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Genetic Management of Virus Diseases in Peanut

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ABSTRACT

Peanut, also known as groundnut (*Arachis hypogaea* L.) is a major oilseed crop in the world. About 31 viruses representing 14 genera are reported to naturally infect peanut in different parts of the world, although only a few of these are of economic importance. These include groundnut rosette disease in Africa, tomato spotted wilt disease in the United States, peanut bud necrosis disease in south Asia, and peanut stripe virus disease in east and southeast Asia. Cucumber mosaic virus disease in China and Argentina and peanut stem necrosis disease in certain pockets in southern India are also economically important. Host plant resistance provides the most effective and economic option to manage virus diseases. However, for many virus diseases, effective resistance gene(s) in cultivated peanut have not been identified. With a few exceptions, the virus resistance breeding work has received little attention in peanut improvement programs. Transgenic resistance offers another option in virus resistance breeding. This review focuses on the status of genetic resistance to various economically important groundnut viruses and use of transgenic technology for the improvement of virus resistance.

KEYWORDS: *Arachis hypogaea*; genetic transformation; groundnut vectors; virus resistance

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ABBREVIATIONS

ADPC	Area under the disease progressive curve
BCMV	Bean common mosaic virus
cp	Coat protein
CMV	Cucumber mosaic virus
CMMV	Cowpea mild mottle virus
DNA	Deoxyribose nucleic acid
DSI	Disease severity index
ELISA	Enzyme-linked immunosorbent assay
GCA	General combining ability
GCSV	Groundnut chlorotic spotting virus
GCV	Groundnut crinkle virus
GEV	Groundnut eyespot virus
GRAV	Groundnut rosette assistor virus
GRAV*cp*	GRAV *coat protein*
GRD	Groundnut rosette disease
GRV	Groundnut rosette virus
IPCV	Indian peanut clump virus
IPCV*cp*	IPCV *coat protein*
IPCV*rep*	IPCV *replicase*
NSs	Nonstructural silencing suppressor gene
PBNV	Peanut bud necrosis virus
PBNV*np*	PBNV *nucleocapsid protein*
PCV	Peanut clump virus
PDR	Pathogen-derived resistance
PMV	Peanut mottle virus
PMMV	Peanut mild mottle virus
PSND	Peanut stem necrosis disease
PStV	Peanut stripe virus
PSV	Peanut stunt virus
PTGS	Posttranscriptional gene silencing
RNA*i*	Ribose nucleic acid interference
*Sat*RNA	Satellite-RNA
SCA	Specific combining ability
TSV	Tobacco streak virus
TSV*cp*	Tobacco streak virus *coat protein*
TSWV	Tomato spotted wilt virus

I. INTRODUCTION

Peanut (*Arachis hypogaea* L., Fabaceae) is also called groundnut in Africa, *mani* in Spanish, *amondoim* in Portuguese, *pistache* in French, *mungphali* in Hindi, and *ying zui dou* in Chinese. With an annual world production of 37.2 million tonnes from 23.4 million ha, peanut is a major oilseed crop (FAOSTAT 2007). It is grown primarily for its high quality edible oil (48%–50%) and easily digestible protein (26%–28%). It is also a rich source of vitamins (particularly B group and E), minerals (Cu, P, Ca, Mg, and Fe) and dietary fiber. Peanut haulms provide excellent fodder for livestock while its cake obtained after oil extraction is used in animal feed industry. Peanut shells are used in cardboard making, as fuel and as filler in feed and fertilizer industries. Being a leguminous crop, it enriches the soil with nitrogen and is, therefore, valuable in sustaining the cropping systems. Globally, about two thirds of the produce is crushed for oil and one-third is used in direct consumption and for confectionery purposes. However, the utilization pattern varies within and among countries. In some countries it is primarily an oilseed crop (such as India and Myanmar), while in others, it is used as a food crop (such as United States and others). Over the years, the food use of peanut has shown an increasing trend. Peanut makes important contributions to the human diet in many countries, and its widespread acceptability is attributed to its economic value to the industry and nutritional benefits to the consumers.

Peanut is grown commercially throughout the tropical, subtropical, and warm temperate regions of the world. It is largely a smallholder crop, grown under rainfed conditions in the semiarid regions. The semiarid tropical region, characterized by unpredictable rainfall, contributes over 90% to the world peanut production. The low yields in peanut are primarily due to low inputs, rainfed cultivation of the crop in marginal lands, nonavailability of seed of suitable high-yielding cultivars and the occurrence of insect pests and diseases at different stages of the crop.

The optimum air temperature for growth and development of peanut is between 25 and 30°C. Temperatures above 35°C are detrimental to peanut production. The reproductive phase of peanut is more sensitive to heat stress than the vegetative phase. Due to favorable weather conditions and availability of irrigation, two crops of peanut in rotation with cereals are grown annually in many southeast Asian countries. However, multiple cropping, particularly when the peanut crops overlap, can lead to pathogen/disease buildup in a region. While sole cropping is generally practiced under irrigation, intercropping and mixed cropping are more common in the rainfed conditions. Fodder is also an important consideration in the rainfed production systems. The mixed cropping is more

prevalent in subsistence farming. When the crop is grown under high input conditions with irrigation, yields exceeding 4.0 t ha^{-1} are not uncommon.

For low input rainfed systems, nutrient- and water-use efficient cultivars with resistance/tolerance to prevailing biotic and abiotic stresses are needed. There are several fungal and viral diseases, which infect peanut crop. However, not all of these are economically important.

II. VIRUS DISEASES

About 31 viruses representing 14 genera are reported to naturally infect peanut in different parts of the world. Economically important virus diseases in peanut include peanut mottle potyvirus (PMV), peanut stripe potyvirus (PStV), peanut clump furovirus (PCV), groundnut rosette disease (GRD) complex, tomato spotted wilt tospovirus (TSWV), peanut stunt cucumovirus (PSV), cowpea mild mottle carlavirus (CMMV), cucumber mosaic cucumovirus (CMV), peanut bud necrosis tospovirus (PBNV), and tobacco streak ilarvirus (TSV). Reddy (1991) reviewed information then available on economically important peanut virus diseases including their distribution, symptoms, and method of identification. Indian isolates of TSWV reported in the review were subsequently found to be different from U.S. isolates and renamed as PBNV. The minor viruses in West Africa include groundnut eyespot virus (GEV, a potyvirus, transmitted by *Aphis craccivora* and *Aphis cirticola* in a nonpersistent manner, no seed transmission recorded), groundnut crinkle virus (GCV, a carlavirus, transmitted by *Bemisia tabaci* in a nonpersistent manner, no seed transmission recorded), groundnut chlorotic spotting virus (GCSV, a potexvirus, transmitted by *A. craccivora* and *Aphis spiraecola* in a nonpersistent manner, seed transmission not tested), and viruslike diseases include groundnut streak disease, groundnut mosaic disease, groundnut golden disease, and groundnut flecking disease (Dollet et al. 1986). In Eastern and Southern Africa, another minor virus disease, groundnut streak necrosis, is reported (Bock 1989). In south and southeast Asia, peanut yellow spot tospovirus (Reddy et al. 1991a), peanut chlorotic fan-spot tospovirus (Chen and Chiu 1996), and peanut green mosaic potyvirus (Sreenivasulu et al. 1981) and in the United States bean yellow mosaic potyvirus are reported as minor virus diseases. A viruslike disease, witches broom, noticed in south and southeast Asia, is caused by phytoplasma and transmitted by leafhoppers (Reddy 1984). However, the major and widespread virus disease in Africa is GRD, in the USA TSWV, in south Asia PBNV, in east and southeast Asia PStV, and in China PStV and CMV.

A. Management Options for Virus Diseases

A thorough knowledge of epidemiology and properties of the virus, ecology of the vector(s), crop and cultivated and wild hosts of both virus and vector(s) and virus–vector–host plant interactions is essential in devising rational methods for minimizing virus-induced losses in a crop. Insect vectors can be managed either through chemical control or cultural methods or both. However, these do not have influence on the virus inoculum once the plant is infected. With genetic resistance, both vector and virus can be managed effectively.

1. Cultural Management. As many peanut viruses are seedborne, the elimination of this primary and randomly distributed source of infection is important to minimize the secondary spread of virus in the field and its long distance dispersal. Seed-transmitted viruses of peanut include CMV, PCV, PMV, PStV, and PSV. Use of quality seed produced under virus-free conditions should be promoted to minimize the damage caused by seedborne virus diseases. Undersized, shriveled, and deformed seeds should be removed from the seed lots. Infected seedlings should be removed carefully from the field and destroyed. Similarly, the volunteer plants in the off-season should also be removed from the field and destroyed. They can serve as carrier of the virus inoculum and provide shelter to insect vectors. In the case of use of virus-free seed, distance isolation (100 m or more) from other peanut fields (as they might be source of virus) will help to minimize the incoming virus infection in the field.

A thorough understanding of the vector ecology can also help in devising cultural practices to minimize virus damage to the crop. Some of these cultural practices include adjusting sowing dates to avoid vulnerable crop stages during the peak migration of vectors that may vary from place to place, season to season, and prevalent cropping patterns and crop profiles in a region, not locating field down wind, creating physical barriers through border cropping with tall growing cereals or intercropping, close row and plant spacing to provide early ground cover, chemical seed treatment to protect from vector damage at seedling stage, and growing a trap crop to attract vectors (and subsequently destroying them mechanically or chemically). Peanut cultivation with polythene mulch, which is popular in China and Vietnam, besides giving higher yield also helps to reduce PStV incidence. It should be evaluated against other virus diseases also. Rogueing of infected plants, especially during early stages of plant growth, should be avoided since this practice creates gaps in the field and can increase

incidence of virus diseases such as PBND, PSND, GRD, and TSWV. However, in a rain-dependent production system, adjustment of sowing dates may not be possible. Similarly, in a multiple cropping situation, adjustment of sowing dates may not be feasible unless the sowing or duration of the preceding or the proceeding crop is suitably altered. Recognizing the conditions that are likely to favor PSND caused by TSV in southern India, Prasada Rao et al. (2003a) recommended the following cultural management practices to contain the disease: removal of weeds, particularly parthenium (*Parthenium hysterophorus*) germinated with early rains and growing wild on fallow lands, roadsides and field bunds, border cropping (7–11 rows) with fast growing (tall) pearl millet (*Pennisetum glaucum*), sorghum (*Sorghum bicolor*), or maize (*Zea mays*), optimum plant population, seed treatment with imidacloprid (Gaucho 70 WS), use of systemic insecticide for vector control and isolation from virus host crops such as sunflower (*Helianthus annuus*), marigold (*Tagetes erecta*), and others. Since the PCV is transmitted by a soilborne fungus, *Polymyxa graminis*, rotation of peanut with cereals in infested fields should be avoided. Grassy weeds and their roots should be removed regularly from the infested fields. Grain from crops grown in infested fields should not be used as seed to sow future crops. Before sowing of peanut crop, a trap crop of pearl millet can be raised to reduce the PCV inoculum load in the soil (Delfosse et al. 1997). Although, the use of biocides and soil solarization to control *P. graminis* in the soil is effective, these practices are not practical or economical in large-scale farming.

No single management tool provides adequate control of peanut virus diseases. Cumulative effects of multiple management practices can significantly reduce disease incidence. Under severe disease pressure, the cultural management alone may not provide enough protection to the crop. Integrated disease management, which employs genetic resistance and cultural practices, can delay or slow down the development of virus diseases in the field. In the United States, Brown et al. (2000) developed a tomato spotted wilt risk index based on cultivar selection, planting date, plant population, in-furrow insecticide, disease history, row pattern, tillage practices and herbicide usage with relative weights, which may require change depending on the location, to enable farmers to assess the relative risk of the disease in a particular field and identify the combination of disease-suppressive factors that best apply to their situation.

2. Vector Control. Common insect vectors in peanut include thrips, aphids, and white flies. In the case of PCV, soilborne fungus *P. graminis* is the vector (Ratna et al. 1991). Effective chemical control measures are available against these insect vectors but they are not eco-friendly and often expensive for rainfed agriculture. Further, to manage virus diseases effectively, these chemical vector control measures are to be implemented early on as prophylactic measures. Efficacy to manage virus diseases through chemical control of vector is also dependent on the nature of the virus (persistent or nonpersistent), its acquisition period, its transmission period, virus retention in the insect and insect knock down time, and so on. It is easy to manage GRD by chemical control as the acquisition period of the virus complex by aphid vector is long thereby giving enough opportunity to chemicals to kill the vector. On the other hand, management of PBNV through chemical control of thrips vector is not effective as the virus is nonpersistent and its transmission period is very short before chemicals could kill the vector. In such situations, it is likely that agitated vectors could spread virus inoculum further before they die.

Insect vectors can also be managed in an environment-friendly manner through bioagents and botanicals. However, their impact on virus diseases needs to be studied further. If they do not result in quick knock down, the vectors would have enough time to spread the virus inoculum in the field before they die.

3. Genetic Resistance. Host plant resistance provides the most effective and economic option to manage virus diseases. Resistant cultivars with superior agronomic traits are easily adopted by the farmers. However, for the development of resistant cultivars, it is essential to have, in addition to an effective screening technique, enough genetic variation in either cultivated or related wild species in their response to virus infection. The strategy for breeding for host plant resistance depends on the crop species, nature of the reproductive biology (self-pollinated or cross pollinated), type of cultivar in use (hybrid, homozygous line, or vegetative clone), and inheritance of the resistance (monogenic, oligogenic, or polygenic; dominant or recessive). If the resistance is available in cultivated species, it is easy to transfer resistance gene(s) into superior agronomic backgrounds. Similar is the case with the related wild species if they are sexually compatible with the cultivated types. If the resistant sources are available only in related wild species that are difficult or impossible to use in crossing, techniques such as *in vitro* culture of immature embryo can be adopted to introgress resistance gene(s) (Rau et al. 1992). However, if the resistance gene(s) is harnessed from the wild species, often there is linkage drag bringing in undesirable agronomic traits along with resistance gene(s).

It is generally a long process requiring decades of crossing and selection to get rid of undesirable traits in an interspecific breeding program.

While focusing on breeding for resistance to virus diseases, it is essential that other agronomic traits also receive equal attention of the breeders. Otherwise, even if a variety is highly resistant, it may not be acceptable to farmers. For example, RG 1, the first GRD resistant peanut variety released in Malawi, was not accepted by the farmers because of difficulty in shelling its pods.

The molecular mechanisms underlying the roles of both dominant and recessive resistance genes have been elucidated, promoting the development of possible new viral control strategies (Ritzenthaler 2005). The concept of pathogen-derived resistance (Sanford and Johnston 1985) has stimulated research on obtaining virus resistance through genetic engineering. With the new developments in genetic engineering, it is now possible to access resistance gene(s) from unrelated species or sources. The genetic transformation of plants with gene(s) derived from virus genomes has been shown to confer useful levels of resistance to virus infection for a number of virus–host combinations (pathogen-derived resistance). Recent research indicates that pathogen-derived resistance to viruses is mediated, in most cases, by an RNA-based posttranscriptional gene silencing (PTGS) mechanism resulting in the degradation of mRNA produced by both the transgene and the virus. In general, protein-mediated resistance provides moderate protection against a broad range of related viruses while RNA-mediated resistance has been shown to offer high levels of protection only against closely related strains of a virus (Pang et al. 1993; Lomonossoff 1995; Baulcombe 1996; Dawson 1996). Several strategies are currently in use to generate virus-resistant transgenic plants that express virus-derived gene sequences. All such methods are based on the dependence of viruses on the host's metabolic apparatus for replication and spread, and on the accessibility of viral genomes, replication intermediates, and gene products in infected plant cells (Reimann-Philipp and Beachy 1993). The coat protein (*cp*) gene of the virus is the gene that has been used most widely in transgenics targeted at early stages of virus multiplication. The *cp*-mediated resistance can reduce virus infection and disease development for a number of different host–virus systems (Beachy et al. 1990). Different protocols are available to genetically transform peanut that include *Agrobacterium tumefaciens* mediated (Cheng et al. 1996; Sharma and Anjaiah 2000), particle bombardment of embryogenic callus derived from mature seeds (Livingstone and Birch 1999), and nontissue culture based transformation using embryo axes of mature seeds (Rohini and Rao 2000). Newly

emerging technology based on ribose nucleic acid interference (RNA*i*) offers another avenue to develop virus-resistant peanut genotypes (Wang et al. 2000; Colbere-Garapin et al. 2005). RNA*i* (RNA silencing or cosuppression of homologous genes) manifests as transcriptional or posttranscriptional gene silencing in plants (Baulcombe 2004).

Where a high level of genetic resistance is not available, a holistic approach combining genetic resistance, cultural management and chemical control may be required to manage the disease. With a few exceptions (such as GRD in Africa, PBND and PSND in India, and TSWV in the United States), breeding for virus resistance has received little attention in peanut due to the limited availability of virologists and suitable infrastructure in national programs in most of the developing countries.

B. Screening for Virus Resistance

A thorough knowledge of the epidemiology and properties of the virus and its strains, ecology of the vector(s), crop, cultivated and wild hosts of both virus and vector(s), virus–vector–host plant interactions and diagnostic tools is essential in devising effective field and laboratory screening techniques for virus resistance. Optimal environmental conditions, particularly in greenhouse screening, are needed to ensure virus infection and multiplication in the plant and promote vector activities, if they are used in inoculation. If facilities to control environmental conditions are not available in the greenhouse, the screening of genotypes should be done during the time of the year when the ambient environmental conditions are within the optimum range to promote virus intake and multiplication and vector activities leading to disease development.

Due to limitations of space only a limited number of plants/genotypes can be screened in the greenhouse. As a large number of plants/genotypes need to be screened for a successful breeding program, the initial screening should be carried out in the field. Promising genotypes, if required, should be further tested under greenhouse conditions. However, this will not be possible in the case of transgenic seeds that need to be screened initially under contained greenhouse conditions. In the case of seed-transmitted viruses, initial screening of seed material for virus contamination by enzyme-linked immunosorbant assay (ELISA) is essential. Seed tested positive should be eliminated from the screening trial.

1. Greenhouse Screening. Young and healthy plants of test materials and a susceptible control, uniform in stage of development, are needed

for greenhouse screening. Seeds should be sown in an appropriate medium in required sized pots and seedlings should be thinned to one per pot. Mechanically transmissible viruses can be inoculated by manual sap inoculation or by using an inoculation gun. If the virus is not readily sap transmissible, virus vectors, reared on infected plants, can be used for inoculation purpose. In some situations, graft inoculation may be required. After inoculation, the plants should be protected from infection from other viruses to avoid confusion at the time of recording observations. Environmental conditions in the greenhouse should be maintained within the optimum range to promote disease development.

Appearance and severity of symptoms and time taken to express symptoms often form the basis of screening. It is advisable to monitor presence of the virus in symptomless plants with sensitive serological or nucleic acid-based detection tools. In cases where inoculation response is highly variable in the plant population, from complete resistance to partial resistance with different grades of symptom intensities in between, scoring system often denoted by a "scale" can be used and a disease severity index devised (e.g., screening for groundnut rosette disease by Olorunju et al. 1991). However, in greenhouse screening, field tolerance differences may not be expressed sometimes as was observed in a TSWV screening study involving Southern Runner and Florunner peanut cultivars in Florida. The cultivar tolerance levels are influenced by the environment to which plants are exposed during development and growth (Pereira 1993; Pereira et al. 1995).

2. Field Screening. Field screening allows evaluation of a large number of genotypes/populations provided the disease with sufficient incidence and severity occurs each year at the location owing to the presence of vectors and of virus reservoir hosts nearby. Alternately, epidemics can be enhanced by growing infector rows of sensitive host plants of the vectors and the virus and interspersing infected host plants with vectors among the test plants in a disease screening nursery without the risk of mixed infection. Knowledge of vector ecology and patterns of primary and secondary spread helps in devising an effective field screening technique to ensure uniform spread of the disease in the screening nursery. In some cases, the sowing dates can be adjusted (such as late sowing in case of GRD in Africa and PBNV in rainy season in southern India) to ensure high disease incidence. The symptom-free test plants/genotypes should be evaluated for the presence or absence of virus by diagnostic tools. However, this does not ensure performance of test plants against different strains of the virus.

A multilocation screening for resistance, usually reserved for advanced generation materials, helps in exposing the genotype to diverse geographic isolates of a virus.

C. Mechanisms of Host Response to Virus Infection

Plant viruses show a very wide diversity of particle size, shape and structure, form and genetic structure of their genomes and in mechanisms by which the genome is expressed and replicated (Goldbach et al. 1990). They cause diverse forms of pathogenesis on their equally diverse host plants. Therefore, plants have evolved a variety of mechanisms of resistance to counter different types of virus attack (Fraser 1990). Three main types of mechanisms operating at different levels of host population complexity are discussed in the following sections.

1. Nonhost Resistance. At the level of plant species, if the whole species is immune to a particular virus, it is said to display nonhost resistance to that virus. For example, bean common mosaic virus (BCMV) is restricted to *Phaseolus vulgaris* and a few other leguminous hosts. All other species appear incapable of supporting multiplication and symptom development by BCMV.

2. Cultivar Resistance. In species, which are normally susceptible to a particular virus, some genotypes may display heritable resistance to it. Sometimes this resistance might have coevolved with the pathogen or it could have been introduced by natural or manual outcrossing with resistant individuals of related wild species. This cultivar resistance is the form used by plant breeders in practical crop protection. However, virulent strains of the virus with the ability to overcome particular resistance gene(s) have evolved in many cases.

3. Acquired Resistance. When a prior infection or chemical or cultural treatment induces resistance in susceptible individuals of the species, it is called acquired resistance (sometimes called induced resistance). Generally, this type of resistance is not heritable. However, genetic transformation of susceptible plants with DNA copies of portions of viral genome or associated nucleic acid can result in heritable resistance. Under control of a suitable promoter to ensure expression of active RNA or protein moieties, their transgenes can confer resistance to infection by the whole virus. If they are stably integrated into the host chromosome, they are heritable and affect a lasting form equivalent to cultivar resistance.

Host plant reaction to virus infection can be broadly classified as immune, resistant, tolerant, and susceptible (Walkey 1985). In immune host, virus is not detected despite repeated inoculations. This is because cells of the immune host lack surface receptors to facilitate virus particle adsorption and entry, or virus particles may enter into cells, but cell machinery does not support the replication of virus nucleic acid or due to both factors. In immunity under field conditions, the virus is not detected under natural virus transmission conditions and conditions typical to the crop environment. However, immunity of such hosts can be overcome through artificial inoculation under conditions favorable for virus transmission and/or for the vector. A susceptible host readily supports rapid virus infection, multiplication, and systemic movement in the host. But a resistant host does not readily support virus infection, multiplication, or movement. In case of passive resistance, the host resists virus entry. However, if the virus enters into the cells, it can multiply and invade it as in susceptible host. This kind of response is mainly due to plant resistance to vector (vector resistance), lack of surface receptors permitting virus entry or interference with virus adsorption to cells. In case of active resistance, the host resists virus replication or translation of its products. This response, sometimes, is influenced by abiotic factors (such as temperature), which can influence cell functions, thus can result in varied host response to virus infection. In the case of hypersensitive reaction, the death of virus-infected cell (localized necrosis) occurs to minimize the rate and extent of virus invasion. The presence of various forms of resistance separately or in combination minimizing incidence of infection in an infectible plant under field conditions is termed field resistance. The tolerance reaction shows only mild symptoms without marked effect on plant growth and vigor or yield. This kind of host response may or may not correlate with virus concentration in the cells. The host may support normal rate of virus multiplication, but show only mild symptoms, such host is susceptible to virus infection, but resistant to disease. If host restricts virus multiplication leading to decrease in virus concentration and show mild symptoms, this host is resistant to virus and also to disease. In latent host, the virus can multiply and invade without causing any effect on the growth, and such plants do not show any symptoms. However, a sensitive host shows conspicuous symptoms markedly affecting the growth pattern and often leading to the plant death. In some cases sensitive reaction depends on the stage at which virus infection occurs.

The dominant or recessive nature of resistance alleles affects the symptom expression in the host. While dominant resistance alleles are

strongly associated with virus localizing mechanisms normally involving local lesions, incomplete dominant and recessive alleles allow spread of virus, but inhibit multiplication or symptom development. Fully recessive alleles may be associated with complete immunity (Fraser 1992).

D. Screening for Vector Resistance

Field resistance to virus diseases observed in some genotypes can also result from their resistance to the vector; although, these genotypes may be susceptible to the virus in artificial inoculation. A number of factors (physical barriers such as leaf hairs or robust leaf surfaces, nonpreferred foliage color, secretion of insect alarm pheromones, and presence in the sap of antifeedant chemicals which reduce feeding time and thus time for virus acquisition and transmission) can affect plant attractiveness to vectors, thereby affecting the efficiency of virus transmission (Jones 1990). Vector-resistant genotypes coupled with the required cultural adjustments can give, barring severe epidemic conditions, significant protection to the crop under normal growing conditions. For maximum gains, it is desirable to have resistance to both vector and virus. Such genotypes will also be able to withstand direct damage caused by the insect vector. Screening procedures for resistance to common insect pests are described in detail by Ranga Rao and Wightman (1997).

III. BREEDING FOR RESISTANCE TO VIRUS DISEASES

A. Groundnut Rosette Disease

An overview of the groundnut rosette disease, properties of the etiological agents, protocols for their detection, information on germplasm screening and resistant sources, and various management options is given in Waliyar et al. (2007).

1. Occurrence. Groundnut rosette disease was first reported by Zimmerman (1907) in Tanganyika (presently Tanzania). The disease remains endemic to the African continent and its offshore islands. Earlier reports on its occurrence based on rosettelike symptoms elsewhere outside Africa were not substantiated as these were found to be caused by other viruses.

GRD occurs in small proportions each growing season. However, its severity increases in late-sown crops (Sauger et al. 1954). A very

late-sown crop may produce little or no yield. In rain-dependent subsistence farming in Africa, where most of the agricultural operations are done manually, peanut is often sown late as the priority goes to staple food crops.

Recurrent GRD epidemics cripple the rural economy of the affected countries in Africa. A severe epidemic has a long lasting effect on peanut production as near total failure of the crop affects seed availability in subsequent years. The 1975 GRD epidemic in Nigeria affected 0.75 million ha of peanut causing an estimated loss of US$ 250 million in regional trade (Yayock et al. 1976). In 1995, GRD epidemic affected approximately 43,000 ha of peanut in Eastern Zambia with an estimated loss of US$ 4.89 million. In the following year, peanut production in Malawi was reduced by 23% due to GRD epidemic (Waliyar et al. 2007). Origin and perpetuation of GRD in nature still remains a mystery.

2. Symptoms. There are two major symptom variants of GRD-chlorotic rosette (Storey and Bottomley 1928) and green rosette (Hayes 1932), each with considerable variation (Murant 1989; Naidu et al. 1998a, 1999). Both forms of the disease cause stunting, shortened internodes, and reduced leaf size resulting in bushy appearance of the plants. Usually bright yellow curled/distorted leaves with a few green islands are the symptoms of chlorotic rosette. These symptoms may appear over almost the entire plant, or only in parts of the plant, affecting perhaps some shoots but not others, or the distal portions of the shoots but not the proximal portions. In green rosette, leaves appear dark green with light green to dark green mosaic and are much reduced in size. The stunting is also severe in the case of green rosette. Chlorotic rosette occurs throughout the sub-Saharan Africa. Green rosette, which was earlier confined to West Africa, has now been reported from several countries of Southern and Eastern Africa (Naidu et al. 1999). There is a less common symptom variant, mosaic rosette, due to mixed infection of plants by the satellite-RNA (*Sat*RNA) causing chlorotic variant and mottle variant reported from East Africa (Storey and Ryland 1957). Variability in *Sat*RNA is mainly responsible for symptom variation (Murant and Kumar 1990; Taliansky and Robinson 1997).

3. Virus Causal Agents and Their Characteristics. Three causal agents, groundnut rosette assistor virus (GRAV), a member of the family Luteoviridae, groundnut rosette virus (GRV), belonging to genus *Umbravirus* and a *Sat*RNA, belonging to Subgroup-2 satellite RNAs, are involved in GRD etiology (Reddy et al. 1985a,b; Murant et al. 1988; Taliansky et al. 2000). These three agents synergistically interact with

each other for survival and spread. GRV is dependent upon GRAV for transmission by its aphid vector (Hull and Adams 1968) and *Sat*RNA (which is largely responsible for rosette symptoms in peanut) is itself dependent on GRV for replication (Murant et al. 1988). GRV and *Sat*RNA do not produce GRD symptoms. GRAV on its own can cause mild yellowing/chlorosis of leaves, which persists throughout the growth period and can cause reduction in plant growth and significant yield loss in susceptible peanut cultivars (Naidu and Kimmins 2007). GRV and *Sat*RNA must be packaged within the GRAV *coat protein* (GRAVcp) to be aphid transmissible. GRV is dependent on its *Sat*RNA for encapsidation in coat protein. GRV on its own causes transient symptoms only. GRV and *Sat*RNA have always been found together in nature. Expression of disease symptoms does not necessarily indicate the presence of aphid-transmissible GRAV in infected plants (Naidu et al. 1998b). Plants that show GRD symptoms but lack GRAV play no role in the spread of disease. GRAV plays a crucial role in the epidemiology and perpetuation of GRD.

The three agents of GRD are not seedborne. GRAV can be transmitted by grafting but not by mechanical sap inoculation, whereas GRV can be transmitted by both. There are no reports on occurrence of strains of GRAV and GRV. *Sat*RNA is transmissible mechanically along with GRV and by aphids in the presence of GRV and GRAV.

4. Vector and its Characteristics. Aphid, *A. craccivora*, is the principal vector of GRD agents. In the tropics, only females have been recorded and they reproduce parthenogenetically throughout the year. The GRD agents are acquired by aphids from the phloem sap. Once acquired, aphids can potentially transmit virus particles up to 14 days and possibly for life (Storey and Ryland 1955; Watson and Okusanya 1967; Dubern 1980; Misari et al. 1988). GRD is regarded as a polycyclic disease because each infected plant can potentially serve as a source for initiating subsequent spread of disease in the field. As the virus agents are not seedborne, the primary infection must come from outside source through viluriferous aphids. The secondary spread of disease in a field is through apterae and nymphs (Evans 1954; Booker 1963; Davies 1972; Farrell 1976a,b). Factors such as plant age, crop density, timing and efficiency of transmission by viruliferous aphids, proximity to the source of primary inoculation, climatic factors, and predators and parasitoids of vector population within the crop affect nature and pattern of spread of the disease.

5. Alternate Hosts of Virus. The source of viruliferous aphids that initiate GRD remains unknown. Peanut and some of its wild relatives

are the only known natural hosts of GRAV, GRV, and *Sat*RNA (Okusanya and Watson 1966). However, GRV and *Sat*RNA, following mechanical sap inoculation, have several experimental hosts in Leguminosae, Chenopodiaceae, and Solanaceae families (Okusanya and Watson 1966; Adams 1967; Hull and Adams 1968; Dubern 1980; Reddy et al. 1985a,b; Murant 1989; Murant et al. 1998). *Chenopodium amaranticolor* and *Chenopodium murale* are local lesion hosts; *C. amaranticolor, Glycine max, P. vulgaris, Nicotiana benthamiana,* and *Nicotiana clevelandii* are systemic hosts of GRV. Using viruliferous aphids, GRAV has been transmitted to *Pisum sativum* L., *Stylosanthes gracilis* Taub., *Stylosanthes hamata* (L.) Taub., *Stylosanthes mucronata* Wild., *Stylosanthes sundaica* Taub., *Trifolium incarnatum* L., *Trifolium pretense* L., *Caspella bursa-pastoris* (L.) Medicus, *Gomphrena globosa* L., *Montia perfoliata* L., and *Spinacia oleracea* L. (Okusanya and Watson 1966; Adams 1967; Hull and Adams 1968; Murant 1989). All these plants except *C. bursa-pastoris* (L.) Medicus show symptomless infection and virus application are confirmed by diagnostic assays.

Apart from the natural host, peanut and some of its wild relatives, experimental hosts of GRAV and GRV including *Sat*RNA are *G. globosa, S. gracilis, S. mucronata, S. sundaica, S. oleracea, T. incarnatum,* and *Trifolium repens* (Murant 1989; Murant et al. 1998).

6. Conventional Genetic Improvement

Screening Methods/Techniques. Bock and Nigam (1988) developed a field screening technique for resistance to GRV (and accompanying *Sat*RNA), which involved management of a field disease nursery during the cropping season and subsequent controlled greenhouse screening tests of apparently healthy field survivors. In Malawi, only primary infections give rise to typical patches of disease in the fields. At normal sowing time, one infector row of a susceptible variety was sown between two contiguous rows of test lines. About 1 week after seedling emergence, diseased seedlings heavily infested with vectors, raised earlier in greenhouse, were transplanted at 1.5 m spacing in each of the infector rows. Infestation with viruliferous aphids harvested from greenhouse cultures was done several times. With this method up to 98% disease incidence among the susceptible genotypes could be created in the field. To raise diseased seedlings, a large number of seedlings of a susceptible variety were raised in greenhouse prior to normal sowing time. These seedlings were inoculated with GRV, and dense populations of viruliferous apterae were allowed to develop on the infected plants. Apparently healthy plants in the field (or survivors) consist of "escapes" and resistant plants

(resistance being governed by two recessive genes). In the following dry season, seedlings from seeds of survivor plants are raised in the greenhouse and screened. The seedlings originating from resistant plants maintain their resistance whereas those from "escapes" succumb to the disease. This method of field screening has successfully been used in Nigeria (Olorunju et al. 1991). As described earlier, this method of screening provides an effective and rapid method for large-scale testing of breeding populations but only against GRV and its *Sat*RNA. The GRAV was detected in all the resistant plants.

Under greenhouse conditions, genotypes can be screened for resistance to GRV and *Sat*RNA by mechanical sap inoculation. For screening for resistance to GRAV, test plants need to be inoculated with viruliferous aphids fed on GRAV infected plants or by grafting scion from GRAV-infected plants (Olorunju et al. 1992; Naidu and Kimmins 2007).

Olorunju et al. (1991) used a Disease Severity Index (DSI), based on a 1–5 scale of individual plant reaction to the disease, to determine the disease reaction of a genotype under field conditions. In this method, disease severity of each plant is scored using a 1–5 scale, where 1 = plant with no visible disease symptoms on foliage, 2 = plant with obvious rosette symptoms but no stunting (1%–20% foliage affected), 3 = plant with rosette symptoms plus stunting (21%–50% foliage affected), 4 = plant with severe rosette leaf symptoms plus stunting (51%–70% foliage affected), and 5 = plant with severe rosette leaf symptoms and stunting or dead plants (71%–100% foliage affected). The disease scores of individual plants are used to calculate a DSI for each genotype as follows: DSI = $(1\ A + 2\ B + 3\ C + 4\ D + 5\ E)$/total number of plants assessed in a genotype, where *A, B, C, D,* and *E* are the number of plants with ratings of 1, 2, 3, 4, and 5, respectively. The DSI varies from 1.0 (no diseased plants) to 5.0 (uniform mortality of all plants). Subrahmanyam et al. (1998) modified the method of Olorunju et al. (1991) by reducing the individual plant disease scoring scale to a 1–3 scale, where 1 = plants with no visible disease symptoms on foliage and no stunting, 2 = plants with obvious rosette leaf symptoms stunted to about 50% of the size of symptomless plants, and 3 = plants with severe rosette leaf symptoms and stunting greater than 50%. The calculation of DSI was unchanged.

Genetics of Virus and Vector Resistance. De Berchoux (1960) was the first one to show that resistance to rosette disease (effective against GRV and its *sat*RNA) was governed by two independent recessive genes. He also stated that resistant lines were not immune and that individual plants could become infected with GRV when subjected to inoculation

by massive number of aphids. This resistance was reported to operate equally against both chlorotic rosette (De Berchoux 1960) and green rosette (Harkness 1977). Harkness (1977) attributed low recovery of resistant plants from Virginia × Spanish crosses to heavy inoculum pressure at early stages of plant growth and suggested that loss of resistance from generation to generation if recessive genes did not confer resistance in all nuclear backgrounds. Nigam and Bock (1990) studied inheritance of resistance to chlorotic rosette (GRV and its *Sat*RNA) in crosses involving different botanical varieties of peanut in Malawi and confirmed findings of De Berchoux (1960) of two recessive genes governing the resistance in all backgrounds. In all the resistant plants, the presence of GRAV was detected. Genes conferring resistance to GRV and its *Sat*RNA did not confer resistance to GRAV (Bock and Nigam 1988; Bock et al. 1990). Similar findings on the inheritance of resistance to green rosette using mixed infection in the field (GRV + *Sat*RNA + GRAV) and single GRV infection under greenhouse conditions were reported from Nigeria by Olorunju et al. (1992). There was one exception of RMP 12 × M 1204.781 cross, where in the F_2 generation, the plants segregated into 1 susceptible:3 resistant. Resistance to GRAV has not yet been identified (Chiyembekeza et al. 1997).

Amin (1985b) reported high levels of resistance to *A. craccivora* in *Arachis chacoense*, *Arachis villosa*, *Arachis correntina*, and *Arachis glabrata* wild species under screenhouse conditions. Progenies of *A. chacoense* and *A. villosa* interspecific derivatives with cultivated peanut also showed high resistance to the insect pest. Resistance to aphid vectors, identified in cultivated peanut, ICG 5240 (EC 36892) (Padgham et al. 1990) and ICG 12991 (Minja et al. 1999), is controlled by a single recessive gene (van der Merwe 2001; Herselman et al. 2004). The basis of resistance to aphid is antibiosis (Grayer et al. 1992). Mapping of this resistance gene in a segregating F_2 population derived from a cross between the aphid-resistant female parent ICG 12991 and the aphid susceptible male parent on linkage Group-1, at a distance of 3.9 cM from a marker originating from susceptible parent IGV-SM 93541 that explained 76.1% of the phenotypic variation for aphid resistance (Herselman et al. 2004) offers possibilities for marker-assisted selection for vector resistance in peanut. Aphid-resistant sources are, however, susceptible to GRV, *Sat*RNA, and GRAV (Minja et al. 1999).

Resistant Sources. Resistance to GRD in cultivated peanut was discovered in 1952 in late maturing Virginia (semierect) type pure lines of landraces (48-7, 48-14, 48-15 A, 48-21, 48-34, 48-35, 48-36, 48-37, 48-44, 48-45, and 48-70 A) under severe natural attack of rosette in

Burkina Faso (Sauger and Catherinet 1954; Sauger et al. 1954; De Berchoux 1958). Their resistance was further confirmed following graft and aphid transmission under artificial inoculation (Sauger et al. 1954). These lines also maintained their resistance at locations in Senegal and Cote d′ Ivoire. However, these lines were not immune under a very heavy infestation of viruliferous aphids. The bordering region between Burkina Faso and Cote d′ Ivoire can be considered as source of resistance genes (De Berchoux 1958).

Subrahmanyam et al. (1998) screened approximately 6,800 accessions of the cultivated peanut originating from South America, Africa, and Asia using an infector row technique (Bock and Nigam 1988) and disease rating system adopted from Olorunju et al. (1991) between the 1990/1991 and 1996/1997 growing seasons. They found 116 of these accessions, including 15 short-duration Spanish types, highly resistant to GRD. Most of these Virginia-type resistant accessions originated from West Africa and were either breeding lines involving original West African sources of resistance in their parentage or landraces. They all tested positive for GRAV, but there was quantitative variation in the level of GRAV accumulation. ICG 11044 (PI 162525), a long-duration Virginia (semierect) type originating from Argentina, was the only resistant germplasm line originating from South America. ICG 9723 (VRR 731), ICG 11735 (RV 055), ICG 11767 (RV 093), ICG 11788 (RV 115), ICG 12622 (RAP 154), ICG 12678 (RV 14), ICG 12680 (RV 15), and ICG 13063 (GSS 181) from India; ICG 10347 (Lok Wow, PI 445925) and ICG 11649 (Lianzhan) from China; and ICG 12876 (RT 12) from Myanmar were the other Virginia-type resistant sources outside Africa. Among the Spanish-type resistant sources, ICG 12988 (US 22), ICG 12989 (US 23), ICG 12991 (US 25), and ICG 12992 (US 26) originated from India.

In another screening of 2,301 cultivated germplasm lines from different sources and 252 advanced breeding lines derived from crosses involving earlier identified sources of resistance to GRD in Nigeria, 65 germplasm lines (55 long-duration Virginia types and 10 short-duration Spanish types) showed high levels of resistance, and 134 breeding lines were also resistant (Olorunju et al. 2001). Out of the 65 germplasm lines, 42 were also found resistant in Malawi (Subrahmanyam et al. 1998). Most of the breeding lines owed their resistance to RMP 40, RMP 91, and RG 1 genotypes. As these resistant sources were evaluated based on disease symptoms, they are resistant to GRV and its *Sat*RNA. However, it is not known whether these resistant sources carry the same or different resistance genes. It would be interesting to initiate studies on allelic relationship among the resistant genotypes. Subrahmanyam

et al. (1998) reported quantitative resistance to GRAV in symptom showing partially infected plants of GRV-resistant germplasm lines and suggested its exploitation in breeding programs. As plants with low GRAV accumulation would be poor source for virus acquisition by aphid vector in the field, further spread of the disease will be lowered as is shown with other persistently transmitted luteoviruses (Barker and Harrison 1986; Gray et al. 1994).

West Africa is considered the tertiary center of diversity of cultivated peanut (Gibbons et al. 1972). It would be interesting to study allelic relationship of resistant sources originating from outside Africa with the original resistant sources reported from West Africa. In the gene bank records at ICRISAT "origin" means the country from which an accession was obtained. There has been a lot of exchange of peanut germplasm among the countries in the past without proper documentation and records.

Twenty-five accessions belonging to nine wild *Arachis* species (*A. appressipila*—ICG # 8127, 8945, and 14860; *A. decora*—ICG 14946; *A. diogoi*—ICG 4983; *A. hoehnei*—ICG # 8190 and 13232; *A. kretschmeri*—ICG # 8191, 8216, 11558, and 13224; *A. kuhlmannii*—ICG # 13225, 14862, and 14875; *A. pintoi*—ICG # 13222, 14855, 14856, and 14888; *A. stenosperma*—ICG # 13171, 13173, 13187, 13210, and 14872; and *A. villosa*—ICG 13168) showed resistance to GRD when 116 accessions representing 28 species were evaluated in Malawi (Subrahmanyam et al. 2001). It is likely that these resistant sources belonging to wild *Arachis* species may carry different resistance genes. Murant et al. (1991) reported *A. chacoense* as immune to both GRV and GRAV.

Two genotypes, ICG 5240 (EC 36892) (Padgham et al. 1990) and ICG 12991 (Minja et al. 1999), are reported to be aphid resistant in cultivated peanut. EC 36892 was also found resistant to aphids in China (Zeyong et al. 1995) and Nigeria (Ozigi and Olorunju 1997). Minja et al. (1999) found higher resistance to aphids in ICG 12991 than ICG 5240 in Malawi. However, both are susceptible to all three agents of GRD. Under natural disease pressure, ICG 12991 showed less GRD than that in a susceptible cultivar (Minja et al. 1999; Naidu et al. 1999).

Resistant Cultivars Released. Using resistant sources identified in Burkina Faso, many rosette-resistant cultivars, both in the Virginia and the Spanish groups, have been released in Africa (Table 4.1). Several other genotypes (ICGV-IS # 96808, 96814, 96855, and 98891 and ICIAR 19BT) are at the prerelease stage in Nigeria.

In all the resistance breeding programs in Africa, resistance genes have come from the same sources. Under natural disease pressure these

Table 4.1. Rosette-resistant cultivars released in Africa.

Cultivars	Origin	Botanical type	Release date	Country
RMP 12	Burkina Faso	Virginia	1963	Burkina Faso, other wetter zones of West Africa
RMP 91	Burkina Faso	Virginia	1963	Burkina Faso
69-101	Senegal	Virginia	1969	Senegal
KH 241 D	Burkina Faso	Spanish	1964	Burkina Faso
KH 149 A	Burkina Faso	Spanish	1964	Burkina Faso
RG 1	Malawi	Virginia	1976	Malawi
Nyanda (ICGV 93437)	ICRISAT, India	Spanish	1999	Zimbabwe
ICGV-SM 90704	ICRISAT, Malawi	Virginia	2000	Malawi, Mozambique, Zambia, Uganda
ICG 12991 (US 25)	Madhya Pradesh, India	Spanish	2001	Malawi, Mozambique, Zambia, Uganda
Chitala				
ICGV-SM 99568	ICRISAT, Malawi	Spanish	2005	Malawi
ICGV-SM 99555	ICRISAT, Malawi	Spanish	2009	Tanzania
ICGV-SM 99557	ICRISAT, Malawi	Spanish	2009	Tanzania
ICGV-SM 01711	ICRISAT, Malawi	Virginia	2009	Tanzania
ICGV-SM 01721	ICRISAT, Malawi	Virginia	2009	Tanzania
Samnut 23 (ICGV-IS 96894)	ICRISAT, Nigeria	Spanish	2001	Nigeria
Samnut 21 (UGA 2)	IAR, Nigeria	Virginia	2001	Nigeria
Samnut 22 (M 572.801)	IAR, Nigeria	Virginia	2001	Nigeria

Source: Mayeux et al. (2003) and Bockelee-Morvan (1983).

genes have maintained their resistant reaction even after more than 60 years of their discovery and have provided succor to peanut farmers in Africa. Only under very heavy infestation by viruliferous aphids do they show some disease symptoms in some plants or some parts of the plants. All of these varieties are resistant to GRV and its *Sat*RNA. But they do not carry resistance to GRAV, which can cause significant yield loss without expressing symptoms (Naidu and Kimmins 2007). It is, therefore, essential to identify resistance to GRAV and incorporate in new cultivars along with that of GRV and *Sat*RNA.

7. Nonconventional Genetic Improvement. Pathogen-derived resistance (PDR) represents a potential strategy for controlling GRD through

the generation of transgenic peanut. Introduction of GRAV or GRV genomic sequences or genes, or *Sat*RNA-derived sequences that down-regulate GRV replication (Taliansky et al. 1996) into suitable peanut cultivars is an ideal approach. However, the success of PDR-, RNA-, or protein-mediated resistance is highly influenced by the degree of sequence homology between the sequence of the transgene and the challenging virus. A high level of sequence homology that exists within the GRAV*cp* genes (97%–99%) clearly indicates that PDR from the GRAV*cp* gene would likely be functional. Similarly, the transformation of *N. benthamiana* with full-length sequences of a mild variant of the GRV *Sat*RNA yielded plants that did not produce symptoms when inoculated with GRV and a virulent *Sat*RNA (Taliansky et al. 1998). This study showed two different mechanisms of resistance that operated in different transgenic events. The first mechanism included plants containing high levels of transcript RNA where the replication of both *Sat*RNA and GRV genomic RNA was inhibited. In the second mechanism, plants contained low transcript RNA levels where the replication of *Sat*RNA but not of GRV genomic RNA was inhibited. This provides another approach of developing transgenic resistance to GRD.

At ICRISAT, peanut transgenic plants were developed using GRAV*cp* gene to induce host plant resistance to GRD (Sharma et al. unpublished). The approach seemed promising also because of a number of reasons including a possible reduction in GRAV particles available for transmission due to expression of GRAV*cp* in transgenic plants which could be either due to gene silencing (RNA-mediated) or *cp*-mediated resistance. Second, since the *cp* of GRAV is required for the encapsidation of the GRV genome and *Sat*RNA, the absence of or reduction in GRAV*cp* would presumably result in diminished packaging of GRV and *Sat*RNA and, subsequently, reduced aphid transmission. The selected transgenic events following their molecular characterization have been transferred to South Africa (because GRD does not occur in India) for phenotyping under greenhouse conditions to assess their effectiveness against the disease pressure.

B. Peanut Bud Necrosis Disease

Occurrence of a disease with symptoms similar to those of peanut bud necrosis disease (PBND) was mentioned in the Annual Report of the Indian Agricultural Research Institute, New Delhi in 1949. This appears to be the first record of occurrence of PBND in India. The name "Bud Necrosis" was given in 1968 and the disease was considered to be

distinct at that time because none of the other peanut virus diseases reported until 1968 were known to produce the bud necrosis symptoms (Reddy et al. 1968). Since 1962, based on symptoms, the disease has been referred to by different names (groundnut mosaic, groundnut rosette, bunchy top, chlorosis, ring mottle, bud blight, and ring mosaic) in literature published by the Indian scientists. The disease was first noticed in farmers' fields in Punjab during 1958–1959. In southern India, it was first noticed in 1962 in Hyderabad.

Since the review by Reddy and Wightman (1988), several advances in knowledge helped to remove confusion in the identity of causal agent of PBND and its vector. It is now well established that PBND is caused by PBNV and the virus is transmitted by *Thrips palmi* and not by *Frankliniella schultzei* (Vijaya Lakshmi 1994).

1. Occurrence. PBND is currently recognized as economically important in south and southeast Asian countries such as India, Nepal, Sri Lanka, Thailand, and parts of China (Reddy et al. 1995).

2. Symptoms. Primary symptoms include appearance of faint chlorotic spots or mottling on young leaflets that may develop into chlorotic and necrotic rings and streaks. Occasionally, the leaflets may show a general chlorosis with green islands. Necrosis of the terminal bud soon follows. If disease occurs in plants less than 1 month old, total necrosis of the plant may follow. Necrosis on older plants usually spreads only to the petiole, or to the portion of the stem immediately below the terminal bud. Stunting and proliferation of axillary shoots are common secondary symptoms. Leaflets formed on these axillary shoots show a wide range of symptoms including reduction in size, distortion of the lamina, mosaic mottling, and general chlorosis. Seeds from early-infected plants are small shriveled and their testae show red, brown, or purple mottling. Seeds from virus-infected plants contain less oil (Mohamed Ali and Prasada Rao 1982).

3. Virus Causal Agent and its Characteristics. Until 1990, PBND in India was reported to be caused by tomato spotted wilt virus (Reddy et al. 1991b). However, when better and more sensitive detection tools became available, it was realized that the causal virus of PBND was distinct from TSWV, and it was named PBNV (Reddy et al. 1992). PBNV is not seed transmitted in peanut. The PBNV genome contains three RNA species. The sRNA has been sequenced and the two genes it codes have been identified (Satyanarayana et al. 1995).

4. Vector and its Characteristics. Like the initial wrong identification of the virus causal agent, the earlier reported thrips vectors, *F. schultzei* and *Scirtothrips dorsalis* (Amin et al. 1981), were found incorrect. Subsequent detailed studies reported that *T. palmi* was the vector of PBNV although the earlier reported vectors were also present on the plant. PBNV is acquired by larvae of *T. palmi* but the transmission is exclusively due to adult thrips (Vijaya Lakshmi et al. 1995). The virus is transmitted in a persistent manner by the vector thrips which, under optimum conditions, retain the virus throughout their life. At two sites near Hyderabad, India, Vijaya Lakshmi (1994) found *T. Palmi* on 44 of 64 cultivated plant species and on 27 of 45 wild plant species. Primary infection is the major source of disease spread in the field (Buiel 1996).

5. Alternate Hosts of Virus. Both PBNV and the vector thrips have wide host ranges that include crop plants, ornamentals, and weeds. The virus may survive in these hosts and provide an inoculum source for thrips. Crop plants such as chilli (*Capsicum annuum*), tomato (*Lycopersicon esculentum*), brinjal (*Solanum melongena*), green gram (*Vigna radiata*), and black gram (*Vigna mungo*), ornamentals such as zinnia and chrysanthemum, weeds such as *Ageratum conyzoides* and *Cassia tora* are frequently present in and near peanut fields and serve as alternate hosts.

6. Conventional Genetic Improvement

Screening Methods/Techniques. Field screening for resistance to PBND is done under natural conditions at hot spot locations of the disease. Wider row spacing and planting of infector rows of cowpea to attract thrips and adjusting sowing time so as to catch thrips migration peak at seedling stage are some of the cultural practices used to enhance uniform PSND pressure in the field screening nursery. Since plant age and age of the inoculated leaves delay the disease incidence even in a susceptible variety (Buiel and Parlevliet 1996), it is important to create virus inoculum and disease pressure at early plant stage to avoid/reduce plant escapes in the screening nursery.

In Thailand, Pensuk et al. (2002) found field disease incidence at 50 or 60 DAS as most appropriate parameter to identify resistance to PBNV in peanut genotypes. They also suggested use of area under the disease progressive curve (ADPC) as an alternative to disease incidence in discerning peanut genotypes for PBNV resistance. As mechanical sap inoculation under greenhouse conditions gave similar results as field disease incidences in their study, they suggested use of mechanical sap inoculation to effectively screen genotypes for resistance to PBNV.

Since no correlation was observed between number of thrips per plant and the incidence of the disease under field conditions, Nagaraja et al. (2005) suggested that resistance to the vector may not be of much help under epidemic conditions. Instead, Ekvised et al. (2006) suggested use of percentage of damaged plants, percentage of damaged leaves, and thrips damage rating in screening of genotypes as they had relatively higher heritability than the thrips number. Dwivedi et al. (1995) used a 1–9 scale for scoring thrips injury, where 1 = highly resistant, 2–3 = resistant, 4–5 = moderately resistant, 6–7 = susceptible, and 8–9 = highly susceptible. The vector-resistant genotypes were subsequently tested for PBNV resistance by mechanical inoculation under controlled greenhouse conditions.

Quantitative resistance to PBNV is determined from the level of disease incidence, that is, the percentage of plants with symptoms. Since PBNV isolates from different locations in India reacted with polyclonal antiserum (Reddy et al. 1992) and with 10 monoclonal antibodies developed against the nucleocapsid protein (Poul et al. 1992), the resistance screening at any one location should result in identification of resistant genotypes which will maintain their resistance at other locations also.

Genetics of Virus and Vector Resistance. From a half-diallel field study in India involving five quantitatively (% disease infection) resistant parents and two susceptible cultivars, Buiel (1996) reported at least three resistance factors responsible for reduced disease incidence, which were inherited additively. Dominance and epistatis factors were apparently absent. The resistance was also reported to be stable across environments.

In a 6×6 F_1 and F_2 diallel field study in Thailand, Pensuk et al. (2002) found highly significant general combining ability (GCA) effects and significant specific combining ability (SCA) and reciprocal effects for PBND incidence. However, the relative contribution of SCA and reciprocal effects was much less than that of GCA. The performance of the parental line was a good indicator of GCA of the line. Due to significant reciprocal effects they suggested use of resistant sources as female parents. ICGV 86388, IC 10, and IC 34 were found to be good combiners for PBND resistance. In another field study of generation mean analysis of populations derived from three crosses among two resistant parents (ICGV 86388 and IC 10) and a susceptible parent (KK 60-1), Pensuk et al. (2004), however, reported nonadditive gene effects for low PBND incidence and suggested that selection may be delayed to later generations. They also reported some genetic differences for PBND resistance in

these two resistant parents. As both studies were carried out in the field, it is not clear whether the resistance to the disease was due to the resistance to the vector or the virus or the plants were escape as only selected diseased plants were tested for the presence of the virus. This could be one of the reasons for differing results obtained by the same group of authors and with the same material involved in both the studies.

Following Hayman's model and Gamble's notations in eight generations of three crosses, Poledate et al. (2007) reported differing gene effects. Additive gene effects were most important contributors to genetic variation in generation means for both disease incidence and disease severity in ICGV 86388 × IC 10 cross. In ICGV 86388 × KK 60-1 cross, additive gene effects and additive × additive epistatic gene effects were important for disease incidence. The dominance gene effects were also significant in this cross for disease incidence. Additive gene effects were significant for disease incidence only in cross IC 10 × KK 60-1. Depending up on the gene effects operating in these crosses, they suggested cross specific selection strategy.

Niyomsil et al. (2007) found dominance and epistatic gene effects significant in cross IC 10 × Khoen Kaen 60-1 cross for thrips damage. The additive gene effect was nonsignificant.

Resistant Sources. The reduced disease incidence in the field can be due to resistance to the vector or to the virus or a combined resistance to both. In the field, genotypes differ considerably in the incidence of PBND due to combined effects of resistance to the virus and resistance to the vector. Reduced disease incidences are recorded as field resistance. In virus-resistant/-tolerant genotypes resulting in reduced disease incidence there is reduced virus multiplication at the site of infection and subsequent systemic spread (Buiel 1996).

More than 8,000 germplasm accessions were screened at ICRISAT Center under field conditions for resistance to PBND. Several germplasm accessions belonging to subspecies *hypogaea* (ICG # 848, 851, 852, 862, 869, 885, 2271, 2306, 2307, 2323, 2741, 3042, 3806, 3873, 5030, 5024, 5043, 5044, 6135, 6317, 6323, 7676, and 7892) showed consistently low disease incidence (field resistance). Eight accessions of wild *Arachis* species [*A. duranensis* (ICG 8199 (PI 468200), ICG 8956 (PI 468201), ICG 11552 (PI 475882), ICG 11553 (PI 475882), and ICG 11555 (PI 475885)) and one accession each of *A. valida* (ICG 8193 (PI 468154)), *A. correntina* (ICG 8132 (PI 262808)), and *A. monticola* (ICG 8189 (PI 468199))] did not show disease under field conditions (Dwivedi et al. 1995). Other than *A. volida*, all of these species are cross compatible with cultivated peanut. In field and laboratory studies, Reddy et al. (2000) found three accessions of *A. cardenasii* (ICG

11564, 13164, and 13165) and two accessions of *A. villosa* (ICG # 8144 and 13168) free from virus in the field and absence of virus in systemic leaves in spite of repeated inoculation. Both the species are cross compatible with cultivated peanut. Among the several breeding lines with vector resistance, ICGV # 86031 and 86388 showed resistance to PBNV, when mechanically sap-inoculated with low virus concentration (Dwivedi et al. 1995). Among 172 genotypes of cultivated peanut field screened for 3 years in Karnataka, India, Gururaj et al. (2002) found 7 genotypes, DRG 18, ICG 7812, ICG(FDRS) 10, ICGV 80325, JSSP 3, KNG 22, and PI 393516 as highly resistant to PBND (0%–1% disease incidence). From three seasons' field screening, Gopal et al. (2004) reported ICGV 92269, 89/94-3-2, 83/151-7, and 85/203-6 as showing consistently low disease incidence in Andhra Pradesh, India.

Amin et al. (1985) observed consistently low damage rating or a low percentage of damaged foliage by thrips in the following *A. hypogaea* genotypes: NC Ac # 2242, 2214, 2243, 2240, 2232, and 2230. Antixenosis mechanism (nonpreference) and dark green leaves (Amin 1985a) are some of the factors associated with resistance to thrips. Pensuk et al. (2002) found ICGV 86388, IC 34, and IC 10 resistant to thrips in Thailand. Thrips-resistant genotypes had lower PBND incidence in open field conditions during the seedling stage (Vijaya Lakshmi 1994).

The breeding strategy for resistance to PBND should aim at improving the levels of resistance to the vector and the virus and combining them into superior agronomic backgrounds. Interspecific breeding utilizing resistant compatible wild *Arachis* species should help to improve the levels of resistance to the vector and the virus. However, linkage drag could be an issue in interspecific breeding.

Resistant Cultivars Released. Resistant genotypes reduce the rate of epidemic development and considerably reduce the incidence of PBND (Buiel 1996). Several high-yielding cultivars with field resistance to PBND have been released. These include CO 3, ICGS 11, ICGS 44 (ICGV 87128), ICGS 37 (ICGV 87187), R 8808 (KRG 2), R 9251, K 134, DRG 12, RSHY 1, Kadiri 4, JCC 88, GG 7, and DRG 17, among others in India (Basu et al. 2002) and Khon Kaen 6 in Thailand (Sarawat et al. 2004). Other cultivars reported with field resistance to PBND in India, among others, are Kadiri 3, ICGS 5, RS 138, CSMG 881, CSMG 888, and CSMG 892 (Singh et al. 1994); ICGS 1 (Nigam et al. 1991a); ICGV 87141 (ICGS 76) (Nigam et al. 1991b); ICGV 87160 (Reddy et al. 1992); and ICGV 86699 (Reddy et al. 1996); ICGV 86325 (Dwivedi et al. 1996); TAG 24 (Patil et al. 1995); TG 37 A (Kale et al. 2004); GPBD 4, JSSP 9, and Dh 53 (Nagaraja et al. 2005); Pratap Mungphali 1 (Nagda and Joshi 2004); and Pratap Mungphali 2 (Nagda and Dashora 2005).

7. Nonconventional Genetic Improvement. Owing to complex ecology of PBNV and the vector, and difficulties in implementing integrated management approaches in subsistence agriculture system, PBND remains a difficult problem to manage under field conditions. Since the most successful approach that has aided so far in the development of virus-resistant plants has been the use of nucleocapsid gene encoding for viral coat protein (Satyanarayana et al. 1996a,b), ICRISAT is pursuing genetic engineering approaches to explore the possibilities of improving PBND resistance in peanut by expressing the PBNV nuleocapsid gene (Chander Rao et al. 2006). Over 48 independent transgenic events in JL 24 cultivar were produced by using two binary vectors encoding for *PBNVnp* gene through *A. tumefaciens* and microprojectile mediated genetic transformation. Based on greenhouse virus challenging experiments with T_1 and T_2 generation transgenic peanut plants, three events that showed a 40%–67% decrease in disease incidence were considered to be superior. However, only one event showed less than 25% disease incidence under field conditions in a contained on-station trial. The expression of symptoms in some plants was delayed by 40–60 days under greenhouse conditions and by 14–21 days in the contained on-station trial as compared to the control plants. Although, all the infected transgenic plants showed severe PBND symptoms, several of these showed recovery, thereby suggesting a modest tolerance to PBND (Chander Rao et al. 2006). Apparent lack of resistance to PBNV in the transgenic plants could be attributed to the presence of RNA silencing suppressor gene, nonstructural silencing suppressor gene (NSs), in the PBNV genome which could be rendering *PBNVnp* gene ineffective. Nevertheless, looking at the unexpectedly lower frequency of virus-resistant events throughout the challenging experiments, it was concluded that an alternate strategy based on RNA interference (antisense and hairpin-RNA) mediated gene silencing could be a potential tool to address a complex constraint such as PBNV. Currently, at ICRISAT, RNA*i*-mediated resistance approach to counter the effect of *NSs* gene in the PBNV genome is being pursued.

C. Tomato Spotted Wilt Virus Disease

Culbreath et al. (2003) have done an extensive review of epidemiology and management of tomato spotted wilt (TSW) in peanut in the United States.

1. Occurrence. Spotted wilt of peanut caused by TSWV was first reported in Brazil (Costa 1941). The virus also infects peanut in South America, but its impact is limited there. Since 1971, when it was first observed in Texas, it has become a major production constraint in peanut in the southeastern United States.

2. Symptoms. The wide array of symptoms caused by TSWV on peanut includes concentric ring spots on leaflets, various patterns of chlorosis on leaflets, stunting of all aboveground plant parts, small or misshapen geocarpophores, pods and kernels, and reddish discoloration and cracking of seed coats. Roots of affected plants typically show varying degree of necrosis that can result in death of the entire plant. Early infection causes reduction in pod number, kernel size, and yield in plants. TSWV has been associated with general yellowing and wilting of plants without accompanying its typical aboveground symptoms. Culbreath et al. (1992a) reported incidence of asymptomatic infections as high as that of disease incidence based on visible foliar symptoms. Most infections are the result of primary transmission and there is limited secondary spread of TSWV after it becomes established in the field (Black et al. 1993; Camann et al. 1995).

3. Virus Causal Agent and its Characteristics. TSWV is transmitted by thrips in a persistent manner but it is not seed or pollen borne (German et al. 1992; Peters 2003). It can be found in the pods and testae of seed from infected plants, but planting seed infested with TSWV does not result in plants infected with the virus (Pappu et al. 1999).

4. Vector and its Characteristics. Thrips are the only proven vectors of TSWV. Eight thrips species are reported as TSWV vectors—*Frankliniella fusca*, *F. intonsa*, *F. occidentalis*, *F. schultzei*, *S. dorsalis*, *Thrips tabaci*, *T. palmi*, and *T. setosus* (Todd et al. 1990; Mound 1996; Ullman et al. 1997).

Thrips can acquire the virus only during larval stages and larvae as well as adults transmit the virus (Peters et al. 1996). TSWV has been shown to multiply in their vectors (Ullman et al. 1993).

5. Alternate Hosts of Virus. TSWV and related viruses are reported to infect over 650 species of plants including 50 families among both monocots and dicots (Culbreath et al. 2003).

6. Conventional Genetic Improvement

Screening Methods/Techniques. Field screening, similar to that used for PBND, can be adopted for TSWV screening. Culbreath et al. (1997)

described a new intensity rating method based on percent of row length severely affected by TSWV, which takes much less time and effort than determining disease incidence based on individual plants. They recommended it as a practical alternative to individual plant assessment for characterization of genotype responses to TSWV. For stable resistance across locations, a multilocation field screening of genotypes is required due to potential strain variation in TSWV (Culbreath et al. 2000). Although earlier workers found mechanical sap inoculation of TSWV difficult, Mandal et al. (2001) developed a highly efficient mechanical transmission protocol to carry out glasshouse screening (temperature: minimum 21°C and maximum 45°C, relative humidity: 16%–80%, light intensity: 26–92 klx). They identified 2–3 days after germination (6–7 days after sowing) as the most susceptible stage of the seedling for TSWV inoculation. However, they cautioned of transmission variability among the isolates as their method was developed using a single isolate of TSWV.

Genetics of Virus and Vector Resistance. From their study on multiple foliar pest resistance in peanut at Khon Kaen, Thailand, Anderson et al. (1990) reported both significant general combining ability and specific combining ability for incidence of TSWV. Virginia parents, NC Ac 2821 and ICGS 4, had the best general combining ability effects for resistance to virus diseases. Holbrook et al. (2003) observed that transgressive segregation for resistance to TSWV was not uncommon, particularly in the heavy selection pressure that is practiced in peanut breeding nurseries in the southeastern United States. In *Capsicum chinense*, however, the resistance (localized hypersensitivelike reaction) to TSWV is governed by a dominant single gene (Boiteux et al. 1994).

Resistant Sources. Among 300 germplasm lines screened in field trials in Georgia, USA, 11 genotypes including 3 genotypes of *A. hypogaea* (PI # 196621, 339967, and 341267), 2 genotypes of *A. glabrata* (PI # 262794 and 338264), and 6 genotypes of other *Arachis* species (PI # 262286, 262828, 276233, 468142, 475883, and S-862) showed no infection 100 days after sowing whereas the susceptible control Florunner had 27% natural infection at harvest (Demski et al. 1991). Of the 24 wild *Arachis* accessions screened by mechanical inoculation in Georgia, Prasada Rao et al. (1993) found 7 accessions in section *Rhizomatosae*, 5 in section *Arachis*, and 1 in section *Erectoides* resistant to TSWV. Those, which belonged to section *Arachis*, included PI 468141 (*A. diogoi*), 468144 (*A. helodes*), 468345 (*Arachis* sp.), 468370 (*Arachis* sp.), and 468371 (*Arachis* sp.). The resistance of these accessions can be exploited in

interspecific breeding program. While pursuing interspecific breeding for combining resistance to root-knot nematode and TSWV in peanut, Holbrook et al. (2003) surmised that the resistance gene(s) to TSWV might be present in *A. cardenasii* (GKP 10017, PI 262141). Within *A. hypogaea*, the breeding line F NC94022-1-2-1-1-b3-B has among the highest levels of field resistance to TSWV (Culbreath et al. 2005). The source of resistance is hypothesized to be its *hirsute*-type parent PI 576638.

Resistant Cultivars Released. Resistant cultivars released in the United States include Southern Runner (Culbreath et al. 1992b), Georgia Brownie (Branch 1994), Georgia Green (Branch 1996), Tamrun 96 (Smith et al. 1998), Georgia Bold (Branch 1998), Georgia Hi-O/L (Branch 2000), Georgia-01R (Branch 2002), C-99R (Gorbet and Shokes 2002a), Florida MDR 98 (Culbreath et al. 1997; Gorbet and Shokes 2002b), Tamrun OL01 (Simpson et al. 2003), Georgia-02C (Branch 2003), Georgia-03L (Branch 2004), Georgia-04S (Branch 2005), Andru II (Gorbet 2006a), Carver (Gorbet 2006b), Tamrun OL07 (Baring et al. 2006), Georgia-O5E (Branch 2006), A-Norden (Gorbet 2007a), Hull (Gorbet 2007b), AP-3 (Gorbet 2007c), Tifrunner (Holbrook and Culbreath 2007), Georgia-06G (Branch 2007a), Georgia Greener (Branch 2007b), Georganic (Holbrook and Culbreath 2008), and Tifguard (Holbrook et al. 2008). Georganic (tested previously as C11-2-39) has the highest level of field tolerance among released cultivars (Culbreath et al. 2005). Along with cultural management practices, these genotypes help to keep the disease incidence at substantially low level. Cultivars with higher levels of resistance to TSWV with other desirable agronomic traits, if available, will reduce the dependence on other cultural and chemical control measures to manage the disease effectively.

7. Nonconventional Genetic Improvement. Several peanut breeding programs in the United States are making significant progress in improving resistance to TSW through conventional breeding methods (Culbreath et al. 1999, 2000). To further reduce yield losses caused by TSW, genetic engineering for generating TSWV-resistant peanut is receiving major attention in these breeding programs. Since, protection of transgenic plants against TSWV is under both RNA- and protein-mediated control (Pang et al. 1993), the approaches include using both sense and antisense TSWV nucleocapsid gene expression. Brar et al. (1994) and Chenault and Payton (2003) introduced nucleocapsid protein gene (*N* gene) from a Hawaiian TSWV lettuce isolate driven by a 35S CaMV promoter via microprojectile bombardment into a runner and a

Valencia-type varieties important to southeast and southwest United States, respectively. The *N* gene, when inserted into New Mexico Valencia A peanut, delayed symptom expression and prevented systemic virus infection (Li et al. 1997). Yang et al. (1998) suggested that stable engineered resistance would require production of numerous independent transformants to allow the selection of one with appropriate level of *N* gene expression. The field ratings for incidence of spotted wilt indicated that there was a potential to combine nucleoprotein-mediated resistance in transgenic peanut with host-plant resistance that already had been identified in the peanut germplasm. Variety AT 120 transgenics with antisense nucleocapsid gene (Magbanua et al. 2000) and selections of Marc 1 transformed with coat protein gene of TSWV (Ozias-Akins et al. 2002) showed lower disease incidence than the respective nontransformed cultivar or lower than that in moderately resistant cultivar Georgia Green. Yang et al. (2004) also observed lower incidence of spotted wilt in a transgenic progeny of Marc 1 peanut cultivar in comparison to the nontransgenic controls in field evaluations over years and locations and during challenge inoculation under controlled environmental conditions in the United States. They stated that this transgenic event could potentially be used in a conventional breeding program to enhance host resistance. As different mechanisms of resistance might be operating in different *N* gene containing lines, Bucher et al. (2003) suggested use of stable pathogen-derived resistance based on homology-dependent RNA silencing for durable TSWV resistance.

D. Peanut Stripe Virus Disease

1. Occurrence. Peanut stripe potyvirus disease (formerly reported as peanut mild mottle virus (PMMV) disease from Peoples' Republic of China) is widespread in east and southeast Asia. It was introduced into the United States through seed imports from Peoples' Republic of China and was first observed in Georgia in 1982 (Demski et al. 1984). It was also observed for the first time in 1987 in experimental materials at various locations in India (Prasada Rao et al. 1989). However, subsequent consistent efforts for a couple of years of destroying infected materials, quarantining the source locations, and monitoring peanut fields throughout the country led to elimination of this disease from India (Basu 1997).

2. Symptoms. Characteristic symptoms include dark green stripes and discontinuous banding along the lateral veins of young leaves

and an okra leaf or blotched pattern of dark green on older leaves. Infected plants show stunting, and unlike PMV and peanut green mosaic, symptoms persist in older leaves. Based on disease reaction on a set of peanut genotypes and some other host species, Wongkaew and Dollet (1990) grouped isolates of PStV, obtained from different countries, into eight strains—mild mottle, blotch, stripe, blotch-stripe, blotch-CP-N (similar to blotch but can induce systemic necrosis on KC 84 R cowpea), chlorotic ring mottle, chlorotic line-pattern, and necrotic strains. Similarity was noted among isolates within the same strain grouping regardless of their origins. Losses in pod yield vary with the strain type and can reach as high as 55% in Peoples' Republic of China (Kunrong et al. 1999). The PStV infection can decrease the nutritive value of peanut and change the flavor of peanut and its products (Ross et al. 1989). While Mn, Se, Zn, Fe, tartaric acid, raffinose, glucose, fructose, and total carbohydrate contents increased, the concentration of K, Mg, protein, and total soluble phenolics decreased in seeds from infected plants as compared to the seeds from uninoculated plants. Sucrose increased in seeds from plants inoculated with PStV at the time of emergence. There was no change in the concentration of stachyose, inositol, P, S, Ca, Cu, and oil.

3. Virus Causal Agent and its Characteristics. The disease is caused by potyvirus, PStV, and is transmitted by *A. craccivora* and *Myzus persicae* in a nonpersistent manner. Seed transmission ranging from 19.3 to 37.6% was observed in the United States in seedlings derived from peanut seed collected from plants infected with PStV when young (Demski et al. 1984; Sreenivasulu and Demski 1988). In majority of the cases in Peoples' Republic of China, the seed transmission rate ranged from 5 to 20% (Xu et al. 1991). Seed transmission rate depends on the virus strain, peanut genotype, plant age, and environmental conditions. Peanut plants grown at higher temperatures (25°C and 30°C) showed lower seed transmission (8% and 8.5%) than those grown at lower temperature (20°C; 18%) (Warwick and Demski 1992).

4. Alternate Hosts of Virus. Hosts of PStV include soybean (*G. max*), cowpea (*Vigna unguiculata*), cluster bean (*Cyamopsis tetragonoloba*), green gram, French bean (*P. vulgaris*), white lupine (*Lupinus albus*), wild tobacco (*N. benthamiana*), crimson clover (*T. incarnatum*), arrow leaf clover (*Trifolium vesiculosum*), subterranean clover (*Trifolium subterraneum*), sesame (*Sesamum indicum*), and Florida beggar weed (*Desmodium tortuosum*).

5. Conventional Genetic Improvement.

Screening Methods/Techniques. Epidemiological studies in the United States indicated localized secondary spread of PStV from a primary source; the major source of primary inoculum being the infected seed (Demski and Reddy 1988). To ensure high incidence of PStV in field screening, Kasno (1988) suggested early sowing of infector rows of a susceptible variety and sowing of infected seed obtained from infected plants at regular interval in infector rows. Wakman et al. (1989) transplanted PStV infected plants in infector rows and also released aphids onto infected plants. Planting of the screening nursery at a time when natural aphid activity is more (dry season) will ensure better spread of the virus in the field. Middleton et al. (1988) suggested the use of locations with high incidence of PStV and to take into account the following while scoring for PStV reaction: percentage disease incidence, types of symptoms observed, serological testing of all plants which failed to show disease symptoms and yield estimation, especially from PStV-infected genotypes which show either mild or no overt disease symptoms; the last being important to identify the sources of tolerance.

Genetics of Virus and Vector Resistance. From their study on multiple foliar pest resistance in peanut at Khon Kaen, Thailand, Anderson et al. (1990) reported both significant general combining ability and specific combining ability for incidence of PStV. Virginia parents, NC Ac 2821 and ICGS 4, had the best general combining ability effects for resistance to virus diseases. However, in soybean, where resistance to a soybean isolate of PStV is available in cultivated varieties, a single incompletely dominant gene was found to confer resistance. The dominant homozygous parent was immune and the heterozygous genotypes showed necrosis different from recessive homozygous parent. The gene was tentatively designated as Pst (Choi et al. 1989).

Resistant Sources and Resistance Breeding. High levels of resistance or immunity to PStV in cultivated peanut has not been reported. No resistance source was found among approximately 10,000 accessions of cultivated peanut from ICRISAT's gene bank when evaluated for resistance to PStV at Muneng in Indonesia. However, in some cases, symptoms were delayed until late in the growing season (Saleh et al. 1989). Similarly, among 1,383 cultivated peanut germplasm accessions screened in China, none showed resistance to PStV. However, among the wild *Arachis* species, *A. glabrata* (PI # 262801 and 262794) showed immune and *A. villosa* (PI 210555-1) and *A. correntina* (GKP 9530-31)

highly resistant reactions to PStV in field screening and on sap inoculation (Xu and Zhang 1987). Culver et al. (1987) also reported wild *Arachis* accessions of the section *Arachis*, *A. diogoi* (PI 468141 and PI 468142), *A. helodes* (PI 468144), and *Arachis* sp. (PI 468345) and of the section *Rhizomatosae* (PI # 468174, 468363, and 468366) resistant to PStV upon mechanical inoculation in the United States. In mechanical, aphid and graft inoculations of PStV of 54 wild *Arachis* in India, accessions in section *Arachis*, *A. cardenasii* [ICG 11558 (PI 475998)] could not be infected by all the three methods of inoculation, while *A. chacoense* [ICG 4983 (PI 276235)], *A. cardenasii* [ICG 11562 (PI 476012) and ICG 12168 (PI 476013)], and accessions of section *Erectoides*, *Arachis* sp. [ICG 11560 (PI 476004) and ICG 8215 (PI 468170)] and *A. paraguariensis* [ICG 8973 (PI 468176)] were infected by grafting but not by aphid and mechanical inoculations (Prasada Rao et al. 1991). Of the 24 wild *Arachis* accessions screened by mechanical inoculation in Georgia, Prasada Rao et al. (1993) found seven accessions in section *Rhizomatosae*, eight in section *Arachis*, and two in section *Erectoides* resistant to PStV. Those, which belonged to section *Arachis*, included PI # 276235 (*Arachis* sp.), 468141 (*A. diogoi*), 468144 (*A. helodes*), 468345 (*Arachis* sp.), 468370 (*Arachis* sp.), 468371 (*Arachis* sp.), 475998 (*Arachis* sp.), and 476012 (*Arachis* sp.). Some of these were also reported to be resistant to TSWV. The resistance of these accessions can be exploited in interspecific breeding program. Genetic variation was also observed in seed transmission rate among peanut genotypes. Cultivars, Haihua 1 and Hua 37, showed much lower seed transmission than cultivar Summer Peanut in Peoples' Republic of China (Xu et al. 1991). Warwick and Demski (1992) reported higher rate of seed transmission in the runner types (15%) than the bunch types (4%). On the contrary, Xu et al. (1991) reported more pronounced disease symptoms and higher seed transmission rates in Spanish types than other types.

No targeted breeding against PStV has been reported. In east and southeast Asia, where the disease is well established and widespread, interspecific breeding utilizing immune/resistant genotypes of *Arachis* species from section *Arachis* should be initiated. The species in section *Arachis* are cross compatible with cultivated peanut *A. hypogaea*. Some of these genotypes, PI # 468141, 468142, 468174, 468363, and 468366, are also reported to be resistant to PMV (Melouk et al. 1984). Thus, combined resistance to both PStV and PMV can be obtained in a resistance breeding program. Resistance to aphid vectors should also be exploited along with the resistance to the viruses. Adala and Natural (1988) observed no PStV infection in aphid-resistant genotype ICG 5240 (EC 36892), when it was exposed to viruliferous aphids in the

Philippines. In Peoples' Republic of China also, this genotype recorded low incidence of PStV (Zeyong et al. 1995). As genetic variation in seed transmission rate exists, this gives an opportunity to breed for zero or low seed transmission to reduce primary source of virus inoculum in the produce if used as seed for the next crop.

Resistant Cultivars Released. Although not specifically bred for PStV resistance, improved peanut cultivars, Huayu 16 (Zhengchao and Qingshu 2000) and Huayu 17 (Shanlin et al. 2000), developed at Shandong Peanut Research Institute, Laixi, Peoples' Republic of China, are reported to be moderately resistant to PStV.

6. Nonconventional Genetic Improvement. Transgenic plants of peanut varieties, Gajah and NC 7, containing one of the two forms of PStV coat protein gene (an untranslatable, full length sequence (*cp* 2), or a translatable gene encoding a *cp* with an N-terminal truncation (*cp* 4)) exhibited high levels of resistance to PStV. The mechanism of resistance appears to be RNA-mediated, since plants carrying either *cp* 2 or *cp* 4 gene had no detectable protein expression but were resistant or immune (no virus replication). Highly resistant *cp* 2 T_0 plants contained transgene-specific small RNAs (Higgins et al. 2004). PStV resistance in these transgenic plants of Gajah variety was stably inherited over at least five generations (Dietzgen et al. 2004). In another study, where three different kinds of response to PStV infection were identified—resistant, recovery, and susceptible, the transgenic peanut lines cv. Gajah proved stable up to seven generations of selfing and some pure lines were identified (Hapsoro et al. 2005, 2007). These lines constitute important germplasm for PStV resistance breeding in peanut, which will provide protection against a wide range of isolates present in different geographic regions where PStV is endemic.

E. Peanut Stem Necrosis Disease

1. Occurrence. Peanut stem necrosis disease caused by TSV came to notice in India in 2000, where it caused an epidemic in Anantapur district in Andhra Pradesh affecting 2,25,000 ha and causing an economic loss of US$ 65 million (Reddy et al. 2002). TSV was reported as occurring frequently on peanut in Brazil (Costa and Carvalho 1961), but it was first noticed on peanut in 1999 in South Africa (Cook et al. 1999) and in 2000 in Anantapur in India. TSV on peanut in India is currently observed in parts of Andhra Pradesh (Anantapur, Kurnool,

Cuddapah, and Chittoor districts) and adjoining areas in Karnataka (Raichur district). It remains a potential threat to peanut in southern states in India. Lava Kumar et al. (2008) have recently discussed emergence and spread and control strategies of TSV in India.

2. Symptoms. Symptoms first appear on young leaves as necrotic lesions and veinal necrosis. The necrosis later spreads to the petiole and stem. Necrotic lesions on the stem later spread upwards killing the bud. Majority of the plants infected within a month after sowing die due to necrosis, which also spreads downwards in case of early infection. Because of the necrosis of stem, the disease was named peanut stem necrosis disease (PSND). In some cultivars, surviving plants produce axillary shoots. The leaflets on these axillary shoots are small and show general chlorosis unlike the secondary symptoms of PBNV where distortion and mosaic mottling of leaf lamina are common. In some cultivars pods harvested from the diseased plants show necrotic lesions.

3. Causal Agent and its Vector and Their Characteristics. PSND is caused by TSV (Reddy et al. 2002). The adults of three thrips species, *F. schultzei*, *S. dorsalis*, and *Megalurothrips usitatus*, have been shown to transmit TSV experimentally in peanut and cowpea in the presence of infected pollen from the susceptible hosts (Shukla et al. 2005). In India, the flower inhabiting *F. schultzei* and *P. hysterophorus*, a symptomless weed host, play a major role in the field spread of the disease in peanut (Prasada Rao et al. 2003b). The pollen-assisted TSV transmission process has been studied in detail in other host plants (Sdoodee and Teakle 1987; Greber et al. 1991). Transmission of TSV occurs when thrips carrying pollen from TSV-infected plants on their bodies land on host plants and cause them (pollen) to dislodge on leaves, and while feeding on host plants wound both leaf tissue and infected pollen to facilitate virus infection of the plants. As thrips do not acquire the virus, there is no leaf-to-leaf transmission. Limited studies conducted with field-infected and mechanically inoculated plants of peanut, sunflower, and parthenium failed to show seed transmission of the virus (Prasada Rao et al. 2009). Further studies using large quantities of seed of more than one cultivar are needed to reach a firm conclusion on seed transmission of the virus in these crops.

4. Alternate Hosts of the Virus. Natural hosts of the virus include peanut, safflower (*Carthamus tinctorius*), sunflower, cotton (*Gossypium hirsutum*), cowpea, black gram, mungbean, marigold, chilli, bottle gourd (*Lagenaria siceraria*), cucumber (*Cucumis sativus*), gherkin

(*C. sativus*), niger (*Guizotia abyssinica*), okra (*Abelmoschus esculentum*), pumpkin (*Cucurbita pepo*), sunnhemp (*Crotalaria juncea*), sesame, soybean, several weed species, and ornamentals. Among the weed species, parthenium, is widespread in India (Prasada Rao et al. 2003a; Jain et al. 2005; Santha-Lakshmi-Prasad et al. 2005; Kumar et al. 2006; Ladhalakshmi et al. 2006).

5. Conventional Genetic Improvement

Screening Methods/Techniques (Field and Laboratory). An effective field screening technique for PSND resistance is yet to be perfected. A screening method, where parthenium was grown 1 month before sowing the test genotypes around the field in which PSND screening would be carried out, gave encouraging results. An artificial inoculation method involving infected sap dilution at 1:10 and inoculation twice at 12 and 15 days after sowing was found to be very good in screening peanut germplasm and to identify stable resistance (Prasada Rao, pers. commun.). Kalyani et al. (2005) used both 1:10 and 1:100 sap dilution for screening peanut germplasm and observed that lower virus concentration provided a more discernible disease picture among the genotypes. They also found virus not producing symptoms at lower temperatures during winter season. Screening for TSV/PSND resistance should be carried out when temperature conditions are favorable (28–32°C) for virus multiplication and symptom expression.

Resistant Sources and Resistance Breeding. All 150 released peanut cultivars in India showed susceptible reaction to TSV in glasshouse screening using mechanical inoculation with sap from virus-infected plants at 1:10 concentration (Prasada Rao et al. 2003b). Among 11 PBND and thrips-tolerant peanut genotypes identified at ICRISAT and screened for TSV resistance using 1:100 and 1:10 virus concentrations, ICGV # 99029, 01276, 92267, and 00068 recorded significantly lower TSV infection than susceptible control JL 24 (Kalyani et al. 2005). Among 56 germplasm accessions from 20 wild *Arachis* species belonging to *Arachis*, *Erectoides*, *Procumbente*, and *Rhizomatosae* sections, eight accessions, ICG # 8139, 8195, 8200, 8203, 8205, and 11550 belonging to *A. duranensis*, ICG 8144 belonging to *A. villosa*, and ICG 13210 belonging to *A. stenosperma* (all from section *Arachis*) did not show systemic spread of virus upon mechanical inoculation in repeated tests. These accessions either showed no infection or 100% infection in inoculated leaves but subsequently emerged leaves did not show any presence of the virus (Kalyani et al. 2007). No further screening for resistant sources and resistance breeding are currently in progress for this disease in peanut in India.

Since the disease remains a potential threat in southern states in India, interspecific breeding utilizing accessions belonging to section *Arachis* should be initiated to develop TSV-resistant varieties. As high levels of resistance are available for thrips vectors in cultivated germplasm, these accessions should also be involved in hybridization. A variety with combined resistance to TSV and thrips vector would withstand the disease onslaught better.

6. Nonconventional Genetic Improvement. At ICRISAT, work is being carried out on engineering TSV resistance through *A. tumefaciens*-mediated transformation of popular peanut cultivar JL 24 (Spanish type) with TSV coat protein gene (*TSVcp* gene), and recovery of transgenic plants that block systemic movement of TSV within the plant (Sharma et al. unpublished). Across various plants batches inoculated in T_1 generation, three different types of resistance responses were observed. While several plants were found to be completely susceptible, a few plants maintained a disease symptom-free phenotype and others recovered from an initial infection and the subsequent new growth was devoid of TSV symptoms. All the symptomatic plants, either transgenic or control, tested positive to TSV in ELISA, and all asymptomatic plants were negative, indicating a correlation between the virus presence and the stem necrosis disease. Primarily, a few highly resistant plants were obtained where no symptoms developed even as late as 21 days postinoculation, although >10% transgenic plants exhibited delayed symptom development. Considering that TSV was detected in the inoculated leaves of these plants and the lack of virus in the subsequently emerged leaves suggests inhibition of the systemic spread of virus. These results clearly indicate that the use of transgenic technology appears appropriate for the development of virus-resistant peanut to combat stem necrosis disease. However, to ascertain the usefulness of this newly acquired resistance, the resistant transgenic events identified under greenhouse conditions must be evaluated under restricted field conditions in the TSV hot spots in the near future. In another study, Bag et al. (2007) generated transgenic peanut lines containing sense and antisense coat protein gene of TSV using *Agrobacterium*-mediated transformation of de-embryonated cotyledons of cultivar JL 24. These lines are under evaluation for their reaction to TSV.

F. Peanut Clump Disease

1. Occurrence. The peanut clump virus disease is widespread in West Africa. It is also found in Indian subcontinent in isolated patches particularly in sandy and sandy loam soils.

2. Symptoms. Diseased plants are severely stunted, dark green and bushy with young quadrifoliate leaves showing mosaic mottling with chlorotic rings. They occur in patches in the field. Infected plants produce flowers but a few poorly developed pods. The disease recurs in the same patches year after year.

3. Causal Agent and its Vector and Their Characteristics. The disease is soilborne. It is caused by a PCV and is transmitted by soil inhabiting fungus *P. graminis*. The fungus lives in roots of cereal crops and grassy weeds. The optimum temperature for infection by *P. graminis* is between 27 and 30°C. Below 23°C, infection is suppressed and fungal development is delayed (Legreve et al. 1998). The virus replicates well over the same range of temperature as the fungus, but temperatures higher than 30°C are not favorable to the virus (Delfosse et al. 2002). The virus is transmitted through peanut seed (6%–50%) and also through seeds of finger millet (*Eleusine coracana*), fox tail millet (*Setaria italica*), pearl millet, maize, and wheat (*Triticum aestivum*). West African PCV isolates are not serologically related to Indian peanut clump virus (IPCV) isolates (Reddy et al. 1983).

4. Alternate Hosts of Virus. The virus causes disease in wheat, barley, sugarcane (*Saccharum officinarum*), chilli, and pigeonpea (*Cajanus cajan*). Other symptomless (or absence of severe symptoms) hosts include sorghum, maize, rice (*Oryza sativa*), mustard (*Brassica juncea*), soybean, and mungbean.

5. Conventional Genetic Improvement

Screening Methods/Techniques (Field and Laboratory). In the past, hot spot locations have been used for screening germplasm for resistance to peanut clump disease. Using French bean as inoculum source, Reddy et al. (2005) showed efficient transmission of one isolate by mechanical sap inoculation with the virus having a 3 week incubation period. They recommended it as a convenient and reliable screening method without going to hot spot locations.

Resistant Sources and Resistance Breeding. West African peanut variety 57-422 is reported as tolerant to peanut clump (Bockelee-Morvan 1983). Among 9,000 germplasm accessions of cultivated peanut tested in India, no resistance source to PCV/IPCV could be identified. No further screening for resistant sources and resistance breeding are currently in progress for this disease either in India or West Africa.

6. Nonconventional Genetic Improvement. The coat protein gene from RNA-2 of IPCV has been cloned and sequenced (Wesley et al. 1994) and its expression in *N. benthamiana* studied (Bragard et al. 2000). At ICRISAT, studies have been carried out to induce resistance to IPCV in peanut using the *cp* and replicase genes of IPCV in *Agrobacterium*-mediated transformation (Sharma and Anjaiah 2000). Over 50 transgenic events were developed using either *cp* or replicase (*rep*) gene of IPCV, and characterized for gene integration and expression. Replicated field evaluations against IPCV were carried out under controlled conditions during the rainy season of 2002–2004 in an on-station sick plot at ICRISAT, Patancheru, India under contained conditions by using 10 transgenic lines carrying single gene inserts (5 each with *IPCVcp* and *IPCVrep* genes). Four transgenic events (three with *IPCVcp* and one with *IPCVrep* gene) showed complete resistance to IPCV (Sharma et al. unpublished). These plants showed resistant phenotype where the virus titer declined with maturity. In the first ELISA test conducted 3 weeks after sowing, all the tested plants showed the presence of IPCV, thereby, showing an initial infection of the test plants, while the subsequent three tests at 15 day intervals did not reveal any virus in these four lines. This indicated the potential of *cp* and *rep* genes in the induction of resistance to IPCV.

G. Peanut Mottle Virus Disease

1. Occurrence. Peanut mottle virus in peanut was first observed in 1961 in Georgia, USA (Kuhn 1965). It is now widely distributed in cultivated peanut throughout the world (Behncken 1970; Demski et al. 1975; Bock et al. 1978; Reddy et al. 1978). It also infects soybean (Demski 1975), lupines (Demski et al. 1983b), cowpea (Demski et al. 1983a), snap bean (*P. vulgaris*) (Silbernagel and Mills 1991), garden pea (*P. sativum*), and some forage legumes (Demski et al. 1981) under natural conditions when planted close to peanut field. The predominant strain of PMV in the United States (PMV-M2) caused a 31% seed loss in a susceptible peanut cultivar Starr (Kuhn et al. 1978). The yield loss caused to peanut by PMV in Georgia in 1974 was estimated to be over US$ 10 million (Paguio and Kuhn 1974). PMV also alters the chemistry (fatty acids, amino acids, and total protein) of peanut seed (Hovis et al. 1979).

2. Symptoms. PMV causes mottling and interveinal depression in peanut leaves with their margins rolled upwards causing a cupping appearance. Occasionally, dark green islands on leaves are also visible. As plants mature, these symptoms become masked, particularly during

hot, dry weather, leading to many infected plants showing no overt symptoms except on new flushes of growth. Both number and size of pods from infected plants are decreased.

3. Causal Agent and its Vector and Their Characteristics. PMV is transmitted in a nonpersistent manner by several aphid species (*A. craccivora* Koch, *A. gossypii* Glover, *Hyperomyzus lactuae* L., *M. persicae* Sulzer, *Rhopalosiphum padi*, and *R. maidis*) and infected peanut seeds. Five strains of PMV have been isolated from peanut in Georgia, USA. These are M 1 and M 2, which induce mild mottle and N, S, and CLP, which cause necrosis, severe mosaic, and chlorotic line pattern symptoms, respectively (Paguio and Kuhn 1973). The necrosis strain is not aphid transmitted except in mixed infection with the mild mottle strain. Transmission through infected seed appears to be the most important source of PMV spread in peanut and a few other crops (mungbean and cowpea). Transmission in peanut seed varies from none to 8.5% depending upon genotype, virus strain and environment.

4. Alternate Hosts of Virus. In addition to *P. vulgaris*, 14 other species are reported to be susceptible to the virus (Kuhn 1965). Mottle symptoms are produced in *Vigna sinensis* (L.), *L. albus* L., *L. angustifolius* L., *Trifolium vesiculosum* Savi (several cultivars), *T. subterranium* L., *Phaseolus lunatus* L. cv. Henderson, *G. max* (L.) Merr. cv. CNS-4, *V. unguiculata* L., *Canavalia ensiformis*, *Cassia laptocarpa* Benth., *C. occidentalis* L., *P. sativum* cvs. Alaska and Little Marvel, and *T. incarnatum* L. *C. tora* produces black local lesions and stem necrosis. Virus was recovered from inoculated but symptomless plants of *V. cylindrical* Skeels, *V. sesquipedalis* Fruwirth, *Phaseolus coccineus* L., *Cymopsis tetragonoloba* (L.) Taub., and *Lathyrus odoratus* L.

5. Conventional Genetic Improvement

Screening Methods/Techniques. Most of the screening for resistance to PMV has been done under greenhouse conditions following mechanical sap inoculation and aphid transmission. Disease reaction was determined by symptoms, local-lesion assays, serology, and electron microscopy. However, a field-screening nursery can be developed for PMV following the approach used in the field screening for resistance to peanut stripe virus.

Genetics of Virus and Vector Resistance. No inheritance study on PMV resistance in peanut is reported in the literature. However, in soybean, the resistance to PMV is reported to be governed by single dominant allele (Boerma and Kuhn 1976).

Resistant Sources and Resistance Breeding. No immunity to PMV is reported in cultivated peanut (Kuhn et al. 1968). However, Kuhn et al. (1978) reported tolerance in two genotypes of cultivated peanut, PI # 261945 and 261946, as they did not suffer yield loss upon inoculation with the virus. Demski and Sowell (1981) reported resistance to PMV in seven rhizomatous introductions (PI 262794, PI 262818, AM 3867, 'Florigraze' (PI 421707), PI 172223, 'Arbrook' (PI 262817), and 'Arblick' (PI 262839)) as they were not infected with the virus by mechanical inoculation, aphid inoculation, or by natural infection when planted near infected peanut field. Melouk et al. (1984) found eight wild *Arachis* species accessions (PI 468171, 468174, 468363, 468366, and 468371; all belonging to section *Rhizomatosae* and PI 468141 (*A. diogoi* Hoehne), 468142 (*A. diogoi*), and 468169; all belonging to section *Arachis*) free from virus. Of the 24 wild *Arachis* accessions screened by mechanical inoculation in Georgia, Prasada Rao et al. (1993) found eight accessions each in section *Rhizomatosae* and section *Arachis* and one in section *Erectoides* resistant to PMV. Those belonging to section *Arachis* included PI 276235 (*Arachis* sp.), 468141 (*A. diogoi*), 468144 (*A. helodes*), 468150 (*Arachis* sp.), 468345 (*Arachis* sp.), 468370 (*Arachis* sp.), 468371 (*Arachis* sp.), and 475998 (*Arachis* sp.). Some of these are also reported to be resistant to TSWV and PStV. As genotypes belonging to section *Arachis* are cross compatible with cultivated peanut, they could represent good sources of multiple resistances to viruses (TSWV, PStV, and PMV) in an interspecific breeding program.

Out of 283 germplasm lines screened using ELISA on individual seed by Bharathan et al. (1984), two lines, EC 76446 (292) and NC Ac 17133 (RF) showed no seed transmission of PMV. As PMV incidence can be reduced or eliminated by using virus free seed (accompanied with isolation with other peanut field), nonseed transmission can be an important trait in PMV resistance breeding program.

In spite of availability of sources of resistance in section *Arachis* and tolerance in cultivated peanut, no targeted breeding for resistance to PMV is being pursued.

6. Nonconventional Genetic Improvement. Currently, no reports are available on the development of nonconventional resistance to PMV.

H. Peanut Stunt Virus Disease

1. Occurrence. The stunt disease of peanut caused by PSV in the United States was first observed in Virginia and North Carolina in 1964. The virus is widespread in white clover and other forage legumes in many countries. It causes a severe disease in beans. It can also cause disease in tobacco, soybean, and snap bean.

2. Symptoms. The virus has several distinct strains. The strain in North America causes severe dwarfing of either the entire peanut plant or one or more of its branches, depending on the age of the plant. A different isolate of the virus in China does not cause severe stunting, but the leaflets on infected plants are malformed, with various chlorotic symptoms and upward curling. Diseased plants produce fewer pods with reduced size. Misshapen small pods sometimes have a split pericarp wall.

3. Causal Agent and its Vector and Their Characteristics. PSV, a member of cucumovirus group, is transmitted by three species of aphids, *M. persicae*, *A. craccivora*, and *A. spiraecola*, in a nonpersistent manner. It is also easily transmissible by sap inoculation. The virus is seed transmitted but infected seeds, unlike PMV, are not considered the most important source of virus spread as graded seeds, which are used in sowing, have very little seed transmission. Two serologically distinct isolates of the virus from United States (Tolin 1984) and three from China (Zeyong et al. 1986) are reported.

4. Alternate Hosts of Virus. PSV has an extremely wide host range. It may infect 115 species in 59 genera of 17 families, including 63 species in 20 genera of the Leguminosae (Tolin 1984).

5. Genetic Improvement. No genetic enhancement is being pursued to develop varieties resistant to PSV. To contain the virus, use of virus-free seed is suggested. The seed production fields should be away from known reservoir of PSV such as white clover (*T. repens*). Infected plants in a seed production fields should be rogued out.

I. Cowpea Mild Mottle Virus Disease

1. Occurrence. Cowpea mild mottle virus has been reported on cowpea and soybean from East and West Africa and Thailand. Its natural occurrence on peanut has been reported from India (Iizuka et al. 1984) and Sudan (El-Hasan et al. 1997). In both countries, the disease was pronounced in irrigated crop. Incidence of CMMV can be as high as 30% in peanut if they are sown adjacent to infected soybean or cowpea fields.

2. Symptoms. Infected plants are conspicuous with outward rolling of their leaflet edges and severe stunting. Younger leaflets often show vein banding and older leaflets and petioles may become necrotic and drop off. Stunted plants rarely produce pods.

3. Causal Agent and its Vector and Their Characteristics. CMMV is transmitted by whitefly (*B. tabaci*) in a nonpersistent manner. It is also easily sap transmissible. Although it is seed transmitted in cowpea and soybean, seed transmission has not been reported in peanut.

4. Alternate Hosts of Virus. CMMV induces systemic symptoms on *C. ensiformis*, *Cassia occidentalis*, *G. max*, *N. clevelandii*, *P. vulgaris*, *P. sativum*, and *V. unguiculata* (Reddy and Rajeshwari 1984).

5. Genetic Improvement. No information is available on sources of resistance and crop improvement efforts for resistance to CMMV in peanut. The disease can be avoided by not sowing peanut adjacent to infected soybean or cowpea fields and not intercropping it with crops that are susceptible to whitefly.

J. Cucumber Mosaic Virus Disease

1. Occurrence. Cucumber mosaic virus disease is recognized as economically important in northern parts of China and Argentina (Zeyong and Barnett 1984; Zeyong et al. 1989; de Breuil et al. 2008). It can cause up to 40% yield loss.

2. Symptoms. Initial symptoms appear as chlorotic spots on leaves, which later become yellow. Adjacent spots coalesce forming large yellow blotches. Some older leaflets also show green stripes along the veins. Plants are stunted.

3. Causal Agent and its Vector and Their Characteristics. The disease is caused by cucumber mosaic cucumovirus and transmitted by *Macrosiphum euphorbiae* in nonpersistent manner and mechanical sap inoculation. It is seed transmitted up to 2% in peanut.

4. Alternate Hosts of Virus. CMV has a wide host range.

5. Genetic Improvement. No information is available on sources of resistance to CMV in peanut. Planting virus-free seed and use of polythene mulch in cultivation are reported to reduce disease incidence in peanut.

IV. THE FUTURE

Of the 31 viruses from 14 genera, reported to naturally infect peanut in different parts of the world, only a few of these are of economic importance. Effective control strategies for virus diseases are best based on the knowledge of the causal viruses and their principal vectors and the availability of genetic resistance sourced from within the crop species and its wild relatives or from virus genomes. All the currently recognized economically important viruses occurring in the major peanut growing countries have now been characterized and their diagnostic aids developed.

Host plant resistance provides the most effective and economic option to manage virus diseases. By harnessing resistance genes from cultivated peanut, cultivars resistant/tolerant to GRD, PBNV, and TSWV have been developed. However, for many other viruses, effective resistance gene(s) in the cultivated peanut have not been identified. Many wild *Arachis* species harbor genes with high levels of resistance against several virus diseases in peanut. Interspecific breeding, although long drawn, offers opportunities to develop cultivars with high levels of resistance to more than one virus disease. The breeding strategy for resistance to virus diseases should aim at improving the levels of resistance to the virus and the vector and combining them into superior agronomic backgrounds. Where a high level of genetic resistance is not available, a holistic approach combining genetic resistance, cultural management and chemical control may be required for effective management of the virus diseases.

The sequencing of genomes of viruses such as PMV, PStV, PCV, IPCV, and TSV permits utilization of genes of these viruses in the production of resistant transgenic peanut plants. However, even transgenics do not ensure complete freedom from viruses. Genetic engineering using nucleocapsid gene (*np*) for generating PBNV- and TSWV-resistant peanut is being attempted at ICRISAT, but RNA silencing suppressor gene, *NSs*, in the genomes of these viruses is rendering *np* gene ineffective. An alternative strategy based on RNA interference

(antisense and hairpin-RNA) mediated gene silencing could be a potential tool to address a complex constraint such as PBNV and TSWV.

GRD-resistant cultivars released in Africa succumb to the disease under very heavy disease pressure. All these varieties are resistant to GRV and its *sat*RNA but not to GRAV, which also causes significant yield loss. It is, therefore, essential to identify resistance to GRAV and incorporate it in new cultivars along with that of GRV and *sat*RNA with desired farmer-preferred agronomic traits. Incorporation of resistance to aphids into GRD-resistant cultivars will not only provide additional defense against the disease complex but will also minimize the direct yield loss caused by aphids. Resistance to PBNV and TSWV, identified in wild *Arachis* species, can improve the levels of currently available genetic resistance to these viruses in peanut cultivars. For peanut clump disease, the resistance available in wild *Arachis* could be exploited. Viral polymerase or those genes, which can offer broad based resistance to this virus, are most suitable for incorporating transgenic resistance. For PStV and PMV, which are seed transmitted and can, therefore, move to new locations, several wild *Arachis* species showed immune or highly resistant reaction, which need to be exploited to develop resistant cultivars. Transgenic plants of peanut varieties, Gajah and NC 7 containing one of the two forms of PStV coat protein gene (*cp 2* and *cp 4*), which exhibited high levels of resistance to PStV, can be used in a regular breeding program to develop resistant cultivars. The limited economic impact observed for the peanut stunt, cowpea mild mottle virus, and cucumber mosaic virus do not warranted concerted research efforts toward conventional and nonconventional genetic improvement.

With a few exceptions (such as GRD in Africa, PBND in India, and TSWV in the United States), breeding for virus resistance has received little attention in peanut due to the limited availability of virologists and suitable infrastructure in national programs in most of the developing countries. If the losses caused by virus diseases in developing countries are to be contained, capacity building and infrastructural development are essential to develop resistant cultivars.

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Common Bean Breeding in the Tropics

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ABSTRACT

Common bean (*Phaseolus vulgaris* L.) breeding in the tropics and subtropics of Latin America, the Caribbean and Africa has undergone significant changes in the past two decades. Molecular markers for important disease and pest resistance traits have been created and deployed, and resistant cultivars have been introduced, especially for viral and fungal diseases. Scientific capacity has expanded, especially in Africa, and market forces now set many of the criteria for new introductions. However, yields have not increased dramatically in most cases, except where crop management and modern agricultural techniques have been deployed. The vast majority of bean producers in the tropics continue to cultivate the crop in low input systems, and soil degradation makes edaphic constraints more acute. Effects of climate change are already felt in some regions with more frequent droughts or excessive rainfall that will alter patterns and intensity of bean diseases. An evolving climatic scenario and the need to increase yields significantly demand more attention to abiotic stresses of drought, low soil fertility, and eventually higher temperatures. The wild ancestor of common bean evolved in a relatively favorable environment with few abiotic stresses, but domestication has broadened the adaptation range of cultivated bean. Traits for greater efficiency in nutrient acquisition and use have been identified, and sources of tolerance to aluminum toxicity have been employed in breeding. The *Phaseolus* genus is adapted to environments ranging from arid deserts to tropical rain forests, and species with which common bean is cross compatible cover most of this range. Interspecific crosses can tap this genetic variability for adaptation to extreme environments. Nutritional quality

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