Effect of Pasmo on Flax in Manitoba and Inference of the Sexual State of the Fungus by Molecular Polymorphism

By

Lynn Grant

A Thesis submitted to the Faculty of Graduate Studies of the

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In partial fulfilment of the requirements of the degree of

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Effect of Pasmo on Flax in Manitoba and Inference of the Sexual State of the Fungus by Molecular Polymorphism

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ABSTRACT

Septoria linicola (Speg) Garassini (teliolmorph *Mycosphaerella linorum* Naumov) causes the disease pasmo in flax in many flax growing areas. The effects of fungicide application and inoculation on flax under field conditions were studied on six varieties at Morden, Manitoba at the Agriculture and Agri-Food Canada Research Station as well as at Winnipeg, Manitoba at the University of Manitoba Field Station during the 2003 and 2004 growing seasons. Yield, seed oil and protein content and 1000 kernel weight were generally reduced under heavy infections for most cultivars.

The use of fungicides increased yield significantly for almost all the cultivars in all years when compared to a control with no fungicide application. Area under the disease progress curve (AUDPC) was also significantly reduced for all fungicide application treatments except for the inoculated with fungicide application treatment in 2004. Seed oil content was significantly improved for all fungicide application treatments in all years for all cultivars except Norlin in 2003. Seed protein content did not show any clear response to fungicide application. Thousand kernel weight was significantly positively affected by the application of fungicides for all treatments in all years except for the cultivar Vimy at the Winnipeg site in 2003.

The structure of two *S. linicola* populations in Manitoba was studied using Random Amplified Polymorphic DNA (RAPD), a PCR based molecular marker system. Plants were collected from two commercial fields in Portage and Sanford and used to generate single spore isolates for use in this study. The level of polymorphism detected using RAPD suggests that it is plausible that there is sexual recombination occurring between the two populations, or that there is extensive movement of

individual isolates throughout the province. Limited grouping based on site was seen in the dendrograms. Analysis of Molecular Variance (AMOVA) revealed that 88% of the total genetic variation was due to within population variation and 12% to between population variation. This suggests a mix of clonal and sexual reproduction during the growing season.

FOREWARD

This thesis is written in the manuscript style, with each manuscript including its own abstract, introduction, materials and methods and results discussion and/or conclusion sections. There is a general introduction and review of the literature prior to the manuscripts, followed by the general discussion and conclusions, and literature cited section.

1. INTRODUCTION

Septoria linicola (Speg) Garassini (teliolmorph *Mycosphaerella linorum* Naumov) is a fungal pathogen that infects flax. It causes a disease known as pasmo, and is found in many flax growing areas (Rashid, 2003).

Pasmo has been reported to reduce yield (Perrryman and Fitt, 2000; Rashid 2004) and seed oil content (Sackston, 1959) under severe infections. No good sources of resistance have been found to date, and commercial cultivars do not show a high level of resistance (Rashid, 2003).

The sexual state of *S. linicola* (Speg) Garassini has been reported by Wollenwebber (1938), who found it on samples of flax obtained from Argentina. Sackston (1949a) found what he believed to be *S. linicola* (Speg) Garassini perithecia in Manitoba in 1944 but was unable to confirm this.

Indirect means have been employed for *Septoria tritici* Roberge in Desmaz. (*Mycosphaerella graminicola* (Fückel) J. Schröt in Cohn) in Manitoba to lend support to determining the population structure of that fungus (Hoorne, 2002). Hoorne (2002) used the AFLP (Amplified Fragment Length Polymorphism) molecular marker system as one tool in showing that the sexual state of that fungus existed in Manitoba.

There is limited knowledge about the life cycle of the fungus in most flax growing areas. It is important to understand the population structure of fungal pathogens as this understanding contributes to a better understanding of the life cycle, which in turn leads to the use of more suitable control practices. There are also

implications for the longevity of control measures such as fungicides or resistant cultivars under different life cycles.

To date, there are no confirmed reports of the sexual state of *S. linicola* in Manitoba. The objectives of this study were to assess the population structure of *S. linicola* (Speg) Garassini in Manitoba and to determine the effects of the disease on a selection of commercial flax cultivars.

2. LITERATURE REVIEW

2.1 THE HOST PLANT

2.1.1 Nomenclature

The scientific name given to flax, *Linum usitatissimum* L., has its origin in Latin where *linum* meant 'flax' (Judd, 1995), and *usitatissimum* meant most useful in Latin (Kolodziejczyk and Fedec, 1995). *Linum usitatissimum* is in the genus *Linum*, which is in the family *Linaceae* Dumort (Diederichsen and Richards, 2003).

2.1.2 Origin

The present annual cultivated flax likely evolved from weedy or wild forms which represent perennial life cycles. *Linum bienne* Mill, a wild flax, which is found in North Africa, the Mediterranean Basin, the near East, the Caucasus, Western Europe, and Iran, has been suggested as a likely ancestor of flax. It has dehiscent bolls or capsules of seed, strong branches, blue flowers and the same chromosome number as commercial flax (2n=30) (Zohary and Hopf, 2000).

Syria is the site with the oldest linseed discovery (9200 -8500 BC). It is thought that this site predates farming (Zohary and Hopf, 2000). Flax is thought to have been domesticated around 7000 BC in the fertile crescent (VanZeist and Bakker-Heeres, 1975; Smith, 1995). Excavation of farming villages in the Near East used by pre-Pottery Neolithic B peoples from the second half of the 8^{th} millennium and the 7^{th} millennium BC have turned up flaxseeds. These seeds are usually found along with domesticated

barley and wheat (Zohary and Hopf, 2000). Since flax is among one of the first domesticated crops it is considered a founding crop (Vaisey-Genser and Morris, 2003). There is uncertainty as to when exactly flax moved from being a perennial crop to an annual crop, but it is believed to have taken place before the Christian era (Singh, 1987).

Flax was spread from the near east to West Asia, Europe and the Nile valley through the spread of agriculture (Zohary and Hopf, 2000). The relatively large seed size found in Iraq, parts of Mesopotamia and in Syria and dated to before 6000 BC suggest that flax was an important crop during the evolution of irrigation in agriculture (Zohary and Hopf, 2000). The most thoroughly documented case for the early use of flax is the fibre produced from the stalks of the flax plant. Flax has also served many other purposes over the course of history, as a medicinal product and as a food (Geijer, 1979). There is archeological evidence that flax oil was in use in China starting between five and two thousand years ago (Pan, 1990).The oil has been used for lamp oil, frying food, in flooring and paints and as a preservative. Many other uses have been made of flax in different countries (Vaisey-Genser and Morris, 2003).

Flax was brought to Canada, to the area of New France, by a European farmer named Louis Hebert (Anonymous, 2008). The introduction of flaxseed into western Canada occurred around 1875, and it became a very important crop, in part because of its value in breaking virgin soil (Lehberg and Anderson, 1941). In areas where peanuts and olives could not be grown, including portions of North America and Europe, interest in the oil produced by the flax seed grew in the first half of the twentieth century (Vaisey-Genser and Morris, 2003). Canadian production peaked in 1912 at 26

million bushels (Lehberg and Anderson, 1941). With the increase in wheat prices during the second world war flax production declined (Daun and DeClercq, 1994). During the second world war restrictions were placed on the importation of edible oils. Around the same time, it became legal to use margarine as a table spread in some provinces in Canada. This resulted in research into linseed to determine its suitability as a domestic oil seed crop (Hunt, 1969). Research conducted in the 1950's found that an off flavour or 'flavour reversion' occurred in shortenings and salad oils produced from flax oil. It was concluded that the high alpha linolenic acid content of the flaxseed oil was causing the off taste (Lemon, 1947; Armstrong and McFarlane, 1994). This caused a halt to commercial production of flax seed oil (Vaisey-Gerner and Morris, 2003). Today a cold press process carried out in a low oxygen environment coupled with lightproof containers are helping to prolong the shelf life of flaxseed oil (Carter, 1993). Flaxseed is being used in baked goods and other foods as a functional food and there has also been an increase in the use of natural linoleum in recent years (Vaisey-Genser and Morris, 2003).

2.1.3 Oil Seed and Fibre Flax

Commercial flax is a herbaceous annual plant. Flax varieties can be divided into two types based on morphology, fibre and oil seed flax (or linseed). Fibre flax varieties grow straight and tall and are less likely to branch. Varieties grown for oil production tend to be shorter and often produce more branches (Singh, 1987). Flaxseed and linseed may mean different things in different regions of use. In Canada and the United States the words are both used to describe the crop, with a slight tendency to use the word

flaxseed to designate flax grown for human consumption. In Europe linseed is used to designate oilseed flax grown for industrial and nutritional uses while the word flax is used when speaking of plants grown for fibre production (Oomah and Mazza, 1998). Different varieties of flax have been developed for oil or fibre production (BeMiller, 1973). The term Solin refers to a flax variety with less than three percent alpha-linolenic acid (ALA) in the seed, which is in contrast to more traditional varieties that contain approximately 57% ALA, an essential omega-3 fatty acid (Oomah and Mazza, 1998).

The average oil content of flaxseed produced in Western Canada ranged between 41 and 46%, based on dry weight, during the years 1934 to 1993. There was a slight increase in oil content during this same period, most likely due to improved agronomic practices as well as breeding. Oil content is important because flaxseed oil can be employed in both industrial and human and animal uses (Vaisey-Genser and Morris, 2003; Scheidler, 2003). Maintaining a high oil content under disease conditions is important from a marketing and consistency standpoint.

The protein content of flaxseed varieties grown in Canada tends to be between 20 and 24 % (Duguid et al. 2003). Flax grown in the southern areas of Canada tends to have a higher protein content and a lower oil content than seed grown in more northern areas (Dorrell and Daun, 1978). Protein content of the seed is important if the seed is going to be used in human or animal supplements or food products. Meal is a by-product of the oil extraction process and is used in animal feeds for its protein content (Scheidler, 2003).

2.1.4 Commercial Production

The 2007/2008 season saw approximately 524 thousand hectares harvested for a total of 634 thousand tonnes, with an average price of $560-600 /tonne. Estimates for the 2008/2009 season are 565 thousand hectares which will yield about 705 thousand tonnes, for a forecast price of $560-600 /tonne (approximately $394 800 to $423 000 thousand in net receipts) (Agriculture and Agrifood Canada, 2008).

2.1.5 Agronomics

In North America flax is principally grown on the Canadian Prairies and in the North Central United States in the Black, Dark Brown, Dark Grey and Brown Chernozemic soil zones. Oil seed varieties are heavily favoured on the Prairies. Manitoba and Saskatchewan make up the largest areas seeded to oil seed flax in Canada. Zero- and minimum-tillage practices have been increasingly employed in recent years and flax can be successfully produced using these practices (Marchenkov et al. 2003).

A minimum of three years between flax crops is advised. Flax should be seeded in rows 15 to 20 cm apart, and can even be planted in rows up to 30 cm apart. In Manitoba flax is generally seeded between the tenth and thirty first of May (Anonymous, 2002). Early seeding favours higher yield (Sackston, 1949b).

Insect pests of flax include grasshoppers, aphids, army cutworms, cutworms, wireworms, aster leafhoppers, beet webworms and bertha army worms (Anonymous, 2002).

There are many diseases that can affect flax. Rust (*Melampsora lini* (Ehrenb.) Desmaz.) has long been an important pathogen but there has not been an outbreak since the 1970's (Hoes and Tyson, 1963; Zimmer and Hoes, 1974; Hoes and Zimmer, 1976; Rashid, 2003). The current situation in North America is such that all commercial cultivars are immune to local rust races (Rashid and Kenaschuck, 1992; 1994). Fusarium wilt (*Fusarium oxysporum* f. sp *lini* (Bolley) W. C. Snyder and H. N. Hans.) is a widespread disease and is commonly problematic when flax has been grown in the same field over a long time. While severe epidemics are uncommon, a severe infection can cause a reduction in yield of between 80 and 100% (Kommedahl et al. 1970; Sharma and Mathur, 1971; Kroes et al. 1999). All commercial cultivars of flax in North America are moderately resistant or resistant to fusarium (Kenaschuk and Rashid, 1993; Kenaschuck et al. 1996). Pasmo (*Septoria linicola* (Speg.) Garassini) is also important in North America. Alternaria blight is seen occasionally but there have been no major epidemics. Powdery mildew (*Oidium lini* skoric) has also been observed in commercial fields (Rashid, 1998; Rashid et al. 1998a). Sclerotinia stem rot (*Sclerotinia sclerotiorum* Lib.) de Bary) has been reported in Canada, particularly in heavily lodged (Rashid, 2000) or irrigated fields (Mederick and Piening, 1982). Browning and stem break (*Aureobasidium lini* (Lafferty) Hermanides-Nijhof) (Henry, 1934; Henry and Ellis, 1971), anthracnose (Rashid, 2003), seedling blight (Vest and Comstock, 1968), damping off (Millikan, 1951), aster yellows phytoplasma (Rashid, 2003), crinkle (Oat blue dwarf virus) (Hoes, 1975; Rashid et al. 2000), and curly top are of limited importance in Canada (Rashid, 2003).

2.2 THE FUNGAL PATHOGEN

2.2.1 Nomenclature and Taxonomy

Pasmo disease of flax may also be called septoriosis or spasm (Rashid, 2003). The word pasmo means spasm in Spanish (Loughnane, et al. 1946, Sackston, 1949a). It is thought that the popular name for the disease, spasm, might have come about since the disease appears and spreads rapidly in fields of flax just before harvest (Sackston, 1949a).

Pasmo of flax is caused by the pathogen *Septoria linicola* (Speg.) Garassini (teliomorph *Mycosphaerella linorum* Naumov). The fungus had been called *Phlyctena* ? *linicola* Speg. n.f. as a provisional name until the fungus could be better classified (Spegazzini 1911). Brentzel (1926) added support to this classification since the pycnidia were not complete, only nearly so, and occurred on the stems and were not limited to leaves. He did however point out that the fungus bore a resemblance to certain *Septoria* species. The current name was first employed by Garassini in 1935. The name was then used by Rost (1937) and Wollenweber (1938). Arguments for the inclusion of the fungus in the *Septoria* genus were made later by Garassini (1939).

The genus *Septoria* Sacc. is anamorphic. The large majority are coelomycetes that are pathogenic on plants. Most taxa employ leaves as food sources and cause leaf spot diseases (Verkley at al. 2004). *Septoria cystis* Desm. is the type species of *Septoria* (Sutton, 1980).

The sexual state is rarely seen, with only a few reports in the literature. Wollenweber (1938) obtained pasmo infected flax samples from Pergamino, Argentina

and found that there were perithecia on the stems. Ascospores collected from the perithecia and grown in culture produced typical *S. linicola* colonies. He named the perfect stage *Sphaerella linorum* n.sp. Kruger (1941) found immature perithecia in Germany, and Sackston (1949a) found what he believed to be perithecia of the fungus in December of 1944 in Manitoba but was unable to complete Koch's postulates.

2.2.2 Distribution, Prevalence and Incidence

Spegazzini was the first person to observe the causal organism of pasmo. He reported finding it near La Plata in Argentina in December of 1909 (Spegazzini, 1911).

The disease has been found on every continent with reports from India (Singh, 1987), Europe (Muskett and Colhoun, 1947; Rost, 1937; Wollenwebber and Kruger, 1938; Naumoff, 1926), North America (Brentzel, 1923, 1926; Rodenhiser, 1930), South America (Spegazzini, 1911; Wollenweber, 1938), Africa (Colhoun and Muskett, 1943; Nattrass, 1943), New Zealand (Cunningham, 1931, Millikan, 1948), and Australia (Millikan, 1948).

It is thought that the fungus was introduced into the United States when flaxseed was imported, possibly from Argentina, and grown for breeding, research or commercial purposes (Brentzel, 1926). Pasmo was initially identified in Canada in 1939, was first observed in Manitoba in 1940 (Sackston, 1947b), and was seen in Manitoba annually beginning in 1942 (Sackston, 1947b, 1948, 1949b; Vanterpool, 1945). It is thought that the pathogen moves from site to site as spores adhered to seeds or that small pieces of diseased plant that remain among the seeds start infections in new areas after being planted along with the seed.

2.2.3 Economic Importance

It has been observed by Sackston (1959) that lower oil content in the seeds can result from severe infection. Seed weight and size are often reduced in heavy infections of pasmo leading to reductions in yield (Sackston, 1950; Perryman and Fitt, 2000; Rashid, 2004).

2.2.4 Symptomology, Infections and Dispersal

The disease is considered to be a disease of leaves and stems; bolls and leaves can all be infected (Rashid, 2003). Seedlings infections may start out as tiny pale flecks on cotyledons, which may be undetectable without a microscope. The flecks then increase in size to 0.5-5 mm or more in diameter. They then become darker and water soaked, with colour changing from green-grey to brown. Lesions then begin to appear on leaves of the plants, following the same progression as is seen on the cotyledons. The outline of the lesions is irregular to round. Lesions appear randomly on the surface and are not limited by leaf veins. Lesions may occupy up to one half of the leaf or more. Individual lesions on the same leaf may remain separated or they may coalesce. Leaves with heavy infections may become chlorotic. On mature plants, colour changes of lesions are from pale green to green-yellow, to light brown ending in dark brown lesions (Sackston, 1949a). Within the brown lesions many pycnidia are formed which are darkly pigmented. Throughout the growing season the disease moves up the stem so that by harvest branches, leaves and bolls of the plant are infected. Under heavy infection many of the plants are almost completely defoliated by harvest as the lesions often cover the entire leaf and cause drying out and death of the leaves. The stems show

a pattern of alternating stripes of brown and green along the length as lesions expand and circle the stem, giving the stems a striped appearance (Rashid, 2003). Flowers and bolls may be infected and blighted, or pedicels may be damaged and result in abscission of flowers or bolls. When sepals, which seem to be more susceptible to infection, are attacked they turn brown and may eventually appear bleached, or silvery. Stems and pedicels often also exhibit this bleaching characteristic late in the infection. Once the bleached stage has been reached there are numerous pycnidia in the tissue (Sackston, 1949a). Pedicels and stems become weakened and rain and wind may cause boll drop and breakage of infected stems (Rashid, 2003).

On a field scale the disease may be observed as brown areas in the field that enlarge as harvest approaches. Sometimes, the entire field may turn brown prematurely. Within patches, plants on the margins may seem healthy or may have only a few small lesions. Plants at the centre of the patches are progressively more infected and often the innermost plants no longer have any leaves and may be entirely brown and dried out (Brentzel, 1926; Sackston, 1949a). The scattered patches created by the disease may give the appearance of irregular ripening and are often quite conspicuous in an unripe field. If the conditions are favourable the patches may spread through the entire field, even large commercial fields, within one to two weeks after the appearance of the first patches. A reddish hue to pasmo infected plants can sometimes serve to distinguish naturally ripened fields from prematurely ripened pasmo infected fields (Sackston, 1949a). Sackston (1949a) has observed some fields that seem to have diseased plants evenly distributed throughout the field in clumps or as single plants.

Often the seed harvested from infected fields will be thinner than healthy seed. In addition, the seed may look dull. In years when the infection occurred early or was particularly severe it has been observed that the seeds will be small and thin, greyish in colour, and scabby or wrinkled. Seeds produced from heavily infested fields may also have pycnidia on their surface but this is relatively rare (Sackston, 1949a).

Pycnidia in lesions tend to be lens shaped. In their early stages they are fairly incomplete, but are almost complete at maturity with small ostioles. On leaves and stems the pycnidia develop below the epidermis, with stem borne mycelia extending into the bast-fibre cells (Brentzel, 1926). Spegazzini (1911) noted that pycnidia size can range between 75 and 150 μ in diameter. Pycnidia were also observed to change from pale brown to dark brown at maturity and to have lens shaped ostioles (Spegazzini, 1911).

Pycnidiospores are hyaline, cylindrical, elongated and can be irregularly curved or straight. Generally the spores have three septa (Millikan, 1951).

The fungus produces large numbers of pycnidia, with numbers ranging between one and 70 per mm^2 of plant tissue. These pycnidia have the capacity to produce between 1000 and 10 000 spores. Since the spores exit the pycnidia in a gelatinous matrix, sometimes called a cirrus, they have not been observed to move readily with only wind as a dispersal mechanism, especially once they have dried. Strong wind, coupled with the force of raindrops is thought to play a role in spore movement. A more probable mode of dissemination is that of animals and insects. It has been shown that insects as well as animals and people can become covered in spores when they move through infected flax fields after a precipitation event while the spores were still moist.

These spores could then introduce the disease into another field or move the infection within the same field (Christensen, 1952).

Sackston (1970) also maintained that spores produced from the pycnidia cannot be easily dispersed by wind alone but proposes that it may be possible that droplets of water remaining on leaves of infected plants may be a possible source of inoculum. Sackton (1970) has studied the germination of pycnidiospores and has found that while most of them produce germ tubes which become hyphal threads, some produced multiple secondary spores. The tendency to produce spores is favoured by high numbers of spores in a suspension, which is often the case when droplets of water remain on leaves and an entire cirrus is dissolved into the droplet. The spores that are produced under these conditions are more likely to be dispersed by wind and are thought to play a role in the dispersal of the fungus in more mature flax fields (Sackston, 1970).

It has been noted that the ability of the spores to adhere to the leaves is one of the most important aspects of the infection process. Some researchers have maintained that flax seems to be somewhat more resistant to pasmo between the cotyledon and the flowering stages (Brentzel, 1926; Kruger, 1941; Loughnane et al. 1946). As Sackston (1949a) noted when he conducted artificial inoculation experiments, leaves during this developmental period do not seem to retain suspensions that are sprayed on, while cotyledons do. The liquid runs off the leaves and thus the spores are unable to infect the leaves. When surfactants were added to the liquid, infections occurred much more consistently. It can then be concluded that leaf surface tension is likely playing a role in this type of resistance and that the resistance is not true resistance, simply physiological escape. Soriano (1928) had previously noted that the ability of young leaves to avoid

wetting likely played an important role in their escaping, temporarily, infection. Field inoculations carried out by Sackston (1949a) showed that as long as the spores were able to adhere to the leaves the plants were susceptible to pasmo at any stage.

Sackston (1949a) reported that the fungus gains access to the leaf through the stomates. No structures such as appresoria were seen. Sometimes germ tubes would pass over multiple stomates before they penetrated one, in other cases germ tubes found stomates almost immediately and turned down into them. It was not determined what triggered the germ tubes to turn down into a stomate.

2.2.5 Environmental Requirements and Epidemiology

Research has been conducted to determine favourable temperatures for the fungus. The fungus does not grow well below 5°C, that it grows best at 21°C and that temperatures above 32°C hinder growth (Brentzel, 1926). Brentzel (1926) concluded that the fungus grew best at a range between 17°C and 29°C. Results reported by Rodenhiser (1930) supported those of Brentzel and further concluded that temperatures of 17°C, 22°C and 27°C gave the largest differences in growth rates between different isolates in laboratory culture. Borges (1946) found that 25°C was the best temperature for the growth of *S. linicola* while a temperature range between 20 °C and 24°C was found by Kruger (1941) and Wollenweber (1938) to be optimal. In culture, it was found by Sackston (1949a) that sporulation was highest at 20°C and 25°C, with few spores produced at 10°C and 30°C.

The rate of disease development is influenced somewhat by temperature. When temperature was maintained around 27°C lesions developed in approximately six days.

When the temperature was around 21°C it took approximately nine days for lesions to appear. Eleven days had elapsed at 15.5°C before lesions began to appear on plants (Sackston, 1949a).

Moisture conditions have a huge impact on the progression of pasmo in the field. Brentzel (1926) noted that areas in fields that had high humidity tended to have more severe pasmo infections. Sackston (1949a) also noted that lower lying areas in a field had more severe infestations and fields that had experienced longer periods or high relative humidity or that had received a large amount of precipitation were more likely to have severe infections. Lodged areas also tended to have a higher rate of infestation. In greenhouse experiments the same pattern was seen, those plants kept in humid conditions longer after inoculation were more severely infected than those with shorter high humidity exposure times. At very low humidities pycnidia formation was reduced.

Seed harvested from fields infested with pasmo may serve as a source of infection if planted. Brentzel (1926) observed that when both clean seed and seed from an infected field were sown on the same field, the area with the infected seed had severe pasmo infection while the area sown with clean seed had relatively minor levels of infection. Pasmo was also observed to appear earlier in the spring in plots that had been seeded with infected seed than in other plots. The appearance of pasmo on flax in research plots widely separated from each other but that were seeded with seed from the same source, and of the same variety, suggested to Natalyina (1932) that the disease was seed-borne. Newhook (1942) felt that either mycelium in the seed coat or spores on the surface of the seed were the sources of infection from the seed. It was shown by Loughnane et al. (1946) that the mycelia had the ability to move from the sepals to the

petals and from there into the placenta. The fungus was then able to penetrate the funiculus and move into the seed coat. Pycnidia were observed to be forming on the seed coat and there were pycnidia on the placenta, which were releasing spores that were contaminating the seed. This meant that there was a possibility that the spores on the seed coat could infect the cotyledons on the germinating seedling since the seed coat is often brought above ground during emergence.

Sackston (1949a) found that in Manitoba seeds obtained from diseased plants did have many spores on their surfaces. When plated some of the seeds did produce colonies of the fungus, other seeds had pycnidia develop on the seed coats. Other seeds were allowed to germinate and the cotyledons of some of these plants developed lesions. When *S. linicola* spore contaminated seeds were planted in soil it was found that very few of them, only one of thousands, developed cotyledon lesions. Thus, although it is very unlikely, under very favourable conditions, it is possible for contaminated seed to be infected by spores on the seed coat. The low frequency of contaminated seed becoming infected is further supported by Loughnane et al. (1946) who stated that they have never seen any experimental proof that seeds that are contaminated on the surface with *S. linicola* spores have ever produced infected seedlings. They maintain that it is pycnidia or mycelia in the seed coat that is responsible for the infected plants. Sackston (1949a) comments that although pycnidia on the seeds are rare in Manitoba, they have been shown in experiments to be a successful method of disease transmission. A more likely source of inoculum is the pycnidia that are found on pedicels, sepals and other small bits of infected plant parts that are often mixed in with the seed. Often this plant tissue is planted with the seed

since it is difficult to remove from the seed and has been shown to successfully infect flax plants (Sackston, 1949a).

The possibility of soil containing spores being able to cause disease has also been ruled out by Sackston (1949a) since, under normal soil conditions, pouring spore suspensions on the soil and allowing seeds to grow did not produce any infected flax plants. The placement of sporulating colonies of *S. linicola* growing on cereal grains on the soil and the maintenance of the plants in a high humidity environment was effective for infecting seedlings. The same was observed when infected stubble and straw from the previous season were placed on the surface of the pots. Driving rain resulting in splash could easily spread the spores from straw or stubble to the flax plants in a field setting (Sackston, 1949a).

Wind is probably involved to some degree if the movement of the spores through the field is rapid. Dry spores have not been successfully trapped, it was found that wind with no water is not a very effective way to spread spores, but that when water is added the transmission of spores in the air current is much more successful (Sackston, 1949a, 1970). Since winds in western Canada can be quite strong, it is possible that some dry cirrhi may be dislodged but it is considered to be unlikely that this would make a significant contribution to disease dispersal. There are heavy dews in western Canada that keep plants wet for long periods of time. Cirri can dissolve in water droplets, leaving the spores suspended in water, and the combination of dews and strong winds likely favours longer range spore dispersal in most commercial fields (Sackston, 1949a).

Once the pathogen is introduced into an area it can survive on straw and diseased stubble. The pathogen is able to overwinter on straw and then serve as a source of inoculum for seedlings in the next season. Once a few plants were infected, spores would be continuously produced and this would in turn cause neighbouring plants to become infected (Brentzel, 1926). Early infections may affect only the lower portions of the plants, as was seen by Bolley (1931) in Argentina, while the tops of the plants may be infected in late plantings. This seemed to suggest that the late planted crops were being infected by wind blown spores from earlier plantings. Newhook (1942) also attributed secondary infection to spores blown by wind. Rain and wind were more important to Garassini (1935) when it came to disease spread, since he found that the spore mass of the fungus (the cirrus) became completely dissolved when water droplets covered them, and this allowed the individual spores to be dispersed by wind. Loughnane et al. (1946) believed that wind was the main means of spore movement but that insects and rain splash also played a role in plant-to-plant transfer of spores. Wollenweber (1938) thought that the conidia produced by the pathogen might be involved in the spread of the disease.

In the field pasmo will reach 100% of leaf area infected if conditions are favourable (Perryman et al. 1999). Ferguson et al. (1987) reported that the period after anthesis is the time when the effects of the disease are most important for yield components. Pasmo generally does not impact the number of flowers produced by the plant.

2.2.6 Epidemiological Prediction

It has been observed by Perryman et al., (1999) that rainfall events caused increases in the number of spores in the air, as long as the daily mean temperature reached 12°C. They reported that generally spore counts are high in June and increase around the middle of July. Since the disease is already present and well established on leaves and stems by this point they felt that the use of spore samplers to predict the severity of the disease would not be effective.

2.3 Disease Control

2.3.1 Resistance

It has been reported by many researchers that some resistance was observed in certain cultivars (Rodenhiser, 1930; Bolley, 1931; Garassini, 1935; Dillman, 1939; Kruger, 1941; Flor, 1943, 1944; Turvet, 1944; Spangenberg, 1944; Millikan 1948; Sackston, 1949a; Hannah, 1953; Covey, 1962; Loshakova and Korneeva, 1979; Loshakova, 1984; Turley and Snowdon, 1998). This has been attributed to factors such as date of maturity (Pederson and Michaelson, 1960), the wettability of the leaves (Covey, 1962; Sackston, 1949a), erratic distribution of the inoculum in the plots as well as the possibility of different races of the pathogen being present in different regions (Sackston, 1949a). Resistance testing has been extensive, but has not proven successful to date.

2.3.2 Cultural Control

The disease can be controlled by using proper field sanitation and by burning infected crop debris (Muskett and Colhoun, 1947; Girola, 1920). Pycnidia can overwinter on stubble and straw to provide inoculum in the spring. Overwintered pycnospores can initiate infections on healthy flax plants. Infected straw may serve as a source of inoculum well into the growing season (Brentzel, 1926). It is therefore recommended that infected straw be destroyed immediately after harvest (Butler, 1949).

In fields where straw was plowed under as a means of controlling the disease, it was observed that seedlings of flax grown in these fields the next year are often infected with the disease (Brentzel, 1926). Proper rotation of three or four years has been recommended as a control measure but is only effective if there are no sources of infection in the area (such as straw, other fields of flax or stubble) and if seed is completely disease free, both of which may be hard to achieve (Butler, 1949; Girola, 1920; Rashid and Kenaschuk, 1998). Rashid et al. (1994) report that conventional tillage and summer fallow were the most effective tillage systems to control pasmo.

Girola (1920) and Rashid and Kenaschuk (1998) recommended not using seed from infected fields, using measures such as weed control and recommended seeding rates to ensure that the microclimate does not favour disease development. Additionally, Rashid and Kenaschuk (1998) recommend planting early to avoid warm weather and early infections. Early seeding was also recommended by Sackston (1951) as was using long season varieties since these had the greatest yield potential if they were planted at the right time. Rashid et al., (2001) have reported that later seeding and lower seeding rates reduced the incidence of pasmo significantly early on in the season as well as the

final pasmo severity. However, later seeding dates had lower yields than early seeded dates. Lower rates of nitrogen were also found to reduce disease severity, however the lower rates also reduced yield. Differences in results between the two studies could be accounted for by environmental factors.

Fungicides have been employed with varying rates of effectiveness in research trials but to date no fungicides have been registered for use in flax. Perryman et al. (1999) found that Benomyl was the most effective fungicide. Halley et al. (2004), tested multiple fungicides and concluded that azoxystrobin was the most effective fungicide. Mancozeb has also been shown to be effective as a disease control measure (Ferguson et al., 1987).

Rashid and Kenaschuk (1998) reported that two applications of fungicide was the most effective method of reducing disease severity and increasing yield. Ferguson et al. (1987), Perryman and Fitt (2000) and Perryman et al. (1999) reported that a single application of fungicide was almost as effective as multiple applications as long as it is applied around the time the plants were flowering, preferably at mid flowering.

Timing of fungicide seems to have an impact on yield, with application at mid flowering and mid flowering and late capsule development being found to reduce severity by 20% (Perryman et al. 1999). Perryman and Fitt (2000) have also observed increases in yield when fungicides are applied, particularly when June and July precipitation is high. Perryman and Fitt (2000) observed that fungicide applications reduce the browning of leaves and stems, and this in turn delayed senescence when compared to unsprayed plots. They also reported that leaf browning later in the growing season was strongly associated with yield loss. Additionally they observed that as the

season progressed there were smaller and smaller differences in disease severity on leaves between plots, however there was an increase in the difference in browning of the stems in plots treated with fungicides and those not treated with fungicides. Their study further suggested that there was a loss of 0.1 to 0.18 t/ha for each 10% increase in leaf browning. They recommended application of fungicide about one year in three or four, since the disease did not cause enough damage to justify the costs of the fungicide and application in the other years.

As noted by Sackston (1959), the use of fungicides to control pasmo in flax may not be a control method pursued by many producers since the severity of the disease varies from year to year (he reported it tended to be severe only one in five years). Additionally, no effective forecasting system has been developed to help farmers determine when fungicides might be warranted. The returns gained from the fungicide must also be high enough to justify application and fungicide costs, which often only occurs under severe infection conditions (Sackston, 1959; Rashid and Kenaschuk, 1998).

It has been noted by many researchers that wild *Linum* species can also act as alternate hosts for the disease. In New Zealand the introduced weed *Linum marginale* Cunn. was found to be highly susceptible to the pathogen and became a source of inoculum for commercial flax crops (Newhook, 1942; Lafferty and McKay, 1944). Lafferty and McKay (1944) observed infected plants of *L. angustifolium* in Eire, but were unable to find infected commercial fields. In Manitoba and Saskatchewan there have been suspected pasmo infections on *L. lewisii* Pursh. In greenhouse trials *L.*

austriacum L., *L. flavum* L., *L. grandiflorum* Desf., *L. perenne* L., *L. striatum* Walt., and *L. tenue* Desf. were also susceptible to pasmo (Sackston, 1949a).

2.4 DETECTION OF POLYMORPHISMS

2.4.1 Polymorphisms within Populations

There are many techniques that can be used to detect polymorphisms, or variations within the genetic code of a population. One option is to study morphological traits of the organism to elucidate variations. However, Johns et al. (1997) have noted that there is a limit on the number of morphological traits that can be used to study variability and the morphological and molecular data do not always agree well. They found that morphological data often obscured groups that were clearly seen with genetic data. Gupta et al. (1999) note that molecular markers do not have the limitations that are imposed by the use of morphological traits in studies of genetic variability.

A clonal population is assumed to have a limited number of polymorphisms within the genome while a population that is reproducing sexually is expected to have a higher number of polymorphisms, due to the exchanges of DNA that occur during sexual reproduction. Few reports of the sexual state of *Septoria linicola* have been made. Molecular work done by Verklay et al. (2004) backs up the genetic association between the anamorph and the teliomorph. The sexual states of some other Septoria species have recently been found, including *S. tritici*, (Hoorne, 2002).

2.4.2 Random Amplified Polymorphic DNA (RAPD)

This method is used for fingerprinting genomic DNA and is considered to be simple. A single short primer is used for the polymerase chain reaction for the Random Amplified Polymorphic DNA (RAPD) procedure. The primers for this technique must be selected such that they are complementary within a limited number of base pairs to sequences on the two opposite strands. Amplification of the DNA between these two sites occurs during PCR. Mutations at the site of attachment of the primer show up as polymorphisms as no amplification can occur, so a particular DNA fragment will not show up on the gel. This technique is useful because it is not necessary to know the sequence of the species to generate primers, so this makes it a less expensive technique. Additionally, a large number of fragments are generated with a single primer (Williams et al. 1990). RAPDs are useful in that they require only a small amount of DNA, they are easy to carry out and they are quick, which is beneficial when dealing with large populations (Haanstra et al. 1999). However, they are difficult to reproduce because there is a chance that the primer will randomly generate a product during PCR even if the primer is not specific (Penner at al. 1993).

2.4.3 Simple Sequence Repeat (SSR)

This technique produces markers that are mainly co-dominant. One of the downsides of the technique is that it takes a large amount of time and is expensive (Li and Quiros, 2001; Vos et al. 1995). SSRs are often used in genetic studies because they provide a large number of polymorphic DNA fragments while being relatively simple

(Plaschke et al. 1995; Huang et al. 2002). They also have the advantage of being very reproducible (Roder et al. 1998).

2.4.5 Amplified Fragment Length Polymorphisms (AFLP)

This technique is another PCR based technique used by many researchers for many applications including assessing differences within populations, among individuals and within species based on evolution (Mueller and Wolfenbarger, 1999). Vos et al. (1995) designed this marker to be used with no advance knowledge of the genetic sequence, and to work independent of template DNA amount as long as a minimal amount of DNA, 2.5 pg, is present. The technique uses a generic and limited set of primers. Selecting specific sets of primers allows the number of DNA fragments generated to be increased or decreased. Vos et al. (1995) cite the specific conditions required for annealing during PCR as being one of the characteristics that makes the marker reliable.

However, other researchers feel that the procedure is complex, and it is difficult to optimize the conditions for each individual step, as the procedure requires DNA digestion, ligation and amplification. A further complication arises if a methylation sensitive restriction enzyme is used on the methylated DNA, which can cause pseudo polymorphisms (Li and Quiros, 2001). Another limitation of the technique is that one of the restriction enzymes used, MseI, recognizes the AATT restriction sites, and this may cause uneven marker distribution within the genomes of some species (Haanstra et al. 1999). This technique is similar to RAPDs in that it is not necessary to know the DNA sequence of the organism you are using.

2.4.5 Random Fragment Length Polymorphism (RFLP)

This procedure involves the cutting of DNA by enzymes. The restriction enzymes cut the DNA when they recognize particular sequences. A mutation in the sequence will either cause it to be unrecognizable by the enzyme and it will not be cut, or the mutation may create additional cutting regions, creating a larger number of shorter fragments. This way the DNA fragment may be shorter or longer depending on where mutations arise and this will be detectable on a gel. The differences in the genetic code are also called polymorphisms, giving the name restriction fragment length polymorphisms. The use of more restriction enzymes produces more DNA fragments. RFLP's are successful when studying co-dominant markers, but it requires a large amount of DNA to act as a template for the reaction. However, it also is not able to show you more than a few loci in each reaction (Vandemark et al. 2006).

2.4.6 Sequence-related Amplified Ploymorphisim (SRAP)

The polymerase chain reaction (PCR) based technique known as SRAP has become a popular molecular tool for population studies as well as studies involved in gene mapping for breeding as well as gene tagging (Li and Quiros, 2001). SRAP was developed to amplify open reading frames. The technique uses two primers, a forward and a reverse primer. It has been used for construction of genetic linkage maps (Li and Quiros, 2001). SRAP has been noted to be more repeatable than RAPDs and faster than many of the other currently employed molecular markers (Li and Quiros, 2001). It has also been used to do genetic diversity studies by many researchers (Ferriol et al. 2003; Vandemark et al. 2006; Fernando et al. 2005). Ferriol et al. (2003) found that the SRAP

marker provided information that matched more closely with the known evolutionary history and the morphology of the organism than any other molecular marker they had tried. Shu-Jing et al. (2006) used SRAP to help establish the genealogical classification of medicinally important *Ganoderma* strains. SRAP was better at detecting variation in the genetic code among isolates of *Apiosporina morbosa* than ITS (Fernando et al. 2005). Fernando et al. (2005) also report that because the entire genome is sampled with SRAP, more polymorphic fragments are created than are seen with ITS. SRAP has been reported to generate as many polymorphic bands as AFLP (Vos et al. 1995; Li and Quiros, 2001). SRAP is less expensive than AFLP because it does not require the enzyme restriction, the pre-amplification step or the ligation of the primer (Fernando et al. 2005). SRAP markers were observed by Fufa et al. (2005) to provide an estimate of genetic diversity that was more conservative than that seen with SSR, and it was felt that SRAP had the potential for the identification of genotype and genetic diversity but in a different manner than SSR. Li et al. (2003) have hypothesized that genes with low levels of expression may be detected well by SRAP. One of the disadvantages of SRAP markers is that they may not be distributed randomly across the genome (Li and Quiros, 2001). One of the main differences between SSRs and SRAP is that SRAP amplifies many polymorphic and reproducible alleles and loci, while SSR markers identify individual multiallelic loci. This allows SRAP markers to be used more efficiently for gene mapping, diversity analysis and fingerprinting (Fufa et al. 2005).

3. INFERENCE OF THE SEXUAL STATE OF *SEPTORI LINICOLA* (SPEG.) GARASSINI IN MANITOBA

3.1 ABSTRACT

The fungus *Septoria linicola* (Speg.) Garassini is pathogenic on flax and has the ability to significantly reduce yield as well as other quality parameters. Pasmo is observed on an annual basis in commercial flax fields in Manitoba and Saskatchewan, with the incidence of the disease reaching 100% of sampled fields by September (Rashid et al. 2005, 2006, 2007). With the increasing importance of flax as both a functional food and for industrial uses there has been renewed interest in control measures to reduce the impact of the disease. The presence or absence of the sexual state in the province could impact control recommendations to producers as well as influence the direction of research into control measures.

The sexual state of the fungus has not been reported in Canada, thus our objectives were to gain information about the genetic structure of the population in order to infer the reproductive mode, sexual or asexual, from two geographically separated populations of *Septoria linicola* (Speg.) Garassini in Manitoba, Canada.

The molecular method Random Amplified Polymorphic DNA (RAPD) was used to gain information about the two populations. From these two populations, 163 isolates were used in DNA extraction and subsequent PCR reactions. Within this population, four stations were chosen from which a larger number of single spore isolates were generated. In total 128 polymorphic DNA fragments were scored from those generated

from six selected primers. These polymorphic fragments were used for building a Maximum Parsimony tree and for statistical analysis.

The results from the Analysis of Molecular Variance (AMOVA) indicated that 88 % of the variability seen was due to within population variability, and 12 % was due to among population variability. The AMOVA comparing the four smaller subpopulations from each location indicated that in both cases the two subpopulations from the same location were subdivided, there was limited gene flow between them. In contrast, comparisons of the subpopulations between locations indicated that the populations were not subdivided, that there was significant gene flow between locations. A single most parsimonious tree was generated from 100 bootstraps. The branching pattern within the tree showed that the locations were not grouping separately.

It is highly plausible that sexual recombination is occurring in Manitoba. The levels of variability within and between populations, in addition to the structure of the Maximum Parsomony tree all suggest that the population in Manitoba is in fact one large population, and not many individual subdivided clonally reproducing populations. Based on these results, it is highly plausible that sexual recombination is occurring in Manitoba, Canada. It is likely that within the growing season asexual reproduction is the main mode of reproduction, with sexual reproduction occurring in the spring or during the growing season, and contributing to long range dispersal in the spring.

3.2 INTRODUCTION

Pasmo disease of flax, caused by *Septoria linicola* (Speg.) Garassini, has been known to occur in North America since 1919 (Brentzel, 1926). It has been present in Manitoba since 1940 (Sackston, 1946). In the last 6 years, pasmo was found in all commercial flax fields sampled in September in Manitoba and Saskatchewan (Rashid et al. 2002, 2003, 2004, 2005, 2006, 2007). There are no reported races of the fungus, nor does there appear to be any significant variation in the virulence of the isolates in the province of Manitoba. In order to better understand the pathogen and the control options, it is important to attempt to determine the mating status of the fungus. In most cases fungicides, plant breeding, and cultural methods, or a combination of all three are pursued in order to control the pathogen. Breeding of resistant or tolerant lines is being pursued and is preferred over fungicide use due to the environmental impacts of pesticide use. The apparent lack of variability in the virulence of the fungus, and the apparent lack of highly resistant flax genotypes (Hannah, 1953; Sackston, 1959) will most likely lead to the search for tolerance. If the pathogen is reproducing sexually, cultural controls, such as rotations and field sanitation, while still important, will be less effective in controlling the disease.

To date, the sexual state of the pathogen has not been confirmed in Canada. Sackston (1949a) reported that he found structures that might represent the sexual state on flax straw in Manitoba. He was not able, however, to confirm that these were in fact *S. linicola* (Speg.) Garassini. The objective of the current study was to use the PCR-based molecular method known as Random Amplified Polymorphic DNA (RAPD) to

infer the presence of sexual reproduction in the fungal population in Manitoba, based on the level of genetic polymorphisms within the DNA of two separate sample populations of the fungus.

3.3 MATERIALS AND METHODS

3.3.1 Plant Sampling

Plant samples, with evidence of pasmo, were collected from two commercial flax fields in two different areas in Manitoba. One field was near Portage la Prairie and the other was near Sanford. The distance between the fields was approximately 90 km. Thirty six sampling stations were used in each field. The sampling stations were 50 m away from each other in every direction (Appendix 1). At each station, about 40 plants were removed from a 1 m^2 sampling area within the crop in a random sampling pattern and placed into labelled paper bags. The dry samples were stored in sealed plastic containers at 4°C until they were processed.

3.3.2 Production of Fungal Material

Individual plant stems were cut in half lengthwise and cut into 2 cm lengths. Samples were surface sterilized using 0.5 % Javex ™ (NaOCl with an initial concentration of 5%) and then rinsed three times for one minute with 30 ml of autoclaved distilled water. They were then incubated in the dark on moist filter paper in sterilized glass Petri plates at 20 °C until pycnidia on the stem pieces released pycnidiospores (usually between 4 and 7 hours). Pycnidiospores from individual pycnidia were then placed on plates of yeast malt agar (YMA) using a sterilized needle and allowed to incubate in the dark at 20 °C until colonies developed and produced spores. The spores were then streaked onto fresh YMA plates and again allowed to incubate until colonies were formed and spores were produced. The spores from these

cultures were then streaked on water agar plates and single spores were isolated from these plates using a compound microscope and a needle. The single spores were grown to sporulation on new YMA plates at 20°C in the dark. Once sporulation occurred, the plates were flooded with 5 ml of water to obtain a spore suspension. Flasks containing 80 ml of sterilized Yeast Sucrose liquid medium were inoculated with 400 µl of the spore suspension, and sealed with foam plugs. These developing cultures were then incubated for 7 days at room temperature on a shaker set at 150 RPM. Following this incubation period, the fungal material was harvested from the liquid medium by spinning the contents of the flasks in a Centra Cl2 centrifuge (Thermo IEC, Needham Heights, MA, USA) at 12000 g for 10 minutes. The resulting material was collected in micro-centrifuge tubes and frozen in liquid nitrogen. The material was then lyophilized using a Freeze Dryer 8, (Labconco Corporation, Kansas City, Missouri, USA) and stored at -20°C until used for DNA extraction. For all stations, at least two stems were randomly selected for pycnidiospore isolation. From the cultures generated, one culture was carried forward from each stem piece for single spore culturing. Four stations were randomly selected from which a larger number of isolates were generated. These stations were P-5-3 (14 isolates), P-2-3 (16 isolates), S-3-2 (22 isolates) and S-6-5 (10 isolates) and were chosen to test for clonality "within rainsplash distance" (Appendix 1).

3.3.3 DNA Extraction

DNA extractions were carried out using the Wizard® genomic DNA purification kit (Promega, Madison, Wisconsin, USA). The extraction protocol used

was that provided by the manufacturer with the addition of a phenol chloroform cleaning step at the end of the extraction. The DNA was then quantified using an Ultrospec 2100 pro (Biochrome Ltd., Cambridge, England) and 25% of the samples were run on 0.8% agarose gels stained with ethidium bromide to confirm the results of the spectrophotometric quantification and to examine the quality of the DNA. The quantified DNA was then stored in TE (10mM Tris Cl and 1mM EDTA) buffer at a pH of 7.4 in microcentrifuge tubes at -20°C until needed. Standardized concentrations of 3.5 ng of DNA/µl of distilled water were made up using the DNA concentration values obtained during quantification. The concentrated DNA solution was added to the appropriate amount of distilled autoclaved water for use in amplification. DNA extraction was carried out on 191 isolates for use in RAPD screening, 101 from Sanford and 90 from Portage la Prairie (Appendix 1). Of these isolates, 78 from Sanford and 85 from Portage la Prairie were used to generate a consensus tree and to calculate the values for AMOVA. Twenty eight isolates that did not amplify with one or more primers were removed from the data set for analysis and tree building, resulting in 163 isolates being used in the analysis, as missing data may affect the reliability of results for these two procedures.

3.3.4 Amplification of DNA

PCR reactions contained 1.61 mM of Tris pH 8.4 and 40.29 mM Potassium Chloride (10x TAQ polymerase reaction buffer (Invitrogen, California, USA)), 0.97 mM Mg^{2+} (Invitrogen), 0.097 mM of each of the dNTP's A, C, G, and T (Invitrogen), 5 units/µl Taq polymerase (Invitrogen), 0.388 pmole of primer (Alpha DNA, Quebec,

Canada) , 7 ng of template DNA, and 6.988 µl of HPLC grade water, for a total volume of 10.3 µl per reaction. The rection volume was determined by recommendations of the manufacturer of the PCR plates due to well size. Primer sequences (Table 3.1) were obtained from the UBC website from the NAPs Unit standard Primers (http://www.michaelsmith.ubc.ca/services/NAPS/Primer_sets). Six primers were chosen based on a high degree of variability between isolates during preliminary screening with eight randomly chosen isolates, four from each site.

PCR conditions were the following: five minutes at 94°C for denaturing, then 40 cycles of 30 seconds at 95°, one minute at 34°C for annealing and 1 minute and thirty seconds at 72°C for extension. The annealing temperature was chosen during the primer screening phase.

The PCR products were then electrophoresed in 1.5 % agarose gels made with TAE (0.04 M Tris Acetate and 1 mM EDTA) buffer containing 0.35ng/ml of ethidium bromide. One kilobase and 100 base pair ladders were run on all gels to aid in scoring of bands on the gels. Gel images (Figure 3.1) were captured digitally using an Alphaimager HP (AlphaInnotech, California, USA) and were stored electronically for analysis.

Table 3.1. RAPD Primer sequences used in amplification of selected isolates of *Septoria linicola.*

Primer	Sequence (5'-3')	GC content (%)
UBC522	TCG TCT AGC A	50
UBC536	GCC CCT CGT C	80
UBC608	GAG CCC GAA A	60
UBC634	CCG TAC ACG C	70
UBC676	GCT AAC GTC C	60
UBC681	CCC CCG GAC T	80

3.3.5 Data Collection and Analysis

Gels were scored visually by assigning a value of one (presence) or zero (absence) for all bands for a given primer for each isolate (Appendix 3). A visual threshold was determined for exclusion of gels. In order to increase the in-lab reliability of the bands used in scoring the RAPDs, 60 isolates were randomly selected for re-extraction and were then used in a new PCR reaction. The bands generated in the second reactions were compared to the bands generated when the entire population was used and only those that appeared in both reactions were used. In total 128 reproducible polymorphic DNA fragments from 163 isolates were used for tree building and Analysis of Molecular Variance (AMOVA) analysis. The program GenAlEx (Peakall and Smouse, 2006) was used to perform AMOVA as well as to calculate genetic distances and genetic identities. Analysis of Molecular Variance was also run on the four sub-populations generated from stations P-5-3, P-2-3, S-3-2 and S-6-5. Maximum Parsimony was performed in PAUP 4.0 (Swofford, 2003) using 100 bootstrap replicates to generate a phylogenetic tree with the binary data. A heuristic search was done with stepwise addition for branch swapping. The tree bisection reconnection algorithm was

used to swap the branches. The tree was unrooted. A single most parsimonious tree resulted. A tree was also generated using the Neighbour Joining program but is not presented here as it was similar to the Maximum Parsimony tree for most isolates.

3.4 RESULTS

3.4.1 Analysis of Entire Population using AMOVA

In total 128 polymorphic bands scored from the PCR products generated from 163 isolates from two locations. The results obtained from the AMOVA calculations for the entire population performed by GenAlEx (Peakall and Smouse, 2006) are outlined in Table 3.2.

Table 3.2. Analysis of Molecular Variance (AMOVA) for 163 single spore isolates of *Septori linocola* [1].

Source of Variation	df	Sum of Squares	Estimate of Variability
Among Populations	1	199.544	12%
Within Populations	161	2735.401	88%
Total	162	2934.945	

[1] AMOVA measured variance among groups at two sites. Significance level for the data set was P> 0.001.

As is seen in the Table 3.2 only 12% of the variability in the population is attributable to variations between the populations, the remainder (88%) is within population variability. A low PhiPT value of 0.117 sugests that the subdivision between the two populations is minimal.

3.4.2 Genetic Distances and Identities of the Entire Population

Genetic distances and genetic identities were also calculated for the two populations (Tables 3.3 and 3.4 respectively) following the procedures developed by Nei (1972, 1978).

Genetic distance values indicate how different a population is from a larger population. It is a measure of the number of genes that have changed in the population or populations under study from an original theoretical population (Avise, 2004).

Table 3.3. Pairwise population matrix of Nei genetic distance and unbiased genetic distance for two populations of *Septoria linicola* from Portage la Prairie and Sanford, Manitoba, Canada.

Population	Portage la Prairie	Sanford	Portage la Prairie	Sanford
	Genetic distance		Unbiased genetic distance	
Portage la Prairie	0.0		0.0	
Sanford	0.053	0.00	0.049	0.0

The results given in Table 3.3 indicate that the proportion of genes that have changed in the two populations relative to the larger population have been small, as the numbers are close to zero.

Genetic identity indicates how genetically similar a sub-population is when compared to a larger population. It indicates whether or not a sub-population is in fact part of a larger population or if it should be considered to be a genetically distinct population based on the number of similar genes in the populations (Avise, 2004).

Table 3.4. Pairwise population matrix of Nei genetic identity and unbiased genetic identity for two populations of *Septoria linicola* from Portage la Prairie and Sanford, Manitoba, Canada.

Population	Portage la Prairie	Sanford	Portage la Prairie	Sanford
	Genetic identity		Unbiased genetic identity	
Portage la Prairie	1.0		1.0	
Sanford	0.948	1.0	0.952	1.0

The results given in Table 3.4 show that the genetic identity values of the Sanford and Portage populations are close to one, suggesting that the two populations are genetically similar to the larger population.

3.4.3 Analysis of Four Sub-Populations using AMOVA

Four sub-populations where generated, two from each location, to test for variability within a small area. In total 62 isolates were analyzed. When AMOVA was run on the subset of four subpopulations from the two locations, the varibiltity within populations was again large (Table 3.5).

Table 3.5. Analysis of Molecular Variance (AMOVA) for 62 single spore isolates of *Septoria linocola* made up of four sub-populations [1].

Source	df	Sum of Squares	Estimate of Variability (%)
Among Regions	1	84.80	10
Among Populations	2	50.62	3
Within Populations	58	980.81	87
Total	61	1116.23	100

[1] AMOVA measured variance among groups at two sites, with two sub-populations being used from each site. Significance level for the data set was P> 0.001.

When the isolates from the four smaller sub-populations were analyzed, the within-population variation was 87 %, while the among region variability was 10 %. The variability accounted for among populations was only 3%. The PhiPT value was 0.127 for the analysis, suggesting that the populations were not significantly different from each other.

3.4.4 Pairwise Population Analysis of Four Sub-Populations

Pairwise population analysis was run on the 62 isolates from the four sub-populations to determine the level of similarity of these populations to each other.

Table 3.6. Pairwise Population Analysis of 62 isolates of *Septoria linicola* made up of four sub-populations.[1]

Populations compared	PhiPT	P value
P-5-3 and P-2-3	0.034	0.05
P-5-3 and S-3-2	0.111	0.001
P-2-3 and S-3-2	0.115	0.001
P-5-3 and S-6-5	0.158	0.001
P-2-3 and S-6-5	0.153	0.002
S-3-2 and S-6-5	0.033	0.078

[1]The four sub-populations originated from two locations, those designated as P isolates originated from the Portage la Prairie location and those designated as S originated from the Sanford location. Isolate P-5-3 contained 14 isolates, population P-2-3 contained 16 isolates, population S-3-2 contained 22 isolates and population S-6-5 contained 10 isolates.

Based on the data shown in Table 3.6 we can see that comparisons of ssub-populations within locations (Sanford and Portage la Prairie) gave PhiPT values that were below 0.05, which indicated the populations were nearly genetically identical, there was little variability between them. The higher PhiPT values seen for comparisons

of populations between the two locations indicated that there was a higher level of variability between these populations (Figure 3.6).

3.4.5 Agarose Gels

A typical example of polymorphic bands generated by random amplified polymorphic DNA in the current study is given below.

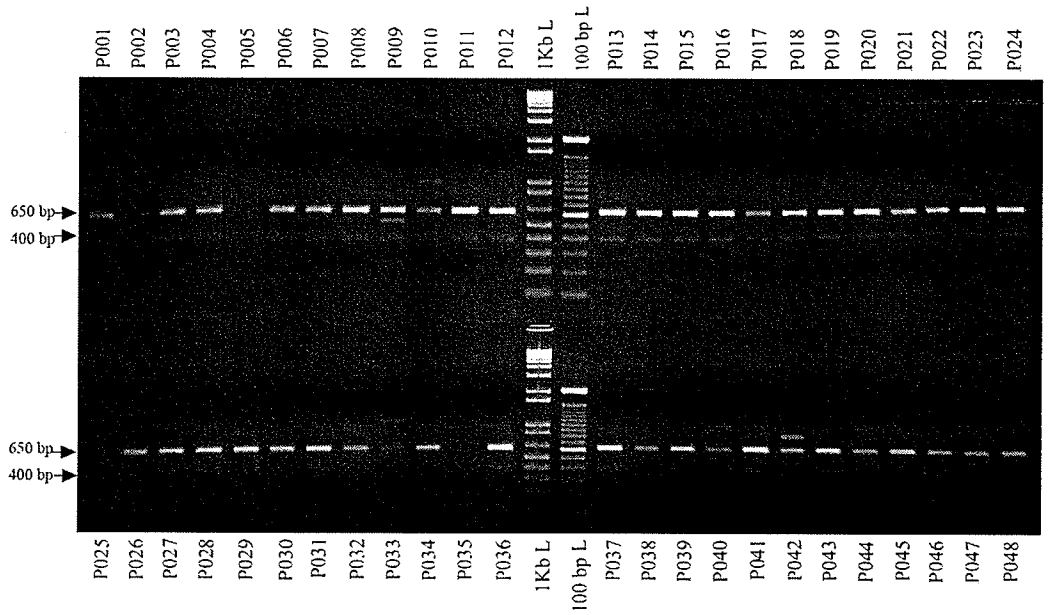
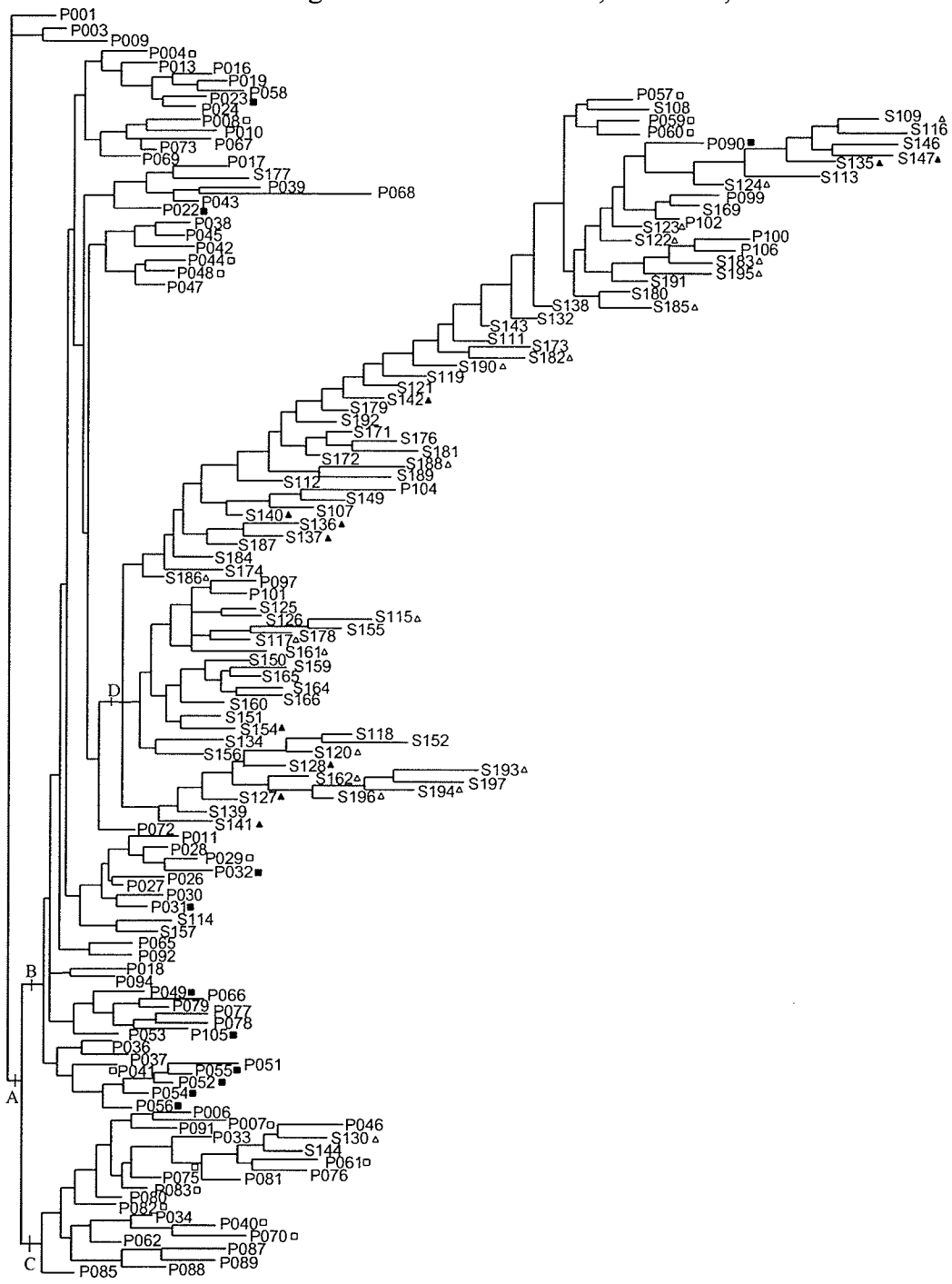


Figure 3.1. Agarose gel depicting polymorphic loci generated using 48 isolates from the Portage la Prairie, Manitoba, Canada site with RAPD Primer UBC522.

3.4.6 Phylogenetic Tree

The phylogenetic tree generated with the PAUP 4.0 program (Swofford, 2003) was created using Maximum Parsimony. In total 163 isolates were used, giving a large number of branchings. Branches were broken into 4 clades.

Figure 3.2. Phylogenetic tree generated by PAUP 4.0 program showing relationships between isolates from Portage la Prairie and Sanford, Manitoba, Canada*, [1].



* Isolates beginning with P are from the Portage la Prairie site and those beginning with S and from the Sanford site.

[1]Isolates with a Δ are from station S-3-2, while those with a ▲ are from station S-6-5; isolates with a ■ after the number are from station P-2-3 and those with a □ following the number are from station P-5-3. Letters A, B, C and D designate clades within the tree.

The tree generated by the PAUP program 4.0 (Swofford, 2003) shows that the isolates from the two locations are intermingled, with no locational division (Figure 3.2). Clade A is comprised exclusively of isolates from the Portage la Prairie location and clade C mostly of isolates from the Portage la Prairie location as well, with the exception of isolates S130 and S144 (Figure 3.2). Clade D contained a branch at the bottom that consisted exclusively of isolates from the Sanford area, but the other two branches contained a combination of Portage la Prairie and Sanford isolates (Figure 3.2). Clade B, encompassing a large number of branches, included branches that were exlusively from Portage la Prairie (the topmost and bottom most branches), with most of the other branches being more mixed (Figure 3.2). Clade C contained a number of isolates from station S-3-2, but these were not all on the same branch. The bottom most branch of clade B contained a large number of isolates from station P-2-3 which are branching together.Within clade D one of the topmost braches included three isolates from the P-5-3 station (Figure 3.2).

3.5 DISCUSSION

3.5.1 Life Cycle of the Septoria linocola *Population in Manitoba*

The genetic structure of the *S. linicola* population in Manitoba is not known. To date the sexual state of the fungus has not been conclusively demonstrated in the province (Sackston, 1949a).

The proposed life cycle, based on the results of this study, consists of both the asexual cycle as outlined above as well as the production of pseudothecia or the sexual reproductive structures. Pseudothecia production is most likely occuring primarily in the fall, as is seen with *Mycosphaerella graminicola* (Shaw and Royle, 1989), as an additional source of overwintering inoculum available in the spring. One of the benefits of sexual reproduction to the pathogen is that the type of sexual spores for *S. linicola* reported by Wollenbeber (1938) from flax samples obtained from Argentina are thought to be much more amenable to longer distance travel than are the asexual spores.

If the fungus was reproducing solely in an asexual manner, the life cycle would consist of either overwintered pycnidiospores (on stubble or straw) or pycnidiospores on seeds that are then able to infect the seedling (infected seeds) or leaves of the young plant in the spring. Since the asexual spores are reliant on rain-splash and possibly wind for dispersal, they are only able to infect plants close to the site of release of the spores (the overwintered pycnidiospores or infected seedlings). Once infection occurres and the fungus reaches maturity it will produce new pycnidia which will act as a secondary source of inoculum throughout the season and will also be able to overwinter to serve as a source of inoculum in the spring (Sackston, 1949a).

3.5.2 Reproduction and Local Dispersal

Overall in the populations studied, there was not a distinct separation of isolates based on location or station from which they were sampled. In the two populations studied there was a tendency towards smaller groupings based on geographic sites, such as clade A and some branches in clade B, suggesting local dispersal during the growing season. Within the clades, with the exception of clade A, isolates from the other population were often interspersed on many of the branches. Some groups within these larger branchings have short branch lengths, indicating genetic similarity, which is expected due to the large numbers of pycnidia being produced over the growing season. If there were no sexual reproduction the expectation would be that the two populations sampled would have large numbers of identical isolates.

Even in a mating population, there would be a certain level of similarity within sites, since clonal reproduction is also occurring. Clonal reproduction seems to be occurring quite frequently during the summer due to it being rapid and not requiring any other individual to occur. This has been shown to be the case for *Mycosphaerella graminicola* (*Septoria tritici)* by Eriksen et al. (2001).

A pair of populations that are reproducing exclusively clonally and that have a minimal amount of genetic exchange would be expected to segregate into two very distinct groups. If clonal reproduction was occurring exclusively, it would be expected that a large number of genetically identical isolates would be seen (Avise, 2004). The pycnidia, or the asexual reproductive bodies are known to occur here (Sackston, 1949a). To dated no confirmed reports have been made of the sexual state in Manitoba.

3.5.3 Genetic Diversity

The current study employed a number of individuals from two areas of Manitoba in order to provide as much information about the genetic diversity in the population as possible. Based on the results obtained from both the consensus tree building and AMOVA analysis, it is highly plausible that there is sexual recombination occurring within the *S. linicola* populations sampled. A within-population variability estimate of 88% suggests that there is genetic exchange occurring within the population or that there are a high number of isolates that are being moved from one site to another. It is possible that mutations or the long range movement of the pathogen could be contributing to the variability within the populations, but it seems unlikely that these factors could be occurring with a high enough frequency to account entirely for the observed values.

Comparisons of two sub-populations within each location (Sanford and Portage la Prairie) indicated the populations were nearly genetically identical within locations, there was little variability between them. This means that there is genetic exchange occurring within this population. The higher PhiPT values seen for comparisons of populations between the two locations indicate that there is a higher level of variability between locations, but the populations are not acting like isolated populations, which is what a PhipT value of one would indicates.

If a population is clonal, mutations would be expected, but it seems doubtful that the random mutations would reach such high numbers as were observed here. Joseph and Hall (2004) reported that the haploid mutation rate was around $6.3x10^{-5}$ mutations per haploid genome per generation in yeast. Based on the fact that the fungus has only

been observed in Manitoba since 1940 (Sackston, 1946), if the population were mutating at a similar rate, and was only reproducing asexually, the contribution to the level of polymorphism would be small, and would not account for the differences seen.

Hoorne (2002) studied two populations of *S. tritici* in Manitoba with isolates collected in a similar manner to that done here, with the establishment of sampling stations and collection of pycnidiospores from individual pycnidia to generate a collection of single spore isolates. The fields in that study were 200 km apart. Fourty four isolates were used to compare two locations. Amplified fragment length polymorphisms were used to compare the levels of polymorphism between and within the two populations. Hoorne (2002) found that almost all of the isolates were genetically different and that the populations were, as in this study, part of one larger population, and that within populations there was a large amount of variability. The phylogenetic trees generated also show a similar trend of large numbers of branches and dispersal of isolates from both locations with small groupings from individual locations.

3.5.4 Dispersal

Studies conducted on spore movement within fields have shown that it is very difficult to move the asexual spores over long distances without the aid of rain splash and wind (Sackston, 1949a). As a result the long distance dispersal of the asexual spores is likely dependant on the movement of infected material such as leaf bits and straw or on infected seed. For seed transmission to have a large impact, the seed lot would have to be severely infected in order to have a large enough number of individual isolates moving into new distant fields. Sackston (1949a) found that out of 1000

contaminated seeds planted only one of them developed cotyledon lesions. If this is representative of typical field conditions, the introduction of new isolates into a field would only occur at a relatively low rate. If seed transmission had a significant impact the expectation would be to see limited differences in the isolates if only asexual reproduction were occurring, and the small number of genotypes should be present in most fields seeded with that seed. If, however, seed from multiple sources were mixed and planted into fields we might expect to see a higher number of groups of identical isolates.

Heavily infected seed does not qualify for certification, so it is unlikely that a farmer would be in a position to purchase such a heavily infected seed lot. Low levels of infected seed could be expected based on the prevalence of the disease in Manitoba (Rashid et al. 2005, 2006, 2007) however, it is difficult to asses their contribution to the variability seen in the sampled populations, as the seed was not assessed prior to planting.

The asexual spores are most effective at short range dispersal, and since flax is rarely grown after flax on the same field, it would be more difficult for a population reproducing solely by asexual means to effectively maintain and disperse a large number of highly genetically different isolates solely through seed transmission. The existence of sexual spores for *S. linicola* could have led to more genetic variability in the populations sampled, as well as to more genetic exchange between these populations. This is consistent with the parsimony tree, because of the ability of the ascospores to move over long distances and mate with genetically different individuals. This is also consistant with the PhiPT values measured for both the larger population

and the sub-populations indicating that the two populations were in fact part of one larger population.

The wind dispersal of small bits of leaf material from stubble could potentially play a role in dispersal of the pathogen, but has never been proven to be an effective dispersal method. Since many farmers remove their flax straw or chop it and spread it on the field, because of its resistance to decomposition, the amount available for easy dispersal is likely small. These bits of straw and leaves would have to avoid decomposition and then have to be blown into new fields, where the flax was being planted. While it is possible that a small contribution is made by debris dispersal, we would still expect a small number of genetically different isolates if only clonal reproduction was occurring. Therefore there would still be fields composed largely of the same clonal isolate. The likelihood of all of the conditions being met for debris dispersal and subsequent infection is low. In addition, if the mode of reproduction in a field is exclusively clonal, there will be low contributions to genetic diversity made by the propagules on the debris.

It has been suggested that farmers themselves could be spreading the disease and this could potentially be the case. It is, however, somewhat unlikely as most farmers are careful not to move in wet fields due to the risk of spreading diseases within and between fields. Farmers are generally careful not to move within heavily infected fields and subsequently visit other fields of the same crop on the same day. Most farmers practice field sanitation and clean machinery between fields, which also reduces the transmission of inoculum between fields.

There are many modes of possible transmission of fungal isolates between sites. For *S. linicola*, the asexual spores, which are known to be present in Manitoba, are able to move only within a limited distance from their origin due to their reliance on transmission via rain and wind, on infected seed or bits of tissue mixed in with seed. Their ability to over-winter on straw is beneficial only if the straw is placed close enough to new plants that the spores can find new hosts. The sexual state of the fungus, conversely, has the ability to travel over longer distances due to the nature of the spores, which are wind-borne. The results of this study suggest that the sexual state of the fungus exists in Manitoba, based on the variability seen within two sampled populations. The amount of genetic variability calculated by AMOVA, as well as the consensus tree results, suggest that gene exchange is occurring between these populations, most likely through mating, with contributions being made via the introduction of unique isolates to the fields each season through wind, infected seeds or tissue bits.

3.6 CONCLUSION

The phylogenetic and statistical evidence suggest that it is plausible that the sexual state of *S. linicola* exists in Manitoba. This is the first report of the inference of the sexual state of S. linicola in Manitoba. It is probable that the sexual state is also present in the other flax growing areas in Canada as well as in the United States. Few reports have been made of the physical existence of the sexual structures (Wollenbeber, 1938), and none have been confirmed in Manitoba (Sackston, 1949a). The presence of the sexual state of the fungus has implications for both control recommendations made to farmers as well as attempts to breed resistant cultivars. The presence of the sexual state means farmers have less control over the initial inoculum levels in their fields and have to rely on other cultural control methods. The presence of the sexual state also has implications for the longevity of resistant lines that may be developed in the near future.

4. EFFECTS OF PASMO ON FLAX

4.1 ABSTRACT

Septoria linicola (Speg) Garassini (teliomorph *Mycosphaerella linorum* Naumov) is the causal agent of pasmo disease of flax (*Linum usitatissimum* L.). Pasmo has been reported to cause yield losses and affect oil and protein content of the seed (Rashid and Kenaschuk, 1998; Sackston, 1949a). To date no good source of resistance has been found (Hannah, 1953; Sackston, 1959). Six flax cultivars (AC Emerson, AC Linora, AC Macbeth, McGregor, Norlin and Vimy) were studied to determine their response to fungicide protection from pasmo disease under field conditions. The response characteristics studied included seed yield, oil and protein content of the seed under disease pressure with and without the aid of a fungicide. Infected straw was used to introduce the disease into the plots and two to five applications of Headline ™ (Pyraclostrobin) were used to control the disease in the fungicide treated plots. The use of fungicide produced marked decreases in Area Under the Disease Progress Curve (AUDPC) values for all cultivars. Application of the fungicide provided significant increases in yield, with the exception of AC Macbeth in Winnipeg in 2004. Yield increases were especially high in the absence of added inoculum, with many cultivars having nearly twice the yield as the control. Seed oil content was significantly higher in the fungicide application treatments compared to the control for all cultivars except Norlin in 2003. Seed protein contents were significantly better with fungicide application for all cultivars in Winnipeg in 2003, but the same was not true for the

Morden site in the same year. In 2004 almost all cultivars did significantly worse with the application of fungicides compared to the control in Winnipeg, while at the Morden site treatments had little significant effect. The 1000 kernel weight tended to be significantly higher with fungicide application over all years and at all sites.

4.2 INTRODUCTION

Flax has been grown in Western Canada since approximately 1875 (Lehberg and Anderson, 1941). The area seeded to flax in Canada is estimated to be approximately 565 thousand hectares in 2008 (Agriculture and Agri-Food Canada, 2008). Due to the increasing importance of flax as a functional food in the North American market (Vaisey-Genser and Morris, 2003) the flax acreage is likely to remain stable or increase over the next few years.

Pasmo has been observed in Canada since 1939 (Sackston, 1947b). In each of the last seven years pasmo has been found in commercial fields in Manitoba and Saskatchewan, ranging in incidence from 58 % to 96 % of surveyed fields, with most fields surveyed late in the season (late August and September) having incidences of 100% (Rashid et al. 2001, 2002, 2003, 2004, 2005, 2006, 2007). Pasmo has been reported to reduce yields (Sackston 1947a, 1951) as well as oil and protein content in flax seed (Pederson and Michaelson, 1960). Rashid and Kenaschuk (1998) observed a 20% reduction in yield of flax under moderate infection. Due to the stable acreages being planted to flax and the potential decreases in yield that can result from the disease, this study attempted to determine the impact of fungicide use, as well as the impact of the disease on six flax cultivars. The ability of the six selected cultivars to maintain the yield, oil and protein content of the flaxseed produced under different disease pressure conditions was examined.

4.3 MATERIALS AND METHODS

4.3.1 Experimental Design

This study compared the yield, oil and protein contents of six flax cultivars with and without disease pressure and fungicide applications. The experiment was carried out over two years at two sites in Manitoba, at the Agricultural and Agri-Food Canada Research Station at Morden, and at the University of Manitoba Research Station at Winnipeg. The cultivars used were AC Emerson, McGregor, AC Macbeth, Norlin, AC Linora and Vimy, which were selected for their perceived differences in their reaction to the fungus (K. Rashid, unpublished data).

The experiment was designed as a split plot in 2003, with the design being improved to a split plot with a 2 by 2 factorial set of treatments applied to main plots in 2004. In 2003, the treatments consisted of inoculum with no fungicide application and fungicide application with no inoculum. In 2004 the main treatments consisted of inoculum or no inoculum, with the secondary treatment being the application or lack of application of a fungicide, producing a total of four treatments. The treatments were replicated four times with the cultivars being randomly assigned a plot within each replication. At the Morden site, four rows of each cultivar were planted in each plot with a row spacing of 30 cm. Flax plots were grown within a larger block of other flax trials in both years at the Morden site. At the Winnipeg site, six rows of each cultivar were planted in each plot with a row spacing of 25 cm. Corn was planted on all sides of the trial in both years. At the Morden site one plot consisting of four rows of sunflowers in each replicate was used as a physical barrier between inoculated and uninoculated

treatments, while at the Winnipeg site one plot consisting of four rows of corn was used as a barrier. Because of extremely poor emergence in the first seeding of the Winnipeg trial in 2004 the trial was reseeded on June 10th. The Morden trial was seeded in mid-May.

The inoculum used to induce the disease consisted of one year old naturally pasmo infected straw that was harvested, baled and stored outside as this has been used as a source of inoculum by other researchers (Brentzel, 1926; Flor, 1943; Sackston, 1949a, 1970; Rashid, 2003; Halley et al., 2004). The straw was placed between the rows of flax when the flax plants were 20 to 25 centimeters in height and prior to flowering. A misting system was used to generate high humidity conditions in the crop canopy, which favours the development of the disease (Rashid, 2003). The system was not used, however, when it was raining. In 2003, at the Morden site the straw was placed between the rows on June 23rd, and at the Winnipeg site on July 3rd. In 2004, straw placement occurred on July 7th at the Morden site, and on July 20th at the Winnipeg site. Three bales were used per site in 2003 and six bales were used per site in 2004. A larger number of bales was required in 2004 because the number of plots requiring straw application had doubled.

In 2003, the treatments were: i) inoculum with no fungicide application (I/NF) and ii) no inoculum with fungicide application (NI/F), which was to serve as the control. In 2003, the trial at Morden was misted from July 11 to July 31 while at Winnipeg misting occurred from July 20 to August 3. The misting occurred for 5 minutes every half hour from approximately 4:30 pm until 8:30 am. The NI/F treatment was sprayed with Headline ™ (Pyraclostrobin) fungicide (BASF Canada) every 10 days

to prevent the disease. Spraying occurred between July 11 and August 29 at the Morden site and between July 18 and September 3 at the Winnipeg site for a total of 5 applications per site. The centre two rows of each plot were sprayed using a backpack sprayer in Morden and a CO_2 pressurized backpack sprayer in Winnipeg, with the total volume of fungicide applied being identical in both sites. The fungicide was applied five times giving a final rate of 1.385 kg Active Ingredient/acre.

In 2004, an inoculated and treated with fungicide (I/F) and non-inoculated with no fungicide (NI/NF) treatment were added in addition to the treatments employed in the previous year (I/NF and NI/F). The trial was again carried out at the Morden and Winnipeg sites with the misting system being used in the same manner as it was in 2003. In Morden the plots were misted from July 13 to July 22, for a total of 10 days of misting. In Winnipeg misting was carried out from July 20 to July 27, for a total of 7 days of misting. The misting period in Winnipeg was shortened because of prolonged rainfall and the resulting high humidity conditions, which rendered the misting system redundant. Fungicide application methods were the same as in 2003, however only 2 fungicide applications were made at each site to give a final rate of 0.562 kg AI /acre in 2004. At the Morden site the applications were made on July 28 and August 12, while at the Winnipeg site they were made on August 14 and 28.

4.3.2 Disease Evaluation

Cultivars were rated every week for severity and incidence of the disease using all the plants in the centre two rows of each plot to give a rating. Severity was rated on a

scale of 1 to 9 according to criteria set out in Table 4.1 (K. Y. Rashid, unpublished data). Incidence was based on the percentage of plants infected in the centre two rows with the number ranging between 1 and 100.

Table 4.1. Severity rating descriptions for pasmo on flax used in the current study.

Rating	Symptoms
1	No disease.
2	One or two very small lesions on a small number of leaves, no or a very limited number of lesions on stems.
3	Small lesions on a small number of leaves, no lesions or very few on stems.
4	Lesions on a moderate number of leaves, small lesions on most stems.
5	Medium sized lesions on more than half of the leaves, less than half of the stem covered in lesions.
6	Large lesions with chlorotic halos on more than half of the leaves, half of the stem being covered in lesions.
7	Large lesions on almost all of the leaves, some of the leaves being dead and brown, leaves being lost, large portions of the stem covered in lesions, minor lodging of plants.
8	All of the leaves infected with large lesions and many being brown and dead, loss of dead leaves is obvious, most of the stems covered by lesions, moderate lodging of plants
9	All leaves dead, stems mostly defoliated, no green patches left on the stems, many of them turned grey and severe lodging of the stems

4.3.3 Quality Evaluation

The centre two rows of each plot were harvested using a 2 row cutter model # G510L (Mitsubishi, Shimane, Japan) in Morden or by hand with a sickle in Winnipeg and were then bagged and placed in a drying room at 27 °C for 2-5 days. The bundles were threshed using a Nursery Master Combine unit # 4 (Wintersteiger, Saskatchewan,

Canada) in Morden, and a stationary Hage thresher in Winnipeg. Seed samples were cleaned using a Clipper seed and grain conditioner Model #F80003540 using a number eight screen (Blufton Agr/Industrial Corp., Indiana, United States of America). Cleaned samples were weighed and 100 seeds were counted by hand. The 100-seed sample weights were used to generate a 1000 seed weight. Oil and protein contents of the cleaned seed were analyzed using an NIR (Near Infrared) machine. NIR uses the absorbance and reflectance of light energy to analyze, among other characteristics, moisture and oil content (Panford et al., 1988).

Analysis of the data using the Mixed Model program in the SAS ® software, version 9.1 (©2002-2003, SAS Institute Inc. Gary, NC, USA)., to run Analysis of Variance (ANOVA) showed that the data could be pooled over sites for individual cultivars for yield, and seed oil content in 2003. The seed protein content, AUDPC and the 1000 kernel weight were analyzed separately for each site. The 2004 data was analyzed based on site because the data were not suitable to be pooled based on the ANOVA analysis. The treatments were analyzed as one factor initially to determine whether or not the treatment had an effect on the model. Relative differences were calculated by dividing the treatment value by the control value and multiplying by 100. Dunnett's test was used to determine significance level of the differences between the control and the treatment values. Correlations were also calculated using the correlation function in GLM in SAS, version 9.1.

4.4 RESULTS

4.4.1 Disease Development

2003

The following results are described individually for each of the two study sites, with no pooling of data.

Agriculture and Agri-food Canada Research Station at Morden, Manitoba, Canada

Pasmo symptoms had already appeared by the first rating date on July 8th for the I/NF plots and July 11th for NI/F plots. Average disease severity for inoculated plots showed a noticeable increase starting in late July and continued to increase (with the exception of August 22nd) until harvest in late August (Figure 4.1.a). Severity increased from three on July 25th to 6 to 7 on August 15th.

For the NI/F plots the disease severity was more variable but rose from 2 to 4 as the crop matured, reaching a peak at the last day of ratings, August 28th. The trend was consistent increases in severity on successive observation dates, but with smaller increases than those seen for the I/NF plots (Figure 4.1.b). The severity was approximately half that seen in the I/NF plots. Maturity was not reached as early for the NI/F plots as they remained actively growing longer and some plots continued to flower and produce bolls until harvested. The first set of bolls in the NI/F plots matured shortly after the I/NF plots (September 2nd and August 21st respectively).

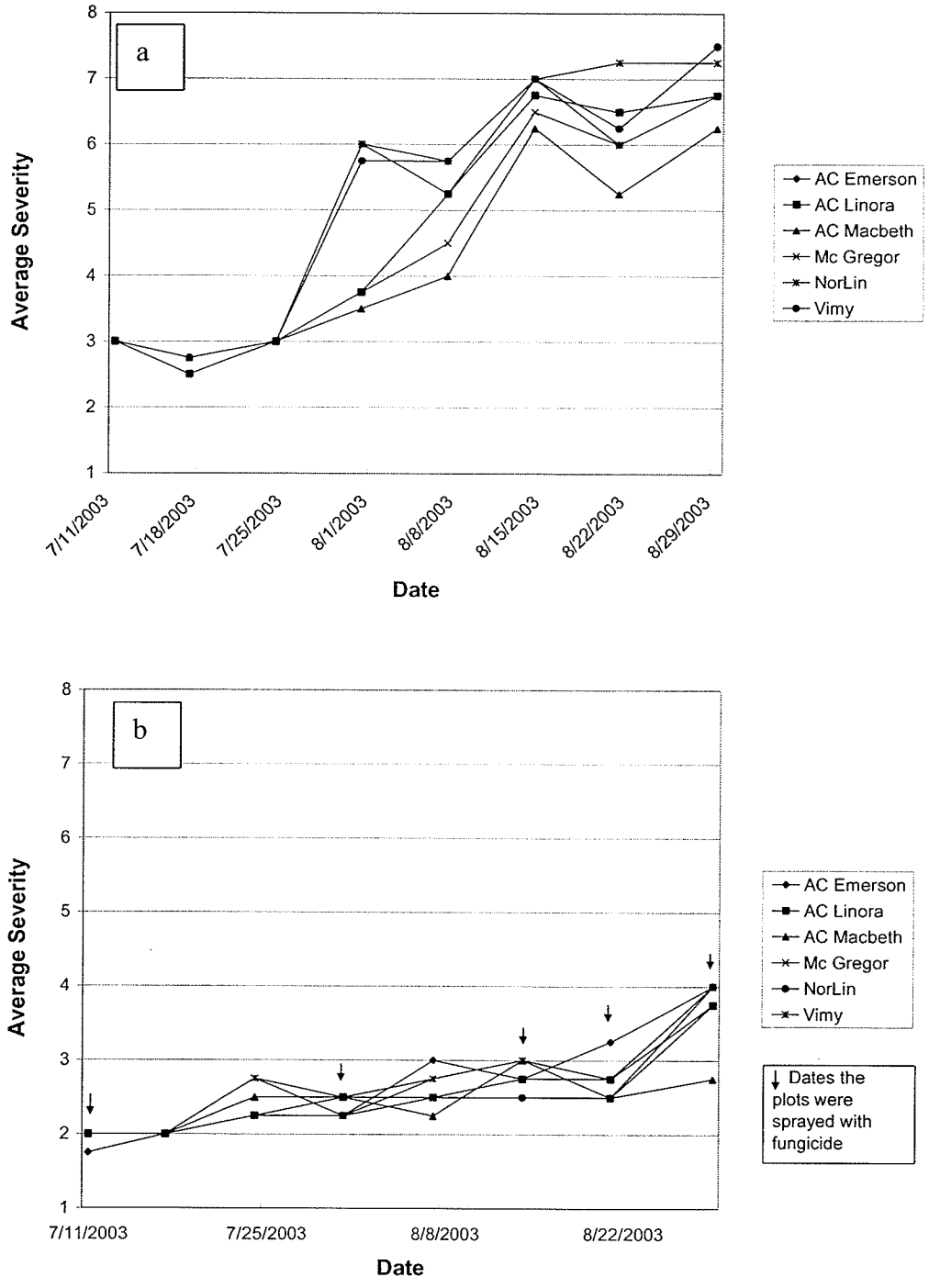


Figure 4.1. a, b. Average disease severity rating over a growing season of six different cultivars of flax with control (inoculated no fungicide application (I/NF)) treatment (a) and the non-inoculated with fungicide application (NI/F) treatment (b) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2003[1]. [1]Spray dates are indicated with arrows.

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

Symptoms were first seen on July 16th for the I/NF plots and July 18th for the NI/F plots. Plots were first rated on July 18th. The average disease severity increased steadily from 2.75 to reach a maximum of 7.25 for the I/NF plots with the final date giving the highest severity ratings of the season (Figure 4.2.a).

In the NI/F plots, disease severity was relatively low (remaining around 2) for much of the season with the exception of Vimy, which saw an increase in severity on August 8th to 3, followed by a decrease in severity in September. All cultivars with the NI/F treatment had their highest severity of the season recorded on the last date, September 8th. For many cultivars there was a large increase in severity between September 2nd and September 8th. Many of the cultivars had very low severity ratings for nearly the entire season until the point when severity increased to between 2.75 and 3.25 (Figure 4.2.b).

The Winnipeg plots matured at a similar time to the Morden plots. The inoculated plots reached maturity by August 16th, while the fungicide treated plots reached maturity by September 7th. The fungicide treated plots had begun to flower again by the second week of September.

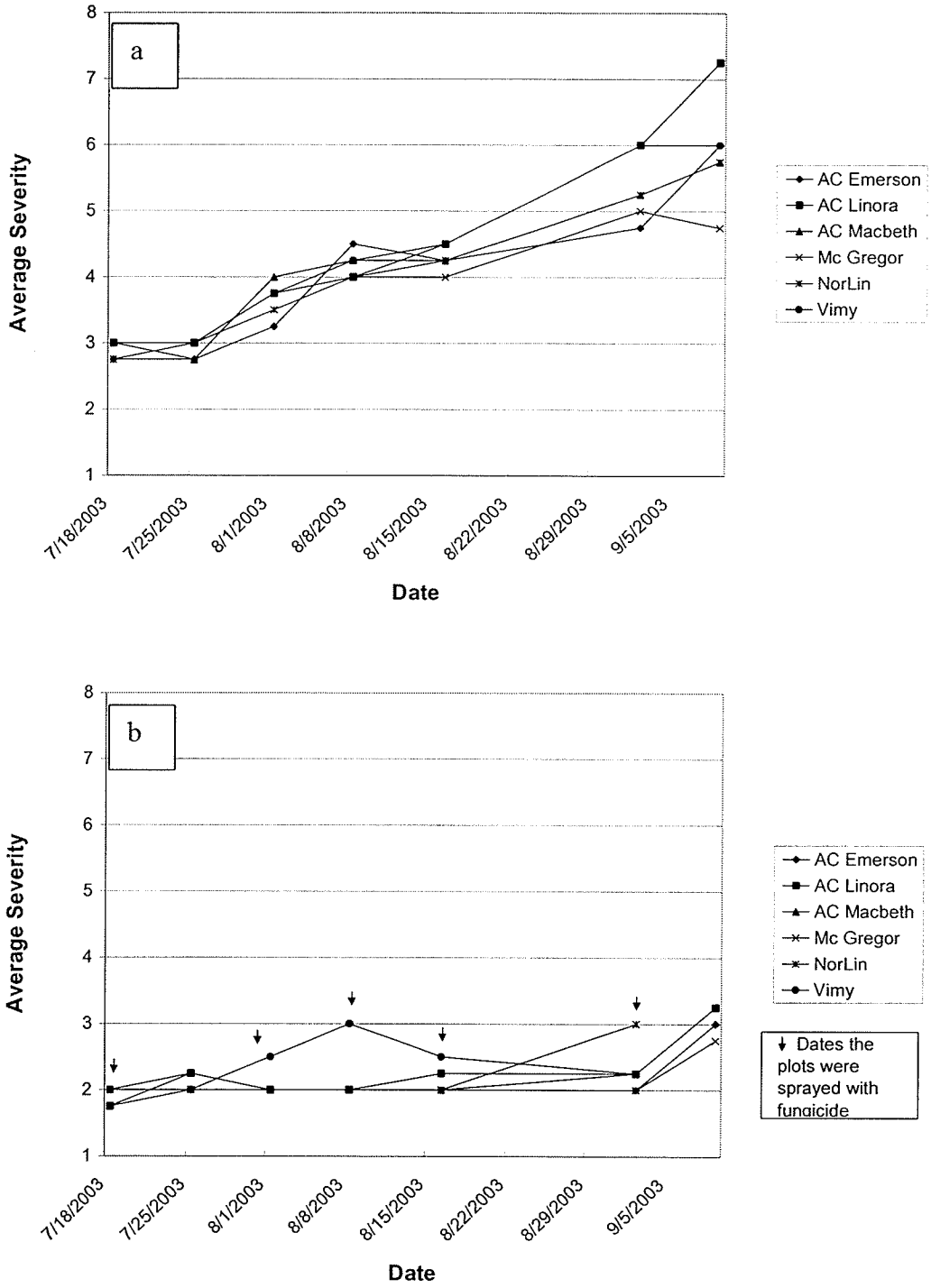


Figure 4.2. a, b. Average disease severity ratings over a growing season of six different cultivars of flax with control (inoculated no fungicide application (I/NF)) treatment and non-inoculated with fungicide application (NI/F) treatment (b) at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].
[1]Spray dates are indicated with arrows.

2004

Agriculture and Agri-food Canada Research Station at Morden, Manitoba, Canada

In 2004 disease developed much later than it did in 2003. The disease was visible on the plants by July 27th in all plots, over two weeks later than in 2003. Average disease

severity for plots under the I/F treatment was low for July and the first half of August, remaining below 3, and then climbed between August 13th and August 20th to reach an average severity of 4 (Figure 4.3.a).

Plots under the I/NF treatment had low average disease severity (2 or just above) until mid-August, after which the severity increased dramatically to 7, with the highest ratings being observed on the last date, September 9th (Figure 4.3.b).

Plots under the NI/F treatment had low disease severity in July and the early part of August, remaining around 2, with increases late in August to a range of 2.5 to 4, and larger increases through the first week of September to end at an average severity of 4 or 4.25 (Figure 4.4.a).

Non-inoculated with no fungicide application treated plots (Figure 4.4.b) had pasmo severity ratings of 2 or less until the middle of August when severity increased steadily to a minimum of 4 at the last observation date on September 7th, 2004.

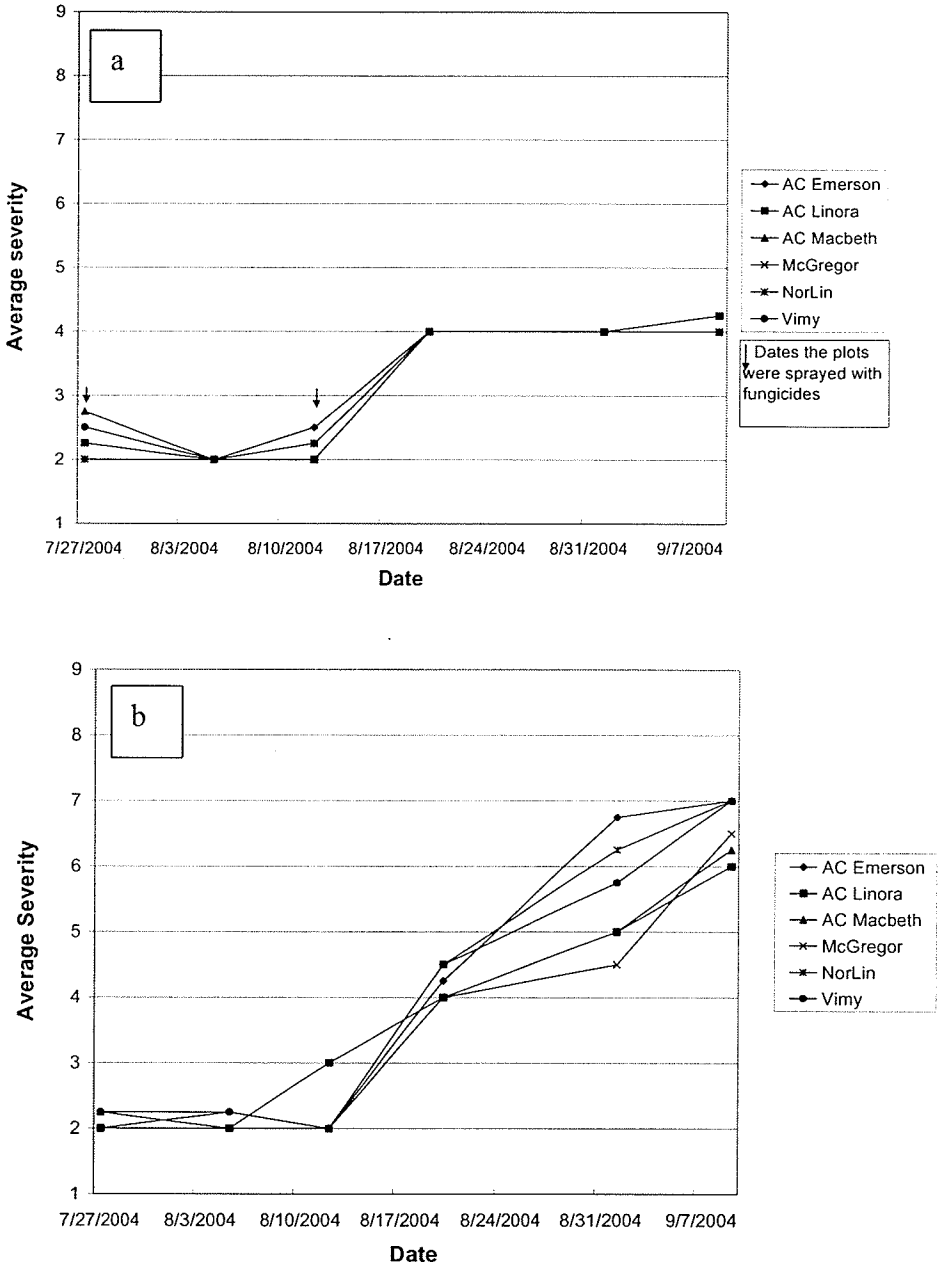


Figure 4.3. a, b. Average disease severity ratings over a growing season of six different cultivars of flax with the inoculated and fungicide application (I/F) treatment (a), and the control (inoculated with no fungicide application (I/NF)) (b) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004[1].
[1]Spray dates are indicated with arrows.

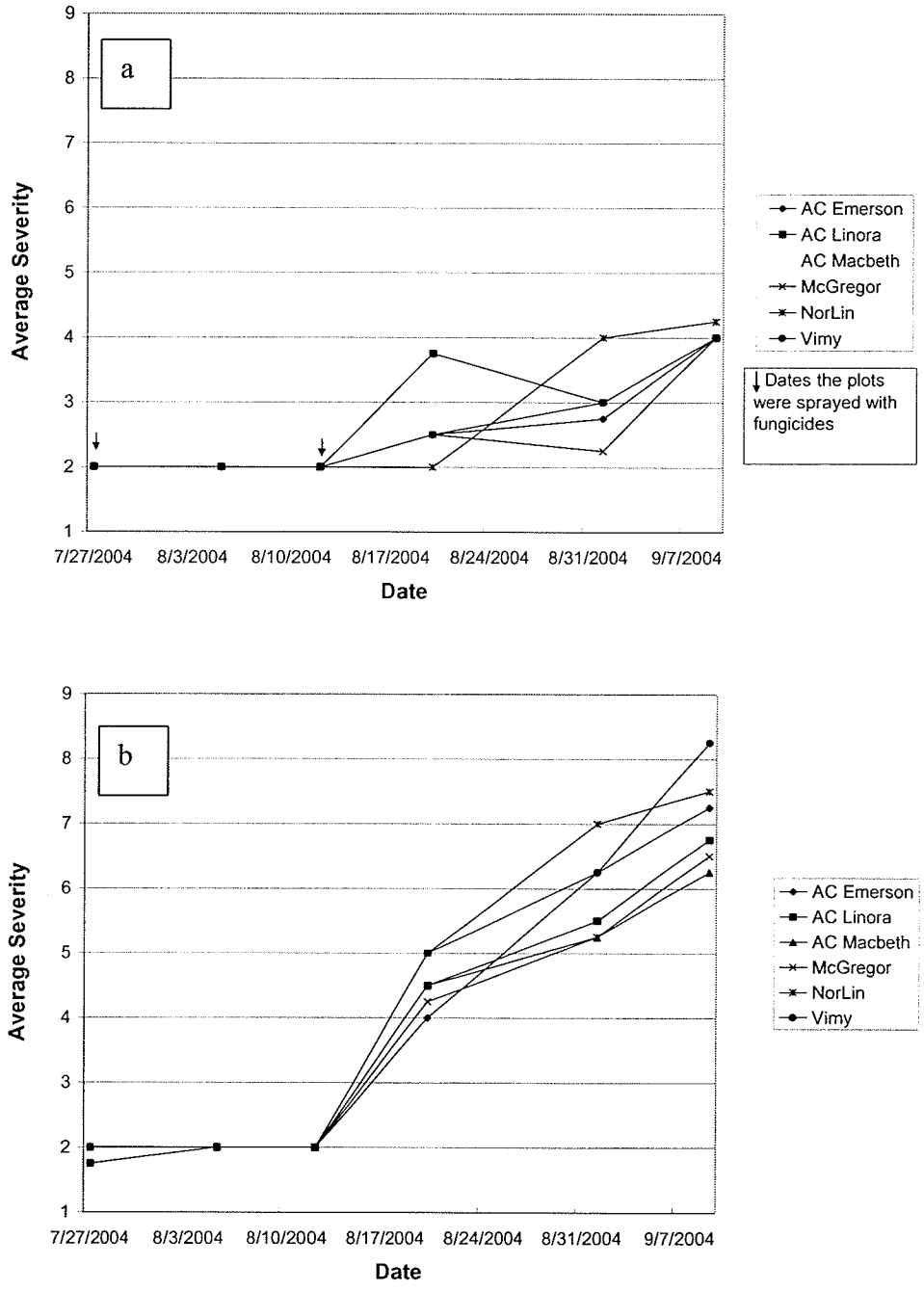


Figure 4.4. a, b. Average disease severity ratings over a growing season of six different cultivars of flax with non-inoculated with fungicide application (NI/F) treatment (a) and the non-inoculated with no fungicide application (NI/NF) treatment (b) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004. [1]Spray dates are indicated with arrows.

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

The disease was first observed on August 13th, which was quite late in the season, and much later than the previous year. Average disease severity for plots with the I/F treatment initially were rated at four in the third week of August, where they remained, until the middle of September. At this time, severity increased slightly to 4.25 and 5.25 (Figure 4.5.a).

Plots with the I/NF treatment were initially rated at a disease severity of 4. Severity ratings increased into early September to reach a maximum of 6, and again into mid September for a final maximum rating of 6.25 (Figure 4.5.b).

Plots with the NI/F treatment initially had a low disease severity ratings (2) which increased in the third week of August to reach 4. The severity then remained stable at this level until the middle of September when severity increased slightly to a maximum of 5 (Figure 4.6.a).

Non-inoculated non-fungicide treated plots also initially showed minimal disease symptoms, but disease ratings increased to 4 in the last week of August. Severity remained stable until the middle of September when it increased slightly, with the most severely infected plots reaching severities of 6 (Figure 4.6.b).

4.4.2 Area Under the Disease Progress Curve (AUDPC)

2003

Statistical analysis showed that AUDPCs were significantly different between sites and thus were analyzed separately (Table 4.2).

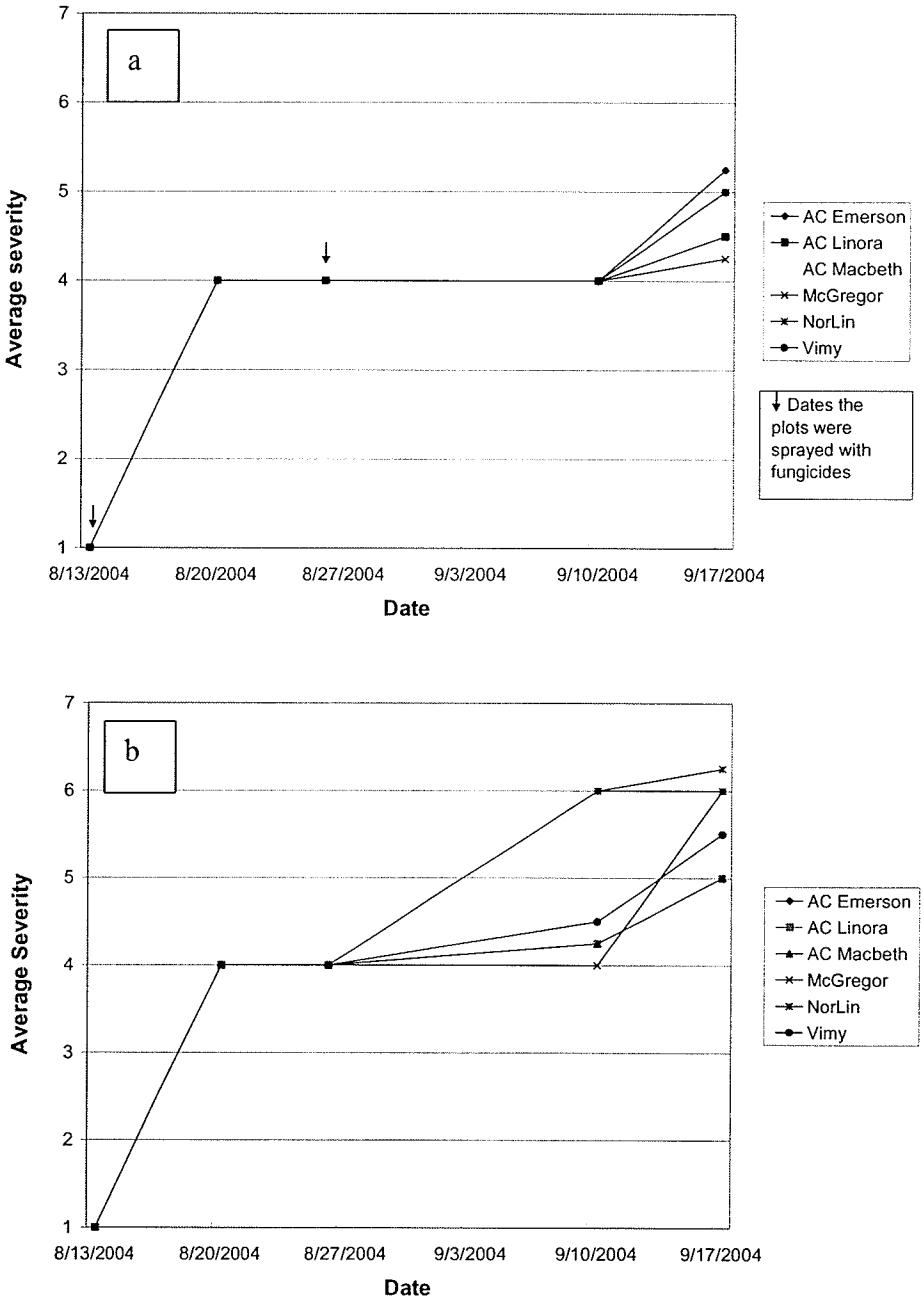


Figure 4.5. a, b. Average disease severity ratings over a growing season of six different cultivars of flax with the inoculated with fungicide application (I/F) treatment (a), and the control (inoculated with no fungicide application (I/NF)) treatment (b) at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].
[1]Spray dates are indicated with arrows.

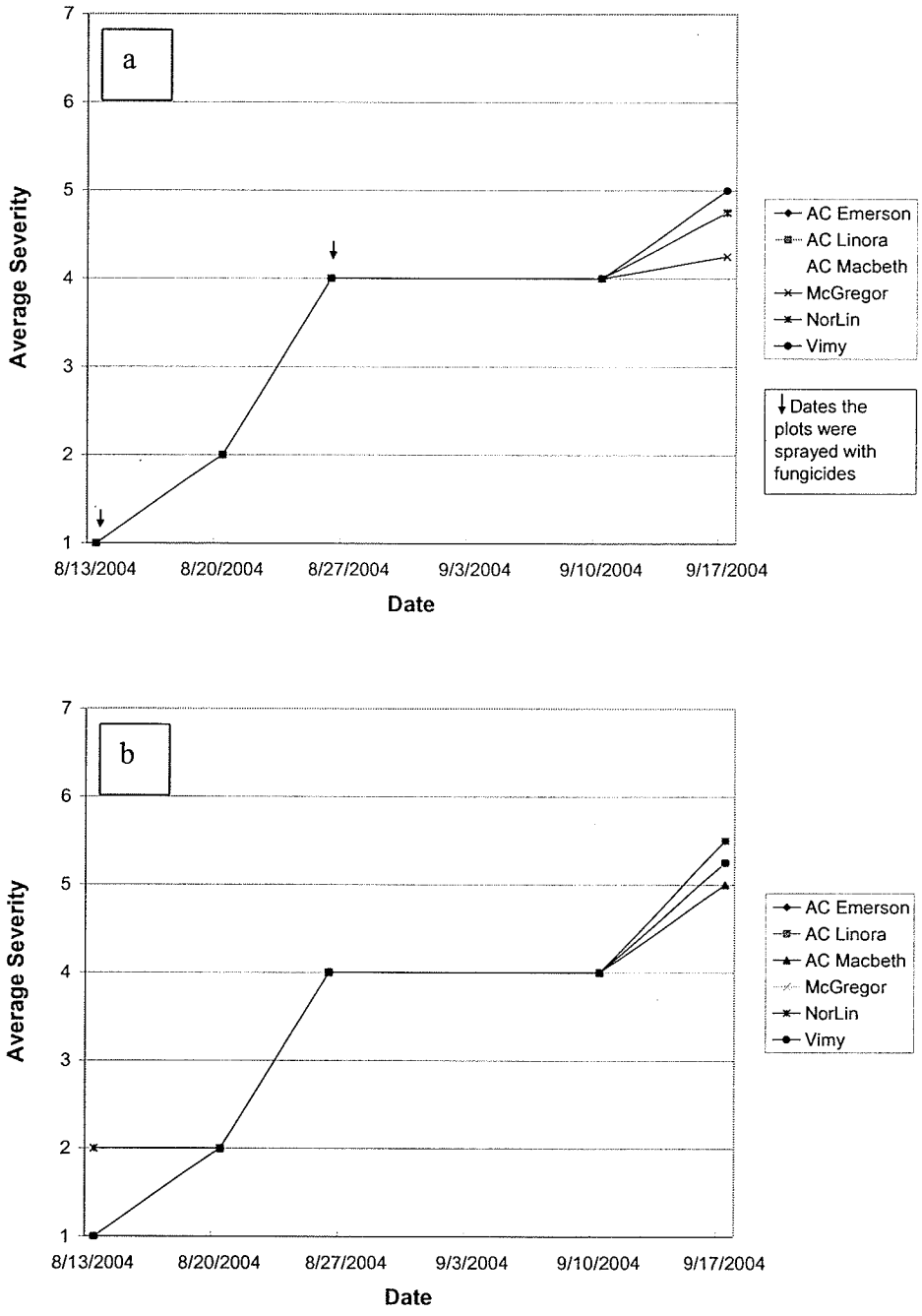


Figure 4.6. a, b. Average disease severity ratings over a growing season of six different cultivars of flax with the non-inoculated with fungicide application (NI/F) treatment (a) and non-inoculated no fungicide application (NI/NF) treatment (b) at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].
[1]Spray dates are indicated with arrows.

Table 4.2: Analysis of variance (ANOVA) for pooled area under the disease progress curve (AUDPC) values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and at Winnipeg, Manitoba, Canada at the University of Manitoba Field Station in 2003[1].

Source	F Value	Pr>F
Treatment	1388.31	<0.0001
Cultivar	2.23	0.0601
Rep x Treatment	0.57	0.7494
Site	8.15	0.0055

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

Agriculture and Agri-Food Canada Research Station at Morden, Manitoba, Canada

According to the ANOVA analysis the treatment (non-inoculated with fungicide application and inoculated with no fungicide application treatments) was highly significant and accounted for the largest portion of the error in the model. Cultivar differences were significant but did not account for as large a portion of the variability within the model (Table 4.3). As a result treatments were compared within cultivars.

Table 4.3: Analysis of variance (ANOVA) for area under the disease progress curve (AUDPC) values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2003[1].

Source	F Value	Pr>F
Treatment	440.44	<0.0001
Cultivar	2.78	0.0351
Rep x Treatment	2.57	0.0393

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

AUDPC was reduced by approximately 41 % to 55% across the cultivars with the application of the fungicide in the absence of inoculum. The cultivars Norlin and Vimy showed the largest response to the non-inoculated with fungicide application treatment. The difference between the treatments was significant for all cultivars (Table 4.4).

Table 4.4: Area under the disease progress curve (AUDPC) values (%) of the non-inoculated fungicide application (NI/F) treatment relative to the control (inoculated no fungicide application (I/NF)) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2003.[1]

Cultivar	AUDPC of (I/NF) control (%)	AUDPC values of NI/F treatment as a % of control
AC Emerson	100	50.25*
AC Linora	100	52.58*
AC Macbeth	100	57.79*
McGregor	100	58.30*
NorLin	100	44.18*
Vimy	100	49.07*

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

Only treatment (non-inoculated with fungicide application and inoculated with no fungicide application treatments) was highly significant at the Winnipeg site (Table 4.5), thus treatments were compared within cultivars.

Table 4.5: Analysis of variance (ANOVA) for area under the disease progress curve (AUDPC) values at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].

Source	F Value	Pr>F
Treatment	570.33	<0.0001
Cultivar	2.19	0.0821
Rep x Treatment	0.82	0.5663

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

There was less variability in the reduction in AUDPC at the Winnipeg site. The cultivars that were most positively affected by the application of fungicide without inoculation were AC Linora and Norlin (Table 4.6).

Table 4.6: Area under the disease progress curve (AUDPC) values (%) of the non-inoculated fungicide application (NI/F) treatment relative to the control (inoculated no fungicide application (I/NF)) at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].

Cultivar	AUDPC of (I/NF) control (%)	AUDPC value of NI/F treatment as a % of control
AC Emerson	100	50.29*
AC Linora	100	48.40*
AC Macbeth	100	50.67*
McGregor	100	49.95*
NorLin	100	48.50*
Vimy	100	54.53*

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

2004

The AUDPC was significantly affected by site, treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) and cultivar (Table 4.7). The error attributed to site was the largest of all the sources and thus sites were analyzed separately.

Table 4.7: Analysis of variance (ANOVA) for pooled area under the disease progress curve (AUDPC) values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Source	F Value	Pr>F
Treatment	178.04	<0.0001
Cultivar	3.36	0.0065
Rep x Treatment	0.39	0.9659
Site	237.78	<0.0001

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Agriculture and Agri-Food Canada Research Station at Morden, Manitoba, Canada

Treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments), and cultivar were both significant, but only the treatment effects accounted for a large part of the error seen in the model (Table 4.8). The treatments were therefore compared within cultivars.

Table 4.8: Analysis of variance (ANOVA) for area under the disease progress curve (AUDPC) values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station 2004[1].

Source	F Value	Pr>F
Treatment	149.39	<0.0001
Cultivar	14.15	<0.0001
Rep x Treatment	2.99	0.0025

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

AUDPC was generally lower at this site than it was in 2003. Within cultivars, treatments with fungicide (I/F and NI/F) had significantly lower AUDPC's than plots that were not treated with fungicides (I/NF and NI/NF). The non-inoculated with fungicide application treatments had AUDPC's that were 28 % to 40 % lower than those of the inoculated with no fungicide application treatment (the control). The inoculated with fungicide application treatment had significantly lower AUDPC values as well but the differences were smaller (Table 4.9).

Table 4.9: Area under the disease progress curve (AUDPC) values (%) of the non-inoculated with fungicide application (NI/F), the non-inoculated with no fungicide application (NI/NF) and the inoculated with fungicide application (I/F) treatments relative to the control (inoculated with no fungicide application (I/NF)) at Morden, Manitoba, Canada, at the Agriculture and Agri-food Canada Research Station in 2004[1].

Cultivar	AUCPC of (I/NF) control (%)	AUDPC of NI/F treatment as a % of the control	AUDPC of NI/NF treatment as a % of the control	AUDPC of I/F treatment as a % of the control
AC Emerson	100	59.10*	96.53	77.64*
AC Linora	100	71.13*	102.09	85.53*
AC Macbeth	100	70.70*	103.84	89.27*
McGregor	100	66.97*	107.95*	90.07*
NorLin	100	64.67*	109.05*	78.56*
Vimy	100	62.35*	106.77	80.71*

*Differences were significant at the Adjusted $P<0.05$ level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

The AUDPC was significantly affected by both treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) and cultivar (Table 4.10). The cultivar effects were small compared to treatment effects thus treatments were compared within cultivars.

Table 4.10: Analysis of variance (ANOVA) for area under the disease progress curve (AUDPC) values at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Source	F Value	Pr>F
Treatment	204.90	<0.0001
Cultivar	12.48	<0.0001
Rep x Treatment	0.57	0.8536

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

AUDPC was lower for all treatments when compared to the control (I/NF). The differences were significant for the NI/F treatment and the NI/NF treatment for each of the cultivars (Table 4.11).

Table 4.11: Area under the disease progress curve (AUDPC) values (%) of the non-inoculated with fungicide application (NI/F), the non-inoculated with no fungicide application (NI/NF) and the inoculated with fungicide application (I/F) treatments relative to the control (inoculated with no fungicide application (I/NF)) at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Cultivar	AUDPC of (I/NF) control (%)	AUDPC of NI/F treatment as a % of control	AUDPC of NI/NF treatment as a % of control	AUDPC of I/F treatment as a % of control
AC Emerson	100	77.04*	78.90*	82.54*
AC Linora	100	91.86*	93.34*	96.19
AC Macbeth	100	91.86*	92.60*	97.67
McGregor	100	89.81*	92.75*	94.85*
NorLin	100	76.56*	78.41*	81.41*
Vimy	100	89.51*	89.92*	94.09

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

4.4.3 Yield

2003

The effects of treatment (non-inoculated with fungicide application and inoculated with no fungicide application treatments) on yield were significant. Site and cultivar were not significant and were thus pooled for analysis (Table 4.12).

Table 4.12: Analysis of variance (ANOVA) for pooled yield values for Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].

Source	F Value	Pr>F
Treatment	186.24	<0.0001
Cultivar	1.23	0.3053
Rep x Treatment	1.27	0.2820
Site	1.73	0.1925

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

Yields for the NI/F treatment were significantly higher than yields for the control (I/NF) treatment (Table 4.13). In the case of the cultivar NorLin the yield was nearly doubled when fungicides were used in the absence of inoculation.

Table 4.13: Pooled yield values (%) of the non-inoculated fungicide application (NI/F) treatment relative to the control (inoculated with no fungicide application (I/NF)) for Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].

Cultivar	Yield of (I/NF) control (%)	Yield of NI/F treatment as a % of the control
AC Emerson	100	172.42*
AC Linora	100	176.51*
AC Macbeth	100	182.40*
McGregor	100	186.41*
NorLin	100	195.52*
Vimy	100	168.50*

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

2004

The treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) and site were both statistically significant in the yield model thus sites were analyzed separately (Table 4.14).

Table 4.14: Analysis of variance (ANOVA) for pooled yield values for Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Source	F Value	Pr>F
Treatment	79.31	<0.0001
Cultivar	1.61	0.1615
Rep x Treatment	0.88	0.5703
Site	46.89	<0.0001

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Agriculture and Agri-Food Canada Research Station at Morden, Manitoba, Canada

Cultivar and treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) were both significant at the Morden site (Table 4.15). Treatments were compared within cultivars as treatment effects represented a larger portion of the error in the model.

Table 4.15: Analysis of variance (ANOVA) for yield values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004[1].

Source	F Value	Pr>F
Treatment	19.13	<0.0001
Cultivar	7.95	<0.0001
Rep x Treatment	2.62	0.007

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Compared to the control (I/NF), the three treatments had higher yields. The increases seen for the non-inoculated no fungicide application treatment were significantly better than the control only for the cultivars AC Emerson and McGregor. AC Macbeth did not show any statistically significant differences in yield when treatments were compared (Table 4.16).

Table 4.16: Yield values (%) of the non-inoculated with fungicide application (NI/F), the non-inoculated with no fungicide application (NI/NF) and the inoculated with fungicide application (I/F) treatments relative to the control (inoculated with no fungicide application (I/NF)) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004[1].

Cultivar	Yield of (I/NF) control (%)	Yield of NI/F treatment as a % of control	Yield of NI/NF treatment as a % of control	Yield of I/F treatment as a % of control
AC Emerson	100	267.25*	182.03*	247.59*
AC Linora	100	206.66*	157.34	191.36*
AC Macbeth	100	151.75	110.12	138.10
McGregor	100	209.53*	161.63*	199.05*
NorLin	100	242.39*	165.55	221.93*
Vimy	100	158.55*	114.19	178.57*

*Differences were significant at the Adjusted $P<0.05$ level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

Treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) effects were statistically significant at the Winnipeg

site. Cultivar was also significant (Table 4.17). Treatment effects on the model were larger than were the effects of cultivar thus treatments were compared within cultivars.

Table 4.17: Analysis of variance (ANOVA) for yield values at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Source	F Value	Pr>F
Treatment	46.76	<0.0001
Cultivar	5.08	0.0006
Rep x Treatment	2.03	0.0366

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Yields were increased for all treatments compared to the control (I/NF) treatment. The increase was significant for all cultivars under all treatments except AC Linora and McGregor for the NI/NF treatment. The largest yield increases were seen for the NI/F treatment, with AC Macbeth showing the largest increase (Table 4.18).

Table 4.18: Yield values (%) of the non-inoculated with fungicide application (NI/F), the non-inoculated with no fungicide application (NI/NF) and the inoculated with fungicide application (I/F) treatments relative to the control (inoculated with no fungicide application (I/NF)) at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Cultivar	Yield of (I/NF) control (%)	Yield of NI/F treatment as a % of control	Yield of NI/NF treatment as a % of control	Yield of I/F treatment as a % of control
AC Emerson	100	218.59*	187.99*	197.88*
AC Linora	100	158.01*	139.98	145.52*
AC Macbeth	100	367.92*	353.28*	260.39*
McGregor	100	236.11*	157.11	208.06*
NorLin	100	212.31*	155.63*	176.41*
Vimy	100	248.34*	177.66*	187.85*

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

4.4.4 Seed Oil Content

2003

Treatment (non-inoculated with fungicide application and inoculated with no fungicide application treatments) was statistically significant as was cultivar in the model, but cultivar accounted for slightly less of the error in the model than did treatment. Site was not a significant factor in the model so data was pooled over sites (Table 4.19).

Table 4.19: Analysis of variance (ANOVA) for pooled seed oil content values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].

Source	F Value	Pr>F
Treatment	15.87	0.0072
Cultivar	10.52	<0.0001
Rep x Treatment	3.57	0.0036
Site	3.7	0.0581

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

Oil content of seeds produced from the NI/F treatment was significantly higher relative to the control (I/NF) treated plots in 2003 in both sites for all cultivars except Norlin, with the differences ranging from 3.01 % to 5.877 % across the six cultivars (Table 4.20).

Table 4.20: Pooled seed oil content values (%) of the non-inoculated with fungicide application (NI/F) treatment relative to the control (inoculated with no fungicide application (I/NF)), at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].

Cultivar	Oil content of (I/NF) control (%)	Oil content of NI/F treatment as a % of control
AC Emerson	100	103.11*
AC Linora	100	105.88*
AC Macbeth	100	104.76*
McGregor	100	104.19*
NorLin	100	104.09
Vimy	100	104.57*

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

2004

Site, cultivar and treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) were all significant for seed oil content in 2004. Sites were analyzed separately, as were cultivars, in order to look more closely at the treatment effects within cultivars (Table 4.21).

Table 4.21: Analysis of variance (ANOVA) for pooled seed oil content values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Source	F Value	Pr>F
Treatment	111.41	<.0001
Cultivar	44.77	<.0001
Rep x Treatment	1.40	0.1724
Site	94.04	<.0001

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Agriculture and Agri-Food Canada Research Station at Morden, Manitoba, Canada

Treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) had a significant effect on the seed oil content as shown in Table 4.22. Cultivar also had a significant effect in the model at the Morden site.

Table 4.22: Analysis of variance (ANOVA) for seed oil content values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004[1].

Source	F Value	Pr>F
Treatment	63.13	<0.0001
Cultivar	32.58	<0.0001
Rep x Treatment	1.75	0.0776

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Seed oil content was higher for all treatments when compared to the control (I/NF), but was not statistically higher for the cultivars AC Macbeth and Norlin under the NI/NF treatment (Table 4.23).

Table 4.23: Seed oil content values (%) of the non-inoculated with fungicide application (NI/F), the non-inoculated with no fungicide application (NI/NF) and the inoculated with fungicide application (I/F) treatments relative to the control (inoculated with no fungicide application (I/NF)) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004[1].

Cultivar	Oil content of (I/NF) control (%)	Oil content of NI/F treatment as a % of control	Oil content of NI/NF treatment as a % of control	Oil content of I/F treatment as a % of control
AC Emerson	100	107.97*	104.48*	107.91*
AC Linora	100	107.47*	105.81*	106.92*
AC Macbeth	100	105.84*	102.37	105.09*
McGregor	100	107.74*	104.56*	107.99*
NorLin	100	106.48*	103.15	107.35*
Vimy	100	109.14*	102.90*	108.40*

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

Oil content of the seed was significantly affected by treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) and cultivar as indicated by Table 4.24. Treatment effects accounted for a larger portion of the error in the model thus treatment were analysed within cultivars to determine the effects of the treatments relative to the control.

Table 4.24: Analysis of variance (ANOVA) for seed oil content values at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Source	F Value	Pr>F
Treatment	130.16	<0.0001
Cultivar	44.70	<0.0001
Rep x Treatment	2.11	0.4085

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Seed oil content was significantly increased for both the NI/F and I/F treatments compared to the control. For the NI/NF treatment increases were small, and in some cases seed oil content fell, with none of the changes being significant (Table 4.25).

Table 4.25: Seed oil content values (%) of the non-inoculated with fungicide application (NI/F), the non-inoculated with no fungicide application (NI/NF) and the inoculated with fungicide application (I/F) treatments relative to the control (inoculated with no fungicide application (I/NF)) at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Cultivar	Oil content of (I/NF) control (%)	Oil content of NI/F treatment as a % of control	Oil content of NI/NF treatment as a % of control	Oil content of I/F treatment as a % of control
AC Emerson	100	105.00*	100.79	104.33*
AC Linora	100	106.81*	99.149	103.83*
AC Macbeth	100	105.4*	100.72	104.54*
McGregor	100	109.43*	102.30	107.80*
NorLin	100	105.39*	100.74	105.06*
Vimy	100	104.10*	98.49	102.59*

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

4.4.5 Seed Protein Content

2003

Site and treatment (non-inoculated with fungicide application and inoculated with no fungicide application treatments) were statistically significant for seed protein content in 2003. Site accounted for the largest portion of the error in the model. As a result data was analysed by site. Cultivar was not significant in this year (Table 4.26).

Table 4.26: Analysis of variance (ANOVA) for pooled seed protein content values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].

Source	F Value	Pr>F
Treatment	17.97	0.0054
Cultivar	1.85	0.1128
Rep x Treatment	2.70	0.0197
Site	46.89	<0.0001

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

Agriculture and Agri-Food Canada Research Station at Morden, Manitoba, Canada

At the Morden site cultivar was statistically significant but did not account for a large portion of the error in the model. Rep and treatment interactions were highly significant and accounted for most of the error in the model (Table 4.27).

Table 4.27: Analysis of variance (ANOVA) for seed protein content values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2003[1].

Source	F Value	Pr>F
Treatment	0.03	0.8771
Cultivar	5.42	0.0011
Rep x Treatment	15.08	<0.0001

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

Seed protein contents were not significantly different between the control (I/NF) and the NI/F treatment, with the exception of the cultivar McGregor, which had a protein content increase of over 7.5 % compared to the control (I/NF) (Table 4.28).

Table 4.28: Seed protein content values (%) of the non-inoculated with fungicide application (NI/F) treatment relative to the control (inoculated with no fungicide application (I/NF)) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2003[1].

Cultivar	Protein content of I/NF (control) (%)	Protein content of NI/F treatment as a % of control
AC Emerson	100	98.12
AC Linora	100	101.99
AC Macbeth	100	97.25
McGregor	100	107.56*
NorLin	100	100
Vimy	100	98.80

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

Treatment (non-inoculated with fungicide application and inoculated with no fungicide application treatments) effects were significant in the model, as was cultivar, but the treatment accounted for a much larger portion of the error in the model (Table 4.29). This resulted in treatments being compared within cultivars.

Table 4.29: Analysis of variance (ANOVA) for seed protein content values at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].

Source	F Value	Pr>F
Treatment	212.77	<0.0001
Cultivar	2.63	0.0445
Rep x Treatment	1.76	0.1435

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

At the Winnipeg site seed protein content was significantly increased in the NI/F treatment when compared to the control (I/NF) for each of the cultivars (Table 4.30).

Table 4.30: Seed protein content values (%) of the non-inoculated with fungicide application (NI/F) treatment relative to the control (inoculated with no fungicide application (I/NF)), at Winnipeg, Manitoba, Canada at the University of Manitoba Field Station in 2003[1].

Cultivar	Protein content of I/NF (control) (%)	Protein content of NI/F treatment as a % of control
AC Emerson	100	114.72*
AC Linora	100	117.70*
AC Macbeth	100	116.35*
McGregor	100	118.81*
NorLin	100	118.62*
Vimy	100	118.01*

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

2004

Site was statistically significant in the model in 2004, and accounted for the largest portion of the error (Table 4.31).

Table 4.31: Analysis of variance (ANOVA) for pooled seed protein content values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Source	F Value	Pr>F
Treatment	0.65	0.5986
Cultivar	1.96	0.0883
Rep x Treatment	0.98	0.4734
Site	156.33	<0.0001

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Agriculture and Agri-Food Canada Research Station at Morden, Manitoba, Canada

Treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) was significant at the Morden site in 2004, and had the largest error term in the model (Table 4.32).

Table 4.32: Analysis of variance (ANOVA) for seed protein content values at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004[1].

Source	F Value	Pr>F
Treatment	43.29	<0.0001
Cultivar	1.19	0.327
Rep x Treatment	1.09	0.2895

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Seed protein content increased for all cultivars in all treatment except for NorLin under the I/F treatment. The increase was only significant for a few cultivars, as indicated in Table 4.33.

Table 4.33: Seed protein content values (%) of the non-inoculated with fungicide application (NI/F), the non-inoculated with no fungicide application (NI/NF) and the inoculated with fungicide application (I/F) treatments relative to the control (inoculated with no fungicide application (I/NF)) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004[1].

Cultivar	Protein content of (I/NF) control (%)	Protein content of NI/F treatment as a % of control	Protein content of NI/NF treatment as a % of control	Protein content of I/F treatment as a % of control
AC Emerson	100	104.04	109.28	102.28
AC Linora	100	107.33*	104.25	107.12
AC Macbeth	100	105.60	105.12	104.37
McGregor	100	109.57*	109.87	105.93*
NorLin	100	107.13	107.59	100
Vimy	100	108.18*	104.07	105.56*

*Relative differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

Treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) and cultivar were both significant in the model and accounted for approximately the same amount of error, with treatment accounting for slightly more (Table 4.34). In order to determine the effects of treatment, treatments were compared within cultivars.

Table 4.34: Analysis of variance (ANOVA) for seed protein content values at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Source	F Value	Pr>F
Treatment	14.74	0.0003
Cultivar	14.10	<0.0001
Rep x Treatment	4.9	<0.0001

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Decreases in seed protein content occurred for the NI/F as well as the I/F treatments, but was not always statistically significant. Seed protein contents under the NI/NF treatment were not significantly different from the control (I/NF) for any cultivar (Table 4.35).

Table 4.35: Seed protein content values (%) of the non-inoculated with fungicide application (NI/F), the non-inoculated with no fungicide application (NI/NF) and the inoculated with fungicide application (I/F) treatments relative to the control (inoculated with no fungicide application (I/NF)) at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Cultivar	Protein content of (I/NF) control (%)	Protein content of NI/F treatment as a % of control	Protein content of NI/NF treatment as a % of control	Protein content of I/F treatment as a % of control
AC Emerson	100	89.66*	100.95	93.88*
AC Linora	100	91.95*	102.07	99.54
AC Macbeth	100	95.16	101.43	98.57
McGregor	100	90.13*	98.55	89.09*
NorLin	100	93.16*	100.32	94.01*
Vimy	100	92.12*	101.53	93.65

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

4.4.6 1000 Kernel Weight

2003

In 2003 the treatment (non-inoculated with fungicide application and inoculated with no fungicide application treatments) and cultivar were both statistically significant, but the treatment accounted for the majority of the error in the model (Table 4.36). Site was statistically significant so sites were analyzed separately.

Table 4.36: Analysis of variance (ANOVA) for pooled 1000 kernel weights at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and at Winnipeg, Manitoba, Canada at the University of Manitoba Field Station in 2003[1].

Source	F Value	Pr>F
Treatment	462.78	<0.0001
Cultivar	1.8543	<0.0001
Rep x Treatment	1.60	0.1592
Site	7.27	0.0086

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

Agriculture and Agri-Food Canada Research Station at Morden, Manitoba, Canada

When sites were analyzed separately the treatment (non-inoculated with fungicide application and inoculated with no fungicide application treatments) and cultivar were both statistically significant at the Morden site, as indicated in Table 4.37. Treatment effects were larger than those of cultivar in the model thus treatments were analyzed within cultivars.

Table 4.37: Analysis of variance (ANOVA) for 1000 kernel weights at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2003[1].

Source	F Value	Pr>F
Treatment	440.44	<0.0001
Cultivar	41.65	<0.0001
Rep x Treatment	2.57	0.0375

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

The 1000 kernel weights were increased significantly between the control (I/NF) and the NI/F treatment for all cultivars. Increases reached 42 % for the cultivar AC Linora (Table 4.38).

Table 4.38:1000 kernel weight values (%) of the non-inoculated with fungicide application (NI/F) treatment relative to the control (inoculated with no fungicide application (I/NF)) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2003[1].

Cultivar	1000 kernel weight of (I/NF) control (%)	1000 kernel weight of NI/F treatment as a % of control
AC Emerson	100	125.73*
AC Linora	100	142.20*
AC Macbeth	100	140.22*
McGregor	100	137.42*
NorLin	100	131.68*
Vimy	100	134.21*

*Differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

Both cultivar and treatment (non-inoculated with fungicide application and inoculated with no fungicide application treatments) were statistically significant at the Winnipeg site, with treatment accounting for the largest portion of the error in the model (Table 4.39). This resulted in the decision to analyze treatments within cultivars.

Table 4.39: Analysis of variance (ANOVA) for 1000 kernel weights at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].

Source	F Value	Pr>F
Treatment	159.08	<0.0001
Cultivar	24.36	<0.0001
Rep x Treatment	1.51	0.2105

[1] Treatment comprises both the non-inoculated with fungicide application (NI/F) and the control (inoculated with no fungicide application (I/NF) treatments.

The 1000 kernel weight was increased significantly for all cultivars except Vimy when the NI/F treatment was compared to the control (I/NF) (Table 4.40).

Table 4.40: 1000 kernel weight values (%) of the non-inoculated with fungicide application (NI/F) treatment relative to the control (inoculated with no fungicide application (I/NF)) at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2003[1].

Cultivar	1000 kernel weight of I/NF (control) (%)	1000 kernel weight of NI/F treatment as a % of the control
AC Emerson	100	126.76*
AC Linora	100	132.07*
AC Macbeth	100	129.85*
McGregor	100	133.75*
NorLin	100	125.96*
Vimy	100	122.10

*Relative differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

2004

In 2004, treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments), cultivar and site were all statistically significant effects in the model (Table 4.41). The sites were therefore analyzed separately to determine the cultivar and treatment effects.

Table 4.41: Analysis of variance (ANOVA) for pooled 1000 kernel weights at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station and at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Source	F Value	Pr>F
Treatment	253.85	<0.0001
Cultivar	56.52	<0.0001
Rep x Treatment	.078	0.6691
Site	172.95	<0.0001

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Agriculture and Agri-Food Canada Research Station at Morden, Manitoba, Canada

Both treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) and cultivar had significant effects on the model at the Morden site, with treatment accounting for slightly more or the error (Table 4.42). To better determine the effects of treatment in the model treatments were analyzed within cultivars.

Table 4.42: Analysis of variance (ANOVA) for 1000 kernel weights at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004[1].

Source	F Value	Pr>F
Treatment	136.88	<0.0001
Cultivar	101.77	<0.0001
Rep x Treatment	6.12	0.2664

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

Significant increases were seen between the control (I/NF) and the NI/F and I/F treatments in 2004 at the Morden site. Increases were also seen for the NI/NF treatment but the differences were not always significant (Table 4.43).

Table 4.43: 1000 kernel weight values (%) of the non-inoculated with fungicide application (NI/F), the non-inoculated with no fungicide application (NI/NF) and the inoculated with fungicide application (I/F) treatments relative to the control (inoculated with no fungicide application (I/NF)) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004[1].

Cultivar	1000 Kernel weight of (I/NF) control (%)	1000 kernel weight of NI/F treatment as a % of control	1000 kernel weight of NI/NF treatment as a % of control	1000 kernel weight of I/F treatment as a % of control
AC Emerson	100	132.71*	109.28*	128.09*
AC Linora	100	160.44*	104.25	124.48*
AC Macbeth	100	127.15*	105.12	123.56*
McGregor	100	134.03*	109.87*	131.55*
NorLin	100	131.66*	107.59*	130.23*
Vimy	100	130.92*	104.07	127.99*

*Relative differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

At the Winnipeg site both treatment (non-inoculated with fungicide application, inoculated with no fungicide application, non-inoculated with no fungicide application, and inoculated with fungicide application treatments) and cultivar were statistically significant and accounted for approximately the same amount of error in the model with treatment being slightly higher (Table 4.44). As a result treatments were compared within cultivars in order to determine the relative effects of the treatments.

Table 4.44: Analysis of variance (ANOVA) for 1000 kernel weights at Winnipeg, Manitoba, Canada, at the University of Manitoba Field Station in 2004[1].

Source	F Value	Pr>F
Treatment	117.79	<0.0001
Cultivar	113.95	<0.0001
Rep x Treatment	1.13	0.3537

[1] Treatment comprises the non-inoculated with fungicide application (NI/F), control (inoculated with no fungicide application (I/NF)), non-inoculated with no fungicide application (NI/NF) and inoculated with fungicide application (I/F) treatments.

The 1000 kernel weight was significantly different for the NI/F and I/F treatments when they were compared to the control (I/NF). AC Macbeth had a significantly lower 1000 kernel weight for these two treatments, while all the other cultivars had increases. Within the NI/NF treatment only AC Emerson had a significantly higher 1000 kernel weight when compared to the control (I/NF), as seen in Table 4.45.

Table 4.45:1000 kernel weight values (%) of the non-inoculated with fungicide application (NI/F), the non-inoculated with no fungicide application (NI/NF) and the inoculated with fungicide application (I/F) treatments relative to the control (inoculated with no fungicide application (I/NF)) at Winnipeg, Manitoba, Canada at the University of Manitoba Field Station in 2004[1].

Cultivar	1000 kernel weight of (I/NF) control (%)	1000 kernel weight of NI/F treatment as a % of control	1000 kernel weight of NI/NF treatment as a % of control	1000 kernel weight of I/F treatment as a % of control
AC Emerson	100	117.94*	107.67*	110.97*
AC Linora	100	119.58*	104.58	107.95*
AC Macbeth	100	93.64*	104.29	97.02*
McGregor	100	122.48*	108.47	113.00*
NorLin	100	121.05*	104.67	114.30*
Vimy	100	118.84*	104.30	109.96*

*Relative differences were significant at the Adjusted P<0.05 level.
[1] Values were calculated by dividing the value of the treatment by the value of the control and multiplying by 100.

4.4.7 Correlations

2003

Agriculture and Agri-Food Canada Research Station at Morden, Manitoba, Canada

At the Morden site in 2003 (Table 4.46), yield was significantly correlated with all the other factors. It was positively correlated with oil content and 1000 kernel weight, and negatively correlated with protein content and AUDPC. Oil content was significantly negatively correlated with protein content but positively correlated with 1000 kernel weight. The 1000 kernel weight was negatively correlated with AUDPC.

Table 4.46. Correlation values for six flax cultivars under non-inoculated with fungicide application (NI/F) and control (inoculated with no fungicide application (I/NF) treatments at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2003.

Factors	Correlation	Significance Value[1]
Yield & Oil Content	0.63	0.0002
Yield & Protein Content	-0.60	0.0004
Yield & 1000 Kernel Weight	0.57	0.0007
Yield & AUDPC	-0.43	0.017
Oil Content & Protein Content	-0.73	<0.0001
Oil Content & 1000 Kernel Weight	0.58	0.0007
Oil Content & AUDPC	-0.31	0.0898
Protein Content & 1000 Kernel Weight	-0.33	0.0699
Protein Content & AUDPC	0.12	0.5145
1000 Kernel Weight & AUDPC	-0.36	0.0437

[1]Probability >| r |

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

At the Winnipeg site in 2003 (Table 4.47), there were significant negative correlations between oil and protein content as well as between 1000 kernel weight and AUDPC.

Table 4.47. Correlation values for six flax cultivars under non-inoculated with fungicide application (NI/F) control (inoculated with no fungicide application (I/NF)) treatments at Winnipeg, Manitoba, Canada at the University of Manitoba Field Station in 2003.

Factors	Correlation	Significance Value[1]
Yield & Oil Content	0.09	0.6509
Yield & Protein Content	-0.27	0.1477
Yield & 1000 Kernel Weight	-0.006	0.977
Yield & AUDPC	0.31	0.1007
Oil Content & Protein Content	-0.78	<0.0001
Oil Content & 1000 Kernel Weight	0.06	0.7435
Oil Content & AUDPC	-0.27	0.1416
Protein Content & 1000 Kernel Weight	0.07	0.7161
Protein Content & AUDPC	0.20	0.2997
1000 Kernel Weight & AUDPC	-0.40	0.0297

[1]Probability >| r |

2004

Agriculture and Agri-Food Canada Research Station at Morden, Manitoba, Canada

Yield and oil content had the largest number of significant correlations at the Morden site in 2004 (Table 4.48). Yield was positively correlated with both oil content and protein content. Oil content was positively correlated with protein content, but was negatively correlated with AUDPC.

Table 4.48. Correlation values for six flax cultivars under non-inoculated with fungicide application (NI/F), non-inoculated with no fungicide application (NI/NF) inoculated with fungicide application (I/F) and control (inoculated with no fungicide application (I/NF)) at Morden, Manitoba, Canada, at the Agriculture and Agri-Food Canada Research Station in 2004.

Factors	Correlation	Significance Value[1]
Yield & Oil Content	0.45	0.0003
Yield & Protein Content	-0.44	0.0004
Yield & 1000 Kernel Weight	0.15	0.2382
Yield & AUDPC	-0.05	0.7108
Oil Content & Protein Content	-0.78	<.0001
Oil Content & 1000 Kernel Weight	0.17	0.1859
Oil Content & AUDPC	-0.28	0.0288
Protein Content & 1000 Kernel Weight	-0.12	0.3716
Protein Content & AUDPC	-0.01	0.9296
1000 Kernel Weight & AUDPC	0.06	0.6635

[1]Probability >| r |

University of Manitoba Field Station at Winnipeg, Manitoba, Canada

At the Winnipeg site in 2004 (Table 4.49), yield was positively correlated with 1000 kernel weight. Oil content was negatively correlated with protein content and positively correlated with 1000 kernel weight.

Table 4.49. Correlation values for six flax cultivars under and non-inoculated with fungicide application (NI/F), non-inoculated with no fungicide application (NI/NF), inoculated with fungicide application (I/F) and control (inoculated with no fungicide application (I/NF)) at Winnipeg, Manitoba, Canada at the University of Manitoba Field Station in 2004.

Factors	Correlation	Significance Value[1]
Yield & Oil Content	0.10	0.4622
Yield & Protein Content	0.03	0.8256
Yield & 1000 Kernel Weight	0.28	0.0278
Yield & AUDPC	0.93	0.4757
Oil Content and Protein Content	-0.50	<0.0001
Oil Content & 1000 Kernel Weight	0.43	0.0005
Oil Content & AUDPC	-0.23	0.0776
Protein Content & 1000 Kernel Weight	-0.02	0.875
Protein Content & AUDPC	0.06	0.6718
1000 Kernel Weight & AUDPC	-0.24	0.0664

[1]Probability > | r |

4.5 DISCUSSION

The effects of fungicides on several variables including disease severity, yield, seed oil content and seed protein content were addressed. Severity ratings were also examined briefly. The correlations between yield, seed oil and protein content and disease severity were computed and relationships were explored.

The variables investigated in this study responded differently to fungicide treatments and to inoculation over the two years and at the two sites. Some of these responses may have been the result of weather and cultural effects, as outlined below, which may have altered the effects of the fungicide application at particular sites or in a particular year. Weather and cultural effects may also have affected the ability of the pathogen to infect the plants. These effects may also have modified its effect the pathogen had on the plants, resulting in different effects on the quality characteristics.

4.5.1 Weather Effects

Weather conditions were quite different between the 2 years of this study. The differences may help account for variable responses of both the disease severity and progression and the measured characteristics of the cultivars (Appendix 6).

Temperatures during the 2003 season were favourable for the development of pasmo (Appendix 4 a, and b). The hot temperatures seen in late July and early August may have slightly hindered disease development.

In contrast, temperatures in July and August of 2004 were cool (Appendix 4 c and d), and may have been sufficiently low to negatively impact both the flax plants and the pathogen, especially at the Winnipeg site.

Leaf wetness was created artificially by the misting system, thus conditions should have been favourable for disease development in late July at the Morden site and into early August at the Winnipeg site in 2003 (Appendix 4 a, and b). Disease development may have been slowed by dry conditions during August after misting had been completed. Yield may also have been affected by the dry conditions in 2003.

In 2004, the humidity conditions were favourable for the disease during most of the growing period. At the Winnipeg, site the misting system was only used minimally due to high levels of humidity during the misting period. Overall the 2004 season was wetter than the 2003 season in both locations, which may have had an impact on yield .

Sackston, (1951) observed that cool dry weather seemed to impede the spread of the disease. Flor (1943) and Rashid (2003) stated that warm moist conditions were ideal for pasmo disease development. Dybing and Zimmerman (1965) reported that exposure to low temperatures, such as 11° C, for periods of two weeks or longer slowed growth, reduced seed and boll number, decreased oil content and delayed flax maturity.

Perryman and Fitt (2000) reported that when the weather was wet between flowering and harvest, yield losses, as well as yield, are higher than in dry years. Perryman and Fitt (2000) also observed that in years with above average

temperatures, plots not treated with fungicides tended to have lower yields. In the current strudy, the yield increases in 2004 were larger than those in 2003, when treatments were compared to the control (Appendix 6). This may be attributable to the wetter season, which may have provided the plants better growing conditions. The higher rainfall, especially when compared to the hot dry conditions seen in 2003, may have allowed the-fungicide protected plants to perform better than plants without the benefit of fungicide protection. Conversely, it may be that wetter conditions provided a much more favourable environment for the pathogen, thus the unprotected treatments did not perform as well as they had in the previous year.

4.5.2 Cultural Effects

It was noted, during the course of the current trial, that the application of the fungicide slowed maturation slightly. It was also observed that inoculated plots tended to mature faster than those that had not been inoculated. At the Morden site in 2003, the plots that did not receive any fungicide application matured much more rapidly, so there was a conspicuous difference between the two treatments by the end of the season. The same effect was seen at the Winnipeg site, but was not as dramatic. The 2004 trial did not give as clear results but the same trend was seen in Morden, as the inoculated plots which did not receive fungicide applications matured earlier than those that had fungicide applications. Sackston (1949a) also observed the early ripening phenomenon in his heavily infected experimental plots.

In 2003, sites were inoculated on June 23rd and July 3rd, respectively, for Winnipeg and Morden, and disease was first observed July 8th at the Morden site and

July 16th at the Winnipeg site. The high temperatures may also have hastened maturity in the unsprayed plots, which stopped disease development.

Plots were inoculated later in 2004 (July 7th at the Morden site and July 20th at the Winnipeg site) due to cooler weather slowing plant development. Disease was not observed until July 27th at the Morden site and August 20th at the Winnipeg site. It is possible that due to the delay in seeding and inoculation, which was more pronounced at the Winnipeg site, the disease appeared later with reduced severity, even though humidity conditions were ideal. It may be that the conditions in 2004 were actually more favourable for the disease, but since conditions were less favourable for the plant, and since the season was shorter for the Winnipeg site, the maximum severity of the disease was not reached.

Later seeding in 2004 may also have affected the seed oil content. Since flowering started later in the Winnipeg trials and continued into late August, it is probable that the maximum oil content was never reached. The combination of the cool temperatures which seem to have slowed maturation, along with late flowering, likely led to lower oil contents and smaller differences between the treatments in 2004 at the Winnipeg site.

Ford and Zimmerman (1964) reported that oil content was reduced when seeding was delayed. It has been reported by Sims et al. (1961) that deposition of oil in the seed starts 10 days after flowering and peaks 30 days after flowering, thus late seeding and flowering could potentially reduce oil deposition.

Five fungicide applications were made in 2003, thus a high level of protection was achieved in fungicide application plots. In 2004, only two fungicide applications

were made in order to more closely represent real world conditions and prevent late maturity.

Disease severity ratings are based in part on visual assessments of leaf and stem browning resulting from lesions created by the pathogen. Differences in the amount of brown leaves decreased late in the season between fungicide-treated plots and non- fungicide-treated plots. This was much more pronounced at the Winnipeg site in 2003 and in 2004 at both sites.

The control (NI/F) plots generally had lower final disease severity ratings in 2003 than they did in 2004, possibly due to the reduced number of fungicide applications in 2004. Smaller differences in final severity between plots treated with fungicide and those not treated may also be due to the reduced number of fungicide applications in 2004. Often, the stems of the fungicide treated plots did not show lesions for an extended period, even when the leaves had begun to be heavily infected.

Perryman and Fitt (2000) made the observation that, as the end of the season approached, the difference in leaf browning between fungicide treated and non-fungicide-treated plots decreased. The differences between the treatments, when stem browning was compared, were noticeable.However, the sprayed plots retained more green tissue.

Plant stands were not as thick at the Winnipeg site in 2004 as they were at the Morden site, possibly due to late seeding and cooler temperatures. Initially the stands were similar. As the season progressed, however, the plants in Winnipeg did not branch as much as they did at the Morden site or in the previous years' trials, leaving

a thin plant stand. The thin plant stands likely resulted in less humidity within the canopy, creating an environment that was less favourable for infection. Rashid and Kenaschuck (1998) noted that a dense canopy was important for disease development and this was not seen in Winnipeg in 2004.

4.5.3 Disease Severity

An unexpected result was that some of the highest severity ratings for AC Emerson, AC Linora, Norlin and Vimy at the Morden site in 2004 were seen in the NI/NF plots. It is possible that these plots received extra outside inoculum from a neighbouring trial but if that were the case we would expect the inoculated trial to have also experienced this. With the exception of Vimy, most of the NI/NF treatment final severity ratings were not dramatically higher than those seen in the inoculated plots, so it may be that there was a microclimate effect. It could also be that in the inoculated plots exposure to the disease occurred earlier in the season when there was less leaf matter. This could have resulted in the severity not reaching as high level due to less overall tissue to infect as well as a less favourable microclimate. Thin plant stands in Winnipeg in 2004 may have resulted in less available nutrients and carbohydrates once infections had occurred, as the plants were overall less healthy.

Weather and cultural effect can have pronounced effects on overall variability within and between years and growing sites, as outlined above. The effects of the different treatments on each of the measured variables will now be addressed. Interactions of the yield, oil and protein contents of the seed, and the disease severity measure will also be touched on.

Diseases can have a significant impact on yield and other quality parameters. It is important to understand the relationship between different levels of disease severity and the impact on yield and other economically important measures. It is useful to determine if fungicides could be a beneficial tool for producers to employ under severe infestations of the disease.

4.5.4 Effects of Fungicide Application on Yield

The yield increases with fungicide application compared to the control in 2003 were approximately 50 %. When the control (I/NF) was compared to the treatments in 2004, yields nearly doubled for most cultivars receiving fungicide applications. Lack of protection appears to have the potential to cut yield by at least 50 % under severe pasmo infestations.

Individual farm fields in Manitoba experienced estimated flax yield losses of up to 50 % in 1947 (Sackston, 1959), which is similar to the results seen in the current study. Researchers working in other countries have also noted that pasmo caused a significant reduction in yield (Butler, 1949). Perryman and Fitt (2000) in the United Kingdom observed that when they could associate the yield loss with pasmo infection, leaf infections were associated with a 25.5 % yield loss while stem infection was associated with a 23.7 % yield loss.

4.5.5 Effects of Fungicide Application and Inoculation on Seed Oil Content

Overall seed oil content was significantly higher in the fungicide treatments than in the control in both years at all sites. Seed oil content could be up to 9.43 %

higher for fungicide treatments compared to the control (I/NF). Increases were generally smaller at the Winnipeg site compared to the Morden site in 2004, which may be attributable to late seeding at the Winnipeg site. Sackston and Carson (1951) report that oil content was higher in non-inoculated plots than in inoculated plots. This may be due to reduced photosynthetic area and disease induced premature ripening.

4.5.6 Effects of Fungicide Application and Inoculation on Seed Protein Content

Seed protein content did not show a strong tendency to increase or decrease across sites and years. At Morden there were very few cultivars for which any of the treatments gave significantly higher seed protein contents than the control in either year.

At the Winnipeg site the application of fungicide significantly increased the seed protein content over the control in 2003 for all cultivars. In 2004 most of the treatments produced lower seed protein contents compared to the control. In Winnipeg the seed protein contents for NI/F treatment were all significantly lower than the control, while those of the NI/NF treatment were not significantly different. For the I/F treatment the seed protein contents were significantly lower for AC Emerson, McGregor, and NorLin, while the other cultivars showed no differences.

The lower protein contents observed in the fungicide protected plots in Morden in 2003 and Winnipeg in 2004 may be a normal response of the plants. As yields increase, protein contents are known to decrease, owing to a negative correlation between yield and protein, found in many crops.

4.5.7 Correlations Between Variables

In the current study significant negative correlations between yield and AUDPC was seen in Morden in 2003 but were not seen in 2004 or at the Winnipeg site in either year. Perryman and Fitt (2000) found that the amount of leaf area turned brown after infection was often was correlated to yield decrease. Sackston (1947a) and Ferguson et al. (1987) also found a negative correlation between the severity of the disease and the yield. However, when infections were severe, the correlation between yield loss and symptom severity was no longer observable (Sackston, 1959).

The lack of correlation between yield and AUDPC in 2004 may in part have been due to the low severity observed in 2004. Under low disease severity, weather factors may have had more of an impact on yield than the disease itself. Sackston (1959) noted that although there was reported cultivar resistance to pasmo, visual assessment of the disease was not a direct indication of the effect of the disease on yield. Cultivars with the same level of infection may in fact vary noticeably in their yield response to the disease. This may explain why there were no significant correlations in Winnipeg or in Morden in 2004.

In 2003 at the Morden site, there was a significant positive correlation between yield and kernel weight. In 2004 at the Winnipeg site the correlation was positive, but was not as strong as in the previous year. This seems logical, as bigger, plumper seeds are associated with higher yield. Ferguson et al. (1987) found that there was a significant positive correlation between yield and seed weight and that seed weight was the most important component of yield. Sackston (1947a, 1959) also observed that yield reductions were mainly the result of seed weight and size

reductions rather than reductions in seed numbers. This was not the case, however, when pedicels were weakened and seed was lost due to boll drop (Sackston, 1959). Perryman and Fitt (2000) found that the seed weight was increased when fungicides were applied and that this had a positive effect on yield.

Oil content and AUDPC were significantly negatively correlated at the Morden site in 2004. The correlations were not significant in 2003 or at the Winnipeg site in either year. Sackston (1959) found that heavily infected plants produced seeds with a lower oil content when compared to healthy plants' seed.

The oil and protein content of the flax seed were significantly associated in all years and at all sites and the correlation was always negative. The negative correlation seen suggests that the plant was sacrificing oil production in the seed in order to have more protein. It may be that in these interactions the plant may not have had enough time or enough photosynthetic resources to deposit as much oil in the seed as it might otherwise have. Naqvi et al. (1987) and Oomah and Mazza (1993) reported a significant negative correlation between oil and protein content in flax.

A significant positive association was seen between the oil content and 1000 kernel weight at the Morden site in 2003 and at the Winnipeg site in 2004. The positive association between oil content and kernel weight seems logical as we would expect larger seeds to contain more oil. Sackston and Carson (1951) found that seed size and oil content were generally positively correlated. They found that oil content and seed size were generally affected in the same way by environmental factors.

The correlation between yield and oil content was positive in 2003 but was negative in 2004. Since the location was overall not statistically significant in the

model in 2003, the correlation may not be as meaningfull as if the locations had been pooled for that year. Sackston and Carson (1951) found that there were generally positive correlations between yield and oil content, with most of them being significant. They also had one cultivar and year where the correlation was negative. This suggests that the overall expected correlation between yield and oil content is positive. Under certain conditions, possibly cool weather as was seen in 2004, the correlation can be negative.

Yield and protein content were negatively associated in both years in Morden, with the association being slightly stronger in 2003. Dybing and Lay (1982), reported that they found a negative correlation between yield and protein content, but that it was not significant. They suggest that the reduction in protein content is due to the plant devoting more photosynthetic resources to oil content in the seed for a given yield.

4.6 CONCLUSIONS

The application of fungicides was observed to significantly improve the measured quality characteristics in both years for most of the 6 cultivars. Under severe infestations the use of a fungicide could prove beneficial to flax producers.

Fungicide protection of flax from the pasmo disease can have dramatic effects on yield. Measured average yield differences in 2003 between I/NF (control) and NI/F treated plots reached 95.5 %. Maximum relative yield differences between the control and the NI/F treatment reached 267.9 % at the Winnipeg site and 167 % at the Morden site.

When the NI/F treatment was compared to the control, relative seed oil content was increased by up to 5.87 % in 2003, and by up to 9.43 % at the Winnipeg site in 2004.

Average seed protein content was variable between years and sites, but the cultivar McGregor tended to show increases in relative seed protein content up to 10.92 % under the I/F treatment when compared to the control.

Area under the disease progress curve (AUDPC) was decreased by a maximum of 55% in 2003 in the fungicide-treated plots. In 2004 AUDPC in the NI/F treatment was decreased by up to 40.9 % at the Morden site compared to the non-treated control.

Average 1000 kernel weight was significantly affected by the disease. The relative difference in average 1000 kernel weight between the non-treated control and NI/F treatments reached 42.2 % in 2003. In 2004, 1000 kernel weights tended to be

highest under the NI/F treatment, producing kernel weight up to 60.44 % higher than those of the control.

The effects of weather and growing site were also observed to have significant impacts on the quality characteristics of the selected cultivars. Microclimate may have impacted both the flax plants and the pathogen, resulting in some of the observed variabilty between growing sites and years.

5. GENERAL CONCLUSION

This is the first report of the usage of molecular tools to determine the population structure of *S. linicola* in Manitoba. The effects of fungicides and inoculation with the pathogen on flax under field conditions were also studied. Six flax cultivars were chosen to highlight the effects of the disease as well as the potential benefits of fungicide protection of flax from the pathogen.

The results provided by AMOVA (anlaysis of molecular variance) as well as from the phylogenetic tree generated from the polymorphism data suggest that it is highly plausible that sexual reproduction is occurring in the two populations studied from Manitoba. From the groupings shown in the phylogenetic tree it seemed likely that both sexual and asexual reproduction was occurring. The low PhiPT values obtained from analysis of the entire population from two different locations as well as the four sub-populations from these two locations suggested that the populations were part of one larger population, but that there was a small amount of genetic difference between locations. The comparison of sub-populations within locations suggested that populations within locations were nearly genetically identical.

With only one sampling time during the season, and this being late in the season just prior to harvest, it is difficult to determine exactly when and with what frequency each type of reproduction is occurring. The contributions of ascospores and pycnidiospores to the yearly pasmo infestations in Manitoba are therefore unknown. It is likely that ascopore production is occurring in the spring and perhaps throughout the growing season, with pycnidiospore production predominating during the growing

season. Stubble or straw that has overwintered seems to be the predominant source of inoculum for new epidemics with both the sexual and asexual reproductive structures having the ability to overwinter on these materials and infect new plants in the spring.

The presence of sexual reproduction in Manitoba has implications for the long term management of the disease in the province. If longer range sexual spores are being produced on a regular basis, the cultural control methods available to the farmer are reduced. Rotation away from flax is less successful as a control method if large numbers of spores are being introduced into a field from outside inoculum sources. Resistance in the host, where it exists, can be overcome more rapidly by a sexually reproducing pathogen population than one that is clonal.

Data obtained during the current study suggests that the protection of flax plants from pasmo using fungicides provides marked increases in yield as well as maintaining oil contents when fungicide protected plots are compared to inoculated plots. A significant reduction in the severity of pasmo infestations was also observed. Further investigations into fungicides that provide a high level of protection could be warranted depending on the costs of the fungicide and the price received for flax by the farmer. The fungicide used in the current study, Headline, might be a good choice for further research given the positive results seen in this study.

End use markets will also determine the level of interest shown by producers in fungicides. Flax seed used for neutraceutical and food production markets may be less desirable if it has received fungicide applications. Thus the end use market will likely drive the decisions of producers if a fungicide should ever be registered for flax.

None of the six flax cultivars tested in this study showed a marked resistance to pasmo. Within the commercial cultivars currently being grown there is no good source of resistance to pasmo. If infestations were to become severe due to a predominance of weather that is more favourable to the pathogen, producers are likely to seek out fungicide protection as a means of maintaining yield and other quality characteristics. As there are currently no fungicides registered for flax in Manitoba, severe infestations could potentially have a large impact on flax producers, and on the flax production industry in Manitoba as a whole.

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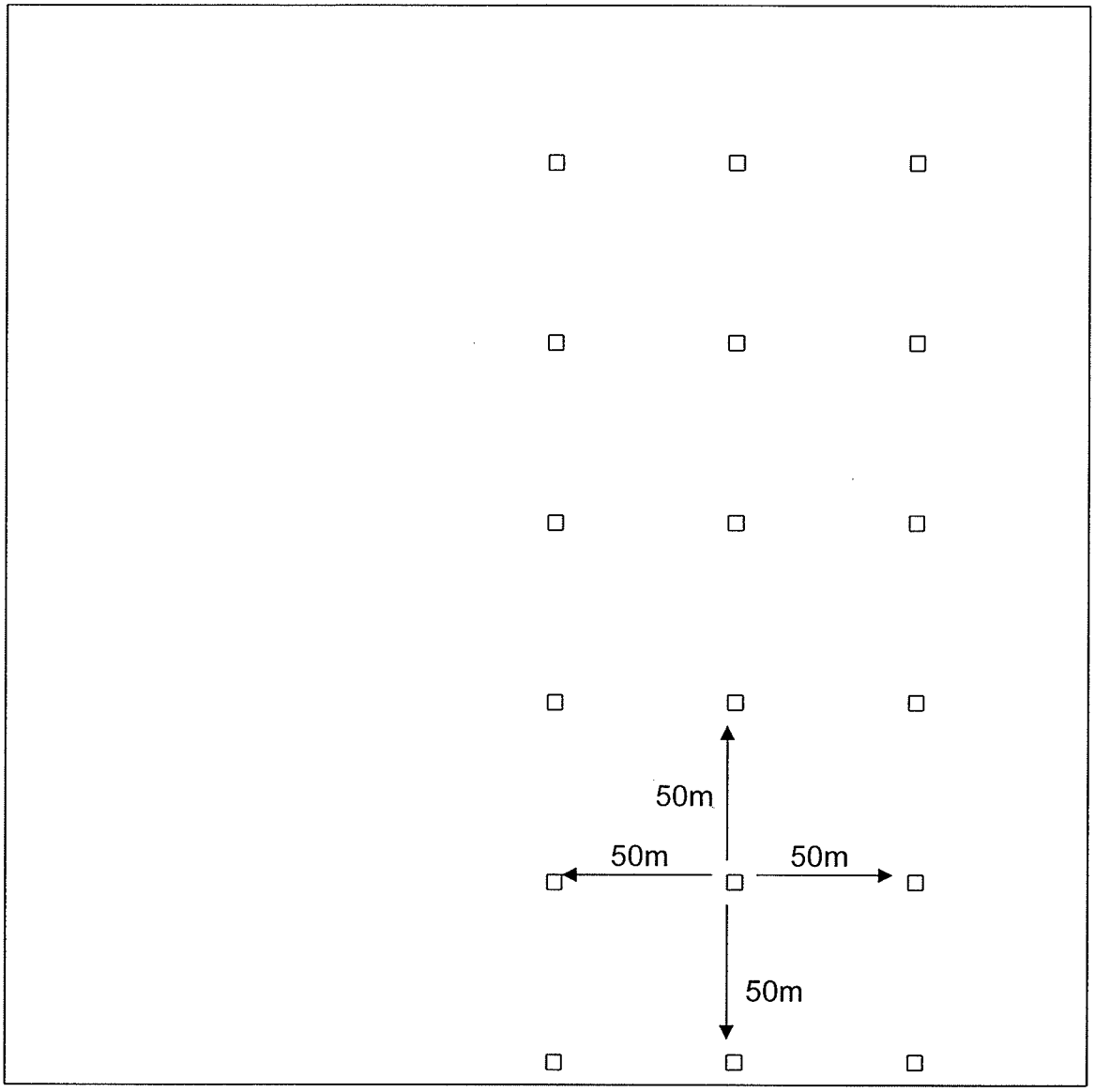
7. APPENDICES

APPENDIX 1: Layout of systematic sampling grid used in commercial fields for isolate collection



APPENDIX 2. Isolates, number designations and site origin of isolates used in RAPD fingerprinting.[1]

Number	Isolate	Site
P001	P-5-4-1-1	Portage la Prairie
P002	P-5-3-30-2	Portage la Prairie
P003	P-4-3-9-3	Portage la Prairie
P004	P-5-3-36-2	Portage la Prairie
P005	P-4-1-2-3	Portage la Prairie
P006	P-3-2-2-1	Portage la Prairie
P007	P-5-3-35-2	Portage la Prairie
P008	P-5-3-40-1	Portage la Prairie
P009	P-5-1-35-2	Portage la Prairie
P010	P-6-5-4-1	Portage la Prairie
P011	P-4-3-1-1	Portage la Prairie
P013	P-5-1-1-1	Portage la Prairie
P014	P-5-1-8-2	Portage la Prairie
P016	P-6-6-1-1	Portage la Prairie
P017	P-6-3-3-1	Portage la Prairie
P018	P-1-3-4-1	Portage la Prairie
P019	P-2-3-14-2	Portage la Prairie
P022	P-2-3-11-3	Portage la Prairie
P023	P-2-3-17-2	Portage la Prairie
P025	P-1-2-5-2	Portage la Prairie
P026	P-4-5-2-1	Portage la Prairie
P027	P-3-4-2-1	Portage la Prairie
P028	P-3-1-4-1	Portage la Prairie
P029	P-5-3-31-2	Portage la Prairie
P030	P-5-5-1-1	Portage la Prairie
P031	P-2-3-27-1	Portage la Prairie
P032	P-2-3-25-1	Portage la Prairie
P033	P-5-6-3-1	Portage la Prairie
P034	P-3-6-2-1	Portage la Prairie
P035	P-1-5-5-1	Portage la Prairie
P036	P-4-5-3-2	Portage la Prairie
P037	P-6-5-5-1	Portage la Prairie
P038	P-1-6-2-1	Portage la Prairie
P039	P-3-1-2-1	Portage la Prairie
P040	P-5-3-27-1	Portage la Prairie
P041	P-5-3-28-1	Portage la Prairie
P042	P-1-6-3-1	Portage la Prairie
P043	P-2-1-4-1	Portage la Prairie
P044	P-5-3-38-1	Portage la Prairie
P045	P-2-6-2-1	Portage la Prairie
P046	P-2-3-30-1	Portage la Prairie

APPENDIX 2. Isolates, number designations and site origin of isolates used in RAPD fingerprinting.[1]

Number	Isolate	Site
P047	P-2-2-2-1	Portage la Prairie
P048	P-5-3-2-2	Portage la Prairie
P049	P-2-3-20-1	Portage la Prairie
P050	P-6-4-3-2	Portage la Prairie
P051	P-5-6-1-2	Portage la Prairie
P052	P-2-3-23-1	Portage la Prairie
P053	P-2-2-3-1	Portage la Prairie
P054	P-2-3-22-1	Portage la Prairie
P055	P-2-3-21-1	Portage la Prairie
P056	P-2-3-24-1	Portage la Prairie
P057	P-5-3-32-1	Portage la Prairie
P058	P-5-4-3-1	Portage la Prairie
P059	P-5-3-11-2	Portage la Prairie
P060	P-5-3-10-2	Portage la Prairie
P061	P-5-3-3-3	Portage la Prairie
P062	P-6-1-2-3	Portage la Prairie
P063	P-4-4-2-1	Portage la Prairie
P064	P-3-4-1-1	Portage la Prairie
P065	P-3-2-3-3	Portage la Prairie
P066	P-5-2-3-1	Portage la Prairie
P067	P-4-6-2-3	Portage la Prairie
P068	P-4-6-1-1	Portage la Prairie
P069	P-6-2-1-1	Portage la Prairie
P070	P-5-3-5-1	Portage la Prairie
P071	P-5-5-2-1	Portage la Prairie
P072	P-6-4-3-3	Portage la Prairie
P073	P-3-6-1-1	Portage la Prairie
P074	P-4-2-2-3	Portage la Prairie
P075	P-5-3-1-3	Portage la Prairie
P076	P-4-2-2-1	Portage la Prairie
P077	P-2-5-3-3	Portage la Prairie
P078	P-4-4-5-1	Portage la Prairie
P079	P-2-1-3-3	Portage la Prairie
P080	P-6-5-38-1	Portage la Prairie
P081	P-4-2-2-2	Portage la Prairie
P082	P-5-3-29-1	Portage la Prairie
P083	P-5-3-12-3	Portage la Prairie
P084	P-2-5-6-3	Portage la Prairie
P085	P-6-3-2-1	Portage la Prairie
P086	P-2-3-25-3	Portage la Prairie
P087	P-6-2-3-3	Portage la Prairie

APPENDIX 2. Isolates, number designations and site origin of isolates used in RAPD fingerprinting.[1]

Number	Isolate	Site
P088	P-1-5-2-1	Portage la Prairie
P089	P-1-1-3-1	Portage la Prairie
P090	P-2-3-13-1	Portage la Prairie
P091	P-2-3-9-1	Portage la Prairie
P092	P-2-3-32-1	Portage la Prairie
P094	P-5-3-6-1	Portage la Prairie
P095	P-2-3-31-1	Portage la Prairie
P096	P-5-3-13-1	Portage la Prairie
P097	P-5-2-1-2	Portage la Prairie
P098	P-2-5-3-2	Portage la Prairie
P099	P-2-4-1-1	Portage la Prairie
P100	P-2-3-34-1	Portage la Prairie
P101	P-1-1-1-2	Portage la Prairie
P102	P-1-3-1-2	Portage la Prairie
P103	P-2-3-26-1	Portage la Prairie
P104	P-2-5-3-1	Portage la Prairie
P105	P-2-3-17-3	Portage la Prairie
P106	P-3-3-4-1	Portage la Prairie
S107	S-2-5-1-1	Sanford
S108	S-2-3-1-2	Sanford
S109	S-3-6-4-1	Sanford
S110	S-3-2-29-1	Sanford
S111	S-2-2-2-2	Sanford
S112	S-3-5-1-1	Sanford
S113	S-5-2-1-1	Sanford
S114	S-4-1-2-2	Sanford
S115	S-3-2-30-1	Sanford
S116	S-3-2-20-2	Sanford
S117	S-3-2-15-1	Sanford
S118	S-1-6-2-1	Sanford
S119	S-6-3-3-1	Sanford
S120	S-3-2-33-1	Sanford
S121	S-3-3-1-1	Sanford
S122	S-3-2-12-1	Sanford
S123	S-3-2-2-2	Sanford
S124	S-3-2-23-1	Sanford
S125	S-4-2-1-2	Sanford
S126	S-4-2-4-2	Sanford
S127	S-6-5-30-1	Sanford
S128	S-6-5-18-1	Sanford
S129	S-1-1-3-2	Sanford

APPENDIX 2. Isolates, number designations and site origin of isolates used in RAPD fingerprinting.[1]

Number	Isolate	Site
S130	S-3-2-24-1	Sanford
S131	S-4-6-2-2	Sanford
S132	S-5-1-7-1	Sanford
S133	S-3-2-26-1	Sanford
S134	S-5-1-3-2	Sanford
S135	S-6-5-24-3	Sanford
S136	S-6-5-19-3	Sanford
S137	S-6-5-25-1	Sanford
S138	S-6-2-4-1	Sanford
S139	S-5-6-6-1	Sanford
S140	S-6-5-20-3	Sanford
S141	S-6-5-29-2	Sanford
S142	S-6-5-45-1	Sanford
S143	S-5-6-3-1	Sanford
S144	S-2-3-24-1	Sanford
S145	S-6-5-36-3	Sanford
S146	S-6-6-3-1	Sanford
S147	S-6-5-35-3	Sanford
S148	S-6-5-17-1	Sanford
S149	S-1-3-5-2	Sanford
S150	S-1-5-4-2	Sanford
S151	S-1-4-2-3	Sanford
S152	S-3-4-2-1	Sanford
S153	S-6-5-44-1	Sanford
S154	S-6-5-42-1	Sanford
S155	S-1-4-1-1	Sanford
S156	S-1-2-4-2	Sanford
S157	S-1-2-1-3	Sanford
S158	S-1-5-1-1	Sanford
S159	S-1-1-1-2	Sanford
S160	S-6-3-2-1	Sanford
S161	S-3-2-32-1	Sanford
S162	S-3-2-19-2	Sanford
S164	S-6-2-1-1	Sanford
S165	S-4-6-3-1	Sanford
S166	S-6-6-5-1	Sanford
S167	S-5-3-4-1	Sanford
S168	S-3-6-2-1	Sanford
S169	S-6-4-4-2	Sanford
S170	S-2-3-4-1	Sanford
S171	S-2-6-4-2	Sanford

APPENDIX 2. Isolates, number designations and site origin of isolates used in RAPD fingerprinting.[1]

Number	Isolate	Site
S172	S-4-3-4-3	Sanford
S173	S-4-4-5-1	Sanford
S174	S-2-6-1-1	Sanford
S175	S-2-1-1-1	Sanford
S176	S-3-2-31-1	Sanford
S177	S-3-2-10-1	Sanford
S178	S-3-3-2-1	Sanford
S179	S-4-1-1-1	Sanford
S180	S-4-4-1-1	Sanford
S181	S-2-6-3-1-2	Sanford
S182	S-3-2-8-2	Sanford
S183	S-3-2-25-3	Sanford
S184	S-4-5-9-2	Sanford
S185	S-3-2-18-1	Sanford
S186	S-3-2-17-1	Sanford
S187	S-2-5-2-1	Sanford
S188	S-3-2-21-2	Sanford
S189	S-4-5-3-1-2	Sanford
S190	S-3-2-34-1	Sanford
S191	S-5-4-4-1	Sanford
S192	S-4-3-2-3	Sanford
S193	S-3-2-36-1	Sanford
S194	S-3-2-9-2	Sanford
S195	S-3-2-35-2	Sanford
S196	S-3-2-39-1	Sanford
S197	S-6-4-3-3	Sanford

[1] Isolates are named by site, row, station within the row, pycnidia from which they were selected, and single spore.

APPENDIX 3: RAPD binary scores of 163 isolates of *S. linicola* collected at different field stations in Portage la Prairie, Manitoba, Canada, and Sanford, Manitoba, Canada using different primers.

Appendix 3 a: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 608.

	Marker																
Isolate	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P001	0	1	0	0	1	0	0	1	1	0	0	0	1	0	0	1	1
P003	0	1	1	0	1	1	0	1	1	0	0	0	1	0	0	1	1
P004	0	1	1	0	1	0	1	1	1	0	0	0	1	0	0	1	1
P006	0	1	0	0	1	0	0	1	0	0	0	0	1	0	0	1	1
P007	1	0	0	0	1	1	0	1	1	0	0	0	1	0	0	1	1
P008	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
P009	1	1	1	0	1	1	0	1	1	0	0	0	1	0	1	1	1
P010	0	1	1	0	1	0	0	1	0	0	0	0	1	0	1	1	1
P011	0	1	1	0	1	1	0	1	1	0	1	0	1	0	1	1	1
P013	0	1	1	0	1	0	0	1	1	0	0	0	1	0	0	0	1
P016	0	1	1	0	1	0	0	1	1	0	1	0	1	0	1	1	1
P017	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
P018	0	1	1	0	1	0	0	1	1	1	1	0	1	0	0	1	1
P019	0	1	0	0	1	0	0	1	0	0	1	0	1	0	0	1	1
P022	0	1	0	0	1	0	0	1	0	0	0	0	1	0	0	0	1
P023	0	1	1	0	1	0	1	1	1	0	1	0	1	0	0	1	1
P024	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
P026	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
P027	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
P028	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	0	1
P029	0	1	1	0	1	0	0	1	1	0	0	0	1	0	0	1	1
P030	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
P031	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
P032	0	1	0	0	1	0	0	1	1	0	0	0	1	0	0	1	1
P033	0	1	1	0	0	1	0	1	0	0	0	0	1	0	0	1	1
P034	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
P036	0	1	1	0	0	0	0	1	0	0	0	0	1	0	0	1	1

Appendix 3 a: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 608.

	Marker																
Isolate	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P037	0	1	1	0	0	0	0	1	0	0	0	0	1	0	0	1	1
P038	0	1	1	0	1	0	0	1	1	0	0	0	1	0	0	1	1
P039	0	0	1	0	0	0	0	1	0	0	0	0	1	0	0	0	1
P040	0	1	1	0	1	0	0	1	1	0	1	0	1	0	1	1	1
P041	0	1	0	0	1	0	0	1	1	0	0	0	1	1	1	1	1
P042	0	1	1	0	0	0	0	1	1	0	1	0	1	0	0	1	1
P043	0	1	0	0	1	0	0	1	0	0	0	0	1	0	1	0	1
P044	0	1	1	0	0	0	0	1	0	0	1	0	1	0	0	1	1
P045	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
P046	0	1	1	0	1	0	1	1	1	0	0	0	1	0	0	1	1
P047	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	1	1
P048	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	1	1
P049	0	1	1	0	1	1	0	1	1	0	1	0	1	0	1	1	1
P051	0	1	1	0	1	0	1	1	1	0	1	0	1	0	1	1	1
P052	0	1	1	0	1	0	1	1	1	0	0	0	1	0	1	1	1
P053	0	1	1	1	1	0	1	1	1	0	1	0	1	0	0	1	1
P054	0	1	1	0	1	0	1	1	1	0	0	0	1	0	1	1	1
P055	0	1	1	0	1	0	1	1	1	0	0	0	1	0	1	1	1
P056	0	1	1	0	1	0	1	1	1	1	0	0	1	0	1	1	1
P057	0	0	0	0	1	0	0	1	0	0	0	0	1	0	1	1	1
P058	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	1	1
P059	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	1	1
P060	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	1	1
P061	0	1	1	0	1	0	1	1	1	0	1	0	1	0	0	1	1
P062	0	1	1	0	1	0	0	1	1	0	1	0	1	0	1	1	1
P065	0	1	1	0	1	0	0	1	1	0	0	0	1	0	0	1	1
P066	0	1	1	0	1	0	0	1	1	0	0	0	1	0	0	1	1
P067	0	1	1	0	1	1	0	1	1	0	1	0	1	0	1	1	1
P068	0	1	1	0	1	1	0	1	1	1	0	1	0	0	0	0	1
P069	0	1	1	0	1	0	0	1	1	0	0	0	1	0	0	1	1

Appendix 3 a: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 608.

	Marker																
Isolate	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P070	0	1	1	0	1	0	0	1	1	0	0	0	1	1	0	1	1
P072	0	1	1	0	1	0	0	1	1	0	0	0	1	0	0	1	1
P073	0	1	1	0	1	1	0	1	1	0	0	0	1	0	1	1	1
P075	0	1	1	0	0	0	0	1	0	0	0	0	1	0	0	1	1
P076	0	1	1	0	1	0	0	1	0	1	0	0	1	0	0	1	1
P077	0	1	1	0	1	0	0	1	0	0	1	0	1	0	0	1	1
P078	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	1	1
P079	1	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
P080	1	1	1	0	1	0	0	1	1	0	0	0	1	0	0	1	1
P081	0	1	1	0	0	0	0	1	0	0	0	0	1	0	0	0	1
P082	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	1	1
P083	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	1	1
P085	0	1	1	0	1	0	0	1	1	0	0	0	1	0	0	1	1
P087	0	1	0	1	1	0	0	1	1	0	0	0	1	0	0	1	1
P088	0	1	1	0	1	1	0	1	0	0	0	0	1	0	0	0	1
P089	0	1	1	0	1	1	0	1	0	0	0	0	1	0	0	1	1
P090	0	0	0	1	1	0	0	0	0	0	0	0	1	0	0	1	1
P091	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1
P092	0	1	1	0	1	1	0	1	1	0	0	0	1	0	0	1	1
P094	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	1	1
P097	0	1	1	0	0	0	0	1	1	0	0	0	1	0	0	0	1
P099	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1
P100	0	0	0	0	1	1	0	1	0	0	1	0	1	0	0	1	1
P101	0	1	1	0	1	0	0	1	0	0	1	0	1	0	1	0	1
P102	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1	1
P104	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
P105	0	1	1	0	1	0	0	1	0	0	1	0	1	0	0	1	1
P106	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	1	1
S107	0	1	1	0	1	0	0	1	0	0	1	0	1	0	0	1	1
S108	0	0	0	0	1	0	0	0	0	0	0	0	1	1	0	1	1

Appendix 3 a: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 608.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S109	0	1	1	0	1	0	0	1	0	0	1	0	1	0	0	1	1
S111	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1
S112	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
S113	0	1	1	0	1	0	0	1	1	0	1	0	1	0	1	1	1
S114	0	1	1	0	1	0	0	1	0	0	0	0	1	0	0	0	1
S115	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
S116	0	1	1	0	0	1	0	1	0	0	1	0	1	0	0	1	1
S117	0	1	1	0	1	0	0	1	1	0	1	0	1	0	1	1	1
S118	0	1	1	0	1	1	0	1	1	0	1	0	1	0	1	1	1
S119	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
S120	0	1	1	0	1	1	0	1	1	0	0	0	1	0	1	1	1
S121	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	0	1
S122	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	1
S123	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	1	1
S124	0	0	1	0	0	0	0	0	0	0	1	0	1	1	0	1	1
S125	0	1	0	0	1	1	0	1	1	0	1	0	1	0	1	1	1
S126	0	1	1	0	1	1	0	1	1	0	1	0	1	0	1	1	1
S127	0	1	1	0	1	1	0	1	1	0	1	0	1	0	0	1	1
S128	0	1	1	0	1	1	0	1	1	0	1	0	1	0	1	1	1
S130	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
S132	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1
S134	0	1	1	0	1	0	0	1	0	0	1	0	1	0	0	1	1
S135	0	1	1	0	1	0	0	1	0	0	1	0	1	0	0	0	1
S136	0	0	1	0	1	0	0	0	0	0	0	0	1	0	0	0	1
S137	0	1	1	0	1	0	0	1	0	0	1	0	1	0	0	1	1
S138	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	1
S139	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
S140	0	1	1	0	0	0	0	1	0	0	0	0	1	0	0	1	1
S141	0	1	1	0	1	0	1	1	0	0	1	0	0	0	0	1	1
S142	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1

Appendix 3 a: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 608.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S143	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1
S144	0	1	1	0	0	0	1	1	0	0	0	0	1	0	0	1	1
S146	0	1	1	0	1	0	0	1	0	0	1	0	1	0	0	1	1
S147	0	1	1	0	1	1	0	1	0	0	1	0	1	0	0	1	1
S149	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	1	1
S150	0	1	1	0	0	0	1	0	0	0	0	0	1	0	1	1	1
S151	0	1	0	0	1	0	1	1	1	0	0	0	1	0	1	1	1
S152	0	1	1	0	1	1	0	1	1	0	1	0	1	0	1	1	1
S154	0	1	1	0	1	1	1	1	1	0	0	0	1	0	0	1	1
S155	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1
S156	0	1	1	0	1	1	1	1	1	0	1	0	1	0	0	1	1
S157	0	1	0	0	1	0	1	1	1	0	0	0	1	0	0	1	1
S159	0	0	1	0	0	0	1	0	0	0	0	0	1	0	0	0	1
S160	0	1	1	0	0	0	1	0	0	0	0	0	1	0	1	1	1
S161	0	1	1	0	1	1	1	1	1	0	1	0	1	0	1	1	1
S162	0	1	1	0	1	1	0	1	1	0	1	0	1	0	1	1	1
S164	0	1	1	0	0	0	1	0	0	0	0	0	1	0	1	1	1
S165	0	1	1	0	0	0	1	1	0	0	0	0	1	0	1	1	1
S166	0	1	1	0	0	1	1	0	1	1	0	0	1	0	1	1	1
S169	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1
S171	0	0	1	0	1	0	0	0	0	0	1	0	1	0	0	1	1
S172	0	0	1	0	0	1	0	0	0	0	0	0	1	0	0	1	1
S173	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0
S174	0	0	1	0	1	0	1	1	0	0	1	0	1	0	0	1	1
S176	1	0	1	0	1	0	0	1	0	0	1	0	1	0	0	1	1
S177	0	0	1	0	1	0	0	1	1	0	0	0	1	0	0	0	1
S178	0	0	1	0	1	0	0	0	0	0	1	0	1	0	1	1	1
S179	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1
S180	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	0	1
S181	0	0	1	0	1	0	0	0	0	0	1	0	1	0	1	1	1

Appendix 3 a: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 608.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S182	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	0	1
S183	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	1	1
S184	0	1	1	0	1	0	0	0	0	0	1	0	1	0	1	0	1
S185	0	0	0	0	0	0	0	1	1	0	1	0	1	0	0	1	1
S186	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
S187	0	1	1	0	1	0	0	1	1	0	1	0	1	0	0	1	1
S188	0	0	1	0	1	0	0	0	0	0	0	0	1	0	0	0	1
S189	0	1	0	0	0	0	0	1	0	0	0	0	1	0	0	0	1
S190	0	0	0	0	0	0	0	0	0	0	1	0	1	0	0	0	1
S191	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	1	1
S192	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	1
S193	0	0	0	0	1	1	1	1	1	0	1	0	1	0	0	1	1
S194	0	0	0	0	1	1	0	1	1	0	1	0	1	0	0	1	1
S195	0	0	0	0	1	1	1	1	0	0	0	0	1	0	0	1	1
S196	0	0	0	0	1	1	0	0	1	0	1	0	1	0	0	1	1
S197	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	1	1

Appendix 3 a: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 608.

	Marker				Marker				Marker		
Isolate	18	19	20	Isolate	18	19	20	Isolate	18	19	20
P001	0	0	0	P038	0	0	0	P070	0	1	0
P003	0	0	0	P039	0	0	0	P072	0	0	0
P004	0	1	0	P040	0	1	1	P073	0	1	1
P006	0	0	0	P041	0	1	0	P075	0	0	0
P007	0	0	0	P042	0	1	0	P076	0	0	0
P008	0	0	0	P043	0	0	1	P077	0	0	0
P009	0	0	0	P044	0	0	1	P078	0	0	0
P010	0	0	0	P045	0	0	0	P079	0	0	0
P011	0	1	0	P046	0	0	1	P080	0	0	0
P013	0	1	0	P047	0	0	1	P081	0	0	0
P016	0	1	1	P048	0	0	1	P082	0	0	0
P017	0	1	0	P049	1	1	0	P083	0	0	0
P018	0	1	0	P051	1	1	0	P085	0	0	0
P019	0	0	0	P052	0	1	0	P087	0	0	0
P022	0	0	0	P053	0	0	0	P088	0	0	0
P023	0	0	0	P054	0	1	0	P089	0	0	0
P024	0	0	0	P055	0	1	0	P090	0	0	0
P026	0	0	0	P056	0	1	0	P091	0	0	0
P027	0	0	0	P057	0	0	0	P092	0	1	0
P028	0	0	0	P058	0	0	0	P094	0	0	0
P029	0	0	0	P059	0	0	0	P097	0	0	0
P030	0	0	0	P060	0	0	0	P099	0	0	0
P031	0	0	0	P061	0	0	0	P100	0	0	0
P032	0	0	0	P062	0	0	0	P101	0	0	0
P033	0	0	0	P065	0	1	0	P102	0	0	0
P034	0	0	0	P066	0	0	0	P104	0	0	0
P035	0	0	0	P067	0	1	1	P105	0	0	0
P036	0	0	0	P068	1	0	0	P106	0	0	0
P037	0	0	0	P069	0	0	0	S107	0	0	0

Appendix 3 a: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 608.

	Marker										
Isolate	18	19	20	Isolate	18	19	20	Isolate	18	19	20
S108	0	0	0	S141	0	0	0	S179	0	0	0
S109	0	0	0	S142	0	0	0	S180	0	0	0
S111	0	0	0	S143	0	0	0	S181	0	1	0
S112	0	0	0	S144	0	0	0	S182	0	0	0
S113	0	0	0	S146	0	0	0	S183	0	0	0
S114	0	0	0	S147	0	0	0	S184	0	0	0
S115	0	0	0	S149	0	0	0	S185	0	0	0
S116	0	0	0	S150	0	0	0	S186	0	0	0
S117	0	0	0	S151	0	0	0	S187	0	0	0
S118	0	0	0	S152	0	0	0	S188	0	0	0
S119	0	0	0	S154	0	0	0	S189	0	0	0
S120	0	0	0	S155	0	0	0	S190	0	0	0
S121	0	0	0	S156	0	0	0	S191	0	0	0
S122	0	0	0	S157	0	0	0	S192	0	0	0
S123	0	0	0	S159	0	0	0	S193	0	0	0
S124	0	0	0	S160	0	0	0	S194	0	0	0
S125	0	0	0	S161	0	0	0	S195	0	0	0
S126	0	0	0	S162	0	0	0	S196	0	0	0
S127	0	0	0	S164	0	0	0	S197	0	0	0
S128	0	0	0	S165	0	0	0				
S130	0	0	0	S166	0	0	0				
S132	0	0	0	S169	0	0	0				
S134	0	0	0	S171	0	0	0				
S135	0	0	0	S172	0	0	0				
S136	0	0	0	S173	0	0	0				
S137	0	0	0	S174	0	0	0				
S138	0	0	0	S176	0	0	0				
S139	0	0	0	S177	0	0	0				
S140	0	0	0	S178	0	0	1				

Appendix 3 b: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 522.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12
P001	0	1	0	1	1	1	1	1	0	0	0	0
P003	1	1	0	1	1	1	0	0	0	0	0	0
P004	1	1	1	1	1	1	1	1	0	1	0	0
P006	0	1	0	1	1	1	0	1	0	1	0	0
P007	0	1	0	1	1	1	1	1	0	1	0	0
P008	1	1	1	1	1	1	1	1	0	1	0	0
P009	1	1	0	1	1	1	0	1	0	0	0	0
P010	1	1	1	1	1	1	1	1	0	1	0	0
P011	0	1	1	1	1	1	1	1	0	1	0	0
P013	1	1	0	1	1	1	0	1	0	1	0	0
P016	0	1	0	1	1	1	1	0	0	1	0	0
P017	0	1	0	1	1	1	1	0	0	1	0	0
P018	1	1	0	1	1	1	0	1	1	1	0	0
P019	0	1	0	1	1	1	1	1	0	1	0	0
P022	1	1	0	1	1	1	1	1	0	1	0	0
P023	0	1	0	1	1	1	1	0	0	1	0	0
P024	0	1	0	1	1	1	0	0	0	0	0	0
P026	1	1	1	1	1	1	1	0	0	1	0	0
P027	1	1	1	1	1	1	1	0	0	1	0	0
P028	0	1	1	1	1	1	1	1	0	1	0	0
P029	0	1	1	1	1	1	1	1	0	1	0	0
P030	1	1	0	1	1	1	0	0	0	1	0	0
P031	1	1	0	1	1	1	0	0	0	1	0	0
P032	1	1	1	1	1	1	1	1	0	1	0	0
P033	1	1	0	1	1	1	0	1	0	1	0	0
P034	1	1	1	0	1	1	1	0	0	1	0	0
P036	1	1	0	1	1	1	0	1	0	1	0	0
P037	0	1	1	1	1	1	1	1	0	1	0	0
P038	1	1	0	1	1	1	0	1	1	1	0	0
P039	0	1	0	1	1	1	0	1	0	1	0	0
P040	1	1	1	1	1	1	1	0	1	1	1	0

Appendix 3 b: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 522.

	Marker											
Isolate	1	2	3	4	5	6	7	8	9	10	11	12
P041	1	1	1	1	1	1	1	0	0	1	0	0
P042	1	1	0	1	1	0	0	1	1	1	0	0
P043	0	1	0	1	1	1	1	1	0	1	0	0
P044	1	1	1	1	1	1	1	1	1	1	0	0
P045	1	1	0	1	1	1	0	1	1	1	0	0
P046	1	1	0	1	1	1	0	1	1	1	0	0
P047	1	1	1	1	1	1	1	1	1	1	0	0
P048	1	1	1	1	1	1	1	1	1	1	0	0
P049	1	1	0	1	1	1	1	1	0	1	0	0
P051	1	1	0	1	1	1	1	1	0	1	0	0
P052	1	1	0	1	1	1	1	1	0	1	0	0
P053	1	1	0	1	1	1	1	1	0	1	0	0
P054	1	1	0	1	1	1	1	1	0	1	0	0
P055	1	1	0	1	1	1	1	1	0	1	0	0
P056	0	1	0	1	1	1	1	1	0	1	0	0
P057	0	0	0	0	1	0	1	0	0	1	0	0
P058	0	1	0	1	1	1	1	1	0	1	0	0
P059	1	0	0	0	1	0	1	0	0	1	0	0
P060	0	0	0	1	1	0	1	1	0	1	0	0
P061	1	1	0	1	1	1	1	1	0	1	0	0
P062	1	1	1	1	1	1	0	0	0	1	0	0
P065	1	1	0	1	1	1	1	1	0	1	0	0
P066	1	1	0	1	1	1	1	1	0	1	0	0
P067	1	1	0	1	1	1	1	1	0	1	0	0
P068	0	1	0	0	1	1	1	1	0	0	1	1
P069	1	1	0	1	1	1	1	0	0	1	0	0
P070	1	1	0	1	1	1	1	1	0	1	0	0
P072	1	1	0	1	1	1	1	1	0	1	0	0
P073	1	1	0	1	1	1	1	1	0	1	0	0
P075	1	1	0	1	1	1	0	0	0	1	0	0

Appendix 3 b: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 522.

Marker / Isolate	1	2	3	4	5	6	7	8	9	10	11	12
P076	1	1	0	1	1	0	0	0	0	1	0	0
P077	1	1	0	1	1	1	0	1	0	1	0	0
P078	1	1	0	1	1	1	1	1	0	1	0	0
P079	1	1	0	1	1	1	1	1	0	1	0	0
P080	1	1	1	1	1	1	1	1	0	1	0	0
P081	0	1	0	1	1	1	0	1	1	1	0	0
P082	1	1	1	1	1	1	1	1	0	1	0	0
P083	0	1	0	1	1	1	1	1	0	1	0	0
P085	1	1	0	1	1	1	1	1	0	0	0	0
P087	1	1	0	1	1	0	1	0	1	1	0	0
P088	1	1	0	1	1	1	1	0	1	0	0	0
P089	1	1	0	1	1	0	1	0	1	1	0	0
P090	0	0	0	0	0	0	0	1	1	0	0	0
P091	1	1	0	1	1	1	0	1	0	0	0	0
P092	1	1	0	1	1	1	0	1	1	1	0	0
P094	1	1	0	1	1	1	1	1	1	1	0	0
P097	0	1	0	1	1	1	1	1	0	0	0	0
P099	0	0	0	0	1	0	0	1	0	0	0	0
P100	1	1	0	1	1	0	0	0	0	1	0	0
P101	1	1	1	1	1	1	1	1	1	1	0	0
P102	1	0	1	0	1	0	0	0	0	0	0	0
P104	1	1	1	1	1	1	1	1	1	1	0	0
P105	1	1	1	1	1	1	1	0	1	1	0	0
P106	1	1	0	1	1	0	0	1	0	1	0	0
S107	1	1	1	1	1	1	1	0	0	1	0	0
S108	1	0	0	1	1	0	1	0	0	1	0	0
S109	1	1	0	1	1	0	1	0	0	1	0	0
S111	1	1	0	1	1	1	0	0	0	1	0	0
S112	0	1	0	1	1	1	1	0	0	1	0	0
S113	1	1	0	1	1	1	1	1	0	1	0	0

Appendix 3 b: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 522.

	Marker											
Isolate	1	2	3	4	5	6	7	8	9	10	11	12
S114	1	1	0	1	1	1	1	0	0	1	0	0
S115	1	1	0	1	1	1	1	0	0	1	0	0
S116	1	1	0	1	1	1	1	1	0	1	0	0
S117	1	1	0	1	1	1	1	1	0	1	0	0
S118	1	1	0	1	1	1	1	1	0	1	0	0
S119	0	1	1	1	1	1	1	1	0	1	0	0
S120	1	1	1	1	1	1	1	0	0	1	0	0
S121	0	1	0	1	1	1	0	1	0	1	0	0
S122	1	0	0	0	1	0	0	0	0	1	0	0
S123	0	0	0	0	1	0	0	0	0	0	0	0
S124	0	0	0	0	1	0	0	1	0	1	0	0
S125	1	1	0	1	1	1	1	1	0	1	0	0
S126	1	1	0	1	1	0	1	1	0	1	0	0
S127	1	1	0	1	1	1	1	1	0	1	0	0
S128	1	1	0	1	1	1	1	1	0	1	0	0
S130	1	1	0	1	1	0	0	1	0	1	0	0
S132	0	1	0	1	1	0	1	0	0	1	0	0
S134	1	1	0	1	1	1	1	0	1	1	0	0
S135	1	1	0	0	1	1	0	0	0	0	0	0
S136	1	1	0	1	1	1	0	1	0	1	0	0
S137	1	1	0	1	1	1	0	1	1	1	0	0
S138	0	0	0	1	1	1	1	0	0	1	0	0
S139	1	1	1	1	1	1	1	1	0	1	0	0
S140	1	1	0	1	1	1	0	1	1	1	0	0
S141	1	1	0	1	1	1	0	1	0	1	0	0
S142	0	1	0	1	1	1	1	1	1	1	0	0
S143	0	1	0	1	1	1	1	1	0	1	0	0
S144	1	0	0	1	1	1	0	1	0	1	0	0
S146	1	1	0	1	1	1	1	0	1	1	0	0
S147	1	1	0	1	1	1	0	0	0	1	0	0

Appendix 3 b: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 522.

Marker												
Isolate	1	2	3	4	5	6	7	8	9	10	11	12
S149	1	1	0	1	1	1	1	1	0	1	0	0
S150	1	1	0	1	1	0	1	0	0	1	0	0
S151	1	1	0	1	1	1	0	1	0	1	0	0
S152	1	1	0	1	1	0	0	1	0	0	0	0
S154	1	1	0	1	1	1	1	0	0	1	0	0
S155	1	1	0	1	1	1	0	0	0	1	0	0
S156	1	1	0	1	1	1	0	0	1	1	0	0
S157	1	1	0	1	1	1	1	0	0	1	0	0
S159	1	1	0	1	1	1	1	0	1	1	1	0
S160	1	1	0	1	1	1	1	0	1	1	0	0
S161	1	1	0	1	1	1	0	1	1	1	1	1
S162	1	1	0	1	1	1	0	0	0	1	0	0
S164	1	1	0	1	1	1	1	0	0	1	1	0
S165	1	1	0	1	1	1	1	0	0	1	1	0
S166	1	1	0	1	1	1	1	0	0	1	1	0
S169	0	0	0	0	0	0	0	0	0	1	0	0
S171	0	1	0	0	1	1	0	1	0	1	1	0
S172	0	1	0	0	1	1	0	1	0	1	0	0
S173	0	1	0	0	1	0	0	0	0	1	0	0
S174	1	1	0	1	1	0	1	1	0	1	0	0
S176	1	1	0	1	1	1	0	1	1	1	0	0
S177	0	1	0	1	1	1	0	1	0	1	0	0
S178	0	1	0	1	1	0	1	0	1	1	0	0
S179	0	1	0	1	1	1	0	0	0	1	0	0
S180	0	0	0	1	1	0	0	0	0	1	0	0
S181	0	1	0	1	1	1	1	1	1	1	0	0
S182	0	0	0	0	1	0	0	1	1	1	0	1
S183	1	0	0	0	0	0	0	0	1	1	0	1
S184	1	1	0	1	1	0	1	0	0	1	0	1
S185	0	0	0	0	0	0	0	0	0	1	0	0

Appendix 3 b: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 522.

	Marker											
Isolate	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**
S186	1	1	0	1	1	0	1	1	1	1	0	1
S187	1	1	0	1	1	0	1	1	1	1	0	0
S188	1	1	0	1	1	1	1	1	0	1	0	0
S189	0	1	0	1	1	1	1	1	0	1	0	0
S190	1	1	0	1	1	1	1	1	1	1	0	0
S191	0	0	0	0	1	1	1	0	0	1	0	0
S192	0	1	0	1	1	1	1	0	1	1	0	0
S193	1	1	0	1	1	1	0	0	0	1	0	0
S194	1	1	0	1	1	1	1	1	1	1	0	0
S195	0	1	0	1	1	0	0	1	1	1	0	0
S196	0	1	0	1	1	1	0	0	1	1	0	0
S197	1	1	0	1	1	0	1	0	1	1	0	0

Appendix 3 c: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 536.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P001	0	0	0	0	1	1	1	0	1	1	0	0	0	0	1	0	0
P003	0	0	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
P004	0	0	0	0	1	1	1	1	1	1	0	1	0	0	0	0	0
P006	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P007	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P008	0	1	0	0	1	0	1	1	1	1	0	1	0	0	0	0	0
P009	0	0	0	0	1	0	1	0	0	1	0	0	0	0	0	0	0
P010	0	1	0	0	1	0	1	0	1	1	0	1	0	0	1	0	0
P011	0	0	0	0	0	1	0	0	1	1	0	1	0	0	0	0	0
P013	0	0	0	0	0	1	1	0	1	1	0	1	0	0	1	0	0
P016	0	0	0	0	0	0	1	1	1	1	0	0	0	1	0	0	0
P017	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
P018	0	0	0	0	0	1	1	0	1	1	0	1	0	0	0	0	0
P019	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0
P022	0	0	0	0	0	1	1	0	1	1	0	0	0	0	0	0	0
P023	0	0	0	0	0	1	1	1	1	1	0	1	0	0	0	0	0
P024	0	0	0	0	0	1	1	1	1	1	0	0	0	0	0	1	1
P026	0	0	0	0	1	1	0	0	1	1	0	1	0	0	1	0	1
P027	0	0	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
P028	0	0	0	0	1	1	0	0	1	1	0	0	0	0	0	0	0
P029	0	0	0	0	1	0	0	0	1	1	0	0	0	1	1	0	1
P030	0	0	0	0	0	1	0	0	1	1	0	1	0	1	0	1	0
P031	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
P032	0	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P033	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P034	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
P036	0	0	0	0	0	1	1	0	1	1	0	1	0	0	0	1	0
P037	0	0	0	0	1	1	1	0	1	1	0	1	0	0	1	1	1
P038	0	0	0	0	0	0	1	0	1	1	0	1	0	0	0	0	0
P039	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0

Appendix 3 c: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 536.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P040	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
P041	0	0	0	0	1	1	1	0	1	1	0	1	0	1	0	1	1
P042	0	1	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
P043	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
P044	0	0	0	0	0	0	1	0	1	1	0	1	0	0	0	0	0
P045	0	0	0	0	1	0	1	0	1	1	0	1	0	0	0	0	0
P046	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	1	0
P047	0	0	0	0	1	0	1	0	1	1	0	1	0	0	1	1	0
P048	0	0	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
P049	0	0	0	0	1	1	1	0	1	1	0	0	0	1	0	0	0
P051	1	0	0	1	1	1	1	0	1	1	0	1	0	1	1	0	0
P052	1	0	0	1	1	0	1	0	1	1	0	1	0	0	1	0	1
P053	0	0	0	1	0	1	1	1	1	1	0	1	0	0	0	0	0
P054	1	0	0	1	1	1	1	0	1	1	0	1	0	1	1	0	1
P055	1	0	0	1	1	1	1	0	1	1	0	1	0	0	0	0	0
P056	1	0	0	1	1	1	1	1	1	1	0	1	0	0	1	1	1
P057	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P058	0	0	0	0	1	0	1	1	1	1	0	0	0	0	0	0	0
P059	0	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0
P060	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P061	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0
P062	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0
P065	1	1	0	1	1	1	1	0	1	1	0	1	0	0	0	0	0
P066	1	1	0	0	0	0	1	0	1	1	1	1	0	0	0	0	0
P067	0	0	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
P068	1	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0
P069	0	0	0	1	1	0	0	1	1	1	0	1	0	0	0	0	0
P070	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0
P072	0	0	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
P073	0	0	0	0	1	0	1	1	1	1	0	1	0	0	1	0	0

Appendix 3 c: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 536.

	Marker																
Isolates	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P075	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P076	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P077	0	0	0	1	1	1	1	0	1	1	0	0	0	0	1	0	0
P078	0	0	0	1	1	0	1	0	1	1	0	0	0	0	1	0	0
P079	0	0	0	0	0	0	1	0	1	1	0	1	0	0	0	0	0
P080	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
P081	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P082	0	0	0	0	1	0	1	0	1	1	0	0	0	0	0	0	1
P083	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P085	0	0	0	0	0	0	1	0	1	1	0	1	0	0	0	0	0
P087	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0
P088	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
P089	0	0	0	0	0	0	1	0	1	1	0	1	0	0	0	0	0
P090	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P091	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P092	1	1	0	1	1	1	1	0	1	1	0	1	0	0	0	1	0
P094	0	0	0	0	1	1	0	0	1	1	0	1	0	0	0	0	0
P097	0	0	0	1	1	1	1	0	1	1	0	0	0	0	0	0	0
P099	0	0	0	0	0	0	0	0	1	1	0	1	0	0	0	0	0
P100	0	0	0	0	0	0	1	0	0	1	0	1	0	0	0	0	0
P101	0	0	0	1	1	1	1	0	1	1	0	0	0	0	0	0	0
P102	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
P104	0	0	0	1	1	1	1	0	1	1	1	0	1	0	1	0	0
P105	0	0	0	1	1	1	1	0	1	1	0	1	1	0	1	0	0
P106	0	0	0	0	0	0	1	0	1	0	0	1	0	0	0	0	0
S107	0	0	0	0	1	0	1	0	1	1	0	1	0	0	0	0	0
S108	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0
S109	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
S111	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0
S112	0	0	0	0	0	1	1	1	1	1	0	0	0	0	0	0	0

Appendix 3 c: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 536.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S113	0	1	1	1	1	1	1	0	1	1	0	1	0	1	0	1	0
S114	0	0	1	0	1	1	1	0	1	1	0	0	0	0	0	1	0
S115	1	0	0	1	1	1	1	1	1	1	1	1	0	1	0	1	0
S116	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
S117	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
S118	1	1	0	0	1	1	1	1	1	1	0	1	0	0	0	1	0
S119	0	0	0	0	1	1	1	0	1	1	0	0	0	0	0	1	0
S120	0	0	0	0	1	1	1	1	1	1	0	1	0	0	0	0	0
S121	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
S122	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
S123	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
S124	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
S125	0	0	0	0	1	0	1	0	1	1	0	1	0	0	0	0	0
S126	0	0	0	0	1	1	1	0	1	1	0	1	0	1	0	0	0
S127	0	0	0	1	1	1	1	0	1	1	0	1	0	0	0	1	1
S128	0	0	0	0	0	1	1	0	1	1	0	1	0	0	0	1	0
S130	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
S132	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
S134	0	0	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
S135	0	0	0	0	1	0	0	0	1	1	0	0	0	0	0	0	0
S136	0	0	0	0	0	0	1	0	1	1	0	1	0	0	0	0	0
S137	0	0	0	0	0	1	1	0	1	1	0	1	0	0	0	0	0
S138	0	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0
S139	0	0	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
S140	0	0	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
S141	0	0	0	0	1	0	1	0	1	1	0	1	0	0	0	1	0
S142	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
S143	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
S144	0	0	0	0	0	0	0	0	1	1	0	1	0	0	1	0	0
S146	0	0	0	0	1	0	1	0	1	1	0	0	0	0	0	0	0

Appendix 3 c: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 536.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S147	0	0	0	0	1	0	1	1	0	1	0	1	0	0	0	0	0
S149	0	0	0	1	1	1	1	0	1	1	0	1	0	0	0	0	0
S150	0	0	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0
S151	0	1	0	1	1	1	1	0	1	1	0	1	0	0	1	0	0
S152	0	1	0	1	1	1	1	0	1	1	0	1	0	0	0	0	0
S154	0	1	0	1	1	1	1	0	1	1	0	1	0	0	1	0	1
S155	0	0	0	1	1	1	1	1	1	1	0	1	0	0	0	0	1
S156	0	0	0	0	1	1	1	0	1	0	0	1	0	0	1	0	1
S157	1	0	1	0	1	0	1	0	1	1	0	1	0	0	0	1	0
S159	0	0	0	1	1	0	1	0	1	1	0	0	0	0	0	0	0
S160	0	0	0	1	1	0	0	0	1	1	0	1	0	0	0	0	0
S161	1	1	1	0	0	1	1	0	1	1	0	1	0	0	0	0	0
S162	0	1	0	0	1	0	1	0	1	1	0	1	0	0	0	0	0
S164	0	1	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
S165	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0
S166	0	1	0	0	1	1	1	0	1	1	0	1	0	0	0	1	0
S169	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
S171	0	0	0	0	0	0	1	0	1	1	0	1	0	0	0	0	0
S172	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0
S173	0	0	0	0	0	1	1	0	1	0	0	0	0	0	0	0	0
S174	0	0	0	0	0	0	1	0	1	1	0	1	0	0	1	0	0
S176	0	1	0	0	1	1	1	0	1	1	0	1	0	0	0	0	0
S177	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0
S178	0	0	0	0	0	1	1	0	1	1	0	1	0	0	0	1	0
S179	0	0	0	0	0	1	1	0	1	1	0	0	0	0	0	0	0
S180	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1	0
S181	0	0	0	0	0	1	1	0	1	1	0	0	0	0	0	0	1
S182	0	0	0	0	0	1	1	0	1	1	0	0	0	0	0	0	0
S183	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
S184	0	0	0	0	0	1	1	0	1	1	0	1	0	0	0	1	0

Appendix 3 c: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 536.

	Marker																
Isolate	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**
S185	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
S186	0	0	0	0	0	1	1	0	1	1	0	1	0	0	0	0	0
S187	0	0	0	0	0	1	1	0	1	1	0	1	0	0	0	0	0
S188	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
S189	0	0	0	0	0	1	1	0	1	1	0	0	0	0	0	0	0
S190	0	0	0	0	0	1	1	0	1	1	0	1	0	0	0	0	0
S191	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
S192	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	1
S193	0	0	0	0	0	0	1	0	1	1	1	1	0	0	0	1	0
S194	0	0	0	0	0	0	1	0	1	1	1	0	0	1	0	1	1
S195	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
S196	0	0	0	0	0	0	1	0	1	1	1	0	0	0	0	1	1
S197	0	0	0	0	0	0	0	0	1	1	1	1	0	0	0	0	1

Appendix 3 c: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 536.

	Marker		Marker		Marker		Marker		Marker		Marker
Isolate	18	Isolate	18	Isolate	18	Isolate	18	Isolate	18	Isolate	18
P001	0	P038	0	P069	0	P106	0	S138	0	S174	0
P003	0	P039	0	P070	0	S107	0	S139	0	S176	0
P004	0	P040	0	P072	1	S108	0	S140	0	S177	1
P006	0	P041	0	P073	0	S109	0	S141	1	S178	0
P007	0	P042	0	P075	0	S111	0	S142	0	S179	0
P008	0	P043	0	P076	0	S112	0	S143	0	S180	0
P009	0	P044	0	P077	0	S113	0	S144	0	S181	0
P010	0	P045	0	P078	0	S114	0	S146	0	S182	0
P011	0	P046	0	P079	0	S115	0	S147	0	S183	0
P013	0	P047	0	P080	0	S116	0	S149	0	S184	0
P016	0	P048	0	P081	0	S117	0	S150	0	S185	0
P017	0	P049	0	P082	0	S118	0	S151	0	S186	0
P018	0	P051	0	P083	0	S119	0	S152	0	S187	0
P019	0	P052	0	P085	0	S120	0	S154	0	S188	0
P022	0	P053	0	P087	0	S121	0	S155	0	S189	0
P023	0	P054	0	P088	0	S122	0	S156	0	S190	0
P024	0	P055	0	P089	0	S123	0	S157	0	S191	0
P026	0	P056	0	P090	0	S124	0	S159	0	S192	0
P027	0	P057	0	P091	0	S125	0	S160	0	S193	0
P028	0	P058	0	P092	0	S126	0	S161	0	S194	0
P029	0	P059	0	P094	0	S127	0	S162	0	S195	0
P030	0	P060	0	P097	0	S128	0	S164	1	S196	0
P031	0	P061	0	P099	0	S130	0	S165	0	S197	0
P032	0	P062	0	P100	0	S132	0	S166	0		
P033	0	P065	0	P101	0	S134	0	S169	0		
P034	0	P066	0	P102	0	S135	0	S171	0		
P036	0	P067	0	P104	0	S136	1	S172	0		
P037	0	P068	0	P105	0	S137	0	S173	0		

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P001	0	1	1	1	1	1	1	1	0	1	1	0	0	0	1	0	1
P003	1	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P004	0	1	1	1	1	0	1	1	0	1	1	0	1	1	1	0	1
P006	0	1	1	1	1	0	1	1	0	1	1	0	1	0	0	0	1
P007	0	1	1	1	1	1	1	1	0	1	1	0	1	0	1	0	1
P008	0	1	1	1	1	0	1	1	0	1	1	1	1	0	1	0	1
P009	1	1	1	1	1	0	1	1	1	1	1	0	1	0	0	0	1
P010	0	1	1	1	1	0	0	1	0	1	1	1	1	0	1	0	1
P011	1	1	1	1	1	0	1	1	0	1	1	1	1	0	1	0	1
P013	1	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P016	0	1	1	1	1	0	1	1	0	1	1	0	0	0	1	0	1
P017	0	0	1	1	0	0	1	1	0	1	0	0	0	1	1	0	1
P018	0	1	1	1	1	1	1	1	0	1	1	1	1	0	1	0	1
P019	0	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P022	0	1	1	1	0	1	1	1	0	1	1	0	0	0	1	0	1
P023	0	1	1	1	1	0	1	1	0	1	1	1	1	0	1	0	1
P024	0	1	1	1	1	1	1	1	0	1	1	1	1	0	1	0	1
P026	1	0	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P027	0	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P028	0	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P029	0	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P030	0	1	0	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P031	0	1	1	1	1	0	1	1	0	1	1	1	1	0	1	0	1
P032	0	1	1	1	0	0	1	1	0	1	1	0	1	0	1	0	1
P033	1	1	1	1	0	0	0	1	0	1	0	0	0	0	0	0	1
P034	0	0	1	1	1	0	0	1	0	1	1	0	0	0	1	0	1
P036	0	1	1	1	1	0	1	1	0	1	1	0	0	0	1	0	1
P037	0	1	1	1	1	1	1	1	0	1	1	0	0	0	1	0	1
P038	1	1	0	1	1	0	1	1	0	1	1	0	0	1	0	0	1
P039	0	0	0	1	0	0	1	0	0	1	1	1	0	0	1	0	1

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P040	0	0	0	1	1	0	0	1	0	1	1	0	1	0	0	0	1
P041	0	1	1	1	1	1	1	1	0	1	1	0	1	0	1	0	1
P042	0	1	0	1	1	0	0	1	0	1	1	0	0	0	1	0	0
P043	1	1	1	1	0	0	1	1	0	1	1	1	0	1	1	0	1
P044	0	1	1	1	0	0	1	1	0	1	1	0	1	0	1	0	1
P045	1	1	1	1	1	0	1	1	0	1	1	0	0	0	1	0	1
P046	0	1	1	1	0	0	1	1	0	0	0	0	0	1	0	0	0
P047	0	1	1	1	1	0	1	1	0	1	1	0	0	0	1	0	1
P048	0	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P049	0	1	1	1	1	1	1	1	1	1	1	0	1	0	1	0	1
P051	0	1	1	1	0	0	1	1	0	1	1	1	0	0	1	0	1
P052	0	1	1	1	0	0	1	1	0	1	1	0	1	1	1	0	1
P053	1	1	1	1	1	1	1	1	1	1	1	0	0	0	1	0	1
P054	1	1	1	1	0	0	1	1	0	1	1	0	0	0	1	0	1
P055	0	1	1	1	0	0	1	1	0	1	1	0	1	0	1	0	1
P056	0	1	1	1	1	1	1	1	1	1	1	0	1	0	1	0	1
P057	0	0	0	1	1	0	0	0	0	0	0	0	1	0	0	0	1
P058	0	1	1	1	0	0	1	1	0	1	1	0	0	0	1	0	0
P059	0	0	0	1	0	0	0	1	0	1	0	0	0	1	0	0	0
P060	0	0	0	1	0	0	0	1	0	0	0	0	1	0	0	0	0
P061	0	0	0	1	0	0	1	0	0	1	0	0	0	1	1	0	1
P062	0	1	1	1	0	1	0	1	1	1	1	1	1	0	1	0	1
P065	0	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P066	0	1	1	1	1	1	1	1	0	1	1	1	0	1	1	0	1
P067	0	1	1	1	1	0	1	1	0	1	1	1	1	0	1	0	1
P068	0	1	1	1	0	0	1	0	0	1	1	1	0	0	0	1	0
P069	0	1	1	1	1	0	1	1	1	1	1	1	1	0	1	0	1
P070	0	0	0	1	0	0	0	0	0	1	1	1	0	0	0	0	1
P072	0	1	1	1	1	0	1	1	0	1	1	0	0	1	1	0	1
P073	0	1	1	1	1	1	1	1	0	1	1	1	1	0	1	0	1

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P075	0	1	0	1	0	0	1	1	0	1	1	0	1	0	1	0	1
P076	0	0	0	1	0	0	1	0	0	0	0	0	0	1	1	0	1
P077	0	1	0	1	1	1	1	1	1	1	1	0	1	1	1	0	1
P078	0	1	1	1	0	1	1	1	1	1	1	1	0	1	1	0	1
P079	0	1	1	1	0	1	1	1	0	1	1	1	0	1	1	0	1
P080	0	1	1	1	1	0	1	1	0	1	1	1	0	1	1	0	1
P081	0	0	0	1	0	0	1	0	0	0	0	0	0	1	1	0	1
P082	0	1	0	1	1	0	1	1	0	1	1	0	1	0	1	0	1
P083	0	1	0	1	0	0	1	1	0	1	1	0	0	0	1	0	1
P085	0	1	1	1	1	1	1	1	0	1	0	0	1	0	1	0	1
P087	0	0	0	1	1	1	1	1	0	1	1	0	0	1	1	0	1
P088	0	1	1	1	1	1	1	1	1	1	1	0	1	1	0	0	1
P089	0	0	0	1	0	1	1	1	0	1	1	0	0	0	1	0	1
P090	0	0	0	1	0	0	0	0	0	0	1	0	0	1	0	0	0
P091	0	0	1	1	1	0	0	1	1	1	1	0	1	0	1	0	1
P092	0	1	1	1	1	0	1	1	1	1	0	0	1	0	1	0	1
P094	0	1	1	1	1	1	1	1	0	1	1	1	1	0	1	0	1
P097	1	1	1	1	0	0	1	1	0	1	1	0	0	0	1	0	1
P099	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0
P100	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	1
P101	0	1	1	1	1	0	1	1	1	1	1	0	0	0	1	0	1
P102	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
P104	0	0	1	1	0	0	1	1	0	1	0	0	0	1	0	0	1
P105	0	1	1	1	1	1	1	1	1	1	1	0	0	1	1	0	1
P106	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
S107	0	1	0	1	0	1	1	1	0	1	0	0	1	0	0	0	1
S108	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
S109	0	1	1	1	0	0	0	0	1	0	0	0	1	0	0	0	1
S111	0	0	1	1	0	0	0	0	0	1	0	0	1	0	0	0	1
S112	1	1	1	1	1	1	1	1	0	1	1	1	0	0	1	0	1

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S113	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0
S114	0	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
S115	1	1	1	1	1	1	1	1	1	1	1	0	1	0	0	0	1
S116	0	0	0	1	0	0	0	1	0	0	0	0	1	0	0	0	1
S117	1	1	1	1	1	0	1	1	0	1	1	0	1	0	0	0	1
S118	1	1	1	1	1	0	1	1	0	0	1	0	0	1	0	0	1
S119	0	0	0	1	0	0	1	1	0	0	1	0	0	0	0	0	1
S120	0	1	1	1	1	1	1	1	0	1	1	0	1	0	0	0	1
S121	0	0	0	1	0	0	1	1	1	1	1	1	0	0	0	0	1
S122	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
S123	0	0	0	1	0	0	0	0	0	1	0	0	0	0	1	0	0
S124	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
S125	0	0	0	1	0	0	1	1	0	1	1	0	1	0	1	0	1
S126	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1	0	1
S127	0	1	1	1	1	1	1	1	1	1	1	0	0	0	1	0	1
S128	0	1	1	1	0	0	1	1	0	1	1	0	1	0	1	0	1
S130	0	0	0	1	0	0	0	0	0	0	0	0	0	1	0	0	1
S132	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	1
S134	0	0	0	0	0	0	1	1	1	1	1	0	0	1	0	0	1
S135	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0	1
S136	0	1	0	1	0	0	0	1	0	1	0	0	0	0	1	0	1
S137	0	1	0	1	0	0	0	1	0	1	0	0	0	1	1	0	1
S138	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1
S139	0	1	1	1	1	1	1	1	0	0	1	0	0	0	1	0	1
S140	0	1	1	1	1	1	1	1	0	1	0	0	0	0	1	0	1
S141	1	1	1	1	1	1	1	1	0	1	1	0	0	0	1	0	1
S142	0	1	1	1	1	0	1	1	0	1	1	0	0	0	1	0	1
S143	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1
S144	0	0	0	1	0	0	1	1	0	0	0	0	0	0	0	0	1
S146	1	0	0	1	1	0	0	1	0	0	0	0	0	1	1	0	1

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

	Marker																
Isolate	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S147	1	0	0	1	1	0	1	0	0	0	0	0	0	0	0	0	1
S149	0	0	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0
S150	0	1	1	1	1	0	1	1	1	1	1	0	1	0	1	0	1
S151	0	1	1	1	1	0	1	1	0	1	1	0	0	1	1	0	1
S152	0	1	1	1	1	0	0	0	0	0	0	0	0	1	0	0	1
S154	0	1	1	1	1	0	1	1	0	1	1	0	0	1	1	0	1
S155	1	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
S156	0	1	1	1	1	0	1	1	0	1	1	0	0	1	1	0	1
S157	1	1	1	1	0	0	1	1	0	1	1	0	1	0	1	0	1
S159	0	1	1	1	1	0	1	1	1	1	1	0	0	0	1	0	1
S160	0	1	0	1	1	0	1	1	0	1	1	0	0	1	0	0	1
S161	0	1	1	1	1	0	1	1	0	1	1	0	0	0	1	0	1
S162	1	1	1	1	1	0	1	1	1	1	1	0	1	1	0	0	1
S164	0	1	1	1	1	0	1	1	0	1	0	0	0	1	1	0	1
S165	0	1	1	1	1	0	1	1	0	1	1	0	1	0	1	0	1
S166	0	1	1	1	1	0	1	1	1	1	1	0	1	0	1	0	1
S169	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
S171	0	1	1	1	1	0	1	1	0	1	0	0	0	1	1	0	1
S172	0	1	1	1	1	0	0	1	0	1	0	0	0	0	1	0	1
S173	0	0	0	1	0	0	0	0	0	1	0	0	0	0	1	0	1
S174	0	1	1	1	1	0	1	1	0	1	0	0	0	0	1	0	1
S176	0	1	1	1	1	0	1	1	0	1	0	0	0	0	1	0	1
S177	0	1	1	1	0	0	1	1	0	1	0	0	0	1	0	0	1
S178	1	1	1	1	1	0	1	1	1	1	1	0	1	0	1	0	1
S179	0	1	1	1	1	0	0	1	0	1	1	0	0	0	1	0	1
S180	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1	0	0
S181	0	1	1	1	0	0	1	1	0	1	0	0	0	0	1	0	1
S182	0	0	0	1	0	0	0	0	0	1	0	0	1	0	1	0	1
S183	0	0	0	1	0	0	0	0	0	1	0	0	0	1	0	0	0
S184	1	1	1	1	1	0	1	1	0	1	0	0	0	0	1	0	1

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

	Marker																
Isolate	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**
S185	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1
S186	0	1	1	1	1	0	1	1	0	1	0	0	0	0	1	0	0
S187	0	1	1	1	0	0	0	1	0	1	0	0	0	1	0	1	0
S188	0	1	1	1	1	0	1	0	0	0	0	0	0	0	1	0	0
S189	0	1	1	1	0	0	1	1	0	1	0	0	0	0	0	1	1
S190	0	1	1	1	0	0	0	1	0	1	0	0	0	0	0	0	0
S191	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
S192	0	1	1	1	1	0	1	1	0	1	0	0	0	0	1	0	1
S193	1	1	0	1	0	1	1	1	0	0	0	0	0	0	1	0	0
S194	1	1	1	1	1	1	1	1	1	1	1	0	1	0	0	0	1
S195	0	0	1	1	0	0	0	0	0	0	1	0	0	0	0	0	0
S196	1	1	1	1	1	1	1	1	0	1	1	0	1	0	0	0	1
S197	1	1	0	1	1	1	1	1	0	1	1	1	0	0	1	0	1

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

Isolate	Marker 18	19	20	21	22	23	24	25	26
P001	0	0	1	0	1	1	0	0	0
P003	1	0	0	1	1	1	0	0	0
P004	1	1	0	1	1	1	0	1	0
P006	0	0	0	1	1	0	0	0	0
P007	0	0	0	1	1	1	0	1	0
P008	1	0	0	0	1	1	0	0	0
P009	1	0	0	1	0	1	0	0	0
P010	0	0	1	1	1	1	0	1	0
P011	1	0	0	0	1	1	0	1	0
P013	1	0	0	1	1	1	0	1	0
P016	1	0	0	0	1	1	0	1	0
P017	1	0	1	0	1	1	0	1	1
P018	0	0	1	0	1	1	0	1	0
P019	1	0	1	0	1	1	0	0	0
P022	1	0	1	1	1	1	0	1	0
P023	1	0	0	0	1	1	0	1	0
P024	1	0	0	0	1	1	0	1	0
P026	1	0	0	0	0	1	0	1	0
P027	1	0	0	0	1	1	0	1	0
P028	1	0	1	0	1	1	0	1	0
P029	1	0	1	0	1	1	0	1	0
P030	1	0	0	1	1	1	0	1	0
P031	1	0	0	0	1	1	0	1	0
P032	1	0	0	0	1	1	0	1	0
P033	1	0	0	1	1	1	0	1	0
P034	0	0	0	0	1	1	0	1	0
P036	1	0	0	0	1	1	0	1	0
P037	1	0	0	0	1	1	0	1	0
P038	1	0	0	0	1	1	0	1	0
P039	1	0	0	0	1	1	0	1	0
P040	1	0	0	0	1	1	1	1	1

Appendix 3D: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

Isolate	Marker 18	19	20	21	22	23	24	25	26
P041	0	0	0	0	1	1	0	1	0
P042	0	0	1	0	1	1	0	0	0
P043	0	0	1	0	1	1	0	1	0
P044	1	0	0	1	1	1	0	1	0
P045	1	0	0	1	1	1	0	1	0
P046	1	0	0	1	1	1	0	1	0
P047	0	0	0	1	1	1	0	0	0
P048	1	0	0	0	1	1	0	0	0
P049	1	0	0	1	1	1	0	1	0
P051	1	0	1	0	1	1	0	1	0
P052	1	0	1	1	1	1	1	1	0
P053	1	0	0	1	1	1	0	1	0
P054	1	0	0	1	1	1	0	1	0
P055	1	0	0	1	1	1	0	1	0
P056	1	0	0	0	1	1	0	0	0
P057	0	0	0	0	0	1	0	0	1
P058	1	0	0	0	1	1	0	0	0
P059	1	0	0	0	0	1	0	0	1
P060	1	0	0	0	0	1	0	0	1
P061	0	0	0	0	0	1	0	1	1
P062	0	0	0	0	1	1	0	0	0
P065	1	0	0	1	1	1	0	1	1
P066	1	0	0	1	1	1	0	1	1
P067	1	1	0	0	1	1	0	1	1
P068	1	0	1	0	0	0	1	1	0
P069	1	0	1	0	1	1	0	1	0
P070	0	0	1	0	1	1	0	0	1
P072	1	0	1	0	1	1	0	0	0
P073	1	0	0	1	1	1	0	1	0
P075	1	0	0	1	1	1	0	0	0
P076	0	0	0	0	1	1	0	0	1

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

Isolate	Marker 18	19	20	21	22	23	24	25	26
P077	1	1	0	1	1	1	0	0	0
P078	1	0	0	1	1	1	0	1	1
P079	1	0	1	1	1	1	0	1	1
P080	1	0	0	1	1	1	0	0	0
P081	1	0	0	0	1	1	0	0	0
P082	1	0	0	0	1	1	0	0	0
P083	1	0	0	1	1	1	0	1	0
P085	1	0	0	1	1	1	0	0	0
P087	0	0	0	1	1	1	0	1	0
P088	1	0	0	0	1	1	0	0	0
P089	1	0	1	0	1	1	0	0	0
P090	0	0	0	1	0	1	0	0	0
P091	0	0	0	1	1	1	0	0	0
P092	1	0	0	0	1	1	0	1	0
P094	1	0	1	0	1	1	0	1	0
P097	1	0	0	1	1	1	0	0	1
P099	0	0	0	0	0	0	0	0	0
P100	0	0	0	0	0	0	0	0	0
P101	1	0	0	1	1	1	0	0	1
P102	0	0	0	0	0	0	0	0	0
P104	0	0	0	0	1	1	0	0	0
P105	1	0	0	0	1	1	0	1	1
P106	0	0	0	0	1	0	0	0	0
S107	0	1	1	0	1	1	0	1	1
S108	0	0	0	0	0	0	0	1	1
S109	0	0	0	0	0	0	0	0	1
S111	0	0	0	0	0	1	0	0	0
S112	1	0	0	0	1	1	0	0	0
S113	0	0	0	0	0	0	0	0	0
S114	0	0	0	0	1	1	0	0	0
S115	1	0	0	0	1	1	0	0	0

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

Isolate	Marker 18	19	20	21	22	23	24	25	26
S116	1	0	0	0	0	0	0	0	0
S117	0	0	0	0	1	1	0	0	0
S118	0	0	1	0	0	1	0	0	0
S119	1	0	0	0	0	1	0	0	0
S120	1	0	0	0	0	1	0	0	0
S121	1	0	0	0	1	1	0	0	0
S122	0	0	0	0	0	0	0	0	0
S123	0	0	0	0	0	0	0	0	0
S124	0	0	0	0	0	0	0	0	0
S125	0	0	0	0	1	1	0	1	0
S126	0	0	0	0	1	1	0	1	0
S127	1	1	0	0	1	1	0	0	0
S128	1	0	0	0	1	1	0	0	0
S130	1	0	0	0	1	0	0	1	0
S132	0	0	0	0	1	1	0	0	0
S134	1	1	0	0	1	1	0	1	1
S135	0	0	0	0	1	0	0	0	0
S136	1	0	0	0	1	1	0	0	0
S137	0	0	0	0	1	1	0	0	0
S138	0	0	0	0	0	1	0	0	0
S139	0	0	0	0	0	1	0	0	1
S140	1	0	0	0	1	1	0	0	0
S141	1	0	0	0	1	1	0	0	1
S142	1	0	0	0	1	1	0	0	1
S143	0	0	0	0	0	1	0	0	0
S144	1	0	0	0	1	1	0	0	0
S146	0	0	0	0	0	0	0	1	0
S147	0	0	0	0	0	0	0	0	0
S149	0	0	0	0	1	1	0	0	0
S150	1	0	0	0	1	1	0	0	0
S151	1	0	0	0	1	1	0	0	0

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

Isolate	Marker 18	19	20	21	22	23	24	25	26
S152	1	0	0	0	1	1	0	0	0
S154	1	0	0	0	1	1	0	0	0
S155	1	0	0	0	1	1	0	0	0
S156	1	1	1	0	1	1	0	0	0
S157	1	0	0	0	1	1	0	0	0
S159	1	0	0	0	1	1	0	0	0
S160	1	0	1	0	1	1	0	0	0
S161	1	0	0	0	1	1	0	0	0
S162	0	0	0	0	1	1	0	0	0
S164	1	0	0	0	1	1	0	0	0
S165	1	0	0	0	1	1	0	0	0
S166	0	0	0	0	1	1	0	0	0
S169	0	0	0	0	0	0	0	0	0
S171	0	0	0	0	1	1	0	0	0
S172	0	1	0	0	1	1	0	0	0
S173	0	0	0	0	1	1	0	0	0
S174	0	0	1	0	1	1	0	0	1
S176	1	0	0	0	1	1	0	0	0
S177	0	0	1	0	0	1	0	0	0
S178	0	0	0	0	1	1	0	0	0
S179	0	1	0	0	1	1	0	0	0
S180	0	0	0	0	0	0	0	0	0
S181	0	0	1	0	1	1	0	0	0
S182	1	0	0	0	0	1	0	0	0
S183	0	0	0	0	0	1	0	0	0
S184	1	0	0	0	1	1	0	0	0
S185	0	0	0	0	0	0	0	0	0
S186	1	0	0	0	1	1	0	0	0
S187	1	0	0	0	1	1	0	0	0
S188	1	0	0	0	1	1	0	0	0
S189	0	0	0	0	1	1	0	0	0

Appendix 3 d: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 676.

Isolate	Marker 18	19	22	21	22	23	24	25	26
S190	1	0	0	0	0	1	0	0	0
S191	0	0	0	0	0	0	0	0	0
S192	0	0	0	0	1	1	0	0	0
S193	0	0	0	0	0	1	0	0	0
S194	1	0	0	0	1	1	0	1	1
S195	0	0	0	0	0	1	0	1	1
S196	0	0	0	0	1	1	0	0	0
S197	0	0	0	0	1	1	0	0	0

Appendix 3 e: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 634.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P001	1	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
P003	0	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
P004	1	1	1	1	0	0	1	1	0	0	1	1	1	1	1	1	1
P006	0	1	1	1	1	0	1	1	0	0	1	0	0	1	1	1	1
P007	1	1	1	1	0	0	0	1	0	0	1	0	0	1	1	1	1
P008	1	1	1	1	0	1	0	1	0	1	1	0	1	1	1	1	1
P009	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
P010	1	1	1	1	0	0	1	1	0	0	1	0	0	1	1	1	1
P011	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
P013	1	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
P016	0	1	1	1	0	1	0	1	0	1	1	0	1	1	1	1	1
P017	1	1	1	1	1	0	0	1	0	1	1	1	1	1	1	1	1
P018	0	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1
P019	1	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
P022	1	1	1	1	1	0	0	1	0	1	1	1	1	1	1	1	1
P023	1	1	1	1	0	1	1	1	0	1	1	1	1	1	1	1	1
P024	1	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
P026	0	1	1	1	0	0	0	1	0	1	1	1	1	1	1	1	1
P027	0	1	1	1	0	0	1	1	0	0	1	1	1	1	1	1	1
P028	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
P029	0	1	1	1	0	1	0	1	0	1	1	1	1	1	1	1	1
P030	1	1	1	1	0	1	0	1	0	0	1	0	0	1	1	1	1
P031	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
P032	1	1	1	1	0	1	0	1	0	1	1	0	1	1	1	1	1
P033	0	1	1	1	0	0	1	1	0	1	0	0	0	1	1	1	1
P034	0	1	1	1	0	0	1	1	0	1	1	0	0	1	1	1	1
P036	0	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
P037	1	1	1	1	0	0	1	1	0	1	1	1	0	1	1	1	1
P038	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
P039	0	1	1	1	0	0	0	1	0	0	1	0	0	1	1	1	1
P040	1	1	1	1	0	0	1	1	0	0	1	0	0	1	1	1	1

Appendix 3 e: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 634.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P041	1	1	1	1	1	0	0	1	0	1	1	1	1	1	1	1	1
P042	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
P043	1	1	1	1	0	0	1	1	0	1	1	0	0	1	1	1	1
P044	1	1	1	1	0	0	0	1	0	1	1	1	1	1	1	1	1
P045	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
P046	1	1	1	1	0	0	1	1	0	0	1	0	1	1	1	1	1
P047	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
P048	1	0	1	1	1	0	0	1	0	1	1	0	1	1	1	1	1
P049	0	1	1	1	1	0	1	1	0	0	1	1	1	1	1	1	1
P051	1	1	1	1	1	0	0	1	0	1	1	1	1	1	1	1	1
P052	0	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
P053	1	1	1	1	1	0	0	1	0	0	1	1	1	1	1	1	1
P054	0	1	1	1	1	0	1	1	0	1	1	1	1	1	1	1	1
P055	0	1	1	1	1	0	1	1	0	0	1	1	1	1	1	1	1
P056	0	1	1	1	1	0	1	1	0	1	1	1	1	1	1	1	1
P057	0	1	1	1	0	1	0	1	1	1	1	0	0	1	1	1	1
P058	0	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
P059	0	0	1	1	0	1	0	1	0	1	1	0	0	1	1	1	1
P060	0	0	1	1	0	0	0	1	0	1	0	0	0	1	1	1	1
P061	0	0	1	1	1	0	0	1	0	0	1	0	1	1	1	1	1
P062	0	1	1	1	1	0	0	1	0	1	1	0	0	1	1	1	1
P065	0	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1
P066	0	1	1	1	0	1	0	1	0	1	1	0	1	1	1	1	1
P067	0	1	1	1	0	0	1	1	1	0	1	1	1	1	1	1	1
P068	1	0	1	0	0	0	1	0	0	0	0	0	1	0	1	1	1
P069	0	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
P070	0	1	1	1	0	0	1	1	0	0	1	1	0	1	1	1	1
P072	1	1	1	1	1	0	1	1	0	1	1	1	1	1	1	1	1
P073	1	1	1	1	0	0	1	1	0	0	1	1	1	1	1	1	1
P075	0	1	1	1	1	0	1	1	0	1	1	0	0	1	1	1	1

Appendix 3 e: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 634.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P076	0	1	1	1	1	0	1	1	0	0	0	1	0	1	1	1	1
P077	0	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
P078	0	1	1	1	0	0	1	1	0	1	0	1	1	1	1	1	1
P079	0	1	1	1	1	0	1	1	0	1	1	1	1	1	1	1	1
P080	0	1	1	1	1	0	1	1	0	1	1	0	0	1	1	1	1
P081	0	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
P082	0	1	1	1	1	0	1	1	0	0	0	0	0	1	1	1	1
P083	0	1	1	1	1	0	1	1	0	1	1	0	0	1	1	1	1
P085	0	1	1	1	1	0	1	1	0	1	1	0	0	1	1	1	1
P087	0	0	1	1	1	0	1	1	1	0	1	0	0	1	1	1	1
P088	0	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1
P089	1	0	1	1	0	0	1	1	1	0	0	1	0	1	1	1	1
P090	0	0	0	0	0	0	1	1	0	1	0	1	0	1	1	0	0
P091	0	1	1	1	1	0	1	1	0	1	1	0	0	1	1	1	1
P092	1	1	1	1	1	0	1	1	0	1	1	1	1	1	1	1	1
P094	1	1	1	1	1	0	1	1	0	1	1	0	0	1	1	1	1
P097	1	1	1	1	0	0	1	1	0	1	1	1	0	1	1	1	1
P099	0	0	1	0	1	0	1	1	0	1	1	0	0	1	1	1	0
P100	0	0	1	1	0	0	1	1	0	1	1	0	0	1	1	1	0
P101	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
P102	1	0	1	1	0	1	1	1	0	1	1	0	0	1	1	0	0
P104	1	1	1	1	0	1	1	1	0	1	1	0	0	1	1	1	1
P105	0	1	1	1	1	0	1	1	0	1	1	1	1	1	1	1	1
P106	0	0	1	1	1	0	1	1	0	1	1	0	0	0	0	0	0
S107	1	1	1	1	0	0	1	1	0	1	1	1	0	0	0	1	0
S108	0	0	1	1	0	0	0	1	0	0	0	0	0	0	0	0	0
S109	1	1	1	1	0	0	1	1	0	1	1	1	0	0	0	1	0
S111	1	1	1	1	1	0	1	1	0	0	1	0	0	1	1	1	1
S112	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S113	0	0	0	1	0	0	0	0	0	1	1	0	0	0	1	0	0

Appendix 3 e: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 634.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S114	1	1	1	1	0	0	0	1	0	1	1	1	1	1	1	1	1
S115	1	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
S116	0	1	1	1	1	0	1	1	0	1	1	0	0	1	1	1	1
S117	1	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
S118	1	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
S119	1	1	1	1	0	1	1	1	0	1	1	0	0	1	1	1	1
S120	1	1	1	1	1	0	1	1	0	1	1	0	0	0	1	1	1
S121	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
S122	0	0	1	1	0	0	0	1	1	1	1	0	0	0	1	1	0
S123	0	0	1	1	0	0	1	1	0	1	1	0	0	1	1	1	0
S124	0	0	0	1	0	0	1	0	0	1	0	0	0	1	1	1	1
S125	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S126	0	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S127	0	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
S128	0	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
S130	0	0	1	1	0	0	1	1	0	0	1	0	1	1	1	1	1
S132	0	0	1	1	1	0	1	1	0	1	1	0	0	1	1	1	1
S134	0	0	1	1	0	0	1	1	0	1	1	0	0	0	1	1	0
S135	0	0	1	0	0	0	1	1	0	1	1	0	1	1	1	1	1
S136	0	0	1	1	1	0	1	1	0	1	1	0	1	1	1	0	0
S137	1	0	1	1	1	0	0	1	0	0	1	0	1	1	1	1	1
S138	0	0	1	0	0	0	1	1	0	1	1	0	1	1	1	1	1
S139	1	1	1	1	0	0	1	1	0	1	1	0	0	1	1	1	1
S140	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
S141	0	1	1	1	0	0	0	1	0	1	1	1	1	1	1	1	1
S142	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
S143	0	1	1	1	1	0	1	1	0	0	1	0	1	1	1	1	1
S144	0	1	1	1	0	0	1	1	0	0	1	0	0	1	1	1	1
S146	1	1	1	1	1	0	1	1	0	1	0	1	1	1	1	1	1
S147	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	0	1
S149	1	1	1	1	0	0	1	1	0	1	1	1	0	1	1	1	1

Appendix 3 e: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 634.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S150	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S151	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
S152	1	1	1	1	0	0	1	1	0	0	1	1	0	1	1	1	1
S154	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S155	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S156	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S157	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
S159	1	1	1	1	1	0	1	1	0	1	1	1	1	1	1	1	1
S160	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S161	1	1	1	1	0	1	0	1	0	1	1	1	0	1	1	1	1
S162	1	1	1	1	0	1	1	1	0	1	1	1	1	1	1	1	1
S164	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S165	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S166	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
S169	1	0	1	0	0	0	0	1	0	1	0	0	1	1	1	1	1
S171	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	0	0
S172	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
S173	0	1	1	1	0	0	0	1	1	1	1	0	1	1	1	1	1
S174	1	1	1	1	1	0	1	1	0	1	1	1	0	1	1	1	1
S176	1	1	1	1	1	0	0	1	0	1	1	0	1	1	1	1	1
S177	1	1	1	1	0	0	1	1	0	0	1	1	0	1	1	1	1
S178	1	1	1	1	1	0	1	1	0	1	1	1	1	1	1	1	0
S179	1	1	1	1	1	0	1	1	0	1	1	1	1	1	1	1	1
S180	0	0	1	0	0	1	0	1	0	0	1	1	1	1	1	1	1
S181	0	1	1	1	1	0	1	1	0	1	1	0	1	1	1	1	1
S182	1	1	1	1	0	0	1	1	0	0	1	1	1	1	1	1	1
S183	0	0	1	0	1	0	1	1	0	1	1	0	0	1	1	1	1
S184	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S185	0	0	1	0	0	1	1	1	0	0	0	0	1	1	1	0	1
S186	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S187	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1

Appendix 3 e: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 634.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S188	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
S189	0	0	0	0	0	1	0	0	1	1	1	0	0	0	1	0	0
S190	0	1	1	1	0	0	0	1	0	0	1	0	0	1	1	1	1
S191	0	1	1	1	0	0	0	1	0	0	1	0	0	1	1	0	1
S192	1	1	1	1	0	1	1	1	0	1	1	0	0	1	1	1	1
S193	0	0	1	1	0	0	1	1	0	0	1	0	1	1	1	1	1
S194	1	1	1	1	0	0	1	1	0	1	1	0	1	1	1	1	1
S195	0	0	1	1	0	0	0	1	0	1	1	0	1	1	1	1	1
S196	1	1	1	1	0	0	1	1	0	1	1	1	1	1	1	1	1
S197	1	1	1	1	0	1	1	1	1	0	1	1	1	1	1	1	1

Appendix 3 e: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 634.

Isolate	Marker 18	19	20	21	22
P001	0	1	1	0	0
P003	0	1	1	1	0
P004	0	1	1	1	0
P006	0	1	1	1	0
P007	0	1	1	0	0
P008	0	0	1	1	0
P009	0	0	1	1	0
P010	1	0	1	1	1
P011	0	0	1	1	1
P013	0	1	1	1	0
P016	0	1	1	1	0
P017	0	1	1	1	0
P018	0	1	1	0	0
P019	0	1	1	1	0
P022	1	1	1	1	1
P023	0	1	1	1	1
P024	0	1	1	1	1
P026	0	1	1	1	0
P027	0	1	1	1	0
P028	0	1	1	1	1
P029	0	1	1	1	1
P030	0	1	1	1	0
P031	0	1	1	1	0
P032	0	1	1	1	1
P033	0	1	1	1	0
P034	0	1	1	0	0
P036	0	1	1	1	1
P037	0	1	1	1	1
P038	0	1	1	0	0
P039	0	1	0	0	0
P040	1	1	1	1	0

Isolate	Marker 18	19	20	21	22
P041	0	1	1	1	0
P042	0	1	1	1	0
P043	0	1	1	0	0
P044	0	1	1	1	0
P045	0	1	1	0	0
P046	0	1	1	1	1
P047	0	1	1	1	1
P048	0	1	1	0	0
P049	0	1	1	1	0
P051	0	1	1	1	0
P052	1	1	1	1	0
P053	0	1	1	1	0
P054	0	1	1	0	1
P055	1	1	1	1	0
P056	0	1	1	0	0
P057	0	1	1	0	0
P058	0	1	1	1	0
P059	0	1	1	1	0
P060	0	1	1	1	0
P061	0	1	1	1	1
P062	0	1	1	1	0
P065	0	1	1	1	1
P066	0	1	1	1	0
P067	0	1	1	0	1
P068	0	1	0	0	0
P069	0	1	1	1	0
P070	1	1	1	1	0
P072	0	1	1	1	1
P073	0	1	1	1	0
P075	0	1	1	1	0
P076	0	1	1	1	0

Appendix 3 e: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 634.

Isolate	Marker 18	19	20	21	22
P077	0	1	1	1	0
P078	0	1	1	0	0
P079	0	1	1	1	0
P080	0	1	1	1	0
P081	0	1	1	1	0
P082	0	1	1	0	1
P083	0	1	1	1	0
P085	0	1	1	0	1
P087	0	1	1	1	0
P088	0	1	1	1	0
P089	0	1	1	1	0
P090	0	1	0	0	0
P091	0	1	1	1	0
P092	0	1	1	1	0
P094	0	1	1	0	0
P097	0	1	1	0	0
P099	0	0	0	0	1
P100	0	1	1	1	0
P101	0	1	1	1	0
P102	0	0	0	0	1
P104	0	1	1	1	1
P105	0	1	1	0	1
P106	0	0	1	0	0
S107	1	1	0	1	1
S108	0	0	0	0	0
S109	0	0	0	0	0
S111	0	1	1	1	0
S112	0	0	1	0	1
S113	0	1	0	0	1
S114	0	1	1	0	1

Isolate	Marker 18	19	20	21	22
S115	0	1	1	0	0
S116	1	1	1	1	0
S117	0	1	1	0	0
S119	0	1	1	1	0
S120	0	1	1	1	0
S121	0	1	1	1	0
S122	0	1	1	0	0
S123	0	1	1	0	0
S124	0	0	0	1	0
S125	0	1	1	1	1
S126	0	1	1	1	0
S127	0	1	1	0	0
S128	0	1	1	0	0
S130	0	1	1	1	0
S132	0	1	1	1	0
S133	0	0	1	0	0
S134	0	1	1	1	0
S135	0	1	0	0	0
S136	0	1	1	1	1
S137	0	1	1	0	0
S138	0	1	1	1	0
S139	0	1	1	0	0
S140	0	1	1	0	1
S141	0	1	1	0	0
S142	0	1	1	1	0
S143	1	1	1	1	0
S144	0	1	1	1	0
S146	0	1	1	0	1
S147	0	1	1	1	0
S149	0	1	1	0	1

Appendix 3 e: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 634.

Isolate	Marker 18	19	20	21	22
S150	0	1	1	1	0
S151	0	1	1	1	0
S152	0	1	1	0	0
S154	0	1	1	0	0
S155	0	1	1	0	1
S156	0	1	1	1	0
S157	0	1	1	1	0
S158	0	1	1	1	0
S159	0	1	1	1	0
S160	0	1	1	1	0
S161	0	1	1	0	0
S162	1	1	1	0	0
S164	0	1	1	1	0
S165	0	1	1	1	0
S166	0	1	1	0	1
S169	0	0	0	0	1
S171	0	1	1	0	1
S172	0	1	1	0	1
S173	0	1	1	1	1
S174	0	1	1	1	0
S176	0	1	1	0	0
S177	0	1	1	1	0
S178	0	1	1	0	0
S179	0	1	1	0	0
S180	0	1	1	1	0
S181	0	1	1	0	1
S182	0	1	1	1	1
S183	0	1	1	1	1
S184	0	1	1	0	1
S185	0	1	0	1	0
S186	0	1	1	1	0

Isolate	Marker 18	19	20	21	22
S187	0	1	1	0	1
S188	0	1	1	0	0
S189	0	1	0	0	1
S190	0	1	1	1	0
S191	0	1	1	0	1
S192	0	1	1	0	1
S193	0	1	1	1	0
S194	0	1	1	1	0
S195	0	1	1	0	1
S196	0	1	1	0	0
S197	0	1	1	0	1

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P001	1	1	0	1	1	0	1	1	0	0	0	1	0	1	0	1	0
P003	1	1	0	1	1	0	1	1	0	0	0	1	0	0	0	1	0
P004	1	1	1	0	1	0	1	1	0	0	1	0	0	1	0	1	0
P006	0	0	1	1	1	0	1	1	0	0	1	0	0	0	0	1	0
P007	1	1	0	0	1	0	1	1	0	0	1	0	0	0	0	1	0
P008	1	1	1	0	1	0	1	1	0	0	0	1	0	1	0	0	0
P009	1	1	0	1	1	0	1	1	0	0	1	0	0	0	0	1	0
P010	0	1	0	1	1	0	1	1	0	0	1	1	0	0	0	0	0
P011	0	1	0	1	1	0	1	1	0	0	1	1	0	1	0	1	0
P013	1	1	0	1	1	0	1	1	0	0	1	1	0	1	0	1	0
P016	0	1	0	0	1	0	1	1	0	0	1	1	0	1	0	1	1
P017	1	0	1	1	1	0	1	1	0	0	0	1	0	1	0	1	0
P018	1	1	1	0	1	0	1	1	0	0	1	1	0	1	0	1	1
P019	1	1	0	1	1	0	1	1	0	0	1	0	0	1	0	1	0
P022	1	1	1	0	1	0	1	1	0	0	0	1	0	1	0	0	0
P023	1	1	0	0	1	0	1	1	0	0	0	0	0	1	0	1	0
P024	1	1	1	0	1	0	1	1	0	0	1	1	0	1	0	1	1
P026	1	1	1	0	1	0	1	1	0	0	1	1	0	1	0	1	0
P027	1	1	1	0	1	0	1	1	0	0	1	0	0	1	0	1	0
P028	1	1	1	0	1	0	1	1	0	0	1	0	0	1	0	1	0
P029	1	1	0	1	1	0	1	1	0	0	1	1	0	1	0	1	0
P030	1	1	1	1	1	0	1	1	0	0	1	0	0	1	0	1	0
P031	1	0	0	0	1	0	1	1	0	0	1	0	0	1	0	1	0
P032	0	0	0	0	1	0	1	1	0	0	1	0	0	1	0	1	0
P033	0	1	0	0	1	0	1	1	0	0	1	0	0	1	0	1	0
P034	1	0	0	1	1	0	1	1	0	0	1	1	0	0	0	1	0
P036	1	1	1	1	1	0	1	1	0	0	1	0	0	1	0	1	0
P037	1	1	0	1	1	0	1	1	0	0	1	1	0	1	0	1	0
P038	1	1	1	0	1	0	1	1	0	0	0	0	0	0	0	1	0
P039	0	1	0	0	1	0	1	1	0	0	0	0	0	0	0	1	0
P040	1	0	0	0	1	0	1	1	0	0	1	0	0	1	0	1	0

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

Isolate \ Marker	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P041	0	1	1	1	1	0	1	1	0	0	1	1	0	1	0	1	0
P042	1	1	1	1	1	0	1	1	0	0	0	0	0	1	0	1	0
P043	0	0	1	0	1	0	1	1	0	0	0	1	0	0	0	1	0
P044	1	1	0	0	1	0	1	1	0	0	1	0	0	1	0	1	0
P045	1	0	0	0	1	0	1	1	0	0	1	0	0	1	0	1	0
P046	0	1	0	0	1	0	0	1	0	0	1	0	0	0	0	0	0
P047	1	1	0	1	1	0	1	1	0	0	0	1	0	0	0	1	0
P048	1	1	0	0	1	0	1	1	0	0	0	1	0	1	0	0	0
P049	0	1	1	0	1	0	1	1	0	0	1	1	0	0	0	1	0
P051	1	1	0	1	1	0	1	1	0	0	0	0	1	1	0	1	0
P052	1	1	1	1	1	0	1	1	0	0	1	0	0	1	0	1	0
P053	1	0	1	1	1	0	1	1	0	0	1	1	0	1	0	1	0
P054	0	1	1	0	1	0	1	1	0	0	1	1	0	1	0	1	0
P055	0	1	0	0	1	0	1	1	0	0	0	0	0	1	0	1	0
P056	0	1	1	1	1	0	1	0	0	0	1	1	0	1	0	1	0
P057	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
P058	1	1	1	0	1	0	1	1	0	0	1	0	0	0	0	0	0
P059	0	0	0	0	1	0	1	0	0	0	1	0	0	0	0	0	0
P060	1	0	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0
P061	0	1	1	0	1	0	1	1	0	0	1	1	0	1	0	0	0
P062	1	1	1	1	1	0	1	1	0	0	1	1	0	1	0	1	0
P065	1	1	1	0	1	0	1	1	0	0	1	1	1	0	0	1	0
P066	1	1	1	1	1	0	1	1	0	0	1	1	0	1	0	1	0
P067	1	1	1	1	0	1	1	1	0	0	1	1	1	1	0	1	0
P068	0	0	1	0	1	0	0	1	0	0	1	1	0	1	0	1	1
P069	1	1	1	0	1	1	1	1	0	0	1	0	0	1	0	0	0
P070	1	0	0	1	1	0	1	1	0	0	1	0	0	1	0	1	1
P072	0	0	1	0	1	0	1	1	0	0	0	0	0	1	0	1	0
P073	1	1	1	1	1	0	1	1	0	0	1	1	0	1	0	0	0
P075	0	0	1	0	1	0	1	1	0	0	0	1	0	0	0	1	0
P076	0	1	1	0	1	0	1	1	0	0	1	0	0	1	0	0	0

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
P077	0	0	1	0	1	0	1	1	0	0	1	1	0	0	0	0	0
P078	0	1	0	0	1	0	1	1	0	0	0	0	0	1	0	1	0
P079	0	1	1	1	1	0	1	1	0	0	0	1	0	0	0	0	0
P080	1	1	1	0	1	0	1	1	0	0	1	1	0	0	0	1	0
P081	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0	1	0
P082	1	1	1	0	1	0	1	1	0	0	1	1	0	0	0	1	0
P083	1	1	1	0	1	0	1	1	0	0	1	1	0	0	0	0	0
P085	1	1	1	1	1	0	1	1	0	0	1	0	1	1	0	1	0
P087	1	1	1	1	1	1	1	1	0	0	1	1	0	0	0	1	0
P088	0	1	1	1	1	0	1	1	0	0	0	0	0	1	0	0	0
P089	0	0	1	0	1	0	1	1	0	0	1	1	1	0	0	0	0
P090	0	0	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0
P091	1	1	1	0	1	0	1	1	0	0	1	1	0	0	0	1	0
P092	1	1	1	0	1	0	1	1	0	0	1	1	0	0	0	0	0
P094	1	1	1	0	1	0	1	1	0	0	1	1	0	0	0	1	0
P097	1	1	1	0	1	1	0	0	0	0	1	0	0	1	0	1	0
P099	0	0	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0
P100	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0
P101	0	1	1	0	1	1	0	0	0	0	1	0	0	1	0	1	0
P102	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
P104	1	1	1	0	1	1	0	0	0	0	0	0	0	0	0	1	0
P105	1	1	1	0	1	1	0	0	1	0	1	1	0	1	0	0	0
P106	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
S107	0	0	1	0	1	1	0	0	0	0	0	0	0	0	0	1	0
S108	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0
S109	0	1	0	0	1	1	0	0	0	0	1	0	0	0	0	0	0
S111	0	0	1	0	1	1	0	0	0	0	1	0	0	0	0	1	0
S112	0	0	0	1	1	1	0	0	0	0	1	1	0	0	0	1	0
S113	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
S114	0	1	0	1	1	1	0	0	1	0	1	0	0	1	0	1	0
S115	1	0	1	1	1	1	0	0	1	0	1	1	0	1	0	1	0

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S116	1	0	0	1	1	1	0	0	0	0	1	0	0	0	1	0	1
S117	0	1	1	1	1	1	0	0	1	0	1	1	0	1	0	1	0
S118	1	1	0	1	1	1	0	0	1	0	1	1	0	1	0	1	0
S119	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1	0	0
S120	0	1	0	1	1	1	0	0	0	0	0	0	0	1	0	1	0
S121	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	1	0
S122	0	0	0	0	0	1	0	0	0	0	1	0	0	0	0	1	0
S123	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
S124	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
S125	0	1	1	0	1	1	0	0	0	0	1	1	0	0	0	0	0
S126	0	1	1	0	1	1	0	0	0	0	0	0	0	1	0	0	0
S127	1	1	1	0	1	1	0	0	0	0	1	1	0	0	0	1	0
S128	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
S130	1	0	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0
S132	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0
S134	0	1	0	1	1	1	0	0	0	0	1	1	0	1	0	1	0
S135	0	0	0	0	1	1	1	0	0	0	1	0	0	1	0	0	0
S136	0	0	1	0	1	1	0	0	0	0	0	1	0	0	0	0	0
S137	0	0	1	0	1	1	0	0	0	0	1	0	0	0	0	0	0
S138	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0
S139	1	0	1	0	1	1	0	0	0	0	0	1	0	1	0	1	0
S140	0	0	1	0	1	1	0	0	0	0	1	0	0	0	0	1	0
S141	0	0	1	0	1	1	0	0	1	0	0	1	0	1	0	1	0
S142	0	0	1	0	1	1	0	0	0	0	0	1	0	0	0	0	0
S143	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0
S144	0	1	1	0	1	1	0	0	0	0	1	0	0	1	0	0	0
S146	1	0	0	1	1	1	0	0	0	0	1	1	0	0	0	0	0
S147	0	0	0	0	1	1	0	0	0	0	1	0	0	1	0	0	0
S149	0	0	1	1	1	1	0	0	0	0	0	0	0	0	0	1	0
S150	0	1	1	1	1	1	0	0	1	0	1	1	0	0	0	1	1
S151	1	1	1	1	1	1	0	0	1	0	0	1	0	0	0	1	1

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

Isolate \ Marker	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S152	1	1	0	0	1	1	0	0	1	0	1	0	0	1	0	0	0
S154	0	1	1	1	1	1	0	0	1	0	1	1	0	1	1	0	0
S155	0	0	1	1	1	1	0	0	0	0	0	0	0	0	0	1	0
S156	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	1	0
S157	1	1	1	0	1	1	0	0	1	0	1	0	0	1	0	1	0
S159	1	0	1	0	1	1	0	0	0	0	1	0	0	1	0	1	0
S160	0	1	1	1	1	1	0	0	0	0	0	1	0	1	0	1	0
S161	0	0	0	1	1	1	0	0	0	0	1	1	0	1	0	0	0
S162	0	0	1	0	1	1	1	0	0	0	0	0	0	0	0	0	0
S164	0	1	1	0	1	1	0	0	0	0	0	1	0	1	0	0	0
S165	0	1	1	0	1	1	0	0	0	0	0	0	0	1	0	1	0
S166	0	1	1	0	1	1	0	0	0	0	0	1	0	1	0	1	0
S169	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
S171	0	0	0	1	1	1	0	0	0	0	1	1	0	0	0	1	0
S172	1	0	0	1	1	1	0	0	0	0	1	1	0	0	0	1	0
S173	0	0	0	0	1	1	0	0	0	0	1	0	0	0	1	0	0
S174	0	0	0	1	1	1	0	0	1	0	1	1	0	0	0	1	0
S176	1	0	0	1	1	1	0	0	0	0	0	1	0	0	0	1	0
S177	1	0	0	0	1	0	1	0	0	0	0	0	0	1	0	0	0
S178	0	0	0	1	1	1	0	0	0	0	0	0	0	1	0	1	0
S179	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0
S180	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
S181	1	0	1	0	1	1	0	0	0	0	1	0	0	1	0	1	0
S182	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
S183	0	0	0	0	0	1	0	0	0	0	1	1	0	0	0	0	0
S184	0	0	1	0	1	1	0	0	0	0	0	1	0	0	0	1	0
S185	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0
S186	0	0	1	0	1	1	0	0	0	0	0	1	0	1	0	1	0
S187	0	0	1	0	1	1	0	0	0	0	1	1	0	0	0	1	0
S188	0	1	0	0	1	1	0	0	1	0	1	1	0	0	0	0	0
S189	1	0	0	0	1	1	0	0	1	0	1	1	0	0	0	1	0

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

Isolate	Marker 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
S190	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0
S191	0	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0
S192	0	0	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0
S193	0	0	1	0	1	0	1	0	1	0	0	1	0	1	0	0	1
S194	0	0	0	0	1	1	0	0	1	0	1	1	0	1	0	1	0
S195	1	1	0	0	1	1	0	0	0	0	1	0	0	1	0	0	0
S196	1	0	1	0	1	1	0	0	0	0	0	0	0	0	0	1	0
S197	0	0	1	0	1	1	0	0	0	0	0	0	0	1	1	0	0

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

Isolate	Marker 18	19	20	21	22	23	24	25	26	27	28	29	30
P001	1	0	0	1	0	0	1	1	0	0	0	0	0
P003	1	0	0	1	0	0	0	1	0	0	0	1	0
P004	1	1	1	1	1	1	1	1	0	0	1	1	0
P006	1	0	0	1	0	1	0	1	0	1	1	1	1
P007	1	1	1	1	0	1	0	1	0	0	0	0	0
P008	1	1	1	1	0	0	1	0	0	1	0	0	0
P009	1	0	0	1	0	0	1	0	0	1	0	0	0
P010	1	0	0	1	0	0	1	0	0	1	1	1	1
P011	1	0	0	1	0	1	1	0	0	0	1	0	1
P013	1	1	1	1	0	0	1	1	0	0	1	1	1
P016	1	1	1	1	0	0	1	1	0	0	0	1	1
P017	1	1	1	1	0	0	0	1	0	1	1	1	1
P018	1	0	0	1	1	1	1	1	0	0	1	1	0
P019	1	1	1	1	0	1	1	1	0	1	1	0	1
P022	1	0	0	1	1	0	1	1	0	1	1	1	1
P023	1	0	0	1	0	0	1	1	0	0	1	1	1
P024	1	1	1	1	0	0	1	1	0	0	1	1	1
P026	1	1	1	1	0	1	1	1	0	0	1	1	1
P027	1	0	0	1	0	1	1	1	0	1	1	0	1
P028	1	0	0	1	1	1	0	0	0	1	1	0	1
P029	1	0	0	1	0	1	1	0	0	1	1	0	1
P030	1	0	0	1	0	1	1	0	0	0	1	0	0
P031	1	0	0	1	0	1	1	0	0	1	1	0	1
P032	1	0	0	1	0	1	1	1	0	1	1	0	1
P033	1	0	0	0	0	1	0	0	0	1	1	0	0
P034	1	0	0	1	0	0	0	0	0	0	1	0	0
P036	1	0	0	1	0	1	1	1	0	0	1	0	0
P037	1	1	1	1	0	1	1	1	0	1	1	0	1
P038	1	1	1	1	0	0	1	0	0	1	1	0	0
P039	1	0	0	1	0	0	1	1	0	0	1	1	0
P040	1	0	0	1	0	0	0	0	0	0	1	1	1

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

Isolate	Maker 18	19	20	21	22	23	24	25	26	27	28	29	30
P041	1	0	0	1	0	1	1	1	0	1	1	1	0
P042	1	0	0	1	0	1	0	1	0	1	0	0	0
P043	1	0	0	1	0	0	1	1	0	0	1	1	0
P044	1	0	0	1	0	0	1	1	0	0	1	1	1
P045	1	0	0	1	0	0	1	1	0	0	1	1	1
P046	1	0	0	1	0	0	0	1	0	0	0	1	0
P047	1	0	0	1	0	0	1	1	0	0	1	1	1
P048	1	0	0	1	0	0	1	1	0	0	1	1	1
P049	1	1	1	1	0	0	1	1	0	0	1	1	1
P051	1	0	0	0	1	0	1	1	0	1	1	0	1
P052	1	0	0	1	0	0	1	1	0	1	1	1	0
P053	1	0	0	1	0	1	1	1	0	1	1	0	1
P054	1	0	0	1	0	1	1	1	0	0	1	0	1
P055	1	0	0	1	0	0	1	1	0	1	1	0	1
P056	1	0	0	1	0	1	1	1	0	1	1	0	1
P057	0	0	0	0	0	0	0	1	0	1	1	0	1
P058	1	1	0	1	1	1	1	0	0	0	0	0	0
P059	0	0	0	0	0	0	0	1	0	1	1	1	1
P060	0	0	0	1	0	0	0	1	0	0	0	0	1
P061	1	0	0	0	1	0	0	0	0	1	0	0	1
P062	1	0	0	1	0	0	0	0	0	0	1	1	1
P065	1	0	1	1	0	1	0	1	0	0	1	1	0
P066	1	0	1	1	1	0	1	1	0	1	1	1	1
P067	1	0	1	1	0	0	0	1	0	0	0	1	1
P068	1	0	0	1	0	1	1	0	0	1	1	1	1
P069	1	0	1	1	0	0	1	0	0	1	1	0	1
P070	1	0	0	1	0	1	1	1	0	0	1	1	1
P072	1	0	1	1	0	0	1	1	0	1	1	1	1
P073	1	0	1	1	0	0	1	1	0	0	1	1	1
P075	1	0	0	1	0	0	1	0	0	1	1	1	1
P076	1	0	1	0	0	1	0	0	0	0	0	1	0

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

Isolate	Marker 18	19	20	21	22	23	24	25	26	27	28	29	30
P077	1	0	0	1	0	0	0	1	0	0	1	1	1
P078	1	0	1	1	0	1	0	1	0	0	1	1	1
P079	1	0	0	1	0	0	1	1	0	0	1	0	1
P080	1	0	0	1	0	0	0	1	0	1	1	0	1
P081	1	0	0	0	0	0	1	0	0	1	1	0	0
P082	1	0	1	1	0	0	0	1	0	0	1	0	0
P083	1	0	0	1	0	1	0	1	0	0	1	0	1
P085	1	0	0	1	0	0	1	1	0	0	1	0	0
P087	1	0	0	1	0	0	1	1	0	1	1	1	1
P088	1	0	0	0	0	0	0	1	0	0	0	1	1
P089	1	0	1	0	0	0	0	1	0	1	1	1	1
P090	0	0	0	0	0	1	0	1	0	1	0	0	0
P091	1	0	0	1	1	0	0	0	0	0	1	0	1
P092	1	0	0	1	0	0	1	1	0	1	1	1	1
P094	1	0	0	1	0	0	0	1	0	0	0	1	1
P097	1	0	1	1	0	1	1	1	0	0	1	0	1
P099	0	0	0	1	0	0	1	0	0	0	0	0	0
P100	0	0	1	0	0	0	1	0	0	1	1	0	1
P101	1	0	1	1	0	1	1	1	0	0	1	0	0
P102	0	0	0	0	0	0	0	0	0	1	0	0	1
P104	1	0	1	1	0	1	0	1	0	1	1	0	0
P105	1	0	1	1	0	0	1	1	0	0	1	0	1
P106	1	0	1	0	0	0	0	1	0	1	1	0	1
S107	1	0	0	0	0	1	0	1	0	0	1	0	1
S108	0	0	0	0	0	0	0	0	0	0	0	0	0
S109	0	0	1	1	1	0	1	1	0	0	0	0	1
S111	1	0	1	0	0	1	0	0	0	0	0	1	1
S112	1	0	0	1	0	0	1	1	0	1	1	1	1
S113	1	0	0	1	0	0	0	0	0	0	1	0	0
S114	1	0	0	1	0	0	1	0	0	0	1	1	1
S115	1	0	1	0	0	1	1	0	0	0	0	1	0

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

Isolate \ Marker	18	19	20	21	22	23	24	25	26	27	28	29	30
S116	0	0	1	1	0	0	1	1	0	0	0	0	1
S117	1	0	1	0	0	1	1	1	0	0	1	1	0
S118	1	0	1	1	0	0	0	0	0	0	0	0	0
S119	1	0	0	1	0	1	1	0	0	0	0	1	0
S120	1	0	1	1	0	0	0	0	0	0	0	0	0
S121	1	0	0	1	0	0	0	0	0	0	0	0	0
S122	0	0	0	0	0	0	0	1	0	0	0	0	0
S123	0	1	0	1	0	0	0	1	0	0	0	0	0
S124	1	0	0	1	0	0	0	1	0	0	0	0	0
S125	1	0	1	1	0	1	1	1	0	1	1	0	1
S126	1	0	1	1	0	1	1	1	0	0	1	0	1
S127	1	0	1	0	0	0	0	0	0	0	1	0	0
S128	0	0	0	0	0	0	0	0	0	0	0	0	0
S130	1	0	0	1	0	0	0	0	0	0	1	1	0
S132	1	0	0	0	0	0	0	1	0	0	1	0	1
S134	1	0	0	1	0	0	1	1	0	0	1	1	0
S135	0	0	0	1	0	1	0	0	0	1	0	0	0
S136	1	0	0	1	0	0	0	1	0	0	0	1	0
S137	1	0	1	0	0	0	0	1	0	1	0	1	0
S138	0	0	0	0	0	0	0	0	0	0	0	1	1
S139	1	0	0	0	0	0	0	0	0	0	0	0	0
S140	1	0	0	0	0	1	1	1	0	1	1	0	1
S141	1	0	0	1	0	0	0	1	0	1	1	0	1
S142	1	0	0	1	0	0	0	0	0	0	0	1	1
S143	0	0	1	0	0	0	0	0	0	0	0	1	1
S144	1	0	0	1	0	0	0	0	0	0	1	0	0
S146	1	0	0	1	0	0	1	0	0	0	0	0	0
S147	1	0	0	1	0	0	0	0	0	1	0	1	0
S149	1	0	0	0	0	0	0	1	1	0	0	0	1
S150	1	0	0	1	1	0	1	1	0	0	1	1	1
S151	1	0	0	1	0	1	1	0	0	0	1	1	1

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

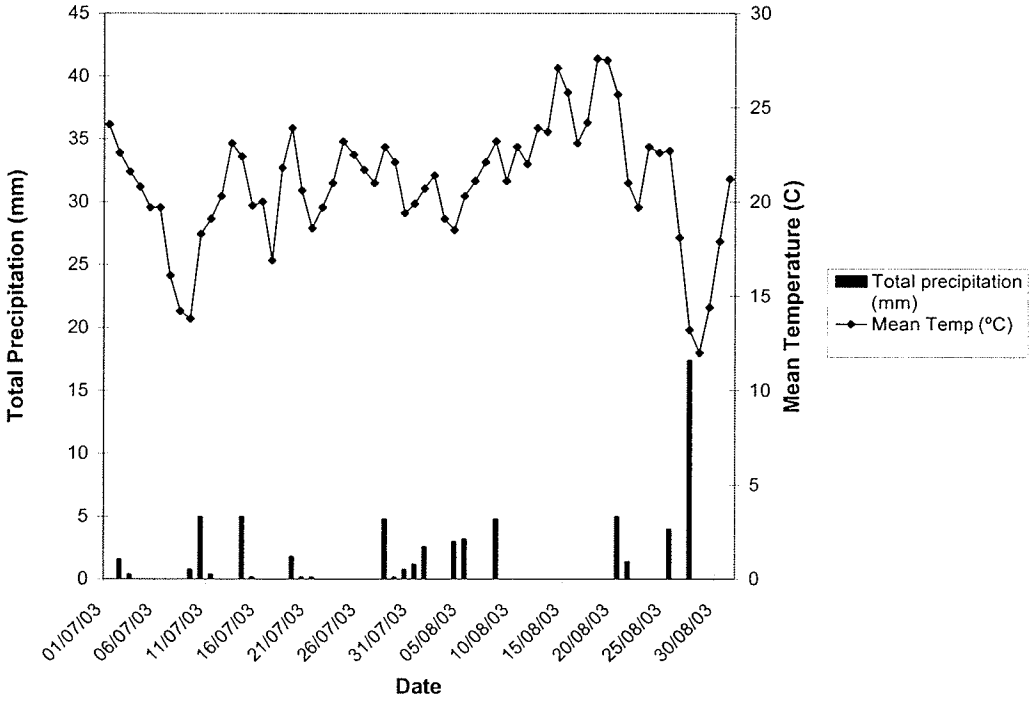
Isolate \ Marker	18	19	20	21	22	23	24	25	26	27	28	29	30
S152	1	0	0	0	1	0	0	0	0	0	0	0	0
S154	1	0	1	1	0	1	0	1	0	1	0	1	1
S155	1	0	1	1	0	1	1	0	0	0	0	1	0
S156	1	0	0	1	0	0	1	1	0	0	1	1	0
S157	1	0	0	1	0	1	1	0	0	0	1	1	0
S159	1	0	0	1	0	1	1	1	0	0	1	1	1
S160	1	0	0	0	0	1	1	0	0	1	1	1	1
S161	1	0	1	1	0	1	1	0	0	0	1	1	0
S162	1	0	1	0	0	0	0	0	0	0	1	0	0
S164	1	0	0	1	0	0	0	0	0	0	0	1	0
S165	1	1	1	0	0	1	1	1	0	0	0	1	1
S166	1	0	1	1	0	1	0	1	0	0	0	1	1
S169	0	0	0	0	0	0	0	0	0	0	0	0	0
S171	1	0	1	0	0	0	0	0	0	0	0	0	1
S172	1	0	0	1	0	0	0	0	0	0	0	1	1
S173	0	0	0	0	0	1	0	0	0	0	0	0	1
S174	1	0	0	1	0	0	1	1	0	1	1	1	0
S176	1	0	1	1	0	0	0	0	0	0	1	1	1
S177	1	0	1	0	0	0	0	1	0	1	1	1	0
S178	1	0	1	1	0	0	1	1	0	0	1	1	1
S179	1	0	1	1	0	0	0	0	0	0	1	1	0
S180	1	0	0	0	0	0	0	0	0	0	0	0	1
S181	1	0	1	1	0	1	0	0	0	0	1	1	1
S182	1	0	0	0	0	1	0	1	0	1	0	1	1
S183	1	0	0	0	0	0	0	1	0	1	1	0	1
S184	1	0	0	1	0	0	1	1	0	1	1	1	0
S185	1	0	0	0	0	0	0	0	0	1	0	1	1
S186	1	0	1	1	0	0	1	1	0	1	1	1	1
S187	1	0	0	1	0	1	1	1	0	0	1	1	0
S188	1	0	1	1	0	1	0	0	1	0	1	1	0
S189	1	0	0	1	0	1	1	1	0	0	1	1	1

Appendix 3 f: RAPD binary scores for isolates at Portage la Prairie and Sanford, Manitoba, Canada using primer 681.

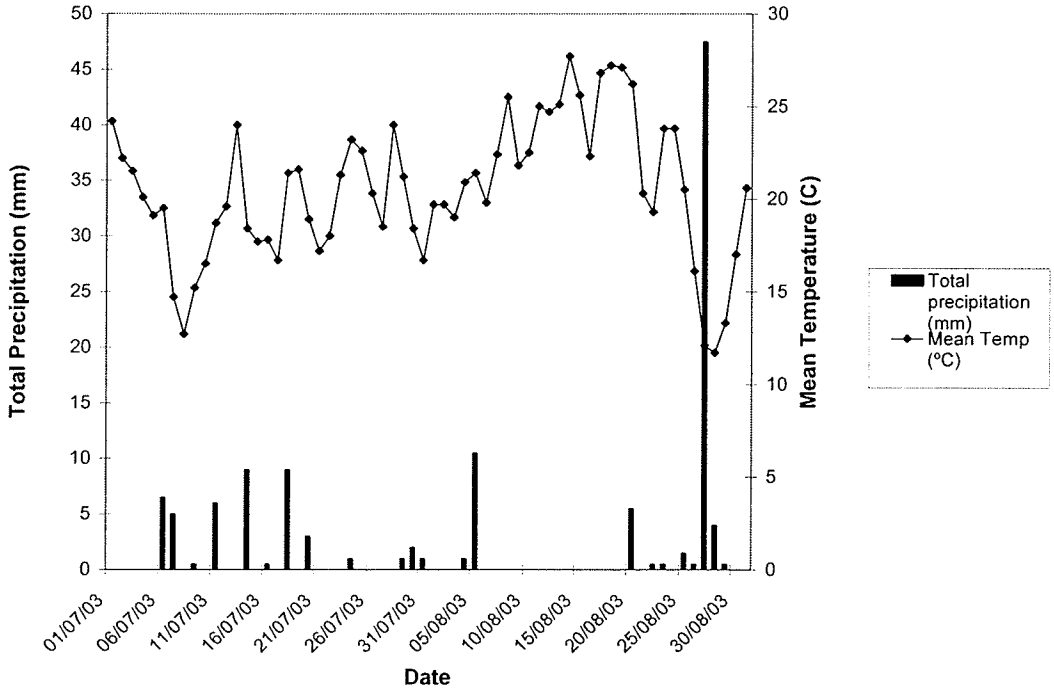
	Marker												
Isolate	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**	**30**
S190	1	0	0	1	0	0	0	0	0	0	0	1	1
S191	0	0	1	0	1	0	0	0	0	1	1	0	1
S192	0	0	0	1	0	0	0	0	0	1	0	0	1
S193	1	0	1	0	0	1	0	0	0	0	1	0	0
S194	1	0	1	1	0	1	1	1	0	1	1	0	1
S195	1	0	1	0	0	0	1	1	0	1	1	1	1
S196	1	0	1	0	0	0	0	0	0	0	0	0	0
S197	1	0	1	1	0	1	0	1	0	0	1	0	1

APPENDIX 4: Weather Data

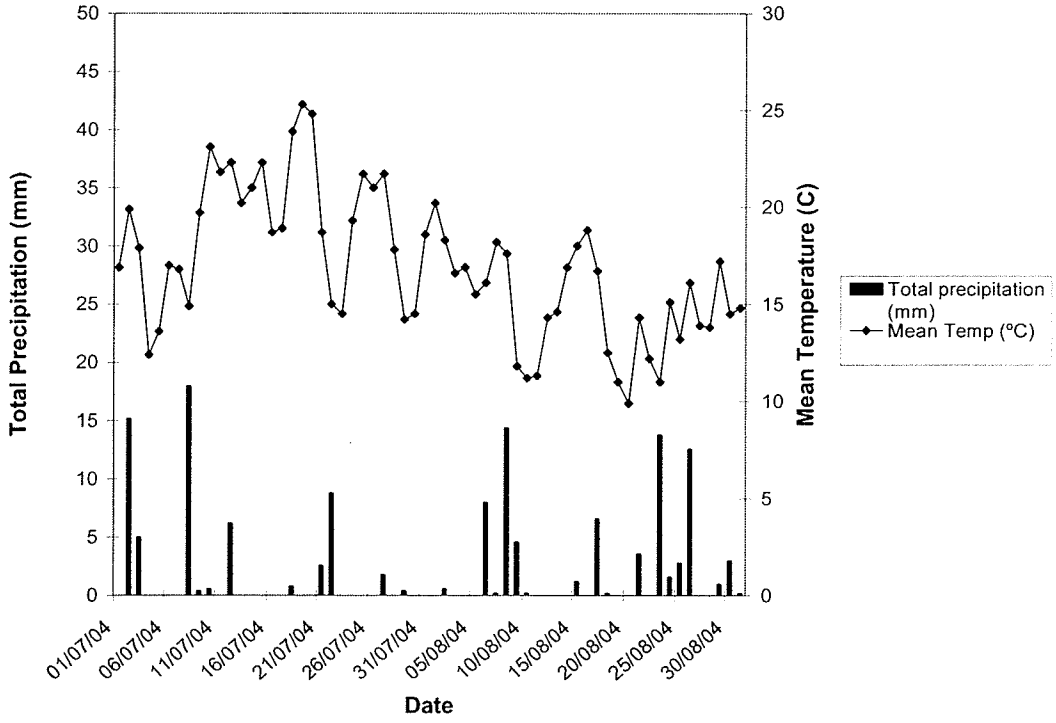
Appendix 4. a: Daily rainfall and mean temperature at Morden, Manitoba, Canada at the Agriculture and Agri-Food Research Station in 2003.



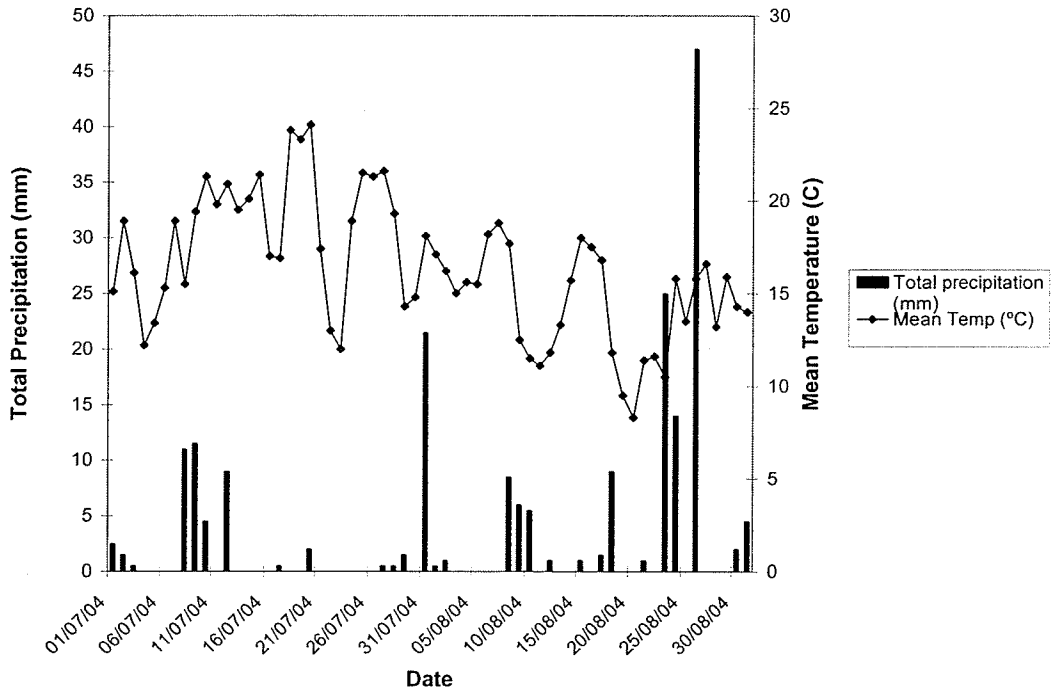
Appendix 4. b: Daily rainfall and mean temperature at Winnipeg, Manitoba, Canada at the University of Manitoba Field Station in 2003.



Appendix 4. c: Daily rainfall and mean temperature at Morden, Manitoba, Canada at the Agriculture and Agri-Food Research Station in 2004.



Appendix 4. d: Daily rainfall and mean temperature at Winnipeg, Manitoba, Canada at the University of Manitoba Field Station in 2004.



Appendix 5: Calculation of the Relative Difference Between Control and other Treatments

$$\frac{\text{Treatment value}}{\text{Control value}} \times 100 = \%\ \text{Difference}$$

Appendix 6: Supporting Data

Cultivar	Treatment	Rep	Plot	Location	Yield (g/m^2)	Oil content (%)	Protein content (%)	1000 kernel weight (g)	AUDPC	Year
AC Emerson	I/NF	1	1	Morden	408.80	44.7	24.0	5.3	233.5	2003
AC Emerson	I/NF	2	9	Morden	297.27	40.6	25.1	4.9	267	2003
AC Emerson	I/NF	3	13	Morden	386.54	42.7	23.9	5.1	253.5	2003
AC Emerson	I/NF	4	19	Morden	416.91	44.4	22.9	5.3	230	2003
AC Emerson	NI/F	1	25	Morden	421.68	45.1	24.8	6.6	120.5	2003
AC Emerson	NI/F	2	34	Morden	505.79	43.9	25.1	6.5	127.5	2003
AC Emerson	NI/F	3	42	Morden	516.87	45.6	22.9	6.4	121	2003
AC Emerson	NI/F	4	44	Morden	442.46	46.2	21.3	6.4	125.5	2003
AC Linora	I/NF	1	5	Morden	339.14	43.1	23.7	4.5	228	2003
AC Linora	I/NF	2	11	Morden	331.04	41.5	24.0	4.1	242	2003
AC Linora	I/NF	3	18	Morden	313.37	41.4	25.4	4.3	217	2003
AC Linora	I/NF	4	21	Morden	364.50	43.6	22.2	4.4	242	2003
AC Linora	NI/F	1	27	Morden	487.25	47.2	24.7	6	102	2003
AC Linora	NI/F	2	35	Morden	577.40	45.5	25.4	6.4	120.5	2003
AC Linora	NI/F	3	38	Morden	528.59	46.3	23.6	6.1	141.5	2003
AC Linora	NI/F	4	43	Morden	403.29	45.6	23.5	6.1	124.5	2003
AC Macbeth	I/NF	1	3	Morden	391.14	45.1	25.1	4.6	187.5	2003
AC Macbeth	I/NF	2	12	Morden	260.78	40.2	27.6	4.3	210	2003
AC Macbeth	I/NF	3	17	Morden	297.45	43.1	25.3	4.5	234.5	2003
AC Macbeth	I/NF	4	20	Morden	403.29	45.8	23.8	5	202	2003
AC Macbeth	NI/F	1	30	Morden	554.22	47.5	25.3	6.6	105	2003
AC Macbeth	NI/F	2	31	Morden	455.52	45.9	26.0	6.5	123	2003
AC Macbeth	NI/F	3	37	Morden	422.5	46.7	25.7	6.5	123.5	2003
AC Macbeth	NI/F	4	48	Morden	508.89	48.4	22.0	6.2	130.5	2003
McGregor	I/NF	1	2	Morden	407.70	42.1	23.7	4.3	195.5	2003
McGregor	I/NF	2	10	Morden	298.74	39.2	25.4	3.9	239	2003
McGregor	I/NF	3	15	Morden	329.88	39.6	24.5	4	241.5	2003
McGregor	I/NF	4	24	Morden	311.94	42.7	23.0	4.1	210	2003
McGregor	NI/F	1	26	Morden	521.96	43.6	26.8	5.6	120	2003
McGregor	NI/F	2	32	Morden	540.99	42.8	27.0	5.7	141.5	2003
McGregor	NI/F	3	39	Morden	557.11	44.7	25.1	5.6	99.5	2003
McGregor	NI/F	4	47	Morden	509.37	44.6	25.0	5.5	155.5	2003

Appendix 6: Supporting data

Cultivar	Treatement	Rep	Plot	Location	Yield (g/m^2)	Oil content (%)	Protein content (%)	1000 kernel weight (g)	AUDPC	Year
NorLin	I/NF	1	4	Morden	377.32	42.4	23.4	5.3	235.5	2003
NorLin	I/NF	2	8	Morden	319.11	43.0	23.4	5.1	249.5	2003
NorLin	I/NF	3	16	Morden	225.28	40.1	25.2	4.8	295.5	2003
NorLin	I/NF	4	23	Morden	316.89	42.2	22.9	5.0	236	2003
NorLin	NI/F	1	28	Morden	541.20	45.7	24.5	6.5	106	2003
NorLin	NI/F	2	36	Morden	552.10	43.9	25.8	6.7	106	2003
NorLin	NI/F	3	40	Morden	507.60	46.5	22.7	6.7	124.5	2003
NorLin	NI/F	4	46	Morden	530.49	46.3	21.9	6.7	124.5	2003
Vimy	I/NF	1	6	Morden	281.79	40.4	25.8	4.6	221	2003
Vimy	I/NF	2	7	Morden	381.96	43.7	25.1	5.0	227	2003
Vimy	I/NF	3	14	Morden	162.70	40.9	26.3	4.4	306.5	2003
Vimy	I/NF	4	22	Morden	311.86	43.6	22.6	5.0	262.5	2003
Vimy	NI/F	1	29	Morden	510.84	46.3	24.8	6.5	109	2003
Vimy	NI/F	2	33	Morden	260.21	42.9	27.1	6.3	134	2003
Vimy	NI/F	3	41	Morden	434.21	46.3	23.4	6.2	124.5	2003
Vimy	NI/F	4	45	Morden	486.95	46.3	23.3	6.5	131.5	2003
AC Emerson	I/NF	1	1	Winnipeg	271.52	42.7	24.7	5.7	175	2003
AC Emerson	I/NF	2	10	Winnipeg	280.79	42.0	24.4	4.5	221.5	2003
AC Emerson	I/NF	3	18	Winnipeg	254.02	44.0	22.0	5.4	224.5	2003
AC Emerson	I/NF	4	20	Winnipeg	250.64	43.0	24.0	5.7	234	2003
AC Emerson	NI/F	1	25	Winnipeg	575.24	42.5	28.1	6.7	107.5	2003
AC Emerson	NI/F	2	33	Winnipeg	497.84	42.8	27.7	6.8	107.5	2003
AC Emerson	NI/F	3	37	Winnipeg	870.09	44.6	26.0	6.7	107.5	2003
AC Emerson	NI/F	4	43	Winnipeg	595.21	44.1	27.3	6.8	107.5	2003
AC Linora	I/NF	1	3	Winnipeg	358.24	43.6	23.3	4.1	261	2003
AC Linora	I/NF	2	11	Winnipeg	309.41	43.0	23.7	5.1	226	2003
AC Linora	I/NF	3	14	Winnipeg	268.54	42.2	23.7	5.1	221	2003
AC Linora	I/NF	4	19	Winnipeg	278.14	43.6	23.1	4.1	245.5	2003
AC Linora	NI/F	1	29	Winnipeg	675.07	44.8	27.4	6.0	135.5	2003
AC Linora	NI/F	2	35	Winnipeg	598.65	44.2	27.6	6.2	107.5	2003
AC Linora	NI/F	3	42	Winnipeg	675.85	43.9	28.4	6.1	114	2003
AC Linora	NI/F	4	45	Winnipeg	577.10	44.6	27.0	6.0	135.5	2003

Appendix 6: Supporting data

Cultivar	Treatement	Rep	Plot	Location	Yield (g/m^2)	Oil content (%)	Protein content (%)	1000 kernel weight (g)	AUDPC	Year
AC Macbeth	I/NF	1	6	Winnipeg	295.21	44.3	24.2	4.8	221	2003
AC Macbeth	I/NF	2	7	Winnipeg	313.31	44.2	23.4	5.2	242	2003
AC Macbeth	I/NF	3	13	Winnipeg	278.95	43.4	24.0	4.8	230	2003
AC Macbeth	I/NF	4	24	Winnipeg	210.34	47.0	22.0	5.3	199	2003
AC Macbeth	NI/F	1	27	Winnipeg	593.99	45.2	27.6	6.6	107.5	2003
AC Macbeth	NI/F	2	36	Winnipeg	597.14	45.2	26.7	6.6	107.5	2003
AC Macbeth	NI/F	3	41	Winnipeg	703.28	45.0	27.9	6.5	126	2003
AC Macbeth	NI/F	4	44	Winnipeg	634.10	46.0	26.7	6.4	111	2003
McGregor	I/NF	1	2	Winnipeg	274.74	41.0	24.7	3.7	231	2003
McGregor	I/NF	2	8	Winnipeg	206.17	41.4	24.3	3.9	195	2003
McGregor	I/NF	3	15	Winnipeg	243.54	41.4	23.9	4.1	214	2003
McGregor	I/NF	4	23	Winnipeg	225.69	42.0	22.8	4.3	205.5	2003
McGregor	NI/F	1	26	Winnipeg	528.99	41.7	28.8	5.4	107	2003
McGregor	NI/F	2	34	Winnipeg	537.04	41.7	27.7	5.4	104	2003
McGregor	NI/F	3	39	Winnipeg	560.46	42.1	28.7	5.3	107.5	2003
McGregor	NI/F	4	48	Winnipeg	528.53	42.0	28.5	5.3	104.5	2003
NorLin	I/NF	1	4	Winnipeg	290.52	43.3	32.7	4.9	250.5	2003
NorLin	I/NF	2	12	Winnipeg	275.70	42.7	24.1	5.1	245.5	2003
NorLin	I/NF	3	16	Winnipeg	266.97	44.9	22.1	5.4	198.5	2003
NorLin	I/NF	4	22	Winnipeg	194.17	43.9	22.5	5.4	192	2003
NorLin	NI/F	1	28	Winnipeg	621.63	43.4	26.9	6.7	111	2003
NorLin	NI/F	2	32	Winnipeg	512.67	44.4	27.4	6.5	107.5	2003
NorLin	NI/F	3	40	Winnipeg	584.44	43.7	27.5	6.5	107.5	2003
NorLin	NI/F	4	47	Winnipeg	580.27	42.6	27.8	6.5	104	2003
Vimy	I/NF	1	5	Winnipeg	292.84	42.6	25.2	5.0	270	2003
Vimy	I/NF	2	9	Winnipeg	248.61	44.1	22.9	5.2	210.5	2003
Vimy	I/NF	3	17	Winnipeg	.	.	.	.	224.5	2003
Vimy	I/NF	4	21	Winnipeg	318.73	43.9	24.3	6.2	238.5	2003
Vimy	NI/F	1	30	Winnipeg	613.44	43.4	28.4	6.5	150	2003
Vimy	NI/F	2	31	Winnipeg	547.13	43.7	28.5	6.8	122	2003
Vimy	NI/F	3	38	Winnipeg	507.68	44.4	28.5	6.7	127.5	2003
Vimy	NI/F	4	46	Winnipeg	514.18	44.4	28.5	6.7	115	2003

Appendix 6: Supporting data

Cultivar	Treatment	Rep	Plot	Location	Yield (g/m^2)	Oil content (%)	Protein content (%)	1000 kernel weight	AUDPC	Year
AC Emerson	I/F	1	50	Morden	432.94	44.1	24.5	6.74	136	2004
AC Emerson	I/F	2	59	Morden	292.97	43.4	24.4	6.54	151	2004
AC Emerson	I/F	3	63	Morden	436.29	43.9	25.1	6.91	136	2004
AC Emerson	I/F	4	70	Morden	497.46	44.6	24.8	6.99	136	2004
AC Emerson	I/NF	1	74	Morden	138.14	39.9	25.5	5.04	196	2004
AC Emerson	I/NF	2	83	Morden	223.08	40.4	24.1	5.3	178	2004
AC Emerson	I/NF	3	88	Morden	88.26	41.4	23.2	5.52	168	2004
AC Emerson	I/NF	4	96	Morden	220.84	41.4	23.8	5.36	178	2004
AC Emerson	NI/F	1	30	Morden	442.39	44.2	24.9	7.06	103.5	2004
AC Emerson	NI/F	2	32	Morden	466.35	43.4	25.8	7.07	109.5	2004
AC Emerson	NI/F	3	38	Morden	429.33	44.0	24.3	6.95	116.5	2004
AC Emerson	NI/F	4	48	Morden	453.24	44.5	25.5	7.09	96	2004
AC Emerson	NI/NF	1	05	Morden	203.36	42.2	23.5	5.59	186.5	2004
AC Emerson	NI/NF	2	12	Morden	297.74	42.6	23.9	5.85	161.5	2004
AC Emerson	NI/NF	3	16	Morden	309.62	42.5	23.5	5.91	176.5	2004
AC Emerson	NI/NF	4	24	Morden	409.45	43.1	22.4	5.84	170.5	2004
AC Linora	I/F	1	52	Morden	341.83	44.5	24.4	6.59	140.5	2004
AC Linora	I/F	2	55	Morden	247.17	43.0	26.2	6.32	140	2004
AC Linora	I/F	3	65	Morden	406.78	44.3	25.1	6.49	136	2004
AC Linora	I/F	4	67	Morden	421.40	42.9	26.6	6.69	136	2004
AC Linora	I/NF	1	77	Morden	196.13	40.8	23.3	5.01	168	2004
AC Linora	I/NF	2	84	Morden	199.43	40.9	23.8	5.3	154	2004
AC Linora	I/NF	3	90	Morden	154.02	40.7	23.8	5.21	169	2004
AC Linora	I/NF	4	91	Morden	191.0	41.0	24.6	5.44	155	2004
AC Linora	NI/F	1	25	Morden	248.39	42.6	27.0	6.82	110	2004
AC Linora	NI/F	2	34	Morden	444.95	45.0	24.8	6.76	116.5	2004
AC Linora	NI/F	3	40	Morden	416.39	44.7	24.9	6.95	116.5	2004
AC Linora	NI/F	4	44	Morden	420.75	43.3	25.8	6.81	116.5	2004
AC Linora	NI/NF	1	6	Morden	240.42	42.0	23.0	5.27	187	2004
AC Linora	NI/NF	2	9	Morden	392.54	43.6	21.7	5.78	157.5	2004
AC Linora	NI/NF	3	13	Morden	228.27	43.5	22.7	5.43	157.5	2004
AC Linora	NI/NF	4	21	Morden	304.0	43.8	22.1	5.37	157.5	2004

Appendix 6: Supporting data

Cultivar	Treatment	Rep	Plot	Location	Yield (g/m^2)	Oil content %	Protein content (%)	1000 kernel weight	AUDPC	Year
AC Macbeth	I/F	1	49	Morden	311.96	44.7	27.2	7.17	140.5	2004
AC Macbeth	I/F	2	57	Morden	291.18	44.6	25.7	6.5	140.5	2004
AC Macbeth	I/F	3	66	Morden	438.63	45.7	25.4	6.83	140.5	2004
AC Macbeth	I/F	4	72	Morden	445.5	46.7	24.3	7.03	136	2004
AC Macbeth	I/NF	1	76	Morden	255.86	42.9	24.6	5.51	168	2004
AC Macbeth	I/NF	2	82	Morden	259.23	42.5	24.6	5.4	164	2004
AC Macbeth	I/NF	3	87	Morden	318.68	43.6	24.7	5.68	148.5	2004
AC Macbeth	I/NF	4	94	Morden	243.19	43.9	24.4	5.69	144	2004
AC Macbeth	NI/F	1	28	Morden	534.83	45.8	25.3	7.29	110	2004
AC Macbeth	NI/F	2	31	Morden	212.28	44.7	27.4	6.84	109.5	2004
AC Macbeth	NI/F	3	42	Morden	457.62	47.0	24.4	7.06	116	2004
AC Macbeth	NI/F	4	47	Morden	429.55	45.5	26.7	7.14	106	2004
AC Macbeth	NI/NF	1	4	Morden	296.86	43.6	23.9	5.64	175.5	2004
AC Macbeth	NI/NF	2	8	Morden	356.85	44.2	23.9	6.01	158	2004
AC Macbeth	NI/NF	3	17	Morden	283.00	45.3	22.5	5.95	157.5	2004
AC Macbeth	NI/NF	4	19	Morden	249.21	43.9	23.3	5.82	157.5	2004
McGregor	I/F	1	53	Morden	349.63	43.5	26.0	6.02	136	2004
McGregor	I/F	2	60	Morden	297.29	43.7	25.0	5.96	136	2004
McGregor	I/F	3	62	Morden	337.88	42.4	26.1	5.97	136	2004
McGregor	I/F	4	68	Morden	429.20	43.4	24.7	5.9	136	2004
McGregor	I/NF	1	73	Morden	126.78	39.3	24.4	4.26	158	2004
McGregor	I/NF	2	79	Morden	166.69	40.1	24.8	4.69	144	2004
McGregor	I/NF	3	86	Morden	189.28	39.6	23.9	4.7	158	2004
McGregor	I/NF	4	95	Morden	227.63	41.2	23.0	4.48	144	2004
McGregor	NI/F	1	27	Morden	434.22	42.7	26.8	6.1	116.5	2004
McGregor	NI/F	2	36	Morden	379.84	43.2	26.5	5.97	96	2004
McGregor	NI/F	3	37	Morden	332.19	43.5	25.5	6.08	96	2004
McGregor	NI/F	4	46	Morden	342.36	43.2	26.5	6.15	96	2004
McGregor	NI/NF	1	3	Morden	207.5	40.4	24.0	4.76	175.5	2004
McGregor	NI/NF	2	10	Morden	300.23	42.4	22.3	5.2	161.5	2004
McGregor	NI/NF	3	18	Morden	335.59	42.5	22.4	4.94	157.5	2004
McGregor	NI/NF	4	20	Morden	304.86	42.2	22.8	5.02	157.5	2004

Appendix 6: Supporting data

Cultivar	Treatment	Rep	Plot	Location	Yield (g/m^2)	Oil content (%)	Protein content (%)	1000 kernel weight	AUDPC	Year
NorLin	I/F	1	51	Morden	459.09	43.5	24.2	6.88	136	2004
NorLin	I/F	2	56	Morden	279.86	44.6	23.3	6.52	143.5	2004
NorLin	I/F	3	61	Morden	286.98	42.8	26.5	6.7	136	2004
NorLin	I/F	4	71	Morden	432.68	43.0	24.9	7.17	136	2004
NorLin	I/NF	1	75	Morden	157.96	40.6	23.8	5.14	188	2004
NorLin	I/NF	2	80	Morden	139.58	40.2	24.5	5.12	168	2004
NorLin	I/NF	3	85	Morden	122.33	39.8	25.3	5.2	168	2004
NorLin	I/NF	4	93	Morden	237.38	41.4	23.2	5.48	178	2004
NorLin	NI/F	1	29	Morden	504.98	43.1	25.3	6.78	111	2004
NorLin	NI/F	2	35	Morden	276.37	42.8	25.6	6.84	121	2004
NorLin	NI/F	3	39	Morden	441.36	43.6	25.6	7.14	111	2004
NorLin	NI/F	4	43	Morden	370.39	43.0	27.2	6.81	111	2004
NorLin	NI/NF	1	1	Morden	267.29	42.1	23.1	5.35	187	2004
NorLin	NI/NF	2	11	Morden	223.94	40.6	24.5	5.68	195	2004
NorLin	NI/NF	3	14	Morden	267.03	42.8	22.8	5.82	193	2004
NorLin	NI/NF	4	23	Morden	329.84	41.6	23.9	5.68	190.5	2004
Vimy	I/F	1	54	Morden	226.67	43.9	25.4	6.75	140.5	2004
Vimy	I/F	2	58	Morden	238.01	43.6	25.2	6.51	136	2004
Vimy	I/F	3	64	Morden	349.38	44.1	24.9	6.89	140.5	2004
Vimy	I/F	4	69	Morden	322.54	44.0	25.1	6.92	143.5	2004
Vimy	I/NF	1	78	Morden	154.75	41.4	23.8	5.28	168	2004
Vimy	I/NF	2	81	Morden	129.30	41.0	23.4	5.5	190.5	2004
Vimy	I/NF	3	89	Morden	129.41	40.7	24.1	5.05	168	2004
Vimy	I/NF	4	92	Morden	223.05	41.1	24.0	5.32	168	2004
Vimy	NI/F	1	26	Morden	252.17	44.2	24.5	7.03	110	2004
Vimy	NI/F	2	33	Morden	238.78	43.9	26.5	6.98	109.5	2004
Vimy	NI/F	3	41	Morden	267.88	44.2	25.9	6.7	110	2004
Vimy	NI/F	4	45	Morden	250.37	44.5	26.2	6.98	103.5	2004
Vimy	NI/NF	1	2	Morden	212.27	41.5	24.4	5.19	190	2004
Vimy	NI/NF	2	07	Morden	143.96	41.5	23.7	5.79	187	2004
Vimy	NI/NF	3	15	Morden	130.93	41.36	24.8	5.66	186.5	2004
Vimy	NI/NF	4	22	Morden	239.65	42.4	23.6	5.37	178	2004

Appendix 6: Supporting data

Cultivar	Treatment	Rep	Plot	Location	Yield (g/m^2)	Oil content (%)	Protein content (%)	1000 kernel weight	AUDPC	Year
AC Emerson	I/F	1	52	Winnipeg	287.50	42.8	21.8	5.94	119	2004
AC Emerson	I/F	2	58	Winnipeg	355.05.	42.0	21.7	6.02	115.5	2004
AC Emerson	I/F	3	66	Winnipeg	221.06	43.2	23.6	6.18	115.5	2004
AC Emerson	I/F	4	67	Winnipeg	177.55	43.1	21.9	5.74	115.5	2004
AC Emerson	I/NF	1	75	Winnipeg	141.93	40.8	24.0	5.37	141	2004
AC Emerson	I/NF	2	81	Winnipeg	147.10	41.0	24.0	5.28	137.5	2004
AC Emerson	I/NF	3	89	Winnipeg	88.80	41.0	23.7	5.62	130	2004
AC Emerson	I/NF	4	94	Winnipeg	149.00	41.2	23.1	5.25	155.5	2004
AC Emerson	NI/F	1	29	Winnipeg	269.82	43.7	21.4	6.18	109.5	2004
AC Emerson	NI/F	2	31	Winnipeg	354.99	43.4	21.1	6.42	106	2004
AC Emerson	NI/F	3	42	Winnipeg	275.28	42.8	21.4	6.36	109.5	2004
AC Emerson	NI/F	4	48	Winnipeg	250.01	42.3	21.1	6.42	109.5	2004
AC Emerson	NI/NF	1	3	Winnipeg	259.13	41.2	24.4	5.82	113	2004
AC Emerson	NI/NF	2	7	Winnipeg	237.15	41.3	23.9	5.93	113	2004
AC Emerson	NI/NF	3	15	Winnipeg	232.68	41.4	23.4	5.66	109.5	2004
AC Emerson	NI/NF	4	19	Winnipeg	260.18	41.4	24.0	5.76	109.5	2004
AC Linora	I/F	1	49	Winnipeg	219.71	42.8	20.9	5.37	112	2004
AC Linora	I/F	2	57	Winnipeg	325.33	42.8	21.8	5.33	115.5	2004
AC Linora	I/F	3	61	Winnipeg	248.01	41.8	22.1	5.16	115.5	2004
AC Linora	I/F	4	71	Winnipeg	251.84	43.4	21.7	5.59	112	2004
AC Linora	I/NF	1	74	Winnipeg	152.31	41.4	22.3	5.2	115.5	2004
AC Linora	I/NF	2	84	Winnipeg	194.65	40.7	22.2	4.82	115.5	2004
AC Linora	I/NF	3	85	Winnipeg	208.43	40.9	21.2	4.78	126.5	2004
AC Linora	I/NF	4	91	Winnipeg	162.63	41.5	21.2	5.07	115.5	2004
AC Linora	NI/F	1	27	Winnipeg	294.89	44.3	20.0	5.95	109.5	2004
AC Linora	NI/F	2	32	Winnipeg	314.05	44.1	19.6	5.85	109.5	2004
AC Linora	NI/F	3	41	Winnipeg	238.09	43.6	19.9	5.89	109.5	2004
AC Linora	NI/F	4	43	Winnipeg	287.49	43.7	20.7	6.07	106	2004
AC Linora	NI/NF	1	5	Winnipeg	297.45	40.7	23.3	5.13	109.5	2004
AC Linora	NI/NF	2	10	Winnipeg	261.82	40.6	22.5	5.24	109.5	2004
AC Linora	NI/NF	3	18	Winnipeg	235.63	40.7	21.6	5.22	113	2004
AC Linora	NI/NF	4	24	Winnipeg	210.17	41.1	21.3	5.19	109.5	2004

Appendix 6: Supporting data

Cultivar	Treatment	Rep	Plot	Location	Yield (g/m^2)	Oil content (%)	Protein content (%)	1000 kernel weight	AUDPC	Year
AC Macbeth	I/F	1	53	Winnipeg	202.01	44.2	22.0	5.61	115.5	2004
AC Macbeth	I/F	2	60	Winnipeg	225.03	43.7	22.2	5.69	115.5	2004
AC Macbeth	I/F	3	63	Winnipeg	176.41	43.6	22.9	5.46	115.5	2004
AC Macbeth	I/F	4	68	Winnipeg	242.60	43.5	22.5	5.41	115.5	2004
AC Macbeth	I/NF	1	78	Winnipeg	68.59	41.7	22.9	5.20	115.5	2004
AC Macbeth	I/NF	2	79	Winnipeg	102.32	41.2	23.7	5.04	126.5	2004
AC Macbeth	I/NF	3	88	Winnipeg	50.90	41.7	23.0	5.25	115.5	2004
AC Macbeth	I/NF	4	95	Winnipeg	103.14	42.8	21.3	5.27	115.5	2004
AC Macbeth	NI/F	1	30	Winnipeg	286.89	44.3	21.3	5.75	109.5	2004
AC Macbeth	NI/F	2	35	Winnipeg	272.22	43.8	22.4	5.8	109.5	2004
AC Macbeth	NI/F	3	40	Winnipeg	369.56	43.9	22.6	6.06	109.5	2004
AC Macbeth	NI/F	4	47	Winnipeg	266.92	44.5	20.2	5.87	106	2004
AC Macbeth	NI/NF	1	2	Winnipeg	293.60	42.5	23.4	5.5	109.5	2004
AC Macbeth	NI/NF	2	11	Winnipeg	258.68	41.3	23.7	5.22	109.5	2004
AC Macbeth	NI/NF	3	13	Winnipeg	315.56	42.8	21.9	5.42	109.5	2004
AC Macbeth	NI/NF	4	21	Winnipeg	280.16	42.0	23.2	5.37	109.5	2004
McGregor	I/F	1	54	Winnipeg	239.05	42.3	20.8	4.8	112	2004
McGregor	I/F	2	55	Winnipeg	227.48	41.2	21.3	4.41	112	2004
McGregor	I/F	3	64	Winnipeg	265.38	41.1	22.5	4.93	112	2004
McGregor	I/F	4	69	Winnipeg	282.04	41.2	21.1	4.81	115.5	2004
McGregor	I/NF	1	77	Winnipeg	130.17	38.7	24.3	4.18	115.5	2004
McGregor	I/NF	2	83	Winnipeg	107.69	38.4	24.7	4.35	122.5	2004
McGregor	I/NF	3	86	Winnipeg	100.92	38.6	23.6	4.13	119	2004
McGregor	I/NF	4	93	Winnipeg	148.55	38.1	23.6	4.11	119	2004
McGregor	NI/F	1	25	Winnipeg	291.17	42.8	21.8	5.29	106	2004
McGregor	NI/F	2	36	Winnipeg	265.49	40.5	22.2	4.81	109.5	2004
McGregor	NI/F	3	37	Winnipeg	354.2	42.8	21.3	5.37	106	2004
McGregor	NI/F	4	46	Winnipeg	248.75	42.2	21.4	5.07	106	2004
McGregor	NI/NF	1	1	Winnipeg	234.64	39.2	24.3	4.72	109.5	2004
McGregor	NI/NF	2	8	Winnipeg	234.27	39.9	24.0	4.7	109.5	2004
McGregor	NI/NF	3	16	Winnipeg	141.66	39.1	23.2	4.52	113	2004
McGregor	NI/NF	4	23	Winnipeg	155.08	39.2	23.3	4.25	109.5	2004

Appendix 6: Supporting data

Cultivar	Treatment	Rep	Plot	Location	Yield (g/m^2)	Oil content (%)	Protein content (%)	1000 kernel weight	AUDPC	Year
NorLin	I/F	1	50	Winnipeg	219.75	42.7	22.1	5.7	115.5	2004
NorLin	I/F	2	56	Winnipeg	258.33	43.1	21.6	5.94	115.5	2004
NorLin	I/F	3	65	Winnipeg	272.67	43.1	22.3	6.29	115.5	2004
NorLin	I/F	4	70	Winnipeg	243.07	42.6	21.9	6.29	115.5	2004
NorLin	I/NF	1	76	Winnipeg	162.03	40.3	24.2	5.57	141	2004
NorLin	I/NF	2	80	Winnipeg	142.17	40.8	23.7	5.25	141	2004
NorLin	I/NF	3	87	Winnipeg	103.42	40.9	22.8	5.20	141	2004
NorLin	I/NF	4	92	Winnipeg	155.73	41.2	22.8	5.17	144.5	2004
NorLin	NI/F	1	28	Winnipeg	248.64	43.2	21.7	6.07	106	2004
NorLin	NI/F	2	34	Winnipeg	313.74	43.3	21.4	6.55	109.5	2004
NorLin	NI/F	3	38	Winnipeg	316.69	42.3	21.9	6.40	109.5	2004
NorLin	NI/F	4	45	Winnipeg	319.95	43.2	22.1	6.63	109.5	2004
NorLin	NI/NF	1	6	Winnipeg	258.52	41.1	23.7	5.76	113	2004
NorLin	NI/NF	2	9	Winnipeg	231.55	40.4	24.5	5.43	113	2004
NorLin	NI/NF	3	17	Winnipeg	201.75	41.8	23.2	5.59	109.5	2004
NorLin	NI/NF	4	22	Winnipeg	184.93	41.1	22.4	5.40	109.5	2004
Vimy	I/F	1	51	Winnipeg	213.74	42.8	21.2	5.78	115.5	2004
Vimy	I/F	2	59	Winnipeg	381.75	41.9	22.7	5.78	115.5	2004
Vimy	I/F	3	62	Winnipeg	287.99	42.5	21.2	5.58	115.5	2004
Vimy	I/F	4	72	Winnipeg	253.28	43.1	20.5	6.15	115.5	2004
Vimy	I/NF	1	73	Winnipeg	116.06	40.9	24.2	5.4	126.5	2004
Vimy	I/NF	2	82	Winnipeg	185.45	41.5	22.8	5.1	133.5	2004
Vimy	I/NF	3	90	Winnipeg	129.30	41.5	22.4	5.38	115.5	2004
Vimy	I/NF	4	96	Winnipeg	174.33	42.1	22.0	5.3	115.5	2004
Vimy	NI/F	1	26	Winnipeg	331.56	42.4	21.8	6.14	109.5	2004
Vimy	NI/F	2	33	Winnipeg	334.8	43.6	20.6	6.25	109.5	2004
Vimy	NI/F	3	39	Winnipeg	440.72	43.3	20.9	6.28	109.5	2004
Vimy	NI/F	4	44	Winnipeg	395.75	43.5	20.8	6.5	109.5	2004
Vimy	NI/NF	1	4	Winnipeg	251.87	41.0	24.0	5.5	113	2004
Vimy	NI/NF	2	12	Winnipeg	271.06	40.2	24.1	5.45	109.5	2004
Vimy	NI/NF	3	14	Winnipeg	281.59	40.4	23.4	5.54	109.5	2004
Vimy	NI/NF	4	20	Winnipeg	270.58	41.9	21.3	5.6	109.5	2004