CHAPTER 13

Diseases of Garden Peas (*Pisum sativum* L.) and Their Management

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13.1 INTRODUCTION

Garden pea (*Pisum sativum* L.) is an important pulse crop belonging to family *Fabaceae* and sub-family *Papilionaceae*. It is an important Rabi, herbaceous, frost-hardy annual crop. It has a global importance but mainly grown, particularly in Asia, Europe, and North America. In India, its cultivation mainly confined to northern and central parts.

13.2 ORIGIN AND DISTRIBUTION

The origin and progenitor of *Pisum sativum* L. is not clearly well known. The Mediterranean region, eastern, and Central Asia and Ethiopia have been indicated as the center of origin. Recently the Food and Agriculture Organization (FAO) designated Ethiopia and western Asia as the center of diversity, with a secondary center in southern Asia and the Mediterranean region. Peas were popular with the ancient Greeks and Romans and the word 'pea' is derived from the Latin word 'pisum.' The first cultivation of pea appears to have been in western Asia from where it spread to Europe, China, and India. Presently it is found in all temperate and tropical countries.

13.3 CLIMATIC REQUIREMENTS

Garden pea may be grown in varied climatic and weather conditions. It requires cold and dry climate and the longer cold spell helps enhancing

yield. Seeds can germinate even at high temperature but the process is slow. The optimum temperature requires for germinations 20–22°C.

13.4 SOIL REQUIREMENTS

Garden peas can be grown on all types of soils but it prefers well-drained sandy loam soil. The soils should rich an organic matter as it enhances better growth by supplying nutrient sat as lower rate. It grows best at pH of 6.5 and does not thrive in highly acidic or alkaline soils or saline soils. The ideal soil is clay loam. If soil conditions are good, its cultivation becomes very easy and successful. Garden pea is grown mainly as a Rabi crop which normally sown in October and November and harvested in the month of February and March. Being a leguminous crop has capacity of fixing atmospheric nitrogen in the soil. In spite of high yielding varieties and improved agronomic practices the productivity of pea is low. Diseases are one of the most limiting factors responsible for low yield per hectare. Among the diseases, powdery mildew (*Erysiphe polygoni*), Downy mildew (*Peronospora pisi*), wilt (*Fusarium oxysporum* f.sp. *pisi*), Rust (*Uromycesfabae*) and *Ascochyta* blight (*Ascochyta* spp.) are the most important diseases of pea in India (Sharma, 1998).

13.5 POWDERY MILDEW

Powdery mildew is one of the most important worldwide distributed airs borne disease of garden pea. Its prevalence has been reported from Australia, Canada, China, India, Japan, Malaysia, Russia, Spain, South Africa, Sudan, Tanzania, Turkey, USA, and many other countries (Singh and Singh, 1988). The disease is widespread and often economically important in semi-arid regions of the world. The disease was first reported in India from Dehradun by Butler in 1918. The disease is usually more destructive during late season crop, which attains a serious threat and causing huge losses in both quality and quantity of the produce. The disease can reduce the number of pods per plant, number of seeds per pod, plant height, biomass, and ultimately 25–50% yield loss. Munjal et al. (1963) observed that in severely infected crop, there was reduction of 21–31% pod number, 25–47% in pod weight. Griton and Ebert (1975) reported that 50% reduction in yield was due to powdery mildew infection. However, the reduction in yield is mainly due to reduced photosynthetic activity in infected plants by the attack of this pathogen. Epidemic development of the disease is very often, fast, and it progresses as compound

interest. In dry and warm condition the disease becomes more destructive, while downy mildew flourishes in humid weather condition.

13.5.1 SYMPTOMS

First symptom appears on the upper surface of the lower leaves as a very small, slightly discolored spots. These soon give rise to white powdery areas which continue to enlarge as white patches. These white patches combine together to form larger whitish floury areas. As the disease advances, the upper leaves of the plants also show similar symptoms. In severe conditions, both leaves surface adversely infected by the pathogen. Due to the presence of conidia and conidiophores on leaves looks as dusted with flour and the whole crop in the field appears as white from a distance. At the end of conidial formation or in a later stage, the leaf surface shows yellow to brownish patches, and finally, foliage dies. In severe conditions, entire tendrils and petioles are covered with white powdery mass. Pods are also infected in all stages and show white floury patches consisting of white powdery mass. Pods become small in size, shriveled, pod number, and pod weight are also reduced multiple infections may cover the entire aboveground plant. Severely infected plants are unthrifty and have poor yield and quality. Small, oval, black fruiting structures may form in mature lesions.

13.5.2 CAUSAL ORGANISM

The causal agent of the disease is *Erysiphe pisi*, although other species such as *Erysiphe trifolii* and *Erysiphe baeumleri* have also been reported causing this disease. According to Paul and Kapoor (1983), the various species described under the name *Erysiphe polygoni* on different hosts in India were found to comprise eight species viz., *E. polygoni, E. pisi, E. berberidis, E. betae, E. heraclei, E. martii, E. rananculi* and *E. salviae*. The pathogen is ectoparasitic in nature and withdraws their nutrition from host surface. The fungus overwinters on infected plant debris and in alternate hosts. Air current helps in spreading the fungus locally and over long distance.

13.5.3 DISEASE CYCLE AND EPIDEMIOLOGY

Powdery mildew is an air-borne disease of worldwide distribution**.** Cleistothecia develop on dead plant debris and serve as source of primary inoculums

for the next season. Ascospores formed in these fruiting bodies are released by decay of the fruiting bodies and blown by wind to lower leaves where cause infection and produce powdery mass of spores. Secondary spread occurs through windblown conidia. Rainfall suppresses the disease by washing off the spores and causing them to burst instead of germinates. Free moisture on plants will also restrict germination of the spores and does not promote the epidemic. Epidemics of powdery mildews frequently start from foci of infection. Spatial distribution of fungal plant pathogens is determined by components of the disease cycle such as pathogen survival, source of primary inoculums, mode, and amount of inoculums dispersal. Host plant genotypes with quantitative resistance may have less disease because attacking pathogens have reduced infection efficiency, longer latent period or reduced propagule production. One or more of these components can reduce the disease progress (temporal increase) and may spread (spatial increase) in the field. Powdery mildew develops quickly in warm and dry condition for 4–5 days, particularly at flowering and podding stage. If infection occurs earlier than four weeks from maturity, yield losses due to powdery mildew arise from the infection covering stems, leaves, and pods, which will lead to shriveled seeds (Yarwood et al., 1954).

13.5.4 MANAGEMENT

13.5.4.1 CULTURAL

Cultural practices should be applied to avoid the favorable condition for infection and spread of the powdery mildew. Collection and destruction of plant debris and avoidance of close planting are helpful in reducing disease incidence. Pratibha and Amin (1991) reported that sowing of pea from late September to early October, late November or early December show more powdery mildew and give reduced yields. The adjustment of date of sowing may be important in avoiding or reducing powdery mildew infection. The disease does not develop fast under sprinkler irrigation system (Hagedorn, 1984) and it may help to reduce powdery mildew because spores are washed off the plant.

13.5.4.2 HOST RESISTANCE

Genetic resistance is acknowledged as the most effective, economical, and environmental friendly method of disease control. However, only three genes

(*er1*, *er2* and *Er3*) have been described so far in pea germplasm and only *er1* has been widely used in breeding programs. The use of polygenic resistance or combining several major genes could enhance the durability of the resistance. Many workers have investigated varietal resistance of pea to powdery mildew, but very few varieties have been reported to be resistant (Singh et al., 1988). Resistance in powdery mildew is controlled by a single gene pair 'er' (Azmat et al., 2010). Fondevilla et al. (2006) reported that in pea, two single recessive genes, er1 and er2 have been identified for resistance to powdery mildew caused by *E. pisi*. Gupta and Mate (2009) reported that conidia of *E. polygoni* varied in size according to susceptibility of the cultivars.

13.5.4.3 BIOLOGICAL

Introduction of resistance in pea with nonpathogenic pea powdery mildews (*Oidium* sp., *Phyllactinia corylea*) against a subsequent infection with *Erysiphe pisi* resulted in reduced conidial germination, appresorium formation and secondary branch development up to 12 days after inoculation (Singh et al., 2003). Application of aqueous extract of vermicompost also inhibits spore germination and development of powdery mildews on pea (*Pisum sativum* L) in the field at very low incidence (0.1–0.5%). *Trichoderma harzianum* (0.5%) was found effective and economical for controlling the disease and giving better seed yield (Surwase et al., 2009). Plant extracts and oils are better than chemical because they are environment-friendly and safe from residual effect. It adversely affects the germination of conidia or conidial density (Singh et al., 1984). Turmeric extract significantly reduced the disease severity and increased the number of pods, number of grains, and grain yield (Shabeer and Irfan, 2006). The botanical NSKE (5%) was found highly effective and economical for controlling the disease and giving higher seed yield (Surwase et al., 2009).

Maurya et al. (2004) reported that neem bark methanol extract (NBM) and motha (*Cyperus rotundus*) rhizome ethyl acetate extract (CRE) significantly reduced disease intensity. Ginger, tulsi, mahua, and cashew nut extracts were also effective at concentration 2000 ppm *in vivo.*

13.5.4.4 CHEMICAL

Chemical control is feasible with a choice of protective and systemic fungicides. Among the large number of fungicides tested, high level of control of

the disease was observed with the use of tridemorph, dinocarp, and wettable sulfur as foliar spray but sulfur is the most economical fungicide. Foliar sprays of EBI fuhgicides like propiconazole, hexaconazole, tebuconazole, diclobutrzol, triadimenol, enarimol, etc. have been found effective at lower doses than sulfur and posses good antisporulant activity (Rana et al., 1991; Gupta and Sharma, 2004). Tilt (propiconazole) was the most effective treatment, which not only increased seed yield but also reduced disease severity significantly as compared to unsprayed check followed by kerathane (0.1%) and carbendazim (0.1%) during the crop seasons for two years (Prasad and Dewivedi, 2007). The fungicides hexaconazole (0.05%) and Propiconazole (0.05%) were found very effective and economical for controlling the disease and giving higher seed yield (Surwase et al., 2009). Sulfur has a significant chemical cost advantage over systemic fungicides and is acceptable to biodynamic/organic growers.

13.6 DOWNY MILDEW OF PEA

Downy mildew of pea was first discovered by Berkeley in England in 1846 (Chupp and Sherf, 1960), and since then, it has been found in all parts of the world. Dixon (1981) has also reported that downy mildew of peas is widely distributed all over the world. Olofsson (1966) and Biddle et al. (1988) reported yield losses of 30% in Sweden and 45% in the UK, respectively. In India, the disease is prevalent throughout the Indo-i plain and causes considerable yield loss.

13.6.1 SYMPTOMS

Downy mildew causes a different kind of symptoms on pea plants. Three different infection types with different symptoms can be recognized during a crop cycle. The symptom is first visible on the developing third and fourth leaves, and it first appears on the lower leaves then spread to upper leaves. Scattered yellow to brown patches of indeterminate shape appear on the leaves surface. Systemic infection in seedlings causes stunted growth with conidia sporulation, which often covers a major part of the plant surface, and this is caused by oospores in the soil which infect germinating seeds. These infections can seriously reduce the plant population. A lower frequency of infection (50%) was obtained when placed 3 cm above the seed level, and infection was even more reduced (1%) when the oospores were placed 3 cm

below the seeds. Later in the season, top systemic symptoms can develop with stunting and sporulation occurring over the entire surface of the top of plants. Taylor et al. (1990) showed that systemic infection could also originate from leaf infection. Top systemic infection is the result of direct infection of the top meristem. This type of infection is found more frequently in varieties with reduced stipule size, which may be determined by the gene st (Taylor et al., 1990). In these varieties, the top meristem is not protected by the stipules, which wrap around the apex in varieties with normal-sized stipules. Following infection, the mycelium develops in the intercellular spaces penetrating the stem, the leaf stalks, and even the pods through the veins (Kosevskii and Kirik, 1979).

Local foliar and tendril lesions with conidia sporulation on the abaxial foliar surface is a typical symptom. Local infections on tendrils may develop from conidia present on the plant surface. Pod infection causes yellow lesions on the pod surface and epithelial proliferations on the endocarp. Pod infection develops from conidia deposited on young pods rather than by mycelial growth through the peduncle and pedicel (Mence and Pegg, 1971). Oospores are formed within the yellow lesions. Pod infection often causes distorted pods, seed abortion, and brown discolored small peas with a bitter taste. Pod infections directly affect pea quality and are, therefore, a serious expression of the disease. The seeds beneath the lesions are aborted and undersized. Later in the season, oospore develops in the senescent tissues and can survive in the soil for up to 15 years (Van der Gaag and Frinking, 1997).

13.6.2 CAUSAL ORGANISM

Downy mildew of pea is caused by *Peronospora pisi* Syd. and belongs to kingdom- chromista, phylum- Oomycota, class- Oomycetes, order- Peronosporales and family- peronosporaceae. Mycelium of the fungus is hyaline, aseptate, intercellular, branched with finger-shaped haustoria. Sporangiophores first appear as simple elongating hyphae from stomata on abaxial leaflet surface then branching from a single axis to produce multiple terminal sporangia. Gametangia developed extensively on inner surface of pods from smooth, bulbous hyphae, adhering to the host epidermis. Species of *Peronospora* produce conidia that lack modification in the apical region, the operculum which do not contain zoospores and germinate by germ tubes (Shaw, 1981). *P. viciae* also produces oospores, which have a typical reticulate pattern of the exosporium. These species are also capable of regular and

predictable production of oospores in large numbers. Both heterothallic and homothallic isolates of these two species have been found. Sexual reproduction is probably important for the adaptation of the fungus to various host genotypes by recombination of virulence genes. The vegetative stage is probably diploid like in other species of *Peronospora* (Tommerup, 1981). The fungus is an obligate parasite which can only grow on living plant tissue. Forma specialis pisi can only infect *Pisum* species and not species of the genus *Vicia* within the tribe *Vicieae* (Campbell, 1935).

13.6.3 DISEASE CYCLE AND EPIDEMIOLOGY

The disease is both seed and soil-borne in nature. In the seed, the fungus may be present as a contaminant, or the seed infection may extend beneath the seed coat. Oospores on germination, the infected seeds might act as the source of perpetuation; however, the main sources of primary inoculums are the oospores on in the diseased crop debris where these can survive up to two years. Oospores on germination produce a germ tube that infects the seedlings systemically. Oospores in the soil also act as a source of primary inoculums early in the season. The oospores can survive for a long time in the soil. Oospores survive for 10–15 years in the soil (Olofsson, 1966). Secondary spread of the disease occurs through sporangia disseminated by the moist wind. Under favorable temperatures for sporangial germination, four hours of leaf wetness is required for Infection. High humidity also helps in the dissemination of sporangia. Cool nights coupled with foggy weather or the presence of dew favor the disease development. Optimum temperature for infection to occur is 16°C.

13.6.4 MANAGEMENT

13.6.4.1 CULTURAL

Field sanitation, destruction of infected crop debris and 3–4 years crop rotation with nonhost crops are important in reducing the primary inoculums. Proper drainage and wider spacing create micro conditions that are unfavorable for disease development. Since the pathogen is also seed-borne in nature, it is always recommended to use disease-free seed. Seed treatment in hot water at 50°C for 25 minutes has also been found effective to eradicate the seed-borne inoculums. Watering early in the morning so that leaf

surfaces dry out rapidly and avoid it in the evening, which can lead to high humidity or leaf wetness and persists throughout the night.

13.6.4.2 HOST RESISTANCE

During recent years improved varieties have been reported from India, and it is imperative that they are protected against the major diseases of pea. Pea varieties are known to differ in their reaction to downy mildew disease. Bains et al., (1993) reported the pea varieties PWR-3 and Bonneville have resistance under Punjab condition. Variation in resistance between pea cultivars has been reported by Stegmark (1988). Some cultivars are completely resistant to some isolates but are fully susceptible to others. Race-specific resistance of pea was found in several cultivars, but there is no genotype with complete resistance to all known pathogen races (Matthews and Dow, 1983). The cultivar 'Dark Skin Perfection' is more resistant to downy mildew than some other cultivars used for the production of peas for canning and freezing (Stegmark, 1988).

13.6.4.3 CHEMICAL

The pathogen is known to overwinter in the form of oospores in the infected plant tissues and in the seed. Seed borne inoculums should be eradicated through seed dressing with systemic acylalanine fungicides like metalaxyl, cymoxanil, and fludioxanil are very effective against systemic seedling infections (Brokenshire, 1980). However, later in the season, the pod infection can still be severe. There is no real effective fungicide treatment against pod infection in peas. In the long run, the current acylalanine fungicides may become ineffective due to the development of tolerance by the pathogen. The secondary spread of the disease should be checked by spraying fungicides like carbendazim + chlorothaonil, Carbendazim, + mancozeb. To avoid buildup of fungicide resistant strains, it is always better to rotate the fungicide and apply in mixture with non systemic.

13.7 FUSARIUM WILT

Wilt of pea caused by *Fusarium oxysporum* f. sp. *pisi.* (Linford) Snyder and Hansen is one of the most important diseases in pea growing areas. The *Fusarium* wilt of pea was first reported in 1918 by Bisby in Minnesota

(Chupps and Sherf, 1960). In India, the occurrence of Pea wilt report was made available by Patel et al. (1949) from Bombay. Peshne (1966) made a comparative study on morphology, physiology, and pathogenicity of *Fusarium oxysporum* f. sp. *pisi.* Wilt of pea causes serious losses world-wide and is one of the major yield reducers. Sharma et al. (1998) found that soil born disease like root rot and wilt are the limiting factors in producing early crop of pea. Maheshawari et al. (1983) made a survey of wilt and root rot complex of pea in the various pea growing areas in Northern India and reported 13.9 to 95% yield loss. Losses up to 60% were reported in the crop during the years of epiphytotics in Himachal Pradesh (Kumar, 1983).

13.7.1 SYMPTOMS

Plants are susceptible to the disease at any stage of crop growth. Symptoms of pea wilt have been described by different workers from time to time. Linford (1928) observed symptoms as a distortion and wilting of leaflets by sudden collapse of the plants and extensive cortical decay of the roots. The first and the foremost characteristics symptoms is a downward incurving of the margins of younger leaves and stipules, accompanied by a slight yellowing of the leaves and a superficial grayness suggesting an excessive development of waxy bloom. The lower internodes increase in diameter and the entire stem becomes rigid (Schroeder and Walker, 1942). Sukapure et al. (1957) reported symptoms of pea wilt as rolling of leaves, upper parts of the plants may be pale and the growth of terminal bud is checked. Stem and upper leaves may become more rigid than normal and the roots crisp, while the lower leaves turn pale and commence to wither. Sometimes, the entire plant becomes yellow and the lower leaves wither progressively upwards; however, after the collapse of a few basal leaves, the upper part of the plant wilts abruptly and may become dry while still green in color. After such wilting the stem shrivels downwards from the tip to basal internodes, which remains firm and turgid till the end discoloration of vascular system and partial wilting is characteristic symptom of *Fusarium* wilt. If pods are formed, they contain only a few shrunken, immature seeds and dry earlier.

13.7.2 CAUSAL ORGANISM

Wilt is an important soil born disease, caused by *Fusarium oxysporum* f. sp. *pisi* (Linford) snyder and Hansen is one of the most important diseases

of pea. The fungus belongs to kingdom fungi, Phylum Ascomycota, Order Tuberculariales and Family Tuberculariaceae. The fungus hyphe is septate, delicated, white to peach colored, usually with a purple tinge. Microconidia are borne on simple phialades arising laterally on hyphae or from short, less branched conidiophores. These are oval ellipsoid to cylindrical or curved and measures 5–12 x 2.2–3.5 micrometer. Macroconidia are borne on elaborately branched conidiophores or on the surface of sporodochia. These are thin-walled, 3–5 septate, fusoid-subulates pointed at both ends and measure 27–46 x 3–4.5 micrometer. Chlamydospores are both terminal and intercalary.

13.7.3 DISEASE CYCLE AND EPIDEMIOLOGY

Fusarium oxysporumf. sp. *Pisi* is mainly soil-borne pathogen that is commonly found in soil. It can survive in soil as chlamydospores without pea crop for more than 10 years. The pathogen is not a seed-borne in nature, but it can be carried on the seed coat. Anwar et al. (1994) isolated the pathogen from both ingeminated seed and abnormal seedlings. The fungus causes infection on the fibrous roots or epicotyls region and grows inter and intercellular in the cortex and ultimately concentrates in the xylem vessels. The mycelium may grow systemically through the vascular system and reach to the seed causing infections. The infected seeds germinate and resulting in production of abnormal seedlings. After the death of the plant, the fungus continues to grow and sporulate on the stem cortex, resulting in production of soil-borne inoculums. The spores penetrate the pea plant through the root hairs and fibrous roots. The pathogen mainly enters through cut surface and the exposed stele is necessary for infection. It grows upward through the stem often well into the upper branches in the xylem. This process interferes with the passage of water from the roots to the stems, leaves, and pods resulting in yellowing, dwarfing, and wilting of plants.

The pathogen is soil-borne and also survives on the seed. The spores can be carried from one field to another on farm equipment, on crop debris and in wind or water-borne soil. The disease is more prevalent in alkaline soil. Favorable condition for plant growth reduces fungus growth and does not permit the disease to progress rapidly. The reduction in shoot length could be used to supplement the visual severity rating for *fusarium* wilt in field pea (Neumann and Xu, 2003). The pathogen establishes in some areas quickly than others and is serious under moist conditions. A soil temperature of 23 to 27°C is most favorable for disease development.

13.7.4 MANAGEMENT

13.7.4.1 CULTURAL

The use of disease-free seed, practicing field sanitation, and long crop rotations are important in reducing the primary inoculums of the pathogen. Early planting of pea crop has also been suggested to lower the disease levels. Soil amendment and repeated cropping with different non-host crops such as wheat, oats, maize, and sorghum reduced the wilt population. Minimum disease incidence and maximum grain yield were observed during the 1st and 15th November when the soil temperature are low for the development of wilt and it was very severe when the planting were done in first week of September (Sharma and Sharma, 2003). Use of balanced fertilizers reduces the wilt complex in pea (Sagar and Sugha, 1998). Minimum disease incidence and apparent infection rate were observed when seeds were sown at 8 cm distance spacing as compared to other spacing (Verma and Dohroo, 2005a).

13.7.4.2 HOST RESISTANCE

The only economic control in wilt infected soil is to grow resistance or tolerant varieties of pea against wilt. Virin and Walker (1939, 1940) evolved a system of numerical disease evaluation based on the symptoms of disease by calculating the disease indices. In this method, the index represents the average number of days from sowing to the particular stage of the disease concerned. Ramphal and Choudhary (1978) observed soil inoculation with wilt pathogen was the most effective method of producing pea wilt disease. They screened pea cultivars and reported Kalanagni as immune and Alaslea as a resistant variety. Datar (1983) tested 36 cultivars against *Fusarium oxysporum* f. sp. *pisi* in field condition, out of which five cultivars namely BR-12, Khapera Khada, 15/1, 4–3 and 479–13 were found resistant and rest of them were found susceptible. Kumar and Kohli (2001) reported that sixteen cultivars of pea (*Pisum sativum*) were screened against *Fusarium oxysporum* f. sp. *Pisi,* in which Arkel was most susceptible while accession DPR-3 was found most resistant. Inheritance of wilt resistance revealed that single dominant gene is governing the resistance. "Sanjunichi" and "Tsurunashiakahana" among the pea cultivars were found resistant cultivars (Mashita and Fukaya, 2006).

13.7.4.3 CHEMICAL CONTROL

Seed dressing with ceresan, dexon, captan, carbendazim, and benomyl has been found effective for the control of pea wilt (Gupta et al., 1989). Seed treatment of pea with Bavistin and Benlate gave complete control of *Fusarium oxysporum* f. sp. *pisi.* (Utikar et al., 1978). Shukla et al. (1979) tested the efficacy of some seed dressing fungicides for the control of pea wilt and reported that Bavistin have the best result in improving germination, reducing mortality and giving significant higher yield. Maheshwari et al. (1981) reported that seed dressing with Benomyl and Dithane M-45 and soil treatment with Phorate + Captan reduced plant mortality against *Fusarium oxysporum* f. sp. *pisi* and increased yield. The pathogen was also controlled by soaking pea seeds in a 1:1 combine's suspension of Captafol (0.1%) and Captan (0.2%) (Gangopadhyay and Kapoor, 1979). Sinha and Upadhyay (1990) tested 11 chemical compounds and found that pathogen growth was completely inhibited by Emisan-6 and Sulfex (80% S) at all concentrates tested. Wang et al. (1995) reported that seed treatment with fungicide like organomercurials, thiram or carbendazim in known to reduce seed borne inoculums of pathogen and has been recommended. Pandey and Upadhyay (1999) reported Bavistin was highly effective fungicide while *T. viride* and *T. harzianum* best among all bio-control agents. Integration of all bio-agents with Bavistin was not beneficial but bioagent + Thiram was highly effective. Verma and Dohroo (2002) conducted field experiments and reported that Bavistin treatment resulted in the highest mean seed germination, lowest pre emergence rot and highest yield with the lowest wilt incidence. Maheswari et al. (2008) reported that Carbendazim proved most effective fungitoxicant for checking the mycelial growth of *Fusarium oxysporum* F. sp. *lentis* (5.6 mm) followed by Captan (9.9 mm), Hexaconazole (12.5 mm) and Diniconazole (16.44 mm). Phosphoorganic insecticides are powerful cutinase inhibitors and inhibited cutinase released by *Fusarium oxysporum* F. sp. *Pisi* structures for infection (Koller et al., 1982). Carbendazim and thiophanate-methyl applied as seed treatment were highly effective in increasing fresh and dry weight, root, and shoot length, nitrogen content in pea plants and also improved seed germination and plant survival (Neweigy et al., 1985).

13.7.4.4 BOTANICALS

Sharma et al. (2003) reported the antifungal activity of different plant extracts against *Fusarium oxysporum* f. sp. *pisi in vitro* condition the leaf

extracts of *Datura stramonium* and *Azadirachta indica* had superior antifungal activity. Verma and Dohroo (2003) studied fungitoxic effect of different plant extracts against *Fusarium oxysporum* f. sp. *pisiin vitro* and found the leaf extract of garlic showed 100% inhibition followed by *ocimum* extract. Devi and Paul (2003, 2005) the fungitoxic activity of extract of 10 plant species was evaluated against the pea wilt caused by *Fusarium oxysporum* f. sp. *pisi.* The *Ranuculus muricatus* extract completely inhibited the growth of the wilt pathogen followed by Lantana, *Ocimum*, and *Datura*. Sahni and Saxena (2009) reported the antifungal activity of ethanolic extracts of medicinal plants were evaluated against *Fusarium oxysporum* f. sp. *pisi* by "Modified disc technique" and various plant extracts resulted inhibition on the growth of mycelium however bark of *Euphorbia nerifolia* exhibited absolute toxicity against the test fungus.

13.7.4.5 ESSENTIAL OIL

Sharma et al. (2003) reported the antifungal activity of different plant oils were evaluated against *Fusarium oxysporum* f. sp. *pisi in vitro* condition. The oils of *Datura stramonium* and *Azadirachta indica* have considerable antifungal activity against the wilt pathogen. Abo-El Seoud et al. (2005) reported essential oils of fennel, peppermint, caraway, eucalyptus, geranium, and lemon were tested for their antimicrobial activities against *Fusarium oxysporum* and essential oils of fennel, peppermint were selected as an active ingredient for the formulation of biocides due to their efficiency in controlling the tested *Fusarium oxysporum.* Sitara et al. (2008) reported essential oils extracted from the seed of neem, mustard, black cumin and asafetida were evaluated for their antifungal activity against *Fusarium oxysporum, F. moniliforme, F. nivale,* and *F. semitectum*.

13.7.4.6 BIOLOGICAL CONTROL

Pre-inoculation application of *Gliocladium roseum* provided good control of wilt of pea (Saksirirat et al., 1994). Velikanov et al. (1994) reported that *Trichoderma aureoviride, Trichoderma harzianum, T. viride* and *Gliocladiumvirens* were found to be hyperparasitic on both *Fusarium oxysporum* and *Fusarium solani.* Verma and Dohroo (2003) studied the efficacy of the fungal antagonists *Trichoderma harzianum* and *Trichodermaviride* against Fusarium wilt of pea caused by *Fusarium oxysporum* f. sp. *pisi in vitro*. *Trichoderma*

harzianum and *Trichoderma viride* showed the maximum growth inhibition of the wilt pathogen. Seed treatment with antagonists like *Trichoderma viride* and *Trichoderma harzianum* was found effective in the management of pea wilt both under glasshouse and field conditions (Verma and Dohroo, 2005c).

13.8 RUST

It is an important disease of garden pea particularly in wet areas of pea cultivation throughout the world. The rust fungus was first identified by Jordi in 1904 (Buchheim, 1922). In India, pea rust pathogen *U. viciae fabae* by Sydow and Butler on *Vicia faba* from Pusa, Bihar in 1906. The first report of its occurrence on pea in India was by Butler (1918). Two species of *Uromyces* have been reported to cause rust of pea. One of them *Uromyces pisi* (Persoon) de Bary, has been reported from several European countries (Deutelmoser, 1926; Jorstad, 1948). It is a heteroecious species having its aecial stage on *Euphorbia cyparissias* and rarely occurs in India, other species *U. viciae-fabae* (Pers.) has been found to cause pea rust in India (Prasada and Verma, 1948). The disease may cause epidemic proportions under favorable weather conditions resulting in considerable yield losses.

13.8.1 SYMPTOMS

The rust pustules appear on all green above-ground parts of the plant. The minute raised yellow rust pustules to appear on above-ground parts of the plants and most preferably on underside of the leaves and less abundant on the pods and stems. All the four stages develop on green part of the host, including the pods. The first symptoms appeared with the development of aecia. The yellow spots have aecia appear first on the undersurface of the leaves, stems, and petioles persist for longer time The formation of the aecial stage is preceded by a slight yellowing, which gradually turns brown. The uredopustules are powdery light brown in appearance. Late in season dark brown to black teleutopustules develop on the leaves but most commonly on stem and petioles.

13.8.2 CAUSAL ORGANISM

The disease is caused by two species of *Uromyces* viz, U. pisi (Pers.) de Bary and *U. viciae-fabae* (Schroet). Later one is worldwide distributed

pathogen of pea and also reported from faba bean (*Vicia faba* L.), lentil (*Lens culinaris* Medic.) and sweet pea (*Lathyrus sativus* L.) (Shroff and Chand, 2010). The fungus is an autoecious with aeciospores, urediospores, and teliospores on the surface of host plant and completes its life cycle on the same host. In India, urediospores are converted to teliospores under field condition during the month of March due to the higher temperature. Urediospores are short-lived while teliospores can survive in plant debris from one season to another (Hebblethwaite, 1983). Germination of teliospores takes place between 17 to 22°C temperatures and at the start of next season producing basidiospores which initiated new infection cycle (Joshi and Tripathi, 2012). The disease is favored by high humidity, cloudy, and rainy weather condition. Disease development in field condition is favored between 20°C to 22°C (Kushwaha et al., 2006). On peas the fungus starts its life cycle with the formation of pycnia, aecia, uredia, and finally telia. The yellow spots having aecia in round or elongated clusters are the earliest visible symptoms of the disease on the leaves. Pycnia occurs in small groups associated with the aecia. The aecia are cupulate and 0.3 to 0.4 mm in diameter. The peridium is short and whitish. The aeciospores are round to angular or elliptical with the hyaline wall 1 micron thick and verrucose. The aeciospores measures 14 to 22 microns in diameter. The uredia develop on both the sides of the leaves and on other parts of the plant. They represent a powdery light brown appearance. The urediospores are round to ovate, light-brown, echinulate with 3–4 equatorial germ pores and measure 20–30 (22–28) × 16–26 (19.22) microns. The telia occur in the same sorus as the uredia are developed from the same mycelium. The teliospores are dark-brown or black, subglobose, ovate or elliptical with rounded or flattened apex which is considerably thickened and appears papillate and measure 25–38 (40) × 18–27 micron. The mycelium in intercellular, branched, septate having yellowish or orange-red oil drops in the cytoplasm knob-like haustoria are formed in the host cell. The teliospores represent the sexual stage of the fungus and on germination give rise to a promycelium from each cell. The diploid nucleus passes into the promycelium, undergoes meiosis, and four haploid nuclei are formed. Each nucleated cell of the promycelium then produces short sterigmata at the tip of which swells to form a globular basidiospore in which the single nucleus form the cell of the promycelium moves. The basidium in the uredinales thus consists of two stages: the probasidium or hypobasidium (teliospore and epibasidium (promycelium). Polymorphism is the *Uromyces* spp. exhibited by development of following types of spore in the sequence listed:

1. **Stage 0:** Pycnia or spermogonia bearing pycniospores or spermatia and they are produced on a haploid thallus resulting from infection by basidiospores. This is the only monocaryotic stage of the rust mycelium on the host. They contain a palisade of sporogenous cells which cut off at their tips single-celled, uninucleate pycniospores in sweet nectar. The spore laden nectar is exuded from the pycnia and carried by insects. The spermatia affect fertilization by fusion with receptive or flexuous hyphae present in the mouth of pycnia of the apposite mating type. The pycnia are highly variable in shape, being globose, conical, hemispherical, lens shape or undefined shape without proper delimiting boundary.
2. **Stage I:** Aecia bearing aeciospores and are formed as a result of dicaryotization of the monocaryotic mycelium. They are associated with pycnia and also act as repeating spores or uredia. The aecia contain a palisade of binucleate sporogenous cells at their base. These cells invariably bear unicellular, binucleate, hyaline aeciospores in chains. They germinate to produce a dicaryotic mycelium initiating the dicaryon which bears the uredia and telia.
3. **Stage II:** Uredia or uredinia bearing urediospores or urediniospores. The uredia are sori produced by binucleate thallus and bearing one-celled urediospores singly on pedicel. The urediospores are binucleate with hyaline or colored walls and are perforated by conspicuous pores (germ pores). The wall is echinulated with pointed conical spines.
4. **Stage III:** Telia bearing teliospores or teleutospores and which are vary enormously. The spores may be single or multicellular, smooth, stalked or sessile and embedded in the host or free. The young teliospore is binucleate but at maturity it has a single diploid nucleus representing the diplophase in the life cycle.
5. **Stage IV:** Basidium or promycelium bearing basidiospores. It represents the transition of diplophase to haplophase by the germination of teliospore, meiosis of diploid nucleus and formation of haploid basidiospores.

13.8.3 DISEASE CYCLE AND EPIDEMIOLOGY

The rust pathogen is mainly soil-borne in nature as teliospores survive in crop debris. In India, the rust appears to survive on weed hosts belonging

to *Lathyrus*, *Vicia*, etc., and the spores are windblown to the main crop (Singh, 1987). Wild hosts may serve as primary or secondary source of infection. Aeciospores in *U. viciae-fabae* were found to be repeating spores and play an important role in outbreak of pea rust in north India. Inoculation of pea plants either by aeciospores or urediospores resulted in the production of aeciospores (Kushwaha et al., 2006).

Very little information is available on the effect of environmental factors on development of pea rust. Prasada and Verma (1948) reported that at relatively low temperature 17–22°C results in formation of secondary aecia while at 25° the infection causes development of uredia. No infection by aeciospores occurs at 30°C. These spores remain viable for 8, 6, 4, 3 and 2 weeks at temperatures of 3–8°C, 10–12°C, 17–18°C and 30°C, respectively. No viability is retained after 6 weeks showing that the aeciospores do not survive during the off season for the crop. Singh (1998) reported that optimum temperature for germination of uredospores is 16–25°C and no germination occurs at 28–29°C. In the plain of North India, where warm-season sets towards the end of March. These spores remain viable for 16–17 weeks, when stored at 3–8°C and only for 2 weeks at 36–37°C. Thus these spores do not survive in the hot summer interning two successive crop seasons. The teliospores of the rust fungus have been found to have no dormancy and can germinate at 12–22°C soon after their formation. Batra and Stavely (1994) working on *Uromyces appendiculatus* (Pers.) reported that the urediospore germinate at 15°C to 24°C while the teliospores in crop debris germinate at 10°C to 15°C under favorable conditions the spore complete the infection cycle within next 5–10 days. Recently Shroff and Chand (2010) reported that infection process of *Uromyces viciae fabae* started after deposition of aeciospores on the surface of pea leaves at a temperature 25–30°C and relative humidity (RH) of 99–100%. Kushwaha et al. (2006) observed that aeciospores in *Uromyces fabae* were found to be repeating spores and play an important role in pea rust outbreaks in the North Eastern Plain Zone (NEPZ) of India. Among the different growth stages of pea, the pod formation stage was highly susceptible and production the maximum number (744) aecidia/leaf at 20–25°C. Urediospore production mainly coincided with the senescence of the pea plants. Atmospheric temperatures around 20°C maximum and 5°C minimum with high RH (60–70% mean weekly) and light shower or drizzle favors for *U. viciae-fabae* development and spread. Maximum Temperature 25°C and minimum 7–8°C and less or more rains disfavor rust spread (Mittal, 1997).

13.8.4 MANAGEMENT

13.8.4.1 CULTURAL PRACTICES

Cultural practices mostly affect the environmental conditions favorable for host plant and natural enemy of pathogen and make unfavorable to pest and pathogens. The cultural practices may be very important tool in avoiding or reducing the inoculums/disease without any unwanted side effects like pollution. Generally, the disease appears late in crop season, and hence, the losses can be reduced by early planting. Delayed sowing after 5 October had increased the severity of rust severity and decreased grain yield. Cultivar 'khaparkheda' gave the highest seed yield (1.54 t/ha) and had the lowest severity (Sangar and Singh, 1994). Singh et al. (1996) reported that three pea cultivars were sown on 4 dates between 5 October and 4 December in a field trail and the severity of rust was increased as sowing delayed. Field sanitation to destroy diseased crop debris and long crop rotations avoiding broad beans, *Vicia, lathyrus* should be followed for minimizing losses from the disease (Singh, 1987). Efforts should be also made to locate and destroy the weed hosts.

13.8.4.2 HOST RESISTANCE

The evolvement of rust-resistant varieties seems to be the most effective, but there is a need for screening of existing lines/germplasm/cultivars against pea rust. Singh and Tripathi (2004) reported that KFP 106, DMR 11, HUP 8603, Type 163, and KPMR 22 showed a good level of resistance, which being conditioned by a number of genes. Xue and Warkentin (2002) observed that Tara and Century were the most resistant to both UF-1 and UF-3 isolates while Victoria and Topper were the most resistant to UF-2 only. Barilli et al. (2009) collected 2759 pea accessions and screened for resistance against pea rust. All accessions displayed a complete interaction (high infection type) both in adult plants under field condition and in seedlings under growth chamber conditions, but with varying levels of disease reduction and no complete resistance was observed. Khan et al. (2009) reported that among all tested pea cultivars only Climax gave the highest yield due to lowest disease severity and Meteor had the lowest yield due to maximum disease severity.

Induced resistance seems to a new approach for the control of pea rust. Walter and Murray (1992) observed that inoculation of the lowest two leaves of *Vicia faba* with urediospores of rust fungus *Uromyces viciae-fabae*. The resistance was seen as diminished infected areas on the leaves and as fewer urediospores for standard area for 29 days from challenge. The resistance was very high when first days separated the two inoculations but had disappeared when 12 days separated to two. In further experiments with plants at the same stage and using the same isolates of the rust fungus, Walter, and Murray (1992) found that treatment of the first two leaves with either 10 m^M tripotassium phosphate or 5m^M EDTA in place of rust inoculation also caused significant increase in resistance of the upper leaves to challenge inoculation with the rust fungus. In general resistant genotypes contained higher level of phenolics and susceptible ones had more sugar content.

13.8.4.3 CHEMICAL CONTROL

Chemical pesticides are backbone to control of rust disease in pulses and cereals crops till today. Spraying of fungicides alone or in combination has been considered necessary to provide adequate protection to the crop from rust incidence. Fungicides found effective against pea rust are Diathane M-45 (0.2v a.i.) and Calixin (0.2% a.i.). First spraying is done as soon as the disease appears in the field and three more sprays are given at ten days interval. Upadhyay and Gupta (1994) reported that Tridimefon, Maneb + Tridemorph were effective against rust disease under field conditions. Systemic sterol biosynthesis inhibiting fungicides were effective against *Uromyces viciae-fabae* (Fuzi, 1995). Khaled et al. (1995) reported that fungicides (benomyl) carboxin, metalaxyl, oxycarboxin, thiram, triadimefon, and triforine) alone or with Dithane M-45 (mencozeb) were effective against rust but Propiconazole (Tilt) gave the best control, reducing rust intensity and increased pod yield. Folicur and calixin were also effective against rust (Ayub et al., 1996). Gupta et al. (1998) observed that hexaconazole (0.1%) and difenoconazole (0.01%) were best against rust and increased yield. Mancozeb seed treatment was the most effective followed by carboxin and benomyl. Gupta and Shyam (2000) reported that seven ergostral biosynthesis inhibiting fungicides cyperconazole, flusilezole, propiconazole, and hexacanozole completely inhibited rust incidence and rust severity on leaves. Singh and Tripathi (2004) observed that Baycor

(0.1%) prophylactic 2 to 3 sprays at 15 days interval was found most effective in reducing the disease severity and appreciable increase in grain yield. Individual fortnightly foliar sprays, commenced when the disease appeared with carbendazim, score, Tebuconazole, and hexaconazole among systemic and at 10 days interval of antracol, microsul, and shareamong the nonsystemic fungicides proved effective for combating the rust disease and in ameliorating the crop yield (Sugha et al., 1998).

13.9 ASCOCHYTA BLIGHT

Ascochyta blight was first reported from the North Western Province of India presently in Pakistan (Butler, 1918). Ascochyta disease complex has been reported from Poland, Germany, Chile, India, Austria, East Africa, Bulgaria, Scandinavia, and Netherland (Kaiser et al., 1998). *P. medicaginis* var. *pinodella* associated with Ascochyta disease complex of pea was isolated from pea cultivar Lincoln from Bajaura, in Himachal Pradesh by Sagar and Bhardwaj (1997). Srivastava and Gupta (1990) reported pea blight (*Ascochyta pisi*) from Sikkim, where it caused heavy losses from December to March. Under favorable weather conditions, it causes significant yield losses. The disease may cause yield losses up to 40%, but sometimes in blight phase alone the losses may go up to 60% (Bretag et al., 1995; Tivoli et al., 1996).

13.9.1 SYMPTOMS

Ascochyta blight is characterized by presence of brown to purplish, irregular areas on the foliage. Under high moisture condition for long period, the lesions become circular and larger in size. The small, brown to purplish irregular spots appear on the pods and enlarge to irregular, purplish, large area could become blotched with the coalescing of lesions. The early symptoms on stem appear as black to purplish streak which is more pronounced at the nodes and could enlarge into brown to purplish irregular areas on the stem. Pycnidia are usually darker in color and produced on lesions especially on leaves and pods in characteristic ring pattern. Fruiting bodies, the pycnidia, form concentric rings, which is the characteristic symptom of the disease. The lesions are circular on leaves and pods, whereas these are elongated on stem and branches. The apical twigs, branches, and stems

often show girdling and plant parts above girdle portion are break-off even before drying. Lesions on pods are prominent and usually circular with dark margins. Pod infection often leads to seed infection through testa as well as cotyledons. Dark lesions with pycnidia in the concentric rings are formed even on the seed coat. In the field condition, disease appears in patches after 6–8 days of infection and rapidly spread to the entire field under congenial environmental conditions.

13.9.2 CAUSAL ORGANISM

Ascochyta blight disease complex consists of three pathogens, which include *Ascochyta pisi*, which causes spots on leaves and pods; *Ascochyta pinodes* (teleomorph *Mycosphaerella pinodes*) causes blight and *Phoma medicaginis* var. *pinodella* which causes foot rot. The fungus is homothallic. The ascostromata are globose with beaked ostioles. Asci are cylindrical-clavate with a wall made up of two membranes. The inner membrane is thickened at the tip and is provided with an apical pore. At the time of ascospores discharge the outer membrane is ruptured at the tip and the inner membrane stretches to approximately three times its length. The spores move towards the apex and when the membrane ruptures at the pore the spores are ejected and the stretched membrane contacts.

13.9.3 DISEASE CYCLE AND EPIDEMIOLOGY

Ascochyta blight is both externally and internally seed-borne. The fungus may be present on the seed surface, within seed coat, cotyledons, and embryo. The infested seeds are the main source carrying pathogens from one season to the next and one place to another. Infected seedling dies quite early and may serve as substrate for growth of the fungus and formation of pycnidia and conidia for secondary spread. In *M. pinodes* development of perithecia in crop refuse serves as another source of perennation. Development of pycnidia and perithecia of *M. pinodes* was studied on pea cultivar Solara under greenhouse and field conditions (Roger and Trivoli, 1996). Development and quantity of pycnidia were related to inoculums concentration and physiological state of the plants. Pycnidia were produced on both green and senescent organs while perithecia on senescent organs only. Spores trapping showed that both pycnispore dispersal and ascospore discharge were

initiated by rainfall or dew. Pycniospores and ascospores were dispersed through the growing season, indicating that ascospores also play an important role in secondary infections. Waterlogging of pea already infected with *M. pinodes* may result in more severe infection and greater reductions in plant growth, cultivars more sensitive to waterlogging may suffer greater losses from disease (McDonald and Dean, 1996).

Spore germination of *A. pisi* was 85–87% in a drop of water or at 100% RH when temperature was 20–21C (Susuri, 1976). Singh (1987) reported that the infection did not occur below 80% RH, but it rapidly increased above 90%**.** Singh et al. (2005) reported the significant effect of temperature, moisture duration, and their interaction on *Ascochyta* blight development. Temperature ranging between 20–30°C with moisture duration of 12–24 hours resulted in severe disease development.

13.9.4 MANAGEMENT

13.9.4.1 CULTURAL PRACTICES

The use of disease-free or healthy seed is very important for managing Ascochyta blight disease. Disease-free seed can be produced if the crop is grown in low rainfall areas. The disease can be reduced by following long crop rotation and by reduction of the crop refuse by burning either in the field or after threshing (Bedlan, 1985).

13.9.4.2 HOST RESISTANCE

Kavasnikov and Krotova (1977) reported, out of 260 genotypes of pea only 13 were showed resistant against *Ascochyta pisi*. Sandhu and Dhillon (1984) reported from Ludhiana and Gurudaspur that pea cultivars Pleiofila and ML-21 were moderately resistant to *M. pinodella*. Iqbal et al. (2001) found three lines 89P117-5, 88P022-6-28 and 88P0-6-29 of pea as highly resistant to blight (*A. pisi*). Pea lines Bartel, Brite, Bodil, Borek, Karo, Meteor, Rondo, Zolty Pomorski, KM01, KM02, KM03, Solara, Bohatyr, and Lu15/92 were found to be resistant against *M. pinodes.* Lines K1632, K3055, K5072, K5117, K5513, K6391, K7354, and K8195 (Vladimirtseva et al., 1990). Pea cultivar viz., Oscar, Pony Express and Ru/53 were found resistant to *Ascochyta pisi* (Obradovic et al., 1994).

13.9.4.3 BIOLOGICAL CONTROL

The effect of seed coating with antagonist *Pythium oligandrum* or fungicides (thiabendazole (TBZ) + fosetyl-aluminum + captan or TBZ + metaalxyl + thiram) under *in vitro* and *in vivo* conditions and found that *P. oligandrum* was an aggressive parasite of pathogens under *in vitro* condition but failed to control footrot under field conditions while fungicidal seed treatments significantly reduced foot rot under field condition (Bradshaw-Smith et al., 1991). Lacicowa and Pieta (1996) reported that seed dressing with *Trichoderma koningii* and *Gliocladium roseum* was found effective in protecting the seed from *Ascochyta pisi.* Pretorius et al. (2002) observed that crude *Eucomis autumnalis* bulb extract prevented the *M. pinnodes* spore infection of the leaves under *in vivo* conditions by inhibiting the spore germination and showed no phytotoxic reaction on the leaves. Four isolates were tested alone and in combinations for suppression of the disease and promotion of plant growth under field conditions. The mean of disease reduction with the most promising isolate 51 was 60% in foliar and 55% in the plant debris treatment. In addition to disease suppression, pseudomonads promoted plant growth in terms of increased plant height and grain yield. Moreover, pseudomonads were compatible with some fungicides at concentrations as high as 100 ppm in *vitro* condition.

13.9.4.4 CHEMICAL CONTROL

Seed treatment with fungicides is an effective measure to reduce the severity of disease. Seed treatment with fungicides viz., thiram, TBZs (Bretag, 1985) were found effective in reducing the seed-borne infection of Ascochyta spp. The seeds treated with cymoxanil + oxadixyl + carbendazim + thiram showed less rotting than the cymoxanil + oxadixyl treated seeds (Sanssene et al., 1998). Foliar application of fungicides such as TBZ (Bretag, 1985), copper oxychloride, chlorothalonil, and benomyl (Warkentin et al., 1996) have been found effective in reducing the severity of Ascochyta blight and increasing the yield and seed weight of pea. Single application of mancozeb at the early flowering stage was effective in reducing the disease severity and in increasing yield (Warkentin et al., 2000). Combined seed treatment with carbendazim and thiram and two foliar sprays each with mancozeb and dinocap reduced the severity of the disease and increased yield (Singh et al., 1992). EBI fungicides like Prochloraz were also found effective in reducing the disease and increasing yield (Nasir and Hoppe, 1997). Fungicides like

chlorothalonil and benomyl are found effective in managing Ascochyta blight epidemics (Warkentin et al., 1996).

KEYWORDS

- **ascochyta blight**
- **disease cycle**
- **Food and Agriculture Organization**
- **neem bark methanol extract**
- **northeastern plain zone**

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