Sources and Nature of Resistance to Cercospora Arachidicola Hori and Cercosporidium Personatum (Beck & Curtis) Deighton in Arachis Species¹

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ABSTRACT

Various subspecies, botanical varieties and cultivars of peanuts (Arachis hypogaea L.) from widely separated areas of South America and Africa were evaluated for resistance to C. personatum and C. arachidicola. All cultivated peanuts tested were highly susceptible to both fungi except one collection from Peru, which showed a high degree of tolerance to defoliation.

Sources of resistance were found in the wild Arachis species. Several immune and many highly resistant collections of Arachis were found in the sections Erectoides, Rhizomatosae, and Extranervosae. In section Axonomorphae, A. chacoense (10602 GKP) was highly resistant to C. arachidicola but susceptible to C. personatum, and C. cardenasii (10017 GKP) was susceptible to C. arachidicola but immune to C. personatum. Both Arachis species are cross-compatible with A. hypogaea.

Host response in terms of pathogen penetration was classified into: immune, moderately and highly susceptible; and the reactions after penetration were: highly resistant, moderately and highly susceptible. On highly susceptible peanuts, the germtubes showed directed growth toward open stomata through which these fungi penetrate. On moderately susceptible peanuts a few germ-tubes grew toward the stomata, and on the immune entries no directed growth of the germ-tubes was observed. Resistance after penetration was associated with the formation of a barrier in advance of and around the infection site in the form of cell wall swelling and thickening, and the deposition of pectic substances on the cell walls and in intercellular spaces.

Woodroof (1933) concluded that Cercospora arachidicola Hori and Cercosporidium personatum (B. & C.) Deighton (Cercospora personata) are prevalent in all countries in which peanuts are grown commercially, and noted that C. arachidicola is probably more common than C. person-atum. All known varieties of the cultivated peanut are susceptible in varying degrees to the two leafspot diseases (Chevaugeon, 1952; Hemingway, 1957; Higgins, 1935; Reyes and Romasanta, 1940; Smartt, 1961; Jackson and Bell, 1969). Cooper and Gregory (1960) reported degrees of variation in leafspot susceptibility in X-ray-induced mutants of a progeny of Virginia Bunch peanuts (NC 4). More recently, Smith (1971) has reported the screening of 800 strains of A. hypogaea by inoculation with Cercospora arachidicola. None of these showed more than the usual variations in susceptibility previously encountered in cultivated peanuts by the workers cited above (personal communication).

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Although peanut leafspots are successfully controlled by fungicides in the United States, the cost is significant and may be higher, relative to other costs, in other peanut - producing areas. The development of host resistance is therefore a worthwhile objective. This paper presents sources of resistance found in some of the wild species relatives of the cultivated peanut, some characteristics of that resistance and the distribution of the resistance by taxonomic sections of the genus Arachis. Previous reports have already indicated resistance to leafspot diseases in wild Arachis species. Chevalier (1934) reported that six species, all native to Brazil, were susceptible to C. personatum. Frezzi (1960) reported that the wild species of Arachis from Corrientes and Misiones, Argentina, were attacked primarily by C. personatum. Gibbons and Bailey (1967) reported resistance to C. arachidicola in three wild species of Arachis.

Materials and Methods

Inoculum for pathogenicity studies was produced by the methods of Abdou and Cooper (1974). Stock cultures were maintained by transfer to fresh medium every 2 weeks and 10 days in the case of C. arachidicola and C. personatum, respectively. Inoculum was prepared from stock cultures by making a concentrated conidial suspension and transferring 0.5 ml of this suspension to a petri dish containing peanut leaf agar (WA + PLX) medium (Abdou and Cooper, 1974). Each petri dish was shaken by hand to spread the inoculum over the surface of the medium. After 7-10 days incubation at room temperature under continuous light, the sporulating culture was flooded with 30 ml distilled water and after 2 to 3 min the conidia were brushed free by means of a camel's hair brush. The resulting conidial suspension was then filtered through a layer of cheesecloth. Slants of WA + PLX medium were also used in preparing small quantities of inoculum.

Preliminary tests conducted with A. hypogaea indicated that inoculum of approximately 15,000 spores per ml was adequate to give lesions in quantities suitable for disease evaluation. The addition of three to four drops of Tween-80 (polyethylene oxide sorbitan monooleate; ICN Nutritional Biochemicals Corporation, Cleveland, Ohio 44128) per 100 ml inoculum gave uniform distribution over the entire plant.

Inoculum was applied to the peanut plants with an electric atomizer at 8-10 lb air pressure. Inoculated plants were immediately placed in clear polyethyleneenclosed chambers built under the greenhouse benches where the temperature fluctuated between 25-30 C. The chambers were equipped with a humidifier which was adjusted to atomize for 10-15 min every 4 hr, resulting in 100% relative humidity without excess free water on the leaves. After 4 days incubation the humidifier was readjusted to atomize for the same period only twice during the day and once at night. After additional days incubation the plants were returned to the greenhouse benches. Small necrotic spots usually appeared within 8-11 days after inoculation.

Spore germination and mycelial development on the leaf surface were studied by placing 1 cm² leaflet samples on a microscope slide, adding a drop of acid-fuchsin in lactophenol and covering with cover slip for microscopic examination. With a strong light source, the germinating conidia and mycelial growth appeared red on unstained green leaf tissues. In order to observe the mode of penetration of germ-tubes, leaflet tissue was killed and fixed in F.A.A. solution for 24 hr, cleared by placing in chloral hydrate solution at room temperature for 4-7 days, transferred to lactophenol for 24-48 hr, then stained with acid-fuchsin in lactophenol for 10-15 min or more for examination. This method was suitable for mature and old leaves, but young and succulent leaves were disintegrated by the chloral hydrate treatment. The use of 70 to 75% lactic acid as a clearing agent as reported by Debenham (1939) overcame this difficulty.

For histological studies infected leaflet samples from greenhouse-grown plants were killed and fixed in F.A.A. for 24-48 hr. Satisfactory results were also obtained with FBA (Johansen, 1940). Air was removed from the tissues by subjecting the vials containing the samples to a partial vacuum for 15 min. Prior to sectioning, all materials were dehydrated according to the tertiary butyl alcohol method (ibid) and embedded in Tissuemat or paraffin wax of 56 C melting point. Serial sections were cut 8 u thick. Of several staining schedules tried, safranin-fast green combination (ibid) was the most satisfactory for the study of host-parasite reactions, although crystal violet-erythrosin (ibid) also gave dependable results.

Specific stains were tested to determine the chemical nature of the materials deposited in the cell walls of resistant plants. Sudan IV was used for cutin and suberin, phloroglucinol in hydrochloric acid for lignin, and ruthenium red for pectic substances (ibid).

Disease ratings in the greenhouse were usually made 2-6 weeks from first symptoms. The apparently diseaseresistant survivors were re-inoculated and rated again. Disease severity ratings were based on number of lesions per leaflet, diameter of lesion, area infected [infected area/leaflet = no. lesions x (average diameter/lesion 12)² x 3.1416], sporulation index, and percentage defoliation (five leaves/plant on the main stem, the cotyledonary laterals, or main cutting axis). The evaluated leaves were below the third terminal leaf.

The host materials used consisted of 30 lines of A. hypogaea [5 from Peru (2 subspecies hypogaea and three sequential forms of unique and undescribed subspecific status restricted to the Peruvian region); 9 from Bolivia (6 subspecies hypogaea, 2 fastigiata, and 1 intermediate); 9 from the Guarani region, Brazil, Paraguay, Argentina (1 hypogaea, 8 fastigiata); 1 from Amazonas (hypogaea); 4 African (1 hypogaea and 3 fastigiata); NC 2 and NC 4] and wild species accessions as follows by sections of the genus Arachis: 9 Axonomorphae (2 annuae and 7 perennes); 1 Caulorhizae; 22 Erectoides (1 trifoliolatae, 21 tetrafoliolatae); 56 Rhizomatosae; and 6 Extranervosae. No Triseminalae or Pseudoaxonomorphae species were included in the inoculations.

A detailed listing of the host materials appears in the tables of Results. The collection numbers used there correspond to those in Gregory et al. (1973).

Results

HOST-PARASITE RELATIONSHIPS AND MECHANISM OF RESISTANCE TO INFECTION

Examination of leaflets from selected plants inoculated with *C. arachidicola* and *C. personatum*, showed that the conidia began to germinate within 6 to 12 hr after inoculation. After 48 hr, 95-99% of the conidia had germinated on all inoculated plants examined. Conidia usually germinated from the terminal cells, although other cells occasionally produced germ-tubes. The germ-tubes grew from 5 to 50 μ in *C. arachidicola* and from 7 to 35 μ in *C. personatum* within the first 6 days.

After 1 week of incubation germ-tubes began to show differential responses to specific plants. On plants which were classified as highly susceptible, the majority of the germ-tubes of both fungi began to show attraction toward the open stomata. Usually only one germ-tube, occasionally two to three, from each conidium showed this directional response. If a conidium was far from the stoma, the germ-tube occasionally branched and grew toward the opened stomatal pore. No competition between two germ-tubes was observed and only one was found to penetrate a stoma. Some of the germ-tubes also showed directional growth toward the epidermal radial cell walls. After the germ-tube of C. arachidicola reached a stomatal pore, the distal end enlarged and became filled with dense protoplasm; whereas, the proximal cells appeared to be partially evacuated. In C. personatum the distal end of the germ-tube enlarged to form an irregularly shaped "appressorium-like" structure over the stomatal pore or the epidermal radial cell walls. In stained preparations, a dark point marking the penetration peg could be distinguished in the center of the germ-tube swelling. The macroscopic symptoms of the disease in the form of minute necrotic spots occurred 48-72 hr after penetration. These necrotic spots included the guard cells and some of the surrounding cells. No penetrations were observed from the germ-tubes which were directed to the epidermal cell walls. These observations indicate that C. arachidicola and C. personatum normally penetrate their host only through open stomata.

On plants which were moderately susceptible, the behavior of the germinating conidia was similar to the previous group for the first 7 days of incubation. After that, however, only a few germtubes showed directional growth toward stomata. All infections observed were from these apparently attracted germ-tubes. The majority of the germ-tubes grew at random and did not penetrate. Occasionally a germ-tube grew beside or even over a stoma without entering. The conidia and their germ-tubes that failed to penetrate eventually lost their stainability and became vacuolated and transparent.

On immune plants, the germ-tubes of these fungi apparently were not attracted toward the stomata and no penetration or subsequent infection was observed. Eventually the conidia and their germ-tubes lost their stainability and became transparent.

HOST-PARASITE RELATIONSHIPS

AND MECHANISM OF RESISTANCE AFTER INFECTION

Highly Susceptible Group

Cross sections of very highly susceptible peanut leaves (i.e., A. hypogaea, P.I. 262074) shortly after infection showed that as soon as penetration and the formation of secondary hyphae were evident in the stomatal chamber, the adjacent host cells began to react. The guard cells, the adjacent epidermal cells and the region of the palisade-spongy mesophyll tissues below and surrounding the stomatal chamber stained deeply with safranin. The contents of these cells were granulated with numerous vacuoles and the nuclei were stained deeply and located against the cell walls. The cytoplasm coagulated and increased in density. The cell walls remained intact and evident. These changes in the host cells occurred after infection by either fungus.

With advancement of the disease, the fungal mycelium branched and continued proliferation in the leaf tissues causing the cells to collapse and produce the necrotic spot surrounded by lightly stained cells which make up the yellow halo so characteristic of these lesions. The advancing *C*. *arachidicola* mycelium was at first intercellular, but developed intracellularly after the host cells were killed. No evidence of cicatrice formation was observed surrounding the lesions on this group of *Arachis* species. It appeared that host cells were killed in advance of the growing mycelium of *C*. *arachidicola* as hyphae were not found in living cells but were frequent in colonized dead cells. Host cells were not killed in advance of *C*. *personatum* hyphae. Many preparations revealed the intercellular fungus with haustoria in apparently normal cells.

In both fungi, after the collapse of the host cells, the mycelium produced a stroma either subcuticularly or subepidermally, and most frequently in the stomatal chambers. The stroma developed on the lower or on both lower and upper surfaces of *C. personatum* lesions and largely on the upper surface in *C. arachidicola* lesions. About 2 weeks after infection under conditions of high relative humidity, the stroma produced fascicles of conidiophores which protruded from the stomata or the ruptured epidermis.

Moderately Susceptible Group

In the moderately susceptible Arachis species, the early stages of infection and disease development were similar to those in the highly susceptible group. Observations of advanced stages revealed that there was moderate to little mycelium colonizing the cells. The only completely destroyed cells were those in the center of the lesions. No definite stroma was formed and conidiophores developed through the stomata in only a few instances under favorable conditions. A heavily stained deposit in the intercellular spaces and cell walls in advance of infection at early stages was observed, but in advanced lesions no evidence of such deposits was present and all cells were invaded.

Highly Resistant Group

In highly resistant Arachis species, the reactions of the host and parasite were quite different from the previous groups. Sections of 10 - 20 - day - oldlesions showed that the fungus had stopped its proliferation and development was limited to only a few mycelial strands at the center of the lesion.

The infection appeared to be restricted and confined to the region between two vascular bundles in which cells of the bundle sheath extension reacted to form a barrier on both sides of the infection. Both epidermal and water storage cells also reacted to form this barrier at the upper and lower sides of the lesion as indicated by greatly thickened cell walls which were deeply stained with safranin. The intercellular spaces were also filled with a deeply stained amorphous material. The palisade cells did not share in the formation of this type of barrier, and the infected area was limited to the center of the infection site. No mycelium was detected outside this barrier and lesion development was completely stopped. An attempt to study the chemical nature of the material deposited in and between the cell walls of these barriers with differential stains indicated that it was a pectic substance giving a positive reaction with ruthenium red stain. A negative reaction to this stain was observed in the leafspot lesions on members of the moderately and highly susceptible *Arachis* species.

Sources of Resistance in the Axonomorphae

All strains of the cultivated peanut tested proved to be highly susceptible. Disease reaction of a sample of 30 strains (12 subspecies hypogaea, 16 subspecies fastigiata, and 2 intermediate varieties) is shown in Table 1.

Table 1. Reaction of different A. hypogaea strains to inoculation with Cercospora arachidicola and Cercosporidium personatum.

P.I.	Subspecies	Lesior leafl		Area in mm	fected [†]	Percentage defoliation*	
no.		arach.	pers.	arach.	pers.	arach.	pers
262130	hypogaea	14.6	13.1	183.5	163.4	100	90
268837	مرمن باللي طيانين	12.9	13.2	63.2	64.7	100	90
NC 4		14.0	13.3	98.9	93.9	100	95
274201		12.9	13.7	91.2	96.8	100	95
269092		13.3	13.8	64.7	67.6	100	95
274190		13.5	13.8	42.4	48.0	80	80
262123		13.7	13.9	67.2	64.7	100	100
262107		13.1	14.1	92.6	99.6	70	70
268839		13.9	14.5	68.1	71.1	100	95
262125		13.8	14.6	67.6	71.5	100	100
274180		14.5	14.8	102.5	104.6	100	100
261982		12.7	15.1	89.7	106.7	100	100
NC 2	Intermediate	13.5	13.0	95.4	91.9	100	95
262090		14.8	13.3	104.6	94.2	100	100
262075	fastigiata	12.5	13.4	91.5	94.7	80	90
262129		14.4	13.6	101.8	96.1	40	50
261906		15.3	13.6	108.2	96.2	100	100
261991		12.3	13.7	86.9	96.8	90	100
262012		13.4	13.9	94.7	98.3	100	98
262121		13.9	14.2	98.2	100.2	100	95
261945		13.2	14.2	93.3	100.4	90	100
268579		13.7	14.3	96.8	101.1	100	90
261942		15.3	14.7	108.2	103.9	100	95
262122		14.2	14.7	100.4	103.9	100	100
262092		14.7	14.8	103.9	104.6	100	100
268563		14.7	14.8	184.7	185.3	100	100
261936		13.0	15.1	91.9	106.7	100	100
262074		14.5	15.1	182.2	188.5	100	100
268696		13.1	15.1	64.2	37.9	100	90
261962		13.3	15.6	93.9	110.1	80	100

¹ These on diameters of 20 lesions in each of two replications. *Average of five leaves (20 leaflets) in each of two replications.

Table 2 shows the disease reaction of eight wild species relatives of cultivated peanuts in the section Axonomorphae. Except for the outstanding resistance of A. chacoense (10602 GKP) to C. arachidicola and the immunity of A. cardenasii (10017 GKP) to C. personatum, members of the section Axonomorphae were generally susceptible to both fungi.

The disease reactions of one member of the section Caulorhizae and 22 collections of the section Erectoides (1 trifoliolatae and 21 tetrafoliolatae), 56 collections of section Rhizomatosae, and 6 collections of the section Extranervosae are shown in Tables 3, 4 and 5, respectively. The one species of section Caulorhizae was moderately resistant to both fungi. The members of the section Erectoides were mostly susceptible. Only two accessions, 10573 (b) GKP and 10574 GKP, exhibited a consistent high resistance to both fungi. Accession 10543 GKP was moderately resistant to *C. personatum* and 9795 GKP moderately resistant to both fungi. Accessions of the section Rhizomatosae were mostly resistant to both fungi. Exceptional instances of susceptibility may be noted in Table 4. One instance (accession 10596 GKP) of immunity to both fungi was recorded. In the section Extranervosae a most interesting case of absolute immunity was observed in *Arachis villosulicarpa*, the only other species of *Arachis* known to cultivation. Unfortunately, present prospects for genetically combining this or any other species of the section with *A. hypogaea* are remote.

Table 2. Reaction of Arachis species of the section Axonomorphae to Cercospora arachidicola and Cercosporidium personatum.

Collection no.	Lesions per leaflet*		Diameter per lesion, mm		Percentage defoliation [‡]		Sporulation index	
(or name)	arach.	pers.	arach.	pers.	arach.	pers.	arach.	pers
. monticola	25	23	2.5	2.5	95	95	+++	+++
A. duranensis	8		0.6		90		+	
A. <u>villosa</u>	10	8	2.5	2.0	90	85	+++	+++
9548 GKP	15	15	1.0	0.8	90	90	+	+
9901 GKP	35	25	2.0	1.5	90	100	+	+
9926 GKP	8	7	1.0	0.9	90	85	+	+
10602 GKP	5	5	0.5	2.0	0	90	0	++
10017 GKP [¶]	5	0	1.0	0	100	0	+	0

*Average of five leaves (20 leaflets) in each of two replications.

⁺Average of 20 lesions in each of two replications.

*Average of 20 leaflets in each of two replications.

\$0 =.no sporulation; + = light; ++ = moderate; +++ = heavy.

^WA. <u>chacoense</u> and <u>A. cardenasii</u>, respectively.

Table 3. Reaction of Arachis species of the sections Caulorhizae and Erectoides to Cercospora arachidicola and Cercosporidium personatum.*

Collection no.	Lesions per leaflet		Diameter lesion.		Percer defoli		Sporulation index	
(or name)	arach.	pers.	arach.	pers.	arach.	pers.	arach.	pers
			Caulort	nizae				
10538 GKP	40	42	0.5	0,5	0	0	0	0
			Erectoi	ldes				
9837 GKP (tri- foliolatae)	2	5	1.5	1.5	90	100	++	++
9646 GKP (tet- rafoliolatae)		15	1.0	1.5	90	90	+	+
9764 GKP	10	12	1.5	1.0	100	100	+	+
9769 GKP	12	15	2.0	1.0	90	85	+	+
9788 GKP	5	5	1.5	1.5	100	100	+++	+
9795 GKP	12	10	0.6	0.5	0	0	+	0
9812 GKP	15	14	0.6	0.6	80	90	+	+
9820 GKP	25	28	1.0	0.8	95	90	+	+
9841 GKP	15	15	0.5	0.5	85	85	+	+
9990 GKP	5	8	0.6	0.6	100	90	+	+
9993 GKP	12	12	0.7	0.8	90	95	+	+
10002 GKP	10	12	1.0	0.5	90	85	+	+
10541 GKP	25	21	0.8	1.5	100	100	++	+
10543 GKP	25	25	2.0	0.5	90	0	+	0
10573(a) GKP	8	15	2.0	2.0	90	85	+	+
10573(b) GKP	8	10	0.5	0.5	0	0	0	0
10574 GKP	8	7	0.5	0.5	0	0	0	0
10580 GKP	15	14	0.8	0.7	100	90	+	+
10582 GKP	25	15	0.6	0.6	100	100	+	+
10585 GKP		31		0.6		85		+
10588 GKP	42	38	1.0	1.0	100	80	+	+

*See footnotes in Table 2.

Table 4. Reaction of Arachis species of the section Rhizomatosae to inoculation with Cercospora arachidicola and Cercosporidium personatum.*

					•				
Collection no.	Lesior leaf		Diamete lesion		Percer defoli		Sporulation index		
(or name)	arach.	pers.	arach.	pers.	arach.	pers.	arach.	pers.	
······									
A. glabrata (a) 5	8	0.5	0.5	0	0	0	0	
A. glabrata (b		15	0.6	0.5	ō	ō	ō	õ	
A. glabrata (c		14	0.5	0.5	Ō	ō	ō	ŏ	
P.1, 151922	25	26	0,5	0.5	ō	ō	ō	ŏ	
9553 GKP	5	15	0.5	0.5	Ō	ō	ŏ	ŏ	
9562 GKP	7	0	0.6	0	ō	ō	ŏ	ŏ	
9564 GKP	35	25	0.5	0.5	Ö	ō	õ	ŏ	
9566(a) GKP	25	26	0.5	0.5	Ó	ō	ŏ	ŏ	
9566(b) GKP	26	27	0.5	0.5	ō	ō	ō	ŏ	
9567 GKP	5	8	0.5	0,5	ō	ō	ŏ	ŏ	
9568 GKP	5	6	0.5	0.5	Ó	Ō	õ	ō	
9569 GKP	12	12	0.5	0.5	0	Ó	õ	ō	
9570 GKP	5	8	0.5	0.5	0	Ó	ō	õ	
9571 GKP	15	15	0.5	0.5	Ó	ō	ō	ō	
9572 GKP	10	13	0.5	0.5	0	ō	õ	ō	
9574(a) GKP	38	28	0.5	0.5	0	Ó	ō	ō	
9574(b) GKP	8		1.0		95		+		
9574(c) GKP	15	13	1.0	0.5	100	0	+	0	
9575 GKP	5	10	0.5	0.5	0	Ō	Ó	ō	
9576 GKP	7	5	0.5	0.5	0	0	ō	Ō	
9577 GKP	8	7	0.5	0,5	0	0	Ó	Ō	
9578 GKP	20	25	0.5	0.6	0	0	Ó	0	
9580 GKP	25	22	0.6	0.5	0	0	0	0	
9587 GKP	10	15	0.5	0.5	0	0	0	0	
9592(a) GKP	12	15	0.6	0.5	0	0	0	0	
9592(b) GKP	8	6	0.5	0.5	0	0	0	0	
9610 GKP	5	15	0.6	0.5	0	0	0	0	
9629 GKP	26	28	0.5	0.5	0	0	0	0	
9634 GKP	15	15	0.5	0.5	0	0	0	0	
9642 GKP	15	10	0.6	0.5	90	0	+	0	
9644 GKP	5	15	0.5	0.5	0	0	0	0	
9645 GKP	45	15	0.5	0.5	0	0	0	0	
9649 GKP	5	6	0.5	0.5	0	0	0	0	
9664 GKP	40	38	0.5	0.5	0	0	0	0	
9667(a) GKP	24	15	0.5	0.5	0	0	0	0	
9667(b) GKP	5	6	0.5	0.5	0	0	0	0	
9797 GKP	38	32	0.5	0.5	0	0	0	0	
9806 GKP	12		0.6		85		+		
9813 GKP	0	34	0	0.5	0	0	0	0	
9815 GKP	5	15	0.5	0.5	0	0	0	0	
9822 GKP 9827 GKP	30 40	29 35	0.5	0.5	0	0	0	0	
9830 GKP	40	32	0.5	0.5		0	0	0	
9834 GKP	42 5	5	0.5 0.5	0.5	0	0	0	0	
9882 GKP	7	6	0.5	0.5	0	0	0	0	
9893 GKP	5	10	0.5	0.5	ő	0	0	0	
9918 GKP	ŝ	10	0.5	0.5	ő	0	0	0	
9921 GKP	5	12	0.5	0.5	ŏ	ő	ŏ	0	
9922 GKP	5	15	0.5	0.5	ŏ	ő	ŏ	0	
9925 GKP	12	8	0.5	0.5	ŏ	ŏ	ŏ	ŏ	
9935 GKP	12	15	0.5	0.5	ŏ	ŏ	ŏ	o	
10105 GKP	25	27	0.5	0.5	ŏ	ŏ	ŏ	ŏ	
10120 GKP	15	15	0.5	0.5	ŏ	õ	ő	ŏ	
10559 GKP	38	20	1.0	1.5	100	95	+	÷	
10566 GKP	25	22	1.0	0.8	0	ő	ò	ŏ	
10596 GKP	ō	ō	0	0	ŏ	ŏ	ŏ	ŏ	
	-	-	-	-	•		•	•	

*See footnotes in Table 2.

Table 5. Reaction of Arachis species of the section Extranervosae to inoculation with Cercospora arachidicola and Cercosporidium personatum.*

	Collection no.		Lesions per leaflet		Diameter per lesion, mm		Percentage defoliation		Sporulation index	
	(or name)		arach.	pers.	arach.	pers.	arach.	pers.	arach.	pers.
	10406 GKP		8	5	0.5	0.5	0	0	0	0
	9906 GKP		6		0.6		90		+	
<u>A</u> .	villosulicarpa	(a)	0	0	0	0	0	0	0	0
<u>A</u> .	villosulicarpa	(b)	0	0	0	0	0	0	0	0
<u>∧</u> .	villosulicarpa	(c)	0	0	0	0	0	0	0	0
	10127 GKP		28	20	1.5	1.0	95	95	++	++

*See footnotes in Table 2.

Discussion

The small sample of A hypogaea tested here reinforces the widely held opinion that resistance to the Cercospora and Cercosporidium leafspots in this species is only moderately variable and has not been observed to extend to such a range as would allow any reduction in the cost of artificial control or to mitigate serious economic losses in its absence.

This situation is in striking contrast to that encountered in species from five of the seven known taxonomic sections of the genus *Arachis*; two of these species of the section Axonomorphae being known to be cross-compatible with cultivated peanuts (Smartt and Gregory, 1967). Unpublished work indicates that the genetic resources of species of other sections of the genus will soon become available to peanut breeders possibly giving rise to multifactorial genetic control of leafspots of peanuts.

It is recognized that such an achievement will not be simple and that the introduction of such amounts of foreign germ plasm into cultivated peanuts risks the appearance of unforeseen new diseases and other difficulties. On the other hand, the wild species are suspected to contain resistance to peanut rust (W. K. Bailey, personal communication), resistance to stunt virus (T. T. Hebert, personal communication), rosette virus (R. W. Gibbons, personal communication), nematode resistance (Donald J. Banks, personal communication), mite resistance (Leuck and Hammons, 1968), lesser corn stalk borer resistance (W. C. Gregory, personal observation) and possibly other barriers to important pests of peanuts.

It appears that the economic challenge here, taken together with the difficulties associated with interspecific transfers of genetic information and the relatively small band of peanut breeders in the world, would justify a concerted and somewhat coordinated attack on peanut improvement through species hybridization.

Literature Cited

- ABDOU, Y. A-M. AND W. E. COOPER. 1974. Effect of culture medium and light on sporulation of two peanut leaf spotting fungi, Cercospora arachidicola Hori and C. personata (B. & C.) Ell. & Ev. Peanut Sci 1:11-14.
- CHEVALIER, A. 1934. Monographie de l'arachide. Res. Bot. Appl. et. Agron. Trop. 24:709-755.
- CHEVAUGEON, J. 1952. Recherches sur la cercosporiose de l'arachide en Moyenne Casamance. Ann. Inst. Nat. Rech. Agron., Ser. C 3:489-510. (Rev. Appl. Mycol. 32:661-662, 1953).
- COOPER, W. E. AND W. C. GREGORY. 1960. Radiationinduced leafspot resistant mutants in the peanut (Arachis hypogaea L.). Agron. Jour. 52:1-4.
- DEBENHAM, E. M. 1939. A modified technique for the examination of the xylem of whole plants or plant organs. Ann. Bot. 3:369-373.
- FREZZI, M. J. 1960. Enfermedades del mani en la provincia de Cordoba (Argentina). Revista de Investigaciones Agricolas 14:113-228.
- GIBBONS, R. W. AND B. E. BAILEY. 1967. Resistance to Cercospora arachidicola in some species of Arachis. Rhod. Zamb. Mai. J. Agric. Res. 5:57-59.
- GREGORY, W. C., M. P. GREGORY, A. KRAPOVICKAS, B. W. SMITH AND J. A. YARBROUGH. 1973. Structures and genetic resources of peanuts, p. 47-133. Peanuts—culture and uses. Amer. Peanut Res. and Educ. Assoc., Inc., Stillwater, Okla.

- HEMINGWAY, J. S. 1957. The resistance of groundnuts to Cercospora leafspot. Empire Jour. Expt. Agric. 25:60-68.
- HIGGINS, B. B. 1935. Breeding peanuts for disease resistance. Phytopathology 25:971-972. (Abstr.).
- JACKSON, C. R. AND D. K. BELL. 1969. Diseases of peanut (groundnut) caused by fungi. Univ. of Ga. Agric. Expt. Sta. Res. Bull. 56, 137 p.
- JOHANSEN, D. A. 1940. Plant microtechnique. McGraw-Hill Book Co., New York. 523 pp.
- LEUCK, D. B. AND R. O. HAMMONS. 1968. Resistance of wild peanut plants to the mite Tetranychus tumidellus. Jour. Econ. Ent. 61:687-688.
- REYES, G. M. AND R. ROMASANTA. 1940. Varietal susceptibility of peanuts to black spot [Cercospora personata (B. & C.) Ell. & Ev.] Phillipp. Jour. Agr. 11:371-382.
- SMARTT, J. AND W. C. GREGORY. 1967. Interspecific cross-compatibility between the cultivated peanut Arachis hypogaea L. and other members of the genus Arachis. Oleagineux 22:1-5.
- SMITH, D. H. 1971. A simple method for producing Cercospora arachidicola conidial inoculum. Phytopathology 61:1414.
- WOODROOF, N. C. 1933. Two leafspots of peanut (Arachis hypogaea L.) Phytopathology 23:627-690.