Effect of Reducing Oxidant Injury and Early Blight on Fresh Weight and Tuber Density of Potato

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ABSTRACT

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Ethylene diurea (EDU) significantly protected potato foliage from visible injury due to ozone (O_3) in laboratory and field experiments, but did not induce significant increases in fresh weight or specific gravity of tubers at harvest. Ambient O_3 concentrations were low, and the severity of oxidant injury was too small to have an impact on yield. Fentin hydroxide (Du-Ter) did not diminish O_3 injury on potato leaves in controlled fumigations, but did significantly reduce adaxial bronzing in the field. The potato early blight pathogen, *Alternaria solani*, was frequently isolated from leaf tissue with adaxial bronzing. Significant increases in the fresh weight of tubers of the cultivar Norchip, and the overall specific gravity of tubers from all cultivars, showed a greater-than-additive response in the treatment combination of Du-Ter and EDU when compared to increases in either single spray treatment. The effects of Du-Ter, either singly or in combination with EDU, indicated that adaxial O_3 lesions became colonized by *A. solani* under natural field conditions. Regression equations relating changes in yield to percentage changes in apparent infection rate or rate of defoliation from early blight and O_3 showed that cultivar tolerance is a major factor to consider when assessing the importance of controlling symptoms of oxidant injury or early blight on potato.

Additional key words: air pollution, *Solanum tuberosum*, yield.

A number of agents including insects, wind, pesticides, and air pollutants can cause leaf injury on potato. Some injury symptoms can be attributed to a specific environmental factor, while others cannot, particularly when two or more factors have been present concurrently. Ozone-induced injury has been observed frequently on potatoes in southern Ontario (1,13,17) and in other areas of North America (3–7,12,16,18,19). Symptoms of oxidant injury are adaxial bronzing (1,3,5,12,17,19) or abaxial speckle (3,5,12). The expression of oxidant symptoms may be related to the duration and concentration of ozone (O_3), either singly or in combination with modifying factors such as soil moisture, relative humidity or plant disease (10,20,22). That oxidant injury on potato leaves can enhance the development of foliar fungal diseases (1,16) has significant implications for disease control and needs to be more clearly documented.

Several studies have been conducted to assess the direct impact of O_3 injury on potato in the field (1,4,5,13). Spraying foliage with the antioxidant ethylene diurea (EDU) significantly increased yields in one study (1), but not in another (4). Foliar sprays (13) or soil drenches (5) of EDU did not always increase yields significantly in other studies (5,13). The direct impact of O_3 on yields of field-grown potato has not been well established because of variability in cultivar sensitivity (6,7,12,18) and inconsistent responses of the same cultivar to antioxidant treatments (1,4,5,13).

Oxidants interact with plant diseases in several ways (11,20,22). Viral, bacterial or biotrophic fungal infections interfere with O_3 stress, or are inhibited by oxidant effects (10,11,20,22). Necrotrophic fungal diseases of potato (16), onion (23,24), geranium (22), poinsettia (22), and pine (20) are enhanced by oxidant injury. Potato early blight, caused by a necrotrophic fungal pathogen, *Alternaria solani* Sorauer, is evident each season in southern Ontario. This disease can become quite severe on susceptible potato cultivars if routine chemical controls are not utilized (14). Chemical controls are costly and do not always control this disease. One preliminary report showed that a spray mixture of EDU and the fungicide chlorothalonil (Bravo) gave better control of early blight, based on visual assessment and significantly better harvest weights than a single application of Bravo on the susceptible cultivar Norchip in 1979 (1).

To determine whether potato production is significantly affected by O_3 directly or through an enhancement of early blight development, the following approach was used: To separate the chemical control of O_3 injury and of early blight, a number of fungicides were screened to select one that possessed no antioxidant properties, but gave effective control of early blight. The selected fungicide and the antioxidant EDU were applied singly or together in field plots of various cultivars at a number of locations.

MATERIALS AND METHODS

Chemical screening. Five fungicides, metiram (Polyram), mancozeb (Manzate), chlorothalonil (Bravo), captafol (Difolotan), and fentin hydroxide (Du-Ter), each registered in Ontario for control of potato early blight, were screened and compared to EDU for antioxidant properties on two cultivars, Norland and Chieftain, in controlled fumigation experiments. Sprayed or unsprayed potato plants were transferred into four exposure chambers 24 hr prior to fumigation. All plants were exposed for 6 hr per day to O_3 at 530 $\mu g/m^3$ (0.25 ppm) for 5 consecutive days. Ozone was generated with an Elcar Viva O_3 generator and measured with a Dasibi model 1003AH analyzer calibrated against an O_3 calibrator (Monitor Labs model 8500). Two fully expanded leaves were assessed 24 hr after the final exposure for the proportion of leaf covered with black spots, using the Horsfall-Barratt rating system (15). The experiment was repeated three times.

The five fungicides, EDU, and a mixture of equal parts of EDU and Du-Ter were tested for fungitoxicity on a pathogenic isolate of *A. solani*. Cultures were maintained on potato-dextrose agar (PDA). Chemicals were added to 10 ml of PDA in 9-cm-diameter petri plates at concentrations of 500, 50, 5, 0.5, and 0 (check) $\mu g/ml$. Twenty plates were used for each concentration of each chemical. A 3-mm-diameter plug removed from the actively growing edge of a culture was transferred to the center of each test plate. Cultures

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were incubated for 20 days in the lab at 20 C. Colony diameters were measured and expressed as percentages of the check. The percentage values were plotted on semilogarithmic paper, and the curves were used to estimate the concentration of chemical required to inhibit growth by 50%. In addition, percentage inhibition values for Du-Ter and the Du-Ter + EDU mixture were transformed to natural logarithms and regressed against the concentration of dissolved chemical. The transformed observations were used in an analysis of covariance to determine whether observations from both treatments could be pooled to form a common regression relationship, and to determine if EDU affected the toxicity of Du-Ter to isolates of *A. solani*.

Field experiments. Treatments designed to assess the impact of O_3 injury on early blight development were established on commercial farms in 1980, 1981, and 1982 in a randomized complete block design. Plots within experimental blocks consisted of four rows, each 7 m long and consisting of 20 healthy plants. Plots were spaced 2–3 m apart. The two outer rows of the plots were unsprayed guard rows, designed to minimize interplot interference. Each experimental block included three cultivars and four chemical treatments. Four plots were assigned randomly to each cultivar within each block. Treatments were also assigned randomly to individual cultivars. Treatments in each experimental block were separately randomized. The large block design was replicated four times at Alliston in 1980, six times at Alliston and Carlisle in 1981, and six times at Alliston and Burlington in 1982. Atmospheric O_3 concentrations were monitored continuously with Bendix or Mast O_3 meters with values corrected against the calibrator. Artificial inoculum was not introduced into any field experiment. Ridomil was applied to the entire plot where needed to prevent late blight. Sevin, Guthion, and Monitor were applied to all the plots (including guard rows) to control Colorado potato beetle, flea beetle, or aphids. Thimet was banded into the furrows at planting time to control early insect infestations.

Treatments. The cultivars Kennebec, Chieftain, and Norchip were chosen for the field studies because they are widely grown in southern Ontario, have different sensitivities (Kennebec [insensitive], Chieftain [intermediate], and Norchip [sensitive]) to ambient O_3 (3,6,7,12,18), and different levels of resistance (Kennebec [resistant], Chieftain [intermediate], and Norchip [susceptible]) to early blight (8,9,14). Plots were sprayed with Du-Ter to control early blight, with EDU to reduce oxidant injury, with a tank mixture of both to control both symptoms simultaneously, or were left unsprayed. EDU was applied at 1.5 kg a.i./ha and Du-Ter at 0.3 kg a.i./ha, whether applied singly or in a mixture. Plots were sprayed at 7- to 9-day intervals from the first week of July to the second week of September of each season. Sprays were applied to the two center rows of the small plots by using CO_2-pressurized hand sprayers.

Injury assessment. Plots were assessed every 5–14 days in July, August, and September. Both leaf surfaces were examined. The percentage of the row canopy affected by early blight lesions, i.e., lesions that showed evidence of concentric ring expansion or were rough rather than smooth in appearance, was estimated according to the Horsfall-Barratt system (15). The percentage of the canopy affected by oxidant lesions (i.e., superficial, smooth lesions with no evidence of concentric ring expansion affecting either surface) was estimated in the same way. The total percentage of the row canopy showing defoliation from both early blight and oxidant lesions was estimated as well. The mean percent areas of canopy diseased from early blight, injured from O_3, or affected by both stresses combined (referred to as "defoliated") were calculated from the Horsfall-Barratt ratings. Observations from the dates when maximum injury was seen were used to compare the severity of O_3 injury in different treatments. Small plot means from all locations, except Burlington, were used in an analysis of variance for treatment differences. Injury symptoms at Burlington from unidentified causes obscured the O_3 injury which could not be quantified, and therefore was not analyzed. Treatment means were compared using the protected LSD test (21) at $P=0.05$. Each cultivar was analyzed separately. Kennebec rarely showed injury greater than 1% and therefore could not be used in the analysis. Since O_3 injury had not been observed in the field when O_3 concentration was below 107 $\mu g/m^3$, the hours that O_3 concentrations exceeded 107, 160, and 214 $\mu g/m^3$ were summed from the first week of July to the date of maximum injury and to the end of August to estimate the oxidant dosage.

Plot means for percent disease and percent defoliation that were less than 50% were transformed using the logit transformation, $\log_e [x/(1-x)]$ (25), and plotted against time. Slopes derived from the regression equations for each curve provided estimates of the apparent rates of infection or defoliation. These rates were subjected to an analysis of variance and compared with a protected LSD test at $P = 0.01$. Observations from all experiments were analyzed by cultivar and then were pooled to determine the effects of treatments as described below.

Yield assessment. At the end of each season, the two center rows of each plot were harvested separately, all tubers were weighed and mean plot weights were calculated. Specific gravity of tubers was determined on two subsamples of medium-sized tubers from each row, and means for each plot were calculated. Plot means for fresh weight and specific gravity from all locations except Carlisle were analyzed by cultivar or were pooled. Yield data from Carlisle were unavailable because plots were too wet to harvest and tubers had rotted. Plot means were used in an analysis of variance and treatment means were compared with a protected LSD test at $P=0.05$.

Mathematical models associating observed rates of apparent infection and defoliation with total weight of tubers per plot at harvest were constructed. Rates of infection and defoliation calculated for sprayed plots were expressed as percentages of the rates observed in the unsprayed check plots within each block. Observations from each cultivar were treated separately. Tuber weights were expressed in the same way. The percentage change in total tuber weight at harvest, due to treatment, was regressed against the percentage change in the apparent infection rate or rate of defoliation. Data from all cultivars were pooled initially, but when the associated regression strengths and residuals indicated a poor linear fit, observations from each cultivar were used to form separate equations.

TABLE 1. Effectiveness of five fungicides and EDU in reducing O_3 injury on leaves of potato cultivars Norland and Chieftain exposed to O_3 at 530 $\mu g/m^3$, and in inhibiting the growth of pathogenic isolates of *Alternaria solani* by 50% in culture (EC_{50})

Spray treatment	Injury on Norland (%)	Injury on Chieftain (%)	EC_{50} (mg/L)
Du-Ter	20.0 a[z]	15.0 a	0.1
Polyram	20.0 a	10.0 a	80.0
Check	17.0 a	15.0 a	–
Difolatan	6.0 b	5.0 ab	4.0
Mancozeb	5.0 b	3.0 b	15.0
Bravo	5.0 b	2.0 b	5.0
EDU	0.5 c	0.1 c	250.0
Du-Ter + EDU	–	–	0.3

[z] Values in each column followed by the same letter are not significantly different at $P=0.05$, according to protected LSD test.

TABLE 2. Ambient O_3 concentrations from 1 July to the date of maximum injury to potato foliage and to 31 August

Location and year	Cumulative hours to time of maximum injury of O_3 ($\mu g/m^3$) exceeding:			Cumulative hours to 31 August of O_3 ($\mu g/m^3$) exceeding:		
	107	160	214	107	160	214
Alliston 1980	273	60	5	286	62	5
Alliston 1981	112	18	0	115	18	0
Carlisle 1981	195	19	0	232	22	0
Alliston 1982	130	26	0	139	26	0

RESULTS

Chemical screening. EDU provided significantly better protection against O_3 injury than any other chemical tested in the controlled-fumigation experiments (Table 1) and was not appreciably toxic to pathogenic isolates of *A. solani* (Table 1) (2). Du-Ter, Polyram, and Difolatan did not significantly reduce O_3 injury in fumigation experiments (Table 1). Du-Ter, however, was the most fungitoxic (Table 1) and was used for the field experiments. Toxicity data for Du-Ter alone and mixed with EDU could be used to form a common regression equation to describe fungal inhibition over several chemical concentrations. EDU, when mixed with Du-Ter, did not alter the fungitoxicity of Du-Ter (Table 1).

Foliar injury. Concentrations of atmospheric O_3 rarely exceeded 214 $\mu g/m^3$ (Table 2) and the percentage canopy affected by oxidant injury was rarely greater than 10% (Table 3). Oxidant symptoms were more severe at locations where concentrations were higher. Injury at Alliston in 1981 was more severe than expected from the O_3 dose relative to other locations. This apparent inconsistency may have resulted from high relative humidity, moist soil, or from the rapid development of disease symptoms just before maximum O_3 injury was seen. EDU and Du-Ter alone or in combination significantly reduced O_3 injury on Norchip and Chieftain (Table 3). The combination spray significantly reduced O_3 injury on Norchip compared to either chemical alone (Table 3).

Many superficial lesions characteristic of O_3 injury (1,12,17–19) were seen on the adaxial leaf surface. They showed evidence of concentric ring expansion, typical of early blight, 1–2 wk after first being observed. *A. solani* commonly was isolated from leaf tissue showing adaxial bronzing when leaves were collected in fields with rapid disease development. The pathogen was rarely recovered from leaves with adaxial bronzing collected in fields with slow rates of disease development.

The apparent infection and defoliation rates were not affected by EDU, but were both reduced by Du-Ter in all cultivars (Table 4). The combination treatment gave a further small reduction in both rates that was not significant at $P = 0.05$. When all the observations were pooled, the apparent infection rate of 0.149 per unit per day was significantly less than the apparent defoliation rate of 0.164 per unit per day, according to a protected LSD test at $P = 0.01$, indicating that O_3 injury was contributing significantly to the rate of development of foliar necrosis.

Yield response. Du-Ter significantly increased yield in all three cultivars (Table 4). Although EDU by itself did not significantly increase yield, in combination with Du-Ter it further increased yield in the O_3-sensitive, early blight-susceptible cultivar Norchip. Du-Ter also significantly increased specific gravity in all cultivars. The further small increase from the combination treatment was only statistically significant at $P = 0.05$ when the data for all the cultivars were combined (Table 4). The harvest weight for Norchip and the overall mean specific gravity for all cultivars were not only significantly greater, but showed a greater-than-additive response in the combination treatment compared to the single treatments (Table 4).

Regression equations describing change in fresh weight, due to treatment, had similar coefficients of determination regardless of which rate parameter was used (Table 5). Models for Chieftain and Norchip had coefficients ranging from 0.52 to 0.66 (Table 5). No significant equation relating percentage change in fresh weight to percent change in apparent infection and defoliation rates could be demonstrated for Kennebec. The rate of development of foliar symptoms did not appear to affect the fresh weight of Kennebec at harvest. Therefore, it is concluded that Kennebec showed tolerance to both O_3 and infection by *A. solani*.

TABLE 3. Effect of EDU and Du-Ter alone or in combination on the percentage row canopy of potato cultivars Norchip and Chieftain affected by apparent oxidant injury

Cultivar and location	Canopy injury (%)			
	Check	EDU	Du-Ter	Du-Ter + EDU
Norchip				
Alliston 1980	9.8	2.6	11.5	2.3
Alliston 1981	7.8	7.2	4.9	1.7
Carlisle 1981	2.6	2.0	0.8	0.6
Alliston 1982	3.0	1.6	0.9	0.3
Mean response[z]	5.7 a	3.5 b	4.5 b	1.1 c
Chieftain				
Alliston 1980	5.9	4.0	5.9	3.0
Alliston 1981	12.4	9.5	8.3	5.6
Carlisle 1981	3.3	2.7	1.8	1.8
Alliston 1982	2.4	0.8	2.7	1.8
Mean response	6.0 a	4.3 bc	4.6 b	3.0 c

[z] Treatment means followed by the same letter are not significantly different at $P = 0.05$, according to the protected LSD test.

TABLE 4. Effect of EDU and Du-Ter singly and in combination on apparent infection and defoliation rates, harvest weights, and tuber densities of three potato cultivars

Cultivar	Treatment	Apparent infection rate (per unit/day)	Apparent defoliation rate (per unit/day)	Fresh weight (kg)	Tuber density (g/L)
Norchip	Check	0.201 a[z]	0.212 a	17.2 a	1.0708 a
	EDU	0.194 a	0.202 a	17.7 a	1.0709 a
	Du-Ter	0.159 b	0.174 b	19.1 b	1.0751 b
	Du-Ter + EDU	0.154 b	0.164 b	20.8 c	1.0758 b
Chieftain	Check	0.147 a	0.174 a	21.1 a	1.0650 a
	EDU	0.147 a	0.168 a	20.4 ac	1.0655 a
	Du-Ter	0.133 b	0.156 b	22.6 c	1.0667 b
	Du-Ter + EDU	0.134 b	0.157 b	22.3 bc	1.0672 b
Kennebec	Check	0.139 a	0.149 a	22.8 a	1.0697 a
	EDU	0.136 a	0.148 a	21.6 a	1.0703 a
	Du-Ter	0.124 b	0.134 b	25.2 b	1.0721 b
	Du-Ter + EDU	0.122 b	0.131 b	24.8 b	1.0721 b
All cultivars	Check	0.162 a	0.178 a	20.4 a	1.0686 a
	EDU	0.159 a	0.172 a	20.0 a	1.0689 a
	Du-Ter	0.139 b	0.155 b	22.3 b	1.0709 b
	Du-Ter + EDU	0.137 b	0.151 b	22.6 b	1.0716 c

[z] Values within the same column followed by the same letter are not significantly different at $P = 0.05$, according to the protected LSD test. Harvest data from the Carlisle experiment were not available.

TABLE 5. Regression equations[z] relating the precentage change in fresh weight (Y) due to treatment to percentage change in the apparent infection rate and defoliation rate (X)

Cultivar	Regression equation	Regression significance (P)	Coefficient of determination
Apparent infection rate			
Norchip	$Y = 3.5 - 0.6\ X$	0.01	0.66
Chieftain	$Y = 0.9 - 0.8\ X$	0.01	0.52
Kennebec	$Y = 3.9 - 0.2\ X$	0.62	0.03
Apparent defoliation rate			
Norchip	$Y = 5.0 - 0.7\ X$	0.01	0.56
Chieftain	$Y = 3.2 - 0.9\ X$	0.01	0.56
Kennebec	$Y = 4.4 - 0.1\ X$	0.69	0.001

[z]Equations were based on data collected in four locations for three seasons. The independent variable ranged from −35% to +7.5% in both cases.

DISCUSSION

The effects of O_3 on potato foliage are well documented (1,3–7,12,13,16–19) as are the effects of early blight (8,9,14). An interaction between the two agents had also been indicated (1). In this study it was found that tissue injured by O_3 became rapidly colonized by the early blight fungus under conditions of rapid disease development. When disease pressure was low, O_3 lesions showed no signs of expansion or concentric ring formation.

Because O_3 injury was low during the 3 yr of this study, it was difficult to show a clear interactive effect between O_3 injury and early blight injury on yield and specific gravity. Reducing ozone injury with EDU had little effect on fresh weight and tuber density. Reducing O_3 injury with EDU appeared to improve the control of early blight, especially in the cultivar Norchip, which is particularly sensitive to both O_3 and early blight. However, the reduction in apparent infection rate was not significant at $P = 0.05$. The effects of EDU in increasing tuber density, although small, were more consistent across the cultivars, but required the pooling of all the data from the combination treatment to show statistical significance.

The reduction in weight of the different cultivars due to O_3 on early blight is not directly related to the percentage leaf area diseased or injured. Even though the loss in Kennebec is greater than that in Chieftain, Chieftain had the higher infection and defoliation rates (Table 4). Cultivars differ in the relation of loss of leaf area to loss in yield.

Du-Ter, a chemical with no significant antioxidant properties, significantly reduced the development of oxidant injury symptoms in the field. Presumably Du-Ter reduced the colonization of small oxidant lesions making them less apparent. The early blight fungus was recovered frequently from leaf tissue with adaxial bronzing. Either oxidant-caused lesions became infected and colonized by *A. solani* or injury to potato foliage predisposes it to greater infection of uninjured tissue. It seems advantageous to control both disease and other injuries concurrently to minimize the development of early blight. Understanding the level of cultivar tolerance to disease and O_3 is very important to growers balancing costs of chemicals with losses in yield resulting from poor control of foliar injury. The advantage of using a fungicide with antioxidant properties before or during the early stages of an early blight epidemic warrants investigation in sensitive cultivars like Norchip.

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