Chapter 4
Genomic Designing for Biotic Stress Resistant Peanut

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Abstract Peanut is an oilseed crop that is essential for food and nutritional protection around the world. It is a source of livelihoods to smallholder growers of Asia and Sub-Saharan Africa. However, yield losses keep increasing under present climate change accompanied by rising CO_2 levels, erratic rainfall, rising and fluctuating atmospheric temperature, despite a considerable genetic gain in yield since the 1960s. Moreover, climate change and global warming lead to the ocurrence of a number of biotic stresses that severely affect crop yield and productivity. Furthermore, the cultivated peanut's genetic architecture and tetraploid nature have resulted in low genetic diversity for many economically significant traits. Significant achievement in yield and tolerance against biotic stresses has been made by conventional approaches, although time consuming, and laborious. Recent developments in genomics, combined with the use of available genetic resources, have raised the peanut to that of a "genomic

The original version of this chapter was revised: The authors affiliations have been updated. The correction to this chapter is available at
https://doi.org/10.1007/978-3-030-91035-8_9

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C. Kole (ed.), *Genomic Designing for Biotic Stress Resistant Oilseed Crops*,
https://doi.org/10.1007/978-3-030-91035-8_4

resource-rich oilseed crop.” As a result, a comprehensive approach that includes the application of genomic knowledge and techniques in crop improvement programs is critical for furthering peanut productivity advancement. Molecular markers are the most useful genomic tools for characterizing and harnessing usable genetic variability. Researchers are now moving faster towards traits and their genetic mapping studies. In addition, the existence of a diploid progenitor reference genome, tetraploid genotype, and 58 K SNPs, a high-density genotyping assay have greatly aided high-resolution genetic mapping. There has also been an important progress in developing multiparental genetic mapping populations namely, nested association mapping (NAM) and multi-parents advanced generation intercross (MAGIC) for mapping of quantitative and multiple traits simultaneously with high-resolution. The low cost of sequencing aided the development of mapping techniques based on sequencing especially QTL-sequencing for dissecting complex traits such as resistance to diseases. In peanut, there are a few promising examples of diagnostic markers for biotic stresses being developed and deployed in genetic improvement. In this context, this chapter provides recent information on the various biotic stresses faced by the crop across the globe, progress made through conventional breeding programs, transgenic approaches, and achievements in genomics with a special emphasis on QTL discovery, mapping of desirable traits and molecular assisted breeding approaches. The chapter also offers an overview of the most recent genomic discoveries, methods, and techniques used, as well as their possible applications for peanut improvement.

Keywords Peanut · Biotic stresses · Genomics · Transgenics · Molecular markers · Trait mapping

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4.1 Introduction

Peanut (*Arachis hypogaea L.*), also known as groundnut, is an essential oilseed-food-feed-fodder crop of choice, cultivated in more than 100 countries worldwide. The crop is cultivated as a sole and intercrop on nearly 28.5 million ha area globally, with record production of 45.95 million tonnes and productivity of 1611 kg/ha of pods-in-shell in the year 2018 (http://www.fao.org/faostat/en/#data/QC) (Fig. 4.1). Peanuts are grouped into two sub-species "*hypogaea*" and "*fastigiata*", mainly on the basis of pattern of branching and vegetative and reproductive axes distribution. The subspecies '*hypogaea*' consist of two botanical varieties, '*hypogaea*' (spreading-Virginia runner and semispreading—Virginia bunch types) and '*hirsuta*' (Peruvian runner), whilst the subspecies '*fastigiata*' is grouped into four botanical types ('*fastigiata*'-valencia types; '*vulgaris*'-spanish types; '*peruviana*' and '*aequatoriana*') (Gregory et al. 1973; Krapovickas and Gregory 1994). The cultivated Peanut is an amphidiploid/ disomic tetraploid designated as $2n = 4x = 40$. Peanut is an economically important oilseed crop and its kernels are rich with 45–55% oil, 25–30% protein, and 10–20% carbohydrate (Jambunathan et al. 1985). Peanut haulm contain carbohydrates (38–45%), minerals (9–17%), protein (8–15%) and lipids (1–3%), and has a digestibility of around 53% when fed to cattle. Peanuts are treated as

Fig. 4.1 Healthy peanut crop in the farmers' field

functional food as it is also an important source of minerals such as calcium (Ca), phosphorus (P), iron (Fe), magnesium (Mg), zinc (Zn), potassium (K), vitamins such as vitamin E, thiamine, riboflavin, pantothenic acid, niacin, antioxidants includes primarily *p*-coumaric acid, and bioactive compounds to promote health such as tocopherol, resveratrol, arginine. Over 60% of peanut produced worldwide is crushed for oil extraction while, 40% is used in food purpose and others (Birthal et al. 2010). Several fatty acids are present in peanut oil, of which palmitic, a saturated acid (7–12%), and unsaturated fatty acids viz., linoleic (25–35%) and oleic (40–50%) together make up about 90% of the total fats (Arya et al. 2016; Bera et al. 2018; Kamdar et al. 2020). Also available are high oleic lines with more than 80% oleic acid. There is a growing demand in the international market for peanut and peanut derived products, especially in confectionary use. The most popular peanut commodity in the Australia, Canada and USA, is peanut butter. Peanut kernels can either be eaten raw or roasted or boiled and can also be used to make baked and confectionary products. Peanut, as a legume crop, also helps to improve soil health quality and fertility by leaving organic matter and N_2 back in the soil.

Although the domesticated peanuts originated in region of southern part of Bolivia and north-western Argentina (Simpson et al. 2001), but 95% of peanut area globally is concentrated in Asia and in Africa in the semi-arid tropical regions (SAT) where small and marginal farmers grow the crop under rain-fed conditions (FAO 2017). Moreover, climate change leads to the ocurrence of number of biotic stresses that severely affects crop yield and productivity (Pandey et al. 2015). Nearly 75–80% of the world's peanuts are cultivated in developing countries by smallholder farmers who normally harvestpod yield of 500–800 kg ha^{-1} compared to the ptential yields of more than 2.5 ton per hectare. Low yields are mainly due to various diseases caused by nematodes, bacteria viruses and fungi (Kokalis-Burelle et al. 1997; McDonald et al. 1998). Major fungal diseases that target foliages are rust and leaf spots (early leaf spot and late leaf spot). Major fungal diseases that infect seed and seedlings are crown rot or *Aspergillus* crown rot, dipodia collar rot, yellow mold, damping off by *Rhizoctonia* spp., and smut. The major diseases affecting roots, stems, and pods include *Sclerotinia* root rot, *S.* blight, *Botrytis* blight, pod rot, *Fusarium* wilt, and charcoal rot. The major viral and mycoplasmal diseases are bud necrosis, stem necrosis, peanut mottle, peanut clump, peanut stripe, tomato spotted wilt, peanut rosette and stunt. Two major bacterial diseases are bacterial leaf spot and bacterial wilt. Peanut is also attacked by nematodes and certain insect-pests viz., *Spodoptera*, *Helicoverp*a, leaf miner, white grubs, aphids, thrips and jassids.

Good success has been achieved in peanut by conventional breeding approaches but the process is laborious and time consuming. The improved varieties of peanut with high production potential and resistance against biotic agents were developed and released for cultivation worldwide. A huge repository of variation of the cultivated peanut is present as germplasm accessions in the gene banks. The largest collection of peanut germplasm is being held at ICRISAT, India (15,445 accessions) followed by ICAR-National Bureau of Plant Genetic Resources (ICAR-NBPGR) with 14,585 accessions; ICAR-Directorate of Groundnut Research (ICAR-DGR) in India with 9024 accessions; 9917 accessions at the U.S. Department of Agriculture

(USDA); Oil Crops Research Institute (OCRI) of the Chinese Academy of Agricultural Sciences (CAAS) with 8083 accessions and 4210 accessions in China at the Crops Research Institute of the Guangdong Academy of Agricultural Sciences. Further, few to medium germplasm collections are held at the North Carolina State University (NCSU) and Texas A & M University (TAMU) in the USA; Brazil, at the Instituto Agronomico de Campinas and EMBRAPA-CENARGEN; and Instituto de Botánica del Nordeste (IBONE) and in Argentina, Instituto Nacional de Technologia Agropecuaria (INTA). Mostly, wide hybridization is being used to tap the usable genes from wild species (Kalyani et al. 2007; Stalker; Malikarjuna; Bera et al. 2010). However, genetic bottleneck in historical origin of the polyploid peanut from natural cross between the diploid ancestors *A. ipaensis* and *A. duranensis* followed by duplication of chromosome limits the available genetic diversity (Kochert et al. 1996). This limits the success of traditional breeding methods. Morever, the unlimited potential of wild species and wild forms, a reservoir of novel and useful alleles, remains under-utilized due to genetic barrier in introgression of genes into elite genotypes, compounded with the transfer of undesirable gene blocks. With the development of genetic linkage maps followed by marker discovery and identification of quantitative trait loci (QTLs) and genetic mapping of the target traits peanut improvement programm has accelerated during the last decade.

However, the impacts of climate change can be seen all over the world, stressing the urgent need for designing climate-smart (CS) crops to be able to cope-up these unfavorable conditions and aid in sustaining agriculture in order to achieve food and nutritional security. For improvement of two or more traits simultaneously, it is important to identify markers for important traits and use them in breeding programme. The cultivated peanut (*A. hypogaea*) is an allotetraploid (AABB) with a total genome size of 2.7 Gb formed from closely related sub genomes (Bertioli et al. 2016). Peanut genomic tools, such as molecular markers (Wang et al. 2012; Bosamia et al. 2015), genetic/linkage maps (Gautami et al. 2012b), and genome sequences of cultivated and progenitors species (Bertioli et al. 2019; Chen et al. 2019; Zhuang et al. 2019), have rapidly developed in the last decade. These advanced genomic tools and resources have facilitated the use of modern genetics and breeding methodologies such as genome-wide association studies (GWAS) for mapping multigenic trait and genomic selection (GS) for improvement of peanut crop. Genomic selection is one approach to broaden the genetic diversity by mining usable alleles from the wild species, landraces or wild relatives. An integrated breeding strategy is needed that will allow multiple desirable alleles to be selected facilitating pyramiding of number of genes as well as the deployment of GS approaches. Moreover, the transgenic approaches are being followed worldwide for the peanut improvement. Several useful genes either from wild species or synthetic genes could be transferred into established cultivars (Tiwari et al. 2008; 2011; Mehta et al. 2013; Sarkar et al. 2014, 2016; Bala et al. 2016; Patil et al. 2017; Bhalani et al. 2019). This chapter describes the major biotic constraints to peanut production (Table 4.1) and reviews the stages and extent of damage, and management options. It also reviews the genetic resources available, and the conventional and molecular breeding approaches to mitigate the

Table 4.1 Major biotic constraints to peanut production

S. No.	Disease	Causal organism	Distribution
1	Early leaf spot	*Cercospora arachidicola*	Worldwide
2	Late leaf spot	*Phaeoisariopsis personata Cercosporidium Personatum*	Worldwide
3	Rust	*Puccinia arachidis*	Worldwide
4	Web blotch	*Phoma arachidicola*, *Didymella arachidicola*	Angola, Argentina, Australia, Brazil, Canada, China, Commonwealth of Independent States, Japan, Lesotho, Malawi, Nigeria, South Africa, Swaziland, USA, Zambia, and Zimbabwe
5	Scab	*Sphaceloma arachidis*	Argentina, Brazil, Japan, and Swaziland
6	*Alternaria* leaf spot and veinal necrosis	*Alternaria alternate*	India, Vietnam, and Thailand
7	*Phyllosticta* leaf sPot	*Phyllosticta arachidis-hypogaea*	Burkina Faso, India, Malawi, Mozambique, Niger, Nigeria, Swaziland, Thailand, and Zimbabwe
8	Powdery mildew	*Oidium arachidis*	India and Israel
9	*Cercospora* leaf blight	*Cercospora canescens*	Thailand
10	*Myrothecium* leaf blight	*Myrothecium roridum*	India and Thailand
11	Zonate leaf spot	*Cristulariella moricola*	India, Thailand, and USA
12	*Sclerotium* leaf spot	*Sclerotium rolfsii*	India, Malawi, and Thailand
13	*Choanephora* wet blight	*Choanephora cucurbitarum*	Thailand and Philippines
14	Pepper spot and leaf scorch	*Leptosphaerulina crassiasca*	Angola, Argentina, Burkina Faso, India, Madagascar, Mauritius, Malawi, Mozambique, Niger, Nigeria, Senegal, Swaziland, Thailand, Taiwan, USA, Vietnam, Zambia, and Zimbabwe
15	Anthracnose	*Colletotrichum arachidis*, *C. dematium*, *C. mangenoti*	India, Niger, Nigeria, Sudan, Senegal, Taiwan, Tanzania, Thailand, Uganda, and USA
16	*Alternaria* leaf blight	*Alternaria alternate*, *A. tenuissima*, *A. arachidis*	India, Nigeria, and Thailand
17	*Pestalotiopsis* leaf blight	*Pestalotiopsis arachidis*	India, Nigeria, and Thailand
18	*Aspergillus* crown rot/collar rot	*Aspergillus niger*	Worldwide

(continued)

Table 4.1 (continued)

S. No.	Disease	Causal organism	Distribution
19	Yellow mold	*Aspergillus flavus*	Worldwide
20	*Diplodia* collar rot	*Lasiodiplodia theobromae*	Australia, India, Israel, South Africa, Thailand, USA, and Venezuela
21	*Rhizoctonia* damping-off	*Rhizoctonia solani*	Worldwide
22	Stem rot	*Sclerotium rolfsii*	Worldwide
23	*Sclerotinia* blight	*Sclerotinia minor*, *S. sclerotiorum*	Argentina, Australia, China, Taiwan, USA, and Zimbabwe
24	*Cylindrocladium* black rot	*Cylindrocladium crotalariae*	Australia, India, Japan, and USA
25	*Botrytis* blight	*Botrytis cinerea*	Australia, Commonwealth of Independent States, Japan, Malawi, Romania, South Africa, Swaziland, Tanzania, USA, Venezuela, Vietnam, and Zimbabwe
26	*Verticillium* wilt	*Verticillium albo-atrum*, *V. dahlia*	Argentina, Australia, Israel, and USA
27	*Fusarium* wilt	*Fusarium oxysporum*	Worldwide
28	Charcoal rot	*Macrophomina phaseolina*	Worldwide
29	Black hull/black pod rot	*Thielaviopsis basicola*, *Chalara elegans*	Israel, Argentina, Italy, South Africa, and USA
30	Pod rot	*Pythium myriotylum*, *Rhizoctonia solani*, *Fusarium solani*, *Fusarium oxysporum*, *Macrophomina phaseolina*	Worldwide
31	Bacterial wilt	*Ralstonia (Pseudomonas) solanacearum*	Angola, China, East Indies, Ethiopia, Australia, Fiji, Indonesia, Sri Lanka, Libya, Madagascar, Malaysia, Mauritius, Nigeria, Papua New Guinea, Philippines, Somalia, South Africa, Swaziland, Taiwan, Thailand, Uganda, USA, Vietnam, Zambia, and Zimbabwe
32	Bacterial leaf spot	*Unidentified bacterium*	India and Vietnam
33	Peanut mottle virus	*Peanut mottle virus*	All peanut-producing countries in Africa, the Americas, Asia, and Oceania

(continued)

Table 4.1 (continued)

S. No.	Disease	Causal organism	Distribution
34	Peanut stripe virus	*Peanut stripe virus*	Most peanut-producing countries in South and Southeast Asia, and USA
35	Peanut clump virus	*Peanut clump virus*	India and West Africa. Probably several other countries in Asia
36	Peanut bud necrosis	*Peanut bud necrosis virus*	South and Southeast Asia
37	Tomato spotted wilt virus	*Tomato spotted wilt virus*	Africa, the Americas, Australia and Europe
38	Stem necrosis	*Tobacco Streak Virus*	India, Australia, Brazil
39	Peanut rosette disease virus	A complex of two viruses (*Peanut rosette assistor virus*, *Peanut rosette virus*) and a satellite RNA	Sub-Saharan Africa, Madagascar
40	Peanut stuntvirus	*Peanut stunt virus*	North America and southern China
41	Peanut streak necrosis virus	*Sunflower yellow blotch virus*	Southern Africa
42	Cowpea mild mottle virus	*Cowpea mild mottle virus*	Asia and Africa
43	Peanut yellow spot virus	*Peanut yellow spot virus*	Thailand and India
44	Witches' broom	*Mycoplasma-like* (*organism MLOs*)	Burkina Faso, China, India, Indonesia, Japan, Niger, Taiwan, Thailand, and USA
45	Root-knot nematode	*M. arenaria*, *M. hapla*, *M. javanica*, *M. incognita*	*M. arenaria*: Egypt, India, Israel, Malawi, Senegal, Taiwan, USA, and Zimbabwe *M. hapla:* Australia, China, India, Israel, Japan, South Africa, South Korea, USA, and Zambia. M. javanica: USA, *M. incognita*: USA
46	Root-lesion nematode	*Pratylenchus brachyurus*	Australia, Benin, Egypt, Gambia, India, Nigeria, Senegal, Thailand, USA, and Zimbabwe
47	Kalahasti malady	*Tylenchorhynchus brevilineatus*	India
48	Peanut smut	*Thecaphora frezii*	Argentina

Source http://oar.icrisat.org/7190/1/IB_PeanutDiseases-2012.pdf

effect of biotic stresses. This chapter provides updates on QTL mapping for economically important traits. In addition, we also discussed identification of SNPs linked to gene/QTLs based on next generation sequencing (NGS) approaches.

4.2 Description of Different Biotic Stresses

4.2.1 Fungal Diseases

4.2.1.1 Foliar Fungal Diseases

Stages and extent of damage

Peanut rust (*Puccinia arachidis* Speg, the causal agent) is a serious foliar disease. The pathogen *P. arachidis* is host-specific and known to produce at both uredial and telial stages. It is, however, almost entirely known for its uredial stage, which is abundant. The pathogen spreads quickly by repeated infection cycles of wind-borne inocula of uredospores (Hennen et al. 1976). It is characterized by orange-red/brown-colored, circular to elliptical pustules (uredinia) ranged in size from 0.3 to 2.0 mm in diameter on the lower surface of the leaves. Though uredia are the main stage of the infection cycle, there are also a few records of the occurrence of the telial stage. Telia chiefly occur on the under surface of peanut leaves (Bromfield 1971). Teliospores are light or golden yellow spores with acute to rounded and thickened apex that are oblong, obovate, ellipsoid, or ovate in shape. They germinate at maturity without a dormancy phase. Rust causes significant yield loss to peanut globally (Subrahmanyam and McDonald 1983). However, disease incidence and severity vary with locations and seasons. The pathogen can cause up to 57% economic damage to the peanut crop when environment is warm and humid (Subrahmanyam and McDonald 1987). Under favorable conditions and the presence of susceptible cultivars, however, rust-related losses can reach to 70% (Subrahmanyam et al. 1985a, b, c; Dwivedi et al. 2002a). Rust losses are compounded if the crop is also affected by leaf spots, such as early leaf spot caused by fungus, *Cercospora arachidicola* and late leaf spot caused by fungus *Phaeoisariopsis personata*, which can result in yield losses of up to 70% (Nutter and Shokes 1995; Shokes and Culbreath 1997). Both pathogens are soil-borne, with conidia produced directly from mycelium in crop debris in the soil, deposited on the first-formed leaves, and then carried to later-formed leaves and other plants by rain splash, wind and insects. Ascospores, chlamydospores, and mycelial fragments, on the other hand, are possible inoculum sources. On volunteer peanut plants and infected crop debris, early and late leaf spot pathogens can survive from season to season. Outside of the *Arachis* genus, no host species has been identified. The early leaf spot pathogen's telemorph and telemorphs of late spot pathogens, *Mycosphaerella arachidis* Deighton and *Mycosphaerella berkeleyi* Jenk, respectively are rarely seen on peanut. Leaf spots damage the plant by causing lesion formation and inducing leaflet abscission, both of which reduce the total photosynthetic area

of the plant (Fig. 4.2). *Cercospora arachidicola* forms subcircular lesions of more than one mm in diameter (Tshilenge 2010). Most sporulation occurs from the lesions on the upper leaf surface where dark brown with always yellow halos, and a lighter shade of brown lesions are formed on the lower leaflet surface. Lesions caused by *Phaeoisariopsis personata* are usually small in size, more nearly circular, and darker (black) and slightly rough than those of *C. arachidicola*, usually do not have yellow halos and most sporulation occurs on the lower surfaces. In addition to leaf spots, these pathogens cause lesions on all above-ground sections of the plant, including stipules, petioles, roots, and pegs (Subrahmanyam et al. 1982a, b).

Fig. 4.2 Wild Arachis sp. infected with *Alternaria* leaf blight

Management

Between successive crops, a fallow period of at least one month should be observed. Crop rotations involving cereals or other non-host crops are successful in preventing disease spread (Mondal et al. 2014a, b). To avoid inoculum buildup and carryover, volunteer peanut plants should be eradicated, sowing times should be planned to avoid contamination from outside, and environmental conditions conducive to the disease should be avoided. Maintaining field sanitation by weeding and proper plant spacing should be added to this (Kokalis-Burelle et al. 1997). Since leaf spot pathogens are primarily soil-borne, crop rotation out of peanuts for 2–3 years and burial of peanut crop residues are used to reduce inoculum load. Leaf rust can be managed with a variety of fungicides and fungicide mixtures. Chlorothalonil, tridemorph, mancozeb-zinc combinations, hexaconazole, strobilurinsterol-inhibitors, and other sulphur-based fungicides are effective in reducing peanut rust incidences (Kokalis-Burelle et al. 1997). Benomyl, chlorothalonil, copper hydroxide, fentin hydroxide, maneb and mancozeb, sulfur, copper/sulpher dusts, propiconazole, and tebuconazole are some chemicals that are being used to reduce the threat due to leaf spot epidemics (Smith and Littrell 1980).

Several biological agents *viz.*, *Acremonium persicinum*, *A. obclavatum*, *Eudarluca caricls*, *Penicillium islandicum*, *Tuberculina costaricana* and *Verticillium lecanii* have been reported significantly inhibiting *invitro* germination of rust spores (Ghewande 1990). Also, pre-treatment with conidia of *T. harzianum* has shown to significantly inhibit germination percentage and germtube growth of *P. arachidis* (Govindasamy and balasubramanian 1989). *Fusarium chlamydosporum*, a mycoparasite that releases chitinase capable of cell wall lysis of fungi can also act as a biocontrol agent (Mathivanan et al. 1998). However, no serious or significant attempts have been made in the field to use any of these species for controlling peanut rust biologically. Mycoparasites, *Dicyma pulvinata* and *Verticillium lecani*, *Acremonium obclavatum*, *Fusarium spp* and *Penicillium spp* are also known to parasitize the leaf spot pathogens. In glasshouse trials, *Pseudomonas* spp., which has broad-spectrum antifungal activity, was also found to significantly reduce late leaf spot (Haas and Keel 2003). Further, foliar spray of chitinolytic bacteria, *B. circulans* and *S. marcescens* for control of LLS of peanut has been documented (Kishore et al. 2005).

4.2.1.2 Fungal Diseases Affecting Stem, Root and Pod

The major fungal diseases attacking root, stems, and pods include *Sclerotium*/Stem rot, *Sclerotinia* blight and *Botrytis* blight, *Fusarium* wilt, pod rot and charcoal rot.

Stages and extent of damage

Stem rot/white mold/southern blight of peanut is caused by a soil dwelling necrotrophic fungal pathogen, *Sclerotium rolfsii*. It is one of the most severe biotic stresses that can affect peanuts, and it is most prevalent in the tropics and subtropics regions and other temperate regions of the world with warm and humid climates

Fig. 4.3 Artificially inoculated peanut field with *Sclerotium rolfsii* for screening resistance to stem rot

(Deepthi and Reddy 2013). *Sclerotium rolfsii* is a deuteromycete fungus belonging to the group "*Mycelia Sterilia*" (Alexopoulos et al. 1962). Although the basidiomycete *Athelia rolfsii* (Cruz) Tu and Kimbrough has been described as the sexual stage of *S. rolfsii*, but it is very rarely seen in the peanut field (Tu and Kimbrough 1978). White mycelia and round, brown sclerotia with diameters ranging from 0.5 to 2 mm distinguish the fungus (Figs. 4.3, 4.4 and 4.5). In the absence of a host, it persists for several years as mycelia in crop debris and as sclerotia in the soil (Punja 1985). The pathogen does not produce any asexual spores. The pathogen primarily infects stems, but it also targets leaves, pods, and other plant parts, resulting in severe damage at all stages of crop growth. Chlorosis and/or wilting of a lateral branch are the first signs of infection; however, if the main stems become infected, the entire plant may appear wilted or chlorotic (Backman and Brenneman 1997). By forming oxalic acid and cell-wall degrading enzymes, stem rot fungus kills plant tissues before colonization (Cilliers et al. 2000; Ganesan et al. 2007). If the fungal pathogen attacks the pods, they develop a brown rot that appears mashed and water-soaked (Punja 1985). Stem rot causes yield losses that typically range from 10 to 40%, but can reach up to 80% in heavily infected fields (Mehan and McDonald 1990; Akgul et al. 2011; Bera et al. 2014a; 2016a).

The soil-borne fungi *Sclerotinia minor* Jagger and *Sclerotinia sclerotiorum* (Lib.) de Bary trigger *Sclerotinia* blight. S*clerotinia* blight is a devastating peanut disease marked by thick tufts of white mycelium and broad, irregularly formed sclerotia. It is a economically significant disease that causes significant yield losses and affects kernels quality. The loss of yield due to disease occurrence is estimated to be 10%,

Fig. 4.4 Sclerotia of *Sclerotium rolfsii* on a heavily infected peanut plant

Fig. 4.5 Peanut plant and pods damaged by *Sclerotium rolfsii*

but in extreme cases, it may be as high as 50% (Porter and Melouk 1997). Sclerotia of *Sclerotinia minor* are often small and abundant, while those of *Sclerotium sclerotiorum* are large and less abundant. Peanut is contaminated by mycelia from germinating sclerotia in majority of the cases. The plant finally dies, and sclerotia proliferate on the dead tissue in large numbers. Some sclerotia are shed from plant tissue into the soil or may be preserved as overwintering inoculum on dead plant tissue. Sclerotia germinate into mycelium or apothecia under ideal conditions. *Sclerotinia minor* and *Sclerotinia sclerotiorum* are ascomycetes. One or more pale orange to white apothecia (sexual stage) may emerge from a single sclerotium. The fruiting body produces ascospores that range in size from 8–17 × 5–7 μm (Porter and Melouk 1997). Watery lesions appear on all infected tissues, including pegs and pods, and the tissues are quickly coated with white fluffy mycelium. On roots, pegs, and pods, yellowish-brown bleached lesions appear after mycelium penetrates the tissues. The stems become girdled and die, and the leaves become chlorotic and necrotic (Backman and Brenneman 1997).

Botrytis blight is also known as gray mold of peanuts and is due to fungus, *Botrytis cinerea* that occurs only sporadically in cold, wet weather. *Botrytis cinerea* Pers.: Fr. (anamorph) belongs to molds/deuteromycete class that rapidly colonizes plants. The fungus can cause plant tissue as well as the entire plant to wilt and die. Blight caused by *B. cinerea* is marked by the abundance of conidia and sclerotia produced on infected plant sections. The fungus overwinters as massive sclerotia, which are irregular structures and colored dark-brown to black (Porter 1997). The ascomycetous stage of *Botrytis* blight, *Botryotinia fuckeliana* (de Bary) Whetzel, is rarely spotted. Mycelium, which comes from germinating sclerotia or conidia, is the primary source of inoculum. *Botrytis* blight is not a serious peanut disease, and the damage it causes is generally minor. Several *Pythium* spp., specifically *P. myriotylum*, *P. irregulare*, and *P. ultimum* (Wheeler et al. 2005), have been found to be associated with diseased peanuts, causing damage to the pod and kernels, as well as substantial yield loss of up to 80% (Beute 1997). Peanut damping-off, root rot and vascular wilt may all be caused by *Pythium* spp. Peanut pod rot is an economically significant disease that affects the quality and yield potentiality of the crop. *Rhizoctonia solani*, *Sclerotium rolfsii*, and *Pythium* spp. are the most common soil-borne mycelial pathogens that cause pod rot (Kokalis-Burelle et al. 1997). *Pythium* spp. caused pod rot is marked by browning and water-soaking of pods in the early stages, accompanied by a brown to black appearance in the later stages (Wells and Phipps 1997). *Pythium* spp. are fungi with white fluffy mycelia that produce sporangia, asexual reproductive structures that germinate by forming motile zoospores. Sexual spores *i.e.*, oospores serve as the primary survival structure of *Pythium* species. Due to the lack of above ground symptoms, it's difficult to estimate yield losses caused by *Pythium* pod rot, but losses of up to 80% have been recorded (Beute 1997). *Rhizoctania solani* Kühn is another soil-borne pathogen capable of causing seed decay, damping off, root rot, limb rot, and pod rot (Garren 1970). The anamorph, *Rhizoctonia solani* Kühn, is a Deuteromycete that does not produce asexual spores and the teleomorph, *Thanatephorus cucumeris*, is a Basidiomycete. Pigmented and septate hyphae, as well as non-differentiated sclerotia, are found on plant debris that germinate to infect host tissues (Brenneman

1997). *Rhizoctonia* pod rot is distinguished by a dry, brown or russet-colored rotted pod, as opposed to *Pythium* spp that form dark greasy-appearing lesions. Pod rot, caused by *R. solani*, can result in yield losses of 22–28% in favorable environmental conditions (Besler et al. 2003). Another soil-borne fungus, *Fusarium solani*, is involved in pod rot, as a predisposing factor as well as one of the saprophytic fungus that aggravates the pod's final breakdown. *Fusarium* spp. reproduces on plant debris and lives saprophytically in soil. Conidia are formed in abundance but are short-lived. Chlamydospores are the long lasting survival structures (Frank 1972; Garcia and Mitchell 1975). *F. solani* makes pods more susceptible to *Pythium myriotylum* infection. Later colonization of pods by *P. myriotylum* is accompanied by rapid increase in pod rot. Finally, pod disintegration is caused by *F. solani* and saprophytic species.

Management

The key technique for controlling stem rot is to prevent inoculum build-up. Disease build-up can be reduced by deep plowing, weed control, and crop rotation with corn or grain sorghum (Backman and Brenneman 1997). Excess canopy growth and irrigation should be avoided because they encourage disease development. Solar heating of moistened soils under a polyethylene tarp, combined with the application of *Trichoderma harzianum*, reduces *S. rolfsii* disease (Grinstein et al. 1979). To reduce *Sclerotinia* disease incidence, it is strongly recommended to minimize damage to peanut plants caused by farm machinery and other mechanical means (Porter et al. 1982). To avoid fungal colonization due to frost damage, *Botrytis blight* should be managed to a large extent by avoiding excessive irrigation, good drainage, mulching, and planting early maturing peanut varieties. Overwatering and flooding should be prevented because *Pythium* spp. forms motile zoospores that travel in water. Peanut rotation with grasses like corn, sorghum, or other pasture grasses may help minimize *Pythium* spp. and *R. solani* (Baird et al. 1995; Brenneman 1997). Rotation of crops has also been shown to minimize *Pythium* spp. inoculum density while having little impact on disease incidence (Beute 1997).

Numerous fungicides are known to inhibit the germination of sclerotia or the mycelia growth of various fungi. To combat stem rot, pentachloronitrobenzene (PCNB) and carboxin have been used. Tebuconazole and other sterol-inhibiting triazole-type fungicides have provided more than 80% control on stem rot (Backman and Brenneman 1997). Propiconazole and flutolanil also offer excellent control of stem rot (Csinos 1987; Grichar 1995). Pruning of peanut vines along with the application of benomyl is reported to control stem rot (Backman 1975). Further, fumigation of soils with methyl bromide, chloropicrin, or metham-sodium is toxic to sclerotia (Elad et al. 1980). Fungicides such as iprodione and fluazinam are known to control *Sclerotinia* blight disease (Bailey and Brune 1997; Butzler et al. 1998). The use of fungicide chlorothalonil against leaf spots should be avoided because it has been shown to trigger *S. minor* to germinate (Beute and Rodriguez-Kabana 1979). However, under conditions conducive to *Sclerotinia blight* chlorothalonil is highly effective and widely used to control the disease. Some protection against *B. cinerea* is

provided by foliar sprays with fungicides including benomyl and chlorothalonil. Iprodione also inhibits the spores germination and inhibit the growth of fungus (Langston et al. 2002). Pesticides such as PCNB and metalaxyl, also have inhibitory activity on *Rhizoctonia* and *Pythium* spp., respectively (Filonow and Jackson 1989). Tebuconazole and Azoxystrobin are systemic fungicides with a wide spectrum of activity that can be used to control R. *solani* (Baird et al. 1991; Brenneman 1997). Metalaxyl and mefenoxam may be effective against oomycetes including *Pythium* spp. (Filonow and Jackson 1989; Lewis and Filonow 1990). High rates of gypsum application at flowering are recommended. In certain areas, the application of high doses of gypsum greatly reduced pod rot caused by *P. myriotylum* (Alva et al. 1989). It is well established that adequate calcium nutrition in the soil is critical for pod rot control (Walker and Csinos 1980; Csinos et al. 1984). Fungicides such as Tebuconazole and flutolanil or fluazinam offer an effective chemical control against *Rhizoctonia* induced pod rot. *Fusarium* populations are selectively suppressed by soil solarization and treatments of soil with biocide metham sodium in sublethal doses. Biological control with antagonistic fungi have also been demonstrated. The fungi *Trichoderma harzianum*, *T. viride*, *T. hamatu*, *T. koningii* and *Pseudomonas fluorescens* have successfully suppressed stem rot severity. They inhibit mycelia growth of the pathogen and suppress sclerotial formation (Karthikeyan et al. 2006; Kwee and Keng 1990). *Talaromyces flavus* parasitized hyphae as well as sclerotia of *S. rolfsii* (Madi et al. 1997). *T. harzianum* proved to be the most efficient biocontrol agent against *S. typhimurium.* When compared to other possible biocontrol agents, *T. harzianum* comes out to be the most effective biocontrol agent to control *S. rolfsii* (Kulkarni and Kulkarni 1994). Further, soil inoculation with *Rhizobium* reduced the population of *S. rolfsii* in the rhizosphere (Bhattacharyya and Mukherjee 1990). *P. fluorescens*, *P. aeruginosa*, *Serratia marcescens* and *B. subtilis* are also antagonistic to stem rot fungus where, *P. aeruginosa* completely inhibited the growth of *S. rolfsii* by producing a siderophore (Podile et al. 1988; Ordentlich et al. 1987). Antagonistic species such as *Gliocladium spp.*, *Penicillium spp., Sporodesmium spp.*,*Talaromyces spp.*, and *Trichoderma spp.*, release compounds such as chitinases, and β-1, 3-glucanases which are enzymes that can pierce the cell walls and cause complete cell death, and also attack on sclerotia of *S. minor* (Sherwood et al. 1995). *Teratosperma oligocladum* and *Sporidesmium sclerotivorum* effectively reduce the survival of sclerotia of *S. minor* in soil (Bullock et al. 1986; Adams 1989; Adams and Wong 1991). *Coniothyrium minitans*, another biocontrol agent, disrupts the life cycle of *Sclerotinia* by targeting the sclerotia and rendering the sclerotia useless as inocula (Jones et al. 1974). *Trichoderma harzianum*, a competitive fungus is also effective against gray mould. A *Gliocladium* species has been known to parasitize conidia, conidiophores and sclerotia of *Botrytis.* The hyperparasites, *Botryotrichum piluliferum*, *Coniothyrium sporulosum*, *Dicyma olivacea*, *Gliocladium catenulatum*, *Stachybotrys chartarum*, *Stachylidium bicolor*, *Stachybotrys elegans*, *Trichothecium roseum*, *Verticillium chlamydosporium*, *V. tenerum*, and *V. bigguttatum* parasitize the hyphae of *Rhioctonia. G. virens*is is known to colonize mycelia as well as sclerotia of *R. solani* (Turhan 1990; Morris et al. 1995; Bertagnolli et al. 1996). In the presence of *T. harzianum*, the growth of *R. solani* was significantly slowed (Tu and Vaartaja

1981). *Pseudomonas fluorescens*, *P. aeruginosa*, *B. subtilis* and *B. megaterium* also inhibit the growth of *R. solani* (Savithiry and Gnamanickam 1987; Podile et al. 1988; Turner and Backman 1991; Badel and Kelemu 1994).

4.2.1.3 Fungal Diseases Affecting Seed and Seedlings

Major fungal diseases that affect seed and peanut seedlings include collar rot or *Aspergillus* crown rot caused by *Aspergillus niger*, yellow mold caused by *Aspergillus flavus*, diplodia collar rot caused by *Lasiodiplodia theobromae* and *Verticillium* wilt.

Stages and extent of damage

Collar rot or seedling blight or crown rot is caused by the fungus *Aspergillus niger* Tiegh., a necrotrophic fungus that exists in an anamorph stage in soil and on crop residues. Soil-borne conidia attack seeds and cause rotting. Infected seeds are covered with masses of conidia and fail to germinate (Subrahmanyam et al. 1992). The pathogen attacks the emerging young seedling and brown discolored spots appear on the collar region. The affected portion becomes soft causing yellowing of lower leaves, blighting of the shoot, finally leading to the death of the crown (Suzui and Makino 1980). While rotting of seeds and preemergence damping-off are general symptoms, infection may also affect mature plants. Large lesions form below the soil line on the stem and spread upwards along the branches, causing leaf drooping and sudden wilting in young plants. The pathogen lives in soil plant litter. The percentage of plants that die as a result of collar rot varies between 28 and 50% (Ghewande et al. 2002).

Yellow mold is a seedling disease caused by the saprotrophic and pathogenic fungus *Aspergillus flavus*. It lives in the soil on organic sources of nutrients in the form of mycelia and resistant structure sclerotia. These structures germinate directly to either produce mycelia or give rise to conidiophores and conidia. Both mycelia and conidia serve as the primary sources of inocula (Scheidegger and Payne 2003). *A. flavus* has an extraordinary ability to colonize seeds. The mold causes pre-emergence rotting of seed, reduce seed viability and germination and causes seedlings to rot (Kumar et al. 2012). After seedlings emerge, infection is mainly confined to the cotyledons. The diseased plants are chlorotic and stunted. Aflatoxin, a form of secondary metabolite produced by the pathogen, is the most toxic carcinogen among known mycotoxins. (Calvo et al. 2002; Klich 2007; Krishnamurthy et al. 2008). As a result, either by killing the plant or by contaminating peanut kernels with aflatoxins, which are then either unmarketable or cause significant health issues to both human and animals that consume contaminated kernels.

Diplodia collar rot of peanut, caused by the soil-borne saprophyte *Lasiodiplodia theobromae* (Pat.) Griffon and Maubl. and by *Diplodia gossypina* are known to cause wilting in immature and mature plants (Porter and Garren 1968). For long periods of time, mycelia and mature conidia of the fungus may be found dormant in soil and plant debris. Heat-stressed peanut plant tissue is more susceptible to *D. gossypina* colonization. Mycelia originating from germinating or mature conidia and

mycelial fragments may cause primary infection. Necrotic areas that are elongated and characterized by light brown centers with dark brown margins are formed on above ground stems. On the surfaces of necrotic tissues, single or compound pycnidia can be seen individually or in groups. Diplodia collar rot occurs infrequently around the world, causing only minor economic losses. Collar rot normally causes yield reductions of less than 1%, but reductions of 25% or more have been recorded (Porter and Phipps 1994).

Verticillium wilt is caused by *Verticillium dahlia* Kleb., which can survive in the soil as microsclerotia for long periods of time. White fluffy mycelia and hyaline single cellular conidia are also produced by the fungus. The fungus infects the host plant systemically by entering the roots and spreading through the xylem, causing vascular discoloration in the crowns, stems, roots, and petioles (Melouk and Damicone 1997). Plant death is preceded by general yellowing, defoliation, leaf necrosis on margins, wilting, general stunting, and dehydration as the disease progresses (Purss 1961; Melouk and Wadsworth 1990).

Management

Irrigation and weed management can be effective in reducing fungal disease. Irrigation alleviating drought stress or early harvest to escape drought are the best control measures for minimizing aflatoxin contamination. Furthermore, planting noninfected, high-quality seeds are the safest way to prevent seed and pre-emergence seedlings rotting caused by A. *flavus*. Diplodia collar rot incidence can be reduced by rotating peanut with crops other than hosts. Furthermore, by manipulating row orientation and maintaining adequate foliage during the growing season, heat induced injury to basal stems of plants can be minimized, and disease severity can be reduced. High temperatures and moisture tension exacerbate the severity of *Verticillium wilt.* As a consequence, infested fields should be irrigated on a daily basis. It's also a good idea to plant V*erticillium*-free seed. Since certain weeds are also susceptible to *V. dahliae*, weed control may help reduce the occurrence of *Verticillium* wilt. Peanuts grown in the presence of nonhost crops like grain sorghum/ Sudan grass produce less wilt than peanuts grown in the presence of susceptible crops like cotton, okra, or peanut. *Verticillium dahliae* has a longer lifetime in the soil than microsclerotia, and short-term crop rotations have no effect on their levels.

Triazole compounds including propiconazole, tebuconazole and difenconazole, carbendazim, carboxin and captan are known to inhibit the mycelial growth and spore production of the collar rot fungus. *Verticillium* wilt cannot be regulated with chemicals. While metham sodium applied via sprinkler irrigation has been effective in controlling the disease in sandy soil (Krikun and Frank 1982).

Biological control has shown to control infection with varying degree of success. *Trichoderma* spp (Harman et al. 1981), *Bacillus* spp. (Capper and Campbell 1986) and *Pseudomonas* spp (Vidyasekharan and Muthamilan 1995) are known to be antagonistic are used to control the crown root fungus with varying degrees of success. In soil treated with *T. harzianum* at both the seedling stage and vegetative growth stage, disease incidence was reduced (Garren et al. 1969; Harder et al. 1979). Further, the treatment of peanut seeds with *Bacillus subtilis* significantly controls crown rot

(Podile and Prakash 1996). *Streptomyces* spp. have a strong antagonistic effect on the growth and development of *Aspergillus* (Zucchi et al. 2008; Zhang et al. 2013). Also, the bio-control agent, *Trichoderma harzianum*, and *T. viride* are known to control *A. flavus* infection as they showed the ability to parasitize *A. flavus* by coiling around its hyphae (Chiuraise et al. 2015). *A. shirousamii* lessen the formation of mycotoxin-aflatoxin by *A. flavus* (Kim and Kim 1986). An atoxigenic strain of *A. parasiticus* is used as a competitive agent to reduce aflatoxin contamination in peanut kernels (Dorner et al. 1992). More recently, pre-harvest aflatoxin contamination of peanut has been effectively be controlled by use of commercial products namely, AflaGuard and Aflasafe derived from atoxigenic strains in the United States (Luis et al. 2017). *A. flavus* produced less aflatoxins in peanut kernels when *Flavobacterium odortum* was present, and *Pseudomonos cepacia* absolutely stopped *A. flavus* from growing (Chourasia 1995; Misaghi et al. 1995). Treatment with a mixture of chitosan or *Bacillus* reduced the growth of *A. flavus* (Cuero and Osuju 1991).

4.2.2 Bacterial Diseases

Two major bacterial diseases are bacterial wilt and bacterial leaf spot.

Stages and extent of damage

Ralstonia solanacearum (Smith) causes bacterial wilt, which is a severe global disease and poses a serious risk to peanut production in many wet and humid regions. *Ralstonia solanacearum* is a aerobic, rod-shaped, and gram-negative bacterium that does not form any spores and accumulate poly-p-hydroxybutyrate as a carbon source (Hayward and Hartman 1994). The phenotypic properties of *R. solanacearum* are heterogeneous, and it has been grouped into five biovars based on its ability to use unique carbon sources. Biovars 1, 3, and 4 have been identified as peanut pathogens. *R. solanacearum* isolates have been tentatively classified into five groups, with race 1 being known in peanut (He et al. 1983). This soil-borne pathogen infects plant roots through lesions/wounds and spreads easily through the conducting system, causing dark xylem and pith discoloration. When the cut ends of stems are immersed in water, milky white ooze with masses of bacteria appears. The roots and pods of infected plants are discolored and rotten. In the advanced stage, drooping and death of branches and the entire plant may occur (Kelman 1953; Mehan et al. 1994; Vasse et al. 1995). In China, Indonesia, and Vietnam, bacterial wilt is a major constraint to peanut production. Yield losses of 10–30% are normal, with losses as high as 60% in heavily infected fields (Mehan et al. 1994).

An unspecified *Pseudomonas* species causes bacterial leaf spot. Small, circular to irregular shaped light-brown water-soaked lesions develop on the leaves in the early stages of infection. Lesions enlarge and grow as chlorotic halos as the disease progresses, resulting in shedding of leaf (Subrahmanyam et al. 1992).

Management

The key sources of bacterial wilt inoculum are susceptible hosts or weed hosts, as well as infected crop residues. Rotation of peanut with non-host crops is effective in reducing losses due to wilt. Seeds infected with fungus are also a possible source of prime inoculum, with seed transmission rates ranging from 4 to 15%. Drying seeds to moisture content below 9% is recommended to control seed borne infection. Flooding fields of peanut for 15–30 days prior to sowing, enhancing soil drainage, preserving sufficient soil moisture, early sowing to avoid high temperatures, burning crop residues, weed reduction, quarantine, and cleaning farm tools after operations in infested fields are all cultural control steps (Mehan et al. 1993).

Some predominant avirulent strains such as *R. solanacearum* and *Pseudomonas* spp., have been found to be antagonistic to the bacterial wilt pathogen, followed by *Acinetobacter* spp., *Bacillus* spp., and *Streptomyces* spp.

4.2.3 Viral Diseases

Viral diseases in peanut caused by cucumber mosaic virus (CMV), peanut bud necrosis virus (GBNV), peanut rosette assistor virus (GRAV), peanut rosette virus (GRV), satellite RNA associated with GRV and/or GRAV, Indian peanut clump virus (IPCV), peanut clump virus (PCV), peanut mottle virus (PeMoV), peanut stripe virus (PStV) and peanut stunt virus (PSV) and tomato spotted wilt virus (TSWV) are the most economically important viral pathogens of peanut and are responsible for serious yield losses globally or regionally.

Stages and extent of damage

Among viruses, peanut rosette disease causes greater yield loss than any other virus disease affecting peanut in the semiarid tropics. Peanut rosette disease has a complex etiology involving three agents: peanut rosette assistor luteovirus (GRAV; Murant 1989), peanut rosette umbravirus (GRV; Murant and Kumar 1990), and a satellite-RNA (sat-RNA; Murant et al. 1988) of GRV. GRV and sat-RNA are packaged within the GRAV coat protein to be transmitted by the aphid, *Aphis craccivora* in a persistent manner. Since none of these agents are carried by seeds, viruliferous aphids are the main vectors of primary infection into the crop. The two predominant symptoms of peanut rosette are "chlorotic" and "green" rosette plants. Due to shortening internodes and decreased leaf size, the virus causes extreme stunting, that cause a bushy appearance. The amount of yield loss due to peanut rosette disease depends on the plant's growth stage; infection before flowering will result in a 100% loss in pod yield.

Tomato spotted wilt is caused by Tomato spotted wilt virus (TSWV), a species of the genus Tospovirus and family Bunyaviridae. TSWV is transmitted by several species of thrips viz., *Thrips tabaci*, *T. palmi*, *T. setosus*, *Frankliniella spp.*, *Scirtothrips* spp. but the virus is not transmitted through seed or pollen (Mandal et al.

Fig. 4.6 Peanut plants infected with peanut bud necrosis disease

2001; Peters 2003). The most significant species is *F. fusca*, which is the most common vector that reproduces on peanuts. The virus produces a broad range of symptoms from chlorotic and/or necrotic to severe stunting and subsequent death of susceptible peanut plants. It also causes early germination of seeds reducing further crop yield. The disease reduces the number of pods produced, kernel size and yield per plant. Losses up to 100% have been reported due to spotted wilt (Culbreath et al. 2003).

Bud necrosis (Fig. 4.6) is a major problem in dry areas, resulting in yield reductions up to 80% (Chohan 1974; Kamdar et al. 2014). Crop losses worth up to US$89 million from India were reported (Reddy and Devi 2003). The causal virus of this disease was initially identified as tomato spotted wilt virus (TSWV) in India (Ghanekar et al. 1979) but now it is studied to be caused by TSWV or PBNV (Peanut Bud Necrosis Virus) (Reddy et al. 1992; Adam et al. 1993; Satyanarayana et al. 1996). Chlorotic spots on leaves or mottling of immature leaflets or necrotic and chlorotic rings and streaks are formed as a result of viral infection (Bera et al. 2014b). In the later stages of plant growth, petioles bearing infected leaflets become flaccid and droop, finally followed by necrosis of terminal buds (Jasani et al. 2018a). The entire plant shows a highly stunted bushy appearance. Early-infected plants produce thin, shriveled seeds with red, brown, or purple mottling on the testae. Plants that are late infected can produce normal-sized seeds, but the testae are mottled and cracked (Reddy 1991). Both viruses are mechanically transmitted. GBNV is also transmitted by thrips vector, *Thrips palmi* (Reddy and Devi 2003) and TSWV is transmitted probably by vector, *Frankliniella fusca* and *F. occidentalis.*

Peanut clump is caused by two distinct, serologically unrelated viruses viz., peanut clump virus (PCV) mostly confined inwestern Africa, and Indian peanut clump virus (IPCV), virus from India. On newly emerging quadrifoliates of young plants, mottling, chlorotic, and mosaic rings appear. Infected leaves turn dark green, either

with or without faint mottling as a result of the virus infection. Plants that have been infected early are severely stunted, but they may produce flowers. If pods form, they are underdeveloped, and seed weights can be decreased upto 60%. These viruses are transmitted through seed, soil-borne plasmodiophoromycete fungi, *Polymyxa graminis* and mechanically by sap inoculation (Reddy et al. 2005). Since viruses are present on the seed coats of all kernels from infected plants, both viruses are transmitted by seed in peanuts with a frequency of more than 6%. In peanut almost 100% crop loss has been reported if the disease occurs in the early growing season, and up to 60% yield loss in late infected plants (Reddy 1991). The annual loss due to this disease globally is estimated to surpass US$38 million (Reddy and Devi 2003).

Peanut mottle caused by the potyvirus, peanut mottle virus (PeMoV), is another viral disease of economic importance. On young leaflets, the virus produces a faint mottle or a mosaic of irregular size and shapes and islands of dark green colour. The number of pods and root nodules along with size of pods are reduced in plants infected with virus. Also, diseased plants are slightly stunted. Varied symptoms are caused by different strains of the virus as reported by Paguio and Kuhn (1973) and Bijaisoradat et al. (1988). Symptoms caused by chlorosis and necrosis strains of PeMoV are similar to those caused by TSWV (Sreenivasulu et al. 1988). PeMoV is easily transmitted by infected seed and sap at the rates ranging from 0 to 8.5%. PeMoV is spread by *Aphis craccivora*, *A. gossypii*, *Hyperomyzuslactucae*, *Myzus persicae*, *Rhopalosiphum padi*, and *R. maidis*in a non-persistent mode (Paguio and Kuhn 1976; Highland et al. 1981). In Georgia yield losses because of this virus infection were approximated up to 20–70% (Kuhn and Demski 1975), and in India losses may be observed upto 40% in susceptible cultivars.

Peanut yellow mosaic caused by cucumber mosaic virus (CMV) is capable of causing yield losses of upto 40%. CMV, a type species of the genus Cucumovirus and belongs to the family, *Bromoviridae.* Chlorotic spots and rolling of younger leaflets are symptoms of the infection. These spots further coalesce and form large blotches of yellow colour. The leaf lamina of subsequently formed younger leaflets shows yellowing, with green lines running down the lateral veins. The virus is promptly sap transmitted by many aphid species such as *Macrosiphum euphorbiae* in a non-persistent way. Further, it is also observed to be transmitted via the infected seed upto 2–4% (Xu and Barnett 1984). The CMV-CA isolate is peanut seed transmissible and thus the initial spread is probably initiated through the seed-infected with virus. Aphids may play role in secondary spread of virus in peanut fields.

Peanut stripe is caused by PStV, a potyvirus. The characteristic symptoms of a viral disease are intermittent stripes and green bands along lateral veins of peanut leaflets. Striping, mosaic as green islands, and pattern of oak leaf kind can be seen on older leaflets. The plants that have been infected have slightly stunted growth (Demski et al. 1984). Some isolates also result in localized death of tissues on leaves. This leads to stunted growth, severe mosaic patterns and systemic distortion of foliages or stripes symptoms (Chang et al. 1990). The virus is transmitted by sap and is also transmitted through seed up to 37%. Aphids namely, *Myzus persicae*, *Aphis craccivora* and *A. gossypii* transmit the virus in a non-circulative and non-persistent manner.

Shortening of petioles, reduction in the size of leaflets, chlorosis, malformation, and extreme dwarfing of one or more branches or the whole plant are all symptoms caused by the potato stunt virus (PSV). The virus, which belongs to the cucumovirus family, has the potential to cause losses of up to 75%. PSV is spread by three species of aphid namely, *M. persicae*, *A. craccivora* and *A. spiraecola*, by sap inoculation and nature of transmission is non-persistent. It is also transmitted by seeds at the lowest possible frequency of 0.01–0.2% (Xu et al. 1986).

Management

Controlling the virus disease requires cultural practices such as uprooting of all volunteer plants and non-harvested seeds that are infected, sowing of early maturing varieties, manipulating sowing dates, using high-quality pre-treated seed, high seeding rate, and maintaining optimum plant stands. Since, TSWV and PBNV have such wide host ranges, as well as vectors capable of sustaining virus infection and supporting thrips vector multiplication (Reddy et al. 1983), it is not practicable to manage the disease by killing weeds and volunteer peanuts (Reddy et al. 1983). When one row of a fast-growing cereal crop like maize, jowar, or bajra is intercropped with every three rows of peanuts, disease occurrence is reduced (Reddy 1998). Repeated cultivation of dicots and fortuitous hosts like peanut, cowpea, and pigeonpea is likely to reduce the inoculum in the soil (Legreve et al. 1999; Delfosse et al. 2002). Early sowing of the peanut crop prior to monsoon arrival, use of pearlmillet as a bait plants to minimize the inoculum burden in the soil, sowing of peanut during the post-rainy season, avoiding rotation with highly susceptible cereal crops such as maize and wheat, and soil solarization can all help to reduce the incidence of peanut clumps. The initial or early spread of the PeMoV virus is aided by low-level transmission via the infected seed of a few grain legumes (cowpea, mung bean, common bean) as well as peanut (0–8.5%). In nature, substitute crops such as soybean, cowpea, navy bean, clover, peas, French bean, white lupine and weeds (*Desmodium*, *Cassia* spp.) as well as aphids help the virus survive and spread (Demski 1975). The incidence of the virus in young peanut fields appears to be very low (<1%). As the crop reaches maturity, the disease progresses to nearly 80% under congenial conditions that favor vector activity in the fields. So, use of virus-free seed for palnting is important to avoid the disease. Planting should be done with seed lots collected from disease-free areas, as seed is the primary source of PStV virus inoculum. In order to regulate the spread of PStV, the production and subsequent use of virus-free seed should be prioritized. Only certified seeds are permitted to be transported within or outside the countries. The use of plastic film for mulching peanut fields in China is reported to lessen PStV incidence.

Pesticides to reduce vector populations of viruses are available but only little success is achieved. Insecticidal control of thrips vectors is largely ineffective for suppressing spotted wilt in peanut (Culbreath et al. 2003). The use of some insecticides (imidacloprid) was found to increase the disease incidence. Aldicarb, acephate and carbofuran were found to be ineffective. However, chlorophyrifos and phorate (furrow application) reduced spotted wilt in peanut and phorate application is used commercially in the US.

4.2.4 Nematode Diseases

Nematodes are microscopic unsegmented roundworms found in soil. The species of nematodes that cause the most damage to peanuts are peanut root-knot nematodes, root-lesion nematodes, and peanut pod nematodes.

Stages and extent of damage

Among nematodes, the highest loss in peanut is caused by root-knot nematodes *i.e.*, *Meloidogyne arenaria*, *M. javanica*, *M. hapla* and *M. incognita.* Root-knot nematodes result in root galls due to internal swelling of roots and pegs, limit the development of *Rhizobium* nodules, and increase attack by other soil-borne pathogens. Infected pegs and pods may also form galls. Infected plants also exhibit stunting and chlorosis to varying degrees. Root growth is slowed, and vascular elements are disturbed, resulting in poor nutrient and water uptake and transport. Egg masses, infective second-stage juveniles, and adult males of root-knot nematodes can all be found in the soil. Infectious juveniles emerge from the eggs and enter roots, pegs, or pods, moving intercellularly and intracellularly to a location near vascular tissue (McSorley et al. 1992). Under favorable environmental conditions, sedentary juveniles either form males of 1–2 mm length or globose-pyriform shaped mature females that lay large numbers of eggs (about 200–1500 from each female) in a gelatinous matrix. These masses of eggs can either be retained in the roots or squeezed out into the soil. The new second-stage juveniles from hatchecd eggs enter into the soil around the roots. Peanut root nematodes cause yield losses ranging from 20 to 90%. *Pratylenchus coffeae* (Godfrey) Filipjev & Schuurmans-Stekhoven and *P. brachyurus* (Zimmermann) Schuurmanns-Stekhoven (Boswell 1968) are two species of lesion nematodes that target peanut (Chhabra and Mahajan 1976). Lesion nematodes have six life stages, like all nematodes: an embryo, four juvenile stages, and an adult stage and produce. These nematodes are endoparasites that invade the pegs, roots, and pods of peanuts and produce necrotic root lesions and pod lesions followed by discolouration. The infection of pegs also leads to necrotic lesions. The pegs are weakened as a result of these lesions, and pods are shed prematurely. The percentage of sound mature seeds, seed weight, and kernel quality can all be affected by root-lesion nematodes. So, losses results from decreased pod yield and poor yield quality.

Peanut pod nematode (*Ditylenchus africanus* Wendt) is a migratory endoparasite prevalent in limited regions of the world (De Waele et al. 1989). The nematode reaches peanut pegs at the point of pod's attachment and passes through the hull. The nematode reproduces in the hulls and seeds before they are harvested. Approximately 90% of the population of nematode existing within or around a plant is carried inside the pods when they are harvested (De Waele et al. 1989; Basson et al. 1993). A gray, bruise-like soiling of the pod at the point of peg attachment is the first apparent symptom. Premature germination occurs in up to 25% of seeds. The weight of the seeds can also be decreased by 20–50%. The most significant economic effect is the crop's decreased market value as a result of discolored seed (Venter et al. 1991).

Management

Meloidogyne species are holo parasites, and without a host, their populations rapidly decline. Peanut rotation with crops such as maie, cotton, sorghum, and some soybean cultivars will significantly reduce root-knot nematode infestation in soils. Cotton, velvet bean (*Mucuna deeringiana)* and Bahia grass *(Paspalum notatum)* are excellent rotational crops. In addition, since many weeds act as suitable hosts, weed management and volunteer plant eradication are required for a rotating plan to be successful (Taylor and Sasser 1978; Rodríguez-kábana and Canullo 1992; Rodríguez-kábana et al. 1994). However, crop rotation with nonhost crops offers limited success to manage lesion nematode populations, since most *Pratylenchus* species have wide range of hosts that include both dicots and monocots. Nevertheless, crop rotation with the non-host crop *i.e.*, maize reduce the nematode population significantly. The use of nematode- free seed and field-sanitation are important measures. Farmers in *D. africanus*-infested fields are advised to harvest their crops early (Venter et al. 1992).

The fumigant nematicides such as dibromochloropropane (DBCP), ethylene dibromide (EDB), 1,3-dichloropropene (1,3-D) and metham sodium are very effective for the control of root-knot nematodes. Non-fumigants and systemic nematicides that is available for use in peanut are- aldicarb, carbofuran, ethoprop, fensulfothion and phenamiphos (Rodríguez-kábana and King 1985). Phenamiphos at sowing time, aldicarb at sowing or peg formation stage, and oxamyl at peg forming stage are among the registered chemicals for use against the peanut pod nematode (McDonald and Van Den Berg 1991).

Viruses, bacteria, fungi, non-related nematodes, insects, mites, and protozoa, are among the microorganisms and invertebrates that target nematodes. *Pasteuria penetrans*, is one obligate parasite of root-knot nematodes found in many peanut fields. *Arthrobotrys* species and *Monacrosporium* species are the nematophagous fungi that have the potential to control *D. africanus* (Swart and Jones 1994*)*.

4.2.5 Insect-Pests

The important insect pests of peanut are aphids (*Aphis craccivora* Koch), many species of thrips (*Frankliniella fusca*, *F. schultzei*, *Thripspalmi*), jassids (*Empoascakerri* and *E. fabae*), leaf miner (*Aproaeremamo dicella*), red hairy caterpillar (*Amsacta albistriga*), and *Spodoptera.* Aphids, thrips and jassids are sap-sucking pests and also carriers of major viral diseases (Fig. 4.7). Termites and white grubs may also cause significant damage to peanuts (Figs. 4.8 and 4.9). Despite the fact that many insect species have been found in the peanut crop, only a few cause major damage and yield losses. Insect pests are responsible for 10–20% of crop losses in general.

Fig. 4.7 Peanut plant infected with sucking pest

4.2.5.1 Sap Sucking Pests

Stages and extent of damage

Peanut aphid, *Aphis craccivora* (Koch), is one of the most serious and injurious pests of peanut of order Hemiptera, with a worldwide distribution. The aphid is ovoviviparous; females retain eggs inside their bodies and give birth to small larvae. Males are alate and sexual form. Crop losses are caused by *A. craccivora* either directly or indirectly, mainly through the transmission of plant viruses. *A. craccivora* attacks plants at their seedling stage, vegetative stage, and reproductive stage. Aphids tend to feed on immature pods, shoots, young and tender leaves, and fruits. The highest losses in yield due to direct damage are incurred when aphid colonies target developing tips of plants in the spring. Large numbers of aphids feeding directly on peanuts can cause partial sterility of the plants (Mayeux 1984). Peanut yield losses

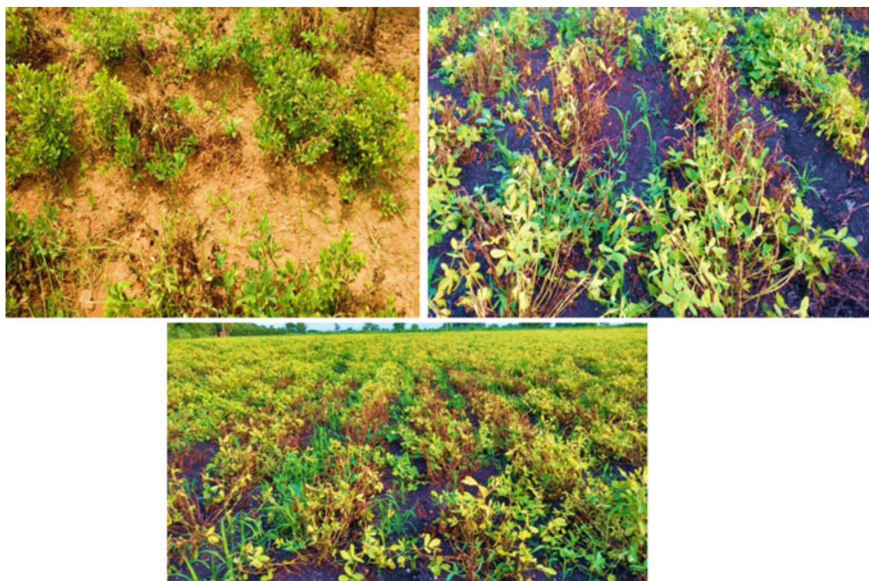

Fig. 4.8 Peanut crop damaged by termite

Fig. 4.9 Peanut pods damaged by termite

of 16% have been reported in India due to insect pests, the most common of which is *A. craccivora* (Jagtap et al. 1984). The development of honeydew, which serves as a substrate for growth of fungus, and the spread of plant viruses such as peanut rosette, peanut (peanut) mottle, and peanut stunt viruses cause indirect damage from *A. craccivora.* Thrips, from order Thysanoptera are small in size (less than 2 mm long) and slim insects having fringed wings that live in the flowers and folded leaflets of peanut plants. The most important thrips on peanut are *Scirtothrips dorsalis*, *Thrips palmi* and *Frankliniella schultzei* (Amin 1985; Ekvised et al. 2006). They are hemimetabolous insects that go through four stages: embryo, larvae, nymphs (two nymphal, and the 'prepupal' and 'pupal' instars), and adult. Adults and larvae are mobile, and adults have wings of their own (Lewis 1997). The sap is sucked from the surface of the leaflets by nymphs and adults. This causes white patches on the upper surface of the leaves, known as silvering, and necrotic patches on the lower surface, known as necrotic patches. As the leaflets expand they split as newly developing leaflets are distorted due to formation of patchy necrotic areas that puncture eventually. Seedlings are often injured. Thrips are vectors for many viruses like PBNV, TSWV, and stem necrosis virus, all of which can lead to widespread yield loss. Jassids (leafhoppers) are another important foliage-sucking pest of peanut and act as limiting factors in the successful cultivation of the peanut crop. *E. kerri* Bachlucha is the most common jassid that attacks peanuts in Asia, and it can be found in abundance in western India, mainly Gujarat. In Africa, *E. facialis* and *E. dolichi* are common jassid species on peanut, and *E. fabae* is widely distributed in the Americas. Both the nymphs as well as adults suck the sap from the tender leaf and mostly from the lower surface of the leaflet causing whitening of the veins, yellowing in the form of patches of the leaflets, leaf curling and necrosis (necrosis of leaf tips in V shape known as hopper burn), stunted growth and eventually death of plants. Jassids also act as a vector of leaf curled, tomato spotted and other viruses (Amin and Palmer 1985; Singh et al. 1990).

Management

Early and dense sowings are highly recommended to control aphids. Early sowings enable plants to initiate flowering before aphids' arrival, while dense sowings provide a barrier to aphids entry into the field (Mayeux 1984). Sanitary measures are important within crops and between seasons to prevent the transmission of viruses by *A. craccivora.* Virus-infected plant materials should be eliminated after harvest and any volunteer plants or weeds that harbour viruses should be destroyed. Thrip populations in peanuts can be substantially reduced by cultural practices. Lower thrip densities are achieved by manipulating sowing dates to avoid peak thrips dispersal and during the susceptible seedling period (McKeown et al. 2001; Culbreath et al. 2010). Likewise, heavy plant residue from conservation tillage systems, increased plant density and twin-row planting reduces thrips infestation on peanut (Brown et al. 1996; Culbreath et al. 2008; Tubbs et al. 2011).

The insecticides such as chlorinated hydrocarbons, organophosphates, carbamates, and pyrethroids have all been used against *A. craccivora.* Systemics that have a high level of persistence during the plant's growth stage are favored. Furthermore,

neem formulations have been shown to be effective against *A. craccivora*, making them a viable alternative to use of insecticides (Egho et al. 2009; Baidoo et al. 2012; Chaudhari et al. 2015). The most regulary used category of insecticides against thrips are carbamates, neonicotinoids, organophosphates, phenylpyrazole and pyrethroids (Todd et al. 1996; Mandal et al. 2012; Marasigan et al. 2016; Srinivasan et al. 2017). Insecticides from newer groups, such as diamides and spinosyns have also been discovered to be effective against thrips (Marasigan et al. 2016, 2018). Seed treatment with Imidacloprid protects for almost a month against sucking pests. If more than 10% of leaves have the typical 'hopper burn' symptoms of thrips, dimethoate can be sprayed during the initial crop development, which is up to 30 days after emergence. However, chemicals should not be used indiscriminately and should be used depending on the economic threshold level of insect population. In India and Africa, coccinellids, *Cheilomenes sexmaculata*, is recommended as a significant natural agent in peanuts (Agarwala and Bardhanroy 1999). Release of the reduviid predators namely, *Rhynocoris marginatus* (Sahayaraj and Martin 2003), *R. kumraii* (Sahayaraj and Ravi 2007), and *Chrysoperla zastrowi sillemi*, a chrysopid predator (Baskaran and Rajavel 2013) and spraying fungus *Verticillium lecanii* reduced populations of *A. craccivora* in Indian fields of peanuts (Sahayaraj and Namachivayam 2011).

4.2.5.2 Foliage Feeders or Defoliators

Many leaf eating insects species are found in peanut crop, of which *Spodoptera*, hairy caterpillar and leaf miner are of economic importance.

Stages and extent of damage

Spodoptera litura (Fab.), tobacco caterpillar/tobacco armyworm and *Spodoptera littoralis*, cotton leaf worm are the two dominant leaf worm species. The adults are light brown moths and lay eggs in group of hundreds, primarily on the upper leaf surfaces. There are six larval instars, which disperse from egg batches. Larvae are regarious feeder and eat leaves, bulbs, and fruits, and are considered a significant defoliator. As a result, *S. litura* is one of a number of pests that can be problematic during the peg initiation stage, pod development stage, and maturation stages of crop growth (Singh and Sachan 1992). The red hairy caterpillar, *Amsacta albistriga* Walk. *and Amsacta moori* Butler, are the most common hairy caterpillars that target peanuts. At the start of the southwest monsoon, the brownish white adults emerge from the soil. They eat all plant bits, including buds, flowers, and leaves and are voracious feeders. They often move from one field to another for food after destroying the vegetation and hatching in one field, resulting in a significant reduction in yield. Peanut leaf miner, *Aproaerema modicella* (Deventer) is a usual pest of peanuts in South and South-East Asian contries and a major pest of India. Young larvae dig into the leaves of hatcheries, depositing single gleaming white eggs on the underside of the leaflets. There are five larval instars stages and pupation takes place inside webbed leaves. For peanut, yield losses of >50% have been reported due to feeding

on the leaves (Islam et al. 1983). From a point, a heavily attacked field appears to be 'burned,' and epidemics can result in complete crop loss.

Management

To expose pupae of *Spodoptera* to natural enemies and adverse weather-related factors, clean cultivation and deep plowing are recommended. Sunflower, taro and castor plants allure *Spodoptera* and thus, may be sown to collect egg masses and larval instars both around and within fields, as trap crops (Zhou 2009). Light traps or pheromone traps can be used to collect moths of defoliators. Crop rotation with sorghum, pearlmillet or maize should be followed. The migration of larvae of red hairy caterpillar can be avoided by digging deep trenches. To reduce the larval densities of leaf miner intercropping of peanut with sorghum, millet or cowpea is preferred. Also, cotton-sorghum-peanut is the best crop rotation combination to give better yields and reduce the incidence of leaf miner. Removing the alternative hosts and weeds viz., lucerne, amaranthus, berseem and *Indigofera hirsuta* can be effective to control the growth of the leaf miner population.

S. litura and other defoliators have gained resistance to most of the available pesticides used commercially (Ramakrishnan et al. 1984; Naeem Abbas et al. 2014), so control is becoming increasingly difficult, although, spraying of dimethoate, fenthion, phosphomidon, Imidacloprid, carbaryl, dichlorovos, and Quinalphos, is practiced. Chlorantraniliprole, spinosad, and emamectin benzoate, are among other new chemicals that have shown optimistic results against *S. litura* (Gadhiya et al. 2014). When adult stage of leaf miners is discovered in the attacked area, fruit powder extract of neem can be used to effectively reduce oviposition. Insecticides, ideally dimethoate or imidacloprid can be used.

Telenomus remus, egg-larval parasitoid and larval parasitoid species namely, *Apanteleruficrus*, *A. kazak*, *Cotesia marginiventris*, *Campoletes chloridae*, and *Hyposoterdidymator* are some biological controls reported but have varying efficiency (Braune 1982; Michael et al. 1984). *Trichogramma* parasitize on eggs and young larvae of red hairy caterpillar. Spraying of bioinsecticides based on Nuclear Polyhedrosis virus (NPV) or *Bacillus thuringiensis* can manage spodoptera effectively.

4.2.5.3 Root and Pod Feeders

Stages and extent of damage

White grub species, *Lachnosterna* (=*Holotrichia) consanguinea* (Blanch.) and *L. serrata* are the two most important soil inhabiting polyphagous pests of peanut. Adults are dark brown and emerge out of the soil within 3–4 days after the onset of rain. The eggs are white and round in shape, while larvae are whitish yellow in colour, fleshy and C-shaped. The young grubs in their second, third and fourth instar larval stages feed on organic matter and fine rootlets while mature grubs feed on both roots and pods. Wide patches of dead plants can be found in heavily infested

fields, and the remaining plants are often stunted and wilting. The damage to peanut crops in endemic areas varies from 20 to 80%. Peanut plants are harmed by termites, mostly *Microtermes* spp. and *Odontotermes* spp. They burrow within the root and stem, killing the plant; they make holes in the pods, damaging the kernels; and they cause scarification (stripping of the soft corky tissue between the pods veins). As a result, pods are more vulnerable to *Aspergillus* species infection.

Management

Summer ploughing exposes the pupae to scorching solar radiation and predation by birds. Crop rotation with sorghum and pearl millet, early sowing, and use of light traps and pheromone traps should be practiced. Clearing mounds of termites around peanut fields and injecting chlorpyriphos into the termite mounds are two cultural operations that can effectively reduce termite populations in cropping areas. Termite control was also found to be successful when peanuts were harvested at the optimum maturity stage and debris was removed from the field. Although, soil insects are expensive and difficult to manage insecticides namely, carbofuran, chlorpyrifos and phorate can be incorporated in soil prior to sowing and seed treatment with chlopyriphos and imidacloprid can be practiced.

4.3 Genetic Resources and Trait Discovery

Genetic resources are important sources of variability and serve as repository of many desirable alleles for current and future programmes for peanut improvement. Genetic variability preserved in gene banks are important sources of variability and harbor many useful genes for utilization in breeding programs. Thousands of peanut accessions are conserved in national and global gene banks around the world, including ICRISAT, the United States, Brazil, India, and China, where biotic stress variations can be seen (Ntare et al. 2006; Pandey et al. 2012a, b). Furthermore, cultivated peanut accessions, gene banks have a large number of wild peanut accessions. Since cultivated peanuts are the result of a single hybridization among diploid ancestors, they have a narrow genetic base and genetic variability in response to biotic stresses. Wild *Arachis* species, on the other hand, have been reported to have higher tolerance/resistance to a variety of stresses (Figs. 4.10 and 4.11). In addition, several interspecific hybridization lines have been established to create new variability (Fig. 4.12), and some improved varieties have also been released. The genus *Arachis* has 80 species (Valls and Simpson 2005). Initially, Krapovickas and Gregory in the year 1994 grouped the genus *Arachis* into nine sections based on cross compatibilities, morphology, phylogeny and geographic distribution namely, *Arachis* with 31 species., *Erectoides* 14, *Extranervosae* 10, *Procumbentes* 10, *Rhizomatosae* 4, *Heteranthae* 6, *Caulorhizae* 2, and *Trierectoides* 2 and *Triseminatae* with single species. The *A. hypogaea*, a cultivated and tetraploid peanut, *A. monticola*, another non-cultivated tetraploid species, and 29 diploid species make up the *Arachis* section.

Fig. 4.10 Wild *Arachis* sp. maintained in field conditions

Fig. 4.11 Wild Arachis sp. resistant to foliar fungal diseases

Fig. 4.12 Synthetic amphidiploid maintained under field conditions

Genetic diversity in the peanut is grouped into different gene pools as suggected by Singh and Simpson (1994). Breeders benefit from the idea of gene pools because it helps them choose germplasm to use in hybridizations to widen the genetic base of crop and enhance the crop's genetics. Landraces and typical cultivars of peanut from 1° as well as 2° centres of genetic diversity, along with wild *A. monticola*, make up the primary gene pool (GP1). Hybridization within the GP1 results in routine chromosome pairing and thus, fertile progeny, so gene transfer from GP1 to *A. hyogaea* is easy. The secondary gene pool (GP2) consists of diploid species of the *Arachis* segment that are congenial in cross with *A. hypogaea* but contain sterile to partly fertile hybrids because of ploidy variations. The tertiary gene pool (GP3) consists of species from section *Procumbentes*, which are compatible in cross with diploid species of *Arachis* section (Mallikarjuna 2005; Mallikarjuna and Hoisington 2009), section *Erectoides*, whose species have low cross-compatibility with and *A. hypogea* (Singh 1998); and *Rhizomatosae*, whose tetraploid species can be crossed both with diploid species of section *Arachis* and *A. hypogea* (Gregory and Gregory 1979; Mallikarjuna and Sastri 2002). The remaining *Arachis* species that are incompatible or weakly compatible with *A. hypogaea* and other *Arachis* species are included in the Quaternary Gene Pool (GP4). The most open sources GP1 and GP2, which have been successfully used in peanut improvement, and their probable benefit

is now much more efficient and predictable. However, the use of biotechnological techniques is needed to exploit tertiary and quaternary gene pools. The use of GP1 for many traits has been restricted, and wild *Arachis* species have frequently shown desired variability and a higher degree of resistance than GP1. For example, in the case of PStV, despite screening 9000 accessions, no resistant source was established in cultivated peanuts, but a negative reaction was observed in many wild *Arachis* accessions (Culver et al. 1987; Prasada Rao et al. 1991). Wild *Arachis* spp., such as *A. batizocoi*, *A. correntina*, *A. cardenasii*, *A. duranensis*, *A. diogoi*, *A. pusilla* and *A. villosa*, have higher resistance and tolerance to peanut-rust (Abdou et al. 1974; Subrahmanyam et al. 1982a, b; 1985a, b, c), but their pods are catenate and small. Many wild species from the *Arachis* section that are cross-compatible with the cultivated species displayed either an immune response or highly resistant response to the late leaf spot pathogen, including *A. diogoi*, *A. cardenasii*, *A. glabrata*, *A. stenosperma*, *A. repens*, *A. appressipila*, *A. paraguariensis*, *A. villosulicarpa* and *A. hagenbeckii*, were among the highly resistant species found in other sections (Subrahmanyam et al. 1985a, b, c). Further, several resistance sources to ELS were identified in *A. hypogaea* and two diploid wild species, *A. stenosperma* and *A. diogoi* were also scored as highly resistant (Foster et al. 1981). Also, considerable genetic variation for virus resistant was found in wild species. *A. cardenasii A. diogoi*, *A. correntina*, and *A. pusilla* showed no infection to TSWV under field conditions. Two species namely, *A. diogoi* and *A. pusilla* also exhibited no infection from Peanut mottle virus (Subrahmanyam et al. 1985a, b, c; Demski and Sowell 1981). Both reproductive resistance and hypersensitive necrosis to *Meloidogyne* spp. have been reported recently in tetraploids derived from complex crosses of *A. hypogaea* (Nelson et al. 1989; Holbrook and Noe 1990) comprising of three species viz., *A. batizocoi*, *A. cardenasii*, and *A. diogoi* Hoehne that are resistant to nematode. There was considerable variation for resistance in different accessions of wild species *(*Sharma et al. 2003*)*. *A. batizocoi*, *A. diogoi*, *A. correntina*, *A. villosa*, *A. spegazzini*, *A. cardenasii*, *A. stenosperma*, *A. duranensis*, *A. rigonii*, *A. paraguariensis*, *A. pusilla*, *A. glandulifera*, *A. ipaensis* and *A. repens* are species that possess resistance to thrips (Yang et al. 1993; Michelotto et al. 2017; Srinivasan et al. 2017). *A. cardenasii*, *A. duranensis*, *A. kempff-mercadoi*, *A. monticola*, *A. stenosperma*, *A. paraguariensis*, *A. pusilla*, and *A. triseminata* showed multiple resistances to the leaf miner and thrips. *A. cardenasii*, *A. appressipila A. ipaensis* and *A. paraguariensis* showed antibiosis to *Spodoptera* and also resistance to leaf feeding (Sharma et al. 2003).

Fertility obstacles triggered by species incompatibilities and ploidy level differences; association of desirable traits with traits that are agronomically unadapted and undesirable; and monitoring introgressed segments have all hampered the transfer of genes from wild species. Many methods are being used for the introgression of wild genes in cultivated peanut with varied success of which the hexaploid and tetraploid routes are most successful. In the hexaploid route, a triploid hybrid derived from a cross between the cultivated allotetraploid species and the diploid wild species is colchicine treated to produce a hexaploid plant, followed by generations of selfing to select tetraploid plants with resistance to multiple disease resistances (Stalker et al. 1979; Stalker and Beute 1993; Reddy et al. 1996). In tetraploid route as suggested

by Simpson et al. (2001) firstly, an A genome hybrid was made by crossing *A. cardenasii* with *A. diogoi*. Then, the B genome species *A. batizocoi* was crossed with the A genome hybrid to create a sterile AB hybrid. This sterile hybrid was treated with colchicine to double the chromosome number and restore fertility. This tetraploid, also known as amphidploid [*A. batizocoi* × (*A. cardenasii* × *A. diogoi)*], was registered as TxAG-6, that has a strong resistance to nematodes and later used as a source in breeding two cultivars, COAN and NemaTAM. More recently, amphidiploids were developed using *A. duranensis* and *A. ipaensis* (Fávero et al. 2006) and *A. gregoryi* and *A. linearifolium* (Simpson and Starr 2001; GCP 2005; Simpson et al. 2003). Further, considering the potential use of amphidiploids ICRISAT has developed many tetraploids and amphidiploids peanuts using wild species. Synthetic amphidiploids, such as ISATGR 278–18 (*A. duranesis* × *A. batizocoi)* and ISATGR 5B *(A. magna* × *A. batizocoi*), were developed by ICRISAT and have been used in backcross breeding program to transfer useful genes into elite cultivars/genotypes that possess many traits of interest, including resistance to foliar diseases (Kumari et al.2014). The sterile diploid hybrids from *A. magna* V 13,751 and *A. kempff-mercadoi* V 13,250 were treated with colchicine for polyploidization, and the amphidiploids were crossed with *A. hypogaea* cv. IAC OL 4 to initiate the introgression of the wild genes for pest resistance into the cultivated peanut (de Paula et al. 2017). Furthermore, the release of an Indian variety (GPBD 4) with foliar disease resistance due to chromosome segments from *A. cardenasii* is an example of achievement from wide hybridization. Further, with the advent of marker technologies and biotechnological tools, prebreeding activities have been accelerated. Molecular markers are being used to test hybridity, to characterize the introgression lines for wild genes and molecular diversity analysis. To overcome the problems of barriers between the cultivated species and the wild species and to get rid of undesirable gene blocks genetic engineering techniques would be an ideal option in peanut improvement.

4.4 Conventional Breeding Methods for Biotic Stresses Resistance

Many of the biotic stresses can be controlled to a lesser degree by adopting appropriate cultural practices and chemical control measures. However, farmers can afford to use very little pesticides in general and still less for controlling biotic stresses. So, using disease-resistant cultivars is one of the most effective and cost-effective ways to reduce disease-related crop losses. Peanut breeding for biotic stresses involves the identification of sources of resistance either from existing variability in cultivated germplasm accessions, from wild *Arachis* species or creating new variability by mutation breeding and their introgression into elite genotypes. This approach has resulted in the development of many disease resistant cultivars coupled with higher yield. Availability of potential donors, understanding of genetic control of resistance and proper screening methods are prerequisite to begin any disease resistance breeding

program. The general approach includes the screening of germplasm, crossing and development of hybrids, and effecting selections in segregating generations advanced through pedigree, bulk method, single seed descent, backcross or their modifications. The pedigree method enables breeders to concentrate on high-heritability traits, while the bulk-pedigree methodology, a simplified variant of the bulk method aimed at enhancing traits with low heritability (Wynne and Gregory 1981). The single seed descent method is gaining popularity because it saves both space and money (Isleib et al. 1994). In 1927, a Dutch scientist from East Java (Indonesia), made the first effort to use genetic resources to order to develop a disease resistant peanut and as a result, Schwarz 21, a variety resistant to bacterial wilt was developed (Buddenhagen and Kelman 1964). Despite these early achievements in leveraging host-plant heterogeneity, biotic stresses resistance breeding was not given much attention until the late of 1970s. Most of the resistant germplasm lines against foliar fungal diseases are primeval and land races that have unwanted pod and kernel characteristics. Rust resistance sources presently used by peanut breeders have factors for "slow rusting" and reported to have either recessive inheritance or dominant with duplicate recessive or partial dominant, or polygenic inheritance. Some sources of rust resistance governed by a few major genes are relatively easy to transfer into agronomically adaptable and desirable types. GPBD 4 is a most popular rust-resistant variety produced at UAS, Dharwad, from the parental genotype ICGV 86855, which is an interspecific derivative derived from cross, *A. hypogaea* × *A. cardenasii* (Stalker 1997). Some tetraploid lines or nearly-tetraploid lines originated from crosses of cultivated allotetraploid peanuts with wild *Arachis* species have shown a high level of resistance to ELS and LLS (Subrahmanyam et al. 1985a, b, c). Genetic resistance shows complex inheritance and factors including initial infection, sporulation, size of lesions, and defoliation, all play a role (Green and Wynne 1986; Chiteka et al. 1988a, b; Anderson et al. 1993; Waliyar et al. 1993, 1995). Rate-reducing resistance to leaf spots is quantitative and governed by both additive and non-additive gene effects along with maternal effects (Anderson et al. 1986a; Dwivedi et al. 1993). Some of the released cultivars that are tolerant to early leaf spot (ELS) in India and USA are BG 3, Bailey, C-99R, CSMG 84-1, DP 1, GG 7, Florida 07, Georganic, ICGS 44, M 335, ICGS 76, M 522, Prutha, Somnath, Sugg and VA 81B. LLS tolerant cultivars released from India are ICGV 86590 and ICGV 86325, ICG (FDRS) 10, Girnar 1, K 134, GBPD 4, ALR #s 1, 2, and 3, BSR 1, R 8808, VRI (Gn) 5, CSMG 84-1 and RG 141. In the USA, C-99R, Florida 07, Florida MDR 98, Southern Runner, TUF Runner, TM '727', and others were released (Gorbet et al. 1987).

In order to integrate resistance to both leaf spots in a single line, two strategies are being used. Selecting for LLS resistance among germplasm lines that has already been screened for ELS resistance is one approach. A strategy is to combine individual sources for resistance to LLS and ELS in a single cultivar. Genes for resistance to LLS and ELS are inherited singly and can be consolidated into a single genotype (Kornegay et al. 1980; Anderson et al. 1986b). Multiple foliar fungal disease resistant cultivars namely, ALR 1, ALR 2, DOR 8-10, Girnar-l, GPBD4, ICGS (FDRS) 10, and ICGV 86590 were developed in India but are not popular because of poor kernel and pod characteristics. Partially resistant cultivars can also be cultivated to decrease

the inocula build up and rate of spread of leaf spot epidemics, but this resistance is not complete and stable (Subrahmanyam et al. 1982a, b).

Resistance to soil-enduring fungi is difficult to breed for, and progress has been slow. Until recently, low to average levels of resistance to stem rot is reported in peanut germplasm. To date, resistance to soil-borne fungus is attributed to polygenic with minor but additive effects (Fry 1982), and is thought to be similar to horizontal or field resistance. However, integrating this form of field resistance into germplasm with desirable agronomic traits has proven difficult. If soil-inhabiting fungus of peanut is to be controlled using the available sources that is incomplete, extensive cooperative breeding and pathology research is needed. Peanut cultivars viz., Virginia 81B, Virginia 93B, Southwest Runner, Tamspan-90, and Tamrum OL07 possess considerable resistance to pathogen, *S. minor* (Akem et al. 1992; Baring et al. 2006). Some cultivars in USA are known to show partial resistant to *S. rolfsii* namely, Southern runner, Toalson, Pronto, Tamrun 96 and Georgia Browne (Simpson et al. 1979; Banks and Kirby 1983; Gorbet et al. 1987; Branch 1994; Smith et al. 1998; Backman and Brenneman 1997). Moderately resistant cultivars such as VA-98R, VA 93B, and Perry are being utilized commercially (Chappell et al. 1995). Certain peanut lines have been confirmed to have high production potential along with average resistance to *Pythium* spp. Georgia Browne, a runner peanut, has been found to have partial resistance to *R. solani.* Resistance to both *Pythium* spp. and *R. solani* may be found in Spanish cultivars, mainly Toalson (Beaute 1997; Brenneman 1997).

Preharvest resistance, resistance by seed coat against *invitro* seed colonization (IVSC), and cotyledons aversion to aflatoxin formation are all independently inherited resistance mechanisms against *Aspergillus flavus*, provide future achievement from gene pyramiding (Upadhyaya et al. 2002). But to date, no effective efforts have been made because the genetics and mechanisms of resistance are complex and not fully understood. One released variety, J 11 is reported to have resistance to initial infection and subsequent colonization by the fungus *A. flavus*, and this resistance is associated with the hardening of its hypocotyl tissues (Hadwan and Bhowmik 1991; Nayak et al. 1992). Yueyou 9 and Yueyou 20 are *A. flavus* resistant cultivars released from China (Liang et al. 2009). ICRISAT has identified some germplasm with limited resistance in their Minicore collection (Waliyar et al. 2016). The Senegal variety 55-437 is reported to have some resistance (Clavel 2004). More recently, two accessions, Zh.h0551 and Zh.h2150 resistant to aflatoxin production were identified from China's minicore collections (Yu et al. 2020).

Southern Runner' was the first released cultivar of peanut with average resistance to TSWV (Culbreath et al. 1992a, b, 1994, 1996). Further, additional cultivars having TSWV resistance similar to Southern Runner including 'Georgia Browne', and 'Georgia Green' 'C 99R', 'Florida MDR 98' and 'Tamrun 96', were released (Branch 1996; Culbreath et al. 1994, 1996). All currently grown cultivars in the southeastern region of the U.S. have higher resistance to TSWV.

Excellent resistance sources to rosette disease are available in several genotypes from different maturity groups (Bock et al. 1990; Subrahmanyam et al. 1998; Naidu et al. 1999). Subramanyam et al. (2001) have identified several wild *Arachis* species resistant to all the three causative agents of peanut rosette. Resistance to

rosette virus is controlled by a monogenic dominant or two independent recessive genes, so these resistances are relatively easy to transfer into agronomically desirable types (Nigam and Bock 1990; Olorunju et al. 1992). GRD resistance sources were first discovered in Senegal in the year 1952, and subsequently they were used as parents in developing high-yielding, rosette-resistant peanut varieties, RMP 91, RG1, RMP12. In Nigeria, UGA2 (Samnut21), M572.80I (Samnut22), and ICGV-IS96894 (Samnut23), medium duration and resistance to GRD were released in 2001, and following three early maturing varieties with GRD resistant Samnut24, Samnut25, and Samnut26, were released more recently (Ajeigbe et al. 2015). Rosette resistance is successfully introgressed by backcrossing with a commercial cultivar, 28–206(R) (Mauboussin et al. 1970). Also, GBNV resistant peanut cultivars viz., ICGS 11 and ICGS 44 were released in India.

The higher resistance in the cultivar Schwarz 21 to bacterial wilt was first identified in Indonesia. A series of resistant cultivars have been released commercially in China since 1980s (Mehan et al. 1994). Bacterial wilt resistant sources from wild *Arachis* species (Tang and Zhou 2000) and cultivated species (Liao et al. 2005) were used as sources to develop and release resistant peanut cultivars viz., Zhonghua 4, Tianfu 11, Zhonghua 6, and Zhonghua 21 in China (Yu et al. 2011) and in other countries.

Garcia et al. (1996) reported that resistance to nematode in *A. cardenasii* was governed by two genes, dominant in nature, where one gene designated as *Mag*, is responsible for inhibiting root galling and another gene named as *Mae*, is responsible for hindering egg production by nematode, *M. arenaria.* In complex hybrids (tetraploid) of *A. hypogaea* (Nelson et al. 1989; Holbrook and Noe 1990) derived from three species, *A. batizocoi*, *A. cardenasii*, and *A. diogoi* Hoehne, resistant to nematode, both hypersensitive and necrotic cell death and reproductive resistance to *Meloidogyne* sp. have been identified. As a result, the first breeding line (TxAG-7), resistant to *Meloidogyne* was commercially released for cultivation (Simpson et al. 1993). TxAG-7 was originated from a backcross of *A. hypogaea* cv. 'Florunner' with TxAG-6 (Simpson et al. 1993). A backcross program was also used to introduce root-knot nematode resistance from TxAG-7 into Florunner, resulting in the release of 'COAN,' the first peanut cultivar with *M. arenaria* resistance (Simpson and Starr 2001). The resistance in this cultivar was governed by a single gene of dominant nature. Subsequently, introgressing genes from TxAg-6 to *A. hypogaea*, resulted in release of two cultivars, NemaTAM (Simpson et al. 2003) and Webb (Simpson et al. 2013).

When resistance to multiple biotic stresses is needed, it is hard to accumulate enough polygenes, inherited independently with conventional breeding approaches to provide good resistance levels to all diseases. Exceptions to this will happen if the same genes/or set of genes confer resistance to more than one diseases, for example several genotypes resistant to *Pythium* pod rot also shows resistant to *S. rolfsii* (Smith et al. 1989). One successful example is Tifguard, a peanut variety bred with resistance to nematode, root-knot nematode and virus, TSWV released from USA (Holbrook et al. 2008). However, the lack of major or complete resistance sources for biotic stresses may partly be the reason for the slow gain in breeding for disease-resistant cultivars in peanut (Allen 1983).

Due to the difficulty in screening a huge number of germplasm accessions and segregating populations under erratic and variable insect strains, insect resistance breeding has received little attention. Repellent, antibiosis, immunity, physical structures, and avoidance are some of the resistance mechanisms that can be used alone or in combination. Many genotypes with insect pest resistance have also been reported (Nigam et al. 1991). Resistance to thrips and jassids is related to high trichome density, distribution, and length, as well as thick leaf cuticles. Antibiosis works by reducing growth and fecundity in aphid resistant genotypes (Padgham et al. 1990). Resistance against *A. craccivora* was reported in the breeding line, ICG 12991, governed by a single recessive gene (Minja et al. 1999). ICGV 87160 (ICG (FDRS), Serenut 10R, SGV0023, SGV 002, SGV 0053, SGV 0084, Samnut 22 and 23 are released cultivars reported to a have higher yield in leaf miner infested fields. A higher tolerance to leaf miner and *Spodoptera* in a breeding line ICGV 86031 is seen as an enhanced ability of the vegetative tissue to regrow after defoliation (Wightman and Rao 1994).

Traditional breeding programs has been successful in some areas but has failed in others due to a lack of improved and more efficient screening methods and techniques, as well as a lack of knowledge about the underlying mechanisms of resistance. Before starting any breeding program, we need to know about the inheritance/genetics of certain traits. Furthermore, in breeding programs, greater diversification of parental resources is needed to expand the genetic base and produce new cultivars that will perform better under adverse conditions. To access genes from GP3 and GP4 pools, recombinant DNA technology with a cis-transgenic approach must be used. Emerging molecular tools offer a way to improve the efficiency, effectiveness and gain from traditional breeding programs, especially for complex polygenic traits. A comprehensive approach incorporating traditionaland molecular breeding, with transgenics techniques would offer solutions to the complex problems presently confronting the peanut improvement.

4.5 Molecular Breeding in Peanut

Marker-assisted breeding implies the application of molecular markers in combination with genomics tools and techniques to improve traits in the desired direction using modern breeding strategies such as marker-assisted selection (MAS), marker-assisted recurrent selection (MARS), marker-assisted backcrossing (MAB), and genomic selection (GS). For the application of markers in breeding program availability of markers/marker techniques along with dense genetic linkage maps are necessary.

Progress in marker work has been heavily dependent on advances in marker technology. Initially, molecular marker discovery in peanut was focused on proteins and isozymes, followed by rapid progress on discovery of DNA-based markers such as RFLP, RAPD, AFLP, SSR and SNPs. The earlier genomics studies were focused on the use of polymorphic RFLP and RAPD markers for screening interspecific

breeding lines and cultivated peanuts genotypes (Burow et al. 1996, 2001; Subramanian et al. 2000; Dwivedi et al. 2001, 2002b; Garcia et al. 1995). *Eco*RI/*Mse*I and *MIu*1/*Mse*I primer pairs initially observed polymorphisms within cultivated peanut accessions and interspecific tetraploid derivatives in AFLP assays (He and Prakash 1997; Herselman 2003). However, use of these markers is not suitable for the application in MAS. Although RFLP is co-dominating and highly reproducible marker, method is more time consuming, laborious and based on radioactive-based probes. Further, dominant marker RAPD is distributed in whole genome but have less reproducibility. Whereas, assays of STS (PCR-based sequence tagged site markers derived from closely linked RFLP markers) and SCAR (sequence characterized amplified region originated from polymorphic RAPD bands) are more accurate, co-dominant in nature and can be used for high-throughput genotyping (Olson et al. 1989; Paran and Michelmore 1993). Similarly, dominating nature of AFLP can be more suitable for diversity analysis compared to MAP. This marker can be converted into co-dominating markers namely, STS and SCAR (Konieczny and Ausubel, 1993; Negi et al. 2000; Huaracha et al. 2004). Due to multitude characteristics of SSRs (simple sequence repeats) such as reproducibility, polymorphism, multiallelic, genome distribution, co-dominance inheritance, simple assay and transferability across species, SSRs are markers of choice for the molecular breeding (Weber 1990). As a result, several novel SSRs have been found in peanut and utilized in breeding program. In recent years, more than 2500 SSR markers have been produced in peanut using methods such as the construction and subsequent sequencing of SSR-densed genomic DNA libraries, the sequencing and mining of Bacterial Artificial Chromosome (BAC)-end sequences (BES) for repeats motifs, and the mining of transcript sequences developed either by Sanger method of sequencing or more advanced developed next-generation sequencing (NGS) approaches (Mace et al. 2007; Cuc et al. 2008; Gautami et al. 2009; Pandey et al. 2012a, b). Efforts by several researchers to develop SSRs markers for peanut have resulted in more than 9000 repeats (Guo et al. 2016). The degree of polymorphisms in cultivated peanuts, however, remains low. The use of more robust techniques such as SNPs, kompetitive allele-specific PCR or KASPar and genotyping by sequencing (GBS) approaches are required due to the lower genetic variation at molecular level. There have been major developments over the last decade, with the discovery of massively parallel technology, next generation sequencing technology (NGS). Several multiple approaches to bioinformatics, whole genome study using de novo assembly, resequencing have enabled the development of large numbers of SNPs and SSRs (Bertioli et al. 2016). In addition, NGS and data mining have made it easier to discover cost-effective, large-scale generation of EST-SSRs and SNPs (expressed sequence tags) (Pandey et al. 2012a; b; Zhao et al. 2012; Guimaraes et al. 2012; Nagy et al. 2012; Zhang et al. 2012; Bosamia et al. 2015). With the advantages of most abundance and widely distribution of SNP throughout genome, cost efficient SNP genotyping platform are not freely available for the tetraploid peanut and microsatellites are still considered as best choice as markers for tetraploid peanuts because it is co-dominant and easy to score (Pandey et al. 2012a; b). Miniature Inverted-Repeat Transposable Elements

(MITEs) based markers have also been developed in peanut (Bhat et al. 2008; Shirasawa et al. 2012) and a large number of polymorphic *AhMITE*1 markers have recently been identified from the peanut genome re-sequencing data (Gayathri et al. 2018).

4.5.1 Genetic Linkage Maps

The development of genetic mapping populations by crossing genetically divergent parents is the first step in developing linkage maps and the identifying QTLs/genes linked to the trait of interest. Several genetic populations for mapping traits have been developed including F_2 population, $F_{2:3}$ populations, recombinant inbred lines (RILs), backcross introgression lines (BILs), near isogenic lines (NILs), and association mapping populations based on natural populations, nested association mapping (NAM), and multi-parent advanced generation inter-cross (MAGIC) populations (Pandey et al. 2012a, b; Varshney et al. 2013; Janila et al. 2013). Higher levels of polymorphism greatly encourage the development of more saturated genetic linkage maps that form the basis for identifying markers of economically significant characteristics closely linked to governing QTLs. Based on F_2 mapping population derived from *A. stenosperma* (AA) × *A. cardenasii* (AA), the first linkage map of 11 LGs consisting 117 RFLP markers loci was constructed (Halward et al. 1993). Later, population derived from cross between synthetic amphidiploids [*A. batizocoi; BB* × *(A. cardenasii; AA* × *A. digoi; AA*] and cv. Florunner were used to construct linakge map that comprised of 370 RFLP loci on 23 LG (Burow et al. 2001). The first incomplete/partial linkage map based on population derived from cultivated peanutwas made, which had 12 AFLP markers distributed on five linkage groups (Herselman et al. 2004). Further, agenetic 88 BC_1F_1 individuals from cross of synthetic amphidiploids (*A. ipaënsis* × *A. duranensis*) with *A. hypogaea*cultivar Fleur11 was constructed using 298 SSRs loci that distributed on 21 LGs. (Fávero et al. 2006). Thes elow-density maps have minimal use in QTL mapping. Later, several SSR based genetic maps have been constructed by various research groups including 131 SSR loci map distributed on 20 LGs from the population of cross between Yueyou 13 and Zhenzhuhei (Hong et al. 2008), 135 loci on 22 LGs, from a RILs population derived from crossing parents, ICGV 86031 and TAG 24 (Varshney et al. 2009), composite map of 175 SSR in 22 LGs (Hong et al. 2010), 101 SSRs in 17 LGs (Zhang 2011) and integrated composite map of 897 SSRs distributed on 20 LGs was constructed by Gautami et al. (2012b). In a similar vein, two other genetic maps based on RIL derived from TAG24 × GPBD4 (188 SSR loci) and TG26 × GPBD 4 (181 SSR loci) were created and used to generate a 225 SSR loci consensus map (Sarvamangala et al. 2011; Sujay et al. 2012). In addition to these maps, two linkage maps are generated one with 119 SSR loci from the RILs of ICGS 76 3 × CSMG 84-1 and another with 82 SSR loci from RILs derived from cross, ICGS 44 × ICGS 76 (Gautami et al. 2012a) along with consensus linkage map population derived from TAG 24 × ICGV 86031. More recently, Qin et al. (2012) built individual genetic maps consisting of 236 and 172 EST-SSR marker loci, respectively,

from the two RILs populations, one from cross, Tifrunner × GT-C20 and other from cross, SunOleic 97R X NC94022. A consensus map consisting 324 marker loci spanning 1352 cM of genetic distance was then constructed (Qin et al. 2012). Wang et al. (2012) constructed linkage map based on single mapping population with a total of 318 SSRs mined from BAC-end sequences (BES) covering 1674.4 cM map distance. Shirasawa et al. (2012a) used sequence data from the parental lines to mine marker in silico and mapped 1114 loci in 21 LGs. Later, 897 marker loci (895 SSRs and 2 CAPS) were mapped on 20 LGs spanning a total genetic distance of 3607.97 cM, followed by 3693 marker loci mapped on 20 LG with total map distance spanning 2651 cM (Gautami et al. 2012b; Shirasawa et al. 2013).

Nearly all maps, however, constructed using low-throughput markers, including RFLPs, SSRs have produced comparative low density map and are unable to provide reliable information of complex trait. In contrast, the most abundant marker, SNPs was used to construct genetic map for the "A" genome for the first time in 2012. With advent of high-throughput sequencing technologies, different methods have been established to genotype the mapping population of peanut such as restriction site-associated sequencing (RAD-seq) double digest RAD-seq, genotyping by sequencing (GBS) and high density SNPs or insertion/deletions (InDel) (Miller et al. 2007; Peterson et al. 2012; Poland et al. 2012; Zhou et al. 2014; Han et al. 2018). The first genetic map based on SNPs for cultivated peanuts was constructed using ddRAD seq with 1621 SNPs (Zhou et al. 2014). Recently, SLAF-seq technology (specific length amplified fragment sequencing (SLAF-seq) was used to construct high density linkage map in peanut (Wang et al. 2018a, b; Hu et al. 2018). These dense genetic maps would have a greater effect on genetic studies in peanuts and marker-assisted selection programs to improve traits. Table 4.2 provides a list of genetic maps constructed using various molecular markers for the *Arachis* species.

4.5.2 Marker Trait Associations and QTLs Discovery

4.5.2.1 Mapping Populations and Approaches

The two prerequisites for molecular breeding are the discovery of linked markers associated significantly with traits to be improved and the identification of QTLs by genetic mapping. Trait mapping can be done by various approaches including linkage mapping, linkage disequilibrium (LD) based association mapping and joint use of linkage and LD based, linkage-cum- association mapping (JLAM). In linkage mapping, bi-parental populations (RILs, NILs, BILs and $F_{2:3}$) are commonly used however, recent advances in the area of marker trait association, linkage disequilibrium based association mapping like candidate gene-based association (CGAS) and GWAS were also used in natural populations (Zhu et al. 2008). Bi-parental populations have high trait mapping ability, but have disadvantages in being able to have few traits and low resolution with allelic variation. In contrast, association mapping has advantages of use of large number of germplasm to cover huge amount

Table 4.2 Comprehensive list of genetic maps developed in peanut

Populations used	Markers used	No of loci mapped	Coverage (cM)	References
Genome AA				
A. stenosperma × *A. cardenasii*	RFLP	132	1063.00	Halward et al. (1993)
[*A. stenosperma* × (*A. stenosperma* × *A. cardenasii*)]	RAPD, RFLP	206	800	Garcia et al. (2005)
A. duranensis (K7988) × *A. stenosperma* (V10309)	SSR	204	1230.89	Moretzsohn et al. (2005)
A. duranensis (K7988) × *A. stenosperma* (V10309)	SSR, anchor, AFLP, NBS profiling, SNP	369	–	Leal-Bertioli et al. (2009)
A. duranensis (PI 475,887) × *A. duranensis* (Grif 15,036)	SNP, SSR, SSCP, RGC	1724	1081.30	Nagy et al. (2012)
A. duranensis (K7988) × *A. stenosperma* (V10309)	SSR, TE	597	544.00	Shirasawa et al. (2013)
A. duranensis (K7988) × *A. stenosperma* (V10309)	SNP, SSR	384	705.10	Bertioli et al. (2014)
A. duranensis (K7988) × *A. stenosperma* (V10309)	SNP, SSR, RGA	502	1004.10	Leal-Bertioli et al. (2016)
Genome BB				
A. ipaensis (K30076) × *A. magna* (K30097)	SSR	149	1294.00	Moretzsohn et al. 92009)
A. ipaensis (K30076) × *A. magna* (K30097)	SSR, TE	798	461.00	Shirasawa et al. (2013)
A. ipaensis (K30076) × *A. magna* (K30097)	SSR, TE	399	678.00	Leal-Bertioli et al. (2015)
K 9484 (PI 298,639) × GKBSPSc 30,081 (PI 468,327) of *A. batizocoi*	SSR	449	1278.60	Guo et al. (2012)
Genome AABB				
Florunner × TxAG-6 {[*A. batizocoi* K9484 × (*A. cardenasii* GKP10017 × *A. diogoi* GKP10602)]4 × }	RFLP	370	2210.00	Burow et al. (2001)
ICG 12991 × ICGVSM 93541	AFLP	12	139.4	Herselman et al. (2004)
[Fleur 11 × (*A. ipaensis* × *A. duranensis*)4 ×]	SSR	298	1843.70	Fonçéka et al. (2009)
Yueyou 13 × Zhenzhuhei	SSR	131	679.00	Hong et al. (2008)

(continued)

Table 4.2 (continued)

Populations used	Markers used	No of loci mapped	Coverage (cM)	References
TAG 24 × ICGV 86031	SSR	135	1270.50	Varshney et al. (2009)
TAG 24 × ICGV 86031	SSR	191	1785.40	Ravi et al. (2011)
Yueyou 13 × Zhenzhuhei	SSR	132	684.90	Hong et al. (2010)
Yueyou 13 × Fu 95-5	SSR	109	540.69	Hong et al. (2010)
Yueyou 13 × J11	SSR	46	401.70	Hong et al. (2010)
TAG 24 × GPBD 4	SSR	56	462.24	Khedikar et al. (2010)
TAG 24 × GPBD 4	SSR	188	1922.40	Sujay et al. (2012)
TG 26 × GPBD 4	SSR	45	657.90	Sarvamangala et al. (2011)
TAG 24 × GPBD 4	SSR	181	1963.00	Sujay et al. (2012)
ICGS 44 × ICGS 76	SSR	82	831.40	Gautami et al. (2012b)
ICGS 76 × CSMG84-1	SSR	119	2208.20	Gautami et al. (2012b)
SunOleic 97R × NC94022	SSR, CAPs	172	920.70	Qin et al. (2012)
SunOleic 97R × NC94022	SSR, CAPs	206	1780.60	Pandey et al. (2014)
Tifrunner × GT-C20	SSR	318	1674.40	Wang et al. (2012)
Tifrunner × GT-C20	SSR, CAPs	239	1213.40	Qin et al. (2012)
YI-0311 × Nakateyutaka	SSR, TE	326	1332.90	Shirasawa et al. (2012a)
Satonoka × Kintoki	SSR, TE	1114	2166.40	Shirasawa et al. (2012b)
VG 9514 × TAG 24	SSR	95	882.90	Mondal et al. (2012)
A. hypogaea "Runner IAC 886" × (*A. ipaensis* × *A. duranensis*)4×	SSR, TE	1469	1442.00	Shirasawa et al. (2013)
Tifrunner × GT-C20	SSR, CAPs	378	2487.40	Pandey et al. (2014)
Tifrunner × GT-C20	SSR	418	1935.40	Pandey et al. (2014)
A. hypogaea "Runner IAC 886" × (*A. ipaensis* × *A. duranensis*)4×	SNP, SSR	772	1487.30	Bertioli et al. (2014)
Zhonghua 5 × ICGV 86699	SNP, SSR	1685	1446.70	Zhou et al. (2014)
VG 9514 × TAG 24	SSR, ISSR, TE, RGC	190	1796.70	Mondal et al. (2014a; b)

(continued)

Table 4.2 (continued)

Populations used	Markers used	No of loci mapped	Coverage (cM)	References
Zhonghua 10 × ICG12625	SSR	470	1877.30	Huang et al. (2015)
Zhonghua 10 × ICG12625	SSR, TE	1219	2038.75	Huang et al. (2016)
TAG 24 × GPBD 4	SSR, TE	289	1730.80	Kolekar et al. (2016)
SunOleic 97R × NC94022	SSR	248	1425.90	Khera et al. (2016)
Fuchuan Dahuasheng × ICG 6375	SSR	347	1675.60	Chen et al. (2016)
Xuhua 13 × Zhonghua 6	SSR	228	1337.70	Chen et al. (2016)
Florida-EP™ "113" × Georgia Valencia	SSR, SNP	30	157.80	Tseng et al. (2016)
ICGV 00350 × ICGV 97045	DArT, DArTseq	1152	2423.12	Vishwakarma et al. (2016)
79266 × D893	SSR	231	905.18	Li et al. (2017)
Florunner × TxAG-6 {[*A. batizocoi* K9484 × (*A. cardenasii* GKP10017 × *A. diogoi* GKP10602)]4 × }	SSR	91	1321.90	Wilson et al. (2017)
Yuanza 9102 × Xuzhou 68-4	SSR	743	1232.57	Luo et al. (2017)
Yuanza 9102 × Xuzhou 68-4	SSR	830	1386.19	Luo et al. (2017)
ICGV 07368 × ICGV 06420	DArT, SSR	854	3526.00	Shasidhar et al. (2017)
ICGV 06420 × SunOleic 95R	DArT, DArTseq	1435	1869.00	Shasidhar et al. (2017)
ICGV 06420 × SunOleic 95R	SNP	1211	–	Liang et al. (2017)
TMV 2 × TMV 2-NLM	TE	91	1205.66	Hake et al. (2017)
GG20 × CS19	SSR	12	558.74	Bera et al. (2016b)
ZH16 × sd-H1	SNP	3630	2098.14	Wang et al. (2018a; b)
Xuhua 13 × Zhonghua 6	SNP	2595	2465.62	Liu et al. (2020)
TG37A × NRCG CS85	SNP	266	1092	Dodia et al. (2019)
Tifrunner × NC 3033	SNP, SSR	1524	3382	Chavarro et al. (2020)
NC 3033 × Tifrunner	SNP, SSR	1524	3381.96	Luo et al. (2020a, b)
Consensus				

(continued)

Table 4.2 (continued)

Populations used	Markers used	No of loci mapped	Coverage (cM)	References
3 populations	SSR	175	885.40	Hong et al. (2010)
3 populations	SSR	293	2840.80	Gautami et al. (2012b)
2 populations	SSR	225	1152.90	Sujay et al. (2012)
13 maps	SSR, TE	3693	2651	Shirasawa et al. (2013)
8 populations	SSR, TE	5874	2918.62	Lu et al. (2018)

of allelic variation in nature which can provide high resolution mapping, however, QTL detection power is very low. Further, multiparent populations namely, MAGIC population, training population and recombinant inbred advanced intercross line (RIAIL) populations (Morrell et al. 2012) are being exploited. MAGIC populations involve recombination of alleles from multiple parents and provide a high mapping resolution and high power of detecting QTL (Cavanagh et al. 2008). By choosing different founder parents and creating a wide collection of interrelated RILs populations, NAM population captures genetic diversity, which allows achieving high resolution mapping by using power of ancestral meiotic recombination. In addition to that, whole-genome average interval mapping (WGAIM) along with the joint association mapping approaches have been developed to analyses QTL accurately (Verbyla et al. 2014). Further, WGAIM method concurrently integrates all probabilities at each marker for all individuates. Two NAM populations have been developed for peanut, *i.e.*, one each in Spanish type (cross of ICGV 91114 with 22 testers) and other in Virginia type (cross of ICGS 76 with 21 testers) and could be used for higher resolution of mapping (Varshney 2016; Pandey et al. 2016). Sixteen populations have been developed in a community wide project in the US and numerous QTLs have been identified for biotic stresses in a limited subset of these populations (Chu et al. 2018).

4.5.2.2 Trait Mapping and QTLs Discovery for Biotic Stresses

For most biotic stresses, various types of markers have been identified. Stalker and Mozingo (2001) established an association between ELS sporulation and RAPD marker AM 1102 in a peanut population derived from a cross between an *A. hypogaea* and *A. cardenasii* introgression line with 'NC 7'. Mondal et al. (2008) identified RAPD marker J 7 (1300) as a suitable genetic marker associated with rust. Genetic linkage maps with 188 and 181 loci respectively, were constructed from population derived from TAG 24 × GPBD 4 and TG 26 × GPBD 4. Morever, RILs mapping populations were used to associate SSR markers (IPAHM103, GM2009, GM1536,

GM2301 and GM2079 with major QTLs for rust. Using genotyping and phenotyping data, 13 QTLs for rust and 13 QTLs for late leaf spots were discovered from these RILs populations, explaining 2.54 to 82.96% and 10.07 to 67.8% phenotypic variance, respectively (Sujay et al. 2012). In $F_{2:3}$ progenies of cross between two contrasting parents, TMV 2 (susceptible) × COG 0437 (resistant), Shoba et al. (2012) identified SSR marker, PM384 associated with LLS and rust. Shoba et al. (2013) also reported a QTL for LLS in the same mapping population with 37.9% phenotypic variation. However, large QTLs that contribute > 20% phenotypic variation and must be confirmed should be targeted for active QTL introgression in elite breeding lines (Varshney et al. 2013). Mondal et al. in the year 2012 reported two EST derived SSR markers named as SSR HO115759 and SSR GO340445 and these were appropriate candidates for use in marker-assisted selection as they are closely linked to rust resistance. Two transposable element (TE) based markers, TE 498 and TE 360, were reported to be in association with the rust resistance in a RIL population of VG 9514 × TAG 24. But, these linked markers need further validation to speed up the process of introgressing resistance into megavarieties (Sujay et al. 2012; Gajjar et al. 2014).

Lei et al. (2006) detected an AFLP named as, E45/M53-440 originated SCAR primer, AFs-412 to be closely associated with resistance to infection by *A. flavus.* For protection against *A. flavus* invasion, Liang et al. (2009) idebtified six QTLs, each of which is located on a separate linkage group and can explain phenotypic variance of 6.2 to 22.7%. Two large QTLs for TSWV resistance were discovered by Qin et al. (2012). The AFLP marker was used by Herselman et al. in 2004 to map aphid resistance in ICG12991. A number of DNA markers linked to root-knot nematode resistance were also discovered. For the root-knot nematode, *Meloidogyne arenaria*, RAPD markers (Z3/265, RKN410, KKN229 and RKN440), RFLP loci (*R2430E* and *R2545E)* and SSR markers were found to be linked tightly to dominant resistance genes, *Mae* (for restricting egg number) and *Mag* (for restricting gall formation) (Burow et al. 1996; Garcia et al. 1996; Church et al. 2000; Wang et al. 2008; Carpentieri-Pípolo et al. 2014). This marker was cloned and SCAR (197/909) and RFLP (R2430E, R2545E and S1137E) probes obtained from cDNA libraries further confirmed linkages with nematode resistance (Burow et al. 1996; Chu et al. 2007). Nagy et al. (2010) used high-resolution mapping for nematode resistance to establish another SSR marker, GM565. Later, another tool, single base pair extension (SBE) was discovered to be efficient for high-efficient SNP mapping in peanut, and the genetic map revealed five candidate genes conditioning resistance to biotic stresses (Alves et al. 2008). Later, Khera et al. in the year (2013) used a collection of 96 explanatory SNPs to establish KASPar assays, named as GKAMs (Groundnut KASPar Assay Markers), and validated 90 GKAMs against different biotic stresses. Clevenger et al. (2017) used QTL-seq approach to identify KASP markers from an RIL population segregating for quantitative field resistance to LLS. QTL analysis from cross,'Tifrunner × GT-C20' derived F_2 genetic population detected two QTLs for thrips, 15 for TSWV, and 37 QTLs for LS. However, in the advanced F_5 population, one for thrips, nine for TSWV, and 13 for leaf spots have been identified. This is the first research to report new QTLs for thrips, TSWV, and leaf spots, and it will

need to be improved and validated in the future (Wang et al. 2013, 2014). Using a common RILs population derived from cross, VG 9514 X TAG 24, two main QTLs, qTDP-b08 for total development period and qAE2010/11-a02 for adult emergence with 57–82% and 13–21% PVE respectively, were detected for bruchid resistance (Mondal et al. 2014a; b). A mapping population derived from the SunOleic 97R x NC94022 cross yielded 155 QTLs, including one and three significant QTLs for TSWV and LLS resistance, respectively (Guo et al. 2013). Further, many marker-trait associations (MTAs) for *Aspergillus flavus* (01, 24.69% PV), ELS (06, 9.18–10.99% PV), LLS (01, 18.10% PV) and GRD (31, 10.25–39.29% PV) were discovered using GWAS approach (Pandey et al. 2014). Recently, Jasani et al. (2021) reported one major QTL from cross JL-24 x NRCGCS-85 for PBND resistance. Details of some main QTLs that have been reported in peanut to be associated with disease stresses are given in Table 4.3.

Table 4.3 QTLs reported for biotic stresses in peanut

S. No.	Traits/biotic stress	Marker system	Source/population	QTLs identified	PVE (%)	References
1	Rust and LLS	SSRs	GJG17 × GPBD4	Two	29.06–70.52	Ahmad et al. (2020)
2	*Sclerotinia blight*	SNPs	Tamrun OL07 × T × 964117	Seven	6.6–25.6	Liang et al. (2020)
3	*Aspergillus flavus*	SNPs	Yueyou 92 × Xinhuixiaoli	Two	5.15–19.04	Khan et al. (2020)
4	Bacterial wilt	SSRs and SNPs	Xuhua 13 × Zhonghua 6	One	37.79 -78.86	Luo et al. (2020a, b)
5	Stem rot	SSRs and SNPs	Tifrunner × NC 3033	33	4.76–20.01	Luo et al. (2020a, b)
6	Stem rot	SNPs	Tifrunner × NC 3033	Two	9–13	Cui et al. (2020)
7	PBND	SSRs	TAG 24 × ICGV 86031	5	3.92–12.57	Jadhav et al. (2019)
8	ELS and LLS	SNPs	Florida-07 × GP-NC WS16	6	5–41	Chu et al. (2019)
9	Tomato Spotted wilt virus	SNPs	SunOleic 97R × NC94022,	One	36.51	Agarwal et al. (2019)

(continued)

Table 4.3 (continued)

S. No.	Traits/biotic stress	Marker system	Source/population	QTLs identified	PVE (%)	References
10	Aflatoxin	SSRs	Zhonghua 10 × ICG 12625	12	9.32–21.02	Yu et al. (2019)
11	Bacterial wilt	SNPs	Xuzhou 68–4 × Yuanza 9102	4	7.72–23.33	Wang et al. (2018a; b)
12	ELS, LLS and TSWV	SNPs	Tifrunner × GT-C20	35	6.32–47.63	Agarwal et al. (2018)
13	PBND	SSRs	JL-24 × NRCGCS-85	2	12.38–16.88	Jasani et al. (2018b)
14	Stem rot	SSRs	GG-20 × NRCGCS-319	1	25.36	Kamdar et al. (2018)
15	ELS and LLS	SNPs	Florida-07 × GP-NC WS 16	15	4.93–16.60	Han et al. (2018)
16	ELS, LLS and TSWV	SSRs	Tifrunner × GT-C20	42	6.36–15.6	Pandey et al. (2017a)
17	Leaf spot	SNPs	Tamrun OL07 × Tx964117	Six	11- 24	Liang et al. (2017)
18	Bacterial wilt	SSRs and SNPs	Xinhuixiao × Yueyou 92	Two	12–21	Zhao et al. (2016)
19	LLS	SNPs	Zhonghua 5 × ICGV 86699	20	3.41–19.12	Zhou et al. (2016)
20	Rust and LLS	SSRs and TE	TAG24 × GPBD4	Five	10.2–53.7	Kolekar et al. (2016)
21	Root-knot nematode	SNPs	*A. duranensis* × *A. stenosperma*	Eight	5.70–43.70	Leal-Bertioli et al. (2016)
22	ELS, LLS and TSWV	SSRs and ESTs	SunOleic 97R × NC94022	48	3.88–29.14	Khera et al. (2016)
23	TSWV	SSRs	Florida EPTM “113” × GeorgiaValencia	2	10.02–22.70	Tseng et al. (2016)
24	Stem rot	SSRs	GG-20 × CS-19	1	17.15	Bera et al. (2016b)
25	Rust	SSRs and TE	*A. ipaënsis* (accession K 30076) × *A. magna* (accession K 30097)	13	5.8–59.3	Leal-Bertioli et al. (2015)

(continued)

Table 4.3 (continued)

S. No.	Traits/biotic stress	Marker system	Source/population	QTLs identified	PVE (%)	References
26	Bruchid	SSRs	VG 9514 × TAG 24	44	11.00–82.00	Mondal et al. (2014a; b)
27	Root-knot nematode	RFLP	Florunner × TxAG-6	10	-	Burow et al. (2014)
28	LLS	SSR	TMV 2 × COG 0437	1	20.2–24.1	Shoba et al. (2013)
29	TSWV, LS, Thrips	SSRs	Tifrunner × GT-C20	77	5.20–34.92	Wang et al. (2013)
30	TSWV	SSRs	Tifrunner × GT-C20 and SunOleic 97R × NC94022	2	12.90–35.80	Qin et al. (2012)
31	Rust and LLS	SSRs	TAG 24 × GPBD 4 and TG 26 × GPBD 4	43	2.54–82.96	Sujay et al. (2012)
32	LLS and Rust	SSRs	TAG 24 × GPBD 4	23	1.70–55.20	Khedikar et al. (2010)

4.5.2.3 Advanced Trait Mapping Approaches

In addition, advanced-backcross QTL (AB-QTL) is proposed by Tanksley et al. (1996) to save the time and increase the precision of identifying associated markers and simultaneous ingression of desirable traits from wild species and wild forms to cultivated genotypes. Some QTLs for root-knot nematode resistance (Fonceka et al. 2012; Burow et al. 2014), LLS and rust resistance (Varshney et al. 2013) was identified using the same approach. Further higher resolution towards mapping efforts can be gained with NGS methods and mapping by sequencing approaches (Huang et al. 2009; Schneeberger and Weigel 2011). Furthermore, QTL-seq, MutMap, and BSR-seq are three new trait mapping methods that have demonstrated for rapid recognition of candidate genomic regions and diagnostic markers for the targeted traits. The DNA samples pooled from F_2 segregating progeny derived from a cross between a mutant type and corresponding wild type are used in the MutMap method to conduct whole-genome re-sequencing (WGRS) (WT). The SNP index is used to identify new SNPs, and then the sequence of bulk DNA is compared to the reference sequence. The SNPs that have sequence reads containing only the mutant sequences (SNP index = 1) are assumed to be related to the causal SNP responsible for the mutant phenotype. MutMap strategy was conceptually integrated to the standard F_2 and RIL populations in the QTL-seq technique (Takagi et al. 2013). For accelerated detection of agronomically significant QTLs, a combination of BSA and whole genome re-sequencing is used. BSR-Seq uses RNA-Seq reads for mapping traits effectively, even in populations in which no molecular polymorphic survey have previously been

conducted (Liu et al. 2012). Allele-specific functional markers and SNPs markers for rust resistance and LLS resistance were identified in peanut using the QTL-seq method (Pandey et al. 2016, 2017b). ICRISAT recently released a 10-SNP panel with related SNPs for two foliage fungal diseases (rust and LLS) mapped on chromosomes A02 (LLS) and A03 (rust).

4.5.3 Molecular Breeding for Disease Resistance

Some of the diagnostic markers reported to be linked with QTLs of significant effect have been validated and established for use in marker-assisted selection (MAS) and marker-assisted backcross (MABC) breeding programme. MABC is most commonly employed to introgress transgene or loci with major effect into a commercial cultivar. (Figs. 4.13 and 4.14). Further, to improve the genotype MARS and genomic selection (GS approaches are now days are being used to accumulate desirable alleles with small effects). Using MABC approach first variety with resistance to root-knot nematode,-NemaTAM was released in the USA (Simpson et al. 2003). Since then, several other cultivars with the use of *A. cardenasii*, as a source of resistance have been released in the USA named as, Tifguard (Holbrook et al. 2008), Webb (Simpson et al. 2013), Georgia-14 N (Branch and Brenneman 2015) and TifNV-High O/L (Holbrook et al. 2017). Major QTLs governing rust and LLS explaining up to 82.62% and 67.98% phenotypic variation respectively, was transferred from

Fig. 4.13 Peanut plants tagged for genotyping in early generation in the field

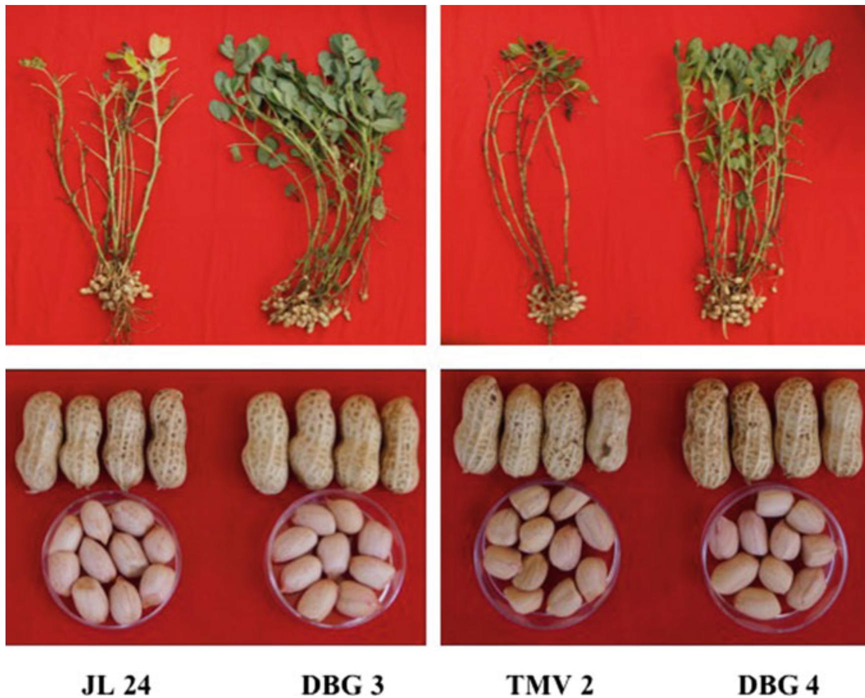

Fig. 4.14 Late leaf spot resistant marker-assisted backcross breeding lines DBG 3 and DBG 4 developed from JL 24 and TMV 2, respectively (Yeri and Bhat 2016; Kolekar et al. 2017)

'GPBD 4' into three rust susceptible varieties viz., ICGV 91114, TAG 24 and JL 24 by using four linked markers namely, IPAHM103, GM2301, GM2079 and GM1536 in MABC program (Varshney et al. 2014). Two developed amphidiploids synthetics from ICRISAT, one is ISATGR 278-18 derived from cross, *A. duranensis* × A. *batizocoi* and other is, ISATGR 5B derived from cross, *A. magna* × *A. batizocoi* were utilied to introgress resistance to foliar diseases in five mega-varieties namely, ICGV 91278, ICGV 91114, ICGS 76, JL 24 and Dh86 using backcrosses (Kumari et al. 2014). Furtherefforts to use the linked markers for resistance to foliar diseases for pyramiding desirable QTLs in the three popular peanut cultivars viz., GJG 9, GG 20 and GJGHPS 1 are underway (Fig. 4.15).

Marker-assisted selection (MAS) aims to improve tolerance against biotic stresses by targeting major QTLs and eventually omits the possibility of stacking minor effect and epistatic QTLs. Thus, combining the desirable genes or pyramiding of minor and epistatic QTLs through the MABC is a big challenging task (Peleman and Voort 2003). To accumulate beneficial alleles with small phenotypic effects in a single genotype, the MARS and GS approaches can be used. GS is a kind of MAS that at a time predicts all loci, haplotype, or marker effects across the genome to calculate Genomic Estimated Breeding Values (GEBVs). It is a tool in plant breeding to predict the genetic value of untested lines based on genome-wide marker

Fig. 4.15 High yielding peanut breeding lines with huge pod bearing

data. Estimated GEBVs are then used for selecting desirable types for advancing the breeding cycle without need of phenotyping. Unlike MABC and MARS, GS or genome wide selection (GWS) aims to sort out superior lines with higher breeding value in a breeding program using marker profile data of whole genome and high throughput genotyping. As a result, GS appears to be a possible strategy for breeding complex traits in the near future. But these approaches in peanut have not been widely explored. However, more recently initial GS usage attempts have identified four GS-models and suggested the use of the best models to achieve higher accuracy in predicting characters with large G $\times$ E effects in peanut (Pandey et al. 2020).

4.6 Transcriptomics and Proteomics

Transcriptomic analysis has been employed to identify the differentially expressed genes for resistance to ELS (Gong et al. 2020), LLS (Han et al. 2017) and leaf rust (Rathod et al. 2020a, b). The results suggest that a few major genes and several factors

mediate the resistance to ELS disease, showing the characteristics of quantitative trait in defense responses. Most of these studies identified the defense-related genes. Molecular responses of the wild peanut challenged with the LLS pathogen were studied using cDNA-AFLP and 2D proteomic study. A total of 233 differentially expressed genes, involved in cell wall strengthening, hypersensitive reaction and resistance related proteins were identified in wild peanut, *A. diogoi* (Kumar and Kirti 2015). Transcriptomic analysis in the *A. flavus* resistant peanut cultivar J11 led to the detection of 663 differentially expressed genes. Further functional analysis revealed that these genes encoded a wide range of defense or PR- proteins (pathogenesis related proteins). Changes in the expression patterns of these genes might contribute to peanut resistance to *A. flavus* (Zhao et al. 2019). Bosamia et al. (2020) used RNA-Seq to unravel the mechanisms of resistance to stem rot caused by *Sclerotium rolfsii* using a resistant (NRCG-CS85) and susceptible (TG37A) genotype. Differentially expressed genes and translated proteins in wild peanut indicate its defense mechanism upon interaction with pathogen and provide initial breakthrough of genes possibly involved in sensing or recognizing and early signalling responses to fight the infection through subsequent development of resistance.

4.7 Transgenic Approaches for Genetic Improvement of Peanut Against Biotic Stresses

As a consequence of ploidy barrier between the cultivated species and the wild species, introgression of stress-related genes from the diploid progenitors by conventional breeding becomes complex. Further, introgression lines developed by crossing wild species with cultivated peanuts carried undesirable gene blocks. To overcome the problem of lack of beneficial genes within crossable germplasms, genetic engineering/recombinant DNA techniques such as *Agrobacterium tumefaciens* mediated or direct transfer of desired genes from wild species would be an ideal option to impart resistance against diseases (Vasavirama and Kirti 2012).

Resistance to several fungal and virus diseases has been achieved through the use of transgenes coding for cell wall components such as chitinase, glucanase etc., PR proteins, coat proteins, bacterial chloroperoxidase, oxalate oxidase, RNA interference (RNAi), and crystal proteins. Sunkara et al. (2013) reviewed the use of chitinase, glucanase, Rs-AFP2 (*Raphanus sativus* antifungal protein-2) and SniOLP (*Solanum nigrum* osmotin like protein) for LLS and ELS, oxalate, chitinase and glucanase for *S.* blight, chitinase for rust, and anionic peroxidase, glucanase, stilbene synthasesynthetic peptide D4E1, chitinase, mod1, nonheme chloroperoxidase (cpo), LOX 1, and Pn LOX 3 against *A. flavus* infection and aflatoxin production. When compared to the parent variety, transgenic lines of the Okrun cultivar harboring chitinase gene from rice and glucanase genes from alfalfa showed a 43–100% reduction in *S. blight* incidence (Chenault et al. 2005). Two genes viz., Rchit and CHI coding for chitinase

enymes against *Fusarium* wilt and leaf spots fungi have been evaluated for inheritance in peanut transgenic events (Rohini and Sankara 2001; Iqbal et al. 2011, 2012). Late leaf spots incidence was decreased in transgenic lines of peanut expressing a defensin gene, BjD from mustard (Anuradha et al. 2008). Transgenics with cDNA sequence of barley oxalate oxidase conferred enhanced resistance to blight by *Sclerotinia* (Livingstone et al. 2005). Transgenics developed using bacterial non-heme chloroperoxidase gene from *Pseudomonas pyrrocinia (cpo-p)* and rice chitinase gene (*Rchit*) showed hyphal growth inhibition of *A. flavus* (Niu et al. 2009; Prasad et al. 2013).

The complete nucleotide sequence (4019 nts long) and genome organization (4 ORFs) of GRV are known (Taliansky et al. 1996). Because the coat protein gene of virus, GRAV has been sequenced and transformation constructs is created, the chances of producing rosette-resistant cultivars by inserting the coat protein genes into peanut have improved significantly (Taliansky et al. 1998). Peanut cultivar JL 24 was transformed with the GBNV nucleoprotein gene at ICRISAT, and T2 transgenic events were tested for virus resistance. If these events are successful, they will provide reliable GBNV resistance that can be bred into other peanut cultivars through back-cross breeding programs. Also, the genomes of viruses namely, PCV and IPCV is sequenced, so there are excellent chances of using viral coat protein genes to cuase resistance in peanut using unorthodox methods (Sharma and Anjaiah 2000). At ICRISAT, peanut cultivar JL24 was transformed with IPCV-H coat protein and replicase genes to induce pathogen-derived resistance. Genetically modified peanut cultivars that carry viral coat protein gene exhibited high levels of resistance to PStV (Franklin et al. 1993). Further, transgenic peanut plants of Gajah and NC 7 that contained untranslatable full length sequence (*CP2*) and translatable *CP* gene with an N-terminal truncation (*CP4*) of PStV, offered resistance to virus (Higgins et al. 2004). Insertion of viral nucleocapsid protein-coding gene (*tswvnp)* in peanut genome has resulted in resistance to TSWV (Brar et al. 1994). Furthermore, by activating RNA silencing, a natural virus defense mechanism, high-level resistance or immunity can be induced in plants (Waterhouse et al. 2001). RNAi technology such as,RNA silencing or homologous gene cosuppression are powerful methods for developing resistance to viruses in peanut genotypes (Wang et al. 2000). At ICRISAT, an RNAi-mediated approach is being used to counteract the effect of the PBNV genome's nonstructural silencing suppressor gene (NSs gene). Transformed plants with specific small RNAs, the products of RNA silencing were highly resistant to PStV infection and the resistance was stably inherited over atleast five generations (Dietzgen et al. 2004). Resistance derived from pathogens by introducing GRAV or GRV genes/ genome sequences, or SatRNA-derived sequences that inhibit/slow down GRV replication is a possible strategy against GRD via transgenic plant generation (Taliansky et al. 1996). Cry1 EC gene against *S. litura* (Tiwari et al. 2008) and cry1 X gene against *H. armigera* and *S. litura* (Entoori et al. 2008) are two synthetic genes that have shown promise against their respective insect pests. When the trypsin inhibitor gene from cowpea was introduced into peanuts, it increased tolerance to insects (Xu et al. 2003). The success and achievement of transformation techniques is still poor due to its allopolyploidy, genotype specificity, low transformation and

regeneration efficiency and low level of transgene expression. Although many transgenic lines have been developed against biotic stresses, to date no transgenic cultivars of peanut is released commercially. Targeted genome editing technology for functional genes is an exciting new advancement. It has the potential to be an effective tool in driving disease-fighting varietal development. Plant targeted genome editing has proven to be effective using zinc finger nucleases (ZFNs) and transcriptional activator-like effector nucleases (TALENs), which involve two DNA binding proteins flanking a sequence of interest (Lloyd et al. 2005; Wright et al. 2005; Cermak et al. 2011; Li et al. 2012; Mahfouz et al. 2011). Furthermore, CRISPRs (clustered regularly interspaced short palindromic repeats), a high-throughput genome editing technology focused on the prokaryotic immune system, offer a promising hope for further peanut improvement. Recently, CRISPR/ Cas9 technology has become very popular for genome editing, trait discovery and manipulating genome in desired direction. However, utilization of CRISPR based genome modification in peanut is challenging, because of complexity of genome. Also, CRISPR/Cas9 technology does not transfer DNA sequences from one species to another. However; CRISPR/Cas9 technology has the ample scope for enhancing the limited resistance available against biotic stresses.

4.8 Future Prospects

Peanut is a high nutritional value, multipurpose food-feed-fodder crop that has gained global significance. The key to maintain competition and meet the potential future demand is the genetic enhancement of peanuts for increased yield and enhanced tolerance to biotic and abiotic stresses. Knowing the presence of higher diversity, allelic variations and presence of novel alleles in wild *Arachis* species, more conserted multiinstitutional and multidisciplinary efforts with greater investment are required to intensively evaluate and properly characterize the desirable quest in wild *Arachis* and their use in breeding program supported with modern genomic technologies. New genetic and genomic innovations have given tremendous optimism to achieve higher genetic gains with high precision and accuracy in less time and resources. Peanuts now have enough genomic and genetic resources required to speed up the process of peanut improvement. There are presently few but successful examples of molecular breeding products available in peanut; however in the coming years there will be more of such successful tales. In genomics research, still, more efforts are required to saturate the peanut linkage map so that MAS can be deployed for peanut improvement. At the same time, new breeding technologies such as genomic selection and genome editing are also being implemented to develop next-generation model peanut varieties that can give better performance under changing climatic conditions. Moreover, to combine conventional breeding and molecular breeding approaches, a comprehensive approach is needed to improve complex traits governed by multigenes and other problems that peanut is currently facing.

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