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Soilborne Diseases and their Control

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Abstract

Seed and seedling diseases, root rots, and wilts are caused by a number of soilborne fungi, all of which are facultative saprophytes and can survive in soil for long periods in the absence of a susceptible host. In general, these diseases are serious yield constraints where short rotations or monoculture of legume crops are the rule. Seedling diseases and root rots are enhanced by poor seed vigor, poor seedbed preparation, and other biotic and abiotic stresses which predispose the host plant. Control of these diseases requires an integrated approach of genetic resistance/tolerance, cultural practices, appropriate seed treatments, and high seed vigor. The most economical and durable control of Fusarium wilt is to grow resistant varieties. New races of a wilt pathogen have arisen due to increased selection pressure from growing resistant varieties in short rotations but have not outpaced the development of resistant cultivars.

INTRODUCTION

Cool season food legumes, including chickpea (*Cicer arietinum* L.), lentil (*Lens culinaris* Medik.), pea (*Pisum sativum* L.), and lupin (*Lupinus angustifolius* L.), are susceptible to a number of soilborne fungal diseases. Seed and seedling rots are caused primarily by *Pythium* spp. and *Rhizoctonia solani*. Full season root rots and root diseases are caused by *Aphanomyces euteiches, Fusarium solani, Sclerotium rolfsii, Rhizoctonia solani,* and *Pleiochaeta setosa*. Wilt of these crops is caused primarily by various forma specialis of *Fusarium oxysporum*.

SEEDLING DISEASE

Pythium Seed and Seedling Rot

Factors which delay emergence or result in uneven plant stands including: a) cold, wet soil, b) poor seed vigour, and c) herbicide injury, predispose developing plants to seedling disease. Diseases caused by *Pythium* spp. are referred to as seed rot, damping-off, or root rot. Damage due to *Pythium* is more prevalent and severe when soil moisture is high and soil temperatures are in the 10-15°C range. When conditions are favourable for the fungus but less for the host, *Pythium* species can become very pathogenic. However, as the plant matures, *Pythium* attack is usually focused on root tips. Infection takes place when zoospores produce germ tubes or hyphal elements form appressoria, and penetrate the host plant by means of infection pegs (Kraft *et al.,* 1967). According to Stasz *et al.* (1980), there are at least three kinds of genetic resistance in pea to Pythium seed and seedling rot: 1) seeds lose juvenile susceptibility within 48 hrs. after imbibition begins, thus decreasing the number of seeds rotted; 2) peas with round, not wrinkled, seed exude reduced amounts of substances stimulatory to *Pythium*; and 3) peas with pigmented seed coats are nearly immune to *Pythium* due to the presence of fungistatic, phenolic compounds.

Seed quality and vigour have a major influence on *Pythium* seed infection. Seed with poor vigour and/or mechanical damaged seed exude more water soluble and volatile exudates than do high vigour seeds and are

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more stimulatory to *Pythium* germination and infection (Matthews, 1971). In the United Kingdom, seed lots of cool season legumes are categorized based on electroconductivity tests of imbibed seed. Only seeds with relatively low EC readings are recommended for planting in very cold, wet soil where *Pythium* attack is most likely.

Rhizoctonia Seedling Rot

Rhizoctonia solani can survive in field soils for extended periods and can attack legume seedlings when warm, moist conditions are prevalent. For seedling infection to occur, the sclerotium or hyphal fragment germinates and grows up to several millimeters through soil to form an infection cushion on the host surface to penetrate the seedling plant. *Rhizoctonia* invades both inter- and intracellulary, and seedlings become less susceptible with maturity. *Rhizoctonia* is warm temperature dependent, occurring most frequently and severely when surface soil temperatures are in the 24-30°C range and in sandy soils because they warm rapidly. Seedling damping off, caused by *R. solani*, can be especially severe when soil moisture and surface soil organic matter are high and the legume crop is direct drilled into fields with reduced or no tillage.

Seedling hypocotyl and epicotyl symptoms, caused by *R. solani*, appear as water-soaked lesions turning reddish-brown to brown (Flentje & Hagedorn, 1964). The growing tip of a seedling may die as it emerges from the soil. On older plants, reddish brown, sunken lesions may occur on the epicotyl or hypocotyl, sometimes girdling the entire plant, resulting in severe plant stunting or death. *Rhizoctonia solani* (AG4) prefers well-aerated areas at or near the soil surface, so plant parts at or near the soil surface are most vulnerable to attack.

There are no reported varieties of any cool season legumes resistant to *Rhizoctonia*, but pea cultivars with vigorous, thick stems, which emerge rapidly may escape serious damage (McCoy & Kraft, 1984). Rotation with cereals, clean tillage of fields prior to sowing, and fungicidal seed treatment chemicals, such as PCNB or Demosan, provide some protection against *R. solani* (Hagedorn, 1984). Biological seed treatments, such as *Trichoderma viride* and/or *Gliocladium virens*, have also shown promise in protecting against Rhizoctonia seed and seedling rot (Chet & Baker, 1981; Harman *et al.*, 1980).

ROOT ROT

Root rots of cool season legumes are caused by several different soilborne fungal pathogens that produce similar symptoms. These disease complexes can encompass the whole root system and/or extend short distances above the soil surface. All of the pathogens discussed in this paper can be disseminated by water, movement of infected plant debris, or infested soil carried on farm implements. In most cases, where soilborne diseases are severe, a short or no interval between legume crops in a given field is the rule. Crop rotation has historically been a primary means of soilborne disease control (Bruehl, 1986). However, research is often focused on making monoculture or short rotations possible through developing resistant varieties and on biological or chemical control. In the Columbia Basin of Central Washington, some farmers were growing seed and processing peas in a double cropping sequence with sweet corn for processing. These growers received a double income from a given field, but peas were being grown every year. The result has been the development of severe root rot in less than 5 years that in some instances has caused a total loss of the crop. In Western Australia, the population of *Pleiochaeta setosa* spores in soil declines by about 50% every 12 months. Consequently, the longer the break between legume crops the lower the spore numbers and disease risk (Sweetingham, 1996).

Rhizoctonia Root Rot of Lupine

In Western Australia, a wheat-lupin rotation is the preferred rotation. As a result, both Rhizoctonia bare patch (AG8) and a slow-growing binucleate *Rhizoctonia* spp. have become serious problems in lupin production (MacLeod & Sweetingham, 1997). Yellow lupins are resistant to the binucleate form. Affected or disease patches caused by *Rhizoctonia solani* (AG8) and the binucleate *Rhizoctonia* range from 0.3 to 12 m in diameter. Patches caused by the binucleate *Rhizoctonia* are usually not visible before 7 wk after sowing. This is in contrast to Rhizoctonia bare patch (*R. solani* AG8), which is usually visible in lupin crops 4 wk after sowing. Symptoms caused by *R. solani* (AG8) are usually spear-tipped lateral and tap

roots, compared to sloughing of the tap root and pinched-off lateral roots caused by the binucleate *Rhizoctonia* spp.

Rhizoctonia bare patch is most severe in minimum tilled crops and cultivation reduces disease severity (Rovira, 1986). Rhizoctonia bare patch control increased with cultivation depth. However, cultivation does not control hypocotyl rot caused by other strains. Severe outbreaks have been observed following a long pasture phase, which suggests inoculum build-up under clover or annual medics. At sites with a history of severe hypocotyl rot, increased seeding rates are recommended to compensate for losses in stand density. No known resistance to Rhizoctonia bare patch or hypocotyl rot has been reported in lupins.

Pleiochaeta Root Rot and Brown Spot of Lupins

Pleiochaeta setosa (Kirchn.) Hughes is the cause of a brown spot of lupins and is also a virulent and widespread root pathogen of lupin seedlings in Western Australia (Sweetingham, 1989). Pleiochaeta root rot occurs only in fields previously cultivated with lupins where soilborne inoculum exists in sufficient quantities. The severity of Pleiochaeta root rot is greatly reduced as sowing depth increases, due to avoidance of concentrated inoculum on or near the soil surface. Inoculum of *P. setosa* originates from sporulation on lupin leaf litter. Pleiochaeta root rot is more severe where the surface 10-cm of soil dries out shortly after sowing.

Spore numbers are usually high in fields sown to lupins the previous season. Consequently, double cropping of lupins is not a recommended practice. Retaining cereal stubble mulch on the soil surface reduces rain-splash of spores onto the foliage of plants, thus reducing brown spot severity. Seed treatment with fungicides is recommended and can reduce brown spot losses up to 4 wk after sowing. In Western Australia, a sowing depth of 5 cm is recommended for lupin establishment in fields where high concentration of *Pleiochaeta* spores exist (Sweetingham, 1996). Shallower seeding rates lead to increased disease, and planting deeper than 7 cm results in reduced plant stands. The variety 'Myallie' is reported more resistant to Pleiochaeta brown spot than other varieties of narrow-leafed lupin and is recommended for planting in high inoculum fields. However, Myallie is not root rot resistant and cultivars with resistance to Pleiochaeta root rot should be released in the near future (Sweetingham, personal communication).

Aphanomyces Root Rot of Peas

Aphanomyces root rot of pea is the most destructive disease of peas worldwide, most often occurring in poorly drained fields with heavy-textured soils (Hagedorn, 1984). The pathogen, *Aphanomyces euteiches* Drechs., can also infect roots of other legumes such as alfalfa, clover, common bean, lentil, and faba bean (Papavizas & Ayers, 1974). Aphanomyces root rot was first described in 1925 in Wisconsin and has continued to be a serious problem there (Hagedorn, 1984; Papavizas & Ayers, 1974). Since 1985, Aphanomyces root rot has been observed in northern Idaho, in the Palouse and Blue Mountain areas in eastern Washington, and in the Columbia Basin of central Washington.

Where inoculum levels are high, yellowing and stunting are evident soon after emergence, but symptoms can occur at any age given high soil moisture and warm temperatures. Infected cortical tissue is straw coloured and darkens as secondary organisms colonize. When infected plants are pulled from the soil, a strand of vascular tissue is all that remains of the root system. Microscopic observation of infected, cortical tissue reveals typical, thick-walled oospores (25-30 μ diam.), which can survive in soil for years.

There are no economically feasible control practices for Aphanomyces root rot other than to avoid planting a susceptible legume in heavily infested fields (Kraft *et al.*, 1990). Long-term rotations with crops that delay or reduce inoculum build-up, coupled with field indexing to determine field root rot potential, is the only means of control (Hagedorn, 1984; Kraft *et al.*, 1990). Commercial varieties are now being developed that have measurable levels of resistance to avoid severe economic loss in fields moderately infested with *A. euteiches* (Kraft *et al.*, 1995). The development of commercial varieties with resistance to *A. euteiches* is due to public breeding efforts in the last 15 years that have produced resistant germplasm lines approaching a commercial type (Davis *et al.*, 1995; Gritton, 1990; Kraft, 1981, 1989, 1992). However, resistance is not sufficient to withstand adverse environmental conditions and/or increased inoculum levels (Kraft & Boge, 1996).

Green manure plowdown of oats (*Avena sativa* L.) and several species of crucifers have shown promise in lowering the inoculum potential of *A. euteiches* in Wisconsin and Minnesota (Fritz *et al.*, 1995;

Muehlchen *et al.*, 1990). Saponins are produced in oat roots and tops, and they are considered inhibitive to *Aphanomyces* and *Pythium* zoospores (Maizel *et al.*, 1963). Research in Minnesota (Fritz *et al.*, 1995) has shown a significant decrease in Aphanomyces root rot severity where green oat residue was incorporated to a shallow depth in the fall prior to sowing peas the next spring on *Aphanomyces* infested ground. Chisel plowing usually incorporates crop residue into the top 10 cm (Wilkins & Kraft, 1988). Placing the oat residue in the top 10 cm, where saponins are available to reduce primary zoospore inoculum and consequent infection of seedling roots, is necessary for significant control (Fritz *et al.*, 1995).

Since Aphanomyces root rot was first described in northern Idaho (Bowden *et al.*, 1985), a modified paper towel baiting technique was developed to determine field inoculum levels of *A. euteiches* (Kraft *et al.*, 1990). Because oospores are the survival structure of this pathogen and are buried in susceptible legume root debris, a baiting technique using wet-sieved organic matter was developed. Using this technique, *Aphanomyces* was readily detected in several areas and fields. Use of this procedure also revealed that infective oospores of *A. euteiches* were as deep as 60 cm in the soil profile and were present in areas with poor drainage in fields with low overall inoculum levels.

The fungicide Tachigaren (hymexazole) has shown promise as a seed treatment to reduce Aphanomyces root rot. Both greenhouse and laboratory studies demonstrated that 1.8 to 3.7 g a.i./kg seed plus 1.6 g a.i./kg seed Apron and 1/8 g a.i./kg seed Captan resulted in significant control. In further work at Prosser, the combination of resistant germplasm, fungicides, or biological seed treatments improved pea seedling stands, disease control, and seed yields (Kraft, 1982; Kraft & Papavizas, 1983; Kraft *et al.*, 1995).

Control of Aphanomyces root rot will depend on a multi-faceted program of resistant/tolerant varieties, green manure plowdowns, seed treatment chemicals, and longer intervals between susceptible legume crops.

Fusarium Root Rot of Chickpeas and Peas

Fusarium root rot of pea and chickpea is caused by *Fusarium solani* (Mart.) Appel and Wr. f. sp. *pisi* (F.R. Jones) Snyd. & Hans. (Kraft *et al.*, 1981; Bhatti & Kraft, 1992a). Symptoms on both pea and chickpea consist of yellowing of the basal foliage, stunted growth, and reddening of the vascular tissue below the soil line. The common site of seedling infection by *Fusarium* is the cotyledonary attachment area, below ground epicotyl, and upper taproot (Kraft & Roberts, 1967). Penetration of pea seedlings often occurs through stomates on the epicotyl (Bywater, 1959). Infection can then extend upward to the soil line and downward into the root zone. Initial symptoms on seedling roots consist of reddish-brown to blackish-brown streaks, which usually coalesce. A red discoloration of the vascular system can occur in the taproot but usually does not progress above the soil line.

Data on actual pea crop losses due to Fusarium root rot are scarce but yield losses up to 30% have been reported (Kraft & Berry, 1972; Raghavan *et al.*, 1982). Halila and Strange (1996) observed Fusarium root rot in 3% of Tunisian chickpea fields. The formae specialis responsible for Fusarium root rot of chickpea in many places has not been determined. However, *F. solani* f. sp. *pisi* was shown to be a virulent pathogen of chickpea roots when soil temperatures were 30°C or above (Bhatti & Kraft, 1992a).

Yield reductions in pea due to Fusarium root rot are influenced by previous cropping history, soil temperature, moisture, compaction, aeration, acidity, and fertility (Kraft *et al.*, 1981; Allmaras *et al.*, 1988; Bhatti & Kraft, 1992b). Likewise, Fusarium root rot of chickpea is more severe in compacted than in loose soil and root growth of chickpea was inversely correlated with soil compaction (Bhatti & Kraft, 1992b). In fact, the degree of root infection and damage caused by *F. solani* f. sp. *pisi* is directly dependent on the stress level to which the plant is exposed. Any condition(s) which decrease root growth will increase Fusarium root rot severity (Allmaras *et al.*, 1988; Kraft *et al.*, 1981; Kraft & Wilkins, 1989).

Chlamydospores of *F. solani* f. sp. *pisi* germinate to produce pre-infection growth when stimulated by root and seed exudates (Cook & Flentje, 1967). Rhizosphere effects may extend no more than 2 mm from the root surface and chlamydospore mobility is nil. Exudation from healthy pea roots is greatest near the root tip and along the zone of maturation (Rovira, 1973). *Fusarium* chlamydospores require 6 to 10 h for germination, and growth toward a substrate would probably miss the root tip with resultant contact with the zone of maturation where exudation is reduced. Poor aeration and/or soil compaction can reduce root growth and induce lateral root branching closer to the root apex, thus enhancing the probability that the germinating chlamydospore will make contact with the root tip and the exudation zone. *Fusarium solani* f. sp. *pisi* typically produces initial disease symptoms in the region of cotyledonary attachment, epicotyl, and hypocotyl, which are stationary (peas are hypogeal in germination) due to seed exudation that stimulates

chlamydospore germination. It has been our experience that only when the entire root system is invaded does *F. solani* f. sp. *pisi* cause serious disease losses.

In eastern Washington and northeastern Oregon, a definite tillage pan in all pea, wheat, or wheat-fallow sites was found, regardless of soil type and whether dryland or irrigated (Kraft & Allmaras, 1985). Typically, *F. solani* f. sp. *pisi* propagules were found throughout the upper 60 cm of soil, but their numbers were low in the tillage pan. The low numbers in the tillage pan and their presence below it could be related to impaired drainage from tillage pan compaction and the saprophytic survival of *F. solani* f. sp. *pisi* in the drier subsoil. In fields not cropped to peas for five or more years, *F. solani* f. sp. *pisi* was not detected in the plow layer but was recovered in the subsoil and there was a corresponding increase in yield. Long-term cultivation has apparently produced an environment beneath the plough layer that is favourable for survival of *F. solani* f. sp. *pisi*. In the absence of other stress factors, inoculum of *F. solani* f. sp. *pisi* deep in the soil profile has little detrimental effect on pea growth and development up to anthesis, when the upper 20 cm of the root system is not infected (Rush & Kraft, 1986). When inoculum levels of *F. solani* f. sp. *pisi* were significantly reduced by methyl bromide fumigation in the 0-20 cm depth and compaction was reduced due to tillage, there was a decrease in root disease and an increase in root growth and dry seed yields (Kraft & Wilkins, 1989).

Glyphosate is being used as an alternative to mechanical weed control in the winter wheat-green pea rotation of southeastern Washington and northeastern Oregon. Glyphosate was found to stimulate proliferation of *F. solani* f. sp. *pisi* in the rhizosphere of some common weeds sprayed with it (Kawate *et al.*, 1997). Apparently, after exposure to glyphosate, nutrients were released in sufficient quantities for *F. solani* f. sp. *pisi* to increase in population numbers.

A breeding program to incorporate resistance to *F. solani* f. sp. *pisi* in pea has been ongoing at Prosser, WA, since 1967. High seed vigour is an important consideration in comparing one pea line with another for resistance to *Fusarium*. A line with poor seed vigour may appear susceptible to Fusarium root rot when in fact it is genetically resistant (Kraft, 1986). Because seed and seedling vigour is important in the development of Fusarium root rot, a seed soak test to screen peas for resistance is now used (Kraft & Kaiser, 1993). Seeds of test lines with high vigour are soaked overnight in a conidial suspension of *F. solani* f. sp. *pisi* adjusted to 1 x 10^6 per ml and planted in coarse-grade perlite. Inoculated lines are read after 14 days and scored on a 0-5 scale. Good progress has been made and will continue to be made in developing peas with acceptable horticultural traits and inheritable resistance to Fusarium root rot (Kraft & Kaiser, 1993). Because resistance is not of a high level, an integrated control approach is needed which includes cultural practices, maintenance of good seed vigour, and genetic resistance.

Sclerotium Rot of Cool Season Legumes

Sclerotium rolfsii Sacc. (teleomorph *Athelia rolfsii* (Curzi) Tu & Kimbrough, 1978) is a serious pathogen of chickpea and causes a collar rot of lentil. This fungus has a wide host range of nearly 500 species with Graminaceous species being less susceptible (Punja, 1985). This pathogen is widely distributed in warm climates and the disease is usually observed under wet warm conditions and in fields where chickpea and lentil are sown following a paddy rice crop. Sclerotia formed on undecomposed tissues in the field are capable of initiating infection and are the primary source of inoculum (Punja & Grogan, 1981). *Sclerotium rolfsii* can cause damping-off of seedlings, stem canker, and/or root rot. When chickpea or lentil seedlings are attacked, this pathogen is capable of invading all parts of the host and they die quickly. Usually the infection on more mature plants begins on the succulent stem as a dark-brown lesion just below the soil line. The first visible symptoms appear as yellowing or wilting of the lower leaves which progresses to the upper leaves. The fungus grows upward in the plant and downward into the roots. White mycelium is always present in infected tissues and grows over the soil to adjacent plants to start new infections. On all infected tissues the fungus produces numerous small round-shaped sclerotia which are brown in colour. The mature sclerotia are not connected with mycelial strands and have the size, shape, and color of mustard seed (Punja & Rahe, 1992). *Sclerotium rolfsii* kills and disintegrates tissues by secreting oxalic acid, and also pectinolytic, cellulolytic, and other enzymes. Once established, production of mycelium and sclerotia are very rapid, especially during periods of high moisture and temperature (30-35°C). This pathogen usually attacks plants near the soil line and sclerotia are capable of surviving for long periods. The only economic control consists of long-term rotations with non-susceptible hosts and deep plowing of sclerotia.

Collar rot of lentil, caused by *S. rolfsii,* can be reduced by altering the sowing date so the seedling stage does not coincide with high soil moisture and temperatures above 25°C (Agrawal *et al.*, 1975). Crop rotation is unlikely to be an effective method of control due to the wide host range and *S. rolfsii's* persistence on numerous types of host debris. Seedling mortality in lentil can be significantly reduced by treating seed with combinations of fungicides, such as thiram + pentachloronitrobenzene or thiram + carbendazim (Agrawal *et al.*, 1975). Mancozeb seed treatment has also been found to reduce collar rot of lentil (Singh *et al.*, 1985). No biological control of this pathogen has been achieved under field conditions with either chickpea or lentil. However, several resistant lentils have been identified (Khare *et al.*, 1979; Kannaiyan & Nene, 1976; Abu-Mohammad & Kumar, 1986).

FUSARIUM WILT OF CHICKPEA, PEA, AND LENTIL

Fusarium wilt of pea, lentil, and chickpea is caused by the formae specialis *pisi*, *lentis,* and *ciceri*, respectively of *F. oxysporum*. These pathogens are soil inhabitants that can survive indefinitely in soil and be seedborne.

Chickpea Wilt

Among the diseases reported on chickpea, wilt caused by *F. oxysporum* Schl. emnd. Snyd. Hans. f. sp. *ciceri* [Padwick] Snyd. & Hans. is one of the most important diseases in North Africa, South Asia, and Southern Europe (Nene & Reddy, 1987), some areas in the United States (Buddenhagen & Workneh, 1988) and causes up to 10% losses in yield. In Tunisia, wilt is present in 30-40% of chickpea fields (Halila & Strange, 1996). The disease is more prevalent in the lower latitudes (0-30 N) where the chickpea-growing season is relatively dry and warmer than in the higher latitudes (30-40 N). The pathogen is soilborne surviving in soil for more than 6 years in the absence of a susceptible host (Haware *et al.*, 1986; Haware & Nene, 1982b) and is also seedborne (Haware & Nene, 1978).

This pathogen exhibits physiologic specialization and seven races have been reported from India, Spain, and the USA. Of these seven races, designated 0-6, races 1, 2, 3, and 4 were reported in India (Haware & Nene, 1982a) and 0, 5, and 6 in Spain (Jiménez-Díaz *et al.*, 1989). In Tunisia, morphological and pathogenic variability studies determined that race 0 predominates (Halila & Strange, 1996). In Morocco, race 1 was found to be the predominant pathogen (El-Hadi, 1993). Research conducted in Tunisia showed that chickpea cultivars varied in wilt symptoms from very early wilting to very late wilting (Halila & Strange, 1996). Late wilting is thought to be a form of partial resistance, governed by more than one gene, and that complete resistance can be obtained by crossing late wilting parents (Upadhaya *et al.*, 1983). Considerable progress has been made in identifying wilt resistant sources and the development of wilt-resistant and high-yielding chickpea cultivars. Breeding programs at national and international centers have developed and released resistant cultivars; however, these cultivars have not maintained resistance across locations due to area specific races of the wilt pathogen (Infantino *et al.,* 1996). At the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), over 13,500 chickpea accessions from 40 countries have been evaluated for wilt resistance (Haware *et al.*, 1992) and 160 accessions were found resistant to race 1. Of these, 150 were "desi" types.

Zote *et al.* (1996) reported that wilt susceptible, moderately susceptible, and resistant cultivars of chickpea all supported multiplication of *F. o.* f. sp. *cicèri* in the rhizosphere. Apparently, it is not possible to eliminate the chickpea wilt pathogen from infested soil by growing resistant cultivars alone. There is a need to employ other management practices such as long-term crop rotation to reduce the inoculum density in soil.

Pea Wilt

The pea wilt pathogen, *Fusarium oxysporum* Schlecht f. sp. *pisi* (van Hall) Snyd. & Hans, is a soil inhabitant that can survive indefinitely and can be seedborne (Kraft, 1994). Typical symptoms of pea wilt are pale foliage, downward curling of leaves, and vascular discoloration both below and above ground (Hagedorn, 1984). Often the above- and below ground vascular system turns a light yellow to brick-red color, and the lower subterranean portion of the stem becomes larger than normal. Penetration often

occurs through root tips (Nyvall & Haglund, 1972). The pathogen concentrates in the larger xylem elements and can reproduce in the rhizosphere of both resistant and susceptible cultivars (Charchar & Kraft, 1989).

Race 1 (common wilt) of pea, caused by *F. oxysporum* Schl. f. sp. *pisi* race 1 Snyd & Hans., was first reported in Wisconsin in 1924 and resistance was attributed to a single, dominant gene. Race 1, after the release of resistant cultivars, was not a problem in commercial production in the United States again until 1972 (Kraft *et al.*, 1974). Race 2 (near wilt) of pea was described in 1933 and is found in most pea growing areas of the world (Hagedorn, 1984). Resistance to race 2 is also controlled by a single dominant gene. In 1970, *F. oxysporum* f. sp. *pisi* race 5 (Haglund & Kraft, 1970) was described from western Washington which killed varieties resistant to races 1 and 2. In 1979, an additional race of *F. oxysporum* f. sp. *pisi* was described, which killed varieties resistant to races 1, 2, and 5. This strain was named race 6 (Haglund & Kraft, 1979). Resistance to races 5 and 6 are again attributed to separate, single, dominant genes.

The pathogenicity of races 1, 2, 5, and 6 of *F. oxysporum* f. sp. *pisi* can be distinguished by their reaction on differential pea varieties (Kraft, 1994). The disease reactions are based on a resistance response (no observable disease) and a susceptible reaction (dead or severely stunted, chlorotic plants). However, pathogenicity tests are subjective because they are influenced by temperature, host plant age, method of inoculation, etc. Research during the last 10 years has classified strains of *F. oxysporum* using vegetative compatibility groupings (VGC) by pairing nitrate nonutilizing (nit) mutants generated on a potassium chlorate medium. Strains, or races of *F. oxysporum,* can be further characterized based on fungus genetics along with host-pathogen interactions. Within *F. oxysporum* f. sp. *pisi*, 4 VGS's have been reported (Correll, 1991). Molecular techniques have revealed that races 1, 5, and 6 are closely related, and that race 2 is distinct (Coddington *et al.*, 1987; Kistler *et al.*, 1991). So far, all isolates of race 2 have exhibited a highly conserved banding pattern, whereas isolates of the other races exhibited much more variability with the primers used.

There has been disagreement in the literature on classifying races of *F. oxysporum* f. sp. *pisi* (Armstrong & Armstrong, 1974; Haglund, 1974; Kraft & Haglund, 1978). Currently, the accepted method of classifying any isolate for a race designation is by host-pathogen response. Because inoculation procedures, genetic homozygosity of host and pathogen, environmental conditions, and inoculum levels all influence the host-pathogen response, standardization of procedures is important for repeatability (Huebbeling, 1974; Kraft & Haglund, 1978).

The only economic control of pea wilt is to use resistant varieties, index prospective fields for presence of a given race, and avoid planting a susceptible variety in infested soil.

Lentil Wilt

Several species of *Fusarium* have been associated with wilted lentils (Khare *et al.*, 1979). However, the primary pathogen appears to be *Fusarium oxysporum* Schlecht ex. Fr. f. sp. *lentis* Vasudeva and Srinivasan (Chattopadhyay & Sengupta, 1967). The disease is widespread where lentil is grown. Lentil wilt has been reported in Argentina, Canada, Chile, Colombia, Czechoslovakia, Egypt, Ethiopia, France, Hungary, India, Jordan, Morocco, Nepal, Sudan, Syria, Turkey, Tunisia, Uruguay, the USA, and the former USSR. The host range is primarily lentil, however *Vicia montbretii* Fisch. & C.A. can be infected when artificially inoculated (Bayaa *et al.*, 1995). Although variability in fungal sensitivity to nutrition, fungicides, and temperature exists in strains of *F. oxysporum* f. sp. *lentis,* races of this pathogen have not been defined.

Symptoms appear as patches of infected plants in the field at either the seedling or adult plant stage. Seedling wilt is characterized by sudden drooping, followed by drying of leaves and death of seedlings. Wilt in mature plants can appear from the flowering to late pod filling stage and is also characterized by sudden drooping of top leaflets, dull green foliage color, and wilting of individual branches or the whole plant. The root system of either infected seedlings or mature plants appears healthy, with a slight reduction of lateral roots but with very little vascular discoloration.

In India, seedling wilt of lentil appears during November and/or in the adult plant stage during April-May. The pathogen can survive in soil more than five years due to chlamydospore formation. Lentil wilt is more severe in sandy loam soil than in clay soil and the mortality of infected lentil plants increases with soil pH up to 7.5. Wilt is most severe during warm weather (Izquierdo & Morse, 1975; Bayaa *et al.*, 1986; Agrawal *et al.*, 1993). Sowing date also affects wilt incidence. In India, delayed sowing reduced disease incidence but late sowing reduces yield (Kannaiyan & Nene, 1975). A rotation out of lentils for 4 to 5 years reduced inoculum density but did not completely eradicate the disease. In India, cultivation of paddy rice or

sorghum during the rainy season reduced wilt incidence the next winter (Kannaiyan & Nene, 1979). Biological control of lentil wilt under field conditions has not been achieved.

Cultivars with resistance to lentil wilt have been released from Bulgaria, Lebanon, and India. Sources of resistance have been found by many authors in cultivated lentil (Kannaiyan & Nene, 1976; Bayaa & Erskine, 1990; Hamdi *et al.*, 1991; Hossain *et al.*, 1985; Khare *et al.*, 1979; Tiwari & Singh, 1980) and made available through the Lentil International Fusarium Wilt Nursery. Resistance has also been found among wild lentil relatives *L. culinaris* ssp. *orientalis* and *L. nigricans* spp. *ervoides* (Bayaa *et al.*, 1995).

CONCLUSION

Soilborne fungal diseases of cool season grain legumes are described as seed and seedling blights, root rots, and wilts. Seed and seedling diseases are caused primarily by *Pythium* spp. and *Rhizoctonia solani*. However, in Western Australia, Pleiochaeta seedling and root rot has become a serious yield constraint. The most important fungi causing root rots include *Aphanomyces euteiches*, *Fusarium solani*, *Rhizoctonia solani*, and *Sclerotium rolfsii*. Wilt is caused primarily by various host-specific forms of *Fusarium oxysporum*.

Such diseases as *Aphanomyces, Fusarium, Pythium, Pleiochaeta,* and Sclerotium rot are dramatically increased by short rotations, which facilitate significant increases in soil inoculum. Resistance to these diseases is usually not sufficient to withstand high inoculum levels, especially in concert with other biotic and abiotic stresses which predispose these legumes to root diseases. Consequently, a multifaceted approach is needed to control these diseases including host resistance/ tolerance, biological and chemical seed treatments, longer rotations between susceptible crops, and cultural practices which enhance rapid root growth.

High levels of genetic resistance exist for most races of Fusarium wilt, which attack chickpea, pea, and lentil. Resistance to various races of wilt has been stable for long periods. However, new races of the wilt pathogen have developed in areas where the crop has been grown in monoculture or short rotations and where resistance to an existent race(s) is extensively grown. This has certainly been the case for the pea wilt pathogen in the Skagit Valley of Western Washington.

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