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Preface

While it is true that excellent progress has been made in developing new and widely adopted wheat cultivars, diseases still pose a big challenge for sustainable production of this cereal. The first book on wheat diseases in Brazil, entitled, "Doenças do Trigo e seu Controle," was published in Portuguese by Editora CERES, São Paulo, in 1978. Fifteen years later, the second updated edition of the book entitled, "Manejo Integrado de Enfermedades del Trigo," was published in Spanish, under the patronage of World Bank, by Imprenta Landivar, Santa Cruz de la Sierra, Bolivia, in 1993. The first book was well received especially in Brazil, whereas the second book had a wide readership in a number of Latin-American countries since it was written in Spanish. In a few years both the books were out of print. Later, after almost 20 years, several people from the wheat community expressed the need for a book in English with updated information including re-occurrence of old diseases and emergence of new diseases as well as new races of pathogens, and their impact on global wheat production.

By and large, the severity of some diseases caused by necrotrophic pathogens is directly related to change in the tillage system. In the modern era of precision agriculture, the conservation tillage system is fast expanding and demands adequate changes which are also addressed in this book.

The objective of this book was to offer necessary information on biotic and abiotic stresses that adversely affect the wheat production, descriptions of the most important diseases including necessary illustrations to help the reader the correct diagnosis of diseases and comprehend their epidemiological aspects. The book also deals with pillars of integrated disease management which would be eco-friendly and reduce severity of diseases and yield losses. It encompasses different tools of disease management and their implications especially for the tropical and subtropical areas of the world with acquired Latin-American experiences of over 40 years.

The book neither deals with descriptions and recommendations of different fungicides nor with the various wheat cultivars because they are considered outside the scope of the present objective. Nonetheless, it offers a comprehensive list of references for each chapter to enable the reader to look for specific details on a

given aspect including fungicides and wheat cultivars. It is hoped that this will serve as one of the reference books for students, young scientists, extension workers, and progressive farmers dealing with wheat and wheat production. Undoubtedly, it is a timely, and much needed publication considering the present world food crisis.

Londrina, Paraná, Brazil Y.R. Mehta

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Contents

Chapter 1
Wheat and Wheat Production Constraints

1.1 Wheat and Wheat Production Constraints

Wheat is staple food for the major part of the world's population. Approximately 630 million tons of wheat are produced annually, roughly half of it in developing countries (Peña 2007; Singh et al. 2011). It is especially important in India, in the USA, in Europe, and in the Latin and central-American countries including Argentina, Brazil, Bolivia, Chile, Mexico, Paraguay and Uruguay. Wheat production in the USA in 2011, was around 34.4 million tons harvested from 18.6 million hectares (Savary et al. 2012). Annual wheat production in the Latin American region, for example, used to be rather low compared to that in some technologically advanced countries and remained so, for some years (15 and 20×106 t). However, as in other countries, wheat productivity in this region has gradually increased during the past 20 years, reaching an average of over 2.0–3.5 t/ha, depending upon the country. This significant increase in wheat yield is mainly due to the introduction of high yielding cultivars and improvements in integrated disease management practices which are dealt with in the following chapters, followed by individual descriptions of some important wheat diseases that cause substantial yield losses in different wheat growing areas, of the world. Besides several diseases, the reoccurrence of scab, the emergence of an aggressive race of stem rust Ug99 and the spread of a relatively new disease—the Pyricularia blast, attacking cereals other than rice, are causing serious threats to wheat cultivation in much of the world (Vurro et al. 2010; Ralph et al. 2012).

1.1.1 Natural Limitations for Wheat Cultivation

Most wheat cultivation in Latin America is mechanized. In some cases soils are acidic with low pH and low fertility (<5.5), deficient in phosphorus and in some regions the level of exchangeable Al causes toxicity to the plant which is normally

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expressed by inhibition of root growth referred to as "Crestamento". In Al toxic soils lime application allied with the use of Al tolerant cultivars would be the best solution to overcome the problem (Hede et al. 2001). However, lime application in excess predisposes the plant to take-all disease caused by *Ophiobolus graminis*. The majority of Mexican wheats cultivated in the past were with high production potential but were sensitive to Al toxicity and to different fungal diseases as well. At present, most of the wheat cultivars are of local origin. In recent years, lot of progress has been achieved towards the development of Al resistant cultivars especially in Brazil. Matzenbacher (1988) reported that irrespective of the application of large quantities of lime the soil pH has not yet been corrected satisfactorily in the major wheat areas.

To solve the aluminum problem for example, the first efforts to incorporate aluminum tolerance of Brazilian wheats in Mexican wheats, with their wider adaptability, started in the early seventies with the collaboration of the Centro Internacional de Mejoramiento de Maiz y Trigo (CIMMYT), Mexico. As a result of this collaboration a number of semi-dwarf cultivars with high production potential and with aluminum tolerance were released (Rajaram et al. 1988a, b). In recent years, the national wheat breeding programs of Brazil released several aluminum tolerant cultivars with high production potential (EMBRAPA 2011). Similarly, much progress has been made during the past 20 years to minimize other problems like soil compaction, low soil fertility and soil erosion.

Wheat cultivation is subjected to a number of other limitations like diversity of climatic and soil conditions, pests and diseases. Unfavorable conditions for wheat cultivation include hail storms, heavy and prolonged rainfall, germination on spike, drought and frost. Over 100 diseases caused by biotic and abiotic stresses affect wheat in the USA and in other countries (Bockus et al. 2011; Savary et al. 2012).

1.1.2 The Wheat Plant

Wheat is a self-pollinated plant however, in some cases the cross-pollination may reach up to 2–3 %. The wheat species can be classified in three categories; *Triticum aestivum* L., *T. compactum* Host (club wheat) and *T. durum* Dest. (hard wheat). The first two species are hexaploids containing three genomes AABBDD ($n=21$), whereas the third species is tetraploid [*Triticum turgidum* (L.) Thell.] containing 14 chromosomes AABB ($n=14$). The three species together represent about 90 % of the cultivated wheat in the world (Wiese 1987; Gill et al. 1991; Blanco et al. 1998; Bockus et al. 2011).

First of all it is necessary to get acquainted with the wheat plant to understand the plant-pathogen interaction and the recent advances that have been made in genetical, molecular and chemical aspects. Since the botany of the wheat plant is described in several publications, it is considered dispensable here in this book. However, a description of some aspects of the anatomy of wheat seed and the growth cycle of the wheat plant are considered worthwhile (Fig. 1.1, Tables 1.1 and 1.2).

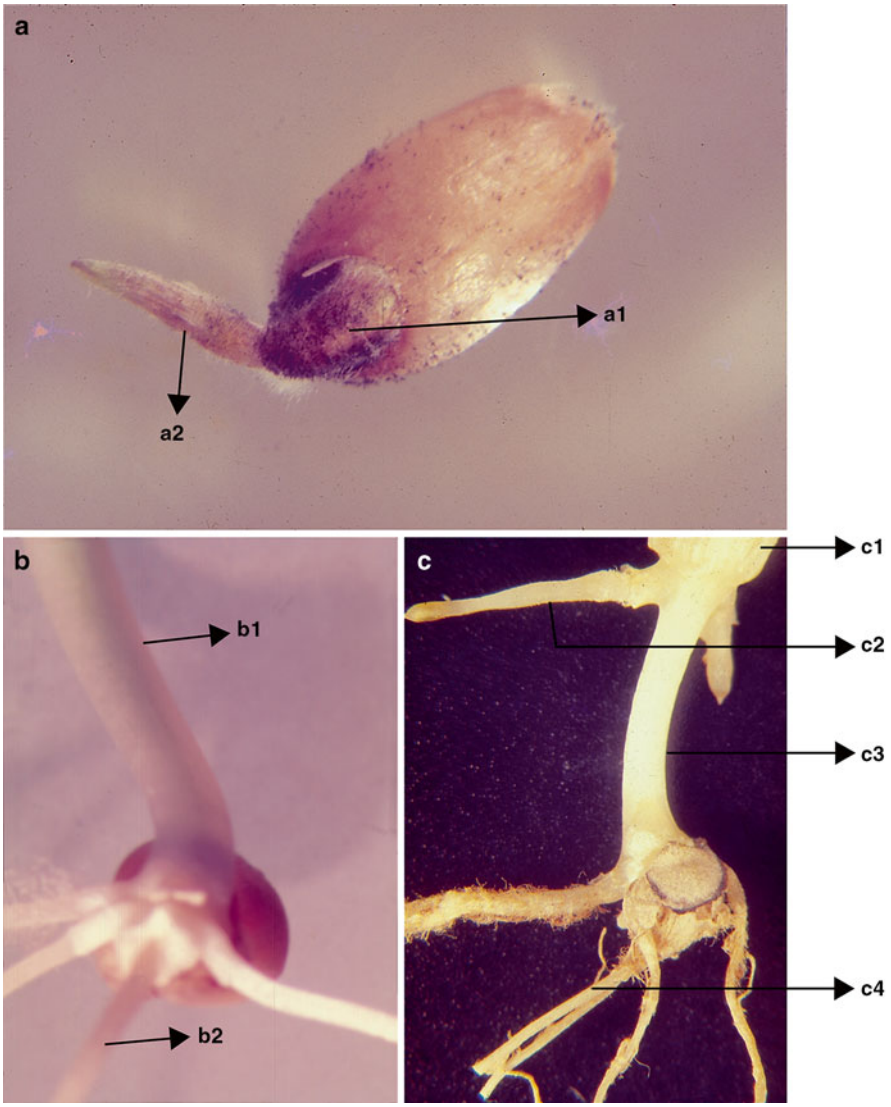

Fig. 1.1 Wheat seed germination. a1-plumule/coleoptile; a2-radicle; b1-coleoptile; b2-radicle; c1-stem; c2-crown roots; c3-coleoptile; c4-seminal roots

In order to make the reader familiar with a normal cereal plant, in the following pages the growth cycle as developed by Zadoks et al. (1974), is presented as; (a) Principal growth stages Table 1.1 and (b) Secondary growth stages Table 1.2.

Table 1.1 Decimal code for the growth stages of cereals (Zadoks et al. 1974)

1 Digit code	Description
0	Germination
1	Seedling growth
2	Tillering
3	Stem elongation
4	Booting
5	Inflorescence emergence
6	Anthesis
7	Milk development
8	Dough development
9	Ripening
T	Transplanting and recovery (rice only)

Principal growth stages

Table 1.2 A decimal code for the secondary growth stages (Zadoks et al. 1974)

2-Digit code	General description	Feekes' scale	Additional remarks on wheat, barley, rye, and oats
	Germination		
00	Dry seed		
01	Start of imbibition		
02	–		
03	Imbibitions complete		
04	–		
05	Radical emerged from caryopsis		
06	–		
07	Coleoptiles emerged from caryopsis		
08	–		
09	Leaf just at coleoptiles tip		
	Seedling growth		
10	First leaf through coleoptile	1	
11	First leaf unfolded*		Second leaf visible (<1 cm)
12	Two leaves unfolded		
13	Three leaves unfolded		
14	Four leaves unfolded		
15	Five leaves unfolded		50 % of laminae unfolded
16	Six leaves unfolded		
17	Seven leaves unfolded		
18	Eight leaves unfolded		
19	Nine or more leaves unfolded		

(continued)

Table 1.2 (continued)

2-Digit code	General description	Feekes' scale	Additional remarks on wheat, barley, rye, and oats
	Tillering		
20	Main shoot only		
21	Main shoot and 1 tiller	2	
22	Main shoot and 2 tillers		
23	Main shoot and 3 tillers	3	This section to be used to supplement record from other sections of the table: 'concurrent codes'
24	Main shoot and 4 tillers		
25	Main shoot and 5 tillers		
26	Main shoot and 6 tillers		
27	Main shoot and 7 tillers	3	This section to be used to supplement record from other sections of the table: 'concurrent codes'
28	Main shoot and 8 tillers		
29	Main shoot and 9 or more tillers		
	Stem elongation		
30	Pseudo stem erection	4–5	In rice: vegetative lag phase
31	First node detectable	6	
		7	Jointing stage
32	Second node detectable		
33	Third node detectable		
34	Fourth node detectable		Above-crown nodes
35	Fifth node detectable		
36	Sixth node detectable		
37	Flag leaf just visible	8	
38	–		
39	Flag leaf ligule/collar just visible	9	Pre-boot stage In rice: opposite auricle stage
40	–		
41	Flag leaf sheath extending		Little enlargement of the inflorescence, early boot stage
42	–		
43	Boots just visibly swollen		Mid-boot stage
44	–		
45	Boots swollen	10	Late-boot stage
46	–		
47	Flag leaf sheath opening		
48	–		
49	First awns visible		In awned forms only
	Inflorescence emergence		
50	First spikelet of inflorescence just visible	N 10-1	N = non-synchronous crops
51		S	S = synchronous crops
52	1/4 of inflorescence emerged	N	
53		S 10-2	

(continued)

Table 1.2 (continued)

2-Digit code	General description	Feekes' scale	Additional remarks on wheat, barley, rye, and oats
54		N	
55	1/2 of inflorescence emerged	S 10-3	
56	3/4 of inflorescence emerged	N 10-4	
57		S	
58	Emergence of inflorescence	N	
59	completed	S 10-5	
	Anthesis		
60	Beginning of anthesis	N	Not easily detectable in Barley.
61		S 10-51	In rice: usually immediately following heading
62	–		
63	–		
64			
65			
66	–		
67	–		
68	Anthesis complete	N	
69		S	
	Milk development		
70	–		
71	Caryopsis water ripe	10–54	
72	–		
73	Early milk		
74	–		
75	Medium milk	11-1	
76	–		Increase in solids of liquid endosperm notable when crushing the caryopsis between fingers
77	Late milk		
78	–		
79	–		
	Dough development		
80	–		
81	–		
82	–		
83	Early dough		Finger nail impression not held
84	–		
85	Soft dough	11-2	
86	–	11-2	Finger nail impression held, inflorescence
87	Hard dough		

(continued)

Table 1.2 (continued)

2-Digit code	General description	Feekes' scale	Additional remarks on wheat, barley, rye, and oats
88	–		Losing chlorophyl
89	–		
	Ripening		
90	–		
91	Caryopsis hard (difficult to divide by thumb-nail)	11-3	In rice: terminal spikelets In rice: 50 per cent of spikelets ripened
92	Caryopsis hard (can no longer be dented by thumb-nail)	11-4	In rice: over 90 % of spikelets ripened
93	Caryopsis loosening in daytime		
94	Over-ripe, straw dead and collapsing		
95	Seed dormant		
96	Viable seed giving 50 % germination		
97	Seed not dormant		
98	Secondary dormancy induced		
99	Secondary dormancy lost		
	Transplanting and recoery (rice only)		
T1	Uprooting of seedlings		
T2	–		
T3	Rooting		
T4	–		
T5	–		
T6	–		
T7	Recovery of shoot		
T8	–		
T9	Resumption of vegetative growth		

The proposal of this codified system of secondary growth stages is of special interest in epidemiological studies, herbicide, gametocyte and fungicide applications and assessment of yield losses. Besides, it has an additional advantage over the Feekes and Large scale since it takes into account the post anthesis growth stages which are not well defined by the Feekes and Large scale (Tables 1.1 and 1.2). The new decimal codification system of the growth stages has been used by most research scientists worldwide.

1.1.3 Types of Diseases

Although the aluminum toxicity problem has been solved to a great extent, there are other threats to wheat cultivation such as diseases caused by fungus, bacteria and virus. These diseases can be divided into: (a) diseases caused by viruses; (b) diseases caused by mycoplasmas; (c) diseases caused by bacteria; (d) diseases caused by fungi; (e) diseases caused by nematodes; and (f) nonparasitic diseases.

Basic information about the causal agents of these diseases is herewith described.

Diseases Caused by Bacteria

Plant pathogenic bacteria are prokaryotic, unicellular, rod shaped, most of them gram negative, motile via flagella and aerobic or facultative anaerobic. They are introduced in the plant via heavy rain-splash and insects, but always through wounds. They are capable of producing toxins and enzymes which decay plant tissues. Their size and shape vary but in general they could be up to 2 μm long (Bockus et al. 2011). Most of the bacteria are also seed transmissible. They can be seen under a common microscope not requiring an electronic microscope as viruses do. Identification of bacteria is done through biochemical properties and pathogenicity tests in different hosts, contrary to the fungal pathogens whose identification is based mostly on morphological characters. In recent years several bacteria have been classified through serological tests using monoclonal antibodies and by using biotechnological methods like RAPD, RFLP, rDNA, ERIC-REP PCR, microarray, sequencing, etc. The multiplication of bacteria is by asexual means and their movement in plants can be localized or systemic.

There are five different bacteria causing diseases in wheat. They are *Clavibacter*, *Bacillus*, *Erwinia*, *Pseudomonas* and *Xanthomonas*. Among these bacteria Pseudomonads and Xanthomonads are of major importance since they cause heavy yield losses in a number of important plant species. In general, the phytopathogenic Pseudomonads produce green fluorescent pigment in culture medium deficient in phosphorus, whereas in common culture media colonies of these bacteria are whitish, shiny and somewhat raised. Xanthomonads, on the other hand, constitute a group of bacteria which produce yellow colonies in common culture media. Both *Pseudomonas syringae* and *Xanthomonas campestris* (*axonopodis*) include different forms which are distinguished by their pathogenicity on some host species and are referred to as "pathovar". *X. translucens* pv. *undulosa* for example, attacks

wheat whereas *X. c*. pv. *hodei* attacks barley. Over eight bacterial diseases of wheat are reported: bacterial leaf stripe, also called black chaff (*X. t*. pv. *undulosa*), bacterial mosaic (*Clavibacter michiganensis* subsp. *michiganensis*, white blotch (*Bacillus megaterium* pv. *cerealis*), pink seed (*Erwinia rhapontici*), stem melanosis (*Pseudomonas cichorii*), bacterial sheath rot (*Pseudomonas fuscovaginae*), basal glume rot (*Pseudomonas syringae* pv. *atrofaciens* and bacterial leaf blight (*Pseudomonas syringae* pv. *syringae*), the first being the most important one worldwide (Mehta 1993; Mathur and Cunfer 1993; Maraite et al. 2007; Bockus et al. 2011).

Diseases Caused by Fungi

Fungal diseases are caused by biotrophic (obligate parasites which attack only the living plants) and necrotrophic fungi (facultative parasites which survive on dead tissues and do not necessarily need living plants). Numerous fungi are pathogenic to plants, animals and human beings. Fungal pathogens that attack plant species are grouped into four classes: phycomycetes, ascomycetes, basidiomycetes and imperfect fungi. In general, these fungi reproduce by spores which are resistant to ample temperature and humidity variation. Under adverse climatic conditions they can survive in the form of mycelia, sclerotia or clamydospores in the soil or in dead plant tissues. Most of the fungal pathogens are facultative parasites. Among different pathogens, the fungal pathogens represent the greatest threat worldwide. Depending upon the pathogen, the cultivar and the year, some of the fungal pathogens can cause 100 % yield losses.

Unlike viruses and bacteria, the fungi can be easily visualized through a simple microscope and in the majority of cases their presence can be verified by the naked eye. The causal organism of some of the common plant diseases can be identified based on symptoms whereas others need morphological and pathological characterization including their reaction on different host cultivars. A large majority of necrotrophic fungal pathogens can be easily cultivated on common artificial media.

The wheat fungal pathogens are widely distributed wherever the crop is grown and can attack different plant parts.

Diseases Caused by Mycoplasmas

Mycoplasma like organisms are prokaryotic and although they belong to one group of bacteria, they are smaller than the bacteria and lack a rigid cell wall. They are similar to pneumonia like organisms and can be cultivated in artificial culture media. As plant pathogens the mycoplasmas are responsible for diseases called “aster yellows”. Mycoplasmas are transmitted by insects and their movement in the plant is systemic. So far there has only been one mycoplasma disease of wheat and it has not yet reported from Latin America. Aster yellows are reported to attack monocotyledonous plants like wheat, barley, rye and oats, but over 300 dicotyledonous plants are also known to be hosts of this disease (Bockus et al. 2011).

Diseases Caused by Nematodes

Nematodes are roundworms and are found in soil and water. They are eel-shaped and some females are sac-like and they can be observed with an ordinary microscope and multiply by eggs. The nematodes penetrate the host, especially the roots, through their stylet. Diseases caused by nematodes can be important especially those caused by a complex of fungal and nematode or fungal and bacterial pathogen (*Rhizoctonia solani* + *Pratylenchus* spp., *Anguina tritici* + *Corynebacterium tritici*). To date, several nematodal wheat diseases have been reported among them Cereal Cyst Nematodes, Root Gall Nematodes and Root Knot Nematodes may be of some economic importance in specific wheat areas (Mehta 1993; Bockus et al. 2011).

Diseases Caused by Virus

The morphological form of plant viruses differs greatly. Wheat viruses, for example, are filamentous, spherical or bacilliform. The main virus vectors are aphids, whitefly, mites, leafhopper, nematodes, etc. They are also transmitted by seed, sap, grafting, fungi and even by mechanical means. At least two viruses; Barley Stripe Mosaic (BSM) and Wheat Chlorotic Streak Virus are known to be transmitted by seed (Phatak 1974). Today over 30 different virus diseases are known to occur in wheat and among them two stand out as of special economic importance. Barley Yellow Dwarf (BYD) is the most widely distributed disease worldwide and Wheat Soil-Borne Mosaic transmitted by the fungus *Polymyxa graminis* is especially important in the southern region of Brazil, although its occurrence is reported in other countries like Argentina, the USA, Egypt and Italy. Some of the virus diseases are dealt with in one of the following chapters of this book. For other virus diseases of minor economic importance the reader may refer to other publications (Zaitlin and Palukaitis 2000; Bockus et al. 2011; Jones et al. 2010).

Diseases caused by viruses were discovered towards the end of the nineteenth century and thereafter several virus diseases of different plants were described. Diseases caused by viruses affect the biological and physiological process of plants and can be responsible for reduction in production potential of plants. Among the plant pathogens, viruses occupy a special position. They are sub-microscopic agents and constitute the central part of the nucleic acid (RNA or DNA) surrounded by a protective protein coat. They cannot be multiplied in artificial culture medium. They lack self metabolism process and hence depend totally on the synthesizing system of the host for their multiplication. Thus, the viruses differ drastically from other causal organisms like fungi, bacteria, nematodes, etc. A high resolution electron microscope is an essential equipment to visualize the extremely small virus particles measured in nanometer (millimicron). One nanometer is equal to 1/1,000 of a micrometer (micron) which in turn is equal to 1/1,000 of a millimeter. Plant viruses vary between 20 and 1,000 nm with some exceptions at either end of the scale. The potato spindle virus having lower molecular weight has no protective

protein coat. Thus, a new term "virod" was proposed for this virus and for some other similar plant pathogens. For characterization of a virus, serological tests as well as some physio-chemical tests are deemed necessary along with electron microscopy to visualize the format of virus particles.

Nonparasitic Diseases

Nonparasitic diseases are not caused by any pathogen. They may be caused by several other factors like nutritional disorders, toxic effects of chemicals (like herbicides), very high or very low temperatures (like frost), insects, or nutritional imbalance (see Chap. 8).

1.1.4 Factors that Affect the Development of Diseases

Among the climatic conditions, the temperature and the atmospheric humidity play an important role. Almost all the plant pathogens are active under high humidity conditions provoked by continuous or intermittent rains for several days. Helminthosporium diseases can reach epidemic proportions under continuous rains and warm temperatures, whereas Septoria diseases require continuous rains but lower temperatures for several days. Wheat mildew caused by *Erysiphe graminis* (*Blumeria graminis*), on the other hand, can be severe under warm temperatures followed by humid and cloudy weather (Amuzescu 2009; Shaw and Osborne 2011; Pritchard 2011).

Predisposition of wheat plants to different diseases is another factor to be considered. In general, attack by one pathogen predisposes the plant to some other pathogen which may not necessarily be considered as a secondary pathogen. *Ophiobolus graminis* for example, predisposes the plant to *Septoria tritici* infection. In this case, *S. tritici* may be considered as a secondary pathogen since *O. graminis* causes almost 100 % damage to the plant. Nonetheless, epidemiologically speaking, *S. tritici* infection may become important since it adds to the amount of inoculum in the field. The same may be true when the plant is predisposed to *S. tritici* after it is infected by *Rhizoctonia solani*. While predisposition of a plant to another pathogen is known to occur, there is an antagonistic effect between *S. tritici* and leaf rusts. Some virus pathogens can also predispose the plants to infection by *S. tritici* (Sanderson 1964). Wheat powdery mildew as a predisposing factor was demonstrated several decades ago (Jonston 1934; Manners and Gandy 1954). Predisposition of wheat to *Septoria* after *Erysiphe graminis* infection was reported by Brokenshire (1974). Similarly, the susceptibility to *S. nodorum* was believed to be due to the infection of leaf rust (Van der Wal et al. 1970). Undoubtedly, this kind of predisposition drastically reduces the yield.

The principal factors that affect the development of wheat diseases are basically related to climatic conditions, wheat cultivars and virulence of the pathogen. All these factors are inter-related. If a cultivar is susceptible but a virulent pathogen

is absent then the disease will not occur or else will occur in a very low severity. If the cultivar is susceptible and a virulent pathogen is present but the weather conditions are not favorable for the disease in question, the disease will not occur. In contrast, if all three factors are present, the disease will occur. This is referred to as "the disease triangle".

Wheat cultivars can be classified into four categories: highly resistant; moderately resistant; moderately susceptible; and highly susceptible. Cultivars that are highly resistant or highly susceptible to all the pathogens do not exist. This is because resistance is a genetic character of a plant and also because different pathogens demand specific climatic conditions for their development (Van der Plank 1963).

1.1.5 Economic Importance of Diseases

Importance of disease is estimated through the yield loss it causes. At times, the importance of a particular disease is ignored or else underestimated because of the lack of accurate research data on assessment of losses (Nutter 1993; Vurro et al. 2010; Savary et al. 2012).

Losses caused by wheat diseases vary a lot from country to country. An interesting review on crop losses due to diseases and their implications for global food production losses and food security is presented by Savary et al (2012). These authors stated that direct yield losses of global agricultural productivity could be between 20 and 40 %. The indirect yield losses refer to the quality of product and may affect the human and animal health. Losses caused by wheat diseases in the USA, France and Nordic countries are summarized by McMuller et al (1997) and Savary et al (2012). Most of the losses in these countries are due to the fungal diseases like leaf blotch and Septoria nodorum blotch (Jain 2011; Savary et al. 2012). Some examples on yield losses caused by wheat diseases are given in the following pages, however losses caused by individual diseases are cited under their respective chapters.

Experiments to obtain quantitative data on loss assessment are difficult to conduct since they depend on several factors such as:

(a) the experiments have to be conducted in "Hot spot" locations for different diseases to avoid disease escape. Cultivars exhibit differential responses, some are highly susceptible to a particular pathogen while others are moderately susceptible and the data are valid only for the cultivar tested;
(b) success in experimentation also depends on the climatic conditions favorable to the disease. In case of unfavorable climatic conditions, artificial inoculations and repeated irrigations become necessary;
(c) in general, experiments on assessment of yield losses depend on the availability of specific fungicides, or in other words, fungicides which control all the diseases but not the one against which yield losses are being estimated. Normally, wheat is attacked by several diseases during different stages of its development. Selective and highly efficient fungicides to control leaf rust

and/or powdery mildew have become outdated and are no longer being commercialized;

(d) relatively new diseases like bacterial leaf stripe and Pyricularia blast are not controlled by the use of fungicides and available fungicides against Fusarium head blight (scab) are not highly effective;

(e) in addition, soil-borne root rot diseases cause appreciable yield losses and are invariably overlooked. Yield losses caused by common root rots of wheat may vary between 18 and 30 % (Diehl et al. 1983; Reis 1985). Mehta and Gaudêncio (1991) studied the relation between the severity of common root rot caused by *Bipolaris sorokiniana* and the wheat yield during four years and reported that for each 1 % increase in the root rot severity there was corresponding yield loss of 49 kg/ha;

(f) size of the experimental plot also plays an important role in the success of assessment of yield losses. For wheat, usually the plot size is 10–12 m^2 and for this size of field plots invariably some inter-plot interference is observed hence the losses are somewhat underestimated (Forster and Schaad 1988; Mehta and Bassoi 1993). To minimize this problem, distance between the plots should be increased. Although there are no definite rules for this, it is expected that the higher the distance between the plots the lower will be the inter-plot interference. Forster and Schaad (1988) reported inter-plot interference for the bacterial stripe of wheat even when the distance between the plots was 4.6 m;

(g) the experiments on yield losses should be continuous using newly released cultivars, since data on old cultivars may lose its importance;

(h) finally, assessment of yield losses provoked by a particular pathogen at different growth stages of wheat would be of special interest, especially to establish an appropriate fungicidal spraying schedule. However, such experiments are difficult to conduct and would demand dedication from a team of research workers.

Some information about yield losses is available in Brazil. Most of the information refers to losses caused by a complex of diseases but some information on losses caused by a specific pathogen is also reported. Yield losses caused by *B. sorokiniana* in a susceptible cultivar were estimated to be over 86 %. Such susceptible cultivars do not exist anymore, but a loss of over 86 % only demonstrates the potential of the pathogen (Mehta 1993). Mehta and Bassoi (1993), estimated yield losses of about 20–40 % depending upon the cultivar and the level of seed infection by *X. t.* pv. *undulosa*. In the case of leaf rust, it was possible to demonstrate that the earlier the disease epidemic starts the higher the yield losses will be (Fig. 1.2). There are a number of other reports about yield losses (Reis et al. 2000).

In Bolivia, yield losses caused by helminthosporium and leaf rust diseases, were reported to be between 38 % and 60 % respectively (Languidey and Barea 1993). In Brazil, yield losses of up to 100 % may be caused by Pyricularia blast (Kohli et al. 2011).

Irrespective of all the aforesaid problems, research data on yield losses provoked by a particular pathogen on a particular cultivar are very much needed. Information generated on yield losses would permit producers to adopt the appropriate control measures and determine the cost-benefit ratio of the existing measures.

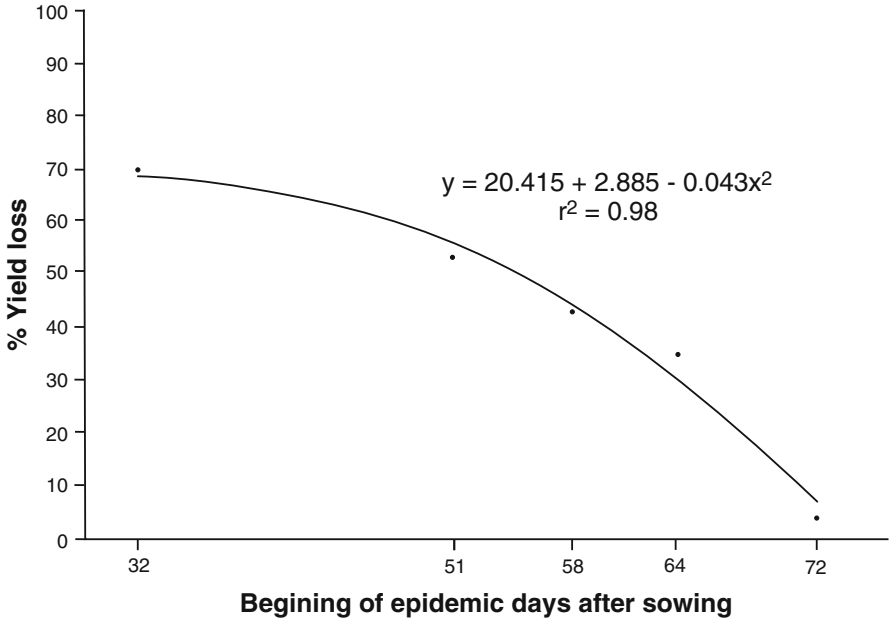

Fig. 1.2 Loss in yield in relation to the beginning of leaf rust epidemic in cv. Jupateco. Source: Mehta and Igarashi (1985)

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Chapter 2
Pillars of Integrated Disease Management

Economical and sustainable disease control can be obtained through the establishment of an integrated disease management system. The first integrated management system was developed by Dwight Isely to manage the population of cotton boll worm (*Anthonomus grandis*), which gave positive results for over 60 years in the United States (Newson 1980). Later, several other integrated management systems were developed and their basic concept was introduced to develop integrated management systems for diseases as well. Although there are several definitions of the integrated management system, according to Ledbetter et al. (1979) cited by Blair and Edwards (1980), "it is a system where all the possible pest control techniques are used to keep the pest population below the economic threshold. Each technique is eco-friendly and is compatible with the objectives of the user. Integrated management is more than merely the control of pests through chemicals. In several cases it includes the biological, cultural and sanitary control techniques for a complex of pests.

Thus the pillars of an integrated management system include several and all possible control measures and in case of wheat it should include cultivar resistance, seed health, cultural practices and fungicides (Mehta 1993; Cook 2000; Zambolim et al. 2001). As a rule, an integrated management system must always be eco-friendly. Some of these aspects are discussed in the following pages.

2.1 Genetic Resistance

Wheat cultivars developed after a long period of breeding work become vulnerable to new diseases or new races of a pathogen and thus lose all the investment made in creating new and high yielding cultivars (Van der Plank 1963). According to Van der Plank (1963), there are two kinds of resistance; one is referred to as vertical resistance (specific resistance) and the other as horizontal resistance (partial resistance). Vertical resistance is also known as complete resistance, specific resistance and monogenic resistance. The resistant cultivars can be classified into three groups: (a) specific resistance; (b) partial resistance and; (c) generalized resistance.

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2.1.1 Cultivars with Specific Resistance

Cultivars with specific resistance are those which show resistance to a few races of a pathogen but not to all. Breeding for specific resistance is simple and is inherited according to Mendel's law of inheritance. Biffen (1905), studied this for the first time and reported that for yellow rust of wheat the plants segregated in a ratio of 1:3 (one resistant plant and three susceptible plants). Since then numerous studies have been made and several resistant cultivars against a number of diseases were created. The resistance is considered specific when the cultivars are resistant to a single or few races. By and large, this kind of resistance is governed by a single dominant gene. When a resistant cultivar is crossed with a susceptible cultivar, in the F2 generation segregation of plants can be observed. If the resistance is controlled by a major gene the plants segregate in a ratio of 3:1 (three resistant and one susceptible) and when the resistance is recessive the plants segregate in a ratio of 1:3 (1 resistant and 3 susceptible).

To control diseases plant breeders and pathologists have been using major gene since it is simple and easy to select because of the clear difference between resistant and susceptible plants. However, cultivars with this kind of resistance last only for a short duration because the resistance is lost as soon as a new race of the pathogen capable of attacking the cultivar evolves in nature (Van der Plank 1963). It is for this reason that the sowing of a single cultivar in a large area should be avoided (also see chapter on disease control by cultural practices).

Virulence and aggressiveness are the two terms to express the parasitic ability of a pathogen to cause disease. In fact, virulence is the capacity of a pathogen (race or pathotype) to overcome the resistance gene of the host plant. Aggressiveness is the ability of a virulent pathogen to colonize the host and develop symptoms at a rapid pace. A virulent pathogen may be aggressive or not depending upon the environmental conditions, nonspecific resistance, latent period, etc.

2.1.2 Gene-for-Gene Theory

The inheritance of resistance and susceptibility in plants and the virulence and avirulence in parasites were studied for the first time by Flor (1947). After completing studies on linseed rust, this author presented the theory of gene-for-gene. Flor's theory of gene-for-gene implies that each gene that governs avirulence or virulence in a pathogen has a corresponding gene in the host that governs resistance or susceptibility. If one avirulent gene in the pathogen does not match with a resistance gene in the host no infection occurs. The disease occurs when the gene that governs avirulence in the pathogen matches with a corresponding gene in the host. If the host has no gene for resistance the disease will result irrespective of whether the pathogen has a gene for virulence or not. Similarly, the disease will also occur when one gene that governs virulence in the pathogen matches a corresponding gene

governing susceptibility in the host. This is an important aspect to be considered (Wilcoxson and Saari 1996; Vog et al. 2013). According to Elligboe (1976), the resistance and avirulence are normally dominant, whereas susceptibility and virulence are normally recessive.

The pairing of genes in other words is also called "compatibility" of genes. When the disease occurs it can be said that there is a compatible reaction between the gene of the pathogen and the gene of the host. It can also be said that the pathogen is avirulent and the host is resistant. In fact, compatibility and incompatibility are the specific reactions between the genes.

The gene-for-gene theory can be explained as follows. One gene of the host corresponds with a gene in the pathogen and makes a pair of genes. For each corresponding pair of genes, there exist at least two alleles in the host and two alleles in the pathogen.

There are four possible host-pathogen combinations for specific pair of genes. Interactions occur when two genes exist in the host R1 that governs the resistance and the r1 that governs susceptibility or when the complementary genes in the pathogen, called P1 which governs avirulence and the other p1 that governs virulence exist. The gene R1 shows an incompatible reaction with gene P1 but the gene p1 is compatible with both R1 and r1 genes. Specific recognition between the genes occurs in an incompatible reaction. Compatible reactions are the result of lack of recognition between genes (Elligboe 1976). The basic patterns of resistance about the host-pathogen interaction are explained in detail by several workers (Person 1959; Elligboe 1976; Mcintosh and Watson 1982; Loegering 1984; Roelfs 1988a, b; Roelfs et al. 1992; Wilcoxson and Saari 1996; Vog et al. 2013). Tosa (1989) has demonstrated that gene-for-gene relationship exists between forme specialis of *Erysiphae graminis* and genera of gramineous plants. Complete notion of the gene-for-gene theory is necessary to understand the specific host-pathogen interaction.

2.1.3 Fitoalexins × Specific Resistance

Resistance caused by rapid death of a host cell at the site of infection by a pathogen is normally referred as hypersensitive. Hypersensitivity may be considered as an essential component of specific resistance.

Hammerschmidt (1999) has given a comprehensive review about the research on phytoalexins.

While working on host-selective toxins produced by *Stagnospora nodorum* Friesen et al. (2009) reported that *S. nodorum* produces at least four proteinaceous host-selective toxins that interact with dominant host sensitivity/susceptibility gene products to induce Septoria nodorum blotch in seedlings.

The resistance of plants is governed by genes. In resistant cultivars the pathogen dies soon after penetration, due to extreme sensitivity of plant tissue, resulting in a hypersensitive reaction. This hypersensitive reaction is characterized as minute

specks of infection resulting in tissue necrosis (Dixon et al. 2002; Silva et al. 2010; Purwar et al. 2012).

Several hypotheses have been postulated for the mechanism involved in the hypersensitive reaction of resistant tissue (Hammerschmidt 1999; Fan and Doemer 2012; Jalali 1999; Muhovski 2012; Zang et al. 2013). It is believed that in some host-pathogen interactions the genes that govern resistance in the plant activate the formation and the concentration of some phenolic compounds in the lesions that are toxic to pathogens as well as to the infected cells. The concentration of phenolic compounds may depend on the degree of resistance of the host plant. In other words, this means that the production of phytoalexins depends on the resistance genes. If the genes are strong, the production of phytoalexin and its accumulation near the point of infection is high and hence the pathogen dies immediately. In some cases, no hypersensitive reaction is observed and the host is considered as immune.

Phenolic compounds (aromatic compounds) are produced in the plants via the Shikimic acid pathway or else by other biosynthetic procedures in innumerable host-pathogen interactions. Such phytoalexins could be specific or non-specific. A specific phytoalexin is produced only as a result of invasion by a specific pathogen whereas a phytoalexin is produced by mechanical damage is referred to as non-specific.

However, in recent years, the development of specific phytoalexins in the resistance process has continued to be a matter of controversy. While according to Elnanghy and Shaw (1966), resistant cultivars after infection produce higher concentrations of phytoalexins than susceptible ones, Seevers and Daly (1970) believes that there is no correlation between the concentration of phytoalexins in the tissues of resistant wheat cultivars and the susceptible cultivars for leaf rust. Several other examples regarding this subject are cited in the literature. It is possible that the concentration of phenolic compounds necessary to inhibit the growth of the pathogen could be very low and may not reach the detection limit by normal analytic procedures. More investigations are necessary regarding the relationship between resistance and the biosynthesis of aromatic compounds.

There is still some controversy about the primary gene product involved in the host-pathogen interactions. Although a vast amount of literature is available on the high rates of phytoalexin production in resistant cultivars, there is still no evidence to conclude whether this is the primary product of the gene or genes that govern a specific host-parasite reaction (Hammerschmidt 1999; Dangl and Jones 2001; Divon et al. 2002; Purwar et al. 2012; Jalali 1999). It is not yet very clear how resistance genes function to confer avirulence recognition. Clear understanding about resistance gene functions requires focus towards biochemistry and cell biology (Dangl and Jones 2001).

Different theories have been put forward by researchers on phytoalexins (VanEtten et al. 1989; Kaué 1996; Nicholson and Hammerschmidt 1992). One is that the plants are resistant because they can rapidly produce phytoalexins in sufficient quantities to check the progress of the pathogen. In the susceptible plants, it is possible that the pathogen grows rapidly because the plants do not produce phytoalexins or else produce them in insufficient quantities.

The other hypothesis is that the plants produce phytoalexins when they are invaded by pathogens. Only those pathogens which are capable of degrading the phytoalexins can normally multiply and provoke disease. Pathogens which cannot degrade the phytoalexins will be paralyzed and cannot produce disease. Elligboe (1976) introduced arguments for both the hypotheses. One of his arguments is that if the basis for the pathogen's restriction and development is its sensitivity to phytoalexins and if a mutation in the pathogen occurs which is not sensitive to such phytoalexins, then in this case the mutant can grow in the plant and cause disease. The second hypothesis is that if a mutant of a pathogen (normally capable of causing disease) incapable of degrading the phytoalexin is created then it will not be able to develop in the host and provoke disease. If both the arguments are correct, it would mean that the production of phytoalexin is not a pre-requisite for any host-parasite combination and that phytoalexins would be the secondary product of a gene governing any host-pathogen interaction. The example of mutants of a pathogen in studies of phytoalexins is a relatively new concept and could lead to important discoveries.

2.1.4 Use of Multilines

Multilines are lines that are agronomically similar to each other but differ genetically as regards their resistance to different races of a pathogen. Multilines may be referred as a different form of specific resistance. Each line has specific resistance to a particular pathogen and when several such lines are mixed together they form a "multiline". Due to their large diversity, the multilines have a special advantage over the specific resistance cultivars since they reduce the initial inoculum (Xo) as well as the rate of infection (r). Each line contributes to an additional genetic factor without phenotypic uniformity of the mixture. Multilines are created based on appropriate knowledge about the characters of agronomically compatible lines and genetically incompatible ones and are mixed in equal proportions. The use of specific resistance can be more advantageous when more resistance genes are introduced in a cultivar.

The advantages of the use of multilines were recognized in 1898, but investigations into multilines were intensified only in 1960 (Bourlaug 1953). The wheat breeding program of the Rockefeller Foundation in Mexico, released two multiline wheat cultivars in 1960. The first commercially used multiline wheat cultivars in Colombia were Miramar 63 and Miramar 65. Research on multilines was also started in India and in 1979 a multiline KSML 3 was released in the State of Punjab. In the same year a multiline cultivar called Tumult was released in Holland and another named Crew was released in the United States in 1982. Multiline cultivar of oats composed of 13 pure lines in two maturity classes were cultivated with success in more than 40,000 ha in the State of Iowa, USA (Browning 1988). However the development and utilization of multiline cultivars was not significantly successful mainly because of the time-consuming and expensive development process. Besides, the genetic diversity of multiline was very much reduced because of its pure line nature.

2.1.5 Cultivar Mixture

Because of their excessive uniformity, multilines lost their importance and a new concept of cultivar mixture was introduced. Within the Integrated Disease Management concept diseases can be kept under low intensity in cultivar mixture without the use of fungicides. Advantages of cultivar mixture in wheat, soybean, maize, rice, oats, beans, onion and gram (chickpea) were reported by several researchers. In East Germany for example, about 60 % of barley used for malt was cultivated through the cultivar mixture. The use of cultivar mixture to control diseases was studied by Wolfe (1988) for 11 years using 152 types of mixtures and in 122 types of mixtures an increase in yield of about 8 % was obtained. Later, Cowger and Mundt (2002), studied four mixtures of moderately resistant and susceptible winter wheat cultivars naturally infected with *Mycosphaerella graminicola* to investigate impacts on disease progress in the field. They reported that mixture yields were on average 2.4 and 6.2 % higher than mean component pure-stand yields in 1999 and 2000, respectively, but the differences were not statistically significant. Most of these studies were performed considering agronomical and pathological aspects (Faraji 2011). There is a concern among scientists that in a cultivar mixture natural selection of the pathogen with combined virulence may occur.

Another alternative strategy to the use of multilines and cultivar mixture is the pyramidation of resistant genes in an agronomically desirable cultivar. The more the major resistance genes are incorporated in a cultivar the more it becomes resistant to different races of the pathogen. Incorporation of major resistance genes is a relatively simple process and the cultivar with different resistance genes will be long lasting because its resistance will not be easily met by the creation of new races of the pathogen. This is a modern tendency in developing new disease resistant cultivars in several breeding programs.

2.1.6 Advantages and Disadvantages of Specific Resistance

Generally speaking, specific resistance is expressed by several terms like specific resistance, vertical resistance, monogenic resistance, hypersensitivity and unstable resistance, but the first is most used. This is the most interesting type of resistance as long as a new race of the pathogen capable of attacking the cultivar is not created in the nature. Normally, this kind of cultivar is short lived because a single change in the genetic constitution of the pathogen may be necessary to overcome the resistance and such changes are very common in nature (Van der Plank 1963). In recent years, because of the short lived nature of this resistance different types of resistance mechanisms have been sought.

2.1.7 Cultivars with Partial Resistance

Non-specific resistance, is also referred to by different terms, like horizontal resistance, field resistance, non-specific resistance, polygenic resistance, uniform resistance, stable resistance and partial resistance. Here again, the terms partial resistance and non-specific resistance are widely used. Partial resistance is effective against all the races of the pathogen and is governed by different genes. Contrary to specific resistance, partial resistance is of longer duration. The genes that govern this type of resistance are denominated as "minor genes" or non-specific genes and are not easily identifiable. Being polygenic in nature breeding for partial resistance becomes difficult. The difference between resistant and susceptible plants is not very clear and hence selection of plants is hampered or becomes doubtful.

Since partial resistance is governed by polygenes, it is less probable that a new race will appear in nature and be capable of matching all the genes of the host and breaking its resistance. In other words, partial resistance is difficult to be overcome by the evolution of new races of the pathogen. It is believed that, generally all cultivars have at least some quantity of partial resistance and the level of this resistance varies from cultivar to cultivar. It is not known how many genes are needed for the partial resistance to be highly effective and to satisfactorily control the diseases.

To accumulate partial resistance in a cultivar against a given pathogen, it becomes necessary to know different sources of the partial resistance. If there is no way to identify the genes that govern partial resistance, how can one be sure that different sources of partial resistance which show some level of partial resistance have the same or the different genes? If the genes are the same the breeder will be wasting his time and if the genes are different it will be possible to increase the level of resistance through breeding procedures. However, partial resistance can easily be lost during the traditional process of breeding. That being the case, it will be necessary to use different breeding procedures such as recurrent selection which is being used by several breeders (Singh et al. 2007).

For an effective selection for this kind of resistance the quantity of inoculum present in the field becomes very important. This is because partial resistance only reduces the rate of infection. If the selection for resistance is made in populations planted beside a highly susceptible cultivar, then the partial resistance may be ignored or else may be under estimated. Normally, the selection pressure in the experimental fields is very high and for this reason some breeding material with lower level of partial resistance may be lost. However, for the selection of high level of partial resistance even a high natural selection pressure is felt desirable (Mehta and Igarashi 1978). These are some of the aspects which make breeding for this kind of resistance rather difficult.

Partial resistance is preferred when the rate of infection of a disease is very high as is the case with biotrophic leaf rust and stem rust pathogens. Efficiency and economy in controlling the diseases will depend on the level of partial resistance of

a given cultivar. The higher the level of partial resistance the higher will be the efficiency and economy in controlling the disease through the use of fungicides.

Partial resistance is considered durable. On the other hand, resistance governed by specific genes can also be long lasting in some cultivars and hence long durability does not necessarily mean partial resistance. Partial resistance governed by non-specific polygenes could last for several years more than the resistance governed by a combination of specific genes. Durable resistance against stem rust of wheat for example, is conferred by a combination of specific genes like *Sr2*, *Sr23*, *Sr36*, whereas for leaf rust it is conferred by the combination of specific genes *Lr13* and *Lr34* (Roelfs 1988a, b; McDonald 2010).

Roelfs (1988a, b) and Singh et al. (2007), reported that some wheat cultivars having the gene *Sr2* in combination with other genes are being cultivated in North America to control stem rust without having been attacked by stem rust in the past 30 years. Similarly, examples of durable resistance for leaf rust are based on the utilization of a group of specific genes like *Lr12*, *Lr13* and *Lr34*. There exist other examples of durable resistance using a combination of specific genes. At times, this kind of resistance is referred to as "multigenic resistance" (Knott 1988; Parlevliet 1988).

Sometimes partial resistance is confused with "tolerance". The concept of the word "tolerance" is completely different from the concept of resistance. A cultivar tolerant to a particular disease is in fact susceptible and no resistance mechanism operates against the disease but it tolerates the infection and could perform well in the field.

2.1.8 Controversies About the Genes That Govern the Partial Resistance

The concept of partial resistance has created lot of interest among the pathologists and the plant breeders. As a result more and more reports on this issue have raised new ideas and concepts. Nelson (1971) believed that the genes which govern specific resistance or those which govern partial resistance are the same genes. According to this author, when more the specific genes are present in a cultivar more will be its chance to express partial resistance to which it has no genes for such kind of resistance.

While working on powdery mildew Ellingboe (1975) observed the phenomenon of "slow mildewing" in wheat cultivar "Genesse" either in the field or under controlled conditions. After inoculating the F2 plants derived from the cross between Genesse and a cultivar where the powdery mildew used to develop rapidly in the field, it was observed that if the plants were maintained in the glasshouse then segregation was continuous without showing highly resistant plants. Based on these results he concluded that the "slow mildewing" was governed by several genes. However, when the plants were maintained under controlled conditions he observed a segregation ratio of 3 slow mildewing plants and 1 fast mildewing plant and believed that this was due to a dominant gene for 'slow mildewing'. Later, he concluded that the genes that govern specific or partial resistance are the same genes.

Theoretically, there could be genes that do not follow the gene-for-gene theory. However in the experimental work conducted by different scientists to investigate the natural occurrence of variability, a gene-for-gene relationship always existed irrespective of the presence of specific major genes or the partial resistance genes (Elligboe 1976; Parlevliet and Zadoks 1977; Parlevliet 1981; Parlevliet and van Ommeren 1975; Gonzalez et al. 2012). Roelfs (1988a, b) and Tosa (1989) also reported that in the majority of cases there exists a gene-for-gene relationship. This evidence does not support the concept of Van der Plank's (1963) view of partial resistance where he believed that gene-for-gene relationship exists only in case of specific resistance.

Different genes that govern specific resistance show intermediate effects and produce results similar to partial resistance (polygenic or non-specific). Parlevliet (1985) reported that the partial resistance genes also follow the gene-for-gene relationship. Considering these two aspects, Knott (1988), raised doubts about whether there exist difference in resistance mechanism or physiological difference between the two types of resistance. However, this author believes that the basic difference between these types of resistances is that major genes of specific resistance acts independently from one another, whereas the polygene of partial resistance act additively.

Strictly speaking, with the exceptions of leaf rust and yellow rust of wheat, substantial success in wheat breeding for partial resistance has not been achieved so far. Some success has been achieved in accumulating partial resistance against leaf rust and powdery mildew in barley by the recurrent selection method of breeding.

It may still take some time before the existence of a gene-for-gene relationship in partial resistance becomes conclusive and throws more light on the revolutionary idea that the same genes govern both types of resistance.

2.1.9 Cultivars with Generalized Resistance

When dealing with partial resistance one should specify the pathogen in question. While partial resistance is effective against all the races of a single pathogen, the cultivars with generalized resistance offer partial resistance against all the pathogens and their respective races. This may be considered as a modified form of partial resistance. One practical method for this kind of resistance was suggested by Robinson (1976) and it includes polycrossings between different cultivars susceptible to a specific race of each pathogen against which partial resistance is desired. This author suggested that polycrossing and high selection pressure should be exerted continuously for 6–8 generations until a uniform cultivar with a satisfactory level of partial resistance is achieved against different pathogens and their races. In this method complete selection pressure can be exerted only in the absence of specific resistance. When specific resistance is operating the partial resistance cannot be easily identified in the segregating populations. So far, success in this methodology has not been obtained.

2.1.10 *Production of Dihaploid Through Wheat × Maize Hybrids*

Breeding efforts to transfer resistance in desirable high yielding cultivars has been a high priority in recent years especially for *Gibberella zeae*, *B. sorokiniana, Pyricularia grisea, Pyrenophora tritici-epentis and X. t. pv. undulosa,* using the available sources of resistance. Since resistance to these pathogens is not complete, success in breeding is not very encouraging. The production of wheat haploids via chromosome elimination is one of the latest and most useful techniques in gene transfer experiments (Laurie and Bennet 1988; Riera-Lizarazu and Mujeeb-Kazi 1990; Riera-Lizarazu et al. 1992; Zang et al. 1996). Production of haploid wheat plants and subsequent production of dihaploids (double haploids) can fix characters in a single generation. The procedure using wheat × maize hybrids allows complete homozygosity within one or two generations and facilitates the somaclonal variant selection process.

Somaclonal variation exists and has been proven to be genetically inherited (Vasil and Vasil 1986). Although several transformation techniques have been developed, generally speaking such techniques are highly sophisticated and depend on availability of an efficient and reproducible tissue culture system (Jahne et al. 1994). Somaclonal variation, on the other hand, is a relatively simple technique, usable where other methods are not feasible or where resistance genes of interest are not available.

Gametoclonal variation is known to occur in doubled haploids (Rode et al. 1987; Gallais 1988; Bjornstand et al. 1993; Bakshi et al. 2012; Kelm et al. 2012; Christiane et al. 2012). Kelm et al. (2012) studied inheritance of seedling resistance to seven worldwide isolates of *Mycosphaerella graminicola* in a doubled-haploid population. Multiple quantitative trait loci mapping revealed major and minor genetic effects on resistance. These authors suggested a complex inheritance of resistance to Septoria tritici blotch in the seedling stage in terms of isolate-specificity and resistance mechanisms.

According to Zang et al. (1996), haploid embryo production frequency and plant regeneration are affected significantly by maize genotypes but not by wheat genotypes.

Mehta and Angra (2000) reported that it was possible to produce hybrid embryos and haploids in six wheat cultivars, thereby indicating that wheat genotype did not affect haploid plant production. According to these authors hybrid embryo production varied between 0 and 25% and the chromosomes had a constant number as observed in their original hexaploid wheat genotypes ($2n = 6x = 42$), whereas the haploid plants had $n = 21$. Fig. 2.1 shows wheat caryopsis with embryo formation through wheat × maize hybridization (Mehta and Angra 2000).

Further research in this area might lead to the creation and release of new cultivars with a desirable level of resistance especially against the necrotrophic plant pathogens.

Seeding resistance to Septoria tritici blotch in the winter wheat doubled-haploid population (Solitar × Mzurka) was studied by Kelm et al. (2012). According to these

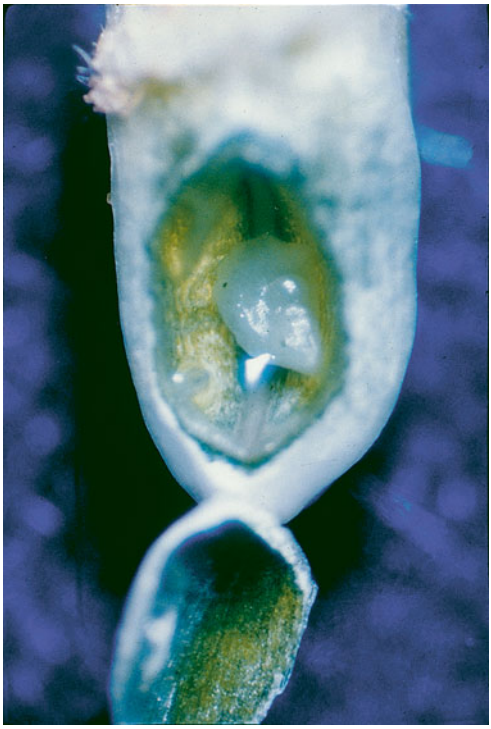

Fig. 2.1 Wheat caryposis with embryo formation through wheat × maize hybridization. Source: Mehta and Angra (2002)

authors, multiple quantitative trait locus (QTL) mapping revealed major and minor effects on resistance as well as several epistatic relationships in the seedling stage. The results suggest a complex inheritance of resistance to STB in the seedling stage in terms of isolate-specificity and resistance mechanism.

2.1.11 General Considerations

Breeding for resistance to some necrotrophic pathogens is still not adequate. As a rule, in breeding for disease resistance, it is necessary to have ample genetic variability within the host populations, as well as within the pathogen populations. To exploit existing genetic variability in the host plant, it is important to determine the genetic variability of the pathogen populations. For this purpose, different virulent strains of the pathogen must be identified. In this case, establishment of a differential set of cultivars may be very helpful. Screening for resistance is an important step in breeding for resistance. Screening techniques must be reliable, so that the resistant material thus selected can be incorporated into the crossing blocks with confidence.

Screening for resistance is generally done in the glasshouse or in a walk-in cold chamber, but always under controlled conditions. All plant material must be tested under standard and uniform conditions. Any change in the quality of inoculum, inoculation technique, incubation period and environmental conditions may alter the reaction pattern.

For screening purposes, a good inoculum must include a mixture of several virulent isolates and an appropriate and constant amount of conidia in the suspension. It is also preferable that plant material be tested in the glasshouse for resistance at two growth stages, at seedling stage and also at adult plant stage. It has been shown that seedling reaction does not necessarily correspond to adult plant reaction. Although adult plant resistance is always preferred, tests done also on seedlings stage help to determine lines that show resistant reaction at both growth stages. Such lines are of great interest in breeding programs for resistance.

Although glasshouse tests are reliable, it is often necessary to conduct field trials to confirm cultivar reaction under natural conditions. It is important that the tests be performed at three to four "hot-spot locations" each year. Resistant and susceptible checks should be included and the trial be surrounded by a susceptible spreader also inoculated with a mixture of isolates varying in virulence. Disease ratings should be taken at different stages of crop development. For the disease progress curves the rate of infection can be calculated: the lower the rate of infection, the higher the degree of resistance. Agronomically desirable lines with low infection rates are used as sources of resistance in the breeding programs.

There is a need to identify resistant sources in alien species like *Aegilops squarrosa, Agropyron curvifolium, Elymus curvifolius, Hordeum chilense, Triticum tauschii* and *Thinopyrum curvifolium*. These species may offer genes of major interest. Transfer of resistance from such species to *T. aestivum* is a rather complicated and difficult task. Difficulties in making interspecific crosses are mainly due to differences in levels of ploidy. However, problems such as lack of chromosome pairing and crossing over, failure of crosses after fertilization, difficulties in rearing hybrid plants and the lack of vigor and fertility of hybrid plants can be overcome by the use of various techniques and chemicals such as colchicine and gibberellin.

Finally, it is evident that for the Integrated Disease Management system, cultivar resistance plays an important role. As far as rusts are concerned, spectacular achievements have been obtained in the creation of new and resistant wheat cultivars. In Brazil, for example, the majority of wheat cultivars until the 1980's were susceptible to two rusts, because most of the cultivars were introduced from other States or from other countries. Today, most of the wheat cultivars are of Brazilian origin, although some have CIMMYT germplasm when used as parent. These cultivars have wide adaptability and disease resistance. It is believed that these cultivars possess genes for specific resistance as well as for partial resistance. (*Lr13, Sr2*, etc.). Rajaram et al. (1988) reported that during the last several years semi-dwarf cultivars occupied over 50 million hectares in the world without having reported any severe leaf or stem rust epidemics. The modern tendency is to develop new wheat cultivars with a combination of specific and non-specific resistance genes against major diseases.

2.2 Fungicides and Their Application in the Field

Within the Integrated Management concept use of fungicides also play an important role, however, they are applied only when their use becomes necessary. Although the cultivars have specific and non-specific genes against leaf rust, they are not protected against all the races and hence one or two applications of some specific fungicides become necessary. Considering different problems involved in fungicidal applications like development of new mutants of a pathogen resistant to fungicide or to a group of fungicides, their toxic and residual effect, fungicides are being used rationally.

Besides, some reduction in the use of fungicides is recommended in some countries. In Sweden for example, the use of agricultural chemicals was reduced by over 50 %, whereas Denmark established a limit of 50 % in the use of agro-chemicals till the year 1997. Similarly, Great Britain experienced a reduction of 41 % in the use of active ingredients. Considering the complexity of the problem, Germany decided to reduce the use of agricultural chemicals and adopt Integrated Disease and Pest Management practices (Warrel 1990).

In Latin America including Brazil, there is no fixed limit for the use of agro-chemicals. However, substantial emphasis is being placed on the rational use of these chemicals as well as development of Integrated Disease and Pest Management systems by ANDEF. Through the development of different Disease Management Systems rational and effective use of fungicides can be obtained, as evidenced by several publications (Mehta 1978; Reis 1985, 1987). The generalized fungicidal use for wheat in Brazil, between 1974 and 1980, was 2–3 applications during the crop cycle, which after 1984 was reduced to 0–2 applications (Mehta et al. 1992; Mehta 1993). Thus it is estimated that due to the rational use of fungicides, Brazil has been economizing around 50 million dollars annually.

Reduction in the use of fungicides in the Rio Grande do Sul, Brazil, for example, is in part because of the modeling systems to predict the severity of wheat diseases, especially the wheat scab (Fernandez et al. 1993; Fernandes and Picinnini 1999; Vargas et al. 2000; Fernandes and Pavan 2002; Fernandes et al. 2004, 2005; Del Ponte et al. 2009; Pavan et al. 2011). In Brazil, efforts have been made during the last 30 years in developing mathematical models for predicting severity of several diseases with the aim of achieving rational and effective use of fungicides. None-the-less, during the past few years Brazil has been using increasing amounts of fungicides to combat newly emerging diseases like soybean rust, Ramularia leaf blight of cotton, among others.

2.2.1 Selection of Fungicides

Different types of fungicides exist for aerial application in wheat. These fungicides can be protectants or erradicants. The former control the infection but cannot eradicate it once it has established itself in the plant. For this reason the protectant fungicides are applied before the onset of the infection. On the other hand, eradicant fungicides are those which eliminate the fungus after it has caused infection and

thus cure the plant. Most systemic fungicides are classified in this group. For loose smut of wheat for example, carboxin + thiram is considered an eradicant fungicide. New fungicides whether specific or non-specific are constantly emerging in the market. For basic information about fungicides in general, the reader may refer to some specific publications.

While a wealth of information is available about the use of different fungicides under different epidemiological conditions, a lot still needs to be done regionally. Each region poses a different and specific problem demanding an independent approach. While wheat rust can be controlled using the existing fungicides, wheat scab and Pyricularia blast for example, are not satisfactorily controlled by using the existing fungicides and the technology of their application, whereas wheat leaf rust can be controlled through the existing fungicides.

Experiments on fungicides are somewhat difficult to conduct. A global knowledge about the development of an epidemic is essential to study the efficiency of fungicides. Before a fungicide is recommended several aspects are taken into consideration like: (a) Adequate dose of the fungicide, (b) Methods of application, (c) Appropriate spraying schedule, (d) Final effect on the yield and the quality of product; (e) Economy in use of certain types of fungicides; (f) Residual effect on the plant; and finally, toxic effects on plants, human beings and animals.

Normally fungicides have been evaluated based on the gain in yield (Mehta 1978). Thus, if the efficiency of fungicides is only based on the yield data then all the fungicides which increase the yield should be recommended, irrespective of whether they controlled the disease satisfactorily or not. Fungicidal evaluation can also be based on the economy involved in the operation. However, the economical aspects involved in the use of fungicides are somewhat complicated since they also depend on the resistance level of the cultivar (fungicides may be economical for a specific cultivar but not for another), fungicidal dose and finally the cost of the fungicide which varies from year to year.

Considering these aspects Mehta et al. (1978) established a criterion for the evaluation and selection of fungicides against foliar diseases of wheat. According to this criterion all fungicides which maintained the level of disease below 50 % of the leaf area infected at growth stage 83 (Zadoks et al. 1974), were selected and recommended. Growth stage 83 was used as a reference because after this stage of development the leaves, especially the flag leaf and the flag leaf-1 do not contribute to the formation of grain and hence fungicide use becomes unnecessary. According to these authors, all the fungicides classified using this criterion, without exception, were superior to the check plots in yield as well. However, further research is needed to establish appropriate criteria for the new moderately resistant cultivars.

2.2.2 Fungicide Spraying Schedule

An appropriate fungicide spraying schedule plays an important role in Integrated Management Systems. The spraying schedule includes the time of first application, interval between the applications, the number of applications and the time of last application, considering always the cultivar and the disease in question.

Under the Latin-American conditions the first application is of the utmost importance. Fungicides should be applied starting from the first appearance of the disease symptoms. The first symptoms of the disease especially spot blotch and leaf rust are observed 40–45 days after sowing in early maturing cultivars and 50–55 days after for the late maturing cultivars. Thus the first application can be made after 45–55 days after sowing depending upon the type of cultivar. However, if the first disease symptoms appear 70 days after sowing, for example, the first application should be made then and not before. With some exceptions, fungicidal application before the onset of the disease is not advisable.

It is well known that the earlier the disease epidemic starts the higher will be the loss in yield. Fixing a foliar disease level of 5–10 % of the leaf area infected for the first application, for example, is difficult and risky. If the farmer has to wait until the level of infection reaches this level and if it is followed by a prolonged rainy period, the disease will proliferate rapidly and the farmer will have to wait for several days before he can enter the field for application with tractor. Besides, it must be remembered that field applications with tractor are time consuming and the rate of infection of the majority of diseases is very high. If the disease is not controlled at the beginning the control may then be inadequate or even uneconomical. Thus the tolerance limit for most of the foliar diseases could be traces of infection for the first fungicidal application. The objective is not to control totally the disease but to retard the start of the epidemic by 30–40 days, or else reduce the rate of infection so that the disease level does not reach over 50 % of the leaf area infected at soft dough stage 83 (Zadoks et al. 1974; Mehta et al. 1978).

The rate of infection of different pathogens could be very high and would depend on the spore production potential of each pathogen. Leaf rust and Heminthosporium for example, are considered diseases of high infection rate. Table 2.1 shows comparison between the spore production potential of the pathogens of these diseases. *Puccinia triticina* for example, needs 8 days of incubation period, whereas *Bipolaris sorokiniana* needs only 2 days. Irrespective of the period of infection in both the cases the sporulation starts 11 days after the incubation. The delay in 6 days in the incubation period of *P. triticina* is compensated by double the number of spores produced per day per lesion. However, *P. triticina* loses 1–2 days in relation to *B. sorokiniana* as regards the maximum duration of spore production. Once again, this loss is compensated by a superior period of double the amount of sporulation

Table 2.1 Spore production potential of *Puccinia triticina* (PT) and *Bipolaris sorokiniana* (BS), in susceptible wheat cultivars under controlled conditions

Pathogen	PT	BS
Period of incubation	8 days	48 h
Beginning of sporulation days after inoculation	11	11
Maximum No. of spores produced per day/lesion	767	487
Maximum duration of spore production	1 day	2–3 days
Maximum period of sporulation	72 days	30 days
Relation between weight of spores and the weight of the spore producing leaf or % leaf area infected by a single lesion	01:01	22.70 %

per lesion, lasting for 72 days and 30 days in the case of *P. triticina* and *B. sorokiniana,* respectively (Mehta and Zadoks 1971; Mehta 1981).

Finally, as a result of high spore production potential the dry weight of total spore produced during 72 days by *P. triticina* equals the dry weight of the leaf that produced the spores. On the other hand, a single lesion caused by *B. sorokiniana* could reach up to 22.7 % of the leaf area. Thus, considering the compensation in spore production potential of the two pathogens using different parameters, it can be concluded that both pathogens are very aggressive and so special care must be taken towards the management strategies in controlling these two diseases.

Success in fungicidal applications also depends on the interval between the applications. Systemic fungicides for foliar diseases offer 20–22 day of protection whereas the non-systemic fungicides (protectant fungicides) offer a protection of only 12–15 days. When the climatic conditions are favorable for diseases the fungicidal applications can be repeated considering the interval between the applications and the type of fungicide. However, if the climatic conditions are not favorable for the disease as happens with prolonged periods of drought, then the rate of disease multiplication is drastically reduced and hence the interval between the applications may be increased. In any case, constant field monitoring for the disease spread is deemed necessary.

The number of application for foliar diseases cannot be pre-established and is variable depending on the cultivar, fungicide and weather conditions. It also depends on the time of appearance of the first symptoms of the disease before which fungicides may not be applied. In some years the first symptoms of the disease may appear after 60–70 days after sowing necessitating only one application, of a systemic fungicide. Similarly, if the first disease symptoms appear only after the growth stage 83 for example, then in this case fungicidal application may not be necessary. Irrespective of the number of applications the last application may be performed preferably up to the time of flowering but not after the milk-stage. A susceptible cultivar may need more applications than a less susceptible or moderately resistant cultivar.

Generally speaking, non-systemic fungicides are applied more number of times than the systemic fungicides, during the crop cycle especially when the weather conditions are favorable for the disease. The number of applications also depends on the system of cultivation. In no-tillage cultivation, for example, the first disease symptoms of tan spot are observed as early as 20–25 days after sowing. If crop rotation is not followed, the number of applications will no doubt be more than for the conventional system of cultivation.

By and large, wheat is attacked by a number of diseases. As stated earlier, cultivars that are moderately or highly resistant to all the diseases do not exist. Cultivars differ in their degree of resistance and susceptibility to a particular disease or a group of diseases and hence different fungicidal schemes need to be considered for each group of cultivars in a particular area. For moderately susceptible or moderately resistant cultivars, only some reduction in the rate of infection is necessary. On the other hand, for susceptible or highly susceptible cultivars, it will be necessary to delay the start of a disease epidemic by 30–40 days. Delay in the initiation of epidemic can be obtained by systemic fungicides, whereas reduction in the rate of infection may be obtained by non-systemic fungicides.

2.2.3 Management of Systemic Fungicides

Resistance of pathogens to some fungicides was discovered at the beginning of the 1940s. One of the examples of this kind of resistance is experienced by Japanese farmers in controlling rice blast caused by *Pyricularia grisea*. In the 1960's antibiotics were introduced to control rice blast replacing organomercurial compounds. In the year 1971, a few years after the introduction of the antibiotic kasugamycin some resistant biotypes of *P. oryzae* were identified especially in some districts where kasugamycin was extensively and intensively used. In the following year 97 % of the *P. oryzae* isolates were resistant to kasugamycin in those districts. The application of kasugamycin was stopped and as a result the resistance level was reduced to 20 % within 3 years (CERES 1982).

Boukef et al. (2012) studied frequency of mutations associated with fungicide resistance and population structure of *Mycosphaerella graminicola* in Tunisia. They reported that few mutations associated with fungicide resistance were detected. They further reported that no evidence for strobilurin resistance was found among 357 Tunisian isolates and only two among 80 sequenced isolates carried mutations associated with azole resistance.

There are several examples of resistance of some pathogens to fungicides of the group benzimidazol. Other examples of resistance like powdery mildew fungus of Cucurbitacea to dimetirimol, of *Alternaria kikuchina t*o polioxina and *Erwinia amylovora* to streptomycin are wellknown. The history of resistance to fungicides was well covered by Delp (1980), who used a theoretical (mathematical) model to understand the development of resistance to benomyl considering leaf spot disease of the perennial crop caused by *Cercospora* sp. According to this model, the problem of resistance was drastically reduced when benomyl was used in combination with maneb from the beginning of its utilization (Heitefuss 2012).

Considering these examples, it is evident that the use of systemic fungicides should always be based on some criteria. However, irrespective of the criteria used, constant monitoring of the biotypes of the pathogen is indispensable so that the resistant biotypes be identified as soon as they emerge. Investigations in this area are encouraged by Fungicide Resistance Assessment Committee (FRAC) and intensified because the range of chemical groups is very much narrow and may favor the emergence of resistant biotypes. In addition to this, there also exists the problem of cross resistance where a biotype resistant to one fungicide is also resistant to another fungicide of the same group.

The dose of the fungicide should not be altered during the crop cycle. Considering economical aspects sometimes a farmer uses half of the recommended dose of the fungicide to control the powdery mildew at its initial stage and later when other foliar diseases start appearing, he uses the same fungicide but with a normal or even higher dose, because the lower dose will not control other foliar diseases. This is a very dangerous practice since it may provoke the pathogen to adopt the fungicide and create resistant biotypes. The problem of resistance can be minimized by using a mixture of systemic fungicides with non-systemic fungicides.

2.2.4 Fungicide Application Techniques

The efficiency of a fungicide in controlling a disease will depend on the application method. Normally, a good coverage of the plant is important and can be obtained by high or low volume sprays. High volume sprays using 200 l of water per hectare are applied by tractor. In this case the equipment should be well regulated in order to avoid the run-off of the fungicide. It should however be remembered that the better the coverage of the plant with fungicide the better will be the efficiency in controlling the disease. Tractor spraying causes mechanical damage to the crop, but it is compensated by the gain in yield obtained by the disease control (Boller et al. 2007, 2008; Cunha et al. 2011; Tormen et al. 2012).

While the low volume (30–40 L ha^{-1}) aerial applications overcome the problem of mechanical damage to the crop, they pose different problems. Good aerial application depends on the velocity of the wind, experience of the pilot and the height of flying. The smaller the spray particles the higher will be their dispersion and evaporation and consequently the higher their deposition on the plant. Normally speaking, an average of particle size in aerial applications of 200 μm is considered optimum, since particle size smaller than this does not give a good coverage of the plant.

Another type of equipment for aerial applications is called "micronair" and is being used with success. This kind of equipments has a special advantage over the conventional hydraulic equipments, in which the size of the spray particles can be easily adjusted. However they are more expensive than the conventional ones. For aerial applications cross winds of about 10 km ha^{-1} are considered ideal. Presence of free water including dew formation on the plants is not considered prejudicial. On the contrary, it helps in the distribution of the fungicide because in the aerial applications the size of the spray particles is rather small. On the other hand, with tractor application (high volume) the size of the spray particles is big and in the presence of free water may cause run-off of the spraying product. In such a case fungicidal applications during the early hours of the day may be avoided. In both kinds of application the quantity (dose) of the fungicide should remain the same. The dose of the fungicide should be calculated per unit of area and not by volume of the water to be used either for tractor or for aerial application.

2.3 Disease Forecast Modeling

Besides the above mentioned aspects, in recent years emphasis has been given to the disease forecasting computer models. Through such models it is possible to provide estimates of disease likelihood and forecast outbreaks which in turn avoid unnecessary fungicidal applications. They give guidelines for timely applications and consequently make the control measures more cost effective. Computer modeling when validated, should necessarily become a part of the integrated disease management systems.

Traditional plant disease climatological models have used accumulated hours of wetness duration combined with temperature requirements to predict the infection process and identify times of high disease risk. These types of models use recorded weather data to track enough favorable disease hours to warrant management action. In Brazil, the revolutionary web-based technologies have been studied for the past few years; these are known as SISALERT (Vargas et al. 2000; Fernandes et al. 2007, 2011; Ponte et al. 2005; Pavan et al. 2011). A simulation model developed for Fusarium head blight (FHB) has a component for the disease cycle and component for growth and development of wheat spikes. According to this model, a successful infection on a particular day depends on host tissue susceptibility factor, inoculum density, temperature, daily precipitation and mean relative humidity in a 24-h window. FHB risk maps were elaborated which are computer-generated images depicting infection risk using special interpolation techniques for point estimations of the risk by site-specific weather stations and forecasted weather within a wheat growing area. The goal of this predictive system is to help growers assess the risk of the FHB in their region (CNPtrigo—Embrapa, personal communication with J. M. Fernandes).

A disease forecasting computer model is being developed also for Pyricularia blast of wheat. However, because of the absence of a significant amount of quantitative data, exploratory simulation was developed. Model output was used for producing maps for a large geographical region.

To date, SISALERT is operating to predict risk of infection of the two aforesaid wheat diseases (FHB and Pyricularia blast). However, one of the major obstacles in computer disease forecasting models is the lack of their validity data. Although a lot of progress has been made, such models need to be improved further and validated regionally so that they can be successfully used by a variety of wheat growers practicing different cropping systems, which would finally make the integrated wheat disease management programs eco-friendly and cost effective.

2.4 Seed Transmitted Pathogens

The importance of proper seed health testing in general has been somewhat neglected. In Brazil for example, in the case of wheat there was little or no need to study seed health since most of the seed-borne fungi were controlled by compulsory seed treatment through legislation. During the early seventies this law was relaxed and also the use of mercurial fungicides was restricted or even prohibited due to their high toxicity. New fungicides have been introduced as substitutes for the highly effective mercurial fungicides and therefore it has become inevitable to perform the proper seed health testing and evaluate the fungicides to avoid their indiscriminate use.

Seed health testing has been practiced for a long time and was initially started by L. C. Doyer as the first official seed pathologist in the Netherlands. Since then much progress has been made in the world but it has not yet reached the point of meeting the expectations of the seed producing industries.

Normally, pathogens transmitted through wheat seed are *Stagnospora nodorum*, *Bipolaris sorokiniana*, *Drechslera tritici-repentis*, *Fusarium graminearum*, *Pyricularia grisea*, *Ustilago tritici* and some bacteria including *X. t. pv. undulosa*. Results of seed health testing are necessary to advocate proper sanitary practices which, in turn, may considerably reduce the yield losses caused by several diseases and to check the introduction and spread of new diseases from one region to another. Recommendations about sanitary practices should be made after careful considerations of several factors like, testing procedures, level of seed infection of a particular pathogen or a group of pathogens, epidemiology of the disease, cost and efficiency of fungicides and in certain cases the earliness of treatment after harvest.

2.4.1 Seed Health Testing

Seed health testing has the principal aim of establishing percentages of infection of different pathogens. Needless to say, saprophytes should not be taken into consideration. Several methods for seed health testing are available and selection of a particular method will depend upon the pathogens under study. For example, in the case of *S. nodorum* and *F. graminearum* a good correlation is observed between results from the laboratory and field (De Tempe 1958; Hewett 1975) and hence the commonly used tests such as pre-treated seed on malt extract agar, pretreated seed on potato-dextrose agar (PDA) + 0.2 % oxgall or non-pretreated seed on PDA + 0.2 % oxgall are quite satisfactory. For details on specific methods related to specific pathogens the reader may refer to Mathur and Cunfer (1993) and the ISTA publication—*Seed-borne fungi:* A contribution to routine seed health analysis (Machado et al. (2002).

For *B. sorokiniana* and *D. tritici-repentis* the commonly used tests are not satisfactory since these two fungi are very variable. Several strains of these two species exist of which some may be pathogenic whereas others may not. Infection percentage determined by routine methods would be an overestimation of the true infection that may occur in field conditions. According to the author's experience, very little or no correlation between field and laboratory testing with routine methods was observed. Such findings were observed also by Jorgensen (1974) and Mead (1942), even when the barley seeds were rather heavily infected by *B. sorokiniana. Drechslera* spp. may only cause severe root infections (root browning) without affecting emergence. These effects cannot be examined in field tests and consequently poor correlation is observed.

Undoubtedly, in certain cases the poor effect of the seed-borne inoculum on the emergence may be due to some phytotoxic effect of the fungicides. Special health test techniques should be used in the laboratory to determine the infection percentage of seedlings and not the seeds. Such percentages would be much closer to field conditions and would eliminate all saprophytic strains of the pathogen. If good correlation between such percentages and the infection percentages in the field are observed, then the recommendations on seed treatment can be made depending on the level of infection.

A method for detection of loose smut infection in the seeds of barley for example, was introduced in early fifties (Pederson 1956). Later, several modifications were made in the technique and also a few new techniques were introduced. These techniques were also used for wheat without having sufficient experimental evidence regarding their efficiency and reliability for wheat. Pederson (1956) reported that diseased embryos with loose smut were less easily extracted than healthy ones and advocated the extraction of all embryos. Later, Hewett (1972), in contrast to Pederson's results, demonstrated that partial or complete extraction of embryos is not important and does not alter the results even at lower infection rates when sampling errors are greater. In most techniques currently used for barley, extraction of all the embryos is not considered important. However, sufficient evidence is still lacking regarding the extraction of wheat embryos. Standardization of the technique is extremely important and without it a good correlation between laboratory and field tests could not be expected.

Determination of loose smut infection percentage in wheat is a problem. Loose smut infections in wheat and barley are determined by extracting the embryos and examining them under a binocular microscope for the presence of the mycelium of the fungus. The correlation between the infection percentage in the laboratory test and the field test is expected to be almost 1:1.

While establishing loose smut infection percentage and making recommendations about the seed treatment against loose smut, germination percentage of the seed sample should be considered. In case of very low germination percentage (below 70) it is possible that many of the infected seeds are incapable of germination and may affect results in field tests. Consequently, this would give a low correlation ratio when compared with laboratory results. The use of seed dressing fungicides will be uneconomical, especially when infection percentage in a seed sample with low germination percentage reaches the limit of tolerance and the seed treatment is recommended without correcting ratio of infection percentage to germination percentage. Hence, infection percentage must always be correlated with the germination percentage.

Correlation between laboratory and field tests depends upon cultivar resistance. Hewett (1975) reported that no smutted ears were produced in a field test using three samples of barley (cv. Emir) when the infections determined in laboratory test were 0.4 %, 0.8 % and 1.0 % respectively.

Several fungicides are available on the market for seed treatment. Most of these are effective against a particular pathogen or a group of pathogens. Broad-spectrum fungicides that are effective against all the important pathogens of a particular crop are rare. Moreover, degree of effectiveness may vary from fungicide to fungicide. Economics in the use of seed dressing fungicides invariably depends on the level of seed infection and resistance of the cultivar (Rubiales and Moral 2010).

It is wellknown that seed infections with fungal and bacterial pathogens tend to decline during storage. This leads to another question. Why cannot seed health testing be done just before sowing? But, if the seed health testing is left till the sowing time then the seed health testing laboratories may not be able to analyze large quantities of seed sample before the end of the officially recommended sowing period.

Also they may not have enough time to perform different tests for different pathogens. This leads to another question. Should the test be performed twice, once soon after harvest and again just before sowing? The cost of repeated tests would thus be very high.

Wheat bunt has been eradicated in Brazil. However, it is still an important disease in many countries. Seed infection percentage of *Tilletia* spp. causing bunt is determined by a relatively simple method. In this method, concentration of spores in a suspension is determined with the help of a hemocytometer and finally the percentage of seed infection/contamination is determined. Spores of *Tilletia* spp. are heavier than water and consequently their rate of sedimentation is very high. Because of the sedimentation problem in suspension with water, correct determination of spore concentration cannot be achieved. Spore suspension should be made in a solution (water + glycerin) in such a way that its specific gravity is equal to the specific gravity of the spores. This would avoid spore sedimentation. The spore suspension made in this way may be further subjected to a vibrator for one minute after adding a few drops of Tween-20. This would be beneficial, especially to get a uniform spore suspension. These are some of the points one needs to consider while using the techniques for *Tilletia* infections.

2.4.2 Level of Seed Infection

What should be the level of seed infection of a particular pathogen or a group of pathogens to advocate the use of fungicides? What is the minimum level of infection which permits recommendation of seed treatment and guarantees economical return in the field? Answers to such questions would prevent the indiscriminate use of fungicides. In certain cases, recommendations on seed treatments are based more on personal opinions and judgments rather than actual experimental evidence. If the minimum tolerance limits of infections are not established, then the whole purpose of seed health testing vanishes. Since bunt diseases are eradicated, tolerance level for seed infection with *Tilletia* spp. in Brazil is zero. Undoubtedly, recommendations for seed treatments may be made to check the introduction and spread of pathogens from one region to another or one country to another without any immediate considerations.

2.4.3 Epidemiological Aspects of the Disease

Epidemiological aspects of the disease are also important while considering the effectiveness of seed treatment. If the pathogen is not only seed-borne but also soil-borne and if the soil is heavily infested with such a pathogen, then seed treatment would be of little or no practical importance. For example, soils in the southern region of Brazil are heavily infested with some pathogens like *S. nodorum* and

P. tritici-repentis. Viable perithecia of these pathogens are observed throughout the year on wheat stubble (Mehta 1975, 1993; Mehta et al. 1992). By and large, these fungi are highly predominant in soils with no-tillage cultivation. In such cases, fungicidal seed treatment would only improve emergence and for some time the general health of the seedling. Other control measures like crop rotation and aerial fungicidal applications and use of resistant cultivars thus become necessary (Singh et al. 2007; Gurung et al. 2011).

2.4.4 Time of Seed Treatment

As mentioned earlier, questions such as when should the seed be treated, are most frequently asked by the seed growers and answers to these questions are manifold. Early seed treatment soon after harvest could be an important factor in economical seed treatment, depending upon the storage conditions and the moisture content of the seed. Seeds with about 12 % moisture content, when stored at about 5–10 °C, will not pose any problem. In such a case, earliness of treatment would not alter the results and seeds could be treated just before sowing. On the other hand, seeds with high moisture content stored at a high temperature will allow the fungal pathogens to grow which in turn will affect seed germination and emergence. Once the seed quality is affected by fungi, seed treatment would not do any good. Hence, in poor storage conditions seed treatment should be practiced soon after the harvest to prevent seed damage during the storage period.

All the above mentioned factors should be taken into consideration towards the production and use of healthy seed. Generalization regarding seed treatment cannot be made merely on the basis of high infection percentages. There are several ways of controlling seed-borne diseases of which seed treatment with chemicals is one that has been much talked about in recent years. It may be remembered that for seed transmitted bacterial pathogens, to date, no chemicals are available. Seed health problems can be minimized to a great extent by proper fungicidal sprays in the seed multiplication farms and also by strict inspection of such farms throughout the growing season.

The spot blotch pathogen (*B. sorokiniana*) is transmitted through seed, air and soil. In Brazil, for example, from 1970 to 1980, spot blotch was very important due to the cultivation of highly susceptible Mexican dwarf cultivars like Jupateco, Inia, Tanori, etc. in large areas. After 1980's new resistant or moderately resistant wheat cultivars were released. Tan spot caused by *Drechslera tritici-repentis* has now become much more important than the spot blotch caused by *B. sorokiniana*. It is important to note that in 1975–1980 only 80.000 ha were covered by no-tillage cultivation whereas at present more than 85 % of the wheat area of the State of Paraná, for example, is covered by no tillage cultivation system either fully or partially (without crop rotation). This has provoked the intensity and spread of tan-spot disease.

The existing reports demonstrate a higher transmission rate of *B. sorokiniana* through seed (Mehta 1993; Forcelini 1995). However it must be remembered that

B. sorokiniana is a facultative parasite, attacks various grass hosts and survives on the crop residue of different plant species throughout the year. The pathogen survives in the soil and with a higher concentration of its propagules in the no-tillage cultivation system (Mehta et al. 1992; Mehta 1993).

The pathogen survives in the soil in the form of conidia, mycelium and clamydospores. According to Diehl et al. (1983), root rot of wheat caused by a complex of pathogens could be responsible for a yield loss of about 18 %. In recent years, the severity of root rot has been drastically reduced because of cultural practices and cultivar resistance (Mehta 1993). During harvest time 2–3 days rain favor the fructification of the fungus and contaminate the seed (Mehta 1978). In the majority of cases the seed is externally contaminated and not truly infected. The severely infected seeds are shriveled and are eliminated during the seed processing. Besides, during the storage the level of seed contamination/infection falls drastically depending upon the time and condition of storage (Mehta 1993). This is a common phenomenon for several fungal and bacterial seed infections. For this reason, the recommendation would be to treat seed only if the infection/contamination is below 30 % and seeds having higher infection level are discarded. This criterion helps to a great extent in reducing the initial infection of seedlings and guarantees a good and uniform "stand". However, it must be remembered that the use of uncertified seed (pirated seed) with no seed health control represents 30 % of the total commercialized wheat seed sold in Brazil.

Glume blotch (netch blotch) of wheat caused by *Stagonospora nodorum* (Syn. *Septoria nodorum*) is an important disease especially in the southern region of Brazil. During 1980s glume blotch infections were noticed in northern Brazil on some Mexican cultivars like Tanori, Jupateco and Anahuac, due to excessive applications of nitrogen. The yield potential of Mexican cultivars could only be exploited to its maximum when heavy doses of fertilizers, especially the nitrogen fertilizers, were applied. This in turn predisposes the plant to *S. nodorum* infection (Mehta 1978). Similar to tan spot, *S. nodorum* survives in the left-over wheat stubble from one season to another in its sexual form *Leptosphaeria nodorum* and serves as an important source of primary infection. It is for this reason that the severity of netch blotch is higher in no-tillage cultivation system. However, the severity of the disease depends on the weather conditions such as continuous rain fall for 3–4 days or more and the temperatures varying between 15 and 22 ° C.

The level of tolerance for seed infection is interesting and even necessary in certain cases. Tolerance levels for seed infections must be established considering several aspects. For this purpose, the seed health tests must be based on research data, must be repeatable, easy to perform in different laboratories and should be based on epidemiological aspects of the disease in question. Ideally, the seed should be free from any pathogen, especially when the seed is the only source of infection.

It is evident that as a first step, comparative seed health tests should be performed in different laboratories to verify that there exist very few or no discrepancies between the results of different laboratories. This will permit the recommendations on tolerance limits for seed infections with much accuracy and credibility. In fact, comparative seed health tests have been practiced in several countries since 1975

(Yorinori et al. 1979; Machado et al. 2002). Such tests are necessary especially for newly developed seed health techniques. In the case of wheat, soybean and beans (*Phaseolus* spp.), there are several pathogens to be tested in seed health testing and some of the fungal and bacterial pathogens demand specific media and methodology.

In the USA, for example, almost 100 % of the bean seeds (in the State of Idaho >80 % and in Michigan 18 %) are official and there is very little or no pirated seed. The seed certification laws in these two States establish zero level of infection for seed multiplication farms as well as for laboratory tests for *Pseudomonas syringae* pv. *phaseolicola* and *Xanthomonas axonopodis* pv. *phaseoli* (Lahman and Schaad 1985). It must also be remembered that *X. a*. pv. *phaseoli* and *X. a. pv. phaseoli* var. *fuscans* are two distinct pathogens that cause bacterial blight in beans and also there are different non-pathogenic strains of these two pathogens.

2.4.5 General Considerations

Brazil is considered to be a "showcase" for the international agro-business. Establishment of tolerance limits would be useful especially when the use of illegal pirated seed is reduced to zero because pirated seeds are largely responsible in a major part for the dissemination of different pathogens. Brazil, for example, annually loses over 50 million tons of agricultural production due to the pirated seed of different crops, other than causing numerous phytosanitory problems.

2.5 Cultural Practices

Cultural practices constitute an important aspect in the integrated disease management programs. Through these practices severity of some of the diseases can be minimized or even eliminated without the use of the agrochemicals. Some of the aspects of cultural practices are discussed below.

2.5.1 Fertilizers

The growth and the productivity of a plant will depend on the availability of the macro and micro nutrients in adequate and balanced quantities. If these nutrients are not available in sufficient quantities in the soil, it will be necessary to complement them to the economic threshold level.

The principal macronutrients like N (nitrogen), P (phosphorus) and K (potassium) are needed in large quantities by plants and are responsible for increments in yield. In some cases the application of P alone for example, could be necessary to obtain

maximum economic yield. However, the unilateral application of fertilizers could be only transitory and later may induce deficiency of other nutrients in the soil and may limit the yield.

The application of N may be fractioned by applying a part of it during seeding and the rest as top dressing between tillering and early boot stage. Other than the macronutrients, for achieving higher productivity, a supply of secondary elements like Ca (calcium), Mg (magnesium) and S (sulfur), as well as micronutrients is necessary. The lack of one element or its presence in excessive quantities in the soil will reduce the efficiency of other elements and consequently reduce the yield.

The supply of one element in excess could induce physiological problems which in turn could reduce the yield and even could favor the attack of some pathogens. Excessive calcium in the plant reduces its resistance to loose smut and susceptibility to leaf rusts (Hubber 1976).

The availability of P may be limited in many tropical soils necessitating its application to increase the yields. In Brazil, in many cases this element has been applied in excessive quantities in the soil. Potassium is an essential element to increase plant vigor and in some cases it is also responsible for inducing plant resistance to pathogens. Vergenes et al. (2007), reported that potassium deficiency significantly increased spot blotch severity in two genotypes BL 2217 (moderately resistant) and Ciano 79 (susceptible) and stressed the importance of the soil fertility as part of an integrated crop management of Helminthosporium leaf blights.

In general, because of the lack of response in yield to the application of K many farmers have lost interest in applying this element to the soil in sufficient quantities. This is an essential element to increase plant vigor and in some cases it is also responsible for inducing resistance of the plant against pathogens.

Balanced fertilization implies consideration of a series of factors (EMBRAPA 2011). Although health and vigor of the plant have a major influence on its predisposition to diseases, no generalization can be made for all the host-pathogen interactions with respect to a particular nutrient. Some diseases are not influenced by nutrients while others show drastic effects. Although resistance is genetically controlled, it is expressed through the physiological process inter-connected with the nutritional state of the plant and the pathogen (Hubber 1976).

2.5.2 Soil Conservation and Tillage

Since the most remote antiquity, accumulated experiences have evidenced a series of advantages in soil mobilization for good crop development. Soil mobilization destroys the seeds of the weeds, larva and insects. However, for the tropical and sub-tropical regions soil mobilization is very much condemned and a no-tillage cultivation system has been introduced in the recent years. Several advantages of this system of cultivation are well documented. There is a general agreement among the growers, the scientific community and extension workers that soil mobilization as well as excessive traffic on the soil should be reduced as far as possible.

Tropical and subtropical regions of the world are known for their notorious instability in agricultural production and for their fragile eco-system. The gradual degradation of the soil structure is attributed to the excessive and heavy mechanization reducing the water infiltration rate, increasing soil compaction and soil erosion and consequently loss of soil organic matter.

The cultivation systems practiced in the Southern-Cone Region of Latin America can be classified into four categories:

1. The traditional system which includes residue burning after wheat harvest followed by one heavy disk plowing and two to five light disk harrow plowing to level the soil before seeding. This leads to soil compaction layer of 10–15 cm deep.
2. The conventional system which includes one heavy disk plow (Rome plow), followed by two light disk harrow.
3. The reduced tillage system (vertical tillage) which includes one chisel plow followed by one field cultivator.
4. The no-tillage cultivation system (direct drilling) where the soil is not mobilized and wheat is sown directly by special equipment.

No-tillage is a part of the conservation system of cultivation and as mentioned earlier it is practiced either partially or completely in over 80 % of the area of the State of Paraná, Brazil. A somewhat similar area exists also in the State of Rio Grande do Sul. The no-tillage system includes operations which maintain a sufficient quantity of crop residue on the soil surface, not revolving the soil, improves the soil quality, involves minimum tillage and consequently reduces soil traffic with agricultural machinery, avoids soil erosion and reduces infestations of weeds. The advantages of this system are well-known and its use is being widely practiced all over the world (Roberts and Johnston 2007). The conservation system of cultivation includes, other than no-tillage, crop rotation, crop-livestock integration (crop-pasture rotations in mixed farming), consortium of crops, permanent soil coverage with mulch or green crops and integrated management of pest and diseases.

Crop-livestock integration has twofold aims to achieve, in other words, production of fodder and mulch to keep the soil covered. While the brachiaria has an alellopathic effect for some soil pathogens, in 2011 some root and stem infections of soybean (*Glycine max*) caused by *Macrophomina phaseoli* were observed in some brachiaria fields in the State of São Paulo. One of the principal objective of the conservation system is to keep the soil permanently covered with mulch or with green crops (Denardin et al. 2007).

Since the advantages of conservation tillage including the no-tillage and crop rotations are widely accepted it is also necessary to admit and accept the challenges they face for their long term adoption. Under certain situations, some modifications in the no-tillage cultivation system may seem necessary. Influence of some of these practices on the severity of diseases is discussed in the following pages.

To start with, the no-tillage system should be implemented after careful consideration of several factors. Areas with heavy infestation of weeds, areas with heavy infestation of *Sclerotinia sclerotiorum*, areas with soil compaction, areas with soil erosion and sloping land (steep inclination of land), etc., should be avoided for

implementing no-tillage system. Some of the farmers are destroying terrace mainly to facilitate the field operations, thereby creating once again the problem of soil erosion and canceling most of the benefits of no-tillage. Thus, the soil terracing practice must be maintained. The reader may refer a specific publication available in this respect (Denardin et al. 2007; Caviglione et al. 2010).

Several fungal, bacterial and viral diseases of different crops under no-tillage cultivation can cause severe yield losses. Some pathogens attack all the plant parts while others attack only the above ground parts causing leaf necrosis and defoliation. The majority of diseases are transmitted through seed but some are transmitted through soil and air. The fungal diseases which survive in the soil or on non-crop residue from one season to another in the absence of living plants, are called necrotrophic (facultative parasites). Thus they serve as the initial source of inoculum and infect the plant soon after its emergence. On the other hand, the pathogens which need living plants for their survival are called obligate parasites or biotrophic pathogens, like rusts, smuts and bunts of wheat, rust of soybean and beans (*Phaseolus vulgaris*). The biotrophic pathogens are disseminated by seed or by air from one region to another and can cause infection in plants in both systems of cultivation traditional or no-tillage, depending on the weather conditions. The volunteer plants give shelter to some biotrophic pathogens and play an important role in the epidemiology of the disease.

The necrotrophic pathogen *Stemphylium* attacks different crops like potato (*Solanum tuberosum*), tomato (*Solanum esculentum*), onion (*Allium cepa*) and garlic (*Allium sativum*). The Stenphylium spot bloch caused a severe cotton (*Gossypium hirsutum*) leaf blight epidemic, in the State of Paraná, Brazil. The pathogen is not seed transmitted but transmitted through the crop residue. The severity of this disease was three times more in no-tillage than the conventional system of cultivation. The pathogen survives on the crop residue from one season to another and produces large quantities of spores under no-tillage cultivation.

Angular leaf spot of beans caused by *Phaeoisariopsis griseola*, is economically very important. In Brazil, beans are cultivated during the whole year being termed the rainy season crop, dry season crop and autumn/winter crop, but mainly it is a dry season crop. The pathogen survives on the bean crop residue from one season to another, mainly under the no-tillage cultivation system and continues producing the spores. The rate of seed transmission is between 1.5 and 2.0 %. Since the pathogen survives on the crop residue, the air-borne inoculum is not very important for the onset of the disease. In the United States of America, for example, the survival of *P. griseola* on the crop residue was up to 12 months (Celetti et al. 2005). Under Brazilian conditions the survival of the pathogen was observed for only 4 months under no-tillage cultivation

Tan spot of wheat *Pyrenophora tritici-repentis* is more severe in no-tillage than in the conventional system of cultivation. As mentioned earlier, in recent years the area under no-tillage has increased to over 80 % of the total area of the State of Paraná, under wheat cultivation and in the State of Rio Grande do Sul, it is around 1.5 million hectares. The quick expansion of this system of cultivation has brought some disease problems. The severity of some diseases is directly related to the introduction of the no-tillage cultivation system.

Mehta (1978, 1993) reported that the severity of tan spot in no-tillage during three years of experimentation was substantially higher than the conventional system of cultivation and that the disease caused a yield loss of 40 %. In no-tillage the perithecia of the sexual stage of the fungus survives on the crop residue for 2 years or more and continues liberating the ascospores which serve as the initial source of inoculum. The ascospores are not carried to long distances. In no-tillage the symptoms of the disease are noticed within 20–25 days after sowing. The disease lesions produced by the ascospores produce the first cycle of asexual spores (conidia) which in turn infect the wheat in the conventional as well as in the no-tillage cultivation system.

Unlike the ascospores, the conidia travel long distances. In the conventional system the symptoms of tan spot are observed 15–20 days after the appearance of the first symptoms in the no-tillage cultivation. Consequently, the secondary conidial production cycle disseminates the disease still further in other fields either of conventional or no-tillage. Normally, the high severity of the disease in no-tillage is related to continuous precipitations during the initial period of the crop cycle (Mehta and Gaudêncio 1991). It must be remembered however, that wheat is still one of the best options for winter and appropriate crop rotations would reduce the intensity of tan spot in no-tillage cultivation (see chapter on crop rotation and their role in disease management).

The spike diseases of wheat and triticale (*X.Triticosecale*) caused by *Pyricularia grisea* and *Gibberella zeae*, can cause losses of up to 40 % (Mehta and Baier (1998). Very little information is available as regards the influence of system of cultivation on the severity of these two diseases (Zambolim et al. 2001).

Black oat has been used in crop rotations over two decades because of its resistance to some soil-borne diseases and because of its large amount of green matter (mulch). During this period only the cv. IAPAR 61 of black oats was used without any cultivar diversification. As a result, the resistance of this cultivar to *P. grisea* was broken in 2005. Later, cultivars of white oat (*A. sativa*) were found resistant to *P. oryzae* (also see chapter on *P. grisea*). Recently, the resistance of one of the white oat cultivars IAC 7, to *P. grisea* was also overcome.

The severity of Cercospora leaf spot of maize caused by *Cercospora zeae-maydis* is normally more severe in no-tillage. However, there is no exact information about the loss in yield caused by this pathogen in no-tillage cultivation.

The severity of soybean stem canker caused by *Diaporthe phaseolorum* f. sp. *meridionalis*, has always caused more problems in no-tillage than in the conventional system of cultivation. The pathogen survives on crop residue for over 2 years. Because of the availability of resistant cultivars, this disease now poses no problem.

The bacterial diseases caused by different pathovars of *Pseudomonas*, *Xanthomonas* and *Curtobacterium*, attacking several economically important crops, are basically seed transmitted and hence their severity does not depend on the system of cultivation.

In tropical and semi-tropical regions, it is known that most of these bacteria do not survive in the soil because of the high temperatures which prevail during 4–5 months in the summer (Mehta 1993). Nonetheless, some of the bacteria like

Curtobacterium flaccumfaciens pv. flaccumfaciens (Cff) of beans may survive on the leftover stubble from one season to another (Leite Junior et al. 2001).

These are only a few examples of some diseases in which the tillage practices may have some influence on disease incidence and severity.

2.5.3 Crop Rotations and Their Role in Disease Management

Crop rotation implies altering plant species in a given period of time within the same cropping area. The objective should be to maintain the diseases below threshold level in no-tillage because total control will be difficult to obtain and it will not be necessary or cost effective. Intelligent crop rotation is an inherent part of conservation tillage and it basically includes six aspects: (1) Maintaining biodiversity, it must be remembered that diversity includes diversity of plant species as well as diversity of cultivars of the plant species in question; (2) Reducing infestation of weeds; (3) Breaking the disease cycle; (4) Keeping the soil always covered with mulch or green crop; (5) Supplementing some of the nutrients essential for crop development; and finally; (6) Increasing the profit of the farmers over a period of time.

The use of different plant species in crop rotation is very important since most diseases are specific to a given species and do not attack other species of plant. Thus, an appropriate use of non-host crops for subsequent planting helps in breaking the disease cycle especially of the necrotrophic plant pathogens. Sufficient care should be taken while selecting a particular crop and its cultivar to be used in the rotation. In wheat growing areas, triticale and rye (*Secale cereale*) are not ideal crops for rotation since both are susceptible to pathogens that attack wheat and may contribute to maintain the cycle of the diseases. Wheat is a cash crop and economically more important than the other two crops. Similarly, if foxtail (*Setaria italica*) is preferred for crop rotation, it should not precede wheat, barley, triticale and rye, since it is also highly susceptible to Pyricularia blast (*P. grisea*).

As mentioned earlier, rusts and smuts are biotrophic pathogens and do not survive in crop residue so they are not controlled by crop rotation. Triticale and rye are susceptible to tan spot pathogen. White oats (*A. sativa*), maize, pigeon pea (*Cajanus cajan*), crotalaria (*Crotalaria juncea*), millets *(Pennisetum americanum;* Syn. *P. typhoides*), radish (*Raphanus sativus*) and the leguminous crops can be used for rotation. These crops can be used for grain production and for green manuring as well (Calegari et al. 1993; Denardin et al. 2007).

The cotton pathogen *C. gossypii* var. *cephalosporioides* can survive on crop residue for over 2 years. While seed inoculum is normally eliminated through fungicidal seed treatment, the soil inoculum can be eliminated or drastically reduced through crop rotation with non-host crops like millet, pigeon pea (*Cajanus cajan*), maize and soybeans. Millet serves as an alternative to soybean and pigeon pea as a complement to maize (Zancanaro and Tessaro 2006; Scaléa 2007).

Potential fungal pathogens transmitted through soil and causing economic losses include species of *Sclerotinia, Rhizoctonia, Phytopthora, Fusarium, Macrophomina, Ophiobolus* and *Diaporthe*.

The millet and sorghum (*Sorghum vulgare*) are attacked by *Claviceps purpurea*, commonly referred as ergot, posing a new threat since these crops are used in crop rotation especially for animal food and/or for keeping the soil covered. Animal feedstuffs contaminated with sporidia of this fungus are poisonous to animals. In this case appropriate alternatives should be worked out (Bogo and Boff 1997; Cultivar 1999).

Beans and peas (*Pisum sativum*) are susceptible to *Fusarium oxysporum* f. sp. *phaseoli*, *F. semitectum* and *F. solani* f. sp. *phaseoli* (Mehta and Gaudêncio 1991; Zambolim et al. 2001). Besides, *F. oxysporum* f. sp. *vasinfectum*, *F. moniliforme* and *F. semitectum*, are also pathogenic to cotton, wheat, maize and soybean and hence resistant cultivars of these crops should be used for rotation only in soils that are not highly infested with these pathogens. In the case of cotton, the severity of *Fusarium* (*F. oxysporum* f. sp. *vasinfectum*), tends to increase when associated with nematodes (*Meloidogyne incognita*, *Rotylenchus reniformis* and *Pratylenchus brachyurus*) making a disease complex. The integration of livestock-cropping especially in the Cerrado region of Brazil and the use of *B. decumbens* reduce populations of *M. incognita*, *M. javanica* and *P. brachyurus* in no-tillage cultivation (Campos et al. 1997).

Other than the *Fusarium* spp. the soil-borne pathogens include *Sclerotinia sclerotiorum*, *Rhizoctonia solani*, *Sclerotium rolfsii* and *Macrophomina phaseoli*. These are problematic pathogens since they have a wide host range and demand 4–6 years of rotation with non-host crops. For this reason, constant soil monitoring is desirable, besides reducing the frequency of using susceptible hosts in time and space. On the other hand for the necrotrophic foliar pathogens like *B. sorokiniana*, *Drechslera tritici-repentis*, *P. griseola*, *C. gossypii* var. *cephalosporioides* and *C. zeae-maydis*, crop rotation of 1–2 years would be sufficient. Because of its high susceptibility to *S. sclerotiorum* sunflower is not usually chosen for large scale planting in the State of Paraná, Brazil (Cardoso and Mehta 1997). Some *Vicia* spp. (*Vicia villosa, V. sativa*), *C. cajan* and cowpea (*Vigna unguiculata*) may be considered as good options (Calegari et al. 1993).

Chickpea (*Cicer arietinu*m) can be another option for crop rotation especially for the semi-arid tropics. Being a leguminous winter crop and with a crop cycle of only 120 days, chick-pea is also tolerant to long dry periods. The deep root system of chickpea helps in breaking the soil compacted layer, produces abundant nodulation and hence fixes atmospheric nitrogen. It tolerates low temperatures and its yield potential can vary between 2.0 and 4.0 t ha^{-1}. Other than these advantages, introduction of chickpea in a crop rotation system would help break the disease cycle of some wheat pathogens and would contribute towards increasing bio-diversity in cropping systems.

However, as regards crop rotations, there is still a lack of information with respect to: (a) Influence of different crops used either to cover the soil and/or for grain production, on the severity of diseases of the principal crop; (b) Knowledge about the host range of the major pathogens of different plant species; (c) The level of disease resistance of different cultivars of each one of the plant species to be used in crop rotation.

Very few farmers use the crop rotation. There are several reasons including lack of adequate orientation, lack of enough seed, lack of appropriate machinary for

seeding and difficulty in commercialization of the harvested grain at the right time because of lack of demand. As a consequence, the use of some crops in rotation may not be cost effective. It is also necessary that the majority of farmers should practice crop rotation because the farmers who do not do so may let their land become infested with necrotrophic pathogens and serve as a source of primary inoculum to the other neighboring farms.

Monoculture of cotton grown over the crop residue of millet for example, is a common practice in the Cerrado region of Brazil. For cotton it may be difficult to follow all the basic rules of no-tillage in the Cerrado region of Brazil, mainly because of the nature of the crop itself. Besides having a long crop cycle, the cotton crop residue has to be deeply buried mainly to break the life cycle of boll worm (Zancanaro and Tessaro 2006).

Finally, the crop rotation which does not bring economic returns for a period of 3–4 years, will not be sustainable. When a mixture of seeds of different crops is recommended for planting for green manuring and to create a mulch on the soil, it is necessary to take into consideration the cost of seed, the cost of operations involved, the quantity of green matter really required and the frequency of such operations in a given time and space. The majority of farmers do not possess machinery either for planting or for managing the cover crops. It must be remembered once again that the basic objective of crop rotation is always to maintain the soil covered for most of the year. Depending upon microbial activity, a part of the nitrogen is consumed by the micro-organisms. Nitrogen is easily leached into the soil and also volatilized.

One of the arguments is that the higher the amount of dry matter on the soil surface the higher will be the richness of soil in terms of nitrogen and phosphorus. However, one should fix a limit for this. A minimum quantity of the dry material on the soil should be worked out. In fact, the maximum amount of organic matter on the soil surface is the one which the soil can mineralize. It is time to establish the optimum quantity of dry organic matter needed to cover the soil so that the operation becomes economical and sustainable and the crop rotation does not become a "fairy tale".

Wheat is still one of the best options for winter in the Latin-American region. There are several options for crop rotation and selection of a particular crop depends on whether it is for grain production, for green manuring, or for mulch (Gazziero 1994). It also depends on the government incentive and the local commercial needs. Choosing white oats as well as maize and sunflower seems appropriate for most of the area. However, wheat should not be followed by maize or sunflower because of the deficiency of nutrients and the resulting diseases like Fusarium root rot. Use of some leguminous crops is interesting since they generally are not attacked by most of the wheat and soybean pathogens and at the same time improve the soil fertility.

In Argentina, in some disease prone areas the soil is ploughed once a year after the soybean harvest and before sowing wheat to incorporate the crop residue and minimize the early infections of tan spot (Fernando et al. 1987).

Integrated foliar disease management to prevent yield loss in Argentina wheat production was investigated by Simón et al. (2011). They evaluated the combine

effect of tillage, N fertilization, fungicides and resistant cultivars in reducing foliar disease severity. According to these authors the disease was less severe in zero tillage which received a fungicide compared to conventional tillage plots that were not treated with fungicide. They concluded that in spite of the increase of necrotrophic diseases, developing no-till system in wheat monoculture is possible without significant yield losses if effective disease management practices are applied.

The effect of crop rotation on the severity of wheat root diseases has been extensively studied in Brazil (Diehl 1979; Diehl et al. 1982, 1983; Reis 1985; Reis and Baier 1983; Fernandez et al. 1993). Diehl et al. (1982) reported a loss of 20 % because of the monoculture (wheat-soybean-wheat). Reis and Ambrosi (1987) reported that the increase in wheat yield due to crop rotation was noticed only after 5 years. In the following years, Reis and Santos (1989), reported that 1, 2 and 3 years of rotation without wheat in the winter, did not increase wheat yields statistically. Mehta and Gaudêncio (1991), also did not observe any gain in wheat yields after 4 years of crop rotation without wheat in the winter. Thus, it seems that crop rotation with more than 1 year without wheat in the winter may not be very profitable. However, due to the expansion of no-tillage cultivation and consequent increase in tan spot severity, an appropriate crop rotation should be worked out considering the epidemiological aspects of this particular disease. Soils severely infested with the fungus *Gaeumannomyces graminis*, need special attention. In this case, crop rotation with non-host leguminous species becomes extremely important. White oats are more resistant to this pathogen and may be a good option for winter.

2.5.4 Crop Residue

Residue burning to reduce the soil-borne inoculum of some pathogens has been extensively discussed during the past few years. Considering the problems of loss of organic matter and soil erosion, residue burning is generally condemned. It may reduce the soil inoculum but does not eliminate it completely. Rees and Platz (1979) reported that burning wheat residue drastically reduced the soil inoculum of tan spot pathogen but did not eliminate it completely and with the result that the little inoculum left-over on the soil surface could still be enough for tan spot epidemic. Destruction of crop residue or its deep incorporation in the soil is practiced in some special cases like the boll worm of cotton caused by *Anthonomus grandis*.

The organic material in the form of left-over crop residue undergoes the process of decomposition (degradation of protein) and liberates nitrogen for the plant and remains in the soil as a fertilizer (Roberts and Johnston 2007). The burning of crop residue signifies loss of fertilizer and thus increases the cost involved in supplying an additional amount of nitrogen in the soil. The process of degradation of protein and the cycle of organic material is somewhat complex as can be summarized in Fig. 2.2.

The discovery of bacteriostatic substances could bring additional benefits. Bacteriostatic nitrapirina, inhibits the growth of bacteria *Nitrosomonas* of the soil and blocks the conversion of ammonia into a leaching form of nitrogen. Consequently

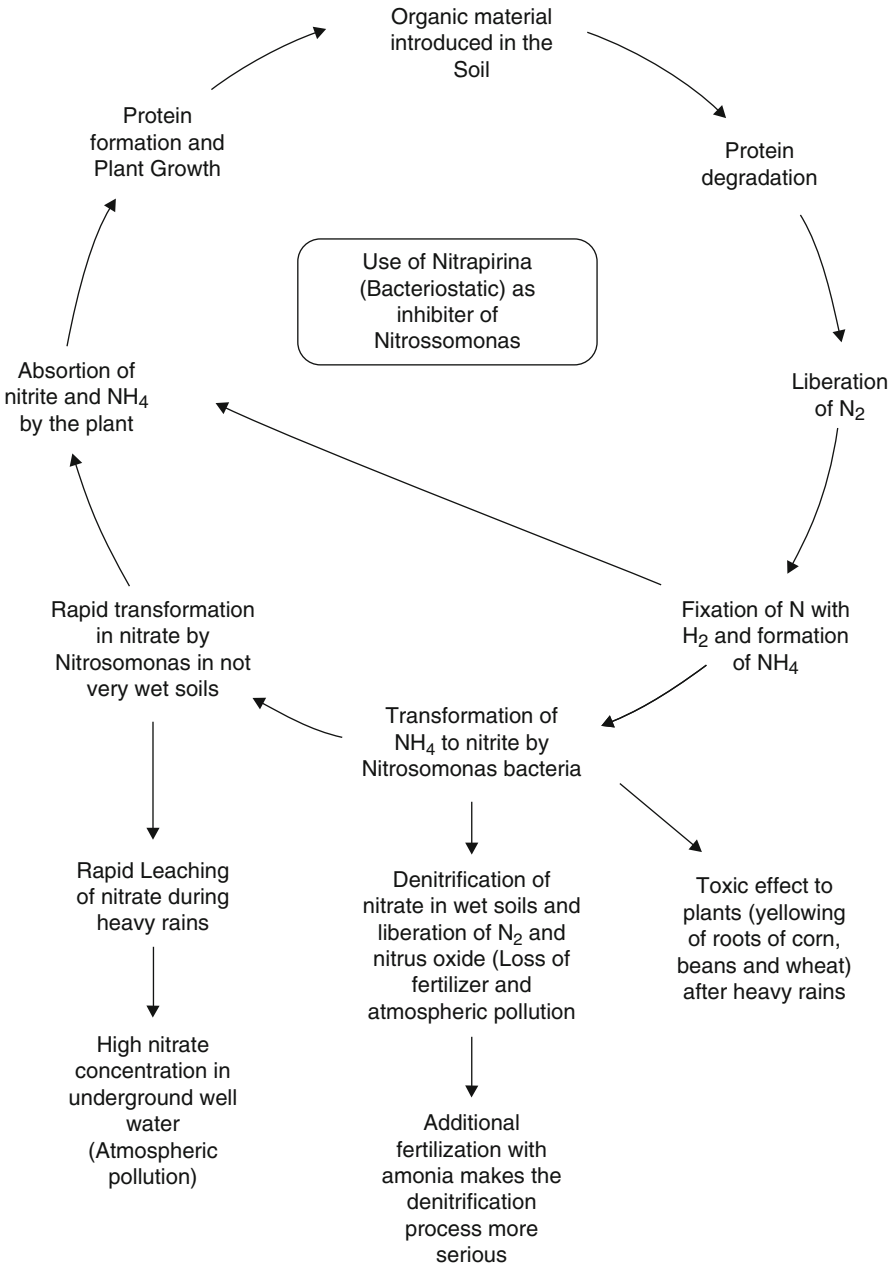

Fig. 2.2 Process of degradation of protein of organic matter

the loss of nitrogen is reduced and the crop productivity is increased (Barrons 1980). Nitrapirina can be incorporated in the soil together with the fertilizer, but this is not yet a common practice on a commercial scale.

2.5.5 *Diversification of Sowing Dates and Cultivars*

The severity of some diseases could be reduced by simply changing the seeding dates and/or by diversification of cultivars. Stem rust of wheat for example, is controlled in the South of Brazil by changing the sowing dates. Before the 1980's wheat had been sown until the end of June. No severe epidemic of this disease has been noticed since 1982 when change in sowing dates to the period between April 1 and May 10 was effected.

Stem rust prefers hot temperatures (25–28 °C) which normally occur after the first week of August. During this time, the wheat planted between April and May has reached its maturity and the rust does not reach epidemic proportions such as to cause damage to the plant.

Wheat blast caused by *Pyricularia grisea*, is partially controlled by change in sowing dates. In the wheat region above parallel 24 south for example, the sowing dates were altered and recommendations were made to seed wheat not before 10th of April. This strategy is working well since the late 1980's (Kohli et al. 1996).

Diversification of cultivars is another strategy to reduce the severity of diseases. As far as possible, more than one cultivar should be planted in a particular area and planting the whole area with a single cultivar should be discouraged even when a particular cultivar is most preferred by the farmer.

The problem of wheat blast caused by *P. grisea*, during 1985–1987 was probably provoked by planting a single rice cultivar in the North of Paraná, Brazil. Rice cultivar Cica 9 was immune to rice blast pathogen and hence within a few years over 90 % of the rice area of the State was covered by Cica 9. Later, within few years its resistance was broken by a new race (pathotype of *P. grisea*). It is presumed that this is the race which attacked wheat eliminating unnecessary virulence genes of rice and creating a new virulence gene to wheat. Perhaps for this reason the *P. grisea* isolates of wheat do not attack rice. Valent and Chumley (1991), also believed that the pathogen that attacks wheat is distinct form than the one that attacks rice.

In fact, during 1985–1987, late rice was planted in the month of December-January, whereas early wheat was planted during the same years during the second week of February. During the rice harvest the release of clouds of *P. grisea* spores coincided with the emergence of wheat spikes which was planted not too far from the rice fields and the spores were deposited on the rachis of the wheat spikes causing infection.

Thus, from this point onwards, the *P. oryzae* of rice became adapted to wheat which had never been its host, at least in Brazil. This assumption seems to be more convincing. Since there is no experimental proof for this assumption it is considered only as circumstantial evidence. There have been several unanswered questions

about the origin of the inoculum which initially attacked wheat and they may remain so, for a long time to come. In any case, the wheat blast story, once again emphasizes the need for diversification of cultivars and the sowing dates.

2.5.6 Alternative Methods for Disease Control

Management of Sclerotinia (*S. scerotiorum*), *Rhizoctonia solani* and *Fusarium* spp. of soybeans and other leguminous plants could be achieved through biological control using *Tricoderma asperellum* and *T. harzianum* as well as mulch of *Brachiaria ruziziensis* (syn. *Urochloa ruziziensis*) (Pomella 2007). Gorgen et al. 2007) reported a reduction in initial inoculum of the pathogen in the soybean crop through the application of spores of *T. harzianum* (2×109), at a rate of 1.5 L ha^{-1}, along with the fungicidal seed treatment. They observed 100 % parasitism and death of 70–100 % scleroids. In contrast, they observed only 16–75 % parasitism in uncovered soils. Further experimentation seems necessary before the use of *Trichoderma* spp. can be practiced on a commercial scale.

It is believed that maize cultivated in consortium with *U. ruziziensis* can drastically reduce the soil inoculum of *S. sclerotiorum* in comparison with maize cultivated alone. Costa and Rava (2003) reported that in the integrated crop-livestock system, the crop residue of *U. brizantha* and *U. ruziziensis* cv. Marandu, has a positive effect in controlling *F. solani, R. solani* and *S. sclerotiorum*. On the other hand, as stated earlier, infections of *Urochloa* spp. caused by soil-borne charcoal-rot disease (Macrophomina) caused by the fungus *M. phaseoli* have been recently observed in the State of São Paulo. Since *M. phaseoli* has a wide host range including maize and sorghum, soils not highly infested with *M. phaseoli* should be carefully identified for crop rotations.

The integrated crop-livestock system has several advantages including the reduction in disease severity as well as reduction in pests and weed outbreaks (Studdart et al. 1997; Franzluebbers 2007; Gorgen et al. 2010; Vilela et al. 2012). Considering several advantages of the integrated crop-livestock system (agrisilvipasture), especially in the Cerrado region of Brazil, the USA and a part of Africa, it is believed that this system will gain a much greater momentum over the course of time.

The use of calcium silicate (Si) in agriculture has long been investigated. Depending upon the plant species the quantity of Si in the plant biomass varies between 1 and 10 %. Other than this, one of the effects of the presence of Si is in the reduction of disease severity. A literature review in this matter is presented by Rodrigues and Datnoff (2007). According to Seebold et al. (2004), calcium silicate applied at the rate of 0.1 t ha^{-1} was efficient in controlling rice blast of wheat.

Control of other pathogen species like *Sphaerotheca, Pythium, Uncinula, Blumeria* and *Fusarium* was also demonstrated by (Rodrigues and Datnoff 2007). Control of powdery mildew of beans (*Erysiphe poligoni*) was observed by us

under glasshouse conditions with soil application of calcium and magnesium silicate at the rate of 0.2 t ha^{-1}. The treated plants showed higher vigor than the untreated ones (Unpublished data). Further research is needed to shed more light on this matter.

Undoubtedly, the conservation system of cultivation would be most welcome to overcome several problems including the disease problems allied with other integrated disease management practices as discussed in earlier chapters.

To achieve success in sustainable and eco-friendly conservation system of cultivation an integration of specialist of different disciplines and the collaboration of farmers and the extension workers become inevitable (Mehta 1996a).

2.5.7 Precision Agriculture and General Considerations

In recent years, precision agriculture has been much talked about. Initially, precision agriculture was aimed at image-based satellite remote sensing for soil monitoring for rational use of fertilizers and to detect water logging and sloping areas, so as to make appropriate use of natural resources. Now precision agriculture is dealt with in a much broader sense. According to Moran et al. (1997), multispectral images can be used for: (1) identifying and monitoring soil moisture content; (2) crop phenology stage; (3) crop biomass and yield production; (4) crop evapotranspiration-rate; (5) crop nutrient deficiencies; (6) crop disease; (7) weed infestation and; (8) insect infestation.

By and large, precision agriculture is achieving accuracy in different aspects of agriculture production, in order to obtain food security through more efficient use of natural resources.

Any change in agricultural practices towards increasing safe food production without degrading soil and water resources and the atmosphere, would necessarily be a part of precision agriculture. In this respect, a few examples can be cited.

In Loess Plateau (China), for example, the use of plastic film mulch on over 51,000 km^2 is being used to increase soil temperature (Turner et al. 2011). According to these authors, the use of plastic film mulch to warm the soil in spring has enabled the economic production of maize in the colder regions of the Loess Plateau where it was not possible without mulch. For precision farming, on the other hand, Romanenko et al. (2007), suggested planting of different cultivars in a commercial wheat farm, each one having a different gene for disease resistance and thus forming a mosaic pattern of genes in the field and consequently increasing yield.

Precision agriculture demands future trends in developing cultivars through modern biotechnological tools in resource poor areas, including areas with frequent droughts and heat waves (Turner et al. 2011).

Precisely, all aspects dealt with in the preceding chapters should form the basis for precision agriculture and even a little more (Fig. 2.3). In addition to eight points raised by Moran et al. (1997), issues pinpointed in the preceding chapters should be intrinsic of precision agriculture and can be summarized as: use of

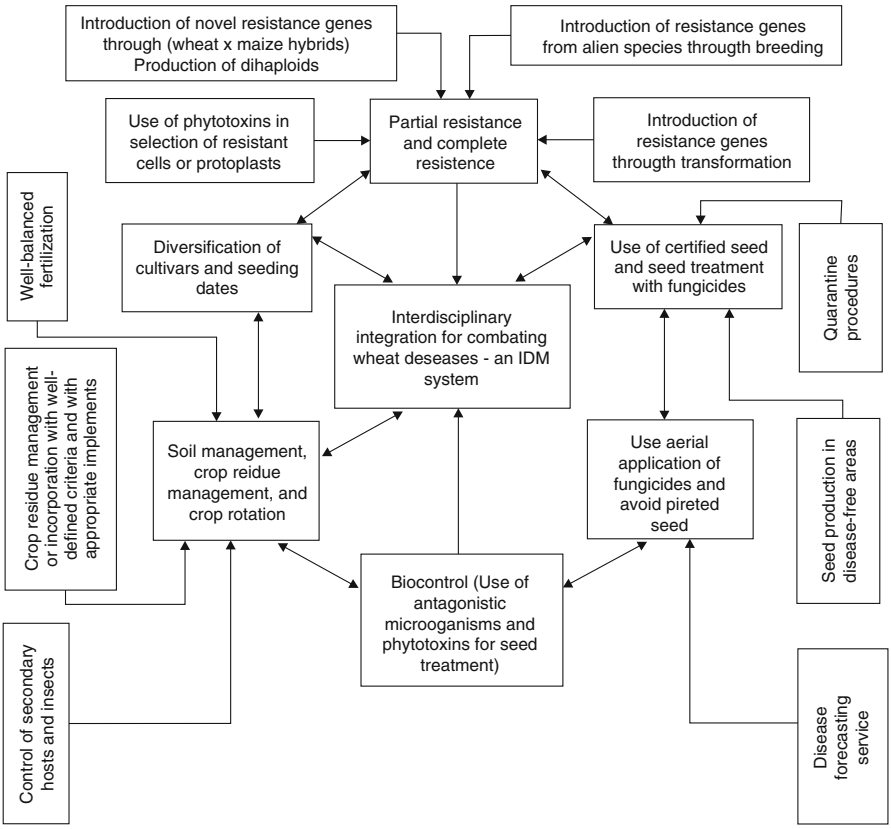

Fig. 2.3 Wheat disease control tools and their integration

appropriate mineral fertilization depending upon the soil analysis; use of healthy and certified seed and avoidance of pirated seed; use of conservation tillage practices like appropriate control of weed and soil structure before implementing no-tillage cultivation; rain water-use efficiency; intelligent crop rotation including biodiversity of plant species and their cultivars for green manuring or for grain production; crop residue management; integrated pest and disease management practices; use of disease forecasting models; as well as analysis of pesticide residue in grains (Cook 2000).

Precision agriculture should also deal with demand of wheat buyers to insist phytosanitory certificates to insure no risk for consumers especially for fungi associated toxins [alkaloids produced by *Claviceps purpurea* (ergot); Ochratoxin A, produced by *Penicillium* and *Aspergillus* during storage (tolerance limit 5 μg/kg for grains); Vomitoxin produced by *F. graminearum* (tolerance limit 500 μg/kg in grains)]. (Peña 2007).

Wheat quality attributes, other than phytosanitary and flour qualities like gluten and protein, involve a series of transactions in the process of value chains, adding value at each stage till the processing and marketing of the farm produce to the final consumer. While the precision agriculture may reduce the cost involved in some inputs, in the end it may in some cases, increase the cost of the final product.

The recent food crisis has been provoked by population growth, climate change, water scarcity and the use of crops for biofuels (Amuzescu 2009; Chakraborty et al. 2011; Pritchard 2011; Shaw and Osborne 2011; Turner et al. 2011; Barak and Schroeder 2012; Serge et al. 2012). Due to the climate change phenomenon crop yields are predicted to decrease in the near future especially in the semi-arid regions. Precision agriculture is expected to address these issues and suggest appropriate changes for a more effective, sustainable and eco-friendly agriculture production system as a whole.

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Chapter 3
Spike Diseases Caused by Fungi

Wheat spikes are attacked by several fungal diseases out of which only Scab, Pyricularia blast, Smuts, Bunts and Ergot are described in the following pages. Other fungal diseases are dealt with under a different chapter since they also attack leaves and seeds. Smut and bunt diseases are sometimes referred as "Smuts". There are two kinds of smut diseases; the Loose smut and the flag smut. The Loose smut is of common occurrence wherever wheat is grown whereas Flag smut occurs in restricted areas. As regards bunts, there are three kinds called Common bunt, Dwarf bunt and Karnal bunt. These bunt diseases have been eradicated from some countries including Brazil, due to an obligatory act of seed treatment with mercury based fungicides. After the Common bunt and the Dwarf bunt have been eradicated the compulsory seed treatment law was lifted in the 1960s and since then their occurrence has not been reported from Brazil. The bunts are still important in some other countries. Comprehensive information especially on smuts and bunts is presented by several workers (Heald and Holton 1940; Fischer and Holton 1957; Western 1971; Wilcoxson and Saari 1996; Bockus et al. 2010).

3.1 Bunts

There are three kinds of bunts called common bunt, dwarf bunt and Karnal bunt. These bunt diseases have been eradicated from some countries including Brazil, due to an obligatory act of seed treatment with mercury based fungicides. After the common bunt and the dwarf bunt have been eradicated the compulsory seed treatment law was lifted in the 1960s and since then their occurrence has not been reported. The bunts are still important in some other countries. A short description of these diseases is presented in the following pages. However, comprehensive information is presented by Wilcoxson and Saari (1996) about all bunt and smut diseases and the reader may refer to this publication for more details.

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3.1.1 Common Bunt and Dwarf Bunt

Common bunt and dwarf bunt are more important diseases than Karnal bunt in some parts of the world. Dwarf bunt is also known as short smut, stunt smut, stubble smut and TCK smut and it was first recognized in 1935. It occurs in the USA, Canada, continental Europe and Sweden, where winter wheat is subject to prolonged snow cover. It has not been observed in spring sown wheat (Fischer and Holton 1957). Dwarf bunt reduces both yield and grain quality. Dwarf bunt in the USA for example, has caused much more damage in yield and quality than any other wheat disease and this was especially so during the first half of the twentieth century (Wilcoxson and Saari 1996). In 1927, bunt reduced wheat yields by more than 760,000 metric tons (Holton and Heald 1941). According to Gasperi (1961), the importance of common bunt was reduced after 1945 but earlier years it had provoked yield losses of about 70 %.

Symptoms

Infected plants are stunted and produce more tillers than normal healthy plants. Infected grains have a strong fishy odour. The infected spikes are transformed into a black mass of teliospores. The seed pericarp remains intact but it can be easily disintegrated and a mass of spores is then released (Fig. 3.1). All the tillers of infected plant show infected spikes. No grains are formed in the infected spikes and the normal kernel is replaced by a bunt ball containing a mass of spores.

The presence of only a few spores in a grain/seed sample may drastically affect international marketing. Thus the effect of the disease is greater than the yield losses it may cause. Because of strict international regulations the spread of the disease from one country to another is kept under control.

Causal Organism and Epidemiology

Common bunt is caused by fungus *Tilletia laevis* Kuhn and *T. tritici* (Bjerk.) Wint and dwarf bunt is caused by *T. controversa* Kuhn. *T. laevis* and *T. tritici* are two very similar fungi differing only by their spore morphology. In the earlier years the common bunt fungi were named as *T. caeris* and *T. foetida*, but now the names *T. laevis* and *T. tritici* are internationally accepted. The teliospores of *T. laevis* are smooth. They are sub-globose, yellowish-brown, similar to those of *T. tritici* but with broad deep reticulations on the spore walls and measure 17–22 μm in diameter (Line 1993).

Pathogenic races of dwarf bunt and common bunt are known to exist. Several resistance genes are identified. Virulence of dwarf bunt and common bunt in wheat is governed by the same genes (Holton and Heald 1941; Hoffmann and Metzger 1976; Metzger and Hoffmann 1978; Wilcoxson and Saari 1996).

The dwarf bunt pathogen infects wheat and triticale. It also occurs on grasses like *Aegilops*, *Agropyrin*, *Agrostis*, *Alopecurus*, *Arrhenatherum*, *Beckmannia*, *Bromus*,

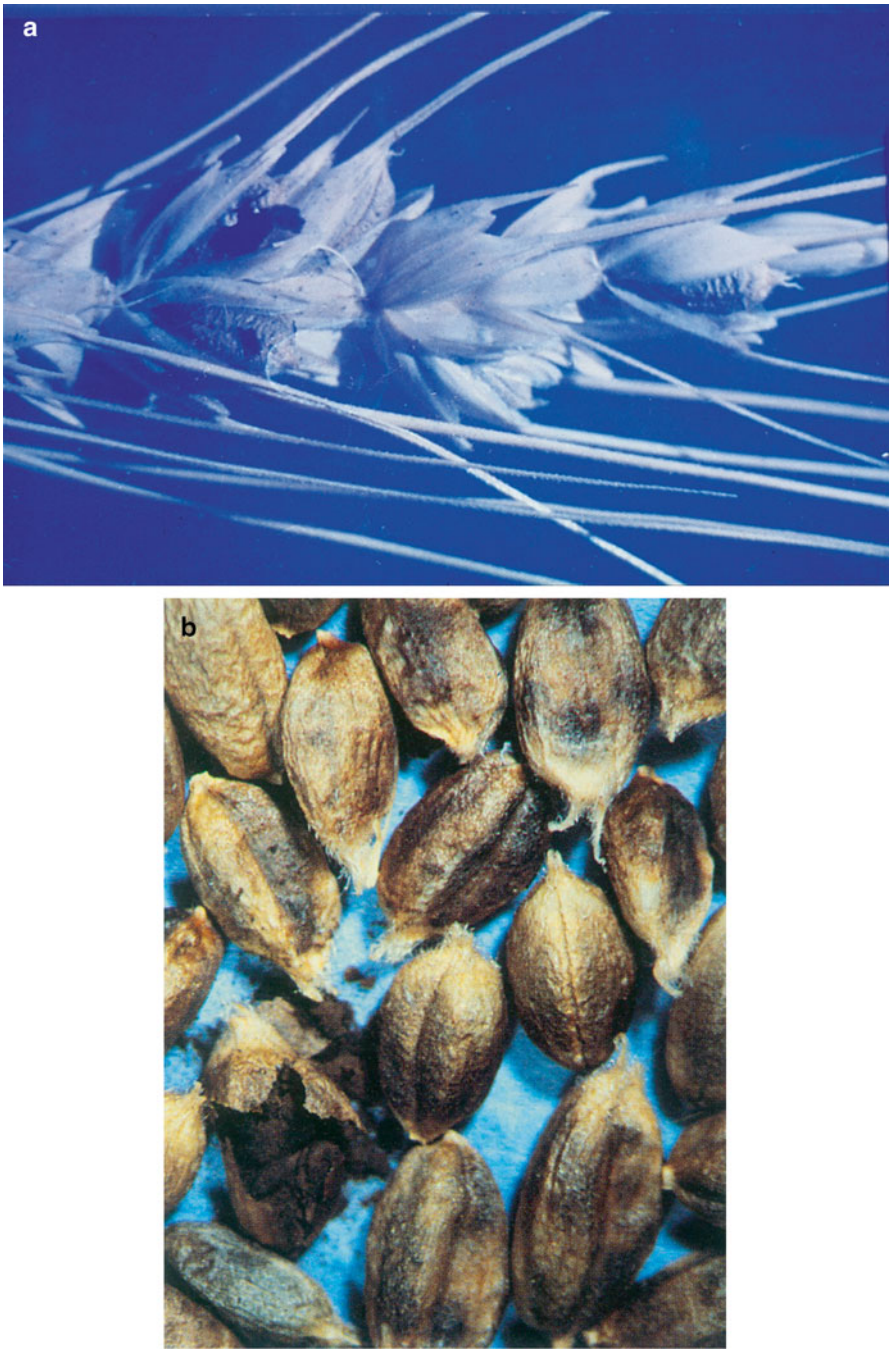

Fig. 3.1 Common bunt (*Tilletia laevis* and *T. tritici*). (**a**) Infected wheat spike; (**b**) infected wheat kernels (Courtesy: CIMMYT)

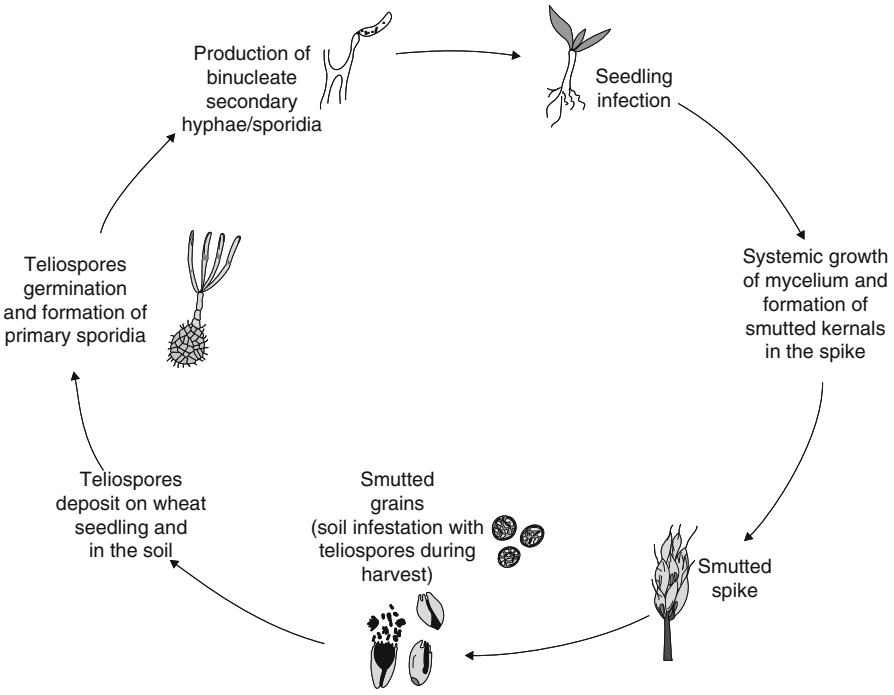

Fig. 3.2 Life cycle of common bunt

Dactylis, *Elymus*, *Festuca*, *Holcus*, *Hordeum*, *Koeleria*, *Lollium*, *Poa*, *Secale* and *Trisetum* (Wilcoxson and Saari 1996).

The pathogen is heterothallic and fusion occurs between opposite mating type sporidia and later produce dikaryotic hyaline secondary sickle-shaped sporidia which in turn produce infectious hyphae. Both the pathogens have a similar life cycle and may occur together in the same plant.

Upon germination of teliospores (present on the contaminated seed or in the soil), the infectious hyphae enters in the young plant at the tillering stage. The mycelium grows inter and intra-cellularly, colonizes the ovaries and transforms the grain into a mass of teliospores (Line 1993). The life cycle is presented in (Fig. 3.2) (See Wilcoxson and Saari 1996).

Control

Seed treatment with some systemic fungicides like triazole may provide complete control of this bunt. However, strict quarantine measures including seed assays are the best procedures to check the spread of the disease from one country to another.

According to Matanguihan and Murphy (2011), common bunt has re-emerged as a major disease in organic wheat. In conventional agriculture, common bunt is

managed with the use of chemical seed treatments, however, since synthetic chemicals are prohibited in organic agriculture, common bunt is a major threat once more in organic wheat. Matanguihan and Murphy (2011), have given a review on management of common bunt under organic farming systems, mainly though host resistance. It also includes studies on the physiological and molecular basis of host resistance. The reader is encouraged to refer to this comprehensive and up-dated review on common bunt of wheat.

3.1.2 Karnal Bunt

Karnal bunt is also known as partial bunt. It was first reported on wheat from Karnal district in northern India (Mitra 1931; Bedi et al. 1949). Thereafter, the disease appeared on a larger scale in 1996, 1978–1979, 1981–1983 and 1986, in different parts of India as well as Pakistan (Joshi et al. 1983; Agarwal 1986; Singh and Dhaliwal 1989). In Mexico the disease occurred in 1982 and 1983 and later in severe proportions in the Valle de Yaqui (Warham 1986). A good revision of literature was presented by Joshi et al. (Joshi et al. 1983). The disease is also reported in other countries like Nepal, Afghanistan and Iraq (Zang et al. 1984). Karnal bunt occurs on *Triticum aestivum*, *T. turgidum* and *X. Triticosecale* (Agarwal and Verma 1979). Yield losses caused by this disease are very low. Yield losses between 5 and 20 %, have been reported but normally the losses may vary between 0.2 and 0.5. *Tilletia indica* differs from the other bunts like *T. foetida*, *T. caries*, *T. controversa* and *Urocystis agropyri*, in that the pathogen infects during anthesis and sporulates during the same growing season of the host. Because of the color and bad odour, only 1–4 % infected kernels may render the wheat grain unacceptable for human consumption (Joshi et al. 1983; Wilcoxson and Saari 1996).

Symptoms

The symptoms of Karnal bunt are somewhat different from those produced by other bunts. Only a few grains per spike are infected and hence it is referred as partial bunt (Fig. 3.3). Infected kernels have a bad odour because of the production of trimethylamine. Initially, the infected part of the grain looks dark brown later becoming black (Fig. 3.4).

Causal Organism and Epidemiology

Karnal bunt is caused by *Tilletia indica* (Mitra) Mundkur (Syn. *Neovossia indica*). The teliospores are twice as large as the other *Tilletia* spp. and measure 22–49 μm (Fig. 3.4b, c). According to Bonde et al. (1989), the teliospores of *T. indica* can sometimes be confused with those of *T. barclayana*—a rice pathogen. Four pathotypes of *T. indica* are reported to occur (Aujla et al. 1980, 1985; Carris et al. 2006).

Fig. 3.3 Wheat spike infected with *T. indica* (Courtesy: CIMMYT)

The teliospores of *T. indica* have three layers around their cell wall: periospore, epispore and endospore. Teliospores can remain viable in the laboratory for 5–7 years. They germinate at a variable temperature of 5–30 °C after 1 h of incubation. Upon germination of teliospore meiosis occurs and promycelium and primary sporidia are produced. The sporidia germinate to form hypha or secondary falcate sporidia. The secondary sporidia infect the ovary at the boot stage or at the flowering time and the fungus develops in the ovary and finally transforms the grain partially with sori of teliospores. According to Wilcoxson and Saari (1996), the teliospores are disseminated during the harvest and cause infection to other spikes, thus completing the life cycle of the pathogen.

Control

Several cultivars of wheat and related species have been found to be resistant to *T. indica*. Besides, several breeding lines originating from India, Brazil and China have been found resistant (Wilcoxson and Saari 1996).

Cultural practices like crop rotation are not efficient in controlling the disease (Singh and Dhaliwal 1989). Bedi et al. (1949) reported that the incidence of Karnal bunt was higher in fertile soils with artificial irrigation. Since the pathogen survives

Fig. 3.4 (**a**) Wheat kernels infected with *T. indica*; (**b**, **c**) teliospores and teliospore germination of *T. indica* (Courtesy: CIMMYT)

in the soil for 4 years, no crop rotation could be effective. Similarly, several systemic fungicides have been tested, but no conclusive evidence of their efficiency has been reported.

The disease can be kept at low levels through different management practices such as: (a) Seed multiplication in areas free from the disease; (b) Fungicidal seed treatment for partial control; (c) Rigorous seed inspection (Warham 1986; Mehta 1993).

Avoidance of contaminated seed is of course the best way to control the disease. Thirumalaisamy et al. (2011) have developed specific primers for detection of Karnal bunt pathogen in wheat seed.

Dutt et al. (2011), reported that jasmonic acid (JA) may act as a potential activator of induced resistance against Karnal bunt of wheat by up-regulating cystain gene expression (Purwar et al. 2012).

3.2 Ergot

Ergot has been known to occur in cereals for the last two centuries. Ingestion of ergot alkaloids in grain provokes toxic effects in human beings and animals. The disease occurs in cultivated cereals and grasses.

Ergot can cause 5–10 % yield loss in small grain cereals and forage crops. Wegulo and Carlson (2011), has given a good account on the consequences of this disease including information on the health effects of ergot on human being and the animals.

3.2.1 Symptoms

At first, the symptoms of the disease can be identified by the presence of sugary slime or sticky yellowish droplets "honey dew" (Fig. 3.5a) exudates by infected florets. Later, the characteristics symptoms of the disease are the presence of sclerotia on spikes replacing some of the kernels. The sclerotia are horn-like, larger than the glumes and break during harvest and contaminates the grains (Fig. 3.5b). The fungus contains alkaloids in variable quantities and under severe field infestation the grain harvest becomes unsuitable for human and animal consumption (Peña 2007).

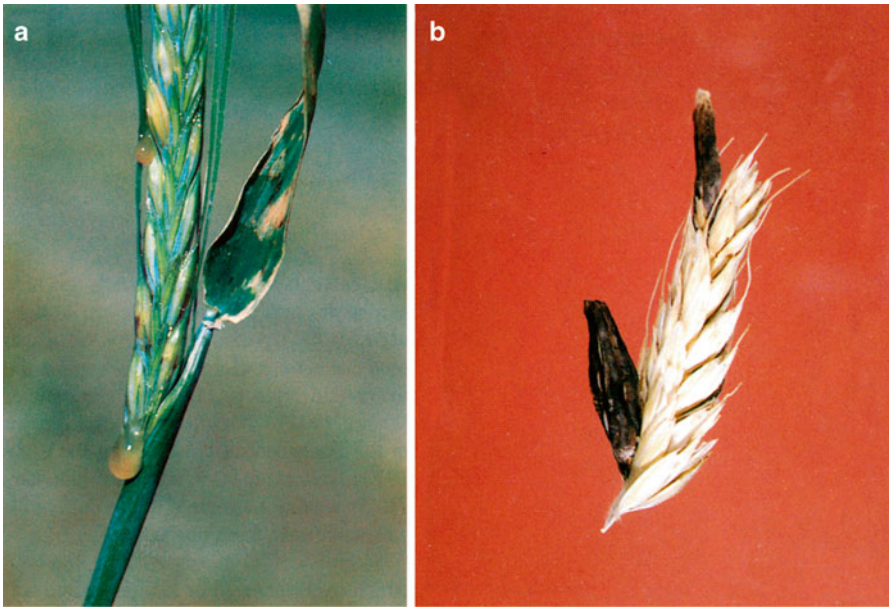

Fig. 3.5 (**a**) Honeydew stage of *Claviceps purpurea*; (**b**) sclerotia of *C. purpurea* (Courtesy: B.M. Cunfer)

3.2.2 Causal Organism and Epidemiology

Ergot of wheat is caused by the fungus *Claviceps purpurea* (Fr.) Tul. Sclerotia are black and measure 2–20 mm. On germination the sclerotia produce perithecia, asci and ascospores which in turn infect the wheat spikes and thus complete the life cycle of the pathogen. Sclerotia remain viable in the soil for at least 1 year. Other than soil infestation perithecia may also be introduced in the soil through contaminated seeds.

The conidia of the "honey dew" stage are responsible for the secondary spread of the disease. They are disseminated by wind, rain splashing and insects. The disease is favored by prolonged rainy and cool periods. Other than wheat, triticale and rye are susceptible to *C. purpurea* (Bove 1970; Darlington and Mathre 1976).

3.2.3 Control

Ergot can be controlled by using healthy seed. Cleaning of the seed is necessary especially when it originated from infested fields. The disease is more frequent in sorghum field than in wheat fields. Other than seed cleaning, crop rotation with non-host crop species will reduce severity of the disease. No other control measures are recommended because in general, the disease is not considered important.

3.3 Pyricularia Blast

For a long time Pyricularia blast has been a major disease of rice and has been intensively studied during the past 100 years. Prabhu and Fillippi (2006), have given an excellent account of this disease of rice encompassing a number of different aspects.

Pyricularia wheat blast was first reported in the northern part of the State of Paraná, Brazil, in 1985 (Igarashi et al. 1986). Soon after its first occurrence, it spread to other Brazilian States as well as to some neighboring countries. The disease was observed on barley (Yaegashi and Udagawa 1988) and was reported to be very common in ryegrass in the United States (Bain et al. 1972; Carver et al. 1972). As stated earlier a survey was conducted to find out which are the top ten fungal pathogens in the world based on scientific and economic importance (Ralph et al. 2012a). The survey generated 495 voters from the international community. In first place appeared *Magnaporthe grisea* causing blast on rice and wheat. According to Khush (2005), approximately one half of the world's population relies on rice for its primary caloric intake. Perhaps a somewhat similar situation exists for wheat because it is a staple food for some of the world's most populated countries.

At present wheat blast disease is restricted to the tropical and sub-tropical regions of South America (north-eastern Argentina, lowlands of Bolivia, central and south-

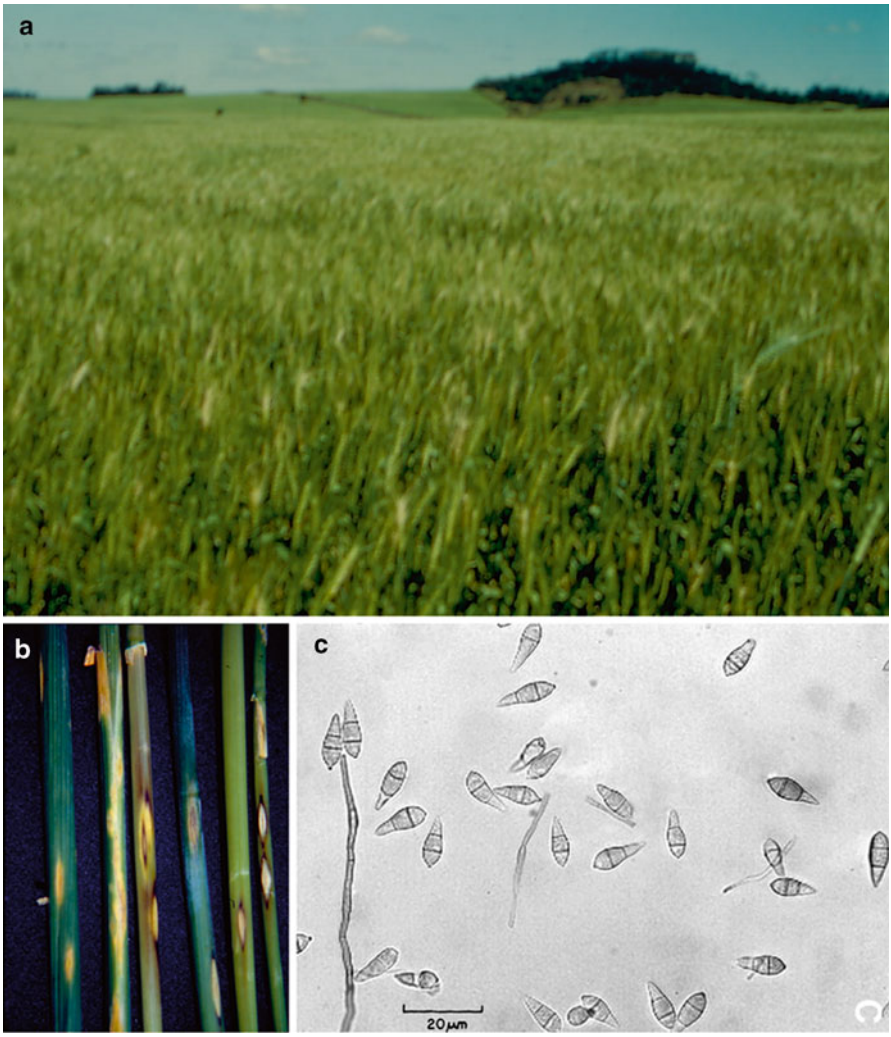

Fig. 3.6 (**a**) Heavily infested wheat field with *P. grisea* showing white (bleached) spikes; (**b**) symptoms of *P. grisea* on triticale stems; (**c**) conidia and conidiophores of *P. grisea*

central Brazil and Paraguay). However, climatic changes associated with global warming could trigger its spread to other parts of the world (Kohli et al. 2011). Contrary to rice blast, wheat blast is basically a spike disease however, in highly susceptible cultivars leaf infections can at times be observed. In triticale and oats the disease can be observed on leaves and culms (Fig. 3.6a, b). In oats it is basically a leaf disease like rice blast but in susceptible cultivars it can attack rachis and the glume at its base.

The grain yield losses caused by Pyricularia blast can vary from very low to almost 100 %. Highest losses occur when the fungus attacks the rachis at the base of the spike affecting total or partial grain filling depending upon the time of infection. In 1987, the weather was dominated by the El Niño climatic phenomenon and yield losses incurred by the three Brazilian wheat producing states (Paraná, Mato Grosso do Sul and São Paulo) varied between 10.5 and 53 % (Goulart and Paiva 1992). During the same year, the disease also appeared in the traditional wheat region of Rio Grande do Sul causing variable losses (Piccinini and Fernandes 1990). In 2009, excessive rains during the critical wheat period in the North and West region of the State of Paraná, Brazil, provoked heavy incidence of scab and Pyricularia blast.

While Cunfer et al. (1993) observed the disease in the border region of Brazil/Paraguay in 1987, the first epidemic in Paraguay occurred in 2002, causing production losses of more than 70 % in the early seeded fields (Viedma and Morel 2002). Most of the harvested grain did not meet commercial standards for test weight and had to be used for animal feed.

The first severe infections of wheat blast in Bolivia were observed in the lowland Santa Cruz region in 1996 and resulted in a loss of almost 80 % of the production (Barea and Toledo 1996; Kohli et al. 2011). Argentina reported its first blast infections and associated losses in a summer seeded wheat experimental crop in the north-eastern state of Chaco in 2007/2008 (Alberione et al. 2008).

3.3.1 Symptoms

The disease symptoms on spikes are somewhat similar to those caused by scab (*Gibberella zeae*) and can easily be confused with scab (Figs. 3.7 and 3.8). Rather than attacking individual spikelets, wheat blast attacks the rachis. The portion of the spike above the point of infection becomes bleached and no grains are formed, whereas the portion below remains healthy and produces normal grains. On wheat leaves the symptoms are not common but in black oat cultivars they are very common and are characterized by elliptical lesions with dark-brown margin (Fig. 3.9). On oats the pathogen also attacks rachis at different points and the whole spike becomes bleached. Abundant sporulation of the fungus on the rachis and at the base of the glumes can be observed. The base of the glume looks dry, strangulated and dark brown in color. Abundant sporulation of the pathogen can be observed on these lesions (Fig. 3.9c, d). While the rice blast fungus mainly attacks the leaves, the wheat blast fungus mainly attacks spikes. Heavily infected seeds when examined by "blotter test", show profuse sporulation of the fungus, although these 'so called' seeds are shriveled and are eliminated during the seed processing and are not epidemiologically important.

Fig. 3.7 (**a**) Commercial wheat field heavily infested with *Pyricularia grisea* (Courtesy: J.M.C. Fernandes); (**b**) infected spike showing blackening of rachis and a portion of spike bleached

Fig. 3.8 Comparison of symptoms of *G. zeae* (*left*) and *P. grisea* (*right*)

Fig. 3.9 (**a**) Spread of Pyricularia blast from severely infested black oat field of cv. IAPAR 61 to the adjacent wheat field showing 100 % infected spikes; (**b**) symptoms of Pyricularia blast on leaves of white oat; (**c**) infected and healthy panicles of white oat; (**d**) sporulation of *P. grisea* at the base of glume of white oat cv. IAC 7

3.3.2 Causal Organism and Epidemiology

Pyricularia grisea (Cooke) Sacc. [telemorph *Magnaporthe grisea* (Herbert) Barr]. *P. grisea* is a well known pathogen of rice and is a very highly variable. The pathogen that attacks rice is different from the one that attacks wheat (Valent and Chumley 1991; Mehta 1993; Urashima et al. 1993, 2004a, b). The fungus can be easily cultivated on common culture media, but for abundant sporulation oatmeal-agar medium

is considered more appropriate (Prabhu et al. 1992). The conidia are borne on short conidiophores singly or in groups of 2–3, pyriform, obclavate, hyaline and with one or two septa (Fig. 3.6c). The size of the conidia varies a lot but in general they measure 23 × 17 μm (Agarwal and Mortensen 1989).

Seed transmission of the wheat fungus has been shown by Goulart and Paiva (1990). However, as mentioned earlier, seed infection seems to play little or no role in the epidemiology of the disease because the pathogen is not systemic in nature and spike infection comes from the air-borne conidia. The air-borne conidia originating from several different hosts are splashed by wind and rain on the rachis of wheat spike, germinate in a few hours and cause infection. In just a few days profuse sporulation occurs on the rachis at the point of infection, the spores are transported by rain and wind to other plants and other fields and thereby the pathogen continues its life cycle.

The primary infection may come from several secondary hosts (Prabhu et al. 1992; Urashima et al. 1993, 2004a, b). Several grass weeds (*Cenchrus echinatus*, *Eleusine indica*, *Digitaria sanguinalis*, *Brachiaria plantaginea*, *Echinocloa crus-galli*, *Pennisetum setosum*, *Hyparrhenia rufa* and *Rhynchelytrum roseum*) occur commonly in wheat and rice fields of Brazil and are secondary hosts of *Pyricularia* but their role in the epidemiology of wheat blast is not well understood (Prabhu et al. 1992). In Bolivia, *Eleusine indica*, *Digitaria sanguinalis* and *Rottboelia exaltata* have been identified with blast symptoms (Hurtado and Toledo 2005).

Blast infection in commercially grown black oats (*Avena strigosa*) has also been reported quite recently (Mehta et al. 2006). Black oats were considered resistant until the commercial cultivation of this species was attacked by *P. grisea* throughout the State of Paraná (Brazil), in the year 2004. The Pyricularia blast spread from severely infested black oat fields to the adjacent wheat field of cv. CD 103 (Mehta et al. 2006). In the year 2012, one of the white oats cultivar IAC 7 was found severely attacked for the first time, probably by a new biotype of *P. grisea*, under natural field conditions in Assis, in the State of São Paulo (Fig. 3.9b, c d,). The breakdown of resistance of white oats is of the utmost importance since this crop was the only resistant source of *P. grisea* among the cereals (Marangoni et al. 2013). Planting white oats during winter is one of the best options in crop rotation with wheat. As mentioned elsewhere, it is not yet known if all the white oats cultivars are susceptible to this biotype. Screening all the available white oats cultivars for R gene sources is imminent. The first report of blast on triticale (*X. Triticosecale*) was made by Mehta and Baier (1998). According to these authors, Pyricularia blast was not found before 1998 in commercial fields of triticale in Brazil. Black oats and foxtail millet (*Setaria italica*) are widely used in the crop rotation system in the Southern Cone region and their susceptibility to *P. grisea* (Fig. 3.10) is a matter of concern (see chapter on crop rotation).

Prabhu et al. (1992) reported that all of the *P. grisea* isolates from rice, wheat and grass weeds were pathogenic on the wheat cultivars and barley, but none of the 10 wheat and 7 grass isolates infected any of the 30 rice cultivars. Similarly Mehta and Baier (1998) reported variation for virulence and host specificity among *P. grisea*

Fig. 3.10 Symptoms of *P. grisea* on leaves and spike of foxtail millet (*Setaria italica*)

isolates from triticale. In this study, isolates from triticale were aggressive on triticale and oats, less aggressive on wheat and not compatible with rice, indicating that the wheat and the rice pathotypes are distinctly different in origin. The leaf reaction of some wheat and oat cultivars to a mixture of *P. grisea* isolates from black oats is presented in Tables 3.1, 3.2, 3.3 and 3.4. The wheat cv. BRS 229 has been showing reasonable level of resistance under field conditions for several years, but its susceptibility to mixture of oat isolates, like the triticale isolates, suggests a different origin for the oat isolates (see Table 3.4).

Although exact weather conditions required for a field epidemic are not clear, most severe blast years have coincided with wet years. These are characterized by several days of continuous rains and average temperatures between 18 and 25 °C during the flowering stage of the crop followed by sunny, hot and humid days.

Table 3.1 Differential response of black oat (*Avena strigosa*) cultivars artificially inoculated with *Pyricularia grisea* isolates from black oats

Cultivar	Average severity index[a]	Grouping category (Scott and Knott 1974)
EMBRAPA 140	1.0000	A
EMBRAPA 139	1.0000	A
EMBRAPA 29	0.9583	A
SI 90045	0.8333	A
IA 03187	0.7917	A
SI 83002	0.7917	A
SI 90173	0.7083	A
IAPAR 61 (susceptible check)	0.7083	A
IA 03687	0.6667	A
SI 83400	0.6667	A
PRETA COMUM	0.6250	A
IA 00887	0.6250	A
CTC 88P16	0.6250	A
SI 0061-USA	0.6250	A
SI 90112	0.6250	A
IA 01587	0.5833	A
IPR 126 (resistant check)	0.0000	B

[a]Disease severity scale of 0–1 (Zadoks 1972). Disease rating was done 7 days after inoculation in the glasshouse

Table 3.2 Differential response of white oat (*Avena sativa*) cultivars artificially inoculated with *Pyricularia grisea* isolates from black oats

Cultivar	Average severity index[a]	Groping category (Scott and Knott 1974)
IAPAR 61 (susceptible check)	0.7083 A	A
FAPA 4	0.1667 B	B
IAC 7	0.1667 B	B
UFRGS 14	0.1250 B	B
UFRGS 19	0.1250 B	B
URS 22	0.1250 B	B
UPF 16	0.0833 B	B
UFRGS 991200211	0.0417 B	B
ALBASUL	0.0417 B	B
UPF 15	0.0417 B	B
UPFA 20	0.0417 B	B
URS 20	0.0417 B	B
UPF 18	0.00 B	B
UPFA 22	0.00 B	B
URS 21	0.00 B	B
URS GUAPA	0.00 B	B
IPR 126 (resistant check)	0.00 B	B

[a]Disease severity scale of 0–1 (Zadoks 1972). Disease rating was done 7 days after inoculation in the glasshouse

Table 3.3 Pathogenic variation between the *Pyricularia grisea* isolates from black oats (*Avena strigosa*), on some wheat and oat cultivars

	Blast severity 7 days after inoculation in wheat and oat cultivars[a]			
	Wheat cultivar		Oat cultivar	
Isolates	CD 103	BR 18	IAPAR 61	IPR 126
15,700	0.08	0.08	0.75	0.08
15,701	0.58	0	0.33	0
15,714	0.33	0	0.5	0
15,715	0.83	0	0.33	0
15,718	0.58	0	0.33	0.08
15,720	0.58	0	1	0
15,721	0.5	0	1	0.42
15,722	0.67	0.08	0.92	0
15,723	0.08	0	0.58	0
15,724	0.33	0.08	1	0.25
15,725	0.58	0	0.92	0
15,726	0.17	0.42	1	0.67
15,728	0.25	0.25	0.42	0.08
15,730	0.58	0.58	1	0.25
15,732	0.92	0	1	0.17
15,741	0	0	0.25	0.17
15,742	0.75	0.25	0.83	0.08
15,749	0.75	0.58	0.75	0
15,756	0.58	0	1	0.58

[a]Average of three replications. Disease severity scale of 0–1 (Zadoks 1972). *Source*: Marangoni et al. (2013)

Table 3.4 Differential response of wheat cultivars to infection caused by mixture of five *P. grisea* isolates from black oats, under glasshouse conditions

Cultivar	Average severity index[a]	Grouping category (Scott and Knott 1974)
BRS 229[b]	0.9163	A
ONIX	0.89	A
BRS 249	0.89	A
BR 18	0.743	B
IPR 118	0.6868	B
CD 114	0.612	B
CD 108	0.612	B
BRS 248	0.4073	C
BRS 220	0.4073	C
BRS 193	0.2367	D

[a]Severity scale of 0–1 (Zadoks 1972)
[b]Always showered good level of field resistance
Source: Marangoni et al. (2013)

3.3.3 Control

Identification of genetic resistance sources in wheat is more difficult, due to wide virulence diversity in the fungus (Urashima et al. 2004a, b). Twenty-seven cultivars have shown moderate resistance in evaluation made under natural field conditions

of infection (EMBRAPA 2011). New Bolivian cultivars such as Parapeti-CIAT has also shown some level of resistance. Some Brazilian cultivars like BR 18 and CD 103 (having resistance from cv. Milan (see Table 3.3)) have shown high levels of resistance over the years. As said earlier, the oat isolates have different origin from the wheat isolates and perhaps for this reason the cv. BR 18 showed a susceptible reaction (Table 3.4). Recently, several cultivars and advanced lines derived from the CIMMYT line, Milan, have been observed to carry a very high level of resistance to blast disease in the endemic region. While the genetic basis of resistance in Milan has yet to be studied, Mehta et al. (2001) reported that leaf resistance of the cv. OR1 was based on a single recessive gene. The increased area under Milan-based resistant wheat cultivars such as Sausal CIAT, CD 113, CD 116 and Caninde 1, released in Bolivia, Brazil and Paraguay, respectively, need to be combined with other sources urgently to prevent the selection of a virulent pathotype in the fungus (Kohli et al. 2011). According to recent studies conducted in Brazil, some cultivars derived from Milan like, Milan CD 116, Milan3/Atila/Cimmyt 3 and other cultivars like, BRS 210 and BRS 229 were found to be highly resistant under field conditions (Marangoni et al. 2013).

Besides genetic resistance, avoidance of early dates of seeding helps reduce the disease severity (Mehta et al. 1992; 1993). Fungicidal seed treatments help in eliminating the seed-borne infection but do not protect the plant from spike infection. Fungicides combining triazols with strobilurins have been used with only a little success at the heading stage to control the disease, especially in the moderately resistant varieties (Kohli et al. 2011). However, in general, fungicide applications on susceptible and moderately susceptible cultivars have not resulted in good control of the disease and hence are not cost-effective.

The pathogen is highly variable and other than cv. Milan, so far highly resistant sources in wheat are not available, although some white oats cultivars as mentioned earlier, are found highly resistant to this disease (Table 3.2).

Genetic variability in the pathogen was also reported in the USA. Recently, University of Kentucky, UK college of Agriculture Research Scientist Lloyd Murdock found wheat blast caused by *P. grisea* for the first time in the USA on a single wheat head in 2011 in a research plot in Princeton. According to a report (NEWS) presented by Kaite Pratt of the Agricultural Communication Services, UK College of Agriculture, no additional instances of the disease were found even after extensive scouting of the involved research plots and neighboring fields. According to this report, the rice blast fungus does not infect wheat and vice versa. Katie Pratt further informed that the genetic structure of UK wheat blast was different from the blast fungus occurring on wheat in South America and hence it is not an exotic pathogen. According to the recent report of 2012 of the Dept. of Plant Pathology, Ohio State University Extension, OARDC researchers in the USA are using tools of molecular genetics and genomics for the development of resistant varieties for rice and other crop plant. According to this report, Guo-Liang Wang and researchers have determined that the AvrPiz-t effector from the fungal pathogen *M. oryzae* has both avirulence and virulence functions and suppresses the host ubiquitin proteosome system during infection. In addition, the Mitchell and Wang laboratories found that the AvrPi-t gene is present in the two sequenced wheat blast strains from South

America, suggesting the resistance gene Piz-t might be effective against wheat blast (Guo-Liang et al. 2012).

An integrated management of the disease especially by combining less susceptible cultivars and avoidance of early seeding, have been successfully implemented to reduce yield losses in the endemic regions.

In recent years, emphasis has been laid on the disease forecasting by computerized mathematical models (see chapter on Disease forecast modeling). Through such models it is possible to provide estimates of disease likelihood and forecast outbreaks which in turn avoid unnecessary fungicidal applications. Computer modeling thus may become a part of the integrated disease management systems in the near future (Fernandes et al. 2005, 2011).

Other than the disease forecasting modeling, resistance of white oat cultivars suggests that some specific resistance genes are operating in such oats. However, it remains a challenge to transfer the resistance genes of oats to wheat. It is believed that the modern technologies for gene transfer including embryogenesis, transformation techniques, sequencing, etc. may come up with some exciting solutions.

3.4 Scab—The Fusarium Head Blight

The Fusarium head blight (FHB) also known as wheat scab, has become one of the most important diseases of cereals including wheat and barley. Worldwide re-emergence of FHB and its occurrence in severe epidemic forms in the nineties, left some of the largest wheat producing countries in turmoil (McMullen et al. 1997; Leonard and Bushnell 2003; Ralph et al. 2012b).

Similar to Pyricularia blight, Fusarium blight, received one of the top priorities for scientific research (Ralph et al. 2012b). Losses in yield are quite variable. Heavy yield losses have been recorded in several countries (Chester 1950; Mehta 1993; Nicholson et al. 2007). According to Nicholson et al. (2007), in Britain for example, the estimated yield losses were of 50–60 %, whereas in Paraguay they were as high as 70 %, during 1972–1976. In the USA and in Canada, the monetary losses were US$ 3 billion and US$ 220 million, in the epidemic years 1991 and 1996, respectively. An excellent account of scab epidemics and losses caused in wheat and barley in the USA and Canada has been presented by McMullen et al. (1997). Scab is endemic in China where heavy yield losses were reported in epidemic years (Cook 1981a, b).

In China, FHB is a newly expanded disease. According to Yang and Lu (2012), the epidemic area has expanded to the major wheat production, including the Yellow and Huai River Valleys Wheat Zone and Northern Winter Wheat Zone. Lu and Chen (2012) reported that in China, severe FHB epidemic occurred in 2012 in southern Shandong, Henan, Shanxi and Hebei provinces and northern Jiangsu, Anhui and Hubei provinces, due to the lack of resistant cultivars in these areas and due to the increase in acreage with no-tillage cultivation. Similarly, in Sweden and Norway, the

increased occurrence of mycotoxin producing *Fusarium* spp. among other reasons, seems to be associated with increased use of reduced tillage (Brodal et al. 2012).

In Brazil, the losses are reported to reach 75 % (Mehta 1993). Severe epidemics of FHB occurred during the years 1976, 1983, 1986, 1987 and 2010. In 1987 yield losses in the State of Rio Grande do Sul were estimated to be 274 kg/ha (Reis 1986; Casa et al. 2004). In Argentina losses in yield varied between 5 and 30 % depending upon the year, the wheat region and the cultivar (Galich 1989; Moschini and Fortugno 1996). In Uruguay the disease caused about 80 % yield losses during 1977–1985 (Torres 1989).

Other than yield losses, infected grains are low in test weight, low in quality and high in mycotoxin vomitoxin (deoxynivalenol) harmful for human health and animal feed, which further reduce marketability and price (Peña 2007). As a result, many producers are penalized with huge discounts. Scabby barley is also not accepted on the market by malters. According to (McMullen et al. 1997), during severe epidemic years in the USA, many farmers had to decide in conjunction with the Federal Crop Insurance representatives and farm Service Agency agents, to either destroy the crop in the field or to try to salvage some yield. These authors reported that approximately 18 % of the wheat acreage in northwestern Minnesota was not harvested due to heavy incidence of scab in 1994.

3.4.1 Symptoms

Symptoms of the disease are noticed at the time of spiking. The pathogen attacks individual spikelets or the whole spike and as mentioned elsewhere in this book, the symptoms can be confused with Pyricularia blight. One of the differences is that the Pyricularia blight fungus does not attack individual spikelets (see chapter on *Pyricularia blight*). The blighted spikes look bleached and almost white and in humid conditions pink masses of spores can be seen on or in between the spikelets (Fig. 3.11b). These pink masses of conidia are not observed with Pyricularia infections, instead, the conidial masses of *Pyricularia* on the rachis are of ash color. In cases of severe infections the whole crop looks green but with white spikes.

FHB affects grain yield quality. When infections occur early, the grains look pinkish and shriveled (Fig. 3.12). Such infected grains contaminate other grains during the storage especially when the grain has high moisture content. In general, the shriveled grains being lighter in weight are eliminated by the combine during harvesting and by the gravity table in the seed processing unit. Infected seed show variation in color and shriveling. Infected seeds give rise to infected seedlings or death of the seedlings.

Black hard perithecia are frequently observed on infected spikes at the time of harvest (Fig. 3.11c, 3.13b, c). They are also observed abundantly on the crop residue of wheat and corn (Bergstrom 1993). According to Khonga and Sutton (1988), maize and wheat debris infested with *G. zeae* which had overwintered for 2 years, served as a source of infection for wheat in Ontario, Canada.

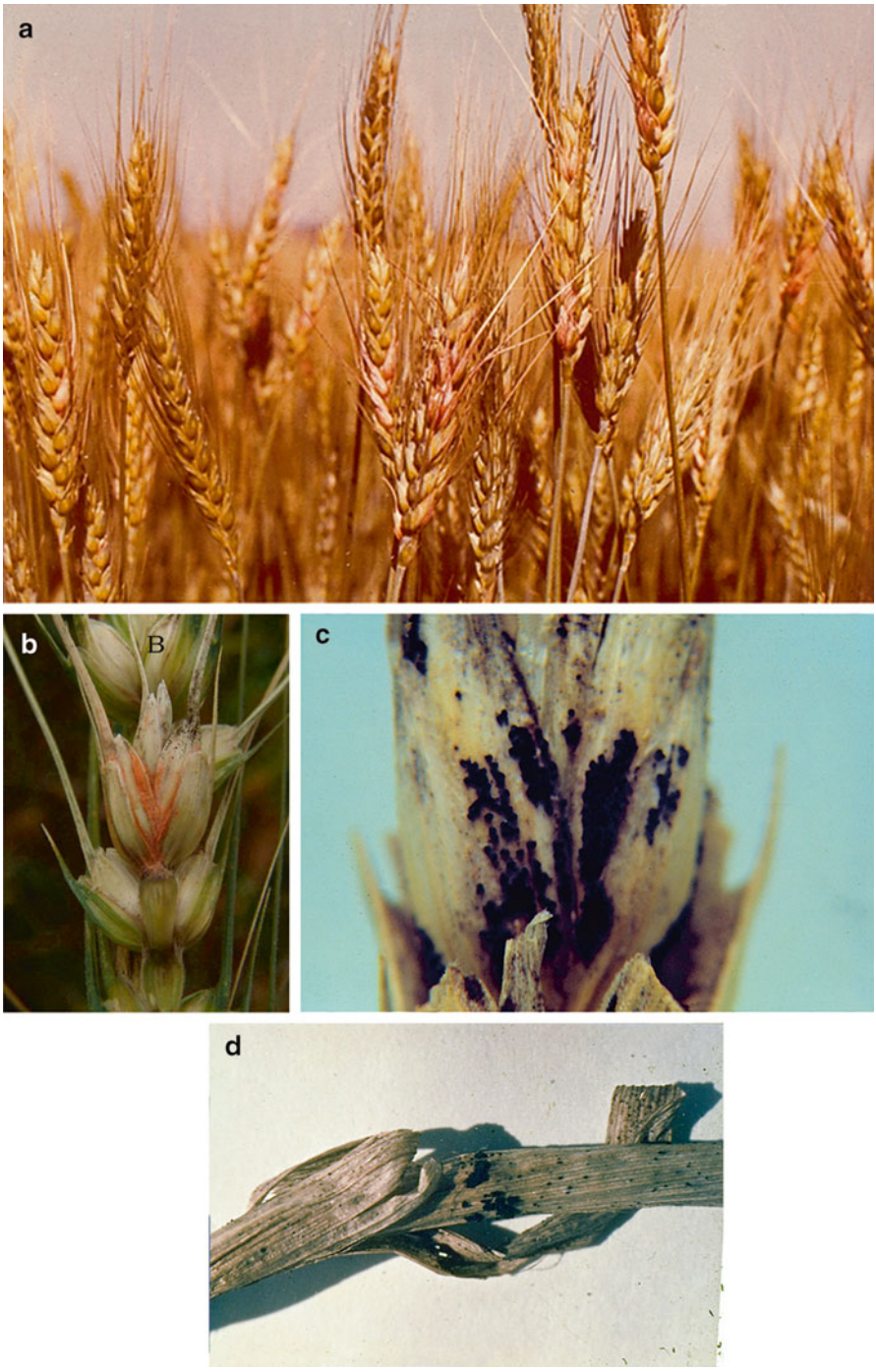

Fig. 3.11 Scab-the Fusarium Head Blight. (**a**) Infected spikes in the field; (**b**) spikelet showing pinkish mass of conidia of *Fusarium graminearum* (Courtesy: J.M.C. Fernandes); (**c**, **d**) perithecia of *Gibberella zeae* on spike and leaf sheath

Fig. 3.12 Wheat kernels infected with *F. graminearum* showing pinkish discoloration (Courtesy: P.D. Hewett)

3.4.2 Causal Organism and Epidemiology

FHB is caused by *Fusarium graminearum* Schwbe (Syn, *F. roseum* LK emed Snyder and Hans. f. sp. *cerealis* (CKe.) Snyd and Hans. Cv. "*Graminearum*"). (Syn. *G. roseum* f. sp. *cerealis* "*graminearum*"), telemorph *Gibberella zeae* (Schw.). Petch. Two varieties of *Microdochium nivale* like *M. nivale* var. nivale and *M. nivale* var. *majus* are also known to cause FHB (Nicholson et al. 2007).

The perfect stage *G. zeae* can be observed in nature on the infected spikes and on the stems (Fig. 3.13b, c). The macroconidia of the imperfect stage *F. graminearum* are produced in phialides on ramified conidiophores. Microconidia are absent whereas clamydospores are observed in artificial culture media. The macroconidia are curved, sickle-shaped, 3–7 septate, the basal cell is foot-shaped and measure 25–62 × 2.5–5.0 µm. On common artificial culture media (PDA) the fungus grows well with abundant sporulation at approximately 25 °C. The aerial mycelium is dense, yellow to tan at the margins and carmine red at the center. The asci and ascospores are produced in hard, black oval perithecia which measure 140–350 µm in diameter. The asci are clavate, bitunicate, contain 4–8 ascospores and measure 60–85 × 8–11 µm. The ascospores are hyaline, slightly curved with rounded ends, three septate and measure 17–25 × 3–4 µm (Bergstrom 1993; Mehta 1993).

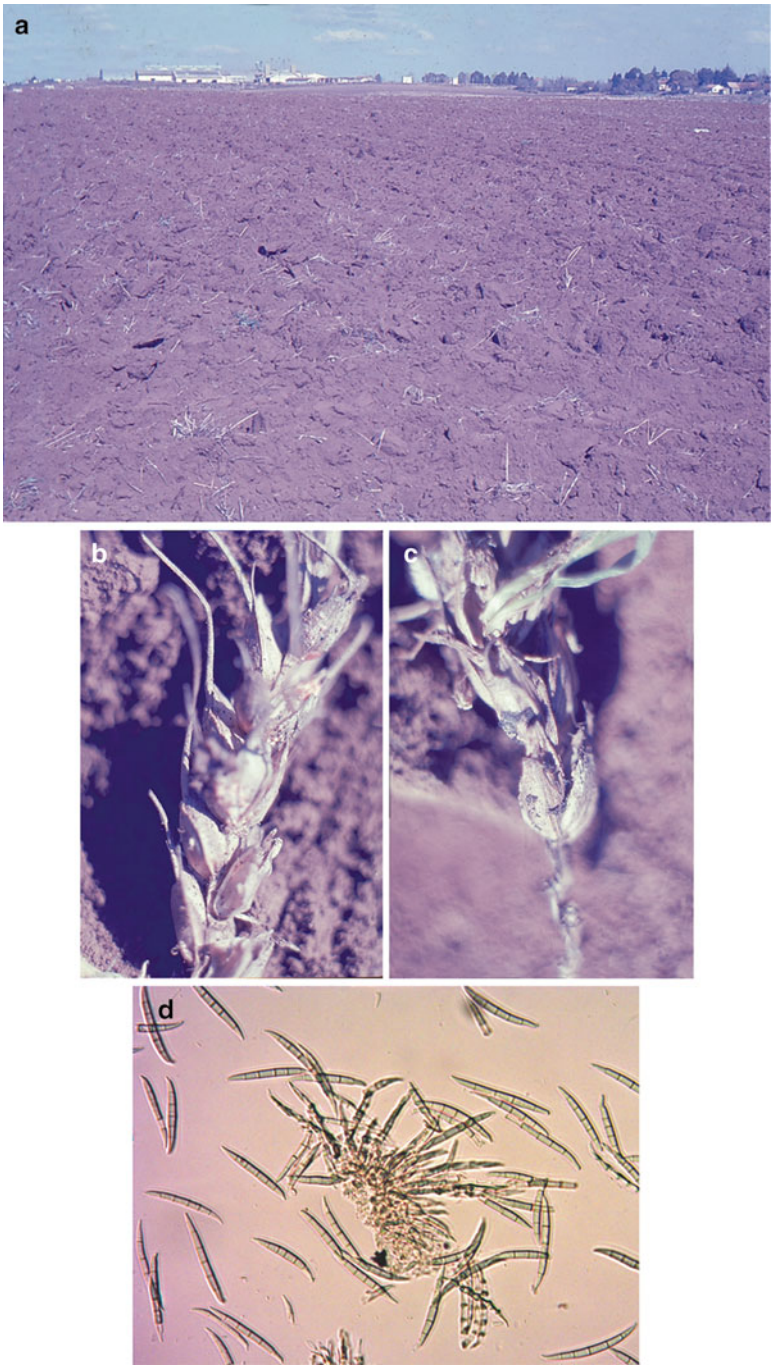

Fig. 3.13 (**a**) Wheat field with crop residue; (**b**, **c**) survival of perithecia of *G. zeae* on left-over wheat spikes in the field; (**d**) sporodochium and conidia of *Fusarium graminearum*

F. graminearum produces several vomitoxin (trichothecene) of which the most important mycotoxin is deoxynivalenol (DON). *F. graminearum* is considered to be the most important one in toxin production. FHB can be caused by several species of *Fusarium* like *F. graminearum, F. culmorum, F. avenaceum, F. poae* and *F. nivale* (*Calonectria nivale*). The last of these can be important in countries or part of countries where cooler climate predominates (Nicholson et al. 2007; Bergstrom 1993). However, *F. graminearum* is still considered to be the major pathogen in the FHB syndrome. Recently, *F. graminearum* was seen to consist of seven lineages (O'Donnel et al. 1999; Carter et al. 2000; Nicholson et al. 2007; Zhang et al. 2012). In the USA and Canada, for example, severe measures are taken to limit the grain contamination by vomitoxin. In the USA it is 1 mg/kg in wheat food products (Mathur and Cunfer 1993).

Both *F. graminearum* and *G. zeae* can cause infection to wheat spikes. *F. graminearum* infects roots and spikes but the infection is not systemic. Spike infections can occur at any stage of development between flowering period up through the soft dough stage, but the most damaging stage of infection is the flowering. At this stage the conidia blown by wind and rain splash get deposited on the spike (florets), soon germinate and infect the grain at its initial formation.

Optimal conditions for infection are warm and humid weather followed by several days of rain. Temperatures of 22–25 °C are most favorable for infection and disease development. When the infection occurs at the flowering stage, the conidia germinate and infect the ovary. In this case there is no formation of grain. Infections occurring after the grain formation are external infections and the seed serves as a source of primary infection. The disease affects different parts of the spike. When the rachis is infected at different points, the entire spike show bleached appearance. At the time of harvest, the conidial mass contaminates the healthy seed and can even cause significant damage during the storage especially when the moisture content of the seed is more than 12–13 % (Telles Neto et al. 2007).

Other than the seed, the conidia and the perithecia can exert an important role in the epidemiology of the disease. They are produced on the wheat crop residue and can remain viable for a long period of time or until the next wheat crop is sown in (Fig. 3.13d). The perithecia produce asci and the ascospores, which in turn infect the wheat roots and/or are blown by wind and infect the spikes of wheat grown in the nearby fields. The ascospores do not travel long distances whereas conidia produced on crop residue can travel long distances and infect wheat spikes and complete the life cycle of the pathogen (Fig. 3.14).

By and large, conidia produced on crop residue or on other hosts play the key role in the epidemiology of the disease. It must be remembered that the pathogen attacks several other hosts like maize, sorghum, triticale, rye, barley and other Gramineae hosts. Reis (1985) reported several collateral hosts such as *Brachiaria plantaginea*, *Pennisetum purpureum*, *P. cladestinum*, *Digitaria sanguinalia*, *Paspalum* spp. *and Ropogon bicornis* and *Elyanthus* spp., which is interpreted as the survival mechanism of the pathogen. Infection of other non-host crops like soybean is also known to occur.

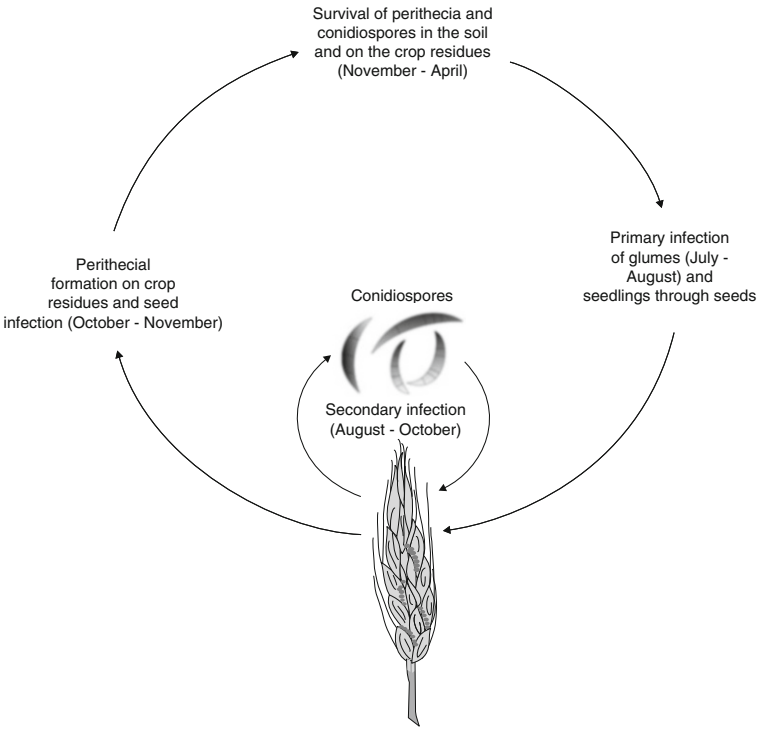

Fig. 3.14 Life cycle of *G. zeae*

The re-emergence and constant increase in severity of scab is also attributed to the increase in area with no-tillage cultivation as well as to the substantial increase in area with maize (Muhovski 2012). Cultivation of maize throughout the year (cultivation during summer as well as in winter in some countries like Brazil) is problematic.

Since the pathogen attacks maize and also survives on its left-over stubble, the inoculum of *F. graminearum* and of its perfect stage *G. zeae*, remains in the air throughout the year. The concomitant increase in acreage with no-tillage and the cultivation of two crops per year of maize help in building up scab epidemics year after year.

3.4.3 Control

Crop rotations in no-tillage cultivation may help in reducing the density of inoculum but will not have much impact on controlling the disease since the infection of the spikes is through the air-borne conidia at the time of spiking and flowering. Under the conventional system of cultivation crop rotation with non-cereal hosts

and deep plowing of left-over stubble of the previous cereal crop would help to some extent. Tillage rotation with deep ploughing in severely affected areas, perhaps once in 2 or 3 years, may be a feasible alternative to continuous no-till cultivation. As mentioned elsewhere for tan spot, this kind of tillage rotation is already practiced by some farmers in Argentina.

Seed treatment with an appropriate fungicide would eliminate the seed infection and help guarantee a uniform stand in the field. However, the disease is not satisfactorily controlled with the use of aerial applications of fungicides (Reis 1986). There are some sporadic reports about success in fungicidal applications, but by and large, successful results have not been obtained by the majority of farmers during the past several years especially in the Latin-American region. This may be attributed to the constant presence of inoculum, favorable weather conditions at the time of spiking and flowering, lack of highly resistant cultivars and lack of exact timing of fungicidal applications which it is not always possible to follow (Paul et al. 2010). Nonetheless, 2–3 applications with a mixture of systemic and non-systemic fungicides made during spiking and flowering period may offer reasonable disease control. In such a case, disease forecasting models after its appropriate validation would help to a great extent (Fernandes 1997; Fernandes and Pavan 2002; Fernandes et al. 2004; Fernandez et al. 1993; Fedak et al. 2007).

For the aforesaid reasons, in recent years emphasis has been directed towards the control of FHB through genetic resistance. It has long been known that the Chinese, Japanese and Brazilian wheat cultivars like Sumai 3, Nyu Bay and Frontana, respectively, carry resistance to FHB pathogen. It was believed that these cultivars carried different genes for resistance to FHB fungus (Van Ginkel et al. 1996). The resistance is governed by QTL.

F. graminearum produces the trichothecene toxin deoxynivalenol (DON) which is a protein biosynthesis inhibitor and has been shown to be a virulence factor. (Desjardins 2006). As stated earlier, most of the resistance to FHB of wheat comes from Chinese germplasm. According to Lu and Chen (2012), resistance genes different from those of Sumai 3 and Wangshubai have been transferred from wheat relative species into common wheat. These authors further reported that, new genetic resources with high FHB resistance and high yield have been obtained by using somaclonal variation in tissue culture combined with addition of mycotoxin selection pressure in the medium.

In the USA Mergoum et al. (2012) reported that more than three decades of breeding efforts have resulted in releasing many cultivars with varying levels of resistance which are being grown in large areas in the Northern-Central plains of the USA. These include Alsen in 2000, Steele-ND in 2004, Glenn in 2005, Howard in 2006, Faller in 2007 and Barlow in 2009. Crop rotation, genetic resistance, selected fungicides applied at heading and staggering flowering dates are currently the control practices widely used in the USA (North Dakota and Minnesota) to control the FHB (Ransom 2012). Carbendazim is no more used because of resistance of the *Fusarium* spp. involved in the FHB syndrome to this fungicide.

Other than the sources of resistance in wheat, introgression of resistance from alien species seems feasible. Resistance to FHB has recently been found in acces-

sions of *Triticum* and *Aegilops*, such as *T. timopheevi*, *T. monococcum*, *T. miguschovae*. Fedak et al. (2007), reported that usable FHB resistance has been isolated from several wild species. These authors successfully transferred the resistance into bread wheat from *T. monococcum*, *T. timopheevi* and *Aegilops speltoides.* They also reported the existence of resistance in *T. miguschovae*.

The index of scabby kernels can be estimated by a simple procedure described by Bergstrom (1993). According to this procedure: (a) Collect 100 ears randomly, preferably at a set distance in a W-pattern; divide ears into healthy and symptomatic groups and record the proportion of ears that show any prematurely senescented spikelets or signs of Fusarium; (b) Randomly select ten ears from the symptomatic group and dissect the spikelets from the ears; (c) Divide the spikelets into healthy and symptomatic group, then multiply the proportion of infected ears × the proportion of infected spikelets per ear × 100 to estimate disease index.

Thus based on the index of scabby kernels a decision could be taken either to use the field for grain harvest or for animal feed.

For the similar purpose, seed health testing can be performed using the freezer blotter method for Fusarium infections as described by Limonard (1966). This method is widely accepted and is still practicable. It is described as:

(a) Surface sterilize a sample of 400 seeds for 30 s in 95 % ethanol, then for 30 s in 1 % available chlorine from sodium hypochlorite, followed by rinsing in sterile distilled water;
(b) Place the seed on moist filter paper in 9 cm Petri dishes (25 seeds per dish) and incubate for 24 h at 20–25 °C in a cycle of 12 h NUV light and 12 h darkness;
(c) After incubation freeze the plates at −20 °C for 24 h to prevent germination and re-incubate under conditions mentioned earlier for 5–6 days, after which the seeds can be examined microscopically for growth of different fungi. The scab pathogen produces white mycelium on the seed surface and shiny sporodochia with macroconidia. In *F. graminearum* the sporodochia appear as pale to dull orange.

Thus, it is apparent that the wheat scab can be controlled by an integration of different practices, like production and use of healthy seed (also see chapter on seed transmitted pathogens), use of less susceptible cultivars, cultural practices including crop rotation, tillage rotation, seed treatment with fungicide, timely application of fungicides at flowering, diversification of cultivars and date of planting and by judicious use of fungicides based on disease forecasting (Paul et al. 2010). None of these method used alone would effectively control the disease. Although severity of FHB is also related to the Fusarium infested crop residues of wheat and maize, residue burning is not recommended as a control measure for this disease.

3.5 Smuts

There are two kinds of smut diseases; the loose smut and the flag smut. The loose smut is of common occurrence wherever wheat is grown whereas flag smut occurs in restricted areas. Smut and bunt diseases are sometimes referred as “smuts”. Both

smut fungi are old diseases of wheat and are being studied over centuries. A brief description of two smuts is presented in the following pages. Detail information on these diseases is presented by several workers (Heald and Holton 1940; Fischer and Holton 1957; Western 1971; Wilcoxson and Saari 1996; Bockus et al. 2010).

3.5.1 Flag Smut

Flag smut of wheat was first reported in South Australia in 1968 (Wilcoxson and Saari 1996) and in the USA in 1940 (Heald and Holton 1940). In Latin and Central America, it has been reported only from Chile and Mexico, Its occurrence in India, Pakistan and South Africa was also reported in 1929 (Wilcoxson and Saari 1996). It is not reported from Brazil. In Australia and the USA losses caused in individual fields reached around 50 % (Fischer and Holton 1957). However, in recent years this disease has not been considered to be of economic importance.

Symptoms

Flag smut is typically a leaf and vein disease. Infected leaves and seedlings are twisted and become most conspicuous during stem elongation and early boot stage (Fig. 3.15), where long grayish-black streaks of blistered smut sori develop between the veins. Later the sori break through the epidermis and release a mass of black spores. In the field infection can be distinguished by plants showing stunted, wilted and yellowish-green leaves (Line 1993).

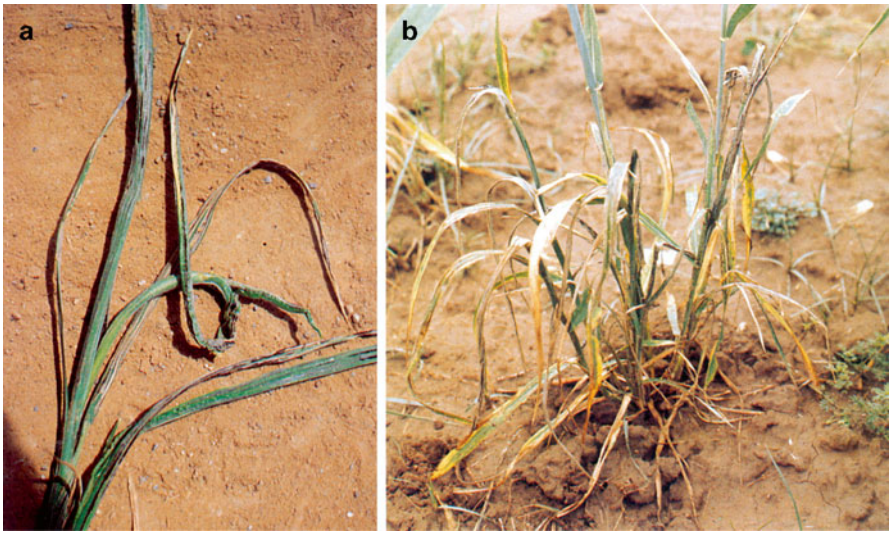

Fig. 3.15 (**a**) Flag smut (*Urocystis agropyri*) symptoms on wheat showing twisted foliage; (**b**) flag smut symptoms showing stunted plant (Courtesy: CIMMYT)

Causal Organism and Epidemiology

Flag smut is caused by *Urocystis agropyri* (Preus) Schroet. (Syn. *U. tritici* Koern). Some strains of this fungus also attack several other grasses. The sori of this pathogen consist of 1–6 black teliospores. Teliospores are globose to subglobose 8–18 μm in diameter, whereas spore balls measure 18–52 μm.

During the harvest, the spore balls are crushed and teliospores are released. The teliospores can survive on the contaminated seed and in the soil they can remain viable for 4–7 years (Line 1993; Wilcoxson and Saari 1996). Teliospores germinate and produce sporidia which infect wheat coleoptiles early in the process of seed germination. The infection is systemic in the plant and produces smut galls between the veins of the leaves. Pathogenic variability and differential response of *U. agropyri* collections on *Triticum* spp. was studied by Sharma et al. (1995). The life cycle of this pathogen is similar to that of common bunt.

Control

The disease can be controlled through the use of resistant cultivars and cultural practices such as rotation with non-host species for at least 2–3 years. Resistant cultivars are known to exist in several countries (Line 1972, 1993).

Fungicidal seed treatment with carboxin, tebuconazole or triademefon gives good control of seed-borne and soil-borne inoculum (Wilcoxson and Saari 1996).

3.5.2 Loose Smut

Loose smut of wheat, is caused by a fungal pathogen belonging to the class of Basidiomycetes and occurs all over the world on cultivated wheats; *Triticum aestivum*, *T. dicoccum*, *T. durum*, *T. monococcum*, *T. polonicum*, *T. spelta* and *T. turgidum* (Heald and Holton 1940; Neergaard 1979). It also occurs on triticale (*X. Triticosecale*). The disease is of little or no economic importance in most of the countries, except in very rare cases. In some individual fields the yield losses could reach up to 17–27 % (Western 1971; Mehta 1993). In Latin America, the infection levels between 0 and 0.2 % in susceptible cultivars at times may be observed in some fields where the seed is not treated. The loss in yield is proportional to the percentage of infection.

Symptoms

Loose smut directly affects the wheat spikes. Infected plants are shorter than the normal plants, but loose smut symptoms are visible only at the time of spike emergence (Fig. 3.16). The infected spikes emerge before the healthy ones and are completely transformed into a black mass of clamydospores. All the tillers of infected plant show infected spikes in which no grains are formed.

Fig. 3.16 Infected wheat spikes with loose smut fungus (*Ustilago tritici*)

Causal Organism and Epidemiology

Loose smut of wheat is caused by *Ustilago tritici* (Pers) Rostr. [Syn. *Ustilago nuda* var. *tritici* Schaf. and *U. nuda* (Jens.) Rostr.]. Although the rusts and smuts belong to the same class of fungi (Basidiomycetes), they differ from each other in relation to their spore production system. The rust fungi produce five types of spores whereas the smut fungi produce only two types of spores: the clamydospores (teliospores) and the basidiospores (sporidia).

The teliospores are black, finely raticulated and measure 5–8 μm in diameter. They germinate and form four or more septate promycelium. Studies on physiologic specialization were conducted some years ago (Jonston 1959; Medeiros 1976).

Loose smut is a systemic disease and is internally seed-borne. The infection occurs at the time of flowering. The teliospores are blown by the wind, get deposited on the flowers of wheat spike where they germinate and form promycelium. The promycelia fuse and produce infective hyphae which penetrate through the ovary wall and thus causes seed infection. After infection the mycelium stays dormant in the embryo until the germination of the seed. The infected seed cannot be distinguished from the healthy seed by the naked eye. Mycelium of *U. tritici* in infected seed can be visualized through the extraction of embryo (Fig. 3.17). Infected seed gives rise to an infected plant. When infected seed germinates, the loose smut fungus grows along with the growing plant in its intracellular spaces until the time of spiking when it transforms the spike into a loose mass of spores (clamydospores). These spores are later blown by wind and infect other spikes at the time of flowering, thus completing the life cycle of the pathogen (Fig. 3.18). The loose smut fungus *U. tritici* of wheat does not attack barley and vice versa.

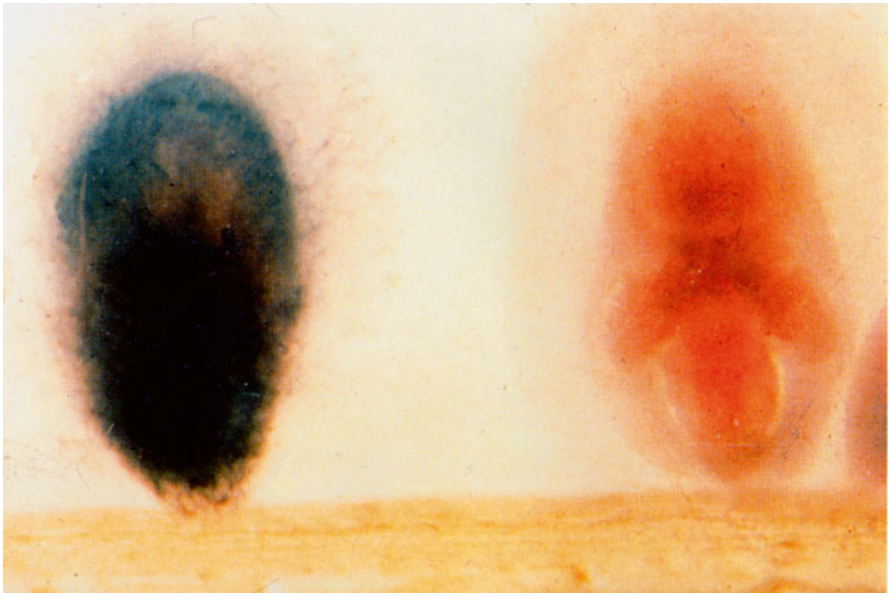

Fig. 3.17 Wheat embryos. Healthy (*left*) and infected (*right*) with *U. tritici*

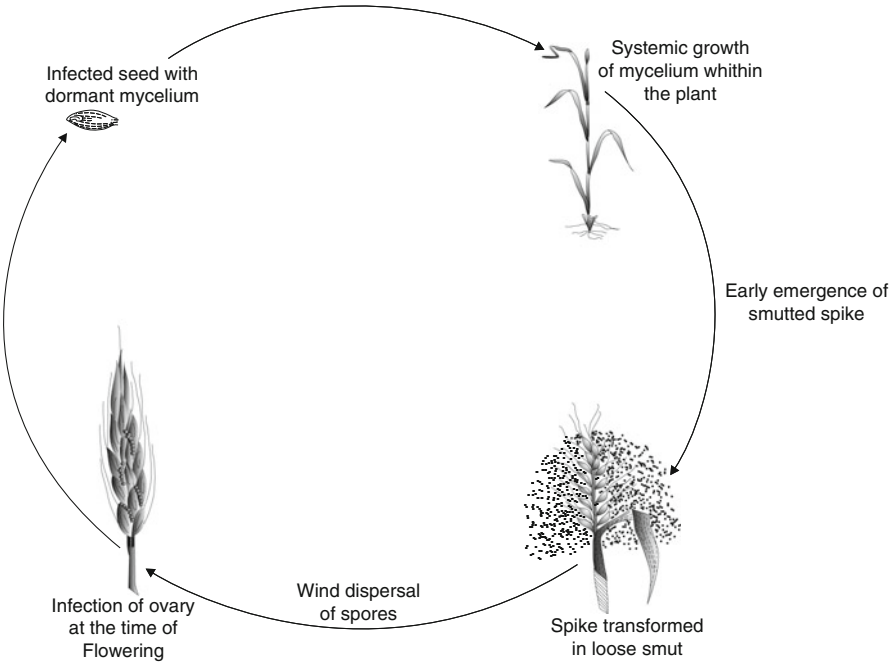

Fig. 3.18 Life cycle of *U. ritici*

Control

In the past, this disease was controlled by the hot water seed treatment (Mehta 1993), but this kind of treatment is now outdated. Loose smut is effectively controlled through the use of resistant cultivars and through seed treatment with some systemic fungicides. Some of the efficient fungicides are thiram 93.7 + carboxin 93.7 a.i./100 kg seed and triadimenol (400 g a.i./100 kg of seed). At present most of the wheat cultivars are resistant to loose smut.

Loose smut is also controlled through the seed certification procedures as followed in different countries like Brazil, the UK, India and the USA. The maximum tolerance limits for field infections are 0.2 %, 0.5 % for basic seed, first generation certified seed and second generation certified seed, respectively (Rennie et al. 1983).

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Chapter 4
Spike Diseases Caused by Bacteria

As stated earlier there are eight bacterial diseases known to occur in wheat, these being bacterial streak and chaff (*Xanthomonas translucense* pv. *undulosa*), bacterial mosaic (*Corynebacterium michiganensis* subsp. *tesselarius*), white blotch (*Bacillus megaterium* pv. *cerealis*), pink seed (*Erwinia rhapontici*), stem melanosis (*Pseudomonas cichorii*), bacterial sheath rot (*Pseudomonas fascovaginae*), basal glum rot (*Pseudomonas syringae* pv. *atrofaciens*) and bacterial leaf blight (*Pseudomonas syringae* pv. *syringae*). Some of the pathogens also cause diseases on other parts of the plant and are dealt with in the following Chapters. Out of these diseases, only a few are economically important or else have a wide host range and hence are described in the following pages. For other bacterial diseases the reader may refer to other publications like: McBeath (1993) and Maraite et al. 2007; Bockus et al. 2010).

Another disease described as "Tundu" (ear cockle) in India, is a complex disease of wheat spikes occurring in association with *Rathaybacter tritici* (*Corynebacterium tritici, Corynebacter michiganense* pv. *tritici, Clavibacter tritici*) and the seed gall nematode *Anguina tritici*. Its occurrence is restricted only to India, Pakistan, Egypt, Australia, Canada and China.

4.1 Bacterial Leaf Streak and Chaff

Bacterial leaf streak and black chaff was first described by Smith (1917) and later Xanthomonas streak was described as a causal agent of bacterial blight of barley. In 1919 the pathogen was named *X. translucens* var. *undulosa* (Smith 1917; Smith et al. 1919; Schaad and Forster 1985). Xanthomonas streak or black chaff has been increasing gradually in Latin America since 1979, and became one of the important diseases of wheat in this part of the world. The disease exploded in 1982, attacking almost all the wheat cultivars and advanced breeding lines. Later, severe outbreaks of the disease were observed in commercial wheat field in Brazil and other neighboring countries.

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Severe yield losses were reported in different countries (Boosalis 1952; Cunfer and Scolari 1982; Schaad and Forster 1985; Mohan and Mehta 1985). Yield losses of about 40 % are reported in sprinkler-irrigated fields in southern Idaho (Schaad and Forster 1985). In Argentina similar yield losses provoked by this disease were reported (Mehta et al. 1992). Usually, the pathogen does not cause seedling losses. Mehta and Bassoi (1993) reported that the disease caused up to 39.7 % losses in yield, depending upon the wheat cultivar and the level of bacterial infection/contamination in the seed. Under severe infections favored by cool and rainy periods, commercial fields become damaged (Fig. 4.1). The disease normally becomes severe after the heading stage, when most of the yield components, including the number of grains per spike, are already defined. The reduction in yield is due to the reduction in grain weight. On average, reduction in yield was proportional to the increase in disease severity (Mehta and Bassoi 1993).

Although the disease has become important in different countries, its occurrence in epidemic form is sporadic and very much depends on the weather conditions of the year.

4.1.1 Symptoms

Bacterial streak symptoms can be observed at any stage of plant growth. Initially, small yellowish water-soaked, oily, translucent streaks can be observed on the leaves (Fig. 4.2). Under humid and cold conditions abundant exudation of the bacteria can be observed on the leaves and on the stems as small yellowish granules which later become hard shiny crystals (Fig. 4.3). On the stems, initially, water-soaked yellowish patches can be observed with whitish exudation, becoming dark brown to violet in color (Fig. 4.4). Upon ageing the streaks on leaves coalesce into light brown blotches which later become dark brown and with abundant exudation giving an appearance of whitish-yellow crystals. Symptoms on culms, rachis and awns are characterized by alternating bands of diseased and healthy areas, yellowish in color and with violet areas at both ends of the lesion (Figs. 4.4 and 4.5). On glumes, symptoms are distinct from those produced by pseudo-black chaff or melanosis provoked by the stem rust resistance gene *Sr2* (Mehta 1993; Mehta and Bassoi 1993). However they can sometimes be confused with melanosis. On the culms and rachis the lesions may reach 5–6 cm in length.

4.1.2 Causal Organism and Epidemiology

Bacterial streak of wheat is caused by different pathovars of *Xanthomonas translucens* (*Xanthomonas translucens* pv. *undulosa* (Xtu); *X. campestris* pv. *undulosa* Hagb (Syn. *X. c*. pv. *Translucens* J. J. and Dye; *Phytomonas translucens* var.

Fig. 4.1 (**a**, **b**) Severely infested wheat fields with bacterial streak (*Xanthomonas ttranslucens* pv. *undulosa*)

Fig. 4.2 Initial symptoms of bacterial streak on wheat leaves

undulosa Hagb., *X. campestris* (Pam.) Downs; and *X. translucens* (J. J. and R.) Downs var. *undulosa* (J. J. and R.). Several pathovars are known but the pathovars that attack wheat are *undulosa* and *cerealis*. Other than wheat, the pathogen attacks different hosts of Gramineae including triticale, but rarely rye and barley.

Bacterial streak (*X. t.* pv. *undulosa*) symptoms showing a water-soaked and oily appearance, can be observed seven days after inoculation, using seedling injection technique (Fig. 4.6a). The bacterial colonies on nutrient agar with glucose are round and yellow due to the production of the pigment xanthomonadin, mucoid, slightly elevated and shiny (Fig. 4.6c). The bacterial cells are rod shaped, with one polar flagellum (Fig. 4.7), gram negative, aerobic, catalyze positive, oxidase negative, reduce nitrate to nitrite and positive to hydrolysis of gelatin (Mohan and Mehta 1985).

Mehta (1996a, b), studied several isolates of Xtu and classified them in two groups. In the first group ten isolates were highly aggressive on wheat and with little necrosis on oats. The second group of six isolates showed less aggressive symptoms on wheat, without oily and translucent appearance, did not produce exudation on wheat and caused only chlorosis on oats. Positive immunofluorescence reaction using monoclonal antibody was obtained only for the first group of isolates.

Based on their host range and molecular markers, Bragard et al. (1993, 1995, 1997), grouped the different pathovars of Gramineae into *X. t.* pv. *undulosa*. Recently, pathogenic and genetic diversity of Xcu was reported by Adhikari et al. (2012).

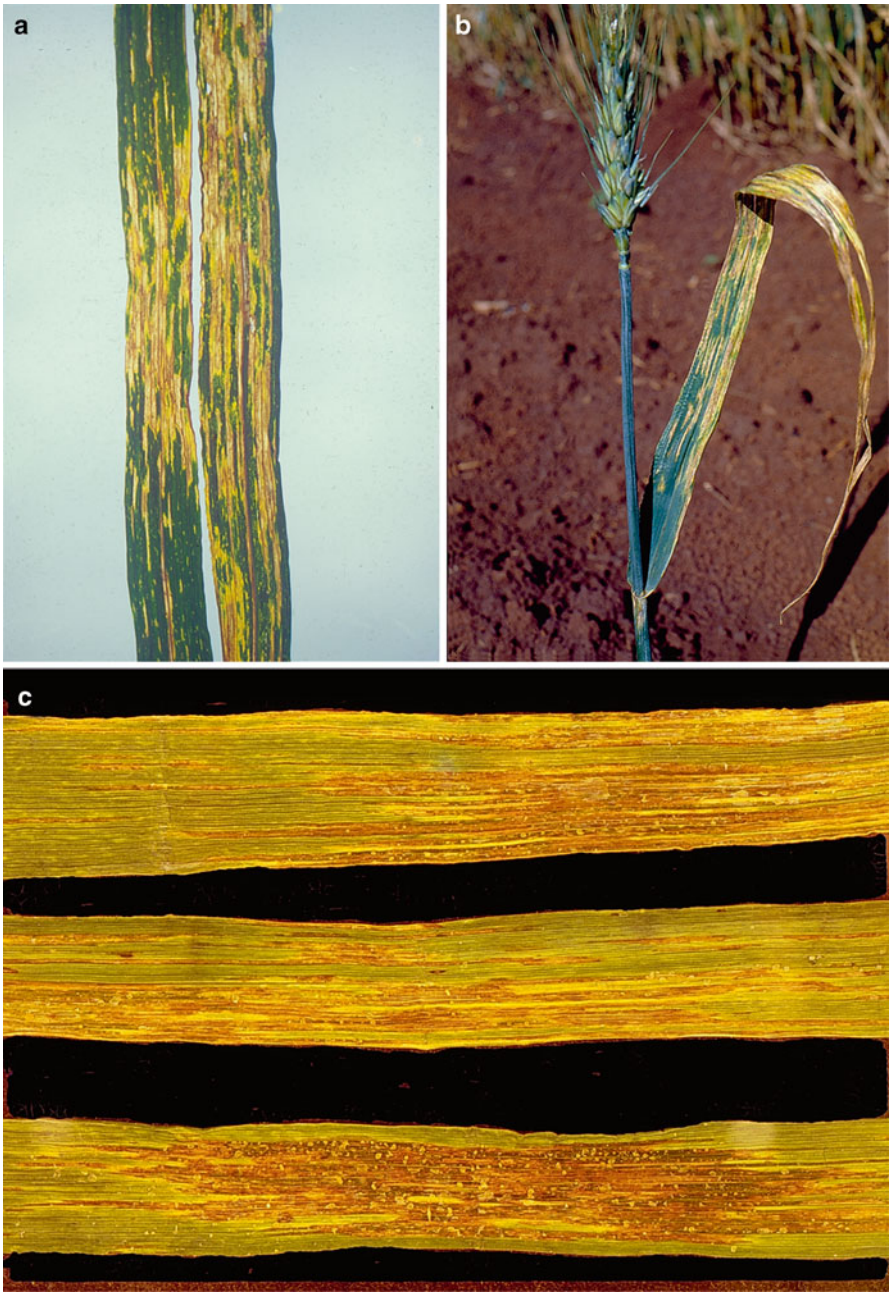

Fig. 4.3 (**a**–**c**) Severely infected leaves with bacterial streak

Fig. 4.4 (**a**–**d**) Bacterial streak symptoms on wheat culms with water-soaked, oily appearance and exudation

Fig. 4.5 (**a, b**) Bacterial streak symptoms on wheat glumes and awns

An extensive review on integrated approaches for detection of plant pathogenic bacteria and diagnosis of bacterial diseases was presented by Alvarez (2004).

Xanthomonas streak (Xcu) is transmitted through infected/contaminated seed (Schaad and Forster 1985; Mohan and Mehta 1985; Mehta 1990; Mehta and Bassoi 1993). It is suspected that in the USA the pathogen survives in the soil during the absence of wheat. In tropical and sub-tropical areas, the pathogen does not survive more than a few months on the crop residue because of high temperatures during the summer (Mehta et al. 1992; Mehta 1993). The bacterium Xtu survives in the infected seed for about three years under normal seed storage conditions. However, the percentage of seed infection drastically reduces during the storage period, so that longer the storage period lower would be the percentage of seed infection (Mehta and Bassoi 1993; Bragard et al. 1993).

Normally, the disease is more severe on the contours (terrace) than in the remainder of the field because in the contours there is a high concentration of organic matter, which promotes higher density and vigor of the plants and consequently more favorable micro-environment for the development of Xanthomonas streak.

The principal reasons for the spread of the disease are attributed to three factors: (a) Free exchange of germplasm; (b) Lack of practical laboratory methods to identify infected seed lots; (c) Lack of resistant cultivars.

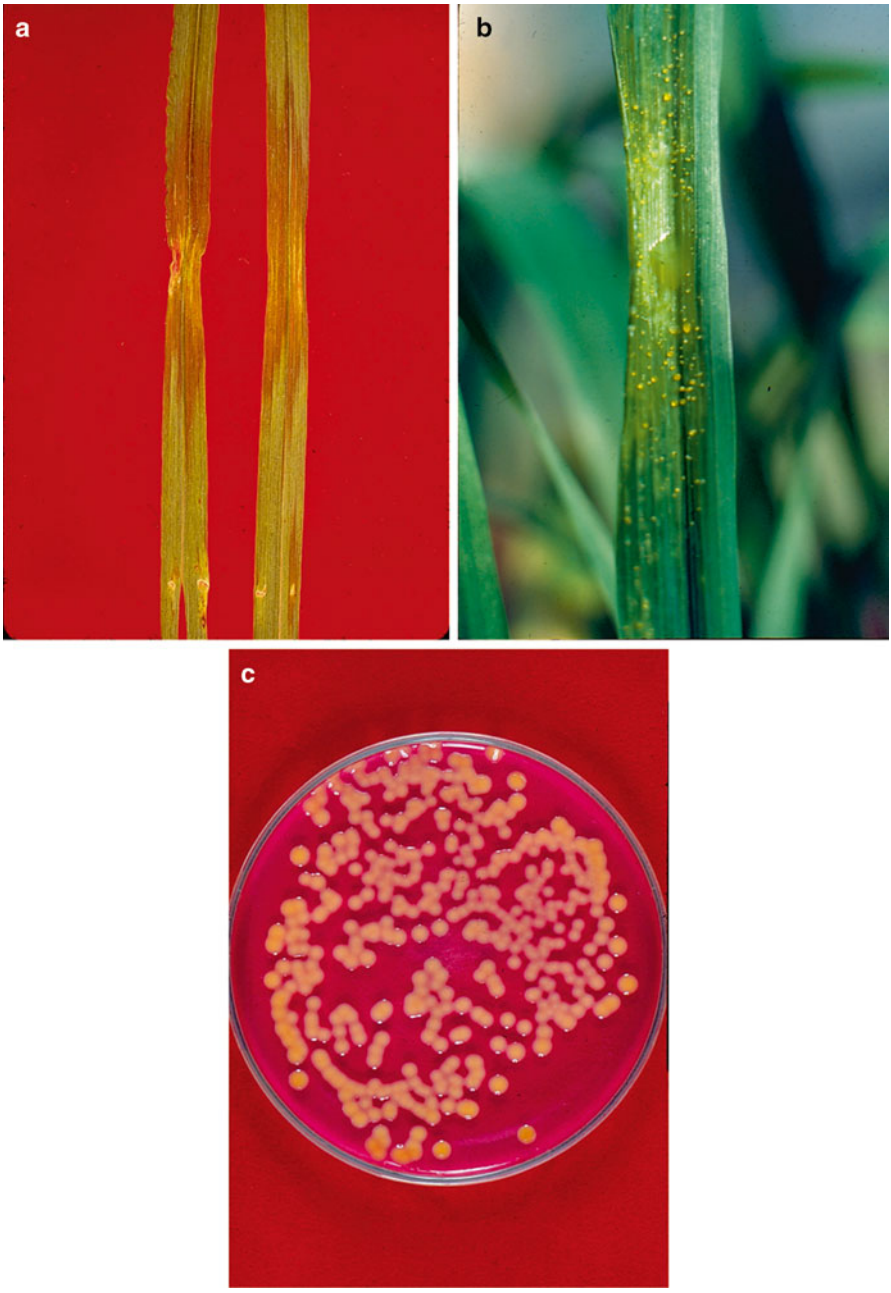

Fig. 4.6 (**a**) Bacterial streak symptoms showing water-soaked and oily appearance, seven days after inoculation, using seedling injection technique; (**b**) exudation of bacteria as *small yellowish* granules, 12 days after inoculation; (**c**) colonies of *X .t.* pv. *undulosa* on nutrient agar

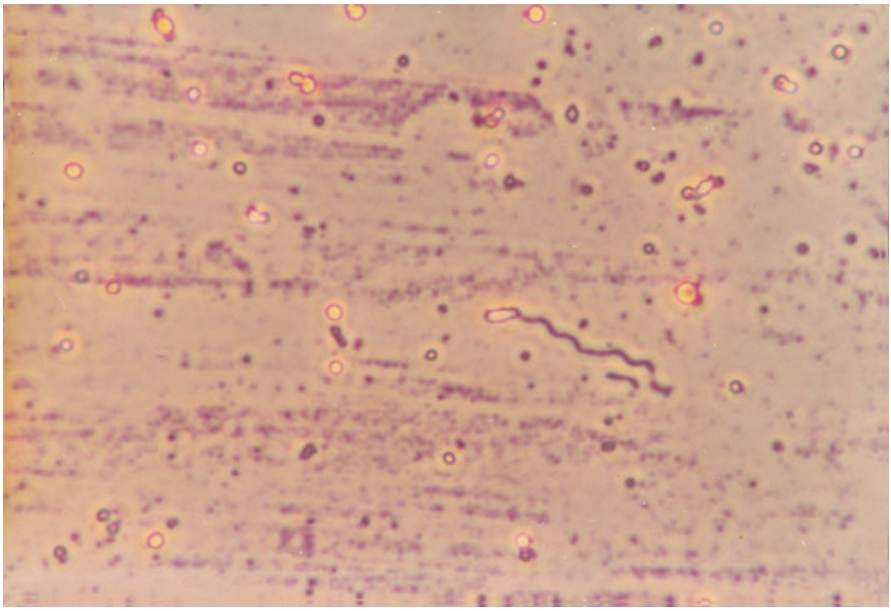

Fig. 4.7 *X. t.* pv. *undulosa* bacterium with one polar flagellum. Source: Mohan and Mehta (unpublished)

The spread of the disease from one field to another is through the wind and rain splashing and is best favored by average temperatures between 18 and 20 °C (Mehta et al. 1992; Mehta 1993). Unlike in other countries, under Brazilian conditions the disease does not develop when the temperatures are high.

Resistant cultivars have not so far been developed due to lack of resistant sources in the germplasm. However, under field conditions, visible differences can be observed among some cultivars. Mehta (1996a, b) identified some wheat cultivars with different degrees of resistance based on the rate of lesion extension.

4.1.3 Control

Since Xanthomonas streak is seed transmitted, it can be controlled through seed treatment. However, for this it is necessary to analyze the seed to verify the level of seed infection/contamination. The threshold level problems are well discussed by some workers (Zadoks 1985; Kuan 1988; Forster and Schaad 1988; Nutter 1993). For establishing a threshold level for infected seeds, a precise seed assaying method is a prerequisite. Some problems with the semi-selective medium (Xts–agar) developed by Schaad and Forster (1985), have already been reported (Mehta 1990) and a more specific seed assaying method is not yet available. Mehta and Bassoi (1993), reported that due to a prolonged rainy period during harvest time, probably the

saprophytic bacterial population in the seed lots increased and the Xts-agar detected very little or no Xtu, even in seed lots coming from severely infested fields with 50–70 % of the leaf area infected at the soft dough stage. According to these authors, too many saprophytic bacteria appeared on Xts-agar which might have inhibited the development of Xtu. In fact, it is very difficult to establish precisely the threshold level for seed infection especially for un-irrigated areas with unpredictable climatic conditions. Thus, a more efficient semi-selective medium needs to be worked out.

The practical utility of quantification of bacteria in a particular seed lot is questionable when using the existing culture media. The following questions still remain unanswered: What should be the sample size and how many sub-samples must one use for routine seed health testing before a particular seed lot is released? Can colony forming units (cfu/g) be used as a parameter to predict or not an epidemic in the subsequent crop grown under unirrigated areas?

Mehta (1990), developed a modified seedling injection technique to detect the presence of *X. t.* pv. *undulosa* in the wheat seed lots (Saettler 1971). Mehta (1990), reported that in none of the wheat seed samples (out of 88 tested), did the seedling injection technique fail to detect the presence of pathogenic bacteria. A little variation using seedling injection technique is possible because of the natural heterogenicity of number of contaminated seeds and/or the presence of less aggressive strains of the bacteria.

Later, Bragard et al. (1993), tested several other methods like Xts-agar (Schaad and Forster 1985), immunofluorescence microscopy (IF) and enzyme linked immunobsorbent assay (ELISA) and compared them with the modified seedling injection technique. They concluded that the seedling injection technique can be used to detect the contamination of bacteria in the seed.

For small amounts, seed treatment with acidified cupric acetate can eliminate the bacteria from the seed. According to Forster and Schaad (1988), cupric acetate treatment is toxic to seed and causes reduction in germination. A little reduction in germination is also reported by Sands et al. (1989), using dry heat seed treatment. Mehta (1990) reported a drastic reduction in the level of seed contamination with bacteria using dry heat seed treatment. Furthermore, Mehta and Bassoi (1993) reported almost complete eradication of Xcu by seed treatment with Guazatine Plus (guazatine 33 % + imazalil 2.0 %). This fungicide is manufactured by Kenogard, Sweden and is not available for commercial use.

As far as the tolerance limit for bacterial seed infection is concerned, for the southern cone region of Latin America, apart from considerations of quarantine, a zero level of tolerance is not necessary either for seed contamination or for the resultant infection in the crop. Mehta (1990) studied the relationship between the percentage of leaf area infected observed in the field and the contamination of the corresponding seed lot. Out of a total of 50 wheat cultivars with <10 % leaf area infected at the soft dough stage in the field, seeds of 38 wheat cultivars either had no seedling infection, as indicated by seed health analysis (seedling injection technique), or yielded <1,000 cfu g^{-1} on XTS agar. Based on this finding, a tolerance limit of 10 % leaf area infected with Xanthomonas streak at the flowering-soft dough stage is being employed in seed-multiplication fields, especially in rain-fed areas (Mehta et al. 1992; Mehta 1993).

Use of resistant cultivars is the best way to control Xanthomonas streak. So far, complete resistance in wheat and triticale has not been found. However, Mehta (1996a, b) identified some cultivars having partial resistance like Ibiara, Piratan, Cacatu and Batuira, based on the rate of lesion extension. Under field conditions, visible differences could be observed among some cultivars like Batuira and BR 14, showing less visible bacterial streak symptoms. According to Mehta et al. (1992), although cvs. Caite and Tapejara were susceptible to foliar infection, they were considered tolerant to the disease. Duveiller et al. (1993), reported that resistance to four cultivars (Pavon, Mochis, Angostura and Alondra) was governed by five different genes. Under Brazilian conditions, however, cv. Alondra was always found to be susceptible under natural field conditions.

Raja et al. (2010) screened six wheat cultivars against ten different strains of *X. translucens pv. undulosa* at various stages of plant growth to check virulence against bacterial streak. Their results showed that all the six cultivars were susceptible to the bacterial streak, however the severity of bacterial streak was variable among the wheat cultivars.

Somaclonal variation for disease resistance in wheat and production of double haploids (wheat x maize) may offer new perspectives for developing disease resistant cultivars (Mehta and Angra 2000; Silva et al. 2010).

For efficient control of Xanthomonas streak, an integrated approach would be desirable to manage the disease, like seed multiplication in disease-free areas, use of tolerance level of disease severity in the field and seed treatment of the genetic seed (foundation seed) (Mehta and Bassoi 1993; Bragard et al. 1993).

4.2 Pink Seed

Pink seed is a bacterial disease and occurs in several cold climate countries of Northern hemisphere like Canada, England, France, Russia, Ukraine and USA. It is also reported in Australia and Israel (Diekmann and Putter 1995). According to Maraite et al. (2007), the disease is recently reported from common beans (Huang et al. 2002), which demonstrates the wide host range of this pathogen.

Although some parts of Argentina, Chile and Brazil may favor the disease, its occurrence is not yet reported. The bacteria causing this disease is considered an opportunistic pathogen and hence is not responsible for appreciable yield losses in wheat.

4.2.1 Symptoms

Seeds from infected plants show pinkish coloration and are slightly shrunken, however, morphologically they look normal. The pinkish discoloration of seed also resembles seeds treated with some fungicides (Fig. 4.8). Normally, the percentage of infected seed in seed lots is relatively very low as compared to other bacterial diseases.

Fig. 4.8 Symptoms of pink seed caused by *Erwinia rhapontici*. Courtesy R.L Forster

4.2.2 Causal Organism

Pink seed disease is caused by the bacteria *Erwinia rhapontici* (Mill.) Burk. [Syn. *E. carotovora* var. *rhapontici* (Mill.) Dye]. The bacterium is rod shaped, gram negative, with 3–7 peritrichous flagella, catalyze-positive, oxidase-negative, positive for glucose fermentation and measures 1.2–1.5 × 0.5–0.8 μm. In culture medium the bacteria produces a diffusible pink pigment (McBeath 1993).

4.2.3 Epidemiology and Control

Pink seed bacteria invades injured kernels. Since the disease is not economically important no control measures are prescribed.

4.3 Pseudomonas Leaf Blight

Bacterial leaf blight was first described by Otta in 1974. Later, the disease spread on different crops in the USA. The disease occurs in several other countries like Argentina, Australia, Bangladesh, Bulgaria, Canada, New Zealand, Pakistan, Russia Tunisia and Ukraine (McBeath 1993). In Brazil, it was reported in 1976, but without causing any substantial damage in wheat (Mehta 1978, 1993). In general, economic losses caused by this disease are considered very low (Maraite et al. 2007; Valencia-Botin and Cisneros-Lopez 2012).

4.3.1 Symptoms

Bacterial leaf blight is basically leaf disease but spike infections can also occur. Spike infections can also be caused by *P. syringae* pv. *atrofaciens* (Otta 1974; Mehta 1978, 1993; Maraite et al. 2007). Initial symptoms are characterized as water-soaked spots which expand and coalesce into large yellowish areas covering almost 50 % of the leaf area (Fig. 4.9).

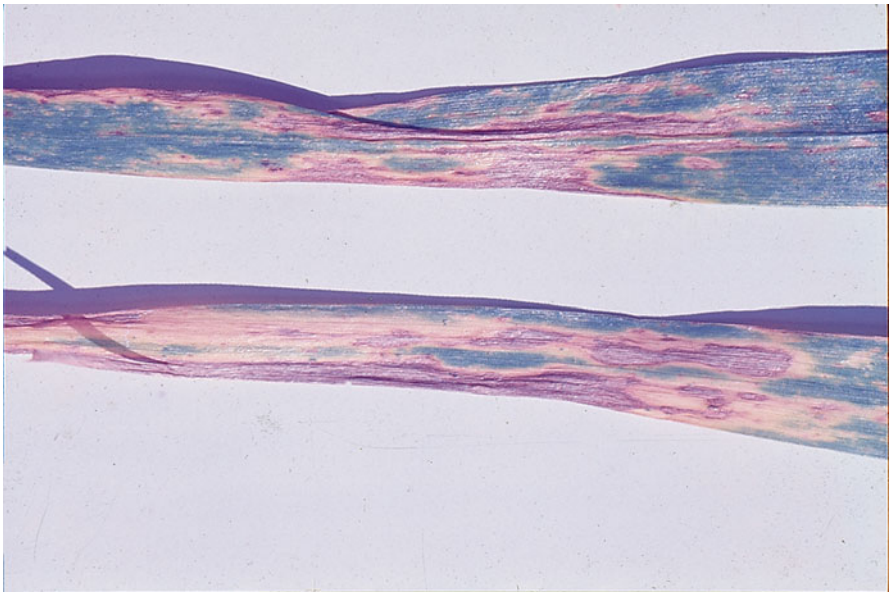

Fig. 4.9 Wheat leaves infected with *Pseudomonas syringae*

4.3.2 Causal Organism and Epidemiology

Bacterial leaf blight is caused by *Pseudomonas syringae* pv. *syringae* Van Hall. The bacterium is rod shaped, gram negative and is motile with one polar flagellum. According to Maraite et al. (2007), *P. syringae* pv. *syringae* and *P. syringae* pv. *atrofaciens* cannot be distinguished by colony morphology nor by physiological, serological and basic genetic features.

Strains belonging to the heterogeneous *P. syringae* group have been associated with a wide range of diseases on herbaceous as well as arborescent plants (Maraite et al. 2007). *P. syringae* pv. *Japonica* was considered as a synonym of *P. syringae* pv. *syringae* by Young (1992). The bacterium is seed transmitted. Prolonged rainy and cool periods are essential for the disease development.

4.3.3 Control

As the disease is seed transmitted, the best way to control is to produce seed in disease-free areas. No seed treatment procedures are known for commercial seed lots. Cultivar resistance is known to exist. Resistance was identified in Russian winter wheat and spring wheat cultivars (Maraite et al. 2007).

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Chapter 5
Spike Diseases Caused by Viruses

As mentioned elsewhere, there are over 30 viral diseases reported to occur in wheat but only a few are economically important. Some of the viral diseases are overlooked because of their low economic importance or else because their symptoms are rather ambiguous and at times attributed to nutritional deficiencies of the plant. Viruses cause several disorders in the wheat plant including foliar chlorosis appearing as mottles, dashes, blotches or streaks (Zaitlin and Palukaitis 2000; Jones et al. 2010; Bockus et al. 2010). Only four viral diseases are described in the following pages, especially considering their economic importance.

5.1 Barley Stripe Mosaic

5.1.1 Symptoms

Barley stripe mosaic is the only viral disease of wheat transmitted through seeds. Infected seed show no symptoms of the disease. It occurs in several countries where wheat is grown but without causing any appreciable yield losses. It was first described in the USA in 1910, but its causal agent was not identified until 1951 (McKinney 1951). Its presence in some countries including Paraguay in 2012 was reported (McKinney 1951; Mehta 1993).

The symptoms of the disease are similar in wheat and barley and depend on the strain of the virus and the wheat cultivar (McKinney and Greeley 1960). Infected plants are stunted with excessive tillering and the leaves show chlorotic stripes which later become brownish (Fig. 5.1).

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Fig. 5.1 Symptoms of barley stripe mosaic on wheat. *Courtesy*: M.C. Bassoi

5.1.2 Causal Organism and Epidemiology

The disease is caused by Barley yellow stripe Mosaic Virus. The virus particles are rod-shaped and measure 30–160 nm. The virus can survive for several years in the seed (McKinney 1951; McNeal and Afanasiev 1955; McKinney and Greeley 1960; Phatak 1974; Albrechtsen 1993).

The disease occurs on oats, rye, maize, millet and several grass species. No virus vector of this disease is yet known. Several strains of this virus are known to exist (McKinney and Greeley 1960). The dissemination of the disease in the field is probably through the contact of healthy plants with diseased plants. Natural hosts are members of the Gramineae, but species of Chenopodiaceae, Solanaceae, Amaranthaceae and Primulaceae can be artificially infested (Atabekov 1971). Normally the percentage of seed infection is not very high, but in certain cases it can reach up-to 67 % (Phatak 1974) and very rarely reach 81 % (McNeal and Afanasiev 1955). In barley for example, the seed infection can reach 90 % (Albrechtsen 1993).

5.1.3 Control

Certification schemes and seed health testing using ELISA could help discard seed lots showing high levels of seed infection (Carroll 1983).

5.2 Barley Yellow Dwarf

Barley yellow dwarf occurs all over the world and is one of the most important viral diseases of wheat. Infected plants produce 5–20 % less grains. Gill (1970a, 1970b), estimated a yield loss of 34 % in Manitoba. In Brazil, the losses could be between 20 and 30 % (Caetano 1982). In an epidemic year, however, the yield losses have reached up-to 70 % in England (Western 1971).

5.2.1 Symptoms

Symptoms of the disease depend on the strain of the virus, the temperature and the resistance of the cultivar. By and large, the infected plants are stunted and their leaves show yellow, red and purple discoloration. This kind of coloration starts from the tip of the leaf and advances to its base. Sometimes green stripes between the yellowish leaf areas can be seen along with some necrosis. In case of severe infection the whole plant becomes yellow with little, or at times, excessive tillering (Fig. 5.2). The root system of severely infected plants is drastically reduced and the plant does not produce any grains. Normally, the infected plants are scattered in the field.

5.2.2 Causal Organism and Epidemiology

The disease is caused by a virus called barley yellow dwarf virus (BYD). The virus moves systemically within the plant and occurs in phloem cells. There are seven identified strains of the virus. They are characterized as: **RMV**: transmitted regularly by aphids *Rhopalosiphum maidis*; **RPV and PAS**: transmitted regularly by *R. padi*; **MAV**: transmitted regularly by *Macrosiphum avenae*; **PAV and PAS**: transmitted regularly by *R. padi* and *M. avenae*; **SGV and GPV**: transmitted by *Schizaphis graminum* and *R. padi* (Rochow 1969; Gill 1970a; Rochow and Muller 1971; Bockus et al. 2010).

The barley yellow dwarf virus is transmitted only by aphid vectors. The dissemination of this disease depends on the presence of different species of aphids. After a few hours of feeding on an infected plant, the aphids are capable of transmitting the virus to other plants. The virus is persistent in the aphids. Cool temperatures of about 20 °C are favorable for the development of the disease (Figueira 2010).

Fig. 5.2 Wheat plant infected with barley yellow dwarf virus (BYD)

5.2.3 Control

The disease can be kept at tolerable levels by controlling the aphids through the use of insecticides. Biological control of aphids however, has been successful to some extent in Brazil and Bolivia. Although cultivars differ in their level of resistance, no high level of resistance is yet known to exist in wheat. Nevertheless, Ohm et al. (2005) reported that highly effective resistance to yellow dwarf virus was introgressed into wheat (*Triticum aestivum*) from *Thinopyrum intermedium*. They further reported that such lines also have resistance against other diseases like soil-borne mosaic virus, powdery mildew, stem rust, stripe rust and tan spot.

5.3 Rice Hoja Blanca

Rice hoja blanca occurs in Japan and in the American continent (Gibler et al. 1961; Caetano et al. 1970). The disease is of little economic importance.

5.3.1 Symptoms

Infected plants show leaf mottling and complete chlorosis. Severely infected plants dry prematurely without forming grains. The spikes become white and are distributed in the field. Similar white spikes may also be due to the attack of the stem borer *Diatrea saccharalis* Fabr., or because of frost. The Rice hoja blanca can be distinguished from the other causes, since *D. saccharalis* creates a small hole at the base of the stem which can be easily observed and in the case of frost damage, the spikes can be easily removed by fingers from the stem.

5.3.2 Causal Organism and Epidemiology

Hoja blanca is caused by RHBV (Rice hoja blanca virus). Other than wheat the RHBV attacks rice (Lamey et al. 1964). It is transmitted by insects of the genus *Sogata*. Two species of *Sogata S. cabana* Crawf. and *S. orizicola* Muir, are known as vectors of this virus. In these insects the virus is circular and persistent. In the absence of wheat the vector migrates to other fields of rice. After the rice harvest, the vector comes back to the wheat fields and completes the life cycle of the disease.

5.3.3 Control

No specific control measures are recommended.

5.4 Soil-Borne Wheat Mosaic

Soil-borne mosaic has been known to occur in the USA since 1919. It is also known to exist in the Latin American region (Argentina and Brazil) and is quite common in the State of Rio Grande do Sul, Brazil. In 1971, severe outbreaks of this disease were observed in 30 different districts. Later, the disease occurred in lower intensities in the same State (Caetano 1982). Yield losses caused by this disease are very variable (Dalbosco et al. 2001). Quinsberry and Reitz (1967) reported losses of about 1.5–3.0 million dollars during some epidemic years. Later, Kucharek and Walker (1974) reported losses between 42.5 and 52.5 % in commercial fields in Florida. Kuhne (2009) has given a comprehensive review of soil-borne viruses attacking cereals. According to this author, soil-borne viruses could still be a threat to wheat cultivation.

Fig. 5.3 Symptoms of soil-borne wheat mosaic

5.4.1 Symptoms

Soil-borne mosaic virus encompasses different strains showing variable symptoms between whitish-green to yellow mosaic on leaves (Quinsberry and Reitz 1967). The leaves of infected plants are stunted, mottled and show chlorotic stripes parallel to veins (Fig. 5.3). Rye, barley and *Bromus* spp. are the hosts of this virus.

5.4.2 Causal Organism and Epidemiology

The virus particles of soil-borne mosaic are rod-shaped and are present in the infected tissues. They measure 35–422 × 24–34 nm. The in vitro dilution end point of the virus is between 10^{-3} and $10^{-3.5}$ and remain viable for 48 h. The thermal inactivation is between 58 and 60 °C for a few minutes (CMI 1971). The virus is transmitted through the obligate fungus parasite *Polymixa graminis* Led. However, recently some doubts were raised whether *Polymixa* spp. are the specific carriers of plant viruses (Legreve et al. 2008).

P. graminis invades the wheat plant through its roots but does not produce any symptoms on the plant. After penetration the fungus forms plasmodial bodies in the epidermal cells of the cortex of the primary root. As a consequence, cristoforos

(compacted mass of spores) are formed in the plasmodial bodies and later, mature zoosporangia can later be observed on the epidermal cells of the root.

The disease spreads from one field to another through agricultural machinery. The resistant spores of *P. graminis* gives shelter to the virus and can survive in the soil for several years (Figueira 2010).

5.4.3 Control

Being a soil-borne disease, no specific control measures are indicated. However, crop rotation with non-host leguminous species may offer good results. Black oats (*Avena strigosa*) are considered resistant to this virus in Brazil. Continuous wheat in the infested area should be avoided.

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Chapter 6
Foliar and Stem Diseases

6.1 Foliar and Stem Diseases

Sixteen foliar and stem diseases including some sporadical diseases caused by fungi are described in this chapter. Among them Tan Spot, Spot blotch, Powdery mildew, three rusts, and two Septoria diseases are economically more important. As stated elsewhere, at present the occurrence of a new stem rust race Ug99 is causing a serious threat to wheat cultivation in the World. Two Septoria diseases namely Septoria nodorum blotch and Septoria tritici blotch are of major concern in several countries. A worldwide loss was estimated at US$1 billion, and in the USA alone the two Septoria diseases cause a yearly loss of over 1 % (Eyal et al. 1987). Normally, these diseases individually are capable of causing about 30–40 % loss in yield depending upon the country and the year. In the UK for cereal crops, annually 200 million Pounds are spent for fungicidal applications against Septoria diseases, without which the yield losses may amount up to 465 million Pounds (Gullino and Kuijpera 1994). Spot blotch was an important disease in several tropical and sub-tropical countries till the late 1980s. Later, due to the change in tillage practices and the development of moderately Spot blotch resistant cultivars Tan spot has become increasingly more important than the Spot blotch. In the recent years, severe incidences of Powdery mildew have also been observed in many countries (Ralph et al. 2012).

6.1.1 Alternaria Leaf Blight

Alternaria leaf blight is a specific disease of wheat mostly restricted to Indian subcontinent. The disease was first reported in 1924, but remained uncharacterized until 1962. From 1960 to 1964 the disease damaged all commercial cultivars in the subcontinent. The disease affects all parts of the plant including glumes and awns and causes significant yield losses (Prasada and Prabhu 1962; Prabhu and Prasada 1965, 1966, 1967).

Y.R. Mehta, *Wheat Diseases and Their Management*,
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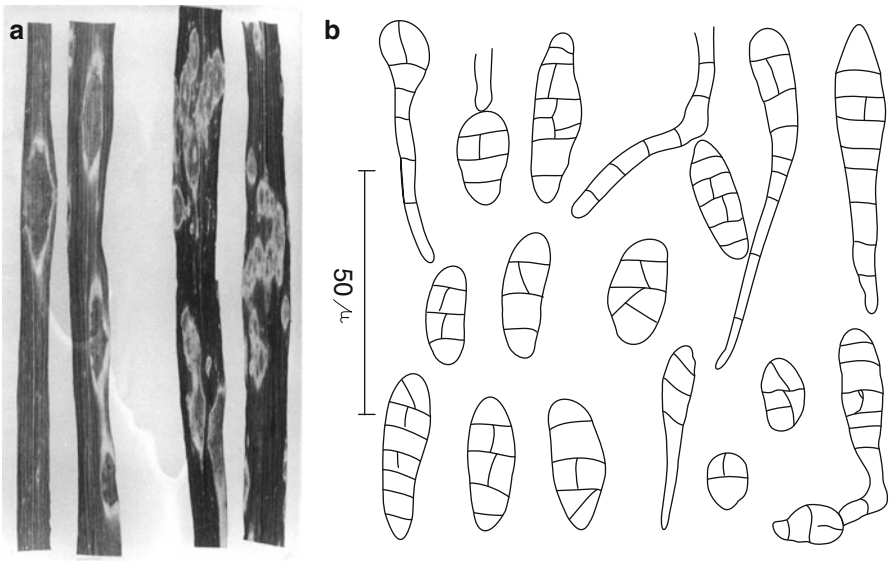

Fig. 6.1 Symptoms of *Alternaria triticina* on wheat leaves (*left*) and conidia of *A. triticina* on *right*. Courtesy A.S. Prabhu

Symptoms

The initial symptoms of the disease appear as small oval discolored lesions, irregularly scattered on the leaves. When the lesions enlarge they become irregular in shape and brown to gray in color and measure 1 cm in length. Later, the lesions coalesce and cover large areas causing premature death of the leaves. Under moist conditions, black conidial dust appears on the lesions and infection spreads to other parts of the plant including spikes and awns. In the field the disease appears when the plants are 6–8 weeks old. A bright yellow marginal zone is sometimes seen around the well-developed lesions (Fig. 6.1). Heavily infested fields become straw colored to dark brown and the yield is affected. Seeds originating from heavily infested fields show discoloration and are shriveled. The darkening of the seed may extend over the whole seed, in contrast to the black point symptoms caused by *Bipolaris sorokiniana* which are restricted to the embryo region.

Causal Organism and Epidemiology

Alternaria leaf blight is caused by *Alternaria triticina* Prasada and Prabhu. *A. triticina* is distinguished from other *Alternaria* spp. by its specificity to attack wheat. Conidia of *A. triticina* germinate at temperatures ranging from 5 to 35 °C, but the optimum varies between 15 and 27 °C. A minimum period of 48 h of near saturated atmosphere is a prerequisite for successful infection (Prabhu and Prasada 1966).

Conidia are borne on conidiophores, sometimes in chains of 2–4, but normally most of them are solitary. Conidia are smooth, irregularly oval, with both ends rounded, gradually tapering into a beak and measure 2–37 × 3–7 μm. Conidia have 1–10 transverse septa and 0–5 longitudinal septa. Spores in culture medium are very variable and may show all gradations from beakless to moderately beaked. The spores on natural hosts are moderately beaked. The hyphae, conidiophores and conidia are hyaline initially and later deep olive buff. Conidiophores are single and measure 3–6 × 17–28 μm.

A. triticina grows well in common artificial culture media. According to Prabhu and Prasada (1967), standard nutrient agar is the best medium to identify the seed infection caused by *A. triticina*, since it permits to distinguish from *A. tenuis* based on the colony characters after 6 days at 25 °C and also promoted abundant sporulation. According to these authors, the extent of seed infection varies from region to region in the country.

Young vigorous seedlings offer resistance to *A. triticina*. Other than wheat, *A. triticina* attacks triticale and barley. Wheat cultivars differ in their degree of resistance. Survival of the propagules of *A. triticina* in the soil or in the crop residue is very poor. The dissemination of the disease is by wind dispersal of the conidia. The pathogen is also seed transmitted and the fungus can remain viable in the seed till the next wheat season.

Control

Cultivar resistance and use of healthy seed offers the best way to reduce yield losses caused by *A. triticina*. Some of the genotypes like Hope, Klein, Frongtoso and La Previsión, offer a good level of resistance.

Fungicidal application applied to foliage at the initial stages of infection gives satisfactory results. Fungicidal application based on weather forecasting data would be the best way to reduce the severity of the disease (Bhowmic 1974; Sokhi 1974).

6.1.2 Downy Mildew

Other than wheat, downy mildew occurs on several hosts of Gramineae including barley, oats, rice and sorghum. Besides Brazil, it occurs in several countries like Australia, Austria, France, India, Iran, Iraq, Italy, Japan, Mexico, Pakistan, Turkey and the USA, but without causing appreciable damage to the crop (Chahal and Singh 1993). In maize and sorghum, it may cause appreciable yield losses in localized wet areas (Payak et al. 1970; Thakur and Mathur 2002). Normally, individual plants are affected. Occurrence of the disease is not very common and hence no information on yield losses in wheat is reported.

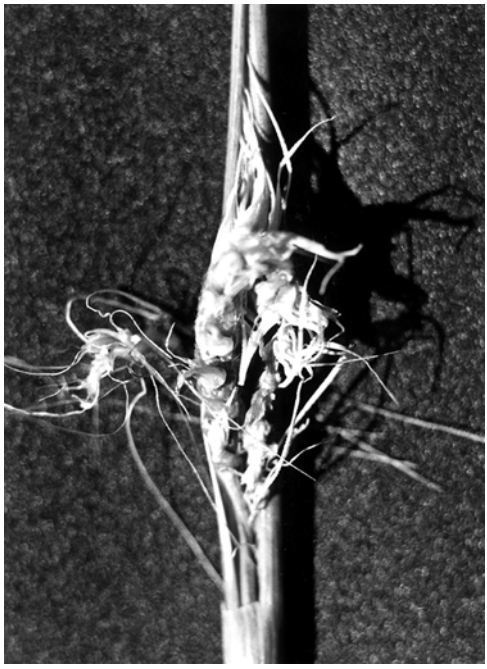

Fig. 6.2 Wheat plant infected with downy mildew showing "Crazy top" symptoms caused by *Sclerophthora macrospora*

Symptoms

The main characteristics symptoms of the disease are stunting, excessive tillering and thickened and twisted leaves. The tillers do not produce spikes and die. The spikes are twisted and sometimes do not emerge completely showing "Crazy top" symptoms. Production of excessive spikes, sometimes more than three in a single plant and excessive and twisted awns are observed (Fig. 6.2). In Brazil, the disease was noticed in individual plants during 1994, in the southern region of the State of Paraná. The disease occurs in excessively wet or water-logged areas and infected plants show stunting and yellowing. Infected plants do not show downy growth of the fungus.

Causal Organism and Epidemiology

The disease is caused by the fungus *Sclerophthora macrospora* (Sacc.) T. S. and N. [Syns. *Sclerophthora graminicola* (Saac.) Schroet., *Sclerophthora macrospora* Sacc.]. The fungus belongs to the family Pythiaceae and is an obligate parasite. According to Chahal and Singh (1993), oospores are hyaline to yellowish, smooth-walled and measure 40–75 μm in diameter.

Infected leaf tissues when examined under microscope show presence of spherical oospores of *S. macrospora* (Fig. 6.3). The oospores germinate in wet soil and

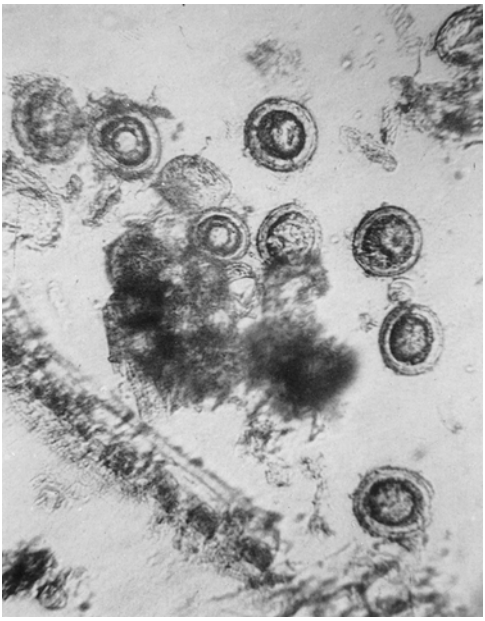

Fig. 6.3 Oospores of *S. macrospora* in infected leaf tissues. Courtesy S.B. Mathur

produce lemon-shaped sporangia which measure 60–100×30–65 μm (Shurtleff 1980). Sporangia germinate in water at 10–25 °C and form motile zoospores with two flagella. Sometimes the sporangia germinate and produce secondary sporangia (Payak et al. 1970; Chahal and Singh 1993). Upon germination the germ tube of zoospores infects the wheat plant and the pathogen continues its systemic growth and multiplication in the plant until it reaches heading. According to Bains and Jhooty (1986), the pathogen could be seed transmitted.

Control

Crop rotation with non-host cereal plant species helps in reducing the severity of the disease. As a precaution use of seeds coming from infested fields should be avoided. In water-logged areas, improved drainage becomes necessary. In India some cultivars like HD 2009, HD 2364, HD 2384, WG 357 and KSML 3, were reported to be resistant (Chahal and Singh 1993).

6.1.3 Leaf Rust

Leaf rust or brown rust of wheat is the most commonly spread disease in the world (Morgounov et al. 2012). Johnston (1931) reported a yield loss of 55 % in some susceptible wheat cultivars. Eversmeyer and Browder (1974) reported a loss in yield

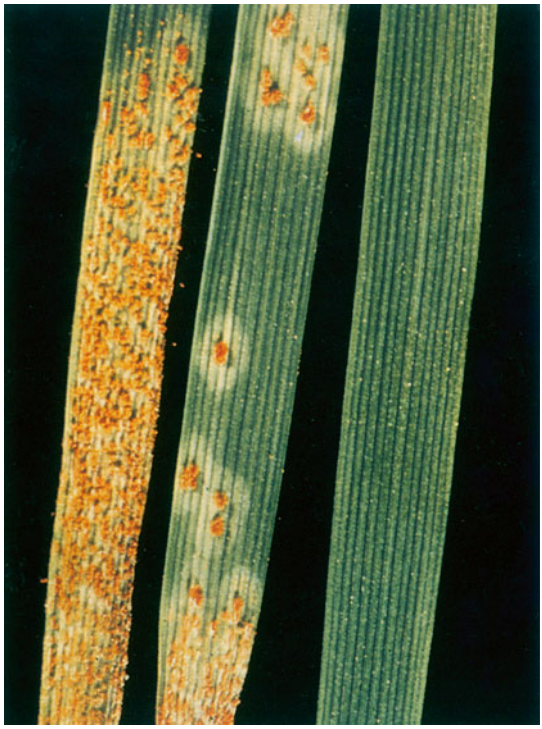

Fig. 6.4 Symptoms of leaf rust

of 44 % due to the joint infections of leaf rust and stem rust. In Brazil, Barcellos (1982) and Mehta and Igarashi (1985a) reported a loss of 50 % and 72 % respectively. In Bolivia, the yield loss due to leaf rust could vary between 51 and 64 % (Languidey and Barea 1993).

Symptoms

The uredia are formed on both sides of the leaf. The rust pustules are scattered, circular to slightly oblong and represent orange color (Fig. 6.4). The telia are small, oblong, formed mainly along the veins and remain covered by epidermis for a long period. The sexual (perfect) form occurs rarely on *Thalictrum* sp. but its presence in Brazil is not yet known. In severe cases of infection roots and grain size and weight are reduced. This rust pathogen also attacks barley, *Aegilops* and *Agropyron* spp. (Bockus et al. 2010).

Causal Organism and Epidemiology

Leaf rust is caused by *Puccinia triticina* Ericks. (syn. *P. recondita* Rob. Ex. Desm. *f. sp. tritici*, *P. rubigo-vera tritici* (Ericks.). Carleton, *P. rubigo-vera* (DC) Wint.

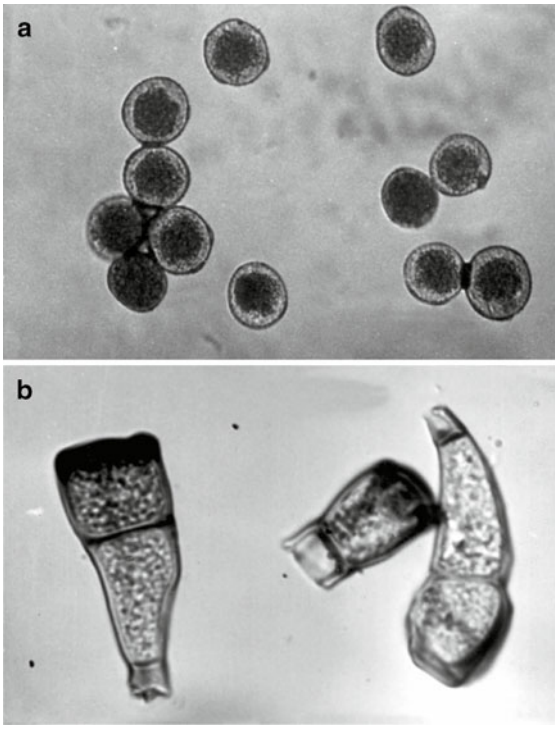

Fig. 6.5 (**a**) Uredinospores; and (**b**) teliutospores of *P. triticina*

and *P. triticina* (Ericks). The uredinospores are brown, round and measure 16–20 µm in diameter. On germination they form an appressorium and infect the plant through stomata. The teliutospores are rarely produced, dark brown with 2–3 septa and are similar to those of yellow rust fungus (Fig. 6.5a, b). The alternate host of *P. recondita* where it completes the life cycle is similar to stem rust and exists only in Europe and the United States (Saari et al. 1968; Bockus et al. 2010). The alternate hosts are; *Anchusa italica* Retz, *Clematis mandscurica* Rupr., *Isopyron fumarioides* W., *Thalictrum flavum* L., *Thalictrum foetidum* L., *Thalictrum japonicum* Thumb. and *Thalictrum speciosissimum* Loeft. (Roelfs et al. 1992; Eversmeyr and Kramer 2000).

Leaf rust races are identified using a standard differential set of cultivars or cultivars that have a combination of resistance genes (Lr-genes). Over 200 races of this fungus are reported in the world (Kolmer et al. 2012). The parasite *Darluca filum* (Biv.-Bern. Ex. Fr.) Cast., imperfect stage of *Eudarlucca cacicis* (Fr.) Ericks., attacks the rust under natural conditions. This fungus grows within the rust pustules, stops the development of the pustule and can be easily recognized by its black color.

Leaf rust develops in a range of atmospheric conditions. The infection normally starts at the growth stages between 8 and 9 (Table. 6.2), however, in some cases the infection can take place much earlier as well. The primary infection comes from the uredinospores blown by wind from other States or countries where the wheat crop is sown early. Nonetheless, the rust-free period is epidemiologically very important. It is a period during which no wheat crop or wheat plant exist in the field. The leaf rust pathogen also survives on the volunteer wheat plants during the off-season (Reis 1991a). In Brazil, wheat is sown in the month of March in the Central-West region and later until July in the Southern region (São Paulo, Paraná, Santa Catarina and Rio Grande do Sul). This long period favors the inter-zonal movement of uredinospores and reduces the wheat free period.

Like stem rust, presence of free water is necessary for uredinospore germination and infection. Although rust infections can take place in a wide range of temperatures, the most favorable temperature is between 16 and 18 °C. To build up epidemics, three factors are necessary: presence of susceptible cultivar, constant presence of inoculumand favorable weather conditions including the presence of free water on the leaves. As mentioned elsewhere, this forms a disease triangle. Besides, light is a decisive factor and has a marked influence on the uredinospores germination and infection. Zadoks (1967) verified that 63 % of the spores germinated in the dark while only 10 % germinated in the presence of light and the inhibition of the infection process occurred at the time of penetration.

The infection cycle of *P. triticina* is completed within 10 days. While studying spore production and sporulation period of *P. triticina* Mehta and Zadoks (1970) verified the high production potential of this pathogen. They showed that the dry weight of the spores produced during the sporulation period was equivalent to the dry weight of the spore-producing leaf and the maximum period of sporulation on the primary leaves was 72 days. The long sporulation period is interpreted as a survival mechanism of the fungus and has an important role in the epidemiology of the disease. Mehta and Zadoks (1970, 1971), reported that the maximum spore production was observed when the pustule density was 920 per leaf. Walker (1969) reported that if a level of 1,000 pustules per leaf is reached a yield loss of 29 % can be predicted.

Mehta and Igarashi (1978) reported that there exists a strong correlation between the latent period and the rate of infection, the longer the latent period the lower will be the rate of infection (Fig. 6.6). The uredinospores are the repeated spores and are the only ones responsible for building up an epidemic through the multiple spore production cycle. The life cycle of *P. triticina* is shown in Fig. 6.7.

Control

Genetic resistance is the best way to control this rust. While specific resistance genes like *Lr 19, Lr22, Lr29, Lr32 and Lr 33* were effective for several years, most of them have already superseded by the emergence of new pathotypes

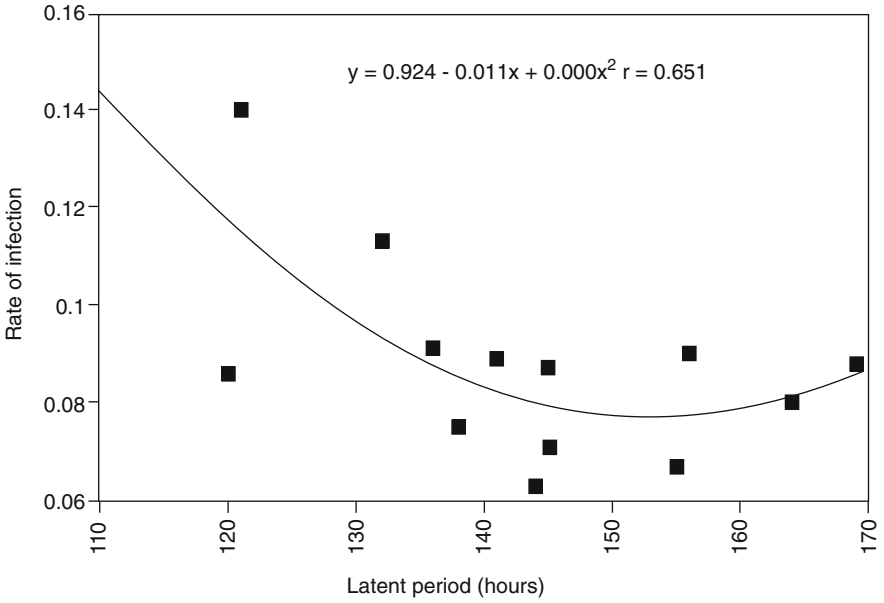

Fig. 6.6 Correlation between the latent period and the rate of infection of *P. triticina. Source*: Mehta and Igarashi (1978)

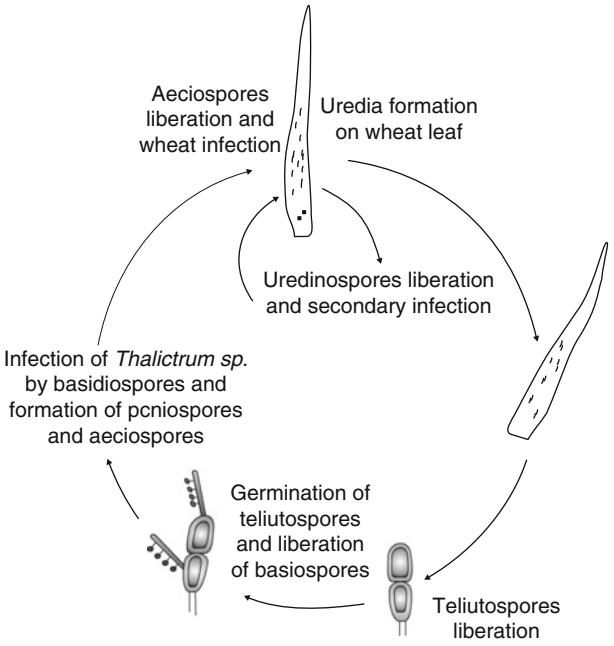

Fig. 6.7 Life cycle of *P. triticina*

(McIntosh et al. 1995, 2003). CIMMYT's germplasm development program has developed several cultivars with an adequate level of durable resistance for leaf rust that are being used in some developing countries (Van Ginkel and Rajaram 1993). The present strategy is to use a combination of specific and non-specific genes to achieve durable resistance. Mehta and Igarashi (1978) identified some cultivars with non-specific genes based on the period of incubation (latent period) and the rate of disease epidemic progress both under natural and controlled glasshouse conditions. According to these authors, cultivars BH 1146, IAC 5-Maringa, and IAS 20 probably have non-specific genes. Other cultivars with slow rusting characters were Genaro 81, Seri 82, Myna "S" and Kauz. According to Rajaram et al. (1988), cultivars Genaro 81, Opata 85and Pavan 76 have the complex gene *Lr13*. The slow rusting cultivars developed over past 50 years which include *Lr34/Yr18* and *Lr46/Yr29* and Pavan 76 with leaf rust and yellow rust resistance genes *Lr46/Yr29* have been effective since their release in 1976 (Navabi et al. 2003; William et al. 2007).

The CIMMYT bread wheat line Saar has good levels of resistance to leaf rust and yellow rust (*Puccinia striiformis* f. sp. *tritici*) based on *Lr34/Yr18* in combination with other minor genes. Lillemo et al. (2007)) reported that there exists a strong correlation between the leaf tip necrosis a phenotypic marker for *Lr34* and the powdery mildew (*Blumeria graminis* f. sp. *tritici)*, resistance in cv. Saar. They further concluded that resistance to yellow rust, leaf rust and the powdery mildew, is not only confined to *Lr34* but could be a general phenomenon of leaf tip necrosis associated resistance genes including *Lr46* and *YrLrPrII*.

Besides genetic resistance, leaf rust is controlled by one or two timely applications with some officially recommended systemic fungicides either alone or in combination with non-systemic fungicides (Mehta 1993; EMBRAPA 2011).

6.1.4 Phoma Leaf Spot

The Phoma leaf spot is not a very common disease of wheat. It occurs in India, the USA and Brazil (Neema et al. 1971; Hosford 1975a). In Brazil, it was observed during 1972 and 1976 and is considered as a secondary disease. Occasionally, some leaf spots are observed in some fields, more often in the seedling stage than in the adult plant stage, probably because of the seed transmission.

Symptoms

On the leaves the initial oval spots of about 1 cm in size are observed with some pycnidia in the center of the lesion (Fig. 6.8). Pycnidia are also observed on glumes but without any specific symptoms. It is a seed transmitted disease (Morgan-Jones 1967).

Fig. 6.8 Symptoms of Phoma leaf spot (*Phoma insidiosa*)

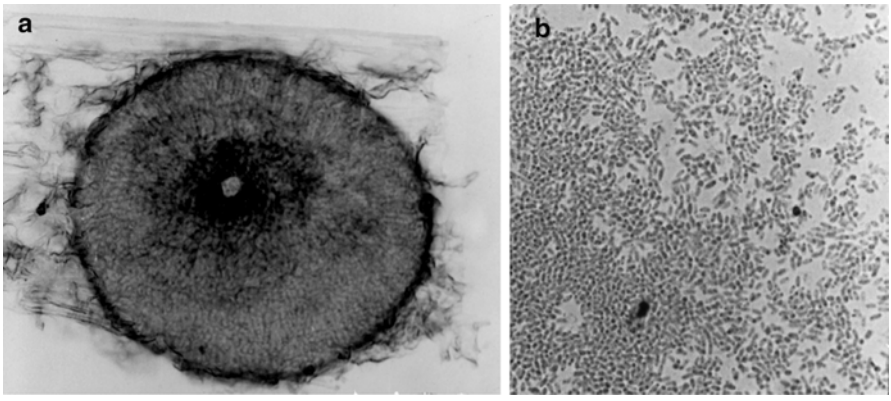

Fig. 6.9 (**a**) Pycnidium; (**b**) pycnidiospores of *P. insidiosa*

Causal Organism and Control

Phoma leaf spot is caused by *Phoma insidiosa* Tassi and *P. glomerata* (Corda) WR. and Hachapf. The pycnidia are globose, black and smaller than the pycnidia of *Septoria* spp. Pycnidium and pycnidiospores are numerous, oval to ellipsoid. Pycnidia measure 2.5–6.0 μm (Fig. 6.9a, b) (Morgan-Jones 1967; Punithalingam

and Holliday 1972). The pathogen grows well on common culture media with abundant pycnidia and pycnidiospores.

Both *Phoma* spp. attack several hosts and can also survive saprophytically on crop residue. The disease is favored by cold and humid weather (20 °C). Being a disease of secondary importance no control measures are recommended.

6.1.5 Pink Snow Mold

Pink snow mold occurs especially in temperate countries like the USA, Canada, Scandinavia and some countries in Western Europe. Although it is a cold loving fungus, it also occurs in the southern region of Paraná (Ponta Grossa), Brazil, where it was observed for the first time on commercial wheat, during the years 2000–2002, in an epidemic form. According to McBeath et al. (1993), the fungus requires at least 2 months of snow cover to cause severe damage (Fig. 6.10a, b), however, in Brazil, such conditions do not exist and the disease occurs in severe form. This suggests the existence of different races (biotypes) of the pathogen in different geographic areas of the world. Although the disease can cause severe damage to the crop, exact data on yield losses are not available (Cassini 1981; McBeath et al. 1993).

Symptoms

The disease is more frequently observed on leaves, leaf sheaths and spikes. On the leaves and leaf sheaths thick masses of aerial mycelium together with abundant salmon-colored sporodochia (conidia) (Richardson and Zillinsky 1972), are observed (Fig. 6.10a, b).

Causal Organism and Epidemiology

The pathogen of pink snow mold is referred to by several names. like: *Fusarium nivale* (Fr.) Ces.; the perfect stage - *Calonectria nivale* Schaf. [syn. *Microdochium nivale* (Fr.) Samuels and I. C. Hallett; *C. gaminicola* Berk and Br., *Griphosphaeria nivale* (Schaf) Mul. and van Arx, *Micronectriella nivalis* (SCAF.) Booth; *Monographella nivalis* (Schaffnit) E. Muller].). However, the pathogen is normally referred to as *Fusarium nivale*. The fungus grows on common culture media at low temperatures. 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 (Fig. 6.10d). The mycelial colonies appear as white to peach-colored because of the pinkish sporodochia. Microconidia or clamydospores are absent. The macroconidia are small and measure 2.8–4 × 16–25 µm, 1–3 septate. The perithecia are immersed, globose and contain clavate ascospores and

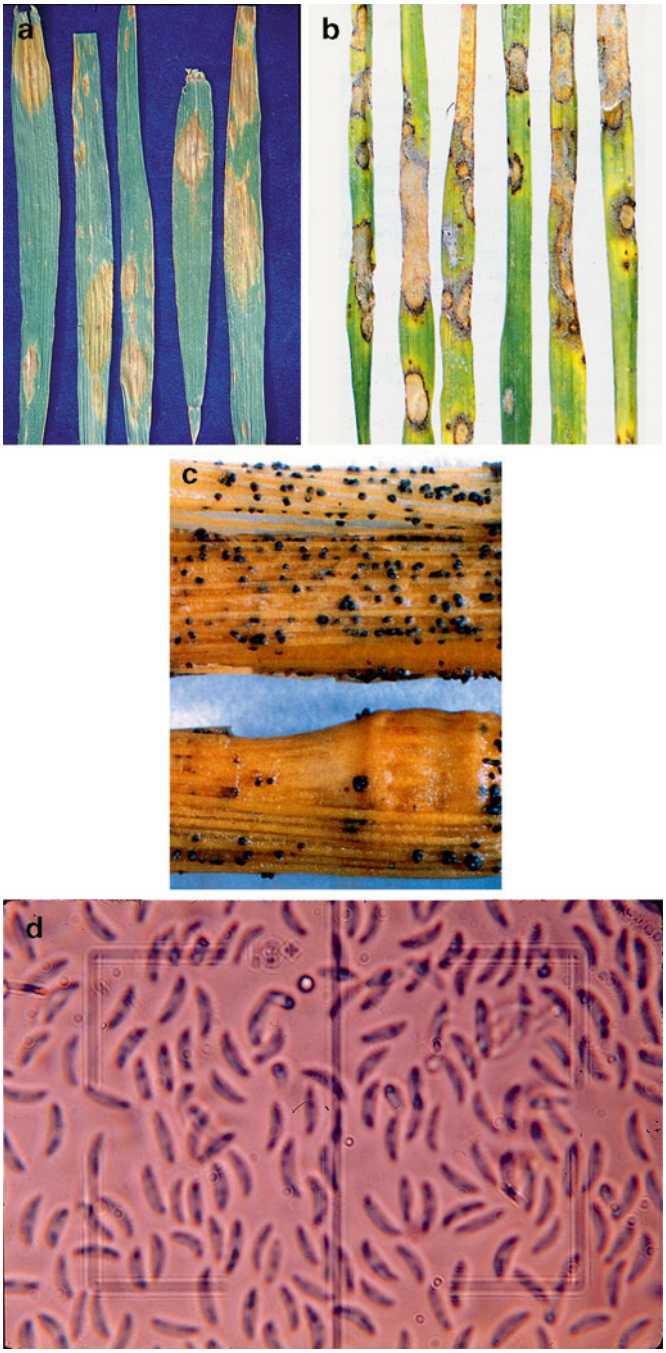

Fig. 6.10 (**a**) Symptoms of pink snow mold (*Fusarium nivale*) on wheat leaves in Brazil; (**b**) symptoms in the USA; (**c**) perithecia of *Microdochium nivale* on leaf sheath (Courtesy S.B. Mathur); (**d**) conidia of *F. nivale*

according to McBeath et al. (1993) measure 120–180 × 100–150 µm (Fig. 6.10c). The ascospores are ellipsoidal, 1–2 septate and measure 10–17 × 3.5–4.5 µm (Lebeau 1968; Booth 1971).

Two varieties of *Microdochium nivale* like *M. nivale* var. nivale and *M. nivale* var. majus are also known to cause FHB (Nicholson et al. 2007). *F. nivale* is a seed as well as a soil-borne pathogen. The spread of the disease is through wind and rain splashing. Cool (10–15 °C) and cloudy weather followed by rain for few days is ideal for the development and dissemination of the disease (Nakajima and Abe 1996). Conidia play an important role in the epidemiology of the disease. Although infections may occur by conidia as well as by the ascospores, the spread of the disease from one field to another is through the conidia. In Brazil, the disease is sporadic and occurs in localized areas and hence has not attracted attention for research. A comprehensive literature review about this disease has been presented by Lebeau (1968), Cook and Bruehl (1968) and McBeath et al. (1993).

Control

Use of healthy seed and crop rotation with leguminous crops would help in controlling the disease.

Hani (1981) cited by McBeath et al. (1993), suggested some seed health testing methods to identify and discard the contaminated seed lots. A simple method for seed health testing as suggested by McBeath et al. (1993) is to incubate the seed on moist filter paper for 14 days at 10 °C followed by 3 days at 20 °C. *F. nivale* is identified by the browning of roots and coleoptiles and by presence of mycelium and typical spores. As yet no tolerance limit as yet is proposed for the level of seed contamination.

Several effective fungicides are available on the market and may be used whenever necessary to complement other control measures. When the climatic conditions are favorable it becomes very difficult to control the disease by fungicidal sprays. The spore production potential of this pathogen is very high. In Brazil, during the epidemic years (2000–2002), three applications of a systemic fungicide mixed with contact fungicide failed to satisfactorily control the disease.

Cultivar resistance is always preferred to control the disease, but there is no information about the level of resistance of wheat cultivars in the Latin American region. Among Mexican cultivars, durum wheat is more susceptible than common wheat (Zillinsky 1983).

6.1.6 Powdery Mildew

Powdery mildew is a well-known disease of wheat and occurs all over the world (Morgounov et al. 2012). Yield losses can reach up to 40 %, but early infections may cause death of seedlings. In Brazil yield losses were estimated to be 55 % on

highly susceptible cultivar IAS 54 (Linhares 1988). The disease has been increasing for the past 2 decades in Brazil, but its severity depends on the climatic conditions of the year. In France yield losses caused by powdery mildew in triticale could reach up-to 40 % without control measures (Matasci et al. 2012).

Symptoms

The characteristic symptoms of the disease are the appearance of white cottony mycelia with spores, sometimes covering almost the whole leaf area (Fig. 6.11a, b). Small whitish elongated spots occur on the stems, ears and awns. In fact, under severe infections all above-ground plant parts can be infected. Infected plants look weak and produce shriveled grains. In advanced stages of infection the presence of cleistothecia as small black bodies (points) interlaced within the light colored mycelia can be observed (Fig. 6.11c). Early infection at the seedling stage may damage the entire crop if not controlled appropriately, since it restricts root development and reduces the number of tillers.

Causal Organism and Epidemiology

Powdery mildew attacks wheat, barley and other hosts of Gramineae. *Blumeria graminis* 'forma specialis' *tritici* attacks wheat, while f. sp. *hordei* attacks barley. Powdery mildew of wheat is caused by *Blumeria graminis* f. sp. *tritici* (Syn. *Erysiphae graminis* Dc. f. sp. *tritici* E. Marchal, anamorph *Oidium monilioides* Link. It is an obligate parasite and is heterocious in nature. It belongs to the class Ascomycetes (order Erysiphales). On wheat leaf lesions, first the imperfect state of the fungus (*Oidium monilioides*) is observed. The conidia are born in chains on short conidiophores (Fig. 6.11d, e and Fig. 6.12a). Conidia are ellipsoid to oblong, unicellular, hyaline and measure 25–37 × 12–17 µm. The cleistothecia are globose, immersed in the mycelium and measure 139–250 µm in diameter. Ascospores are formed in asci, eight in number, elliptical, sub-hyaline and measure 20–25 × 10–15 µm (Fig. 6.12).

In nature, secondary infections are caused by air-borne conidia and ascospores. The pathogen survives in the soil as cleistothecia.

The conidia are short-lived. They germinate within a wide range of temperature, but ideally at 18–22 °C and relative humidity of 60–100 % (Ellingboe 1972). However, once the infection is established, dry periods are necessary for abundant and continuous spore production. In fact, cyclic wet and dry periods help in building up an epidemic. Upon conidial germination, a germination tube is formed and later an appressorium, which penetrates the epidermal cell and forms haustoria. Finally, secondary hyphae are formed which gives rise to new appressoria and haustoria and new colonies of the fungus are formed. The complete formation of haustoria takes 34–36 h (Masri and Ellingboe 1966a, b). The infection cycle is completed within 3 days. Once the infection is established, the spore production potential of this pathogen becomes very high.

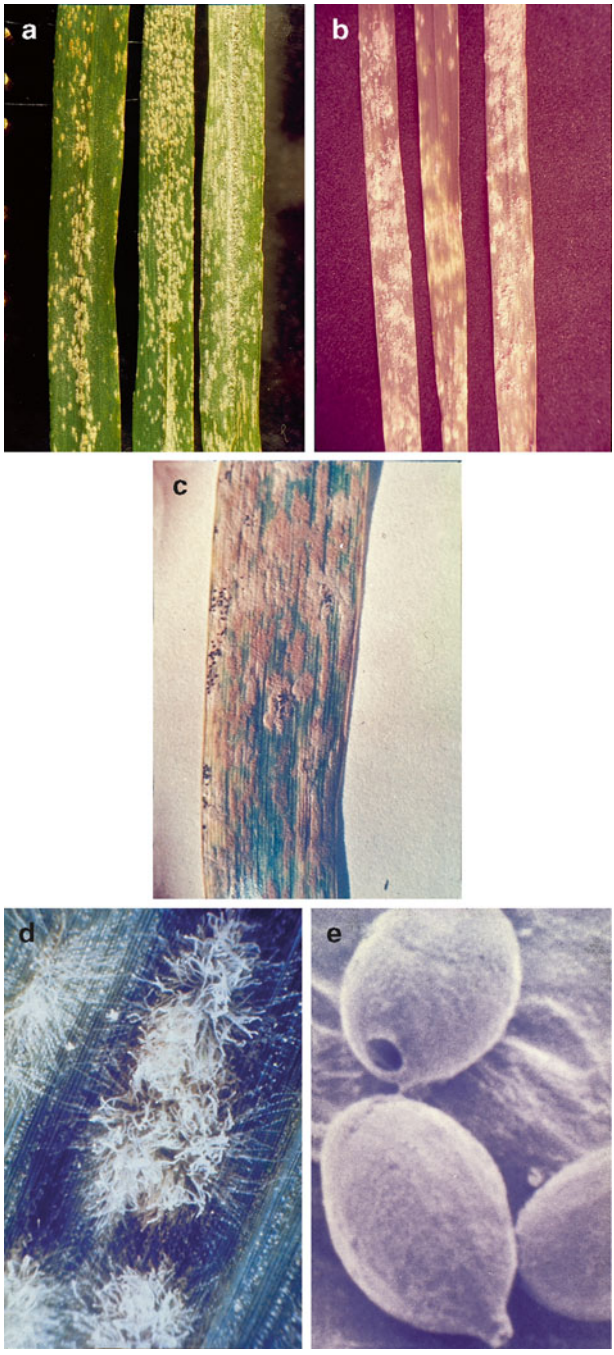

Fig. 6.11 (**a**, **b**) Initial symptoms of powdery mildew on wheat leaves; (**c**) advanced stage of powdery mildew infection showing formation of small, black clestothecia (sexual stage of *Blumeria graminis*); (**d**) powdery mildew colony on wheat leaf; (**e**) conidia of *B. graminis* (Courtesy BASF)

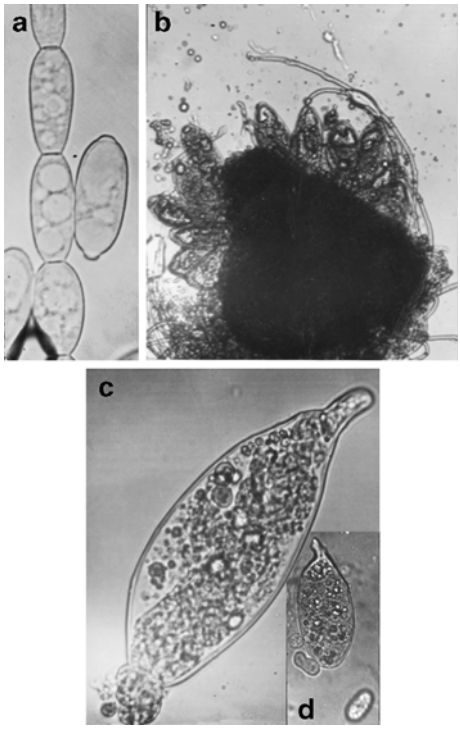

Fig. 6.12 *B. graminis*. (**a**) conidia in chain; (**b**) perithecium containing asci; (**c**, **d**) ascus containing ascospores

According to Shaner (1973a, b), there is wide variation as regards the lesion area and the chain of conidial production which determines or not the characteristics of "slow mildewing".

Conidia are produced in great numbers, are wind-dispersed and travel long distances and are epidemiologically important for the spread and development of epidemics (Nair and Ellingboe 1962, 1965). Although ascospores can cause primary infection, their role in the epidemiology of the disease is not well understood. Cleistothecia, however, would play an important role in creating new races of the pathogen. So far, few races of the pathogen are known to occur, however, several biotypes are frequently emerged. The life cycle of the pathogen is shown in Fig. 6.13.

Control

In general, use of "slow mildewing" cultivars is preferred over other methods of control. Resistance is governed by a single dominant gene and may be overcome in a short period of time due to the emergence of new races and new biotypes of the pathogen. A few "slow mildewing" cultivars like cv. Saar are available in several countries. Lillemo et al. (2007) reported that one or more common genetic factors

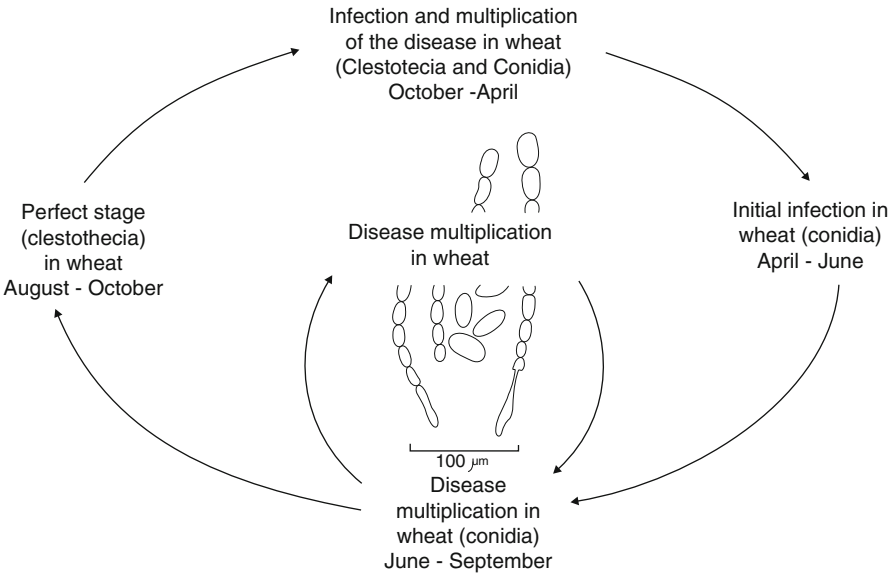

Fig. 6.13 Life cycle of *B. graminis*

were responsible for resistance in cv. Saar against leaf rust, yellow rust and powdery mildew. They believed that this resistance is probably offered by gene *Lr34*and that the combination of three genes *Lr34*, *Lr46* and *YrLrPrll* reduced the level of powdery mildew in wheat. Lillemo et al. (2007) also reported that despite never having been exposed to powdery mildew during the development of the line Saar, it has exhibited high levels of partial resistance to powdery mildew in Europe. In Brazil, some *Pm* genes have been found effective (Costamilan 2002, 2003, 2005; Costamilan et al. 2007; Zhu et al. 2005; Mohler et al. 2010).

Two powdery mildew resistance genes introduced from *Triticum carthicum* accession PS5 to common wheat were identified and tagged using microssatellite marker (Zhu et al. 2005). Mohler et al (2010) conducted genetic studies on the German winter wheat cultivar 'Cortez' to identify genes involved in powdery mildew resistance at seedling and adult plant growth stages using doubled haploid population Atlantis/Cotez. Analysis of association between molecular markers and powdery mildew severity in selected genotypes indicated the involvement of two genes on chromosomes 1A and 7B in seedling resistance.

Bai et al (2012) studied pyramiding adult-plant powdery mildew resistance QTLs in bread wheat using 21 F6 lines. Based on the phenotypic data these authors concluded that there were highly significant effects of QTL combinations on reducing powdery mildew. These authors however, suggested that the marker assisted selection will still require field testing for powdery mildew to validate the resistance.

Cultural practices such as crop rotation with non-host plant species reduce the severity of the disease. Application of excessive amounts of nitrogen fertilizers

should be avoided. Destruction of volunteer wheat plants, whenever possible, is also recommended. In Denmark for example, winter barley has been banned in order to break the "green bridge" and consequently break the disease cycle. Taking into consideration several problems provoked by crop residue destruction, its use is not advisable (see chapter on crop residue).

Several systemic fungicides are available and are highly effective against powdery mildew. In Europe, for example, use of systemic fungicides is a common practice. None the less, prolonged use of a single systemic fungicide for aerial applications may lead to the creation of resistant biotypes of the pathogen. Examples of such resistance are already known for benzimidazol (Schroeder and Provvidenti 1969; Vargas 1973). Some systemic fungicides may be used for seed dressing since they provide control of powdery mildew for a long time or at least until the boot stage.

6.1.7 Septoria Diseases

Septoria diseases of wheat include three diseases namely Septoria nodorum blotch, Septoria tritici blotch and Septoria avenae blotch. The first two diseases are some of the most important diseases of wheat in the world. A worldwide loss was estimated at US$1 billion and in the USA alone these diseases cause a yearly loss of over 1 % (Eyal et al. 1987). Normally, these diseases individually are capable of causing about 30–40 % loss in yield depending upon the country and the year. The third disease, Septoria avenae blotch is a relatively new disease of wheat and may cause appreciable yield losses but its occurrence is not as widespread as the other two diseases. In the following pages these diseases are described in brief.

Septoria Avenae Blotch

Septoria avenae blotch was first described by Johnson (1947), in Canada. Later, the disease was reported in the USA and in some other countries. It was reported from Brazil in 1975 (Mehta 1975a), but without causing any apparent damage to the wheat crop.

Symptoms

Septoria avenae blotch is mainly a leaf disease and the symptoms look somewhat similar to those of Septoria nodorum blotch. However, in the case of severe infections some lesions on the spike and stems can appear. The perithecia and pycnidia are formed on the same lesion. The lesions on leaf are elliptical to oblong with a whitish center. As with *S. nodorum*, perithecia are produced on the same lesion (Fig. 6.14).

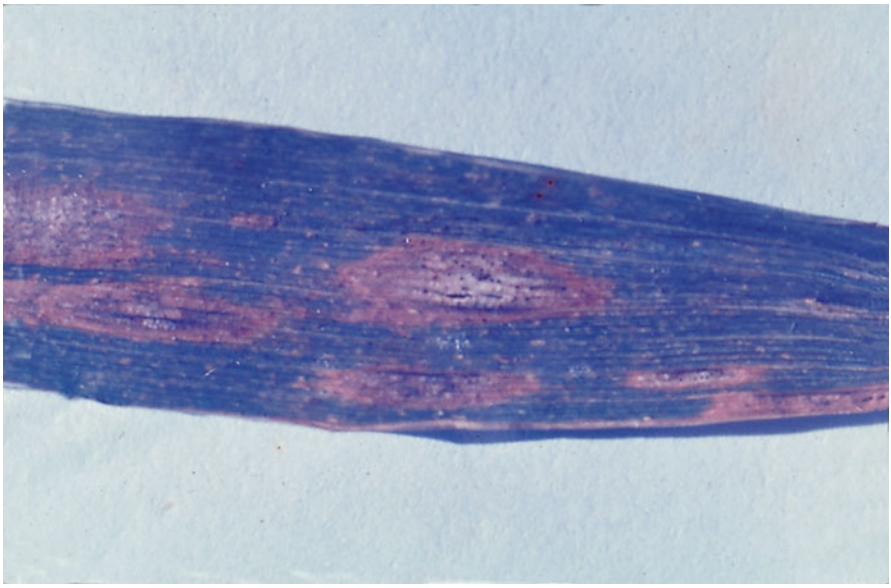

Fig. 6.14 Symptoms of Septoria avenae blotch on wheat leaf

Causal Organism and Epidemiology

The disease is caused by *Leptosphaeria avenaria* Weber f. sp. *triticia* T. Johnson (anamorph *Septoria avenae* Frank f. sp. *triticea* T. Johnson). It differs from *Leptosphaeria nodorum* and *Mycosphaerella graminicola*, based on its ability to infect wheat, barley and oats and also its ability to produce perithecia more easily than the other two pathogens.

The pycnidia measure 93–261 μm in diameter and the pycnidiospores measure 23–29×2.8–4.7 μm. The pycnidiospores are almost cylindrical, hyaline, typically three-septate (rarely four-septate), erect, slightly curved. The fungus is homothallic. The perithecia are globose with an ostiol, dark brown to black and measure 44–80×8–11 μm. The ascospores are three-septate with little constriction at the septum and measure 16–28×4–6 μm (Hosford et al. 1969). Light is necessary for the production of perithecia. The pathogen also differs from *L. nodorum* in spore size and septation.

The fungus also attacks barley and other hosts belonging to Gramineae and survives on the crop residue under field conditions. The spread of the disease is through the wind-borne spores either from the secondary hosts or from the soil inoculum of ascospores or pycnidiospores (Hosford and Busch 1974). Like other Septoria species wet and cool weather is essential for the development of the disease. Johnson (1947) and Shaw (1957) reported that *S. avenaria* f. sp. *triticea* is

a week pathogen of wheat, whereas Hosford and Busch (1974) believed that the pathogen is important.

Control

Since the disease does not cause substantial yield losses, no specific control measures are suggested. Nonetheless, the control measures suggested for other two Septoria diseases could be practiced where the disease is endemic.

Septoria Nodorum Blotch

Septoria leaf and glume blotch was first described in England in 1845. Today, the disease occurs all over the world and especially in countries where cool weather persists during the wheat period. The pathogen attacks all above ground parts of the wheat plant. Severe epidemics have occurred in Europe and in the USA during the last few decades. According to Caldwell (1976), severe incidences of the disease were observed during 1973 and 1975, especially in dwarf wheats. In the States of Alabama and Georgia disease epidemics were registered in 1976 (Cunfer and Nelson 1976; Sapra et al. 1976). The disease is very important in north-western Europe. Yield losses of over 50 % were reported in England (Cook and Jones 1970a). According to these authors losses were also in the quality of grains. In the south of Bavaria (Germany) severe epidemics have caused losses between 10 and 30 %. Losses between 33 and 90 % were reported in the province of Buenos Aires and Santa Fé, Argentina (Annone 1990a). Galich (1981) also reported occurrence of Septoria blotch epidemics during the years 1943, 1944, 1949 and 1954. In the south of Brazil, the severe epidemics of 1972 and 1973 are well-known (Mehta 1975b; Rogenski et al. 2012).

Symptoms

The infected seedlings show elliptical dark-brown lesions on the coleoptile (Fig. 6.15). Such lesions may be confused with those produced by *Bipolaris sorokiniana*. Initially, lesions on leaves are small, elliptical with whitish center and dark brown margin. Well-developed lesions on leaves are elliptical, some-times fusiform and dark brown with whitish center along with pycnidia (Fig. 6.16). Pycnidia on the lesions are not formed if the weather is unfavorable for the disease. Later the lesions coalesce and become irregular. On stems and leaf sheath the lesions are linear or rectangular and appear dark brown to black. In advanced stages of infection of the nodes, abundant pycnidia can be observed with the naked eye. The nodes become shriveled and cause lodging of the plant. Infection of *Fusarium nivale* on nodes also causes lodging.

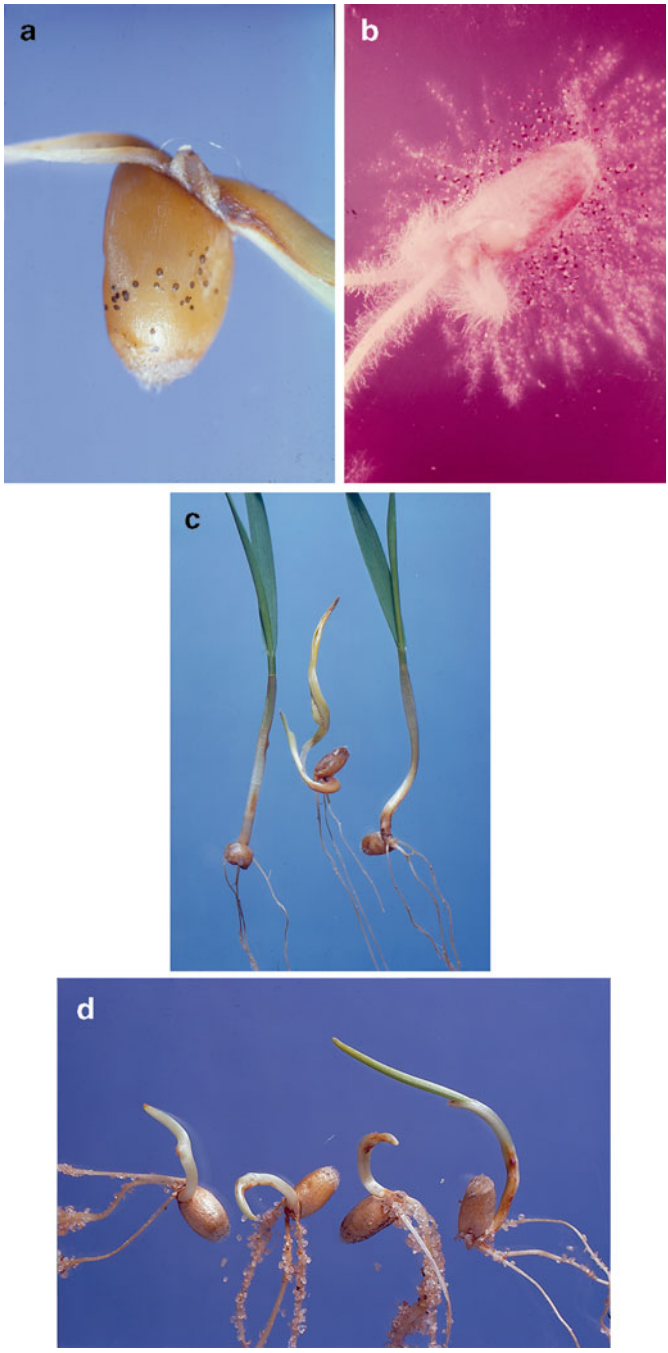

Fig. 6.15 Septoria nodorum blotch (*Septoria nodorum*) on wheat seed and seedlings. (**a**) pycnidial formation on seed; (**b**) pycnidial formation on culture media; (**c**) symptoms on seedlings (coleoptile) grown in sand; (**d**) seedling symptoms in the field. Courtesy P.D. Hewett

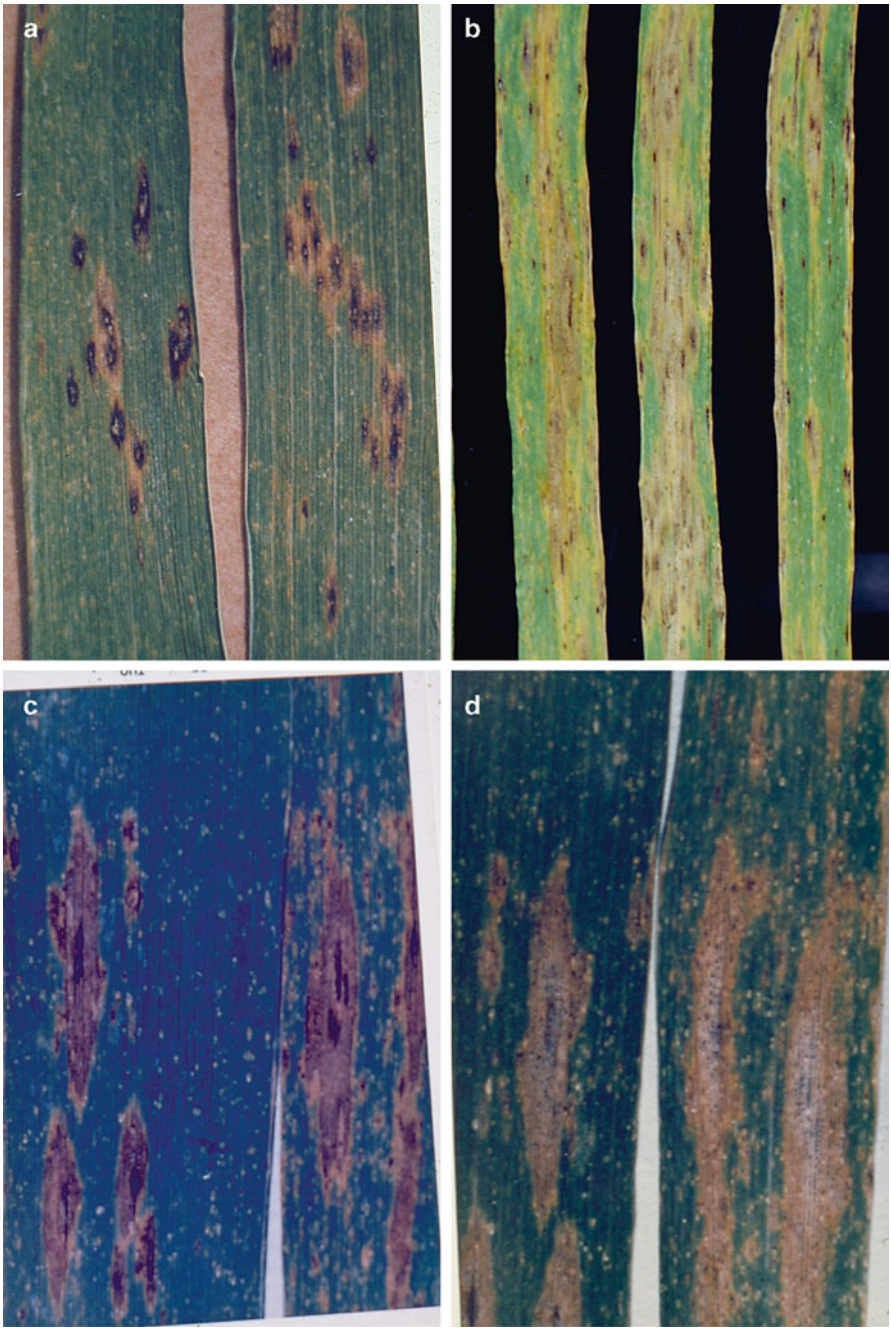

Fig. 6.16 (**a**, **b**) Initial symptoms of Septoria leaf blotch; (**c**, **d**) well developed lesions showing whitish center and presence of pycnidia

Fig. 6.17 (**a**, **b**) Septoria glume blotch on spikes and awns; (**c**) formation of perithecia of *Leptosphaeria nodorum*; (**d**) formation of pycnidia of *S. nodorum*

Infection of *S. nodorum* on glumes and awns is characterized by dark brown to black lesions starting from the top of the glumes and extending towards the base. As the disease advances, numerous pycnidia can be observed with the naked eye on the upper portion of the glumes (Fig. 6.17). In the absence of pycnidia, isolation of the pathogen becomes necessary for correct diagnosis, because glumes are also attacked by other fungi like *Fusarium* spp., *Blumeria graminis*, *Leptosphaeria avenaria*, as well as by melanosis—a physiological disorder. Infection of the glumes does not

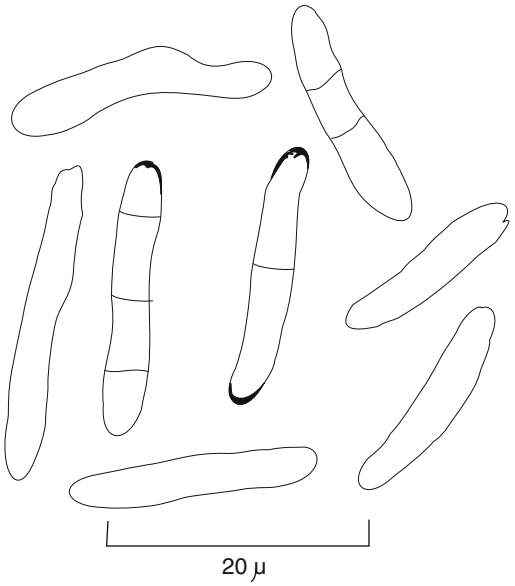

Fig. 6.18 Pycnidiospores of *Septoria nodorum*

necessarily mean infection of the seed. There is no correlation between the glume infection and seed infection (Hewett 1969; Friesen et al. 2009; Friesen and Faris 2010; Rogenski et al. 2012).

Causal Organism and Epidemiology

Septoria leaf and glume blotch is caused by *Leptosphaeria nodorum* (Syn. *Phaeosphaeria nodorum*; anamorph *Stagnospora nodorum* (Berk.) Castellani and E. G. Germano, *Septoria nodorum* (Berk.) berk and apud Berk and Br. (Syn. *S. glumarum* Pass. *Hendersonia nodorum* (Berk Petrak.) (McDonald et al. 2012)

The pycnidia are dispersed on the glumes, spherical, sub-epidermal, flattened at the base and measure 160–210 μm in diameter. The pycnidiospores produced naturally on the plant are almost cylindrical, 0–3 septate, hyaline, erect or slightly curved. In culture media pycnidia measure 22×311 μm in diameter and the pycnidiospores measure 12–26×2–6 μm. The pycnidial cell wall is parenchymatous, delicate and dark brown in color. In general, pycnidiospores measure 12–32×17–40 μm (Fig. 6.18) (Mehta 1993). Normally, most of the pycnidiospores are 0–1 septate.

The perithecia are black, flattened at the base spherical, sub-epidermal and are somewhat larger than the pycnidia (Fig. 6.19). Perithecia have a distinct ostiole and measure 133–240 μm. Asci are bitunicate, cylindrical with eight ascospores and measure 40–70×6–10 μm. The ascospores are triseptate, constricted at the extreme ends, hyaline and measure 12–22.5×3–4 μm (Fig. 6.20).

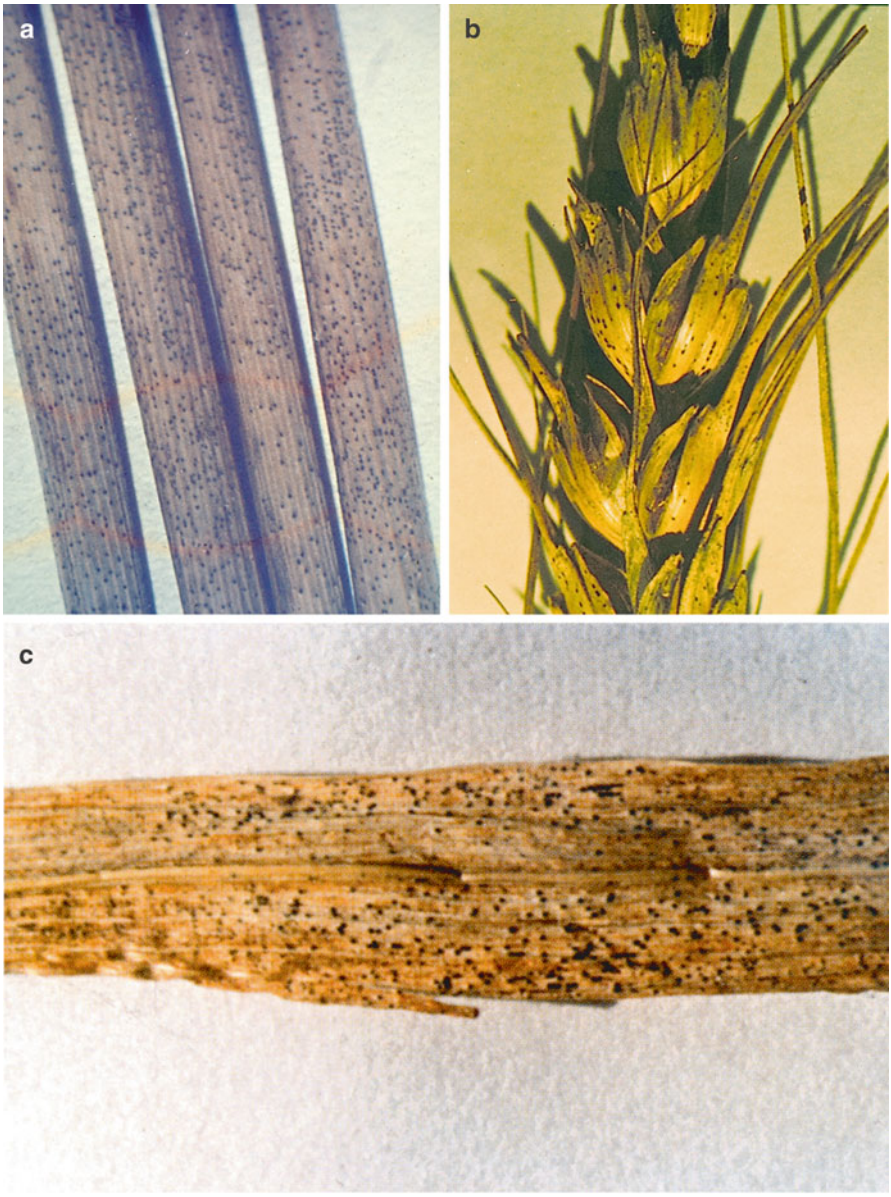

Fig. 6.19 Perithecia of *L. nodorum*. (**a**) on leaf sheaths; (**b**) on glumes; (**c**) on leaf

On artificial culture media the mycelium is at first white and later becomes slightly reddish. Pycnidiospores are produced on artificial media within 4–6 weeks (Hosford 1975b).

S. nodorum sporulates only in the presence of light. Prolonged periods of darkness inhibits the sporulation. Under artificial conditions, continuous light provokes

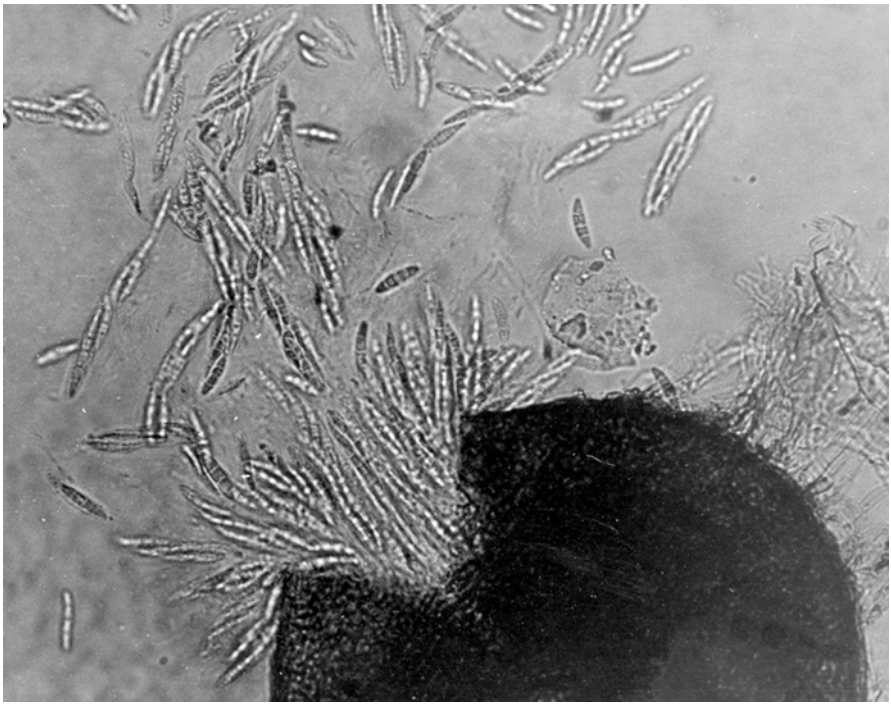

Fig. 6.20 Perithecium containing asci and ascospores of *L. nodorum*

sporulation, but alternative cycles of 12 h darkness and 12 h light are required for abundant sporulation. Details about laboratory and field techniques for Septoria diseases like; collection and handling of infected plant material, isolation of pathogen, maintenance of cultures, production of inoculum, inoculation procedures and disease assessment are very well described by Eyal et al. (1987).

Normally, under field conditions, it takes 11–14 days to complete the infection cycle when pycnidia are produced on leaves. During humid periods the pycnidia liberate spores in the form of cirrus and are dispersed by wind and rain splashing. The saprophytic phase of the pathogen is interesting. The pathogen takes less than half the time to sporulate as a saprophyte on the crop residue than to sporulate on living tissues as a pathogen. According to Von Wechmar (1966), spores remain viable for 8 months inside the pycnidium, whereas Weber (1922) reported that they can remain viable on the crop residue for 18 months. This indicates that the survival of *S. nodorum* depends on the climatic conditions of each country. Scharen (1964), reported that the pycnida produced on crop residue can release spores within 8 h when made wet and that the process of spore production continues for a long period if the crop residue is repeatedly dried and made wet. This discovery is epidemiologically very important since it explains the spore production potential of the pathogen in its saprophytic phase. The cyclic production of spores by *S. nodorum* explains the continuous source of inoculum in the field.

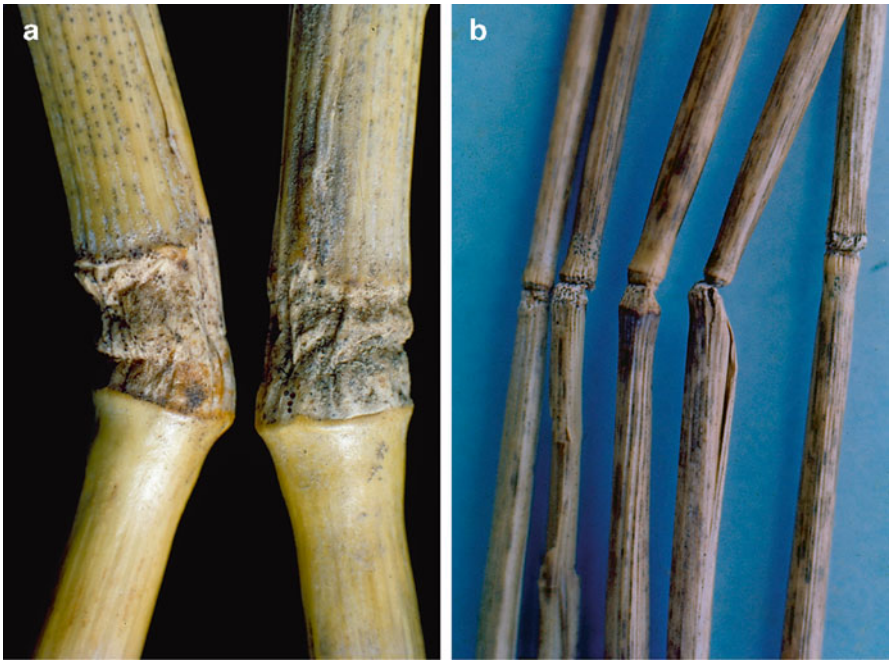

Fig. 6.21 (**a**, **b**) Septoria leaf blotch infection on nodes showing formation of pycnidia. (**a**, courtesy P.D. Hewett)

In Europe, for example, wheat stubble is burned in some countries, reducing thereby the inoculum present on crop residue. Nonetheless, residue burning does not completely eliminate the inoculum including the perithecia which can survive in the soil for a long period of time (Figs. 6.21 and 6.22). The perithecia liberate ascospores which serve as the source of primary infection along with the inoculum present in the seed.

Different host species of *S. nodorum* are known. The pathogen has a wide range of secondary hosts which may also be responsible for the primary source of inoculum (Shearer and Zadoks 1972). The life cycle of the pathogen is presented in (Fig. 6.23).

Differences in virulence among isolates are reported by some workers (Scharen et al. 1985; Friesen et al. 2009; Friesen and Faris 2010). Mahto et al. (2011), reported the existence of 21 different genes operating among the cultivars and affirmed that isolates from Brazil, Chile and Ecuador expressed high relative virulence.

Control

Some cultural practices like deep plowing especially in heavily infested field help reduce the amount of inoculum and the number of seedling infections. Crop rotations with oats, lupines and other leguminous species help to break the cycle of the pathogen and consequently reduce yield losses. Application of excessive amounts

Fig. 6.22 (**a**, **b**) Survival of *L. nodorum* on wheat crop residue

of nitrogen fertilizers should be avoided. As for any other disease, use of resistant or less susceptible cultivars help reduce the disease severity for the following crop.

Seeds originating from highly infested fields should be treated with fungicides. Seed health testing would help to discard heavily infected seed lots (Baker 1970; Eyal 1986; Cunfer et al. 1988).

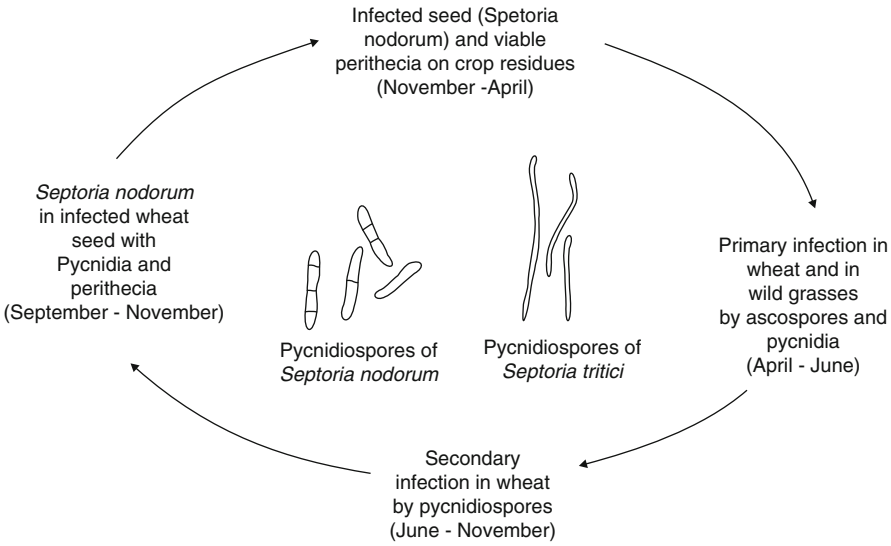

Fig. 6.23 Life cycle of *S. nodorum* and *S. tritici*

Mahto et al. (2011) evaluated local and commercial spring wheat cultivars and advanced breeding lines for resistance to three leaf spot diseases: spot blotch, septoria nodorum blotch and tan spot races 1 and 5, during 2009–2010. Their results indicated that 30, 31, 19 and 10 % of the tested wheat cultivars and advanced lines were resistant to Spot blotch, Septoria nodorum blotch and tan spot races 1 and 5, respectively.

Septoria Tritici Blotch

Septoria tritici blotch is reported in almost all the wheat growing countries. The disease was first described on wheat in 1842 (Eyal et al. 1987; Shaner 1976). After 1940, disease severity on wheat went on increasing in the USA, due to the generalized use of nitrogenous fertilizers. According to Caldwell (1976), the first epidemic of Septoria tritici blotch occurred in the State of Indiana in 1949. Later, in 1957, severe epidemics of the disease were also registered in the States of Arkansas, Missouri and Indiana. Gough and Smith (1976), reported the occurrence of severe epidemics in the State of Oklahoma in 1975 and the yield losses were between 10 and 20 %.

The disease was not important in Israel until the introduction of dwarf wheats which were highly susceptible to this disease. The yield losses were estimated to be between 20 and 50 % (Eyal 1976). The disease was considered important in Tunisia after the occurrence of severe epidemics during 1968 and 1971 (Ghodbane et al. 1976). In humid areas in the south and west of England the disease was very

important (Cook and Jones 1970a, b). Similarly, Sanderson (1972, 1976) reported the importance of the disease in New Zealand. According to this author, the occurrence of sexual stage of this fungus was epidemiologically important.

In South America the importance of Septoria tritici blotch was reported in several countries like Argentina, Chile, Uruguay and Brazil. In Argentina severe epidemics were registered in 1943, 1946, 1948, 1956, 1959 and 1979 (Galich 1981). A somewhat similar situation occurred in Chile. According to Mellado (1990), the increased severity of the disease in recent years was attributed to the use of semi-dwarf wheat cultivars, use of heavy amounts of nitrogenous fertilizers, the monoculture practice (wheat-soybean-wheat) and the early seeding of wheat. Because of such practices a loss in yield of over 14 %, was recorded (Burrows 1981). Heavy yield losses were also recorded in Uruguay, during 1960, 1976 and 1979, causing yield losses between 30 and 64 % (Abrinbana et al. 2012). According to this author, there was a correlation between the percentage leaf area infected and the percentage of yield loss. In Brazil, the losses in yield were estimated to be between 40 and 62 % depending upon the year and the wheat cultivar (Mehta 1989). Although severe epidemics were registered in the State of Rio Grande do Sul, during the 1970s and 1980s, exact data on yield losses were not estimated.

Thus, considering the occurrence of epidemics and consequently heavy reduction in wheat yields in different countries, over the past few decades, it becomes evident that Septoria tritici blotch has considerably more importance than the other Septoria diseases and hence deserves more research efforts towards its management.

Symptoms

S. tritici is basically a leaf pathogen and rarely causes infection on other parts of the wheat plant. At the initial stage of the disease the lesions are small, oblong to linear and light yellow in color. The lesions are narrower (Fig. 6.24) than those of *S. nodorum*. In advanced stages of infection the lesions are linear and parallel to the nerves and when the lesions coalesce the leaves become straw-colored and show the presence of numerous pycnidia. The pycnidia are black and are arranged in a linear fashion. Near wheat maturity, the pathogen produces the perfect stage represented by numerous perithecia on the leaves (Fig. 6.25a–c). The perithecia are black, slightly larger than the pycnidia and can be identified through a hand lens.

Causal Organism and Epidemiology

Septoria tritici blotch is caused by *Mycosphaerella graminicola* (Fuckel) Schroeter (anamorph *Septoria tritici* Rob. and Desm.) (Sanderson 1976).

The pycnidia are globose to oblong, black with an ostiole and sometimes form two kinds of spores, the microconidia and the macroconidia. The macroconidia (Pycnidiospores) are hyaline, filliform, 2–7 septate, slightly curved and measure

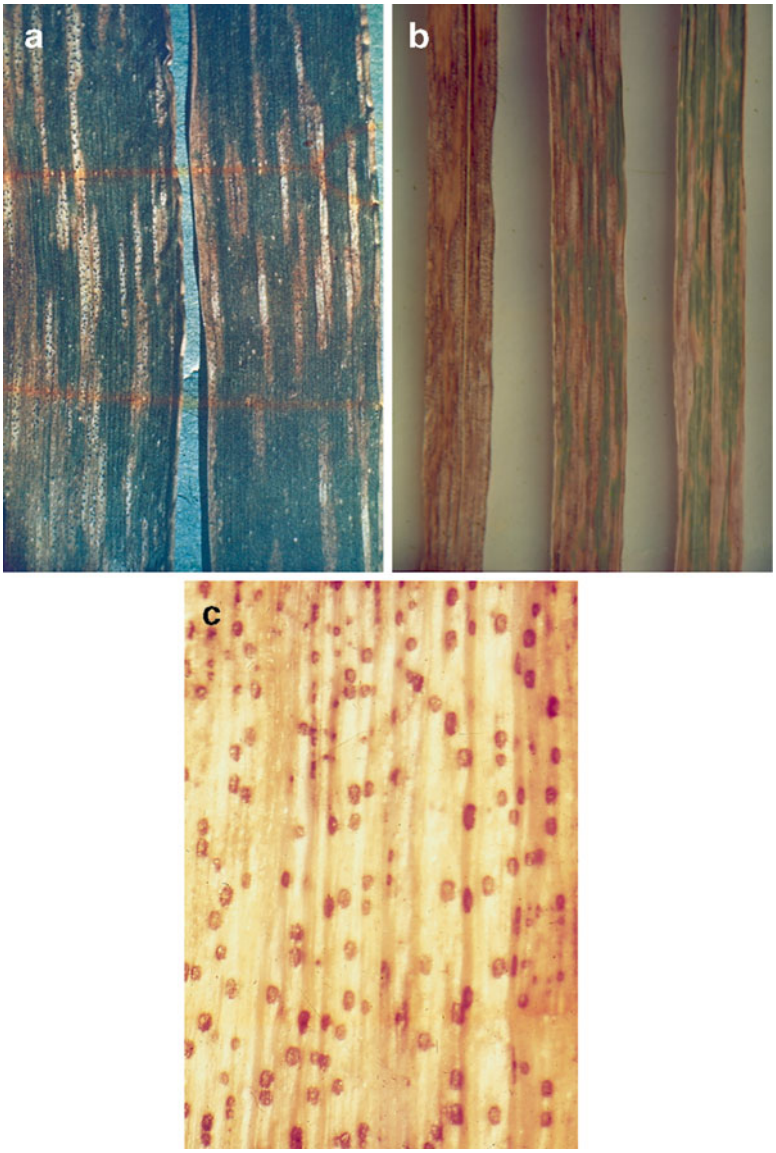

Fig. 6.24 (**a**–**c**) Symptoms of Septoria tritici blotch on wheat leaves showing formation of pycnidia almost arranged in rows

43–70 × 1.5–20 µm (Fig. 6.26). The microconidia are unicellular, curved, hyaline and measure 5–6 × 0.3–1.0 µm. On common culture media (PDA) the fungus colony is reddish, mucous with an appearance like a bacterial or yeast colony. The fungus colony consists of a dense mass of conidia (macroconidia) formed through budding and it does not normally form pycnidia.

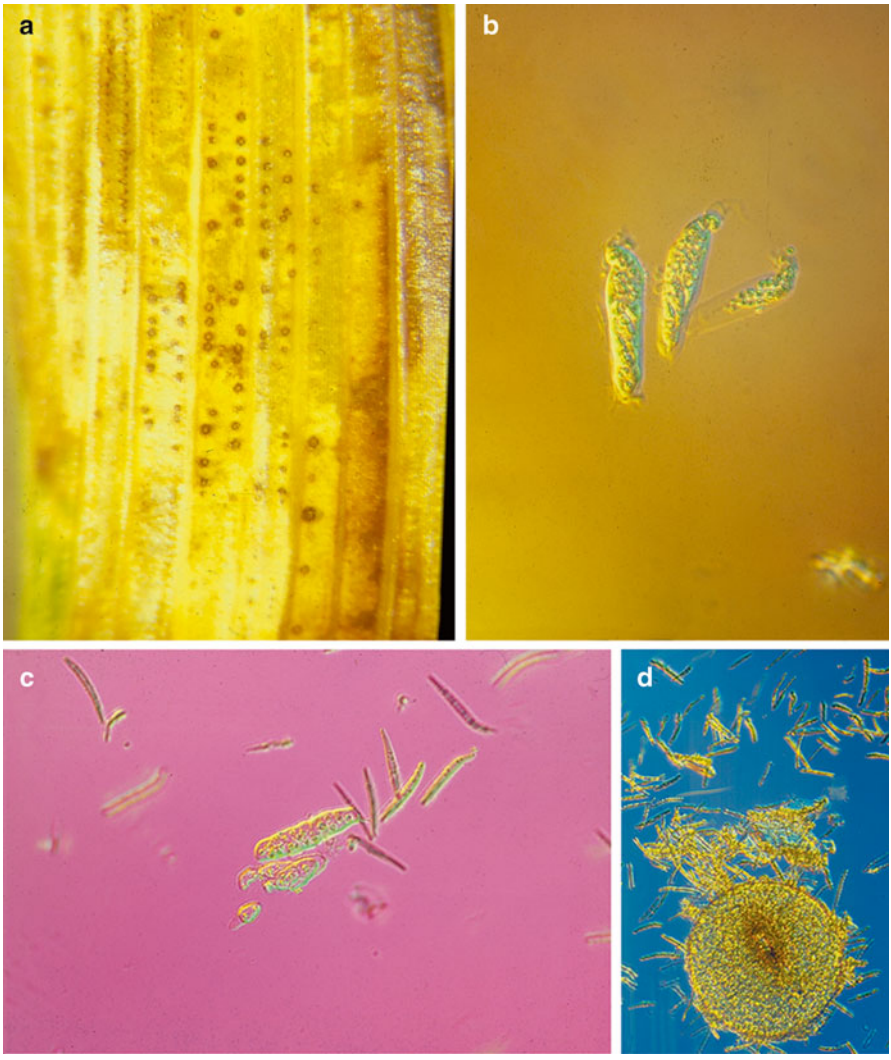

Fig. 6.25 (**a**) Formation of pycnidia (*Septoria tritici*) and perithecia (*Micosphaerella graminicola*) on leaf sheath; (**b**) asci; (**c**) asci and ascospores; (**d**) perithecium containing asci and ascospores

The perithecia are sub-epidermal, globose, dark brown and measure 68–14 µm in diameter (Fig. 6.25d). The perithecia contain bitunicate asci and measure 30–40 × 11–14 µm. The asci contain hyaline ascospores with one septum and measure 9–16 × 2.5–4.0 µm (Sanderson 1976). Physiologic specialization has been reported in the USA, Australia and Uruguay (Eyal et al. 1987).

Pathogenic variability within the isolates of *S. tritici* has been observed in Argentina and Uruguay (Cordo and Arriaga 1990; Perello et al. 1990; Abrinbana

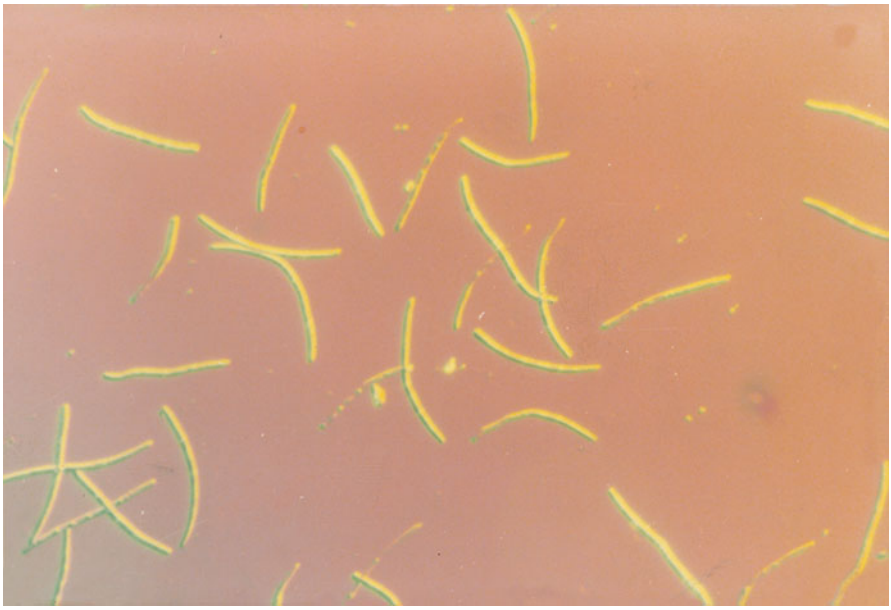

Fig. 6.26 Conidia of *S. tritici*

et al. 2012). Sanderson (1976), established the relationship between *M. graminicola* and *S. tritici* for the first time and reported *M. graminicola* as the perfect stage (sexual stage) of the fungus *S. tritici*. Later, the perfect stage (*M. graminicola*) was reported from England, Australia and Brazil (Scott et al. 1988; Mehta 1989). The genetic diversity and population structure of *S. tritici* was reported by El Chartouni et al. (2011), Kelm et al. (2012), Ghaffary et al. (2012) and Simon et al. (2012).

Shaner (1976) reported that the optimal temperatures for germination and infection are 22 °C and 21 °C, respectively, although infection can normally occur from 16 to 17 °C. Renfro and Yong (1956), reported that under field conditions a leaf wetness period of 15 h is necessary for infection, whereas the optimal period was 35 h followed by 2 days of high humidity.

The pathogen is seed-transmitted. Fig. 6.27 shows infected wheat seed with pycnidia and perithecia of *M. graminicola*. In the field, the primary infection comes from the secondary hosts, from the volunteer plants, as well as from the pycnidia and perithecia surviving on the crop residue. Although, the perfect stage (*M. graminicola*) is known to exist in nature, its role in the epidemiology of the disease is not well understood. Shaner (1976) reported that the pathogen survives on the crop residue from one season to another. Brokenshire (1975a, b), reported the susceptibility of some species of Gramineae to *S. tritici.*

Fig. 6.27 Infected wheat seeds showing formation of numerous perithecia of *Micosphaerella graminicola*

Control

van Beuningen and Kohli (1990) reported that it was possible to identify germplasm with some resistance to *S. tritici* in the Southern cone region of Latin America. According to these authors, the resistance is derived from crosses using Russian wheat Kavkaz with translocation 1B/1R. However, Eyal (1976, 1986) and Eyal et al. (1987), believed that the resistance does not reside in the translocation 1B/1r and that the resistance of Bobwhite was superior to that of progenitor Aurora (Christiane et al. 2012; Goodwin 2012).

Recently new broad spectrum resistance to Septoria tritici blotch derived from synthetic hexaploid wheat was reported by Ghaffary et al. (2012) and Simon et al. (2012). Results of Ghaffary et al. (2012) confirm that common wheat progenitors might be a rich source of new Septoria tritici blotch resistance genes/QTLs that can be deployed in commercial breeding programs.

Disease resistance and escape to the control of Septoria tritici blotch was studied in a set of 226 lines including modern cultivars by Arraiano et al (2009). According to these authors while greater plant height was strongly associated with reduced severity of the disease some lines with low mean levels of the disease was due to unknown genes for partial resistance.

Biological control of Septoria tritici blotch on wheat by four *Trichoderma* spp. strains under field conditions was studied in Argentina (Perello et al. 2008).

Promising results using *Trichoderma* spp. for biocontrol of Septoria tritici blotch were obtained by these authors especially when used as an integrated approach to the problem.

6.1.8 Selenophoma Leaf Spot

Selenophoma leaf spot also known as Halo spot is a common disease of grasses and of some cereals. It attacks wheat and is considered a secondary disease. However, Holton (1965), reported severe incidence on cv Gains. In Brasil, the pathogen occurs in association with *Septoria tritici*.

Symptoms

Selenophoma spots on leaves are small, oblong and gray in color with numerous pycnidia. The pycnidia are smaller than the pycnidia of *S. tritici* (CMI 1973).

Causal Organism and Control

Selenophoma leaf spot is caused by *Selenophoma donacis* (Pass.) Sprague and A. G. Johns. The pycnidia are globose, dark brown and measure 90 µm in diameter. The pycnidiospores are unicellular, hyaline and measure 13–22 × 3–4 µm.

Considering a secondary disease, no information is available about its etiology and thus no specific control measures are recommended.

6.1.9 Spot Blotch

Spot blotch occurs throughout the world and it is especially severe in the tropical and semi-tropical countries. It is most severe in warmer non-traditional wheat growing regions and is of special importance in Bangladesh, Bolivia, Brazil, East India, south-east China, south-east Australia, Paraguay and Zambia. Yield losses from 40 to 85 % have been reported in the Philippines and Zambia respectively (Raemaekers 1988; Kumar et al. 2002).

In India, spot blotch was reported to be severe in some States in 1930. In 1971, the majority of semi-dwarf wheat cultivars (triple gene) showed high susceptibility. Later, the disease extended to other regions with higher severity on some cultivars like NP 884, NP 852, Lerma Rojo and S 227 (Joshi et al. 1969). Recently, the disease has become still more important in South Asia's intensive irrigated wheat-rice production system, due to high temperature and high relative humidity prevailing in that area (Kumar et al. 2002).

In the USA this disease is considered to be of secondary importance, but its generalized occurrence in the State of Minnesota was reported (Vargo et al. 1981).

In Brazil, it was reported for the first time in 1945 and later in 1961 (Gasperi 1961). The importance of the disease in the States of Paraná, São Paulo and Mato Grosso do Sul was recognized in the mid 1970s with the spread of wheat in those States. In July 1975, when these States lost almost all the wheat crop owing to the severe frost (−5 to −9 °C), large quantities of seed were imported from Mexico for seeding in 1976. The imported cultivars such as INIA 66, Jupateco 75 and Tanori 71 were highly susceptible to spot blotch. Besides, appropriate control measures were not then available and were not practiced until 1979. This resulted in severe epidemics of the disease, year after year and all the wheat cultivated soils became infested with the spot blotch pathogen (Mehta 1978; Mehta et al. 1992).

In Latin American countries, especially in Brazil, yield losses have been reported, most of which were attributed to a complex of diseases comprising mainly leaf rust and spot blotch. However, in some years yield losses caused by spot blotch alone could be quantified: for example, in 1982, losses in yield were determined using the cultivar Anahuac at Palotina, where spot blotch was the predominant disease and butyl triazole and ethirimol were used to eliminate the interference of other minor diseases. As mentioned elsewhere in this book, the yield losses were 31–37 %. In 1983, spot blotch alone induced a 79.0–86.5 % loss in yield of the highly susceptible cultivar Mitacoré. There was also a drastic reduction in grain quality.

The root rot phase of the disease, common root rot, is severe in drier areas of Africa, Australia, Brazil, Canada and the USA (Joshi et al. 1969; Mehta 1978; Mehta and Gaudêncio 1991; Mehta and Igarashi 1985a). Yield losses due to common root rot have been reported to be around 20 % in Brazil (Diehl et al. 1983). In Australia, common root rot causes moderate losses (Knight et al. 2010; Agarwal 2011) (see chapter on common root rot).

Symptoms

The disease affects all plant parts including the roots and the seed. The initial symptoms on the leaves are characterized by small, light-brown to black, oval spots (3–4 mm) (Fig. 6.28). At this stage no sporulation of the pathogen can be observed and the spots may be confused with Septoria diseases. Soon the spots enlarge and become typically elliptical with abundant sporulation. Because of the sporulation, the center of the lesion looks dark-brown to almost black with dark brown margin. In such cases the black spore mass can be easily removed with the fingers and can be seen with the naked eye and thus the symptoms can be distinguished from the Septoria diseases, in which the spores are formed in pycnidia and are not black. During the rainy season, the spores are washed and only conidiophores can be seen under a binocular. In severe cases of infection the lesions coalesce and the whole leaf becomes dry (Fig. 6.29). Besides the leaves, the

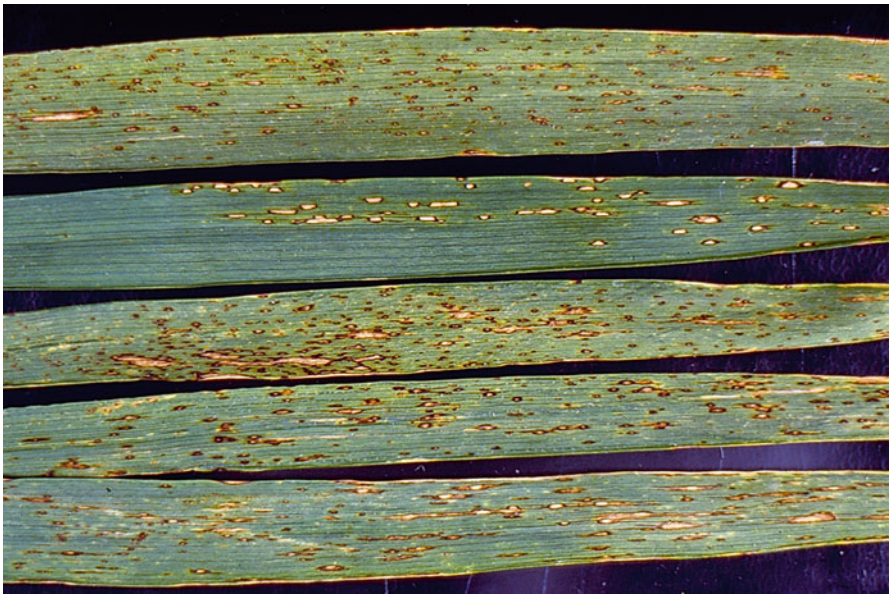

Fig. 6.28 Initial symptoms of spot blotch (*Bipolaris sorokiniana*) on wheat leaves

stems, glumes, rachis and awns can be infected showing dark brown-black spots. Infected glumes show a light center and a dark brown margin. Profuse sporulation on the glumes and on the nodes could be observed within a few days after rain followed by high temperatures (>25 °C). Stem and node infections result in lodging (Fig. 6.30).

Infected spikes give rise to infected seed and consequently produce infected seedlings (Fig. 6.31). The seeds can be infected or get contaminated during the harvest. Infected seeds are shriveled and have oblong lesions with light centers and dark margins. Infected seeds show the black point symptoms. Black point symptoms near the embryo are normally associated with *B. sorokiniana* and or *Alternaria alternata*.

The common root rot phase of the disease occurs on the crown, the sub-crown internodes and the roots, appearing as dark-brown to black discoloration and necrosis. The root system may be partially or completely damaged resulting in poor plant growth or death of the seedlings. Diseased plants are stunted and chlorotic (see chapter on common root rot).

Causal Organism and Epidemiology

Spot blotch is caused by *Bipolaris sorokiniana* (Sacc.) Shoem. (Syn. *Drechslera sorokiniana* (Sacc.) Subram. and Jain; *Helminthosporium sorokinianum* Sacc. Ex Sorok.; *H. sativum* Pamm., King and bake; *H. californicum* Macke and Paxton).

Fig. 6.29 (**a**) Typical sporulating elliptical lesions on leaves; (**b**, **c**) severely infected leaves by spot blotch (*B. sorokiniana*)

Fig. 6.30 (**a**, **b**) Spot blotch symptoms on wheat stems and nodes; (**c**, **d**) spikes showing sporulation of *B. sorokiniana*

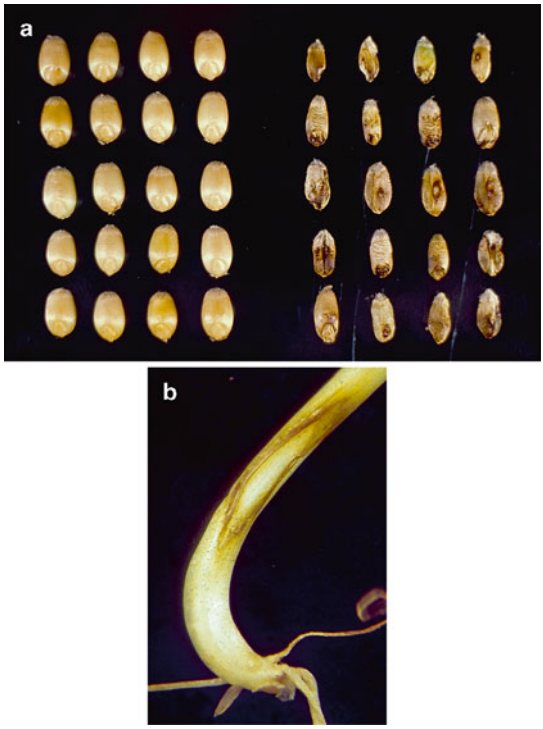

Fig. 6.31 (**a**) Healthy and infected kernels of wheat showing symptoms of spot blotch; (**b**) typical elliptical lesion on coleoptile caused by seed transmitted *B. sorokiniana*

(Telemorph *Cochliobolus sativus* (Ito et Kurib.) Drechs. Ex Dastur). The sexual phase *C. sativus* is not found in nature.

Conidia are born in groups of 2–3, on simple conidiophores. Conidia are 1–8 septate, germinate only by polar cells and measure 15–30×60–134 µm (Fig. 6.32). The perithecia of the perfect stage are bulbous with ostiolar beak and measure 340–470×370–530 µm. The asci are cylindrical or fusiform, rounded at the apex and contain 4–8 curved ascospores. The ascospores are filiform, hyaline and measure 160–360×6–9 µm (Fig. 6.33).

The fungus is a facultative parasite and grows well on common artificial media at 20–25 °C. The colony characters are variable. During excessive replications on culture media, the fungus shows a lot of variation due to mutation, which is a common phenomenon in *B. sorokiniana*. Normally, such mutations do not alter the pathogenicity of the isolates (Christensen 1925, 1929).

During 9 years of seed health testing in Brazil, up to 94 % of the samples tested were infected/contaminated by *B. sorokiniana* (Mehta and Igarashi 1985b). Severe infections in seed samples from some regions were correlated with severe spot blotch epidemics.

The pathogen is transmitted through contaminated or infected seed (Mehta and Igarashi 1985a, b; Reis 1991b). Mycelia in the germinating seed grows onto the coleoptile and seminal roots. Brown elliptical lesions develop on the coleoptile.

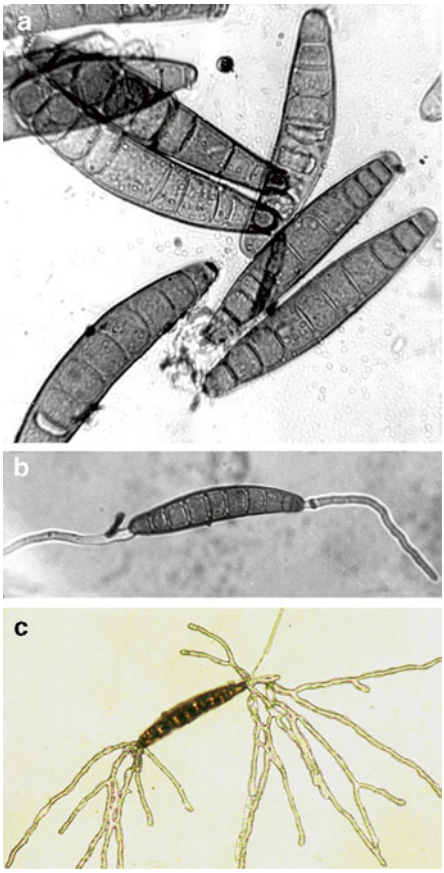

Fig. 6.32 *B. sorokiniana*. (**a**) conidia; (**b**, **c**) bipolar germination of conidia

During the harvest, under epidemic conditions, thick black clouds of conidial dust are seen. While harvesting severely infested fields, the combine harvester gets almost covered by black dust of *B. sorokiniana* spores within 5–6 h of working (Fig. 6.34). The black dust contains almost 80 % spores of *B. sorokiniana* and can be easily removed by hand. The spore dust can be dried and stored under laboratory conditions for several years and can be used for field inoculations for germplasm screening purposes (Fig. 6.35). During harvest, the soil also gets infested with the pathogen. The spores germinate on the soil and get transformed into mycelium and clamydospores which can survive and remain viable for several years (Reis and Abrãao 1983; Chinn and Ledingham 1958; Boosalis 1962). However, in recent years, such a situation does not exist due to the substitution of susceptible cultivars with locally developed resistant ones (Fig. 6.35).

The secondary dissemination of the disease is through wind-borne spores. The spores travel long distances. However, the soil inoculum is most important as a primary source of infection. The first symptoms of the disease are observed 45–55 days after sowing.

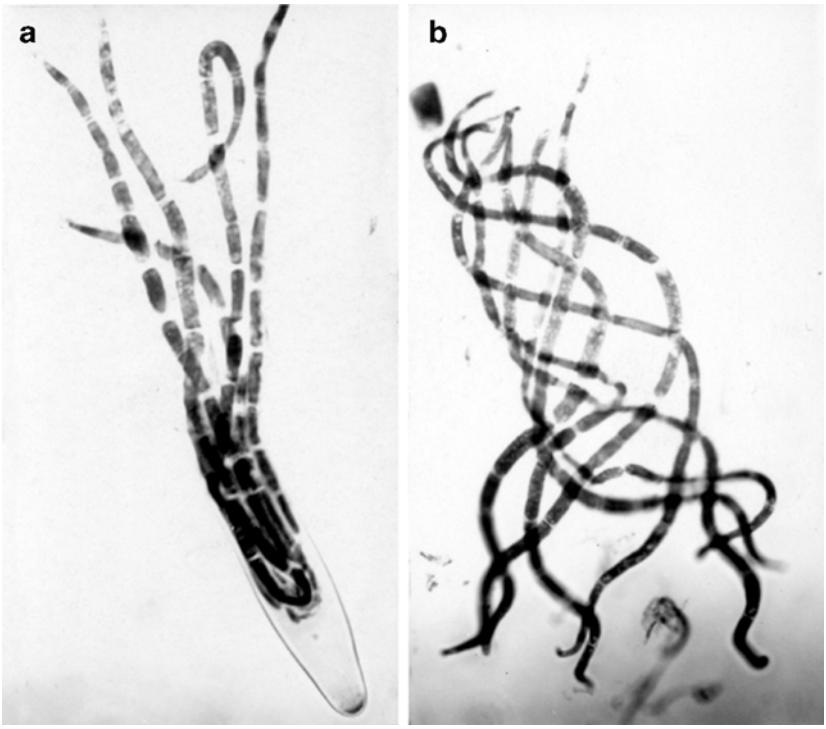

Fig. 6.33 *Cochliobolus sativus*. (**a**) ascus and ascospores; (**b**) ascospores (Courtesy R.M. Hosford)

B. sorokiniana infects several other Gramineae hosts like triticale, barley, rye and other species of *Agropyron*, *Bromus*, *Hordeum* and *Lolium*. The pathogen does not attack maize and oats. The life cycle of the pathogen is presented in (Fig. 6.36). Since the perfect stage does not occur in nature, it plays no role in the epidemiology of the disease.

Oliveira et al. (2002) and Gyawali et al. (2012), studied genetic variability within ten *B. sorokiniana* isolates using RAPD and reported high level of genetic variability among the isolates. Later, Baturo et al. (2004) studied genetic variability in 53 *B. sorokiniana* isolates collected across the Brazilian wheat growing areas during 15 years using RAPD analysis. Contrary to the findings of Oliveira et al. (2002), Baturo et al. (2004) reported a very low variability among the isolates and concluded that no genetic changes occurred in the pathogen population during the past 15 years (Fig. 6.37). These authors further concluded that the limited variability observed among the *B. sorokiniana* isolates, is probably due to the differences in aggressiveness and not due to the differences in virulence (Knight et al. 2010; Agarwal 2011).

Phenotypic experiments revealed that isolates of *B. sorokiniana* collected from barley spot blotch infections showed a high level of pathogenic variability across the differential set of cultivars. In contrast, isolates from common root rot infections produced significantly less spot blotch disease on inoculated barley leaves

Fig. 6.34 (**a**) Black clouds of conidial dust while harvesting heavily infested wheat field in early 1980s; (**b**) absence of clouds of conidial dust while harvesting healthy wheat in early 2005, Brazil, due to the use of resistant or less susceptible cultivars

(Knight et al. 2010). These authors suggested divergence within Australian populations of B. sorokiniana in relation to host tissue specificity. Several other studies on genetic structure of this pathogen have been reported (Knight et al. 2010; Agarwal 2011; Gyawali et al 2012).

Control

Breeding for spot blotch resistance started in the early 1980s and then intensified. Some progress can be seen. The earlier highly susceptible cultivars like INIA, Jupateco, Tanori 71, Mitacoré and Paraguay 214, were withdrawn from the list of

Fig. 6.35 (**a**, **b**) Combine machine covered with *B. sorokiniana* conidia a few hours after wheat harvest; (**c**) storage of conidial dust containing >80 % of spores collected from the combine in a glass vial under laboratory conditions for several years

recommended cultivars and new, less susceptible cultivars such as CEP 11, CEP 14, Tapejara, Igapó, Cacatu, Batuira, Piratan and BR 8 have been included. Besides, several other resistant cultivars have been developed in the recent years. Although accurate data about yield losses induced by spot blotch in the new cultivars are not available, field evaluations during the past 2 decades have demonstrated their reduced susceptibility.

Partial resistance is known to exist in wheat. Mehta (1981), studied the conidial production, sporulation period and rate of lesion extension by *B. sorokiniana*, in order to detect cultivars with higher levels of partial resistance. According to this

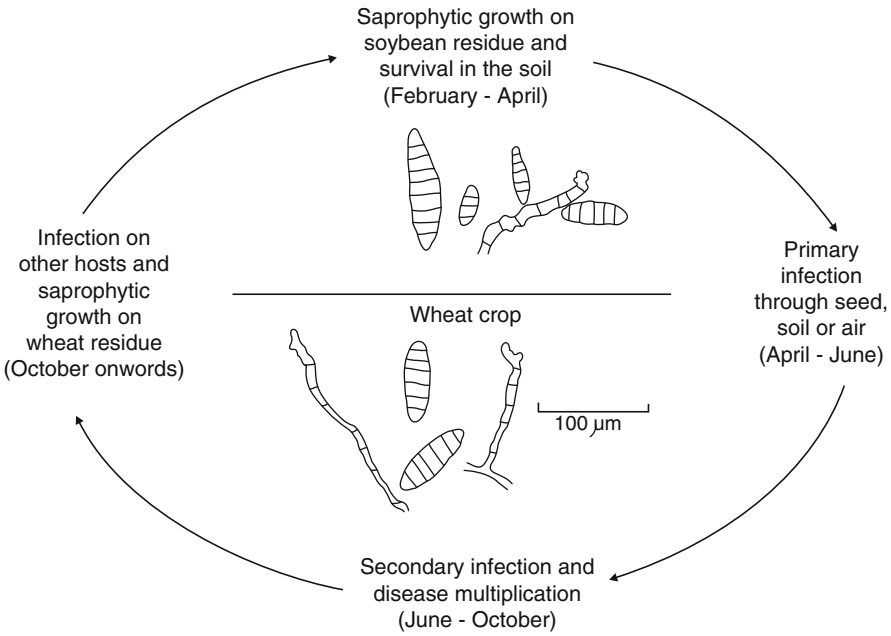

Fig. 6.36 Life cycle of *B. sorokiniana*

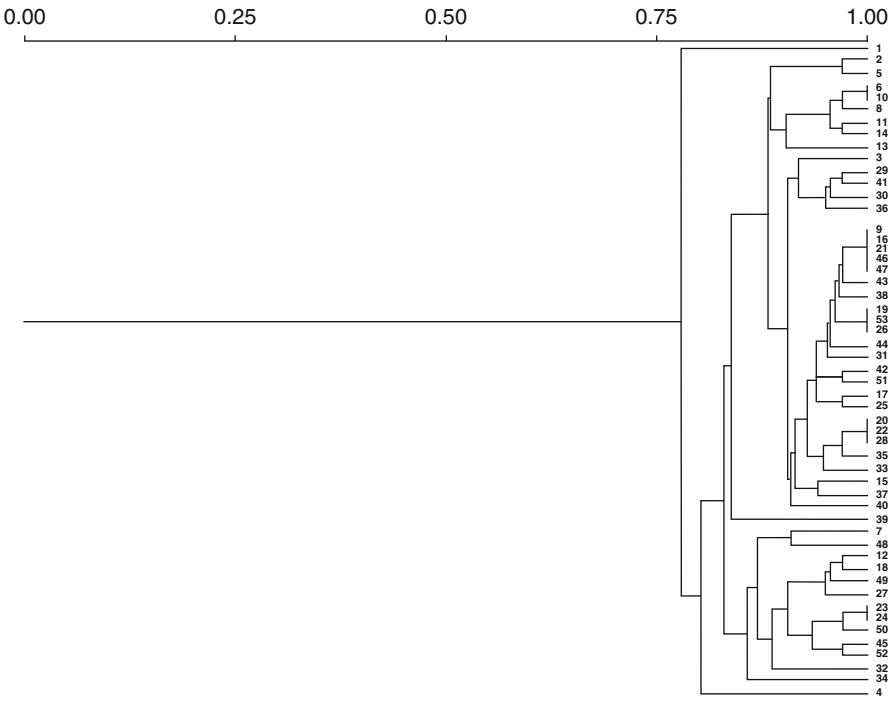

Fig. 6.37 Dendrogram produced by UPGMA cluster analysis based on RAPD markers from seven random primers for *B. sorokiniana. Source*: Baturo et al. (2004)

Fig. 6.38 Disease severity scale of *B. sorokiniana* for glasshouse studies

author, the parameters of partial resistance like conidial production, sporulation period and the rate of lesion extension acted independently and these parameters are considered essential to measure partial resistance.

The degree of resistance of different cultivars can be measured using these parameters as well as the disease severity scale (Fig. 6.38). The resistance parameters can be measured at the decimal growth stage 53, since older leaves become more susceptible for *B. sorokiniana* infection (Fig. 6.39). Mehta (1981) identified BH1146, LD 783 and PAT 7219, as the most resistant cultivars. Nonetheless, in recent years cultivars with a higher level of resistance have become available (Domiciano et al. 2009). Very little information is available about the mechanism of resistance.

Several effective seed treatment fungicides are now available. However, in Brazil, for example, some seed treatment criteria are followed: (1) No seed treatment should be undertaken if the infection level is <20 %, as determined by "blotter test" and the germination percentage is within the seed standards; (2) seed lots with <20 % infection can be treated only if the germination percentage is low and after seed treatment it reaches the seed standard limit; (3) seed lots with >20 % infection can be treated with fungicide only if the germination percentage is lower than the standard and there is a seed shortage problem; (4) seed lots may be treated with fungicides irrespective of the level of infection, especially

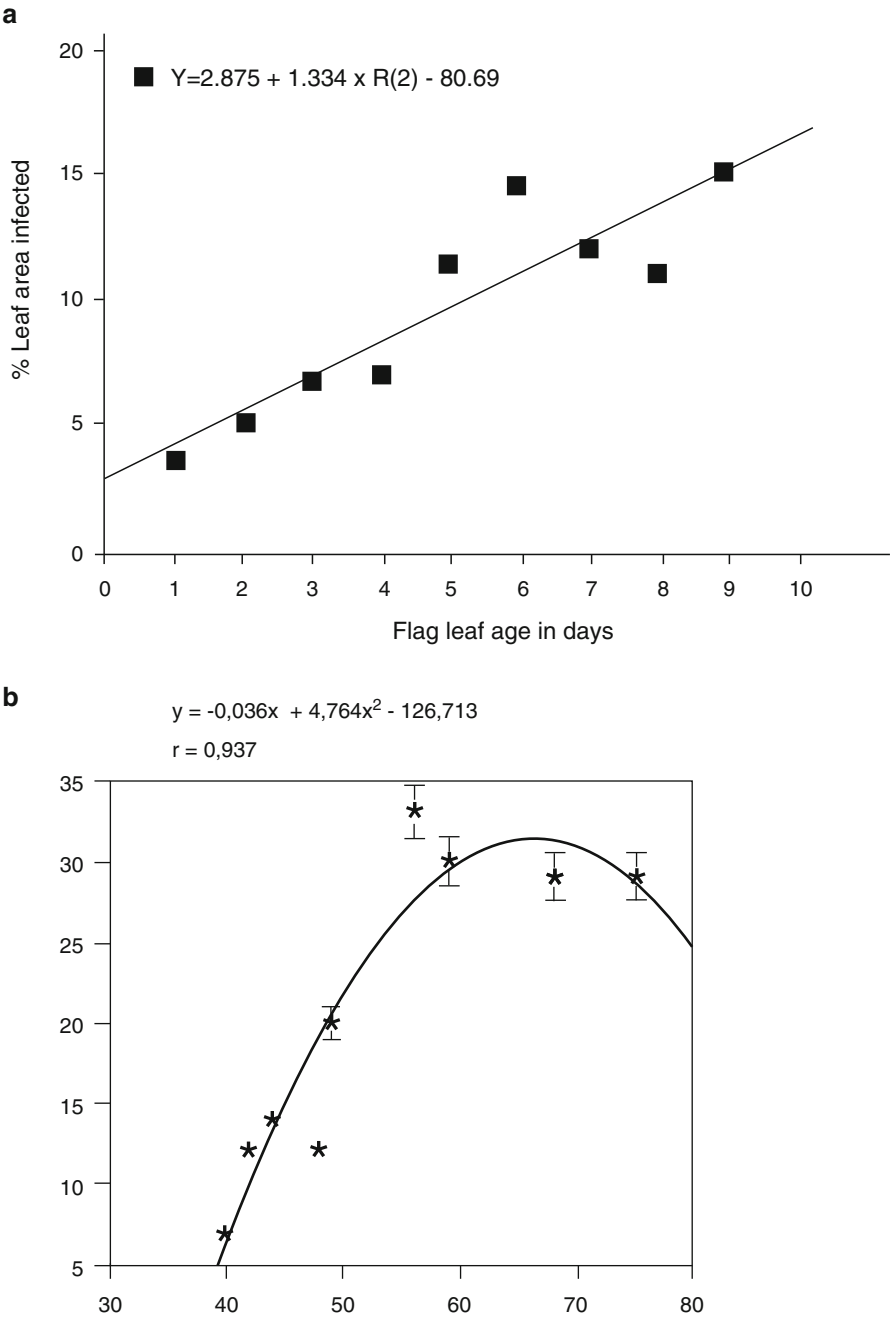

Fig. 6.39 (**a**) Relation between the flag leaf age and the percentage of flag leaf area infected by *B. sorokiniana* (*Source*: Mehta 1981); (**b**) relation between the flag leaf age of 16 wheat cultivars and the percentage of flag leaf area infected by *B. sorokiniana* (*Source*: Triller and Mehta 1997)

for seeding in new areas or areas where crop rotation is practiced. This criterion is based on the belief that the seed treatment would avoid the introduction of additional inoculum into the soil and consequently would minimize the primary source of inoculum.

One of the ways to avoid severe losses due to the foliar diseases is to plant more than one cultivar and on different dates but within the recommended sowing period. Late sowing, especially in tropical and sub-tropical areas should be avoided in order to avoid coincidence between the flowering-milk stage and the hot humid periods. Although the pathogen survives on crop residues, there is nither any indication nor report in the literature about the influence of tillage practices and/or crop rotation, on the severity of foliar phase of this disease (Mehta and Gaudêncio 1991). This is probably attributable to the fact that the disease is also air-borne.

6.1.10 Stem Rust

Stem rust, also known as black rust, is one of the most studied rusts of wheat in the world and occurs wherever the crop is grown. In this rust, stems are more severely attacked than the other parts of the plant like leaves, sheaths and awns and hence the name "stem rust".

Among the three rusts which occur on wheat, the stem rust caused by *Puccinia graminis* f. sp. *tritici* (Pgt) is considered the most notorious and devastating one. As stated elsewhere, a survey conducted by the journal Molecular Plant Pathology, to find out the opinion of the international scientific community as to which ten fungal plant pathogens the community would consider for scientific/economic importance in the world. *M. oryzae* appeared the outstanding pathogen occupying first place in the ranking of ten pathogens. *Puccinia* spp. especially the three wheat rusts appeared in the third place with an imminent threat to wheat cultivation due to the emergence of race Ug99 (Ralph et al. 2012).

Significant and repeated crop failures caused by rusts occurred in North America between 1904 and 1962 (Hodson 2011; Roelfs 1985). Severe epidemics also occurred in Europe and China (Leonard and Szabo 2005).

Yield losses caused by stem rust in the world are very variable in different parts of the world. In Manitoba and Saskatchewan from 1925 to 1935, for example, the losses amounting to approximately 31 million dollars were attributed to yield reduction (Butler and Johns 1955). During the 1960s and 1970s, annual yield losses in the USA were estimated to be over 1 million metric tons (Bockus et al. 2010). In Latin American conditions, this rust appears late in the season when the crop reaches near maturity and hence causes little or no damage in yield. There has been no record of any severe epidemic of this rust since 1982, in the Latin American region.

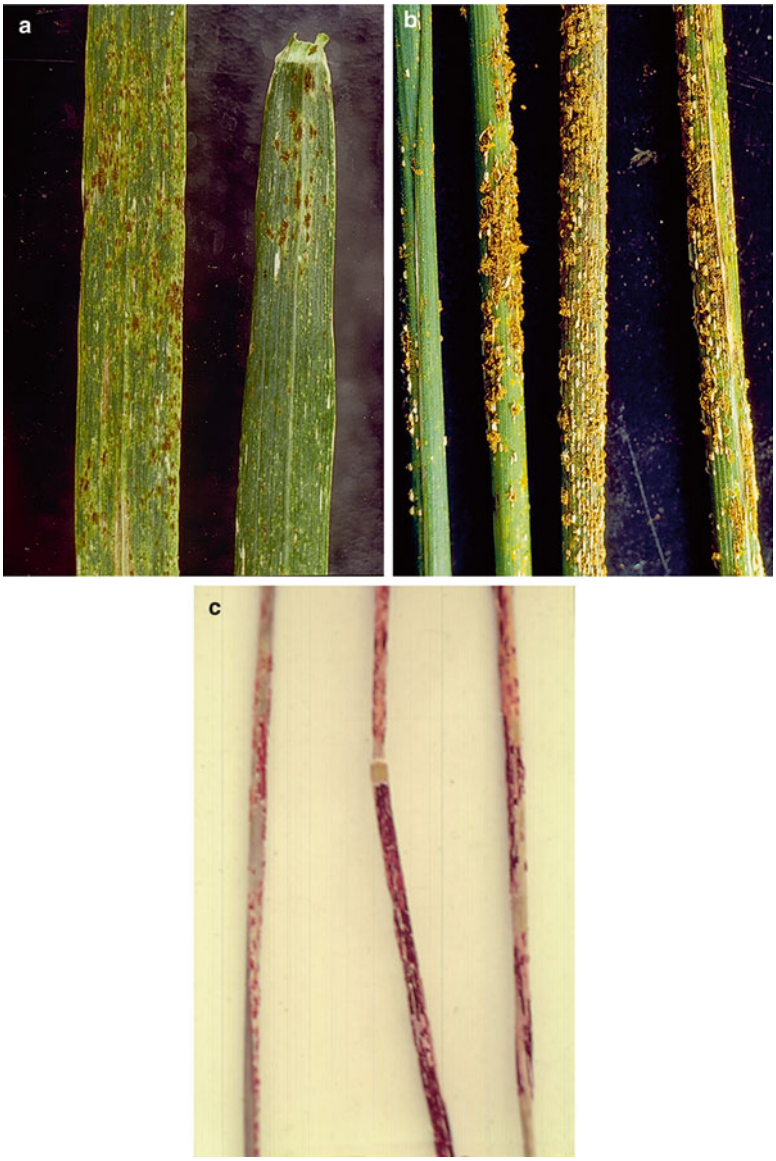

Fig. 6.40 Stem rust. (**a**) symptoms on leaves; (**b**, **c**) symptoms on stems

Symptoms

The rust pustules (uredosori) appear basically on stems and leaves in the form of long stripes. Pustules are reddish to dark brown in color and contain unicellular uredinospores. Later, teliutospores develop in the teliosori within the same uredinial sori or separate telial sori. In this case, the pustules become almost black in color and contain the bicellular teliutospores (Fig. 6.40). When the pustules are fully matured they

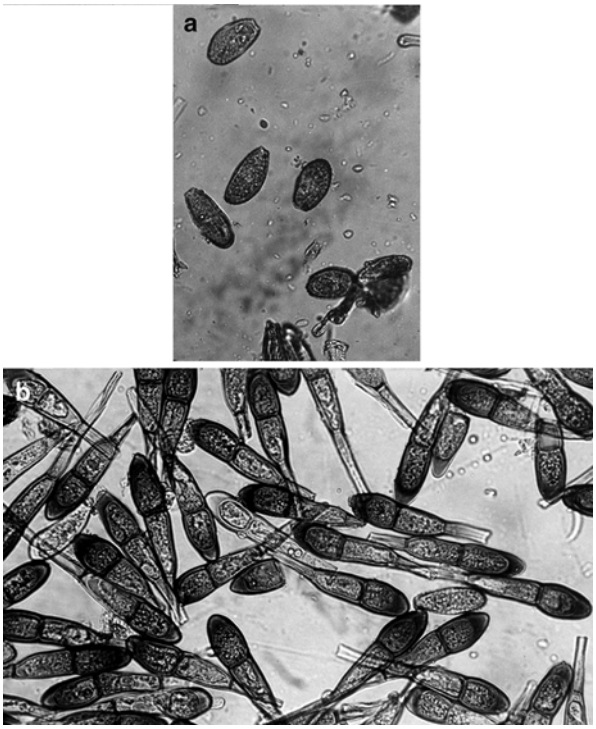

Fig. 6.41 (**a**) Uredinospores; (**b**) teliutospores of *Puccinia graminis tritici*

break the epidermis and release the uredinospores and the teliutospores and show a white collar of epidermis around the pustules.

Causal Organism and Epidemiology

The uredinospores attack, common and durum wheat, barley and triticale and the sexual stage of the fungus basidiospores (produced by the germination of teliutospores) attack a completely different host barberry. The uredinospores are binucleate cells and measure 17–20×25–30 μm (Fig. 6.41). These are the first spores produced in uredinial sori. The uredinospores germinate when there is free water on the leaves and cause infection through stomata. The stem rust fungus is heterothallic and is caused by *Puccinia graminis* f. sp. *tritici* Ericks and Henn. *Puccinia graminis* species is sub-divided in different *forma speciales*, which in turn are sub-divided into races and biotypes. This rust produces five types of spores out of which uredinospores and teliutospores are produced on wheat and the pycnospores and aeciospores are produced on barberry (*Barberis vulgaris*), completing the life cycle of the pathogen (Fig. 6.42).

The teliutospores fallow the production of uredinospores and are produced in teliutosori which are normally produced on the same uredinial sori. The teliutosporos

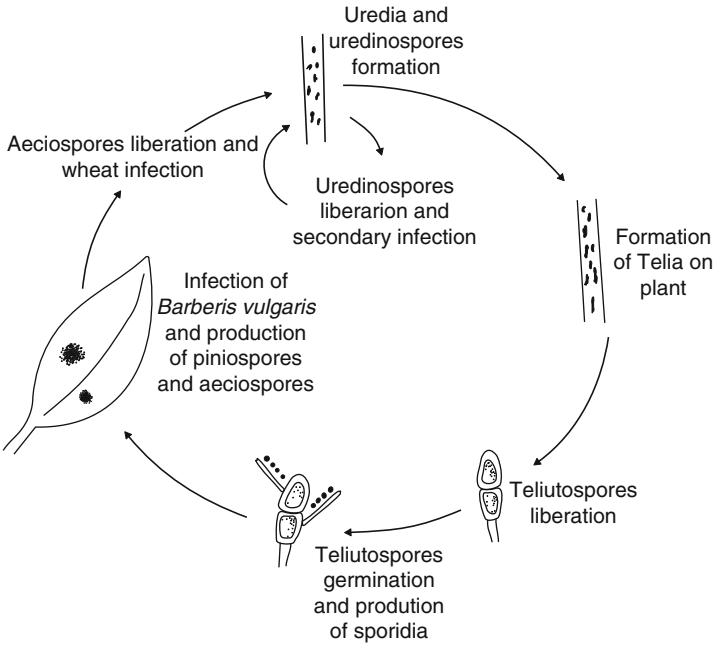

Fig. 6.42 Life cycle of *P. graminis tritici*

are bicellular and measure 35–64 × 11–22 µm. The epical cell is rounded and darker at the epics. Like uredinospores they germinate when there is free water on the leaves and the stems. Both cells of the teliutospores germinate and produce promycelium on which four sporidia are produced. The sporidia are small, hyaline, unicellular and constitute the third type of spores of this rust and are the only spores which infect barberry. On barberry pycnia and picnospores are produced. Picnospores of the opposite sex unite and produce aecia and aeciospores on the lower side of the leaf. The aeciospores are slightly yellowish, global, are produced in chains and measure 14–26 µm. The aeciospores infect wheat.

Being heterothalic (heteroic) rust, the stem rust fungus is comprised of several physiological forms or races distinguished by the virulence patterns on an international set of differential host series. They are further distinguished by "form species" or "forma specialis" depending on their host specificity. New races are created basically through the sexual recombinations in the alternate host (barberry). New races may also develop through mutation and parasexual mechanisms in the uredinial stage.

Being heterothalic (heterocious) rust, half of its life cycle (uredinospores and teliutospores) is completed on wheat and the other half (picnospores and aeciospores) on barberry which serves as an alternate host (Fig. 6.42). The alternate hosts belonging to barberry are: *Barberis vulgaris* L., *B. canadensis* Mill., *B. fendleri* Gray (Wiese 1987). The uredinospores are repetitive spores since they infect the same host in which they are formed. Since barberry is not present in South-America, the only source of primary infection is the uredinospores brought by wind from

other States or countries where wheat is sown early. High humidity and high temperatures favor the disease. Prolonged periods of high humidity and high temperatures (20–25 °C) are considered optimum for the development of an epidemic.

The incubation and latent period of stem rust is 10 days, which means that it takes 10 days between the infection and the first appearance of the visible symptoms (production of new colony of uredinospores). It is for this reason that the uredinospores are the only spores responsible for the development of an epidemic of this rust. The teliutospores do not infect wheat. They remain viable on the wheat residue and after germination produce basidiospores capable of infecting barberry.

There exist over 300 physiological races of the pathogen in the world. New races have emerged as a result of hybridization within the pathogen or sometimes through mutation (Roelfs et al. 1992; Mehta 1993). In recent years, a new race designated as Ug99 appeared in East Africa with virulence to *Sr31*—a commonly used resistance gene all over the world (Pretorius et al. 2000; Singh et al. 2011b). Race Ug99 has virulence for several genes commonly present in wheat germplasm including gene *Sr31* located in the 1B.1R wheat-rye translocation known to be present in several wheat cultivars worldwide (Singh et al. 2007). So far, seven variants within the Ug99 lineage have been reported varying in virulence to *Sr21*, *Sr24*, *Sr31* and *Sr36* (Singh et al. 2011b). Considering the threat to world wheat cultivation posed by Ug99 lineage, releasing new and widely adopted wheat cultivars with resistance to lineage Ug99 has received utmost importance (Lowe et al. 2011; Macintosh and Pretorius 2011). According to Singh et al. (2007), the long-term strategy should focus on rebuilding the combination of slow rusting gene *Sr2* with other unknown additive genes of similar nature to achieve long-term durability.

The Global Rust Initiative (GRI) was launched in 2005 in Nairobi, Kenya, to raise awareness about the risk posed by race Ug99, along with recommendations from the expert panel report of CIMMYT of 2005, to implement a global strategy to overcome the threat of Ug99. According to Singh et al. (2007), the success of GRI would depend on early replacement of susceptible cultivars by resistant ones, especially in the danger zone and consequently, check the migration of Ug99 to other areas and with further evolution. In Latin American region the presence of Ug99 has not so far been reported.

Control

As stated earlier, destruction of the alternate host barberry helped in breaking the life cycle of the pathogen and drastically reduced the chances of creating new virulent races. According to Ralph et al. (2012), the barberry eradication program started in 1918 in the USA and has continued since then in the UK, must be viewed as one of the major achievements in plant pathology in both scope and disease management.

Stem rust is normally controlled through the use of genetic resistance. The adult plant resistance from Hope is a classical example (Roelfs et al. 1992). In Brazil, stem rust has been effectively controlled through the use of genetic resistance, application of fungicides and change in sowing dates (see the chapter on Cultural

practices). Diversity of sowing dates and the use of resistant cultivars is important in reducing the severity of the stem rust. A wheat free period would be an important practice to break the disease cycle. Zadoks and Bouwman (1985) have stressed the importance of the wheat free period (green bridge). This can be achieved through the eradication of volunteer wheat plants through herbicides.

According to Singh et al. (2007), spring wheat cultivars derived from CIMMYT germplasm (having resistance gene *Sr31* located on rye translocation 1B.1R), have been offering resistance to stem rust for several decades. However, given the emergence of stem rust complex race Ug99, this situation may alter completely. Singh et al. (2011b) have given up-to-date information on the threat posed by Ug99 to wheat production around the world.

6.1.11 Tan Spot

Tan spot of wheat caused by *Pyrenophora tritici-repentis* (Ptr), is one of the most important diseases of wheat in various countries and has been reported in more than 21 countries. It was first identified on some grasses in Germany in 1902 and later in Japan in 1928. However, severe epidemics have only occurred in the USA (North Dakota, South Dakota, Kansas, Oklahoma and Nebraska), Canada, Australia, Argentina, Bolivia, Paraguay and Brazil. According to Lamey (1981), the disease was very severe in areas without crop rotation and where there was lot of crop residue from the previous crop in the field.

Severe epidemics of tan spot were also noticed together with infections of *Leptosphaeria avenaria* during 1971–1976 in North Dakota (Hosford 1971a, b, c, 1972). According to Gough and Johnston (1981), the disease was very severe in Oklahoma mainly because of the change in cultural practices and especially the use of no-tillage cultivation. A somewhat similar situation occurred in Australia where *P. tritici-repentis* was identified as a wheat pathogen. During the years 1949–1970, straw burning was a common practice in Australia. Having understood the importance of maintaining the crop residue on the soil surface to retain soil humidity and to check soil erosion, the no-tillage cultivation became a common practice in Australia. While this practice brought several advantages, it provoked severe epidemics of tan spot disease (Klein and Ellison 1981; Rees 1987; Rees and Platz 1980, 1983, 1990).

The disease was reported for the first time in the State of Paraná, Brazil, in 1973 (Mehta 1975c) and later it spread to other Brazilian States and to the neighboring countries (Mehta 1978, 1993). Yield losses in Brazil were estimated to be around 30 % (Mehta 1993).

Symptoms

Under natural field conditions initially the typical disease symptoms are characterized by oval to diamond-shaped lesions which later elongate and develop a tan color with a chlorotic halo and a small brown infection site. The initial lesions are small and measure 0.7–4.0 × 0.5–1.0 mm with a whitish center surrounded by a yellow halo (Fig. 6.43).

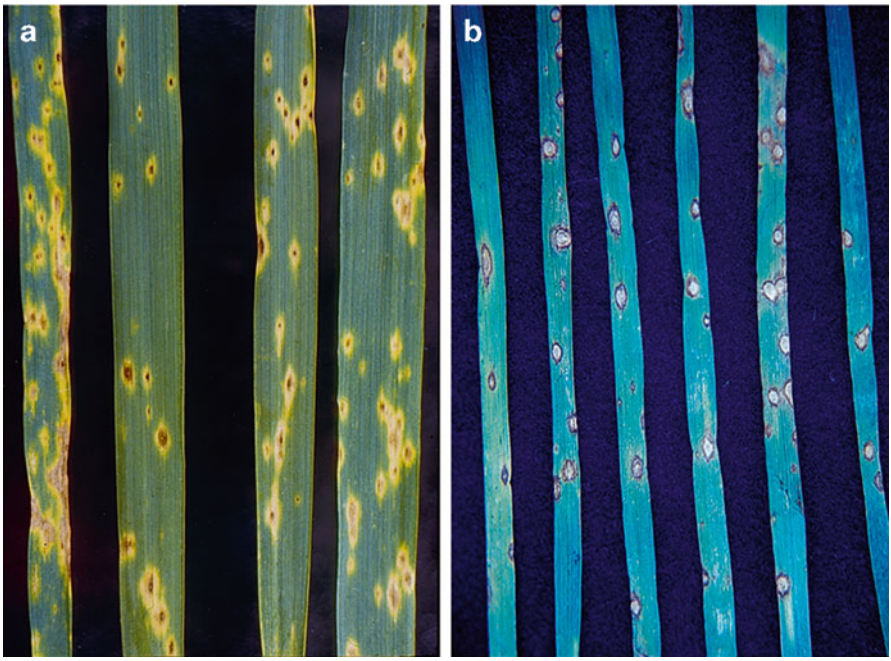

Fig. 6.43 (**a**, **b**) Initial diamond-shaped lesions with chlorotic halo of tan spot (*Drechllera tritici-repentis*) on wheat leaves

Later the lesions become large and tan with yellowish margin (15×2 mm), where the fructification of the fungus can be observed through a hand lens. Heavily infected leaves turn necrotic and dry prematurely (Fig. 6.44). At harvest time pseudothecia (ascostroma) the sexual stage of the pathogen can be observed especially on the stems and sometimes on the infected seed (Fig. 6.45). Depending upon the susceptible genotype tan necrosis and/or extensive chlorosis can be observed. In 1997, severe incidence of tan spot along with typical diamond-shaped lesions, circular to irregular dark-brown lesions without tan necrosis and chlorosis or with very little chlorosis, were also observed. Occasionally, sporulation of Ptr in the form of conidia and conidiophores has been observed on these lesions. According to De Wolf et al. (1998), on resistant and partially resistant wheats lesion size is reduced and chlorosis and necrosis may be absent.

Causal Organism and Epidemiology

Tan spot is caused by *Pyrenophora tritici-repentis* (Died) Drechs. (anamorph *Drechslera tritici-repentis* (Died) Shoemaker) (Syn. *Helminthosporium tritici-repentis*; *Pyrenophora trichostoma* (Fr.) Fckl. *Pyrenophora* Fr. is a genus of Loculoascomycetes with multi-locular and binucleate ascostroma.

Fig. 6.44 (**a**, **b**) Severely infected wheat leaves showing tan spot symptoms (*D. tritici-repentis*)

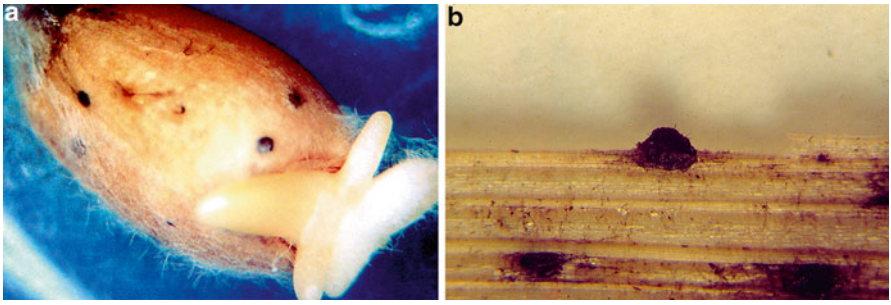

Fig. 6.45 (**a**) Infected wheat seeds showing presence of pycnidia and perithecia; (**b**) formation of ascostroma of *Pyrenophora tritici-repentis* (sexual stage of *D. tritici-repentis*) on wheat stems after harvest

Several species of *Pyrenophora* are the perfect stages (sexual stages) of *Drechslera* spp. (*Helminthosporium* spp.). *Pyrenophora tritici-repentis* is the perfect stage of *Drechslera tritici-repentis* (Died) Shoemaker (Syn. *D. tritici- vulgaris* (Nishikado) Ito. *H. tritici vulgaris* Nishikado and *H. tritici-repentis*).

The nomenclature of this fungus was discussed in the past by several mycologists (Hosford 1971a, b; Wehmeyer 1949, 1954) and *P. tritici-repentis*, *H. tritici-repentis* and *H. tritici vulgaris* are considered the same fungus.

P. tritici-repentis is a homothallic fungus and can be easily cultivated in common artificial culture media. When cultured on autoclaved wheat or maize leaves the fungus produces conidiophores and conidia within 4–6 days. Later, the conidia

germinate and transform into mycelia and finally produce ascostroma (Fig. 6.46). The ascostroma on such leaves mature within 3–4 weeks. When completely matured they are black, spherical, flattened at the base, with prominent ostiole and measure 445–756 × 445–676 µm. The ascostroma are multi-locular with polyascal locules with bitunicate and clavate asci measuring 178–267 × 36–53 µm (Fig. 6.47a). The asci contain 8 three septate ascospores, lined in four, with one or two longitudinal septa, constricted at the septum, slightly yellowish-brown and measure 36–53 × 13–12 µm. The conidia are erect, long, 3–11 septate without constriction at the septa, light brown and measure 53–143 × 13–18 µm (Fig. 6.47b). The conidia germinate through the end cells and through one or two middle cells.

A similar fungus *Platyospora pentamera* (Karst) Wehm, occurs as a secondary pathogen along with *P. tritici-repentis* (Hosford 1975b).

Tan spot causes severe yield losses in wheat. The pathogen has two reproductive phases - the asexual phase [*Drechslera tritici-repentis*] and the sexual phase [*Pyrenophora tritici-repentis*—(Ptr)]. In the absence of a wheat crop the pathogen survives on the left-over wheat stubble from one season to another. When the wheat is sown again the pathogen releases ascospores which attack the seedlings and completes the cycle (Fig. 6.48).

Five races of the pathogen are reported to occur based on virulence patterns on a differential set of wheat cultivars in the United States and Canada (Luz and Hosford 1980; Lamari and Bernier 1989; Lamari et al. 1995; Singh et al. 2010; Gurung et al. 2011). Under glasshouse conditions, Lamari and Bernier (1989) and Lamari et al. (1991), grouped the Ptr isolates into four pathotypes based on production of different symptoms on differential lines. Pathotypes (races) one through four were respectively designated as Nec+ Chl+, Nec+ Chl-, Nec- Chl+ and Nec- Chl-(Table 6.1). Later, a different form of pathotype 3 has been designated as race 5 (Lamari and Bernier 1989; Lamari et al. 1995). Differences in aggressiveness among 84 Ptr isolates collected from three different States were also reported from the USA (Krupinsky 1992).

According to the recent reports eight races and three toxins have been characterized base on three differential host genotypes (Ciuffetti et al. 1998; Bockus et al. 2010). Knowledge regarding the diversity of the pathogen population is a prerequisite for breeding aimed at development of resistant cultivars. Among other methods, assessment of genetic diversity of pathogen populations based on genomic DNA fingerprinting has been investigated (Mehta et al. 2004). Information based on pathogenicity and DNA fingerprinting can be used to test the germplasm of wider adaptability for resistance against all the different genotypes of the pathogen occurring in a particular country.

Mehta et al. (2004) reported genetic diversity of 40 Ptr isolates collected from seven wheat cultivars grown in 12 different regions of the State of Paraná, Brazil, during 1996–1998, using pathological and molecular analyses (PCR-RFLP, ERIC and REP-PCR banding patterns). According to these authors, out of 40 isolates, 18 were in sporulating form and hence only these were used for pathological studies whereas all the 40 isolates were used for molecular analysis and toxin sensitivity test. Out of the 18 isolates one isolate was non-pathogenic to wheat. Virulence test

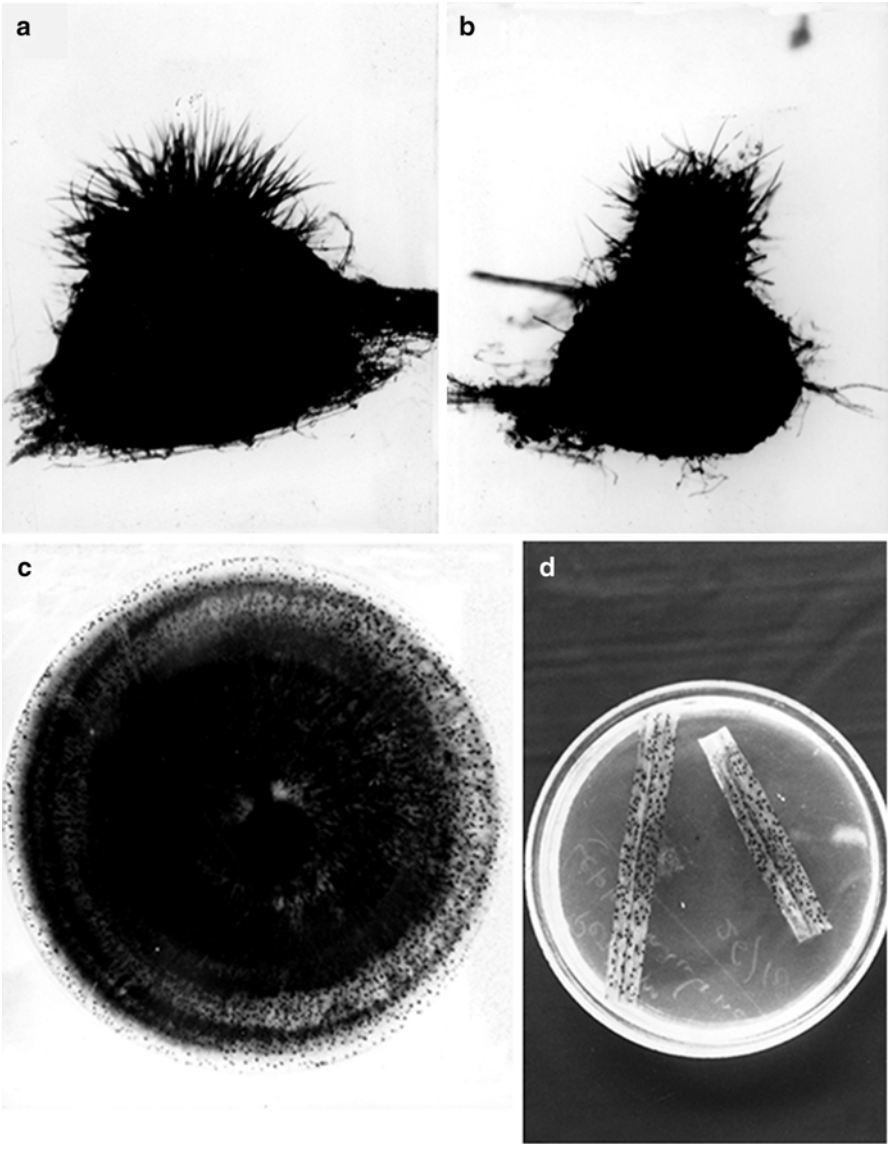

Fig. 6.46 (**a**–**c**) Formation of ascostroma of *P. tritici-repentis* on artificial culture media; (**d**) on autoclaved corn leaves

conducted on six wheat cultivars against 18 Ptr isolates, revealed a very low cultivar × isolate interaction indicating that the isolates differed in aggressiveness but not in virulence. The varietal screening test on 38 wheat cultivars using a mixture of seven aggressive isolates showed compatible reaction to all wheat cultivars. Cultivars BRS 23, BRS 177 and CEP 17 were resistant, whereas the rest of the cultivars were susceptible to highly susceptible. Out of the 40 Ptr isolates only one

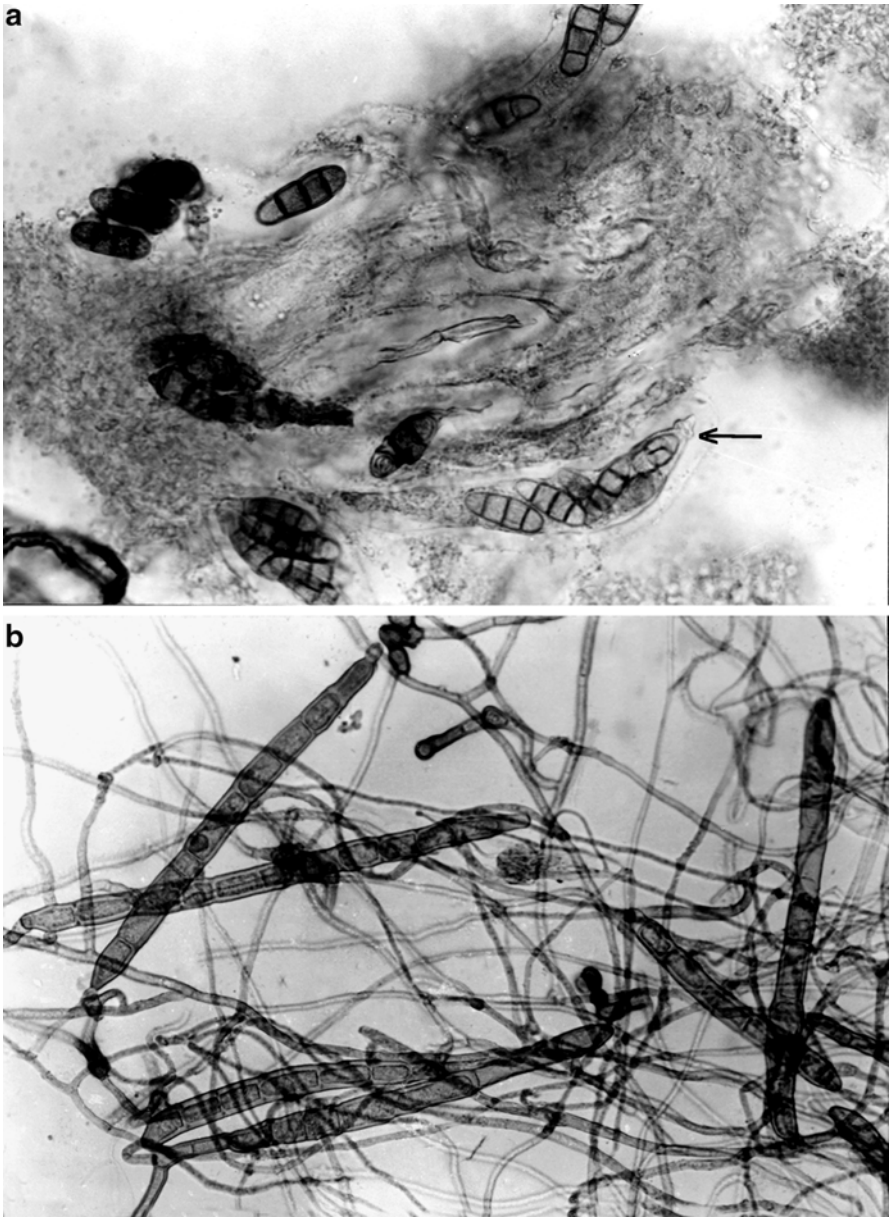

Fig. 6.47 (**a**) Asci and ascospores of *P. tritici-repentis*; (**b**) conidia of *D. tritici-repentis*

avirulent isolate was identified as pathotype 4 (race 4). The rest of the isolates belonged to pathotype 1 (race1).

Regarding the molecular analysis, Mehta et al. (2004) reported that Ptr on wheat in the State of Paraná, Brazil, is genetically uniform (Fig. 6.49). According to these authors, small molecular variabilities observed between the isolates are due to the

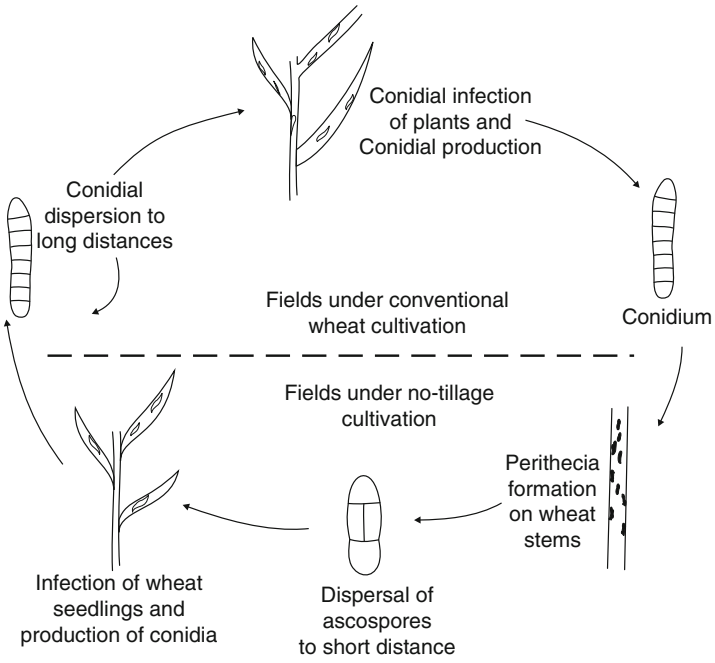

Fig. 6.48 Life cycle of *P. tritici-repentis*

Table 6.1 Pathotypes of *Pyrenophora tritici repentis*

Pathotype	Necrosis (nec)	Chlorosis (cl)	Designation[a]
1	+	-	nec+ cl+
2	+	+	nec+ cl−
3	-	+	nec− cl+
4	-	-	nec− cl−

[a]+ = Positive; − = Negative

intra-specific variants of Ptr. This implies that screening wheat germplasm for resistance to this disease need not involve a wide range of isolates of this pathogen. Such results may be useful for the breeding programs seeking wheat cultivars resistant to tan spot fungus *P. tritici-repentis*.

Pathogenicity and activity of phytotoxin produced by Ptr, to six wheat cultivars is reported by Mehta et al. 2004; Bockus and Claassen 1992; Ciuffetti et al. 1998; Bach and Kimati (2012), studied purification and characterization of toxins from wheat isolates of *Drechslera tritici-repentis*, *Bipolaris bicolor* and *B. sorokiniana* and reported that the low molecular weight metabolites produced by *B. bicolor*, *B. sorokiniana* and *D. tritici-repentis* were considered to be toxins that facilitate disease in wheat cultivars.

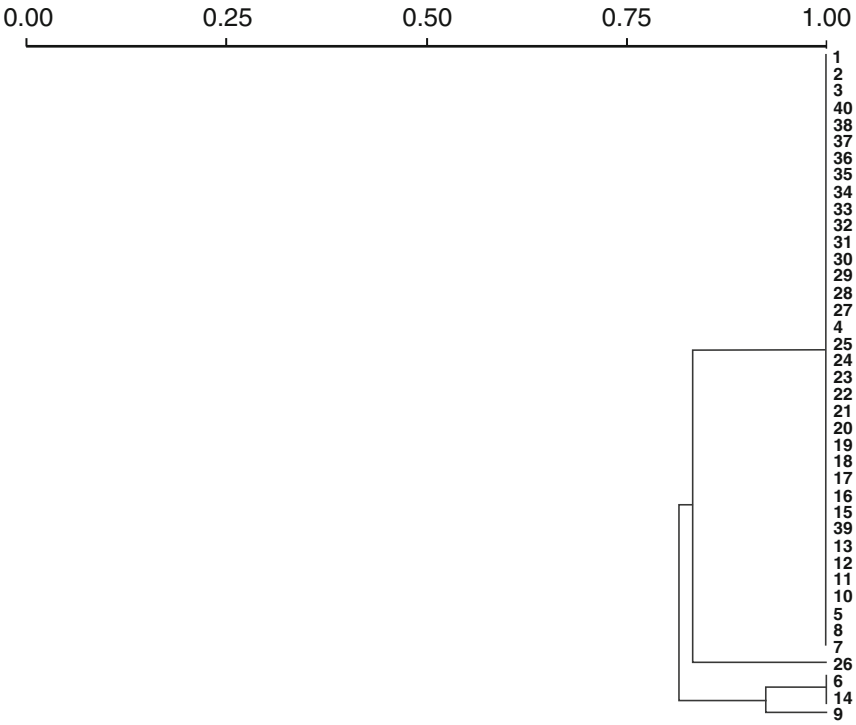

Fig. 6.49 Dendrogram produced by UPGMA cluster analysis based on primer pairs ERIC1/ERIC2 and REP1/REP2, for *P. tritici-repentis. Source*: Mehta et al (2004)

Lack of a significant cultivar×isolate interaction was interpreted by Van der Plank (1963) as difference in aggressiveness and that the possibility of physiologic specialization is low (Krupinsky 1992; Onfroy et al. 1996). Differences in aggressiveness of Ptr isolates were also reported by Krupinsky (1992) and Abrinbana et al. (2012). Cultivars Ibiara and OR 1 were resistant to moderately resistant, BH 1146 and Mirim were moderately susceptible whereas, cvs. IPR 84 and IPR 85 were susceptible to most of the Ptr isolates.

Thus differences observed between the isolates are due to the intra-specific variants of Ptr. The perfect state Ptr is very commonly observed in Brazil (Mehta 1993) and so these variants could be due to the sexual hybridization occurring within the pathogen populations (Mehta et al. 1993).

In Kenya infected seed is considered to be the primary source of infection (Hosford 1981). Seed infection was also observed in Australia and Brazil (Fig. 6.45a). Shilder and Bergstrom (1991), conclusively demonstrated the seed infection and seed transmission of *P. tritici-repentis.* However, inoculum present on the crop resi-

Table 6.2 Presence of pseudothecia of *P. tritici-repentis* on crop residue as the source of primary infection, in Brazil

Sampling date	Conventional system[a]	No-tillage cultivation[a]
October	+	+++
Dezember	+	+++
January	+	++
March	0	+
June	0	+
September	0	+
October	+	++
January	0	+++

[a]0 = Pseudothecia abscent, + = pseudothecia present, ++ = few pseudothecia, +++ = pseudothecia abundant
Source: Mehta and Gaudêncio (1991), Mehta (1993)

due is still the most important element in the epidemiology of the disease all over the world. Hosford (1981) emphasized the importance of crop residue and suggested that the inoculum in the wheat crop residue can continue producing conidia and ascospores for a period of 3 years. Higher severity of tan spot in no-tillage cultivation was reported by several other workers (Mehta 1975c, 1993; Odvody and Boosalis 1978; Lamey 1981; Rees 1987; Mehta and Gaudêncio 1991).

Complete resistance to tan spot in wheat is not available. The relationship of wheat seedling and adult plant resistance to *P. tritici-repentis* was studied by Tadesse et al. (2011). These authors reported significant positive correlation between seedling resistance evaluated in greenhouse and adult plant resistance estimated in field conditions. They further reported that tan spot resistance is controlled by a single recessive gene.

While crop residue is the most important source of primary infection, a part of primary infection may also come from the secondary hosts as well (Table 6.2). In Canada, Krupinsky (1982) reported the following secondary hosts: *Agropyron cristatum*, *A. desertorum*, *A. intermedium*, *A. smithii*, *Elymus angustus*, *E. cinereus*, *E. triticoides*, *E. giganteus*, *E. junceus*,*Andropogon gerardii*, *Bouteloua gracilis*, *Alopecurus arundinaceus*, *Stipa viridula*, *Sorgastrum nutans*, *Dactylis glomerata*, *Calamivilfa longifolia*, *Phalaris arundinacea*, *Bromus inermis*, *Panicum virgatum*, *Hordeum vulgare*, *Avena sativa* and *Secale cereale*.

The survival mechanism of Ptr was studied by Mehta 1993. The pathogen is capable of surviving under extreme climatic conditions of −0 °C in North Dakota (USA) and +40 °C in Australia (Hosford 1971a, b; Rees and Platz 1980; Mehta 1993). The initial infection basically comes from the inoculum present on the left over wheat stubble of the previous wheat crop (Fig. 6.45b). To a lesser extent the initial inoculum may also come from the infected seed and from other secondary grass hosts, but epidemiologically speaking this source of infection is not considered important (Fig. 6.45a).

Control

In the varietal screening test Mehta et al. (2004), showed a compatible reaction for all the 38 wheat cultivars. Cultivars BRS 23, BRS 177 and CEP 17 were resistant whereas cvs. IPR 84 and IPR 85 were highly susceptible. Further work to

quantitatively measure the degree of resistance through the rate of lesion extension, may identify some new cultivars with an adequate level of resistance to be used in the breeding programs.

Resistance in wheat was reported in the USA and Australia (Hosford 1981; Rees and Platz 1990). Some of the Bobwhite and Veery lines and cultivars derived from CIMMYT germplasm having substitution 1B.1R, have been offering some degree of resistance to tan spot.

Some modification in the conservation tillage system may be necessary. Soil in no-tillage can be plowed (perhaps deep plowed) once in 2–3 years would reduce the tan spot severity. As mentioned elsewhere, in Argentina, in some disease prone area the soil is ploughed once a year after soybean harvest and before the wheat sowing to incorporate the crop residue and minimize the early infections of tan spot (Fernando et al. 1987).

According to Bockus (1998), Chisel plowing is intermediate between moldboard plowing and no-till in controlling tan spot and that currently, continuous no-till wheat production is not popular in Kansas.

Besides, fungicidal seed treatment also eliminates the seed- transmitted inoculum and would help eliminate the initial inoculum especially where crop rotation is practiced (see chapter on crop rotation). Tan spot of wheat can be controlled through fungicidal sprays with officially recommended fungicides (Embrapa 2011).

6.1.12 Yellow Rust

Yellow rust is also known as stripe rust and is one of the most important rusts of wheat in Argentina, Chile, China, India, England, Kenya, Northeastern of Europe, Mexico and Uruguay. Losses in yield caused by this rust could be between 5 and 50 % depending on the year and the country. Stripe rust is the most destructive disease in autumn-sown wheat in northwest and southwest China. Severe epidemics occurred in China in 1950, 1964, 1990 and 2002, which caused yield losses between 1.3 and 6.0 million tons (Xia et al. 2007).

Morgounov et al. (2012) studied disease incidence and severity for winter wheat variety Bezostaya 1 considering the data from international nurseries from 1969 to 2010 and from 51 countries across major wheat production regions totaling 1,047 reports. According to their studies, while the frequency of leaf rust and stripe rust occurrence was stable over time with some exceptions, substantial global reductions in stem rust were recorded in spite of the recent emergence of race Ug99. In Brazil, the disease is not economically important.

Symptoms

Yellow rust can infect all above ground parts of the plant, but infections are more common on the leaves and the glumes. The symptoms of this rust are quite different than the other two wheat rusts. On the leaves the formation of uredia in a linear

Fig. 6.50 (**a**, **b**) Symptoms of yellow rust (*P. striiformis*) on wheat leaves; (**c**) cross section of wheat leaf showing uredinial sori (Courtesy BASF, Brazil)

fashion can be easily observed (Fig. 6.50). The uredinospores are also produced on glumes and the mass of spores represent yellow color.

The telia and the teliutospores are observed when the wheat plant reaches maturity. The telia are shiny, black in color and like uredia are also arranged in a linear fashion across the veins. Under severe infections the root system is affected and the size of the grain is reduced.

Causal Organism and Epidemiology

The stripe rust is caused by *Puccinia striiformis* Westend. (Syn. *Puccinia striiformis* f. sp. *tritici* West (Syn. *P. glumarum* Ericks and Henn). The uredinospores and teliutospores are formed in sori. The teliutospores are dark brown flattened at the

extremities, two celled and measure 35–65 × 12–20 μm. The teliutospores remain covered in the sori for a long time. The teliutospores germinate but do not infect wheat and have no role in the progress of an epidemic.

Epidemiological aspects of stripe rust have been well documented by several workers (Zadoks 1961; Stubbs 1977; Rapilly 1979; Zadoks and Bouwman 1985; Wellings 2011). Stripe rust develops well in countries where cooler weather predominates during most of the year. In Europe *P. striiformis* over-summers on wheat. Although *P. striiformis* is cold loving rust, recently severe widespread epidemics have been attributed to new and aggressive races from warmer environments (Hovmeller et al. 2011).

Other than wheat, *P. striiformis* attacks barley, triticale, rye and some grasses. However, the *Triticum* spp. are the main hosts of this rust (Roelfs et al. 1992). According to Zadoks and Bouwman (1985), a single pustule (uredinium) per hectare can cause an epidemic in the Netherlands. So far, no alternate hosts are known for this rust in the Latin-American region. The aecial and pycnial stages are known to exist on *Berberis* spp. (Roelfs et al. 1992; Jin et al. 2010). Uredinospores alone are responsible for the epidemiology of the disease. Several races of this rust are known to exist even in the absence of an alternate host.

Control

Stripe rust is controlled by a combination of genetic resistance and fungicide applications. Resistance in wheat is of the adult plant type incurred by minor and major genes. As mentioned earlier, the slow rusting cultivars developed over the past 50 years which include *Lr34/Yr18* and *Lr46/Yr29* and Pavon 76 with leaf rust and yellow rust resistance genes *Lr46/Yr29* have been effective since their release in 1976 (Van Ginkel and Rajaram 1993; Navabi et al. 2003; William et al. 2007). The CIMMYT bread wheat line Saar has good levels of resistance to leaf rust and yellow rust (*Puccinia striiformis* f. sp. *tritici*) based on *Lr34/Yr18* in combination with other minor genes.

Chemical control has been used in Europe with success where the yield potential is around 6–7 tons to 1 ha (Buchenauer 1982). In other parts of the world like India, China and Latin-America, the use of fungicides to control stripe rust is limited to one or two applications only in epidemic years.

6.1.13 Zonate Eyespot

Zonate eyespot occurs in the USA, Mexico and Costa Rica and is important in subtropical humid areas. In South America, the disease was observed for the first time in 1992, in Samaipata, in Santa Cruz Department, Bolivia. In the following year, it

Fig. 6.51 (**a–d**) Symptoms of zonate leaf spot (*Drechslera gigantea*) on wheat leaves

was observed again in the same area but with higher severity of 20 % leaf area infected in susceptible cultivars. According to Zillinsky (1983), it can be a limiting factor for wheat production in some countries.

Symptoms

Initial symptoms on the leaf are circular spots with whitish center and well defined brownish border. Later the lesions develop and give an appearance of zonate spots (Fig. 6.51), which are the distinguishing characteristics of this disease from other

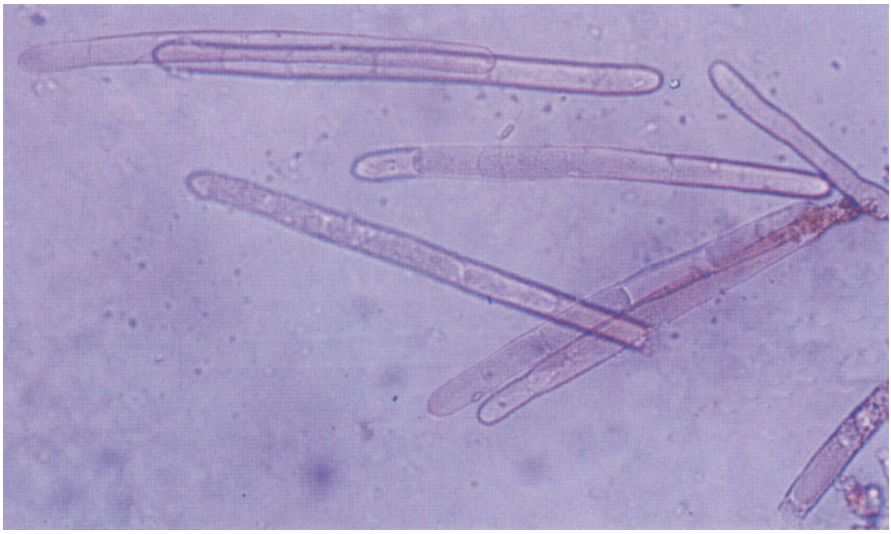

Fig. 6.52 Conidia of *D. gigantea*

leaf spots. Severely infected leaves become yellow or straw colored and die prematurely. With the aid of a hand lens presence of conidiophores can be observed on the older lesions. So far, symptoms on other parts of the plant are not observed (Sivanesan 1992).

Causal Organism and Epidemiology

Zonate eyespot is caused by *Drechslera gigantea* (Heald and F. A. Wolf) S. Ito. (*Helminthosporium giganteum* Heal and F. A. Wolf.). The conidiophores are long, dark brown with 5–6 septa (normally five) and measure 255–408 × 10 µm (average 335 × 10 µm (Fig. 6.52). Conidia are hyaline, cylindrical, erect, and measure 160–469 × 18.4–30.6 µm (average 256 × 22 µm) and rarely 81.6 × 15.3 µm. The conidia are very sensitive to dehydration and easily lose viability. So far, the perfect stage of this fungus is not known, but it is believed that it may belong to the genus *Pyrenophora*. When incubated in a humid chamber infected leaf portions produce abundant conidia within 72–86 h. The fungus growth is slow on common culture media like PDA. Well-developed colonies on PDA are olive green and show concentric rings.

According to Zillinsky (1983), the pathogen attacks hosts of Gramineae which adds to the source of primary inoculum. Ellis (1971) reported that the disease is common on *Cynedon dactylon*. In the USA the pathogen also attacks species of *Agropyron*, *Agrostis*, *Bromus*, *Caulophyllum*, *Echinochloa*, *Eleusine*, *Elymus*, *Eragrostis*, *Lersia*, *Muhlenbergia*, *Muas*, *Panicum*, *Pennisetum*, *Phalaris*, *Phleum*,

Poa, *Sporobolus* and *Zizannia*. In Bolivia, the disease was observed on *Cynedon dactylon* and *Eragrostis* sp. No information is available about the seed transmission of this pathogen.

Control

In Bolivia, cultivars Opata and K5 were more attacked than other cultivars like Genaro and Kea. Considering the presence of different hosts of the pathogen, crop rotation may not show encouraging results. As a precaution seeds coming from infested fields should be treated with appropriate fungicides. No other specific control measures are recommended.

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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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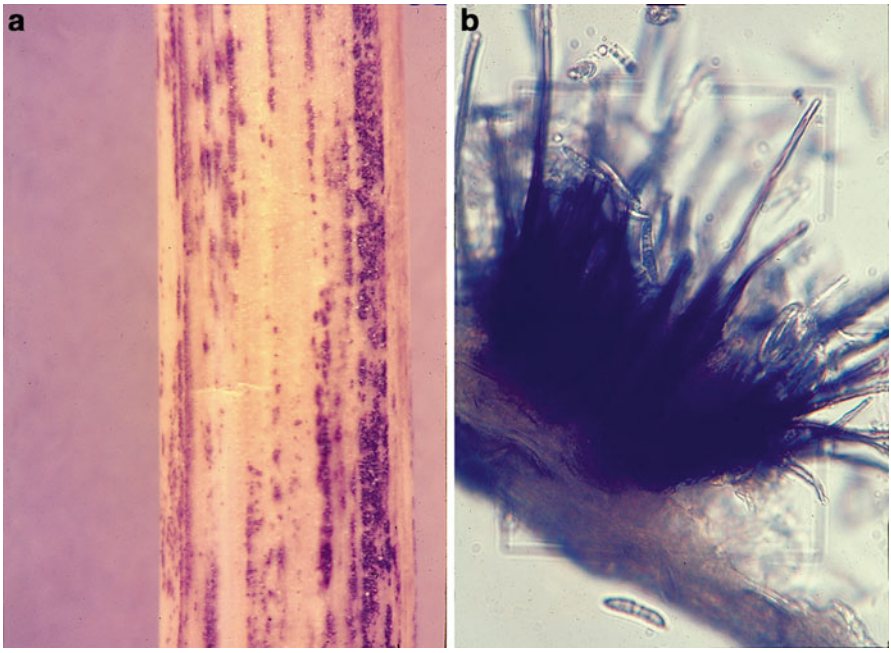

Fig. 7.1 Anthacnose (*Colletotrichum graminicola*). (**a**) symptoms on wheat stem; (**b**) acervulus and conidia

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.

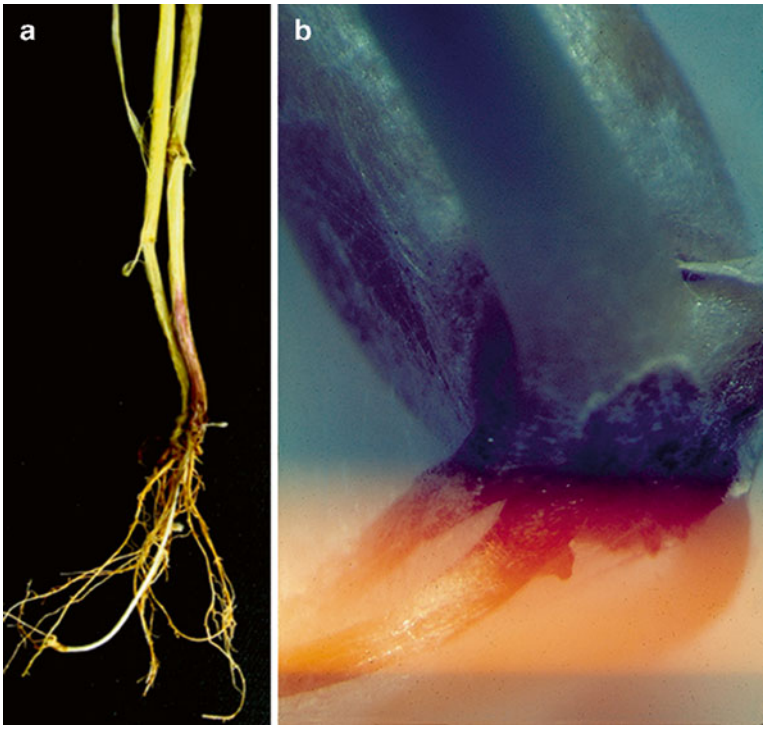

Fig. 7.3 (**a**) Symptoms of *Fusarium graminearum* root rot of wheat showing pinkish discoloration of the infected portion; (**b**) wheat crown infected with *F. culmorum*

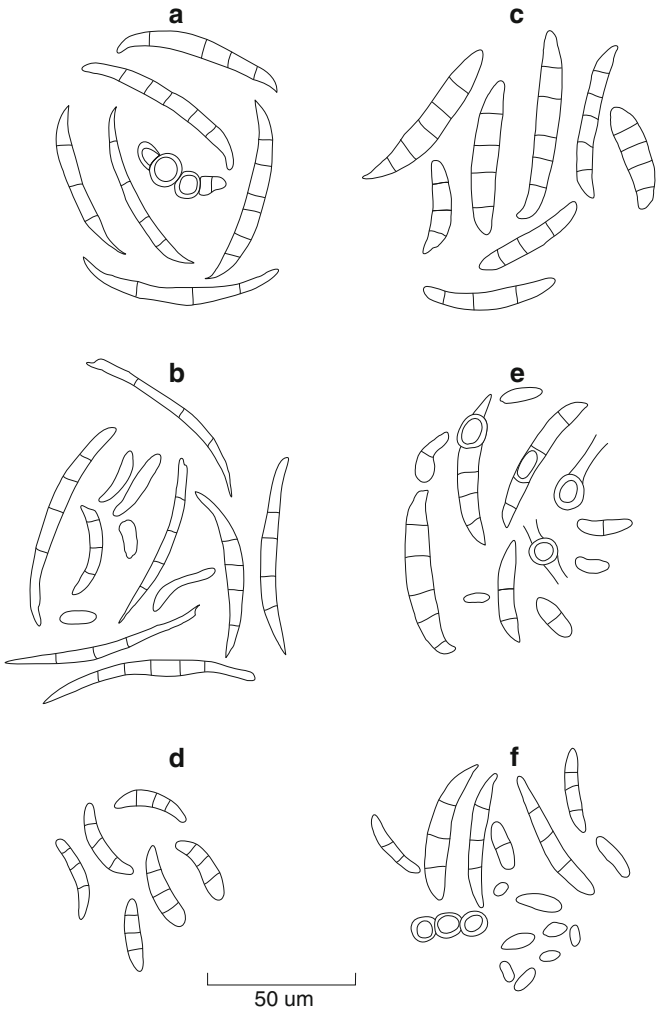

Fig. 7.4 *Fusarium* spp. causing Fusarium root rot. (**a**) macroconidia and clamydospore of *F. graminearum*; (**b**) macroconidia of *F. avenaceum*; (**c**) macroconidia of *F. culmorum*; (**d**) macroconidia of *F. nivale*; (**e**) macroconidia of *F. solani*; (**f**) macro and microconidia of *F.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

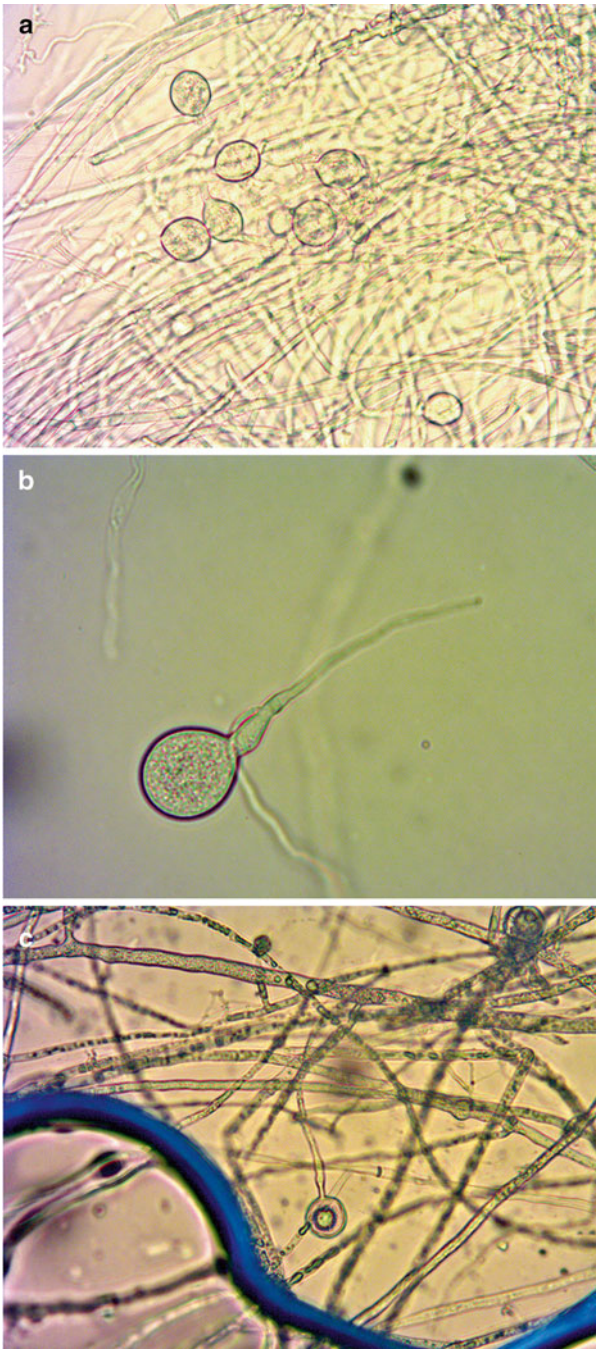

Fig. 7.5 Pythium root rot. (**a**) sporangia; (**b**) sporangium germination; (**c**) oospore

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.

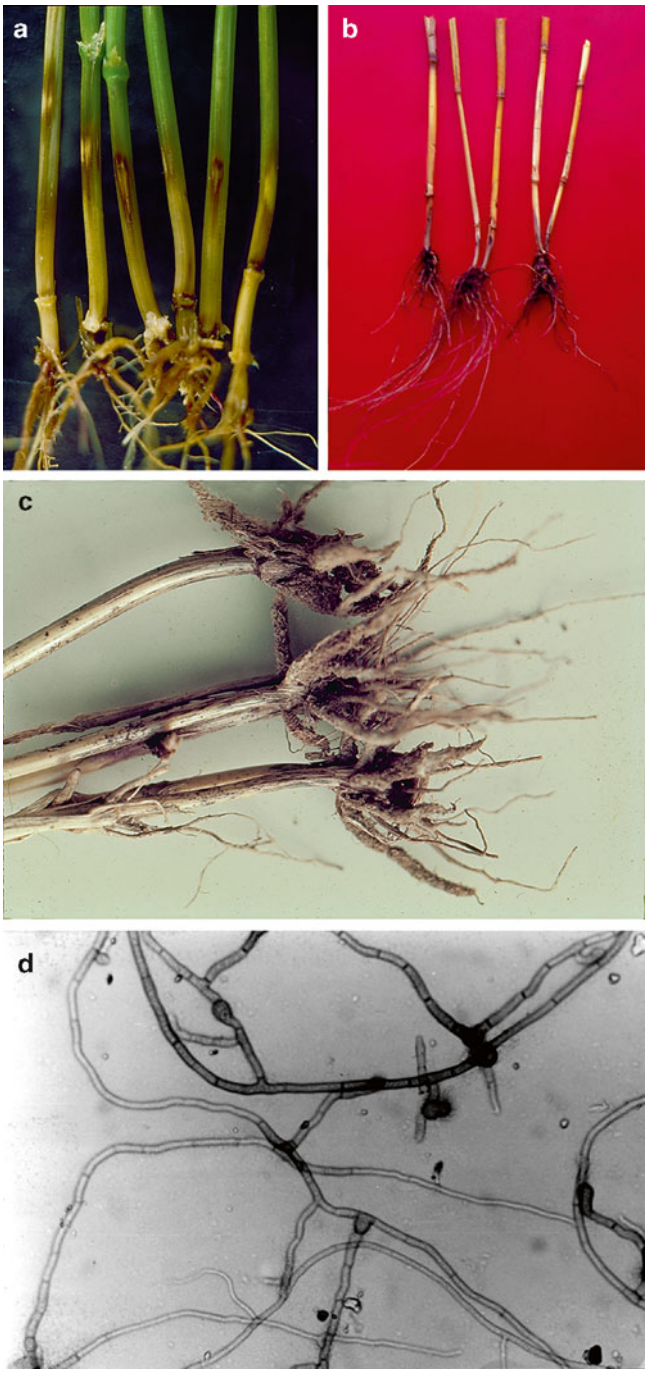

Fig. 7.6 Symptoms of Rhizoctonia root rot. (**a**) infection on the lower part of wheat stem showing elliptical lesions; (**b**, **c**) severe infection on roots and on the lower part of stems; (**d**) mycelium of *Rhizoctonia solani*. Note the branching of mycelium at right angles and constriction at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

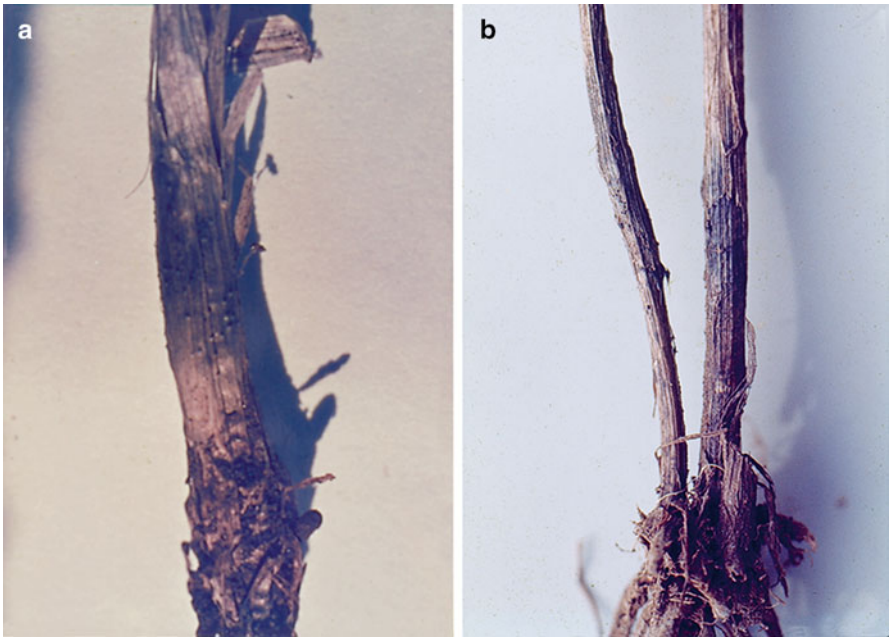

Fig. 7.9 (**a**, **b**) Wheat plants infected with *G. graminis* showing presence of black, hook-like perithecia

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.

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Chapter 8
Physiological Diseases

8.1 Physiological Diseases

Physiological disorders can be caused by several abiotic factors such as frost (Fig. 8.1), high temperatures, low soil pH, phytotoxicity caused by agricultural chemicals (Fig. 8.2), "melanosis" (Fig. 8.3), as well as nutritional imbalance. The gene *Sr2* conferring resistance (slow rusting) to stem rust, is known to be linked with pseudoblack chaff expression in glumes and near nodes of the stems, also called "melanosis". Soil acidity and low pH caused stunting of the plants which remain sterile and the phenomenon is referred to as "crestamento" in Latin-America. For details, the reader may refer to some specific publications (Snoball and Robson 1991; Mcfadden 1939; Sanderwirth and Roelfs 1980).

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Fig. 8.1 Physiological disorders caused by frost. (**a**) Seedling death due to severe frost, (**b**) leaf bleaching, (**c**) bleaching and premature death of spikes, (**d–f**) spike emergence affected by frost

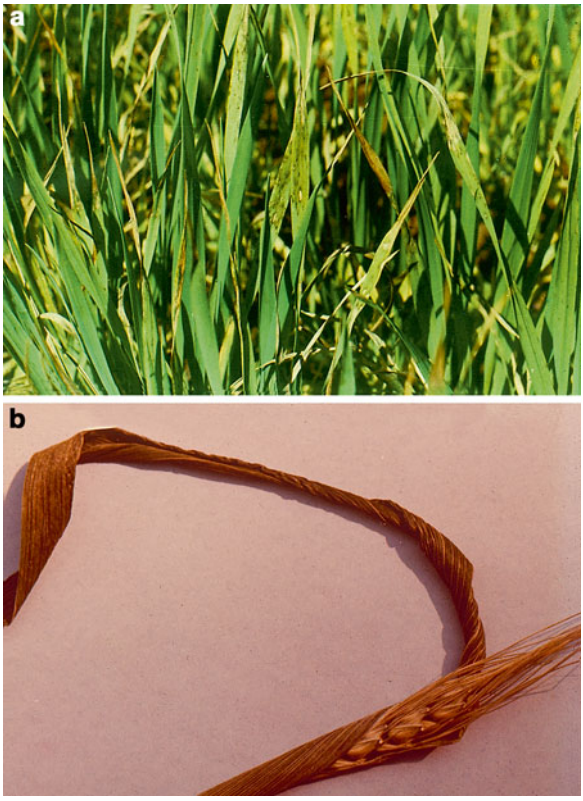

Fig. 8.2 (**a**–**b**) Phytotoxic effect caused by herbicide 2,4-D

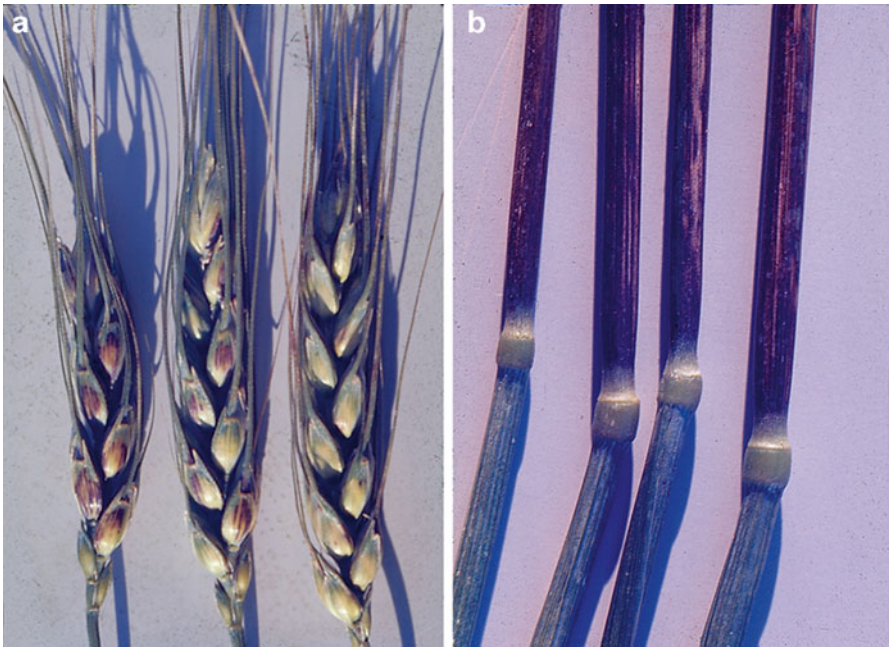

Fig. 8.3 (**a**, **b**) Symptoms of melanosis on spikes and stems

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Chapter 9
Disease Appraisal Scales

9.1 Disease Appraisal Scales

The evaluation of disease severity is necessary, especially for epidemiological experiments as well as for measuring the level of resistance. Several scales are proposed for different diseases but none of them is suitable for all the diseases. Use of specific scales are necessary for leaf blight fungi, powdery mildew (Fig. 9.1), rusts (Fig. 9.2), for some spike diseases (Fig. 9.3) and for hemilthosporium (Fig. 9.4).

Rogenski et al. (2012) suggested application of artificial neural networks to estimate infection by leaf spot in wheat crop. These authors proposed a system based in artificial neural networks in order to estimate the percentage of infections of diseases, assisting in decision making and facilitating the monitoring of potentially infected areas. However, the system needs to be validated under different wheat growing conditions.

In the following pages, some diagrammatic scales are presented to aid the reader in disease appraisal and its interpretation. Normally, disease ratings should be made 3–4 times during the crop cycle to plot the progress of the disease epidemic. For further details the reader may refer to specific publications in this respect (Chester 1950; Browder 1971; James 1971a, b; Roelfs 1992).

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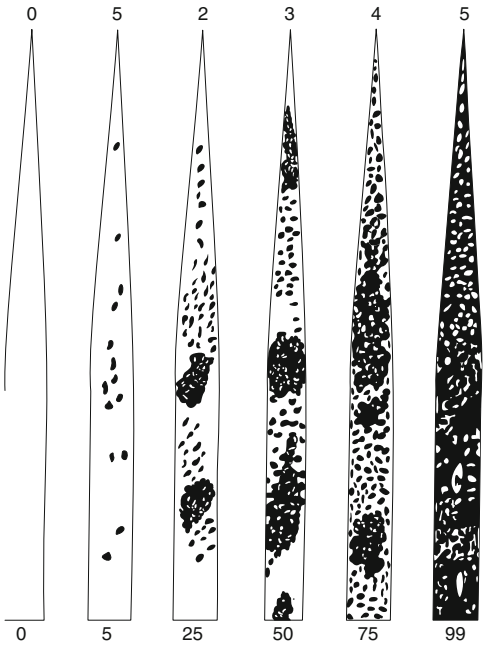

Fig. 9.1 Disease appraisal scale for powdery mildew. *Source*: Mehta (1993)

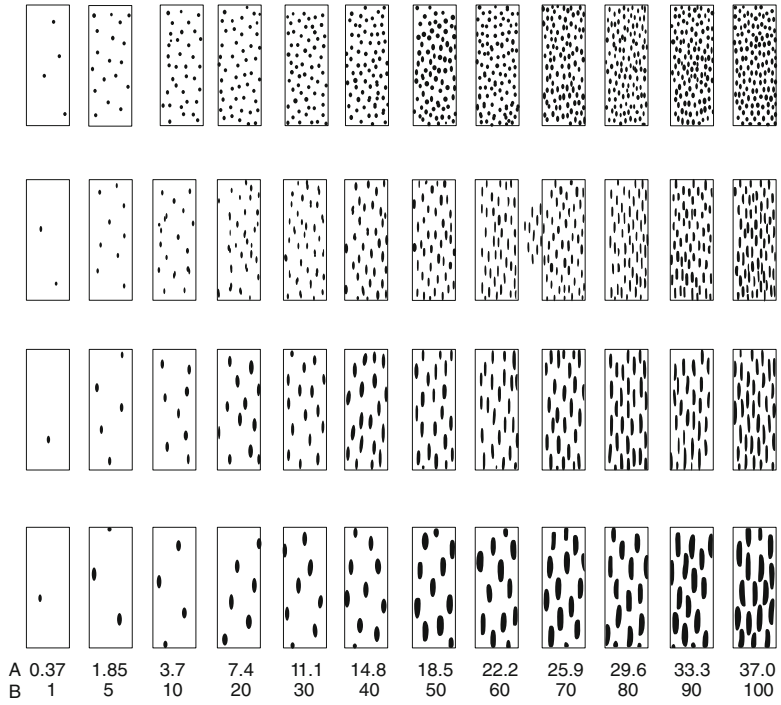

Fig. 9.2 Disease appraisal scale for rusts the modified Cobb scale (**A**) actual percentage occupied by rust uredinia; (**B**) rust severities of the modified Cobb scale. *Source*: after Peterson et al. (1948), Roelfs (1992)

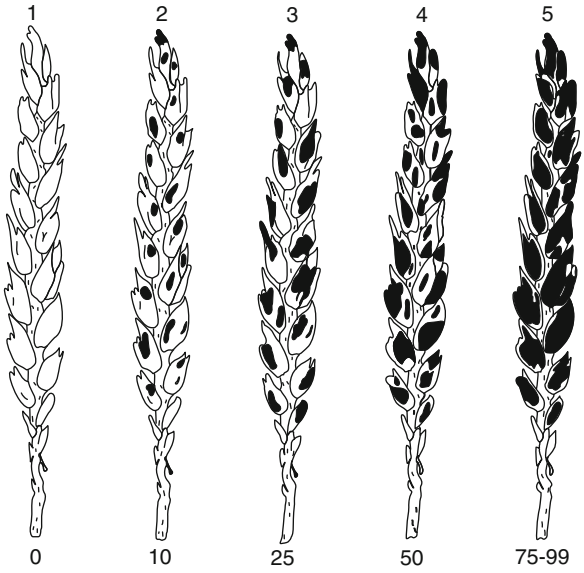

Fig. 9.3 Disease appraisal scale for scab-the fusarium head blight (FHB). *Source*: James (1971a, b)

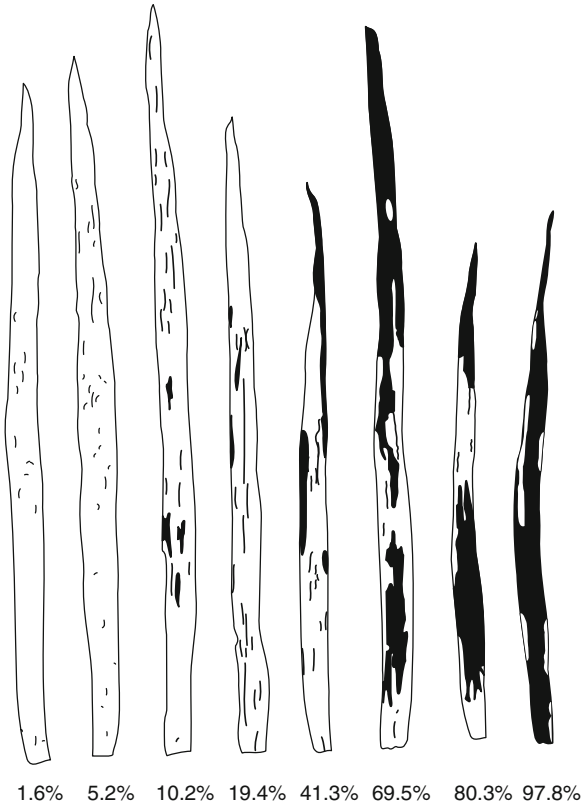

Fig. 9.4 Disease appraisal scale for Helminthosporium leaf blights

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