Characterization of Nutrient Disorders of *Pericallis* × *hybrida* 'Jester Pure Blue'

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Abstract

***Pericallis* × *hybrida* 'Jester Pure Blue' 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 NO_3-N, 1.0 PO_4-P, 6.0 K, 5.0 Ca, 2.0 Mg and 2.0 SO_4-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 minus one of the nutrients. Reagent grade chemicals and deionized water of 18 mega ohms purity were used to formulate treatment solutions. Boron toxicity was also induced by increasing the element 10× higher than the complete nutrient formula. The plants were automatically irrigated. The solution drained from the bottom of the pot and was captured for reuse. 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

Commonly grown for use as a potted plant, cineraria (*Asteraceae* family) is a floriculture crop that is reported to have a light to moderate fertilization requirement. Aimone (1986) recommended a fertilizer regime of 100 $mg \cdot L^{-1}$ N and K applied as a constant liquid fertilization or 200 $mg \cdot L^{-1}$ 20-20-20 used with every other watering. Dole and Wilkins (2005) recommended fertilization rates of 100 $mg \cdot L^{-1}$ as a constant liquid fertilization or a slightly lower weekly rate of 150 $mg \cdot L^{-1}$ N.

Few nutritional problems are reported (Dole and Wilkins, 2005). Boron deficiency leads to stunted growth and disfigured, mottled leaves. High pH induces iron deficiency which appears on the upper leaves as interveinal chlorosis. Over fertilization can result in excess growth that makes the plants dry out faster than smaller plants (Aimone, 1986). High levels of ammonium nitrogen can lead to rolled leaves and silvery-green foliage (Nau, 1984).

The lack of information provides a disadvantage to growers that need to make an on-site diagnosis when nutritional disorders occur. The objectives of this study were to provide visual and tissue diagnostic values for cineraria.

MATERIALS AND METHODS

Single 'Jester Purple Blue' cineraria plugs (3.4×3.4×5.1 cm cell size) were transplanted into 13.74 cm diameter (1.29 L) plastic pots containing acid washed silica-sand [Millersville #2 (0.8 to 1.2 mm diameter) from Southern Products and Silica Co., Hoffman, NC] on 24 October 2008. This experiment was conducted in a glass greenhouse in Raleigh, NC at 35°N latitude. Plants were grown at 23°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 24 separate irrigation lines (each 1.82 m long). Each line contained 8 openings (12.7 cm diameter) that held the 8 pots for the elemental treatment. Control plants were grown with

a complete modified Hoagland's all nitrate solution: (macronutrients in mM) 15 NO_3-N, 1.0 PO_4-P, 6.0 K, 5.0 Ca, 2.0 Mg and 2.0 SO_4-S (Hoagland and Arnon, 1950), plus μM concentrations of micronutrients, 72 Fe, 18 Mn, 3 Cu, 3 Zn, 45 B and 0.1 Mo. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. The B toxicity treatment was conducted by increasing the 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 as needed from 6:00 hours to 18:00 hours using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma). 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, three symptomatic plants were selected for sampling. The fully expanded leaves were sampled to evaluate the critical tissue concentration for each element. Harvested leaves were washed in a solution of 0.5 N HCl for 1 min and rinsed with deionized water. The remaining shoot tissue was harvested separately. Both sets of tissue were dried at 70°C for at least one week, and the weights were recorded. After drying fully expanded leaf 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.). During the first harvest of nitrogen and iron deficient plants, they were too small to obtain the necessary tissue from one plant, therefore two plants were combined for a single sample. The experiment was terminated when the plants were in full bloom on 19 December, and non-symptomatic plants of the copper, manganese, molybdenum and zinc were sampled for dry weight and nutrient levels. 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

Plants were sampled on six separate harvest dates.

Nitrogen

On nitrogen (N) deficient plants the initial symptoms developed on the lower mature leaves as a yellow-green coloration (Fig. 1, left). At initial sampling, the plants were 35.9% smaller than the control. Tissue concentration at this stage was 6.96% for control plants while the N deficient tissue concentration was 1.59%. Over time, the leaves became completely yellow and some lower leaves became completely necrotic (Fig. 1, right). At the completion of the experiment, the N deficient plants were visually smaller than the control plants.

Phosphorus

Initially phosphorus (P) deficient plants exhibited an overall dull black-green coloration (Fig. 2, left). At initial sampling, P concentration was statistically different between the treatments (1.02 and 0.14%, respectively, for the control and minus P plants), but plant mass was not. As symptoms progressed, the lower leaves initially developed partial yellowing which was followed by necrosis of the entire leaf (Fig. 2, right). When the experiment was terminated, the P deficient plants were visually smaller than the controls.

Potassium

Initial symptoms for potassium (K) deficiency were lower leaf speckling (Fig. 1, left) and marginal interveinal chlorosis on the most recently mature leaves. At harvest plants were 22.0% larger than the control. The control tissue concentration contained 8.68% K while K deficient plants had a value of 1.10% K. Necrosis then occurred and was expressed on individual plants either along the leaf margin or on the inside of the leaf (Fig. 3, right). Necrotic symptoms were more severe on lower leaves than upper. Inflorescences either failed to develop or aborted.

Calcium

Calcium (Ca) deficient plants initially exhibited leaf speckling near the petiole on middle and upper leaves (Fig. 4, left). Upper leaves also exhibited slight interveinal chlorosis. At sampling, the roots were inspected and Ca deficient plants had roots that were a burgundy-reddish color as compared to an off-white color of the controls. Plant masses were not significantly different. Tissue Ca values for control and deficient plants were 1.87 and 0.37%, respectively. Symptoms progressed with lower leaf yellowing, speckling spreading to cover the entire leaf, and leaf curl (Fig. 4, right). This speckling was followed by necrosis. At the completion of the experiment, Ca deficient plants were visually smaller than the control.

Magnesium

Tan spots on older and younger leaf margins were the initial symptoms of magnesium (Mg) deficient plants (Fig. 5, left). A significant difference between the two dry weights of the two samples did not occur. The control tissue Mg concentration at this stage was 0.64% while the Mg deficient plants had a value of 0.15%. Symptoms progressed with spotting developing inward from the margin. New spots were larger and started as dark green regions on lower leaves. After a few days the color changed to a tan brown. Lower leaves exhibited more spotting than the upper leaves. Upper leaves had few spots isolated in the leaf margin. The plant then developed a yellowish coloration, and some lower leaves became completely necrotic (Fig. 5, right). The Mg deficient plants were visually smaller than the controls at the end of the experiment.

Sulfur

Sulfur (S) deficient plants exhibited an overall greenish-yellow coloration (Fig. 6, left). At the time of sampling there were no size differences between the deficient S plants and the controls. Tissue S concentrations for control and S deficient tissue were 0.52 and 0.18% respectively. This color became a more intense yellow over time, and necrosis developed on the leaf margins (Fig. 6, right). At the end of the experiment, S deficient plants were visually smaller than the control plants.

Boron

Plants exhibiting boron (B) deficiency were similar in dry mass with the control. Recently matured leaves had whitish-yellow speckling on the edge of the leaf (Fig. 7, left). It was visible at harvest that the root tips were brown and had limited growth compared to control. No size differences existed between B deficient tissue and the control. Control tissue B concentration was 52.1 $mg \cdot kg^{-1}$ while the B deficient tissue concentration was 12.1 $mg \cdot kg^{-1}$. After two weeks, speckling had spread over the upper leaves of the entire plant. Youngest leaves were thick and distorted and had a rubbery feel to the touch. Lower leaves were asymptomatic. Inflorescences either aborted (Fig. 7, right), or they formed and never produced flowers. Upper leaves exhibited marginal necrosis and leaf wilt. New leaves did not fully develop. Our observations of distorted growth are consistent with symptoms reported by Dole and Wilkins (2005). B deficient plants were smaller than the controls when the experiment ended.

Symptoms of B toxicity began with necrosis on lower leaves where veins reached the edge of the leaf (Fig. 8, left). At initial sampling, the plants were 27.1% larger than

the control. Tissue values for B toxicity plants and control were 396.8 and 76.4 $mg \cdot kg^{-1}$ B, respectively. After four weeks, necrosis covered the entire margin of the leaf. Upper leaves also began exhibiting symptoms. In another four weeks yellowing had developed between the necrotic area and the healthy tissue (Fig. 8, right).

Iron

Iron (Fe) deficient plants exhibited interveinal chlorosis of the recently matured leaves (Fig. 9, left). Tissue dry mass and concentrations were not significantly different. The tissue Fe concentration for the control plants was 85.1 $mg \cdot kg^{-1}$ while the concentration for Fe deficient plants was 64.3 $mg \cdot kg^{-1}$. Because of the small plant size, two plants were combined for each sample. The lack of significance can be attributed to the high standard deviation that occurred with one pair of symptomatic plants. The mean tissue concentration of the remaining two replications was 52.54 $mg \cdot kg^{-1}$ Fe. Chlorosis then spread over the entire plant, even into the inflorescence leaves (Fig. 9, right). These symptoms match the description provided by Dole and Wilkins (2005) for Fe deficient plants induced by a high substrate pH.

Asymptomatic

At the end of the experiment after 8 weeks of growth when the plants had reached full bloom, plants grown under copper (Cu), manganese (Mn), molybdenum (Mo), and zinc (Zn) deficient conditions exhibited no visual symptoms. At termination, these plants were sampled and analyzed for dry mass and tissue concentration to determine if non-visual differences were evident. For all four elements, plant dry mass and tissue concentrations were significantly different for the control and induced plants.

Plants grown under Cu deficient conditions were 26.7% smaller in mass than the control. The control tissue Cu concentration was 16.9 $mg \cdot kg^{-1}$ while Cu deficient was 6.3 $mg \cdot kg^{-1}$.

Plants grown under Mn deficient conditions had a Mn tissue concentration of 14.0 $mg \cdot kg^{-1}$ as compared to 116.4 $mg \cdot kg^{-1}$ for the controls. Plants grown without Mn were 27.3% smaller than the control.

Plants grown under Mo deficient conditions were 38.7% smaller than the control. The control tissue Mo concentration was 0.2 $mg \cdot kg^{-1}$ while Mo deficient tissue concentration was not detected.

Plants grown under Zn deficient conditions had a Zn tissue concentration of 15.7 $mg \cdot kg^{-1}$ as compared to 21.6 $mg \cdot kg^{-1}$ for the controls. Plants grown without Zn were 49.6% smaller than the control.

Even though the plants grown without Cu, Mn, Mo, and Zn were asymptomatic, the dry mass and tissue concentration values will be useful to greenhouse growers when trying to determine if these elements are limited.

CONCLUSIONS

Induced nutrient deficiencies produced the symptoms described for cineraria. Tissue samples taken when initial symptoms occurred provide critical tissue nutrient levels for the crop. These two sets of data will be helpful for commercial growers needing to diagnose cineraria nutritional problems.

ACKNOWLEDGEMENTS

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Tables

Table 1. *Pericallis* x *hybrida* 'Jester Pure Blue' plant dry weight as affected by deficient or toxic nutrient treatments.

Treatment	-N	-P	-K	-Ca	-Mg	-S	-B	+B	-Cu	-Fe	-Mn	-Mo	-Zn
	Dry weight (g)												
Element	N	P	K	Ca	Mg	S	B	B	Cu	Fe	Mn	Mo	Zn
Complete control	0.74A[1]	0.70a	0.70b	3.77a	2.54a	0.70a	1.50a	0.70b	8.90a	0.74a	8.90A	8.90A	8.90A
Treatment	0.48B	0.51a	0.89a	3.50a	2.84a	0.66a	1.89a	0.89a	6.52a	0.77a	6.47B	5.46A	4.49B

[1] Significant differences between sample means based on *F* test are indicated by lower case letters if $P \leq 0.05$ or upper case letters if $P \leq 0.01$.

Table 2. *Pericallis* × *hybrida* 'Jester Pure Blue' tissue nutrient concentration as affected by deficient or toxic nutrient treatments.

Treatment	-N	-P	-K	-Ca	-Mg	-S	-B	+B	-Cu	-Fe	-Mn	-Mo	-Zn
	Tissue nutrient concentration (%)						Tissue nutrient concentration (mg kg^{-1})						
Element	N	P	K	Ca	Mg	S	B	B	Cu	Fe	Mn	Mo	Zn
Complete control	6.96A[1]	1.02A	8.68A	1.87A	0.64A	0.52A	52.1A	76.4B	16.9A	85.1a	116.4A	0.2a	21.6a
Treatment	1.59B	0.14B	1.10B	0.37B	0.15B	0.18B	12.1B	396.8A	6.3B	64.3a	14.0B	ND[2]	15.7b

[1] Significant differences between sample means based on *F* test are indicated by lower case letters if $P \leq 0.05$ or upper case letters if $P \leq 0.01$.
[2] Tissue concentration was below the detectable limit of 1.0 $mg \cdot kg^{-1}$.

Figures

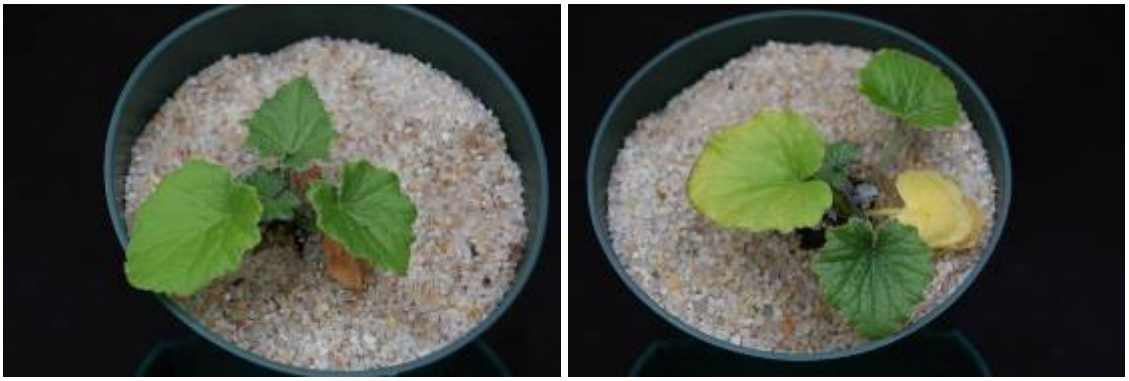

Fig. 1. Nitrogen deficiency began as a lower leaf yellowing (left) and advanced to leaf necrosis (right).

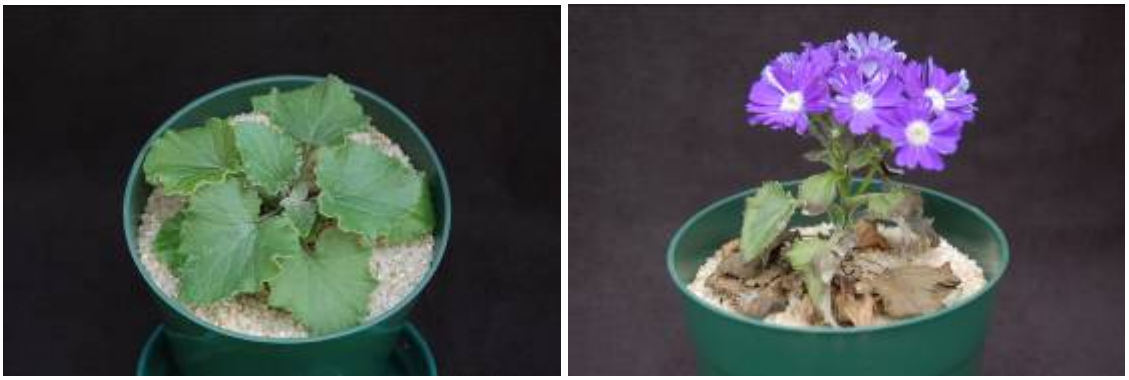

Fig. 2. Phosphorus deficiency appeared as dull black-green coloration of the leaves (left) and advanced to leaf necrosis (right).

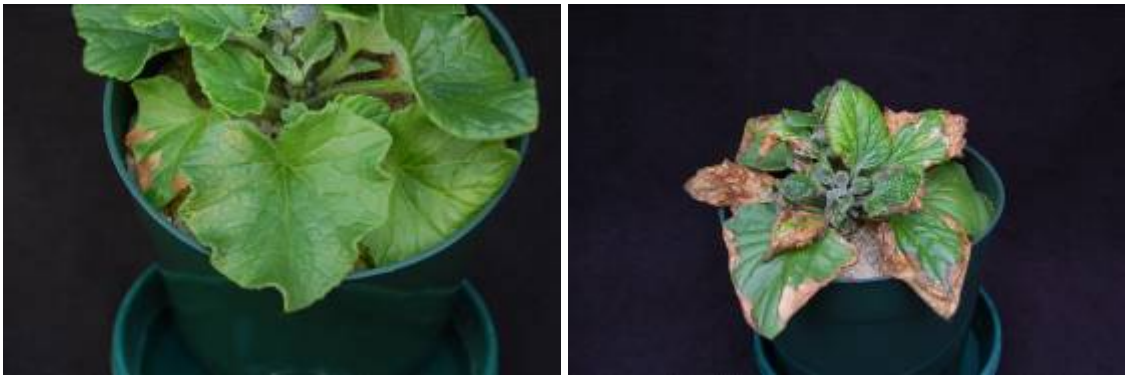

Fig. 3. Potassium deficiency began as lower leaf speckling (left) and was followed by necrosis (right).

Fig. 4. Calcium deficiency (left image illustrates control plant on the left side and Ca deficient plant on the right side) appeared as speckling on lower leaves near the petiole and interveinal chlorosis on the upper leaves (left) and progressed to lower leaf yellowing and leaf curl (right).

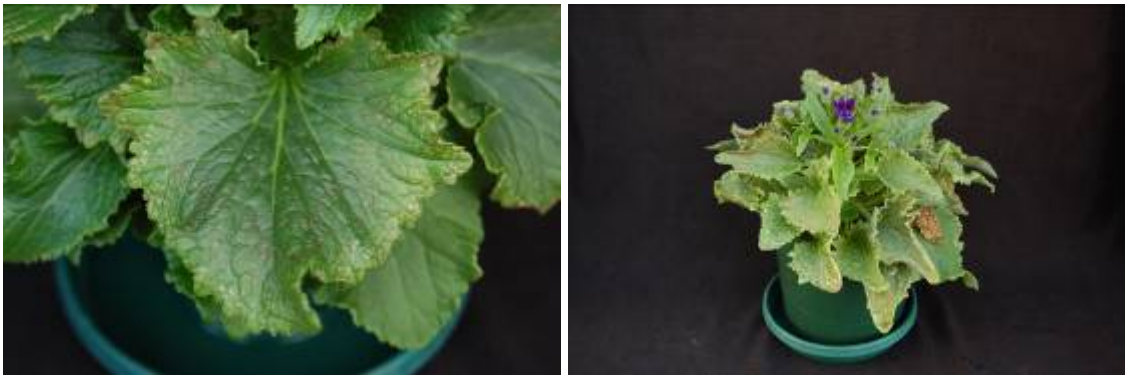

Fig. 5. Magnesium deficiency began as spots on leaf margins (left) and advanced to larger spots and the plant developing a yellow coloration (right).

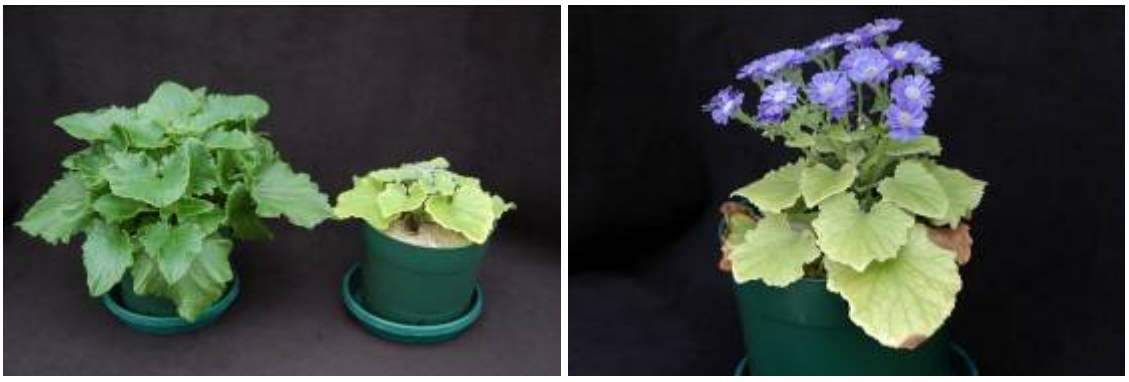

Fig. 6. Sulfur deficiency (left image illustrates control plant on the left side and S deficient plant on the right side) began with plants exhibiting a greenish-yellow color (left) and progressed to a more intense yellow coloration in the leaves and necrosis (right).

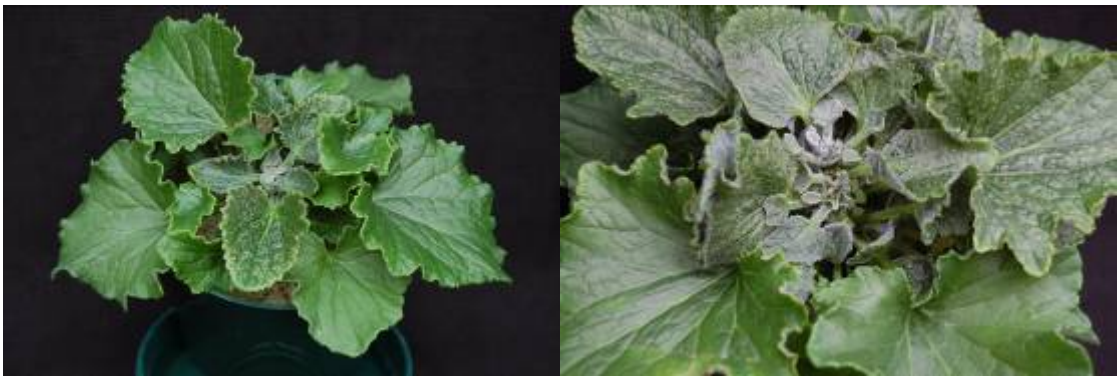

Fig. 7. Boron deficiency appeared as whitish yellow speckling on leaf edge of recently matured leaves (left) and advanced to speckling covering the entire leaf and inflorescence abortion (right).

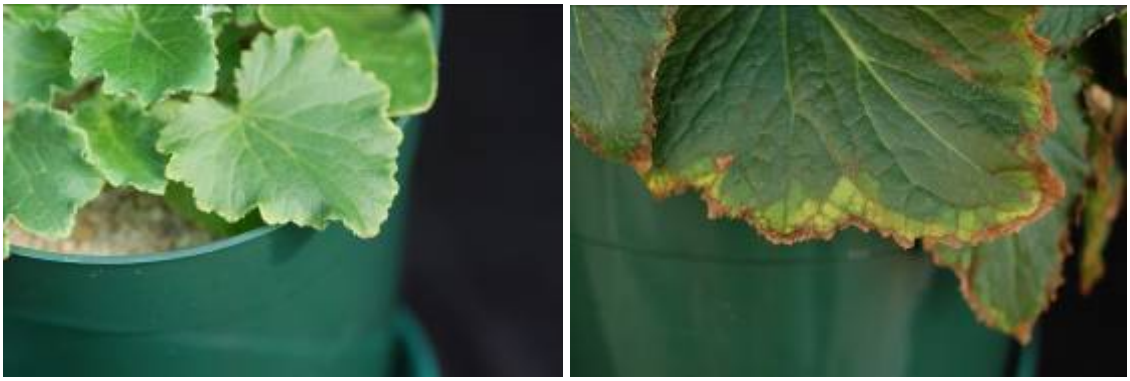

Fig. 8. Boron toxicity began with necrosis on lower leaves along the margin where veins reached the edge of the leaf (left) and progressed to the entire leaf edge becoming necrotic and yellow coloration between the necrotic and healthy tissue (right).

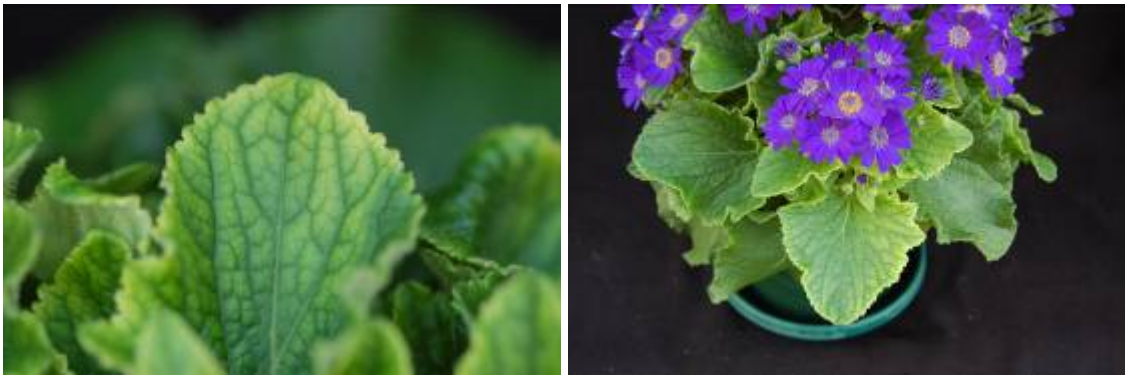

Fig. 9. Iron deficiency appeared as interveinal chlorosis of recently matured leaves (left) and advanced to chlorosis spreading over the entire plant (right).