, UM3132 and Global differentiated isolates at the adult stage. The line R8314.47 was generally susceptible to Western Australian isolates but resistant to isolates from the 2 Canadian locations.

Yield losses due to L. maculans infestation have been reported to be devastating to oilseed rape in localized outbreaks in western Canada. A study was performed to quantify yield losses of 6 cultivars of oilseed rape when seeded into L. maculans infested fields. The Australian cultivar BLC198 appeared to be more tolerant of L. maculans than was Tobin. Under moderate disease intensity at one location BLC198 outyielded Tobin by 25%. The B. napus cultivar Profit had a greater level of resistance to blackleg disease than Westar. Profit outyielded Westar by 59% under moderate disease intensity. Late maturing B. napus cultivars Global and Maluka did not appear to be affected by the disease levels encountered in this study, although disease severity ratings were significantly higher for Global than for Maluka.

FOREWORD

The format of this thesis follows the manuscript style which has been outlined by the Department of Plant Science of the University of Manitoba. Three manuscripts are presented which follow the style of the Canadian Journal of Plant Pathology. Within each manuscript is an abstract, introduction, materials and methods, results and discussion. A general abstract, a general introduction and a review of the literature precede the manuscripts. A general discussion, literature cited and appendices follow the manuscripts.

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GENERAL INTRODUCTION

Oilseed rape production is of major importance to the economy of western Canada. Only wheat and barley exceed the crop in amount of hectares seeded. Oilseed rape supplies over 13% of the world's edible oil; only soybean and palm oil are more important (Downey and Röbbelen 1989). In developed countries the superior nutritional aspects of edible oil derived from oilseed rape are generating increased interest in the crop. The high level of monounsaturated fatty acids in the oil (67% of total) have been suggested to decrease risk of heart disease (Vles and Gottenbos 1989).

Leptosphaeria maculans the causal organism of blackleg disease of oilseed rape, exists in two forms - avirulent which causes only superficial disease symptoms, and virulent which causes severe crown cankers and economic yield loss. The virulent form of L. maculans was first detected at one location in Saskatchewan. It has spread rapidly and has now been found in all oilseed rape producing areas in Saskatchewan (Jesperson 1990), Manitoba (van den Berg and Platford 1990) and in east central Alberta (Evans et al. 1990). Concern in Canada has prompted the development of blackleg resistant oilseed rape cultivars.

In Europe and Australia resistant cultivars of oilseed rape have been developed as the most effective control of the disease. In Canada there are some Brassica napus cultivars recognized to be 'fair' in resistance to L. maculans (Anon 1989). However no B. rapa cultivars presently registered are resistant. Determining sources of resistance within B. rapa is the first step in a breeding program concerned with developing resistant cultivars.

An effective breeding program benefits from information about the nature of host-pathogen interactions. Genetic variation in response to L. maculans within species of oilseed rape, and variation for virulence in the pathogen must be determined. This

information will assist in selection of resistant germplasm, as well as leading to more efficient and effective exploitation of resistance genes.

Yield losses in oilseed rape due to L. maculans have been reported to be severe in many cases. However few studies have quantified yield losses; those which have used field survey methods. More precise measurement of yield losses would be useful in calculating the economic impact of the disease in specific locations and to total production of oilseed rape. Knowledge of yield losses may be used to plan strategy in resource allocation and cost effective measures of control of blackleg disease.

To address these problems the following studies were undertaken: a) to evaluate B. rapa accessions from the University of Manitoba Brassica rapa germplasm collection for resistance to L. maculans, b) to study variation for virulence within isolates of L. maculans over a range of resistant to susceptible brassica lines, and c) to quantify the yield loss that may be expected in oilseed rape in a field situation.

LITERATURE REVIEW

2.1 The Host

Oilseed rape was first produced in 1943 in Canada to supply lubricants for engines of naval and merchant ships. This was necessary during World War II because of the restricted availability of these lubricants from Europe and Asia. In the mid 1950's demand for oilseed rape increased in the food industry for processing into margarine, shortening and salad oil (Youngs 1974). This required the development of low erucic acid level cultivars to satisfy nutritional concerns. Levels of erucic acid were reduced from 44.2% of the fatty acid content to trace levels. Similarly glucosinolate levels in the meal were reduced to low levels to satify the requirements of the feed industry (Stefansson 1983). Specification of acceptable levels of erucic acid content of oil and glucosinolate content of seed and meal were adopted by the crushing industry. These specifications had to be met in order for cultivars of oilseed rape to qualify for use of the name canola. Development of canola quality oilseed rape has lead to its important position in the economy of western Canada.

Two species of oilseed rape, more commonly known as rapeseed or canola, are grown in Canada in approximately equal amounts. Brassica napus L., commonly referred to as argentine rape, and B. rapa L. (syn. B. campestris L.) is commonly known as polish rape. Brassica napus is 20% higher yielding but requires 95 days to mature which is 10 days later than B. rapa (Anon 1989). Brassica rapa is popular when seeding is delayed or in areas where the risk of frost is too high for cultivation of B. napus.

Statistics for the 1987 crop show that 2.67 M hectares were seeded in Canada. The average yield was 1440 kg ha^{-1}, and total production was 3.85 M tonnes (Anon 1988a).

2.2 The Pathogen

2.2.1 Description

Leptosphaeria maculans is found world-wide wherever brassica species are grown (Commonwealth Mycological Institute 1978). It has caused extensive damage to crucifers such as fodder brassicas, turnips (B. rapa) and swedes also known as rutabagas (B. napus) in New Zealand. It has caused serious losses in B. oleracea in the United States, and has threatened the cabbage seed industry in that country (Gabrielson 1983).

Leptosphaeria maculans (Desm.) Ces. et de Not., anamorph Phoma lingam (Tode ex Fr.) Desm., is the causal agent of blackleg disease of oilseed rape. It is an heterothallic, loculoascomycete and a member of the order Pleosporales.

As a parasite, the pathogen produces pycnidia with a pseudoparenchymatous wall structure, while as a saprophyte the pycnidia have a pseudosclerenchymatous wall structure (Boerema 1976). The two types of pycnidia have been designated group I and group II respectively (Cunningham 1927).

Group I pycnidia are initially closed, then develop a papillate opening, sometimes with a neck. Group II pycnidia have dark walls, are often irregular in shape and may or may not have a papilla. Hyaline, oval, single-celled pycnidiospores measuring 1-2 x 2.5-5 μm are exuded from the pycnidia.

The sexual state is usually found only on woody plant tissue. Pseudothecia are composed of pseudosclerenchymatous tissue, round, papillate and 360-500 μm in diameter. Within the pseudothecia are bitunicate asci measuring 10-17 x 70-120 μm. Ascospores within the asci are hyaline when young and tan-yellow when mature, five septate and constricted at the septa. They measure 4-9 x 30-70 μm (Smith and Sutton 1964, Boerema 1976).

2.2.2 Symptoms

Infection of canola plants by the blackleg pathogen may cause damping off of seedlings, leaf spots and stem and crown cankers. All parts of the plant including the siliques and seed may become infected (Gabrielson 1983).

Indefinite water-soaked lesions form on cotyledons, hypocotyls, and leaves. The lesions turn a white to grey color and dark scattered pycnidia usually appear. Pycnidiospores are exuded in a pink ooze from the pycnidia under appropriate conditions of temperature and moisture.

Invasion of the host follows a systemic pathway of infection (Hammond et al. 1985). After infection of the leaves or cotyledons where the fungus colonizes the intercellular spaces of the lamina, it follows a vascular strand into the petiole. The pathogen spreads down the petiole and may pass into the stem. In the stem the fungus kills the cells of the cortex causing cankers on upper portions of the stem and more commonly at the crown. Hammond and Lewis (1987a) and Mithen et al. (1987) found resistance to the pathogen was expressed in the leaves of Brassica species and therefore prevented systemic colonization. Hammond and Lewis (1987b) found resistance expressed in the stem of oilseed rape plants after the leaves and petiole had been colonized.

Stem cankers are black to whitish-grey in color and are of two types; at temperatures of 24/15^{o}C cankers develop rapidly, and are dry with abundant pycnidia, while at lower temperatures (12/7^{o}C) they develop slowly, and are soft and slimy with no pycnidia (Barbetti 1975). These cankers may encircle the entire stem seriously impairing pod filling, or causing lodging and death of the plant.

2.2.3 Disease Cycle and Epidemiology

The disease cycle of Leptosphaeria maculans is illustrated in Figure 2.1. The organism survives from season to season in infected seed or in infested residue of

previous crops. Pseudothecia are produced on infested residues as a result of conjugation of mycelium between mating types. Within the pseudothecia ascospores are produced which serve as the wind-borne primary inoculum. Ascospores are released under moist conditions and moderate temperatures. McGee (1977) found that at least one millimeter of precipitation was necessary to release large quantities of ascospores. Ascospores have been found to travel 5-8 km in Australia (Bokor et al. 1975) and 1.5 km in Britain (Gladders and Musa 1980). In Canada, Petrie (1978) found severe infections of oilseed rape crops immediately adjacent to fields containing infested residue but only traces of infection in a crop 1 kilometer from the nearest inoculum source.

Discharge of ascospores follows a seasonal pattern in France, Australia and Canada (Alabouvette and Brunin 1970, McGee 1977, McGee and Petrie 1979). In Canada the discharge begins in July of the year after establishment of the disease and continues throughout the summer and autumn. In second and subsequent years it begins in early spring and continues until late autumn. The quantity of inoculum has been found to decrease markedly by the third year (McGee 1977).

Oilseed rape plants may become infected at any stage of development. However the severe stem canker phase generally develops only when plants are infected between emergence and the six leaf stage of growth (McGee and Petrie 1979). Ascospores land on the leaves and germinate to produce a lesion. Pycnidia produced on these lesions exude pycnidiospores which serve as secondary inoculum. Pycnidiospores are dispersed by water-splash which limits the distance travelled to a maximum of about 1.5 meters. Barbetti (1976) found that under suitable climatic conditions pycnidiospores could produce a severe disease epidemic.

Pycnidiospores or mycelium in infected seed may serve to infect the developing seedlings directly. Seedlings may be killed during or shortly after emergence resembling

damping off. Seed-borne inoculum is not considered to lead to epidemics, but is responsible for spread of the disease to previously uninfested areas.

2.3 Variability of the Pathogen

2.3.1 Differential Interactions

The phenomenon of differential interactions is observed when a series of host lines are inoculated with a series of pathogen individuals (usually single spore cultures) which differ in virulence. The occurrence of differential interactions suggests that a gene-for-gene relationship is present in the host-pathogen relationship. The gene-for-gene concept was proposed by Flor (1942) who studied the inheritance of resistance in the host and the inheritance of virulence in the pathogen in flax (Linum usitatissimum L.) and the rust which attacks it (Melampsora lini Desm.). He found the inheritance of each was controlled by matching pairs of genes, and demonstrated simple Mendelian ratios in the inheritance of resistance and virulence. In the flax-flax rust system, host resistance was dominant to susceptibility, and at the complementary locus in the pathogen, which is diploid, avirulence was dominant to virulence. The only combination of alleles which resulted in a resistant response was that of the dominant resistance allele and the dominant avirulence allele. This dominance relationship has generally been found to be true in most cases where inheritance has been studied, although exceptions do exist (Crute 1985).

In a differential interaction there is a variable ranking of lines of different resistance when inoculated with pathogen individuals of varying virulence. The ranking is likely to change with each pathogen individual. If no differential interaction occurs the ranking of lines is in the order of their resistance regardless of which pathogen individual they are tested against. All pathogen individuals can be ranked according to their virulence,

regardless of which host differential they are tested against when differential interactions do not occur.

Person (1959) showed that a gene-for-gene relationship could be demonstrated by host-pathogen interaction studies only, without the accompanying genetic studies of the host and the pathogen as Flor (1942) had done. Person studied only the ratios of resistant and susceptible lines when tested with various pathogen individuals. From this he constructed what is known as the Person differential interaction model of host-pathogen interactions. All possible combinations of resistant and susceptible genes were arranged in a mathematical sequence of the binomial distribution, in both host differentials and pathogen differentials. The importance of the model is that it reveals the nature of the gene-for-gene relationship and demonstrates the gene-for-gene relationship without any genetic studies in either the host or the pathogen.

There is controversy among plant pathologists about the existence of non-differential interactions. Ellingboe (1981) believes that all host-pathogen interactions follow the gene-for-gene theory and that any apparent non-differential interaction is due to environment, poor methods of measurement or experimental error. Parlevliet and Zadocks (1977) suggest interactions which appear to be non-differential are due to additive effects of minor genes. On the other hand Vanderplank (1968) and Robinson (1987) claim non-differential interactions do exist. They suggest resistance and virulence are under the control of one, a few or many genes; however there is no matching of host and parasite genes.

2.3.2 Morphology

Cunningham (1927) detected variability for virulence in Leptosphaeria maculans. Two strains were described based on virulence to crucifers: avirulent and virulent. The two strains differ in appearance on culture media. In culture the avirulent strain produces

a distinct brownish-yellow pigment and grows faster than the virulent strain (McGee and Petrie 1978). The avirulent strain produces few or no pycnidia in culture as opposed to the virulent strain which produces an abundance of pycnidia. Petrie (1988) has developed an in vitro test to distinguish between these two strains based on length of the germ tube produced by conidia in a specified time period.

The relationship between avirulent and virulent strains is unknown. Cultural mutants have been found in the avirulent strain but none of the mutants were typical of virulent L. maculans (Calvert et al. 1949). Attempts to mate avirulent by virulent strains in culture have not been successful (Delwiche 1980, Bonman et al. 1981) although in the field both strains produce pseudothecia.

2.3.3 Virulence

Vanterpool (1961) described the avirulent strain of Leptosphaeria maculans for the first time in Canada in 1961. McGee and Petrie (1978) isolated the virulent strain for the first time in Canada in 1975 from the Melfort-Star City area of Saskatchewan.

L. maculans has been shown to be host-strain specific. Vanterpool (1963) and Petrie and Vanderpool (1968) demonstrated host specificity. L. maculans was found on Thlaspi, Brassica and Sisymbrium species. McGee and Petrie (1978) found that the Thlaspi strain of the fungus which was virulent on stinkweed (Thlaspi arvense) and avirulent on oilseed rape. Isolates that were virulent on rapeseed were avirulent on stinkweed.

Another level of variability is the ability of L. maculans to infect differentially a set of Brassica napus or B. rapa lines. A number of researchers have used various oilseed rape lines as a differential series to test isolates from several locations (Thurling and Venn 1977, Delwiche 1980, Cargeeg and Thurling 1980a, Newman 1984, Hammond and

Lewis 1987a, 1987b). Virulence was determined by the amount of damage the isolates caused to the oilseed rape lines over time.

Thurling and Venn (1977) looked at the responses of brassica lines to isolates from three sites in Australia. They found significant interactions between cultivars and fungal isolates. There was evidence of genetic variation in resistance to blackleg among cultivars. They also suggested that there was variation for virulence among different populations of pathogens from different sites.

Variation in response has been found to be continuous by Cargeeg and Thurling (1980a), suggesting that the system is polygenic in both host and pathogen. However they thought that cases of clear specific interaction may have been due to the presence of one or more resistance or virulence genes with relatively large effect.

Newman (1984) found isolates could be divided into groups; those avirulent on all cultivars and those showing differential interactions between different host lines. This suggests that the fungus can respond to changes in host resistance.

2.4 Disease Control

2.4.1 Management Practices

Leptosphaeria maculans persists in the soil on infested host residue for as long as it takes for the residue to breakdown. Infested residues are the source of primary inoculum and depending on environmental conditions may take 5 years or longer to break down (Alabouvette and Brunin 1970). Crop rotations of 3-5 years may not be effective or economic in many areas. Also since the primary inoculum is airborne, control in adjacent fields is required (McGee and Emmett 1977).

Control of susceptible weed species and volunteer oilseed rape must also be maintained. Wild mustard (B. kaber) is susceptible to the virulent oilseed rape form of L. maculans (Petrie 1979).

Seeding of oilseed rape crops has been delayed in Australia as a means of avoiding periods of heavy ascospore release (McGee 1977). This is not a viable alternative in most areas due to other environmental constraints, and the fact that ascospores are released throughout the growing season.

2.4.2 Chemical Control

Seed-borne inoculum is not believed to play an important role in the initiation of primary infection (McGee 1977). However it is important in transmission of the disease to previously uninfested areas. For this reason preventative measures to control seed infection have been sought. Seed testing methods which detect Leptosphaeria maculans have been developed (International Seed Testing Association 1965).

In the past hot water treatments have been used to eradicate the pathogen from crucifer seed. Walker (1923) found this treatment to be effective, however others reported that it failed to eradicate the pathogen (Cunningham 1927, Gabrielson 1983). The treatment is impractical for large seed lots.

A number of chemical seed treatments have been found to be effective in eradicating the pathogen. A thiram soak developed by Maude et al. (1969) was useful in controlling seed coat infection. Maude et al. (1984) found fenpropimorph to be an effective control of L. maculans. Gabrielson (1983) cites Maude et al. (1981) who reported benomyl, thiabendazole, and iprodione to be reliable seed treatments. Kharbanda (1989) found iprodione to be an effective seed treatment in growth chamber tests.

Various methods of chemical control have been tested to combat the disease in infested fields. Humpherson-Jones and Burchill (1982) were able to restrict pseudothecia

formation and ascospore release from infected stubble in vitro by chemical means. However in commercial situations this proved uneconomical.

Attempts have been made at disease control by foliar application of benomyl at various stages of crop development. However Brown et al. (1976) found poor control or no control of crown canker when foliar applications of benomyl were made to oilseed rape crops. Xi and Morrall (1988) tested foliar applications of triadimefon, flutriafol, iprodione and diniconazole on B. napus cv. Westar and B. rapa cv. Tobin. They found none of the fungicides were effective in controlling blackleg disease under the conditions encountered. Morrall and Xi (1989) obtained only limited control of blackleg disease in the field on B. napus cvs. Westar and Profit using multiple applications of prochloraz. They concluded that at present there is limited potential to control blackleg disease in western Canada through use of foliar fungicide application.

In Australia the systemic chemical flutriafol has been shown to be an effective control of blackleg in oilseed rape. Flutriafol is coated on superphosphate fertilizer granules and applied in furrow with the seed. Ballinger et al. (1988) recommend rates of 75-100 g ai ha^{-1} flutriafol. In Canada, Xi et al. (1989) have evaluated flutriafol by a similar method but found only limited effectiveness.

2.4.3 Resistance and Tolerance

Resistance has been defined as a characteristic of the host enabling it to resist growth and colonization of a pathogenic organism. The ability of one host cultivar to support invasion and colonization by the pathogen, similar to a second host cultivar, and yet to suffer less damage to its growth, development and reproduction was defined as tolerance (Crute 1985). Both resistance and tolerance among oilseed rapes has been found.

Resistance to blackleg disease in oilseed rape has been a major breeding objective in Europe, Australia and more recently Canada. Cultivars have been developed in France

and Australia that possess a degree of blackleg resistance or tolerance (Brunin 1970, Roy and Reeves 1975, Wratten 1977). It is thought that the resistance in many of the existing cultivars of B. napus may have been derived from the same source (Brunin, 1970). Other sources of resistance include B. juncea (Roy 1978, 1984), B. insularis (Mithen and Lewis 1988) and B. carinata (Sacristan and Gerdemann 1986). Various weedy cruciferous species may also serve as resistance sources (Salisbury 1987).

2.5 Yield Loss Assessment

In order to develop economic control measures to restrict disease it is necessary to quantify the magnitude of disease loss. Disease-loss appraisal involves two stages, the first of which is to characterize the relationship between disease and yield loss through field experimentation. The second stage is to assess disease loss through surveys in particular fields or areas using the assessment method developed in stage one.

In the experimental phase the objective is to measure disease and develop a reliable method for translating this into yield loss. It involves studying the disease throughout the season and monitoring growth of healthy and diseased plots. Models derived from field experiments are commonly classified as one of three types (James 1974). The critical point model estimates yield loss from a measured level of disease at a given growth stage; or at a certain growth stage or time when a given level of disease is attained. The critical point model assumes there is one point in a crop's development when it is particularly sensitive to the effects of the disease. A multiple point model estimates yield loss by determination of disease level at more than one point during growth of the crop. The multiple point model improves precision over the critical point model but requires increased labor and cost for data collection. A third model is the Area Under the Disease

Progress Curve. The area under the curve, measured in arbitrary units, is used to estimate yield loss.

The majority of disease-loss models are regression equations with percent yield loss as the dependent variable and one or more values of disease intensity as the independent variable.

$$\% \text{ yield loss} = b_0 + b_1X + U$$

where: b_0, b_1 - regression parameters and,

X - disease intensity and,

U - error term, the random dispersal of observations of yield loss on either side of the true regression line relating percentage yield loss to disease intensity.

Normality, homoscedasticity and the absence of autocorrelation are important assumptions of linear regression. If these assumptions are not valid reliability of the model as evaluated by the analysis of variance becomes suspect (James and Teng 1979).

The experimental phase of disease-loss studies should be conducted in all of the geographical areas where the crop is important and should be continued for at least three years. The study should include the major cultivars grown commercially and be carried out under conditions similiar to common farming practises. The use of multiple-treatment experiments with varying levels of disease and one disease free treatment allows the comparison of the effects of different types of epidemics at one location. Disease-loss is calculated as the difference in yield between diseased and the disease-free plot. Standard experimental designs are often employed such as the split plot or randomized complete block design with the two main variables, yield and disease

recorded. Various epidemics may be generated using fungicides, isogenic lines or cultivars.

Disease assessment is commonly measured in one of two ways. Disease incidence, where the number of infected plants or plant units (i.e. leaves) are expressed as a percentage of total plants or plant units is one method. This is often accurate at low levels of disease, when less than 85% of plants are infected, but is inaccurate at higher levels of disease. Disease severity is used to refer to the area of tissue affected by the disease expressed as a percentage of total area. The term disease intensity is used to refer to disease measurement by either of the preceeding terms (James 1974). In disease assessment of blackleg the percentage of the circumference of the stem girdled with canker is a common measure of assessment (Newman 1981, Newman and Bailey 1987, Hammond and Lewis 1987a, Xi and Morrall 1988, Sawatsky 1989), although percentage of the stem penetrated and length of the canker have also been measured (Newman 1981, Newman and Bailey 1987, Hammond and Lewis 1987a). Growth stage of the plant is a vital requirement of disease assessment. A key to the growth stages of oilseed rape has been developed by Harper and Berkenkamp (1975).

Oilseed rape production has been seriously hampered in France (Alabouvette and Brunin 1970), Australia (Roy and Reeves 1975) and Canada (McGee and Petrie 1978) due to Leptosphaeria maculans. Losses have been found to be due to the virulent strain of the pathogen in all cases. The avirulent strain causes only superficial lesions on host plants and is not of economic significance in oilseed rape production. The rapid spread of the virulent strain in Canada has caused concern about production losses in all areas of the prairies.

McGee and Emmett (1977) quantified crop losses in Australia through surveys. They assessed crops for disease at harvest based on the number of plants they considered

severely cankered (more than 50% of the stem girdled). Yield loss was estimated according to the formula:

$$\% \text{ yield loss} = 100 - \frac{W}{W_1} * \frac{100}{N}$$

where; W = total weight of seed in sample

W_1 = average weight of seed per plant from healthy plants in sample

N = total number of plants in sample

A highly significant correlation between disease severity rating and yield loss was found. This was expressed by the formula:

$$\% \text{ yield loss} = 0.7D - 2.0 \quad r = 0.95 \quad (P = 0.001)$$

where D is the disease intensity rating. When half of the plants sampled are considered severely cankered (D = 50), a 38% yield loss can be expected.

2.6 Evaluation of Blackleg Resistance

2.6.1 Methodology

In vitro experiments require use of either pycnidiospore or ascospore inoculum. Wood and Barbetti (1977) compared both types of inoculum when screening for resistance; there was no difference in the type of infection between the two. Ascospore inoculum required only one to two ascospores per seedling leaf to give rise to an infection. Pycnidiospore inoculum required large numbers of pycnidiospores by comparison.

Brassica line evaluation for resistance to L. maculans has been done using a variety of methods and at a variety of growth stages (Sawatsky 1989). A simple and efficient screening method is inoculation of cotyledons shortly after emergence with a pycnidiospore suspension (Delwiche and Williams 1979, Alabouvette et al. 1974). Cotyledons are rated for disease reaction using a scale based on size and appearance of lesion, and the presence or absence of sporulation (Delwiche 1980).

McGee and Petrie (1978) inoculated seedlings by placing pycnidiospore infected oat kernels at the base of each plant in order to facilitate infection. Sawatsky (1989) found this an inefficient method due to the large number of uninfected plants noted. Direct infection of intact stems has not been documented under natural conditions. Helms and Cruickshank (1979) used pycnidiospore infected perlite placed on top of germinating seedlings to cause infection.

In the seedling stage lines have been evaluated using a petiole inoculation method (Newman 1981) or leaf inoculation method (Sawatsky 1989). In the former case, the base of the petiole of the first or second true leaf is wounded with a needle. Then an assay disk soaked in pycnidiospore suspension is placed over the wound. Plants are placed in a humidity chamber for 3 days and kept at 100% rh. The latter method was similar but did not require wounding. Pycnidiospore inoculum was sprayed at 10 psi directly onto leaves and then placed under high humidity .

A method used in the adult stage has been to inoculate the pycnidiospore suspension directly into the stem with a syringe at bolting. This is usually done between the 1st to 4th node (Newman and Bailey 1987).

In the field, lines are sown into soil containing blackleg infested residue (Sawatsky 1989). A variation of this method has been to inoculate plants artificially by pipetting or spraying suspensions of pycnidiospores or ascospores onto leaves with or without wounding (Barbetti 1975, Alabouvette et al. 1974). In some cases additional infested

residue has been spread at the testing site to ensure adequate amounts of primary inoculum.

2.6.2 Correlation Between Methods

Correlation between methods used for screening have been investigated in detail by Sawastsky (1989). Correlation of results between methods appears to vary with the growth stage and organ of the plant used to determine resistance to the disease. Sawatsky found no significant differences between the cotyledon and adult test for 4 of the 5 cultivars of B. napus tested. She also found no significant differences for 3 of 5 cultivars, between the seedling test, where crown cankers were rated, and the adult test. Highly significant differences were found between the cotyledon and seedling test for 4 of 5 cultivars. Newman and Bailey (1987) found good agreement between seedling and adult tests and concluded there was no advantage to screening adult plants for resistance.

Indoor evaluation of Brassica species for resistance to L. maculans has usually been performed by other workers using one or at most a few isolates of the pathogen. In field testing many isolates are present, each of which may vary for the genes for virulence. This may be one explanation for the differences in disease severity ratings which have often been found when accessions or cultivars are evaluated using both indoor and field tests. Variation for virulence of L. maculans isolates has been found by other researchers as discussed previously.

Correlations between cotyledons and field tests have been found to be low or non-significant (Sawatsky 1989, Helms and Cruickshank 1979). This may be due to variation for virulence of isolates in field tests. It may also be a reflection of differences which exist between resistance expressed in the cotyledon and resistance expressed at other points in the infection pathway. Helms and Cruickshank (1979) found field tolerant cultivars showed no resistance when inoculated as seedlings and rated for symptoms on

the cotyledons and hypocotyls. However resistance was observed when rating took place later and was based only on hypocotyl symptoms. Thurling and Venn (1977) did not consider cotyledon symptoms to be a good measure of field resistance. They suggested that resistance was more accurately measured if rating took place later in plant development.

Sawatsky (1989) found the best correlation between greenhouse and field results was obtained when using the seedling test, which rated crown cankers. Newman and Bailey (1987) found results between seedling tests corresponded well with field results for highly resistant or very susceptible brassica lines, but found conflicting results for lines with intermediate resistance. Cargeeg and Thurling (1980b) found good correlation between seedling and field results for field selected lines. They concluded that provided selection intensity was not too high, greenhouse testing was effective in screening for blackleg resistance.

Adult plant test results did not correspond well with field results (Sawatsky 1989). The level of resistance noted in the field test for most cultivars was underestimated by the adult test. Sawatsky suggested this was due to the deposition of inoculum directly into the cortical and vascular tissue of the stem, which bypassed resistance mechanisms which might have existed earlier in the infection pathway.

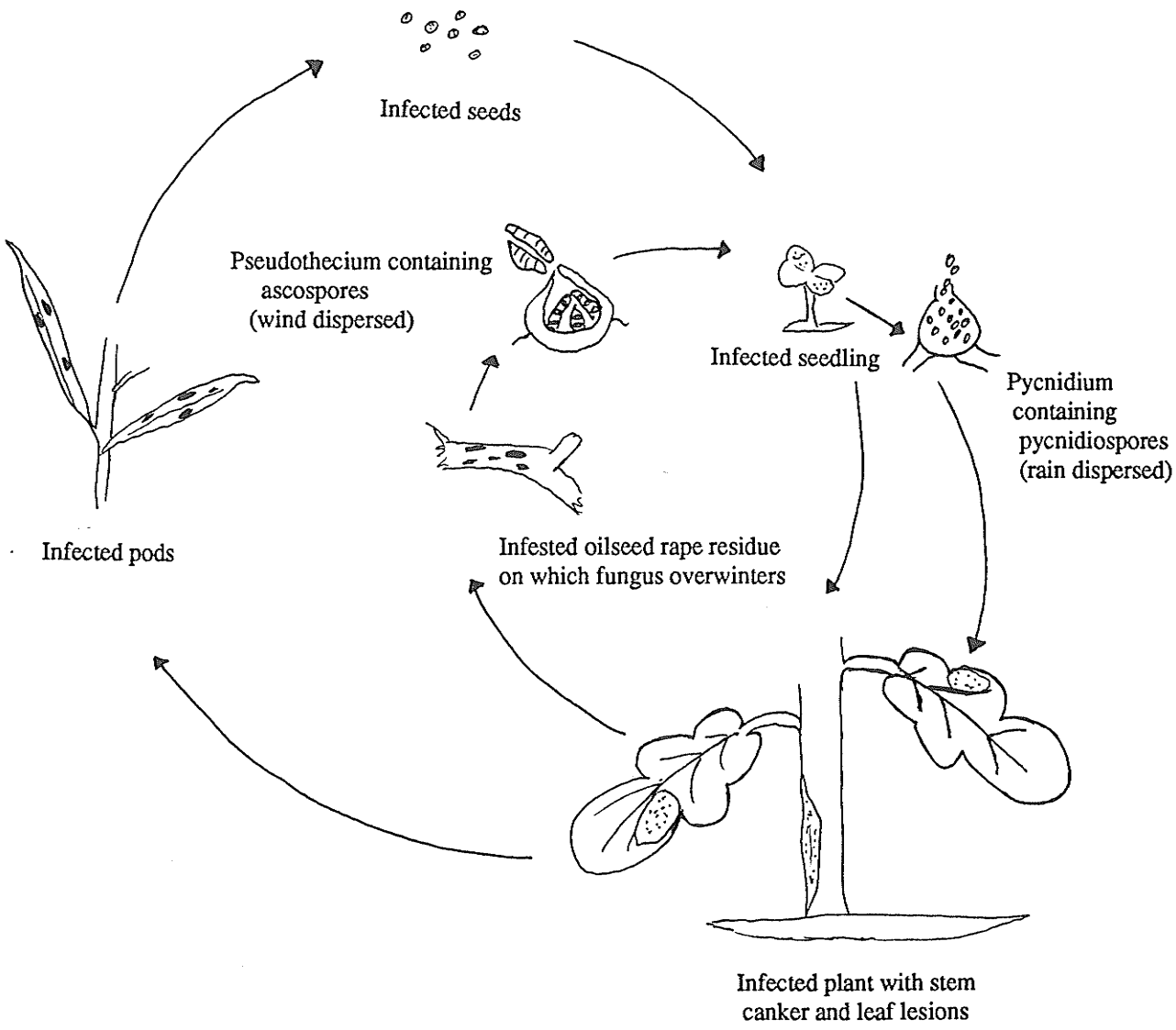


Figure 2.1. Disease cycle of blackleg of oilseed rape, caused by Leptosphaeria maculans.

Evaluation of Brassica rapa L. accessions for resistance to Leptosphaeria maculans (Desm.) Ces. et de Not.

3.1 Abstract

Accessions of B. rapa were evaluated for resistance to L. maculans at the cotyledon and adult growth stages under controlled environmental conditions. No accessions were defined as highly resistant to the pathogen at either stage. Nine accessions of a total of 231 at the cotyledon stage, and 7 accessions of a total of 232 at the adult stage, were defined as moderately resistant. Field screenings performed in 1988 and 1989 identified many accessions which contained greater resistance to L. maculans than the susceptible check, Tobin. A weak correlation ($r = 0.27^{***}$) was found between the cotyledon and adult test; there were no significant correlations among other tests.

A number of accessions which were defined as moderately resistant in the adult test were retested against isolates from different geographical locations. Differences in variation for virulence among isolates was noted. Winter type accessions were generally found to be more resistant than spring types to most isolates of the pathogen.

3.2 Introduction

Blackleg disease of oilseed rape and oilseed turnip rape (Brassica napus L. and B. rapa L. respectively), causal agent Leptosphaeria maculans causes significant yield reductions of these crops. Yield losses in Europe and Australia have been reduced by use of resistant or tolerant oilseed rape cultivars (Brunin 1972, Wratten 1977). In Canada a number of B. napus oilseed rape cultivars have been registered which are recognized as 'fair' with respect to blackleg resistance (Anon 1989).

There are no cultivars of B. rapa in Canada which have significant blackleg resistance. The intent of this study was to screen accessions of B. rapa from the University of Manitoba B. rapa collection for resistance to one isolate of L. maculans at 2 growth stages. Accessions were also screened in the field disease nursery for 2 seasons.

3.3 Materials and Methods

Inoculum preparation. Accessions were screened at the cotyledon stage (growth stage (GS) 1 of the Harper and Berkenkamp (1975) scale), and the adult stage (GS 3.3) in the growthroom and greenhouse against one isolate of the pathogen (Pl86-14). The isolate had been collected from infected oilseed rape in 1986 from southwestern Manitoba. Inoculum was increased by surface sterilizing infected cotyledons of Westar with a 5% solution of sodium hypochlorite for 4 minutes, and plating on V8 juice agar containing rose bengal and 1% streptomycin sulfate. After 7 days pieces of mycelium were cut out and used to seed other V8 juice agar plates. Plates were incubated under blacklight (Sylvania R30T8/350BL) at room temperature until cultures sporulated (7-10 days). After 10 days plates were flooded with sterile distilled water and the surface was rubbed with a sterile glass slide. The resulting pycnidiospore suspension was strained through 4 layers of autoclaved cheese cloth and centrifuged for 30 minutes at 5000 rpm.

The pellet was resuspended in approximately 5 ml of sterile distilled water and stored at -15°C. For each inoculation pycnidiospore suspension was thawed and the inoculum concentration adjusted to 10^7 pycnidiospores ml^{-1} with sterile distilled water and the aid of a haemocytometer.

Cotyledon test. Six seeds of each of 232 accessions were planted in soilless potting mix (W.R. Grace and Co. Ltd., Ajax, Ontario) in 'jiffy' pots within flats. Flats were placed in the growth chamber at 20/18°C day/night temperatures and a 16 h photoperiod was supplied by fluorescent light banks. Flats were watered daily with tap water and at 12 days after seeding with a solution of 20-20-20 (nitrogen-phosphorus-potassium) fertilizer. At 6 days after planting when the cotyledons had fully expanded, each cotyledon was wounded with a sterile needle and a 10 µl drop of pycnidiospore suspension placed over the wound. Ten days later cotyledons were rated for disease severity (DS) according to the scale devised by Delwiche (1980) (Appendix 3.1).

Adult test. Cotyledons of the seedlings used in the cotyledon test were removed to halt progression of the disease. Six seedlings of each line were potted (3 seedlings per pot) in soilless mix and placed on benches in the greenhouse. (Winter types were vernalized for 6 weeks at 4°C, 12 h photoperiod, after removal of cotyledons, but before potting). Natural daylight was supplemented to 16 h with fluorescent light banks. Plants were watered daily with tap water and fertilized at 2 week intervals with a solution of 20-20-20. At bolting, growth stage (GS 3.3) the 2nd internode of each plant was injected using a hypodermic syringe with 10 µl of pycnidiospore inoculum. Plants were rated for disease severity 10 days later and once a week thereafter for 4 weeks using a scale based on the lesion length and circumference of the stem girdled by the lesion (Table 3.1). Accessions which rated lowest in the adult test, and other lines of interest were retested against other L. maculans isolates.

Field tests. Most accessions which were screened in the greenhouse tests were seeded in the blackleg nursery at Souris, Manitoba in 1988. Accessions were seeded in a randomized complete block experiment of 2 replications. Each accession was seeded in single row plots of 3.1 meters, with 0.61 m between rows. Carbofuran (Furadan CR-10) was applied with the seed at 5.6 kg ha^{-1}. Disease severity ratings for 10 plants from each plot were taken at GS 5.3 for the circumference of stem girdled by canker: 0 - no disease, 1 - <25%, 2 - 25-50%, 3 - 50-75%, 4 - 75-100%, 5 - plant dead. Those accessions rating less than 1.0 in 1988 were retested in 1989 in the same manner at the Souris blackleg nursery.

Accessions were ranked according to disease severity rating and correlations were made using the Spearman correlation procedure (rank correlation).

3.4 Results and Discussion

The results of each test for each accession tested against L. maculans are presented in Appendix 3.2. An analysis of variance was performed on weekly disease severity ratings of the adult test, using plants within lines as replications. The disease severity ratings from week 4 were used to represent the adult test as this week had a wide range of reactions (DS 4.6 to 10.0) and a lower error mean square than other weeks.

None of the accessions were resistant when resistance is defined by a disease severity rating from 0.0 to 2.9 in the cotyledon test and from 0.0 to 3.2 in the adult test (Table 3.2). Nine accessions were moderately resistant (DS 3.0 - 5.9) in the cotyledon test and 7 in the adult test (DS 3.3 - 6.5). The 20 accessions with the lowest disease severity ratings in the cotyledon test and adult test had 7 accessions in common - UM1086W, UM1090W, UM1092W, UM1094W, UM1067 and UM1124 (Table 3.3). Spearman correlation coefficients indicated that there was a weak correlation ($r = 0.27^{***}$ $P = 0.001$) between the two tests over all accessions tested. Winter type accessions were

moderately resistant in both of these tests. Wratten (1977) found that winter type oilseed rape and oilseed turnip rape often contain greater resistance to blackleg than do spring types.

In the 1988 field test, 136 of a total 222 accessions tested scored lower than the susceptible check Tobin (DS 1.4) (Table 3.4). In 1989, 53 of a total of 70 accessions tested scored lower than Tobin (DS 1.6). The top 20 accessions from each field test year have 6 accessions in common - UM1072W, UM1063, UM1095, UM1117, UM1155, UM1204. Spearman correlation coefficients were non-significant between the tests.

Two accessions, UM1072W and UM1063 scored among the top 20 accessions in the cotyledon, field 1988 and field 1989 tests. However, apart from a weak correlation between the cotyledon and adult test, all other correlations between tests were non-significant. Most of the accessions in the collection (62%) are of Indian or Pakistani origin. Therefore it is not surprising most of the accessions with low disease severity ratings are from India and Pakistan. Winter type accessions UM1088W, UM1090W, UM1092W, and UM1094W were all from Finland. They were not tested in the field due to the vernalization requirement. UM1072W is a winter type from Turkey and was inadvertently tested in the field.

Some accessions which had low disease severity ratings in week 4 were tested against a number of isolates from Australia (WA14, WA32, WA51, and OA23), Manitoba (Pl86-14 and Plat2) and Wisconsin (PHW478) at the cotyledon and adult stage (Figures 3.1 and 3.2). There was considerable variation of disease severity ratings within accessions to isolates of the pathogen. This was possibly due to the outcrossing nature of B. rapa. Mean disease severity ratings were generally high on all spring type accession-isolate combinations in either cotyledon or adult tests. Winter type accessions exhibited greater resistance than spring types to most isolates. Significant differences ($P = 0.05$) existed among accessions for all isolates in the cotyledon test. In the adult test there were no

significant differences between isolates WA51 and OA23 among accessions, but there were among accessions tested against all other isolates.

Disease severity ratings were classified as resistant, moderately resistant and susceptible for each accession-isolate combination (Tables 3.5 and 3.6). Some of these isolates have previously shown variation for virulence on B. napus cultivars (Mengistu et al. 1989). The results in this study indicated variation for virulence among isolates over the B. rapa accessions. Also, there was variation in the disease reactions of accessions to isolates of the pathogen, in the cotyledon and the adult test. In the cotyledon test the winter types tested were resistant to the isolate from Wisconsin (PHW478) but variable to other isolates. The Manitoba isolates (Plat2 and Pl86-14) elicited similar disease reactions over the accessions tested. Australian isolates elicited variable responses among accessions. In the adult test susceptible responses on all accessions were elicited by the Australian isolates WA51 and OA23. All other isolates elicited variable reactions on spring type accessions, while moderately resistant responses were elicited on most winter types. The UM1090W-WA14 accession-isolate combination resulted in a susceptible disease reaction.

This study indicated variability in disease reactions to L. maculans among the accessions tested. None of the accessions were resistant to Pl86-14 in either the cotyledon or adult tests, however a number of accessions were moderately resistant. Disease severity ratings of winter types was lower than most spring types in these tests, although only a limited number of winter types was tested. There were many accessions with low disease severity ratings in the field tests, compared to the susceptible check Tobin, although there was no relationship between field tests and the cotyledon or adult tests. The usefulness to a breeding program, of the accessions identified as moderately resistant in cotyledon or adult test requires further evaluation.

Cotyledon and adult test results were not significantly correlated with field test results. Greenhouse testing using one isolate of the pathogen may elucidate specific genes for resistance to that particular isolate. However, as was found when the better lines from the adult test were tested against a number of other isolates, there was variability for virulence among isolates. In field tests many isolates of the pathogen, with different genes for virulence may be present, which necessitates that accessions be evaluated against a range of isolates. Environmental factors such as temperature (Barbetti 1975) and precipitation (McGee 1977) have been shown to influence the host-pathogen interaction in the oilseed rape-L. maculans system. Therefore variation in disease reactions may be expected between indoor and field tests when evaluating accessions for resistance to L. maculans. Sources of resistance determined from greenhouse screening must be tested in the field in order to evaluate their effectiveness in a breeding program.

Table 3.1. Rating scales for crown canker due to Leptosphaeria maculans based on length and circumference measurements. Disease severity rating (DS) obtained by addition of scales (l + c).

score	lesion length on stem (l)	circumference of stem girdled by lesion (c)
0	no infection	no infection
1	< 10 mm	< 25%
2	10-19 mm	25-50%
3	20-29 mm	50-75%
4	30 mm and greater	75-100%
5	plant dead	plant dead

Table 3.2. Disease reactions of accessions of Brassica rapa tested at the cotyledon and adult growth stages with one isolate of Leptosphaeria maculans (isolate Pl86-14).

Disease reaction	Cotyledon test	Adult test
Resistant	0	0
Moderately resistant	9	7
Susceptible	222	225
Total	231	232

Cotyledon test: Resistant 0.0 - 2.9
Moderately resistant 3.0 - 5.9
Susceptible 6.0 - 9.0

Adult test: Resistant 0.0 - 3.2
Moderately resistant 3.3 - 6.5
Susceptible 6.6 - 10.0

Table 3.3. Disease severity of lowest scoring Brassica rapa accessions in cotyledon and adult test using one isolate of Leptosphaeria maculans (Pl86-14). Values expressed as the mean of 6 plants ± SE.

Cotyledon test[1] (231 accessions tested) (0 to 9 scale)		Adult test (232 accessions tested) (0 to 10 scale)	
UM1070	3.3 ± 1.1	UM1094W	4.6 ± 0.6
UM1090W	3.5 ± 3.5	UM1088W	5.0 ± 0.6
UM1226	3.7 ± 1.6	UM1092W	5.5 ± 1.5
UM1092W	4.0 ± 3.0	UM1090W	6.0 ± 0.3
UM1472	4.0 ± 1.1	UM1130	6.2 ± 0.7
UM1086W	4.8 ± 1.7	UM1022	6.2 ± 1.4
UM1225	4.8 ± 1.5	UM1140	6.5 ± 1.3
UM1094W	5.5 ± 0.5	UM1158	7.0 ± 0.8
UM1200	5.5 ± 0.3	UM1141	7.2 ± 0.9
UM1065	6.0 ± 1.3	UM1144	7.2 ± 0.3
UM1187	6.0 ± 1.1	UM1314	7.3 ± 1.1
UM1124	6.3 ± 1.2	UM1150	7.5 ± 0.8
UM1236	6.5 ± 1.5	UM1337	7.5 ± 1.4
UM1067	6.7 ± 0.8	UM1124	7.7 ± 0.6
UM1068	6.7 ± 0.6	UM1075	7.7 ± 0.6
UM1072W	6.7 ± 1.5	UM1472	7.8 ± 0.8
UM1202	6.8 ± 1.4	UM1067	7.8 ± 0.8
UM1063	7.0 ± 0.9	UM1120	7.8 ± 0.7
UM1071	7.0 ± 0.9	UM1333	7.8 ± 0.8
UM1082	7.0 ± 1.4	UM1086W	8.0 ± 1.4
Westar	8.6 ± 0.1	Westar	8.6 ± 0.1

[1] W indicates a winter type line.

Table 3.4. Disease severity of lowest scoring Brassica rapa accessions in field tests in 1988 and 1989. Values expressed as the mean of 10 plants ± SE.

Field test 1988[1] (222 accessions tested) (0 to 5 scale)		Field test 1989 (70 accessions tested) (0 to 5 scale)	
UM1287	0.3 ± 0.1	UM1449	0.10 ± 0.2
UM1258	0.4 ± 0.1	UM1206	0.25 ± 0.2
UM1117	0.4 ± 0.4	UM1226	0.50 ± 0.2
UM1072W	0.4 ± 0.4	UM1440	0.50 ± 0.3
UM1057	0.4 ± 0.2	UM1328	0.50 ± 0.1
UM1015	0.4 ± 0.0	UM1117	0.55 ± 0.4
UM1153	0.5 ± 0.4	UM1287	0.65 ± 0.5
UM1248	0.5 ± 0.5	UM1197	0.65 ± 0.2
UM1236	0.5 ± 0.1	UM1057	0.70 ± 0.1
UM1145	0.5 ± 0.2	UM1063	0.70 ± 0.1
UM1095	0.6 ± 0.3	UM1192	0.70 ± 0.3
UM1179	0.6 ± 0.3	UM1095	0.75 ± 0.3
UM1257	0.6 ± 0.2	UM1155	0.80 ± 0.3
UM1070	0.6 ± 0.0	UM1177	0.80 ± 0.6
UM1204	0.6 ± 0.3	UM1061	0.85 ± 0.4
UM1386	0.6 ± 0.1	UM1240	0.90 ± 0.2
UM1141	0.7 ± 0.2	UM1072W	0.95 ± 0.4
UM1155	0.7 ± 0.1	UM1204	0.95 ± 0.3
UM1063	0.7 ± 0.2	UM1238	1.00 ± 0.3
UM1297	0.7 ± 0.2	UM1292	1.00 ± 0.3
Tobin	1.4 ± 0.1	Tobin	1.60 ± 0.1

[1] W indicates a winter type line.

Figure 3.1. Disease severity of Brassica rapa accessions challenged with isolates of Leptosphaeria maculans from different geographical origins (cotyledon test). Values expressed as the mean of 6 plants ± SE.

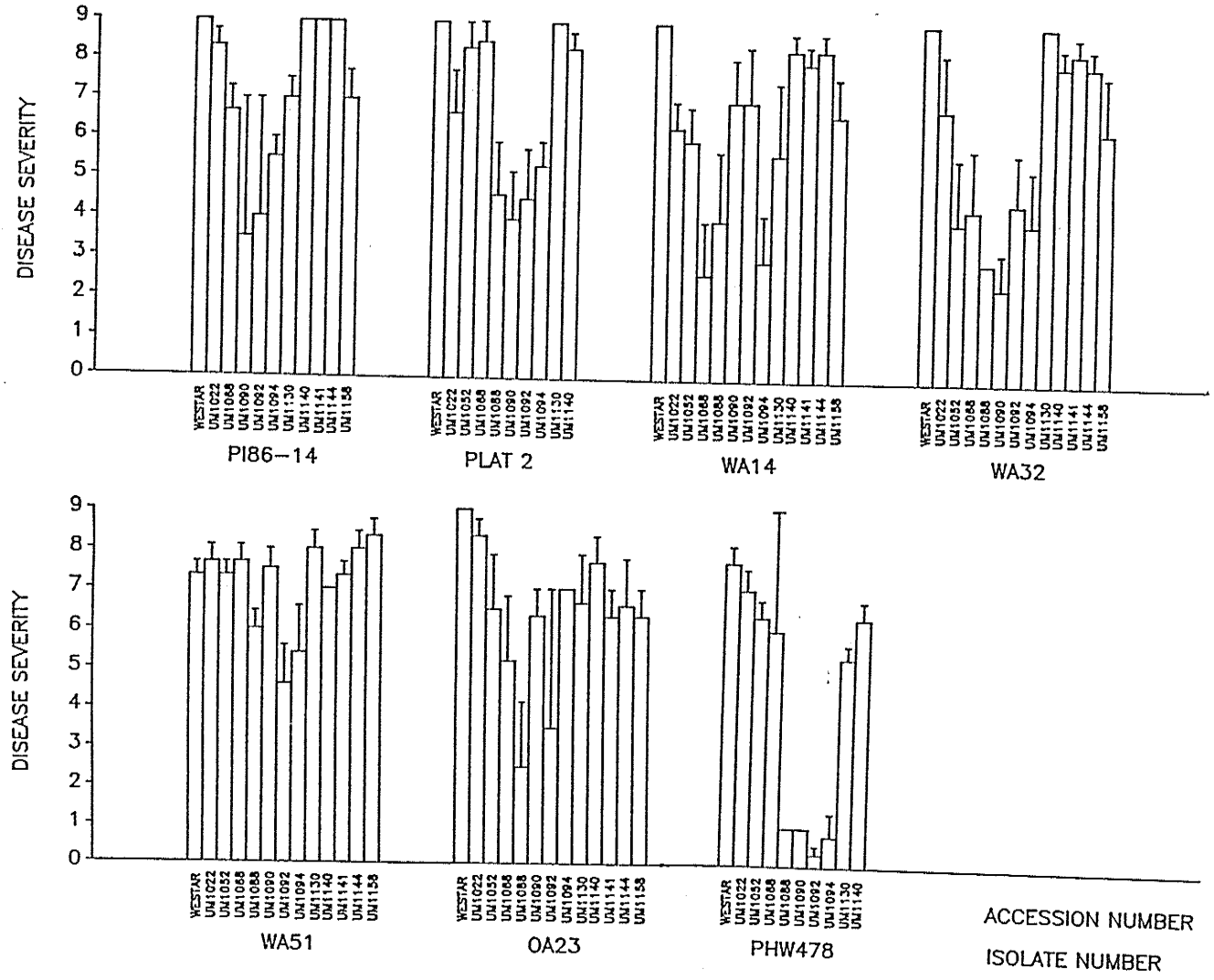


Figure 3.2. Disease severity of Brassica rapa accessions challenged with isolates of Leptosphaeria maculans from different geographical origins (adult test). Values expressed as the mean of 6 plants ± SE.

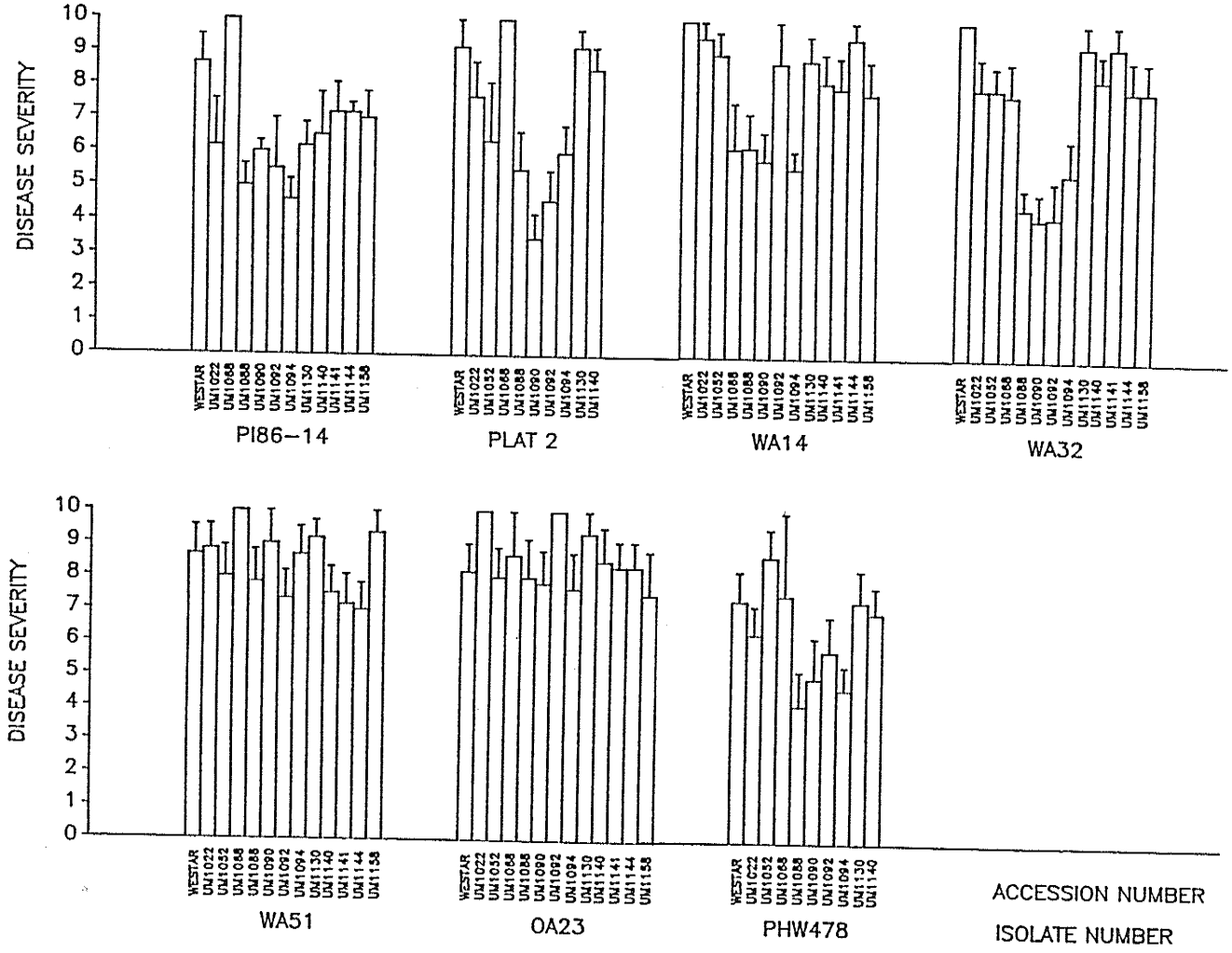


Table 3.5. Disease reactions of accessions of Brassica rapa challenged with isolates of Leptosphaeria maculans (cotyledon test).

Accession Number	Isolate Number						
	Pl86-14	Plat2	WA14	WA32	WA51	OA23	PHW478
Westar	S	S	S	S	S	S	S
UM1022	S	S	S	S	S	S	S
UM1052	-	S	S	M	S	M	S
UM1068	S	S	R	M	S	M	S
UM1088W	M	M	M	M	S	R	R
UM1090W	M	M	S	R	S	S	R
UM1092W	M	M	S	M	M	M	R
UM1094W	M	M	M	M	M	S	R
UM1130	S	S	M	S	S	S	M
UM1140	S	S	S	S	S	S	M
UM1141	S	-	S	S	S	M	-
UM1144	S	-	S	S	S	S	-
UM1158	S	-	S	S	S	S	-

Resistant (R) 0.0 - 2.9
Moderately resistant (M) 3.0 - 5.9
Susceptible (S) 6.0 - 9.0

Table 3.6. Disease reaction of accessions of Brassica rapa challenged with isolates of Leptosphaeria maculans (adult test).

Accession Number	Isolate Number						
	Pl86-14	Plat2	WA14	WA32	WA51	OA23	PHW478
Westar	S	S	S	S	S	S	S
UM1022	M	S	S	S	S	S	M
UM1052	-	M	S	S	S	S	S
UM1068	S	S	M	S	S	S	S
UM1088W	M	M	M	M	S	S	M
UM1090W	M	M	M	M	S	S	M
UM1092W	M	M	S	M	S	S	M
UM1094W	M	M	M	M	S	S	M
UM1130	M	S	S	S	S	S	S
UM1140	M	S	S	S	S	S	S
UM1141	S	-	S	S	S	S	-
UM1144	S	-	S	S	S	S	-
UM1158	S	-	S	S	S	S	-

Resistant (R) 0.0 - 3.2
Moderately resistant (M) 3.3 - 6.5
Susceptible (S) 6.6 - 10.0

Variation for virulence of Leptosphaeria maculans isolates from different geographical origins on lines of the genus Brassica

4.1 Abstract

A study was conducted to investigate the virulence of 30 isolates of Leptosphaeria maculans from Souris, Manitoba; Melfort, Saskatchewan; and Western Australia over a range of susceptible to resistant brassica lines. Isolates were categorized into groups based on disease reactions elicited on host lines at 2 growth stages; the cotyledon stage (cotyledon test) and the adult stage (seedling test).

In both tests highly significant differences in disease severity on lines were obtained among isolates within origins and among origins. A highly significant line x isolate interaction was found in both tests. Two lines (R8314.47 and UM3132) at the cotyledon stage and 3 lines (R8314.47, UM3132 and Global) at the adult stage were used in differentiating isolates. At the cotyledon stage R8314.47 was resistant or moderately resistant to all isolates from Manitoba and Saskatchewan, but susceptible to most isolates from Western Australia. At the adult stage R8314.47 was resistant to all isolates from Manitoba and Saskatchewan, but moderately resistant or susceptible to Western Australian isolates. UM3132 exhibited variation in disease reaction to isolates within origins at both stages. Global was susceptible to almost all isolates at the cotyledon stage but exhibited variable disease reactions to isolates within origins at the adult stage.

The significance of these findings is discussed with reference to the nature of resistance and its implications in breeding for resistance to L. maculans.

4.2 Introduction

Blackleg disease of oilseed rape (Brassica napus L. and B. rapa L.), causal agent Leptosphaeria maculans (Desm.) Ces. et de Not., has become a serious production constraint in many areas of the Canadian prairies. Previously production of oilseed rape has been seriously hampered in France (Alabouvette and Brunin 1970) and Australia (Roy and Reeves 1975). Development of blackleg resistant cultivars has been of primary importance for the continued production of oilseed rape in these countries.

The development of resistant cultivars requires an understanding of host-pathogen interactions. A host line may be used in a differential series of lines to identify pathogen isolates when the disease reaction (interaction phenotype) is known. Knowledge of the variability for virulence of isolates in a pathogen population will facilitate plant breeding strategy, and possibly indicate the consequences of introducing new sources of host resistance into an area.

Variation for virulence of isolates of L. maculans has been determined in Australia (Thurling and Venn 1977, Cargeeg and Thurling 1980a), and in Britain (Newman 1984). Detection of differential interactions between L. maculans and brassica populations indicate that strains of varying virulence exist among geographical origins and within each origin. This study reports on the variation for virulence of isolates of L. maculans from specific origins over a range of resistant to susceptible lines of the genus brassica. Lines that may be useful in establishing a differential series are discussed.

4.3 Materials and Methods

Eight double haploid lines of Brassica napus (Westar, Regent, Marnoo, Karat, Global, R8311, R8314.26, R8314.47), 2 B. rapa lines of breeders seed (Tobin and BLC198), and

2 B. juncea lines each of which had been selfed 4 times (UM3132 and UM3233) were used in this study.

Thirty isolates of the pathogen were used, each derived from single ascospores. Seventeen were obtained from one field at Souris, Manitoba (MA), 4 from one field at Melfort, Saskatchewan (SA), 7 from Western Australia (WA), 1 from France and 1 from New South Wales (NSW), Australia, the latter 2 isolates designated OA. Those isolates from Manitoba and Saskatchewan were obtained from infested pieces of oilseed rape residue, while the others were obtained from the University of Manitoba L. maculans collection.

Pieces of infested oilseed rape residue were attached to the underside of the lid of a petri dish with petroleum jelly. Lids were flooded with distilled water, then drained 2 minutes later. Lids were placed over the bottoms of the petri dishes which contained water agar. Dishes were then sealed with parafilm and placed under continuous fluorescent light at room temperature for 24 to 36 h. At this time germinating single ascospores were taken from the water agar and plated on V8 juice agar containing rose bengal and 1% streptomycin sulfate. One ascospore was obtained from each piece of trash.

Inoculum preparation. All isolates were increased on V8 juice agar. After 10 days plates were flooded with sterile distilled water and the surface was rubbed with a sterile glass slide. The resulting pycnidiospore suspension was strained through four layers of autoclaved cheese cloth and centrifuged for 30 minutes at 5000 rpm. The pellet was resuspended in approximately 5 ml of sterile distilled water and stored at -15°C. For each inoculation pycnidiospore suspension was thawed and the inoculum concentration adjusted to 10^7 pycnidiospores ml^{-1} with the aid of a haemocytometer.

Cotyledon test. Ninety-six seeds were planted in 'Koral' flats (Koral Co., Bramalea, Ontario) in 'Metromix' a soilless potting mix (W.R. Grace and Co. Ltd., Ajax, Ontario).

The flats were placed in a growth room with a daylength of 16 h and 20/15^{o}C day/night temperatures. Flats were watered daily with tap water and fertilized with a solution of 20-20-20 (nitrogen-phosphorus-potassium fertilizer) on the 5th and 10th day after seeding.

Cotyledons were wounded on the 6th day after seeding with a needle and inoculated with a 10 µl droplet of the spore suspension. Disease severity was rated 10 days after inoculation on a scale devised by Delwiche (1980) (Appendix 3.1).

The experiment was conducted as a split-plot design with 4 replications in which isolates were the whole plots and lines the subplots. Four plants of each line were tested in each replication. Isolates were not randomized within replicates due to the method of inoculation. The same design was used for the seedling test described below.

Seedling test. Forty-eight seeds were planted in 'Koral' flats in 'Metromix' using the same experimental design as in the cotyledon test. The 'Koral' flats were placed on flats that had been filled with a mixture of 2 g granular fertilizer (20-20-20) per 500 grams vermiculite. Flats were placed on greenhouse benches with normal daylight supplemented with fluorescent light to 16 h. Flats were watered with tap water daily and fertilized twice weekly with full strength Hoagland solution for crucifers (Williams 1985).

Petioles of the second true leaf were inoculated 21 days after seeding when the second true leaf had fully expanded. The petiole was wounded in the leaf axil with a needle in four places 1 mm apart. A 6 mm filter disk (Whatman #3) that had been soaked in a spore suspension was applied over the wounds. Plants were covered with polyethylene for 72 h.

Disease severity was rated at the crown 8 weeks after inoculation. Ratings were taken for the following measurements; length of crown lesion, circumference of stem girdled (using the 0 to 4 scale for each found in of Table 3.1) and penetration of stem (Figure

4.1). These ratings were used to determine the volume of diseased tissue (VDT) in cubic centimeters.

$$VDT = (\pi r^2_1 - \pi r^2_2) \times l \times c/4$$

where: πr^2_1 - the total area in the cross-section of the stem.

πr^2_2 - the area of healthy tissue in cross section of the stem.

l - the length of the lesion.

c - the circumference of the stem girdled in 25% increments.

4.4 Results

Cotyledon test. Mean disease severity ratings and standard errors for all line-isolate combinations are presented in Appendix 4.1. High disease severity ratings were obtained on many of the lines from most isolates. The B. juncea line UM3233 was immune or resistant to all isolates.

Isolates were categorized into 7 groups based on the reaction elicited on brassica lines. Reactions of brassica lines to pathogen isolates were classified as resistant, moderately resistant and susceptible (Table 4.1). Most groups could be differentiated with 2 lines, R8314.47 and UM3132.

The analysis of variance was used for lines selected on the basis of differences in disease reaction to isolates. This selection negated the null hypothesis (that there was no difference between lines). In spite of this the significance of factors and interactions between factors illustrated the differences. Therefore the levels of significance of the F tests must be interpreted with caution. There were no significant differences among isolate origins for lines R8314.47 and UM3132 (Table 4.2). However there were highly significant differences among isolates and among isolates within each origin.

Interactions among lines and origins and among isolates and lines (within origins) were obtained.

Disease severity ratings of isolates on the differentiating lines are presented in Figure 4.2. Disease severity on R8314.47 ranged from 1.1 ± 0.3 to 8.8 ± 0.5. Isolates from Manitoba and Saskatchewan gave lower disease severity ratings than isolates from Western Australia. On UM3132 disease severity was high for most isolates except the isolate from France.

Seedling test. Mean disease severity ratings and standard errors for all line-isolate combinations are presented in Appendix 4.2. Isolates were grouped into 9 groups of a possible 27 on the basis of the disease reactions of 3 lines; R8314.47, UM3132 and Global (Table 4.3). The disease reactions were determined by the VDT of each line-isolate combination. A resistant reaction (VDT 0.0 to 0.2) was defined by a maximum rating of 1.3 for each of the three measurements comprising the VDT. A moderately resistant reaction (VDT of 0.3-1.5) was defined by ratings between 1.3 and 2.5 for each measurement determining the VDT. A susceptible reaction (VDT 1.6-4.0) was determined by ratings of 2.6 and greater for each disease measurement of the VDT.

There were highly significant differences among isolates and lines (Table 4.2). Within origins, Manitoba and Western Australia, isolates differed highly significantly from each other. However there were no significant differences among isolates from Saskatchewan or any difference between the isolate from France and the isolate from NSW, Australia. There were highly significant line by origin interactions and isolate by line (within origin) interactions.

Disease severity caused by isolates for each of the 3 brassica lines are presented in Figure 4.3. Infection of R8314.47 with isolates from Manitoba and Saskatchewan resulted in very low disease severity ratings. However most isolates from Western Australia, France and NSW, Australia elicited high disease severity ratings. Disease

severity of isolates on Global varied between 0.1 ± 0.1 and 2.5 ± 0.8. Global was susceptible to 1 isolate from Manitoba and 2 isolates from Western Australia, resistant to 4 isolates from Manitoba and moderately resistant to all others (Table 4.3). No trends were visible for isolates from specific origins. Generally disease severity was high on UM3132 from all origins although there were isolates which elicited a moderately resistant reaction. The isolate from France elicited a resistant reaction.

4.5 Discussion

Virulence of Leptosphaeria maculans was defined by the extent of lesion development on cotyledons (cotyledon test) or the extent of canker development in adult plants (seedling test). Differential interactions between isolates of the pathogen and lines of the host were indicated by the significant isolate by line (within origin) interaction. When isolates were grouped according to their reactions on brassica lines, 2 lines in the cotyledon test and 3 lines in the seedling test were found to be useful for differentiation among isolates.

The groupings of isolates suggest that differences in virulence exist among different geographical areas. In the cotyledon test isolates from France and NSW, Australia and all but one from Western Australia (WA40), elicited a susceptible reaction on R8314.47. All Manitoba and Saskatchewan isolates elicited a resistant reaction. In the seedling test R8314.47 was susceptible or moderately resistant to all Western Australian isolates except WA40, the isolate from France (OA45) and the NSW, Australian isolate (OA23). It was resistant to all isolates from Manitoba and Saskatchewan.

The B. napus line R8314 has two genes for resistance to L. maculans (Sawatsky 1989). It was suggested three reactions would be elicited in the host depending on the number of alleles confering resistance; dominant alleles at each loci conditioning complete resistance, a dominant allele at one locus conditioning moderate resistance and

recessive alleles at both loci conditioning a susceptible reaction. In the present study most of the isolates from Western Australia and the isolates from France and NSW, Australia possess the virulence genes necessary to overcome the resistance of R8314.47, while the isolates from Manitoba and Saskatchewan do not. R8314.47 appears to be a useful line to determine variation for virulence in L. maculans isolates among and within geographical origins.

There was a wide range of disease severity ratings on UM3132 in both cotyledon and seedling tests. The disease severity ratings formed a continuous distribution between resistance and susceptibility in both tests. Cargeeg and Thurling (1980a) also found a continuous distribution of disease severity ratings on B. napus lines when inoculated with isolates of L. maculans. They suggested that a continuous distribution of disease severity ratings indicated polygenic control of resistance. In this study as in that of Cargeeg and Thurling specific interactions between lines and isolates were found which suggest one or more genes have a large effect on resistance.

The variable disease reactions observed on UM3132 make it a useful line in differentiation of isolates. In the cotyledon test more of the isolates from Manitoba and Saskatchewan are virulent on UM3132 than isolates from Western Australia. Most of the Manitoba and Saskatchewan isolates elicit a susceptible disease reaction, while most of the Western Australian isolates elicit a moderately resistant reaction. The isolates from France and NSW, Australia elicited a resistant reaction. In the seedling test UM3132 was moderately resistant or susceptible to Manitoba, Saskatchewan and Western Australian isolates. It was resistant only to the isolate from France. It appears there is a difference in the virulence of Manitoba and Saskatchewan isolates with that of Western Australian isolates at the cotyledon stage, but not at the adult stage.

Variable disease reactions were obtained on Global in the seedling test, although it was susceptible to almost all isolates at the cotyledon stage. As with UM3132 there were

isolates within most of the origins which were capable of overcoming the resistance of Global. Global proved to be useful to differentiate isolates in the seedling test. Western Australian isolates appear to be more virulent on Global, eliciting moderately resistant and susceptible reactions, than Manitoba or Saskatchewan isolates which elicited moderately resistant and resistant reactions. However a susceptible disease reaction was elicited by a Manitoba isolate which indicates that the complete range of virulence exists in isolates from Manitoba for this cultivar.

There was not a strong relationship between disease reactions in the cotyledon and seedling test. Only 15 of 30 isolates gave the same disease reaction in both tests for R8314.47. For UM3132, 20 of 30 isolates gave the same reaction. The resistance of Global was expressed in the seedling test but not in the cotyledon test. Useful blackleg resistance genes found in Global would be overlooked if only the cotyledon test were used. Other researchers have suggested that a disease severity rating reflecting the rate of development of disease over a longer time period provides a better measure of resistance than a short time period (Thurling and Venn 1977, Helms and Cruickshank 1979).

A wide range of variation for virulence within each geographic origin studied as well as among origins was found in this study. The fact that isolates exist which can overcome the resistance of R8314.47, suggest that isolates should be screened prior to the introduction of the resistance genes to be sure it will be effective. The existence of virulent isolates suggests that the resistance of R8314.47, if used as a source of resistance in breeding programs, may be short-lived in Canada.

Petrie and Lewis (1985) have produced fertile pseudothecia from crosses of virulent isolates of L. maculans from Britain, Australia and Canada. Since the pathogen is capable of recombination, and possibly mutation, there is the potential that isolates carrying virulence genes with the ability to overcome the resistance of R8314.47 may arise. Resistance dependent on one or a few genes has been termed differential or

vertical resistance (Vanderplank 1968). The major advantage to this type of resistance is the ease with which the resistance genes may be transferred from a source to a desirable cultivar. However, the fewer the genes for resistance, the greater is the possibility of new virulent isolates with the ability to overcome the resistance. Loss of resistance dependent on one or a few genes has been clearly documented in host-pathogen systems, such as potato-Phytophthora infestans (Gallegly 1968). When using only a few genes for resistance in a breeding program it has been suggested the genes should be from various sources and possibly used with particular strategies in mind such as gene pyramiding or regional deployment of resistance genes (Zadoks and Schein 1979).

Resistance dependant on many genes, has been called polygenic or horizontal (Vanderplank 1968). Cargeeg and Thurling (1980a) suggested the oilseed rape-L. maculans association may follow the model of Parlevliet and Zadocks (1977) which suggests individual genes in a polygenic system could be vertical and operate on a gene-for-gene basis with virulence in the pathogen. They suggested this type of resistance to be more stable and longer lasting than differential resistance. The probability that all resistance genes will be overcome by a virulent isolate would be less than if resistance were dependant on only one or a few genes. The results obtained in this experiment for the lines Global and UM3132 are similar to the results obtained by Cargeeg and Thurling. Although the resistance of Global and UM3132 does not confer complete resistance, these lines may still contain resistance genes useful in a breeding program.

The results from this study indicate that there was variation for virulence among isolates from different geographical origins as well as within isolates from the same origin over the brassica lines used here. This has implications for breeding strategy because it suggests resistance dependent on one or a few genes may be rapidly overcome by virulent isolates of the pathogen arising from sexual recombination or mutation. Differential interactions were seen between isolates of L. maculans from Western

Australia and those from Canadian origins on R8314.47. This line might be investigated further for its potential value in establishing a recognized differential series of lines to identify pathogen isolates.

Figure 4.1. Penetration measurement of diseased stem (r). Results used with measurements taken in Table 3.1 to determine disease severity (VDT) in seedling test.

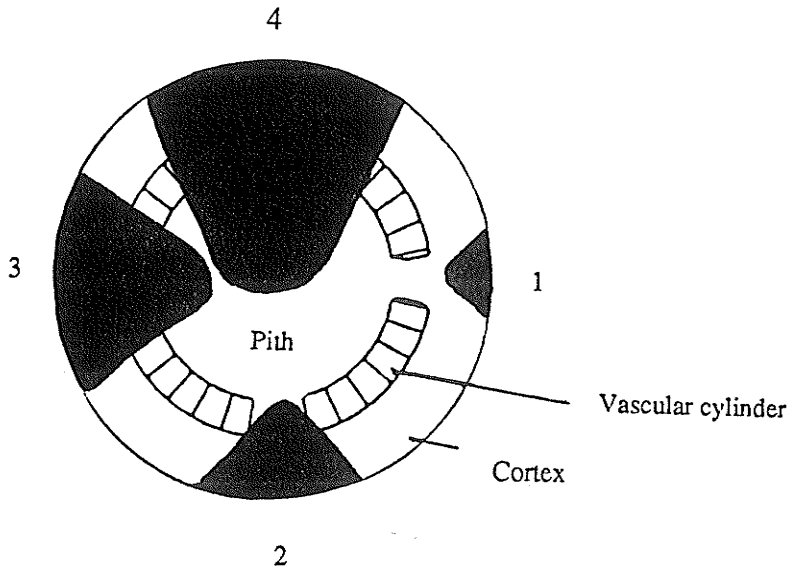


Table 4.1. Disease reactions of brassica lines challenged with isolates of Leptosphaeria maculans from different geographical origins (cotyledon test).

Isolates[1]	Lines	
	R8314.47	UM3132
MA: 2,4,9,17	R[2]	M
MA: 3,8,10,13,14,16,18,25 SA: 1,4	R	S
WA40	M	M
MA: 1,5,6,7,11 SA: 10,15	M	S
OA: 45,23	S	R
WA: 32,53,58,17	S	M
WA: 51,14	S	S

[1] Origins of isolates: MA - Souris, Manitoba
SA - Melfort, Saskatchewan
WA - Western Australia
OA - miscellaneous (France, NSW Australia)

[2] Disease reactions of lines: Resistant (R) 0.0 - 2.9
Moderately resistant (M) 3.0 - 5.9
Susceptible (S) 6.0 - 9.0

Table 4.2. Analysis of variance of isolates of Leptosphaeria maculans for variation for virulence on selected brassica lines (cotyledon and seedling test).

Source	Cotyledon test df	Cotyledon test MS	Seedling test df	Seedling test MS
Origin (O)	3	16.03	3	2.48
Isolate (I)\O	26	9.32**	26	2.21**
within Souris	16	8.68**	16	1.44**
within Melfort	3	6.40**	3	0.75
within Western Australia	6	13.62**	6	5.19**
within miscellaneous	1	2.48	1	0.90
Replicate\I*O error a	90	0.93	90	0.52
Lines (L)	1	344.95**	2	77.59**
L*O	3	190.60**	6	56.01**
I*L\O	26	75.31**	52	1.06**
Error	90	0.59	180	0.60
Total	239		359	

** significant at the 1% level (see discussion).

Figure 4.2. Disease severity of brassica lines challenged with isolates of Leptosphaeria maculans from different geographical origins (cotyledon test). Values expressed as the mean of 4 replications ± SE. (Standard errors included in mean squares.)

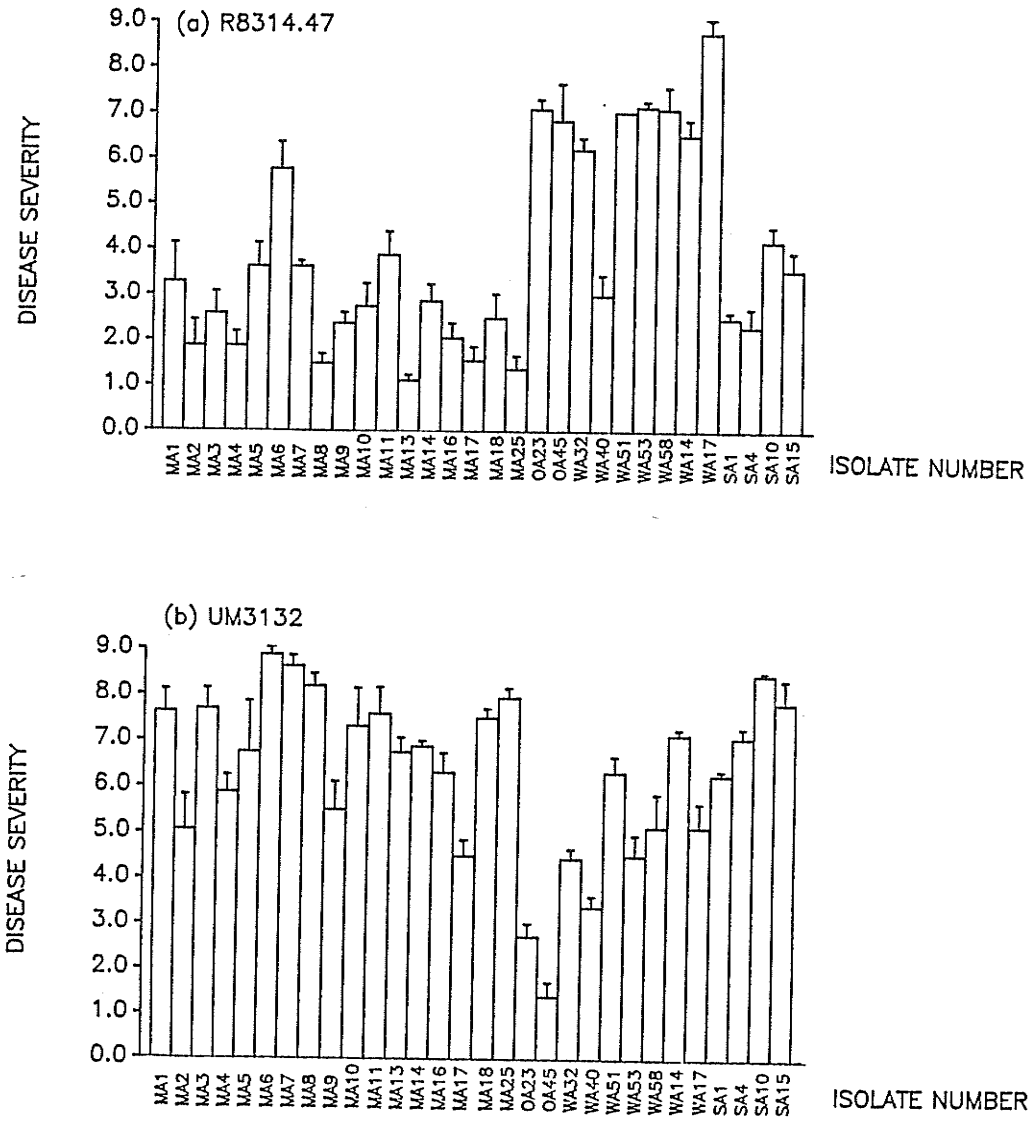


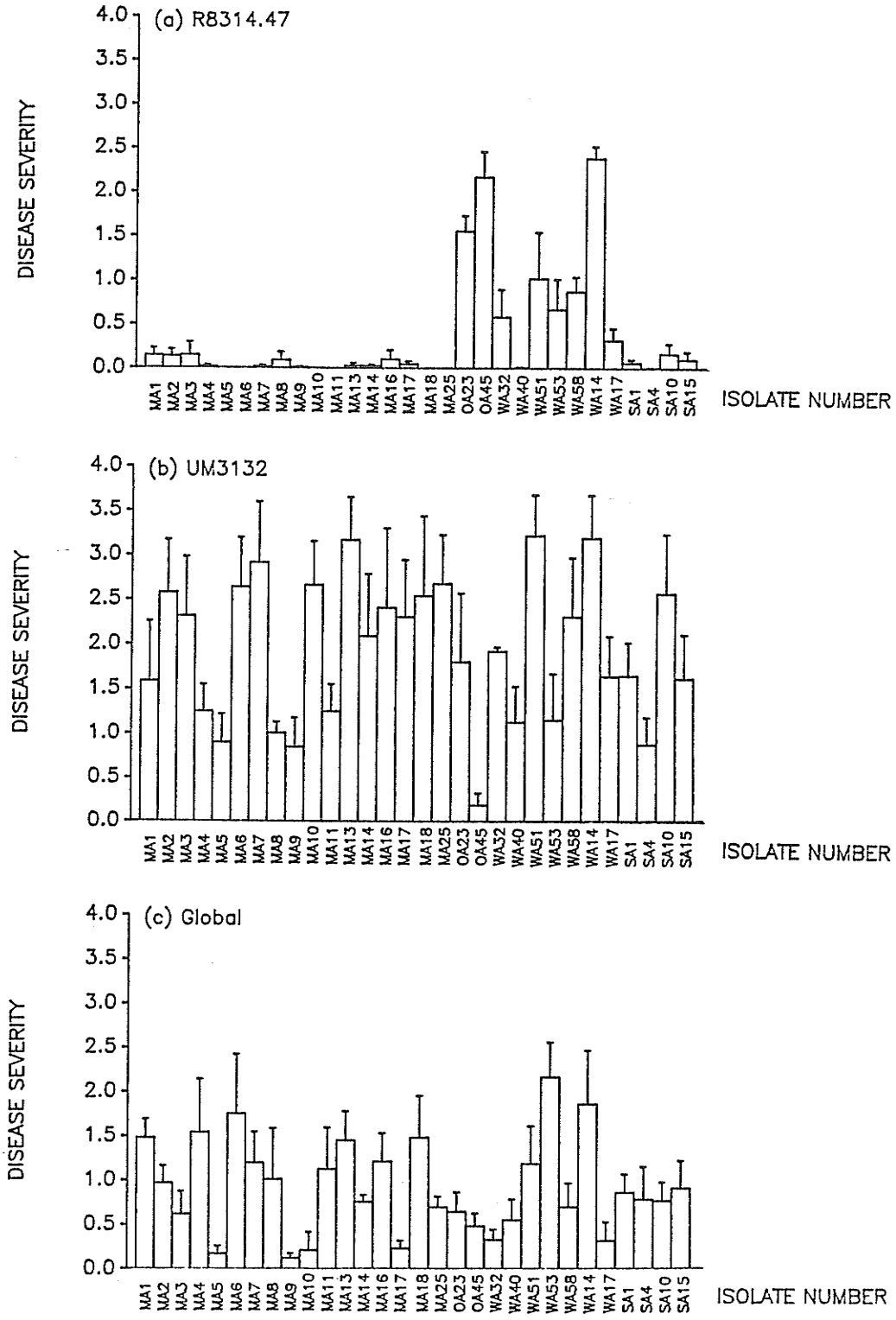
Table 4.3. Disease reactions of brassica lines challenged with isolates of Leptosphaeria maculans from different geographical origins (seedling test).

Isolates[1]	Lines		
	R8314.47	UM3132	Global
MA: 5,9	R[2]	M	R
MA: 10,17	R	S	R
MA: 4,8,11 SA4 WA40	R	M	M
MA: 1,2,3,7,13,14,16,18,25 SA: 1,10,15	R	S	M
MA6	R	S	S
WA53	M	M	S
OA23 WA: 32,51,58,17	M	S	M
OA45	S	R	M
WA14	S	S	S

[1] Origins of isolates: MA - Souris, Manitoba
SA - Melfort, Saskatchewan
WA - Western Australia
OA - miscellaneous (France, NSW Australia)

[2] Disease reactions of lines: Resistant (R) 0.0 - 0.2
Moderately resistant (M) 0.3 - 1.5
Susceptible (S) 1.6 - 4.0

Figure 4.3. Disease severity (VDT) of brassica lines challenged with isolates of Leptosphaeria maculans from different geographical origins (seedling test). Values expressed as the mean of 4 replications ± SE. (Standard errors included in mean squares.)



Yield losses in oilseed rape and oilseed turnip rape (Brassica napus and B. rapa) due to blackleg disease (Leptosphaeria maculans)

5.1 Abstract

Experiments to determine yield loss of 6 cultivars of oilseed rape were established at blackleg infested and blackleg free sites in Manitoba and Saskatchewan in 1988 and 1989. Four sites contained infested rape trash of previous years, while 3 sites were free of disease. Disease severity ratings were high on BLC198, Tobin and Westar at disease sites. Disease severity ratings were much lower on cultivars Profit, Global and Maluka. Quantification of crop loss at the various sites, for each cultivar is discussed.

5.2 Introduction

Blackleg disease of oilseed rape, causal agent Leptosphaeria maculans, has reduced oilseed rape production in Australia (Roy and Reeves 1975) and in France (Alabouvette and Brunin 1970). In western Canada the virulent form of the pathogen was first discovered in the Melfort-Star-City area of Saskatchewan in 1975 (McGee and Petrie 1978) and has since spread throughout most of the prairie provinces.

The disease has been recognized to be responsible for significant yield losses. However few studies have tried to quantify these losses. McGee and Emmett (1977) in Victoria, Australia used a survey approach to crop loss quantification and found a strong relationship between yield loss and amount of disease damage.

This paper reports on yield loss of oilseed rape cultivars under controlled experimental conditions.

5.3 Materials and Methods

Field studies were undertaken at 2 sites in 1988; Souris and Minnedosa, Manitoba which are located 150 km apart in south-western Manitoba. In 1989, 5 sites were seeded; Melfort and St. Brieux in north-east Saskatchewan, 50 km apart, Carman in southern Manitoba, and Souris and Elgin in south-western Manitoba. The latter two sites were separated by 5 km. All sites were located in the black chernozemic soil zone (Anon 1977).

Experiments at Souris, 1988 and 1989, Elgin, 1989, and Melfort, 1989, were carried out on land previously cropped to oilseed rape in which blackleg epidemics had occurred in the previous two years and earlier. Infested crop residue was present at all of these sites. Minnedosa, 1988, Carman, 1989 and St. Brieux, 1989, were chosen as check sites, and presumed disease free.

At each site a split-split plot experiment of 4 replications was conducted. The systemic fungicide flutriafol was used to try to achieve a range of disease severities between main plots. Flutriafol was applied at 0, 35, 70, 105, and 140 grams active ingredient per hectare (g ai ha^{-1}) in 1988 and at 0, 50, 100, 150, and 200 g ai ha^{-1} in 1989. Flutriafol was carried on superphosphate fertilizer granules (0-45-0) and applied in furrow with the seed, at the maximum recommended rate of actual phosphate with seed (20 kg ha^{-1}) (Anon 1988b).

Main plots were divided into 3 subplots. Each subplot contained 2 cultivars of similar maturity. Sub-subplots were seeded to a range of susceptible and resistant cultivars. This was done to facilitate harvest, and pair wise analysis of the cultivars within each maturity group. Maturity groups, cultivars and their resistance to blackleg are presented in Table 5.1.

All plots were seeded with carbofuran (Furadan CR-10) at 5.6 kg ha^{-1} in furrow with seed. St. Brieux was sprayed for cutworms twice in the seedling stage, growth stage (GS) 1 (Harper and Berkenkamp 1975), with deltamethrin (Decis 2.5 EC) at 0.20 l ha^{-1}. (All growth stages here will refer to the Harper and Berkenkamp scale.) Carman was sprayed with carbofuran at 0.2 l ha^{-1} in the rosette stage (GS 2.1), and Souris was sprayed with malathion 50 EC in the late flowering stage (GS 4.4) to control flea beetles.

Trifluralin (Treflan EC) was applied at 1.35 kg ai ha^{-1} and incorporated at Minnedosa, 1988, Carman, 1989, and St. Brieux, 1989. Edafluralin (Edge) was applied at 0.89 kg ai ha-1 at Melfort, 1988 and 1989. Fall applied trifluralin (Treflan QR5) at 1.1 kg ai ha-1 was used at Souris, 1989. In 1989 Souris, Carman, St. Brieux and Melfort were sprayed with sethoxydim (Poast) at 0.26 kg ai ha^{-1} at GS 2.1 for grassy weed control. All sites were hand weeded.

Seeding date was May 23, 1988 at both Souris and Minnedosa. In 1989 seeding dates were May 22 - St. Brieux, May 23 - Melfort, May 27 - Carman, and May 29 - Souris and

Elgin. B. rapa cultivars were seeded at 9.5 kg ha^{-1} and B. napus at 7.5 kg ha-1. Seeding was performed with a double disk drill followed by packing wheels. Seed was placed in 6 rows, 3-5 cm deep; each sub-sub-plot was 1.15 m wide and 6 m long. Guard plots of Westar surrounded the length and width of the experiment.

Disease was allowed to develop naturally throughout the growing season. At swathing time (GS 5.3), plots were rated for disease severity. On the basis of the percentage circumference of stem girdled by canker a scale was devised; 0 - no disease, 1 - <25%, 2 - 25-50%, 3 - 50-75%, 4 - 75-100%, 5 - plant dead. Two hundred plants per plot were rated at random from the 2 center rows (or 4 center rows if insufficient plants existed in the 2 center rows). Rating in both years was performed between August 1st and 15th for B. rapa cultivars depending on the site as both cultivars had a similar maturity. B. napus cultivars were rated between August 8th and 31st depending on the site and cultivar. BLC198 and Tobin, as well as Westar and Profit, matured similarly, Maluka 4-7 days later than Westar and Global 4-7 days later than Maluka.

In 1988 all six rows of each plot were cut, rated and laid in a swath. Once seed was dry each plot was threshed with a 1988 Nurserymaster elite Wintersteiger plot harvester. In 1989 plants in the 4 center rows were cut at time of rating and placed in burlap sacks to air dry until mature. Material was threshed with the Wintersteiger plot harvester.

Data was analysed by analysis of variance and the least significant difference test (LSD) at the 0.05 level of significance, using the Statistical Analysis System (SAS Institute Inc. Box 8000 Cary, North Carolina).

5.4 Results and Discussion

Data for field sites are presented in Table 5.2 and 5.3. The analysis of variance for each site is presented in Appendices 5.1-5.7. Disease levels for each site are compared by the disease severity ratings of the highly susceptible cultivar Westar. High disease

levels were recorded at Elgin, 1989 (DS 4.8), moderate disease ratings at Souris,1988 and 1989 and Melfort, 1989 (DS 2.9, 2.8 and 3.5 respectively) A low disease rating was obtained at the check sites Minnedosa, 1988 and St. Brieux, 1989 (DS 0.2 in both cases). No disease was detected at Carman, 1989.

Among replications highly significant differences for disease occurred at Souris and Minnedosa, 1988 and at Elgin, 1989, and significant differences at Souris and Melfort, 1989. There was a highly significant difference among replications for yield at Souris, 1989 and a significant difference at Minnedosa, 1988

No significant differences among flutriafol treatments were found for yield or disease severity rating at any sites in either year. In this study flutriafol was completely ineffective in controlling blackleg disease, but did not appear to have any detrimental effect on crop yield. In Saskatchewan, Xi et al. (1989) found flutriafol to be only marginally effective as a control of blackleg disease in oilseed rape. However Ballinger et al. (1988) in Australia found flutriafol to be an effective control for blackleg in oilseed rape. Differences may be explained by variation in soil types, soil pH or climatic conditions between the two countries. Xi et al. (1988), suggest there may be differences in fertilizer composition and fungicide application to the fertilizer granules. Further research to clarify these discrepancies is needed.

Highly significant differences were found among maturity groups for both disease and yield at all sites except Souris, 1988, where there was no significant difference in yield among maturity groups. This was expected as cultivars were chosen for their differences in resistance to blackleg. Yield varied with the cultivar chosen.

Drought limited yields of all cultivars at Souris in 1988, although late maturing cultivars were more strongly affected than early cultivars. Early growing season conditions were very dry which was coupled with above average temperatures lasting throughout the growing season. Severe drought conditions occurred at the Souris and

Elgin sites in 1989 beginning late June. Yields for the B. rapa cultivars were very low and B. napus cultivars were a complete loss.

BLC198, although considered moderately resistant in Australia, had a disease severity rating not significantly different from Tobin at all disease sites (Table 5.2). Yield of BLC198 was significantly higher than Tobin at all sites (disease and check) in both years, except for St. Brieux, 1989 a check site. At Minnedosa, 1988 which was a check site BLC198 outyielded Tobin but the difference was non-significant (109% as a percentage of Tobin), while under moderate disease levels at Souris, 1988, BLC198 yielded 134% of Tobin. In 1989 BLC198 yielded (as a percentage of Tobin); 106% at Souris (moderate disease level), and 118% at Elgin (high disease level). At Carman, the check site BLC198 was 111% of Tobin. At Melfort (moderate disease level) BLC198 yielded 114% of Tobin, but only 91% of Tobin at St. Brieux, a check site. Disease ratings on both cultivars are similar, however BLC198 outyields Tobin under moderate disease conditions. BLC198 appears to be somewhat tolerant of blackleg infection compared with Tobin. Yield results from the disease sites at Elgin and Souris are similar to that obtained at the check site at Carman. The data from Souris and Elgin in 1989 should be considered in light of the extreme drought conditions that prevailed. Drought may have been the more seriously limiting factor for yield than was disease at these sites.

Westar and Profit are compared, under the moderate disease intensity at Souris, 1988 and at the check site Minnedosa, 1988. The disease severity rating of Westar was 2.9 vs 1.5 for Profit, at Souris. However Westar out-yielded Profit 1032 to 746 kg ha^{-1}, or as a percentage of Maluka, 106% to 77%. At the check site Westar yielded 3070 vs 2502 kg ha^{-1} for Profit (107% vs 87% of Maluka respectively). The greater yield of Westar over Profit was not expected at Souris, since Westar was much more susceptible to blackleg infection. Better drought resistance of Westar may have been a contributing factor to its

increased yield over Profit. Westar appeared to mature marginally earlier than Profit which may also have contributed to increased seed set.

At Melfort, 1989 (disease site) disease ratings of Westar and Profit were 3.5 and 2.0 respectively. Profit outyielded Westar at this site 1665 to 583 kg ha^{-1} or 91% vs 32% of Maluka. At the check site, St. Brieux, 1989, Profit outyielded Westar 105% vs 93% of Maluka respectively. Under the moderate blackleg epidemic at Melfort, 1989, Westar yielded 61% less than at the check site (St. Brieux, 1989), while the reduction in yield was only 14% for Profit. The increased yield of Profit over Westar found at these sites illustrates the benefit of using a resistant cultivar in areas where blackleg is a threat. Due to the extreme drought in 1989 data was not available for B. napus cultivars at Elgin and Souris.

The late maturing cultivars Global and Maluka are significantly different in disease resistance, at both Souris, 1988 and Melfort, 1989. However there is no significant difference in yield between the two cultivars within either disease or check site in either year. Global, like BLC198, appears to be tolerant of L. maculans. It becomes infected and exhibits disease symptoms, however under the disease levels in this study no significant reduction in yield, in comparison to Maluka, result. Possibly Maluka could be considered an alternative cultivar for use in Canada. Its strong blackleg resistance is highly desirable, although it is later in maturity than Westar.

Table 5.1. Maturity groups and cultivars used to evaluate yield loss/disease relationships in 1988 and 1989.

Maturity group (subplots)	Cultivar (sub-subplots)	Blackleg Resistance
Early (B. rapa)	BLC198	moderate †
	Tobin	poor *
Medium (B. napus)	Westar	poor *
	Profit	fair
Late (B. napus)	Global	fair *
	Maluka	good †

* Anon (1989)
† N. Wratten (personal communication)

Table 5.2. Disease severity rating and yield of Brassica napus and B. rapa cultivars at 2 sites in 1988. Values expressed as the mean of 4 replications and 5 treatments.

Site	Year	Cultivar	Disease Severity[1]	Yield (kg/ha)
Souris	1988	BLC198	2.4	936
		Tobin	2.4	699
		Westar	2.9	1032
		Profit	1.5	746
		Global	1.1	823
		Maluka	0.4	970
		lsd (0.05)[2]	0.1	183
Minnedosa	1988	BLC198	0.4	2258
		Tobin	0.3	2072
		Westar	0.2	3070
		Profit	0.2	2502
		Global	0.2	2818
		Maluka	0.1	2872
		lsd (0.05)	0.1	238

[1] Disease severity rating on 0 (no disease) to 5 (plant dead scale).

[2] LSD (0.05) compares culitvars within each maturity group, comparisons cannot be made between maturity groups.

Table 5.3. Disease severity rating and yield of Brassica napus and B. rapa cultivars at 5 sites in 1989. Values expressed as the mean of 4 replications and 5 treatments.

Site	Year	Cultivar	Disease Severity[1]	Yield (kg/ha)
Souris	1989	BLC198	2.9	124
		Tobin	3.1	117
		Westar	2.8	15
		lsd (0.05)[2]	0.3	1
Elgin	1989	BLC198	4.3	226
		Tobin	4.4	192
		Westar	4.8	12
		lsd (0.05)	0.3	2
Carman	1989	BLC198	0.0	1316
		Tobin	0.0	1189
		Westar	0.0	1262
		lsd (0.05)	0.0	121
Melfort	1989	BLC198	3.4	1352
		Tobin	3.5	1289
		Westar	3.5	583
		Profit	2.0	1665
		Global	1.4	1896
		Maluka	0.4	1830
		lsd (0.05)	0.3	140
St. Brieux	1989	BLC198	-	1536
		Tobin	-	1692
		Westar	0.2	1718
		Profit	-	1939
		Global	-	1908
		Maluka	-	1842
		lsd (0.05)	-	143

[1] Disease severity rating on 0 (no disease) to 5 (plant dead) scale.

[2] LSD (0.05) compares culitvars within each maturity group, comparisons cannot be made between maturity groups.

GENERAL DISCUSSION

Evaluation of Brassica rapa germplasm from the University of Manitoba collection indicated no accessions were resistant to Leptosphaeria maculans in the cotyledon and adult stage. Several accessions were considered to be moderately resistant at both stages of testing. There were few accession which had a low disease severity rating in the field evaluation as well as the cotyledon or adult test. Accessions defined as moderately resistant in cotyledon and adult tests and those which had a low disease severity rating in the field might be tested for tolerance to L. maculans by determining yielding ability under disease conditions. As found in part 5 of this thesis, the B. rapa cultivars BLC198 and Tobin exhibited similar disease symptoms but varied in yield.

Variability for resistance among accessions was found when plants were evaluated at the cotyledon and adult growth stages. Winter type accessions generally had lower disease severity ratings than spring type accessions. Further examination of winter types may be useful in determining whether these accessions have resistance genes of benefit to breeders.

There was weak or no correlation between the various evaluation tests. The cotyledon and adult test measure the resistance at the beginning and end of the infection pathway respectively. Resistance at these two points may or may not be under the control of the same genes (Hammond et al. 1985). It has been suggested that a more accurate measure of resistance is obtained when the pathogen is allowed to progress as naturally as possible along the infection pathway before rating is performed (Thurling and Venn 1977, Helms and Cruickshank 1979, Sawatsky 1989). The seedling test, in which the 2nd to 4th true leaves or petioles were inoculated and crown cankers rated 6-8 weeks later, gave the best

correlation with field results in the methods tested by Sawatsky (1989). Future researchers may want to consider this method of evaluation.

Considerable variation for virulence of isolates of L. maculans was determined by the ability to attack a particular line of brassica. Significant interactions between isolates and lines were indicative of differential interactions. Two lines of brassicas at the cotyledon stage, R8314.47 and UM3132, and a third line at the adult stage, Global, differentiated most isolates. R8314.47 was resistant and/or moderately resistant at the cotyledon and adult stages respectively, to isolates from both Canadian locations but susceptible to most Western Australian isolates. Sawatsky (1989) suggested this line has two genes determining resistance to L. maculans. This line may be useful in development of a standard differential series to test isolates of L. maculans, particularly if the two genes for resistance could be separated into isogenic lines.

The fact that major genes for resistance exist within brassica species, should make it reasonably easy for plant breeders to transfer this resistance to agronomically desirable cultivars. However due to the frequency of genetic recombination, and the possibility of mutation to virulence in the pathogen, the resistance of one or a few genes for resistance may be overcome. With introduction of new cultivars carrying resistance genes, pathogen populations should be monitored for changes in virulence. Since genes for virulence to overcome the resistance of R8314 already exist in Australian populations of the pathogen, but do not appear to be present in Canadian populations, care must be taken not to introduce Australian isolates of L. maculans. Resistance depending on many genes has been suggested to be preferable (Fleming and Person 1982, Parlevliet and Zadoks 1977). Polygenic resistance may be present in Brassica species, as indicated by the disease severity ratings of the line UM3132.

Yield loss was quantified for 6 cultivars of oilseed rape. Significant reductions in yield were found for a number of cultivars. The Australian cultivar BLC198 and the

Canadian cultivar, Tobin did not differ in terms of disease severity due to L. maculans. BLC198 consistently outyielded Tobin at all sites, but by a larger margin under high L. maculans infection than under disease free conditions. This suggests BLC198 has a greater degree of tolerance to L. maculans than Tobin.

The B. napus cultivars Westar and Profit most clearly show the benefit of using a resistant cultivar where risk of blackleg is high. Profit exhibited much lower disease symptoms than Westar at all sites where disease was present. At one particular site yield of Westar was reduced from that of Profit by 59%. Later maturing B. napus cultivars Global and Maluka did not suffer any significant yield reduction due to L. maculans infection. Global did exhibit mild symptoms, while Maluka remained nearly free of crown canker.

The systemic fungicide flutriafol was not effective in protecting oilseed rape from L. maculans in this study. In Canada there has not been any economic method of chemical control of blackleg disease in infested fields. The use of cultural methods is of limited effect due to the longevity of the pathogen on infested residue and due to the long distance dispersal of the primary inoculum. Resistant cultivars appear to be the most effective and economic method of combatting blackleg disease.

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APPENDICES

Appendix 3.1. Disease reaction of cotyledons of brassica lines inoculated with Leptosphaeria maculans isolates (Delwiche 1980).

Disease reaction	Description
0	No darkening of tissue around wound. Typical response of non-inoculated cotyledon.
1	Limited blackening around wound; lesion diameter 0.5-1.5 mm. Faint chlorotic halo may be present. Sporulation absent.
3	Dark necrotic lesion, 1.5-3.0 mm. Chlorotic halo may be present. Sporulation absent.
5	Non-sporulating, 3-6 mm lesion, sharply delimited by darkened necrotic tissue. May show greyish-green tissue collapse characteristic of susceptible reactions (7&9), or dark necrosis throughout.
7	Greyish-green tissue collapse. Lesion 3-5 mm, with sharply delimited, non-darkened margins.
9	Rapid tissue collapse at about 10 days accompanied by profuse sporulation in large lesions (> than 5 mm) with diffuse, non-darkened margins.

Appendix 3.2. Disease severity ratings for accessions of Brassica rapa evaluated for resistance to one isolate of Leptosphaeria maculans (Pl86-14) in the greenhouse, and at the Souris, Manitoba blackleg nursery in 1988 and 1989. Values expressed as the mean of 6 plants ± SE in cotyledon and adult tests and as the mean of 10 plants ± SE in field tests.

[1] upper value is the mean, lower value is the standard error.

Accession Number	Coty-ledon test[1]	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1086W	4.75	6.50	6.50	6.50	8.00	.	.
	1.65	1.62	1.63	1.63	1.37	.	.
1088W	.	2.40	3.00	3.20	5.00	.	.
	.	0.40	0.77	0.80	0.63	.	.
1090W	3.50	2.80	4.00	4.60	6.00	.	.
	3.50	0.58	0.95	0.87	0.32	.	.
1092W	4.00	3.67	4.67	5.33	5.50	.	.
	3.00	2.03	1.69	1.54	1.50	.	.
1094W	5.50	0.80	2.20	3.00	4.60	.	.
	0.50	0.49	0.20	0.45	0.60	.	.
1012	8.33	5.00	6.67	8.17	8.67	.	.
	0.67	1.59	1.50	1.17	0.84	.	.
1014	7.33	7.50	8.00	9.67	9.67	1.00	1.80
	1.31	1.59	1.29	0.33	0.33	0.50	0.08
1015	9.00	10.00	10.00	10.00	10.00	0.40	1.15
	0.00	0.00	0.00	0.00	0.00	0.00	0.90
1016	8.60	10.00	10.00	10.00	10.00	1.10	.
	0.40	0.00	0.00	0.00	0.00	0.20	.
1019	8.67	5.50	7.67	9.67	10.00	1.30	.
	0.33	1.54	1.12	0.33	0.00	0.50	.
1021	9.00	4.50	6.17	8.50	9.00	0.90	1.60
	0.00	1.12	0.83	0.72	0.45	0.00	0.20
1022	8.33	4.67	4.83	5.00	6.17	1.10	.
	0.42	1.69	1.64	1.61	1.40	0.10	.
1047	9.00	10.00	10.00	10.00	10.00	1.25	.
	0.00	0.00	0.00	0.00	0.00	0.35	.

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1049	9.00	4.50	5.67	8.00	8.67	1.10	.
	0.00	1.18	0.92	0.93	0.61	0.10	.
1051	8.00	6.00	8.00	8.50	8.83	1.80	.
	1.00	1.29	0.95	0.75	1.67	0.50	.
1054	8.00	7.67	9.17	9.50	10.00	1.25	.
	0.68	1.50	0.83	0.50	0.00	0.05	.
1055	8.67	9.17	9.67	0.00	10.00	1.50	.
	0.33	0.83	0.33	0.00	0.00	0.50	.
1056	7.67	7.67	8.67	8.83	9.00	1.30	.
	0.67	1.48	0.84	0.75	0.63	0.30	.
1057	8.20	10.00	10.00	10.00	10.00	0.40	0.70
	0.49	0.00	0.00	0.00	0.00	0.20	0.10
1058	8.67	8.67	9.00	9.00	9.50	1.00	1.25
	0.33	1.33	1.00	1.00	0.50	0.20	0.15
1059	8.67	7.67	8.17	8.33	9.33	2.15	.
	0.33	1.48	1.22	1.09	0.67	0.35	.
1061	8.33	7.67	8.33	8.83	9.33	0.95	0.85
	0.67	1.48	1.05	0.75	0.67	0.05	0.35
1063	7.00	5.67	7.83	8.50	8.67	0.65	0.70
	0.89	1.67	1.11	0.72	0.61	0.15	0.10
1065	6.00	4.17	6.17	7.50	8.33	1.65	.
	1.34	1.22	1.38	0.81	0.76	0.95	.
1066	9.00	9.00	9.17	9.50	9.67	1.20	.
	0.00	1.00	0.83	0.50	0.33	0.70	.
1067	6.67	4.17	5.17	7.33	7.83	1.00	1.90
	0.80	1.19	1.05	0.88	0.79	0.60	0.10
1068	6.67	9.17	9.50	10.00	10.00	1.70	.
	0.61	0.83	0.50	0.00	0.00	0.10	.
1069	8.67	3.67	5.00	7.33	8.17	1.15	.
	0.33	1.31	1.06	0.61	0.65	0.05	.

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1070	3.33	6.00	6.33	7.00	8.67	0.60	1.05
	1.09	1.79	1.65	1.44	0.99	0.00	0.05
1071	7.00	4.17	6.83	7.83	9.17	1.55	.
	0.89	1.19	1.47	1.11	0.54	0.25	.
1072W	6.67	10.00	10.00	10.00	10.00	0.40	0.95
	1.50	0.00	0.00	0.00	0.00	0.40	0.35
1073	9.00	9.00	9.50	9.67	9.67	.	.
	0.00	1.00	0.50	0.33	0.33	.	.
1074	9.00	9.00	9.17	9.33	9.67	1.55	.
	0.00	1.00	0.83	0.67	0.33	0.25	.
1075	9.00	3.50	6.17	6.83	7.67	2.00	.
	0.00	0.22	0.83	0.65	0.56	0.30	.
1082	7.00	8.67	8.67	8.83	9.33	1.80	.
	1.37	1.33	1.33	1.17	0.67	0.20	.
1083	9.00	4.50	7.33	8.67	9.17	1.05	.
	0.00	1.15	0.71	0.61	0.54	0.15	.
1095	7.67	4.50	7.17	8.00	9.00	0.55	0.75
	0.42	1.12	0.91	0.89	0.68	0.25	0.25
1098	9.00	7.00	8.33	8.33	9.00	0.80	1.40
	0.00	1.91	1.67	1.67	1.00	0.00	0.50
1100	9.00	10.00	10.00	10.00	10.00	1.55	.
	0.00	0.00	0.00	0.00	0.00	0.25	.
1102	7.00	4.50	7.00	8.00	8.67	1.65	.
	0.73	1.17	1.32	1.03	0.84	0.35	.
1108	8.00	5.67	7.50	8.00	8.67	1.60	.
	0.68	1.50	1.12	0.89	0.61	0.40	.
1109	9.00	3.83	4.83	8.00	8.50	1.05	.
	0.00	0.40	0.31	0.68	0.50	0.05	.
1110	7.50	8.67	8.83	9.17	9.17	1.15	.
	1.50	1.33	1.17	0.83	0.83	0.55	.

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1111	9.00	3.67	6.33	8.17	9.67	0.95	1.44
	0.00	0.42	0.76	0.83	0.33	0.35	0.56
1112	8.67	7.33	9.33	9.50	9.67	2.40	.
	0.33	1.69	0.67	0.50	0.33	0.20	.
1113	8.33	5.67	6.83	7.50	8.67	2.30	.
	0.67	1.50	1.05	0.81	0.61	0.50	.
1115	9.00	8.00	8.50	8.67	8.67	0.85	1.25
	0.00	1.26	0.96	0.84	0.84	0.25	0.45
1117	9.00	8.67	8.67	8.67	9.67	0.35	0.55
	0.00	1.33	1.33	1.33	0.33	0.35	0.35
1118	7.33	2.67	4.67	5.83	8.00	1.80	.
	0.80	0.49	0.56	0.54	0.68	0.10	.
1119	9.00	5.50	6.33	8.83	10.00	1.90	.
	0.00	1.45	1.23	0.75	0.00	0.40	.
1120	7.33	4.17	5.50	7.50	7.83	1.05	.
	0.95	1.19	1.06	0.85	0.70	0.65	.
1121	9.00	2.83	9.33	8.50	10.00	1.60	.
	0.00	0.17	0.67	0.96	0.00	0.40	.
1122	8.00	6.50	8.33	8.33	8.50	1.20	.
	0.68	1.59	1.09	1.09	1.02	0.40	.
1123	9.00	4.00	6.67	7.67	8.67	2.35	.
	0.00	1.24	1.26	0.84	0.61	0.95	.
1124	6.33	4.00	5.50	6.83	7.67	1.85	.
	1.23	0.89	0.72	0.79	0.61	0.85	.
1125	9.00	5.17	8.00	10.00	10.00	2.05	.
	0.00	1.25	1.00	0.00	0.00	0.05	.
1127	9.00	5.17	8.00	8.50	10.00	1.60	.
	0.00	1.56	1.29	0.96	0.00	0.50	.
1129	9.00	7.83	9.17	9.67	10.00	1.35	.
	0.00	0.98	0.54	0.33	0.00	0.05	.

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1130	7.00	2.67	4.17	5.83	6.17	2.25	.
	0.52	0.33	0.79	0.87	0.70	0.25	.
1131	9.00	6.17	8.00	9.00	9.67	2.50	.
	0.00	0.83	0.89	0.68	0.33	0.00	.
1132	8.67	0.00	10.00	10.00	10.00	1.45	.
	0.33	0.00	0.00	0.00	0.00	0.35	.
1133	7.33	5.50	7.17	8.83	10.00	2.55	.
	0.61	1.45	0.95	0.75	0.00	0.15	.
1134	9.00	8.67	8.67	8.67	8.83	1.40	.
	0.00	1.33	1.33	1.33	1.17	0.80	.
1135	8.67	4.50	7.17	9.67	9.67	1.40	.
	0.33	0.67	1.01	0.33	0.33	0.40	.
1136	7.33	6.17	10.00	10.00	10.00	1.45	.
	1.31	1.80	0.00	0.00	0.00	0.05	.
1137	9.00	7.50	8.33	8.50	9.67	0.95	2.05
	0.00	1.31	1.05	0.96	0.33	0.15	0.15
1138	9.00	8.67	8.83	9.33	9.50	0.85	1.45
	0.00	1.33	1.17	0.67	0.50	0.45	0.45
1139	9.00	4.17	6.67	8.50	10.00	1.40	.
	0.00	1.22	0.76	0.72	0.00	0.50	.
1140	9.00	5.00	4.83	5.33	6.50	1.35	.
	0.00	2.24	1.64	1.61	1.28	0.15	.
1141	9.00	3.50	4.67	6.67	7.17	0.65	0.02
	0.00	1.31	1.15	1.15	0.91	0.15	0.25
1143	9.00	7.83	8.83	10.00	10.00	1.25	.
	0.00	1.38	1.17	0.00	0.00	0.35	.
1144	9.00	3.50	5.00	6.67	7.17	1.65	.
	0.00	0.50	0.58	0.42	0.31	0.25	.
1145	7.67	3.83	6.50	7.67	9.00	0.50	1.25
	0.67	0.17	0.89	0.56	0.63	0.20	0.15

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1146	9.00	4.67	6.17	7.33	8.50	0.90	1.80
	0.00	1.09	0.83	0.56	0.67	0.40	0.00
1147	8.67	4.83	8.00	8.33	8.83	1.35	.
	0.33	1.19	0.63	0.56	0.54	0.15	.
1148	9.00	6.67	7.83	9.33	9.33	1.55	.
	0.00	2.11	1.38	0.67	0.67	0.05	.
1149	8.67	6.00	6.67	8.00	8.67	1.30	.
	0.33	1.26	1.12	0.63	0.61	0.00	.
1150	8.00	4.17	5.83	7.17	7.50	1.50	.
	0.68	1.90	1.35	0.91	0.81	0.30	.
1151	9.00	5.50	7.33	8.67	9.33	1.30	.
	0.00	1.48	1.20	0.67	0.67	0.40	.
1153	8.67	5.17	6.83	7.83	8.83	0.45	1.55
	0.33	0.98	1.05	0.70	0.54	0.35	0.35
1155	9.00	4.83	6.33	7.33	8.17	0.65	0.80
	0.00	1.05	1.17	0.95	0.65	0.05	0.30
1157	8.67	4.50	6.83	9.17	9.67	0.95	1.05
	0.33	0.72	1.11	0.54	0.33	0.25	0.55
1158	7.00	4.00	5.17	6.17	7.00	1.10	.
	0.73	1.44	1.51	1.08	0.82	0.30	.
1159	8.67	4.33	6.00	8.50	8.67	1.35	.
	0.33	1.38	1.34	1.02	0.88	0.55	.
1160	8.33	6.17	7.17	8.50	8.83	0.95	1.15
	0.42	1.72	1.33	0.96	0.75	0.65	0.35
1162	8.67	10.00	10.0	10.00	10.00	1.10	.
	0.33	0.00	0.00	0.00	0.00	0.50	.
1165	8.67	6.00	7.67	8.83	9.33	1.90	.
	0.33	1.26	0.80	0.54	0.42	0.40	.
1168	8.00	6.50	7.83	9.00	9.33	0.95	1.30
	0.45	1.57	0.98	0.63	0.42	0.45	0.10

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1169	9.00	5.67	10.00	10.00	10.00	2.15	.
	0.00	1.43	0.00	0.00	0.00	0.15	.
1170	9.00	10.00	10.00	10.00	10.00	2.40	.
	0.00	0.00	0.00	0.00	0.00	0.80	.
1171	9.00	6.50	7.33	8.83	9.00	1.40	.
	0.00	1.59	1.23	0.75	0.63	0.90	.
1172	7.00	6.17	7.17	8.17	8.17	0.85	1.25
	0.82	1.28	0.98	0.65	0.65	0.45	0.05
1173	9.00	6.83	7.67	8.33	8.50	0.75	1.05
	0.00	1.47	1.05	0.76	0.72	0.25	0.55
1177	9.00	9.33	9.50	9.50	9.67	1.00	0.80
	0.00	0.67	0.50	0.50	0.33	0.30	0.60
1178	8.33	7.50	8.33	9.00	9.67	0.70	1.20
	0.42	1.59	1.09	0.63	0.33	0.40	0.60
1179	8.33	5.17	7.50	9.67	9.67	0.55	1.10
	0.67	1.17	0.56	0.33	0.33	0.25	0.60
1180	8.67	6.50	7.50	8.00	8.33	0.85	2.05
	0.33	1.57	1.15	0.89	0.76	0.35	1.05
1181	8.33	5.33	7.50	8.00	8.50	0.85	1.55
	0.67	1.48	0.96	0.93	0.72	0.85	0.55
1182	8.33	6.67	7.67	8.83	8.83	0.90	1.60
	0.42	1.54	1.17	0.75	0.75	0.00	0.90
1183	9.00	10.00	10.00	10.00	10.00	1.10	.
	0.00	0.00	0.00	0.00	0.00	0.10	.
1184	9.00	8.60	8.80	9.20	9.40	1.00	1.10
	0.00	1.40	1.20	0.80	0.60	0.40	0.90
1186	7.67	8.33	9.33	9.50	9.50	0.75	1.10
	0.99	1.67	0.67	0.50	0.50	0.55	0.40
1187	6.00	9.00	9.17	9.33	10.00	1.60	.
	1.13	1.00	0.83	0.67	0.00	0.20	.

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1188	8.60	6.67	8.17	8.50	9.67	0.80	1.35
	0.42	1.52	0.83	0.67	0.33	0.20	0.05
1189	7.50	8.00	8.67	8.83	9.00	0.70	1.30
	1.50	1.29	0.84	0.75	0.63	0.00	0.30
1190	7.17	8.67	9.00	9.33	9.33	1.95	.
	1.47	1.33	1.00	0.67	0.67	0.15	.
1191	8.67	5.83	7.67	7.83	9.67	.	.
	0.33	1.40	0.95	0.83	0.33	.	.
1192	7.00	4.83	8.00	8.67	9.17	0.95	0.70
	0.52	1.17	0.93	0.88	0.54	0.35	0.30
1193	7.67	7.50	8.33	9.17	9.67	0.95	1.15
	0.84	1.59	1.05	0.54	0.33	0.05	0.45
1194	8.33	6.67	8.50	8.67	8.83	1.05	.
	0.42	1.50	0.96	0.84	0.75	0.15	.
1196	8.67	9.00	9.50	9.50	9.50	1.45	.
	0.33	1.00	0.50	0.50	1.12	0.45	.
1197	8.67	5.17	6.33	7.50	8.83	0.95	0.65
	0.33	1.56	1.28	1.20	0.75	0.15	0.15
1198	8.00	7.17	7.83	9.00	9.33	1.10	.
	0.68	1.30	1.01	0.63	0.42	0.20	.
1199	8.67	9.50	10.00	10.00	10.00	1.25	.
	0.33	0.50	0.00	0.00	0.00	0.15	.
1200	5.50	6.00	7.83	8.67	9.67	0.85	1.60
	1.71	1.34	0.98	0.84	0.33	0.45	0.20
1202	6.83	7.00	7.50	8.33	8.50	1.40	.
	1.42	1.44	1.23	0.76	0.67	0.30	.
1203	9.00	3.40	7.20	8.00	9.60	1.10	.
	0.00	0.75	0.73	0.63	0.40	0.20	.
1204	9.00	5.83	6.83	8.00	9.50	0.60	0.95
	0.00	0.95	0.70	0.93	0.50	0.30	0.25

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1205	9.00	5.50	9.00	9.50	9.67	0.90	2.00
	0.00	1.43	0.68	0.50	0.33	0.30	0.60
1206	8.00	7.83	8.33	8.50	8.67	0.95	0.25
	0.68	1.38	1.09	1.02	0.99	0.35	0.15
1207	8.33	7.00	8.00	8.83	10.00	1.50	.
	0.42	1.37	1.00	0.75	0.00	0.40	.
1208	7.67	6.83	9.50	9.50	10.00	0.80	2.15
	1.33	1.47	0.50	0.50	0.00	0.20	0.55
1215	8.67	6.17	10.00	10.00	10.00	2.00	.
	0.33	1.72	0.00	0.00	0.00	0.10	.
1217	8.67	7.17	7.67	8.33	8.50	1.80	.
	0.33	1.38	1.17	1.17	1.15	0.10	.
1220	8.67	5.67	6.67	10.00	10.00	1.45	.
	0.33	1.48	1.20	0.00	0.00	0.35	.
1222	8.67	7.50	8.33	9.67	10.00	2.10	.
	0.33	1.71	1.17	0.33	0.00	0.00	.
1224	9.00	7.67	8.33	9.50	9.50	0.85	1.10
	0.00	1.20	1.09	0.50	0.50	0.25	0.40
1225	4.83	1.67	5.33	7.50	8.17	1.25	.
	1.47	0.84	0.71	0.56	0.60	0.25	.
1226	3.67	4.50	6.33	8.17	8.50	0.70	0.50
	1.58	1.20	0.95	0.87	0.81	0.10	0.20
1227	8.67	6.67	7.50	8.17	9.00	0.85	1.40
	0.33	1.52	1.31	0.83	0.63	0.05	0.10
1228	8.67	5.17	7.17	9.67	10.00	.	.
	0.33	1.56	0.98	0.33	0.00	.	.
1229	9.00	6.67	6.83	8.00	9.17	1.10	.
	0.00	1.50	1.45	0.93	0.54	0.40	.
1230	8.33	7.00	8.00	9.17	9.33	1.30	.
	0.67	1.91	1.37	0.83	0.67	0.30	.

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1231	9.00	6.50	8.00	9.33	9.50	1.45	.
	0.00	1.59	1.26	0.67	0.50	0.25	.
1232	7.67	8.67	9.33	10.00	10.00	1.05	.
	1.33	1.33	0.67	0.00	0.00	0.05	.
1233	8.67	6.83	8.67	9.33	10.00	0.95	1.20
	0.33	1.47	0.88	0.42	0.00	0.15	0.60
1234	7.00	5.17	6.83	8.50	9.17	1.60	.
	1.03	1.68	1.14	0.72	0.54	0.30	.
1235	7.67	4.00	6.17	8.67	8.83	1.70	.
	0.99	1.24	1.28	0.61	0.54	1.00	.
1236	6.50	4.33	5.83	6.83	8.17	0.50	1.05
	1.45	1.15	0.87	0.75	0.65	0.10	0.55
1238	7.33	6.50	7.33	9.67	10.00	0.80	1.00
	0.95	1.63	1.23	0.33	0.00	0.10	0.30
1239	9.00	10.00	10.00	10.00	10.00	1.25	.
	0.00	0.00	0.00	0.00	0.00	0.65	.
1240	9.00	10.00	10.00	10.00	10.00	1.00	0.90
	0.00	0.00	0.00	0.00	0.00	0.50	0.20
1245	7.50	10.00	10.00	10.00	10.00	1.20	.
	1.50	0.00	0.00	0.00	0.00	0.40	.
1246	7.50	6.00	6.67	8.17	8.50	0.70	1.40
	1.50	1.84	1.52	1.17	0.96	0.50	0.80
1247	8.67	6.33	8.33	8.83	9.67	1.85	.
	0.33	1.65	1.09	0.75	0.33	0.05	.
1248	8.00	7.17	7.83	0.00	10.00	0.45	1.35
	1.00	1.83	1.38	0.00	0.00	0.45	0.05
1249	8.00	8.83	8.83	0.00	10.00	1.05	.
	1.00	1.17	1.17	0.00	0.00	0.05	.
1250	8.67	7.67	9.67	10.00	10.00	1.30	.
	0.33	1.50	0.33	0.00	0.00	0.30	.

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1251	9.00	7.50	9.00	9.33	10.00	1.90	.
	0.00	1.59	1.00	0.67	0.00	0.40	.
1252	8.00	7.50	8.00	8.50	10.00	1.50	.
	1.00	1.59	1.29	1.02	0.00	0.50	.
1253	8.67	6.33	7.67	8.00	8.83	1.20	.
	0.33	1.67	1.09	0.93	0.75	0.70	.
1254	8.67	6.17	7.83	9.17	9.33	0.70	2.00
	0.33	1.72	1.38	0.83	0.67	0.10	0.20
1257	9.00	10.00	10.00	10.00	10.00	0.55	1.10
	0.00	0.00	0.00	0.00	0.00	0.15	0.30
1258	9.00	8.67	8.67	8.67	9.17	0.35	.
	0.00	1.33	1.33	1.33	0.83	0.05	.
1259	9.00	3.33	5.17	8.00	8.67	1.10	.
	0.00	1.33	1.05	1.03	0.99	0.50	.
1260	8.00	3.83	9.00	9.17	9.33	2.10	.
	0.68	1.28	1.00	0.83	0.67	0.30	.
1261	9.00	4.83	7.33	8.67	9.33	1.40	.
	0.00	1.64	1.26	0.88	0.67	0.50	.
1262	8.67	4.67	8.33	8.83	9.67	1.05	.
	0.33	1.69	1.09	0.75	0.33	0.15	.
1264	8.67	3.67	7.33	9.67	8.67	1.50	.
	0.33	1.36	1.20	0.33	0.61	0.10	.
1265	8.00	5.17	7.50	9.17	10.00	1.35	.
	1.00	1.56	1.23	0.54	0.00	0.45	.
1267	9.00	4.00	7.00	8.00	8.83	1.90	.
	0.00	1.29	1.03	0.68	0.54	0.90	.
1270	9.00	4.83	6.67	7.33	8.00	2.10	.
	0.00	1.64	1.54	1.23	0.93	0.00	.
1271	9.00	8.67	8.67	9.17	9.50	0.95	1.70
	0.00	1.33	1.33	0.83	0.50	0.35	0.10

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1274	9.00	5.60	6.80	7.20	8.20	1.15	.
	0.00	1.81	1.32	1.16	1.11	0.35	.
1287	9.00	7.33	10.00	10.00	10.00	0.25	0.65
	0.00	1.69	0.00	0.00	0.00	0.05	0.45
1291	9.00	6.67	10.00	10.00	10.00	1.55	.
	0.00	2.11	0.00	0.00	0.00	0.35	.
1292	8.00	4.50	7.50	8.00	9.33	1.00	1.00
	1.00	1.78	1.59	1.29	0.67	0.10	0.30
1293	9.00	7.33	7.83	9.17	10.00	1.95	.
	0.00	1.69	1.38	0.83	0.00	0.75	.
1294	7.50	6.00	7.50	9.67	10.00	1.90	.
	1.50	1.86	1.23	0.33	0.00	0.70	.
1295	9.00	7.50	7.83	9.50	9.67	1.30	.
	0.00	1.59	1.38	0.50	0.33	0.50	.
1297	9.00	6.17	8.33	10.00	10.00	0.65	1.15
	0.00	1.72	1.09	0.00	0.00	0.15	0.25
1298	9.00	10.00	10.00	10.00	10.00	1.75	.
	0.00	0.00	0.00	0.00	0.00	0.55	.
1301	9.00	4.67	7.50	9.17	9.33	1.10	.
	0.00	1.69	1.20	0.54	0.42	0.70	.
1302	9.00	6.00	7.67	8.50	9.50	1.25	.
	0.00	1.79	1.48	0.96	0.50	0.05	.
1304	9.00	6.17	8.00	8.83	9.17	2.15	.
	0.00	1.72	1.29	0.75	0.54	0.55	.
1308	9.00	10.00	10.00	10.00	10.00	1.70	.
	0.00	0.00	0.00	0.00	0.00	0.20	.
1309	9.00	10.00	10.00	10.00	10.00	3.25	.
	0.00	0.00	0.00	0.00	0.00	0.05	.
1312	9.00	4.33	5.67	8.33	0.00	.	.
	0.00	1.82	1.48	1.05	0.00	.	.

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1313	9.00	7.67	9.33	9.50	9.50	3.25	.
	0.00	1.48	0.67	0.50	0.50	0.35	.
1314	8.33	2.33	6.00	6.83	7.33	1.75	.
	0.67	0.21	1.13	1.01	1.09	0.05	.
1317	9.00	3.33	7.83	9.67	9.67	0.75	1.65
	0.00	1.33	1.28	0.33	0.33	0.15	0.15
1319	9.00	5.17	8.83	8.83	9.00	1.30	.
	0.00	1.56	0.75	0.75	0.63	0.30	.
1320	9.00	6.17	8.17	8.33	9.00	1.95	.
	0.00	1.72	0.83	0.76	0.63	0.15	.
1321	9.00	2.17	9.17	9.33	9.67	0.90	1.60
	0.00	0.17	0.83	0.67	0.33	0.20	0.90
1322	8.33	3.83	6.00	8.17	9.00	1.25	.
	0.67	1.28	0.82	0.83	0.63	0.05	.
1324	9.00	6.00	7.67	8.50	10.00	2.05	.
	0.00	1.79	1.17	1.02	0.00	0.45	.
1326	9.00	4.17	10.00	10.00	10.00	2.40	.
	0.00	1.90	0.00	0.00	0.00	0.20	.
1327	9.00	5.00	8.17	9.67	10.00	2.10	.
	0.00	1.61	1.11	0.33	0.00	0.20	.
1328	9.00	3.67	6.17	8.67	9.17	0.85	0.50
	0.00	1.31	1.30	0.84	0.54	0.05	0.10
1329	9.00	4.83	8.17	8.33	9.50	1.55	.
	0.00	1.64	1.17	1.05	0.50	0.75	.
1330	7.67	3.00	5.67	6.17	8.67	2.05	.
	1.33	1.44	1.41	1.28	0.84	0.15	.
1331	9.00	6.17	9.33	9.67	10.00	1.45	.
	0.00	1.72	0.67	0.33	0.00	0.65	.
1332	9.00	3.67	6.00	7.83	8.83	1.90	.
	0.00	1.28	1.13	0.70	0.54	0.70	.

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1333	8.33	3.83	5.33	5.67	7.83	1.60	.
	0.67	1.28	1.20	1.12	0.75	0.30	.
1334	9.00	6.33	7.50	9.33	9.50	1.55	.
	0.00	1.65	1.12	0.67	0.50	0.25	.
1335	9.00	5.20	5.80	6.40	9.20	2.40	.
	0.00	1.96	1.71	1.57	0.80	0.50	.
1336	9.00	6.00	7.00	10.00	10.00	1.10	.
	0.00	1.79	1.37	0.00	0.00	0.10	.
1337	8.00	1.33	3.67	6.00	7.50	1.35	.
	0.68	0.42	0.95	1.53	1.36	0.05	.
1338	9.00	4.67	6.50	7.67	8.50	0.85	1.55
	0.00	1.69	1.18	1.23	0.72	0.05	0.45
1343	9.00	4.83	7.50	7.83	9.17	1.90	.
	0.00	1.64	1.20	1.14	0.83	0.50	.
1346	9.00	2.17	7.50	8.17	8.83	1.10	.
	0.00	0.48	0.85	0.83	0.75	0.10	.
1351	9.00	4.83	6.67	8.83	9.33	0.85	1.80
	0.00	1.72	1.09	0.75	0.67	0.35	0.10
1381	9.00	8.00	9.20	9.20	9.20	1.85	.
	0.00	2.00	0.80	0.80	0.80	0.15	.
1382	9.00	6.00	7.33	8.67	9.33	1.10	.
	0.00	1.79	1.33	0.84	0.67	0.00	.
1383	9.00	7.00	9.33	10.00	10.00	1.35	.
	0.00	1.91	0.67	0.00	0.00	0.25	.
1384	9.00	5.67	9.17	9.33	9.50	1.90	.
	0.00	1.96	0.83	0.67	0.50	0.10	.
1385	9.00	6.17	9.50	9.50	10.00	1.05	.
	0.00	1.72	0.50	0.50	0.00	0.45	.
1386	9.00	1.67	10.00	10.00	10.00	0.60	1.20
	0.00	1.67	0.00	0.00	0.00	0.10	0.20

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1396	9.00	2.83	6.83	9.33	9.50	1.25	.
	0.00	1.51	1.14	0.67	0.50	0.35	.
1397	9.00	3.33	4.83	8.33	8.67	2.25	.
	0.00	1.33	1.14	1.09	0.99	0.95	.
1398	8.00	6.00	7.67	9.00	9.17	1.10	.
	0.45	1.79	1.12	0.68	0.54	0.00	.
1399	9.00	7.50	8.83	9.33	10.00	1.15	.
	0.00	1.59	0.75	0.42	0.00	0.15	.
1400	8.67	6.33	7.67	8.17	9.50	1.75	.
	0.33	1.65	1.05	0.83	0.50	0.95	.
1401	8.33	7.67	9.33	9.50	9.50	1.80	.
	0.67	1.48	0.67	0.50	0.50	0.10	.
1402	9.00	5.17	7.50	8.00	8.83	1.55	.
	0.00	1.54	0.81	0.68	0.54	0.05	.
1404	9.00	6.00	8.17	9.50	9.67	2.20	.
	0.00	1.37	0.83	0.50	0.33	0.60	.
1405	9.00	7.67	8.50	9.33	9.33	1.45	.
	0.00	1.48	0.96	0.67	0.67	0.35	.
1417	9.00	5.00	8.00	8.33	8.67	1.65	.
	0.00	1.59	1.00	0.76	0.67	0.65	.
1418	9.00	9.00	9.67	9.67	9.67	1.05	.
	0.00	1.00	0.33	0.33	0.33	0.05	.
1421	9.00	3.17	10.00	0.00	10.00	1.95	.
	0.00	1.42	0.00	0.00	0.00	0.85	.
1425	9.00	3.67	7.17	9.00	9.17	2.40	.
	0.00	1.28	1.30	0.68	0.54	0.00	.
1426	9.00	8.83	10.00	10.00	10.00	1.30	.
	0.00	1.17	0.00	0.00	0.00	0.40	.
1427	9.00	4.67	9.33	9.50	9.50	1.45	.
	0.00	1.69	0.67	0.50	0.50	0.45	.

Appendix 3.2. (continued)

Accession Number	Coty-ledon test	Adult test Week 1	Week 2	Week 3	Week 4	Field tests 1988	1989
1428	9.00	3.33	7.83	9.50	9.67	0.85	2.40
	0.00	0.71	0.91	0.50	0.33	0.25	0.20
1429	9.00	7.17	8.67	8.83	9.00	1.15	.
	0.00	1.83	0.88	0.75	0.63	0.65	.
1430	9.00	3.67	7.50	9.33	10.00	1.85	.
	0.00	1.28	0.85	0.67	0.00	0.65	.
1432	8.33	6.17	9.17	9.33	9.50	1.45	.
	0.67	1.72	0.83	0.67	0.50	0.25	.
1440	9.00	7.50	7.83	9.00	10.00	0.75	0.50
	0.00	1.59	1.38	1.00	0.00	0.05	0.30
1446	7.00	8.67	10.00	10.00	10.00	1.50	.
	1.37	1.33	0.00	0.00	0.00	0.40	.
1449	9.00	10.00	10.00	10.00	10.00	0.90	0.10
	0.00	0.00	0.00	0.00	0.00	0.40	0.15
1465	9.00	8.67	9.67	10.00	10.00	1.25	.
	0.00	1.33	0.33	0.00	0.00	0.55	.
1472	4.00	2.50	5.50	7.00	7.83	0.75	1.50
	1.13	0.34	1.06	1.13	0.79	0.35	0.50
Tobin	8.17	3.00	5.83	7.67	9.33	1.42	1.60
	0.40	1.44	1.33	0.80	0.67	0.06	0.08
Westar	8.59	5.43	7.28	8.10	8.61	.	.
	0.14	0.26	0.20	0.16	0.13	.	.

Appendix 4.1. Disease severity means and standard error of each isolate-line combination (cotyledon test). Values are the mean of 4 replications ± SE.

Isolate[1]	Lines[2]											
	1	2	3	4	5	6	7	8	9	10	11	12
MA1	2.31[3]	3.28	8.19	8.31	6.94	8.56	9.00	8.94	8.06	8.06	0.00	7.63
	0.56	0.85	0.49	0.28	0.21	0.30	0.00	0.06	0.70	0.48	0.00	0.48
MA2	1.19	1.88	7.69	8.44	6.38	7.44	8.88	9.00	7.06	8.19	0.00	5.06
	0.06	0.56	0.39	0.26	0.39	0.36	0.13	0.00	0.28	0.19	0.00	0.75
MA3	2.15	2.58	7.88	8.88	7.00	7.75	8.81	9.00	8.15	7.81	0.25	7.69
	0.24	0.49	0.26	0.13	0.00	0.20	0.12	0.00	0.39	0.45	0.25	0.45
MA4	1.00	1.88	7.00	8.25	6.13	7.13	7.75	7.75	6.75	7.25	0.00	5.88
	0.18	0.31	0.35	0.32	0.52	0.13	0.48	0.48	0.83	0.32	0.00	0.38
MA5	1.31	3.63	8.50	8.38	8.88	9.00	9.00	9.00	5.56	6.50	0.00	6.75
	0.62	0.52	0.29	0.38	0.13	0.00	0.00	0.00	1.02	0.74	0.00	1.11
MA6	2.13	5.75	8.25	8.13	7.29	8.50	9.00	8.88	7.00	7.38	0.00	8.88
	0.31	0.60	0.32	0.43	0.41	0.20	0.00	0.13	1.08	0.80	0.00	0.13
MA7	2.63	3.63	7.75	8.50	7.00	8.63	8.88	9.00	5.50	6.13	0.00	8.63
	0.38	0.13	0.43	0.29	0.00	0.38	0.13	0.00	0.54	0.83	0.00	0.24
MA8	0.85	1.49	8.06	8.65	7.75	8.38	9.00	9.00	7.25	8.16	0.00	8.19
	0.09	0.21	0.31	0.15	0.27	0.39	0.00	0.00	0.60	0.36	0.00	0.28
MA9	0.94	2.38	6.13	7.00	6.50	7.00	7.50	6.75	5.75	4.69	0.00	5.50
	0.06	0.24	0.31	0.20	0.20	0.20	0.20	0.43	0.32	0.80	0.00	0.61
MA10	1.28	2.75	7.44	8.00	7.63	7.50	8.50	8.06	5.94	6.94	0.00	7.31
	0.38	0.51	0.75	1.00	0.47	0.61	0.50	0.48	1.39	0.36	0.00	0.83

Appendix 4.1. (continued)

Isolate	Lines											
	1	2	3	4	5	6	7	8	9	10	11	12
MA11	2.63	3.88	7.63	7.75	7.50	7.63	8.63	8.25	8.42	7.25	0.00	7.58
	1.46	0.52	0.47	0.48	0.50	0.47	0.24	0.48	0.34	0.43	0.00	0.58
MA13	1.00	1.13	6.75	7.50	6.00	8.50	8.25	8.50	6.00	7.13	0.00	6.75
	0.18	0.13	0.32	0.29	0.35	0.35	0.32	0.20	0.89	0.31	0.00	0.32
MA14	1.75	2.88	6.13	8.50	6.88	7.25	8.75	8.88	5.75	6.25	0.00	6.88
	0.25	0.38	0.24	0.35	0.13	0.14	0.14	0.13	0.25	0.72	0.00	0.13
MA16	1.31	2.06	7.13	8.31	6.94	8.23	8.88	9.00	5.25	6.44	0.00	6.31
	0.12	0.33	0.56	0.45	0.06	0.30	0.13	0.00	0.43	0.40	0.00	0.43
MA17	1.30	1.56	7.13	7.00	0.81	7.06	8.38	7.88	0.48	7.13	0.00	4.50
	0.18	0.31	0.07	0.53	0.28	0.06	0.16	0.26	1.04	0.60	0.00	0.35
MA18	1.38	2.50	7.75	7.13	7.13	7.50	8.00	8.00	6.79	7.21	0.00	7.50
	0.38	0.54	0.43	0.13	0.13	0.20	0.00	0.20	0.39	0.64	0.00	0.20
MA25	0.78	1.38	7.71	7.75	7.38	8.73	9.00	8.75	6.93	7.08	0.00	7.94
	0.33	0.29	0.27	0.27	0.22	0.10	0.00	0.25	0.60	0.33	0.00	0.21
OA23	6.31	7.06	6.88	5.35	6.94	5.31	6.81	6.00	6.56	6.00	0.00	2.72
	0.61	0.21	0.30	0.84	0.06	1.61	0.37	0.84	0.49	0.53	0.00	0.29
OA45	3.35	6.81	4.63	4.69	3.19	6.07	6.00	6.94	1.91	2.31	0.00	1.40
	0.73	0.80	0.43	0.57	0.62	0.29	0.37	0.12	0.55	0.83	0.00	0.32
WA32	5.44	6.19	6.94	6.50	6.25	6.81	7.19	6.81	6.00	6.88	0.00	4.44
	0.54	0.26	0.16	0.40	0.32	0.12	0.21	0.45	0.50	0.47	0.00	0.21

Appendix 4.1. (continued)

Isolate	Lines											
	1	2	3	4	5	6	7	8	9	10	11	12
WA40	1.13	3.00	7.13	6.75	6.88	7.00	7.00	7.00	4.38	5.50	0.00	3.38
	0.31	0.46	0.13	0.14	0.13	0.00	0.00	0.00	0.72	0.20	0.00	0.24
WA51	7.00	7.00	7.06	6.94	7.00	7.00	7.00	7.00	6.81	6.56	0.00	6.31
	0.00	0.00	0.06	0.06	0.00	0.00	0.00	0.00	0.12	0.26	0.00	0.34
WA53	6.75	7.13	7.00	6.00	7.00	7.00	6.44	6.50	7.00	5.63	0.00	4.50
	0.25	0.13	0.00	0.71	0.00	0.00	0.41	0.29	0.00	0.38	0.00	0.46
WA58	6.88	7.08	7.13	5.75	7.00	7.00	6.92	7.04	6.29	5.75	0.00	5.13
	0.13	0.48	0.13	0.43	0.00	0.20	0.34	0.24	0.41	0.43	0.00	0.72
WA14	5.38	6.51	6.88	6.50	5.88	7.13	7.00	6.88	3.38	6.63	0.00	7.13
	0.13	0.34	0.13	0.35	0.38	0.13	0.00	0.13	0.52	0.24	0.00	0.13
WA17	8.38	8.75	9.00	8.13	8.88	9.00	9.00	8.88	7.13	7.75	0.00	5.13
	0.38	0.25	0.00	0.52	0.13	0.00	0.00	0.13	0.52	0.43	0.00	0.52
SA1	1.13	2.50	8.88	8.63	7.75	8.13	9.00	9.00	7.94	8.31	0.00	6.25
	0.07	0.14	0.13	0.13	0.32	0.36	0.00	0.00	0.50	0.31	0.00	0.10
SA4	1.13	2.31	7.65	8.69	7.25	7.73	9.00	9.00	7.25	7.44	0.00	7.06
	0.07	0.41	0.34	0.19	0.37	0.31	0.00	0.00	0.42	0.84	0.00	0.21
SA10	3.50	4.19	8.56	8.56	7.63	8.06	9.00	9.00	8.50	8.88	0.00	8.44
	0.35	0.33	0.16	0.21	0.31	0.33	0.00	0.00	0.29	0.13	0.00	0.06
SA15	2.60	3.56	8.06	7.75	6.88	7.19	9.00	9.00	7.06	6.76	0.00	7.81
	0.41	0.40	0.33	0.25	0.13	0.19	0.00	0.00	0.52	0.78	0.00	0.51

Appendix 4.1. (continued)

1	Isolates of Leptosphaeria maculans:		MA - Manitoba SA - Saskatchewan WA - Western Australia OA - miscellaneous
2	Lines of brassica species:	B. napus	1 - R8314.26 2 - R8314.47 3 - Global 4 - Marnoo 5 - Karat 6 - R8311 7 - Regent 8 - Westar
		B. rapa	9 - BLC-198 10 - Tobin
		B. juncea	11 - UM3233 12 - UM3132

3 upper value is the mean, lower value is the standard error (0 - 9 scale).

Appendix 4.2. Disease severity means and standard error of each isolate line-combination (seedling test). Values are the mean of 4 replications ± SE.

Isolate[1]	Lines[2]											
	1	2	3	4	5	6	7	8	9	10	11	12
MA1	0.00[3]	0.14	1.48	3.09	1.73	3.34	2.72	3.83	2.07	3.75	0.00	1.59
	0.00	0.08	0.21	0.20	0.72	0.44	0.90	0.10	1.02	0.25	0.00	0.67
MA2	0.08	0.13	0.97	3.92	1.66	2.23	2.94	3.48	3.25	3.53	0.00	2.57
	0.00	0.08	0.19	0.08	0.36	0.36	0.43	0.23	0.34	0.47	0.00	0.59
MA3	0.00	0.15	0.62	3.29	1.17	2.93	1.92	2.44	2.83	3.51	0.00	2.31
	0.00	0.15	0.26	0.32	0.74	0.55	0.78	0.38	0.59	0.21	0.00	0.66
MA4	0.01	0.02	1.54	3.54	2.82	1.83	2.77	2.97	3.92	4.00	0.00	1.25
	0.01	0.02	0.60	0.36	0.47	0.54	0.36	0.53	0.08	0.00	0.00	0.30
MA5	0.00	0.00	0.17	3.04	0.83	0.68	2.08	3.01	1.61	3.66	0.00	0.90
	0.00	0.00	0.09	0.40	0.36	0.25	0.38	0.45	0.48	0.20	0.00	0.32
MA6	0.00	0.00	1.75	2.83	2.91	2.84	3.55	3.75	3.61	3.68	0.00	2.64
	0.00	0.00	0.67	0.95	0.54	0.38	0.34	0.25	0.39	0.23	0.00	0.55
MA7	0.00	0.02	1.20	4.00	2.41	3.37	3.68	3.75	3.55	4.00	0.00	2.91
	0.00	0.02	0.35	0.00	0.79	0.15	0.22	0.25	0.45	0.00	0.00	0.68
MA8	0.05	0.09	1.01	4.00	1.28	2.55	3.15	3.42	3.75	4.00	0.00	1.00
	0.05	0.09	0.58	0.00	0.52	0.37	0.34	0.58	0.16	0.00	0.00	0.12
MA9	0.00	0.01	0.12	2.04	1.26	0.97	0.79	2.58	2.68	4.00	0.00	0.84
	0.00	0.01	0.05	0.52	0.14	0.21	0.21	0.42	0.69	0.00	0.00	0.33
MA10	0.00	0.00	0.21	3.37	1.15	1.83	2.81	3.45	3.91	4.00	0.00	2.66
	0.00	0.00	0.20	0.38	0.28	0.22	0.23	0.26	0.09	0.00	0.00	0.49

Appendix 4.2. (continued)

Isolate	Lines											
	1	2	3	4	5	6	7	8	9	10	11	12
MA11	0.00	0.00	1.13	3.92	2.83	1.83	2.77	2.97	3.92	4.00	0.00	1.25
	0.00	0.00	0.47	0.08	0.47	0.54	0.36	0.54	0.08	0.00	0.00	0.30
MA13	0.00	0.03	1.45	2.24	3.54	2.34	2.99	3.83	1.85	4.00	0.00	3.16
	0.00	0.03	0.33	0.26	0.36	0.21	0.43	0.17	0.67	0.00	0.00	0.48
MA14	0.08	0.03	0.76	3.60	1.57	2.69	3.74	3.92	3.89	4.00	0.00	2.09
	0.05	0.02	0.08	0.30	0.65	0.27	0.26	0.08	0.11	0.00	0.00	0.70
MA16	0.01	0.10	1.21	3.33	1.86	3.17	3.17	3.83	3.55	3.91	0.00	2.04
	0.01	0.10	0.32	0.47	0.38	0.23	0.63	0.17	0.31	0.09	0.00	0.89
MA17	0.01	0.05	0.23	1.31	1.41	1.61	2.36	2.56	3.57	3.58	0.00	2.30
	0.01	0.03	0.09	0.02	0.46	0.41	0.71	0.24	0.33	0.31	0.00	0.64
MA18	0.02	0.00	1.48	2.62	3.38	3.01	2.04	3.66	2.61	3.52	0.00	2.54
	0.02	0.00	0.47	0.25	0.36	0.55	0.27	0.13	0.75	0.31	0.00	0.89
MA25	0.00	0.00	0.70	3.53	2.00	3.24	4.00	3.67	3.34	4.00	0.00	2.67
	0.00	0.00	0.12	0.30	0.50	0.31	0.00	0.33	0.45	0.00	0.00	0.55
OA23	0.18	1.55	0.64	1.68	0.73	2.24	2.30	2.25	3.83	3.16	0.12	1.80
	0.10	0.18	0.22	0.68	0.30	0.14	0.62	0.13	0.10	0.40	0.07	0.77
OA45	0.64	0.13	0.48	1.84	1.07	2.74	1.69	3.03	2.84	4.00	0.11	0.19
	0.17	0.29	0.14	0.41	0.20	0.49	0.36	0.56	0.80	0.00	0.11	0.14
WA32	0.01	0.58	0.33	2.14	0.97	1.26	1.14	2.13	3.67	3.36	0.00	1.91
	0.00	0.31	0.11	0.23	0.13	0.35	0.29	0.21	0.24	0.42	0.00	0.05

Appendix 4.2. (continued)

Isolate	Lines											
	1	2	3	4	5	6	7	8	9	10	11	12
WA40	0.00	0.01	0.55	1.01	1.63	2.56	0.99	2.43	3.11	4.00	0.00	1.12
	0.00	0.01	0.23	0.46	0.17	0.61	0.20	0.08	0.57	0.00	0.00	0.40
WA51	0.32	1.02	1.18	2.24	0.75	2.08	3.18	3.42	3.53	4.00	0.01	3.21
	0.21	0.52	0.43	0.79	0.15	0.37	0.50	0.24	0.47	0.00	0.01	0.46
WA53	0.24	0.67	2.16	2.46	1.75	1.65	2.71	2.45	3.02	3.56	0.00	1.14
	0.15	0.34	0.39	0.54	0.81	0.82	0.44	0.29	0.65	0.44	0.00	0.52
WA58	0.29	0.87	0.70	1.88	0.96	1.89	1.57	2.69	3.37	3.84	0.04	2.30
	0.15	0.17	0.27	0.73	0.14	0.28	0.27	0.34	0.33	0.16	0.04	0.66
WA14	2.45	2.37	1.85	3.22	1.97	2.48	3.01	3.13	3.83	3.92	0.00	3.18
	0.53	0.13	0.61	0.78	0.68	0.74	0.33	0.42	0.17	0.08	0.00	0.48
WA17	0.19	0.32	0.32	2.09	2.25	1.43	2.09	2.92	4.00	2.48	0.00	1.63
	0.10	0.13	0.21	0.06	0.25	0.31	0.82	0.49	0.00	0.34	0.00	0.45
SA1	0.08	0.06	0.86	2.01	1.46	2.68	2.20	2.35	3.15	3.75	0.02	1.64
	0.05	0.04	0.21	0.51	0.63	0.50	0.37	0.23	0.65	0.25	0.02	0.37
SA4	0.02	0.00	0.78	2.51	1.11	1.46	2.33	2.79	3.91	4.00	0.00	0.87
	0.01	0.00	0.37	0.52	0.24	0.28	0.56	0.42	0.08	0.00	0.00	0.31
SA10	0.08	0.17	0.76	3.60	2.27	3.56	3.24	3.67	3.88	4.00	0.00	2.56
	0.05	0.11	0.21	0.24	0.41	0.28	0.54	0.23	0.13	0.00	0.00	0.66
SA15	0.04	0.10	0.91	0.56	1.94	3.92	2.37	3.99	3.03	4.00	0.00	1.61
	0.02	0.09	0.31	0.28	0.31	0.08	0.34	0.01	0.57	0.00	0.00	0.49

Appendix 4.2. (continued)

1 Isolates of Leptosphaeria maculans:

MA - Manitoba
SA - Saskatchewan
WA - Western Australia
OA - miscellaneous

2 Lines of brassica species:

B. napus
1 - R8314.26
2 - R8314.47
3 - Global
4 - Marnoo
5 - Karat
6 - R8311
7 - Regent
8 - Westar

B. rapa
9 - BLC-198
10 - Tobin

B. juncea
11 - UM3233
12 - UM3132

3 upper value is the mean, lower value is the standard error (0 - 4 scale).

Appendix 5.1. ANOVA for blackleg disease and yield of oilseed rape on cultivars, maturity groups and fungicide treatments at Souris, 1988.

Source	df	Disease MS	Yield MS
Replicate (R)	3	0.57**	234569.22
Treatment (T)	4	0.13	259393.00
R*T (error a)	12	0.76	408338.63
Maturity (M)	2	32.21**	30137.85
T*M	8	0.06	59560.24
R*M\T (error b)	30	0.12	45656.81
Cultivar (C)\M	3	8.80**	209041.60**
T*V\M	12	0.09	11767.48
error c	45	0.03	32605.70

* significant at 5% level
** significant at 1% level

Appendix 5.2. ANOVA for blackleg disease and yield of oilseed rape on cultivars, maturity groups and fungicide treatments at Minnedosa, 1988.

Source	df	Disease MS	Yield MS
Replicate (R)	3	0.09**	671228.63*
Treatment (T)	4	0.02	17910.48
R*T (error a)	12	0.01	158832.82
Maturity (M)	2	0.59**	2234230.90**
T*M	8	0.01	35765.92
R*M\T (error b)	30	0.02	61975.51
Cultivar (C)\M	3	0.08**	471613.10**
T*V\M	12	0.01	68237.53
error c	45	0.01	54542.94

* significant at 5% level
** significant at 1% level

Appendix 5.3. ANOVA for blackleg disease and yield of oilseed rape on cultivars, maturity groups and fungicide treatments at Elgin, 1989.

Source	df	Disease MS	Yield MS
Replicate (R)	3	1.50**	1761.08
Treatment (T)	4	0.06	1589.20
R*T (error a)	12	0.10	1454.43
Maturity (M)	2	25.48**	94976.06**
T*M	8	0.07	1334.16
R*M\T (error b)	30	0.22	1449.19
Cultivar (C)\M	3	10.12**	736.37*
T*V\M	12	0.80	226.03
error c	45	0.22	197.44

* significant at 5% level
** significant at 1% level

Appendix 5.4. ANOVA for blackleg disease and yield of oilseed rape on cultivars, maturity groups and fungicide treatments at Souris, 1989.

Source	df	Disease MS	Yield MS
Replicate (R)	3	2.45*	817.86**
Treatment (T)	4	0.91	125.13
R*T (error a)	12	0.63	310.44
Maturity (M)	2	47.71**	31110.91**
T*M	8	0.36	85.17
R*M\T (error b)	30	0.21	365.27
Cultivar (C)\M	3	6.89**	141.23*
T*V\M	12	0.10	27.02
error c	45	0.15	33.81

* significant at 5% level
** significant at 1% level

Appendix 5.5. ANOVA for yield of oilseed rape on cultivars, maturity groups and fungicide treatments at Carman, 1989.

Source	df	Yield MS
Replicate (R)	3	50565.79
Treatment (T)	4	9062.40
R*T (error a)	12	86648.45
Maturity (M)	2	423467.83**
T*M	8	84119.26
R*M\T (error b)	30	11796.72
Cultivar (C)\M	3	43389.97**
T*V\M	12	4841.10
error c	45	6129.67

* significant at 5% level
** significant at 1% level

Appendix 5.6. ANOVA for blackleg disease and yield of oilseed rape on cultivars, maturity groups and fungicide treatments at Melfort, 1989.

Source	df	Disease MS	Yield MS
Replicate (R)	3	1.80*	34489.31
Treatment (T)	4	0.44	10870.37
R*T (error a)	12	0.21	18902.22
Maturity (M)	2	68.60**	994414.85**
T*M	8	0.14	7344.00
R*M\T (error b)	30	0.14	5604.00
Cultivar (C)\M	3	10.96**	669003.37**
T*V\M	12	0.79	2722.99
error c	45	0.15	8342.55

* significant at 5% level
** significant at 1% level

Appendix 5.7. ANOVA for yield of oilseed rape on cultivars, maturity groups and fungicide treatments at St. Brieux, 1989.

Source	df	Yield MS
Replicate (R)	3	4961.43
Treatment (T)	4	25696.86
R*T (error a)	12	19362.31
Maturity (M)	2	131824.40**
T*M	8	11457.56
R*M\T (error b)	30	13460.85
Cultivar (C)\M	3	44030.26**
T*V\M	12	11620.78
error c	45	8550.62

* significant at 5% level
** significant at 1% level