CHAPTER 26

Groundnut bud necrosis virus (GBNV) and the management of peanut bud necrosis disease (PBND)

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Introduction

Groundnut (peanut) crop has been reported to be infected by 23 viruses under natural conditions and another 39 viruses are capable of infecting groundnut under experimental conditions (Sreenivasulu et al., 1991). Among those, the Groundnut Bud Necrosis Virus (GBNV) causes significant economic loss resulting from the necrosis of terminal bud and subsequent death of the plant. The disease was named peanut bud necrosis disease (PBND) by Reddy et al. (1968), and based on virion morphology, host range, vector transmission, antigenic relationship, and similarity of the virus causing PBND to tomato spotted wilt virus (TSWV), it was concluded that TSWV is also the causal organism of PBND (Chohan, 1972, 1974; Ghanekar et al., 1979). However, further serological cross-reaction studies (Adam et al., 1993), and sequence similarity of amino acids and the homology of the nucleoprotein (De Avila et al., 1993), led to the conclusion that the causal virus of PBND, specifically reported in India, was different from TSWV and was named as groundnut bud necrosis virus (GBNV). Though GBNV is primarily known to affect groundnut, it has now been reported to cause different types of necrosis disease in several crop species including horticultural crops and ornamental plants and weeds.

Peanut bud necrosis disease occurring in the early stages of the crop can cause up to 100% yield loss, while at a later stage of crop growth, it may cause up to 70% (Prasada Rao et al., 1989; Narayanasamy and Ramiah, 1977). The incidence of the disease generally can range from 5% to 80% as observed in the different groundnut-growing regions of India.

In the PBNV-affected regions of the world such as the South/South-eastern Asia, South America, and the Southern United States, the virus is reported to be transmitted by different species of thrips, like *Thrips palmi*, *Scirtothrips dorsalis*, and *Frankliniella schultzei* (Pappu et al., 2009; Riley et al., 2011; Mandal et al., 2012). The incidence of bud necrosis

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is largely dependent on migrant thrips infesting the groundnut crop (Reddy et al., 1983; Reddy and Wightman, 1988).

Total resistance, i.e., absence of systemic infections at the genotype level, has not been reported in the cultivated groundnut, *Arachis hypogaea* (Reddy et al., 1991). However, a good number of genotypes with field resistance to PBNV (Amin, 1985; Dwivedi et al., 1993, 1995; Reddy et al., 2000; Kesmala et al., 2004; Mandal et al., 2012) have been identified. There are crop management practices that can take care of reducing the vector population which are generally advocated control measures to PBND since durable host plant resistance is not available.

In this chapter, the progress and prospects toward managing the virus have been consolidated.

Peanut bud necrosis disease

Peanut bud necrosis disease (PBND) caused by groundnut bud necrosis virus (GBNV) is an economically important Tospovirus infecting several crops, horticultural, and ornamental species. In the US, the annual yield losses due to GBNV have been estimated to be over U.S.$89 million (Reddy et al., 1995). The yield losses due to the PBND in India have been reported up to 90% (Chohan, 1974; Ghanekar et al., 1979; Mayee, 1987; Gopal and Upadhyaya, 1988; Singh and Gupta, 1989; Dwivedi et al., 1993; Basu, 1995; Singh and Srivastava, 1995; Dharmaraj et al., 1995). The extent of loss may depend upon the time of incidence of the disease and the yield loss will be 100% up to 60-day-old crop, and infection after pod formation may lead to a minimal loss only.

The initial symptoms of PBND appear on young leaves with light chlorotic spots progressing to chlorotic or necrotic rings or streaks or green islands with chlorosis on leaves. This will lead finally to the wilting and necrosis of terminal buds. If the disease occurs within 30 days after germination of the crop, there will be a total loss of the crop due to terminal bud necrosis. Stunting of the plant with distorted leaf lamina, chlorosis of the leaves, axillary shoot proliferation, and mottling appear as secondary symptoms. In most cases, the secondary symptoms are apparent only in the terminal buds or only a few branches will be affected (Fig. 1). The pods of the diseased plants turn out to be small with shriveled seeds and mottled testa, and with reduced oil content (Mohamed Ali and Prasada Rao, 1982).

Distribution

In India, PBND has been considered as a minor disease of groundnut (Reddy, 1988) and later on the disease was reported in epidemic proportions (Vijayalakshmi, 1994). PBND has been reported as one of the major production constraints in groundnut from the states of Andhra Pradesh, Maharashtra, Uttar Pradesh, Gujarat, Tamil Nadu, Karnataka, and Madhya Pradesh, where the crop is grown during the rainy seasons. PBND has been

Fig. 1 (A) Axillary shoot proliferation and chlorosis of leaf lamina without major damages to terminal bud, due to infection at the final stage of crop growth; (B) terminal bud affected due to infection at a later stage of crop growth; (C) total necrosis of the terminal bud due to the infection at an early stage of crop growth and may lead to the death of the plant.

reported in post rainy crops also from the Saurashtra region of Gujarat, Northern, and Vidarbha regions of Maharashtra; Nizamabad, Nalgonda, and Mahbubnagar districts of Telangana; and northeastern parts of Karnataka (Radhakrishnan et al., 2016). Raichur in Karnataka, Jagtial in Telangana, Hyderabad in Andhra Pradesh, Latur in Maharashtra, Saurashtra in Gujarat, Tikamgarh in Madhya Pradesh, and Mainpuri in Uttar Pradesh are considered as hot spots for PBND in India.

Groundnut bud necrosis virus (GBNV)

Tomato spotted wilt virus (TSWV) was the lone virus under genus tospovirus until 1992 when groundnut bud necrosis virus (GBNV) was described as distinct from the TSWV based on serology (Reddy et al., 1992). Groundnut bud necrosis virus (syn. Peanut bud necrosis virus) is a tospovirus, (RNA virus) under the order *Bunyavirales*, which has more than 350 named isolates (Pappu et al., 2009; Elliott, 2014; Oliver and Whitfield, 2016; Adams et al., 2017). The family *Bunyaviridae* comprises five genera, viz., *Orthobunyavirus, Hantavirus, Nairovirus, Phlebovirus*, and *Tospovirus* (Nichol et al., 2005). Of these, *Tospovirus* is the only genus having plant viruses and has four serogroups according to their serological relationships and sequence homology of the nucleocapsid protein with tomato spotted wilt virus (TSWV), the type member of this genus (Adkins, 2000).

As in other tospoviruses, the GBNV-GN (type isolate, groundnut) is also a tripartite ambisense RNA virus with three negative-stranded linear RNA genomes. The three strands are designated as L (large, 8.9 kb), M (medium, 4.8 kb), and S (small, 3.05 kb) which are encapsidated in quasispherical particles of 80–120 nm in diameter (Satyanarayana et al., 1996a,b; Gowda et al., 1998). Functionally, the L segment which is of negative polarity is responsible for coding an *RNA-dependent RNA polymerase* (RdRp; 330 kDa L-protein with 2877 amino acids); the segment M codes in the sense orientation of the virion for the precursors to glycoproteins Gn and Gc (127.3 kDa) and

the movement protein NSm (34.3 kDa). The segment S with 1320-nucleotide long ORF codes in the complementary sense of the virion for a nonstructural protein NSs (49.5 kDa), which is the silencing suppressor, and the other ORF of 831 nucleotides in the complementary strand produces the nucleocapsid protein N (30.6 kDa). The NSs protein is reported to have *RNA-stimulated ATPase* and 5′ phosphatase activities helping overcome the host defense mechanism. The N protein is involved in the viral RNA replication, transcription, and acts as a protective layer in the encapsidation of the genome (Prins and Goldbach, 1998; Takeda et al., 2002; Lewandowski and Adkins, 2005; Li et al., 2009; Mandal et al., 2012; Singh et al., 2014a,b; Li et al., 2015).

GBNV which shares about 29% sequence identity with the N protein of TSWV is included in the serogroup IV. The sequence identity of the N and NSm genes of other isolates of GBNV-infecting plants from *Leguminosae, Solanaceae*, and *Cucurbitaceae* plants is also included in the serogroup IV (Satyanarayana et al., 1996b; Bhat et al., 2002; Anjaneya Reddy et al., 2008; Jain et al., 2004; Akram et al., 2004; Lokesh et al., 2010). Comparison of the amino acid sequences of the Ns and N proteins of GBNV revealed an identity of 22%–30% with the other reported tospoviruses from other serogroups while it had 82%–86% identity with the serogroup IV. However, the sequences of M RNA and Gn/Gc protein regions had only 56%–89% sequence identity in intergenic regions of GBNV isolates from groundnut, tomato spotted wilt virus (TSWV, serogroup I), and impatiens necrotic spot virus (INSV, serogroup III) genomes, and 44%–47% similarity with the genomes of other viruses from the genus *Bunyavirus* (Mandal et al., 2012).

Host range

The host range of GBNV is very wide infecting a large number of crop plants like brinjal, carrot, *Citrullus lanatus,* cotton, cowpea, chili, *Cucurbita maxima*, groundnut, jute, mungbean, pea, pepper, rajmash, potato, *Solanum nigrum*, soybean, sunflower, tobacco, tomato, and ornamental plants like *Anthurium*; cosmos, *Gomphrena globose*, *Impatiens* sp., *Petunia hybrida*, *Vinca rosea*, and zinnia. The weed species such as *Acalypha indica, Ageratum conyzoides, Alysicarpus rugosus, A. longifolia, Ageratum conyzoides, Acanthospermum hispidum, Achyranthus aspera, Cassia tora, Catharanthus roseus, Chenopodium quinoa, C. amaranticolor, Corchorus tilocularis, Commelina benghalensis, Commelina jacobi, Calotropis gigantea, Cyanoptis cuculetta, Datura stramonium, D. metal, Desmodium triflorum, Eclipta alba, Euphorbia geniculata, Lagasca mollis, Lochnera pusilla, Physalis minima, Parthenium hysterophorus, Sesbania rostrata, and Vigna triloba* have been reported to have GBNV and symptoms thrips damage near groundnut-growing fields (Reddy et al., 1991; Bhat et al., 2002; Umamaheswaran et al., 2003; Thien-Xuan et al., 2003, Akram et al., 2004; Akram and Naimuddin, 2010; Raja and Jain, 2006; Hemalatha et al., 2008; Sivaprasad et al., 2011; Gopal et al., 2011a Akram and Naimuddin., 2013, Vemana et al., 2015; Pavithra et al., 2016; Basavaraj et al., 2017; Babu et al., 2020; Renuka et al., 2020; Amruta et al., 2020, Bhat et al., 2020).

Transmission

Though the presence of GBNV particles was confirmed in the pods and seeds of groundnut, the disease was not found to be seed transmitted as the presence of the virus was not reported in the embryos (Pappu et al., 2009). However, the high population density of thrips was recorded in crop fields that are affected by viruses (Garcia et al., 2000; Sharma et al., 2003; Culbreath et al., 2003). Initially, the thrip species, *Frankliniella schultzei* and *Scirtothrips dorsalis,* were reported to be the vectors of GBNV (Amin et al., 1981). Subsequent investigations have established that *Thrips palmi* (*Thysanoptera: Thripidae*) is the vector of GBNV (Vijayalakshmi, 1994; Reddy et al., 1995; Ghosh et al., 2017). Thrips can affect the crop directly through feeding injuries (Childers and Achor, 1995; Childers and Bullock, 1999; Heming, 1993; Lewis, 1973) and indirectly by transmitting plant viruses (Riley et al., 2011). A vector count of 10 adults of *T. palmi* per plant could lead to the transmission of the virus up to the extent of 100% (Vijayalakshmi, 1994). The relationships between thrips and tospoviruses are yet not very clear. Though the disease is mainly transmitted by the adult thrips, the second instar larvae can also transmit the virus if they acquire the virus during the first-instar larval stage (Sakimura, 1963; Moritz et al., 2004; Ullman et al., 1992). Studies on *Frankliniella occidentalis* spreading tomato spotted wilt virus (TSWV) have revealed that the virus infection starts from the midgut epithelium and progresses toward the surrounding muscles, and is then transported through the midgut to the salivary gland (Ullman et al., 1992, 1997; Moritz et al., 2004; Whitfield et al., 2005). The virus gets multiplied in the salivary glands, while feeding process virus gets transmitted to healthy plants. Further, alterations in the feeding behavior of the infected vectors as compared to uninfected ones also have been reported (Stafford et al., 2011; Ogada and Poehling, 2015). Daimei et al. (2017) reported a significant difference in the development period of the GBNV-infected thrips as compared to the uninfected ones. However, their preadult mortality, adult longevity, and fecundity were not affected. The probable receptors in the thrips to TSWV have been predicted through differential transcriptomic and proteomic studies (Schneweis et al., 2017; Badillo-Vargas et al., 2012). The possible interactions of glycoproteins of GBNV were predicted with *T. palmi* C-type lectin, enolase, clathrin, cathepsin, and *vacuolar ATP synthase* subunit E. It has been proposed through in silico analysis that C-type lectin is the primary cellular receptor interacting with GBNV-GN which facilitates the entry of the virus particles to the vector cells by clathrin-mediated endocytosis (Jagdale and Ghosh, 2019).

Management of the PBND/GBNV

Cultural practices

Planting time has been found to lower the incidence of PBND by avoiding the coincidence peak population of thrips with a more susceptible seedling stage (Brown et al.,

1996; McKeown et al., 2001; Culbreath et al., 2010). Early sown (first fortnight of June) and paired row planted crops generally get less incidence of the disease while the incidence was maximum in the late sown crops (first fortnight of August) and face dry spells during the crop growth (Ghewande, 1983; Reddy et al., 1983; Dharmaraj et al., 1995; Singh and Srivastava, 1995; Brown et al., 1996; McKeown et al., 2001; Culbreath et al., 2010; Tubbs et al., 2011). The weather parameters during the crop growth stages influencing the incidence of PBND in groundnut were studied in detail by Vijayalakshmi et al. (2017) and Naresh et al. (2018). The peak activity of thrips and GBNV was reported when plants were 30-days old, in *Kharif* season during August-September and in *rabi* season by the end of February and March. There was a negative correlation of thrips density with RH of morning and evening and a positive correlation with maximum temperature (T_{max}) and minimum temperature (T_{min}), rainfall and sunshine hours, respectively. Evening RH and T_{min} showed a negative correlation with GBNV infection while $T_{max,}$ morning RH, rainfall, and sunshine hours were positively correlated. The disease development depends on infection by the viruliferous thrips which had acquired the virus from their alternate hosts as well as their extent of migration. The migration has been reported to be maximum when the air temperature is between 20°C and 30°C and wind velocity of 10 km/h at 3 m above the canopy (Thiara et al., 2004; Prasada Rao and Reddy, 2005; Pensuk et al., 2010). Thus warm dry weather is conducive for the build-up of thrip population and disease development.

Planting density also was found to play a role in managing the PBND. Lower plant population (<23 plants/m^2) as against optimum population (33 plants/m^2) was reported to have a higher incidence of the disease compared with crops planted at higher population density (Reddy et al., 1991; Bhatnagar et al., 1995). Removing the infected plants from the field, especially during the early stages of plant growth, can result in lower plant density and may increase the incidence of viral infections (Reddy et al., 1991, 1995). However, roughing may help in keeping the viral inoculum in the field at a lower level. Early planting and increasing the planting density have limitations and other disadvantages depending upon the geographical, climatic, and varietal characteristics. Approaches like conservation tillage also were reported to reduce the thrips density in groundnut as compared to conventional tillage (Brown et al., 1996; Monfort et al., 2007).

Intercrops and trap-crops have been reported to be effective in reducing the incidence of PBND through controlling the vector movement. Bajra, maize, and sorghum planted in 4–8 rows around the crop field will act as a barrier crop that will prevent wind-borne pollen from weeds and alternate hosts carrying virus and thrips movement. Sunkad et al. (2005) reported that intercropping groundnut with bajra or sorghum or pigeon pea or maize in the ratio of 3:1 could reduce PBND incidence by 60.3% to 69.1%. Gopal et al. (2010a,b) reported that intercropping groundnut with redgram in the ratio 5:1 could reduce PBND to an extent of 10%. Intercropping groundnut+castor in 3–6:1 ratio also was reported to be effective in controlling the incidence of PBND (Singh et al.,

2014a,b). Several weed species (as listed under the hosts for GBNV) can support the virus inoculum during the offseason when the crop is not grown and will contribute to active disease development during the cropping season. Control of these weed species can be done by weeding or by using appropriate weedicides.

Chemical control

Chemical control of the vector *T. palmi* has been reported to be effective in reducing the incidence of PBND (Amin, 1985; Singh and Srivastava, 1995). However, only a limited number of insecticides are available for this (Reddy et al., 1995; Todd et al., 1995, 1996; Herbert et al., 2005; Culbreath et al., 2008; Marasigan et al., 2016). Timmanna et al. (2021) reported the insecticides Spinosad 45SC, Diafenthiuron 50WP, Cyantraniliprole 10.26OD, Fipronil 5SC, Imidacloprid 17.8SL, and Dimethoate 30EC to be effective against thrips. Seed treatment with dimethoate at 1000 mL or imidacloprid @2 mL/kg or drenching with imidacloprid at 200 mL/ha in 500 L of water at 25–30 days after sowing has been recommended for epidemic regions. Chemical control has to be resorted before the appearance of the disease symptoms (Prasada Rao and Reddy, 2005). However, the choice of the right insecticide may depend on their registration in the growing areas and label claims for relevant crops, etc.

Thrips palmi is a difficult-to-control pest and chemicals alone may not be sufficient (Young and Zhang, 1998). The small size and cryptic habits of thrips help them in evading direct contact with insecticides on spaying. Further, their mass migration also necessitates more number of sprays or chemicals with extended residual effect to avoid the recurrence of the thrip population (Lewis, 1973). There are reports of insecticide resistance in thrip populations and thrips have very efficient detoxification mechanisms making them also resistant to several insecticide classes (Espinosa et al., 2005; Bielza, 2008; Bielza et al., 2007, 2008; Bao et al., 2015; Srinivasan et al., 2018).

Bioproducts

With the enhanced use of insecticidal chemicals, the concern on environmental issues is also being flagged, especially on the mortality of the nontarget insects like pollinators and natural enemies by the use of broad-spectrum insecticides (Nicodemo et al., 2014). In this context, the use of biocontrol agents and botanicals for vector control becomes relevant. Kulkarni et al. (2001) used sorghum/coconut leaf extract spray in reducing PBND incidences. Though weekly spray of monocrotophos was found to be very effective in vector control, the spray of 10% neem seed extract or 10% neem cake extract also recorded a significantly low incidence of PBND (Gopal et al., 2011b). Use of a mixture of quinalphos 25 EC (0.05%) and Sorghum leaf extract (10%), or quinalphos 25 EC (0.05%) with coconut leaf extract (10%), could considerably reduce the thrip population in groundnut (Vijayalakshmi et al., 2014).

Vanthana et al. (2022) has reported the exploitation of microbe-associated molecular pattern (MAMP)-triggered immunity (MTI) in controlling GBNV infecting tomato. The use of flagellin (Flg) and elongation factor (EF-Tu) could induce the defense response in GBNV. When *Agrobacterium tumefaciens* expressing these MAMP genes was used for drenching the root zone or as a foliar spray, the secondary metabolites of *Bacillus amyloliquefaciens* (VB7) released by the *A. tumefaciens* could effectively suppress the symptoms of GBNV.

There are a few reports on the antiviral activities of endophytes and other SAR-inducing agents which are tested in other plant viral diseases. These can also be profitably adapted for the control of GBNV. Recently, Sorokan et al. (2020) reported that endophytic *Bacillus thuringiensis* B-5351, *B. subtilis* 26D, and *Bacillus* sp. TS2 could directly produce *RNAase*s in the plant tissue resulting in effective control of viral diseases of potatoes. They have further proposed the use of these strains in developing biocontrol agents (insecticide+viricide) which are multifunctional. ZhiNengCong (ZNC), a plant immune inducer, even at a very low concentration, could enhance resistance to the potato X virus through the salicylic acid pathway by positively regulating the RNA silencing (Peng et al., 2020). This points to the possibility of using ZNC as a new antiviral bioagent. Harish et al. (2009) reported that banana bunchy top virus could be controlled up to 80% under green-house conditions and up to 52% in the field using bioformulations containing isolates of *Pseudomonas fluorescens* (Pf1), a rhizobacterium and endophytic *Bacillus* spp. (EPB22). Plant growth-promoting rhizobia (PGPR) strain GBO3 was reported to work as inducers in controlling blackeye cowpea mosaic strain of bean common mosaic virus (BCMV) in cowpea in both screen-house and field conditions (Udaya Shankar et al., 2009). The antiviral activity of phenanthroquinolizidine alkaloids was tested in tobacco mosaic virus (TMV) and was found that the alkaloid interacts with the viral RNA. Their antiviral activity was more than that of antofine and commercial ningnanmycin, ribavirin, etc. The compounds containing both phenanthrene and N-heterocyclic ring could maintain the anti-TMV activity of phenanthroindolizidines; though their mode of action was different from that of antofine (Wang et al., 2012; Yu et al., 2015). Yan et al. (2021) developed 9-ethoxy methyl tylophorine with a high anti-TMV activity which has the potential to be used as an effective inhibitor of plant viruses. Despite the reports on several such molecules with proven antiviral activities, as well as the commercially available antiviral molecules, they are yet to be used in the management of the GBNV.

Use of resistant/tolerant/transgenic genotypes

Growing genetically resistant cultivars are considered the most reliable and eco-friendly approach to reduce economic losses due to the diseases. However, the virus resistance reported in groundnut may not be the sole outcome of host-virus interactions but could

also be influenced by host-vector interactions (do Nascimento et al., 2006; Sundaraj et al., 2014; Shrestha et al., 2015) or both. Most often, the genetic resistance in groundnut to GBNV has been referred to as a field resistance (determined by field-level screening of the genotypes) which is the cumulative outcome of host plant resistance to the virus as well as to the thrip vector. The plants which are identified as genetically resistant in the field have very poor multiplication and systemic movement of the infecting virus from the site of infection (Buiel, 1996). The host plant resistances along with the cultural practices and chemical management targeted at thrips are the most adopted approach. The mechanism and genetics of host plant resistance to GBNV and thrips are not yet completely known (Sundaraj et al., 2014; Shrestha et al., 2015). The use of resistant cultivars could reduce the extent of epidemic development by reducing the GBNV incidence considerably (Culbreath et al., 1993; Buiel and Parlevliet, 1996). It can further reduce the usage of insecticides and allied nontarget effects leading to the enhancement of the sustainability of the production system.

Wild species and interspecific derivatives

The wild species of the cultivated groundnut (*Arachis hypogaea*) are reservoirs of genes for disease and pest resistance. The cultivated groundnut is an allotetraploid with a very narrow genetic base and without innate resistance to thrips, or to viruses that are thrip-transmitted (Ratnaparkhe et al., 2011). However, several wild species of *Arachis* were reported as resistant to GBNV. At International Crop Research Institute for Semiarid Tropics (ICRISAT), Hyderabad, India, over 8000 germplasm accessions were screened for their resistance to PBND (Nigam et al., 2012). Reddy et al. (2000) did an extensive screening of 83 accessions of wild *Arachis* species both in the field and under artificially inoculated conditions. Under field conditions, *A. benensis* (ICGI 1551), *A. cardenasii* (ICG 11564), and *A. villosa* (ICG 13168 and ICG8144) from the section *Arachis*; *A. appressipila* (ICG 8945 and ICG 8946) from the section *Procumbentes*; and *A. triseminata* (ICG 8131) from the section *Triseminatae* did not develop the disease even under adequate disease pressure or under artificially inoculated conditions. This could probably be due to the failure of virus movement within the plants. Though *A. benensis* (ICG 11551) belonging to section *Arachis* and *A. appressipila* (ICG 8945 and ICG 8946) belonging to the section *Procumbentes* were not infected by GBNV in the field, they developed PBND symptoms on artificial inoculation suggesting that their field resistance may be due to their vector resistance. The species in the section *Arachis* are cross-compatible with the cultivated species *A. hypogaea*, and *A. cardenasii* has already been used in crop improvement programs. Several other wild species of *Arachis* having resistance to thrips are been reported by other workers as well (Amin and Mohammed, 1980; Stalker and Campbell, 1983; Kolte, 1984; Reddy, 1988; Lynch, 1990; Dwivedi et al., 1995; Reddy et al., 2000; Bera et al., 2004; Stalker, 2017; Michelotto et al., 2017) (Table 1).

Table 1 Reaction of wild species of *Arachis* to thrips.

Sl. No	Species	Response to thrips
1.	*Arachis*. sp. GKP 105773	Immune
2.	*A. batizocoi* K 9484	Immune
3.	*A. repens* GKP 20538	Immune
4.	*A. kempff-mercadoi* GKP 10127	Immune
5.	*A. pusilla* GKP 12922	Immune
6.	*A. glabrata* GKP 9830	Immune
7.	*A.* sp. GK 12934	Highly resistant
8.	*A.* sp. GK 12946	Highly resistant
9.	*A. correntina* GKP 9530,	Highly resistant
10.	*A. chacoense* GKP 10602	Highly resistant
11.	*A. cardenasii* GKP 10017, ICG 11564, 13164, 13165	Highly resistant
12.	*A. appressipila* GKP 9990	Highly resistant
13.	*A. stenosperma* HLK 410	Highly resistant
14.	*A. sp* PI 262848	Resistant
15.	*A. spegazzinii* GKP 10038	Resistant
16.	*A. hagenbeckii* HL 436	Resistant
17.	*A. villosa* ICG 8144	Resistant
18.	*A. duranensis* 30064, 30065, 36002, 36002-2, 36005	Resistant
19.	*A. paraguariensis*	Resistant
20.	*A. vallsii*	Resistant
21.	*A. williamsii*	Resistant
22.	*A. diogoi*	Resistant
23.	*A. benensis*	Resistant
24.	*A. volida* 30011	Resistant
25.	*A. monticola* 30063	Resistant
Amphidiploids		
1.	*A. batizocoi*×*A. kempff-mercadoi*	Resistant
2.	*A. gregoryi*×*A. stenosperma*	Resistant
3.	*A. magna*×*A. cardenasii*	Resistant

However, most of the wild species are diploids while the cultivated groundnut (*Arachis hypogea*) is an allotetraploid (AABB, $2n = 4\times = 40$) resulted from the hybridization of the two diploids progenitors, *A. duranensis* (AA, $2n = 2\times = 20$) and *A. ipaensis* (BB, $2n = 2\times = 20$) followed by the process of evolution and speciation, and *A. monticola* is considered as the immediate predecessor wild species of *A. hypogaea*. This difference in ploidy makes introgression of these traits difficult through conventional breeding methods. The bottleneck is further compounded by incompatibility issues in hybridizations where only the members of the section *Arachis* are crossable. To overcome this, approaches like development and use of synthetic tetraploid or amphidiploids have been attempted (Leal-Bertioli et al., 2015). Under the ongoing programs of the ICAR-Directorate of Groundnut Research (ICAR-DGR), Gujarat, India, several interspecific

derivatives were developed and tested under field conditions for the tolerance to PBND. Of these, CS-2, CS-8, CS-36, CS-46,CS-51, CS-55, CS-57, CS-58, CS-75, CS-79, CS-82, CS-85, CS-86, CS-102, CS-103, CS-108, CS-159, CS-177, CS-244, CS-246, CS-262, CS-268, CS-285, CS-286, CS-294,CS-300, CS-301, CS-319, CS-327, CS-329, CS-417, and CS-421 were found to be resistant to PBNV (Srinivasaraghavan et al., 2013). Thirty-four prebreeding lines developed from interspecific hybridizations were tested at the hot spot for their field resistance and the lines NRCGCS nos. 79, 81, 85, 86, 159, 246, 267, 271, 275, 282, 285, 286, 300, 301, 319, and 327 were found to be resistant while NRCGCS nos. 36, 46, 51, 55, 57, 58, 82, 102, 103, 153, 161, 177, 262, 269, 417, and 421 were moderately resistant (Jasani et al., 2018). Further, 435 breeding lines developed by interspecific hybridizations of *Arachis hypogaea, Arachis cardenasii, Arachis pusilla, Arachis duranensis, Arachis diogoi, Arachis correntina, Arachis helodes, Arachis monticola, Arachis batizocoi, Arachis stenosperma, Arachis villosa, Arachis kempff-mercadoi, Arachis pintoi, Arachis kretschmeri, Arachis oteroi,* and *Arachis villosulicarpa* developed at the ICAR-DGR were screened at a hot spot for their field resistance (Jignesh et al., 2014). Of these, the lines with NRCGCS numbers 2, 7, 8, 36, 46, 51, 55, 57, 58, 75, 79, 81, 82, 85, 86, 102, 103, 108, 153, 159, 161, 177, 244, 246, 262, 267, 268, 269, 271, 275, 277, 281, 282, 285, 286, 300, 301, 319, 327, 328, 417, and 421 were found to be resistant while the lines with NRCGCS numbers 10, 27, 28, 37, 40, 41, 43, 45, 54, 59, 73, 76, 77, 83, 92, 94, 96, 97, 98, 99, 104, 105, 107, 113, 116, 120, 137, 144, 151, 156, 167, 184, 186, 187, 190, 192, 196, 201, 202, 203, 205, 206, 211, 212, 215, 220, 227, 228, 230, 232, 250, 263, 264, 293, 308, 311, 337, 338, 339, 375, 377, 378, 379, 381, 396, 398, 402, 412, 420, 422, 423, 425, and 431 were moderately resistant. Of these derivatives, eight stable lines were registered with the ICAR-National Bureau of Plant Genetic Resources (ICAR-NBPGR), New Delhi, India, as germplasm (Bera et al., 2010a,b,c,d,e,f,g,h) (Table 2).

Conventional plant breeding could also identify/develop sources of resistance of varying degrees to GBNV or PBND (Amin, 1985; Dwivedi et al., 1995; Reddy et al., 2000; Kesmala et al., 2004; Mandal et al., 2012) (Tables 3 and 4). Tabassum et al. (2017) took up field screening as well as artificial inoculation of 40 advanced breeding lines and could identify 13 genotypes with ICGV numbers 00187, 00191, 00202, 00211, 00247, 07222, 06146, 00201, 00206, 00213, 86699, 87846, and 07220 as resistant in field condition. However, none of these genotypes was showed resistance to GBNV under artificial inoculated conditions, probably because of the high inoculum pressure. Further, the study revealed a very significant difference in the disease incidence in the genotypes ICGV 91114 and ICGV 99058, 99072, 00162, 86590, 00308, 93468, ICGS 44 which may be attributed to the genetic factor and morphological characters which may deter the feeding of thrips. Factors like the number of trichomes on leaf stem and petioles, hairiness of the leaf lamina, the thickness of the leaf, dark green leaf color, and higher phenol content were reported to be influencing in deterring the vectors on

Table 2 Interspecific derived germplasm with PBND registered at ICAR-NBPGR.

Sl. No.	Germplasm ID	DGR accession No	Features of the germplasm
1.	INGR 10037	NRCGCS-83	Resistant to PBND, stem rot, late leaf spot, rust, and *Alternaria* leaf blight
2.	INGR 10038	NRCGCS-124	Resistant to PBND, stem rot, late leaf spot, early leaf spot, rust, and *Alternaria* leaf blight
3.	INGR 10039	NRCGCS-180	Resistant to PBND, stem rot, late leaf spot, early leaf spot, rust, and *Alternaria* leaf blight
4.	INGR 10040	NRCGCS-222	Resistant to PBND, stem rot, late leaf spot, early leaf spot, *Alternaria* leaf blight, and tolerant to rust
5.	INGR 10029	NRCGCS-77	Resistant to rust, early leaf spot, late leaf spot, *Alternaria* leaf blight, and tolerant to PBND and stem rot
6.	INGR 10030	NRCGCS-85	Resistant to PBND, stem rot, late leaf spot, early leaf spot, *Alternaria* leaf blight, and tolerant to rust
7.	INGR 10031	NRCGCS-86	Resistant to stem rot, late leaf spot, early leaf spot, rust, *Alternaria* leaf blight, and PBND
8.	INGR 10036	NRCGCS-21	Resistant to PBND, stem rot, late leaf spot, rust, and tolerant to early leaf spot

Table 3 The genotypes reported being tolerant to PBND in field screenings at hot spots.

Genotype	References
EAH1010, EAH1232, and TMV7	Ramapandu and Raychaudhuri (1980)
ICGV86031	Dwivedi et al. (1993)
Robut 33-1	Amin (1985)
ICGS 1	Nigam et al. (1991a)
ICGV 87141 (ICGS 76)	Nigam et al. (1991b)
Kadiri 3, ICGS 5, RS 138, CSMG 881, CSMG 888, and CSMG 892	Singh et al. (1994)
Spanish 5512, Spanish 67-5, ICGS 18, ICGV 86699, J 14, R 8821, R 7015, R 9021, ICG 1703, ICG 2711, EC 2215, ICG 5042, ICGV 98304, RSG 1	Basu (1995)
CSMG-12. ICG 869, ICG 6317, CSMG-15	Singh and Srivastava (1995)
R 8806, R 8970, R 8976, R 9621, R 9251, R 9214, R 9227, R 9204, ICGV numbers 86029, 86030, 86031, 89304, ICG 2271	Dharmaraj et al. (1995)
ICG 239	Ghewande and Desai (1995)
ICGV 86430, 2192-8(50), and 2169-5(9)	Buiel et al. (1995)

Table 3 The genotypes reported being tolerant to PBND in field screenings at hot spots—cont'd

Genotype	References
ICG numbers 848, 851, 852, 862, 869, 885, 2271, 2306, 2307, 2323, 2741, 3042, 3806, 3873, 5030, 5024, 5043, 5044, 6135, 6317, 6323, 7676, and 7892; ICGV 86031, 86,388	Dwivedi et al. (1995)
ICGV 86699	Reddy et al. (1995)
ICGV 86325	Dwivedi et al. (1996)
ICG5323, ICG2866, NRCG 1015, R13, NRCG4400	Desai (1998)
CSMG 12, EC 21070, ICG 98, GBFDS 92, GBPRS 4, GBPRS 15, U 4-7-7, 28/207, Ah 7215, Ah 7286, Ah 7913, BPG 511, Chandra, CSMG 5, CSMG 5-1, CSMG 9, CSMG 15, CSMG 17, EC Nos., 1246, 20957, 21161	Singh et al. (1998)
DRG-18, ICG-7812, ICG (FDRS)-10, ICGV-80325, JSSP-3, KGN-22, PI-393516	Gururaj et al. (2002)
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, DRG 17	Basu et al. (2002)
TCGS-635	Vasanthi (2003)
Pratap Mungphali 1	Nagda and Joshi (2004)
ICGV 92269, 89/94-3-2, ICGV 91229, ICGV 91193, 89/94-7-3, 83/151-7, 85/203-6, ICGV 91248, ICGV 91117, and ICGV 86031	Gopal et al. (2004)
GPBD 4, JSSP 9, and Dh 53	Nagaraja et al. (2005)
Pratap Mungphali 2	Nagda and Dashora (2005)
IC 10, IC 34, ICGV 86031, and ICGV 86388	Kesmala et al. (2006)
ICGV 90009, ICGV 86699, ICGV 86329, 91177, 91234, ICGV-94252, and TG 26	Gopal et al. (2010a,b)
CS-43, CS-45, CS-54, CS-73, CS-77, CS-83, CS-92, CS-94, CS-104, CS-137, CS-156, CS-202, and CS-212	Srinivasaraghavan et al. (2013)
NRCGCS nos. 79, 81, 85, 86, 159, 246, 267, 271, 275, 282, 285, 286, 300, 301, 319, 327	Jasani et al. (2018)
Kadiri Amaravathi	Lakshmi et al. (2019)

Table 4 Groundnut cultivars released in India which have moderate resistance to PBND.

Name of cultivar	Habit	Release	Pedigree
1. M 197	VR	1982	C 501×U 4-7-2
2. ICGS 11 (ICGV 87122)	SB	1986	Selection from Robut 33-0
3. ICGS 44 (ICGV 87128)	SB	1988	Selection from Robut 33-0
4. RG 141	SB	1989	Robut 33-1×NCAc 2821
5. ICGS 1 (ICGV 87119)	SB	1990	Selection from Robut 33-0
6. ICG (FDRS) 10 (ICGV 87160)	SB	1990	Ah 65×NCAc 17090

Continued

Table 4 Groundnut cultivars released in India which have moderate resistance to PBND—cont'd

Name of cultivar	Habit	Release	Pedigree
7. ICGS 37 (ICGV 87187)	SB	1990	Selection from Robut 33-1
8. ICGV 86590 (ICGS 86)	SB	1991	X 14-4-b-19-B×PI 259746
9. Pragathi (RSHY 1)	SB	1991	Selection from "Spanish Mutant-1"
10. JCG-88 (Jagtial 88)	SB	1993	J 11×TG (E)-1
11. Birsa Bold (BAU 13)	VB	1993	BAU 6×MI 3
12. B 95	VB	1993	M13×Shulamit
13. Manikya (DRG 12)	SB	1994	Robut 33-1-mp-1×TAP 4
14. DRG 17 (Mukta)	VB	1994	Robut 33-1×TAP 4
15. ICGV 86325	VB	1994	CGS 20×G 201/KAUSHAL
16. BSRG 1 (ICGV 86143)	SB	1994	ICGV 44×(Robut 33-1xNCAc 2821)
17. K 134 (Vemana)	SB	1995	Kadiri 3×JL 24
18. TG 26	SB	1995	BARCG1×TG 23
19. Apoorva (R 8808)	SB	1997	ICGS 11×Chico
20. R 9251 (KRG-3)	SB	1997	JLM-1×TG-23
21. Kadiri 4 (K 150)	SB	1997	Dh 3-30×NCAc 2230
22. CSMG 884 (Prakash)	VB	1999	Kaushal×Chandra
23. Co 3 (TNAU 256)	SB	1999	VRI 3×JL 24
24. SG 99	SB	2004	ICGV 86829×ICGV 87160
25. Ratneshwar (LGN 1)	SB	2005	JL 24×NCAc 17,090
26. Pratap Mugphali 2 (ICUG 92195)	SB	2005	ICGV 86055×ICG (FDRS 10)
27. Pratap Mugphali 1 (ICUG 92035)	SB	2005	ICGV86033×ICG 2214
28. Co(GN) 5	VB	2005	(Co-2×ICGS-8601)×(Co-2×VG-119×ICGS-50)
29. GG 8 (J 53)	SB	2006	(27-5-1×JL24) 30-3-2-B-B
30. GG 16 (JSP 39)	VR	2006	JSP 14×JSSP4 S-94-15-B-10-1-B-B
31. Vasundhara (Dh 101)	SB	2007	R 9241×Dh 51-2
32. Ajeya (R 2001-3)	SB	2008	ICGS 11×ICG 4728
33. VRI (Gn) 6 (VG 9816)	SB	2009	ALR 2×VG 9513
34. Mallika (ICHG 00440)	VB	2009	(ICGV 88386×ASHFORD)×ICGV 95172
35. Vijetha (R 2001-2)	SB	2010	ICGS 11×ICG 4728
36. Kadiri Haritandhra (K 1319)	SB	2010	91/57-2×PI 476177
37. GJG-HPS-1 (JSP-HPS-44)	VR	2010	JSP 21×VG 5
38. Divya (CSMG 2003-19)	VR	2011	Amber×ICG-1697
39. Pratap Raj Mungphali	SB	2011	Selection from ICGV-98223

Table 4 Groundnut cultivars released in India which have moderate resistance to PBND—cont'd

Name of cultivar	Habit	Release	Pedigree
40. Central Groundnut ALG-06-320	SB	2017	(J 11 × CG 52)×ICGV 86015
41. Kadiri Amaravathi (K 1535)	SB	2017	Kadiri 6×NCAc 2242
42. Konkan Bhuratna (RTNG-29)	VB	2019	PBS 24030×GPBD 4
43. K 1719 (Kadiri Chithravati)	SB	2020	Kadiri 7 Bold×TAG 24
44. K 1812 (Kadiri Lepakshi)	SB	2020	((ICGV 92069×ICGV 93184) SIL4×ICGV 98300)

VB, Virginia Bunch; *VR*, Virginia Runner; *SB*, Spanish Bunch.
Compiled based on the data available with the All India Coordinated Program on Groundnut (AICRP-G), ICAR-Directorate of Groundnut, Gujarat, India.

feeding on the plants and hence, reducing the incidence of PBND (Campbell and Wynne, 1980; Yang et al., 1993; Kandakoor et al., 2014; Jasani et al., 2018). The higher cuticular vax content of the wild species of groundnut (*Arachis glandulifera, A. batizocoi, A. paraguariensis, A. chacoense, A. glandulifera, A. monticola,* and *A. ipaensis*) might be playing a role in deterring thrips from feeding (Yang et al., 1993; Souza et al., 2010).

The association of particular habit group/market types of cultivated groundnut with the resistance has been reported by several authors (Amin and Mohammed, 1980; Campbell and Wynne, 1980; Lynch, 1990; Mulder, 1999; Mulder and Seuhs, 2002; Herbert et al., 2005; Whalen et al., 2014), and Virginia-type cultivars are more susceptible as compared to the other market types. The observed genotypic difference in the resistance to thrips may also be due to the antixenosis by which the vectors may not prefer to feed on certain types of host genotypes (Painter, 1951; Kogan and Ortman, 1978; Smith, 2005). However, it is very difficult to explain this genotypic difference when the groundnut crop itself has a very limited variability. This phenomenon has again been explained as a mechanism of resistance called Antibiosis where host plants affect the biology of the vector resulting in a reduction in the fecundity and longevity or higher mortality (Teetes, 1996).

Genetics of host resistance

Three additively inheriting factors, without epistasis and dominance, were reported to be involved in reducing the incidence of PBND, which was stable in different environments (Buiel, 1996). Pensuk et al. (2002) reported that the inheritance of resistance (reduced disease incidence) is additive while it has highly significant general combining ability (GCA) affecting significantly SCA and reciprocal effects. Hence, it was also suggested

to use the resistant source as the female parent. For the incidence as well as the severity of the disease, high heritability was reported in the parental lines and had both genotypic and phenotypic correlations with desirable agronomic traits and disease resistance (Kesmala et al., 2003, 2004; Tonsomros et al., 2006). Later on, Pensuk et al. (2004) also reported nonadditive gene effects associated with the low incidence of PBND and suggested selection in later generations. Poledate et al. (2007) found both additive and additive epistasis for disease severity and disease incidence, respectively, while dominance and epistatic gene effects were reported by Niyomsil et al. (2007).

Cultivar development

Programs on breeding for resistance to PBND should focus on improving host plant resistance to both vector and virus which are to be deployed in desirable agronomic backgrounds. The possibility of utilizing the resistant wild species which are cross-compatible with the cultivated groundnut also can be exploited profitably through appropriate breeding procedures for wide gene transfer.

The relatively new cultivars which are bred using already identified resistant parental lines show this difference in the field (Table 4). There is an inherent limitation in field screening as the disease intensity is highly influenced by the environment and hence varies from year to year (Culbreath et al., 2010; Tseng et al., 2016), while the artificial inoculation possibly bypasses some of the plant defense responses and finally not fully reliable (Zhao et al., 2018).

Modern tools

The host plant resistance to the GBNV and the vector thrips is not absolute and requires additional management practices to contain the PBND incidence (Mandal et al., 2012; Marasigan et al., 2016). The advent of modern tools, especially the availability of genomic resources and tools, has rekindled the zeal in breeding for resistance to the virus and vectors. Recent developments in screening groundnut genotypes for thrips resistance in the lab have given an edge over the field screening which has been followed conventionally (Shrestha et al., 2013; Sundaraj et al., 2014; Thoen et al., 2016). To boost this now, a behavior-analyzing software "Ethovision® XT 10" is also available which could determine the parameters on the vector-host plant interactions. Another tool now available is "Electronic nose," which can be effectively used in the screening of thrips populations in groundnut.

During the last two decades, a good number of genomic resources including the whole genome sequence of the cultivated groundnut (*A. hypogea*), its two progenitor wild species (*A. duranensis* and *A. ipaensis*), and several other wilds species are made available. Several molecular markers are also now available for trait-based mapping and associations so that those can be used profitably in breeding programs. Marker-assisted selection can be exploited in pyramiding genes responsible for GBNV resistance as well

as thrips resistance in one genotype. Thus, the selection pressure on the virus will be reduced and delay the resistance development by the virus leading to the reduction in the use of insecticides and protect nontarget insects, and finally, result in a sustainable production system (Srinivasan et al. 20,018).

Efforts are on to associate molecular markers with resistance to PBND, which involve both virus and vector. The only report in marker association thrips is of groundnut rosette virus which is transmitted by *Aphis craccivora* (Herselman et al., 2004). The first report of QTLs associated with GBNV resistance was from Jadhav et al. (2019). Of the 14 QTLs identified from recombinant inbred lines from the cross TAG 24 × ICGV 86031, one QTL q60DI located on LG_AhII and the other QTL q90DI were identified with a high PVE. For disease incidence, nine significant additive×additive (AA) interactions were detected, and for yield-related traits, two interactions in parental types while seven interactions displaying effects favoring combinations of the recombinant genotypes were also observed. Employing inclusive composite interval mapping in a RIL population of the cross JL-24 × NRCGCS-85, Jasani et al. (2021) reported two major QTLs (QTL_{PBND}-01 and $QTLP_{BND}$-02) for PBND resistance which could explain 12.38%–16.88% of phenotypic variance. The marker AHS0006 associated with the QTL_{PBND}-01 was found to be coding for sialyl transferase-like protein 1 responsible for protein glycosylation and sialyltransferase activity.

QTLs associated with TSWV, which is transmitted by the *T. palme*, have been reported by Khera et al. (2016) and Tseng et al. (2016). From these reports, it is not apparent that the resistance is for the virus or the vector as the phenotyping was done under field conditions. Khera et al. (2016) prepared an enhanced genetic linkage map accommodating 248 maker loci with a density of 5.7 cM/loci. Using multiseason phenotyping data and the linkage map, 48 quantitative trait loci (QTLs) were discovered of which six QTLs were associated with resistance to TSWV. The source of resistance in the RILs may be NC94022, one of the parental lines (Culbreath et al., 2005; Qin et al., 2012). A major QTL associated with TSWV was reported in Florida-EP™ "113" (Tseng et al., 2016). One of the parental materials *A. hirsuta* (PI 576638) which has TSWV resistance might have contributed to the resistance in the RILs. If these QTLs associated with resistance to TSWV are contributing to the thrip resistance as well, those can be profitably used in breeding for resistance to GBNV also.

Transgenic resistance

With the limited availability of sources of durable genetic resistance in the germplasm of cultivated groundnut, transgenics become a viable option for virus resistance. A transgene can be pathogen-derived (from the genome of the virus itself) on other sources including resistant host plants, other organisms, or synthetic. Among pathogen-derived genes, viral coat protein, movement protein, and replicase are used frequently (Magbanua et al.,

2000; Baulcombe, 2002; Yang et al., 2004; Raja and Jain, 2008, 2012; Yadav, 2009; Phaneendra et al., 2010; Mehta et al., 2013; Peng et al., 2014; Patil et al., 2017; Senthilraja et al., 2018; Gogoi et al., 2019).

The first report on genetic transformation in groundnut was from Brar et al. (1994) who used the transgene *cry* derived from *Bacillus thuringiensis* which could give resistance to lepidopteran pests. However, transgenic resistance to thrips in groundnut has not been reported so far even though such resistance to thrips has been reported in other crops (Outchkourov et al., 2004). Venkatesan et al. (2009) studied the efficiency of the N gene of GBNV in rendering PBNV resistance in tobacco transgenic lines using the sense or antisense orientations of the gene. The transgenics were showing varying levels of RNA-mediated resistance through posttranscriptional gene-silencing (PTGS). Transgenic resistance to GBNV was developed by expressing the nucleocapsid protein gene of the virus in the cultivar Kadiri 6 using Agrobacterium-mediated transformation (Yadav, 2009). Groundnut transgenics expressing nucleocapsid protein (N gene) of GBNV were developed by Rao et al. (2006, 2013) in the cultivar JL24. Though they could evaluate more than 200 transgenic lines, only one line could show a 75% reduction in disease incidence, and they have concluded that the transgenics have partial and nondurable resistance to PBND. Transgenic cultivar Kadiri 6 expressing the NC gene of GBNV was produced and evaluated for their resistance to the virus. The transgenic plants on artificial inoculation with GBNV showed delayed and less intense symptoms showing their resistance acquired through the transgene (Yadav, 2009; Patil et al., 2017). Further, they have produced Kadiri 6 transgenics expressing both CP gene of a tobacco streak virus (TSV) causing peanut stem necrosis disease (PSND) and NC gene of GBNV which also reported resistance to both the viruses on challenge inoculation (Patil et al., 2017).

RNA interference (RNAi)

The role of RNAs in gene expression has been profitably exploited in the control of plant viruses. Silencing of the RNA or RNA interference (RNAi) regulates and restricts the number of transcripts through suppression of transcription (TGS) or by degrading sequence-specific RNA. The viral dsRNA is degraded with the help of an enzyme complex into 21 to 22 bp long dsRNA (short interfering, siRNAs). Small interfering RNAs are recognized by the regulatory mechanism of RNAi leading to the sequence-specific degradation of target mRNA (Fire et al., 1998; Fire, 2007; Whyard et al., 2009; Gan et al., 2010; Zhang et al., 2013; Gandhi et al., 2021).

To obtain a wide spectrum resistance, gene-silencing approach targets the conserved sequences in the GBNV genome (Goswami et al., 2017). To harbor GBNV-derived siRNA, a microRNA159a was synthesized with 21-nt siRNA sequences from RNAi suppressor (GBNV-NSs and PRSV-Hc-Pro) genes. It was found that transgenics expressing virus-specific siRNAs using artificial-miRNA could silence GBNV. Swamy et al. (2015) used *Agrobacterium* based on planta transformation technique in the groundnut

cultivar GPBD 4 to transfer gene constructs containing the coat protein gene of GBNV. The challenge-inoculated transgenic plants were reported to be resistant to GBNV. RNAi has been successfully demonstrated in reducing reproductive fitness of thrip species *Frankliniella occidentalis* by silencing vacuolar ATP synthase (Badillo-Vargas et al., 2015). The technology of RNAi is very target-specific, and hence, there may be minimal nontarget effects.

The spray of culture filtrates of *Ganoderma lucidum* at 0.1% could reduce the GBNV infection in tomatoes (Sangeetha et al., 2020). Foliar spray of an aqueous suspension of bacterially expressed double-stranded RNA (dsRNA) derived from the full-length NSs gene of GBNV, containing 0.01% celite could reduce the infection of GBNV (Gupta et al., 2021). Such exogenous application of dsRNA specific to the tobacco mosaic virus (TMV) could give resistance to TMV by limiting the systemic movement of the virus (Konakalla et al., 2016). However, the amount of dsRNA that can enter the plant system by such exogenous application is minimal (Gogoi et al., 2017; Kaldis et al., 2018; Namgial et al., 2019). Hence, a suitable and efficient delivery system has to be developed to utilize this for plant protection from the virus. Thagun et al. (2022) recently devised a high-throughput nucleic acids delivery system using cell-penetrating peptide nanocarriers spray on leaves. This approach, which may be a replacement for genetic transformation, could induce gene silencing mediated by siRNA-CPP complexes. Further, this strategy of exogenous applications of RNA may help in addressing the regulatory hurdles and concerns associated with transgenics of the consumer.

Conclusion

With detailed field studies and artificial inoculations, it has now been established that PBND is caused by GBNV. It has also been established that the disease-causing virus is transmitted by the vector *Thrips palmi*. The virus genome has been well worked out and fully known, including the variability in the isolates across different groundnut growing areas. For the control of the PBND, there are different approaches including utilization of the genetic resistance to the thrip vector, genetic resistance to the casual virus GBNV, and a combination of management practices including cultural and chemical control. Our knowledge of the genetic resistance to thrips in groundnut is derived from the field level screening of several genotypes including the cultivated and wild species of *Arachis*. Though host plant resistance to the vector is considered as one of the major options, such resistance sources are very rare in the cultivated species but are available in some of the wild species. The genetics of thrips resistance is not worked out in groundnut although such information is available in other crop species like tomato and pepper. Similarly, several wild species of *Arachis* also have genes for resistance to the casual virus by delaying the virus multiplication and arresting the systemic movement of viral particles in the infected plant. However, the cultivated groundnut does not have or very little

inherent resistance to GBNV. The genetic resistance reported from the field screening may not be solely due to the host-virus interaction, and host-vector interaction also plays a very vital role. The genetic resistance available in the wild species remains largely underutilized due to the breeding barriers resulting from the ploidy difference between the wild species and the cultivated groundnut as well as the cross-compatibility issues. Thus, the breeding for resistance to PBND resistance should encompass high levels of resistance to both virus and the vector, expressed in superior and acceptable agronomic backgrounds. There are different strategies of conventional and modern plant breeding including genomics-assisted/molecular breeding available now for pyramiding these genes for both vector- and viral-resistant genes to attain this goal. Pyramiding of multiple resistance genes can reduce the selection pressure and thus slow down the evolution of the vector/virus to more virulent ones ensuring the sustainability of groundnut cultivation.

With the availability of a good number of genomic resources in cultivated and wild species of groundnut together with the approaches in genomics-assisted and molecular breeding, exploitation of the resistance is from the far related species of *Arachis*. Marker-assisted breeding and identification of QTLs which are tagged with molecular markers are making this effort easier. The information on the genome sequence of GBNV has opened up the possibility of utilizing this information in developing transgene constructs and developing groundnut plants expressing these transgenes and hence improved resistance. The understanding of the RNAi also has allowed designing several small RNAs which can silence the viral gene expression in host plants. The utilization of transgenics in agriculture is a matter for statutory regulation which may vary from country to country and several countries do not allow their use. However, recent studies have come up with viral gene silencing by an extraneous spray of transformation vectors or dsDNA itself. Though the approach was found to be effective, the delivery mechanisms were not efficient. Very recently, a delivery mechanism using nanoparticles has been reported with is kindling good hope on exploiting this environment-friendly and non-transgenic approach for viral gene silence and hence control of PBND.

Undoubtedly, host plant resistance is the first order of management option for PBND, but it can be better manifested in combinations with cultural practices and chemical control of the vector. However, concerns about the indiscriminate use of insecticides and the ecological damage have been on the rise. This option of using host plant resistance together with other agronomic management practices becomes more relevant in the absence of high levels of host plant resistance.

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