

## Root rot of wheat

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### SUMMARY

The expansion of cereal to marginal and stressed environments in order to satisfy the increasing food demand of population is common to many countries. Dryland root rot disease of wheat is an other challenge that faces the agronomist in general to improve productivity in these environments. This paper review the major research works conducted in many parts of the world on root rot disease of cereals. Major emphasis is made-whenver it is possible-on most of the research accomplished on root rot on Morocco since 1984. This review paper includes most references on root rot and might be useful for any person involved in root rot research.

**Key words:** Root rot, cereals, Morocco

### RESUME

**Titre:** pourritures racinaires du blé

L'extension de la culture des céréales aux zones marginales et stressées afin de combler le déficit croissant en produits céréaliers est un phénomène très commun à plusieurs pays. Les maladies causant les pourritures racinaires très fréquentes dans ce type d'environnements, reste un déficit crucial à soulever par les chercheurs pour augmenter la production céréalière dans ces zones. Cet article est une revue bibliographique des principaux travaux de recherches menés sur les pourritures racinaires à travers le monde. Les études menées par les chercheurs au Maroc sur cet aspect sont aussi incluses dans ce manuscrit. Nous estimons que cet article englobe les principaux résultats et références sur les pourritures racinaires des blés utiles à tout chercheur qui veut entamer des recherches liées à ce sujet.

**Mots Clés:** Pourritures racinaires, céréales, Maroc

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## ملخص

### العنوان: التعفنات الجدرية في القمح

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بدأت زراعة الحبوب، التي تعتبر من أهم المنتوجات الفلاحية عبر العالم تغطي مساحات شاسعة من المناطق الجافة وشبه الجافة وهذا راجع للنقص المتزايد في منتوجات الحبوب. ويعتبر التعفن الجدرية من بين الأمراض التي تحد من منتوجية القمح في هذه المناطق. يلخص هذا التقرير أهم الأبحاث التي أجريت عبر العالم حول التعفنات الجدرية للحبوب. كما يحتوي على أهم النتائج والأبحاث التي أجريت في المغرب على هذه الأمراض. ويشتمل على أهم المراجع والنتائج التي يحتاجها كل باحث مهتم بالبحوث في مجال أمراض التعفنات الجدرية.

**الكلمات المفتاحية:** التعفنات الجدرية، الحبوب، المغرب

## INTRODUCTION

Common root rot, foot rot, crown rot, pink rot, culm rot, and stalk rot are descriptive terms used to designate a group of wheat diseases caused by one or more organisms, alone or in association (Burgess *et al.* 1975). Common root rot has been reported from most cereal growing areas of the world. However, the occurrence and frequency of causal agents vary regionally and from year to year (Fernandez *et al.* 1985; Lyamani 1988).

*Cochliobolus sativus* and several species of *Fusarium* including *F. graminearum* Schwabe, *F. culmorum* (W.G. Smith) Sacc., and *F. avenaceum* (Fr.) Sacc. are considered the primary causal agents of root rot of wheat (Burgess *et al.* 1975; Specht and Rush 1988; Lyamani 1988).

Root rot is a persistent and inconspicuous disease that reduces seedling vigor and impairs functioning of roots and crowns resulting in loss of stands, reduced yield and lower grain quality (Fenster *et al.* 1972; Mergoum 1991; Mergoum *et al.* 1994). The damage from root rot pathogens varies from year to year and from field to field depending upon the amount of inoculum present as well as soil and climatic factors (Statler and Darlington 1972; Lyamani 1988; Tinline and

Ledingham 1979). In addition to the environmental factors, certain cultural practices may play an important role in the expression of this disease (Tinline 1986; Broschious and Frank 1986; Wildermuth and McNamara 1991; Mergoum 1991; Houmaïri 1993).

This paper reviews works on root rot disease of wheat conducted in many parts of the world with emphasis on those realized in Morocco. It includes sections on economic importance, causal agents, source of inoculum, virulence and competition, control, inoculation technics and selection for root rot tolerance or resistance. For the purpose of consistency, and to avoid confusion in this report, all symptoms previously indicated will be referred to as "root rot".

## ECONOMIC IMPORTANCE

Root rot was described as an "insidious, presistent and inconspicuous disease" that reduces yields to some degree each year (Fenster *et al.* 1972). In the USA, root rot disease is considered the third greatest cause of yield reduction of wheat after the rusts and smuts (Merrill 1977). In the northwestern region of the USA, yield reduction of wheat by *F. culmorum* was estimated to be at least 10% (Cook 1968b) and reached 50% (Cook 1968a) in some infected fields. Under favorable conditions, yield reductions of up to 50 and 70% in wheat and barley, respectively, were attributed to common root rot in some areas of Nebraska (U.S. Department of Agriculture 1979) and North Dakota (Stack 1982). Overall stand reductions caused by *Cochliobolus sativus* ranged from 11 to 29% and from 27 to 62% in wheat and barley, respectively (Frank 1985). Similar stand reductions were reported in areas of Colorado and Wyoming (Hill and Fernandez 1983), Montana (Grey *et al.* 1991), and Tennessee (Reed 1952). Average yield losses due to root rot in North America were estimated to 3 to 4% (Weise 1977).

Loss surveys in the Canadian regions have shown that root rot is a widespread and important disease of wheat and barley (Ledingham *et al.* 1973; Piening *et al.* 1976; Verma *et al.* 1976a; Tinline and Ledingham 1979). These studies showed that average yield losses in wheat ranged from 5 to 12% over the period of 1939 to 1973. In barley, Canadian researchers estimated an average 10,3% yield loss caused by root rot from 1970 to 1972 (Piening *et al.* 1976). Grain loss of "Manitou" wheat ranged from 22.5 to 34.9% depending on disease severity and cropping systems (Verma *et al.* 1976b). In the Canadian prairie provinces, estimated yield loss caused by root rot of wheat was 5.7% from 1969 to 1971 (Ledingham *et al.* 1973). Yields of barley were reduced by 16% and 40% on fallow land and stubble, respectively (Piening *et al.* 1983).

In Australia, reduction of 26.6% in average yield was reported (Purss 1966). Up to 20% dead heads and 100% infection by *F. graminearum* were recorded (Wearing and Burgess 1977).

In brazil, reports indicate losses caused primarily by *C. sativus* (Luz 1984) of 14 and 19%, respectively, in the wheat cultivars "Nobre" and "CNT 10".

In Morocco, field studies conducted in 1985-86 and 1986-87 in the West Central regions showed average yield losses of 4 and 6%, respectively (Lyamani 1988). Under artificial inoculation by root rot pathogens *Helminthosporium sativum* and *Fusarium culmorum*, yield losses as high as 60% were recorded on durum cultivar Marzak in 1989-90 cropping season (Mergoum 1991).

Yield losses due to common root rot of wheat and barley resulted from a reduction in emergence (Verma *et al.* 1976 a; Grey *et al.* 1991; Mergoum 1991; Mergoum *et al.* 1988; 1994), tiller number (Verma *et al.* 1976a; Grey *et al.* 1991; Mergoum, 1991; Mergoum *et al.* 1994), number of spikes per plant (Piening 1973; Ledingham *et al.* 1973; Piening *et al.* 1976), spike size (Verma *et al.* 1976a; Weise 1977), number of kernels per spike (Sims *et al.* 1961; Lidingham *et al.* 1973; Verma *et al.* 1976a; Weise 1977; Mergoum 1991; Mergoum *et al.* 1994), kernel weight (Sims *et al.* 1961; Lidingham *et al.* 1973; Verma *et al.* 1976; Weise 1977; Mergoum 1991; Mergoum *et al.* 1988), and kernel size (Sims *et al.* 1961). However, the number of tillers per plant and number of kernels per spike appear to be the yield components most affected by root rot in wheat (Verma *et al.* 1973). Wheat kernel protein levels seem to be only slightly affected by root rot (Ismail and Michalik 1980). Plants can recover from initial stunting due to early infection by root rot pathogens if favorable environmental conditions occur during the late growth stages (Sallans 1959). Recovery from *F. culmorum* infection was less marked than from *C. sativus* infections and is variable among wheat cultivars. Simultaneous infection of environmental conditions occur during the late growth stages (Sallans 1959). Recovery from *F. culmorum* infection was less marked than from *C. sativus* infections and is variable among wheat cultivars. Simultaneous infection of wheat seedling with both *F. culmorum* and *C. sativus* caused more initial stunting followed by a greater recovery than with either pathogen alone (Sallans 1965). When severe damage occurs in the crowns, however, the plants will not recover even though under optimum environmental conditions. Recovery depends on the amount of damage caused to crown tissue (Fenster *et al.* 1972).

## CAUSAL ORGANISMS

The root rot disease complex of cereals can be caused by one or several organisms, alone or in combination (Statler and Drington 1972). *Cochliobolus sativus* (Ito & Kuribayashi) Drechsler ex Dstur, (conidial stage = *Helminthosporium sativum* Pammel, King and Bakke, Syn. *Bipolaris sorokinianum* (Sacc. in Sorokin Shoemaker), has long been known as an important component of the root rot complex ( Verma *et al.* 1974; 1976a; Frank 1985; Specht and Rush 1988).

In addition to *C. sativus*, several species of *Fusarium* including *F. culmorum* (W. G. Smith) Sacc., *F. graminearum* Schwabe; and *F. avenaceum* (Fr.) Sacc. which are considered the most virulent species, can cause root rot disease alone (Cook 1968a, 1980; 1981; Couture 1982; 1983); or in association with *C.*

*sativus* (Watking and Kerr, 1980; Scardaci, and Webster 1982; Diehl *et al.* 1982; Hill and Fernandez 1983; Saari and Wilcoxson, 1974). In Morocco, Although, most studies demonstrated that root rot of wheat is caused mainly by *Helminthosporium sativum* and *Fusarium culmorum* (Baye 1988; Lyamani 1988; Mergoum 1991; Houmairi 1993), many prevalent fungi including *F. equiseti*, *F. oxysporum*, *F. solani* and *F. graminearum* were associated with root rot (Lyamani 1988; Houmairi 1993).

## SYMPTOMS

Root rot symptoms on cereals may consist of discoloration, necrosis or rotting of roots, subcrown internode, crown, and/or the stem base (Sallans 1965; Watkins and Kerr 1980; stack 1982). Under severe root rot conditions, symptoms such as stunting, late death of tillers, and premature ripening and bleaching of the spikes commonly known as "white heads" or "dead heads" may occur (Walkins and Kerr 1980; Scardaci and Webster 1982; Stack 1982; Lyamani 1988; Mergoum 1991; Mergoum *et al.* 1994). Early infection may result in preemergence and postemergence seedling blight (Butler 1961; Watkins and Kerr 1980).

## DISTRIBUTION

Root rot surveys have been extensively conducted in the USA (Fenster *et al.* 1972; Frank 1985; Hill and Fernandez 1983; Grey and Mathre 1984), Canada (Ledingham 1961; 1973; Verma *et al.* 1974; 1976b; Duczeck *et al.* 1985), Australia (McNight and Hart 1966; Wearing and Burgess 1977), Brazil (Diehl *et al.* 1982; Luz 1984), India (Neima and Joshi 1973), England (Snyder and Nash 1968), Italy (Piglionica *et al.* 1975), Hungary (Mesterhazy 1974), France (Cassini 1967), Morocco (Baye 1984; Lyamani 1988), and Poland (Manka *et al.* 1985).

In Colorado and Wyoming (Hill and Fernandez 1983) and in Texas, USA (Specht and Rush 1988), root rot of winter wheat is caused primarily by the association of *C. sativus* and *Fusarium acuminatum*. *F. acuminatum* is more prevalent in the spring and summer (Hill and Fernandez 1983), while *C. sativus* usually infects winter wheat during the fall or early spring.

In Morocco, several species of *Fusarium* are associated with root rot symptoms in addition to *C. sativus* (Lyamani 1988). The most prevalent *Fusarium* species are *F. equiseti*, *F. culmorum*, *F. oxysporum*, and *F. solani*. *C. sativus* and *F. culmorum* are considered the major pathogens causing root rot of spring wheats and barley in Morocco (Baye 1984; Lyamani 1988).

## SOURCE OF INOCULUM

The relative importance of seed and soil as sources of inoculum for root rot organisms has been intensively investigated in the cereal growing areas throughout the world. Seed loss surveys conducted in Australia (Chambers 1962), Canada (Ledingham 1961; 1970), England (Hewett 1967), Morocco (Lyamani 1975; 1988), Finland (Ylimaki 1970; Uoti and Ylimaki 1974) and France (Cassini 1970) showed that seed infection by root rot pathogens was generally low.

The primary inoculum of *Fusarium* spp. is soil born chlamydospores in plant debris which is localized mainly in the upper 10 cm of the soil. *F. culmorum* exists in the soil as single, double and clumps of chlamydospores free or embedded in organic matter (Cook 1980). *F. graminearum* chlamydospores are more likely to lose viability under high soil temperatures and rapid desiccation than are those of *F. culmorum*. Therefore, mycelium in crop residue appears to be the main form of viable inoculum of *F. graminearum* (Wearing and Burgess 1977; Sitton and Cook 1981).

Conidia and/or mycelium in plant debris in or on the soil are the primary survival structures of *C. sativus* (Ledingham *et al.* 1960; Chinn 1976a; 1976b; Weise 1977). *C. sativus* conidia can remain viable in the soil for many years (Ledingham 1970) and therefore, soil born inoculum has a major role in infection (Verma *et al.* 1976b).

## SURVIVAL

The thick-walled chlamydospores produced by *F. culmorum* in the soil can survive there for years (Cassini 1970; Sitton and Cook 1981; Williams and Schimmitthener 1962; Inglis and Cook 1986). These spores tend to remain inactive unless hosts are available. This pathogen can also survive as mycelium on plant debris. Both *F. culmorum* and *F. nivale* can infect hosts other than cereals and this may contribute to their prolonged survival in certain fields (Jamalainen 1959).

*F. graminearum* can also produce chlamydospores under appropriate temperature conditions. Chlamydospores were developed in non-autoclaved soil stored at 21 to 30°C but not at 5 to 11°C (Nyval 1970; Sitton and Cook 1981). However, this fungus survives primarily as mycelium in plant debris (Wearing and Burgess 1977). *F. culmorum* and *F. graminearum* are not good competitors against antagonistic soil fungi. Both colonize plant tissue mainly through the parasitic process (Cook and Bruehl 1968; Cook 1970; Warren and Kommedahl 1973; Wearing and Burgess 1977).

*C. sativus* survives mainly as conidia or as a mycelium in host plant debris. Conidia can survive several years in the soil (Ledingham *et al.* 1960; Ledingham

1970; Weise 1977). *C. sativus* may also be carried through storage on seeds especially under wet conditions during harvest (Williams *et al.* 1980).

## VIRULENCE AND COMPETITION

Variability in virulence among isolates of *C. sativus* and *Fusarium* species is well known and was described as a type of physiologic specialization which is common among many fungi. Most isolates of *C. sativus* tested for their pathogenicity to cereal seedlings showed moderate to weak virulence. No differences among these isolates were observed when tested on barley (Kidambi *et al.* 1985).

Studies made on the pathogenicity of *Fusarium* pathogens showed considerable variability in virulence among and within pathogenic *Fusarium* species. *F. culmorum* and *F. graminearum* are strongly virulent and *F. avenaceum* is a weakly virulent, while the remaining *Fusarium* species are non-pathogenic to weakly virulent (Osmald 1949; chambers 1972; Colhoun *et al.* 1968; Uoti 1976a; Mesterhasy 1978; Lyamani 1988). Virulence of *F. culmorum* isolates to both seedling and adult plant under field conditions was greater than that found under greenhouse conditions.

Competition between *Fusarium* species and *C. sativus* in the wheat subcrown internodes has been reported (Tinline 1977). *C. sativus* infection of already colonized subcrowns by *F. culmorum* and *F. acuminatum* is inhibited. *Fusarium*, however, is able to infect and colonize internodes already infected by *C. sativus* (Tinline 1977). Wheat seedlings inoculated with both *F. culmorum* and *F. acuminatum* were shown to enhance the effects of *C. sativus* (Fernandez *et al.* 1958). In some cases however, infection with several fungi was less damaging than single infections.

## EPIDEMIOLOGY

### Climatic factors

There is a general agreement among workers that soil moisture and temperature are critical factors in root rot disease development.

In the Pacific Northwest region of the USA, root and foot rot of wheat caused by *F. culmorum* is most severe in the 200 to 350 mm annual precipitation, stubble mulched, wheat-fallow areas (Cook 1968 b). Other investigations (Cook and Christen 1967) in the same regions showed that *F. graminearum* and *F. culmorum* both cause foot and root rot of wheat in warm, dry soil. However, *F. graminearum* is associated with slightly warmer and drier soil than *F. culmorum*. The occurrence of severe *Fusarium* crown and root rot of wheat in this region is probably the result of water potentials harmfully low to host physiology but nearly ideal for the pathogen (Cook 1973).

Several reports (Sasi 1980; Sasi *et al.* 1981; Hill 1984) demonstrated the existence of differences in lesion size due to *F. culmorum* among cultivars when wheat seedlings were grown under water stress conditions. Under Moroccan conditions of high root rot incidence and severity, high yield losses were detected during the 1986-87 dry season compared to the wetter 1985-86 season (Lyamani 1988). Similarly, root rot development under water stressed conditions was significantly superior to that scored under irrigated trials in the Moroccan West Central region during 1988-1991 period. White heads scores reached 65% for Durum wheat cultivar Marzak at Tessaout Station (Mergoum 1991; Mergoum *et al.* 1994). Positive correlations have been established between the onset of water stress, foot rot severity, and yield reduction (Papendick *et al.* 1971; Grey *et al.* 1991).

Fungal responses to water potential vary with temperatures (Bruehl and Cunfer 1971; Manandhar and Bruehl 1973). Isolates of *F. culmorum* from different areas exhibit optimum growth at -0,8 to -1,4 Mega Pascal (MPa) at 20 to 30°C and -2,8 MPa at 35°C, (Cook and Christen 1976). Prolonged moisture stress coupled with relatively high soil temperature in the fall are important factors which enhance early development of root rot on the underground plant parts (Fenster *et al.* 1972). Soil temperatures at planting time and subsequent root and crown intensity were strongly correlated. It was concluded that a soil temperature of 65°F at planting time was optimum (Fenster *et al.* 1972). Higher temperatures which usually coincided with early plantings, caused yield losses due to severe root rot. Low temperatures, however, caused less plant development (Fenster *et al.* 1972) and freezing temperatures may predispose wheat plant to damage by fungi normally considered weak such as *F. roseum* types and *C. sativus* (Fernandez *et al.* 1985).

### **Agronomic factors**

Development of plant and soil microflora including root rot pathogens are affected by soil fertilization. There is a general agreement that inadequately nourished wheat and barley seedlings are more disposed to disease attack (Papendick and Cook 1974; Verma *et al.* 1975). On the other hand preliminary studies on the effects of nitrogen on root rot incidence conducted in the Gharb region of Morocco showed that high levels of nitrogen enhanced disease development on wheat (Baye 1984); these results however, conflicted with those obtained in the West Central regions of Morocco (Mergoum 1991). It is believed that excess of nitrogen in cereals enhances root rot severity (Smiley *et al.* 1972) as a result of promoted growth and increased transpiration of plants which rapidly deplete the soil moisture (Garrett 1976; Cook 1980; Patly 1981). Seedling blight caused by *Fusarium* spp. averaged 11 to 14% in the presence of fertilizer (N, P, K) and residues and 1 to 2% when both were absent.

The form of nitrogen fertilizer may influence the severity of many diseases specially of those caused by facultative parasites (Smiley *et al.* 1972). Studies on *Fusarium* root rot (Rowaished 1981) showed that infection and disease severity



was higher in plants supplied with ammonium nitrogen ( $\text{NH}_4$ ) than in those receiving nitrate nitrogen ( $\text{NO}_3$ ). The influence of phosphate fertilizer has also been investigated and results were variable. High soil phosphate levels resulted in limited root rot infection compared to low phosphate levels (Piening *et al.* 1969; Verma *et al.* 1975).

Potassium effects on root rot pathogens are not well defined. While nitrogen and phosphate seemed to decrease disease incidence in early stage of crop growth, potassium tends to enhance it. At later stages however, the roles are reversed, i.e. nitrogen and phosphate tend to increase disease development (Onuorah 1969).

Several investigators have studied the relationship between date of planting and root or crown disease intensity and severity (Fenster *et al.* 1972; Willams *et al.* 1980; Mergoum 1991). In the USA, root and crown rot intensity was the greatest in early planted seeding and progressively diminished with late planting of wheat (Fenster *et al.* 1973; Mergoum 1991). Early planting is related to high soil temperatures. High correlations between soil temperature at planting time and subsequent root and crown rot incidence were established (Willams *et al.* 1980). Early planting results in a depletion of soil moisture because of heavy fall plant growth, thus enhancing root and crown rot disease because of drought stress (Watkins and Kerr 1980; Fenster *et al.* 1972; Mergoum 1991).

Seeding rate reported to significantly affect root rot disease development (Papendick and Cook 1974). Disease severity increased with increased seeding rates. However, no correlation could be detected between disease severity and seeding rate by other workers (Tinline 1986; Broschous and Frank 1986; Mergoum 1991). Similar results were obtained for the effects of spacing and interplanting barley plants on root rot (Piening 1983; Broschous and Frank 1986).

Rotation of crops resulted in more diverse soil microflora including *Fusarium* species (Nash and Snyder 1965; Windel and Kommedahl 1971) compared to monocropping the same crop (Ledingham 1961; Willams and Schmitthenner 1962). Rotations with crops other than wheat, barley, corn, or western ryegrass will reduce the level of root rot in succeeding wheat or barley crops (Cassini 1967; Cook 1968a; Stack 1982; Wildermuth and McNamara 1991).

## CONTROL

### Chemical

Seed treatments with available chemicals have a limited value for the control of cereal root rot since the pathogens are in the soil and infection can take place after the protective period of the fungicide treatment is over (Colhoum 1972; Watkins and Kerr 1980; Stack 1982; Mergoum 1991). However, seed treatment with an appropriate fungicide may be used to reduce preemergence and

postemergence "damping off" of seedlings (Fenster *et al.* 1972; Watkins and Kerr 1980). Several chemicals have been evaluated for root rot control and have yielded variable results (Burrage and Tinline 1960; Mergoum 1991). Seed treatments with systemic fungicides such as Thiabendazole (Cassini 1967; 1970) and Benomyl + Thiram (Diehl *et al.* 1983) are the most effective fungicides to control *Fusarium* infection. Other fungicides such as Nuarimol and Fenapronil were reported to be effective against *C.sativus* (Diehl *et al.* 1983). Promising results were obtained in controlling root rot in spring wheat by using Triadimenol, Imazalil and Nuarimol (Verma *et al.* 1986). However, Imazalil was effective to reduce significantly root rot winter wheat under Colorado, USA conditions (Mergoum 1991).

Population of *F. culmorum* and saprophytic *Fusarium* were reduced within retention zone following injection of anhydrous ammonia (NH<sub>3</sub>) into the soil in the field (Smiley *et al.* 1972). Further studies, however, showed small or no reduction of *Fusarium* propagules in the tillage layer when anhydrous ammonia was applied to the soil. This was attributed primarily to insufficient distribution of anhydrous ammonia throughout the tillage layer (Smiley *et al.* 1972).

### Biological

Early work (Anwar 1949) on biological control of root rot demonstrated a depressive effect of natural soil microflora on cereal rotting pathogens. *Trichoderma harzianum* was reported to have excellent inhibiting action against both *F. culmorum* and *C. sativus* (Dymovych 1960; Uoti 1976b). However, this microorganism was not effective against root and foot rot pathogens of winter wheat in Colorado, USA (Mergoum 1991). Other organisms including *Pyronema confluens*, *Penicillium* spp., *Chaetomium cochhoides*, and *Chaetomium globosum* have also shown some degree of biological control of cereal root rot pathogens (Uoti 1976 b).

### Cultural practices

The most effective means to control cereal root and crown rot is by good crop management practices which reduce environmental stress, particularly water stress. Crop rotation is an important tool for the farmer to reduce root rot effects (Stack 1982). Rotation to any crop resistant to the causal agent of the disease to allow an appropriate time between successive wheat or barley crops probably will reduce the pathogen populations to a low level (Stack 1982; Baye 1984; Wildermuth and McNamara 1991).

Planting seedbed, tillage which permits the incorporation of the straw residues deep into the soil, the use of large and heavy seeds which produce vigorous plants, and seed treatment with appropriate chemicals may reduce root rot damage (Fenster *et al.* 1972; Williams *et al.* 1980; Watkins and Kerr 1980).

Adequate levels of nitrogen (Ledingham 1970; Smiley *et al.* 1972; Papendick and Cook 1974; Cook 1980) and potassium (Stack, 1982) can contribute to a significant reduction in the root rot severity.

While early planting gives good plant stands, it generally coincides with high soil temperatures and may result in the depletion of soil moisture which enhances disease development. Late planting conserves soil water reserves but may result in poor plant growth which increases the chances of winter injury and "blow out" of plants (Fenster *et al.* 1972; Williams *et al.* 1980; Mergoum 1991).

### Genetic resistance

The key to the practical control of root rot appears to lie in the development of resistant cultivars (Sallans and Tinline 1965). Lack of suitable techniques for consistently distinguishing differences in resistance among cultivars and the absence of sources of immunity or resistance are the main obstacles in identifying varieties (Butler 1961; Mergoum 1991). However differences between wheat cultivars was obtained under field conditions with the greatest differences occurring when inoculum was spread alongside the growing plants (Dodman and Wildermuth 1985). Differences in varietal reaction in the field have been demonstrated (Dodman and Wildermuth 1987; Purss 1965; Mergoum 1991; Mergoum *et al.* 1994), but none of the cultivars tested showed evidence of high resistance (Hanson *et al.* 1950; Purss 1965; Mergoum 1991). Selection for yield has undoubtedly eliminated the most susceptible lines, but it has not been successful in improving cultivar resistance enough to prevent yield losses.

Comparisons of the reaction to root rot caused by *C. sativus* and *Fusarium* spp. of 14 lines *Triticum* species revealed the resistance of lines of the diploids *T. aegilopoides* and *T. monococcum* and the susceptibility of lines of the hexaploid *T. compactum*. *T. timopheevi* lines appeared more resistant than those of the other tetraploid species (Harding 1972). Test of 10 cultivars of durum wheat and 10 cultivars of bread wheat demonstrated that although significant differences existed between cultivars, in general durum wheat were the most susceptible (Mergoum 1991; Mergoum *et al.* 1994). However, screening 1130 accessions of durum wheat from the world collection showed the existence of excellent sources of tolerance to root rot in Morocco. Unfortunately, these accessions are in general agronomically undesirable and therefore need many genetic improvements (Mergoum *et al.* 1994). Test of the chromosome substitution for the D genome in winter wheat by chromosome 1 to 7 of *Aegilops squarrosa* L. and *Agropyron* species demonstrated that substitution had no consistent effects on root rot severity caused by *C. sativus* (Conner *et al.* 1989). In general, common wheat appears to be more resistant than durum wheat (Cassini 1967; Statler and Darlington 1972; Baye 1984; Mergoum 1991; Mergoum *et al.* 1994).

When both *C. sativus* and *Furarium* root rot pathogens were used as inoculum, there was no high resistance exhibited by any tested wheat genotype (Hanson *et al.*, 1950; Sims *et al.* 1961) although some cultivars showed significant

differences in reaction (Statler and Darlington 1972; Lyamani 1988; Mergoum 1991; Mergoum *et al.* 1988; 1994).

The seedling stage of wheat appeared to be more susceptible than later stages (McNight and Hart 1966; Statler and Darlington 1972; Mergoum 1991). However, some cultivars were severely damaged by *F. roseum* alone or in combination with *C. sativus* at the seedling stage as well as at the adult stage (Statler and Darlington 1972). Other findings confirmed that the degree of resistance to *F. graminearum* infection depends upon cultivars and maturity stage (Schroeder and Christensen 1963).

Studies on the inheritance of varietal reaction to root and crown rot yielded variable results. Evidence of segregation to the disease in the F3 (Hanson *et al.* 1950) and other segregating generation progenies (Sallans and Tinline 1965) was obtained. Reports on the inheritance of root rot resistance in various crosses showed that the reaction was governed by a single gene pair (Arny 1948; 1951; Larson and Atkinson 1981; 1982), or many gene pairs (polygenic) (Cohen *et al.* 1969).

## INOCULATION AND SELECTION TECHNIQUES

Selection of resistant cultivars to root pathogens has been hindered by inadequate, inconsistent inoculation methods, and lack of accurate and suitable techniques for measurement of the disease response. Several inoculation techniques have been tried including natural infection (Tinline and Ledingham 1979), incorporation of the inoculum onto or into the soil (Dodman and Wildermuth 1987; 1985; Fernandez *et al.* 1985; Cook 1980), seed inoculation (Grey *et al.* 1991; Dodman and Wildermuth 1987; 1985; Harding 1971, Ismail and Michalik 1980; Mergoum 1991; Mergoum *et al.* 1994), spraying the plants with a suspension of conidia (Dodman and Wildermuth 1987), and taping infested wheat straw to the plant subcrown internodes (Tinline 1977). An inoculation method using an agar disc containing conidia of the pathogen was used on roots (Armitage 1984; Hill *et al.*, 1987; Mergoum 1991; Mergoum *et al.* 1988), on leaves (Hill 1984), and on cut stems (Sasi *et al.* 1981).

The incidence and severity of lesioning and discoloration of the subcrown internodes have been used for disease assessment since the above ground symptoms are seldom observed (Ledingham *et al.* 1973; Mergoum 1991). Plants are classified into 4 groups: severe, moderate, slight, and clean depending upon the extent of discoloration of the subcrown internodes.

Disease rating classes from 1 to 4 based on disease severity and percent of diseased plants were elaborated and have been used in several studies to assess yield loss due to root rot (Verma *et al.* 1976b; Tinline and Ledingham 1979; Mergoum 1991). Premature ripening and bleaching of the spikes commonly known as "white heads" or "dead heads" percentages were successfully used in

order to discriminate between reactions of wheat genotypes (Mergoum 1991; Mergoum *et al.* 1994).

Other disease scoring techniques have been used including the percent of diseased plants or tillers (Dodman and Wildermuth 1985; 1987; Lyamani 1988), the ease in pulling plants from the soil (Kommedahl and Patel 1960), seedling reactions (Harding 1971; Hill *et al.* 1987; Mergoum 1991; Mergoum *et al.* 1988), percentage of recovery of disease seedlings (Armitage 1984), changes in amino acids, total protein content, and production of toxic substances which inhibit seedling growth (Ismail and Michalik 1980; Rabie *et al.* 1986). All of these techniques have yielded variable and inconsistent results and have not been effective in differentiating small quantitative differences in disease resistance among genotypes.

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