Smog DAMAGE TO COTTON

O. C. TAYLOR • J. D. MERSEREAU

EXTENSIVE COTTON PRODUCING areas of California suffer from air pollution. There is reason to believe that this problem will increase in severity and extent with increases in industrial and urban development. Premature aging and dropping of leaves of susceptible plants are known to be caused by natural and experimental, photochemically produced, compounds in smog. Ozone and peroxyacetyl nitrate (PAN), two known phytotoxicants in smog, have been demonstrated to produce such leaf separation under laboratory conditions.

To investigate the potential hazard of these two agents, cotton plants grown in pots under greenhouse conditions were exposed to atmospheres containing known concentrations of manufactured ozone or PAN. Activated charcoal filters on the greenhouse removed ozone, PAN, and other pollutants from the air supplied to cotton plants during the entire pretreatment period. Exposure to air pollutants was started when the plants had developed two or three squares and in some instances when one or more bolls had formed. Concentrations of ozone used

Photo 1, left, shows chlorosis and suppression of leaf expansion on cotton exposed to 0.25 to 0.35 ppm ozone 9 hours a day for 12 days. Dark green thrifty plant received activated carbon filtered air. Photo 2, right, shows leaf drop 10 days after exposure to 0.25 to 0.35 ppm ozone 9 hours a day for 12 days. Companion plant is from activited carbon filter treatment.

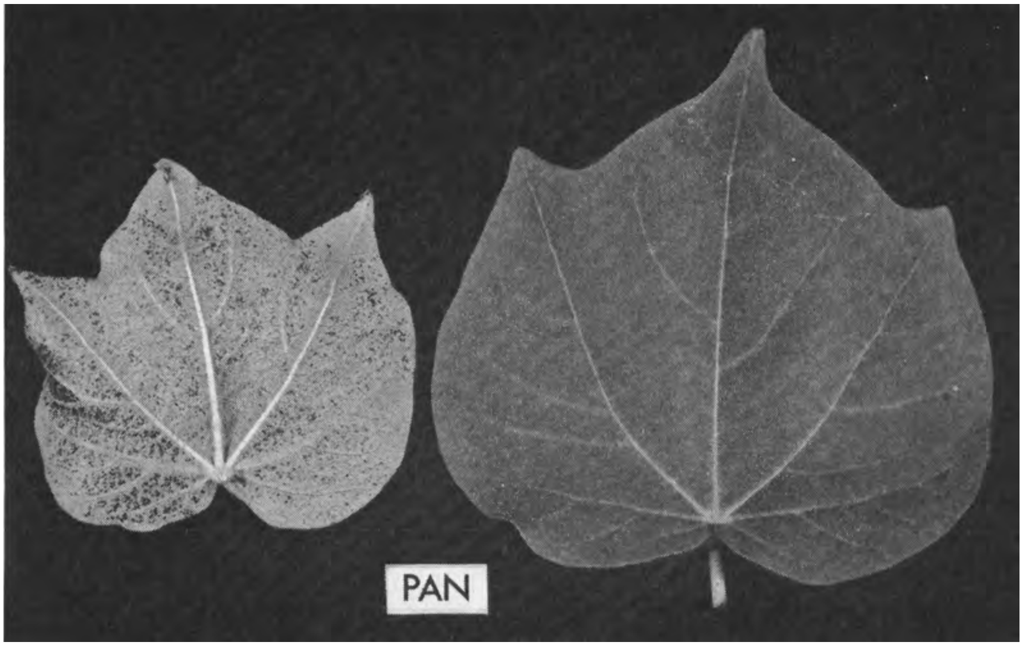

Photo 3. Young expanding cotton leaves exposed to 1.0 ppm PAN for 2 hours. Left—Glazing and bronzing appears on lower surface. Right—Upper surface is not damaged.

(0.25 to 0.35 parts per millon for seven hours a day), were sufficient to cause moderate to heavy damage on sensitive vegetable crops such as tomatoes, beans and lettuce. PAN concentrations used in these experiments (0.5 to 1.0 ppm for one to two hours), were sufficient to cause severe damage to these same vegetable crops.

The effects of ozone on the cotton plants appeared gradually as the experiment progressed. The first evidence of chlorosis on the oldest leaves appeared about the fifth to seventh day, depending on the ozone concentration being used. The higher the concentration, the shorter the time required for symptoms to appear. As the fumigations continued, yellowing increased and became evident on successively younger leaves as shown in photo 1. Ultimately the yellowed leaves dropped leaving only young immature leaves at the top of the plant (photo 2). Dropping of the oldest leaves started on the eighth to tenth day and continued for a week or 10 days after the 12-day experiment ended. Necrotic lesions usually surrounded by a red area were evident on the upper surface of many leaves of plants exposed to ozone concentrations above about 0.35 ppm. Similar symptoms on the upper leaf surface are characteristic of ozone damage on grapes, avocado, citrus and, under some conditions, on bean leaves.

Severe burning and decay of cotton leaves occurred when they were exposed to 1.0 ppm ozone for seven hours. This damage did not resemble any of the symptoms observed in the field.

The high concentrations of PAN for short exposures, 1.0 ppm for two hours or 0.5 ppm for four hours, caused a glazing on the lower surface of young expanding leaves (photo 3). The glazed areas subsequently become bronzed and decayed. Facilities were not available to produce the quantities of PAN required to investigate its effect during a long term, low-concentration exposure.

Leaf symptoms indistinguishable from those produced experimentally by ozone have been observed on cotton near Bakersfield, Indio and in experimental field plots at Riverside. Similar symptoms occur on cotton in the vicinity of Phoenix, Arizona. These occurrences indicate that cotton growers face increasing losses from stunted growth, and possibly premature drop of bolls and squares, wherever the inroads of photochemical smog are occurring.

O. C. Taylor is Associate Horticulturist in the Experiment Station and J. D. Mersereau is Laboratory Technician, University of California, Riverside.

MOSAIC DISEASE OF PASSION VINE

A VIRUS DISEASE of ornamental passion vines (*Passiflora caerulea* and *P. alato-caerulea*) in northern California is characterized by stunting of the plant, and mottling and distortion of the leaves. The disease is now known to be caused by cucumber mosaic virus.

Transmission of the virus from passion vines to cucumber and cowpea plants has been effected mechanically by brushing the juice from macerated leaves onto the leaves of the indicator plants with Carborundum as an abrasive. Symptoms of infection in the indicator plants resembled those usually produced by common strains of cucumber mosaic virus. Juice from macerated leaves of mosaic-diseased cucumber plants reproduced the mosaic disease in *Passiflora caerulea* when brushed over the leaves.

The virus from passion vine was also transmitted successfully from diseased to healthy cucumber plants by the green peach aphid (*Myzus persicae*). Aphids acquired the virus after a relatively short feeding time of 15 to 20 seconds. The infectivity of the aphids decreased rapidly, a fact which indicates that the virus is of the nonpersistent type in this insect vector. Further confirmation of the identity of the virus was obtained with serological tests, in which antiserum for cucumber mosaic virus gave positive reactions with sap from diseased plants that had been inoculated with virus from passion vine.

Cucumber mosaic virus is known to have a wide host range in ornamental and crop plants. Under natural conditions, therefore, it is believed that aphids may act as vectors which transmit the virus from other host plants to passion vine.

In countries such as Australia, where *Passiflora edulis* is grown commercially for its fruit, cucumber mosaic is one of the viruses which cause the "woodiness disease." This economically important disease is so named because the rind of the fruit becomes woody and thick, leaving little space for the edible pulp. Yields are further reduced because infected plants produce fewer fruits.—*D. S. Teakle, Miller Fellow; C. C. Gill, Research Assistant; R. D. Raabe, Associate Professor, Department of Plant Pathology, University of California, Berkeley; and R. H. Taylor, Senior Pathologist, Department of Agriculture, Victoria, Australia, on leave at Department of Plant Pathology, U. C., Davis. The antiserum was provided by R. G. Grogan and K. A. Kimble, Department of Plant Pathology, U.C., Davis.*