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G rain sorghum is the staple food of low income farmers in many developing countries in semi-arid tropical areas of Africa and Asia. The yields harvested by these farmers are commonly less than 30% of average sorghum yields in developed countries due to use of low-yielding genotypes, drought, pests, diseases, and nutrient constraints. Nutritional disorders are a major limitation because grain sorghum is grown in areas with impoverished soils and because farmers generally use little fertilizer. Accurate diagnosis is the essential first step before these disorders can be corrected by the judicious use of fertilizers.

Foreword

The preparation and publication of this book were prompted by the recommendation, at the 'Sorghum in the Eighties Symposium' in Hyderabad, India in 1981, that an appropriate handbook be produced to document improved diagnostic techniques for nutrient deficiencies and excesses. To achieve this, ACIAR commissioned a collaborative project between the All-India Coordinated Micronutrient Program of the Indian Council of Agricultural Research, and the plant nutrition group of the Department of Agriculture, University of Queensland. The two groups shared the task of gathering the material for the book, with the Indian partners taking major responsibility for the field program and the Queensland group undertaking much of the controlled glasshouse work. Previous experience by the Queensland group in producing books on the nutritional disorders of cassava and ginger was most valuable.

ACIAR wishes to express its gratitude to the dedicated Indian and Australian scientists who made this publication possible. We also appreciate the valuable contribution to the project made by Dr R.B. Clark, of the United States Department of Agriculture and the University of Nebraska. The International Crops Research Institute for the Semi-Arid Tropics deserves special thanks for its general support of the project and for releasing an Indian staff member, Dr T.J. Rego, to work on the project at the University of Queensland.

ACIAR hopes that this book will prove valuable in the accurate diagnosis of nutritional disorders of grain sorghum, and that the agronomists, soil scientists, and extension specialists using it will in turn pass on this knowledge to farmers, with the ultimate goal of increasing sorghum yields.

E.T. Craswell
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Introduction

Grain sorghum (*Sorghum bicolor* (L.) Moench) is the sixth most important dietary source of calories for the world's population, after rice, wheat, sugar (cane and beet), maize, and *Solanum* potatoes (Cock 1985), and is the fifth most important cereal grain on a world production basis, after wheat, maize, rice, and barley (FAO 1985). It is the major staple food grain of many people living in the semi-arid tropical regions of Africa and Asia, and is also used extensively in numerous countries throughout the world as a livestock feed, either as green forage, dry straw, or a grain concentrate.

Little change has occurred in world production of sorghum (60–77 million t/annum) over the last 10 years (Table 1). About 84% of the area harvested and about 65% of the world production comes from the developing nations, especially those in the semi-arid tropics of Africa and Asia. Average yields of sorghum in many African and Asian countries (300–900 kg/ha) are much lower than those in other countries (e.g. 3000–4200 kg/ha in the USA) and have shown little improvement since 1948 (Leng 1982).

Myers and Asher (1982) and Clark (1982a, d) drew attention to the diverse range of soils on which sorghum is grown. The most predominant soils used are: Vertisols, Inceptisols, Entisols, and Alfisols in Asia; Vertisols, Alfisols, and Mollisols in North and Central America; Mollisols, Alfisols, Oxisols, and Ultisols in South America; Vertisols in Australia; and Vertisols, Alfisols, Entisols, and Inceptisols in Africa. The range of soils is so wide that it is not surprising that some are exceptionally low in certain nutrients or have excessive quantities of certain nutrients. Nutritional stress problems in soils are often related to the type of parent material and the soil-forming processes characteristic of that soil. Therefore, many nutrient deficiencies or toxicities can often be predicted from the soil type and parent material (Dudal 1976; Clark 1982a, d).

Acid soils (e.g. Oxisols, Ultisols, and some Entisols, Alfisols and Inceptisols) are usually low in exchangeable bases, and are frequently low in available phosphorus. Acidity increases the solubility of iron, aluminium, and manganese, and the concentration of these elements in the soil solution may reach toxic levels in very acid soils. The major nutritional problems for sorghum growing on acid soils are toxicities of aluminium, iron, and manganese, and deficiencies of phosphorus, calcium, magnesium, molybdenum, and zinc (Clark 1982a).

	1974–76[a]	1983[b]	1984[b]	1985[b]
World				
Production (Mt)	60.1	59.6	68.1	77.4
Area (Mha)	47.5	47.4	48.2	50.2
Yield (kg/ha)	1266	1257	1413	1543
Developing Nations				
Production (Mt)	40.2	45.3	43.1	46.5
Area (Mha)	40.4	42.0	40.6	41.9
Yield (kg/ha)	994	1078	1061	1109
Developed Nations				
Production (Mt)	19.9	14.3	25.1	30.9
Area (Mha)	7.1	5.4	7.7	8.2
Yield (kg/ha)	2824	2659	3283	3751

TABLE 1
Trends in grain sorghum production, 1974–85

Source: [a]FAO 1984; [b]FAO 1985.

Alkaline soils (e.g. Mollisols, Vertisols, and some Inceptisols) often contain fairly high concentrations of salt in the profile, and if the groundwater is close to the surface or if irrigation is mismanaged, these soluble salts may accumulate in surface layers and the soil may become saline. Alkaline soils are often well supplied with calcium, magnesium, and potassium, but deficiencies of sulfur and toxicities of boron may occur. Most alkaline soils contain sufficient iron to supply the needs of sorghum, but the iron is not readily available. The most common nutritional disorders observed in sorghum growing on alkaline soils are deficiencies of iron (Clark 1982a; Tandon and Kanwar 1984), zinc, and manganese (Tandon and Kanwar 1984).

Soils which were once well supplied with nitrogen and phosphorus often became depleted in these nutrients following a long history of cropping. As a result, deficiencies of nitrogen and phosphorus are widespread in many countries (Vance 1981; Clark 1982a; Myers and Asher 1982; Tandon and Kanwar 1984; Grundon and Vance 1986). Potassium deficiency has been reported in India (Tandon and Kanwar 1984), while sulfur deficiency has been reported in Papua New Guinea (Vance 1981) and India (Tandon and Kanwar 1984). Iron and zinc deficiency are widespread in the USA (Clark 1982a) and India (Tandon and Kanwar 1984), while responses to applied manganese have been demonstrated in India (Tandon and Kanwar 1984). Boron, copper, and molybdenum deficiency have been shown to limit sorghum yield on a small number of Indian soils (Tandon and Kanwar 1984).

While visible symptoms are often used to help identify the cause of nutritional disorders, good descriptions and high

quality colour photographs of symptoms of deficiencies and toxicities on a range of grain sorghum cultivars are not readily available. Agarwala and Sharma (1979) published colour photographs and descriptions of symptoms of iron, manganese, zinc, and boron deficiency. Frederiksen (1986) reported similar data for nitrogen, phosphorus, potassium, calcium, magnesium, sulfur, manganese, iron, and zinc deficiency, and for toxicities of phosphorus, boron, zinc, sodium, and aluminium. McLachlan (1978) published a colour photograph and description of sulfur deficiency.

Black and white photographs plus descriptions have been published of symptoms of calcium deficiency (Kawasaki and Moritsugu 1979; Moritsugu and Kawasaki 1985); phosphorus, copper, and iron deficiency (Brown et al. 1977); phosphorus and iron deficiency and aluminium toxicity (Clark 1982a, b); iron, boron, aluminium, cobalt, and strontium toxicity (Clark 1982d); phosphorus toxicity (Clark 1982c; Furlani et al. 1986a, b); iron deficiency (Kannan 1980); manganese deficiency (Ohki 1974); zinc deficiency and toxicity (Ohki 1984); and copper, iron, and zinc deficiency together with manganese and aluminium toxicity (Brown and Jones 1977).

Written descriptions without photographs have been published for deficiency and toxicity symptoms of various essential elements (Krantz and Melsted 1964; Tucker and Bennett 1968; Gallaher et al. 1975; Clark et al. 1981; Clark 1982d; Grundon et al. 1986; Shorrocks (undated)), and for toxicity symptoms of aluminium (Clark et al. 1981; Furlani and Clark 1981; Grundon et al. 1986), and a number of heavy metals (Clark et al. 1981; Clark 1982d).

Most of the symptoms described in this book were developed on plants grown in a glasshouse under controlled conditions of nutrient supply (soil or nutrient solution culture) at the University of Queensland, St. Lucia, Australia. Where possible, these symptoms have been checked against symptoms expressed by plants growing in the field. Unless otherwise acknowledged, soil and plant analysis data have been derived from glasshouse trials at the University of Queensland, Australia, or from greenhouse and field trials undertaken at Ludhiana, Hyderabad, Coimbatore, and Anand, India.

Diagnosis of nutritional disorders

Visible symptoms

Most nutritional disorders produce characteristic symptoms on leaves, stems, or roots which can be used in diagnosis under field conditions. The use of visible symptoms has the advantage of direct field application without the need for costly equipment or laboratory support services, as is the case with soil and plant analysis. A disadvantage is that the disorder can be diagnosed only after it is sufficiently severe to produce visible symptoms. For some disorders, considerable yield loss may have already occurred by the time visible symptoms appear or it may be too late to alleviate the problem within the current growing season. Because different nutritional disorders, or attacks by pests and diseases, may sometimes produce rather similar visible symptoms, visual diagnosis of nutritional disorders should be considered a preliminary diagnosis to be confirmed by other methods such as plant analysis, soil analysis, or fertilizer trials.

The normal patterns of distribution and redistribution of mineral nutrient elements within the plant cause symptoms of nutritional disorders to occur in particular positions on the plant (Asher et al. 1980). Thus, in addition to observing the appearance of affected leaves, attention must be directed to their location on the plant.

Nutrient elements generally tend to be distributed among the various parts of the shoot in a pattern resembling that of water loss due to transpiration. Thus, fully expanded leaves tend to receive a larger share of the water and nutrient elements entering the shoot than do the tightly rolled immature leaves in the whorl at the shoot apex. When above-optimum supplies of a mineral element are provided, the highest concentrations will normally be found in the oldest leaves because accumulation has been occurring for the longest period of time in these leaves. The older leaves will usually show symptoms of nutrient toxicity first and most markedly. Where an excess of one element reduces the uptake of another element or interferes with its utilisation in the plant tissues, the main symptoms may be those of a deficiency of the other element, with the symptoms appearing at the position characteristic for that element.

When a deficiency of nitrogen, phosphorus, potassium, or magnesium occurs, plants tend to withdraw these elements from older leaves and redistribute them to young, actively growing parts of the plant (Robson and Snowball 1986). These elements are redistributed via the

phloem. Hence, the first and most obvious symptoms of deficiency of phloem-mobile elements generally occur on lower, older leaves.

Elements such as calcium, iron, manganese, and boron are not redistributed to any great extent under deficiency conditions and are referred to as phloem-immobile elements (Robson and Snowball 1986). The first and most obvious symptoms of deficiency of phloem-immobile elements occur on young, actively growing parts of the plant, including root tips. With phloem-immobile elements, the plants must have a continuous external supply if healthy growth is to be maintained. If the supply has fluctuated widely during the growing season, it is common to find deficiency symptoms on leaves formed during a period of deficiency, with healthy leaves both above and below the affected leaves.

The nutrient elements sulfur, zinc, copper, and molybdenum often have variable mobility in the phloem. Under some conditions, they appear to be phloem-mobile while under other conditions they behave as phloem-immobile elements (Robson and Snowball 1986). Hence, for these elements, symptoms may appear on young or old growth depending on the species and other factors, among which nitrogen supply appears important. However, in our experience with grain sorghum, only sulfur and zinc exhibited variation in the location of visible symptoms of deficiency, and then only when the deficiency was very severe and persisted for some time.

Plant analysis

The principles and use of plant analysis have been covered in a number of reviews, including those by Macy (1936), Ulrich (1952), Smith (1962), Bates (1971), Jones (1972), Munson and Nelson (1973), Moraghan (1985), and Smith (1986). Plant analysis involves a number of steps, including sampling, sample preparation, laboratory analysis, and interpretation of data. All of these must be undertaken with care if the technique is to be used successfully, to evaluate the nutritional status of a plant. Jones and Steyn (1973), Moraghan (1985), and Reuter et al. (1986) have addressed, in general terms, the problems associated with sample selection, sample handling and preparation, and laboratory analysis, while Jones and Eck (1973) reviewed similar information specifically in relation to corn and grain sorghum.

The use of plant analysis as a diagnostic tool is based on the existence of a functional relationship between crop yield and concentration of nutrients in the whole plant or an index tissue. However, most elements are distributed unevenly between plant parts and even within the same tissue (Jones 1970, 1983). In addition, the concentration of elements in a given tissue often varies with stage of growth, plant age, genotype (cultivar), climatic conditions, and interactions between elements (Ulrich and Hills 1967; Bates 1971; Lockman 1972a, b, c; Jones and Eck 1973; Jones 1983). Each of these factors can affect the relationship between crop yield and concentration of the nutrient in the plant or index tissue, and, as a result, the concentration of nutrient which may represent healthy growth can vary considerably. Standardisation of time of sampling, tissue sampled, stage of growth, and age of plant helps decrease this variation.

Unfortunately, published information for grain sorghum shows little evidence of standardisation of procedures. For grain sorghum during the vegetative stages of growth, Lane and Walker (1961) bulked all leaves, while Agarwala and Sharma (1979) sampled middle leaves. In studies with very young plants, whole shoots have been analysed (Boawn and Rasmussen 1971; Lockman 1972a, b, c; de Boer and Reisenauer 1973; Shukla et al. 1973; Brown and Jones 1977; Salardini and Murphy 1978; Kuo and Mikkelsen 1981; and Clark et al. 1981). Lockman (1972a, b, c) took the 'third leaf' (i.e. the newest, fully-developed leaf) of older vegetative plants, and Ohki (1974, 1984) recommended sampling 'blade 1' (i.e. the leaf with the most recently developed ligule). When grain sorghum was in head, Francois et al. (1984) sampled the fourth blade below the flag leaf, Gallaher

et al. (1975) took the fourth leaf from the apex, Lockman (1972a, b, c) analysed the third leaf below the head, and Brawand and Hossner (1976), Vance (1981), and Weir (1983) used the second leaf from the apex. Clearly, there is a need to select a standard index leaf for all stages of growth. *We recommend that the Youngest Emerged Blade (YEB), which we define as the blade of the youngest leaf on which the ligule has fully emerged, be chosen as the standard index leaf.* Hence, during vegetative growth, the YEB would be equivalent to the third leaf of Lockman and blade 1 of Ohki. However, once the flag leaf or head has emerged, the YEB is equivalent to the flag leaf.

No studies could be found which have critically examined differences in tissue concentrations for healthy growth between cultivars of grain sorghum. However, data from Lockman (1972b), Mikesell et al. (1973), Brown and Jones (1975), Brown et al. (1977), Esty et al. (1980), Kannan (1980), and Furlani et al. (1984, 1986b) suggest that significant differences may occur in tissue concentrations for healthy growth, especially for phosphorus and iron.

Knowledge of the response curve which describes the relationship between crop yield and nutrient concentration in the plant or index tissue is essential for interpretation. Two main approaches have been used in attempts to quantify the response curve in terms of yield-nutrient concentration relationships: (a) the use of a 'critical nutrient concentration' (CNC) or 'standard value' (Macy 1936; Ulrich 1952; Ulrich and Hills 1967; Bates 1971), and (b) the use of sufficiency ranges (Lockman 1972c; Jones and Eck 1973). Ulrich and Hills (1967) defined the CNC as the concentration corresponding to 90% of maximum yield, and this approach has been used widely for grain sorghum (de Boer and Reisenauer 1973; Agarwala and Sharma 1979; Ohki 1974, 1984; Vance 1981; Weir 1983). While the CNC is frequently reported as a single value, Ulrich (1952) recognised that a narrow critical range more truly represented the situation, and P.F. Smith (1962), Dow and Roberts (1982), and F.W. Smith (1986) strongly supported this view. Hence, the use of sufficiency ranges may go some way towards satisfying the concept that the CNC represents a range rather than a single value.

Another method which is sometimes used to interpret plant analyses is based on nutrient ratios. Where a sound physiological basis exists for the interaction between two elements, such as sulfur and nitrogen in the formation of proteins (Freney et al. 1977), the ratio of their concentrations

has proved to be a valuable aid in diagnosing the nutritional status of some crop and pasture species (Spencer et al. 1977; Randall et al. 1981). Beaufils (1971, 1973) used nutrient ratios or other ratio indices of nutrient status, in his Diagnosis and Recommendation Integrated System (DRIS) for assessing the nutritional balance of plants. Diagnosis using DRIS rests on a comparison of the nutrient concentration in plant tissue with DRIS reference parameters (norms) and their coefficients of variation in a special calibration formula for determining, in descending order, the most limiting elements. Although DRIS norms (Table 2) have been proposed for use with grain sorghum (Arogun 1978; Sumner et al. 1983), and methods for refining these norms have been

T A B L E   2   DRIS norms and coefficients of variation for grain sorghum (from Arogun 1978).

Tissue parameter	Norm	Coefficient of variation (%)
p/n	0.112	19
n/k	2.355	23
p/k	0.259	21
n/ca	7.200	30
p/ca	0.975	31
k/ca	3.080	24
mg/n	0.079	26
p/mg	1.518	45
mg/k	0.183	26
mg/ca	0.553	30

suggested (Cornforth and Steele 1981; Escano et al. 1981a, b; Elwali et al. 1985; Walworth et al. 1986), Smith (1986) considered that there was no apparently sound physiological basis for the use of DRIS in Australia.

Soil analysis

The principles and uses of soil analysis have been reviewed in a number of publications, including those of Bray (1948), Cox and Kamprath (1972), Walsh and Beaton (1973), and Lindsay and Cox (1985). The use of soil analysis as a diagnostic tool is based on the existence of a functional relationship between the amount of nutrient element extracted from the soil by chemical methods and crop yield. While much of the present-day use of soil analysis is directed towards making sound fertilizer recommendations, the same soil test values can be used also to assist in diagnosing nutritional disorders. As a diagnostic tool, soil analysis has a major advantage over both plant analysis and visual symptoms in that it can be used to predict the nutritional status of a soil before the crop is planted.

Soil analysis involves a number of steps, including sample collection, handling and preparation, chemical extraction, and analysis, which must be carefully undertaken if the technique is to be used successfully to estimate the nutritional status of the soil. Peck and Melsted (1973) addressed the problem of obtaining a representative sample from the field to be tested, while Cox and Kamprath (1972), Melsted and Peck (1973), and Lindsay and Cox (1985) have considered the problems of choosing the extractant and the method of chemical analysis.

Before a soil analysis can be used to diagnose the nutritional disorder, the soil test (i.e. the chemical extractant and conditions prevailing during extraction) must be carefully calibrated against the field response of the crop to applications of the test element on the range of soils for which the soil test is to be used. When the soil test is to be used to separate deficient from non-deficient soils, only a single or 'critical' value which separates these two classes of soils is needed. The calibration and interpretation of soil tests have been discussed by Cate and Nelson (1965, 1971), Cox and Kamprath (1972), Melsted and Peck (1973), Cope and Rouse (1973), Hanway (1973), Rayment (1985), Moody (1985), and Fergus and Little (1985).

It is assumed that the extractant removes the whole or some part of the plant-available fraction of the element in the soil. As numerous factors affect the 'availability' of a nutrient, many soil tests do not have a universal application and may be limited in their use to a range of soils of similar chemical and physical characteristics. Furthermore, crop species often have different critical values for the same soil type and climatic environment because of differences in their ability to extract the nutrient from the soil, in their requirement for the

nutrient, in their rates of nutrient uptake, or in their utilisation coefficients, i.e. dry matter or grain yield produced per unit of nutrient absorbed (Cope and Rouse 1973; Kanwar and Youngdhal 1985). Grain sorghum cultivars have been shown to differ in their ability to extract native soil nitrogen (Tandon and Kanwar 1984) and in their susceptibility to aluminium toxicity (Furlani and Clark 1981), iron deficiency (Mikesell et al. 1973; Esty et al. 1980; Clark et al. 1982; Williams et al. 1982; Tandon and Kanwar 1984), zinc deficiency (Randhawa and Takkar 1976; Tandon and Kanwar 1984), phosphorus deficiency (Brown and Jones 1975; Brown et al. 1977; Clark et al. 1978; Furlani et al. 1984), and phosphorus toxicity (Furlani et al. 1986a, b). It is usually assumed that traditional open-pollinated cultivars of grain sorghum are better adapted to local soil fertility conditions than are the more fertilizer-responsive hybrids, but the possibility that such cultivar differences may be reflected in different soil test values does not appear to have been critically examined for grain sorghum.

For data obtained from the greenhouse and field experiments conducted in India as part of this study, and for other unpublished data of Indian origin, the statistical procedure of Cate and Nelson (1971, 1973) was used to determine critical values for nitrogen, phosphorus, potassium, sulfur, zinc, manganese, and iron.

Pot experiments

Glasshouse pot experiments provide a rapid means of screening soils for potential nutritional limitations, and for estimating approximate rates of fertilizer which will be needed to correct these limitations. Such information is an extremely valuable aid to the efficient design of field experiments. By comparison with field trials, pot trials usually require less time and materials, and provide diagnostic results more quickly. However, pot trials may sometimes identify disorders which do not occur in the field, (a) because either water or climatic factors may limit growth in the field, or (b) because the amount of nutrients available to the plant may differ between the pot and field situation.

Fertilizer trials

Fertilizer field trials are an important part of the overall diagnostic process, because the only way to establish the fertilizer requirement accurately is by means of field experiments where the full interplay of plant, soil, management, and environmental factors can occur (Asher et al. 1983). However, field trials require large inputs of time and materials, and for practical reasons only a small number of possible nutritional limitations can be tested, often only nitrogen, phosphorus, and potassium. As a result, field trials sometimes fail to identify the disorder or lead to grossly inaccurate assessments of the true fertility status of the soil when multiple nutrient disorders exist. Furthermore, field trials are often slow to produce diagnostic results, and so are an inefficient means of making an initial diagnosis.

Key to symptoms of nutritional disorders of grain sorghum

Key:

A1 Symptoms begin or are more severe on older leaves **B**

A2 Symptoms begin or are more severe on young leaves **D**

A3 Symptoms prominent on both old and young leaves **G**

A4 No specific leaf symptoms but roots thickened and fail to elongate **Aluminium toxicity**

B1 Older leaves mainly pale green with pale yellow chlorosis and pale brown necrosis **Nitrogen deficiency**

B2 Older leaves mainly pale green with pale yellow interveinal chlorosis and sometimes brown, orange, and purple lesions **Magnesium deficiency**

B3 Older leaves mainly dark green with or without dark yellow chlorosis but often with purple, red, or dark brown lesions or pigmentation **C**

C1 Older leaves with small dark brown to red-purple lesions resembling spots **Manganese toxicity**

C2 Older leaves with purple suffused pigmentation, no orange or brown lesions **Phosphorus deficiency**

C3 Older leaves with large orange, yellow, purple, or brown streaks and lesions **Zinc toxicity**

C4 Older leaves with prominent red-purple lesions which have an irregular outline **Phosphorus toxicity**

C5 Older leaves with marginal yellow chlorosis and brown necrosis, or older leaves with marginal brown lesions surrounded by red-purple halo with smooth outline, plants not wilted **Potassium deficiency**

C6 Older leaves with yellow, white, or brown margins, plants often wilted **Toxicity of sulfur, sodium, or chlorine**

C7 Older leaves with grey to white necrotic lesions and brown marginal necrosis, plants often appear wilted **Sodium toxicity**

C8 Plants often appear wilted, yellow interveinal chlorosis and red-purple lesions on middle leaves **Chlorine toxicity**

D1 Young leaves mainly pale green or pale yellow   **E**

D2 Young leaves mainly dark green   **F**

E1 Whorl of young leaves twisted to
one side   **Copper deficiency**

E2 Prominent yellow or white interveinal chlorosis
on young leaves   **Iron deficiency**

E3 Young leaves may develop faint yellow
interveinal chlorosis but usually turn an even
pale yellow   **Sulfur deficiency**

F1 Young leaves with brown twisted
leaftips   **Copper deficiency**

F2 Young leaves with torn or serrated leaf margins
and leaftips deformed, missing or
joined together   **Calcium deficiency**

F3 Young leaves with broad yellow or white bands
between the margins and midvein in lower
half of leaf   **Zinc deficiency**

F4 Young leaves with transparent white
interveinal lesions   **Boron deficiency**

F5 Young leaves with yellow interveinal chlorosis and
red-brown interveinal lesions   **Manganese deficiency**

G1 Older leaves mainly dark green with large
purple or orange lesions, young leaves
pale yellow   **Zinc toxicity**

G2 Older leaves mainly dark green with small
brown or red-purple lesions, young leaves
pale yellow   **Manganese toxicity**

G3 Older leaves and younger leaves mainly pale green
or pale yellow, with or without interveinal
chlorosis   **Deficiency of sulfur or
toxicity of aluminium inducing
deficiencies of iron and/or
magnesium**

Disorders producing symptoms mainly on lower leaves

Nitrogen deficiency

Symptoms

Deficient plants are pale green to pale yellow in colour (Plate 1a), grow more slowly, and often show delayed flowering and maturity (Plate 1b). The stems tend to be thin and spindly, and in some cultivars red streaks develop on the lower leaf sheaths (Plate 1c).

Symptoms appear first on older leaves and then advance up the stem to younger leaves. The leaf blades progressively change colour from dark green to pale green (Plate 1a). A pale yellow chlorosis develops and this is followed by a brown necrosis (Plate 1d, e). The chlorosis and necrosis begin near the tip of the leaf blade and advance towards the base until the whole blade turns yellow, then brown (Plate 1a, d, e). The chlorosis and necrosis may advance along the midvein in a V-shaped pattern, along the margins, or on a broad front (Plate 1a, d, e). The boundary between the healthy green tissue and the chlorotic tissue is diffuse. In most cultivars, the chlorosis is a uniform pale yellow colour (Plate 1d), but in others, small brown or dark grey necrotic spots or streaks appear within the chlorotic tissues (Plate 1e). Frederiksen (1986) described the development of a 'firelike or deep yellow' chlorosis by some cultivars. Eventually, the affected leaves wither, die, and hang down around the lower stem (Plate 1a).

The dominant symptom of nitrogen deficiency is the development of pale yellow lower leaves. Similar symptoms occur when sorghum plants are attacked by the yellow sugarcane aphid (*Sipha flava*) (Teetes et al. 1983). Aphid attack can be distinguished from nitrogen deficiency by the presence of the aphid on the yellow lower leaves, and by the dark green colour of the healthy, upper leaves which are usually pale green in colour when nitrogen is deficient.

Nitrogen deficiency

a **Severe nitrogen deficiency in young plants of a local cultivar of grain sorghum growing on an Alfisol at Hyderabad, India. Note the pale green, yellow, and brown lower leaves.**

b **Effect of nitrogen fertilizer on stage of development in cv. Goldrush on a Vertisol at Cambooya, Australia. Plants on left received 50 kg N/ha; plants on right have received no nitrogen fertilizer and show delayed flowering.**

c **Comparison of stems from nitrogen-deficient (left) and healthy (right) plants of cv. Texas 610SR grown in solution culture. Note the thinner stems and greater red streaking on the lower leaf sheaths of the nitrogen-deficient plant (left).**

d **Comparison of healthy (left) and nitrogen-deficient (centre and right) leaves of cv. Texas 610SR.**

e **Nitrogen-deficient leaves of cv. M35-1 showing yellow chlorosis and brown necrotic spots and streaks.**

a

b

c

d

e

Tissue analysis

Boawn et al. (1960), Lockman (1972a, b, c), and Reuter (1986) presented data on the nitrogen concentration for various parts and stages of growth of grain sorghum plants. Lockman (1972c) and Reuter (1986) classed nitrogen concentrations of 3.0–5.1% in whole shoots of young plants (i.e. up to growth stage 3; collar of 8th leaf visible – Vanderlip and Reeves 1972), and 1.9–4.0% in the upper leaves of plants at anthesis and during grain filling, as adequate for grain sorghum growth. Critical nitrogen concentrations at several stages of growth are shown in Table 3.

TABLE 3   Concentrations of nitrogen in grain sorghum corresponding to 90% of maximum yield.

Growth stage[a]	Plant part[b]	N conc (%)	Reference
2	YEB	5.3	This study cv. Texas 610SR
30 DAS	WS	3.0	Hariprakash (1979)
3–4	YEB	4.8	This study cv. Texas 610SR
3–4	YEB	3.3	This study cv. M35-1
60 DAS	WS	1.8	Hariprakash (1979)
6, 7	2BBH	2.5	Weir (1983)

[a]As in Vanderlip and Reeves (1972); DAS = days after sowing.
[b]YEB = youngest emerged blade; WS = whole shoot; 2BBH = second blade below head.

Dahnke and Vasey (1973) reviewed the methods used in testing soils for nitrogen and divided them into two groups: (a) those which estimate the potential of the soil to mineralise nitrogen from organic nitrogen reserves, and (b) those which measure the inorganic nitrogen content (i.e. ammonium and nitrate) at a given point in time, usually just prior to sowing the crop. The alkaline-permanganate test is an example of the first group. This test has been used widely in India where ratings of below 280 kg N/ha, between 280 and 560 kg N/ha, and above 560 kg N/ha correspond to low, medium, and high nitrogen status respectively for general crop growth (Tandon and Kanwar 1984). More recent unpublished data of Takkar indicate a critical range of 218–247 kg N/ha for grain sorghum.

In carefully controlled solution culture studies, Forno (1977) showed that grain sorghum can effectively utilise both ammonium and nitrate nitrogen. With ammonium nitrogen alone, the maximum dry matter yield was within a few per cent of that for nitrate alone. When urea is applied to soil, hydrolysis to ammonium nitrogen is usually quite rapid, but nitrification of ammonium to nitrate may take considerable time, particularly at high rates of application. For example, when 336 kg N/ha as urea was added at planting to a sorghum crop, 88% of the mineral nitrogen in the top 10 cm was present as the ammonium-ion 11 days after planting, and 65% was still present in that form after 25 days (Cowie and Asher 1986). Nevertheless, soil tests for plant available mineral nitrogen often measure only the nitrate fraction. Among studies in which the measurement of soil nitrate failed to adequately characterise the nitrogen status of a soil are those of Dahnke and Vasey (1973) and Hibberd et al. (1986). In Australia, 15–20 mg N/kg has been used as a critical range (1:5 water-extractable nitrate in the top 60 or 90 cm) for sorghum production (Rayment and Bruce 1984).

Studies of Asher and Cowie (1986) and Cowie and Asher (1986) clearly showed that for maximum grain yields of sorghum, adequate mineral nitrogen is needed in the root zone until the season is well advanced, nitrogen stress prior to anthesis resulting in yield reductions due to abortion of florets. Mineral nitrogen in the root zone beyond anthesis had little effect on yield, but was an important determinant of grain protein concentration.

Soil analysis

Phosphorus deficiency

Symptoms

Except for a general lack of vigour and slowness to flower and mature, few clearly recognisable symptoms appear when the deficiency is mild. Affected plants appear stunted with thin stems and dark green leaves. When the deficiency becomes more severe, growth is greatly reduced and overtones of dark red or purple develop on the sheaths and blades of older leaves (Plate 2a, b). In some cultivars, the red to purple overtones advance more rapidly along the veins to give a red or purple streaked pattern. Eventually, the red to purple pigmentation may extend the full length of the leaf blade. When the deficiency is very severe, a dark brown necrosis often develops near the tip of the leaf blade and advances inwards and along the margins towards the base. Eventually, the affected leaves wither, die, turn dark brown, and hang down around the stem (Plate 2a).

The degree of red or purple pigmentation varies from cultivar to cultivar. Some cultivars develop very little red or purple pigmentation (Plate 2c). In these cases, the older leaves usually turn yellow and brown as a dark yellow chlorosis develops near the tip and advances inwards and along the margins towards the base of the leaf blade (Plate 2c). The dark yellow chlorosis is usually followed by a dark brown necrosis. The boundary between the healthy green tissue and the chlorotic or necrotic tissue is usually sharp and distinct.

Some cultivars develop an additional symptom on their young mature leaves when grown in solution cultures in the glasshouse (Grundon et al. 1986); a pale yellow, interveinal chlorosis develops on one or both sides of the midvein about halfway along the leaf blade. This symptom remains very localised and does not extend outwards towards the margins or towards the base or tip of the leaf blade (Plate 2d).

Clark et al. (1981) reported that excess selenium or molybdenum caused a diffuse reddish purple pigmentation to develop within chlorotic interveinal tissues on older leaves. Presumably, these were symptoms of phosphorus deficiency induced by the excess selenium and molybdenum.

Phosphorus deficiency.

a **Phosphorus-deficient plants in the field at Garden City, Kansas, USA. Note stunted growth and purple pigmentation on leaves.**

b **Comparison of stems from healthy (left) and phosphorus-deficient (centre and right) plants of cv. Pride grown in solution culture. Note the thinner stems and the purple pigmentation on the leaf sheaths of the deficient plants (centre and right).**

c **Marginal chlorosis and necrosis on phosphorus-deficient leaves of cv. Goldfinger grown in solution culture. Note the lack of extensive purple pigmentation.**

d **Young mature leaves from healthy (left) and phosphorus-deficient (right) plants of cv. Texas 610SR grown in solution cultures in a glasshouse. Note the pale yellow interveinal chlorosis on the deficient leaf (right).**

a

b

c

d

−P
PRIDE

Tissue analysis

Lockman (1972a, b, c), Clark et al. (1978), Kuo and Mikkelsen (1981), Reneau et al. (1983), and Reuter (1986) presented data on concentrations of phosphorus for various parts and stages of growth of grain sorghum plants. Lockman (1972c) and Reuter (1986) classed phosphorus concentrations of 0.21–0.60% in whole shoots of young sorghum plants (up to growth stage 3; collar of 8th leaf visible – Vanderlip and Reeves 1972), and 0.15–0.35% in upper leaves of plants at anthesis and during grain filling, as adequate for sorghum growth. Critical phosphorus concentrations at several stages of growth are shown in Table 4.

T A B L E 4   Concentrations of phosphorus in grain sorghum corresponding to 90% of maximum yield.

Growth stage[a]	Plant part[b]	P conc (%)	Reference
3–4	YEB	0.47	This study cv. Texas 610SR
3–4	YEB	0.50	This study cv. M35-1
60 DAS	WS	0.23	Hariprakash (1979)
6	YEB	0.52	This study cv. Texas 610SR
6–7	2BBH	0.25	Weir (1983)

[a]As in Vanderlip and Reeves (1972); DAS = days after sowing.
[b]YEB = youngest emerged blade; WS = whole shoot; 2BBH = second blade below head.

Bingham (1965) and Thomas and Peaslee (1973) have described the various methods used in testing the phosphorus status of soils. Extraction with 0.5M sodium bicarbonate has been used in many countries as an estimate of plant-available phosphorus, and critical values for sorghum growth have been found to range from 6 to 23 kg P/ha in India (P.N. Takkar, unpublished data; Singh and Dass 1984; Tandon and Kanwar 1984), and from 15 mg P/kg (0–60 cm) to 35 mg P/kg (0–10 cm) in Australia (Rayment and Bruce 1984). On Australian soils with a pH less than 7.0, extraction with 0.005M sulfuric acid is preferred, and critical values range from 35 to 45 mg P/kg (0–10 cm) (Rayment and Bruce 1984) and from 40 to 60 mg P/kg (0–10 cm) (G.H. Price, pers. comm.).

Soil analysis

Potassium deficiency

Symptoms

Potassium deficient plants lack vigour, flowering and maturing more slowly than healthy plants. The stems are shortened and thin, and the older leaves display a marginal necrosis which may extend the full length of the leaf blade (Plate 3a, b). However, the pattern of development and pigmentation of the necrosis differs considerably among cultivars.

In some cultivars, a yellow to white chlorosis develops near the margins at the tip of the leaf blade (Plate 3a, b, c). The chlorosis then advances along the margins towards the base of the leaf blade as the severity of the deficiency increases. The chlorosis is followed by pale brown necrotic lesions which often join together and may become continuous from the tip to the base of the leaf blade (Plate 3b). In other cultivars, the lesions are reddish purple (Plate 3d) or reddish brown in colour, and develop pale brown necrotic centres in time (Plate 3d). These lesions develop near the leaf tip in the interveinal tissues and advance initially towards the margins (Plate 3d), and then along the margins towards the base of the leaf blade. The boundary between the healthy green tissue and the yellow, purple, or brown tissue is usually distinct and sharp (Plate 3b, c, d).

There appears to be some uncertainty regarding the leaves on which the first symptoms appear. Krantz and Melsted (1964) reported that the symptoms appeared firstly on older leaves and advanced towards younger leaves if the deficiency persisted, while Morard (1973) and Grundon et al. (1986) found that the symptoms appeared firstly on middle or young mature leaves and then spread to lower, older leaves and finally to younger leaves as the severity increased.

A number of diseases of sorghum cause lesions which could be confused with the purple and brown lesions (Plate 3d) developed on some cultivars under potassium deficiency. These diseases include leaf blight (*Helminthosporium turcicum*), oval leaf spot (*Ramulispora sorghicola*), and anthracnose (*Colletotrichum graminicola*) (Williams et al. 1978). However, none of these diseases causes the development of marginal necrosis (Plate 3a, b, c) which

Potassium deficiency.

a Potassium-deficient plant of cv. Goldfinger showing marginal necrosis on the lower leaves.

b Comparison of healthy (right) and potassium-deficient (centre and left) leaves of cv. Texas 610SR grown in solution culture.

c Symptoms of potassium deficiency on leaves of cv. Texas 610SR (left) and cv. Pride (right) grown in solution culture.

d Close-up of the purple and brown lesions on a potassium-deficient leaf of cv. Goldrush. Note the purple halo surrounding the brown necrotic lesions.

a

b

c

d

appears on all sorghum cultivars when potassium is deficient.

Infestations of corn leaf aphid (*Rhopalsiphum maidis*) may cause yellowish mottling and brown marginal necrosis on middle and younger leaves (Teetes et al. 1983), which should not be confused with the white, yellow, purple, and brown marginal symptoms (Plate 3a, b, c, d) developed on middle to older leaves when potassium is deficient.

Tissue analysis

Lockman (1972a, b, c), Reneau et al. (1983), and Reuter (1986) presented data on potassium concentrations for various parts and stages of growth of grain sorghum plants. Lockman (1972c) and Reuter (1986) classed potassium concentrations of 2.5–4.5% in the whole shoot of young plants (up to growth stage 3; collar of 8th leaf visible– Vanderlip and Reeves 1972), and 1.0–1.7% in upper leaves of plants at anthesis and during grain filling, as adequate for sorghum growth. Critical potassium concentrations of 2.1% for the cultivar M35-1 and 2.2% for Texas 610SR in the youngest emerged blade of plants at growth stage 3–4 (Vanderlip and Reeves 1972) were found in the present study. Weir (1983) reported a critical potassium concentration of 1.8% in the second blade below the head at the full heading stage of growth.

Soil analysis

Doll and Lucas (1973) reviewed the methods used in testing soils for potassium, and commented that 'at the present time there appears to be little justification for using any tests other than exchangeable plus water soluble potassium for soil testing'.

In many laboratories, neutral 1M ammonium acetate is used to measure extractable potassium, for which critical values ranging from 53 to 280 kg K/ha have been established in India (Singh and Dass 1984; Tandon and Kanwar 1984), and from 100 to 150 mg K/kg in Australia (G.H. Price, pers. comm.).

Magnesium deficiency

Symptoms

Plants suffering from magnesium deficiency lack vigour and are often stunted with thin stems. The leaves are usually pale green to yellow in colour with many brown lesions (Plate 4a). The symptoms develop firstly on older leaves and advance upwards to younger leaves. When the deficiency is severe, the whole plant appears pale green or pale yellow in colour (Plate 4a). The first symptom to develop is a yellow chlorosis which appears in interveinal tissues, usually about halfway along the leaf blade. However, there are large differences among the cultivars in the further development of symptoms.

In some cultivars, the veins remain dark green and the interveinal chlorosis extends towards the tip and base of the leaf blade, giving the blade a distinctive striped appearance (Plate 4b). In other cultivars, the veins fade in colour until the whole leaf blade turns yellow in colour. In yet other cultivars, orange, purple, or brown lesions develop within the chlorotic tissues (Plate 4c). Eventually these lesions expand and often join together until the margins of the leaf turn orange, red, or purple in colour, depending on the degree of pigmentation (Plate 4d). The boundaries between the healthy green tissue and the orange, purple, or brown lesions are distinct and sharp. When the deficiency is very severe, the margins of the older leaves turn brown, wither, and die.

The symptoms developed by some diseases may be confused with the orange, purple, or brown lesion symptoms (Plate 4c, d) appearing on some cultivars when magnesium is deficient. These diseases include leaf blight (*Helminthosporium turcicum*) and oval leaf spot (*Ramulispora sorghicola*) (Williams et al. 1978). However, these diseases never cause the development of interveinal chlorosis (Plate 4b, c) or marginal necrosis (Plate 4d) which occurs when magnesium is deficient.

Magnesium deficiency.

a   **Magnesium-deficient young plant of grain sorghum cv. E57.**

b   **Well developed interveinal chlorosis on a magnesium-deficient leaf of cv. Texas 610SR grown in solution culture. Note the onset of purple lesions within the chlorotic tissues.**

c   **Leaf of cv. Texas 610SR showing symptoms of severe magnesium deficiency. Note the uniform yellow colour of the leaf with prominent brown and purple lesions and red suffusion.**

d   **Comparison of magnesium-deficient leaves of cvv. Sundowner, Pride, Goldfinger, and Goldrush grown in solution culture.**

a

b

c

SUNDOWNER

PRIDE

Mg   GOLDFINGER

GOLDRUSH

d

Tissue analysis

Lockman (1972a, b, c), Gallaher et al. (1975), and Reuter (1986) presented data on the concentration of magnesium for various parts and stages of growth of grain sorghum plants. Lockman (1972c) and Reuter (1986) classed magnesium concentrations of 0.4–0.8% in whole shoots of young sorghum plants (up to growth stage 3; collar of 8th leaf visible –Vanderlip and Reeves 1972), and 0.1–0.5% in upper leaves of plants at anthesis and during grain filling, as adequate for sorghum growth. Critical magnesium concentrations of 0.16% for the cultivar M35-1 and 0.20% for Texas 610SR were found, in the present study, in the youngest emerged blade (YEB) of plants at growth stage 3–4 (Vanderlip and Reeves 1972), while at anthesis, the critical concentration was found to be 0.21% in the YEB of Texas 610SR. Weir (1983) reported a critical value of 0.15% in the second leaf below the head at the full heading stage of growth.

Soil analysis

Doll and Lucas (1973) reviewed the methods used in testing soils for magnesium and suggested that both neutral 1M ammonium acetate-exchangeable magnesium and the K/Mg ratio should be considered when interpreting the test results. In Europe, it is considered that the K/Mg ratio should be less than 5 on a weight basis, or less than 1.5 on an equivalent basis for the growth of field crops (Doll and Lucas 1973). In Australia, neutral 1M ammonium acetate-exchangeable magnesium levels of more than 100 mg Mg/kg to 200 mg Mg/kg are considered adequate for grain sorghum growth (G.H. Price, pers. comm.).

Salinity and toxicity of sodium, chlorine (as chloride), or sulfur (as sulfate)

Symptoms

Saline conditions occur when excessive levels of soluble salts, often salts of sodium (chloride or sulfate), are present in the root environment. The presence of high concentrations of soluble salts raises the osmotic pressure of the soil solution and predisposes plants to drought injury. In addition, toxic accumulations of one or more of the elements present in excess in the soil may occur, leading to the development of toxicity symptoms. Because toxicities of sodium, chlorine, and sulfur produce somewhat similar symptoms, tissue analysis may be needed to dintinguish between these disorders.

Addition of up to 50 mM sodium, 25 mM chlorine, or 50 mM sulfur to solution cultures (see footnote), produced similar symptoms. Plants became flaccid and appeared wilted. The leaves turned a dull green colour and became leathery to the touch. Initially, a grey discoloration developed near the tips of the older leaves and advanced along the margins. The margins sometimes rolled upwards, forming a tube along each edge of the leaf blade (Plate 5a). The grey discoloration did not persist and rapidly turned yellow, then brown, until the healthy green tissue around the midvein was outlined by marginal bands of yellow and brown (Plate 5b). These symptoms may be due to osmotically induced water stress, because similar symptoms were produced when the osmotic potential of nutrient solutions was lowered from −0.1 to −1.1 MPa by the addition of polyethylene glycol-6000 (Plate 5c).

When 100–200 mM sulfur was supplied, plants were severely stunted but did not develop symptoms additional to those described above. However, when 100–200 mM sodium was supplied, additional symptoms were observed. Plants had thick stems with short internodes (Plate 5d). A marginal chlorosis developed on the older leaves and small

Salinity, general osmotic effects, and toxicity of sodium and chlorine (as chloride).

a **Inward rolling of the margins on an old leaf of cv. Texas 610SR supplied with 100 mM chloride in nutrient solution.**

b **Lower leaf from cv. CSH 6 irrigated with highly saline water on a Vertisol at Coimbatore, India. Note the development of marginal chlorosis and necrosis.**

c **Leaves of cv. Texas 610SR showing marginal wilting, chlorosis, and necrosis; youngest (right) to oldest (left). Plants were grown in solution cultures in which the solution osmotic potential was lowered from − 0.1 to −1.1 MPa by the addition of polyethylene glycol-6000.**

d **Young plant of cv. Texas 610SR supplied with 200 mM sodium in solution culture.**

e **Lower leaf of cv. Texas 610SR showing marginal chlorosis and necrosis with small, grey necrotic lesions within the chlorotic tissue.**

a

b

c

d

e

grey necrotic lesions then appeared within the chlorotic tissue and the adjacent healthy tissue (Plate 5e). The boundary between the necrotic lesions and the surrounding tissue was sharp and distinct (Plate 5e). In time, a pale brown necrosis developed along the margins and extended inwards towards the midvein (Plate 5e). Eventually, the margins became completely necrotic, and circular to oval dark brown lesions appeared within the pale brown necrotic tissue (Plate 5f).

When 50 mM chlorine was supplied, characteristic symptoms appeared on the middle leaves. These leaves were bright green in colour and a thin dark reddish purple band developed on the margins in the basal half of the leaf blades. A pale yellow chlorosis developed in interveinal tissues near the tip of the leaf blade and advanced towards the base (Plate 5g). Eventually, rusty red lesions developed within the chlorotic tissue; these lesions were usually confined to the marginal areas of the leaf (Plate 5g).

Large excesses of chlorine (100–200 mM) greatly reduced growth and plants were severely stunted. However, the internodes were longer than those of sodium toxic plants, and stems were thin rather than thick as in sodium toxicity (compare Plate 5d and 5h). Dark red lesions developed within interveinal areas, mainly towards the margins and the leaf tips, on the blades of older leaves (Plate 5i).

f **Lower leaf of cv. Texas 610SR showing circular to oval dark brown lesions within the marginal necrotic tissue.**

g **Middle leaf of cv. Texas 610SR showing the early stages of chlorine toxicity symptoms in solution culture. Note the interveinal marginal chlorosis and red-purple lesions within the chlorotic tissue.**

h **Plant of cv. Texas 610SR supplied with 100 mM chlorine in solution culture.**

i **Older leaf of cv. Texas 610SR supplied with 200 mM chlorine in solution culture. Note the development of interveinal rusty-red lesions.**

FOOTNOTE: Plants were grown in solution cultures where either sodium, chlorine, or sulfur was supplied in the absence of an excess of the other two elements. Sodium was supplied as a mixture of sodium sulfate and sodium nitrate where the molar ratio of sulfate : nitrate was 1 : 23; chlorine as a mixture of calcium chloride, potassium chloride, and magnesium chloride where the molar ratio of calcium : potassium : magnesium was 1 : 2.05 : 0.25; and sulfur as a mixture of calcium sulfate, potassium sulfate, and magnesium sulfate where the molar ratio of calcium : potassium : magnesium was 1 : 2.05 : 0.25.

f

g

h

i

Tissue analysis

Data do not appear to have been published on concentrations of sodium, chlorine, or sulfur in sorghum plants suffering from a toxicity of any of these elements. Weir (1983) considered that concentrations of chloride of less than 2% in the second blade below the head at full heading were safe, while Francois et al. (1984) reported a critical concentration of 0.71% in the fourth blade below the flag leaf after heading.

In the present study, the following concentrations of sodium and chlorine in the youngest emerged blades were associated with 10 and 50% yield reduction in plants of the cultivar Texas 610SR at growth stage 3–4 (Vanderlip and Reeves, 1972).

Element	Level of yield reduction	
	10%	50%
Sodium (mg/kg)	30	50
Chlorine (%)	0.2	1.3

Reisenauer et al. (1973) reviewed the methods used in testing soils for chlorine and sulfur, while Lunt (1965) addressed the determination of sodium levels and Eaton (1965) considered techniques for measuring total salt and soil salinity.

Lunt (1965) commented that when the amount of exchangeable sodium exceeds 10–20% of the cation exchange capacity of a soil, soil physical properties begin to deteriorate and plant growth is adversely affected by both the poor physical conditions of the soil and the high level of exchangeable sodium. However, no specific data for sorghum production seem to be available.

Bruce and Rayment (1982) classed soils with an electrical conductivity between 0.9 and 2.0 mS/cm as high in soluble salts, and those above 2.0 mS/cm as very high. Evidence indicates that grain sorghum is only moderately tolerant of salinity, being more tolerant than wheat, cotton, or barley (Eaton 1942; Hart 1974). Hart (1974) found that sorghum yield was reduced by 50% at an electrical conductivity (saturation extract) of 12 mS/cm, while Maliwal (1967) suggested that sorghum cultivars may differ in their tolerance to salinity. Thus, Taylor et al. (1975) found that when 5000 mg/l of salt (approximately 10 mS/cm) was added to solution cultures, yield of the cultivar Midland was reduced by 71%, whereas yield of the cultivar Desert Maize was reduced by only 21%.

Critical values for sulfur or chloride toxicity in soils do not appear to have been published for sorghum production. On the other hand, Eaton (1965) reported that yield reductions of 25% are predicted for beans (sensitive) and barley (tolerant) with 9 and 170 mM chlorine respectively.

Soil analysis

Phosphorus toxicity

Symptoms

Plants supplied with a small excess of phosphorus often show no reduction in growth but develop purple to rusty-brown lesions on their dark green lower leaves (Plate 6a). These lesions are characteristic of phosphorus toxicity and develop initially near the tip of the leaf blade and advance along the margins towards the base. The lesions are irregular in shape and have sharp boundaries with the healthy green tissue (Plate 6b). Plants supplied with a large excess of phosphorus lack vigour and have short stout stems. The leaves are dark green in colour and the blades of lower leaves may be almost covered by reddish-purple lesions (Plate 6c). Eventually, the tips and margins of the blades turn brown, wither, curl upwards, and die (Plate 6d).

Clark (1982c) and Furlani et al. (1986a, b) described similar reddish purple lesions on plants growing in solution cultures, potted sand or soil, and in the field, and referred to them as 'red-speckling'. The level of phosphorus which induced toxicity symptoms depended on the cultural method, cultivar, age of the plants, and source of phosphate. With a susceptible cultivar, 'red-speckling' appeared 3 days after 16 μM potassium dihydrogen phosphate had been added to solution cultures of 7-day-old plants (Furlani et al. 1986b), and also 3 days after 5 μM ammonium dihydrogen phosphate had been added to solution cultures of 2-day-old seedlings of the cultivar Texas 610SR (N.J. Grundon, unpublished data).

The 'red-speckling' of phosphorus toxicity can be confused with the reddish purple or brown lesions developed by plants suffering from a severe deficiency of potassium or magnesium. However, there are differences which help to distinguish between these disorders. In magnesium deficiency, the lower leaves are yellow-green in colour (Plate 4a), whereas the lower leaves are dark green in colour in both potassium deficiency (Plate 3a) and phosphorus toxicity (Plate 6a). With potassium deficiency, the lesions are regular in outline and the reddish purple colour usually surrounds the brown necrosis (Plate 3d), whereas the lesions of phosphorus toxicity are irregular in outline (Plate 6b) and the brown necrosis is confined to the margins (Plate 6d).

Phosphorus toxicity.

a Appearance of plants of cv. Texas 610SR with 700 μM phosphate (left) and 70 μM phosphate (right) in solution culture. Note well developed symptoms on lower leaves of plants on the left, but no reduction in growth.

b Early stages of purple to rusty red interveinal lesions, known as 'red-speckling', on lower leaves of cv. Texas 610SR supplied with 350 μM phosphate in solution culture.

c 'Red-speckling' on lower leaves of plants of cv. Texas 610SR supplied with 1400 μM phosphate in solution culture.

d Lower leaves of cv. Texas 610SR supplied with 2800 μM phosphate in solution culture. (Phosphate supplied as a mixture of calcium tetrahydrogen phosphate, potassium dihydrogen phosphate, sodium dihydrogen phosphate, and ammonium dihydrogen phosphate where the molar ratio of calcium : potassium : ammonium : sodium was 1.00 : 1.93 : 0.71 : 0.71.)

a

b

c

d

Tissue analysis

Phosphorus toxicity does not occur frequently under field conditions, but is often observed in sorghum seedlings growing in nutrient solutions and in potted soils under greenhouse conditions (Clark 1982c; Furlani et al. 1986a, b; Grundon and Vance 1986). Kuo and Mikkelsen (1981) found phosphorus concentrations of more than 1% in whole shoots of 35-day-old sorghum plants suffering from phosphorus toxicity. Lockman (1972c) classed phosphorus concentrations greater than 0.5% in whole shoots of young plants (up to growth stage 3; collar of 8th leaf visible – Vanderlip and Reeves 1972) as high, while in plants at anthesis or during grain filling, concentrations of more than 0.35 and 0.25% respectively in the third leaf below the head were considered high. In the present study, we found that dry matter yields of the cultivar Texas 610SR plants at growth stage 3–4 (Vanderlip and Reeves 1972) were depressed by 10% when the phosphorus concentration in the youngest emerged leaf was 1.6%, and were depressed by 50% or more when the concentration was above 1.9%.

Soil analysis

Because phosphorus toxicity does not occur naturally in soils, and overfertilisation with phosphatic fertilizers is usually rare, no attempts have been made to develop a soil test for excess phosphorus.

Manganese toxicity

Symptoms

Plants suffering from manganese toxicity lack vigour and are slower to flower and mature. Growth is usually stunted and stems are thin. The foliage is dark green in colour, but young leaves may become pale yellow due to manganese-induced iron deficiency.

The characteristic symptom of manganese toxicity is the development of prominent, small, reddish-brown spots or lesions on the dark green older leaves (Plate 7a, b). These lesions develop initially in interveinal tissues towards the tip of the blade (Plate 7a, c), but may eventually extend over the full length of the blade (Plate 7b, c). Furthermore, they usually advance upwards to younger leaves as the severity of the toxicity increases, and may appear on all leaves when the toxicity is very severe (Plate 7c).

Clark et al. (1981) reported similar symptoms on grain sorghum plants grown in solutions supplied with 1600 μM manganous chloride, but also noted the development of necrotic lesions surrounded by dark red, irregularly shaped halos on the margins and tips of older leaves.

When the toxicity is severe, symptoms of manganese-induced iron deficiency may develop on the young leaves (Plate 7c). A pale yellow interveinal chlorosis develops in the basal tissues of the young leaf blades (Plate 7d), and in time, extends the full length of the young blades (Plate 7c). Another symptom which does not closely resemble those of iron deficiency, is the development of white necrotic lesions in the basal tissues of the young leaf blades (Plate 7e). Faint red streaks may appear near or within the white lesions (Plate 7e). Some cultivars develop a thin white lesion across the width of the leaf blades within the whorl.

Manganese toxicity.

a Reddish-brown necrotic spots developed on older leaves of cv. Texas 610SR supplied with 12 μM manganous chloride in solution culture.

b Severe manganese toxicity symptoms on old leaves of cv. Texas 610SR supplied with 48 μM manganous chloride in solution culture.

c Severe manganese toxicity in cv. Texas 610SR supplied with 480 μM manganous chloride in solution culture. Note the development of symptoms on both young (yellow chlorosis) and old (reddish-brown necrotic spots) leaves.

d Interveinal yellow chlorosis due to iron deficiency on young leaf of cv. Texas 610SR supplied with 8 μM manganous chloride in solution culture.

e White necrosis and red streaking presumably due to iron deficiency on young leaf of cv. Texas 610SR supplied with 32 μM manganous chloride in solution culture.

a

b

c

d

e

Tissue analysis

Manganese toxicity is sometimes encountered in sorghum growing on strongly acidic soils (Clark 1982a). Lockman (1972a, b, c), Clark et al. (1981), Kuo and Mikkelsen (1981), and Reuter (1986) presented data for high and toxic concentrations of manganese for various parts and stages of growth of grain sorghum plants. High concentrations ranged from over 70 to over 150 mg/kg in whole shoots of young plants (up to growth stage 3; collar of 8th leaf visible – Vanderlip and Reeves 1972), and above 50 mg/kg in upper leaves of plants at anthesis (Lockman 1972c). Kuo and Mikkelsen (1981) reported that concentrations above 860 mg/kg in whole shoots of 35-day-old plants were toxic, while in the present study, concentrations of 278 and 1850 mg/kg in the youngest emerged blade of the cultivar Texas 610SR at growth stages 3–4 (Vanderlip and Reeves 1972) were associated with dry matter yield depressions of 10 and 50% respectively.

Soil tests for manganese toxicity do not appear to have been developed for grain sorghum. However, studies of manganese toxicity with other crops have used soil pH, exchangeable manganese (extracted with neutral 1M amonium acetate, or 1M ammonium dihydrogen phosphate, or 0.5M calcium nitrate), and easily reducible manganese (extracted with 0.2% quinol or hydroquinone in neutral 1M ammonium acetate) as parameters of plant available manganese (Lohnis 1951; Fergus 1954; Lovett and Johnson 1968; Cheng and Ouellette 1971). Usually, there has been a better relationship between exchangeable manganese and plant growth than between easily reducible manganese and plant growth. However, neither parameter of plant available manganese could reliably predict soils likely to have manganese toxicity, and a combination of soil pH, exchangeable manganese, and easily reducible manganese was often used (Fergus 1954). Many workers advocated the use of test species sensitive to excessive manganese or of plant analysis rather than soil analysis as a means of predicting manganese toxicity (Lohnis 1951; Fergus 1954; Andrew and Hegarty 1968).

Manganese toxicity generally occurs on poorly drained soils high in manganese or on strongly acidic soils where it is associated with other problems of acid soil infertility such as toxicities of hydrogen ions, aluminium, and iron, and deficiencies of calcium, phosphorus, zinc, magnesium, and molybdenum (Clark 1982b; Adams 1984a). Correction of aeration or drainage problems in poorly drained soils, or liming of acid soils to a pH above about 5.3–5.5 decreases the solubility of manganese and frequently overcomes manganese toxicity for sorghum production on such soils (Adams 1984a).

Manganese-induced iron deficiency has been attributed to direct competition between manganese and iron for a position within the haem nucleus of certain iron-containing enzymes (Weinstein and Robbins 1955), or within protoporphyrin-9, the chlorophyll precursor (Sideris and Young 1949). Therefore, it has been sometimes possible to overcome manganese toxicity by increasing the iron concentration in the nutrient solution (Hiatt and Ragland 1963), or by applying foliar sprays of ferrous sulfate to crops growing on acid soils (Fergus 1954).

Soil analysis

Zinc toxicity

Symptoms

Plants affected by a mild zinc toxicity have reduced height and mature slowly. When the toxicity is very severe, the stems are very short (Plate 8a) and the lower leaf sheaths often pull away from the stem to give the appearance of an open fan. A pale yellow interveinal chlorosis develops between the margins and midvein on the blades of older leaves. A pale grey necrosis sometimes develops within the chlorotic tissues, but usually streaks of red, brown, purple, or orange pigmentation appear within or near the chlorotic tissues (Plate 8b). The boundary between the healthy green tissue and the chlorotic or pigmented tissue is very diffuse (Plate 8b). When the toxicity is very severe, dark reddish brown, necrotic lesions may develop near the margins on older leaves. These necrotic lesions often join together to produce a marginal necrosis (Plate 8c). The boundary between the necrotic tissue and the remainder of the leaf is sharp and distinct (Plate 8c).

Another set of symptoms may appear on younger leaves and resemble those of iron deficiency; a pale yellow interveinal chlorosis develops in the basal tissues of the young leaf blades, and advances towards the leaf tip until it extends over the full length of the leaf blade (Plate 8a).

Zinc toxicity.

a   **Response of plants of cv. Texas 610SR to zinc supply in solution culture; 240 μM zinc sulfate (left), 16 μM zinc sulfate (centre), 2 μM zinc sulfate (right). Note the development of zinc-induced iron deficiency at the intermediate level of zinc.**

b   **Red, purple, brown, and orange pigmentation on old leaf of cv. Texas 610SR supplied with 240 μM zinc sulfate in solution culture.**

c   **Brown marginal necrosis developing within the red, purple, and brown pigmented tissues on an old leaf of cv. Texas 610SR supplied with 240 μM zinc sulfate in solution culture.**

a

b

c

Tissue analysis

Zinc toxicity is not a common occurrence in soils used for sorghum production but may occur on some acid soils high in zinc, or on soils overfertilized with zinc fertilizers or foliar zinc sprays. Nevertheless, some data are available on possible toxic concentrations of zinc in various tissues of sorghum plants at different stages of growth. Clark et al. (1981) found a zinc concentration of 270 mg/kg in whole shoots of 24-day-old plants suffering from zinc toxicity, whereas Boawn and Rasmussen (1971) reported concentrations of 470–570 mg/kg in whole shoots of zinc toxic 35-day-old plants. In whole shoots of young plants, Lockman (1972c) classed zinc concentrations above 60 mg/kg as high, while Agarwala and Sharma (1979) classified concentrations above 30 mg/kg in middle blades of 49-day-old plants as high. In the present study, we found that zinc concentrations of 60 and 445 mg/kg in the youngest emerged blade were associated with depressions of dry matter production of 10 and 50% respectively in plants of the cultivar Texas 610SR at growth stage 3 (collar of 8th leaf visible–Vanderlip and Reeves 1972).

Soil analysis

Viets and Lindsay (1973) reviewed the methods used in testing soils for zinc but made no mention of methods which could be used to test for zinc toxicity.

Although soil data for zinc toxicity do not appear to be available for sorghum, some information does exist for maize. Barnette (1936) found that 400 mg/kg of exchangeable zinc was toxic, while Gall and Barnette (1940) reported that 890 mg/kg was toxic in pot trials. In the present study, sorghum plants developed zinc toxicity symptoms in solution cultures when supplied with 16 μM zinc chloride.

Disorders producing symptoms mainly on upper leaves

Calcium deficiency

Symptoms

Plants suffering from mild calcium deficiency or from a temporary shortage of calcium supply may show little depression in growth, but develop a characteristic 'torn leaf' symptom on their upper leaves (Plate 9a). These younger leaves are short and are often held erect. The margins of the leaf blade usually turn pale green or pale yellow and tear easily to give a characteristic serrated or saw-tooth appearance (Plate 9a, b). In some cultivars, the leaf tips become deformed and curl to form sword-like tips, or bend down and break off leaving blunt tips to the leaves (Plate 9c).

When the deficiency becomes severe, growth is greatly depressed (Plate 9d). The upper internodes may be very short and the young leaves crowded together to give the appearance of a rosette. The young leaves often fail to unroll from the whorl and a number of leaves may become joined together at their tips (Plate 9e). As the leaves try to expand, the blades become folded and sometimes assume a laddered appearance.

The apical meristem often dies when calcium is severely deficient. When this occurs, many tillers develop from the basal nodes (Plate 9c, d, e) and the newly formed young leaves on these tillers show symptoms of severe calcium deficiency (Plate 9c, e).

Flowering and maturity are delayed by mild calcium deficiency, but head size is often unaffected. However, when calcium deficiency is severe, the head may not form or it may be partly barren (Plate 9d).

Calcium deficiency.

a Young leaves of a calcium-deficient grain sorghum plant cv. RS671.

b Calcium-deficient leaves of cv. M35-1 grown in solution culture. Note the serrated, torn margins.

c Young plant of cv. Texas 610SR showing symptoms of severe calcium deficiency. Note the development of basal tillers and the torn and deformed leaves.

d Response to calcium in solution culture by cv. Texas 610SR (left, deficient; right, adequate).

e Severely calcium-deficient plant of cv. Texas 610SR grown in solution culture. Note that young leaves of the primary shoot are joined together at their tips and also note the development of basal tillers.

a

M35-1 -Ca

b

c

d

e

Tissue analysis

Lockman (1972a, b, c) and Reuter (1986) presented data on the calcium concentration for various parts and stages of growth of grain sorghum plants. Lockman (1972c) and Reuter (1986) classed calcium concentrations of 0.9–1.5% in whole shoots of young plants (up to growth stage 3; collar of 8th leaf visible – Vanderlip and Reeves 1972), and 0.2–0.6% in upper leaves of plants at anthesis and during grain filling, as adequate for sorghum growth. The critical calcium concentration in the youngest emerged blade of Texas 610SR plants at anthesis was found, in this present study, to be 0.34%. Weir (1983) reported a critical value of 0.2% in the second leaf below the head in plants at the full heading stage of growth.

Soil analysis

Calcium is usually the dominant basic cation in soils and only in strongly acid soils is the level of calcium so low that it becomes limiting as a plant nutrient (Doll and Lucas 1973). Liming of acid soils to raise pH also corrects any calcium deficiency. McLean (1973) has reviewed the methods used for determining the lime requirement of acid soils, while the subjects of soil acidity and the use of liming materials were comprehensively addressed in the book edited by Adams (1984b). Liming to achieve a soil pH from 5.3 to 5.5 appears to supply sufficient calcium for the growth of grain sorghum (Adams 1984a).

Sulfur deficiency

Symptoms

Affected plants lack vigour, are stunted, pale green to yellow in colour, and have thin stems (Plate 10a). The blades of young leaves are shorter and held more erect than usual. Initially, these blades turn pale green in colour while the older leaves remain dark green (Plate 10b). A pale yellow chlorosis develops in the basal tissues of these young leaves (Plate 10b) and advances in a broad front towards the tip until the whole blade turns a uniform pale yellow colour (Plate 10c). In some cultivars, the chlorosis advances more rapidly down the interveinal tissues and a pale interveinal chlorosis appears (Plate 10d) before the blades turn a uniform yellow colour.

When the deficiency is prolonged or very severe, a second set of symptoms may develop on the older leaves of some cultivars. Initially, the older leaves turn from dark to pale green in colour. A dark yellow chlorosis then develops near the tip of the leaf blade and advances along the margins towards the base. Eventually, the chlorotic margins turn brown, and these older leaves wither, die, and hang down around the lower stem (Plate 10c).

Sulfur deficiency.

a   Response of cv. Pacific 710 to sulfur supply in solution culture. Compare the dark green healthy plants (left) with the pale yellow deficient plants (right).

b   Sulfur-deficient young grain sorghum plant of a local cultivar growing on an Alfisol at Hyderabad, India. Note the deficient, pale yellow younger leaves and healthy, dark green lower leaves.

c   Severely sulfur-deficient plant of cv. Texas 610SR grown in solution culture. Note the development of symptoms on both young (pale yellow chlorosis) and old (brown necrosis) leaves.

d   Close-up of sulfur-deficient young leaf from a local cultivar growing on an Alfisol at Hyderabad, India. Note the pale yellow interveinal chlorosis.

a

b

c

d

Tissue analysis

Lockman (1972a, b, c) and Reuter (1986) presented data on the concentration of sulfur for various parts and stages of growth of grain sorghum plants. Adequate sulfur concentrations were 0.24% in whole shoots of young plants (up to growth stage 3; collar of 8th leaf visible – Vanderlip and Reeves 1972) and 0.16% in the youngest emerged blades of plants at anthesis. Critical sulfur concentrations at several stages of growth are shown in Table 5.

TABLE 5   Concentrations of sulfur in grain sorghum corresponding to 90% maximum yield.

Growth stage[a]	Plant part[b]	S conc (%)	Reference
3–4	YEB	0.20	This study cv. M35-1
3–4	YEB	0.25	This study cv. Texas 610SR
8th leaf	YEB	0.27	Brar and Takkar (unpubl.)
9th leaf	YEB	0.23	Brar and Takkar (unpubl.)
10th leaf	YEB	0.20	Arora and Takkar (unpubl.)
6	YEB	0.21	This study cv. Texas 610SR
6	2BBH	100 mg/kg SO_4-S	Vance (1981)
6–7	2BBH	0.15	Weir (1983)

[a]As in Vanderlip and Reeves (1972).
[b]YEB = youngest emerged blade; 2BBH = second blade below head.

Soil analysis

Reisenauer et al. (1973) reviewed the methods used in testing soils for sulfur and found that the two most frequently used extractants were water and 0.01M monocalcium phosphate. Based on a limited number of calibration studies, Reisenauer et al. (1973) concluded that the critical value of extractable soil sulfur for the normal growth of most cereal crops ranged from 6 to 12 mg/kg. In India, critical values of sulfur for sorghum production were found to be 8 mg/kg water-extractable sulfur (Badhe and Lande 1980), and from 7 to 10 mg S/kg by extraction with 0.15% calcium chloride (C.L. Arora, M.S. Brar, and P.N. Takkar, unpubl.).

Iron deficiency

Symptoms

Mild iron deficiency can produce characteristic symptoms on younger leaves without greatly reducing growth. If the deficiency is also temporary, it is quite common to see iron deficiency symptoms on leaves midway up the stem, with healthy green leaves both above and below the affected leaves. Severely affected plants may be pale green, yellow, or almost white in colour, and have thin stems (Plate 11a).

The characteristic symptom of iron deficiency is a pale yellow interveinal chlorosis which develops on the younger leaves. The chlorosis begins in interveinal tissues near the base of the leaf blade and eventually extends the full length of the blade (Plate 11b). The distinctive striping of the leaf is caused by the sharp boundaries between the dark green veins and the yellow interveinal tissues (Plate 11c). However, as the severity of the deficiency increases, the veins fade in colour and turn pale yellow. Eventually, white necrotic areas develop within the chlorotic tissues and the whole leaf blade turns pale yellow to white in colour. In some cultivars, pale red streaks may develop within the yellow chlorotic tissues, while in other cultivars, long brown lesions may develop within the white necrotic areas, and large lenticular sections between the midvein and the margin may wither and die.

Toxicities of a number of heavy metals, including manganese (Plate 7d), zinc, mercury (Plate 11d), aluminium (Plate 16b), cobalt, copper, nickel, and lead, may cause symptoms closely similar to those of iron deficiency to appear, perhaps as a result of interference with the uptake or metabolism of iron in the sorghum plant. In some cultivars, the symptoms of the very early stages of sulfur deficiency (Plate 10b, d) may appear as a faint interveinal chlorosis on the young leaves which could be confused with iron deficiency (Plate 11b, c). However, the subsequent pattern of development of the symptoms is different; in iron deficiency, the interveinal chlorosis becomes more prominent (Plate 11c) before fading to a yellow then white chlorosis, whereas in sulfur deficiency, the interveinal chlorosis becomes less distinct and fades rapidly to a uniform yellow, but never white, chlorosis (Plate 10b, d).

Systemic infection by sorghum downy mildew (*Sclerospora sorghi*) may produce yellow and white streaks and stripes on young leaves (Williams et al. 1978) which resemble the
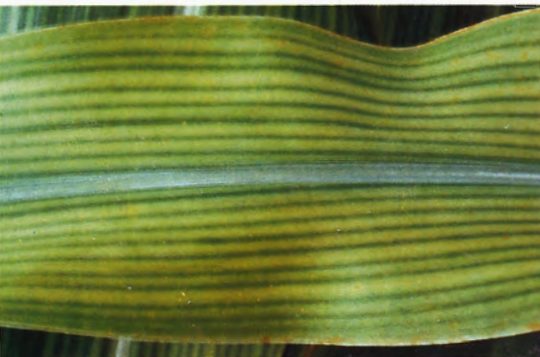
Iron deficiency.

a Iron deficient sorghum crop near Garden City, Kansas, USA.

b Young grain sorghum plant of a local cultivar growing on a Vertic Inceptisol at Anand, India. Note iron deficiency symptoms on youngest leaves and healthy dark green older leaves.

c Interveinal chlorosis on young leaves of a local cultivar growing on a calcareous Inceptisol at Coimbatore, India.

d Iron deficiency symptoms in a young leaf of cv. Texas 610SR supplied with 24 μM mercuric chloride in nutrient solution.

a

b

c

d

symptoms of iron deficiency. The presence of downy white fungal growth on the under surface of infected leaves (Williams et al. 1978) distinguishes attack by sorghum downy mildew from iron deficiency.

Tissue analysis

Lockman (1972a, b, c), Brown and Jones (1977), Salardini and Murphy (1978), Clark et al.(1981), and Reuter (1986) presented data on the concentration of iron for various parts and stages of growth of grain sorghum plants. Lockman (1972c) and Reuter (1986) classed iron concentrations of 33–250 mg/kg in whole shoots of young plants (up to growth stage 3; collar of 8th leaf visible – Vanderlip and Reeves 1972), and 40–200 mg/kg in upper leaves of plants at anthesis and during grain filling, as adequate for sorghum growth. de Boer and Reisenauer (1973) found a critical iron concentration of 65 mg/kg in the whole shoots of 35-day-old plants, while Takkar et al. (1986) reported a critical value of 45 mg/kg in the fourth and fifth leaves of 20-day-old plants. In the present study, we found a critical concentration of 88 mg/kg in the youngest emerged blade of the cultivar Texas 610SR at anthesis.

Soil analysis

Viets and Lindsay (1973) reviewed the methods used in testing soils for iron and recommended the use of the DTPA test which was subsequently described in greater detail by Lindsay and Norvell (1978). On the basis of a greenhouse pot experiment, a critical value of 4.5 mg/kg DTPA-extractable iron was found to separate responsive from non-responsive soils (Lindsay and Norvell 1978). In Australia, a value of 2 mg/kg DTPA-extractable iron is used (G.H. Price, pers. comm.), while in India, critical values for the DTPA extraction range from 4.5 mg Fe/kg to 6.4 mg Fe/kg (Takkar and Mehta 1986; Takkar et al. 1986; R. Ramadoss and R.P. Ramaswami, unpubl.).

Copper deficiency

Symptoms

A deficiency of copper depresses growth and affected plants become stunted with thin stems and pale green foliage (Plate 12a). The symptoms appear only on leaves which are still within the whorl and on young mature leaves. In mild deficiencies, these leaves turn pale green in colour and an interveinal pale yellow chlorosis develops at the base of the blades (Plate 12b). The whorl of expanding leaves may remain tightly rolled and become bent to one side (Plate 12b).

When the deficiency is severe, a pale brown necrosis develops at the tips of the youngest leaves. The affected leaf tips wither and die, often twisting into a spiral shape. The basal tissue of the leaf blade usually remains green, and the twisted brown tip hangs down from the green basal tissue (Plate 12c). In very severe deficiencies, this symptom develops on leaves still within the whorl and a number of young leaves often become joined together at their tips to give a laddered pattern (Plate 12d) resembling that of calcium deficiency (Plate 9e).

Somewhat similar symptoms were described by Brown et al. (1977) and Clark et al. (1981) and attributed to copper deficiency. These authors suggested that copper deficiency may, in reality, be calcium deficiency. However, the symptoms of copper deficiency and calcium deficiency are readily distinguishable. The twisted, dead leaf tips which characterise copper deficiency do not develop when calcium supply is limited, while the characteristic serrated, saw-toothed margins of calcium deficiency do not occur when copper supplies are inadequate.

Copper deficiency.

a **Response of cv. M35-1 to copper in a copper-deficient Vertisol. Rates of copper applied were (left to right) 0, 8.8, and 28.2 mg Cu/pot (equivalent to 0, 5, and 16 kg Cu/ha). Note the marked depression in growth and paler colour of the copper-deficient plants (centre and left).**

b **Mild copper deficiency in cv. Texas 610SR growing in solution culture. Note the shortened upper internodes which cause the young leaves to become crowded together, the pale green to yellow colour of the unopened leaves in the whorl, and the young leaves bent to one side.**

c **Brown necrotic tip on the blade of a young copper-deficient leaf of cv. M35-1 grown in copper-deficient soil.**

d **Dead leaf tips which remain joined together in a copper-deficient young plant of cv. M35-1.**

a

b

c

d

Tissue analysis

Lockman (1972a, b, c), Brown and Jones (1977), Clark et al. (1981), and Reuter (1986) presented data on concentrations of copper for various parts and stages of growth of grain sorghum plants. Lockman (1972c) and Reuter (1986) classed copper concentrations of 8–27 mg/kg in whole shoots of young plants (up to growth stage 3; collar of 8th leaf visible–Vanderlip and Reeves 1972), and 1–15 mg/kg in upper leaves of plants at anthesis and during grain filling, as adequate for sorghum growth. Agarwala and Sharma (1979) reported a critical copper concentration of 3.6 mg/kg in the middle blades of 30-day-old sorghum plants, while, in the present study, we found a critical concentration of 2.7 mg/kg in the youngest emerged blade of the cultivar Texas 610SR at anthesis.

Soil analysis

Viets and Lindsay (1973) reviewed the methods for testing soils for copper and recommended the use of DTPA as an extractant. In a subsequent paper, Lindsay and Norvell (1978) described the DTPA method in detail and proposed a tentative critical value of 0.2 mg/kg DTPA-extractable copper for grain sorghum. In Australia, a critical value of 0.3 mg/kg DTPA-extractable copper is used (G.H. Price, pers. comm.).

Zinc deficiency

Symptoms

Zinc deficiency causes the internodes to become very short, resulting in stunted plants (Plate 13a, b). Flowering and maturity are also delayed by zinc deficiency (Plate 13a). The first symptoms on the foliage appear on the youngest leaves even before they unroll from the whorl; a pale yellow chlorosis develops between the margins and midvein near the base of the leaf blade (Plate 13b). As the deficiency becomes more severe, the chlorosis extends towards the leaf tip and often turns nearly white (Plate 13c) or sometimes pale brown. The midvein and margins usually remain green and the boundary between the healthy green tissue and the chlorosis is diffuse.

In some cultivars, a reddish-purple or brown pigmentation which is often seen more easily from underneath the unrolling leaf develops adjacent to the yellow chlorosis in the basal tissue of the blades of the young leaves (Plate 13d). Similar symptoms have been reported previously by Clark et al. (1981), who also described the development of dark red bands on the margins of the affected leaves.

When a severe zinc deficiency continues for a relatively long time, symptoms develop also on some of the older leaves about halfway down the stem. A white or brown necrosis and purple pigmentation develop in interveinal tissues near the tip of affected leaves (Plate 13e). The necrosis and pigmentation advance towards the leaf base, but usually extend only about halfway down the leaf blade. The boundary between the healthy green tissue and the necrotic tissue is sharp (Plate 13e). Ohki (1984) reported similar symptoms in zinc-deficient grain sorghum plants, but noted that the brown necrosis was preceded by an interveinal chlorosis which extended the full length of the leaf blade.

Infections of grain sorghum plants by maize dwarf mosaic virus and sugarcane mosaic virus cause the development of an irregular mottling of dark and light green, often interspersed with longitudinal white or yellow streaks (Williams et al. 1978). Sometimes these symptoms may resemble the yellow chlorosis developed in the base of the young leaves of zinc-deficient plants (Plate 13c), but usually the chlorotic streaks of viral infection are far more irregular in shape and are not confined to the basal area of the leaf, being scattered over the full length of the leaf blade.

Zinc deficiency.

a **Response of cv. Texas 610SR to zinc supply in solution culture. Note the stunted growth and delayed maturity of the zinc-deficient plants on the right.**

b **Zinc-deficient plant of a local cultivar growing on an Alfisol at Hyderabad, India.**

c **Close-up of the base of the blade of a zinc-deficient leaf showing broad, chlorotic bands on either side of the midvein.**

d **Close-up of the underside of a young leaf of a zinc-deficient plant of cv. CSH 9 growing on an Alfisol at Hyderabad, India. Note the brown coloration developed adjacent to the yellow chlorosis.**

e **Interveinal white and brown necrosis and purple pigmentation developed on middle leaves of cv. Texas 610SR grown in solution culture.**

a

b

c

d

e

Tissue analysis

Boawn et al. (1960), Boawn and Rasmussen (1971), Lockman (1972a, b, c), Shukla et al. (1973), Brown and Jones (1977), Agarwala and Sharma (1979), Clark et al. (1981), Ohki (1984), and Reuter (1986) have reported zinc concentrations for various parts and stages of growth of grain sorghum plants. Lockman (1972c) and Reuter (1986) classed zinc concentrations of 40–70 mg/kg in whole shoots of young plants (up to growth stage 3; collar of 8th leaf visible– Vanderlip and Reeves 1972), and 7–40 mg/kg in upper leaves of plants at anthesis and during grain filling, as adequate for sorghum growth. Critical zinc concentrations at several stages of growth are shown in Table 6.

T A B L E 6   Concentrations of zinc in grain sorghum corresponding to 90% maximum yield.

Growth stage[a]	Plant part[b]	Zn conc (mg/kg)	Reference
49 DAS	MB	20	Agarwala and Sharma (1979)
63 DAS	MB	15	Agarwala and Sharma (1979)
3–4	YEB	10	Ohki (1984)
3–4	YEB	8.6	This study cv. Texas 610SR
9th leaf	YEB	9	Bansal & Takkar (unpubl.)
6–7	2BBH	18	Weir (1983)

[a]Growth stages as in Vanderlip and Reeves (1972). DAS = days after sowing.
[b]MB = middle blades; YEB = youngest emerged blade; 2BBH = second blade below head.

Viets and Lindsay (1973) reviewed the methods of testing soils for zinc and recommended the use of DTPA as an extractant. Lindsay and Norvell (1978) described the DTPA method in detail, and, on the basis of glasshouse pot trials, set a critical value of 0.6 mg/kg DTPA-extractable zinc for sorghum growth. Two critical values are used in Australia, depending on the pH of the soil. When the pH is less than 7.0, a critical value of 0.3 mg/kg DTPA-extractable zinc is used, whereas a value of 0.8 mg/kg is employed for soils with a pH greater than 7.0 (G.H. Price, pers. comm.). In India, critical values were found to range from 0.3 to 3.2 mg Zn/kg as determined by DTPA extraction (Singh and Dass 1984; and unpublished data of P.N. Takkar, I.M. Chibba, R.L. Bansal, V.V. Subbaiah, A.P. Rao, Z. Ahmed, P.N. Cherry, R.T. Dangawala, A.J. Patel, M.F. Raj, and V. George), and from 0.5 to 0.8 mg Zn/kg as determined by dithizone extraction (Deshmukh et al. 1974; Khan and Zende 1976).

Soil analysis

Manganese deficiency

Symptoms

Manganese-deficient plants are pale green to yellow in colour and have thin spindly stems. If the deficiency is only mild, growth may be depressed, but flowering and maturity will not be greatly delayed (Plate 14a). However, severe deficiencies can cause death of young plants.

The symptoms develop on young leaves which may be still emerging from the whorl (Plate 14b). Initially, small discrete chlorotic lesions develop between the veins, usually about midway along or towards the tip of the blade. These chlorotic lesions join together to produce a faint interveinal chlorosis (Plate 14c, d) which often affects only a short length of the leaf blade but sometimes may extend almost the full length of the blade. Small, discrete dark reddish brown or black lesions develop within the chlorotic tissues (Plate 14c, d). Eventually, these brown or black lesions join together producing brown interveinal stripes over the length affected by the interveinal chlorosis (Plate 14e).

In a mild deficiency, the veins usually remain green, but when the deficiency is severe, the brown lesions often join together across the minor veins to form necrotic areas covering large sections of the leaf blade. The boundary between the healthy green tissue and the white, yellow, or brown lesions is sharp (Plate 14b, c, d, e).

Ohki (1974) reported similar symptoms but believed that the reddish brown lesions which had not previously been associated with manganese deficiency, may have been a characteristic of the cultivar DeKalb 'BR64'. Our experience indicates that the reddish brown lesions may be rather common, because they were present on all cultivars when Texas 610SR, Pride, Goldrush, Goldfinger, Pacific 710, Sundowner, E57, and M35-1 were grown in solution cultures deficient in manganese.

Manganese deficiency.

a Response of plants of cv. Texas 610SR to manganese supply in solution culture. Note the depressed growth of the manganese-deficient plants (left) at similar stage of maturity.

b Manganese-deficient young plant of cv. E57 grown in solution culture. Note the symptoms on the younger leaves, with the older leaves remaining healthy.

c Close-up of manganese-deficient leaf of cv. Texas 610SR showing early stages of interveinal chlorosis and brown necrosis.

d Close-up of white chlorotic lesions and dark brown to black necrosis on a manganese-deficient leaf of cv. E57 grown in solution culture.

e Advanced symptoms of reddish brown necrotic lesions on a manganese-deficient leaf of cv. Texas 610SR grown in solution culture.
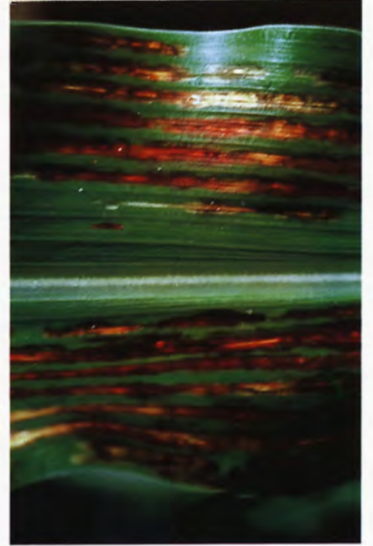
Tissue analysis

Lockman (1972a, b, c), Ohki (1974), Agarwala and Sharma (1979), Clark et al. (1981), Kuo and Mikkelsen (1981), and Reuter (1986) reported concentrations of manganese for various parts and stages of growth of grain sorghum plants. Lockman (1972c) and Reuter (1986) classed manganese concentrations of 40–150 mg/kg in whole shoots of young plants (up to growth stage 3; collar of 8th leaf visible– Vanderlip and Reeves 1972), and 8–190 mg/kg in upper leaves of plants at anthesis and during grain filling, as adequate for sorghum growth. Critical manganese concentrations at several stages of growth are shown in Table 7.

T A B L E   7   Concentrations of manganese in grain sorghum corresponding to 90% maximum yield.

Growth stage[a]	Plant part[b]	Mn conc (mg/kg)	Reference
63 DAS	MB	15	Agarwala and Sharma (1979)
9th leaf	YEB	12	Sadana and Takkar (unpubl.)
10th leaf	YEB	10	Sadana and Takkar (unpubl.)
5	YEB	10	Ohki (1974)
5	2BBH	15	Ohki (1974)
6	YEB	29	This study cv. Texas 610SR

[a]Growth stages as in Vanderlip and Reeves (1972). DAS = days after sowing.
[b]YEB = youngest emerged blade; MB = middle blades; 2BBH = second blade below head.

Soil analysis

Viets and Lindsay (1973) reviewed the methods used for testing soils for manganese and recommended the use of DTPA as an extractant. In a later paper, Lindsay and Norvell (1978) described the DTPA method in detail and proposed a tentative critical value of 1 mg/kg DTPA-extractable manganese. In India, critical values ranging from 3–7 mg/kg DTPA-extractable manganese have been established for grain sorghum (V.K. Nayyar, U.S. Sadana, and P.N. Takkar, unpublished data), while, in Australia, a critical value of 2 mg/ kg DTPA-extractable manganese is used (G.H. Price, pers. comm.).

Boron deficiency

Symptoms

A deficiency of boron depresses growth and causes shortening of the internodes. It may lead to severe stunting (Plate 15a). If heads are produced, they may be partly or completely barren (Plate 15b). The leaves are short, dark green in colour, and held erect (Plate 15a). They are often brittle and easily torn or broken. When the deficiency is severe, the apical meristem often dies and tillers develop (Plate 15a). If the deficiency occurs just before the boot stage of growth, the tillers may appear from nodes well above ground level.

Characteristic white or transparent lesions develop in interveinal tissues on the young leaves (Plate 15c), often while they are still within the whorl. Initially, the lesions develop in an intermittent pattern and appear firstly in the midsection of the leaf blade (Plate 15d). The boundary between the healthy green tissue and the white lesions is usually diffuse (Plate 15d). As the deficiency increases in severity, these lesions become almost transparent and join together. In some cultivars, the transparent lesions may extend beyond the midsection of the blade (Plate 15d, e). The veins appear to remain intact and green, even when the deficiency is very severe (Plate 15e).

Boron deficiency.

a Severely boron-deficient young plant of cv. Texas 610SR grown in solution culture. Note the development of tillers from the base of the plant and the appearance of deficiency symptoms on the youngest leaves.

b Comparison of heads from healthy (left) and boron-deficient (right) plants of cv. E57 grown in solution culture. Note the reduced head size, and evidence of missing florets on the deficient plant.

c Boron-deficient plants of cv. Texas 610SR grown in solution culture.

d Development of white to transparent interveinal lesions in an intermittent pattern in the midsection of a boron-deficient young leaf of cv. Texas 610SR grown in solution culture.

e Well developed transparent interveinal lesions on a boron-deficient leaf of cv. M35-1 grown in solution culture. [The apparent pink colour of the lesions is caused by the red colour of the background showing through the transparent lesions].

a

b

c

d

e

Tissue analysis

Lockman (1972a, b, c), Agarwala and Sharma (1979), Clark et al. (1981), and Reuter (1986) presented data on the concentration of boron for various parts and stages of growth of grain sorghum plants. Lockman (1972c) and Reuter (1986) classed boron concentrations of 1–25 mg/kg in whole shoots of young plants (up to growth stage 3; collar of 8th leaf visible–Vanderlip and Reeves 1972), and 1–10 mg/kg in upper leaves of plants at anthesis and during grain filling, as adequate for sorghum growth. Agarwala and Sharma (1979) reported a critical boron concentration of 10 mg/kg in middle blades of 63-day-old grain sorghum plants, while Weir (1983) found a critical concentration of 5 mg/kg in the second blade below the head at the full heading stage of growth.

Soil analysis

Reisenauer et al. (1973) reviewed the methods used to test soils for boron and suggested that soils which contained from 1 to 5 mg/kg hot-water-soluble boron would supply sufficient boron for normal growth of most field crops.

Disorders producing no specific leaf symptoms

Aluminium toxicity

Symptoms

Aluminium toxicity can severely restrict crop growth on strongly acidic soils $(pH[H_2O] < 5.0)$, sorghum being more sensitive than maize (Brenes and Pearson 1973) but less sensitive than sunflower (Blamey et al. 1986). Substantial differences in sensitivity to aluminium toxicity exist among sorghum cultivars (Plate 16a) (Furlani and Clark 1981).

In sorghum and most other crops, there are no specific leaf symptoms associated with aluminium toxicity, although a specific symptom has been reported for cassava (Asher et al. 1980). However, aluminium-induced symptoms of phosphorus, calcium, iron, and magnesium deficiency have been observed on some sorghum cultivars (Clark et al. 1981; Clark 1982b; Blamey et al. 1986; Frederiksen 1986), presumably due to impaired growth and function of the roots. These symptoms, an example of which is given in Plate 16b, are of little diagnostic value because they are not specific to aluminium toxicity.

Severe aluminium toxicity is associated with strong depression of root extension and fine lateral root development, all roots being short and thick, and often brown or black in colour (Plate 16c, d). Loss of apical dominance when root apices are injured or killed leads to a bunching of stubby lateral roots close to the tips of the main roots (Plate 16d). The number of nodal roots may be increased but their extension growth may be quite limited.

External concentrations of aluminium too low to produce clearly recognisable root symptoms may still cause substantial reductions in the growth of roots and shoots. Thus, in a carefully controlled solution culture study of Blamey et al. (1986), an aluminium concentration of only 16 μM reduced the dry weight of tops (Plate 16e) and roots by about 72% without producing clear root symptoms.

Aluminium toxicity.

a Response of grain sorghum cultivars which are tolerant (left) and susceptible (right) to excess soluble aluminium. The plants are growing on a highly acidic soil in Brazil.

b Young plant of cv. Texas 610SR grown in solution culture containing 16 μM monomeric aluminium. Note the presence of iron deficiency symptoms on young leaves and magnesium deficiency symptoms on old leaves.

c Aluminium toxicity on roots of grain sorghum supplied with high (right) and medium (centre) levels of aluminium, and no aluminium (left).

d Close-up of roots of grain sorghum supplied with high levels of aluminium in solution cultures.

e Response of cv. Texas 610SR to 0.4 μM (left) and 16 μM (right) monomeric aluminium in solution cultures.

a

b

c

d

e

Tissue analysis

Lockman (1972a, b, c), Brown and Jones (1977), and Reuter (1986) presented data on concentrations of aluminium for various parts and stages of growth of grain sorghum plants. Usually, plant analysis is not a very reliable method of diagnosing aluminium toxicity in crop plants, because of variable relationships between concentration in the tissues and growth. For example, Brown and Jones (1977) reported that growth was not affected at an aluminium concentration of 72 mg/kg in whole shoots of 18-day-old plants, whereas Lockman (1972c) classed concentrations above 70 mg/kg in whole shoots of young plants (growth stage 2–3 — Vanderlip and Reeves 1972) as high. In plants at anthesis, aluminium concentrations above 225 mg/kg in upper leaves were classed as high, while 0–25 mg/kg was considered unlikely to affect growth of plants at the grain-filling stage of development (Lockman 1972c).

Soil analysis

Aluminium becomes more soluble as the soil becomes more acidic (Thomas and Hargrove 1984). In many soils with pH values below 5.0, the concentration of aluminium in the soil solution reaches levels which are toxic to the growth of many crop plants (Adams 1984a). Attempts to predict the incidence and intensity of aluminium toxicity have included measurements of soil pH, exchangeable aluminium, per cent saturation of the exchange complex with aluminium, and extractable aluminium (Thomas and Hargrove 1984). The critical value for each of these factors varies from soil to soil (Adams 1984a), thus limiting their application as a 'universal' soil test.

Solution culture experiments have demonstrated that the phytotoxic components of soil solution aluminium are inorganic monomeric ions (Blamey et al. 1983; Alva et al. 1986). Therefore, the best hope for a 'universal' soil test for aluminium toxicity probably lies in some method which estimates the concentration of phytotoxic soluble aluminium.

Acknowledgments

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