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SUDDEN DECLINE OF CABBAGE TREE (*CORDYLINE AUSTRALIS*): SEARCH FOR THE CAUSE

Summary: Many cabbage trees (*Cordyline australis*) are dying throughout much of the North Island and the northern South Island of New Zealand. The symptomatology of those dying in urban environments is described, and is concluded to be consistent with the hypothesis that death is caused by a biotic agent entering through a leafy tuft of the branch system. This disease, which has been named Sudden Decline, usually leads to almost total defoliation of affected trees within 2-12 months. Disease incidence has increased linearly at about 11% per annum since 1987/88. Cultivated trees of *C. kaspar, C. obtecta*, and various *Cordyline* hybrids have also been observed dying with Sudden Decline symptoms. Investigations aimed at identifying the causal agent are described, and the hypothesis is advanced that a phytoplasma (mycoplasma-like organism or MLO) is the cause. Sudden Decline is contrasted with the widespread ill-health apparent in many pastoral populations of cabbage tree throughout the country. This Rural Decline is characterised by a general loss of branch and leaf vigour and occasional tree death. It is suggested that Rural Decline is a complex disease (decline disease) caused by various biotic and abiotic agents interacting with an ageing population growing in situations where regeneration is prevented. In many pastoral situations Sudden Decline is superimposed on Rural Decline. The ecological implications of Sudden Decline are discussed.

Keywords: *Cordyline australis*; disease; decline; phytoplasma.

Introduction

Since the late 1980s widespread death of cabbage trees (*Cordyline australis*) throughout much of the North Island has caused concern to the general public, managers and users of parks and reserves, and researchers concerned with indigenous biota. We have been studying the nature of these deaths, in an effort to understand the causes and thus underpin future management and control of the problem (Beever, 1990). A number of other researchers have studied particular aspects, and there has been widespread speculation as to the cause (Rudman and Nixon, 1992; Simpson, 1993a).

In this paper we distinguish two syndromes: Sudden Decline, the death of apparently healthy trees over a period of months (Rees-George, Robertson and Hawthorne, 1990); and Rural Decline, the general loss of branch and leaf vigour and occasional death of trees that is occurring over much of the rural landscape often in the presence of grazing stock (Beever and Forster, 1992). We conclude that Sudden Decline is a biotic disease caused by an as yet unidentified agent, and Rural Decline is a complex disease caused by various biotic and abiotic agents interacting with an ageing population. In many lowland North Island areas Sudden Decline is superimposed on Rural Decline.

Some Definitions

The terms 'disease', 'dieback', and 'decline', are used in different ways. We apply them as follows.

Disease - "Any malfunctioning of host cells and tissues that results from continuous irritation by a pathogenic agent or environmental factor and leads to development of symptoms" (Agrios, 1988). Can be divided into biotic (or infectious) diseases caused by pathogenic organisms, and abiotic (or non-infectious) diseases caused by environmental factors. This definition usually excludes animal (pest) damage, except in the case of nematodes and protozoans, and mechanical injury. We prefer not to use the term 'disorder', which has been variously used to refer to both biotic and abiotic diseases (often when the etiology is not known), and also is used as a synonym for abiotic disease (physiological disorder).

Dieback - "Progressive death of shoots, branches, and roots generally starting at the tip" (apical dieback) (Agrios, 1988). The term is also used to refer to the gradual deterioration of forests (Skelly, 1992), and in the common name of various diseases (Sinclair, Lyon and Johnson, 1987).

Decline - "Progressive loss of vigor and health" (Sinclair *et al*., 1987) resulting from biotic or abiotic diseases or complexes of both acting over a period

of years. The term is also used, as with dieback, to refer to widespread forest deterioration (Skelly, 1992; Manion and Lachance, 1992), and in the common name of various diseases (Sinclair *et al*., 1987).

The concept of forest decline as a distinct syndrome has received intense debate over recent years, but there is no consensus as to its usefulness. Manion and Lachance (1992) argue that "decline concepts are needed for problems associated with an array of interacting biotic and abiotic factors" coupled with a population age component. Specifically with respect to trees, Manion (1991) has championed the use of "decline disease" to refer to harmful deviation from normal functioning caused by the interaction of predisposing, inciting, and contributing factors. The role of these three factors in forest decline (dieback) has been recognised in studies of New Zealand forests for many years (Stewart, 1989).

Sudden Decline of cabbage tree

Rees-George *et al*. (1990) gave the name Sudden Decline to a disease causing rapid death of cabbage trees during the late 1980s around northern New Zealand. The use of the term 'decline' in the name was not intended to imply that the phenomenon in question reflected a 'progressive loss of vigor and health' acting over a period of years or that it was a decline disease *sensu* Manion (1991), although others have subsequently misapplied the name in this manner. Rees-George *et al*. (1990) provided a preliminary description of the symptoms and initial investigations as to possible cause. In this symposium paper we further describe the symptomatology, and outline additional investigations aimed at identifying the cause of the disease. As a tufted woody monocotyledon, *C. australis* has an unusual morphology and anatomy for a tree (Tomlinson and Fisher, 1971), features that should be borne in mind when interpreting the symptomatology.

Symptomatology

Tuft and leaf symptoms

We distinguish the first tuft to show symptoms (the initially affected tuft) from those that show symptoms later (subsequently affected tufts). The first symptoms recognised are the yellowing and subsequent browning from the tip of the lower (older) leaves, followed by premature leaf fall beginning with the oldest leaves (Fig. 1a), on the initially affected tuft. By the time half of the leaves have fallen, other tufts on the tree have usually become affected in a similar manner. The lower leaves of these subsequently affected tufts may become flaccid and droop, before yellowing and then browning from the tip and falling prematurely. The yellowing phase varies, sometimes conspicuously affecting about half of the leaves on the tuft (Fig. 1b), but at other times it is relatively transient. If flowering or fruiting panicles are present, these lose turgidity and become desiccated, but remain attached to the branch tip. Tufts on branches attached closest to the branch subtending the initially affected tuft are affected before more distant ones.

The early stages of leaf yellowing and browning resemble that associated with normal senescence and leaf fall of older (usually 2-year-old) leaves in spring and early summer. Thus the yellowing and browning leaf symptom is not diagnostic of Sudden Decline unless it progresses up to the tuft apex. Many unaffected trees show a general foliar yellowing, possibly associated with nutrient imbalance.

Dissection of the apical region of initially affected tufts before total leaf loss shows browning of some of the vascular strands and limited browning and occasional necrosis of the interstitial tissue (Fig. 1c). On cutting, areas of the interstitial apical tissue sometimes discolour reddish brown. Associated with this apical damage some of the white, unexpanded leaves are necrotic for part of their length, although the most central leaves are usually unaffected. In contrast the apical region of the subsequently affected tufts show no discoloration of any tissue even after most leaves have fallen.

Tufts from healthy trees can be readily made to strike as cuttings. In contrast, subsequently affected tufts of severely affected trees usually die. However, in about 10% of cases, they have been made to strike using a mist propagator within a glasshouse. Thirteen such cuttings have been grown on outdoors in plastic containers for 4-6 years at Mt Albert Research Centre, Auckland. Of these, two died after 5 years with leaf and trunk symptoms resembling those of initially affected tufts. The roots appeared healthy, many retaining the white cortex characteristic of roots less than 1 year old, but the wood of the rhizome was stained pale yellow, similar to the trunk tissue. The other 11 cuttings are still growing normally, although all parent trees are now dead.

Branch and trunk symptoms

The wood tissue of the initially affected branch system, before significant apical necrosis, shows

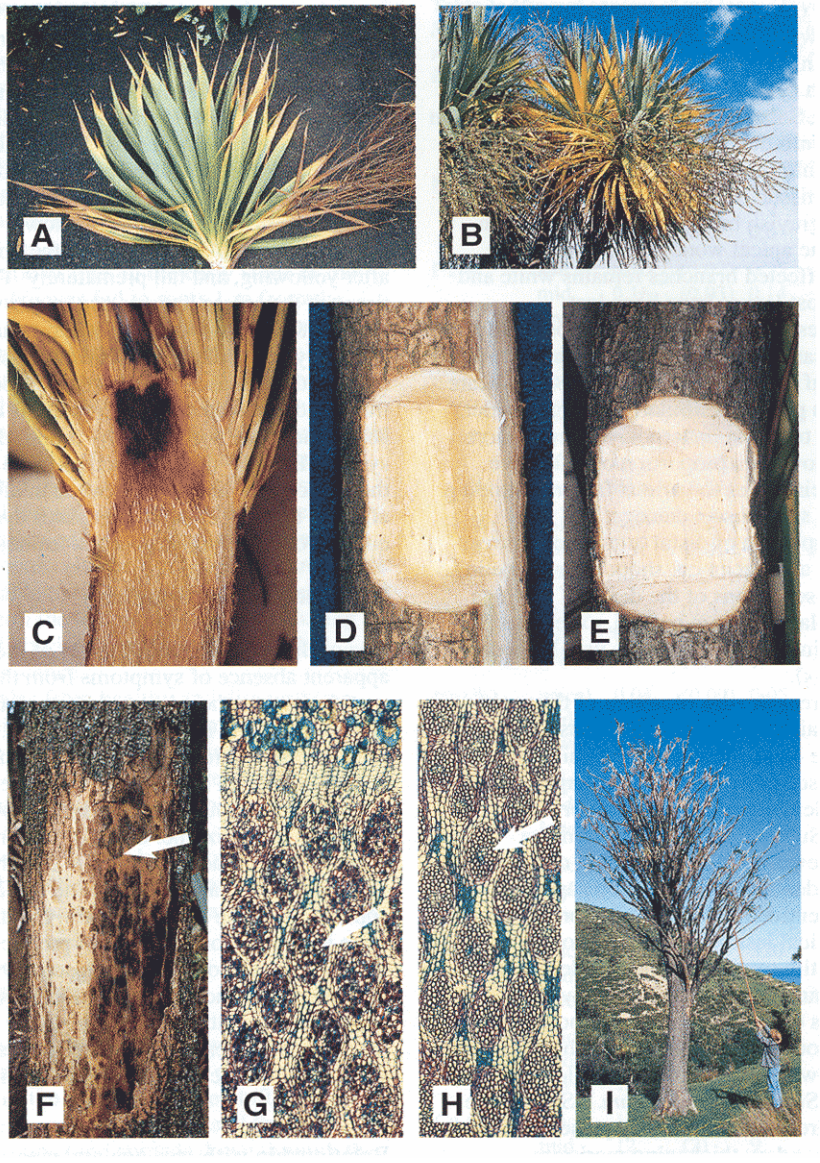

Figure 1: *Symptomatology of Sudden Decline of cabbage tree. (**a**) Initially affected tuft showing yellowing and apical browning of leaves from the tip. Some of the older leaves have fallen prematurely. Other tufts on this tree appeared unaffected (Auckland City, June 1995). (**b**) Subsequently affected tuft showing conspicuous yellowing of leaves. The leaves on the initially affected tuft on this tree had mostly browned by his time (Auckland City, March 1991). (**c**) Initially affected tuft, at a slightly more advanced stage than (a), cut in longitudinal section showing apical necrosis and red-brown discoloration of interstitial tissue (Auckland City, June 1995). (**d**) Branch dissected to show pale yellow to brown discoloration of wood 550 mm below the initially affected tuft in (a) (Auckland City, June 1995). (**e**) Same branch as (d) dissected to show absence of wood discoloration 1500 mm below tuft. (**f**) Trunk lesion of tree at late stage of disease, with bark removed to show progressive browning of cortex tissue centred around abortive root initials (example arrowed). Unaffected cortical tissue is white (Omana, Auckland, October 1990). (**g**) Histological transverse section of wood from tree at middle to late stage of disease (Omana, Auckland, December 1990), showing gummosis (dark staining depositions) within red-stained xylem elements of the vascular strand (arrowed). (**h**) Histological transverse section from nearby unaffected tree (Omana, Auckland, December 1990). Vascular strand (arrowed) containing xylem elements (stained red) surrounding phloem elements (stained blue). (**i**) Tree at advanced stage showing almost total defoliation (Wairarapa, October 1991).*

irregular zones of pale yellow to pale brown discoloration (water soaking) extending down towards the trunk (Fig. 1d), but initially not up into adjacent branch systems. Unaffected wood tissue is white (Fig. 1e). Subsequently the wood discoloration extends down into the trunk, eventually to ground level, and out into the adjacent branch systems. In time the wood tissue of the initially affected tuft becomes pale greyish brown and cracks through desiccation. The apical wood tissue of the subsequently affected branches remains white and turgid, until most leaves have been lost.

The progression of symptoms is most easily observed in relatively small, young trees with up to a dozen or so leaf tufts, but our observations indicate that the pattern proceeds similarly in larger (older) trees. In larger trees the pattern is, however, more difficult to follow, as a proportion of tufts have usually lost their leaves to various factors including drought stress, scale insect damage, and frost damage. A proportion of distal tufts on older trees fail to produce a replacement shoot after flowering. Our limited observations of the death of saplings indicate a similar symptom pattern, except that the leaves are retained on the trunk (as usually occurs in normal saplings).

In larger trees the discoloration of the outer wood of the main trunk initially extends around only a portion of the circumference. Transmission electron microscopy of wood at this stage indicates no obvious xylem or phloem damage or irregularities. Subsequently, more extensive browning and eventually dark staining of the wood occurs. Coincidentally, brown lesions appear in the cortex, often centred on abortive root primordia (Fig. 1f). These cortical lesions eventually coalesce and encircle the entire trunk, from ground level up towards the branches. Light microscopy of the trunk tissue indicates extensive xylem gummosis and occasional phloem necrosis (Fig. 1g, 1h), the phloem necrosis not always associated with xylem gummosis (P. Simpson, *pers. comm.*). Starch-like granules that are characteristically present in the cells of the secondary thickening meristem ('cambium') disappear, and the activity of the meristem is reduced (Fig. 1g). As cortical damage proceeds, the bark over much of the lower trunk loosens from the trunk, sometimes while many of the leaf tufts are still less than half defoliated. At this time and subsequently secondary fungi may infect, and boring insects often become active. Eventually, by the time defoliation is virtually complete, these cortical lesions extend from ground level into the major branches.

In single-trunked trees the period from first symptoms to tree death (almost total defoliation and extensive trunk wood and cortical death, Fig. 1i) ranges from 2 to 12 months (Rees-George *et al.*, 1990). Where the disease occurs in trees which branch into multiple trunks at or near ground level the trunks do not respond synchronously, and individual trunks may die over a period of years. The response of these subsequently affected trunks follows that outlined above, with extensive wood staining and cortical necrosis in the trunk and lower branches. The leaves brown and desiccate from the tip, sometimes after yellowing, and fall prematurely. The internal tissue of the apices remains apparently unaffected, at least up to the time when most leaves have fallen.

Very occasionally diseased trees show a chronic form of the disease, whereby only a sector of the trunk and associated branch system is affected, or sometimes only a terminal portion of the branch system. In these situations a dark zone may form in the trunk at the interface between unaffected and affected tissue. The affected tissues eventually die and dry out, leaving large trunk lesions.

Rhizome and root symptoms

One of the most striking features of the disease is the apparent absence of symptoms from the rhizome, even in trees in the advanced stage of the disease when defoliation is virtually complete, the lower trunk cortex has rotted, and much of the bark has fallen. In Sudden Decline-affected trees that have been cut near ground level, resprouts sometimes arise from the rhizome. Usually these resprouts succumb after reaching 1-2 m in length.

During the early phase of the disease roots appear to be little affected, but in the later phases there is a reduction in new root production and increasing death of roots (Simpson, 1993a).

In one instance apical portions of two rhizomes were taken as cuttings from a severely affected tree that had been excavated. Both regenerated new aerial stems, which are still growing vigorously after 4 years.

Relationship with tree age and size

No correlation between tree age or size and Sudden Decline has been detected, although relatively few saplings have been observed dying before flowering. The youngest tree of precisely known age observed to die was 13.3 years old and 9.5 years from first flowering, but many trees within a few years of first flowering have been affected. The oldest precisely dated tree was at least 61 years old (R.E.F. Matthews, *pers. comm.*) but others probably exceed 100 years. Many vigorous trees estimated to be in the 10-30 age group have succumbed.

Water and mineral relations

The conspicuous browning and desiccation of leaves suggest that the plant may be in severe water stress consistent with root or trunk damage. When secondarily affected leafy tufts from a tree in the mid-stage of the disease were cut and placed in a solution of acid fuchsin the dye was readily translocated through to the leaves and flowers, indicating that the xylem system was functional at this level. In another tree, in the early stage of the disease, when dye was fed to roots 1 m from the trunk base the dye was readily translocated up the trunk. Preliminary measurements of stomatal resistance in green leaves of this affected tree showed little difference from leaves of a nearby healthy tree. Further experimentation is required, but the results suggest that water movement is not severely affected in the early stage of the disease. Yellowing and browning of the leaves - at least in subsequently affected leaves - may reflect vascular blockage, but could also be a physiological response to a translocatable toxin or to altered hormone levels. In general the leaf symptoms of yellowing and active leaf abscission are consistent with premature senescence.

Leaf samples from healthy and diseased trees have been analysed for major and minor nutrients (Table 1). They indicate no major imbalance in affected trees. Chloride levels are relatively high, suggesting that cabbage trees are tolerant of - and indeed may require - high chloride. Zinc levels are high in the lower yellowed leaves of diseased plants, but this probably reflects their advanced senescence; the levels in the younger central leaves do not differ significantly.

Conclusions on symptomatology

These observations are consistent with the hypothesis that an infectious agent enters through one tuft system (the initially affected tuft), causes vascular tissue damage to that tuft and those in close vascular connection, and spreads down through the subtending branch and the trunk, producing wood discoloration. Other more remote branch systems of the tree are not directly affected, but the leaves on these branches are killed, perhaps through toxins produced in the trunk system. The vigorous cuttings that have been grown from subsequently affected tufts of Sudden Decline trees suggest that the infectious agent had not entered these tufts. The two cuttings that died after 5 years may have done so as a result of rare latent infection, or from new independent infections (the cuttings are growing outside). The slow collapse of multiple-trunked trees

Table 1: *Mineral content of tuft leaves from healthy and Sudden Decline-affected cabbage trees. The values represent the mean values (with standard error) for three individual trees.*

		Healthy		Sudden Decline		Adequate[a]
N (%)	apical[b]	1.0	(0.3)	1.5	(0.6)	
	mid	0.9	(0.1)	0.9	(0.1)	1.5
	lower	0.8	(0.3)	0.6	(0.2)	
P (%)	apical	0.20	(0.02)	0.25	(0.05)	
	mid	0.16	(0.03)	0.14	(0.01)	0.2
	lower	0.11	(0.02)	0.08	(0.03)	
K (%)	apical	1.6	(0.2)	1.7	(0.2)	
	mid	1.5	(0.3)	1.1	(0.3)	1.0
	lower	0.6	(0.3)	0.4	(0.2)	
S (%)	apical	0.13	(0.06)	0.14	(0.04)	
	mid	0.10	(0.03)	0.10	(0.02)	0.1
	lower	0.08	(0.03)	0.08	(0.03)	
Ca (%)	apical	0.89	(0.28)	0.60	(0.07)	
	mid	1.15	(0.40)	0.80	(0.20)	0.5
	lower	1.58	(0.57)	1.80	(0.68)	
Mg (%)	apical	0.14	(0.03)	0.12	(0.03)	
	mid	0.11	(0.04)	0.12	(0.02)	0.2
	lower	0.14	(0.07)	0.20	(0.03)	
Na (%)	apical	0.04	(0.02)	0.05	(0.02)	
	mid	0.08	(0.03)	0.08	(0.04)	-
	lower	0.19	(0.13)	0.15	(0.03)	
Cl (%)	apical	0.37	(0.05)	0.44	(0.08)	
	mid	0.54	(0.16)	0.56	(0.12)	0.01
	lower	0.50	(0.21)	0.60	(0.14)	
Fe (μg/g)	apical	27	(9)	21	(8)	
	mid	44	(3)	31	(16)	100
	lower	74	(38)	44	(25)	
Mn (μg/g)	apical	379	(307)	320	(246)	
	mid	359	(307)	400	(298)	50
	lower	523	(417)	797	(605)	
Zn (μg/g)	apical	59	(8)	79	(23)	
	mid	90	(24)	126	(67)	20
	lower	129	(72)	328	(128)	
Cu (μg/g)	apical	17	(5)	12	(2)	
	mid	12	(2)	9	(2)	6
	lower	8	(2)	6	(1)	
B (μg/g)	apical	11	(3)	9	(4)	
	mid	20	(6)	17	(3)	20
	lower	17	(3)	18	(6)	

[a] 'Adequate' levels, based on Table 4.3 of Epstein (1972). They indicate levels considered adequate for 'normal' plant growth.

[b] Samples comprised three fully expanded leaves from each position: near the tuft apex (apical), from mid-tuft (mid), and from the lower part of the tuft (lower). The lower leaves of the healthy plants were still green, those of the affected plants were 50% yellowed.

is interpreted as reflecting the slow spread of the infectious agent via the rhizome system. The browning and necrosis of the trunk cortex tissue may result from toxin action, or from lack of nutrients caused by loss of vascular strand function.

Distribution and epidemiology

A number of different estimates of the extent and geographic distribution of cabbage tree ill-health have been made.

Rees-George *et al.* (1990) describe a survey of at least 50 trees at each of 34 roadside and pastoral sites from Northland to South Canterbury. In this survey, trees with symptoms of Sudden Decline were not found south of Taumarunui. For the 16 northern sites there has been a steady increase in dead trees from 1988 to early 1994, by which time over 60% were dead (Fig. 2). The increase over this period has been linear, averaging about 11% per year, although there was high variability in both the rate of increase and the incidence between individual sites. On a local scale the disease is sporadic, often affecting only one or two trees in a group, and not showing an obvious pattern of tree-to-tree spread. This pattern resembles the jump-spread pattern typical of insect-vectored diseases such as lethal yellowing of palm (Sinclair *et al.*, 1987). The majority of individual trees recorded as dead had

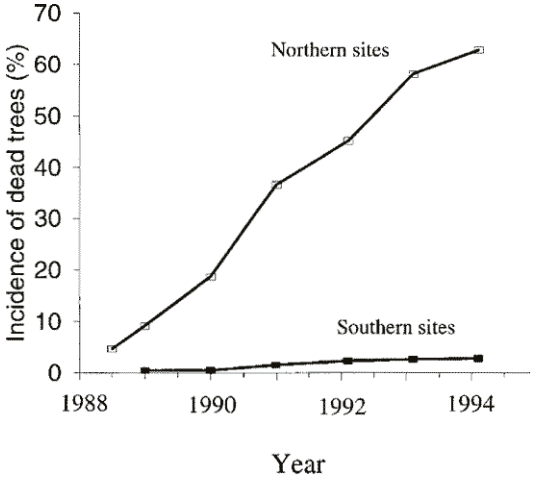

Figure 2: *Disease progress curves for cabbage trees at 16 northern sites (Whangarei to Taumarunui) and 18 southern sites (Palmerston North to Geraldine). Precise localities are provided by Rees-George* et al. *(1990). Values are the mean incidence of dead trees for each group.*

deteriorated from an apparently healthy condition over the 1-year interval between scores, indicating their demise from Sudden Decline. For those plants south of Taumarunui the rate of tree death was low, averaging <1% per year. While the data sets can be satisfactorily fitted by straight lines, such a relationship is not likely to reflect the course of the disease before or after the study interval. On the assumption that the relationship for the northern sites follows a logistic curve, extrapolation backwards would suggest the epidemic began in the early 1980s. Extrapolation forwards suggests that over 75% of the original population will have died within the next few years. This pessimistic assessment must be tempered by pointing out that the survey was based on individuals that were trees in 1987/88. Since that time there has been considerable recruitment of young trees, at least at those sites protected from grazing mammals.

Another survey was undertaken by Hosking and Hutcheson (1992) over the summer 1990-91. They assessed trees at 760 sites under various land uses throughout both the North Island and South Island. At each site they recorded various site and stand characteristics and the presence of disease symptoms on a stand basis, rather than for individual trees. Stands showing what the authors term "typical" symptoms of Sudden Decline were confined to the northern half of the North Island. "Decline was strongly associated with land use, being most common on mature farmland and grazed shrubland, and rare in undisturbed forest and shrubland associations. Symptoms were most severe in old and over mature trees. The most extensive dieback areas were often associated with drastic changes in water table, i.e., where sites had been flooded or drained."

Sudden Decline in the sense that we recognise the disease was probably the major contributor to the disease score in this survey. However, Rural Decline (see below) was undoubtedly a significant contributor in some instances.

A large survey was conducted by R.E. Brockie in 1990-91, by surveying the health of over 5000 trees visible from roads throughout much of the North Island (Brockie, 1995). This survey was repeated in 1994-95 when over 7000 trees were observed. The proportion of sickly and/or dead trees in 1990-91 was highest in Northland (24%); no unhealthy trees were recorded from Tongariro, Wairarapa, and Wellington. By 1994-95 unhealthy trees were present in these three areas, and had increased at most of the other sites, with Northland again being the highest (30%). From Brockie's comments we conclude that many individuals in his sickly and/or dead category are affected by Sudden Decline, but a proportion will reflect Rural Decline.

The mean value of 20% for affected trees in the Northland, Auckland, Waikato, and Rotorua districts in 1994-95 is rather less than the proportion for this general area (northern sites) based on the data of Fig. 2. Brockie's survey will underestimate population death, as trees that died and were removed or fell over the 4-year interval will not have been recorded, and young trees will have been recruited.

These various surveys, and more limited ones such as those of Gannon and Gannon (1992) and Thomson (1991), confirm the widespread ill-health and death of cabbage trees throughout much of the northern half of the North Island. Subsequent to the establishment of the survey of Rees-George *et al.* (1990) we have observed affected trees over a wide altitudinal range, from near sea level to over 450 m (vicinity of Urewera National Park), and also in the southern North Island (Manawatu, Wairarapa, Wellington) and northern South Island (Marlborough, Nelson). We have examined numerous trees elsewhere throughout the South Island, including a number showing signs of ill-health. Trees apparently affected by frost (showing necrotic dead apices) or by fire, and others showing apical dieback due to unknown causes have been examined, but to date (June 1995) we have not confidently identified trees with the symptoms of Sudden Decline south of Nelson/Marlborough.

C. australis is widely cultivated elsewhere in temperate parts of the world. We have observed occasional trees with some of the symptoms of Sudden Decline in Tasmania (1993) and eastern Australia (1990, 1994), but most trees in these areas are unaffected. Correspondence with horticulturists in Europe failed to locate examples of dying trees, apart from those damaged by frost. More evidence is needed if we are to conclude that Sudden Decline is present outside New Zealand.

On the basis of reports to forest health observers (Hosking and Hutcheson, 1992), comments from B. Waller (*pers. comm.*), and our observations there is a strong indication that widespread cabbage tree death began in the Northland region in the early 1980s, a time consistent with the epidemiological data of Fig. 2. Locally in the Auckland area no affected trees were observed at sites on the Waitakere Range coast west of the city in 1990, but affected trees have become common there subsequently. Our observations and those of various informants including Brockie (1995) indicate that the disease has moved from north to south, with reports from the Waikato, East Cape, and Rotorua preceding those from further south.

It has been suggested that there may have been previous episodes of excessive cabbage tree death in Canterbury and Gisborne-Hawkes Bay (Hosking and Hutcheson, 1992), and a number of dying trees were reported in the lower Wairarapa in 1977 (Grehan and Nixon, 1978). It is not clear whether these occurrences involved Sudden Decline.

Host range

There is some controversy as to whether other species of *Cordyline* are affected by Sudden Decline (Hosking and Hutcheson, 1993; Simpson, 1993b). While most of our observations have been made on *Cordyline australis*, we have noted a few trees of *C. kaspar* and one of *C. kaspar x C. australis* cultivated around Auckland City dying with Sudden Decline symptoms. Plants of *C. obtecta* have also been observed dying with similar symptoms, except that the cortex does not show necrosis until a very advanced stage. We have observed plants of *C. banksii* and *C. australis* x *C. banksii* in the Waitakere Ranges near Auckland, and cultivated plants of *C. pumilio,* and *C. pumilio* x *C. australis* in Auckland City, dying with symptoms somewhat resembling those of Sudden Decline. However, more work is needed to firmly establish whether these latter species are indeed affected by Sudden Decline. The Australian species *C. rubra* and C. *stricta*, which are widely cultivated in New Zealand, appear to be unaffected although occasional dying branches have been noted.

The pathogen hypothesis

On the basis of the symptoms described above, we postulate that Sudden Decline is a biotic disease caused by a virulent pathogen capable of rapidly killing trees. As noted, our most detailed observations of symptoms were made on cultivated trees around Auckland, but trees with similar symptoms have been widely observed in both urban and rural areas as far south as Marlborough and Nelson.

We reject explanations implicating causal factors related to tree physiology including aging (affected trees cover a wide age range), vigour (vigorous young to middle-aged trees and poorly growing older trees succumb), and flowering (trees have been observed dying in all seasons and phenological stages). The suggestion that flower removal prevents death (Edwards, 1995) is anecdotal, and other anecdotal evidence suggests that trees die despite regular flower head removal (S.J. Welch, *pers. comm.*).

Likewise we reject explanations based on environmental factors including changing water

table (affected trees occur on well drained volcanic soils, on poorly drained clays and brackish swamplands, usually without any obvious earthworks that would have recently changed the water table), altered climate (disease distribution encompasses a wide climate range), and increased ultraviolet-B radiation (the change in ultraviolet radiation is relatively small, and the symptoms differ from those recorded for other plants (Krupa and Kickert, 1989)). In general the occurrence of healthy trees adjacent to dying ones, and the lack of synchrony of death of multiple-trunked trees argue against a localised environmental cause.

While rejecting these explanations as to the cause of Sudden Decline, both host status and environmental factors must play a role in Sudden Decline. Indeed it is a basic premise of plant pathology that disease is a function of pathogen, host, and environment - the "disease triangle" (Manion, 1991). The challenge is to identify the key factors. The most pertinent observations requiring explanation are: (1) the frequent occurrence of dying trees alongside healthy ones (sometimes the two may be distinct trunks of a presumptive clone); and (2) the restriction of the disease to the North Island and northern South Island.

Formal proof that a pathogen is the cause of a disease requires demonstration of Koch's postulates (Manion, 1991). The first is that the suspected causal agent must be consistently associated with the disease, the second that the agent must be isolated and grown in pure culture, the third that the disease symptoms must be produced when the agent is inoculated into healthy plants, and the fourth that the pathogen must be re-isolated from these plants. While it is straightforward to demonstrate these postulates with many pathogens, in other cases modifications to the postulates have perforce been adopted.

Search for a pathogen

Rees-George *et al*. (1990) describe preliminary investigations as to possible causal agents. In 1990-91 we undertook a broad programme focusing on all known pathogenic groups (Agrios, 1988). Particular attention was paid to agents known to cause lethal diseases of other woody monocotyledons. The symptomatology of the disease is such that we were able to exclude animal pests (insects, mammals, etc.) as primary agents, except for nematodes and protozoa, which are known to produce comparable diseases in plants. The initial aim of this programme was to satisfy Koch's first postulate, i.e., to demonstrate the presence of a pathogen consistently associated with the disease.

1. Nematodes

Plant-parasitic nematodes can be grouped into three ecological classes: stem and petiole nematodes, root and soil nematodes, and virus vector nematodes. Stem and petiole nematodes were looked for because of the resemblance of Sudden Decline of cabbage trees to 'red ring' disease of coconut palms (Griffith, 1987). This sudden tree decline is caused by the nematode *Rhadinaphelenchus cocophilus*, which accumulates in large numbers in a ring of orange-red discoloured tissue beneath the stem surface. Root and soil nematodes can also be associated with tree decline and death. For example, the burrowing nematode genus *Radopholus* attacks banana and citrus trees, causing major crop losses, and New Zealand and Australia have been proposed as the centre of origin for this genus (Sher, 1968). Other plant-pathogenic root and soil nematodes could be implicated in cabbage tree ill-health, but are unlikely to cause rapid tree death. A further group of soil nematodes act as virus vectors, and could be implicated if Sudden Decline is of viral origin.

Twenty-six samples of cabbage tree at various stages of disease, as well as healthy trees were examined over the summer and autumn of 1990-91. Stem-petiole samples were extracted by fibrating stem tissue in water, extracting the tissue by mist, and examining the extraction water by microscopy. Soil-root samples from under healthy and affected trees were extracted by sieving and sugar density centrifugation.

No nematodes nor any other microscopically observable biota were found in the stem-petiole samples. The nematode fauna in all soil-root samples from near healthy and affected trees was similar. The spiral nematodes (*Helicotylenchus* spp.), which are typical of grasslands, were commonest. One sample had high numbers of ring nematode (*Macroposthonia* sp.), another had a few lesion nematodes (*Pratylenchus* sp.). All samples had a background fauna of non-phytoparasitic nematodes. Another sample had a light infection of root knot nematode (*Meloidogyne incognita*) (a young sapling grown at Mt Albert Research Centre, Auckland, with a distorted root system was also found to be infected by this species). No nematodes from groups which are known to act as virus vectors were found in any sample.

We conclude that nematodes are probably not involved in Sudden Decline.

2. Protozoa

Flagellated protozoa are a relatively rare and obscure cause of plant disease (Dollet, 1984). They cause a serious disease of coconuts called hartrot, and of oil palms called marchitez, both in South America.

Symptoms include the sudden wilt and death of the growing point in trees over 2 years old.

Following advice from M. Dollet (*pers. comm.*, 1990), samples were examined from apical regions of 12 affected trees and 4 healthy trees, by squeezing out a drop of sap from plant tissue and examining for elongated mobile organisms (body length 10-20 μm) at x400 magnification.

No biota were observed, and we conclude that protozoa are probably not involved in Sudden Decline.

3. Fungi

Fungi are the major cause of plant diseases. Symptoms of Sudden Decline are at least in broad terms consistent with infection by vascular-wilt and root-rotting fungi, and fungi of other groups (Agrios, 1988). Although previous work (Rees-George *et al.*, 1990) failed to establish a consistent association between any fungal species and Sudden Decline, this work was based on relatively little data, and thus further investigations have been undertaken.

Fungi associated with trunk, branches and leaves

Rees-George *et al.* (1990) reported unsuccessful attempts to isolate pathogenic fungi from branch tips of ailing trees. Isolations were attempted to potato dextrose agar amended with streptomycin (40 mg/l) from a further six trees at Omana, Auckland showing Sudden Decline symptoms. *Cylindrocarpon didymum* was commonly recovered from advanced necrotic zones of the trunk and branches, but is generally considered a saprophyte, and is unlikely to be a primary pathogen. Isolations were also attempted from the slightly yellowish discoloration evident just inside the cortex of trees showing early symptoms of Sudden Decline. Of over 100 explants plated only 3 yielded fungi and none of these was a candidate pathogen.

Two basidiomycetes commonly fruit on dead and dying trees. Fruit bodies of the oyster mushroom *Pleurotus opuntiae*, a species widespread around the world (Segedin, Buchanan and Wilkie, 1995), may appear even while some green leaves are still on the tree. It is generally considered a saprophyte, and is not consistently associated with affected trees. The crust fungus *Phanerochaete cordylines* is an endemic species virtually confined to *Cordyline australis*. Circumstantial evidence suggests that it is capable of killing large branches and occasionally whole trees. Abundant fruiting has been observed on old trees, which show extensive leaf browning and tuft death. Such trees frequently show trunk and branch damage, and may have been compromised. The symptoms of this disease differ from Sudden Decline in that the cortex and outer wood forms a white rot, permeated by fungal mycelium, and the bark does not exfoliate. Only part of the trunk or a major branch system is usually affected, and fresh shoots form from dormant buds below the lesion. This fungus has been observed spreading over and apparently killing dicotyledonous shrubs near to severely affected trees, indicating pathogenic potential. The species is presently known only from Auckland City northwards (records in Herbarium PDD), but may occur to the south. It has seldom been observed or isolated from trees showing Sudden Decline symptoms, although it was sometimes isolated from roots. We conclude that it is a saprophyte and occasional pathogen of cabbage tree, but that it is not the cause of Sudden Decline.

The leaf-spotting fungus *Sphaeropsis cordylines* has been noted in a number of localities, but shows no association with dying trees.

Fungi associated with roots and rhizomes

Fungi associated with roots were assessed over a full annual cycle from November 1990 to October 1991. Samples of roots showing necrosis and sections of outer rhizome tissue from the point of root exit were collected at monthly intervals from three trees showing slight or moderate symptoms of Sudden Decline in the Waimauku district, near Auckland City. Samples were washed, cut into 10 mm lengths, and surface sterilised. Between 20 and 30 root pieces from each of the three trees were plated on cornmeal agar amended with pimaricin (10 mg l^{-1}), and 20-30 pieces on prune agar. The former medium is selective for phycomycetes such as the recognised pathogens *Phytophthora* and *Pythium*. Plates were examined after 5 days' incubation at 22°C, and fungal isolates were transferred to fresh plates of potato dextrose agar for subsequent identification.

Representatives of 33 genera were recovered , together with unidentifiable sterile forms including a number of unknown basidiomycete fungi. No species of *Phytophthora* were recovered from any of the monthly samplings, while *Pythium* was isolated only from two samples, and then in relatively low numbers. The only other candidate fungal pathogens were species of *Fusarium*, some of which are known to cause vascular wilts of a wide range of plants. While *Fusarium* isolates were frequently recovered in each month, for *Fusarium* to be considered as a primary cause of Sudden Decline the fungus should be consistently isolated from the vascular tissue of the rhizome and stem. Isolations made from such tissue of trees with Sudden Decline resulted in only infrequent recovery of *Fusarium* spp.

Chemical treatment, either directly, or indirectly through soil incorporation, is routinely used to control plant diseases caused by soil-borne fungi. If the

primary cause of Sudden Decline is a fungal pathogen, then regular treatment with a suitable chemical should produce an improvement in the health of affected trees. Two chemicals were selected. Phosphorous acid (as Foli-r-fos 400, containing 400 g/l phosphorous acid - U.I.M. Agrochemicals, Australia, Pty Ltd) has specific activity against the root rot pathogens *Phytophthora* and *Pythium*. Carbendazim 75% W.P. (BASF New Zealand, Ltd), with both protectant and eradicant properties, controls a wide range of ascomycetous fungal pathogens.

Twenty-six trees showing early symptoms of Sudden Decline, and six apparently healthy trees, at three sites within 75 km of Auckland City were chosen for study. Six trees with symptoms and the six apparently healthy trees were injected with sterile distilled water. Ten affected trees each were injected with Foli-r-phos 400 (7.5 ml a.i. for each 200 mm of trunk diam., fed by gravity into a hole 5 mm in diam. by 75 mm in depth) and carbendazim (5 g carbendazim 75% W.P. and 20 ml water, inserted into a hole 20 mm in diam. by 100 mm in depth and sealed with a cork). Initial treatments were in September 1990 and, unless a tree was found to be dead, were repeated in December 1990 and March and June 1991. The trees were photographed after first treatment and again in September 1991.

None of the trees exhibited an improvement in health. The degree of deterioration in tree health was similar in both chemical treatments and the 'sick' control group of trees.

In summary, we conclude that fungal pathogens are probably not the cause of Sudden Decline.

4. Culturable bacteria

The symptoms of Sudden Decline, especially the development of chlorosis of leaves progressing to necrosis, and the associated discoloration and subsequent girdling necrosis of sections of trunk cortex, are characteristic of many bacterial diseases. Furthermore, pathogens in the genera *Erwinia, Pseudomonas,* and *Xanthomonas* are associated with apical dieback. Symptoms of a range of so-called fastidious bacteria (Agrios, 1988) also resemble Sudden Decline. These include stunting pathogens in the genus *Clavibacter,* which are associated with xylem-limited activity leading to diseases such as ratoon stunting of sugar cane. *Xylella fastidiosa,* the cause of a slow decline of grape (Pierce's disease) and many other hosts, and fastidious *Spiroplasma* spp. such as *S. citri* or *S. kunkelii*, are also possible candidates. Technically, the fastidious bacteria are difficult to diagnose because they are 'fastidious', requiring complex supporting media for their isolation, and are very slow growing (non-culturable bacteria are discussed in the next section).

A number of media were chosen for their capacity to support known bacterial pathogens. Non-fastidious organisms were isolated to non-selective media (nutrient agar and/or King's medium B agar - Lelliott and Stead, 1987). Complex selective media were chosen which support various groups of fastidious prokaryotes: (a) Gram-positive stunting pathogens in the genus *Clavibacter* (SC medium - Davis *et al.,* 1980a); (b) *Xylella fastidiosa* (PD2 and BYCE media - Wells *et al*., 1981; Davis, Purcell and Thomson, 1980b). SC, PD2, and BYCE were confirmed as supporting media. Medium M1A of Jones *et al*. (1977) was used to look for spiroplasmas following success in growing imported strains of *Spiroplasma citri*, *S. kunkelii*, and *Spiroplasma* spp. (three strains) on this medium.

Twelve trunk specimens with early symptoms characteristic of Sudden Decline were examined between October 1990 and October 1991. Samples were taken from at least three affected zones on trunks, and ten isolations were made on to chosen media from each zone. Initially, isolations were made using a standard bacteriological tissue maceration step. Subsequently, cortical tissue samples were trimmed aseptically into short stakes and crushed, to force the exudation of droplets of plant fluids. These were spread on agar media and incubated at 27° C for 28 days. Most plates of all media remained sterile for the period of incubation. Growth which did occur exhibited the heterogeneous colony patterns of contaminating organisms and not those of any bacterial pathogen.

It is concluded that if a bacterium is the causal pathogen of Sudden Decline, then it was not of a group readily capable of isolation using commonly used general or complex bacterial growth media.

5. Phytoplasmas (Mycoplasma-like organisms or MLOs)

Phytoplasmas (once called mycoplasma-like organisms or MLOs) are a group of bacterial plant pathogens that have not yet been grown in artificial culture (Kirkpatrick, 1989; McCoy *et al*., 1989; Lee and Davis, 1992). They were first recognised as disease agents in 1967, although many of the diseases they cause have been known for much longer. They are grouped with the spiroplasmas in the class Mollicutes which differ from the 'true' bacteria in their lack of a rigid cell wall, the protoplasm being merely enclosed by the plasma membrane, and small genome size. They are confined to the sieve tubes in the phloem of infected plants, and are transmitted by insects (leafhoppers, planthoppers, psyllids) that feed specifically on the phloem. Recent molecular evidence indicates these parasites form a monophyletic group, and the trivial

genus name phytoplasma was proposed (Gunderson *et al*., 1994).

The possibility that a phytoplasma could be the cause of Sudden Decline was suggested early in our investigations, because of the resemblance of the disease to so-called yellows diseases of other plants. Many yellows diseases are now known to be caused by phytoplasmas. The best known in New Zealand is yellow-leaf disease of flax (*Phormium* spp.), a presumably endemic disease once of major concern to the now defunct flax industry (Boyce and Newhook, 1953; Ushiyama, Bullivant and Matthews 1969). The native flax leafhopper *Oliarus atkinsoni* is known to transmit this disease (Cumber, 1953).

Because these organisms cannot be grown in culture, diagnosis of phytoplasma diseases is difficult. Three main techniques have been used: antibiotic treatment; electron microscopy; and techniques based on identifying phytoplasma DNA. Additional evidence may come from transmission studies.

Evidence from antibiotic treatment involves injecting trees with tetracycline antibiotics and observing symptom remission. Preliminary work gave no indication of symptom remission in response to such treatment, although experimental variability, including that of the water controls, was high and the experimental results were not conclusive.

Phytoplasmas: Electron microscopy

Diagnosis of phytoplasma infection by electron microscopy involves finding phytoplasma profiles in the phloem of affected plants. Phytoplasmas appear as circular bodies distinguished by their variable size (50-1000 μm), bounded by a plasma membrane, and often containing densely staining DNA strands.

Standard EM methods were used, involving fixing material in 2.5% glutaraldehyde, post-fixing in 1% osmium tetroxide, dehydrating through an ethanol series and embedding in Spurr's resin. Sections were stained with uranyl acetate followed by lead citrate (Reynolds, 1963).

Samples from 17 trees showing Sudden Decline symptoms have been examined, along with a smaller number from apparently healthy trees. Initial focus was on the vascular system in the leaf bases and the apex of subsequently affected tufts (at the time this work was carried out, the distinction between initially affected and subsequently affected tufts was not recognised). No structural indication of phytoplasmas was detected, nor any other pathogen, including xylem-limited bacteria or viruses. Subsequently, rhizome tissue, root tissue, and trunk tissue from trees in the early stage of the disease were examined, again without detecting the presence of any pathogens. In general the phloem and xylem elements in these samples appeared essentially normal, but occasionally the phloem was collapsed and the xylem contained electron-dense material. The xylem gummosis observed later in disease development (Fig. 1g) resembles that observed in yellow-leaf disease of flax (Newhook, 1953).

The results neither implicate a phytoplasma in Sudden Decline, nor do they exclude it. Detection of phytoplasmas by electron microscopy is often difficult in woody plants, as in the various yellows diseases of grape (Credi, 1994), presumably because of their often sparse and erratic distribution (Kirkpatrick, 1989).

Phytoplasmas: DNA approaches

Modern DNA technology offers highly sensitive and specific approaches to the diagnosis of plant diseases, including phytoplasma diseases. We initially applied DNA dot blot technology for the detection of phytoplasmas using phytoplasma probes supplied by R.E. Davis (Davis *et al*., 1988, 1992), and obtained preliminary evidence for involvement of a phytoplasma (Beever and Forster, 1992; Rudman and Nixon, 1992). After a break in experimentation we were unable to confirm these preliminary findings, and subsequently abandoned the probe approach in favour of the more specific and sensitive PCR technology that had increasingly become the method of choice in the intervening period (Ahrens and Seemüller, 1992; Davis and Lee, 1993). We have used the methodology to devise a highly specific and sensitive detection system for the *Phormium* phytoplasma, causal agent of yellow leaf of *Phormium,* and are applying these methods to Sudden Decline-affected cabbage trees.

Phytoplasmas: transmission experiments

Phytoplasmas can be transmitted artificially by grafting and via the haustorial parasite, dodder. However, our grafts were unsuccessful, and we were unable to produce dodder infections of cabbage trees. Transmission experiments have focused on planthoppers that have been found on cabbage trees in the Auckland urban environment, where Sudden Decline is rampant. This restriction limits the field to two species, both introduced from Australia: the lacy-winged *Scolypopa australis* (passionvine hopper), and the green *Siphanta acuta*. The distribution of *Scolypopa australis* throughout New Zealand closely parallels that of Sudden Decline (Cumber, 1966; Dietz, 1989). *Siphanta acuta* extends further south. The introduced slate-grey *Anzora unicolor (Sephena cinerea*) has been found occasionally on rural roadside cabbage trees, but not yet from urban plants. The experimental design has involved rearing

nymphs of each species on phytoplasma-free host plants, feeding them for 2 or 3 weeks on intact or detached leafy tufts from Sudden Decline trees (acquisition period), and transferring them to seedlings of cabbage tree and flax for up to 6 weeks (incubation plus exposure periods). Controls included nymphs maintained on phytoplasma-free hosts, and another group fed on yellow leaf-affected flax plants for the acquisition period. Experiments with *Scolypopa australis* were conducted over the summer 1993/94, and those with *Siphanta acuta* over the summer 1994/95. As of June 1995, no symptoms indicative of phytoplasma infection have been noted in any of the recipient plants.

In summary, the evidence to date does not unequivocally implicate phytoplasmas in Sudden Decline, but our studies are continuing.

6. Viruses and virus-like agents

Viruses cause a wide range of plant diseases (Matthews, 1991). While many cause plant stunting and mosaic and ringspot symptoms on leaves, others may cause host death. The commonest viruses contain RNA, but some important ones contain DNA. One of the latter causes foliar decay of coconut, a disease resembling Sudden Decline. Viroids differ from viruses in that they are composed of a single strand of RNA, without accompanying protein coat. Cadang-cadang, a viroid disease of coconut, has symptoms resembling those of Sudden Decline (Garnsey and Randles, 1987).

Transmission experiments and electron microscopy

Viruses can be detected by various means including electron microscopy, mechanical transmission to indicator plants, grafting and dodder transmission, and for some soil-transmitted viruses by growing plants in infective soil (Matthews, 1991).

As indicated above, no virus or virus-like symptoms were detected by electron microscopy of various tissues from affected trees. Pollen from three affected trees was examined, but difficulties in fixing the pollen adequately meant that internal structure was poorly preserved, and the results were inconclusive. Mechanical transmission tests to a variety of herbaceous indicator plants using tissues from apices, inflorescences, and pollen, gave no indication of the presence of viruses in affected trees. As indicated above, we were not successful in grafting cabbage trees or inducing dodder infection of leaves. Saplings have been grown in plastic containers of soil collected from under affected trees. While the growth of most has been poor, and one has died, the symptoms of slow growth sometimes associated with leaf distortion are not those of Sudden Decline.

Molecular approaches

Molecular methods offer powerful tools for diagnosing viral diseases. Three approaches have been applied to cabbage trees in a search for viruses correlated with the occurrence of Sudden Decline: (a) to isolate dsRNA, so as to indicate the presence of RNA viruses or infectious RNA species; (b) to isolate aberrant DNA, indicative of DNA virus presence; and (c) to isolate circular single-stranded RNA species, indicative of viroid infection. Viruses and virus-like agents typically become systemically distributed through their hosts. Thus young leaf and apex tissue and young inflorescence tissues were targeted for extraction.

(a) To look for dsRNA, total nucleic acids from foliar and/or floral tissues of six affected cabbage trees were extracted using hot phenol and sodium dodecyl sulphate (SDS), following tissue disruption by freeze-drying and vigorous maceration for 15 min. in liquid nitrogen with abrasives. Nucleic acid species typically found in plants were detected in samples, indicating some success with the extraction methods used. Nucleic acids migrating in a manner typical of dsRNA species were detected in one tree. DNA cloning and partial sequencing of this dsRNA species indicated that it was not of viral origin.

(b) To look for DNA viruses total nucleic acids were extracted from the apical region, leaf blades, and floral tissue of several affected trees with phenol and SDS. The extracts were salt-fractionated and the DNA fraction was centrifuged on CsCl gradients. The gradients were fractionated, and 20 fractions were tested for aberrant DNA by gel electrophoresis. No DNA species were detected.

(c) The method used to check for viroids is based on two unique properties of viroids, the circularity of their RNA and its high stability. Samples are subjected to two-dimensional polyacrylamide gel electrophoresis in different buffer regimes. The method was trialed by successfully detecting potato spindle tuber viroid in tomato leaves. No viroids were detected in extractions from leaf tissue of three affected cabbage trees prepared using phenol/chloroform followed by CF-11 cellulose chromatography.

Our conclusion that Sudden Decline is unlikely to involve a virus or viroid must be tempered by difficulties in obtaining high-quality nucleic acid for study.

Hypothesis as to the nature of the pathogen

Our present working hypothesis is that Sudden Decline is a phytoplasma disease. Support for this hypothesis is as follows.

- Disease symptoms (vivid leaf yellowing, occasional phloem necrosis, xylem gummosis) resemble those of various phytoplasma diseases, including yellow-leaf of *Phormium*.
- Death can be very rapid, as with some phytoplasma diseases such as lethal yellowing of palms.
- The disease has not so far been linked to any of a wide range of other pathogens.
- The rate of disease increase is compatible with other phytoplasma diseases (Sinclair and Griffiths, 1994).
- The sporadic pattern of disease occurrence at the local level resembles the jump-spread pattern typical of insect-vectored diseases such as lethal yellowing of palm (Sinclair *et al*., 1987).
- Planthoppers that could spread a phytoplasma disease occur throughout the range of Sudden Decline.
- Phytoplasma diseases have often proved difficult to diagnose.

As indicated earlier, we were unable to confirm our preliminary dot-blot evidence implicating phytoplasmas. Thus we have not so far satisfied Koch's first postulate, that is the consistent association of a suspected agent with the disease. While the lack of methods to grow phytoplasmas means that no-one has formally demonstrated all of Koch's postulates for any phytoplasma disease, acceptable evidence requires demonstration of the first postulate and preferably transmission of the disease by an appropriate insect.

If the phytoplasma hypothesis is correct, the question as to the origin of the cabbage tree phytoplasma arises. There are a number of possibilities. We favour the suggestion that it may be a variant of the *Phormium* phytoplasma, the pathogen of yellow-leaf disease. A possible scenario is that the cabbage tree phytoplasma arose in the *Phormium* phytoplasma population somewhere in the lower Northland - Auckland area, and has been spread from there by introduced planthoppers.

Rural decline of cabbage tree

We propose the name Rural Decline to refer to the widespread loss of vigour, ill-health, and gradual death of wild cabbage trees that is occurring throughout much of rural New Zealand (Beever and Forster, 1992). Its symptomatology is distinct from that of Sudden Decline, although Sudden Decline is often superimposed on Rural Decline. Following Hosking and Hutcheson (1992), we agree that Rural Decline can be reasonably accounted for by Manion's three-stage concept of decline disease (Manion, 1991). Predisposing factors may include tree age, and soil compaction with poor drainage; inciting factors may include water and nutrient stress and mechanical injury by stock; and contributing factors wood-rotting fungi and microorganisms that provoke saprobic decay and leaf feeding caterpillars. A comparable interaction between a biotic disease (ash yellows, caused by a phytoplasma) and a decline disease (ash decline) has been reported in North America (Sinclair and Griffiths, 1994).

Simpson (1993b) proposes the term "Slow Decline" to refer to the widespread ill-health of both wild and cultivated cabbage trees. His usage (P. Simpson *pers. comm.*) encompasses Rural Decline as defined here, as well as physical damage caused by human activity. We prefer to consider the latter as injury rather than disease.

Rural Decline is characterised by a general loss of tuft and tree vigour over a period of many years. Death of individual tufts is common, but tree death is infrequent, and is often associated with physical damage such as breakage of branches and/or trunk by wind or grazing animals, and entry of water and saprobic decay into the trunk. It is sometimes associated with changes in the water table caused by flooding or drainage. The putatively opportunistic fungal pathogen *Phanerochaete cordylines* may be present on damaged branches and trunk. Rural Decline is characteristic of old trees in pasture and grazed shrubland. Many of these trees probably established at the time the land was cleared for farming, around the turn of the century, and as noted by Esler (1974) are "marching to oblivion".

Ecological significance of Sudden Decline, and cabbage tree conservation

While biotic diseases seldom cause major epidemics in natural or modified systems, there are some relatively well understood examples where they do. These include situations where an exotic pathogen or variant of an existing pathogen is introduced. For example, the introduction of the fungus *Cryphonectria parasitica* probably from China virtually eliminated American chestnut (*Castanea dentata*) from north-eastern North America (Sinclair *et al*., 1987). Closer to home, the apparent introduction of *Phytophthora cinnamomi* resulted in widespread death of a diverse range of woody perennials in indigenous plant communities of Australia (Weste and Marks, 1987; Wills and Keighery, 1994). In other situations, widespread plant death has resulted from an environmental change acting in concert with an existing pathogen. For example, it has been suggested that the death of large numbers of flax (*Phormium tenax*) through much of the North Island beginning early this

century was triggered by drainage of the swampland to encourage flax growth (Boyce and Newhook, 1953). This led to an increased population of the insect vector of the *Phormium* phytoplasma and consequently an increase in yellow leaf disease. In Tasmania human disturbance through logging is associated with an increase in mortality of *Nothfagus cunninghamii* caused by the fungus *Chalara australis* (Kile, Packham and Elliott, 1989). Whether Sudden Decline should be included with these diseases awaits resolution of its cause.

The loss of large cabbage trees in urban areas such as Auckland and Rotorua is of concern in that this plant provides one of the distinctive New Zealand elements in gardens and parks. The disappearance of many trees from the pastoral landscape is likewise affecting the visual values of these areas. However, loss of these trees was inevitable given the lack of regeneration of this species in the presence of grazing stock (Esler, 1974). What is still problematic is the future of trees protected from stock in the rural landscape, and in protected natural areas. Certainly, many trees are dying in some wetland areas (e.g., Dargaville, North Auckland; Lower Kaituna, Bay of Plenty), and Sudden Decline-affected trees have been noted for example in coastal forest communities on Hen Island, and in local reserves around Auckland City. More survey work is needed to assess the impact in these situations.

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