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Influence of Peanut Stripe Virus on Growth, Yield, and Quality of Florunner Peanut¹

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ABSTRACT

The influence of peanut stripe virus (PSV) on growth, yield, and grade of Florunner peanut and percent seed transmissions was determined under field conditions during 1985 and 1986. Plants were artificially inoculated with PSV and infection was confirmed by enzyme-linked immunosorbent assays. Under the conditions of these tests, PSV did not significantly influence growth, yield, or grade of Florunner peanut, and seed infection averaged less than 2 percent.

Key Words: *Arachis hypogaea*, groundnut.

Peanut stripe virus (PSV) was reportedly introduced into the U.S. in peanut (*Arachis hypogaea* L.) seed from the People's Republic of China as early as 1979 (1,2), but symptoms of this new virus were not detected until 1982 in seed-increases of germplasm lines at the

Georgia Experiment Station, Experiment, GA. The virus was identified as a new virus of peanut in 1983 and designated PSV (1). Demski and Lovell (1) and Kuhn et al. (10) used enzyme-linked immunosorbent assays (ELISA) to identify peanut plants infected with PSV in four widespread Georgia counties in 1983. Further ELISA tests also showed PSV in plants from several states cooperating in peanut seed exchange, i.e., Georgia, Florida, Virginia, North Carolina, and Texas. However, with few exceptions, the virus was thought to be restricted to institutional and research test plots since it was not found in randomly sampled commercial fields in any of these states (10).

Peanut stripe virus is a potyvirus that is transmitted mechanically, by aphids in a non-persistent manner, and by seed (3). Characteristic symptoms are striping or discontinuous dark green banding along the lateral veins of young leaves and an oakleaf or blotched pattern of dark green on older leaves. Serologically and symptomatically, PSV differs from peanut mottle virus (PMV) a previously identified endemic virus of peanut (9, 12), by ELISA and by symptoms in peanut and other host plants. Other hosts of PSV include soybean (*Glycine max* [L.] Merr.), cowpea (*Vigna unguiculata* [L.] Walp.), white lupine (*Lupinus albus* L.), wild tobacco (*Nicotiana benthamiana* Domin.), crimson clover (*Trifolium incarnatum* L.), arrowleaf clover (*T. ves-*

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iculostum Savi), subterranean clover (*T. subterraneum* L.), sesame (*Sesamum indicum* L.), and Florida beggarweed (*Desmodium tortuosum* [D.C.] Schw.) (3).

Preliminary greenhouse studies suggested a 20% yield loss due to decreased seed number and weight and an amount of seed transmission of 19 to 37% [compared with 2% for PMV (9)] when plants were inoculated with PSIV at the third to fifth leaf stage (1). Because of the initial yield loss estimate, amount of seed transmission, and the other important agricultural hosts infected by the virus, PSIV was considered a threat to the U.S. peanut industry, and restrictions on movement and testing of infected germplasm were initiated in several states. Therefore, cooperative research among peanut scientists in Georgia was initiated to determine the influence of PSIV on growth, yield, quality of Florunner peanut and amount of seed transmission of the virus under field conditions.

Materials and Methods

Field experiments were conducted on the Agronomy Farm, Coastal Plain Experiment Station, in 1985 and 1986 to determine the effect of PSIV infection initiated at different stages of plant development on Florunner peanut. Certified Florunner seed were planted on Tifton loamy sand (fine, loamy, siliceous, thermic Plinthic Paleudults) at ca. 120 kg/ha in two rows, 51 cm between rows, on a 1.83-m bed. In both years, the fields were treated before planting with benefin (N-Butyl-N-ethyl- α , α , trifluoro 2, 6-dinitro-p-toluidine) (1.25 kg ai/ha) and vernolate (5-Propyl dipropylthiocarbamate) (2.24 kg ai/ha), and prior to complete plant emergence with alachlor [2-chloro-2', 6'-diethyl-N-(methoxymethyl) acetanilide] (3.36 kg ai/ha) and naptalan (N-1 Naphthylphthalamic acid) + dinoseb [2-sec butyl 4,6-dimethylphenol alkanolamine salts] (3.36 + 1.68 kg ai/ha, respectively) for week control as recommended by the Georgia Extension Service. Aldicarb [2-Methyl-2-methylthio-propionaldehyde O-methylcarbamoyloxime] (0.67 kg ai/ha) was applied at planting to control early season insects. Beginning ca. 40 days after plant emergence, all plants were sprayed for leafspot control with chlorothalonil (Tetrachloroisophthalonitrile) (2.48 l ai/ha) at ca. 10- to 14-day intervals using an air-blast sprayer.

Two-row plots, 2.44 m X 1.83 m, were established at plant emergence by removing plants from a 1.83-m space between replications. A Lumite[®] screen cage (Chicopee, P. O. Box 2537, Gainesville, GA 30502), 1.83 m wide X 1.83 m high X 3.66 m long, of 7.9 X 7.9 mesh/cm screen supported by a conduit frame was placed over each plot that was inoculated with PSIV and untreated control plots to prevent aphid transmission of the virus among plots.

The experiment in 1985 was designed in a randomized complete block with 9 to 10 replications. Treatments were: (1) uninoculated, uncaged control, (2) uninoculated, caged control, and inoculated at (3) emergence, i.e., plants with 1-3 tetrafoliates completely expanded; (4) 20 days; (5) 40 days; and (6) 60 days post-plant emergence. Plants in each inoculated plot were dusted with 600 grit Carborundum powder and all plants in each plot were inoculated at several locations, i.e., 3 to 5 leaves/plant, with PSIV. White lupine infected with PSIV was macerated in 0.25 M phosphate buffer (pH 7.2) with a mortar and pestle just before inoculation. Cheesecloth was then dipped into the buffer containing macerated tissue and rubbed on the peanut leaflets of each plant. The Carborundum on these leaves produced microscopic lesions which allowed transmission of PSIV into the plants. Uninoculated plants were not treated with Carborundum or the buffer solution.

In 1986, the experiment was designed in a randomized complete block with a split plot arrangement of treatments and 10 replications. Whole plots were caged versus uncaged plots and subplots were (1) uninoculated control, inoculated at (2) plant emergence; (3) 20 days; (4) 40 days; and (5) 60 days postplant emergence. Plants were inoculated with PSIV as described above. To reduce possible transmission of the virus among uncaged plants, all plants were sprayed at weekly intervals with bifenthrin [2-methyl (1,1'-biphenyl)-3-yl)methyl 3-(2-

chloro-3,3,3-trifluoro-1-propenyl)-2,2-dimethylcyclopropanecarboxylate] (0.067 kg ai/ha) in 1986, since pyrethroids have been shown to control aphids, increase dispersal, and decrease virus transmission (6, 13, 14).

Leaf samples were collected from plants in each plot before each inoculation and at 20-day intervals throughout the growing season. Five fully expanded young leaves were collected from each of the two rows per plot as subsamples. These subsamples were placed in an ice chest in the field and kept cool until analyzed for PSIV by the direct ELISA technique of Demski et al. (3). Leaves from each subsample were bulked and two ELISA assays were conducted per plot on each date. All ELISA reactions were assayed by reading absorbance at 410 nm in a Dynatech ELISA reader (Dynatech Laboratories, Inc., 960 Slaters Lane, Alexandria, VA 22304). Three virus controls, three healthy controls, and three blank wells were randomly distributed in each plate. Absorbance values for blank wells were subtracted from absorbance values from plate wells containing healthy controls to give absorbance values of healthy controls. Samples judged positive for PSIV infection had absorbance values at least twice as large as those of the healthy controls.

The plants in all plots were dug at a depth of ca. 14.4 cm with a peanut inverter ca. 145 days after planting. Number of plants, number of plants infested with *Sclerotium rolfsii* Sacc., number of hits, i.e., number of row feet infested with *S. rolfsii*, and plant height were recorded for each plot. Ten plants then were separated at random from each plot and plant weight, top weight, root weight, and fresh pod weight were recorded. Pods from each of the 10 plants were placed in individual bags with plot labels, and then placed in a larger bag by plot and dried for 24-48 hrs at 36 C to ca. 15% moisture. Other plants in each plot remained in the field for 5 to 7 days. The plots then were harvested with a stationary plot thresher, and pods were placed in a labeled bag and dried as described above. Dry pod weight, number of pods, number of seed, and seed weight for the 10 individual plant samples were recorded. Pod weight for the remainder of the plots was recorded and 1000 g pod samples were shelled and graded according to USDA grading procedures. Seed from the 10 individual plants from each plot were analyzed for seed transmission of PSIV by the technique described by Demski and Warwick (4).

All data were analyzed using SAS (16). Means were compared using orthogonal comparisons (17) for the field data and Duncan's (5) new multiple range test for the ELISA data. In 1986, covariance analyses (17) were used to analyze the field data due to significant differences in *S. rolfsii* infection.

Results and Discussion

ELISA analyses for PSIV infection in peanut leaves in 1985 showed that plants were not infected before inoculation (Table 1). Plants in all control plots (caged and uncaged) remained free of PSIV infection throughout the growing season. PSIV was detected 20 days after inoculation in plants inoculated at emergence. Likewise, later inoculations with PSIV for the other inoculation treatments resulted in significantly higher ELISA values

Table 1. Enzyme-linked immunosorbent assay (ELISA) values for presence of peanut stripe virus (PSIV) when peanut was artificially inoculated at different plant ages (Tifton, Ga., 1985).

Treatment ¹	ELISA values for PSIV on indicated day ²					
	May 10	May 30	June 19	July 9	July 30	August 19
Uncaged control	0a	0.02b	0.03c	0.01c	0.02c	0.00c
Caged control	0a	0.02b	0.11c	0.03c	0.03c	0.01c
Emergence	0a	0.55a	0.58b	0.78a	0.55a	0.20a
Post-20	0a	0.02b	0.49a	0.58b	0.45a	0.20a
Post-40	0a	0.01c	0.01c	0.87a	0.58a	0.20a
Post-60	0a	0.02b	0.01c	0.02c	0.28b	0.09b
Standard error	0	0.25	0.40	0.46	0.32	0.11

¹ The uncaged control and caged control were not inoculated with PSIV. The emergence was inoculated May 10, Post-20 inoculated May 30, Post-40 inoculated June 19, and Post-60 inoculated July 9.

² Means within a column followed by the same letter are not significantly different ($p < 0.05$; Duncan's multiple range test).

than for the caged and uncaged control by 20 days after inoculation. Only the ELISA value for the post-60 treatment recorded August 19 resulted in a questionable mean reading for infection. The mean ELISA value for this treatment was significantly higher than those for the controls, and the mean ELISA value for the previous sampling date showed positive infection for the post-60 treatment.

Orthogonal comparisons for the 1985 test indicated that shading by the Lumite screen cages significantly reduced fresh pod weight, fresh root weight, seed weight/

did not affect yield of Florunner peanut when plants were grown in screened cages.

Orthogonal comparisons in 1985 also indicated that shading had the greatest influence on peanut grade (Table 3). The percent virginia pods, percent meats, weight/100 SMK, percent ELK, and total percent SMK were significantly greater for the caged control plants than for the uncaged control plants. PSTV infection initiated at any plant age tested did not reduce peanut grade, except for the percent virginia pods for plants inoculated 40 days after emergence. Other grade

Table 2. Effects of peanut stripe virus on growth and yield of Florunner peanuts (Tifton, GA, 1985).

Orthogonal comparison	No. plants/plot	No. plants with <u>S. rolfesii</u>	Fresh plant wt. (g)	Fresh pod wt. (g)	Fresh top wt. (g)	Fresh root wt. (g)	Seed wt./plant (g)	No. Seed/plant	Total yield/plot (g)	% yield Reduction
1. Caged Control vs Uncaged control	70.4	3.4	277.7	82.0	181.2	5.9	39.5	52.4	2396.8	-27.1
	71.3	2.1	296.3	100.2	175.8	8.3	37.7	83.3	3290.0	
2. Caged Control vs Inoculated @ Emergence	70.4	3.4	277.7	82.0	181.2	5.9	30.5	52.4	2396.8	- 7.5
	72.2	0.5	274.5	76.0	181.0	5.9	27.8	50.1	2217.1	
3. Caged Control vs Inoculated @ Post 20	70.4	3.4	277.7	82.0	181.2	5.9	30.5	52.4	2396.8	- 1.5
	67.3	1.4	260.0	76.4	170.1	5.2	28.8	50.7	2359.7	
4. Caged Control vs Inoculated @ Post 40	70.4	3.4	277.7	82.0	181.2	5.9	30.5	52.4	2396.8	- 2.8
	74.3	0.8	260.5	77.6	168.1	5.8	29.0	54.7	2329.8	
5. Caged Control vs Inoculated @ Post 60	70.4	3.4	277.7	82.0	181.2	5.9	30.5	52.4	2396.8	+ 0.5
	71.4	1.2	251.9	76.4	163.4	5.3	28.1	51.0	2410.2	

* = comparison significant at p = 0.05; ** = comparison significant at p = 0.01.

plant, number of seed/plant, and yield of plants in the caged-control plots compared with plants in the uncaged-control plots (Table 2). Caged control plants and plants in the various PSTV treatments differed only in fresh root weight, which was significantly lower for plants inoculated with PSTV at 20 days after emergence than for the caged control plants. Yields for plants inoculated with PSTV did not differ significantly from the yields in the uninoculated, caged control. Thus, PSTV

parameters for plants infected at this age did not differ significantly from parameters for uninoculated caged-control plants. Thus, PSTV had little or no effect on grade of Florunner peanut in 1985.

Table 3. Treatment means and significance of orthogonal comparisons for the effects of peanut stripe virus on grade of Florunner peanuts (Tifton, GA, 1985).

Orthogonal comparison	Virginia pods (%)	Meats (%)	wt. 100 SMK (g)	ELK (%)	Total SMK (%)
1. Caged Control vs Uncaged Control	4.6	80.2	69.1	38.6	76.3
	1.7	79.5	59.4	22.9	74.5
2. Caged Control vs Inoculated @ Emergence	4.6	80.2	69.1	38.6	76.3
	4.5	80.0	68.7	35.1	75.9
3. Caged Control vs Inoculated @ Post 20	4.6	80.2	69.1	38.6	76.3
	4.8	80.7	69.6	36.5	76.9
4. Caged Control vs Inoculated @ Post 40	4.6	80.2	69.1	38.6	76.3
	3.1	80.4	67.9	35.4	75.9
5. Caged Control vs Inoculated @ Post 60	4.6	80.2	69.1	38.6	76.3
	4.1	80.4	68.4	38.6	76.8

* = comparison significant at p = 0.05; ** = comparison significant at p = 0.01.

The 7.9 X 7.9 mesh/cm Lumite screen used for cages reduced light penetration by 22 to 27%. Shading altered production of sinks and partitioning of assimilates to the plants; above-ground vegetative plant growth was similar in caged and uncaged plots, but shading reduced root growth and seed production. However, seed that were produced on shaded plants were larger than seed produced on unshaded plants. Thus, plants grown under shade produced fewer seed, whereas under full sun plants produced more seeds but were unable to produce sufficient assimilate for maximum seed yield, resulting in smaller kernels.

The effects of shading on growth, partitioning, and yield for peanut have been reported (7, 8, 11, 18). However, these reports are for shading for various periods during development, rather than from soon after emergence through maturity as reported here. Partial shading from plant emergence to first flower production reduced peg production and number of seed, but seed that were produced had a higher mean weight (18), as noted in the present study. Shading (75%) during peak flowering reduced the number of flowers and inhibited peg formation, while shade during the pegging and pod-

ding phases reduced pod and peg numbers and pod dry weight (7). Prolonged shading reduced shoot dry weight and number of mature pods and seed (11). Complete shading was most critical during peak flowering, but it also reduced vegetative growth if initiated prior to the onset of flowering, or pod fill if initiated after flowering (8). In the present study, it appeared that peanut plants adapted to the reduced light intensity. The total number of seeds and dry pod weight were reduced, probably as a result of shading during flowering, but the plants compensated for the fewer seed by producing larger kernels. However, total compensation was not achieved as reported by Williams (18) for shading during early vegetative growth.

Over 8,000 individual seeds from 3 replications were analyzed using ELISA for seed transmission of PSTV (4). Seed infection averaged 1.75% for plants infected at emergence, 0.19% for plants infected 20 days after emergence, and 0.0% for the caged control, uncaged control, and plants infected 40 and 60 days after emergence. Only the emergence treatment differed sig-

nificantly from the other treatments. Plants in the remaining treatments were not.

Analyses of the July 7 ELISA readings showed no significant differences between PSTV infection for the caged and uncaged plants. Significant PSTV infection occurred in plants that were inoculated at emergence and post-20 days, while there was no infection detected in plants that had not been inoculated (Table 4).

ELISA readings were low for the July 28 leaf samples with questionable values for infection for all treatments (Table 4). A significant cage X stage interaction was detected that, unlike the previous interaction, resulted from the significantly higher ELISA values for caged plants from the emergence, post-20, and post-40 inoculated treatments than for uncaged plants from these treatments. No significant difference in infection was noted for the post-60 and untreated treatments between the caged and uncaged plants.

Analyses of the ELISA values for the August 17 samples showed no significant differences in infection be-

Table 4. Enzyme-linked immunosorbent assay (ELISA) values for peanut stripe virus (PSTV) when peanut was artificially inoculated at different plant ages (Tifton, GA, 1986).

Age at Inoculation ¹	ELISA values for PSTV on indicated day ²																	
	June 17*			July 7			July 28*			August 17			September 6			September 28		
	caged	uncaged	mean	caged	uncaged	mean	caged	uncaged	mean	caged	uncaged	mean	caged	uncaged	mean	caged	uncaged	mean
Emergence	0.31Y	0.56X	0.44a	0.28	0.40	0.34a	0.12X	0.08Y	0.10a	0.27	0.24	0.26a	0.17	0.18	0.17a	0.20	0.31	0.26a
Post-20	0.00Z	0.00Z	0.00b	0.29	0.42	0.36a	0.13X	0.07Y	0.10a	0.22	0.36	0.29a	0.13	0.19	0.16a	0.22	0.22	0.22a
Post-40	0.00Z	0.01Z	0.01b	0.02	0.03	0.02b	0.08Y	0.03Z	0.05b	0.11	0.09	0.10b	0.14	0.13	0.14a	0.09	0.08	0.08b
Post-60	0.00Z	0.00Z	0.00b	0.01	0.02	0.01b	0.02Z	0.01Z	0.02c	0.09	0.07	0.08bc	0.05	0.08	0.07b	0.10	0.08	0.09b
Untreated	0.01Z	0.01Z	0.01b	0.01	0.01	0.01b	0.03Z	0.02Z	0.03c	0.01	0.02	0.01c	0.04	0.08	0.06c	0.04	0.07	0.05b
Mean	0.07b	0.16a		0.12a	0.18a		0.06a	0.04b		0.14a	0.16a		0.11a	0.13a		0.13a	0.15a	
Standard Error	0.02			0.02			0.004			0.01			0.01			0.01		

¹ The emergence treatment was inoculated with PSTV May 28, Post-20 was inoculated June 17, Post-40 was inoculated July 7, and Post-60 was inoculated July 26.

² Means within a sampling date followed by the same lowercase letter are not significantly different, and interaction means for caged versus uncaged within an age at inoculation followed by the same uppercase letter are not significantly different ($p = 0.05$, Duncan's multiple range test).

* Significant cage X stage interaction for this sampling date.

nificantly in PSTV seed infection from all other treatments. These seed infection rates were considerably lower than the 19 to 37% reported for earlier greenhouse studies (1, 2, 3). Thus, possible interactions between shading and expression of PSTV could not be discounted. Therefore, in 1986 the test was conducted under caged and uncaged conditions.

ELISA analyses of peanut leaves for PSTV infection in the 1986 test showed that inoculation at emergence resulted in infection that was detected 20 days later, but also produced a significant cage X stage interaction (Table 4). This interaction resulted from a significantly higher ELISA reading from plants in the uncaged-emergence treatment than for plants in the caged-emergence treatment; there were no significant differences between the caged and uncaged plots for the other treatments. More importantly, the ELISA analyses indicated that plants in the emergence treat-

ment were infected with PSTV whereas plants in the remaining treatments were not. Mean ELISA values for plants from the emergence and post-20 day treatments were significantly higher than ELISA values for all other treatments. ELISA values for plants from the post-40 day inoculation treatment were also significantly higher than ELISA values for plants from the untreated control, but the low ELISA values indicated questionable PSTV infection for plants from these treatments. ELISA values for plants from post-60 day inoculation treatment were not significantly different from the values for plants from the untreated control, even though plants in the post-60 day treatment were inoculated with PSTV 20 days earlier.

Analysis of the September 6 ELISA readings for peanut leaf samples was similar to analyses of previous samples (Table 4). ELISA readings between the caged and uncaged plants did not differ, but ELISA values were significantly different for plants from the different

inoculation treatments. Plants inoculated with PSIV at emergence, post-20 days, and post-40 days had significantly higher ELISA readings than plants inoculated post-60 days or the uninoculated control. Also, the post-60 day inoculation produced variable ELISA readings that were not significantly different from the readings for untreated control. This was due in part to infection in the uncaged control plants; samples from 4 of the 10 control plots tested positive for the virus.

Two days before harvest, leaf samples from caged and uncaged plots had similar ELISA readings, but plants from the emergence and post-20 day treatments had significantly higher ELISA readings than plants from the other treatments (Table 4). Samples from the post-40 day, post-60 day, and uninoculated control plots had similar low ELISA readings. Infection was noted in at least one row of the uncaged, uninoculated control for 6 of the 10 replications. No infected plants were detected in the caged, uninoculated control plots. Additional

control plots at harvest, and all yield and grade data were obtained from these substituted plots.

In 1986, *S. rolfssii* was prevalent in the test plots and undoubtedly reduced yield. Furthermore, *S. rolfssii* incidence was significantly higher outside the cages than inside the cages as measured by both number of diseased plants and number of hits (Table 5). Therefore, analyses of covariance with number of diseased plants, number of hits, or percent diseased plants as the covariant were conducted for field, yield, and grade variables. Results of these analyses indicated that *S. rolfssii* did not influence the effects of PSIV.

As in the previous year, major differences were noted between the caged and uncaged plots (Table 5); plants outside the cages had significantly greater fresh root weight, number of seed/plant, and total yield than plants inside the cages. Shading reduced yield by 33.9%. Plants inoculated at emergence had a significantly lower fresh root weight than uninoculated plants,

Table 5. Treatment means and significance of orthogonal comparisons for the presence of *S. rolfssii* and the effects of peanut stripe virus on growth and yield of Florunner peanuts (Tifton, GA, 1986).

Orthogonal comparison	No. plants/plot	No. hits ¹	No. plants with <i>S. rolfssii</i>	Fresh plant wt. (g)	Fresh pod wt. (g)	Fresh top wt. (g)	Fresh root wt. (g)	Seed wt./plant (g)	No. Seed/plant	Total yield/plot (g)	% Yield reduction
1. Caged vs Uncaged	45.1	3.5 *	10.0 **	341.9	88.6	238.2	5.6 **	37.9	76.9 **	1670.1 **	-33.9
2. Uninoculated vs Inoculated @ Emergence	49.1	4.3	14.9	353.6	92.2	243.0	6.0 **	38.8	83.0	2207.6	- 8.6
3. Uninoculated vs Inoculated @ Post 20	49.1	4.3	14.9	353.6	92.2	243.0	6.7	38.8	83.0	2207.6	- 3.9
4. Uninoculated vs Inoculated @ Post 40	49.1	4.3	14.9	353.6	92.2	243.0	6.7	38.8	83.0	2207.6	- 1.9
5. Uninoculated vs Inoculated @ Post 60	49.1	4.3 **	14.9	353.6	92.2	243.0	6.7	38.8	83.0	2207.6	-10.4
6. Uncaged - Uninoculated vs Uncaged-Inoculated @ Emergence	50.3	5.1	17.8	347.1	95.2	232.5	7.8	40.4	89.9	2680.4	- 9.2
7. Uncaged - Uninoculated vs Uncaged - Inoculated @ Post 20	50.3	5.1	17.8	347.1	95.2	232.5	7.8	40.4	89.9	2680.4	- 7.5
8. Uncaged - Uninoculated vs Uncaged - Inoculated @ Post 40	50.3	5.1	17.8	347.1	95.2	232.5	7.8	40.4	89.9	2680.4	- 0.7
9. Uncaged - Uninoculated vs Uncaged - Inoculated @ Post 60	50.3	5.1	17.8	347.1	95.2	232.5	7.8	40.4	89.9	2680.4	-11.4

* = Comparison significant at p = 0.05; ** = Comparison significant at p = 0.01.

¹ No. hits = number of row feet infected with *S. rolfssii*.

samples from 20 border plots adjacent to the uncaged, uninoculated control plots were analyzed by ELISA for infection with PSIV; 15 of these border plots tested negative for PSIV. The border plots were of the same dimensions as the uncaged, uninoculated plots and had been treated similarly. Therefore, adjacent uninfected border plots were substituted for infected, uncaged con-

and plants inoculated 60 days after emergence had significantly more *S. rolfssii* hits than uninoculated plants. There were no other significant differences for these treatments in any of the yield components. This was especially true for the uncaged-uninoculated treatment compared with the uncaged-inoculated treatments, i.e., orthogonal comparisons 6 through 9. Thus, even under

uncaged conditions, PSTV did not significantly affect yield of Florunner peanut.

Analyses of the 1986 peanut grade data indicated that, as in 1985, shading altered peanut grades (Table 6). Plants grown inside the cages had a significantly higher percent virginia pods, percent ELK, and weight/100 SMK than did plants grown outside the cages. As noted in 1985, plants grown inside the cages produced significantly fewer seeds but were able to partition photosynthate to significantly increase the size of the pods and kernels. Percent meats, percent ELK, weight/100 SMK, and total percent SMK were similar in uninoculated and PSTV-inoculated treatments.

Table 6. Treatment means and significance of orthogonal comparisons for the effects of peanut stripe virus on grade of Florunner peanuts (Tifton, GA, 1986).

Orthogonal comparison	Virginia pods (%)	Meats (%)	Wt. 100 SMK (%)	ELK (%)	Total SMK (%)
1. Caged vs Uncaged	4.1 **	74.6	60.2 **	24.0 **	69.8
	12.0	76.8	52.6	10.5	69.7
2. Uninoculated vs Inoculated @ Emergence	8.1	79.3	56.2	14.6	73.0
	8.6	75.7	56.7	17.1	69.9
3. Uninoculated vs Inoculated @ Post 20	8.1	79.3	56.2	14.6	73.0
	4.0	75.5	57.0	17.4	70.2
4. Uninoculated vs Inoculated @ Post 40	8.1	79.3	56.2	14.6	73.0
	8.2	72.3	55.9	16.8	69.3
5. Uninoculated vs Inoculated @ Post 60	8.1	79.3	56.2	14.6	73.0
	6.9	77.7	56.4	16.1	66.8
6. Uncaged - Uninoculated vs Uncaged-Inoculated @ Emergence	2.9	79.8	60.5	26.7	74.6
	4.8	73.7	60.4	23.9	69.1
7. Uncaged - Uninoculated vs Uncaged - Inoculated @ Post 20	2.9	79.8	60.5	26.7	74.6
	1.4	79.9	60.4	25.0	74.6
8. Uncaged - Uninoculated vs Uncaged - Inoculated @ Post 40	2.9	79.8	60.5	26.7	74.6
	5.5	73.0	59.3	23.0	68.3
9. Uncaged - Uninoculated vs Uncaged - Inoculated @ Post 60	2.9	79.8	60.5	26.7	74.6
	1.9	66.5	60.6	21.3	62.6

** = Comparison significant at $p = 0.01$.

Over 5,000 individual seed from the 1986 test were analyzed for the presence of the virus using ELISA (4). Only one seed tested positive for the presence of the virus, and thus there were no significant differences in seed transmission of PSTV among treatments.

In conclusion, field research in 1985 and 1986 indicated that PSTV did not influence measured components of growth, yield, and grade of Florunner peanut. The amount of seed transmission of the virus averaged less than 2% under field conditions, similar to that reported for PMV (9). The effects of PSTV on peanut appear similar to those reported for PMV (9) under field conditions, i.e., a minimum influence on yield and quality. Ross *et al.* (15) analyzed the chemical constituents of kernels from the 1985 grade samples. Concentrations of manganese, zinc, iron, tartaric acid, raffinose, glucose, fructose, and total carbohydrates were significantly higher in seed from infected plants, while concentrations of potassium, magnesium, and total soluble phenolics were significantly lower in seed from infected plants compared with seed from the uninfected-caged control. The impact of these findings on peanut quality, taste, and nutritive value will require additional research.

Peanut stripe virus can be rapidly disseminated by aphids in a peanut field. In 1984, several peanut cul-

tivars being evaluated for insect resistance were identified by ELISA as infected with PSTV. Seed from these cultivars were tested for PSTV using ELISA techniques (4), and seed that tested negative were planted in 1985 for seed increase in isolation from other peanuts. The ELISA technique for seed was 99.8% effective in identifying infected seed (4). Even with only 0.2% infected seed at planting, over 50% of the plants tested positive for PSTV just before harvest. However, based on the above research and observation of these infected plants, it is doubtful that PSTV had a significant effect on these cultivars.

Breeding of new and improved peanut cultivars with increased yield, grade, insect resistance, or disease resistance offers tremendous potential for improving commercial peanut production. Thus, as a result of research herein reported, all restrictions in Georgia on movement and testing PSTV-infected germplasm have been rescinded.

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