COPPER TOXICITY IN WOODY ORNAMENTALS[1]

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Abstract. *Rhododendron obtusum* Planch. 'Delaware Valley' (Delaware Valley Azalea), *Buxus sempervirens* L. (common boxwood) and *Cotoneaster divaricata* Rehd. and Wils. (spreading cotoneaster) grown in sand culture received a complete nutrient solution containing Cu concns ranging from 0.032 ppm to 100 ppm. Development of Cu toxicity symptoms were recorded for 8 weeks, then Cu, Fe, and Mn levels in roots, stems, and leaves were determined. Chronic Cu toxicity symptoms were interveinal chlorosis and stunted growth. Acute Cu toxicity caused wilting, desiccation, and death of all affected plants. Tissue Fe and Mn levels were not consistently correlated to the Cu concn of the treatment solution. Only tissue Cu levels proved useful as an index of Cu toxicity.

Copper toxicity symptoms have been recorded for a number of fruit and agronomic crops (3), but not for ornamentals. Ornamentals may also be subjected to high Cu levels in several ways. Some industries regularly emit smoke containing Cu which can be deposited on foliage and soil. Repeated applications of Cu fungicides, such as Bordeaux mixture may result in toxic Cu levels in soil (5). Finally, many woody ornamentals are sold balled and burlapped with Cu treated burlap, which may be toxic to enclosed plants if $CuSO_4$ was the Cu source (2).

Diagnosis of a Cu toxicity problem is difficult because soil test results are hard to interpret, the symptoms are almost indistinguishable from other nutrient and physiological disorders, and little information exists regarding nutrient levels in Cu toxic plants. Soil test results are difficult to interpret because Cu is one of the most tightly bound cations, so it may be very localized in the soil. Its availability is also hard to determine since soil pH, cation exchange capacity, and organic matter content all influence the level at which Cu will be toxic (3).

Above ground symptoms of Cu toxicity are generally stunted growth and interveinal chlorosis (3), conditions which mimic both Fe and Mn deficiency. In fact, interrelationships between Cu, Fe, and Mn uptake have been found, with the levels of each affecting the uptake of the others (1). Foliar analysis has shown that leaves of Cu-poisoned plants may be Fe deficient. This deficiency may be overcome by supplying the plant with Fe-EDTA, but it does not reduce the toxic effect of Cu on the roots, which either accompanies or precedes leaf chlorosis (6).

To aid nurserymen and landscape contractors in identifying Cu toxicity of woody ornamentals, an experiment was carried out to obtain visual symptoms of Cu toxicity and tissue analysis data which would be useful in pinpointing a Cu toxicity problem.

Materials and Methods

Softwood cuttings of 'Delaware Valley' Azalea, common boxwood, and spreading cotoneaster were rooted under mist in unsterilized sand, transplanted into acid washed silica sand (particle size 0.3 to 1.0 mm) in 454 ml (16 oz.) containers, and watered with a complete nutrient solution. Iron was supplied in the chelated (EDTA) form to prevent its precipitation from solution. After plant establishment the Cu concn in the nutrient solution was adjusted with $CuSO_4$ to vary from 0.032 ppm (control) to 100 ppm. Plants were observed for visual symptoms of Cu toxicity. After 8 weeks roots, stems, and leaves were dry ashed and analyzed for Cu, Fe, and Mn using a Perkin Elmer 303 atomic absorption spectrophotometer.

Four replications of cotoneaster and boxwood and 3 replications of azalea were grown. However there was not enough dry weight produced for individual analysis of boxwood or all of the cotoneasters. Analyses of all boxwood, and

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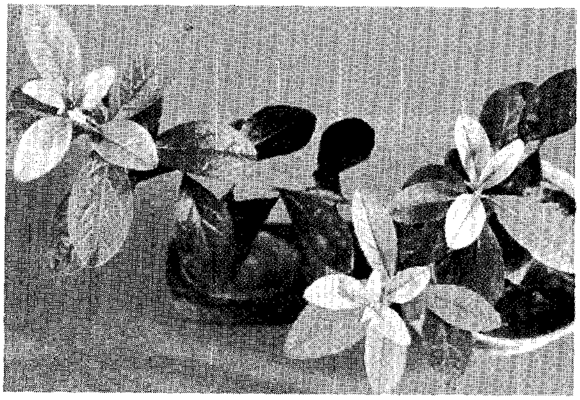

Figure 1. Chlorotic Delaware Valley White Azalea following 7 weeks treatment with 5 ppm copper.

Figure 2. Comparison of the effects of copper treatment on spreading cotoneaster. The plant on the left received 0.032 ppm copper and the plant on the right 5.0 ppm copper.

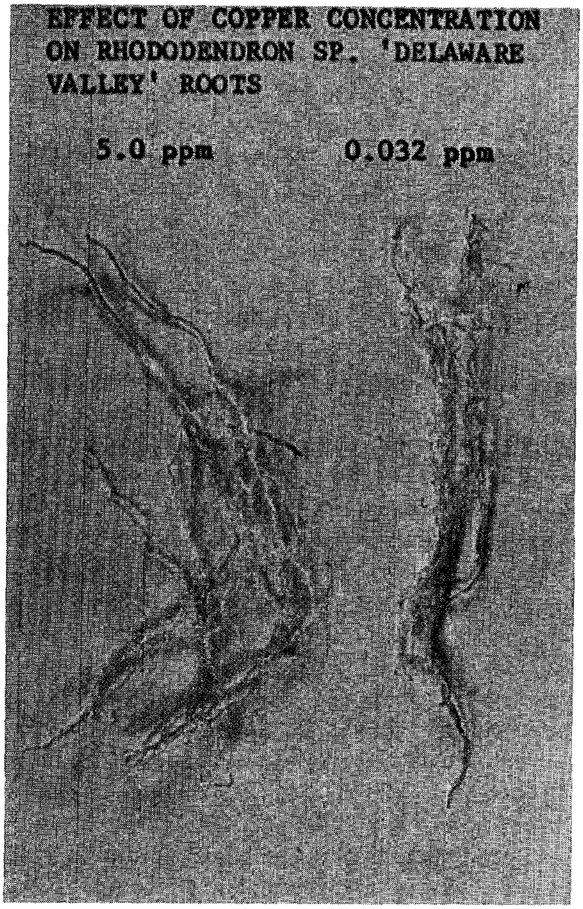

Figure 3. Effect of copper concentration on azalea roots. Lateral branching is reduced by chronic copper levels.

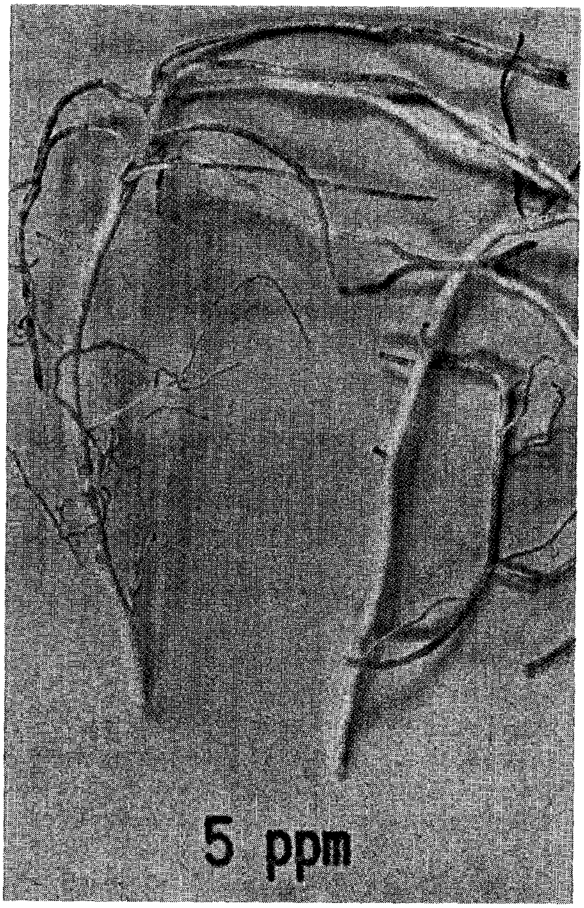

Figure 4. Chronic copper toxicity injury on boxwood roots. Necrotic lesions and undeveloped laterals are characteristic.

cotoneasters receiving 5 ppm Cu or above, were done on a composite of the 4 replications.

Results and Discussion

The Cu concns used caused 2 types of injury to the plants. At concns of 50 ppm Cu and below, chronic injury was induced resulting in the gradual decline of the plants. At 100 ppm Cu acute injury was induced, resulting in the sudden death of all affected plants.

Chronic injury is the type injury which would most commonly be found in ornamentals. It was induced in azalea and cotoneaster by Cu concns between 1 and 50 ppm, and in boxwood between 5 and 50 ppm. Generally, chronic symptoms were interveinal chlorosis and stunted growth (Figures 1 and 2), except for boxwood which did not become chlorotic but was simply stunted. The location of the chlorosis in azalea and cotoneaster was dependent on concn. With Cu concns between 1 and 5 ppm chlorosis began on the new growth, while at 50 ppm bottom leaves became chlorotic first. The higher Cu concns resulted in faster symptom expression. Chlorosis may have developed at lower Cu levels if chelated Fe had not been used, as it has been shown to alleviate the effects of high Cu concns (6). Surviving cotoneasters were pruned during the experiment, and this seemed to accentuate the problem. New growth was very severely stunted and chlorotic with 5 ppm causing all but terminal leaves to drop.

Chronic injury of the roots resulted in thicker main roots and fewer lateral roots (Figure 3). Dark stubs present on the root system were apparently lateral roots which were killed before elongating (Figure 4). Necrotic lesions were common on affected roots, which were also darker than healthy roots.

Copper at 100 ppm caused acute injury, but far exceeds the amount of Cu normally available to a plant. General symptoms of acute injury were wilting (Figure 5), dessication, and death of all affected plants. The youngest leaves were affected last, but newly expanded leaves remained small and poorly developed.

Other symptoms of acute injury were species related. Azaleas showed a gray discoloration of new leaves; and, as the wilting occurred, the hairs on the new growth became very prominent and appeared silver in color (Fig. 5). On cotoneaster and boxwood there occurred either a brown discoloration or chlorosis beginning at the base of the leaf and spreading outward (Figures 6 & 7), until the leaves abscised. With Cu toxicity, as opposed to water stress symptoms, the leaf margin is affected last. Lower leaves curled upward toward the stem, especially on boxwood, and some rosetting of terminal growth was evident. With boxwood only, leaves and stems became chlorotic, then leaf veins turned charcoal gray, beginning with the midvein and proceeding toward the margins.

Acute injury was hard to define on the roots because the plants declined so quickly. However, darkened root tips, necrotic lesions, and some desiccation were apparent when the plant roots were washed (Figure 8).

No consistent correlation was found between the Cu level in the nutrient solution and either Fe or Mn levels in roots, stems, or leaves (Table 1). Again, the FeEDTA in the nutrient solution probably accounts for the normal Fe levels in plants supplied high amounts of Cu. The exceedingly high Fe concn found in azalea roots suggests Fe was accumulating in the azalea roots instead of being translocated. This was not the case though, as the stems and leaves contained normal Fe levels. The Mn concn of cotoneaster roots and stems appeared to be abnormally low, yet the leaves contained adequate levels, precluding the appearance of foliar Mn deficiency symptoms.

The Cu concn of all plant parts of the 3 species tested was highly correlated to the Cu concn of the treatment solution ($r = 0.9$ or greater). Azaleas, boxwoods, and cotoneasters exhibiting Cu toxicity symptoms had leaf Cu levels of 11, 23, and 22 ppm or higher, stem Cu levels of 40, 61 and 37 ppm or higher, and root Cu levels of 257, 673, and 641 ppm or higher, respectively.

Copper toxicity symptoms are very hard to distinguish from other nutrient, and some physiological disorders. Soil tests are generally unsatisfactory in determining a Cu toxicity problem, as interpretation of the results depends on the analytical method and soil characteristics. Copper is toxic at much lower levels in a light sandy soil than in a soil high in organic matter or clay. High Cu levels resulting from Cu treated burlap are confined to the area around the burlap and would probably be missed by a soil test. Tissue analysis seems to be the only way to positively identify a Cu toxicity problem, and because Cu was the only one of the three elements analyzed that was consistently correlated to the Cu concn of the treatment solution, it is the only one which can be used as an index of Cu toxicity. Roots, stems, and leaves can all be used as indicators of Cu toxicity, but because of the magnitude of the difference between normal and toxic Cu levels in the roots, root Cu levels would seem to be the best indicator. In a related experiment though, Cu was found to be toxic at much lower levels (200 ppm) to roots of spreading cotoneasters grown in a media containing soil. Because of this and because the Cu concn which induced toxicity varied so much between both tissue and species, application of this data to other species must be done with caution.

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Table 1. Copper, Fe, and Mn concentrations in roots, stems, and leaves of Delaware Valley Azalea, common boxwood, and spreading cotoneaster grown under varying copper regimes.[z]

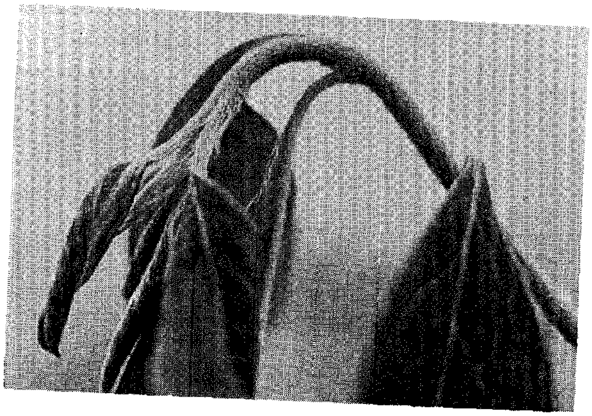
Species	Nutrient Solution Cu Concentration (ppm)	Root Cu concn ppm	Root Fe concn ppm	Root Mn concn ppm	Stem Cu concn ppm	Stem Fe concn ppm	Stem Mn concn ppm	Leaf Cu concn ppm	Leaf Fe concn ppm	Leaf Mn concn ppm
Azalea	0.032	64	10168	171	14	127	27	7	103	18
	0.5	187	2594	54	17	97	18	8	142	17
	1.0[y]	257	11021	173	40	88	18	11	178	16
	2.5[y]	599	5754	125	45	42	12	20	150	20
	5.0[y]	477	4565	125	80	57	25	21	132	21
	50.0[y]	4819	8380	147	x	x	x	35	224	27
	100.0[y]	6046	4716	115	266	132	85	56	321	34
		r = .96[w]	r = .18	r = .07	r = .98	r = .53	r = .97	r = .96	r = .95	r = .97
Boxwood	0.032	37	386	86	16	66	23	13	172	46
	0.5	115	274	105	23	73	19	14	185	37
	1.0	325	294	84	34	58	12	16	150	31
	2.5	569	286	95	37	43	15	17	163	35
	5.0[y]	673	251	97	61	64	8	23	139	19
	50.0[y]	3657	368	26	320	59	9	49	160	31
	100.0[y]	4368	466	18	296	69	12	65	174	40
		r = .96[w]	r = .81	r = .92	r = .9	r = .24	r = .38	r = .98	r = .21	r = .17
Cotoneaster	0.032	63	898	6	11	56	1	12	186	29
	0.5	282	965	8	23	42	1	22	150	26
	1.0[y]	641	1081	8	37	41	2	26	127	16
	2.5[y]	1438	901	9	40	35	2	22	127	32
	5.0[y]	2710	859	7	88	75	2	26	209	59
	50.0[y]	7246	1141	9	130	64	11	47	132	102
	100.0[y]	7460	1232	12	211	45	13	95	224	67
		r = .91[w]	r = .84	r = .86	r = .95	r = .03	r = .96	r = .98	r = .45	r = .66

[z] Data for azalea represent an average of 3 replications. Data for boxwood represent a composite of the 4 replications. Data for cotoneaster represent an average of the 4 replications at 2.5 ppm Cu treatment concentration and below, and a composite of the 4 replications at 5.0 ppm and above.

[y] Plants in these treatment categories exhibited Cu toxicity symptoms.

[x] Samples destroyed in a lab accident.

[x] Coorelation of the nutrient solution Cu concn with the tissue-element concn.



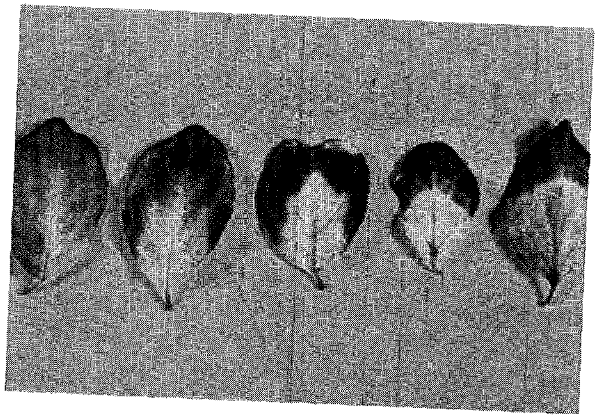


Figure 5. Wilting is a symptom of acute copper injury. Silver hairs were also *prominent* on young azalea leaves.

Figure 6. Basal chlorosis of cotoneaster caused by treatment for 10 days with 100 ppm copper.

Figure 7. Basal necrosis of cotoneaster caused by treatment for 10 days with *100* ppm copper.

Figure 8. Comparison of chronic and acute levels of copper on boxwood roots.