ABSTRACT

BARNES, JARED GENE. Characterization of Nutrient Disorders of Floriculture Species. (Under the direction of Brian Earl Whipker.)

Managing fertility is an important aspect of floriculture crop production, and problems can occur during production. Having disorder symptom descriptions and critical tissue values available provides growers the means to quickly recognize and correct problems. However, such resources are not available for all crops, and growers who cannot confirm that a nutrient disorder is occurring are at a disadvantage.

Twelve species that lack complete disorder descriptions and critical tissue concentrations are abutilon (*Abutilon ×hybridum*), coreopsis (*Coreopsis grandiflora*), cosmos (*Cosmos bipinnatus*), dahlia (*Dahlia ×hybrida*), dianthus (*Dianthus* sp.), fuchsia (*Fuchsia ×hybrida*), gazania (*Gazania rigens*), gomphrena (*Gomphrena globosa*), cineraria (*Pericallis ×hybrida*), primula (*Primula acaulis*), salvia (*Salvia farinacea*), and dusty miller (*Senecio cineraria*). These species were grown in silica sand culture to induce and photograph symptoms of nutritional disorders. 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. Boron toxicity was also induced by increasing the element 10× higher than the complete nutrient formula. Reagent grade chemicals and deionized water of 18-mega ohms purity were used to formulate treatment solutions. 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. The concluding chapter highlights the similarity and atypical symptoms among species.

Characterization of Nutrient Disorders of Floriculture Species

by
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A thesis submitted to the Graduate Faculty of
North Carolina State University
in partial fulfillment of the
requirements for the Degree of
Master of Science

Horticultural Science

Raleigh, North Carolina

2010

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DEDICATION

To “Granddad,” thanks for letting me follow you with a rake.

BIOGRAPHY

Jared Barnes was born in Martin, Tennessee on 11 August 1985 to Cleo and Becky Barnes. Barnes graduated salutatorian of his class from Obion County Central High School in May 2004 and graduated summa cum laude from the University of Tennessee at Martin in May 2008. Barnes interned at The Scott Arboretum of Swarthmore College in the summer of 2008. He currently resides in Raleigh, North Carolina and attends North Carolina State University (NCSU). He will begin work on his doctorate in August 2010 at NCSU.

ACKNOWLEDGEMENTS

I’ve often compared the important people in my life to rungs on a ladder. Each person has been a step on the way to where I am now and a true blessing in my life. Omission of one of them and I believe my life could have taken a drastically different route.

‘Granddad,’ thank you for being the first step into the world of horticulture. I appreciate you letting me play with you in your garden. I enjoyed following you, raking our footsteps out as you pushed the Troy-Bilt® tiller. You’ll never know how much that time spent with you means to me today.

Granny and Bill, thank you for letting me keep gardening on your land after ‘Granddad’ passed away. And, thanks for tolerating my first compost pile in your front yard. Aunt Sue, thank you for all your kindness and support.

To the members of the Northwest Tennessee Master Gardeners Club, thank you for all that you taught me. As a young teenager I learned much about gardening and horticulture with you. Preston and Mary Prather, thank you for your advice and encouragement about graduate school. Ron Shaffer, thanks for being a great friend. I’ve enjoyed our many conversations over the years.

Carol Reese, thanks for laying much of the framework in my head for how landscape design is supposed to be. And, thank you for making me aware of Jimmy Williams. Jimmy, I appreciate you opening your garden to me and all the knowledge (and plants) that you've shared. The scholarship you nominated me for has changed my life, and I consider you my gateway into the world of horticulture.

Perennial Plant Association, thank you so much for the 2007 Academic Scholarship. It changed my life and propelled me to where I am today. Without it I would not have met all the wonderful people at the symposium that gave me such excellent advice and recommendations for graduate school. Many are still friends to this day. Allen Bush, Brent and Becky Heath, Dan Heims, Jason Reeves, Laura Deeter, and Troy Marden, I appreciate all the support and encouragement you have given me. You are great friends. Stephanie Cohen, I never will forget all the advice you've given me. I still remember the day that you emailed me about the internship at The Scott Arboretum in Swarthmore, Pennsylvania. Thanks for having such a 'pay-it-forward' attitude. I hope I can help others one day like you've helped me.

To the staff of The Scott Arboretum and the grounds crew of Swarthmore College, thank you for one of the best summers of my life. Andrew Bunting, Rhoda Maurer, and Jacqui West, thanks for making a southern feel welcome in a northern town. You've all been great friends. I learned so much visually and intellectually about horticulture from all of you and from the opportunities you presented me with. It's not everyday that you have some of the greatest gardens and gardeners in your backyard.

Dr. Tom Blanchard, Dr. Nancy Buschhaus (Buschhaus), Emily Haynes (Aunt Emily), Dr. David Pitts, Michael Poore, Dr. Darrell Ray, Dr. Gopal Sharma, and Dr. Dawn Wilkins (Dawn), thanks for all that you taught me, and thanks for preparing me for graduate school. I had a blast in your classes! Many parts of this thesis are a reflection of the writing methods you taught me; I'll add I don't think this document has a single semicolon in it.

All of the aforementioned people brought me to where I am today, and I turned to many of them for advice on grad schools. While here numerous people in the Horticulture Department have provided so much advice and support. Dr. Brian Whipker, you are top on that list. Thank you for being such a great advisor. Your requests for research were always backed with the knowing that life happens, and sometimes things don't go as expected. Thank you for your patience. You have also provided many educational opportunities by allowing me to do research outside of plant nutrition and to attend and to present at conferences. You opened the world to me, literally, when you allowed me to go to Singapore and present research there. Finally, thank you for the humbling experience of washing sand. I now know how to clean up the beaches of the world, one five-gallon bucket at a time.

Dr. Paul Nelson and Dr. Jamie Gibson, thanks for being great committee members. I appreciate all of your input and edits on this thesis. Jonathan Frantz, thank you for reviewing papers before conferences and for analyzing hundreds of tissue samples. Ingram McCall, thank you so much for all your help in the greenhouse. You were a great and patient companion changing solutions, sampling tissue, and potting up plants. Dr. John Dole and Rachel McLaughlin, thank you for all your help in the graduate program. Anytime I had an issue or question I could count on one of you to answer it. So many other members of the faculty and staff have taught me so much and provided help and great conversations in the hallways and the greenhouses.

I’d also like to thank all of the graduate students for their help and advice. Lis Meyer and Todd Rounsaville, you are two of my best friends and I’m glad y’all will be around for my PhD. I’ve enjoyed our many conversations and lunches together. I hope that many more garden hopping events are in our future. I’d also like to thank the undergrads. Even though I am a grad student, thanks for considering me one of your own. Don’t let your enthusiasm die; you are the future of horticulture.

I’ve also made many friendships outside of the department here in North Carolina, friendships that have been essential to making sure that I don’t go crazy from so much school work and research. Anna, Becca, Becky, Ben, Beth, Brian, Bryan, Casey, Christine, Emily, Eric, Erich, Jenny, Joelle, Julie, Karli, Keegan, Laura, Phillip, Stewart, and Susanna, you are the finest people in the world and have made my stay in North Carolina immensely pleasurable. I cannot imagine a life without you. With you I have learned so much about life and God. You showed me how to think even more intelligently, how to kayak, and even how to dance. Thank you for accepting me and for letting me be myself around you.

Of course, all the steps would fall off if it weren’t for the sides. Dad and Mom, thanks for holding my life together. You have been there every step along the way with me, supporting me and helping me reach my full potential. I don’t know where this ladder is headed, but I know that without you, I would have never gotten off the ground. God has

truly blessed me with wonderful parents, and I thank Him for you and for all the blessings in my life. And, Elizabeth, thanks for being a great sister. I look forward to your texts each and every day.

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INTRODUCTION

Floriculture crop production is a major industry in the United States. In North Carolina the wholesale value of crops grown by producers with greater than $10,000 in sales was $217.2 million dollars for 2008 (USDA, 2009). Quality is important, and in order to grow a saleable crop, commercial growers must manage multiple cultural factors like plant nutrition. Plant nutrients are divided into two categories, essential and nonessential (Mills and Jones, 1996). Essential elements have three characteristics within the plant. Omission of one results in irregular growth, failure to reproduce, or death. An essential element is specific and cannot be replaced by another element. Last, growth and metabolism must be directly affected by an essential element (Arnon and Stout, 1939). Nonessential elements do not meet these three criteria and include nickel (Ni), silicon (Si), vanadium (V), sodium (Na), cobalt (Co), selenium (Se), and aluminum (Al). The sixteen essential elements are further broken down into two categories. Carbon (C), hydrogen (H), oxygen (O), nitrogen (N), phosphorus (P), potassium (K), calcium (Ca), magnesium (Mg), and sulfur (S) are the macronutrients, and the micronutrients are boron (B), chlorine (Cl), copper (Cu), iron (Fe), manganese (Mn), molybdenum (Mo), and zinc (Zn). Water and air are the source of C, H, and O. Plant uptake of the remaining thirteen elements occurs primarily from the substrate (Mills and Jones, 1996). Other sources for nutrients are foliar fertilizers (Dole and Wilkins, 2005) and fungicides (Brian Whipker, personal communication).

According to Dole and Wilkins (2005), fertilization is an “exact process,” and supplying nutrients in the correct amounts under an intense production schedule allows “little margin of error.” Nutritional problems arise when the absence or excess of the one of the essential nutrients occurs. While some similarities of symptoms do occur, growers need to have accurate crop specific diagnosis information available. These diagnostic tools include both symptomology images and critical tissue concentrations.

Twelve species that lack a complete description of symptomology and critical nutrient concentrations are abutilon (*Abutilon* x *hybridum*), coreopsis (*Coreopsis grandiflora*), cosmos (*Cosmos bipinnatus*), dahlia (*Dahlia* x *hybrida*), dianthus (*Dianthus* sp.), fuchsia (*Fuchsia* x *hybrida*), gazania (*Gazania rigens*), gomphrena (*Gomphrena globosa*), cineraria (*Pericallis* x *hybrida*), primula (*Primula acaulis*), salvia (*Salvia farinacea*), and dusty miller (*Senecio cineraria*). The objective of this study was to compile descriptions of initial and advanced symptomology and to determine critical nutrient concentrations. These twelve species were grown under N, P, K, Ca, Mg, S, B, Cu, Fe, Mn, Mo, and Zn deficiencies. Plants were also induced with B toxicity. The essential element Cl was not included in this study. Once the twelve crops were grown comparisons among the symptomology were made in an effort to stereotype nutritional disorders and highlight unique differences.

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Table 1.1 Common name, scientific name, and cultivar of the twelve species grown under deficient or toxicity nutrient treatments.

Common Name	**Scientific Name**	**Cultivar**
abutilon	*Abutilon ×hybridum*	Bella Yellow
coreopsis	*Coreopsis grandiflora*	Santa Fe Yellow
cosmos	*Cosmos bipinnatus*	Sonata ™ White
dahlia	*Dahlia ×hybrida*	Maxi Morelia
dianthus	*Dianthus*	Bouquet Purple
fuchsia	*Fuchsia ×hybrida*	Gartenmeister Bonstedt
gazania	*Gazania rigens*	Daybreak White
gomphrena	*Gomphrena globosa*	Las Vegas Purple
cineraria	*Pericallis ×hybrida*	Jester Pure Blue
primula	*Primula acaulis*	Danova Rose
salvia	*Salvia farinacea*	Evolution
dusty miller	*Senecio cineraria*	Silver Mist

Chapter 2

Characterization of Nutrient Disorders of *Abutilon ×hybridum* ‘Bella Yellow’

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Abutilon ×hybridum* 'Bella Yellow'

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Keywords: Flowering maple, Calcium, Micronutrients, Nitrogen, Phosphorus, Potassium, Sulfur

Abstract

***Abutilon ×hybridum* 'Bella Yellow' plants were grown in silica sand culture to induce and photograph symptoms of nutritional disorders. 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

Abutilon (*Abutilon ×hybridum*, Malvaceae family) is a tender perennial that is grown for its attractive flowers (Dole and Wilkins, 2005). Plants are typically raised from seed (Dole and Wilkins, 2005; PanAmerican Seed, 2010; Hamrick, 2003), but they can be produced vegetatively (Chapman and Lyons, 1987).

It is recommended that if started from seed, growers use a soilless mix that is disease-free, well-drained, has a pH of 5.5 to 6.3 (Hamrick, 2003), and an electrical conductivity (EC) of 0.75 mS/cm using a 1:2 extraction (PanAmerican Seed, 2010). When germination occurs, it is advised to use 15-0-15 at a concentration of 50 to 75 $mg \cdot L^{-1}$ N and increase the concentration to 100 to 150 $mg \cdot L^{-1}$ N once full expansion is observed on the cotyledons (Hamrick, 2003; PanAmerican Seed, 2010). If growth slows, 20-10-20 should be used every third irrigation. While growing the crop it is recommended to maintain the EC in the range of 1.0 mS/cm (PanAmerican, 2010) or 1.0 to 1.5 mS/cm (Hamrick, 2003).

After four or five weeks, plugs will be ready to pot. The substrate should be soilless, disease-free, well-drained (PanAmerican Seed, 2010), a pH of 5.5 to 6.5, and contain a medium initial nutrient charge (Hamrick, 2003; PanAmerican Seed, 2010). Fertilization recommendations vary from 150 $mg \cdot L^{-1}$ N nitrate based fertilizer at every other irrigation (Hamrick, 2003; PanAmerican, 2010) to using 300 $mg \cdot L^{-1}$ N from soluble

20-20-20 twice a week (Chapman and Lyons, 1987.) Using an ammoniacal N source will result in large leaves (Hamrick, 2003; PanAmerican, 2010).

Some nutritional problems are reported with abutilon. Too much nitrogen (N) reduces flowering (Hamrick, 2003; PanAmerican, 2010). Plants that are underfertilized exhibit pale green foliage coloration, and leaves appear droopy (PanAmerican, 2010).

While N is the only nutrient problem covered in the literature, others potentially exist, and the lack of information about those disorders can be a disadvantage to growers. Critical tissue values have also not been published. Therefore, this study was performed to investigate other nutritional problems of abutilon and to determine critical tissue values.

Materials and Methods

Four 72-cell (3.9 x 3.9 x 5.7 cm cell size) flats of 'Bella Yellow' abutilon seed were sown on 20 Aug. 2009. To mitigate germination conditions, seeds were germinated in a cooler at 21˚C. Cotyledons emerged on 23 Aug. 2009, and flats were moved into a glass greenhouse. Seedlings were fertigated as needed using 150 $mg \cdot L^{-1}$ N from 13-2-13 Cal-Mag (SQM North America, Atlanta, GA). Single plugs were transplanted into 12.7 cm diameter (0.76 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 Sept. 2009. Treatments started immediately upon transplanting. This experiment was conducted in a glass greenhouse in Raleigh, NC at 35°N latitude. Plants were grown at 21˚C day and 18˚C night temperature set points. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2 \cdot 4H_2O$, KH_2PO_4, $MgSO_4 \cdot 7H_2O$, FeDTPA, $MnCl_2 \cdot 4H_2O$, $ZnCl_2$, $CuCl_2 \cdot H_2O$, H_3BO_3, and $Na_2MoO_4 \cdot 2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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/or mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, three pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 $\leq$ 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 placing it 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.). The experiment was terminated on 19 Nov. 2009, and non-symptomatic plants grown in molybdenum deficient conditions 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 ten separate harvest dates. Significant differences did not occur for any tissue dry weights. Unless otherwise noted, values for tissue concentrations presented were significantly different.

Nitrogen

Symptoms of nitrogen (N) deficiency began with the plant exhibiting an overall greenish-yellow coloration. Some lower leaves were darker green but appeared to be developing the greenish-yellow coloration. Leaves also oriented parallel to the stem, giving them a 'droopy' appearance. These symptoms match those described by PanAmerican (2010) for plants that were underfertilized. N concentration in the control plant tissue was 4.98% and 2.14% N in the N deficient tissue. The greenish-yellow coloration then intensified to yellow. Blooms that formed were smaller and a lighter yellow color than the control. Isolated yellow leaves then began to exhibit necrosis near the tip.

Phosphorus

Phosphorus (P) deficiency began with the leaves appearing darker green than the control. The leaves also oriented parallel with the stem instead of being more perpendicular. The droopy symptom matches the description provided by PanAmerican (2010) for underfertilization, but the pale green color was not observed with P deficiency.

Only two samples had adequate tissue in order to determine control P concentration, and the average of those two was 0.39%. There was inadequate sample to determine P concentration of P deficient tissue. Flowers that formed were smaller than the control. In the advance stage lower leaf drop occurred.

Potassium

The initial symptom of potassium (K) deficiency was lower to middle leaf tip necrosis. K tissue concentrations in the control and K deficient plants were 4.60% and 0.86%, respectively. As the disorder progressed the necrotic area increased in size, and the leaf margin began to exhibit yellowing. The yellow area then became necrotic and turned a tannish-brown color.

Calcium

Calcium (Ca) deficiency manifested in the most recently developed leaves as a marginal yellow coloration around some parts of the leaf. Ca tissue concentration in the control plants was 2.94% and 0.68% in Ca deficient plants. As symptoms became more moderate, the middle and upper leaves developed a greenish-yellow coloration. In some leaves this intensified to interveinal chlorosis. Affected leaves began to exhibit brown coloration on the edge which became necrotic in the advance stage of Ca deficiency. Upper leaves near the apical bud and in the axillary shoots then dropped. Some flowers aborted; those that formed and opened were smaller than the blooms on control plants. On some plants the apical bud aborted. Plants tried to regrow new leaves near the top.

Magnesium

Symptoms of magnesium (Mg) deficiency started in the upper leaves as tertiary interveinal chlorosis. Control Mg tissue concentration was 0.73% Mg while the Mg deficient tissue concentration was 0.36%. The interveinal chlorosis coloration then became more intense in the middle and upper leaves, and in some leaves the only green left was near the primary and secondary veins. Necrotic spots that varied in size and location then began to appear on affected leaves. Smaller isolated spots appeared on lower leaves. Some leaf drop then occurred, and flowers began to abort.

Sulfur

Sulfur (S) deficiency symptoms were initially manifested in the upper leaves and axillary shoot leaves as the development of a greenish-yellow coloration. Control and S deficient tissue concentrations were 0.38% and 0.17% S, respectively. As the disorder progressed the entire plant developed a greenish-yellow coloration. This coloration intensified to yellow in the middle and upper leaves. Upper leaves then began exhibiting necrosis along the margin. These necrotic spots grew in size. Necrotic spots then began appearing on the lower leaf margins.

Boron

Boron (B) deficiency initially manifested in the upper leaves. The leaves were crinkled and had a shiny appearance. B concentration in the control plants was 35.5 $mg \cdot kg^{-1}$ and 6.3 $mg \cdot kg^{-1}$ in the B deficient plants. The disorder progressed with a yellow coloration developing in the margin of the upper leaves; the yellowing moved inward on the leaf. This marginal area then became limp and advanced to tannish-brown necrosis. Flowers along the stem aborted, and in some plants the apical bud died.

Interveinal chlorosis in the upper leaves was the initial symptom of B toxicity. Tissue concentrations were not significantly different because the controls only had two samples that had adequate sample and the B toxic treatment only had one sample. The average between the two B control samples was 36.6 $mg \cdot kg^{-1}$ which is approximately the same as the control value obtained from the control compared with the B deficient sample. The single B toxic sample had 244.1 $mg \cdot kg^{-1}$ B. Symptoms then began to manifest in the lower leaves; yellow spots appeared on the margin. Upper leaves became more greenish-yellow and exhibited a downward curl. Isolated spots then appeared on the inside of the lower leaves. Lower leaf spots then became necrotic and turned a pinkish-tan coloration. The spotting moved upwards in the plant into the middle leaves. At the end of the experiment, greenish-yellow spotting had begun in the upper leaves.

Copper

The symptom of copper (Cu) deficiency appeared in the lower leaves as small yellow spots. Cu concentration in the control sample was 7.4 $mg \cdot kg^{-1}$ Cu and in the Cu deficient sample was 4.4 $mg \cdot kg^{-1}$ Cu.

Iron

Interveinal chlorosis manifested itself on the upper leaves of plants grown in iron (Fe) deficient conditions. Tissue concentrations were not significantly different between the control and Fe deficient tissue. The lack of significance comes from the control average that only had two samples that had adequate tissue. The two control samples averaged had an Fe concentration of 116.1 $mg \cdot kg^{-1}$. The Fe deficient tissue had an Fe concentration of 47.8 $mg \cdot kg^{-1}$. As the plant grew, the interveinal chlorosis intensified in the upper leaves, and lower leaves developed interveinal chlorosis.

Manganese

A yellow line along the margin of upper abutilon leaves was the first symptom of manganese (Mn) deficiency. Control Mn tissue concentration was 153.2 $mg \cdot kg^{-1}$ while the Mn deficient tissue concentration was 28.3 $mg \cdot kg^{-1}$ Mn. As the disorder progressed, this yellow band increased in size and intensified in coloration; without the control one might assume that it was a variegated sport. Middle to upper leaves exhibited symptoms. In the advanced stage the yellowing turned into interveinal chlorosis.

Zinc

Zinc (Zn) deficiency began with the upper leaves exhibiting interveinal chlorosis. Tissue concentration of control and Zn deficient tissue was 24.7 $mg \cdot kg^{-1}$ and 8.7 $mg \cdot kg^{-1}$ Zn, respectively. As the disorder advanced, the interveinal areas began to appear bleached. This bleaching became necrosis. Interveinal chlorosis appeared in the lower leaves. Flowers developed, but they aborted before fully opening. Leaves with advanced symptoms abscised from the plant. Some of the partially necrotic lower leaves remained on the plant. Abutilon then regrew new leaves. These new leaves began to exhibit interveinal chlorosis and necrosis along the leaf margin and in isolated spots within the leaf.

Asymptomatic

After 8 weeks of growth when the plants had reached full bloom, plants grown under molybdenum (Mo) deficient conditions exhibited no visual symptoms. The experiment was terminated, and these asymptomatic plants were sampled and analyzed for dry mass and tissue concentration to determine if non-visual differences were evident. There was inadequate sample to determine Mo concentration of either the control or Mo deficient plants.

Conclusions

Symptoms of N, P, and Fe deficiencies and B toxicity manifested early. These disorders maybe more likely problems encountered by growers.

Abutilon exhibited some atypical symptoms. When Mn deficiency first manifested, the abutilon plant looked as if it exhibited varigation. However, its manifestation in more plants confirmed that it was a symptom. Growers should take note if they observe variegation in a nonvariegated crop it may indeed by Mn deficiency. Cu deficiency also manifested in abutilon. Cu deficiency is typically difficult to induce (Barnes, 2010), but it appeared as small spots on lower leaves.

It was also atypical for all 13 treatments to exhibit no significant differences in tissue dry weight. Differences in the control and deficient averages for N, Cu, and Zn deficiencies can be considered significantly different while N was close with a P value of 0.07. A high N deficient tissue dry weight, a low Cu control dry weight, a high Zn deficient dry weight and low Zn control dry weight resulted in these three comparisons not being significantly different.

This study demonstrated that other nutritional disorders occur for abutilon. Symptoms that are described and the reported critical tissue concentrations will help growers diagnosis nutritional orders that arise in their greenhouses when producing this crop.

Acknowledgements

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative, and we would like to thank PanAmerican Seed for abutilon seed.

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Table 2.1 *Abutilon* ×*hybridum* 'Bella Yellow' plant dry weight and 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**
	Dry Weight (g)												
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	0.62	0.62	8.60	1.07	1.07	1.41	2.36	0.62	2.36	0.62	1.07	8.60	1.41
Treatment	0.29	0.42	8.56	1.06	0.81	1.38	2.86	0.45	1.65	0.61	1.06	7.64	0.90
p-value[1]	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

	Tissue Nutrient Concentration (%)						**Tissue Nutrient Concentration ($mg\cdot kg^{-1}$)**						
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	4.98	0.39[2]	4.60	2.94	0.73	0.38	35.5	36.6[2]	7.4	116.1[2]	153.2	—[3]	24.7
Treatment	2.14	—[3]	0.86	0.68	0.36	0.17	6.3	244.1[4]	4.4	47.8	28.3	—[3]	8.7
p-value[1]	***	NS	***	***	***	***	***	NS	***	NS	*	NS	***

[1] *, **, or *** indicates statistically significant differences between sample means based on *F* test at $P \leq 0.05$, $P \leq 0.01$, or $P \leq 0.001$, respectively. NS (not significant) indicates the F test difference between sample means was $P > 0.05$.

[2] Concentration listed is averaged between two samples.

[3] Insufficient sample was available to determine concentration.

[4] Only one sample was used in the number presented.

Chapter 3

Characterization of Nutrient Disorders of *Coreopsis grandiflora* 'Santa Fe Yellow'

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Coreopsis grandiflora* 'Santa Fe Yellow'

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Keywords: Calcium, Magnesium, Micronutrients, Nitrogen, Phosphorus, Potassium, Sulfur

Abstract

***Coreopsis grandiflora* 'Santa Fe Yellow' plants were grown in silica sand culture to induce and photograph symptoms of nutritional disorders. 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. Boron toxicity was also induced by increasing the element 10× higher than the complete nutrient formula. Reagent grade chemicals and deionized water of 18-mega ohms purity were used to formulate treatment solutions. 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. Of the thirteen treatments, eleven exhibited symptoms.**

Typical symptomology of nutrient disorders and critical tissue concentrations are presented. Symptoms of nitrogen, phosphorus, potassium, calcium, sulfur, and iron deficiencies were some of the first disorders to manifest in coreopsis, and these disorders may be a more likely problem encountered by a grower.

Introduction

Coreopsis (*Coreopsis grandiflora*, Asteraceae family) is a short lived hardy perennial used for cut flowers (Dole and Wilkins, 2005) and as an ornamental garden plant (Armitage, 2001). In order to produce plants for purchase, growers need to understand the nutritional requirements of coreopsis. Recommendations were provided on how to fertilize the crop (Hamrick, 2003; Dole and Wilkins, 2005); however, little information is provided on how to recognize nutritional disorders. This experiment was conducted to provide growers with diagnostic tools of disorder symptomology descriptions and critical nutrient values should problems arise.

Materials and Methods

Single 'Santa Fe Yellow' coreopsis plugs (2.0 x 2.0 x 2.6 cm cell size) were transplanted into 13.7 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 15 June 2009. In order to get adequate tissue for nitrogen, phosphorus, and iron, a second round of the experiment was conducted. Extra plugs were potted into a 72-plug

(4.0 x 4.0 x 5.8 cm) tray. Plants were fertigated as needed using 150 $mg \cdot L^{-1}$ N from 13-2-13 Cal-Mag (SQM North America, Atlanta, GA). On 26 June 2009 these plants were installed in the previously described manner. This experiment was conducted in a glass greenhouse in Raleigh, NC at 35°N latitude. Plants were grown at 21˚C day and 18˚C night temperature set points. Treatments started immediately upon transplanting. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2 \cdot 4H_2O$, KH_2PO_4, $MgSO_4 \cdot 7H_2O$, FeDTPA, $MnCl_2 \cdot 4H_2O$, $ZnCl_2$, $CuCl_2 \cdot H_2O$, H_3BO_3, and $Na_2MoO_4 \cdot 2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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/or mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, three pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 dry 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 $\leq$ 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 placing it 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.). The experiment was terminated on 28 July 2009, and non-symptomatic plants of copper and molybdenum 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 five separate harvest dates. Values for percentage difference in plant dry weights were presented if control and treatment tissues were significantly different. Unless otherwise noted, values for tissue concentrations presented were significantly different. A survey range of tissue values for *C. grandiflora* 'Sunray' was published by Mills and Jones (1996a), and each is listed in comparison to the value obtained from this experiment.

Nitrogen

Nitrogen (N) deficient plants developed an overall greenish-yellow coloration. Deficient plants were 68% smaller than the controls. Control tissue had 5.37% N while N deficient tissue had 1.56% N. The survey range Mills and Jones (1996a) published was 3.18-3.33% N. The control value was higher than this range, and the deficient value was lower. Some lower leaf veins exhibited chlorosis. As the disorder progressed, necrotic spots appeared on lower foliage.

Phosphorus

Small blackish-green spots appeared on the lower leaves of plants grown under phosphorus (P) deficient conditions. At the time of harvest, P deficient plants were 21% smaller than the controls. P tissue concentration of control plants was 0.46%. There was inadequate sample to determine P tissue concentration. Mills and Jones (1996a) stated that the survey range for P was 0.29-0.31%, and the control was above this range. As the disorder progressed blackish-green spots covered all the leaves. The leaf ends then became necrotic with black coloration. The necrosis then advanced inward on the leaf.

Potassium

Older leaves on plants grown under potassium (K) deficient conditions exhibited darkening veins, which progressed to a tannish coloration, necrotic spotting, and larger necrotic spots that formed on the margin. Tissue from those plants had 0.41% K, and the control had 6.77% K. When compared with the survey range of 2.73-3.33% presented by Mills and Jones (1996a), the control was above the range while the deficient value was below the range. K deficient plants were 49% smaller than the control. As the disorder progressed, lower affected leaves began to develop a yellow coloration. This yellow coloration intensified as new young leaves grew and exhibited no symptomology. Lower leaves became fully necrotic.

Calcium

Calcium (Ca) deficient plant leaves exhibited poor elongation overall, giving the plant the appearance of being smaller. Plants grown under Ca deficient conditions were 60% smaller than the control plants. Control plant tissue Ca concentration was 1.78%, and Ca deficient Ca tissue concentration was 0.46%. The survey range for Ca is 1.14-1.33% (Mills and Jones, 1996a). The control value fell above this range while the Ca deficient concentration fell below this range. Also, necrosis on some young leaves was observed in the leaf midrib. As the disorder progressed necrosis covered the entire leaf and more leaves became affected. Some plants then died.

Magnesium

Overall plant leaves grown under magnesium (Mg) deficient conditions exhibited a greenish-yellow coloration. A significant difference was not observed between control and Mg deficient dry weights. Mg concentration in control tissue was 0.65% and in Mg deficient tissue was 0.05%. Mg concentration survey range published by Mills and Jones (1996a) was 0.49-0.50%. The control Mg concentration was above this range, and the Mg deficient concentration was below this range. The yellow coloration intensified. Necrotic areas began to form along the margins near the tips of the larger leaves. These necrotic spots then began to increase in size as more leaves began to exhibit necrotic spotting.

Sulfur

Young leaves developing a yellow coloration was the initial symptom of sulfur (S) deficiency. S deficient plant dry weight was 40% less than the control. Control tissue concentration was 0.31% which was within the S concentration survey range of 0.28-0.32% presented by Mills and Jones (1996a). The S deficient tissue concentration was 0.10%, and it fell below the range (Mills and Jones, 1996a). This yellow coloration then manifested in all the leaves. Also, the underside of lower leaves developed a reddish coloration.

Boron

Leaves on plants grown under boron (B) deficiency exhibited less elongation. Plants appeared shorter, but a significant difference was not observed between control and B deficient dry weights. The youngest leaves also felt thicker than the control plants. B concentration in control tissue was 75.1 $mg \cdot kg^{-1}$ while B deficient tissue concentration was 7.5 $mg \cdot kg^{-1}$. Young lobed leaves then became deformed. In the latter stages of the disorder, the tips of the larger leaves began to exhibit interveinal whitening that progressed inward. This area then became necrotic.

Older leaves were the first to exhibit B toxicity symptoms; the outer margin of the leaf exhibited yellow discoloration. A significant difference in dry weight did not occur between the two treatments. Control tissue B concentration was 75.1 $mg \cdot kg^{-1}$, and B toxic tissue concentration was 436.0 $mg \cdot kg^{-1}$. More yellow coloration then developed along the margin. Upper leaves also began to exhibit the marginal yellowing.

Mills and Jones (1996a) published a survey range of 26-30 $mg \cdot kg^{-1}$ for B concentration. Control values for both treatments were higher than the range. The same control sample which had a value of 75.1 $mg \cdot kg^{-1}$ was used as a comparison for both B deficient and B toxic treatments because they manifested symptomology at the same time and were included in the same harvest. B deficient tissue concentration was lower than the range, and B toxic tissue concentration was higher than both this range and the control.

Iron

Interveinal chlorosis of the young leaves was the initial symptom of iron (Fe) deficiency. A significant difference did not occur between control and Fe deficient plant dry weights. Control tissue Fe concentration was 40.5 $mg \cdot kg^{-1}$. There was inadequate Fe deficient tissue to determine Fe concentration. The survey range published by Mills and Jones (1996a) was 53-61 $mg \cdot kg^{-1}$. The control concentration fell below this range. Interveinal chlorosis then manifested in the lower leaves.

Manganese

The initial symptom for manganese (Mn) deficiency was tertiary vein interveinal chlorosis on middle and upper leaves, and the overall appearance was a “honeycombing” effect. There was not a significant difference between control and Mn deficient plant dry weights. Mn concentration in control and Mn deficient tissue was 69.2 $mg \cdot kg^{-1}$ and 9.0 $mg \cdot kg^{-1}$. The survey range published by Mills and Jones (1996a) was 74-101 $mg \cdot kg^{-1}$. The control value is just below this range, and the Mn deficient concentration was much lower than this range. The interveinal chlorosis then manifested in lower leaves, and the coloration intensified.

Zinc

The initial symptoms of zinc (Zn) deficiency were a lack of leaf elongation and yellowing or interveinal chlorosis in young leaves. Zn deficient plant dry weight was 48% less than the control. Control plants had a Zn concentration of 17.5 $mg \cdot kg^{-1}$. Zn deficient plants had a concentration of 10.1 $mg \cdot kg^{-1}$. When compared with Mills and Jones's (1996a) survey range of 64-75 $mg \cdot kg^{-1}$, both the control and deficient concentrations were below the range. However, the deficient value was lower than the control. As the disorder progressed, the yellow coloration intensified. Some of the lower leaves then began to exhibit necrotic spots. These spots increased in size until the entire leaf became necrotic.

Asymptomatic

After 6 weeks of growth when the plants had reached full bloom, plants grown under copper (Cu) and molybdenum (Mo) deficient conditions exhibited no visual symptoms. The experiment was terminated, and these asymptomatic plants were sampled and analyzed for dry mass and tissue nutrient concentrations to determine if non-visual differences were evident.

Control and Cu deficient dry weights were not significantly different. Control tissue Cu concentration was 6.4 $mg \cdot kg^{-1}$ while Cu deficient tissue concentration was 1.2 $mg \cdot kg^{-1}$. The survey range presented by Mills and Jones (1996a) was 4-7 $mg \cdot kg^{-1}$ Cu. The control value fell within this range, and the Cu deficient value fell below this range. Although symptoms may not manifest with Cu deficiencies, having the Cu tissue concentrations available for greenhouse growers will be helpful when they try to determine if Cu is limited.

A significant difference did not occur between control and Mo deficient dry weights. Mo levels were nondetectable in both control and Mo deficient samples. Mills and Jones (1996a) published a survey range of 0.14-0.25 $mg \cdot kg^{-1}$ Mo.

It is helpful for growers to know that plants may be deficient in both of these elements but not manifest symptoms.

Conclusions

Symptoms of N, P, K, Ca, S, and Fe deficiencies were some of the first disorders to manifest in coreopsis. Growers may be more likely to encounter these problems in production. With the advanced stage of S deficiency, plants exhibited an atypical symptom of red foliage undersides. The typical color change associated with S deficiency is greenish-yellow or yellow coloration (Barnes, 2010).

When the deficient tissue concentrations were compared with the values published by Mills and Jones (1996a), all the samples with measurable concentrations fell below the optimum range. Control tissue values varied. They were lower than the survey range (Fe, Mn, Zn), in the survey range (S, Cu), or higher than the survey range (N, P, K, Ca, Mg, B). Values for those that fell outside of the range could be due to variation in the leaf concentration, difference in cultivars, or time of harvest (Mills and Jones, 1996b).

When grown in the nutrient treatment system coreopsis exhibited the described symptoms. Tissue samples were taken when the initial symptoms occurred. The symptomology presented here and critical tissue nutrient levels for the crop will be helpful for commercial growers needing to diagnose coreopsis nutritional problems.

Acknowledgements

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative.

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Table 3.1 *Coreopsis grandiflora* ‘Santa Fe Yellow’ plant dry weight and tissue nutrient concentration as affected by deficient or toxic nutrient treatments, and published optimum concentrations.

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	1.26	1.26	1.22	1.22	1.36	1.22	3.01	3.01	8.59	1.26	3.01	8.59	1.36
Treatment	0.40	1.00	0.62	0.49	1.25	0.73	3.03	2.82	6.82	1.21	1.58	8.98	0.71
p-value[1]	***	*	***	***	NS	***	NS	NS	NS	NS	NS	NS	**
	Tissue Nutrient Concentration (%)						**Tissue Nutrient Concentration ($mg\cdot kg^{-1}$)**						
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	5.37	0.46	6.77	1.78	0.65	0.31	75.1	75.1	6.4	40.5	69.2	—[2]	17.5
Treatment	1.56	—[3]	0.41	0.46	0.05	0.10	7.5	436.0	1.2	—[3]	9.0	—[2]	10.1
p-value[1]	***	NS	***	***	***	***	***	***	**	NS	***	NS	*
Sufficiency Range for *Coreopsis grandiflora* 'Sunray'[4]	3.18 - 3.33	0.29 - 0.31	2.73 - 3.33	1.14 - 1.33	0.49 - 0.50	0.28 - 0.32	26 - 30	26 - 30	4 - 7	53 - 61	74 - 101	0.14 - 0.25	64 - 75

[1] *, **, or *** indicates statistically significant differences between sample means based on F test at $P \leq 0.05$, $P \leq 0.01$, or $P \leq 0.001$, respectively. NS (not significant) indicates the F test difference between sample means was $P > 0.05$.

[2] Tissue Mo concentration was below the detectable limit of 1 $mg\cdot kg^{-1}$.

[3] Insufficient sample was available to determine concentration.

[4] Mills and Jones, 1996

Chapter 4

Characterization of Nutrient Disorders of
***Cosmos bipinnatus* 'Sonata™ White'**

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Cosmos bipinnatus* 'Sonata™ White'

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Keywords: Calcium, Magnesium, Micronutrients, Nitrogen, Phosphorus, Potassium, Sulfur

Abstract

***Cosmos bipinnatus* 'Sonata ™ White' plants were grown in silica sand culture to induce and photograph symptoms of nutritional disorders. 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. Boron toxicity was also induced by increasing the element 10× higher than the complete nutrient formula. Reagent grade chemicals and deionized water of 18-mega ohms purity were used to formulate treatment solutions. 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. Of the thirteen treatments, nine exhibited symptoms.**

Typical symptomology of nutrient disorders and critical tissue concentrations are presented. Symptoms of N and P deficiencies and B toxicity appeared early, and these disorders may be a more likely problem encountered by a grower.

Introduction

Cosmos (*Cosmos bipinnatus*, Asteraceae family) is an annual plant grown primarily as a cut flower (Dole and Wilkins, 2005a). Growers who produce cosmos need to have an understanding of the crop fertility and potential nutrient disorders that occur. Nutrient disorders symptoms and critical nutrient tissue concentrations have not been published. Therefore, this experiment was conducted to ascertain what symptoms occur and the critical values for specific nutrient disorders.

Materials and Methods

'Sonata ™ White' cosmos seed was sown into 72-cell plug trays (4.0 x 4.0 x 5.8 cm cell size) containing Fafard 4P on 20 Aug. 2009. Seedlings were fertigated as needed using 150 $mg{\cdot}L^{-1}$ N from 13-2-13 Cal-Mag (SQM North America, Atlanta, GA). Single plugs were transplanted on 17 Sept. 2009 into 12.7 cm diameter (0.76 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]. This experiment was conducted in a glass greenhouse in Raleigh, NC at 35°N latitude. Plants were grown at 21˚C day and 18˚C night temperature set points. Treatments started immediately upon transplanting. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2 \cdot 4H_2O$, KH_2PO_4, $MgSO_4 \cdot 7H_2O$, FeDTPA, $MnCl_2 \cdot 4H_2O$, $ZnCl_2$, $CuCl_2 \cdot H_2O$, H_3BO_3, and $Na_2MoO_4 \cdot 2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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/or mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, three pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 dry 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 $\leq$ 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 placing it 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.). The experiment was terminated on 20 Oct. 2009, and non-symptomatic plants of copper, iron, manganese, and molybdenum 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 five separate harvest dates. Values for percentage difference in plant dry weights are presented if control and treatment tissues were significantly different. Unless otherwise noted, values for tissue concentrations presented were significantly different.

Nitrogen

The initial symptoms of plants grown under nitrogen (N) deficient conditions included smaller leaf size and less growth. N deficient plants had 56% less dry weight than the control plants. Control tissue N concentration was 6.57% while N deficient tissue concentration was 1.86%. As the disorder progressed N deficient plants developed an overall greenish-yellow coloration.

Phosphorus

Phosphorus (P) deficient plants exhibited necrotic spots on the lower leaves and had yellow stem coloration. Plants grown under P deficient conditions had 54% less dry weight than the control plants. P tissue concentration was 0.73% in the control plants. The P concentration was 0.10% in the deficient plants. No advanced symptoms were observed.

Potassium

Plants grown under potassium (K) deficient conditions exhibited overall brown necrosis, primarily in the tips of leaves. Some spots also appeared across the leaf blades. Tissue dry weights were similar. K concentrations in control and K deficient plants were 7.11% and 1.54%, respectively. No advanced symptoms were observed.

Calcium

Calcium (Ca) deficiency symptoms manifested as brown necrosis in leaf tips of young axillary shoots and in flower buds. Some upper leaves also exhibited isolated gray-green spotting in the foliage. Ca concentration in the control was 1.29% and in the Ca deficient tissue was 0.24%. Dry weights were similar. The gray-green spotting progressed to a tan-brown coloration. In the advanced stage, flower stems also exhibited toppling, a condition where the stem below the flower collapses. It is a disorder associated with Ca deficiency (Dole and Wilkins, 2005b). Necrosis also spread in leaves and flower buds.

Magnesium

Brown necrotic areas forming in scattered lower-leaved areas on plants was the initial symptom of magnesium (Mg) deficiency. Tissue dry weights for the control and Mg deficient tissue were not significantly different. Tissue concentrations for the control and Mg deficient tissue were 0.42% Mg and 0.16% Mg, respectively. As the disorder progressed, necrotic spots dried out. No advanced symptoms were observed.

Sulfur

Sulfur (S) deficiency manifested in the flower peduncles; they developed a whitish-green coloration. A significant difference did not occur between control and deficient plant dry weights. Control tissue concentration was 0.47% S, and S deficient tissue concentration was 0.21% S. No advanced symptoms were observed.

Boron

Plants grown under boron (B) deficiency exhibited distorted growth in the young leaves of the apical bud and axillary shoots. This young growth also exhibited a water-soaked appearance. Plant dry weight of the B deficient plants was 25% less than the controls. B concentration in the control was 70.5 $mg \cdot kg^{-1}$, and it was 15.6 $mg \cdot kg^{-1}$ in the B deficient plants. As the disorder progressed, lower and middle leaves began to exhibit a whitish-green coloration that advanced to brownish-green necrosis.

Brown tips appeared on lower leaves of plants grown under B toxicity conditions. A significant difference did not occur between the two treatment tissue dry weights. B tissue concentrations in the control and B toxic tissue were 69.4 $mg \cdot kg^{-1}$ and 431.2 $mg \cdot kg^{-1}$, respectively.

Zinc

Zinc (Zn) deficient plants exhibited less growth, and Zn deficient plant dry weight was 58% less than the control. Zn concentration in the control was 36.1 $mg \cdot kg^{-1}$ while the

Zn deficient tissue concentration was 25.1 mg·kg^{-1}. No addition symptoms were observed. Other species typically manifest advanced Zn deficiencies (Barnes, 2010), but the earlier harvest may not have allowed the symptoms to develop.

Asymptomatic

After 5 weeks of growth when the plants had reached full bloom, plants grown under copper (Cu), iron (Fe), manganese (Mn), and molybdenum (Mo) deficient conditions exhibited no visual symptoms. The experiment was terminated, and these asymptomatic plants were sampled and analyzed for dry mass and tissue concentration to determine if non-visual differences were evident.

For all four asymptomatic deficiencies, significant differences were not observed between the control and treatment dry weights.

Copper (Cu) tissue concentrations were significantly different at $P = 0.06$. Control tissue Cu concentration was 10.3 mg·kg^{-1}, and Cu deficient tissue Cu concentration was 7.3 mg·kg^{-1}. Iron (Fe) tissue concentrations in control and deficient plants were 178.3 mg·kg^{-1} and 72.3 mg·kg^{-1}, respectively. Tissue concentrations of manganese (Mn) in the control plants were 129.7 mg·kg^{-1} and 47.3 mg·kg^{-1} in the Mn deficient plants. Although symptoms may not manifest with Cu, Fe, and Mn, the tissue concentrations will provide greenhouse growers the means to determine if these elements are limited.

Molybdenum (Mo) tissue concentrations in both the control and Mo deficient tissue were nondetectable which indicates the leaf tissue concentration was below the lower limit of 1 mg·kg^{-1}.

For all four deficiencies it is helpful for growers to know that plants may be deficient in these elements, but the plants may not manifest symptoms.

Conclusions

Symptoms of N and P deficiencies and B toxicity appeared early. Therefore, these disorders may be a more likely problem encountered by a grower. It was atypical for this crop to not manifest Fe and Mn deficiency symptoms. Cu and Mo deficiencies are hard to induce, but Fe and Mn deficiencies typically manifest in a crop (Barnes et al., 2010). While it may be said that cosmos is tolerant of Fe and Mn deficiencies, a compounding factor may have been the early flowering of the crop. The plants may not have grown long enough to induce the deficiencies. Future work could investigate these deficiencies by growing cosmos under long days since it is a facultative short day plant (Dole and Wilkins, 2005a). According to Dole and Wilkins (2005a), without the short day treatment cosmos will not flower or flowering will be irregular until days are under 14 hours.

Cosmos exhibited the described symptoms when grown in the nutrient treatment system. Tissue samples were taken when the initial symptoms occurred. Symptomology presented here and the reported critical tissue nutrient levels for this crop will be helpful for commercial growers needing to diagnose cosmos nutritional problems.

Acknowledgements

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative, and we would like to thank PanAmerican for cosmos seed.

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Table 4.1 *Cosmos bipinnatus* 'Sonata™ White' plant dry weight and 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
	Dry Weight (g)												
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	2.45	1.82	4.53	2.94	5.17	4.53	2.94	1.82	5.17	5.17	5.17	5.17	4.53
Treatment	1.07	0.84	3.17	2.66	4.60	3.64	2.22	1.47	4.18	4.73	4.04	5.31	1.89
p-value[1]	***	*	NS	NS	NS	NS	*	NS	NS	NS	NS	NS	**
	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	6.57	0.73	7.11	1.29	0.42	0.47	70.5	69.4	10.3	178.3	129.7	—[2]	36.1
Treatment	1.86	0.10	1.54	0.24	0.16	0.21	15.6	431.2	7.3	72.3	47.3	—[2]	25.1
p-value[1]	***	*	***	***	***	***	***	***	NS	*	***	NS	*

[1] *, **, or *** indicates statistically significant differences between sample means based on *F* test at $P \leq 0.05$, $P \leq 0.01$, or $P \leq 0.001$, respectively. NS (not significant) indicates the F test difference between sample means was $P > 0.05$.

[2] Tissue Mo concentration was below the detectable limit of 1 mg·kg^{-1}.

Chapter 5

Characterization of Nutrient Disorders of *Dahlia* ×*hybrida* 'Maxi Morelia'

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Dahlia ×hybrida* 'Maxi Morelia'

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Keywords: Calcium, Magnesium, Micronutrients, Nitrogen, Phosphorus, Potassium, Sulfur

Abstract

***Dahlia ×hybrida* 'Maxi Morelia' plants were grown in silica sand culture to induce and photograph symptoms of nutritional disorders. 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

Dahlia (*Dahlia ×hybrida*, Asteraceae family) is a floriculture crop grown for garden use, cut flower production, and as a potted plant (Dole and Wilkins, 2005). Nutritional recommendations vary based on growing stage and conditions.

When sowing seed in containers, a soilless substrate with pH 5.5 to 5.8 should be used (Hamrick, 2003). She suggested that plants grown from seed should be fertilized once cotyledons fully expand with 50 to 75 $mg \cdot L^{-1}$ N from 14-0-14 (in $N:P_2O_5:K_20$) for 5 to 7 days. The rate should be increased to 100 to 150 $mg \cdot L^{-1}$ N for the next two to four weeks depending on tray size, and the source should alternate weekly between 14-0-14 and 20-10-20. Finally, plugs can be hardened for an additional week before potting and during this time fed 14-0-14 at 100 to 150 $mg \cdot L^{-1}$ N.

If the plants are grown from cuttings use a medium with a pH of 5.8 to 6.2 (Yoder, 1998); Hamrick (2003) reinforces this recommendation. Claassen (1992) recommended a rate of 250 to 300 $mg \cdot L^{-1}$ N from 20-10-20 for vegetatively propagated dahlietta, dahlias specifically bred for pot culture. Yoder (1998) recommended that dwarf dahlias be fertigated with 200 to 250 $mg \cdot L^{-1}$ N primarily in the nitrate form such as 20-10-20, 20-5-19, or 21-5-20. That recommendation also came with the advice to test the level of soluble salts to prevent elevated electrical conductivity (EC). Hamrick (2003) states that plants grown from cuttings should be fertilized every other irrigation with 250 to 300 $mg\cdot L^{-1}$ N by alternating between 20-10-20 and 14-0-14 or 15-0-15.

For pot culture it is important to monitor salt levels, pH, and nutrition for quality plant production (De Hertogh, 1996). Hamrick (2003) advised that once potted, plants should be supplied 250 to 300 $mg\cdot L^{-1}$ N on every other irrigation by alternating the N source between 15-16-17 and 20-10-20. Dole and Wilkins (2005) stated that each watering should supply 200 $mg\cdot L^{-1}$ N from 20-10-20; this regime can be substituted with controlled-release fertilizer.

For tubers Durso and De Hertogh (1977) used single or multiple applications of Osmocote or fertigated weekly with 200 $mg\cdot L^{-1}$ N from 20-20-20. Aimone (1985) suggested that tubers be fertilized with a balanced N, P, and K fertilizer such as 20-20-20 after shoot growth begins. With 20-20-20 ammonium build up should not be a problem if there are many sunny spring days. Also, growers can add slow-release fertilizer like Osmocote 14-14-14 to the mix at a rate of 2.97 $kg\cdot m^{-3}$. Hamrick (2003) also states that plants grown from tubers can be grown with a slow-release fertilizer added to the mix. Those mixes derived from composted bark are good for growing tubers. Or, growers may choose to use a constant liquid feed rate of 250 to 300 $mg\cdot L^{-1}$ N from 20-10-20; 14-0-14 or 15-0-15 should be alternated with this source. The fertilization regime should be started after the tubers sprout. The EC should be lowered once every week to ensure that there is not an accumulation of salts.

De Hertogh and Nard (1993) stated that dahlias should be grown in well-drained soil that contains some sand and organic matter with a pH 6 to 7. Field production plants can either be side dressed with dry fertilizer or provided with it when spring growth first emerges (Dole and Wilkins, 2005).

Few nutritional problems are reported. El-Gamassy and Moustafa (1963) found that nitrogen (N) and phosphorus were important for growth and that potassium (K) and calcium (Ca) influenced flower and tuberous root number. Ammonical-N should be avoided through all stages of production as it encourages soft growth (Hamrick, 2003).

Growers need to be able to recognize nutritional problems on site, and the lack of information about dahlia nutritional disorders is disadvantageous. Therefore, this study was conducted to document the visual symptoms and diagnostic tissue values that occur with each nutrient deficiency.

Materials and Methods

Single 'Maxi Morelia' dahlia plugs (2.7 x 3.1 x 4.1 cm cell size) were transplanted into 12.7 cm diameter (0.76L) 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 9 March 2009. This experiment was conducted in a glass greenhouse in Raleigh, NC at 35°N latitude. Plants were grown at 18˚C day and 15˚C night temperature set points. Treatments started immediately upon transplanting. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2{\cdot}4H_2O$, KH_2PO_4, $MgSO_4{\cdot}7H_2O$, FeDTPA, $MnCl_2{\cdot}4H_2O$, $ZnCl_2$, $CuCl_2{\cdot}H_2O$, H_3BO_3, and $Na_2MoO_4{\cdot}2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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/or mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, three pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 placing it 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.). The experiment was terminated on 05 May 2009, and non-symptomatic plants of copper and molybdenum 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 four separate harvest dates. Values for percent difference in plant weights and tissue concentrations are presented.

Nitrogen

Nitrogen (N) deficient plants initially exhibited a magenta color in the stem and petioles and yellow-green leaves; plant leaves were poorly elongated. Dry weights for control and N deficient tissue were not significantly different. For the control, two samples did not have enough mass to determine N concentration. The sample that did have enough mass had a concentration of 7.86%. All three N deficient samples did not have enough tissue to determine N concentration. The disorder progressed with the edge of the leaf becoming yellow and tan necrotic spots appearing along the margin. The entire leaf then became necrotic.

Phosphorus

Phosphorus (P) deficiency began with the stem and petioles developing a magenta coloration. P deficient plants were 68% smaller than the control plants. However, the tissue concentrations for control and P deficient tissue were not significantly different. One of the control plants had a P value of 0.23% while the value of the other two averaged 0.89%. The P deficient tissue concentration was 0.18%. The lower value of the control accounts for the lack of significance. Older leaves then began to develop a yellow-green coloration on the outside of the leaf; young leaves were green. The yellow-green coloration then spread over the older leaves and was isolated to the interveinal region. Small tan spots appeared near the midrib. Also, brown necrotic spots appeared on margin and advanced inward over time. The youngest leaves also had a black green coloration. Lower leaves became completely necrotic.

Potassium

Large brown slightly sunken necrotic spots appearing near the margin on lower leaves of dahlia was the initial symptom of potassium (K) deficiency. K deficient plants were 41% smaller than the control plants. The K control tissue concentration was 7.19% while the K deficient plants had a K concentration of 0.86%. These lower leaves then exhibited wilting tissue which became necrotic and dry. As the disorder progressed, necrotic spots began to appear in middle and then upper leaves. Variation in spotting ranged from a few large necrotic spots on the margin to several smaller necrotic spots located throughout the leaf. Necrosis was also observed in the petioles. In the advanced stage all leaves became almost fully necrotic. Flowers either aborted or failed to open.

Calcium

Middle and upper leaves on plants grown in calcium (Ca) deficient conditions initially exhibited small necrotic black sunken spots between secondary veins near the midrib. Ca deficient plants were 47% smaller than the control plants. Tissue Ca concentrations were significantly different with the control having 2.33% while the Ca deficient plants had 0.34%. As the plants grew these spots enlarged, crossed the secondary veins, and merged into one large black necrotic spot. On some leaves this necrotic area separated the tip and base of the leaf; the tip developed a whitish-green coloration. Lower leaves began to exhibit marginal yellowing. Necrosis of the entire leaf followed. Flower buds formed, but aborted before opening. Eventually, almost the entire plant became necrotic.

Magnesium

The two initial symptoms of magnesium (Mg) deficiency were distorted upper leaves and lower leaf interveinal chlorosis. Dry weights for control and Mg deficient plants were not significantly different. Tissue concentrations were significantly different with the control having 0.93% Mg while the Mg deficient tissue had a value of 0.22%. This disorder progressed with a blackish discoloration appearing in the petiole and grayish-green sunken spots appearing along the midrib. The spot color then changed to tan-brown. The entire plant developed a green-yellow coloration. When the plants flowered the blooms were much lighter in color. The typical pinkish-purple blooms were much whiter.

Sulfur

Sulfur (S) deficient plants exhibited an overall greenish-yellow coloration. Also, the petioles and stem exhibited a magenta coloration. Plants grown in S deficient conditions were 43% smaller than the control plants. Tissue concentrations for control and S deficient plants were 0.32% and 0.14%, respectively. Plant leaves then became more yellow in coloration. Some new leaves grew from the axillary buds, and these were green in coloration with magenta petioles. However, the lower leaves retained their yellow coloration. Lower leaves developed some necrotic spots.

Boron

The initial symptom that appeared with boron (B) deficiency was distortion in upper leaves. Upper leaves broke off the plant very easily and felt rigid and rubbery. Dry weights were not significantly different, but tissue B concentration was. Control plants had a value of 73.1 $mg \cdot kg^{-1}$ while deficient plants had a value of 7.4 $mg \cdot kg^{-1}$. This disorder progressed with brown spots appearing in the apical buds and tannish spots appearing in some of the distorted leaves. Lower leaves began exhibiting yellowing in the margin. In the advanced stage, apical buds failed, and flowers began to form but aborted or never fully opened. Also, some leaves developed necrotic spots with a grayish-brown coloration. The plants appeared to wilt even though they had adequate water.

Plants exhibiting B toxicity exhibited partial "bull's eye" necrotic spots on the leaf tips where the veins reach the edge of the leaf. The outer leaf coloration was brown, and a black band formed between the brown and healthy green tissue. Neither dry weight nor tissue B concentration were significant. The control had a value of 64.5 $mg \cdot kg^{-1}$. One of the B toxicity plants had a value of 74.0 $mg \cdot kg^{-1}$; the average B concentration of the other two plants was 291.6 mg·kg^{-1}. The lower value accounts for the lack of significance. Eventually, most of the necrotic area became a brown coloration. The necrotic spots merged together into one large necrotic band. Lower and upper foliage began to crinkle in isolated spots. Some of these crinkles eventually became necrotic tannish-colored spots, and some leaves were disfigured. Upper leaves began to exhibit marginal browning. When the plants flowered the sepals and bud scales exhibited browning on the tips. Some lower leaves then became fully necrotic.

Iron

Lower leaves on iron (Fe) deficient plants were initially a greenish-yellow coloration. At the time of sampling a significant difference did not occur between control and Fe deficient dry weights and Fe tissue concentrations. Control Fe concentration was 80.3 mg·kg^{-1}, but two of the Fe deficient tissue concentrations were 85.9 mg·kg^{-1} and 84.8 mg·kg^{-1}. The third had a concentration of 25.5 mg·kg^{-1}. The large difference between the values accounts for the lack of significance. The greenish-yellow coloration then spread over the entire plant.

Manganese

Manganese (Mn) deficient plants initially exhibited a greenish-yellow coloration in the lower leaves, but this coloration was quickly manifested in the entire plant. Mn deficient plants were 21% smaller than the control plants. Control plants had a Mn concentration of 119.5 mg·kg^{-1}, and Mn deficient plants had a Mn concentration of 10.1 mg·kg^{-1}. As the disorder progressed the leaves exhibited a few isolated tannish necrotic spots.

Zinc

Plants grown under zinc (Zn) deficient conditions had lower leaves that exhibited a slight yellow coloration. Zn deficient plants had 52% less dry mass than the control plants. Tissue concentrations were not significantly different. The control plants had an average of 14.3 mg·kg^{-1} Zn. One of the Zn deficient plants had a Zn concentration of 11.7 mg·kg^{-1}. The average of the other two Zn deficient tissue samples was 7.5 mg·kg^{-1}. The higher value accounts for the lack of significance between the control and Zn deficient tissue concentrations. As the disorder progressed the yellow coloration intensified. Bleached necrotic spots also began to appear on the lower leaves, mostly in the interveinal regions. Sections of the lower leaves then became necrotic.

Asymptomatic

After 8 weeks of growth when the plants had reached full bloom, plants grown under copper (Cu) and molybdenum (Mo) deficient conditions exhibited no visual symptoms. The experiment was terminated, and these asymptomatic plants were sampled and analyzed for dry mass and tissue concentration to determine if non-visual differences were evident.

Plants grown under Cu deficient conditions did not have a significantly different dry weight when compared to the control. Differences in Cu tissue concentration were significantly different. The control tissue Cu concentration was 6.8 $mg \cdot kg^{-1}$ while Cu deficient tissue concentration was 2.1 $mg \cdot kg^{-1}$.

There was not a significant difference in dry weights for control plants and those grown under Mo deficient conditions. Mo was only detected in one control sample, and that level was 0.5 $mg \cdot kg^{-1}$. Mo was not detected in any of the Mo deficient samples. The ICP-OES instrument most optimally detects micronutrient levels of approximately 1 $mg \cdot L^{-1}$, but it can detect lower levels. This fact accounts for the samples with undetectable levels of Mo. It should be noted that of the 51 samples taken, 27 had levels of Mo below the detectable limit; therefore, it is also possible that *Dahlia ×hybrida* 'Maxi Morelia' is not a crop that uptakes significant levels of Mo.

Although symptoms may not manifest with Cu and Mo deficiencies, having the tissue concentrations available to greenhouse growers will be helpful when trying to determine if these elements are limited. It is also helpful to know that Mo levels can be extremely low in dahlia crops, even below the detectable limit in crops fed with a balanced fertilizer.

Conclusions

Dahlia exhibited the described symptoms when grown in the aforementioned nutrient deficiency system, and tissue samples were taken when the initial symptoms occurred. Thus, the symptomology and critical tissue nutrient levels for the crop will be helpful for commercial growers needing to diagnose dahlia nutritional problems.

Acknowledgements

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative.

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Table 5.1 *Dahlia ×hybrida* 'Maxi Morelia' plant dry weight and 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
	Dry Weight (g)												
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	0.97	0.97	1.27	3.45	1.27	1.27	1.27	0.97	10.19	0.97	3.45	10.19	3.45
Treatment	0.28	0.31	0.75	1.83	1.51	0.72	1.26	0.73	8.81	0.86	2.74	10.27	1.67
p-value[1]	*	*	**	***	NS	**	NS	NS	NS	NS	**	NS	**
	Tissue Nutrient Concentration (%)						**Tissue Nutrient Concentration ($mg \cdot kg^{-1}$)**						
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	7.86[2]	0.67	7.19	2.33	0.93	0.32	73.1	64.5	6.8	80.3	119.6	0.5[2]	14.3
Treatment	–[3]	0.18	0.86	0.34	0.22	0.14	7.4	219.1	2.1	65.5	10.2	–[3]	8.9
p-value[1]	NS	NS	***	***	***	***	***	NS	**	NS	**	NS	NS

[1] *, **, or *** indicates statistically significant differences between sample means based on *F* test at $P \leq 0.05$, $P \leq 0.01$, or $P \leq 0.001$, respectively. NS (not significant) indicates the *F* test difference between sample means was $P > 0.05$.

[2] Only one sample represented in the number presented.

3 There was inadequate sample to determine concentration.

Chapter 6

Characterization of Nutrient Disorders of *Dianthus* ‘Bouquet Purple’

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Dianthus* 'Bouquet Purple'

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Abstract

***Dianthus* 'Bouquet Purple' plants were grown in silica sand culture to induce and photograph symptoms of nutritional disorders. 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. Boron toxicity was also induced by increasing the element 10× higher than the complete nutrient formula. Reagent grade chemicals and deionized water of 18-mega ohms purity were used to formulate treatment solutions. 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. All thirteen treatments exhibited symptomology.**

Molybdenum (Mo) tissue concentrations were not reported; therefore, symptoms for Mo deficiency cannot be confirmed. Symptoms of nitrogen, calcium, sulfur, and iron deficiencies were the first disorders to manifest in dianthus. Therefore, these disorders may be more likely problems encountered by a grower.

Introduction

Dianthus 'Bouquet Purple' (*Dianthus* sp., Caryophyllaceae family) is an interspecific hybrid dianthus, and the parents have not been released by the breeder (Pilon, 2003). While some dianthus are biennial (SePro, 2000), this cultivar is a perennial that is grown as a garden perennial, a bedding plant, or a cut flower (Pilon, 2003).

Growers who wish to grow dianthus need to understand the fertility of the crop and potential nutritional disorders that may be encountered. Few problems are reported. Sole use of nitrate fertilizer will result in stunted growth of *D. barbatus* (Post, 1949).

Because symptomology of nutrient disorders is not available, this study was conducted to elucidate symptoms and critical nutrient levels for perennial dianthus.

Materials and Methods

Single 'Bouquet Purple' dianthus plugs (2.0 x 2.0 x 2.6 cm cell size) were transplanted into 12.7 cm diameter (0.76 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 17 Sept. 2009. This experiment was conducted in a glass greenhouse in Raleigh, NC at 35°N latitude. Plants were grown at 18˚C day and 15˚C night temperature set points. Treatments started immediately upon transplanting. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2{\cdot}4H_2O$, KH_2PO_4, $MgSO_4{\cdot}7H_2O$, FeDTPA, $MnCl_2{\cdot}4H_2O$, $ZnCl_2$, $CuCl_2{\cdot}H_2O$, H_3BO_3, and $Na_2MoO_4{\cdot}2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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/or mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, three pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 dry 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 $\leq$ 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 placing it 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.). The experiment was terminated on 19 Nov. 2009. 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. Values for percentage difference in plant dry weights are presented when control and treatment tissues were significantly different. Unless otherwise noted, values for tissue concentrations presented were significantly different. While the parents of Dianthus 'Bouquet Purple' are not known, the values in this experiment are compared with survey ranges obtained by sampling perennial dianthus *D. gratianopolitanus* and *D. gratianopolitanus* 'Bath's Pink' (Mills and Jones, 1996).

Nitrogen

Nitrogen (N) deficiency symptoms started with plants exhibiting less growth, an overall yellowing, and a magenta coloration developing in the lower leaves near where the leaves arise from the base. N deficient plant dry weight was 54% less than the control. Control and N deficient tissue concentrations were 5.38% N and 1.49% N, respectively. The survey range Mills and Jones (1996) published for N was 1.52-1.63%. The control value was above this range while the deficient value fell below this range. As the disorder progressed the yellow and magenta coloration intensified. The lowest leaves became completely magenta in color.

Phosphorus

Plants exhibiting less growth was the initial symptom of phosphorus (P) deficiency. Dry weight of plants grown under P deficient conditions was 56% less than the control dry weight. P tissue concentration in the control was 0.82% and in the P deficient tissue was 0.07%. Mills and Jones (1996) survey range for P was 0.32-0.40%. The control P value fell above this range and the deficient P value was below this range. As the disorder progressed, purple coloration began to develop in the lower leaves. Necrosis also manifested in the lower leaves, and it began to advance inward from the tip. In the advance stage of the disorder these symptoms manifested in middle leaves.

Potassium

Lower leaves of plants grown under potassium (K) deficient conditions exhibited necrotic tips. This symptom manifested during the last harvest. A significant difference in dry weight was not observed. Control plants had a K concentration of 6.80% while the K deficient plants had a K concentration of 2.15%. The survey range published by Mills and Jones (1996) for K was 1.96-2.09%. The control was above this range while the deficient value was slightly higher than this range.

Calcium

New foliage on plants grown under the calcium (Ca) deficient regime exhibited crinkled distorted growth near the leaf tips. The affected area exhibited white necrotic spots. No significant difference occurred between the control and Ca deficient dry weights. Ca tissue concentrations in the control and Ca deficient plants were 1.59% and 0.13%, respectively. Mills and Jones (1996) stated that the survey range was 0.58-1.66%. The control value fell within the upper part of this range, and the deficient value was below this range. During the moderate stage of symptomology, young leaves exhibited less growth, and the white necrotic spots increased in size. The apical bud and axillary shoots aborted. New shoots then emerged from the base, but they also exhibited the crinkled distorted growth near the tips and white necrotic spots.

Magnesium

Lower leaves initially exhibited marginal yellowing with magnesium (Mg) deficiency. Dry weights of the control and Mg deficient plants were not significantly different. Mg concentration in the control was 0.49%, and Mg concentration in the Mg deficient tissue was 0.07%. A survey range of 0.22-0.26% Mg was published by Mills and Jones (1996). The control value fell above this range, and the deficient value was below this range. As the disorder progressed the lower leaf yellow coloration intensified, and some affected leaf tips became necrotic. Older leaves also exhibited purple and white leaf discoloration. This discoloration intensified over time. In the advance stage of the disorder the discoloration advanced into the upper leaves of the inflorescence. New shoots began to grow from the base of the plant and exhibited the yellow coloration or tip necrosis.

Sulfur

Sulfur deficiency manifested in upper leaves with the development of a greenish-yellow coloration. There was not a significant difference between control and S deficient tissue dry weights. S concentration in the control tissue was 0.35% while the concentration in the deficient tissue was 0.14%. Mills and Jones (1996) stated that the survey range for S was 0.16-0.20%. The control value fell above this range and the S deficient concentration was slightly lower than this range. As the disorder advanced the yellow coloration intensified and appeared in all plant leaves, and an overall light purple coloration developed in the leaves. Initially, there was little growth of basal shoots, but in the advance stage of the disorder many new shoots began to grow. New shoot tips had a greenish-yellow coloration.

Boron

Plants that grew under boron (B) deficient conditions exhibited a lack of elongation of the inflorescence and basal shoots. The lower leaves also exhibited more pronounced curling and twisting than the control plants. Plants grown under control and B deficient conditions did not exhibit significant differences in plant dry weight. B concentrations in control and B deficient tissue were 43.5 $mg \cdot kg^{-1}$ and 4.0 $mg \cdot kg^{-1}$ respectively. As the disorder progressed, marginal purpling manifested on lower leaves. Some lower leaves exhibited interveinal chlorosis. As the disorder progressed the purpling appeared in the upper leaves, and the inflorescence stalled in growth. New shoots emerged from the base of the plant. Leaves on these new shoots exhibited curling and distorted growth.

B toxicity symptoms began with the lower leaves exhibiting interveinal chlorosis on the leaf tips. A significant difference was not observed in tissue dry weights between the control and B toxic tissues. Control B concentration was 28.3 $mg{\cdot}kg^{-1}$, and B toxic B concentration was 265.2 $mg{\cdot}kg^{-1}$. The interveinal chlorosis advanced to a white tip necrosis. Necrosis advanced inward on the lower leaves of the plant. Necrotic tips then began to manifest on the middle and upper leaves of the plant.

Mills and Jones (1996) published a survey range of 20-47 $mg{\cdot}kg^{-1}$. The control values for both B deficient and B toxic fell within this range. The B deficient value was below this range, and the B toxic value was above this range.

Copper

Leaves on copper (Cu) deficient plants exhibited a bluish-white coloration. Plants grown under Cu deficient conditions had 40% less dry weight than the control. Cu concentration in control plant tissue was 9.8 $mg{\cdot}kg^{-1}$ and was 1.7 $mg{\cdot}kg^{-1}$ in the Cu deficient tissue. The control value was within the published survey range of 8-11 $mg{\cdot}kg^{-1}$, and the deficient value was below this range (Mills and Jones, 1996). No other symptoms were observed.

Iron

Iron (Fe) deficiency manifested in the upper leaves as interveinal chlorosis. A significant difference was neither observed between the control and Fe deficient tissue dry weights nor tissue concentrations. The lack of significance for the tissue concentrations originates from two factors. One of the control plant Fe concentrations was nondetectable. The average between the two remaining was 31.4 $mg\cdot kg^{-1}$. Also, one of the Fe deficient plants concentrations was 50.9 $mg\cdot kg^{-1}$ which is higher than all control and deficient concentrations. The average between the two lower values was 19.1 $mg\cdot kg^{-1}$. Mills and Jones (1996) published a survey range of 42-82 $mg\cdot kg^{-1}$. Both the control and deficient values reported in Table 6.1 fell below this range. As new axillary shoots grew they exhibited a yellow coloration. In the advance stage of the disorder the entire plant had developed an overall greenish-yellow coloration.

Manganese

Plants grown under manganese (Mn) deficiency initially developed a greenish-yellow coloration. A significant difference did not occur between tissue dry weights. Control tissue Mn concentration was 159.5 $mg\cdot kg^{-1}$. Mn deficient tissue concentration was 7.6 $mg\cdot kg^{-1}$ Mn. A survey range of 47-586 $mg\cdot kg^{-1}$ was published by Mills and Jones (1996). The control value falls within this range while the deficient value falls below this range. As the disorder progressed the shoots arising from the base exhibited less growth.

Molybdenum

After 9 weeks of growth when the plants had reached full bloom, plants grown under molybdenum (Mo) deficient conditions exhibited a lack of axillary shoot growth. A significant difference occurred between control and Mo deficient plants. Symptomatic plants had 57% less dry weight than the controls. Tissue Mo concentrations were not significant because Mo was only detected in one control sample; that concentration was 0.7 $mg \cdot kg^{-1}$ Mo. A survey range of 0.12-1.96 $mg \cdot kg^{-1}$ was published by Mills and Jones (1996). This value fell within this range.

Because concentrations were not reported for the symptomatic plants, it cannot be confirmed that the lack in axillary growth is a symptom. Future work sampling more *Dianthus* 'Bouquet Purple' plants could help to elucidate optimum and deficient Mo concentrations and determine if the difference in axillary growth is a symptom.

Zinc

Plants grown under zinc (Zn) deficiency exhibited less growth of basal shoots. This was obvious in the tissue dry weights. Zn deficient tissue dry weight was 29% less than the control. Control tissue concentration was 45.2 $mg \cdot kg^{-1}$ Zn while the deficient plants contained 19.6 $mg \cdot kg^{-1}$ Zn. Both the control and deficient Zn values were below the survey range of 109-148 $mg \cdot kg^{-1}$ (Mills and Jones, 1996).

Conclusions

Symptoms of N, Ca, S, and Fe deficiencies were the first disorders to manifest in dianthus. Therefore, these disorders may be more likely problems encountered by a grower.

This perennial dianthus exhibited some atypical symptomology. K was very late manifesting symptoms. Typically, K appears earlier in the production cycle (Barnes, 2010). A very atypical symptom for B deficiency was initiation of symptoms on lower leaves. Symptoms manifested with Cu deficiency. Cu deficiency is typically difficult to induce (Barnes, 2010), but it manifested in this crop. Mo deficiency is also difficult to induce and in prior studies grown in this system it has not manifested (Barnes, 2010). Based on this study Mo deficient plants exhibited symptomology. However, without the tissue concentrations, the conclusion that the lack in axillary growth was a symptom cannot be made.

Another factor that was atypical was the regrowth of new shoots from the base after a disorder manifested. Typically after a disorder manifested the crop entered a decline and showed little growth. This characteristic was observed with Ca, Mg, S, and B deficiencies. For each deficiency the axillary shoots exhibited the same symptomology that initially appeared in the plant. Growers that can recognize a Ca, Mg, S, or B deficiency early enough may be able to cut the plants back, make a corrective nutrient application, and allow them to flush back out to produce a healthy crop.

When nutrient disorders were induced in dianthus, the plants exhibited the described symptoms. Tissue samples were taken when the initial symptom manifested. The symptomology presented and critical tissue nutrient levels for the crop will be helpful for commercial growers needing to diagnose dianthus nutritional problems.

Acknowledgements

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative.

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Table 6.1 *Dianthus* 'Bouquet Purple' plant dry weight and tissue nutrient concentration as affected by deficient or toxic nutrient treatments and published survey average concentrations.

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	0.22	0.55	9.15	0.27	0.55	0.27	3.19	0.55	9.15	0.21	3.19	9.15	9.15
Treatment	0.10	0.24	7.24	0.30	0.60	0.27	2.74	0.75	5.47	0.25	2.19	3.92	6.54
p-value[1]	**	*	NS	NS	NS	NS	NS	NS	*	NS	NS	**	*
				Tissue Nutrient Concentration (%)					**Tissue Nutrient Concentration ($mg \cdot kg^{-1}$)**				
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	5.38	0.82	6.80	1.59	0.49	0.35	43.5	28.3	9.8	31.4[2]	159.5	0.7[3]	45.2
Treatment	1.49	0.07	2.15	0.13	0.07	0.14	4.0	265.2	1.7	19.1[4]	7.6	—[5]	19.6
p-value[1]	***	***	***	***	***	***	***	***	**	NS	***	NS	***
Survey Average for *D. gratianopolitanus* species and 'Bath's Pink'[6]	1.52 - 1.63	0.32 - 0.40	1.96 - 2.09	0.58 - 1.66	0.22 - 0.26	0.16 - 0.20	20 - 47	20 - 47	8 -11	42 - 82	47 - 586	0.12 - 1.96	109 - 148

[1] *, **, or *** indicates statistically significant differences between sample means based on F test at $P \leq 0.05$, $P \leq 0.01$, or $P \leq 0.001$, respectively. NS (not significant) indicates the F test difference between sample means was $P > 0.05$.

[2] Value presented averaged between two samples where Fe was detectable.

[3] Only one sample was used for the value presented.

[4] An excessive sample concentration was removed, and the average between the other two presented.

[5] Inadequate sample was available to determine concentration.

[6] Mills and Jones, 1996.

Chapter 7

Characterization of Nutrient Disorders of *Fuchsia* ×*hybrida* 'Gartenmeister Bonstedt'

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Fuchsia* ×*hybrida* 'Gartenmeister Bonstedt'

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Keywords: Calcium, Magnesium, Micronutrients, Nitrogen, Phosphorus, Potassium, Sulfur

Abstract

***Fuchsia* ×*hybrida* 'Gartenmeister Bonstedt' 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. Boron toxicity was also induced by increasing the element 10× higher than the complete nutrient formula. Reagent grade chemicals and deionized water of 18-mega ohms purity were used to formulate treatment solutions. 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

Fuchsia (*Fuchsia ×hybrida*, Onagraceae family) is a floriculture crop treated as a perennial in warmer climates and as an annual flowering plant in areas where it is less hardy (Dole and Wilkins, 2005).

Fertilization requirements vary by method of propagation and stage of plant development. Seed are sown in a substrate with a recommended pH of 6.0 to 6.5. Once cotyledons appear, it is recommended 14-0-14 at a concentration of 50 to 75 $mg \cdot L^{-1}$ nitrogen (N) be applied and changed to 20-10-20 at 100 to 150 $mg \cdot L^{-1}$ N when true leaves appear (Hamrick, 2003). Dole and Wilkins (2005) recommended fertilizing with at least 200 $mg \cdot L^{-1}$ N and potassium (K), with less than 20% of that N being in the ammoniacal form.

If propagating from cuttings, the recommended pH ranges vary from 5.0 to 5.5 (Hamrick, 2003) to 6.0 to 6.6 (Heins et al., 1990). There are no reports of low or high pH problems; however, Hamrick (2003) recommended the application of 20-10-20 which is an acidic fertilizer (Whipker et al., 2001) in case the plants become discolored. Cuttings should be fertilized with 50 to 75 $mg \cdot L^{-1}$ N at sticking and increase the 20-10-20 concentration to 100 to 200 $mg \cdot L^{-1}$ N once a week when approximately 50% of the cuttings have initiated roots. It is also recommended to alternate between acidic (20-10-20) and basic (15-0-15) fertilizers.

Fuchsia should be potted in a substrate that is well-drained, disease free, has a high initial starter charge, and a pH of 5.0 to 5.5. Hamrick (2003) recommended to continue alternating between 20-10-20 and 15-0-15 when fuchsia are potted, increase to a fertilizer concentration of 150 to 200 $mg \cdot L^{-1}$ N while the plants are becoming established, and once well rooted, further increase the concentration to 200 to 300 $mg \cdot L^{-1}$ N. She also advised against using fertilizers that are high in phosphorus (P).

Nutritional problems reported pertain to overfertilization or underfertilization. Overfertilization can lead to excessive vegetative growth (Hamrick, 2003) or plants that will not flower or have delayed flowering (Dole and Wilkins, 2005). Poor branching will result if plants are underfertilized (Hamrick, 2003).

Optimum tissue concentrations and symptomology of other nutrient deficiencies are not present in the literature, and the absence of this information is disadvantageous to growers. Therefore, the objectives of this study were to document the symptomology of nutrient disorders and determine tissue values for fuchsia.

Materials and Methods

Single 'Gartenmeister Bonstedt' fuchsia plugs (2.7 x 3.1 x 4.1 cm cell size) were transplanted into 12.7 cm diameter (0.76 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 12 March 2009. This experiment was conducted in a glass greenhouse in Raleigh, NC at 35°N latitude. Plants were grown at 18˚C day and 15˚C night temperature set points. Treatments started immediately upon transplanting. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2{\cdot}4H_2O$, KH_2PO_4, $MgSO_4{\cdot}7H_2O$, FeDTPA, $MnCl_2{\cdot}4H_2O$, $ZnCl_2$, $CuCl_2{\cdot}H_2O$, H_3BO_3, and $Na_2MoO_4{\cdot}2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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/or mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, three pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 $\leq$ 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 placing it 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 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 on 5 May 2009, and non-symptomatic plants grown in molybdenum deficient conditions 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. Values for percentage difference in plant dry weights are presented if control and treatment tissues were significantly different. Unless otherwise noted, values for tissue concentrations presented were significantly different.

Nitrogen

Nitrogen (N) deficiency began with the upper leaves exhibiting a yellow-greenish coloration with a thin band of red on the margin; lower leaves were green. Dry weights of plants grown under N deficiency were not significantly different from the control. Control tissue had a N concentration of 3.66% while the deficient plant had a concentration of 1.72%. As the disorder progressed upper leaves then exhibited a maroon coloration while lower leaves began to exhibit necrotic brown spots. Flower buds began to form but never fully developed. Some plants in the experiment also died in the advanced stages of the disorder.

Phosphorus

Symptoms of phosphorus (P) deficiency initially manifested as greenish-yellow spots in the upper leaves. Control and P deficient plant dry weights were not significantly different. The control tissue had 0.57% P while the deficient had 0.20% P. The middle and lower leaves then began to exhibit large blackish-brown and tan necrotic spots. Lower leaves became completely necrotic and fell off. Leaf drop progressed up the plant over time.

Potassium

The leaves on plants exhibiting potassium (K) deficiency exhibited an "umbrellaing" effect where the upper leaves curled downward. Also, less plant growth was evident, and a significant difference in dry weight was observed. K deficient plants were 42% smaller than the control plants. Values for K concentration in the control and K deficient plants were 3.56% and 0.31%, respectively. Lower leaves then began to develop necrotic areas near the leaf petiole and along the edge; this necrosis progressed upward on the plant to the leaf exhibiting some yellowing and bleached areas. The leaves then fell off the plant from the base upward. In the final stage of K deficiency most of the leaves fell from the plant, and inflorescences formed but never fully developed.

Calcium

Symptoms of calcium (Ca) deficiency began with small spots forming near the stem on most recently matured leaves. These upper leaves also exhibited a slight yellow coloration. Ca deficient plants were 35% smaller than the control. Leaf tissue concentration of the control was 1.52% Ca and 0.15% in the deficient plants. As the disorder progressed the spots became yellow circles surrounded by purplish-red coloration. The lower leaves began to drop. In the advanced stages the entire plant developed a yellow coloration, more leaves dropped, and the leaves that remained on the plant began to exhibit leaf curl. Inflorescences were smaller on the deficient plants than the inflorescences on the control.

Magnesium

With magnesium (Mg) deficiency the disorder initially manifested on lower leaves as white hazing on the half of the leaf near the leaf tip or white bleached spots on the leaves. No significant difference was observed between the control and deficient dry weights. Control plants had a value of 0.36% Mg while the deficient plant had a value of 0.08% Mg. As the disorder progressed the white spots became more prevalent, and leaf drop from the base upward began. In the advanced stage most of the leaves dropped off, but those that remained became bleached.

Sulfur

Symptoms of sulfur (S) deficiency began with middle and upper leaves exhibiting yellowing that was greenish-yellow on the leaf inside and progressed to yellow on the leaf margin. Control and deficient dry weights were not significantly different. Values for control and S deficient plants were 0.28% and 0.11%, respectively. Entire leaves became yellow as the plants grew. Flowers developed, but they had a pink coloration instead of the typical orange-red. These inflorescences eventually aborted. Lateral shoots then began to grow. These lateral shoots had an intense magenta coloration with some yellowing on the expanded part of the leaf.

Boron

Boron (B) deficiency initially manifested itself with a golden-brown color near the base of the young expanding leaves; this area also had a shiny appearance. Some of these leaves also exhibited pitted spotting on the leaf. B deficient plants were 27% larger in dry weight than the control plants. Control plants had a B concentration of 39.0 $mg \cdot kg^{-1}$, and B deficient plants had a B concentration of 7.0 $mg \cdot kg^{-1}$. As the disorder progressed some inflorescences aborted; flowers that were able to form and open were deformed. Pitted spotting covered the upper leaves. Also, isolated larger tan necrotic spots also appeared on the leaf tips. The golden color was persistent through the experiment, but the shininess became duller over time. Axillary shoots then began to grow after the apical bud aborted.

The initial symptom of B toxicity was the darkening or browning on the tips of lower fuchsia leaves. Dry weights for the control and B toxic treatments were not significantly different. Values for the control and B toxic plants were 32.1 $mg \cdot kg^{-1}$ and 413.5 $mg \cdot kg^{-1}$, respectively. Some spots appeared on the lower leaves, but it was difficult to distinguish these spots from spots occurring on control plants. Leaf drop began with the lower leaves eight weeks into the experiment, and then progressed upward.

Copper

The initial symptom of copper (Cu) deficiency was the appearance of hazy whitish-green bleached areas on the leaf lower to middle leaves. A significant difference between plant dry weights was not observed. Control tissue had a Cu concentration of 5.7 $mg \cdot kg^{-1}$ while the deficient tissue had a Cu concentration of 1.6 $mg \cdot kg^{-1}$. As the disorder progressed the bleached area spread and became a tannish-brown color.

Iron

Upper recently matured leaves exhibiting yellowing was the initial symptom of iron (Fe) deficiency. This symptomology is atypical from the interveinal chlorosis that is normally observed (Barnes et al., 2010). Control and deficient plant dry weights were not significantly different. Control plants contained 143.8 $mg \cdot kg^{-1}$ Fe, and 72.0 $mg \cdot kg^{-1}$ Fe was in the deficient plants. Over time axillary shoots then began exhibiting yellowing.

Manganese

Plants exhibiting manganese (Mn) deficiency developed a yellowish-green coloration. The yellowing was most prevalent in the tips of axillary shoots. Control and

Mn deficient plants exhibited no significant difference in dry weight. Values for the control and Mn deficient tissue were 84.3 $mg \cdot kg^{-1}$ Mn and 5.2 $mg \cdot kg^{-1}$ Mn, respectively. As the disorder progressed, lower leaves dropped.

Zinc

Zinc (Zn) deficient plants initially exhibited "umbrellaing" where the young leaves curled downward. There was yellow coloration near the petiole. As inflorescences formed flowers were a peach coloration instead of the typical orange-red. Plants grown under Zn deficient conditions had 27% less dry weight than the controls. Control plants had a concentration of 13.4 $mg \cdot kg^{-1}$ Zn while deficient plants had a concentration of 4.7 $mg \cdot kg^{-1}$ Zn. The lower leaves then began to drop. The yellow coloration area near the petiole became necrotic. Seven weeks into the experiment, later inflorescences that formed were smaller than the control inflorescences, aborted, or never fully formed. Both K and Zn deficient plants exhibited umbrella-like leaf growth, but the symptomology of the two can be differentiated by the fact that a Zn deficient plant retains its lower leaves.

Asymptomatic

After 8 weeks of growth when the plants had reached full bloom, plants grown under molybdenum (Mo) deficient conditions exhibited no visual symptoms. The experiment was terminated, and asymptomatic plants were sampled and analyzed for dry mass and tissue concentration to determine if non-visual differences were evident. A significant difference did not occur for control and Mo deficient dry weights. Values for control and Mo deficient tissue were 10.1 $mg \cdot kg^{-1}$ and 2.4 $mg \cdot kg^{-1}$, respectively.

Conclusions

Fuchsia plants were grown under conditions to induce nutrient deficiencies and toxicities, and the documented symptoms manifested in the crop. These descriptions will be helpful for commercial growers needing to diagnose fuchsia nutritional problems.

Because N, P, and S were three of the first deficiencies to manifest in the crop, they may be a more likely problem encountered by growers. Limited information on other nutrient symptomologies and Hamrick's (2003) statement about a lack of N leading to plants that exhibit reduced branching support this conclusion.

Some atypical symptomology manifested in fuchsia. Fe deficiency appeared as leaf yellowing instead of the typical interveinal chlorosis that is observed (Barnes, 2010). Also, Cu deficiency is typically a very difficult problem to induce, but it did with fuchsia (Barnes, 2010).

Acknowledgements

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative.

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Table 7.1 *Fuchsia ×hybrida* 'Gartenmeister Bonstedt' plant dry weight and 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
	Dry Weight (g)												
Element	**N**[1]	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	0.49	0.40	1.25	3.71	0.79	0.40	3.71	1.25	7.76	0.79	7.76	7.76	3.71
Treatment	0.53	0.38	0.72	2.41	0.81	0.39	5.09	1.08	9.60	1.14	7.48	9.29	2.72
p-value[2]	NS	NS	**	*	NS	NS	**	NS	NS	NS	NS	NS	*

	Tissue Nutrient Concentration (%)						Tissue Nutrient Concentration ($mg \cdot kg^{-1}$)						
Element	**N**[1]	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	3.66	0.57	3.56	1.52	0.36	0.28	39.0	32.1	5.7	143.8	84.3	10.1	13.4
Treatment	1.72	0.20	0.31	0.15	0.08	0.11	7.0	413.5	1.6	72.0	5.2	2.4	4.7
p-value[2]	***	***	***	***	***	***	***	***	**	**	***	***	***

[1] Two plants used per sample.

[2] *, **, or *** indicates statistically significant differences between sample means based on F test at $P \leq 0.05$, $P \leq 0.01$, or $P \leq 0.001$, respectively. NS (not significant) indicates the F test difference between sample means was $P > 0.05$.

Chapter 8

Characterization of Nutrient Disorders of
***Gazania rigens* 'Daybreak White'**

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Gazania rigens* 'Daybreak White'

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Keywords: Treasure flower, Calcium, Magnesium, Micronutrients, Nitrogen, Phosphorus, Potassium, Sulfur

Abstract

Gazania rigens **'Daybreak White' plants were grown in silica sand culture to induce and photograph symptoms of nutritional disorders. 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. Boron toxicity was also induced by increasing the element 10× higher than the complete nutrient formula. Reagent grade chemicals and deionized water of 18-mega ohms purity were used to formulate treatment solutions. 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. Out of the thirteen treatments, eleven exhibited symptoms. Deficiency symptoms of N, S, and Ca were first to manifest. Should nutritional disorders arise growers should first consider these three problems when trying to determine the possible cause.

Introduction

Gazania (*Gazania rigens*, Asteracea family) is a tender perennial that is grown as an annual bedding plant. Growers raise plants for hanging baskets, cell packs, or pots (Dole and Wilkins, 2005). Growers who wish to grow gazania need to understand its cultural needs, including its fertility requirements.

Information about the symptomology of nutrient disorders of gazania and critical nutrient levels have not been published. Therefore, growers producing gazania can encounter problems if nutritional disorders manifest because they lack the ability to identify specific disorders. This experiment was conducted to elucidate the symptomology of gazania nutritional disorders and to determine critical nutrient levels of the crop.

Materials and Methods

Single 'Daybreak White' gazania plugs (2.7 x 2.7 x 3.6 cm cell size) were transplanted into 12.7 cm diameter (0.76 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 11 June 2009. In order to get adequate tissue for nitrogen, phosphorus, and iron, a second round of the experiment was conducted. ‘Daybreak White’ gazania seed were sown into a 72-plug (4.0 x 4.0 x 5.8 cm) tray on 13 Oct. 2009. Seedlings were fertigated as needed using 150 $mg \cdot L^{-1}$ N from 13-2-13 Cal-Mag (SQM North America, Atlanta, GA). These plants were installed on 9 Nov. 2009 in the previously described manner. This experiment was conducted in a glass greenhouse in Raleigh, NC at 35°N latitude. Plants were grown at 18˚C day and 15˚C night temperature set points. Treatments started immediately upon transplanting. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2 \cdot 4H_2O$, KH_2PO_4, $MgSO_4 \cdot 7H_2O$, FeDTPA, $MnCl_2 \cdot 4H_2O$, $ZnCl_2$, $CuCl_2 \cdot H_2O$, H_3BO_3, and $Na_2MoO_4 \cdot 2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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/or mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, three pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 dry 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 placing it 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.). The first experiment was terminated on 28 July 2009, and non-symptomatic copper and molybdenum deficient plants were sampled for dry weight and nutrient levels. The second experiment was terminated on 17 Dec. 2009. 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 nine separate harvest dates. Values for percentage difference in plant dry weights are presented if control and treatment tissues were significantly different. Unless otherwise noted, values for tissue concentrations presented were significantly different.

Nitrogen

Nitrogen (N) deficient plants developed an overall yellowish coloration. Dry weight for control plants was not available so a comparison could not be made. Concentrations in control and N deficient plants were 5.68% and 2.31% N, respectively. At the end of the experiment N deficient plants were visually smaller than the control.

Phosphorus

Lower and upper leaves on plants grown under phosphorus (P) deficient conditions were darker green than the control. Plants grown under P deficient conditions exhibited less growth, and dry weights were 43% smaller than the control. Tissue P concentration of plants grown with complete solution was 1.42% and 0.12% for P deficient plants. Lower leaves then developed necrosis which moved inward on the leaf from the tip. In the advance stage most of the lower and middle leaves had become fully necrotic.

Potassium

Potassium (K) deficiency began with lower leaves exhibiting circular, gray-green lesions. K deficient plants were also 31% smaller than control plants. K concentration in the control plants was 8.47% while K concentration in K deficient plants was 0.33%. These lesions then turned a tan-brown coloration. The lesions increased in size until the entire leaf became necrotic. Some lower leaf interveinal chlorosis was also observed. As new foliage grew, it developed the same symptomology exhibited initially by the lower leaves.

Calcium

Browning on the tips of lobed young leaves and the young flower bud sepals were the initial symptoms of calcium (Ca) deficiency. No significant difference was observed between control and treatment dry weight. Control plant Ca tissue concentration was

1.58%. Plants grown without Ca contained 0.40% Ca. As the disorder progressed the young foliage exhibited less development; the young leaves also had browning present on the tips. Some of the flower buds aborted. The older leaves exhibited brown discoloration and necrosis on the leaves. Most of the leaves then became fully necrotic, and the plant died.

Magnesium

Plants grown under magnesium (Mg) deficient conditions developed an overall greenish-yellow coloration; some of the lower and middle leaves had a spotty yellow coloration. Control and Mg deficient dry weights were not significantly different. Control and Mg deficient tissue concentrations were 0.46% and 0.05%, respectively. The overall yellowing then intensified in leaves to a stronger yellow coloration or interveinal chlorosis. Affected leaves then began to exhibit tip browning.

Sulfur

Young leaves of sulfur (S) deficient plants developed a yellow-green coloration. Control S concentration was 0.44% and deficient plants contained a concentration of 0.25% S. This yellow coloration then intensified on the upper leaves. Young flower buds were a vibrant yellow color. In the late stage most of the leaves on the plant had turned a vibrant yellow coloration.

Boron

Boron (B) deficiency manifested with the plants exhibiting poor height development. A significant difference did not occur between control and B deficient dry weights. Control and B deficient tissue had B concentrations of 67.1 $mg \cdot kg^{-1}$ and 13.4 $mg \cdot kg^{-1}$, respectively.

Symptoms of B toxicity initially manifested on lower leaves. The margins had yellowing or sunken greenish-tan discolored areas. The difference between control and B toxic tissue dry weights were not significantly different. Tissue concentration of B toxic tissue was 238.6 $mg \cdot kg^{-1}$ while the control had a lower value of 42.4 $mg \cdot kg^{-1}$. As the disorder progressed the marginal yellowing advanced to marginal necrosis. The sunken discolored areas and yellow margins then appeared on younger leaves.

Iron

Plants grown under iron (Fe) deficiency exhibited yellowing on the lower leaves. Fe deficient plants were 17% smaller than the control plants. Fe tissue concentration in the control plants was 62.6 $mg \cdot kg^{-1}$ and 35.2 $mg \cdot kg^{-1}$ in the Fe deficient plants.

Manganese

Middle and upper leaves on plants grown under manganese (Mn) deficiency developed a greenish-yellow coloration. Closer inspection revealed that the yellowing appearance came from interveinal chlorosis developing between the small veins in the leaf.

A significant difference did not occur between the control and Mn tissue dry weights. Control Mn tissue concentration was 126.9 mg·kg^{-1} while Mn deficient tissue concentration was 7.9 mg·kg^{-1}. The interveinal chlorosis coloration then intensified and became more pronounced.

Zinc

Young leaves displayed a greenish-yellow coloration and were poorly elongated on plants grown under zinc (Zn) deficient conditions. Dry weights for the control and Zn deficient plants were not significantly different. The control plants had 15.6 mg·kg^{-1} Zn while the Zn deficient plants had a value of 7.1 mg·kg^{-1}. As the disorder progressed the leaves began to develop a greenish-white haze on the leaf surface. Lower leaves also began to develop necrotic spots.

Asymptomatic

After 7 weeks of growth when the plants had reached full bloom, plants grown under copper (Cu) and molybdenum (Mo) deficient conditions exhibited no visual symptoms. The experiment was terminated, and these asymptomatic plants were sampled and analyzed for dry mass and tissue concentration to determine if non-visual differences were evident.

Plants grown under Cu deficient conditions did not have a significantly different dry weight when compared to the control. Control tissue Cu concentration was 9.5 mg·kg^{-}

^{1}, and Cu deficient tissue concentration was 5.7 $mg{\cdot}kg^{-1}$. Although symptoms may not manifest with Cu deficiency, having the tissue concentrations available to greenhouse growers will be helpful when trying to determine if Cu is limited.

A significant difference in dry weights did not occur for control plants and those grown under Mo deficient conditions. Detectability of Mo was problematic with the gazania samples. Of the three control samples, one had Mo levels below 1 $mg{\cdot}kg^{-1}$, and of the three Mo deficient samples, two had Mo levels below 1 $mg{\cdot}kg^{-1}$. Although not a complete replication, the mean Mo concentration for the control samples was 1.3 $mg{\cdot}kg^{-1}$, and the single Mo deficient sample Mo concentration was 0.3 $mg{\cdot}kg^{-1}$. These concentrations provide a general idea to the target tissue Mo levels.

Conclusions

Deficiency symptoms of N, S, and Ca were first to manifest. Should nutritional disorders arise growers should first consider these three problems when trying to determine the possible cause. It was atypical for gazania plants to not exhibit advanced B and Fe deficiency symptoms because typically they are readily induced (Barnes, 2010). It is important to note that Cu and Mo levels can be extremely low in gazania and the plants be asymptomatic. Because some Mo samples were below the detectable limit in crops fed with a balanced fertilizer, future experiments that increase the number of plants grown under Mo deficient conditions would be able to better elucidate the optimum and deficient levels of Mo.

When grown under the described nutrient deficient and toxic conditions, gazania exhibited the described symptoms. The symptomology descriptions and tissue samples that were taken during this experiment will allow growers to be able to recognize potential nutrient problems before they become too advanced and result in crop failure.

Acknowledgements

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative, and we would like to thank PanAmerican Seed for gazania seed.

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Table 8.1 *Gazania rigens* ‘Daybreak White’ plant dry weight and 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
							Dry Weight (g)						
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	—[1]	1.40	1.05	0.88	1.24	0.45	4.55	2.26	4.89	1.40	2.26	4.89	2.26
Treatment	0.43	0.80	0.72	0.90	1.40	0.48	4.26	1.86	6.64	1.16	2.28	4.10	1.63
p-value[2]	NS	***	*	NS	NS	NS	NS	NS	NS	**	NS	NS	NS

	Tissue Nutrient Concentration (%)						Tissue Nutrient Concentration ($mg \cdot kg^{-1}$)						
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	5.68	1.42	8.47	1.58	0.46	0.44	67.1	42.4	9.5	62.6	126.9	1.3[3]	15.6
Treatment	2.31	0.12	0.33	0.40	0.05	0.25	13.4	238.6	5.7	35.2	7.9	0.3[4]	7.1
p-value[2]	***	***	***	***	***	**	***	*	**	*	**	NS	**

[1] Complete control dry weight was unavailable for N.

[2] *, **, or *** indicates statistically significant differences between sample means based on *F* test at $P \leq 0.05$, $P \leq 0.01$, or $P \leq 0.001$, respectively. NS (not significant) indicates the F test difference between sample means was $P > 0.05$.

[3] Concentration averaged between two samples.

[4] Concentration listed is from a single sample.

Chapter 9

Characterization of Nutrient Disorders of *Gomphrena globosa* 'Las Vegas Purple'

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Gomphrena globosa* 'Las Vegas Purple'

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Keywords: Globe amaranth, Calcium, Micronutrients, Nitrogen, Phosphorus, Potassium, Sulfur

Abstract

***Gomphrena globosa* 'Las Vegas Purple' plants were grown in silica sand culture to induce and photograph symptoms of nutritional disorders. 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

Gomphrena (*Gomphrena globosa*, Amaranthaceae family) is an annual that is grown for use as a bedding plant (Dole and Wilkins, 2005a) and a cut flower (Dole and Wilkins, 2005b). An understanding of potential nutrient disorders that could occur with gomphrena are important for growers. However, nutrient disorders have not been described for the crop, and critical tissue values have not been published. Therefore, this study was conducted to provide growers with information on gomphrena nutrient disorders and critical nutrient levels.

Materials and Methods

'Las Vegas Purple' gomphrena seed was sown into plug trays (4.0 x 4.0 x 5.8 cm cell size) containing Fafard 4P on 27 Aug. 2009. Seedlings were fertigated as needed using 150 $mg \cdot L^{-1}$ N from 13-2-13 Cal-Mag (SQM North America, Atlanta, GA). Single plugs were transplanted into 12.7 cm diameter (0.76 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 Sept. 2009. This experiment was conducted in a glass greenhouse in Raleigh, NC at 35°N latitude. Plants were grown at 18˚C day and 15˚C night temperature set points. Treatments started immediately upon transplanting. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2{\cdot}4H_2O$, KH_2PO_4, $MgSO_4{\cdot}7H_2O$, FeDTPA, $MnCl_2{\cdot}4H_2O$, $ZnCl_2$, $CuCl_2{\cdot}H_2O$, H_3BO_3, and $Na_2MoO_4{\cdot}2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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/or mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, three pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 dry 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 placing it 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.). The experiment was terminated on 20 Oct. 2009, and non-symptomatic plants of iron and molybdenum 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 eight separate harvest dates. Values for percentage difference in plant dry weights are presented if control and treatment tissues were significantly different. Unless otherwise noted, values for tissue concentrations presented were significantly different.

Nitrogen

Leaves on nitrogen (N) deficient plants developed a yellowish-green coloration. Plant dry weight of N deficient plants was 49% less than the controls. Control plants had a concentration of 5.28% N while N deficient plants had a concentration of 2.68% N. As the disorder progressed the coloration intensified, especially in lower leaves where they became yellow. Magenta coloration also appeared in the lower leaf tips, margins, and veins. Some of the lower leaf tips then became necrotic.

Phosphorus

Phosphorus (P) deficient plants were a darker green coloration than the control. Dry weights of plants grown under P deficient conditions were 49% less than the control. Tissue concentration of the control was 1.99% P while the tissue concentration of the P deficient plants was 0.13% P. As the disorder progressed lower leaves began to exhibit a magenta coloration. Tips of lower leaves then became necrotic, and the necrosis advanced inward on leaves until they became fully necrotic. Overtime, necrosis advanced up the plant.

Potassium

Potassium (K) deficiency began with the tips of lower leaves exhibiting a whitish-green coloration. Plants grown under K deficient conditions had 37% less dry weight than the controls. K concentration in the control was 7.42% and in the K deficient plant was 1.59%. As the disorder progressed more of the coloration appeared on the leaf tip. Tan necrotic spots began to appear, and these increased in size. Spots of varying sizes also began to appear on the inside of leaves. The smaller spots were a red circle that surrounded a white center. Spotting also manifested in middle and upper leaves. In the advance stage of the disorder, leaves became fully necrotic.

Calcium

Calcium (Ca) deficient plants appeared smaller, and dry weights were 52% that of the controls. Initially lower leaves had some spots present on them, but it did not appear to differ from the number of spots present on the control. Spots were a red ring that surrounded a white center. Concentrations of Ca in the control and Ca deficient plants were 2.66% and 0.32%, respectively. As the disorder progressed the spots increased in number, and they began to appear in the middle and upper leaves. The spotting on lower leaves increased so much that the leaves appeared to have an overall reddish-magenta coloration.

Magnesium

Plants grown under magnesium (Mg) deficiency became a yellow-green color. Dry weights of plants grown under Mg deficient conditions was 31% that of the control. Control Mg tissue concentration was 2.06%, and Mg deficient tissue concentration was 0.17%. As the disorder progressed leaf tips became necrotic. The initial yellow-green coloration intensified to yellow. The coloration was isolated primarily to the interveinal region of the leaf. White necrotic spots appeared near the leaf tip. In the advanced stage of the disorder interveinal chlorotic leaves also exhibited red coloration on the leaf margin, and the white necrotic spots increased in size until the entire leaf became necrotic.

Sulfur

Upper leaves and axillary leaves on plants grown under sulfur (S) deficiency developed a greenish-yellow coloration. A significant difference did not occur between control and S deficient dry weights. Control plants had a S concentration of 0.30% while S deficient plants had a S concentration of 0.13%. All the leaves on S deficient plants then developed a yellow-greenish coloration. Whitish-tan necrotic spots then manifested on the lower leaf margins. These spots then advanced inward.

Boron

White spotting on young leaves near the apical bud and in the axillary shoots was the initial symptom for boron (B) deficiency. Concentrations of B in the control and B deficient plants were 41.5 $mg \cdot kg^{-1}$ and 10.2 $mg \cdot kg^{-1}$, respectively. As the disorder progressed the affected leaves developed light tan necrosis which advanced inward on the leaf from the tip. Some flower inflorescences began to develop, but they then aborted. In the advance stage new shoots began to grow.

Symptoms of B toxicity manifested in the lower leaves. The tips became necrotic and exhibited a white coloration. Control B concentration was 36.8 $mg \cdot kg^{-1}$, and B toxic tissue concentration was 165.6 $mg \cdot kg^{-1}$ B. The spots then increased inward in size, and a brownish coloration developed on some leaves between the necrotic and healthy tissue. Symptoms progressed upward on the plant and eventually manifested in the upper leaves.

Copper

Copper (Cu) deficiency manifested as lower leaf spotting. The spots were red circular areas that had a white center. Dry weight of Cu deficient plants was 43% less than the control. Tissue concentrations were significantly different at $P = 0.07$. Control Cu concentration was 12.2 $mg \cdot kg^{-1}$, and Cu deficient Cu concentration was 7.7 $mg \cdot kg^{-1}$. The number of spots then increased. Tannish spots then appeared on affected leaves, and increased in size until the entire leaf became necrotic.

Manganese

Manganese (Mn) deficient plants exhibited a whitish-green or yellow-green coloration on the margin near the tips of lower leaves. Concentrations of control and Mn deficient plants were 115.3 $mg \cdot kg^{-1}$ and 22.9 $mg \cdot kg^{-1}$, respectively. As the disorder progressed red circular spots with white centers occurred on lower leaves.

Zinc

Lower leaf spotting was the initial symptom of zinc (Zn) deficiency. Spots were small and were a reddish-brown coloration while others had white centers. Plant dry weight of Zn deficient plants was 35% less than the control. Tissue concentrations were not significantly different for control and Zn deficient plants. Those concentrations for the control and Zn deficient tissue were 118.7 $mg \cdot kg^{-1}$ and 115.2 $mg \cdot kg^{-1}$, respectively. As the disorder progressed larger red or yellow colored spots appeared on upper leaves. Larger white spots then appeared on lower leaves.

Asymptomatic

After 8 weeks of growth when the plants had reached full bloom, plants grown under iron (Fe) and molybdenum (Mo) deficient conditions exhibited no visual symptoms. The experiment was terminated, and these asymptomatic plants were sampled and analyzed for dry mass and tissue concentration to determine if non-visual differences were evident.

Dry weights of the control and Fe deficient plants were not significantly different. Tissue concentrations were significantly different, but Fe deficient concentration was over four times greater than the control. Control tissue Fe concentration was 108.9 $mg \cdot kg^{-1}$, and Fe deficient tissue concentration was 493.5 $mg \cdot kg^{-1}$.

Dry weights and tissue concentrations were not significantly different between control plants and Mo deficient plants. Only one control had Mo reported, and that concentration was 2.6 $mg \cdot kg^{-1}$. Mo deficient tissue concentration was 1.7 $mg \cdot kg^{-1}$. While the values were not significantly different, they will provide growers with an idea of Mo concentration within the plant.

Conclusions

Symptoms of N, P, and S deficiencies and B toxicity manifested early. These disorders may be a more likely problem encountered by a grower.

Some anomalies were observed in gomphrena. Cu deficiency manifested in the crop. Cu deficiency is typically difficult to induce (Barnes, 2010), but it appeared as spotting on the lower leaves and advanced to lower leaf necrosis. Also atypical was the high level of Fe in the Fe deficient samples. Reviewing the original data revealed that the control Fe concentrations were consistent through the eight harvests. Values ranged from 63.9-187.3 $mg \cdot kg^{-1}$ Fe. However, higher than normal values for Fe were observed in Cu deficiency with an average of 1318.3 $mg \cdot kg^{-1}$ Fe and in Zn deficiency 994.5 $mg \cdot kg^{-1}$ Fe.

While symptoms have not been previously described for gomphrena nutritional disorders, this study illustrated that they do occur. Also, the critical tissue concentrations reported will help growers diagnosis nutritional disorders that arise in their greenhouses when producing this crop.

Acknowledgements

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative, and we would like to thank Benary for gomphrena seed.

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Table 9.1 *Gomphrena globosa* 'Las Vegas Purple' plant dry weight and 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**
	Dry Weight (g)												
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	0.43	0.76	10.53	4.83	4.83	1.05	2.41	1.05	2.41	10.64	10.53	10.64	5.54
Treatment	0.22	0.39	6.63	2.34	3.33	0.87	1.96	1.02	1.37	10.19	7.33	11.84	3.58
p-value[1]	***	**	***	**	*	NS	NS	NS	*	NS	*	NS	*

	Tissue Nutrient Concentration (%)						**Tissue Nutrient Concentration ($mg\cdot kg^{-1}$)**						
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	5.28	1.99	7.42	2.66	2.06	0.30	41.5	36.8	12.2	108.9	115.3	2.6[2]	118.7
Treatment	2.68	0.13	1.59	0.32	0.17	0.13	10.2	165.6	7.7	493.5	22.9	1.7	115.2
p-value[1]	***	***	***	***	***	***	***	***	NS	**	***	NS	NS

[1] *, **, or *** indicates statistically significant differences between sample means based on *F* test at $P \leq 0.05$, $P \leq 0.01$, or $P \leq 0.001$, respectively. NS (not significant) indicates the F test difference between sample means was $P > 0.05$.

[2] Only one sample used in the number presented.

Chapter 10

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

(In the format appropriate for submission to Acta Horticulturae)

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

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Keywords: Calcium, Magnesium, Micronutrients, Nitrogen, Phosphorus, Potassium, Sulfur

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. Boron toxicity was also induced by increasing the element 10× higher than the complete nutrient formula. Reagent grade chemicals and deionized water of 18-mega ohms purity were used to formulate treatment solutions. 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 (*Pericallis ×hybrida*, 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 x 3.4 x 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 Oct. 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 temperature set points. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2{\cdot}4H_2O$, KH_2PO_4, $MgSO_4{\cdot}7H_2O$, FeDTPA, $MnCl_2{\cdot}4H_2O$, $ZnCl_2$, $CuCl_2{\cdot}H_2O$, H_3BO_3, and $Na_2MoO_4{\cdot}2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. The solution drained out from the bottom of the pot and was captured 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 pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 placing it 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 Dec., 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. At initial sampling, the plants were 36% 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. 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. 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. 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 and marginal interveinal chlorosis on the most recently mature leaves. At harvest plants were 22% 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. 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. Upper leaves also exhibited slight interveinal chlorosis. At sampling, the roots were inspected and Ca deficient plants had roots that were a burgundyreddish 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. 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. 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. 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. 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. 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. 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, 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. At initial sampling, the plants were 27% larger than the control. Tissue values for B toxicity plants and control were 396.8 $mg{\cdot}kg^{-1}$ 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.

Iron

Iron (Fe) deficient plants exhibited interveinal chlorosis of the recently matured leaves. 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 52.5 $mg \cdot kg^{-1}$ Fe. Chlorosis then spread over the entire plant, even into the inflorescence leaves. 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 27% 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% smaller than the control.

Plants grown under Mo deficient conditions were 39% 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 50% 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

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative.

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Table 10.1 *Pericallis ×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**[1]	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**[1]	**Mn**	**Mo**	**Zn**
Complete Control	0.74 A[2]	0.70 a	0.70 b	3.77 a	2.54 a	0.70 a	1.50 a	0.70 b	8.90 a	0.74 a	8.90 a	8.90 A	8.90 A
Treatment	0.48 B	0.51 a	0.89 a	3.50 a	2.84 a	0.66 a	1.89 a	0.89 a	6.52 b	0.77 a	6.47 b	5.46 B	4.49 B

[1] Two plants used per sample.

[2] 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 10.2 *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**[1]	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**[1]	**Mn**	**Mo**	**Zn**
Complete Control	6.96 A[2]	1.02 A	8.68 A	1.87 A	0.64 A	0.52 A	52.1 A	76.4 B	16.9 A	85.1 a	116.4 A	0.2 a	21.6 a
Treatment	1.59 B	0.14 B	1.10 B	0.37 B	0.15 B	0.18 B	12.1 B	396.8 A	6.3 B	64.3 a	14.0 B	—[3]	15.7 b

[1] Two plants used per sample.

[2] 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 $P \leq 0.01$.

[3] Tissue concentration was below the detectable limit of 1.0 $mg \cdot kg^{-1}$.

Chapter 11

Characterization of Nutrient Disorders of *Primula acaulis* 'Danova Rose'

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Primula acaulis* 'Danova Rose'

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Keywords: Primrose, Calcium, Magnesium, Micronutrients, Nitrogen, Phosphorus, Potassium,
Sulfur

Abstract

***Primula acaulis* 'Danova Rose' 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 was drained from the bottom of the pot and 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

Primula (*Primula acaulis*, Primulaceae family) (Dole and Wilkins, 2005) is a floriculture crop grown for use as a cool season colorful bedding plant or as a pot plant in the home (Linwick, 1996). It is not a heavy feeder (Erwin, 1999; Armitage et al., 1994) and requires a low to moderate level of fertilization (Linwick, 1996; Hartnett, 1993).

Hartnett (1993) recommended during Stage 2 of germination, the period when the cotyledons emerge and first true leaves develop, the fertility regimen should include 60 $mg \cdot L^{-1}$ nitrogen (N) from potassium nitrate and calcium nitrate, 74 $mg \cdot L^{-1}$ potassium (K), balanced micronutrients, and a pH of 6.0. Linwick (1996) suggested three weeks after sowing seedlings should be fertilized with 50 to 75 $mg \cdot L^{-1}$ N from a 20-10-20 peat-lite mix or other fertilizer which has an electrical conductivity (EC) of 1.25 mS/cm and a pH of 5.5. Armitage et al. (1994) recommended a lower rate of 25 to 50 $mg \cdot L^{-1}$ KNO_3 during Stage 2 and 3. PanAmerican Seed (2009) suggests during Stage 2 seedlings should be fed with 100 $mg \cdot L^{-1}$ of 14-0-14, and during Stage 3 one should alternate between 200 $mg \cdot L^{-1}$ 14-0-14 and 20-10-20. Prior to transplanting, seedlings should be fed 200 $mg \cdot L^{-1}$ N (Dole and Wilkins, 2005; Erwin, 1999; Hartnett, 1993). The potting on mix should be high in organic matter, but starter fertilizers should be omitted to provide more control over fertility requirements (Hartnett, 1993). After transplanting, 90 $mg \cdot L^{-1}$ N with a 1 to 1 ratio of N in the nitrate form and potash fertilizer is recommended (Hartnett, 1993). Dole and Wilkins (2005) and Erwin (1999) suggested that 90 to 100 $mg \cdot L^{-1}$ N and K be used. Armitage et al. (1994) recommended a higher rate of 100 to 125 $mg \cdot L^{-1}$ N for production, and Linwick (1996) suggested a constant liquid feed rate of 100 to 150 $mg \cdot L^{-1}$ N. After the plants have developed a rosette of 6 to 10 leaves the rate can be increased to 200 $mg \cdot L^{-1}$ N and 440 $mg \cdot L^{-1}$ K to enhance bloom production (Hartnett, 1993; Linwick, 1996). In the cooler months, N should originate from calcium nitrate and potassium nitrate (Hartnett, 1993) instead of ammonium nitrate (Linwick, 1996; Erwin, 1999). Cooler temperatures slow the action of microorganisms converting ammonium into useable nitrate. The lack of conversion results in a build up of ammonium (Holcomb, 1983) which can be toxic (Erwin, 1999). In the winter the fertilizer rate should be dropped to 75 to 100 $mg \cdot L^{-1}$ N with each irrigation (Armitage et al., 1994). PanAmerican (2009) suggests for the cold treatment lowering the 20-10-20 rate to 15-0-15 at 50 ppm. The EC of the media should be below 1.2 mS/cm (PanAmerican, 2009).

Some nutritional problems have been reported. Excess N encouraged leaf growth (Hartnett, 1993; Dole and Wilkins, 2005) and can cause root problems which results in foliar problems. Ammonium toxicity occurred during winter and causes marginal yellowing on lower leaves. This symptom can be corrected by leaching the substrate and changing to nitrate-based fertilizers (Erwin, 1999). Manganese (Mn) at toxic levels resulted in necrosis on lower leaf margins (Hartnett, 1993). Low fertility can result in small plants and premature bud set (PanAmerican, 2009). N deficiency manifested itself with chlorotic leaves (Holcomb, 1983; PanAmerican, 2009), discolored and blackening veins, pale young growth, and older growth developing marginal necrosis (Holcomb, 1983). Phosphorus (P) deficiency is reported as being a problem (Hartnett, 1993). Symptoms include bronze colored lower leaves, inward leaf curl with necrotic tips, and brown coloration in the veins (Holcomb, 1983). K deficiency appears as chlorotic lower leaves that eventually become necrotic, a reduction in young leaf expansion, and curled or rippled leaf margins (Holcomb, 1983). Calcium (Ca) deficiency is exhibited as either chlorosis, necrosis, or edge burn of upper leaves (Erwin, 1999). Pale green leaves have also been reported with Ca deficiency (Holcomb, 1983). Applying calcium nitrate will correct the problem (Erwin, 1999). Magnesium (Mg) deficiency manifested itself in older leaves with interveinal chlorosis (Holcomb, 1983; PanAmerican, 2009). This symptom progresses to necrosis of the leaf tips and margins. Younger leaves exhibit curling and backward arching (Holcomb, 1983). The most severe cases of Mg deficiency can be manifested as smaller plants, leaf canker, and burned margins (Dole and Wilkins, 2005). To help chlorosis problems and enhance growth, magnesium sulfate can be used at a rate of 3.7 $g \cdot m^3$. Leaf distortion of young leaves can be caused by boron deficiency and can be corrected by applying 3.77 g/100 L Borax or 1.87 g/100 L Solubor (Erwin, 1999). Iron (Fe) chlorosis can occur when pH is above 6.5 (Hartnett, 1993; PanAmerican, 2009), when EC levels are high (Hartnett, 1993), or when insufficient Fe is provided (Erwin, 1999). Symptoms of Fe deficiency appear in young leaves (Erwin, 1999) as interveinal chlorosis (Holcomb, 1983; PanAmerican, 2009) which can develop to a white coloration and necrosis (Holcomb, 1983). Older leaves can also exhibit marginal chlorosis which progresses inward toward the midrib (Holcomb, 1983). This deficiency can be corrected by lowering pH using an acidic fertilizer that provides iron (Erwin, 1999) or applying iron chelate (Hartnett, 1993). Insufficient Mn caused bleaching in new growth (Hartnett, 1993).

While some information is provided about nutritional disorders of primula, the symptomology for nutritional problems of this crop is not complete. Thus, the goals of this study were to provide visual and tissue diagnostic values for primula so that growers will be able to accurately diagnosis nutritional problems on site.

Materials and Methods

Single 'Danova Rose' primula plugs (3.4 x 3.4 x 5.1 cm cell size) were transplanted into 12.7 cm diameter (0.76 mL) 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 Oct. 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 temperature set points. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2 \cdot 4H_2O$, KH_2PO_4, $MgSO_4 \cdot 7H_2O$, FeDTPA, $MnCl_2 \cdot 4H_2O$, $ZnCl_2$, $CuCl_2 \cdot H_2O$, H_3BO_3, and $Na_2MoO_4 \cdot 2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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 pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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, it was ground in a Foss Tecator Cyclotec™ 1093 sample mill (Analytical Instruments, LLC, Golden Valley, MN) to pass a $\leq$ 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 placing it 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 calcium 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 Dec., and non-symptomatic plants of the copper, manganese, molybdenum, and zinc plants 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. Values for percentage difference in plant weights and tissue concentrations are presented. Comparisons of values obtained from this experiment were compared with optimum values published by Holcomb (1983) for *Primula* x *polyantha*.

Nitrogen

An overall pale green coloration was the initial symptom of nitrogen (N) deficiency. Dry weight was significantly different with N deficient plants being 25% smaller at harvest than the controls. The control N tissue concentration was 3.91% while the N deficient tissue was 1.80%. The control value was within the optimum range of 2.50 to 3.30%. The deficient tissue value was lower than the optimum range and greater than the deficient value of 1.51% (Holcomb, 1983). Symptoms progressed with the outer margin developing a stronger yellow coloration on the middle and lower leaves. The entire leaf then turned a lemon-yellow coloration. This symptom was followed by the leaf turning tan and becoming dry. Our observations were similar to previous reports (Holcomb; 1983; PanAmerican, 2009) except the vein blackening and discoloration did not occur.

Phosphorus

The tips of lower leaves were necrotic for plants grown in phosphorus (P) deficient conditions. Yellow coloration surrounded this necrotic area. A significant difference occurred between the control and P deficient tissue. At sampling time P deficient plants were 45% smaller than the controls. Values for P concentrations of control and deficient tissue were 0.38% and 0.12%, respectively. The optimum range for P is 0.36% to 0.81%.

Thus, the control falls within that range; however, the P deficient tissue concentration was lower than the critical value of 0.22% (Holcomb, 1983). Symptoms progressed with lower leaves exhibiting interveinal chlorosis. Marginal browning continued to move inward on the leaf, and the leaves began to curl inward. Some lower leaves became completely brown. Other than the bronze colored leaves and brown coloration in the veins, our observations match those reported earlier (Holcomb; 1983; Hartnett, 1993).

Potassium

Symptoms of potassium (K) deficiency began with marginal yellowing on the tips of both young and mature leaves. At initial sampling a significant difference did not occur between the dry weights of the K deficient tissue and the control tissue. Control plants had a control tissue concentration value of 6.23% for K while K deficient tissue had a value of 3.13%. The optimum range is 2.11 to 4.20% K (Holcomb, 1983). Thus, the control value falls above this range while the deficient value falls within this range. Within the yellow coloration of mature leaves necrotic spots formed on the margins. Some lower leaves became necrotic. At the termination of the experiment, plants were visually smaller than the controls. The symptoms reported by Holcomb (1983) were similar to those we observed.

Calcium

Initially, calcium (Ca) deficient plants developed necrotic spots on the lower leaf margins. There was no significant difference in control and Ca deficient dry weights. Values for the control and Ca deficient tissue were 0.65% and 0.51%, respectively. The control tissue value is within the optimum range of 0.62 to 1.01% while the Ca deficient tissue value was lower than this range. However, it is higher than the reported deficient level of 0.18% (Holcomb, 1983). Symptoms advanced with the lower leaves developing black veins, yellowing in the leaf margin, and lower leaf curling inward. The yellow coloration moved inward toward the midrib, and brown necrotic spots formed between secondary veins. Leaves then either retained a residual green coloration near the midrib, or they turned brown and became dry. New leaves then developed necrotic margins. At the termination of the experiment, plants were visually smaller than the controls. Our observations were similar to Holcomb's (1983) and Erwin's (1999) observations; however, with our study additional symptoms were observed.

Magnesium

Magnesium (Mg) deficient plants exhibited a thin band of tan coloration on the very outside margin of the lowest leaves while larger mature leaves exhibited more of a yellow color in the margin. There was no significant difference in control and Mg deficient dry weights. Control Mg tissue concentration was 0.31% while the deficient tissue had a value of 0.17%. The optimum range of the values is 0.20 to 0.42%. Thus, the control value falls within this range. The Mg deficient tissue is lower than the range but higher than the deficient value of 0.09% (Holcomb, 1983). As symptoms progressed this band became wider, and bleaching began to occur within this marginal area. The margin became dessicated as tan coloration progressed inward toward the midrib almost completely filling the interveinal areas. Younger leaves exhibited similar symptoms but to a lesser degree and remained mostly green. Plants were visually smaller when the experiment was terminated. Our observations match those described by Holcomb (1983) and PanAmerican (2009), but leaf cankers did not occur (Dole and Wilkins, 2005).

Sulfur

Sulfur (S) deficient plants initially developed a greenish-yellow coloration in the leaves. In older leaves it varied where it appeared either near the tip or near the petiole. In younger leaves yellowing was isolated to the margin of the leaf. Plant dry weights were not significantly different. S tissue concentrations for control and S deficient samples were 0.54% and 0.12%. No optimal range or deficiency values have been reported for S. Symptoms progressed with the entire plant developing the greenish-yellow coloration. Necrosis then occurred on the lower leaf margins. Necrotic areas were a tan color. This symptom advanced until the entire leaf became necrotic. When the experiment ended, it was apparent that the deficient plants were visually smaller than the controls.

Boron

Boron (B) deficient plants exhibited a lack of elongation of young foliage and yellowing on the mature leaf margins. A significant difference was not observed in the dry weights. The control tissue value was 35.9 $mg \cdot kg^{-1}$ B which was similar to the reported optimum level of 35.90 $mg \cdot kg^{-1}$ (Holcomb, 1983). Deficient plants contained 4.5 $mg \cdot kg^{-1}$ B. Symptoms progressed with browning becoming prevalent in the young leaves and yellow coloration progressing inward toward the midrib. The bud then aborted and developed a black coloration, and the leaf margins became crispy with black coloration. Deficient plants were visually smaller at the end of the experiment. The initial distorted growth matches the symptoms described by Erwin (1999); however, symptoms progressed further in our study.

B toxicity plants initially exhibited tan coloration along the leaf margin of the lower leaves. Dry weights were not significantly different. Tissue B concentration for the control plants was 44.5 $mg \cdot kg^{-1}$ while it was 373.7 $mg \cdot kg^{-1}$ for the B toxicity plants. Both values were above the reported optimum value of 35.9 $mg \cdot kg^{-1}$ (Holcomb, 1983). Symptoms progressed with the tan coloration expanding inward, and the border area between the necrotic and healthy green tissue eventually developed a yellow band.

Iron

Iron (Fe) deficient plants exhibited yellowing in both young and old leaves. Yellowing was confined primarily to the margin and outer veinal areas. No significant difference was observed with the dry weights and tissue concentrations. Control and Fe deficient tissue had values of 148.1 $mg \cdot kg^{-1}$ and 95.9 $mg \cdot kg^{-1}$. Both values were within the optimum range of 78 to 155 $mg \cdot kg^{-1}$. One of the Fe deficient samples had a high value which resulted in a high standard deviation which was why no significant difference was observed. The mean value of the other two lowest samples was 69.2 $mg \cdot kg^{-1}$. This value is lower than the optimum range but higher than the reported deficient level of 60 $mg \cdot kg^{-1}$ Fe (Holcomb, 1983). No other symptoms were observed. In this experiment the plants manifested a yellow coloration instead of true interveinal chlorosis described by previous work (Holcomb, 1983; Hartnett; 1993; PanAmerican, 2009). Also, the plants did not progress to the white coloration and necrosis described by Holcomb (1983).

Zinc

Zinc (Zn) deficient plants initially exhibited tan or brown coloration on the leaf margins in isolated spots. A significant difference was not found between the dry weights for Zn. Tissue concentrations were significantly different at $P = 0.06$. Control tissue had a Zn concentration of 23.6 $mg \cdot kg^{-1}$ while the deficient plants contained 13.7 $mg \cdot kg^{-1}$ Zn. Both of these values are lower than the reported optimal concentration of 38.55 $mg \cdot kg^{-1}$ (Holcomb, 1983). The coloration then began to expand inward as affected areas turned crispy.

Asymptomatic

The experiment was terminated when the plants reached full bloom after 8 weeks of growth. No visual symptoms were apparent with those plants grown under copper (Cu), manganese (Mn), and molybdenum (Mo) deficient conditions. To determine if non-visual differences due to the deficiency occurred, plants were sampled and analyzed for dry mass and tissue concentration. Of these three elements, all were significantly different for tissue concentrations, but no significant difference was observed with plant dry mass.

Control tissue Cu concentration was 5.0 $mg \cdot kg^{-1}$. The value for Cu deficient tissue was 3.8 $mg \cdot kg^{-1}$. Both of these values are lower than the optimum reported value of 6.45 $mg \cdot kg^{-1}$ (Holcomb, 1983).

The tissue concentrations of plants grown under control and Mn deficiency conditions were 39.7 $mg \cdot kg^{-1}$ and 7.0 $mg \cdot kg^{-1}$, respectively. The optimum level for Mn is 49.70 $mg \cdot kg^{-1}$ (Holcomb, 1983). Both values are lower than this number. Hartnett's (1993) observation of new growth bleaching was not observed.

The control tissue concentration for Mo was 1.7 $mg \cdot kg^{-1}$ while Mo deficient tissue had a tissue concentration of 0.2 $mg \cdot kg^{-1}$. No value has been reported for Mo. Therefore, the value of 1.7 $mg \cdot kg^{-1}$ is considered to be an optimum value while 0.2 $mg \cdot kg^{-1}$ is a deficient value. An experiment growing more plants would increase the accuracy of these values.

Although the plants grown in Cu, Mn, and Mo deficient conditions exhibited no symptoms, the tissue concentration values will be of use to growers that need to determine if these plant nutrients are low.

Conclusion

Nutrient deficiencies were induced in *Primula acaulis* 'Danova Rose' which generated the described symptoms. Also, tissue samples taken when initial symptoms were observed provide critical tissue nutrient values for the crop. This data will allow growers to have a better grasp on diagnosing nutritional problems of this crop.

Acknowledgements

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative.

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Table 11.1 *Primula acaulis* 'Danova Rose' 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**[1]	**P**	**K**	**Ca**[1]	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	0.35 a[2]	0.57 A	0.31 a	0.35 a	0.57 a	0.31 a	0.97 a	3.53 a	3.53 a	0.31 a	3.53 a	3.53 a	1.44 a
Treatment	0.26 b	0.32 B	0.24 b	0.24 b	0.55 a	0.24 a	1.09 a	2.93 a	3.37 a	0.3 a	3.23 a	3.81 a	1.24 a

[1] Two plants used per sample.

[2] 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 11.2 *Primula acaulis* 'Danova Rose' tissue nutrient concentration as affected by deficient or toxic nutrient treatments and published optimal range and deficient values.

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[1]**	**P**	**K**	**Ca[1]**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	3.91 A[2]	0.38 A	6.23 A	0.65 A	0.31 A	0.54 A	35.9 A	44.5 B	5.0 a	148.1 a	39.7 A	1.7 A	23.5 a
Treatment	1.80 B	0.12 B	3.13 B	0.51 B	0.17 B	0.12 B	4.5 B	373.7 A	3.8 b	95.9 a	7.0 B	0.2 B	13.7 a
***Primula* x *polyantha* Optimum Range[3]**	2.50 - 3.30	0.36-0.81	2.11 - 4.20	0.62 - 1.01	0.20 - 0.42	-	35.90	35.90	6.45	78 - 155	49.70	-	38.55
***Primula* x *polyantha* Deficient Value[3]**	1.51	0.22	1.56	0.18	0.09	-	-	-	-	60		-	-

[1] Two plants used per sample.

[2] 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 $P \leq 0.01$.

[3] Holcomb, 1983.

Chapter 12

Characterization of Nutrient Disorders of *Salvia farinacea* 'Evolution'

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Salvia farinacea* 'Evolution'

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Keywords: Calcium, Magnesium, Micronutrients, Nitrogen, Phosphorus, Potassium, Sulfur

Abstract

Salvia farinacea **'Evolution' plants were grown in silica sand culture to induce and photograph symptoms of nutritional disorders. 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. Boron toxicity was induced by increasing the element 10× higher than the complete nutrient formula. Reagent grade chemicals and deionized water of 18-mega ohms purity were used to formulate treatment solutions. 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. Out of thirteen treatments, twelved exhibited**

symptomology. Symptoms of N, P, S, Ca, and K deficiencies and B toxicity manifested early; therefore, these disorders may be a more likely problem encountered by a grower.

Introduction

Salvia **(***Salvia farinacea*, Lamiaceae family) is a bedding annual planted by gardeners to provide color during the summer (Armitage, 2001). Since this is a popular crop, growers must understand how to grow the species. One factor they must have a grasp on is the nutritional requirements of the crop, and a lack of knowledge can lead to nutrient disorders.

Few nutrient disorders have been reported for salvia. For *S. splendens* necrotic foliage occurs if electrical conductivity levels are too high (Armitage et al., 1994). If a nutritional problem arises in the greenhouse, growers are at a disadvantage with the lack of nutritional problems that occur with *S. farinacea*. Therefore, this experiment was conducted to determine what nutrient disorder symptoms occur and to elucidate critical nutrient levels for the crop.

Materials and Methods

Single 'Evolution' salvia plugs (2.7 x 2.7 x 3.6 cm cell size) were transplanted into 12.7 cm diameter (0.76 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 11

June 2009. In order to get adequate tissue for nitrogen, phosphorus, and iron, a second round of the experiment was conducted with a 72-plug (4.0 x 4.0 x 5.8 cm) tray being sown with 'Evolution' salvia seed on 13 Oct. 2009. Seedlings were fertigated as needed using 150 $mg \cdot L^{-1}$ N from 13-2-13 Cal-Mag (SQM North America, Atlanta, GA). These plants were installed in the previously described manner on 9 Nov. 2009. This experiment was conducted in a glass greenhouse in Raleigh, NC at 35°N latitude. Plants were grown at 18˚C day and 15˚C night temperatures. Treatments started immediately upon transplanting. An automated, recirculating irrigation system was constructed out of 10.2 cm diameter PVC pipe (Charlotte Plastics, Charlotte, NC). The system consisted of eighteen separate irrigation lines (each 1.82 m long). Each line contained eight openings (12.7 cm diameter) that held the eight pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2 \cdot 4H_2O$, KH_2PO_4, $MgSO_4 \cdot 7H_2O$, FeDTPA, $MnCl_2 \cdot 4H_2O$, $ZnCl_2$, $CuCl_2 \cdot H_2O$, H_3BO_3, and $Na_2MoO_4 \cdot 2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14).

For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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/or mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, three pots containing one symptomatic plant each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 dry 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 $\leq$ 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 placing it 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.). The first experiment was terminated on 28 July 2009, and non-symptomatic molybdenum deficient plants were sampled for dry weight and nutrient levels. The second experiment was terminated on 17 Dec. 2009. 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 ten separate harvest dates. Values for percentage difference in plant dry weights are presented if control and treatment tissues were significantly different. Unless otherwise noted, values for tissue concentrations presented were significantly different. Comparisons of values obtained from this experiment were compared with optimum values published by Mills and Jones (1996) for *S. splendens*, another bedding salvia (Dole and Wilkins, 2005).

Nitrogen

Plants grown under nitrogen (N) deficient conditions developed an overall yellow coloration. N deficient plants were 38% smaller than the control plants. The control tissue N concentration was 5.06% while N deficient tissue concentration was 2.57%. When compared with *S. splendens*, both values were within the sufficiency range presented by Mills and Jones (1996) of 2.38-5.61%. The control was in the upper range while the deficient value was in the lower range. As the disorder progressed the yellow coloration intensified, and the leaf margins developed a purple coloration. Lower leaf tips became necrotic. The necrotic area advanced inward on the leaf. In the advance stage the upper leaves also became necrotic.

Phosphorus

Phosphorus (P) deficiency manifested on lower leaves with brown spots appearing on the outside half margin of the leaf. Plants grown under P deficient conditions were 39% smaller than those that received P. Control plants had a concentration of 0.67% P and P deficient plants a concentration of 0.12% P. The control value was within the sufficiency range of 0.30-1.24% presented by Mills and Jones (1996), but the deficient value was below the lower end of the range. As symptoms progressed affected leaves abscised from the plant. Upper leaves then developed the marginal spotting.

Potassium

Potassium (K) deficiency appeared on lower leaf margins as tan-brown spots. K deficient plants were 38% smaller than the control plants. The K concentrations of control and K deficient plants were 6.07% and 0.47%, respectively. Mills and Jones (1996) presented a sufficiency range of 2.90-5.86% for *S. splendens*. The deficient plant value was below the range, and the control plant value was above the range. Overtime these spots then increased in size. Middle and lower leaves then developed necrotic mottled spotting that ranged in coloration from dark black-brown to light tan-green. The necrosis then covered the entire leaf.

Calcium

Young leaves on plants grown under calcium (Ca) deficient conditions exhibited dark spots on the leaf margin and downward curl. Ca deficient plants were 37% smaller than those plants that received a complete fertilizer regime. Control plants had a Ca concentration of 1.66% while deficient plants had a concentration of 0.40% Ca. The control value was within the optimum range of 1.00-2.50% Ca that Mills and Jones (1996) presented. The deficient value was lower than this range. Grey spots then appeared on the most recently matured leaf. The spots on both upper and middle foliage then increased in size; some leaves became fully necrotic. The apical bud became necrotic. The plants then died.

Magnesium

Plants grown under magnesium (Mg) deficient conditions first exhibited gray-brown spots on the interveinal regions of middle leaves. A significant difference was not observed between the control and Mg deficient dry weights. Differences in Mg tissue concentration were significantly different. Control tissue Mg concentration was 0.66%, and Mg deficient tissue concentration was 0.08%. In the optimum range Mills and Jones (1996) presented of 0.25-0.86%, the control value fell within this range, and the deficient value fell below this range. As the disorder progressed the size of the spots increased in size, and the entire leaf became necrotic. The discoloration then manifested in upper leaves, and a yellow coloration began to appear in lower leaves. In the advance stage most of the middle leaves then became fully necrotic. Yellow lower leaves exhibited some brown discoloration.

Sulfur

Initially, sulfur (S) deficient plants developed an overall greenish-yellowish coloration. S deficient plants were 44% smaller than the control plants. Tissue concentrations were significantly different; control tissue was 0.45% S while the deficient tissue had 0.24% S. Both the control and the deficient plant values obtained from this experiment were lower than the value of 0.73% published by Mills and Jones (1996). The yellowish coloration then intensified in the middle and upper leaves. Middle leaves then began to develop marginal brown necrosis. Upper leaves then began to develop necrosis as the middle leaves became fully necrotic.

Boron

Boron (B) deficiency initially manifested in the young leaves. They appeared longer and narrower than the control, and small brownish specks formed near where the leaf and petiole meet. Dry weights between the two treatments were not significantly different. Control tissue contained 56.0 $mg \cdot kg^{-1}$ B, and B deficient tissue contained 13.0

$mg \cdot kg^{-1}$ B. Mills and Jones (1996) presented that the optimum B range was 25-75 $mg \cdot kg^{-1}$. Control tissue concentration fell within this range, and the B deficient tissue concentration was lower. As the disorder progressed those specks developed into larger dark brown or black spots. Spotting also began to appear in the upper leaf petioles. The young foliage on lower axillary shoots began to manifest the spotting. Lower axillary shoot buds died, and then the apical bud died. In the advance stage lower leaves manifested interveinal chlorosis which progressed to a brown coloration. Middle and upper leaves then manifested these symptoms.

Lower leaves exhibiting brown spots along the margin was the initial symptom of B toxicity. Dry weights of the control and B toxic tissue were not significantly different, but tissue B concentration was. The control tissue B concentration was 46.8 $mg \cdot kg^{-1}$ while the B toxic tissue concentration was 242.1 $mg \cdot kg^{-1}$. The control also fell within the range presented by Mills and Jones (1996), but plants grown under B toxic conditions had a B concentration that was three times higher than the upper range limit. As the disorder progressed, the spots spread along the margin. The marginal spotting moved into the middle and upper leaves on the plant.

Copper

Plants grown under copper (Cu) deficiency exhibited a greenish-white haze coloration that developed on middle and upper leaves. No significant difference was observed in plant dry weights. Control Cu tissue concentration was 5.5 $mg \cdot kg^{-1}$; Cu deficient tissue Cu concentration was 1.1 mg·kg^{-1}. Both values were lower than the sufficiency range of 7-35 mg·kg^{-1} presented by Mills and Jones (1996). As the disorder progressed the size of the haze coloration increased to cover the leaf.

Iron

The development of a greenish-yellowish coloration in the plants was the first symptom of iron (Fe) deficiency. The coloration was not very apparent and would be hard to distinguish without a control present. Control and Fe deficient tissue dry weights were not significantly different. The Fe concentration of the control plants was 72.0 mg·kg^{-1} while Fe deficient plants had a concentration of 55.5 mg·kg^{-1}. Mills and Jones (1996) presented an optimum range of 60-300 mg·kg^{-1} Fe. The control falls within this range, and the deficient value is lower than this range. As the disorder progressed some upper leaves developed a faint interveinal chlorosis.

Manganese

Manganese (Mn) deficient plants initially developed a greenish-yellow coloration. Dry weights from the control and Mn deficient treatments were not significantly different. Control tissue had a Mn concentration of 114.4 mg·kg^{-1} and deficient plants contained 7.6 mg·kg^{-1} Mn. Control tissue was within the range presented by Mills and Jones (1996) of 30-284 mg·kg^{-1}. Mn deficient tissue was lower than this range. The greenish-yellow coloration then intensified to interveinal chlorosis in some leaves. Brown spots then appeared on leaves throughout the plant either along the margin or in the interveinal areas; symptoms were more pronounced on the middle leaves. The spotting then progressed to leaf necrosis.

Zinc

Upper leaves on plants grown under zinc (Zn) deficiency developed a yellowish-green coloration. Differences in Zn tissue concentrations were significantly different at $P = 0.06$. The control tissue Zn concentration was 21.4 $mg \cdot kg^{-1}$ while Zn deficient tissue concentration was 12.1 $mg \cdot kg^{-1}$. Both values are lower than the sufficiency range of 25-115 $mg \cdot kg^{-1}$ published by Mills and Jones (1996). As the disorder progressed the upper leaves began to develop brown necrotic spots. Also, the inflorescence exhibited reduced elongation and development. Axillary shoots lacked full development, too. In the advance stage lower leaves exhibited a yellow coloration.

Asymptomatic

After 7 weeks of growth when the plants had reached full bloom, plants grown under molybdenum (Mo) deficient conditions exhibited no visual symptoms. The experiment was terminated, and these asymptomatic plants were sampled and analyzed for dry mass and tissue concentration to determine if non-visual differences were evident.

A significant difference in dry weights for control plants and those grown under Mo deficient conditions did not occur. One control sample had a Mo concentration of 0.6

mg·kg^{-1} while Mo deficient plants Mo levels were all below 1 mg·kg^{-1}. The optimum range was 0.20-1.08 mg·kg^{-1} Mo for *S. splendens* (Mills and Jones, (1996).

Conclusions

Symptoms of N, P, S, Ca, and K deficiencies and B toxicity manifested early; therefore, these disorders may be a more likely problem encountered by a grower. Necrosis was a common symptom for several of the deficiencies: N, K, Ca, Mg, S, Mn, and Zn. The early spotting observed with K, Ca, and Mg deficiencies led to necrosis. K and Ca deficiencies exhibited less yellowing than Mg deficiency. Ca deficiency exhibited less growth than K and Mg deficient plants. Necrosis appeared as mottled spotting with K deficient plants instead of interveinal necrosis observed with Mg deficiency. Ca deficient plants eventually exhibited plant collapse. N, S, Mn and Zn deficiencies manifested necrosis after other symptoms appeared. N deficient plants exhibited yellow and purple coloration before necrosis manifested on lower leaf tips. S deficient plants only manifested yellowing. Mn deficient plants exhibited significant growth before they first manifested yellowing which then developed into necrosis. Zn deficiency developed necrotic spots on upper leaves with inflorescences that lacked full development.

The described symptoms manifested themselves in salvia after being grown under nutrient deficient and toxic conditions, and the reported critical nutrient values originated from samples taken when the initial symptom occurred. It will be helpful for commercial growers to have this information available when diagnosing salvia nutritional problems.

Acknowledgments

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative, and we would like to thank PanAmerican Seed Co. for the salvia seed.

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Table 12.1 *Salvia farinacea* 'Evolution' plant dry weight and tissue nutrient concentration as affected by deficient or toxic nutrient treatments and published optimum concentrations.

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	0.53	1.03	2.33	2.33	2.39	1.04	3.94	2.33	9.91	2.02	6.45	13.99	2.39
Treatment	0.33	0.63	1.45	1.47	2.05	0.58	2.57	2.16	10.45	2.04	5.71	18.44	1.43
p-value[1]	**	*	***	***	NS	**	NS	NS	NS	NS	NS	NS	*
				Tissue Nutrient Concentration (%)						**Tissue Nutrient Concentration ($mg\cdot kg^{-1}$)**			
Element	**N**	**P**	**K**	**Ca**	**Mg**	**S**	**B**	**B**	**Cu**	**Fe**	**Mn**	**Mo**	**Zn**
Complete Control	5.06	0.67	6.07	1.66	0.66	0.45	56.0	46.8	5.5	72.0	114.4	0.6[2]	21.4
Treatment	2.57	0.12	0.47	0.40	0.08	0.24	13.0	242.1	1.1	55.5	7.6	—[3]	12.1
p-value[1]	*	***	***	***	***	***	***	***	***	*	***	NS	NS
Sufficiency Range for *Salvia splendens*[4]	2.38 - 5.61	0.30 - 1.24	2.90 - 5.86	1.00 - 2.50	0.25 - 0.86	0.73	25 - 75	25 - 75	7 - 35	60 - 300	30 - 284	0.20 - 1.08	25 - 115

[1] *, **, or *** indicates statistically significant differences between sample means based on F test at $P \leq 0.05$, $P \leq 0.01$, or $P \leq 0.001$, respectively. NS (not significant) indicates the F test difference between sample means was $P > 0.05$.

[2] Only one sample used in the number presented.

[3] Insufficient sample was available to determine concentration.

[4] Mills and Jones, 1996.

Chapter 13

Characterization of Nutrient Disorders of *Senecio cineraria* 'Silver Mist'

(In the format appropriate for submission to Acta Horticulturae)

Characterization of Nutrient Disorders of *Senecio cineraria* 'Silver Mist'

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Keywords: Dusty Miller, Calcium, Magnesium, Micronutrients, Nitrogen, Phosphorus, Potassium, Sulfur

Abstract

***Senecio cineraria* 'Silver Mist' 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

Dusty miller (*Senecio cineraria*, Asteraceae family) is a annual or biennial floriculture crop (Dole and Wilkins, 2005). Grown for its attractive silver foliage, it is typically used in the landscape or in containers to complement other ornamental plants (Armitage et al., 1994).

Hamrick (2003) recommended seed should be sown into a well-drained substrate with pH 5.5 to 5.8. The electrical conductivity (EC) should be below 0.75 mS/cm. Once the radicle is visible, use 14-0-14 to supply 50 to 75 $mg \cdot L^{-1}$ N. Armitage et al. (1994) recommended applying 50 to 75 $mg \cdot L^{-1}$ N from KNO_3 while the plants were in the plug stage. Hamrick (2003) recommended that after cotyledons have emerged, increase the fertigation rate to 100 to 150 $mg \cdot L^{-1}$ N, and alternate sources between 20-10-20 and 14-0-14. To minimize disease, she encourages growers to keep foliage as dry as possible while fertigating during all stages of production. During the hardening phase, plugs should be watered with 100 to 150 ppm N from 14-0-14 as needed.

Dusty miller should be potted into a disease-free, well-drained substrate that has a moderate starter charge (Hamrick, 2003). Armitage et al. (1994) advised using 100 to 150 $mg \cdot L^{-1}$ N from a complete fertilizer after potting and applying it during each irrigation. Hamrick (2003) suggested using 14-0-14 at a slightly higher rate of 150 to 200 $mg \cdot L^{-1}$ N to increase the “fuzziness” of the foliage.

No nutritional problems have been reported, and that lack of information can be disadvantageous to growers. The goal of this study was to elucidate the nutritional problems by determining the visual symptoms and tissue diagnostic values when they do occur.

Materials and Methods

Two 'Silver Mist' dusty miller plugs (2.0 x 2.04 x 2.9 cm cell size) were transplanted into 12.7 cm diameter (0.76 mL) 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 Oct. 2008. Treatments started immediately upon transplanting. 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 eighteen separate irrigation lines (each 1.82 m long). Each line contained six openings (12.7 cm diameter) that held the six pots for the elemental treatment. Thirteen lines contained the disorder treatments, and five lines contained the control. Pipes were randomly assigned as a control or treatment after the system was constructed. 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. Nutrients originated from the salts of KNO_3, $Ca(NO_3)_2 \cdot 4H_2O$, KH_2PO_4, $MgSO_4 \cdot 7H_2O$, FeDTPA, $MnCl_2 \cdot 4H_2O$, $ZnCl_2$, $CuCl_2 \cdot H_2O$, H_3BO_3, and $Na_2MoO_4 \cdot 2H_2O$. NaOH was added to adjust pH. In order to induce nutrient deficiency treatments, the plants were irrigated with complete nutrient solution excluding one of the nutrients. For macronutrients salt cations were replaced with Na, and anions were replaced with Cl (See Barnes et al., Chapter 14). For micronutrients the salt was omitted from the solution. 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). Irrigation was automated using a drip system utilizing sump-pumps (model 1A, Little Giant Pump Co., Oklahoma City, Oklahoma), and the frequency was adjusted to meet the plant biomass needs. The solution drained out from the bottom of the pot and was captured 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/or mature leaves as they developed.

When the initial deficient symptom of each treatment occurred, three pots containing two symptomatic plants each were selected for sampling. The remaining five pots were grown to document advanced symptoms. 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 leaf tissue, it was ground in a Foss Tecator Cyclotec™ 1093 sample mill (Analytical Instruments, LLC, Golden Valley, MN) to pass a $\leq$ 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 placing it 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 harvest of nitrogen 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 on 13 Feb. 2009, and non-symptomatic plants of copper and molybdenum 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. Values for percent difference in plant weights and tissue concentrations are presented.

Nitrogen

Nitrogen (N) deficient plants exhibited magenta and yellow coloration. There was a significant difference between control and N deficient tissue dry weights. At sampling N deficient plant dry weight was 22% less than the control. There was not enough sample to determine N concentration within the N deficient leaf tissue. The control had a value of

6.05% N. The survey average Mills and Jones (1996) published was 3.56% N. The control value was higher than this average.

Phosphorus

Plants grown in phosphorus (P) deficient conditions exhibited a purplish coloration in the lower leaf petioles. At time of sampling P deficient plants were 33% smaller than control plants. Leaf tissue P concentration in the control was 0.89% and 0.19% in the P deficient plants. Mills and Jones (1996) stated that the survey average for P was 0.66%. The control was above this value, and the deficient value fell below this value. Lower leaves then began to exhibit some yellowing which was followed by the leaf developing a tan coloration as it became necrotic.

Potassium

Symptoms of potassium (K) deficiency began with brown spots appearing on the insides of middle and lower leaves. Affected areas were limp to the touch. A significant difference did not occur between the dry weight control and K deficient tissue. Control K tissue concentration was 7.10% while K deficient tissue concentration was 0.48%. When compared with the survey average of 2.29% presented by Mills and Jones (1996), the control fell above this average while the deficient value fell below the average. Symptoms progressed with the tan spots increasing in size and in some areas turning dark gray. The entire leaf then became necrotic. Necrotic areas were crispy to the touch. On deeply lobed leaves the necrosis started on the tips of the dissected parts and moved inward. Upper leaves exhibited an 'umbrellaing' effect where the leaves had a downward curl. In the advanced stage many of the lower and middle leaves became necrotic, and they remained on the plant.

Calcium

Calcium (Ca) deficiency initially manifested itself as black and brown colored necrotic spots and blackening in the veins appearing on the most recently mature leaf; young leaves were also distorted. Inspection of the roots revealed a lack of development. At the time of harvest Ca deficient plants were 29% smaller than the control plants. Leaf tissue concentrations of control and Ca deficient plants were 1.33% and 0.12%, respectively. The survey average for Ca is 0.98% (Mills and Jones, 1996). The control value was above this value while the Ca deficient concentration was below this value. The disorder progressed with the necrotic spots growing in size. Lower and middle leaves began to exhibit spots. The spots that initially formed had a desiccated texture to them. New leaves failed to fully form and appeared as small "nubs." The spotting consumed lower leaves, and they became fully necrotic. Terminal buds began to develop, but they were very distorted and lacked full development.

Magnesium

Magnesium (Mg) deficiency began with lower leaves developing a greenish-yellow coloration. Also, the leaves looked limp as if they haven't been watered; however, the media was moist. Inspection of the roots revealed a lack of elongation. Mg deficient dry weight was 38% less than the control plants. The Mg concentration in the control plants was 0.46% and in the Mg deficient plants was 0.06%. The Mg concentration survey average published by Mills and Jones (1996) was 0.62%. Both the control and Mg deficient Mg concentrations were below this average. Mg deficiency progressed with the yellowing on the lower leaves becoming more pronounced. Discolored leaves developed brown necrotic spots. The spots then expanded.

Sulfur

The youngest leaves on sulfur (S) deficient plants had a slight greenish-yellow coloration. There was not a significant difference between the control and S deficient dry weights. Control tissue concentration was 0.50% which fell above the S concentration survey average of 0.36% presented by Mills and Jones (1996). The S deficient tissue concentration was 0.23% S, and it fell below the value (Mills and Jones, 1996). As the disorder progressed lower leaves began to exhibit a greenish-yellow coloration.

Boron

The initial symptom of boron (B) deficiency was a thickening in the young leaves. B deficient plants were 37% smaller than the control plants. Tissue B concentration was below the detectable limit of 1 $mg \cdot kg^{-1}$. Control B level was 26.4 $mg \cdot kg^{-1}$. The disorder progressed with the upper leaves not fully expanding. The leaves consisted of only the midrib and a few secondary veins. Advanced symptoms of the disorder like leaf browning, the development of necrotic honey-tan spots, and leaf tip curl developed on the lower leaves.

Plants grown under B toxicity conditions developed whitish-tan spots along the margin of mature leaves. There was not a significant difference between control and B toxicity dry weights. Control tissue B concentration was 36.9 $mg \cdot kg^{-1}$, and B toxicity plants had a concentration of 262.6 $mg \cdot kg^{-1}$. As the disorder progressed the spots grew in size, and the affected regions began to curl and distort. Spots were isolated to the lobe tips of the dissected leaves. Spots then appeared on middle and upper leaves.

Mills and Jones (1996) published a survey average of 17 $mg \cdot kg^{-1}$ for B concentration. Control values for both treatments were higher than the average. Concentration of B in the B deficient tissue was not detectable, and B toxic tissue concentration was higher than both this survey average value and the control.

Iron

The most recently expanded leaves of plants grown under iron (Fe) deficient conditions exhibited interveinal yellowing. Dry weights of Fe deficient plants were 25% less than the control plants. Control plant tissue had an Fe concentration of 114.2 $mg \cdot kg^{-1}$, and Fe deficient plant tissue had a concentration of 59.7 $mg \cdot kg^{-1}$. The survey average published by Mills and Jones (1996) was 79 $mg \cdot kg^{-1}$. The control concentration fell above this value, and the deficient concentration was below this value. As the plant grew new leaves developed interveinal yellowing. Intervienal yellowing eventually appeared in the youngest leaves and also in the young leaves of terminal shoots.

Manganese

The initial symptom of manganese (Mn) deficiency was the development of a greenish-yellow coloration over the entire plant. There was not a significant difference in plant tissue dry weights. Tissue from control plants had a Mn concentration of 212.8 $mg \cdot kg^{-1}$ while Mn deficient tissue had a Mn concentration of 7.9 $mg \cdot kg^{-1}$. The survey average published by Mills and Jones (1996) was 270 $mg \cdot kg^{-1}$. Both concentrations fell below this value. The greenish-yellow coloration was present for the remainder of the experiment. In the advance stage small necrotic tan spots appeared on the lower leaves.

Zinc

Plants grown in the zinc (Zn) deficient regime exhibited brown necrotic spots on the lower leaves. Plants grown under Zn deficient conditions had 28% less dry weight than control plants. Tissue values for the control and Zn deficient plants were 15.0 $mg{\cdot}kg^{-1}$ and 8.0 $mg{\cdot}kg^{-1}$, respectively. When compared with Mills and Jones's (1996) survey average of 73 $mg{\cdot}kg^{-1}$, both the control and deficient concentrations fell below the value. The lower leaves then became almost totally necrotic.

Asymptomatic

The experiment was terminated after 16 weeks of growth. No visual symptoms were apparent with those plants grown under copper (Cu) and molybdenum (Mo) deficient conditions. To determine if non-visual differences due to the deficiency occurred, plants were sampled and analyzed for dry mass and tissue concentration. Of these two elements, both were significantly different for tissue concentrations, but significant differences were not observed with plant dry mass.

The control tissue concentration for Cu was 10.2 $mg{\cdot}kg^{-1}$, and Cu deficient tissue concentration was 1.7 $mg{\cdot}kg^{-1}$. The survey average presented by Mills and Jones (1996) was 54 $mg{\cdot}kg^{-1}$ Cu. Both the control value and the deficient value were below this average. Mo tissue concentration for control was 2.14 $mg{\cdot}kg^{-1}$. In the Mo deficient plants Mo was only detectable in one plant, and that concentration was 0.1 mg kg^{-1}. Mills and Jones (1996) published a survey value of 0.21 $mg{\cdot}kg^{-1}$ Mo. The control concentration was above this value, and the deficient concentration was just below this value. While Cu and Mo deficient plants never exhibited symptoms, the tissue concentrations listed will be useful if growers are trying to determine if plant nutrient levels are low.

Conclusions

While no nutritional problems have been reported for dusty miller, this study revealed that they do indeed occur. The described symptoms and reported tissue values will aid growers in the event that a nutritional problem does arise in the production of this crop.

Acknowledgements

We are grateful for the funding support provided by USDA-ARS and the Floriculture and Nursery Research Initiative.

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Table 13.1 *Senecio cineraria* 'Silver Mist' plant dry weight and tissue nutrient concentration as affected by deficient or toxic nutrient treatments and published optimum concentrations.

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	0.49	0.49	1.64	2.63	2.63	0.49	1.64	3.48	18.81	1.64	3.48	18.81	8.06
Treatment	0.38[1]	0.33	1.40	1.85	1.63	0.45	1.04	3.24	19.80	1.22	3.22	19.60	5.80
p-value[2]	***	**	NS	**	***	NS	**	NS	NS	*	NS	NS	**

	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	6.05	0.89	7.10	1.33	0.46	0.50	26.4	36.9	10.2	114.2	212.8	2.14	15.0
Treatment	—[3]	0.19	0.48	0.12	0.06	0.23	—[4]	262.6	1.7	59.7	7.9	0.1[5]	8.0
p-value[2]	NS	***	***	***	***	***	***	***	***	**	***	**	**
Survey Average for 'Silverdust' *Senecio cineraria*[6]	3.56	0.66	2.29	0.98	0.62	0.36	17	17	54	79	270	0.21	73

[1] Two plants used per sample.

[2] *, **, or *** indicates statistically significant differences between sample means based on F test at $P \leq 0.05$, $P \leq 0.01$, or $P \leq 0.001$, respectively. NS (not significant) indicates the F test difference between sample means was $P > 0.05$.

[3] Insufficient sample was available to determine concentration.

[4] Tissue B concentration was below the detectable limit of 1 mg·kg^{-1}.

[5] Only one sample used in the number presented here.

[6] Mills and Jones, 1996.

Chapter 14

Comparison of the Symptomology of the Twelve Species Grown Under Nutrient Deficient and Toxicity Conditions

Comparison of the Symptomology of the Twelve Species Grown Under Nutrient Deficient and Toxicity Conditions

Introduction

Twelve species (Table 14.1) were grown under nutrient deficient and boron toxicity conditions. After growing these twelve species, similarities in plant response or symptomology were observed amongst the same nutrient treatment of different species. Some atypical symptoms were also observed. This chapter discusses both types of initial and advanced symptoms so that they may be recognized as commonly occurring symptoms on other plants.

Dicussion

Nitrogen Deficiency

Symptoms were observed for all twelve species grown under nitrogen (N) deficient conditions. An initial symptom for N deficiency was less plant dry weight than the control. Nine of the twelve species' plant dry weights were significantly less (22-71%) than the control plants. Another symptom was the overall manifestation of a greenish-yellow or yellow coloration. Initially, ten species exhibited an overall greenish-yellow or yellow coloration. Cineraria first manifested the yellow coloration in the lower leaves while fuchsia first manifested the yellow coloration in the upper leaves. A third symptom that manifested in species was the appearance of red, magenta, or purple coloration in the lower leaves, petioles and/or stem. Dusty miller, dahlia, fuchsia, and dianthus exhibited this disorder.

Similar advanced symptoms were observed in ten of the twelve species. The initial greenish-yellow or yellow coloration then intensified in the entire plant or in the lower and middle leaves. Leaf coloration that initially exhibited red, magenta, or purple color intensified overtime. Gomphrena also exhibited lower leaf magenta coloration six weeks after installation. Necrosis manifested on the lower leaves of eight species. Abutilon exhibited an atypical advanced symptom. The flowers on the N deficient plant were smaller and a lighter color than the control flowers.

Phosphorus Deficiency

Phosphorus (P) deficiency manifested initial symptoms for all twelve species. Nine of the twelve species exhibited less dry weight (21-67%) than the corresponding control species. A dark green coloration expressed over the entire plant or was in the lower leaves was a reoccurring symptom. For some species this coloration originated from small black-green or brown spots. Atypical symptoms occurred with dusty miller and dahlia which exhibited purple or magenta coloration, primula and cosmos which exhibited lower leaf necrosis, cosmos which developed a yellow coloration in the stem, leaves of abutilon which oriented parallel to the stem, and fuchsia which developed upper leaf greenish-yellow spots.

In the advance stage eleven of the twelve species manifested symptomology for P deficiency. Lower leaf yellowing, interveinal chlorosis, magenta or purple discoloration occurred on those species. This discoloration was followed by the development of necrotic spots which then spread over the entire leaf. On fuchsia, salvia, and abutilon the lower leaves abscised after becoming necrotic. Atypical advanced symptoms included upper leaf marginal spotting on salvia plants and smaller flowers on abutilon.

Potassium Deficiency

For potassium (K) deficiency all twelve species manifested initial symptoms. Seven species exhibited less plant dry weight (23-49%) than the control. Cineraria dry weight was 27% greater than the control. Nine species manifested lower leaf necrosis. Spots appeared along the margin or on the inside of the leaves, and spot color varied between whitish-green, gray-green, tan, or brown. Cosmos exhibited necrosis over the entire plant. Atypical symptoms were also observed. Primula exhibited overall marginal leaf yellowing, and upper leaves of fuchsia drooped and gave the upper part of the plant an 'umbrella' appearance.

Ten species out of twelve exhibited advanced symptoms for K deficiency. All of the species exhibited lower leaf necrosis. This necrosis increased in size in every case except primula because it exhibited no necrosis initially. Necrosis then moved upward in the plant leaves on dahlia and fuchsia. Some atypical symptoms manifested. Dusty miller upper leaves drooped and gave the top growth an ‘umbrella’ appearance. Inflorescences aborted on dahlia and fuchsia.

Calcium Deficiency

All twelve species manifested initial symptoms for calcium (Ca) deficiency. Dry weights of seven species were significantly less (26-60%) than the control dry weights. One symptom that was observed in eight species was the expression of upper leaf spotting or necrosis. Coloration of the spotting and necrosis included black, brown, gray-green, or white. Atypical symptoms were observed in primula that exhibited lower leaf marginal necrosis and gomphrena that exhibited lower leaf spotting. Coreopsis exhibited poor leaf elongation. Gazania flower buds exhibited browning.

The twelve species also exhibited advanced symptoms for Ca deficiency. All species exhibited a form of upper leaf necrosis or spotting in the advanced stage. Lower leaves also typically became affected with necrosis, yellowing, or interveinal chlorosis. Dahlia, cosmos, and abutilon inflorescences failed to develop, and fuchsia inflorescences were smaller in size. Apical buds aborted in salvia, dianthus, and abutilon. Necrosis became so pronounced in primula, dahlia, salvia, gazania, and coreopsis that these species died. Dusty miller exhibited an atypical symptom. The upper leaves distorted, and only the midribs and secondary veins of upper leaves formed. Fuchsia lower leaves also dropped.

Magnesium Deficiency

All twelve species exhibited a symptom for magnesium (Mg) deficiency. Dusty miller and gomphrena had significantly lower dry weights (38% and 31%, respectively) than the controls. Most of the species exhibited greenish-yellow or yellow coloration, yellowing margin, or interveinal chlorosis. These symptoms were typically observed in the lower leaves. Coreopsis and gomphrena exhibited an overall yellowing, and abutilon exhibited an upper leaf interveinal chlorosis. Cineraria, fuchsia, salvia, and cosmos did not exhibit yellowing but instead exhibited spotting or necrosis. It was located in the lower leaves except for cineraria which also manifested spotting in the upper leaves and salvia which developed spotting in the middle leaves. Fuchsia spotting was more of a bleached appearance.

All the species also exhibited advanced symptoms for Mg deficiency. Yellow coloration or interveinal chlorosis became more pronounced in the advanced stage. Necrosis was also a common symptom between the twelve species in the advanced stage, and it was typically observed in the lower leaves. Primula, salvia, abutilon, and gomphrena exhibited necrosis or bleaching in the interveinal region of the leaf. Fuchsia necrosis was followed by leaf drop which progressed upward on the plant. An atypical symptom was the lighter flower coloration of dahlia plants grown under Mg deficient conditions. Also atypical was the overall purple and yellow streaked coloration observed in dianthus plants.

Sulfur Deficiency

All twelve species exhibited initial symptoms for sulfur (S) deficiency. A significant difference in dry weight was only observed for dahlia, salvia, and coreopsis whose dry weights were less (40-44%) than the controls. Eleven species manifested a greenish-yellow color. Four of the eleven were an overall greenish-yellow color, and seven of the eleven species exhibited a greenish-yellow color in the upper leaves. Axillary shoots of abutilon and gomphrena also exhibited the greenish-yellow coloration. Cosmos symptomology was atypical; the flower peduncles were a whitish-green color. Dahlia also exhibited a magenta stem coloration along with an overall greenish-yellow color.

Eleven species exhibited advanced symptoms for S deficiency. As the disorder progressed the greenish-yellow coloration intensified to an overall yellow coloration. Some species then exhibited lower leaf necrosis. Abutilon necrosis began on the upper leaves and then progressed downward. Inflorescences of fuchsia were a lighter pink color, and later inflorescences that formed failed. Flower buds of gazania were an intense yellow coloration. Atypical symptoms observed with S deficiency were the manifestation of red leaf undersides on coreopsis plants and an overall light purple coloration on dianthus leaves.

Boron Deficiency

Boron (B) deficiency manifested for all twelve species. A significant difference in dry weight was observed for three species. Fuchsia dry weight was 37% greater than the control. Dusty miller and cosmos dry weights were less (37% and 25%, respectively) than the control. Symptoms varied for B deficiency but included upper leaf white or brown spotting, smaller upper leaf size or elongation, upper leaf distortion or crinkling, and upper leaf thickening or a rubbery feel. Fuchsia upper leaves also had a golden brown, shiny appearance, cosmos leaves also had a water-soaked appearance, and abutilon leaves also had a shiny appearance. Primula exhibited lower leaf yellowing, and dianthus exhibited lower leaf curling.

Advanced B deficiency symptoms manifested for eleven of the twelve species. Upper leaves began to manifest necrosis. In six species inflorescences aborted, and in four species the apical buds aborted. Lower leaves exhibited a variety of symptoms that included yellow, purple, or whitish green-coloration, interveinal chlorosis, brown spotting, or brown necrosis. Dianthus and gomphrena attempted to regrow new axillary shoots.

Boron Toxicity

Symptoms of B toxicity manifested in all twelve species. Cineraria dry weight was 27% greater than the control. The most common symptom was the appearance of lower leaf marginal brown spotting or necrosis. Yellow coloration, interveinal chlorosis, or a tan coloration also appeared on lower leaf margins. An atypical symptom was the manifestation of interveinal chlorosis in upper abutilon leaves.

Advanced symptoms were observed in eleven species. In most species the lower leaf margins then became entirely necrotic. Coreopsis margins stayed a yellow coloration.

The necrosis in the species then advanced upward on the plant into the middle and upper leaf margins. Dahlia exhibited necrosis in the inflorescence. Fuchsia dropped its lower leaves.

Copper Deficiency

Copper (Cu) deficiency manifested in five of the twelve species. Cineraria, dianthus, and gomphrena crop dry weights were significantly less (27-43%) than the control dry weight. Fuchsia and salvia developed whitish-hazed or bleached areas. Fuchsia symptoms appeared on the lower and middle leaves while salvia appeared on the middle and upper leaves. Abutilon and gomphrena both exhibited lower leaf spotting. Dianthus developed an overall bluish-green leaf coloration.

Only fuchsia, salvia, and gomphrena exhibited further symptoms. The bleached area expanded on fuchsia and salvia, and the number of spots on lower gomphrena leaves increased.

Iron Deficiency

Symptoms manifested on ten of the twelve species for iron (Fe) deficiency. A significant difference in dry weight was only observed for dusty miller and gazania whose dry weights were less (25% and 17%, respectively) than the controls. Upper leaf interveinal chlorosis was a common initial symptom. Primula and salvia exhibited an overall development of yellow coloration while dahlia and gazania developed lower leaf yellowing.

Eight species exhibited advanced symptoms. Interveinal chlorosis became more pronounced and appeared in the lower leaves on some plants. Axillary shoots also exhibited a yellow coloration on dusty miller, fuchsia, and dianthus.

Manganese Deficiency

Nine of the twelve species exhibited initial symptoms for manganese (Mn) deficiency. Cineraria, dahlia, and gomphrena crop dry weights were significantly less (21-30%) than the corresponding control dry weights. The most common symptom was the appearance of an overall greenish-yellow coloration. In gazania and coreopsis this symptom was the appearance of a 'honeycombing' effect of the small veins. Abutilon exhibited upper leaf marginal yellowing, and gomphrena exhibited lower leaf marginal yellowing.

Advanced symptoms appeared on the same nine species that exhibited initial symptoms. The yellow color intensified on gazania, coreopsis, and abutilon. Salvia developed an overall interveinal chlorosis. Necrotic spotting occurred on the lower leaves of several plants. Salvia necrosis appeared more on the middle leaves. Dianthus plants also exhibited reduced basal shoot growth.

Molybdenum Deficiency

Molybdenum (Mo) deficiency potentially manifested an initial symptom in cineraria and dianthus. Cineraria plants had significantly less dry weight than the control. However, recognizing this symptom would be difficult for growers because it manifested during the last harvest when the plants were at market stage. Dianthus plants exhibited less basal shoot growth, but the concentrations were not available to determine if it was a difference in Mo concentration that led to this symptom.

The lack of symptoms with Mo can be somewhat expected because it was reported to manifest in a limited number of crops like poinsettia (Dole and Wilkins, 2005), crucifers, and legumes (Gupta, 1997). It was induced in this experiment to see if Mo deficiency was an unknown problem of the 12 species.

Zinc Deficiency

All twelve species manifested symptoms for zinc (Zn) deficiency. Nine crop dry weights were significantly less (27-58%) than the control dry weights. Most often the upper leaves exhibited a yellowish-green coloration, uniform yellowing, or interveinal chlorosis. Other species like primula, dusty miller, dahlia, and gomphrena exhibited lower leaf yellowing, interveinal chlorosis, or brown spots. Atypical symptoms appeared on fuchsia where not only was yellowing present, but the upper leaves also exhibited an ‘umbrella’ appearance. Fuchsia flowers were a peach color instead of the typical red-orange.

In the advance stage nine species manifested symptoms. A variety of symptoms were observed in the advanced stage. Yellowing that had appeared as an initial symptom became more pronounced in color. Dusty miller, dahlia, gazania, and coreopsis exhibited lower leaf necrotic spots. Fuchsia, salvia, and abutilon exhibited upper leaf necrosis. Fuchsia also dropped its lower leaves. Gomphrena exhibited both upper and lower leaf necrosis. Zn deficiency negatively impacted the development of fuchsia, salvia, and abutilon flowers. They lacked complete development or aborted. An atypical symptom was observed on gazania leaves; they developed a greenish-white coloration.

Conclusions

The following initial and advanced symptoms manifested in the twelve species. Some symptoms were atypical for both the crop and the deficiency. Recognizing which disorders manifested symptoms more readily will provide a clue to which ones will occur most often. Symptoms appeared for all twelve species for N, P, K, Ca, Mg, S, and B deficiencies and B toxicity. Eleven species exhibited symptoms for Zn deficiency, ten species for Fe deficiency, nine species for Mn deficiency, five species for Cu deficiency, and 2 species for Mo deficiency. Presenting the symptoms in this grouped manner will provide growers the means to recognize the symptoms that occur with specific disorders.

Literature Cited

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Gupta, U.C. 1997. Symptoms of Molybdenum Deficiency and Toxicity in Crops. p.160-170. In: Gupta, U.C. (ed.), Molybdenum in Agriculture. Cambridge University Press, Cambridge, United Kingdom.

Table 14.1 Common name, scientific name, and cultivar of the twelve species grown under deficient or toxicity nutrient treatments.

Common Name	**Scientific Name**	**Cultivar**
abutilon	*Abutilon ×hybridum*	Bella Yellow
coreopsis	*Coreopsis grandiflora*	Santa Fe Yellow
cosmos	*Cosmos bipinnatus*	Sonata ™ White
dahlia	*Dahlia ×hybrida*	Maxi Morelia
dianthus	*Dianthus*	Bouquet Purple
fuchsia	*Fuchsia ×hybrida*	Gartenmeister Bonstedt
gazania	*Gazania rigens*	Daybreak White
gomphrena	*Gomphrena globosa*	Las Vegas Purple
cineraria	*Pericallis ×hybrida*	Jester Pure Blue
primula	*Primula acaulis*	Danova Rose
salvia	*Salvia farinacea*	Evolution
dusty miller	*Senecio cineraria*	Silver Mist

Table 14.2 Salts used to formulate stock fertilizer and base solutions, the salts' corresponding molecular weights, grams of salt used to mix the solution concentrate for the molarity listed, and mL of stock solutions used to prepare 100 L of nutrient solution.

Fertilizer Salt and Base	**Molecular Weight**	**Grams per Liter**	**Molarity**	**Amount of Stock Solution (ml) Added and Topped Up to 100 L**													
				ALL	**-N**	**-P**	**-K**	**-Ca**	**-Mg**	**-S**	**-Fe**	**-Mn**	**-Zn**	**-Cu**	**-B**	**+B**	**-Mo**
KNO_3	101.10	101.10	1M	500	—	500	—	500	500	500	500	500	500	500	500	500	500
$Ca(NO_3)_2 \cdot 4H_2O$	236.15	236.15	1M	500	—	500	500	—	500	500	500	500	500	500	500	500	500
KH_2PO_4	136.08	136.08	1M	100	100	—	—	100	100	100	100	100	100	100	100	100	100
$MgSO_4 \cdot 7H_2O$	246.48	246.48	1M	200	200	200	200	200	—	—	200	200	200	200	200	200	200
KCl	74.56	74.56	1M	—	500	100	—	—	—	—	—	—	—	—	—	—	—
$CaCl_2 \cdot 2H_2O$	147.02	147.02	1M	—	500	—	—	—	—	—	—	—	—	—	—	—	—
$NaNO_3$	84.99	84.99	1M	—	—	—	500	500	—	—	—	—	—	—	—	—	—
$NaH_2PO_4 \cdot H_2O$	137.99	137.99	1M	—	—	—	200	—	—	—	—	—	—	—	—	—	—
Na_2SO_4	142.04	142.04	1M	—	—	—	—	—	200	—	—	—	—	—	—	—	—
$MgCl_2 \cdot 6H_2O$	203.30	203.30	1M	—	—	—	—	—	—	200	—	—	—	—	—	—	—
FeDTPA	40.00	40.00	1M	100	100	100	100	100	100	100	—	100	100	100	100	100	100
$MnCl_2 \cdot 4H_2O$	197.91	3.9580	20mM	90	90	90	90	90	90	90	90	—	90	90	90	90	90
$ZnCl_2$	136.30	2.7260	20mM	15	15	15	15	15	15	15	15	15	—	15	15	15	15
$CuCl \cdot 2H_2O$	170.48	3.4096	20mM	15	15	15	15	15	15	15	15	15	15	—	15	15	15
H_3BO_3	61.83	6.1830	100mM	45	45	45	45	45	45	45	45	45	45	45	—	450	45
$Na_2MoO_4 \cdot 2H_2O$	241.90	0.2419	1mM	10	10	10	10	10	10	10	10	10	10	10	10	10	—
NaOH (base)	40.00	40.00	1M	~40	~40	~5	~45	~40	~40	~40	~6	~40	~40	~6	~6	~40	~40