Pathology

Sources of Resistance to Bud Necrosis Disease in Groundnut

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Bud necrosis is an economically important viral disease of groundnut (Arachis hypogaea) caused by peanut bud necrosis virus (PBNV), vectored by Thrips palmi. It occurs in all major groundnut-growing areas of India and other parts of Southeast Asia (Ghanekar et al. 1979, Gopal and Upadhyaya 1991, Reddy et al. 1991, Dwivedi et al. 1995). The disease incidence on groundnut genotypes differs considerably in the fields (5 to 80%). Low disease incidence observed in certain genotypes is due to the vector non-preference (Buiel 1993). Yield losses due to bud necrosis mainly depend on the time of infection. Infection in <50-day-old plants results in no pod yield and >70-day-old plants are less susceptible to the disease and such plants will have near normal pod setting (Gopal and Upadhyaya 1991). Host plant resistance to PBNV is scarce in the germplasm. Identification of genotypes that can tolerate the disease during early stages of crop growth are useful in mitigating yield loss due to the disease. Therefore in this study, 242 groundnut accessions were evaluated to identify genotypes with field resistance to bud necrosis in three cropping seasons during 1996ñ97 under epiphytotic conditions at the Regional Agricultural Research Station (RARS), Jagtial, Andhra Pradesh, India. Of the 242 genotypes, 190 were from the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, Andhra Pradesh; 15 from the National Research Centre for Groundnut (NRCG), Junagadh, Gujarat; 10 from the University of Agricultural Sciences (UAS), Dharwad, Karnataka; and 27 from RARS, Jagtial. Bud necrosis resistance in various genotypes was assessed based on the percentage of disease incidence and area under disease pressure curves (AUDPC) (Southern and Wilcoxson 1984).

The three trials were sown on 17 July 1996, 20 November 1996 and 15 July 1997 at 45 cm × 20 cm spacing, in 2 rows of 5 m length and replicated twice. One row of cv JL 24, highly susceptible to PBNV was sown after every four rows of test genotypes. Buffer crop of JL 24 was sown around the experimental field to maintain PBNV inoculum. Field operations followed were as per the package of practices of Acharya NG Ranga Agricultural University (ANGRAU), Hyderabad, Andhra Pradesh (Anonymous 1985). No plant protection measures against diseases or pests were used. Bud necrosis incidence was recorded at 10-day intervals, with first observation from 30 days after sowing (DAS) till a week before crop harvest. The infected plants were marked with colored bamboo pegs to facilitate easy recognition of infected plants and to avoid miscounting in case of premature death of early infected plants. After final observation disease incidence (%) was calculated for each observation and AUDPC (A-value) was calculated by multiplying disease incidence (%) with days (duration between DAS and date of observation) (Nagarajan and Muralidharan 1995).

But necrosis was first recorded in JL 24 at 20, 39 and 22 DAS in rainy (kharif) season 1996, postrainy (rabi) season 1996/97 and rainy season 1997, respectively (Table 1). However, disease incidence in JL 24 was variable and was 98% in rainy season 1996, 66% in postrainy season 1996/97 and 69% in rainy season 1997. But highest disease incidence was always in JL 24 during the three seasons tested (Table 1). Only 89/94-3-2 remained free from PBNV infection during all the 3 rainy seasons (Table 1).

Disease incidence (%) and A-values were considered for evaluating the resistance. Based on this, 10 of the 242 genotypes tested were promising resistant sources (Table 1). The genotypes 89/94-3-2, ICGV 92269, 83/151-7 and 85/203-6 consistently recorded low disease incidence and A-value.

The disease incidence accounts the number of plants infected at a given time whereas the A-values account the disease incidence and age of the crop recorded several times during the cropping season to arrive at a single point scoring and measure the disease progress. Thus, A-value represents multipoint scoring of disease incidence reduced to single statistics and offers distinctive advantage in selecting genotypes possessing field resistance and is very useful in identifying field resistant sources (Southern and Wilcoxson 1984). For instance, bud necrosis incidence in 85/203-6, 89/94-7-3 and ICGV 86031 was 2.7, 7 and 8%, respectively and the A-values were 49.7, 255.6 and 102.7, respectively. Although disease incidence in ICGV 86031 was 8% it had a low A-value

Table 1. A-value and bud necrosis disease incidence of some promising groundnut genotypes at Jagtial, Andhra Pradesh, India1.

	Kharif 1996		Rabi 1996/97		Kharif 1997		Mean	
Genotype ²	A	DI	A	DI	A	DI	A	DI
ICGV 92269	48	5	NT ³	NT	0	0	24	2.5
89/94-3-2	0	0	0	0	0	0	0	0
ICGV 91229	286	26	100	11	0	0	128.7	12.3
ICGV 91193	249	22	NT	NT	180	5	214.5	13.5
89/94-7-3	256	7	0	0	255	7	255.6	7
83/151-7	192	14	85	6	0	0	92.3	6.7
85/203-6	149	8	0	0	0	0	49.7	2.7
ICGV 91248	406	28	266	6	0	0	224	11.3
ICGV 91117	194	19	0	0	478	6	224	8.3
ICGV 86031	129	9	143	5	36	10	102.7	8
JL 24 (susceptible check)	2581	98	1820	66	1665	69	2021.9	77.7

A = AUDPC value; DI = Disease incidence (%).

(102.67) compared to 89/94-7-3 (256.6 A-value) suggesting that 7% incidence recorded in 89/94-7-3 occurred at early stage of crop growth and probably would result in greater yield loss. In ICGV 86031 the disease effect on yield would be low because most of the infection would occur with age of the crop. Data in Table 1 clearly indicate the usefulness of A-values over disease incidence (%) in differentiating the promising genotypes.

Three conclusions are drawn from this study: (1) Of the 242 genotypes tested, the genotypes 89/94-3-2, ICGV 92269, 83/151-7 and 85/203-6 were found to be most promising sources of resistance to bud necrosis; (2) There is a great variability in the reaction of groundnut genotypes to PBNV infection; and (3) The evaluation based on Avalues was found to be more useful in identification of promising groundnut genotypes with field resistance to bud necrosis.

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References

Anonymous. 1985. Package of practices for field crops. Rajendranagar, Hyderabad, Andhra Pradesh, India: Acharya NG Ranga Agricultural University. 225 pp.

Bueil AAM. 1993. Resistance in groundnut to peanut bud necrosis virus. Pages 207ñ210 in Durability of disease resistance (Jacobs Th and Parlevliet JE). Dordrecht, Netherlands: Kluwer Academic Publishers.

Dwivedi SL, Nigam SN, Reddy DVR, Reddy AS and Ranga Rao GV. 1995. Progress in breeding groundnut varieties resistant to peanut bud necrosis virus and its vector. Pages 35ñ 40 in Recent studies on peanut bud necrosis disease: proceedings of a Meeting, 20 Mar 1995, ICRISAT Asia Center, Patancheru, India (Buiel AAM, Parlevliet JE and LennÈ JM, eds.). Patancheru 502 324, Andhra Pradesh, India: International Crops Research Institute for the Semi-Arid Tropics.

Ghanekar AM, Reddy DVR, Iizuka N, Amin PW and Gibbons RW. 1979. Bud necrosis of groundnut (Arachis hypogaea L.) in India caused by tomato spotted wilt virus. Annals of Applied Biology 93:173ñ179.

Gopal K and Upadhyaya HD. 1991. Effect of bud necrosis disease on yield of groundnut (Arachis hypogaea L.). Indian Phytopathology 32:122ñ123.

Nagarajan S and Muralidharan R. 1995. Dynamics of plant diseases. Hyderabad, India: Allied Publishers Ltd. 219 pp.

Reddy DVR, Wightman JA, Beshear RJ, Higland B, Black M, Sreenivasulu P, Dwivedi SL, Demski JW, McDonald D, Smith Jr JW and Smith DH. 1991. Bud necrosis: a disease of groundnut caused by tomato spotted wilt virus. Information Bulletin no. 31. Patancheru 502 324, Andhra Pradesh, India:

Disease incidence was recorded 5ñ8 times during the crop growth period starting from 30 days after sowing.

^{2.} All test genotypes were obtained from ICRISAT.

^{3.} NT = Not tested.

International Crops Research Institute for the Semi-Arid Tropics. 20 pp.

Southern JW and Wilcoxson RD. 1984. Effect of planting date on slow rusting of wheat by Puccinia graminis f. sp. tritici. International Journal of Tropical Plant Diseases 1:21ñ24.

Role of In Vitro Resistance to Aflatoxin Contamination in Reducing Pre-harvest Aflatoxin Contamination Under Drought Stress in Groundnut

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Aflatoxin contamination caused by Aspergillus flavus and A. parasiticus has been an important constraint to groundnut (Arachis hypogaea) industry worldwide. Genetic resistance to aflatoxin in groundnut is the most effective solution to this problem. Resistance to fungal infection as well as to aflatoxin contamination have been reported, but breeding for resistance has been slow due to various reasons. In most cases the resistance has been found unstable across locations or seasons and poorly related to pre-harvest contamination. During the past decade, we have been continuously screening for resistance to aflatoxin contamination by using artificial inoculation and toxin test under laboratory conditions and several resistant genotypes have been identified. In this study, the role of in vitro resistance to aflatoxin contamination in reducing pre-harvest contamination under natural conditions was studied by testing four resistant genotypes in potted trials with end-of-season drought stress treatment.

Four resistant lines, H 2030, H 2060, H 2063 and H 2095, and a susceptible line, 88-1202, were planted in 30-cm diameter plastic pots containing sandy loam soil. In each pot, four plants were grown. Potted plants of all the genotypes were normally managed during the first 80 days after sowing (DAS). For the end-of-season drought stress treatment, the potted plants were transferred to water shelter plot and protected from 80 DAS. Irrigation was controlled and the plants showed slight wilting symptom due to water deficit in the later growth stage. The control plants were normally irrigated and did not show wilting symptom. For each treatment, four replications were tested. The plants were harvested at 120 DAS. The seeds were tested for aflatoxin contamination within 30 days after harvest by using the fluoremeter method.

The results of aflatoxin determination are given in Table 1. Based on statistical analysis, the variances of both genotypes and treatment (drought) were significant. For the susceptible control genotype, 88-1202, drought stress treatment significantly increased the aflatoxin content. Under drought stress, the aflatoxin content of 88-1202 was also significantly higher than that of the four test genotypes which were previously identified with resistance to aflatoxin contamination under laboratory

Table 1. Aflatoxin concentration i	n different samples o	f groundnut under	end-of-season drought stress.
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			Aflatoxin content (µg g ⁻¹)				
Genotype	Treatment	I	II	III	IV	Mean	
H 2030	Drought	0.035	0.152	0.034	0.071	0.073	
	Irrigated	0.063	0.047	0.319	0.088	0.129	
H 2060	Drought	0.560	0.369	0.662	0.341	0.483	
	Irrigated	0.107	0.824	0.657	0.749	0.584	
H 2063	Drought	0.081	0.242	0.168	0.329	0.205	
	Irrigated	0.664	0.017	0.200	0.292	0.293	
Н 2095	Drought	0.412	0.529	0.074	0.425	0.360	
	Irrigated	0.688	0.563	0.434	0.011	0.424	
88-1202	Drought	2.254	2.842	1.220	3.143	2.365	
	Irrigated	0.725	0.558	0.796	0.644	0.681	

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