QUEENSLAND DEPARTMENT OF PRIMARY INDUSTRIES

DIVISION OF PLANT INDUSTRY BULLETIN No. 742

A REVIEW OF DIEBACK—A DISORDER OF THE PAPAW (CARICA PAPAYA L.) IN QUEENSLAND

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SUMMARY

Dieback is a disorder of the papaw which every year causes tree losses in southern sub-tropical Queensland. On occasions, epiphytotics arise and tree deaths in plantations may be as high as 100%.

This paper collates available information relating to dieback since it was first reported in 1922. It includes origin and production areas, symptoms and occurrence with respect to plant characters and cultivars, soils, weather and localties.

Possible causes of the disorder are discussed and an attempt is made to establish working hypotheses aimed at investigating these.

It is noted that in wet, tropical north Queensland, where environmental limitations on production are minimal, the occurrence of dieback is rare.

I. INTRODUCTION

The papaw (*Carica papaya* L.) has been commercially cultivated in Queensland for more than 60 years. However, the industry has had a number of problems. In addition to cultural and disease problems, a disorder known as dieback, first recorded in 1922, has from time to time had a devastating effect on plantations. Tree losses as high as 100% have been recorded in epiphytotic situations, and smaller losses up to 10% occur in all years in coastal Queensland between Rockhampton and the southern border.

The cause of the papaw dieback is not known although the problem has been examined by various workers.

This paper collates available information on dieback from 1922 to 1976 and attempts to establish working hypotheses aimed at isolating the cause or causes of the disorder.

A section on the origin of the papaw and areas of production is included as such information appears relevant to the occurrence of dieback and may prove to be the ultimate key to successful cultivation of the papaw and the maintenance of a viable industry in Queensland.

II. ORIGIN AND PRODUCTION AREAS OF THE PAPAW

The cultivated papaw or papaya (*Carica papaya* L.) is a small, dicotyledonous, semi-woody, tropical fruit tree with a very palatable, melon-like fruit which, in the tropics, produces fruit throughout the year. Of the four genera of the *Caricaceae,* three, including *Carica,* are indigenous to tropical America, while the fourth, *Cylicomorpha,* appears to be native to tropical Africa (Storey 1969). Storey notes that it is generally agreed among botanists that *Carica papaya* L. (from here on referred to as the papaw) originated in the lowlands of Central America somewhere in the region between southern Mexico and Nicaragua. This region of Central America lies between approximately 15° and 18° North latitude and has a tropical environment characterized by warmth and high humidity all year round (Anon. 1967).

Today, the papaw is grown extensively throughout tropical and sub-tropical regions of the world, between 32° North latitude and 32° South latitude (Yee *et al.* 1970). Thus many areas of production are well removed from the region of origin, both in terms of distance and climate. However, when the papaw is grown in different regions the performance of the plant varies markedly. This is well reflected by the time from seeding to harvest, namely, 9 months in Hawaii (Yee *et al.* 1970), 24 months in South Africa (Allan 1967), and 11 to 18 months for tropical and sub-tropical regions respectively, in Queensland (Agnew 1968).

There are a number of generalized statements in the literature on cultural requirements and other topics (Agnew 1951, 1958; Awada 1962; Malan 1964; Yee *et al.* 1970), but the environmental limitations on growth and productivity are not well known.

It is an important fact that some 90% of the production of papaws in Queensland is carried on in a sub-tropical climate (Anon. 1972) which is quite different to that in which the papaw originated. While such a situation may be desirable from the view point of market proximity, it is probably much less desirable in terms of plant productivity.

III. SYMPTOMS OF PAPAW DIEBACK

Papaw dieback has been described by a number of workers since the first recorded report in 1922. The descriptions vary slightly, but it is generally acknowledged that all symptoms are not necessarily present on any one tree. Further, in period of severe tree losses (50 to 100% of trees in any one plantation) 'atypical' symptoms occur.

The first symptom is a bunching of the crown leaves due to a shortening of the petioles. The larger crown leaves yellow rapidly making the disorder obvious to the casual observer. The seventh or eighth leaf develops light brown watersoaked areas at the margins which spread rapidly resulting in collapse of one or two leaves. At this stage, a number of symptoms may be present.

One or more of the yellowed crown leaves have small dark green lines (about 1 to 2 mm long) in and just around some of the small veins. The rest of the veins are clear so, when the leaf is viewed from the underside, the lines appear in an X-Y pattern.

Also apparent is a brown staining in the stem phloem tissue just outside the cambial layer. The upper parts of the stem as well as the lower stem and roots are affected first, with the whole stem staining as the disorder progresses. The petioles of the yellowed leaves are also stained especially in the abscission layer.

The growing point usually, but not always, bends towards the seventh or eighth leaf. A small area at the leaf base becomes watersoaked and brown, followed rapidly by death of the growing point.

Grease spotting of the stem, and sometimes the petioles, is associated with dieback-affected plants but the symptom is often evident on apparently healthy plants. Stem grease spots are mostly on the current season's growth and vary in size up to 5 to 6 mm in diameter. In severe cases, numerous spots can coalesce into large patches. The petioles are affected in more severe outbreaks. The grease marks are confined to the lower part of petioles of young leaves and are 2 to 10 mm long by about 1 mm wide.

Later symptoms are blackening of the dead growing point and yellowing of the older leaves which often remain in position for some time forming a fringe around the dead top. Any fruit present either falls off while green or the larger fruit ripen abnormally becoming flabby, wrinkled and finally rotten. Young trees usually die outright but trees bearing their second or third crop sometimes develop healthy side branches.

The time from development of the symptoms to the death of the growing point varies from about 1 week up to 4 or 5 weeks depending upon the time of year and tree growth rate.

Some dieback occurs throughout the year but it is most commonly found during spring in hot dry periods following heavy rain and in the cooler drier late autumn-winter period. Severe outbreaks occur every few years and then often simultaneously throughout a district, but last only a few weeks. Trees growing vigorously or those bearing a heavy load of fruit just starting to ripen seem to be most susceptible, although losses can be severe in young plants just starting to flower.

The above symptoms are quite distinct from symptoms of other known diseases. However, at times of severe dieback outbreaks, the symptoms are not uniform on all trees and may be confounded with other diseases, by inexperienced observers.

IV. OCCURRENCE OF PAPAW DIEBACK WITH RESPECT TO PLANT CHARACTERS AND CULTIVAR, SOILS, WEATHER, AND LOCALITY

Plant characters and cultivar

Dieback affects both young and old trees and those in vigorous as well as in poor condition (Simmonds 1937). A survey in 1973 by M. E. Nicholson and J. D. Glennie (unpublished data) noted that trees 4 months and older were affected. However, fruiting plants sustained greater losses with most trees collapsing just as the first fruit were ready to harvest.

The vigour of the plant may be involved as dieback will stop in a vigorously growing plant after it has travelled approximately 0·5 m down the stem, while at other times it will progress until the plant eventually dies (Simmonds 1965). Kruger (1968) noted that isolated trees may be affected throughout the warmer months, although more serious outbreaks last only a few weeks. These outbreaks usually coincided with an increase in the rate of plant growth. G. J. W. Agnew (unpublished data 1952) stated that sex of the tree had no bearing on the occurrence of dieback. In his breeding lines at Nambour, males, females and hermaphrodites had all been affected. O. W. Sturgess (unpublished data 1953)

also concluded that both male and female trees were equally susceptible to dieback. Similarly, N. S. Kruger (unpublished data 1962) reported that sex and tree height had no effect on the incidence of the disorder.

Numerous papaw strains grown in the Nambour district for breeding work did not indicate that any one strain showed any greater resistance or susceptibility to dieback (G. J. W. Agnew, unpublished data 1952). This was supported by E. P. Williams (unpublished data 1952) and K. King (unpublished data 1952) at Townsville and Yarwun respectively.

O. W. Sturgess (unpublished data 1953) found that the selections 1G, 3W and 1R were less susceptible to dieback in a trial at Aspley (Brisbane) during 1951–53, with 1G being the most promising selection. He attributed this finding to late flowering and in the case of 1G a very poor flowering. However, the 1R selection which flowered at the normal time at Redlands near Brisbane during the 1951–52 epiphytotic had losses as great as 88%. N. S. Kruger (unpublished data 1962) found that the strain S3 was less susceptible to dieback, but did not qualify this finding.

Therefore, both young and old trees in vigorous as well as in poor condition are affected, but both the symptoms and the severity of outbreaks seem to be governed by plant vigour. It appears that different selections may differ in tolerance to dieback because of flowering time. However these differences need further investigation.

Soils

Dieback occurs in different soil types ranging from sandy loams to clay loams (E. P. Williams, unpublished data 1952) and is especially severe where there is a fairly compact subsoil close to the surface, but plantings on gravelly or stony soil are generally free of the disorder (DaCosta 1944). W. Pont (unpublished data 1949) noted the worst incidence of dieback in 1948 was on clay soils, with clay contents usually greater than 40%.

R. L. Prest (unpublished data 1952) observed that the red brown or grey shaley ridges at Brookfield (Brisbane) were well drained and experienced lower losses from dieback and drainage problems than the red and brown sandy clay loams of Aspley (Brisbane) and the Redcliffe Peninsula. However, he added that the Brookfield area is warmer, more frost free and more sheltered from winds than the open country around Aspley and Redcliffe.

Tree losses in the Yarwun, Yeppoon and The Caves areas were more apparent on heavier red-brown loams than granitic sands but, in a bad outbreak in 1947 after exceptionally heavy rain, the incidence of dieback was also high on the Yarwun granitic sands as well as other local soil types (K. King, unpublished data 1952).

B. L. Oxenham (unpublished data 1952) and G. W. J. Agnew (unpublished data 1952) both reported that in the severe outbreak at Nambour in 1951–52 there was no difference due to soil types.

More recently M. E. Nicholson (unpublished data 1973) observed that dieback was worst on red volcanic soils of a high cation exchange capacity. He also considered that soils with bad dieback in average years have the worst dieback during an epiphytotic.

J. H. Simmonds (unpublished data 1950) found in trials that areas in the field with the most severe dieback had the highest soil moisture, the highest field capacities and the highest average soil moisture below 150 mm deep. The worst outbreaks also coincided with periods of highest soil moisture.

Dieback will occur on comparatively light soils where irrigation has been carried out at irregular intervals and may be associated with excessive alternating of the soil moisture content (E. P. Williams, unpublished data 1952).

In summary, soil type influences the occurrence of dieback in 'normal' years but in epiphytotic situations soil type exerts a smaller and perhaps only marginal influence suggesting that another variable, probably weather, is dominant.

Weather

Throughout the years, weather has been mentioned as a factor involved in the occurrence of dieback. Simmonds (1937) was the first to associate dieback with hot drying weather and excessive plant water loss. He also noted that partial root rots following excessive rain gave rise to typical dieback symptoms by producing artificial drought conditions.

Other workers have mentioned dieback following prolonged droughts, heavy rain during which soil aeration is poor, and hot dry periods following heavy rain (DaCosta 1944; K. King, unpublished data 1952; B. L. Oxenham, unpublished data 1952; G. W. J. Agnew, unpublished data 1952; E. P. Williams, unpublished data 1952; M. E. Nicholson, unpublished data 1973).

In all of these reports, a common denominator is excessive rainfall preceding dieback outbreaks. Probably of equal importance but not seen by all workers to be associated is a hot, dry, follow-up period after excessive rain. Such conditions are common to sub-tropical areas of Queensland during the spring and autumn months, when dieback is invariably more prevalent. We tender the hypothesis that if root damage is occurring as a result of excess rain, a hot dry period following will accelerate water loss from the plant at a time when the roots, due to their damaged state, cannot meet the demand for water and probably other nutrients.

Localities

Dieback occurs in all parts of south-eastern Queensland, but its severity varies markedly, not only between districts but also from farm to farm within an affected district. Some trees are lost throughout the year but most losses occur simultaneously over large areas and are apparently due to weather conditions being unusually favourable to the development of dieback (DaCosta 1944). A statistical analysis showed that the incidence of dieback in one plantation at Aspley (Brisbane) was not random but tended to be made up of clusters of affected plants (Anon., unpublished data 1949). Observations by the authors support this finding.

M. G. Hawson (personal communication 1973) has observed dieback in Carnarvon (Western Australia), but the incidence is generally low in these irrigated plantations. More recently R. E. Barke (personal communication 1974) has reported that dieback occurs in Malawi, Africa, which has a dry tropical climate not unlike that of Townsville.

The locality where dieback is rare is wet tropical north Queensland, an area in the southern hemisphere which corresponds in latitude to that in the northern hemisphere where the papaw originated. This area is characterized by

warmth and high humidity, with less temperature and humidity fluctuations than areas of southern Queensland. Such a climate in the tropics allows the papaw to continue growth almost year round as distinct from southern Queensland areas. This growth pattern is reflected in the time from planting to harvest which is 11 months in north Queensland and 18 months in southern Queensland (Agnew 1968). B. W. Cull (personal communication 1974) pointed out that, in the wet tropics, papaws shed relatively few leaves as compared with those in southern Queensland, probably because winter accelerates senescence of older leaves due to lower temperatures, cool winds and drier conditions. Therefore it is postulated that stop-go growth phases and perhaps leaf shed contribute to papaw dieback occurrence in southern Queensland as such factors are less marked in wet tropical north Queensland.

V. SUMMARY OF CURRENT KNOWLEDGE OF DIEBACK

1. Age and vigour of the plants may govern dieback symptoms and the severity of an outbreak. Plants bearing a full load of fruit approaching maturity are more susceptible. Older plants are less likely to die outright. Vigorously growing plants are more likely to succumb to dieback.
2. Occurrence of dieback is independent of tree sex.
3. There is some evidence to suggest that cultivar may have an influence on dieback mediated via the time of flowering.
4. Soil type influences the occurrence of dieback, with lighter, better-drained soils having a lower incidence than heavier or poorly drained soils. However, this difference is marginal in epiphytotics.
5. Weather exerts an influence when excessive rainfall is followed by a hot dry period.
6. Dieback occurs in sub-tropical Queensland and in dry tropical zones. Dieback is rare in wet tropical north Queensland and this is probably related to climatic influences on plant growth and development.

VI. POSSIBLE CAUSES OF PAPAW DIEBACK

Over the years, different workers have postulated various causes of papaw dieback and these can be summarized under the following headings—

(a) A parasitic disease.
(b) A localized growing point calcium deficiency akin to blossom end rot in tomatoes.
(c) Root damage or loss of root functional efficiency.
(d) Atmospheric and soil drought.

A parasitic disease

Simmonds (1937) reported nematodes and a root rot, from which *Pythium ultimum* was isolated, accompanying the dying of tops of papaw plants. The partial root rot followed excessive rain and produced artificial drought conditions which gave rise to typical dieback symptoms. He also observed that, during a dry season when soil moisture was low and especially if the efficiency of the roots was depleted by the presence of nematodes or decay, a period of hot drying weather caused the plant to transpire from its leaf and fruit surface more moisture than the roots could replace. Furthermore, although the symptoms of dieback suggest a parasitic disease of the top of the plant, investigation showed that no

parasitic organism was present and that the death of the crown was apparently caused by some general disturbance in the health of the plant (DaCosta 1944). In addition, it was observed that dieback was often accompanied by a fungous rotting of the roots, but this appeared to be a consequence of the weakening of the root system by adverse conditions rather than a primary cause of root failure.

Trunk and root rots caused by *P. ultimum* express mild or severe symptoms, usually depending on whether the soil is waterlogged or not. If waterlogged, plant death is sudden and accompanied by dramatic wilting of old and young leaves. In milder cases, plants are unthrifty and display loss of vigour.

Phytophthora palmivora causes a more severe root rot resulting in tree losses during unusually wet weather where free water encourages build up and distribution of spores (Simmonds 1965).

More recent work by R. S. Greber (personal communication 1976) has produced no evidence of viruses or mycoplasmas in dieback plants. He did point out that, with a necrotic disease such as dieback, the concentration of organisms away from necrotic areas would be low and, because of the rapid onset of necrosis, isolation of organisms is made difficult. However, there is no difficulty in locating the mycoplasma-like organism causing yellow crinkle in papaw and it is therefore unlikely that a virus or a mycoplasma was missed with electron microscope studies of dieback. Similarly, transmission studies by R. S. Greber and also by N. S. Kruger (personal communication 1974) have been unsuccessful.

In summary, while severe attacks by *Pythium* and *Phytophthora* are easily identified, a mild attack by either organism may act as a predisposing or a contributing cause of dieback, and to date this possibility cannot be discounted. Nematodes may also act in a similar manner by causing root damage.

A localized growing point calcium deficiency

It has been proposed that papaw dieback may be akin to the disorder in tomatoes known as blossom end rot (BER) (Simmonds 1937, Kruger 1968). BER is related to a localized calcium deficiency in the tomato fruit and is influenced by soil water stress and atmospheric conditions.

To relate papaw dieback to the BER condition, it is necessary to postulate that the localized deficiency of calcium is in the growing point of the papaw rather than in the fruit, which is the case with the tomato. Kruger (1968) recorded a reduction in calcium levels in the growing point of the papaw during a rapid growth phase associated with dieback incidence in the field. This reduction in calcium in the growing point could not be made up with calcium sprays. However, the authors have not found consistent reductions in total calcium levels in the growing points of dieback plants compared with healthy plants.

Leopold (1964) suggested that translocation out of the leaf is essentially restricted to the phloem. Therefore with regard to the absence or very reduced movement of calcium in the phloem, it is pertinent to point out that the first discernible factor noted by the authors in dissections of dieback plants is a breakdown and staining in the young phloem tissue near the growing point and in the young roots.

Munoz *et al.* (1966) demonstrated calcium deficiency in the mountain papaw *C. candamarcencis* Hook, f. R. E. Barke (personal communication 1974) has also produced and described symptoms of calcium deficiency in papaws (*C. papaya* L.). However, neither the work by Munoz *et al.* nor the work by

R. E. Barke can confirm that dieback is caused by calcium deficiency. From the description of the former it is difficult to see any similarity between calcium deficiency and dieback.

R. E. Barke (personal communication 1974) examined the effect of a total calcium deficiency on symptom expression with the papaw whereas the basis relating to the BER hypothesis is a localized calcium deficiency in the growing point. His symptoms of calcium deficiency in common with dieback were bunching of the crown leaves, death of the growing point and death of the plant. However, death of the growing point occurs first in dieback plants but in the calcium deficient plants death occurred acropetally. Also, death of the plant was slow with the calcium deficient plants, while it is fast with dieback. Turning of the growing point occurred with calcium deficient plants and this symptom is supported as evidence of dieback by Kruger (1968) and R. S. Greber (personal communication 1974). R. S. Greber also pointed out that the symptoms of calcium deficiency produced by R. E. Barke resemble winter dieback of papaw in the abscission phenomenon associated with petioles and the slower death of the plant. However, recent studies made by the authors showed that boron deficiency can also induce turning of the growing point.

Recent work by Rangnekar (1975) with tomato plants suggests that calcium deprivation restricts photosynthate movement out of the leaf and this contributes to lowered growth activity and eventual death of the meristem. Such a mechanism may operate with the papaw and warrants investigation, particularly in relation to water stress and boron levels, both of which modify translocation of carbohydrates. Also it may be the soluble calcium fraction which is important rather than the total calcium level.

Root damage or loss of root functional efficiency

Simmonds (1937) reported that partial root rot following excessive rain produced artificial drought symptoms which gave rise to typical symptoms of dieback and that a decreased efficiency of roots damaged by nematodes or decay will, in a period of hot dry weather, cause the plant to transpire more water than the roots can immediately replace. Similarly, DaCosta (1944) related dieback to root failure and indicated that drought and inadequate nutrition can cause such failure, but emphasized that the commonest cause is deficient aeration of the soil usually due to poor drainage.

Root damage may be attributed to many factors of which the following may be important—

1. Pathogen, nematode and insect infestations.
2. Waterlogging of the soil.
3. Physical root damage.
4. Soil nutrient deficiency and nutrient availability.
5. Soil nutrient excesses in relation to osmotic pressure and competitive ion effects.
6. Soil water deficit.
7. Above or below optimum soil temperatures.
8. Soil mechanical resistance to root extension and growth.
9. Lack of soil aeration.
10. Suboptimal or supra-optimal soil pH.
11. Soil toxins.
12. Autotoxin production by plants.

Isolation of those factors which contribute to dieback will be difficult. Any contributing factors should be tested in relation to soil calcium supply and localized deficiency of calcium in the plant to prove or disprove the role of calcium in dieback expression. Also treatments should be imposed during a rapid growth phase, taking care to either include or preclude the role of atmospheric and soil water stress.

Atmospheric and soil drought

Simmonds (1937) recorded the relationship between dieback occurrence and dry seasons, low soil moisture, and hot dry weather conditions, while DaCosta (1944) noted that prolonged drought influenced dieback incidence, although no distinction was made between atmospheric and soil drought.

It is an attractive proposition to relate calcium to atmospheric drought conditions because of the calcium stress syndrome which is known for tomatoes and is involved in the explanation of tip burn in lettuce, cabbage and brussel sprouts. However, such an explanation is tenuous by itself, even if proven, since soil drought and root failure may also produce dieback symptoms.

VII. AREAS FOR RESEARCH

The exact nature of papaw dieback is unknown and the causes may be complex. The four areas for research outlined below aim at inducing dieback and are based on the possible causes presented earlier.

1. Research aimed at determining whether dieback is caused by a localized calcium deficiency of the growing point.
2. Examination of the effects of partial root damage and loss of root functional efficiency on the occurrence of dieback.
3. Examination of the effects of soil drought and atmospheric drought on dieback incidence.
4. Investigation of the concept that dieback may be caused by a parasitic organism.

While the causes of dieback may be complex, the end result or effect is consistent, namely the symptoms including the collapse of the apical meristem. This suggests that the primary mechanism by which dieback is brought about in the papaw plant is the same. Simmonds (1937) suggested that the collapse of the soft sappy tissues of the apex and young leaves was caused by their supplementing excessive water loss from older leaves and fruit surfaces due to hot drying weather. Further, the condition was aggravated when the efficiency of the roots was impaired by dry soils, nematodes, decay or partial root rot after excessive rain.

We agree in principle with the possible causes of dieback cited by Simmonds above. However, since the young phloem-cambial tissue in leaves, stems and fruits is the first to show deterioration, it seems likely that this collapse is brought about by carbohydrate shortage and not water starvation as the primary mechanism. Therefore, a fifth research approach based on failure of carbohydrate supply to the growing point is indicated: we suggest that acute boron deficiency may be involved, since boron is known to be intimately involved in sugar transport in plant system.

Breeding and selection for dieback resistance is, at this stage, difficult because of the irregular occurrence and intensity of the disorder. However, field selections should not be ignored. Ultimately the artificial induction of dieback would enable screening of selections to determine whether genetic variation was useful in control.

VIII. CONTROL OF PAPAW DIEBACK

Because the cause of papaw dieback is not known, control measures can be attempted, based only on observations of factors which influence its severity. At times all measures may fail and perhaps losses have to be accepted in southern Queensland until the causes are known.

Control measures for dieback consist essentially of improving cultural conditions and the encouragement of the recovery of affected trees (DaCosta 1944). In selecting sites for papaw plantings, ground known to be badly drained should be avoided, as should sites where a clay subsoil is close to the surface. The physical condition of the soil should be improved by drainage, liming and incorporation of organic matter. Where irrigation is possible, judicious use of water will do much to minimize losses from dieback, but care should be taken not to over-water trees, during the spring months. Affected plants, especially those which are more than 2 years old, often recover from the disorder and produce healthy side branches, and this process may be encouraged by cutting back the trunk immediately the problem is noticed. If small side branches are already present, the trunk should be cut back to a point about 250 mm above them. If no side branches are present, it may be preferable to cut back the trunk 500 mm or so above ground level. If the trunk is cut through at one of the partitions, and a tin placed over the cut end, there will usually be few losses from rotting of the trunks.

Kruger (1968) has suggested that controlling rapid growth phases, particularly in spring, with growth retardants may reduce dieback by reducing calcium dilution in the plant during rapid growth.

If dieback results from a localized calcium deficiency in the growing point of the plant and is akin to blossom end rot in tomatoes, then similar control measures should be effective in reducing incidence (R. E. Barke, personal communication 1974) namely—

1. Avoid over-fertilization, particularly with ammonical and potassic fertilizers.
2. Use lime or dolomite regularly.
3. Increase soil organic matter levels.
4. Avoid the use of acidifying fertilizers.
5. Ensure that sites are well drained.
6. Use irrigation to eliminate soil water deficits.
7. Apply calcium sprays during times of likely outbreak.
8. Screen varieties for dieback resistance.

R. S. Greber (personal communication 1974) considers that root damage is not necessary for dieback to occur and that over-fertilizing and over-watering should be avoided particularly in the spring with vigorous plants growing in exposed windy situations on the outside of plantations. Both R. E. Greber and N. S. Kruger (personal communications 1974) agree that cutting back the trunk is usually successful in regenerating a new fruiting stem on dieback plants. However, the relating of dieback to a rapid growth phase does not explain dieback in winter during a period of slower growth.

Winter dieback could well result from low soil temperatures, lowered soil moisture and exposure to cool drying winds in southern Queensland, so that altering soil and atmospheric moisture conditions and selection of warm protected sites should help reduce incidence with present varieties.

In the warmer weather, raising the atmospheric humidity in the crop should reduce dieback incidence, particularly during periods of excessive plant water loss in hot dry weather after heavy rainfall when root damage and decreased root function have reduced water uptake. Slowing growth rates of the plant under similar conditions as tried by Kruger (1968) may also prove successful.

IX. CONCLUSIONS

Papaw production in Queensland is primarily conducted in the sub-tropical region of the state. This region has the highest incidence of papaw dieback. The sub-tropical climate of this area is quite different from that of Central America where the papaw originated, and the growth, development and production of the papaw is limited by the environment.

Papaw dieback seldom occurs in wet tropical north Queensland where environmental conditions tend to approximate those of the area of origin.

The symptomology of papaw dieback differs in reports by various officers and this is due to the observers, the rapidity of progression of the disorder, plant vigour differences, confounding due to other diseases, time of year and the type of occurrence, namely 'normal' or epiphytotic. However, with experience the disorder can be easily identified in the field since the end result is quite consistent.

Many factors seem to be implicated: age and vigour of plants seem to govern dieback symptoms and the severity of an outbreak; sex of the plant has no influence; the cultivar may have a bearing on dieback incidence, but it is not certain whether this is mediated through an escape mechanism or through genetic factors; soil type influences dieback occurrence with lighter and better drained soils having a lower incidence than heavier or poorly drained soils, however, in epiphytotics soils seems to exert only a marginal effect; weather exerts an effect through excessive rainfall and a hot dry follow up period; locality effects are likely to be made known through plant growth and development and recovery phases. Therefore a vigorous papaw plant growing in a poorly drained or heavy soil subjected to excessive rain followed by hot dry conditions is more likely to succumb to dieback.

The causes of papaw dieback are for the present unknown and it seems that papaw dieback is a complex syndrome rather than one simple problem. Causes of warm weather dieback, winter dieback and epiphytotic outbreaks may differ although the final effect is similar, suggesting that the primary mechanism is the same.

Although at present, control measures remain empirical, areas for research are indicated and should, if conducted carefully, isolate causes and the primary mechanism involved.

Perhaps the papaw industry should be encouraged to develop in wet tropical north Queensland where dieback is apparently non-existent and environmental limitations on growth and productivity are minimal and equate to those where the papaw originated. As an alternative, papaw breeding and selection for sub-tropical conditions may well provide the answer in the long term.

In the interim, site selection and management practices suggested, should minimize the incidence of dieback.

X. ACKNOWLEDGEMENTS

The authors thank all members of the Queensland Department of Primary Industries who have consented to the many statements made as personal communications in this paper, and other Departmental staff, both past and present, who are quoted from internal records and correspondence.

The authors are indebted to Messrs. H. M. Groszmann, N. S. Kruger, B. W. Cull, R. E. Barke and R. S. Greber of the Department and to Professor G. L. Wilson, University of Queensland, who have offered helpful advice and criticism in the preparation of this paper and to Mrs. D. Swan and Miss L. Smith for the typing of the manuscript.

The authors also pay tribute to the many papaw growers in Queensland, who have co-operated with various workers in attempting to resolve this problem.

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(Received for publication 6 October 1975)

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