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Calcium Deficiency as the Basic Cause of Marginal Bract Necrosis of 'Gutbier V-14 Glory' Poinsettia

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Abstract. A hypothesis was tested that the cause of a damaging marginal bract necrosis common to 'Gutbier V-14 Glory' poinsettia is a deficiency of Ca that is expressed when the rapidly growing bract outdistances the current supply of Ca. Plants were grown in the greenhouse in a sedge peat-EauGallie sand mix with fertility adjustments designed to moderately enhance Ca deficiency. Twice-weekly foliar applications of Ca (432 ppm) were begun at the stage of first bract coloration to attempt to prevent necrosis. In addition to bract necrosis, puckering of bracts occurred on controls but not in Ca-treated plants. In another experiment, induced Ca deficiency symptomology included bract necrosis. Calcium chloride spray essentially prevented the necrosis. Necrotic bract marginal tissue had 0.07% Ca, whereas sprayed tissue contained more than three times this Ca concentration.

Struckmeyer (4) described Ca deficiency syndrome for poinsettia: small leaves, necrotic lesions, and a cupping or curling under of margins of upper leaves. The terminal growing point failed to continue growth, resulting in stunted plants. She made no mention of symptoms on bracts or other flower parts.

Wilfret (5) pointed out in 1981 that 'Gutbier V-14 Glory' was a new cultivar that showed great promise for central Florida if the often severe marginal bract necrosis problem could be controlled. Nell and Barrett (1, 3) found that 'Gutbier V-14 Glory' plants had the most bract necrosis when plants were irrigated frequently and fertilized heavily during bract coloration; bract necrosis was greater in plants receiving 100% NH₄ in con-

trast to those receiving half NO₃ and half NH₄ (2). Based on these published observations and the symptomology of the disorder, we hypothesized that bract necrosis might be a Ca deficiency-related disorder. Two experiments were conducted to test this hypothesis—first, comparing Ca spray chemicals, and 2nd, using soil fertility conditions predisposing to Ca deficiency with and without preventive foliar nutritional sprays.

In the first experiment, a steam-pasteurized mixture of EauGallie fine sand : Florida sedge peat (1:1, v/v) was amended with 1.0 kg technical grade chemical CaCO₃ powder and 2.5 kg technical grade MgCO₃ powder/m³. Diammonium phosphate, K₂SO₄, and MgSO₄·7H₂O at 0.25 kg·m⁻³ each and Perk at 0.75 kg·m⁻³ also were included in the mix. Single 'Gutbier V-14 Glory' rooted cuttings were planted into 15-cm plastic pots 5 Sept. 1984. Plants were pinched one week after planting and grown to flowering without daylength adjustment.

Calcium treatment blocks, each with 14 replicate plants, were sprayed twice weekly with four Ca source compounds (Table 1) from the first appearance of red color in the bracts (2 Nov.) until termination of the ex-

periment (23 Jan.). Calcium nutritional sprays provided 432 ppm Ca from all sources. In addition to three chemical grade compounds, Ca phenolate (This Calcium, Stoller Chemical, Houston) also was employed as a Ca source.

Calcium sprays were about equally effective in the prevention of bract necrosis as well as puckering, cupping, and arching. Apparently the growth rate of bract laminar tissue, especially at the margins, was maintained at a normal rate by Ca sprays, whereas the lack of Ca was accompanied by uneven growth, noticeably restricted at the margins as manifested by puckering, cupping, and measurable bract arching (Table 1). Puckering was rated 0-10, none to most severe, as a visible wrinkling and folding of laminar tissue. Cupping was evidenced by a concave appearance of the bract when viewing the abaxial surface and was rated on the same 0-10 scale. Arching caused by an elevation of the midrib evident when the bract was laid on a flat surface was measured as an objective index of the apparent effect of Ca deficiency on laminar expansion at the bract margin (apparently cupping was also induced by a failure of margin laminar tissue to expand normally). Pot plant appearance was also rated on the 0-10 scale. All quality comparisons indicated favorable response to Ca sprays. Calcium contents showed elevated Ca (0.15% to 0.27%) in marginal tissue of the floral bracts washed with dilute acetic acid, pH 4.5, due to Ca sprays, while the unsprayed control had very low Ca (0.07%). Calcium acetate produced a lower bract Ca value than the other Ca sprays, while This Calcium produced slightly higher Ca contents. The data in Table 1 suggest that bract necrosis occurrence is related to inadequate Ca supply to the bract margin and the Ca sprays should be efficacious in prevention of the disorder. The question of what soil and spray treatments would control most practically remains to be answered.

In a 2nd experiment, a steam-pasteurized 1:1 mixture of EauGallie fine sand and Florida sedge peat was amended with different liming materials (Table 2) to control Ca availability. Single rooted cuttings of 'Gutbier V-14 Glory' were planted in 18-cm plastic pots 30 Aug. 1984. Plants were grown without supplemental light or shade and were pinched 13 Sept.

Substitution of MgCO₃ for CaCO₃ in liming induced what appeared to be Ca deficiency, which was only partially corrected

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Table 1. Effect of Ca spray treatments on aberrant metabolism symptom expression of 'Gutbier V-14 Glory' poinsettias.

	Marginal bract necrosis ^z		Bract puckering ^y	Bract cupping ^y	Bract arching (cm)	Bract necrosis rating ^y	Pot damage rating ^y	Ca bract margin dry wt (%)
	10 Dec. 1984	27 Dec. 1984						
Unsprayed	5.9 a*	23.6 a	5.6 a	7.1 a	2.5 a	3.2 a	8.3 a	0.07 d
CaCl ₂ ^w	0.7 b	0.9 b	2.9 b	3.4 b	0.8 b	0.0 b	1.5 b	0.24 b
Ca(NO ₃) ₂	0.1 b	0.4 b	3.0 b	3.4 b	0.5 b	0.2 b	1.5 b	0.23 b
This Calcium	0.4 b	0.3 b	2.1 b	2.4 b	0.7 b	0.2 b	1.5 b	0.27 a
Ca acetate	0.4 b	0.8 b	1.8 b	3.2 b	0.8 b	0.2 b	1.3 b	0.15 c

^zMean number of bracts affected per pot.

^ySubjective rating: 0 = none, 10 = most.

^xMean separation in each category of data by Duncan's multiple range test, $P = 5\%$.

^wAll four calcium treatments sprayed at concentrations of 432 ppm Ca.

Table 2. Effect of soil and foliar Ca treatments on growth, aberrant metabolism symptoms and Ca content of 'Gutbier V-14 Glory' poinsettias.

Treatment no.	CaCO ₃ (kg·m ⁻³)		Marginal leaf necrosis (No. leaves/pot)		Marginal bract necrosis (No. bracts/pot)		Shoot fresh weight (g)		Root fresh weight (g)		% Ca in young leaves 5 Nov. 1984		% Ca in bract margins 11 Jan. 1985	
	MgCO ₃ (kg·m ⁻³)		- Ca ^x	+ Ca ^x	- Ca	+ Ca	- Ca	+ Ca	- Ca	+ Ca	- Ca	+ Ca	- Ca	+ Ca
	1 ^y	0	3.4	40 a ^w	50 e	21 bc	2.0 fg	12 e	22 e	8 e	9 e	0.15 d	0.28 d	0.085 c
2	0.5	2.9	25 b	0 e	25 a	0.0 g	97 d	130 cd	22 d	29 cd	0.30 d	0.32 d	0.074 c	0.247 b
3	1.0	2.5	24 b	1 e	31 a	0.6 g	195 ab	193 ab	39 abc	39 abc	0.55 c	0.63 abc	0.075 c	0.263 ab
4	2.0	1.7	9 d	1 e	13 cde	1.0 g	212 abc	185 abc	43 ab	44 ab	0.71 abc	0.79 ab	0.067 c	0.262 ab
5	4.0	0	6 de	1 e	6 efg	0.0 g	158 abc	182 abc	33 bc	40 ab	0.61 c	0.80 a	0.070 c	0.330 ab
6	1 × cc ^z		9 d	0.4 e	10 def	0.4 g	175 abc	153 ab	46 a	38 abc	0.62 bc	0.70 abc	0.065 c	0.212 b
7	2 × cc		10 d	0.2 e	13 cde	0.4 g	181 abc	198 abc	42 ab	47 a	0.58 c	0.67 abc	0.067 c	0.248 b
8	4 × cc		17 c	0.2 e	18 bcd	0.2 g	192 ab	198 ab	43 ab	39 abc	0.62 bc	0.65 abc	0.070 c	0.245 b
Means			18	1.1	17	0.6	153	158	35	36	0.52	0.61	0.072	0.253

^z1 × cc = single dose of competitive cations 0.125 kg·m⁻³ each of: (NH₄)₂HPO₄, KCl, MgCl₂, and K₂SO₄; 2 ×, 4 × = double and quadruple rates. These mixes received 6 kg·m⁻³ of dolomitic limestone and 0.75 kg·m⁻³ of Perk.

^yTreatments 1-5 were amended with 0.25 kg·m⁻³ each of (NH₄)₂HPO₄ and K₂SO₄ as well as 0.75 kg·m⁻³ of Perk.

^x- Ca = no calcium spray; + Ca = sprayed twice weekly with 432 ppm Ca as CaCl₂.

^wMean separation in each category of data by Duncan's multiple range test, $P = 0.05$.

by CaCl₂ sprays as shown by data (Table 2) on leaf and bract marginal necrosis. The high rate of competitive cations that decrease uptake of Ca increased the incidence of bract and leaf necrosis over that of the low rate. Growth of poinsettias was severely limited by the two lowest levels of CaCO₃. Characteristic Ca deficiency developed in these treatments. CaCl₂ spray applied late in the production season essentially prevented leaf and bract necrosis except for the lowest CaCO₃ level (0 kg·m⁻³). Calcium contents of leaves and bract margins were reduced to apparent deficiency levels by complete substitution of MgCO₃ for CaCO₃, but CaCl₂ sprays raised Ca to marginally adequate levels of the element. Unsprayed bract margins

were uniformly low in Ca content.

The hypothesis that bract necrosis is an expression of a Ca deficiency disorder is supported by the results of the experiments reported. Further work is indicated to elucidate the role of many environmental factors that induce bract necrosis (and Ca deficiency). Additional research is desirable to find the most practical methods of preventing bract necrosis as well as to identify the physiological features of 'Gutbier V-14 Glory' that predispose to bract necrosis problems.

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