CHAPTER 18

LENTIL DISEASES

PAUL TAYLOR[1], KURT LINDBECK[2], WEIDONG CHEN[3], AND REBECCA FORD[4]

[1]*Center for Plant Health and BioMarka, Faculty of Land and Food Resources, The University of Melbourne, Victoria 3010, Australia*
[2]*Grains Innovation Park, The Department of Primary Industries, Private Bag 260, Horsham, Victoria 3401, Australia*
[3]*USDA-ARS, Grain Legume Genetics and Physiology Research Unit, 303 Johnson Hall, Washington State University, Pullman, WA 99164-6402, USA*
[4]*BioMarka, Faculty of Land and Food Resources, The University of Melbourne, Victoria 3010, Australia*
E-mail: paulwjt@unimelb.edu.au

Abstract: Fungal diseases of lentils are the most important biological constraint to productivity. *Ascochyta lentis* (ascochyta blight) and *Fusarium oxysporum* f. sp. *lentis* (fusarium wilt) are the major fungal pathogens that can cause severe losses in most lentil growing regions of the world. Fungal diseases such as botrytis grey mould (*Botrytis fabae* and *B. cinerea*), rust (*Uromyces viciae-fabae*), stemphylium blight (*Stemphylium botryosum*), and anthracnose (*Colletotrichum truncatum*) are also important in some growing seasons in particular countries when environmental conditions are conducive for infection. Lentil plants can also be infected by a range of viruses but generally the affect on yield is not as great as that caused by fungal pathogens. Lentil yellows disease caused by bean leaf roll virus (BLRV), beet western yellows virus (BWYV), or subterranean clover red leaf virus (SCRLV) is widespread throughout the world. Other important virus diseases of lentil include bean yellow mosaic (BYMV), pea seed borne mosaic (PSbMV), cucumber mosaic (CMV), alfalfa mosaic (AMV) and broad bean stain (BBSV). Integrated disease management practices including use of resistant cultivars, modified cultural practices and use of fungicides or insecticides can reduce the impact of these diseases on lentil production

1. INTRODUCTION

Lentil plants are affected by a wide range of pathogens with fungal diseases being the most important. These decrease productivity through infection and damage to leaves, stems, roots and pods, and reduce marketability by discoloring seed. Most

S.S. Yadav et al. (eds.), Lentil: An Ancient Crop for Modern Times, 291–313.

major economically important diseases are found in all lentil growing regions of the world eg., ascochyta blight, whereas, diseases like fusarium wilt and anthracnose have not been detected in some major lentil producing countries such as Australia. As well, certain virulent pathotypes of a pathogen have restricted geographical range (eg. Ct0 pathotype of anthracnose in Canada). Therefore, it is important for industry and quarantine personnel to be aware of the range of pathogens that can infect lentil, and be able to identify the cause(s) of a disease outbreak, or be able to detect exotic pathogens on imported seed or plant parts. Early correct identification and detection of lentil pathogens will prevent incursion of exotic diseases into areas where the particular disease does not exist.

The major fungal diseases of lentil are described with descriptions of the causal organism, symptoms produced on the plant, epidemiology, and disease management and control. Viruses are then described followed by a listing of minor lentil diseases.

2. ASCOCHYTA BLIGHT

Ascochyta blight, caused by *Ascochyta lentis* Bond. and Vassil, is one of the most important biotic constraints to lentil production (Figure 1a). Able to attack all above ground plant parts at any growth stage under favorable conditions, the disease causes reduction in yield and seed quality. The disease is prevalent throughout the world and has been reported to cause yield losses of up to 70%, 30–50% and 50% in Canada, USA and Australia respectively (Gossen and Morrall, 1983; Kaiser, 1992; Brouwer *et al.*, 1995).

There are two stages within the *A. lentis* life-cycle, the asexual or anamorph stage and the sexual or teleomorph stage. The asexual stage is characterized by the production of pycnidia in the lesions on infected plants, which are 175–300 μm in diameter with a minute round osteole (Bondartzeva-Monteverde and Vassilievsky; 1940 cited by Agrawal and Prasad, 1997). The pycnidia release conidia which are cylindrical, straight or rarely curved, round at the ends with a median septum. The teleomorph (*Didymella lentis*) was observed for the first time on over wintered lentil straw in 1992 in Idaho, USA (Kaiser and Hellier, 1993), confirming the heterothallic nature of *A. lentis*, with two distinct mating types. Compatible mating types (MAT1-1 and MAT1-2) are required for the development of fertile pseudothecia and viable ascospores. Kaiser *et al.* (1997) differentiated the teleomorphs of *A. fabae* and *A. lentis* on the basis of a pathogenicity test and morphology. They also distinguished ascospores of *A. fabae* from *A. lentis* using molecular markers. Kaiser (1997) identified both mating types in isolates from Australia, Canada, Italy, Morocco, New Zealand, Pakistan, Spain, Syria, Turkey and USA. Consequently, *D. lentis* was proposed as a new species that was distinct from *D. fabae.*

2.1. Symptoms

The symptoms of the disease include lesions on leaves, petioles, stems and pods (Figure 1a). The irregularly shaped lesions on leaves, petioles and stem are tan

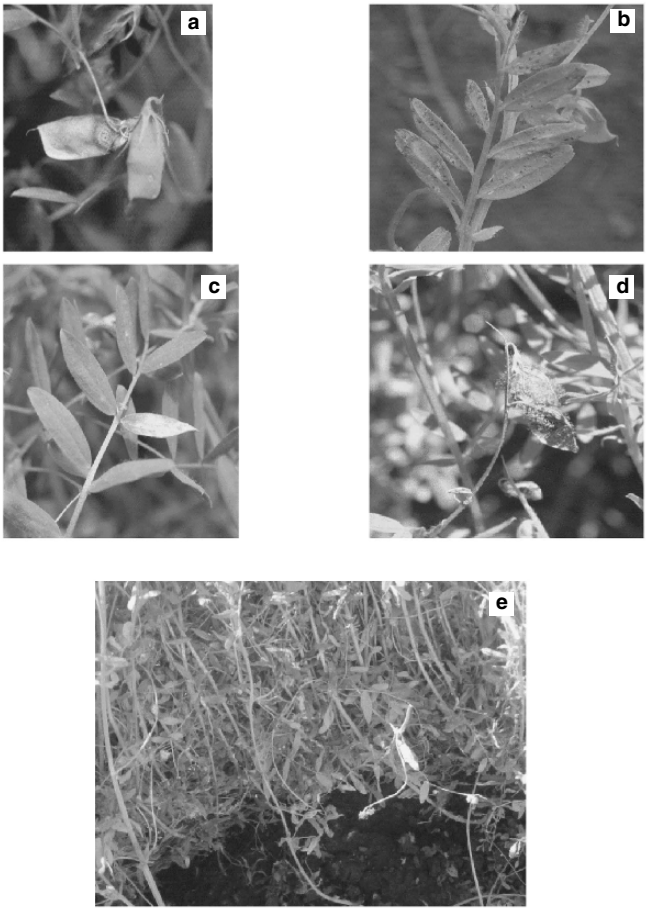

Figure 1. Diseases of lentil. a) Ascochyta blight of pods – *Ascochyta lentis*; b) rust on leaves – *Uromyces viciae-fabae*; c) Stemphylium blight of leaves – *Stemphylium botryosum*; d) Botrytis grey mould of pods – *Botrytis fabae*; e) White mold – *Sclerotinia sclerotiorum*

and darker brown on pods and seeds. Black pycnidia are visible in the centre of mature/older lesions. In severe infection, lesions can girdle the stem, leading to breakage and subsequent death of all tissues above the lesion. Heavily infected seeds are shriveled and discoloured with whitish mycelium and pycnidia (Kaiser and Hannan, 1986).

2.2. Epidemiology

Cool, wet weather is conducive to *A. lentis* infection, disease development and spread. The disease affects all the aerial parts of the plant and is seed borne. Conidia may be dispersed under rain splash up to 15 cm (Pedersen *et al.*, 1994)

while wetness periods of 24–48 hrs and temperatures of 10–15°C are optimum for infection (Pedersen and Morrall, 1994). The pathogen may also be dispersed by wind blown infected leaflets (Pedersen *et al.*, 1994) and through infected seed (Kaiser and Hannan, 1986). Kaiser and Hannan (1986) found *A. lentis* infection in seeds of 46 accessions from 30 countries. They also found the pathogen on seed from countries where the disease was previously unrecorded. The pathogen was found to survive in infected pods and seeds for over three years when stored under optimum conditions. However, the fungus lost viability after 21 weeks on seeds and 29 weeks on pods when buried at a soil depth of 16 cm. Kaiser *et al.* (1989) stored infected lentil seed at 20, 5, −18 and −160 to −196°C and proposed that the pathogen would survive in infected lentil seed as long or longer than the seed remained viable.

The initial infection process was studied by Roundhill *et al.* (1995). Detached leaves inoculated with a spore suspension of *A. lentis* conidia germinated within six hours of inoculation, and germ tubes and appressoria developed after 10 hours. A penetration peg then pierced the cuticle, often near the junction of two epidermal cells and cytoplasm aggregated adjacent to the infection hypha. Within 40 hours, the plasmalemma was disrupted and by 52 hours, the cytoplasm and nucleus broke down and the cell became largely occupied by the fungus with only remnants of the protoplasm present. Once the epidermis was fully colonized, the pathogen invaded the mesophyll with macroscopic symptoms being evident by day nine.

2.3. Management and Control

The most economical and sustainable strategies to control ascochyta blight are through resistance breeding along with cultural practices. Losses due to ascochyta blight can be minimised by crop rotation, early sowing to escape moist weather at harvest, the use of disease free seed and burning of diseased debris from the previous crop (Nene *et al.*, 1988). A three-year break between lentil crops reduced the amount of inoculum in the soil. Sun drying of lentil seed was found to be useful in controlling seed borne inoculum (Beniwal *et al.*, 1989). Hot water and dry heat treatment at 55 °C for 25 minutes and 70 °C for 24 hrs respectively inhibited fungal growth in the seed however, seed germination declined drastically with the hot water treatment (Ahmed and Beniwal, 1991).

A large number of fungicides have been evaluated for control of seed-borne infection with benomyl, carbendazim, carbathiin, ipodion and thiobendazole reported to be effective in varying degrees (Morrall, 1988; Bretag, 1989). Metalaxyl and thiram have also been found to reduce fungus growth but thiram did not control the disease effectively in the field (Bretag, 1989). In particular, Kaiser and Hannan (1987) reported greater seedling emergence from infected lentil seed and increased yield after treatment with thiobendazole and benomyl but thiobendazole showed a phytotoxic effect at 3 g or more a.i./ kg of seed. The effects of foliar application of fungicides were studied by Beauchamp *et al.* (1986) who reported

that captafol, chlorothalonil, folpet and metiram completely inhibited conidia germination at 32 μg/ml or less. Seed yield increased and seed infection was reduced using single applications of these fungicides at early bloom to early pod set.

Lentil breeding programs have developed resistant cultivars however, knowledge of pathogenic diversity is important when choosing appropriate isolates to screen for resistance. Many studies have shown pathogenic diversity among isolates by assaying a set of host-specific differential genotypes or cultivars. Ahmed *et al.* (1996) studied the virulence of 84 *A. lentis* isolates from Canada and 16 isolates from other countries and found that isolates collected in 1978 were less virulent than those collected in 1992. This increase in virulence over time may have been due to genetic recombination and/or host genotype–directed selection for specific virulence in the pathogen population. This highlights the need to evaluate the host reaction to the disease with highly virulent isolates in order to identify the most robust resistance sources. Alternatively, screening of germplasm in the field where breeding lines are exposed to the local population of *A. lentis* can account for variability within the pathogen population (Tivoli et al. 2006).

Nasir and Bretag (1997a) divided a collection of Australian *A. lentis* isolates into six pathotypes based on quantitative differences in pathogenicity. However, there is concern as to whether true pathotype differences exist or if the differences observed in disease severity are a measure of the natural distribution of aggressiveness within a population, ranging from low to high (Taylor and Ford 2007). Banniza and Vandenberg (2006) reported that the host reaction of 16 lentil genotypes to 65 isolates of *A. lentis* collected in Canada resulted in a continuum of severity of infection. These results indicated natural variation of aggressiveness in the population without any distinct pathotypes. Taylor and Ford (2007) defined a pathotype as a subclass or group of a pathogen distinguished from others of the same species by its pathogenicity on a specific host (genotype) ie a qualitative difference in disease severity. Whereas, aggressiveness reflects the natural variation in pathogenicity or level of disease (measured quantitatively) within the pathogen population. However, since resistance to *A. lentis* has been found to be controlled by specific resistance genes (Ford et al., 1999; Nguyen et al., 2001), there is the likelihood that pathotypes of *A. lentis* have evolved that have qualitative differences on lentil genotypes.

3. FUSARIUM WILT

Fusarium wilt of lentil is an important disease reported in every continent where lentil is grown except Australia (Beniwal *et al.*, 1993; Tosi and Cappelli, 2001). The disease may cause complete crop failure under favorable conditions for disease development, and can be the major limiting factor for lentil cultivation in certain areas (Chaudhary and Amarjit, 2002). The common name lentil wilt has been used to describe many general wilting and dying symptoms. Hence a number of pathogens have been reportedly associated with lentil wilt (Khare, 1981) possibly because of the difficulty in species identification and confusion in the fusarium

taxonomy. Strictly speaking, the causal organism of vascular wilt of lentil is *Fusarium oxysporum* Schlecht. Emend. Snyder & Hansen f. sp. *lentis* Vasudeva and Srinivasan. Although its sexual state has not been found, it is generally believed to belong to the Hypocreales of Ascomycetes.

In culture, the mycelium of the pathogen is hyaline, septate and much branched. Growth patterns on media vary from fluffy to appressed and vary in color from no color to pink. *F. oxysporum* f. sp. *lentis* produces three kinds of spores: microconidia; multi-septate macroconidia, which have a distinct foot cell and a pointed apical cell; and chlamydospores (Khare 1980). Microconidia are ovoid or kidney-shaped, hyaline and usually one celled. Macroconidia are long with pointed apical cell and notched basal cell, and two to seven celled. Chlamydospores are oval or spherical, one-celled, and thick walled, formed singly in macroconidia or apical or intercalary in the hyphae.

3.1. Symptoms

Fusarium wilt usually occurs near or at reproductive stages (flowering to pod-filling) of crop growth. Symptoms include wilting of top leaves that resemble water deficiency, stunting of plants, shrinking and curling of leaves from the lower part of the plants that progressively move up the stems of the infected plant. Plants finally become completely yellow and die. Root symptoms include reduced growth with marked brown discoloration, tap root tips that are damaged and proliferation of secondary roots above the area of tap root injury. Discoloration of vascular tissue in the lower stem may not always be visible. However, in India, the disease has also been reported to occur at the seedling stage. General symptoms at the seedling stage include seed rot and sudden drooping more like wilting and damping off (Khare, 1980).

Field diagnosis should be done in connection with field cropping history. Recent lentil production especially with a history of fusarium wilt will indicate potential wilt problems. Suspect stunted and wilted plants should be carefully removed from the soil so that the roots can be checked for reduced growth without external fungal growth. External fungal growth indicates the presence of other diseases such as collar rot. Lower stems should be split to check for vascular discoloration. Although vascular discoloration is not always symptomatic of fusarium wilt the presence of discoloration would confirm the disease. Culturing of infected plant tissue in the laboratory should be done with caution because of the possible presence of other saprophytic *Fusarium* spp. that appear similar to *F. oxysporum* f. sp. *lentis*. A pathogenicity test on lentil is necessary to confirm *F. oxysporum* f. sp. *lentis*.

3.2. Epidemiology

Like many other formae specilaes of *F. oxysporum*, the pathogen has a very limited host range as it only infects lentil in nature. In inoculation studies, *F. oxysporum* f. sp. *lentis* was unable to infect cowpea, french-bean, bengal gram, lathyrus,

mungbean, uribean, pea, soybean or red gram (Khare, 1980). The disease is favored by warm and dry conditions (Bayaa and Erskine, 1998) with an optimal temperature of 22–25 °C.

F. oxysporum f. sp. *lentis* is a soilborne pathogen, although seed infestation and infection is common. The chlamydospores can survive in soil either in dormant form or saprophytically for several years without a suitable host. A survey of soil samples from Sangod Tehsil of Kota, Rajasthan, India, found that *F. oxysporum* f. sp. *lentis* was the most prevalent lentil pathogen (Chaudhary and Amarjit, 2002). Synergistic interaction between *F. oxysporum* f. sp. *lentis* and root knot nematode *Meloidogyne javanica* was observed in lentil cultivars resistant or susceptible to fusarium wilt (De *et al.*, 2001). Presence of the nematode significantly increased wilt incidence, caused significant reduction in shoot length, root length and nodulation in both susceptible and resistant cultivars (De *et al.*, 2001).

3.3. Control Methods

The most economical means to control fusarium wilt of lentil is through the use of resistant cultivars (Bayaa et al., 1997; Stoilova and Chavdarov, 2006). Resistant or moderately resistant lentil cultivars (DPL 58, DPL 61 and DPL 62) significantly reduced wilt incidence and severity of root rot, and increase grain yield (Chaudhary and Amarjit, 2002). Studies in genetics of resistance to fusarium wilt will eventually help to produce more resistant lentil cultivars (Eujayl et al., 1998). Selecting cultivars that mature early and adjusting the planting date if possible can reduce disease incidence by escaping a portion of lentil growth from weather conditions favorable to the disease. The most suitable planting dates vary according to the different production regions. Use of clean seed for sowing and/or the use of fungicidal seed treatments can eliminate or reduce contaminating inoculum sources. Since the pathogen has a very restricted host range, a three to five year rotation will help reduce inoculum level in the field.

Although *in vitro* and greenhouse tests showed fungicides were effective against fusarium wilt, field applications were not always practical because of the cost and technical difficulty of incorporating chemicals into soil during the growing season. Seed treatment with benomyl fungicide reduced the incidence of fusarium wilt. Biological control has been a focus of recent research. El-Hassan and Gowen (2006) tested three formulations to enhance efficacy of the biocontrol agent *Bacillus subtilis*, and found that formulations with either talc or glucose significantly decreased disease severity and showed enhanced plant growth promoting activity by increasing root length. Efficacy and practicality of biocontrol in the field remains to be worked out.

4. BOTRYTIS GREY MOULD

Botrytis grey mould (BGM) of lentil, caused by the fungal pathogens *Botrytis fabae* (Sard) (teleomorph: *Botryotinia fabae*) and *B. cinerea* (Pers.: Fr.) (*Helotiales, Sclerotiniaceae*) (teleomorph: *Botryotinia fuckeliana),* is a serious, but sporadic

disease. Knights (1987) first reported the disease in Australia on lentil in the wet year of 1983 and since then the disease (Figure 1d) has caused considerable damage to commercial lentil crops grown throughout Victoria and South Australia (Lindbeck et al. 2003). In Canada, the disease was first reported in 1970 (Morrall et al. 1972) with serious epidemics of botrytis stem and pod rot occurring from 1992 to 1994 (Morrall 1997). A series of cool, wet summers in those years provided ideal conditions for botrytis epidemics to occur. Elsewhere around the world the disease has been recorded on lentil in the USA in 1964 (Wilson and Brandsberg 1965) and in New Zealand in 1987 (Cromey et al. 1987). Botrytis grey mould has also been reported as being a serious problem throughout the sub-continent including Bangladesh (Gowda and Kaul 1982), Nepal (Karki 1993) and Pakistan (Bashir and Malik 1988, Iqbal et al. 1992). Brouwer et al. (2000) found only *Botrytis cinerea* to be a problem in lentil production in Pakistan but not the rest of the Indian subcontinent, despite *B. fabae* being a common disease in the region. On the South American continent, the disease has been reported as a production constraint in Colombia (Bascur 1993). *B. cinerea* has also been isolated off infected and dying plants in Chile (France et al. 1988) and from lentil with symptoms in the field in northern Egypt (Hamdi and Hassanein 1996). In Europe, the production of lentil is claimed to be limited by low profitability and its susceptibility to *Botrytis* in wet climates (Carrouee et al. 2000).

Colonies of *B. cinerea* grow quickly, reaching 6.0 cm diameter and more in 10 days at 20°C on oatmeal agar, at first hyaline but later becoming grey to greyish brown (Domsch et al. 1980). Conidiophores arise irregularly and often in patches, without a basal swelling, frequently 2 mm or more long, mostly 16−30 μm thick, branched, often with a stipe and a rather open head of branches, smooth, clear, brown below, paler near the apex, with the ends of the branches often quite colourless. The conidia are ellipsoidal or obovoid, often with a slightly protuberant hilum; colourless to pale brown, smooth, 6−18 × 4−11 μm (mostly 8−14 × 6−9 μm) (Ellis and Waller 1974a). Sclerotia are black and usually smaller and thinner than those of *Sclerotinia sclerotiorum*. *Botrytis cinerea* is distributed worldwide, but occurs mainly in humid temperate and subtropical regions (Domsch et al. 1980).

Ellis and Waller (1974b) provided a description of *B. fabae*. Conidiophores are not normally found on leaves under field conditions but develop and produce conidia in a humid chamber. In culture on bean leaf agar conidium production is encouraged by the presence of relatively high concentrations of inorganic salts such as sodium nitrate. Sclerotia are formed abundantly in culture, discrete or sometimes confluent, mostly 1−1.7 mm in diameter, rarely up to 3 mm. Conidia are always much larger than those of *Botryotinia fuckeliana*, 14−29 × 11−20 μm (mostly 16−25 × 13−16 μm).

4.1. Symptoms

All aboveground plant parts of lentil can be affected by botrytis grey mould. Depending on the location of the crop, symptoms may initially appear either on

flowers and pods (Figure 1d), or lower in the crop canopy. The most damaging symptoms become apparent after the crop has reached canopy closure and a humid microclimate is produced under the crop canopy. The disease first appears on the lower foliage as discrete lesions on leaves which are initially dark green, but turn greyish-brown, then cream as they age, that enlarge and coalesce to infect whole leaflets. Severely infected leaves senesce and fall to the ground. These can often act as a secondary source of inoculum by lodging in leaf and stem axils and initiating stem infections. If the canopy remains humid for extended periods infection can spread to the lower stems which quickly become girdled and covered with a furry layer of conidiophores, eventually causing stem death and whole plant infection.

Death of plants can often occur before the onset of flowering and pod fill. Infection will continue to spread resulting in patches of dead plants within crops (Bayaa and Erskine 1998). When the weather turns dry and the infected plants are disturbed, clouds of spores are released into the air. Flowers can show symptoms of infection with typically grey mouldy growth present on petals, causing flower death (Bayaa and Erskine 1998). Pods which become infected will be covered in the grey mouldy growth, rot, and turn brown when dried out. Seeds within these pods will fail to fill properly (Davidson et al. 2004). Infected seeds are discoloured and shrivelled (Bayaa and Erskine 1998). When infected seeds are sown seedling blight can occur. Seedling blight is characterised by the prolific grey mycelial growth of the pathogen on the hypocotyl at the soil line (Morrall 1997). This stage of the disease also has the potential to spread along seedling rows as the pathogen spreads from plant to plant (Morrall 1997), reducing seedling populations.

4.2. Epidemiology

There are several main sources of inoculum of botrytis grey mould, these include; seed-borne inoculum, sclerotia, mycelium in old infected trash, and alternate host plants. In Australia, *B. cinerea* and *B. fabae* have been frequently isolated off lentil seed (T. Bretag and K. Lindbeck unpublished data). Under Canadian conditions *B. cinerea* has been found to be highly seed-borne and can affect seed viability, seedling emergence and crop establishment (Morrall 1997). *Botrytis cinerea* has also been isolated from lentil seed in India (Rajendra et al. 1987), Spain (Diaz and Tello 1994) and the USA (Kaiser 1992). Sclerotia are considered the main survival structure for both *B. cinerea* and *B. fabae*. Sclerotia produced by *B. fabae* were considered an important source of inoculum for chocolate spot of faba beans (Harrison 1979), but only on the soil surface where the bodies are exposed to sunlight and produce conidia. In addition, sclerotia have the ability to produce conidia over an extended period of time. Under laboratory conditions sclerotia of *B. cinerea* were found to continue to sporulate for approximately 12 weeks after the first crop of conidia (Nair and Nadtotchei 1987). Resting mycelium in old host plant debris may survive and produce conidia under humid conditions for extended periods (Bayaa and Erskine 1998). Both *B. cinerea* and *B. fabae* can survive in

lentil trash on the soil surface for at least 12 months under Australian conditions (K. Lindbeck, unpublished data).

The development of botrytis grey mould epidemics is largely determined by the prevailing environmental conditions during periods of inoculum production and dispersal in the presence of the host. The coinciding of all these events can result in the development of an epidemic very quickly when compared to most other diseases (Jarvis 1980b). It is generally assumed that for *B. cinerea*, inoculum is always present in the field and that production, liberation and dispersal of inoculum is an ongoing process (Jarvis 1980b), for *B. fabae* this principle will not always apply given its restricted host range. Environmental conditions and canopy density have also been shown to be primary factors that influence the development of botrytis grey mould epidemics in lentil crops (Kaiser 1992, Morrall 1997, Bailey et al. 2000). A dense crop canopy, especially following canopy closure, and humid conditions following rain favour the sporulation and dispersal of *B. cinerea* on decaying lentil tissue (Kaiser 1992), and its appearance is often characteristic of a lentil crop with rank growth (Morrall 1997). There have been numerous studies on identifying the optimum temperatures and relative humidities for disease development by *B. cinerea* and *B. fabae* on other host crops, namely chickpea and faba bean. Temperatures ranging from 15−25 °C and RH > 95% have been found to be optimal for initiation and development of disease (Harrison 1980, Wilson 1937, Tripathi and Rathi 1992, Rewal and Grewal 1989) particularly at flowering and after canopy closure (Lindbeck et al. 2002).

Botrytis cinerea has a broad crop host range collectively, including faba bean, chickpea, field pea, lupin and pasture legumes such as lucerne and clover. Other host species include a wide range of ornamental and horticultural crops. This provides the pathogens with a wide geographic distribution and alternate host mechanism. *B. cinerea* is known to have over 200 host plant species including many ornamental, horticultural, field crop and weed species (Jarvis 1980a). The wide host range of *B. cinerea* is likely to make the role of alternate hosts an important part in survival from one season to the next (Davidson et al. 2004). Unlike *B. cinerea*, *B. fabae* is known to have a more restricted host range. Yu (1945) found only four plant species were able to become infected, (ie, produce lesions) after inoculating 28 species of leguminous plants with conidia of *B. fabae*, these included *V. faba* L., *Pisum sativum* L., *P. sativum* var. *arvense* Poir, and *Vicia sativa* L. Other recorded hosts of *B. fabae* include *Phaseolus vulgaris* (Ellis and Waller 1974b).

4.3. Control Methods

Practices that have been effective in crop canopy management can be used to avoid the creation of a microclimate which encourages disease epidemics (Bretag and Materne 1998a). Practices that delay or avoid the formation of a dense canopy include the adjustment of sowing dates and rates, use of wider row spacing to increase air flow, weed control and optimum fertiliser use, particularly avoiding high nitrogen levels (Bayaa and Erskine, 1998; Lindbeck et al. 2002). A program

of stubble reduction may also be undertaken by grazing, burning or burying, to reduce the carryover of infected stubble into the following season. In addition, potential alternate host plants can be controlled to reduce the early build up of disease inoculum (Lindbeck et al. 2002). Lentils should also not be grown adjacent or into a lentil, faba bean, chickpea, vetch or lathyrus stubble (Lindbeck et al. 2002).

Farmers can reduce the risk of seedling blight and disease carry-over by retaining seed only from disease free crops for sowing the following year, and using seed with less than 5% infection. Seed treatments with fungicides such as benomyl, carboxin, chlorothalonil or thiabendazole can reduce seed-borne inoculum levels (Bayaa and Erskine 1998, Bretag and Materne 1998b, Lindbeck et al. 2002, Morrall, 1997). Foliar fungicides are recommended in Australia for control of botrytis grey mould in lentil crops (Lindbeck et al. 2002, 2003); however, Bayaa and Erskine (1998) stated that fungicide control for grey mould in lentil was uneconomic. Carbendazim, chlorothalonil, mancozeb and procymidone are the products widely used in Australia (Lindbeck et al. 2002). Iqbal et al. (1992) evaluated 14 fungicides and found that benomyl, thiabendazole and tridemorph were the most effective against *B. cinerea*.

Resistance to botrytis grey mould is poorly understood, but requires a better understanding to enable different sources of resistance to be utilised and subsequent pyramiding of resistance genes (Tivoli et al. 2006). Resistant lentil germplasm has been identified in Australia (Bretag and Materne 1999, Lindbeck et al. 2003), Canada (Kuchuran et al. 2003), Nepal (Karki, 1993) and Pakistan (Erskine et al. 1994, Tufail et al. 1993). Testing in Australia has found variability within Australian lentil germplasm for resistance to botrytis grey mould (Bretag and Materne 1999) and a breeding program to improve the resistance to the disease is currently underway (Lindbeck et al. 2003). The lentil variety 'Nipper' was released in 2006 from the Australian lentil breeding program with resistance to both botrytis grey mould and ascochyta blight.

5. LENTIL RUST

Rust, caused by *Uromyces viciae-fabae* (Pers.) Schroet, (*Uredinales, Pucciniaceae*) is regarded as the most important foliar disease of lentil (Figure 1b) (Erskine et al. 1994). Complete crop failures can occur due to this disease (Beniwal et al. 1993). Rust of lentil is widespread globally, but is considered to be a production problem in Algeria, Bangladesh, Canada, Ethiopia, India, Italy, Morocco, Pakistan, Nepal, Syria and Turkey (Erskine et al. 1994). The disease also occurs widely in South America including Argentina, Brazil, Chile, Colombia, Ecuador, and Peru (Bascur 1993).

Rust is an autoecious fungus, completing its life cycle on lentil. The aecia of *U. viciae-fabae* are amphigenous or hyphyllous, usually in groups surrounding the pycnia or sometimes scattered, cupulate, 0.3−0.4 mm diam. The aeciospores are spheroidal, 18−26 μm diam.; wall hyaline, verrucose, 1 μm thick. Uredia are amphigenous and on the petioles and stems, scattered, cinnamon, 0.5−1 mm diam. Uredospores are ellipsoidal or obovoidal 22–28 × 19–22 μm; wall luteous to sienna, very finely echinulate, 1−2.5 μm thick; pores 3–4, equatorial or occasionally

scattered on *Lathyrus*. Telia are like the uredia but black and larger: 1–2 mm diam. Teliospores are ellipsoidal, obovoidal or cylindrical, rounded or subacute above, 25–40 × 18–26 μm; wall chestnut, smooth, 1–2 μm thick at the sides, 5–12 μm thick above; pedicels sienna to luteous, up to 100 μm long. (Laundon and Waterson 1965).

5.1. Symptoms

Rust starts with the formation of yellowish-white pycnidia and aecial cups on the lower surface of leaflets and on pods, singly or in small groups in a circular form (Agrawal et al. 1993). Later, brown uredial pustules emerge on either surface of leaflets, stem and pods (Figure 1b). Pustules are oval to circular and up to 1 mm in diameter. They may coalesce to form larger pustules (Bayaa and Erskine 1998). The telia, which are formed late in the season, are dark brown to black, elongated and present mainly on branches and stems. In severe infections leaves are shed and plants dry prematurely (Bakr 1993), the affected plant dries without forming any seeds in pods or with small shriveled seeds. The plant has a dark brown to blackish appearance, visible in affected patches of the paddock or in the whole paddock if totally infected (Beniwal et al. 1993).

5.2. Epidemiology

The disease generally starts from low-lying patches in the paddock and radiates towards the border (Bayaa and Erskine 1998). Lentil seed may be contaminated with pieces of rust-infected leaf, stem and pericarps, which can act as primary inoculum for the recurrence of the disease in most years (Khare 1981, Agrawal et al. 1993). Rust may also perpetuate on weed hosts from where it may infect lentil crops by windborne teliospores. High humidity, cloudy or drizzly weather with temperatures 20 to 22 °C favour disease development (Agrawal et al. 1993). The disease generally occurs during the flowering /early podding stage. Aeciospores germinate at 17−22 °C and infect other plants forming either secondary aecia at temperatures of 17 − 22 °C or uredia at 25 °C. Uredosori develop later in the season and are rapidly followed by telia (Beniwal et al. 1993). After harvest, aecia and uredia present on lentil trash die out, but teliospores tolerate high temperatures and allow the fungus to survive the summer. At lower temperatures, uredospores could be an important means of survival (Bayaa and Erskine 1998). Uredomycelium is highly resistant to heat and sunlight and is probably important for continued development and survival of rust in hot, dry conditions. The predominant form of survival will vary with the environment and location (Bayaa and Erskine 1998). Teliospores germinate at 17–22 °C without a resting period and cause new outbreaks of the disease each season.

There are 70 recorded hosts of *U. viciae-fabae* including lentil, chickpea, field pea, *Lathyrus* spp and *Vicia* spp. (Parry and Freeman 2001). Degrees of host specialisation and pathogenic variability do exist within populations of *U. viciae-fabae* worldwide. Much research has been performed regarding race identification

within *U. viciae-fabae* over many years with conflicting outcomes regarding the suggestion of forma speciales within the species.

5.3. Control Methods

Cultural control methods currently recommended for control of *U. viciae-fabae* include: control of volunteer plants over summer; isolation of new season crops from old host crop stubbles (MacLeod 1999) and destruction of old lentil stubbles (Prasada and Verma 1948). Early studies on the control of lentil rust in India found seed treated with Agrosan (phenylmercury acetate) to control seed-borne inoculum (Prasada and Verma 1948). Singh (1985) found Vigil (diclobutrazole), applied as a seed dressing prevented the appearance of *U. viciae-fabae* up to 70 days following inoculation with uredospores; bayleton (triadimefon) prevented disease appearance up to 40 days post inoculation and the untreated control was severely infected with rust 35 days after inoculation. Experiments investigating the use of foliar fungicides for rust control by Agarwal et al. (1976) found Hexaferb (Ferric dimethyldithiocarbamate) and Dithane M-45 to give the best control of *U. viciae-fabae* in experimental plots at Jabalpur, India. In addition, Dithane M-45 also increased plot yield by 82% and grain weight by 24% when compared to the untreated control. The use of host plant resistance is the best means of rust control (Bayaa and Erskine 1998). Genetic differences among genotypes and sources of resistance have been reported worldwide, with several rust resistant lines available. Resistance to rust is reported to be controlled by a single dominant gene (Sinha and Yadav 1989). Studies in factors influencing the mechanism of resistance to rust in lentil (Reddy and Khare 1984) reported that resistant cultivars contained more leaf surface wax, P, K, S, Zn, Fe, Cu levels of phenols than susceptible cultivars which had higher levels of amino acids, N, Mn, and sugars. Structurally there were no significant differences found between resistant and susceptible cultivars.

6. STEMPHYLIUM BLIGHT

Stemphylium blight of lentil is caused by the pathogen, *Stemphylium botryosum* Wallr (*Pleosporales, Pleosporaceae*) (teleomorph: *Pleospora herbarum* (Fr) Rab.). The disease has been reported on lentil from Bangladesh (Bakr 1993), Canada (Morrall 2003), Egypt, Syria (Bayaa and Erskine 1998) and the USA (Wilson and Brandsberg 1965). The disease has the potential to cause yield losses of up to 62% under conducive conditions (Figure 1c) (Bakr 1993).

Conidiophores of *S. botryosum* have 1–7 septate, 20–72 × 4–6 μm, pale brown to brown, with a swollen apical sporogenous cell 7–11 μm diam., and slightly roughened toward the apex. They possess a single apical pore 5–8 μm diam. Conidia are oblong, olive to brown, ovoid to subdoliiform, occasionally constricted at 1–3 transverse septa and at the 1–3 longitudinal septa if complete, 19.5 × 28.5 μm with a single basal pore 8 μm diam. and a roughened outer wall. Ascostromata are scattered, immersed to erumpent in the tissue of the host, 100–500 μm in diam. Asci are 90–250 × 20–50 μm containing eight ascospores, cylindrical to slightly

club shaped. Ascospores are light to yellow brown, ellipsoid to club shaped with 7 septate, slightly constricted at the three primary transverse septa, muriform and 26–50 × 10–20 μm (Booth and Pirozynski 1967).

6.1. Symptoms

Symptoms of stemphylium blight start with the appearance of small pin-headed light brown to tan coloured spots on leaflets. Under ideal conditions the small spots enlarge rapidly, covering the entire leaflet surface within a 2–3 day period. The infected tissue appears light cream in colour, often with angular patterns of lighter and darker areas that spread across, or long, the entire leaflet (Morrall 2003; Figure 1c). The affected foliage and stems gradually turn dull yellow, giving a blighted appearance to the crop (Bakr 1993). The infected leaves can be abscised rapidly, leaving only the terminal leaflets on the stems. The stems bend down, dry and gradually turn ashy white, but pods remain green. White mycelial growth can sometimes be seen on the infected stems (Bakr 1993).

6.2. Epidemiology

Important sources of *S. botryosum* inoculum include infected crop debris and infected seed. Infected crop debris can be a source of primary inoculum in the form of air-borne ascospores or as resting mycelium, based on the studies of the pathogen on other host crops such as alfalfa (Gilchrist 1990). *Stemphylium botryosum* is known to be carried on seed (Booth and Pirozynski 1967) and *Stemphylium* spp. has been isolated off lentil seed in Australia (Nasir and Bretag 1997), but the significance of seed-borne *S. botryosum* inoculum on disease initiation in lentil is not clearly understood (Mwakutuya 2006). Bakr (1993) has reported from Bangladesh that the pathogen commences infection when the ambient night temperature remains above 8°C, and the mean day temperature exceeds 22 °C. The RH inside the crop canopy must also reach 94%. In India, Singh and Singh (1993) found that an average mean temperature of 18 °C ± 2 °C and RH of 85−90% in the morning was favourable for disease development and spread. Most recently, in Canada Mwakutuya (2006) found that symptom development of *S. botryosum* was optimised after 48 h of leaf wetness at temperatures above 25 °C. The host range of *S. botryosum* is wide and includes a large number of ornamental, horticultural and field crop species. These include lentil (Bakr 1993), lupin (Tate 1970), tomato (Bashi and Rotem 1975) spinach (Koike et al. 2001), alfalfa, clover (Smith 1940), lettuce (Tate 1970), apple, onion and gladiolus (Booth and Pirozynski 1967).

6.3. Control Methods

There is little published information available regarding cultural control methods for *S. botryosum* in lentil. Being a stubble-borne disease strategies such as destruction

of old crop residues, and crop rotation would assist in decreasing potential inoculum sources. In Bangladesh, delayed sowing was found to significantly decrease the incidence of stemphylium blight in lentil, but later sowing resulted in reduced crop yields and heavy infection by *U. viciae-fabae* (Bakr 1993). Foliar fungicides have been found to be effective in the management of stemphylium blight. In Bangladesh the application of Roval 50 WP was found to effectively control the disease when applied three times at weekly intervals starting from the initiation of the disease (Bakr 1993). In other horticultural crops, such as asparagus and garlic, *Stemphylium* spp. has been successfully controlled using chlorothalonil (Meyer et al. 2000), tebuconazole and procymidone (Basallote-Ureba et al. 1998). Sources of host plant resistance have been identified in screening nurseries in Bangladesh. The resistant varieties 'Barimasur 3' and 'Barimasur 4' were released with resistance to *S. botryosum* (Sarker et al. 1999a, b). Studies by Chowdhury et al. (1997) found lentil cultivars with resistance to *S. botryosum* had a higher number of epidermal hairs, thicker cuticle, thicker epidermal cell layer and thicker cortical layers. In addition, resistant lines were also found to have fewer stomata than susceptible cultivars.

7. ANTHRACNOSE

Anthracnose is an important disease of lentil, caused by *Colletotrichum truncatum* (Schwein.) Andrus and Moore and has been reported from Bangladesh, Canada, Ethiopia, Morocco, Syria and Canada (Anderson et al. 2000; Kaiser et al. 1998). Although *C. truncatum* has not been recorded as a pathogen on lentil in Australia, isolates have been recorded from soybean (*Glycine max*), *Xanthium occidentale* and peanut (*Arachis hypogaea*) growing in northern Australia. These non-host isolates were shown to be genetically different to the lentil infecting isolates from Canada using molecular markers and morphological descriptors (Ford et al. 2004). However, under optimal inoculation conditions the soybean isolate can infect lentil and faba bean leaves and stems. Further gene sequencing and pathogenicity testing of the Australian isolates of *C. truncatum* on lentil is required to validate the taxonomy of this group of isolates. In the mean time, the Australian lentil industry remains under threat from this exotic disease, which causes severe yield and seed quality loss under epidemic conditions.

In culture, the mycelium of *C. truncatum* growing on PDA at 25 °C is dark brown to black in colour with setae being rare. The conidia are ellipsoidal, hyaline and aseptate with one rounded and one pointed end and are 16.0–20.0 × 3.0–5.0 μm in size. Setae are generally acicular, most swollen at the base, tapered to the apex and comprised one or two septa. Mycelial appressoria and appressoria are produced directly from the germ tube, are generally brown, clavate and occasionally irregular. No teleomorph has been found in the wild (Kaiser et al. 1998) however, the teleomorph was recently induced under laboratory conditions and named *Glomerella truncate* (Armstrong-Cho and Banniza, 2006).

7.1. Symptoms

Irregularly shaped, light brown necrotic lesions start to develop on lower stems and gradually increase in number and size until they coalesce and give the stems a blackish brown appearance. Lesions on leaves are circular with few acervuli in the middle of each lesion and premature leaf drop begins at early flowering. Conidia form in acervuli on infected plants, and secondary spread of conidia to neighboring plants occurs by rain splash. The fungus penetrates the vascular tissue, which results in plant wilting, and large brown patches of dying plants become evident in the field after flowering.

7.2. Epidemiology

The disease is favored by high humidity and temperatures of 25–30°C, and is seed borne but has not been shown to be transmitted from seed to seedling. The pathogen is capable of surviving for up to four years as microsclerotia in crop residue and may become active again when in contact with fresh host tissue, spreading as conidia with rain splash and on plant debris through wind dispersal between crops (Buchwaldt et al. 1996).

A study on the infection process of leaves inoculated with a spore suspension, by Chongo et al. (2002) found that in the initial infection phase, conidia germinated within 3–6 hours after infection (hai) and formed appressoria at 6–12 hai. By 24 hai infection hyphae infected epidermal cells inter- and intra-cellularly. Differential host cell reaction was observed by the resistant cultivar 2 to 3 days after infection. Hyphal spread was slower and phenolic compounds accumulated more quickly in the resistant line, resulting in fewer, smaller lesions than in the susceptible cultivar.

7.3. Control Methods

The current disease management practices are based primarily on application of foliar fungicides such as chlorothalonil or benomyl (Chongo et al. 2002). However, seed treatment with fungicides such as benomyl or thiabendazole provides complete control of the seed-borne fungus. Breeding for resistance has suffered from a lack of highly resistant germplasm to include in breeding programs. In Canada, Buchwaldt et al. (2004) found only 16 out of 1,771 accessions of lentil to contain resistance to anthracnose after field and glasshouse screening. As well two pathotypes of *C. truncatum* were identified with the Ct0 pathotype isolated more frequently from commercial seed samples than the Ctl pathotype, although both pathotypes were isolated with similar frequency from plants in commercial fields planted to susceptible cultivars. Pathotype Ct0, to which no resistance has yet been identified, presents a high risk to lentil production in Canada and potentially worldwide.

PCR-based diagnostics tests have been developed to detect the pathogen in plant tissues (Ford et al. 2004) and are a valuable and reliable alternative to conventional seed health testing methods. These tests can be applied directly to suspect infected

tissues taken from the field, to identify the pathogen much faster and potentially more accurately than traditional culturing techniques.

8. VIRUSES

Lentil plants can be infected by a range of viruses but generally the affect on yield is not as great as that caused by fungal pathogens (Beniwal et al. 1993). Viruses tend to be transmitted by aphids and/or seed infection thus controlling the insect vector, planting disease-free seed and use of resistant cultivars will aid in controlling these diseases. The following viruses of lentil are found in the major lentil growing regions of the world.

Lentil yellows disease is caused by several related luteoviruses such as bean leaf roll virus (BLRV), beet western yellows virus (BWYV), or subterranean clover red leaf virus (SCRLV). BLRV was first reported in Australia in 1999 (Freeman, unpublished) and BWYV was recently reported in Iran (Makkouk et al. 2001). The causal viruses are transmitted in a persistent manner by aphids, but not by seed. Epidemic spread of this disease is always associated with high aphid vector populations. The initial symptoms on leaves of virus infected lentil plants show interveinal chlorosis, which intensifies with time until the whole leaf becomes yellow. Other symptoms include leaf rolling, reduction in leaf size and significant reduction in pod setting.

Bean yellow mosaic is caused by the bean yellow mosaic virus (BYMV) which belongs to the potyvirus group. The virus has a wide host range and is transmitted through sap and by aphids in the nonpersistent manner. Transmission of the virus in lentil seed has not been reported. Leaf symptoms of infected plants include mild mosaic followed by leaf narrowing. The new growth of leaves from leaf axils are narrow, elongated and light green. Early infections adversely affect plant growth and yield (Beniwal et al. 1993).

Pea seed borne mosaic is caused by the pea seedborne mosaic virus (PSbMV) which belongs to the potyvirus group. The virus is seed borne in lentil, but also infects faba beans and field peas (Makkouk et al. 1993; Latham and Jones 2001) and is transmitted in a nonpersistant manner by aphids. The disease is characterized by a mild mosaic and malformation of leaves. The affected plants show little stunting and twisting of stems. Seeds from the affected plants are smaller than normal and deformed (Beniwal et al. 1993).

Cucumber mosaic virus (CMV) belongs to the cucumovirus group. CMV has a very wide host range and can be transmitted through sap, by aphid species in a nonpersistent manner and infected seed (Fletcher et al. 1999). Leaf symptoms include vein clearing followed by mild systemic mottle. Infected plants show stunting as the disease advances.

Alfalfa mosaic virus (AMV) is an alfamovirus and has a wide host range. The virus is spread by aphid species as well as being seed transmitted (Latham and Jones 2001). In areas where large aphid populations occur, crop losses can be high due to reduced plant growth and seed yield. Symptoms can be variable depending on the stage of growth at

infection; environmental conditions and the host however, in lentil plants the leaves become twisted, deformed and stunted leading to necrotic tip growth.

Broad bean stain virus (BBSV) is a comovirus known to occur in Europe, North Africa and West Asia however, this virus has yet to be detected in Australia. BBSV naturally infects faba bean, dry pea and vetch and is transmitted through sap, seed and by weevils. The disease is characterized by a mild mottling on the leaves, which is not easily recognizable because of the small size of the lentil leaf. The affected plants are reduced in growth, which is easily recognized, especially when they are compared with healthy plants. Seeds from infected plants occasionally show dark staining on the seed coat (Beniwal et al. 1993).

9. MINOR DISEASES

Other minor diseases of lentil include White mould caused by *Sclerotinia sclerotiorum* (Lib.) de Bary that occurs from early flowering to pod setting where conditions are wet and cool (Figure 1e). The pathogen produces sclerotia that can survive in the soil until cool moist soil conditions exist that induce the sclerotia to germinate and produce apothecia that release ascospores to reinfect lentil crops. Other root and basal stem fungal diseases include collar rot caused by *Sclerotium rolfsii* Sacc., *Rhizoctonia solani* Kuhn; and dry root rot caused by *Rhizoctonia bataticola* (Taub.) Butler, in which the perfect stage of the fungus is *Macrophomina phaseolina* (Tassi) Goid. Also Pythium seedling and root caused by *Pythium aphanidermatum* (Edson) Fitzp. and *P. ultimum* Trow; and, black root rot caused by *Fusarium solani* (Mart.) Appel & Wr.

Minor foliar fungal diseases include Powdery mildew caused by *Erysiphe polygoni* DC (conidial stage, *Oidium* sp.) that has been recorded in Cyprus, Ethiopia, India, Siberia, Sudan, Syria, Tanzania and the former USSR; Downy mildew caused by *Peronospora lentis* Gäumannn from Egypt, France, India and Syria; Alternaria blight caused by *Alternaria alternata* (Fr.) Keissler that has limited range in Egypt, Ethiopia and India. The following fungi have been recorded as pathogens on lentil: *Cercospora lensi*,*Cladosporium herbarum* (Pers.) Link, *Cylindrosporium* sp., *Helminthosporium* sp., *Phoma medicaginis* (Malbr. & Roum.), *Septoria* sp., *Stemphylium hotryosum* Walr.

REFERENCES

Agarwal SC, Prasad KVV (1997) Diseases of lentil. Science Publishers Inc., USA.

Agarwal SC, Khare, MN, Agarwal PS (1976) Control of lentil rust by use of fungicides. Indian Phytopathology 29:90–91

Agarwal SC, Singh K, Lal SS (1993) Plant Protection of Lentil in India. In: Lentil in South Asia (pp 147–165). Erskine W and Saxena MC (eds), International Centre for Agricultural Research in the Dry Areas, Aleppo, Syria.

Ahmed S, Beniwal SPS (1991) Ascochyta blight of lentil and its control in Ethiopia. Tropical Pest Management 37: 3668–373.

Ahmed S, Morrall RAA, Sheard JW (1996) Virulence of *Ascochyta fabae* f.sp. *lentis* on lentil. Canadian Journal of Plant Pathology 18: 354–361.

Anderson KL, Buchwaldt L, Chongo G, Gossen BD, Morrall RAA, Pearse PG (2000) Diseases of lentil in Saskatchewan 1999. Canadian Plant Disease Survey 80: 96–98.

Armstrong-Cho CL, Banniza S (2006) *Glomerella truncata* sp. nov., the teleomorph of *Colletotrichum truncatum*. Mycological Research 110: 951–956.

Bailey KL, Gossen BD, Derksen DA, Watson PR (2000) Impact of agronomic practices and environment on diseases of wheat and lentil in southeastern Saskatchewan. Canadian Journal of Plant Science 80(4): 917–927.

Bakr MA (1993) Plant Protection of Lentil in Bangladesh. In: Lentil in South Asia (pp 177–186). Erskine W and Saxena MC (eds), International Centre for Agricultural Research in the Dry Areas, Aleppo, Syria.

Banniza S, Vandenberg A (2006) Investigations into the population structure of *Ascochyta lentis* in western Canada. Proceedings of the 1st International Ascochyta workshop on grain legumes. Le Tronchet, France.

Basallote-Ureba M, Prados-Ligero AM, Melero-Vara JM (1998) Effectiveness of tebuconazole and procymidone in the control of *Stemphylium* leaf spots in garlic. Crop Protection 17(6): 491–495.

Bascur GB (1993) Lentil and Faba Bean in Latin America: Their Importance, Limiting Factors and Research. ICRDA, Aleppo, Syria.

Bashi E, Rotem (1975) Sporulation of *Stemphylium botryosum* f. sp. *lycopersici* in tomatoes and of *Alternaria porri* f. sp. *solani* in potatoes under alternating wet-dry regimes. Phytopathology 65: 532–535.

Bashir M, Malik BA (1988) Diseases of major pulse crops in Pakistan: a review. Tropical Pest Management 34: 309–314.

Bayaa B, Erskine W (1998) Diseases of lentils. In: The Pathology of Food and Pasture Legumes (pp. 423–471) Allen DJ and Lenné JM (eds.) CAB International and ICRISAT, Wallingford, UK.

Bayaa B, Erskine W, Singh M (1997) Screening lentil for resistance to Fuarium wilt: methodology and sources of resistance. Euphytica 98:69–74.

Beauchamp CJ, Morrall RAA, Slinkard AE (1986) The potential for control of ascochyta blight of lentil with foliar-applied fungicides. Canadian Journal of Plant Pathology 8: 254–259.

Beniwal SPS, Seid A, Tadesse N (1989) Effect of sun drying of lentil seeds on the control of seedborne *Ascochyta lentis*. LENS Newsletter 16: 26–28.

Beniwal SPS, Bayaa B, Weigand S, Makkouk K, Saxena MC (1993) Field Guide to Lentil Diseases and insect Pests. International Centre for Agricultural Research in the Dry Areas, Aleppo, Syria, 106pp.

Bondartzeva-Monteverde VN, Vassilievsky NI (1940) A contribution to the biology and morphology of some species of Ascochyta on Leguminosae. *Acta Institute of Botany Academic Science USSR* 1938, Ser II, pp. 345–376 (cited by Agrawal and Prasad, 1997).

Booth C, Pirozynski KA (1967) *Pleospora herbarum*. C.M.I. Descriptions of Pathogenic Fungi and Bacteria, No. 150.

Bretag TW (1989) Evaluation of fungicides for the control of ascochyta blight in lentils. Tests of agrochemicals and cultivars. Annals of Applied Biology (Suppl.): 44–45.

Bretag TW, Materne MA (1998a) Lentil: Effect of seeding rate on foliar disease and grain yield. In: 'Summary of Field Research in the Wimmera 1998'. (Ed. J Hyett) pp. 35–36. (Department of Natural Resources and Environment: Horsham, Victoria).

Bretag TW, Materne MA (1998b) Lentil: Evaluation of 15 lines for resistance to botrytis grey mould. In: 'Summay of Field Research in the Wimmera 1998'. (Ed. J Hyett) pp. 37–38. (Department of Natural Resources and Environment: Horsham, Victoria).

Bretag TW, Materne MA (1999) Evaluation of 36 lentil lines for resistance to ascochyta blight and botrytis grey mould. In: 'Summary of Field Research in the Wimmera 1999'. (Ed. J Hyett) pp. 38–40. (Department of Natural Resources and Environment: Horsham, Victoria).

Brouwer JB, Bretag TW, Materne MA (1995) Coordinated improvement program for Australian lentils. Proceeding of 2nd European Conference on Grain Legumes, Copenhagen, Denmark. p. 25.

Brouwer JB, Sharma B, Malik BA, Hill GD (2000) Region 6: Asia-Pacific: Meeting the challenge. In: Linking research and marketing opportunities for pulses in the 21st century. Proceedings of the third international food legumes research conference. Current Plant science and biotechnology in agriculture, Vol 34, pp 115–129 (Ed R. Knight) Kluwer Academic Publishers.

Buchwaldt L, Morrall RAA, Chongo G, Bernier CC (1996) Windborne dispersal of *Colletotrichum truncatum* and survival in infested lentil debris. Phytopathology 86: 1193–1198.

Buchwaldt L, Anderson KL, Morrall RAA, Gossen BD, Bernier CC (2004) Identification of lentil germplasm resistant to *Colletotrichum truncatum* and characterization of two pathogen races. Phytopathology 94: 236–243.

Carrouee B, Gent GP, Summerfield RJ (2000) Production and uses of grain legumes in the European Union. In: Linking research and marketing opportunities for pulses in the 21st century. Proceedings of the third international food legumes research conference. Current Plant science and biotechnology in agriculture, Vol 34, pp 79–97 (Ed R. Knight) Kluwer Academic Publishers.

Chaudhary RG, Amarjit K (2002) Wilt disease as a cause of shift from lentil cultivation in Sangod Tehsil of Kota, Rajasthan. Indian Journal of Pulses Research 15: 193–194.

Chongo G, Gossen BD, Bernier CC (2002) Infection by *Colletotrichum truncatum* in resistant and susceptible lentil genotypes. Canadian Journal of Plant Pathology 24: 81–85.

Chowdhury AM, Ahmed A, Zaman M (1997) Studies on the defence structural factors of some susceptible and resistant varieties of lentil plants. Journal of Mycopathological Research 35: 35–39.

Cromey M G, Mulholland RI, Russell AC, Jermyn WA (1987) *Ascochyta fabae* f. sp. *lentis* on lentil in New Zealand. New Zealand Journal of Experimental Agriculture 15: 235–238.

Davidson JA, Pande S, Bretag TW, Lindbeck KD, Krishna-Kishore G (2004) Biology and control of*Botrytis* spp. in legume crops. In: Botrytis: Biology, Pathology and Control, (pp 295–318) edited by Y. Elad, B. Williamson, P. Tudzynski and N. Delen. Dordrecht, The Netherlands: Kluwer

De RK, Ali SS, Dwivedi, RP (2001) Effect of interaction between *Fusarium oxysporum* f. sp. *lentis* and *Meloidogyne javanica* on lentil. Indian Journal of Pulses Research 14: 71–73.

Diaz D, Tello JC (1994) A fungal inventory of lentil seeds (*Lens culinaris* Medik.) harvested in Castilla-La Mancha. Boletin de Sanidad Vegetal, Plagas 20(4): 857–870 (abstr).

Domsch KH, Gams W, Anderson TH (1980) Compendium of Soil Fungi. Academic Press, London

El-Hassan SA, Gowen SR (2006) Formulation and delivery of the bacterial antagonist *Bacillus subtilis* for management of lentil vascular wilt caused by *Fusarium oxysporum* f. sp. *lentis*. Journal of Phytopathology 154: 148–155.

Ellis MB, Waller JM (1974a) *Sclerotinia fuckelina,* C.M.I. Descriptions of Pathogenic Fungi and Bacteria, No. 431

Ellis MB, Waller JM (1974b) *Botrytis fabae*. C.M.I. Descriptions of Pathogenic Fungi and Bacteria, No. 432.

Erskine W, Tufail M, Russell A, Tyagi MC, Rahman MM, Saxena MC (1994) Current and future strategies in breeding lentil for resistance to biotic and abiotic stresses. Euphytica 73: 127–135.

Eujayl I, Erskine W, Bayaa B, Baum M, Pehu, E (1998) Fusarium vascular wilt in lentil: Inheritance and identification of DNA markers for resistance. Plant Breeding 117: 497–499.

Fletcher JD, Russell AC, Butler RC (1999) Seed-borne cucumber mosaic virus in New Zealand lentil crops: yield effects and disease incidence. New Zealand Journal of Crop and Horticultural Science 27: 197–204.

Ford R, Pang ECK, Taylor PWJ (1999) Genetics of resistance to ascochyta blight (*Ascochyta lentis*) of lentil and identification of closely linked molecular markers. Theoretical and Applied Genetics 98: 93–98.

Ford R, Banniza S, Photitia W, Taylor PWJ (2004) Morphological and molecular discrimination of *Colletotrichum truncatum* causing anthracnose on lentil in Canada Australasian Plant Pathology 33: 559–569.

France I A, Sepulveda P, Tay UJ (1988) Identification of *Botrytis cinerea* Pers. on lentil (*Lens culinaris* Med.). Agricultura Tecnica Santiago 48(2): 158–160 (abstr).

Gilchrist DG (1990) Stemphylium Leaf Spot. In: Compendium of Alfalfa Diseases (2nd ed) (pp 17–20), D Stuteville and D Erwin (eds), APS Press.

Gossen BD, Morrall RAA (1983) Effect of ascochyta blight on seed yield and quality of lentils. Canadian Journal of Plant Pathology 5: 168–173.

Gowda CLL, Kaul AK (1982) Lentil. In: Pulses in Bangladesh (pp 51–106). Bangladesh Agricultural Research Institute, Joydebpur, Dhaka and Food and Agriculture Organisation of the United Nations.

Hamdi A, Hassanein AM (1996) Survey of Fungal Diseases in North Egypt. LENS Newsletter 23(1/2): 52–56.

Harrison JG (1979) Overwintering of *Botrytis fabae*. Transactions of the British Mycological Society 72(3): 389–394.

Harrison JG (1980) Effects of environmental factors on growth of lesions on field bean leaves infected by *Botrytis fabae*. Annals of Applied Biology 95: 53–61.

Iqbal SM, Hussain S, Malik BA (1992) *In vitro* evaluation of fungicides against *Botrytis cinerea* of lentil. Lens Newsletter 19: 49–51.

Jarvis WR (1980a) Taxonomy. In: The Biology of Botrytis. J. R. Coley-Smith, K. Verhoeff and W. R. Jarvis, Academic Press: 1–17.

Jarvis WR (1980b) Epidemiology. In: The Biology of Botrytis. J. R. Coley-Smith, K. Verhoeff and W. R. Jarvis (eds), Academic Press: 219–250

Kaiser WJ (1992) Fungi associated with the seeds of commercial lentils from the U.S. Pacific Northwest. Plant Disease 76(6): 605–610.

Kaiser WJ (1997) Intra- and international spread of ascochyta pathogens of chickpea, faba bean and lentil. Canadian Journal of Plant Pathology 19: 215–224.

Kaiser WJ, Hannan RM (1986) Incidence of seedborne Ascochyta lentis in lentil germplasm. Phytopathology 76: 355–360.

Kaiser WJ, Hannan RM (1987) Seed treatment fungicides for control of seedborne *Ascochyta lentis* on lentil. Plant Disease 71: 58–62.

Kaiser WJ, Hellier BC (1993) *Didymella* sp. The teleomorph of *Ascochyta lentis* on lentil straw. *Phytopathology* 83: 692. (*Abstr.*).

Kaiser WJ, Wang BC, Rogers JD (1997) *Ascochyta fabae* and *A. lentis*: Host specificity, teleomorphs (*Didymella*), Hybrid analysis, and taxonomic status. *Plant Disease* 81: 809–816.

Kaiser WJ, Mihov M, Muehlbauer FJ, Hannan RM (1998) First report of anthracnose of lentil incited by *Colletotrichum truncatum* in Bulgaria. Plant Disease. 82: 128.

Kaiser WJ, Stanwood PC, Hannan RM (1989) Survival and pathogenicity of Ascochyta fabae f. sp. lentis in lentil seeds after storage for four years at 20 to −196 °C. Plant Disease 73: 762–764.

Karki PB (1993) Plant Protection of Lentil in Nepal. In: Erskine W and Saxena MC (eds.) Lentil in South Asia. (pp. 187–193) ICARDA, Aleppo, Syria.

Khare MN (1981) Diseases of Lentils, *In*: Lentils (Eds. C Webb, G Hawtin), Farnham Royal, U.K., pp 163–172.

Khare M N (1980) Wilt of Lentil. Jawaharlal Nehru Krishi Vishwa Vidyalaya, Jabalpur, M.P India 155 pp.

Knights EJ (1987) Lentil: A potential winter grain legume crop for temperate Australia. Journal of the Australian Institute of Agricultural Science 53: 271–280.

Koike ST, Henderson DM, Butler EE (2001) Leaf spot disease of spinach in California caused by *Stemphylium botryosum*. Plant Disease 85: 126–130.

Kuchuran M, Banniza S, Vandenberg B (2003) Evaluation of lentil varieties for resistance to Botrytis gray mould. In: Proceedings of Pulse Field Days, 6–7 January 2003, Saskatoon, Canada (p 89).

Latham LJ, Jones RAC (2001) Incidence of virus infection in experimental plots, commercial crops, and seed stocks of cool season crop legumes. Australian Journal of Agricultural Research 52: 397–413.

Laundon GF, Waterson JM (1965) *Uromyces viciae-fabae*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 60 Commonwealth Mycological Institute, Kew, UK.

Lindbeck KD, Bretag TW, Materne MA (2003) Breeding for resistance to *Botrytis fabae* in Australian lentils. In: Proceedings of 8th International Congress of Plant Pathology, Christchurch, New Zealand, p 291.

Lindbeck KD Materne MA, Davidson JA, McMurray L, Panagiotopoulos K (2002) Lentil Disease Management Strategy for Southern Region GRDC. Pulse Australia and state departments extension article.

MacLeod W (1999) Faba bean: Rust disease. Agriculture Western Australia Farmnote 114/96.

Makkouk KM, Kumari SG, Bos L (1993) Pea seed-borne mosaic virus: occurrence in faba bean (*Vicia faba*) and lentil (*Lens culinaris*) in West Asia and North Africa, and further information on

host range, transmission characteristics, and purification. European Journal of Plant Pathology 99: 115–124.

Makkouk KM, Fazlali Y, Kumari YSG, Farzadfar S (2002) First record of Beet western yellows virus, Chickpea chlorotic dwarf virus, Faba bean necrotic yellows virus and Soybean dwarf virus infecting chickpea and lentil crops in Iran. Plant Pathology 51: 387–387.

Meyer MP, Hausbeck MK, Podolsky R (2000) Optimal fungicide management of purple spot of asparagus and impact on yield. Plant Disease 84: 525–530.

Morrall RAA (1997) Evolution of lentil diseases over 25 years in western Canada. Canadian Journal of Plant Pathology 19(2): 197–207.

Morrall RAA (1998) Using thiobendazole to control seed-borne *Ascochyta* in lentil. Canadian journal of Plant Pathology 10: 370 (Abstr).

Morrall RAA (2003) Diseases of Lentil. In: Diseases of Field Crops in Canada (3rd ed) (pp 191–200) (KL Bailey, BD Gossen, RK Gugel and RAA Morrall eds),The Canadian Phytopathological Society.

Morrall RAA, McKenzie DL, Duczek LJ, Verma PR (1972) A qualitative survey of diseases of some specialty crops in Saskatchewan in 1970 and 1971: Sunflower, safflower, buckwheat, lentil, mustards and field pea. Canadian Plant Disease Survey 52(4): 143–148.

Mwakutuya E (2006) Epidemiology of Stemphylium blight on lentil (*Lens culinaris*) in Saskatchewan. MSc Thesis, Department of Plant Sciences, University of Saskatchewan, Saskatoon.

Nair, NG Nadtotchei A (1987) Sclerotia of Botrytis as a Source of Primary Inoculum for Bunch Rot of Grapes in New South Wales. Journal of Phytopathology 119: 42–51.

Nasir M, Bretag TW (1997a) Pathogenic variability in Australian isolates of *Ascochyta lentis*. Australasian Plant Pathology 26: 217–220.

Nasir M, Bretag TW (1997b) Prevalence of *Ascochyta fabae* f.sp. *lentis* on lentil seed from Victoria, Australia. Australasian Plant Pathology 26: 117–120.

Nene YL, Hanounik SB, Qureshi SH, Sen B (1988) Fungal and bacterial foliar diseases of pea, lentil, faba bean and chickpea. In: Summerfield RJ (ed) World crops: cool season food legumes. Kluwer Academic publishers, the Netherlands pp 577–589.

Nguyen TT, Taylor PWJ, Brouwer JB, Pang ECK, Ford R (2001) A novel source of resistance in lentil (*Lens culinaris* ssp. *culinaris*) to ascochyta blight caused by *Ascochyta lentis*. Australasian Plant Pathology 30: 211–215.

Parry R, Freeman A (2001) *Uromyces viciae-faba*. In: Pathogens of the Temperate Pulse *Genera Cicer, Lathyrus, Lens, Lupinus, Pisum*, and *Vicia* Volume 1 : Pathogen Pest Data Sheets. The State of Victoria, Department of Natural Resources and Environment, p 441.

Pedersen EA, Morrall RAA (1994) Effect of cultivar, leaf wetness duration, temperature and growth stage on infection and development of ascochyta blight of lentil. Phytopathology 84: 1024–1030.

Pedersen EA, Morrall RAA, McCartney HA, Fitt BDL (1994) Dispersal of conidia of *Ascochyta fabae* f sp. *lentis* from infected lentil plants by simulated wind and rain. Plant Pathology 43: 50–55.

Prasad R, Verma UN (1948) Studies on lentil rust, *Uromyces fabae* (Pers.) de Bary in India. Indian Phtyopathology 1:142–146.

Rajendra P, Chaudhary CB, Prasad R (1987) Seed-borne mycoflora of lentil. Lens Newsletter 14(1–2): 20–22.

Reddy RR, Khare MN (1984) Further studies on factors influencing the mechanism of resistance to lentil (*Lens culinaris* M.) to rust (*Uromyces fabae* (Pers.) de Bary). LENS Newsletter 11:29–32.

Rewal N, Grewal JS (1989) Effect of temperature, light and relative humidity on conidial germination of three strains of *Botrytis cinerea* infecting chickpea. Indian Phytopathology 42: 79–83.

Roundhill SJ, Fineran BA, Cole ALJ, Ingerfeld M (1995) Structural aspects of ascochyta blight of lentil. Canadian Journal of Botany 73: 485–497.

Sarker A, Kumar J, Rahman MM, Hassan MS, Zaman W, Afzal MA, Murshed ANMM (1999a) Registration of 'Barimasur-3' lentil. Crop Science 39:5, 1536.

Sarker A, Erskine W, Hassan MS, Afzal MA and Murshed ANMM (1999b) Registration of 'Barimasur-4' lentil. Crop Science 39:5, 876.

Singh K (1985) Effect of seed treatment on lentil rust (*Uromyces fabae)* development. LENS Newsletter 12:26–27.

Singh JN, Singh AD (1993) Effect of environment on the development and spread of *Stemphylium* blight on lentil. Indian Phytopathology 46: 252–253

Sinha RP, Yadav BP (1989) Inheritance of resistance to rust in lentil. LENS Newsletter 16:41.

Smith OF (1940) Stemphylium leaf spot of red clover and alfalfa. Journal of Agricultural Research 61: 831–846.

Stoilova T, Chavdarov P (2006) Evaluation of lentil germplasm for disease resistance to Fusarium wilt (*Fusarium oxysporum* f. sp. *lentis*). Journal of Central European Agriculture 7:121–126.

Tate KG (1970) A foliage disease of blue lupin caused by *Stemphylium botryosum* Wallr. New Zealand Journal of Agricultural Research 13: 710–716.

Taylor PWJ, Ford R (2007) Biology of ascochyta blight of cool season food and feed legumes. European Journal of Plant Pathology (In press).

Tivoli B, Baranger A, Avila CM, Banniza S, Barbetti M, Chen W, Davidson J, Lindeck K, Kharrat M, Rubiales D, Sadiki M, Sillero JS, Sweetingham M, Muehlbauer FJ (2006) Screening techniques and sources of resistance to foliar diseases caused by major necrotrophic fungi in grain legumes. Euphytica 147: 223–253.

Tosi L, Cappelli C (2001) First report of *Fusarium oxysporum* f. sp. *lentis* of lentil in Italy. Plant Disease 85: 562.

Tripathi, HS, Rathi YPS (1992) Epidemiology of botrytis gray mold of chickpea. Botrytis Gray Mold of Chickpea: Summary Proceedings of the BARI/ICRISAT Working Group Meeting to Discuss Collaborative Research on Botrytis Gray Mold of Chickpea, Joydebpur, Bangladesh, ICRISAT, Patancheru, Andhra Pradesh, India.

Wilson AR (1937) the chocolate spot disease of beans (*Vicia faba* L.) caused by *Botrytis cinerea* Pers. Annals of Applied Biology 24: 258–288.

Wilson VE, Brandsberg J (1965) Fungi isolated from diseased lentil seedlings in 1963–64. Plant Disease Reporter 49(8): 660–662.

Yu T F (1945) The red-spot disease of broad beans (Vicia faba L.) caused by *Botrytis fabae* Sardina in China. Phytopathology 35(12): 945–954.