Matthew P. Reynolds
Hans-Joachim Braun *Editors*

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Chapter 9
Globally Important Non-rust Diseases of Wheat

Xinyao He, Navin C. Gahtyari, Chandan Roy, Abdelfattah A. Dababat, Gurcharn Singh Brar, and Pawan Kumar Singh

Abstract While the three rusts are the most predominant wheat diseases in the global scale, various other diseases dominate in different geographical regions. In this chapter, some major non-rust diseases of wheat with global and/or regional economic importance are addressed, including three spike diseases (Fusarium head blight, wheat blast, and Karnal bunt), four leaf spotting diseases (tan spot, Septoria nodorum blotch, spot blotch, and Septoria tritici blotch), and several root diseases.

Keywords Head blight diseases · Leaf spotting diseases · Root diseases

9.1 Learning Objectives

- To learn the major epidemic regions, causal agent(s), epidemiology, management, genetics, resistance breeding etc. of each disease.

X. He · P. K. Singh (✉)
International Maize and Wheat Improvement Center (CIMMYT), Texcoco, Mexico
e-mail: x.he@cgiar.org; pk.singh@cgiar.org

N. C. Gahtyari
ICAR-Vivekananda Parvatiya Krishi Anusandhan Sansthan, Almora, Uttarakhand, India
e-mail: navin.gahtyari@icar.gov.in

C. Roy
Department of Plant Breeding and Genetics, Bihar Agricultural University, Bhagalpur, Bihar, India

A. A. Dababat
International Maize and Wheat Improvement Center (CIMMYT), Ankara, Turkey
e-mail: a.dababat@cgiar.org

G. S. Brar
Faculty of Land and Food Systems, The University of British Columbia, Vancouver, BC, Canada
e-mail: gurcharn.brar@ubc.ca

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9.2 Introduction

Wheat production is challenged by a range of diseases, rusts and non-rusts, causing on average 10–28% of yield losses globally according to a recent estimation [1]. The diseases can cause infection on all parts of the wheat plant (Fig. 9.1) and are strongly influenced by environmental conditions and disease management strategies. In the Sects. 9.3, 9.4 and 9.5, several major wheat diseases are presented according to their infection sites, i.e., spike, leaf, and root, and the most important information of each disease is summarized.

Fig. 9.1 Disease symptoms for (1) Fusarium head blight, (2) wheat blast, (3) tan spot, (4) spot blotch, (5) Septoria tritici blotch, and (6) cereal cyst nematode

9.3 Spike Diseases

9.3.1 Fusarium Head Blight

Fusarium head blight (FHB) is one of the most devastating diseases of wheat globally, with major epidemic regions in North America, Europe, East Asia, and the Southern Cone of South America. Many species in the genus *Fusarium* cause FHB, but it is *F. graminearum* species complex that has global importance and has been found in all major epidemic regions. The disease is favoured by warm and humid environment around anthesis, leading to yield reduction and quality deterioration. More importantly, the disease produces a range of mycotoxins, particularly deoxynivalenol (DON, or vomitoxin), which are toxic to humans and animals, raising a serious concern to food and feed safety. In the USA, losses attributable to FHB in wheat and barley between 1993 and 2001 were estimated at $7.67 billion. In China, the epidemic has increased significantly in the last two decades, affecting on average 5.3 Mha and reached 9.9 Mha in the 2012 great epidemic [2]. Yield reductions can reach up to 70% in Europe and South America [3].

FHB resistance is a typical quantitative trait, conditioned by numerous genes of minor effects. Several types of resistance have been proposed, represented by resistance to initial infection (Type I), resistance to disease spread within spike tissues (Type II), resistance to toxin accumulation (Type III), resistance to kernel infection (Type IV), and resistance to yield loss (Type V) [3]. Numerous sources of resistance were reported in literature; but only a few have been successfully utilized in breeding programs, such as 'Sumai 3', 'Wuhan 1', 'Frontana' etc. [3]. FHB resistance genes/QTL (Quantitative trait loci) have been mapped on all the 21 wheat chromosomes, though, only seven QTL have been formally designated as Mendelized genes, of which only *Fhb1*, *Fhb2*, *Fhb4,* and *Fhb5* are from common wheat, whereas *Fhb3*, *Fhb6,* and *Fhb7* are from wild wheat relatives [4]. So far, only *Fhb1* and *Fhb7* have been cloned, and their functional markers have been developed for marker-assisted selection (MAS).

Generally, two breeding strategies for FHB resistance could be utilized, i.e., exploitation of native resistance and introduction of exotic resistance. There is no strong FHB resistance available in the current CIMMYT gene pool; though, some moderately resistant lines have been identified and a few QTL with major effects have been mapped. Among those lines are 'Shanghai3/Catbird', 'Mayoor', 'Soru#1', 'IAS20*5/H567.71' etc. Apart from a major QTL on 2DL, others are either of low frequencies or of minor effects, but higher level of resistance can still be achieved via accumulating those QTL in elite breeding lines, similar to rust resistance breeding [5]. The limitation of using native resistance is, however, a lack of QTL/gene with strong Type II resistance, which could be compensated via introduction of exotic FHB resistance genes, like *Fhb1* and *Fhb7*. The former is the most well-known FHB resistance gene and has been extensively utilized in China, USA, and Canada; however, its resistance allele is tightly linked with the susceptibility allele of the stem rust gene *Sr2*, limiting its application in the CIMMYT wheat breeding.

To address this problem, several recombinant lines with both *Fhb1* and *Sr2* were introduced from Australia and included in various crosses with elite CIMMYT breeding lines [6].

Since no immunity to FHB has been found in wheat and high level of FHB resistance is difficult to achieve, other disease management strategies are also important in wheat production regions where FHB is a limiting factor. Removal of crop residue and rotation with non-host crops are helpful in reducing inoculum concentration. It is well known that maize-wheat rotation greatly increases the risk of FHB and thus should be avoided; otherwise, integrated disease management including deep tillage, fungicide application, and growing FHB resistant or moderately resistant cultivars are recommended.

9.3.2 Wheat Blast

Wheat Blast (WB) caused by the ascomycetes fungus *Magnaporthe oryzae* pathotype *triticum* (MoT) is one of the devastating diseases in warm and humid growing region. It can infect all the aerial parts of wheat, but completely or partially bleached spike is the typical symptom. WB is a new disease and was initially identified in the Parana state of Brazil in 1985; afterwards, its rapid widespread to the neighbouring states in Brazil and other countries of South America raised serious concerns. The first WB outbreak outside South America was reported in Bangladesh in 2016, raising a major concern on wheat production in South Asia (SA), as nearly 17% of the wheat growing areas in SA are vulnerable to WB. More recently, occurrence of WB has been reported from Zambia which can be a major threat for wheat production and trade in Africa [7]. Under favourable temperatures of 25–30 °C and high humidity, the disease can cause high yield loss ranging from 10% to 100% depending upon the level of infection.

The long-distance spread of the pathogen occurs through infected commercial grains, followed by the air transmission; therefore, grain treatment (chemical or irradiation) can effectively manage the primary inoculum load. For field WB management, foliar fungicides' application such as demethylation inhibitors (DMI), quinone outside inhibitor (QoI), succinate dehydrogenase inhibitors (SDHI) are suggested to be used in combination/rotation so as to reduce the fungal resistance against the fungicides especially QoI [8]. Various agronomic practices *viz.* optimizing planting dates, weed management, crop rotation with non-hosts, and avoid excessive nitrogen application are reported to be effective in WB control. However, these should be used in combination with genetic resistance to achieve a better management.

Regarding host resistance, the 2NS/2AS translocation has been widely acknowledged as a stable and effective resistance source, although virulent isolates have emerged recently in South America. The translocation was introduced from *Ae. ventricosa* and has been widely utilized in wheat breeding due to rust resistance genes (*Yr17, Lr37, Sr38*), as well as resistance genes for nematodes (*Cre5, Rkn3*) and

WB. The 2NS/2AS translocation is an excellent example for the potential from crossing with wild relatives of wheat, for more examples refer to Chaps. 16, 17 and 18. Most well-known WB resistant lines have the 2NS/2AS translocation, e.g. 'Milan' and 'Borlaug #100' in the CIMMYT germplasm, 'Sausal CIAT', 'CD 116', 'Caninde #1' in South America, 'BARI Gom 33' in Bangladesh, 'HD2967', and 'DBW189' in India [9]. Recent genetic studies involving diverse wheat germplasm identified only one stable QTL on 2NS/2AS, whereas the remaining QTL were of small effects and were detected in only some environments (Singh et al. unpublished data). This highlights the importance of identification of new WB resistance genes for breeding use, which could alleviate the selection pressure that is being applied to 2NS virulent isolates, to prolong the lifespan of 2NS varieties.

A few resistance genes have been reported to have major effects at seedling (leaf resistance) but not at adult-plant (spike resistance) stages, among which, *Rmg2, Rmg3, Rmg7, Rmg8,* and *RmgGR119* are effective against MoT, whereas *Rmg1*, *Rmg4, Rmg5*, *Rmg6,* and *RmgTd(t)* are effective against non-MoT species. It is important to mention that *Rmg2, Rmg3,* and *Rmg7* have been overcome by new MoT isolates, whereas *Rmg8* and *RmgGR119* exhibited effective resistance in greenhouse but need to be validated in large scale field trials [9].

Early WB resistance breeding in South America depended heavily on natural infection, which was sporadic and unpredictable, with great variation in disease pressure. As for countries being threat by WB but still do not have the disease (like India), or those have WB but do not have the screening capacity (like Zambia), the request for an international precision phenotyping platform (PPP) is very strong, where interested cooperators can evaluate their wheat lines for reaction to WB. In collaboration with its national partners, CIMMYT has established three WB PPPs, with one in Bangladesh (Jashore), and two in Bolivia (Quirusillas and Okinawa) to screen germplasm and advanced lines from across the globe. High quality phenotypic data have been produced from the three PPPs, which greatly facilitated the WB resistance breeding, germplasm screening, as well as genetic studies [9].

9.3.3 Karnal Bunt

Tilletia indica (syn. *Neovossia indica*) is a hemibiotrophic fungus which was first described to cause disease in the Indian city of Karnal, hence called 'Karnal bunt' (KB). Currently, the disease is distributed in parts of Asia (India, Nepal, Pakistan, Iraq, Iran, Afghanistan), Africa (South Africa), and the Americas (USA, Mexico, Brazil). Though the estimated yield losses in KB affected regions are minimal (below 1%), it is an important disease from international trade perspective, where many member countries of WTO have zero tolerance quarantine laws. KB significantly deteriorates the wheat quality in terms of reduced vitamins, amino acids, weakened dough, and loss in flour recovery, ultimately affecting the human consumption negatively [10].

The conducive conditions for disease development are high humidity with cool temperature (<20 °C) favoring teliospore germination. Infected spikes disperse teliospores that become inoculum for the next season, and the teliospores are reported to remain viable for up to five years in soil under natural conditions, indicating the spatial and temporal dispersal capability of the disease. Boot emergence to anthesis is the optimum stage for a germinated teliospore to infect, however, an infection can happen as late as at late dough stage [11]. Treating seed with Chlorothalonil or mixture of carboxin & thiram and foliar spray with propiconazole, triadimefon and carbendazim are the suggested chemical control measures. The natural populations of *T. indica* have high genetic diversity owing to the sexual recombination leading to high diversity for virulency of KB strains as well as diversity in the wheat genotypes for resistant/susceptible reaction against the disease.

In the early days of KB resistance breeding at CIMMYT, important genetic stocks used were 'Aldan/IAS58' from Brazil, 'Shanghai-7' from China, and native CIMMYT lines 'Roek//Maya/Nac', 'Star', 'Vee#7/Bow' and 'Weaver'. To date, screening programs have resulted in the identification of numerous resistant sources for bread wheat and durum wheat from various countries as reviewed in Bishnoi et al. [10]. Additional resistant sources have been identified in primary to tertiary gene pools of wheat including *T. urartu* (AA) and *Ae. tauschii* (DD). Durum and triticale are generally more resistant than bread wheat.

Genetic resistance against KB is governed by polygenes with quantitative inheritance, although gene-for-gene interaction may exist to some extent. Many genes with small additive effects acting in an additive and epistatic mode impart KB resistance. Stacking additive genes along with an eye for significant epistatic gene interactions can enhance levels of KB resistance. In QTL mapping studies, as expected, majority of the identified QTL had minor effects, and only a few major QTL have been identified on chromosomes 4B, 5B, and 6B, where the one on 4B associated with SSR marker *Xgwm538* had the largest effect (R^2 of 25%). A GWAS study on 339 accessions from Afghanistan led to the identification of a consistent QTL on chromosome 2BL along with some other novel genomic regions [12].

9.4 Leaf Spotting Diseases

9.4.1 Tan Spot

Tan spot (TS) is caused by the necrotrophic fungus *Pyrenophora tritici-repentis* (Died.) Drechs. The disease frequently appears in the warm and humid growing regions of bread and durum wheat, especially in Canada, Australia, USA, and South Africa. Yield and quality losses are common under high disease pressure. Reduced or no-till approaches to prevent soil erosion and water management are important reasons for increased disease pressure and TS infections can therefore be a challenge

in using conservation agriculture practices. Another major reason that corresponds with increased pathogen virulence is the acquisition of a host-selective toxin (HST) PtrToxA by *P. tritici-repentis* from *Stagonospora nodorum* via horizontal gene transfer, which overcame the resistance of most cultivars carrying *Tsn1* gene. So far, three HSTs have been identified from *P. tritici-repentis*, acting as pathogen virulence factors in the TS pathosystem. Based on type of lesion (chlorosis or necrosis) and HSTs produced, *P. tritici-repentis* is classified into eight races using six differential genotypes (Table 9.1).

Pyrenophora tritici-repentis is a necrotroph and follows inverse gene-for-gene relationship where recognition of host sensitivity gene by pathogen produced HST results in a compatible (susceptible) interaction. This is opposite to Flor's classical gene-for-gene model in biotrophic diseases such as mildews and rusts, where host resistance gene is recognized by pathogen avirulence (*Avr*) gene, leading to an incompatible (resistant) reaction. High level of resistance has been found in several wheat genotypes although immunity is not reported [13]. Host resistance in wheat against TS can be qualitative or quantitative and some of the most well-characterized genes are *Tsn1* (interacts with PtrToxA), *Tsc2* (interacts with PtrToxB), and *Tsc1* (interacts with PtrToxC). *Tsn1* is the only cloned TS resistance gene, which is located on chromosome 5BL and a dominate functional marker *Xfcp623* is used for MAS [14]. *Tsc1* is located on chromosome 1A and *Tsc2* on 2BS, for which flanking markers are available for MAS. In addition to these three major genes, a recent meta-QTL study identified 19 QTL/loci for resistance to TS which can be utilized in wheat breeding programs [15].

Resistance breakdown is a major concern in R-genes conferring resistance to biotrophic pathogens as the pathogen *Avr* genes mutate rapidly. In case of TS

Table 9.1 Reaction of eight characterized races of *Pyrenophora tritici-repentis* on bread and durum wheat differential lines. R and S indicates resistant and susceptible response, respectively

Race	Associated toxins	Reaction of differential genotypes					
		Glenlea	6B662	6B365	Salamouni	Coulter	4B1149
1	PtrToxA, PtrToxC	S (necrosis)	R	S (chlorosis)	R	S (necrosis)	R
2	PtrToxA	S (necrosis)	R	R	R	S (necrosis)	R
3	PtrToxC	R	R	S (chlorosis)	R	S (necrosis)	R
4	None	R	R	R	R	R	R
5	PtrToxB	R	S (chlorosis)	R	R	S (necrosis)	R
6	PtrToxB, PtrToxC	R	S (chlorosis)	S (chlorosis)	R	S (necrosis)	R
7	PtrToxA, PtrToxB	S (necrosis)	S (chlorosis)	R	R	S (necrosis)	R
8	PtrToxA, PtrToxB, PtrToxC	S (necrosis)	S (chlorosis)	S (chlorosis)	R	S (necrosis)	R

resistance, if sensitivity genes are knocked-out or mutated, the pathogen cannot evolve as rapidly as biotrophs, so the resistance is more durable. Additionally, the fungus is saprophytic in nature and selection pressure on the pathogen would not be as high as in mildews or rusts. Molecular markers associated with major loci conferring susceptibility or resistance are very useful to select for TS resistant cultivars. Stacking of multiple QTL (including race non-specific) for TS resistance is an important and desirable strategy to manage the disease [15].

9.4.2 Septoria Nodorum Blotch

Stagonospora nodorum, a filamentous ascomycetes fungus, causes wheat leaf and glume blotch and affects wheat yield and quality in the warm and humid areas particularly in Australia, USA, parts of Europe and southern Brazil. Short incubation period enables the pathogen for multiple infection cycles within a season. The fungus can reproduce through asexual conidia and frequent sexual reproduction due to availability of both mating types (MAT1-1 and MAT1-2) that makes sexual reproduction possible.

Stagonospora nodorum produces multiple HSTs, of which 15 have been identified so far. The HSTs (e.g., SnToxA) interact with the corresponding host sensitivity genes (e.g., *Tsn1*) in an 'inverse gene-for-gene' manner that causes infection in the host, just as in TS. So far, nine necrotrophic effector (NE) and sensitivity gene interactions *viz.* SnToxA-*Tsn1*, SnTox1-*Snn1*, SnTox2-*Snn2*, SnTox3-*Snn3-B1*, SnTox3-*Snn3-D1*, SnTox4-*Snn4*, SnTox5-*Snn5*, SnTox6-*Snn6*, and SnTox7-*Snn7* have been identified in wheat. Three important NE genes in the pathogen *viz. SnToxA*, *SnTox1*, *SnTox3* and one important host sensitivity gene in wheat *viz. Tsn1* have been cloned which has helped in the extensive study of three important interactions *viz.* SnToxA-*Tsn1*, SnTox1-*Snn1* and SnTox3-*Snn3-B1* for better understanding the molecular basis of Septoria nodorum blotch (SNB) [16]. *Tsn1* was identified on chromosome 5BL [14], whereas both *Snn1* and *Snn3-B1* were mapped on 5BS [17]. Negative selection of host sensitivity genes during the breeding program would accelerate the breeding progress of resistant varieties.

An integrated disease management strategy including cultural practices, fungicides application, and use of resistant varieties is most effective in managing SNB. Infected seed and straw serve as the primary source of inoculum; therefore, seed treatment, crop rotation, and residue management reduce the chances of an epidemic in the disease-prone areas. SNB infection causes the greatest yield losses at the adult plant stage, for which resistance screening should be emphasized [18]. Genetic analysis revealed both qualitative and quantitative nature of resistance; but the latter dominates in field resistance against SNB [16]. Quantitative resistance is reported to have low to moderate heritability, thus high selection intensity should be kept to obtain higher genetic gain for SNB resistance. QTL associated with SNB resistance have been identified on multiple wheat chromosomes [18], yet few have been utilized in breeding.

9.4.3 Spot Blotch

Spot blotch (SB) caused by *Bipolaris sorokiana* (telemorph *Cochliobolus sativus*) is a destructive disease of wheat in the warm and humid growing regions, especially South Asia, Latin America, and Southern Africa. The pathogen causes average yield loss of 15–20%; but yield loss of up to 87% has been detected on the susceptible varieties [19]. The pathogen can infect all parts of the wheat plant, but leaf infection is the most typical, where infection starts from the older leaves and then progresses upward towards the younger leaves. High temperature (18–32 °C) and humidity (>90%) favours the disease establishment.

Identification of resistance sources through screening of national and international germplasm stocks was initiated in early 1980s and initial success was accomplished by replacing most susceptible varieties with the resistant lines in Brazil. Several resistant lines such as Saar, M 3, Yangmai 6, BH 1146, Shanghai 4, Ning 8201 including synthetic derivatives like 'Chirya 1', 'Chirya 3', 'SYN1' were identified as potential donors. Leaf tip necrosis (*Ltn+*) is associated with moderate resistance to SB, allowing breeders to use it as a phenotypic marker during selection. No host immunity has been reported for SB, and genetic studies on field SB resistance revealed a quantitative nature of inheritance [20].

To date, four major QTL (*Sb1-Sb4*) conferring SB resistance have been mapped. *Sb1* was mapped on chromosome 7DS, co-located with the cloned leaf rust resistance gene *Lr34* having pleiotropic effects on yellow rust (*Yr18*), stem rust (*Sr57*), powdery mildew (*Pm38*) and leaf tip necrosis (*Ltn+*). *Sb2* was identified on chromosome 5BL, *Sb3* on 3BS, and *Sb4* on 4BL [21]. These QTL can be used to develop new varieties or transferred into popular susceptible varieties through marker-assisted back cross (MABC) programme. Apart from these four *Sb* genes, *Tsn1* on 5BL has been shown to have major effects against *B. sorokiniana* isolates with *ToxA* [22]. Such *ToxA+* isolates have been identified in the *B. sorokiniana* populations of Australia, USA, India, and Mexico [23], implying that removing *Tsn1* from popular wheat varieties enhances resistance not only to TS and SNB, but also to SB. Contribution of QTL with minor effects is also significant in reducing SB severity, and such QTL have been mapped on chromosomes 1A, 1B, 1D, 2B, 2D, 3A, 3B, 4A, 4B, 4D, 5A, 5B, 6A, 7A, 7D in bi-parental and GWAS mapping studies [19].

9.4.4 Septoria Tritici Blotch

Septoria tritici blotch (STB) is caused by the fungal species *Zymoseptoria tritici* (teleo. *Mycosphaerella graminicola*). The pathogen is heterothallic with two mating types that have frequent sexual reproduction, resulting in a high level of genetic variation and an accelerated evolution and diversification of the fungal pathogen.

This in turn leads to problems like break down of host resistance and fungal resistance to fungicide. Losses to STB can range from 30% to 50% during severe epidemics, but typically are much lower. Epidemics are most severe in areas with extended periods of cool and wet weather, particularly North America (USA, Canada, Mexico), East Africa (Ethiopia, Kenya), South America (Brazil, Chile, Uruguay, Argentina) and the most damage occurs in Europe and CWANA (Central and West Asia and North Africa) region [24].

Host resistance to STB can be both qualitative and quantitative, but there is no clear difference between them since the gene-for-gene interaction in the wheat-STB pathosystem does not confer complete resistance. So far, 22 resistance genes have been designated, of which 21 were identified from hexaploid wheat, i.e. *Stb1* through *Stb19*, *StbSm3* and *StbWW*, and only one gene, *TmStb1*, has been found in *T. monococcum* [25]. So far, *Stb6* and *Stb16q* are the only two STB resistance genes that have been cloned, and their respective functional markers have been developed for MAS [26]. A total of 89 genomic regions carrying QTL or meta-QTL have been identified on all but 5D chromosomes, as summarized by Brown et al. [25].

The breeding effort for STB resistance began in 1970s in CIMMYT, using resistance sources from Brazil, Russia, Argentina, and China [27]. Nowadays, CIMMYT materials, represented by the International Septoria Observation Nurseries (ISEPTON), exhibit very good STB resistance under Mexican environments due to the consistent selection against the local *Z. tritici* strains. However, their performance in other countries varies greatly, due to different *Z. tritici* populations, although promising lines can still be identified. A vivid example is the resistance of durum wheat, which is nearly immune in Mexico but becomes highly infected in North Africa. Recent genetic studies on the STB resistance mechanism for CIMMYT lines revealed a nature of quantitative inheritance, with multiple minor QTL and limited major QTL (Singh et al., unpublished). This minor gene-based resistance mode is preferred as it likely confers durable resistance, as evidenced in resistance to many wheat diseases represented by rusts [5].

Plant height (PH) and days to heading (DH) are often negatively associated with STB resistance/escape, i.e., tall and late lines tend to have low STB. The association between short stature and high STB infection was a major issue that hampered the promotion of semi-dwarf wheat varieties in STB affected areas, especially in North Africa where STB is a priority biotic constrain. Efforts have been made to break such association, which resulted in the identification of intermediate maturing, high yielding semi-dwarf lines with high STB resistance [27]. It is noteworthy that such association exists in many abovementioned wheat diseases, like FHB, SNB, SB and TS. Such association is contributed mostly by disease escape, although tight linkage between resistance QTL and PH/DH associated genes and pleiotropic effects of the latter genes could be involved.

9.5 Root Diseases

Soil borne pathogens (SBPs) include the *Heterodera* species, cereal cyst nematode (CCN), *Pratylenchus* species, root lesion nematode (RLN) and many additional fungal species. Among the later are Take-all (GGT, *Gaeumannomyces graminis var. tritici*), *Pythium* spp, *Rhizoctonia solani,* Crown rot (CR, *Fusarium* spp), and common root rot (CRR, *Bipolaris sorokiniana*) (Table 9.2). These pathogens are favoured by different soil, cropping system and climate [28], and are found wherever cereal-based farming systems dominate. SBPs attack the roots of cereal crops resulting in a high yield loss and reduced grain quality. The damage caused by these pathogens is more visible in fields where drought and monoculture practices dominate. Rain-fed wheat under sustainable agriculture production, especially those grown under arid and semi-arid conditions, is being impacted by climate change due to hotter and drier soils. Under the harsh climatic condition characterized by low precipitation and high temperature, yield losses can exceed 50%. However, the available reports regarding wheat grain yield losses do not accurately portray the magnitude of economic losses at the regional or national levels, since those reports have been mostly linked to research plots located in infested areas of fields i.e., sick plots [29]. Further complications arise from reports initially attributed to yield reduction by *H. avenae* that are now identified as *H. filipjevi*, *H. latipons*, *H. australis*, or *H. sturhani* [30].

The pathogens have a wide host range and can survive in the soil/organic residue for many years, therefore crop rotation plays a paramount role in reducing their damaging impact. Root rot symptoms are difficult to identify clearly but generally are characterized by discolouration of roots, coleoptiles and stem bases of the infected seedling. Root rot fungi also may attack the upper parts of plants which may result in foliage lesions, head and seedling blight (Table 9.2).

Take-all (*G. graminis*) is the dominant root disease favoured by the moist and cool conditions in winter season followed by the moisture stress during anthesis. Fungicide application and rotation with non-host crops are effective options to control the disease [28]. *Pythium* is a pathogen having a wide host range causing root rot and seedling damping off. *Pythium* infects root system via root tips and root hairs and can also penetrate the embryo of germinated seed, leading to symptoms like stunting and yellowing of leaf tissue. Infected roots are stunted, and light brown-yellow colouration is seen near the tips. *Rhizoctonia* can prune off the root and limit water and nutrient absorption which ultimately leads to crop damage. It survives in the top of the soil (0–10 cm) on organic matter [31]. *Fusarium* spp. especially *F. culmorum* and *F. pseudograminearum* cause root diseases, including foot rot, root rot, and crown rot. Crown rot encompasses symptoms on the lower part of the wheat plant, and diseased plants are characterized by fungal colonization on the wheat stems, crown and root tissues leading to a honey-brown discolouration of the leaf sheaths and lower stem, and necrosis of the crown region. *Bipolaris* spp. especially *B. sorokiniana* cause common root rot of wheat worldwide, which produces a brown to black discolouration of the subcrown internode.

Table 9.2 Basic characteristics of the root rot diseases

Disease/ causal agent	Causal agent	Symptoms	Hosts	Survival
Take-all (GGT)	*Gaeumannomyces graminis var. tritici*	Patches, blackening of roots, plant are easy to pull from the soil	Wheat, barley, rye, oat, grasses	Grass, stubble
Pythium root rot	*Pythium* spp.	Patches yellow to brown root system	Wheat, barley, triticale, oats, grasses	Resting spores
Rhizoctonia bare patch	*Rhizoctonia solani*	Stunting of plants, seedling rots, roots stunted with spear point	Wheat, barley, triticale, grasses	Plant residue, hyphal fragments
Crown Rot (CR)	*F. pseudograminearum, F. culmorum*	Scattered plants, browning of stem base, crown, white heads, pinched no grain, pink lower nodes	Wheat, barley, triticale, grasses	Volunteer grass, stubble residue
Common root rot (CRR)	*B. sorokiniana*	Patches Dark brown discolouration on subcrown internode	Cereals, grasses	Spores in soil, stubble residue
Cereal cyst nematode (CCN)	*H. avenae, H. filipjevi, H. latipons*	Patches, stunted yellow plants, multiple short, branched roots, cysts visible on roots in spring	Wheat, barley, oat, triticale, and grasses	Eggs, cysts
Root-lesion nematode (RLN)	*Pratylenchus* spp.	Patches, chlorosis of lower leaves, stunting, fewer tillers, and delayed plant growth	Wheat, grasses	Eggs, nematodes

Three major species belong to CCN, *viz. Heterodera avenae, H. latipons*, and *H. filipjevi*, and the first is the most widely distributed CCN around the globe. Wheat producing regions with temperate climatic conditions in Asia, Africa, North and South America, Europe and the Mediterranean are typically CCN occurrence zones [29]. The *Pratylenchus* species, especially *P. thornei*, *P. crenatus*, *P. neglectus* and *P. penetrans*, are widely distributed pathogens for RLN [32]. CCN is monocyclic as it completes only one cycle per season while RLN is polycyclic due to a higher multiplication rate of three to five generations per year. RLN causes stunted and poorly tillered plants. The badly damaged roots are thin and poorly branched with short and knotted laterals. Above ground CCN symptoms can be identified easily through patches and stunted plants. Below-ground symptoms are white females on roots (immature cyst) which can be seen with naked eyes in spring time (Fig. 9.1) [32].

Identifying which root rot pathogen is present in the field by classical and/or molecular tools is the most important point to tackle the disease (Table 9.2).

Managing these diseases in the modern farming system is a difficult task due to their hidden nature compared to leaf diseases. A variety of management strategies have been studied to control root rots [28]. Better understanding of the pathogen biology is the first step to apply the best management strategy for targeted root rot disease. Sowing healthy and high-quality seeds at the correct depth and sowing time with adequate levels of nitrogen are main agronomy practices. As these pathogens have a wide range of host crop, rotation with non-host crops may help to reduce inoculum level in the soil [31]. If there is a registered fungicide, its seed treatment may support stand establishment. 'Green bridge' must be broken off, since the volunteer plants or weeds helps the fungi/nematode to survive during offseason [28, 32].

Using resistant crops of high yielding potential combined with good agronomy is the most efficient and economical way to improve the productivity of the crop and manage root rot diseases, especially in dryland areas. Tolerant varieties are also effective in reducing the yield losses; however, they may conduce inoculum build-up/increase in the soil. Wheat and its wild relatives have been screened for resistance against SBPs, and several *Cre* genes (*Cre1* to *Cre9*, *CreX, CreY*) against CCN have been identified, which are reported to follow gene-for-gene hypothesis. International collaborative efforts, *viz.* distribution and utilization of CIMMYT's International root disease resistance nurseries in the respective national breeding programs, is important to achieve desired resistance in locally adapted wheat varieties [32]. Other current and future research will address the use of endophytic microorganisms and other cultural practices to the yield losses incurred by SBPs. There is currently insufficient breeding for resistance to SBPs due to a lack of expertise and recognition of SBPs as a factor limiting wheat production potential, inappropriate breeding strategies, slow screening processes, and increased research funding is required for a more holistic approach to plant health management [30]. In conclusion, nematologists, breeders and agronomists need to draw a good strategy and work together to find solution to the complex issues facing agricultural production and use multidisciplinary approaches to move forward in ensuring food security for all.

9.6 Key Concepts

Host resistance is widely acknowledged as an economic and environment-friendly approach to manage wheat diseases, for which quantitative resistance is preferred over qualitative resistance due to the long-term durability of the former. For diseases where host resistance is less effective, alternative management tools like fungicide application and cultural practices should be utilized to obtain a satisfactory disease control.

9.7 Conclusions

For all wheat diseases, varietal resistance is an indispensable component in disease management, because it is cost-effective, environmentally friendly, and compatible with other management strategies, which is especially valuable to resource-poor farmers in developing countries who often have no access to fungicides. For developed countries, the increasing demand on organic production and the stricter regulation on fungicide application also call for varietal resistance. Therefore, host resistance becomes the focus of CIMMYT's breeding work. Quantitative loci should be preferred over qualitative genes in breeding to prolong the life span of the released resistant varieties, and when disease pressure is high, other management tools especially fungicide and agronomic management (rotation, plant density and sowing time etc.) should be combined with varietal resistance to obtain a reasonable control of the diseases. Wheat relatives have made great contribution to resistance against various diseases mentioned in this chapter, e.g., the 2NS/2AS translocation for resistance to WB, *Fhb7* for FHB, *Stb16q* for STB, etc. More efforts are needed to exploit and identify novel resistance genes from such materials, and some additional relevant information is available in Chaps. 16, 17 and 18.

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