

Response of Potassium-Deficient Tomato Plants to Atmospheric Ozone

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ABSTRACT

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Visible injury caused by ozone was reduced in potassium-deficient Rutgers tomato plants. Injury reduction paralleled increased foliage resistance as indicated by porometer

measurements. Exposure to ozone was followed by increased foliar potassium content in both normal and potassium-deficient plants.

Additional key words: air pollution, ozone injury, potassium deficiency, stomatal resistance, *Lycopersicon esculentum*.

The tendency for plants deficient in a particular nutrient element to be less sensitive to air pollutants than optimally grown plants has been reported (5). This has been attributed in part to the development of increased stomatal resistance when plants were under stress (5). Since potassium has been revealed to be directly involved in stomatal activity (1, 2, 3, 4), a study was conducted to determine the effect of potassium deficiency on the response of tomato plants to atmospheric ozone and to relate the response with stomatal resistance.

MATERIALS AND METHODS

Seeds of tomato (*Lycopersicon esculentum* L. 'Rutgers') were germinated in moist vermiculite and, at the appearance of the first true leaves, seedlings were transplanted to washed quartz sand in 17.5-cm diameter plastic pots. The seedlings were maintained in a charcoal-filtered greenhouse and given 200 ml of distilled water daily for 3 days and 200 ml of half-strength nutrient solution for five additional days. On the 9th day, full-strength nutrient treatments were applied (300 ml every other day with distilled water on alternate days) and continued for 30-37 days until ozone treatment. The low-K treatment consisted of 19.5 mg/liter potassium and the complete treatment of 175.0 mg/liter potassium with all other macro- and micro-nutrients at optimal levels (5).

Ozone exposures were conducted in a glass-walled chamber as previously described (5). Temperature, relative humidity, and light were maintained at 30 C, 50%, and 186 lux, respectively. Plants were placed on a revolving turntable within the chamber with four replicates per treatment and exposed to ozone concentrations of 300 (0.15 ppm), 400 (0.20 ppm), 500 (0.25 ppm), or 600 (0.30 ppm) $\mu\text{g}/\text{m}^3$ for 3 hours. Ozone concentration was monitored continuously with a Mast ozone meter. Control plants were placed in a chamber supplied with charcoal-filtered air during the same period. Ozone exposures were conducted on sunny days from 0900 to 1200 hours. After exposure, all plants were

returned to the charcoal-filtered greenhouse and observed for injury for the next 48 hours.

All leaves on each plant were rated for ozone symptoms as follows: 0 = absence of injury, 1 = injury involving less than 15% of leaf, 2 = 15-25% injury, 3 = 30-65% injury, and 4 = 70% or more injury. An average rating then was calculated for each plant.

Measurement of leaf resistance by means of a Lambda diffusive resistance meter was generally begun at 1030 hours to coincide with the period of optimal stomatal activity. Readings on ozone-treated plants and their controls were made once from 1300 to 1500 hours on the same day. Duplicate readings were made on either surface of the first two lateral leaflets on the fourth oldest leaf of each plant and reported as $\text{sec}\cdot\text{cm}^{-1}$.

Plants were harvested 48 hours after exposure. Fresh weights of leaves were recorded and the leaves were minced, dried at 21 C in a forced-draft oven, and ground through the 420- μm (40-mesh) screen of a semimicro Wiley Mill for potassium determinations. Potassium analyses were made on dry-ashed tissue samples taken up in HCl by atomic absorption techniques (7) and reported as percentage of dry weight. Data were analyzed statistically by Student's *t* test.

TABLE 1. Foliage injury resulting from 3-hour exposures to varying ozone concentrations on tomato plants grown at low potassium or complete nutrient levels

O ₃ Concn. ($\mu\text{g}/\text{m}^3$)	Ozone injury rating ^a	
	Nutrient treatment	
	Low-K	Complete
300	0.22	1.23
400	0.53	1.97
500	1.59	2.27
600	2.23	2.38

^aFoliage injury rating scale: 0 = no visible injury; 1 = < 15% of leaf injured; 2 = 15-25% injury; 3 = 30-65% injury; and 4 = > 70% injury. Each value is the mean of eight replicates.

RESULTS

After 30 days of nutrient treatment, plants receiving 19.5 mg/liter potassium (low-K) exhibited the stunting and mottled yellow leaf symptoms associated with potassium deficiency. Foliage yield of low-K plants averaged 36.7 g as compared with 77.4 g for plants grown at the complete nutrient level.

Injury consisted of the upper surface bleaching usually associated with ozone exposure. The extent of injury (Table 1) increased with increase in ozone concentration regardless of potassium level. However, at the lower ozone concentrations (up to 400 $\mu\text{g O}_3/\text{m}^3$), injury on the potassium-deficient plants was greatly reduced compared with that on plants that received a complete nutrient solution. At an ozone concentration of 600 $\mu\text{g}/\text{m}^3$ there was no difference in visible injury between the two groups.

Data in Table 2 reveal a statistically significant increase in stomatal resistance in the potassium-deficient plants over the normal ones for both leaf surfaces. A comparison of resistance of ozone treated vs. control leaves revealed that in the treated plants, resistance was significantly decreased ($P = 0.05$) in the low-K plants, whereas there was no significant difference between treatments among those receiving complete nutrient solutions.

TABLE 2. Stomatal resistance of fourth oldest leaf of control and ozone-treated tomato plants grown at low potassium or complete nutrient levels

	Stomatal resistance ^a (sec-cm ⁻¹)			
	Nutrient treatment			
	Low-K		Complete	
	Control	O ₃ -treated	Control	O ₃ -treated
Lower surface	10.2* ^b	8.4*	4.2	4.8
Upper surface	14.5*	...	10.0	...

^aEach control value is the mean of four readings on each of twenty plants. Each value under O₃-treated is the mean of four readings on each of four plants.

^bAsterisk (*) indicates significant difference between low-K and complete at $P = 0.01$.

Leaf potassium content (Table 3) varied in both low-K and complete nutrient plants according to the time of year, with the highest content occurring in plants harvested in August. Despite seasonal variations, there was a significantly increased potassium content in plants at both potassium levels as a result of ozone exposure. However, there was no relationship between the magnitude of potassium increase and ozone level.

DISCUSSION

Results of this investigation indicate that in tomato plants, potassium deficiency causes reduced ozone toxicity. Induced ozone tolerance was directly related to stomatal resistance, which was greater by two- to four-fold in the low-K plants than in the normal ones. These results conform to those of Fischer (1, 2), of Graham and Ulrich (3), and of Humble and Hsiao (4), all of whom have demonstrated that stomatal opening in leaves of various species is due to active accumulation of potassium by guard cells.

Results of chemical analyses indicating a significantly increased K content in plants as a result of ozone exposure corroborated evidence to this effect reported earlier (5). The validity of the present data seems to be upheld by the fact that increased K-content in the low-K treatment was accompanied by decreased stomatal resistance on the single occasion when such data were available for comparison.

Finally, Peaslee and Moss (6) contend that reduced CO₂ uptake rates in K-stressed leaves may be attributed to stomatal closure. If stomatal closure, induced by withholding potassium, can interfere with CO₂ uptake there is no reason why uptake of other atmospheric gases also may not be interdicted.

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TABLE 3. Leaf potassium content of control and ozone-treated tomato plants grown at low-K or complete nutrient levels

O ₃ Concn. ($\mu\text{g}/\text{m}^3$)	Plant age (days)	Leaf potassium after treatment ^a % of dry weight					
		Nutrient Treatment					
		Low-K			Complete		
		Control	O ₃ -treated	Increase (%)	Control	O ₃ -treated	Increase (%)
300	35	0.58	0.89* ^b	152	2.38	2.84** ^c	121
400	31-35	0.84	1.20*	135	1.94	2.55**	131
500	33	0.58	1.02*	180	2.35	2.88**	122
600	25	0.85	0.98*	115	2.39	2.83**	118

^aEach value is the mean of four to eight replicates.

^bAsterisk (*) indicates significant difference between control and O₃-treated at $P = 0.05$.

^cDouble asterisk (**) indicates significant difference between control and O₃-treated at $P = 0.01$.

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