NUTRIENT DISORDERS OF SWEET POTATO

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Foreword

Farmers in developing countries grow sweet potato for a variety of uses. In China sweet potato is grown in large quantities as an animal feed and an industrial crop. In the Pacific, the crop is a major staple food needed in greater quantities as the population of the region expands. Importantly, the intensification of cropping caused by population growth is depleting the native soil fertility and exacerbating the nutrient disorders that are the focus of this book.

The Australian Centre for International Agricultural Research (ACIAR) had no hesitation in supporting the research that led to this publication. The geographic focus on the Asia and the Pacific is important to ACIAR and partner scientists in Papua New Guinea, Tonga and Samoa were keen to participate. The scientists from the University of Queensland have a proven track record in research on the diagnosis of plant nutrient disorders. These are the successful ingredients of ACIAR projects.

This publication is based on a systematic approach to research involving controlled experiments and field surveys in the collaborating Pacific Island nations. The book is a credit to the research partners and the ACIAR Publications Unit that helped produce it. I congratulate them all. I hope that the publication is made available to field practitioners who can use it to diagnose nutrient disorders in farmers' fields and recommend appropriate remedies to correct the problems. Such widespread use of the publication will be ample reward for the authors and should greatly benefit farmers producing sweet potatoes.

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About the Authors

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Introduction

Sweet potato (*Ipomoea batatas* (L.) Lam.) is a hardy and nutritious staple food crop, which is grown throughout the humid tropical and subtropical regions of the world. It is a perennial plant of the family Convolvulaceae, with long trailing vines and leaves which vary in shape from simple to deeply lobed. The edible roots, often referred to as tubers, are formed by secondary thickening of the upper part of some feeder roots. Among the great diversity of cultivars grown, two types are commonly recognised. The staple types, grown throughout the tropics, are usually white, red or purple, although yellow-fleshed types are popular in Africa. The orange-fleshed types, preferred in the USA, typically have a higher sugar and lower dry matter content, and are usually eaten only as a supplementary or dessert vegetable.

Originally domesticated in tropical America, sweet potato spread to the South Pacific in prehistoric times. However, in the countries where sweet potato now has greatest importance, it is a relatively recent migrant. European traders and colonists brought the crop to Africa, Asia and the western Pacific. It reached China in 1594 (Kochhar 1981) and Papua New Guinea (PNG) between 300 and 400 years ago (Yen 1974). Rwanda, now one of the highest per capita consumers of sweet potato, began cultivating it only in the 18th century (IITA 1992).

World production of sweet potato is now of the order of 122 Mt, ranking third among the root crops after the 'Irish' potato (281 Mt) and cassava (164 Mt) (FAO 1996) (Table 1). China is by far the largest producer, with 84% of the global production, and Vietnam and Indonesia together account for a further 4%. However, on a per capita basis, sweet potato is most important in countries of highland east Africa and the Melanesian Pacific, where it constitutes a major staple in people's diet (Opio 1990).

The regional and national figures in Table 1 do not reflect the variation in usage within countries.

Table 1. Total production, production per capita and yield of sweet potato in 1993, and the percentage change from 1985 to 1995, by region (derived from FAO 1996).

Region or country	Total production, 1993 (Mt)	Production per capita (kg/year)	Yield (t/ha)	Change in production 1985–1995 (%)	Change in yield 1985–1995 (%)
World	122.0	21.4	13.2	+9.4	–11.1
Africa	7.48	10.3	4.9	+8.6	–20.0
— Uganda, Rwanda & Burundi	4.01	112.5	5.3	+1.2	–12.3
North America	0.584	2.0	17.1	–13.4	+7.9
Central America	0.485	3.0	3.7	–43.3	–24.6
South America	1.31	4.1	11.4	–10.7	+25.2
Asia	111.6	35.4	15.3	+10.4	–11.0
— China	102.2	83.7	16.5	+13.1	–16.3
— Other Asian Countries	9.38	4.9	8.5	–11.8	+3.7
Europe	0.056	0.1	12.1	–46.2	+8.1
Pacific	0.568	54.3	4.6	+2.3	–5.5
— PNG & Solomon Islands	0.513	109.6	4.7	–1.2	–2.4

In fact, sweet potato is the main staple food for many communities. For example, a 1986 survey in Rwanda found per capita sweet potato consumption in the north-central region to be over 300 kg/year, representing 40% of the food consumed (Woolfe 1992). Earlier studies in PNG villages recorded adult consumption of 1.2–1.7 kg/day (approx. 400–600 kg/year), contributing over 90% by weight of the food consumed (Sinnett 1975). Access to imported foods in recent times has reduced this percentage in many communities, but many PNG Highlanders still rely on sweet potato for over half of their dietary energy and over 30% of their protein intake (Harvey and Heywood 1983).

Sweet potato is typically produced for food, eaten by the grower's family or locally marketed in an unprocessed form. However, an increasing volume is being processed into industrial starch, alcohol, noodles and other products, especially in China. In some regions, sweet potato is also the mainstay of livestock production, utilising both the vines and undersized roots. In PNG, pigs raised on sweet potato have traditionally been the main item of trade and a measure of wealth, and may consume up to 60% of the crop harvested (Harvey and Heywood 1983). In the Cañete Valley in Peru, sweet potato supports a modern dairy industry (Woolfe 1992).

Sweet potato is grown in a wide range of environments, from the humid tropics to mild temperate zones, and from sea level to 2700 m altitude (Bourke 1985a). Like cassava, it can be grown in semi-arid conditions, but it has greater tolerance than cassava to the low temperatures encountered at high altitude, and to heavy soils. However, it is intolerant of waterlogging, and is usually grown on ridges or mounds. Under favourable conditions, the vines grow quickly to cover the soil, eliminating weeds and hence minimising the labour required after planting. Clarke (1973) identified a number of factors contributing to the rapid rise of sweet potato to dominance in the agriculture of PNG, supplanting the traditional taro and yam. Importantly, sweet potato has a wider tolerance of soil conditions, allowing extended rotations and cultivation of previously marginal areas, and it requires less labour, produces yield earlier, can be harvested over an extended period as required for consumption, and does not require cooking before feeding to animals. Increasing population pressure, and the use of land and labour for cash cropping, continue to promote the shift to sweet potato production. Many of these factors play a role in other countries which have adopted sweet potato as a subsistence crop.

Sweet potato's short growth period (average 140 days) and the high edible proportion of the harvested product contribute to a very high edible energy yield compared with other staple crops (Table 2). The data in Table 2 are based on average

Table 2. Edible energy yield, and approximate composition per MJ edible portion, of sweet potato and other major tropical staple crops.

	Edible energy yield MJ/ha/day[a]	Protein[b] (g)	Calcium[b] (mg)	Iron[b] (mg)	β-carotene equiv.[b,c] (mg)	Thiamine[a] (mg)	Riboflavin (mg)	Niacin[a] (mg)	Ascorbic acid[a,c] (mg)
Sweet potato	201	3.6	67	1.5	0–42[d]	0.22	0.08	1.5	62
Cassava	146	1.7	66	1.9	0–0.25	1.10	0.05	1.1	48
Potato	205	5.9	25	2.3	Trace	0.31	0.11	3.4	85
Banana	184	3.3	20	1.5	1.0–2.6	0.09	0.09	1.3	38
Rice	138	4.1	14	0.3	0	0.04	0.02	0.7	0
Wheat	142	7.5	21	1.1	0	0.21	0.06	1.4	0
Maize	155	5.7	13	1.9	0.3	0.23	0.09	1.3	0
Sorghum	100	7.6	11	4.7	0	0.33	0.08	2.3	0

Sources: [a] de Vries et al. (1967), derived from Platt (1965); [b] Woolfe (1992)
[c] Losses of 18–78% of ascorbic acid (vitamin C) and 20–25% of carotenoids are incurred during boiling.
[d] Higher carotenoid content is found in orange-fleshed cultivars.

yields in tropical countries, and do not take into account differences in growth conditions. For instance, sweet potato, cassava and sorghum are more likely to be grown as traditional cultivars on poor soils, without irrigation or fertilizer, than are rice, potato or wheat. These considerations further emphasise the high yield potential of sweet potato.

Root crops are often considered to be nutritionally inferior to cereals (de Vries et al. 1967). However, when compared on an equal energy basis (Table 2), sweet potato has an average protein concentration comparable with that of rice. Furthermore, the protein concentration in sweet potato is highly variable, and there is potential for significantly improving the average through both genetic and management factors. A survey of South Pacific crops found protein concentrations from 0.46–2.93%, equivalent to 1.0–6.1 g/MJ (Bradbury and Holloway 1988).

Sweet potato is also a good source of calcium, ascorbic acid (vitamin C) and β-carotene (provitamin A). The yellow- to orange-fleshed cultivars contain particularly high levels of carotenoids, and are equalled only by carrot as a source of provitamin A (Woolfe 1992). Vitamin A deficiency is a common and serious health problem in many countries with rice-based diets, being the main cause of blindness in children. Less acute deficiency decreases children's resistance to infectious diseases, contributing to infant mortality. In response to this problem, efforts are being made in some Asian countries to promote the use of yellow-fleshed cultivars of sweet potato. There is scope for breeding yellow-fleshed sweet potatoes with higher dry matter and lower sugar content, which are more acceptable to users of traditional staple varieties.

The young leafy shoots, which are eaten as a green vegetable in some countries, particularly in Asia, are high in protein (approximately 20% of dry weight), and are also a good source of β-carotene, thiamine (vitamin B_1), riboflavin (B_2), folic acid and ascorbic acid (Villareal et al. 1985; Woolfe 1992).

In many countries, sweet potato is viewed as a poor man's food, with lower per capita consumption in higher income brackets (Woolfe 1992). This view has probably contributed to the relatively small research effort which has been applied to the development of sweet potato, compared with other crops of similar importance. A large proportion of the research effort has been directed towards selection and breeding of improved cultivars. This attention is justified, given both the high potential for crop improvement and the great diversity of cultivars available to the breeder. In PNG alone, about 1200 accessions are maintained, and the total number of indigenous cultivars may be as high as 5000 (Bourke 1985a). However, relatively little attention has been given to improvements in crop management, particularly in crop nutrition, despite large potential gains in the short term.

The potential yield for sweet potato under ideal conditions is approximately 80–100 t/ha. While such yields are unrealistic in most practical conditions, in intensive, high-input systems such as are found in east Java, the best farmers consistently produce yields of 30–50 t/ha within four months of planting (E. van de Fliert, pers. comm. 1996). Even in subsistence gardens, yields recorded in PNG vary from less than 1 t/ha (Floyd et al. 1988) to as much as 50 t/ha (Conroy and Bridgland 1950, cited in Bourke 1982). Yet the global average yield is only about 13 t/ha (Table 1), and that in semi-subsistence economies appears to average only 4–6 t/ha. This yield gap represents an enormous potential for improving food production. This is especially true in Africa, the Pacific and Central America, where high pressure on land resources, through population growth and expansion of cash cropping, necessitates such an improvement. Yet it is disturbing to note that yields in these areas are either declining or almost static (Table 1).

Whilst many factors contribute to low yield under farming conditions, nutritional problems are worthy of attention, firstly because of their prevalence, and secondly because of the large gains that can be made by alleviating them. Nutrient deficiencies almost invariably limit yields of subsistence crops, and are common even in fertilized crops. Mineral toxicities, particularly those associated with acid soils, affect many areas and may cause complete crop failure, but the

problem is often not correctly diagnosed. The decline in yield following continuous cropping of the same land is often largely due to depletion of soil nutrients, as can be seen by the response of sweet potato crops in old gardens to nutrient additions, either in the form of organic mulches or inorganic fertilizers (e.g. Bourke 1985b; D'Souza and Bourke 1986a, b; Floyd et al. 1988). However, the traditional fallow practices relied upon to renew fertility are being compromised due to increasing pressure on land resources (Bourke 1977a; Halavatau et al. 1996). The challenge is to restore sustainability in those areas where intensification has reduced yield, and to improve yield where land resources are limited for the expansion of production.

Accurate diagnosis of nutritional problems is a vital and indispensable first step to finding solutions to them that are technically sound, affordable, and friendly to the environment. Correction of a nutritional disorder does not necessarily require the use of inorganic fertilizers. In some cases, solutions may be available using local resources and improved management practices, including the use of leguminous plants in the cropping cycle. Many traditional practices add or conserve soil nutrients (e.g. D'Souza and Bourke 1986b), and new techniques are continually being developed. However, expanding trade and income opportunities make the use of fertilizers a desirable option for an increasing number of producers. For maximum benefit to producers, amendments will need to be applied on the basis of an understanding of the crop's requirements, and a recognition of which factor or factors are most limiting to production at a particular site.

This book is intended to aid those involved in sweet potato production (producers, advisers and researchers) in the diagnosis of nutritional problems, and to provide guidance towards correcting them. We have attempted to provide a comprehensive coverage of the current knowledge on sweet potato nutrition, although it is appreciated that such detail may make the information difficult for some farmers to access. It is hoped that this book will provide a resource from which farm advisers can extract information appropriate for the environment, culture and resource availability of local sweet potato growers.

The structure and use of this book

This book provides detailed, illustrated descriptions of all nutritional disorders of sweet potato which are likely to occur under field conditions. With each description, some information is given on the occurrence and management of the disorder. Mention is also made of other disorders which may present similar symptoms, and of diagnostic tests which may be applied to confirm a tentative diagnosis based on visible symptoms. The section on managing sweet potato nutrition may help in interpreting symptoms and in deciding what further steps to take. A glossary of specialist terms has also been included (Appendix 1).

To help the reader to locate relevant information quickly, disorders have been grouped according to where on the plant the symptoms are most likely to be seen. At the back of the book (Appendix 2) is a key to nutritional disorders based on their visible symptoms, which serves as a quick reference to the information on each disorder. The reader may wish to consult the key first.

Managing Sweet Potato Nutrition

Plant nutrients are the chemical elements which are essential components of plant tissue. For healthy growth, plants require an adequate supply of each of these elements. The most abundant elements in plants, carbon (C), oxygen (O) and hydrogen (H), are obtained from the air and water. The others, referred to as the mineral nutrients, are supplied by the mineral and organic components of the soil. They are divided into two groups, according to their abundance in plants. The macronutrients, nitrogen (N), potassium (K), phosphorus (P), calcium (Ca), magnesium (Mg) and sulfur (S), comprise from 0.1 to 6 % of dry plant material. The micronutrients, including iron (Fe), chlorine (Cl), boron (B), manganese (Mn), zinc (Zn), copper (Cu) and molybdenum (Mo), have requirements in the order of 0.1–100 mg per kg dry weight.

A deficiency of any one nutrient results in a reduced growth rate and yield of the crop. The problem can only be corrected by increasing the supply of that nutrient. No amount of N or P fertilizer, for instance, can compensate for a deficiency of B. Therefore, it is important to diagnose a nutritional disorder correctly, in order to treat it efficiently.

It is also important to recognise that supplying a nutrient in excess of the crop's requirement is of no benefit to the crop. It is an unnecessary expense, and may cause environmental pollution when excess nutrients leach into streams and groundwater supplies.

Many mineral nutrients (including B, Cl, Mn and Cu) are toxic to plants if they are present at high concentrations. Some other elements, which are not essential nutrients for sweet potato, may also cause toxicity. These include aluminium (Al), a problem in acid soils, and sodium (Na), in saline soils.

Causes of nutrient disorders

The supply of a mineral nutrient to the plant is determined not only by its total abundance in the soil, but also by the proportion of the total which is in an available form at any time, and by the plant's ability to capture this available component.

Usually only a small proportion of the total nutrient is available for uptake. Availability of many essential nutrients is affected by soil pH. At high pH (alkaline soil), the solubility of P, and of many micronutrients (e.g. Fe, Mn, Zn and Cu) is greatly reduced, and the crop may experience deficiencies of these nutrients. At low pH (acid soil), the solubility of some elements, particularly P and Mo, is reduced, while that of Al and Mn may be increased to toxic levels. Disorders such as Al toxicity, which inhibit root development, reduce the plant's ability to capture nutrients and water, and may induce symptoms of secondary disorders such as Mg deficiency or water stress. It is important to recognise the primary agent in such cases.

Deficiencies of macronutrients, particularly N, P and K, are often associated with fertility decline following sequential cropping. These elements are taken up in such large quantities by the crop that the soil's reserves become depleted after a number of crops have been produced. Increasing cropping intensity results in an increasing dependence on external supplies of these nutrients. In low-intensity agriculture, supplies may be replenished during a bush fallow, by allowing time for weathering of mineral particles, by redistributing nutrients into the crop's root zone from below, and by capture of wind-borne nutrients from sea spray. Nutrients accumulated by plants during the fallow become available to the crop as the plant material decomposes, or when it is burnt. Burning makes many nutrients immediately available to the following crop, but it also makes those nutrients easily lost by leaching. Burning also decreases the soil's ability to continue supplying nutrients, as there is less organic material to decompose. Some

nutrients, particularly N and S, are lost into the atmosphere during burning.

Deficiencies of micronutrients are usually associated with low natural abundance in the soil, or unfavourable soil conditions causing insolubility of these nutrients. In the case of low abundance, correction usually requires the application of only a few kilograms per hectare of the deficient nutrient, which may be effective for a number of years. Such inputs are likely to be cost-effective, even when the application of N or P fertilizers is not. The management is more difficult when the deficiency is due to adverse soil conditions, such as the very high pH in coralline soils. Applying the nutrient as a foliar spray is one way to avoid it being fixed in such soils. Increasing the organic matter content of the soil is also beneficial.

Diagnosing nutrient disorders

Plants respond to a deficiency of any nutrient firstly by decreasing their growth rate. Specific symptoms which may allow the deficient nutrient to be identified, usually occur only at relatively severe levels of deficiency. Nevertheless, such symptoms are often the first thing to alert the grower that there is a problem, and they are very useful for diagnosis. Since plant species differ in sensitivity to particular nutrient stresses, observation of symptoms on other crops in the same area should indicate to producers and advisers that the problem may also be affecting sweet potato, even if symptoms are not evident on sweet potato crops. Chemical analysis of the plant tissue provides a second diagnostic tool, which, when available, can be very valuable for confirming a tentative diagnosis based on visible symptoms, or identifying a suspected problem when symptoms are not visible.

If a soil is low in several nutrients, the plant usually shows deficiency symptoms of the element which is most limiting to growth. If that nutrient is supplied, the growth rate will increase until it is limited by the next most scarce nutrient, and a new set of symptoms may develop. It is difficult to establish from plant symptoms or tissue composition which nutrients are likely to be deficient other than the one most limiting. Soil tests may provide some guidance, but they need to be calibrated for each crop and soil type before they can be interpreted with confidence.

One of the simplest and most reliable methods of discovering all the nutrients which are deficient in a soil is through small pot trials. In nutrient omission pot trials, each pot receives an adequate supply of all the mineral nutrients (control, or 'All' treatment), or all minus one. Plants are grown for a short period (usually 4–6 weeks), and the dry weight of tops is compared with that of the 'All' treatment. Any nutrient whose absence results in a significant decrease in weight is regarded as deficient. Usually an indicator plant such as maize is used, which offers rapid and uniform establishment from seed, and well characterised responses to nutrient deficiencies. However, nutrient levels which are deficient for one species may be adequate for another. In recent studies, sweet potato tip cuttings have been used successfully in nutrient pot trials (Dowling et al. 1995, 1996).

If a nutrient deficiency is suspected, the best way of confirming it is by observing a positive response to fertilizer containing that nutrient. When testing a fertilizer response, it is important to have an untreated area for comparison. Usually a narrow test strip in the middle of the field is treated with fertilizer. Alternatively, fertilizer may be applied to the whole field, with the exception of a central strip, which would remain unfertilized. Fertilizers often contain more than one nutrient, an it is important to establish which nutrient is causing the response. For example, N and S deficiencies both produce similar symptoms in the crop. They can be differentiated by applying sulfate of ammonia (containing both N and S) to one strip and urea (containing only N) to another. If the crop greens up in both strips, the problem is N deficiency, but if it only responds to the sulfate of ammonia, it is S deficiency.

Another useful technique for confirming deficiencies of micronutrients is leaf painting. By carefully painting half of an affected leaf with a dilute solution of the suspected nutrient, the response can be seen by comparing the colour or expansion of the painted half with the unpainted half. Leaf painting has been used successfully to

diagnose deficiencies of Fe, Mn and Zn in sweet potato.

Interpreting visible symptoms

Due to the different role played by each nutrient in the plant, each nutritional disorder tends to produce its own characteristic symptoms. Visible symptoms provide a useful diagnostic tool which is not dependent on costly laboratory equipment or time-consuming chemical analyses. However, some disorders produce rather similar symptoms or no symptoms at all, and the effects of insect pests and diseases may produce symptoms similar to those of nutritional disorders. Environmental conditions (e.g. moisture supply, temperature, light) may affect the appearance and severity of nutrient disorders. Cultivars may also differ in their expression of symptoms. Sweet potato, in particular, shows enormous diversity in the appearance of cultivars, and this is reflected in the range of symptoms they display. Nevertheless, there are distinct patterns, and a careful observer can usually reduce the number of possible causes to a few, if not to a single suspect. Tentative diagnoses can then be confirmed by applying fertilizer test strips, by soil tests, leaf painting or plant tissue analyses.

Visible symptoms often take the form of chlorosis, the reduction of green colour (chlorophyll pigment) in the leaves. Chlorotic tissue may be light green, yellow or whitish. Tissue furthermost from the veins is often the most severely affected, as it is last in the supply line. Hence, chlorosis patterns are frequently described as interveinal, if the tissue on and adjacent to the veins retains a darker colour than the remainder of the leaf. To what degree the minor veins retain their colour in addition to the major veins, and the distance over which the colour change is graduated, are additional features which aid diagnosis. If the chlorosis affects the whole leaf blade uniformly, it is referred to as 'general'. 'Vein clearing', the case when the veins become paler than the rest of the blade, is more often a symptom of viral infection than of a nutritional disorder.

Necrosis is the death of tissue. It may occur following chlorosis as part of a progressive degradation, or it may arise in localised zones of the leaf due to a critical disfunction. The location, shape and size of necrotic lesions are useful discriminators, as are the colour and texture of the dead tissue.

Other symptoms include changes in the occurrence and intensity of secondary pigmentation (red or purple colours), changes in the shape or dimensions of plant parts such as thickening, cupping or curling of the leaf blade, size reduction of the blade, deformities causing irregularly shaped or incomplete leaf blades, or shortening of internodes on the stem.

In addition to the appearance of a particular symptom, the position or location of that symptom on the plant must be noted. Nutrients are absorbed by the root system, and distributed among the various plant parts. Some of these nutrients may be redistributed to younger parts of the plant during times of shortage either readily (e.g. K, P), more slowly (e.g. S) or hardly at all (e.g. B, Ca). Thus, deficiencies of K and P are likely to be observed first on older leaves, that of S on both older and younger leaves, and those of B and Ca on the younger leaves. Elements taken up in excess of plant requirements continue to be accumulated during the life of a leaf. Thus, there will be a tendency for toxicity symptoms to appear first on the older leaves where accumulation has been occurring for the longest time.

In many locations, sweet potato flowers sparingly or not at all. An unusually prolific or early production of flowers is usually a sign that the crop is under nutritional stress. However, as a number of nutrient deficiencies can cause increased flowering, this symptom is of little use in diagnosing the particular deficiency.

Interpreting tissue analyses

Chemical analysis of plant tissue is also an important technique in the diagnosis of nutritional disorders. In annual crops, tissue analysis is most often used in 'trouble-shooting' rather than in the recommendation of fertilizer rates. However, if the tissue samples are collected early in crop growth and analyses are completed quickly, a corrective fertilizer application may be possible within the same season.

The interpretation of tissue analyses is based on established relationships between crop yield and nutrient concentrations in plant tissue (Fig. 1). Critical concentrations are those which separate the sufficient (healthy) range from the deficient or toxic range. For practical purposes, critical concentrations are defined as those concentrations associated with 90% of maximum yield (Ulrich and Hills 1973). Between these concentrations is the range of concentrations required for healthy growth.

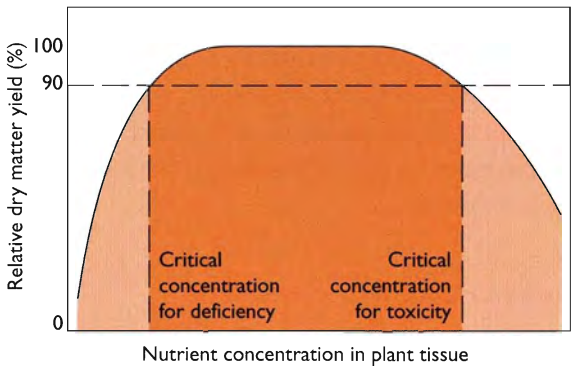


Figure 1. Schematic relationship between nutrient concentration in the plant tissue and dry matter yield relative to the maximum possible yield.

The relationship between crop yield and concentration of a particular nutrient in plant tissue may be determined by means of nutrient solution culture experiments, glasshouse pot experiments, or field experiments. Generally, field experiments are considered the best (Bates 1971), but are considerably more expensive than solution culture and pot experiments. They also depend on the availability of sites which are deficient in each of the nutrients to be studied. The values given in this publication have been derived from solution culture experiments (O'Sullivan et al. 1996a). Where possible, their reliability has been validated under field conditions.

A certain part of the plant rather than the whole plant, is usually collected for analysis. Leaves are usually considered the most satisfactory parts (Bates 1971). Because leaves continue to accumulate some nutrients with age, it is important that nutrient concentrations in leaves of the same physiological age are compared. In many annual crops, the blade of the youngest fully expanded leaf is selected as the 'index' tissue for analysis. However, sweet potato leaves may continue to expand throughout much of their life, so their physiological maturity cannot be judged easily on the basis of full expansion. The many studies referred to in this book have selected different leaves or parts of the vine for analysis, which makes the information difficult to compare. In our research, the blades of the 7th to 9th youngest leaves have been selected as the index tissue (Table 3). They were selected as being sufficiently responsive to disorders of both mobile and immobile nutrients, and having less variable nutrient concentrations than younger leaves (O'Sullivan et al. 1996b).

Apart from the physiological age of the leaf, as indicated by its position on the plant, tissue composition may vary with the age or stage of development of the crop. For instance, some researchers have found the critical concentration for N deficiency to decline with crop age. Caution should therefore be exercised in applying the critical concentrations established for young plants to tissue taken from older crops. With regard to the values given in Table 3, it should be noted that the sweet potato plants grew more rapidly under experimental conditions than could be expected in the field, and that these plants may be equivalent to crops from 6 to 10 weeks of age, depending on the temperature and water supply experienced by the crop. In any case, they represent crops prior to the stage of rapid growth of the storage roots. From the point of view of applying corrective measures to the crop on the basis of the diagnosis, it is preferable to sample the crop at the earliest stage possible. However, in many instances, symptoms of a disorder may not appear until advanced stages of crop development. In such cases, tissue analysis will still be valuable, but discrepancies arising from plant age should be borne in mind when interpreting the results.

Environmental conditions may further affect the concentrations of nutrients found in leaves. The

concept of critical nutrient concentration requires that the nutrient of interest is the only factor limiting growth when the plant material is sampled. It has been shown that water stress can change the nutrient concentrations in leaves, and that plants take some time to recover normal concentrations after restoration of adequate water supply (Reuter and Robinson 1986). For example, to establish the P status of *Stylosanthes humilis*, Fisher (1980) recommended that a period of several weeks without water stress should precede the sampling of tissue. This is not practical in many situations, but users should be mindful of this potential source of error.

To collect leaf samples, the blades are removed without the petiole, and should be dried as soon as possible after sampling, using gentle heat (e.g. 60–70°C for 48 hours) or microwaving. If samples must be stored for more than a few hours before drying, it is preferable to keep them cool (e.g. in an ice box) to minimise the weight loss due to respiration of the living tissue. It is important to sample leaves that have not been contaminated with soil. If the leaves are dusty, they may be gently rinsed and blotted dry, but extended immersion in water and rubbing should be avoided. Only distilled or deionised water should be used.

When sampling a crop, it is best to take a composite sample of leaves from several plants which are equally affected by the symptom of concern (Reuter and Robinson 1986). If the crop is not uniformly affected, several samples could be taken, each from small, uniform areas of the crop, from the most to the least affected. The symptoms observed, and the level of severity, should be recorded for each sample, and the samples clearly labelled.

Soil analysis

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

Table 3. Critical nutrient concentrations for deficiency and toxicity, and the adequate concentration ranges for sweet potato, measured in the 7th to 9th open leaf blades from the shoot tip, sampled at 28 days from planting. Data were obtained from experiments in solution culture using cv. Wanmun.

Nutrient	Unit	Critical concentration for deficiency	Adequate range	Critical concentration for toxicity
Nitrogen	%	4.0	4.2–5.0	
Phosphorus	%	0.22	0.26–0.45	
Potassium	%	2.6	2.8–6.0	
Calcium	%	0.76	0.90–1.2	
Magnesium	%	0.12[a]	0.15–0.35	
Sulfur	%	0.34	0.35–0.45	
Chlorine	%	–	–	0.9–1.5
Iron	mg/kg	33	45–80	
Boron	mg/kg	40	50–200	220–350
Manganese	mg/kg	19	26–500	1600[a]
Zinc	mg/kg	11[a]	30–60	70–85
Copper	mg/kg	4–5	5–14	15.5[a]
Molybdenum	mg/kg	0.2	0.5–7	

[a] These critical concentrations have been found, on occasion, to be inconsistent with field observations, or to vary with environmental conditions. Refer to the relevant sections for a full discussion.

The results of soil analyses are interpreted on the basis of previously-established relationships between crop yield and soil test. These relationships may be established by means of glasshouse pot experiments or field experiments. In either case, they are specific to both the soil type and the crop species (and to some extent, the cultivar) used in the experiments, and can be applied to other crops or soils only with a degree of caution and uncertainty.

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

Correcting nutrient disorders

Once a nutritional problem has been correctly diagnosed, it is usually possible to rectify it, if not for the present crop, then at least for future crops at the same site. Whether or not a corrective measure can improve yields in an existing crop will depend on the nature of the problem and on the age of the crop when the diagnosis is made.

Nutrient deficiencies are alleviated by increasing the supply of the deficient nutrient. Applying inorganic fertilizers is one way of doing this. Another may be to add organic material such as animal manure, if it contains an appropriate balance of the required nutrients. Other approaches aim to change the soil properties, in order to increase the availability of nutrients already present or to reduce the supply of elements causing a toxicity. The pH of acid soils may be increased by adding lime ($CaCO_3$) or dolomite ($CaCO_3.MgCO_3$). Gypsum ($CaSO_4.2H_2O$) may be used to correct problems of soil surface crusting and poor permeability in saline and sodic soils. In soils prone to waterlogging, improved drainage may be necessary to reduce denitrification, to reduce the production of toxic forms of Mn, or simply to ensure that the roots receive enough oxygen to function well. Sweet potato roots are very sensitive to low oxygen supply.

Increasing the organic matter content of the soil has a number of beneficial effects. The gradual decomposition of this material provides a steady supply of plant-available nutrients. The organic particles may also provide a suitable substrate on which soil nutrients can be held in an available form. Organic matter increases the soil's ability to resist acidification. It also increases water retention so that the soil takes longer to dry out, and gives the soil an open texture so that more air can get to the roots. Organic matter is increased by leaving crop or fallow residue on the field, without burning, or by bringing plant material from another site. If the need for field sanitation prevents leaving the crop residue, then options may be to compost it and return it later, or to use it to mulch another crop which is not at risk from the pest. Often sweet potato vines are removed and used to feed stock. Returning the animal manure to the field replaces some of the nutrients, but it will not maintain the organic matter content of the soil unless supplemented by plant material from fallow or green manure crops.

Plant species, and even cultivars within species, differ in their sensitivity to low nutrient levels in the soil. They may vary in their nutrient uptake or in tissue requirements for a particular nutrient. For some crop species, agricultural scientists have developed cultivars resistant to particular nutrient problems, such as sunflower cultivars tolerant of low B supply (Blamey et al. 1984), and barley cultivars resistant to B toxicity. In sweet potato, a great diversity of cultivars exist, and there is evidence of regional adaptations conferring tolerance to low N (Jones and Bouwkamp 1992), and possibly to low B (D'Souza and Bourke 1986a). Researchers have identified lines which are tolerant of Al (e.g. Munn and McCollum 1976; Sangalang and Bouwkamp 1988; Ritchey et al. 1991) or of salinity and B toxicity (Chávez et al. 1995). It is likely that variation exists in the requirements for other nutrients also, which may be used in the future for selection and breeding of cultivars to overcome particular nutritional problems.

The approach taken to crop nutrition will depend on the grower's context. Where fertilizers and water are readily available and relatively inexpensive,

growers may aim to maximise the crop's potential by eliminating any nutritional stress. Where fertilizers are unavailable or too expensive, the aim may be to optimise use of resources in the agroecosystem, in order to gain an adequate and sustainable reward for the grower's labour. At whatever level of operation, it is important to recognise the limitations of the resource base. A traditional cropping system may become unsustainable through intensification, whether by increasing the number of crop cycles between fallows, or shortening the fallow period. However, intensification is frequently paralleled by a shift from subsistence to cash cropping. At some point in this progression, the purchase of inputs, including fertilizers, may become profitable. Grower advisers should remain aware of the options, even if some are not currently cost-effective.

Nutrient requirements of sweet potato

Sweet potato is regarded as being tolerant of poor fertility, as it may produce adequate yields on soils too poor for many other crops. However, the yield obtained on poor soil will be only a fraction of the potential yield for the cultivar. Often large increases in yield can result from a modest increase in nutrient supply.

Most of the nutrients taken up by a sweet potato crop are removed from the site when the crop is harvested. Depending on the extent of nutrient reserves in the soil, this loss may be incurred only once or several times before yields of subsequent crops suffer from nutrient deficiency. The amount of nutrients removed by the crop depends on the yield, and on whether the vines are removed from the field as well as the roots. Table 4 gives approximate rates of nutrient removal for crops yielding 12 t/ha of storage roots (about the global average) and 50 t/ha (a high yield). In an intensive farming system, those nutrients for which the soil has limited reserves may be supplemented by fertilizers. In addition to crop removal, nutrient losses through leaching, soil erosion and fixation will affect the actual fertilizer requirement. In less intensive systems, the rate of cropping that is sustainable will depend on the time required for soil reserves to be mobilised, or organic material to decompose, to replenish the plant-available nutrient pools in the soil.

Table 4. Estimated removal of nutrients from the soil by sweet potato crops of 12 t/ha (average) and 50 t/ha (high), for situations where only storage roots are harvested, and where both roots and vines are removed.

Nutrient	Nutrient removal[a] (kg/ha) by crop with root yield of:			
	12 t/ha		50 t/ha	
	Roots	Roots and Vines[b]	Roots	Roots and Vines[b]
Nitrogen	26	52	110	215
Phosphorus	6	9	25	38
Potassium	60	90	250	376
Calcium	3.6	16	15	65
Magnesium	3	6.5	12.5	27
Sulfur	1.8	4.3	7.5	18
Chlorine	10	18	43	75
Iron	0.060	0.160	0.250	0.670
Boron	0.024	0.074	0.100	0.310
Manganese	0.024	0.175	0.100	0.730
Zinc	0.036	0.062	0.150	0.260
Copper	0.018	0.037	0.075	0.155
Molybdenum	0.004	0.006	0.015	0.023

[a]Concentrations of nutrients in sweet potato roots and tops vary considerably. Quantities of nutrients removed have been based on representative concentrations from a number of sources (including Scott and Bouwkamp 1974; Bradbury and Holloway 1988; Woolfe 1992; Spence and Ahmad 1976; Diem 1962; Hill 1989; and the authors' own data), converted to fresh weight basis assuming 70% moisture in the storage roots and 86% moisture in the vines.

[b]A vine:root weight ratio of 0.6 was assumed. Actual ratios may vary in the approximate range 0.3–1.4.

Phosphorus deficiency

The phosphorus (P) availability to plants may be limited by its low abundance in the soil, but also, and very commonly, by its adsorption onto various soil minerals. In acidic soils, P may be adsorbed by Fe or Al oxides, and various clay minerals. Many of the most fertile and productive soils in tropical zones are derived from volcanic material containing allophane minerals, which have a large P-fixing capacity. Phosphorus deficiency is often the major limitation to crop growth on these soils, particularly where previous cropping has caused a depletion of soil organic matter and increased acidification. Phosphorus deficiency is also common on highly weathered tropical soils and siliceous sands; in fact, few soils are naturally well endowed with this nutrient.

In calcareous soils, P may be adsorbed by calcium carbonate, or precipitated as calcium phosphate (Tisdale et al. 1993). The ability of sweet potato to take up P may also be reduced by high pH. Sweet potato was found to grow poorly in solution culture maintained at pH 8.0 (Ila'ava 1997). This was largely attributed to poor P uptake despite continuous monitoring and maintenance of P in solution.

Sweet potato has been reported to be relatively tolerant of low soil P status (de Geus 1967). A number of fertilizer trials in the USA have shown little or no yield responses of sweet potato to the application of P fertilizers (Bouwkamp 1985; Jones and Bouwkamp 1992). In these trials, it appears that residual P from previous crops was sufficient to supply the needs of the sweet potato crop.

Sweet potato's efficiency in obtaining soil P is due in part to its association with vesicular-arbuscular mycorrhizae (VAM). These ubiquitous soil fungi invade the plant roots and feed on its sugars, and in return they assist with the capture of P from soils with low P availability. Mycorrhizal infection has been shown to increase sweet potato growth and yield in a number of studies (e.g. Paterson et al. 1987; Kandasamy et al. 1988; Mulongoy et al. 1988; Khasa et al. 1992; Paula et al. 1992, 1993; Dowling et al. 1994). Floyd et al. (1988) found that the extent of mycorrhizal infection was positively correlated with yield, and negatively correlated with the crop response to P fertilizer, over a range of soils in the Highlands of PNG. The effect is greatest under low P fertility (Negeve and Roncadori 1985), and VAM may have no benefit or even a negative effect on crops which are well supplied with P.

Although sweet potato may yield relatively well under low P conditions, P deficiency is still a very common cause of reduced yields. Goodbody and Humphreys (1986) found significant positive correlations between sweet potato yield and available P in each of three soil types surveyed in Simbu province of PNG. Also in Highland PNG, Floyd et al. (1988) obtained a three-fold increase in yield in response to P fertilizers, when K was also supplied. Recent field trials have demonstrated a large positive response of sweet potato to P fertilizer on a number of volcanic ash soils in Tonga (Halavatau et al. 1996) and andisols in PNG (Dowling et al. 1994). Crop surveys in Uganda showed P deficiency to be the most severe problem of sweet potato on heavily cropped and acidified soils in the Kabale region (E. Carey and J. Low, pers. comm. 1996).

It is clear from many such examples that wider use of P fertilizers will play an important role in improving sweet potato production, as it has for other crops. However, it is also evident that rates of P fertilizers recommended for other crops may be excessive and wasteful when used on sweet potato.

Symptoms of phosphorus deficiency

Mild to moderate P deficiency may be difficult to recognise in the field. Growth may be reduced to less than one half that of well nourished plants, without the appearance of any identifiable symptoms of P deficiency (Plate 1a). Mild P deficiency is often associated with a darker than normal, bluish green colour of the foliage. Unlike N deficiency, young to mature leaves remain dark green at all levels of severity. Obvious symptoms on the older leaves are associated with severe stunting (Plate 1b).

The first sign of P deficiency is usually the premature senescence of older leaves. In most (but not all) cultivars, yellowing is preceded by the appearance of purple anthocyanin pigments, producing a range of autumnal colours in the senescing leaves (Plate 1c). Yellowing may spread from discrete interveinal patches, which typically become cleared of anthocyanin pigment (Plate 1d), or may be more general, but often affecting one half of the blade more than the other (Plate 1e). In this case, the chlorotic areas may appear orange or red due to the overlying anthocyanin pigments. Necrotic lesions subsequently develop in the chlorotic zones (Plate 1f), and the necroses spread as irregular patches until the leaf blade is entirely brown and dry. In some cultivars (e.g. Markham, Plate 1g) no yellow or purple phase precedes the necrotic lesions, which appear on green tissue. However, as in other cultivars, those parts of the leaf blade which are not yet necrotic turn yellow in the final stages of senescence (Plate 1h).

Some cultivars may develop purple pigmentation on the upper surface of the youngest leaves, particularly on the veins (Plate 1i). This may resemble N deficiency, although in P deficiency it is less common among cultivars, and is less strongly veinal.

Possible confusion with other symptoms

The appearance of red pigmentation on the veins of young leaves may resemble N deficiency. However, in the case of P deficiency, there is no general chlorosis of the plant. Potassium and Mg deficiencies also cause chlorosis on older leaves, but in P deficiency chlorosis usually does not retain a distinct interveinal pattern, as is more typical of K or Mg deficiencies.

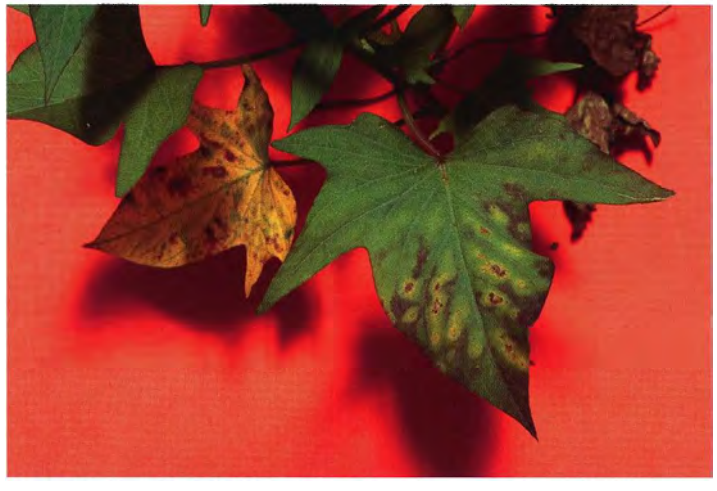
Sweet potato feathery mottle virus (SPFMV) may also induce chlorotic spots surrounded by purple tissue on older leaves. Lesions caused by the virus are randomly scattered over the leaf blade, and are not restricted to the oldest leaves on the vine. The symptoms do not progress to cause necrotic lesions or orange and red colours on the senescing leaves, as seen in P deficiency.

Plate 1. Phosphorus deficiency

a) Healthy (left) and P-deficient (right) plants of cv. Beerwah Gold, grown in solution culture. The P-deficient plant received 6% of the P supplied to the healthy plant and shows severe growth reduction without obvious symptoms of disorder.

b) A severely stunted, six-weeks-old plant of cv. Hawaii, grown in a P-deficient soil in Tonga. Typical symptoms of P deficiency are evident, including purpling and subsequent yellowing of older leaves, with dark green colour remaining in young leaves.

c) Bright autumnal colours in senescing older leaves of a P-deficient crop of cv. Hawaii in Tonga. Note also the numerous flowers, which are often a symptom of stress.



d) Interveinal chlorotic zones developing to necrosis on an older leaf of cv. Hawaii grown in solution culture with low P supply.

Plate 1. Cont'd

e) Generalised chlorosis on an older leaf of cv. Beerwah Gold, affecting one side of the leaf blade more than the other.

f) Necrotic lesions spreading within the chlorotic tissue on an older leaf of cv. Wanmun.

g) Necrotic lesions without chlorosis or purple pigmentation, developing as a result of P deficiency on an older leaf of cv. Markham.

h) Yellowing and extensive necrosis on a senescing leaf due to P deficiency in cv. Markham.

i) Purple pigmentation on the young leaves of a P-deficient plant of cv. Markham. Healthy plants of this cultivar have no purple pigmentation.

Diagnostic soil and plant tissue tests

In solution culture experiments, a critical concentration of 0.22% P has been estimated in the blades of the 7th to 9th youngest leaves. Concentrations between 0.26 and 0.45% P were associated with maximal growth (Table 3). These concentrations concur with those of Leonard et al. (1949) and Weir and Cresswell (1993).

Little work has yet been done to calibrate soil tests for predicting yield response of sweet potato to applied P. Bingham (1962) suggested that available P levels in the range 5–7 mg/kg (Olsen's method) correspond to the threshold for deficiency in crops with a low P requirement, such as sweet potato. Goodbody and Humphreys (1986) obtained positive regressions between available P and sweet potato yield, on soils ranging from 0.6 to 5 mg/kg available P (Olsen's method). Fox et al. (1974) gave 0.001 ppm (0.3μM) as the minimum soil solution concentration of P associated with 95% of maximum yield of sweet potato, and Nishimoto et al. (1977) found that a soil solution concentration of 0.003 ppm (0.1μM) corresponded to a yield 70% of the optimum. It should be noted that these measurements include only part of the P in soil organic matter, which may represent a considerable proportion of the P available to the crop in some soils.

Measurements of phosphate binding capacity, in addition to plant-available P, have been used to estimate the quantity of P fertilizer required on

phosphate-fixing soils. Phosphate binding can be estimated using phosphate sorption isotherms (Fox and Kamprath 1970), or phosphate retention (Saunders 1974). These methods have been described by Rayment and Higginson (1992).

Correction of phosphorus deficiency

Phosphorus deficiency can be corrected by broadcast, band or spot application of soluble P sources, such as single or triple superphosphate, ammonium phosphate, or mixed fertilizers (containing N, P and K, with or without other nutrients). Band or spot application of P fertilizers is recommended on strongly P-fixing soils.

Rock phosphate is a relatively cheap alternative to more soluble fertilizers. Rock phosphate should be well incorporated into the soil, and is usually only effective on acidic soils, due to its very low solubility at neutral to high pH. On an acid lateritic soil, Kabeerathumma et al. (1986) found rock phosphate to be equally effective as superphosphate on the first sweet potato crop, and to have a greater residual effect.

Single superphosphate (10% P) also contains S and Ca, and is recommended in soils where these nutrients are also low. Triple superphosphate (TSP, 24% P) also contains Ca, but not S.

A sweet potato crop will remove about 8–40 kg P/ha from the soil depending on yield (Table 4), but on P-fixing soils, much higher rates of application (>100 kg P/ha) may be needed in the first year of application. In subsequent years, lower rates may suffice to maintain an adequate supply of P to the crop.

Traditional subsistence methods of cultivating sweet potato, including the incorporation of substantial amounts of organic matter and using large, healthy runners for planting, add significant amounts of P, as well as other nutrients. Decomposition of organic matter provides a steady supply of plant-available P even on highly P-fixing soils. An important part of the response of sweet potato crops to organic matter additions is due to improved P nutrition (Floyd et al. 1988).

Potassium deficiency

Root crops have a high requirement for potassium (K) compared with cereals, as the content of K in the harvested roots is high (see Table 4). A 20 t/ha sweet potato crop removes approximately 100 kg K/ha in the storage roots, and considerably more is removed if both roots and vines are harvested (Table 4). Even soils which are naturally high in K may become depleted after several successive crops. On various soils in PNG which had been continuously cropped with sweet potato for several years, large yield increases were obtained by the application of either inorganic K fertilizer or organic composts high in K (Bourke 1985b; D'Souza and Bourke 1986a, b; Floyd et al. 1988).

In contrast to N or P deficiencies, K deficiency tends to have a much greater effect on storage root yield than on the growth of the tops (Edmond and Sefick 1938; Bourke 1985b). Thus, correction of an apparently mild deficiency may result in large yield increases.

Symptoms of potassium deficiency

As with other major nutrients, K deficiency can cause substantial growth reduction before specific symptoms develop (Plate 2a). In the field, symptoms often develop after two to three months, when the expanding storage roots begin to place increased demand on K supplies. Yellowing appears on the oldest leaves, while the youngest leaves retain a normal colour, size and texture (Plate 2b). If young leaves are normally purple, this pigmentation may be reduced in K deficient plants (Plate 2a).

The oldest leaves develop a yellow chlorosis in marginal and interveinal zones (Plate 2c). Brown necrotic lesions develop within the chlorotic zones (Plate 2d) and eventually spread to cover the entire leaf blade. Cultivars vary in the extent to which lesions spread predominantly from the margins to interveinal zones, are initiated in interveinal regions nearer the midrib, or spread with relatively little regard for veinal distribution. Necrosis associated with K deficiency is usually dark in colour, and the necrotic areas become dry and brittle.

The yellowing and necrosis may be preceded by a light green interveinal mottle affecting mature to older leaves, and often most obvious on the leaves of axillary shoots. This may be the earliest, or only sign of the disorder, but depending on cultivar and conditions, it may not develop. Initially, leaves of intermediate age may be more affected than the older leaves (Plate 2e). Minor veins retain their green colour, finely dividing the chlorotic tissue. In some cases, the interveinal tissue may become necrotic, either remaining as isolated pits (Plate 2f) or coalescing to form small, irregular lesions (Plate 2g).

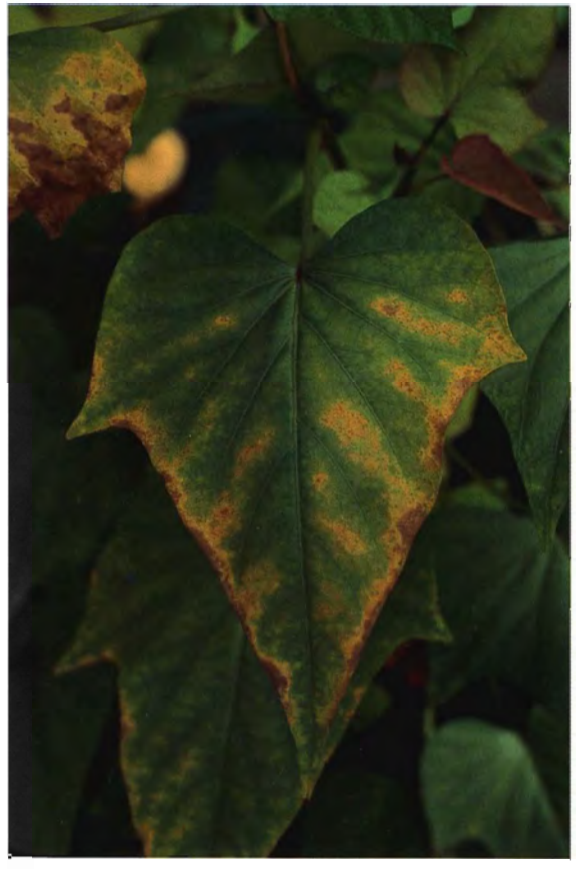
Potassium deficient crops tend to produce small, thin storage roots of poor quality (Plate 2h). Orange-fleshed varieties usually have a paler-than-normal flesh colour.

Plate 2. Potassium deficiency

a) Growth response of sweet potato cv. Wanmun to K supply in a solution culture experiment. Plants from left to right received 100%, 14% and 1.7% of the optimum K supply. Note that yellowing of the oldest leaves is apparent only on the more severely deficient plant (right).

Plate 2. Cont'd

b) A K-deficient crop of cv. Foketi grown in Tonga on an old volcanic ash soil low in K.



c) Yellow chlorosis spreading from margins and interveinal patches of an older leaf of cv. Wanmun, and early the development of necrotic lesions.

d) The spread of chlorosis and necrosis on senescing older leaves of cv. Wanmun.

e) Light green interveinal chlorosis on a leaf of intermediate age, cv. Wanmun.

f) Small necrotic lesions following interveinal chlorosis on a mature leaf of cv. Markham.

Plate 2. Cont'd

g) Irregular necrotic patches developing from the joining of small interveinal lesions on mature leaves of cv. Beerwah Gold.

h) Slender, under-sized storage roots from a K-deficient crop of mixed cultivars, on the Nembi Plateau, PNG. The garden had been cropped with sweet potato for a number of years. (Photo: R.M. Bourke)

Possible confusion with other symptoms

The appearance of an interveinal chlorotic mottle or small necrotic pits in interveinal tissue of leaves of intermediate age may resemble symptoms of Mn deficiency. However, in the case of Mn deficiency, the green zones around veins are broader and more diffuse. The absence of symptoms on the youngest leaves, and the occurrence of necrotic lesions on the oldest leaves indicate K deficiency.

Interveinal patches of necrosis surrounded by chlorotic zones are also symptomatic of Mg deficiency. Mg-deficient crops are generally pale in colour, and the pattern of interveinal chlorosis is usually more regular, with the major veins remaining green for their entire length.

Diagnostic soil and plant tissue tests

The determination of a critical concentration for K in sweet potato leaves is complicated by the fact that sweet potato has some capacity to substitute Na for part of its K requirement. In the presence of sufficient Na, a critical concentration of 2.6% K has been determined in the 7th to 9th youngest leaf blades (Table 3). In solution culture experiments with very low Na concentrations, the critical concentration for K was found to be approximately 4.0% (O'Sullivan et al. 1997a; Ivahupa 1997). The maximum substitution effect seems to be achieved at relatively low Na concentrations

(Ivahupa 1997), which would be found in most soils. Therefore, a critical concentration of 2.6% would apply in most field situations. This appears to be consistent with field observations of the response of crops to K fertilizer.

Soil exchangeable K measurements are frequently used to predict the K status of crops, but they do not reflect the soil's reserves of potentially available K, which may be released over a period of time. Crop responses to K fertilization are generally expected at exchangeable K values in the range 0.2–0.6 cmol(+)/kg soil, although this relationship depends on the soil texture and total cation exchange capacity (CEC): sandy soils may respond only in the range 0.05–0.25 cmol(+)/kg (Landon 1991). In the Highlands of PNG, Goodbody and Humphreys (1986) found a positive correlation between sweet potato yield and exchangeable K over a range of 0.2–1.0 cmol(+)/kg soil.

Correction of potassium deficiency

Since sweet potato crops remove a considerable amount of K from the soil (approximately 8 kg K in storage roots plus vines per 1 t of roots), continuous cropping without fertilization can lead to the exhaustion of soil K reserves. Potassium deficiency occurs most commonly on sandy soils, which have a low CEC, and on oxisols and ultisols with low base status. Some volcanic ash soils have large K reserves, while others may have a low CEC, and therefore low available K and a poor ability to retain added K.

Potassium can be added to the soil either in the form of inorganic fertilizer or in organic mulches and composts. Recommendations for fertilizer additions range from 80 to 200 kg K/ha (de Geus 1967). Organic mulches are much more bulky than inorganic fertilizers, therefore requiring more labour, but the efficiency of nutrient use by the crop may be higher (Floyd et al. 1988). D'Souza and Bourke (1986b) recommended an application of coffee pulp compost at 20 t/ha in the Nembi Plateau of PNG.

Split applications of K, before planting and after 4 weeks of growth, are often used (Hill 1989), with the fertilizer banded along the row. However, Fujise and Tsuno (1967) recommended ploughing in K fertilizer to a depth of 40 cm, noting that it is the deeper roots which most actively provide K for tuber growth in the late stages of crop development. Such deep placement is rarely practised, other than by the burial of compost under mounds in some traditional production systems.

Excessive application of K may lead to Mg or Ca deficiency, due to depression of the uptake of these elements (Spear et al. 1978). On sandy soils in particular, Mg and Ca applications may be necessary in addition to K to maintain a favourable balance (Landon 1991).

Magnesium deficiency

Magnesium (Mg) deficiency may result either from low Mg content in the soil or from an overabundance of K or Ca which inhibit Mg uptake by the crop (Leonard et al. 1948). Therefore, the disorder is most likely to occur on sandy soils with low CEC, on volcanic ash soils of high K status, or on some calcareous soils. Overfertilization with K may also induce Mg deficiency. In strongly acidic soils, Mg deficiency may be induced by the presence of toxic concentrations of Al in the root environment, which inhibit Mg uptake by the plant (see Aluminium toxicity).

Symptoms of magnesium deficiency

A Mg-deficient crop will tend to have a pale overall colour. The earliest specific symptom of Mg deficiency is an interveinal chlorosis of older leaves (Plate 3a). Typically, the main veins retain a relatively broad margin of dark green tissue, but the minor veins are less well defined, resulting in radial bands of pale tissue between the main veins (Plates 3b & c). However, in some cultivars the chlorosis is more mottled, composed of isolated patches, or the veins retain little green margin, and appear as a green network on a generally pale leaf (Plate 3d). Chlorosis first appears on the oldest leaves, but may spread to quite young leaves (Plate 3e). It may be accompanied by upward or downward curling of the leaf margins (Plates 3d & a), or a wilty drooping of the leaf blades (Plate 3d).

Purple or red-brown pigment may appear on older leaves in conjunction with chlorosis. Most commonly, pigmentation affects the upper surface of interveinal patches near the leaf tip and margins (Plate 3f), but in some cultivars the veins under the leaf may become red (Plate 3g).

Chlorosis generally progresses to yellowing and necrosis of the oldest leaves. Leaves may become entirely yellow and wilted before browning off (Plate 3h), or necrosis may develop on interveinal and marginal tissue without prior necrosis (Plate 3i). Most commonly, localised yellowing precedes necrotic lesions, which spread from interveinal zones (Plate 3j).

The vines of Mg-deficient plants may become thin and twining, with lengthened internodes, a response similar to etiolation (Plate 3k). Lengthening of vines in response to Mg deficiency was also reported by Edmond and Sefick (1938).

Possible confusion with other symptoms

Potassium deficiency may also produce interveinal necrotic lesions surrounded by chlorosis in oldest leaves. However, in the case of Mg deficiency, necrosis is usually in a more regular pattern around the leaf, confined to interveinal and marginal zones. In addition, the senescing leaves are generally wilted, and the necrotic tissue is usually paler and remains soft.

Plate 3. Magnesium deficiency

a) Healthy (left) and Mg-deficient (right) plants of cv. Beerwah Gold, grown in solution culture, showing growth reduction accompanied by chlorosis, downward curling and some red pigmentation of the older leaves.

b) Symptoms of Mg deficiency in cv. Kisozi in the Kabale district of Uganda. Interveinal chlorosis begins on recently mature leaves and increases in intensity with leaf age. Some brown pigmentation is also present on the older, chlorotic leaves. (Photo: J. Low)

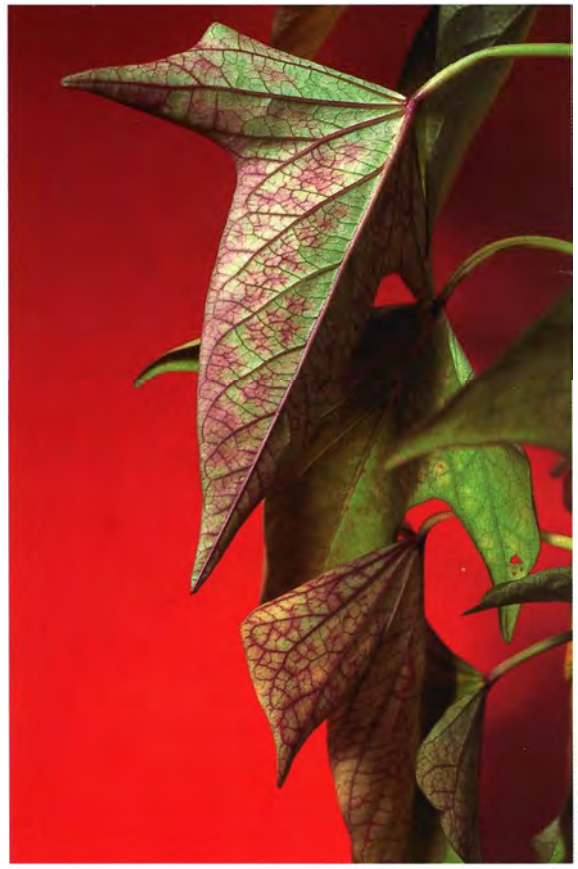
c) Interveinal chlorosis caused by Mg deficiency in cv. Hawaii, showing a broad margin of dark green tissue flanking the midvein and main veins, but little definition of the minor veins.

Plate 3. Cont'd

d) Symptoms of Mg deficiency in cv. Wanmun, showing little retention of green colour in the tissue adjacent to veins, giving a more mottled pattern of chlorosis. Leaf margins tend to curl upwards, and the blades hang limply.

e) Interveinal chlorosis on young (but not the youngest) leaves of Mg-deficient sweet potato from Ubili, New Britain, PNG.

f) A Mg-deficient crop of cv. Mushemeza in the Kabale district of Uganda, showing red-brown pigmentation of the upper surface of interveinal tissue. Pigmentation is initially near the leaf margin, but may spread over all interveinal zones. (Photo: J. Low)



g) Red pigmentation of the veins on the lower surface of Mg-deficient leaves of cv. Wanmun.

Plate 3. Cont'd

h) Chlorosis in cv. Wanmun, progressing to general yellowing and wilting before necrosis spreads on the senescing leaf blade.

i) Necrotic lesions on interveinal and marginal tissue of Mg-deficient leaves of cv. Hawaii, not preceded by general yellowing.

j) Localised yellowing and necrosis beginning in interveinal zones, and spreading during the senescence of an old leaf of cv. Lole.

k) A Mg-deficient vine tip of cv. Hawaii, with a thin, twining stem and lengthened internodes.

Diagnostic soil and plant tissue tests

The critical concentration for Mg in sweet potato leaves is not yet well defined, as various sources of information are not in good agreement. In particular, estimates based on solution- or sand-culture experiments tend to be lower than those observed in the field. Differences between the experimental results are probably mostly attributable to the selection of different tissues for analysis. In solution culture experiments, a critical concentration (leaf blades 7–9 at 4 weeks) of 0.12% Mg was estimated for Mg deficiency, while healthy plants were observed to contain 0.15–0.35% Mg (Table 3). Bolle-Jones and Ismunadji (1963) induced Mg deficiency in sand culture, and reported a concentration of 0.06% Mg in Mg-deficient plants (all leaf blades at 8 weeks), and 0.14–0.22% Mg in the leaves of non-deficient plants. Also using sand culture, Spence and Ahmad (1967) reported a concentration of 0.16% Mg (whole shoot to 9th leaf at 10 weeks) in plants showing early symptoms of Mg deficiency, while plants receiving adequate Mg supply contained from 0.37% to 0.94% Mg. However, symptoms observed in field crops in Uganda and Australia have been associated with concentrations in the index leaves of 0.29–0.37% Mg (authors' data), and in USA 0.40% Mg has been reported in an Mg-deficient crop (from Reuter and Robinson 1986).

Soil tests have not been calibrated for sweet potato response to Mg. For other field crops, neutral 1 M ammonium acetate-exchangeable Mg levels of greater than 80–160 cmol(+)/kg are generally considered adequate. European standards recommend consideration of soil test K levels also, with a K:Mg ratio of less than 5, on a weight-of-element basis, being desirable (Doll and Lucas 1973).

Correction of magnesium deficiency

Magnesium deficiency may be corrected by incorporation of dolomitic lime or magnesium oxide into acid soils (20–50 kg Mg/ha), or by band application of kieserite or fertilizer-grade magnesium sulfate (10–40 kg Mg/ha). As magnesium sulfate is the most soluble of these sources, it is the preferred source where it is necessary to correct an observed Mg deficiency in an established crop, although it is often more expensive than other sources. If top-dressing of the established crop is difficult, magnesium sulfate may be applied as a foliar spray or by fertigation.

Boron toxicity

Boron (B) toxicity is most likely to arise on saline and alkaline soils, following over-fertilization with B fertilizer, or following the application of irrigation water high in B. Chávez et al. (1995) reported that a combination of salinity and B toxicity was the main cause of low yields of irrigated sweet potato in southern coastal Peru.

Sweet potato appears to be only moderately tolerant of B toxicity, but this assessment was made according to the appearance of symptoms on sand-cultured plants, and may not reflect yield reduction (Landon 1991).

Symptoms of boron toxicity

Boron toxicity causes conspicuous necrotic lesions in the interveinal areas of older leaves (Plate 4a), leading to premature senescence and shedding of the leaves. Affected leaves are usually cupped downwards, or curled under at the tip. In solution culture, symptoms may be evident on plants suffering only a minor reduction in growth due to B toxicity, but in the field root damage may result in severe stunting and poor survival of cuttings.

In some cultivars, initial symptoms may be the development of a pale green to whitish interveinal chlorosis (Plate 4b), but in other cultivars necrotic spots may be preceded by only a localised chlorosis, with the surrounding tissue remaining green (Plate 4c). Necrotic spots may be light brown (Plate 4b), dark brown (Plates 4c & d), or have a dark outline (Plate 4e). The necrotic tissue is brittle, and in some instances may drop out, leaving 'shot-holes'.

Lesions are usually concentrated near the leaf margins and in interveinal tissue midway between main veins, but may appear initially to be more scattered (Plate 4c). They may spread and coalesce to occupy most of the interveinal and marginal tissue (Plates 4d & e). However, in cultivars with deeply divided leaf blades, the distribution may be less regular (Plate 4f).

Leaf senescence proceeds rapidly, with the remaining live tissue turning yellow, and necrosis spreading from the initial lesions over the entire blade (Plates4g & e).

Chávez et al. (1995) found considerable variation in tolerance to B toxicity among cultivars of sweet potato, but noted that the severity of foliage damage was not related to the root yield of the cultivar.

Possible confusion with other symptoms

Salinity may induce symptoms of necrotic lesions in interveinal tissue, similar to those caused by B toxicity. However, the necrotic lesions in the case of salinity are usually a dark, steel-grey colour and first appear on the margins of older leaves, although as symptoms spread to younger leaves, they become less marginal and more interveinal. Plants suffering from B toxicity may develop interveinal chlorosis well in advance of necrotic lesions. With salinity, chlorosis is uncommon except immediately surrounding the necrotic lesions.

In the early stages of symptom development in some cultivars, B toxicity may induce interveinal chlorosis similar to that of Mg deficiency. The appearance on older leaves of discrete necrotic spots would distinguish B toxicity from Mg deficiency.

Necrotic lesions, if produced with little preceding chlorosis, may be mistaken for those caused by fungal pathogens, such as *Alternaria* spp., *Cercospora bataticola* and *Phyllosticta batatas*. Fungal lesions may be recognised by their random distribution on the leaf blade, not showing a regular interveinal pattern. While fungal lesions may be more common on older leaves, they do not show a consistent decrease in severity from the oldest to younger leaves.

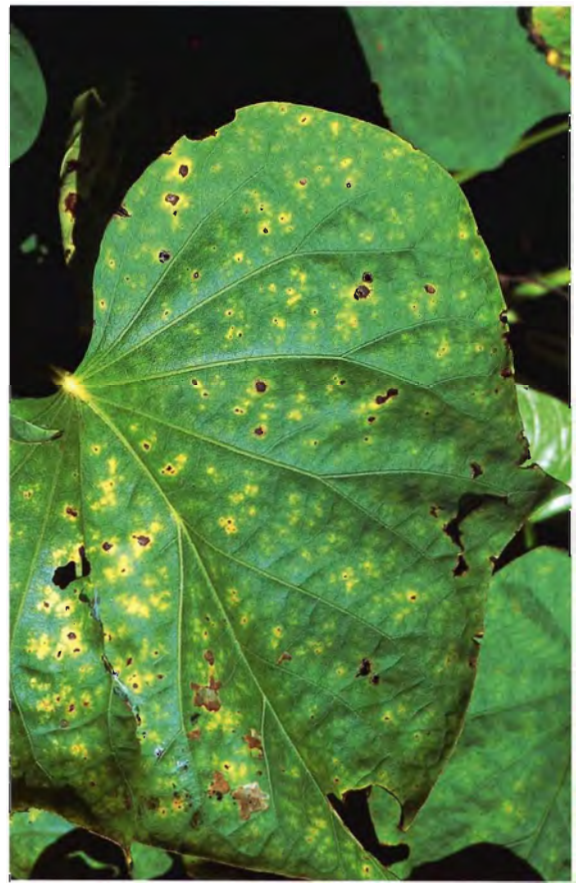
Diagnostic soil and plant tissue tests

The critical concentration for B toxicity in the 7th to 9th youngest leaf blades of sweet potato has been found to lie between 220 and 350 mg B/kg (Table 3). Concentrations of 400–800 mg B/kg were associated with 30–50% growth reductions, while concentrations from 1200 to more than 2000 mg B/kg were found in severely affected plants. Index leaves from healthy plants usually contain 50–200 mg B/kg.

Plate 4. Boron toxicity

a) Symptoms of B toxicity in cv. Beerwah Gold, showing interveinal chlorosis, necrotic lesions and downward cupping of older leaves.

b) Pale interveinal chlorosis caused by B toxicity in cv. Hawaii. Note the necrotic lesions towards the leaf tip.



c) Early appearance of necrotic spots in cv. Centennial, as a result of overfertilization with B. Spots have a scattered distribution and little preceding chlorosis.

d) Necrotic lesions in interveinal and marginal zones on cv. Beerwah Gold.

e) The spread of necrotic spots in interveinal and marginal tissue of an older leaf of cv. Wanmun, and general yellowing prior to death of the leaf. Previously shed leaves are visible below.

f) Symptoms of B toxicity in an older leaf of cv. Lole, showing an uneven distribution of lesions.

Plate 4. Cont'd

g) Leaf senescence in cv. Wanmun. Yellowing is followed rapidly by necrosis of the entire leaf blade, and leaf shedding.

Hot water extraction is most frequently used to estimate the concentration of plant-available B in soil. The threshold for B toxicity in sweet potato is in the order of 4 mg/kg hot water-extractable B, or approximately 0.15 mg/kg B in the soil saturation extract (Landon 1991). However, cultivars may vary considerably in their tolerance of excess B.

Correction of boron toxicity

Should B toxicity be encountered in the field, it may be possible to leach the excess B from the root zone, if sufficient water of good quality (low B concentration) is available. Alternatively, liberal application of N fertilizer, especially calcium nitrate, may alleviate the problem (Bradford 1966). Additions of lime and organic matter have also been reported to be effective (Olsen 1972).

Sweet potato cultivars vary in their tolerance of B toxicity, and breeding programs by the International Potato Center (CIP) have been successful in identifying genotypes with useful levels of tolerance, as well as having good agronomic and quality characteristics (Chávez et al. 1995).

Manganese toxicity

Manganese (Mn) is frequently an abundant constituent of soils, but its low solubility at neutral and alkaline pH prevents excessive uptake by plants. Therefore, Mn toxicity is nearly always associated with acid soils. Waterlogging may also induce or exacerbate Mn toxicity, as anaerobic conditions cause higher oxides of Mn to be reduced to plant-available Mn^{2+}. Thus in some instances Mn toxicity may appear during wetter periods, with plants recovering as the soil dries out. Manganese is also a component of some fungicides, and may accumulate through repeated use of these fungicides, especially to crops grown on sandy soils.

Symptoms of manganese toxicity

Like B toxicity, Mn toxicity causes necrotic spots to appear on the older leaves of the vine. Manganese toxicity also frequently causes chlorosis, most severe on the younger leaves, due to an induced Fe deficiency. Either one or both of these symptoms may be observed in crops affected by Mn toxicity (Plate 5a).

Symptoms on older leaves begin with the appearance of small, irregularly shaped patches of pale tissue in interveinal zones (Plate 5b). The pale tissue is slightly sunken, and small (0.5–2 mm), roughly circular spots of dark necrosis soon develop (Plate 5c). The necrotic spots are usually more concentrated towards the tip and margins of the leaf. They are scattered within the interveinal tissue, and do not usually form a regular alignment between the main veins, as is seen in the case of B toxicity or salinity. These lesions may be associated with the blackening of minor veins on the lower side of the leaf (Plate 5e). The necrotic lesions multiply and coalesce until they occupy most of the leaf area (Plate 5d). Affected leaves eventually turn yellow and are shed.

The necrotic spots are caused by the accumulation of Mn in the tissue over a period of time. At relatively mild levels of Mn toxicity, necrotic spots on older leaves may be associated with little apparent reduction in vine growth. At greater severity, root function is impaired, and stunting in conjunction with Fe deficiency symptoms may develop, often without necrotic spots. Symptoms indicative of induced Fe deficiency are a pale yellow to white interveinal chlorosis of young leaves, and eventually necrosis of the young leaves and apex, resulting in arrested growth (Plate 5f).

Possible confusion with other symptoms

The necrotic lesions caused by Mn toxicity may be confused with those of salinity or B toxicity. In the case of Mn toxicity, necrotic spots are smaller and more evenly scattered across the interveinal tissue, rather than being predominantly aligned midway between the main veins. The spread of necrosis around the leaf margin is not usually observed until after extensive areas of interveinal tissue have been engulfed.

The necrotic lesions may also be mistaken for those caused by fungal pathogens, particularly *Cercospora bataticola*, which produces small, dark leaf spots (Clark and Moyer 1988). Fungal lesions may be recognised by their random distribution on the leaf blade. While they may be more common on older leaves, they do not show a consistent decrease in severity from the oldest to younger leaves.

Diagnostic soil and plant tissue tests

A critical concentration of 1600 mg Mn/kg in the 7th to 9th youngest leaf blades was associated with growth reductions due to Mn toxicity, in solution culture experiments using cv. Wanmun (Table 3). Concentrations as high as 8000 mg Mn/kg were measured in severely affected plants. However, we measured a concentration of 1500 mg Mn/kg in field-grown plants showing severe symptoms and considerable stunting. Rufty et al. (1979) found that tolerance of tobacco to Mn toxicity increased with increasing temperature, despite greatly increased concentrations of Mn in the leaves of plants grown at the higher temperature. The combined effect of these two factors meant that the critical tissue concentration associated with the appearance of symptoms increased seven-fold, from 700 to

5000 mg Mn/kg, with an increase in the day/night temperature regime from 22/18°C to 30/26°C. It is likely that sweet potato crops experience a similar interaction between Mn toxicity and temperature. Crops have been observed to recover from Mn toxicity as the season becomes warmer. This obviously makes interpretation of tissue Mn concentrations difficult.

In soils, levels of total (perchloric acid extractable) Mn of more than 2000 mg/kg are regarded as high (Landon 1991). Measurement of 'easily-extractable' Mn is more common, using chelating agents such as diethylenetriamine–pentaacetic acid (DTPA) as extractant (Rayment and Higginson 1992). Concentrations of DTPA-extractable Mn above 45 mg/kg are considered potentially harmful to root crops such as potato and carrot (CFL 1983). In conjunction with low pH (<5.3 measured in water) or waterlogging, such concentrations may indicate a risk of Mn toxicity.

Correction of manganese toxicity

As Mn toxicity often results from low soil pH, it can often be corrected by application of lime or dolomite to raise the pH above about 5.3 (measured in 1:5 soil:water). If the problem is associated with waterlogging, improved drainage may be effective. Raising the height of mounds or ridges may be enough to avoid problems associated with waterlogging in most situations.

Plate 5. Manganese toxicity

a) Symptoms of Mn toxicity in cv. Puerto Rico, grown on the Atherton Tableland in northern Australia. Necrotic spots caused by Mn accumulation are seen in conjunction with pale yellow interveinal chlorosis caused by induced Fe deficiency.

b) Early symptoms of Mn toxicity in cv. Wanmun: pale green and sunken patches in interveinal zones of an older leaf.

c) Dark necrotic spots developing in interveinal tissue on an older leaf of cv. Wanmun.

d) Extensive areas of necrotic tissue formed by the expanding and merging lesions, and yellowing of the remaining leaf tissue indicating the onset of leaf senescence.

Plate 5. Cont'd

e) The underside of an affected leaf, showing blackening of veins.

f) Severe Fe deficiency resulting from excess Mn, causing tip death in cv. Markham.

Salinity

High levels of soluble salts in the soil (usually sodium chloride or sodium sulfate) affect the plant in a number of ways. As the osmotic pressure of the soil solution is increased, plants become more susceptible to drought injury. Also, excessive uptake by the plant of Na, chloride or sulfate may result in toxicity symptoms on the vines. In addition to the direct effects on plant function, high Na concentrations have detrimental effects on soil properties, including surface crusting, increased bulk density, poor aeration and waterlogging. Plants grown in saline-alkaline soils may also be suffering effects of high soil pH, including low availability of the micronutrients Fe, Mn, Cu and Zn.

Salinity-alkalinity problems are associated mainly with coastal areas, or with irrigated crops in areas of low precipitation and high evaporation. In this case, salts may accumulate in the root zone either from the irrigation water or from raising a saline water table.

Symptoms of salinity stress

The separate effects of toxic concentrations of Na, Cl and sulfate have not been studied in sweet potato. The following symptoms were observed in solution culture studies, in which sodium chloride (NaCl) concentrations of 25, 50 100 or 200 µM were applied (equivalent to electrical conductivities in solution of 2.9, 5.6 10.7 or 20.4 dS/m respectively). Plants suffered severe growth reductions at concentrations in the root zone of 50–100 µM; 200 µM NaCl resulted in necrosis of the roots, shedding of leaves and eventually death of the whole plant (Plate 6a).

Typical symptoms are dark necrotic lesions on the older leaves, followed by rapid senescence and leaf drop (Plate 6b). Discrete chlorotic or water-soaked interveinal zones may appear before necrotic symptoms in some cultivars; they are centred between the main veins and may be separate, irregularly shaped zones of approximately 3–10 mm diameter (Plate 6c), or a continuous strip from near the midrib to the margin. Necrosis first appears on oldest leaves at the leaf margins or the tips of lobes (Plate 6d), from where it spreads into interveinal tissue (Plate 6e). As the symptoms spread to younger leaves, however, necrosis may arise more commonly as discrete, well separated lesions away from the margin, in the chlorotic zones if they are present, and concentrated midway between main veins (Plate 6f). These soon become linked with the margin by a spreading necrosis which surrounds the original spots (Plate 6g). The tissue at the centre of the original lesions is generally paler than the necrosis which subsequently spreads between them. The secondary necrotic tissue is typically a dark steel-grey. Leaf senescence proceeds with the spread of yellow chlorosis from the edge of necrotic zones, and leaves become dry and shrivelled before they are shed (Plate 6h). Leaves of axillary shoots, being younger, are less severely affected (Plate 6h).

Plate 6. Salinity

a) Growth of cv. Markham, in the presence of (left to right) 0, 100 µM and 200 µM NaCl in the root zone.

Plate 6. Cont'd

b) Salinity symptoms in cv. Hawaii, progressing from interveinal necrotic lesions on younger leaves to extensive necrosis and yellowing of older leaves. Leaves below the yellow leaf have been shed and only the leaves from axillary shoots remain.

c) Interveinal chlorotic zones on a mature leaf of cv. Wanmun, before the development of necrotic lesions.

d) The first necrotic lesions developing at the margins of an older leaf of cv. Markham.

e) Necrosis spreading from marginal to interveinal zones on an older leaf of cv. Hawaii.

f) Discrete interveinal lesions developing on a mature leaf of cv. Wanmun.

Plate 6. Cont'd

g) On an older leaf of cv. Markham, the initial lesions have become linked by a dark, spreading necrosis.

h) Leaf senescence in cv. Markham progressing from yellowing around the necrotic zones to complete desiccation of the leaf. Young leaves on axillary shoots exhibit no symptoms.

i) Wilting and leaf abscission in cv. Wanmun without development of extensive necrotic lesions, resulting from root death at high salt concentrations.

In severe cases in which the roots are extensively damaged, the plant becomes slightly wilted and generally chlorotic, and older leaves may be shed before the development of extensive necrosis (Plate 6i). Necrosis of stem tissue may lead to the death of the shoot tip.

Salinity appears to have a greater effect on the development of storage roots than on the vines. Greig and Smith (1962) studied effects of chloride salts of Na, K, Ca and Mg, and found that all produced similar symptoms on the vines, and that vine growth was correlated with the osmotic concentration in the culture solution. However, only Na treatments significantly reduced storage root yield. At levels of NaCl which had little or no effect on top growth, root yield was considerably reduced. Sodium-affected storage roots were smaller and thinner than those of healthy plants, but NaCl did not reduce the number of storage roots per plant (Greig and Smith 1961).

Possible confusion with other symptoms

Boron toxicity also produces necrotic lesions on older leaves. Necrotic lesions resulting from B toxicity usually remain discrete, or expand and fuse, while secondary necrosis does not spread until after the leaf has turned yellow, at an advanced stage of senescence. Those resulting from salinity are usually rapidly engulfed by a dark grey secondary necrosis, which spreads from the margin into interveinal tissue without being preceded by chlorosis.

Diagnostic soil and plant tissue tests

In the case of NaCl salinity, the chloride concentration in the leaf tissue appears to correlate more closely with growth response, and to be less variable among cultivars, than the Na concentration. This suggests that Cl is the more toxic element to sweet potato, so the response obtained is that of Cl toxicity. A critical concentration for Cl toxicity of 1.5% (15,000 mg Cl/kg) was determined in 7th to 9th youngest leaf blades in solution culture experiments (Table 3). In a soil-based experiment, a lower critical concentration, of approximately 0.9%, appeared to apply (J. Yauwo, pers. comm. 1996). This may be due to the effect of salinity on plant water uptake, which became limiting in the soil-based culture.

Electrical conductivity of the soil saturation extract (EC_e) is the most commonly quoted measure of soil salinity. Crop species vary widely in their salt tolerance, with an EC_e of 4 dS/m corresponding to 50% yield reduction in sensitive crops, and over 10 dS/m for highly tolerant crops. Sweet potato is regarded as moderately tolerant of salinity, suffering a 50% yield reduction at 6 mS cm^{-1}. The threshold for response of sweet potato to salinity is approximately 2.5 dS/m (Bernstein 1964).

Soil measurements of both exchangeable Na, and exchangeable Na percentage (ESP = 100 × Exchangeable Na/Cation Exchange Capacity) are used to assess soil sodicity.

While a number of factors affect the amount of Na required to cause dispersion in a particular soil, as a rough guide soils with exchangeable Na greater than 1 cmol(+)/kg soil, or an ESP ≥15 may be regarded as potentially sodic (Landon 1991). In such soils, sweet potato crops may suffer from both Na toxicity, and the deterioration of soil permeability and structure caused by high Na.

Correction of salinity stress

Appropriate measures to overcome the detrimental effect of salt accumulation depend on a proper understanding of the complex of problems present at the site. Improved drainage and more efficient water delivery systems (minimising the volume of water required to maintain the crop) may slow salination of irrigated land so that seasonal rains may leach sufficient salt to maintain fertility. The application of gypsum may reduce the deleterious effects of Na on soil structure. Some benefit may be gained by leaching the salts with non-saline water to which gypsum has been added, or after application of gypsum to the soil. Provision should be made for the safe disposal of saline leachate.

The most practical solution in many instances may be to select salt-tolerant cultivars for use on affected areas. CIP researchers have had some success in selecting cultivars tolerant of salinity in coastal Peru (Chávez et al. 1995) and Bangladesh (International Potato Center 1995).

Nitrogen deficiency

Nitrogen (N) is one of the most abundant elements in plants and animals, as it is a major component of proteins. The amount of N required by a crop is large compared with the natural N reserves in most soils, and thus most crops respond positively to additional N, whether from animal manures or inorganic fertilizers. However, this pattern does not always hold for sweet potato. In some studies, N application was reported to reduce sweet potato yields (e.g. de Geus 1967; Bourke 1977b; Navarro and Padda 1983; D'Souza and Bourke 1986a). More commonly, the pattern is for low rates of N to increase yield to some extent, but higher rates to cause a yield decline.

The reason for this confusing response is that N supply has a strong influence on the distribution of dry matter within the plant, particularly affecting root growth relative to top growth. When N supply is high, plants tend to grow more tops relative to roots. In the case of sweet potato, high N may cause luxuriant growth of the vines at the expense of storage root yield.

Cultivars vary greatly in the level of N required to maximise yield, and in their tendency to reduce yield at higher levels of N. In particular, negative responses to N are more common in cultivars developed in low-fertility areas where soil amendments are not traditionally used. Jones and Bouwkamp (1992) reported that an application of 60 kg N/ha increased yields of three USA cultivars but decreased the yields of three African cultivars. Watanabe (1979) classified cultivars into those which readily produce luxuriant vine growth, frequently resulting in reduced yield of storage roots, and those which do not, these being better adapted to fertile soils and high N inputs.

There is a common belief in developing countries that N fertilizers are bad for sweet potato. This is unfortunate because N deficiency is very common. Large responses to N are often obtained on soils which have been heavily cropped in the past, or those subject to heavy leaching (Bourke 1977b, 1985b; Halavatau et al. 1996). The response to N may be poor, however, if deficiencies of other nutrients such as K are not also corrected (Bourke 1977b, 1985a). Sweet potato tends to respond better to composts of plant materials which contain high K relative to N, than to animal manures, which are lower in K (Bourke 1982; D'Souza and Bourke 1986b). However, this depends on the balance of nutrients present in the soil.

Improved N nutrition of the crop also leads to higher protein concentrations in the tubers (Constantin et al. 1974; Kimber 1976; Purcell et al. 1982), and this may be of considerable significance in communities which obtain much of their protein from sweet potato (Heywood and Nakikus 1982). In the Kaintiba District of PNG, for instance, the mean protein concentration in tubers sampled, at 0.62%, was less than half the average for the South Pacific region (Bradbury and Holloway 1988), suggesting that these crops were N deficient. The protein intake of PNG Highlanders is typically suboptimal unless supplemented by imported foodstuffs (Heywood and Nakikus 1982; Harvey and Heywood 1983).

Sweet potato has been shown to form root associations with the nitrogen fixing soil bacterium *Azospirillum brasilense* (Hill et al. 1983; Hill and Bacon 1984). Inoculation of crops with the bacterium increased the root yield and the N concentration in leaf tissue when no N fertilizer was applied (Crossman and Hill 1987; Mortley and Hill 1990). The prevalence of such symbiotic associations in the various regions and environments in which sweet potato is grown, and its significance for yield of subsistence crops, have not yet been investigated.

Symptoms of nitrogen deficiency

Deficiency of N causes dramatic reductions in growth of sweet potato plants (Plate 7a), and yet it is not easily recognised in the field. General symptoms are a uniform light green chlorosis of the leaves, and slow growth resulting in a delayed or sparse ground cover (Plate 7b). These symptoms may be obvious in comparison with healthy plants, but less apparent in a uniform crop.

The development of N deficiency symptoms vary according to conditions experienced by the crop. When N is initially adequate during the establishment phase but becomes depleted during crop growth, plants may appear normal or near normal in colour and habit, except for yellowing and premature shedding of older leaves due to remobilisation of N from these tissues (Plate 7c). In this case, the oldest leaves become uniformly yellow and slightly wilted. A pale coloured necrosis may spread from the tip or margins, but often the leaf is shed before it develops extensive necrosis. Necrotic tissue is supple rather than brittle.

Alternatively, if N supply is low throughout the growth of the crop, no senescence of older leaves may be evident. Symptoms of chronic N deficiency include uniformly pale colour (Plate 7b), reduced leaf size (Plate 7c), loss of the normal sheen resulting in a dull appearance of the leaves, thin spindly vines and reduced activity of axillary buds leading to less branching. In severe cases, small purple-pigmented or necrotic spots have been observed on the surface of older leaves of some cultivars (Plate 7e).

Increased anthocyanin pigmentation of the young leaves and especially the leaf veins is a noticeable symptom of N deficiency, which has been observed on all cultivars studied. However, it is not unique to N deficiency, since P- or S-deficient plants may show a similar symptom. This symptom is observed on plants suffering both types of N deficiency described above. In cultivars in which young leaves are normally pigmented, the purple colour is deepened and is retained for longer in the veins (Plate 7f), whereas the leaves of healthy plants change colour uniformly from purple to green. In cultivars which normally display little or no anthocyanin pigment, veins of the young leaves become red or purple. In some cultivars, the pigment may be most obvious on the upper surface of the leaves (Plate 7g); in others, the pigment may be almost absent from the upper surface but distinct on the lower leaf surface (Plates 7d & h). The red pigmentation usually also extends to the petiole and stem.

Possible confusion with other symptoms

Red veins on young leaves and yellowing of older leaves may also indicate P deficiency. However, most leaves on P-deficient plants remain dark green. Purple pigmentation on the older leaves before senescence is seen in some cultivars suffering P deficiency but not N deficiency.

Sulfur deficiency also induces a general chlorosis of the plant, and may be difficult to distinguish from N deficiency. Sulfur deficiency is indicated if the chlorosis is greater on the young leaves than on the old, if leaf veins are paler than the interveinal tissue, or if red-purple pigmentation is at least as great on the oldest leaves as on the youngest.

Plate 7. Nitrogen deficiency

a) Healthy (left) and N-deficient (right) plants of cv. Beerwah Gold, showing growth reduction, lack of branching, and small, pale green leaves.

b) A N-deficient plot (front) of cv. Markham compared with an area fertilized with N (rear), in a fertilizer trial at Lae, PNG.

Plate 7. Cont'd

c) Small leaf size and pale, dull colour caused by N deficiency (left) in cv. Markham, compared with a healthy leaf (right).

d) A crop in the Kabale district of Uganda, which has become deficient in N after establishment. Older leaves become uniformly yellow and limp, and younger leaves show reddening of veins on their lower surface. (Photo: J. Low)

e) Small purple spots on an older leaf of cv. Wanmun suffering severe N deficiency.

f) Pigmentation of young leaves of cv. Wanmun, showing retention of red pigment in the veins.

g) Red pigmentation of veins on the upper surface of young leaves of cv. Markham.

Plate 7. Cont'd

h) Red pigmentation visible only on the lower surface of veins in cv. Hawaii.

Diagnostic soil and plant tissue tests

A critical concentration of 4.0% N was determined for the 7th to 9th youngest leaf blades of sweet potato, on the basis of vine growth in solution culture over 28 days (Table 3). This agrees closely with the critical concentration in field-grown plants in Tonga, sampled at two months from planting, and corresponding to 90% of maximum yield (S. Halavatan, pers. comm.). The N concentration in leaves tends to decline with plant age (Spence and Ahmad 1967; Scott and Bouwkamp 1974), and it is likely that the critical concentration varies with the age of the crop. Leonard et al. (1949) found N concentrations in the first five expanded leaf blades, necessary for good yield, to be 4.7–5.0% N in early stages, or 3.0–3.8% N at harvest. Using a similar leaf sample taken at 10 weeks, Mascianica et al. (1985) gave a critical concentration of 4.9% N. Jackson (1972) found that yield responded linearly to N concentration, up to a maximum of 4.2%, in the 5th–6th youngest leaf blade sampled at 10 weeks. Walker and Woodson (1987), however, found no significant difference in leaf blade N concentration with age of sampling from 44 to 97 days. They determined a critical concentration associated with 95% of maximum marketable root yield of 3.0–3.2% N in the fifth leaf blade from the apex.

These differing estimations of critical N concentration may be due in part to cultivar differences in response to N. Villareal et al. (1979) found that cultivars differed in the relationship between crop age and protein concentration in sweet potato shoot tips: some showed a steady decline in protein concentration from 40 to 120 days, while others maintained their protein concentration throughout the growing season.

The concentration of nitrate-N in the petiole is often used to assess the N status of crops. Lorenz (1965) quoted nitrate-N concentrations in the petiole of the sixth leaf of sweet potato in mid season, as 1500, 2500 and 3500 mg/kg for deficient, intermediate and sufficient N status, respectively. However, Walker and Woodson (1987) found that, while petiole nitrate-N concentration was sensitive to N supply, it was highly variable among cultivars and with age of sampling, and was a poor predictor of root yield.

In the field, fertilizer test strips may be used to confirm a deficiency of N, and to distinguish N from S deficiency. For example, urea (containing only N) and sulfate of ammonia (containing both N and S) can be applied to separate strips of one or two ridges, at a rate of about 50 kg N/ha (e.g. 1 kg N for a 2 m × 10 m strip). If plants green up equally with either sulfate of ammonia or urea, it would be appropriate to conclude that they were suffering from N deficiency; if only the sulfate of ammonia produces a response, the problem would be S deficiency.

Soil N measurements are difficult to interpret as sources of N with differing availability to plants are not distinguished. As a rough indication, concentrations below 0.1% N (Kjeldahl method) are regarded as very low, while concentrations of 0.5–1.0% N may be adequate for maximum crop growth (Landon 1991, quoting Metson 1961). On a sandy-loam with moderate organic matter content, Mascianica et al. (1985) determined a critical soil nitrate-N concentration for sweet potato of 37 mg N/kg soil, sampled 29 days after transplanting.

Correction of nitrogen deficiency

A sweet potato crop of 20 t/ha removes approximately 87 kg N/ha, if roots and vines are harvested (Table 4). The optimum rate of fertilization will depend on the amount of plant-available N in the soil, and on yield potential, which may be dictated by the available soil water and rainfall. Reported recommendations for application of N fertilizers to sweet potato generally lie between 30 and 90 kg N/ha (de Geus 1967). Overfertilization with N may lead to reduced yields, as it may encourage excessive vine growth at the expense of the storage roots.

Organic mulches of various materials including foliage from leguminous trees and animal manure may be used to provide N to the crop. Nitrogen also accumulates in organic material during a fallow period, particularly if the fallow is dominated by leguminous species. This becomes available to the subsequent crop gradually as the organic material decomposes. Burning the fallow residue rapidly releases the N from the organic matter, but much of this is lost to the atmosphere as N oxides (NO_2, N_2O) during the burn. The remaining nitrate is readily lost through leaching and denitrification, and the long-term effect of burning is to reduce the supply of N and other nutrients, as well as degrading soil physical properties. Nitrogen supply can be increased by growing a leguminous crop, such as peanut in rotation with sweet potato, but only if the crop residues remain in the field.

Nitrogen deficiency is sometimes associated with waterlogging of the soil. Under anaerobic conditions, soil bacteria quickly convert soil nitrate into nitrogen gas (N_2), which is lost to the atmosphere. Improved drainage can be achieved by increasing the height of ridges or mounds, keeping the troughs between them clear of weeds, and providing adequate channels for excess water to leave the field. Sweet potato is sensitive to waterlogging, and crops tend to yield poorly if waterlogging occurs for even a short period, particularly in the early stages of crop growth. Therefore, good drainage is necessary regardless of N supply.

Sulfur deficiency

Sulfur (S) deficiency may not be recognised easily in the field, as the symptoms are not obvious except in severely deficient plants. However, recent soil fertility studies have shown that sweet potato production may be frequently limited by low S supply. Deficiencies have been documented on ultisols from the PNG Highlands (Dowling et al. 1994), andesitic volcanic ash soils in Tonga (Halavatau et al. 1996) and coralline soils from coastal PNG (M. Johnston, unpublished data).

On the other hand, it appears that sweet potato may be more tolerant of soils low in S than are a number of other crops. Vance et al. (1983), reviewing literature on S deficiency in the agriculture of PNG, noted numerous reports of S deficiency in legume, cereal and tree crops, but none in sweet potato. On low-S soils of the North Guadalcanal Plains in the Solomon Islands, Chase and Widdowson (1983) noted that, although a number of pasture grass, legume and tree species responded to additional S, sweet potato did not.

Sulfate is readily leached from the soil, and weathered soils in high rainfall areas frequently have a low S status. Burning of vegetation results in gaseous losses of S (as SO_2) from the farming system. Regular high levels of P application may displace S from the soil matrix and contribute to S depletion. In areas close to industrial or volcanic activity, rainfall may provide sufficient S to maintain crop production. Sea spray may also be a significant source of S in coastal areas, but S deficiency has been reported even on small islands (Halavatau et al. 1996) where aerial accession of S is likely to be in excess of 10 kg/ha/year (Fox et al. 1983).

Symptoms of sulfur deficiency

Sulfur deficiency results in a uniform pale green chlorosis throughout the plant (Plate 8a). Veins do not retain a green colour, and in many cases, they may be more chlorotic than the interveinal tissue (Plates 8b & c). In cultivars in which young leaves are normally green (ie. lacking red pigmentation), the youngest leaves may appear chlorotic earlier or more severely than mature leaves. This is not obvious in cultivars with red or purple tips.

A considerable reduction in growth may be suffered without the appearance of any visible symptoms. Clear symptoms are associated with severe stunting, accompanied by a reduction in leaf size (Plate 8d), and reduced activity of axillary buds, resulting in less branching (Plate 8a).

Purple or red-brown pigmentation may develop on both young and old leaves. In cultivars with normally green shoot tips, the petiole and margins of young leaves may become red, and this may extend in a mottled pattern over interveinal areas of the upper leaf surface (Plates 8e & f). A similar pattern is seen on the oldest leaves, although it is often confined to the tips of lobes rather than extending around the entire margin. Leaves of intermediate age generally lack purple pigmentation. In plants which normally have purple-pigmented shoot tips, pigment is lost in the normal manner as leaves mature (in contrast with N deficiency).

Possible confusion with other symptoms

The general growth reduction and chlorosis of the whole plant closely resemble the symptoms of N deficiency. A useful distinguishing character is the red pigmentation of veins of young leaves in N-deficient plants, but no pigmentation on the oldest leaves. While S deficiency may increase pigmentation of young leaf margins and petiole, a strong veinal pattern is not typical, and the oldest leaves are usually also pigmented.

Diagnostic soil and plant tissue tests

A critical concentration of 0.34% S in the 7th to 9th youngest leaf blades was estimated in solution culture studies using the cv. Wanmun (Table 3). In healthy plants, concentrations were generally between 0.35 and 0.45% S.

The chemistry of S in soils is complex and there is no reliable measure of plant-available S which can be applied over a wide range of soil conditions. Total S measurements may be related to crop response to S fertilizer only when the soil S reserves are very low. Calcium phosphate extraction is used to estimate the

soluble plus adsorbed sulfate fraction (Rayment and Higginson 1992). This may be used to predict S deficiency in soils with a low organic matter content, but in most humid tropical soils, S is predominantly supplied from the degradation of organic material (Landon 1991). Landon (1991) quoted approximate critical levels of 200 mg/kg total S, or 6–12 mg/kg extractable S, below which S deficiency is likely in soils with a low organic matter content.

In the field, fertilizer test strips may be used to distinguish S deficiency from N deficiency. If plants green up with sulfate of ammonium, but not with urea, it would be appropriate to conclude that they were suffering from S deficiency; if both treatments were equally effective, the problem would be N deficiency.

Correction of sulfur deficiency

Sulfur deficiency is usually corrected by the addition of an S-containing fertilizer. The fertilizer chosen may be primarily for supplying S, such as gypsum or elemental S, or S may be added as a constituent of fertilizers intended to supply other nutrients, such as ammonium sulfate (24% S) or single superphosphate (11% S). It should be noted that many 'high-analysis' fertilizers such as triple superphosphate, monoammonium phosphate, diammonium phosphate and urea, contain only trace amounts of S.

Plate 8. Sulfur deficiency

a) Healthy (left) and S-deficient (right) plants of cv. Beerwah Gold, showing reduced growth, lack of branching, small size of leaves and general pale colour resulting from S deficiency.

b) Mild S deficiency in cv. Wanmun, resulting in chlorosis which is more pronounced on and adjacent to the leaf veins than on the interveinal tissue.

Plate 8. Cont'd

c) Mild S deficiency in cv. Hawaii, in which the veins appear paler than the surrounding tissue.

d) Chlorosis and reduced leaf size in S-deficient cv. Hawaii (L), compared with healthy leaves (R).

e) Chlorosis is accompanied by purple pigmentation of the petioles and margins of young leaves of cv. Beerwah Gold.

f) Pigmentation on margins and interveinal areas of a young leaf of cv. Lole.

g) An older leaf of cv. Hawaii, showing pale veins and purple pigmentation extending from tips to interveinal patches of the leaf blade.

The maintenance of a high organic matter content in the soil will also increase the availability of S to the roots, and decrease the rate of nutrient leaching. Burning of crop and fallow residues leads to direct losses of S to the atmosphere, and loss of organic matter inputs to the soil. This practice is particularly ill-advised where S supply is poor.

Manganese deficiency

Manganese (Mn) is present in the soil as free Mn^{2+}, which is readily available to plants, and as oxides of low solubility. The proportion of Mn in various forms in the soil is dependent both on chemical reactions and on microbial activity. High soil pH greatly reduces the solubility of soil Mn, and therefore its availability to roots. Thus, Mn deficiency is most likely to occur in soils that are alkaline or have been limed. Johnston (1996) suspected Mn deficiency in sweet potato grown on coralline soils near Madang, PNG, although sweet potato was much less affected by the disorder than was yam (*Dioscorea esculenta*).

Microbial activity in the soil results in the oxidation of Mn^{2+} to insoluble higher oxides. As this is favoured in well-aerated soils, Mn deficiency may arise intermittently during drier periods, or appear more severe in well drained parts of the field. Cycles of Mn deficiency and recovery may result in symptoms normally associated with young leaves appearing on older parts of the plant.

Symptoms of manganese deficiency

Usually the first sign of Mn deficiency in sweet potato is an indistinct interveinal chlorosis throughout the plant, but particularly on leaves of intermediate age (Plate 9a). The green zones around major veins are relatively broad and fade gradually toward the interveinal zone. In some cultivars, the chlorosis may appear more general than interveinal. Chlorosis may be accompanied by drooping of the leaves (although they remain turgid), slight puckering of the leaf surface or downward curling of the leaf margins (Plate 9b). The drooping is caused by bending of the petiole and is not accompanied by wilting of the leaf blades, nor does it progress to the point of leaf senescence and abscission.

On expanding leaves, small areas between minor veins become pale and sunken, and eventually develop into necrotic spots. While all interveinal zones of the leaf are affected, they do not develop at the same rate. Indeed, it is common to see a peppering of brighter, cleared spots across the leaf blade (Plate 9c). Plate 9d shows the development of the necrotic pits in detail. Initially, the leaves most affected are those which are rapidly expanding, a few nodes back from the tip. But as the symptom intensifies, both older and younger leaves are affected. Pits in older leaves tend to be darker and are often concentrated near the base of the midrib and main veins (Plate 9e). The young leaves become pale, thickened and brittle, and may curl under or buckle as they expand. When pitting occurs in very young leaves, their subsequent expansion causes the pits to develop into larger holes (Plate 9f). In severe cases only a lacy skeleton of the leaf remains. Severely-affected leaves become necrotic, the necrosis spreading from the tip or lateral margins (Plate 9g). The growing points at the shoot tip and in leaf axils remain active. Axillary shoots are generally less affected than the subtending leaf.

In cultivars where the leaf blade is deeply divided into lobes, the above symptoms may be poorly developed. Instead, small, chlorotic, thickened and deformed young leaves may be produced (Plate 9h). The leaf surface may be finely pitted especially in the vicinity of main veins.

Pillai et al. (1986) reported that Mn-deficient plants produced small, thin tubers which had brownish streaks in the flesh.

Possible confusion with other symptoms

Copper deficiency may also induce interveinal chlorosis and drooping in mature leaves, but in the case of Cu deficiency, there is a distinct wilting of the leaves, and this may progress to leaf senescence and abscission. This does not occur with Mn deficiency. The appearance of small, deformed young leaves may also be mistaken for Cu deficiency, although the latter does not typically induce chlorosis or pitting of the leaf surface.

The clearing of small islands of interveinal tissue may also be a symptom of Cu toxicity. In this case, however, the chlorosis usually only affects mature leaves, and may occur on only localised areas of the blade. Symptoms of Mn deficiency tend to be expressed uniformly across the leaf blade.

Diagnostic soil and plant tissue tests

A critical concentration of 19 mg Mn/kg in the 7th to 9th youngest leaf blades was determined in solution culture studies using cv. Wanmun (Table 3). In healthy plants, concentrations were generally between 26 and 500 mg Mn/kg.

Manganese availability in the soil may be estimated by extraction with a chelating agent such as diethylenetriaminepentaacetic acid (DTPA) (Rayment and Higginson 1992). Concentrations of DTPA-extractable Mn < 4 mg/kg are considered potentially deficient for a range of root and vegetable crops (CFL 1983).

Suspected cases of Mn deficiency can be confirmed by leaf painting with a 1% solution of manganese sulfate. Manganese application causes regreening of chlorotic tissue (Plate 9i), and in young leaves it arrests the development of interveinal pits and enhances expansion of the treated portion of the leaf blade.

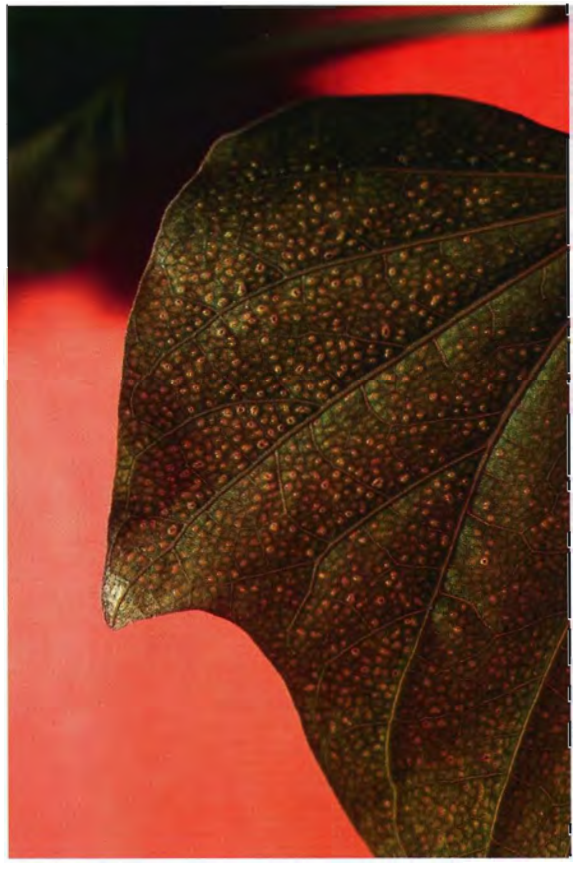
Plate 9. Manganese deficiency

a) Mild Mn deficiency symptoms in cv. Wanmun (right) compared with a healthy plant (left). Interveinal chlorosis is present on leaves of all ages, but especially those of intermediate age.

b) Interveinal chlorosis in cv. Markham accompanied by leaf drooping, slight puckering and downward curling of the leaf margins.

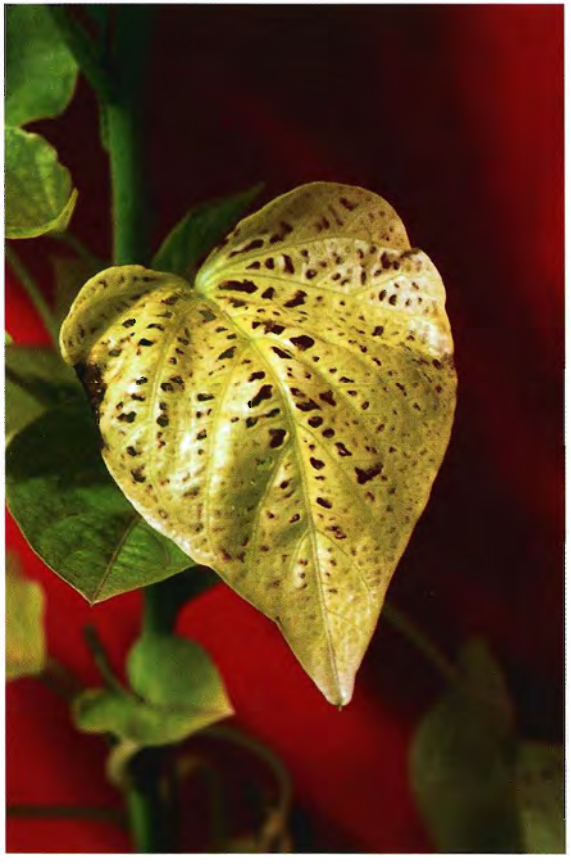
Plate 9. Cont'd

c) An expanding leaf of cv. Wanmun, in the early stages of the development of interveinal pits, seen as bright spots on the leaf.



d) Detail of the development of necrotic pits in a young, expanding leaf of cv. Wanmun.

e) Necrotic pits in a mature leaf of cv. Wanmun.



f) A young leaf of cv. Markham, in which the interveinal pits have developed into holes during the expansion of the blade.

g) Severely Mn-deficient young leaves of cv. Hawaii, showing chlorosis, curling and necrosis. The tip and axillary shoots are less severely affected.

h) Young leaves of cv. Lole showing chlorosis, thickening and deformity but without necrosis or holes. Fine pitting can be seen on the leaf surface.

Plate 9. Cont'd

i) A Mn-deficient leaf of cv. Wanmun, the right-hand side of which was painted with a 1% solution of manganese sulfate, showing complete recovery from chlorosis.

Correction of manganese deficiency

Except on some sandy soils, Mn deficiency usually arises due to soil conditions which limit the availability of Mn to plants. Therefore, fertilization of the soil with Mn may be ineffective, as the added Mn may become unavailable. Foliar sprays of 0.1% manganese sulfate or chelate, at 2 to 4 kg Mn/ha, may be effective (Tisdale et al. 1993; Weir and Cresswell 1993). The elevation of soil organic matter content, by mulching or composting, will help to improve the availability of Mn and other nutrients to the crop.

When deficiency occurs at neutral to acidic soil pH, Mn may be applied to the soil, as either manganese sulfate or oxide. Rates used for other crops are typically in the range of 10 to 20 kg Mn/ha. Soils low in Mn should not be limed above pH 6.4 (measured in water).

Copper deficiency

Copper (Cu) deficiency in sweet potato is encountered on some acid, sandy soils of low total Cu content, such as those in the vicinity of Mareeba in northern Australia (O'Sullivan et al. 1997b). It may also occur on calcareous soils, in which Cu availability is low due to its insolubility at high pH. In some organic soils, Cu may be tightly bound to soil constituents and poorly available to plants. Liming of soils low in Cu can lead to the appearance of Cu deficiency.

Symptoms of copper deficiency

A number of visible symptoms of Cu deficiency have been observed on both mature and young leaves, their extent and order of development varying among cultivars. Conspicuous symptoms on the vines are usually associated with considerable growth reduction (Plate 10a).

Chlorosis, wilting and drooping of mature leaves may be the first visible symptom of Cu deficiency (Plates 10b & c). Leaves of intermediate age are first affected, but in time the turnover of leaves will mean that the oldest leaves show symptoms (Plate 10c). The chlorosis is interveinal, with a gradual fading of colour with distance from the main veins. Usually the minor veins retain less green colour than the main veins, but may be sufficiently well defined to give the chlorosis a mottled appearance (Plate 10c). Chlorotic leaves may develop spots or patches of necrosis, which spread until the entire leaf is dead.

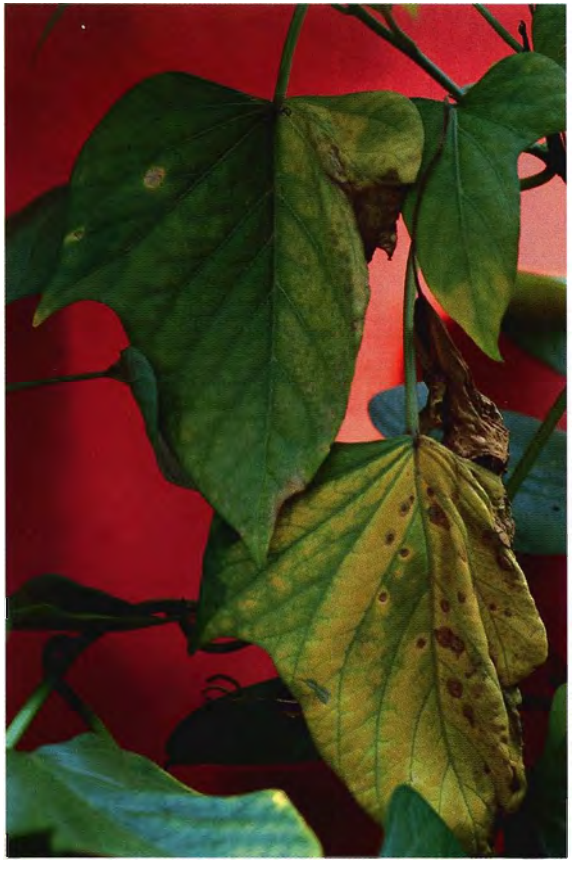
In some cultivars, necrotic spots have been observed on mature leaves without prior chlorosis. Initially the necrotic spots are small, dark and sharply defined (Plate 10d). They may be clustered close to the point of petiole attachment, or more commonly scattered over the leaf surface. The necrotic spots are clearly visible on the lower side of the leaf, and are often adjacent to minor veins (Plate 10d). Subsequently, a yellow chlorosis develops around them, spreading to encompass a number of lesions (Plate 10e). Later, the area between the initial lesions becomes necrotic (Plate 10e), and eventually the whole leaf dies.

Symptoms affecting the young leaves and growing point may develop earlier or later than those described above, and in some cultivars they may be the only visible symptoms. Initially, the surface of young leaves may take on a silvery appearance (Plate 10f). A variety of leaf deformities may occur (Plates 10f–h). New leaves are usually small and may be misshapen, puckered, or thickened (Plate 10g). They may develop holes due to uneven expansion of the blade (Plate 10h). Some reduction in internode length is common. Unlike Zn deficiency, narrowing and reduction in size of lateral lobes is not characteristic of Cu deficiency.

Plate 10. Copper deficiency

a) Growth reduction and symptoms caused by Cu deficiency in cv. Wanmun (right), compared with a healthy plant (left).

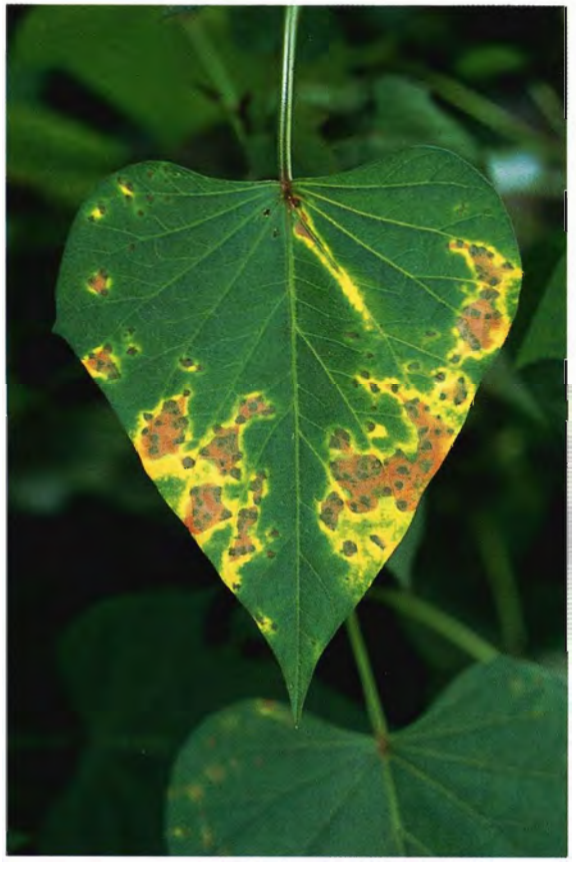
Plate 10. Cont'd



b) Chlorosis and wilting of mature leaves progressing to necrosis and leaf senescence in cv. Wanmun.

c) Mottled interveinal chlorosis, and the development of necrotic spots on the most severely affected leaves, in a Cu-deficient crop of cv. Centennial in northern Australia.

d) Small necrotic spots on leaves of cv. Wanmun, which may appear with no chlorosis: comparison of the upper (right) and lower (left) leaf surfaces.



e) Development of chlorosis and secondary necrosis around the initial necrotic lesions, cv. Wanmun.

f) Silvery appearance and puckering of young leaves as a result of Cu deficiency, in northern Australia (unnamed breeding line).

Plate 10. Cont'd

g) Symptoms of Cu deficiency in young leaves of cv. Centennial grown in northern Australia, including puckering, deformity and holes in the leaf blade.

h) Symptoms of Cu deficiency in young leaves of cv. Wanmun, including thickened veins, asymmetric leaf shape, upward cupping and holes due to uneven expansion of the blade.

i) A storage root from a Cu-deficient crop of cv. Centennial in northern Australia, with blackened areas of vascular tissue immediately below the root surface.

Pillai et al. (1986) observed that Cu-deficient sweet potato plants produced storage roots which were normal in external appearance but contained brown streaks in the flesh. Similar symptoms have also been observed in Cu-deficient crops in northern Australia (O'Sullivan et al. 1997b). Necrotic patches of vascular tissue may appear on the surface as a brown area (Plate 10i). Other roots, which appear healthy at harvest, may develop subsurface necrosis followed by extensive tissue breakdown after a short period of storage. Roots which appear sound may blacken rapidly after cutting. These storage disorders may occur in crops showing little or no visible symptoms on the vines.

Possible confusion with other symptoms

Interveinal chlorosis on mature leaves, resulting from Cu deficiency, may resemble deficiencies of Mg or Mn. In the case of Mg deficiency, the interveinal pattern of chlorosis is usually more pronounced, and internodes near the tip may be lengthened, whereas those in Cu-deficient plants are likely to be shortened. In the case of Mn deficiency, the symptoms are more uniform throughout the plant and do not generally progress to yellowing and senescence of mature leaves.

Zinc deficiency also causes the development of small, abnormally-shaped young leaves, but Zn-deficient leaves are usually chlorotic, elongated,

with reduced lateral lobing. They are usually not asymmetrical or puckered, as is often the case with Cu deficiency. Aphide may also cause puckering of young leaves.

Diagnostic soil and plant tissue tests

The critical concentration for Cu deficiency was found to be in the range 4–5 mg Cu/kg in the 7th to 9th youngest leaf blades (Table 3). Concentrations below 2 mg Cu/kg have been recorded in crops showing symptoms of Cu deficiency in northern Australia (O'Sullivan et al. 1997b).

Soil extraction using EDTA with ammonium bicarbonate has been recommended for evaluation of Cu status, particularly in alkaline, calcareous soils. Using this test, a critical concentration of 0.3–0.4 mg Cu/kg was determined for wheat (Best et al. 1985). Soil tests have not been calibrated for Cu deficiency in sweet potato.

A quick and inexpensive diagnostic test may be made by painting the surface of a young leaf with a dilute solution containing Cu (e.g. 0.25% $CuSO_4.5H_2O$ + 0.25% $Ca(OH)_2$). If Cu deficiency is the cause of the observed symptoms, the treated leaf will show reduced symptoms and increased expansion of the lamina over the following week. It is important to clearly label the leaf so that it can be identified on later inspection.

The result is most obviously seen if only one half of the leaf is painted, so it can be compared with the untreated half. A small amount of agricultural wetting agent or mild detergent may assist to ensure even wetting of the leaf surface, but this is not essential with sweet potato, as the foliage wets relatively easily.

Correction of copper deficiency

Copper may be applied to the soil or as a foliar spray. On alkaline or organic soils, foliar sprays may be more effective due to the rapid fixing of soil-applied Cu, but in general, soil application is considered preferable (Reuther and Labanauskas 1966). Rates of application for sweet potato crops have not been optimised. For other crops, soil applications of copper sulfate at 1 kg Cu/ha on acid, sandy soils with low organic matter content, to 7 kg Cu/ha on alkaline, peaty or heavy-textured soils have been used. Overfertilization with Cu can lead to Cu toxicity, so the minimum effective dose should be sought. A single application of Cu may be effective for up to 10 years (Weir and Cresswell 1993).

For foliar application, rates as low as 0.25 kg Cu/ha have been sufficient for wheat. Spray mixtures of 0.5% copper sulfate plus 0.5% hydrated lime (Asher and Lee 1975) or 0.5% copper oxychloride (Weir and Cresswell 1993) have been recommended for ginger and vegetable crops, respectively. Copper sulfate alone may cause leaf burn.

Molybdenum deficiency

Molybdenum (Mo) is unusual among the micronutrients in becoming less available to plants at low soil pH. Thus, Mo deficiency is usually associated with acid soils (pH <5.5), particularly those which are geologically old and highly leached. Mo deficiency has not been reported in sweet potato crops to date. As soils low in Mo are often also low in P and S, Mo may not be apparent unless P and S deficiencies have been corrected by fertilization (Johnson 1966).

Mo is required by plants in very small quantities. Its main (and possibly only) function in non-leguminous plants is as a component of the enzyme nitrate reductase, which is essential for the metabolism of nitrate, the main form of plant-available N in most soils. Therefore Mo deficient plants may appear as if they are deficient in N, having general chlorosis and stunted growth. Symptoms unlike those of N deficiency may occur as a result of nitrate accumulation in the tissue.

Symptoms of molybdenum deficiency

Although Mo deficiency has not been described in field-grown sweet potato, symptoms resembling those of N deficiency are expected. Such symptoms would include a general pale green colour, stunted growth with small leaf size, and possible reddening of veins on the young leaves.

In plants grown in solution culture, the following symptoms were associated with a mild growth reduction from Mo deficiency. In cv. Wanmun, older leaves developed silvery patches over interveinal tissue, as a result of the upper epidermis separating from the underlying tissue (Plate 11a). Eventually these patches became necrotic, but necrosis did not spread readily from the isolated interveinal patches (Plate 11b). In cv. Beerwah Gold, the young to recently mature leaves displayed a mild interveinal chlorosis, with some interveinal areas becoming necrotic (Plate 11c). In cv. Hawaii, chlorosis was more uniform, and the recently mature leaves commonly developed a marginal scorch, especially at the tips of lobes (Plate 11d).

Possible confusion with other symptoms

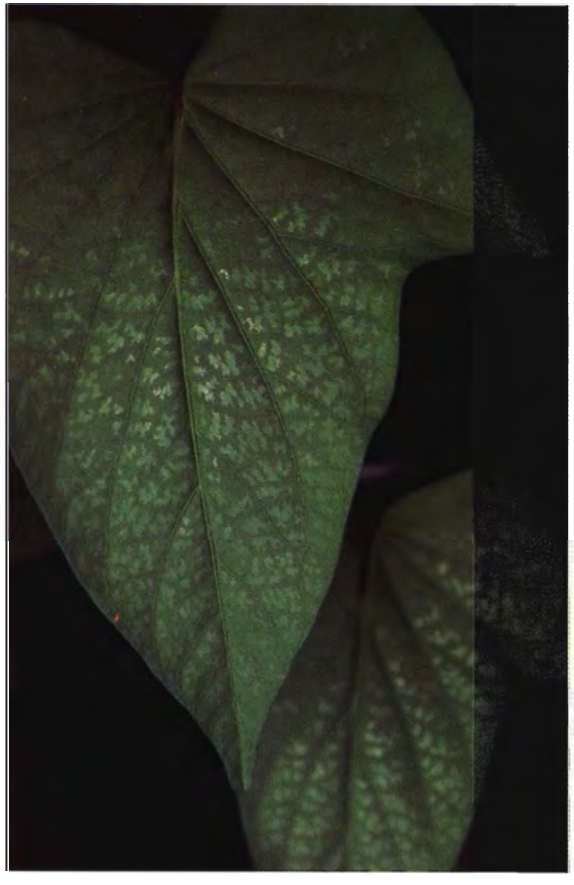
Molybdenum deficiency may induce symptoms similar to those of N deficiency, as Mo is required for the assimilation of nitrate taken up by the plant. Necrosis on the margins and interveinal areas of older leaves may bear some resemblance to salinity damage or B toxicity. However, these disorders generally cause much more extensive necrosis than has been seen in the case of Mo deficiency, and are not usually associated with acid soils.

Diagnostic soil and plant tissue tests

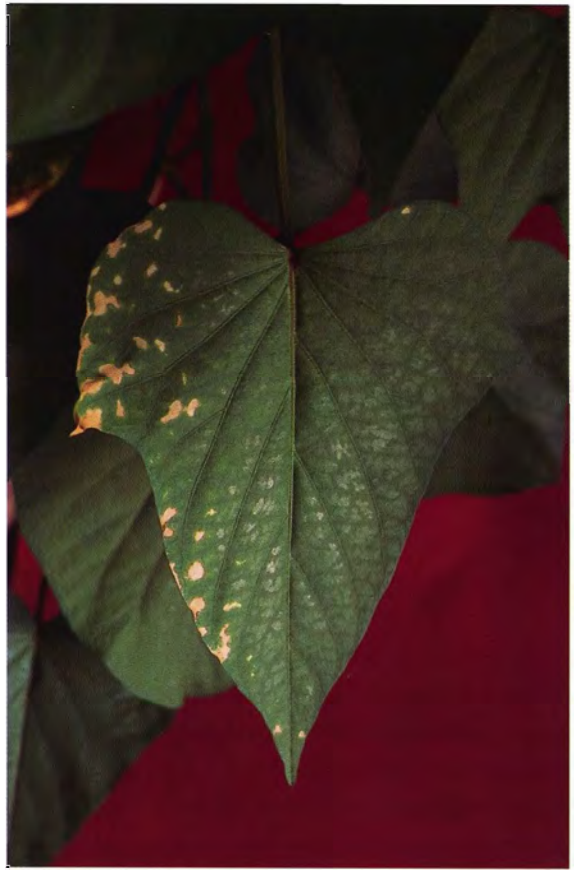
From solution culture studies, the critical concentration for deficiency of Mo was found to be approximately 0.2 mg Mo/kg in the 7th to 9th youngest leaf blades (Table 3). Equivalent leaves from healthy plants contained 0.5–7 mg Mo/kg.

As Mo is required for metabolism of nitrate, deficiency of Mo can result in accumulation of nitrate in the plant tissues. Measurements of sap nitrate concentration have been used to distinguish Mo deficiency from N deficiency in a number of crops, including sunflower (McDonald 1978) and potato (Ulrich 1993). A simple, semi-quantitative measurement of sap nitrate can be performed using nitrate test strips, such as those manufactured by Merck. Reagent-impregnated paper is mounted on the plastic strips, and changes from white to deep purple when wet with a solution containing nitrate, over the range 10–500 ppm. Sweet potato sap can be tested by cutting through a stem or petiole, and applying the side edge of the paper to the droplet of sap which exudes from the cut, allowing the moisture to move in by capillary action. The sap should not be applied directly to the paper surface, as it will stain darkly. Sap from Mo-deficient plants will cause a strong colour change, while that from N-deficient plants produces no change. Sap from healthy plants will usually give a weak reaction (Plate 11e). Note that S deficiency may also increase sap nitrate levels.

Plate 11. Molybdenum deficiency



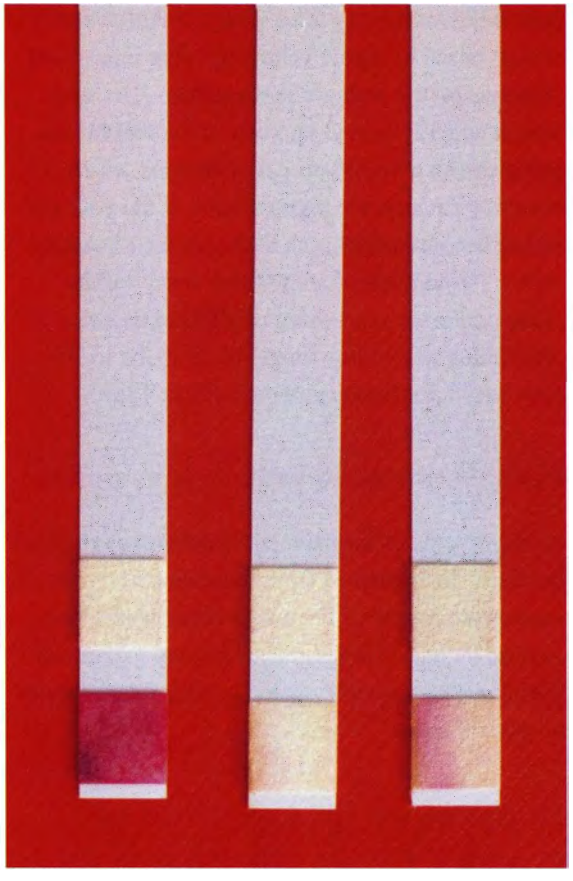
a) Silvery interveinal patches on mature leaves of cv. Wanmun, resulting from Mo deficiency.



b) Necrosis developing in the affected interveinal areas on a leaf of cv. Wanmun.

c) Interveinal chlorosis and necrotic patches on a mature leaf of cv. Beerwah Gold.

d) General chlorosis with marginal necrosis, particularly at the tips of lobes, of a young expanded leaf of cv. Hawaii.



e) Merckoquant® nitrate test strips wet with sap from (left to right) a Mo-deficient plant, a N-deficient plant and a plant adequately supplied with all nutrients.

Correction of molybdenum deficiency

Molybdenum deficiency is relatively easily corrected, either by the application of small quantities of Mo to the soil, or by raising the soil pH. Application of sodium molybdate or ammonium molybdate at rates of 0.2–0.3 kg Mo/ha should be sufficient to correct the disorder in most situations, and may be effective for several years. Sodium molybdate may also be applied as a foliar spray. A solution of 50 g sodium molybdate/100 L water has proven successful with other crops such as sunflower (Blamey et al. 1987). Some commercially-available fertilizer mixtures also contain Mo.

Liming to raise the soil pH above 5.5 is usually effective in alleviating Mo deficiency, and may improve conditions for crop growth in other ways also, such as through improved availability of P and alleviation of Mn or Al toxicity.

Zinc toxicity

Incidence of zinc (Zn) toxicity is rare and has not been reported in sweet potato crops. Cases in other crops have been reported on acid soils high in Zn, or on soils overfertilized with Zn fertilizer. Zinc is also a component of some fungicides, and may accumulate following their persistent use.

Symptoms of zinc toxicity

Zinc toxicity may cause severe growth reduction or prevent the establishment of transplanted cuttings. In solution culture experiments, concentrations higher than 10 µM Zn in the root medium decreased sweet potato growth (Plate 12a). At 50 µM Zn, growth was completely arrested and necrosis of roots was evident.

Specific visible symptoms are not always evident. Symptoms include the development of dark pigmented spots or blotches on older leaves (Plate 12b), or in more severe cases, red pigmentation throughout the vine, particularly on petioles and around margins and veins of the leaf blades (Plate 12c). However, pigmentation is not observed in all cultivars. Severe damage to the roots at high Zn concentration may cause general chlorosis and wilting (Plate 12d).

High levels of Zn inhibit the uptake of Fe, and it is common to find symptoms of severe Fe deficiency induced by Zn toxicity. Iron deficiency is characterised by a pale yellow to white interveinal chlorosis on the younger leaves (Plate 12e), and may eventually lead to necrosis of the leaf blades and growing point (Plate 12f). Cultivars which normally have purple pigmentation in the youngest leaves become bright pink at the tip. Leaf painting with a 1% solution of ammonium ferric sulfate causes dramatic regreening of affected tissue, confirming that a poor supply of Fe to the leaf is responsible for these symptoms (Plate 12g).

Possible confusion with other symptoms

Symptoms of Zn-induced Fe deficiency may not be easily distinguished from those caused by an inadequate supply of Fe, unless other symptoms such as increased red pigmentation are expressed. Zinc toxicity may be revealed by chemical analysis of the leaf tissue.

Diagnostic soil and plant tissue tests

Concentrations of more than 70–85 mg Zn/kg in the 7th to 9th youngest leaf blades were associated with reduced growth due to Zn toxicity in solution culture experiments (Table 3). Concentrations above 150 mg Zn/kg corresponded with severe stunting, while concentrations as high as 900 mg Zn/kg were measured in severely affected plants.

In soil, a DTPA-extractable Zn concentration above 10 mg/kg is considered potentially harmful in acid soils. 'Total Zn' concentrations in soil (perchloric acid extractable Zn) usually fall in the range 10 to 300 mg/kg, with concentrations above 150 mg/kg regarded as high (Landon 1991), and likely to result in reduced plant growth.

Correction of zinc toxicity

Should Zn toxicity be found to occur in field-grown sweet potato, liming to raise the pH of the soil may alleviate the problem by reducing the concentrations of plant-available Zn. Large applications of P fertilizers may also have a beneficial effect.

Plate 12. Zinc toxicity

a) Growth of sweet potato cv. Wanmun in solutions containing (left to right) 10 μM, 20 μM, 30 μM and 40 μM Zn. Growth in 10μM Zn was similar to that of control plants receiving trace amounts of Zn. Chlorosis of plants grown at the higher Zn levels is due to induced Fe deficiency.

b) Dark red-brown pigment spots, concentrated near veins on an older leaf of cv. Wanmun.

c) Development of purple pigmentation on cv. Hawaii as a result of Zn toxicity.

Plate 12. Cont'd

d) Severe wilting due to root damage from Zn toxicity in cv. Markham. This cultivar does not produce anthocyanin pigments in response to Zn toxicity.

e) Typical symptoms of Fe deficiency in cv. Lole, induced by high Zn concentrations.

f) Symptoms of severe Zn toxicity in cv. Wanmun, including complete chlorosis and marginal necrosis of young leaves caused by induced Fe deficiency, stunted growth and dark red spots on the older leaves.

g) Regreening of chlorotic leaf tissue two days after the application of 1% solution of ammonium ferric sulfate to the leaf surface, demonstrating that the chlorosis was caused by Fe deficiency.

Copper toxicity

Toxic levels of copper (Cu) rarely occur naturally in soils. However, Cu may accumulate due to application of sewage sludge, pig slurries or mine slag, or more commonly through persistent use of Cu-containing fungicides or fertilizers (Tiller and Merry 1981). Mild symptoms of Cu toxicity have been observed on sweet potato in northern Australia on land with a history of heavy fungicide use (O'Sullivan et al. 1997b).

Symptoms of copper toxicity

Excess Cu is highly toxic to sweet potato (Plate 13a), concentrations as low as 5 µM in the root zone being sufficient to cause significant growth reduction. Concentrations above 20 µM prevent root growth and result in the death of the transplanted cutting. Root damage may cause severe wilting, and death of the shoot tip (Plate 13b). Unlike Zn toxicity, there is little or no general chlorosis of the leaves, nor increased red pigmentation.

Moderate levels of Cu toxicity may be accompanied by a pale green to white interveinal chlorosis on mature leaves (Plates 13c–e). Often the symptom appears on only a few leaves on the vine, with both younger and older leaves lacking chlorosis (Plate 13a). Small interveinal areas become pale and slightly sunken, while the tissue around both major and minor veins retains normal coloration. The effect may vary from a faint mottle (Plate 13c) to a striking, sharply defined pattern (Plate 13e). The symptom is often unevenly distributed across the blade, affecting one region, such as the leaf tip or a lobe, more than others (Plate 13d).

Like toxicities of Mn and Zn, Cu toxicity may also induce symptoms of Fe deficiency, seen as a conspicuous pale yellow interveinal chlorosis on the younger leaves (Plate 13a).

Possible confusion with other symptoms

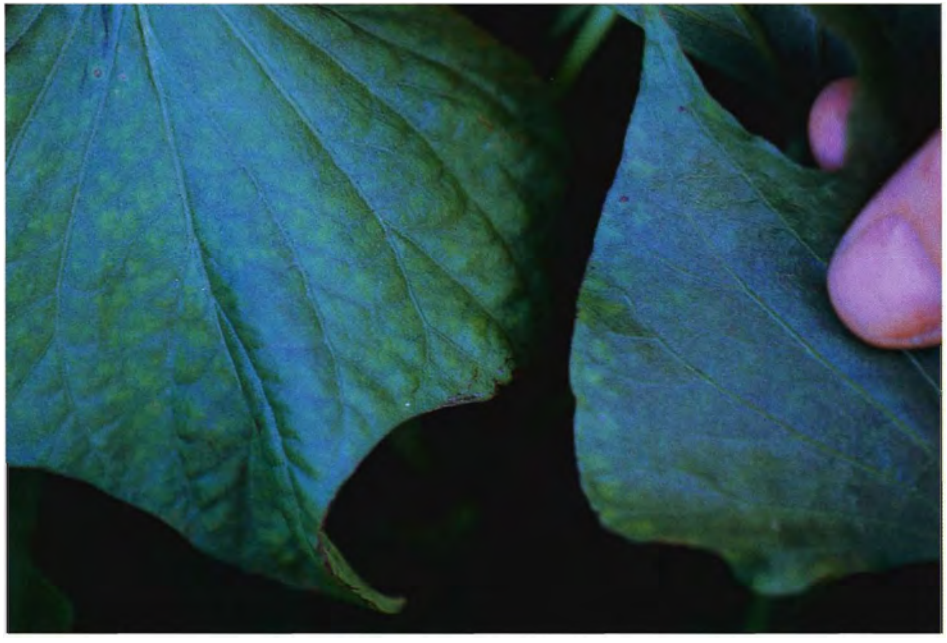
The interveinal chlorosis may be confused with the pitting caused by Mn deficiency. In the case of Cu toxicity, it is most intense on mature rather than on younger leaves and is not accompanied by a general chlorosis of the leaf blade. Symptoms of Mn deficiency typically affect the entire blade uniformly, while those of Cu toxicity may be localised or unevenly distributed.

It may be difficult to distinguish between severe Cu toxicity and Zn toxicity, although the latter more frequently causes increased red pigmentation on the vines. Some knowledge of the site history and a chemical analysis of leaf tissue may be necessary to confirm the diagnosis.

Plate 13. Copper toxicity

a) Growth of sweet potato cv. Wanmun in solutions containing (left to right) trace, 8 µM and 12 µM Cu.

b) Wilting due to severe root damage from Cu toxicity in cv. Markham.



c) A mild interveinal mottle caused by excess Cu in older leaves of cv. Beerwah Gold, grown in northern Australia.

Plate 13. Cont'd

d) Interveinal chlorotic spots on an older leaf of cv. Wanmun at moderate levels of Cu toxicity. The chlorotic spots are localised or distributed unevenly on the leaf blade, and are sharply defined, with the remaining tissue retaining a uniform green colour.

e) Striking white interveinal chlorosis resulting from Cu toxicity on a mature leaf of cv. Wanmun, affecting larger interveinal areas, but adjacent tissue remains dark green.

Diagnostic soil and plant tissue tests

No soil tests have been calibrated for Cu toxicity in sweet potato. For many crops, 'total' soil Cu concentration (perchloric acid extractable) above 100 mg/kg have been regarded as high (Landon 1991), while concentrations of DTPA-extractable Cu above 20 mg/kg are potentially toxic in acid soils (CFL 1983). For citrus, soils containing more than 112 kg/ha total Cu in the top 15 cm, using Spencer's test (Spencer 1954), are considered at risk, particularly if the soil pH is below 6.5 (Koo et al. 1984).

Copper toxicity appears to affect primarily the plant roots, and therefore the concentration in the above-ground parts may be a poor indicator of plant status. Alva et al. (1995) found no significant increase in foliar Cu concentrations in citrus trees, as soil Cu was increased to levels which decreased growth. In sweet potato, critical tissue concentration for toxicity of 15.5 mg Cu/kg in the 7th to 9th youngest leaf blades was determined in solution culture (Table 3). However, analyses of healthy field-grown plants have been found to commonly exceed this concentration. In crops where no fungicides have been applied, concentrations above 30 mg Cu/kg have been recorded, while crops receiving fungicidal sprays may contain much higher concentrations without ill effect. In north Queensland, a concentration of 270 mg Cu/kg was measured in plants showing mild symptoms of Cu

toxicity, while over 800 mg Cu/kg was measured in leaves of an apparently healthy crop. These high concentrations are probably achieved by direct adsorption of Cu onto the leaf surfaces from fungicidal spray, and reflect the recent spraying history of the crop. In contrast, toxicity is more likely to result from accumulated spray residues in the soil, and depends on the extended history of the site, combined with the ability of the soil to fix Cu.

Correction of copper toxicity

As Cu toxicity usually results from excessive application of Cu, prevention rather than correction should be stressed. Heavy applications of P fertilizers may reduce the availability of excess Cu to the plants. Liming may be beneficial, as Cu becomes less available to plants at high pH. Maintaining the soil pH above 6.5 has been recommended for amelioration of Cu toxicity in citrus (Koo et al. 1984).

Calcium deficiency

Calcium (Ca) is usually the dominant basic cation in soils, and only in acid soils with low CEC is the level of Ca likely to be so low that it limits plant growth. Strongly acid soils tend to be high in exchangeable Al, and the toxic effects of Al on root growth may exacerbate the symptoms of Ca deficiency. High levels of K and Mg may reduce Ca uptake, and therefore overuse of fertilizers containing these nutrients may lead to Ca deficiency.

Symptoms of calcium deficiency

The primary symptom of Ca deficiency is the development of necrotic tissue on young leaves. The necrosis usually begins along the lateral margins (Plate 14a), and extends inward mainly in interveinal tissue (Plates 14b & c). Necrotic tissue is mid to dark brown and brittle. Necrosis is not usually preceded by localised chlorosis, although the leaves may be uniformly paler than normal. The symptom is first seen on young expanding leaves, two or three leaves below the tip (Plate 14c), but as the disorder intensifies, newly formed leaves are affected, and finally the apex may die (Plate 14d).

Necrotic leaves may be prematurely shed. In some cultivars, leaf drop is spectacular, leaving some length of bare stem below the tip (Plate 14e). Other cultivars may show little abscission, retaining completely dry leaves on the stem (Plate 14d).

A second symptom may develop on older leaves, which is apparently unrelated to the necrosis of young leaves. Necrotic spots appear, which are either clustered along the main veins from the petiole attachment point to about half-way to the margin (Plate 14f), or scattered uniformly across the interveinal areas (Plates 14f & g). They are roughly circular with abrupt, irregular edges, and do not seem to accelerate leaf senescence. The necrotic tissue in this case is mid brown, and is not brittle. These symptoms were seen on two of four cultivars studied, and were reported also by Bolle-Jones and Ismunadji (1963) in the Indonesian cultivar Djarak.

Calcium deficiency inhibits root growth, and in severe cases root tips may die back. Solution concentrations of 13 μM Ca or below inhibit the growth of roots on sweet potato cuttings (Plate 14h) (Ila'ava 1997).

Possible confusion with other symptoms

Boron deficiency may also cause death of the shoot tip. However, necrosis on expanding leaves is uncommon in B deficiency; rather the young leaves become thickened, puckered and brittle, and are often chlorotic.

Diagnostic soil and plant tissue tests

A critical Ca concentration of 0.76% of dry weight in the 7th to 9th youngest leaf blades was estimated in solution culture studies using cv. Wanmun (Table 3). In healthy plants, concentrations were generally between 0.9 and 1.2% Ca. Calcium concentration is dependent on the

actual age of the leaves sampled, as Ca accumulates in the leaf over time. A caution is therefore given against interpreting the concentration in leaf samples from other parts of the vine than that for which the critical concentration is specified. Other factors such as temperature may influence the actual age of the leaves selected, adding to the difficulty of interpreting leaf analyses for Ca status.

Attempts to measure plant-available Ca in the soil are generally unreliable, since the availability is dependent on a number of factors. Exchangeable Ca levels less than 0.2 cmol(+)/kg soil indicate that Ca deficiency is likely (Landon 1991). Very high availability of K (through heavy K fertilization) or Na (sodic soils) may cause Ca to be deficient to plants even though the exchangeable Ca levels may be in what would normally be considered an adequate range.

Plate 14. Calcium deficiency

a) Necrosis on the lateral margins of young leaves is an early sign of Ca deficiency in sweet potato grown on a granitic sand in northern Australia. (Holes in the leaf blades are insect damage).

b) Necrosis spreading from the lateral margins of a young leaf of cv. Hawaii. Older leaves have been shed.

c) Young leaves of cv. Wanmun, showing the development of necrosis on expanding leaves below the tip.

d) Severe Ca deficiency resulting in death of the growing point in cv. Wanmun. Note retention of dead leaves on the stem.

Plate 14. Cont'd

e) Abscission of young necrotic leaves exposing a bare stem in cv. Hawaii.

f) Older leaves of cv. Wanmun, with necrotic spots clustered along the main veins on some leaves, and evenly distributed across the leaf blade on others.

g) An older leaf of cv. Markham, with necrotic spots spread across the leaf blade.

h) Rooting of sweet potato cv. Hawaii tip cuttings, in nutrient solutions containing, from left to right, 4, 13, 41, 144, 397 and 1328 µM Ca. (Photo: V.P. Ila'ava)

Correction of calcium deficiency

Addition of lime (40% Ca) has the dual effect of providing Ca and raising the soil pH. Lime is poorly soluble, and consequently has low mobility in the soil, so it should be broadcast and incorporated thoroughly into the soil before planting the crop. If Ca deficiency is not associated with soil acidity, gypsum (22% Ca) may be used. It is usually also incorporated into the soil, although it is more soluble than lime. Single and triple superphosphate also contain Ca (23% and 16% Ca respectively) and may be suitable sources where both P and Ca are in short supply.

Applications of Ca (particularly as gypsum) may intensify deficiencies of Mg or K. It may be necessary to add these nutrients in conjunction with Ca, so that a suitable cation balance is maintained.

Iron deficiency

Iron (Fe) deficiency is frequently encountered on calcareous soils. In these soils, the availability of Fe to plants is reduced through precipitation of Fe at high pH, or by high concentrations of P. Under such conditions, Fe deficiency may often be associated with deficiencies of other micronutrients, such as Mn, Cu and Zn, whose availability is also pH-dependent. Heavy applications of lime or P fertilizers can likewise induce Fe deficiency. Iron deficiency may also be expected on sandy soils, organic soils or acid soils having excessively high levels of Mn.

A number of other disorders which impair root function, including toxicities of Mn, Al or Zn, or Ca deficiency, can inhibit Fe uptake and induce symptoms of Fe deficiency in the leaves. In all cases of Fe deficiency, care should be taken to determine the conditions responsible for the disorder.

Iron deficiency is one disorder where visible symptoms are obvious when crop growth is only mildly affected, a feature which probably contributes to the frequent observation of Fe deficiency in sweet potato crops (e.g. Bourke 1983).

Symptoms of iron deficiency

The distinctive symptom of Fe deficiency is chlorosis of the youngest leaves (Plate 15a & b). As the chlorosis is a result of photobleaching, leaves in bright sunlight will be more affected than those in shade. Initially, a yellow interveinal chlorosis develops, which is characterised by a sharply contrasting green network of veins (Plate 15b). However, as the condition becomes more severe, the chlorosis becomes white and the veins may lose their green colour (Plates 15c & d). If young leaves are normally purple, they become pink as a result of Fe deficiency (Plate 15d). While all the leaves of the plant may become affected, youngest leaves generally show the greatest intensity (Plate 15c). However, if Fe supply has been restored, new leaves become bright green and chlorotic leaves may be found below them.

Severely affected leaf blades become necrotic, the necrosis usually spreading from the tip and margins into interveinal zones (Plate 15e). Eventually, the growing points may die, both at the terminal apex and in axillary shoots (Plate 15f). Necrotic tissue on expanded leaf blades is generally light brown and soft, but may be dark and brittle on the shoot tips.

Possible confusion with other symptoms

Boron deficiency may also cause death of growing points and chlorosis of young leaves, but the chlorosis is not distinctly interveinal. In particular, the minor veins are not sharply defined, as is seen in Fe deficiency. Also, thickening and deformation of young leaves is not observed in Fe deficiency.

Plate 15. Iron deficiency

a) Chlorosis of young leaves caused by Fe deficiency on a calcareous soil in New Ireland, PNG. (Photo: R.M. Bourke)

Plate 15. Cont'd

b) Typical interveinal chlorosis caused by Fe deficiency in cv. Markham.

c) Symptoms of Fe deficiency in cv. Lole, varying from a light green interveinal chlorosis on mature leaves to pale yellow or almost white on younger leaves.

d) Iron deficiency in cv. Wanmun causing symptoms varying from yellow interveinal chlorosis to complete bleaching of leaf blades, and necrosis on leaf blades and tip. The youngest leaves, which are normally purple in this cultivar, have become pink due to bleaching of the green pigments.

e) Necrosis on a young leaf blade in cv. Lole.

f) Severe Fe deficiency in cv. Markham, resulting in death of the terminal and axillary growing points.

g) The response to leaf painting with Fe on an Fe-deficient leaf of cv. Hawaii. The right-hand side of the blade was painted with a solution of 1% ammonium ferric sulfate, five days previously.

Iron deficiency may be induced by a number of disorders which adversely affect root function. These include Ca deficiency and toxicities of Mn, Zn or Cu. Correction depends on identifying the primary cause in these cases.

Diagnostic soil and plant tissue tests

A critical concentration of 33 mg Fe/kg in the 7th to 9th youngest leaf blades was estimated in solution culture studies using cv. Wanmun (Table 3). In healthy plants, concentrations were generally between 45 and 500 mg Fe/kg. This is consistent with the findings of Spence and Ahmad (1967) who reported 35 mg Fe/kg dry weight in young shoots of plants which showed mild symptoms of Fe deficiency without growth reduction, and 28 mg Fe/kg in a similar sample from plants showing severe chlorosis.

Due to the large number of factors which affect the availability of soil Fe to plants, soil measurements are not generally reliable for diagnosing Fe deficiency.

Diagnosis can be confirmed by painting a chlorotic leaf with a 1% solution of ammonium ferric sulfate, which will cause regreening after a few days. It is important to clearly label the painted leaf so that it can be identified on later inspection. By painting only one half of the leaf, the other half may be used for comparison of the response (Plate 15g). Including a small amount of an agricultural wetting agent or mild detergent in the solution may aid the uniform wetting of the leaf surface, but it is not essential for sweet potato leaves, which wet relatively easily.

Correction of iron deficiency

On soils of high pH, application of Fe compounds as a fertilizer to the soil is likely to give a poor response, as the added Fe will be precipitated and become unavailable to plants. Treatment of an Fe deficient crop is best achieved by a foliar spray of chelated Fe or 1–2% ammonium ferric sulfate solution. Burying small pieces of scrap iron, such as nails and steel food cans, in the mound at planting, can also be effective in reducing Fe deficiency. Soaking cans in sea water before burying accelerates rusting and has been successful in increasing their effectiveness as an Fe supplement on calcareous atoll soils (Cable 1992).

If Fe deficiency occurs on an acid soil, it may be a secondary effect of another nutritional disorder, such as Mn toxicity or Ca deficiency. In this case, liming may alleviate the Fe deficiency by eliminating the underlying problem.

Boron deficiency

Sweet potato seems to be more susceptible to boron (B) deficiency than many other crops. Boron deficiency has long been recognised as a significant problem of commercial sweet potato crops in the USA, particularly in relation to poor root quality (Willis 1943; Nusbaum 1946; Miller and Nielsen 1970). Cases have been recorded in many other countries, in a range of environments, including highly weathered ultisols in the Highlands of PNG (Bourke 1983), on granitic sands in northern Australia (O'Sullivan et al. 1997b) and on river flats in Malawi (J. Mkumbira, pers. comm. 1995). Boron deficiency is most likely on soils derived from acid igneous rocks and freshwater sediments, which are naturally low in B, or in acid, sandy soils in high rainfall areas, from which B has been leached (Bradford 1966). Dry or cold conditions, which restrict root development and the movement of water from roots to tops, seem to exacerbate B deficiency. Recovery may occur following rain or warmer weather.

Symptoms of boron deficiency

Boron deficiency affects actively growing tissue, both of the shoots and roots. The first sign is usually a thickening of young leaves. The leaves and stem near the shoot tip are brittle and break easily when bent. Other symptoms are variable, depending on the cultivar and growing conditions. Young leaves are usually paler than the older leaves (Plate 16a), but the extent and pattern of chlorosis is variable: it may be uniform (Plates 16a & b), or diffusely interveinal, fading gradually with distance from the main veins (Plates 16d & h). In other cases it may be a less distinct interveinal mottle (Plate 16c). Leaves may become puckered (slightly raised in interveinal zones) and the tip and lateral lobes may curl under (Plates 16b, c & e). Petioles may twist. Internodes may be shortened, producing a compact habit around the apex (Plate 16c). In severe cases, the leaf veins may appear callused or overgrown with rough, whitish or pink tissue (Plate 16d). Deeply lobed cultivars may display reduced development of the lateral lobes (Plate 16d).

At greater severity, B deficiency causes death of the growing points (Plate 16e) The dominant tip is usually the first affected, but the axillary buds also become necrotic.

Fibrous roots of B-deficient sweet potato plants become short, stumpy and highly branched (Plate 16f), producing the coralline structures typical of B deficiency in many plant species.

Storage roots are often short and blunt-ended (Plate 16g), and may split and overgrow, producing cankers and deformities (Plate 16h) (Willis 1943; von Stieglitz and Chippendale 1955). The skin may be rough and may wrinkle towards the ends. The cut root exudes less white sap than normal, and the flesh may be mottled or corky in places (Plate 16i). Orange-fleshed roots are generally paler than normal. The flavour is less sweet, or even bitter. Nusbaum (1946) reported irregular thickening of roots, resulting in dumbbell and spindle shapes. Other disorders reported to be associated with B deficiency are 'internal brown spot' (Nusbaum 1946) and 'sweet potato blister' (Miller and Nielsen 1970). Internal brown spot is characterised by the occurrence of brown necrotic areas in the flesh, irregularly distributed but more prevalent in the cambial zone, close to the surface of the root. Sweet potato blister refers to raised dark spots which develop on the skin of some cultivars following a period of storage, and may be sparsely scattered or numerous, covering a large proportion of the root surface.

Storage root development may be greatly reduced or completely inhibited by severe B deficiency (Pillai et al. 1986)

Possible confusion with other symptoms

Calcium deficiency may also result in the death of shoot and root tips. However, in the case of Ca deficiency, young leaves below the shoot tip are not thickened but develop a necrosis which spreads from the lateral margins.

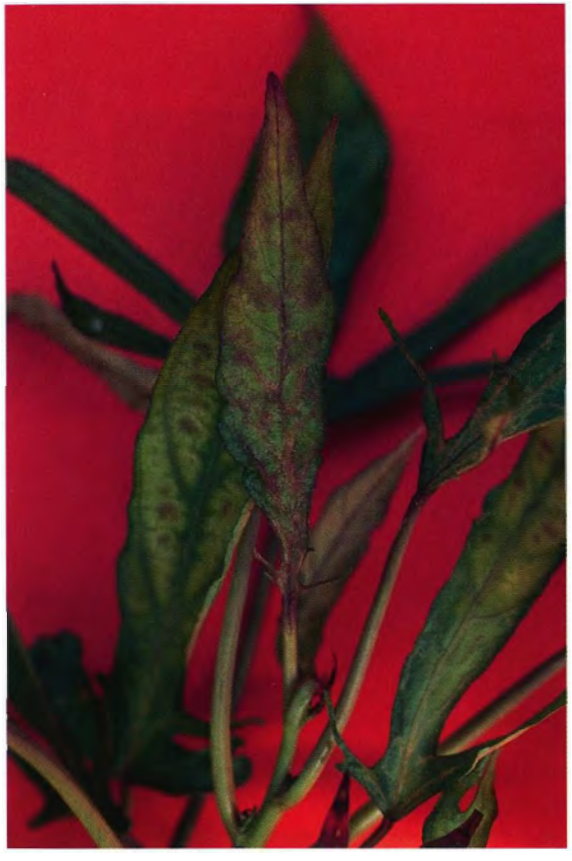
Mites may also cause thickening and deformities on young leaves. They generally do not cause tip death, and may have a more scattered or patchy occurrence in the crop.

Plate 16. Boron deficiency

a) A B-deficient vine of cv. Babache in Malawi, on which young leaves are chlorotic, small and thickened, and the shoot top is dead.

c) Reduced internodes in cv. Hawaii producing a compact growth habit. Leaves are puckered and curled downwards, and have a mottled chlorosis.

b) Puckering and downward curling of the leaf tips and lobes, on young leaves of cv. Wanmun; note the death of the shoot tip.



d) Young leaves of cv. Lole showing interveinal chlorosis, reduced lateral lobes and callus-like growth along leaf veins.

e) Death of the apical and axillary growing points in cv. Wanmun.

f) Short, thickened and highly branched roots of a B-deficient plant.

Plate 16. Cont'd

g) Misshapen storage roots from a B-deficient crop in Fiji, with short, blunt-ended shape and, on the right, an overgrown split. (Photo: A.J. Dowling)

h) Severely deformed roots bearing logitudinal cankers from a B-deficient crop in Kulukulu, Fiji.

i) Symptoms of B deficiency in sweet potato from the Central Highlands of PNG. The vine shows interveinal chlorosis of the young leaves, and the vine tip is dead. The storage root is undersized, has an irregular cross-section resulting from longitudinal cankers, and mottled flesh. (Photo: R.M. Bourke)

Diagnostic soil and plant tissue tests

A critical concentration of 40 mg B/kg in the 7th to 9th youngest leaf blades was determined in solution culture studies using cv. Wanmun (Table 3). This seems to coincide well with field observations on a wide range of cultivars.

With regard to soil analyses, the relationship between hot water extractable B and B deficiency is modified by a number of factors including soil texture, pH and available Ca. There is no published information on soil B in relation to B deficiency in sweet potato. Critical concentrations quoted in the literature for a wide range of crops mostly lie in the range 0.3–0.5 mg/kg hot water extractable B, but may be up to 1 mg/kg for susceptible species such as beet, and may be higher in alkaline soils than in acid soils (Bradford 1966).

Correction of boron deficiency

Boron deficiency can be corrected by fertilising with borax or other borates (e.g. 'Solubor') applied to the soil before planting. Recommended rates are 1–1.5 kg B/ha on sandy, acid soils, or up to 4 kg B/ha on clayey, alkaline soils (von Stieglitz and Chippendale 1955). Overfertilization may result in B toxicity, so it is best to aim for the lowest effective rate, which may be further reduced on subsequent crops.

Foliar application of B is often recommended for other crops, but appears to give a poor response in sweet potato. Boron is not transported within the plant from vines to roots, and while the tops may appear healthy after foliar spraying, symptoms persist on the storage roots.

Zinc deficiency

Zinc (Zn) deficiency may occur on acidic soils low in Zn, or on alkaline soils in which the solubility of Zn is reduced. Due to the declining availability of Zn with increasing soil pH, applying lime or dolomite to acid soils low in Zn may induce Zn deficiency. Applications of Cu fertilizer, or large amounts of P fertilizer, may also exacerbate Zn deficiency (Olsen 1972).

Crops vary considerably in their sensitivity to Zn deficiency. Sweet potato appears to be of moderate sensitivity, and is less susceptible than, for example, cassava or citrus. These species may provide good indicators, if present in the vicinity of a sweet potato crop suspected of Zn deficiency, as they develop clear and distinctive symptoms in situations where symptoms on sweet potato are mild or not evident.

Sweet potato seems to suffer most severely from Zn deficiency at early stages of establishment, and in cool weather. It is common for the crop to 'grow out of it', or to recover as the season warms. However, the effect on final yield of an early Zn stress has not been determined.

Symptoms of zinc deficiency

The most distinctive symptom of Zn deficiency in sweet potato is a reduction in the size of young leaves (Plates 17a & b). The leaves are thickened but usually not distorted, and may be as small as 1–3 cm in length. After the onset of this symptom, plant growth is severely limited. In some cultivars, internodes are also shortened (Plates 17b & c), but in others this occurs to a far lesser extent than the reduction in leaf size (Plates 17d & e). General chlorosis of the young leaves is usual, but may vary from mild to almost complete bleaching (Plate 17c). Increased purple pigmentation of the shoot tips may occur in some cultivars (Plate 17e). Characteristic changes in leaf shape are narrowing of the blade, and repositioning of the lateral lobes (if present) to point towards the leaf tip more acutely than normal (Plate 17f).

Mature leaves develop an interveinal chlorosis in which a diffuse green margin flanks the major veins, and usually the minor veins to a lesser extent (Plate 17g). Interveinal chlorosis on mature leaves is often the first sign of Zn deficiency, preceding obvious symptoms in the young leaves. However, in other cases it may not appear at all.

Pillai et al. (1986) observed that the tubers of Zn-deficient sweet potato plants were of normal shape and size, but displayed a brown discoloration of the flesh.

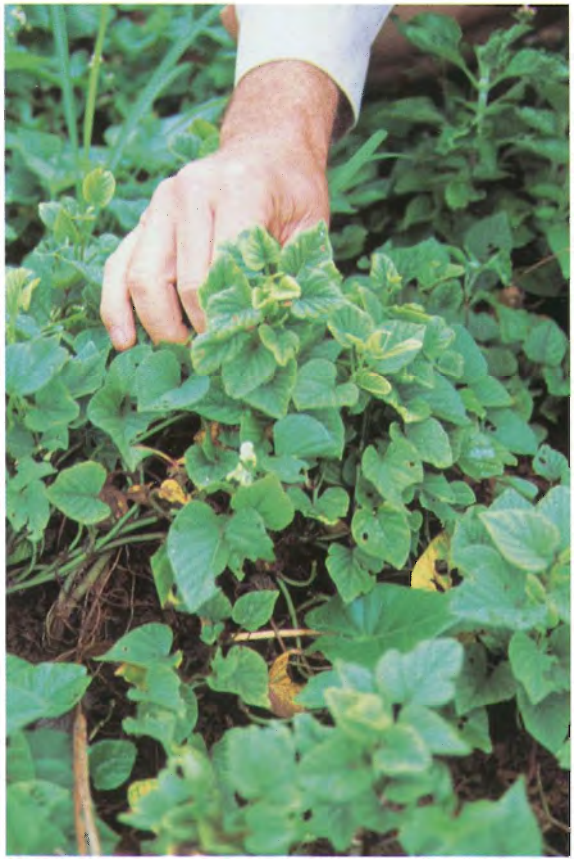
Possible confusion with other symptoms

The appearance of a fine interveinal mottle on mature leaves may be mistaken for early signs of K deficiency. On K-deficient plants, however, the oldest leaves are usually the most affected, and leaves do not respond to painting with zinc sulfate.

Some cases of Zn deficiency may be mistaken for Fe deficiency. However a number of distinguishing features will be evident. With Zn deficiency, chlorosis on older leaves is more diffuse, while in Fe deficiency, the veins contrast sharply with the interveinal tissue. In young leaves, the change in leaf shape and thickening of the leaf blade are not observed in Fe deficiency, and necrosis of young leaves or the shoot tip is not typical of Zn deficiency. Again, leaf painting may be used to separate the two deficiencies.

Little-leaf, or witches' broom, is a disease of sweet potato caused by a mycoplasma-like organism. Affected plants have small, thickened and sometimes chlorotic young leaves which may be mistaken for Zn deficiency. As it rarely affects all the plants in a plot, the observation of healthy plants adjacent to severely stunted, small-leaved plants indicates little-leaf, while Zn deficiency tends to affect adjacent plants similarly. Little-leaf also induces proliferation of side shoots from leaf axils, and latex is absent from cut stems or roots (Clark and Moyer 1988). Symptoms of little-leaf often appear or intensify following a dry period.

Plate 17. Zinc deficiency



a) A Zn-deficient crop of cv. Foketi in Tonga. The young leaves are much reduced in size and show an interveinal to general chlorosis.

b) Extreme size reduction of young leaves caused by Zn deficiency in northern Queensland. Note also shortening of stem internodes.

Plate 17. Cont'd

c) Symptoms of Zn deficiency in cv. Hawaii, showing chlorosis and reduction in size of young leaves, narrowing of the leaf blade and a greater tendency of the lateral lobes to point more acutely towards the leaf tip. Internodes are also shortened below the tip.

d) Severe Zn deficiency in cv. Wanmun, resulting in dramatic size reduction of young leaves with relatively little reduction in internode length.

e) Zn deficiency in young leaves of cv. Lole, showing narrowing of leaf lobes, a more acute angle between lateral lobes and the midvein, purple pigmentation (which is not usual in this cultivar) and reduced leaf size.

f) A comparison in four cultivars of young leaves from Zn-deficient plants (right) with leaves of equivalent age from healthy plants (left): cultivars from the top down are Lole, Hawaii, Markham and Wanmun.

g) Interveinal chlorosis in a mature leaf of cv. Wanmun, caused by Zn deficiency.

Plate 17. Cont'd

h) A young leaf of cv. Siale on a Zn-deficient soil in Tonga, showing regreening and expansion of the right-hand side several days after it was painted with a solution containing zinc sulfate. The leaf held to the left is from a healthy plant. (Photo: S. Halavatau)

Diagnostic soil and plant tissue tests

Suspected cases of Zn deficiency can be confirmed by a positive response to painting the leaf surface with a solution of 0.5% zinc sulfate plus 0.25% calcium hydroxide. Normally, one half of a leaf blade is painted, so the response can be compared directly with the untreated half. After a few days, this should result in regreening of chlorotic tissue on either mature or young leaves, and may increase expansion of the treated area of young leaves (Plate 17h). It is important to label the painted leaf clearly so that it can be identified on later inspection.

A critical leaf concentration of 11 mg Zn/kg was determined in experiments using solution culture (7th to 9th youngest leaf blades at 4 weeks) (Table 3). However, field data suggest that this underestimates the actual critical concentration. A concentration of 17 mg Zn/kg was associated with severe symptoms in young plants growing on granitic sand in northern Australia (O'Sullivan et al. 1997b). These plants responded positively to leaf painting with zinc sulfate solution. Data from other sweet potato crops in the same region suggest that the critical concentration may lie between 20 and 30 mg Zn/kg, and may increase with age of the crop. Leaf analyses from crops in a wide range of situations indicates a normal range of approximately 30–60 mg Zn/kg.

Various extractants have been used to estimate plant-available Zn in soils, including hydrochloric acid, dithizone and DTPA. These tests are influenced to varying degrees by the soil pH, free lime content and phosphate concentration, and reported critical concentrations for Zn deficiency in a range of crops vary widely (1.0–7.5 mg Zn/kg with hydrochloric acid, 0.3–2.3 mg Zn/kg with dithizone) (Landon 1991). An extractant containing 0.01 M EDTA and 1 M ammonium carbonate has been found to be suitable over a range of soils, including alkaline, calcareous soils (Trierweiler and Lindsay 1969). Using this test, a critical concentration of 1.4 mg Zn/kg was determined for Zn deficiency in maize, a crop regarded as being sensitive to low Zn supply.

Correction of zinc deficiency

Foliar spraying is probably the most convenient method of supplying Zn to a Zn-deficient crop, and is particularly recommended on alkaline soils, where soil-applied Zn may have low availability. Rates for sweet potato have not been optimised, but guidance may be taken from the rate used for ginger (0.5% zinc sulfate heptahydrate with 0.25% calcium hydroxide) (Asher and Lee 1975) and cassava (1–2% zinc sulfate heptahydrate solution) (Asher et al. 1980).

On neutral and acid soils where Zn deficiency is known to occur, soil application at or before planting is likely to be more effective than foliar sprays after crop establishment (Weir and Cresswell 1993). Soil application rates of 3–10 kg Zn/ha as zinc sulfate heptahydrate (23% Zn) or zinc oxide (60–80% Zn) are typical for vegetable crops. The lesser amount may suffice on light-textured acidic soils, whereas clayey or alkaline soils may require the higher amount. Zinc oxide should be broadcast and incorporated into the soil before planting. Zinc sulfate heptahydrate is more soluble, and band application at the time of planting is acceptable. Zinc applications may be effective for several years.

Prevention of Zn deficiency during establishment of cassava has been achieved by dipping the cuttings in 2–4% zinc sulfate solution for 15 minutes prior to planting (Asher et al. 1980). A similar strategy may be effective with sweet potato cuttings.

Burying small pieces of scrap galvanised iron in the mound or ridge may provide an effective source of Zn to the crop. This is a particularly useful strategy where Zn fertilizers are not available or are poorly effective due to high alkalinity of the soil.

Maintenance of a high soil organic matter content increases the availability of Zn to plants (Chapman 1966).

Aluminium toxicity

Aluminium (Al) toxicity is the most common cause of growth reduction on strongly acid soils. Aluminium is the most abundant metal in the earth's crust, but its solubility at neutral to alkaline pH is very low. However, below about pH 5.0 (measured in water), Al solubility increases rapidly and may reach toxic concentrations. Solution culture experiments have shown that sweet potato will grow normally at pH values as low as 4.0 in the absence of Al (Ila'ava et al. 1996). Therefore, in the field, sweet potato crops probably encounter problems of Al toxicity before suffering any direct effects of low pH.

Sweet potato is considered to be moderately tolerant of Al. In solution culture experiments, some cultivars showed no reduction in root growth at 25 µM Al, while 50 µM Al caused an average of 40% root growth reduction (Ila'ava et al. 1996). Aluminium-sensitive species may suffer severe root growth reduction at concentrations of 10 µM Al or less (Blamey et al. 1986). Sweet potato was found to be more tolerant of soil acidity than taro, yam, tobacco, maize, green bean or soybean, but less tolerant than cassava (Abruna et al. 1979; Abruna-Rodriguez et al. 1982).

Symptoms of aluminium toxicity

The primary effect of Al toxicity is to reduce root development (Plate 18a). Roots appear short and thickened, with short laterals, and may be discoloured yellow to brown. Root hair development is suppressed. Where the Al concentration increases with soil depth, the downward extension of the roots may be restricted, resulting in a very shallow root system.

In addition to poor growth and stunted appearance (Plate 18b), a number of symptoms may appear in the tops as a result of poor root development, depending on which secondary factor is most limiting. Symptoms of water stress are common. As Al in the soil solution inhibits the uptake of Ca and Mg by roots, symptoms of Ca or Mg deficiency may develop. In addition, the solubility of P is reduced in the presence of high concentrations of Al, so that P deficiency is often associated with Al toxicity (Plate 18c).

Possible confusion with other symptoms

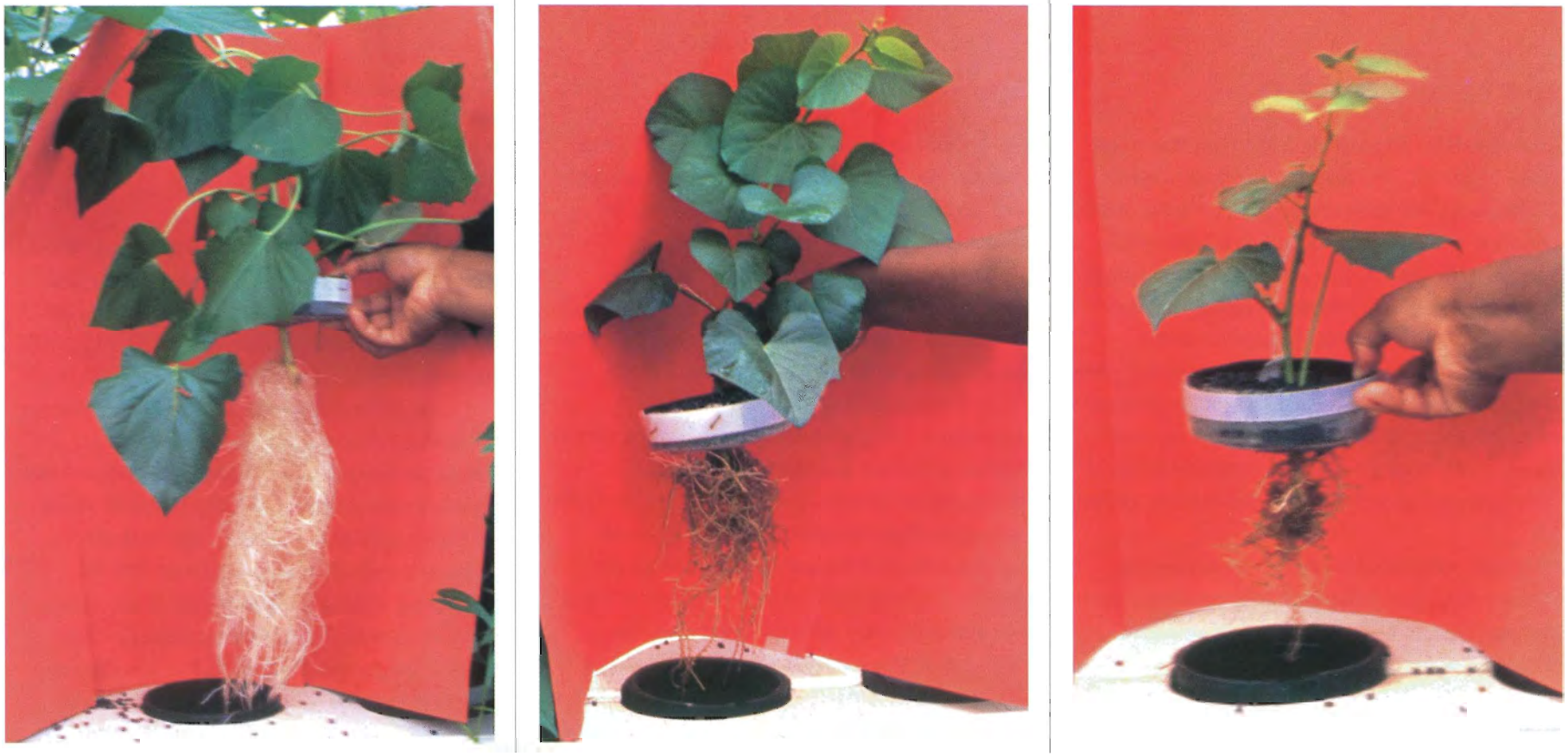
As mentioned above, Al toxicity is often associated with the expression of other nutritional disorders, such as a deficiency of Ca, Mg or P. Carefully uprooting plants may reveal a root system of limited extent, and brown in appearance.

Diagnostic soil and plant tissue tests

Aluminium toxicity is best diagnosed by testing the soil. Soils with a pH below 5.0 (measured in 1:5 soil:water) are at risk of Al toxicity, and in soils below pH 4.5 (other than organic soils), Al is likely to severely reduce sweet potato production.

Tests for exchangeable Al and Al saturation may be useful. Abruna et al. (1979) found that Al saturation correlated well with the response of sweet potato yields to liming on several tropical oxisols and ultisols, with the greatest beneficial effect of lime on those soils with greater than 60% Al saturation. Liming a soil with 35% Al saturation resulted in only a 10% yield increase. Ila'ava et al. (1996) demonstrated that the tolerance of sweet potato to solution Al is greatly increased in the presence of high concentrations of Ca, and it may be necessary to take Ca availability into account when using soil tests to predict crop response to Al.

Plate 18. Aluminium toxicity



a) A comparison of root growth in sweet potato cv. Meriken grown in nutrient solution containing 0 (left), 25 (middle) and 50 (right) µM Al (Photo: V.P. Ila'ava).

b) Severely stunted sweet potato plants on an acid soil in Kabale, Uganda. (Photo: J. Low)

c) Stunting accompanied by symptoms of severe P deficiency on sweet potato grown on an acid soil in Kabale, Uganda. (Photo: J. Low)

Correction of aluminium toxicity

Correction requires elevation of the soil pH, through the incorporation of lime or dolomite into the soil. In addition, maintenance of high soil organic matter levels through the return of crop residues, cover cropping, or mulching will help as components in the organic matter are capable of binding free Al into non-toxic complexes. Organic matter also helps to slow the rate of acidification of the soil, which may result from sequential cropping. Choosing nitrate fertilizers instead of urea or ammonium fertilizers may also reduce acidification.

Phosphate forms insoluble complexes with Al. Some of the beneficial effect of adding P fertilizers to acidic volcanic ash soils appears to result from decreasing solution Al concentrations.

Sweet potato cultivars have been shown to vary in their tolerance to high concentrations of Al (Munn and McCollum 1976; Sangalang and Bouwkamp 1988; Ritchey et al. 1991; Ila'ava et al. 1996), and there is scope for the selection of cultivars which yield well under acid soil conditions. A rapid selection technique, measuring root length of cuttings grown for a few days in a solution containing Al, correlated well with Al tolerance in the field (Ritchey et al. 1991). Sangalang and Bouwkamp (1988), in a study involving 379 sweet potato genotypes, found that Al-tolerant clones yielded less than Al-sensitive ones in nonstressed conditions. In contrast, Ritchey

et al. (1991) found differences in Al tolerance among a selection of high-yielding genotypes, suggesting that it is possible to breed for some degree of tolerance without loss of yield potential.

A number of pathogenic agents, including viruses, fungi and mycoplasma-like organisms, may cause abnormalities on the foliage of sweet potato, which could be confused with symptoms of nutritional disorders. It is not intended in this section to give a full description of all such disorders, but only to help users to determine whether the symptoms they observe are due to a nutritional disorder or a pathogenic agent. Clark and Moyer (1988) have provided a detailed description of pathogenic diseases of sweet potato.

Viruses

Sweet potato feathery mottle virus (SPFMV) is a widespread and serious pathogen of sweet potato, which is transmitted by aphids. Yield losses of 12–25% have been reported due to SPFMV alone, and losses up to 90% have been attributed to co-infection by SPFMV and a whitefly-transmitted virus (Pan 1996). SPFMV typically produces pale green chlorotic spots (2–8 mm) mainly on older leaves, which may be bordered by a zone of purple pigmentation (Plate 19a and b). In some cases, purpling may spread over most of the leaf blade, the chlorotic spots appearing as islands in it. SPFMV may also cause vein clearing, or 'feathering', but a number of other viruses also produce this symptom. Disorders of the storage root including 'russet crack' and 'internal cork' are also induced by some strains of SPFMV in susceptible cultivars (Clark and Moyer 1988; Skoglund and Smit 1994).

While P deficiency in sweet potato may also induce purpling of leaves with cleared chlorotic spots, the two disorders should be easily distinguished. Lesions caused by SPFMV are randomly scattered over the leaf blade, and are not restricted to the oldest leaves on the vine. Symptoms of SPFMV do not progress to cause necrotic lesions, or general chlorosis with the appearance of orange and red leaf colours, as seen in P deficiency.

A number of other viruses of sweet potato have been described, including sweet potato mild mottle virus (SPMMV), sweet potato chlorotic stunt virus (SPCSV) and sweet potato caulimovirus (SPCV), each of which has a limited geographic range (Clark and Moyer 1988). Several are transmitted by the whitefly *Bemisia tabaci*. Symptoms include stunting, mottled chlorosis, vein clearing and leaf distortion.

Mycoplasma-like organisms

Sweet potato little-leaf disease (also known as witches' broom and ishuku-byo) is caused by a mycoplasma-like organism, which invades the vascular system of the plant. Symptoms often become more apparent after a dry spell. It causes a marked reduction in leaf size and a general or mottled chlorosis of the leaves, together with increased branching producing a dense, bushy habit. Cut stems and roots exude no latex sap. It is usually easily recognised, as it will not affect all the plants in a field simultaneously, so that stunted, small-leafed plants appear conspicuously beside healthy plants (Plate 19c). In contrast, disorders such as Zn deficiency, which also cause reduction in leaf size, will tend to affect nearby plants to a similar extent.

Little-leaf disease may be transmitted by several species of leafhopper. It has a long incubation period in sweet potato before symptoms appear, and so is easily transferred in planting material, either in infected vines or in the eggs of infected leafhoppers. It is best controlled by removing and destroying affected plants as they appear, and by choosing planting material from crops with a minimal incidence of the disease.

Fungal diseases

Fungal pathogens such as *Phyllosticta batatas*, *Cercospora bataticola, C. timorensis* and *Alternaria* spp. cause necrotic spots on the leaf blade, which may resemble those caused by toxicities of Mn or B. Fungal lesions may be recognised by their random distribution on the leaf blade (not showing a regular interveinal pattern, although they may be delimited by veins). While lesions may be more common on older leaves, they do not show a consistent decrease in severity from the oldest to younger leaves. Lesions caused by *Alternaria* spp. are characterised by having concentric rings of lighter and darker tissue. Those of *Phyllosticta batatas* may be recognised by the pycnidia, the fungal fruiting bodies, which appear as dark specks near the centre of the lesion (Plate 19d).

Fungal leaf spots do not usually cause significant yield reduction. However, anthracnose or blight, caused by some strains of *Alternaria* spp., can be a serious problem particularly at higher elevations in eastern Africa. In this case the fungus also produces lesions on the stem which can cause death of the vine (Skoglund and Smit 1994).

Fusarium wilt is caused by a vascular infection by *Fusarium oxysporum* f. sp. *batatas*. Symptoms usually appear in young plants which have just started rapid growth. Initially plants develop a dull yellow chlorosis (Plate 19e) which progresses to wilting, shedding of older leaves, stunting and often death. Symptoms may initially resemble N or Mg deficiency, but unlike nutritional disorders, it usually does not affect all plants at the same time. If the stem is cut obliquely, discolouration of the vascular tissue can be seen. Fusarium wilt is best controlled by planting resistant cultivars.

Scab, caused by *Sphaceloma batatas* (also known in its ascigerous form as *Elsinoe batatas*), causes scab-like or scaly patches on the stem and petioles, and on the veins on the lower surface of the leaves (Plate 19f). On young leaves they may restrict the growth of veins, causing puckering and curling of the blade. Eventually the entire infected shoot may appear pale with shrivelled, rough-textured leaves (Plate 19g). In contrast to the leaf deformities caused by sweet potato scab, deformities caused by nutritional disorders such as Cu or B deficiency tend to affect the interveinal tissue more than the veins, resulting in puckering or holes in the blade.

Other symptoms of fungal attack include necrosis on the stems, particularly at the base, causing wilting and death of vines, and rots in the flesh of the storage roots. For more information, see Clark and Moyer, 1988.

Pest damage

A variety of insects feed on sweet potato leaves and signs of their grazing are usually easily recognised. They are generally of little concern, as they rarely affect yield significantly. However, sap feeders, including aphids and mites, can cause more serious damage if infestations are high. They also produce leaf deformities which may resemble nutrient disorders, while the animals themselves may not be conspicuous.

Aphids (*Aphis* spp., particularly *A. gossyppi*) feed predominantly on the young shoots (Plate 19h). Leaves damaged at an early stage in their development may become misshapen, puckered and irregularly curled (Plate 19i). Persistent heavy infestations can result in a stunted crop, displaying general or interveinal chlorosis with yellowing and necrosis on older leaves (Plate 19j). Broadmites (*Polyphagotarsonemus latus*) can also cause leaf distortions (Plate 19k). Tissue along the main veins becomes thickened, and the remaining interveinal tissue reduced producing narrow, shrivelled, downward-curling leaves. In severe cases, the leaves may become brown and scaly. Spider mites (*Tetranychus* spp.) may also cause thickened, shrivelled leaves with a dull, brownish appearance. As symptoms of both aphids and mites tend to be most severe on young leaves, they may resemble those of Cu or B deficiencies. They are generally more irregular and asymmetrical, compared with the nutrient deficiencies.

Aphid and mite populations are usually kept low by predatory insects. Severe infestations are often associated with inappropriate use of insecticides, which remove their natural predators.

Intumescence

Under conditions of high humidity and low light intensity, some sweet potato cultivars have a tendency to produce a proliferation of small calluses (1–2 mm) on the leaf surface (Plate 19l). This condition is called intumescence or tumefaction. The calluses are initially pale green to white, but soon become brown and shrivelled. They may occur on either the upper or lower leaf surface, and are usually scattered over the entire leaf surface but sometimes grow only or predominantly over the major veins. Intumescence is not associated with any pathogenic agent, and has not been reported to impair growth or reduce yield (Clark and Moyer 1988).

Plate 19. Foliar symptoms produced by pathogens

a) Chlorotic spots caused by SPFMV on cv. Puerto Rico, growing in northern Australia.

b) Chlorotic spots bordered by purple pigmentation are typical symptoms of SPFMV in cv. PB growing in central Java, Indonesia.

Plate 19. Cont'd

c) A plant affected by sweet potato little-leaf disease (right), showing severe stunting and general chlorosis, compared with a symptomless plant (left).

d) Necrotic leaf spots caused by *Phyllosticta batatas*. The dark centres seen in some lesions are the fungal fruiting bodies. (Photo: A. Braun)

e) Chlorosis caused by Fusarium wilt in cv. Centennial, in northern Australia. Other plants in the background are of resistant cultivars. (Photo: L. Loader)

f) Scaly lesions on the stem, petioles and veins under leaves of sweet potato in Indonesia. (Photo: A. Braun)

Plate 19. Cont'd

g) A pale, withered shoot infected with sweet potato scab in PNG.

h) Aphids feeding on a shoot tip of cv. Bustak in Indonesia.

i) Puckering and deformities of young leaves caused by aphid feeding. (Photo: A. Braun)

j) A severely stunted crop of cv. Bustak in Indonesia, which has suffered from heavy aphid infestation.

Plate 19. Cont'd

k) Leaf distortions and thickening caused by broadmites on cv. Beerwah Gold.

l) Intumescence on the lower surface of a leaf of cv. Beerwah Gold, grown in humid conditions in a greenhouse.

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Appendix 1. Glossary, Scientific Names, Abbreviations, Conversions, and Symbols

acidic
(of a solution or soil) containing more free hydrogen ions than free hydroxyl ions, a characteristic which affects the behaviour of many of the chemical species contained in it (see *soil pH*)

alkaline
(of a solution or soil) containing fewer free hydrogen ions than free hydroxyl ions, a characteristic which affects the behaviour of many of the chemical species contained in it (see *soil pH*)

anion
a soluble chemical unit (atom or molecule) which carries a net negative electrical charge

anthocyanin
a red pigment sometimes present in plant tissues. In combination with varying levels of green and yellow photosynthetic pigments, it may produce colours from pale pink to bright orange or dark purple

apex
(adj. *apical*) The shoot or root tip

apical meristem
the tissue in a shoot or root tip which gives rise to new leaves or roots

axil
the acute angle formed by the junction of the stem with a leaf petiole, from where lateral shoots arise

axillary bud
the dormant or undeveloped shoot tip in a leaf axil

cation
a soluble chemical unit (atom or molecule) which carries a net positive electrical charge

cation-exchange capacity
the capacity of a soil to loosely bind cations, determined by the surface area and charge density of particles (e.g. clay, organic matter) contained in a given weight of soil

chlorophyll
the green pigment in plant tissue, which transfers light energy into chemical reactions

chlorosis
(adj. *chlorotic*) Loss of green pigmentation, resulting in a paler than normal colour, either light green, yellow or whitish

dolomite
a mixture of calcium carbonate and magnesium carbonate

exchangeable cation
a cation which is loosely bound to a negatively charged site on the surface of a soil particle, and which may be displaced into solution by another cation which takes its place at the particle surface

fertigation
the application of nutrients by adding them to irrigation water

gypsum
naturally occurring calcium sulfate

internode
the length of stem between two nodes

interveinal
relating to those parts of the leaf blade which lie between the veins

lamina
the leaf blade

latex
the milky sap which exudes from cut surfaces of some species of plants, including sweet potato

leaf blade
the broad, flat part of the leaf which provides most of the photosynthetic surface of the plant

lesion
localised injury (usually necrotic) of the plant tissue

lime
naturally occurring calcium carbonate

major / main veins
thick veins on the leaf blade which branch directly from the midvein or from the point of attachment to the petiole, and radiate outwards to the marginal vein

marginal vein
the leaf vein which runs just inside the margin of the blade and parallel to it

midvein / midrib
the main vein bisecting the leaf blade, from the petiole to the tip

minor veins
secondary and tertiary branches of the major veins

mottle
an uneven or blotchy appearance

mycorrhizae
soil fungi which form an intimate association with plant roots, often to mutual benefit

necrosis
(adj. *necrotic*) Death, affecting any section of the plant tissue, not the entire plant

node
the point on a stem where a leaf is attached

petiole
the leaf stalk which supports the leaf blade. Both the petiole and the blade make up the leaf

photosynthesis
(adj. *photosynthetic*) The process by which plants utilise light energy to synthesise organic material

senescence
(v. *senesce*) The processes leading up to death of a plant part, such as a leaf, or the whole plant

soil pH
a measure of the acidity or alkalinity of a soil. Soil pH may be measured by mixing soil in water (e.g. 1:5 soil:water), or in a solution (e.g. 0.002 M $CaCl_2$). Measured in water, a pH of 6.5–7.5 denotes a neutral soil. Acid soils have pH<6.5, with soils having pH<5 being very acid. Alkaline soils have pH>7.5. The presence of undissolved lime or coral may raise the pH up to 8.2; a higher pH indicates the presence of sodium salts

storage roots
the starchy, edible roots of sweet potato, sometimes referred to as tubers

Scientific names for plant species mentioned

barley	*Hordeum vulgare*
beet	*Beta vulgaris*
cassava	*Manihot esculenta*
citrus	*Citrus* spp.
ginger	*Zingiber officinale*
green bean	*Phaseolus vulgaris*
maize	*Zea mays*
peanut	*Arachis hypogaea*
potato	*Solanum tuberosum*
soybean	*Glycine max*
sugar beet	*Beta vulgaris*
sunflower	*Helianthus annuus*
sweet potato	*Ipomoea batatas*
taro	*Colocasia esculenta*
tobacco	*Nicotiana tabacum*
wheat	*Triticum aestivum*
yam	*Dioscorea* spp.

Abbreviations

ACIAR	The Australian Centre for International Agricultural Research
µM	micromolar concentration
CEC	cation-exchange capacity
CIP	International Potato Center
cm	centimetre
cmol(+)	centimoles of positive charge
cv.	cultivar
DTPA	diethylenetriaminepentaacetic acid
EC	electrical conductivity
EDTA	ethylenediaminetetraacetic acid
g	gram
ha	hectare
kg	kilogram
L	litre
m	metre
M	molar concentration = mole/L
me	milliequivalent
mg	milligram
MJ	megajoule = million joules
mm	millimetre
dS	decisiemen
Mt	megatonne = million metric tonnes
PNG	Papua New Guinea
S	Siemen
t	metric tonne = 1,000 kg
TSP	triple superphosphate
VAM	vesicular-arbuscular mycorrhizae

Conversions

Units of measurement used in literature have been converted to standard units for inclusion in this text. Following are the standard units and their synonyms or conversions to other commonly used units.

cmol(+)/kg	=	me/100g
mg/kg	=	ppm = µg/g
dS/m	=	mS/cm = millimhos/cm
1 kg K/ha	=	1.20 kg K_2O/ha
1 kg P/ha	=	2.29 kg P_2O_5/ha

Symbols denoting chemical elements

Throughout most of the text in this booklet, chemical elements are referred to by their chemical symbols. However, the names of chemical compounds are written in full. Following is a list of the elements mentioned and their corresponding symbols.

Symbol	Name
Al	Aluminium
B	Boron
C	Carbon
Ca	Calcium
Cl	Chlorine
Cu	Copper
Fe	Iron
H	Hydrogen
K	Potassium
Mg	Magnesium
Mn	Manganese
Mo	Molybdenum
N	Nitrogen
Na	Sodium
O	Oxygen
P	Phosphorus
S	Sulfur
Zn	Zinc

Appendix 2. Key to Nutrient Disorders

This key is intended as a quick reference to help determine which disorders are the most likely cause of the symptoms observed. It is recommended that the user then refer to the detailed descriptions of each suspected disorder.

This key uses symptoms visible on the above-ground parts of the plant only. Some disorders may be keyed out through a number of routes, depending on which symptoms are expressed most clearly in the crop being investigated. Nutritional disorders may induce a variety of symptoms, which may be expressed to a greater or lesser degree (or not at all) in certain cultivars, or under certain environmental conditions. Hence, a number of symptoms may be described for each disorder. It is not necessary that each of these is observed; the presence of any one, or combination, of the described symptoms may indicate the disorder.

In addition to nutritional disorders, reference is made to a few pathogenic diseases, whose symptoms may be easily confused with those of a nutritional disorder. The key is not intended to give a comprehensive diagnosis of pathogenic disorders.

A **1.** Symptoms are most severe on the young leaves or shoot tip. ..... **B**

2. Symptoms affect older leaves, or the whole plant equally. ..... **G**

B **1.** Shoot tips are dead. ..... **C**

2. Shoot tips are not dead. ..... **D**

C **1.** Young leaves have conspicuous interveinal chlorosis, with green major and minor veins contrasting sharply with the yellow to whitish interveinal tissue; severely affected leaves may be entirely chlorotic or have brown patches; leaves are of normal shape and texture. ..... ***Iron deficiency*** p.75

2. Young leaves are thickened and brittle, often puckered or chlorotic; leaf tip and lateral lobes may be curled under. ..... ***Boron deficiency*** p.79

3. Young expanding leaves may have necrosis on the lateral margins; leaves immediately below may be dead or abscised; older leaves may have circular chlorotic or necrotic spots. ..***Calcium deficiency*** p.72

D **1.** Young leaves are paler than older leaves. ..... **E**

2. Young leaves are not chlorotic. ..... **F**

E **1.** Young leaves have conspicuous interveinal chlorosis, with green major and minor veins contrasting sharply with the yellow to whitish interveinal tissue; severely affected leaves may be entirely chlorotic or have brown patches; leaves are of normal shape and texture. ..... ***Iron deficiency*** p.75

1.1 Necrotic spots are present, particularly on older leaves. Iron deficiency symptoms may be induced by ... ***Manganese toxicity*** p.37

1.2 Red or purple pigmentation is present, on older leaves or leaves of all ages. Iron deficiency symptoms may be induced by ..... ***Zinc toxicity*** p.65

2. Young leaves show general chlorosis, or clearing of small interveinal islands which may form necrotic pits. Older leaves show a diffuse interveinal chlorosis, with darkest green near main veins and colour fading gradually into interveinal zones; these leaves may be puckered or drooping. ..... ***Manganese deficiency*** p.54

3. Chlorosis is general on young leaves, but distinctly interveinal on mature leaves. Young leaves are very small, elongated, and may be curled but not puckered. ..... ***Zinc deficiency*** p.83

4. Chlorosis on young leaves may be diffusely interveinal or general; young leaves are thickened and brittle, and may be puckered. Older leaves are not chlorotic. ..... ***Boron deficiency*** p.79

F **1.** Young leaves may be puckered, deformed, or very small, but not thickened and without interveinal pitting. The leaf blade may have holes, especially near the midrib, and the young leaves may have a silvery appearance, compared with the mature leaves.

1.1 The symptoms affect adjacent plants similarly, although they may be more severe in one part of the field than another. ..... ***Copper deficiency*** p.58

1.2 The symptoms affect some plants more than others. There may be healthy plants adjacent to severely affected plants. Veins may be paler than the rest of the leaf. ..... ***Virus disease***

2. Young leaves are small, and may be curled but rarely puckered. Upper internodes may be very short, but not in all cultivars.

2.1 Branching is increased giving a bushy appearance. Some plants may be much more affected than others, even adjacent plants. The symptom may appear more severe after dry weather. ..... ***Little- leaf disease*** p.91

2.2 Branching is not excessive. Adjacent plants are similarly affected. Young leaf shape is elongated, with lateral lobes reduced in extent and pointing forward. ..... ***Zinc deficiency*** p.83

3. Young leaves are thickened and brittle, may be puckered and have tips and lateral lobes curled under. May have corky growth of tissue over the veins. Upper internodes usually shorter than normal; the shoot tip and axillary buds may be necrotic. .. ***Boron deficiency*** p.79

4. Young expanding leaves have necrotic lateral margins. Leaves below them may be dead, but older leaves are less affected. Older leaves may have circular brown or russet necrotic spots. ..... ***Calcium deficiency*** p.72

G **1.** The whole plant is pale green, with a generally stunted appearance and small, dull leaves. ..... **H**

2. Chlorosis or necrosis is only or most severe on mature to old leaves. ..... **K**

3. No above-ground symptoms other than poor establishment of the crop, wilting or death of plants at an early stage. ..... **O**

H **1.** Red or purple pigment is present on the young leaves. ..... **I**

2. No red or purple pigmentation is present on the young leaves. ..... **J**

I **1.** Pigment is present on the veins and petioles of young leaves (upper or lower surface); cultivars which are normally purple-tipped have purple veins on maturing leaves which are losing general pigmentation. No pigmentation is present on oldest leaves. ..... ***Nitrogen deficiency*** p.45

1.1 The crop does not respond to either nitrogen or sulfur fertilizers. Some mature leaves may have necrotic patches in interveinal tissue or at the margins. ..... ***Molybdenum deficiency*** p.63

2. Pigmentation on the young leaves is more on the margins than veins. Oldest leaves are also pigmented, especially on the margins and tips. On mature leaves, veins may be paler than the interveinal tissue. ..... ***Sulfur deficiency*** p.50

J **1.** Veins are paler than the interveinal tissue; this may be obvious only on mature or older leaves. ..... ***Sulfur deficiency*** p.50

2. The entire leaf blade is uniformly chlorotic. Oldest leaves become mostly or entirely yellow before browning off. ..... ***Nitrogen deficiency*** p.45

2.1 The crop does not respond to either nitrogen or sulfur fertilizers. Some mature leaves may have necrotic patches in interveinal tissue or at the margins. ..... ***Molybdenum deficiency*** p.63

K **1.** Chlorosis or necrosis is not accompanied by discrete necrotic spots. ..... **L**

2. Chlorosis is accompanied by discrete necrotic spots. ..... **M**

L **1.** Young leaves are dark green; yellowing of the oldest leaves may be patchy, asymmetric or interveinal and is accompanied by irregular necrotic patches. Older leaves may have purple pigmentation, becoming red and orange as the leaves senesce. ..... ***Phosphorus deficiency*** p.20

1.1 Poor crop establishment and root growth. Soil is acidic (pH<5.0 measured in water).
Phosphorus deficiency may be induced by ..... ***Aluminium toxicity*** p.88

2. Yellowing of the interveinal sectors between main veins is present on the oldest leaves, and is accompanied by marginal to interveinal necrosis; younger leaves may show light green interveinal chlorosis, with darker green retained around both major and minor veins. ..... ***Potassium deficiency*** p.25

3. Symptoms progress gradually from young mature leaves to oldest leaves. Sectors between main veins become pale green, while the veins are flanked with a zone of greener tissue. On older leaves, the interveinal tissue may become yellow or develop necrotic patches, or the leaf may be entirely yellow and wilted. Red pigment may be present on older leaves. The stem near the tip may be thin and twining. ..... ***Magnesium deficiency*** p.29

3.1 Poor crop establishment and root growth. Soil is acidic (pH<5.0measured in water).
Magnesium deficiency may be induced by ..... ***Aluminium toxicity*** p.88

4. Chlorosis is well developed on mature leaves, but not necessarily the oldest leaves. The chlorosis is diffusely interveinal, mottled or general, and may be accompanied by drooping, puckering or localised necrotic lesions. Youngest leaves may be deformed, puckered or undersized. ..... ***Copper deficiency*** p.58

5. Light green to white chlorosis of small interveinal islands on mature leaves; may affect only a few leaves, and only part of the leaf blade, and does not progress to general yellowing or leaf senescence. ..... ***Copper toxicity*** p.68

6. Leaves have irregularly scattered chlorotic spots (not a regular interveinal distribution), which are roughly circular, and approx. 2–7 mm across. Spots may be surrounded by purple pigmentation. ..... ***Feathery mottle virus*** p.91

7. Oldest leaves become necrotic, initially around the margins but rapidly spreading, causing early death. Necrosis may be preceded by a narrow front of yellowing. Whole crop stunted, and with a tendency to wilt. ..... ***Salinity*** p.40

M **1.** Necrotic lesions are initially small (1–2 mm), dark, and scattered over the interveinal tissue. They may proliferate and merge to form irregular patches of interveinal necrosis. General yellowing of the entire blade occurs on the oldest leaves, immediately preceding senescence. Young leaves may have pale green chlorosis due to induced iron deficiency. ..... ***Manganese toxicity*** p.37

2. Necrotic lesions are most frequent and largest midway between the main veins. ..... **N**

N **1.** Necrosis extends around the leaf margin, and may spread from the initial lesions to engulf large areas of the blade. Necrosis is usually preceded by a front of yellowing. Dead leaves may be retained on the stem. ..... ***Salinity*** p.40

2. Interveinal necrotic spots usually precede the spread of necrosis around the leaf margin. They may expand and fuse, but the surrounding tissue does not become yellow or necrotic until the leaf is about to die. Dead leaves are readily shed. .... ***Boron toxicity*** p.34

O **1.** Saline soil, often irrigated semi-arid land, possibly showing surface crusting or salt deposition around hollows ..... ***Salinity*** p.40

2. Acid soil, having pH <5.0 (measured in water). ..... ***Aluminium toxicity*** p.88