Diseases of Edible Oilseed Crops

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Foreword

Annual edible field-grown oilseed crops have wide adaptability and are grown under varied agroclimatic conditions. They occupy a special place in agricultural economies all over the world. Almost all such crops have a great potential for the diversification of major cropping systems in developing countries. In the period 2009–2012, these crops recorded impressive compound growth rates in terms of area and production. However, the average yield per hectare is hardly 30%–50% of what is obtainable under real-farm situations in rain-fed areas as well as in areas with assured moisture supply in developing countries. One of the major reasons for this huge gap is the occurrence of diseases that adversely affect these crops.

In the war against hunger and malnutrition, it is necessary to enhance and update knowledge about these diseases, their occurrence, epidemiology, and disease management, including transgenic technology. To this end, the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) has developed and characterized transgenic peanut (groundnut) lines with aflatoxin resistance conferred by the rice chitinase gene and also lines with bud necrosis virus resistance imparted by expressing the viral coat protein gene. Elsewhere, similar approaches in the development of stem necrosis virus–resistant transgenic sunflower and *Alternaria*-resistant transgenic mustard have been cultivated. Enriched with these recent developments, this book is useful as an updated basic reference volume in the conduct of research and development activities toward obtaining increased productivity and sustainability of oilseed production in the world.

In view of this, I appreciate and compliment all three authors for bringing out this important book. I hope that readers of this book will contribute to pushing forward the frontiers in the war against hunger and malnutrition.

William D. Dar
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Preface

The proposal for bringing out this book is the result of the pressing need and demand from edible oilseed crop researchers, university faculty members and students, and the interest taken by the publishers in the proposed authors based on the previously published book *Diseases of Annual Edible Oilseed Crops* by CRC Press in 1984–1985. The new volume is an improved one covering the latest developments in host–pathogen interactions and disease management, including molecular breeding for disease resistance and developments of transgenics, if any, in edible oilseed crops. Accordingly, the proposal was prepared and submitted for review along with the expert opinion of scientists as per CRC Press norms and regulations, and was approved after incorporating suggestions given by the experts.

This book deals primarily with the diseases of cultivated annual edible oilseeds, that is, peanut (groundnut), rapeseed–mustard, sunflower, sesame, safflower, and soybean. In recent years, soybean, though not a high-oil-containing seed, has been identified more as an oilseed crop than a bean crop by the United Nations Food and Agriculture Organisation (UN FAO). Diseases of other annual crops, for example, cotton, corn, rice bran, and perennial oil palm, which also contribute significantly to the world supply of edible vegetable oils and fats, have been excluded. Linseed oil is mostly useful for industrial purposes. However, a new edible grade oil crop termed *Linola* has been created (through collaborative research between Australia and the United States) out of conventional linseed varieties through mutation breeding, which is likely to expand as a good source of vegetable oils for human consumption.

A great deal of information has been accumulated on the diseases of peanut, rapeseed–mustard, sesame, sunflower, safflower, and soybean since the publication of the 1984–1985 edition. Besides a pressing need for such a comprehensive work, the experience of the authors in research in this pertinent field has prompted the attempt to bring together the scattered information on the subject in a comprehensive manner in order to present it in a useful form. An attempt has been made to present a broader view of the subject than that generally included in bulletins and manuals. Discussions on the development of a straightforward and also of a controversial nature have been included to stimulate thinking especially among graduate students. The information presented represents a careful synthesis of research articles. The survey of literature has been made as complete as possible up to the beginning of 2014. In most cases, original papers are consulted, and the temptation to use review articles or abstracts as a major source of information is avoided.

The "Introduction" deals with the uses and chemistry of vegetable oils and fats, trends in world production and consumption, production constraints, crop management, and disease problems. Depending upon the available literature, the treatment of all the previously mentioned crop diseases follows a uniform pattern under headings such as Symptoms, Geographical Distribution and Losses, Pathogen, Epidemiology, Disease Cycle, and Diseases Management covering Host Plant Resistance, Molecular Breeding, Cultural Control, Biological Control, etc., in each chapter. The aim has been to make the subject matter regarding each disease as complete and self-contained as possible. At first, the reader is introduced to the respective edible oilseed crop in each chapter with a brief botanical description of the crop and its genomics, origin, and distribution. The diseases are arranged under each crop on the basis of their global economic importance.

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Acknowledgments

The authors first wish to express their appreciation to their respective work organization for providing them with opportunities to independently investigate the disease problems of oilseed crops: the Indian Council of Agricultural Research (C. Chattopadhyay), G.B. Pant University of Agriculture and Technology (S.J. Kolte), and the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) (F. Waliyar). It is basically because of this research work and their experience as the team leaders of the oilseed pathology discipline in their respective institutes that the authors could come together and make a genuinely convenient and desirable, firm, support base to write this book, while being involved at some time or an other in some active collaborative research on certain aspects of edible oilseed crop diseases.

During the process of preparing the manuscript, the authors sacrificed time they could spend with their family members and friends. They were constantly encouraged by their near and dear ones to prioritize the writing of this book. C. Chattopadhyay acknowledges the encouragement given by his parents (Amiya Kumar and Arati Chattopadhyay), wife (Aparna), daughter (Nivedita), teachers (Dr. Chitreshwar Sen and Dr. Bineeta Sen) and research managers (Dr. S. Ayyappan [DG-ICAR], Dr. S.K. Datta [DDG-CS-ICAR], and all mentors, scientists, staff of ICAR: NCIPM, other scientist colleagues, all dear friends, and other relatives, especially Chaitali. S. J. Kolte expresses his appreciation to his wife, Rekha, for being the best critic and for being very supportive of him continuing his job even after retirement, and to his great teacher, Professor Y.L. Nene, for encouragement. F. Waliyar acknowledges the encouragement from his family by dedicating this book to his parents and to Marion, Simine, Edrice, Engela, Martin, Charline, and Alice.

Most of the illustrations, particularly the photographs of the signs and symptoms of different diseases, are the results of the authors' original work. A few photographs have been obtained from other scientists working in India, the United States, and Nigeria, whose help is acknowledged in the captions. The following people deserve particular mention: Dr. Tom Gulya, research plant pathologist, USDA-ARS, Northern Crop Science Lab, Fargo, North Dakota, USA; Dr. Shrishail Navi, associate scientist, Department of Plant Pathology and Microbiology, Iowa State University, Ames, USA; Dr. B.A. Tunwari, Department of Crop Production and Protection, Federal University, Wukari (Taraba), Nigeria; Dr. H. Nahunnaro, Department of Crop Protection, Modibbo Adama University of Technology, Yola, Nigeria; Dr. G.K. Gupta, head, Division of Crop Protection, ICAR-Directorate of Soybean Research, Indore, India; Dr. S. Chander Rao, principal scientist, and Dr. K.S. Varaprasad, project director, ICAR-Directorate of Oilseed Research, Hyderabad, India; Dr. Anil Kotasthane, head, Department of Plant Pathology, Indira Gandhi Krishi Vishwavidyalaya, Raipur, India; and Dr. A.K. Tewari, professor of plant pathology, G.B. Pant University of Agriculture and Technology, Pantnagar, India.

Some of the figures are partly adapted from previous published records, and the sources of illustrations have been included as needed. The authors are grateful to Manish M. Patil for his help in sending the finished manuscript to CRC Press. The courteous cooperation in all matters relevant to the development of this book that the authors received from the publisher, especially Randy Brehm, acquiring editor and main contact person, and Kari Budyk, senior project coordinator, CRC Press, is gratefully acknowledged.

The authors are grateful to Dr. William D. Dar, director general, ICRISAT, Patancheru, Telangana, India, for writing the foreword for this book.

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Authors

Chirantan Chattopadhyay is currently the director of the National Research Centre for Integrated Pest Management (NCIPM) in New Delhi under the Indian Council of Agricultural Research (ICAR) system. He earned a PhD in mycology and plant pathology from the Indian Agricultural Research Institute (IARI), New Delhi, and is a fellow of the Indian National Academy of Agricultural Sciences. He has more than 22 years of research experience in oilseed crop disease management and has significantly contributed in the areas of understanding the host–pathogen interactions, epidemiology, and integrated disease management in sunflower, safflower, sesame, peanut, and rapeseed–mustard crops. He has published more than 70 peer-reviewed research papers and 4 books. Dr. Chattopadhyay has to his credit several academic awards and one patent. His significant research achievements include the development of *Fusarium* wilt–resistant genotypes in safflower, the identification of genotypes of *Brassica juncea* resistant to a mixture of isolates of *Albugo candida*, the development of a biocontrol agent formulation to reduce the use of fungicides in the management of major diseases of oilseeds (*Brassica*), and seed treatment (and foliar application) of aqueous bulb extract of *Allium sativum* for the management of major foliar diseases (*Alternaria* blight, white rust, powdery mildew, and *Sclerotinia* stem rot) of mustard.

S.J. Kolte, professor of plant pathology (Retd.), has 36 years of experience in teaching both undergraduate and postgraduate courses in the Department of Plant Pathology, G.B. Pant University of Agriculture and Technology, Pantnagar, India. He has contributed considerably in the areas of oilseed pathology and plant pathology by being principal investigator of three international collaborative research projects funded by the Department for International Development (United Kingdom) and the International Development Research Centre (Canada) on disease and drought resistance in oilseed crops for more than two decades. He has contributed outstanding research work in the area of oilseed crop pathology, written 8 books and guided 26 students for their PhD and MS programs. He has also published 125 research papers in reputed national and international journals and has contributed several review articles and book chapters. In recognition of his contributions in plant pathology, Dr. Kolte has been bestowed with more than half a dozen awards, notable among them being the Lifetime Achievement Award by the Society for Rapeseed–Mustard Research, Y.L. Nene Outstanding Best Teacher award by the Indian Society of Mycology and Plant Pathology, and the IPS Recognition Award by the Indian Phytopathological Society. Induced host resistance, genetics of host resistance, and development of a new concept of the Divya mustard ideotype are some of his significant contributions.

Farid Waliyar is a principal scientist of plant pathology at the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT). He is currently ICRISAT director for West and Central Africa with regional office in Bamako. He earned a PhD in plant pathology (1984) from the University of Paris M&C, where he specialized in research on the role of *Aspergillus flavus* and safety measures for aflatoxin contamination and other soil fungi on peanut and their evolution during the cropping season in Senegal. Dr. Waliyar has a total of 30 years experience in strategic basic research related to the development of integrated crop disease management particularly in the peanut crop. He has published widely (more than 200 publications) as well as supervised and examined several undergraduate, MS, and PhD students. He has expertise in administration and management of research projects involving international collaboration. Dr. Waliyar has received several awards, the most recent from the Malian Ministry of Agriculture in recognition of his contribution to the development of agriculture in Mali.

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Section 1

Introduction

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1 Edible Oilseed Crops

Edible oilseed plants are those whose seeds bear fixed nonvolatile oil, and oilseed crops are grown primarily for the oil contained in the seeds. The oil content of small grains (e.g., wheat) is only 1%–2%, and that of oilseeds ranges from about 20% for soybeans to over 40% for sunflowers and canola rapeseed. Crops like rapeseed–mustard, peanut, and sunflower have oil recovery ratio of 45%, 40%, and 30%, respectively, whereas cottonseed and soybean have oil recovery ratio of 11.5% and 17% only (Kumar 2014). Some of the oilseeds like peanut, sesame, sunflower could be consumed directly or may be eaten fried, roasted, or pounded and mixed with sugar; or the oil may be extracted from such seeds and directly used for cooking food or for confectionery purposes. Usually, refining of the oil is done before it is used as food. Edible vegetable oils may, however, be used occasionally for industrial purposes, for example, manufacturing of soaps, varnishes, hair oils, and lubricants. The residues left, that is, the oil cakes, serve as excellent animal or poultry feed. Oil cakes may also be used as manure to increase the fertility status of soils. The demand for edible oilseeds for human consumption in different parts of the world is principally derived from three categories of cultivated crop plants: (1) primarily cultivated annual oilseed crops, for example, peanut (*Arachis hypogaea* L.), rapeseed–mustard (*Brassica campestris* L., *Brassica napus* L., *Brassica juncea* [L.], Czern and Coss. *Eruca sativa* Lam.), sunflower (*Helianthus annuus* L. var. *macrocarpus* [DC] Ckll.), sesame (*Sesamum indicum* L.), safflower (*Carthamus tinctorius* L.), niger seed (*Guizotia abyssinica* Cass.), and soybean (*Glycine max* [L.] Merrill); (2) an annual fiber crop cotton *(Gossypium hirsutum* L.) through its seed by-products; and (3) perennial oilseed plants such as coconut palm (*Cocos nucifera* L.) and oil palm (*Elaeis guineensis* Jacq). Corn (*Zea mays* L.) also contributes significantly to the world edible oil supply. Besides traditionally grown oilseed crops, technological innovations in refining, bleaching and deodorization, newer oils like cottonseed and rice bran oils have also become popular in the recent times. Thus, the range of plants that could be cultivated for edible oils is extensive, but only a few that are included in the first (1) category are suitable for large-scale commercial production or produce oil that is required in large quantities. In this chapter, only this category of primarily cultivated annual oilseed crops is considered with respect to diseases and their management.

CHEMICAL NATURE OF EDIBLE OILS AND FATS

Edible oils and fats of vegetable origin are composed of triglycerides that are esters of one molecule of glycerol and three molecules of fatty acids. A reaction leading to the formation of a triglyceride is shown in Figure 1.1.

Triglycerides that are solids at room temperature are termed as *fats*, whereas the liquid ones are termed as *oils*. The latter contain ester-bound unsaturated fatty acids.

Fatty acids by and large are straight-chain aliphatic monocarboxylic acids. Most of the members of this series contain an even number of carbon atoms in the molecule. Individual fatty acids are distinguished from one another by the nature of the hydrocarbon chain. This chain can vary in length from 4 to 24 carbon atoms. When fatty acid contains one or more double bonds in the molecule, it is said to be unsaturated. Thus, the fatty acid may be saturated (no double bond) as stearic acid, monounsaturated (one double bond) as oleic acid, or polyunsaturated (with two or more double bonds) as linoleic acid. The fatty acids are abbreviated according to the number of carbon atoms in the molecule and degree of unsaturation (number of double bonds). The common names,

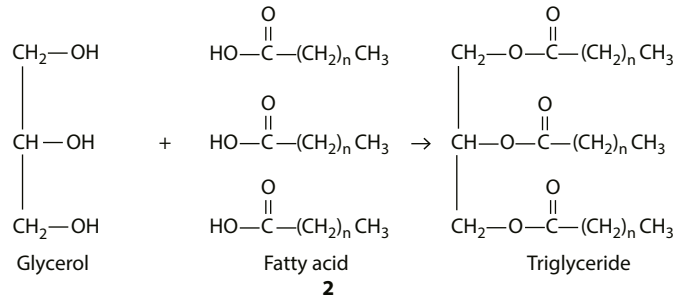

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FIGURE 1.1 A chemical reaction leading to the formation of triglyceride.

TABLE 1.1
Common Names, Symbols, Systematics, and Structural Formulae of Certain Important Fatty Acids Found in Vegetable Oils

Common Name	Symbol	Systematic	Structural Formula
Saturated fatty acids (*1–4*)			
1. Myristic	$C_{14.0}$	Tetradecanoic	$C_{13}H_{27}COOH$
2. Palmitic	$C_{16.0}$	Hexadecanoic	$C_{15}H_{31}COOH$
3. Stearic	$C_{18.0}$	Octadecanoic	$C_{17}H_{35}COOH$
4. Arachidic	$C_{20.0}$	Eicosanoic	$C_{19}H_{39}COOH$
Unsaturated fatty acids (*5–10*)			
5. Palmitoleic	$C_{16.1(9)}$	9-Hexadecenoic	$C_{15}H_{29}COOH$
6. Oleic	$C_{18.1(9)}$	9-Octadecenoic	$C_{17}H_{33}COOH$
7. Linoleic	$C_{18.2(9,12)}$	9,12-Octadecadienoic	$C_{17}H_{31}COOH$
8. Linolenic	$C_{18.3(9,12,15)}$	9,12-Octadecatrienoic	$C_{17}H_{29}COOH$
9. Gadoleic	$C_{20.1(9)}$	Eicosenoic	$C_{19}H_{37}COOH$
10. Erucic	$C_{22.1(13)}$	13-Docosenoic	$C_{21}H_{41}COOH$

Sources: Dutcher, R.A. et al., *Introduction to Agricultural Biochemistry*, John Wiley & Sons, New York, 1951, p. 72; Vaisey-Genser, M. and Eskin, N.A.M., Canadian rapeseed oil—Properties, processes and food quality, Publication No. 54, Rapeseed Association of Canada, Winnipeg, Manitoba, Canada, 1978, p. 13.

Note: Figures in the parenthesis indicate the position of double bonds (=) in the fatty acid chain at carbon numbers starting from carboxyl group.

abbreviated symbols, systematic, and structural formulae of certain important fatty acids found in vegetable oils are given in Table 1.1. The natural configuration of fatty acids is the *cis* configuration, which is considered to be nutritionally more desirable.

TRENDS IN WORLD PRODUCTION AND CONSUMPTION OF VEGETABLE OILS AND FATS

Commonly Cultivated Annual Edible Oilseed Crops

The oilseeds sector has remained vibrant globally with 4.1% growth per annum in the last three decades. The production of annual oilseed field crops has increased considerably since 1960, and now constitutes over 50% of the total production of fats and oils in the world. However, the supply of vegetable oils from annual field crops tends to remain quite flexible from year to year in relation to the total world supply of vegetable oils and fats (Sharma et al. 2012). The present average

TABLE 1.2
Average World Oilseed Production (2010–2012) and Projected Oilseed Crop Production (2022)

	Production (kilotons)		Growth (%)		Per Capita Food Use (kg/annum)	Per Capita Food Use (kg/annum)
World/Country	Average (2010–2012 Estimated)	Projected (2022)	2003–2012	2013–2022	Average (2010–2012 Estimated)	Projected (2022)
World	300,414	400,460	3.13	2.07	18.3	20.0
Developed countries	165,474	203,242	3.70	1.63	24.0	24.4
North America	107,682	128,468	2.35	1.13	37.9	32.2
Canada	18,184	23,367	8.05	1.62	23.9	20.8
United States	89,497	105,101	1.44	1.03	39.5	33.5
Europe	52,349	66,678			21.8	23.3
Oceania developed	2,861	4,130	9.33	2.53	26.4	27.0
Developing countries	224,040	28,728	2.71	2.39	16.7	19.0
Africa	10,043	12,910	1.11	2.65	11.4	12.4
Latin America and Caribbean	139,470	189,415	4.17	2.84	19.2	22.3
Asia and Pacific	175,427	84,893	0.62	1.42	17.7	20.5
China	44,380	47,951	0.01	1.20	21.9	26.3
India	23,222	27,165	1.29	1.71	13.3	16.0

Source: OECD-FAO, OECD-FAO agricultural outlook 2013, Chapter 5—Oilseeds and oilseed products, OECD/FAO Secretariats, pp. 139–282.

per capita consumption of edible oils and fats is 39.5 kg/annum (highest) in the United States, 13.3 kg/annum (low) in India and other south Asian countries, and 11.4 kg/annum (lowest) in Africa. Thus, the consumption of fats and oils in Asia and Africa and in other developing countries is much less as against the required minimum consumption level of 30 kg/annum (Table 1.2). This is a serious situation, particularly when it comes to meeting the requirement of an essential fatty acid, linoleic ($C_{18.2}$), and the energy supply for body functions under a balanced diet pattern. Considering the global minimum per capita consumption as required for keeping human health, the increasing world population by about 2% every year, the present rate of oilseed production on a global scale is not and will not be satisfactory. However, developed countries such as the United States, Canada, and the Russian Federation have been and should continue to be the major producing areas. Population growth and rising per capita income are expected to lead to an average 2.1%/annum growth of food vegetable oil use in developing countries. Annual food vegetable oil use per capita is expected to average 19 kg/annum across developing countries, but no more than 9.5 kg/annum in least developed countries by 2022. As a group, developed countries are showing a stable consumption level of 24–25 kg/annum, but individual countries differ based on tastes and preferences (OEDC-FAO 2013). Biotechnology offers a number of solutions to meet the growing need for affordable vegetable oils with improved fatty acid composition for food and industrial uses (Lu et al. 2011). The six annual edible oilseed crops, as considered in this chapter, are grown in different parts of the world, covering a wide range of geographical areas. Total world's oilseed production from major oil crops has been 423.55 million tons from 205.08 million hectares during 2009–2010. The leading countries in oilseed production are the United States, Brazil, Argentina, China, and India (Yadav et al. 2012). The yield of these crops is of higher magnitude in the developed countries as compared with the developing ones (Table 1.2). For example, the average yield of peanuts in the developed countries, particularly in the

TABLE 1.3
Edible Oilseed Crop Productivity (q/ha) in India vis-à-vis World (2012)

Crop	India	World	Country with Highest Productivity[a]
Peanut	11.7	16.7	46.9 (United States)
Rapeseed–mustard	11.4	18.7	36.9 (Germany)
Soybean	12.0	23.7	27.8 (Paraguay)
Sunflower	07.6	14.8	24.9 (China)
Sesame	04.26	05.1	13.1 (Egypt)
Safflower	06.5	09.6	14.8 (Mexico)

Sources: FAO, FAOSTAT world oilseed production, 2012, available at: http://faostat.fao.org; Paroda, R.S., The Indian oilseeds scenario: Challenges and opportunities, in: *The First Dr. M.V. Rao Lecture*, Indian Society of Oilseeds Research, Hyderabad, India, August 24, 2013, p. 26.

[a] Among the countries with >80% global contribution.

United States, is 46.9 q (quintals)/ha; whereas in India and in other semiarid countries, it is only about 11 q/ha as given in Table 1.3 (FAO 2012, Paroda 2013). A similar situation appears to be true with respect to the high production of rapeseed (now canola) in Canada and sunflower in Russian Federation, compared with the yield performance of these crops in developing countries. Safflower production is about 16.4 q/ha in the United States, 16.8 q/ha in Mexico, and only 6.3 q/ha in India (FAO 2011, Padmavati and Virmani 2012). The average yield of sesame varies from a high of 11.75 q/ha in Egypt to a low of 1.52 q/ha in Sudan (Ranganatha et al. 2012).

Linola: A New Annual Edible Oilseed Crop

There are reports showing that in certain linseed species extent and degree of polyunsaturated fatty acids are so low that the oil extracted is perfect for edible purposes. For example, *Linum strictum* L. is largely cultivated for edible oil and fodder purposes in Afghanistan (Richaria 1962). The edible oils are characterized by rather having higher content of oleic, palmitoleic, and linoleic acids. The linseed oil obtained from the seeds of *Linum usitatissimum* cannot normally be used for edible purposes directly or in the edible products because of its high linolenic acid contents; though in certain regions of Chhattisgarh (formerly a part of Madhya Pradesh) and adjoining eastern part of Vidarbha Region of Maharashtra State in India, linseed oil is used for cooking food. It is noteworthy that Green (1986a,b) has been successful in obtaining two low-linolenic acid (28%–30%) mutants *M1589* and *M1722* by treating the seeds of the linseed (*Linum usitatissimum* L.) cultivar *Glenelg* with ethyl methanesulfonate. The two mutants through crossing together have been further combined within a single genotype that has only 1% linolenic acid and increase in linoleic acid to 50%–70% depending on the temperature during seed maturation. Consequently, through traditional plant-breeding procedures, a joint venture between Commonwealth Scientific and Industrial Research Organization (CSIRO) of Australia and United Grain Growers Ltd. of Winnipeg, Canada, has led to the development of edible linseed oil (Gunstone 2011). The fatty acid composition of the new oilseed crop named *linola* (a registered trademark of CSIRO) has been changed, and the level of linolenic acid substantially reduced from 50%–60% to 2%. This greatly increases the oxidative stability of the oil that is a polyunsaturated oil identical to sunflower, safflower, or corn oil in fatty acid composition. The oxidative stability of oil of this newly created linseed genotype is equivalent to that of sunflower oil and much better than high-linolenic common linseed oil (Green 1986a–c). The color of the linola seed is also changed to pale yellow, which allows it to be distinguished from brownish traditional flaxseed/linseed.

The new oilseed crop can be grown wherever flax and linseed varieties are currently cultivated. The climate in northern Europe is highly suitable for the production of linola where sunflower and

corn cannot be produced. Linola seed can be processed in existing crushing plants using standard procedures, and linola meal can also be used in ruminant feed in the same way as linseed meal. Refining of crude linola oil by conventional steps produces a pale-colored oil with good oxidative stability. The Food and Drug Administration has given GRAS approval to linola (Solin: the common generic name) oil for use as a general-purpose cooking oil, frying, and salad oil. Thus, linola oil and seed of the new oilseed crop appear to have a promising future. Anticipating the adoption and likely expansion of acreage of linola as a new edible oilseed crop, the linola crop is likely to be affected by the same diseases that affect the traditional nonedible grade linseed/flax (Kolte and Fitt 1997).

PRODUCTION CONSTRAINTS

Basic Constraints

It is true that high-yielding varieties of oilseeds do not have the genetic potential to yield at par with cereals, even at the optimum management level. Besides, it could be observed that production of a unit quantity of fats and oils by a plant requires more energy than production of carbohydrates by cereals. In making comparisons, one should always keep in view the differential energy requirements for the plants to produce a quintal of oil. If, for example, a plant produces 1 g of glucose, the conversion of this results in the formation of 0.83 g carbohydrate, while if glucose is converted into lipid, only 0.38 g is formed (Swaminathan 1979). It is because of this high-differential energy requirement that oil yield from oilseeds has continued to be restricted.

Other Constraints in Developing Countries

Poor plant population arising from poor-quality seed, particularly in the case of soybean, peanut, and sunflower, inadequate nutrient status of soil and nutrient supply, no rhizobial inoculation or use of inefficient rhizobial cultures in the case of soybean and peanuts, poor plant protection measures, and poor postharvest technology have been some other constraints for poor yields of oilseeds in developing countries. Besides, much of the oilseed acreage in developing countries—particularly in India—is rainfed, and therefore, a certain degree of instability is inherent in the production process. Absence of rain or lack of irrigation water at critical stages of the crop growth before maturity causes significant loss in yield. Thus, productivity in developing countries is still low compared to other oilseed-producing countries in the world. The main cause is low cultivation of oilseeds on account of switchover to other profitable crops and dependence on rainfall rather than on irrigation (Narayan et al. 2011).

CROP MANAGEMENT

Oilseed crop management must be seriously considered in view of the very low yield of these crops in developing countries. Considerable advancement in research has led to an increase in the productivity of the oilseed crops, both in developed and in developing countries particularly in China and India. In some crops, like and safflower, it is now possible to plan on the exploitation of hybrid vigor. Higher productivity of the sunflower in Canada and other developed countries is attributed to the cultivation of hybrid cultivars. In developing countries, adoption of a package approach (technological package) supported by package of services (seed, fertilizer, chemical supplies, etc.) constitutes an important major thrust to intensify oilseed production. There is still considerable scope for introduction of short-duration varieties of oilseeds in irrigated as well as in dry-farming systems favoring multiple cropping pattern all over the world.

It becomes necessary to obtain a thorough updated knowledge of a particular crop in terms of land preparation, techniques of sowing, varieties, fertilizer requirements, and intensive care during

crop-growing season. Preparation of seedbeds, with sufficient conservation of soil moisture, is necessary for the most oilseed crops with special reference to peanut, sunflower, and rapeseed crops. Seed treatment with most recently recommended fungicides (thiram, carbendazim, or with a mixture formulation of such fungicides) at the rate of 2–3 g/kg of seed may be necessary for the soybean, peanuts, and sunflowers to get good seed germination and plant stand, directly increasing yields through such treatment. Some crops like rapeseed and mustard are still sown by broadcast method in India. It has now been demonstrated through planned field experiments and on-farm farmers participatory research that the yield of rapeseed–mustard crop can be increased considerably by line sowing. It is, therefore, considered best that rapeseed–mustard crop be sown in lines through seed drills. The requirements for fertilizers will be determined by the fertility status of the soil, the nature of oilseed crop to be grown, and time of sowing. For the peanut crop, application of calcium through gypsum may be quite important for better pod and seed development. Some other nutritional problems with respect to deficiency of boron, zinc, and iron have been encountered in oilseed crops in different geographic areas. A direct yield loss of U.S. $1.5 billion/annum is estimated due to low crop yields besides huge loss due to disease concerns arising out of Zn malnutrition in the country (Singh 2010, Suresh et al. 2013). Timely steps should be taken to correct the aforementioned deficiencies. Other management practices include spraying of suitable insecticides and fungicides at the appropriate time for the management of insect pests and diseases. In the case of rapeseed–mustard, the crop must be essentially protected from aphid attack under Indian conditions.

DISEASE PROBLEMS

Peanut, rapeseed–mustard, sunflower, sesame, safflower, and soybean are subject to attack by several infectious and noninfectious diseases. The loss in yield of the crop may vary, depending upon the nature of the pathogen and the severity of the attack. Considering all the vegetable oil–producing crops, the quantity lost, on a world basis, is estimated to be more than about 14.00 million tons/year—amounting to a monetary loss of about U.S. $16 million. This excludes the newly developed diseases for which loss estimates have not yet been determined. Thus, the overall losses may be of a higher magnitude. With an increasing emphasis on oilseed production, it is expected that limited land resources through intensive farming, higher cropping intensity, better seeds, and greater use of fertilizers and herbicides, the production of oilseeds will increase; however, this might create new disease problems under the changed environments, in addition to the already existing diseases. Such a shift in the disease situation, as discussed in the following chapters, has already taken place in the case of peanuts due to the use of benomyl for early and late leaf spot management, consequently favoring more peanut rust and Sclerotium rot development in the United States, and with respect to rapeseed–mustard due to the use of Barban® herbicide favoring development of *Sclerotinia* rot in Canada. Use of dalapon herbicide has increased the susceptibility of rapeseed to light leaf spot (*Pyrenopeziza brassicae* Sutton and Rawlinson) in the United Kingdom (Kolte 1985). A similar situation appears to be true with respect to nutrient status and susceptibility of rapeseed and sunflowers to fungal diseases at a lower concentration of erucic acid and glucosinolates. Derivatives of glucosinolates have been known to be fungitoxic. Some volatile derivatives of glucosinolates are reported to be more abundant in light leaf spot–resistant varieties than in susceptible types of rapeseed. So, the consequences of this trend, that is, breeding for low glucosinolates and for other quality characters, must be thoroughly examined in the general context of rapeseed diseases. Although climate change and variability is considered an altering situation and a big challenge to oilseed production, there is sometimes a positive impact of it regarding the disappearance of sesame phyllody disease (caused by phytoplasma) in an unusually cool and rainy growing season in the west Mediterranean region of Turkey. This is a unique case of influence of climate variability characterized by higher and frequent rainfalls and consequently causing lower temperatures but higher humidity on the *nonoccurrence* of phyllody disease transmitted and

spread by leaf hopper vectors (Cagirgan et al. 2013). In contrast to this insect- and vector-borne phytoplasma, the fungal pathogen, *Sclerotinia sclerotiorum*, and several other pathogens find such weather conditions with higher rainfalls and lower temperatures most congenial to cause epidemics in rapeseed–mustard and sunflowers (Boomiraj et al. 2010, Evans et al. 2010).

Oilseed crops are affected by foliage diseases such as the rusts, downy mildews, leaf spots and blights. The management of these diseases through the use of chemical sprays and host resistance has been achieved in a satisfactory manner, but the situation with respect to control of a number of soil-borne root diseases, for example, charcoal rot, *Sclerotinia* rots, *Verticillium* wilts, and *Fusarium* wilts, is not satisfactory. Oilseed crops have a rather low-yield genetic potential. Therefore, the least expensive management measures, such as use of host resistance and cultural control, will find favor with farmers and others concerned with more oilseed production. In recent years, the gains in productivity of oilseed crops have been achieved primarily through exploitation of genetic variability (Anjani 2012, Azeez and Morakinyo 2011, Zhang and Johnson 1999). Conventional breeding coupled with modern tools such as biotechnology should now be the primary focus in crop improvement programs. Investigations to develop disease-resistant transgenics are underway all over the world. In India, for example, *Alternaria*-resistant mustard transgenics using antifungal chitinase and glucanase genes have been successfully developed. Similarly, transgenic peanuts have been developed with coat protein genes for resistance to peanut bud necrosis and peanut stem necrosis viruses (Paroda 2013).

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Section II

Peanut

Peanut (groundnut) belongs to the family Fabaceae (= Leguminosae) and the genus *Arachis*, derived from the Greek *a-rachis*, meaning without spine, and refers to the absence of erect branches. The species name *hypogaea* is derived from *hupo-ge*, which in Greek means below the earth. It is an allotetraploid having South American origin (Nigam 2000, Wang et al. 2011). Recent studies reveal that peanut originated in northern Argentina or southern Bolivia from hybridization between the diploid wild species *Arachis duranensis* and *Arachis ipaensis*. Cultivated peanut thus has an allotetraploid genome (AABB, $2n + 4x = 40$). It is an annual herbaceous plant growing to a height of 30–60 cm with an angular hairy stem and spreading branches. Leaves occur alternately, one at each node; they are pinnate with two pairs of ovate leaflets. The peanut flowers are perfect, and self-pollination is the general rule although natural cross-pollination may occur at times. After pollination, the perianth withers and at the base of the ovary a meristematic region grows into a stalklike peg that pushes the ovary into the soil. Groundnut has a taproot system that is often covered with root nodules resulting from a symbiosis with nitrogen-fixing bacteria (collectively called rhizobia). Among the rhizobia identified on groundnut, *Bradyrhizobium* species are the most prominent ones. The rhizobia penetrate the root tissue, induce cell division, and settle inside root cells where they convert atmospheric nitrogen (N_2) into ammonia, which in turn is used by the plant.

Because its ancestors were two different species, today's peanut is a polyploid, meaning this species can carry two separate genomes, designated as A and B subgenomes. *A. duranensis* serves as a model for the A subgenome of the cultivated peanut, while *A. ipaensis* represents the B subgenome. Very recently in April 2014, peanut genome has been successfully sequenced as a result of the collaborative research done by the International Peanut Genome Initiative (IPGI)—a group of multinational crop geneticists from the United States, China, Brazil, India, and Israel. The peanut genome sequences will now provide researchers access to 96% of all peanut genes in their genomic context and provide the molecular map needed to more quickly breed for peanut disease resistance and other economically important traits producing more improved high-yielding peanut cultivars all over the world (UGA Today—University of Georgia News Services—October 20, 2014; Mallikarjuna and Varshney 2014).

The varieties in cultivation fall into two main groups: the bunch or erect and the runner or spreading types. The basic chromosome number of *Arachis hypogaea* is 20 pairs ($2n = 40$) with large genome size (2.82 Gb DNA, 2800 Mb/1C). The oil content of the seed varies from 44% to 55%, and protein content of the seed is about 25%–28% in different varieties. About two-thirds of the world's production is crushed for oil and the remaining one-third is consumed as food. Today, peanut is

widely distributed and is cultivated in more than 80 countries in tropical and subtropical regions of the world. Peanut requires warm, sunny climate with a well-distributed rainfall of at least 500 mm and temperatures ranging from 25°C to 30°C. It thrives best in well-drained sandy-loam soils with a pH ranging from 5.5 to 7.0. Asia with 63.4% area produces 71.7% of the world's peanut production followed by Africa with 31.3% area and 18.6% production and North–Central America with 3.7% area and 7.5% production. Important peanut-producing countries are China, India, Indonesia, Myanmar, Thailand, and Vietnam in Asia; Nigeria, Senegal, Sudan, Zaire, Chad, Uganda, Republic of Ivory Coast, Mali, Burkina Faso, Guinea, Mozambique, and Cameroon in Africa; Argentina and Brazil in South America; and the United States and Mexico in North America (Hegde 2009). The most favorable conditions for peanuts are moderate rainfall during the growing season, an abundance of sunshine, and relatively high temperature. The plants need ample soil moisture from the beginning of blooming up to 2 weeks before harvest. The crop thrives best on sandy-loam, loam, and well-drained black soils. The crop is affected by several diseases, causing large losses in both yield and quality of seeds. The peanut diseases are described in Chapters 2 through 4 as follows.

2 Fungal Diseases

SEED ROT AND SEEDLING DISEASE COMPLEX

Symptoms

A wide range of fungi, acting synergistically or in succession, attack the plant from the time seed is planted, until a few weeks after emergence to cause the seedling disease syndrome. The symptoms may be divided into four categories according to the development stage of the plant when the damage occurs. These categories are seed rot, preemergence damping-off, postemergence damping-off, and seedling blight. The rotted seeds become soft and mushy, turn brown, shrink, and finally disintegrate, often showing the presence of the fungal growth. This gives a patchy stand of the crop. The patchy stand of the crop may also be due to infection of seedlings after the seed has germinated but before the seedling has emerged above the soil level, which is called as preemergence damping-off. They may be killed even before the hypocotyls have broken the seed coat. The radicle and the plumule, when they come out of the seed, undergo complete rotting. Since this happens under the soil surface, the disease is often not visible except for the resulting patchy stand. In both cases, infection takes place before emergence of the seedlings above the soil level.

The postemergence seedling blight is characterized by the toppling over of infected seedlings any time after they emerge from the soil until the stem of the peanut plant soon becomes lignified and resistant to postemergence damping-off. The fungi that cause seedling blight symptoms infect the cotyledons and leaves as they emerge, either from inoculum carried on the seed or when the cotyledons come into contact with contaminated crop residues. Seedlings may also be attacked by certain species of fungi at the roots and sometimes at or below the soil line. Such seedlings usually show collar rot, root rot, brown root rot (caused by *Fusarium solani* [Martius] Sacc. in Argentina), and wilt-like and damping-off symptoms. In the case of collar rot caused by *Lasiodiplodia theobromae* (Pat.) Griff. & Maubl., the leaflets and stem remain green until the seedlings die and black pycnidia are found on the collar region at the soil level (Chi Mai Thi et al. 2006).

Geographical Distribution and Losses

These diseases occur all over the peanut-growing countries in the world and can cause serious reduction in yield through reduced plant stand. Losses recorded in any one field for a given inoculum level will vary from one season to the next depending on crop residues, soil conditions, seed quality, and climatic factors during the critical 3–4 weeks after planting. Extensive losses have been reported in the range of 25%–50% in Malawi, Senegal, Sudan, Niger, Nigeria, and other West African countries (Subrahmanyam et al. 1991, Kolte 1997, Thiessen and Woodward 2012), Southern African countries (Subrahmanyam et al. 1997), Egypt (Wakil and Ghonim 2000), India (Kolte 1984), and Pakistan (Riaz et al. 2002). Under conducive conditions like drought stress, losses due to brown root diseases in peanuts could reach 95% in some fields in Argentina (Rojo et al. 2007). Patchy stands of peanuts due to these diseases are the single most important factor for low production of the crop in almost all peanut-growing states of India.

Pathogens

The causal fungi associated with the preemergence seed rot, preemergence and postemergence damping-off of peanut seedlings, and seedling blights are *Rhizoctonia solani* Kuhn, *F. solani* (Mart) Apple and Wr., *Pythium ultimum* Trow., *P. myriotylum* Drechs, *P. debaryanum* Hesse emend. Middleton, *P. aphanidermatum* (Edson) Fitzp., and *P. butleri* Subram (Kolte 1984, Rashid et al. 2004, Cavallo et al. 2005, Thiessen and Woodward 2012).

Both the preemergence and postemergence damping-off of peanut seedlings can also be the result of infection of *Sclerotium rolfsii* Sacc., *Rhizoctonia bataticola* (Taub.) Butl., a sclerotial stage of *Macrophomina phaseolina* (Tassi) Goid (Chakrabarty et al. 2005), *Aspergillus niger* van Tieghem, and *A. flavus* (Link) ex Fries. The latter four fungal pathogens have the capacity to cause root rots, collar rot diseases beyond the stages of crop growth that could reasonably be considered as seedling. Diseases caused by these are described separately in "*Sclerotium* Stem Rot," "*Aspergillus* Collar Rot," "Yellow Mold and Aflarroot," and "Charcoal Rot" Sections under *F. solani* causing the brown root disease is one of the most serious diseases of peanuts in the southern region of Argentina in recent years (Oddino et al. 2008). Collar rot of peanut seedlings caused by *L. theobromae* (Pat.) Griff. & Maubl. (syn *Botryodiplodia theobromae* Pat.) has been reported to become a major disease problem in peanut production in North Vietnam (Chi Mai Thi et al. 2006). Two or more of the aforementioned pathogens can act together as a complex of the cause of seedling diseases.

Factors Affecting Infection

Normally, the peanut seed contains a tannin-like substance that acts as an antioxidant to retard the breakdown of the oil and entrance of the fungi that cause decay of seeds. Fungi associated with the peanut seed may decay it, particularly if it is damaged. In the mechanically damaged seeds, the seed coat is scratched and broken. Unless done with extreme care, machine-shelled seeds show reduction in germination by 25%–75%, as compared with hand-shelled seeds. The scratches on the seed surface provide points of entry of fungi. Delayed germination because of deep sowing or lack of moisture in the soil or waterlogging conditions may all influence the development of seed rot and seedling blight diseases within the first week after planting.

Soil temperature influences the involvement of the kind of pathogens in seedling disease complex and the severity of incidence of seedling diseases. For example, at higher soil temperature (>35°C), *R. solani* isolate from peanuts (a warm weather crop) is more virulent than the isolate from wheat (a cool weather crop) (Sreedharan et al. 2010). Thus, soil temperature as affected by planting date should be considered in areas where wheat is planted following peanut, if root disease caused by *Rhizoctonia* is a concern. There appear to be biotypes of *R. solani* capable of causing the disease over a wide range of optimum temperature. For seedling disease caused by *S. rolfsii* and *R. bataticola*, the optimum temperature is 25°C or above. There is a definite wide range of temperature optima for different species of *Pythium*. *P. ultimum* requires low soil temperatures, while *P. aphanidermatum* is more damaging at higher temperatures. Interestingly, *R. bataticola*, on the other hand, can survive and its incidence can substantially increase in infected peanut seed even at −18°C, a temperature recorded for long-term storage. The implication of these results is that for ensuring a high level of germination in peanut seeds under such situations, only dry and pathogen (*R. bataticola*) free or seeds with very low infection grade be used for long-term storage (Singh et al. 2003b).

Disease Management

Though there is a low to high range variation in the degree of susceptibility of peanut genotypes to seedling diseases caused by various pathogens, for example, *S. rolfsii*, there is the least possibility of obtaining acceptable level of seedling blight disease resistance in peanut seeds (Gour and

Sharma 2009). Therefore, a combined management strategy utilizing cultural, biological and chemical management practices is important in reducing losses caused by seed rot and seedling diseases in peanut crop production.

Cultural Control

Only sound seed without any evidence of injury should be used for sowing. Care should be taken to avoid injury to seeds during shelling and while the seeds are sown through seed drills. Hand-shelled seeds, if sown, without injury to the seed coat, give a higher stand of the crop even without fungicidal treatment. The loss in the crop stand because of seed rot and seedling blights can also be compensated by increasing the seed rate from 60 to 75 kg/ha. The effect appears to be similar to that of the fungicidal seed treatment. Crop rotation and tillage practices greatly influence the seedling disease complex in peanuts. For example, reduction of brown root rot of peanut seedlings caused by *F. solani* is greater in a 2-year rotation including corn–soybean or soybean–corn prior to peanuts than in a 1-year crop rotation, and the tillage system using the paratill subsoiler before seeding peanuts in a no-till system is a suitable strategy to improve peanut root growth and reduce the disease incidence, which provides a promising alternative in the control of peanut seedling brown root rot in Argentina (Oddino et al. 2008). In Egypt, seedling diseases of peanuts caused by *F. solani* and *R. solani* could be managed by amending the soil with gypsum (500 kg/acre) and by balanced application of nitrogen (100 kg/acre) and potassium (50 kg/acre) fertilizers as well as by soil moisture (55%–70% of field capacity) (El-Korashy 2001).

Chemical Control: Fungicidal Seed Treatment

The shelled seeds should be immediately treated with the fungicide rather than treating them after lapse of time. It should also be noted that no amount of seed treatment will change poor seeds into good ones when they are stored under conditions detrimental to their keeping quality. Therefore, seed treatment is not a corrective for improper storage. The seed should be treated with effective fungicide, usually a mixture of thiram and carbendazim (2:1) or thiram and carboxin (2:1) at 3% that takes care of the variety of seed-borne pathogens (Kolte 1994, 1997, Akgul et al. 2011). Seed treatment with a mixture of thiram and carbendazim at 2 g ai/kg seed is recommended as a routine treatment in plant quarantine labs in India to prevent the seed transmission of *R. bataticola* infection from one region to another (Chakrabarty et al. 2005). A mixture of the aforementioned three fungicides (thiram + carboxin + carbendazim) is known to be the most efficient seed treatment for the control of most seedling diseases of peanuts in Argentina (Cavallo et al. 2005), whereas a mixture of thiram and thiophanate methyl is reported to be the best in comparison to other fungicides (Meena and Chattopadhyay 2002). A number of other fungicides are recently investigated to be of potential use in the management of seedling disease complex of peanuts caused by Basidiomycetes such as *R. solani* (= *Thanatephorus cucumeris* (Frank) Donk.) and *S. rolfsii* (= *Athelia rolfsii* (Curzi) Tu & Kimbrough). These are triazole fungicides, like tebuconazole and propiconazole, and flutolanil and strobilurins, such as azoxystrobin. But these are not effective in controlling the infection caused by *Pythium*, which can, however, be controlled by seed treatment with phenylamides such as metalaxyl. Hence, if *Pythium* species is involved in the seedling disease complex, a component of metalaxyl be included in the mixture of fungicides for seed treatment (Thiessen and Woodward 2012). A new seed treatment fungicide *Stamina* (Headline) provides broader spectrum of control of seed-borne and seedling diseases caused by species of *Rhizoctonia*, *Pythium*, and *Fusarium*, which needs to be investigated for peanut crop.

Presowing treatment of peanuts with antioxidant hydroquinone (in 20 mM water solution) for 12 h is reported to be useful in completely inhibiting seed-borne pathogenic fungi and enhancing the plant growth parameters producing a 50% increase in yield (El-Wakil 2003). Use of properly cleaned seeds and integrating mancozeb seed treatment (3 g/kg seed) is reported to be beneficial in getting significantly higher seedling emergence and higher dry pod yield in eastern Ethiopia (Tarekegn et al. 2007).

Biological Control

Peanut seed pelleting with *Trichoderma harzianum* Rifai strain Th-5 is reported to be effective in protecting seeds from *M. phaseolina* infection by 79.6%, resulting in improved seedling vigor (Malathi and Doraisamy 2004). Similarly, seed treatment with another isolate of *T. harzianum* at 10 g/kg provides maximum protection to peanut seedlings, which is reported to be superior to fungicidal seed treatment under similar conditions (Rakholiya and Jadeja 2010). Rojo et al. (2007) have also reported the control of brown root rot of peanuts (*F. solani*) with *T. harzianum* strain ITEM 3636 through seed treatment in Argentina. The mass inoculum of the antagonist (*T. harzianum*) could be produced on low-cost agricultural waste products (rice bran, wheat bran, mustard cake) for the control of seedling collar rot of peanuts caused by *S. rolfsii* by seed treatment followed by soil application of *T. harzianum* along with farmyard manure (FYM) (Bhagat and Sitansu 2007). A combination of *Trichoderma* strain 5 MI with Rovral 50 WP fungicide and garlic extract gives the best control of seedling diseases caused by *R. solani* and *S. rolfsii* (Islam et al. 2005). The presence of *Bacillus subtilis* (Ehrenberg) Cohn has been naturally observed on peanut kernels. It is thus possible that the seeds that show the presence of this bacterium remain free from attack due to seed-borne fungi (Kolte 1984). El-Shehaby and Morsy (2005) from Egypt have demonstrated the usefulness of soil treatment of four isolates of an antagonist bacterium, *Bacillus sphaericus*, for the management of seedling diseases of peanuts caused by *R. solani*, *F. solani*, and *S. rolfsii*.

Extracts of leaves of *Azadirachta indica* A. Juss. (Kadam et al. 2008b) and *Moringa* seed (Donli and Dauda 2003) can be used for treating peanut seed, as alternative methods to fungicidal seed treatments for the control of seed and seedling diseases of peanuts.

EARLY AND LATE LEAF SPOTS

Symptoms

Peanut leaf spot diseases, the early leaf spot (ELS) and late leaf spot (LLS), are caused by two distinct, but closely related fungal pathogens. These are *Cercospora arachidicola* Hori (causing ELS) and *Phaeoisariopsis personata* (Berk. & Curt.) V. Arx. (= *Cercosporidium personatum* (Berk. & M.A. Curtis) Deighton) (causing LLS); both may occur simultaneously on the same leaf. (Other diseases too may cause spots on leaves, but they are not referred to as *leaf spots*). The ELS appears earlier about 10–18 days after emergence than the LLS, which appears 28–35 days after emergence or may appear at the time of harvest. In both cases, symptoms become visible as pale areas on the upper surface of the leaves. As the spot develops, it becomes yellow; necrosis occurs from the center of the lesion, and later the entire spot becomes necrotic. Infection with either leaf spot fungus produces hormonal changes in the leaf that cause leaf drop. Defoliation usually starts at the base of the central or lateral stem and then progresses upward. Initially, the ELS and LLS are indistinguishable. The distinguishing features, as the spots are fully developed, become quite evident as described under ELS and LLS.

ELS: Circular to irregular, larger, measures 1–10 mm in diameter; spots are characterized by a yellow halo of visible width (Figure 2.1). At maturity, the spots are reddish brown to black on upper surface but the lower surface of spot is distinctly orange in color. Cushions of conidiophores are formed at first on the upper surface (epiphyllous), but sometimes these are found on the lower surface of older spots and during periods of heavy cloud cover and frequent showers particularly when defoliation is at the peak (Figure 2.2); masses of clear to olive colored spores may be seen with the use of a hand lens on the upper surface of the spot; thus, the intensity of sporulation usually on the upper surface of the spot becomes a visible sign of the causal fungus.

LLS: Tends to remain distinctly round, 1.5–5.00 mm in diameter; yellow halos are not visible around the newly formed spots but are found only with mature spots. Spots are almost black on both surfaces, but lower surface of the spot is distinctly carbon black (Figure 2.3). Conidiophores are always found confined to the lower leaf surface (hypophyllous),

FIGURE 2.1 ELS of peanuts.

FIGURE 2.2 Defoliation of the peanut crop at harvest due to ELS.

and these are usually in the plainly visible concentric circles. Because the LLS pathogen produces many spores especially on the lower surfaces, the lesions usually have a raised or tufted appearance. Spots on stem, petiole, and pegs are similar and are irregular or elliptical in shape, when defoliation occurs (Figure 2.4).

Geographical Distribution and Losses

Berkeley (1875) from the United States was the first to describe peanut leaf spot. Later, Woodroof (1933) gave a clear account of the existence of two distinct spots: *ELS* and *LLS*. Presently, the two diseases have been reported throughout the world wherever peanuts are grown. The two diseases,

FIGURE 2.3 LLS of peanuts. Note the carbon black color of the undersurface of the spot.

FIGURE 2.4 Defoliation of the peanut crop at harvest due to LLS.

though occur simultaneously in the same area, differ quite considerably in their relative preponderance from one region to another, depending upon the prevailing weather conditions and type of peanut varieties under cultivation (Kolte 1984, Das and Roy 1995, Sawargaonkar et al. 2010). Reduction in yield is largely due to loss in photosynthetic tissue and defoliation (Naab et al. 2005, Singh et al. 2011a,b). Spots on the pegs also tend to decrease the yield by restricting translocation of food to the seeds. Annual crop losses in the range of 10%–50% or more are common worldwide particularly in the peanut-growing regions in Australia (Kelley et al. 2012), the southeastern United States (Nutter and Shokes 1995), and savanna zones in Nigeria (Izge et al. 2007, Iwo and Olorunju 2009) and Ghana (Nutsugah et al. 2007a,b); in other areas of West Africa (Waliyar 1991, Waliyar et al. 2000), DR Congo and Central Africa (Tshilenge 2010, Tshilenge-Lukanda et al. 2012), sub-Saharan Africa (Hamasselbe et al. 2007), and Malawi (Kisyombe et al. 2001); and in South Asia covering India (Sarkar and Chowdhury 2005, Gopal et al. 2006a), Pakistan (Ijaz et al. 2008), Bangladesh (Hossain et al. 2010), and Nepal (Thakur et al. 2013). The leaf spots have been a serious problem in case of early-season rains and near maturity of the peanut crop in the northern parts of Vietnam, while in the

southern parts of that country, it causes damage from the beginning of the crop (Mehan and Hong 1994). Losses of great magnitude have been documented by comparing yields in plots in which the leaf spot diseases have been controlled with fungicides to those in untreated check plots in which the diseases have been allowed to progress. Plants not treated with fungicides may shed most of their leaves (90% in severe cases) prematurely, causing significant yield losses. Since there are established negative relationships between biomass and disease rating or defoliation and between pod yield and disease rating or defoliation, the visual rating of disease and measured defoliation could be used as a rapid and inexpensive tool to initially assess yield losses caused by the ELS and LLS diseases (Naab et al. 2005). Of several disease assessment methods, the best is proved to be percent main-stem defoliation above the fourth node and percent diseased leaf area estimated from visual leaf spot score (Adomou et al. 2005). Besides the loss in yield of kernels, the value of the hay that may be used as fodder for cattle is also adversely affected. When the leaf spots occur in combination with rust (caused by *Puccinia arachidis*), the losses involved are still more and need more attention (Gughe et al. 1981, Vidyasekaran 1981). The relative importance of each of the leaf spot disease varies from place to place and from season to season, depending on the cropping system and the environmental conditions.

The ELS and LLS lower the peanut haulm yield and quality (Pande et al. 2003). Crude fiber, crude protein, fat, and dry matter content of haulm are significantly lower in severely infected haulm, whereas ash, moisture content, and nitrogen-free extracts get increased with increasing leaf spot severity (Bdliya 2006, 2007).

Pathogens: *C. arachidicola* Hori (perfect stage, *Mycosphaerella arachidis* Deighton) and *P. personata* (Berk. & M.A. Curtis) Arx (perfect stage, *Mycosphaerella berkeleyi* W. Jenkins)

Classification
Kingdom: Fungi
Phylum: Ascomycota
Class: Dothideomycetes
Subclass: Dothideomycetidae
Order: Capnodiales
Family: Mycosphaerellaceae
Genus: *Mycosphaerella*
Species: *arachidis* or *berkeleyi*

C. arachidicola: The perfect stage of this fungus is *M. arachidis*. Its mycelium is septate. In plant tissue, initially, it is intercellular and then it becomes intracellular. Mycelium penetrates directly in plant cell and does not form haustoria. Conidiophores are supported by dark-brown stromata of 25–100 μ in diameter. In the early stages of development, conidiophores mostly are epiphyllous, but in the later stages of disease development, these become amphigenous. Conidiophores arise from stroma and are fasciculate and geniculate, of yellowish-brown color. Conidiophores are usually continuous but may also be with several septations. These measure 20–45 μ × 3–6 μ. Conidiospores are obclavate to clavate, mostly curved, and subhyaline to olivaceous in color. These measure 35–108 μ × 2–5.4 μ, having 4–12 septa with rounded to distinctly truncate base and subacute tips, and germinate by producing germ tubes from different cells. Secondary conidiospores and conidia are seen on slide made from host tissue kept under extremely favorable environmental conditions (Kolte 1984). In case of teleomorphic state, perithecia are scattered mostly along margins of lesions produced by spores of imperfect state. These are amphigenous, somewhat embedded in leaf tissue, erumpent, ovate to nearly globose, and black in color. These are of a size 47.6–84 μ × 44.4–74 μ. Ostiolem is slightly papillate. Asci are cylindrical, club-shaped short stipitate,

fasciculate, aparaphysate, and bitunicate with eight ascospores. Asci measure 27–37 μ × 7–8.4 μ. Ascospores are uniseriate to biseriate in the ascus. These are bicellular with upper cell slightly curved and hyaline. Their sizes range from 7 to 15.4 μ × 3 to 4 μ (Kolte 1984).

P. personata (= *C. personatum*): The mycelium of *P. personata* is septate and exclusively intercellular. Its haustoria puncture into the palisade and mesophyll tissue. Dense, globular, brown to black stromata measuring a diameter of 20–30 μ are produced. Conidiophores are mostly hypophyllous, but sometimes amphigenous. In the later stages of disease development, conidiophores arise in clearly concentric tufts from heavy stromatic base. These are fasciculate, geniculate, and reddish brown in color with mostly hyaline tips and nonseptate or severally septate. Conidiophore sizes range from 24 to 54 μ × 2 to 8.2 μ. Conidia or conidiospores of the fungus are obclavate with attenuated tips and pale-brown dilutely olivaceous color measuring 18–60 μ × 5–11 μ with one to nine septa and bluntly rounded top cells. In *P. personata*, secondary conidia and conidiophores are not reported. The perithecia, asci, and ascospores of teleomorphic stage of *P. personata* only differ from *C. arachidicola* in size. The sizes of perithecia, asci, and ascospores are 84–140 μ × 70–112 μ, 30–40 μ × 4–6 μ, and 19.6 μ × 2.9–3.83 μ, respectively (Kolte 1984). The teleomorphic stage of the LLS pathogen, *M. berkeleyi* Jenkins, is rarely seen on peanuts (Shokes and Culbreath 1997). Molecular variation in the pathogen has been studied (Kumari et al. 2009a,b, 2012).

Epidemiology and Disease Cycle

The fungi that cause ELS and LLS reproduce and infect by means of asexual spores conidia. Both fungi are capable of producing tremendous numbers of conidia on infected plant parts. Conidial production is favored by high humidity. The primary inocula that cause the initial leaf spot infections during the growing season are spores produced on infested peanut residues in the soil. Visible spots develop 10–14 days after infection. New conidia are produced in spots on infected leaves. These conidia will subsequently infect plants and produce secondary infections. Conidia are spread by wind, splashing rain, and insects. Leaf spot can increase rapidly under favorable conditions as several secondary cycles may occur per season. The ELS/LLS stage characterized by the higher level of symptom expression is found not associated with the plant phase of highest emerged leaves, but the disease symptoms reach the peak only after the phase of intense leaf development in peanut crop (Tshilenge-Lukanda et al. 2012).

When enough precipitation of monsoon rains makes a film of water on leaves or a relative humidity more than 90% prevails with a temperature of 20°C–29°C for 6–7 days, peanut crop is severely affected by the ELS and LLS. Disease incidence and severity may vary depending on prevailing climatic conditions. Intermittent rains from flowering to pod development stage of the crop favor the infection and development of leaf spots (Pande et al. 2000). The maximum temperature range of 31°C–35°C and minimum temperature range of 18°C–33°C favor leaf spot outbreak on peanuts. The influence of climatic elements like temperature and relative humidity on the development of ELS and LLS of peanuts has extensively been studied (Dubey 2005, Kadam et al. 2008a, Ijaz et al. 2011). Abundant peanut residue in fields where peanuts are cropped continuously often results in early and rapid development of leaf spot. The first appearance of leaf spot and its continuous progress throughout the growing season are heavily dependent upon weather conditions. Environmental conditions required for both types of leaf spots are warm temperatures and long periods of high humidity or leaf wetness (Pande et al. 2004). Wet periods of sufficient duration to support infection usually consist of dew periods at night or extended rainy periods. When adequate moisture is present, leaf spot infections may occur in a relatively short period when temperatures are warm, but a longer wet period is required when temperatures are cool. For these reasons, potential for damage from leaf spot is greater where levels of humidity and rainfall are high (Muhammad et al. 2008). Frequent irrigation with small amounts of water can also create prolonged periods of high humidity and leaf wetness favorable for infection. A model has been developed by taking into consideration the relative humidity of more than 95% and the minimum temperature of 22°C and maximum

30°C. The model is used or compared with a calendar-based schedule in the United States (Smith 1986), Argentina (Pezzopane et al. 1998), and Brazil (Moraes et al. 2002). Based on prediction of favorable weather conditions and application of high-resolution Weather Research and Forecasting model, the model for ELS control in peanut crop has been developed. The short-term prediction of weather parameters and their use in management of the leaf spot diseases is a viable and promising technique, which could help the growers make accurate management decisions through optimum timing of fungicide applications (Olatinwo et al. 2012).

There appears to be a reduction in lesion size and intensity of sporulation (inoculum potential) of ELS pathogen due to infection (Subhalakshmi and Chowdhury 2008).

Disease Management

Host Plant Resistance

In Cultivated Arachis hypogaea

Host resistance to both ELS and LLS diseases is reported in *A. hypogaea*, and it is quite variable (Varman 2001, Izge et al. 2007, Padi 2008, Giri et al. 2009, Tallury et al. 2009, Dolma et al. 2010, Visnuvardan et al.2011). Generally, late-maturing alternately branched cultivars are either runner or spreading bunch type with dark-green foliage possessing very high degree of resistance to the leaf spot pathogens, whereas early forms with sequential branching are usually erect bunch type with light-green foliage showing a high degree of susceptibility to the disease (Kolte 1984). Sequentially, branched early cultivars are more susceptible to *Cercospora*, possibly because there are a greater proportion of stomata of penetrable size (more than 13.4 μ) on the upper leaf surface. In alternately branched forms, there is thicker palisade tissue in the leaf, which may partly explain the slower rate of lesion growth on them as well as account for their dark-green foliage (Hemingway 1957, Gibbons and Bailey 1967). Because of the reduced infection rate characterized in part due to lower infection frequencies, smaller lesions, lower sporulation index, longer latent periods, and lesser leaf area damage and disease score in resistant varieties, the resistance in these varieties is considered to be a partial one (Hossain and Ilag 2000, Dwivedi et al. 2002, Pande et al. 2002, Cantonwine et al. 2008b). Selection based on components of resistance to LSS may not lead to plants with higher retained green leaf area. The remaining green leaf area on the plant should, therefore, be the major selection criteria for resistance to LLS in breeding programs (Dwivedi et al. 2002). Improving levels of resistance along with foliar application of fungicides to manage the disease in locally adapted varieties would substantially increase peanut yields in developing countries (Waliyar et al. 1993, 1995, 1998). Currently, there are only a few varieties possessing tolerance to foliar diseases. However, one to two sprays depending upon the suitable time of application increase the pod yield significantly (Waliyar et al. 1998). A Bolivian land race cultivar *Bayo Grande* and several other Bolivian-derived genotypes show promise for use in a reduced fungicide and/or conservative tillage system with a potential to lessen fungicides compared to standard production practices (Gremillion et al. 2011a). Some of the most promising genotypes resistant or moderately resistant (tolerant) to either or both of the leaf spots are given in Table 2.1. High heritability coupled with high genetic variation is usually noticed for LLS and rust and pod yield indicating that additive gene effects are functional for these characters (Venkataravana and Injeti 2008).

Two genotypes of peanut, namely, cv 850 and cv 909, are reported to be resistant to *P. personata* (*C. personatum*) and show symptoms similar to hypersensitive response (HR) lesions, and the production of HR lesions is due to a novel *O′*-methyltransferase gene (Nobile et al. 2008). Higher amount and greater accumulation rate of total free phenol and stilbene phytoalexin production are the most possible biochemical mechanisms of resistance in peanuts against the two leaf spot pathogens (Motagi et al. 2004, Sobolev et al. 2007, Bhaskar and Parakhia 2010). The isoenzyme variability, for example, the presence of phosphatase band and two esterase bands, and stilbene phytoalexins are found to be specific to resistant cultivars, which can possibly be used as biochemical

TABLE 2.1
Peanut Genotypes Resistant (R) or Moderately Resistant (MR) to ELS and/or LLS as Reported from Different Countries in the World

Genotype	Country	R/MR	Reference(s)
ICGV 98369	South Africa	R	Mathews et al. (2007)
ICGV SM99529	Malawi		
ICGV 91225	Sub-Saharan Africa	R to ELS	Hamasselbe et al. (2007)
Samnut II	Sub-Saharan Africa	R to LLS	
Golden Mutant 96 C	Pakistan	R to CLS	Naeem-ud-Din et al. (2009)
INS-1-2006, AIS-2006-11	India	R to LLS	Sheela (2008)
ICGV-IS-96805		R to ELS + LLS	Iwo and Olorunju (2009)
C689-2, Georgia-01R, C12-3-114-58, C11-154-6, Tifguard, and Georganic	Southern United States	R to ELS	Li et al. (2012)
Charmwon, HyQ(CG)S-10 (*A. hypogaea* ssp. *fastigiata*)	Korea	R to ELS	Pae et al. (2008)
ICGV 05033, ICGV 03037, ICGV 05099, ICGX020063-P11, ICGV 04093, ICGV 03016, ICGV 04071, ICGV 86031, ICGV 03157, PAFRGVT60	India	R to LLS + rust	Venkataravana and Injeti (2008)
ICGV 99057, ICGV 00228, ICGV 99068, ICGV 99057, ICGV 00169	India	MR or R to LLS	Venkataravana et al. (2008)
259/88, 262/88	Bangladesh	High R to LLS + ELS	Hossain et al. (2007)
269/89	Bangladesh	MR to ELS	Hossain et al. (2007)
DP-1, Georganic	United States	R to ELS + LLS	Cantonwine et al. (2008)
Georgia-01R, Georgia-05E	United States	R to ELS + LLS + TSW	Branch and Culbreath (2008)
CV100, PI648033	United States	R to ELS + LLS + TSW	Holbrook and Culbreath (2008)
ICGV-IS-96808	Nigeria	R to ELS/LLS	Izge et al. (2007)
CV 850, CV 909		High R to LLS	Nobile et al. (2008)
PI 390590	India	R to LLS	Suryawanshi et al. (2006)
R8972	India	R to LLS + rust	Gopal et al. (2006b)
ICGV 92099, ICGV 90084	Ghana	R to ELS + LLS	Frimpong et al. (2006)
TFDRG1, TFDRG2, TFDRG3, TFDRG4, TFDRG5, VG9514	India	R to LLS + rust	Badigannavar et al. (2005)
N96076L (GP-125, PI641950)	United States	Multiple disease resistance including LLS	Isleib et al. (2006)
Nkatiesari	Ghana	R to ELS + LLS	Padi et al. (2006)
SP 8638	Korea	R to LLS	Pae et al. (2005)
Huayu 22	China	R to LLS + web blotch	Chen et al. (2005)
Kokwang	Korea	R to ELS	Park et al. (2004)
Jakwang	Korea	R to ELS	Pae et al. (2004)
FDRS-10	India	R to LLS	Jyosthna et al. (2004)
Zhonghua 9	Hubei Province of China	R to LLS + rust	Liao et al. (2004)
GPBD-4	India	R to LLS + rust	Gowda et al. (2002a)
Mutant 28-2	India	R to LLS	Gowda et al. (2002b)
C-99R	United States	R to LLS + stem rot + TSW	Gorbet and Shokes (2002a)
Florida MDR 98	United States	MR-R to LLS + stem rot + TSW	Gorbet and Shokes (2002b)

(*Continued*)

TABLE 2.1 (*Continued*)
Peanut Genotypes Resistant (R) or Moderately Resistant (MR) to ELS and/or LLS as Reported from Different Countries in the World

Genotype	Country	R/MR	Reference(s)
ICGV 92267	ICRISAT, India	MR to LLS + rust	Upadhyaya et al. (2002)
Georgia-01R	United States	R to ELS + LLS + rust	Branch (2002)
GP-NCWS11, GP-NCWS12, GP-NCWS-13, GP-NCWS14, GP-NCWS15	United States	R to ELS + LLS	Stalker et al. (2002)
VRI Gn 5	India	R to LLS + rust	Vindhiyavarman and Mohammed (2001)
ICGV 92080, ICGV 92093	India	R to LLS	Mohammed et al. (2001)
Huayu 17	Shandong Province, China	High R to LLS	Yu et al. (2000)

ELS, early leaf spot; LLS, late leaf spot; TSW, tomato spotted wilt.

markers in the identification of the leaf spot pathogen–resistant cultures in peanuts (Jyosthana et al. 2004, Sobolev et al. 2007). In order to be effective in improving the efficiency of identifying genes of interest of ELS/LLS resistance in the entire germplasm collection, core collection as developed in the United States could be of immense value (Holbrook and Dong 2005, Gremillion et al. 2011b).

As resistance to ELS and LLS is inherited independently (Higgins 1935) and in some genotypes, duplicate complementary recessive genes are reported to control the LLS resistance (Motagi et al. 2000), and these diseases vary in their relative preponderance in different regions based on prevalent cropping system and spectrum of locally prevalent races, a separate breeding program may have to be used, if survey work reveals that only one of these is only important in a particular area.

In Wild Arachis *Species*

The cultivated peanut (*A. hypogaea* L.) is an allotetraploid with an AABB genome and low genetic diversity. Because of its limited genetic diversity, this species lacks resistance to a number of important pests and diseases. In contrast, wild species of *Arachis* are genetically diverse and are rich sources of disease-resistant genes (Varman et al. 2000, Fávero et al. 2009). The genus *Arachis* is native to South America and consists of 22 described species and possibly more than 40 undescribed. Collections are maintained in Brazil, the United States, and India. Many of these accessions have been screened for resistance to pathogens and insect pests, and it is demonstrated that sources of resistance are available in the wild species belonging to the taxonomic section *Arachis* with either A or B (or *non-A*) genomes and these can be used for introgression of resistance genes against two leaf spot diseases and other diseases (Mallikarjuna et al. 2004, 2012, Yadav et al. 2007, Fávero et al. 2009). International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) is currently using diploid species of section *Arachis*, which are cross compatible with the tetraploid-cultivated peanut to transfer useful genes through interspecific hybridization. Thus, stable, tetraploid interspecific hybrids with resistance to the LLS and rust showing high yield potential have been developed (Yadav et al. 2007). Twenty-nine percent (29%) of interspecific derivatives from the cross *A. hypogaea* ($2n = 40$) × *A. kempff-mercado* ($2n = 20$) at the BC2 F2 generation have been established to show resistance to both ELS and LLS (Mallikarjuna et al. 2004). Hybrids formed between cultivated and wild species are generally alternately branched, giving low yield, but these may provide the basis for selection in a breeding program.

Resistance to *P. personata* is found in accessions of different species of *Arachis*, from A genome species, namely, *A. stenosperma*, *A. kuhlmannii*, *A. helodes*, *A. simpsonii*, *A. diogoi*, *A. aff. diogoi*, *A. microsperma*, *A. linearifolia*, and *A. cardenasii*, and *non-A* genome species,

namely, *A. cruziana*, *A. hoehnei*, *A. magna*, *A. valida*, *A. batizocoi*, and *A. williamsii*. Differential gene expression in *A. diogoi* upon interaction with *P. personata* reveals that the pathogen induces cyclophilin-like proteins (Kumar and Kirti 2011). Similarly, resistance to *C. arachidicola* is found in accessions of different species of *Arachis*, from A genome species, namely, *A. kuhlmannii*, *A. helodes*, *A. cardenasii*, *A. kempff-mercado*, *A. linearifolia*, and *A. stenosperma*, and from *non-A* genome species, namely, *A. hoehnei*, *A. magna*, and *A. batizocoi* (Fávero et al. 2009). The resistance to LLS and rust (*P. arachidis*) studied by Pande and Rao (2001) in the 74 accessions of wild species of *Arachis* revealed the accession *KG3006* of *A. hoehnei* immune to either of the two leaf spot diseases. Thus, there is resistance to *P. personata* and *C. arachidicola* in many accessions of wild species, and these accessions may be different among accessions of the same species. Interestingly, molecular markers for resistance genes (R genes) that encode a putative nucleotide-binding site (NBS) domain and a leucine-rich repeat (NBS-LRR genes) have been developed to generate resistance gene analogues (RGAs) in wild *Arachis* spp. in Brazil (Guimarães et al. 2005, Garcia et al. 2006). Inclusion of these RGAs in *Arachis* genetic map will be of paramount importance in breeding for disease resistance not only to ELS and LLS diseases but also to rust and other economically important diseases of peanuts.

Molecular Breeding and Transgenic Peanuts for ELS and LLS Resistance

Genetic transformation has launched a new era in peanut breeding and germplasm creativity through transformational methods including *Agrobacterium tumefaciens*–mediated, particle bombardment, and nontissue culture techniques (Wei et al. 2008). Comparison of molecular profile among peanut cultivars and breeding lines with differential reaction against LLS and other diseases is facilitated by the use of random amplified polymorphic DNA (RAPD) and intersimple sequence repeat (ISSR) markers (Dwivedi and Gurtu 2002, Dwivedi et al. 2003, Mondal et al. 2008a, 2009, Khedikar et al. 2010). For example, transgenic peanut plants constitutively expressing the mustard defensin gene have been generated by cloning the complete cDNA containing an open reading frame (ORF) of 243 bp of a defensin of mustard (defensins are small positively charged antimicrobial peptides, 5 kDa in size, and show potent antifungal activity). Such transgenic peanut plants show enhanced resistance against both the leaf spot diseases (Anuradha et al. 2008). The ISSR marker UBC810 (540) has been found associated with LLS + rust resistance but UBC810 (500) only with LLS resistance (Mondal et al. 2009). Leal-Bertioli et al. (2009) have identified candidate genome regions that control disease resistance and placed candidate disease-resistant genes and quantitative trait loci (QTLs) against LLS disease on the genetic map of the A genome of *Arachis*, which is based on microsatellite markers and legume anchor markers enabling mapping of a total of 34 sequence confirmed candidate disease-resistant genes and 5 QTLs. Among the polymorphic sclerotium stem rot (SSR) markers developed through crossing LLS-susceptible cultivar TMV-2 and LLS-resistant genotype COG-0437, the primer PM 384(100) has association with resistance and could therefore be utilized in the marker-assisted breeding program over a wide range of genetic background (Shoba et al. 2012). A double-gene construct with *Solanum nigrum* osmotin-like protein (SniOLP) and *Raphanus sativus* antifungal protein 2 (Rs-AF2) genes under separate constitutive 35S promoters has been developed to transform peanut plants. Such transgenic peanut plants expressing the SniOLP and Rs-AF2 genes show enhanced resistance to LLS based on reduction of number and size of lesions on leaves and delay of the onset of the LLS (Gowda et al. 2010, Vasavirama and Kirti 2012). The molecular diversity analysis using SSR reveals high level of genetic polymorphism for resistance to LLS and rust diseases, which provides valuable information for peanut breeders designing strategies for incorporating and pyramiding LLS and rust resistances and creating inbred line populations to map these traits (Mondal et al. 2005, Mace et al. 2006). Similarly, RAPD assays using 10 oligonucleotide primers have revealed existence of DNA-level variation within the LLS and rust-resistant genotypes. The susceptible lines can be clustered distinctly away from the resistant group, and clustering of genotypes based on phenotyping of LLS and rust can serve as basis for tagging resistant genes (Reddy et al. 2004). Marker-assisted backcross breeding should be able to minimize the linkage drag as it greatly facilitates monitoring of

introgressed chromosome segments carrying disease-resistant genes from wild species to cultivated peanuts. Transgenic peanuts with resistance to biotic stresses as ELS and LLS and others have been reported to be produced and in various stages of characterization under containment and/or controlled field conditions (Dwivedi et al. 2003, Luo et al. 2005). For example, fertile transgenic plants of peanut cv TMV-2 expressing tobacco (*Nicotiana* spp.) chitinase and neomycin phosphotransferase (nptII) genes have been generated using *A. tumefaciens*–modified transformation system, and peanut plants containing transgenically increased activity of chitinase are resistant to attack by *C. arachidicola* to different degrees (Rohini and Rao 2001). Similarly, transgenic peanut plants possessing rice chitinase gene expressing resistance to ELS pathogen have been obtained through *Agrobacterium* mediation (Iqbal et al. 2012). Such strategies are of great potential for the control of the leaf spot diseases.

Chemical Control

Fungicides

The economic benefit of using fungicides to control the leaf spot diseases depends on the climatic conditions, the variety of peanut grown, the relative importance of the fungus species affecting the crop, and the general pattern of farming in the area concerned (Johnson et al. 2007a, Hoque et al. 2008, Naab et al. 2009, Muhammad and Bdliya 2011, Wann et al. 2011). Better results with increase in yield have been reported on early-maturing sequentially branched varieties compared with alternately branched late varieties. The reason is that early-maturing sequential type cannot produce branches and leaves at the end of growing season; consequently, all the assimilates produced at this stage are available for the growth of the nuts so that when the disease is controlled, there is considerable increase in the kernel yield. Conversely, the alternate forms continue to produce many new branches and leaves even up to the end of the season, and therefore, the effect on kernel yield as a result of disease control is proportionately less. The time of the first application may be dependent on the susceptibility of the variety. In long-duration varieties like 28-206 and 47-16, it is better to apply fungicides at the later stages of growth. Both of these lines produced 3.16 and 2.94 tons/ha pod yield when fungicide is applied at 70 days after sowing (DAS) (Waliyar et al. 1993, 1995, 1998). A new Australian cultivar Sutherland has significantly higher resistance to LLS and would need reduced or fewer number of fungicide spray in the management of the disease (Kelley et al. 2012). Research results obtained by Carley et al. (2009) reinforce the value of controlling ELS and LLS (and web blotch) with timely fungicide applications and importance of digging at optimum pod maturation of peanuts contradicting the general recommendation or belief that the percentage of canopy defoliation justifies early digging to prevent the yield loss. Similarly, such experimental evidence has been obtained in relation to fungicide application in refinement harvest timing guidelines based on the distribution of pod maturity as defined by mesocarp color categories (Chapin and Thomas 2005). An interaction of number and properly timed fungicide sprays for the control of ELS and LLS can result in substantial monetary gains for peanut farmers in West Africa (Waliyar et al. 2000). Several fungicides that have been effective in the control of the ELS and LLS are given in the following paragraphs.

During the initial stages of the development of fungicides, elemental sulfur (sulfur dust, wettable sulfur) and copper compounds (Bordeaux mixture, copper oxide, copper oxychloride) were in use for the control of the leaf spot diseases of peanuts (Kolte 1984). Later as many modern compounds like dithiocarbamates (maneb, zineb, mancozeb) and chlorothalonil became available, these then largely replaced copper-based compounds as protective fungicides. However, currently, both copper-based and elemental sulfur fungicides are considered to be organically acceptable fungicides on cultivars with partial resistance to one or both the ELS and LLS pathogens (Cantonwine et al. 2006, 2007a, 2008). Chlorothalonil spray at 0.2% has been found better than mancozeb and copper oxychloride, which is proved to be an economical fungicide (Culbreath et al. 1992a,b). Organotin compounds, though performing better than dithiocarbamates, could not become a commercially successful class of fungicides for the control of ELS and LLS. In 1968, a systemic fungicide, benomyl, was introduced and registered for better and efficient control of both the leaf spots through its

protective and curative effects. Consequently, similar other two systemic fungicides, for example, carbendazim (0.05%) and thiophanate methyl (0.05%), found a commercially successful use all over the world for the control of the leaf spot diseases (Kolte 1984, Noriega-Cantú et al. 2000, Biswas and Singh 2005). All these systemic fungicides are reported to give better control of the leaf spots than mancozeb or copper oxychloride (Vidyasekaran 1981, Bolonhezi et al. 2004, Biswas and Singh 2005). However, curative action of systemic fungicide, that is, carbendazim when combined with a dithiocarbamate (mancozeb), has given good control of the leaf spots at longer intervals at various locations in the peanut-growing regions in different countries (Joshi et al. 2000, Srinivas et al. 2002, Biswas and Singh 2005, Satish et al. 2007, Johnson et al. 2007a, Lokesh et al. 2008). The first spray of mancozeb on initiation of the leaf spots followed by the carbendazim spray 10 days after mancozeb and again the spray of mancozeb 10 days after carbendazim give maximum control of the leaf spot disease, avoiding the possibility of development of fungicide resistance (Sawant 2000). The continued use of sprays of benomyl or benomyl-like compound carbendazim resulted in the development of resistant strains of *C. arachidicola* and *P. personata* in the mid-1970s. Consequently, use of benomyl on peanuts in the southeastern United States was discontinued, but chlorothalonil still proved to be an effective standard fungicide and is used extensively for the control of ELS and LLS diseases not only in the United States but also in Malawi (Kisyombe et al. 2001, Culbreath et al. 2002). Both mixtures and alternate applications of chlorothalonil and benomyl are effective for the management of the leaf spot in fields where benomyl alone did not provide season-long leaf spot control due to fungicide resistance (Culbreath et al. 2002). Additional options became available for the leaf spot control in the 1990s with registration of the demethylation inhibitors (DMIs), that is, sterol (ergosterol) biosynthesis–inhibitor (SBI) fungicides, which are triazole fungicides, namely, difenoconazole, propiconazole, hexaconazole, cyproconazole, tebuconazole, and epoxiconazole for use on peanut crops. These fungicides improved the management of one or both leaf spot diseases compared to chlorothalonil alone (Moraes et al. 2001). They control not only the leaf spots but peanut rust also (Culbreath et al. 1992b, Dahmen and Staub 1992, Jadeja et al. 1999). Similarly, quinol oxidation (Qo site) inhibitor (QoI) fungicides, also referred to as strobilurin fungicides, first launched in 1996, are very effective for the control of the leaf spots. The three most commonly used strobilurin fungicides used for the ELS and LLS on peanuts are azoxystrobin, pyraclostrobin, and trifloxystrobin. Recently, the new pyrazole carboxamide fungicide penthiopyrad at 0.20 kg ai/ha is reported to have excellent potential for the management of the LLS and may complement current SBI and QoI fungicides (Culbreath et al. 2009). Prothioconazole applied alone (0.18–0.20 kg/ha) or in combination, that is, 0.085 kg ai/ha of prothioconazole + 0.17 kg ai/ha of tebuconazole or mixtures of prothioconazole at 0.063 kg ai/ha with trifloxystrobin at 0.063 kg ai/ha, gives similar or better leaf spot control than chlorothalonil (Culbreath et al. 2008). Pyraclostrobin (Chapin and Thomas 2005, Culbreath et al. 2006), azoxystrobin (Bowen et al. 2006, Hagan et al. 2006), tebuconazole (Hossain et al. 2005, Culbreath et al. 2006, Nutsugah et al. 2007), and a new systemic fungicide Nativo (Khan et al. 2014) have been found as effective fungicides for the control of leaf spot diseases of peanuts in different countries around the world. Weather-based fungicide advisory sprays of these fungicides in the United States reflect the improved disease control, consequently improving the yield of the peanut crop (Grichar et al. 2005). The cost–benefit analysis of fungicidal control of the leaf spots of peanuts in the Sudan savanna region of Nigeria revealed positive returns per hectare from the use of fungicides. Application of Bentex T (benomyl + thiram), for instance, has been found to give 78.13% seed yield increase over untreated plants, which could be translated into a mean net profit in the range of U.S. $387–$909/ha depending on the number of effective efficacies of sprays (Bdliya and Gwio-Kura 2007a,b). Similarly, high incremental cost–benefit ratio has been obtained with hexaconazole (1:4.72) followed by propiconazole (1:2.05), difenoconazole (1:1.29), and chlorothalonil (1:1.13) in the control of leaf spot diseases in India (Gururaj et al. 2002, 2005a). In some other studies, however, the highest cost–benefit ratios (1:6.9–1:675) with 0.1% difenoconazole spraying at 30, 50, and 70 DAS (Gopal et al. 2003) and with hexaconazole at 60 and 75 DAS (Johnson and Subramanyam 2003) and effective control with single spray (Chandra et al. 2007) have been obtained in India. Rainfall-based advisories

for the chemical control of LLS in Brazil reveal that tebuconazole sprays at an interval of 15 days show higher efficiency in controlling the LLS in comparison to a scheme of control with chlorothalonil sprayings at five fixed dates (the first at 49–50 days after planting and the following ones at intervals of 14–15 days) besides promoting an average reduction of one to three sprayings (Moraes et al. 2002). When the proportion of ELS- and LLS-affected leaves exceeds 10% and the wet index total exceeds the threshold value (<2.3), the first application of fungicide is advised, followed by at least two successive sprays at a 14-day interval (Butler et al. 2000).

Nonconventional Chemicals

Among nonconventional chemicals using inorganic and metal salts, catechol at 10(-3M) (Maiti et al. 2005), nickel chloride, and cupric sulfate at 10(-3M) (Kishore et al. 2001b) have been found significantly effective in controlling the ELS and LLS infection through induction of host resistance in peanuts. Resistance interaction is correlated with early and rapid induction of phenylalanine ammonia lyase (PAL) enzyme regulating the biosynthesis of antifungal phytoalexin medicarpin (Kale and Choudhary 2001). Similarly, the effect of salicylic acid (SA) on induction of resistance in peanuts against LLS through foliar application of SA at a concentration of 1 mM is observed to be due to the enhanced activity of PAL, chitinase, β-1,3-glucanase, peroxidase (PO), and polyphenol oxidase (PPO) (catechol oxidase) enzymes and in total phenolic contents in peanuts after application of SA and inoculation with *P. personata* (Meena et al. 2001a).

Cultural Control

Since the ELS and LLS pathogens have a very restricted host range and largely to *Arachis* species, effective crop rotation of a 2–3-year duration of peanuts with nonsusceptible crops, such as cotton or corn or soybean or maize alternating with peanut or not following peanut with peanut, has been found useful in delaying the initial infections by both leaf spot fungi and reducing the incidence of the disease early in the season by 88%–93% (Kucharek 1975). This level of reduction permits growers to take some of the *pressure off* of a fungicide spray and delay the first fungicide application where crop rotation is followed requiring fewer or at least less expensive fungicide application. Though there can be regional differences based on different cropping schemes, rotation into nonhost crops is essential to sustainable, long-term production of peanuts. About 72%–86% more yield has been reported by following crop rotation. Eliminating volunteer peanut plants in the fields that follow peanut main or forage crops immediately after harvest of the peanut is a critical component of successful crop rotation program. Keeping the peanut crop free from weed (weeding once at either 4 or 6 weeks after planting) is also helpful in minimizing the severity of the leaf spots (Abudulai et al. 2007) in Ghana. Clewis et al. (2001) from the state of North Carolina in the United States provided evidence as to how high density of common ragweed (*Ambrosia artemisiifolia*) in peanut fields increases the incidence and severity of LLS on the peanut crop.

The relationship between disease severity and yield increase at different plant spacings is reported. Lower incidence and severity of the leaf spots is reported at a 50 × 30 cm intrarow spacing in comparison to 50 × 20 cm in Nigeria (Garba et al. 2005). Reduced tillage, the strip-tilled peanut crop with the presence of cover crop residue at the soil surface of strip-tilled fields in the United States, is less severely affected by ELS epidemics than conventionally tilled fields. The strip tillage delays ELS epidemics due to a fewer initial infections, most likely due to cover crop residue interfering with the dispersal of primary inoculum from overwintering stroma in the soil to the peanut plant (Cantonwine et al. 2007b). Consequently, the number of fungicide applications, if any, could be reduced without compromising the control of ELS when reduced tillage is used especially when combined with moderately resistant cultivars (Monfort et al. 2004). Intercropping of millet (Bdliya and Muhammad 2006) and that of maize (Ihejirika 2007) in Nigeria and of pearl millet in India (Srinivas et al. 2002) with peanut show significantly lower ELS/LLS severity on peanut in comparison with sole peanut crop without adversely affecting the yield of inter crops. NPK fertilizer rates and peanut plant population per hectare significantly influence the severity

of ELS, the least severity of the ELS being at the density of 250,000 peanut plants/ha in comparison to 1,000,000 plants/ha (Ihejirika et al. 2006a). Among several methods of irrigation studied (Jordan and Johnson 2007, Woodward et al. 2008), subsurface drip irrigation is useful in avoiding the increase and spread of the ELS compared with overhead sprinkler irrigation (Lanier et al. 2004).

Depending on the more frequent occurrence of the disease in a particular area, a choice of suitable planting dates (either early or late) and a choice of using early- or late-maturing variety may be important in reducing the severity of the disease (Reddy and Reddy 2000, Naidu and Vasanthi 2002). The least incidence and severity of the leaf spots is noted when the crop is planted in the first week of May in the state of Gujarat in India (Hazarika et al. 2000). Planting date and maturity of the variety then also significantly influence the effectiveness of fungicide spray application. For example, fungicide spraying in early-sown crop in the state of Chhattisgarh in India is reported to be uneconomical because reduction in pod yield due to the leaf spots in the early-sown crop is not significant. In contrast, two sprays (or even only one spray) are economical for late-sown crops under similar conditions (Tiwari et al. 2005). Interestingly, long-duration (120 days) variety (F-mix) of peanuts when sown early and treated with fungicide for the leaf spot control under optimum and timely crop management practices is reported to have produced greater yield than short-duration (90 days) variety (Chinese) under both with and without fungicide-treated environments and more than three- to fourfold increase over the average peanut yields in Ghana (Naab et al. 2005). Deep burying of crop residues in the soil by mold board plow and destruction of crop residues by burning have been recommended as additional aids for the control of the leaf spot diseases of peanuts.

Biological Control

Biological control of leaf spot diseases using antagonistic fungi or bacteria may be an alternative approach to use of chemical fungicides. This appears to be desirable in view of the development of resistance against the most effective systemic chemicals and side effects from frequent use of chemicals, as is evident from the increase in population of phylloplane mycoflora and foliar mites as a result of the spray of chemicals. In India, a mycoparasite, *Hansfordia pulvinata*, has been found parasitizing *C. arachidicola* and *C. personatum* (Krishna and Singh 1980, Siddaramaiah and Jayaramaiah 1981). *Hansfordia* sp. (exact species not mentioned) has also been reported from the United States to be parasitizing only *C. personatum* and not *C. arachidicola* (Taber and Pettit 1981). It is observed that the leaf spots, which are parasitized by the aforementioned fungus, do not show conidiophores and conidia of the respective causal fungus species. Among several isolates of *Pseudomonas fluorescens* screened for their efficacy for the control of the LLS, the *strain Pf1* has been found to be effective in reducing the LLS disease index through seed treatment (10 g/kg seed) combined with soil application of *P. fluorescens* strain Pf1 (2.5 kg/ha at 30 and 45 DAS) or foliar spray application of talc-based powder formulation (1–2.5 kg/ha) of the strain Pf1 (Meena et al. 2000, 2002, 2006, Zhang et al. 2001, Johnson and Subramanyam 2009, Meena 2010, Meena and Marimuthu 2012). Following the seed treatment, the antagonist colonizes soil in the peanut rhizosphere. All such treatments also show increase in plant height with enhanced yield of pod possibly due to the production of IAA and induction of *P. fluorescens*–mediated systemic resistance against the leaf spot pathogens. *P. fluorescens*–treated peanut plants show increase in the activity of PAL, phenolic content, and lytic enzymes. Chitin-supplemented application of antifungal and chitinolytic bacteria *Bacillus circulans* GRS 243 and *Serratia marcescens* GPS 5 effectively results in the control of LLS (Kishore et al. 2005a) through enhanced activity of four defense-related enzymes such as chitinase, β-1,3-glucanase, PO, and PAL (Kishore et al. 2005b). The nonchitinolytic *Pseudomonas aeruginosa* GSE 18 (Kishore et al. 2005b) and chlorothalonil-tolerant *P. aeruginosa* have also given effective control of the LLS (Kishore et al. 2005b). Kondreddy and Podile (2012) reported a new integrated approach, where both direct antagonism and induced resistance got combined to reduce the incidence of the LLS in peanuts. Chlorothalonil-tolerant chitinolytic bacterium

has been genetically engineered to secrete elicitor protein harpin Pss of *Pseudomonas syringae* pv. *syringae* for the dual benefit of growth promotion of peanut plants and the control of LLS.

Mycorrhizal (*Glomus* sp. and *Gigaspora* sp.) symbiosis with peanut roots increases the resistance of plants to leaf spot pathogens reducing the severity of the leaf spot diseases by 54% (Zachée et al. 2008).

Effect of Plant Extracts

Seed extract of *A. indica* (Srinivas et al. 2000, Alabi and Olorunju 2004, Nandgopal and Ghewande 2004, Ambang et al. 2007, 2011, Badliya and Alkali 2010a,b); aqueous leaf extracts of *A. indica* (Aage et al. 2003, Ihejirika et al. 2006b); Hemi Fern, that is, *Hemionitis arifolia* (Sahayaraj et al. 2009), *Prosopis juliflora* at 2% (Kishore and Pande 2005b), *Polyalthia longifolia* at 10% (Adiver 2004), *Lawsonia inermis* at 5%, and *Datura metel* at 2% (Kishore et al. 2001a, 2002, Kishore and Pande 2005a); seed extract of *Thevetia peruviana* (Ambang et al. 2007, 2011); and *Mahogany* bark extract (Salaudeen and Salako 2009), when sprayed on peanut plants, give reductions in the leaf spot disease index on peanuts. Aqueous leaf extracts of *A. indica* and that of *D. metel* and neem (*A. indica*) seed could be of potential economical and eco-friendly alternative usages for the control of the leaf spots particularly in areas where farmers cannot afford to use fungicides particularly in the Sudan savanna region of Nigeria.

RUST DISEASE OF PEANUTS

Symptoms

Orange-red to chestnut-brown elliptical raised uredo pustules appear on the abaxial surface of the leaves (Figure 2.5). The pustules are 0.3–2.00 mm in diameter and usually surrounded by a yellow halo. The adaxial surface of the leaf might present a gray appearance due to the formation of flecks that correspond to the position of the uredo pustules below. The uredo pustules are either isolated or in groups; they are formed subepidermally on compact stomata but soon burst through the epidermis and become exposed. Consequently, a reddish-brown mass of spores becomes visible on the

FIGURE 2.5 Peanut rust symptoms on the leaflet. Note the numerous small-sized uredo pustules.

FIGURE 2.6 Field view of peanut rust–affected crop.

surface of the leaves. The uredo pustules are also formed on the adaxial surface at advanced stages of infection. They may also be formed on stipules, petioles, and stem. As the infection advances, the pustules turn dark brown and frequently coalesce to cover larger areas. Eventually, the leaflets may curl and drop off resulting in defoliation. Severely affected plants appear as light-brown patches in normal green plants in the field and can be easily seen from a longer distance (Figure 2.6). Pods of severely affected plants are low in number and mature 2–3 weeks early. The seeds of such plants remain small in size. The sequence of the development of symptoms has been studied on artificially inoculated plants (Mallaiah and Rao 1979).

Geographical Distribution and Losses

Peanut rust has been known since 1884 from specimens on cultivated peanut plants collected in Caaguazu, Paraguay (Spegazzini 1884). It has now become a disease of major economic importance in almost all peanut-growing areas in North, Central, and South America, the Caribbean Islands, the West Indies, Asian and West and East African countries, and Australia (Kolte 1984, Subrahmanyam et al. 1985). In recent years, peanut rust has spread to and become established in South Africa (Mathews et al. 2007). In the United States, rust causes considerable economic losses in South Texas, and earlier rust, as such, had not been considered a serious problem in peanut production (Hammons 1977), but in recent years, the disease has been recorded to occur and impact yields in the southeastern United States (Gremillion 2007).

Since the appearance of the rust coincides with the appearance of ELS and LLS, several workers have adopted the method of estimating the loss in yield due to the disease by controlling the leaf spots by spraying plants first with benomyl or carbendazim and then superimposing this treatment with another fungicide (tridemorph or chlorothalonil or hexaconazole) effective against the rust. Thus, information on loss in yield due to rust or the leaf spots alone or due to both has been obtained. The loss in pod yield due to rust alone has been reported in the range of 14%–70%, depending on the variety and geographical region and climatic conditions (Kolte 1984, Gururaj and Kulkarni 2006). It is reported that early establishment of the disease in Australia may advance

harvesting of the crop by up to 28 days. Significant yield loss to the extent of 100% is observed in most rust-affected peanut crops in Northern Australia in every season (Middleton and Shorter 1987). A serious outbreak of peanut rust appeared in 1973 in northern Territory in Australia (O'Brien 1977) and in the Maharashtra State of India in 1976–1977, and the crop yield then declined by 35% (Mayee 2009, Tashildar et al. 2012).

In Nicaragua, the commercial crop cost for peanuts has increased by 48% because of measures to control peanut rust and leaf spots in nonrotated fields. In the P.R. of China, losses due to rust are estimated to be 49%, 41%, 31%, and 18% at flowering, pegging, pre-pod-forming, and mid-pod-forming stages, respectively (Zhou et al. 1980).

In India, the combined infections of rust and leaf spots cause losses to the extent of 29%–70% in pod yield and 27% in kernel weight depending on the variety (Ghuge et al. 1981, Tashildar et al. 2012). In addition to direct yield losses, rust can lower down seed quality by reducing seed size and seed viability and oil content (Subrahmanyam et al. 1991, 1997). The loss in oil content due to rust infection alone has been estimated to be about 7%–10% (Kenjale et al. 1981).

Pathogen: *P. arachidis* Speg.

Classification
Kingdom: Fungi
Phylum: Asidiomycota
Class: Urediniomycetes
Subclass: Incertae Sedis
Order: Uredinales
Family: Pucciniaceae
Genus: *Puccinia*
Species: *arachidis* Speg.

Usually, the pathogen is observed in the uredial stage on peanut plants. Uredosori are subepidermal, amphigenous, and scattered. Each sorus contains numerous uredospores. The uredospores are round to oval and pedicellate. The pedicel of the uredospore is short, fragile, and hyaline. The epispore is thin walled, echinulate, and cinnamon colored. The germ pores are 2–3 or 3–4 and are located equatorially in the spore. The spores measure in the range of 18.56–33.00 × 17.47–26.48 μm, with an average size of 24.96 × 21.22 μm.

The telial stage is reported on the peanuts in Brazil (Spegazzini 1884) and India (Chahal and Chohan 1971), but its presence has been found to be quite rare. It is interesting that Chahal and Chohan (1971) have reported only the telial stage and not the uredial stage of peanut rust while noting that it is the first report of peanut rust occurrence in India. Such a situation could be attributed to possible differences in variability in the isolates of *P. arachidis*, for example, out of several isolates collected from the state of Karnataka in India, only one *Gadag isolate* shows a rare phenomenon of teliospore formation (Tashildar et al. 2012). Aecia and pycnia are not known. An alternate host, if any, is also not known.

Epidemiology and Disease Cycle

Little new information on epidemiology and disease cycle of peanut rust has become available in the last three decennia. The sexual stage (teleutospore formation) is rare in the main areas of peanut cultivation and is epidemiologically insignificant, and the absence of an alternate host or a collateral host indicates that in the endemic areas, the fungus perennates in the uredinial stage on either volunteer self-sown plants or autumn-sown crops. There has been a large swing to autumn and spring crops in many peanut-growing countries including India, and this combined with the increased number of peanut crops has favored the pathogen survival. Uredospores, as such, without the living host have a

very short life except at very low temperature. Uredospores can be stored at low temperature without loss of viability, but at 40°C temperature, they rapidly loose viability. For instance, uredospores in the exposed crop debris lose all viability within 4 weeks under postharvest conditions at Hyderabad under Indian conditions. It thus appears that in tropical and subtropical countries, the pathogen does not survive uredospores per se from year to year. Hence, survival and development of the disease are limited by both temperature and survival of host tissues. Such epidemiological studies on peanut rust have also been carried out in detail in Mexico (Noriega-Cantú et al. 2000). Logistic model for prediction of the rust of peanuts under Karnataka conditions has been given by Gururaj et al. (2006). Similar prediction model has been developed by Narayana et al. (2006).

The possibility of the pathogen being carried through seed as a surface contaminant has been indicated, as introduction of rust on the peanuts in the United States, Brunei, Australia, and Papua New Guinea is reported to be the result of seed transmission of the pathogen through imported seed (Kolte 1984). Usually, the primary infection is attributed to uredospores transported by wind from short or long distances where the volunteer or self-sown rust-affected peanut plants are located or where the unexposed affected plant debris is present (Gururaj and Kulkarni 2007). In the southern part of India, there is an extensive and continuous cropping of peanuts at all times of the year, and thus, there is an easy availability of uredospores as primary inoculum from one season to another. It is clear that peanut rust is now well established in India and that it shows a clear pattern of spread from one or two origins where the crop circumstances permit inoculum buildup. Thus, the infection appears in July–August in southern India, in September and October in central India, and in November and December in the northeast. It appears, therefore, that the initial inoculum infecting peanuts in northern Andhra Pradesh and Maharashtra is derived from southern states and that, in turn, central Indian crops serve to provide the initial inoculum infecting those in West Bengal and Assam (Mayee et al. 1977). In the United States, the fungus does not overwinter but blows in from subtropical areas (Van Arsdel 1974).

After deposition of the uredospores on the leaves of the peanut plants, they germinate by giving rise to a germ tube at the temperature range of 15°C–30°C, with an optimum range being 20°C–25°C (Kono 1977). For spores to germinate on a leaf surface, the presence of free water is a must. The uredospores do not germinate at a relative humidity below 100%. Relative humidity above 80% supports the germination when spores are placed in a thin film of water (Cook 1980b). After germination, the germ tube grows and forms an appressorium over stomata. Cook (1980a) has studied the infection process in detail. According to him, close adhesion of the germ tube leaf surface is essential for appressorium formation. From the appressorium, the infection hypha arises and penetrates the tissue through stomata. After transversing the length of the stomatal passage, the infection peg swells and forms a vesicle in the substomatal chamber. Several infection hyphae then arise from such a vesicle, and the subsequently formed mycelia become intercellular by producing knob-shaped haustoria into the cells. Most infections take place successfully at 22°C–26°C. About 8–10 days after incubation, symptoms become visible with the production of a new crop of spores that become wind borne and cause secondary infection.

The factors that directly affect the spore germination have an indirect effect on disease development. Light inhibits uredospore germination and germ tube elongation (Subrahmanyam et al. 1980). Thus, it appears that there are more chances of getting a crop infected with the pathogen during evening or night hours than through the day. The density of spore concentration also affects the spore germination and subsequent infection process. Spores do not germinate in clumps and dense patches because of a high concentration of self-inhibitor within them and in surrounding water. The self-inhibitor has been isolated by Foudin and Macko (1974) and identified as methyl *cis*-3,4-dimethoxy cinnamate. It is found that *P. arachidis* is more sensitive to self-inhibitor chemicals than any other rust fungus. Leaf surface influences the infection, probably because of the differences in wettability of the leaf surfaces. It is usually seen that the abaxial surface of the leaf is more wettable compared with adaxial surface, and therefore, more infection sites develop on abaxial surface.

Under conditions of high rainfall and humidity in the *postrera* planting season in Honduras and Nicaragua (Central America), the disease becomes devastating, and it then becomes difficult

to control (Arneson 1970). In Venezuela, the rust becomes severe when the rainy season is nearly over or when dew is abundant (Hammons 1977). In India, a continuous dry period characterized by high temperature (>26°C) and low relative humidity (<70%) is reported to delay rust occurrence and severity, whereas intermittent rain, high relative humidity, and 20°C–26°C temperature favor disease development (Siddaramaiah et al. 1980). Aerial dissemination of uredospores has been studied, and diurnal periodicity with peak occurrences around noon was reported under Indian conditions (Mallaiah and Rao 1982), and currently changing scenario of prevalence of rust in the late 1990s from the three important southern states Andhra Pradesh, Karnataka, and Tamil Nadu has been described by Pande and Rao (2000).

In India, during summer months (May/June) when incidence of the rust is low, the incubation period is long (18 days), while in winter, when the rust is abundant, the incubation period is only 7 days (Mallaiah 1976). More or less similar observations, with respect to incubation in summer and winter seasons, have been reported from Taiwan (Fang 1977).

Susceptibility of the plants appears to be related to age, with plants becoming susceptible to disease at 5–6 weeks of age (McVey 1965).

There appears to be a relationship between altitude and appearance of the rust on peanuts. In lowveld areas (altitude 430 m) in Zimbabwe, the crop is severely affected, whereas in highveld areas of that country, rust appears quite late and in less severe form. A close relationship in the climatic requirements of rust and LLS has also been noted, as both are favored at lower elevations (Rothwell 1975). A similar situation appears to be true with the development of rust and LLS in Malawi (Sibale and Kisyombe 1980) and perhaps in southern parts of India. Though the pathogen is mainly restricted in its host range to *A. hypogaea*, it has recently been found to naturally infect *Arachis repens* with the formation of both uredospores and otherwise rarely formed teliospores in Brazil (Rodrigues et al. 2006).

Disease Management

Host Plant Resistance

In Cultivated Arachis *Species*

Screening has been conducted on large scale under both natural conditions and artificial inoculation of plants (4–5 weeks old) with uredospores to locate the sources of resistance especially at ICRISAT where a range of techniques have been developed (Subrahmanyam et al. 1982, Waliyar et al. 1993, Sudhagar et al. 2009).

On the basis of the percentage of leaves infected due to rust or percentage of defoliation due to rust, rating scales for discriminating resistant and susceptible genotypes have been used. No symptoms of rust, leaves showing slight infection, and less than 25% of leaves showing severe symptoms or defoliation due to rust disease have been taken as criteria for determining resistance.

It is found that highly and moderately rust-resistant genotypes are characterized by higher cuticular and epidermal cell thickness, lesser and smaller epidermal cells, lower number of stomata, and more wax content at the later stages of crop growth (Gururaj and Kulkarni 2006). Differences in the degree of and development of rust mycelium in the substomatal cavities are manifested in resistant and susceptible reactions. In highly resistant and immune types, the germ tube dies after penetration through stomata without the further development (Nevill 1980). In the nonphysiological resistant types, chloronemic flecks are formed without the formation of uredia and uredospore release, but in the physiological type of resistance, less than half of the chloronemic flecks are developed into uredia as in NCI3 cultivar (Cook 1972, 1980a). In nonphysiological resistant cultivars, resistance is related to leaf wettability, which in turn determines the spore retention capability of the cultivar. Cultivars with thin and less waxy leaves are generally affected earlier than those with thick and waxy leaves (Chen et al. 1981). Sudhagar et al. (2009) reported that the activity of PO and PPO is maximum at 80 DAS and that ascorbic acid oxidase and chitinase enzymes exhibit their maximum activity at 80 DAS

TABLE 2.2
Peanut Genotypes Resistant (R) or Moderately Resistant (MR) to Rust Disease of Peanuts as Reported from Different Countries in the World

Genotype	Country	R/MR	Reference(s)
DH22 (red), DH22 (tan), GPBD-4, K-134, R8808, R9214, R9227, R2001-1, R2001-2, and R2001-3	India	MR (partial resistance)	Gururaj and Kulkarni (2008)
ICGV 93207 (named as Sylvia)	Released for Mauritius (ICRISAT)	R	Reddy et al. (2001a)
ICGV 94361	India (ICRISAT)	MR (early)	Upadhyaya et al. (2001a)
ICGV 87354	India (ICRISAT)	R	Reddy et al. (2001b)
ICG 8954 (*A. kuhlmannii*)	India (ICRISAT)	Immune (asymptomatic)	Pande and Rao (2001)
ICGV 87853 (Venus)	India (ICRISAT)	R	Reddy et al. (2000)
M-5, 255/88	Bangladesh	MR	Hossain et al. (2007)
JALW-20, JL-501	India	MR to rust + LLS	Deshmukh et al. (2009)
VL-1	India	R	Kumar et al. (2012)
ICGV 98383	India	R	Patil et al. (2010)
ICGV 99003, ICGV 99005, ICGV 99012, ICGV 99015	India (ICRISAT)	R	Singh et al. (2003a), Dwivedi et al. (2002)
NC17090	—	R	Pensuk et al. (2003)

with prominent expression of a 56 kDa protein in rust-resistant genotypes of peanut. The potential amount and activity of these are genetically determined, and such changes in the quantity of isozyme and protein can be relied for screening rust-resistant/rust-tolerant genotypes. Induction and accumulation of phenols and PPO enzyme have been found to be at a faster rate in rust-resistant genotypes ICG 1697 and ICG 10053 on inoculation with uredospores (Kumar and Balasubramanian 2000).

Marked sources of resistance in the cultivated peanut have been reported by several workers (Tables 2.1 and 2.2). Some genotypes are resistant to both rust and LLS. Some peanut genotypes such as GPBD-4, DH22 (red and tan), and R9214 (Gururaj and Kulkarni 2008) and genotypes such as ICGVs 99003, 99005, 99012, and 99015 (Dwivedi et al. 2002) show the slow-rusting ability characterized by longer incubation period, low sporulation index, lesser number of pustules per unit area, and smaller pustule size. In such cases, the resistance is controlled by several genes. A new Australian peanut cultivar *Sutherland* has significantly higher level of resistance to rust (and also to LLS) under the Queensland conditions in Australia (Kelly et al. 2012).

In Wild Arachis *Species*

The genus *Arachis* is native to South America and also consists of 22 described species and more than 40 undescribed. Gene banks are maintained in Brazil, the United States, and India. Many germplasm accessions have been screened and several peanut genotypes with immunity or high level of resistance to peanut rust have been identified, with sources for resistance mainly originating from Peru, Bolivia, and India (Wynne et al. 1991, Varman et al. 2000, Yadav et al. 2007). A very high degree of rust resistance in wild species of the genus *Arachis* has been reported. Such species are *A. duranensis* (PI 219823, section *Arachis*), *A. correntia* (PI 331194, section *Arachis*), *A. cardenasii* (PI 262141, section *Arachis*), *A. chacoense* (PI 276235, section *Arachis*), *A. chacoense* × *A. cardenasii* (F1 hybrid), *A. pusilla* (PI 338448, section *Triseminalae*), *A.* sp. 9667 (PI 262848, section *Rhizomatosae*), *A.* sp. 10596 (PI 276233, section *Rhizomatosae*), *A. glabrata* (PI 118457, 231318, 262287, 262801, section *Rhizomatosae*), *A. villosulicarpa* (PI 336985, section *Extranervosae*), and *A. villosa* (Subrahmanyam et al. 1982). *A. stenosperma* accession V 10309 is resistant to rust and LLS and the experimental evidence reveals that in *A. stenosperma*, infection is hampered at the stage of penetration (Leal-Bertioli et al. 2010).

Resistance to rust in some wild species has been found to be mostly recessive and governed by monogenic (3:1), digenic (15:1), and trigenic (63:1) F2 segregation ratios (Joel et al. 2006). Susceptibility is dominant to resistance and F2 population segregates in the ratio of 3:1 with resistance governed by a single recessive gene (Paramasivam et al. 1990). However, rust resistance in peanuts is also reported to be controlled by both additive and nonadditive gene actions and additive gene effects are predominant (Ghewande 2009). Thus, wild germplasm accessions of both A and B genome types are available to be used for the introgression of resistance genes against rust fungal pathogen (Fávero et al. 2009).

Molecular Breeding and Transgenic Peanuts for Rust Disease Resistance

Comparison of molecular profile among different peanut cultivars, genotypes, and breeding lines with differential disease reaction against rust has been carried out using RAPD (Mondal et al. 2005, 2008a,b) and ISSR marker analysis (Varma et al. 2005, Mace et al. 2006, Mondal et al. 2008a, 2009). Of the two markers, ISSR reveals higher polymorphism (74.5%) than RAPD (47.1%) with the average number of polymorphic bands per assay unit being 5.4 in ISSR and 3.3 in RAPD (Mondal et al. 2008b). Mondal et al. (2008b) have been the first to report on the identification of RAPD markers linked to rust resistance in peanuts in India and that RAPD marker $J7_{1300}$ is reported to be applicable for marker-assisted selection in the peanut rust resistance breeding program. The ISSR primer UBC 810_{540} is found to be associated with both rust and LLS resistances (Mondal et al. 2009). A study has been conducted by Mace et al. (2006) using ISSR analysis to identify diverse rust disease–resistant germplasm for the development of mapping populations and for their introduction into breeding programs. Twenty-three SSRs have been screened across 22 groundnut genotypes with differing levels of resistance to rust and LLS. Rust resistance in peanuts is associated with two SSR alleles (pPGPseq3A1$_{271}$ and pPGPseq3A1$_{390}$) in ICGV 99003 × TMV-2 and seven SSR alleles (pPGPseq5D5$_{270}$, pPGPseq5D5$_{295}$, pPGPseq5D5$_{325}$, pPGPseq16F1$_{315}$, pPGPseq16F1$_{424}$, pPGPseq7F6$_{128}$, and pPGPseq13A7$_{292}$) in ICGV 99005 × TMV-2. SSR markers associated with rust resistance should facilitate the rapid identification and transfer of chromosomal region(s) into elite breeding lines by using marker-assisted backcross breeding in peanuts (Varma et al. 2005).

QTL analysis using inbred lines of a mapping population TAG 24 × GPBD-4 segregating for rust reaction reveals 12 QTLs for rust. Interestingly, a major QTL associated with rust ($QTL_{rust}01$), contributing 6.90%–55.20% variation, has been identified by both composite interval mapping and single-marker analysis. A candidate SSR marker (IPAHM 103) linked with this QTL has been validated using a wide range of resistant/susceptible breeding lines as well as progeny lines of another mapping population (TG 26 × GPBD-4). Therefore, this marker is considered to be useful for introgressing the major QTL for rust in desired lines/varieties of peanut through marker-assisted backcrossing (Khedikar et al. 2010). Similarly, the two more SSR markers pPGpseq4A05 and gi56931710 have been found to show significant association with the rust reaction, and these flank the rust resistance genes at map distances of 4.7 and 4.3 cM, respectively, in linkage Group 2 (Mondal and Badigannavar 2010, Mondal et al. 2012). Thus, tagging of the rust resistance locus with linked SSR markers can be useful in selecting the rust-resistant genotypes from segregating populations and in introgressing the rust resistance genes from diploid wild species.

Chemical Control

In order to ensure elimination of infection from the surface-contaminated seeds, treatment of seed with thiram or captan at 3–4 g/kg of seed or with any effective seed treatment fungicide is advisable.

Mancozeb and Calixin (chlorothalonil, fentin hydroxide, tridemorph, triadimefon, and benodanil) have had been the fungicides of choice for peanut rust control through foliar sprays in the past (Kolte 1984). Chlorothalonil appears to be more effective than mancozeb. In recent years, some newer fungicides such as hexaconazole (Johnson and Subramanyam 2003, Hossain et al. 2010), difenoconazole (Gopal et al. 2003, Kalaskar et al. 2012), tebuconazole (Besler et al. 2006, Hagan et al. 2006), and azoxystrobin (Hagan et al. 2006) have been found to be more effective in

controlling the peanut rust in comparison to mancozeb and chlorothalonil. In case the rust-resistant or partially resistant peanut cultivar is used, for example, the use of peanut cultivar *Sutherland* in Australia or in years with low rust disease pressure, few or reduced number of fungicide application will be needed to manage the disease (Kelly et al. 2012).

In order to control both rust and leaf spots, a mixture of two chemicals has been found effective. A mixture of systemic fungicide benomyl or carbendazim (0.05%) and mancozeb (0.2%) or a mixture of benomyl (0.05%) and mancozeb (0.2%) (Kolte 1984, Ghewande 2009) or a mixture of chlorothalonil (0.2%) and hexaconazole or a combination of tridemorph plus carbendazim (Mathur and Doshi 1990) has been reported to be superior in bringing about the control of rust and leaf spot diseases of peanuts with increase in peanut yield by 30%–40%.

Among the inorganic and metal salts, ammonium dihydrogen orthophosphate (monoammonium phosphate) and cobalt chloride have been found to be effective in controlling rust infection caused by *P. arachidis* (Kishore et al. 2001b).

Cultural Control

Cultural practices that destroy volunteer peanut plants or crop debris are an important measure to limit primary sources of inoculum. This is particularly important in Caribbean and Central American countries. In Australia, in the Atherton Tableland region, growers are encouraged to eliminate volunteer plants within 2 months before sowing to reduce the amount of inoculum early in the season. It has also been possible to introduce a degree of uniformity in planting time in Australia to minimize the probability of late plants being close to an early planted rust-affected crop. Under Indian conditions, early planting (15 days earlier than normal), plant spacing of 45 × 10 cm, and intercropping with red gram (*Cajanus cajan*) and castor (*Ricinus communis*) (Kodmelwar and Ingle 1989, Ghewande 2009) and with sorghum and pearl millet (Reddy et al. 1991) have been effective in reducing the peanut rust incidence and severity. Care should be taken to use seeds for sowing from healthy plants and noninfested regions.

Biological Control

The possibility of biological control of the disease by the use of antagonistic microorganisms has been indicated. Uredosori on peanut leaves have been found to be parasitized by mycoparasites such as *Darluca filum* (Biv.) Bem. ex. Fr, *Eudarluca caricis* (Fr.) O. Erik, *Daluea phylum* Byv and *Tuberculina* cos-*traricana* Sy, *Verticillium lecanii* (Zimm.) Viégas, and *Penicillium islandicum*. Mycophagous thrips, *Euphysothrips minozzi* Bagnall and *Dipteron* maggots (Patil et al. 2000) have also been reported to feed on uredospores of *P. arachidis* (Kolte 1984). However, no serious attempt has been made to use mycoparasites in the control of rust in peanuts, though sprays of culture filtrate of *V. lecanii* and *P. islandicum* were demonstrated to be effective in reducing the rust severity under field conditions (Ghewande 1993, 2009). A new fungal antagonist, *Fusarium chlamydosporum* Wollenw. & Reinking, has been isolated from the pustules of peanut rust significantly reducing the rust infection by *P. arachidis* using conidia and culture filtrate of the antagonist in artificial infection studies (Mathivanan and Murugesan 2000). Similarly, antagonistic isolate of *F. solani* has also been isolated from peanut rust pustules, and its antagonistic activity against *P. arachidis* is found to be due to chitinase and β-1,3-glucanase enzymes (Mathivanan 2000).

Effect of Plant Extracts

Foliar application of neem (*Azadirachta indica*) seed kernel extract (NSKE) at 3% (Gururaj et al. 2005b, Ghewande 2009) and aqueous leaf extracts of *Azadirachta indica* (Zade et al. 2005, Ghewande 2009) at 2% spray and that of *Prosopis juliflora* at 2% (Kishore and Pande 2005b), *Lawsonia inermis* at 5%, and *Datura metel* at 2% (Kishore et al. 2001, 2002, Kishore and Pande 2005, Zade et al. 2005) give reduction in the severity of rust disease index by 65%–74%. Aqueous plant extracts (30–40 g/L) of *Lippia multiflora*, *Boscia senegalensis*, and *Ziziphus mucronata*, the three local plants from the vicinity of Burkina Faso, show very strong antifungal activity against *P. arachidis* in vitro, and these have been found to be as good as or superior to fungicides in controlling the *P. arachidis* infection

on peanut leaves (Koïta et al. 2012). Integrating neem leaf extract (2%) with potassium (1.0% K_2O) (Hossain and Rahman 2007) and the NSKE (5%) or leaf extract (2%) of *D. metel* with fungicide chlorothalonil or difenoconazole is more effective in disease control (Kishore and Pande 2005, Kalaskar et al. 2012). More recently, flaxseed oil has been found to be effective in the control of peanut rust by adversely affecting the uredospore germ tube length and by completely suppressing appressorium formation, which is essential for pathogen (*P. arachidis*) to form an infection peg to pass through the stomatal aperture and infect the host tissue (Chen and Ko 2014). All such measures may be more economical, eco-friendly, and useful in improved control of the rust with lesser dependence on fungicides particularly for the resource-poor farmers in less developed countries.

Sclerotium STEM ROT

Symptoms

All the aboveground and underground plant parts can be affected. Several kinds of symptoms of the disease become visible depending upon the stage of plant growth, but the stem rot is more common. Usually, the disease appears more frequently as the plant approaches maturity. Infection may take place on the stem just above the soil surface or at the foot of the plant 1–2.5 cm below the ground level. In the beginning, the symptoms become visible in the form of a deep-brown lesion around the main stem at the soil level. It may occur on the stem below the soil surface under dry conditions or above the ground in wet weather. Soon after, the lesion becomes covered with white radiating mycelium that encircles the affected portion of the stem. The distinct rot occurs beneath the fungal weft leading to wilt-like symptoms characterized by yellowing and browning of the foliage that show drooping while remaining attached on the plants. Such plants remain upright in the row under field conditions. The entire plant or one or two branches may be killed. Death of the aboveground portion of plant sometimes takes place very rapidly, particularly in extremely hot weather. Coarse white strands of the pathogenic fungus growing in a fan-shaped pattern may be present on the surface of the affected plant parts or on the soil surface or leaf litter adjacent to affected plants. Later, brown-colored mustard seed–like sclerotia are often noticed intermingled with the fungal strands on affected plant parts or around the affected plants on soil surface (Figure 2.7). This facilitates spread of the disease on plants sown in rows, and thus, *the row effect* becomes evident under field conditions.

Geographical Distribution and Losses

The *Sclerotium* stem rot or SSR of peanuts is variously named as southern blight, sclerotium wilt, sclerotium blight, white mold, crown rot, foot rot, sclerotial disease, and sclerosteosis. The disease occurs throughout peanut-growing areas of the world in the tropics and in warmer parts of the temperate zones (Kolte 1984, Subrahmanyam et al. 1991, Momotaz et al. 2009, Shakil and Noor 2012). It is the most important disease on peanuts in India, Israel, and the southeastern United States, and the average annual peanut crop losses due to SSR are usually in the range of 25%–27% in India (Kolte 1984), Israel (Bowen et al. 1992), the United States (Damicone and Melouk 2009, Thiessen and Woodward 2012), and Australia (Middleton 1980). The disease incidence in the farmer's fields may be in the range of 0%–60%, and yield losses usually do not exceed 25% but may be as great as 80%. The losses are usually greater than what are apparent from field observations, as peg decay severs many nuts from the plant and they are left in the soil at harvest. The disease occurs in distinct foci as it spreads to adjacent plants in the row, and the number of disease foci is reported to be linearly related to yield loss in peanuts as reported by Rodriguez-Kabana et al. (1975). However, yield loss models from data from selected fungicide treatments indicate that the loss caused by SSR at low disease incidence may be proportionately greater than the yield loss at higher disease incidence and indicate that the relationship between SSR incidence and peanut yields may be nonlinear (Bowen et al. 1992). The SSR is, however, becoming a greater threat to irrigated peanut crop

FIGURE 2.7 SSR of peanuts. Note the mycelial growth and small mustard seedlike bodies of the fungus on rotted parts of the peanut plant.

in tropical countries. The temperature relation in the development of the disease and growth of the fungus seems to be the limiting factor in geographical distribution of the disease. More recently, the disease has spread in a more severe form over 80% in the heavily infested fields in the main peanut production regions of Henan Province in the P.R. of China (Xu et al. 2011) and to the extent of 25% in central Vietnam (Le 2011), and its prevalence is increasing in Indonesia (Taufiq et al. 2007).

Pathogen: Asexual anamorph stage (*S. rolfsii* Sacc); sexual stage (teleomorph, *A. rolfsii* (Curzi) Tu & Kimbrough)

Classification
Kingdom: Fungi
Phylum: Basidiomycota
Subphylum: Agaricomycotina
Class: Agaricomycetes
Order: Atheliales
Family: Atheliaceae
Genus: *Athelia*
Species: *rolfsii* (Curzi) C.C. Tu & Kimbr.

S. rolfsii does not produce conidia and is a deuteromycete in the group mycelia sterilia. The mycelium is septate and hyaline with conspicuous branching at right angles. A bud-like structure forms at the growing tip. It continues to grow and gives rise to a branch. The main branch hyphae are relatively large (5–9 μ in diameter) compared to many fungi that are more typically

with hyphal diameter of 2–4 μ. The well-developed mycelium, which forms the main vegetative body of the fungus, is in cord-like strands and grows as a creeping mycelium. The hyphae show the presence of clamps. Many a time, a number of hyphae are found to anastomose among them. Clamp connections in the form of forks and hooks or H-like in shape can be noticed. The young growing mycelial mass on the host surface, as well as on the medium, is generally snow-white with a silky luster.

Smaller-diameter (2–4 μ) hyphal cells called *feeding branches* arise from the main branches and penetrate the plant tissue. The well-developed mycelium growing in strands at its tips initiates the sclerotial formation in about 6–12 days, either on a host surface or on a medium. Sclerotia arise singly or quite close to each other, so as to assume a cluster or group at the tips of the growing mycelium. Initially, the sclerotia are whitish, but later they become yellowish white. A water drop is given out from the sclerotium during the course of its development, which on drying becomes dark brown and forms an outer coating. As the sclerotia age, they become dark brown to chocolate brown in color and are like mustard seed in appearance and size. The outer dark-brown ring of sclerotium consists of thickened cells followed by the formation of pseudoparenchymatous tissue.

The development of the perfect (sexual) stage is very rarely found in nature, and it has been the common practice to use the name of the sclerotial stage. Interestingly, the fungus is known to occur in its perfect stage as *A. rolfsii* causing the collar rot phase of the disease in the Marathwada Region of Maharashtra in India (Kadam et al. 2011). The development of perfect stage consists of the formation of a structure called a basidium in which meiosis occurs. Four haploid basidiospores are produced at the tips of small structures on the basidium called as sterigmata. *A. rolfsii* produces basidia in an unprotected layer (hymenium) that develops under humid conditions at the margins of lesions. Hymenial production consists of aerial, button-like loosely formed white growth, and it measures 0.1–0.75 mm. Basidia are club shaped and slender. They measure 20–25 μ × 4–6 μ. The sterigmata are shorter but more stout and measure 1.5–3 μ and vary from 1 to 4 in number. Basidiospores are hyaline, smooth, obclavate and measure 4–4.8 μ × 2–3.5 μ; when mature, the basidiospores are forcibly discharged.

Epidemiology and Disease Cycle

The pathogen is a facultative parasite found on a wide range of soils. The fungus can affect plants in about 100 families representing about 500 plant species. Most of these plants are dicotyledons comprising mainly composites and legumes. Members of the Gramineae family are regarded less susceptible.

Several investigators have compared the morphology and host range of different isolates from various hosts and concluded that the differences are small. Geographical variability among *S. rolfsii* populations is demonstrated. The importance of variability has been realized in the light of the discovery of pentachloronitobenzene (PCNB) tolerant strains of *S. rolfsii* isolated from a Texas peanut field in 1985 (Nelin 1992, Shim et al. 1998, Sharma and Singh 2002). There exists a variability among isolates of *S. rolfsii* in their ability to produce oxalic acid, which is correlated with the mortality of the seedlings; the lower the oxalic acid production by the isolate, less pathogenic it is in causing the seedling mortality (Palaiah and Adiver 2004). The variability in population structure of *S. rolfsii* isolates from peanut fields in Japan reveals the presence of four mycelial compatibility groups (MCGs). These are MCGs A, B, C, and D, and most isolates of the same MCG are proven to be clonal (Okabe and Matsumoto 2000). Similarly, in Vietnam, three distinct groups among isolates of *S. rolfsii* have been identified to display their diversity of genetic and phenotypic traits in mycelial compatibility, growth rate, and sclerotial characteristics (Le et al. 2012a). Based on internal transcribed spacer (ITS) ribosomal DNA sequence analyses, three distinct groups have been identified among field isolates of *S. rolfsii* from peanuts, tomatoes, and *taros* in India, and these show differences in aggressiveness, suggesting thereby that the most aggressive isolates be used in a consortium for the development of sick field plots for screening of resistance (Bagwan 2011a).

S. rolfsii survives through sclerotia or mycelium on plant debris in the top 6 cm of soil. It has a high oxygen demand and soil aeration affects survival; hence, viable sclerotia are found at greater depths in lighter sandy soil compared to heavier soils. Mycelia from germinated sclerotia first colonize dead or senescent plant tissue on the soil surface before infecting healthy plants. Colonized food bases supply energy for invasion of living plant organs and bridge distances between germinated sclerotia and the host. The fungus is most active at the soil surface and a mat of hyphae is formed over the basal portion of peanut. It clings to the epidermis but does not apparently penetrate living cells. It grows into host cells killed in advance by metabolites such as oxalic acid and/or pectolytic or cellulolytic enzymes produced by the fungus as it grows over the host tissues (Kolte 1997). Warm (30°C–35°C) aerated conditions in the upper few centimeters of the soil favor the development of the fungus. Generally, temperatures remaining at 29°C–35°C during most of the day and seldom dropping below 23°C during the night are more favorable for disease development. A soil moisture content of 40%–50% of water-holding capacity is optimum for disease development.

Sclerotial germination throughout the growing season is favored by low humidity and particularly by alternate drying and wetting (Porter et al. 1984). The accumulation of dried leaves around the peanut plants following defoliation due to leaf spots, insect damage, and drought creates optimum conditions for disease development by providing a food base for *S. rolfsii*. Phosphorus fertilization is reported to increase disease incidence but this is decreased by potassium. Peanut plants treated with benomyl often exhibit greater SSR problems, primarily because benomyl reduces the soil population of antagonistic *Trichoderma*. Rodriguez-Kabana et al. (1979) reported that 1,2-dibromo-3-chloropropane (DBCP) is stimulatory for the germination of dormant sclerotia in soil by producing mycelium that can use available organic debris to produce new sclerotia. This therefore leads to the production of more sclerotia leading to a higher inoculum density in DBCP-treated soil, subsequently resulting in higher incidence of the disease. Peanut plants infected by *S. rolfsii* emit a blend of organic compounds such as methyl salicylate and linalool, which appear to be inhibitory to the growth of *S. rolfsii* in vitro, suggesting thereby that the emission of these compounds by infected plants may constitute a direct defense against *S. rolfsii* (Cardoza et al. 2002). It has been shown that *S. rolfsii* can be seed borne in the peanut. But the possibility of survival and subsequent infection from this source is greatly overshadowed by soilborne inoculum.

Disease Management

Host Plant Resistance

Significant negative correlation between SSR incidence and yield per unit area reveals the importance of the development of SSR-resistant cultivars in obtaining higher yield under pathogen stress conditions (Krishnakanth et al. 2005). Several workers have attempted to spot out resistant varieties of peanuts to SSR. Peanuts of the bunch type are killed outright, but plants of runner peanuts are not killed by the disease, since portions away from the attack on runner peanuts are usually supported by the adventitious root system. It is probably because of this behavior that runner peanuts have been reported as less susceptible (Garren 1966).

Although the development of resistant cultivars to *S. rolfsii* is rather difficult, in recent years, a few peanut cultivars that have been registered to be resistant to tomato spotted wilt disease (caused by tomato spotted wilt virus [TSWV]) are also known to be resistant to SSR (Branch and Brenneman 2009). Such cultivars are Georgia-07W, Georgia-03L, and AP-3 (Gorbet 2007, Branch and Brenneman 2008, 2009), Florida-07 (Gorbet and Tillman 2009), DP-1 (Gorbet and Tillman 2008), Andru II (Gorbet et al. 2006a), and Carver (Gorbet et al. 2006b). Ashok et al. (2004a,b) from India confirmed through artificial infection studies that 11 genotypes, 3 belonging to Virginia bunch (*A. hypogaea* var. *hypogaea*; TCG 1525, PI 269710, NCAc 38), 2 belonging to Virginia runner (*A. hypogaea* var. *hypogaea*; Haryanawadi, ND 8-2), 4 belonging to *vulgaris* (*A. hypogaea* var.

fastigiata; SS 34, VRR 472, Tai son, and PI 1268559), and 2 belonging to Spanish (*A. hypogaea* var. *vulgaris*; NCAc18019 and RR5290), are highly resistant to SSR.

Moderate resistance to SSR is reported in registered peanut cultivars such as Tamrun OL 02 (Simpson et al. 2006), Phule Unap (Patil et al. 2005), Dh 8 (Krishnakanth et al. 2003, 2005), C-99R (Gorbet and Shokes 2002a), Florida MDR 98 (Gorbet and Shokes 2002b), GG-11 and GG-13 (Rakholiya and Jadeja 2010), R9227 (Pujar et al. 2011), JL-365 (Thakare et al. 2007), and Local 235 (Abd-El-Moneem et al. 2003). Root exudates of the moderately resistant *Local 235* cultivar from Egypt are characterized by the presence of arabinose, lysine, and tryptophan (Abd-El-Moneem et al. 2003). Host genotype and biocontrol treatment combinations have established that the biological control using *T. harzianum* is more convenient, less costly, eco-friendly, and more effective in partially resistant genotypes (Krishnakanth et al. 2003). The same conclusion should be valid when chemical control using propiconazole is combined with the partial resistance of the peanut cultivars.

Chemical Control

The application of sterol biosynthesis inhibitors (SBI) also referred to as DMIs and quinone outside inhibitor (QoI) fungicides are now known to be more effective than previously recommended fungicides such as quintozene and carboxin for the control of SSR (Johnson and Subramanyam 2000, Besler et al. 2006, Grichar et al. 2010, Augusto and Brenneman 2011, 2012). The SBI triazole systemic fungicides, namely, diniconazole, propiconazole, tebuconazole (Minton et al. 1990, 1991, Culbreath et al. 1992, 2009, Adiver and Anahosur 1995), cyproconazole (Culbreath et al. 1992, Adiver and Anahosur 1995), hexaconazole (Johnson et al. 2007b), difenoconazole (Cilliers et al. 2003), and prothioconazole (Augusto and Brenneman 2012), and the QoI strobilurin fungicides, that is, azoxystrobin (Johnson and Subramanyam 2000, Rideout et al. 2002, Bowen et al. 2006, Sconyears et al. 2007, Hagan et al. 2010) and pyraclostrobin (Hagan et al. 2007, Grichar et al. 2010), have been proved to give very efficient and effective control of SSR of peanuts. The QoI fungicides should be applied preventively or as early as possible in the disease cycle as these are effective in inhibiting early mycelial growth. Once the fungus is growing inside the plant tissues, QoI fungicides have little or no effect. The strobilurins act at one specific site in the cytochrome system in the fungus and inhibit the mitochondrial respiration and are in the same cross-resistance group (same mode of action) and belong to Fungicide Resistance Action Committee (FRAC) Code 11. Similarly, SBI fungicides belong to FRAC Code 3, which include triazoles. Although these fungicides pose less risk to human health and/or environment than alternative pesticides, they appear to be vulnerable to rapid buildup of resistance in fungal population, and hence, their use must be managed carefully to avoid appearance of fungicide resistance (Vincelli 2002). Mixing triazole (SBI fungicides) or strobilurins (QoI fungicides) with other fungicides that have different mode of actions is desirable in minimizing the development of fungicide resistance. Fontelis (penthiopyrad), the new pyrazole carboxamide, is recently established to show excellent control of SSR of peanuts in the United States and may complement current SBI and QoI fungicides. Fontelis would be an acceptable rotation partner for resistance management purposes with SBI triazole (Group 3) and strobilurin (Group 11) fungicides (Culbreath et al. 2009, Hagan 2012) in the management of SSR of peanuts. These chemicals are most frequently applied as granules at the pegging stage but may also be directed as sprays (Hagan et al. 1991). Flutolanil, a benzanilide systemic compound, has been found to show protective and curative effect for the control of SSR of peanuts in the United States (Csinos 1987, Timper et al. 2001) and Nicaragua (Augusto et al. 2010c). Seed treatment with triadimenol, another systemic triazole-type fungicide, or insecticide chlorpyrifos or carboxin, or ipconazole or azoxystrobin is also reported to be effective in controlling the SSR in the seedling stage of peanuts (Bowen et al. 1992, Rodriguez-Kabana and Kokalis-Burelle 1997, Rakholiya and Jadeja 2010, Akgul et al. 2011, Rakholiya et al. 2012).

Presowing treatment of susceptible peanut seeds for 24 h using 10^{-4} to 10^{-7} M dilute solutions of four growth-regulating chemicals, that is, indoleacetic acid, Cycocel (chlormequat), 2,4-dichloroacetic acid, and 2,4,5-trichloroacetic acid, significantly inhibits the development of *sclerotium* blight symptoms and reduces mortality in 2-week-old plants inoculated with the

S. rolfsii (Chowdhury 2003). Chitosan, a deacylated product of chitin, when used as seed treatment at 0.05%–1%, gives effective control of collar rot phase of the disease caused by *S. rolfsii* (Chowdhury 2002). Peanut plants raised from seeds treated with the aforementioned growth-regulating chemicals and chitosan show increased activity of PO and PPO enzymes producing more phenols and proteins. A combination of soil applications of insecticide and fungicide such as aldicarb + flutolanil (Minton et al. 1991) and chlorpyrifos + quintozene (Hagan et al. 1988) has been found to be more effective than either alone in the control of SSR.

Cultural Control

Deep burial of surface organic matter and crop debris by plowing it to a depth of 8–10 in. in soil during land preparation can improve the yield of peanuts by more than 50%, largely due to elimination of the food base of the pathogen and subsequent reduction of disease incidence (Kolte 1984, Desai and Bagwan 2005). The nondirting of peanuts during cultivation (as an interculture operation) likewise has been shown to reduce losses from SSR in the peanut crop, and a similar effect can be achieved by planting the peanut on slightly raised beds.

Since the pathogen is omnivorous, there is little chance of any control of the disease by following crop rotation. However, inoculum buildup of the pathogen in soil can be brought under control by rotating peanuts with less susceptible crop plants belonging to Gramineae family. For example, when maize precedes the peanut crops and the crops are under conservation tillage, population of biocontrol agents becomes higher, which then appears to be an important practice in the SSR management strategies as per the investigations carried out in Argentina (Vargas Gil et al. 2008). Crop rotation of maize, pearl millet, sorghum, garlic, and onion with peanut may be useful for the management of SSR of peanuts and also for the reduction of soil population of *S. rolfsii* due to the presence of certain antifungal compounds in the root exudates of these crops (Bagwan 2010, Vinod Kumar et al. 2012). The SSR incidence can be reduced to 62% with increase in pod yield by 15.5% when onions precede peanuts in the crop rotation (Zeidan et al. 1986).

Nondirting cultivation in combination with minimizing defoliation due to the leaf spot control can bring about significant control of SSR of peanuts in the subsequently planted crop. Soil amendment with basal application of gypsum at 500 kg/ha (additional to the normal practice of gypsum application at flowering) followed by neem cake at 150 kg/ha has been found to be useful in reducing the SSR of peanuts by 31%–39%, resulting in increase in pod yield by 200–260 kg/ha (Johnson et al. 2003). Among different forms of nitrogen and potassium fertilizers, application of calcium ammonium nitrate results in minimizing the incidence of SSR (Johnson et al. 2007b). Interaction of fungicide application timing and postspray irrigation is significant for SSR control and peanut yield. Applying fungicides at night when the leaves are folded and using irrigation water after fungicidal spray have both been shown to increase deposition of fungicides in the lower plant canopy, which subsequently improves control of SSR (Augusto and Brenneman 2011). Mulching the soil surface with wheat straw (80%–90% soil coverage) helps in restricting the increase in the SSR incidence, despite the reported increase in the density of *S. rolfsii* inoculum in such soils (Ferguson and Shew 2001).

Soil solarization is a nonchemical method for controlling the disease by means of solar heating of the soil. In this method, naturally infested soil when mulched for 6 weeks during July–August with transparent polyethylene (TPE) sheets raises the temperature of soil to 40°C–53°C, enabling killing or inactivation of the fungus. When such a field area is sown with peanuts, the following spring, the disease in the spring-planted crop is kept to minimum level, giving 52% more yield compared with the crop raised on untreated plots. This method has become more useful in Israel (Grinstein et al. 1979) and can be used in most tropical and subtropical countries including India (Reddy et al. 2007a,b).

Biological Control

Biocontrol agents particularly the fungi (*T. harzianum*, *Trichoderma viride*) and bacteria (*B. subtilis*, *Ps. fluorescens*, *Ps. aeruginosa*, *Ps. chlororaphis*) are seen to be an alternative and viable option for the management of SSR of peanuts as these have been found antagonistic to the growth of

S. rolfsii reducing its inoculum potential (Biswas and Sen 2000, Ray and Mukherjee 2002, Abd-Alla et al. 2003, Sahu and Senapati 2003, Desai et al. 2004, Pal et al. 2004, Saralamma and Reddy 2004, Abd-Allah 2005, Kishore et al. 2005c, Saralamma and Reddy 2005, Bagwan 2011b, Sharma et al. 2012). But the newly introduced biocontrol agents should be able to survive in the new ecological niche (Podile et al. 2002). For this purpose, low-cost local agricultural waste products such as wheat bran; oil cakes like mustard cake, castor cake, and neem cake; and FYM or compost can be used as the substrate for supporting the growth of effective antagonists as well as for retention of appropriate population ($\times 10^8$ cfu/g) of antagonists under field conditions (Vikram and Hamzehzarghani 2001, Nandagopal and Ghewande 2004, Bhagat and Pan 2007, Thiruvudainambi et al. 2010). Thus, combined soil application of 2.5 kg *T. viride* + 6 kg compost + 500 kg neem cake/ha gives effective control of SSR (Dandnaik et al. 2006), and oil cakes (mahua cake, neem cake, pungam cake) in combination with *T. viride* or *Ps. fluorescens*, each at 5 kg/ha of soil, give best degree of SSR control in peanuts (Varadharajan et al. 2006). Soil application of *T. harzianum* or *T. viride* in combination with thiophanate methyl + neem cake or with wheat bran saw dust + carboxin results in sustaining the antagonist population in soil giving better control of SSR (Patibanda et al. 2002, Saralamma and Reddy 2005). Similarly, lowest SSR incidence is reported when soil application of *Ps. fluorescens* is combined with FYM and tryptophan (Johnson et al. 2008a,b). A combined application of *Rhizobium* and *T. harzianum* is also beneficial in reducing the incidence of SSR (Ganesan et al. 2007). A diatomaceous earth with granules impregnated in a 10% molasses solution has been found suitable for the growth and delivery of *T. harzianum* to peanut fields. Granules coated with the growth of *T. harzianum* are applied 70 and 100 days after planting to the infested soil; this brings about significant disease control (Backman and Rodriguez-Kabana 1975, 1977). Besides antagonistic effects of *T. harzianum* or *T. viride* and that of *Ps. fluorescens*, plants treated with these biocontrol agents do show additional enhanced activity of plant defense-related enzymes, that is, *PO* and *polyphenol oxidase* (Varadharajan et al. 2006), whereas *Ps. aeruginosa* inhibits the plant cell wall–degrading enzymes (polygalacturonase and cellulose) of *S. rolfsii* and reduces the severity of peanut SSR (Kishore et al. 2005c). Some other strains of *Ps. chlororaphis* are, however, known to produce phenazines and lipopeptide surfactants—the thanamycin inhibiting the hyphal growth of *S. rolfsii* and suppressing the incidence of SSR (Le 2011, Le et al. 2012a,b). Some other strains of *Pseudomonas* sp. (strain BREN6) and *Bacillus* sp. (strain CHEP5) are capable of mobilizing infection-induced cellular defense responses (priming) in peanut plant. Inoculation of these strains increases the activity of PAL and PO enzymes, after challenge inoculation of peanut plants with *S. rolfsii*, and reduces the severity of the disease (Tonelli et al. 2011), indicating the induction of induced systemic resistance (ISR). Similar report on ISR against *S. rolfsii* infection has been made using fungal components of *S. rolfsii* in the form of fungal culture filtrate and the mycelial cell wall (Durgesh et al. 2010). Talc-based bioformulation mixture consisting of *Beauveria bassiana* (B2 strain) + *Ps. fluorescens* (strain TDK 1) + *Ps. fluorescens* (strain Pf1) amended with chitosan when applied through seed, soil, and foliar spray has been found to give effective control of *S. rolfsii* infection in peanuts (Senthilraja et al. 2010).

ISR in peanuts also becomes functional in response to use of certain arbuscular mycorrhizal fungi such as *Glomus caledonium* or *G. fasciculatum* when used alone or in combination with *Trichoderma* species for the control of SSR of peanuts (Ozgonen et al. 2010, Doley and Jite 2012).

In case of the seed treatment, *T. harzianum* or *T. viride* can be combined with compatible fungicides iprodione (Raihan et al. 2003, Manjula et al. 2004, Saralamma et al. 2004, Islam et al. 2005), thiophanate-methyl (Saralamma and Reddy 2005), difenoconazole (Cilliers et al. 2003), and chlorpyrifos (Rakholiya and Jadeja 2010) for the control of SSR in peanuts. Peanut seed treatment with *Ps.* cf. *monteilii* has been found to decrease the SSR incidence by 45%–66% (Rakh et al. 2011).

Water-soluble substances that occur naturally in the oat suppress the growth of *S. rolfsii*. Soil microorganisms that decompose oat residue also become more numerous and active during the decomposition process and suppress or destroy *S. rolfsii*. Based on this information, rotation of peanut with oat or rye to reduce the incidence of the disease is suggested (Webb 1971).

Effect of Plant Extract

Aqueous leaf and seed kernel extracts of neem (*A. indica*) (Ume-Kulsoom et al. 2001), aqueous leaf extract of *P. juliflora* and *Agave americana* (Kiran et al. 2006), and garlic, onion, pearl millet, sunflower, and sorghum at 5% (Vinod Kumar et al. 2009) show inhibitory effect on mycelial growth and sclerotial formation of *S. rolfsii*, indicating their potential uses in the management of SSR.

Aspergillus COLLAR ROT OR CROWN ROT

Symptoms

Collar rot or crown rot of peanut seedling is essentially a postemergence disease, but the preemergence phase where the seeds may rot and become covered with sooty black masses of spores can occur. On germination, the emerging hypocotyl is rapidly killed by the lesion below ground, resulting in rotting of the seedlings before their emergence from the soil. In the postemergence phase, crown rot is characterized in the field by wilting and death of seedlings accompanied by rotting of the hypocotyl. Under relatively moist conditions, accompanied by the high atmospheric humidity and high temperature prevailing during the monsoon period, hypocotyl rot itself is seen first as a yellowish-brown lesion that extends into the plant tissue, and affected collar region becomes shredded with a lapse of time and shows profusely sporulating, black growth of the causal fungus. Eventually, the hypocotyl becomes blackened and rotten. Most affected plants die within 30 days of planting, which leads to *patchy* crop stand. As plants develop woody stems and taproots, the disease is less likely to occur. However, later in the season, individual branches or entire plants may develop similar symptoms. Splitting the crown and taproot of affected plants reveals an internal discoloration of the vascular system that is dark gray in color. The dried branches are readily detached from the disintegrated collar region and are blown away by wind. Under dry conditions, the lesion in the collar region remains restricted, bringing about slow wilting and death of shoots in the proximity of lesions, and the rest of the shoots survive. If plants escape early infection, that is, immediate postemergence phase, the plants reach maturity, and crown rot symptom may develop. Occasionally, rotting is continued to the lower portion of the main root, in which case the plants produce adventitious roots above the diseased area. Such plants seldom thrive and usually die during dry weather (Kolte 1997).

Geographical Distribution and Losses

Collar rot or crown of peanuts caused by *A. niger* van Tieghem was perhaps first reported from Sumatra in 1925 (Jochem 1926). It was then reported from all over the peanut-growing countries in the world (Kolte 1984, Cantonwine et al. 2011). Since the disease causes considerable seedling mortality in the early stages of crop growth, crop yield is directly affected by reduction in the stand of the crop. The disease may cause an average 5% loss in yield but in some areas it may cause as high as a 40% loss. Collar rot is a more serious problem in sandy soil (Gibson 1953, Chohan 1965). In Punjab (India), the mortality losses of plants due to the disease may amount to 40%–50% (Aulakh and Sandhu 1970). Similarly, Ghewande et al. (2002) reported that losses in terms of mortality of plants due to collar rot range from 28% to 50%. Plant stand losses as high as two plants due to the disease per meter of planted row have been recorded in the North Territory (Queensland) in Australia.

A serious incidence of the disease was reported in Australia in 1951 (Morwood 1953) and in the United States, resulting in serious stand deterioration, in Georgia in 1961 (Jackson 1962), in Texas in 1962 and 1963 (Ashworth et al. 1964), and in New Mexico in 1965 (Hsi 1966). With increasing interest in producing organic peanuts in traditional peanut production areas in the southeastern United States, postemergence phase of collar rot (*A. niger*) has become the major obstacle regardless of the peanut cultivar being used in organic peanut production, though the disease along with

other seedling diseases could be controlled by standard chemical seed treatments (Ruark and Shew 2010, Cantonwine et al. 2011). Infection occurring within 50 days of sowing from untreated seed causes serious losses and can kill up to 40% of the plant stand.

Pathogen

The disease is known to be caused by the fungus *A. niger* van Tieghem. (In some instances, *Aspergillus pulverulentus* (McAlpine) Thom has been found as the cause of the disease.)

Classification

Domain: Eukaryota
Kingdom: Fungi
Phylum: Ascomycota
Subphylum: Pezizomycotina
Class: Eurotiomycetes
Order: Eurotiales
Family: Trichocomaceae
Genus: *Aspergillus*
Species: *niger* van Tieghem

A. niger is a member of the genus *Aspergillus* that includes a set of fungi that are generally considered asexual, although perfect forms (forms that reproduce sexually) have been found. Conidial heads are globose and black and measure 700–800 μm in diameter; conidiophores have thick smooth walls and measure 1.5–3.00 mm × 15–20 μ and show colorless to brownish shades in the upper half, with sterigmata in two series; primary sterigmata vary with the strain and with the age of the conidial heads and measure 20–30 μ × 5–6 μ at the beginning of sporulation, but often reach 60–70 μ × 8–10 μ at maturity; secondary sterigmata are more uniform, ranging usually from 7 to 10 μ × 3 to 3.5 μ; conidia are globose at maturity, echinulated, somewhat variable in size, and mostly measure 4–5 μ in diameter. Sclerotia are produced in some strains and may dominate the colony character; sclerotia are globose to subglobose, 0.8–1.2 mm in diameter (Kolte 1984).

A. niger has a total genome size that ranges from 35.5 to 38.5 Mb and is composed of about 13,000 genes. Of these genes, about 8000–8500 genes have functional assignments. In addition, about 14,000 ORFs have been identified in the genome that could potentially encode a protein. The DNA sequence of *A. niger* consists approximately of 33.9 million base pairs. The possible function of 6500 genes could be established, which is only about 45% of its total gene count (Debets et al. 1990).

Epidemiology and Disease Cycle

A. niger is a saprophyte found in almost every type of tropical soil. It can tolerate low soil moisture and develops best at temperature between 30°C and 35°C. It can survive on seed or in the soil. It may be carried in or under the seed testa (Kolte 1984, Desai and Bagwan 2005). Seeds become infected during the last days of maturation in the soil and also during harvesting, shelling, and handling. Both soilborne and seed-borne inocula serve as primary sources of infection and adversely affect the seed germination (Mohapatra 2011). The fungus enters the host through a wound on the seed coat (testa) or through the stem, and the cotyledons usually act as the site for primary infection. It is interesting that conidia alone are not capable of causing infection of uninjured tissues. The presence of mycelium is essential for the infection of the uninjured tissue (Nema et al. 1955). Conidia can cause infection only when the testa of the seed is broken, and infection through conidia as the inoculum source occurs only to the extent of 25%, whereas the combination of mycelium and conidia can cause 100% infection through injured seeds. If the injury is extended to the cotyledons, the infection is so rapid that seedlings die before emergence. No spread of the disease in a particular crop season is seen. This is probably because of the absence of secondary infection and critical stages of infection

period. A seed carrying infection gives rise to cotyledons with the development of the lesion, which in turn affects the hypocotyl or stem of the seedlings. The pathogen present in the soil infects either the cotyledons or the hypocotyls directly. What is usually observed is that peanut seedlings grow out of soil in such a way that their cotyledons remain slightly covered with a thin layer of soil. Even if they are not covered with the soil just after germination, they get covered immediately thereafter by the wind-blown soil. The soil-covered cotyledons of the peanut seedlings form a good substrate and are in proper environment for the growth of *A. niger* already present in the soil. Once the fungus is established on cotyledons, it grows into the collar region and causes collar rot of seedlings. In most of the cases, infection takes place within 30–35 days after planting. This period corresponds to maturity of the hypocotyl and shedding of the cotyledons.

The incidence of the disease is positively correlated with high soil inoculum levels and is more prevalent in fields continuously cropped with peanuts than in fields grown with nonhost crops. The main carryover from season to season is through plant debris in soil. Predisposition is a major factor in the development of *Aspergillus* crown rot. Adverse weather conditions, extreme fluctuations in soil moisture, poor seed quality, seedling damage from pesticides or fertilizer application, and any other factor that delays emergence are associated with the disease (Kolte 1997). Young plants are particularly susceptible especially if the seeds are planted too shallow (<3 cm) or too deep (>8 cm) and the soil is exceptionally wet or dry. High soil temperatures also increase the risk of infection. As plants mature and the soil cools, plants become less susceptible and the mortality rate declines. *A. niger* produces oxalic acid and pectinase (polygalacturonase) enzyme *in vitro* and *in vivo*. This indicates that oxalic acid and pectinase enzymes are involved in the pathogenesis, which is further substantiated by the fact that only virulent isolates of the fungus produce oxalic acid and avirulent ones are not capable of doing so. At a soil moisture level of 13%–16%, peanut seedlings are affected most by collar rot. Sandy or sandy-loam soil and amendment of soil with sulfur, FYM, and gypsum have been found to increase the incidence of collar rot.

Etiolated plants are more susceptible to the disease, and high light intensity has been found to render seedlings almost immune to infection (Ashworth et al. 1964). Lesions induced by drought stress and high temperatures predispose the plant to infection. This type of predisposition can occur in the field during the early period of crop growth by partially covering seedlings with hot sand (Ashworth et al. 1964, Kokalis-Burelle et al. 1997).

Disease Management

Host Resistance

Breeders have not actively selected and bred for peanut resistance to *A. niger* in cultivar development perhaps because the disease is successfully brought under control due to fungicidal seed treatment and only partial resistance has been reported in cultivated peanut germplasm. For example, genotypes, namely, EC 21115 (U-4-47-7), B-4, B-21, B-60, B-76, B-101, B-18L, Asiriya Mwitunde (Kolte 1984), N03081T (Bailey), Perry5 (Ruark and Shew 2010), J-11, and GG-2 (Gajera et al. 2013), have been found to be moderately or partially resistant to the disease. The runner types of peanut genotypes are less susceptible to collar or crown rot in comparison to bunch types (Wynne et al. 1991).

Chemical Control

Effects of Fungicides

Many seed-dressing fungicides are reported to be effective against collar rot of groundnut (Gangopadhyay et al. 1996, Karthikeyan 1996). Seed treatment with thiram at 0.5% or captan at 0.25% or Vitavax 200 (carboxin 37.5% + thiram 37.5%) at 0.4% has been found effective for the control of both the phases of the disease under field conditions almost all over the world (Purss 1960, Jackson 1964, Sidhu and Chohan 1969, Agnihotri and Sharma 1972, Rakholiya et al. 2012).

The better efficacy of thiram and carboxin is related to its fungitoxic ability and its influence in increasing the population of *T. viride* antagonistic to *A. niger* around the treated seed. Among the newer fungicides, azoxystrobin alone (Rideout et al. 2002, 2008) or Dynasty PD (a mixture of azoxystrobin + fludioxonil + metalaxyl-M) at 2.5 g/kg of peanut seed in dry form can be used, using equipment specifically designed to apply a dust seed treatment to peanut seed (Australian Govt: Australian Pesticides and Veterinary Medicine Authority Permit No.: Per 13513).

Effects of Nonconventional Chemicals

Copper hydroxide at 0.25 g/kg of peanut seeds could provide an option for growers in organic peanut production for the control of the disease (Tarekegn et al. 2007, Ruark and Shew 2010). Seed treatment with nonconventional inorganic salt chemicals such as barium sulfate, zinc sulfate, and zinc chloride has been found to be effective in the control of collar rot of peanuts (Dasgupta et al. 2000). Supplementing zinc ions as an antioxidant treatment during peanut seed germination has been found to be effective in controlling the hyper increase of reactive oxygen species (ROS) in certain peanut varieties (as GG-11 and GG-24), which then imparts host resistance to *A. niger* infection due to the formation of oligomeric protein of 110 kDa. It, therefore, reveals that control of ROS could control the *A. niger* infection in peanuts (Jajda and Thakkar 2012).

Cultural Control

Only sound, undamaged healthy seeds should be selected and treated with thiram or captan before sowing. The seeds should be sown in good soil moisture conditions, avoiding deep sowing (preferably not more than 2 in.) so that the emergence of seedlings is hastened and cotyledons come above the soil soon, and the pathogen is less liable to cause infection, thereby escaping the disease (Chohan and Kapoor 1967). During interculture operations, care should be taken to avoid injury to seedlings and deposition of soil particles on cotyledons. If cotyledons and the collar region remain exposed to aeration and light under field conditions, the symptoms of collar rot do not develop on peanut seedlings. This constitutes another important measure to control the disease, which can be achieved by ordinary hand hoe during the hoeing operation.

In India under the Punjab conditions, mixed cropping with short-stature crops like moth (*Phaseolus aconitifolius*) in alternate rows has been found useful in decreasing the incidence of collar rot caused by *A. niger*. Moth is a leguminous short-stature crop, and it does not compete with peanut, with regard to both nutrients and light (Chohan and Kapoor 1967).

A. niger fungal populations are rich in soils where continuous cultivation of peanut is a regular practice or peanut is included in the cropping system (Emmanuel et al. 2011). Therefore, it is appropriate that crop sequence of chickpea peanut or wheat peanut has been suggested for reducing the intensity of collar rot under Indian conditions (Chohan and Kapoor 1967). Planting of peanuts on land that has been kept fallow or cropped with grain sorghum the year before (or for longer periods) has been suggested under the U.S. conditions in New Mexico area (Hsi 1966). Irrigation of the fields within 28 days of sowing has been reported to be useful in protecting plants from severe damage in China (Lin 1982). Soil solarization with TPE tarping at 0.05 or 0.10 mm during April for 30 days is useful in reducing the crown rot incidence by 70%–95% (Reddy et al. 2007a,b).

Biological Control

Treatment of premoist peanut seed with talc and 0.5% carboxymethyl cellulose (CMC)-based formulation of antagonistic fungi *T. viride*, particularly strain 60, and *T. harzianum* at 3–4 g/kg seed alone and/or soil application of *T. harzianum* or *T. viride* at 25–62 kg/ha preferably in conjunction with organic amendment such as castor cake or neem cake or mustard cake at 500 kg/ha has been found to be effective in the management of collar rot disease (Raju and Murthy 2000, Rao and Sitaramaiah 2000, Sheela and Packiaraj 2000, Kishore et al. 2001c, 2006, Devi and Prasad 2009,

Mohapatra and Sahoo 2011, Gajera et al. 2011, 2013, Bagwan 2011b, Gajera and Vakharia 2012). *T. viride* is also reported to be tolerant or compatible with seed treatment fungicides like thiram and captan, and seed treatment with *T. viride* can be combined with reduced half dose of thiram or captan, which gives better control of the disease (Kishore et al. 2001c, Devi and Prasad 2009) than either of the *Trichoderma* or fungicidal seed treatment.

At least six plant growth-promoting rhizobacteria (PGPR) have been successfully investigated as biocontrol agents for the control of *Aspergillus* crown rot disease of peanuts. These are (1) fluorescent pseudomonads like *P. fluorescens* (Dilip et al. 1999, Haggag and Abo-Sadera 2000, Sheela and Packiaraj 2000, Dey et al. 2004, Anand and Kulothungan 2010), (2) *P. aeruginosa* strain GSE 18 (Achira et al. 2002, Kishore et al. 2005c, 2006), (3) *B. subtilis* strain G303 formulated for commercial seed treatment use as Kodiak FL (Ruark and Shew 2010, Cantonwine et al. 2011), (4) biofilm-producing *Paenibacillus polymyxa* (Haggag 2007, Haggag and Timmusk 2008), (5) other *Bacillus* species (Prabakaran and Ravimycin 2012, Yuttavanichakul et al. 2012), and (6) *Methylobacterium* sp. (Madhaiyan et al. 2006).

Out of those, Kodiak, a flowable formulation (Bayer Crop Science) that contains a select strain of *B. subtilis* G303 (not less than 5.5×10^{10} viable endospores), is worth mentioning. It is designed to use in combination with other registered seed-applied fungicides such as thiram, captan, and carboxin to extend window of protection. Within 4–8 h of planting, the bacterial endospores in Kodiak begin to reproduce, reaching a population of up to 1 million cells/g of root, and the actively growing bacteria surround the growing roots blocking the intrusion of pathogen, *A. niger*, into the plants. They also produce a chemical inhibitor that can slow the growth of the pathogen. Thus, *B. subtilis* or Kodiak is often effective and can be considered as standard bioagent seed treatment at 2.5 g/kg of seed for the control of crown rot and peanut stand establishment not only in intensive peanut crop production but also in organic peanut crop production (Ruark and Shew 2010, Cantonwine et al. 2011).

Postulated mechanism for better crown rot control due to the aforementioned biocontrol agents (*Trichoderma*, *Pseudomonas*, *Bacillus*, and *Methylobacterium* species) includes inhibition of growth of the pathogen by lytic enzymes (β-1,3-glucanase, chitinase, protease) produced by the antagonists and induction of systemic host resistance followed by promoted plant growth in terms of plant height, increased plant vigor, and efficient rhizosphere colonization and biofilm formation (Sailaja et al. 1998, Lashin et al. 1989, Haggag and Abo-Sadera 2000, Kishore et al. 2005d, 2006, Haggag et al. 2007, Haggag and Timmusk 2008, Devi and Prasad 2009, Anand and Kulothungam 2010, Gajera and Vakharia 2012, Yuttavanichakul et al. 2012). Interestingly, biocontrol of *Aspergillus* crown rot (dry rot) disease has been reported by using transconjugants obtained by the horizontal gene transfer from *P. fluorescens* to *Rhizobium*, and the percentage control efficacy has been found to be better due to the application of transconjugants (Ade and Gangawane 2010).

Effects of Plant Extracts

Seed treatment with *Calotropis procera* leaf extract at 10 mL/kg seed alone or in combination with *T. viride* at 4 g/kg seed has been reported to give significant control of *Aspergillus* collar rot of peanuts (Srinivas et al. 2005). Essential oils (Kishore et al. 2007) and Xenorhabdus metabolites (Vyas et al. 2005) are also reported to be inhibitory to *A.niger* pathogen causing the collar rot disease.

YELLOW MOLD AND AFLAROOT

Symptoms

Yellow Mold Phase

Because of the fungal growth and its secretion, the peanut seed disintegrates within 4–8 DAS, and the seed becomes yellowish brown in color. The testa loses its natural color, turns dark purple to black, and becomes brittle. The seeds become rancid, shrivelled, and turn leathery in texture (Figure 2.8). When the seeds are split open, the mycelium and sporulation of the fungus are clearly visible in the cavity

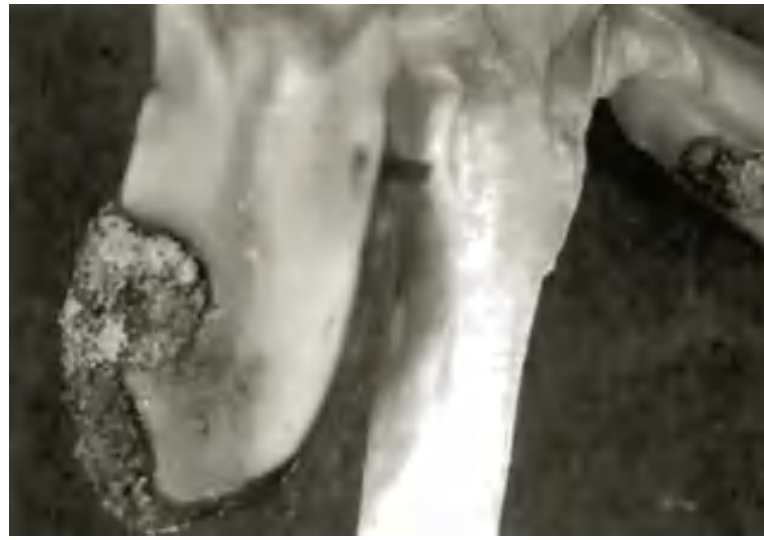

FIGURE 2.8 Yellow mold of peanuts caused by *A. flavus*.

between the cotyledons of the seeds. Under low soil moisture conditions, the decay is very rapid, since the activity of the fungus gets prolonged, owing to the delay in emergence of seedlings. Seedlings and ungerminated seeds shrivel to become a dried brown to black mass covered by yellow or green spores.

Aflaroot Phase

The germinating seeds, which escape the yellow mold phase, may show symptoms on the cotyledons. It is interesting that the hypocotyl is not affected. The pathogen is first seen on cotyledons, and from there it inhibits the growth of the plumule and the root. Cotyledon surfaces are covered with masses of yellow-green spores. The affected cotyledons show necrosis of the central tissues by forming reddish-brown lesions. Necrosis of the cotyledons terminates at or near the cotyledonary axis. The true leaves, which emerge from the affected seedlings, become reduced in size, with pointed tips, and show much variation in shape (Figure 2.9). The color of the affected

FIGURE 2.9 Aflaroot disease of peanuts caused by *A. flavus*.

leaves is yellowish green in comparison with the deep-green color of the leaves of healthy plants. The leaflets also show vein-clearing symptoms. The auxiliary branch arising from the side of affected cotyledons does not grow normally, but remains quiescent. If both the cotyledons are affected, plants remain quite stunted and show great variation in shape and size of the leaflets. The leaves remain thin with a shortened petiole. Leaves are rough to the touch and leathery and appear to be deficient in chlorophyll. When the affected plants are pulled out and examined, it becomes evident that the radicle is without secondary root development denoting the condition described as *aflaroot* by Chohan and Gupta (1968). Under field conditions, the diseased plants can be easily spotted because of their reduced growth and general chlorosis. Since the necrosis of the seedlings does not proceed to the hypocotyl, the aflaroot disease-affected seedlings continue to live till maturity.

Geographical Distribution and Losses

These diseases occur in most peanut-growing countries of the world particularly in Egypt, India, and Sudan (Mehan et al. 1991, Kolte 1997). Incidence of *Aspergillus* (*A. flavus*) contamination of peanut seeds favoring potential occurrence of seedling infection (aflaroot) is recently highlighted from Ethiopia (Mohamed and Chala 2014). Yellow mold was first observed in 1984 in a commercial peanut farm in South Texas in the United States (Subrahmanyam et al. 1987). Losses of 10%–20% in seedling emergence due to yellow mold phase and 5%–11% due to the aflaroot phase have been reported from India (Chohan and Gupta 1968).

Pathogen: *A. flavus* (Link) ex. Fries

Classification
Domain: Eukaryota
Kingdom: Fungi
Phylum: Ascomycota
Subphylum: Pezizomycotina
Class: Eurotiomycetes
Order: Eurotiales
Family: Trichocomaceae
Genus: *Aspergillus*
Species: *flavus* (Link) ex. Fries

The fungus is ubiquitous and saprophytic, and it grows rapidly on a variety of media. Conidial heads typically radiate measuring 500–600 μ in diameter, with the conidiophore coarsely rough, heavy walled, and usually less than 1 mm in length—but it may measure up to 2–2.5 mm—sterigmata are either uniseriate or biseriate with the two conditions rarely occurring in the same heads. Primary sterigmata measure 6–10 μ × 4–5.5 μ. Secondary sterigmata measure 6.5–10 μ × 3–5 μ. Uniseriate sterigmata are variable in size from 6.5 to 14 μ × 3 to 5.5 μ with conidium forming tips that are usually phialiform; conidia are globose to subglobose and inconspicuously echinulate and vary in diameter size from 3 to 6 μ, but most measure 3.5 to 4.5 μ. Sclerotia are produced in many strains and sometimes these dominate the colony characters. Sclerotia are globose to subglobose and red brown in color and measure about 400–700 μ. The toxigenic isolates produce more aflatoxin B and show stronger pathogenicity, whereas nontoxigenic isolates are poor producers of the aflatoxin (Tang et al. 2002). Maximum production of the aflatoxin is produced by toxigenic isolate at 25°C. It is reported that highly toxigenic isolates produce abundant sclerotia in culture media.

Epidemiology and Disease Cycle

The pathogen is seed borne as well as soilborne and survives in crop debris, and its ability to cause disease is related to strong saprophytic competitive ability. The pathogen is very active when soil moisture is below the field capacity and the atmospheric relative humidity is high. It can grow over a temperature range of 17°C–42°C and the optimum temperature for aflatoxin production is 25°C–35°C. The population of *A. flavus* propagules remains high in the top 5 cm of soil and gradually decreases with soil depth. Vertisol soils support a smaller population of *A. flavus* than the alfisols. Damage to pods by the lesser cornstalk borer, *Elasmopalpus lignosellus* (Zeller), exacerbates the disease (Kolte 1997). The development of infection is related to the production of aflatoxin. Nontoxigenic isolates of the fungus are not pathogenic to seedlings. However, no correlation between mycelial growth and aflatoxin production is observed (Lisker et al. 1993). The extent of yellow mold damage and aflatoxin is dependent on the environmental conditions, production, harvesting, and storage practices. Following harvest, further infections may develop with fungal growth covering the seed surface and invading the seed itself. A yellow or brown discoloration of pods and weight loss of pods result in subsequent losses (Satish Kumar and Popat 2010).

Disease Management

Since the fungus is a weak parasite, agronomic practices that favor rapid germination and vigorous growth of the seedlings will reduce the chances of *A. flavus* infection. Peanut germplasm lines possessing thick seed testa structure and protein characteristics can resist the infection by *A.flavus*. (Shan et al. 2006, Upadhyay et al. 2001a,b, Wang et al. 2010). Seed treatment with some of the fungicides, for example, carbendazim, captafol, mancozeb, or thiram, at 3–3.5 g/kg peanut seed has been found to manage the disease significantly under field conditions (Kolte 1984). The yellow mold and the possibility of controlling the aflaroot by the use of antagonistic isolates of *T. viride* and *T. harzianum* have been studied (Desai et al. 2000, Anjaiah et al. 2006, Bagwan 2011b); besides, there exists a potential of using certain isolates of plant growth-promoting fluorescent pseudomonads for the control of the infection caused by *A. flavus* and yield enhancement attributes in peanuts (Dey et al. 2004), whereas biocontrol of aflaroot disease has been reported by using transconjugants obtained by the horizontal gene transfer from *P. fluorescens* to *Rhizobium*, and the percentage control efficacy has been found to be better due to application of transconjugants (Ade and Gangawane 2010) and transgene over-expressing a tobacco β-1,3-glucanase in peanut (Sundaresha et al. 2010). Gamma-ray peanut mutant resistant to *A. flavus* infection has been reported (Azza et al. 2003).

CHARCOAL ROT

Symptoms

The symptoms of the disease appear in different phases. The development of water-soaked lesions on the hypocotyl near the soil surface is a characteristic symptom of this disease. After the hypocotyl is girdled, the seedling dies. Usually, the plants show typical symptoms when they approach maturity. Initial symptoms become visible in the form of the development of a red-brown water-soaked lesion on the stem just above the soil surface. Gradually, the lesion then becomes dark and spreads upward as well as downward, covering larger areas of the stem and root. The stem rot symptoms develop partially or completely by girdling of the stem by the lesion. If the stem is completely girdled by the lesion, the affected plants show wilting, followed by rapid colonization of the branches, which might result in defoliation. The plant then turns brown and subsequently dies.

Usually, rotting of the stem is associated with the rotting of the roots also. The root rot symptoms, independent of stem rot symptoms, appear rarely. If the root alone is affected, the taproot shows rotting, which becomes visible by a shredded appearance of the tissue. The dead tissue is covered with abundant minute black sclerotia, giving a charcoal or ashy-gray appearance to the tissue. Although not normally classified as a foliar pathogen, *M. phaseolina* does cause leaf spots (Gupta and Kolte 1982).

Geographical Distribution and Losses

Charcoal rot of peanuts has a wide geographical distribution and is especially found in tropical and subtropical countries with arid to semiarid climates in Africa, Asia, and North and South America. It is particularly important in Burma, Gambia, India, Israel, Kenya, Malawi, Mauritius, Nigeria, Senegal, Sudan, Syria, and Venezuela, but is of minor importance in the United States (Kolte 1997). All phases of the disease are economically important. Reduction in yield of the crop may be because of poor stand due to seedling rot or by killing of the plant at maturity. The percent incidence of mortality due to the disease may be as high as 65%–72% as reported from India and from Argentina. About 10% loss in yield due to the root rot phase of the disease has been reported from India and Palestine (Kolte 1997).

Pathogen: *M. phaseolina* (Tassi) Goid (syns. *M. phaseoli* (Maubl.) Ashby, *R. bataticola* (Taub.) Briton-Jones, *Sclerotium bataticola* (Taub.), and *Botryodiplodia phaseoli* (Maubl.) Thrium)

Classification
Kingdom: Fungi
Phylum: Ascomycota
Class: Dothideomycetes
Subclass: Incertae Sedis
Order: Botryosphaeriales
Family: Botryosphaeriaceae
Genus: *Macrophomina*
Species: *phaseolina* (Tassi) Goid

M. phaseolina is an anamorphic fungus in the ascomycete family Botryosphaeriaceae. It is normally being without an observable sexual stage, but many isolates have been shown to be anamorphs with sexual teleomorphs so they are generally grouped as mitosporic fungi (Coelomycetes) that have enclosed conidia. It is highly variable, with isolates differing in microsclerotial size and the presence or absence of pycnidia. Microsclerotia are of uniform texture and jet black in color and appear smooth and round to oblong or irregular. Across isolates, microsclerotia vary on size and shape and on different substrates. They are made up of anastomosing mycelial cells that are thick walled and dark brown, and several such cells compose an individual microsclerotium. The pycnidial stage is common on peanuts. Pycnidia are initially embedded in host tissue, then erumpent at maturity. They are 100–200 μm in diameter; dark to grayish, becoming black with age; globose or flattened globose; and membranous to subcarbonaceous with an inconspicuous or definite truncate ostiole. The pycnidia bear simple, rod-shaped conidiophores, 10–15 μm long. Conidia (14–33 × 6–12 μm) are single-celled hyaline, elliptical, or oval. Despite its wide host range and wide phenotypic diversity among isolates, the genus *Macrophomina* contains only one species *M. phaseolina* (Edraki and Banihashemi 2010). Molecular studies using species-specific probes and primers and RFLP and RAPD techniques show no variation among isolates from different hosts in restriction pattern of DNA fragments amplified by PCR of ITS region and also confirm that *M. phaseolina* constitutes a single species (Su et al. 2001, Babu et al. 2007). Sequencing of 92.3% of the genome of *M. phaseolina* has been done, and about 14,249 ORFs, that is, protein-encoding genes, are predicted and 9,934 validated by transcriptome (Islam et al. 2012). It has an abundance of oxidases, POs, and hydrolytic enzymes for degrading cell wall polysaccharides and lignocelluloses to penetrate into the host tissues.

Epidemiology and Disease Cycle

The causal fungus is both seed borne and soilborne. Mycelium in seed and mycelium and microsclerotia or pycnidia in plant debris in soil are primary sources of inoculum. The pathogen can be

detected in the seed coat, cotyledons, and embryo of peanuts (Chakrabarty et al. 2005). Peanut germplasm stored for various durations at 4°C commonly shows 10%–29% infection of this pathogen. Infected seeds do not germinate or produce seedlings that die soon after emergence (Singh et al. 2003b). The microsclerotia can remain viable in dry soils for many years, but rapidly loose viability in wet soils. The microsclerotia are black and spherical to oblong in structure that are produced in the host tissue and released into the soil as the infected plant decays. These multicelled structures allow the persistence of the fungus under adverse conditions such as low soil nutrient levels and temperature above 30°C. Microsclerotial survival is greatly reduced in wet soils surviving no more than 7–8 weeks and mycelium no more than 7 days. Germination of the microsclerotia occurs throughout the growing season when temperatures are between 28°C and 35°C. Sclerotia are then stimulated to germinate by giving rise to the mycelium that grows in the direction of the host surface. A certain amount of vegetative growth on the host surface appears to be essential prior to penetration, and then penetration occurs through cotyledons during emergence and through rootlets. Peanut seedlings are infected more rapidly and severely at 29°C and 35°C than at 18°C or 24°C, with plumules being invaded more frequently than roots. The intact pods are invaded most rapidly at 26°C–32°C. Mature pods are invaded, whereas actively growing pods remain free of the disease. When dry pods are allowed to hydrate over a 6-day period at 26°C, 32°C, or 39°C, pods are penetrated by the fungus quite extensively. Microsclerotia germinate on the root surface and germ tubes form appressoria that penetrate the host epidermal cell walls by mechanical pressure and enzymatic digestion or through natural openings. The hyphae grow first intercellularly in the cortex and then intracellularly through the xylem colonizing the vascular tissue (Okwulehie and Okpara 2002). Once in the vascular tissue, *M. phaseolina* spreads through the taproot and lower stem of the plant producing microsclerotia that plug the vessels. The rate of infection increases with higher soil temperatures, and low soil moisture will further enhance disease severity. Hot, dry weather promotes infection and development of charcoal rot. The charcoal rot is a greater problem when the plant is under drought stress. It is reported that incidence of the disease is higher in shallow cultivated fields than in those planted at a depth of 9 in. Damaged pods and kernels during harvesting and shelling are liable to be affected more by *M. phaseolina*. The fungal growth in pods is increased by rain after harvest. The mechanical plugging of the xylem vessels by microsclerotia, phaseolinone toxin production, hydrolytic and lignocellulose enzymatic action, and mechanical pressure during penetration lead to disease development (Islam et al. 2012). The population of *M. phaseolina* in soil will increase when susceptible hosts are cropped in successive years and can be redistributed by tillage practices.

Disease Management

Host Plant Resistance

Some of the peanut lines possessing less susceptibility or tolerance to the disease have been identified. For example, Spanish bunch peanut (*A. hypogaea* subsp. *fastigiata* var. *vulgaris*) cultivar TG 38 developed by irradiating with 300 Gy gamma-ray F1 seeds of the cross Girnar 1 × TG 26 has been released as resistant cultivar to charcoal rot in India (Kale et al. 2007). Drought-, waterlogging-, and PStV-tolerant peanut cultivar Huayu 16 is also resistant to *M. phaseolina*, and the same is released for wider adaptability in northern China (Li and Qiu 2000).

Chemical Control

Seed treatment with captafol or captan (0.38%) or thiram (0.45%) or Rizolex T (50%) has been found effective in improving seed germination and seedling stand. This results in the reduction of the disease incidence, increasing the yield by 13%–23%. However, the effect of seed treatment does not persist for so long period as to check the development of the disease at maturity or on developing pods. Soil treatment with benomyl drench (0.1%) followed by quintozene drench (0.5%) or with Rizolex T (50%) during the growth period is helpful to control the disease at a later period of crop

growth (Kolte 1997, El-Wakil and Ghonim 2000). If the seed colonization is <20%, seed treatment with a mixture of carbendazim and thiram (1:1) at 2 g ai/kg is used, routinely in plant quarantine laboratories to eliminate *M. phaseolina* (= *R. bataticola*) from peanut seeds, and seed samples having >20% colonization are rejected (El-Habbaa et al. 2002, Chakrabarty et al. 2005).

Cultural Control

Late sowing of the crop is suggested to reduce the incidence of the disease, but late sowing should not be done in areas where rosette is an important disease problem. Deficiency in soil moisture affects the physiology of peanut plants influencing the increase in the incidence of the disease that can, however, be reduced by irrigating the crop field (Okwulehie 2000, 2004, Chougule and Kore 2004). Balanced fertilization inclusive of trace elements such as copper, manganese, and zinc and insect pest control ensure good vigorous growth of the plants and help in the reduction of incidence of charcoal in peanuts. Application of gypsum at 150 kg/ha in severe cases results in a significant reduction of the disease (Kolte 1997).

Biological Control

The use of fungal antagonists *Trichoderma virens*/*Gliocladium virens* (Maheshwari et al. 2001, Christopher et al. 2008), *T. viride* (Malathi and Doraisamy 2004), *T. harzianum* (Malathi and Doraisamy 2004), *T. hamatum* (Vimala et al. 2000), and bacterial antagonist *B. sphaericus* (El-Shehaby and Morsy 2005) has been investigated to be useful in the control of charcoal of peanuts. Generally, seed treatment with the fungal antagonist at 4 g/kg of peanut seed in conjunction with soil application of the antagonist at 100 g/m^2 at the time of sowing and 30 DAS combined with FYM shows maximum reduction in the charcoal rot incidence (Christopher et al. 2008). Seed bacterization with talc-based formulation of PGPR such as *P. fluorescens* strain Pf1 or GRC strain (Meena et al. 2001b, Gupta et al. 2002, Shanmugam et al. 2002, 2003, Ramesh and Korikanthimath 2010) and *Bradyrhizobium* sp. (Deshwal et al. 2003) results in reduction of incidence of charcoal rot, making the PGPR as potential biocontrol agents for the control of the disease. With a view to understanding the broader aspects of control of the disease, the qualitative and quantitative aspects of rhizosphere mycoflora of peanuts as influenced by seed bacterization or seed-dressing fungicides, sprays of chemicals on the crop, and inorganic and organic soil amendments have been studied.

Effects of Plant Extracts

Cold-water extracts of *Allium sativum*, *Polyalthia longifolia*, and thyme have been proved to be effective in significantly reducing the incidence of charcoal of peanuts (El-Habbaa et al. 2002, Udhayakumar et al. 2008).

Sclerotinia BLIGHT

Symptoms

Sclerotinia species are causing the disease often referred to as stem rot, but the disease is truly a blight, characterized by sudden and serious damage to all aerial parts of the plant. Usually, the pegs are invaded first at the soil level, facilitating the colonization of the lateral branches. Light-tan to brown lesions demarcating the healthy and affected tissue appear on the lateral branches. The lesions then become dark brown, and shredding of the tissue becomes evident from an affected branch, and the fungus moves into and colonizes the main branch also. Leaves of such plants become chlorotic, turn brown, and wither, resulting in defoliation and death of the lateral branches or of the whole plant.

Pods of severely affected plants also show rotting. The taproot becomes necrotic and turns black in color. Abundant white fluffy mycelium appears on the soil surface in close proximity of the affected parts or debris in the field. Sclerotia of the fungus are also produced on the surface and within the affected branches, in the central portion of the taproot, on the pegs, on the surface of the pods, on the interface of the shell, and inside the seed.

Geographical Distribution and Losses

Occurrence of the disease is likely to be restricted only to such areas in temperate regions and under cool tropical conditions where the mean temperature drops below 25°C during the growing season of the crop often at intermediate altitude. The first report of occurrence of the disease caused by *Sclerotinia* sp. on peanuts was from Argentina in 1922 by Marchionatto (1922). Subsequently, the disease was reported from Australia, China, India, Israel, Japan, Australia, Mauritius, Taiwan, and the United States (Kolte 1984, Thiessen and Woodward 2012). The first reports of *Sclerotinia minor* causing the disease in the United States were made in Virginia in 1971 and then in North Carolina in 1972. Since its inception in the United States, the fungal disease has become widespread, having moved to Oklahoma, Texas, and New Mexico as well as becoming severe in Virginia and North Carolina. *S. minor* is more prevalent in most peanut-growing states in the United States, but *Sclerotinia sclerotiorum* has also been reported to be associated with the disease in Oklahoma (Wadsworth 1979), Georgia (Woodward et al. 2006), New Mexico (Sanogo and Puppala 2007), and Texas (Woodward et al. 2008) in the United States and in Argentina (Kolte 1984). Epidemic outbreaks of *Sclerotinia* blight of peanuts caused by *S. minor* have been of regular occurrence from 1994 to 1999 in Argentina (Marinelli et al. 2001).

Under low-temperature conditions, the disease can be quite destructive. Yield losses of up to 50% for peanut producers in the southwestern United States, particularly in Oklahoma, North Carolina, and Virginia, have been reported (Chenault et al. 2006). Pod yield losses have been correlated with aggravated disease incidence resulting from damage done to the plant. Midseason onset of a crown infection has the greatest impact on plant productivity, causing a severe decrease in pod yield and seed quality. Yield losses of near 80% in some areas have been reported in some areas in the United States. The requirement of calcium and intensive use of fungicides for disease control cause producers in the Virginia–North Carolina regions to have a higher production cost than most other growers in the United States (Partridge et al. 2006). Consequently, peanut crop production acreage in Virginia reduced from 23,473 ha in 2002 to only 6,880 ha in 2006.

Pathogen(s): *S. minor* Jagger and *S. sclerotiorum* (Lib.) de Bary

Classification

Kingdom: Fungi
Phylum: Ascomycotina
Class: Leotiomycetes
Subclass: Leotiomycetidae
Order: Helotiales
Family: Sclerotiniaceae
Genus: Sclerotinia
Species: *minor* Jagger and *sclerotiorum* (Lib.) de Bary

Small sclerotia type, for example, *S. minor* Jagger, and large sclerotia type, for example, *S. sclerotiorum* (Lib.) de Bary, have been reported to cause the disease. Both these species are necrotrophic fungal pathogens in the phylum Ascomycota, the order Helotiales, and the family Sclerotiniaceae.

Both produce dense mat of mycelium on the surface of the host and on adjacent soil surfaces; dense white bodies then form within this fluffy white mass of mycelium. *S. minor* produces small (0.5–2 mm in diam.), rough angular sclerotia, while *S. sclerotiorum* produces large (2–10 mm diam.) smooth rounded sclerotia. Identification should be made based on a group of sclerotia from the same colony rather a single sclerotium. In general, *S. minor* sclerotia are more numerous, smaller, and more angular than the sclerotia of *S. sclerotiorum*. *S. minor* sclerotia usually germinate by producing a mass of fungal threads and seldom produce ascospores, but sclerotia of *S. sclerotiorum* can produce ascospores from apothecia.

Epidemiology and Disease Cycle

Overwintering sclerotia of commonly occurring *S. minor* in the soil provide the primary inoculum for the disease. They germinate myceliogenically, producing a mass of mycelium that directly penetrates peanuts and causes infection on the pegs or lateral branches near the soil initiating the disease (Faske et al. 2006, Smith et al. 2006). Low temperatures (10°C–25°C) and high soil moisture favor infection, and the disease becomes severe when there are more cold days in a growing season. Myceliogenic sclerotial germination of *S. minor* and growth as well as infection and colonization of peanut tissues are optimum at soil matric potential of −7.2 kPa and optimum temperature of 30°C with 95%–100% relative humidity (Smith et al. 2006). *Sclerotinia* blight becomes more severe as soil pH increases from 6.0 to 6.5. The presence of volatile substances (aldehydes, esters, and halogenated hydrocarbons) from moist undecomposed peanut plant tissues has been shown to initiate sclerotial germination of *S. minor.* Field indices of available moisture in the form of rainfall or relative humidity >95%, air or soil temperatures <29°C, plant growth, and density of foliar canopy are used for predicting outbreaks of *Sclerotinia* blight and the need for fungicide sprays in the United States. As the *Sclerotinia* blight develops in the peanut canopy, numerous sclerotia are produced in and on diseased tissues. Sclerotia are then shed into the soil and increase the inoculum density of the pathogen, which subsequently becomes available in future growing seasons. Sclerotia of *S. minor* are able to remain viable for as many as 8 years in the soil. Since *S. minor* seldom produces ascocarps and ascospores, the latter are unimportant in the epidemiology of *S. minor*–caused disease. It usually attacks its host root and stem at or near the soil line in contrast to the most common formation of apothecia and ascospores in case of *S. sclerotiorum*. Hence, it is not uncommon to see *S. sclerotiorum* infections on the foliar parts of the plants because ascospores become airborne to spread the fungus and cause disease throughout the field. Senescent flower parts are an ideal site for ascospores of *S. sclerotiorum* to colonize. From this tissue, the pathogen can quickly invade healthy leaves, stems, and nuts. It can be a more serious pathogen at the flowering stage of the crop growth (Thiessen and Woodward 2012). Interestingly, unlike *S. sclerotiorum*, *S. minor* does not need a food base of dead or senescing tissues to infect. It has the ability to infect stems and branches in contact with the soil with no food bases (Shew 2011). Pathogenic *Sclerotinia* species produce oxalic acid, termed as pathogenicity factor, which predisposes plants to infection. Godoy et al. (1990) showed that *S. sclerotiorum* mutants that are unable to produce oxalic acid cannot infect susceptible plants. This indicates that oxalic acid is a necessary pathogenicity factor for the development of the disease. Plants injured during interculture operation are predisposed to infection. Peanut plants sprayed with captafol or chlorothalonil (0.56–2.24 kg/ha) are affected more severely by the disease, possibly because plants sprayed with these chemicals favor more production of oxalic acid by *S. minor.*

Disease Management

Host Plant Resistance

In Cultivated Arachis

It is difficult to find a high degree of resistance to the disease, but differences in the degree of host reaction through screening for resistance can be noticed. In general, Spanish and Valencia genotypes because of their upright plant canopy tend to exhibit greater resistance to the disease than the dense spreading Virginia and runner genotypes. The increasing level of resistance exhibited by these erect genotypes appears to be a plant developmental trait escape and early-maturity mechanism (Faske et al. 2006, Damicone et al. 2010). Some of the peanut varieties/lines, for example, Tamspan 98 (Simpson et al. 2000), Tamrun (Damicone et al. 2010), TxAG-4, VA 93B (Cruickshank et al. 2002), GP-NC-WS 12 (Hollowell et al. 2003b), Tamrun 98 (Tx 901417), and Perry (N93112C), and advanced breeding line N92056C (Lemay et al. 2002) have been found to show moderate resistance or tolerance or less susceptibility to the disease, but none have allowed or have a potential for doing away with the use of fungicide sprays.

Molecular Breeding and Transgenic Peanuts for Sclerotinia *Blight Resistance*

Genetically modified peanut lines (1) N70-8-B, P53-28-B, and W73-27-B (Partridge-Telenko et al. 2011) and (2) N70, P39, and W171 (Chriscoe 2008) expressing oxalate oxidase (Oxox) gene from barley have been developed successfully to enhance the resistance to *Sclerotinia* blight through degradation of oxalic acid by Oxox enzyme to produce carbon dioxide and hydrogen peroxide preventing predisposition of peanut cells to infection caused by the pathogen. It is demonstrated that the transgene Oxox in the peanuts has the potential to eliminate costs of fungicide use, increasing profits and promoting a more environmentally sound strategy for *Sclerotinia* blight management (Livingstone et al. 2005). Similarly, peanut transgenic lines No. 654 and No. 487 transformed with chitinase and glucanase genes (Douglas 2004), and about 32 some other peanut transgenic peanut lines possessing a rice chitinase and/or an alfalfa glucanase antifungal gene (Chenault et al. 2005) have been developed for resistance to *Sclerotinia* blight that may be useful in traditional breeding and disease management strategies. Simple sequence repeat (SSR) primer has been identified as a molecular marker associated with resistance to *Sclerotinia* blight in peanuts. Thus, identification of the marker and development of PCR-based screening method is extremely useful to peanut breeders in screening germplasm collections and segregating populations as well as in pyramiding *S. minor* resistance with other desirable traits into superior peanut lines (Chenault et al. 2009).

Chemical Control

While the use of resistant cultivars remains a viable option for the management of *Sclerotinia* blight, the use of fungicides may be necessary in cases of severe infestation. Presently, a number of fungicides have been tested for use in the control of this disease (Bowen et al. 2000, Ryley et al. 2000, Smith et al. 2008). The two most preferred fungicides for use in the control of this disease are fluazinam (Omega 500 F from Syngenta) and boscalid (Endura 70 WG from BASF). These fungicides are effective against *Sclerotinia* blight when applied as preventive measures. Timing of the first spray is critical. Fields with a history of serious problems should be scouted carefully beginning when plants are within 6 in. of touching. Spray the crop when *Sclerotinia* blight is first observed or 60–70 days after planting (calendar program) or according to a *Sclerotinia* blight advisory. A weather-based *Sclerotinia* blight advisory can be used to time applications and prevent unnecessary fungicide applications. If the disease continues to spread, one or two more applications may be made at 3–4-week intervals or according to the advisory (Shew 2011). In general, two or three sprays of fluazinam at 0.75–1.00 kg ai/ha can reduce the disease incidence in the range of 56%–80% on partially resistant cultivars. Boscalid performs marginally better than fluazinam (Smith et al. 2008). Foliar application of procymidone (0.68–0.75 kg ai/ha) has been found effective in the control of the disease.

Cultural Control

Damaging levels of the disease can be prevented by rotating the peanuts with nonhost cereal crops such as corn, sorghum, or cotton, avoiding cool-season vegetables. However, this practice has shown limited effectiveness in controlling the *Sclerotinia* blight because the sclerotia are able to remain viable for as many as 4 years in the soil (Partridge et al. 2006, Shew 2011). In addition, many winter annual weed species serve as hosts for *S. minor* during winter fallow, potentially reducing the benefits of crop rotation (Hollowell et al. 2003a). Hence, it is imperative that sanitation by way of weed control be practiced as one of the disease management strategies. Planting density influences the disease incidence and severity and could become a useful aid in disease management depending on the cultivars used (Maas et al. 2006). To reduce the spread of sclerotia of infested farm from one location to another by contaminated soil and plant debris lodged in farm equipment and shoes, it is recommended that the infested items be removed from these sites and be immersed in 6% sodium hypochlorite (NaOCl) solution

for 60 min. Sodium hypochlorite is proved to be lethal to bare sclerotia when immersed in its solution of 6% for 5 min or 3% for 10 min (Wilson et al. 2010).

Biological Control

Among the antagonistic fungi parasitizing sclerotia of *Sclerotinia* spp., *Coniothyrium minitans* has been identified to have great potential in the management of *Sclerotinia* blight of peanuts caused by apothecium-producing *S. sclerotiorum*. The effects have been shown to be long term (Partridge et al. 2006, Whipps et al. 2008). A spore suspension of *C. minitans* applied across field crops infected by *S. sclerotiorum* at the first appearance of apothecia results in a reduced population of sclerotia in soil at the end of a 7-year period even when susceptible crops are planted. However, less information is known about the effectiveness of *C. minitans* on *S. minor*. If *C. minitans* is active on *S. minor*, as has been found in some studies, then it may provide a way to reduce sclerotial numbers in peanut fields heavily infested by *S. minor*, thereby reducing losses. However, more studies are needed to confirm the effectiveness of *C. minitans* against *S. minor* causing *Sclerotinia* blight of peanuts. The possibility of some endophytic and epiphytic bacteria as potential bacterial antagonists against some soilborne pathogens of peanuts including *S. sclerotiorum* has been evaluated (Tonelli et al. 2010).

CYLINDROCLADIUM BLACK ROT

Symptoms

Under field conditions, the affected plants show chlorosis and wilting. Such plants also exhibit blighting of the leaf tips and margins. The lateral foliage is usually less affected than the erect primary branches. On artificial inoculation, circular brown spots of 0.5–1 mm diameter appear on leaves. The spots are surrounded by chlorotic halos that measure up to 2 mm in diameter. The hypocotyls and taproots become necrotic and black, with necrosis terminating at the soil level. The tips of the lateral roots and taproots are sloughed off, leaving short dark-brown to black fragmented stubs. Adventitious roots often develop on diseased plants at the soil level. Dark-brown to black and slightly sunken lesions appear on the pegs and pods, but the size of the lesions on pods is larger. The distinguishing feature of the disease is the presence of numerous orange-red perithecia at the base of the stems at the soil level or on the pegs and pods under the soil.

Geographical Distribution and Losses

The causal fungus is found in tropical and subtropical regions, and it is thought the fungus was introduced from Asia during the establishment of a tea plantation in coastal Georgia in the 1950s. Bell and Sobers (1966) first observed the occurrence of *Cylindrocladium* black rot (CBR) of peanuts in Georgia in 1965. Since then, this disease has been reported in all peanut-producing areas of the southeastern United States (Kucharek et al. 2000, Wheeler and Black 2005) and in Japan, India, and Australia (Kolte 1984). The disease has been a cause of major concern, particularly in Virginia and North Carolina because of its widespread occurrence and chronic threat to peanut production (Branch and Brenneman 2003). In other parts of the United States, outbreaks of CBR have remained static, and yield losses are generally of secondary importance to other soilborne diseases. The first report of its occurrence with 50% incidence from the Guangdong Province in China has been made in 2008 (Pan et al. 2009) followed by another first report of its occurrence from the Jiangxi Province in China in 2012 (Gai et al. 2012). Depending on the age of plant at the time of infection, the effect of the disease can be devastating. When the underground parts of the plants are destroyed due to the disease, the loss in pod yield and mature kernels may occur up to 50% and 30%, respectively.

Pathogen: Anamorph, *Cylindrocladium parasiticum* Crous, Wingfield & Alfenas (teleomorph, *Calonectria ilicicola* Boedijn & Reitsma)

Anamorph: *C. parasiticum* Crous, Wingfield & Alfenas (teleomorph, *C. ilicicola* Boedijn & Reitsma). It is an ascomycete in the Sordariomycetes group. The fungus is homothallic and produces orange-red subglobose to oval or obviate perithecia, 300–500 × 290–370 µm; asci are hyaline, clavate, long-stalked, and thin walled, contain eight ascospores, and measure 95–138 × 13–19 µm; ascospores are hyaline, granular, and fusoid to falcate, with one to three septa, slightly constricted at the medium septum, and measure 34–58 × 6.3–7.8 µm; conidia are cylindrical and hyaline, with one to three septa, are produced by apical budding, and measure 58–107 × 4–7 µm; conidiophores bearing stipes appear at right angles from the host.

The fungus grows well on potato dextrose agar and produces light, gray-to-white web-like aerial mycelium. It can produce conidia, ascospores, and microsclerotia in culture and infected plants. The microsclerotia vary in size with an average size of 52.7 × 88.4 µm. A selective medium named as sucrose-QT medium has been developed for isolation and inoculum quantification of *C. parasiticum* (Griffin 1977).

Epidemiology and Disease Cycle

The pathogen survives through microsclerotia produced in the roots of affected plants. They are produced abundantly in cortical tissues and *Rhizobium* nodules of infected peanut roots. After disintegration of the affected roots, the microsclerotia are released into soil and spread locally during cultivation and aqueous runoff. Soil movement on equipment can redistribute microsclerotia within and between fields. Plant debris blown into the air during harvesting and combining can be carried by wind to other fields. Birds have been implicated in disseminating the pathogen by ingesting infected peanuts and depositing the microsclerotia in droppings. Conidia and ascospores are quite susceptible to desiccation, and these cannot remain viable to play any effective role in the survival. The primary infection mainly appears through surviving microsclerotia in soil. Inoculum levels as low as 10 microsclerotia/g of soil have been reported to initiate epidemics. The number of observed infections on roots and the level of symptom expression by plants are directly proportional to microsclerotial densities in soil. Soil temperatures of 20°C–25°C and moisture levels near field capacity are most conducive to infection and rot of peanut roots by *C. parasiticum*. Randall-Schadel et al. (2001) confirmed seed transmission of the pathogen and CBR-infected seed could also be a source of primary inoculum to initiate disease epidemics. Infection cushions are formed on the epidermis and complete colonization of the cortex by the mycelium occurs. Epidermal cells beneath the infection cushion die and the necrosis of the surrounding cortical cell appears, suggesting the involvement of phytotoxins in pathogenesis. Fibrous roots emerging from the taproot of the peanut plant disrupt the protective periderm, which subsequently provides favorable courts of infection. Evidence for the occurrence of multiple root infection is reported, but it appears that only a portion of the multiple infection contributes appreciably to disease development. Since conidia are formed infrequently in nature, besides being less viable, the possibility of secondary infection through conidia is eliminated. Perithecia are formed on peanut stems after the establishment of infection in large quantities if adequate moisture is available. Mature ascospores subsequently develop and are discharged between 20°C and 30°C and maximally at 25°C more or less coinciding with vegetative growth. Ascospore formation and discharge appear to be controlled by day–night relative humidity fluctuations and can be dispersed by rain splash and runoff and appear to play a significant role in secondary disease spread within a growing season. All legumes are susceptible to *C. parasiticum*. The incidence of CBR on peanuts increases when soybean is included in the crop rotation sequence (Jordan et al. 2008). Nonleguminous crops such as cotton and tobacco are also known to be susceptible, but infection of these plants does not increase the inoculum in soil. The root-knot

nematode species, *Meloidogyne hapla* and *M. arenaria*, have been reported to interact with the fungus enhancing the disease severity and incidence on both susceptible and resistant varieties of peanuts. For example, root-knot (*M. arenaria*)-resistant peanut genotype C724-19-15 can withstand the interaction effect of infection due to *C. parasiticum* and *M. arenaria* when both pathogens are inoculated simultaneously, but other root-knot-resistant peanut genotypes such as C724-19-25 and Georgia-02C show mortality under similar conditions (Dong et al. 2009). Because of its sexual stage and wide host range, there is a possibility of the development of physiological races of *C. parasiticum*. But currently *C. parasiticum* appears to consist of a genetically homogeneous population with mainly clonal reproduction or inbreeding contributing to the population genetic structure. This is because of absence of random mating due to homothallic nature of *C. parasiticum* as well as the clonality of the population (Wright et al. 2010).

Disease Management

Host Plant Resistance

Use of resistant varieties appears to be more useful. In general, Spanish and runner-type varieties are most resistant to CBR (Dong et al. 2008). Some of the most resistant runner-type peanut genotypes are Georgia-06G, Georgia-07W, Georgia-02C, and Carver (Branch and Brenneman 2012). A breeding peanut line, 90x7-1-5-1-b2-B, has been developed to be resistant to the CBR and tomato-spotted wilt virus (Kucharek et al. 2000). Argentine and NC 3033 varieties grow and survive in soil having as high as 1000 microsclerotia/g soil, whereas only 0.5 microsclerotia/g soil is sufficient to cause severe disease in susceptible varieties. Histological study of the nature of resistance in peanut genotypes suggests that in response to infection in resistant genotypes, additional effective periderm is formed, which sloughs an entire quadrant of infected taproot.

Chemical Control

Several broad spectrum chemical fumigants such as Vapam and Vorlex 201 have been evaluated as preplant (4 weeks before planting) treatments, and other chemicals such as captafol, quintozene, chloroneb, tebuconazole (Kucharek et al. 2000), and gypsum have been evaluated for the management of the disease, which appears to be less useful. The soil fumigant, metam sodium, was first recognized in 1981 to have commercial value for the control of CBR in Virginia. Following applications into soil, metam sodium converts rapidly to methyl isothiocyanate (MIT), which is the active ingredient. MIT is a highly toxic, broad spectrum biocide. Because of its highly toxic and nonspecific nature, MIT should be applied at least 2 weeks prior to planting to avoid crop injury. The severity of CBR increases when roots are parasitized by the northern root-knot nematode (*M. hapla*) and the ring nematode (*Criconemella ornata*). Metam sodium treatment along with the application of Temik 15G at 5–7 lb/A in the seed furrow provides good control of these nematodes as well as CBR.

Cultural Control

Very little is known about methods of control of the disease through cultural practices. It is, however, reported that corn and small grains are highly resistant to the disease, and these should be useful as rotational crops to check the incidence of the disease in peanuts. But cotton can be a better rotation crop than corn with respect to peanut yield and gross economic return (Jordan et al. 2002). Well-drained fields should be used for peanut production, and to prevent soil movement, farm equipment should be thoroughly cleaned prior to field-to-field transport. The harvest of peanut seeds in areas of fields with high incidence of CBR should be avoided. This measure is expected to lower the number of speckled seeds entering commercial seed lots and reduce the risk for the spread of CBR (Glenn et al. 2003).

PEG AND POD ROTS

Symptoms

No apparent symptoms of pod rot appear above-ground except that plants with severe pod rot may flower profusely and appear abnormally dark green in color late in the season. Below ground, symptoms of pod rot caused by *Rhizoctonia* and *Pythium* are difficult to distinguish because both pathogens are often present. Light-brown areas develop on pods that later turn dark brown or black (Figure 2.10). A few to nearly all of the pods on one plant may be affected. *Pythium* usually causes a black, watery rot. Pods rotted by *Rhizoctonia* have a firm brown decay, and the seeds and inner pod wall may be lined with a cream-colored fungus. The seeds within rotted pods are usually completely decayed or severely damaged.

Geographical Distribution and Losses

Peanut plants with a few rotted pods are found in most fields all over the peanut-growing countries in the world. Severe outbreaks of pod rot are less common, but can be devastating where they occur. Pod rot tends to be severe in sandy soils and in fields intensively cropped to peanuts. Peanut pod rot primarily caused by *P. myriotylum* is one of the most important diseases in the pacific coast region of Cosiguina in Nicaragua (Augusto et al. 2010a,b). *Neocosmospora striata* Udagawa & Y. Horie causing peanut pod rot to the extent of 90% incidence in the Old Yellow River drainage area (Sun et al. 2012a) and *Neocosmospora vasinfecta* E. F. Smith causing similar pod rot disease in peanuts to the extent of 30%–100% incidence in the Sha River drainage area (Sun et al. 2012b) have been reported as first reports of occurrences in China. Depending on the geographical location, the losses in yield due to rotting of the pods might vary in the range of 5%–50%.

FIGURE 2.10 Pod rot complex of peanuts caused by fungi.

Pathogens: Complex of Fungi in Preharvest Peg and Pod Rots

There are several types of pod-rotting fungi. The specific pod rots could be caused by fungi such as *Pythium* spp., especially *P. myriotylum*; *R. solani*; *Fusarium* spp., especially *F. solani*; *Verticillium albo-atrum*; and *Botrytis cinerea*. Recently, two species of fungi, namely, *N. striata* and *N. vasinfecta*, have been reported as the causes of peanut pod rots (Sun et al. 2012a,b). Rotting of pegs and pods caused by *S. rolfsii*, *M. phaseolina*, *S. sclerotiorum*, and *C. parasiticum* has been described in the preceding pages. The fungi that cause pod rot are normally found at some level in most peanut soils. Most grow on the above-ground parts of the plant also. *Pythium* is the exception as it only grows below ground. Advanced stages of *Pythium* and *Rhizoctonia* pod rots result in complete decay of the pod and kernels. In most peanut-growing areas, *Rhizoctonia* is the main problem in terms of total acreage. On a field-by-field basis, *Sclerotinia* is the most devastating, but it is not so wide spread. Nematode (*Pratylenchus brachyurus*) and black hull fungus (*Thielaviopsis basicola*) also cause pod discoloration, but the decay is usually superficial.

Epidemiology and Disease Cycle

A complex of factors in addition to the fungi is probably responsible for these severe outbreaks of pod rots. These factors include excessive soil moisture, wide fluctuations in soil moisture, calcium deficiency, insect and nematode feeding, and irrigation with poor-quality (salty) water (Choppakatla et al. 2008). One very important factor among these is low level of available calcium in soil, especially in large-seeded cultivars. Pod surface area and surface to volume ratio are important in determining the quantity of calcium in seeds, and this explains why large-seeded cultivars are more sensitive to calcium deficiency–induced susceptibility to pod rots caused by a complex of fungi (Augusto et al. 2010a). Sudden increase in maturity of kernels and concomitant development of pale testa kernels influence more pod rots. The increase in pod rot due to *Fusarium oxysporum* and *F. solani* later in the season is associated with pale testa kernels found in pods, which to the unaided eye appear healthy. Early defoliation of plants hastens maturity and causes peg breaking. Thus, any disease or insect injury resulting in leaf shedding will increase the incidence of rotting of pegs and pods. For example, under Malawi conditions, the increase in pod rot is associated with higher incidence of ELS and LLS and senescence (Kolte 1984). A long wet season and wetness of the top soil increase the incidence of pod rot caused by *P. myriotylum* particularly in sandy well-aerated soil under Israel conditions. Frequent sprinkling of sandy Israeli soil encourages infection of pods by *P. myriotylum*. Application of fertilizers in midseason increases the amount of succulent tissue, favoring more pod breakdown by *P. myriotylum*. While nitrogen amendments with nitrogenous compounds may improve plant health, an overabundance of nitrogen may cause an increase in disease such as *Rhizoctonia* pod rot (Thiessen and Woodward 2012). Several instances of definite interactions taking place between insect or nematode injury and invasion of pods by *P. myriotylum* have been reported. Feeding sites of corn root worm, *Diabrotica undecimpunctata*, and of a mite, *Caloglyphus michaeli*, on peanut pods favor the entry of *P. myriotylum* resulting in the development of rotting of pods. Interaction studies between *P. myriotylum*, *F. solani*, and *M. arenaria* revealed that *P. myriotylum* interacts synergistically with *F. solani* and *M. arenaria*, but not with *R. solani* in causing pod rots of peanuts. *F. solani* alone has been found to predispose the pods to pathogenic activity of *P. myriotylum*.

Disease Management

Host Resistance

Pod rot management through host resistance may be effective but is dependent on the identification of the causal agent. Spanish cultivars, especially Toalson, may provide resistance to both *Pythium* spp. and *R. solani*. Partial resistance to *R. solani* has also been identified in the runner peanut Georgia Browne. Resistance to *Sclerotinia* has been shown in the varieties Virginia 81B, Virginia 93B,

Tamspan 90 and Southwest Runner, and Tamrun OL 07 (Thiessen and Woodward 2012). Some of the peanut varieties have been found to possess field resistance to pod rots (Gopal 2003, Krishnakanth et al. 2005, Gopal et al. 2006c). Resistance in *Sehwar Z-21* and *TMV-2* is attributed to the shortness of the gynophore (inherited independently) enabling the development of pods in a shallow layer of soil and subsequent escaping of the attack by *P. myriotylum*. The phenotype of pod is positively correlated with pod rot severity (Yang et al. 2002). For example, in the *Amani* variety, resistance to pod rot is related to the thicker shell of the pods. It is thus seen that efforts to breed varieties with a shorter gynophore and thick pod shell be encouraged in order to combat losses due to pod rots.

Chemical Control

Some of the fungicides such as Vitavax, thiram, Rizolex T and Topsin M 70 (El-Deeb et al. 2002), mefenoxam, azoxystrobin (Augusto et al. 2010b), and quintozene when applied to the soil before planting or at the fruiting zone at the flowering time give good control of pod rots. The use of metalaxyl + quintozene (PCNB) or metalaxyl + tolclofos-methyl is useful in controlling pod rot diseases (*Pythium* and *Rhizoctonia* spp.) of peanuts (Filonow and Jackson 1989).

Cultural Control

Since pod rot is caused by a complex of several fungi, cultural control measures assume special significance in the management of the pod rot diseases of peanuts. Planting of peanuts in infested soil should be avoided. Care should be taken to avoid mechanical injury to the developing peg and pods during interculture operations. Defoliation of plants due to leaf spot diseases and insect pests should be controlled to check rotting of the pods. Infrequent irrigation resulting in drying out of the top soil brings about reduction in the incidence of pod rot under Israel conditions. The pod rots can be brought under control by mixing gypsum with the upper 15 cm soil layer at the peak of flowering period. The gypsum reduces the soil inoculum and increases calcium contents of pods resisting invasion of pods by fungi (Kolte 1997).

Biological Control

Experimental evidence indicates that infection of fungi on peanut pods can be checked by certain formulation (e.g., plant guard) of antagonistic microorganisms particularly *T. harzianum* (Kanth et al. 2000, El-Deeb et al. 2002). Endomycorrhizal fungus, *Glomus mosseae*, has been proved to be of potential usefulness in protecting the peanut plants from infection by pod rot fungal pathogens (Abdalla and Abdel-Fattah 2000). But the possibility of actual use of such method over large area has not been investigated.

PROBLEM OF AFLATOXIN CAUSES

Geographical Distribution and Losses

In 1960, in Britain, widespread fatalities occurred in poultry fed on peanut meal that had been imported from Brazil, India, and African countries to Britain, the source of toxin being traced to *A. flavus*. Attempts to isolate any microorganism from the peanut meal resulted in isolation of the fungus, *A. flavus*, which was capable of producing four closely related toxins. The toxins were named as aflatoxins (*A. flavus* toxins) identifying their generic origin. Such toxins were isolated from the imported peanut meal also. Thus, aflatoxins were established as the cause of *Turkey X* disease. Species of *Aspergillus* are normal components of soil microflora, and the principal producers of aflatoxins are the species of *A. flavus* group including *A. parasiticus*. *A. flavus* is the most common species in Africa and Asia, while *A. parasiticus* is predominant in America. In South Africa, *A. parasiticus* is predominant and is associated at twice the frequency of *A. flavus* (Ncube et al. 2010). Aflatoxins are known to cause liver cirrhosis in livestock and humans and they possess potent carcinogenic properties. Aflatoxin contamination in peanuts is a serious and worldwide

problem especially in developing countries concerning food safety and human health (Williams et al. 2004, Alwakeel and Nasser 2011). Aflatoxins can result in serious economic hardships to producers and adverse health impacts in both humans and domestic animals (Duran et al. 2009). Aflatoxin contamination costs the U.S. peanut industry over $20 million annually (Holbrook et al. 2009). It is reported to be more serious in the southern parts of China and considered to be a crucial factor affecting sustainable development of peanut industry in that country (Liao et al. 2009). Similar is the situation in India where aflatoxins are found in a very high range of 1400–3600 μg/kg of peanut cake. Therefore, aflatoxin contamination is also of significance in relation to public health and export. Aflatoxin levels are higher than the minimum parts per billion (ppb) permitted level set by the Food and Drug Administration in the United States (20 ppb), the European Union (6 ppb), the Department of Health in South Africa (10 ppb), and the Brazilian Regulatory Authorities in Brazil (20 μg/kg) for peanuts that are intended for direct human consumption (Oliveira et al. 2009). Interestingly, aflatoxin levels of up to 131–160 ppb are reported to be present in peanuts produced in the three northern provinces KwaZulu-Natal, Mpumalanga, and Limpopo in South Africa (Ncube et al. 2010). Peanuts stored and consumed in rural areas in Mali (West Africa) have been observed to be highly contaminated by *A. flavus* and aflatoxin B1, with average rates of aflatoxin B1 significantly above the accepted international standards (Passone et al. 2005, Soler et al. 2010). *A. flavus* population in soils from the peanut-growing regions in Argentina (South America) indicates prevalence of three strains (sclerotium-forming S and L and nonsclerotial strains). The S strains produce higher mycotoxin levels than the L and nonsclerotial strains, and about 10% of the S strains simultaneously produce aflatoxins B and G and cyclopiazonic acid. These strains are of great concern in food safety as there is a higher probability of aflatoxin contamination in peanuts in Argentina (Barros et al. 2005, 2006, Nesci et al. 2011). Prevalence of such similar strains of *A. flavus* is also reported from peanut soils in Iran (Amani et al. 2012) and India (Raina and Desai 2006). Consequently, aflatoxin contamination of peanuts becomes one of the most important constraints to peanut production in many countries and becomes a crucial factor in restricting the export of peanuts from one country to another (Asis et al. 2009, Xie et al. 2009). Not only animals but also plants are susceptible to aflatoxins (El-Khadem 1968). Most countries/institutions in Asia and Africa give high priority to research on the peanut aflatoxin problem (Wynne et al. 1991, Waliyar 1997, Zobia et al. 2012), and there appears to be the need for aflatoxin awareness campaigns and management programs to be implemented in rural areas in most countries of Asia and Africa.

Chemical Nature of Aflatoxins

Hartley et al. (1963) were the first to successfully isolate and characterize four closely related furanocoumarin compounds, which have been designated as aflatoxins B_1, B_2, G_1, and G_2. Four more aflatoxins are also known, and these are designated as M_1, M_2, B_2a, and G_2a. The structural formulae of different aflatoxins are given in Figure 2.11. The aflatoxins B and G give a characteristic blue and green fluorescence, respectively, under ultraviolet light.

Invasion of Peanut Pods and Kernels and Conditions for Aflatoxin Production.

As a rule, the seeds are free of *A. flavus* at maturity. However, invasion of peanut seeds by aflatoxigenic strains of *A. flavus* and subsequent aflatoxin contamination can occur both before and after harvest especially during drought stress situation. Colonization of seeds by *A. flavus* increases after maturity and during the period between removal from soil and drying. It is observed that conidia of *A. flavus* do not germinate in peg geocarposphere and germination of conidia occurs only in traces in the fruit geocarposphere. This is possibly because of the presence of volatile and nonvolatile fungistatic substances and increased competition from other soil microorganisms in the peanut geocarposphere. What has been observed is that injury of peanut pods, due to growth cracks, mechanical agents, or biological agents such as root-knot nematode (*M. arenaria*) (Timper et al. 2001, 2004), pod burrower bug (*Pangaeus bilineatus*) (Chapin et al. 2004), and insect pest particularly *Tribolium confusum* in storage (Mohale et al. 2010), predisposes the peanut pods to colonization by *A. flavus* or the development of

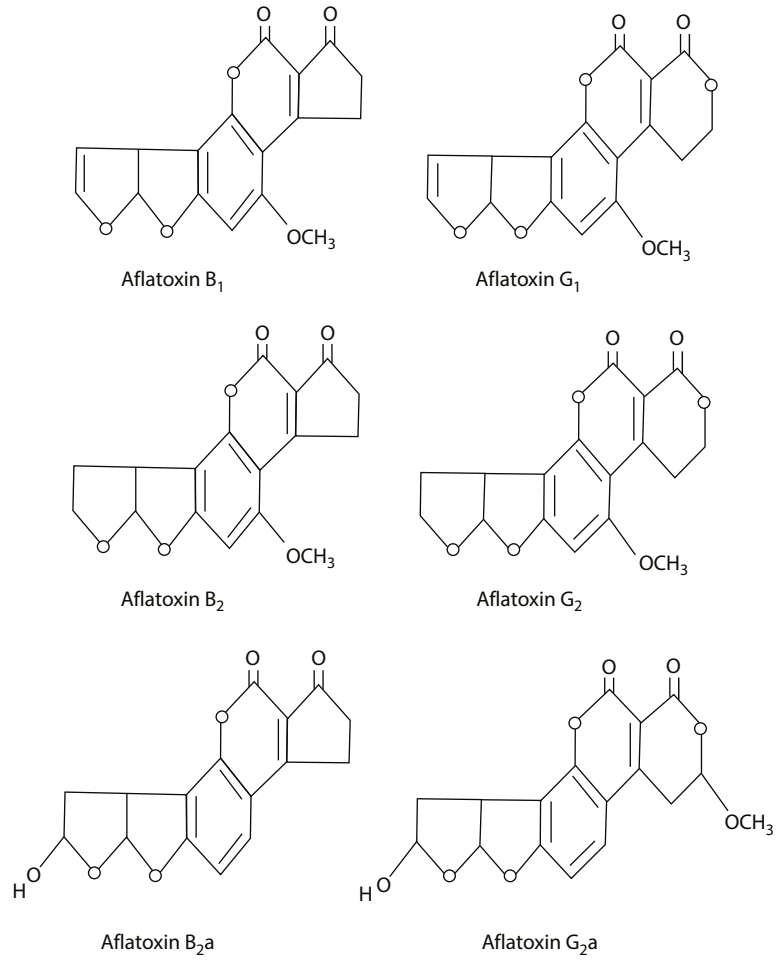

FIGURE 2.11 The structural formulae of different aflatoxins. (From Kolte, S.J., *Diseases of Annual Edible Oilseed Crops*, Vol. I, Peanut Diseases, CRC Press, Boca Raton, FL, 1984.)

aflatoxin in kernels. Following injury to pods or kernels, certain carbon (glucose) and nitrogen substrates (amino acids) are exuded that support the conidial germination of *A. flavus*, enabling it to invade the damaged pods and kernels. Low levels of colonization of peanut fruits by *A. flavus* via flower and aerial peg colonization appear to be possible under field conditions (Kolte 1984). Cropping system appears to have an influence on *A. flavus* infection and accumulation of aflatoxin in the peanut (Kumar et al. 2008). Peanuts harvested from lands planted with peanuts during the previous season are infested with more fungi and contain more aflatoxin than in the case of peanuts raised on lands planted with rye, oats, etc., as the previous crops. Population dynamics of *A. flavus* in soil is also reported to be dependent on the varieties of peanut used in cultivation (Yang and Ma 2003), and its density and genetic diversity based on analysis of vegetative compatibility group (VCG) can vary greatly among regions and fields used for cultivation (McAlpin et al. 2002, Barros et al. 2003, 2006, Horn 2006). Molecular characterization reveals that each VCG represents a single isolate that produces unique DNA fingerprints. This has, therefore, been useful to identify isolates of toxigenic potentials and/or VCG affiliations (McAlpin et al. 2002, Victoria Novas and Cabral 2002, Chen et al. 2002, 2005, Pildain et al. 2004, 2005, Barros et al. 2005, 2007, Reis et al. 2012). Population of *A. flavus* is low in summer crop than in rainy season crop, and its population increases toward pod development stage, and aflatoxin production is negatively related with relative water content, pod wall integrity, and moisture content at harvest (Thakur

et al. 2003a,b, Sudhakar et al. 2007). Infection and aflatoxin concentration can be related to the soil types, alfisol soils being more conducive than vertisols, and to the occurrence of soil moisture stress during pod filling when soil temperatures are near optimal for *A. flavus*. Average soil temperatures of 28°C–34°C are favorable for aflatoxin contamination (Craufurd et al. 2006). Thus, late-season drought stress is the most important factor for *A. flavus* invasion and aflatoxin contamination. Peanuts invaded by aflatoxigenic strains of *A. flavus* in the soil before harvest can lead to a serious contamination during drying and storage, and the postharvest drying conditions tend to influence the degree of seed infection and aflatoxin contamination. Delayed harvesting also leads to increased fungal invasion. *A. flavus* grows best between 10°C and 45°C temperature and at relative humidity of 75% or more. The optimum aflatoxin production, however, takes place at 25°C–30°C temperature at 80%–85% relative humidity. Peanut seeds containing more than 9% moisture are likely to be affected by *A. flavus*, and moisture content of seeds only up to 30% is favorable for aflatoxin production. Prematurely dried pods and rainfall at the time of harvesting favor production of aflatoxin during storage (Kolte 1984).

Aflatoxin Management

Host Plant Resistance

a. In cultivated *Arachis* species: Efforts on the development of screening techniques especially at ICRISAT Center for resistance to *A. flavus* infection and aflatoxin production have led to the foundations for conventional resistance breeding program that has resulted in the identification of accessions (genotypes) and development of breeding lines that have resistance to seed infection by *A. flavus* and low aflatoxin production relative to standard susceptible control cultivars in several peanut-growing countries (Jiang et al. 2005, Ntare et al. 2006, Holbrook et al. 2000, 2008, Nigam et al. 2009). Table 2.3 gives the list of peanut genotypes/lines from different countries that show seed infection and colonization equal to or less than most commonly used standard resistant control peanut genotypes *J 11* from India. Some such genotypes show stability of resistance and high yield across seasons and in multilocation environments. For example, three lines (ICGVs 87084, 87094, and 87110), bred at ICRISAT Center in India for resistance to seed infection, had also been found to be resistant in Niger, Senegal, and Burkina Faso in West Africa (Waliyar et al. 1994). Several land races (local germplasm lines) in China have been identified as resistant to seed infection by *A. flavus* and aflatoxin production (Liao et al. 2009). The resistance of peanut seeds to *A. flavus* and aflatoxin production is associated with certain morphological and biochemical characteristics, namely, structure of seed coat, size of wax layer, junction between epidermal cells, thickness of cell wall, and presence of cracks. Resistance depends upon the intact and undamaged testa. So, protective role of seed testa has been emphasized in case of selection for resistance to seed colonization by aflatoxigenic isolates of *A. flavus* (Asis et al. 2005, Lei et al. 2006, Liang et al. 2009). Resistance to peanut seed infection by *A. flavus* is associated with higher content or higher activity of some biochemical constituents such as resveratrol (Liang et al. 2006a,b, Wang et al. 2012), lipoxygenase (LOX) (Liang et al. 2002, Tsitsigiannis et al. 2005, Kumari et al. 2012), β-1,3-glucanase (Liang et al. 2005), oleic acid (Jiang et al. 2006, Ebrahimi et al. 2009), trypsin inhibitor (Liang et al. 2003), superoxide radical generation (Liu et al. 2012, Zhou et al. 2012), storage protein including proteinase inhibitor (Yan et al. 2012b), hydrogen peroxide (H_2O_2), malondialdehyde accumulation (Zhou et al. 2012), and high total phenols (Kumar et al. 2002, Latha et al. 2007, Kumari et al. 2011). In contrast to the preceding report on oleic acid, high-oleic peanut lines are reported to have nearly twice as much aflatoxin as normal lines (Xue et al. 2003). Some accessions have been identified that exhibit low preharvest aflatoxin contamination (PAC) in multiple environments that are tolerant to drought stress conditions (Cleveland et al. 2003, Wang et al. 2004, Liang et al. 2006b, Arunyanark et al. 2010, 2012, Girdthai et al. 2010a,b). Some such genotypes are J 11, 55-437, and PI 337394F, HY 22 (Liu et al. 2012). Traits related to efficient ability to nitrogen fixation under drought conditions may be used as indirect selection criteria for

TABLE 2.3
Peanut Genotypes Resistant (R) or Moderately Resistant (MR) to *A. flavus* Infection and Aflatoxin Contamination as Reported from Different Countries in the World

Genotype	Country	R/MR	Reference(s)
Tifguard	United States	R (also possesses resistance to root-knot nematode and TSWV)	Holbrook et al. (2009)
Minhua 6, Kanghuang 1, Yueyou 9 (released as *A. flavus*–resistant cultivars)	China	R	Zhuang et al. (2007)
ICGVs 86590, 89104, 94350, 99029; IC 48, ICGS-76	India	R (also shows resistance to drought)	Sudhakar et al. (2007)
J 11	India	R	Kumari et al. (2012)
J 11, PI 337394, PI 337409; breeding lines Manfredi 68, Colorado Irradiado, and Florman INTA	Argentina	R/MR	Asis et al. (2005)
9843-26-2, 9817-36-2	China	R	Chen et al. (2005a)
S230, R8808, ICGV 86590, Spanish improved, mutant 28-2	India	R	Harish Babu et al. (2004a)
Significantly superior to J 11 genotypes, ICGV 86155, ICGV 86699, and ICGV 96266; comparable to J 11 genotypes, ICGV 96262, ICG 1697, and R 9227	India	R	Harish Babu et al. (2004b)
ICGV 86590, ICGV 93280, ICGV 95322 (tolerant to end-of-season drought)	Indonesia	R/MR	Rahmianna et al. (2004)
H2030, H2060, H2063, H2095 (tolerant to end-of-season drought)	China	R	Wang et al. (2004)
S206, KRG1, GPBD-4	India	MR	Varma et al. (2001)
KB 153 (high content of storage protein + proteinase inhibitor)	China	R	Yan et al. (2012a)
ICG 12625	China	R (to aflatoxin production)	Jiang et al. (2010)
ICG 4750	China	R (to invasion)	Jiang et al. (2010)
G 845, G 8	China	R	Jiang et al. (2006)
GT-YY 9, GT-YY 20	United States	R (to *A. flavus*)	Liang et al. (2005)
EF 7284	China	MR	Jiang et al. (2002)
J 11, HY 22	China	R	Liu et al. (2012)
J 11, IC 48, ICGV 89104, ICGS-76	India	R	Latha et al. (2007)
Xiaohongmao (high oleic acid content and small seed size; bacterial wilt resistant)	China	R	Liao et al. (2003)
Taishan Zhenzhu, 93-76	China	R (to aflatoxin production)	Liao et al. (2003)

resistance to aflatoxin production in peanuts (Arunyanark et al. 2012). Resistance to *A. flavus* infection in peanuts is independently attributed to three genes: (1) ARAhPR10 (Xie et al. 2009, 2013), (2) PnLOX2 (Yan et al. 2012a), and (3) $PnAG_3$ (Liu et al. 2012). The $PnAG_3$ gene has been found to be expressed more prominently in *A. flavus*–resistant genotypes than susceptible ones under drought stress conditions (Liu et al. 2012). LOXs are nonheme, nonsulfur iron dioxygenases and are encoded by a multigene family and widely distributed in higher plants. Its metabolic products as jasmonic acid, SA, etc., are anti-insect or antibiotic active substances in which active oxygen radicals can destroy cytomembrane and inhibit fungus and aflatoxin generation. Certain agronomic traits as associated with resistance to *A. flavus* in peanuts are valuable. About 13 common loci (63 alleles having increasing effect) are found to be associated with both agronomic traits and resistance to *A. flavus* (Huang et al. 2012). Microanalysis of resistant and susceptible peanut cultivars infected with *A. flavus* (or with *A. parasiticus*) has resulted in

the identification of 62 genes in resistant cultivars that are upexpressed in response to *A. flavus* infection, whereas 22 putative *Aspergillus* resistance genes have been identified to be constitutively upexpressed in resistant cultivars in comparison to the susceptible ones (Guo et al. 2009, 2011). These sources among others have been used in breeding programs, and several lines have been derived to possess resistance and produce high yield. The most promising breeding lines developed at ICRISAT reported to be resistant to seed infection and colonization are ICGVs 87084, 87094, 87110, 91278, and 91284. These sources of resistance to PAC have been crossed with cultivars and breeding lines that have high yield and acceptable grade and resistance to TSWV and root-knot nematode (*M. arenaria*). One such first exemplary high-yielding PAC-resistant peanut cultivar is *Tifguard*, which is also resistant to TSWV and root-knot nematode, has been released in the United States (Holbrook et al. 2009). More such resistant cultivars adapted to different production systems need to be developed to meet the requirements of producers and users. The levels of resistance could be improved further by pyramiding resistance genes from different and diverse sources. Liang et al. (2009) have reviewed the peanut host resistance mechanisms to aflatoxin contamination and suggested functional genomics approach as a valuable tool to understand the comprehensive mechanism of the resistance pathways.

b. In wild *Arachis* species: *Arachis chiquitana* has been identified as one of the few wild species of *Arachis* showing resistance to *A. flavus* colonization, and initial seed screening of *A. chiquitana* for *A. flavus* has shown promise of obtaining hybrids resistant to *A. flavus* colonization, though further studies remain to be done whether interspecific hybrid so obtained is also resistant to aflatoxin production (Mallikarjuna 2005).

c. Molecular breeding and transgenic peanuts for resistance to aflatoxin production: It is expected that transgenic resistance against *A. flavus* (or *A. parasiticus*) infection and aflatoxin production in combination with conventional breeding may lead to the development of agronomically superior peanuts that are free of aflatoxin contamination (Ozias-Akins et al. 2002, Nigam et al. 2009). For example, a nonheme chloroperoxidase gene (cpo-p) from *Pseudomonas pyrrocinia*, a growth inhibitor of mycotoxin-producing fungi, has been introduced into peanuts by particle bombardment method. Such transgenic peanuts show inhibition of *A. flavus* hyphal growth that could be translated to a reduction in aflatoxin contamination (Niu et al. 2009). Transgenic peanut lines developed by Xie et al. (2013) overexpressing the effects of ARAhPR10 gene have been established to play an important role in peanut host resistance to *A. flavus* infection and alleviation of aflatoxin production in peanuts. Besides this, the recourse to biotechnology, through modification of the aflatoxin biosynthesis pathway, or the use of variants of hydrolytic enzymes (chitinases and glucanases) to provide transgenic protection to peanuts against infection by aflatoxin-producing fungi may help in obtaining peanuts free from aflatoxin. For example, at ICRISAT, transgenic peanut lines with aflatoxin resistance conferred by rice chitinase gene have been developed and characterized (Sharma et al. 2006).

Chemical Control

Aflatoxin production during storage can be prevented by treating peanut pods with aureofungin. Penetration and invasion by *A. flavus* on pods and kernels can also be prevented by spraying chemicals like propionic acid (5%), sorbic acid (0.1%), and chlorothalonil (0.15%) on pods of peanut plants (harvested at proper stage of maturity) inverted in windrows for drying under field conditions (Kolte 1984). Postharvest control of *A. flavus* infection and aflatoxin contamination can be obtained by the use of formulations of food-grade antioxidants such as butylated hydroxyanisole, butylated hydroxytoluene, and propylparaben (Passone et al. 2007, 2008, 2009). Methyl jasmonate treatment to peanut seeds promotes resistance of peanut plants to *A. flavus* infection and production of aflatoxin B1 (He et al. 2004).

Cultural Control

Several workers (Waliyar 1997, Kasno 2004, Rahmianna et al. 2007) have suggested measures to prevent damage to peanut pods and kernels during cultivation, harvesting, and storage conditions

for the prevention of infection due to *A. flavus* and aflatoxin production. Some of the important precautionary measures are (1) avoiding damage to plants and pods from soilborne diseases and during cultivation; (2) avoiding late-season drought stress by the manipulation of crop duration and supplementary irrigation; (3) harvesting and lifting the crop at optimum maturity; (4) discarding damaged pods; (5) drying pods to below 8% moisture content; (6) storing under clean, dry, and insect-free conditions; and (7) avoiding rewetting of pods/seeds during storage. Peanut seeds washed with table salt solution (Sumartini and Yusnawan 2005) and liming the soil have been reported to be useful in reducing the seed infection of *A. flavus* and aflatoxin production (Pereira and Rossetto 2008). Cultural control treatments such as soil application of FYM at 15 tons/ha + gypsum at 500 kg/ha when combined with *Trichoderma* soil application + soil drenching with mancozeb at 1000 ppm result in significant reduction of aflatoxin content (6.6 ppb) with reduced seed infection (13.3%) and maximum pod yield (Ramanaiah et al. 2008). The possibility of inhibition of aflatoxin B_1 production by irradiating the peanut seeds with gamma irradiation has been studied (Prado et al. 2005, Borges et al. 2007). *Musa paradisiaca* fruit peel can be used to suppress aflatoxin production (Sharma and Sharma 2011)

Biological Control

Application of Atoxigenic Strains of A. flavus *or* A. parasiticus

Aflatoxin contamination of peanuts in the field can be reduced by 77%–98% with biological control through the application of nontoxigenic strains of these species, which competitively exclude native aflatoxin-producing strains from developing peanuts (Cotty 1990, Horn et al. 2001, Barros et al. 2003, 2006, Horn and Dorner 2009, Yin et al. 2009). These technologies rely on application of highly competitive atoxigenic strains on solid nutritive substrates. They must be applied at a time and in a manner that allows successful competition with aflatoxin producers. Application timing and placement greatly influence the efficacy of these formulations in preventing aflatoxin contamination. The retention of conidia of atoxigenic strains in the upper soil layers is important in reducing aflatoxin contamination of peanuts (Horn et al. 2001). Proven atoxigenic strains can be routinely applied once per growing season at 11.2–22.4 kg/ha (10–20 lb/acre) of formulation. Reduction of aflatoxins in peanut seeds depends on both the density and the aflatoxin-producing potential of native populations and on the fungal strain used for biological control and nitrate-nonutilizing mutants, which can be used for evaluating the efficacy of biocontrol strains (Horn and Dorner 2009).

Application of Native Antagonistic Fungi and Bacteria

Antagonistic fungi, namely, *T. viride* (Thakur et al. 2003a,b), *T. harzianum* (Thakur et al. 2003a,b, El-Moneim et al. 2010), *Saccharomycopsis schoenii* and *S. crataegensis* (Prado et al. 2008), and *S. cerevisiae* (Prado et al. 2011), and antagonistic bacteria, namely, *B. subtilis* (El-Moneim et al. 2010), a strain of marine bacterium, *Bacillus megaterium* isolated from the Yellow Sea of East China (Kong et al. 2010), *Streptomyces* sp. strain ASBV-1 (Zucchi et al. 2008), and *Burkholderia* sp. strain TNAU-1 (Ayyathurai et al. 2010), have been found to be significantly effective in reducing the aflatoxin production in peanuts. The dose of 20 g of *T. harzianum* or *B. subtilis* formulation per kg of peanut pods and the seed treatment at 10 g/kg or soil application at 2.5 kg/ha at 30, 45, and 60 DAS with the formulation of *Burkholderia* sp. result in significant reduction in the infection by *A. flavus* and aflatoxin B_1 contamination in peanut kernels.

Effect of Plant Extracts

Garlic bulb extract (Sumartini and Yusnawan 2005, Bora et al. 2010), aqueous extract of leaves of lemon (Tewari et al. 2004), aqueous *Moringa* seed extract (Donli and Dauda 2003), and onion and neem extracts (Bora et al. 2010) have shown promise through peanut seed treatment in the control of peanut seed infection by *A. flavus* and aflatoxin production.

TABLE 2.4
Other Fungal Diseases of Peanuts

Disease	Pathogen	Geographical Distribution	Reference(s)
Aerial blight	Rhizoctonia solani (= *Thanatephorus cucumeris*)	India, Malaya, United States	Kolte (1984)
Alternaria leaf spot/ blight	*Alternaria tenuissima* (*Kunze ex. Pers.*) *Wilts*, *A. alternata (Fr.) Keissler*, *A. arachidis*	India, United States	Kolte (1984), Kumar et al. (2012)
Anthracnose	*Colletotrichum arachidis*, *C. dematium* (*Pers. and Fries) Grov. Sensuvona*	Argentina, India, Panama, Taiwan, Uganda, United States	Kolte (1984)
Black root rot	*Thielaviopsis basicola* (Berk and Berk) Ferr.	United States	Kolte (1984)
Botrytis blight	*Botrytis cinerea* (= *Botryotinia fuckeliana*)	Australia, Japan, Romania, Taiwan, Tanzania, United States, Venezuela, Zambia	Kolte (1984), Thiessen and Woodward (2012)
Brown blotch (of Bambara groundnut)	*Colletotrichum capsici*	Nigeria	Obagwu (2003a,b)
Bud blight	*Phoma glomerata*	India	Kolte (1984)
Chlorosis and tip spot	*Psedoplea trifolii*	Mauritius	Kolte (1984)
Choanephora leaf spot	*Choanephora* spp.	United States	Porter (1993)
Collar rot	*Lasiodiplodia theobromae* (= *Diplodia gossypina*)	United States	Porter (1993)
Diaporthe blight	*Diaporthe phaseolorum* var. *sojae* (Lehm.) Wehm.	United States	Kolte (1984)
Fusarium disease (wilt, dry stem rot)	*Fusarium oxysporum*, *F. oxysporum* var. *orthoceras*	Bulgaria	Vitanova (2003)
Fusarium wilt	*Fusarium martii phaseoli* Burkh	United States	Kolte (1984)
Leaf scorch	*Leptosphaerulina trifolii* (*Rost.*) *Pert.*	India, Malawi	Kolte (1984), Desai and Bagwan (2005)
Leaf spot	*Cristulariella pyramidalis Wat. and Marshall*	India, United States	Kolte (1984)
Leaf spot	*Macrophomina phaseolina*	India	Gupta and Kolte (1982)
Leaf blight	*Drechslera spicifera*	India	Jat et al. (2004)
Limb rot (foliar blight phase)	*Rhizoctonia solani*	United States	Thiessen and Woodward (2012)
Melanosis	*Stemphylium botryosum*		Porter (1993)
Myrothecium leaf blight	*Myrothecium roridum* Tode ex Fr.	India	Kolte (1984)
Muddy spot	*Ascochyta* sp.	Brazil	Kolte (1984)
Passalora leaf spot	*Passalora arachidicola* (= *Mycosphaerella arachidis*)	China	Zhang et al. (2010)
Pepper spot and scorch	*Leptosphaerulina crassiasca* (Sechet) Jackson and Bell (= *L. arachidicola* Ye, Chen, and Huang)	Argentina, India, United States	Kolte (1984)
Phomopsis stem blight	*Phomopsis* sp., *P. longicolla*	India, New Mexico (United States)	Kolte (1984), Sanogo and Etarock (2009)
Phyllosticta leaf spot	*Phyllosticta arachidis hypogaea* Vasant Rao	Argentina, India, Israel, Myanmar, Sudan, Taiwan, United States	Desai and Bagwan (2005), Kolte (1984)
Powdery mildew	*Erysiphe polygon*i DC	Mauritius	Kolte (1984)
	Oidium arachidis Chorin	Bulgaria, Israel	Kolte (1984)

(*Continued*)

TABLE 2.4 (*Continued*)
Other Fungal Diseases of Peanuts

Disease	Pathogen	Geographical Distribution	Reference(s)
Olpidium root rot	*Olpidium brassicae*	India	Porter (1993)
Scab	*Sphaceloma arachidis* Bit. and Jenk.	Argentina, Brazil, China	Desai and Bagwan (2005), Fang et al. (2007), Kolte (1984), de Godoy et al. (2001), Moraes et al. (2006), Wang et al. (2009)
Smut (peanut smut)	*Thecaphora frezii* Carranza and Lindquist	Cordoba (Argentina)	Marinelli et al. (2008)
Texas root rot	*Phymatotrichum omnivorum*	United States	Kolte (1984)
Verticillium wilt	*Verticillium dahliae*	Argentina, Australia, Bulgaria, Israel, Southern High Plains of the United States	Kolte (1984), Nakova et al. (2003a,b), Thiessen and Woodward (2012)
Web blotch (net blotch)	*Phoma arachidicola* Marasas, Pauer, and Boerema (= *Mycosphaerella arachidicola* Khokhr)	Australia, South Africa, United States, Zimbabwe	Kolte (1984), Mozingo et al. (2004)
Web blotch	*Ascochyta arachidis Woron.* (= *Mycosphaerella argentinensis*)	Argentina, Russia, United States	Kolte (1984)
Web blight	*Rhizoctonia solani*	India	Dubey (2000)
Zonate leaf spot	*Cristulariella moricola* (*Grovesinia pyramidalis*)	United States	Porter (1993)

OTHER FUNGAL DISEASES

Other less important fungal diseases affecting the peanut crop in different peanut-growing regions of the world are given in Table 2.4.

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3 Virus Diseases of Peanut

PEANUT ROSETTE DISEASE COMPLEX

Symptoms

In order to understand the symptomatology of this disease, it is at the outset important to understand that the disease is caused by a complex of three agents: peanut rosette virus (PRV) and its satellite RNA (SatRNA) and peanut rosette assistor virus (PRAV).

Peanut rosette disease (PRD) complex occurs as two symptom variants, chlorotic rosette and green rosette, with considerable variation within each type (Murant 1989, Naidu et al. 1999). Both forms of the disease cause plants to be severely stunted, with shortened internodes and reduced leaf size, resulting in a bushy appearance of plants. In chlorotic rosette, leaves are usually bright yellow with a few green islands, and leaf lamina is curled (Figure 3.1). In the green rosette, leaves appear dark green, with light green to dark green mosaic (Figure 3.2). Chlorotic rosette occurs throughout the sub-Saharan Africa (SSA), whereas green rosette has been reported from Angola, Kenya, Malawi, Swaziland, Uganda, and West Africa (Naidu et al. 1999). Variability in SatRNA is mainly responsible for symptom variations (Murant and Kumar 1990, Taliansky and Robinson 1997). In addition, differences in genotypes, plant stage at infection, variable climatic conditions, and mixed infections with other viruses also contribute to symptom variability under field conditions (Naidu and Kimmins 2007). Stunting is more severe in diseased peanut plants containing all the three agents than in diseased peanut plants containing only PRV and SatRNA (Ansa et al. 1990). Some reports have suggested that PRAV or PRV infection alone in peanut results in transient mottle symptoms with insignificant impact on the plant growth and yield (Taliansky et al. 2000). These results have, however, been contradicted by studies that provide evidence for the first time that PRAV infection alone, without PRV and Sat RNA, affects plant growth and contributes to significant yield losses in susceptible groundnut cultivars (Naidu and Kimmins 2007).

Geographical Distribution and Losses

PRD, first reported in 1907 from Tanganyika (presently Tanzania), is endemic in peanut-growing areas of SSA including its offshore islands such as Madagascar; it is limited to peanut crop and the African continent (Zimmermann 1907, Reddy 1991, Naidu et al. 1999). There is no evidence of PRD occurrence anywhere outside Africa. Earlier reports on its occurrence based on rosette-like symptoms in peanut in India, Java, and Australia were later confirmed as caused by other viruses such as Indian peanut clump virus (IPCV).

Yield losses due to PRD depend on the growth stage at which infection occurs (Olorunju et al. 1991). Infection due to chlorotic or green rosette disease occurring in young plants (prior to flowering) will result in 100% yield loss. In contrast, plants infected during later growth stages (between flowering and pod setting) may show symptoms only in some branches or parts of branches, and yield loss depends on the severity of infection. Infection after pod setting/maturation causes negligible effects on pod yield. An average annual yield loss due to PRD is estimated to be between 5% and 30% in nonepidemic years, and epidemics often result in 100% yield loss (Alegbejo and Abo 2002).

FIGURE 3.1 Chlorotic rosette of peanut.

FIGURE 3.2 Green rosette of peanut.

PRD usually occurs in small proportions every growing season, but its severity increases in groundnut crops sown late in the season. When epidemics do occur, peanut production is significantly reduced, and the disease has the potential to cripple rural economies in SSA (Naidu et al. 1999). An epidemic in northern Nigeria in 1975 destroyed approximately 0.7 million ha of groundnut, with an estimated loss of U.S. $250 million (Yayock et al. 1976). Similarly, an epidemic in 1995 in eastern Zambia affected approximately 43,000 ha causing an estimated loss of U.S. $4.89 million. In the following year in the central region of Malawi, peanut production was reduced by 23%. As per the estimates of ICRISAT, PRD causes an annual yield loss of U.S. $156 million in SSA (Waliyar et al. 2007).

Pathogen(s): The Causal Virus Complex

Three causal agents, as mentioned earlier, are involved in PRD etiology: PRAV, PRV, and SatRNA (Murant et al. 1988, Murant 1990, Taliansky et al. 2000). The three agents, PRAV, PRV, and SatRNA, synergistically interact.

PRAV: PRAV virions are nonenveloped, isometric shaped with 28 nm diameter particles of polyhedral symmetry. There are no reports on the occurrence of strains of PRAV. The genome is a nonsegmented, single molecule of linear positive-sense, single-stranded RNA of c. 6900 nucleotides which encodes for structural and nonstructural proteins (Murant et al. 1998). It was first recognized as a component of PRD by Hull and Adams (1968) who later identified it as a *Luteovirus* belonging to the family Luteoviridae (Casper et al. 1983, Reddy et al. 1985a). In general, any *Luteovirus* purification protocol can be applied for the purification of PRAV particles from infected peanut plants (Murant 1989, Waliyar et al. 2007). Like other members of the *Luteovirus*, PRAV is thought to encode for six open reading frames (ORFs). Only coat protein (CP) region of the genome has been sequenced (Scott et al. 1996, Murant et al. 1998). Virions are made of single CP subunits of size 24.5 kDa, and the virus is antigenically related to bean/pea leaf roll virus, beet western yellows virus, and potato leaf roll virus (Scott et al. 1996). The virus replicates autonomously in the cytoplasm of phloem tissue. PRAV is transmitted by *Aphis craccivora* in a persistent manner, and experimentally by grafting, but not by mechanical sap inoculation, seed, pollen, or contact between the plants. Peanut is the only known natural host of the PRAV. The virus is reported to occur wherever PRD has been reported. The virus on its own causes symptomless infection or transient mottle and can cause significant yield loss in susceptible peanut cultivars (Naidu and Kimmins 2007).

Groundnut rosette virus (*GRV*): The virus is restricted to SSA and its offshore islands. It is first isolated and characterized by Reddy et al. (1985b) and has no structural (coat) protein (Taliansky et al. 2003), and thus, no conventional virus particles of GRV are formed. Enveloped bullet-shaped structures that can be detected in the ultrathin sections of infected cells could be shown to be cytopathological structures due to GRV infection, as opposed to real virions (Taliansky et al. 2003). The virus genome is a nonsegmented, single linear molecule of single-stranded, positive-sense RNA of size c. 4019 nucleotides which encodes for four ORFs (Taliansky et al. 1996). The genome of an isolate has been completely sequenced (GenBAnk accession # Z 66910), and several partial sequences are available in the gene bank. The virus replicates autonomously in the cytoplasm of the infected tissues (Taliansky and Robinson 2003). GRV on its own causes transient symptoms, but a SatRNA associated with GRV is responsible for rosette disease symptoms. GRV depends on groundnut rosette assistor virus for encapsidation of its RNA and transmission by *A. craccivora* in a persistent mode (Robinson et al. 1999). The virus is transmitted by grafting and mechanical inoculation, but not through seed, pollen, or contact between the plants. Peanut is the only known natural host, but several experimental hosts in the

families Chenopodiaceae and Solanaceae have been reported (Murant et al. 1998). GRV belongs to the genus *Umbravirus*. No strain of GRV has been reported. The virus is restricted to SSA and its offshore islands.

SatRNA: SatRNA is responsible for rosette symptoms and plays a critical role in helper virus. The SatRNA (subviral RNAs) of GRV belongs to the Subgroup-2 (small linear) SatRNAs. It is a single-stranded, linear, nonsegmented RNA of 895–903 nucleotides (Murant et al. 1988, Blok et al. 1994, Taliansky et al. 2000). It totally depends on GRV for its replication, encapsidation, and movement, both within and between the plants.

Diagnosis

Various diagnostic techniques based on biological, serological (protein based), and genomic properties (nucleic acid) of the PRD agents have been developed (Kumar and Waliyar 2007, Waliyar et al. 2007). PRD can be diagnosed in the field based on the characteristic symptoms on peanut. Mechanical inoculation on to *Chenopodium amaranticolor* indicates the presence of PRV (infected plants show minute necrotic lesions on inoculated leaves about 4 days after inoculation) (Murant et al. 1998). Serological and nucleic acid–based diagnostic methods can be used for the detection of PRAV, but only nucleic acid–based methods can be used for the detection of PRV and SatRNA. Triple-antibody sandwich-enzyme-linked immunosorbent assay (ELISA) has been developed for the detection of PRAV (Rajeshwari et al. 1987) and dot blot hybridization and reverse transcription-polymerase chain reaction (RT-PCR) to detect all the three PRD agents in plants and aphids (Blok et al. 1995, Naidu et al. 1998b).

Transmission

1. *Sap transmission*: Transmissibility of sap-transmissible component is best achieved by extracting the sap in potassium phosphate buffer (K_2HPO_4) of pH 7.3 containing Mg or Na bentonite 25 mg/mL and 0.01 M diethyldithiocarbamate. By artificial mechanical sap inoculations, experimental hosts of PRV and SatRNA have been identified in several species in Leguminosae, Chenopodiaceae, and Solanaceae (Murant et al. 1998, Waliyar et al. 2007). *C. amaranticolor* and *Chenopodium murale* are local lesion hosts; *C. amaranticolor*, *Glycine max*, *Phaseolus vulgaris*, *Nicotiana benthamiana*, and *Nicotiana clevelandii* are systemic hosts of PRV. Apart from peanut, experimental hosts of both PRAV and PRV and SatRNA are *Gomphrena globosa*, *Stylosanthes gracilis*, *Stylosanthes mucronata*, *Stylosanthes sundaica*, *Spinacia oleracea*, *Trifolium incarnatum*, and *Trifolium repens* (Murant et al. 1998).
2. *Aphid transmission*: *A. craccivora*, commonly known as the cowpea aphid, is the principal vector involved in the transmission of all the PRD agents in a persistent and circulative manner (Hull and Adams 1968). PRV and SatRNA must be packaged within the PRAV CP to be aphid transmissible. Studies have shown that all the PRAV particles whether they contain PRAV RNA or PRV RNA and SatRNA are acquired by the aphid vector from phloem sap in 4 and 8 h acquisition access feeding for chlorotic and green rosette, respectively (Misari et al. 1988). Then, there is a latent period of 26 h 40 min and 38 h 40 min for chlorotic and green rosette, respectively, and the inoculation access feeding period of 10 min for both forms (Misari et al. 1988). Once acquired, aphid can transmit virus particles for up to 2 weeks and beyond. All stages of the aphid can acquire and transmit the disease agents. Transmission rates of 26%–31% have been reported with one and two aphids per plant and 49% with five aphids per plant (Misari et al. 1988).

Aphid vector does not always transmit all the three agents together (Naidu et al. 1999). Under natural conditions, some PRD-affected plants (PRV and SatRNA positive) have been found to be free

from PRAV, and PRAV can be detected in some nonsymptomatic plants (no PRV and SatRNA) (Naidu et al. 1999). This situation appears to be due to the difference in inoculation feeding behavior of the vector leading to the transmission of (1) all the three agents together, (2) only PRAV, or (3) PRV and SatRNA, as demonstrated by the electrical penetration graph studies of aphid stylet activities (Naidu et al. 1999). This reveals that during short inoculation feeding (test probe or stylet pathway phase) vector aphids probe peanut leaves without reaching the phloem, transmitting only PRV and SatRNA, which multiply in the epidermal and mesophyll cells. Even if PRAV particles are deposited in the mesophyll cells, they cannot replicate, as they can replicate only in the phloem cells (Naidu et al. 1999). However, vector aphids can transmit PRAV and PRV–SatRNA when the stylets penetrate sieve elements (salivation phase) of the phloem cells. Therefore, the success of transmitting all the three agents together is high when inoculation feeding period is longer or increasing the number of aphids per plant (Misari et al. 1988). Vector aphids fail to acquire or transmit PRV and SatRNA from diseased plants lacking PRAV, and such plants become dead-end sources. However, if such plants receive PRAV later due to vector feeding, the plants again serve as source of inoculum.

Epidemiology and Disease Cycle

The epidemiology of PRD is complex, involving interactions between and among two viruses and a SatRNA, the vector, the host plant and environment. Since none of the causal agents is seed borne, primary infection of crops depends on the survival of infected plants (virus sources) and vectors (aphids) (Naidu et al. 1998a). Possible source from which rosette could spread are infected peanut plants surviving between cropping seasons. In regions where there are no sources of infection, initial infection may depend on the influx of viruliferous aphids from other parts of Africa on prevailing wind currents (Bunting 1950, Adams 1967). The vector *A. craccivora* is polyphagous and can survive on as many as 142 plant species in addition to peanut. One or more of these 142 plant species could be a source of the rosette complex (Adams 1967, Naidu et al. 1998a). Efforts thus far have failed to identify any alternative natural hosts of the PRD agents (Waliyar et al. 2007). Kenyan isolates of the virus are closer to the Malawian than to the Nigerian isolates (Wangai et al. 2001).

PRD is a polycyclic disease because each infected plant serves as a source for initiating subsequent disease spread in the field. Winged aphids are responsible for primary spread of the disease. Secondary spread from the initial foci of disease within the fields also occurs by way of the movement of aphid vector, but largely apterae and nymphs (Naidu et al. 1998a). In general, primary infection at early stages of the crop growth provides a good opportunity for repeating cycles of infection to occur before crops mature and vector populations decline. The nature and pattern of disease spread is influenced by plant age, cultivar, crop density, time of infection, transmission efficiency of aphids, proximity to the source of infection, and climatic conditions (van der Merwe et al. 2001, Herselman et al. 2004, Waliyar et al. 2007).

Disease Management

Host Plant Resistance

Efforts in breeding for host plant resistance and evaluation of peanut germplasm collection held in ICRISAT genebank have contributed to the development of several peanut genotypes and identification of germplasm lines with acceptable levels of field resistance to rosette disease (Olorunju et al. 1991, 2001, van der Merwe and Subrahmanyam 1997, Subrahmanyam et al. 1998, 2001). Evaluation of 12,500 lines from ICRISAT's genebank collection of peanut germplasm has resulted in the identification of 150 resistant sources, of which 130 are long-duration Virginia types and 20 are short-duration Spanish types (Subrahmanyam et al. 1998, Olorunju et al. 2001). Evaluation of

116 wild *Arachis* accessions representing 28 species identified 25 accessions resistant to rosette disease (Subrahmanyam et al. 2001). Out of 2301 germplasm lines evaluated in Samaru in Nigeria, only 65 new sources of resistance to rosette could be identified, 55 of which are long-duration Virginia types and 10 are short-duration Spanish types (Ntare and Olorunju 2001). It is not known whether these resistant sources carry the same or different kinds of resistance genes. Generally, resistance to rosette disease in a genotype is assessed by the lack of symptom expression, and therefore, such resistance is largely against PRV and SatRNA (the two components responsible for rosette symptoms) (Bock et al. 1990, Subrahmanyam et al. 1998, Olorunju et al. 2001). This resistance has been shown to be controlled by two independent recessive genes and is effective against both chlorotic and green forms of rosette (Nigam and Bock 1990, Olorunju et al. 1992). This resistance is directed against PRV and consequently to SatRNA and is not effective against PRAV (Bock et al. 1990). This form of resistance has been transformed into early maturing cultivars that are useful for cultivation in regions that are often characterized by short length of growing periods. Some of the rosette-resistant early varieties released in the West and Central Africa region are ICGV-SM 90704, ICG 12991, ICGV-SM 99568, ICGV 93437, SAMNUT 23 (ICGV-IS 96894), SAMNUT 21 (UGA 2), and SAMNUT 22 (M572.80I) (Ntare et al. 2002, Waliyar et al. 2007).

Yield reduction in genotypes that are resistant to PRV and SatRNA is observed to be presumably due to their susceptibility to PRAV (Subrahmanyam et al. 1998, Olorunju et al. 2001). This is finally confirmed in a study that separated PRAV from PRV and SatRNA and demonstrated that PRAV infection alone can significantly reduce peanut seed yield (Naidu and Kimmins 2007).

There is a possibility for the development and deployment of transgenic forms of resistance using genes derived from the virus itself (pathogen-derived resistance) (Deom 1999, Deom et al. 2000). Resistance to PRV has been detected in plants transformed with constructs derived from a mild variant of the SatRNA in *N. benthamiana* (Taliansky et al. 1998). Research in this direction is in progress at the ICRISAT centers.

Chemical Control

Seed treatment with imidacloprid and followed by regular systemic insecticide spray in the early stages of the crop growth (from emergence to 40th day) will control vector aphids and consequent protection against PRD. Long acquisition access feeding period required by the vector provides an opportunity to control aphids with chemical sprays before they can spread the disease. Various insecticides have been used to control *A. craccivora* to minimize or prevent the spread of rosette disease in field trials (Waliyar et al. 2007). Dosage and type of insecticide utilized is critical for controlling aphids. However, insecticides are an unviable option in SSA due to high costs and scarcity, thus seldom preferred by the farmers. Furthermore, insecticide applications pose detrimental effects on health and environment, and their usage is being discouraged.

Cultural Control

Information on the control of PRD by cultural practices has been obtained in different parts of SSA (Naidu et al. 1998a, 1999, Waliyar et al. 2007). Early sowing (particularly in June) and high seed rate (80–120 kg/ha) have been recommended in much of Africa as a standard measure for the control of peanut rosette. Early sowing in the season is to take advantage of low aphid populations, and maintaining good plant density without any gaps (aphids prefer widely spaced plantings for landing) has been shown to reduce rosette disease incidence. However, early sowings may not be effective in areas where groundnut is grown continuously, as this allows perpetuation of virus and vector. Peanut crops intersown with field beans, maize, and sesame remain less affected with the rosette disease that is subsequently useful in preventing the spread of the disease (Alegbejo 1997). Roguing of voluntary sources and early-infected plants prevents the spread of the rosette (Kolte 1984, Waliyar et al. 2007). Overall, the combination of resistant genotypes with early sowing and optimum plant population is economically useful in the management of the disease even under high disease pressure (Subrahmanyam et al. 2002).

PEANUT STEM NECROSIS DISEASE

Symptoms

The first appearance of symptoms becomes visible in the form of the development of necrotic lesions on terminal leaflets and petioles and the death of top growing bud on the main stem followed by necrosis of all top buds on primaries. Complete stem necrosis and often total necrosis of the entire plant occur in early infection. The plants that survive the viral infection become stunted and show proliferation of axillary shoots and reduction in leaflet size and exhibit chlorosis as secondary symptoms in contrast to mosaic and mottling of leaf lamina in the case of peanut bud necrosis disease (PBND). Pods from affected plants show black necrotic lesions on the pod shells, and kernels become smaller adversely affecting the marketability of the pods and kernels.

Geographical Distribution and Losses

The first known case of the occurrence of epidemic of peanut stem necrosis disease (PSND) caused by tobacco streak virus (TSV) was recorded in the monsoon season crop in the year 2000 from the major peanut-growing district of Anantapur in the state of Andhra Pradesh in India (Reddy et al. 2002). The disease now occurs in almost all peanut-growing states of India particularly in those regions, where sunflower necrosis disease caused by the same virus (TSV) is endemically present (Ravi et al. 2001, Bhat et al. 2002). During the 2000 epidemic in Andhra Pradesh, crop losses have been estimated to exceed U.S. $64 million. The TSV, the causal virus, because it infects a wide range of crops like sunflower, safflower, cotton, cowpea, okra, urd bean, and mung bean, assumes a great significance of economic importance, and hence, the TSV is currently regarded as an emerging threat to crop production in India.

Pathogen: The Causal Virus

TSV belonging to the genus *Ilarvirus* in the family Bromoviridae is the cause of the PSND (Reddy et al. 2002). Purified virions of TSV are nonenveloped, isometric, measuring 25–35 nm in diameter. It consists of a single capsid protein of 28 kDa. The virus genome is a single-stranded RNA, has positive polarity, is linear, and is a tripartite of size 3.7, 3.1, and 2.2 with 0.9 kb subgenomic RNA. The TSV genome has been sequenced. Cowpea (*Vigna unguiculata* cv. C-152) and *P. vulgaris* (cv. Top Crop) are the suitable hosts for propagating the virus.

Diagnosis

Symptom-based identification can be misleading as similar symptoms can also be due to peanut bud necrosis virus (PBNV). Hence, diagnosis of the infection caused by TSV should be done using indicator host plants reaction test and ELISA.

The two most important diagnostic hosts are cowpea cv. C-152 and French bean cv. Top Crop. On sap-inoculated leaves of cowpea and French bean, TSV produces necrotic lesions and veinal necrosis as early as within 2–3 days, whereas PBNV produces concentric chlorotic/necrotic lesions on these indicator hosts as late as 4–5 days after inoculation.

ELISA and RT-PCR polyclonal antibodies to TSV have been produced, and the direct antibody-coated-ELISA-based virus detection technique has been developed for the reliable diagnosis of PSND. Oligonucleotide primers from CP gene have been designed for RT-PCR-based virus detection (Prasad Rao et al. 2004).

Transmission

The virus is transmitted through thrips *Frankliniella schultzei*, *Scirtothrips dorsalis*, and *Megalurothrips usitatus* in a very peculiar manner. The thrips fed on infected leaves alone and do not transmit the virus. But they do so through their wounding of plants during feeding only in the

presence of infected pollens particularly from *Parthenium hysterophorus* and/or sunflower in the vicinity of these wounds rather than entering into specific virus–vector relationships. The virus in peanut is not transmissible through seed, but it can be transmitted through sap inoculation.

Epidemiology and Disease Cycle

Though TSV is pollen borne, the virus is not transmitted through seed in the case of peanut (Reddy et al. 2007). Thrips vectors, viz., *F. schultzei*, *S. dorsalis*, and *M. usitatus*, aid in passive transmission of the virus as carriers of pollens from infected plants. Among the three thrips species, *F. schultzei* plays a major role in transmission and spread of the virus particularly from the flowers of infected *P. hysterophorus* and other weed plants (*Abutilon indicum*, *Ageratum conyzoides*, *Croton sparsiflorus*, *Commelina benghalensis*, *Cleome viscosa*, *Euphorbia hirta*, *Lagascea mollis*, *Tridax procumbense*), sunflower, and marigolds. *F. schultzei* carries 8–10 pollen grains on its body from these ranges of weed hosts and 60–70 pollen grains from sunflower flowers, and when these thrips become wind borne and visit groundnut plants, the pollen grains then get dislodged from the insect's body, and during the feeding process, virus present in the pollen grains infects the peanut plants. Since peanut is self-pollinated and early-infected peanut plants do not flower, the peanut plants on its own do not contribute to spread of the virus inoculum and the PSND in a crop field. *Parthenium* is widely distributed and present all year-round in the vicinity of peanut crop fields producing several flushes of pollen grains during its life cycle ensuring continuous supply of virus-infected pollens for effective transmission through the thrips (Prasad Rao et al. 2004, Kumar et al. 2008).

Based on 2000 PSND epidemic in Anantapur district in Andhra Pradesh in India, the following factors are conducive for the occurrence of PSND epidemic: (1) early rains during late May or early June that encourage germination and growth of *Parthenium*, (2) sowing peanut during July by which time *Parthenium* is in full bloom, (3) normal rain that promotes good growth of peanut crop as well as *Parthenium*, and (4) one or two dry periods of 3 week duration that encourages thrips multiplication and movement for the spread of virus.

Disease Management

Host Plant Resistance

1. *In peanut germplasm*: Resistance to PSND has not been found in the germplasm of cultivated peanut (Kalyani et al. 2007). However, a considerable number of peanut genotypes have been reported to be resistant or moderately resistant or promisingly less susceptible to the disease. These genotypes are ICGV 99057, 00169, 99068, 86325, 92267, 94379, ICG 4983 (*Arachis chacoense*), ICGS 37, RP 251, S 206, DH 40, CSMG 84-1, and M-22. Among these, the multiple disease-resistant genotypes ICGV 99057 and ICGV 00169 besides being of high potential of PSND resistance also exhibit good shelling percentage and oil content. Hence, it is rewarding to incorporate resistance into good agronomic types by hybridization and selection (Kumar et al. 2008, Venkataravana et al. 2008).
2. *Transgenic peanut for resistance to PSND*: Transgenic resistance in peanut to PSND has been obtained by transferring CP gene of TSV through *Agrobacterium*-mediated transformation of deembryonated cotyledons and immature leaves of peanut cultivars Kadiri 6 and Kadiri 134. The transgenic lines are reported to remain symptomless throughout and show traces or no systemic accumulation of virus indicating the tolerance/resistance to the TSV infection. CP gene expression has been confirmed in transgenic lines by RT-PCR, real-time PCR, and ELISA. This is an effective strategy for developing peanut with resistance to PSND (Mehta et al. 2013).

Chemical Control

Seed treatment with imidacloprid (Gaucho 70 WS) at 2 mL/kg seed followed by spraying of monocrotophos at 800 mL or dimethoate at 1000 mL or imidacloprid at 200 mL/ha in 500 L of water at 25–30 days after sowing is recommended in epidemic regions. It is noteworthy that in the case of most of the thrips-transmitted viruses, the use of insecticides after the appearance of the disease has no effect on the control of the disease (Prasad Rao and Reddy 2005).

Cultural Control

Removal of weeds such as *P. hysterophorus* and other weeds around the peanut fields is helpful to reduce the disease incidence; however, roguing of early-infected peanut plants may not limit further spread of the disease in the field. Barrier crops like bajra, maize, and sorghum should be planted in four to eight rows around the peanut field. These will prevent thrips and wind-borne weed pollen-carrying virus. Practicing intercropping with bajra, maize, and sorghum in the ratio of 7:1 or 11:1 is also helpful in decreasing the incidence of PSND in the peanut crop. Maintenance of optimum plant density is important to discourage landing of the thrips (Prasad Rao et al. 2004, Kumar et al. 2008).

PEANUT BUD NECROSIS DISEASE

Symptoms

Under field conditions, initially quadrifoliate leaf immediately below the terminal bud shows distinct chlorotic ring spots or chlorotic speckling and becomes flaccid about 30–40 days after planting. These symptoms in peanut due to PBNV are difficult to distinguish from those caused by tomato spotted wilt virus (TSWV). The vascular tissue of the shoot just below the growing tip becomes necrotic and the terminal bud is killed, which later dries and becomes brown. This is a characteristic symptom that occurs on peanut plants of crops grown in the rainy and post-rainy seasons, when ambient temperatures are relatively high above 30°C. The necrosis may proceed downward, and the whole branch may become blighted. Necrosis may also be seen on petioles and along the stems. Proliferation of axillary shoots takes place, but the leaves of such shoots remain smaller than normal and show a wide range of symptoms, including distortion, mosaic mottling, and general chlorosis. Infection with PBNV reduces the concentration of chlorophyll *a* and *b* and increases the specific activity of chlorophyll oxidase and peroxidase enzymes (Hema and Sreenivasulu 2002). Affected plants remain stunted because of the reduction in the length of the internodes, and the whole plants may show a bushy appearance. If plants are infected early, they are stunted and bushy. If plants older than 1 month are infected, the symptoms may be restricted to a few branches or to the apical parts of the plants. Seeds from such plants are small, shriveled, mottled, and discolored. Such seeds show poor germination, or they may fail to germinate. Late-infected plants may produce seed of normal size. However, the testae on such seed are often mottled and cracked.

Geographical Distribution and Losses

PBND was first recorded in India in 1949 as per the reports made from Indian Agriculture Research Institute, New Delhi (Reddy et al. 1995). The economic importance of the disease was realized during the late 1960s when incidences up to 100% were recorded in many peanut-growing regions in India. The disease was described under different names such as ring spot of peanut, ring mosaic, spotted wilt, bud blight, and bud necrosis (Kolte 1984). It was shown to be economically important in parts of Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra, and Uttar Pradesh. Although it was earlier reported to be caused by TSWV, currently the causal virus of PBND in India has been shown to be a serologically distinct *Tospovirus*, now referred to as PBNV, transmitted by *Thrips palmi*. Surveys in many groundnut-growing countries indicate that PBNV is restricted to South and Southeast Asia (Reddy et al. 1995, Poledate et al. 2007, Damayanti and Naidu 2009). Reports of its

occurrence have been made from the main peanut-growing province of Córdoba, in Argentina in South America (de Breuil et al. 2008), and from the Golestan Province of Iran (Golnaraghi et al. 2002). The disease poses a major threat to peanut production in Thailand in dry seasons (Poledate et al. 2007). In India, average yield loss caused by the PBND is more than 50% in peanut that amounts to estimation of about U.S. $89 million/annum (Reddy et al. 1995, Kendre et al. 2000). The causal virus, the PBNV, appears to be economically more significant because different isolates (strains) of PBNV have been proved to be the primary pathogens in causing a number of diseases in different crop plants such as chilli (Gopal et al. 2011a), cucumber (Gopal et al. 2011a), cowpea (Jain et al. 2002, Akram and Naimuddin 2009, Gopal et al. 2011a), mung bean (Jain et al. 2002, Thien et al. 2003, Sreekanth et al. 2006a, Saritha and Jain 2007), okra (Kunkalikar et al. 2012), sesame (Gopal et al. 2011a), soybean (Kumari et al. 2003), sunflower (Pranav et al. 2008), taro (Sivaprasad et al. 2011), tomato (Jain et al. 2002, Raja and Jain 2006, Venkat et al. 2008, Venkatesan et al. 2009, Manjunatha et al. 2010a, Ramana et al. 2011, Akhter et al. 2012), potato (Akram et al. 2003, Pundhir et al. 2012), urd bean (Prasad Rao et al.2003, Kumar et al. 2006), pea (Akram and Naimuddin 2010), and watermelon (Gopal et al. 2011a).

Causal Virus: Peanut Bud Necrosis Virus

The virus causing the PBND does not react with the antisera to TSWV obtained from different sources (Reddy et al. 1992), and based on serological cross reactions (Adam et al. 1993) and amino acid sequence homology of the nucleoprotein (de Avila et al. 1993), it has revealed the existence of a distinct virus, different from the TSWV and impatiens necrotic spot virus (INSV), and thus, the virus-causing PBND has been identified as a distinct *Tospovirus* and named as PBNV. With ELISA as well as Western blots, PBNV has been shown to be serologically distinct from TSWV and INSV (Reddy et al. 1992). PBNV contains three RNA species of about 9.0 kb (large, L RNA), 5.0 kb (medium, M RNA), and 3.0 kb (small, S RNA), and the nucleotide sequences are 8911 for L RNA, 4801 for M RNA, and 3057 for S RNA (Satyanarayana et al. 1996a,b). The virus protein consists of four polypeptides of molecular weights of 27, 52, 58, and 78×10^3 Da. The particles are 70–90 nm in diameter and are surrounded by a double membrane of protein and lipid and sediment at 520–530s. Nonstructural protein, NSs, of the PBNV encoded by the S RNA is a bifunctional enzyme, which could participate in viral movement, replication, or suppression of the host defense mechanism (Lokesh et al. 2010, Bhat and Savithri 2011). Typical of a *Tospovirus*, the PBNV has extremely low thermal inactivation point of 45°C for 10 min; the dilution end point is between 10^{-2} and 10^{-3} and short longevity in vitro of less than 5 h at room temperature. PBNV classified as a virus in serogroup IV of *Tospoviruses* (Bunyaviridae) (Akram et al. 2004).

Transmission

Though the PBNV is graft transmissible, it is not transmitted through aphids and seed. Transmission through sap and thrips transmission are most common that are as follows.

Sap transmission: PBNV can be transmitted by mechanical sap inoculations if care is taken to extract the virus only from young infected leaflets with primary symptoms. Extracts should be prepared in neutral phosphate buffer (0.05 M phosphate buffer, pH 7.0) containing an antioxidant such as mercaptoethanol (0.2%) or thioglycerol (0.075%) and must be kept cold throughout the inoculation process.

Thrips transmission: *T. palmi* transmits PBNV. Other thrips species *S. dorsalis* and *F. schultzei*, which are also present on the plants, do not transmit the PBNV. Adults of *T. palmi* cannot acquire the virus; however, their larvae can acquire the virus and such larvae and subsequently developed adults transmit the virus persistently (Reddy et al. 1991, Sreekanth et al. 2006a,b). A minimum of 15 min acquisition access period by larvae and

a 45 min inoculation access period by adult thrips are required for successful transmission of the PBNV (Sreekanth et al. 2004a, 2006a). Maximum transmission (100%) can be obtained when there are 10 adults per plant. The majority of individual adult thrips transmit the virus for more than half of their life period. Cowpea has been found to be the best host for rearing and multiplying *T. palmi* under laboratory conditions.

Diagnosis

Sap inoculations of virus extracts on diagnostic hosts like cowpea (*Vigna unguiculata*) cv. C-152 and *Petunia* hybrid can be used to identify the PBNV. Cowpea produces concentric chlorotic and necrotic lesions on inoculated primary leaves 4–5 days after inoculations, and subsequently systemic infection develops on newer leaves (Akram and Naimuddin 2009), whereas *Petunia* produces only necrotic lesions on inoculated leaves 3 or 4 days after inoculation (Reddy et al. 1991). ELISA using polyclonal antibodies clearly distinguish PBNV from TSWV and INSV (German et al. 1992, Reddy et al. 1992, Jain et al. 2005, Nagaraja et al. 2005b, Raja and Jain 2006). If young tissues showing initial symptoms are used, PBNV particles can be observed in leaf extracts or even in leaf dip preparations. They are 80–100 nm in diameter and are surrounded by a double membrane of protein and lipid.

Epidemiology and Disease Cycle

There is a possibility of existence of different strains of PBNV, as different isolates of the virus collected from different regions show some differences in host range and reaction of susceptible hosts. Though the detailed study in strain differentiation has not been done, it is revealed through the nucleotide and amino acid sequences of the movement protein (NSm) genes of different isolates that the NSm genes of PBNV isolates are identical in length (924 bp encoding 307 amino acids) suggesting their common origin (Akram et al. 2003, 2004, 2012). The primary sources of inoculum may be different host plant members of Leguminosae and Solanaceae specially crop plants such as tomato, mung bean, urd bean, sunflower, and cucumber that are cultivated in summer and weed hosts such as *Ageratum conyzoides*, *Acanthospermum hispidum*, and *Cassia tora*, which are more commonly present in and around the peanut crop fields and which sustain virus infection and effective thrips vector population (Nagaraja et al. 2005a, Reddy et al. 2011, Gopal et al. 2011a). Thus, the incidence of the PBND in peanut depends on infection by viruliferous thrips that acquire the virus from such alternative hosts and their mass migration flights from alternative hosts to peanut crop fields. Most migrations occur when air temperature is in the range of 20°C–30°C. Warm and dry weather favor disease buildup and prevalence of thrips (Thiara et al. 2004, Pensuk et al. 2010). An optimum temperature of 25°C is the best for rearing *T. palmi* and the total number of larvae produced per female is greater at 25°C (Vijayalakshmi et al. 2000). A wind velocity of 10 km/h at 3 m above the crop canopy is more conducive to mass flights of thrips (Prasad Rao and Reddy 2005). Interestingly, secondary spread from infected peanut plants within a peanut field is considered to be negligible.

Disease Management

Host Plant Resistance

Three peanut germplasm lines, viz., IC 10, IC 34, and ICGV 86388, have been confirmed to be the best PBND-resistant parental lines and useful sources of resistance not only to PBNV (Reddy et al. 2000, Pensuk et al. 2002a–c, 2004, Kesmala et al. 2006) but also to TSWV (do Nascimento et al. 2006), a closely related species of PBNV. Heritability estimates for both incidence and severity of PBND are found to be favorably high enough in these three PBNV-resistant parental lines for further improvement of these characters. Both genotypic and phenotypic correlations between PBNV resistance parameters and desirable agronomic traits are also high (Kesmala et al. 2003, 2004, Tonsomros et al. 2006). However, susceptibility of peanut to PBNV is somewhat associated with large-seed size,

and this, therefore, might be an interfering factor for breeding large-seeded peanut cultivars with resistance to PBNV (Puttha et al. 2008). Two peanut cultivars such as ICGS 11 and ICGS 44 possessing resistance to PBND have been released in India, and some other promising PBND-resistant peanut genotypes are ICGV 92269, 89/94-3-2, ICGV 91229, ICGV 91193, 89/94-7-3, 83/151-7, 85/203-6, ICGV 91248, ICGV 91117, and ICGV 86031 (Gopal et al. 2004). Three more peanut genotypes, viz., GPBD-4, JSSP-9, and DH-53, are reported to be least affected by PBND indicating their very high degree of tolerance to the disease (Nagaraja et al. 2005b). Among 83 wild *Arachis* germplasm screened for resistance to PBNV, one accession each of *Arachis benensis* and *Arachis cardenasii* and two accessions of *Arachis villosa* are confirmed to be resistant to PBNV (Reddy et al. 2000). In these wild *Arachis* PBNV-resistant accessions, the inoculated leaves do show infection, but subsequently developed leaves do not show the presence of virus in spite of repeated sap inoculation indicating the resistance in these accessions appears to be due to a block in systemic movement of the virus. Since *A. cardensii* and *A. villosa* are the progenitors of cultivated peanut and can be hybridized with the latter, the resistant wild *Arachis* accessions can be successfully utilized in conventional breeding program to transfer PBNV resistance to widely cultivated peanut cultivars (Reddy et al. 2000).

Cultural Control

The occurrence and severity of the disease depends on the migration of the thrips. Therefore, there exists a scope for choice of planting dates in reducing or avoiding the disease (Sreekanth et al. 2002, Gopal et al. 2007). Under Andhra Pradesh (India) conditions, early planting at the onset of the rainy season decreases disease incidence. Interrow and intrarow spacing of 20 × 7.5 or 10 cm gives a high density of plant population (2–3 million plants per hectare), which ensure close canopy, leading to reduction in incidence of PBND (Gopal et al. 2007). Roguing of infected peanut plants should not be practiced as this will reduce the density of crop canopy leading to increased incidence of PBND. The movement of the thrips vector is decreased when pearl millet, sorghum, pigeon pea, or maize is intercropped with peanut in the ratio 3:1 resulting in significant decrease in the incidence of PBND (Sreekanth et al. 2004b, Gururaj et al. 2005, Gopal et al. 2010).

Several weed species particularly *Achyranthes aspera*, *Ageratum conyzoides*, *Alysicarpus rugosus*, *Commelina benghalensis,* and *Vigna trilobata* have been found to be reservoirs of PBNV in and around the peanut fields in major peanut-growing regions in India. Removal and destruction of these weed species acting as primary sources of infection would be useful in reducing the incidence of PBND (Gopal et al. 2011a).

Effect of Botanicals

PBNV-susceptible peanut cultivar sprayed separately with sorghum leaf, coconut leaf, and neem kernel or neem cake extracts (10%) 20 and 35 days after planting alone or in combination with 1.25 mL of monocrotophos significantly reduce the incidence of PBND that subsequently results in increase in pod yield by 60%–100% (Kulkarni et al. 2003, Gopal et al. 2011b). Antivirus principle in sorghum and coconut leaf extracts appear to be proteinous compound, and its translocation mechanism might afford protection besides induction of its effect in the accumulation of high concentration of defense-related enzymes such as phenylalanine ammonia lyase, peroxidase, and polyphenol oxidases (Manjunatha et al. 2010b).

SPOTTED WILT

Symptoms

TSWV-infected peanuts first appear at random throughout a field as early as 21 days after the seedlings emerge. Earliest symptoms of the disease are brown speckles on the underside of the first leaf below one or more terminal buds along the leaf. The leaf below the terminal bud, showing typical yellowing and mottling, appears wilted, while the rest of the plant looks healthy. With time, clusters

of diseased plants may be seen. The virus usually spreads within a field down the row from plants infected at the start of the growing season. Brown necrotic spots or streaks may also be seen on the leaf petiole and stem and at times on the terminal bud. These spots may develop into a shoot dieback, which may ultimately kill the plant. Any new leaves are about half their normal size, crinkled and display a range of symptoms including chlorosis, concentric chlorotic ring spots, ring spots with green centers, and chlorotic line patterns. Severe stunting is a common symptom of TSWV infection of susceptible peanut cultivars and is generally more severe when young plants are infected.

Geographical Distribution and Losses

Significant economic losses have been recorded by peanut growers in the southeastern United States since the peanut growers in one Texas County suffered an estimated U.S. $3 million loss in 1986 due to TSWV. By 1988, symptomatic plants could be seen quite commonly in peanut crop stand in Alabama, Florida, and Georgia. However, the numbers of TSWV-infected plants in most fields remained extremely low. In recent years, severe outbreaks of spotted wilt in peanuts have occurred in South Central Georgia. In some fields, an estimated 40% to nearly 100% of peanut plants have been found to be infected with the disease. The disease has been particularly damaging in mid-April planted peanut (Olatinwo et al. 2009). TSWV is now well established throughout the southeastern peanut belt and has become a serious problem in the Virginia/Carolina peanut-growing regions of the United States. During 2002, the disease was present in 47% of the North Carolina hectarage and caused a 5% yield reduction in Virginia (Herbert et al. 2007). There appears to be a significant correlation between spotted wilt intensity and peanut yield (Olatinwo et al. 2010). Recent epidemics in Alabama, Florida, Georgia, Mississippi, and Texas show that the virus is a serious threat to peanut production in the region (Culbreath et al. 2011). The disease is also reported to occur in the province of Cordoba in Argentina (de Breuil et al. 2008).

Pathogen

Spotted wilt disease of peanut (*Arachis hypogaea*) is caused by TSWV (genus *Tospovirus*, family Bunyaviridae). Virions of TSWV are complex compared to many plant viruses. There are three RNAs in the virus genome that are individually encapsidated and are collectively bound by a membrane envelope, that is, of host origin. This complex virion structure is a characteristic that distinguishes TSWV from most other plant viruses. TSWV virions are roughly spherical and are 80–110 nm in diameter. Two virus proteins processed during replication to contain sugars, that is, glycoproteins (GPs), are dispersed throughout the surface of the viral envelope. These proteins are called GP C and GP N and differ slightly in size. Inside the viral envelope is each of the three viral RNAs, individually bound by multiple copies of a nucleocapsid protein. The three RNAs differ in size and are called large, middle, and small. Also, inside the envelope are several copies of a virus-encoded *replicase* protein that is required to initiate virus replication in a new host. The GPs in the envelope function in the maturation and assembly of virions and appear to play a role in the acquisition of TSWV by thrips. Envelope-deficient isolates of TSWV, generated by serial mechanical passage in plants, are infectious in plants but are not transmitted by thrips. It is evident that the GPs are not required for replication in plants, but are required for virus infection of thrips leading to subsequent virus replication in and transmission by thrips.

In addition to the GPs, the M RNA segment encodes a nonstructural protein (NSm). The NSm is unique to *Tospoviruses* in the family Bunyaviridae and is thought to be an adaptation of *Tospoviruses* to plants to facilitate *Tospovirus* the movement from cell to cell through plant cell walls via the plasmodesmata. Because enveloped particles are too large to be transported through plasmodesmata, the role of NSm is to form tubules that facilitate the movement of nucleocapsids (RNA plus protein) from cell to cell. In addition to the nucleocapsid protein, the S RNA segment encodes a nonstructural protein (NSs). Crystalline-like structures of NSs are produced in infected insect cells and plant cells. The NSs protein has RNA-silencing suppressor activity and may play a role in posttranscriptional

gene silencing or RNA metabolism. TSWV is one member of the dozen or so different viruses in the genus *Tospovirus*. One striking difference in these viruses is the variation in their host ranges. TSWV is renowned for having an extensive host range, whereas other members of the genus *Tospovirus* such as peanut yellow spot virus or *Iris* yellow spot virus have narrow host ranges.

Diagnosis

TSWV can be detected using ELISA in both leaf and root crown tissues throughout the peanut-growing season to determine the time and percentage of infected plants (Rowland et al. 2005, Murakami et al. 2006). Diagnosis of TSWV in peanut can also be accomplished by RT-PCR. ELISA and RT-PCR are comparable for detecting TSWV infection rate in field-grown peanuts. A delayed accumulation of TSWV in a cultivar is a reliable indicator of host plant resistance (Dang et al. 2009, 2010).

Transmission, Epidemiology, and Disease Cycle

Worldwide, seven species of thrips are known to be vectors of TSWV. Two of these thrips vectors, the tobacco thrips, *Frankliniella fusca*, which is by far the most abundant, followed by the western flower thrips, *Frankliniella occidentalis*, are known to be efficient vectors of TSWV (Riley et al. 2011). However, the western flower thrips are only a minor component of the total thrips population on peanuts. Thrips may be the primary source of TSWV. Adult thrips carrying TSWV overwinter in the soil and crop debris and transmit the virus at or shortly after seedling emergence. Possibly, weed and crop reservoirs of TSWV also determine whether virus-carrying thrips overwinter in these hosts. Newly emerged peanut seedlings are infested by adult thrips migrating into the field. Adult female thrips usually lay eggs between the young, folded leaflets. After 3–5 days, the first-stage larvae emerge and feed for about 2 days before changing into larger, second-stage larvae. These larvae feed for 3–5 days before changing into a nonfeeding, inactive prepupal stage. Adult thrips then emerge 3 days later. The average time required to complete the cycle from egg to adult is about 13 days for tobacco thrips. Thrips damage to peanut is characterized by scarring and deformation of new leaves, which often results in a stunted, slow-growing seedling. Adult female tobacco thrips are small (1.3 mm) and dark brown. Male tobacco thrips are smaller (1 mm) and pale yellow. Tobacco thrips of both sexes occur in winged or wingless forms. During the growing season, the ratio of females to males may be 6:1 or greater. Female western flower thrips are also small (1.5 mm), with a yellow to blotchy brown abdomen. Males are smaller (1 mm), with a pale yellow body. Larvae of both species range from pale to bright yellow and have bright red eyes. Thrips larvae acquire TSWV by feeding on virus-infected plants. However, the thrips are capable of transmitting the virus only as adults, and they can do so throughout the remainder of their lives. The average life span of an adult female tobacco thrips is about 33 days. TSWV must be acquired by thrips during the larval stage of their development to be transmitted. Thus, only immature thrips that acquire TSWV, or adults derived from such immatures, transmit the virus. The ability of thrips to acquire TSWV decreases as the thrips age. Although the time in development that thrips can acquire the virus is limited, the wide host range for both virus and thrips facilitates the development of epidemics (Culbreath et al. 2011). Once acquired by the larvae, the virus is passed transstadially, that is, TSWV persists through insect molts from larval to adult stages. The virus replicates in thrips, and the thrips can transmit the virus during their entire life. Some evidence indicates that the viral GPs bind to the midgut epithelium and have a role in the process of virus uptake in the midgut. The virus then moves to other cells and organs, becomes well established in the muscle cells epithelium, and have a role in the process of virus uptake in the midgut. Another perspective is that the temporary association between the midgut, visceral muscle, and salivary gland complex in the larval stage provides the avenue for the virus to become systemically established in the thrips. Eventually, the virus enters the salivary glands.

Indirect evidence indicates that virions are excreted with the saliva into host plants during thrips feeding. The TSWV can be transmitted mechanically, and transmission efficiency is improved by the use of two antioxidants (sodium sulfite and mercaptoethanol) and two abrasives (Celite and Carborundum) in extracting the sap inoculum and by application of the inoculum rubbing with a cotton swab dipped in the inoculum as well as pricking with an inoculation needle (Mandal et al. 2001, 2006, Al-Saleh et al. 2007).

Disease Management

Host Plant Resistance

1. *In peanut germplasm*: Although genes to confer resistance to TSWV have been found in some peanut germplasm lines and used to develop new cultivars, there has been rapid adaptation of new forms of the virus to cultivars that have been released. The genetic diversity in this virus group may be fostered by their replication in both different species of plants and different species of thrips. Biological diversity of TSWV may, however, be useful in developing more durable TSWV-resistant crop through induced systemic resistance (Mandal et al. 2006). Thus, currently there are virtually no cultivars of peanut with significant levels of resistance to TSWV that have remained resistant in the field for more than a few years. However, the development of tolerant cultivars has proven to be one of the most promising methods to manage the disease (Riniker et al. 2008). Peanut genotypes reported to be resistant/tolerant to TSWV are given in Table 3.1. High levels of field resistance to TSWV in peanut breeding lines have been derived from hypogaea and hirsuta botanical varieties (Culbreath et al. 2005).

TABLE 3.1
Peanut Genotypes Resistant (R) or Moderately Resistant (MR) to Tomato Spotted Wilt Virus (TSWV) as Reported from Different Countries in the World

Genotype	Country	R/MR	Reference(s)
Georgia-08V (PI 655573)	United States	R	Branch (2009)
Georgia-07W, Georgia-03L, AP-3	United States	R to both TSWV and SR	Branch and Brenneman (2009)
AP-3, York, Tifguard, Georgia-03L	United States	R/MR	Culbreath et al. (2008)
C724-19-15, Tifguard	United States	R to both TSWV and root-knot nematode	Holbrook et al. (2008a)
F NC94022-1-2-1-1-b3	United States	R	Culbreath et al. (2005)
Georgia-01, Georgia-05	United States	R (multiple pest resistance)	Branch and Culbreath (2008)
Geoorganic cv. 100 (PI 648033)	United States	R	Holbrook and Culbreath (2008)
TifGP-1 (PI 648354)	United States	R to both TSWV and root-knot nematode	Holbrook et al. (2008b)
Georgia-06G (CV 94, PI 644220)	United States	R	Branch (2007a)
Georgia Greener (CV 95, PI 644219)	United States	R	Branch (2007b)
ANorden (CV 97, PI 636442)	United States	R	Gorbet (2007a)
Tifrunner (CV 93, PI 644011)	United States	R	Holbrook and Culbreath (2007)
AP-3 (CV 99, PI 633912)	United States	R	Gorbet (2007b)
CHAMPS	United States	MR (less susceptible)	Mozingo et al. (2006)
Tamrun OLO7	United States	R	Baring et al. (2006)
IC 10, IC 34, ICGV 86388	Brazil	R (higher resistance than standard *Georgia Green* for TSWV resistance)	do Nascimento et al. (2006)
C-99R, C11-2-39	United States	R	Mandal et al. (2002)

2. *Transgenic peanut for resistance to TSWV*: Considerable effort has been expended to develop transgenic peanut plants that have virus-derived genes to confer resistance to TSWV. Transgenic peanut progenies that express antisense nucleocapsid (N) gene of TSWV when subjected to natural infection of the virus under field conditions or to challenge inoculation under controlled environmental conditions show significantly lower incidence of the spotted wilt disease. But these have not been used commercially, but could be used in a traditional breeding program to enhance host resistance (Magbanua et al. 2000, Schwach et al. 2004, Yang et al. 2004).

Vector Control through Insecticides

Use of insecticides alone to control thrips populations in the field is often ineffective. Contact insecticides generally do not reach where the thrips are located on the plant, and systemic insecticides do not act rapidly enough to prevent virus transmission. As more is learned about thrips feeding, treatments that deter feeding or induce host resistance to deter thrips feeding may be used. Some success, however, has been achieved as furrow application of aldicarb and phorate results in significant levels of thrips control with reduced incidence of TSWV and significant increase in peanut pod yield (Wiatrak et al. 2000, Herbert et al. 2007).

Cultural Control

In general, timing of planting to avoid major thrips migrations during critical early plant growth periods is feasible to reduce disease. An integrated approach that addresses many parameters that affect tomato spotted wilt development has been successful in mitigating tomato spotted wilt in peanut in the southeastern United States. Peanut variety, planting date, plant population, insecticide application, disease history, row pattern, and tillage have been identified as factors affecting disease development. These factors are weighted to determine a *risk index* for TSWV in the crop. The grower obtains a low, moderate, or high risk value that can be considered when implementing crop production practices. Establishment of high peanut populations of the most resistant cultivars through the use of high seed germination rate in a well-prepared seedbed in early to mid-May plantings, when soil temperatures and moisture conditions favor uniform germination and rapid seedling growth, helps suppress epidemics of spotted wilt (Tillman et al. 2007, Culbreath et al. 2008, 2012).

PEANUT STRIPE

Symptoms

Symptoms on peanut plants vary, depending on virus isolate and peanut cultivar. For most isolates, the initial symptoms appear as chlorotic flecks or rings on young quadrifoliates. The plants are slightly stunted. Subsequently, the older leaves show symptoms that are more specific to the isolate: mild mottle, blotch, stripe, chlorotic ring mottle, chlorotic line pattern, oak leaf pattern, or necrosis (Wongkaew and Dollet 1990). The name *peanut stripe* has been given to the disease on the basis of stripes and green banding symptoms along lateral veins (Demski et al. 1984), characteristic of infected peanut plants. Subsequently, research on peanut stripe virus (PStV) obtained from different regions of the world indicated the existence of specific strains of the virus producing distinct symptoms on peanut. The stripe isolate produces discontinuous stripes along the lateral veins on young quadrifoliates; older leaflets show striping, mosaic in the form of green islands, and an oak leaf pattern. For most other PStV isolates, the initial symptoms appear as chlorotic flecks followed by mild mottle, blotch, or chlorotic ring mottle symptoms. Some isolates have been reported to produce leaf necrosis (Wongkaew and Dollet 1990). PStV differs from peanut mottle virus (PeMoV) in that the stripe symptoms persist in older leaflets, and early-infected plants are stunted in the case of isolates from Asia.

Geographic Distribution and Losses

The PStV is a major cause of yield reductions in peanut crops in many countries. Naturally occurring infections have been reported in China, Japan, Thailand, Philippines, Malaysia, Indonesia, and Myanmar, and the virus has entered the United States from China in 1982 (Demski et al. 1984) and India in 1987 (Demski et al. 1993) with germplasm introductions. Yield losses due to infection under dry season of peanut production are frequently as high as 75%–80%.

In Gujarat, India, the disease incidence has been recorded up to 40%, and it is prevalent in all other four major peanut-growing states (Andhra Pradesh, Karnataka, Maharashtra, and Tamil Nadu) in India (Jain et al. 2000). In Southeast Asia, high incidences of up to 38% have been reported causing yield reduction of 30%–60% in the peanut-growing area in Indonesia (Saleh et al. 1989) and the Philippines (Adalla and Natural 1988) and up to 100% disease incidence in South Korea (Choi et al. 2001). In northern China, where more than 65% of the nation's peanuts are produced, an incidence of over 50% has been reported (Xu et al. 1991). The virus has also been detected in Senegal. The risk of accidental introduction of the virus in any peanut-growing country in imported raw peanuts is considered high, and aphids capable of transmitting it are widespread in peanut crops. PStV infection has a highly variable effect on peanut yield, depending on the geographical conditions, cultivar, and virus isolate.

Causal Virus: Peanut Stripe Virus

Other scientific names for the virus are groundnut stripe virus, peanut stripe potyvirus, groundnut mild mottle virus, groundnut mosaic virus, peanut chlorotic ring mottle virus, sesame yellow mottle virus, peanut mild mottle virus, peanut chlorotic ring virus, peanut mosaic virus, and sesame yellow mosaic virus. PStV is a member of the potyvirus group and consists of filamentous flexuous rods, approximately 752 nm long and 12 nm in diameter, which have a sedimentation coefficient of 150 S and buoyant density in cesium chloride of 1.31 g/cm. Each particle consists of a single protein species of 33,500 Da. Molecular sequencing and analysis of the viral genome of Ts strain of PStV has been done (Wang et al. 2005). The genome is a single-stranded positive-sense RNA molecule of about 9500 nucleotides. The CP contains 287 amino acid residues with a molecular weight of 32,175 Da (Mckern et al. 1991, Cassidy et al. 1993). The particles are relatively stable and can be stained with 2% phosphotungstate or ammonium molybdate pH 6.5 (Demski et al. 1993). PStV strains have a CP sequence variability of below 10% and can be defined according to geographic origin and symptom type (Higgins et al. 1999). A study of the biological and genetic variability of PStV isolates in Indonesia, Thailand, and China found geographically related groups with wide symptom diversity. Indonesian isolates of PStV have been identified as intraspecies recombinants. This information is significant for future diagnosis.

Transmission

The virus is transmitted by several species of aphids in a nonpersistent manner, which is also the only means of disease spread under field conditions. *A. craccivora* is the major vector for the transmission of PStV. Apart from *A. craccivora*, *Myzus persicae* and *Aphis gossypii*, *Hysteroneura setariae* have been shown to be highly efficient PStV vectors for the transmission of the disease.

PStV transmission through groundnut seed can be as high as 37% in artificially inoculated plants (Demski et al. 1984, Demski and Warwick 1986). Under natural conditions, however, the transmission frequency is up to 7%. PStV seed transmission frequency can be influenced by the virus isolate, groundnut cultivar, and environment. The virus can be detected in both the embryo and the cotyledon, but not in the seed testa. Most cultivars tested from natural infection show less than 4% seed transmission (Demski and Reddy 1988).

Diagnosis

It is a common experience that symptoms of the disease caused by PStV resemble those of PeMoV. However, PStV can be distinguished by infectivity assays using indicator hosts reaction. For example, *C. amaranticolor* on sap inoculation produces chlorotic or necrotic lesions in response to PStV, whereas PeMoV does not produce any infection. *Phaseolus vulgaris* cv. Top Crop, on the other hand, remain uninfected due to PStV, but it produces reddish local lesions in response to PeMoV infection. *Pisum sativum* remains uninfected due to PStV inoculation, but it produces systemic mosaic due to infection caused by PeMoV.

PStV particles react strongly with antisera of black-eye cowpea mosaic, clover yellow vein, and soybean mosaic viruses but not with PeMoV antiserum. An immunocapture (IC)-RT-PCR technique that detects the virus in seed lots, which is more sensitive than ELISA, has been developed (Gillaspie et al. 2000). A technique has been developed for the detection of PStV in individual seeds without affecting their germination. The virus can be detected in both the embryo axis and the cotyledon. ELISA can detect one PStV-infected seed in a pool of 25 healthy samples. A dot blot hybridization technique has also been applied to detect PStV in seeds. The sensitivity of this technique is about 10 times greater than ELISA (Bijaisoradat and Kuhn 1988). Further, the technique can differentiate between the presence of PStV and PeMoV in peanut seeds.

Host Range

Besides *A. hypogaea* (peanut), the PStV can infect *Calopogonium caeruleum, Centrosema pubescens* (centro), *Crotalaria pallida* (smooth crotalaria), *Desmodium* (tick clovers), *G. max* (soybean), *Indigofera* (indigo), *Lupinus albus* (white lupine), *Medicago sativa* (lucerne), *Pogostemon cablin* (patchouli), *Pueraria phaseoloides* (tropical kudzu), *Senna obtusifolia* (sicklepod), *Senna occidentalis* (coffee senna), *Senna tora* (sicklepod), *Sesamum indicul* (sesame), *Stylosanthes* (pencil flower), *Uraria crinita* (medicinal plant in Taiwan), *Vigna radiata* (mung bean), and *V. unguiculata* (cowpea) (Liao et al. 2004, Singh et al. 2009).

Epidemiology and Disease Cycle

Though the main source of primary infection in the new planting is PStV-infected peanut seed, several crop plants and weed hosts in peanut-cropping system that fall in the host range of the virus do also serve as effective source of primary infection. Consequently, many aphid species mentioned earlier transmit the PStV in a nonpersistent manner and do play the solitary means of spread of the disease under field conditions (Demski et al. 1993).

Disease Management

Host Plant Resistance

1. *Among the cultivated and wild Arachis germplasm*: Resistance to PStV among the cultivated peanut accessions is not available. However, several accessions of wild *Arachis* spp. are either immune or highly resistant to the virus. For example, *Arachis diogi* accessions PI 468141, PI 468142; *Arachis helodes* accession PI 468144; *A. cardenasii* accessions PI 475998, PI 476012, and PI 476013; *A. chacoense* accession PI 276235; and *Arachis paraguariensis* accession PI 468176 are highly resistant to PStV (Prasad Rao and Reddy 2005, Nigam et al. 2012). But efforts of making crosses to introduce this trait have not been successful, due to incompatibility between species.
2. *Transgenic peanut for resistance to PStV*: A practical and efficient genetic transformation and regeneration system for cultivars of peanut has been developed. Using particle bombardment technology in Australia and China, viral resistance genes have been introduced

into peanut. Also, an alternative *Agrobacterium*-mediated transformation system is investigated. RNA silencing, an intrinsic defense mechanism, has been successfully induced in transgenic peanut plants to specifically eliminate PStV RNA (Waterhouse et al. 2001, Dietzgen et al. 2004). These plants are highly resistant to PStV infection, and the resistance is stably inherited. An international collaborative research program funded by the Australian Centre for International Agricultural Research has now applied this technology to peanuts to control stripe disease in commercial peanut cultivars (Higgins et al. 2004). These plants will be particularly useful for Indonesian growers to combat a major constraint in production and may provide a source of resistance in peanut breeding programs. The genetic improvement of the major Indonesian cv. Gajah for PStV resistance is of particular significance, since this cultivar is also resistant to bacterial wilt, another economically important disease in Southeast Asia. Transgenic peanut lines carrying a CP gene of PStV and showing resistance to the virus have been developed in Indonesia (Hapsoro et al. 2005, 2007a,b, 2008). The transgene has been proven to be stabile up to seven generations of selfing (Hapsoro et al. 2007b). The transgenic peanut lines have been shown to carry the PStV CP transgene and inherited according to the Mendel law (Hapsoro et al. 2008). Therefore, these transgenic pure lines could be used as parents in a breeding program of pyramiding character of resistance to PStV and other novel characters in peanut plants. For example, peanut cultivar Gajah that is resistant to PStV obtained through genetic engineering could be combined with resistance to leaf spot disease, a nontransgenic high yielding character, through hybridization. This demonstrates that transgenic character can be treated just as nontransgenic character in a breeding program employing hybridization (Hapsoro et al. 2010). Resistance to PStV mediated by inverted repeat of the CP gene in transgenic tobacco plants had been developed (Yan et al. 2007, 2012).

Chemical Control

Since the PStV is transmitted by the aphid vectors in nonpersistent manner, managing the disease through vector control through insecticide sprays is not effective.

Cultural Control

It is advisable not to use seeds from crops infected with the PStV from the preceding crop season. Virus-free seed can be produced by growing healthy seed, tested by ELISA in areas where PStV is currently not known to occur. Moreover, in areas where aphid vectors are not likely to be active when peanut crop is young, the proportion of seed containing PStV is not likely to be high. However, if a small number of plants with PStV infection from seeds are observed, they can be rogued. Selection of large-size seeds could reduce the source of primary inoculum, and thus, this practice appears to be useful in decreasing the incidence of the peanut stripe disease. Peanut seed production should be done in the wet season when aphid populations are low and PStV incidence is negligible. Roguing of infected peanut is not effective in controlling PStV, because by the time the symptoms develop, aphids could already have transmitted the virus to other plants (Demski et al. 1993).

Regulatory Control

One way of controlling PStV is through early detection by using PStV antisera for virus detection as well as seed certification and follow-up of plant quarantine. For example, Australian peanut crops are free of the PStV, and it is important that quarantine remains effective in keeping it out. The risk of accidental introduction of the virus in imported raw peanuts is considered high, and aphids capable of transmitting it are widespread in Australian peanut crops (Persley et al. 2001). Therefore, under similar conditions in other countries too, strictly observing plant quarantine regulations to exchange only PStV-free peanut germplasm is of great significance. Seed lots for

experimental purposes should be tested by nondestructive methods before their distribution to non-infested areas. Sowing near leguminous crops or other potential hosts of PStV should be avoided (Demski et al. 1993). Growing groundnuts for seed at a distance from commercial groundnut fields is important. For example, in the United States, 100 m is regarded as a safe distance (Demski et al. 1993), whereas in China, it is 200 m (Xu and Zhang 1986). In seed production fields, roguing diseased plants when they are noticed should reduce the chance of having contaminated seed in the lot. This method may not be practical in large-scale production, but is a common practice in countries such as Thailand.

PEANUT MOTTLE

Symptoms

The symptoms of PeMoV infection can vary with cultivar, time of infection, and environment. The most common symptom, although it may not be readily noticed, is a mild mottle as irregular dark green island or mosaic on the youngest leaves of infected plants. The symptoms are not as clear on older leaves and thus can be easily missed even when the virus is in epidemic proportions in the field. The light and dark green areas of affected leaves can best be seen if leaves are held up to light. Margins of leaflets may curl up and depressions in the leaf tissue between the veins may become prominent. Plants are generally only mildly stunted, if at all. As plants mature, the symptom expression generally declines, particularly during hot and dry weather. Pods from infected plants may be reduced in size and have irregular gray to brown patches. The seed coat of affected seed may also be discolored.

Geographical Distribution and Losses

Peanut mottle disease caused by PeMoV was first reported to occur in Georgia in the United States in 1965 (Kuhn 1965). Since then, it has been reported to occur in many southeastern states of the United States, East Africa, Northeast Australia, China, India, Indonesia, Japan, Malaysia, Philippines, Bulgaria, Sudan, and Venezuela (Kolte 1984). In 2008, reports of its occurrence have been made from the Gilan Province of Iran (Elahinia et al. 2008) and Israel (Spiegel et al. 2008). Five major strains of the PeMoV as mild mottle strains (PeMoV-M1 and PeMoV-M2), severe mosaic strain (PeMoV-S), necrosis strain (PeMoV-N), and chlorotic line pattern strain (PeMoV-CLP) are known to occur, and the losses in yield caused by the disease in a particular geographical area depend on the prevalence of the particular strain of the virus, the severe mosaic strain being more damaging as reported from North Carolina in the United States reducing the yield by 41%–72% (Sun and Herbert 1972). PeMoV causes substantial yield losses in many parts of the world. In some Southeast Asian countries, yield losses up to 30%–48% have been reported, and in the Indian subcontinent, the virus is a potential threat to peanut production (Reddy 1991, Prasada Rao and Reddy 2005).

Causal Virus

PeMoV belongs to the genus *Potyvirus* in the family Potyviridae. As with other members of this virus family, PeMoV has flexuous, filamentous, nonenveloped particles ranging from 740 to 750 nm in length and 15 nm in diameter with one molecule of positive-sense, single-stranded RNA ($3.0–3.5 \times 10^6$ Da) and one coat polypeptide species (32–36,000 Da). Virus infection is often associated with intracellular cytoplasmic and nuclear inclusions, pinwheels, bundles, and laminated aggregates. It is difficult to purify the virus due to its tendency to aggregate and to be inactivated by most clarifying agents. Purified virus shows typical absorption spectrum of nucleoproteins with minimum and maximum absorbance at 246 and 260 nm, respectively (Paguio and Kuhn 1973, Kolte 1984).

Although the virus infects several species within the Leguminosae, its host range outside this family is extremely limited. PeMoV is also known by other names such as peanut green mosaic virus, peanut chlorotic mottle virus, and peanut mild mosaic virus.

Transmission

In addition to being mechanically transmissible, PeMoV is also transmitted in a nonpersistent manner by several species of aphid, including *A. craccivora*, *A. gossypii*, *Hyperomyzus lactucae*, *M. persicae*, *Rhopalosiphum maidis,* and *Rhopalosiphum padi* (Pietersen and Garnett 1992). Out of the five strains, the PeMoV-N is not transmitted by aphid. PeMoV is seed borne up to 20% in peanuts (Bashir et al. 2000). Adams and Kuhn (1977) reported that seed transmission is due to the presence of the virus in the embryo. The seed transmission, however, varies depending on the virus strain, host cultivar, time of infection, and temperature.

Diagnosis

PeMov can be initially diagnosed by its sap inoculation on the leaves of *P. vulgaris* cv. Top Crop that produces its characteristics reddish-brown local lesions. ELISA is also commonly used to detect PeMov in leaves as well as seeds (Bharathan et al. 1984, Puttaraju et al. 2001). In infected plant cells, the virus makes characteristic potyvirus cylindrical inclusions that are visible in the light microscope with proper staining. Since 2000, a new procedure known as an IC-RT-PCR method has been in use for testing large number of seed lots of peanut germplasm to detect PeMoV, and the IC-RT-PCR could be adopted to test other plants and detect other plant viruses (Gillaspie et al. 2000). In this method, a small slice is removed from each seed distal to the radicle of a 100-seed sample, the slices are extracted in buffer and centrifuged, and a portion of the supernatant is incubated in a tube that has been coated with antiserum to the PeMoV. Following immunocapture of the virus (PeMoV), the tube is washed, the RT-PCR mix (with primers designed from conserved sequences within the capsid region of the virus) is placed in the same tubes, and the test then is said to be complete. Results indicate good correlation between the virus detected by the IC-RT-PCR method and the virus detected from the same seed lots by ELISA. But the IC-RT-PCR method is more sensitive and efficient than ELISA. This method has been used for the first time as molecular evidence for the occurrence of PeMoV in China, and the phylogenetic studies done in that country reveal that PeMoV can be clustered into three groups, America, Asia, and Australia, which are found to be consistent with their geographical origins (Liu et al. 2010).

Epidemiology and Disease Cycle

Besides peanuts, this virus is known to infect several legume crops particularly soybeans (*G. max*), French bean (*P. vulgaris*), peas (*P. sativum*), and various weeds that occur in peanut fields. All these can serve as the sources of primary inoculum. In the United States, the virus is known to affect peanuts as well as soybeans. Since soybeans and peanut production areas in the United States are contiguous and overlapping in several southeastern states, the virus infection chain is maintained from one season to another. The susceptibility of several *Cassia* species both in the East Africa and in the United States indicates the presence of a potential reservoir of infection. Since 2007, PeMoV has been reported to infect Rhizoma peanuts (*Arachis glabrata*) in Georgia (Nischwitz et al. 2007). This plant is propagated by cuttings and is a perennial crop. If this virus spreads in perennial peanuts in the southern United States, this plant could become a reservoir of the virus and increase its spread to field peanut and soybean via aphid transmission. Infected seeds are also considered as the source of survival of virus. Seed samples collected from the farmers and fields in India have been found to show 2%–7% PeMoV infection (Puttaraju et al. 2001). In Zimbabwe, the Bambara groundnut (*Vigna subterranea*) seeds have been detected to be infected by PeMoV (Sibiya et al. 2002). The infected seed thus can therefore be one very important source of primary inoculum in a newly planted peanut crop.

Disease Management

Host Plant Resistance

The most promising peanut germplasm lines with resistance to PeMoV are PI 261946, PI 261949, and ICG 504, but this resistance has not been incorporated into any commercial varieties. Some accessions of wild species of *Arachis* that are resistant to PeMoV are PI 172223, 262817, 262818, 262794, 421707, 468141, 468142, 468169, 468171, 468174, 468363, 468366, 468371, and AM 3867 (Prasad Rao et al. 1993, Prasad Rao and Reddy 2005). These accessions can be of potential use in breeding for resistance to PeMoV.

Chemical Control

The spread of the disease may also be controlled by the use of effective insecticide sprays through aphid control, though the control of nonpersistently aphid-transmitted viruses as PeMoV is difficult.

Cultural Control

The use of PeMoV-free seed is the most feasible approach for control, as this prevents the disease from becoming initially established in the field. Thus, cultivars with low or no seed transmission can be of immense use in eliminating the initial source of virus inoculum. It is noteworthy to note that at least there are two peanut genotypes, viz., EC 76466 and NCAC 17133 (PI 259747), which do not show any evidence of transmission of mottle strains of PeMoV through seed (Bharathan et al. 1984, Prasad Rao and Reddy 2005). If PeMoV-free seed is used, volunteer plants must be completely removed and the field situated so that PeMoV hosts, such as clovers, southern pea, and navy bean, are at least 100 yards away. Geographical locations in peanut-growing countries, where there is low or no incidence of occurrence of the disease, are reported to be identified for virus seed production.

PEANUT CLUMP

Symptoms

Plants affected by clump disease are conspicuous in the field because of their severe stunting and dark green appearance (Figure 3.3). Initial symptoms appear on young leaflet as mottling, mosaic, and chlorotic rings, but later turn dark green with or without faint mottling as the leaves mature. Early-infected plants become severely stunted. Late-infected plants may not show conspicuous stunting but appear dark green with faint mottling on younger leaflets. Clump symptoms are similar to those of green rosette, and it is likely that the two diseases are confused in some areas of Africa where they both occur (Reddy 1991). In late-infected plants, clumping may be restricted to few branches. Infected plants become bushy and produce several flowers, but the pegs do not develop pods of normal size. Early-infected plants may not produce any pods and late-infected plants may produce poorly developed pods. These plants often occur in patches, and the disease reoccurs in the same area of the groundnut field in successive years.

Geographical Distribution and Losses

The disease first reported from Senegal in West Africa now affects peanut in several countries in Africa including Benin, Burkina Faso, Chad Congo, Gabon, Ivory Coast, Mali, Niger, Sudan, and also in South Africa and in Asia (the states of Punjab Haryana, Andhra Pradesh, Gujarat, Rajasthan and Uttar Pradesh of India, and in Pakistan) (CABI 2006, Dieryck et al. 2009).

Peanut clump virus (PCV)-infected plants do not produce pods, and yield losses in peanut grown in light sandy soils are as high as 60% even in late-infected crops (Reddy et al. 1988). Peanut clump disease is known to cause losses exceeding U.S. $40 million to peanut alone on a global scale.

(a)

(b)

FIGURE 3.3 Peanut clump caused by the peanut clump virus. Note the severe stunting with dark green leaves (a) and the mosaic mottling on young quadrifoliate leaves (b).

The virus has also been shown to infect a range of monocotyledonous plants, which include wheat, barley, maize, sorghum, foxtail millet, pearl millet, and various grassy weeds (Delfosse et al. 2002). In addition to peanut, the virus has the potential to cause crop losses at least in sugarcane, wheat, barley, maize, sorghum, chillies, and Bambara groundnut. PCV also infects such important crops as cowpea (niebe) and forage legumes (e.g., *Stylosanthes* spp.) and highlights a correlation between the countries cultivating these crops and the virus distribution, but crop losses in these hosts have not been investigated experimentally.

Causal Virus: Peanut Clump Virus

PCV that causes clump disease is characterized by straight tubular particles of two lengths, containing single-stranded RNA, precisely termed as RNA-1 and RNA-2 (Thouvenel et al. 1976, Thouvenel and Fauquet 1981a, Hemmer et al. 2003). However, isolates of the virus (PCV) causing the clump disease in West Africa are serologically distinct from the isolates that cause the clump disease in India and are referred to as isolates of IPCV. However, IPCV has been shown to resemble PCV in symptomatology, in particle morphology, as well as in other aspects. Both PCV and IPCV belong to the genus *Pecluvirus* whose members are typified by a bipartite S RNA genome and by having a fungus vector, now better known as Plasmodiophorid protozoa, the *Polymyxa graminis*. The nucleotide sequence of IPCV RNA-2 and the amino acid sequence of the CP of IPCV have been found to be 61% identical to PCV supporting the contention that the Indian and West African diseases are caused by distinct but related viruses (Naidu et al. 2000, 2003). Both viruses are known to exist as a range of strains: IPCV isolates from India have been grouped into three distinct serotypes—IPCV-H (Hyderabad), IPCV-D (Durgapura), and IPCV-L (Ludhiana)—whereas the common and yellowing strains of PCV have been recognized in West Africa (Thouvenel and Fauquet 1981b). The variability in both PCV and IPCV has been elucidated by studying the respective virus genome, and the complete nucleotide sequence of one of the two RNA species in each virus has been determined (Naidu et al. 2003). Since a polyclonal antiserum produced from one serotype usually does not detect others, a DNA probe derived from RNA has been used to detect all the known serotypes of IPCV (Reddy et al. 1994).

Diagnosis

RT-PCR technique can be used to detect PCV in the host tissue and to detect virus acquisition and transmission of PCV by the vector (Dieryck et al. 2011). *Phaseolus vulgaris* cv. Local and *Chenopodium quinoa* are found to be good diagnostic hosts. Although existence of diversity among various isolates of PCV has been reported, no attempts have been made to determine the distribution and biological characteristics of different isolates. For example, rice stripe necrosis virus (RSNV) is also transmitted by *Polymyxa* sp. and occurs in Côte d'Ivoire, Nigeria, Liberia, and Sierra Leone. It is a virus very similar to PCV, but it is not fully described; its serological and genomic properties need to be clarified, and it is possible that RSNV is a member of the *Pecluvirus*, the genus to which PCV and IPCV belong. This information is vital for the diagnosis of these viruses and for implementing the management practices. Additionally, very little is known about the diversity among the isolates of *Polymyxa* spp. in West Africa. The *Polymyxa* sp. transmitting RSNV infects rice roots, whereas this crop is not a host for *Polymyxa* sp. transmitting IPCV. The studies on the host range of both *Polymyxa* and the viruses it transmits are crucial before developing strategies for the management of this group of viruses in West Africa.

Transmission, Epidemiology, and Disease Cycle

Both PCV and IPCV are seed and soil transmitted and are vectored by the persistent, soil-inhabiting root parasite, Plasmodiophorid protozoa, the *Polymyxa graminis* f. sp. *tropicalis* (Delfosse et al. 2005, Dieryck et al. 2005, 2008, Otto et al. 2005). This fungus-like parasite survives for many years in the soil in the form of highly resistant resting spores, and clump disease occurs in patches in fields. The disease recurs when groundnut and certain IPCV-susceptible cereal hosts like pearl millet (*Pennisetum glaucum*), sorghum (*Sorghum bicolor*), wheat (*Triticum aestivum*), and barley (*Hordeum vulgare*) are grown regularly.

PCV is seed transmitted in peanut and suspected to be transmitting through the seed of a range of monocotyledonous and dicotyledonous hosts (Dieryck et al. 2005). Evidence has been obtained to show that IPCV can establish in disease-free areas if virus-containing seed from monocots is planted in soils containing *Polymyxa* species. IPCV is seed transmitted up to 11% in groundnut and

also through the seeds of finger millet, pearl millet, fox tail millet, wheat, and maize (Ratna et al. 1991). Seed transmission frequency to the extent of 4.29% has been recorded in the case of pearl millet accessions, and the virus can be detected in 96% of the root tips of pearl millet seedlings infected through seed, raising concern regarding their role in the spread of the disease. It is revealed that capsidial proteins of CPV are localized in several parts of the root apexes, notably in the root caps of pearl millet, indicating that the virus is perhaps able to multiply in parts of the apical meristems, which is uncommon to the general characteristics of plant virus infection (Otto et al. 2005). Accumulation of PCV during infection is accompanied by specific association of PCV RNA-1 encoded proteins with membranes of the endoplasmic reticulum and other organelles (Dunoyer et al. 2002).

There is some evidence that quantity and distribution of rainfall influences the incidences of IPCV-H and *P. graminis*; that is, high rainfall with temperatures ranging from 23°C to 30°C results in high incidences of the virus and *P. graminis*, and a weekly rainfall of 14 mm is sufficient enough for *P. graminis* to initiate infection for natural virus transmission (Delfosse et al. 2002).

Disease Management

Host Plant Resistance

The host resistance to PCV and IPCV could not be identified in any of nearly 10,000 *Arachis* germplasm lines. The variation in resistance/tolerance reaction in genotypes in the sick plots has been found to be due to uneven distribution of virus inoculum in the fields, which depends on the germination of resting spores of the vector *P. graminis* and environmental conditions. A reliable virus inoculation procedure is therefore essential to accurately evaluate peanut germplasm for resistance to IPCV/PCV. One such method is mechanical sap inoculation reported by Reddy et al. (2005), where infected peanut seed stored at –70°C is used as initial virus inoculum source, that is, infected seed material (1:10 w/v) is macerated in chilled inoculum buffer and immediately inoculated to French bean (*P. vulgaris* cv. Top Crop) to get IPCV/PCV-infected French bean that then should serve as the source of inoculum for further efficient transmission of the virus to peanut by mechanical sap inoculation. This method is convenient and allows reliable screening of elite peanut germplasm for resistance to various PCV/IPCV isolates in a relatively short period in comparison to soil-borne inoculum that depends on the germination of resting spores of the vector *P. graminis* (Reddy et al. 2005). On biotechnological front, molecular work is in progress to obtain one or more virus CP genes that could be possibly used to transform peanut plants to induce transgenic resistance (Reddy et al. 1994).

Chemical Control

Soil application of biocides such as Nemagon and Temik or furrow application of a systemic insecticide, carbofuran at 5 kg ai/ha, 1 week before planting, although effective, is found to be either hazardous or uneconomical (Reddy 1991, Delfosse et al. 2005).

Cultural Control

Extreme caution is essential in selecting the virus-free seed. Based on epidemiological studies, early sowing of the peanut crop, before the onset of monsoon rains and using judicious irrigation, has been shown to be simple and the most effective cultural method of reducing disease incidence in irrigated areas (Delfosse 2000). As a result of the baiting technique used to monitor IPCV and *P. graminis* infection, a trap-cropping method using pearl millet has been developed and tested successful at different sites in India. Pear millet is planted soon after the monsoon rains and uprooted in 2 weeks after germination. This permits the infection by *P. graminis* but not the development into sporosori. This is useful in reducing the inoculum load and peanut crop sown subsequently shows lower incidence of the clump disease. As *P. graminis* multiplies intensively in monocots, these should be avoided in cropping systems without peanut (Delfosse 2000). Hence, crop rotation with these crops is not to be recommended.

Soil solarization has been found to reduce the incidence of clump disease in India where well-cultivated soils are profusely irrigated before being covered with layers of transparent polyethylene sheeting for at least 70 days during summer (Reddy 1991). But the economic benefit of this method needs to be determined.

PEANUT STUNT

Symptoms

Symptoms vary depending on the host plant and host plant cultivar and the strain of the virus. In peanuts, there are various degrees of stunting, shortening of the petioles, reduced leaf size, mild mottling, and malformation of pods. Severe reduction in leaf size, especially in width, occasionally results in the complete absence of leaflet lamina. Seeds from infected plants appear deformed, frequently with a split pericarp wall, and have poor viability.

Geographical Distribution and Losses

Though the disease is considered to be of less economic importance, there have been records of epidemic occurrence of peanut stunt in the 1960s in Virginia, North Carolina, and Georgia in the United States (Miller and Troutman 1966, Herbert 1967). In 1966, about 50%–90% loss in yield due to the disease was reported in Virginia. The causal peanut stunt virus (PSV) sporadically causes a high incidence of peanut stunt disease in Hebei, Henan, and Liaoning provinces in China (Xu et al. 1992). The PSV, otherwise, is an economically important pathogen of legumes worldwide including several countries in Europe (France, Hungary, Italy, Poland, and Spain), in Asia (China, Georgia, Japan, Korea, and India), in Africa (Morocco and Sudan), and also in the United States (Subrahmanyam et al. 1992). In Kentucky as well as in the southeastern United States, PSV is widespread in forage legumes and is considered a major constraint to production and stand longevity.

Causal Virus: Peanut Stunt Virus

The PSV belongs to the family Bromoviridae. It is a member of the genus *Cucumovirus*, the type member of which is *Cucumber mosaic virus*. PSV particles are isometric or polyhedral, with a diameter of ca. 25–30 nm. The CP of PSV contains a single polypeptide with an apparent molecular weight of about 26 kDa. PSV has a positive-sense tripartite genome (designated RNA-1, -2, and -3 in order of decreasing size), largest genome 3.355 kb (RNA-1), the second largest 2.946 kb (RNA-2), and the third largest 2.186 kb (RNA-3), and has base composition being 24% G, 26% A, 21% C, and 29% U. In addition to the genomic RNAs, the virions also encapsulate a fourth RNA (called RNA-4), which is a subgenomic RNA that functions as mRNA for the viral CP (Naidu et al. 1995, Suzuki et al. 2003). Three types of native particle exist, each consisting of the same protein shell, yet containing different RNA species. One type of particle contains genomic RNA-1, another contains RNA-2, and the third contains genomic RNA-3 and subgenomic RNA-4. However, all the particles have the same sedimentation coefficient (S20w). Nongenomic nuclei found in virions are subgenomic mRNA that encodes the CPs, are named RNA-4, and are of 0.986 kb. Naturally occurring virions of PSV may also package a fifth RNA designated as SatRNA along with their genomic and subgenomic RNAs. PSV-associated SatRNAs are linear, single-stranded RNA molecules, ranging in size from 391 to 393 nucleotides. PSV SatRNA has essentially no sequence homology with its helper virus (i.e., PSV) genomic RNAs. Depending on the PSV strain and host species involved, SatRNAs can modulate the symptoms caused by PSV. PSV supports the replication of its SatRNAs but not those associated with *Cucumber mosaic virus*.

Infectivity of the virus in sap is lost between 51°C and 56°C temperature when exposed to 10 min, between 10^{-4} and 10^{-5} dilutions and between 4 and 24 h at room temperature (Kolte 1984).

Several strains of the PSV-infecting peanut have been described. PSV-V from Virginia and PSV-W from Washington have been described from the United States, and these have been referred to as eastern (PSV-E) and western (PSV-W) American strains of PSV, respectively (Naidu et al. 1995). Some other strains such as PSV-P (Polish) from Poland (Obrepalska-Steplowska et al. 2008), PSV-RP (Robina) from Hungary (Kiss et al. 2009), and PSV-Mi from China (Xu et al. 2004) have been reported.

Diagnosis

Plants suspected of a viral infection should be sent to a plant diagnostic laboratory where the presence or absence of the virus can be confirmed by serological double-antibody sandwich-ELISA and an indirect ELISA, RT-PCR, or host range tests (Dai et al. 2011). Antiserum and sequence data are available for this virus. The PSV produces chlorotic lesions followed by systemic mottle on *Cucumis sativus* and *P. vulgaris* cv. Top Crop, Kentucky wonder, and Bountiful. The virus also produces chlorotic lesions on *C. amaranticolor* and *Cyamopsis tetragonoloba* (Kolte 1984).

Transmission, Epidemiology, and Disease Cycle

All three genomic RNAs, but not subgenomic RNA 4, are essential for infection, and its CP, as in other viruses, plays an important role in many processes during viral life cycle and has great impact on the infectivity (Obrepalska-Steplowska et al. 2008). The CP gene is essential and sufficient for the production of unusual cytoplasmic ribbonlike inclusions that is a strain-specific trait of the virus (Bashir et al. 2004). PSV is transmitted from plant to plant by several species of aphids (*A. craccivora*, *A. spiraecola*, and *M. persicae*) in a nonpersistent manner. It can also be transmitted by mechanical inoculation. It has been shown to be transmitted by seeds in peanuts at a very low level, but this is not considered to be very important to the spread of this virus. The virus can be introduced into a susceptible field crop by aphids from a nearby reservoir (infected perennial hosts like clover, alfalfa, or perennial peanuts) and then is spread further into the field by aphids (Blount et al. 2002). It can be spread in perennial crops by harvesting (mechanical transmission) and possibly by the root grafts.

Disease Management

In the absence of any satisfactory source of resistance among peanut germplasm or PSV-resistant cultivar, good control of the disease appears to be only through vector control by means of insecticides. Incidence of the disease can be brought under control if peanut fields are kept away from clover fields.

It is worth mentioning that there is an occurrence of attenuating PSV SatRNAs that, when coinoculated with PSV, elicit the suppression of virus replication and spread. The symptom-attenuating properties of SatRNAs have been successfully exploited in the development of SatRNA-mediated transgenic protection against *Cucumber mosaic virus* and tobacco ring spot virus, and there is a possibility of developing this technology-based transgenic peanut for the management of the stunt disease (Naidu et al. 1995).

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4 Other Diseases of Peanut

BACTERIAL WILT OF PEANUT

Symptoms

Affected young plants show conspicuous and sudden rapid wilting, and death of roots becomes evident. The whole plant or only a branch of the plant may be wilted. Leaflets of affected plants curl up at the ends and become slightly flaccid, while leaves retain their green color with little fading of normal green color (Mehan et al. 1994). Veins become greener than the lamina, giving leaflets a stripped appearance. A unilateral effect, as can be observed under natural conditions, is noted and only one branch may be affected under artificial conditions also. Wilt symptoms can be observed 3 weeks after sowing, the peak of disease occurrence being 40–50 days after sowing. Plants may wilt entirely over days under conducive conditions, such as after high temperatures. There can also be latent infection. This disease can be distinguished from other wilt diseases by placing infected tissue, freshly sectioned, into water to observe masses of bacterial ooze streaming out. The rapid wilting also distinguishes this from fungal wilts.

Bacterial oozing may be seen on root, stem, and lower branches and the oozing becomes evident as streaks of brown or black discoloration. The affected tissues then become black and show necrosis. When young plants are infected, the pods may remain small, or pods of such plants become wrinkled and may show rotting. When external symptoms are not evident, the infection can be detected in cross sections of stems and roots. Brown pigment formation in host tissue in the cut xylem and pith regions is considered a diagnostic criterion for the identification of the disease.

Geographical Distribution and Losses

Bacterial wilt (BW) disease (*Ralstonia solanacearum*) is widely distributed in tropical, subtropical, and some warm temperate region in the world and poses a great threat to peanut production in China and Southeast Asia including Indonesia and Vietnam (Shan et al. 1998, Doan et al. 2006). More than 10% of the area under peanut is affected in southern China. In Shandong Province in China, where large-seeded cultivars dominate, BW is a very serious problem in Rizhao and Linyi. The disease is reported to affect about 35,000 ha crop area in these two areas (Zhang et al. 2008). Yield reduction generally ranges from 10% to 20%; however, in heavily infested field, over 50% yield losses are not uncommon. The disease is more severe in lands not used for paddy rice in Southeast Asia. In extreme cases, the disease may even cause total crop losses (Mehan et al. 1994, Yu et al. 2011). Although the disease had been reported from several African countries in the 1930s and 1940s, it is not considered economically important there with the exception of Uganda, East Africa (Mehan et al. 1994, Elphinstone 2005, Mace et al. 2007). The worldwide distribution of *R. solanacearum* has been summarized by Commomwealth Agricultural Bureaux International and the European and Mediterranean Plant Protection Organization in the updated series of distribution of maps of plant diseases (Schell 2000).

Pathogen: *Ralstonia solanacearum* (Smith) Yabuuchi et al.

R. solanacearum is the causal pathogen of BW, formerly known as *Pseudomonas solanacearum* E.F. Smith, with similarities in most aspects except that it does not produce a fluorescent pigment like *Pseudomonas* (Fegan and Prior 2005). *R. solanacearum* is a Gram-negative, non-spore-forming, rod-shaped, strictly aerobic bacterium that is 0.5–0.7 × 1.5–2.0 μm in size. For most strains, the optimal growth temperature is between 28°C and 32°C, and it does not grow above 41°C. On solid agar media, individual bacterial colonies are usually visible after 36–48 h growth at 28°C, and colonies of the normal or virulent type are white or cream colored, irregularly shaped, highly fluidal, and opaque. Occasionally, colonies of the mutant or nonvirulent type appear; these are uniformly round, smaller, and butyrous, or dry. A tetrazolium chloride (TZC) medium can differentiate the two colony types. On this medium, virulent colonies appear white with pink enters and nonvirulent colonies are a uniform dark red (Champoiseau et al. 2009).

Variability and Pathotypes in *R. solanacearum* Species Complex

R. solanacearum is classified into five races based on the hosts affected and five biovars based on the ability to use or oxidize several hexose alcohols. Race 1 strains (biovars 1, 3, and 4) are pathogenic to a broad range of hosts, including tomato (*Solanum lycopersicum*), tobacco (*Nicotiana tabacum*), and peanut (*Arachis hypogaea*) and occur in Asia, Australia, and Americas; race 2 strains (biovars 1 and 3) have a more limited host range than race 1 and infect banana (*Musa acuminata*), plantain (*Musa paradisiaca*), heliconia (*Heliconia* spp.), and other plants in the Musaceae family and occur in the Caribbean, Brazil, and the Philippines; race 3 strains (biovar 2) occur in cool upland areas in the tropics and cause severe wilt in potato (*Solanaum tuberosum*), tomato, and geranium (*Geranium* spp.) and occur worldwide, but it is not generally reported in North America covering the United States and Canada and is, therefore, the focus of sanitation and plant quarantine of management practices to prevent the introduction of or spread of the pathogen; race 4 strains (biovars 3 and 4) infect ginger and occur in Asia; and race 5 strains infect mulberry (*Morus alba*) and occur in China (Denny and Hayward 2001). Pathogen diversity and the relationship among races, biovars, and phylotypes have been described (Chen et al. 2000, Alvarez 2005, Fegan and Prior 2005), but this classification system has proved to be unsatisfactory. There are no laboratory tests to define the *race* of an isolate because host ranges of strains are broad and often overlap. The biovars do not correspond to phylogenetically coherent groups, with the exception of biovar 2A, which corresponds to R3bv2.

A phylogenetically meaningful classification scheme has now been developed based on DNA sequence analysis. This scheme divides the species complex into four phylotypes that broadly reflect the ancestral relationships and geographical origins of the strains. Phylotype I strains originated in Asia, phylotype II strains originated in the Americas, phylotype III strains in Africa, and phylotype IV strains in Indonesia. Phylotypes are further subdivided into sequevars based on the sequence of the endoglucanase gene. Multilocus sequence typing and other analyses have confirmed that this system of classification reflects the phylogeny of the group (Prior and Fegan 2005). The 5.8-megabase (Mb) genome of *R. solanacearum* has been completely sequenced. The genome encodes many proteins potentially associated with their role in pathogenicity (Salanoubat et al. 2002).

Diagnosis

Diagnosis can be determined from a section of stem pruned from near the base of a suspect plant. Immediately after pruning the stem, suspend it in a glass of clean water for several minutes. Milky threads will begin to leak from the stem and the water will quickly become white if BW is present. A diagnostic schedule involving direct seed plating and grow-out of peanut seeds for 4 weeks and the leaf bits and twig pieces on tetrazolium chloride agar (TZCA) can be useful for the detection

of BW infection in imported peanut germplasm seeds (Anitha et al. 2004). Serological methods are generally quick and reliable but suffer from problems with specificity or sensitivity or both. Additionally, they do not distinguish live cells from dead cells. A number of *R. solanacearum*–specific nucleic acid–based methods that use the polymerase chain reaction (PCR) amplification can detect both living and dead cells, which are more specific and simple than serological approaches.

Epidemiology and Disease Cycle

The bacterium survives in the soil and can maintain infectious populations over several years. Alternative weed hosts may also play a role in survival and over seasoning. The pathogen infects roots through wounds and colonizes the vascular tissue causing plugging of the xylem and leaf wilting. There is often an association between nematode infection and BW, where the nematodes create wounds in the root tissue to allow an entry point for the bacterium to infect the plant. The bacteria get access to the wounds partially by flagellar-mediated swimming motility and chemotaxis attraction toward root exudates (Yao and Allen 2006). Unlike many phytopathogenic bacteria, *R. solanacearum* potentially requires only one entry site to establish a systemic infection that results in the development of BW. After invading a susceptible host, *R. solanacearum* multiplies and moves systemically within the plant before BW symptoms occur. When the pathogen gets into the xylem, tyloses may form to block the axial migration of bacteria within the plant that may lead to vascular dysfunction. *R. solanacearum* possesses genes for all six protein secretion pathways that have been characterized in Gram-negative bacteria; the best known is Type III secretion system (T3SS or TTS), which secretes infection-promoting effect or proteins (T3 Es) into the host cells. Despite being just one of several secretion systems, the T3SS is necessary for *R. solanacearum* to cause disease. Molecular determinants involved in pathogenicity, virulence, and host range specificity have been described using representative strains of the main phylogenetic groups of *R. solanacearum* (Genin and Denny 2012).

Temperature is a major determinant in the distribution of this pathogen, which is widespread in tropical, subtropical, and warm temperate regions where the mean soil temperature is greater than 15°C (Hayward 1991). Wet soil increases the incidence of disease and water movement contributes to the dissemination of inoculum with water movement. The incidence and rate of wilting therefore increase with high temperatures and soil moisture. Continuous cropping of susceptible plants will also favor infection. Young plants are more rapidly diseased than older ones. BW is more prevalent in slightly acid to acid soils. For example, the disease is more prevalent in the acid soil area south of the Sichuan Basin and yellow alluvial soil, sandy alluvial soil, but no peanut BW occurs in purple soil in China (Chui et al. 2004). The pathogen is disseminated by contaminated farming equipment, in soil on tires and footwear, drainage water carrying inoculum through the soil, infested seed, and plants raised in infected soil, spreading the pathogen to new areas (Ziang et al. 2007).

Disease Management

Host Plant Resistance

Compared to many other crops, a relatively broad genetic diversity of resistance to BW has been found in the cultivated peanut, and the development of BW-resistant peanut cultivars has been more successful. Planting resistant cultivars is deemed as the sole economically viable means for effective control (Ding et al. 2011, Tang and Zhou 2000, Zhou et al. 2003, Yu et al. 2011). Some of the BW-resistant peanut genotypes mostly reported from China are R15, R16, R87, R106, K81 (Wang et al. 2009), Xiaohongmao (Liao et al. 2003), Ju and Zhonghua 6 (Liu et al. 2011), and Yuanza 9102 (Bang et al. 2011), and moderately resistant ones are Jihua 1012, Quancha 10, Quancha 646, Yucyou 193, and 38F5-45-21-CS1 (Yuan et al. 2002). It is significant that some of the highly

resistant landraces have been identified in dragon-type peanuts mostly related to *A. hypogaea* subsp. *hirsuta* collected from South China where BW is generally serious. The dragon varieties have had been traditionally cultivated in many regions of China for at least 600 years before the varieties of three other types (Virginia, Spanish, and Valencia) were introduced in the late nineteenth century. Interestingly, all the BW-resistant landraces had been from South China and no BW-resistant germplasm has been collected from the BW-free north regions of China. The evolution of resistance, therefore, seems to be associated with regional disease pressure, and the BW in South China must have been a major factor influencing the natural selection of the dragon lines in the region. The resistance to BW in tested dragon lines is dominant, and the degree of dominance is higher in the dragon lines compared to the Spanish- or Valencia-resistant genotypes, and both additive and dominant genes are involved in resistance of dragon to BW, though a significant cytoplasmic effect is associated with the resistance in dragon types (Liao et al. 1986, Shan et al. 1998). As the dragon lines possess some other desirable traits such as drought tolerance, good flavor, and high oleic and linoleic acid ratio, they appear to be more promising in improving resistance to BW in peanuts (Muitia et al. 2006). However, latent infections (infection without visible symptoms) by *R. solanacearum* have been found in some resistant cultivars/dragon-type landraces that affect root proliferations and reduce symbiotic nitrogen fixation and tolerance to drought and yield, which explain the low productivity of the crop in infested areas, and this becomes a challenging task to the breeders (Liao et al. 1998, Huang et al. 2011, Jiang et al. 2013).

Most of the resistant germplasm lines identified are small-seeded genotypes with low yield potential; transferring BW resistance to high-yielding adapted peanut cultivars has therefore become an urgent task (Yu et al. 2011). A large-seeded peanut cultivar Rihua 1 is resistant to BW (Zhang et al. 2008, Ding et al. 2012).

Understanding the mechanism underlying BW resistance at the molecular level should hasten the breeding process (Liao 2001, Chen et al. 2008a). The DNA polymorphism among the promising peanut genotypes resistant to BW has been assessed by simple sequence repeats (SSR) and amplified fragment length polymorphism (AFLP) analysis. There is enough polymorphism in the peanut genotypes with BW resistance based on SSR and AFLP analysis (Jiang et al. 2006, 2007, Mace et al. 2007). Thus far, in peanut, there have been several reports regarding the identification of DNA markers related to BW resistance (Yu et al. 2011). Although several DNA markers related to BW resistance have been identified, the map distances are too large to be used in peanut-breeding programs (Yu et al. 2011). Peng et al. (2011) identified 119 transcription-derived fragments (TDFs) after root wounding inoculation with *R. solanacearum*, from Yuanza 9102 (a Spanish-type peanut cultivar with BW resistance) and Zhonghua 12 (a susceptible Spanish-type peanut cultivar) using cDNA-AFLP, and further studied their expression patterns. A total of 98 TDFs have been cloned and sequenced.

Chemical Control

The use of chemicals, antibiotics, and soil fumigation has shown little effect on the control of BW disease.

Cultural Control

In regions where the disease is endemic, cultural methods appear to be effective under some conditions for reducing bacterial population of *R. solanacearum* and subsequent disease control. Crop rotation of at least 2–5 years involving different nonhost crops particularly paddy rice, maize, and sugarcane may be used for significant disease reduction (Machmud and Hayward 1993, Nawangsih et al. 2012). Intercropping can be better for small farmers as cultivation of beans/maize can reduce disease incidence. Controlling weeds that have the potential to serve as inoculum reservoirs in conjunction with crop rotation can also be effective. Control of root-knot nematode population and cultural practices that minimize root damage can also reduce BW severity. Modification of soil pH by using a combination of organic amendment, fertilizers, and soil solarization is also effective in disease control (Machmud and Hayward 1993, Nawangsih et al. 2012).

Biological Control

Pseudomonas fluorescens strains B16 and VK18 and that of *Bacillus subtilis* strain B11 have been identified as promising biocontrol agents for BW control, the B16 strain yielding more effective results in increased yield of peanut by 0.7–0.94 tons/ha. B16 has a positive effect on the growth and yield of peanut and can replace 20% mineral nitrogen, phosphorus, potassium (NPK) fertilizer without significant changes in crop yield under Vietnam conditions (Doan et al. 2006). Similar results have been obtained with other strains of plant growth–promoting bacteria in the control of BW in Indonesia (Nawangsih et al. 2012). An endophytic *Bacillus amyloliquefaciens* strain BZ6-1, isolated from the stem of healthy peanut plants from *R. solanacearum*–infested fields, has been found to suppress *R. solanacearum* greatly, and the field trials demonstrate the control efficiency of strain BZ6-1 against BW by 62.3% (Wang et al. 2011).

ROOT KNOT OF PEANUT

Symptoms

Under field conditions, areas of root-knot nematode-affected peanut plants are usually round to oblong in shape, and rows of infected plants may never overlap as those of healthy plants. It is not uncommon for plants to wilt and eventually die in areas where nematode populations are high. Foliar symptoms of the root-knot disease may be expressed anytime during the growing season. These symptoms of nematode damage on peanut plants include stunting, yellowing, wilting, and even plant death. Generally, however, root-knot nematode damage symptoms are most evident in a peanut crop beginning about 100 days after planting and during or after periods of hot weather. Stunting of the plants results as the nematode larvae feed in the vascular system of the peanut, causing the formation of giant cells that disrupt the vascular system. Affected plants, when uprooted, show the presence of galls on pods and roots. The feeding roots are deformed. The galls on the roots usually are similar in size and shape to the nodules formed by nitrogen-fixing bacteria. On pods and pegs, the galls are corky and variable in shape.

Geographical Distribution and Losses

Root-knot nematodes (*Meloidogyne* spp.) are among the most serious plant pests in the world. Several species of root-knot nematodes are pathogenic on peanut and cause considerable yield loss annually. Of these, *Meloidogyne arenaria* (Neal) Chitwood race 1, *M. hapla* Chitwood, and *M. javanica* (Treub) Chitwood race 3 are the major pathogenic species of peanut (Abdel-Momen and Starr 1997, Minton and Baujard 1998; Koenning et al. 1999). These three species are known to occur in many peanut-producing regions, including North, Central, and South America, Africa, Asia, Europe, and Australia. *M. arenaria* and *M. javanica* are common in warm peanut-growing regions, whereas *M. hapla* occurs mainly in cool regions. In the United States, *M. arenaria* and *M. hapla* exist throughout the peanut-producing areas. *M. arenaria* is the predominant species parasitizing peanut in the southern regions, especially in Alabama, Florida, Georgia, Texas, and South Carolina in the United States where up to 40% of the fields are infested and yield losses in heavily infested fields can exceed 30%. All three species may cause significant losses in the yield and quality of peanut (Abdel-Momen and Starr 1997). Individual peanut fields heavily infested with the root-knot nematode have sustained yield losses greater than 75%. *M. hapla* is the most prevalent species in more northerly states, including North Carolina, Virginia, and Oklahoma (Koenning and Barker 1992). Populations of *M. javanica* parasitic on peanut are common in Egypt (Tomaszewski et al. 1994) and India (Sharma et al. 1995), but they are rare in the United States, having been described from only a few fields in Florida, Georgia, and Texas (Lima et al. 2002). Only one root-knot nematode, *M. arenaria* race 1, is a major nematode pest of peanut, and unlike most plants, peanut is a poor host or a nonhost to other commonly found root-knot nematodes in Florida (*M. incognita* and *M. javanica*).

Pathogen: (*M. arenaria*) Life Cycle

The egg of a root-knot nematode develops into a vermiform first-stage juvenile that undergoes one molt into a second-stage juvenile. The second-stage juvenile hatches from the egg, moves freely in the soil, penetrates the root just behind the root cap, migrates intercellularly in the root, and establishes a feeding site within the developing vascular cylinder. As it feeds on the nematode-induced giant cell system, the second-stage juvenile loses its mobility and begins to increase in girth. After it has imbibed a sufficient quantity of sustenance, the flask-shaped second-stage juvenile molts three times without feeding and matures into a saccate adult female. Females of *M. arenaria* reproduce by mitotic parthenogenesis; as soon as they are mature adults, they begin producing eggs. The second-stage larvae that cannot find a suitable host plant can survive in the soil up to 18 months unless affected by adverse conditions or attacked by predacious fungi.

Male second-stage juveniles undergo metamorphosis during the third molt into elongate vermiform fourth-stage juveniles. The fourth-stage juvenile male remains enclosed in the cuticle of the second and third stages where it molts again to form an adult vermiform male. The male escapes from the cuticles and the root system. It moves freely in the soil, not feeding, only mating with mature adult females. As populations of *M. arenaria* reproduce by mitotic parthenogenesis, males serve no reproductive function.

The length of one generation of *M. arenaria* is greatly affected by temperature. At very high temperatures (>29°C), the life cycle takes approximately 3 weeks, but at very cool temperatures, it can be extended to 2–3 months.

Diagnosis

Morphology of perineal patterns, shape and measurements of the stylet of the female, shape and measurements of the head and stylet of the male, and measurements of the second-stage juveniles are useful characters for species identification. Additional host range tests may be necessary to confirm the identification of the species and determination of the host race. Hosts of *M. arenaria* in the North Carolina differential host range test include tobacco (*Nicotiana tabacum* cv. NC95), pepper (*Capsicum annuum* cv. California Wonder), tomato (*Lycopersicon esculentum* cv. Rutgers), and watermelon (*Citrullus vulgaris* [*C. lanatus*] cv. Charleston Gray). Host race 1 populations infect and reproduce on peanut, whereas host race 2 populations do not.

A DNA probe that is specific for *M. arenaria* has been developed and may be useful for the diagnosis of this species. Cytological and biochemical characterization provide additional characters for the identification of *M. arenaria*.

Disease Management

Host Plant Resistance

Resistance to *M. arenaria* and *M. javanica* is highly correlated, indicating that, in many peanut genotypes, the same gene or genes may confer resistance to both species, or the resistance genes for each species are closely linked. But resistance to *M. hapla* is not correlated with resistance to *M. arenaria* or *M. javanica*. The mechanisms of resistance to *M. hapla* may be different from that of *M. arenaria* (Dong et al. 2008). The resistance genes in peanut may be related to differential recognition by the plant of the three *Meloidogyne* spp. Resistance to all three *Meloidogyne* spp. exists within cultivated peanut (*A. hypogaea*), either with or without introgressed genes from wild species (Simpson 1991). Breeding cultivars with resistance to root-knot nematode, however, has been slower because no meaningful resistance has been found in the peanut germplasm collection of *A. hypogaea*. Genes conferring resistance to peanut root-knot nematode have not been found in cultivated peanut, but a number of other *Arachis* spp. have been identified that are highly resistant or immune to the peanut root-knot nematode. Successful crosses to transfer a high level of nematode resistance

into *A. hypogaea* have been reported by several researchers (Starr et al. 1995, Garcia et al. 1996, Church et al. 2000, Muitia et al. 2006). The resistance had been obtained from a wild species *Arachis cardenasii*. Two peanut germplasm lines "GP-NC WS 5 and GP-NC WS 6" have been derived from *A. hypogaea* × *A. cardenasii* interspecific cross (Stalker et al. 2002). The first of the two root-knot nematode-resistant peanut cultivars developed by the Texas Agricultural Experiment Station are (1) "COAN" released in 1999 (Simpson and Starr 2001) and (2) "NemaTAM" released in 2002 (Simpson et al. 2003). The resistance in COAN is controlled by a single dominant gene and is expressed as a reduction in nematode reproduction. Although nematodes invade the roots of COAN, most emigrate from the roots, but the few that remain in the roots develop to reproductive adults (Bendezu and Starr 2003). NemaTAM has greater yield potential than COAN and possesses the same level of resistance to the peanut root knot. Both COAN and NemaTAM have been proven resistant to the peanut root-knot nematode in southeastern United States. Neither peanut variety, however, could be successfully grown in southeastern United States because they are highly susceptible to tomato spotted wilt virus (TSWV). Later, some promising peanut germplasm lines with resistance to both the root knot and TSWV could be successfully developed (Holbrook et al. 2003), and in 2008, the USDA released a cultivar, Tifguard, which has resistance to both TSWV and root-knot nematode (Holbrook et al. 2008b). Similarly, another runner-type peanut germplasm line TifGP-1 (PI 648354) is reported to be resistant to both the root-knot nematode and TSWV (Holbrook et al. 2008a). The root-knot nematode resistance present in Tifguard is derived from the single dominant gene in COAN. The University of Georgia and University of Florida field trials have found excellent root-knot nematode resistance with the Tifguard cultivar and good final peanut yields in root-knot-infested fields. Interaction between root-knot nematode *M. arenaria* and *Cylindrocladium* black rot (CBR) fungal pathogen *Cylindrocladium parasiticum* in runner peanut genotypes such as "C724-19-15," "C724-19-25," and "Georgia-O2C" that possess different levels of resistance to nematode and *C. parasiticum* reveals that *C. parasiticum* greatly increases mortality on "C724-19-25" and "Georgia-O2C" but not on "C724-19-15" in the presence of *M. arenaria*, indicating that root-knot resistance in peanut can be broken down due to fungal infection caused by *C. parasiticum* (Dong et al. 2009). The peanut germplasm line "TifGP-2," a nematode-susceptible sister line of nematode-resistant *Tifguard*, that is, peanut closely related sister lines with and without nematode resistance, can be valuable research tools to obtain better understanding of the interactions of nematodes with other pathogens of peanut (Holbrook et al. 2008c, 2012).

Chemical Control

Nematicides have often been used for limiting the damage that nematodes cause on plants. Nematicides are usually used as a soil treatment before planting. However, a few nematicides can be applied after planting. These chemicals are relatively expensive and they require costly equipment and trained personnel to apply them. Peanut crops that are good hosts of *M. arenaria* can be protected with soil application of nematicides such as aldicarb at 31 g ai/100 m row (Timper et al. 2001). The effectiveness is dependent on adequate amount of soil moisture. If an optimum amount of water is available, the optimum effect is achieved; if too much or too little water is present, very little control is achieved.

Cultural Control

Meloidogyne species are obligate parasites and populations decline rapidly in the absence of a host. Rotation of susceptible host crop plants with those that are immune or poor hosts is a useful way to reduce the effect that *M. arenaria* has on plant growth. Unfortunately, the nonhost, when it does occur, is usually less profitable than the susceptible crop. *M. arenaria* has a very large host range, and nonhosts or cultivars that have been reported resistant should be used with caution because of the innate variability that occurs in the root-knot nematodes. Switchgrass (*Panicum virgatum*), for example, do not support the population of root-knot nematode but support the population of nonparasitic nematodes (Kokalis-Burelle et al. 2002). Populations of *M. arenaria* are lower in peanut

in the cotton–cotton–peanut than in peanut–peanut–peanut, corn–corn–peanut, or bahiagrass–bahiagrass–peanut cropping systems (Timper et al. 2001). Other agronomic and economic factors are also important in the selection of a rotation crop. An adequate weed control program is absolutely necessary for a crop rotation scheme to be effective because many weed species serve as suitable hosts. While good crop rotation should be continued to reduce all peanut diseases, the advent of resistant peanut varieties will help reduce the need for costly nematicides in peanut production. Soil organic amendment with crab shell chitin has been found not only to reduce *M. arenaria* population but also enhances soil microbial activity and promotes plant growth by 67.7% resulting in reduction in root-knot index on peanut, the number of galls per plant being negatively correlated with the accumulation of total phenols and activities of chitinase and peroxidase subsequently increasing the yield of peanut over 56% (Kalaiarasan et al. 2008b).

Biological Control

Numerous attempts have been made to control root-knot nematodes with parasitic and predacious organisms or various organic amendments, with varying degrees of success. Naturally occurring organisms, such as *Pasteuria penetrans*, which are obligate parasites of *Meloidogyne*, may prove to be effective for biological control (Timper et al. 2001). Peanut seed treatment with *P. fluorescens* at 10 g/kg of seed inhibits *M. arenaria* development in the peanut roots due to reduced and poor development of giant cells (Kalaiarasan et al. 2008a). Possibility of use of facultative Gram-negative symbiotic bacteria belonging to the *Xenorhabdus* species isolated from the gut of the nematode *Steinernema riobrave* is indicated in the control of root-knot nematode of peanut (Vyas et al. 2008). Metabolites of *Xenorhabdus* species so obtained from *S. riobrave* contain the proteins of high molecular weight, 76–90 kDa apart from regular proteins, and appear to play a role in the suppression of root-knot development in peanut.

PEANUT WITCHES' BROOM

Axillary buds of affected plants proliferate to produce numerous small stuff leaves, and the internodes are reduced. The pod stalks (gynophores) grow upward, showing a loss of positive geotropism resulting in the loss of pod formation. The peanut witches' broom (PnWB), first discovered in a geographically isolated area, the Penghu Islands, in 1975 in Taiwan and now reported or suspected to be prevalent in most Asian countries, is a plant disease associated with plant pathogenic phytoplasmas.

Phytoplasmas are a group of phytopathogenic bacteria that are transmitted by sap-feeding leafhopper insect vectors. Phylogenetically, phytoplasmas are related to the animal pathogenic mycoplasmas. Both groups are unique among bacteria in their lack of cell wall and are assigned to the class Mollicutes. However, unlike mycoplasmas that can be cultured and are amenable to genetic manipulations, the *in vitro* cultivation of phytoplasmas has remained unsuccessful despite decades of efforts. The inability to culture phytoplasmas outside of their host has resulted in the designation of the *Candidatus* (*Ca.*) status to their taxonomic assignment and also greatly hampered the research progress in characterizing these pathogens. The PnWB is caused by *Ca. phytoplasma asteris*–PnWB group 16SrII, and it is the first representative of the 16SrII group (Chung et al. 2013).

For the detection and identification of phytoplasmas, polyclonal and monoclonal antibodies, DNA probes, and PCR primers have been developed for various phytoplasmas (Chen and Lin 1997). Besides detection, extrachromosomal DNA, insertion sequence, and various genes of PnWB phytoplasma have been cloned (Chuang and Lin 2000, Wei and Lin 2004, Chi and Lin 2005, Chu et al. 2006, Chen et al. 2008b). With advancement in genomic science, genome sequence has been adopted as a powerful tool to characterize the gene contents of the uncultivated bacteria. Four open reading frames (ORFs) have been identified in the order of hrcA, grpE, dnaK, and dnaJ through the PCR-based technique. Chromosomal arrangement of these genes in PnWB phytoplasma is identical to those of aster yellows witches' broom phytoplasma, onion yellows phytoplasma, and other bacteria phylogenetically related to phytoplasma (Chu et al. 2007a,b). It is also indicative that three

rpoD homologous sigma factor genes may exist in PnWB phytoplasma (Chen et al. 2008b). Whole-genome shotgun sequencing of PnWB phytoplasma has also been done (Chung et al. 2013).

RecA protein, the product of recA gene, a key protein involved in DNA recombination and DNA repair of eubacteria, has been cloned and analyzed from phytoplasma-associated PnWB. Gene organization, the nucleotide sequence, and a sequence in the conserved regions of the ORF are similar to those of the other recA genes of eubacteria. Therefore, this gene from phytoplasma-associated PnWB is identified as a putative recA gene (Chu et al. 2006).

Phytoplasmas related to the *Ca. phytoplasma asteris*–PnWB group have been found to cause symptoms of phytoplasma diseases like leaf roll, rosetting, shoot proliferation, and phyllody in Japanese plum trees (Zirak et al. 2009), sweet cherry (Zirak et al. 2010) in Iran, and sesame in Turkey (Cengiz et al. 2013).

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Section III

Rapeseed–Mustard

Rapeseed–mustard is an important group of edible oilseed crops constituting oilseed Brassicas and crucifers, namely, *Brassica juncea*, *Brassica rapa* subsp. *trilocularis* (yellow sarson), *Brassica rapa* subsp. *dichotoma* (toria and brown sarson), *Brassica napus* (oilseed rape), *Brassica carinata* (Karan rai), *Brassica nigra* (Banarasi rai), and *Eruca vesicaria* subsp. *sativa* Miller (*taramira*). These cultivated *Brassica* has two types of genomes, that is, the diploid (elementary) and amphidiploid genomes. The elementary species include *Brassica rapa* (AA, 2*n* = 20,468-516 Mbp), *B. nigra* (BB, 2*n* = 16,468-760 Mbp), and *Brassica oleracea* (CC, 2*n* = 18,599-618 Mbp), and the DNA sequence variation reveals 51.2 Mb of the *Brassica* A and C genomes based on 10 diverse rapeseed–mustard genotypes (Ahmad et al. 2002, Clarke et al. 2013).

Globally, on 36.4 m ha, 72.5 mt of rapeseed–mustard seed is produced (FAO 2014). However, a wide gap exists between the potential yield and that realized at the farmers' field, which is largely because of the number of biotic and abiotic stresses to which the rapeseed–mustard crop is exposed. Diseases are major hurdles toward achieving higher production in rapeseed–mustard. The intensive cultivation of the crop with more inputs has further compounded the problem, and now the incidence of diseases and insect pests have become more frequent and widespread. Severe outbreak of diseases deteriorates the quantity and quality of seed and oil content drastically in different oilseed *Brassica* crops. The expression of full inherent genetic potential of a genotype is governed by inputs that go into the production system. This can be very well illustrated with examples that involve disease management of rapeseed–mustard. The loss in oilseed crops due to biotic stresses is about 19.9%, out of which diseases cause severe yield reduction at different growth stages. Various plant pathogens are reported to distress the crops. Among them, 18 are considered to be economically important in different parts of the world. To overcome such losses, it is essential to know the causal agents, their behavior, and the means to attack the vulnerable phase of the pathogen.

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5 Rapeseed–Mustard Diseases

Alternaria BLIGHT

Symptoms

Symptoms of the disease are characterized by the formation of spots on leaves, stem, and siliquae. The pathogen has been reported to affect seed germination and quality and quantity of oil (Meena et al. 2010a). On seedlings, symptoms include dark stem lesions immediately after germination that can result in damping-off or stunted seedlings. Generally, disease appeared at 40–45 days after sowing (DAS), and most critical stages have been reported at 75 and 45 days of plant growth (Meena et al. 2004). Spots produced by *Alternaria brassicae* appear to be usually gray in color compared with black sooty velvety spots produced by *Alternaria brassicicola*. Spots produced by *Alternaria raphani* show distinct yellow halos around them. However, the symptoms may vary with host and environment. Symptoms are first visible on lower leaves with appearance of black points, which later enlarge to develop into prominent, round, and concentric spots of various sizes. With progress of the disease, symptoms appear on middle and upper leaves with smaller-sized spots (Figure 5.1), when defoliation of lower leaves occur. Later, round black conspicuous spots appear on siliquae (Figure 5.2) and stem. These spots may coalesce leading to complete blackening of siliquae or weakening of the stem with the formation of elongated streaks. Rotting of the seed may be seen just beneath the black spot on siliqua of yellow or brown sarson. Spots on mustard siliqua are brownish black with a distinct gray center. When older plants become infected, symptoms often occur on the older leaves, since they are closer to the soil and are more readily infected as a consequence of rain splash or wind-blown rain. Fruit-bearing branches and pods show dark or blackened spots that result in yield loss due to premature pod ripening and shedding of the seeds. The infection of *Alternaria* blight on leaves and silique reduces the photosynthetic area drastically. The phase of infection on silique adversely affects the normal seed development, weight, color, oil content, and the quality of seed. *Alternaria* blight–infected leaves of Indian mustard showed significant decrease in oil, triglyceride, 18:2, and 22:1 fatty acid content and also in the level of different lipid classes (phospholipids, glycolipids, and sterols) (Atwal et al. 2005). Two *Alternaria* toxins, namely, alternariol and alternariol monomethyl ether, were found in high concentration in the seeds infected with *Alternaria* species (Gwiazdowski and Wickiel 2009, Jajor et al. 2011). Several biochemical constituents are found to impart resistance to rapeseed–mustard against *A. brassicae*. Total sugars, reducing sugars, flavonol, and chlorophyll content were present in high amount in healthy leaves, while total phenol, *o*-dihydroxy phenol, carotenoids, and protein content rose with increase in infection of *A. brassicae* (Neeraj and Verma 2010, Mathpal et al. 2011, Gupta et al. 2012, Prakash et al. 2012). However, total sugar, total phenol, and *ortho*-dihydroxyphenol were higher in chlorotic areas than necrotic areas of infected leaves. Flavonol and chlorophyll content was observed lower in different infected parts of leaves than in healthy ones and was prominently lower in necrotic areas than chlorotic zones (Atwal et al. 2004).

Activities of some oxidative enzymes, namely, peroxidase (PO) and polyphenol oxidase, increased in *B. juncea* leaves after infection (Chawla et al. 2001). Total phenol content and specific activities of phenylalanine ammonia lyase (PAL) and tyrosine ammonia lyase (TAL) were higher in *Alternaria* blight–infected leaves and siliquae walls compared to healthy leaves, which suggests their possible involvement in plant protection against the disease (Gupta and Kaushik 2002). Transpiration in oilseed rape reduced after infection with *Alternaria* species (Baranowski et al. 2009).

FIGURE 5.1 Alternaria blight on leaves.

FIGURE 5.2 Alternaria blight on pods.

Geographical Distribution and Losses

Rapeseed–mustard crops are ravaged by *Alternaria* blight or black spot; the most common widespread and destructive disease is caused mostly by *A. brassicae* (Berk.) Sacc. infecting all aboveground parts of the plant, reported from all the continents of the world and considered an important constraint in husbandry of oilseed Brassicas. The disease has been reported from Argentina, Australia, Austria, Bangladesh, Brazil, Bhutan, Bulgaria, Canada, Chile, China, Cyprus,

Czechoslovakia, Denmark, Ethiopia, Finland, France, Germany, Ghana, Hong Kong, Hungary, India, Ireland, Italy, Jamaica, Japan, Kenya, Libya, Malawi, Malaya, Mauritius, Morocco, Mozambique, Myanmar, Nepal, the Netherlands, New Zealand, Nicaragua, Nigeria, Norway, Pakistan, Papua New Guinea, Philippines, Poland, Romania, Russia, Saudi Arabia, Scotland, Sierra Leone, Singapore, Spain, South Africa, Sri Lanka, Sudan, Sweden, Switzerland, Taiwan, Tanzania, Trinidad, Turkey, Uganda, the United Kingdom, the United States, Zambia, and Zimbabwe (Saharan et al. 2005). Pathogens of the disease, *A. brassicicola* and *A. raphani*, are also encountered but rarely. Though total destruction of the crop due to the disease is rare and usually yield losses at harvest are 5%–15%, they can reach up to 47% (Kolte et al. 1987) accompanied by reduction in seed quality, namely, seed size and viability. Severity of *Alternaria* blight on oilseed Brassicas differs among seasons and regions as also between individual crops within a region.

Pathogen

It is easy to recognize *Alternaria* sp. by the morphology of their large conidia. They are catenate, formed in chains or solitary, typically ovoid to obclavate, often beaked, pale brown to brown, multicelled, and muriform (Simmons 2007). Formation of chlamydospores is reported in *A. brassicae* and *A. raphani*, while microsclerotia are found to be produced by the former. Although the use of species-group designation does not resolve definitive species boundaries within *Alternaria*, advantages of its use are that it organizes at the subgeneric level the morphologically diverse assemblage of *Alternaria* species and permits the generalized discussion of morphologically similar species without becoming overrestricted due to nomenclatural uncertainty. Cultural, morphological, pathogenic, and molecular variation in isolates of *A. brassicae* has been indicated by several workers (Gupta et al. 2004a, Mehta et al. 2005, Patni et al. 2005a, Khan et al. 2007a,b, Singh et al. 2009, Goyal et al. 2011a, 2013a, Khulbe et al. 2011, Kumawat et al. 2011, Sharma et al. 2013a,b). Some extracellular enzymes, namely, cellulase and pectinases (polygalacturonase [PG] and pectin methyl esterase), are produced by *A. brassicae* under different cultural conditions (Atwal and Sangha 2004a). However, their exact role in pathogenesis is not known. *Alternaria longipes* and *Alternaria napiforme* are also reported on rapeseed–mustard from India (Kolte 1985). *A. brassicicola* produce some detoxifying enzymes, namely, brassinin hydrolases, which are dimeric protein of 120 kDa and catalyze the detoxification of brassinin, a phytoalexin produced in crucifers after fungal infection (Pedras et al. 2009a). The most selective phytotoxic metabolite, namely, brassicicolin A, and the major phytoalexin, namely, spirobrassinin, were produced by *A. brassicicola* in liquid cultures and in infected leaves of *B. juncea*, respectively (Pedras et al. 2009b).

Epidemiology and Disease Cycle

Efficient, economical, and environment-friendly control of *Alternaria* blight may be obtained through the knowledge of its timing of attack in relation to weather factors, which may enable prediction of its occurrence so as to allow growers to take timely action in an efficient manner for crop management. Weather is an exceptionally important factor in the severity of *Alternaria* blight of oilseed Brassicas. Preliminary work indicates effects of temperatures, relative humidity (RH), and sunshine hours on the occurrence of the blight on the oilseed Brassicas (Chattopadhyay 2008). These reports indicate relationships between different weather factors and *Alternaria* blight occurrence through empirical models. Severity of *Alternaria* blight on leaves and pods was higher in later sown crops (Chattopadhyay et al. 2005). Gupta et al. (2003) found positive correlation between sowing date of rapeseed–mustard crop and *Alternaria* blight disease severity. A delayed sowing results in coincidence of the vulnerable growth stage of plants with warm (maximum temperature: 18°C–26°C; minimum temperature: 8°C–12°C) and humid (mean RH > 70%) weather. Initiation of *Alternaria* blight disease on leaves of mustard occurred during 36–139 DAS, highest being at 45 and 75 DAS. Initiation of the disease on pod occurred between 67 and 142 DAS, highest being at 99 DAS. Severity of *Alternaria* blight disease on leaves was favored by a maximum temperature

of 18°C–27°C in the preceding week, minimum temperature of 8°C–12°C, mean temperature >10°C, >92% morning RH, >40% afternoon RH, and mean RH of >70%. Disease severity on pods was positively influenced by 20°C–30°C maximum temperature, >14°C mean temperature, >90% morning RH, >70% mean RH, >9 h sunshine, and >10 h of leaf wetness (Chattopadhyay et al. 2005). The regional and crop-specific models devised thereby could predict the crop age at first appearance of *Alternaria* blight on the leaves and pods, the peak blight severity on leaves and pods, and the crop age at peak blight severity on leaves and pods at least 1 week ahead of first appearance of the disease on the crop, thus allowing growers to take necessary action (Chattopadhyay et al. 2005). Sporulation of *A. brassicae* has been reported to be favored by darkness (Humpherson-Jones and Phelps 1989).

Survival of the pathogen on diseased seed or affected plant debris in tropical or subtropical India (Mehta et al. 2002) and in Nepal (Shrestha et al. 2003) has been ruled out, although under temperate conditions, the pathogen is known to survive noncrop season on seeds and infected crop debris (Humpherson-Jones and Maude 1982). *A. brassicae* was observed predominantly in the seed coat and rarely in embryos of *Brassica campestris* var. toria and *B. juncea* (Shrestha et al. 2000). In Indian subcontinent, oilseeds *Brassica* are sown from late August to November depending on the crop, prevailing temperature, and availability of soil moisture for seed germination. Crop harvest occurs from February to May. Off-season crops are grown in nontraditional areas from May to September and this, coupled with harboring of the fungal pathogen by vegetable *Brassica* crops and alternative hosts (*Anagallis arvensis*, *Convolvulus arvensis*), could be a reason for carryover of the *A. brassicae* from one crop season to another (Mehta et al. 2002). Thus, airborne spores of *A. brassicae* form the primary source of inoculum of this polycyclic disease (Kolte 1985). During the crop season, the pathogen(s) could have several cycles, whereby it is known as a polycyclic disease. Germ tube from germinated spores of *A. brassicae*, *A. brassicicola*, *A. raphani*, and *Alternaria alternata* generally penetrate the undamaged tissues of many brassicaceous hosts directly (Kolte 1985, Goyal et al. 2013a), although indirect penetration through stomata has been reported in *A. brassicae* (Kolte 1985, Goyal et al. 2013b). Black spot lesions develop within 48 h after inoculation. According to Tewari (1986), *A. brassicae* in rapeseed becomes subcuticular after direct penetration. This is followed by colonization of epidermal and the mesophyll cells. In the leaves of rapeseed, the pathogen heavily colonizes the necrotic center and is not present in the chlorotic area indicating that a diffusible metabolite may be directly or indirectly responsible for leaf chlorosis. The plasma membrane is the first target of the diffusible metabolites. Subsequently, the chloroplasts are directly or indirectly affected leading to leaf chlorosis. Recently, Goyal et al. (2013b) found that conidia of *A. brassicae* germinated on the upper epidermis of *B. juncea* leaf, by producing one or several germ tubes and penetrating the host directly without the formation of appressorium. The mycelia ramified, colonized mesophyll and palisade tissue caused necrosis of the cells by producing toxins or metabolites that resulted in the formation of necrotic spots and reduction in photosynthetic area of different plant parts. This infection also decreased the amount of all the cell constituents like lignin, lipids, suberin, and protein, except phenolic compound in all the tissues of *Alternaria*-infected *B. juncea* leaves as compared to healthy leaves.

Disease Management

Host Plant Resistance

Several sources of tolerance against *Alternaria* blight have been reported (AICRP-RM 1986–2014; Chattopadhyay and Séguin-Swartz 2005). A short stature *B. juncea* cv. Divya was found tolerant to *Alternaria* blight (Kolte et al. 2000). Among the different species of oilseed, *Brassica*, *B. juncea*, and *B. rapa* are more susceptible than *B. carinata* and *B. napus*. Lines found tolerant to the disease in *B. juncea* include PAB-9511, PAB-9534, JMM-915, EC-399296, EC-399301, EC-399299, EC-399313, PHR-1, PHR-2, Divya, PR-8988, PR-9024, and RN-490; those in *B. carinata* are HC-1, PBC-9221 (Kiran), NRCDR-515, and DLSC-1; the ones of *B. napus* happen to be PBN-9501,

PBN-9502, PBN-2001, and PBN-2002 (Kolte 1985, AICRP-RM 1986–2014, Patni et al. 2005b, Kolte et al. 2006, Kumar and Kolte 2006). Sources of resistance to *A. brassicae* have been spotted in wild Brassicas, namely, *Brassica alba* (Kolte 1985, Chattopadhyay and Séguin-Swartz 2005), *Camelina sativa*, *Capsella bursa-pastoris*, *Eruca sativa*, *Neslia paniculata* (Chattopadhyay and Séguin-Swartz 2005), *Brassica desnottesii*, *Coincya pseuderucastrum*, *Diplotaxis berthautii*, *Diplotaxis catholica*, *Diplotaxis cretacea*, *Diplotaxis erucoides*, and *Erucastrum gallicum* (Sharma et al. 2002). Three cultivars, namely, Yajima kabu, Saishin, and Shimofusa kabu of *B. rapa*, were identified as resistant against *A. brassicicola* on pods, and these are useful not only for breeding programs for *B. rapa* but also for *B. napus*, a derivative from *B. rapa* and *B. oleracea*, and has little genetic variation due to the limited size of the descendent population (Doullah et al. 2009). A breeding program using number and size of lesions to find the differential response of different genotypes against *Alternaria* blight has been proposed to genetically enhance the level of resistance by Yadav et al. (2008). Resistance to *Alternaria* blight of rapeseed–mustard is found to be associated with factors like phenolic compounds, namely, polyphenol oxidase, PO, catalase in leaves, higher sugars and N, lower in resistant species (Chattopadhyay 2008) or discouragement to conidial retention on plant surface like high deposits of epicuticular wax that forms a physical barrier as a hydrophobic coating to reduce the deposition of waterborne inoculum, and reduce rate of conidia germination and germ-tube formation. *B. napus* (Tower, HNS-3), *B. carinata* (HC-2), and *B. alba* have more wax on plant/leaf surface compared to *B. rapa* (BSH-1, YSPB-24) and *B. juncea* (RH-30) (Chattopadhyay 2008). Two phytoalexins, namely, camalexin and brassinin, and two isothiocyanates (ITCs), namely, allyl- and benzyl-ITCs, were reported to be have antifungal activity at different developmental stages of *Alternaria* blight pathogens, namely, *A. brassicae* and *A. brassicicola* of crucifers (Sellam et al. 2007). Wild crucifers are found to elicit phytoalexins on challenge inoculation (Conn et al. 1988). Activities of some compounds related to camalexin ($C_{11}H_8N_2S$) and 6-methoxycamalexin ($C_{12}H_{10}N_2SO$) are found to be most toxic to *A. brassicae*. Phytotoxin destruxin B elicits phytoalexin response in *B. alba*. Parada et al. (2007) reported that destruxin B is not a host-selective toxin and does not induce accessibility of host plants to *A. brassicae*. Resistance to *A. brassicae* is found to be layered and multicomponent with sensitivity to host-specific toxin destruxin B, quantitative and qualitative elicitation of phytoalexins, hypersensitive reaction, and Ca sequestration determining the fate of host–pathogen interaction (Chattopadhyay and Séguin-Swartz 2005). The resistant *Brassica* varieties also produce some metabolites, namely, sesquiterpenes, deoxyuvidin B, albrassitriol, isoalbrassitriol, and brassicadiol (Saharan et al. 2003).

Molecular Breeding

Since resistance to *Alternaria* blight is governed by additive or polygenes, breeding for resistance to these diseases could involve pyramiding of minor genes, introgression of genes from material found resistant, reciprocal recurrent selection or diallel selective mating (Krishnia et al. 2000), wide hybridization (*B. alba*), molecular breeding (viz., from *C. sativa* by somatic hybridization; transgenic expressing *Trichoderma harzianum* endochitinase gene: Mora and Earle 2001), pollen culture, and sensitivity test to destruxin B. While in studies on the mechanism of tolerance to *Alternaria* blight some have indicated the effect of additive genes or polygene or cluster gene (Krishnia et al. 2000) with resistance being controlled by nuclear genes of partial dominance, there has also been indication of components of resistance being significantly correlated to each other regarding slow blighting (Kumar and Kolte 2001), and dominance (h) having a predominant role in genetic control of time of appearance; additive × dominance predominant for other disease progression factors, namely, area under the disease progress curve (Lakshmi and Gulati 2002, Chattopadhyay and Séguin-Swartz 2005, Meena et al. 2011a). The chitinase enzyme when overexpressed degrades the cell wall of invading fungal pathogens and plays an important role in plant defense response. Indian mustard, which has been transformed with chitinase gene tagged with an overexpressing 35S cauliflower mosaic virus (CaMV) promoter, showed delay in the onset of disease as well as reduction in number and size of lesions (Mondal et al. 2003). Transgenics of Indian mustard with barley

antifungal genes class II chitinase and type I ribosome-inactivating protein, which coexpressed in plants, showed some resistance against *A. brassicae* infection through delayed onset of the disease and restricted number, size, and expansion of lesions as compared to wild plants (Chhikara et al. 2012). The pathogenesis-related (PR) proteins are toxic to invading fungal pathogens but are present in plant in trace amount. Thus, overexpression of these proteins may increase resistance to pathogenic fungi in several crops. Indian mustard plants transformed with class l basic glucanase gene showed restricted number, size, and spread of lesion caused by *A. brassicae*. This gene produces a PR protein glucanase that hydrolyzes a major cell-wall component, glucan, of pathogenic fungi and acts as a plant defense barrier (Mondal et al. 2007). Various technologies, namely, embryo rescue, somatic hybridization, somaclonal variations, genetic transformation, molecular markers, and signal transduction, have been used for incorporation of resistance against this pathogen in oilseed Brassicas by Aneja and Agnihotri (2013).

Rapid advances in techniques of tissue culture, protoplast fusion, embryo rescue, and genetic engineering make transfer of disease resistance traits across wide crossability barriers possible. A cDNA encoding hevein (chitin-binding lectin from *Hevea brasiliensis*) was transferred into *B. juncea* cv. RLM-198. Southern analysis of the putative transgenics showed integration of the transgene. Northern and Western analyses proved that the integrated transgene is expressed in the transgenics. In whole plant bioassay under glasshouse conditions, transgenics were found to possess parameters that are associated with resistance such as longer incubation and latent period, smaller necrotic lesion size, lower disease intensity, and delayed senescence (Kanrar et al. 2002). Insight has been gained into genes being expressed during *Alternaria* infection of *Brassica* (Cramer and Lawrence 2004). The authors used suppression subtractive hybridization between RNA isolated from the spores of *A. brassicicola* incubated in water and on the leaf surface of an ecotype of *Arabidiopsis thaliana* followed by cloning and sequencing of cDNA clones that were differentially expressed. One gene (P3F2), only expressed during infection, was identified, although its function remains to be determined. A similar approach with other pathogens could lead to advances in the understanding of pathogenicity.

Induced Host Resistance

Systemic acquired resistance (SAR) is induced by inoculation with avirulent race of *A. brassicae* (Vishwanath et al. 1999, Vishwanath and Vineeta 2007). Pretreatment of *Brassica* plants with beta-aminobutyric acid (BABA) induced resistance in plants and is thought to be mediated through an enhanced expression of PR protein genes, independent of salicylic acid (SA) and jasmonic acid accumulation (Kamble and Bhargava 2007). Some elicitors like benzothiadiazole (BTH) alone and in combination with SA may play a significant role in eliciting the defense-related enzymes, namely, PO, PAL, and superoxide dismutase (SOD) and phenolics, which may help in the reduction of disease severity by empowering the plant to restrict the invasion of *A. brassicae* in *B. juncea* (Sharma et al. 2008a, Sharma and Sohal 2010). Spray of SA on leaves increased the total sugar content but decreased the starch content in the leaves, which was linked with induction of disease resistance by maintaining a healthy flora of saprophytic microbes that are active against pathogens (Atwal and Sangha 2004b).

Mutation breeding may be one of the feasible techniques for breeding pathogen-resistant cultivars in the absence of a useful donor for resistance to the pathogen in the available germplasm of crops. Some *A. brassicicola*–resistant *B. napus* plants were regenerated from selected and unselected calli after mutation with gamma rays (physical) and ethyl methanesulfonate (chemical) mutagens (Sharma et al. 2012a).

Cultural Control

Hot-water treatment of seeds reduced the growth of *Alternaria* (Humpherson-Jones and Maude 1982). However, spores of these fungi can survive on the leaf tissue for 8–12 weeks and that on stem tissue till 23 weeks. Hence, fields that are replanted soon after harvest often coincided with

a large amount of inoculum, which is likely to affect the crop's emergence and early growth stages (Humpherson-Jones 1992). Thus, rotation with noncruciferous crops and eradication of cruciferous weed hosts can help control these pathogens while fungicide spray in fields needs to be done at the same time. Early sowing (Meena et al. 2002) of well-stored clean certified seeds after deep ploughing at 45 cm row spacing (Kumar and Kumar 2006), clean cultivation, timely weeding and maintenance of optimum plant population, avoidance of irrigation at flowering, and pod formation stages may help manage the disease. Sowing of seeds should not be done by broadcasting method, because it increases disease severity, which could be decreased on leaves and pods by applying 40 kg K/ha along with the recommended dose of nitrogen (N) or sulfur (40 kg/ha) or along with recommended NPK (Kumar and Kumar 2006). However, a higher dose of N makes the crop susceptible to disease. Soil application of K as basal has been found to check *Alternaria* blight disease in mustard (Khatun et al. 2011). It has been reported that sulfur, zinc, and boron decrease the development of *Alternaria* blight and increase seed yield of mustard crop (Khatun et al. 2010). Application of some micronutrient, namely, B at 1 g/L, Mo at 1 g/L, S at 2 g/L, and Zn at 2 g/L, in various combinations reduced *Alternaria* blight disease and increased yield of rapeseed–mustard (Mondal 2008). Inorganic fertilizers, namely, phosphorus (P) and potassium (K), also decrease the disease, while N increases it (Singh 2004a). These are significant at a time when growers report increasing prevalence of *Alternaria* blight disease and decline in its control.

Biological Control

The GR isolate of *Trichoderma viride* was at par with mancozeb in checking blight severity on mustard leaves and pods (Meena et al. 2004). Conidial suspension of *T. viride* was more effective in comparison to culture filtrate in reduction of disease intensity on leaves and pods (Reshu and Khan 2012). *Bacillus subtilis* strain UK-9 isolated from reclaimed soil caused morphological alternations in vegetative cells and spores by disruption, lysis of cell wall of the pathogen, which resulted in reduction in disease severity, and spore germination on leaves (Sharma and Sharma 2008). Seed treatment with bioagents resulted in increase in lipid (phospholipids, glycolipids, and sterol) and protein content in seeds from treated plants. However, seed treatment and foliar spray with bioagents on leaves of Indian mustard enhanced the content of dry matter, total phenol, *ortho*-dihydroxyphenols, starch, total soluble sugars, reducing sugars, total lipids, and different membrane lipids in the leaves but the total protein content decreased after treatment with biocontrol agents at 30 and 60 DAS, which could be associated with defense mechanisms and enhanced growth of the plants (Sharma et al. 2010a,b).

Effect of Plant Extracts

Extracts of several plants have been evaluated against *A. brassicae* (Patni and Kolte 2006, Bhatiya and Awasthi 2007, Meena and Sharma 2012a, Sasode et al. 2012). The level of efficacy of *Azadirachta indica* extract increases as the number of sprays increases (Mohiddin et al. 2008). Spray of garlic bulb and neem leaf extract at flowering stage suppressed disease incidence (DI) and increased yield of mustard crop (Ferdous et al. 2002). Application of 1% (w/v) aqueous bulb extract of *Allium sativum* at 45 and 75 DAS in checking the disease severity on leaves and pods was at par ($P < 0.05$) with mancozeb as also in highest seed yield (Meena et al. 2004, 2008, 2011b, 2013, Yadav 2009). Two foliar sprays of *Eucalyptus globosus* at 2% (w/v) at 75 and 90 DAS could be done for eco-friendly management of black spot disease of rapeseed–mustard (Chandra et al. 2009). Foliar spray of extract of *Calotropis procera* leaves, *A. indica* kernel, and *A. sativum* bulbs may induce resistance against *A. brassicae* by increasing soluble phenol, sugar content, and soluble proteins, namely, PO, polyphenol oxidase, and PAL content in mustard leaves (Surendra et al. 2012). Among several essential oils evaluated, that of *Mentha piperita* provided complete inhibition of fungal growth at 2000 µg/mL, followed by oil of *Cyperus scariosus* (Dhaliwal et al. 2003).

Chemical Control

Spray with iprodione (Rovral) was effective in checking silique infection due to *A. brassicae*. Both reduction in disease and increase in seed yield and test weight were observed by the application of iprodione (Chattopadhyay and Bhunia 2003, Alam et al. 2010), and its residues in the edible parts of plants were lower than the maximum residue level that indicated the safety of this fungicide at the recommended rate (Mukherjee et al. 2003). Higher number (3–4) of iprodione sprays exerted significant reducing effect on the number of spots per siliquae (Hossain and Rahman 2006). Nowadays, there is a need to adopt new molecules of fungicides for the control of such pathogen keeping in mind their fungicidal resistance. Mycelial growth, conidial germination, and germ-tube elongation revealed the existence of *A. brassicicola* isolates highly resistant (EC_{50} > 100 mg/L) to both dicarboximides (e.g., iprodione and procymidone) and phenylpyrroles (e.g., fludioxonil) (Vasilescu et al. 2004). Application of fungicides on seeds reduced the content of two *Alternaria* toxins, namely, alternariol and alternariol methyl ester (Gwiazdowski and Wickiel 2011). Benlate (Anwar and Khan 2001), Contaf (Singh and Maheshwari 2003), and mancozeb (Meena et al. 2004) were effective in reducing disease severity on leaves and increasing seed yield in mustard. Two consecutive foliar sprays of mancozeb 75 WP (0.2%) followed by one spray of metalaxyl + mancozeb (ridomil MZ 72: 0.25%) resulted in high-seed yield and 1000-seed weight (Singh and Singh 2006). The highest net profit as well as the highest cost–benefit has been obtained with carbendazim/zineb (1:3.2) combination followed by carbendazim/captan (1:1.3) combination (Khan et al. 2007c). Seed treatment with carbendazim and foliar spray of metalaxyl + mancozeb (ridomil MZ 72 WP) was found most effective in reducing disease severity and in increasing seed yield (Prasad et al. 2009b). Exposure of *Alternaria* blight–affected leaves to high concentration (214.5 μg/m^3) of SO_2 resulted in suppression of the disease (Khan and Khan 2010).

WHITE RUST

The most widely recognized fungal species, *Albugo candida* (Pers.) Roussel, had been thought to be the exclusive white rust pathogen of the Brassicaceae, infecting as many as 63 genera and 241 plant species (Choi et al. 2009). According to the USDA-ARS Systematic Botany and Mycology Laboratory, *A. candida* was recorded on more than 300 hosts (Farr et al. 2004). Only recently was it realized that a high degree of genetic diversity is present within *Albugo* on Brassicaceae (Voglmayr and Riethmüller 2006, Choi et al. 2008) and that several of the observed lineages might constitute distinct species (Choi et al. 2011). The host specificity of *A. candida* has been recorded from Australia (Kaur et al. 2008a), Canada, Germany (Kolte 1985), India (Saharan et al. 2005), Japan, Romania, and the United States (Kolte 1985). Following the recent lectotypification of *A. candida*, the taxonomic status of which had previously been unclear (Choi et al. 2007), two specialized *Albugo* species parasitic to Brassicaceae have been described within *Albugo* (Choi et al. 2007, 2008). It was also demonstrated that *A. candida* has a broad host range extending over more than a dozen genera of the Brassicaceae and into the Cleomaceae, as the type of *Albugo chardonii* W. Weston (Kolte 1985) was found to be nested within *A. candida* (Choi et al. 2007). Capers (*Capparis spinosa*) are affected by white blister rust attributed to *Albugo capparidis* or, applying a broad species concept, to *A. candida* (Choi et al. 2009).

Symptoms

Disease appearing on leaves is characterized by the appearance of white or creamy yellow–raised pustules up to 2 mm in diameter, which later coalesce to form patches. The pustules are found scattered on the lower surface of the leaves. The part of upper surface corresponding to the lower surface is tan yellow, which enable recognition of the affected leaves. After the complete development of the pustule (Figure 5.3), it ruptures and releases a chalky dust of spores (sporangia). With aging of white rust pustules, affected leaves become senescent when necrosis around or in the pustule can

FIGURE 5.3 White rust on leaves.

FIGURE 5.4 Staghead (hypertrophied inflorescence) caused by *Albugo*.

be seen. Such rust pustules are also observed on the surface of well-developed siliquae. These are noted in local infection. Unlike other crucifers (Mundkur 1959), thickening or hypertrophy of the affected leaves is usually not seen in rapeseed–mustard. However, in systemic infection or infection through stem or flower, hypertrophy and hyperplasia are observed, which result in the formation of stagheads (Figure 5.4). Affected flowers become malformed, petals become green like sepals,

and stamens may be transformed to leaf-like club-shaped sterile or carpelloid structures, which are found to persist on the flower rather than falling early as in normal plants. Ovules and pollen grains are usually atrophied leading to complete sterility. Association of symptoms of downy mildew with that of white rust is frequented. Whole plant infection at very early plant growth stage due to systemic infection is stunted, thickened with no branching, and beared white rust pustules on surface. Thickening of stem may be due to the modification of cortex into large thin-walled cells with fewer intercellular spaces. In floral parts also, there is an increase in size and number of cells of parenchymatous tissue with few intercellular spaces, lesser differentiation of tissue and organs, and increased accumulation of nutrients. Multiplication and spore production by the pathogen result in consumption of the accumulated nutrients leading to collapse and death of cells, drying of affected plants (Kolte 1985).

Geographical Distribution and Losses

White rust caused by *A. candida* (Pers. ex. Fr.) Kuntz. can result in yield loss up to 47% on oilseeds *Brassica* (Kolte 1985) and 89.9% in *B. juncea* (Varshney et al. 2004) with each percent of disease severity and staghead formation causing reduction in seed yield of about 82 and 22 kg/ha, respectively (Meena et al. 2002). The highest avoidable yield loss up to 28.2% with highest disease intensity, namely, 70.8%, has been reported in late sown *B. juncea* var. Varuna (Singh and Bhajan 2005). The disease has been reported from Australia, Austria, Brazil, Canada, China, Egypt, Fiji, Finland, Germany, India, Iran, Japan, Korea, the Netherlands, New Zealand, Pakistan, Palestine, Poland, Portugal, Romania, Saudi Arabia, Sweden, Spain, Turkey, Venezuela, the United Kingdom, and the United States (Meena et al. 2014).

Pathogen

The obligate parasite *A. candida* or *Cystopus candidus* causes white rust of oilseeds *Brassica* and several other crucifers (Choi et al. 2009). Mycelium is aseptate, intercellular with nuclei-free globular haustoria (Coffey 1975). Masses of mycelia beneath host epidermis form a palisade of cylindrical-shaped sporangiophores, which are thick walled at base and free laterally. The sporangiophores give rise to chains of spherical hyaline, smooth, and 12–18 μ diameter sporangia in a basipetal succession, which germinate to give rise to concave biflagellate zoospores or at times to germ tubes (Walker 1957). Temperature most favorable for the germination of sporangia is 10°C (Khunti et al. 2004). Once zoospores are released from the sporangium, they exhibit chemotactic, electrotactic, and autotactic or autoaggregation responses to target new hosts for infection (Walker and West 2007). Thereafter, zoospores come to rest, retract their flagella, and encyst and germinate by the formation of a germ tube. If germination occurs on a susceptible host, then the germ tube penetrates through stomata to form an intercellular mycelium (Walker 1957). Oogonia and antheridia are formed from the mycelium in intercellular spaces, particularly in systemically affected plants (Webster 1980). Oogonia are globose, terminal or intercalary, each containing up to 100 nuclei; its contents defined into a peripheral zone of periplasm and a single central oosphere. Antheridia are clavate, each with 6–12 nuclei on the sides of an oogonium (Heald 1926, Walker 1957). The heterothallic fungus produces restings spores or oospores, which are highly differentiated with five-layered cell wall and at maturity are tuberculate, 40–55 μ in diameter. Germination of oospores has been described by de Bary (1887) and Vanterpool (1959). Germination by sessile vesicles was the most common. Treatment of oospores with 200 ppm $KMnO_4$ for 10 min induced increased germination (Verma and Bhowmik 1988). Oospores do not appear to require any dormancy period. Meena and Sharma (2012b) used animal gut enzymes (1% β-glucuronidase and arylsulfatase, Sigma make) for the germination of the oospores from hypertrophied plant tissue. Physiological specialization of the pathogen has also been reported. Zoospores produced from germinating oospores constitute the primary source of inoculum for the infection of rapeseed–mustard (Verma and Petrie 1980),

particularly when mixed with seeds. There appears to be no dormancy in the case of oospores, as they have been found to germinate just 2 weeks after their collection from affected tissues. The most likely primary infection site is the emerging cotyledon. The production of large masses of zoosporangia on cotyledons seems to require establishment of a large mycelial base inside the host tissue, and in the *Albugo–Brassica* system, such a base apparently develops with a minimum disturbance of the host's synthetic abilities (Harding et al. 1968). The sporangia become visible after the epidermis is ruptured, as a white powdery mass, which can readily be dispersed by wind to cause secondary infection. If sporangia alight on a suitable host leaf or stem surface, they are capable of germinating within a few hours in films of water to form biflagellate zoospores, about eight per sporangium. After swimming for a time, a zoospore encysts and then forms a germ tube, which penetrates the host epidermis. A further crop of sporangia may be formed within 10 days. The establishment and maintenance of a compatible relationship between *A. candida* and its hosts seem to hinge on the successful formation of the first haustorium. In the susceptible host, such as *B. juncea*, the first haustorium forms within 16–18 h after inoculation. Haustoria are small and capitate with spherical heads averaging 4 μ in diameter and are connected to hyphae by slender stalks about 2 μ in length. A haustorium usually originates near the tip of a young hypha, which then continues its growth leaving the haustorium as a side branch. After the formation of the first haustorium in the susceptible host–parasite combination, hyphal growth rate increases rapidly. An encapsulation similar to that observed in *Raphanus sativus* is seen only infrequently around haustoria in a virulent *Albugo*-susceptible *B. juncea* system. In a susceptible host, the hyphae appear to grow around palisade mesophyll cells as a downward spiral, penetrating individual cells with a variable number of haustoria. Verma et al. (1975) found the presence of as many as 14 haustoria in a single cell in "green island" tissue of artificially infected *B. juncea* cotyledons. In the susceptible host, most of the intercellular spaces appear to get occupied by mycelium within 3 days after inoculation. Some biochemical compounds, namely, total phenols, total sugars, and reducing and nonreducing sugars, are generally negatively correlated with DI, but with low disease levels, these are not always consisent (Singh 2005, Mishra et al. 2009). Phenol content reduced more in infected inflorescences than leaves and cotyledons, while free amino acids increased more in infected leaves than inflorescences and cotyledons. But total mineral contents did not show significant variation in both infected and healthy plant parts. However, total chlorophyll content decreased in cotyledons and leaves and increased in the malformed inflorescences (Singh 2005). Studies on quantitative changes in amino acid content of white rust–induced hypertrophies of the mustard plant indicated possible breakdown of protein due to pathogen to release tryptophane and subsequently increasing IAA content of such tissues. However, decrease in IAA, free proline, total proteins, and phenolic compounds in the infected host tissue are also reported. One protein, peptidyl-prolyl *cis-trans* isomerase (PPIase) isoform CYP20-3, was detected only in the susceptible variety and increased in abundance in response to the pathogen. PPIases play an important role in pathogenesis by suppressing the host cell's immune response (Kaur et al. 2011a). Cellulase, endo-polymethyl galacturonase (PMG), and endo-PGs were produced in *B. juncea* leaves infected with *A. candida*. Swelling and disruption of subcellular particles rich in lysomal acid hydrolases were produced by acid phosphatase activity centered primarily in the infected tissues of *B. juncea*. Acid phosphatase activity in antheridia, oogonia, and oospores of *A. candida* indicates that this enzyme plays a role in the synthesis of fungal organs. Increase in PO (Jain et al. 2009), invertase, alpha-amylase, IAA oxidase (Singh et al. 2011a), PAL, TAL, and lipoxygenase (LOX) (Jain et al. 2002) enzyme activity has been reported in infected leaves. However, nitrate reductase enzyme activity increased in infected cotyledons, leaves, and inflorescences (Singh 2005). Erucic acid content is positively correlated with disease infection, which indicates that low-erucic lines are resistant, medium-erucic lines are tolerant, and high-erucic lines are highly susceptible to the disease (Malik et al. 2004). Beta-1,3-glucanase was found to be induced more in the resistant cultivars of *B. juncea* after inoculation with *A. candida* (Kapoor et al. 2003). Infection of the resistant lines with *A. candida* showed significant increase in the glucosinolates as compared to susceptible lines (Pruthi et al. 2001). Much work remains to be done on mutual interaction of *Albugo* and associated

microorganisms. Some of the *Alternaria* species are known to produce toxins and these could conceivably have an adverse effect on the survival of *A. candida*. Pathogenic and genetic diversity among different *A. candida* isolates collected from *B. juncea*, *B. rapa*, *B. oleracea*, *B. tournefortii*, *Raphanus raphanistrum*, *R. sativa*, *E. vesicaria* subsp. *sativa*, *C. bursa-pastoris*, and *Sisymbrium irio*, from different locations in western Australia, has been reported (Kaur et al. 2011b).

Under Indian conditions, the *A. candida* isolate obtained from *B. rapa* was distinct in pathogenicity from the one obtained from *B. juncea* (Kolte et al. 1991). Verma et al. (1999) identified two new races of *A. candida* in India, namely, race 12 from *B. juncea* and race 13 from *B. rapa* var. *toria*, using 14 crucifer host differentials. Twenty distinct pathotypes of *A. candida*, 17 from *B. juncea* (AC 18–AC 34), 2 from *B. rapa* var. *brown sarson* (AC 35–AC 36), and one from *B. nigra* (AC 37), have been identified (Jat 1999). Four distinct and new pathotypes of *A. candida*, namely, AC I4 from RL 1359, AC 15 and AC 16 from Kranti, and AC 17 from RH 30 cultivars of *B. juncea*, have been identified on the basis of their differential interactions on 11 host differentials by Gupta and Saharan (2002). Out of these, pathotypes like AC 23, AC 24, and AC 17 infect only one, two, and three host differentials, respectively, indicating a limited virulence potential, while pathotypes of wider virulence, namely, AC 29, AC 27, AC 30, AC 18, and AC 21 infected 21, 18, 16, 12, and 10 host differentials, respectively (Jat 1999, Gupta and Saharan 2002).

Epidemiology and Disease Cycle

Berkenkamp (1980) reported an increase in DI due to soil application of trifluralin herbicide. The optimum temperature for disease development ranged 12°C–18°C. Only 3 h of wetness was required for disease development at 12°C–22°C. First appearance of white rust disease (*A. candida*) on leaves and pods (staghead formation) of mustard occurred 36–131 DAS, highest being at 50 and 70 DAS and 60 and 123 DAS, respectively. Severity of white rust disease on leaves was favored by >40% afternoon RH, >97% morning RH, and 16°C–24°C maximum daily temperature. Staghead formation was significantly and positively influenced by 20°C–29°C maximum daily temperature, further aided by >12°C minimum daily temperature, and >97% morning RH. It was possible to predict the highest severity of white rust disease of the crop season in the initial weeks after sowing with the models developed by stepwise regression (Chattopadhyay et al. 2011). By using hourly weather data, a simple weather-based forewarning model to evaluate DI has been developed (Kumar and Chakravarty 2008). Oospores formed in infected plants overwintered in plant debris and soil, which function as the source of primary inoculum of the pathogen (Butler and Jones 1961, Verma et al. 1975). Oospores have also been observed in naturally infected senesced leaves of *B. juncea* and *B. rapa* var. toria. Oospores can remain viable for over 20 years under dry storage conditions (Verma and Petrie 1975). Possibility of survival and spread of the pathogen by means of oospores, sporangia, and mycelia carried externally on seeds have been reported (Petrie 1975, Meena et al. 2014). The oospores germinate by releasing zoospores, which infect lower leaves during crop season. Secondary infection of the pathogen occurs through sporangia produced on leaves.

Disease Management

Host Plant Resistance

Though a few sources of host resistance ([*B. juncea*: PWR-9541, JMMWR 941-1-2, PAB-9534, PAB-9511, PHR-1, PHR-2, EC-129126, EC-399299, EC-399301, EC-399300, EC-399296, BIO YSR] [*B. rapa*: PT-303, Tobin] [*B. carinata*: HC-1, 2, 3, 4, 5, NRCDR-515, PBC-9921, BC-2, DLSC-1] [*B. napus*: TOWER, EC-33897, EC-339000, DGS 1, GS-7055, HNS-4, GSL-441, PBN-2001, PBN-2002] [*E. sativa*: RTM-1471] [*B. alba*: Exotic-1, Exotic-2]) have been identified (Kolte 1985, AICRP-RM 1986–2014, Chattopadhyay and Séguin-Swartz 2005, Kumar and Kalha 2005), their success is limited to a few pockets keeping in view the volatile race pattern of the pathogen. Three cultivars of Indian mustard PBR 181, EC-399301, and EC-399299 and two cultivars of *B. campestris*

were found resistant against white rust (Yadav and Sharma 2004). Meena et al. (2005) also found 38 genotypes free from white rust infection, out of 90 genotypes of 5 oileferous Brassicas, that is, *B. juncea*, *B. napus*, *B. carinata*, *B. campestris,* and *E. sativa* in India. In the Terai region of Uttar Pradesh (India), 5 genotypes, namely, WRR-98-01, NDRS-2004, NDRS-2013, NDRS-2005, and NDRS-2007, were found disease free, and 18 genotypes, namely, RC-781, RIK-75-5, RIK-78-4, CSR-721, PI-43, YRT-3, NDRE-7, NNRE-10, NDYR-29 × NDRE-04, NDRE-190 × NDRE-4, CSCN-5, CSCN-3, CSCN-10, CSCN-12, NSRS-2006, NDRS-2009, NDRS-12, and NDRS-2014, were found resistant (Sinah and Mall 2007). Yellow-seeded mustard (*B. juncea*) variety *T4*, YRT-3184 and rapeseed (*B. rapa* var. yellow sarson), type 6 (Chattopadhyay and Séguin-Swartz 2005) have been reported to be resistant to white rust infection. Three Australian genotypes JM 018, JM 06021, and JM 06026; JR 049 and JN 033; and some Chinese genotypes, namely, RK 2, Ringot, RH 13, Amora III, Quianxianjiecai, Yilihuang, Hatianyoucai, Jinshahuang, Manushuang, *B. juncea* 1, 2, and 3, were resistant to white rust (Singh et al. 2010). Some lines of *B. juncea* var. Cutlass, namely, RESJ-1052, RESJ-1004, RESJ-1005, RESJ-1033, and RESJ-1051, have been found to be resistant to all the Indian isolates as well as 2 V (Canadian isolate). These resistant sources with combined resistance to different white rust isolates could be putative donors for further oilseed *Brassica* crop improvement programs (Awasthi et al. 2012).

Transfer of white rust resistance in rapeseed–mustard from *B. carinata* to *B. juncea* could be partially successful by growing disease-free plants under high disease pressure followed by their repeated backcrossing with *B. juncea* cultivar (Singh et al. 1988). Resistance to white rust in rapeseed–mustard is dominant, governed by one or two genes with either dominant-recessive epistasis or complete dominance at both gene pair but either gene when dominant is epistatic to the other. These genes could be located on the same locus or different loci (Kumar et al. 2002). Resistance to the disease at true leaf infection and susceptibility at the cotyledonary leaf stage of the same genotype EC-399301 of *B. juncea* appears to be governed by two independent genes. Hence, screening for white rust resistance at the cotyledonary leaf stage needs to be carefully considered (Mishra et al. 2009). Interspecific crosses between *B. juncea* and *B. napus* suggested that resistance in WW-1507 and ISN-114 to *A. candida* was controlled by a single dominant gene (Jat 1999). In their study of three interspecific crosses between *B. juncea* and *B. napus*, Subudhi and Raut (1994) revealed digenic control with epistatic interaction for white rust resistance trait and a close association of parental species and different grades of leaf waxiness. Sachan et al. (1995), in their study using diallel crosses between two white rust–resistant Canadian *B. juncea* cvs. Domo and Cutlass and two susceptible *B. juncea* Indian cvs. Kranti and Varuna, reported that F1 hybrids, except susceptible × susceptible, were resistant; segregation pattern for resistance in F2 and test crosses was under the control of a single dominant gene in Domo and Cutlass, and that a recessive gene for susceptibility was present in Kranti and Varuna. Sridhar and Raut (1998) reported a monogenic inheritance showing complete dominance in four crosses and lack of dominance in seven crosses attempted between *B. juncea* and resistance sources derived from different species. According to Jat (1999), the resistance was dominant in all the crosses except susceptible × susceptible, where it was recessive. Under controlled conditions, inoculation with three different races of *A. candida* on F2 population of crosses from resistant × resistant revealed that the resistant genes may be located on the same locus or on different loci. Partial resistance in *B. napus* to *A. candida* was controlled by a single recessive gene designated as *wpr* with a variable expression (Bansal et al. 2005). White rust resistance in *B. juncea* (Somers et al. 2002, Manjunath et al. 2007, Singh et al. 2012, Yadava et al. 2012) and avirulence in race AC 2 of *A. candida* to *B. rapa* cv. Torch (Adhikari et al. 2003) is governed by single dominant gene. The resistance of *B. napus* var. *Regent* is conditioned by independent dominant genes at three loci designated as AC 7-1, AC 7-2, and AC 7-3. Two loci also controlled resistance in *B. napus* to *A. candida* race AC 2 collected from *B. juncea*. The Chinese *B. napus* accession 2282-9, susceptible to AC 7, has one locus controlling resistance to an isolate of *A. candida* collected from *B. carinata*. These studies indicated that only one allele for resistance was sufficient to condition an incompatible reaction in this pathosystem. In addition, a

single locus controlling resistance to AC 2 in *B. napus* and *B. rapa* was mapped using restriction fragment length polymorphism (RFLP) marker (Chattopadhyay 2008). A dominant allele at a single locus or two tightly linked loci were reported to confer resistance to both races AC 2 and AC 7 of *A. candida* (Kole et al. 2002). According to Borhan et al. (2008), a dominant white rust–resistant gene, *WRR* 4, encodes a toll-interleukin receptor–nucleotide-binding site–leucine-rich repeat protein that confers broad-spectrum resistance in *A. thaliana* to four races (AC 2, AC 4, AC 7, and AC 9) of *A. candida*. Four Chinese (CBJ-001, CBJ-002, CBJ-003, and CBJ-004) and two Australian (JR049 and JM 06011) genotypes were consistently highly resistant to an *A. candida* pathotype prevailing in Australia throughout the different plant growth stages (Li et al. 2007a, 2008a, 2009a).

Molecular Breeding

White rust susceptible cultivars of *B. juncea* and *B. napus* transformed with WRR4 gene from *A. thaliana* showed resistance to the corresponding *A. candida* races for each host species, which indicates that this gene could be a novel source of white rust resistance in oilseed Brassicas (Borhan et al. 2010). However, there is a need to guard against the danger of breakdown of resistance due to mixed infection with *Hyaloperonospora parasitica* (Singh et al. 2002a,b). Resistant genes have been mapped and identified on the chromosomes of *B. juncea*, namely, *ACr* (Cheung et al. 1998), *AC*-21 (Prabhu et al. 1998), *AC*-2 (Varshney et al. 2004), *ACB*1-*A*4.1, and *ACB*1-*a*5.1 (Massand et al. 2010); *B. rapa*, namely, *ACA*1 (Kole et al. 1996); *B. napus*, namely, *ACA*1 (Ferreira et al. 1994) and *AC* 2*V1* (Somers et al. 2002); and *A. thaliana*, namely, *RAC*-1, *RAC*-2, *RAC*-3, and *RAC*-4 (Borhan et al. 2001, 2008), effective against one or more than one race of *A. candida*. A single gene (*Acr*) responsible for conferring resistance to *A. candida* was mapped on a densely populated *B. juncea*. Two closely linked RFLP markers identified (X42 and X83) were 2.3 and 4 cM from the *Acr* locus, respectively (Cheung et al. 1998). Kole et al. (2002) have worked out the linkage mapping of genes controlling resistance to white rust in *B. napus.* A tightly linked marker for white rust resistance was developed using amplified FLP (AFLP) in conjunction with bulk segregant analysis (Varshney et al. 2004). A polymerase chain reaction (PCR)-based cleaved amplified polymorphic sequence (CAPS) marker for closely linked random amplified polymorphic DNA (RAPD) marker $OPB06_{1000}$ was developed. Data obtained on 94 recombinant inbred lines revealed that the CAPS marker for $OPBO6_{1000}$ and AFLP marker E-AAC/M-CAA_{350} flank the *Ac2*(t) gene at 3.8 and 6.7 cM, respectively. Validation of the CAPS marker in two different F_2 populations of crosses Varuna × BEC-144, and Varuna × BEC-286 established its utility in marker-assisted selection for white rust resistance. The use of both flanking markers in marker-assisted selection was estimated to only allow 25% misclassification, thus providing greater selection efficiency than traditional approaches (Varshney et al. 2004).

Induced Host Resistance

Tirmali and Kolte (2011, 2012) found nonconventional chemicals (plant defense activators) effective in reducing disease index on leaves and staghead incidence. A plant defense activator, BTH, was found effective in protection from staghead development against the challenge inoculation with *A. candida* (Kaur and Kolte 2001, Kumar 2009).

Cultural Control

Crop rotation with nonhost crops helps in managing this pathogen. Roguing and burning of disease-affected plants, particularly stagheads, help in minimizing inoculum buildup in soil. Overirrigation of crop should be avoided that helps in reducing DI. Clean, healthy, and certified seed should be used to avoid seed-borne white rust disease. Soil application of K as basal at 40 kg/ha resulted in significantly ($P < 0.05$) lesser white rust on leaves and number of stagheads than control. Early sowing of seeds may help decrease DI and staghead formation and increase seed yield (Yadav et al. 2002, Meena et al. 2004, Thapak and Dantre 2004, Biswas et al. 2007). Suitable date of sowing based on location and other epidemiological considerations needs to be decided to enable the escape of the disease in different locations.

Biological Control and Effect of Plant Extracts

In recent years, an increasing consciousness about environmental pollution due to pesticides and development of fungicide-resistant strains in plant pathogens have challenged plant pathologists to search for eco-friendly tools in disease management. Aqueous bulb extract of *A. sativum* 1% (w/v), an isolate of *T. viride*, as seed treatment and in combination as respective foliar sprays was statistically at par with that of mancozeb, combination of metalaxyl 35 ES 6 mL/kg seed treatment + 0.2 g/L spray of combination of metalaxyl + mancozeb in checking the rust severity on leaves and number of stagheads per plant (Meena et al. 2003). Inhibition of oospore development in *A. candida* by a natural bioagent *Psuedomonas syringe* under field condition has been reported (Tewari et al. 2000). Spray with the extract of *Eucalyptus* spp. (Kumar 2009) leaves can effectively manage the disease.

Chemical Control

Metalaxyl (Khunti et al. 2001, Biswas et al. 2007), metalaxyl + mancozeb (ridomil MZ) (Pandya et al. 2000, Godika et al. 2001, Yadav 2003), aluminium tris (Girish et al. 2007), and combination of metalaxyl 35 ES 6 mL/kg seed treatment + 0.2 g/L spray of combination of metalaxyl + mancozeb at 50 and 65 DAS (Kolte 1985, Meena et al. 2003) are reported to be able to manage the disease.

DOWNY MILDEW

Symptoms

Symptoms of the disease appear on all aboveground parts but usually on leaves and inflorescence. Usually a few days after sowing, small angular translucent light green lesions first appear on cotyledonary or the first true leaves during seedling stage and at times could be even restricted to these leaves with subsequently emerging ones not showing any symptom. Such lesions later enlarge and develop into grayish white, irregular necrotic patches on the leaves bearing downy growth of the pathogen (conidia and conidiophores) on its undersurface. In severe attack, the affected leaves dry up and shrivel. The extent to which the necrosis occurs depends upon the type of crop species. Leaf symptoms (Figure 5.5) at the seedling stage, as mentioned earlier, are more conspicuous on

FIGURE 5.5 Downy mildew affected leaves at seedling stage.

B. juncea compared to *B. rapa*. Very late in the season, downy growth may be seen on siliquae as well (Figure 5.6). Thickening of the peduncle/inflorescence (Figure 5.7) due to the disease suggest hypertrophy of affected cells, pith of the stem being more affected than the cortex (Vasudeva 1958). Formation of oospores in the inflorescence takes place as it dries up. The disease is also found to be associated with white rust symptoms on leaves and inflorescence. Systemic infection results in

FIGURE 5.6 Downy mildew affected pods.

FIGURE 5.7 Staghead (hypertrophied inflorescence) caused by *Hyaloperonospora*.

thickened stunted growth of plant bearing profuse sporulation (Kolte 1985). In all the infected plant parts of oilseeds *Brassica*, total sugar, total phenol, total protein, and chlorophyll and nitrate reductase activity decreased, while chlorophyll content only in inflorescence and total free amino acids in the infected plant parts increased. However, total mineral content did not differ in infected and healthy plants (Singh 2000, 2004).

Geographical Distribution and Losses

The disease is found to appear more frequently in varying proportions, wherever rapeseed–mustard cultivation has been intensified. The disease has been reported from Argentina, Australia, Austria, Brazil, Bulgaria, Canada, Chile, China, Cuba, Cyprus, Czechoslovakia, Denmark, Ethiopia, Fiji, Finland, France, Germany, Greece, Hong Kong, Hungary, India, Iran, Iraq, Ireland, Israel, Italy, Jamaica, Japan, Kenya, Korea, Malaysia, Mexico, Nepal, the Netherlands, New Zealand, Norway, Panama, Pakistan, Philippines, Poland, Portugal, Romania, Russia, South Africa, Switzerland, Taiwan, Turkey, Uganda, the United Kingdom, the United States, Vietnam, and Yugoslavia (Kolte 1985, Saharan et al. 2005). Reports of its occurrence either alone (Porter 1926) or in association with white rust on leaves or inflorescence have been made, which could result in losses up to 58% (Kolte 1985). Seedling death could be even up to 75% when infection occurs at cotyledonary stage and congenial weather conditions are prevalent.

Pathogen

The causal pathogenic fungus *H. parasitica* (Pers.) Constant is an obligate parasite affecting all crucifers though variation exists in conidial size and other fungal structures among strains infecting different species of cruciferae. Mycelium is hyaline, coenocytic, remains intercellular in host, produces large, lobed intracellular haustoria, often branched, which nearly fill the entire cell. Erect conidiophores singly or in groups of determinate growth emerge vertically through the epidermis on the undersurface of the leaves through the stomata. Conidiophores are hyaline with a flattened base, stout main axis, twisted at a point crossing the stomata, and measure 100–300 μm. At the tip, conidiophores are dichotomously branched six to eight times, sterigmata slender, and acutely pointed. Conidia are hyaline, broadly elliptic to globose, 24–27 μm × 12–22 μm. A single conidium is borne at the tip of each branch, and the same is deciduous. Detachment of conidia is possibly caused by hygroscopic twisting of conidiophore related to changes in humidity. Conidiophore wall is uniformly thick. Spherical oogonia and tendril-like antheridia are developed on separate hyphae in hypertrophied tissue to produce oospores that enable the survival of the pathogen for long times withstanding harsh conditions. On germination of the oospore or conidia, the germ tube penetrates the host tissue directly or through the stomata. This pathogen, in the absence of fungal reproductive structures, during early interaction of seedlings, in infected young leaves packed in sealed plastic bags, and seed stocks can be diagnosed by a fast and reliable molecular identification technique, that is, multiplex PCR amplification of full internal transcribed spacer (ITS) and ITS2 regions of *H. parasitica* (Casimiro et al. 2004).

Epidemiology and Disease Cycle

The disease is favored by cool (8°C–16°C) and moist weather with low-light intensity and high (152 mm) rainfall (Kolte 1985). The oospores that survive in infected crop residue, soil, and seed serve as primary source of inoculum. These oospores germinate to infect cotyledonary and primary leaves. Systemic infection could result in staghead formation. Secondary infection of the plants occurs through airborne conidia or waterborne zoospores produced from germinating sporangia.

Disease Management

Host Plant Resistance

EC-129126 (*B. juncea*), PBN-9501, PBN-2002, and GSL-1 (*B. napus*) are reported to be resistant to the disease (AICRP-RM 1986–2014, Nashaat et al. 2004, Chattopadhyay and Séguin-Swartz 2005). However, there is danger of breakdown of resistance due to mixed infection with white rust. Seven genotypes, namely, *Sinapis alba*, *B. carinata* (HC-1), *B. juncea* (DIR-1507 and DIR-1522), and *B. napus* (GS-7027, Midas, and Tower) exhibited stable resistance to the disease (Dang et al. 2000). Genotypes RC 17, RC 346, RC 89, RC 110, and RC 280 were also resistant to the disease (Singh and Singh 2005). Australian genotypes JM06014 and JM06015 and Indian genotypes JM 3 and Kranti were resistant to downy mildew (Singh et al. 2010). Two Australian spring-type oilseed rape genotypes Pioneer 45Y77 and Pioneer 46Y78 were resistant to *H. parasitica* (Ge et al. 2008). Walters et al. (2005) observed three oxylipins, namely, TriHOE1, TriHOE2, and 13-HOT, in incompatible interaction between *B. napus* and downy mildew pathogen while these were not observed in compatible interaction, which indicates their involvement in signaling and/or as antimicrobial compounds in rapeseed-resisting infection by the pathogen. The resistance of some genotypes of rapeseed–mustard to Indian isolate of *H. parasitica* seems to be conditioned by a single dominant gene (Nashaat et al. 2004).

Cultural Control and Effect of Plant Extracts

Selective picking of affected hypertrophied racemes immediately after formation followed by their destruction; rotation with noncruciferous crops could also be helpful. Suitable planting time need to be worked out as per location. Seed treatment and successive foliar spray with garlic bulb extract was found an eco-friendly alternative to manage this disease (Kolte 1985, Bhatt et al. 2009).

Chemical Control

Seed treatment with metalaxyl 35 SD at 6 g/kg and spray of metalaxyl at 0.01% ai were effective in managing the disease (Chattopadhyay 2008). Gopal (2003) found that the seed treatment with metalaxyl along with its two sprays could reduce the DI significantly and increase the seed yield.

Sclerotinia ROT

Symptoms

Based on the symptoms, the disease has been named white blight, white rot, stem blight, stalk break, stem canker, or rape canker. Usually, under natural conditions, the stem of the plant is seen affected more frequently, though all aboveground parts are subject to attack by the disease. Symptoms on the stem become visible as elongated water-soaked lesions that later are covered by a cottony mycelial growth of the fungus (Figure 5.8). Infected plants are at times overlooked until the fungus grows completely throughout the stem to rot it. When the stem is completely girdled by such lesions, the plant wilts and dries. Foliage may show little sign of attack while at times may even start on leaves, which wilts and droops downward and then moves on to the stem. Sometimes, the infection is restricted to a smaller area of pith, which results in slow stunting of the plant and premature ripening rather than their sudden collapse. Such plants under field conditions can be easily identified because of premature ripening. The affected stem tends to shred; numerous grayish-white to black, spherical sclerotia appear either on the surface or in the pith of the affected stem. When the crop is at seed maturity, the plants tend to lodge, touching the siliquae with the soil level. Such plants, though remain free from stem or aerial infection throughout, show rotting of the siliquae with profuse fungal growth, along with sclerotial bodies just above the soil level. Appearance of the disease at an early stage of crop growth results in the death of whole plant (Kolte 1985). Sharma and Sharma (2001) reported significant reduction in seed germination percentage, radicle growth,

FIGURE 5.8 Sclerotinia rot-affected stem.

plumule growth, plant height, secondary branches, and number of silique on primary and secondary branches in Indian mustard cultivars in Haryana, India. Younger plants (up to of 40 days) are highly susceptible as compared to older ones (Ghasolia and Shivpuri 2009). After infection, Alizadeh et al. (2006) observed an increase in glucosinolate and erucic acid content, while the test weight and oleic acid decrease in rapeseed oil.

Geographical Distribution and Losses

Rot of mustard caused by *Sclerotinia sclerotiorum* (Lib.) de Bary has become important in recent times with high (up to 66%) DI and severe yield losses (up to 39.9%) leading to discouragement of growers of the crop (Chattopadhyay et al. 2003), although reports of even 100% yield loss due to the disease are available (Saharan et al. 2005). *Sclerotinia* rot is also a serious threat to oilseed rape production with substantial yield losses worldwide including Australia, Europe, India and North America (McCartney and Lacey 1999, Hind et al. 2003, Koch et al. 2007, Malvarez et al. 2007, Singh et al. 2008). There may be a great variation in losses in yield in the same area from year to year. Yield losses vary with the percentage of plants infected and the stage of growth of the crop at the time of infection. Plants infected at the early flowering stage produce little or no seeds and those infected at the late flowering stage set seed and may suffer little yield reduction. For predicting yield (Y), a linear equation (R^2: 0.89) was fitted on DI ($Y = 310.25 - 2.04^{**}DI$) using *Sclerotinia* susceptible cv. Rohini (Chattopadhyay et al. 2003). The disease affects broad-leaved crop species and is most common in temperate regions of the world. The first record of its occurrence on rapeseed and mustard appears to have been made from India (Shaw and Ajrekar 1915). Since then, frequent occurrences of the disease in severe form have been reported from Argentina (Gaetan and Madia 2005), Brazil, Canada (loss up to 28%), China, Denmark, Finland (Kolte 1985),

Florida (Young et al. 2012), France, Germany (Kolte 1985), Greece (Tziros et al. 2008), India (Kolte 1985), Italy (Corato and de Baviello 2000), Sweden (Kolte 1985), Texas (Isakeit et al. 2010), and the United Kingdom (Kolte 1985). The disease has been found to be causing severe losses in Rajasthan, Bihar, Uttar Pradesh, Uttarakhand, and Haryana states of India. In one of the surveys conducted at the Directorate of Rapeseed–Mustard Research, Indian Council of Agricultural Research, India (Jha and Sharma 2003), *Sclerotinia* rot has been rated as the most important of eight problems being faced by the farmers of Bharatpur district (Rajasthan, India) in mustard culture. Due to this disease, Shukla (2005a) reported 50.88% yield loss in mustard crop. The pathogen is reported to have a wide host range, known to infect about 408 plant species (Boland and Hall 1994) with no proven source of resistance against the disease reported till date in any of the hosts.

Pathogen

The pathogen is *S. sclerotiorum* (Lib.) de Bary (Syn. *Sclerotinia libertiana* Fuckel; *Whetzelinia sclerotiorum* [Lib.] Korf and Dumont). Mycelium is thin, 9–18 μ in diameter with lateral branches of smaller diameter than the main hyphae. The vegetative hyphae are multinucleate ($n = 8$). Mycelial growth rate on solid agar media is fast and forms a moderate to abundant amount of aerial mycelium. The sclerotia are black, round or semispherical in shape measuring 3–10 μm. These are formed terminally and produced in one or two concentric rings on agar culture media. Sclerotia can be easily detached from the medium. The fungus does not have an obvious true conidial stage, though formation of microconidia in culture media has been reported. The mature sclerotium consists of an outer-pigmented rind and a medulla of prosenchymatous tissues partly embedded in a gelatinous matrix. Several other aspects on the morphology of sclerotium development, physiological and biochemical aspects of sclerotia formation, maturation, and structure of sclerotium have been reviewed by Willets and Wong (1980).

The sclerotial germination is mycelogenic (by mycelium) or carpogenic (by the formation of apothecia). On germination, the sclerotia form stalked apothecia. One to several apothecia may grow from a single sclerotium. The hymenium is made of palisades of asci and paraphyses. The asci measure 119–162.4 μ × 6.4–10.9 μ in size. These are inoperculate, cylindrical, narrow, rounded at the apex with eight ascospores in each ascus. Ascospores are uniform in size ($n = 8$). They measure 10.2–14.0 μ × 6.4–7.7 μ in size. Each ascospore is hyaline, ellipsoid, and has smooth walls. The spores are bi- or triguttulate. The paraphyses are about 100 μm long, 1–2 μm in diameter, slightly swollen at their tips, multinucleate, sparsely septate, and occasionally branched at the bases. The effects of some factors, such as age of sclerotium, temperature, light, and moisture, on apothecial production and ontogeny of apothecia have been reviewed by Willets and Wong (1980). The fungus grows over a range of 0°C–35°C, optimum being 20°C–25°C. Initiation and development of apothecia occur over a range of 10°C–20°C, while 20°C ± 1°C was found better for the same on the sterilized moist sand substrate (Goswami et al. 2012). It attacks field, forage, vegetable and ornamental crops, trees and shrubs, and numerous herbaceous weeds. There is little or no evidence of physiological specialization (Kolte 1985), though variation in pathogenicity has been reported (Goyal et al. 2013c).

Ascospores discharged from the apothecia at the base of the plants in soil constitute important primary source of infection. These ascospores could be stored at −80°C in 30%–40% glycerol for up to 12 months and used as a reliable source of inoculum for pathogenicity test (Olivier and Seguin-Swartz 2006). Mycelium in soil or those arising from sclerotia is a less important initial source of infection because of the low competitive saprophytic ability of the fungus (Kolte 1985). The ascospore can germinate in the presence of a thin film of water, in less than 24 h at 5°C–30°C, optimum being 5°C–10°C. The ascospore gives rise to infection hypha, and initial penetration of the tissue takes place directly by mechanical pressure through the cuticle, or the infection hypha may also penetrate already wounded or injured tissue. After the entrance of the fungus into the host, the mycelia cause enzymatic dissolution of the cell wall in advance, and cells die some distance ahead of the invading hyphae. Pectolytic enzymes are responsible for tissue maceration indirectly

damaging the cell membrane, which results in subsequent death of cells (Kolte 1985). Production of cell wall–degrading PMG and cell (C_x) enzymes by *S. sclerotiorum*–infecting *Brassica* plants has been reported (Kolte 1985) that results in the colonization of plant tissues. Virulence of different isolates appears to be associated with the activity of PMG and C_x enzymes. These PGs can activate defense reactions in hosts, having no relation with the enzyme activity (Wang et al. 2008a). Li et al. (2004) reported that an sspg1d gene (endo-PG) is highly expressed in the pathogen under pathogenic conditions. Activities of PO and SOD enzymes were higher in resistant cultivars than susceptible cultivars of rape (Qi et al. 2004). A selective phytotoxin, sclerin, has been found to be produced by *S. sclerotiorum* in three susceptible cruciferous species, namely, *B. napus*, *B. juncea*, and *S. alba* (Pedras and Ahiahonu 2004), but not in a resistant species, namely, *E. gallicum*. However, three phytoalexins, namely, indole-3-acetonitrile, arvelexin, and 1-methoxyspirobrassinin, were found to be elicited in *E. gallicum* in response to the pathogen infection. The role of protease activity in infection of plants of *B. juncea* is also reported (Kolte 1985). A gene (ssv263) encoding a hypothetical, novel protein with unknown function (Liang et al. 2013), and an arabinofuranosidase/beta-xylosidase precursor (Yajima et al. 2009) from *S. sclerotiorum* has been identified as a possible virulence factor in the pathogen. A phytoalexin-detoxifying gene, namely, brassinin glucosyltransferase 1, that detoxifies brassinin phytoalexin in the host has been found to be induced in *S. sclerotiorum* in response to the infection of the host (Sexton et al. 2009). It appears that invasion of tissues of *B. juncea* is also related to the infection process, mediated by production of a toxin that is identified as oxalic acid (Kolte 1985); the oxalic acid is formed in culture filtrate as well as in infected *B. juncea* plants, which is reported to be thermostable, translocatable, and treatment of the host plant with culture filtrate results in disease. Oxalic acid may play a significant role in activating the glucosinolate–myrosinase system during infection (Rahmanpour et al. 2010). Resistance at the cellular level in *B. napus* against the pathogen is a result of retardation of pathogen development on the plant surface and within plant tissues. In resistant lines, formation of appresoria and infection cushions is suppressed that caused extrusion of protoplast from hyphal cells and produces a hypersensitive reaction. In susceptible lines, calcium oxalate crystals are found throughout the leaf tissues, while they are mainly confined to the upper epidermis of the resistant lines, and starch deposits are also more prevelant in susceptible lines (Garg et al. 2010a). Modulation of 32 proteins involved in photosynthesis and metabolic pathways, protein folding and modifications, hormone signaling, and antioxidant defense has been observed in *B. napus* in response to *Sclerotinia* infection (Liang et al. 2008).

According to Huang et al. (2008), on the surface of leaves and stems, infection cushions of different sizes develop that are often flattened and increased in diameter. These infection cushions and network of mycelia are covered by mucilage produced by the pathogen. After removing the infection cushions, numerous penetration pegs enter the cuticle of leaves and stems through the pores. Small changes are observed in cuticle. After penetration, the hyphae grow inter- and intracellularly between the cuticle and epidermal cells and also colonize xylem and phloem. Pathogen may secrete cell wall–degrading enzymes, namely, cellulases, pectinases, and xylanases, which degrade cellulose, pectin, and xylan in the host cell walls during infection and spread in the host tissues. Increase in proline content and PO activity during decrease in malonaldehyde content, free amino acid contents, and polyphenol oxidase activity and conductivity have positive correlation with rape resistance to the pathogen (Zhao et al. 2006).

Cultural, morphological, pathogenic characteristics and carpogenic germination of *S. sclerotiorum* have been studied by some workers (Goswami et al. 2012). Potato dextrose agar medium was the best supporting mycelia growth of the fungus and produced maximum number of sclerotia (Nguyen et al. 2006). Differences in the morphology of *S. sclerotiorum* isolates have previously been observed by Li et al. (2003a), where isolates producing tan sclerotia were identified. Sexton et al. (2006) demonstrated genotypic diversity utilizing microsatellite markers among *S. sclerotiorum* isolates of oilseed rape crops from Southeast Australia. Very few reports exist to date describing dark-pigmented isolates of *S. sclerotiorum*, such as those from Canada and

southwestern region of the United States (Lazarovits et al. 2000, Sanogo and Puppala 2007). Recently, the molecular biology approaches enable to evaluate similarity and differences between strains within plant pathogens. Akram et al. (2008) worked on variability among isolates of *S. sclerotiorum*. Irzykowski et al. (2004) observed genetic diversity in natural populations of *S. sclerotiorum* from China by using RAPD molecular marker in addition to comparison of sequences of ITS1-5.8s-ITS2 region. Sharma et al. (2013b) used RAPD analyses of 15 geographical isolates, while Chen et al. (2010a) did sequence-related amplified polymorphism analyses of 76 isolates and observed high polymorphism among them. Genetic diversity based on morphological characteristics (Barari et al. 2011) was assessed by using rep-PCR genomic fingerprinting (Karimi et al. 2011) among geographically different isolates of *S. sclerotiorum*. Pathogenic diversity and genetic structure of the pathogen have been assessed through comparison in virulence and mycelial compatibility between isolates (Karimi et al. 2012). In Turkey, genetic and morphological diversity has been demonstrated for the first time within a population of *S. sclerotiorum*–infecting oilseed rape (Mert-Turk et al. 2007).

Morphogenic and pathogenic diversity among 38 isolates of *S. sclerotiorum* from different locations of Rajasthan, India has been reported (Ghasolia and Shivpuri 2007). Nie et al. (2010) and Ling et al. (2011) observed pathogenic diversity among 495 isolates of *S. sclerotiorum* from different regions of Shaanxi Province and 24 isolates of *S. sclerotiorum* from different regions of Anhui region of China. Garg et al. (2010b) studied the pathogenicity of morphologically different isolates of *S. sclerotiorum* from different regions of western Australia. High degree of pathogenic and genetic diversity has been observed among 17 isolates of *S. sclerotiorum* from India and the United Kingdom (Goyal et al. 2013c). Hence, similar holistic study should be conducted with higher number of *S. sclerotiorum* isolates from different geographical regions, which could provide a better picture of divergence among the pathogen and could be helpful in the generation of resistant material against the stem rot in oilseed Brassicas.

Inoculum of *S. sclerotiorum* can be detected in field-based air samples (using a Burkard spore trap) and from petals by PCR assay of nuclear ribosomal ITS sequences (Freeman et al. 2002). The presence of *S. sclerotiorum* on plants may be detected by using immunological detection method, namely, dimeric single-chain fragment variable (scFv) antibody with affinity for the pathogen (Yajima et al. 2008) and polyclonal antibody-based immunoassay (Bom and Boland 2000). Petal infection by *S. sclerotiorum* can be rapidly detected by real-time PCR (RT-PCR) (Yin et al. 2009) and nested PCR (Qin et al. 2011) techniques. Although detection of ascospores of *S. sclerotiorum* can be done by using passive trap, volumetric trap, and PCR techniques (Rogers et al. 2008, 2009) can also be used for the quantification of the ascospores (Penaud et al. 2012).

Epidemiology and Disease Cycle

Sclerotinia rot was positively correlated with increase in soil moisture and RH (R^2: 0.87 and 0.99, respectively), both at sowing and during the flowering period (50–60 DAS). Gupta et al. (2004b) found delay in sowing of rapeseed–mustard crop and reduced DI. High seeding rate and plant density increase the potential for lodging, which may be responsible for plant-to-plant spread of this disease (Jurke and Fernando 2008). Combination of cool weather and high soil moisture during the critical stage of 60–70 days age of crop favored higher incidence on Indian mustard (Sharma et al. 2009). Sharma et al. (2010c) observed petal infection with ascospores during full bloom stage and found rainfall as an important factor in carpogenic infection of *S. sclerotiorum* in *B. juncea*. Detection of healthy Indian mustard crop and its early differentiation from *Sclerotinia* rot–affected *B. juncea* plants was possible using remote-sensing technique, which could help in multistage disease tracking and forecasting (Dutta et al. 2006, Bhattacharya and Chattopadhyay 2013). Singh et al. (2000) developed a stepwise multiple linear regression model for *Sclerotinia* rot of Indian mustard. Under epidemiological study of *Sclerotinia* rot, based on DI and 10 independent weather variables, a multiple linear regression model has been described. The equation of the fitted model is percent

Sclerotinia rot incidence = −11.2351 + 0.9529*BSSH + 4.93924*Eva + 3.83308*pH + 0.60885*RF (mm) − 0.406458*RH 720 + 0.524095*RH 1420 + 0.17386*Soil moisture (%) − 0.30461*T_{max} − 0.677744*T_{min} − 2.19556*WS (DRMR 2010). The ScleroPro system is easy to handle and fully computerized, and based on the weather and field-site-specific data, this program has been available for growers and advisors since 2006 (Koch et al. 2007).

The pathogen primarily survives from one crop period to another in the soil through sclerotia. Such sclerotial bodies get mixed with the soil through affected plant debris after the crop is harvested, or when seeds contaminated with the sclerotial bodies are sown in the soil. Samples have been found to contain up to 432 sclerotia/kg seed, and a certain level of sclerotia in soil is reported to be maintained by the formation of secondary sclerotia (Kolte 1985). There are reports that the fungus can also survive through either mycelium or ascospores in dead or live plants, on testa of seed (Kolte 1985). Some wild plants that act as primary source of inoculum are hogweed (*Heracelum sphondylium* L.), cow parsley (*Anthriscus sylvestris* [L.] Holfm.), *Chenopodium* spp., and *Asphondilia* spp., which are also found to be infected and carry over the pathogen.

The disease is also known to be airborne, while seed treatments were assessed for protection against seed- and soil borne nature of the pathogen. Since aerial infection, apart from that taking place in the soil, is dependent entirely on continued production and dissemination of ascospores, epidemics are common in areas of continuously cool moist weather concurrent with the susceptible stage of crop, particularly the flowering period. Fields sown with rapeseed for 2 years favor more germination of sclerotia than fields sown with the crop for 1 year. Pollen and petals of rapeseed are known to stimulate ascospore germination. Rapeseed crops do not appear to restrict the movement of airborne ascospore. Ascospores are carried into the air current as high as 147.0 cm above the soil level. Spores could be trapped at a horizontal distance of 150 m from source indicating that the ascospores being airborne are carried to sufficient distance and cause spread of the disease from field to field. There did not appear to be a correlation between total rainfall and ascospore incidence. It is observed that ascospores on pollen grains of rapeseed adhere tightly. Honeybee-carried pollen and pollen in honeycombs have also been reported to carry ascospores. However, in view of the readily available wind-borne inoculum, the relative importance of transfer of spores by the honeybees is of less significance (Kolte 1985).

Spray of herbicide barban on rapeseed crop, used for managing *Orobanche*, increased its susceptibility to infection by *S. sclerotiorum*, possibly through altering the physiology of the plant as the herbicide has no inhibitory effect on the *B. juncea* plants. The disease has been noted to be high in plots, where *Orobanche* incidence is low and *vice versa*. Susceptibility of the plants to *Sclerotinia* rot is more when ammonium sulfate was applied, while thiourea spray showed less intensity of the disease (Kolte 1985).

Disease Management

Host Plant Resistance

Because of the wide host range and lack of tissue specificity, breeding resistant varieties appears to be less successful. However, differences in general growth habit and morphological characters of plants might be important characteristics for tolerance of the disease. For example, the *Omi nature* variety of *B. napus*, of medium height, early maturity, with a stiff stem and many branches, was resistant to the disease. The *Isuzu* variety of *B. napus* had a high degree of resistance (Kolte 1985). Another character, that is, stem diameter of the plants, may be a useful parameter for tolerance of the pathogen (Li et al. 2006a). It has also been reported that the high-glucosinolate lines are more susceptible to *S. sclerotiorum* as compared to the low-glucosinolate ones (Song and Guan 2008). Several genotypes of rapeseed–mustard have been screened against *Sclerotinia* rot caused by *S. sclerotiorum* by using different methods under natural and artificial conditions (Ghasolia and Shivpuri 2005a, Chand and Rai 2009, Prasad et al. 2009a, Sharma et al. 2012b). Responses of some genotypes (e.g., cv. Charlton) were observed relatively consistent

irrespective of the isolates of the pathogen, whereas highly variable responses were observed in some other genotypes (e.g., Zhongyouang No. 4, Purler) against the same isolates. Genotypes with higher levels of resistance need to be included in oilseed *Brassica* breeding programs to enhance the level of field resistance in cultivated *B. napus* and *B. juncea*. Although complete resistance has not been identified in canola, partial field resistance to *Scleritinia* rot in Chinese cultivars Zhongyou 821 (Li et al. 1999) and Zhongshuang No. 9 (Wang et al. 2003) has been identified. Four cultivars of *B. napus*, namely, BOH 2600, Bermuda, Capio, and Mohican, were found resistant to *S. sclerotiorum* after a 3-year study (Starzycka et al. 2004). For the three consecutive cropping seasons, eight genotypes, namely, Hyola-401, PBN-9501, PWR-9541, Kiran, RH-9401, RH-492, RW-8410, and PAB-9511, were found resistant (percent DI [PDI] < 1%) to moderately resistant reaction (PDI = 1%–10%) to *S. sclerotiorum* (Ghasolia and Shivpuri 2005a). Genotype Ringot I of *B. juncea* was reported resistant to the rot (Goyal et al. 2011b). Other resistant *B. napus* genotypes that have been previously reported are 06-6-3792 (China), ZY004 (China), RT 108 (Australia) with mean stem lesion lengths < 3.0 cm (Li et al. 2007b, 2008b), and ZY006 with mean stem lesion lengths < 0.45 cm (Li et al. 2008b). In addition, the levels of resistance reported previously in *B. juncea* were, in particular, far lower, for example, *B. juncea* JM 06018 and JM 06006 with mean stem lesion lengths of 4.8 cm (Li et al. 2008b), as compared with *B. napus* genotypes. However, the situation has begun to improve because of the screening of the sources of *Sclerotinia* resistance from Chinese native cultivars (Li et al. 2009b). Garg et al. (2010c) reported high levels of resistance against *S. sclerotiorum* in introgression lines derived from *Erucastrum cardaminoides*, *Diplotaxis tenuisiliqua*, and *Eriospermum abyssinicum*. The novel sources of resistance identified in this study are a highly valuable resource that can be used in oilseed *Brassica* breeding programs to enhance resistance in *B. napus* and *B. juncea* cultivars against *Sclerotinia* rot. Results indicate that more than one *S. sclerotiorum* isolate should be included in any screening program to identify host resistance. It has also been reported that concentration and culture time of mycelial suspension of the pathogen and time for maintaining high RH after inoculation play a major role in disease development in the inoculated plants (Zang et al. 2010). Unique genotypes, which show relatively consistent resistant reactions (e.g., cv. Charlton) across different isolates, are the best for commercial exploitation to breed for resistance in oilseed *Brassica* against *Sclerotinia* rot (Garg et al. 2010c).

Molecular Breeding

PG inhibitor genes (Bnpgip1 and Bnpgip2) (Li et al. 2003b, Hegedus et al. 2008) and EIN3 gene (Xu et al. 2009a) in *B. napus* may play an important role in resistance to *S. sclerotiorum*. Early induction of germin-like genes, namely, BnGLP3 and BnGLP12, that participates in an oxidative burst could play a vital role in defense of *B. napus* against the pathogen (Rietz et al. 2012). This oxidative burst can be detected in vivo in infected oilseed rape by using a modified platinum electrode on which Pt microparticles were dispersed and coated with a poly(*o*-phenylenediamine) film (Xu et al. 2009b). Sequential activation of salicylic and jasmonic acid signaling has been found to be associated with defense in oilseed rape against the pathogen (Wang et al. 2012). The SA levels in *Sclerotinia* rot–infected oilseed rape can be detected by using copper nanoparticles–modified gold electrode (Wang et al. 2010a). The LOX2 gene (Ren et al. 2010a) and PDF1.2 gene (Ji et al. 2009) in *B. napus* may be involved in jasmonate-mediated defense against *S. sclerotiorum*. Transformed lines of canola showed improved resistance to *S. sclerotiorum* with A9Ss gene from *Pseudomonas alcaligenes* strain A9 (Guo et al. 2006), oxalate oxidase gene (Zou et al. 2007, Dong et al. 2008), hrf2 gene (harpinXooc protein) from *Xanthomonas oryzae* pv. *oryzicola* (Ma et al. 2008), Ovd gene from *Orychophragmus violaceus* (Wu et al. 2009), pathogen-specific scFv antibody (Yajima et al. 2010), and pgip1 gene (PG-inhibiting proteins [PGIPs]) from bean cv. Daneshjoo (Abedi et al. 2011). Introduction of glucose oxidase gene into *B. napus* has also been reported to increase resistance to the pathogen (He et al. 2007).

Cultural Control

Management is difficult, inconsistent, and uneconomical due to the presence of wide host range and long-term survival of the resting structures. Since the disease is carried over through sclerotia with crop debris and refuse of the plants stimulate sclerotial formation, it is advisable to collect and burn all the infected stubbles to kill the sclerotia (Vasudeva 1958). For sowing, sclerotia-free clean seeds should be used. In view of the airborne infection through ascospores and a wide host range of about 408 species, use of crop rotation appears to be a less successful method for managing the disease. However, deep summer ploughing and crop rotation with nonsusceptible hosts (rice, maize), use of only recommended dose of nitrogenous fertilizer, irrigation and keeping plant population within limits of recommendation, and flooding of soil, if possible, appear to minimize the sclerotial population in the soil, which subsequently might prove useful in control of the disease resulting from soil borne inoculum (Yuan et al. 2009). Avoidance of overcrowding of plants in a row to minimize plant-to-plant contact through root and stem to aid reduction of disease spread by mycelial means appears effective. Keeping a check on broad-leaf weeds like *Chenopodium* spp. is important in checking the disease. Occurrence of *Sclerotinia* blight can be reduced or avoided by late sowing of the rape (Fei et al. 2002). Late sowing might be helpful under the conditions of Canada by shortening the overlap between phenological susceptibility and exposure to maximum ascospore load (Kolte 1985). In Rajasthan (India), some conditions like sandy soil with late sowing and less irrigation were helpful in lowering DI (Ghasolia et al. 2004), while in Haryana (India), conditions like late sowing with presowing and three supplemental irrigations at branching, flowering, and pod formation were helpful in reducing DI (Sharma et al. 2001). Soil application of compost inhibited carpogenic germination of *S. sclerotiorum* and reduced *Sclerotinia* infection (Couper et al. 2001). Extracts of five organic amendments, namely, sunflower cake, safflower cake, mustard cake, neem cake, and farmyard manure, significantly reduced mycelial growth of the *S. sclerotiorum* (Tripathi et al. 2010). The enzymatic hydrolysis of glucosinolates in the *Brassica* releases ITCs, which could be potentially useful in curbing the pathogen (Kurt et al. 2011). Combination effect of micronutrients, namely, B at 1 g/L, Mo at 1 g/L, S at 2 g/L, and Zn at 2 g/L, in reduction of *Sclerotinia* rot incidence and increase in yield in rapeseed–mustard have been reported (Mondal 2008). Role of N in incidence of *Sclerotinia* rot of oilseeds *Brassica* is confusing (Gupta et al. 2004c, Shukla 2005b).

Biological Control

Chattopadhyay et al. (2002) reported a few of the biological treatments (seed treatment with *T. viride* and *A. sativum* aqueous bulb extract) to be effective against the disease coupled with effects of growth promotion and better plant stand and yields, which surpassed the efficacy of carbendazim on *Sclerotinia*-infested farmers' fields (Meena et al. 2006). Integration of the seed treatment with foliar sprays reaped better reduction of the disease (Chattopadhyay et al. 2004, 2007, Yadav 2009). *Trichoderma atroviride* showed coil formation and penetration of pathogen hyphae (Matroudi et al. 2009). Soil application of *T. harzianum* at 15 g/kg soil simultaneously or 7 days prior to the pathogen resulted in low disease intensity (Mehta et al. 2012). Wu and Wang (2000) reported that W-1 strain of *Caseobacter* spp. can manage the pathogen. Carpogenic germination of sclerotia of the pathogen could be reduced by using a bioagent *Gliocladium virens* (Ghasolia and Shivpuri 2005b). Antifungal activity of 11-3-1 strain of S*treptomyces longisporoflavus* against *S. sclerotiorum* has been observed (Han et al. 2012). The *Pseudomonas fluorescens* P13 isolated from oilseed rape field soil produced hydrogen cyanide (Li et al. 2011), and *Pseudomonas chlororaphis* PA-23 induced canola plants to produce more hydrolytic enzymes, namely, chitinase and beta-1,3-glucanase (Fernando et al. 2007), in response to the infection of *S. sclerotiorum*, thus was effective against the pathogen. However the control of pathogen by *Psuedomonas* strain DF41 is dependent upon lipopeptide production and the presence of a functional Gac system in the bioagent (Berry et al. 2010). Chitinase activity of different genotypes of *B. napus* is significantly correlated with their *Sclerotinia* rot scores and suggested that chitinase can be used in breeding program for improving disease resistance in rape.

Seed treatment with a bacterial strain, namely, *Mesorhizobium loti* MP6, isolated from root nodules of *Mimosa pudica* resulted in enhanced seed germination, early vegetative growth, and seed yield with drastic decline in incidence of *Sclerotinia* rot (Chandra et al. 2007). Y1 (Yan et al. 2005), NJ-18 (Yang et al. 2009a), YS45 (Zhang et al. 2009), Tu-100 (Hu et al. 2005, 2011), and EDR2 (Gao et al. 2013) strains of *B. subtilis* and BS6 strain of *Bacillus amyloliquefaciens* (Fernando et al. 2007) have been promising against this disease in oilseed rape. A new antifungal protein produced by *Bacillus licheniformis* W10 could be used as biofungicide to curb this disease (Sun et al. 2007). A bioagent, namely, *Coniothyrium minitans*, that destroys the hyphae (Jiang et al. 2000) and the sclerotia (Cael et al. 2001, Penaud and Michi 2009) of *S. sclerotiorum* has been used to control the disease. This bioagent degrades oxalic acid to nullify the pH effect thereof. Further, this may stimulate the production of beta-1,3-glucanase by the bioagent and may improve the mycoparasitism of the agent on *S. sclerotiorum* to result in protection of the plants from infection by the pathogen (Ren et al. 2007). Water-assisted application of *C. minitans* at the time of transplanting oilseed rape seedlings has been found effective in suppressing carpogenic germination of the pathogen (Yang et al. 2009b). Treatment of soil with this bioagent was found effective in reducing ascospore production by the pathogen (Huang and Erickson 2004). Drenching of deep soil with spores of *C. minitans* could be done before or after crop planting for increasing long-term efficacy on a regular basis on infected plots (Luth et al. 2012). Li et al. (2006b) reported aerial application of this bioagent as an effective method to curb the mycelial growth of the pathogen on petals. Tautomycin produced by *Streptomyces spiroverticillatus* and other related compounds, namely, 2,3-dimethylmaleic anhydride, diphenylmaleic anhydride, and dimethyl maleate, have significant potential against the pathogen (Chen et al. 2011). Sawdust soil–based bioformulation of *P. fluorescens* PS1 caused morphological alternation by hyphal perforation that enable to curb the disease (Aeron et al. 2011).

Chemical Control

In addition to contamination of seed, viable sclerotia present a potential quarantine hazard in export of seed. Viable sclerotia in infested seed of oilseed *Brassica* could be eradicated by fumigation with methyl bromide (Kolte 1985). In order to check the secondary spread of the disease, the possibility of control of the disease through foliar sprays of chemicals has been investigated. Since the pathogen is soil borne, application of chemicals to soil for managing the disease is not only of limited value but also hazardous to environment. Certain chemicals such as quintozene, fentin acetate, and calcium cyanamide have been found effective to inhibit the apothecial development of the fungus. The efficacy of calcium cyanamide in controlling the disease by 40%–90% has been confirmed under field conditions in Germany (Kolte 1985). Ridomil MZ (mancozeb + metalaxyl) as a seed dresser effected highest germination with no postemergence mortality by *S. sclerotiorum* (Pathak and Godika 2002a). Seed treatment at sowing and foliar spray at first budding/flowering with 0.2% of benomyl proved best on farmers' field (Chaudhary et al. 2010). Use of carbendazim at 0.25% as foliar spray could be effective in controlling this disease (Kolte 2005). Application of fungicides at full flowering phase (Jajor et al. 2010) by using venturi nozzle technology (Kutcher and Wolf 2006) was effective in reducing infection by the pathogen. Foliar spray of zinc pyrithione curbed the pathogen (Wang and Yang 2007).

Since no single method can effectively manage *S. sclerotiorum*, the best approach to control the pathogen is by integration of various eco-friendly measures. In recent years, an increasing consciousness about environmental pollution due to pesticides, and development of fungicide-resistant strains in *S. sclerotiorum* (Penaud et al. 2003) has challenged plant pathologists to search for eco-friendly tools for *Sclerotinia* rot management. Boscalid (trade name *Cantus* in China) is a new broad-spectrum fungicide belonging to carboxamides class. It inhibits the enzyme succinate ubiquinone reductase (Complex II), also known as succinate dehydrogenase, in the mitochondrial electron transport chain (Wang et al. 2009, Zhang et al. 2009, Gu et al. 2012). Use of such methyl benzimidazole fungicides in oilseed Brassicas to manage *Sclerotinia* rot has been reported to result in widespread fungicide-resistant strains of *S. sclerotiorum* (Penaud et al. 2003). A new fungicide,

namely, prochloraz-manganese chloride, is found to be effective in delaying both myceliogenic and carpogenic germination of *S. sclerotiorum*; thus, it has both protective and therapeutic effects on the disease (Ren et al. 2010b).

POWDERY MILDEW

Symptoms

Powdery mildew appears in the form of dirty-white, circular, floury patches on both sides of lower leaves (Figure 5.9) of the infected plants. Under favorable environmental conditions (relatively higher temperature), the floury patches increase in size and coalesce to cover the entire stem and leaves. Severely affected plants remain poor in growth and produce less siliquae. Green siliquae also show white patches in the initial stage of infection. Later, such siliquae become completely covered with a white mass of mycelia and conidia. Severely diseased siliquae remain small in size and produce small shrivelled fewer seeds at the base with twisted sterile tips. As the season advances, under favorable conditions, cleistothecia may be formed on both sides of affected leaves, stems, and siliquae, which become visible in the form of black scattered and/or concentrated bodies (Kolte 1985).

Geographical Distribution and Losses

Occurrence of powdery mildew on oilseeds *Brassica* is reported from France, Germany, India, Japan, Argentina, Australia, Sweden, Turkey, the United Kingdom, and the United States (Kolte 1985, Gaetan and Madia 2004, Kaur et al. 2008b). It is generally believed that the disease does not cause much damage to oilseed *Brassica* crops except in occasional severe outbreaks, when all the leaves and siliquae get covered with the powdery growth of the fungus at early phonological stage. In certain states of India such as Gujarat, Haryana, Madhya Pradesh, Rajasthan, and Uttar Pradesh, the disease has been found to occur quite severely, possibly as an effect of climate change (Kumar et al. 2013) resulting in considerable loss in yield. Kohire et al. (2008a) observed 40% yield loss in

FIGURE 5.9 Powdery mildew.

Indian mustard. Considering the differences in disease intensity from year to year, it appears that the loss is proportional to the disease intensity, which varies considerably depending on the stage at which it occurs.

Pathogen

The pathogen is *Erysiphe cruciferarum* Opiz ex. Junell. The mycelium is ectophytic. Penetration is confined to the epidermal cells, in which haustoria form, the remainder of the fungus being extramatrical. Conidiophores arise from the superficial hyphae on the host surface. Conidiophore is septate, and conidia are borne singly. Conidia are ellipsoid to cylindrical. Ripe conidia fall off quickly and are disseminated by wind. Conidial size ranges 8.3–20.8 μm × 20.8–45.8 μm with an average range of 12.58–14.9 μm × 31.0–36.9 μm. Conidia germinate at optimum of 20°C–25°C by the formation of two types of straight germ tubes, one is short with slightly lobed appressoria and another is long with unlobed appressoria. The length of germ tube ranges 20–30 μm. Cleistothecia are globose to subglobose with numerous hypha-like brownish septate appendages. They are pinkish brown when young and turn brown to dark brown on reaching maturity. Cleistothecia measure 83.2–137.3 μm in diameter (av. 104.4–119.1 μm) on different species and varieties of *Brassica*. The number of asci varies 3–8 per cleistothecium, each ascus producing 2–6 ascospores. Asci are subglobose to broadly ovate, not stalked, light brown to yellowish in color, and measure 25.0–37.4 μm × 41.6–66.6 μm with an average range of 31.7–34.5 μm × 52.3–62.0 μm on different species and varieties. Ascospores are ovoid and measure 19–22 μm × 11–13 μm.

Epidemiology and Disease Cycle

The disease is favored by relatively dry weather conditions (Kohire et al. 2008b). Initiation of powdery mildew disease in mustard occurred during 50–120 DAS. Severity of the disease was favored by >5 days of ≥9.1 h of sunshine, >2 days of morning RH of <90%, afternoon RH 24%–50%, minimum temperature >5°C, and a maximum temperature of 24°C–30°C. Regression analysis showed maximum temperature and afternoon RH of the week preceding the date of observation was positively and negatively linked, respectively, to the disease severity (R^2: 0.9) (Desai et al. 2004). It is possible to forecast the occurrence of the disease using weather-based models (Laxmi and Kumar 2011). Cleistothecial formation appears to be favored by alternating low and moderate temperature, low nutrition of the host, low RH, dry soil, and aging of the host (Kolte 1985). Late sowing and frequent crop irrigation increase the incidence and severity of disease (Kohire et al. 2008c). *B. rapa*, *B. nigra*, *B. juncea*, *C. bursa-pastoris*, *Coronopus didymus*, and *R. sativus* have been found susceptible to *E. cruciferarum* (Kolte 1985). The pathogenic fungus is likely to carry over from season to season through cleistothecia or as mycelium in volunteer host plants. These cleistothecia release ascospores that cause infection on lower leaves during favorable condition. The secondary spread of the disease occurs through the conidia produced in infected leaves.

Disease Management

Host Plant Resistance

Limited sources of resistance against the powdery mildew has been reported in *B. alba*, *B. alboglabra*, *B. rapa* var. brown sarson, *B. chinensis*, *B. japonica*, and *E. sativa* (Kolte 1985). Five genotypes, namely, RN-490, RN-505, PBC-9221, PBN-9501, and PBN-9502, were resistant against powdery mildew (Pathak and Godika 2002b). Two Australian (JM06009, JM06012) and two Indian (JM3, Kranti) genotypes were resistant to powdery mildew (Singh et al. 2010).

Molecular Breeding

Transgenic plants of *B. napus* expressing the bacterial catalase katE in the chloroplast could inhibit the growth of *E. cruciferarum*. These plants revealed constitutive expression of the catalase enzymes, PO, polyphenoloxidase, and high levels of free polyamines like putrescine, spermidine, and spermine (El-Awady et al. 2008).

Cultural Control

Choice of suitable planting dates according to the locale appears to offer a promising method of managing the disease. Irrigation scheduling only at the 50% branching stage could manage the disease (Hingole and Mayee 2003).

Biological Control

Some trends of efficacy of seed treatment and foliar sprays by *T. viride* and aqueous bulb extract of *A. sativum* against the disease have been reported by Meena et al. (2003, 2013), which may need confirmation.

Chemical Control

Should the disease become serious, it no doubt could be managed by dusting plants with sulfur. Karathane when sprayed thrice at 10-day intervals also gives good control of the disease (Kolte 1985). Foliar spray of carbendazim at 0.25% could be effective in controlling this disease (Kolte 2005). Dinocap or tridemorph (0.1%) could be sprayed thrice on leaves to reduce the disease severity significantly, which may also increase the yield of mustard (Shete et al. 2008). Spray of 0.04% tridemorph followed by 0.05% hexaconazole, 0.05% tebuconazole, and 0.20% wettable sulfur on leaves was found effective against this disease (Patel and Patel 2008).

BLACKLEG OR STEM CANKER

Symptoms

Severe infection of the pathogen can cause seedling death, but stem cankering may occur at any plant growth stage on any plant part. During infection, the pathogen grows systemically down toward the tap root of the plant. Blackleg disease causes two distinct types of symptoms, namely, leaf lesions and stem canker. Stem cankering is the major reason of yield loss associated with blackleg. Root rot symptom on oilseed rape has also been reported in Australia as an extension of the stem canker disease caused by *Leptosphaeria maculans*. It appeared before flowering and increased in severity during flowering and at maturity. Infection of *B. napus* roots by *L. maculans* can occur via invasion of cotyledons or leaves by airborne ascospores and directly by the entry of hyphae at sites of lateral root emergence in the soil. The pathogen grew within stem and hypocotyl tissue during the vegetative stages of plant growth and proliferated into the roots within xylem vessels at the onset of flowering. Hyphae grew in all tissues in the stem and hypocotyl but were restricted mainly to xylem tissue in the root (Sprague et al. 2007, 2009).

Symptoms appear first as water-soaked lesions on cotyledons, hypocotyls, and leaves of the host. These lesions turn white to gray color, round to irregular in shape, and become dotted with numerous pinhead-sized black asexual fruiting bodies called pycnidia. When in a mature state and under moist conditions, the pycnidia exude spores in pink ooze on the host. This disease can be distinguished from *A. brassicae* infection by the presence of pycnidia, which are not formed by the *A. brassicae* on *Brassica* crops. Black lesions are generally also seen on the leaves and deep brown lesions with a dark margin that can be seen on the base of stem (Marcroft and Bluett 2008). In severe epidemic conditions, the pathogenic fungus girdles the stem at the crown, leading to lodging and death of the plant. Typical lesions of blackleg can also occur on pods. Pod infection may lead to premature pod shatter and seed infection. The seed beneath pod lesions may be sunken or

shrivelled and pale gray in color. Li et al. (2008b) reported cytological changes, namely, condensation of cytoplasm, shrinkage in cell size, nuclear DNA fragmentation, shrinkage and condensation of the cytoplasm, chromatin fragmentation, and lobing of the nucleus due to hypersensitive reaction in cotyledon and stem tissues of *B. napus*, respectively, after infection by an avirulent strain of *L. maculans.*

Li et al. (2008c) reported that *L. maculans* may elicit apoptosis as a dependent component of pathogenesis in susceptible *B. napus*, and that the pathogen may use apoptotic cells as a source of nutrition for reproduction and further growth. Some proteins, namely, SOD, nitrate reductase, and carbonic anhydrase, were identified as being unique in the resistant plants, and upon pathogen challenge, some other proteins like photosynthetic enzymes (fructose bisphosphate aldolase, triose phosphate isomerase, sedoheptulose bisphosphatase), dehydroascorbate reductase, peroxiredoxin, malate dehydrogenase, glutamine synthetase, *N*-glyceraldehyde-2-phosphotransferase, and PPIase were observed to be increased in the resistant plants that were generated by an interspecific cross between the highly susceptible *B. napus* and the highly resistant *B. carinata* plants (Subramanian et al. 2005).

Geographical Distribution and Losses

Blackleg or stem canker is one of the major diseases of *Brassica* crops such as turnip rape (*B. rapa* L.), cabbage (*Brassica oleracea* L.), rapeseed (*B. napus* L.), and Indian mustard (*B. juncea* L.) grown in temperate regions of the world. For the first time it was reported on stems of red cabbage (Tode 1791). This disease is found in all continents and its world-wide importance and spread has been reported (Fitt et al. 2006). It has been reported as a serious disease also in Argentina (Gaetan 2005a), Australia (Lamey 1995), Brazil (Fernando et al. 2003), Canada (Lamey 1995), China (West et al. 2000), France (Lamey 1995), Greece (Vagelas et al. 2009), Latvia (Bankina et al. 2008), Lithuania (Brazauskiene et al. 2011, 2012), Poland (Kaczmarek et al. 2009a, Dawidziuk and Jedryczka 2011), the United Kingdom (Stonard et al. 2010a), and the United States (Mendoza et al. 2011). Yield losses up to 20% due to blackleg disease were recorded in Canada (Petrie 1978). Pedras et al. (1995) indicated crop losses due to blackleg in Canada alone exceed $30 million annually. It is the most important global disease of *B. napus* crops and causes annual yield losses of more than $900 million in Europe, North America, and Australia (West et al. 2001, Howlett 2004, Fitt et al. 2006). Both spring and winter types are affected by blackleg disease, particularly in Australia, Europe, and North America. Under epiphytotic conditions, this disease can cause yield losses of up to 90% (Kolte 1985, Sosnowski et al. 2004, Marcroft and Bluett 2008).

Pathogen

Blackleg or stem canker is caused by the heterothallic ascomycete fungus *L. maculans* (Desm.) Ces. et de Not. (anamorph: *Phoma lingam* Tode ex. Fr.). Another species, namely, *Leptosphaeria biglobosa* Shoem et Brun, has also been identified as causal agent of blackleg of canola in Australia (Wouw et al. 2008, Zhou et al. 2010), Canada (El-Hadrami et al. 2010), Lithuania (Brazauskiene et al. 2011, 2012), Poland (Kaczmarek et al. 2009, Dawidziuk and Jedryczka 2011), the United Kingdom (Stonard et al. 2010b), and the United States (Dilmaghani et al. 2009). Both species differ in their biochemical and molecular characteristics as well as in pathogenicity (Kaczmarek et al. 2009a), but are often found together in infected tissues of the same host (Dawidziuk et al. 2010). *L. maculans* can infect a wide variety of cruciferous crops, including cabbage, oilseed rape, and cruciferous weeds. Up to 28 crucifer species have been reported as hosts (Petrie 1969). *L. maculans* reproduces both sexually by forming pseudothecia and asexually by forming pycnidia on host species. The development of pseudothecia on stubble and the subsequent discharge of ascospores are greatly influenced by the genotype of the crop species (Marcroft et al. 2003). Pseudothecia

of the pathogen are black, immersed, globose with protruding ostioles, ranging 300–500 μm in diameter, and are normally found on woody plant tissues. Asci are cylindrical to clavate, sessile or short stipitate measuring 80–125 × 15–22 μm; the ascus wall is bitunicate. Ascospores are hyaline and spindle shaped when young but yellow tan and five septate at maturity measuring 30–70 μm × 4–9 μm (Boerema 1976, Sawatsky 1989).

Two types of pycnidia of *P. lingam* (anamorph phase), designated Group I and Group II, have been found on *Brassica* spp. As a parasite, the pathogen produces Group I pycnidia with a pseudosclerenchymatous wall structure, which are initially closed developing a papillate opening (sometimes with a neck), while as a saprophyte, the pathogen produces Group II pycnidia with a pseudoparenchymatous wall structure. Group II pycnidia have dark walls, are often irregular in shape and may or may not have a papilla. Hyaline, oval, single-celled pycnidiospores measuring 1–2 × 2.5–5 μm are exuded from the pycnidia (Boerema 1976). Under high-humidity conditions, ascospores and pycnidiospores adhere to cotyledons or young leaves and germinate to produce hyphae that penetrate through stomata and wounds (Chen and Howlett 1996, West et al. 2001, Hua et al. 2004) and grow into substomatal cavities without forming appressoria (Hammond et al. 1985). After entering into substomatal cavities, the fungus grows between the epidermis and palisade layer and then into intercellular spaces in the mesophyll of lamina. The pathogen then reaches the vascular strands of the petiole and grows systemically within the plant without forming symptoms. The pathogen moves down the petiole and into the stem where it eventually invades and kills the cells of the stem cortex more commonly at the crown and cause the stem canker symptom (Hammond et al. 1985, Sprague et al. 2007, Travadon et al. 2009).

L. maculans exists in two forms, namely, avirulent and virulent. The avirulent form usually infects plants near maturity and causes only superficial disease symptoms, which results in shallow stem lesions, and rarely forming extended cankers that girdle the stem, while the virulent form attacks the crop earlier and causes severe stem canker and economic yield losses. It is especially virulent on *B. napus*. If basal infection begins early, stem cankers appear from flowering onward. As the season progresses, cankers penetrate, deepen, and may girdle stem bases, often completely severing the plant.

A pathogenicity gene that encodes isocitrate lyase has been identified in *L. maculans*. Isocitrate lyase is a component of the glyoxylate cycle and is essential for the successful colonization of *B. napus* (Idnurm and Howlett 2002). Sexton et al. (2000) have cloned a gene encoding endo-PG, pg1, and two genes encoding cellulases, cel1 and cel2, in *L. maculans*. The sp1 and sp2 genes are also expressed in *L. maculans* during the infection of *B. napus* plants, and later the gene secretes a serine protease with protease activity (Wilson and Howlett 2005). The Lmpma 1 gene of *L. maculans* encodes a plasma membrane H+-ATPase isoform, which is essential for pathogenicity toward oilseed rape (Remy et al. 2008). Remy et al. (2009) reported that the Lmepi gene encodes a highly conserved UDP-glucose-4-epimerase enzyme of the Leloir pathway, which is involved in galactose metabolism, and indicated a link between this primary metabolism and pathogenicity in *L. maculans* toward oilseed rape.

A non-host-selective phytotoxin, Sirodesmin PL, which causes blackleg disease of canola, is produced by *L. maculans* (Elliott et al. 2007). The selective phytotoxin maculansin A has been isolated from *L. maculans*, which was more toxic to resistant (*B. juncea* cv. Cutlass) than the susceptible plants (*B. napus*). However, it did not elicit phytoalexin production either in resistant or in susceptible plants (Pedras and Yu 2008).

The pathogen induced production of chitinase in cotyledons of the host in a time-dependent manner. This enzyme started to accumulate before symptom appearance. The proteins, namely, antioxidant enzymes, photosynthetic, and metabolic enzymes, and those involved in protein processing and signaling were found to be significantly affected by the pathogen in the host. The enzymes specifically involved in the detoxification of free radicals increased in response to the pathogen in the tolerant *B. carinata*, whereas no such increase was observed in the susceptible *B. napus* (Sharma et al. 2008b). *Brassica* plants produce some phytoalexins, namely, brassinin and

camalexin, in response of *L. maculans*. Camalexin was found a substantially stronger inhibitor of the pathogen than brassinin (Pedras et al. 2007).

For the first time, variability for virulence in *L. maculans* was reported by Cunningham (1927). Pathogenic variability among *L. maculans* isolates of oilseed rape has been reported in southern Australia (Sosnowski et al. 2001) and western Canada (Kutcher et al. 2007). Australian populations of *L. maculans* have a high level of genetic variability as compared to European and North American isolates (Kutcher et al. 1993), along with a high diversity of avirulence genes (Balesdent et al. 2005). However, a low degree of genetic differentiation between isolates of *L. maculans* from seven sites in both eastern and western Australia has been observed by using AFLP marker (Barrins et al. 2004). Genetic diversity among different Australian, European, and North American isolates of *L. maculans* has also been studied by using AFLP marker (Purwantara et al. 2000). Molecular analyses of populations of *L. maculans* have shown high gene flow within and between populations. Pathogenic and genetic variation in *L. maculans* isolates on rapeseed–mustard has been reported in Australia, and microsatellite marker has been developed to study genetic variation in the Australian *L. maculans* population (Hayden et al. 2003). Isolates of *L. maculans* are usually classified either on the basis of their aggressiveness or by pathogenicity groups (Koch et al. 1991). Chen and Fernando (2006a) reported five pathogenicity groups (PG1, PG2, PG3, PG4, and PGT) of *L. maculans* on the basis of a series of inoculations on canola cultivars (*Westar*, *Glacier*, and *Quinta*) in western Canada and the United States. The PG3 and PG2 of *L. maculans* have also been reported on winter rape in Hungary (Szlavik et al. 2006) and Iran (Mirabadi et al. 2009), respectively. Nine races of *L. maculans* have been described and designated as AvrLm 1–9 (Mitrovic and Trkulja 2010).

Kenyon et al. (2004) developed a method for the detection of systemic growth of *L. maculans* in oilseed rape using quantitative RT-PCR. Sosnowski et al. (2006) used a quantitative PCR assay for the detection of *L. maculans* in soil. PCR-based molecular diagnostic techniques enabled detection, identification, and accurate quantification of airborne inoculum at the species level. Species-specific primers targeted at the ITS region of *L. maculans* and *L. biglobosa* were used to detect the quantity of the pathogen by traditional end point and quantitative RT-PCR methods, the latter being comparatively more sensitive, especially in years with low ascospore numbers (Kaczmarek et al. 2009b). The pathogen *L. maculans* on *B. napus* seeds was detected by using PCR-based techniques (Chen et al. 2010b, Yi et al. 2010). The ratio between airborne propagules of species, namely, *L. maculans* and *L. biglobosa*, of oilseed rape in Poland was evaluated by using a molecular approach based on species-specific primers and quantitative RT-PCR (Kaczmarek et al. 2009a).

Epidemiology and Disease Cycle

Infection of *B. napus* by *L. maculans* and subsequent development of leaf and stem lesions is influenced by cultivar resistance and weather conditions. Agronomic practices such as cultivar choice and fungicide use may also indirectly influence phoma stem canker epidemics at the regional level (Stonard et al. 2010b). Temperature and rainfall affect not only the development of pathogen but also the resistant response of the host (Fitt et al. 2008a). Elliott et al. (2011) for the first time isolated the *L. macunlans* and *L. biglobosa canadensis* isolates from *B. juncea* stuble in Australia. Dry climates lengthen the persistence of infected debris and may synchronize the release of airborne ascospores with seedling emergence; disease spread within plants is most rapid in regions with high temperatures from flowering to harvest (West et al. 2001). Kruse and Verreet (2005) reported that precipitation is of particular importance for *L. maculans* ascospore release during September in Germany. This effect was reduced in October and November while the influence of temperature increased. A very close correlation could be established between *L. maculans* ascospore release and leaf infection in autumn (September–November: $r = 0.82^{**}$). The correlation between the autumn infection of the leaves and root collar was highly significant (October–November: $r = 0.83^{**}$). Huang et al. (2006) reported that Rlm6-mediated resistance to *L. maculans* in *B. napus* leaves is affected by ambient temperature. High humidity and moderate temperatures during vegetative growth promote

disease development (Ghanbarnia et al. 2009). Under high RH condition, *L. biglobosa* can cause increase in disease, which may coincide with reduced accumulation of lignin at early stages of infection (El-Hadrami et al. 2010). According to Brazauskiene et al. (2007), the abundance of ascospores in the air depended on the weather factors, especially the amount and frequency of rainfall. During the daily period, the abundance of ascospore spread was influenced by the ambient RH. Magyar et al. (2006) found that high RH, rainfall, melting snow, and moderate wind act as the most important factors in the dissemination of the ascospores.

Dawidziuk et al. (2012) reported that higher winter temperatures may increase the ability of pseudothecia to release ascospores and the discharge of ascospores of the pathogen into the air, and cause early plant infections. This in turn will increase the number of infected plants, the DI at harvest, and reduce the yield of oilseed rape. Differences in climate, especially temperature, and cultural conditions may affect the proportions of *L. maculans* and *L. biglobosa* in stem base lesions of oilseed rape in the United Kingdom (Stonard et al. 2008). Lo-Pelzer et al. (2009) reported that it is possible to forecast the quantity of available primary inoculum for a given disease severity.

A regional preharvest forecast for stem canker incidence and a crop-specific risk assessment method that predict the onset of *Phoma* leaf spotting using postharvest weather data and thermal time relationships for canker development and canker severity, have been developed (Gladders et al. 2004). Evans et al. (2006) developed an empirical model to predict the date when incidence (percentage plants affected) of phoma leaf spot can be expected to reach 10% on oilseed rape and to guide timing of fungicide applications against this disease to prevent pathogen spread from leaf to stem and the subsequent development of damaging stem cankers. Two weather-based models (Improved Blackleg Sporacle and SporacleEzy) were developed to predict the first seasonal release of ascospores of *L. maculans* or *L. biglobosa* from oilseed rape debris under many climates and thus could contribute to the development of the strategies for the control of the disease (Salam et al. 2003, 2007). A forecasting system for a autumn application of fungicide against the important rape pathogen *L. maculans* has been developed (Bremer 2007). Ghanbarnia et al. (2009) developed a nonlinear model to evaluate the combined effect of total rainfall and average maximum temperature per week on the mean blackleg disease severity of canola. However, Fitt et al. (2008b) developed a model to describe the spread of *L. maculans* across Alberta Province, Canada and was used to estimate the potential spread of *L. maculans* across the oilseed rape growing areas of Yangtze River, China and its associated costs.

The System for Forecasting Disease Epidemics (SPEC) is a joint initiative of the Institute of Plant Genetics PAS and DuPont Poland for stem canker forecasting in the world. It monitors the concentration of ascospores of the *L. maculans–L. biglobosa* species complex as a tool for disease prevention against stem canker (blackleg), and it is addressed to oilseed rape farmers, associated farm service personnel, breeders, commercial company representatives as well as to students and researchers with an interest in plant pathology and plant protection (Jedryczka et al. 2004, 2006, 2008).

L. maculans has a very complicated life cycle. It survives as a saprophyte by forming mycelium, pycnidia, and pseudothecia on crop residues, mainly on stubble (Hall 1992) subsisting from one season to the next. The inoculum production of *L. maculans* decreases with the increasing burial duration in field soil over 10 months, before ceasing, which may be due to associated microbiota (Naseri et al. 2008). This pathogen has both a teleomorph (ascospores) and an anamorph (pycnidiospores) phases on host species and can complete several disease cycles during a single growing season. In Australia and Europe, the main sources of infection of seedlings are infected seed, and for mature plants are wind-dispersed ascospores that are produced within pseudothecia on crop residues during summer. Maturation of pseudothecia is greatly affected by wetness (Liu et al. 2007). Soil borne ascospores and pycnidiospores of *L. maculans* were also able to cause seedling death, even after the spores had remained in a plant growth medium for up to 21 days before sowing (Li et al. 2007c). Ascospores can travel up to 8 km in Australia (Bokor et al. 1975) and 1.5 km in the United Kingdom (Gladders and Musa 1980) and enter into the host through stomata to infect the plant. Soon after the

infection, they produce gray whitish lesions and black pycnidia on the leaves. During the growing season, these pycnidia produce conidia or pycnidiospores that are dispersed by rain splash. These spores cause a secondary infection, which is usually less severe than primary infection with ascospores. However, in western Canada and Poland, asexual pycnidiospores are the primary source of inoculum (Ghanbarnia et al. 2011). The pathogen overwinters as pseudothecia and mycelium in the stubble. In spring, the pseudothecia release their ascospores and the cycle repeats itself.

Krause et al. (2006) reported that the severity of *P. lingam* stem infection increased significantly with increasing number of oviposition punctures of *Ceutorhynchus napi*, which is one of the most destructive insect pests of winter oilseed rape in Central Europe. Females of *C. napi* deposit their eggs into the top of elongating stems that cause punctures in the stem and thought to predispose the stems to early secondary infections by *P. lingam* (*L. maculans*).

Disease Management

Host Plant Resistance

Two types of genetic resistance to *L. maculans* are usually identified in *Brassica*, that is, qualitative resistance (monogenic/race-specific/vertical resistance) that is expressed at the seedling stage and the quantitative one (polygenic/race-nonspecific/horizontal resistance) that is expressed in the adult plants. Qualitative resistance controlled by single major dominant gene has been reported in several spring and winter cultivars of *B. napus*, namely, Cresor, Maluka, Dunkeld, Maluka, Skipton, and Major (Stringam et al. 1992, Dion et al. 1995, Ferreira et al. 1995, Mayerhofer et al. 1997, Rimmer et al. 1999, Raman et al. 2012). Eighteen major genes for resistance to *L. maculans*, *Rlm1* to *Rlm11*, *RlmS*, *LepR1* to *LepR4*, *BLMR1,* and *BLMR2*, have been identified in *Brassica* species; *B. rapa*, *B. napus*, *B. juncea,* and *B. nigra* (Rimmer and van den Berg 1992, Balesdent et al. 2002, 2013, Yu et al. 2005, Delourme et al. 2006, Rimmer 2006, Van de Wouw et al. 2008, Yu et al. 2008a, Long et al. 2011, Raman et al. 2012). Six of them, *Rlm1*, *Rlm2*, *Rlm3*, *Rlm4*, *Rlm7,* and *Rlm9,* were identified in *B. napus*, all of them except *Rlm2* were clustered genetically on chromosome A07 (Delourme et al. 2004). *Rlm2* was mapped on chromosome A10 (Delourme et al. 2006). The *Rlm5* and *Rlm6* were identified in *B. juncea*, *Rlm8* and *Rlm11* in *B. rapa*, and *Rlm10* was identified in *B. nigra*. Four resistance genes, *LepR1*, *LepR2*, *LepR3*, and *LepR4,* were introgressed into *B. napus* from *B. rapa* subsp. *sylvestris.* Recently, two genes *BLMR1* and *BLMR2* were identified in Surpass 400, which is an Australian cultivar developed from an interspecific cross between wild *B. rapa* subsp. *sylvestris* and *Brassica oleracea* subsp. *alboglabra* (Buzza and Easton 2002, Long et al. 2011).

Christianson et al. (2006) reported that resistance to *L. maculans* in *B. juncea* populations is controlled by two independent genes, one of them being dominant and positioned on linkage group J13 and a recessive gene positioned on linkage group J18 based on segregation for resistance in the F2 population. In *B. rapa*, it is governed by three specific genes, namely, Rlm1, Rlm2, and Rlm7 (Leflon et al. 2007). Saal et al. (2004) identified a *B. juncea*–derived recessive gene termed rjlm2 that conferred resistance to *L. maculans* in oilseed rape. Gladders et al. (2006) reported that *B. napus* lines with Rlm6 resistance gene gave very effective control of leaf spot and stem canker caused by *L. maculans* in Europe, while Stachowiak et al. (2006) found both Rlm6 and Rlm7 resistant genes effective for the same. However, populations of *L. maculans* in Europe are known to have a high frequency of virulence to overcome resistance genes Rlm1–4 and Rlm9, and therefore, quantitative resistance makes an important contribution to stem canker control. Li et al. (2003c) reported the breakdown of a *B. rapa* subsp. *sylvestris* single dominant blackleg resistance gene in rape field of western Australia. Sprague et al. (2006) also reported that the *B. napus* cultivars derived from *B. rapa* ssp. *sylvestris* with single major gene resistance showed higher disease severity than cultivars with polygenic resistance in South Australia. Jedryczka et al. (2009) reported that Rlm6 and Rlm7 resistance genes for genetic protection of rapeseed against the present population of *L. maculans* in Poland.

Till date, nine resistance genes (Rlm1–9) have been identified in *Brassica* species (Gout et al. 2006). The corresponding nine avirulence genes designated as AvrLm1–9 have been identified in *L. maculans* (Balesdent et al. 2006), mapped at four independent loci, thereby revealing two clusters of three-and four-linked avirulence genes (Gout et al. 2006). The avirulence gene, AvrLepR1, of *L. maculans* corresponds to a resistance gene LepR1 of *B. napus*, and this plant gene control dominant, race-specific resistance to this pathogen (Ghanbarnia et al. 2012). Tollenaere et al. (2012) identified and characterized candidate Rlm4 blackleg resistance genes in *B. napus* by using next-generation sequencing technique. This major qualitative resistant locus (Rlm4) was mapped on chromosome A7 by using simple sequence repeat marker (Raman et al. 2012). *B. napus* cv. Surpass 400 was reported to have a single dominant resistant gene to *L. maculans* (Li and Cowling 2003), while Wouw et al. (2009) found at least two resistance genes, one of which is Rlm1 in *B. napus* cv. Surpass 400, with *sylvestris-derived* resistance. Two blackleg resistance genes, namely, LepR1 and LepR2, were mapped on N2 and N10 linkage groups of DHP95 and DHP96 lines of *B. napus*, respectively. The LepR1 generally conferred a higher level of cotyledon resistance than LepR2, because LepR1 prevented hyphal penetration, while LepR2 reduced hyphal growth and inhibited sporulation (Yu et al. 2005). The resistant gene LepR3 was found in *B. napus* cv. *Surpass 400* (Yu et al. 2008a). This gene provides race-specific resistance to the fungal pathogen *L. maculans*. LepR3 is the first functional *B. napus* disease resistance gene to be cloned and encodes a receptor-like protein. It has also been demonstrated that avirulence toward LepR3 is conferred by AvrLm1 avirulence gene, which is responsible for both the Rlm1- and LepR3-dependent resistance responses in *B. napus* (Larkan et al. 2013, Rouxel and Balesdent 2013).

The pathogen *L. maculans*, carrying AvrLm1 avirulence gene, when inoculated on *B. napus* plants carrying Rlm1 resistance gene, increased the biosynthesis of SA and ethylene (ET) and induced expression of the SA-associated genes ICS1, WRKY70, and PR-1, and ET-associated genes ASC2a, HEL, and CHI (Sasek et al. 2012a). Huang et al. (2009) found that quantitative resistance to *L. maculans* operates during colonization of *B. napus* stems by the pathogen.

Sinapis arvensis contains high resistance against various aggressive isolates of the blackleg fungus; so this species is valuable for the transfer of blackleg resistance to oilseed rape (*B. napus*) (Snowdon et al. 2000). *Brassica* species containing the B genome (i.e., winter *B. napus*, *B. nigra*, *B. juncea,* and *B. carinata*) are resistant to blackleg disease. Promising recombinant katanning early maturing (KEM) breeding lines derived from *B. napus* × *B. juncea* crosses were crossed with the spring-type *B. napus* cv. Dunkeld, which has useful polygenic resistance to blackleg. KEM recombinant lines showing regular meiotic behavior and a high level of blackleg resistance were screened using isolates of *L. maculans* having different AvrLm genes, which indicated *B. juncea* resistance gene Rlm6 had been introgressed into a *B. napus* spring-type cv. Dunkeld carrying polygenic resistance. The combination of both resistances would enhance the overall efficacy of resistance against *L. maculans* (Chevre et al. 2008).

Several *B. napus* and *B. juncea* germplasm from Australia, China, and India have been evaluated against Australian populations of *L. maculans*. *B. napus* genotypes from Australia were found more resistant than the Chinese and Indian genotypes (Li et al. 2008d). Two cultivars, namely, Aviso and Twister, of *B. napus* were found resistant to *L. maculans* in all seasons of the United Kingdom (Stonard et al. 2007). Light et al. (2011) reported that winter *B. napus* and *B. nigra* lines have outstanding potential for improving blackleg disease resistance under Australian conditions. Two blackleg-resistant lines, 16S and 61446, have been developed through interspecific hybridization between *B. napus* and *B. rapa* subsp. *sylvestris* and backcrossing to *B. napus* (Yu et al. 2013). In these lines, resistance to *L. maculans* is controlled by a single recessive gene (at LepR4 locus), and resistance alleles are allelic. Line 16S that carry LepR4a was found highly resistant, while line 61446 that carry LepR4b was found moderately resistant to stem canker under field conditions.

Molecular Breeding

Ananga et al. (2006) demonstrated that RAPD primers could be effectively used to identify DNA markers that are associated with blackleg disease resistance, which might also exist in the A and C genomes. Derivation of double haploid lines with superior levels of resistance to *L. maculans* compared with parental populations, and their multiyear, multisite (in locations with high pathogen diversity) evaluations could be an efficient practice to develop lines with high resistance to blackleg disease (Delourme et al. 2008). Dusabenyagasani and Fernando (2008) developed a sequence characterized amplified region marker available for marker-assisted selection in breeding canola for resistance against blackleg caused by PG3 of *L. maculans*.

Transgenic *B. napus* plants expressing pea DRR206 constitutively are resistant to the PG2 of *L. maculans* (Wang and Fristensky 2001). Kazan et al. (2002) reported that transgenic canola expressing MiAMP1 from the seeds of *Macadamia integrifolia* may be useful for the management of blackleg disease. A transgenic oilseed rape, *B. napus* cv. Hanna, with increased blackleg resistance has been developed by transferring Lm1 gene from *B. nigra* (Wretblad et al. 2003).

Induced Host Resistance

Resistance in canola can be induced by either pre- or coinoculation with the weakly aggressive isolates of the *L. biglobosa* and *L. maculans* (Chen and Fernando 2006b, Li et al. 2006c, El-Hadrami and Daayf 2009). After inoculation, the accumulated hydroxycinnamates act as precursors for the synthesis of lignin and phenylamide phytoalexins that could explain the restricted development of further inoculated highly aggressive isolates of the pathogen. Pretreatment of *B. napus* leaves with ascospores of *L. biglobosa* or chemical defense activators, namely, acibenzolar-*S*-methyl or menadione sodium bisulfite (MSB), delayed the appearance of *L. maculans Phoma* leaf spot lesions on the plants (Liu et al. 2006). MSB induced resistance locally and systemically (Borges et al. 2003). Treatment of *B. napus* plants with the SAR-inducing chemical benzo-(1,2,3)-thiadiazole-7-carbothioic acid *S*-methyl ester (BTH) significantly enhanced resistance against *L. maculans* (Potlakayala et al. 2007). Abscisic acid (Kaliff et al. 2007) and BABA (Sasek et al. 2012b) can induce callose- and SA-independent resistance, respectively, in *B. napus* against *L. maculans*. The gacS gene of *P. chlororaphis* was found to be responsible for antifungal and biocontrol activity against *L. maculans* of canola. Some low level of induced systemic resistance was observed in *P. chlororaphis* biocontrol of blackleg of canola (Ramarathnam et al. 2011).

Cultural Control

Various management practices such as crop rotation, careful stubble and residue management, time of sowing, use of certified seed, hot-water treatment of seeds, and control of volunteer cruciferous weeds have been recommended. A rotation including barley, field peas, and wheat for 3 years following oilseed rape helped eliminate potential sources of pathogen inoculum of *L. maculans* under all tillage systems (Turkington et al. 2000). The appropriate combination of rotation and tillage may lower airborne inoculum and reduce infection of rape by *L. maculans* (Guo et al. 2005, 2008), while Marcroft et al. (2003, 2004) reported that canola crops should be sown at distances greater than 100 m and preferably 500 m from last season's canola stubble, rather than extending rotation length between crops. Low seed rate and row spacing can increase the percentage of infestation of rape stem by *P. lingam* (Pusz 2007). Infested residue should be buried deep, and a shallow tillage or direct seeding method should be used in the spring to avoid bringing infected canola residue back to the surface. Since the primary infection of the plants occur by the airborne ascospores, canola should not be seeded within 1 km of infested land for 3–4 years (McGee and Emmett 1977). Soil borne ascospores and pycnidiospores can be managed by allowing the sand to dry between infestations and sowing by adding a 20 mm layer of uninfested sand over the top of the infested sand, respectively (Li et al. 2007c). The control of volunteer oilseed rape and susceptible cruciferous weeds should be done to prevent the establishment of the pathogen in fields. Wild mustard (*S. arvensis*) is highly susceptible to the virulent isolate of *L. maculans* (Petrie 1979).

Damage from blackleg could be minimized by sowing canola crops as early as possible before the onset of maturation of pseudothecia thus avoiding major ascospore showers at the seedling stage of maximum susceptibility and by doing fungicide protection in case of a late break season (Khangura and Barbetti 2004). Sowing of *B. napus* cultivars with different complements of resistance genes in subsequent years, that is, rotation of resistance genes minimizes disease pressure by manipulating fungal populations (Marcroft et al. 2012). Sprague et al. (2010) reported that defoliation of plants before stem elongation tended to develop less disease than defoliation during the reproductive phase of plant growth. In the future, this management strategy could be applicable in canola crops defoliated by grazing animals.

Biological Control

A bacterial isolate *Paenibacillus polymyxa* can inhibit the growth of *L. maculans* by producing antifungal peptides (Beatty and Jensen 2002). *Cyathus striatus* can reduce the production of the initial inoculum (pseudothecia) of the pathogen on rape stubble (Maksymiak and Hall 2000, 2002). A mixture of biological agents, namely, designated strain 17-1 (an associative endorhizosphere bacterial strain stimulating plant growth and protecting plants from pathogens) and 38-22, gave the highest increase in disease resistance and the best yield of spring rape (Farniev et al. 2009). Seed treatment with a commercial biofungicide of *Serratia plymuthica* reduced *L. maculans* activity by 50% in Germany (Marquardt and Ehlers 2010). Seed treatment with *S. plymuthica* and *P. chlororaphis* bioagents reduced mean disease by 71.6% and 54.0%, respectively, in canola (Abuamsha et al. 2011). More frequent treatments with commercial product, namely, Trifender WP of *Trichoderma asperellum* bioagent, during vegetation of oilseed rape could be effective against the blackleg disease (Kowalska and Remlein-Starosta 2011).

Chemical Control

Canola plants after 3–5 leaf growth stage are known to be less susceptible to blackleg than seedlings, so the protection of seedlings at that growth stage is an important method to manage this disease. Seed treatment with some fungicides, namely, thiram, fenpropimorph, benomyl, thiabendazole, and iprodione, has been found effective in managing the disease. However, these treatments do not protect plants grown in infested fields.

Ballinger et al. (1988) found that flutriafol applied as a fertilizer dressing on superphosphate granules significantly reduced the levels of stem canker in areas where the disease was prevalent. However, in western Canada, this fungicide had only limited efficacy (Xi et al. 1989). Dressing of canola seeds with fluquinconazole fungicides before sowing was found effective against *L. maculans* in situations of high disease severity, and grain yield increased when cultivars had lower blackleg resistance (Marcroft and Potter 2008). Chemical treatment of canola residues is a significant method to reduce the disease pressure on seedling. A number of chemical fungicides, such as fluquinconazole, flutriafol, and glufosinate ammonium (glufosinate), were able to delay pseudothecial development and decreased the subsequent ascospore discharge by more than 95% (Wherrett et al. 2003). However, impact (flutriafol) at 0.5 and 1 g/L, roundup (glyphosate) at 40 g/L, and copper sulfate inhibited the development of pseudothecia of *L. maculans* on canola residues and subsequently reduced ascospores production by 99% (Khangura 2004). Application of fungicides tended to be more beneficial at higher N rates and on upper slope positions since incidence was greatest under these conditions (Kutcher and Malhi 2004, Kutcher et al. 2005).

Pretreatment of host leaves with acibenzolar-*s*-methyl decreased the incidence of *Phoma* leaf lesions on seedling leaves (Liu et al. 2007). Azoxystrobin (Amistar 250 SC at 0.7 dm^3/ha) was found effective in decreasing blackleg infection of rape (Ratajkiewicz et al. 2009). Cytokinin, especially 6-benzyl amino purine, is able to significantly reduce disease symptoms and mycelial growth within plant tissues (Sharma et al. 2010c). CaraxReg is an innovative combination of 210 g/L mepiquat chloride and 30 g/L metconazole and is approved for the control of blackleg on oilseed rape (Gerber et al. 2010). Eckert et al. (2010) studied the effect of flusilazole, tebuconazole,

and methyl benzimidazole carbamate fungicides (benomyl and carbendazim) on the germination of ascospores, conidia, and germ-tube growth of *L. maculans* and *L. biglobosa*. Triazole-based fungicides, namely, metconazole, protioconazole, tebuconazole, and flusilazole, were most efficient, and mixture of protioconazole and tebuconazole or flusilazole and carbendazim were very active against the *L. maculans* on oilseed rape (Jedryczka and Kaczmzrek 2011). The most effective timings for the application of flusilazole + carbendazim were when leaves 7–11 were present on most plants and at least 10% of plants were affected by phoma leaf spot. Two half-dose applications of fungicide reduced *Phoma* stem canker and increased yield more than a single full dose application when *Phoma* leaf spot epidemics were early (Steed et al. 2007). Kaczmarek et al. (2009c) found that the application of fungicides at the time following the maximum ascospores concentration significantly reduced DI and caused the highest increase of yield. Early treatment of carbendazim + flusilazole fungicides was found more effective than the late treatment (Hood et al. 2007). Only one spray of flusilazole fungicide may result in the highest reduction of infected plants when it was done on the day of the highest ascospore release till no longer than 3 weeks afterward (Kaczmarek et al. 2011). Application of flusilazole fungicide on oilseed rape increased the glucobrassicin, protein content, and yield while decreased the total alkenyl glucosinolate content in seeds (Brachaczek et al. 2011).

DAMPING-OFF AND SEEDLING BLIGHT

Symptoms

A necrotic lesion 1–2 cm long may be seen at the base of the stem, with girdling sometimes taking place near the soil level. The taproot may be discolored and sometimes wire-stem symptoms may be seen. Salmon-colored spore masses of *Fusarium* are often observed on affected tissues. Sometimes, the symptoms are confined to roots consisting of light-brown lesions on the taproot and at the bases of larger lateral roots. Girdling of the main root may take place, which may lead to loss of the entire root system. Damping-off and seedling blight are mostly encountered due to the use of infested seed. Primary lesions consisting of small, circular necrotic spots, along with secondary lesions with large irregular borders, appear on leaves. Under high-moisture conditions, whitish hyphae appeared on the stems (Yang et al. 2004).

Geographical Distribution and Losses

Several species of fungi are involved in causing seed rot and seedling blight around the world. Among them, *Rhizopus stolonifer* is reported to be a more important cause. Postemergence mortality is not frequent, with *Pythium aphanidermatum*, *Pythium butleri*, *Rhizoctonia solani*, *Sclerotium rolfsii*, *Macrophomina phaseolina*, and *Fusarium* spp. being the pathogens involved in India, causing 6%–15% incidence (Kolte 1985, Khan and Kolte 2002). Bottom rot of *B. campestris* L. caused by *R. solani* has also been reported in Japan (Eimori et al. 2005). They mostly survive on crop debris and soil as different resting structures to infect the following crop.

Disease Management

Molecular Breeding

Transgenic *B. napus* plants expressing pea DRR206 were found resistant against biotrophic root pathogen *R. solani* (Wang and Fristensky 2001). Development of transgenics by the transfer from bean (*Phaseolus vulgaris*) cv. Goli of pgip2 gene, which encodes PGIPs, can be useful in the future (Akhgari et al. 2012).

Cultural Control

Drainage from the crop field should be ensured at the time of sowing the crop in order to avoid water stagnation. Clean cultivation and removal of crop debris before sowing are important to manage the problem.

Biological Control

Significant combined effect of *B. napus* green manuring as well as of *Trichoderma* seed treatment against different soil pathogenic fungi (*Pythium* and *Rhizoctonia*) could be useful (Galletti et al. 2006). The mutant strain of *T. viride* 1433, namely, Tvm6, can be used to control *P. aphanidermatum* pathogen of mustard (Khare et al. 2010). Root colonization by *P. fluorescens* can prevent the establishment of *R. solani* on the root system (Tehrani et al. 2007, Zanjani et al. 2011).

Chemical Control

Seed treatment with thiophanate methyl 70 WP at 2 g/kg ensured better plant stand with protection against *S. rolfsii*, *R. solani*, and *Fusarium oxysporum* (Khan and Kolte 2002). Seed treatment with Metalaxyl 35 SD 6 g/kg + carbendazim 1 g ai/kg or with any other suitable seed protectant fungicide may be helpful in increasing the stand of the crop. Application of glyphosate herbicide 10 days before seeding increased seedling emergence and seed yield of canola in field infested with *R. solani* (Rashid et al. 2013).

CLUBROOT

Symptoms

At the initial stages the affected plants show normal healthy growth, but as the disease develops, the plants become stunted showing pale green or yellowish leaves. The plant is then killed within a short time. When the plants are pulled, overgrowth (hypertrophy/hyperplasia) of the main and lateral roots (Figure 5.10) becomes visible in the form of small or spindle or spherical-shaped knobs, called clubs. Depending on the type of root of a species, the shape of the club varies. When many

FIGURE 5.10 Club root.

infections occur close together, the root system is transformed into various-shaped malformations. The swollen roots contain large numbers of resting spores and plasmodia. The older, more particularly the larger, clubbed roots disintegrate before the end of the season.

Geographical Distribution and Losses

Incidence and severity is greater in regions with severe winters than in regions with spring-type climates. It occurs more frequently in soils, which are acidic and poorly drained. More damage due to the disease results on vegetable crops such as cabbage (*B. oleracea* L.) and turnip (*B. rapa* var. *rapifera*) than on oilseed rape (*B. rapa* var. *oleracea*) and mustard (*B. juncea*). Woronin (1878) was the first to study the disease in a systematic manner, life cycle of the fungus, and its relation to host tissue in detail. Walker (1952) described the disease in detail on cabbage. On oilseeds *Brassica*, the disease is reported to occur in East Germany, Malaya, New Zealand, Poland, Sweden, the United Kingdom, and the United States (Kolte 1985). The disease has been reported from the hills of Darjeeling (Chattopadhyay and Sengupta 1952) and Nilgiri (Rajappan et al. 1999) in India on vegetable Brassicas. On *B. rapa* var. yellow sarson (Laha et al. 1985) and var. toria (Das et al. 1987), the disease has been reported from West Bengal and Orissa, respectively, with losses in yield being up to 50% (Chattopadhyay 1991). For the first time, this disease has also been reported on *E. sativa* in Brazil (Lima et al. 2004). This disease has also been reported on canola in Australia (Khangura and Wright 2012) and on rapeseed in Luxembourg (Desoignies et al. 2009). Internationally, this disease causes up to 50% yield loss and is considered a serious disease of rapeseed in France, Canada, Czechoslovakia, Sweden, the United Kingdom, and Germany (Donald and Porter 2003). In southern districts of New Zealand, losses due to clubroot on rape are reported high, and this factor has been the major cause of decline in crop acreage in that country (Lobb 1951). It has been reported to cause 10.2% yield loss on rape in China (Wang et al. 2008b) and 70%–90% in Canada (Pageau et al. 2006).

Pathogen

The pathogen is *Plasmodiophora brassicae* Woronin, which is an obligately biotrophic fungus. Biology of the pathogen has been reviewed (Kolte 1985). There is no evidence that pathotypes of *Plasmodiophora* exist with a single genus or group of related genera within the host family, Cruciferae. Hence, the taxonomic concept *formae speciales* has not been applied to *P. brasssicae*. There is also much morphological variation to justify the taxonomic division of the species on the basis of morphology. Genetic and pathogenic variability in the field isolates of *P. brassicae* has been reported by some workers (Xue et al. 2008, Strehlow et al. 2010). The fungus has a plasmodial vegetative stage characterized by a naked, amoeboid, multinucleate protoplast without a definite cell wall. The plasmodium is produced only in the cells of the host plant and remains intracellular, with two distinct phases. The first, the primary one, usually results from infection by primary zoospores derived from the resting spores, and the secondary one results from infection by secondary zoospores derived from a zoosporangium.

The resting spore is hyaline, spherical, and measures up to 4 μ in diameter. It germinates by giving rise to single biflagellate primary zoospores (the first motile stage) having one long and one short flagella. The zoospore swims by means of its flagella, the long flagellum trailing and short flagellum pointing forward. This zoospore penetrates the host root hairs, and there it develops into a primary plasmodium in the affected cell. The plasmodium formed in this manner later cleaves into multinucleate portions surrounded by separate membranes, and each portion develops into zoosporangia. The zoosporangia come out of the host tissue through pores formed in the host cell wall. About 4–8 biflagellate secondary zoospores are formed upon germination of a single zoosporangium. Each secondary zoospore, except for their small size, is indistinguishable from the primary zoospore. The exact role of the secondary zoospores is not known, but it is likely that the

secondary zoospores pair and unite to produce a zygote to cause fresh infection of the roots, producing new plasmodium called secondary or zoosporangial plasmodium, which in turn forms resting spores (Kolte 1985).

Though there are no *formae speciales* in *P. brassicae*, the fungus shows a lot of variation in pathogenicity. Physiologic specialization in *P. brassicae* was first demonstrated by Honig (1931). Information on the variation of the fungus has been reviewed, and a uniform set of differential hosts is described and proposed for research as an international approach for the identification of physiologic races of *P. brassicae*. Such a set of host genotypes is referred to as the European Clubroot Differential (ECD) set. The set consists of 15 different host varieties: 5 each of *B. rapa*, *B. napus*, and *B. oleracea*. Using the ECD set, 34 physiologic races have been identified in Europe (Kolte 1985).

The resting spores in soil serve as primary source of inoculum. Infection of the host takes place when uninucleate primary biflagellate zoospores are released on the germination of the resting spores. Germination of resting spores was most favored at 24°C, 2.6 pH, and 5 days of dark period (Wang et al. 2002). The zoospores may collide several times with a root hair before becoming attached; later, it appears to be attached at a point opposite to the origin of the flagella through adhesorium. The zoospores then encyst and penetrate the root hair or epidermal cells. The process of penetration of such cells appears to be direct but it has not been ascertained whether enzymes or toxins are involved in pathogenesis. After the entrance of the pathogen through root hairs, the formation of plasmodium and the subsequent development of zoosporangia take place in the infected tissue as described earlier. Then the zoospores derived from the zoosporangia are believed to reinfect the root and initiate formation of secondary plasmodia. Primary zoospores can directly cause secondary infection when the host is already in primary infection (Feng et al. 2013). Whether the secondary plasmodia penetrate the cell wall or if they are transformed passively from cell to cell during cell division is not certain. The plasmodium has no specialized feeding structure such as haustoria. It remains immersed in the host cytoplasm surrounded by a thin plasmodial envelope. There is also no evidence for phagocytic inclusion of the host cell organelles with the plasmodium. The plasmodium enlarges, and repeated nuclear division takes place, and the cells containing these become hypertrophied, although the host nucleus remains active. Hypertrophy of the host cells is apparently brought about by increased DNA synthesis and restriction of the cell division process. Presence of plasmodia in the host (*B. napus*) cell is associated with increased nuclei, at least in callus culture. Galling of susceptible *B. rapa* roots is the result of *P. brassicae* infection, enabling the enzyme glucosinolase to act on glucobrassicin, the indole glucosinolate. It is that the formation of the auxins, 3-indole acetonitrile and/or 3-indoleacetic acid, the characteristic extensive proliferation of tissue takes place. Since crucifers commonly contain indole glucosinolates, it has been suggested that this explains their susceptibility to galling. It appears that there is a close correlation between increase in the oxidative process and gall growth. As the galls develop on roots of the rape plant, the activity of glucose-6-phosphogluconate dehydrogenase, aldolase, triose phosphate isomerase, isocitrate dehydrogenase, and malate dehydrogenase increase is to reach a peak at 28–33 DAS. Then there is accumulation of glucose-6-phosphate, pyruvate, ketoglutarate, and malate in the affected cells. At sporulation, the activity of the aforementioned enzymes and concentration of the metabolites is decreased. During pathogenesis, these phenomena parallel the vegetative growth of the fungus. The metabolic regulation of phytoanticipins and phytoalexins has been found to be correlated with the infection period in the infected roots of oilseed canola (Pedras et al. 2008). Infectious pathogen spores can be detected by one-step PCR protocol, a quantitative/semiquantitative PCR-based technique in the soil and on the seed or tubers harvested from disease infested fields (Cao et al. 2007, Perek et al. 2010, Yin et al. 2010, Rennie et al. 2011).

Epidemiology and Disease Cycle

Development of the disease is favored by high soil moisture and cool weather; however, the disease can occur at any soil temperature between 9°C and 30°C. While development of clubroot was not observed at or below 17°C, it was slower above 26°C than at 23°C–26°C temperature (Gossen et al. 2012).

The fungus survives in the form of resting spores in soil. After the death of the galls, the resting spores are released in the soil; the pathogen thus becomes soil borne and is dispersed in soil as resting spores through farm implements, footwear, floodwater, etc. There is no evidence that the fungus lives as a saprophyte, yet soils are known to remain infested for 10 years or longer without the presence of a host. The pathogen can also survive on cruciferous weeds, namely, *C. bursa-pastoris*. Some of the noncruciferous hosts are also affected by *P. brassicae*. They are *Agrostis* spp., *Dactylis* sp., *Holcus* sp., *Lolium* spp., *Papaver* sp., and *Rumex* sp. Whether these noncruciferous plants play any part in maintaining the continuity of the disease in the absence of a cruciferous host is not known. However, it has been reported that secondary zoospores produced on *Lolium* spp. can infect canola (Feng et al. 2012). Wallenhammar (2010) studied the presence of clubroot in soil samples from 190 fields using a bioassay based on baiting the soils with *B. rapa* subsp. *pekinensis* (Chinese cabbage) *Granaat*. Clubroot incidence was significantly decreased after *Brassica* crops ceased to be grown. The half-life of spore inoculum was determined to 3.6 years for a field with 100% infestation. The level of infestation declined to below the detection level after a period of 17.3 years. Observations on yield loss from *P. brassicae* infections in spring oilseed rape (*B. napus* L.) are reported. In field tests of partly resistant cultivars of spring oilseed turnip (*B. rapa* L.), multiplication of clubroot was moderate.

Repeated cropping of susceptible host results in greater gall mass, reduced plant height, and increased numbers of resting spores in the soil mix compared to resistant host (Hwang et al. 2013). Increase in inoculum density, inoculation of young seedling could increase the disease severity and decrease the plant height and seed yield (Hwang et al. 2011a). Clubroot infection decreases abundance of adenosine kinase, which is involved in cytokinin homeostasis and also reduces host lignin biosynthesis. Enzymes level of ROS metabolism also declined sharply at 12 h after infection but increased at 24–72 h. These observations exhibit major changes in crop metabolism shortly after infection, which may result in the susceptibility of the host (Cao et al. 2008).

Disease Management

Host Plant Resistance

The control of the disease is difficult because of the longevity of resting spores in the soil. Among different methods, use of resistant varieties appears important to manage the disease. Certain kinds of *Brassica* spp. seem to have a natural resistance to the disease. Some genotypes of *B. juncea*, *B. rapa* var. toria, and *B. rapa* var. yellow sarson were found resistant in field condition (Sharma et al. 2012c). Deora et al. (2012) found 45H29 cultivar of canola resistant to disease. Hasan et al. (2012) observed pathotype-specific resistance in diploid species, namely, *B. rapa* (AA), *B. nigra* (BB), and *B. oleracea* (CC), and in the amphidiploid *B. napus* (AACC). Among *B. rapa* genotypes, turnip was most resistant, followed by winter- and spring-type oilseed rape. Contrastingly, rutabaga group of *B. napus* was observed homogeneous for resistance to Canadian *P. brassicae* pathotypes. The European winter canola (*B. napus*) cultivar Mendel has been used for the development of open-pollinated as well as hybrid canola cultivars (Rahman et al. 2011). It appears that resistance in *B. rapa* lines of a known genotype is associated with hypersensitive cortical cell death following invasion of *P. brassicae* from infected root hairs. Black mustard (*B. nigra* L.) is commonly reported as a resistant host due to volatile mustard oil, which remains to be proven (Kolte 1985). However, a positive correlation between clubroot susceptibility and clubroot-induced accumulation of several amino acids was found (Wagner et al. 2012). Sowing of a resistant variety could reduce the inoculum potential, while the cropping of susceptible variety increased the same (Hwang et al. 2011b, 2012a).

There are several physiologic races of *P. brassicae*, which vary in their ability to infect *Brassica* spp., and this complicates the problem of breeding-resistant varieties. Resistant varieties bred for clubroot resistance in one country may be completely susceptible to strains of the pathogens derived from another. Differential hosts used in ECD set are resistant to some races and susceptible to others. Development of resistant varieties through interspecific hybridization appears to be logical.

Resistance to *P. brassicae* Race 3 was successfully transferred from the turnip rape (*B. rapa*) variety Wasslander to rape (*B. napus*) variety Nevin by production of the fertile species, *Brassica napocampestris*, followed by two generations of back crossing of Nevin. Some cultures of *B. napus* (GSL-1, WBBN-1, WBBN-2, PCRS-80, WW-1507, ISN-700, MNS-3), *B. carinata* (HC-1, HC-4, HC-5, 9221, PC-3, PCC-2, PPSC-1, PC-5), and *B. nigra* (ACCBN-479) are reported resistant to the disease (Chattopadhyay et al. 2001).

Molecular Breeding

Suwabe et al. (2003) found that clubroot resistance in *B. rapa* is under oligogenic control, and at least two loci, that is, Crr1 and Crr2, are necessary for resistance. A resistance gene Crr1a has been identified in *B. rapa* L. (Hatakeyama et al. 2013). Yu et al. (2008b) reported that the resistant character in the resynthesized *B. napus* line HW243 is controlled by a single dominant gene for resistance to the disease. But durability of resistance seems unlikely to be profitable due to the development of newer pathotypes of *P. brassicae* keeping in view the faster rate of sexual reproduction in the pathogen. Wu et al. (2012) estimated the expression of stage-specific genes, namely, Pb-YPT, Pb-Brip9, and Pb-PSA, during infection of the pathogen in *B. rapa* by RT-PCR.

Development of transgenic lines by introducing thaumatin-like protein Hv-TLP8 from barley into oilseed rape via *Agrobacterium*-mediated transformation has been reported, which exhibits enhanced resistance to the pathogen (Reiss et al. 2009).

Cultural Control

Development of clubroot is significantly affected by cultivars, sowing date, soil moisture, and infection date (Wang et al. 2002). In Germany, pot experiments conducted (under field conditions) indicated that clubroot in yellow mustard can be reduced from 100% to 66% by mixing 50% compost into naturally infested soil. Early sowing can reduce infection compared to the late sowing of the crop (Hwang et al. 2012b). In view of the long viability of resting spores in soil, short-term crop rotation is not feasible, and traces of the pathogen could be detectable after more than 19 years of host plant absence, making its eradication very difficult (Rastas et al. 2012). Since *P. brassicae* also infects cruciferous weeds such as *C. bursa-pastoris*, it may be important to control the weeds in order to check the incidence of the disease. Use of 10–30 mg/kg boron and calcium nitrate in soil of pH 6.5 or 7.3 was effective in reducing clubroot severity (Ruaro et al. 2009).

Growing the crop in fields known to be infested with the clubroot pathogen should be avoided. General measures aimed at mitigating the incidence of the disease through improved drainage and application of lime brings about control of the disease. Spores of *P. brassicae* do not germinate or germinate very poorly in alkaline soils. On this basis, amendment of infested soil with lime is suggested. The amendment is done so as to raise the pH of the soil to 7.2. Treating the soil with lime 1 kg/m^2 area has been reported to control clubroot in mustard (AICRP-RM 2000). Application of worm cast as base fertilizer could effectively control the clubroot of rape, with an efficacy of 56.3%–61.4%, decrease the soil acidity, and increase the soil organic content (Wang et al. 2010b). Plant tolerance and resistance can be effectively increased against clubroot disease of mustard by using nutrients, namely, B, Mo, and Ca. These nutrients result in increase in yield and reduction in average weight of clubs per plant (Sen 2005, Deora et al. 2011). Nitrogenous fertilizers, namely, calcium ammonium nitrate and calcium nitrate, could be used to control the disease and increase shoot dry weight and seed yield (Bhattacharya and Mandal 2006).

Biological Control

Mixing or pouring of some antagonists, namely, *Serratia* spp. and *Trichoderma* spp., by using mushroom compost as a carrier for the antagonists was found effective in reduction of infection by up to 40% (Preiss et al. 2010). Lahlali et al. (2013) found that the biofungicide serenade (*B. subtilis*) suppresses the disease on canola via antibiosis and induced host resistance.

Chemical Control

Although certain chemicals like azoxystrobin, benomyl, fluazinam, flusulfamide, methyl thiophanate, quintozene, limestone, and other soil fumigants are known to be effective against *P. brassicae*, such disease management methods are not feasible and economical because of the high cost of chemicals and their application.

Fusarium WILT

Symptoms

The leaves of the affected plants show drooping, vein clearing, and chlorosis, followed by wilting, drying, resulting in the death of the plant. The symptoms progress from the base upward. The expression of the disease symptoms varies with the age of the plants. In the early stage of development, affected plants do not show all the typical symptoms. Plants affected in preflowering and early flowering stages show defoliation, and stems of such plants externally develop longitudinal ridges and furrows, which are generally not observed in the later stages. Diseased plants often show stunting, which is more pronounced when the plants are attacked in preflowering stages. Such plants have small pods with no seeds. Unilateral development of the disease is also observed in some of the cases when only one side of the plant shows symptoms of the disease. Roots of the diseased plants show no external abnormality or decay of the tissue until the plants are completely dried. Vascular tissues of stem and root show the presence of the mycelium and/or microconidia of the pathogen. Such tissues show browning of their walls and their plugging with a dark gummy substance, which is one of the characteristic symptoms of vascular wilts. At later stages of the disease, epidermis of roots sloughs off. The diseased plants eventually collapse and die (Kolte 1985).

Geographical Distribution

Mustard is affected by *Fusarium* wilt caused by *F. oxysporum* f. sp. *conglutinans* (Wr.) Snyder and Hansen. The first authentic report of *F. oxysporum* f. sp. *conglutinans* as the cause of the disease in *B. juncea* was made from India, followed by another on *B. nigra* (Kolte 1985). It has also been reported on canola in Argentina (Gaetan 2005b).

Pathogen

The causal fungal pathogen is *F. oxysporum* f. sp. *conglutinans*. Two types of cultures were isolated as Group A and B isolates and found that both were pathogenic to *B. rapa* var. toria, *B. rapa* var. yellow sarson, *Brassica oleracea* var. *botrytis*, *B. oleracea* var. *capitata*, *E. sativa*, *Matthiola incana*, *B. nigra*, *S. alba*, *Symphytum officinale*, and *R. sativus*. Susceptibility of *B. carinata*, *Crambe abyssinica*, and *C. hispanica* has been reported from the United States (Kolte 1985). Devi et al. (2009) reported *Fusarium moniliforme* as causal agent of *Fusarium* wilt of rapeseed from four districts of Manipur, India.

Disease Management

Extracts of plants *Vitex trifolia* and *Artemisia nilagirica* were found to have significant fungicidal properties (Devi et al. 2009). Seed treatment with carbendazim at 0.1% ai or a suitable biofungicide could be effective in managing the disease.

OTHER FUNGAL DISEASES

Rotting of seed is reported to be caused by *Nematospora sinecauda* (Oram et al. 2003), while white leaf spot caused by *Pseudocercosporella capsellae* has also been reported, when grayish white to brownish lesions on leaf (often with a distinct brown margin) and some grayish stem lesions occur (Eshraghi et al. 2005).

Bacterial Stalk Rot

Symptoms

Symptoms of the disease are characterized by the appearance of water-soaked lesions at the collar region of plants, which is usually accompanied by a white frothing. The tender branches are also affected as the lesions advance further to cover larger areas. The leaves show signs of water stress and wither. The affected stem and branches, particularly the pith tissues, become soft, pulpy, and produce dirty white ooze with a foul smell. The infected collar region becomes sunken and turns buff white to pale brown. Badly affected plants topple at the basal region within a few days.

Geographical Distribution and Losses

The first report about the occurrence of stalk rot caused by *Erwinia carotovora* (Jones) Holland appears to have been made in *B. juncea* in Rajasthan (India) by Bhowmik and Trivedi (1980). On an average, about 40%–60% of plants may be affected by the disease. Presence of the disease in fodder varieties of *Brassica* spp. is also observed. Vigorously growing succulent plants, due to an extra dose of N, as well as those growing in poorly drained soil are more severely affected. Root rot caused by *Erwinia carotovora* pv. *carotovora* (Jones) Bergy is an emerging threat for rapeseed–mustard production system, recently reported from the farmers' field in some pockets of India (AICRP-RM 2006–2008, Meena et al. 2010b).

Pathogen

The bacterium is Gram negative, rod shaped with blunt ends, capsulated, and motile with peritrichous flagella. It forms grayish, circular, translucent, shining, smooth colonies on nutrient agar with a raised centre and wavy margin. The bacterium can infect *B. oleracea* var. *botrytis*, *Daucus carota*, *Lycopersicon esculentum*, and *Nicotiana tabacum*.

Epidemiology and Disease Cycle

The disease is favored by warm and humid weather. It usually appears after first irrigation in mustard. The pathogen survives on diseased plant debris in soil.

Disease Management

Bacterial stalk rot can be managed to some extent by using cultural practices, namely, crop rotation with nonhost crops, deep ploughing in summer months, and roguing and burning of diseased debris. These practices help in minimizing inoculum buildup in soil. Early sowing of crops, removal of weeds, and avoidance of overirrigation are effective in reducing the DI. This disease can be reduced by spraying streptocycline 100 ppm and copper oxychloride at 0.2%.

Bacterial Rot

Symptoms

Symptoms appear when the plants are 2 months old. In the initial stages, dark streaks of varying length are observed either near the base of the stems or 8–10 cm above the ground level. These streaks gradually enlarge and girdle the stem. Finally, the diseased stem becomes very

soft and hollow due to severe internal rotting, and this often results in total collapse of the plant. Sometimes, cracking of the stem is observed before the toppling of the plant. Occasionally, symptoms appear on leaves. Lower leaves show the symptoms first, which include midrib cracking and browning of the veins; when extensive, it brings about withering of the leaves. Profuse exudation of yellowish fluid from affected stems and leaves may also occur. Blackened veins and V-shaped necrotic lesions on the leaf margins are surrounded by yellow halos. The advanced phases of the disease include lesion enlargement, foliar chlorosis, and death of leaves. The disease develops from the lower leaves to the apex, resulting in complete leaf necrosis and defoliation. The affected plants, on stripping, show a dark brown crust full of bacterial ooze. The black rot does not cause any disagreeable odor.

Geographical Distribution and Losses

Patel et al. (1949) first observed the black rot symptoms in *B. juncea* in India under natural conditions. In 1970, 100% incidence on cauliflower was reported, and during 1969, National Seed Corporation of India suffered heavy losses of about 0.5 million rupees in 10 ha of cauliflower seed crop due to combined infection of stump rot and black rot. The disease is now reported to occur in a severe form (60% incidence) in the Indian State of Haryana (Kolte 1985). Occurrence of the disease has also been reported in Brazil, Canada, Germany, Serbia (Popovic et al. 2013), Mozambique (Bila et al. 2013), Sweden, and the United States. In fact, monoculture is presently the dominant form of crop management worldwide, which plays a major role in disease progression (Zhu et al. 2000).

Pathogen

The pathogen is *Xanthomonas campestris* pv. *campestris* (Pammel) Dowson. The bacterium is a short rod with rounded ends, occurring singly, rarely in pairs. In culture on potato dextrose agar, it measures 1.5 μ (1.2–2.1 μ) × 0.7 μ (0.5–1.0 μ). It is motile with a single polar flagellum, Gram negative, not acid fast, aerobic, and capsulated without spore formation. On nutrient dextrose agar, colony is dark yellow, circular, nonfluidic, convex, and opaque. The thermal death point is 50°C–58°C. Genetic and pathogenic variability among the isolates of *X. campestris* pv. *campestris* has been identified by several workers (Gaetan and Lopez 2005, Miguel-Wruck et al. 2010, Singh et al. 2011b, Raghavendra et al. 2013). A DNA probe has been developed for rapid identification of strain of this pathogen in plant tissues (Shih et al. 2000). This pathogen can be specifically and rapidly detected by several methods, namely, multiplex polymerase chain reaction (Berg et al. 2005, Leu et al. 2010), multiplex RT-PCR assay (Berg et al. 2006), Bio-PCR (Singh and Dhar 2011), classical biochemical assays, enzyme-linked immunosorbent assay (ELISA) with monoclonal antibodies, Biolog identification system, and PCR with specific primers and pathogenicity tests (Bila et al. 2013).

Epidemiology and Disease Cycle

The host range includes *B. alba*, *B. rapa* var. brown sarson, *B. rapa* var. yellow sarson, *B. carinata*, *B. chinensis*, *B. hirta*, *B. napus*, *B. nigra*, *B. oleracea*, *B. rapa*, *B. tourneforti*, and *R. sativus*. The pathogen does not infect *E. sativa* and *C. sativa* (Kolte 1985). Details of the mode of penetration and the infection process have not been studied using rapeseed–mustard plants. However, it is believed that the pathogen overwinters in diseased plant refuse or in seed and penetrates the host through either stomata or hydathodes and establishes the infection in a similar manner as in other crucifers (Berg et al. 2005).

Disease Management

Host Plant Resistance

Race-specific resistance to the pathogen has been found in Brassicas with B and D genomes (Ignatov et al. 2001). Griffiths and Nickels (2001) reported that a single dominant gene may control the resistance. In progeny of *B. carinata*, the resistance is conferred by a major dominant gene Rb that can

be used for breeding purposes (Ignatov et al. 2001). Some new alien addition lines resistant to black rot have been generated by somatic hybridization between cauliflower and black mustard (*B. nigra*) (Wang et al. 2011). Some degree of resistance to races 1 and 4 of *X. campestris* pv. *campestris* in different *B. napus* crops, mainly in underexplored pabularia group, has been identified (Lema et al. 2011). Resistance was identified in five accessions of *B. carinata* (PI 193460, PI 193959, PI 194254, PI 280230, PI 633077) and four accessions of *B. nigra* (PI 197401, A 25399, A 25401, PI 458981) determined by repeated symptomless responses after inoculation. Five accessions of *B. rapa* (PI 633154, A9285, PI 340208, PI 597831, PI 173847) represent promising new sources of resistance to the pathogen. Incomplete resistance was identified in an accession of *E. sativa* (PI 633207), *Lepidium* spp. (PI 633265), *S. arvensis* (PI 296079), and two accessions of *B. napus* (PI 469733 and PI 469828). These identified accessions represent germplasm that can be used in breeding for resistance to *Xcc* in the future (Griffiths et al. 2009).

Biological Control

Foliar spray or the combined seed soaking and soil drenching with *Pseudomonas aeruginosa* (KA19 strain) and *Bacillus thuringiensis* (SE strain) are also found effective in reducing black rot lesions compared to untreated control (Mishra and Arora 2012).

Chemical Control

Captafol spray (0.2% ai) at 20-day intervals is reported to give good control of the disease; aureomycin (chlorotetracycline) 200 µg/mL was most effective in reducing the infection from 85% to about 15% resulting in an increase in yield by 60%. Among fungicides, carboxin was most effective, reducing the infection by 79% with a corresponding increase in yield by 49%. Spray application of copper oxychloride is also reported to give a considerable degree of control of the disease (Kolte 1985).

OTHER BACTERIAL DISEASES

Bacterial leaf spots are also reported to be caused by *Pseudomonas viridiflava*, which result in white and corky brown spots on leaves and sometimes water-soaked spots on the lower leaf surface (Myung et al. 2010). Brownish-black color leaf spots are also reported to be triggered by *Pseudomonas syringae* pv. *maculicola* (Peters et al. 2004) and *Pseudomonas cannabina* pv. *alisalensis* (Bull and Rubio 2011).

Mosaics

Symptoms

Symptoms on *B. juncea* appear as vein clearing, green vein banding, mottling, and severe puckering of the leaves. The affected plants remain stunted and do not produce flowers, or very few flowers are produced on such plants. When siliquae are formed, they remain poorly filled and show shrivelling, which results in decrease in yield and oil content (Jasnic and Bagi 2007). Symptoms of vein clearing, stunting, and pod malformation have been observed (Sahandi et al. 2004). According to Sahandi et al. (2004), the symptoms appear as systemic conspicuous vein clearing, vein banding, yellowing, and distortion of young leaves. During the later stages of infection, numerous raised or nonraised dark green islands of irregular outline appear in the chlorotic area between the veins, giving rise to a mottled appearance. Curvature of the midrib and distortion of the leaf blade on affected leaves can also be a prominent symptom. Plants infected early are usually stunted and killed, but those infected late show reduced growth only slightly (Kolte 1985). The number of primary branches, seeds per pod, and percentage oil per seed is reduced; the glucosinolate concentration in the oil is also significantly increased in infected plants (Stevens et al. 2008).

Geographical Distribution and Losses

Occurence of the virus disease has been reported on different cruciferous plants in Iran (Shahraeen et al. 2002, Tabarestani et al. 2010), New Zealand (Kolte 1985), Taiwan (Chen et al. 2000), on winter oilseed rape in Austria (Graichen et al. 2000), and on *B. nigra* (Thurston et al. 2001) and *B. rapa* ssp. *sylvestris* (Pallett et al. 2002) in the United Kingdom. Biswas and Chowdhury (2005) reported the disease in *B. juncea* and *R. sativus* in the Himalayan regions of West Bengal, India, and indicated that it may be a strain of turnip yellow virus (TuYV).

Yield could decrease by more than 70% (Jasnic and Bagi 2007). Over 30% of the crop has been reported to be destroyed by the disease in China resulting in 37%–90% loss in yield (Kolte 1985). In Australia, yield loss due to TuYV on oilseed rape may reach up to 46% (Stevens et al. 2008). In Iran, canola field infection with turnip mosaic virus (TuMV), CaMV, and beet western yellows virus (BWYV) was 1.7%–8.3% (Tabarestani et al. 2010).

Pathogen

Some of the more common crucifer mosaic diseases are caused by viruses included in turnip virus I group. On rapeseed–mustard, the mosaic diseases caused by this virus group are described under different names, namely, (1) rape mosaic in China and Canada, (2) mustard mosaic in the United States and Trinidad, (3) Chinese sarson mosaic in India, (4) *B. nigra* virus in the United States, and (5) turnip mosaic in China, Germany, Hungary, Soviet Union, and the United Kingdom (Kolte 1985).

Occurence of six viruses, namely, BWYV, CaMV, turnip crinkle virus, TuMV, turnip rosette virus, and turnip yellow mosaic virus (TYMV), were detected on *B. nigra* (Thurston et al. 2001) and *B. rapa* ssp. *sylvestris* (Pallett et al. 2002) in the United Kingdom. TuMV, CaMV, and BWYV are considered as the most important viruses of canola in Iran (Tabarestani et al. 2010) and of rape (including TYMV) in Europe (Mamula 2008). Cai et al. (2009) found two new strains, youcai mosaic virus–Br and oilseed rape mosaic virus (ORMV)-Wh, which are related to the ORMV cluster of tobamoviruses and distantly to tobacco mosaic virus as a pathogen of oilseed rape in China. The pathogen can be detected by tissue blot immunoassay (Coutts and Jones 2000), ELISA (Thurston et al. 2001, Pallett et al. 2002), double-antibody sandwich-ELISA (Chen et al. 2000, Graichen et al. 2000, Sahandi et al. 2004), antibody sandwich-ELISA and PCR (Farzadfar et al. 2005), and RT-PCR technique (Tabarestani et al. 2010). Polyclonal antiserum has been produced against CaMV isolate that can also be used in indirect ELISA system for virus survey and identification (Sahandi et al. 2004). Graichen et al. (2000) detected no TuYV infection in virus-like symptoms having plants and infection in symptomless plants, which highlights the necessity of serological testing of plant samples for the determination of virus infection in oilseed rape.

Epidemiology and Disease Cycle

Disease intensity varies among years and depends on weather conditions during season, source of inoculum, and vector population (Jasnic and Bagi 2007). Coutts and Jones (2000) found wild radish (*R. raphanistrum*) as a substantial virus reservoir in canola field in Southwest Australia. If TuMV and CaMV viruses are transferred from *S. arvensis* to oilseed rape with aphid, then wild mustard could be a reservoir of these virus infections for oilseed rape under natural condition (Dikova 2008).

TuYV can be transmitted by *Myzus persicae* (act as main vector), *Brachycorynella asparagi*, *Cavariella aegopodii*, *Macrosiphoniella sanborni*, *Macrosiphum albifrons*, *Myzus nicotianae*, *Nasonovia ribisnigri*, *Pentatrichopus fragaefolii*, *Rhopalosiphum maidis*, *Acyrthosiphon pisum* (green race), *Aphis gossypii*, *Aulacorthum circumflexum*, *Aulacorthum solani*, *Brevicoryne brassicae*, *Rhopalosiphum padi*, and *Sitobion avenae* (Schliephake et al. 2000). Green peach aphid (*M. persicae*), turnip or mustard aphid (*Lipaphis erysimi*), cabbage aphid (*B. brassicae*) and cowpea aphid (*Aphis craccivora*) may serve as vector of BWYV, TuMV, and CaMV on rape and Indian mustard in New South Wales, Australia (Hertel et al. 2004). Stevens et al. (2008) found peach-potato aphid, *M. persicae*, as vector of TuYV and reported 72% of winged *M. persicae* carry this virus on oilseed rape in Australia. He also reported that milder autumn and winter conditions favor

the development of the aphid vectors and encourage virus spread. Green peach aphid requires minimum 0.5–1.0 h for acquisition and inoculation process for BWYV transmission on mustard. This transmission is influenced by temperature, and the highest transmission rate could be obtained at 20°C–25°C. After virus acquisition, aphid could retain BWYV for at least 2 weeks. The virus could not pass onto the progeny of its vector. BWYV replicated well in mustard at 15°C–25°C (Chen 2003).

Maling et al. (2010) modified a previously developed hybrid mechanistic/statistical model, which was used to predict vector activity and epidemics of vector-borne viruses, to simulate virus epidemics in the BWYV-*B. napus* pathosystem in a Mediterranean-type environment.

Disease Management

Coutts et al. (2010) found that *B. napus*, which has some resistance to BWYV, can be used in conjunction with imidacloprid seed dressings as component of an integrated pest management strategy to manage BWYV in *B. napus* crops. The genetically engineered cross protection of *Brassica* crops with weak strain Bari-1 Gene VI of CaMV and its genetic regularity have been studied by Gong et al. (2001). Lehmann et al. (2003) were able to induce coat protein–mediated resistance to TuMV in *B. napus*. Control measures include the elimination of inoculum source, isolation from areas, which contain inoculum, aphid control, and growing virus resistant or tolerant oilseed rape genotypes (Jasnic and Bagi 2007). Seed dressing with imidacloprid in sufficient amount (525 g ai/100 kg of seed) before sowing is a good prospect for the control of BWYV and *M. persicae* in *B. napus* crops (Jones et al. 2007). Seed treatment of oilseed rape with thiamethoxam is an excellent alternative to insecticide spray for controlling TuYV transmission by *M. persicae* (Dewar et al. 2011).

Phyllody and Aster Yellows

Symptoms

The characteristic symptom is the transformation of floral parts into leafy structures. The corolla becomes green and sepaloid. The stamens turn green and become indehiscent. The gynoecium is borne on a distinct gynophore and produces no ovules in the ovary. In addition, there are some leafy structures attached to the false septum. The affected plants may show varying degrees of severity of the disease, and the affected part of the raceme does not form siliquae. Some plants may show only terminal portion of the branches affected with the disease, whereas in others, the whole branches show the symptoms.

Geographical Distribution and Losses

Under natural conditions, the phyllody has been reported to occur on oilseed rape (*B. napus*) in Greece (Maliogka et al. 2009), Italy (Rampin et al. 2010), Poland (Zwolinska et al. 2011), Iran (Salehi et al. 2011), Canada on *B. rapa* (Olivier et al. 2006), India on toria (*B. rapa* var. toria), and yellow sarson (*B. rapa* var. yellow sarson) in the states of Punjab, Haryana, New Delhi, and Uttar Pradesh (Kolte 1985, Azadvar and Baranwal 2010) in India. In Canada, aster yellows (AY) has also been reported on *B. napus* and *B. rapa* (Olivier et al. 2010). Yield loss may go up to 90% (Kolte 1985).

Pathogen

The phyllody disease is reported to be caused by the *Candidatus Phytoplasma asteris* (*Phytoplasma asteris*) phytoplasma of subgroup 16Srl-A, 16Srl-B, and 16SrlX-C (Olivier et al. 2006, Maliogka et al. 2009, Azadvar and Baranwal 2010, Rampin et al. 2010, Zwolinska et al. 2011). The AY disease is reported to be caused by the phytoplasma of 16Srl-A and 16Srl-B subgroup (Olivier et al. 2010). Phytoplasma are nonhelical, mycoplasma-like bacteria that lack cell walls. They almost exclusively inhabit the phloem sieve-tube elements of the infected plant and is transmitted from plant to plant

by phloem-feeding homopteran insects mainly plant hoppers (*Laodelphax striatellus*) (Azadvar and Baranwal 2010) or leafhoppers (*Circulifer haematoceps*) (Salehi et al. 2011), and less frequently psyllids and Cicadellidae (Jajor 2007). Seed transmission of phytoplasma in winter oilseed rape has also been reported (Calari et al. 2011). This pathogen can be detected by several methods, namely, nested PCR (Olivier et al. 2006, Azadvar et al. 2011, Calari et al. 2011), PCR, and RFLP (Wang and Hiruki 2001, Zwolinska et al. 2011). Sequencing and phylogenetic analysis of 16S rRNA, a part of 23S rRNA, partial sec A genes, rp gene and 16S–23S intergenic spacer region, and RFLP pattern of 1.25 kb 16S rDNA sequences of the pathogen has also been done (Azadvar and Baranwal 2010).

Epidemiology

Early planting of toria in late August or at its normal planting time in September has been shown to favor the development of the disease in toria under Indian conditions. As high as 24% incidence of the disease, depending on the variety, can be seen in plants sown in August (Kolte 1985). The disease development is favored by prolonged dry and warm weather. The pathogen survives on alternate hosts like sesame, which serves as primary source of infection. The disease is transmitted through leafhopper and seeds. The disease spreads by repeated cycles of secondary infection through the process of transmission.

Disease Management

Weed management in and around fields that serve as hosts for the pathogen and increasing planting population may reduce the incidence of phyllody. Roguing and destruction of the infected plants will reduce further spread of disease. The population of insect vectors should be controlled by using appropriate insecticides when they are at their peak. Two sprays of dimethoate or metasystox at 0.1% at an interval of 15 days starting from the initiation of symptoms should be done to manage the insect vectors.

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Section IV

Sunflower

The cultivated sunflower (*Helianthus annuus* L. var *macrocarpus* (DC) Ck II) belongs to the Compositae (an Asteraceae) family. It is a sparingly branched annual herb about 1.0–3.0 m in height. It is insensitive to photoperiods. The basic chromosome number of *H. annuus* is 20 pairs ($2n = 40$). Sunflower has a large genome (3600 Mbp) with abundant repetitive sequences (Baack et al. 2005, Kane et al. 2011).

Sunflowers are referred to as *composites* because what looks like a single large sunflower head is actually an inflorescence composed of a composite of many tiny, usually 1000–2000, individual flowers joined to a common base called the receptacle. The flowers around the circumference are lingulate ray florets with neither stamens nor pistil. The fertile disk florets are located within the head. Each disk floret is a perfect flower. The flowering behavior facilitates cross-pollination, and insects, particularly the bees, represent the essential vector of sunflower pollen. The degree of cross-pollination may be to the extent of 100%. The sunflower seed is a specific type of elongated rhomboid indehiscent achene, which may be white, black, or striped gray and black. The oil content is more than 40%. The oil is characterized by a high concentration of linoleic acid and a moderate level of oleic acid. While the sunflower seed could still be harvested for edible oil, the woody stalk could be used as a biofuel. By producing food and biomass, such a crop would be both economically and politically viable.

The center of origin of the sunflower is believed to be North America from where it has spread to Europe and Asia. Now it is grown in all continents except the Antarctica. Europe and America account for nearly 70% and 80% of the total production, respectively (Harter et al. 2004, Damodaran and Hegde 2007). Sunflower cultivation in Asian countries is comparatively recent. Asia accounts for nearly 20%–22% of the global sunflower and contributes to about 18% of the production. The productivity of sunflower in Asia is about 1.0 ton/ha, which is lower than the world average. India is the largest grower of sunflower in the Asian continent. This is a short-duration crop that is adaptable to a wide range of agroclimatic situations, having high yield potential, suitable for cultivation in all seasons due to its day neutral nature and can fit well in various intercropping and sequence cropping systems. However, the average yield of this crop in India is lowest; it is less than half the world average and static hovering around 0.5–0.6 ton/ha.

The crop performs well and yields more oil in temperate zones. It grows well in a well-drained soil, ranging in texture from sandy to clay. The emergence of new diseases and large climatic variations, particularly recurrence of drought stress during critical growth stages, has affected stability and yield on a regular basis. With continuous cropping in the same field area, the crops suffer from diseases resulting in large losses in yield. Sunflower diseases are described in the following chapter.

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6 Sunflower Diseases

RUST

Symptoms

Symptoms of sunflower rust appear on all the aboveground plant parts but are more prevalent on leaves. Small, orange to yellowish spots appear in compact circular groups followed by brownish, circular to elongated, and pulverulent uredinia scattered over the upper and lower surfaces of the leaf. Uredial pustules usually appear first on the lower leaves. They are small, circular, 0.5–1.00 mm in diameter, powdery, orange to black in color, and usually surrounded by chlorotic areas (Figure 6.1). The uredia may coalesce to occupy large areas on the affected plant parts. Usually late in the crop season, as the plant approaches maturity or is subjected to physiological stress, teliospores appear in the uredia and develop into telia on the affected senescent tissues, and the black rust stage appears.

In the case of highly resistant varieties, no uredia are produced, and only small chlorotic or necrotic flecks develop at the point of infection.

Geographical Distribution and Losses

The first report of sunflower rust described by Lewis von Schweinitz from the southeastern United States dates from 1822 (Sackston 1981). The disease now occurs in virtually all the sunflower-growing areas of the world and is more common in temperate and subtropical regions (Kolte 1985). It is, however, considered an important disease of sunflower in Argentina (Gutierrez et al. 2012), Australia (Sendall et al. 2006), Canada (Rashid 2004, Gulya and Markell 2009), Cuba (Perez et al. 2002), India (Mayee 1995, Amaresh and Nargund 2002a), Israel (Shtienberg and Johar 1992), Pakistan (Mukhtar 2009), Russia, South Africa (Los et al. 1995), Turkey (Tan 1994, 2010), and the United States (Harveson 2010, Friskop et al. 2011). It has also been recorded in almost all European and adjacent Mediterranean countries engaged in sunflower production (Sackston 1978).

Severe infection can decrease head size, seed size, oil content, and yield. On an average, in North American conditions, yield losses ranging from 25% to 50% have been reported in areas of intensive sunflower cultivation. Loss estimates are based on field observations or yield comparisons of resistant and susceptible varieties in performance trials (Zimmer et al. 1973). Still further, the severity of sunflower rust in the states of Manitoba in Canada in 2003 (Rashid 2004) and in Nebraska, North Dakota, South Dakota, and Minnesota in the United States has increased steadily from 17% to 77% through the 1990s and 2000s, and dramatic yield reductions have been recorded in localized *hot spots* (Gulya et al.1990a, Gulya and Markell 2009, Harveson 2010, Friskop et al. 2011).

Quantitative assessment of the effect of rust on yield of sunflower in Australia has shown reduction up to 76% (Middleton and Obst 1972, Brown et al. 1974). Siddiqui and Brown (1977) from Australia reported that oil yield losses in sunflower are generally influenced by the growth stages of plant when infection occurs and by the degree of intensity of infection. The effect of rust on growth parameters varies according to the moisture stress to which the plant is subjected. In Kenya, the disease caused 60% yield losses in severe cases; consequently, sunflower acreage dropped dramatically from 24,280 ha in 1949 to 1,420 ha in 1952 (Singh 1974).

The quality of seed is also adversely affected by reduction in test weight and oil content and by increased hull to kernel ratio (Middleton and Obst 1972).

FIGURE 6.1 Sunflower rust. Note the minute uredopustules on the leaf. (Courtesy of Dr. Chander Rao and Dr. Varaprasad, DOR, Hyderabad, India.)

Pathogen: *Puccinia helianthi* Schw.

Classification
Kingdom: Fungi
Phylum: Basidiomycota
Class: Urediniomycetes
Subclass: Incertae sedis
Order: Uredinales
Family: Pucciniaceae
Genus: *Puccinia*
Species: *helianthi*
Binomial name: *Puccinia helianthi* Schwein.

P. helianthi is a macrocylic heterothallic, autoceious fungus. Production of all the stages of spore forms on sunflower has been reported from important sunflower-producing countries (Kolte 1985, Sendall et al. 2006) and India (Mathar et al. 1975). The morphological characteristics of different fruiting structures of the fungus have been described in detail by Baily (1923) and Sendall et al. (2006). The life cycle of *P. helianthi* is represented by five (a–e) spore types:

a. *Urediniospores*: These are unicellular, dikaryotic, repeating spores produced in uredosori in 5–7 days after infection. They are brown and vary from subglobose to obovate in shape measuring 25–32 µm × 10–25 µm in size. The wall of these spores is cinnamon brown, 1–2 µm thick, and finely echinulate, usually with two equatorial germ pores. The spores are often slightly thickened at the apex and base. The urediniospores best germinate at 18°C–20°C by giving rise to germ tubes from equatorial germ pores. Germination of fresh spores is little affected by light intensity (2,200–4,300 lux) during spore production, but increasing light intensity is unfavorable for germination of urediniospores (Sood and Sackston 1972).
b. *Teliospores*: These are diploid resting spores produced in teliosori. They are bicelled, smooth, oblong, elliptical, and slightly constricted at the septum and measure 40–60 µm × 18–30 µm in size. The wall of the spore is smooth, chestnut brown, 1.5–3 µm thick at the sides, and 8–12 µm thick above with an apical pore. The spores are pedicellate; the pedicel may be colorless or pale luteous, fragile, and 60–150 µm. The teliospores produced at