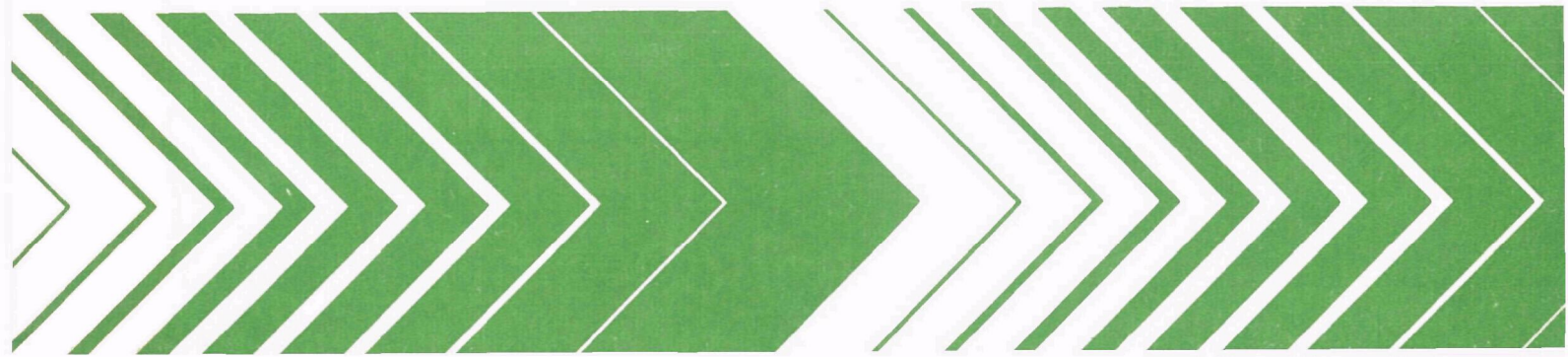




Nitrogen Dioxide

Time-Concentration Model to Predict Acute Foliar Injury



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NITROGEN DIOXIDE: TIME-CONCENTRATION MODEL
TO PREDICT ACUTE FOLIAR INJURY

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FOREWORD

Effective regulatory and enforcement actions by the Environmental Protection Agency would be virtually impossible without sound scientific data on pollutants and their impact on environmental stability and human health. Responsibility for building this data base has been assigned to EPA's Office of Research and Development and its 15 major field installations, one of which is the Corvallis Environmental Research Laboratory (CERL).

The primary mission of the Coravllis Laboratory is research on the effects of environmental pollutants on terrestrial, freshwater, and marine ecosystems; the behavior, effects and control of pollutants in lake and stream systems; and the developmnt of predictive models on the movement of pollutants in the biosphere.

This report contains previously unpublished research results that were originally planned for publication in BioScience in 1971. This reference was used in the 1971 Air Quality Criteria document for Nitrogen Oxides (Chapter 8, ref. 8). Data from Taylor was deleted from the present manuscript. The authors are publishing these results because they still represent the best and most extensive dose-response information for nitrogen dioxide on vegetation. The research was completed in 1968. The authors believe this information should be available to the scientific community.

James C. McCarty
Acting Director, CERL

ABSTRACT

An understanding of the response of plant species to specific doses of an air pollutant or a group of pollutants is essential before air quality standards can be established in areas under crop production. Selected plant species were exposed to two nitrogen dioxide concentrations chosen to produce threshold and severe injury at five time periods ranging from 0.5 to 7 hr. From these data for each species, an equation was developed using concentration as the dependent variable, and foliar injury and time as independent variables. The model allows for the development of a three-dimensional response surface within the limits of the exposure times and concentrations used. The model is useful in predicting the concentration of nitrogen dioxide that will produce a given amount of injury to a specific crop during a single 12-hr day. Research to date suggests the model may be used for other pollutants and for other plant species that show injury following exposure to high ambient nitrogen dioxide concentrations.

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SECTION 1

INTRODUCTION

The introduction of nitrogen dioxide as an air pollutant was first inferred in California when photochemical oxidants were shown to adversely affect vegetation. Through the photochemical production of atmospheric oxidants, including peroxyacetyl nitrate (PAN) and ozone, nitrogen dioxide exerts a secondary, though significant effect on vegetation. At ambient concentrations, the direct effects of nitrogen dioxide on vegetation are more difficult to assess except around localized sources.

Glaser (1970) stated that increasing ambient levels of nitrogen dioxide may have been responsible for some of the chronic injury to vegetation which was prevalent in the Los Angeles Basin. The increase of nitrogen oxides was attributed to the increase in emissions from motor vehicles whose control devices reduced hydrocarbons and carbon monoxide but failed to reduce nitrogen oxides. Glaser suggested that injury from nitrogen dioxide was replacing the PAN-type of injury. The symptoms discussed by Glaser, however, were not found by Taylor and Eaton (1966) after exposing plants for several days to low concentrations of nitrogen dioxide. The nitrogen dioxide injury (reported by Glaser) is similar to that reported from low-level, controlled ozone exposures and/or high ambient oxidant concentrations in eastern urban areas by the authors of this paper and others. The responses reported by Glaser probably are more related to ozone or to pollutant interactions than to the nitrogen oxides alone.

Direct plant responses to phytotoxic levels of nitrogen dioxide can be divided into three broad categories: physiological, chronic and acute injury. Physiological injury includes growth alterations, reduced yields (Taylor and Eaton, 1966) and reduced photosynthesis (Hill and Bennett, 1970). Chronic injury results from intermittent low level exposure over long time periods. Chronic injury produces chlorotic and/or other pigmented patterns in leaf tissues and may be accompanied by an increase in leaf drop (Glaser, 1970; Thompson et al , 1970). Acute foliar injury often resembles the intercostal bifacial necrosis associated with the response of plants to sulfur dioxide. High nitrogen dioxide concentrations produce foliar markings that first appear as water-soaked areas and may develop into white, tan, brown, or bronze necrotic lesions. Lesions, although normally intercostal, are often marginal and more toward the leaf apex. A discoloration of leaves with a waxy appearance has been reported for several weed species (Benedict and Breen, 1955). Short exposures (measured in hours) to high levels of nitrogen dioxide may produce acute symptoms within 2 to 48 hr after exposure. Two publications give good descriptions of nitrogen dioxide injury and include colored plates showing acute injury (Taylor and MacLean, 1970; van Haut and Stratmann, 1967).

Investigators have reported plant responses to several time-concentration combinations of nitrogen dioxide, but not for the express purpose of generalizing their data to develop predictive equations. Several reports in the literature are of value, however, and will be summarized. The threshold concentration for visible injury to pinto bean was 3 to 4 ppm in an 8-hr exposure; a 2-hr exposure at 30 ppm killed the leaves (Middleton et al., 1958). A group of agricultural and horticultural crops exposed to 30 ppm nitrogen dioxide for 1 hr developed little or no injury (Czech and Nothdurft, 1952). Exposure of 10 weed species to 20 or 50 ppm nitrogen dioxide for 4 hr during midday caused from zero to medium injury on leaves of well-watered plants; only mustard showed well defined injury (Benedict and Breen, 1955). Sixty plant species were exposed to 2.5 to 10 ppm nitrogen dioxide for 4 to 8 hr and compared with plants exposed to similar levels of sulfur dioxide; the plants were approximately 2.5 times less sensitive to the nitrogen dioxide (van Haut and Stratmann, 1967). Cotton, pinto bean and endive were slightly injured at 1 ppm nitrogen dioxide for 48 hr, were uninjured at 0.5 or 2.0 ppm for 21 hr or at 1.0 ppm for 12 hr, while 3.5 ppm for 21 hr produced slight injury to cotton and pinto bean and death of endive leaves (Heck, 1964). Tobacco was uninjured at 2.3 ppm for 8.7 hr; pinto bean was not injured until exposed for 4 hr to 10 ppm (Taylor and Eaton, 1966). Navel orange trees continuously exposed to 0.06, 0.12, 0.25, 0.5 and 1.0 ppm nitrogen dioxide showed extensive chronic injury after 35 days at the two highest concentrations. Increased leaf drop and reduced yields occurred with the 0.25 ppm treatment after 8 months (Thompson et al., 1970). Fourteen ornamental species and six citrus varieties were exposed to from 10 to 250 ppm for periods of 0.2 to 8 hr with extensive injury reported in all plants; young shoot necrosis occurred at exposures of 200 ppm for 4 to 8 hr or 250 ppm for 1 hr (MacLean et al., 1968).

The degree of injury to vegetation by nitrogen dioxide is influenced by factors such as plant species, stage of plant development, plant environment (temperature, light, humidity, soil moisture, mineral nutrition), variable susceptibility within species (cultivar or clone), and the interaction of more than one phytotoxic gas in the plant atmosphere (Heck, 1968). Depending upon the kind of plant and its environment, one factor may be more important than another, but all factors must eventually be understood before adequate effects modeling can be completed.

Taylor and MacLean (1970) recognized the increased sensitivity of plants to nitrogen dioxide caused by low light intensity. They reported that 2-hr exposures of sensitive plants to 3 ppm nitrogen dioxide under low light conditions caused as much injury as 6 ppm in light equivalent to full sunlight. Several reports have shown that night exposures to nitrogen dioxide may cause more injury than day exposures (Czech and Nothdurft, 1952; Tingey, 1969; van Haut and Stratmann, 1967). van Haut and Stratmann (1967) reported that rye plants were most sensitive to nitrogen dioxide from noon to 4 pm. Oats had a bimodal sensitivity to nitrogen dioxide with more injury occurring from mid-night to 2 am than at noon to 2 pm.

The report by Tingey et al. (1971) is of special interest. They exposed six species to various mixtures of nitrogen dioxide and sulfur dioxide for 4 hr. They found that over 2.0 ppm of nitrogen dioxide or 0.5 ppm of sulfur

dioxide were required to produce injury when the gases were administered separately, but all species were injured by various combinations of the two gases in the concentration ranges of 0.05 to 0.25 ppm. The relative sensitivity of the six species from highest to least sensitive was soybean, radish, pinto bean, oats, tobacco, and tomato. Injury developed as a chlorotic or necrotic flecking on the upper leaf surface and was similar to injury produced by ozone.

This paper reports experiments designed to more accurately predict acute injury to a selected group of plants from nitrogen dioxide exposures that are limited in time. The factors discussed above, which influence injury response, have not been controlled in the design used. The predictive model reported here allows for the prediction of concentrations of nitrogen dioxide that will produce acute foliar injury when the environmental conditions are not known. The model can be used to suggest combinations of time and concentrations of nitrogen dioxide that should not be exceeded in the atmosphere without injuring specific types of vegetation.

SECTION 2

EXPERIMENTAL METHODS AND PROCEDURES

Plants used in this study (Tables 1 and 2) were grown in 10 cm diameter pots, in a 1:1 peat-perlite mix, in charcoal-filtered greenhouses at about 27 C day and 21 C night temperatures. Supplemental fluorescent lighting insured a minimum 12-hr photoperiod of 10 klux throughout the year. Relative humidity varied from 30 to 80% depending on outside conditions. Plants were watered daily with a half strength Hoagland's nutrient solution. Rapid growing plants were seeded directly into the peat-perlite potting mix; slow developing seedlings were seeded in vermiculite and transplanted into the regular potting mix. The five ornamentals (Table 2) were purchased as young plants from a local nursery and established in the potting mix before exposure. Plants started directly from seed in the potting mix were overseeded and thinned to one plant per pot 7 days after seeding.

Plants were exposed to nitrogen dioxide in greenhouse exposure chambers (Heck et al., 1968) at a young stage of growth (three to six fully expanded leaves) for the crop species and when the ornamentals were still actively growing. One percent nitrogen dioxide was injected into the chamber using a two-stage dilution system and was continuously monitored with a Mast oxidant meter to insure a uniform concentration in the chamber. Chamber concentrations were determined after Saltzman (1954) using one to three 5-min bubbler samples. The number of samples varied with the length of exposure. Values are reported as parts of nitrogen dioxide per million parts of air (ppm)² on a v/v basis.

Plant injury was assessed 2 to 3 days after exposure when all leaves were examined to determine the percent area of each leaf, showing chlorosis and/or necrosis. Injury on each leaf was visually estimated in 5% increments (0 to 100% scale), and an injury index was computed on the basis of the average injury to the three most severely injured leaves per plant. The injury index included a three leaf average even if any or all of the leaves showed no injury. The injury indices for the five ornamental plants (Table 2) were based on a visual estimation of the whole plant rather than individual leaves.

² One ppm of nitrogen dioxide is equivalent to 1.9 mg/m³ of nitrogen dioxide at 760 mmHg and 25 C.

SECTION 3

EXPERIMENTAL DESIGNS AND RESULTS

Two experimental designs were used to study time-concentration effects of nitrogen dioxide on a group of plants. The first design was used to help select times and concentrations for the second, which was designed to develop time-concentration response equations for the plants studied.

The first design was for a 1-hr time period using three nitrogen dioxide concentrations (8, 16 and 32 ppm). Treatments were replicated four times. The results are shown as the average of four replicates per concentration (Table 1). The plants, listed in order of decreasing sensitivity, show the variability in sensitivity of different plant species to nitrogen dioxide. The results also show the effects of environment. The design was purposefully developed to include Bel W₃ tobacco in the summer and winter exposures. The plants exposed in winter were much more resistant.

The second experimental design was used in developing time-concentration response equations for a group of plants exposed to nitrogen dioxide using a time scale of 0.5 to 7 hr. These equations were developed by exposing selected species to nine different combinations of time and concentration (Table 2). Each time-concentration combination was replicated on 4 successive days using 36 observations (plants) of each species to develop each equation. Plant injury indices were assessed as previously described except for begonia, sultana, chrysanthemum, periwinkle and azalea. For these species a single percent injury rating was determined for the whole plant. Mean plant injury values for each time-concentration combination of the design are shown in Table 2. A multiple regression model was applied to the plant injury indices to develop the time-concentration response equations shown in Table 3.

The amount of variation explained by the model, the coefficient of variation (R^2), is shown for each equation in Table 3. Plants in this table are listed in order of their sensitivity to nitrogen dioxide, using three susceptibility groupings (susceptible, intermediate, tolerant). Examples of calculated concentrations for four combinations of percent foliar injury and time are also shown in Table 3. These equations and the survey of literature presented in the introduction of the response of plants to acute doses of nitrogen dioxide, aided in the development of the susceptibility groupings shown in Table 4. Values in this table are not absolute and should be treated as suggested limits for the given susceptibility grouping.

SECTION 4

DISCUSSION

The interrelations of time and concentration (dose) as they affect injury to vegetation are essential for an understanding of air pollution effects. Time-concentration effects have been inadequately studied and are therefore poorly understood. A discussion of time-concentration relations should consider acute, chronic, and physiological responses. At this time, there is insufficient literature relating the effects of time and concentration to the production of chronic injury, or to the reduction of growth, yield, or quality of plant material. The acute effects of nitrogen dioxide, as related to a series of times and concentrations, have not been widely studied, but the reports discussed in the introduction along with the results presented in this paper have permitted the development of estimates of concentrations that will produce injury to plants.

Researchers in the area of air pollution effects on vegetation have generally been content to view a plant's response to a pollutant or group of pollutants in a subjective way. They have preferred to look at effects and make their own interpretations. In the biological as well as the physical sciences, response should be quantified whenever possible to remove the subjective interpretations of the investigator. The response of plants to specific times and concentrations of pollutants is subject to quantification through mathematical modeling. The first such model was developed by O'Gara (1922) for the relations of dose (time x concentration) to acute plant injury from sulfur dioxide.

The O'Gara equation is a mathematical form that fits experimental data obtained from exposures of relatively short duration (less than 1 day). Guderian et al. (1960) did not believe the O'Gara equation would fit their observations (derived from continuous exposures over several hundred hours) and suggested an exponential relationship to best describe their data. In the short time span (1 to 12 hr), both equations give a reasonable fit to available data. The exponential form fits over a wider range of time for the sulfur dioxide work reported. Both of these equations relate time and concentration to a specific percentage injury and thus are capable of developing only a two-dimensional model; both also require a good estimate of a threshold injury concentration before they can be solved.

Heck et al. (1966) developed a response surface showing the variation in injury to pinto bean and tobacco from ozone exposures as time and concentration varied. Surfaces of this type make apparent the steep slope that is frequently observed in the injury versus concentration or injury versus time planes. The steep portions of the slopes indicate that relatively slight changes in many factors (environmental, nitrogen dioxide concentration, time, and others) can cause large variations in the amount of injury produced.

The models discussed above give an insight into what may happen under a given set of circumstances. These relationships are probably universal and could be derived for any toxicant producing a definite acute-type of injury. Relationships of this type permit the prediction, with reasonable assurance that no acute injury will occur as long as a certain threshold concentration is not exceeded for a given period of time.

The predictive equation reported in this paper for nitrogen dioxide was first developed for ozone (Heck and Tingey, 1971). The equation handles time and concentration separately and permits the development of a three-dimensional injury response surface similar to that reported by Heck *et al.* (1966). The equation treats concentration as the dependent variable and both injury and time as independent variables and is represented as:

$$C = A_0 + A_1 I + A_2/T$$

where C is nitrogen dioxide concentration in ppm, I is percent foliar injury, T is exposure time in hours, and A_0 , A_1 , and A_2 are constants (partial regression coefficients) which are specific for the pollutant, plant species, and the environmental conditions used. The equation permits the development of either a two-dimensional curve or a three-dimensional response surface and eliminates the necessity of determining a threshold concentration before the equation can be solved. The predictive equations reported in Table 3 eliminate the necessity for each researcher or control official to give a subjective interpretation of the data shown in Table 2 and permit a uniform interpretation of results by all who review them. The equation can also be used by a researcher or control official to predict the nitrogen dioxide concentration that could be in the atmosphere over a limited time period that would produce zero or slight injury to a given variety or species of plant. The model indicates that a given dose (concentration x time) of nitrogen dioxide over a range of times does not give constant injury.

The experimental data presented in Table 3 were developed from greenhouse exposures over several days in time and thus include an averaging effect of many environmental variables. They do not represent any specific combination of environmental conditions that would tend to make any given plant particularly responsive to nitrogen dioxide. Thus, in some cases, plants exposed under field conditions could be more sensitive to nitrogen dioxide than would be predicted from a given equation. The equations do not consider fluctuations in concentration over a given time interval or the effect of repeated fumigations over either several days or even several hours in one day. The data are from short exposures and thus should not be extrapolated to long time periods.

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TABLE 1. ACUTE INJURY TO SELECTED CROPS AFTER A
1-HOUR EXPOSURE TO NITROGEN DIOXIDE^{a/}

Plants by Name (Common, Cultivar, Scientific) ^{b/}	Injury Index (%)		
	8 ppm	16 ppm	32 ppm
Tomato, Roma ^{c/} (<u>Lycopersicon esculentum</u> , Mill.)	1	48	100
Wheat, Wells ^{d/} (<u>Triticum durum</u> , Desm.)	0	47	90
Soybean, Scott ^{c/} (<u>Glycine max</u> , (L.) Merr.)	0	26	100
Tobacco, Bel W ₃ ^{c/} (<u>Nicotiana tabacum</u> , L.)	0	23	97
Bromegrass, Sac Smooth ^{c/} (<u>Bromus inermis</u> , L.)	2	17	97
Swiss Chard, Fordhook Giant ^{d/} (<u>Beta vulgaris</u> L.)	0	11	62
Tobacco, White Gold ^{c/} (<u>Nicotiana tabacum</u> , L.)	0	1	70
Cotton, Acala 4-42 ^{d/} (<u>Gossypium hirsutum</u> , L.)	0	0	54
Beet, Perfected Detroit ^{d/} (<u>Beta vulgaris</u> , L.)	0	0	36
Orchard Grass, Potomac ^{d/} (<u>Dactylis glomerata</u> , L.)	0	1	18
Tobacco, Bel W ₃ ^{d/}	0	0	5

^{a/} Plants were exposed in Cincinnati, Ohio.

^{b/} Scientific name is given when plant is first listed.

^{c/} Plants were exposed in August with light intensity at 2200 ft-c, temperature 28°C, humidity 75 percent.

^{d/} Plants were exposed in January with light intensity at 1400 ft-c, temperature 21°C, humidity 70 percent.

TABLE 2. ACUTE INJURY TO SELECTED PLANTS USED IN DEVELOPING CONCENTRATION-TIME RESPONSE EQUATIONS FOR NITROGEN DIOXIDE^{a/}

Plants by Name (Common, Cultivar, Scientific)	Dose ^{b/} Conc. Time	Injury Index (%)								
		2.5	4	6	10	14	15	20	20	35
		5	4	3	20	2	15	10	5	5
		0.5	1	2	0.4	7	1	2	4	7
Oats, Clintland 64 (<u>Avena sativa</u> , L.)		0 ^{c/}	0	0	80	2	84 ^{c/}	39	0	21
Radish, Cherry Belle (<u>Raphanus sativus</u> , L.)		0 ^{c/}	0	0	95	0	90 ^{c/}	31	1	2
Bromegrass, Sac Smooth		0 ^{c/}	0	0	69	0	50 ^{c/}	26	1	0
Begonia, Thousand Won- ders, White ^{e/} , (<u>Begonia Rex</u> , Putz.)		0	1	0	26	0	35	49	4	5
Chrysanthemum, Oregon ^{e/} (<u>Chrysanthemum</u> , sp.)		1	1	1	34	0	41	25	4	1
Sultana, White Imp ^{e/} (<u>Impatiens sultani</u> , Hook)		0	0	0	51	0	26	24	0	0
Oats, 329-80 ^{c/}		2	2	1	32	1	18	14	9	14
Cotton, Paymaster		0	0	6	50	0	27	2	2	1
Wheat, Wells		3	2	1	31	3	34	2	3	1
Cotton, Acala 4-42		0	0	0	28	0	28	1	0	1
Periwinkle, Bright Eyes ^{e/} (<u>Vinca minor</u> , L.)		0	0	0	13	0	20	23	1	1
Oats, Pendek ^{d/}		1	2	0	39	0	2	2	1	2
Broccoli, Calabrese (<u>Brassica oleracea</u> <u>botrytis</u> , L.)		0	0	0	19	0	21	0	0	0
Tobacco, Bel B		0	0	3	18	0	17	0	0	0
Tobacco, White Gold		0	0	1	18	0	6	0	0	0

continued .

TABLE 2 (continued)

Plants by Name (Common, Cultivar, Scientific)	Dose ^{b/} Conc. Time	Injury Index (%)								
		2.5	4	6	10	14	15	20	20	35
		5	4	3	20	2	15	10	5	5
		0.5	1	2	0.4	7	1	2	4	7
Tobacco, Bell W ₃		0	0	6	15	0	2	0	0	0
Tobacco, Burley 21		0	0	0	8	0	0	0	0	0
Corn, Pioneer 509-W (<i>Zea mays</i> , L.)		1	0	0	1	0	1	0	0	0
Corn, Golden Cross		0	0	0	0	0	0	0	0	2
Azalea, Alaska (<i>Rhododendron</i> , sp.) ^{e/}		0	0	0	0	0	1	0	0	0
Sorghum, Martin (<i>Sorghum</i> , sp.)		0	0	0	0	0	0	0	0	0
Cucumber, Long Marketer (<i>Cucumis sativus</i> , L.)		0	0	0	0	0	0	0	0	0

^{a/} Plants were exposed in Cincinnati, Ohio. Each value is the average of 4 replicate plants except as noted. Plants are listed in general order of sensitivity. Scientific names are included except when already given in Table 1. Equations are shown in Table 3. Plants were exposed from July 22 through September 20, 1968 except for wheat, oats (Pendek and 329-80) broccoli and cucumber which were exposed in early November

^{b/} Dose = ppm/hrs, conc. = ppm, time = hr.

^{c/} Three replications per treatment.

^{d/} Two replications per treatment.

^{e/} Ornamental plants obtained from nursery, injury indices are determined as whole plant values.

TABLE 3. TIME-CONCENTRATION RESPONSE EQUATIONS FOR A SELECTED GROUP OF PLANTS TO NITROGEN DIOXIDE^{a/}

Plants by Name ^{b/} (common, Cultivars)	Equation ($C = A_0 + A_1 I + A_2/T$) ^{c/}	R^2 ^{d/}	Concentrations (ppm) to produce the I (in %) in T (hr)			
			I=5, T=1	I=5, T=8	I=50, T=1	I=50, T=8
<u>Susceptible</u>						
Oats (Clintland 64)	$c = 1.45 + 0.13 I + 2.39/T$	0.76	4.5	2.3	10.3	8.3
Radish (Cherry Belle)	$C = 2.43 + 0.14 I + 1.02/T$	0.83	4.1	3.2	10.5	9.6
Oats (329-80)	$C = 1.75 + 0.15 I + 3.24/T$	0.56	5.7	2.9	12.5	9.7
Bromegrass (Sac Smooth)	$C = 2.49 + 0.16 I + 1.90/T$	0.71	5.2	3.5	12.4	10.7
Begonia (Thousand Wonders White)	$C = 2.45 + 0.15 I + 2.99/T$	0.63	6.2	3.5	12.9	10.3
Chrysanthemum (Oregon)	$C = 3.16 + 0.16 I + 2.14/T$	0.72	6.1	3.7	13.3	11.4
Oats (Pendek)	$C = 2.79 + 0.14 I + 2.88/T$	0.50	6.4	3.8	12.7	10.2
Wheat (Wells)	$C = 2.80 + 0.13 I + 2.94/T$	0.52	6.4	3.8	10.2	7.7
Sultana (White Imp)	$C = 3.93 + 0.13 I + 1.73/T$	0.67	6.3	4.8	12.2	10.7
Broccoli (Calabrese)	$C = 3.07 + 0.20 I + 2.94/T$	0.53	7.0	4.5	16.0	13.4
Periwinkle (Bright Eyes)	$C = 2.92 + 0.23 I + 3.02/T$	0.55	7.1	4.5	17.4	14.8
<u>Intermediate</u>						
Cotton (Paymaster)	$C = 2.97 + 0.23 I + 1.94/T$	0.58	7.1	5.3	17.4	15.7
Cotton (Acala 4-42)	$C = 3.68 + 0.22 I + 3.15/T$	0.50	7.9	5.2	17.8	15.1
Tobacco (Bel B)	$C = 3.62 + 0.21 I + 3.98/T$	0.38	8.7	5.2	18.1	14.6
Tobacco (Bel W ₃)	$C = 3.65 + 0.18 I + 4.40/T$	0.31	9.0	5.2	17.1	13.2
Tobacco (White Gold)	$C = 4.03 + 0.30 I + 3.56/T$	0.40	9.1	6.0	22.6	19.5

continued ...

TABLE 3 (continued)

Plants by Name ^{b/} (common, Cultivars)	Equation ($C = A_0 + A_1 I + A_2/T$) ^{c/}	R^2 ^{d/}	Concentrations (ppm) to produce the I (in %) in T (hr)			
			I=5, T=1	I=5, T=8	I=50, T=1	I=50, T=8
<u>Tolerant</u>						
Tobacco (Burley 21)	None		1 of 36 plants injured; 0.5 hr., 26 ppm, 33 percent			
Corn (Pioneer 509-W)	None		4 of 36 plants injured; all injuries were 1.6 percent			
Corn (Golden Cross)	None		1 of 36 plants injured; 7.0 hr, 6 ppm, 7 percent			
Azalea (Alaska)	None		1 of 36 plants injured; 1.0 hr, 17 ppm, 5 percent			
Sorghum (Martin)	None		0 of 36 plants injured			
Cucumber (Long Marketer)	None		0 of 36 plants injured			

^{a/} Equations were developed from exposures limited in time (0.5 - 7.0 hr) and denote acute injury symptoms to the plants. Concentrations used ranged from 1 to 20 ppm of nitrogen dioxide. Plants are grouped in 3 susceptibility categories. Specific injury averages are given in Table 2.

^{b/} Scientific names are given in Tables 1 and 2.

^{c/} C is nitrogen dioxide concentration in ppm; I is percent injury; T is time in hr; and A_0 , A_1 , and A_2 are constants (partial regression coefficients) specific for pollutant, plant species, and environmental conditions used.

^{d/} R^2 , multiple correlation coefficient squared which represents the percent variation explained by the model.

TABLE 4. PROJECTED NITROGEN DIOXIDE CONCENTRATION RANGES WHICH WILL PRODUCE, FOR SHORT-TERM EXPOSURES, FIVE PERCENT INJURY TO VEGETATION GROWN UNDER SENSITIVE CONDITIONS^{1/}

Time (hr)	Concentrations (ppm) Necessary to Produce Injury in Three Susceptibility Groupings of Plants		
	Susceptible	Intermediate	Tolerant
0.5	6 - 10	9 - 17	≥ 16
1.0	4 - 8	7 - 14	≥ 13
2.0	3 - 7	6 - 12	≥ 11
4.0	2 - 6	5 - 10	≥ 9
8.0	2 - 5	4 - 9	≥ 8

^{1/} The values in this table were developed from a subjective evaluation of the earlier tables and references included in the introduction.

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

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16. ABSTRACT <p>An experimental design was developed utilizing five time periods from 0.5 to 7 hours with two nitrogen dioxide concentrations at each time period. Concentrations were chosen that would produce threshold and severe injury at these time periods. From these data for each plant species, an equation was developed utilizing concentration as the dependent variable, and both foliar injury and time as independent variables. The model allows for the development of a three-dimensional response surface within the limits of the times and concentrations used. The model should be of practical importance in predicting the concentration of nitrogen dioxide that will produce a given amount of injury to a specific crop during a single 12-hour day. Research to date suggests the model may be used for other pollutants and for other plant species that show injury following exposure to high ambient nitrogen dioxide concentrations.</p>					
17. KEY WORDS AND DOCUMENT ANALYSIS					
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