

Characterization of Nutrient Disorders of Pot Rose ‘Karina Parade’

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Abstract

Pot rose cultivar ‘Karina Parade’ plants were grown in silica sand culture to induce and photograph nutritional disorder symptoms. Plants were grown with a complete modified Hoagland’s all nitrate solution: (macronutrients in mM) 15 NO₃-N, 1.0 PO₄-P, 6.0 K, 5.0 Ca, 2.0 Mg and 2.0 SO₄-S, plus μM concentrations of micronutrients, 72 Fe, 18 Mn, 3 Cu, 3 Zn, 45 B and 0.1 Mo. The nutrient deficiency treatments were induced with a complete nutrient formula and a complete minus one of the nutrients. Reagent grade chemicals and deionized water of 18-mega ohms purity were used to formulate treatment solutions. Boron toxicity symptoms were also induced by increasing the element 10× higher than the complete nutrient formula. The plants were automatically irrigated every 2 hours and the solution drained from the bottom of the pot and recaptured for use. A complete replacement of nutrient solutions was done weekly. Plants were monitored daily to document and photograph sequential series of symptoms as they developed. Typical symptomology of nutrient disorders and critical tissue concentrations are presented.

INTRODUCTION

Rosa L. hybrids (*Rosaceae* family) are popular ornamental plants for cut flowers and garden use. Miniature flowering potted cultivars have been bred for 4- to 6-in. (10- to 15-cm) pot production (Dole and Wilkins, 2005). Pot roses are considered heavy feeders. A desirable leaf area to flower ratio can be produced by providing a balanced fertilization program. Periodic monitoring can assure the nutritional requirements are being met. Langhans (1987) documented and photographed the nutrient deficiency symptoms of roses for cut flowers. The symptomology of most nutrient disorders with critical tissue concentration for greenhouse pot plant production have not been described. When growers face nutritional problems with visual symptoms, knowing key symptoms of nutrient disorders would assist them in problem identification. Also, the critical leaf tissue concentration is helpful to determine when nutritional status of plants nears detrimental concentrations.

MATERIALS AND METHODS

Pot rose liners of *Rosa hybrida* L. ‘Karina Parade’ were transplanted two plants per 13.97 cm diameter (1.29 L) plastic pot containing acid washed silica-sand [Millersville #2 (0.8 to 1.2 mm diameter); Southern Products and Silica Co., Hoffman, NC] on October 4, 2007. The experiment was conducted in a glasshouse in Raleigh, NC at 35°N latitude. Plants were grown at 20°C day and 18°C night temperatures. An automated, recirculating, irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of 2 blocks with each block assigned to a single bench. There were 28 separate irrigation lines (each 1.82 m long) per block with each line containing six openings (12.7 cm diameter) to hold the pots. Three replications per block, each consisting of one pot with one plant, were assigned to each elemental treatment. Control plants were grown with a complete modified Hoagland’s all nitrate solution: (macronutrients in mM) 15 NO₃-N, 1.0 PO₄-P, 6.0 K, 5.0 Ca, 2.0 Mg and

2.0 SO₄-S (Hoagland and Arnon, 1950), plus μM concentrations of micronutrients, 72 Fe, 18 Mn, 3 Cu, 3 Zn, 45.0 B and 0.1 Mo. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrients solution excluding one of the nutrients. The B toxicity treatment was conducted by increasing B concentration (450 μM) in the Hoagland's solution. Reagent grade chemicals and deionized water of 18-mega ohms purity were used to formulate treatment solutions (Pitchay, 2002). The plants were automatically irrigated every 2 h using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, OK). The solution drained out from the bottom of the pot and was recaptured for reuse. Nutrient solutions were replaced weekly. Plants were monitored daily to document and photograph sequential series of symptoms on youngest, young, recently mature, and mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, plant shoot dry weight was recorded and the fully expanded leaves were sampled to evaluate the critical tissue concentration for each element. The harvested leaves were washed in a solution of 0.5 N HCl for 1 min, and rinsed with deionized water before drying at 70°C for tissue analysis. Dried tissue was ground in a Foss Tecator Cyclotec™ 1093 sample mill (Analytical Instruments, LLC, Golden Valley, MN) to pass a ≤ 0.5 mm sieve. Tissue analysis for N was performed with a C-H-N analyzer (Model 2400 series II, Perkin-Elmer, Norwalk, CT) by weighing 3.5 mg of dried tissue into tin cups and placed into the analyzer. Other nutrient concentrations were determined with inductively coupled plasma optical emission spectroscopy (ICP-OES; Model IRIS Intrepid II, Thermo Corp., Waltham, Mass.). All the data were subjected to ANOVA using PROC ANOVA SAS program (SAS Inst., Cary, N.C.). Where the F test indicated evidence of significant difference among the means, LSD ($P \leq 0.05$) was used to establish differences between means.

RESULTS AND DISCUSSION

Tissues sampling was conducted on five different dates as initial symptom occurred for each element.

Nitrogen

Initial symptom of nitrogen (N) deficient plants was a yellowish-green chlorosis on the bottom leaves and the entire plant was lighter green than control. As N symptoms advanced, chlorotic leaves developed reddish interveinal patches that were irregularly shaped, and a purplish pigmentation developed at the tips of the outermost leaflets. Nitrogen deficient plant size was smaller than that of control plants. Plant dry weight of nitrogen deficient plants was significantly smaller (Fig. 1A). Tissue nitrogen concentrations were 1.59 and 3.69% for the nitrogen deficient and control plants, respectively. The N concentration of deficient potted rose plants was below 2.14%, the upper limit concentration of deficient plants for cut roses (Johnsson, 1978) and slightly higher than the deficient range of 1.0 to 1.5% for cut roses (Oerti, 1966) (Table 1). The control plants were within the recommended range of 3.0 to 5.0% (Table 1).

Phosphorus

Phosphorus (P) deficiency resulted in significantly smaller plants than the controls (Fig. 1B). Fewer leaves and auxiliary shoot growth were observed. Phosphorus deficiency initially appeared as darker green leaf coloration, except the older leaves had a uniform lighter green or yellowish coloration as the initial symptom (Fig. 2). Tissue P concentration of deficient plants of 0.10% was below the recommended critical level of 0.2% (Table 1), versus 0.34% for the control plants. Our values were lower than the upper limit 0.143% of deficiency reported by Johansson (1978) for cut roses grown in sand culture.

Potassium

Potassium (K) deficient plants initially developed lighter green interveinal

chlorosis on the lower leaves (Fig. 3). Potassium deficiency resulted in significantly smaller plant dry weight (Fig. 1B). Stunted growth has been reported as a result of potassium deficiency (Horst and Cloyd, 2007). While tissue K concentration of the control plants was 2.88%, K deficient plants contained 1.36% K in the leaf tissue. The K concentration of deficient potted rose plants was higher than the reported deficient range of 0.3 to 1.0% (Oerti, 1966) and <1.01% (Johansson, 1978), but lower than <1.8% (Boodley and White, 1969) for cut roses (Table 1).

Calcium

Plant dry weight was not significantly smaller when symptoms first appeared by withholding calcium (Ca). The initial symptoms of calcium deficiency were interveinal chlorosis on young leaves, collapse of flower stalks, and droplets of exuded clear liquid (Fig. 4). When the initial symptoms were observed, the tissue Ca concentration of 0.20% was within the deficient range 0.1 to 0.4% Ca for cut roses (Table 1). The control plants contained 1.25% Ca.

Magnesium

Magnesium (Mg) deficiency symptoms initially developed on older leaves as yellowish chlorosis. Then the chlorotic leaves developed white patches along the edge of the leaf (Fig. 5), and blotch patches developed interveinal necrosis. Tissue magnesium concentrations were 0.10 and 0.19% for the deficient and control plants, respectively. Both values were below the previously reported recommended range of $\geq 0.2\%$ Mg for cut roses (Oerti, 1966; Johansson, 1978).

Sulfur

Initially, sulfur (S) deficient plants developed uniform chlorosis on the recently matured leaves (Fig. 6). In nutrient solution culture systems, slight interveinal chlorosis on young leaves was reported by Horst and Cloyd (2007), however, the interveinal chlorosis was not observed in this study. The tissue S concentration of deficient plants was 0.22 and 0.26% for control plants. The 0.22% S concentration found to occur when symptoms first appeared is above the range of 0.16 to 0.21% cited by Sadasivaiah and Holley (1973). It is unclear as to why our critical S range when symptomology first appeared is higher than the only previous range reported. Common greenhouse practices for cut roses included the use of sulfur pots for the control of foliar diseases (Langhans, 1987), which would make the incidence of S deficiency unlikely.

Boron

Boron (B) deficient plants initially developed interveinal chlorosis on the young leaves (Fig. 7), and produced 23% less plant dry weight compared to control plants (Fig. 1D). New developing leaves were smaller than on control plants and exhibited distorted growth in the midleaf blade area (Fig. 7). Leaf tissue B concentration for deficient and control plants was 2.55 and 71.74 mg kg⁻¹, respectively. The B concentration of deficient plants was significantly lower than the recommended ranges, 20 to 60 mg kg⁻¹ (Table 1).

The matured leaves of B toxic plants developed brown necrosis along the leaf margin (Fig. 8). The dry weight of toxic plants was only 46% of control plant dry weight (Fig. 1D). Leaf tissue B concentration for toxic and control plants were 473.99 and 71.74 mg kg⁻¹, respectively. Both B concentrations were higher than the recommended optimal range (Table 1).

Copper

The initial symptom of copper (Cu) deficiency was interveinal chlorosis on the recently matured leaves. When initial copper deficient symptoms were observed, tissue Cu concentrations were not detectable in the tissue analysis (lower detection limit is 0.56 mg·kg⁻¹) and 1.07 mg·kg⁻¹ for control plants. Both concentrations are lower than the recommended critical Cu range 5 to 15 mg kg⁻¹ (Table 1). On the harvest day, the dry

weight of a copper deficient plant was 1.96 and 3.63 g for a control plant (Fig. 1C).

Zinc

The upper young leaves developed a completely uniform yellow-green coloration between the veins as the initial symptom of zinc (Zn) deficiency. The dry weight of Zn deficient plants was 60% of control plant's dry weight (Fig. 1C). Tissue Zn concentration of deficient and control plant were 5.89 and 8.58 mg kg⁻¹, respectively. The Zn concentration for both the control and deficient potted rose plants were lower than the recommended optimal range of 15 to 50 mg kg⁻¹ for cut roses (Table 1).

Iron

The initial symptom of iron (Fe) deficiency developed as chlorosis on young leaves. About 10 days after the first symptoms, an interveinal chlorosis developed over almost the entire plant. As symptoms advanced, the entire leaves had severe interveinal chlorosis (Fig. 9). The dry weight of deficient plants was 17% smaller when compared to control (Fig. 1A). When initial symptoms developed, tissue Fe concentration was 65.18 mg kg⁻¹, as compared to 110.26 mg kg⁻¹ for the control plants. The Fe concentration of deficient potted rose plants was within recommended optimal range of 50 to 150 mg kg⁻¹ for cut roses (Table 1).

Manganese

Manganese (Mn) deficiency symptoms initially developed on recently matured leaves as interveinal chlorosis and progressed to whitish-tan spots (Fig. 10). Plant dry weight was not statistically smaller (Fig. 1E). Tissue manganese concentration of deficient plants was 7.13 and 106.32 mg kg⁻¹ for the control plants. The Mn concentration for potted rose plants was within the deficient range of below 30 mg kg⁻¹ for cut roses (Boodley and White, 1969) (Table 1).

CONCLUSIONS

The descriptions of visual symptoms derived from each nutrient deficiency were presented for potted rose plants. When initial symptoms occurred, tissue nutrient concentration was analyzed as critical tissue nutrient levels. The standards will provide guidelines for commercial growers when trying to diagnose nutritional problems in pot roses.

ACKNOWLEDGEMENTS

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Tables

Table 1. Previously reported leaf tissue concentrations for cut roses.

Nutrient	Oerti (1966)		Boodley and White (1969)		Carlson (1966)	Sadasivaiah and Holley (1973)	Johansson (1978)
	Deficient	Healthy	Below standard range	Standard range	Optimal	Normal range	Upper limit of deficiency
Nitrogen (% N)	1.0-1.5	4.0-6.0	Below 3.0	3.0-5.0	3.2-4.0	3.00-3.50	2.14
Phosphorus (% P)	0.01-0.03	≥0.2	Below 0.2	0.2-0.3	0.2-0.3	0.28-0.32	0.14
Potassium (% K)	0.3-1.0	≥1.0	Below 1.8	1.8-3.0	1.5-1.8	2.00-2.50	1.01
Calcium (% Ca)	0.1-0.4	1.0-3.5	Below 0.25	0.25-0.35	0.28-0.34	0.28-0.32	0.39
Magnesium (% Mg)	0.1-0.2	≥0.2	-	-	-	-	0.20
Sulfur (% S) ¹	-	-	-	-	-	0.16-0.21	
Boron (mg kg ⁻¹ B)	0.03-0.05	≥0.1	Below 30	30-60	20-40	40-60	
Copper (mg kg ⁻¹ Cu)	10-20	≥20	Below 5	5-15	10-14	7-15	
Iron (mg kg ⁻¹ Fe)	20-60	≥60	Below 50	50-150	80-100	80-120	
Manganese (mg kg ⁻¹ Mn)	-	-	Below 30	30-250	300-900	70-120	
Zinc (mg kg ⁻¹ Zn)	-	-	Below 15	15-50	40	20-40	

¹ The unit reported is listed as mg·kg⁻¹ S instead of as a percent S.

Figures

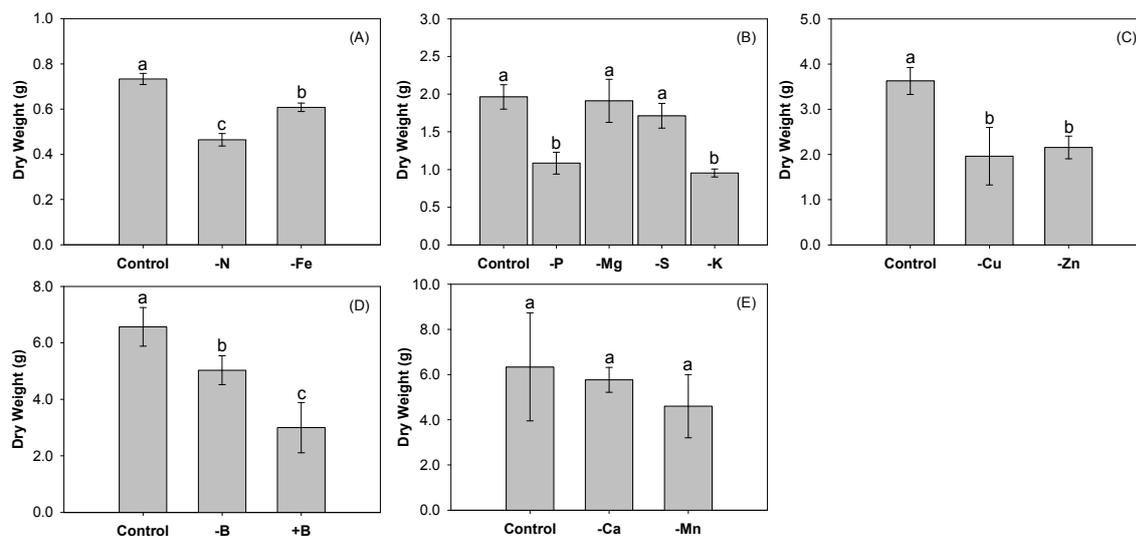


Fig. 1. Mean shoot dry weight (g) for plants grown with a complete nutrient solution (Control), with deficient levels of each nutrient (-N, -P, -K, -Ca, -Mg, -S, -Cu, -Fe, -B, -Mn, and -Zn), or with a toxic level of boron (+B). Five different harvest dates; plants harvested on 19 October, 2007 (A), on 2 November, 2007 (B), on 4 December, 2007 (C), on 14 December, 2007 (D), and on 27 December, 2007 (E). (Letter on the each bar in each harvest date represents a mean separation using pairwise t-test at $\alpha=0.05$).



Fig. 2. Darker green coloration of leaves except for uniform yellow-green chlorotic old leaves by phosphorous deficiency.



Fig. 3. Interveinal chlorosis on the lower leaves occurred with potassium deficiency.



Fig. 4. The collapse of flower stalks and the formation of exuded droplets occurred with calcium deficiency.



Fig. 5. White tan patches progressed from interveinal chlorosis progresses by magnesium deficiency.



Fig. 6. Chlorotic upper leaves were observed with sulfur deficiency.



Fig. 7. Interveinal chlorosis on the young leaves occurred with boron deficiency.



Fig. 8. Tip brown necrosis of the recently matured leaves occurred with boron toxicity.



Fig. 9. Interveinal chlorosis induced by iron deficiency.



Fig. 10. The progression of manganese deficiency initially as an interveinal chlorosis which progressed to whitish-tan spots.

