NUTRITIONAL DISORDERS OF SUNFLOWER

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by

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Preface

The common wild sunflower *(Helianthus annuus* L.) is indigenous to the South Western region of the United States of America, and was used by the native Indians for food, for medicinal purposes, and in ceremonies. The cultivated form (*Helianthus annuus* var. *macrocarpus*) is believed to have evolved from a mutation which was cultivated by the North American Indians, and was subsequently introduced into Europe in the 16th century. It spread rapidly, and later became an oilseed crop in Russia where commercial production of sunflower oil commenced between 1830 and 1840. The cultivars now grown are of European rather than American origin, the crop being reintroduced to North America about 1800.

The cultivated sunflower is among the four most important annual crops in the world which are grown for edible oil, the others being soybean, rapeseed and peanut. The oil is of high linoleic content, and generally commands a premium in the market place because of this characteristic, which is so necessary in the manufacture of polyunsaturated products.

The high oil sunflower crop was grown commercially on a large scale for the first time in Australia in 1969 from a self pollinating Russian cultivar, Peredovik. With the discovery of cytoplasmic male sterility by Dr. Leclercq of France in 1968, hybridisation of the crop became possible and was the big breakthrough for plant breeders.

While Australia is not a major producer of sunflower seed in quantity by world standards, the crop is important in the cropping scene in Eastern Australia, particularly in Queensland and New South Wales. However, the extent and quality of research work relating to the crop which has been done by Australian scientists has been immense, and well recognized by researchers throughout the world.

This publication, with the excellent photographs, will assist all involved in the crop, whether they be researchers, industry advisers or farmers, to readily identify the various nutritional disorders which can affect the raising of the sunflower crop across the whole spectrum of growing conditions which may be encountered. As such it will be an important reference book for all concerned.

Alan J. Lemon, MBE
President: Australian Sunflower Association (1983–1986)

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Introduction

In comparison with many other crops, sunflower (*Helianthus annuus* L. var. *macrocarpus*) has only recently become important on a world scale. The centre of origin of sunflower is recognized as being south-west to central North America (Heiser, 1976). Seeds of the plant were used by North American Indians, and the crop spread to Europe via Spain. Sunflower has been an important crop in the USSR for decades (Putt, 1978).

In recent times, sunflower production has increased rapidly (Fig. 1) so that this crop now ranks with soybean (*Glycine max* (L.) Merr.), cottonseed (*Gossypium* spp.), peanut (*Arachis hypogaea* L.) and rapeseed (*Brassica campestris* L. and *B. napus* L.) as one of the five most important annual oilseed crops (Table 1). The USSR remains the major producer of sunflower seed, but in 1983, 15 countries produced more than 100,000 tonnes of sunflower seed (Table 2). These countries produced collectively 97% of the world's production, with average yields ranging from approximately 600 kg/ha to over 2000 kg/ha.

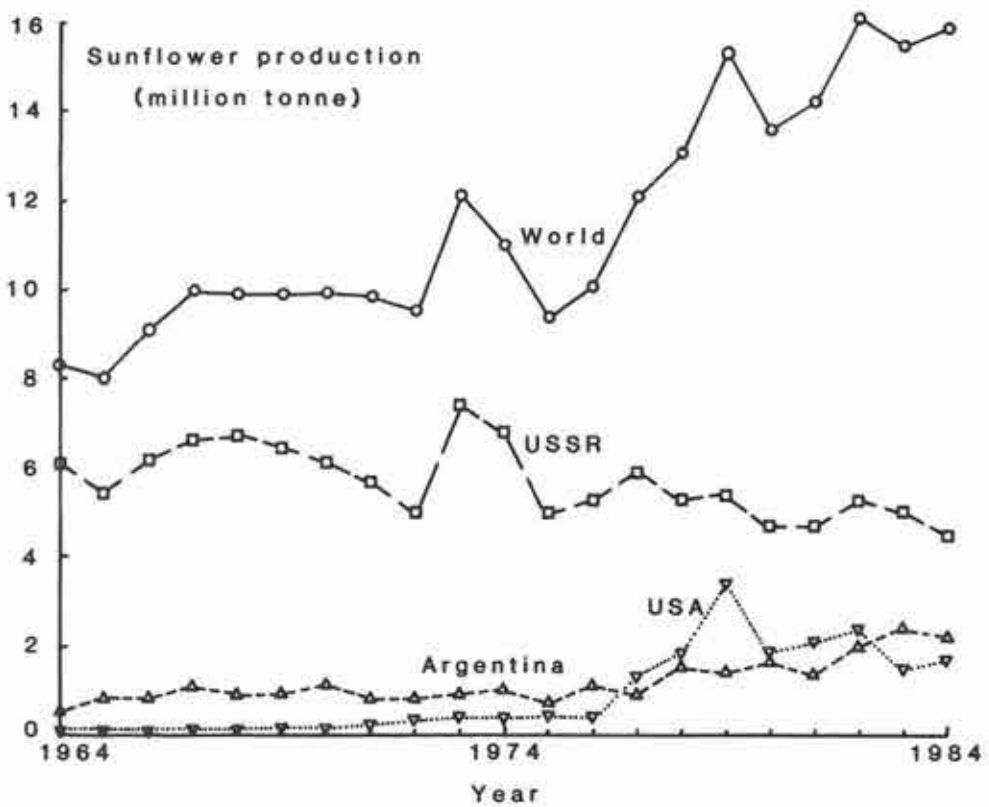

Fig. 1 World sunflower seed production and production in USSR, Argentina and USA from 1964 to 1984 (FAO, 1984)

Table 1. World Production (1983) of oilseed crops (FAO, 1984)

Crop	Production (million tonne)	Oil (%)
Soybean	79.40	19[1]
Cottonseed	27.79	30[1]
Groundnuts (in shell)	19.07	33[1]
Sunflower seed	15.50	44[1]
Rapeseed	14.02	43[1]
Olives	9.00	19
Copra	4.51	10
Linseed	2.23	39[1]
Palm kernels	2.14	10
Sesame seed	2.11	10
Castor beans	0.92	10
Safflower seed	0.86	35[1]

[1]Sims (1981)

Table 2. Sunflower seed production in 1983 of the major producing countries (FAO, 1984)

Country	Area harvested (million ha)	Average Yield (kg/ha)	Production (million tonne)
USSR	4.26	1,181	5.04
Argentina	1.90	1,262	2.40
USA	1.24	1,171	1.45
China	0.73	1,829	1.34
France	0.43	1,933	0.83
Turkey	0.55	1,300	0.71
Romania	0.49	1,431	0.70
Spain	0.92	736	0.68
Hungary	0.28	2,071	0.59
Bulgaria	0.26	1,734	0.45
India	0.46	497	0.23
South Africa	0.32	622	0.20
Yugoslavia	0.08	1,837	0.14
Italy	0.07	1,941	0.13
Australia	0.18	591	0.10
World	12.82	1,208	15.50

With the increasing importance of this crop, production has spread to include new areas that may have soils with different problems to those of the traditional production areas. One such problem is the poorer chemical fertility of many soils recently planted to sunflower. For example, molybdenum deficiency has been reported in sunflower in Australia (McDonald, 1978) and South Africa (Blamey and Chapman, 1979).

The study of nutritional disorders of crop plants is particularly important to agricultural producers. Failure to correct nutritional disorders may have major consequences on crop growth and production. Fortunately, very often it is possible to correct nutritional disorders, both from technological and economic points of view. However, it is necessary to know the cause of the nutritional disorder in order to correct the problem (e.g. by selecting the appropriate type and quantity of fertilizer to apply).

Since sunflower is a relatively recent crop of world importance, information is not readily available on the effects of nutritional disorders on this crop. Also, as production spreads to areas of lower native fertility than have been used traditionally for sunflower production, nutritional disorders other than those occasioned by macronutrient deficiencies are likely to increase in importance.

The aim of this publication is to assist all involved in sunflower production – researchers, advisers and producers – in the identification and correction of nutritional disorders. Emphasis has been placed on symptoms of nutritional disorders. However, where possible, tissue concentrations and soil tests associated with yield reduction have been presented also. Unless otherwise indicated, tissue concentrations are those in the blade of the youngest expanded leaf (YEL) at Growth Stage R-2 (Schneiter and Miller, 1981) from programmed nutrient addition studies (Asher and Blamey, 1987) conducted at the University of Queensland from 1982 to 1986.

Nutrient requirements of sunflower

The Essential Elements

As with other plant species, sunflower requires 13 chemical elements for growth in addition to carbon (C), oxygen (O) and hydrogen (H). Plants cannot grow without a supply of these elements, and they have thus become known as essential elements or nutrient elements. To be considered an essential element, a particular element must meet the following criteria:

i. a deficiency of the element makes it impossible for the plant to complete its life cycle;
ii. a deficiency of the element can only be alleviated by supplying the element;
iii. the element must be involved directly in the nutrition of the plant.

The essential elements, other than C, O and H, have been divided into macronutrients (major nutrients) and micronutrients (trace elements) on the basis of the quantities that plants require.

Macronutrients	*Micronutrients*
Nitrogen (N)	Iron (Fe)
Potassium (K)	Manganese (Mn)
Phosphorus (P)	Zinc (Zn)
Calcium (Ca)	Copper (Cu)
Magnesium (Mg)	Boron (B)
Sulfur (S)	Chlorine (Cl)
	Molybdenum (Mo)

In addition to the essential elements, other elements may cause nutritional disorders because of their toxicity. In particular, aluminium (Al) toxicity is an important consideration in acid soils, i.e. soils with low pH (< 5.5 when measured in water). Sodium (Na) may be present in saline and sodic soils at concentrations that are deleterious to plant growth. Toxicities of heavy metals, e.g. cobalt (Co), chromium (Cr), nickel (Ni), lead (Pb), thallium (Tl) and cadmium (Cd), may result from industrial pollution or the amendment of soil with sewage effluent. Other elements, e.g. selenium (Se), may be naturally present in high concentrations.

In addition, some essential elements may reach levels that are deleterious to plant growth. Examples include Mn toxicity in acid soils and waterlogged soils (Labanauskas, 1966); B toxicity in alkaline soils, through the application of irrigation water high in B or the over-application of B; and Cu and Zn toxicities, through the excessive use of fertilizers containing these elements. Additionally, the application of one nutrient may reduce the absorption of another nutrient to such an extent that a deficiency results.

Robinson (1973) measured concentrations of macronutrients and micro-

Table 3. Concentration of elements in sunflower seed and stover, and content of elements in a crop producing 1 tonne seed/ha (after Robinson, 1973)[1]

Element	Element concentration		Element content in a crop producing 1 tonne seed/ha		
	Seed	Stover	Seed	Stover	Total
	(%)			(kg/ha)	
N	2.58	1.03	25.8	15.5	41.3
P	0.39	0.08	3.9	1.2	5.1
K	0.59	1.51	5.9	22.7	28.6
Ca	0.11	1.10	1.1	16.5	17.6
Mg	0.23	0.58	2.3	8.7	11.0
S	0.17	0.20	1.7	3.0	4.7
Na	<0.02	0.10	<0.2	1.5	<1.7
	(mg/kg)			(g/ha)	
Fe	33	150	33	228	261
Mn	14	27	14	41	55
Zn	48	34	48	51	99
Cu	13	4	13	6	19
B	14	34	14	51	65
Mo	6	15	6	23	29
Al	3	189	3	284	287

[1] Harvest Index of 40% assumed.

nutrients in sunflower seed and stover as well as the total amounts of these elements in a crop (Table 3). Concentrations of some of these elements in sunflower seed have been measured recently by Seiler (1984) as follows: 3.0%N; 0.69% P; 0.82% K; 0.35% Ca; and 0.18% Mg. In both studies, the sunflower plants were apparently healthy.

Diagnosis of Nutritional Disorders

Visible symptoms

Nutrient deficiencies and toxicities often produce characteristic visible symptoms which can play an important role in the diagnosis of nutritional disorders in the field. This method of diagnosis has the advantage that it is not dependent on costly laboratory equipment or time-consuming chemical analysis. Unfortunately, once symptoms become visible, considerable crop loss may have already occurred. Additionally, some disorders produce rather similar symptoms or no symptoms at all, and the effects of insect pests and diseases may produce symptoms similar to those of nutritional disorders. To complicate the situation even further, plants may suffer multiple nutritional disorders producing complex symptoms, and the application of one nutrient to overcome a deficiency may result in another nutrient becoming deficient. Thus, confirmation of a disorder from visible symptoms requires experience; and in any event should be seen as a first step in diagnosis to be confirmed by soil or tissue analysis or both.

In addition to the appearance of a particular symptom, the position or location of that symptom must be noted (Asher *et al.*, 1980). Nutrients are absorbed by the root system, and distributed among various plant parts. Some of these nutrients may be redistributed to younger parts of the plant during times of shortage either readily (e.g. N, P), more slowly (e.g. S), or hardly at all (e.g. B, Ca). Thus, deficiencies of N and P are likely to be observed on older leaves, that of S on both older and younger leaves, and those of B and Ca on the younger leaves. Environmental conditions (e.g. moisture supply, temperature, light) may affect considerably the appearance and severity of nutrient disorders. For example, B deficiency symptoms in sunflower are most severe under drought conditions, and Mn toxicity symptoms are severe in lucerne (*Medicago sativa*) in waterlogged soils (Graven *et al.*, 1965). Elements in excess amounts continue to be accumulated in the leaves. Thus, there will be a tendency for toxicity symptoms to appear first on the older leaves where accumulation has been occuring for the longest time.

In spite of the difficulties and the need for caution, visible symptoms of nutrient deficiencies and toxicities remain an important tool in the diagnosis of nutritional disorders.

Tissue analysis

Tissue analysis is also an important technique in the diagnosis of nutritional disorders. In annual crops, tissue analysis is most often used in 'trouble shooting' rather than in the recommendation of fertilizer rates. However, the application of fertilizer is not precluded, provided the tissue samples are collected early in crop growth and the chemical analyses swiftly completed.

The method of tissue analysis is based on an established relationship between crop yield and nutrient concentration in plant tissue (Fig. 2). Critical concentrations for deficiency and toxicity have been defined as those concentrations associated with 90% of maximum yield (Ulrich and Hills, 1973). Between these concentrations is a range of concentrations required for healthy growth.

The relationship between crop yield and nutrient concentrations in plant tissue may be determined by means of nutrient solution culture experiments, of which there are many techniques (Asher and Edwards, 1983), glasshouse pot experiments, and field experiments. Generally, field experiments are considered the best method (Bates, 1971), but are considerably more expensive than solution culture and pot experiments. They also depend on the availability of sites at which each of the problems to be studied is well developed.

A certain part of the plant rather than the whole plant, is usually selected for analysis, leaves being considered the most satisfactory parts (Bates, 1971). Because leaves may continue to accumulate some nutrients with increasing age, it is important that nutrient concentrations in leaves of the same physiological age be compared. In sunflower, as in many other annual crops, the blade of the youngest expanded leaf (YEL) is most often selected for tissue analysis.

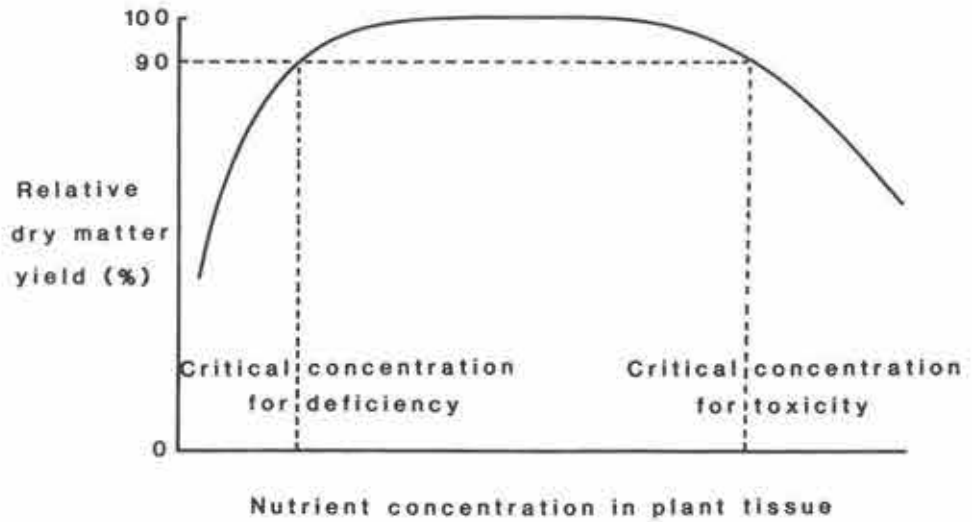

Fig. 2 Schematic relationship between nutrient concentration in plant tissue and dry matter yield relative to the maximum possible

Soil analysis

The total amount of nutrient in the soil does not reflect the quantity available for uptake by plant roots. Thus, chemical methods have been developed, and continue to be developed, to estimate that quantity of a nutrient that is available to the plant. In addition to the requirement that the method provide a good estimate of nutrient availability, soil analysis methods must be rapid, accurate, and reproducible to be accepted for routine use in soil testing laboratories.

The results of soil analyses must be interpretable, i.e. based on previously-established relationships between crop yield and soil test. These relationships may be established by means of glasshouse pot experiments or field experiments. Also, relationships need to be established between soil test values and fertilizer applied in order that recommendations on fertilizer rates may be made.

One advantage of soil analyses is the fact that they can be conducted and fertilizers applied before a crop is planted. Disadvantages of soil analyses include the difficulty of obtaining methods suited to varied soil types; problems in sampling due to variability in fertility across a field; and problems in estimating the likely effects of environmental conditions in the forthcoming season (Melsted and Peck, 1973).

Correction of Nutritional Disorders

Once a nutritional problem has been correctly diagnosed it is usually possible to correct the disorder. Often, a fertilizer containing the particular nutrient

will be applied either to the soil or as a foliar spray to overcome a deficiency. In other cases, an amendment or ameliorant may be applied to correct a soil problem causing the disorder. The most important amendments are gypsum ($CaSO_4.2H_2O$) to correct problems of soil surface crusting and poor permeability in saline and sodic soils, and lime ($CaCO_3$) or dolomite ($CaCO_3.MgCO_3$) to overcome problems associated with soil acidity. In the case of salinity, water management methods may be used to leach toxic elements from the root zone.

It has long been known that plant species, and even cultivars within a species, differ in their sensitivity to low nutrient levels in the soil (Brown *et al.*, 1972). Two reasons for these variations have been proposed, viz. (i) variation in nutrient uptake and (ii) variation in tissue requirement for a particular nutrient. Agricultural scientists have developed cultivars for particular nutrient situations, an example being the breeding of sunflower cultivars to overcome the problem of B deficiency (Blamey *et al.*, 1984).

Disorders producing symptoms mainly on the older leaves

1. Nitrogen Deficiency

Nitrogen (N) deficiency has been found to be the most common nutritional disorder limiting sunflower growth and yield (Robinson, 1978). Irrigated sunflower has been found to remove about 130 kg N/ha in producing a seed yield of 3500 kg/ha (Cheng and Zubriski, 1978).

Symptoms of nitrogen deficiency

Although N deficiency is the most common nutritional disorder in field-grown crops, recognition of N deficiency is difficult. Indeed, the overall, paler colour of the N-deficient crop may easily be overlooked in a field uniformly suffering this disorder.

As with other crop plants, N deficiency appears first as a reduction in growth. This growth reduction may or may not be accompanied by visible symptoms. When symptoms occur, they may include a general chlorosis of seedlings (Plate 1.1) and of plants at a more mature stage of growth (Plate 1.2). On the other hand, reductions in yield (Blamey and Chapman, 1980) have been found in crops with no visible reduction in height nor apparent chlorosis, though leaf area was reduced (Plate 1.3).

Nitrogen deficient crops may exhibit an overall chlorosis with young and old leaves equally affected (Plate 1.1, 1.2). On the other hand, as found in many other crops, a striking yellow chlorosis may be more evident on the older leaves (Plate 1.4, 1.5). Also, the oldest leaves may become completely necrotic, or develop an overall yellow chlorosis with necrotic areas along the leaf margins. In pot experiments, N deficiency has resulted also in plants with thin stems (Plate 1.6).

Possible confusion with other symptoms

Nitrogen deficiency can be confused most easily with sulfur deficiency. Deficiencies of both N and sulfur result in reduced growth and plants with an overall chlorotic appearance (Plate 1.2, 11.2). Nitrogen deficiency may be distinguished from sulfur deficiency in that sulfur deficiency often results in a slightly mottled chlorosis (Plate 11.3), and the chlorosis may be more pronounced on the upper leaves. However, considerable potential for confusion exists in diagnosis of N and sulfur deficiencies from visible symptoms alone.

Nitrogen deficiency may be confused also with molybdenum deficiency, particularly in the early stages of growth, when molybdenum deficient seedings exhibit an overall pale chlorosis. However, a distinction arises between these two disorders in that molybdenum deficient seedlings develop

a marked upward cupping of the leaves (Plate 8.1, 8.2), a symptom not observed with N deficiency. Also, N deficiency is generally visible on older plants whereas molybdenum deficiency appears most commonly on young seedlings. (Recovery from molybdenum deficiency is both rapid and spectacular following a foliar spray with sodium molybdate solution.)

Diagnostic soil and plant tissue tests

Soil tests for nitrate-nitrogen (NO_3–N) have proved useful in the diagnosis of possible N deficiency and in predicting N fertilizer requirements of sunflower, particularly in drier areas of the world. Recommendations for the application of N fertilizer have been based on the difference between NO_3 – N in the soil profile and the N requirement of the expected crop.

The application of N fertilizer has increased the N concentration in plant tissues, including experiments in which yield responses to N fertilizer have been recorded. Cheng and Zubriski (1978) found, that as seed yields increased from 2,734 to 4,197 kg/ha, N concentrations (whole plant) increased from 4.91 to 5.21% when plants were 30 cm high and from 1.38 to 2.02% at the ray floret stage (Growth Stage R-5.1) (Schneiter and Miller, 1981).

The differences in N concentration at different growth stages pose obvious problems in the diagnosis of N deficiency. In a 3-year study, Loubser (1983) found concentrations of 4.0 to 5.5%N in the topmost mature leaves at flowering (Growth Stage R-5.1) in the highest-yielding plots. In solution culture, a critical concentration of 3.3% N has been found in the youngest expanded leaf of plants at Growth Stage R-2.

Correction of nitrogen deficiency

In flowing solution culture, Forno (1977) found that nitrate (NO_3^-) was a better source of N for sunflower than was ammonium (NH_4^+). At optimal external NO_3 – N concentrations (500-5000 μM N), the relative growth rate (RGR) was 20% per day, whereas at optimal NH_4-N concentrations (30-120 μM N), the RGR was only 13% per day.

In irrigated sunflower in the Murrumbidgee Irrigation Area (MIA),

Plate 1. Nitrogen deficiency.

1.1 Nitrogen deficiency in a sunflower seedling (L) grown in nitrogen deficient nutrient solution compared with a seedling (R) grown with adequate nitrogen in solution.

1.2 Nitrogen deficiency in sunflower in the field (Source: B.T. Steer).

1.3 Lack of symptoms in sunflower grown with inadequate nitrogen (no N applied) (R) compared with sunflower fertilized with 180 kg N/ha (L).

1.4 Nitrogen deficiency symptoms on the lower leaves of sunflower grown in nutrient solutions with inadequate (R) nitrogen compared with those grown with adequate nitrogen (L) (Source: N.J. Grundon).

1.5 Chlorosis of the lower leaves of sunflower plants due to nitrogen deficiency.

1.6 In pot culture, nitrogen deficiency may result in plants with thin stems (L) compared with plants adequately supplied with nitrogen (R). (Source: N.J. Grundon).

1.1

1.2

1.3

1.5

1.4

1.6

Hocking and Steer (1982) found that most of the N absorbed was in the NO_3^- form. Nitrogen added as urea was rapidly converted to NH_4^+, which was subsequently converted to NO_3^- Thus, fertilization with different forms of N fertilizer should have little effect on sunflower response.

The timing of N fertilizer application can have marked effects on crop response. Although visible signs of N deficiency may be absent in the young plant, it is particularly important to ensure adequate N nutrition before floret initiation, especially during the period between 20 and 40 days after sowing (Steer *et al.*, 1984). Ensuring adequate N status during this period is particularly important since N deficiency reduces floret number per plant, and hence the number of seeds per plant.

Because of marked interactions between N requirement and environmental factors such as soil N status, moisture availability (Hunter and McCosker, 1982), and soil phosphorus levels (Blamey and Chapman, 1980; Hunter and McCosker, 1982), as well as genetic differences (Muirhead *et al.*, 1982), the response of sunflower to N fertilization is difficult to predict. In Minnesota, N fertilizer recommendations have been made on the basis of expected yields (Fenster *et al.*, 1978). For expected seed yields of over 2,800 kg/ha, an application of 135 kg N/ha is recommended. Nitrogen rates are lower with lower yield expectations, 65 kg N/ha being recommended for yields < 1,680 kg/ha. In western Minnesota, NO_3-N in the top 60 cm of soil is subtracted from recommended N fertilizer rates. In Queensland, a response to N fertilizer is considered unlikely when the soil (0–10 cm) contains > 18 mg NO_3-N/kg (Rayment and Bruce, 1984).

Substantial seed yield increases have been reported following N fertilization at rates up to 180 kg/ha (Blamey and Chapman, 1980) and 200 kg/ha (Tomov, 1976 according to Robinson, 1978). Zubriski and Zimmerman (1974) found that fertilization with 112 kg N/ha increased seed yield from 2,193 to 3,043 kg/ha (average of 5 trials). Cheng and Zubriski (1978) reported a yield increase of 54% (2,734 to 4,197 kg/ha) following an application of 112 kg N/ha to irrigated sunflower. Blamey and Chapman (1980) reported yield increases of over 1,500 kg/ha with 180 kg N/ha, but noted that yield responses to N were dependent on soil phosphorus status. Mathers and Stewart (1982) found that fertilization with 84 kg N/ha was sufficient for a maximum seed yield of 2,900 kg/ha. Hunter and McCosker (1982) reported substantial yield increases through N fertilization, but that the magnitude of the response was dependent on moisture supply. Also, the two cultivars tested responded differently to applied N. Indeed, marked genetic differences in sunflower response to N fertilization have been reported (Muirhead *et al.*, 1982): one group of sunflower cultivars responded to rates up to 30 kg N/ha; a second group responded to up to 90 kg N/ha; and a third group to rates up to 150 kg N/ha.

In spite of marked increases in sunflower vegetative and seed yield from N fertilization, N supply was found to have no effect on the timing of floret initiation, anthesis, or seed maturity (Hocking and Steer, 1982). Although phenology was not affected, N deficiency has been reported to cause a marked reduction in seed number per plant (Steer *et al.*, 1984). Indeed, seed number

per plant was the yield component most sensitive to N deficiency. To prevent yield loss, it is most important to alleviate N deficiency before floret initiation.

Sunflower has been found to be relatively efficient in the recovery of fertilizer N, and particularly in translocating N to the seed. Although sunflower seed was found to account for only 38% of the total (above ground) plant dry weight, N in the seed accounted for 68% of that in the above ground parts of the plant (Cheng and Zubriski, 1978). Recovery of applied N in the seed, while decreasing with increasing N fertilization, has been found to vary from 36–40% (Cheng and Zubriski, 1978) to 60% (Blamey and Chapman, 1980).

Nitrogen fertilization has been found to substantially increase protein concentration in the seed (Cheng and Zubriski, 1978; Robinson, 1978; Blamey and Chapman, 1980). In contrast with protein, oil concentration is reduced by N fertilization. Cheng and Zubriski (1978) found that an application of 112 kg N/ha decreased the oil concentration in the seed from 50.0 to 48.5%. Blamey and Chapman (1980) reported up to 3.8% reduction in oil concentration following an application of 180 kg N/ha, and Muirhead *et al.* (1982) found that 150 kg N/ha reduced the oil concentration of a number of cultivars by an average of 2.5%. Over 10 sites, on soils with differing N status, Scott and Mead (1980) found that fertilization with 60 kg N/ha decreased oil concentration in the seed by 0.5 to 2.0%. However, in spite of decreased oil concentration following N fertilization, yields of oil per hectare were substantially increased through the beneficial effect of N fertilization on seed yields.

In irrigated sunflower, N fertilization increased water use efficiency from 39 kg seed/ha/cm water without N application to 58 kg seed/ha/cm water at 112 kg N/ha (Cheng and Zubriski, 1978). However, yields may be depressed by over-fertilization of dryland sunflower if the increased vegetative growth results in moisture stress before seed maturity.

2. Phosphorus Deficiency

After nitrogen deficiency, phosphorus (P) deficiency is probably the most limiting nutritional disorder to sunflower production throughout the world (Robinson, 1978; Spencer and Chan, 1981; Blamey and Chapman, 1980; Mead and Scott, 1980). Marked responses to P fertilization have been recorded on both alkaline and acid soils. In Australia, seed yield increases of 170 kg/ha have been recorded through applying 20 kg P/ha (Mead and Scott, 1980); and 310 kg/ha through applying 52 kg P/ha (Spencer and Chan, 1981).

Although much evidence suggests that P deficiency in sunflower is widespread, and that considerable yield responses to applied P may occur, the study of P nutrition has not received as much research attention as it has in other crops. Also, recent research by Hibberd *et al.* (1984) and Hunter and Kochman (1985) has suggested that the P nutrition of sunflower may be con-

siderably more complex than that of many other crops. This results from the symbiotic relationship that sunflower is able to form with vesicular arbuscular mycorrhizae (VAM). The formation of this symbiosis considerably improves the efficiency with which sunflower is able to extract P from the root environment. For example, Hunter and Kochman (1985) found that sunflower without VAM infection was P deficient when grown in soil with 13 mg/kg bicarbonate extractable P. On the other hand, sunflower with roots infected with VAM grew adequately in soil with concentrations as low as 6 mg/kg bicarbonate extractable P (Hibberd *et al.*, 1984).

Symptoms of phosphorus deficiency

In the field-grown crop and in plants grown in solution culture, P deficiency is first evident as reduced growth compared with plants adequately supplied with P (Plate 2.1). Indeed, this reduction in growth may persist through the growth of the crop without any other symptoms of P deficiency. This lack of characteristic symptoms renders diagnosis of P deficiency difficult.

On closer examination of the field-grown crop (Plate 2.1, 2.2), a dark grey necrosis of the lower leaves of P deficient plants may be evident. Phosphorus deficiency in solution culture has sometimes been found to reduce plant growth without any characteristic symptoms. Using the technique of programmed nutrient addition (Asher and Cowie, 1970), the regular addition of small quantities of P in solution (albeit quantities of P inadequate for maximum growth) reduced plant growth but produced very few necrotic symptoms of P deficiency (Asher and Blamey, 1987) (Plate 2.3).

In severe cases of P deficiency in solution culture, a dark grey necrosis of the lower leaves develops (Plate 2.4), similar to the symptom observed in the field. Closer examination of this symptom on the lower leaves showed a clear demarcation between healthy and necrotic tissue with no chlorosis surrounding the necrotic areas (Plate 2.4). A further characteristic of this symptom of P deficiency was the appearance of watersoaked areas that became necrotic in what appeared to be concentric circles (Plate 2.5). The development of this symptom was not unlike that produced by some fungal pathogens (Hunter and Kochman, 1985). The necrotic areas were particularly prevalent in the interveinal regions between the major veins (Plate 2.6). Bergmann (1986) reported poor head development in P deficient sunflower.

Plate 2. Phosphorus deficiency.

2.1 Response of sunflower to 60 kg P/ha (background) compared with no P application (foreground) on a soil with 6 mg/kg bicarbonate extractable P.

2.2 Comparison of the effect of a pre-plant application of 60 kg P/ha (R) with no P applied (L) on sunflower at flowering.

2.3 Lack of symptoms on sunflower plants grown in solutions with inadequate (L) and adequate (R) phosphorus.

2.4 Necrosis of a lower leaf of a sunflower plant inadequately supplied with phosphorus.

2.5 Watersoaked and necrotic areas due to phosphorus deficiency (Source: M.N. Hunter).

2.6 Interveinal necrosis due to phosphorus deficiency in sunflower (Source: N.J. Grundon).

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2.2

2.3

2.4

2.5

2.6

Possible confusion with other symptoms

Because the main symptom of P deficiency in sunflower is reduced plant growth, it may be extremely difficult to diagnose P deficiency, particularly in the early stages of growth. Even with relatively severe P deficiency and at late stages of growth, no charactertistic symptoms may be evident.

A further problem in correctly diagnosing P deficiency results from similarity between the lower leaf necrosis produced with this disorder and the symptoms produced by biotic diseases, viz. *Alternaria* and *Septoria* blights. Indeed, Hunter and Kochman (1985) were led to investigate this possibility, but concluded that no organisms were responsible for the necrosis observed (Plate 2.5). However, the problem remains that this symptom of inadequate P may be confused with symptoms caused by *Alternaria helianthi* or *Septoria helianthi*.

Diagnosis and correction of phosphorus deficiency

Because of the relative immobility of P in soils, P should be applied to row crops in adequate amounts before planting. Thus, it would be of greatest benefit to use a soil test for plant available P, and to apply the required quantities on the basis of such a test. Blamey and Chapman (1980) found that seed yield of sunflower was closely related to bicarbonate extractable P, and that seed yields increased up to a soil test value of 19 mg P/kg. In Minnesota, Fenster *et al.* (1978) recommended an application of 18 kg P/ha when the extractable P (Bray # 1) was < 10 mg/kg. In Queensland, response of sunflower to P fertilizer is considered unlikely if the bicarbonate extractable P is >35 mg/kg (Rayment and Bruce, 1984).

In spite of success with soil tests, the recent findings that sunflower response to P applications varies depending whether or not the roots are adequately infected with VAM (Hunter and Kochman, 1985) casts doubt on much of the earlier findings. Because VAM infection decreases with increasing length of the fallow period prior to planting, soil tests for extractable P may need to be evaluated with this in mind.

Plant tissue tests may serve a valuable role in assessing the P status of the crop. Difficulties have been experienced, however, due to changing critical P levels as the plants age (Spencer and Chan, 1981). In a field experiment, the critical P concentration in the youngest expanded leaf decreased from 0.35% at the fourth week from sowing to 0.21% at the tenth week. Loubser and Human (1983) found that the critical P concentration varied over years and cultivars from 0.20 to 0.31%, even though leaves were sampled at the same physiological age. A review of several other studies showed that the critical P concentration in leaf blades varied from 0.20 to 0.32% (Reuter, 1986).

3. Potassium Deficiency

Data from Robinson (1973) indicate that sunflower has a rather high potassium (K) requirement, the seed containing 0.6% K and the mature stover 1.5% K. Hence, high-yielding sunflower crops may remove considerable amounts of K from the soil.

However, sunflower is usually grown on soils of high K status, making K deficiency in the field an uncommon problem. In a comprehensive review, Robinson (1978) reported only one instance (Weibel, 1951) where K fertilizer increased seed yields (from 465 to 1,375 kg/ha). Most Australian soils, particularly those soils used currently for sunflower production, contain adequate levels of plant-available K (Williams and Raupach, 1983). However, K deficiency may occur in sunflower produced on coarse-textured soils low in available K.

Symptoms of potassium deficiency

Symptoms of K deficiency in sunflower first affect the lower leaves, with younger leaves showing symptoms of the disorder only in severe cases. In seedlings, the oldest leaves develop a generally yellow colour and large necrotic patches develop accompanied by severe buckling of the leaf (Plate 3.1). In severe cases, this distortion affects most leaves on the plant (Plate 3.2). In older plants, K deficiency symptoms first appear as a chlorosis of the margins and interveinal regions of the lower leaves (Plate 3.3). The leaves with chlorotic areas often develop an upward cupping, especially towards the tips (Plate 3.1, 3.2), although downward cupping of the leaves has also been observed (Plate 3.2, 3.3). The chlorotic areas of the lower leaves eventually become necrotic and complete senescence of the lower leaves may occur.

Bussler (1964) noted that the necrosis is sometimes preceded by chlorosis, but that the chlorosis quickly vanishes, and the necrosis often borders on healthy tissue. The necrosis is more severe towards the leaf tip and in the interveinal areas, and the leaf base remains green for the longest time. Similar symptoms have been reported by Bergmann (1986).

Possible confusion with other symptoms

Because of the interveinal chlorosis and downward cupping of the lower leaves, symptoms of K deficiency may be most easily confused with those of magnesium deficiency (Plate 4.1–4.5). However, no upward cupping of the leaves has been observed with magnesium deficiency. Also, chlorosis and necrosis due to K deficiency are more severe toward the tip of the leaf (Bussler, 1964; Bergmann, 1986).

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Plate 3. Potassium deficiency

3.1 Chlorosis and necrosis on an older leaf of a sunflower seedling with potassium deficiency.
3.2 Sunflower seedling with severe potassium deficiency.
3.3 Comparison of potassium deficient plants (L) with healthy plants (R) in solution culture..

Diagnostic soil and plant tissue tests

In Minnesota, Fenster *et al.* (1978) suggested that sunflower would be likely to respond to K fertilizers on soils with < 0.26 meq/100g exchangeable K. In Queensland, sunflower response to K fertilizer is considered unlikely if the exchangeable K in the soil is > 0.25 meq/100g (Rayment and Bruce, 1984).

In flowing solution culture where K concentrations were kept constant, Spear *et al.* (1978) reported a concentration of 1.8% K in the youngest expanded leaf associated with a 50% reduction in sunflower vegetative yield. In our study with cv. Hysun 31, solution culture experiments yielded a critical concentration (associated with 90% maximum yield) of 2.4% K in the youngest expanded leaf. Bergmann (1986) reported a K concentration of 0.58% in leaves of K deficient plants while the leaves of healthy plants contained 3.74% K.

Correction of potassium deficiency

Correction of K deficiency, if confirmed, can be most easily accomplished by the application of K fertilizers. Rates of between 37 and 56 kg K/ha have been recommended with soil tests from 0.13 to 0.26 meq/100g and < 0.13 meq/100g, respectively (Fenster *et al.*, 1978).

4. Magnesium Deficiency

Sunflower is produced mainly on soils high in available magnesium (Mg), and no reports of Mg deficiency in field-grown sunflower have been found. In spite of the general lack of Mg deficiency in most sunflower producing areas, Mg deficiency might be encountered in sunflower grown on coarse-textured soils low in available Mg, or where Mg deficiency has been induced by excessive applications of potassium fertilizer.

Symptoms of magnesium deficiency

The first symptom of Mg deficiency is a mottled, interveinal chlorosis on the lower leaves (Plate 4.1). The mottling may affect the whole leaf or only be visible on one side of the midrib. The veinal area remains green, and indeed the mottling of the interveinal area may be quite striking with little change in colour of the region near to the veins (Plate 4.2). Similar symptoms were reported by Bergmann (1986).

As the severity of the disorder increases, a marked downward cupping and bronzing of the leaves occurs (Plate 4.3), and necrotic patches appear (Plate 4.4). The necrotic areas are particularly evident in the interveinal regions of the lowest leaves (Plate 4.5). Severe leaf distortion often accompanies the necrosis.

Possible confusion with other symptoms

Because the interveinal chlorosis and necrosis develops on the lower leaves, symptoms of Mg deficiency may be confused easily with those resulting from potassium deficiency (Plate 3.1–3.3). With potassium deficiency, however, leaves with upward cupping have been observed, whereas upward cupping of leaves has not been observed in plants with Mg deficiency. Also, symptoms of potassium deficiency are more severe towards the leaf tip (Bussler, 1964; Bergmann, 1986).

Diagnostic soil and plant tissue tests

A consequence of the apparent lack of reports of Mg deficiency in field-grown crops is the lack of an adequate diagnostic soil test.

In our studies with cv. Hysun 31 in solution culture, a critical concentration (corresponding to 90% of maximum yield) of 0.18% Mg in the youngest expanded leaf has been found.

Correction of magnesium deficiency

In other crops, Mg deficiency is particularly a problem of sandy soils, an example being 'sand drown' of tobacco (*Nicotiana tabacum*). If encountered in sunflower, Mg deficiency could be corrected by the application of magnesium sulphate. In strongly acid soils, the possibility exists that Mg deficiency may be induced by the presence of toxic concentrations of aluminium in the root environment. In such a situation, both Mg deficiency and aluminium toxicity would be corrected by the addition of dolomite.

Plate 4. Magnesium deficiency.

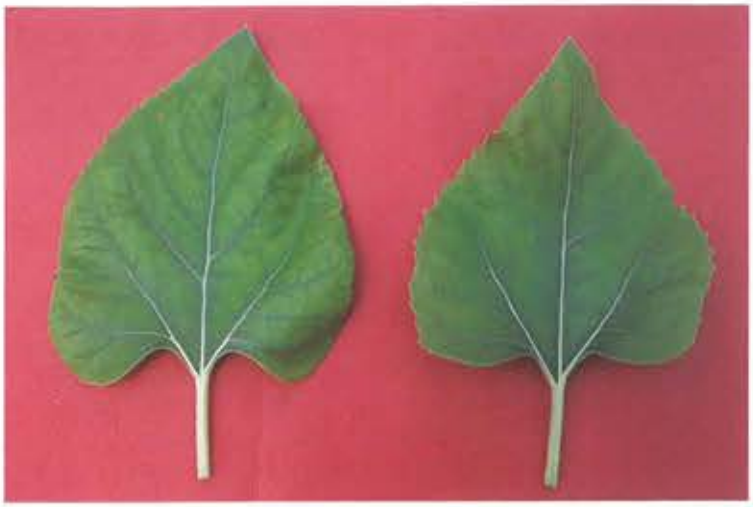
4.1 The initial, mottled, interveinal chlorosis observed on the lower leaves of plants with magnesium deficiency.

4.2 Severe interveinal chlorosis on the lower leaf of a plant with magnesium deficiency (Source: N.J. Grundon).

4.3 Bronzing and downward cupping of a leaf due to magnesium deficiency.

4.4 Moderate (R) to severe (L) chlorosis and necrosis due to magnesium deficiency.

4.5 Severe chlorosis and necrosis and downward cupping of leaves due to magnesium deficiency.



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5. Boron Toxicity

Although sunflower is extremely sensitive to boron (B) deficiency (Schuster and Stephenson, 1940), it is highly tolerant of B toxicity. No reports of B toxicity in field-grown crops have been encountered. Indeed, Blamey and Chapman (1979) reported no yield depression in field-grown sunflower when the B concentration in the leaf was as high as 180 mg/kg. It is unlikely that B toxicity would be a problem in sunflower. Firstly, B toxicity would be encountered in other crops, e.g. navy bean (*Phaseolus vulgaris*), soybean (*Glycine max*), peanut (*Arachis hypogaea*), before being evident in sunflower. Secondly, B toxicity generally arises from irrigation with water high in B or from over-fertilization with B fertilizer (Richards, 1954; Tisdale and Nelson, 1966). Because of the extreme tolerance of sunflower, neither of these situations would be likely to cause problems.

However, one problem that would arise concerns other crops grown in rotation with sunflower. Indeed, yields may be reduced in subsequent crops following the over-fertilization of sunflower with B. For example, Blamey *et al.* (1981) reported a yield reduction in peanut following 3 years' fertilization of sunflower at 3 kg B/ha/year.

Symptoms of boron toxicity

The first symptom of B toxicity appears as a slight chlorosis along the margins of the lower leaves (Plate 5.1). It is characteristic that the chlorosis first appears approximately 2 mm in from the serrated edge of the leaf.

With increasing severity, necrosis may appear in the previously chlorotic areas, particularly along the leaf margins and in the area between the midrib and the two major veins originating from the base of the leaf blade (Plate 5.2, 5.3). The necrosis may be surrounded by a yellow chlorosis. Also, the necrosis has a mottled appearance, with dark brown areas within the lighter brown areas. The leaves not affected by the chlorosis or necrosis remain dark green, and may develop a downward cupping (Scott, 1960; Cerda *et al.* 1981). Symptoms of B toxicity progress from the lower leaves to those higher up the plant.

Plants showing symptoms of B toxicity do not always have reduced yield.

Possible confusion with other symptoms

Symptoms of B toxicity in sunflower are characteristic, particularly in severe cases. However, with mild B toxicity, symptoms may be confused with those

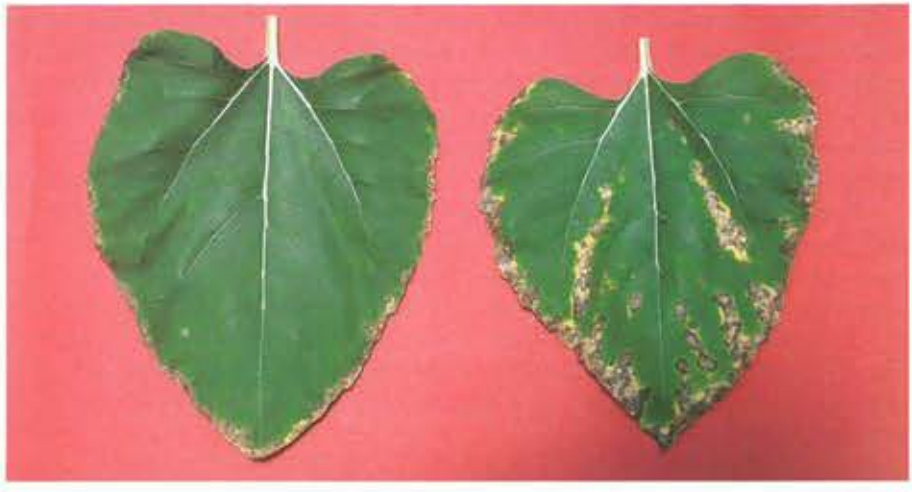
Plate 5. Boron toxicity.

5.1 Initial marginal chlorosis (R) and later marginal necrosis (L) as well as interveinal chlorosis and necrosis due to boron toxicity.

5.2 Moderate (L) and severe (R) marginal necrosis, the latter accompanied by interveinal necrosis, due to boron toxicity.

5.3 Severe marginal and interveinal necrosis due to boron toxicity.

5.1



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caused by excess sodium chloride in the root environment (Plate 10.1). However, in the case of salinity, the mottled necrosis does not appear nor have necrotic areas been observed in areas away from the leaf margin as does B toxicity. Boron toxicity symptoms may also be confused with those where excess sodium sulphate is present in the root zone (Plate 10.3). In the latter case, interveinal chlorosis and necrosis do occur. However, the necrosis due to excess sodium sulphate is not mottled and affects the entire plant, not only the lower leaves as in B toxicity.

Diagnostic soil and plant tissue tests

Because B toxicity has not been reported in sunflower grown in the field, no data are available to suggest soil test levels that might be associated with B toxicity in this crop. In solution culture, Cerda *et al.* (1981) found that concentrations higher than 185 μM B in solution significantly decreased sunflower growth and yield.

Decreases in sunflower growth have been associated with increased B concentrations in leaf tissue. In a solution culture study, Cerda *et al.* (1981) reported that, at flowering, concentrations higher than 500 mg B/kg in the 4th leaf from the cotyledons were in the severe B toxicity range (resulting in 30% maximum yield). In our solution culture studies with cv. Hysun 31, a critical B concentration for toxicity (associated with 90% maximum yield) of 1,150 mg/kg was found in the topmost mature leaf. When present in excess supply, B accumulates in a characteristic pattern in the leaf. Increasing B concentrations have been found from the middle of the leaf towards the leaf margins. Scott (1960) reported B concentrations of 807, 1,100, and 2,177 mg/kg in the healthy tissue, chlorotic tissue and necrotic tissue of sunflower leaves suffering B toxicity. This finding and the pattern of symptom development are in keeping with the hypothesis that B moves passively in the transpiration stream.

Should B toxicity occur, it would be most difficult to correct. Indeed, it would be advisable to prevent the problem by not applying irrigation water high in B or over-fertilizing with B fertilizer. However, once encountered it might be possible to overcome the problem by leaching out the excess B from the root zone, by liming or by the liberal use of nitrogen fertilizer, especially calcium nitrate (Bradford, 1966).

Because of the extreme tolerance of sunflower compared with other crops, the potential exists for producing sunflower on soils unsuitable for the production of other crops sensitive to B toxicity (e.g. bean).

6. Phosphorus Toxicity

Phosphorus (P) toxicity can occur in plants growing in solution culture or in light sandy soils (Snowball and Robson, 1986). The disorder most often occurs when P fertilizer is applied to P deficient plants. No reports of P toxicity in field-grown sunflower have been found.

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Plate 6. Phosphorus toxicity.

6.1 Pale necrosis of the interveinal areas of the older leaves due to phosphorus toxicity.

6.2 Symptoms of phosphorus toxicity resulting from a combination of high phosphorus supply and low zinc supply in solution culture (R). The lower leaves have a marginal and interveinal chlorosis and necrosis. Other plants (L) developed no symptoms when given the same level of phosphorus and an adequate zinc supply (Source: N.J. Grundon).

6.3 With high phosphorus concentrations and low zinc concentrations in solution, the interveinal areas of lower leaves develop distinct necroses surrounded by a pale yellow chlorosis (Source: N.J. Grundon).

Symptoms of phosphorus toxicity

In solution culture experiments in which low P concentrations (approx. 0.1 μM P) had developed through plant removal of P, increasing the P concentration to 4.8 μM resulted in the development of P toxicity symptoms. One day after the addition of P to the solution, the older leaves developed watersoaked areas between the major veins. One day thereafter, the watersoaked areas became necrotic (Plate 6.1).

In solution culture experiments attempting to produce zinc (Zn) deficiency symptoms, a disorder due to P toxicity (Loneragan *et al.*, 1982) has been observed (N.J. Grundon, *pers. comm.*). In these solutions, a concentration of about 1,000 μM P produced adequate growth as long as Zn levels were maintained. With low Zn, chlorotic and necrotic areas were visible on the lower leaves (Plate 6.2, 6.3).

Diagnosis and correction of phosphorus toxicity

We have not found any information on the diagnosis and correction of P toxicity. It would appear best to prevent P toxicity from occurring by ensuring that plants do not become P deficient, and that an adequate Zn supply is maintained in the root environment.

Disorders producing symptoms either on the younger or older leaves

7. Calcium deficiency

Because sunflower is produced mainly on neutral to alkaline soils, calcium (Ca) deficiency is unlikely to be encountered in field-grown crops. Calcium deficiency would be most likely in crops grown on acid soils. However, because of the extreme sensitivity of sunflower to aluminium toxicity, crops grown on acid soils would be more likely to suffer from aluminium toxicity than from Ca deficiency.

Symptoms of calcium deficiency

The first symptom of Ca deficiency encountered is a reduction in stem elongation. At this stage, the leaves appear a normal dark green colour, though less pubescent and more shiny than leaves of plants adequately supplied with Ca. Thereafter, the emerging leaves appear crinkled, and contrast strikingly with leaves formed before stem elongation was reduced (Plate 7.1). The crinkled leaves at the top of the plant and the bracts of the head exhibit interveinal necrosis with little, if any, chlorosis. Another distinct symptom of Ca deficiency also involved the upper leaves. These leaves appear wilted and curled, with a severe bronzing (Plate 7.2).

The lower leaves also develop distinct symptoms of Ca deficiency in addition to those symptoms on the upper, recently emerged leaves. On the lower leaves, tissue breakdown is observed, particularly on the petioles and along the major veins (Plate 7.3). This symptom appears as a dark necrosis.

Bergmann (1986) reported tissue breakdown and death of the upper stem due to Ca deficiency.

Possible confusion with other symptoms

Copper deficiency has been observed to cause leaf crinkling (Plate 14.1, 14.2), a symptom not unlike that caused by Ca deficiency. However, no bronzing nor wilting was observed with copper deficiency. Also, copper deficiency caused extreme reductions in the elongation of the growing point, and the youngest leaves were prominently pubescent.

Deficiency symptoms of Ca and boron, which both affect the growing point and the emerging leaves, may also be confused. However, boron deficiency causes a marked thickening, downward cupping and bronzing of the leaves, which are hard and leathery (Plate 16.2).

The tissue necrosis along the petioles and major veins of the lower leaves may be confused with one of the symptoms of manganese toxicity (Plate 9.3, 9.4). However, with Ca deficiency, there were no small dark brown to black

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Plate 7. Calcium deficiency

7.1 Marked crinkling of the recently emerged leaves as a result of calcium deficiency.

7.2 Wilting and a bronzed necrosis of the recently emerged leaves as a result of calcium deficiency.

7.3 Tissue breakdown, resulting in a dark coloured necrosis on the petioles and major veins of the lower leaves of a calcium deficient plant.

spots associated with the leaf hairs that were evident with manganese toxicity. The tissue necrosis due to Ca deficiency may be confused also with lesions caused by *Alternaria helianthi*.

Diagnostic soil and plant tissue tests

In our studies with cv. Hysun 31 in solution culture, a critical concentration of 1.4% Ca was found in the blade of the youngest expanded leaf of plants at Growth Stage R-2. No studies on the relationship between sunflower growth and soil Ca status have been found in the literature.

Correction of calcium deficiency

Calcium deficiency is unlikely in the neutral to alkaline soils used commonly for sunflower production. In acid soils, aluminium toxicity is likely to be more severe than Ca deficiency. However, should Ca deficiency occur, it may be corrected by applying lime (40% Ca), gypsum (22% Ca), superphosphate (20% Ca) or other Ca fertilizer to the soil.

8. Molybdenum Deficiency

Molybdenum (Mo) deficiency in sunflower has been reported in field-grown crops in the Central Tablelands of New South Wales (McDonald, 1978), in the S.E. of South Australia (McFarlane *et al.*, 1980), and in South Africa (Blamey and Chapman, 1979). This is not particularly surprising since maize (*Zea mays*), which is relatively insensitive to Mo deficiency, has also been found to be Mo deficient in many of the affected areas (Noonan, 1953; Weir *et al.*, 1966; Blamey, 1972).

Symptoms of molybdenum deficiency

As with many other crops, Mo deficiency symptoms become apparent in the early seedling stage (McDonald, 1978; Blamey and Chapman, 1979). Initially, the older leaves exhibit a pale yellow chlorosis. The chlorosis may appear to be uniform over the leaf, although yellowing and mottling may be visible between the veins (McDonald, 1978). As the severity of the symptom increases, the leaves become cupped and necrotic along the leaf margins (Plate 8.1). Agarwala and Sharma (1979) reported that the foliage of Mo deficient plants is pale green with interveinal chlorosis. The young leaves become cupped and the old leaves develop a marginal scorching. The difference between healthy seedlings and those with Mo deficiency is often striking (Plate 8.2). In severe cases the seedlings die, and those that survive are often stunted.

Molybdenum deficient crops often exhibit poor establishment (Blamey, 1972). Also, sunflower crops suffering Mo deficiency have varied from

uniformly affected plants to a patchy appearance. The latter is the more common, even to the extent of one plant being severely affected with the adjacent plant being completely healthy. Crop damage has ranged from very little to complete crop loss (McDonald, 1978). Plants may grow out of the deficiency, particularly in situations where it is not severe.

Possible confusion with other symptoms

Molybdenum deficiency symptoms are striking and characteristic, and are unlikely to be confused with symptoms of nitrogen (Plate 1.1) and sulfur (Plate 11.2) deficiency. Molybdenum deficiency appears early in the growth of the crop, whereas nitrogen and sulfur deficiencies become more severe as the crop develops. Also, neither nitrogen nor sulfur deficiencies result in leaf cupping although the lower leaves of nitrogen deficient plants may develop a marginal necrosis.

Diagnostic soil and plant tissue tests

Molybdenum deficiency is most likely to occur on acid soils since the availability of Mo is reduced at lower soil pH values. However, mild symptoms of Mo deficiency have been recorded at pH > 5.5 (McDonald, 1978).

Molybdenum concentrations in sunflower tissue are extremely low (*c.* 0.1 mg Mo/kg) and are of little use in defining the Mo status of the plant, because of small differences in the Mo concentration between deficient and healthy plants (McDonald, 1978). However, Mo is involved in the enzymatic reduction of nitrate, and high nitrate levels in the plant may be indicative of Mo deficiency. McDonald (1978) reported that healthy sunflower tissue contained 1960 mg NO_3-N/kg whereas Mo deficient tissue contained 12,000 mg NO_3-N/kg.

Correction of molybdenum deficiency

Although Mo deficiency may appear striking, the deficiency is relatively easily corrected. Sodium molybdate may be applied as a fertilizer to the soil, generally as a spray because of the small quantity required, or as a commercially-available mixture with macronutrient fertilizers. McFarlane *et al.* (1980) found that an application of 280 g sodium molybdate/ha increased sunflower seed yields. A rate of 25 g sodium molybdate/25 kg seed has been suggested as a seed dressing (Blamey and Chapman, 1979). Foliar application rates that have proved successful include 280 g sodium molybdate/ha (McDonald, 1978) and 50 g sodium molybdate/100 litres water applied to give good coverage of seedlings (Blamey and Chapman, 1979).

The effect of a foliar spray of sodium molybdate applied to a Mo deficient crop can be spectacular. Sunflower seedlings with Mo deficiency symptoms have been reported to become a healthy green colour within 2 to 8 days of spraying (McDonald, 1978; Blamey and Chapman, 1979).

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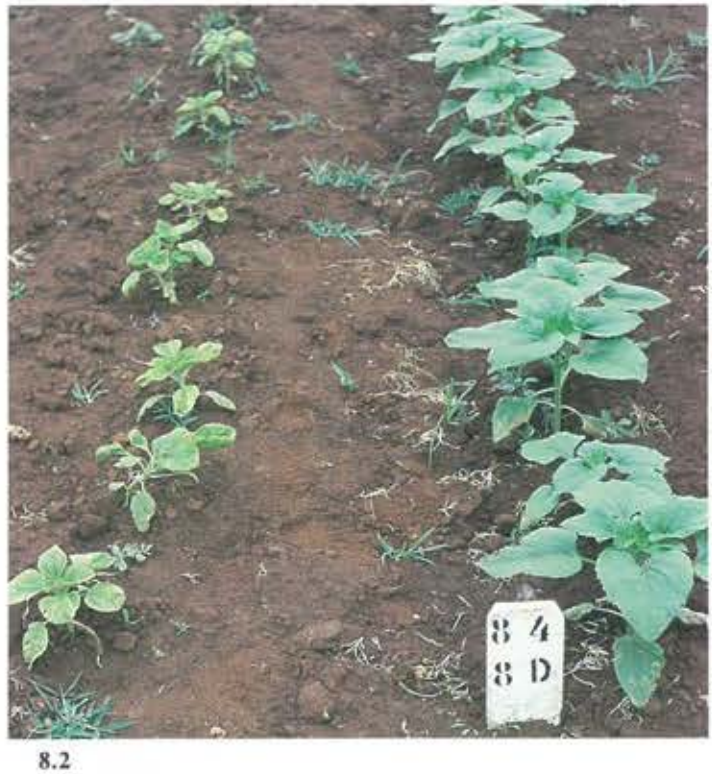

8.2

Plate 8. Molybdenum deficiency

8.1 Chlorosis, accompanied by some necrosis, and upward cupping of leaves of sunflower seedlings as a result of molybdenum deficiency.

8.2 A comparison of healthy sunflower seedlings (R) with those suffering from molybdenum deficiency (L).

9. Manganese Toxicity

Manganese (Mn) toxicity is a particular problem of acid soils and of soils high in easily-reducible Mn that become waterlogged. Crop and pasture species differ markedly in tolerance to high levels of available Mn in soils and nutrient solutions (Foy, 1973; Foy *et al.*, 1978; Edwards and Asher, 1982).

Symptoms of manganese toxicity

The first symptom of excess Mn supply in nutrient solution (30 μM Mn) is the appearance of small, dark-brown to black spots (< 0.5 mm in diameter) on the lower stem and on the petioles and blades of the lower leaves (Plate 9.1, 9.2, 9.3). The spots are not necrotic, and are visibly associated with the trichomes (hairs) on these plant parts. Examination with a hand lens or under a microscope shows that these trichomes are varied in appearance. In some cases, the entire trichome may be blackened; in others, there may be a blackened basal cell or tip cell; in others, cells of the trichome and cells around the base of the trichome may have darkened walls; and in still others, there may be a blackening that spreads from the base of the trichome. The extent of the blackening is not related to the proximity of the trichome to the leaf veins (Plate 9.2).

Electron microprobe analysis has shown that the darkening in and around the trichomes is due to an accumulation of Mn (Blamey *et al.*, 1986c). This suggests that sunflower is able to tolerate high Mn concentrations in the plant by a localization of the excess Mn in a metabolically inactive form in the trichomes.

It is only at much higher Mn concentrations in solution (approx. 300 μM Mn) that other symptoms of excess Mn appear. Irregular, dark-brown, necrotic lesions (> 2 mm in size) develop on the lower leaves, especially along the veins (Plate 9.3, 9.4). These lesions are quite dissimilar to the small, dark coloured spots associated with the trichomes. Also, the upper leaves of the plants develop a striking yellow veinal chlorosis accompanied by leaf crinkling (Plate 9.5). The chlorosis spreads to the interveinal area, and the leaf crinkling becomes severe, followed by the development of large irregular patches of white to buff coloured necrotic tissue (Plate 9.6). These symptoms appear most severe on the rapidly expanding leaves.

Plate 9. Manganese toxicity

9.1 Small, dark spots (manganese accumulations) on the lower stem as a result of excess manganese in solution. Growth of plants with these symptoms alone was not decreased.

9.2 Photomicrograph of darkened trichomes on a lower leaf of a sunflower plant supplied excess manganese in solution.

9.3 Lower leaf showing numerous small dark spots and larger necrotic areas. Growth was reduced by manganese toxicity in this treatment.

9.4 Necrotic lesions on a lower leaf due to manganese toxicity.

9.5 Veinal chlorosis of recently expanded leaves due to manganese toxicity.

9.6 Severe manganese toxicity resulted in chlorosis, necrosis and distortion of the upper leaves.

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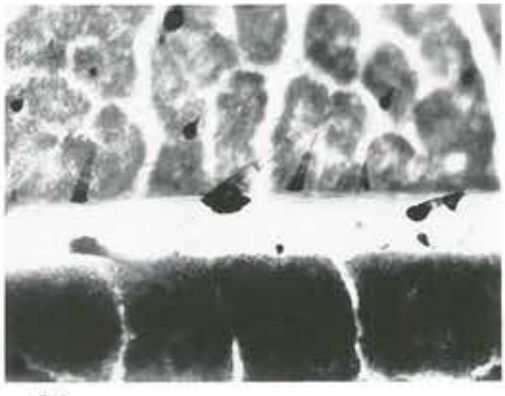

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Possible confusion with other symptoms

Manganese toxicity symptoms in sunflower are quite distinctive, and are difficult to confuse with other known symptoms. At moderate Mn levels (though levels that would be deleterious to the growth of other crops), the dark-brown to black trichomes are distinctive. At higher Mn levels, the veinal chlorosis and leaf crinkling are also characteristic.

The necrotic areas on the petiole and major veins may be confused with one of the symptoms of calcium deficiency (Plate 7.3) which also caused crinkling of the upper leaves (Plate 7.1). Indeed, these symptoms may result from reduced calcium transport to the leaves under conditions of excess Mn supply as has been found in bean plants (Horst and Marschner, 1978). Also, the necrosis may be confused with lesions caused by *Alternaria helianthi* or *Septoria helianthi*.

Diagnostic soil and plant tissue tests

Soil tests for available Mn have been found to be difficult to interpret (Shuman and Anderson, 1974), and no information has been found regarding field-grown sunflower.

Sunflower has been found to be particularly tolerant of Mn toxicity, being the most tolerant of 13 crop and pasture species grown in flowing solution culture with a wide range of constant solution Mn concentrations, viz. 1.3 to 1,160 μM Mn (Edwards and Asher, 1982). Critical concentrations for toxicity (associated with 90% maximum yield) in solution and in whole tops were 65 μM and 5,300 mg Mn/kg, respectively. Corresponding figures for maize, the most sensitive species studied, were 1.4 μM Mn and 200 mg Mn/kg (Edwards and Asher, 1982). Because of the compartmentation of Mn in the trichomes, total Mn concentrations in plant tissue are probably unreliable for diagnostic purposes. Blamey *et al.* (1986c) found that a whole top tissue concentration of 2,205 mg Mn/kg, approximately one half of that reported by Edwards and Asher (1982), was associated with a 10% reduction in plant dry matter yield.

Correction of manganese toxicity

The evidence suggests that the tolerance of sunflower to high Mn concentrations that are lethal to other species (approx. 30 μM Mn) is due to an ability to tolerate high concentrations of Mn in the tops rather than to preventing excess Mn from entering the plants. Because of the tolerance of sunflower to Mn toxicity, it is unlikely that it would be necessary to correct this disorder in field-grown crops. Other crops grown in rotation with sunflower, e.g. maize and wheat (*Triticum aestivum*), would be far more likely to require action to overcome Mn toxicity. However, Mn toxicity often results from low soil pH or from waterlogging. Thus, correction of Mn toxicity would involve the application of lime or dolomite to correct soil acidity or drainage to correct waterlogging.

10. Salinity

Sunflower has been classified as having low salt tolerance. Thus, Fenster *et al.* (1978) recommended that sunflower be grown in soil in which the electrical conductivity (EC) of the saturation extract was less than 2–4 mS/cm. Of the field crops, sunflower was classified as more tolerant to salinity than only soybean, field bean and pea (*Pisum sativum*).

The effect of salinity *per se* on crop growth should not be confused with the consequences of the detrimental effects of high sodium (Na) concentrations on soil properties (e.g. increased bulk density, surface crusting, poor aeration and waterlogging).

Symptoms of salinity stress

The lower leaves of plants grown in nutrient solutions, in which high concentrations of sodium chloride (NaCl) (EC > 7 mS/cm) were maintained, developed a marginal chlorosis (Blamey *et al.*, 1986a). This chlorosis rapidly became necrotic, and the leaves developed a downward cupping (Plate 10.1, 10.2). In addition to the marginal necrosis on the lower leaves, these leaves developed a pale chlorosis that was particularly noticeable in the interveinal areas between the major veins (Plate 10.2). Although growth was reduced in plants grown in solutions with EC > 7 mS/cm (supplied with NaCl), no distinct symptoms were evident on the upper leaves.

Markedly different symptoms developed on plants grown in nutrient solutions in which high concentrations of sodium sulphate (Na_2SO_4) (EC > 4 mS/cm) were maintained. Although symptoms of the disorder first appeared on the lower leaves, both upper and lower leaves were later affected. A chlorosis first developed in the interveinal areas, particularly between the larger veins (Plate 10.3). The chlorosis rapidly became necrotic. Initially, the unaffected areas of the plant remained bright green and the leaves were shiny (Plate 10.4). In solutions in which EC remained constant, the effects of high Na_2SO_4 concentrations became more severe with time, leaves becoming yellow, severely distorted, and necrotic (Plate 10.5).

Possible confusion with other symptoms

Symptoms caused by high salinity, either by excess NaCl or excess Na_2SO_4, are distinctive and little confusion should result with symptoms of other disorders. Although severe symptoms of boron toxicity (Plate 5.2, 5.3) and of NaCl injury are quite different, the early or less severe symptoms of these disorders could be confused (Plate 5.1).

Diagnostic soil and plant tissue tests

Fenster *et al.* (1978) classified sunflower as having low salt tolerance, and recommended that this crop not be grown on soils when the EC of the satura-

tion extract was > 4mS/cm. In solution culture, however, the vegetative growth of sunflower appeared little affected by the addition of NaCl or Na_2SO_4 up to an EC of 4 mS/cm (Blamey *et al.* 1986a). The vegetative growth of sunflower (45 day-old plants) was reduced by 15 and 44% in NaCl solutions with EC values of 7 and 10 mS/cm, respectively. Sunflower was considerably more sensitive to salinity resulting from the addition of Na_2SO_4, vegetative growth being reduced by 42 and 92% in Na_2SO_4 solutions with EC values of 7 and 10 mS/cm, respectively. By comparison, yields of grain sorghum (*Sorghum bicolor*) have been found to decline when the EC of the saturation extract was greater than *c.* 6.5 mS/cm (Francois *et al.*, 1984).

Germination of sunflower seed was reduced by 11% in solutions of NaCl or Na_2SO_4 with an EC of 10 mS/cm (Blamey *et al.*, 1986a). In salt-affected soil, the emergence of seed differed among cultivars (Karami, 1974). Emergence varied from 68 to 96% of that in soil without added NaCl at an EC of the saturation extract of 4.4 mS/cm, and from 33 to 72% at an EC of 7.6 mS/cm. Chhabra *et al.* (1979) found that an exchangeable sodium percentage (ESP) > 16 delayed the germination of sunflower seed and decreased plant height. Seed yield was significantly reduced by 14% at ESP > 25, but at this value, oil concentration was not affected.

Chhabra *et al.* (1979) found that a decrease in seed yield was associated with 0.4% Na in the upper leaves at maturity. Although Na_2SO_4 was more toxic to sunflower than NaCl in our studies, the leaf Na levels associated with a 10% yield reduction were higher with Na_2SO_4 (1.8%) than with NaCl (0.9%). Hence, the associated anions Cl and SO_4 appear to play an important role in salt injury.

Plate 10. Salt injury in sunflower

10.1 Marginal chlorosis and necrosis and interveinal chlorosis as a result of excess sodium chloride in solution.

10.2 Sunflower seedling grown in a nutrient solution containing excess sodium chloride.

10.3 Interveinal chlorosis and necrosis of a lower leaf resulting from excess sodium sulphate in solution.

10.4 Severe necrosis and distortion of the upper leaves due to excess sodium sulphate in solution.

10.5 Very severe chlorosis, necrosis and leaf distortion in sunflower due to excess sodium sulphate in solution.

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Disorders producing symptoms mainly on the younger leaves

11. Sulfur Deficiency

Dicotyledonous plants generally have a higher sulfur (S) requirement than monocotyledons. However, no instances of S deficiency in field-grown sunflower have been reported. This is in spite of S deficiency occurring in other crops in sunflower producing areas. For example, Chisholm and Dowling (1985) reported low reserves of S in soils of the Darling Downs of Queensland, an important sunflower producing area, which resulted in S deficiency in winter cereals and soybean. However, S deficiency has not been reported in sunflower growing on these soils.

Symptoms of sulfur deficiency

In many crop species, S deficiency is characterized by leaves that are pale green to yellow. In general, S deficiency symptoms are either spread rather uniformly over all leaves of the plant or are most noticeable on the younger leaves.

McLachlan (1978) and Platou and Irish (1982) described symptoms of S deficiency in sunflower as a general chlorosis of younger leaves while the older leaves remained green. Because of the high requirement for S, deficiency symptoms may become evident early in the growth of sunflower plants (Plate 11.1). In this case, the cotyledons may remain a healthy green, with the younger leaves being pale green in colour. In older plants, the pale green chlorosis is generally spread rather uniformly over all leaves with growth being severely restricted by S deficiency (Plate 11.2). The chlorosis usually is uniform over the whole leaf although darker green areas may be evident, randomly distributed in areas pale green in colour. Also, the chlorosis may take on a mottled appearance (Plate 11.3).

Possible confusion with other symptoms

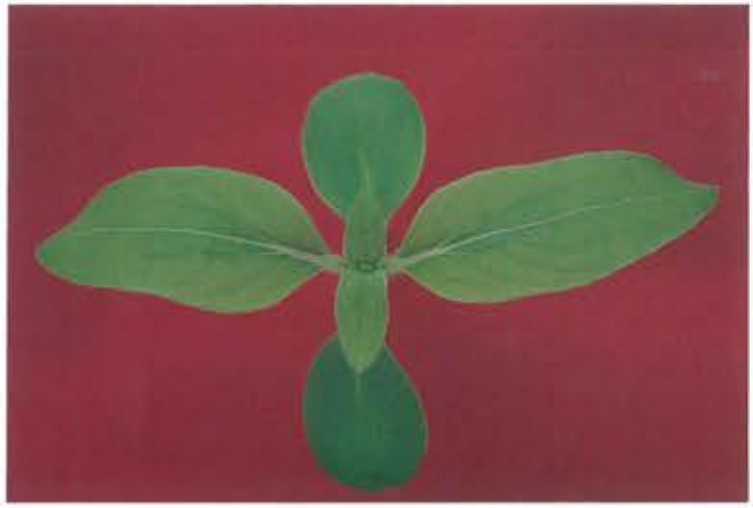
As with many other crop species, S deficiency may be confused easily with nitrogen deficiency (Plate 1.1, 1.2). In general, however, nitrogen deficiency

Plate 11. Sulfur deficiency

11.1 Chlorosis of the youngest leaves (while the cotyledons remain dark green) due to sulfur deficiency in a young sunflower seedling.

11.2 General chlorosis of sunflower plants in nutrient solution culture (L) due to sulfur deficiency compared with healthy plants adequately supplied with sulfur (R).

11.3 Slightly mottled chlorosis due to sulfur deficiency (R) compared with a healthy leaf (L) (Source: N.J. Grundon).



11.1

11.2

11.3

symptoms tend to be more prominent on the older leaves, since nitrogen is rapidly withdrawn from these leaves under conditions of deficiency and redistributed via the phloem to young, actively-growing parts. Bukovac and Wittwer (1957) showed that S also was mobile in the phloem, but considerably less so than nitrogen.

Since molybdenum deficiency also may produce a generally chlorotic plant, a shortage of molybdenum may be confused with S deficiency. However, unlike S deficiency, molybdenum deficiency produces marginal necrosis and upward cupping of leaves (Plate 8.1, 8.2). Also, with molybdenum deficiency, the oldest leaves are more severely affected.

Diagnostic soil and plant tissue tests

A high proportion of Australian soils have low total S levels (40–200 mg/kg), the exception being soils of basaltic origin (e.g. black earths and krasnozems with 105—1,100 mg/kg total S) (Williams and Raupach, 1983). These latter soils have high S levels largely due to high organic matter content and in some cases retention of sulfate by adsorption. However, many soils of the Darling Downs have been continuously cropped for up to 70 years with a consequent marked reduction in organic carbon (Dalal and Mayer, 1986). In these situations, S deficiency might be expected (Chisholm and Dowling, 1985). A further factor in increasing the possibility of S deficiency has been the increasing use of high analysis fertilizers low in S (e.g. triple superphosphate with 2% S).

Although soil tests for plant-available S have been available for many years (Reisenauer *et al.*, 1973), no interpretation of these results has been proposed for sunflower.

Robinson (1970) found a S concentration of 0.36% in whole sunflower plants at heading that showed no symptoms of S deficiency. In our solution culture experiments with cv. Hysun 31, maximum growth of sunflower was found with 0.44% S in the youngest expanded leaf at Growth Stage R-2. With less than 0.40% S, plant dry matter yield was reduced by more than 10%.

Correction of sulfur deficiency

Sulfur deficiency may be corrected most easily by the addition of S-containing fertilizers. These fertilizers may be applied specifically to add S to the soil, e.g. elemental S or gypsum (19% S). Additionally, S is added to the soil along with other fertilizers, e.g. ammonium sulphate (24% S) and single superphosphate (11% S), the application of which is not primarily aimed at correcting S deficiency. Indeed, in Australia the occurrence of S deficiency has been restricted by the widespread use of single superphosphate to correct phosphorus deficiency (Williams and Raupach, 1983).

12. Iron Deficiency

The availability of iron (Fe) to plants decreases with increase in soil pH, and Fe deficiency most commonly occurs in crops grown on neutral to alkaline soils. Most of the world's sunflower is produced on such soils, but no report of Fe deficiency in field-grown sunflower has been found, although Alcantara and de la Guardia (1986) reported that some plants from two hybrids were chlorotic when grown in calcareous soil. In sensitive crops, e.g. sorghum and soybean grown on neutral to alkaline soils, severe yield losses have been reported due to Fe deficiency (Clark, 1982). Mathers *et al.* (1980) suggested that sunflower could be an alternative crop on Fe-deficient soils.

Symptoms of iron deficiency

The initial symptom of Fe deficiency in young seedlings is a pale, mainly interveinal, yellow chlorosis of the younger leaves (Plate 12.1). In older plants, there is a characteristic and distinct interveinal chlorosis of the younger leaves, with the youngest leaves being pale yellow to white (Plate 12.2). In severely Fe deficient plants, the pale yellow interveinal chlorosis rapidly becomes necrotic, and the leaves become severely distorted (Plate 12.3, 12.4). The necrosis is a pale buff colour.

In solution culture, it was difficult to maintain symptoms of a given severity since the leaves either died if Fe deficiency was too severe, or recovered if small amounts of FeEDTA were added. Spontaneous recovery occurred through marked reduction in solution pH by Fe deficient plants which probably caused dissolution of previously precipitated Fe.

Roots of sunflower plants grown in Fe deficient nutrient solutions develop characteristic symptoms as described by Romheld and Marschner (1981). With severe Fe deficiency, roots appear stunted and the root tips swollen. With slight alleviation of Fe deficiency, the swelling is often located in the region slightly behind the root tip (Plate 12.5). Romheld and Marschner (1981) reported that sometimes several swollen parts occur on the same root axis.

Possible confusion with other symptoms

Iron deficiency symptoms in sunflower are distinctive, but may be confused with symptoms caused by zinc toxicity (Plate 17.1) which may in fact be due to zinc-induced Fe deficiency. However, with Fe deficiency, no necrosis was observed in the region of petiole attachment to the stem as with zinc toxicity (Plate 17.2). The early stage of Fe deficiency in young seedlings may be confused with sulfur deficiency (Plate 11.1).

Diagnostic soil and plant tissue tests

Sunflower has been found to be an Fe-efficient plant, in which morphological and physiological changes occur in plant roots in response to Fe stress (Romheld and Marschner, 1981; Romheld *et al.*, 1982). In particular, Fe deficiency increased root diameter and increased the reducing capacity of sunflower roots, resulting in conversion of insoluble Fe^{3+} to the soluble Fe^{2+} form. In addition, the roots acidify their environment which further aids Fe availability. Landsberg (1982) reported a rapid acidification of nutrient solution, from approximately pH 6.7 to 4.0, when the youngest leaves of sunflower plants started exhibiting Fe deficiency symptoms. Alcantara and de la Guardia (1986) showed that genetic variability exists in sunflower in the capacities of roots to lower solution pH and to reduce Fe^{3+}. Plants without these characteristics developed Fe deficiency symptoms.

In spite of the widespread production of sunflower on neutral to alkaline soils prone to Fe deficiency, no reports of Fe deficiency in crops grown on these soils have been found. Thus, no diagnostic soil tests are available. Also, because of the difficulty of assessing biologically-active Fe in plant tissues (total Fe in the plant is of little value), no plant tissue tests have been reported.

Correction of iron deficiency

No reports on the correction of Fe deficiency in sunflower have been found. Also, because of the ability of sunflower roots to improve conditions for Fe uptake, it is unlikely that Fe deficiency would occur in field-grown crops except under unusual conditions.

Plate 12. Iron deficiency

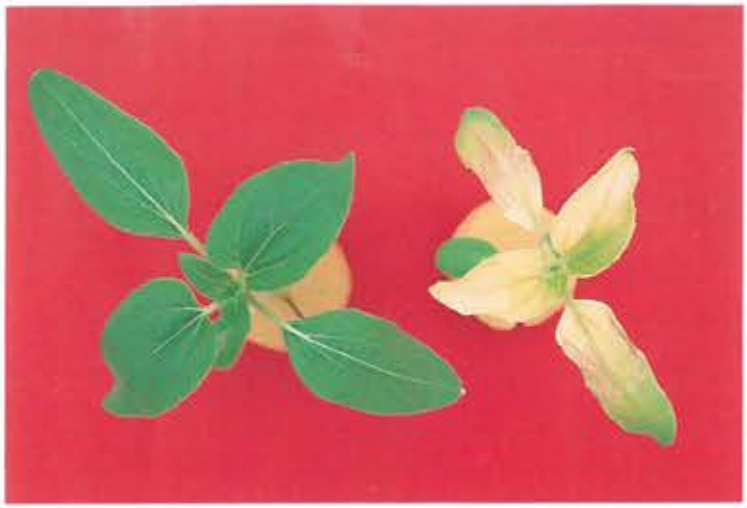
12.1 Severe iron deficiency symptoms on a sunflower seedling (R) with a pale chlorosis and necrosis of upper leaves in comparison with a healthy seedling (L).

12.2 Pale interveinal chlorosis of upper leaves due to iron deficiency.

12.3 Upper leaf from a healthy plant (L) compared with those from plants with increasingly severe iron deficiency (R).

12.4 Severe iron deficiency resulting in a pale chlorosis and leaf necrosis.

12.5 Swollen tips of sunflower roots in response to iron deficiency.



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13. Manganese Deficiency

Manganese (Mn) deficiency is most likely to occur in soils that are low in plant-available Mn, that are alkaline or have been limed. However, there appear to have been no reports of Mn deficiency in field-grown sunflower.

Symptoms of manganese deficiency

The first symptom of Mn deficiency in solution-grown sunflower plants is the appearance of small (< 2 mm) chlorotic spots on recently-expanded leaves. This symptom has been reported also by Bergmann (1986) as being due to Mn deficiency. Neither the older leaves nor the leaves close to the growing point are affected. The small chlorotic spots were generally in the interveinal area of the leaves. As the deficiency of Mn became more severe, the chlorotic spots beame necrotic (Plate 13.1). These necrotic spots were pale brown in colour and tended not to coalesce. Also, in severe cases, symptoms have been reported to spread to the young and old leaves (Agarwala and Sharma, 1979).

Possible confusion with other symptoms

The small chlorotic and necrotic spots caused by Mn deficiency are characteristic of the disorder, and should not be confused with symptoms of other disorders.

Diagnostic soil and plant tissue tests

No reports of soil tests associated with Mn deficiency were found. In sunflower (cv. Hysun 31) grown in solution culture, the critical concentration was 40 mg Mn/kg in the youngest expanded leaf. Agarwala and Sharma (1979) reported that 60 mg Mn/kg in the leaves of sunflower was adequate for growth, and that < 15 mg Mn/kg was associated with visible symptoms of Mn deficiency.

Correction of manganese deficiency

Manganese deficiency in sunflower could be corrected either by soil or foliar applied Mn fertilizers. For other crops, soil applied rates of 34 to 68 kg manganese sulphate/ha have been used, and foliar applied rates have ranged from 5.6 to 11.2 kg manganese sulphate/ha (Tisdale and Nelson, 1966). Since Mn deficiency may be induced by overliming, it is necessary to ensure that soils low in Mn not be limed above pH 6.4 (Tisdale and Nelson, 1966).

In the USSR, Semikhnenko *et al.* (1973) (according to Robinson 1978) reported that foliar applications of Mn 10 days after flowering increased sunflower yields.

13.1

Plate 13. Manganese deficiency
13.1 Small, pale chlorotic and necrotic spots caused by manganese deficiency.

14. Copper Deficiency

No reports of copper (Cu) deficiency in field-grown sunflower have been found. Copper deficiency has been reported in wheat, oats (*Avena sativa*) and barley (*Hordeum vulgare*) growing on the Darling Downs of Queensland (Grundon and Best, 1982; Grundon *et al.*, 1985), but there are no reports of Cu deficiency in sunflower. However, in Queensland, Cu deficiency is primarily found in winter cereals grown on soils of the Eastern Brigalow belt (Western Downs), whereas sunflower is grown primarily in the Eastern Uplands.

Symptoms of copper deficiency

In solution culture, Cu deficiency first becomes apparent as a decrease in stem elongation. As the severity of the deficiency increases, this decrease in stem elongation becomes most striking so that the growing point appears considerably below the earlier, expanded leaves (Plate 14.1). The expanded leaves remain dark green and shiny, and develop an upward cupping. The young leaves emerging from the growing point are severely crinkled and thickened. Also, these leaves are prominently pubescent, having a grey-green appearance (Plate 14.2). In our studies, no chlorosis or necrosis was evident on any of the leaves, but Shorrocks and Alloway (1986) noted that the newly emerging leaves are often necrotic at the tips. Bergmann (1986) and Shorrocks and Alloway (1986) also reported that the petioles of Cu deficient sunflower plants were reflexed (i.e. pointed downwards). The roots of Cu deficient plants are extensively branched (Plate 14.3).

Possible confusion with other symptoms

Symptoms caused by deficiencies of calcium (Plate 7.1) and Cu could be confused because deficiencies of both nutrients restrict stem elongation and cause leaf crinkling. However, Cu deficiency caused no petiole or vein necrosis, a symptom evident on the lower leaves of calcium deficient plants (Plate 7.3). Also, leaf pubescence was prominent in Cu deficient plants in contrast with the rather shiny emerging leaves of calcium deficient plants. Crinkling of upper leaves is also caused by manganese toxicity that, unlike Cu deficiency, is accompanied by leaf chlorosis and necrosis (Plate 9.6).

Root symptoms caused by Cu deficiency could be confused superficially with those caused by aluminium toxicity (Plate 19.2), since both disorders cause extensive root branching. However, unlike Cu deficiency, aluminium toxicity causes discoloration and thickening of the roots.

Diagnostic soil and plant tissue tests

No information has been found on soil tests associated with Cu deficiency in sunflower. In our studies, a critical concentration of 3 mg Cu/kg was established in the youngest expanded leaf of sunflower (cv. Hysun 31) grown in solution culture.

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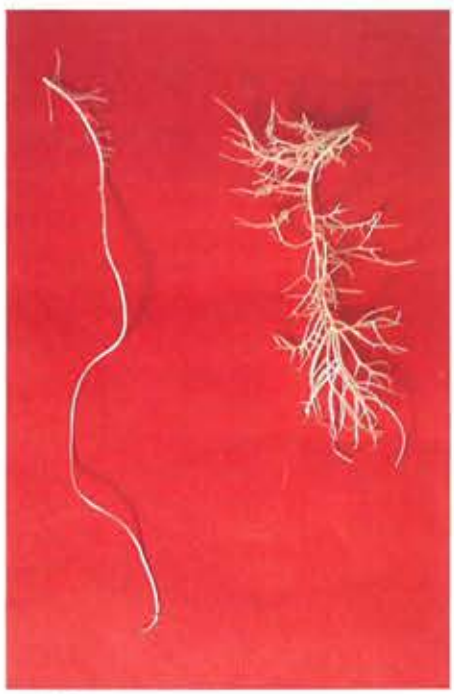
14.2



14.3

Plate 14. Copper deficiency

14.1 With copper deficiency there is a marked decrease in stem elongation so that recently expanded leaves (which may be cupped upwards) appear above the growing point.

14.2 In plants with copper deficiency, young leaves emerging from the growing point are crinkled and thickened, prominently pubescent and have a grey-green colour.

14.3 In solution culture, the roots of plants with copper deficiency are extensively branched (R) in comparison with those of plants adequately supplied with copper (L).

Correction of copper deficiency

Copper deficiency in sunflower could be corrected by either soil or foliar applications of Cu fertilizers. However, since no reports of Cu deficiency in field-grown sunflower have been found, the rates that should be applied are not known. For other crops on mineral soils, a single application of 1 kg Cu/ha on acid or sandy soils to 7 kg Cu/ha on alkaline or heavy-textured soils has been sufficient (Reuther and Labanauskas, 1966). Higher rates may be needed on organic soils.

In wheat, foliar applications of 0.13 to 0.25% Cu as copper sulphate have proved satisfactory (N.J. Grundon, *pers. comm.*). Applications need to be timed to allow proper vegetative development and to coincide with the period of pollen formation. Leaf scorch in ginger (*Zingiber officinale*) caused by foliar Cu applications has been prevented by the addition of calcium hydroxide (0.5%) to the copper sulphate solution (Asher and Lee, 1975). Soils with marginal Cu status should not be limed to pH >6.4 since the availability of Cu decreases with increased pH. High rates of Cu fertilizers should be avoided because of possible problems with Cu toxicity.

15. Zinc Deficiency

No reports of zinc (Zn) deficiency in field-grown sunflower have been obtained.

Symptoms of zinc deficiency

Zinc deficient plants are shorter than those receiving adequate Zn, but the growing point remains higher than the recently expanded leaves. The youngest leaves are narrow and the leaf margin is wavy (Plate 15.1). As the deficiency intensifies, the recently expanded leaves become hard and leathery. With severe Zn deficiency, there is a sudden wilting and collapse of the upper leaves (Plate 15.2). This symptom of Zn deficiency is probably associated with the important role of Zn in maintaining membrane integrity (Welch *et al.*, 1982). Accompanying the severe leaf wilting, brown necrotic spots (approx. 5 mm in diameter) appear, and a grey-green necrosis is observed over a large part of the leaf.

Plate 15. Zinc deficiency

15.1 The youngest leaves of plants with zinc deficiency are narrow and the leaf margin is wavy.

15.2 With severe zinc deficiency, there is a rapid wilting and collapse of upper leaves accompanied by interveinal bronzing.

15.3 The roots of zinc deficient plants (M,L) are thickened and have short, 'spikey' laterals in comparison with roots of plants adequately supplied with zinc (R).

15.1

15.2

15.3

Agarwala and Sharma (1979) reported that the margins and apices of recently-expanded leaves of Zn deficient plants are discoloured, and that necrotic spots in the interveinal areas coalesce to form large necrotic patches. Also, Zn deficient plants develop flower buds earlier than healthy plants, but the buds fail to develop normally and any seed produced is shrivelled.

The roots of plants grown in Zn-deficient solution culture are severely affected. In addition to reduced growth, the roots are thick with many, short, 'spikey' laterals (Plate 15.3).

Possible confusion with other symptoms

Symptoms caused by Zn deficiency are distinctive, and should not be confused easily with symptoms of other disorders. The hard, leathery leaves could be confused with boron deficiency (Plate 16.2). However, in Zn deficiency the affected leaves are those that have recently expanded whereas in boron deficiency, it is the younger, expanding leaves that are hard, leathery and exhibit bronzing. Also, no wilting of the young leaves has been observed in boron deficient plants.

The severe wilting of the upper leaves could be confused with the effects of moisture stress. Also, plants infected with *Sclerotinia sclerotiorum* (Lib.) (Sclerotinia wilt and head rot) and some cultivars infected with *Verticillium dahliae* (Verticillium wilt) may show rapid wilting (Zimmer and Hoes, 1978). Zinc deficiency may be distinguished from these biotic diseases by the absence of causal organisms.

Diagnostic soil and plant tissue tests

Since no reports of Zn deficiency of sunflower grown in soil (field or glasshouse) have been found, no diagnostic soil tests are available. In our solution culture experiments with cv. Hysun 31, a concentration of 14 mg Zn/kg in the youngest expanded leaf has been found to be associated with a 10% reduction in vegetative growth.

Correction of zinc deficiency

Should Zn deficiency be found in field-grown crops, this disorder may be corrected by the application of Zn fertilizers. The rate of application should be in the range 2.7 to 5.4 kg Zn/ha (as zinc sulphate monohydrate), which is commonly used for the field crops (Chapman, 1966). Applications at the lower rates would be for soils that are acid or sandy while the higher rates would be for those that are alkaline or heavy textured. Foliar fertilization with 0.5% zinc sulphate heptahydrate solution (with 0.25% calcium hydroxide) has also been successful on other field crops (Asher and Lee, 1975). If the Zn status of the soil is low, the soil should not be limed to pH > 6.5, since the availability of Zn decreases with increased pH.

Cultivar differences in sensitivity to Zn deficiency have been reported (Agarwala and Sharma, 1979).

16. Boron Deficiency

It has long been known that the sunflower plant is particularly sensitive to boron (B) deficiency. Indeed, this characteristic of sunflower has been used to test soils for B status (Schuster and Stephenson, 1940). In contrast, it is only relatively recently that B deficiency in field-grown sunflower in South Africa has been recognized as being of economic significance (Blamey, 1976; Blamey *et al.*, 1979; Birch *et al.*, 1981; Armstrong and McGee, 1982; Blamey and Chapman, 1982). Sunflower cultivars have been found to differ greatly in B status in plant tissue (Blamey *et al.*, 1980), and in sensitivity to B deficiency (Blamey *et al.*, 1978a). These differences were found to have considerable economic implications. Under conditions of B deficiency, B fertilization increased seed yields of one cultivar by 49% but increased yields of another by only 11.3%. In Australia, Haddad and Kaldor (1984) reported B deficiency in sunflower grown in soils low in plant-available B from the Central Tablelands of New South Wales. Fernandez *et al.* (1985) reported B deficiency in sunflower in Spain. Boron deficiency is widespread in sunflower in Bulgaria, and Stoyanov (1985) estimated that 35% of the crop is fertilized with B. Boron deficiency has also been recorded in sunflower grown in the Chiang Mai region of Thailand (Rerkasem, 1986).

Symptoms of boron deficiency

Boron deficiency produces a number of characteristic symptoms in sunflower that are not easily confused with other nutritional disorders. In very severe cases of B deficiency, the emerging seedling may fail to develop beyond the expansion of the cotyledons (Shkolnik, 1984) (Plate 16.1). Root elongation also ceases or is decreased, in keeping with the finding that B is essential for apical cell division in roots (Cohen and Lepper, 1977). In solution culture, the roots appear excessively branched and brittle.

In general, it is likely that symptoms of B deficiency on plant tops would first appear around the time of flowering (Blamey, 1976), although seedlings with fewer than 8 leaves have been found to exhibit leaf symptoms of B deficiency in solution culture, in pot culture (Bergmann, 1986) and in the field. The upper leaves become hardened, malformed, and necrotic and may have a bronze colour (Plate 16.2, 16.3). Agarwala and Sharma (1979) reported that B deficient sunflower plants exhibit collapse and necrosis of the apex, the internodes are short and the young leaves show basal fading. Symptoms on older leaves include patchy discoloration, water soaked areas and necrosis, the leaves being thick and leathery. Since B is passively translocated in the transpiration stream, symptoms of B deficiency may become more severe during times of moisture stress. Leaves produced after the relief of the moisture stress may not show symptoms of B deficiency (Plate 16.4).

In addition to leaf symptoms, B deficiency may result in a corky appearance of the stem (Plate 16.5) and peduncle. This has considerable economic implications since the stems and peduncles are brittle and may break

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16.7

Plate 16. Boron deficiency

16.1 Failure of seedling development due to severe boron deficiency.

16.2 Boron deficiency causes the recently emerged leaves to be malformed and leathery. The recently expanded leaves have a bronze colour and are also hard and thick.

16.3 With severe boron deficiency, sunflower leaves are thick, necrotic and deformed.

16.4 The field-grown sunflower plant in the foreground has a group of leaves with boron deficiency symptoms in the mid-stem region. These leaves formed during a period of water stress. The leaves that formed after the relief of water stress show no symptoms (Source: J.W. Snyman).

16.5 Boron deficiency may result in a plant with a corky and brittle upper-stem and peduncle.

16.6 Head deformation resulting from boron deficiency.

16.7 Malformation of the head and poor seed set in sunflower resulting from boron deficiency.

resulting in severe yield reduction. Fernandez *et al.* (1985) reported that this symptom was severe in some sunflower crops in Spain, and Rerkasem (1986) reported the occurrence of this symptom in Thailand.

Further symptoms of B deficiency are observed during the reproductive stage. The head is often deformed (Plate 16.6), and in some cases ray florets or bracts grow in the middle of the head (Blamey, 1976; Bergmann, 1986). This results in severe yield loss due to poor seed set. Also, even when malformation of the head is not severe, areas of the head may produce no seed (Plate 16.7). Head deformity has been found to be closely related to B concentration in the upper leaves (Blamey *et al.*, 1978b).

Possible confusion with other symptoms

In the vegetative stage, B deficiency symptoms may be confused with calcium (Plate 7.1) or copper (Plate 14.2) deficiency symptoms, since deficiencies of these nutrients affect the meristem and upper leaves. Both B and calcium are immobile nutrients, and affect the growing point of the plant. However, unlike B deficiency, calcium deficiency causes leaf crinkling and wilting of the upper leaves. In addition, calcium deficiency causes tissue breakdown in the petioles of the lower leaves. Copper deficiency results in plants with expanded leaves higher than the growing point but there is no leaf bronzing or necrosis.

In the reproductive stage, B deficiency may be confused with damage caused by sunflower midge (*Contarinia schulzi* Gagne) (Schulz, 1978), since this pest results in gnarled heads. Indeed, any mechanical damage to the developing head may cause symptoms similar to the deformed head caused by B deficiency (Palmer and Marc, 1982).

Diagnostic soil and plant tissue tests

Since sunflower is extremely sensitive to B deficiency, it is expected that high levels of plant available B would be required for adequate growth. Fernandez *et al.* (1985) reported that no symptoms were found in sunflower growing in soils with $>$ 0.26 mg/kg hot-water soluble (HWS) B. Recently, Cartwright *et al.* (1983) suggested the use of 0.01 M $CaCl_2$ + 0.05 M mannitol as a more convenient extractant for estimating plant-available B. This method was highly correlated with the HWS method.

Critical B concentrations have been reported for a number of sunflower cultivars, and the evidence suggests limited variation exists among cultivars, including those differing markedly in sensitivity to B deficiency. Blamey *et al.* (1979) established a critical B concentration of 34 mg/kg in the YEL at flowering of two field-grown sunflower cultivars. Seed yield was reduced by an average 1.5% for each 1 mg B/kg reduction below this critical concentration. Research on two cultivars differing markedly in sensitivity to B deficiency (Blamey and Chapman, 1982) indicated a similar critical concentra-

tion. Bergmann (1986) reported that the upper, fully-developed leaves of sunflower plants, adequately supplied with B, contained 35 to 100 mg B/kg while those that were severely deficient contained 10 to 13 mg B/kg. Fernandez *et al.* (1985) reported that abnormal head fall due to neck (peduncle) break decreased in sunflower with > 34 mg B/kg in the top mature leaf. Stoyanov (1985) reported that fertilization with B was necessary in sunflower with B concentration < 26 mg/kg.

Correction of boron deficiency

Although sunflower is particularly sensitive to boron deficiency, it is relatively easy to correct this disorder. In South Africa, fertilization with 1 kg B/ha on sandy soils and 3 kg B/ha on clay soils has been found to adequately overcome B deficiency (Blamey *et al.*, 1979), even in sensitive cultivars. In Bulgaria, Stoyanov (1985) recommended the application of 1 to 4 kg B/ha, depending on soil type, to correct the problem. Alternatively, two foliar sprays of 0.4 to 0.5 kg B/ha, applied at the beginning of flowering and during the main period of flowering, have been recommended (Stoyanov, 1985).

Recently, it has been found that it is possible to breed sunflower cultivars efficient in B uptake (Blamey *et al.*, 1984). Also, Jodice *et al.* (1981) found that infection of sunflower roots with vesicular arbuscular mycorrhizae increased B uptake from a B-deficient soil.

Considerable research has shown that B deficiency may be induced by liming acid soils low in plant-available B (Wear and Patterson, 1962; Gupta and Cutcliffe, 1972). In contrast with this research, however, Blamey and Chapman (1982) and Haddad and Kaldor (1984) found that liming had no detrimental effect on B uptake.

17. Heavy Metal and Selenium Toxicities

Although rare, toxicities of the heavy metals, cadmium (Cd), cobalt (Co), chromium (Cr), lead (Pb), nickel (Ni), and thallium (Tl) may result from industrial pollution or the application of sewage sludge to agricultural land. Overfertilization with zinc (Zn) or copper (Cu) fertilizers may also cause problems. Sometimes, toxicities of heavy metals and selenium (Se) occur on soils derived from particular parent materials. Acidification of soils with marginal levels of heavy metals may result in toxicities through increased availability to the plant.

No reports have been found of heavy metal toxicities nor of selenium (Se) toxicity in field-grown sunflower.

Toxicity symptoms

Bazzaz *et al.* (1974) found that the primary mode of action of high concentrations of heavy metals was an interference with stomatal function. Thus, wilting is a common symptom of heavy metal toxicity.

17.1

17.2

17.3

17.4

Plate 17. Heavy metal and selenium toxicity

17.1 Interveinal chlorosis of recently expanded leaves caused by zinc toxicity.

17.2 Necroses at the point of petiole attachment to the stem caused by zinc toxicity.

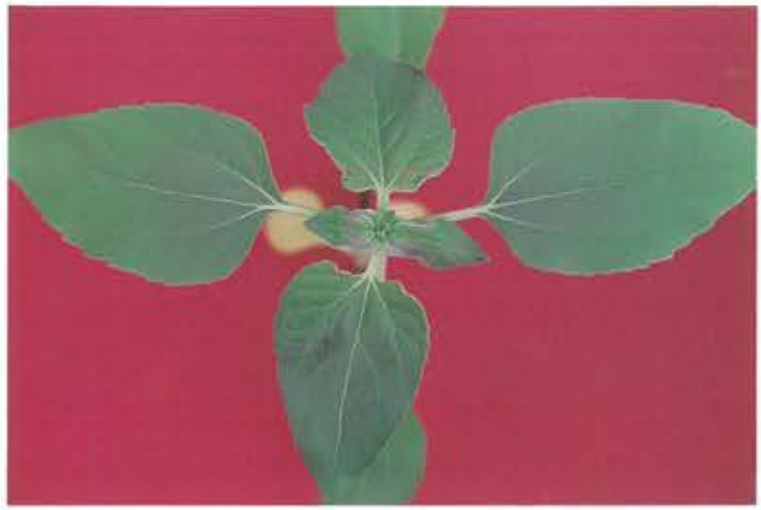
17.3 Marginal chlorosis and upward cupping of leaves from plants with cobalt toxicity.

17.4 Chlorosis and necrosis of areas close to major veins resulting from cobalt toxicity.

17.5 Grey colour and distortion of youngest leaves due to chromium toxicity.

17.6 Interveinal chlorosis of upper leaves due to selenium toxicity.

17.7 Distortion of upper leaves due to selenium toxicity.



17.5

17.6

17.7

Zinc toxicity in sunflower first appears as a light-yellow interveinal chlorosis on the upper, rapidly-expanding leaves (Plate 17.1). The youngest leaves remain green. The interveinal chlorosis may appear on only part of a leaf, or may be of different severity on different parts of the same leaf. The veins remain a striking green colour in contrast to the interveinal area. No necrosis was evident on the leaves of plants in our experiments.

However, a further characteristic symptom of Zn toxicity was the development of brown necroses around the area where the petiole is attached to the stem (Plate 17.2). The necrosis may be localized and may also spread down the stem from the petiole attachment. The necrosis was commonly found approximately halfway up plants suffering Zn toxicity.

In nutrient solutions, high concentrations of Co caused an upward leaf cupping and a distinct chlorosis that is particularly evident towards the edge of the leaf (Plate 17.3). The chlorosis also appeared in a narrow band adjacent to the major veins. These symptoms have been observed mainly on the expanding leaves midway up the plant. On the upper leaves, Co toxicity caused a characteristic interveinal chlorosis followed by a buff-coloured necrosis along the veins or a grey-green necrosis in the interveinal areas (Plate 17.4).

Chromium toxicity was found to cause the older leaves of sunflower plants to become dark green. The youngest leaves developed a grey coloration and a distortion towards the base (Plate 17.5).

The addition of Ni to solution culture caused severe wilting and death of sunflower seedlings.

In spite of vegetative yields being reduced by 50%, no symptoms of Cu toxicty were evident. Plant height was greatly reduced by Cu toxicity, but leaves, stems and roots appeared healthy.

Selenium toxicity caused a distinct interveinal chlorosis spread over the whole leaf surface (Plate 17.6.) A certain amount of leaf crinkling was evident, though this was not severe. In more severe cases of Se toxicity, the older leaves had a grey-green colour, and the upper leaves were distorted (Plate 17.7). The distortion was accompanied by necrosis along the leaf margins and towards the ends of the major veins. Sharma and Gangwer (1985) described Se toxicity in sunflower as chlorosis, cupping and stunting, the severity increasing with increased Se added to the soil and decreasing with added sulfur.

Possible confusion with other symptoms

Severe cases of heavy metal or Se toxicity may be difficult to diagnose without plant analysis because many of these disorders result in a grey-green or bluish colour of the foliage. Also, high concentrations of many heavy metals result in wilting (Bazzaz *et al.*, 1974). However, milder cases are more easily diagnosed on the basis of symptoms.

Cobalt toxicity may be confused with molybdenum deficiency (Plate 8.1, 8.2), except that Mo deficiency can cause the whole plant to become chlorotic.

Leaf symptoms of Zn toxicity may be confused with those caused by iron deficiency (Plate 12.3). In the latter case, however, in addition to the yellow, interveinal chlorosis, the chlorosis became almost white, and severe necrosis occurred. This differed from the observed symptoms of Zn toxicity. Also, no stem necrosis was observed with iron deficiency.

Correction of heavy metal toxicities

The prevention (rather than correction) of heavy metal toxicities should be the aim, since these toxicities generally result from human action. Liming (to reduce availability) may prove effective in alleviating heavy metal toxicities.

Bazzaz *et al.* (1974) reported a 50% reduction in the photosynthesis of sunflower leaves with heavy metal concentrations of 96 mg Cd/kg, 79 mg Ni/kg, 193 mg Pb/kg, or 63 mg Tl/kg. Sharma and Gangwer (1985) found that sunflower dry matter yield was reduced by 47% when the Se concentration in the tops increased from 3.8 to 32.8 mg/kg.

In our solution culture study with cv. Hysun 31, growth was reduced by 10% with a Zn concentration of 210 mg/kg in the youngest expanded leaf. The critical concentration for deficiency was 14 mg Zn/kg.

Also with cv. Hysun 31, a critical concentration (90% maximum yield) of 85 mg Cu/kg in the youngest expanded leaf was found. Since the critical concentration for deficiency is about 3 mg Cu/kg in the youngest expanded leaf, it would appear that sunflower is relatively tolerant of excess Cu.

Role of soil pH in sunflower nutrition

Potential Problems in Acid Soils

18. Low pH

The liming of acid soils to increase soil pH has been an agricultural practice for centuries. However, the reasons for poor growth on acid soils are not always clear, and in all probability vary from site to site (Vlamis, 1953; Coleman *et al.*, 1958). It is generally accepted that there are two main causes of poor plant growth in acid soils: (i) the presence of toxic concentrations of aluminium or manganese and (ii) deficiencies of plant nutrients e.g. phosphorus, calcium, magnesium, or molybdenum. Although pH *per se* is usually not a primary cause of poor plant growth on acid soils, pH plays an important role in determining the availability to plants of chemical elements which markedly affect plant growth.

Sunflower is not highly sensitive to pH (Robinson, 1978), provided toxic substances or essential nutrients do not limit growth. Robinson (1978) reported that the crop is grown on soils ranging from pH 5.7 to over 8, with no single pH optimal for all soil conditions. In solution culture, sunflower has been found to tolerate pH values maintained at from 4.0 to 6.5, although one of the four cultivars studied required pH $\geq$ 5.0 for maximum growth (Blamey *et al.* 1982).

Symptoms at low solution pH

In very young seedlings (*c.* 3 days old), the detrimental effects of maintaining the nutrient solution at pH 3.5 are evident within 1 day. Roots and hypocotyls in contact with the solution rapidly lose turgidity and die. The leaves have a grey-green colour (Blamey *et al.*,1982). A similar rapid response to pH 3.5 has been noted in slightly older seedlings, although these seedlings tended to survive. The plants appear wilted, the older leaves senesce, and the remaining leaves have a grey-green colour (Plate 18.1). The roots are brown and lack turgidity. The root symptoms are quite dissimilar to those observed with aluminium toxicity.

Possible confusion with other symptoms

Symptoms similar to those caused by low solution pH have been observed with high amounts of the heavy metals, cobalt, chromium, and nickel, in solution, in that the young seedlings rapidly lost turgidity and died. Also, in these cases the seedlings had a grey-green colour.

Symptoms caused by low pH could be confused with those caused by water stress, but symptoms caused by low pH cannot be relieved by watering.

18.1

Plate 18.1 Low pH
18.1 Wilting and lower leaf senescence of a sunflower seedling grown at pH3.5.

Correction of low pH

Field-grown sunflower is unlikely to encounter problems of low pH *per se*. In most cases, aluminium toxicity problems would be encountered before the effect of pH became evident.

19. Aluminium Toxicity

Sunflower is extremely sensitive to aluminium (Al) toxicity, which is in marked contrast to the tolerance of this crop to manganese toxicity, another problem encountered in some acid soils. In field studies, sunflower responded to liming an acid sandy loam soil with Al saturation > 5% (Blamey and Nathanson, 1977). In solution culture, Al concentrations as low as 5 μM reduced dry matter yields by 44% (Blamey *et al.*, 1986b).

In the field, Al toxicity has been found to have a most marked detrimental effect on seedling emergence and growth, and on plant survival (Blamey, 1975) (Plate 19.1). Indeed, an Al saturation level of 60% reduced seedling emergence by 10%, decreased the mass of 2 weeks' old seedlings by 66%, and resulted in the survival of less than 20% of the plants at 8 weeks. Sunflower cultivars have been found to differ in sensitivity to Al toxicity in solution culture and in Al toxic soil (Foy *et al.*, 1974).

Symptoms of aluminium toxicity

As with other plants, the primary effect of Al toxicity is evident on the roots. In solution culture, root growth has been found to be markedly reduced by Al present in solution, and the roots show characteristic symptoms of Al toxicity (Plate 19.2). With Al present in solution, primary roots are discoloured, short and thick, with many short and thick lateral roots. Lateral roots are formed close to the root tip, unlike healthy roots where a considerable length (*c.* 10 cm) may be free of lateral root development (Plate 19.2).

In the field, sunflower roots also show abnormalities, although the thickened root system is not as evident. However, root development is much restricted by Al toxicity, and roots have been found not to enter Al toxic zones (Plate 19.3).

Plate 19.1 Aluminium toxicity

19.1 Sunflower affected by aluminium toxicity (foreground) that has been corrected by liming (background).

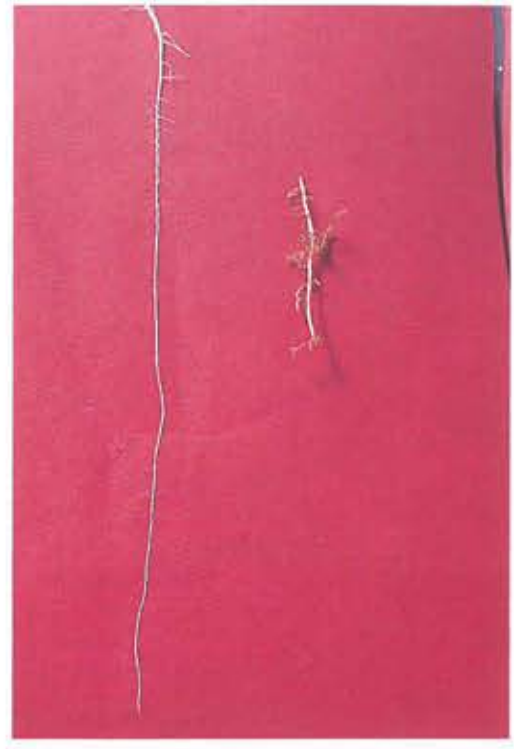
19.2 Short, stubby and discoloured roots (R) caused by aluminium in solution culture in comparison with roots growing in solution without aluminium (L).

19.3 Restricted root development in a soil in which toxic aluminium increased with depth.

19.4 Leaf chlorosis and necrosis in a sunflower seedling caused by aluminium toxicity.

19.5 Effect of aluminium in solution on growth of sunflower (L to R: 0, 5, 10 and 25 μM Al in solution).

19.1



19.2

19.3

19.4

19.5

Because the primary effect of Al toxicity is in reducing root development, many different symptoms have been observed on the tops of plants suffering Al toxicity. Symptoms similar to those caused by moisture stress, phosphorus deficiency (Plate 19.4; 19.5) and magnesium deficiency have been observed. These symptoms have probably been due to decreased water or nutrient uptake (possibly of the nutrient in shortest supply in a particular study) as a result of decreased root proliferation. However in oat plants, magnesium uptake was decreased by Al in solution, despite its lack of effect on top or root growth (Grimme, 1983). Hence, there may be specific effects of Al on root function, even where root growth is not decreased.

Possible confusion with other symptoms

While the symptoms of Al toxicity on the roots of sunflower (and other plants) are characteristic, symptoms on plant tops may be confused with symptoms caused by deficiencies of elements such as phosphorus and magnesium. However, none of these deficiencies cause the root system disorders seen with Al toxicity.

Diagnostic soil and plant tissue tests

Soil tests are probably the best method of diagnosing Al toxicity. Because of the extreme sensitivity of sunflower to Al toxicity, soil pH < 4.5 (measured in 1MKCl) (*c.* pH 5.5, measured in H_2O) has been associated with decreased sunflower growth (Blamey and Chapman, 1979). Also, soil tests for exchangeable Al and Al saturation have been useful for predicting the effects of Al toxicity. Blamey and Nathanson (1977) recommended that sandy loam soils should be limed to decrease Al saturation to $< 5\%$. Above this level, seed yields were increased by 17% for each 10% reduction in Al saturation.

Aluminium saturation of the cation exchange complex has been more useful for predicting Al toxicity than has exchangeable Al (Kamprath, 1984). However, Al saturation has not proved satisfactory across a wide range of soils, and recent studies have suggested the possibility of improved soil tests based on soil solution chemistry.

In solution culture, very low concentrations of monomeric Al (1 μM Al) in solution have been found to be detrimental to sunflower growth (Blamey *et al.*, 1986b). In general, Al concentrations in plant tops have proven unsatisfactory indicators of Al toxicity.

Correction of aluminium toxicity

Since the availability of Al is closely related to soil pH, the application of lime or dolomite to increase soil pH is the major procedure available to correct Al toxicity. These soil ameliorants should be fine and thoroughly mixed with the soil, and applied at rates to increase soil pH to values ≥ 5.5, or to decrease Al saturation below 5%.

Potential problems in neutral and alkaline soils

Sunflower is well adapted to neutral and alkaline soils. Indeed, most sunflower production is on such soils, and few nutritional problems are likely to be encountered that can be directly attributed to high pH.

One problem that may be encountered in alkaline soils is iron deficiency. Iron deficiency may be severe in other crops, e.g. sorghum and soybean (Clark, 1982). However, iron deficiency is unlikely in sunflower due to the morphological and physiological changes that occur in the roots in response to iron stress (Romheld and Marschner, 1981; Romheld *et al.*, 1982). On the other hand, Alcantara and de la Guardia (1986) demonstrated that not all sunflower genotypes are iron-efficient, with some lines showing chlorosis when grown on iron deficient soils.

Other problems associated with some alkaline soils are salinity and sodicity. Sunflower, like other crops grown on these soils, would be prone to nutritional disorders in these situations (Loveday and Bridge, 1983). Excess soluble salts may interfere with water uptake through osmotic effects; nutritional imbalances may be induced; and specific ions may be toxic. Additionally, many saline and sodic soils have adverse physical properties (e.g. surface crusting, reduced permeability to water and air, increased bulk density) that would be detrimental to plant growth.

In contrast with soils that have pH > 7 in the natural state, the liming of acid soils to pH 7 and above may have detrimental effects. Possible reasons for reduced plant growth following liming to near-neutral pH include decreased permeability of the soil to air and water, and decreased availability of phosphorus, boron, manganese, copper and zinc (Kamprath, 1972; Farina *et al.*, 1980).

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