Chapter 7
Root and Stem Rots

Root and stem rots are caused by several nematodal and fungal diseases, however, only one nematodal and seven fungal diseases are described in this chapter. Most of these diseases occur either alone or in combination with others.

7.1 Anthracnose

This disease is widely spread in cereals and grasses. It was reported to be important in soft winter wheat in 1914 (Chester 1950). Later several reports about its occurrence appeared in the literature. The disease can cause 100 % plant infections and can reduce yields by up to 25 %. In Brazil, the disease was reported in cvs. IAS 54 and IAS 58, in the States of Paraná and Rio Grande do Sul.

7.1.1 Symptoms

The disease appears at the spiking stage, on leaves and stems. Numerous black acervuli of the pathogen are observed especially on the lower portion of the stems at the time of host maturity (Fig. 7.1). Under microscopic examination setae and conidia of *Colletotrichum graminicola* can be observed. Severely infected plants may lodge and produce shriveled grains.

7.1.2 Causal Organism and Epidemiology

The disease is caused by *Colletotrichum graminicola* (Ces.) G. W. Wils. (Syn. *C. cereale* Mann). The fungus belongs to the order Melanconiales. The acervuli are oval, black and are produced superficially on the stems. The acervulus contains fusiform, curved, hyaline, unicellular conidia which measure 23–65 × 3–6.5 µm. After germination the conidia form appressoria and infect the host.

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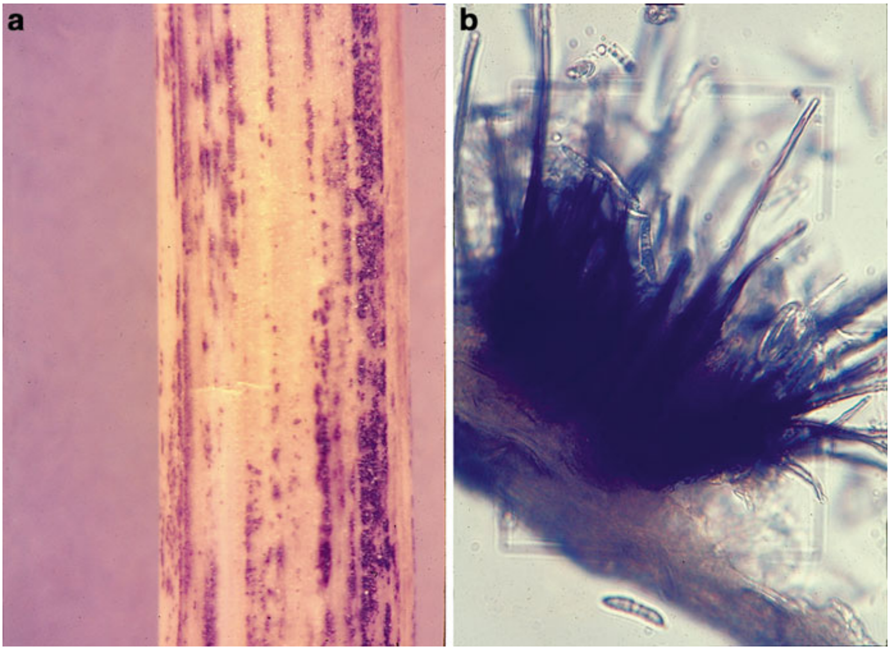

Fig. 7.1 Anthacnose (*Colletotrichum graminicola*). (**a**) symptoms on wheat stem; (**b**) acervulus and conidia

The fungus grows well on common artificial culture media and produces abundant sporulation. The sexual stage of the pathogen perhaps belongs to *Glomerrella* sp. (Politis 1975; Politis and Wheeler 1972; Mehta 1993; Bockus et al. 2010).

7.1.3 Control

C. graminicola survives in the soil and on the crop residue in the form of sclerotia. Although the disease can be seed transmitted, wind-borne conidia serve as the primary source of inoculum. Wheat infections occur under temperature between 20 and 25 °C. Fungicidal seed treatment and crop rotations may reduce the disease intensity. Being a disease of secondary importance, no specific control measures are recommended.

7.2 Common Root Rot

Common root rot of wheat occurs in a number of countries and its severity is reported in some countries like Africa, Australia, Bolivia, Brazil, Canada and the USA (Lindingham et al. 1973; Diehl 1979; Wildermuth 1986; Mehta and Gaudêncio 1991). Yield losses caused by this disease in Canada were estimated to be 6 %, whereas

Fig. 7.2 Common root rot of wheat. Healthy (*left*) and infected (*right*) root system

in Brazil they were about 20 %. Although the disease is widely distributed in Australia and the USA, exact losses in yield are not reported (Specht and Rush 1988; Duckez 1989).

7.2.1 Symptoms

Symptoms of common root rot are not easily visible in the field. The development of infected plants is retarded and the plants look yellowish. When the infected plants are removed from the soil, they show necrosis and dark brown to black discoloration of roots, crown and the sub-crown internode (Fig. 7.2). In the case of early infections the plants die prematurely.

7.2.2 Causal Organism and Epidemiology

The disease is caused by a complex of pathogens, like *Bipolaris sorokiniana* Sacc. In Sorok.; *Fusarium* spp.; *Gaeumannomyces graminis* (Sacc.) Arx and Olivar var. *tritici* Walker. However, the most predominant pathogen is *Bipolaris sorokiniana*.

The pathogen *B. sorokiniana* survives in the soil for a period of 3 years (Reis and Abrão 1983; Frank 1985; Reis 1986a, 1986b; Stack 1994). Infection of the root system occurs from the inoculum present in the soil as well as from the inoculum present in the seed (Kumar et al. 2002; Knight et al. 2010; Gyawali et al. 2012). The leaf blight phase and the root rot phase of the disease attained high severities during the 1980s. Mehta and Igarashi (1985a, 1985b) reported that during 9 years of seed health testing, the intensity of seed infection/contamination varied between zero and 94 %. According to these authors, the high incidence of seed infection was correlated with the occurrence of severe epidemics of the disease in the field during those years.

By and large, the disease intensity and incidence are greater in no-tillage cultivation than in conventional cultivation (Widermuth et al. 1997). Nonetheless, the number of propagules of *B. sorokiniana* was reported to be much lower in no-till cultivation than in the conventional cultivation. At this point, some of the questions remain unanswered, such as: What is the real importance of the quantity of propagules of *B. sorokiniana* in the soil? Does the disease severity only depends on the quantity of inoculum present in the soil? How important will be the seed and the stubble inoculum in relation to the soil inoculum?

The rate of disease progress and its intensity is drastically reduced as a result of heavy and continuous rains during the initial growth stages of the wheat plant. This is also observed by Lindingham et al. (1973). According to these author, the progressive decline in longevity of the spores was due to the progressive increase in the water-holding capacity (WHC) of the soil. The germination of the conidia was zero when the WHC was 88 %. Lindingham et al. (1973), reported that the disease severity increased after a long period of drought.

According to Mehta and Gaudêncio (1991), the intensity of common root rot was lower when wheat was cultivated with an interval of 2–3 years as compared to wheat cultivated every year in the same soil. As stated earlier, these authors reported that for every 1 % increase in root rot severity there was a corresponding yield loss of 46 kg ha^{-1} (See chapter on economic importance of diseases).

7.2.3 Control

Use of resistant cultivars is always emphasized. In Brazil, for example, the earlier semi-dwarf cultivars were very susceptible to common root rot and spot blotch. Since these cultivars were substituted by locally developed resistant ones during the early 90s, the disease is kept under control. When resistant cultivars are not available, deep plowing once every 2–3 years, seed treatment with fungicides and crop rotations with oats and some leguminous crops, may reduce the incidence of common root rot (see also chapter on spot blotch).

7.3 Fusarium Root Rot and Crown Rot

Fusarium root rot and crown rot may occur alone or in association with other organisms. It is a very common root disease and occurs especially in acidic soils. Under favorable conditions the disease may cause severe losses. According to Booth and Waterston (1964a, 1964b, 1971), losses between 50 and 70 % are registered. Epidemics of this disease have occurred in Australia, New Zealand, Canada, USA and France. Although the disease occurs all over the Latin American region, severe epidemics have not been registered (Liddell 1985; Mehta 1993; Monds et al. 2005; Smiley et al. 2005).

7.3.1 Symptoms

The initial symptoms of the disease are characterized by brown discoloration of the coleoptile. Depending on the species of *Fusarium*, the plants die rapidly. The infected plants are underdeveloped, produce white spikes with shriveled grains and when uprooted show pinkish to brown discoloration of the roots (Fig. 7.3). Under severe infections big patches of extremely weak plants are observed.

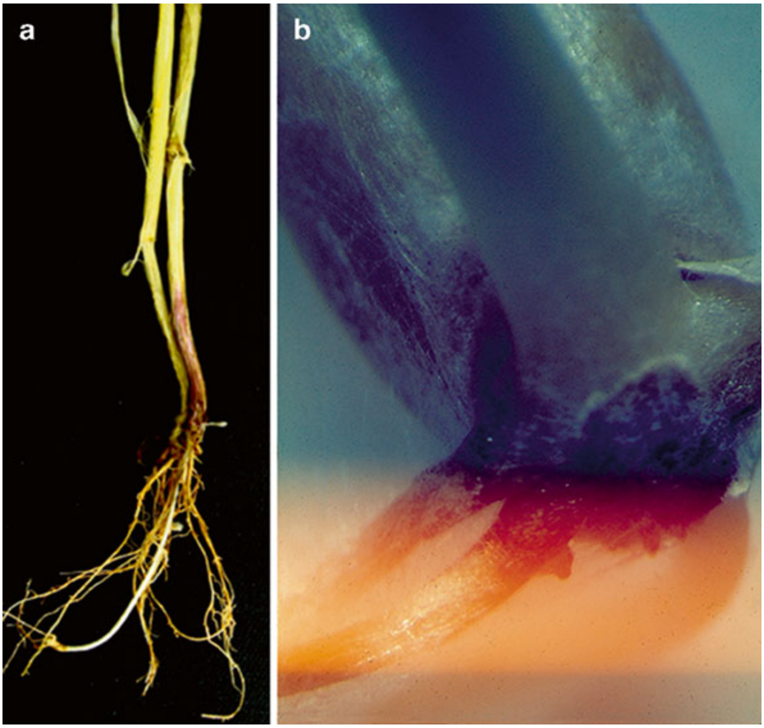

Fig. 7.3 (**a**) Symptoms of *Fusarium graminearum* root rot of wheat showing pinkish discoloration of the infected portion; (**b**) wheat crown infected with *F. culmorum*

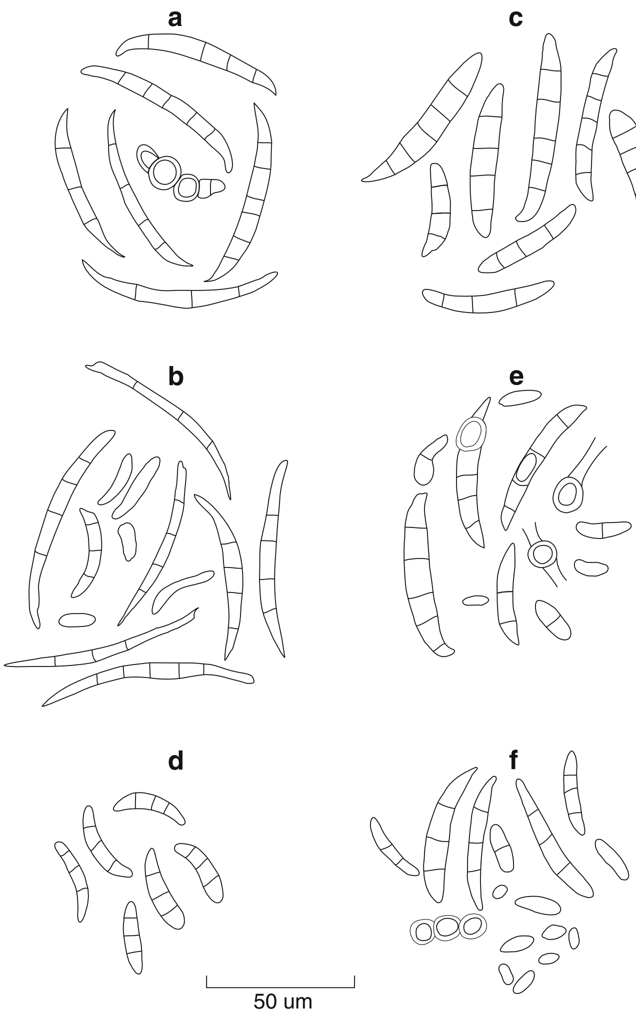

Fig. 7.4 *Fusarium* spp. causing Fusarium root rot. (**a**) macroconidia and clamydospore of *F. graminearum*; (**b**) macroconidia of *F. avenaceum*; (**c**) macroconidia of *F. culmorum*; (**d**) macroconidia of *F. nivale*; (**e**) macroconidia of *F. solani*; (**f**) macro and microconidia of *F. oxysporum*

7.3.2 Causal Organism and Epidemiology

Fusarium root rot is caused by at least six species of *Fusarium: Fusarium-graminearum* Schwabe; *F. avenaceum* (Corda ex. Fr.) Sacc.; *F. culmorum* (Smith), Sacc.; *F. oxysporum* Schlecht.; *F. solani* (Mart.) Sacc.; *F. nivale* (Fr.) Ces. (Fig. 7.4) (Nath et al. 1970; Smiley et al. 2005). According to Bockus et al. (2010) the three most important species are *F. graminearum*, *F. pseudograminearum* (O'Donnell and T. Aoki (telemorph *Gibberella coronicola* T. Aoki and O'Donnell) and *F. culmorum*.

F. oxysporum and *F. solani* are weak or secondary pathogens of wheat. *F. graminearum* is the predominant pathogen in the root rot syndrome of wheat.

F. culmorum produces yellow-red colonies in artificial culture media. Macroconidia are abundant, whereas microconidia are absent.

The macroconidia have a prominent foot cell, 3–5 septate, 4–7 × 25–50 µm. Clamydospores are present.

F. avenaceum (*Gibberella avenacea* Cook), occurs in almost all the countries, but it is more common in countries where a cool climate prevails during the wheat season. It occurs in Latin America including Brazil. Colonies on artificial culture medium appear reddish and as they age, become yellowish. Microconidia are curved, 1–3 septate and measure 3–4 × 850 µm. Macroconidia are uniform 3.5–4 × 40–80 µm, 4–7 septate with a foot cell (Bockus et al. 2010).

F. graminearum develops well on artificial culture media at 20–25 °C. On PDA the fungus produces pinkish pigmentation. Conidia develop on brown mycelium. Sporodochia are uncommon, but sometimes can be observed. They are whitish, containing numerous macroconidia. The macroconidia are sickle-shaped, curved with rounded epical cell and well-marked foot cell and measure 16–39 × 3-5 µm. The perithecia are globose, light orange with gelatinous appearance. The asci are cylindrical, clavate, contain eight ascospores and measure 60-80 × 8-21 µm. The ascospores are uni-septate, ellipsoid and measure 11–18 × 4–7 µm.

F. nivale is very much distinct from the other *Fusarium* species. The colonies are white to slightly yellowish. The mycelium is dense and the conidia are dispersed, abundant, curved, 1–3 septate and measure 10–30 × 2–5 µm. The mycelial colonies appear as white to peach-colored because of the pinkish sporodochia. This fungus has no microconidia or clamydospores. The macroconidia are small, measure 2.8–4 × 16–25 µm and are 1–3 septate. The perfect stage of the fungus belongs to *Calonectria nivale* Schaf. (*Monographella nivalis).* The perithecia are immersed, globose and contain clavateascospores and measure 60–70 × 609 µm. The ascospores are ellipsoidal, 1–2 septate and measure 10–17 × 3.5–4.5 µm.

F. oxysporum and *F. solani* are weakly pathogenic on wheat, whereas *F. culmorum* and *F. graminearum* are of special interest and may cause significant yield losses in wheat. Severely infected seeds are shriveled and are eliminated during the seed processing, whereas slightly contaminated seeds may add inoculum to uninfested soils. All the six *Fusarium* species have a wide host range and survive in the soil and on crop residue. Physiologic specialization is common in *Fusarium* species. Singh et al. (1991) have published a practical methodology for identification of Aspegilli, Fusaria and Penicillia, transmitted through seed.

7.3.3 Control

The *Fusarium* spp. are seed transmitted and also survive in the soil. Fungicidal seed treatment and deep plowing help reduce the soil inoculum. Excessive application of nitrogen predisposes the plant to *Fusarium* spp. infection (Toussoun et al. 1960; Weinke 1962; Smiley et al. 1972).

7.4 Pythium Root Rot

Pythium root rot is a soil-borne fungal disease. The disease is more severe wherever wheat is cultivated in humid and acid soils. Pythium root rot occurs in several wheat growing countries but it is more severe in some parts of the United States and Australia. Pythium root rot is known to cause substantial yield losses to pastures, all major cereal crops including wheat, brassica and pulse crops.

7.4.1 Symptoms

The symptoms of the disease are characterized by browning and necrosis of roots which ultimately causes reduction in plant height. The infected plants become yellowish due to lack of nutrient absorption and the disease appears as yellowish patches in the field. In the field the disease symptoms are nonspecific. The symptoms are usually confused with those of Rhizoctonia root rot. Pythium root rot could also be associated with other soil-borne pathogens like *Rhizoctonia solani*, and *Gaeummanomyces graminis* (Fig. 7.5).

Pythium root rot is also referred as "Damping off" caused by infection of seedlings during the earlier growth stages of the plant resulting in reduced emergence, decrease in root mass and yellowing of plants. Other than "Damping off", the pathogen can attack at all growth stages of the plant and hence in recent years it is not regarded solely as a seeding disease. and could appear in patches similar to the Rhizoctonia root rot. Infected plants show reduced root system with yellow to light brown discoloration of lateral roots and root tips. Grain formation in infected plants is severely affected.

7.4.2 Causal Organism and Epidemiology

Pythium root rot is caused by several species of *Pythium* either alone or in combination. So far 30 species of *Pythium* attacking wheat and barley are known (Paulitz 2010).

According to Chamswarng and Cook (1985), out of 10 *Pythium* species infecting wheat *P. ultimum* and *P. regulare* were most virulent on wheat. Later, Higginbotham et al. (2004), detected differences in virulence among species and among isolates within species. They reported that isolates *P. debaryanum* 90136 and *P. ultimum* 90038 were the most virulent whereas *P. rostratum*, *P. heterothallicum* and *P. intermedium* were the least virulent among species tested. Recently, Paulitz (2010), reported 13 species are associated with wheat, the most predominant species being: *P. aristosporum*; *P. arrhenomanes*; *P. debaryanum; P. graminicola*; *P. irregulare* and *P. ultimum*.

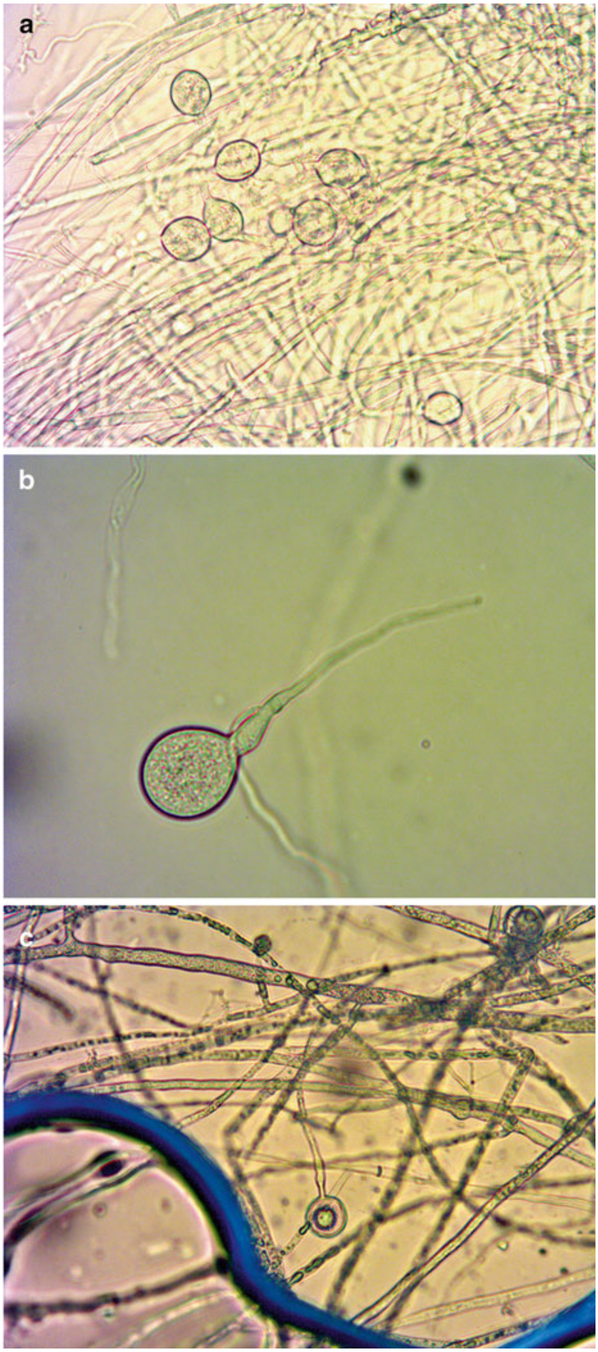

Fig. 7.5 Pythium root rot. (**a**) sporangia; (**b**) sporangium germination; (**c**) oospore

The pathogen survives in the soil and infects germinating seed and roots continuously throughout the growing season. The disease is normally severe in no-tillage cultivation (Bockus and Shroyer 1998; Cook et al. 1980; Cook 2001; Cook et al. 1990; Pankhurst et al. 1995; Schroeder and Paulitz 2006). The pathogen grows rapidly on the left-over stubble specially in the no-tillage cultivation and produces abundant quantities of spores. Spores are also produced on infected plant tissues.

The pathogen produces spherical to lobate asexual sporangia. Each sporangium contains several zoospores having two flagella. It also produces thick-walled sexual oospores. The oospores are uninucleate (Fig. 7.5), can remain dormant and can survive in the soil for a very long period and serve as primary inoculum. Upon germination the oospores produce zoospores which in turn infect the embryo of germinating seed, roots, especially the root tips and root hairs (Bruehl 1953; Cook et al. 1987). The oospores can also infect the roots directly after germination. The measurements of sporangia and oospores are very variable and depend on the species of *Pythium* (Paulitz 2010). Normally, the sporangia are bigger than the oospores and are >140 µm whereas the oospores are approximately 40 µm in diameter.

7.4.3 Control

Crop rotation is one of the best methods to reduce the incidence of Pythium root rot. Wheat cultivars resistant to Pythium root rot are not known. However different species of plants show different levels of resistance. Although cereal crops like wheat and barley are less susceptible than canola for example, continuous cropping with wheat should be avoided in heavily infested soils. In such soils wheat could be grown with an interval of 2–3 years. According to Lawrence and Harvey (2006), grain legumes are most susceptible to infection followed by canola, wheat and barley. For pasture, ryegrass could be a better choice.

Chemical seed treatments with metalaxyl-based fungicides if locally registered, can help reduce the incidence of "Damping off" but do not protect the plant further during its later growth stages. According to Lawrence and Harvey (2006), treating wheat with metalaxyl improved crop emergence by 36 % and root infection by 51 %. Other than chemicals, seed treatment with potential microbial inoculant-savailable in the market offer new perspectives (Weller and Cook 1986a, 1986b).

An integrated disease management approach such as avoidance of pirated seed, seed treatment with chemicals and or biological crop protectants, and adequate crop rotation would reduce crop losses.

7.5 Rhizoctonia Root Rot

Rhizoctonia attacking crown and roots of cultivated plants has been known for a long time. The disease is also referred to as Rhizoctonia root rot and "bare patch". It is more severe in oats, wheat and rye (Pitt 1964a, 1964b). The importance of the disease is recognized in Australia (Roget et al. 1987), in the USA (Weller and Cook 1986a, 1986b) and in the South Asia (Hobbs et al. 1988). In wheat the disease may occur alone or in combination with take-all and nematodes in the USA, Australia and in parts of Europe. There are no reports about the yield losses caused by this disease, but it is known that no grains are formed in infected individual plants.

7.5.1 Symptoms

Symptoms of culm infections are very common. Lesions of the disease appear on just above ground portion of the stem (lower leaf sheaths). Initially, they are elliptical or irregular of about 1 cm in length, brown with a whitish center and dark brown margin and are referred to as "sharp eyespot". Sometimes the lesions are observed only after removing the leaf sheath (Fig. 7.6a–c). In the case of severe infections the roots and the crown become black, the root system is severely damaged and the plants can be uprooted easily. Sometimes black sclerotia develop between the culm and the leaf sheath. As with take-all disease the infected plants are short with white heads, may be scattered in the field or are noticed as patches of groups of plants.

7.5.2 Causal Organism and Epidemiology

The disease is caused by *Rhizoctonia solani* Kuhn. Some strains are perfect stages of *Thanatephorus cucumeris* (Frank) Donk [Syns. *Pellicularia filamentosa* (Pat.) Rog. *Corticium solani* Prill and Delacr.]. Since the perfect stage of the fungus is not commonly observed on infected plants, the pathogen is normally referred as *R. solani*. The perfect stage is not observed on wheat plants. Since there is no sporulation, the fungus is identified by its growing habits. The fungal mycelium is white to light brown and branched at right angles (Fig. 7.6d). Near the branching a slight constriction and a septum can be seen which are the diagnostic characteristics of the pathogen.

Rhizoctonia solani has a very wide host range. Almost all the host belonging to the Gramineae are susceptible to this pathogen. Many isolates from wheat are aggressive in grasses and dicotyledonous plants such as potatoes, peas and soybeans (Bockus et al. 2010).

Infection can occur at any stage of the plant. Infections at the early stages of the plant growth are fatal. Infections after heading may result in shriveled grains but not total loss of grain. The disease occurs at a wide range of temperature and soil pH.

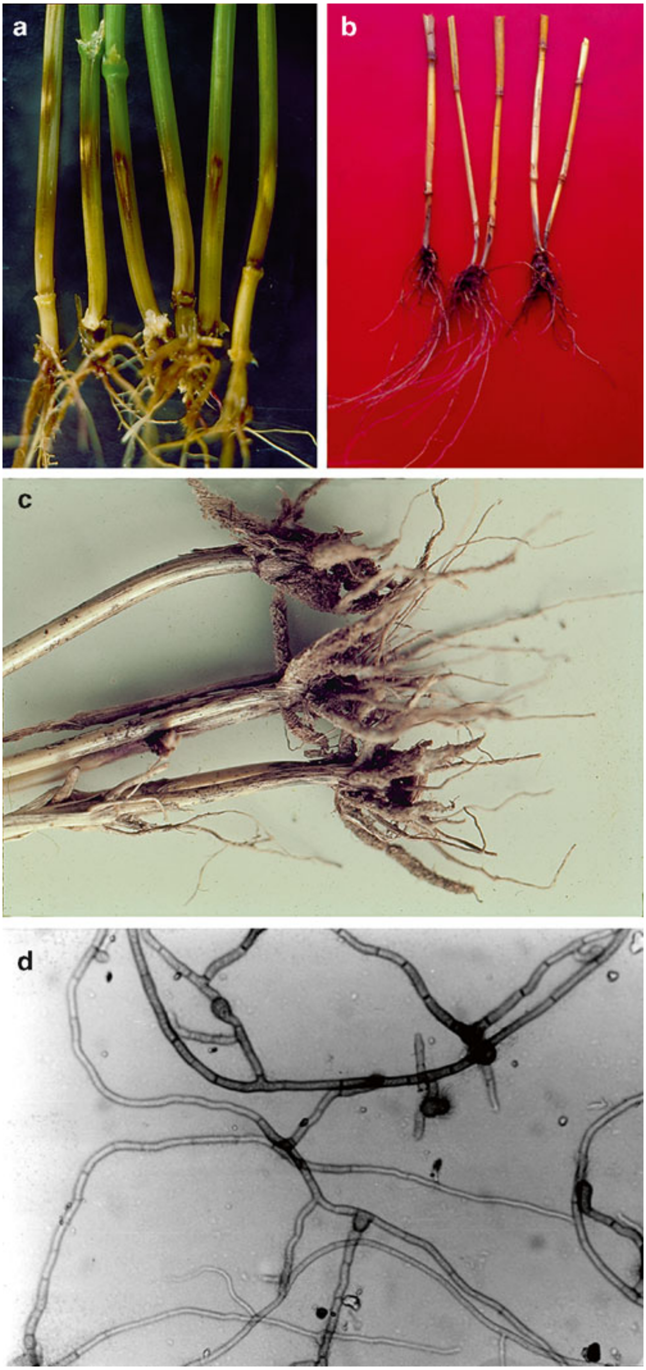

Fig. 7.6 Symptoms of Rhizoctonia root rot. (**a**) infection on the lower part of wheat stem showing elliptical lesions; (**b**, **c**) severe infection on roots and on the lower part of stems; (**d**) mycelium of *Rhizoctonia solani*. Note the branching of mycelium at right angles and constriction at the point of branching

7.5.3 Control

Considering the fact that the pathogen is soil-borne and has a very wide host range, its control becomes very problematic. Crop rotation with non-host crop or with less susceptible crop for 4–5 years would reduce the severity of infection. Rovira (1986) and Weller and Cook (1986a, 1986b), reported that the disease was more severe with no-tillage cultivation than the conventional system of cultivation. No chemicals for soil treatment are recommended. Some strains of bacteria like *Streptomyces* and *Bacillus* are antagonists to the pathogen and offer hope for biological control (Merriman et al. 1974a, 1974b).

Marvodi et al. (2012) identified eleven strains of *Pseudomonas* spp. as biological control agents with broad spectrum activity against pathogenic soil-borne fungi and pests including Rhizoctonia and Pythium root rot of wheat. Their studies indicated that the strains differed in potential antifungal metabolite activities and could be useful for long term development of integrated management of soil-borne diseases of wheat.

7.6 Root Lesion Nematodes

More than 10 species of nematodes are pathogenic to wheat. In some cases yield losses may reach up to 50 %. Descriptions of the nematodal diseases are beyond the scope of this book and so only one nematode disease is briefly described here, since it occurs on wheat in association with a fungal disease caused by *Rhizoctonia solani*. In Brazil, a disease complex caused by *Pratylenchus* spp. and *R. solani* has been reported.

7.6.1 Symptoms

Nematodes attack the wheat plant through its roots and cause necrosis of the root system. Infection caused by nematodes predisposes the plant to infection with *R. solani* (Jenkins 1948). Upon microscopic examination of the infected roots, motile nematodes can be observed. In some cases mycelia of *R. solani* can be observed together with the nematodes (Benedicts and Mountain 1956).

7.6.2 Causal Organism

Wheat may be attacked by a number of *Pratylenchus* spp, descriptions of some of these species on wheat are available (Bockus et al. 2010). It is not clear whether in the disease complex the nematodes invade roots first and thus predispose the plant to *R. solani* infection or vice versa. Nematodes are motile in the soil and invade the root system without forming galls or cysts.

7.6.3 Control

Control of the nematodes is very difficult since the host range of this nematode is very wide. Crop rotation with a non-host for a few years may help reduce the nematodal populations in the field. Soil fumigation is antieconomical and not practicable for large areas.

7.7 Sclerotium Root and Crown Rot

Sclerotium root and crown rot also known as Southerm blight of wheat is reported in some Latin American countries such as Bolivia, Brazil, Equator and Peru. It is also reported in some Asian countries (Igarashi et al. 1983; Dubin 1985). The occurrence of the disease is very frequent in Santa Cruz de la Sierra, Bolivia, especially in waterlogged areas (Mehta 1993). In the State of Paraná, Brazil, severe infections of this disease in commercial soybean fields have obliged some farmers to plow down their fields at the initial growth stages of soybean. Punja (1988), has presented an extensive revision of literature about this disease. According to Hobbs et al. (1988), yield losses caused by this disease could be as high as 30 %.

7.7.1 Symptoms

In some fields, infected plants appear short and with premature senescence. The infected plants can be easily up rooted because of the deteriorated and debilitated root system (Fig. 7.7). Infected plants show presence of white mycelia as well as white or light brown sclerotia which are the diagnostic features of the disease. Lesions on the first above ground short internode are at first linear and soon coalesce forming irregular patches almost circumflexing the culm (Igarashi et al. 1983).

7.7.2 Causal Organism and Epidemiology

The disease is caused by *Sclerotium rolfsii* Sacc. The fungus forms compact mass of mycelia known as sclerotia which measure 0.5–2.0 mm in diameter. The sclerotia are at first white and later become brown and may remain covered with the leaf sheath. Pinheiros et al. (2010) studied development of *Sclerotium rolfsii* sclerotia on soybean, corn and wheat straw and reported that sclerotia were produced on all types of straw. However, wheat straw produced the lowest amount of sclerotia.

Sclerotium root and crown rot is severe in humid and hot areas. The fungus is soil-borne. Sclerotia germinate at temperatures between 27 and 30 °C. After germination they form mycelium, basidia and basidiospores. The mycelial growth, formation

Fig. 7.7 *Sclerotium rolfsii* root and crown rot. (**a**, **b**) infected wheat plants; (**c**) formation of sclerotia in the leaf sheath; (**d**) sclerotial formation on filter paper 10–12 days after incubation of lower portion of infected wheat stems in a moist chamber

and germination of sclerotium is abundant within the first 8–10 cm of the soil and at deeper levels the germination is inhibited (Punja 1985, 1988). Sclerotia are formed on the moist filter paper 10–12 days after incubation of the lower portion of infected wheat stem in a moist chamber (Fig. 7.7d). The pathogen attacks monocotyledonous and dicotyledonous plants encompassing about 500 species of plants (Punja 1988). The disease spreads from one plant to another by root contact and spreads from one field to another through the agricultural implements.

7.7.3 Control

Although the disease is observed in some wheat and soybean fields, in Brazil, its occurrence is sporadic. It is favored by high moisture and high temperatures and higher severities are expected in no-tillage cultivation than in the traditional system of cultivation. Deep plowing reduces the severity of the disease (Punja 1988). Thus in highly infested areas, some modification in no-tillage cultivation may become necessary. Deep plowing of soils under no-tillage cultivation every 3–4 years may be beneficial.

7.8 Take-All

The take-all disease of wheat is a root and basal stem (foot) disease and is known to have occurred in several countries for over 100 years. In 1902 McAlpine identified *Ophiobolus graminis* as a pathogen of take all disease for the first time (Quisenberry and Rietz 1967). Since then interest was created to study this disease in Australia. Losses from this diseases could be over 50 % (Bockus et al. 2010). The disease occurs in the State of Rio Grande do Sul, Brazil, especially in acidic soils low in fertility where the loss can reach 100 %. In the State of Paraná, Brazil, for example, in 1975, one wheat fields of 200 ha was completely destroyed due to take-all disease (Mehta 1993). Such reports are rare, however, losses in Brazil could be between 20 and 30 %, in some fields.

7.8.1 Symptoms

The disease is characterized by foot and root rot. Symptoms are apparent at the time of heading when the spikes turn white and produce a few shriveled grains or no grains, depending upon the severity of infection. Normally, the disease is noticed in the field as white patches of infected plants referred as "whiteheads" (Milus et al. 2009) (Fig. 7.8). In less severely infested fields, individual plants with white spikes

Fig. 7.8 Wheat field severely infested with take-all fungus (*Gaeumannomyces graminis)* showing white patches

distributed in the field can be observed. Infected plants are shorter than the healthy plants and are easily uprooted. The crown portion and the roots become black due to the infected tissue necrosis. In early stages of infection no perithecia are observed on the infected parts of the basal stem, but at later stages of infection, erumpent hook like perithecia can be observed with a hand lens. Perithecia can be easily examined under a stereomicroscope after washing the infected portion. The perithecia are hard and are not easily removed.

7.8.2 Causal Organism and Epidemiology

The take-all fungus was initially known as *Ophiobolus graminis* Sacc. (Fig. 7.9) but later it was transferred to *Gaeumannomyces graminis* (Sacc.) Arx and Oliv. Wheat take-all fungus is designated as *G. graminis* var. *tritici*. It is a soil fungus and belongs to the class of ascomycetes. The perithecia are erumpent, black and measure up-to 400 µm in diameter. The infection pads (hyphopodia) are unitunicate asci containing eight hyaline ascospores. The ascospores are 5–7 septate and measure 3×70–80 µm (Fig. 7.10). The hyphopodia are somewhat like appressoria and infect the host via hyphal peg. The fungus can be easily grown on common culture media. Infected plants sometimes show presence of the fungus *Thanatephorus cucumeris* and the presence of both fungi represents the symbiotic relationship between them.

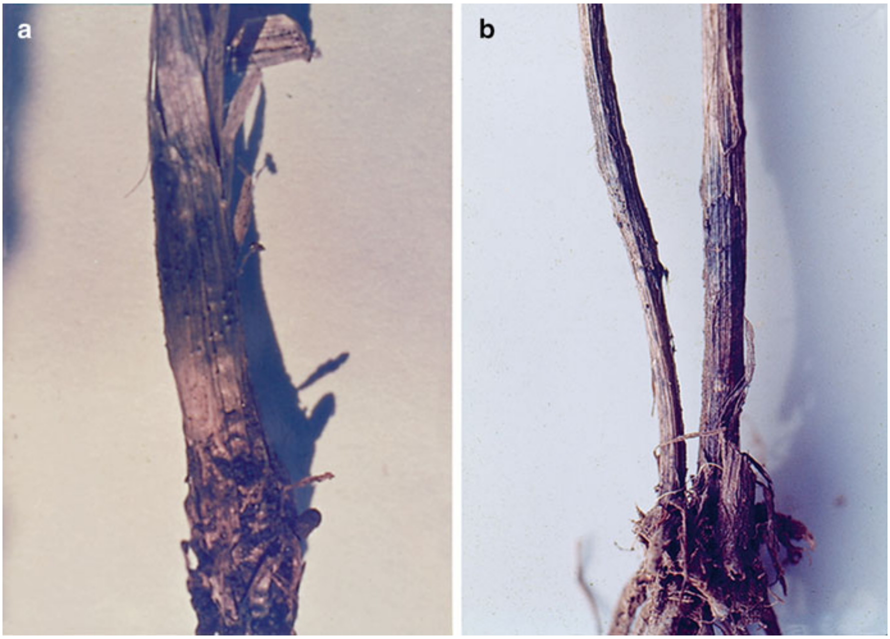

Fig. 7.9 (**a**, **b**) Wheat plants infected with *G. graminis* showing presence of black, hook-like perithecia

Fig. 7.10 Asci and ascospores of *G. graminis*

G. graminis is a soil-borne pathogen and survives as a saprophyte in the soil (Butler and Jones 1955; Warcup 1957; Bockus et al. 2010). Recent works corroborating much of the previous research conducted on this pathogen. The spread of the disease is through the hyphae from one plant to another and through agricultural implements from one field to another. Infection through ascospores is also reported (Garrett and Mann 1948). The ascospores are released through rain and wind splashing them onto nearby healthy plants. A single infected plant can spread the disease to at least another 50 plants (Wehrle and Ogilve 1956). Crop residue of the host plant gives shelter for the survival of the pathogen. Infection can occur at any stage of the plant. The inoculum density of the fungus is high in the first 20 cm of the soil layer, but fungal propagules are also found below this level (Mehta 1993). The fungus first infects the terminal roots and later the crown of the newly emerged seedling. The pathogen *G. graminis* also infects barley and some other species of Gramineae such as *Agropyron repens*, *Holcus lanatus* and *Agrotissto lonifera* (Daval et al. 2010). In the absence of host plant the fungus does not multiply in the soil. Thus, crop rotation with oats, maize and other legumes is recommended especially for heavily infested soils. The disease becomes severe under a monocropping system like wheat after wheat or wheat-soybean-wheat.

Alkaline soils deficient in nitrogen and phosphorus favor the disease more than the acid soils. This is because the concentration of $C0_2$ near the roots in acidic soils is much higher than in alkaline soils thereby restricting, the development of the pathogen. The wheat plant develops well at pH between 5 and 5.5, whereas the *G. graminis* develops well at pH 5.5–7.0. In general, extremely cold weather delays the process of decomposition of organic matter which in turn favors longer survival of the pathogen in the crop residue. The optimal soil temperatures for infection are between 18 and 25 °C. However, soil moisture is essential for the dissemination of the disease. The disease is more severe in Chile where annual rainfall is around 449–1,600 mm.

7.8.3 Control

Little information is available as regards the level of resistance of wheat cultivars. Some oat cultivars are less susceptible than others. Take-all is rather difficult to control because being a soil-borne disease there are no effective fungicides. Besides, complete resistance in wheat is not available (Bithell et al. 2011).

Susceptibility to take-all of cereal and grass species, and their effects on pathogen inoculum was studied by Bithell et al. (2011). They reported that the grass and the cereal species differed in susceptibility to take-all, in their impact on the pathogen multiplication and in associated take-all severity in following wheat crop.

The disease can be managed through cultural practices. Crop rotations of 1–2 years with non-host plant species reduce soil infection (Bailey et al. 2005). Milus et al. (2009), reported that for dryland fields summer fallow was the best option for managing take-all, whereas for irrigated fields, rotation out of wheat for at least 1 year reduced incidence and severity of take-all and rice was the most effective rota-

Fig. 7.11 Lime application in a commercial wheat field

tional crop. Bockus et al. (1994) concluded that temperatures ≥35 °C for 6 h on 12 days inactivate take-all inoculum. The disease could be severe in some cases where wheat is followed by alfalfa, soybeans and grass crops (Bockus et al. 2010). Other than crop rotation, deep plowing would bury the soil inoculum and help reduce the severity of the disease.

The severity of the disease is directly correlated with the application of excessive amounts of lime to acidic soils (Fig. 7.11). Thus application of large quantities of lime at one single time should be avoided. A specific source of nitrogenous fertilizer can increase the severity of the disease. Ammonium nitrate, at all levels of nitrogen, increases the severity of the disease as compared to the use of ammonium sulphate.

The system of cultivation affects the severity of take-all. According to Moore and Cook (1984), severity of take-all is normally greater in no-tillage cultivation than in conventional cultivation.

Normally, disease incidence goes on increasing during the first few years of wheat cultivation. Later, after 3–4 years, the disease incidence goes on decreasing gradually because of the increase of soil microorganisms antagonistic to *G. graminis* (Shipton 1972; Chng et al. 2013). This phenomenon is referred to as "take-all decline". Cook (1984), reported that the disease can be controlled through seed treatment with *Pseudomonas* spp.

It is known that *Didymella exitiales* normally present in the soil rhizosphere, can parasitize *G. graminis*. Specific antagonism by *Phialophorara dicicola* was demonstrated by Deacon (1974). Such discoveries open new perspectives for biological control of *G. graminis.*

Milus et al. (2009) studied susceptibility of inoculated, cool-season and warm-season grassy weeds to take-all in growth chamber and reported that all grasses supported colonization by the take-all pathogen.

Selected References

Angus JF, gardner PA, Pitson GD, Wong PTW (1998) A comparison of six methods to control take-all in wheat. Aust J Agric Res 49:1225–1240

Asher MJC, Shipton PJ (eds) (1981) Biology and control of take-all. Academic, New York

Bailey DJ, Paveley N, Pillinger C, Foulkes J, Spink J, Gilligan CA (2005) Epidemiology and chemical control of take-all on seminal and adventitious roots of wheat. Phytopathology 95:62–98

Benedicts WG, Mountain WB (1956) Studies on the etiology of a root rot of winter wheat in south-western Ontario. Can J Bot 34:159–174

Bithell SL, Butler RC, Harrow S, McKay A, Cromey MG (2011) Susceptibility to tale-all of cereal and grass species and their effects on pathogen inoculum. Ann Appl Biol 159:252–266

Bockus WW, Shroyer JP (1998) The impact of reduced tillage on soilborne plant pathogens. Ann Rev Phytopathol 36:485–500

Bockus WW, Davis MA, Norman BL (1994) Effect of soil shading by surface residues during summer fallow on take-all of winter wheat. Plant Dis 78:50–54

Bockus WW, Bowden RL, Hunger RM, Murray TD, Smiley RW (2010) Compendium of wheat diseases and pests. American Phytopathological Society, St. Paul, p 171

Boosales MG (1962) Precocious sporulation and longevity of conidia of *Helminthosporium sativum* in soil. Phytopathology 52:1172–1177

Booth C (1971) The genus *Fusarium*. Comm. Mycol. Inst. England, 237 pp

Booth C, Waterston JM (1964a) *Fusarium avenaceum*. C.M.I. Descriptions of pathogenic fungi and bacteria. No. 28, Comm. Mycol. Inst. England.

Booth C, Waterston JM (1964b) *Fusarium solani*. C.M.I. Descriptions of pathogenic fungi and bacteria.No. 30, Comm. Mycol. Inst. England.

Bruehl GW (1953) Pythium root rot of barley and wheat. U.S. Deptartment of Agriculture. Technical Bulletin No. 084.

Butler EJ, Jones SC (1955) Plant pathology. Macmillan, New York, p 977

Caetano VR, Pierobom CR (1972) Os problemas sanitários do sistema radicular do trigo. Indicação de pesquisa MA/IPEAS, Pelotas, Brasil 32:1

Chamswarng C, Cook RJ (1985) Identification and comparative pathogenicity of *Pythium* species from wheat roots and wheat-field soils in the Pacific Northwest. Phytopathology 75:821–827

Chester KS (1950) Nature and prevention of plant diseases. McGraw Hill, New York, 525 pp

Chinn SHF (1978) Influence of seed treatment with imazalil on common root rot of winter wheat. Plant Dis 70:857–859

Chng SF, Stewart A, Cromey MG, Dodd SL, Butler RC, Jaspers MV (2013) Effects of different rates of *Gaeumannomyces graminis* var. *tritici* inoculum for detecting take-all suppression in soils. Aust Pl Pathol 42:103–109

Choppakatla V, Hunger RM, Mclout HA (2006) First report of seed-ling blight caused by *Sclerotium rolfsii* on wheat in Oklahoma. Plant Dis 90:986

Christensen JJ (1925) Physiologic specialization and mutation in *Helminthosporium sativum*. Phytopathology 15:785–795

Christensen JJ (1929) The influences of the temperature in the frequency of mutation in *Helminthosporium sativum*. Phytopathology 19:155–162

Colbach RJ, Lucas P, Meynard JM (1997) Influence of crop management on take-all development and disease cycles on winter wheat. Phytopathology 87:26–32

Cook RJ (1967) *Gibberella avenacea* sp. nov. perfect state of *Fusarium roseum* f. sp. *cereals* "*Avenaceum*". Phytopathology 57:732–736
Cook RJ (1968) Fusarium root and foot rot of cereals in the Pacific Northwest. Phytopathology 58:127–131
Cook RJ (1980) Fusarium root rot of wheat and its control in the Pacific Northwest. Plant Dis 64:1061–1066
Cook RJ (1984) Root health: Importance and relationship to farming practices. In: Organic farming: current technology and its role in sustainable agriculture. Madison, 111–127
Cook RJ (2001) Management of wheat and barley root diseases in modern farming systems. Aust Pl Pathol 30:119–126
Cook RJ, Zhang BX (1985) Degree of sensitivity to metalaxyl within the *Pythium* spp. Pathogenic to wheat in the Pacific Noth-west. Plant Dis 69:686–688
Cook RJ, Sitton JW, Waldher JT (1980) Evidence for *Pythium* as a pathogen of direct-drilled wheat in the Pacific Northwest. Plant Dis 64:102–103
Cook RJ, Sitton JW, Haglund WA (1987) Influence of soil treatment on growth and yield of wheat and implications for control of Pythium root rot. Phytopathology 77:1192–1198
Cook RJ, Chamswarng C, Tang WH (1990) Influence of wheat chaff and tillage on *Pythium* populations in soil and *Pythium* damage to wheat. Soil Biol Biochem 22:939–947
Cook RJ, Schillinger WF, Christensen NW (2002) Rhizoctonia root rot and take-all of wheat in diverse direct-seed spring cropping systems. Can J Pl Pathol 24:349–358
Costa Neto JP (1943) Fungos do Rio Grande do Sul observados nos anos 1940–41. Bull Secretaria do Estado dos negócios da Agricultura, Industria e Comércios 99:1–11
Cox J (1965) Continuous wheat growing and the decline of take-all. Rep Rothomstead Exp Sta 1964:133–134
Daval S, Lebreton L, Gazengel K, Guilerm-Ercklboudt AY, Sarniguet A (2010) Genetic evidence for *Gaeummanomyces graminis* var. *tritici* into two major groups. Plant Pathol 59:165–178
Deacon JW (1974) Interactions between varieties of *Gaeumannomyces graminis* and *Phialophora radicicola* on roots, stem and rhizomes of the Gramineae. Plant Pathol 22:85–92
Dickson JG (1956) Diseases of field crops. McGraw-Hill, New York, p 517
Diehl JA (1979) Common root rot of wheat in Brazil. Plant Dis Reptr 63:1020–1022
Diehl JA (1982) Reação de cultivares de trigoà podridão comum de raízes. Pesq Agropec Bras 17(2):1733–1735
Dielh JA, Oliveira MAR, Igarashi S, Reis EM, Mehta YR, Gomes EP (1983) Levantamento de ocorrência de doenças radiculares do trigo no Paraná. Fitopatologia Brasileira 9:179–188
Dubin HJ (1985) Reflections on foot rots of wheat in warmer non-traditional wheat growing climates. In: Wheats for more tropical environments. Proceedings of the international symposium, CIMMYT, Mexico, pp. 182–185
Duckez LJ (1989) Number and viability of *Cochliobolus sativus* in soil profiles in summer fallow fields in Sasketchewan. Can J Plant Pathol 3:12–14
Elnur E, Chester CG (1967) A note on two isolates of *Rhizoctonia solani* Khun from wheat. Plant Pathol 16:104–107
Fish S (1970) The history of plant pathology in Australia. Ann Rev Phytopathol 8:13–36
Frank JA (1985) Influence of root rot on winter survival and yield of winter barley and winter wheat. Phytopathology 75:1039–1041
Garrett SD (1939) Soil conditions and the take-all disease of wheat. IV. Factors limiting infection by ascospores of *Ophiobolus graminis*. Ann Appl Biol 26:47–55
Garrett SD, Mann HH (1948) Soil conditions and the take-all disease of wheat. X. Control of the disease under continuous cultivation of a spring-sown cereal. Ann Appl Biol 35:435–442
Gasperi AJ (1961) Moléstias do trigo no Rio Grande do Sul. Bull Tec. Secretaria da Agricultura, s. d. 36 pp
Gill JS, Sivasithamparam K, Smettem KRJ (2001) Effect of soil moisture at different temperatures on Rhizoctonia root rot of wheat seedlings. Plant Soil 231:91–96

Gutteridge RJ, Zhang JP, Jenkyn JF, Bateman GL (2005) Survival and multiplication of *Gaeumannomyces graminis* var. *tritici* (the wheat take-all fungus) and related fungi on different wild and cultivated grasses. Appl Soil Ecol 29:143–154

Gyawali S, Neate SM, Adhikari TB, Puri KD, Burlakoti RR, Zhong S (2012) Genetic structure of *Cochliobolus sativus* populations sampled from root and leaves of barley and wheat in North Dakota. J Phytopathol 160:637–646. doi:10.1111/j.1439-0434.2012.01956

Henry AW (1931) Occurrence and sporulation of *Helminthosporium sativum* P.K. & B. in the soil. Can J Res 5:407–413

Higginbotham RW, Paulitz TC, Kidwell KK (2004) Virulence of *Pythium* species isolated from wheat fields in Eastern Washington. Plant Dis 88:1021–1026

Hobbs PR, Mann CE, Butler L (1988) A perspective on research needs for the rice-wheat rotation. In: Klatt AR (ed) Wheat production constrains in tropical environments. CIMMYT, Mexico, pp 197–211

Horneby D (1969) Methods of investigating populations of the take-all fungus (*Ophiobolus graminis*) in soil. Ann Appl Biol 35:435–442

Hosford RM, Solangi GRM, Kiesling RL (1975) Inheritance in *Cochliobolus sativus*. Phytopathology 65:699

Huber DM (1972) Spring versus fall nitrogen fertilization and take-all of spring wheat. Phytopathology 62:434–436

Huber DM, Painter CG, McKay HC, Peterson DL (1968) Effect of nitrogen fertilization on take-all of winter wheat. Phytopathology 58:1470–1472

Huberli D, Connor M, Miyan S, MacLeod W (2012) Integrated disease management options to control Rhizoctonia bare-patch in wheat. In:7th Australian soil-borne diseases symposium, South Perth

Igarashi S, Mehta YR, Nazareno NRX (1983) Occorrênciade *Sclerotium rolfsii* na cultura de trigo (*Triticum aestivum*) no estado do Paraná, Brasil. Fitopatologia Brasileira 8:513–515

Inglis DA, Cook RJ (1986) Persistence of chlamydospores of *Fusarium culmorum* in wheat field soils of eastern Washington. Phytopathology 76:1205–1208

Jenkins WA (1948) A root disease complex of small grains in Virginia. Phytopathology 38:519–527

Joshi LM, Goel LB, Renfro BL (1969) Multiplication of inoculum of *Helminthosporium turcicum* on sorghum seeds. Indian Phytopathol 22:146–148

Juhnke ME, Mathre DE, Sands DC (1984) A selective midium for *Gaeumannomyces graminis* var. *tritici*. Plant Dis 68:233–236

Knight NL, Platz GJ, Lehmensiek A, Sutherland MW (2010) An investigation of genetic variation among Australian isolates of *Bipolaris sorokiniana* from different cereal tissues and comparison of their abilities to cause spot blotch on barley. Australian Pl Pathol 39:207–216

Kumar J, Schafer P, Huckelhoven R, Lagen G, Baltruschat H, Stein E, Nagarajan S, Kogel KH (2002) *Bipolaris sorokiniana*, a cereal pathogen of global concern: cytological and molecular approaches towards better control. Mol Pl Pathol 3(4):185–195

Lawrence L, Harvey P (2006) Rooting out *Pythium* and its allies. Farming Ahead 177:42–44

Liddell CM (1985) The comparative pathogenicity of *Fusarium graminearum* group 1, *Fusarium culmorum* and *Fusarium crookwellense* as crown, foot and root rot pathogens of wheat. Aust Pl Pathol 14:29–32

Lindingham RJ, Atkinson TG, Horricks JS, Mills JT, Piening LJ, Tinline RD (1973) Wheat losses due to common root rot in the Prairie Provinces of Canadá, 1969–71. Can Plant Dis Surv 53:113–122

Lutrell ES (1964) Taxanomic criteria in *Helminthosporium*. Mycologia 56:119–132

Luzzardi GC, Pierobom RC (1970) Moléstias do trigo na região sul do Brasil. Circular No. 42, EMBRAPA, Rio Grande do Sul, Brasil, 24pp

Luzzardi GC, Reis EM, Pierobom CR (1976) Epifitia de *Colletotrichum graminicola* (Cesati) G. W. Wilson, nos trigais no sul do Brasil, e, 1975. Trabalho apresentado em VII RENAPET, Ponta Grossa, Brasil

MacNish GC, Neate SM (1996) Rhizoctonia bare patch of cereals: an Australian perspective. Plant Dis 80:965–971

Mai WF, Mullin PG (1996) Plant parasitic nematodes: a pictorial key to Genera. Cornell University Press, Ithaca, NY

Marvodi OV, Walter N, Elateek S, Tayler CG, Ocubara PA (2012) Suppression of Rhizoctonia and Pythium root rot of wheat by new strains of *Pseudomonas*. DOI: 10.1016/j.biocontrol

Mazzola M, Wong OT, Cook RJ (1996) Virulence of *Rhizoctonia oryzae* and *R. solani* AG-8 on wheat and detection in *R. oryzae* in plant tissue by PCR. Phytopathology 86:354–360

Mehta YR (1978) Doenças do trigo e seu controle. Editora CERES, São Paulo, p 190

Mehta YR (1981) Conidial production, sporulation period and extension of lesion of *Helminthosporium sativum* on flag leaves of wheat. Pesq Agrop Bras 16(1):77–99

Mehta YR (1993) Manejo integrado de enfermedadesdel trigo. Imprenta Landivar, Santa Cruz de la Sierra, Bolivia, 314pp

Mehta YR, Gaudêncio C (1991) Effects of tillage practices and crop rotation on the epidemiology of some major wheat diseases. In: Saunders C (ed) Proceedings of the international conference on wheat for non-traditional warmer areas. CIMMYT, Mexico, pp 266–283

Mehta YR, Igarashi S (1985a) Chemical control measures for major diseases of wheat with special reference to spot blotch. pp 196–200. Proc. Inter. Sym. CIMMYT, México, D.F., 364 pp.

Mehta YR, Igarashi S (1985b) Fungos associados nas sementes de trigo *Triticum aestivum* L. e seu efeito na infecção do sistema radicular das plantas. Revista Brasileira de Sementes 7:133–159

Merriman PR, Price RD, Baker KF (1974a) The effect of inoculation of seed with antagonists of *Rhizoctonia solani* on growth of wheat. Aust J Agric Res 25:213–218

Merriman PR, Price RD, Kolimorgan JF, Piggott T, Ridge EH (1974b) Effect of seed inoculation with *Bacillus subtilis* and *Steptomycesgriseus* on the growth of cereals and carrots. Aust J Agric Res 25:219–226

Milus EA, Cartwright RD, Rothrock CS, Anders M, Slaton N (2009) Impact of cropping sequences and alternative hosts on take-all management of winter wheat in Arkansas. Plant Health Progress DOI: 10.1094/PHP 2009-0512-02-RS

Monds RD, Cromey MG, Lauren DR, di Menna M, Marshall J (2005) *Fusarium graminearum, F. cortaderiae and F. pseudograminearum in New Zeland:* Molecular phylogenetic analysis, mycotoxin chemotypes and co-existance of species. Mol Res 109:410–420

Moore KJ, Cook RJ (1984) Increase in take-all of wheat with direct drilling in the Pacific Nothwest. Phytopathology 74:1044–1049

Mordue JEM (1974) *Thanatephorouscucumeris*. C.M.I. Descriptions of pathogenic fungi and bacteria. No. 406. Comm. Mycol. Inst. England.

Nash SM, Christou T, Snyder WC (1961) Existence of *Fusarium solani* f. *phaseoli*as clamydospores in soil. Phytopathology 51:308–312

Nath R, Neergaard P, Mathur SB (1970) Identification of *Fusarium* species on seeds as they occur in blotter test. Proc Int Seed Test Assoc 35:121–144

Nicol JM, Davies KA, Hancock TW, Fisher JM (1999) Yield loss caused by *Pratylenchus thornei* on wheat in South Australia. J Nematology 31:367–376

Nillson HE (1973) Varietal differences in resistance to take-all disease of winter wheat. Swedish J Agric Res 3:89–93

Pankhurst CE, McDonald HJ, Hawke BG (1995) Influence of tillage and crop rotation on the epidemiology of *Pythium* infections of wheat in a red-brown earth of South Australia. Soil Biol Biochem 27:1065–1073

Parmeter JR (1970) *Rhizoctonia solani*, biology and pathology. University of California Press, Berkeley, 255pp

Paulitz TC (2010) Pythium root rot. In: Bockus WW et al (eds) Compendium of wheat diseases and pests, 3rd edn. American Phytopathological Society, St. Paul, pp 45–47

Paulitz TC, Adams K (2003) Composition and distribution of *Pythium* communities in wheat fields in eastern Washington State. Phytopathology 93:867–873

Paulitz TC, Adams K, Mazzola M (2003a) *Pythium abappressorium*—a new species from eastern Washington. Mycologia 95:80–86

Paulitz TC, Smith JD, Kidwell KK (2003b) Virulence of *Rhizoctonia oryzae* on wheat and barley cultivars from Pacific Northwest. Plant Dis 87:51–55

Paulitz TC, Zang H, Cook RJ (2003c) Spatial distribution of *Rhizoctonia oryzae* and rhizoctonia root rot in direct-seeded cereals. Can J Pathol 25:295–303

Paultiz TC, Shroeder KL (2005) A new method for the quantification of *Rhizoctonia solani* and *R. oryzae* from soil. Plant Dis 89:767–772

Pinheiros VR, Seixas CDS, Godoy CV, Soares R, Oliveira MCN, Almeida AMR (2010) Development of *Sclerotium rolfsii* sclerotia on soybean, corn and wheat straw, under different soil temperatures and moisture contents. Pesq Agrop Bras 45(3):1–4

Pitt D (1964a) Studies on sharp eyespot disease of cereals. I. Ann Appl Biol 54:77–89

Pitt D (1964b) Studies on sharp eyespot disease of cereals. II. Ann Appl Biol 54:231–240

Politis DJ (1975) The identity and perfect state of *Colletotrichum graminicola*. Mycologia 67:56–62

Politis DJ, Wheeler H (1972) The perfect stage of *Colletotrichum graminicola*. Plant Dis 56:1026–1027

Prew RD, Mcintosh HM (1975) Effect of benomyl and other fungicides on take-all, eyespot and sharp eyespot diseases of winter wheat. Plant Pathol 24:67–71

Pumphrey FV, Wilkins DE, Hane DC, Smiley RW (1987) Influence of tillage and nitrogen fertilizer on Rhizoctonia root rot (bare patch) of winter wheat. Plant Dis 71:125–127

Punja ZK (1985) The biology, ecology and control of *Sclerotium rolfsii*. Ann Rev Phytopathol pp 23:97–127

Punja ZK (1988) *Sclerotium rolfsii*: Potential impact on wheat production and possible means of control. In: Klatt AR (ed) Wheat production constrains in tropical environments. CIMMYT, México, pp 153–174

Punja ZK, Huang JS, Jenkins FS (1985) Relationship of mycelial growth and production of oxalic acid and cell wall degrading enzymes to virulence of *Sclerotium rolfsii*. Can J Plant Pathol 7:109–117

Quisenberry KS, Rietz LP (eds) (1967) Wheat and wheat improvement. American Society of Agronomy, Madison, 560pp

Raaijmakers JM, Paulitz TC (2009) Therhizosphere: a playground and battlefield for soilborne pathogens and beneficial microorganism. Plant Soil 32:341–361

Reeves TG, Ellington A, Brooke HD (1984) Effect of lupin-wheat rotations on soil fertility, crop disease and crop yields. Aus J Exp Agric Husb 24:595–600

Reis EM (1986a) Densidade de inóculo de *Helminthosporium sativum* no solo, indicativo da interferência entre parcelas experimentais. Fitopatologia Brasileira 11:89–94

Reis EM (1986) Doenças do trigo II. Mal-do-pé. Apassul, Passo Fundo, Brasil, 29 pp

Reis EM, Abrão (1983) Effect of tillage and wheat residue management on the vertical distribution and inoculum density of *Cochliobolus sativus* in soil. Plant Dis 67:1088–1089

Reis EM, Baier AC (1983) Reação de cereais de inverno à podridão comum de raízes. Fitopatologia Brasileira 8:277–281

Roget DK, Venn NR, Rovira AD (1987) Reduction of Rhizoctonia root rot of direct-drilled wheat by short term chemical fallow. Aust J Exp Agric 27:425–430

Rothrock CS, Cunfer BM (1991) Influence of small grain rotations on take-all in a subsequent wheat crop. Plant Dis 75:1050–1052

Rovira AD (1986) Influence of crop rotation and tillage on Rhizoctonia bare patch of wheat. Phytopathology 76:669–673

Russel RS, Igue K, Mehta YR (eds) (1981) Soil root system in Brazilian Agriculture. IAPAR, Londrina, Paraná, Brazil, 372pp

Sallans BJ, Tinline RD (1965) Resistance in wheat to *Cochliobolus sativus*, a cause of common root rot. Can J Plant Sci 45:343–351

Sanford GB (1956) Factors influencing formation of sclerotia by *Rhizoctonia solani*. Phytopathology 46:281–284

Schoeder KL, Okubara PA, Tambong JT, Lévesque CA, Paulitz TC (2006) Identification and quantification of pathogenic *Pythium* spp. from soils in eastern Washington using real-time polymerase chain reaction. Phytopathology 96:637–647

Schroeder KL, Paulitz TC (2006) Root diseases of wheat and barley during the transition from conventional tillage to direct seeding. Plant Dis 90:1247–1253

Sequeira L (1963) Effect of urea application on survival of *Fusariumoxy sporum* f. *cubense* in soil. Phytopathology 53:322–336
Shipton PJ (1972) Take-all in spring sown cereals under continuous cultivation. Disease progress and decline in relation to crop succession and nitrogen. Ann Appl Biol 71:33–46
Shipton PJ, Cook JR, Sitton JW (1973) Occurrence and transfer of a biological factor in soil that suppresses take-all of wheat in eastern Washington. Phytopathology 63:511–517
Siegle H (1961) Ubermischinfektionenmit*Ophiobolus graminis* und *Didymellaexitales*. Phytopath Z 42:305–348
Singh K, Frisvad JC, Thrane UIF, Mathur SB (1991) An illustrated manual on identification of some seed-borne Aspergilli, Fusaria, Penicillia and their mycotoxins. Danish Govt. Inst. Seed Path. For Develop. Countries, Copenhagen, Denmark, 133 pp
Singleton LL (1988) Wheat root rot in tropical environments: potential impact and control. In: Wheat production constrains in tropical environments. Proceedings of international conference 1987. CIMMYT, Mexico, pp. 251–262
Smiley RW (2009) Water and temperature parameters associated with winter wheat diseases caused by soil-borne pathogens. Plant Dis 93:73–80
Smiley RW, Machado S (2009) *Pratylenchus neglectus* reduces yield of winter wheat in dryland cropping systems. Plant Dis 93:263–271
Smiley RW, Uddin W (1993) Influence of soil temperature on Rhizoctonia root rot (*R. solani* AG-8 and *R. oryzae*) of winter wheat. Phytopathology 83:777–785
Smiley RW, Cook RJ, Papendick RI (1970) Unhydrous ammonia as a fungicide against *Fusarium* and fungicidal activity in the ammonia and ammonia-potassium azide solutions. Phytopathology 60:1227–1232
Smiley RW, Cook RJ, Papendick RI (1972) Fusarium root rot of wheat and peas as influenced by soil applications of anhydrous ammonia and ammonia-potassium azide solutions. Phytopathology 62:86–91
Smiley RW, Gourlie JA, Easley SA, Patterson LM (2005) Pathogenicity of fungi associated with the wheat crown rot complex in Origon and Washington. Plant Dis 89:949–957
Specht LP, Rush CM (1988) Fungi associated with root rot and foot rot of winter wheat and populations of *Cochliobolus sativus* in the Texas Panhandle. Plant Dis 72:959–963
Stack RW (1977) A simple selective medium for isolation of *Cochliobolus sativus* from diseased cereal crowns and roots. Plant Dis Reptr 61:521–522
Stack RW (1994) Susceptibility of hard red spring wheats to common root rot. Crop Sci 34:276–278
Sward RM, Kollmorgen JF (1986) The separate and combined effects of barley yellow dwarf virus and take-all fungus (*Gaeumannomyces graminis* var. *tritici*) on the growth and yield of wheat. Aust J Agri Res 37(1):11–22
Tinline RD (1977) Multiple infections of sub-crown internodes of wheat (*Triticum aestivum*) by common root rot fungi. Can J Bot 55:30–34
Toussoun TA, Nash SM, Snyder WC (1960) The effect of nitrogen sources and glucose on the pathogenesis of *Fusarium solani* f. sp. *phaseoli*. Phytopathology 50:137–140
Tyner LE (1956) The incidence of root disease fungi in wheat fields of central and northwestern Alberta. Plant Dis Reptr 40:358–360
Vanterpool TC (1938) Some species of *Pythium* parasitic on wheat in Canada and England. Ann Appl Biol 25:528–543
Verma PR, Morral RAA, Randell RL (1975) The epidemiology of common root rot in Manitoba wheat. III. Development of lesions on sub-crown internodes and the effect of added phosphate. Can J Bot 53:601–606
Walker J (1973) *Gaeumannomyces graminis* var. *tritici*. In: C.M.I. Descriptions of Pathogenic Fungi and Bacteria no. 383, Kew, Surrey
Walker J (1975) Take-all disease of Gramineae: a review of recent work. Rev Plant Pathol 54:113–144
Warcup JR (1957) *Gaeumannomyces graminis* var. *tritici*. C.M.I. Descriptions of pathogenic fungi and bacteria. No. 33, Comm. Mycol. Inst. Kew Surrey

Warren HL, Kommendahl T (1973) Fertilization and wheat refuse effects on *Fusarium* species associated with roots in Minnesota. Phytopathology 63:103–108

Wehrle VW, Ogilve L (1956) Spread of take-all from infected wheat plant. Plant Pathol 5:106–107

Weinke KE (1962) The influence of nitrogen on the root disease of bean caused by *Fusarium solani* f. sp. *phaesioli*. Phytopathology 52:757 (abstr.)

Weller DM, Cook RL (1986a) Increased growth of wheat by seed treatment with fluorescent pseudomonads, and implications of *Pythium* control. Can J Plant Pathol 8:328–334

Weller DM, Cook RJ (1986b) Rhizoctonia root rot of small grains favored by reduced tillage in the Pacific Northwest. Plant Dis 70:70–73

Weller DM, Raaijmakers JM, McSpadden Gardner BB, Thomashow LS (2002) Microbial populations responsible for specific soil suppressiveness to plant pathogens. Annu Rev Phytopathol 40:309–348

Weste G (1972) The process of root infection by *Ophiobolus graminis*. Trans Br Mycol Soc 59:133–147

Widermuth GB, Thomas GA, Radford BJ, NcNamara KA (1997) Crown rot and common root rot in wheat grown under different tillage and stubble treatments in southern Queensland. Aust Soil Tillage Res 44:211–223

Wildermuth GB (1986) Geographic distribution of common root rot and *Bipolaris sorokiniana* in Queensland wheat soils. Aust J Exp Agric 26:601–606

Wood LS (1962) Relation of variation in *Helminthosporium sativum* to seedling blight in small grains. Phytopathology 52:493–497

Yarham DJ, Hirst JM (1975) Diseases in reduced cultivation and direct drilling systems. EPPO Bull 5:287–296

Yun-Nung T, Mei-Ju L, Wen-Hsiung K (2011) A simple method for production of uniform inoculum of *Rhizoctonia solani* with strong pathogenicity. doi: 10.1016/j.bcab.2011.08.006

Zawart R, Thomson J, Milgatte A, Bansal U, Williamson P, Raman H, Bariana H (2010) QTL mapping of multiple foliar disease and root-lesion nematode resistances in wheat. Mol Breeding 26:107–124

Zillinsky FJ (1983) Common diseases of small grains of cereals—A guide for identification. CIMMYT, Mexico, 141