Eastern Redbud (*Cercis canadensis*) Toxicity with Increasing Rates of Sulfentrazone

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Sulfentrazone, an experimental herbicide from the FMC Corporation, has shown promising results for long-term weed control in field trials with ornamentals by controlling weeds such as morningglory and yellow nutsedge that are difficult to manage with currently labeled products (Collins et al., 1996). This compound has recently been labeled for use in both soybeans and tobacco. A possible hindrance to the labeling of sulfentrazone for use in ornamentals is the phytotoxicity that occurs in certain sensitive ornamental species.

Sulfentrazone works by inhibiting protoporphyrinogen oxidase in the chlorophyll biosynthetic pathway in susceptible plants. As a result, a phytodynamic toxicant (protoporphyrin IX) builds up, leading to membrane disruption. Sulfentrazone is absorbed by both the roots and shoots of plants, which turn necrotic and die shortly after exposure to light. Postemergence application of sulfentrazone, resulting in foliar contact of weeds, can cause rapid desiccation and necrosis in affected species, particularly smaller ones (Theodoridis et al., 1992; Van Saun et al., 1991).

In a field trial using sulfentrazone during 1996, *Cercis canadensis* exhibited foliar damage, but phytotoxicity ratings were not noted because the damage resembled *Botryosphaeria* canker, a common disease of redbuds in this area. After much discussion about that trial, an experiment was designed to determine if sulfentrazone had detrimental effects on *C. canadensis*, and if increasing rates were related to increased phytotoxicity.

Cercis canadensis liners measuring 18 to 24 inches were planted in 3-gal containers in April 1997 using a medium consisting of pine bark and expanded shale (2:1, v/v). The plants were allowed to leaf out completely and establish new roots. Sulfentrazone 80WP was applied in May 1997 at increasing rates for a total of six treatments (0.125, 0.25, 0.375, 0.5, and 0.625 lb ai acre $^{-1}$, plus a control). Ten single plant reps were used for each treatment, arranged in a completely randomized design of 60 plants. Plants were watered using trickle irrigation. Treatments were applied using a $\rm CO_2$ -pressurized backpack sprayer calibrated to 26 GPA using 8004 nozzles at 30 lb psi at the boom. Plant phytotoxicity ratings were taken weekly after herbicide application, measured on a scale from 0 to 10 (0 representing no phytotoxicity and 10 representing plant death). Plants were harvested 6 weeks after treatment (WAT). At that time, a visual root rating was taken on a scale from 0 to 10 (0 representing no root damage compared to the control and 10 representing plant death). Shoot and root dry weights were determined after drying for 2 days in a 40C oven.

Phytotoxicity ratings are reported in Table 1 and Fig. 1. Phytotoxicity appeared as a reddish-brown necrotic area around the leaf margin, eventually spreading throughout the whole leaf. A more severe form of phytotoxicity was exhibited as current season stem death in some plants. At 1 WAT, phytotoxicity increased with increasing sulfentrazone rates as expected. At 2 WAT, we observed increasing

Table 1. Phytotoxicity ratings.	0				
Treatment	Rate	1 WAT	2 WAT	3 WAT	4 WAT
Sulfentrazone 80WP	0.125	2.9 c	2.2 c	2.7 c	3.6 b
Sulfentrazone 80WP	0.250	3.1 c	3.1 с	3.7 bc	4.2 ab
Sulfentrazone 80WP	0.375	4.7 b	3.2 bc	3.9 abc	4.1 ab
Sulfentrazone 80WP	0.500	5.7 ab	4.4 ab	4.7 ab	4.7 a

4.8 a 0.0 c 0.95

5.1 a 0.0 d 1.28

5.2a 0.0 d 1.29

6.6 a 0.0 d 1.54

 $0.625 \\ 0.000$

Sulfentrazone 80WP

LSD at P < 0.05

Control

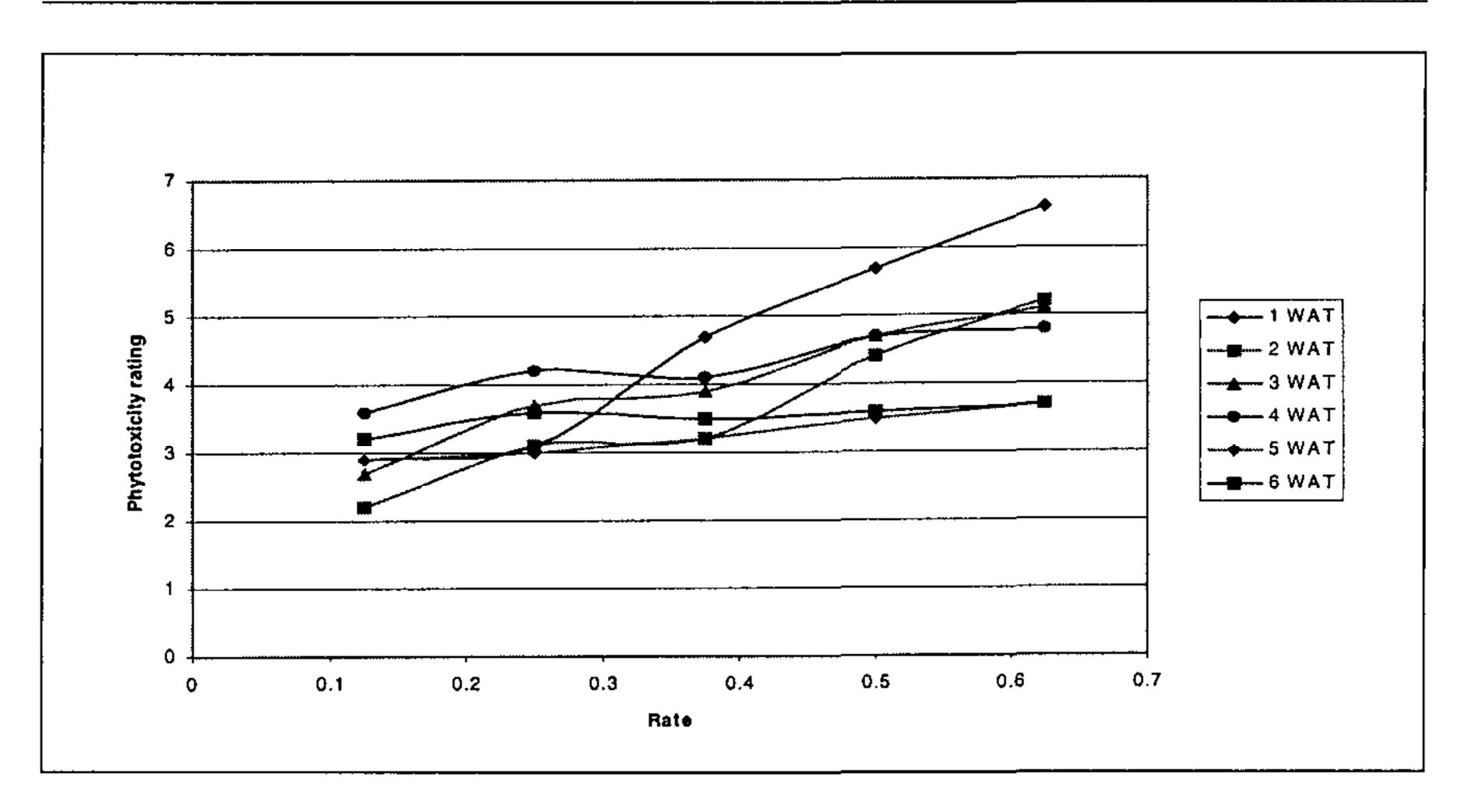


Figure 1. Sulfentrazone toxicity on Cercis canadensis.

phytotoxicity with increasing rates, but this response was not as linear or exponential with increasing rate. This phenomenon was due to the fact that the more severely damaged plants were producing new growth which was undamaged, while the plants that initially had less damage were exhibiting increasing toxicity. By 4 WAT, response was similar among all treatment combinations, with a difference of only 1.2 between the lowest and highest rates. The higher rates had a considerable amount of new growth by this time, thereby negating some of the initial negative effects of sulfentrazone. An explanation for the continued decline of the plants sprayed with lower rates could be that the higher rate effects were initially so severe that the stressed plants temporarily shut down growth and translocation processes, no longer absorbing the herbicide, while the plants sprayed with lower rates continued to absorb the herbicide readily, allowing continued translocation throughout the plant. By 5 and 6 WAT, there were no significant differences among the rates of sulfentrazone. The phytotoxicity ratings were slightly higher at 6 WAT, due to abnormal growth noticed on the new leaves of plants in all treatments, indicating sulfentrazone phytotoxicity persisted 6 WAT from visual analysis.

Table 2. Root and shoot evaluation.

Treatment	Rate	Root rating	Root dry weight(g)	Shoot dry weight(g)
Sulfentrazone 80WP	0.125	5.4 c	7.86 b	13.76 ab
Sulfentrazone 80WP	0.250	$6.0 \ bc$	7.86 b	11.19 bc
Sulfentrazone 80WP	0.375	7.3 ab	4.65 b	7.85 cd
Sulfentrazone 80WP	0.500	7.7 a	3.88 b	6.02 d
Sulfentrazone 80WP	0.625	6.4 abc	6.75 b	9.17 bcc
Control	0.000	0.0 d	13.88 a	17.97 a
LSD at P < 0.05	_	1.64	4.40	4.87

At 6 WAT, plants were harvested and a visual root rating was given to each plant (Table 2). Roots were then separated from shoots and placed in a drying oven for 2 days. At this time, root and shoot dry weights were measured in grams (Table 2). The visual root rating increased with increasing sulfentrazone rates, but dropped at the highest rate. The shoot dry weight followed a similar trend, decreasing with increasing sulfentrazone rates, but increased again at the highest rate. These observations could be attributed to the fact that the plants sprayed with the highest rate of sulfentrazone started to show new growth earliest after the initial treatment, and had the most time to regenerate. The root dry weights showed no significant difference among sulfentrazone treatments, although there were differences visually. Future experiments could utilize more replications to evaluate treatment effects.

LITERATURE CITED

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