Phytotoxic Action of Desmedipham: Influence of Temperature and Light Intensity[1]

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Abstract. Inhibition of growth and photosynthesis in sugarbeet (*Beta vulgaris* L. var. 'USH10') treated with desmedipham [ethyl m-hydroxycarbanilate carbanilate (ester)] was most severe between 25 and 30 C and decreased with higher or lower temperatures. Transfer of sugarbeet plants grown at temperatures from 10 to 35 C to higher temperatures after treatment increased injury and photosynthetic inhibition. Higher temperatures prior to treatment reduced injury at all posttreatment temperatures. When the temperature was changed from 25 to 40 C, inhibition was most severe immediately after treatment. Two days after treatment this 15 C temperature change did not cause additional injury. High posttreatment light intensities caused greater inhibition of photosynthesis than low light intensities.

INTRODUCTION

The 3-alkoxycarbonyl aminophenyl-N-arylcarbamates (biscarbamates) were assayed for herbicidal properties in the late 1960's and were found to cause varying degrees of photosynthetic inhibition (11). Of the compounds tested, phenmedipham (methyl m-hydroxy-carbanilate m-methylcarbanilate) has been used to control a variety of broadleaf weeds in beet crops[3]. Desmedipham, a phenmedipham analog, has recently been developed for the control of redroot pigweed (*Amaranthus retroflexus* L.), which is a problem weed in sugarbeet and is resistant to phenmedipham (8). Variations in light and temperature conditions affect phenmedipham selectivity (1, 5). We studied responses of sugarbeet growth and photosynthesis to desmedipham under various temperature and light conditions.

MATERIALS and METHODS

Sugarbeet seeds were germinated in plastic pots at 30 C in a greenhouse, and transferred to growth chambers upon emergence. The seedlings were separated into seven sets; each set consisted of five to seven groups of 10 pots with six plants per pot. The sets of plants were grown at day temperatures ranging from 10 to 40 C at 5 C intervals. Night temperatures were lowered by 10 C, and for those plants grown at 10 C day temeprature, by 5 C. Day and night lengths were 16 and 8 hr, respectively. Light intensity at plant height in all chambers was 490 microeinsteins per m²/sec (quantum flux measured with a Lambda Instruments LI-185 quantum sensor in the 400–700 nm spectral range). Pots were watered twice daily with 0.5 strength Hoagland solution (6). Relative humidity was not controlled and decreased almost linearly from 67% at 10 C to 30% at 40 C. Plants in half the pots in each group were sprayed with 1.1 kg/ha of desmedipham with a greenhouse belt sprayer when the third true leaf was 3 to 5 mm. The unsprayed plants were controls.

Immediately following spraying plants were either returned to their original temperature regime or transferred to another. Temperature differences spanned up to 20 C at 5 C increments towards higher and lower temperatures; e.g., groups of plants grown at 20 C were transferred to 10, 15, 20, 25, 30, 35, or 40 C. Seven hours after spraying all plants were exposed to $^{14}CO_2$ in a 250-L, air tight, fan-equipped box at room temperature and at a light intensity of 300 microeinsteins per m²/sec. The $^{14}CO_2$ injected into the box was produced by reacting 100 μCi of $Na_2$$^{14}CO_2$ with excess lactic acid and amounted to 10% of the total CO_2 content of the box. Following a 15-min exposure the shoots of four plants from each pot were cut at ground level, air dried at 80 C, and ground in a Wiley mill. Thirty mg samples were placed in 2.5-cm stainless steel planchettes in even, non-overlapping layers using 0.1%Tergitol in 80% ethanol as a fixative. Radioactivity was determined with an end window gas flow detector. Data were expressed as percent inhibition of the unsprayed plants and represent the average of two experiments with five replications each.

The remaining two plants per pot were grown for 7 days after spraying. Then they were cut at ground level, air dried at 80 C, and weighed.

The effect of time between spraying and the temperature change was determined by growing plants at 25 C and transferring them to a 40 C regime at increasing time intervals after spraying. All other criteria were as previously described.

Plants for the light intensity experiment were grown in a greenhouse at 35 C. After spraying they were transferred to a growth chamber at 35 C and exposed to light intensities of 480, 320, 210, and 33 microeinsteins per m²/sec, corresponding to 25, 17, 9, or 1.5 klux. Fluorescent and incandescent lights were used as light sources. Leaf temperatures were measured with a Wescor psychrometric microvoltmeter using an iron-constantan thermocouple appressed to the lower leaf surfaces. The CO_2 concentration (320 ppm) of the ambient air was not a limiting factor within the light intensity range used (4). Light intensity dependent CO_2 fixation was determined in one set of plants using the methods described before. However, $^{14}CO_2$ exposures were either 3 or 9 hr after spraying. The inhibition of net photosynthesis after spraying was measured after the same time intervals in a second set of plants.

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These were placed into a 3-L, airtight, plexiglass box at the same light and temperature conditions as those utilized during the $^{14}CO_2$ exposure. Nitrogen gas containing 580 ppm CO_2 was passed through the box and into an infrared gas analyzer at a flow rate of 1.25 L/min. Upon equilibration the gas cylinder was disconnected and the system closed. The loss of CO_2 was recorded as Δ ppm/min and expressed as percent inhibition. All other conditions were as described for the temperature change experiments.

All experiments were conducted as completely random designs. Data were subjected to factorial analyses of variance.

RESULTS and DISCUSSION

At a light intensity of 490 microeinsteins per m^2/sec the temperature optimum for growth was 25 to 30 C. Inhibition of photosynthesis 7 hr after spraying was more severe than growth inhibition 7 days later, but the temperature response curves were similar in both cases (Figure 1). Transfer of plants grown at 25 and 30 C to higher temperatures resulted in greater inhibition, while transfer to lower temperatures resulted in less inhibition (Figure 2). Plants grown at 10 and 15 C had more inhibition following a rise in temperature to 20 C. Plants grown at 35 and 40 C showed increased inhibition
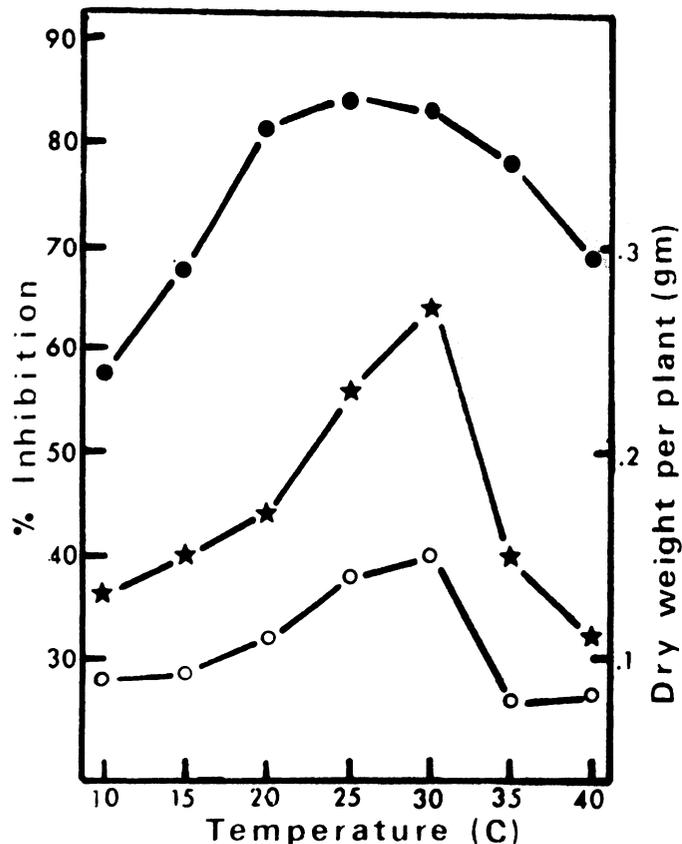

Figure 1. Influence of temperature on photosynthesis inhibition and growth of sugarbeets following application of 1.1 kg/ha of desmedipham. Dry weight per plant of controls 7 days after treatment *——*, percent inhibition of photosynthesis 7 hr after treatment ●——●, percent inhibition of growth 7 days after treatment o——o. Pre- and post-treatment temperatures are as indicated along the abscissa.
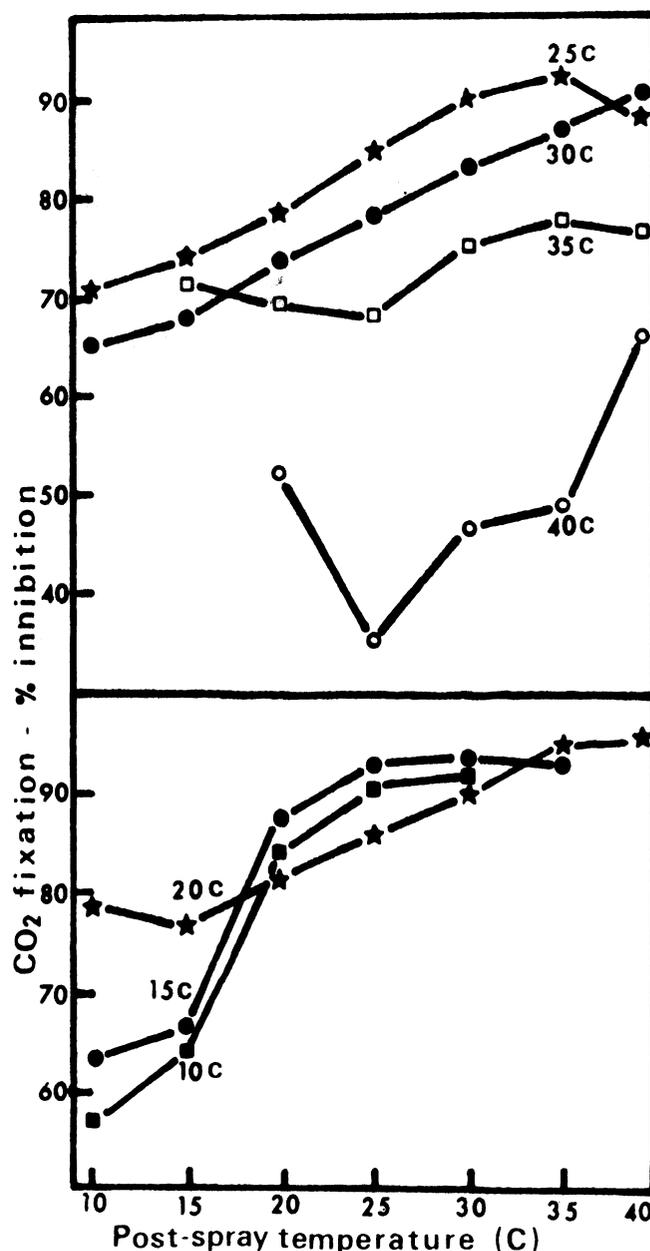

Figure 2. Effect of postspray temperatures on CO_2 fixation following treatment with 1.1 kg/ha of desmedipham. Prespray temperatures are indicated adjacent to appropriate curves.

when transferred to 15 or 20 C. Decreasing inhibition was observed for increasing prespray temperatures (Figure 3). The interaction between prespray and postspray temperatures was highly significant at the lower temperatures and significant at the higher temperatures. The prespray temperature and postspray temperature main effects were highly significant and not signi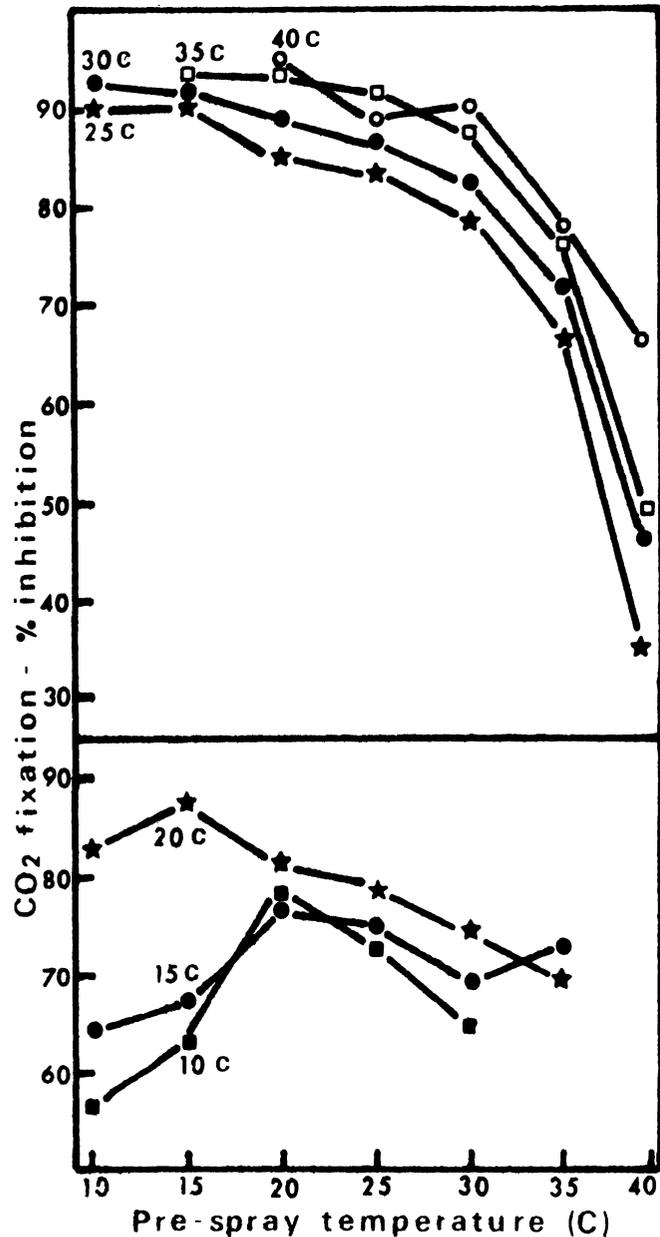ficant, respectively. This indicated that the differences between the responses to postspray temperatures varied with prespray temperatures, but not vice versa, and that the responses were more pronounced at lower prespray temperatures. In general, the more temperatures were decreased following spraying the lower was the inhibition of photosynthesis (Figure 4, A). Conversely, the greater the increase in temperature following treatment the greater the inhibition (Figure 4, B). Elevation of temperature produced more consistent inhibition than lowering of temperature decreased inhibition.

Temperatures optimal for growth in sugarbeets caused maximal inhibition in desmedipham treated plants. This indicates that temperature is a major influence on the herbicidal activity. When temperature was increased immediately after spraying with desmedipham, injury increased. When temperature decreased injury decreased. Sugarbeet plants grown at high temperatures were more resistant to the increased des-
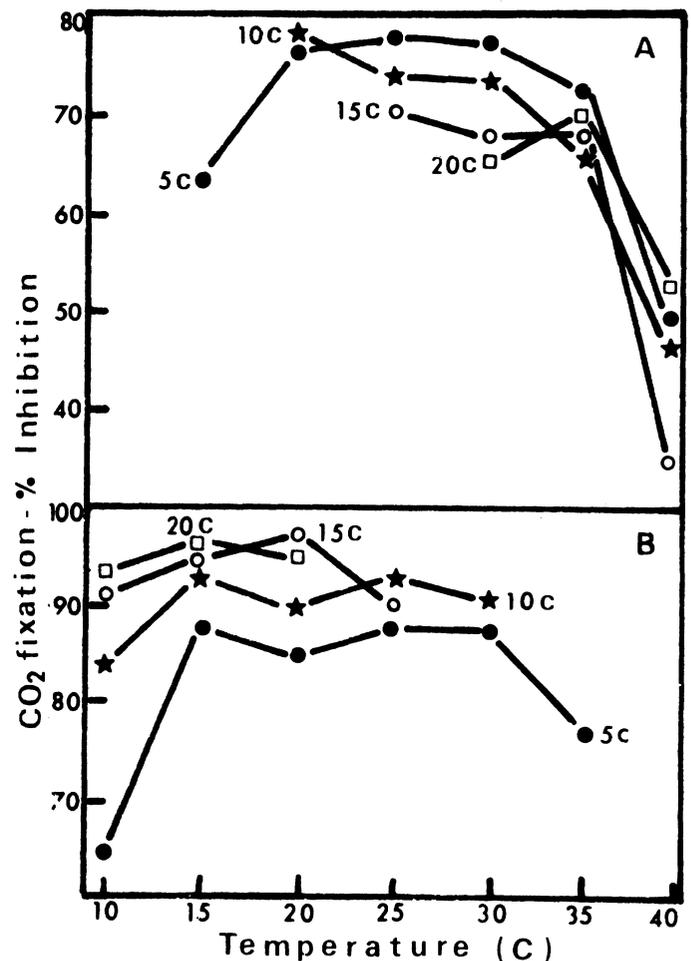

Figure 3. Effect of prespray temperatures on CO_2 fixation following treatment with 1.1 kg/ha of desmedipham. Postspray temperatures are indicated adjacent to appropriate line.

Figure 4. Effect of difference between prespray and postspray temperatures on CO_2 fixation following treatment with 1.1 kg/ha of desmedipham. Temperatures adjacent to curves indicated degrees by which the temperatures were (A) lowered or (B) raised following treatment.

medipham injury caused by raised postspray temperatures than plants grown at lower temperatures.

When plants were not subjected to postspray temperature changes, the colder and warmer temperature regimes showed relatively low inhibition, and the optimal growth temperature increased inhibition. In plants grown at 10 to 40 C and subjected to 25 to 30 C after spraying high inhibition was observed only in the plants grown at the lower temperatures. When 35 or 40 C were used, low inhibition occurred only in plants grown at high temperatures. An asymmetrical correlation between the growth temperature and the postspray temperature existed. The role of prespray temperatures in modifying the activity of desmedipham may be in anatomical and physiological adaptation (9). Temperature-dependent modifications of epidermis, and of enzyme activities are examples. Postspray temperatures affect the physiological processes of the plant.

When the temperature rise after spraying was delayed, inhibition decreased with increasing time (Figure 5). The process most affected by the change is expected to be one whose contribution to toxicity is greatest immediaely after spraying. Penetration is the most likely process (7).

Exposure of sprayed plants to increasing light intensities resulted in increased inhibition (Figure 6). The inhibition decreased with longer exposure times. Although this trend was confirmed by two different assay methods neither the light intensity-exposure time interaction, nor the light intensity main effect was significant. The main effect for exposure time was significant. Increased toxicity with higher light intensities may be regarded as an effect separate from temperature, as

leaf to air temperature differences were less than 1 C at all light intensities. The increase in phytotoxicity with increased light intensity, found also for other herbicides (2), indicates the mechanism of toxicity (10) to be the process most affected by light intensity. Temperature and light intensity modification of desmedipham toxicity generally paralleled those observed previously for phenmedipham toxicity (1, 2, 3).

ACKNOWLEDGMENTS

LITERATURE CITED

1. Arndt, F., R. Rush, H. Lenzner, and K.V. Gierke. 1970. Die Beeinflussing der Selektivität von Phenmedipham durch verschiedene Faktoren. Z. Pflanzenkr. Sonderheft V:89–93.
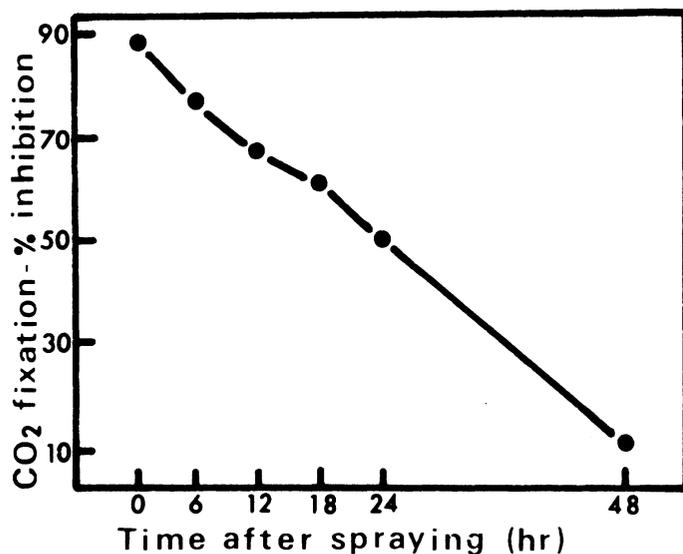

Figure 5. Influence of delaying temperature rise on CO_2 fixation in sugarbeets following treatment with 1.1 kg/ha of desmedipham. Plants were grown at 25 C and transferred to 40 C following spraying.
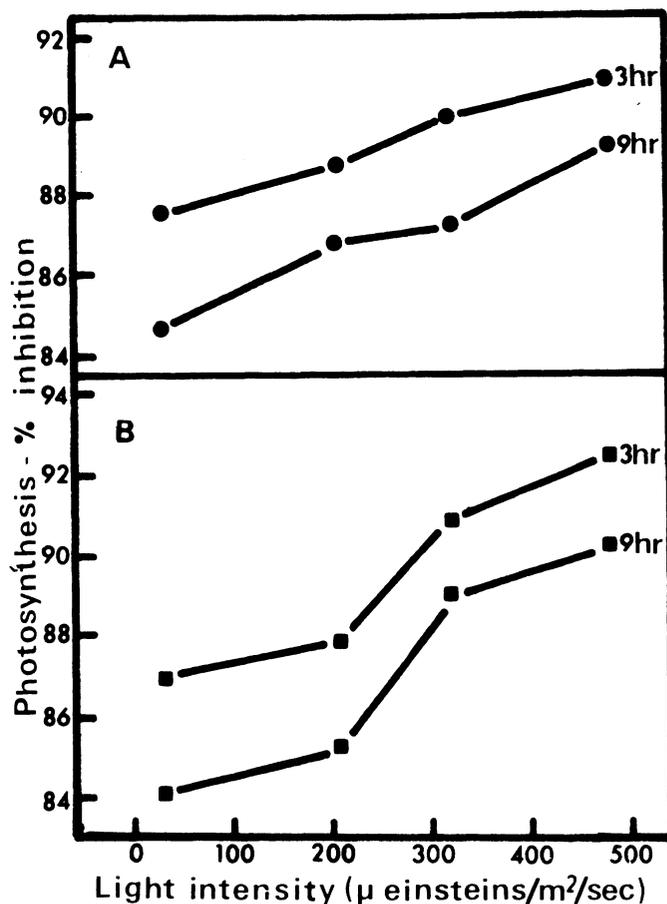

Figure 6. Effect of light intensity on level of photosynthesis inhibition in sugarbeets following application of 1.1 kg/ha of desmedipham. Inhibition was determined by (A) $^{14}CO_2$ exposure and by (B) infrared gas analysis.

2. Ashton, F.M. 1965. Relationship between light and toxicity symptoms caused by atrazine and monuron. Weeds 13:164–169.

3. Bischof, F. von, W. Koch, J.C. Majumdar, and F. Schwerdtle. 1970. Retention, penetration und Verlust von Phenmedipham in Abhangigkeit von einigen Faktoren. Z. Pflanzenkr. Sonderheft V:95–102.

4. Gaastra, P. 1959. Photosynthesis of crop plants as influenced by light, carbon dioxide, temperature and stomatal diffusion resistance. Meded. Landbouwhogeschool, Wageningen 59(13): 1–69.

5. Hendrick, L.W., W.F. Meggitt, and D. Penner. 1974. Basis for selectivity of Phenmedipham and desmedipham on wild mustard, redwood pigweed, and sugar beet. Weed Sci. 22:179–184.

6. Hoagland, D.R. and D.I. Arnon. 1950. The water culture method of growing plants without soil. Calif. Agr. Exp. Sta. Circ. 374. 17 pp.

7. Kassebeer, H. 1970. Aufnahmegeschwindigkeit, Metabolismus, und Verlagerung von Phenmedipham bei verschieden empfindlichen Pflanzen. Z. Pflanzenkr. 79:158–174.

8. Laufersweiler, H. and C.M. Gates. 1972. Response of weeds and sugarbeets to EP-475, a phenmedipham analog. J. Am. Soc. Sugarbeet Tech. 17:53–57.

9. Levitt, J. 1972. Response of Plants to Environmental Stress. Academic Press, New York. pp. 272–321.

10. Stanger, C.E. and A.P. Appleby. 1972. A proposed mechanism for diuron-induced phytotoxicity. Weed Sci. 20:357–363.

11. Trebst, A., E. Pistorius, G. Boroschewski, and H. Schultz. 1968. Die Hemmung photosynthetischer Herbicide des Biscarbamat Typs. Z. Naturforsch. 23b:342–348.