Silica Sprays Reduce the Incidence and Severity of Bract Necrosis in Poinsettia

Richard J. McAvoy and Bernard B. Bible
Department of Plant Science, University of Connecticut, Storrs, CT 06269-4067

Additional index words. sodium silicate, calcium, Euphorbia pulcherrima

Abstract. Silica sprays (Na$_2$SiO$_3$ or Si$_2$O$_5$H$_2$O) markedly reduced the incidence and severity of bract necrosis (BN) of Euphorbia pulcherrima Willd. cv. Supjibi Red compared to plants not sprayed with silica. BN has been associated with low Ca concentrations or high K : Ca ratios in tissues of bract margins. Silica had no effect on Ca or K concentrations in bract margin tissues, and BN was not associated with the macro- or micronutrient composition of bract margin tissues. Sixteen days after initial anthesis, nontreated and deionized-water-sprayed poinsettias developed a higher incidence of BN than did plants sprayed with Na$_2$SiO$_3$ or CaCl$_2$. However, sprays of 3.56, 5.34, and 7.12 mM Na$_2$SiO$_3$ were as effective as 9.98 mM CaCl$_2$ sprays in protecting against BN of ‘Supjibi Red’ and ‘Angelika White’ bracts for up to 30 days after initial anthesis. ‘Supjibi Red’ developed a higher incidence of bract necrosis than did ‘Angelika White’, but both cultivars showed a similar response to the treatments and similar symptoms of necrosis. In both cultivars, initial symptoms appeared as small necrotic lesions on bracts at the looped ends of lateral veins that displayed a closed-vein pattern after the plants reached initial anthesis.

Bract necrosis (BN) is a serious disorder of the poinsettia, characterized by small brown spots or lesions near the bract margins that begin to appear soon after plants reach initial anthesis (Moe et al., 1992; Nell and Barrett, 1986). As symptoms progress, the spots enlarge, darken, and the number of bracts affected increases until the appearance of the poinsettia is seriously degraded. Stromme et al. (1994) observed a Ca gradient in poinsettia bracts with higher concentrations in midportions of bracts and lower concentrations in the bract margins. Woltz and Harbaugh (1986) demonstrated that Ca sprays, applied to bracts during their development, suppressed BN in ‘V-14 Glory’ bracts. They concluded that a deficiency of Ca in bract margins caused BN.

At first glance, BN of poinsettia is another instance of a disorder resulting from localized Ca deficiencies, but is it? Localized Ca deficiencies cause necrosis in the tissues of various plant species, including leaf-edge burn of the poinsettia (Bierman et al., 1990), blackheart of young celery (Apium graveolens L.), leaves (Bible and Stiehl, 1986), and tipburn of young Chinese cabbage (Brassica rapa L. Pekinensis Group) leaves (Van Berkel, 1988). The one common characteristic in these three examples is that the necrosis appears on young tissues during rapid cell expansion. However, with BN, it is the mature bracts that show the first evidence of necrosis as they begin to age, at or shortly after anthesis (McAvoy and Bible, 1994).

Elevated nighttime humidity suppresses blackheart of celery (Bible and Stiehl, 1986) and tipburn of Chinese cabbage (Van Berkel, 1988), whereas it does not suppress BN of the poinsettia (Stromme et al., 1994). Furthermore, Nell and Barrett (1986) reported that water stress (to wilt) applied to poinsettias during bract development did not affect the incidence of BN, unlike water stress induced edge burn of poinsettia leaves (Jacques et al., 1990). In fact, when poinsettias were exposed to abundant water and high fertilizer levels, applied from the onset of bract coloration until anthesis, BN increased (Nell and Barrett, 1986). Although Ca sprays suppress BN, the fundamental cause of this poinsettia disorder is unclear.

The symptoms of BN are similar to the necrotic spots on the margins of lettuce (Lactuca sativa L.) leaves. This lettuce disorder can be suppressed by silicate applications (Blatt and Van Diest, 1981). Also, silica treatments suppress the development of necrotic freckling on sugar cane (a complex trispecies hybrid of Saccharum) leaves (Elawad et al., 1982) and barley (Hordeum vulgare) leaves (Williams and Vlamis, 1957). Based on the nature of the symptoms, we speculated that silicate applications might reduce BN in poinsettia. The objectives of our study were to determine the effects of silica treatments on BN, and compare the efficacy of silica, over time, to the standard calcium chloride (CaCl$_2$) spray treatment.

Materials and Methods

Experiments were conducted in a glass greenhouse with temperature set points main-
bract with most expanded beyond the affected vein; \( s = 11 \) to 15 spots per bract; \( 6 = \) greater than 15 spots per bract. The ratings were multiplied by the number of bracts in each category, and the sum of these values \((>100)\) was divided by the total number of bracts to obtain the total necrosis index (TNI). This value represents the incidence of bract necrosis weighted for severity; a TNI value of 100 would represent no damage and a value of 600 would represent a plant with category 6 damage on all bracts. The TNI is the best measure of overall poinsettia damage because it combines both the severity rating and the percent incidence in a single index.

After bracts were evaluated for damage, the tissue was processed for nutrient analysis. Bract samples were washed with 0.1 M HCl for 60 s and then rinsed in deionized water. Tissue from a 1- to 1.5-cm-wide strip along each bract margin was collected from all bracts on each plant and dried at 70°C. Because the quantity of tissue from each plant was too small for complete nutrient analysis, samples from adjoining replicated blocks were combined to yield four samples for each treatment group. Samples were then ground to pass a 0.25-mm²-pore mesh screen, and analyzed for mineral nutrient content with an ICP-emission spectrophotometer (Thermo Jarrell Ash, Franklin, Mass.) using the dry ash procedure (Cornell Univ., Fruit and Vegetable Science Analytical Lab, Ithaca, N.Y.).

‘Supjibi Red’ and ‘Angelika White’—Na\(_2\)SiO\(_3\) or CaCl\(_2\) sprays (Expt. 2). In 1994/95, ‘Supjibi Red’ and ‘Angelika White’ were transplanted into 1.6-L pots on 8 Aug. 1994 and subsequently pinched on 2 Sept. leaving four to five nodes per plant. Photoperiod lighting (2 \( \mu\text{mol-m}^{-2}\text{-s}^{-1} \)) was provided from 2200 to 0200 h nightly from 30 Aug. to 26 Sept. All plants were fertilized on a constant basis using N at 14.3 mol·m\(^{-3}\a0) from a 15N–2.2P–14.9 mol·m\(^{-3}\) (200 mg·L\(^{-1}\)) of Ca(NO\(_3\))\(_2\) every third irrigation. Sodium silicate sprays increased the Na content of bract margin tissue relative to the nontreated plants (0.13% vs. 0.04% of dry mass, respectively); the Na\(_2\)SiO\(_3\) drench did not affect the Na concentration in bract margins (0.04% of dry mass). There was no effect of the 17.8 mmNa\(_2\)SiO\(_3\) spray or drench on contents of macro- or micronutrients in the bract margin tissue other than Na. The average macronutrient levels measured in bract margins were (all in \( \mu\text{g g}^{-1} \) of dry mass) 17.5 for Mn, 89 for Fe, 23.9 for Cu, 49.2 for Zn, 5.5 for Mo, and 26.5 for B; AI averaged 109.

‘Supjibi Red’ and ‘Angelika White’—Na\(_2\)SiO\(_3\) or CaCl\(_2\) sprays (Expt. 2). The incidence and severity of BN was slightly higher on ‘Supjibi Red’ than on ‘Angelika White’, but both cultivars showed a similar response to treatments (Table 1) and similar symptoms of BN (Fig. 2). In both cultivars, initial symp- toms appeared as small necrotic lesions at the looped ends of lateral veins that displayed the closed-vein pattern after the plants had reached anthesis.

Poinsettias not sprayed with Na\(_2\)SiO\(_3\) or CaCl\(_2\) were the most prone to BN (Fig. 3). As in Expt. 1, necrotic lesions first began to appear on mature bracts in the weeks following initial anthesis. By 16 days after initial anhe- sis, 5.7% of the bracts on nontreated controls had developed necrotic lesions while only 0.2% of the bracts on Na\(_2\)SiO\(_3\)-sprayed plants had developed necrotic spots by this date. Once BN began to develop, the percentage of affected bracts on poinsettias in all three treatment groups increased at a steady linear rate over time (\( r^2 = 0.99, 0.98, 0.93 \) for the nonsprayed, Na\(_2\)SiO\(_3\)-drenched, and Na\(_2\)SiO\(_3\)-sprayed plants, respectively). However, the rate of increase in the incidence of BN was much higher for the nonsprayed and Na\(_2\)SiO\(_3\)-drenched plants (9% and 12% per week, respectively) than for the Na\(_2\)SiO\(_3\)-sprayed plants (4.5% per week).

At harvest, Na\(_2\)SiO\(_3\) sprays had reduced both the incidence and severity of BN compared to nontreated plants. The total necrosis index (TNI = 129) for Na\(_2\)SiO\(_3\)-sprayed plants was 39% lower than for the nontreated plants (TNI = 212) and 49% lower than for the plants drenched with Na\(_2\)SiO\(_3\) (TNI = 255). Thus, drenching the potting medium with a 17.8 mm Na\(_2\)SiO\(_3\) solution was ineffective for control- ling BN.

Discussion

Our results clearly show that silica sprays markedly suppress BN. In a study we conducted in 1994, meta-silicic acid (SiO\(_2\)-\( \text{H}_2\text{O} \)) suppressed BN as effectively as Na\(_2\)SiO\(_3\), indicating that silica is the active agent and not Na (data not shown). Our results also support previous reports on the effectiveness of Ca sprays in suppressing BN (Meinken and Fischer, 1991; Wissmeyer et al., 1992; Woltz and Harbaugh, 1986).

Stromme et al. (1994) associated BN with a Ca concentration <0.16% dry mass and a K : Ca ratio >20 in bract margin tissue of ‘Lilo’. In our Expt. 1, the nontreated and the medium drenched ‘Supjibi Red’ plants had Ca concentrations >0.19% dry mass in bract margin tissue and K : Ca ratios <20. Nevertheless, the bracts had a high incidence and acute severity of BN. Sprays of sodium silicate protected against BN, even though Ca and K levels in bract margins were similar in plants from all treatments. Thus, the protective effect of silica was not related to alteration of Ca or K concentrations in bract margin tissue.

While it is clear that silica sprays at rates as low as 3.56 mm are as effective as 9.98 mm Ca sprays at reducing the incidence and severity of BN in poinsettias for up to 30 days after initial anthesis, it is not clear how either treat- ment works. Although not considered an es- sential nutrient for most terrestrial plants (Epstein, 1994), silica altered the microdistribution of Mn in barley (Lewin and Reimann, 1969) and bean (Phaseolus vulgaris L.) leaves (Horst and Marschner, 1978), and ameliorated Al toxicity (Baylis et al., 1994). In cucumber (Cucumis sativus L.), silica also moderated the effects of a P : Zn imbalance (Marschner et al., 1990). However, in our study, there was no indication of toxic concentra- tions of Mn or Al in tissues of bract margins of nontreated plants. Neither was there an imbalance of P and Zn in bract margin tissues that could be associated with the BN. Unless
small areas of nutrient concentration, not measurable by our sampling technique, give rise to necrotic spots, BN was not associated with the concentrations of any macro- or micronutrient in tissues of bract margins.

Nell and Barrett (1986) postulated salt transport through the pitted vein ending could lead to BN. Blom (1994) suggested that increased BN may be associated with guttation. This evidence is consistent with the slight protection observed against BN afforded by the deionized water spray (Table 1). Deionized water spray could have a diluting effect or the surfactant could disperse toxic droplets, thereby reducing the risk of localized damage (Neumann and Prinz, 1975). While linking of BN with guttation is intriguing, we have never observed guttation on poinsettia bracts and Metcalfe and Chalk (1979) do not list any of the genera in the family Euphorbiaceae as having hyathodes. Furthermore, Van Berkel (1988) observed the least tipburn in Chinese cabbage plants that guttate the most.

The symptoms of BN are distinct from those caused by localized Ca deficiencies in green leafy tissues in that the first visible evidence of necrosis occurs after cell expansion is completed. In our studies, initial symptoms of BN appeared after poinsettia reached anthesis (Figs. 1 and 3). Others (Nell and Barrett, 1986; Wolz and Harbaugh, 1986) observed initial BN at a similar stage of bract development. These observations do not rule out the disorder occurring earlier in bract development, but not manifesting itself until bracts mature. On both ‘Supjibi Red’ and ‘Angelika White’ bracts, BN appeared at the looped ends of lateral veins that displayed the closed-vein pattern (Fig. 2). However, Nell and Barrett (1986) observed that necrotic lesions first appeared on ‘V-14 Glory’ bracts at lateral veins that ended in a blind-end pattern; damage rarely occurred on the veins that had a closed-end or loop pattern.

With localized Ca deficiencies in crops such as celery and Chinese cabbage, visible necrosis occurs in rapidly expanding leaf tissue (Shear, 1975). With these developmental disorders, impaired membrane function—resulting in membrane leakiness—causes the tissue damage (Bangerth, 1979). If BN is primarily a Ca deficiency disorder caused by impaired membrane function during bract development, then, to suppress this disorder, silica would have to substitute for Ca. We know of no reports of silicates substituting for the metabolic or structural functions of intracellular Ca in higher plants. This fact is not surprising in that silica and Ca are dissimilar chemical species.

With topical application of Ca, there is a question as to whether it actually enters the cytosol. The concentration of intracellular Ca is low (submicromolar) and carefully regulated by the cell. In contrast, extracellular Ca concentrations are in the millimolar range (Helper and Wayne, 1985). Extracellular Ca concentrates in the cell wall and middle lamella, strengthening the walls; however, cell wall integrity is less of a problem with developmental Ca disorders and more important for disease resistance. Silica can contribute to cell wall strength and it has been used to suppress powdery mildews in cucumber (Menzies et al., 1992) and grapes (Vitis vinifera L.) (Bowen et al., 1992). Or, by strengthening cell walls, silica and Ca may be preventing laticifer rupture (Tibbitts et al., 1985) and consequent damage analogous to tipburn of lettuce.

Whatever the etiology of BN, if it is a Ca deficiency disorder, then we have reported the first instance of silica partially rectifying a deficiency symptom of Ca. Finally, while silica is not a cure for this disorder, investigating its mode of action may give further clues into the nature of BN.

### Literature Cited


Blom, T.J. 1994. Bract edge burn on poinsettias. Cana...


Fig. 2. Necrotic lesions on poinsettia bracts typical of bract necrosis. (top) Schematic representation of a poinsettia bract showing necrotic lesions developing at the looped-end of lateral veins near the bract margins, (bottom left) photograph of ‘Angelika White’ bract with necrotic lesions near lateral veins on bract margin, (bottom right) photograph of ‘Supjibi’ with necrotic lesion beginning to develop on looped-end of lateral vein.

Fig. 3. Development of bract necrosis (percentage of total bracts with necrotic spots) over time for nontreated poinsettias, or poinsettias sprayed with CaCl₂ (9.98 mM), or Na₂SiO₃ (1.78, 3.56, 5.34, or 7.12 mM), or deionized water. Data represent mean values for the cultivars ‘Supjibi Red’ and ‘Angelika White’; both cultivars showed a similar response to treatments. LSD values for each sample date were 1.4 (day 16), 2.2 (day 23), 3.3 (day 30), 3.4 (day 37), 3.7 (day 42), and 4.1 (day 47).