INHERITANCE OF RESISTANCE TO ASCOCHYTA BLIGHT IN LENTIL

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1989

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INHERITANCE OF RESISTANCE TO ASCOCHYTA BLIGHT IN LENTIL

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ABSTRACT

The inheritance of resistance to ascochyta blight in lentil (Lens culinaris Medik.) caused by Ascochyta fabae Speg. f. sp. lentis Gossen et al. (Syn. A. lentis Vassil.) was studied using as parents the Canadian cultivars Eston (susceptible) and Laird (moderately resistant) and two resistant lines from ICARDA, ILL-5588 and ILL-5684. The F_2, F_2-derived F_3 families and F_2-derived F_4 families of each cross were evaluated for ascochyta resistance under field conditions in an ascochyta nursery during 1987, 1988 and 1989, respectively. The parents and segregating populations were rated for ascochyta reaction on the basis of foliage symptoms, using a 1 to 9 disease rating scale, with plants rated 1 to 5 considered resistant and plants rated 7 to 9 considered susceptible. In addition percent seed-borne ascochyta infection was evaluated, using the seed plating technique.

The cultivar Eston was susceptible. Laird lentil was resistant to foliar infection by ascochyta, but its resistance breaks down in the late podding stage and under the wet conditions of the ascochyta nursery percent seed-borne ascochyta infection was even higher than in the susceptible cultivar Eston. The lines ILL-5588 and ILL-5684 were highly resistant with resistance persisting after maturity and the seed coats do not become infected and discolor materially even with prolonged exposure to wet weather at harvest.

A chi-square test for goodness-of-fit of the F_2 and F_2-derived F_3 families indicated that resistance to foliar infection by ascochyta in Laird lentil was conditioned by a single recessive gene, ral_1. Results also indicated that the resistance to foliage and seed infection by ascochyta of ILL-5588 and ILL-5684 was due to two dominant genes, Ral_2 and

Ral$_3$. ILL-5588, but not ILL-5684, also carried the ral$_1$ gene for resistance to foliar infection by ascochyta and is the better source of resistance to ascochyta. The high correlation between percent seed-borne ascochyta infection in F_2-derived F_3 families and in F_2-derived F_4 families plus the medium to high heritability estimates (0.52 to 0.81) indicate that it will be easy to select for ascochyta resistance in these crosses.

An effective method of selecting for ascochyta resistance in lentil was developed. An ascochyta nursery is developed by spreading infected lentil straw between the lentil rows prior to flowering. This nursery is then sprinkled intermittently once or twice each day until about two weeks after maturity. The crop is permitted to dry naturally and selections made for ascochyta resistant F_2 plants or replicated progeny rows in later generations. Ascochyta resistance is based on a low level of discolored seed (0 to 5%), reconfirmed by plating the seed to determine percent seed-borne ascochyta infection in replicated progeny rows. Only a few selections have a high level of clean bright seed and require seed plating. This technique is quick, easy, effective and efficient. Resulting selections are resistant to both foliar infection and seed infection by ascochyta.

In memoriam of my father, Eusebio Tay Sam and my brother Lorenzo.

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1. INTRODUCTION

Lentil (Lens culinaris Medik.) is one of the oldest pulse and has been cultivated for millenia in Western Asia, Egypt and Southern Europe. The center of origin of the cultivated lentil apparently is the Near East Arc and Asia Minor (Purseglove 1968, Westphal 1974).

Lentil production has remained relatively constant in countries such as India, Pakistan and Bangladesh, where domestic consumption approximates production. However, lentil production in Turkey quadrupled from 1974 to 1984, when over 500,000 tonnes were produced (Muehlbauer et al. 1988). This large increase in production resulted largely from replacing summerfallow with lentil in various Turkish cereal production systems. Currently Turkey is the largest lentil exporter in the world (Muehlbauer et al. 1988). Lentil production has remained relatively stable in the Americas except for Canada. Canada recently became a major producer of lentil with a production of 300,000 tonnes in 1987 (Belisle 1988). Most of this production is exported, and now Canada is the second largest lentil exporter (Belisle 1988).

Ascochyta blight, caused by Ascochyta fabae Speg. f.sp. lentis Gossen et al. [Syn. A. lentis Bond. and Vassil.] (Gossen et al. 1986), was first reported in Canada in 1978 and now is the most serious disease of lentil in western Canada (Morrall and Sheppard 1981). It is seed-borne and stubble-borne and attacks all above ground parts of the lentil plant, reducing photosynthetic area through necrotic lesions and premature defoliation. This disease can markedly reduce both yield and seed quality. In susceptible cultivars, reduction in yield and seed quality may cause loss of more than 70% of potential income (Gossen and Morrall 1983).

Chemical control by seed treatment and repeated foliar application has been developed (Morrall and Gossen 1979, Morrall 1980, Morrall and Gossen 1981, Gossen and Morrall 1983, Morrall and Beauchamp 1984, Beauchamp and Morrall 1985, Beauchamp et al. 1986a,b, France et al. 1987, Kaiser and Hannan 1987, Morrall 1987 and Russell et al. 1987). However, the most effective and economic control is by resistant cultivars. Laird lentil (Slinkard and Bhatty 1979) is the most important cultivar in Canada and is moderately resistant. Another cultivar, Estòn (Slinkard 1987), is susceptible. It is imperative that cultivars with higher levels of resistance be developed.

The objectives of this study were to (1) determine the mode of inheritance of ascochyta blight resistance in crosses between two local lentil cultivars (Laird and Eston) and ascochyta blight resistant lentil lines (ILL-5588 and ILL-5684) from The International Center for Agricultural Research in the Dry Areas (ICARDA), Aleppo, Syria and (2) develop an effective and efficient method of screening for ascochyta resistance in lentil.

2. LITERATURE REVIEW

2.1 Origin and distribution of A. fabae f. sp. lentis

According to Sattar (1933), ascochyta on lentil was firstly reported by Bondartzeva-Monteverde and Vassilievsky in Russia in 1930. They described the fungus as a new species, Ascochyta lentis Vassil. (Bondartzeva-Monteverde and Vassilievsky 1938). In India, Sattar (1933) isolated a fungus from lentil that he identified as a variety of A. pisi Lib. However, Grewal (1988) concluded that the fungus identified by Sattar was actually A. lentis Vassil. which was renamed A. fabae f. sp. lentis by Gossen et al. (1986), based on morphological similarities of isolates of A. lentis from lentil compared with isolates of A. fabae from faba bean (Vicia faba). This new name has been accepted by Cromey et al. (1987) and Kaiser et al. (1989). According to Gossen (1985), A. fabae f. sp. lentis has been reported in most lentil producing countries including Argentina (1974), Brazil (1974), Syria (1979), Greece (1980), Chile (1982) and Pakistan (1983). Cromey et al. (1987) reported this disease in New Zealand, and Bretag (1989) reported ascochyta blight as an important disease of lentil in Australia. Moreover, A. fabae f. sp. lentis has been isolated from seeds originating in Australia, Canada, Chile, Ethiopia, Greece, Hungary, India, Italy, Morocco, Pakistan, Russia, Spain, Syria, Turkey and Yugoslavia (Kaiser and Hannan 1982, Kaiser 1983, Kaiser and Hannan 1986). In Canada A. fabae f. sp. lentis was first reported in 1978, and surveys of seed samples clearly demonstrated that the disease was already widespread in Saskatchewan and Manitoba by then (Morrall and Sheppard 1981, Gossen and Morrall 1986).

2.2 Pathogen taxonomy and morphology

Bondartzeva-Monteverde and Vassilievsky (1938) separated A. fabae Speg. and A. lentis Vassil. primarily on the basis of their pathogenic specialization for faba bean and lentil, respectively. However, Gossen et al. (1984), Gossen (1985) and Gossen et al. (1986) studied the variability of 68 isolates of A. lentis from lentil for 6 cultural and 7 morphological characters and compared them to 13 isolates of A. fabae from faba bean. Multivariate analysis of various cultural and morphological characters failed to separate the two species. However, differences in host specificity were unequivocal, with isolates infecting only the host from which they were originally isolated. Therefore, they concluded that the name A. lentis was incorrect, because there were no consistent morphological differences between A. lentis and A. fabae. Furthermore, they advocated that the two groups should be synonymized under A. fabae Speg., since this name had priority, and proposed two special forms: A. fabae f. sp. fabae for isolates pathogenic on faba bean and A. fabae f. sp. lentis for isolates pathogenic on lentil. Some important physiological differences, such as host specificity, are not covered by the International Code of Botanical Nomenclature and are often indicated through the use of formae specialis (Gossen et al. 1986).

A. fabae f. sp. lentis produces small lesions on lentil and gregarious, yellow-brown, immersed pycnidia and mostly 2-celled, cylindrical conidia (Bondartzeva-Monteverde and Vassilievsky 1938, Gossen 1985). Pycnidia are globose to sub-globose; dark, ostiolate and 75 to 225 µm in diameter (Khan et al. 1983).

Sattar (1933) described A. pisi, considered as A. lentis by Grewal (1988), as having conidia with a range of 10-20 x 3.0-6.0 µm and averaging 13.1 x 3.8 µm when grown in oatmeal agar. Bondartzeva-Monteverde and Vassilievsky (1938) describe A. lentis as having conidia with a range of 11.5-19.5 x 3.5-5.8 µm when grown in oatmeal agar. Conidia from Saskatchewan isolates grown in V-8 agar averaged 14.7 x 4.1 µm (Morrall and Sheppard 1981), well within the prescribed size range.

2.3 Symptomatology

Several workers have described symptoms of the disease as it occurs in different countries (Sattar 1933, Bondartzeva-Monteverde and Vassilievsky 1938, Mitiederi 1974, Davatzi-Helena 1980, Morrall and Sheppard 1981, Sepulveda and Alvarez 1982, Khan et al. 1983, Martens et al. 1984, Kaiser and Hannan 1986, Cromey et al. 1987, France et al. 1987). All these descriptions are remarkably similar. The symptoms appear on all plant growth stages from seedling to mature plant, and all above-ground parts of the plant are attacked. Lesions on the stems and leaves are initially whitish to greyish, becoming light tan colored. Mature lesions usually have darker margins and the centres are speckled with pycnidia. The pycnidia may be scattered or in concentric circles. Coalescing lesions cause blighting and leaflet abscission. Seedlings are killed when lesions girdle the lower stem.

Lesions on pods are generally darker than those on leaves. After the pods have ripened the infected areas often have a purplish hue. Symptoms on infected seeds may range from no visible symptoms to various degrees of discoloration and shrinkage. Severely infected seeds are shrivelled and show a purplish brown discoloration, sometimes in patches and

sometimes covering the entire seed. Occasionally pycnidia and small flecks of mycelium are present on the seed surface.

A. fabae f. sp. lentis has been detected in all parts of the seed, with the cotyledons having a higher level of infection than the plumule and radicle (Morrall and Beauchamp 1988). Vishunavat et al. (1988) reported that A. rabiei in chickpea (Cicer arietinum) was detected in the seed coat and cotyledons, but not in the plumule, as reported by Maden et al. (1975).

Stunting and poor vigor are frequently associated with lentil plants that develop from seed naturally infected with A. fabae f. sp. lentis (Kaiser and Hannan 1987). Seeds severely infected with ascochyta may not germinate, especially if they are badly shrivelled and discolored (Cromey et al. 1987).

2.4 Disease development

The onset of ascochyta depends on the amount of infected plant debris, the frequency of seed transmission and weather conditions (Beauchamp 1985). Since the fungus is both seed- and stubble-borne, the primary inoculum comes from either infected seeds or infected plant debris. Stubble-borne inoculum remains highly infective after one winter and can result in rapid epidemic development early in the growing season. Hewett (1973) reported that A. fabae in faba bean spread for distances up to 10 m in an average season. However, Bond and Pope (1980) in a two-year study reported that A. fabae may spread from volunteer plants to the crop in fields at least 120 m away.

Seed transmission introduces a pathogen randomly throughout a field and provides numerous foci of primary

infection (Baker and Smith 1966). Ascochyta blight is established within a field by either random dispersal of conidia from overwintered debris to lentil plants or transmission of the pathogen from infected seed to the epicotyl (Martens et al. 1984, Gossen and Morrall 1986). Russell et al. (1987) reported that the incidence of ascochyta blight in the foliage approached 100% in a trial planted with naturally infected seed.

Infected lentil seed is the main means by which A. fabae f. sp. lentis is spread over great distances and introduced into previously disease-free areas, where it may serve as initial inoculum for disease development. Infected seed also provides the fungus with an important survival mechanism (Gossen and Morrall 1981, Kaiser 1987). Kaiser et al. (1989) reported that A. fabae f. sp. lentis survived in infected seeds stored for 4 years at temperatures of 20, 5, -18 and -160 to -196 C (liquid nitrogen).

Gossen and Morrall (1986) reported that the frequency of transmission from seed to seedlings was inversely proportional to soil temperature and directly proportional to the degree of seed discoloration. At low temperatures, the relative growth rates and vigor of the host and pathogen favored the pathogen and resulted in a higher frequency of transmission. In contrast, at higher temperatures the epicotyl grew away from the infected cotyledons before infection could occur. In plants with hypogeal germination, such as lentil and chickpea, the cotyledons remain below the soil surface and cannot function as a source of infection for foliage after emergence (Gossen 1985). Gossen (1985) and Gossen and Morrall (1986) compared the efficacy of transmission of ascochyta blight from seed and stubble-borne inoculum and found that

naturally infected plant residue was a highly effective source of inoculum, while seed-borne inoculum was less effective. According to these workers, the increased efficacy of the stubble-borne inoculum was due to the moist conditions and low temperatures during early spring which promoted rapid growth of the fungus. Under these conditions production of inoculum from plant residue at the soil surface should generally be high, and, if the conditions are appropriate for dispersal and infection, seedlings will become diseased soon after they emerge. Many seedlings can become infected by spores from a single fragment of infected residue. In contrast, seed transmission generally results in the infection of only one seedling per infected seed. Lesions of these infected seedlings must produce mature spores under conditions appropriate for dispersal and infection before the disease can spread farther.

Outbreaks of the disease in fields are associated with cooler day temperatures, extended cloudy days, and intermittent rains accompanied by winds. Gossen and Morrall (1986) conclude that the rapid spread of ascochyta blight in western Canada in the late 1970s was due to the use of infected seed by growers and favorable conditions for seed transmission and plant-to-plant spread.

2.5 Disease losses

Although ascochyta blight in lentil has been reported in most lentil producing countries, there are few quantitative data on its effect on yield and seed quality. Gossen and Morrall (1983) compared plots inoculated with A. fabae f. sp. lentis and non-inoculated plots sprayed regularly with a fungicide and observed yield losses of 25 to 40% in the

susceptible lentil cultivars Common Chilean and Eston. In Laird lentil the losses were only 8 to 13%. A 40% yield loss, associated with a 50% loss due to quality reduction, resulted in a 70% reduction in potential income for the producer. These workers indicated that levels of foliar blight, similar to those induced in these experiments, have been observed in natural epidemics.

Gossen and Morrall (1983) developed and evaluated several yield loss equations. They reported that the percentage leaf area affected at early pod set provided the best prediction of percent yield loss: percent yield loss = 47 x $\log_{10}$ % leaf area affected (r^2 = 63%).

Kaiser and Hannan (1987) studied the effect of A. fabae f. sp. lentis on growth and survival of lentil plants under field conditions and found no significant difference in the survival of healthy and infected plants, but the yield of infected plants was decreased 43%. The highest loss in yield was generally associated with wet conditions during the growing season or in the swath. Thus, yield losses caused by ascochyta blight in lentil fluctuate from year to year according to weather conditions, as reported by Beauchamp (1985).

A. fabae in faba bean may also inflict significant losses, particularly under cool wet weather conditions. Gaunt and Liew (1981) surveyed fields in New Zealand and reported that A. fabae caused yield losses approaching 40% in faba bean. They also evaluated yield losses in plots with different levels of seed infection and observed a 44% yield reduction from seed with 12% seed-borne infection, compared with seed with only 0.2% seed-borne infection. Losses apparently were associated with infection of leaves at the

reproductive nodes during flowering, which subsequently caused infection of the pods. In this respect, Morrall and Slinkard (1986) indicated that the major way in which ascochyta blight reduces yield in lentil is through peduncle lesions which cause pod abortion.

A. rabiei is one of the most destructive pathogens of chickpea with losses up to 100% (Nene 1981). In Pakistan severe epidemics of A. rabiei in recent years cut production by 50%, causing losses of US $150 million annually (ICARDA 1985).

The ascochyta complex (A. pinodes, A. pinodella, A. pisi) on pea (Pisum sativum) may decrease yield by 10 to 50% (Wallen 1974). Lawyer (1984) reported that a moderate to severe infection of A. pinodes, the causal organism of pea blight, can reduce yield by 50 to 70%.

2.6 Sources of resistance

Resistance to A. fabae f. sp. lentis has been reported by several researchers. Mitiederi (1974) in Argentina tested 509 lines of lentil under natural infection and 115 were assigned to the most resistant category, in which only a few spots developed on the leaves. She also noted that the most severe infection occurred in small seeded lines. Khatri and Singh in India (cited by Morrall and Sheppard 1981) tested 947 lines and found only five with a high level of resistance. Singh et al. (1982) evaluated 188 lines under artificial epiphytotic conditions and reported that only 18 experimental lines were consistently resistant in all four years of testing. In Russia a collection of lentil lines was assessed for reaction to ascochyta blight following artificial inoculation (Voluzneva and Golubev 1982). They found cultivar

differences in resistance and also noted that ecogeographical groups exhibited differential resistance.

According to Erskine (1984) and Summerfield and Roberts (1985), genetic differences in susceptibility to A. fabae f. sp. lentis have been observed in germplasm grown in Syria by ICARDA, and genotypes resistant to A. fabae f. sp. lentis have been identified. In recent years, ICARDA has distributed a "Lentil International Ascochyta Blight Nursery" with resistant lines available to all lentil researchers.

In Canada, Laird lentil is moderately resistant and Eston lentil is susceptible (Gossen 1985, Slinkard 1987). In New Zealand, Cromey et al. (1987) reported that Primera lentil has some resistance to A. fabae f. sp. lentis.

2.7 Inheritance of resistance to A. fabae f. sp. lentis

The mode of inheritance of resistance to A. fabae f. sp. lentis is unknown. Meiners (1981) stated that there are eight destructive diseases of lentil, but the genetics of resistance is known only for pea seed-borne mosaic virus. To date, no other genetic studies on the inheritance of disease resistance in lentil have been reported (Vandenberg 1987).

Many researchers have reported resistance to ascochyta blight in faba bean (Kharbanda and Bernier 1980, Golubev and Demina 1982, Jellis et al. 1984, Hanounik and Maliha 1984, and Tivoli et al. 1987). However, the mode of inheritance has not been studied. Recently, Hanounik and Robertson (1989) identified faba bean germplasm with broad-based general resistance and germplasm with narrow-based specific resistance to ascochyta blight.

In chickpea, resistance to A. rabiei is frequently reported as monogenic and dominant (Vir et al. 1975, Nene

1981, Singh and Reddy 1983, Tewari and Pandey 1986). Singh and Reddy (1983) also identified a recessive gene governing ascochyta resistance in the experimental line ILC-191. However, Boorsma (1980) reported that resistance to A. rabiei in chickpea was a quantitative trait rather than a qualitative trait. Also, Allen (1983) stated that resistance is partial, and the existence of immunity has not been confirmed. Furthermore, although no studies have been made on the genetics of pathogenicity in A. rabiei, evidence from the inoculation of sets of cultivars with a range of isolates suggests quantitative inheritance.

In pea resistance to foot rot, caused by A. pinodes, is due to dominant genes with epistasis (Rubes 1976). Resistance to leaf and pod spot, caused by A. pisi, is due to a single dominant gene (Wark 1950, Darby and Lewis 1985). Similarly, resistance to ascochyta blight of pea, caused by A. pinodella, is also due to a single dominant gene (Rastogi and Saini 1984).

No reports have been published on physiological specialization in A. fabae f. sp. lentis. Gossen (1985) studied the cultural and morphological variability of A. fabae f. sp. lentis and A. fabae f. sp. fabae utilizing several isolates of A. lentis from different countries, but found no differences in pathogenicity among isolates of A. fabae f. sp. lentis on lentil seedlings.

Singh and Reddy (1983) reported numerous races of A. rabiei on chickpea, and suggested breeding for resistance to specific races. They reported that resistance to race 3, probably the most common race in Syria, was due to a single dominant gene. Similarly, different races of ascochyta have

been reported on faba bean in various regions of the world (Rashid and Bernier 1985, Hanounik and Robertson 1989).

Hafiz (1952) studied morphological and physiological characteristics of chickpea cultivars that were susceptible and resistant to A. rabiei. He found denser pubescence on the stems and leaves of the resistant types, resulting in greater secretion of malic acid from the glandular hairs during the later growth stages. This high concentration of malic acid was detrimental to spore germination and germ tube growth. In addition, Allen (1983) reported a strong correlation between seedcoat color and ascochyta resistance. Resistant genotypes were predominantly black seeded (Kaiser 1972), and Singh et al. (1981) reported that no resistant kabuli cultivar (light colored seed coat) has been released for commercial production in West Asia and North Africa.

2.8 Control of A. fabae f. sp. lentis

Ascochyta blight of chickpea can be controlled by (a) utilizing host resistance, (b) adopting cultural practices including sanitation, or (c) using chemicals to treat seed and foliage (Nene 1981). These control methods have also been used to control ascochyta blight of lentil. Russell et al. (1987) emphasized the need for an integrated approach to the control of A. fabae f. sp. lentis, involving crop rotation to reduce spread from infected crop debris, use of clean seed or seed with a low level of infection, seed treatment and foliar fungicides.

2.8.1 Cultural control

Cultural practices frequently can be modified to help suppress the rate of epidemic development (Frey 1977). In Canada, Morrall and Sheppard (1981) recommend crop rotation, early seeding to escape moist weather at harvest and the use of disease-free seed. In Chile, where lentil is a winter crop, planting early in April is not recommended because ascochyta attack is more likely than when planting between May 15 and June 15 (Tay et al. 1987).

Because infected lentil plants are a highly effective source of primary inoculum, crop rotation should limit the chance of crops being infected from diseased volunteer seedlings and/or contaminated debris from the previous year (Beauchamp 1985, Gossen 1985). Deep plowing of lentil debris is also recommended to control the spread of A. fabae f. sp. lentis (Davatzi-Helena 1980, Kaiser 1987). High temperature and humidity increase the rate of stubble decomposition and adversely affect survival of A. rabiei, especially when the stubble is buried (Kaiser 1973).

Since ascochyta blight is seed-borne, it is obvious that the use of clean seed will be beneficial. However, even seed with no visible symptoms can be infected and subsequently infect the lentil crop. Therefore, seed for planting should be tested for the presence of ascochyta and only seed testing zero or very low should be planted (Morrall and Sheppard 1981, Morrall 1986). Agar plate tests indicate the incidence of seed-borne ascochyta. There is no absolute tolerance level for seed infection since in a wet year seed with only 1 to 2% infection may result in a heavily infected crop and in a dry year even seed with as high as 20% infection may result in little disease (Morrall 1986). According to Gabrielson

(1988), two environmental factors, temperature and moisture, profoundly affect inoculum threshold levels. What may become a disastrous epidemic in one area may be insignificant in another area with different environmental conditions.

In Canada, Sheppard (1987) proposed a tolerance of 1% infected seed in Breeder seed, 1.5% infected seed in Foundation and Registered seed, and 2% infected seed in Certified seed of lentil. In faba bean he proposed zero percent infected seed in Breeder seed, 0.2% infected seed in Foundation and Registered seed, and 0.75% infected seed in Certified seed. In pea he proposed a tolerance of 1% infected seeds for A. pisi and 10% infected seed for A. pinodes and A. pinodella in Certified seed.

Hewett (1973) reported that in England seed lots of faba bean with approximately 1% infected seeds seem suitable for production, but little or no A. fabae can be tolerated in seed intended for multiplication. He stated that infection in British-grown commercial seed has been greatly reduced by selection of clean seed.

Conditions after pod set are important in determining percent seed-borne ascochyta in lentil. Premature harvest (10 days before normal cutting) substantially reduced the frequency of seed infection and discoloration (Gossen 1985). Unfortunately, it also decreased seed yield and seed grade and, therefore, is not an economic control option. In general, reducing exposure of the crop to the environment results in a higher seed grade and a lower frequency of seed infection than prolonged exposure because of the reduced probability of exposure to weather conducive to spread of the pathogen (Beauchamp 1985). Thus, the use of desiccants and direct combining, practices that shorten the period of

exposure to adverse environmental conditions, may be a viable alternative to the standard procedure of allowing the swathed crop to dry in the field (Gossen and Morrall 1984, Gossen 1985).

2.8.2 Chemical control

According to Frey (1977), chemical control becomes essential when crop management, host resistance, alterations of environment or alterations of the associated biota are inadequate to suppress the pathogen sufficiently. In this respect Morrall (1986) concluded that Canadian lentil producers need a method of ascochyta control which can be applied in mid-season after infection has been confirmed and which will be effective when the weather is favorable for disease spread.

2.8.2.1 Fungicide seed treatments

Seed treatment with effective fungicides can reduce or eliminate the initial inoculum and prevent spread of seed-borne diseases into new areas. Morrall and Gossen (1979), Morrall (1980), Morrall and Gossen (1981), Morrall and Beauchamp (1984), Beauchamp and Morrall (1985) and Morrall (1988) evaluated several seed-treatment fungicides including benomyl, carbendazim, carbathiin, iprodione and thiabendazole for control of seed-borne A. fabae f. sp. lentis under field conditions at several locations. They found that thiabendazole was the most effective in reducing seed-to-seedling transmission of the fungus. France et al. (1987) and Russell et al. (1987) also reported the best control of ascochyta with a thiabendazole seed treatment, resulting in increased germination and decreased transmission of the

disease by infected seeds. In Australia Bretag (1989) reported the best control of ascochyta with a fungicide containing thiabendazole, thiram and metalaxyl. This treatment also improved plant establishment. Kaiser and Hannan (1987) evaluated 12 seed treatment fungicides, including carboxiin, captan, thiram, benomyl and thiabendazole on naturally infected lentil seeds in laboratory studies and under field conditions. Their results indicated that seed treatment of seed-borne infection of A. fabae f. sp. lentis with thiabendazole or benomyl resulted in significantly greater field emergence than with the other treatments and yields were significantly higher with thiabendazole. However, these workers reported that thiabendazole was not 100% effective in eradicating the pathogen from the cotyledons. Reddy (cited by Haware et al. 1986) reported that thiabendazole at 3 g/kg seed completely eradicated seed-borne inoculum of A. rabiei from chickpea seed and there was no adverse effect on germination. Maude and Kyle (1970) reported that benomyl successfully eradicated A. pisi Lib. from pea seed.

2.8.2.2 Foliar-applied fungicides

Spraying a foliar fungicide should reduce or prevent disease progress for a period of time and consequently reduce the area under the disease progress curve (Morrall 1986). To date, foliar-applied fungicides for control of ascochyta blight in lentil have been studied in Canada only by Gossen and Morrall (1983), Beauchamp (1985) and Beauchamp et al. (1986a,b). Based on lab tests of 11 fungicides, chlorothalonil, captafol, folpet, metiram, mancozeb and benomyl were selected and evaluated under field conditions

using a single foliar application at early bloom to early pod set on the susceptible common Chilean lentil. Results indicated that systemic fungicides were ineffective in increasing seed yield, whereas protectant fungicides were more effective. Captafol (1.2 kg ai/ha) and chlorothalonil (1.7 kg ai/ha) gave the best protection, increased seed yield and reduced the level of seed-borne infection.

Beauchamp et al. (1986b) tested different spraying schedules for three years using eight combinations of three dates. The fungicides used were benomyl, captafol and chlorothalonil, and spraying started at the early bloom stage, using common Chilean, Eston and Laird lentil. Results were dependent on the weather conditions and indicated no yield advantage from spraying during bloom if dry weather restricted disease progress later in the season. On the other hand, when weather conditions were favorable for disease spread, date of the first application was critical. Spraying at the early bloom stage increased yield and reduced percent seed-borne infection, resulting in an economic response to spraying. However, further study is needed to clarify the potential of foliar fungicides in management of ascochyta blight, especially the timing of spraying and the effect of lower rates. Thus, in certain instances it is cost effective to use a single, relatively inexpensive application as part of an integrated program, relying primarily on rotation and high quality seed to keep inoculum levels to a minimum.

2.8.3 Genetic resistance

The most efficient and economical control of plant disease is through the use of resistant cultivars. However, lentil cultivars highly resistant to ascochyta blight are not available in Canada yet. Laird lentil is moderately resistant to foliar infection, but becomes susceptible at the late podding stage (Morrall and Sheppard 1981, Martens et al. 1984).

Line 89S26013 has recently been released in Syria and Lebanon as a commercial cultivar resistant to both vascular wilt, caused by Fusarium oxysporum f. sp. lentis and ascochyta blight (Erskine et al. 1988). Line ILL-5588 is a single plant selection from line 78S26013 and is highly resistant to Ascochyta fabae f. sp. lentis in Saskatchewan and elsewhere.

3. MATERIALS AND METHODS

3.1 General

In 1986 crosses were made among Laird lentil, moderately resistant to ascochyta blight (Gossen 1985); Eston lentil, susceptible (Slinkard 1987); and two putative ascochyta resistant lentil lines from ICARDA, ILL-5588 and ILL-5684. The F_1 plants were grown in an hydroponics system in growth rooms (Vandenberg and Slinkard 1986).

During 1987, 1988 and 1989 F_2, F_2-derived F_3 and F_2-derived F_4 families, respectively, and parents were evaluated as spaced plants in an ascochyta nursery located at the North Seed Farm, Saskatoon. An artificial epiphytotic was created each year by scattering heavily infected crop debris from the previous season between 4 m rows spaced 60 cm apart and lightly sprinkling several times daily starting about two weeks prior to the first bloom stage.

The segregating populations and parents were rated for ascochyta resistance on the basis of foliage symptoms, using the ICARDA scale (ICARDA 1989) for rating ascochyta diseases, where:

1 = no lesions,
3 = few scattered lesions seen after careful searching,
5 = lesions common and easily observed, but little defoliation,
7 = lesions very common and damaging,
9 = lesions extensive, many plants killed.

Planted rated 1 to 5 were considered resistant, whereas those rated 7 to 9 were considered susceptible. The chi-squared test was used to test the goodness-of-fit to different genetic ratios.

As reported earlier, the ascochyta resistance in Laird lentil breaks down as the plant senesces and wet weather at harvest time results in seed infection and in severe cases the seed coats become badly discolored, similar to the susceptible cultivars (Morrall 1986). However, in 1988 it was noted that the ascochyta resistance of the ICARDA lentil lines persisted after maturity and this was reconfirmed in 1989.

3.2 1987 Research

Five F_2 populations of lentil (Eston x Laird, Laird x ILL-5588, Eston x ILL-5588, Laird x ILL-5684, and Eston x ILL-5684) plus Brewer (Muehlbauer 1982), PI 339318 and the parent lines were grown as spaced plants in rows 60 cm apart at the North Seed Farm. Severity of foliar infection was rated on an individual plant basis when the plants were 62 to 75 days old. About 300 plants of each parent and the other pure lines were rated for foliar infection plus all F_2 plants. Percent infected seeds was assessed as percent visibly spotted and damaged seeds, because individual F_2 plants did not produce enough seed so that some could be sacrificed for determination of seed-borne ascochyta.

3.3 1988 Research

The F_2-derived F_3 lentil lines and their parents were planted in single rows 4 m long and 0.6 m apart (25 seeds per row) in a randomized complete block design with two replications. Each cross was grown in a separate experiment. Severity of foliar infection was rated on an individual plant basis within each row when the plants were 87 to 91 days old and averaged.

Percent seed-borne infection of ascochyta was evaluated, using the seed plating technique (Gossen and Morrall 1983, Morrall and Beauchamp 1988). All plants in the row were harvested in bulk. Fifty seeds per replication were plated on V8 juice agar (Morrall and Beauchamp 1988) in petri dishes according to the following procedure:

1. A sample of seeds was washed in a strainer under tap water to remove trash.
2. Seeds were surface sterilized in 0.6% NaOCl for 10 min.
3. After draining, the seeds were placed in sterile petri dishes containing filter paper to dry.
4. The seeds were plated on 20% V8 juice agar, 7 to 8 seeds per petri dish, incubated on a laboratory bench for 9 to 11 days and scored by percent ascochyta colonies. The laboratory was illuminated with cool white fluorescent lamps during a 12 hr photoperiod.

In addition, a trial was planted to determine if the resistance of the parental lines persisted after maturity, ILL-5588, ILL-5684, Laird and Eston lentil were planted in a randomized complete block design with four replications. Each plot consisted of four rows, 4 m long, and 30 cm apart. One row of each plot was harvested each week, starting August 19 when Laird lentil was beginning to mature and the other cultivars and lines were fully senesced. Percent seed-borne infection was evaluated as above. In addition, percent discolored seed was recorded for each sample.

In 1988, due to the heat and drought which delayed disease development, the ascochyta nursery was also sprayed with a spore suspension of A. fabae f. sp. lentis in late

July. The inoculum was prepared from cultures of ascochyta isolates from Saskatchewan grown on V8 juice agar. Conidia were washed from cultures with distilled water, and the conidial suspension was diluted to 100,000 conidia/mL, based on counts of conidia in a hemacytometer. The inoculum was applied to the foliage of 78-day old plants with a knapsack sprayer. The isolates of A. fabae f. sp. lentis were obtained from Prof. R.A.A. Morrall, Department of Biology, University of Saskatchewan.

3.4 1989 Research

In 1989, all F_2-derived F_4 lines of lentil were regrown to reconfirm F_3 disease data, each cross was grown in a separate experiment. A wide range of seed-borne ascochyta infection was present in the F_3, ranging from near zero to over 80%. The F_2-derived F_4 lines and their parents were planted in single rows 4 m long and 0.6 m apart. Twenty-five seeds were planted in each row in a randomized complete block design with two replications. Foliar infection by ascochyta was rated on an individual plant basis within each row and averaged. Percent seed-borne infection was also evaluated as before by plating a seed sample from each row on Difco-Bacto potato dextrose agar (PDA) (Morrall and Beauchamp 1988).

4. RESULTS AND DISCUSSION

4.1 Summer 1987

4.1.1 Rating of foliar infection by ascochyta in lentil cultivars and lines

The use of heavily infected crop debris and frequent light sprinkling of the foliage during the growing season resulted in a severe foliar infection by ascochyta at the North Seed Farm. This is demonstrated by the rating of 7 for the susceptible cultivars Eston and Brewer lentil (Table 1). In addition, Eston and Brewer lentil had about 40% visibly spotted and damaged seed. PI 339318 lentil had red pods and was included with the thought that the phenolic compounds in the pod wall might inhibit ascochyta infection of the seed through the pod wall. However, PI 339318 lentil was given an ascochyta rating of 9, the most susceptible entry, and the percent visibly spotted and damaged seed was over 70%, nearly twice that of the other susceptible entries. Laird, ILL-5588 and ILL-5584 lentil were given an ascochyta rating of 3 (resistant) and the latter two lines had a low percent visibly spotted and damaged seed. However, Laird lentil had a medium high percent visibly spotted and damaged seed (17.6%).

Table 1. Reaction to foliar infection by ascochyta and percent spotted and damaged seed of lentil cultivars and lines at the North Seed Farm, 1987

Cultivar/ line	Origin	Seed size	Matu- rity	Ascochyta rating on foliage	Percent spotted & damaged seed[1]	Percent clean seed
ILL-5588	ICARDA	Small	Early	3	2.0	98.0
ILL-5684	ICARDA	Small	Early	3	3.1	96.9
Laird	Canada	Large	Late	3	17.6	82.4
Eston	Canada	Small	Early	7	38.3	61.7
Brewer	USA	Large	Early	7	45.0	55.0
PI 339318	USDA	Small	Early	9	73.8	26.2

[1]Based on bulk seed of 5 plants.

4.1.2 Rating of foliar infection by ascochyta in F_2 populations of lentil

Five F_2 population were segregating for resistance (R) and susceptibility (S) to foliar infection by ascochyta and chi-square tests were made for goodness-of-fit to various ratios. The 1R:3S ratio in the F_2 of Eston x Laird (Table 2) suggests that the ascochyta resistance in Laird lentil is conditioned by a single recessive gene (ral$_1$ ral$_1$ in Table 3). Various one, two and three gene ratios were tested, but the genetic explanation in Table 2 is the only one that came close to explaining all results. If data had been available from the cross ILL-5588 x ILL-5684, perhaps the genetic hypothesis would have been better substantiated. Eston is assumed to carry no genes for resistance, but is more resistant than PI 339318 (Table 1). The F_2 ratio in the cross Eston x ILL-5684 provides a poor fit to a 15R:1S ratio due to an excess of susceptible plants, possibly the result of misclassification (Table 2). However, if the 15R:1S ratio is correct, this suggests that the ascochyta resistance in ILL-5684 is

conditioned by two dominant genes (Table 3). The 15R:1S ratio in the F_2 of Laird x ILL-5588 (Table 2) suggests that these two lines differ by two dominant genes conditioning ascochyta resistance (Table 3). The 61R:3S ratio in the F_2 of Laird x ILL-5684 (Table 2) suggests that these two lines are segregating for a recessive gene and two dominant genes conditioning ascochyta resistance (Table 3). Likewise, the 61R:3S F_2 ratio in the F_2 of Eston x ILL-5588 (Table 2) suggests that these two lines are segregating for a recessive gene and two dominant genes conditioning ascochyta resistance (Table 3). However, these F_2 populations are too small to provide conclusive evidence. Therefore, the progenies of these crosses were reevaluated as F_2-derived F_3 families in 1988.

Table 2. Reaction to foliar infection by ascochyta in F_2 populations of lentil at the North Seed Farm, 1987

F_2 population	Number of F_2 plants rated			Expected ratio	X^2	Probability
	R[1]	S	Total			
Eston x Laird	45	129	174	1:3	0.07	0.75-0.90
Eston x ILL-5684	60	10	70	15:1	7.71	<0.01
Laird x ILL-5588	35	2	37	15:1	0.04	0.80-0.90
Laird x ILL-5684	33	0	33	61:3	0.08	0.70-0.80
Eston x ILL-5588	29	2	31	61:3	0.22	0.50-0.75

[1]R=resistant, S=susceptible.

The proposed genotypes for ascochyta reaction of each parental line are shown in Table 3. The F_2 genotypes, frequencies and reaction to foliar infection by ascochyta, based on the proposed parental genotypes, are shown in Table 4. Table 2, 3 and 4 agree except for the poor fit to the

15R:1S ratio in the F_2 Eston x ILL-5684 (Table 2). As noted earlier, this was due to an excess of susceptible plants, possibly the result of misclassification.

Table 3. Proposed genotypes for reaction to foliar infection by ascochyta for the four parental lines of lentil

Parental line	Genotype			Ascochyta reaction[1]
Eston	Ral_1 Ral_1	ral_2 ral_2	ral_3 ral_3	S
Laird	ral_1 ral_1	ral_2 ral_2	ral_3 ral_3	MR
ILL-5588	ral_1 ral_1	Ral_2 Ral_2	Ral_3 Ral_3	R
ILL-5684	Ral_1 Ral_1	Ral_2 Ral_2	Ral_3 Ral_3	R

[1]S=susceptible, MR=moderately resistant but breaks down in pod stage, R=resistant.

Note: Genes for ascochyta blight resistance in the parental lines are:
Eston - 0 gene,
Laird - $\underline{ral}_1$ = 1 recessive gene,
ILL-5684 - $\underline{Ral}_2$ $\underline{Ral}_3$ = 2 dominant genes,
ILL-5588 - $\underline{ral}_1$ $\underline{Ral}_2$ $\underline{Ral}_3$ = 3 genes (one recessive and two dominant).

Table 4. Proposed genotypes, frequencies and reaction to foliar infection by ascochyta for F_2 plants of lentil from the five F_2 populations

F_2 population	Genotype	Frequency	Ascochyta reaction[1]
Eston x Laird	ral_1 ral_1 ral_2 ral_2 ral_3 ral_3	1	MR
	Ral_1 ---- ral_2 ral_2 ral_3 ral_3	3	S
Eston x ILL-5684	Ral_1 Ral_1 Ral_2 ---- Ral_3 ----	9	R
	Ral_1 Ral_1 ral_2 ral_2 Ral_3 ----	3	R
	Ral_1 Ral_1 Ral_2 ---- ral_3 ral_3	3	R
	Ral_1 Ral_1 ral_2 ral_2 ral_3 ral_3	1	S
Laird x ILL-5588	ral_1 ral_1 Ral_2 ---- Ral_3 ----	9	R
	ral_1 ral_1 ral_2 ral_2 Ral_3 ----	3	R
	ral_1 ral_1 Ral_2 ---- ral_3 ral_3	3	R
	ral_1 ral_1 ral_2 ral_2 ral_3 ral_3	1	MR-S?
Laird x ILL-5684	Ral_1 ---- Ral_2 ---- ral_3 ral_3	27	R
	Ral_1 ---- Ral_2 ---- ral_3 ral_3	9	R
	Ral_1 ---- ral_2 ral_2 Ral_3 ----	9	R
	ral_1 ral_1 Ral_2 ---- Ral_3 ----	9	R
	Ral_1 ---- ral_2 ral_2 ral_3 ral_3	3	S
	ral_1 ral_1 Ral_2 ---- ral_3 ral_3	3	R
	ral_1 ral_1 ral_2 ral_2 Ral_3 ----	3	R
	ral_1 ral_1 ral_2 ral_2 ral_3 ral_3	1	MR
Eston x ILL-5588	Ral_1 ---- Ral_2 ---- Ral_3 ----	27	R
	Ral_1 ---- Ral_2 ---- ral_3 ral_3	9	R
	Ral_1 ---- ral_2 ral_2 Ral_3 ----	9	R
	ral_1 ral_1 Ral_2 ---- Ral_3 ----	9	R
	Ral_1 ---- ral_2 ral_2 ral_3 ral_3	3	S
	ral_1 ral_1 Ral_2 ---- ral_3 ral_3	3	R
	ral_1 ral_1 ral_2 ral_2 Ral_3 ----	3	R
	ral_1 ral_1 ral_2 ral_2 ral_3 ral_3	1	MR

[1]MR=moderately resistant, R=resistant, S=susceptible.

Some plants in the F_2 of Laird x ILL-5684 exhibited transgressive segregation, i.e., a higher level of ascochyta resistance than either parent (Table 5), indicating that each parent carried different genes for ascochyta resistance. Transgressive segregation was also indicated for the other

three F_2 populations involving the two lines from ICARDA (Table 5), but was not expected according to the genotypes proposed in Tables 3 and 4. Again this discrepancy may be the result of misclassification or disease escape or the presence of a minor gene for resistance in Eston lentil.

Table 5. Transgressive segregation for resistance to foliar infection by ascochyta in parental and F_2 populations of lentil at the North Seed Farm, 1987

Parent or F_2 population	Ascochyta rating	
	1 to 5	7 to 9
Eston	0	All ($\bar{x}$ = 7)
Laird	All ($\bar{x}$ = 3)	0
ILL-5588	All ($\bar{x}$ = 3)	0
ILL-5684	All ($\bar{x}$ = 3)	0
Eston x ILL-5588, F_2	29[1]	2
Eston x ILL-5684, F_2	60[1]	10
Laird x ILL-5588, F_2	35[1]	2
Laird x ILL-5684, F_2	33[1]	0

[1]Includes plants more resistant than either parent.

4.1.3 Percent visibly spotted and damaged seed in the F_2 populations

Individual F_2 plants of lentil did not produce enough seed so that some could be sacrificed for determination of percent seed-borne ascochyta. Accordingly, seed infection by ascochyta was assessed on an individual F_2 plant basis as percent visibly spotted and damaged seed. This is a realistic approach since lentil grades are determined on the basis of discolored and damaged seed. If most of the clear, bright seed are free of seed-borne ascochyta, then it may be possible to screen for ascochyta resistance by selecting plants with clear, bright seed under conditions of severe ascochyta infection. Results in Table 1 suggest a positive correlation between rating of foliar infection by ascochyta and the

frequency of visibly spotted and damaged seed in the parents, i.e., a positive correlation between level of ascochyta resistance and percent clean, bright seed. The type of resistance present in ILL-5588 and ILL-5684 lentil resulted in the highest frequency of clean bright seed. This positive relationship between level of ascochyta resistance and percent clean bright seed was also evident in the F_2 populations (Table 6). Thus, the F_2 population with the highest proportion of susceptible plants (Eston x Laird, Table 2) also had the highest frequency of individual plants with more than 10% visibly spotted and damaged seeds (56.9%, Table 6). Subsequently, this cross was eliminated from further consideration. These results also suggest that the resistance to foliar infection by ascochyta in the parent lines ILL-5588 and ILL-5684 (Table 1) also conditioned resistance to seed infection. This relationship would also be expected in at least some of the more resistant F_2 progeny from crosses involving these two lines. The high frequency of F_2 plants with less than 2% visibly spotted and damaged seed in Table 6 again suggests transgressive segregation when compared with the parents in Table 1. It is also possible that some additional modifier genes for ascochyta resistance are segregating in these crosses.

Table 6. Percent frequency of F_2 plants with differing percent visibly spotted and damaged seed in the F_2 populations of lentil at the North Seed Farm, 1987

F_2 population	No. of plants	Percent visibly spotted and damaged seed/plant				
		0	1-2	2-3.5	3.5-10	>10
Eston x Laird	174	1.2	5.2	6.0	28.7	56.9
Eston x ILL-5684	70	1.4	11.4	7.1	28.6	51.4
Eston x ILL-5588	31	22.6	3.2	12.9	32.3	29.0
Laird x ILL-5588	37	27.0	16.2	21.6	16.2	18.9
Laird x ILL-5684	33	15.2	27.3	9.1	30.3	18.2

4.2 Summer 1988

4.2.1 Rating of foliar infection by ascochyta in F_2-derived F_3 families

The F_2-derived F_3 families of Laird x ILL-5588 gave a good fit to a 7 homozygous resistant: 8 segregating: 1 homozygous susceptible ratio (Table 7), confirming the 15R:1S F_2 ratio (Table 2) and the presence of two dominant genes segregating for ascochyta resistance in this cross (Table 4). The F_2-derived F_3 families of Laird x ILL-5684 gave a good fit to a 37 homozygous resistant: 26 segregating: 1 homozygous susceptible ratio (Table 7), confirming the 61R:3S F_2 ratio (Table 2) and the presence of one recessive and two dominant genes segregating for ascochyta resistance in this cross (Table 4). Similarly, the F_2-derived F_3 families of Eston x ILL-5588 gave a good fit to a 37 homozygous resistant: 26 segregating: 1 homozygous susceptible ratio (Table 7), confirming the 61R:3S F_2 ratio (Table 2) and the presence of one recessive and two dominant genes segregating for ascochyta resistance in this cross (Table 4).

The F_2-derived F_3 families of Eston x ILL-5684 deviated significantly from the expected 7:8:1 segregation (Table 7), based on segregation two dominant genes for ascochyta

resistance (Tables 2 and 4). This deviation was due to an excess of homozygous resistant F_2-derived F_3 families. In contrast, the poor fit to the expected 15R:1S ratio in F_2 was due to an excess of susceptible plants (Table 2). However, if the homozygous resistant and segregating F_3 families were combined into a single class, the F_3 data gave a good fit to a 15:1 ratio (X_2 = 0.00, P = 1.00), giving a measure of confidence in the genetic explanation of these results.

Table 7. Reaction to ascochyta blight of F_2-derived F_3 families of lentil at the North Seed Farm, 1988

	No. of F_2-derived F_3 families				X^2		
F_3 population	R[1]	Seg.	S	Total	7:8:1	37:26:1	P value
Laird x ILL-5588	21	14	1	36	3.32	----	0.10-0.05
Laird x ILL-5684	14	12	2	28	----	5.91	0.10-0.05
Eston x ILL-5588	24	6	0	30	----	5.60	0.10-0.05
Eston x ILL-5684	47	11	4	62	27.46	----	<0.001

[1]R=resistant, S=susceptible, Seg.=segregating.

4.2.2 Percent seed-borne ascochyta infection of F_2-derived F_3 families

The percent seed-borne ascochyta infection for each F_2-derived F_3 family in the four crosses, along with the corresponding ascochyta rating on the foliage, is presented in Tables 8 to 11. The concordance between these two measures of ascochyta resistance is clearly evident in all four crosses (Tables 8 to 11) and is especially marked at the higher levels of resistance. This suggests that initial selection for ascochyta resistance in these populations can be readily done by selecting for plants (families) with a high level of clean,

bright seed when the plants are grown under conditions of severe ascochyta infection.

Transgressive segregation occurred for level of seed-borne ascochyta infection in all four crosses (Tables 8 to 11), since some F_2-derived F_3 families in each cross were significantly lower in percent seed-borne ascochyta infection than the resistant parent. This suggests that both parents (even the highly susceptible Eston) contributed a gene that enhanced the level of seed-borne ascochyta. Alternatively, some seeds of the more resistant F_2-derived F_3 families may have escaped the ascochyta infection, possibly due to development as the crop was drying down. In susceptible plants the pods may have been infected, but the seeds could have escaped infection as the crop dried.

In the cross Laird x ILL-5588 lentil (Table 8) seed-borne ascochyta infection of the F_2-derived F_3 families ranged from 1.4 to 67.5% and averaged 15.5%. The resistant parent ILL-5588 lentil had 5.8% seed-borne ascochyta infection, while the other resistant parent Laird lentil had 52.5% seed-borne ascochyta. Laird lentil was rated 5 (resistant) based on foliage symptoms, but it had a high percent seed-borne ascochyta infection. This high seed-borne ascochyta infection occurred primarily during the final stages of maturity of the plants, and was not evident when the foliar ascochyta infection was rated. This high percent seed infection is produced by rapid saprophytic growth of the fungus after the pods are ripe in the presence of free water as in the ascochyta nursery. Laird lentil is particularly susceptible to a rapid increase in level of seed-borne ascochyta infection at this stage (Martens et al. 1984, Morrall 1986). Gossen (1985) in a study of the epidemiology and yield losses caused

by ascochyta in 1980, also found no relationship between foliar blight and seed-borne infection in Laird lentil, even though it had significantly lower foliar infection levels than Eston and Common Chilean lentil. He concluded that this was partly due to the later harvest date for Laird with weather conditions favourable for disease development, especially during the later stages of pod filling.

In the cross Laird x ILL-5684 lentil (Table 9) seed-borne ascochyta infection of the F_2-derived F_3 families ranged from 3.3 to 56.8% and averaged 23.8%. The resistant parent ILL-5684 lentil had 10.9% seed-borne ascochyta infection, while the other resistant parent Laird lentil had 56.8% seed-borne ascochyta. As before, the resistance to foliar infection by ascochyta of Laird was rendered ineffective as the pods matured and senesced.

In the cross Eston x ILL-5588 (Table 10) seed-borne ascochyta infection of the F_2-derived F_3 families ranged from 1.8 to 22.6% and averaged 9.1%. The resistant parent ILL-5588 lentil had 4.5% seed-borne ascochyta, while the susceptible parent Eston lentil had 21.8% seed-borne ascochyta.

In the cross Eston x ILL-5684 lentil (Table 11) seed-borne ascochyta infection of the F_2-derived F_3 families ranged from 1.2 to 85.1% and averaged 21.3%. The resistant parent ILL-5684 lentil had 11.4% seed-borne ascochyta, while the susceptible parent Eston lentil had 21.6% seed-borne ascochyta. This cross included three families with very high levels of seed-borne ascochyta infection (over 60%) and practically all seeds were badly discolored.

Table 8. Reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_3 families of Laird x ILL-5588 lentil at the North Seed Farm, 1988

Family number	Reaction to foliar infection[1]	Percent seed-borne ascochyta infection[2]
22	R	1.4
10	R	2.0
7	R	2.6
3	R	2.9
29	R	3.7
27	R	4.4
20	R	4.8
34	R	5.5
1	R	5.5
ILL-5588	R	5.8
30	R	6.0
8	R	6.7
14	R	7.3
4	R	7.6
28	R	7.6
32	R	8.7
16	R	9.9
36	R	10.9
26	R	11.1
35	R	11.7
37	R	12.3
9	R	14.3
33	Seg.	17.3
15	Seg.	18.1
21	Seg.	18.2
11	Seg.	19.6
5	Seg.	21.0
17	Seg.	22.9
18	Seg.	23.1
33	Seg.	23.7
12	Seg.	28.3
24	Seg.	29.3
6	Seg.	29.3
25	Seg.	29.5
13	Seg.	38.5
19	Seg.	38.7
Laird	MR	52.7
23	S	67.5
Mean		15.9
SD		0.2
CV (%)		9.3
LSD ($P \leq 0.05$)		1.9

[1]R=resistant, MR=moderately resistant, Seg.=segregating, S=susceptible.

[2]Based on plating 100 seeds on 20% V8 juice agar, mean of two replications.

Table 9. Reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_3 families of Laird x ILL-5684 lentil at the North Seed Farm, 1988

Family number	Reaction to foliar infection[1]	Percent seed-borne ascochyta infection[2]
23	R	3.3
3	R	7.7
13	R	8.4
10	R	9.7
ILL-5684	R	10.9
28	R	11.3
16	R	14.6
5	R	16.8
15	R	17.5
29	Seg.	17.5
8	Seg.	17.7
30	R	18.4
33	Seg.	19.3
11	Seg.	20.1
24	R	22.8
2	Seg.	24.3
12	Seg.	27.1
22	Seg.	27.2
9	Seg.	28.2
18	R	29.2
19	R	30.2
14	R	30.5
1	Seg.	30.7
31	R	31.9
27	Seg.	36.0
20	Seg.	36.5
17	Seg.	39.2
21	S	43.0
6	S	46.2
Laird	MR	56.8
Mean		23.8
SD		1.3
CV (%)		15.9
LSD ($P \leq 0.05$)		5.6

[1] R=resistant, MR=moderately resistant, Seg.=segregating, S=susceptible.

[2] Based on plating 100 seeds on 20% V8 juice agar, mean of two replications.

Table 10. Reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_3 families of Eston x ILL-5588 lentil at the North Seed Farm, 1988

Family number	Reaction to foliar infection[1]	Percent seed-borne ascochyta infection[2]
15	R	1.8
9	R	1.9
6	R	3.6
5	R	4.0
ILL-5588	R	4.5
7	R	4.8
10	R	5.1
26	R	5.4
8	R	5.6
32	R	5.6
19	R	6.2
22	R	6.3
18	R	6.4
20	R	6.7
34	R	6.7
17	R	6.9
25	R	7.0
24	Seg.	7.7
2	R	7.9
4	R	8.2
13	R	9.6
27	R	11.1
12	Seg.	11.2
3	R	13.2
28	R	13.5
11	R	14.6
16	Seg.	15.1
21	R	15.9
23	Seg.	18.0
38	Seg.	20.1
Eston	S	21.8
31	Seg.	22.6
Mean		9.1
SD		0.5
CV (%)		29.4
LSD ($P \leq 0.05$)		3.9

[1] R=resistant, Seg.=segregating, S=susceptible.
[2] Based on plating 100 seeds on 20% V8 juice agar, mean of two replications.

Table 11. Reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_3 families of Eston x ILL-5684 lentil at the North Seed Farm, 1988

Family number	Reaction to foliar infection[1]	Percent seed-borne ascochyta infection[2]
54	R	1.2
25	R	3.8
65	R	4.6
32	R	5.9
27	R	6.8
21	R	7.1
56	R	7.1
70	R	9.6
67	R	9.7
42	R	10.1
69	Seg.	10.6
37	R	11.1
62	R	11.1
ILL-5684	R	11.4
9	R	11.7
30	R	12.2
38	Seg.	12.7
7	R	13.2
61	R	13.3
2	R	13.7
13	Seg.	14.1
17	Seg.	14.2
52	Seg.	14.7
4	R	15.0
5	R	15.2
68	R	16.3
28	R	16.8
1	R	17.3
36	R	17.8
29	Seg.	17.9
19	R	19.5
49	R	20.2
39	R	20.6
33	R	20.9
66	R	21.5
64	R	21.6
Eston	S	21.6
23	Seg.	22.0
57	R	22.0
31	Seg.	22.2
10	R	22.4
35	R	22.6

Cont'd....

Table 11 (cont'd) ...

Family number	Reaction to foliar infection[1]	Percent seed-borne ascochyta infection[2]
45	R	22.7
12	R	23.1
16	R	23.6
18	R	23.6
3	R	24.1
24	R	24.1
44	Seg.	25.5
20	R	25.7
55	R	25.8
11	R	27.1
59	R	27.3
41	R	27.7
26	R	27.8
3	Seg.	28.7
14	R	29.9
46	R	33.9
6	R	35.2
8	Seg.	40.4
60	S	45.4
22	S	61.8
48	S	63.2
15	S	65.1
Mean		21.3
SD		0.9
CV (%)		12.9
LSD ($P \leq 0.05$)		3.8

[1] R=resistant, Seg.=segregating, S=susceptible.
[2] Based on plating 100 seeds on 20% V8 juice agar, mean of two replications.

4.2.3 Effect of harvest date on percent seed-borne ascochyta infection of the parental lines

One row from each 4-row plot was harvested at approximately one-week intervals starting on August 19 when Laird lentil was at the proper swathing stage. The other three parent lines were fully mature at this stage. The data

were transformed by arcsin prior to statistical analysis. The results are summarized in Table 12. The two ascochyta resistant lentil lines ILL-5588 and ILL-5684 averaged only 3.2 and 9.4% seed-borne ascochyta infection, respectively, over the four sampling dates and only increased gradually over time. Thus, the ascochyta resistance of ILL-5588 and ILL-5684 lentil persists after maturity, the seeds exhibit low levels of seed-borne ascochyta infection and the seed coat does not discolor materially, even under conditions favorable for development of ascochyta blight. The susceptible parent Eston lentil averaged 27.9% seed-borne ascochyta infection, while the moderately resistant (foliar infection only) parent Laird lentil averaged 57.1%.

Table 12. Percent seed-borne ascochyta infection of parental lines of lentil at four harvest dates following maturity at the North Seed Farm, 1988

Parental lines	Percent seed-borne ascochyta infection[1]				
	Aug. 19	Aug. 26	Sept. 3	Sept. 10	$\bar{X}$
ILL-5588	2.6	2.1	3.1	4.9	3.2 a[2]
ILL-5684	4.6	7.5	8.4	16.9	9.4 a
Eston	26.0	29.7	28.1	34.8	29.7 b
Laird	38.6	64.9	67.0	58.0	57.1 c
$\bar{X}$	18.0 a	26.1 ab	26.7 ab	28.7 b	

[1]Based on plating 200 seeds on 20% V8 juice agar.
[2]Means followed by the same letter are not significantly different at the 0.05 level according to Duncan's multiple range test.

The first harvest date averaged 16.0% seed-borne ascochyta and this gradually increased to 28.7% at the fourth harvest date (Table 12). The parent by harvest date

interaction was significant due primarily to the large increase in percent seed-borne ascochyta infection in Laird lentil between the first and second harvest dates (26.3%) compared to the small change for the other three parents. Unfortunately, difference in maturity were confounded with differences in percent seed-borne ascochyta infection, the large differences were still evident at the end of the experiment three weeks after all entrees were fully matured.

As reported earlier, the resistance to foliar infection of ascochyta present in Laird lentil breaks down as the plant senesces. Wet weather at harvest time in the presence of a heavy inoculum load will result in seed infection and in severe cases the seed coats become badly discolored. Ochor and Trevathan (1987) also reported that the presence of Fusarium moniliforme in maize kernels increased as harvest was delayed beyond physiological maturity, emphasizing differences among genotypes. According to Wilcox et al. (1974) and Ellis and Sinclair (1976) delay of harvest adversely affects seed quality significantly in soybean (Glycine max) due to seed infection by Diaporthe phaseolorum Cke. and Ellis var. sojae Wehm., the causal organism of pod and stem blight of soybean. They conclude that, in the absence of cultivars resistant to D. phaseolorum var. sojae, harvesting soon after maturity is required to consistently produce high quality soybean seed.

4.3 Summer 1989

All F_2-derived F_3 families from 1988 were grown again as F_2-derived F_4 families in a replicated trial in 1989 and data were collected on ascochyta rating of the foliage and percent seed-borne ascochyta infection as before. The F_4 data in Tables 13 to 16 are presented in the identical family order

as for the F_3 data in Tables 8 to 11, allowing a quick comparison of similarities in ascochyta reaction in the two years. A summary of the two year average data on foliar infection and percent seed-borne ascochyta infection is presented in the Appendices A, B, C, and D.

4.3.1 Rating of foliar infection by ascochyta in F_2-derived F_4 families

The rating of foliar infection by ascochyta in Laird x ILL-5588 F_3 families (Table 8) was similar to that in the F_4 families (Table 13) with changes only in 4 of 36 families listed as segregating in F_3. The ascochyta rating of foliage in Laird x ILL-5684 F_3 families (Table 9) also was fairly similar to that in the F_4 families (Table 14) with changes in 8 of 28 families including one reversal from a resistant F_3 to a susceptible F_4 (Family 31). The ascochyta rating of foliage in Eston x ILL-5588 F_3 families (Table 10) also was fairly similar to that in the F_4 families (Table 15) with changes in 8 of 30 families. The ascochyta rating of foliage of Eston x ILL-5684 F_3 families (Table 11) also was fairly similar to that in F_4 families (Table 16) with changes in 20 of 62 families. In all cases, except the reversal noted above, these changes involved alteration of the rating of families rated as segregating in either F_3 or F_4. In addition, none of the changes in ascochyta rating of foliage from resistant in F_3 to segregating in F_4 involved families with low levels of seed-borne ascochyta infection. Thus, the highly resistant families were properly identified.

Table 13. Reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_4 families of Laird x ILL-5588 lentil at the North Seed Farm, 1989

Family number	Reaction to foliar infection[1]	Percent seed-borne ascochyta infection[2]
22	R	3.1
10	R	10.8
7	R	1.8
3	R	3.0
29	R	1.2
27	R	3.7
20	R	7.5
34	R	6.6
1	R	12.9
ILL-5588	R	3.2
30	R	17.8
8	R	10.6
14	R	8.4
4	R	6.8
28	R	7.1
32	R	6.0
16	R	5.9
36	R	6.3
26	R	2.5
35	R	14.2
37	R	9.8
9	R	13.8
33	R	9.6
15	R	3.7
21	R	13.0
11	Seg.	20.1
5	Seg.	23.7
17	Seg.	12.6
18	Seg.	22.4
33	Seg.	27.3
12	Seg.	26.3
24	Seg.	22.4
6	Seg.	33.7
25	Seg.	11.2
13	S	53.6
19	Seg.	32.6
Laird	MR	44.1
23	S	54.7
Mean		14.6
SD		1.3
CV (%)		27.8
LSD ($P \leq 0.05$)		5.1

[1] R=resistant, MR=moderately resistant, Seg.=segregating, S=susceptible.

[2] Based on plating 100 seeds on potato dextrose agar, mean of two replications.

Table 14. Reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_4 families of Laird x ILL-5684 lentil at the North Seed Farm, 1989

Family number	Reaction to foliar infection[1]	Percent seed-borne ascochyta infection[2]
23	R	16.9
3	R	25.7
13	R	9.6
10	R	10.0
ILL-5684	R	26.5
28	R	7.8
16	R	2.4
5	R	14.1
15	R	29.7
29	R	18.7
8	R.	12.1
30	Seg.	29.9
33	Seg.	23.5
11	Seg.	20.0
24	R	12.1
2	S	30.8
12	R	28.3
22	Seg.	27.8
9	Seg.	12.8
18	R	29.8
19	R	27.7
14	R	19.0
1	R	3.8
31	S	39.4
27	Seg.	29.4
20	S	45.1
17	Seg.	33.2
21	S	39.4
6	S	44.8
Laird	MR	40.8
Mean		23.0
SD		0.9
CV (%)		10.6
LSD ($P \leq 0.05$)		3.6

[1]R=resistant, MR=moderately resistant, Seg.=segregating, S=susceptible.

[2]Based on plating 100 seeds on potato dextrose agar, mean of two replications.

Table 15. Reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_4 families of Eston x ILL-5588 lentil at the North Seed Farm, 1989

Family number	Reaction to foliar infection[1]	Percent seed-borne ascochyta infection[2]
15	R	1.2
9	R	5.2
6	R	1.0
5	R	3.0
ILL-5588	R	2.9
7	R	8.9
10	R	18.7
26	R	17.6
8	R	16.2
32	R	10.3
19	R	12.4
22	Seg.	31.2
18	R	8.3
20	Seg.	30.6
34	R	16.8
17	Seg.	21.9
25	R	15.2
24	R	32.0
2	R	14.4
4	R	18.2
13	R	7.1
27	R	12.3
12	Seg.	22.4
3	R	3.2
28	Seg.	37.4
11	Seg.	20.1
16	Seg.	21.7
21	Seg.	30.2
23	Seg.	25.4
38	Seg.	21.8
Eston	S	34.2
31	R	10.2
Mean		16.5
SD		1.4
CV (%)		24.7
LSD ($P \leq 0.05$)		5.4

[1] R=resistant, MR=moderately resistant, Seg.=segregating, S=susceptible.

[2] Based on plating 100 seeds on potato dextrose agar, mean of two replications.

Table 16. Reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_4 families of Eston x ILL-5684 lentil at the North Seed Farm, 1989

Family number	Reaction to foliar infection[1]	Percent seed-borne ascochyta infection[2]
54	R	5.1
25	R	8.7
65	R	8.1
32	R	1.4
27	R	11.3
21	R	2.8
56	R	30.6
70	R	22.1
67	Seg.	34.9
42	R	27.2
69	Seg.	38.3
37	R	29.4
62	R	5.1
ILL-5684	R	25.7
9	R	22.6
30	Seg.	40.8
38	Seg.	45.2
7	R	26.4
61	R	13.7
2	R	15.3
13	R	29.1
17	R	26.2
52	Seg.	31.8
4	Seg.	41.3
5	R	17.0
68	R	19.2
28	R	26.8
1	Seg.	37.1
36	Seg.	33.3
29	Seg.	37.4
19	R	20.6
49	R	25.3
39	R	27.6
33	Seg.	33.4
66	Seg.	38.2
64	R	23.4
Eston	S	26.1
23	R	28.0
57	R	15.2
31	R	35.9
10	R	27.2
35	Seg.	46.1
45	R	29.7
12	R	29.9
16	R	27.4
18	R	49.3
3	R	33.2

Cont'd....

Table 16 (cont'd) ....

Family number	Reaction to foliar infection[1]	Percent seed-borne ascochyta infection[2]
24	R	24.0
44	R	30.5
20	Seg.	45.4
55	Seg.	38.6
11	R	27.6
59	Seg.	33.9
41	R	28.1
26	R	25.5
3	R	26.3
14	Seg.	21.9
46	Seg.	25.1
6	S	36.8
8	S	46.1
60	S	37.4
22	S	52.4
48	S	28.3
15	S	69.2
Mean		28.7
SD		1.0
CV (%)		10.4
LSD ($P \leq 0.05$)		4.1

[1]R=resistant, MR=moderately resistant, Seg.=segregating, S=susceptible.
[2]Based on plating 100 seeds on potato dextrose agar, mean of two replications.

4.3.2 Percent seed-borne ascochyta infection of F_2-derived F_4 families

The percent seed-borne ascochyta infection for each F_2-derived F_4 family in the four crosses is presented in Tables 13 to 16 with the individual families ranked from low to high, based on F_3 family means. The means and ranges for the F_3 and F_4 data were similar (Table 17), the major difference was the higher levels of seed-borne ascochyta infection in the F_4 than in the F_3 for the two crosses involving Eston lentil.

Table 17. Means and ranges for percent seed-borne ascochyta infection in F_2-derived F_3 families in 1988 and the same F_2-derived F_4 families in 1989 for four lentil crosses at the North Seed Farm

Cross	Mean	Range
Laird x ILL-5588 F_3	15.9	1.4-67.5
Laird x ILL-5588 F_4	14.6	1.2-54.7
Laird x ILL-5684 F_3	23.8	3.3-46.2
Laird x ILL-5684 F_4	23.0	2.4-45.1
Eston x ILL-5588 F_3	9.1	1.8-22.6
Eston x ILL-5588 F_4	16.5	1.0-37.4
Eston x ILL-5684 F_3	21.3	1.2-85.1
Eston x ILL-5684 F_4	28.7	1.4-69.2

The phenotypic correlation between percent seed-borne ascochyta infection in the F_3 and F_4 provides a meaningful estimate of the repeatability of the data. Thus, the cross Laird x ILL-5588 had the highest correlation between percent seed-borne ascochyta infection of F_2-derived F_3 families and the corresponding F_2-derived F_4 families (Table 18). In this cross, r^2 or 76% of the variation in percent seed-borne

ascochyta infection in the F_4 was associated with variation in percent seed-borne ascochyta in the F_3, suggesting that the breeder can confidently select for ascochyta resistance based on replicated F_2-derived F_3 family data. Unfortunately, results of other three crosses are less favourable (Table 18), with correlations ranging from 0.46 to 0.67 and coefficients of determination (r^2) ranging from 21% to 45%.

Narrow sense heritability can be estimated by the progeny-parent regression in self-pollinated crops. Thus, the narrow sense heritability estimates for percent seed-borne ascochyta infection ranged from 0.81 in Laird x ILL-5588 to 0.52 in Eston x ILL-5684 (Table 18). These medium high to high values indicate that selection for resistance to seed-borne ascochyta infection on the basis of replicated F_2-derived F_3 family data would be successful.

Table 18. Phenotypic correlations between generations and progeny-parent regressions (b) for percent seed-borne ascochyta infection of F_2-derived F_3 families and F_2-derived F_4 families for the four crosses of lentil

Crosses	r	b
Laird x ILL-5588	0.87 **	0.81 **
Laird x ILL-5684	0.67 **	0.71 **
Eston x ILL-5588	0.46 **	0.67 *
Eston x ILL-5684	0.57 **	0.52 **

*,** Significant at the 0.05 and 0.01 levels, respectively.

The cross Laird x ILL-5588 resulted in the highest phenotypic correlation between generations and the highest heritability for percent seed-borne ascochyta infection of the four crosses. Thus, this cross shows the greatest promise

for selecting for a high level of ascochyta resistance. The potential contribution of ILL-5588 to ascochyta resistance is suggested by the low level of percent seed-borne ascochyta infection over the two years, 4.1% vs. 18.6% for ILL-5684, 25.9% for Eston and 48.6% for Laird lentil (means from Tables 8 to 11 and 13 to 16). The data on percent seed-borne ascochyta infection averaged over the F_3 and F_4 of each cross (Table 19) substantiate the major effect of ILL-5588 lentil on ascochyta resistance. Thus, the two year average of both crosses involving ILL-5588 lentil as a parent was 13.9% seed-borne ascochyta infection compared with 24.2% for crosses involving ILL-5684 lentil as a parent. There was no difference in percent seed-borne ascochyta infection of crosses involving Eston or Laird lentil as a parent. This further substantiates the earlier finding that Laird lentil has resistance only to foliar infection and loses its resistance as it senesces and becomes as susceptible as Eston lentil to seed infection.

Table 19. Average percent seed-borne ascochyta infection for the four crosses of lentil, showing the average parent effect

	Parent		
Parent	ILL-5588	ILL-5684	Average
Laird	15.0[1]	23.4	19.2
Eston	12.8	25.0	18.9
Average	13.9	24.2	

[1]Each value is the mean of two replications in F_3 and F_4 of all F_2-derived families for this cross.

4.4 Integration of results on rating of foliar infection by ascochyta and percent seed-borne ascochyta infection

Rating of foliar infection by ascochyta on a scale of 1, 3 and 5 (as resistant) vs. 7 and 9 (as susceptible) is a qualitative assessment. This system was successfully used to calculate goodness-of-fit to F_2 segregation ratios which permitted the assignment of gene symbols for ascochyta in the four parents. The F_2 data indicated transgressive segregation for ascochyta resistance and susceptibility. The F_2 classification was corroborated by F_3 and F_4 data, except in the cross Eston x ILL-5684. This approach resulted in assigning a recessive gene to Laird lentil for resistance to foliar infection by ascochyta. However, as noted by previous workers (Morrall and Sheppard 1981, Gossen 1985, Morrall 1986), the resistance to foliar infection of Laird lentil breaks down as the plant senesces and it becomes susceptible to seed infection. Therefore, inheritance of resistance to seed infection was studied in a different manner.

Reaction to seed infection was based on percent seed-borne ascochyta infection which is a quantitative assessment. In this case, both Eston and Laird lentil were susceptible, ILL-5588 was resistant and ILL-5684 had an intermediate level of resistance to seed infection. Again, transgressive segregation was evident in the F_2, F_3 and F_4 for percent seed-borne ascochyta infection. Phenotypic correlations between generations and narrow sense heritability estimates were relatively high, indicating a major additive genetic component with only a moderate environmental component.

Results of the qualitative assessment (based on rating of foliar infection by ascochyta) and the quantitative assessment (based on percent seed-borne ascochyta infection)

agree very well when the loss of resistance by Laird during senescence is considered. Consequently, an effective system can be devised to facilitate selection for ascochyta resistance in lentil crosses using the resistance in ILL-5588 and ILL-5684 lentil.

4.5 Development of an efficient system of screening for ascochyta blight resistance in lentil

According to the results obtained in this study resistance to ascochyta in lentil can be selected in a highly effective and efficient manner, using the F_2-derived Family Method in an ascochyta nursery as follows:

Generation	Procedure
1	Make crosses (produce F_1 seed).
2	Individual F_2 plants: Select for resistance to foliar infection (plants rated 3 or 5) and less than 5% visibly spotted and damaged seed.
3	F_2-derived F_3 families: In a replicated trial, select among and within crosses for resistance to foliar infection and less than 3% visibly spotted and damaged seed. Use the seed plating technique to evaluate percent seed-borne ascochyta infection. Eliminate families with more than 5% seed-borne ascochyta infection. The selected F_2-derived F_3 families are crossed and backcrossed to commercial cultivars to produce ascochyta resistant commercial cultivars after screening as above.

5. SUMMARY AND CONCLUSIONS

The inheritance of ascochyta resistance was studied in F_2, F_2-derived F_3 and F_2-derived F_4 families of crosses between two Canadian lentil cultivars, Eston (susceptible) and Laird (moderately resistant) and two resistant lines from ICARDA, ILL-5588 and ILL-5684. The cultivar Eston was susceptible. Laird was resistant to foliar infection of ascochyta, but this resistance breaks down in the late podding stage. Under the wet conditions of the ascochyta nursery percent seed-borne infection in Laird lentil was even higher than in the susceptible cultivar Eston. The ascochyta resistant lines from ICARDA, ILL-5588 and ILL-5684, were highly resistant to foliar infection of ascochyta blight. This resistance persists after maturity and the seed coats do not become infected and discolor materially even with prolonged exposure to wet weather at harvest. These results suggest that the resistance present in the foliage was also exhibited by the seed in ILL-5588 and ILL-5684 lentil.

Segregation in the F_2, confirmed in the F_2-derived F_3 families, suggested that resistance to foliar infection by ascochyta in Laird lentil was conditioned by a single recessive gene $\underline{ral}_1$. Results also indicated that the ascochyta blight resistance (foliar and seed infection) of ILL-5588 and ILL-5684 was due to two dominant genes $\underline{Ral}_2$ and $\underline{Ral}_3$. ILL-5588, but not ILL-5684, also carried the recessive $\underline{ral}_1$ gene for resistance to foliar infection by ascochyta. Thus, the proposed genotypes and phenotypes are as follows:

Eston (susceptible)	$\underline{Ral}_1\,\underline{Ral}_1\ \underline{ral}_2\,\underline{ral}_2\ \underline{ral}_3\,\underline{ral}_3$
Laird (resistant to foliar infection)	$\underline{ral}_1\,\underline{ral}_1\ \underline{ral}_2\,\underline{ral}_2\ \underline{ral}_3\,\underline{ral}_3$

ILL-5588 (general resistance) $\underline{ral_1}\ \underline{ral_1}\ \underline{Ral_2}\ \underline{Ral_2}\ \underline{Ral_3}\ \underline{Ral_3}$
ILL-5684 (general resistance) $\underline{Ral_1}\ \underline{Ral_1}\ \underline{Ral_2}\ \underline{Ral_2}\ \underline{Ral_3}\ \underline{Ral_3}$

Evidence of transgressive segregation for both foliar infection in F_2 and percent seed-borne ascochyta infection in F_3 and F_4 suggests that each parent carried different genes for ascochyta resistance. However, transgressive segregation was not expected in all crosses, based on the proposed genotypes. Thus, this may be the result of additional unidentified genes or the presence of a few escapes or even a few late developing pods that never became infected as the crop dried.

The high correlation between percent seed-borne ascochyta infection in F_2-derived F_3 families and in F_2-derived F_4 families plus the medium to high heritability estimates (0.52 to 0.81) indicate that it will be easy to select for ascochyta resistance. The keys are to: (1) develop a uniformly high level of infection in the ascochyta nursery by spreading infected residue between the rows prior to flowering and then intermittently sprinkling the nursery twice daily, (2) select, on either an individual F_2 plant basis or on a replicated row basis (F_3 or later), those entries with less than 5% discolored seed, and (3) plate out seed of selected F_3 or later progenies to determine percent seed-borne ascochyta infection. Selection for percent discolored seed avoids selecting for resistance to foliar infection alone and assaying for percent seed-borne ascochyta provides a double check on possible ascochyta infection of the clear, bright seed.

6. REFERENCES

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Appendix A. Two year data on reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_3 families and F_2-derived F_4 families of Laird x ILL-5588 lentil at the North Seed Farm

Family number	Reaction to foliar infection[1]		Percent seed-borne ascochyta infection		
	F_3	F_4	F_3[2]	F_4[3]	Average
7	R	R	2.6	1.8	2.2
22	R	R	1.4	3.1	2.3
29	R	R	3.7	1.2	2.5
3	R	R	2.9	3.0	3.0
27	R	R	4.4	3.7	4.1
ILL-5588	R	R	5.8	3.2	4.5
34	R	R	5.5	6.6	6.1
20	R	R	4.8	7.5	6.2
10	R	R	2.0	10.8	6.4
26	R	R	11.1	2.5	6.8
4	R	R	7.6	6.8	7.2
28	R	R	7.6	7.1	7.4
32	R	R	8.7	6.0	7.4
14	R	R	7.3	8.4	7.9
16	R	R	9.9	5.9	7.9
36	R	R	10.9	6.3	8.6
8	R	R	6.7	10.6	8.7
1	R	R	5.5	12.9	9.2
15	Seg.	R	18.1	3.7	11.0
37	R	R	12.3	9.8	11.1
30	R	R	6.0	17.8	11.9
35	R	R	11.7	14.2	13.0
33	Seg.	R	17.3	9.6	13.5
9	R	R	14.3	13.8	14.1
21	Seg.	R	18.2	13.0	15.6
17	Seg.	Seg.	22.9	12.6	17.8
11	Seg.	Seg.	19.6	20.1	19.9
25	Seg.	Seg.	29.5	11.2	20.4
5	Seg.	Seg.	21.0	23.7	22.4
18	Seg.	Seg.	23.1	22.4	22.8
33	Seg.	Seg.	23.7	27.3	25.5
24	Seg.	Seg.	29.3	22.4	25.9
12	Seg.	Seg.	28.3	26.3	27.3
6	Seg.	Seg.	29.3	33.7	31.5
19	Seg.	Seg.	38.7	32.6	35.7
13	Seg.	S	38.5	53.6	46.1
Laird	MR	MR	52.7	44.1	48.4
23	S	S	67.5	54.7	51.1
Mean			15.9	14.6	
SD			0.2	1.3	
CV (%)			9.3	27.8	
LSD ($P \leq 0.05$)			1.9	5.1	

[1]R=resistant, MR=moderately resistant, Seg.=segregating, S=susceptible.
[2]Based on plating 100 seeds on 20% V8 juice agar, mean of two replications.
[3]Based on plating 100 seeds on DPA, mean of two replications.

Appendix B. Two year data on reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_3 families and F_2-derived F_4 families of Laird x ILL-5684 lentil at the North Seed Farm

Family number	Reaction to foliar infection[1]		Percent seed-borne ascochyta infection		
	F_3	F_4	F_3[2]	F_4[3]	Average
16	R	R	14.6	2.4	8.5
13	R	R	8.4	9.6	9.0
28	R	R	11.3	7.8	9.6
10	R	R	9.7	10.0	9.9
23	R	R	3.3	16.9	10.1
8	Seg.	R	17.7	12.1	14.9
5	R	R	16.8	14.1	15.5
3	R	R	7.7	25.7	16.7
1	Seg.	R	30.7	3.8	17.3
24	R	R	22.8	12.1	17.5
29	Seg.	R	17.5	18.7	18.1
ILL-5684	R	R	10.9	26.5	18.7
11	Seg.	Seg.	20.1	20.0	20.1
9	Seg.	Seg.	28.2	12.8	20.5
33	Seg.	Seg.	19.3	23.5	21.4
15	R	R	17.5	28.7	23.6
30	R	Seg.	18.4	29.9	24.2
14	R	R	30.5	19.0	24.8
22	Seg.	Seg.	27.2	27.8	27.5
12	Seg.	R	27.1	28.3	27.7
2	Seg.	S	24.3	30.8	27.6
19	R	R	30.2	27.7	29.0
18	R	R	29.2	29.8	29.5
27	Seg.	Seg.	36.0	29.4	32.7
31	R	S	31.9	39.4	35.7
17	Seg.	Seg.	39.2	33.2	36.2
20	Seg.	S	36.5	45.1	40.8
21	S	S	43.0	39.4	41.2
6	S	S	46.2	44.8	45.5
Laird	MR	MR	56.8	40.8	48.8
Mean			23.8	23.0	
SD			1.3	0.9	
CV (%)			15.9	10.6	
LSD ($P \leq 0.05$)			5.6	3.6	

[1]R=resistant, MR=moderately resistant, Seg.=segregating, S=susceptible.
[2]Based on plating 100 seeds on 20% V8 juice agar, mean of two replications.
[3]Based on plating 100 seeds on DPA, mean of two replications.

Appendix C. Two year data on reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_3 families and F_2-derived F_4 families of Eston x ILL-5588 lentil at the North Seed Farm

Family number	Reaction to foliar infection[1]		Percent seed-borne ascochyta infection		
	F_3	F_4	F_3[2]	F_4[3]	Average
15	R	R	1.8	1.2	1.5
6	R	R	3.6	1.0	2.3
5	R	R	4.0	3.0	3.5
9	R	R	1.9	5.2	3.6
ILL-5588	R	R	4.5	2.9	3.7
7	R	R	4.8	8.9	6.9
18	R	R	6.4	8.3	7.4
32	R	R	5.6	10.3	8.0
3	R	R	13.2	3.2	8.2
13	R	R	9.5	7.1	8.4
19	R	R	6.2	12.4	9.3
8	R	R	5.6	16.2	10.9
25	R	R	7.0	15.2	11.1
2	R	R	7.9	14.4	11.2
26	R	R	5.4	17.6	11.5
2	R	R	11.1	12.3	11.7
34	R	R	6.7	16.8	11.8
10	R	R	5.1	18.7	11.9
4	R	R	8.2	18.2	13.2
17	R	R	6.9	21.9	14.4
31	Seg.	R	22.6	10.2	16.4
12	Seg.	Seg.	11.2	22.4	16.8
11	R	Seg.	14.4	20.1	17.4
16	Seg.	Seg.	15.1	21.7	18.4
20	R	Seg.	6.7	30.6	18.7
22	R	Seg.	6.3	31.2	18.8
24	Seg.	R	7.7	32.0	19.9
38	Seg.	Seg.	20.1	21.8	21.0
23	Seg.	Seg.	18.0	25.4	21.7
21	R	Seg.	15.9	30.2	23.1
28	R	Seg.	13.5	37.4	25.5
Eston	S	S	21.8	34.2	28.0
Mean			9.1	16.5	
SD			0.5	1.4	
CV (%)			29.4	24.7	
LSD ($P \leq 0.05$)			3.9	5.4	

[1] R=resistant, MR=moderately resistant, Seg.=segregating, S=susceptible.
[2] Based on plating 100 seeds on 20% V8 juice agar, mean of two replications.
[3] Based on plating 100 seeds on DPA, mean of two replications.

Appendix D. Two year data on reaction to foliar infection by ascochyta and percent seed-borne ascochyta infection of the F_2-derived F_3 families and F_2-derived F_4 families of Eston x ILL-5684 lentil at the North Seed Farm

Family number	Reaction to foliar infection[1]		Percent seed-borne ascochyta infection		
	F_3	F_4	F_3[2]	F_4[3]	Average
54	R	R	1.2	5.1	3.2
32	R	R	5.9	1.4	3.7
21	R	R	7.1	2.8	5.0
25	R	R	3.8	8.7	6.3
65	R	R	4.6	8.1	6.4
62	R	R	11.1	5.1	8.1
27	R	R	6.8	11.3	9.1
61	R	R	13.3	13.7	13.5
2	R	R	13.7	15.3	14.5
70	R	R	9.6	22.1	15.9
5	R	R	15.2	17.0	16.1
9	R	R	11.7	22.6	17.2
68	R	R	16.3	19.2	17.8
57	R	R	22.0	15.2	18.6
ILL-5684	R	R	11.4	25.7	18.6
42	R	R	10.1	27.2	18.7
56	R	R	7.1	30.6	18.9
19	R	R	19.5	20.6	20.1
17	Seg.	Seg.	14.2	26.2	20.2
37	R	R	11.1	29.4	20.3
7	R	R	13.2	28.4	20.8
13	Seg.	R	14.1	29.1	21.6
28	R	R	16.8	26.8	21.8
67	R	R	9.7	34.9	22.3
64	R	R	21.6	23.4	22.5
49	R	R	20.2	25.3	22.8
52	Seg.	Seg.	14.7	31.8	23.5
Eston	S	S	21.6	26.1	23.9
24	R	R	24.1	24.0	24.1
39	R	R	20.6	27.6	24.1
69	Seg.	Seg.	10.6	38.3	24.5
10	R	R	22.4	27.2	24.8
23	Seg.	R	22.0	28.0	25.0
3	R	R	24.1	26.3	25.2
16	R	R	23.6	27.4	25.5
36	R	Seg.	17.6	33.3	25.6
14	R	Seg.	29.9	21.9	25.9
45	R	R	22.7	29.7	26.2
30	R	Seg.	12.2	14.8	26.5

Cont'd...

Appendix D. (cont'd)...

Family number	Reaction to foliar infection[1]		Percent seed-borne ascochyta infection		
	F_3	F_4	F_3[2]	F_4[3]	Average
12	R	R	23.1	29.9	26.5
26	R	R	27.8	25.5	26.7
1	R	Seg.	17.3	37.1	27.2
33	R	Seg.	20.9	33.4	27.2
11	R	R	27.1	27.6	27.4
3	Seg.	R	28.7	26.3	27.5
29	Seg.	Seg.	17.9	37.4	27.7
41	R	R	27.7	28.1	27.9
44	Seg.	R	25.5	30.5	28.0
4	R	Seg.	15.0	41.3	28.2
38	Seg.	Seg.	12.7	45.2	29.0
31	Seg.	R	22.2	35.9	29.1
46	R	Seg.	33.9	25.1	29.5
66	R	Seg.	21.5	38.2	29.9
59	R	Seg.	27.3	33.9	30.6
55	R	Seg.	25.8	38.6	32.2
35	R	Seg.	22.6	46.1	34.4
20	R	Seg.	25.7	45.4	35.6
6	R	S	35.2	36.8	36.0
18	R	R	23.6	49.3	36.5
60	S	S	45.4	37.4	41.4
8	Seg.	S	40.4	46.1	43.3
48	S	S	63.2	28.3	45.8
22	S	S	61.8	52.4	57.1
15	S	S	85.1	69.2	77.2
Mean			21.3	28.6	
SD			0.9	1.0	
CV (%)			12.9	10.4	
LSD ($P \leq 0.05$)			3.8	4.1	

[1]R=resistant, MR=moderately resistant, Seg.=segregating, S=susceptible.
[2]Based on plating 100 seeds on 20% V8 juice agar, mean of two replications.
[3]Based on plating 100 seeds on DPA, mean of two replications.