

Effect of Host Genotype on Incubation Period, Receptivity, Lesion Diameter, and Leaf Area Damage of *Didymella arachidicola* on Peanut¹

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ABSTRACT

The effect of host genotype on incubation period, receptivity, lesion diameter and leaf area damage of *Didymella arachidicola* on nine peanut (*Arachis hypogaea* L.) genotypes was investigated under monocyclic infection in the glasshouse. The genotypes, Florunner, P 84/5/256, C 347/5/6, C 346/5/8 and P 105/3/7, resistant to the pathogen in field trials, had a longer incubation period, reduced receptivity, lesion diameter, and percentage leaf area damage, than susceptible genotypes. Among the susceptible genotypes, Tamnut 74 had the shortest incubation period, and highest receptivity, the largest lesion diameter, and percentage leaf area damage. The other susceptible genotypes, Egret, 38/7/20, and P 84/5/112, were intermediate for these variables. Production of pycnidia and pseudothecia of the pathogen could not be demonstrated in infected leaf tissues of any of the genotypes studied. There was significant interaction between plant age and disease development. Younger plants had a shorter incubation period, higher receptivity, larger lesion diameter, and percentage leaf area damage than older plants. Correlation coefficients among incubation period, receptivity, lesion diameter, and leaf area damage were highly significant. The possible role of these variables in disease epidemics and their use in glasshouse screening of peanut germplasm for resistance to *D. arachidicola* are discussed.

Key Words: *Arachis hypogaea*, groundnut, web blotch.

Web blotch caused by *Didymella arachidicola* (Chock) Taber, Pettit & Philley (= *Phoma arachidicola* Marasas, Pauer & Boerema) is one of the most important foliar diseases of peanut (*Arachis hypogaea* L.) in Texas, USA (T.A. Lee, personal communication), Zimbabwe (4,5), and the Republic of South Africa (3,11,25).

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The disease has also been reported in the USSR, Argentina, Brazil, Canada, People's Republic of China, Australia (18,24), Japan (DHS personal observations), and Malawi (PS personal observations). There has recently been increasing interest in screening of peanut and wild *Arachis* germplasm for resistance to web blotch, and several sources of resistance have been reported from various countries (1,12,16,17,22,23). Preliminary field observations in Texas showed that on susceptible peanut genotypes, web blotch develops early in the growing season, progresses rapidly, and causes severe damage to the foliage. On resistant genotypes, the disease appears later in the season, progresses more slowly, and does little apparent damage to the foliage. Recently, the resistance of wild *Arachis* species to *D. arachidicola* was shown to be associated with a reduced receptivity, lesion development, defoliation, and leaf area damaged by the pathogen (23). This article describes an investigation on the effects of host genotypes on incubation period, receptivity, lesion diameter and leaf area damage of *D. arachidicola* on nine peanut genotypes under monocyclic infection in the glasshouse.

Materials and Methods

Test entries are identified by botanical variety and country of origin (Table 1). Tamnut 74 was susceptible and Florunner was resistant to web blotch in Texas field trials (22). Egret, 38/7/20 and P 84/5/112 were susceptible and P 84/5/256, C 347/5/6, C 346/5/8 and P 105/3/7 were resistant to web blotch in Zimbabwe (A.Z. Chitaka, personal communication). Three seeds of each genotype were sown in 10-cm-diameter plastic pots containing sandy loam soil fumigated with methyl bromide. Seedlings were later thinned to one per pot. Plants were fertilized by drenching the soil with a commercial fertilizer mixture (Rapidgro Corp., Danville, NY). Temperature in the glasshouse ranged from 20-25 C during the plant growth period.

Inoculum of *D. arachidicola* (isolate PA/Texas 16) was produced on Difco potato dextrose agar at 20 C under continuous illumination. Pycnidiospores were harvested from 10-day-old cultures by adding

Table 1. Description and source of peanut genotypes included in the experiments, and genotype reaction to *Didymella arachidicola* in previous field trials.

Genotype	Botanical variety	Country of origin	Field reaction to <i>D. arachidicola</i>
Tamnut 74	<i>vulgaris</i>	USA	Susceptible
Egret	<i>hypogaea</i>	Zimbabwe	Susceptible
38/7/20	<i>hypogaea</i>	Zimbabwe	Susceptible
P 84/5/112	<i>hypogaea</i>	Zimbabwe	Susceptible
Florunner	<i>hypogaea</i>	USA	Resistant
P 84/5/256	<i>hypogaea</i>	Zimbabwe	Resistant
C 347/5/6	<i>hypogaea</i>	Zimbabwe	Resistant
C 346/5/8	<i>hypogaea</i>	Zimbabwe	Resistant
P 105/3/7	<i>hypogaea</i>	Zimbabwe	Resistant

sterile distilled water containing (0.2 mL/L) Tween 80 (polyoxyethylene sorbitan monooleate). The suspension was adjusted to a concentration of Ca. 50,000 spores/mL with a hemacytometer. All leaves on the main stem were labeled and inoculated with a plastic atomizer until incipient runoff. Each treatment had five replicated plants arranged in a completely randomized design. Although the method of inoculation was identical in all experiments, plant age and post-inoculation incubation conditions varied.

Experiment 1. Forty-day-old plants were inoculated and placed in a dew chamber (Percival Mfg. Co., Boone, IA) at 20 C with a 12-h dew period (1800-0600) and 12-h photoperiod (0600-1800).

Experiment 2. Forty-day-old plants were inoculated and placed in a polyethylene chamber located in the glasshouse. Plants were misted with water, initially for a 24-h post-inoculation period and subsequently for 14-h periods (1800-0800) until the end of the experiment. Temperature in the polyethylene chamber ranged from 20-25 C.

Experiment 3. Sixty-five-day-old plants were inoculated. Post-inoculation incubation conditions were as in the second experiment.

The method of disease assessment was identical in all experiments. The following variables were assessed.

Incubation period. Four days after inoculation (DAI) and every day thereafter, the number of lesions on the middle leaf of each main stem were counted until there was no further increase in number of lesions. From these data, incubation period was calculated as the number of days between inoculation and appearance of 50% of the lesions.

Receptivity. On the day when increase in the number of lesions on the middle leaf ceased, lesions on each leaflet of the quadrifoliate were counted in a 1 cm² area of the leaf with a CIBA-GEIGY droplet counting aid. Receptivity was expressed as number of lesions/cm².

Lesion diameter. At 30 DAI, the diameters of two randomly selected lesions on each leaflet of the middle leaf (i.e., 8 lesions/leaf) were measured).

Percentage leaf area damaged. At 15 and 30 DAI, the percentage of the area of labeled leaves on the main stem with web blotch damage was estimated with the aid of leaf diagrams with known percentages of their areas affected. In the third experiment percentage leaf area damage was estimated at 30 DAI only.

Sporulation. At 30 DAI, four leaf bits (Ca. 1 cm² size) were excised from the middle leaf of each plant, cleared in saturated chloral hydrate solution for 24 h and examined under a stereomicroscope (x 50) for pycnidia and pseudothecia.

Percentage data were subjected to arcsine transformation. Data from each experiment were analyzed separately and also on pooled data from all experiments. For each character, an analysis of variance was carried out.

Results

The mean values of incubation period, receptivity, lesion diameter and percentage leaf area damage of *D. arachidicola* in all test genotypes are presented in Tables 2 to 6. There were statistically significant ($p <$

0.01) genotypic effects for incubation period, receptivity, lesion diameter, and percentage leaf area damaged. The genotypes which were resistant to *D. arachidicola* in field trials had longer incubation periods (mean 17.0 to 19.8 days) than field susceptible genotypes (mean 7.4 to 16.4 days). There were significant differences in incubation periods between the two botanical varieties of susceptible peanut genotypes. The susceptible Tamnut 74 (var. *vulgaris*) had a shorter incubation period (mean 7.4 days) than the var. *hypogaea* susceptible genotypes (mean 15.1 to 16.4 days) (Table 2). Resistant genotypes exhibited lower receptivities (mean 4.1 to 7.5 lesions/cm²) than the susceptible genotypes. Tamnut 74 showed the highest receptivity (mean 12.9 lesions/cm²). The other susceptible genotypes were intermediate in this character (Table 3). Resistant genotypes had smaller lesions (mean 0.58 to 0.87 mm diameter) than the susceptible ones (mean 1.67 to 2.93 mm diameter) (Table 4). Infected leaflets showed only limited necrosis and defoliation on resistant genotypes resulting in significantly lower percentage leaf area damage (mean 7.8 to 17.2%) compared to susceptible genotypes in which the infected leaflets turned necrotic and defoliated. Tamnut 74 had the highest percentage leaf area damage (mean 83.8%) and the other susceptible genotypes were intermediate (mean 51.07 to 59.8%) (Tables 5 and 6) between it and resistant genotypes. In general, the genotypes which were resistant to *D. arachidicola* in the field had long incubation periods, low receptivities, smaller lesion diameters and lower percentage leaf area damage compared with susceptible genotypes. Among the susceptible cultivars, Tamnut 74 had the shortest incubation period, highest receptivity, largest lesion diameters and the highest percentage leaf area damaged. No pycnidia and pseudothecia of *D. arachidicola* were found in infected leaf tissues of any of the genotypes studied.

Table 2. Effect of host genotype on incubation period¹ of *Didymella arachidicola*.

Genotype	Experiment			Genot. pe mean
	1	2	3	
Tamnut 74	5.2 a ²	5.4 d	11.6 e	7.40 e
Egret	12.6 d	13.6 c	19.2 d	15.13 d
38/7/20	12.8 d	13.8 c	20.2 cd	15.60 d
P 84/5/112	14.0 c	16.0 a	19.2 d	16.40 c
Florunner	14.2 c	15.0 b	21.8 c	17.00 bc
P 84/5/256	14.8 bc	14.4 bc	23.6 b	17.60 bc
C 347/5/6	15.2 ab	16.2 a	26.4 a	19.27 a
C 346/5/8	15.2 ab	16.2 a	26.0 a	19.13 a
P 105/3/7	16.0 a	16.6 a	26.8 a	19.80 a
Experiment mean	13.33	14.13	21.64	
SE	±0.41	±0.05	±0.71	±0.53
CV(%)	5.00	5.43	6.04	3.31

¹ Number of days from inoculation to appearance of 50% of the lesions.

² Means followed by the same letter within a column do not differ significantly at P=0.05 according to Duncan's multiple range test.

Table 3. Receptivity¹ of nine peanut genotypes to *Didymella arachidicola*.

Genotype	Experiment			Genotype mean
	1	2	3	
Tannut 74	13.0 a ²	13.8 a	11.8 a	12.87 a
Egret	10.0 b	9.8 b	7.8 b	9.20 b
38/7/20	9.8 b	9.0 bc	7.0 bc	8.60 bc
P 84/5/112	9.2 b	8.2 bc	7.0 bc	8.13 cd
Florunner	8.6 b	8.0 c	6.0 cd	7.53 d
P 84/5/256	9.8 b	6.0 d	4.0 ef	6.60 e
C 347/5/6	4.6 c	3.6 e	4.8 de	4.33 f
C 346/5/8	5.0 c	4.6 de	2.8 f	4.13 f
P 105/3/7	5.4 c	5.4 d	2.6 f	4.67 f
Experiment mean	8.38	7.60	5.98	
SE	±0.43	±0.48	±0.44	±0.41
CV(%)	14.35	16.70	20.18	8.74

¹ Number of lesions/cm² of leaf area.

² Means followed by the same letter in a column do not differ significantly at P=0.05 according to Duncan's multiple range test.

Table 4. Diameters of lesions¹ caused by *Didymella arachidicola* on nine peanut genotypes 30 days after inoculation.

Genotype	Experiment			Genotype mean
	1	2	3	
Tannut 74	3.1 a ²	2.9 a	2.8 a	2.93 a
Egret	2.6 b	2.3 b	2.0 b	2.29 b
38/7/20	2.6 b	2.2 b	1.9 b	2.24 b
P 84/5/112	1.6 c	1.5 c	1.9 b	1.67 c
Florunner	0.6 d	0.6 de	0.5 c	0.58 c
P 84/5/256	0.8 d	0.9 de	0.6 c	0.77 de
C 347/5/6	0.7 d	0.6 e	0.6 c	0.61 e
C 346/5/8	1.0 d	1.0 d	0.6 c	0.87 d
P 105/3/7	0.9 d	0.9 de	0.4 c	0.76 de
Experiment mean	1.54	1.45	1.25	
SE	±0.14	±0.12	±0.13	±0.12
CV(%)	20.61	18.43	18.26	11.61

¹ Lesion diameter in mm. Measured eight lesions per leaf.

² Means followed by the same letter in a column do not differ significantly at P=0.05 according to Duncan's multiple range test.

There was a significant interaction between plant age and disease development. Younger (40-day-old) plants used in experiments 1 and 2 showed shorter incubation periods (mean 13.33 and 14.13 days) than older (65-day-old) plants (mean 21.64 days) used in experiment 3 (Table 2). Receptivity was higher on younger plants (mean 8.38 and 7.60 lesions/cm²) than on older plants (mean 5.98 lesions/cm²) (Table 3). Lesions were larger on younger plants (mean 1.54 and 1.45 mm diameter) than on older plants (mean 1.25 mm diameter) (Table 4). Younger plants had a higher percentage of leaf area damaged (mean 44.82 and 36.91%) than older plants (mean 23.42%) when estimated at 30 DAI (Table 6). In general, older plants had longer incubation periods, lower receptivities, smaller lesion diameters and lower percentage leaf area damage than younger plants. These

Table 5. Percentage leaf area damaged by *Didymella arachidicola* on nine peanut genotypes 15 days after inoculation.

Genotype	Experiment			Genotype mean
	1	2	3	
Tannut 74	72.6 a ¹	66.6 a		69.60 a
Egret	39.5 b	40.4 b		40.00 b
38/7/20	38.0 b	32.6 c		35.30 c
P 84/5/112	23.0 c	17.0 d		20.00 d
Florunner	11.8 d	3.6 f		7.70 ef
P 84/5/256	11.8 d	8.6 e		10.20 e
C 347/5/6	11.6 d	7.6 ef		9.60 e
C 346/5/8	13.4 d	8.6 e		11.00 e
P 105/3/7	4.0 e	3.4 f		3.70 f
Experiment mean	25.09	20.93		
SE	±3.13	±3.09		±3.09
CV(%)	10.16	10.29		13.83

¹ Means followed by the same letter in a column do not differ significantly at P=0.05 according to Duncan's multiple range test.

Table 6. Percentage leaf area damaged by *Didymella arachidicola* on nine peanut genotypes 30 days after inoculation.

Genotype	Experiment			Genotype mean
	1	2	3	
Tannut 74	94.6 a ¹	89.6 a	67.2 a	83.80 a
Egret	75.0 b	64.4 b	39.0 b	59.80 b
38/7/20	75.0 b	58.6 b	39.0 b	57.53 b
P 84/5/112	64.6 c	49.0 c	39.6 b	51.07 c
Florunner	20.8 de	14.0 de	3.2 cd	12.67 e
P 84/5/256	25.6 d	17.8 d	8.2 c	17.20 d
C 347/5/6	11.8 e	9.6 e	2.0 e	7.80 f
C 346/5/8	19.6 de	15.4 de	6.2 cd	13.73 de
P 105/3/7	16.4 de	12.8 de	6.4 cd	11.87 e
Experiment mean	44.82	36.91	23.42	
SE	±4.64	±4.24	±3.36	±4.00
CV(%)	13.75	14.77	10.56	7.76

¹ Means followed by the same letter in a column do not differ significantly at P=0.05 according to Duncan's multiple range test.

Table 7. Correlation coefficients¹ between incubation period, receptivity, lesion diameter and leaf area damage of *Didymella arachidicola* on peanut.

Variable	Variable						
	1	2	3	4	5	6	7
1. Incubation period		-0.92	-0.73	-0.84	-0.90	-0.84	-0.87
2. Receptivity			0.72	0.77	0.86	0.76	0.84
3. Lesion diameter				0.83	0.88	0.84	0.89
4. Leaf area damage (%), 15 DAI ²					0.87	0.99	0.87
5. Leaf area damage (%), 30 DAI						0.88	0.99
6. Leaf area damage (%), 15 DAI, T ³							0.87
7. Leaf area damage (%), 30 DAI, T							

¹ Spearman correlation coefficients based on 25 observations. All are significant at P=0.01.

² Days after inoculation.

³ After arcsine transformation.

differences were consistent across all test genotypes, irrespective of their field reactions to *D. arachidicola*.

Correlation coefficients for variables of resistance

were highly significant ($p=0.01$). Receptivity, lesion diameter, and percentage leaf area damage correlated positively with one another, and negatively correlated with incubation period (Table 7).

Discussion

Genotypes resistant to *D. arachidicola* in field screening trials in the USA and Zimbabwe were found to have a longer incubation period, reduced receptivity, smaller lesion diameters, and lower percentage leaf area damaged than susceptible genotypes. Resistance to *D. arachidicola* in peanut genotypes appears to be due to fewer successful infections from pycnidiospores. Even if the fungus successfully enters leaf tissues, development is slowed as indicated by an increased incubation period and reduced lesion diameter. The overall effect of this process is that on resistant genotypes, the infected leaflets showed only limited necrosis and defoliation. It is expected that the resistant genotypes would incur less yield loss. The effect of these individual variables on an epidemic progress in the field is difficult to interpret because these variables interact with one another and their effects are cumulative over the course of the epidemic (19,20).

Although, the genotypes Tamnut 74, Egret, 38/7/20, and P 84/5/112 were scored as susceptible at maturity in field screening trials in the USA and Zimbabwe, there were considerable differences in incubation period, receptivity, lesion diameter and leaf area damage in these genotypes as measured in the glasshouse. Tamnut 74 had the shortest incubation period, highest receptivity, largest lesion diameter, and greatest leaf area damaged, while the other three genotypes, although susceptible at maturity in field screening trials, had longer incubation periods, and lower receptivities and percentage leaf area damage. Lesions were also smaller on these genotypes than on Tamnut 74. This kind of reaction to disease is similar to the "partial resistance" reported by several investigators in other host-pathogen systems (2,6-10,14,15,19-21). Although, the genotypes Egret, 38/7/20 and P 85/5/112 showed severe damage from web blotch at maturity in field screening trials, it is suspected that they may have lower apparent infection rates (r) than other susceptible genotypes. The area under disease progress curve (AUDPC) may also be low in these genotypes because of longer incubation period, and reduced receptivity and lesion diameter.

None of the genotypes included in this study showed fructifications of the pathogen on infected leaflets. This is in agreement with observations made by other workers with other peanut genotypes (16, E.S. Luttrell, personal communication). However, production of pycnidia and pseudothecia was abundant on decomposing infected fallen leaflets lying on the soil surface in the plastic pots. This indicates that under field conditions the decomposing infected leaflets are sources of inoculum for fresh infections. Under cool, moist conditions, spore production is continued as freshly fallen leaves are added to leaf litter on the soil surface, further increasing the inoculum potential. The percentage defoliation is less in resistant genotypes than in susceptible ones. This may have some practical implication in reducing the in-

oculum load when a resistant genotype is grown year after year. Field studies will be required to verify this hypothesis. No information is available on genotype differences in production of pycnidia and pseudothecia on decomposing infected leaflets.

The growth stage of the host influenced disease development in the glasshouse. Older plants had longer incubation period, reduced receptivity, lesion diameter, and leaf area damage than younger plants in all test genotypes, irrespective of their field reactions to the disease. These results clearly indicate that plant age is an important factor in evaluating peanut germplasm for resistance to web blotch.

The incubation period, receptivity, lesion diameter and leaf area damage of *D. arachidicola* on peanut measured in this investigation are highly correlated with one another, as was shown in case of wild *Arachis* species (23). These observations suggest a linkage or possible pleiotropic effects of genetic factors controlling components of resistance as observed in other host-pathogen interactions (13).

The present investigation shows that screening of germplasm for resistance to *D. arachidicola* can be accomplished by measuring the incubation period, receptivity, lesion diameter and leaf area damage in glasshouse-grown plants, especially in areas where web blotch epidemics do not occur regularly or where the presence of other foliar diseases complicate screening in the field. Analysis of these variables is also useful for screening for genotypes that are likely to possess rate-reducing resistance, which is difficult to measure in the field because of interplot interference.

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Literature Cited

1. Alcorn, J. L., E. Punithalingam, and J. J. P. McCarthy. 1976. Peanut net blotch caused by *Didymosphaeria arachidicola* (Chochryukov). Comb. Nov. Trans. Br. Mycol. Soc. 66:351-355.
2. Berger, R. D. 1977. Application of epidemiological principles to achieve plant disease control. Ann. Rev. Phytopathol. 15:165-183.
3. Blaney, F. P. C., J. Chapman, and B. W. Young. 1977. Epiphytology of Phoma Web blotch and Cercospora leafspot in spanish groundnuts. Phytophylactica 9:63-64.
4. Cole, D. L. 1981. Diseases of groundnut (*Arachis hypogaea* L). 1. Fungicide spray effects on *Cercospora arachidicola* and *Phoma arachidicola* leaf infection, kernel yield and pod rots. Zimbabwe J. Agric. Res. 19:101-110.
5. Hildebrand, G. L. 1980. Groundnut production, utilization, research problems and further research needs in Zimbabwe, pp.290-296 in Proceedings of the International Workshop on Groundnuts, 13-17 October, 1980, ICRISAT (International Crops Research Institute for the Semi-Arid Tropics), Patancheru, A. P., India.
6. Hooker, A. L. 1967. The genetics and expression of resistance in plants to rusts of the genus *Puccinia*. Ann. Rev. Phytopathol. 5:183-182.
7. Jeger, M. J. 1980. Multivariate models of the components of partial resistance. Prot. Ecol. 2:265-269.

8. Jeger, M. J., D. G. Jones, and E. Griffiths. 1983. Components of partial resistance of wheat seedlings to *Septoria nodorum*. *Euphytica* 32:575-584.
9. Johnson, D. A., and R. D. Wilcoxson. 1978. Components of slow rusting in barley infected with *Puccinia hordei*. *Phytopathology* 68:1470-1474.
10. Mackenzie, D. R. 1976. Application of two epidemiological models for the identification of slow stem rusting in wheat. *Phytopathology* 66:55-59.
11. Marasas, W. F. O., G. D. Pauer, and C. H. Boerema. 1974. A serious leaf blotch disease of groundnuts (*Arachis hypogaea* L.) in Southern Africa caused by *Phoma arachidicola* Sp. - Nov. *Phytophylactica* 6:195-202.
12. Moraes, S. A., I. J. Godoy, and M. A. M. Gerin. 1983. Avaliacao da resistencia de *Arachis hypogaea* A. *Puccinia arachidis*, *Sphaceloma arachidis* E *Phoma arachidicola*. *Fitopatologia Brasileira* 3:499-506 (in Portuguese, with English summary).
13. Ohm, H. W., and G. Shaner. 1976. Three components of slow leaf rusting at different growth stages in wheat. *Phytopathology* 66:1356-1360.
14. Parlevliet, J. E., and J. C. Zadoks. 1977. The integrated concept of disease resistance. A new view including horizontal and vertical resistance in plants. *Euphytica* 26:5-21.
15. Parlevliet, J. E. 1979. Components of resistance that reduce the rate of epidemic development. *Ann. Rev. Phytopathol.* 17:203-222.
16. Philley, G. L. 1975. Peanut web-blotch: Growth, pathogenesis, and hosts of the causal agent, *Mycosphaerella argentinensis*. Ph.D. Thesis, Texas A & M University, College Station, TX.
17. Phipps, P. M. 1985. Web blotch of peanut in Virginia. *Plant Dis.* 69:1097-1099.
18. Porter, D. M., D. H. Smith, and R. Rodriguez-Kabana. 1982. Peanut plant diseases. pp. 326-410 in H. E. Pattee and C. T. Young (eds.), *Peanut Science and Technology*. Amer. Peanut Res. and Educ. Soc., Yoakum, TX 77995.
19. Shaner, G., and F. D. Hess. 1978. Equations for integrating components of slow leaf-rusting in wheat. *Phytopathology* 68:1464-1469.
20. Shaner, G., H. W. Ohm, and R. E. Finney, 1978. Response of susceptible and slow leaf-rusting wheats to infection by *Puccinia recondita*. *Phytopathology* 68:471-475.
21. Shaner, G., and R. E. Finney. 1980. New sources of slow leaf rusting resistance in wheat. *Phytopathology* 70:1183-1186.
22. Smith, O. D., D. H. Smith, and C. E. Simpson. 1979. Web blotch resistance in *Arachis hypogaea*. *Peanut Sci.* 6:99-101.
23. Subrahmanyam, P., D. H. Smith, and C. E. Simpson. 1985. Resistance to *Didymella arachidicola* in wild *Arachis* species. *Oleagineux* 40:553-556.
24. Taber, R. A. 1984. Web blotch. pp. 9-10 in D. M. Porter, D. H. Smith and R. Rodriguez-Kabana (eds.), *Compendium of Peanut Diseases*. Amer. Phytopath. Soc., Minn., USA.
25. Young, B. W., F. P. C. Blamey, and J. Chapman. 1980. Studies on the occurrence, epidemiology and control of leaf and stem diseases of groundnut. Technical communication No. 166, Dept. of Agric. Technical Services, Republic of South Africa.

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5