The Impact of Peroxyacetyl Nitrate on Conductance of Bean Leaves and on Associated Cellular and Foliar Symptom Expression

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ABSTRACT

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Phaseolus vulgaris 'Provider' seedlings were cultured in sand in a growth chamber. Beginning at 1100 hours 6 days after emergence, plants were exposed to one of the following dosages of peroxyacetyl nitrate (PAN): subthreshold—271 $\mu g/m^3$ for 1 hr; split threshold—405 $\mu g/m^3$ for 0.5 hr (subthreshold) or 405 $\mu g/m^3$ for 1 hr (above threshold); or threshold—360 $\mu g/m^3$ for 1 hr. Stomatal conductance of primary leaves was measured at 1200 hours and 2100 hours on three consecutive days beginning 1 day before exposure to PAN. Plants exposed to subthreshold dosages showed no macroscopic symptoms and stomatal conductance for these plants remained similar to that of controls. Dosages above or at threshold levels produced abaxial glazing; stomatal conductance of these plants was higher at 2100 hours each night after exposure and lower at 1200 hours 1 day after exposure. A histological study was conducted on primary leaf tissue of plants exposed to an above-threshold dosage of PAN. Cross sections of tissue samples taken 3 hr after exposure showed small numbers of plasmolyzed abaxial epidermal and spongy mesophyll cells. Larger numbers, but similar percentages, of epidermal and mesophyll cells were collapsed 6 and 9 hr after exposure. Proximity to stomata was directly correlated with cellular injury. Guard cells remained intact and normal in appearance.

When *Phaseolus vulgaris* (L.) 'Provider' was exposed to levels of peroxyacetyl nitrate (PAN) which did not elicit macroscopic symptoms, water potential became more negative, soil moisture decreased, and wilting accelerated (12). Dugger and Ting (4) reported that pinto bean plants which received a PAN dose above threshold lost water more rapidly than did nonexposed control plants.

Histological studies of leaf tissue exposed to PAN (5,6,8,18) revealed general symptomatology and provided a potential explanation for changes in water status. Hindawi (6) reported that typical lower leaf symptoms caused by PAN were due to the plasmolysis of epidermal and spongy mesophyll cells. A decrease in the turgor of epidermal cells could result in a reduction of pressure exerted on the guard cells and subsequent increase in stomatal aperture and associated transpiration. Kohut (8) reported that abaxial epidermal cells and adjacent spongy mesophyll cells of pinto bean leaves collapsed in response to doses of PAN which exceeded the threshold for foliar injury. Thomson et al (18) reported that the first microscopic symptom in pinto bean plants exposed to PAN consisted of plasmolysis and collapse of a small number of substomatal spongy mesophyll cells; guard cells were unaffected. Glater (5) conducted a microscopic examination of foliage from plants exposed to "PAN-type-smog." Injury began with a reversible accumulation of water in affected cells, including guard cells, and resultant enlarged stomatal aperture. Irreversible damage began with the plasmolysis of cells lining the substomatal chambers. Stomata remained functional, but the rate of transpiration was not measured.

The objective of this study was to correlate development of PAN symptoms in primary bean leaves with accompanying changes in water relations by characterizing leaf conductance during the day and night periods following exposure to PAN and by quantifying cellular injury following similar exposures to PAN. The selection of PAN dosages bracketing the macroscopic injury threshold of primary bean leaves was intended to identify responses which occur on either side of this threshold.

MATERIALS AND METHODS

Culture of plants. Provider bean seeds were sown in flats of vermiculite and soaked with a 5.3 g/L solution of 20-20-20 (NPK). The flats were then placed in a growth chamber programmed at 24 C and 70% RH day, 21 C and 70% RH night, with a 12-hr photoperiod (0600 to 1800 hours) of 25 klux provided by fluorescent and incandescent bulbs. Thereafter, the plants were watered daily without fertilizer until they were transplanted. Three days after emergence individual plants were transplanted to plastic pots 7.62 cm in diameter, filled with 220 g of white quartz sand and watered to container capacity with Hoagland's solution #1 (7).

Exposure to PAN. Three days after transplanting, the 6-day-old bean seedlings were exposed to PAN in a modified growth chamber (21) maintained at 24 C, 75% RH. All plants received the 3 hr pre- and post-exposure light treatment required for injury development (3,16,17). Exposures were begun at approximately 1100 hours. In all experiments an equal number of plants were maintained in a control chamber.

The generation, collection and storage of PAN were conducted according to Stephens (15). Exposure and calibration were executed by methods previously described (1,10,14).

Experimental design. *I. Subthreshold.* Plants were exposed to 271 $\pm$ 74 $\mu g/m^3$ (0.05 $\pm$ 0.015 ppm) PAN or charcoal-filtered air for 1 hr. The experiment was conducted twice with 16 plants per treatment in each experiment.

II. Split threshold. Plants were exposed to 405 $\pm$ 74 $\mu g/m^3$ (0.08 $\pm$ 0.015 ppm) PAN or filtered air and half of the seedlings were removed from the chamber after 0.5 hr (subthreshold) while the balance received a 1-hr (above threshold) exposure. The experiment was conducted twice with 10 plants per treatment in each experiment.

III. Threshold. Plants were exposed to 360 $\pm$ 74 $\mu g/m^3$ (0.07 $\pm$

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TABLE 1. Conductance of primary leaves of *Phaseolus vulgaris* 'Provider' exposed to 405 $\mu g/m^3$ of peroxyacetyl nitrate (PAN) for 1.0 hr, a level above the threshold for foliar injury

Time[a]	Time of day (hours)	Leaf conductance (cm/sec) Adaxial +PAN	Adaxial −PAN	Abaxial +PAN	Abaxial −PAN
−24	1200	0.1535[b]	0.1433	0.4057	0.3819
−13	2100	0.0171	0.0176	0.0767	0.0699
0	1200	0.1401	0.1286	0.4342	0.4395
+9	2100	0.0248 *	0.0193	0.1651 *	0.0364
+24	1200	0.1062 *	0.1700	0.2469 *	0.5351
+33	2100	0.0204	0.0199	0.1517 *	0.0261

[a]Hours preceding or following exposure to PAN; "0" refers to values observed at termination of exposure to PAN.
[b]Each entry is the mean of two replicate experiments each comprised of 10 readings. *Indicates significant difference between conductance values for +PAN and −PAN at $\alpha = 0.05$.

TABLE 2. Conductance of primary leaves of *Phaseolus vulgaris* 'Provider' exposed to air containing 360 $\mu g/m^3$ of peroxyacetyl nitrate (PAN) for 1.0 hr, which resulted in visible foliar injury

Time[a]	Time of day (hours)	Leaf conductance (cm/sec) Adaxial +PAN	Adaxial −PAN	Abaxial +PAN	Abaxial −PAN
−24	1200	0.1233[b]	0.1224	0.3875	0.3859
−13	2100	0.0215	0.0181	0.0981	0.1105
0	1200	0.1994	0.1835	0.5758	0.6384
+9	2100	0.0175	0.0190	0.1061 *	0.0557
+24	1200	0.1431	0.1574	0.2246 *	0.4841
+33	2100	0.0129 *	0.0236	0.1119 *	0.0584

[a]Hours preceding or following exposure to PAN; "0" refers to values observed at termination of exposure to PAN.
[b]Each entry is the mean of two replicate experiments each comprised of 15 readings. * Indicates significant differences between conductance values for + PAN and −PAN at $\alpha = 0.05$.

TABLE 3. Conductance of leaf surfaces of *Phaseolus vulgaris* 'Provider' plants exposed to air containing 360 $\mu g/m^3$ of peroxyacetyl nitrate (PAN) for 1.0 hr, which resulted in visible foliar injury in five of 15 plants

Time[a]	Time of day (hours)	Leaf conductance (cm/sec) Adaxial Uninjured	Adaxial Injured	Adaxial Control	Abaxial Uninjured	Abaxial Injured	Abaxial Control
−24	1200	0.1365[b]	0.1445	0.1440	0.3934	0.4305	0.4013
−13	2100	0.0245	0.0196	0.0156	0.0840	0.1188	0.1149
0	1200	0.1121	0.1754	0.1238	0.2855 *	0.4789	0.3694
+9	2100	0.0087	0.0078	0.0087	0.0193	0.0349	0.0247
+24	1200	0.1799	0.1354	0.1760	0.4252 *	0.2748 *	0.4783
+33	2100	0.0120	0.0104	0.0120	0.0260	0.0461	0.0330

[a]Hours preceding or following exposure to PAN; "0" refers to values observed at termination of the exposure to PAN.
[b]Uninjured, injured, and control entries are the mean of 10, 5, and 15 readings, respectively. * Indicates significant difference between conductance values for injured and uninjured or injured and control at $\alpha = 0.05$.

0.015 ppm) PAN or filtered air for 1 hr. The experiment was conducted three times with 15 plants per treatment in each experiment. Presence of foliar injury was noted 48 hr after exposure.

Leaf conductance. Conductance was measured on adaxial and abaxial surfaces of primary leaves of bean plants with an aspirated diffusion porometer (19,20). Measurements were performed at 1200 hours at 25 klux (day) and at 2100 hours (night); the latter readings were taken by using a 15 W safelight with an orange filter. The safelight did not influence leaf conductance.

In experiment I, conductance was measured immediately after exposure to PAN and again at 9, 24, and 33 hr after exposure. Conductance rates of primary leaves were measured on eight plants each from the exposed and control treatments at each time interval in each trial.

In experiment II, conductance was measured on 10 exposed plants from the 1 hr and 0.5 hr exposure treatments and on 10 controls at each time. Measurements were taken 24 and 13 hr prior to the termination of exposure, immediately after exposure and again at 9, 24, and 33 hr after exposure.

In experiment III, conductance was measured on 15 primary leaves of exposed and control plants at each time in each trial. Measurements were taken as for experiment II. The same primary leaf of each plant was used for each pair of readings at each time interval in each experiment.

Statistical analysis. Data within an experiment were combined for statistical analysis by a Student's *t*-test (13). Significance at the $\alpha = 0.05$ level was selected to reject the null hypothesis that the mean conductance of the controls was equal to that of the exposed plants.

Histological studies. Fifteen seedlings were cultured and exposed to PAN for 1 hr as in experiment II. Tissue samples from one primary leaf per plant were taken from each of five plants at 3, 6, and 9 hr after PAN exposure. Two control plants were sampled 3 and 6 hr after exposure. Samples of leaf tissue, 1 × 3 mm, were prepared for sectioning as described (9,11). Sections, 2 μm thick, were cut with glass knives at 40-μm intervals and stained with 1% toluidine blue in 1% sodium borate and examined with a light microsope. Twelve sections per tissue block from each exposed and control plant were studied. For each section examined, the total number of epidermal and spongy mesophyll cells were counted and the number of each cell type exhibiting collapse was determined. In addition, the position of the cell; viz, adjacent or random with respect to a guard cell, was noted. The percentage of cells injured at each location for each time period studied was calculated.

Mesophyll and epidermal cells were classified as either adjacent or random with regard to proximity of stomata. Adjacent mesophyll cells were those cells which could be connected to a stoma by a straight line without intersecting another cell. All other mesophyll cells were designated as random. Adjacent epidermal cells were defined as any injured cell having no uninjured cell between it and a guard cell. All other injured epidermal cells were classified as

TABLE 4. Number of mesophyll cells injured in primary leaves of *Phaseolus vulgaris* 'Provider' after plants were exposed to air containing 405 $\mu g/m^3$ peroxyacetyl nitrate (PAN) for 1 hr

Postexposure time (hr)	Cell location[a]	Cells (no.) Counted	Cells (no.) Injured	Injured cells[b]
3	Adjacent	1,112	0	0 ± 0[c]
	Random	8,112	7	0.10[d] ± 0.30
6	Adjacent	881	377	42.86 ± 21.80
	Random	5,862	1,202	20.55 ± 7.37
9	Adjacent	456	441	96.08 ± 5.93
	Random	2,532	1,562	59.04 ± 14.34

[a]Adjacent = cells which could be connected to a stomate by a straight line without intersecting another cell; random = all other mesophyll cells.
[b]Mean of the percent injured cells per section of 48, 36, and 24 sections at 3, 6, and 9 hr, respectively.
[c]Standard deviation.
[d]Computed only for sections having injured cells.

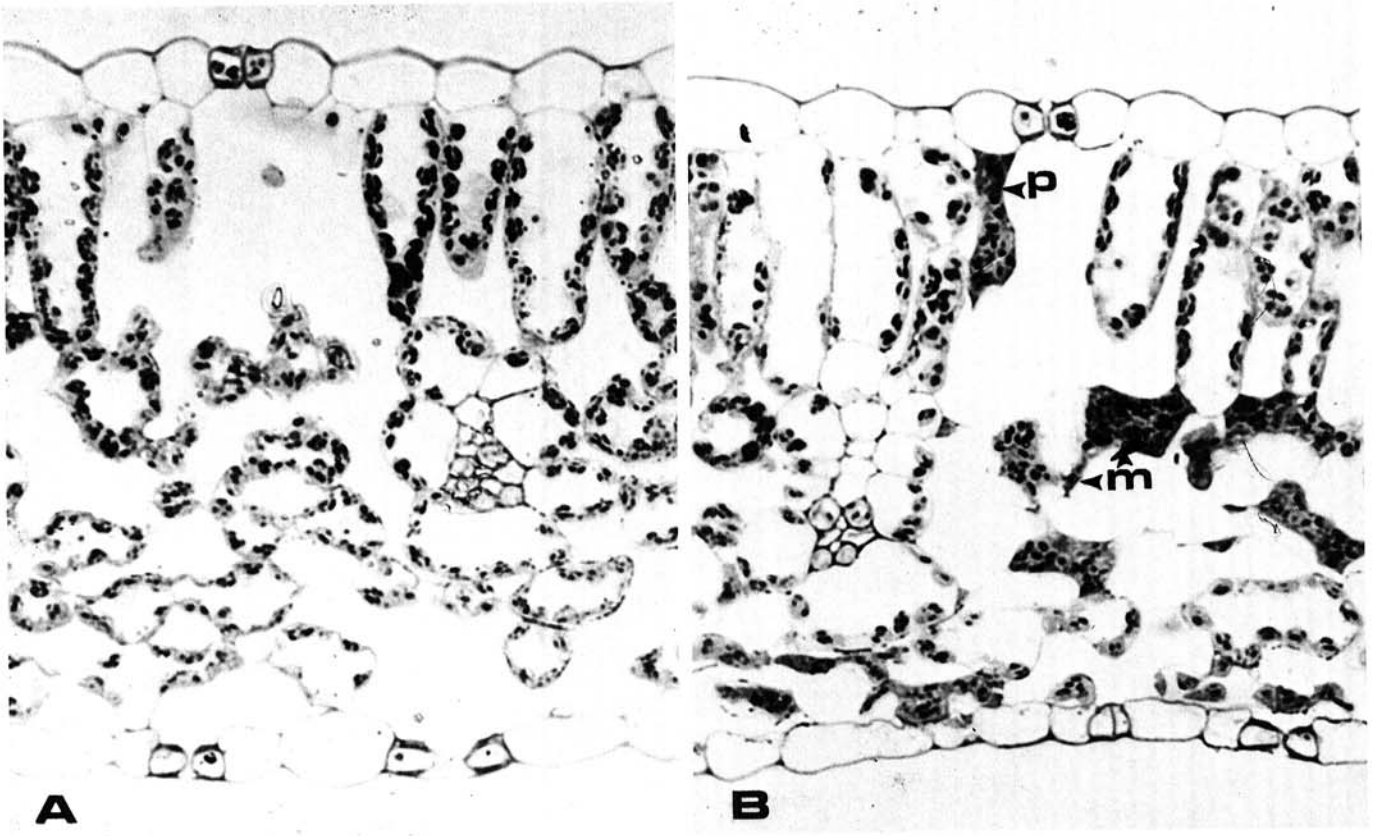

Fig. 1. Histological effects of peroxyacetyl nitrate (PAN) in cross sections of primary leaves of *Phaseolus vulgaris* 'Provider' exposed to air containing 405 $\mu g/m^3$ PAN or charcoal-filtered air for 1 hr. **A,** Control, exposed to charcoal-filtered air (× 1,934). **B,** Six hours after PAN exposure. Note collapsed spongy mesophyll cells (m), and a collapsed palisade parenchyma cell (p) (× 1,716). **C,** Nine hours after exposure. Note the collapsed cells in the abaxial epidermis (a) and the collapsed spongy mesophyll cells (m) (× 1,650).

random. Therefore, only injured epidermal cells could be classified as adjacent or random and an absolute number of total adjacent and random epidermal cells could not be determined. The percentage of total injured mesophyll and epidermal cells may be compared, but not the percentage of injured adjacent and random cells of each type.

Forty-eight hours after exposure to PAN, macroscopic injury was noted on the companion primary leaf which was not sampled for microscopic evaluation. Only the data collected from sections of exposed plants in which the companion primary leaf exhibited macroscopic PAN injury were used in calculating the percentages of injured cells at each location for each time period.

RESULTS

Leaf conductance. *I. Subthreshold.* None of the plants exposed to 271 $\mu g/m^3$ PAN for 1 hr developed macroscopic symptoms. There were no significant differences in conductance between the control and PAN-treated plants for either leaf surface at any time tested after exposure.

II. Split threshold. None of the plants that received 405 $\mu g/m^3$ PAN for 0.5 hr developed macroscopic injury, whereas all plants exposed to that concentration for 1 hr displayed macroscopic symptoms 48 hr after exposure. Symptoms were glazing or bronzing of the abaxial leaf surface. No significant difference in conductance was found between the nonexposed plants and those exposed to 405 $\mu g/m^3$ for 0.5 hr. Alteration of leaf conductance in plants receiving 405 $\mu g/m^3$ for 1 hr was first apparent 9 hr after treatment (Table 1). The conductance of the exposed plants was signficiantly greater than that of nonexposed plants during the night measurement and significantly lower during the day reading. This difference was apparent for both abaxial and adaxial surfaces but was more pronounced for the abaxial surface.

III. Threshold. Three exposures were conducted at 360 $\mu g/m^3$ PAN for 1.0 hr. In two of these exposures, all of the plants developed

TABLE 5. Number of epidermal cells injured in primary leaves of *Phaseolus vulgaris* 'Provider' after plants were exposed to air containing 405 $\mu g/m^3$ peroxyacetyl nitrate (PAN) for 1 hr

Postexposure time (hr)	Cells (no.)					
	Counted (no.)	Injured[a]		Injured cells[b]		
		Adjacent	Random	Total	Adjacent	Random
3	3,135	0	2	0.04 ± 0.20[c]	0	4.00[d] ± 14.43
6	2,671	933	239	43.50 ± 18.05	77.47 ± 27.75	22.50 ± 27.70
9	1,176	522	118	56.41 ± 16.74	82.66 ± 23.35	17.33 ± 23.37

[a] Adjacent = any injured cell having no uninjured cell between it and a guard cell. Random = all other injured cells.
[b] Mean of the percent of injured cells per section of 48, 36, and 24 sections at 3, 6, and 9 hr, respectively.
[c] Standard deviation.
[d] Computed on only those sections having injured cells.

macroscopic injury and the conductance data from these two exposures were pooled for analysis (Table 2). Beginning 9 hr after PAN exposure, the conductance of the abaxial surface of the exposed plants was significantly higher at night and lower for the day readings than that of the controls. Conductance of the adaxial surface of controls was higher than that of the exposed plants at +33 hr. In the third threshold exposure five plants displayed macroscopic symptoms and 10 did not. The conductance values of each group were compared to the 15 controls and to each other (Table 3). Immediately after the exposure to PAN, the plants that would later display macroscopic symptoms had significantly greater leaf conductance on the abaxial surface than did exposed plants that would remain asymptomatic. After 24 hr, the leaf conductance of the abaxial surface of injured plants was significiantly lower than that of uninjured or control leaves.

Histological effects. Representative cross sections of control and injured leaves are illustrated in Fig. 1. All cells are fully turgid in the unexposed leaf tissue (Fig. 1A). Incipient cell injury which occurs 6 hr after exposure to PAN is depicted in Fig. 1B. The spongy mesophyll exhibited several stages of collapse with those injured cells closest to the abaxial epidermis appearing to be more severely collapsed than those farther removed. A collapsed palisade parenchyma cell adjacent to an adaxial stomate also is shown. Injured palisade parenchyma cells were rare and always occurred adjacent to adaxial stomata. At this time abaxial epidermal cells also were showing signs of injury, as evidence by a loss of turgor. All injured spongy mesophyll and epidermal cells appeared to be collapsed 9 hr after exposure (Fig. 1C).

Guard cells appeared uninjured and intact (Fig. 1B, C). Only four of 1,222 abaxial guard cells appeared to be injured. At no time was injury to an adaxial guard cell or epidermal cell noted. In the case of unexposed control tissue (Fig. 1A), no injury was noted in a total of 9,151 mesophyll and 3,681 epidermal cells counted.

Less than 1% of epidermal and mesophyll cells showed symptoms of injury 3 hr after exposure to PAN (Tables 4 and 5). Six hours after exposure to PAN, 24% of the mesophyll and 44% of the epidermal cells were injured. While the mean percentage of injured epidermal cells appeared to be twice that of the percent mesophyll cells injured 6 hr after exposure, the standard deviations showed that the percentage of injured cells in both tissues are statistically similar. Nine hours after exposure, 63% of the mesophyll and 56% of the epidermal cells were collapsed. In both tissue types, larger percentages of adjacent cells than random cells showed injury 6 and 9 hr after exposure (Tables 4 and 5).

DISCUSSION

When PAN caused macroscopic symptoms in primary bean leaves, leaf conductance increased at night and decreased during the day. In a previous report, Dugger and Ting (4) noted a net increase in transpiration of bean plants in response to above-threshold doses of PAN. Starkey (12) reported that bean plants exposed to subthreshold doses of PAN exhibited accelerated wilting, a more rapid decrease in soil moisture, and a more negative water potential. We did not observe any change in conductance in plants receiving subthreshold doses of PAN. Many factors could explain the seeming difference in response reported here and elsewhere (12). The difference in cultural conditions may best explain the altered response. Our plants were grown in sand culture and fertilized with Hoagland's solution while Starkey (12) used an amended soil mixture. An alternate explanation for the apparently different plant response in Starkey's (12) research and our own could relate to genetically determined plant tolerance. While the seed source in both studies was the same, the seed lots were different. Since pinto bean plants are not selected for PAN tolerance, variation in susceptiblity from lot to lot is possible. The impact of PAN on water status of foliage in the absence of macroscopic symptoms remains unclear. It is apparent that when PAN induces foliar symptoms, plant water relations change.

The change in water relations may be related to the observed histological changes. We believe that the diurnal cycle in conductance was maintained, but at a reduced amplitude. Whether the function of some guard cells is completely inhibited or all are partially inhibited cannot be determined histologically. However, guard cells appeared to be intact and not injured when viewed with a light microscope (Fig. 1B, C). It is possible that the altered conductance is related to the collapse of epidermal and spongy mesophyll cells adjacent to guard cells. The collapse of these cells would inhibit the normal exhange of water necessary for guard cell function; cellular collapse also would alter the availability of fluid to the transpiration stream. It is noteworthy that alterations in leaf conductance become apparent 9 hr after PAN exposure when histological injury has progressed to collapse. The sequence of altered structure and function supports but does not prove casuality.

The larger number of cells injured adjacent to stomata is consistent with previous reports (5,18) and logical since this pore is the major site of entry of PAN into the leaf (2). An occasional palisade parenchyma cell, adjacent to a stomate, collapsed in response to PAN, but it is unlikely that injury to so few cells could have any functional impact on the plant. The statistically insignificant change in adaxial conductance following trends of abaxial results would then be related to injury other than to the palisade parenchyma. It is likely that this insignificant change in adaxial conductance would lack biological importance.

LITERATURE CITED

1. DRUMMOND, D. B. 1972. The effect of peroxyacetyl nitrate on petunia (*Petunia hybrida* Vilm.). Pa. State Univ., Center Air Environment Studies (CAES), Pub. No. 260-72, 70 pp.
2. DUGGER, W. M., Jr., O. C. TAYLOR, E. CARDIFF, and C. R. THOMPSON. 1962. Stomatal action in plants as related to damage from photochemical oxidants. Plant Physiol. 37:487-491.
3. DUGGER, W. M., Jr., O. C. TAYLOR, W. H. KLEIN, and W. SHROPSHIRE, Jr. 1963. Action spectrum of peroxyacetyl nitrate damage to bean plants. Nature 198:75-76.
4. DUGGER, W. M., and I. P. TING. 1968. The effect of peroxyacetyl nitrate: Photoreductive reactions and susceptibility of bean plants to PAN. Phytopathology 58:1102-1107.
5. GLATER, R. A. 1970. Smog and plant structure in Los Angeles County. U.C.Los Angeles, School of Engineering, Applied Sci. Rep. 70/17. 39 pp.
6. HINDAWI, R. J. 1970. Air pollution injury to vegetation. Nat. Air Pollut. Control Assoc. (NAPCA) Publ. AP-71. 16-17.

7. HOAGLAND, D. R., and D. I. ARNON. 1950. The water culture method for growing plants without soil. Calif. Agric. Exp. Stn. Circ. 347.
8. KOHUT, R. J. 1975. The interaction of O_3 and PAN on hybrid poplar and pinto bean. Pa. State Univ., Center for Air Environment Studies (CAES) Publ. 428-76. 60 pp.
9. PELL, E. J., and W. C. WEISSBERGER. 1976. Histopathological characterization of ozone injury to soybean foliage. Phytopathology 66:856-861.
10. SMITH, R. A., R. J. BRYAN, M. FELDSTEIN, B. LEVADIE, F. A. MILLER, and E. R. STEPHENS. 1972. Tentative method of analysis for peroxyacetyl nitrate (PAN) in the atmosphere (gas chromatographic methods). Pages 215–219 in: Methods of air sampling and analysis. Am. Public Health Assoc., Washington, DC.
11. SPURR, A. R. 1969. A low-viscosity epoxy resin embedding medium for electron microscopy. J. Ultrastruct. Res. 26:31-43.
12. STARKEY, T. E. 1975. The influence of peroxyacetyl nitrate on bean (*Phaseolus vulgaris* L.) subjected to post-exposure water stress. Pa. State Univ., Center Air Environment Studies (CAES) Pub. 400-75. 45 pp.
13. STEELE, R. G. D., and J. H. TORRIE. 1960. Principles and Procedures of Statistics. McGraw-Hill, New York, NY. 481 pp.
14. STEPHENS, E. R. 1964. Absorptivities for infrared determination of peroxyacetyl nitrates. Anal. Chem. 36:928-929.
15. STEPHENS, E. R. 1965. The production of pure peroxyacetyl nitrates. J. Air Pollut. Control Assoc. 15:87-89.
16. TAYLOR, O. C. 1969. Importance of peroxyacetyl nitrate (PAN) as a phytotoxic air pollutant. J. Air Pollut. Control Assoc. 19:347-351.
17. TAYLOR, O. C., W. M. DUGGER, Jr., E. A. CARDIFF, and E. F. DARLEY. 1961. Interaction of light and atmospheric photochemical products (smog) within plants. Nature 192:814-816.
18. THOMSON, W. W., W. W. DUGGER, Jr., and R. L. PALMER. 1965. Effects of peroxyacetyl nitrate on ultrastructure of chloroplasts. Bot. Gaz. 126:66-72.
19. TURNER, N. C., F. C. FIND, and W. H. WRIGHT. 1969. An aspirated diffusion porometer for field use. Conn. Agric. Exp. Stn. Soils Bull. 29:1-7.
20. TURNER, N. C. and J. Y. PARLANGE. 1970. Analysis of operation and calibration of a ventrilated diffusion porometer. Plant Physiol. 46:175-177.
21. WOOD, F. A., D. B. DRUMMOND, R. G. WILHOUR, and D. D. DAVIS. 1973. An exposure chamber for studying the effects of air pollution on plants. Pa. State Univ., Agric. Exp. Stn. Prog. Rep. 335. 7 pp.