CSIRO Publishing

Australian Journal of Agricultural Research

Volume 53, 2002

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A journal for the publication of original contributions towards the understanding of an agricultural system

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Aust. J. Agric. Res., 2002, **53**, 1105–1110

Peanut resistance to *Sclerotinia minor* and *S. sclerotiorum*

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Abstract. The fungi *Sclerotinia minor* and *S. sclerotiorum* are the causal agents of two similar diseases of peanut (*Arachis hypogaea* L.). Both diseases cause significant losses in the Australian peanut industry. Development of cultivars with resistance to *Sclerotinia* will be an important component of integrated control. The aims of this project are to generate information that will assist in breeding for *Sclerotinia* resistance in peanut: to identify *Sclerotinia*-resistant peanut germplasm, to understand the inheritance and estimate heritability of resistance, and to test the effectiveness of identified sources of resistance against both *S. minor* and *S. sclerotiorum*.

This study has clearly established that material that shows resistance to *S. minor* in the USA is resistant to *S. minor* and likely to be resistant to *S. sclerotiorum* in Australia. The high level of resistance to both *S. minor* and *S. sclerotiorum* in germplasm from Texas, particularly TxAG-4, was confirmed. VA 93B showed good resistance in the field, which is primarily due to the open bush type rather than physiological resistance. Physiological resistance to *S. minor* was also identified in a cultivar and a landrace from Indonesia and a rust-resistant line from Queensland. All germplasm found to have high physiological resistance to *S. minor* belonged to the Spanish type.

Inheritance of physiological resistance to *S. minor* was studied using a Generation Means Analysis (GMA) of the cross TxAG-4/VA 93B and its reciprocal. The broad-sense heritability of physiological resistance on a single plant basis was estimated at 47%, much higher than earlier estimates obtained in field studies. The average gene action of *Sclerotinia* resistance genes from TxAG-4 was found to be additive. No dominance effects were detected in the GMA. A small but significant reciprocal effect between TxAG-4 and VA 93B indicated that VA 93B passed on some physiological resistance maternally.

An experiment was conducted to confirm the value of resistance against both *S. minor* and *S. sclerotiorum*. TxAG-4 was found to have physiological resistance to both *S. minor* and *S. sclerotiorum*. This resistance was expressed against both *Sclerotinia* species by progeny that were selected for resistance to *S. minor*.

On the basis of the information obtained, the comparative advantages of 3 strategies for *Sclerotinia*-resistant cultivar development are discussed: (*1*) introduction of germplasm; (*2*) recurrent backcrossing with screening and crossing in the BC_nF_1 generation; and (*3*) pedigree selection. At present, introduction and backcrossing are recommended as the preferred strategies.

Introduction

Peanut (*Arachis hypogaea* L.) is an important crop of the tropics and subtropics with a total world production of approximately 23 million t (Carley and Fletcher 1995). In Australia, 32 000–47 000 tonnes of peanuts are grown per annum (ABS 2001). The crop is grown in irrigated production systems in eastern Australia from Cooktown in northern Queensland (15° 30′ S, 145° 20′ E) to Wee Waa in New South Wales (30° S, 149° E), and as a rainfed crop in the Burnett region, particularly around Kingaroy (26° 30′ S, 151° 50′ E).

The greatest single determinant of peanut yields in Australia is available soil moisture (Crosthwaite 1994), but fungal diseases cause losses each year and for individual producers the damage caused by disease can be devastating. Leaf diseases are generally adequately controlled by application of fungicides. Important soil-borne fungal pathogens include *Sclerotinia minor* Jagger and *S. sclerotiorum* (Lib) de Bary, *Cylindocladium parasiticum* [Loos] Bell & Sobers, *Sclerotium rolfsii* Sacc., and *Lasiodiplodia theobromae* [Pat.] Griff. & Maubl. Compared with the foliar diseases, these soil-borne diseases are less predictable in their occurrence and harder to control (Ryley *et al.* 1997).

The two species of *Sclerotinia* cause similar diseases of peanut. Both diseases are called Sclerotinia blight and cause significant losses in the Australian peanut industry. In the

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USA, *Sclerotinia minor* blight of peanut is favoured by humidity greater than 95% (Dow *et al.* 1988*a*; Melouk and Backman 1995) and cool temperatures variously described as 18–20°C (Melouk and Backman 1995) or less than 21°C (Porter 1980). The disease is initiated by infection of the lower canopy, particularly the lateral branches and occasionally the hypocotyl (Melouk and Backman 1995). In the Burnett region the initial infection by *S. minor* is either via pegs or via branches (A. N. Kyei, pers. comm. 1998). The first field symptoms are sporadic wilted branches, visible from above the canopy. On parting the canopy, white fluffy mycelium is visible on the infected stems in the lower canopy. Because the pathogen requires a sustained high humidity for infection it 'shows up first in wetter parts of the field where the bush is larger or where it may be in the shade for longer' (Crosthwaite 1994). The disease has been shown to be exacerbated by sprinkler irrigation (Porter *et al.* 1987), injury to the bush (Porter and Powell 1978), and larger canopies (Dow *et al.* 1988*b*; Phipps 1995). It has been suppressed by dinitrophenol herbicides (Porter and Rud 1980) and foliar applications of zinc or copper fertiliser (Hallock and Porter 1981).

Chemical and cultural control methods will not provide complete control (Ryley *et al.* 2000). Development of cultivars with resistance to Sclerotinia will be an important component of integrated control (Melouk and Backman 1995). There have been several *S. minor*-resistant cultivars and germplasm lines released in the USA (Coffelt *et al.* 1982, 1987, 1994; Smith *et al.* 1990, 1991). The capacity to breed and select for such resistance in Australia must be established before committing to cultivar development (Cruickshank 2001).

Hunter *et al.* (1981) developed a method for screening bean (*Phaseolus vulgaris* L.) for 'partial resistance' to *S. sclerotiorum*. Pieces of celery were colonised by the fungus and applied to the stems of plants for a limited period of time. The technique was modified with green bean pods being used instead of celery for screening navy beans for resistance to *S. sclerotiorum* (Middleton and Redden 1990). This technique was in turn modified by JR Tatnell and K Middleton (unpublished data) for experiments on the peanut–*Sclerotinia minor* interaction.

The aims of this project were: (*i*) to confirm the resistance of reported *S. minor*-resistant germplasm and identify new sources of resistance; (*ii*) to understand the inheritance and estimate heritability of resistance to *S. minor*; and (*iii*) to test identified sources of resistance against both *S. minor* and *S. sclerotiorum*.

Materials and methods

Identifying germplasm

Twenty-five germplasm lines, 3 of which have reported resistance to *S. minor* (Table 1), were compared in both field and glasshouse experiments. The field experiment was a 5 × 5 row lattice with

Table 1. Description of 25 lines tested in both field and glasshouse for *Sclerotinia minor* resistance

Genotype	Bush type	Features	Botanical variety (pedigree)
CBR-R2	Prostrate	High CBR resistance	Virginia
NC18229A×NC2-6	Open bunch	CBR resistance	Virginia (NC18229A/NC2)
Chiba Handachi	Erect bunch	Desired quality	Virginia
Tifton-8	Prostrate	High transpiration efficiency	Virginia
Roberts	Erect bunch	High yield, CBR resistant	Virginia (NC 17921*3/NC 18229)
Streeton	Erect bunch	High yield, drought resistant	Virginia (NC 343/Early Bunch)
Southern Runner	Prostrate	High yield, leafspot resistant	Virginia (PI203396/Florunner)
PI362130	Spreading bunch	Narrow leaflets	Virginia
B57-p5-1	Erect bunch	Rust and leafspot resistance	Virginia (Tifrust-1/VA 732818)
VA 93B	Open bunch	*S. minor*-resistant in USA	Virginia (Virg. 81 B./VA 780839)
VA 81B	Open bunch	*S. minor*-resistant in USA	Virginia (F392-8/GA119-20)
CBR-R4	Erect bunch	High CBR resistance	Valencia
Tifrust-1	Open bunch	Rust and leafspot resistance	Valencia
ICGV 86590	Erect bunch	Rust and leafspot resistance	Spanish (X14-4-B-19-B/PI259747)
TxAG-4	Erect bunch	*S. minor*-resistant in USA	Spanish (Toalson/UF 73-4022)
ICGV 86031	Erect bunch	High transpiration efficiency	Spanish (F334A-B-14/NC 2214)
ICGV 87160	Erect bunch	Some rust and leafspot resistance	Spanish (Ah 65/NC17090)
B55-p29 L11	Erect bunch	Rust and leafspot resistance	Spanish (A116L14/Q22298)
Tapir	Erect bunch	High transpiration efficiency	Spanish ('US26'/Kidang)
Q22298	Erect bunch	High harvest index	Spanish
CBR-R3	Erect bunch	High CBR resistance	Spanish
A116 L4	Erect bunch	Rust and leafspot resistance	Spanish
A116 L14	Erect bunch	Rust and leafspot resistance	Spanish
Mani Pintar	Spreading bunch	Well known landrace	Not defined
CS-22	Erect bunch	High rust and leafspot resistance	(Valencia/*A. cardenasii*)

4 replicates, in an established *S. minor*-infested block at Kingaroy. The infestation was established by artificial inoculation of peanut crops in the 2 preceding summers with approximately 720 *S. minor* sclerotia, mixed with sand, per metre of row (Ryley *et al.* 2000). Given the levels of disease achieved in the 2 previous seasons, further inoculation was assumed to be unnecessary. Plots were 1 row 0.9 m apart and 5.4 m long with 40 seeds sown per plot. The measure of resistance was a foci count (FC), the number of 0.3-m sections of row containing plants with *S. minor* infection.

In the glasshouse test, one plant per 15-cm pot was grown until at least 30 cm tall. Inoculum was prepared in the following manner. Fresh pods of bean (*P. vulgaris* L.) were cut into 1-cm lengths; these segments were cut up one side (between the carpels) and any ovule/seed tissue was removed. The segments were sterilised in an autoclave at 121°C and 103 kPa for 15 min, and the sterile bean pieces were placed (cut surface down) on the perimeter of colonies of *S. minor* actively growing on potato dextrose agar, before colonies reached the edge of the Petri dish. A circle of 20–24 bean segments fitted around the Petri dish. The fungus then grew through the bean segment. At 20 ± 2 h after inoculation the segments were cut from the agar for use as inoculum.

Bean segments were wrapped around peanut stems so the stem filled the lumen between the carpels at 78 days after planting (DAP). The bean pieces were applied to the 2 lowest primary branches and the main stem. The inoculated plants were kept in a high humidity environment in a plastic 'tent' for 7 days. Misters operated for 5 min every hour to maintain a saturated atmosphere inside the tent and continuous moisture on the stems. Temperatures were monitored irregularly and only occasionally rose above 25°C in the early afternoon. At 85 DAP the misters were turned off and the plastic curtains raised to allow plants to dry and stop lesion expansion. At 87 DAP, lesion lengths were measured (Fig. 1). There were 16 replicates of each line. The average of all lesions >20 mm was analysed as moderated lesion length (MLL). The number of lesions <20 mm per plant (0–3) was analysed as small lesion count (SLC). Lesions below 20 mm are assumed to be failed or late infections as distinct from slower growing lesions described by lower MLL.

Inheritance and heritability

A generation means analysis (GMA) was conducted using a glasshouse experiment similar to that for the germplasm lines, except the experiment was conducted in 2 runs in a controlled environment cabinet (20–22°C, 12 h light/12 h dark). The GMA included parents and F_1 and F_2 progeny (with reciprocals) of the cross TxAG-4/VA 93B. Average genetic effects were estimated by the joint scaling test (Cavalli 1952) using the matrix algebra of Rowe and Alexander (1980). There were few lesions smaller than 20 cm (i.e. low SLC) and they occurred independently of generations, so only MLL was considered in the GMA. Broad-sense heritability (H) was calculated by the difference method (Nyquist 1991), pooling parent and F_1 variances to estimate environmental variance.

Resistance to S. sclerotiorum

Twelve genotypes (TxAG-4, VA 93B, PI362130, and 9 $F_{4:6}$ lines selected for *S. minor* resistance from the TxAG-4/VA 93B cross) were inoculated in factorial combination with 2 isolates of *S. sclerotiorum*. The 2 isolates of *S. sclerotiorum* used in this experiment were: M9347 collected from watermelon (*Citrullus lanatus* (Thunb.) Matsum. & Nakai.) on the Atherton Tableland in 1997, and UQ898 collected from peanuts at Corndale near Kingaroy in 1994. Colonised bean pieces were attached to the terminal end of 20-cm detached stem segments with all leaves removed and incubated in the controlled environment cabinet (as above) for 7 days. Lesions were measured in mm. No lesions below 20 mm occurred. The $F_{4:6}$ lines were analysed as a group and as individuals.

Results

Identifying germplasm resistant to S. minor

There were highly significant differences among lines for all 3 measures of resistance to *S. minor*: FC, MLL, and SLC (Table 2). The lines with the lowest FC (TxAG-4 and Tapir) also showed the greatest resistance by other measures. Only one of the lines grown commercially in Australia, VA 93B, showed any resistance. It was intermediate in FC. The line PI362130 had the highest values for FC and MLL. The only significant correlation (r = 0.41*) among resistance measures was between FC and MLL (Fig. 1).

Inheritance and heritability of resistance to S. minor

All the descendent generations had MLL values intermediate between the parents (Table 3), which were significantly

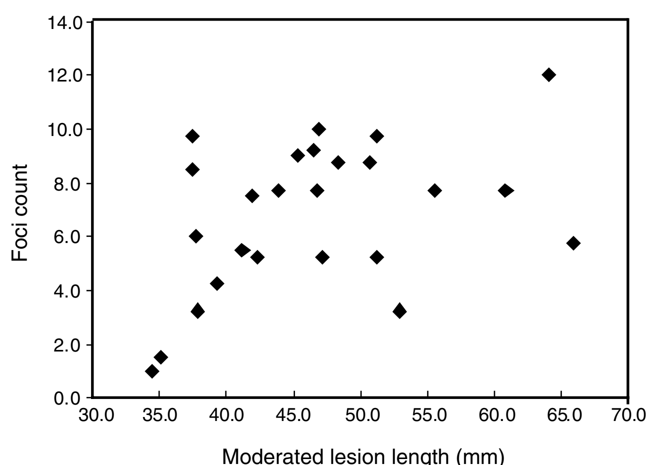

Fig. 1. Moderated lesion length (mm) *v.* foci count (disease foci per 5.4-m plot) for 25 peanut lines.

Table 2. Measures of *S. minor* resistance of a subset of 7 contrasting lines in the field and glasshouse experiments

	Field Foci count[A]	Glasshouse MLL[B]	Glasshouse SLC[C]
Tapir	1.0	34.4	1.69
TxAG-4	1.5	35.1	1.31
Q22298	3.3	37.8	0.75
B55-p29 L11	4.3	39.3	1.44
VA 93B	5.3	47.2	1.00
Streeton	10.0	46.9	0.50
PI362130	12.0	64.1	0.94
l.s.d. (P = 0.01)	3.0	14.5	0.79

[A]Foci count: 0.3-m segments of row containing visible *Sclerotinia* disease, per 5-m plot.
[B]Moderated lesion length: average lesion length (mm) ignoring lesions ≤20 mm.
[C]Small lesion count: the number of lesions ≤20 mm per pot (possible range 0–3).

Table 3. Predicted *S. minor* MLL means (mm) of generations from REML analysis

Generation	Predicted means
TxAG-4	51.7
VA 93B/TxAG-4 F_1	59.2
VA 93B/TxAG-4 F_2	62.7
TxAG-4/VA 93B F_2	67.7
TxAG-4/VA 93B F_1	68.1
VA 93B	73.0
l.s.d. (P = 0.01)	10.2

different from one another ($P \leq 0.01$). The reciprocal F_1s were significantly different from one another ($P \leq 0.05$) and the F_2s were intermediate between the F_1s. These means were the base data for the GMA.

The greatest genetic effect is a net additive effect (a) contributed by TxAG-4, which reduces MLL by 15 mm (Table 4). The 4.6 mm significant reciprocal effect (r) is a positive effect on MLL. It suggests that the maternal or cytoplasmic effect of TxAG-4 is the opposite of the nuclear genetic effect. The net dominance effect (d) is negligible and not significant ($P > 0.05$) regardless of whether the (r) is included in the model.

The pooled phenotypic variance of MLL for the 2 F_2s was significantly greater ($P \leq 0.01$) than the estimate of environmental variance from parents and F_1s. The H of MLL for F_2 plants is estimated as 47%.

Resistance to S. sclerotiorum

Lesion length differed significantly ($P \leq 0.01$) among peanut groups and lines within groups (Table 5). Groups did not interact significantly with fungal isolate but lines within groups did ($P \leq 0.05$).

The mean lesion length of the $F_{4:6}$ selections group was significantly ($P \leq 0.01$) less than VA 93B and PI362130 but not significantly greater than TxAG-4 (Table 6). Three of the 9 lines interacted significantly ($P \leq 0.05$) with fungal isolate. The mean lesion length of each of these lines was significantly lower than VA 93B. Fig. 2 shows lesion lengths in response to the 2 isolates of *S. sclerotiorum*.

Table 4. Parameter estimates, standard errors, and χ^2 tests for GMA of *S. minor* MLL

Genetic effect (mm)	Estimate	s.e.
Mid-point (m)	62.9	1.57
Net additive (a)	–15.1	2.19
Net dominance (d)	0.1	2.46
Reciprocal (r)	4.6	1.44
Calculated χ^2	2.3	
Tabulated χ^2 ($P \leq 0.05$)	5.99	

Table 5. Analysis of variance for *S. sclerotiorum* lesion length (mm)

Source of variance	d.f.	MS	F value
Replicates	9	2937	11.1**
Isolates (*Sclerotinia*)	1	1270	4.8
Residual (main-plots)	9	266	
Peanut groups	3	5452	14.5**
Peanut lines	8	2788	7.4**
Isolates × groups	3	55	0.2
Isolates × peanut lines	8	875	2.3*
Residual (subplots)	198	376	
Total	239		

*$P < 0.05$; ** $P < 0.01$.

Table 6. Mean *S. sclerotiorum* lesion length (mm) of peanut groups

Peanut group/line	Group mean lesion length
Group 1: TxAG-4	99
Group 2: VA 93B	125
Group 3: PI362130	124
Group 4: $F_{4:6}$ selections	103
l.s.d. (P = 0.05)	9–12
l.s.d. (P = 0.01)	12–16

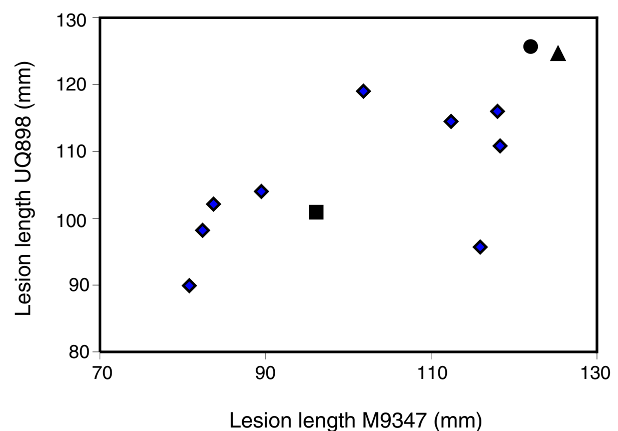

Fig. 2. Comparison of mean lesion length for 12 peanut lines inoculated with two isolates of *Sclerotinia sclerotiorum*. (▲ VA 93B, ■ TxAG-4, ● PI362130, ◆ $F_{4:6}$ lines.)

Discussion

These experiments have established that there is potential to breed for Sclerotinia resistance in Australia. There is useful genetic variation for resistance to both *S. minor* and *S. sclerotiorum*. The inheritance and broad-sense heritability of resistance in one cross have been elucidated. Material selected for resistance to *S. minor* expressed resistance to 2 isolates of *S. sclerotiorum*.

This study has clearly established that TxAG-4 and VA 93B, which have reported resistance to *S. minor* in the USA (Smith *et al.* 1990; Coffelt *et al.* 1994), are resistant to *S. minor* and likely to be resistant to *S. sclerotiorum* in Australia. The high level of physiological resistance (measured by MLL) to both *S. minor* and *S. sclerotiorum* in TxAG-4 was confirmed. VA93 B showed good resistance in the field, which is primarily due to the open bush type rather than physiological resistance. Physiological resistance to *S. minor* was also identified in a cultivar (Tapir) and a landrace (Q22298) from Indonesia, and a rust-resistant breeding line from Queensland (B55-p29 L11). All germplasm found to have high physiological resistance to *S. minor* belonged to the Spanish type (ssp. *fastigiata*). This means that significant recombination is required to introgress the resistance into Virginia and Runner types (ssp. *hypogaea*).

The occurrence of the small or failed lesions quantified by SLC may indicate that 2 components of physiological resistance exist among the lines included in the germplasm experiment: resistance to infection (SLC) and a rate-limiting resistance to post-infection lesion growth (MLL); or SLC may be describing an artefact of the experimental technique, in that some lines may not be so easily infected by the bean piece inoculation. In the latter case, SLC may not contribute to resistance in the field, a conclusion that may be supported by the correlation of FC with MLL but not with SLC. TxAG-4 and Tapir, the lines with greatest resistance according to FC and MLL, also have higher SLC values. TxAG-4 and VA 93B did not differ significantly for SLC, which may explain why the occurrence of small lesions has occurred independently of peanut genotype in other experiments.

The strong additive average genetic effects indicate that early generation selection for MLL may be possible. However, it is unlikely that selection on a single F_2 or F_3 plant basis would be effective, since the heritability of MLL on a single plant basis was low and poor seed production by infected plants will cause loss of the potential variation (genetic drift) within progeny of selected F_2 or F_3 plants. The estimate of H for MLL at 47% compares favourably with the estimates of H for Sclerotinia resistance of 14% and 23% reported by Wildman *et al.* (1992). Wildman *et al.* (1992) were working with different crosses and a different index of resistance measured in the field, so the difference between their results and those obtained in this study is not surprising. The lack of strong dominance effects suggests that the broad-sense heritability estimates approximate narrow-sense heritability. Further improvements in precision would be needed to substantially improve heritability.

Early generation selection for resistance to *S. minor* has achieved correlated genetic advance in physiological resistance to *S. sclerotiorum*. The apparent differential interaction of some lines with isolates of the fungus means that progenies or lines selected using *S. minor* may still require evaluation against more than one isolate of *S. sclerotiorum* to confirm and quantify resistance. However, the presence and magnitude of differential interaction need to be confirmed with further experimentation before committing resources to its management.

On the basis of the information obtained, 3 strategies for development of Sclerotinia-resistant cultivars are seen to have potential. First is the introduction of germplasm, because material reported as resistant in the USA has resistance in Australia and peanut breeding programs in the USA have similar quality objectives to the Australian program. It is highly likely that Sclerotinia-resistant varieties from the USA in the future would be suitable for the Australian industry. Early maturity, drought tolerance, and rust resistance are the desirable traits most likely to be lacking in introductions from the USA. These introductions would be most likely adapted to irrigated environments where foliar disease pressure is not great, e.g. inland southern Queensland.

Second is recurrent backcrossing with screening and crossing in the BC_nF_1 generation. Due to the additive effects of resistance genes, it is expected that the BC_nF_1s will only express half the resistance of the resistant parent. The one serious weakness of this scheme is that greater precision will be required to distinguish the BC_nF_1s carrying the resistance genes compared with distinguishing inbred genotypes expressing greater resistance. Such a backcrossing scheme would benefit from use of detached stems to provide replicated measurement of the resistance of individual plants. Further research is needed to establish that the required precision could be achieved.

Thirdly, a pedigree breeding and selection scheme would allow screening for resistance on a family basis. Heritability, and hence response to selection, would be higher than on a single-plant basis. A possible disadvantage of pedigree breeding is that simple bi-parental crossing will not generate enough genetic recombination between resistant types (ssp. *fastigiata*) and Virginia and Runner types (ssp. *hypogaea*). Pedigree selection among BC_1F_1-derived families may be a way to combine better recombination with efficient selection.

Acknowledgments

The assistance of Jeff Tatnell, Asirifi Kyei, and John Tonks was invaluable. This research was supported by the Queensland Department of Primary Industries and the Grains Research and Development Corporation.

References

ABS (Australian Bureau of Statistics) (2001) 'Australia now—a statistical profile.' (World Wide Web Site) http://www.statistics.gov.au

Carley DH, Fletcher SM (1995) An overview of world peanut markets. In 'Advances in peanut science'. (Eds HE Pattee, HT Stalker) pp. 554–577. (American Peanut Research and Education Society: Stillwater, OK)

Cavalli LL (1952) An analysis of linkage in quantitative inheritance. In 'Quantitative inheritance'. (Eds ECR Reeve, CH Waddington) pp. 135–144. (HMSO: London)

Coffelt TA, Porter DM, Mozingo RW (1982) Registration of Virginia 81 Bunch peanut. *Crop Science* **22**, 1085–1086.

Coffelt TA, Porter DM, Mozingo RW (1994) VA 93B Peanut. *Crop Science* **34**, 1126.

Coffelt TA, Porter DM, Smith JC, Mozingo RW (1987) Registration of six peanut germplasm lines with multiple resistance. *Crop Science* **27**, 1319.

Crosthwaite I (1994) 'Peanut growing in Australia.' (Queensland Department of Primary Industries: Brisbane, Qld)

Cruickshank AW (2001) Peanut (*Arachis hypogaea*) resistance to *Sclerotinia minor* and *S. sclerotiorum*. MAgrSc Thesis, University of Queensland, Australia.

Dow RL, Porter DM, Powell NL (1988*a*) Effect of environmental factors on *Sclerotinia minor* and *Sclerotinia* blight of peanut. *Phytopathology* **78**, 672–676.

Dow RL, Powell NL, Porter DM (1988*b*) Effects of modification of the plant canopy environment on *Sclerotinia* blight of peanut. *Peanut Science* **15**, 1–5.

Hallock DL, Porter DM (1981) Effects of applied plant nutrients on *Sclerotinia* blight incidence in peanuts. *Peanut Science* **8**, 48–52.

Hunter JE, Dickson MH, Cigna JA (1981) Limited-term inoculation: a method to screen bean plants for partial resistance to white mold. *Plant Disease* **65**, 414–417.

Melouk HA, Backman PA (1995) Management of soil-borne fungal pathogens. In 'Peanut health management'. (Eds HA Melouk, FM Shokes) (APS Press: Minnesota)

Middleton K, Redden R (1990) Selection of *Sclerotinia sclerotiorum* resistance from a *Phaseolus* spp. germplasm collection. *Annual Report Bean Improvement Cooperative* **33**, 189.

Nyquist WE (1991) Estimation of heritability and prediction of selection response in plant populations. *Critical Reviews in Plant Sciences* **10**, 235–322.

Phipps PM (1995) An assessment of environmental conditions preceding outbreaks of *Sclerotinia* blight of peanut in Virginia. *Peanut Science* **22**, 90–93.

Porter DM (1980) *Sclerotinia* blight of groundnut—a disease of major importance in the USA. In 'Proceedings of the International Workshop on Groundnuts, 13–17 October 1980'. (Eds RW Gibbons, JV Mertin) pp. 177–185. (ICRISAT: Patancheru, AP, India)

Porter DM, Powell NL (1978) Sclerotinia blight development in peanut vines injured by tractor tyres. *Peanut Science* **5**, 87–90.

Porter DM, Rud OE (1980) Suppression of Sclerotinia blight of peanuts with dinitrophenol herbicides. *Phytopathology* **70**, 720–722.

Porter DM, Wright FS, Powell NL (1987) Effects of sprinkler irrigation on peanut diseases in Virginia. *Plant Disease* **71**, 512–515.

Rowe KE, Alexander WL (1980) Computations for estimating the genetic parameters in joint-scaling tests. *Crop Science* **20**, 109–110.

Ryley M, Kyei A, Trevorrow P, Tatnell J, Garozzo S (1997) Integrated management of soilborne diseases. In 'The 2nd Australian Peanut Conference: Quality peanuts for profit'. (Eds B Fleming, A Cruickshank, S Cruickshank) pp. 88–91. (Queensland Department of Primary Industries: Brisbane, Qld)

Ryley MJ, Kyei NA, Tatnell JR (2000) Evaluation of fungicides for the management of Sclerotinia blight of peanut. *Australian Journal of Agricultural Research* **51**, 917–924.

Smith OD, Aguirre SM, Boswell TE, Grichar WJ, Melouk HA, Simpson CE (1990) Registration of TxAG-4 and TxAG-5 peanut germplasms. *Crop Science* **30**, 429.

Smith OD, Simpson CE, Grichar WJ, Melouk HA (1991) Tamspan 90 peanut. *Crop Science* **31**, 1711.

Wildman LG, Smith OD, Simpson CE, Taber RA (1992) Inheritance of resistance to *Sclerotinia minor* in selected peanut germplasm. *Peanut Science* **19**, 31–34.

Manuscript received 21 January 2002, accepted 31 May 2002