Yellows of Melons Caused by Molybdenum Deficiency in Acid Soil

W. D. GUBLER, Postgraduate Research Plant Pathologist, R. G. GROGAN, Professor, Department of Plant Pathology, University of California, Davis 95616, and P. P. OSTERLI, Farm Advisor, University of California Cooperative Extension, Stanislaus 95352

ABSTRACT

Gubler, W. D., Grogan, R. G., and Osterli, P. P. 1982. Yellows of melons caused by molybdenum deficiency in acid soil. Plant Disease 66:449-451.

Melons growing in acid soil in the San Joaquin and Sacramento valleys of California exhibited severe yellowing, stunting, and failure to fruit. Plant analysis and the prevention or rapid correction of this disorder by the application of dilute foliar sprays of sodium molybdate indicated that it was a molybdenum deficiency. Several melon cultivars, including Honeydew, Crenshaw, Juan Canari, Casaba, Santa Claus, and Persian, were susceptible.

Since molybdenum was shown to be an essential plant element (1), there have been several reports of molybdenum deficiency in various crop plants (4–6) and two reports (4,6) of molybdenum deficiency in melons. Lucas (4) reported that molybdenum deficiency of melons was induced by excessive fertilization with ammonium sulfate, which decreased the availability of molybdenum to the plant. Wilson (6) attributed the deficiency to low soil pH. The symptoms of some crops of melons in the San Joaquin and Sacramento valleys in California resembled molybdenum deficiency symptoms previously described (4,6), and the etiology and control of the disorder in California are described here.

MATERIALS AND METHODS

Affected and apparently healthy leaf and petiole samples from the third, fourth, and fifth nodes were collected from five affected fields for chemical analysis. Leaves were rinsed in distilled water and dried for 24 hr at 104 C. The samples were ground in a Wiley mill to pass through a 40-mesh screen and analyzed for molybdenum content by the method of Evans et al (3) or by that of Chapman and Pratt (2). For both methods, colorimetric determinations were made using a Bausch & Lomb Spectronic 20 spectrophotometer. Analysis for manganese, copper, zinc, calcium, magnesium, and potassium were made with a model 360 Perkin-Elmer atomic absorption spectrophotometer.

Accepted for publication 28 July 1981.

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0191-2917/82/06044903/$03.00/0

Soil for pH determination was taken from 5.0 cm inside the side-dressed fertilizer bands at an average depth of 8.5 cm. Soil pH readings were made on 2:1 suspensions of soil in 0.01 M calcium chloride. Relative concentrations of nitrate nitrogen were determined using a LaMotte plant tissue test kit, model PT-03R (LaMotte Chemical, Chestertown, MD 21620).

Large areas of the fields were affected by this disorder, and plots (randomized or paired plot design, three or four replicates) were established within affected areas. Treatments consisted of sodium molybdate sprays: a 2.3% solution applied at a rate of 2.3 L/ha and a 4.0% solution applied at a rate of 1.2 L/ha.

RESULTS AND DISCUSSION

Symptoms occurred on plants at all stages of growth; they first appeared as a light marginal and interveinal chlorosis of the crown leaves (Fig. 1A). As chlorosis became more severe, a pronounced marginal necrosis developed (Fig. 1B). Symptoms progressed rapidly, with affected plants becoming severely stunted and crown leaves necrotic (Fig. 1C). Few or no fruits set when the deficiency occurred on young plants. When older plants were affected after fruit set, fruit size was reduced.

Natural recovery from deficiency symptoms was observed in a few instances where no control was attempted. In these cases, recovery usually began 6–8 wk after initial symptoms were evident; because of the delay in fruit set, however, no marketable fruits were harvested from affected plants. Typically, plants did not recover naturally; they remained yellowed and stunted, with few or no fruits.

Plant tissue analyses for molybdenum, manganese, copper, zinc, calcium, magnesium, and potassium showed no apparent deficiency, except for molybdenum. Molybdenum concentrations of healthy plants ranged from 0.60 μg/g to 1.03 μg/g (Table 1), whereas concentrations in plants with symptoms ranged from a trace to 0.10 μg/g of oven-dry tissue. In all analyses, the molybdenum concentration of plants exhibiting yellows symptoms was less than that of healthy plants in the same field, and these values are in general agreement with values for deficient and healthy plants reported elsewhere (4).

In field trials over 2 yr, molybdenum salts, when applied as foliar sprays, gave rapid and complete recovery from symptoms on honeydew melon. A 2.3% solution of sodium molybdate applied at a rate of 2.3 L/ha, or a 4.0% solution applied at a rate of 1.2 L/ha, gave

Table 1. Molybdenum concentrations in healthy and deficient melon plants and soil pH ranges from root zones of melon cultivars showing deficiency symptoms

Field location	Cultivar	Molybdenum conc. (μg/g)[a]		pH range[b]
		Healthy	Chlorotic	
Yolo County	Honeydew	1.00	0.10	5.3–5.5
Stanislaus County	Honeydew	0.80	trace	4.9–5.1
Stanislaus County	Honeydew	···	···	5.7–5.9
Stanislaus County	Juan Canari	···	···	5.5–5.6
Stanislaus County	Honeydew	1.03	trace	5.5–5.5
Sutter County	Honeydew	0.60	trace	6.0–6.0
Sutter County	Honeydew	···	···	5.8–5.8
Sutter County	Honeydew	···	···	6.3–6.3
Sutter County	Honeydew	0.85	trace	5.8–5.9

[a] Concentration in leaves taken from the third, fourth, and fifth nodes; blanks indicate no analysis.
[b] Average range of pH from each affected area in nine fields (soil and 0.01M calcium chloride, 2:1). Range represents four sample sites per field.

excellent symptom recovery within 2–3 days regardless of symptom severity or size of plant.

In two fields where plants developed deficiency symptoms just before fruit set, application of a 2.3% solution of sodium molybdate at 2.3 L/ha resulted in fruit set in uniform clusters of four or five fruits midway out on the runner, whereas in unaffected fields and unaffected areas of these same fields, fruits were formed earlier and nearer to the crown.

In a trial established to determine the effect of nontreatment on yield, molybdenum at 2.3 L/ha was applied to deficient plants just before fruit set in an affected area that measured 110 × 300 m, and three rows were left untreated. Symptom recovery in treated plants was evident after only 2 days (Fig. 1D). Total fruits were counted in 76 m of row 1 day

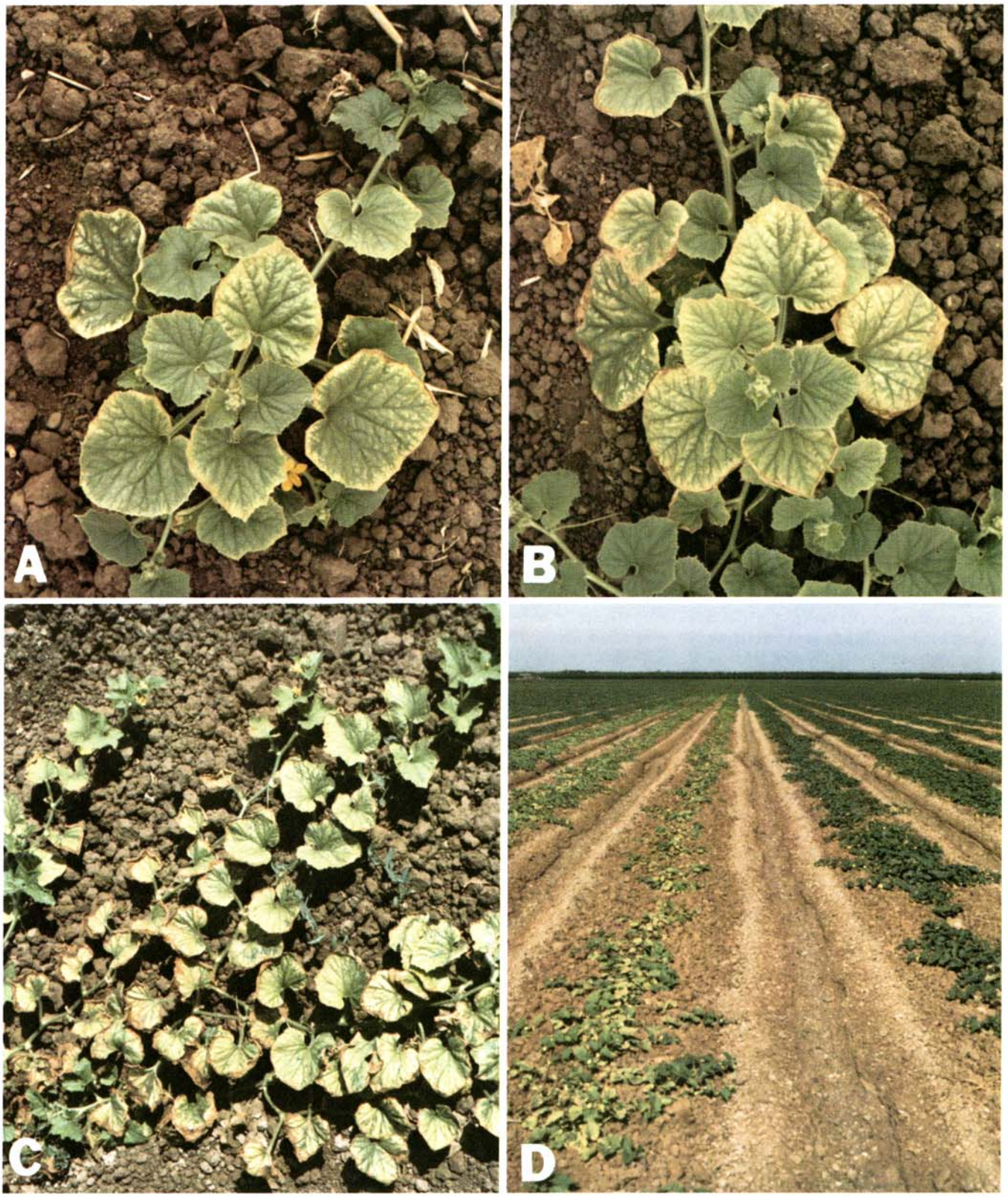

Fig. 1. Symptoms of molybdenum deficiency in melon: **(A)** Early symptoms, showing marginal and interveinal chlorosis and slight marginal necrosis. **(B)** Advanced molybdenum deficiency; note severe chlorosis and marginal necrosis of crown leaves, with progressive symptoms on runners. **(C)** Severe deficiency, resulting in stunting, death of crown leaves, and chlorosis and marginal necrosis on other plant leaves. **(D)** Field view of molybdenum deficiency; note rows on right that were sprayed with sodium molybdate 2 days before photographing.

before harvest in each of three untreated and treated rows. There was an average of 254 melons in each of the three treated rows (σ = 7.78) versus an average of 19 melons in the three untreated rows (σ = 22.19).

Symptoms of this disorder occurred on Honeydew, Crenshaw, Juan Canari, Casaba, Santa Claus, and Persian cultivars in the San Joaquin Valley and on cucumbers and Honeydew melons in the Sutter Basin production area of the Sacramento Valley.

Molybdenum yellows has been more severe in melon plantings along the Delta-Mendota Canal in Stanislaus County and was associated with cut-and-fill areas along this canal. In the Sacramento Valley, molybdenum deficiency occurred more commonly in melon plantings on the heavier soils.

In all instances, the problem has occurred in fields or portions of fields with low soil pH. The pH of soil in affected portions of fields was generally in the 4.9–5.9 range (Table 1). Because the soil in these production areas was usually 6.8–7.1 at the time of planting, the reduction in soil pH that occurred after planting could have been caused by side-dressed ammonium sulfate fertilizers applied at the time of first cultivation. Under these conditions, the release of the $SO_4^=$ anion would result in a lowering of pH, and the $SO_4^=$ anion itself could also compete directly with the $MoO_4^=$ anions for adsorption sites because of their similarity in size and charge (4). In two fields in the Sacramento Valley in which the soil pH was 6.0 and 6.3, the deficiency may have resulted from both the relatively low pH and extended wet soil conditions, but more likely resulted from the competition between molybdate and sulfate anions, possibly exacerbated by fertilization with ammonium sulfate.

Nitrate N concentration in diseased leaves was considerably higher than in healthy leaves. Because molybdenum is an essential component of the nitrate reductase enzyme, these findings supported the hypothesis of molybdenum deficiency. Symptom expression was apparently initiated by a deficiency of molybdenum in the plant and was accentuated by the subsequent high levels of nitrate that accumulated in leaf tissue.

Although in some cases the molybdenum deficiency of melons appeared to be a temporary disorder, the condition was usually prolonged, and affected plants produced no harvestable fruits.

In California, the apparent normal range of molybdenum concentration in healthy melon plants was 0.5–1.0 μg/g; in deficient plants, it ranged from a trace to 0.1 μg/g.

The area of melons in California currently under annual treatment for molybdenum deficiency is about 400 ha in Stanislaus County in the San Joaquin Valley and nearly 800 ha in the Sacramento Valley. Although aerial application was successful, ground-rig application resulted in more rapid recovery from symptoms because of better plant coverage.

LITERATURE CITED

1. Arnon, D. I., and Stout, P. R. 1939. Molybdenum as an essential element for higher plants. Plant Physiol. 14:599-602.
2. Chapman, H. D., and Pratt, P. F. 1961. Methods of analysis for soils, plants and waters. Calif. Univ. Div. Agric. Sci. Publ. 4,034. 309 pp.
3. Evans, H. J., Purvis, E. R., and Bear, F. E. 1950. Colorimetric determination of molybdenum by means of nitric and perchloric acids. Anal. Chem. 22:1568-1569.
4. Lucas, M. D. 1976. Deficiencia de molibdenio em melao num planossolo da regiao de tavira. Agron. Lusit. 37:151-162.
5. Plant, W. 1950. The relation of molybdenum deficiency to the acid soil complex. Trans. Int. Congr. Soil Sci., 44th 2:148-151.
6. Wilson, R. D. 1949. A field response of rock-melons to molybdenum. J. Aust. Inst. Agric. Sci. 15:118-121.