KAMAHI DECLINE IN TONGARIRO NATIONAL PARK

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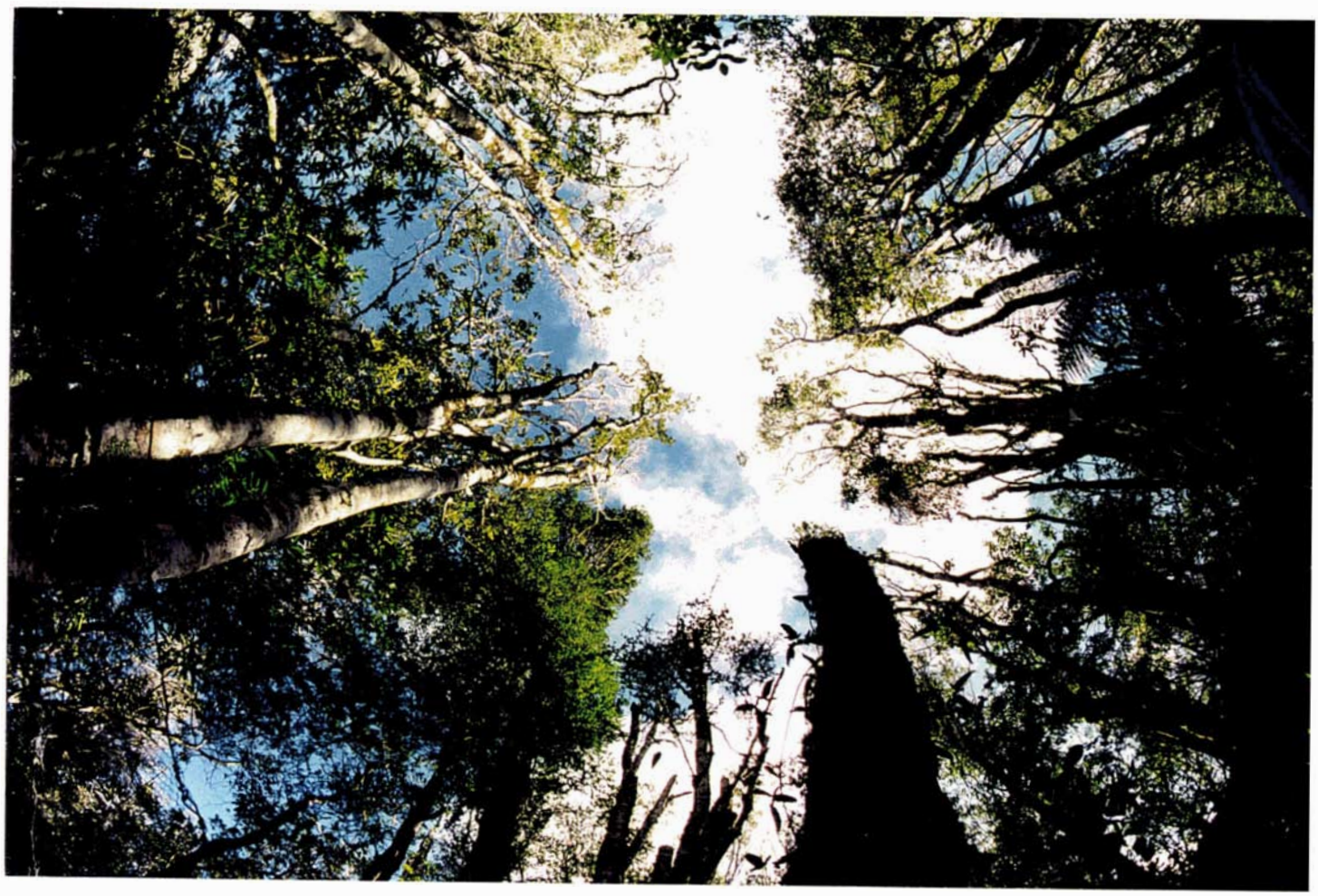
"And I forget just what it takes
and , yeah, I guess it makes me smile,
I found it hard,
it was hard to find,
oh well, whatever, nevermind."

Cobain, 1990.



"I greet him the days I meet him,
and bless when I understand."

Hopkins, 1875.

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Pretty much every morning I get dressed. I don't have many clothes to choose from, but I pick out what I feel like wearing and ask either Megan or my flatmates how I look. Scruffy, usually. Sweet as, I say - because I'm a student. A student. And with that in mind...

TABLE OF CONTENTS

ABSTRACT

Forest dieback is a complex area of study that has led to the development of a number of theories or models which purport to explain it. These models are examined using the example of kamahi dieback in Tongariro National Park. There has long been concern over the health of kamahi in the Park and it is thought kamahi could be in a state of decline. A survey on three transects in the area of the Park where dieback is most apparent compared kamahi health to possible predisposing, triggering and hastening factors (the decline-disease theory of forest dieback) to determine their role in any dieback. Possums, pinhole borer, and *Sporothrix* fungus were highlighted in literature as likely triggering factors in kamahi dieback; an experiment examined their role: possums were excluded from trees, pinhole borer attack simulated, and healthy trees infected with *Sporothrix*, while unhealthy trees were treated with fungicide. To assess the effect of canopy health and vertebrate browsers on regeneration (regeneration is important in both the cohort senescence theory and the model of stand succession), another experiment was conducted using open and exclosure plots under healthy and thinning canopies. A second survey assessed the overall health of kamahi in the area, and compared site and tree factors to levels of dieback.

The survey of an area with high apparent dieback found 14 % of kamahi stems were dead. There was some evidence that age predisposed stems to dieback, and *Sporothrix* was identified as accelerating stem death; no causal factor was determined. The experiment found no evidence that possums, pinhole borer, or *Sporothrix* were affecting the health of kamahi at this site. Sites under a thinning canopy in the regeneration study were much more variable in composition than sites under the healthy canopy; while sites in exclosure plots had higher densities of seedlings than sites that browsers had access to. Sites covered by the broad-scale survey contained very few unhealthy or dead kamahi trees, and none of the factors studied seemed to be impacting on the health of kamahi. There were more small (between 50 cm and 2 m) saplings present at sites with more dead kamahi trees.

It was concluded that kamahi in Tongariro National Park is generally in a healthy state, and not undergoing decline; although localised dieback may be very high. Possums, pinhole borer and *Sporothrix* are not having a large effect on kamahi health in this area. Vertebrate browsers may be maintaining regeneration at a level below natural. Comparison between the theories of dieback, succession and the kamahi data indicate that the successional model may best explain the pattern of kamahi dieback.

The models of dieback and succession can be inserted into an overriding model of dieback management, which will indicate the best path for investigating forest dieback.

Chapter 1: The dieback phenomenon with reference to kamahi (*Weinmannia racemosa*) in Tongariro National Park, New Zealand

Death is a natural event, but accelerated death can be more or less natural. Trees may die for many reasons, and over many scales. Some of these reasons are obvious, such as fire or flooding, or severe droughts, pollution or pestilence (Mueller-Dombois, 1988). The terms below do not refer to these types of death, but rather to death where there is no clear cause, and often a combination of factors involved. Although many trees pass through a period of deterioration or senescence before death, this thesis uses 'dieback' to refer to a category of 'disease' that does not connote any specific cause - it equates to Sinclair's (1988) 'decline disease'. Dieback implies that there is no primary agent capable of causing such death in healthy trees (Houston, 1992). An almost defining feature of this type of tree death is the lack of agreement among researchers on the cause and importance of the factors involved (Manion, 1991), and this is the issue addressed in this thesis.

The following terms are used in this thesis:

Disease - malfunctioning of host cells and tissues that results from continuous irritation by biotic (e.g. pathogens) or abiotic (environmental) factors, and leads to development of symptoms (Agrios, 1988; Beever, Forster, Rees-George, Robertson, Wood & Winks, 1996);

Dieback - progressive loss of health and death of shoots, branches or roots, crown foliage lost out of season, may lead to 'stagheads' and death (Agrios, 1988; Mueller-Dombois, 1992);

Stand-level dieback - synchronous dieback of the canopy of whole stands or parts thereof, rather than isolated trees (Mueller-Dombois, 1988; Steel, 1989);

Decline - widespread dieback, to the extent that a species or vegetation type is deteriorating (Manion & Lachance, 1992b);

Natural - largely non-anthropogenic;

Waldsterben - literally 'forest death', applies to 'novel forest decline' in Europe, and particularly Germany (Kandler, 1992).

DIEBACK

Stand-level dieback occurs over a wide spectrum of forest types and around the globe, and is receiving increasing interest from ecologists (Steel, 1989; Mueller-Dombois, 1993). It is being investigated in many countries, in both southern and northern hemispheres (for example, Agyeman & Safo, 1997; Akashi & Mueller-Dombois, 1995; Arentz, 1983; Auclair, Lill & Rivenga, 1996; Balakrishnan & Mueller-Dombois, 1983; Fisher, 1997; Haemmerli & Schlaepfer, 1993; Hinrichsen, 1987; Hosking & Hutcheson, 1986; Houston, 1974; Huettl & Mueller-Dombois, 1993; Jane & Green, 1983; Kandler, 1992; Landmann, 1993; Landsberg, 1990; Manion & Lachance, 1992a; Mueller-Dombois, 1988; Ogden, Lusk & Steel, 1993; Payton, 1987; Waring, 1987; Ziegler, 1988). It is certainly not a new phenomenon, and in many cases is due to 'natural' causes (Mueller-Dombois, 1987). However, it is often very difficult to distinguish between those and anthropogenic causes of dieback. There is a tendency to blame people in some way for any widespread death of trees in the absence of an obvious cause (Mueller-Dombois, 1986), to the extent that in many industrial countries, particularly European, dieback is always seen as a new and anthropogenic problem (Mueller-Dombois, 1988).

Franklin *et al.* (1987) comment that tree death is an extremely complex phenomenon, and often represents an arbitrary point on a continuum of deterioration; the factors influencing death may not be the factors that were influencing health. They stress tree death as part of the stand dynamic, and the consequence of the same multiple contributing factors that affect that dynamic. Mueller-Dombois (1983a) adds that dieback does not end the life of the community, but that succession and community dynamics continue in spite of it; further, dieback will have special effects on the succession in those stands, for example in allowing podocarps to be released.

The literature on dieback around the world is extensive: there have been symposia and text books published on the subject (e.g. Manion & Lachance, 1992a; Huettl & Mueller-Dombois, 1993). In the northern hemisphere air pollution is often the first factor blamed for any dieback phenomenon, while in the Pacific region similar patterns of dieback occur largely in the absence of high levels of industrial pollution, so other explanations are explored (Mueller-Dombois, 1983b).

In New Zealand, there has been research on a variety of taxa exhibiting dieback in a variety of areas, and with various causes suggested. Browsing by the Australian brushtail possum (*Trichosurus vulpecula*) had been the accepted cause of dieback in

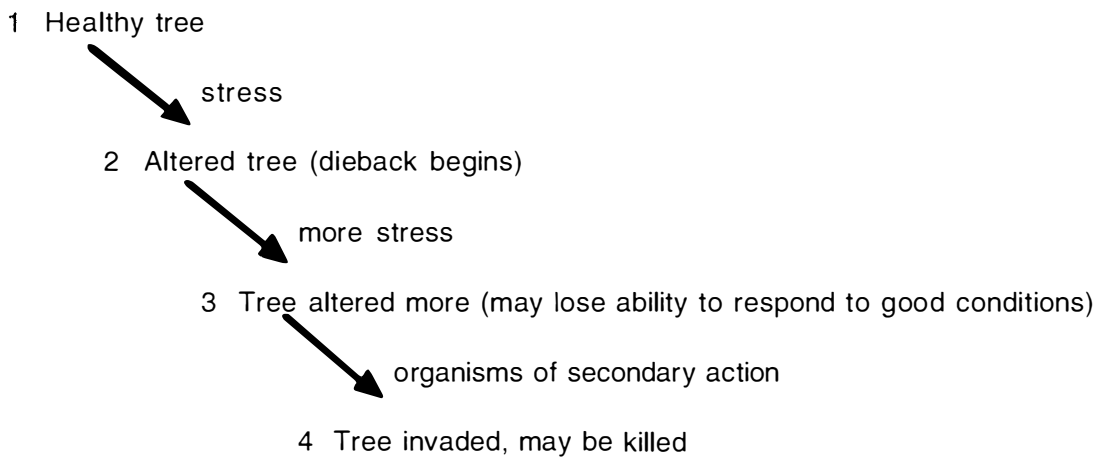
forests dominated by rata (*Metrosideros* spp.) and kamahi (*Weinmannia racemosa*) (as reviewed by Batcheler, 1983). Recently, it has been realised that the problem is more complex, also involving, for example, stand structure and regeneration dynamics (Payton, 1988), as well as a plethora of other factors. On the Huiarau range (Urewera region), where podocarps were observed to be declining, introduced feral browsers were initially blamed, but reduced precipitation effectiveness is now believed to be the cause (Grant, 1963). In the Kaimai Range, again it was thought that introduced browsers were to blame for the deterioration in forest health (Jane & Green, 1983). It is now understood that low levels of soil nutrients were reducing plants' ability to respond to drought and disease, and deer were then affecting the ability of the forest to recover. The 'abnormally high mortality' of canopy trees in the beech forests of the northern Ruahine Range in the early twentieth century is believed to be due to the effect of drought (Grant, 1984). Dieback of pohutukawa on White Island was found to be largely due to toxic fumes from volcanic activity (Clarkson & Clarkson, 1994).

Thus many factors have been implicated in forest decline in New Zealand. Internationally, there has been a move towards developing general models of dieback, or of forests to explain the dieback occurring within them.

MODELS OF DIEBACK

From information on specific cases of dieback, a variety of theories or models have been developed that seek to give more general explanations for forest dieback. These models highlight the ecological processes driving dieback, as opposed to simply describing its purported conservation significance or visual impact. The most general model is from Houston (1984):

1. GENERAL MODEL OF DIEBACK

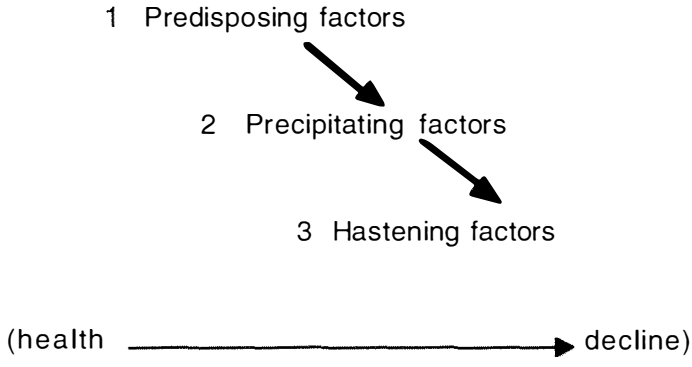


Step 1 begins with a healthy tree, that becomes stressed. If the tree is stressed enough, it will alter its development as a response, often reducing growth as dieback is initiated. If the stressed tree is exposed to further stress, it may become so altered that it is unable to respond to favourable conditions, such as a respite from drought. At this point the tree is effectively 'doomed', and attack by organisms of secondary action will finally kill it. The type of stress is not specified, but any stressor of trees will fit, for example drought, browse pressure, nutrient deficiency or acid rain. The organisms of secondary action are often the proximate causes of tree death, and manifest as plague or pestilence, such as vascular wilt diseases, root rots or insect attack. As they are so often associated with death, they tend to be blamed for the dieback, rather than the original stressor which caused the deterioration in health that leads to death (Hosking, 1993b; Hosking, 1993a).

Mueller-Dombois (1986) highlights the need to focus more on the structure of forests exhibiting dieback, and to examine their spatial and temporal patterns, habitat relationships and associated vegetation and successional responses, in order to assess what is driving the dieback, and whether it is really a problem for the forest. The following models provide frameworks within which these can be studied.

2. DECLINE DISEASE THEORY OF STAND-LEVEL DIEBACK

This theory was developed by Houston (1974), and is discussed further by Mueller-Dombois (1988) and Manion (1991). It sees dieback as a three step process or chain reaction, and is largely a re-expression of the previous model, with the three stages of this model corresponding to the three arrows (or stressors) of the general model :



Predisposing factors affect a stand's **susceptibility** to dieback. Stand structure is considered an important factor, with single-aged stands being particularly susceptible as they approach senescence. Extreme edaphic conditions and recurring perturbations can also predispose a stand to dieback. Landmann (1993) states that stands where soils are rich in nutrients but 'physically unfavourable' (for example, on steeply sloping sites) are perhaps the most predisposed to dieback. The argument is that these stands will mature most rapidly, without the necessary development to avoid the impact of mild stresses, for example a well-developed root-system to avoid drought or nutrient stress. This stage is similar to step 1 of Houston's (1984) model (see above), with trees healthy.

Precipitating factors act to **synchronise** death in stands that contain trees predisposed to die. Precipitating factors may be abiotic stresses, such as drought or severe storms, or biotic agents such as high levels of animal browse (Mueller-Dombois, 1986). They trigger actual death of trees. This is the stage where dieback symptoms are first exhibited, and relates to steps 2 and 3 of the general model.

Hastening factors **accelerate** the dieback, and are generally biotic in nature, such as pathogenic fungi, and insects or other herbivores (Mueller-Dombois, 1992). They correspond to the 'organisms of secondary action' from the previous model (see above).

This theory has served to provide a focus for research on stand-level dieback, and can be used for dieback events of natural or anthropogenic nature (Mueller-Dombois, 1988). Stewart (1989: 243) goes so far as to say that 'all known examples of dieback

in New Zealand *Nothofagus* spp., *Metrosideros* spp., and beech/hardwood forests can be explained using this three-factor framework'. It has been a very influential theory, and seems to have become ubiquitous or all-pervasive in dieback research.

3. COHORT SENESCENCE THEORY

This theory, developed by Mueller-Dombois (1983a; 1986), differs from the previous model, which implies a disease or disorder in the system, only in that it applies to, or describes, senescence and implies succession, which is part of the system. Whereas synchronous dieback of single-aged stands would be treated as a symptom of a problem in the previous model, in this model dieback is a normal and integral part of the system.

1. Catastrophic disturbance allows large cohort to invade the site

↘

2. Stand development proceeds; stands which are under similar conditions will remain synchronised demographically as they approach senescence (≈ the predisposing factor of previous model)

↘

3. Perturbations that usually only affect growth, now initiate dieback in stands predisposed (≈ precipitating factor of previous model)

↘

(leads to patchwork pattern of dieback based on pattern of site suitability)

↘

4. Other factors may hasten decline after initial breakdown of canopy, and may result in death of all life stages and larger areas than a single 'stand' (patchwork breaks down) (≈ hastening factor of previous model)

→

(successional gradient over time)

This model only applies to certain systems as it requires:

a) an environment with catastrophic disturbances;

b) species able to take advantage of such disturbances by rapid (re)invasion; and

c) that the widespread disturbance occurs less frequently over the site than the generation turn-over of the species concerned.

The above process may lead to successive cohorts becoming spatially reduced and out of phase with each other until another catastrophic disturbance resets the cycle.

4. MODEL OF SUCCESSION

Successional models may also include reference to dieback stages, and there are many examples of dieback that seem to be part of the regeneration process of a species, and that operate without catastrophic disturbances to start the cycle. This is classic gap-phase regeneration. One such classic model of succession is included to show that dieback occurs within an ecological system, and has profound significance on that system. This is important to remember, as the cause of dieback is so often considered to be outside the natural system, and thus upsetting the natural cycle. The model is from Mayer and Neuman (1981, cited by Mueller-Dombois, 1987) (fig 1).

For species, such as *Nothofagus* in New Zealand, and *Metrosideros* in Hawai'i, where dieback is very important in succession, this model explains the timing and significance of dieback, without reference to any causal factors.

These issues are investigated in this thesis on kamahi dieback in Tongariro National Park, New Zealand.

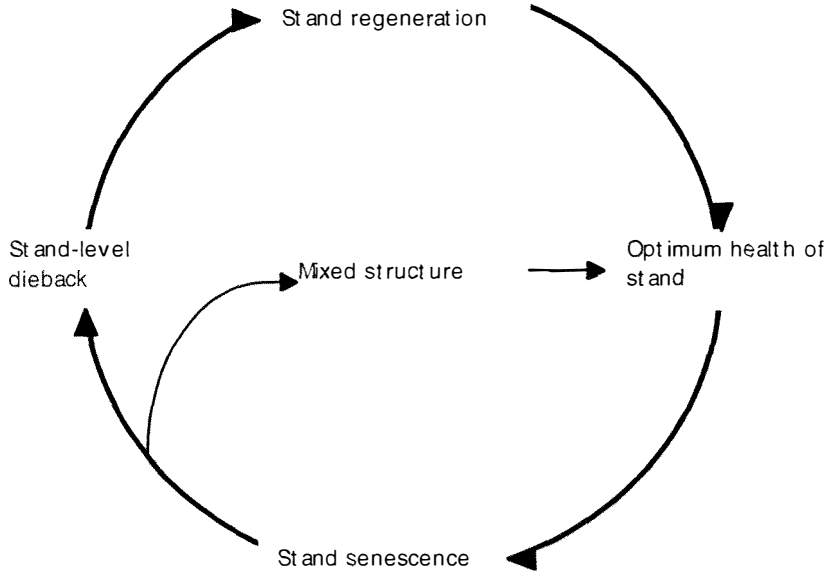


Figure 1 *Generalised model of stand succession from Mueller-Dombois (1987). Senescing stage is the longest, with more open canopy than the optimum health stage.*

TONGARIRO NATIONAL PARK

Tongariro National Park in the central North Island of New Zealand (39° S, 175° E; fig 2) is a park dominated by mountains (figs 3 and 4) .

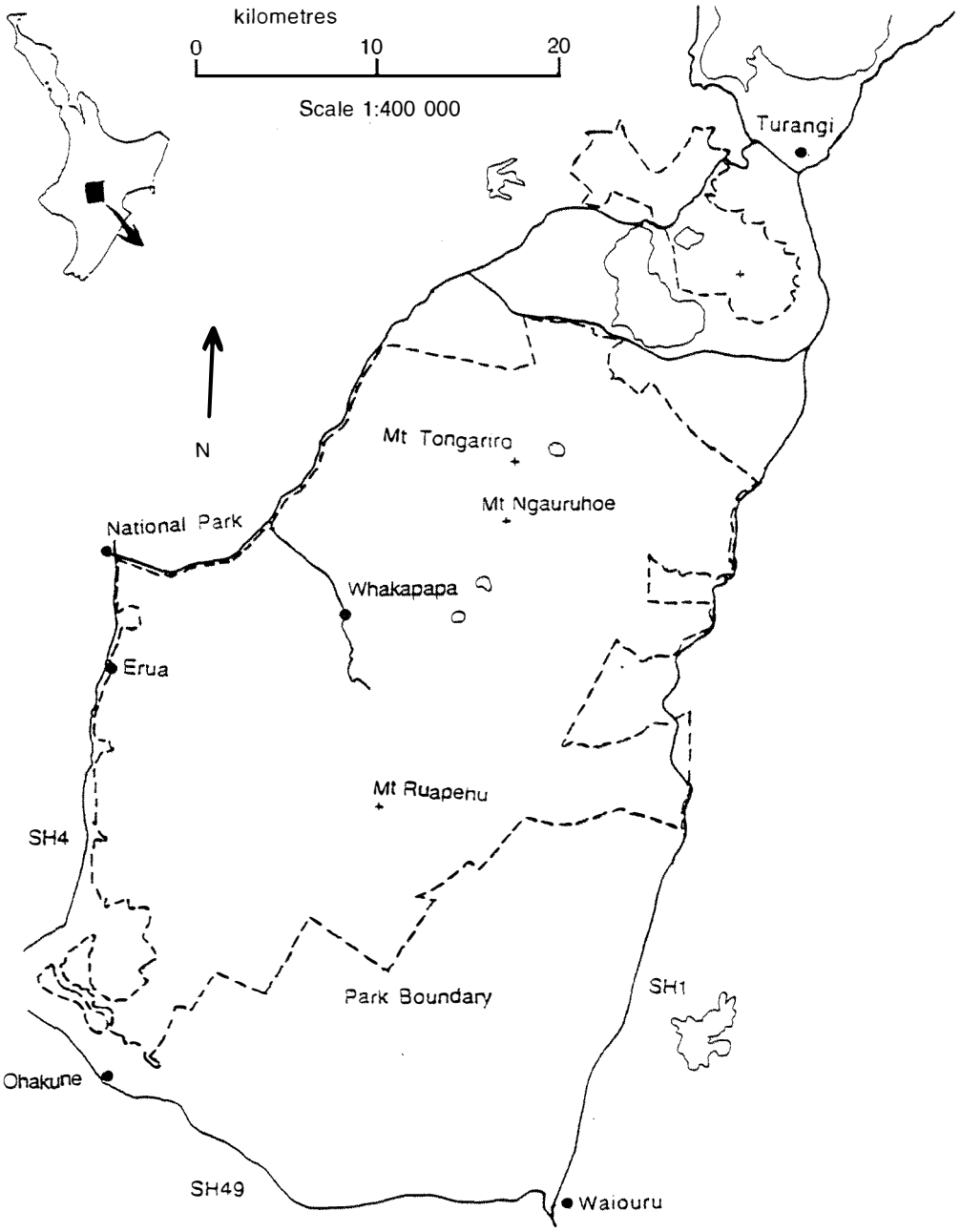


Fig 2 *Location of Tongariro National Park in the central volcanic Plateau, North Island, New Zealand.*

Fig 3 *Tongariro National Park in winter, showing mountains. Ngaurahoe (2291 m) is the conical mountain in centre, Tongariro (1967) the smaller, older mountain to the right (photo G.Rapson).*

Fig 4 *Ruapehu (2797 m) in Tongariro National Park (photo G.Rapson).*

Due to its background, it contains relatively small areas of native forest, and has corresponding problems of encroaching weeds, edge effects, and introduced browsers.

Park History

In 1887, Ngati Tuwharetoa as represented by Te Heuheu, gifted the three mountains Ruapehu, Ngauruhoe and Tongariro to the government of New Zealand. Te Heuheu was concerned that these mountains, the ancestors of Ngati Tuwharetoa, would be divided and sold piece by piece to Pakeha, who had already shown their disrespect for the mountains. These mountains became New Zealand's first National Park (and the fourth in the world) when Tongariro National Park was established in 1894 and gazetted in 1907. As the main concern of Ngati Tuwharetoa was for the integrity of the mountains, the original Park included only the peaks of Ruapehu, Tongariro and Ngauruhoe - 2600 hectares containing very little vegetation.

Following the recommendations of eminent New Zealand botanists Cockayne (1908) and Turner (1909), the Park was expanded with the purpose of including more vegetation types. This expansion has continued, and at present the Park includes over 80 000 ha (Department of Survey and Land Information - Te Puna a Korero Whenua) and is a World Heritage area.

The Park now constitutes a varied landscape, for example the volcanic mountains, plateaux, lakes and desert. It thus contains a diversity of vegetation types (Atkinson, 1981; 1985), from lowland podocarp/hardwood forests, through montane beech (*Nothofagus* spp.) forests, subalpine shrubland, to alpine herbfields; from bogs and almost rainforest on the west, to the Rangipo Desert on the East; and also including areas of heathland and tussockland. It was this diversity of vegetation that Cockayne (1908) had so wanted to preserve. However the Park is not without its conservation problems, as the tussocklands are widely invaded by european heather (*Calluna vulgaris*). Additionally, the health of the forests have caused concern to managers, for example beech in the past (Skipworth, 1981), and today kamahi.

KAMAHI (*WEINMANNIA RACEMOSA*)

Kamahi (sometimes called tawhero around National Park, or Kamai in Southland) is an important component of all but the higher altitude forest associations in Tongariro National Park (Atkinson, 1981). It is a non-offensive, pleasant-looking canopy tree, growing up to 25 m tall, and to a trunk diameter of over 1.2 m (Salmon, 1980). It may grow as a single stemmed tree, or may assume a many-stemmed guise, and is often of epiphytic origin with very irregular stems (Wardle, 1966) (fig 5). Kamahi leaves are thick, leathery and serrated (fig 6), with much variation in shape. Juvenile leaves tend to be tri-foliate, and adult uni-foliate.

Kamahi is endemic, and closely related to *Weinmannia silvicola*, which is the more northerly species, ranging from Waikato and the Bay of Plenty north to Mangonui (Salmon, 1980). Wardle (1966) speculates that kamahi is New Zealand's most abundant canopy tree, and comments that it probably associates with every other tree species present south of the Far North. It ranges from Auckland to Stewart Island, and from sea-level to around 1100 metres above sea level, but may not be tolerant of drought and poorly drained sites.

Kamahi produces racemes of white or red-tinged flowers, abundant some years (figs 7 and 8), and less in others (pers.obs.). Flowers appear in late spring, and may persist as seed pods to the following year. The very small and abundant seeds are wind dispersed, and require a lit situation (although not *direct* sunlight) to germinate, but seedlings prefer shaded conditions (Wardle, 1966). Seedlings may be either epiphytic or terrestrial in habit. Kamahi saplings have extremely fast growth rates, and are the most common canopy species present in forest gaps (Ogden, Fordham, Pilkington & Serra, 1991). This leads to continual recruitment, and the typically all-sized population structure (Lusk & Smith, 1998).

Kamahi is thought to be an important species in succession, particularly following disturbance (Wardle, 1966). And dieback of kamahi may be an important part of the forest system, where podocarps can regenerate under kamahi, but not under adult podocarps (Beveridge, 1973).

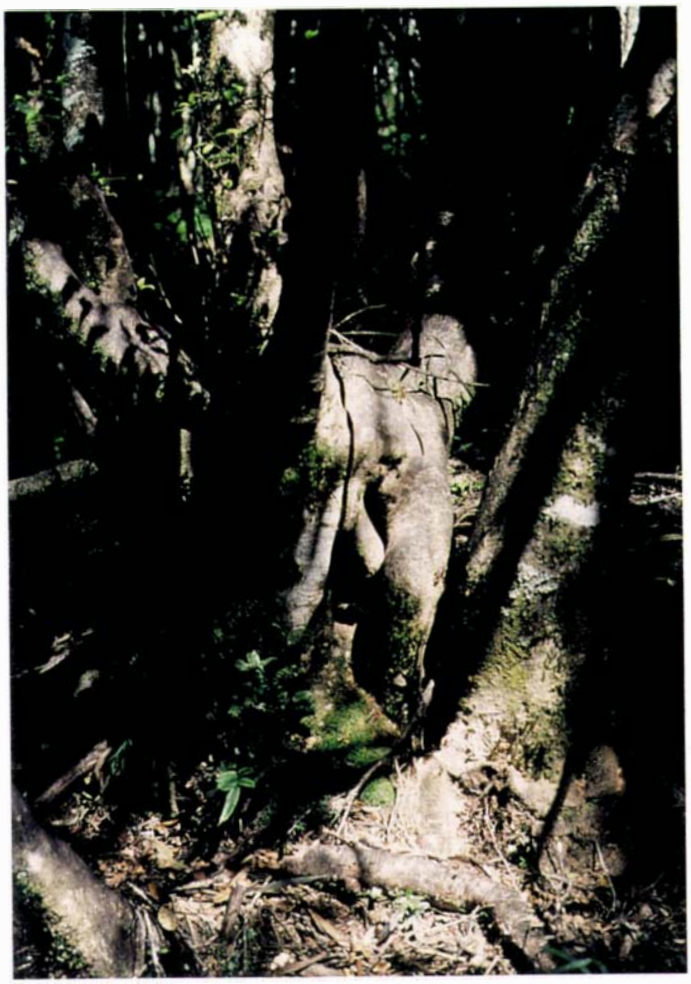
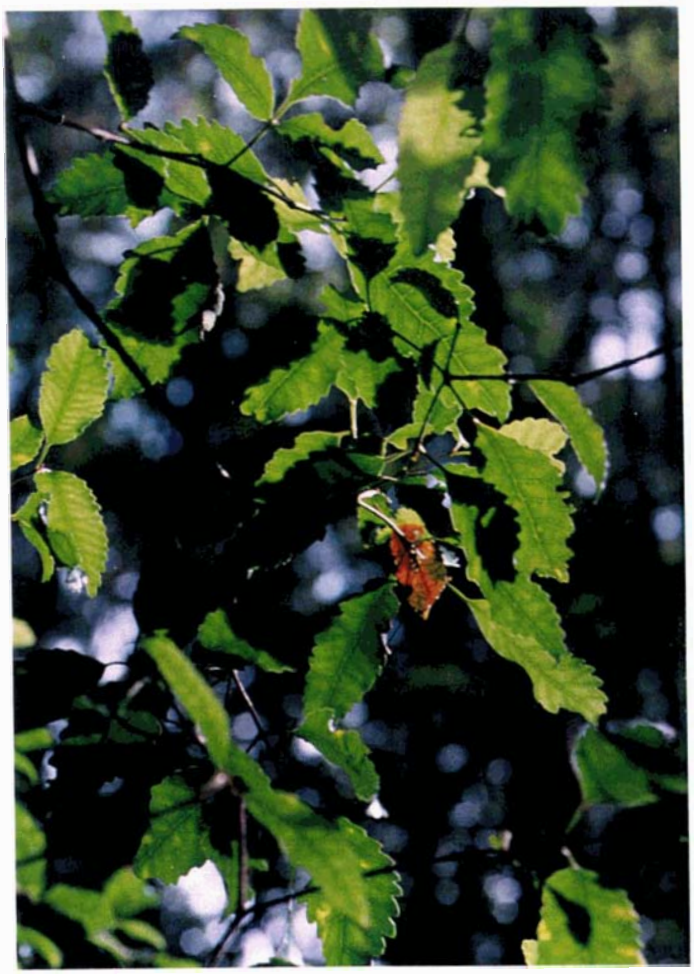


Fig 5 *Kamahi stem, showing irregular nature on trees of epiphytic origin (photo G.Rapson).*

Fig 6 *Kamahi foliage (photo G.Rapson).*



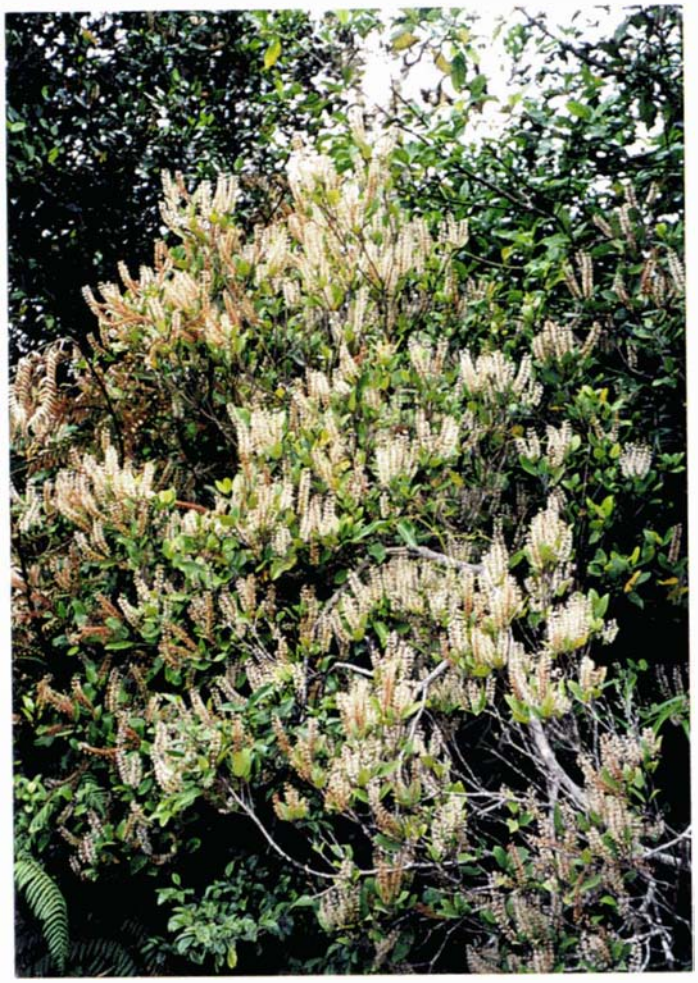


Fig 7 (right) *Kamahi branch showing abundant white racemes of flowers.*

Fig 8 (below) *Kamahi bush with seedpods (brown) and two racemes still with flowers (white).*

AIMS OF THIS STUDY

Dead trees are an obvious feature of the forest of some areas of Tongariro National Park and have given rise to concern that kamahi forests may be in general decline. Possums have been widely assumed to be causal in this situation, possibly in combination with insect and fungal pathogens. Kamahi in Tongariro National Park can be considered a case study of a dieback phenomenon, in the context of models of forest dieback. Superficially it appears to fit each model, with the patchy mortality typical of decline disease, cohort senescence and the successional model presented. History of Tongariro National Park is briefly reviewed, as well as aspects of the ecology of kamahi, as important background information. This research examines whether kamahi is in decline, and attempts to find the causes of the mortality.

The aims of this thesis are :

(1) to establish the extent of kamahi mortality (chapter 2a);

(2) to examine the correlations between dieback and possible predisposing and / or triggering biotic, environmental or demographic features (chapters 2a and 2b, with 2b focusing on an area of more severe dieback);

(3) to establish causality of these relationships by the effect of their manipulation on kamahi health (chapter 3);

(4) to examine the role of kamahi mortality in forest succession (chapter 4); and,

(5) to examine the effect of browsing (largely by introduced mammals) in constraining replacement of kamahi (chapter 4).

Underlying these aims is the desire to identify which (if any) of the various dieback models applies in this case study, and to suggest management strategies for identifying dieback problems and deciding on the most appropriate course of action.

APPROACHES

The approach taken in this thesis to answer the preceding questions is a combination of surveys and experiments. In order to understand the extent and patterns of kamahi mortality in the Park, two surveys were undertaken. The first, a broad-scale survey of kamahi habitats in and near the Park, aimed to assess the extent and scale of kamahi dieback, and to suggest causes (chapter 2a), it aims to give an overview of the situation rather than directly addressing any of the models. The effects of possible causes, such as possum browse, insect browse, and susceptibility to drought and age, on kamahi health were tested by examining correlations between them and the level of dieback observed in trees. The second survey focused on an area of the Park where kamahi mortality had been found to be heaviest (chapter 2b). Using the decline-disease theory

of forest dieback, this survey aimed to ascertain the chief predisposing, triggering and accelerating factors in kamahi dieback, by comparing each of the most likely factors to measures of kamahi stem or stand health. The structure of the fieldwork for chapter 2b was largely determined by the Department of Conservation, who located and marked the transects and each of their quadrats, as well as numbering each stem within each quadrat. The Department of Conservation also collected much of the data describing each stem, although all but Diameter at Breast Height were later checked by myself in the field.

A review of the literature on high levels of kamahi mortality elsewhere in New Zealand highlighted browsing by the introduced brushtail possums, attack by *Platypus* spp. pinhole borer, and invasion by *Sporothrix* fungus as being the most likely, or most commonly discussed, causes. In this study, an experiment was designed and executed to examine the impacts of these factors, both separate and interacting, on kamahi health in Tongariro National Park (chapter 3), and to test any findings from the two surveys. The impacts of reducing possums' access to healthy and unhealthy trees (by banding stems), excluding fungi from unhealthy trees (using systemic fungicide), introducing *Sporothrix* to healthy trees, and boring large numbers of holes in healthy trees (as *Platypus* spp borer often do) were tested. This experiment uses the concept of triggering factors from the dieback models: it identifies the factors most likely to trigger a decline in health in kamahi, and assesses their ability to be causative.

Regeneration at sites under a healthy canopy as opposed to a less healthy canopy, and the effect of excluding larger animals were looked at to assess the importance of stand level dieback in succession (chapter 4). If regeneration is markedly different under a thinning kamahi canopy than under the healthy canopy, particularly if seedlings of canopy species appear more suited to an unhealthy canopy, then stand level kamahi dieback is likely to be important in the forest system (as in the successional model presented by Beveridge, 1973). The effect of exotic herbivores is investigated to determine the role they are having in this process, in particular whether suppression of seedlings by browsing will be having an effect on levels of successful regeneration. High levels of regeneration (in the absence of herbivory) are features of the cohort senescence and successional models (though not necessarily restricted to them).

The thesis concludes with a revision of the evidence for proportion and scale of kamahi dieback in the context of the dieback models, identifies which of these models

(if any) best fits the data, and proposes a model for investigation and management of high tree mortality in native forests.

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Chapter 2: Surveys of Kamahi (*Weinmannia racemosa*) dieback in Tongariro National Park

INTRODUCTION

As discussed in the previous chapter, forest dieback or decline is an important and globally widespread issue, with many different theories which attempt to describe and explain it. These theories tend to separate into those that focus on the mortality as an unnatural, anthropogenic problem, and those which focus on periods of increased mortality as naturally occuring within the forest system, and necessary to that system's survival. In order to examine the validity of the more important of these theories, an example of forest dieback is necessary.

Kamahi (*Weinmannia racemosa*) is a very important canopy tree species in the forest of Tongariro National Park, making up a huge proportion of the total basal area of the forest on the western and southern sides of the Park (approximately 45 % of the basal area of live trees on the western lower slopes of Hauhungatahi is kamahi (unpub. DOC data)). Large scale dieback of kamahi has been noticed on the western side of the Park. The cause of the high rates of mortality observed has been much speculated on, with possibilities ranging from those of an anthropogenic origin, such as browsing by the introduced brushtail possum (*Trichosurus vulpecula*), to completely natural causes, such as drought stress. It appears to be an ideal example with which to examine forest dieback theory. The following two sub-chapters (2a and 2b) present surveys that provide information on kamahi mortality at two scales that can be compared to the theories.

An obvious first approach for a study of species mortality is a survey, covering areas where dieback is and is not evident, to determine the magnitude of the dieback 'problem', any patterns of mortality that may occur, and whether the mortality appears to fit successional models. A broad scale approach is necessary for such a widespread species which occupies a range of forest habitats and roles within the forest community (being important in secondary succession, as well as an important sub-canopy and canopy species). This approach allows generalisations on the environmental preferences and related vulnerabilities of the species to be made, as well as providing baseline data for interpreting its ecology. Chapter 2a investigates the level of kamahi mortality in the National Park area, and compares health of trees to several site factors and possible causal factors, as well as to indices of regeneration.

Another strategy is to focus on the area where mortality appears most common, in an effort to discover the causes of that mortality. This approach is useful when there are management issues involved, as conservation managers are generally *re*-active, responding to public concerns, and high levels of extremely visible dieback are likely to attract adverse comment from Park users. A more detailed approach also allows focus on factors likely to be impacting directly on the health of an area of forest, regardless of whether the dynamics, demographics and ecology of that area is typical of the species under study. Chapter 2b presents a survey conducted on the western slopes of Hauhungatahi, where kamahi mortality is most noticeable. The emphasis is on the fate of individual poles and/or stems within each stand, and uses more detailed observations of health and damage types within these stems (as opposed to the trees of the previous survey). This survey investigates the causes of the mortality using the decline-disease model of forest dieback.

The chapter concludes with a brief comparison of the two surveys.

Chapter 2a: Assessment of extent and possible causes of kamahi (*Weinmannia racemosa*) dieback in Tongariro National Park

ABSTRACT

Concern has been raised over the health of kamahi in Tongariro National Park. Thirty sites representative of kamahi in the area were selected to investigate the health of kamahi at those sites, and to investigate the nature of the relationship between kamahi health and a number of factors thought to impact on that health. Site characters considered to be of particular interest were altitude, slope, aspect, exposure, canopy closure, drainage, canopy height, density of kamahi, presence of possums and level of regeneration. On individual trees, level of dieback was compared to crown density, proportion of dead terminal shoots, levels of reproduction, epicormic sprouting, damage to trunk and foliage by possums, pinhole borer infestation, insect foliar damage, foliar wilt and proportion of twig breakage.

Very few trees encountered in this study were dead (4 %) or dying (3 %). None of the site factors that had been suggested as possible causes of dieback are related to kamahi health at these sites; although, the number of small saplings (50 cm to 2 m tall) was positively related to the number of dead kamahi at each site. None of the causal factors measured on each tree were related to the health of that tree. Crown density was less able to predict the amount of dieback in a tree than was the level of dead terminal shoots.

It was concluded that kamahi in the forests in and around Tongariro National Park is in a healthy state, although patches of high mortality do occur.

INTRODUCTION

In the study of species' declines, attention is often paid to the pattern of the species where it is dying, with little attention paid to the pattern of that species where it is healthy (Acker, Harmon, Spies & McKee, 1996). Acker *et al.* (1996) also comment that ignoring the prevalence and pattern of healthy trees can give a distorted view of the extent and causes of dieback. While it is clear that, *in some areas of Tongariro* National Park (*i.e.* the western side of Hauhungatahi, as studied in chapter 2), kamahi *is* dying at an apparently very high rate (around 14 %), the overall patterns of mortality and regeneration for kamahi in the area are not known.

STAND DYNAMICS AND PATTERNS OF REGENERATION

Only recently has serious attention been paid to the processes of stand dynamics in relation to natural disturbances (Stewart & Veblen, 1982); yet stand dynamics are extremely relevant to understanding dieback, and essential to discussing its implications. It is important to remember that death is an event that will occur in the lifetime of every tree, as it begins a new role in the forest system (Franklin, Shugart & Harmon, 1987). If a cohort of trees establish together, and develop under *similar* conditions, it is plausible they will approach *senescence together* (Mueller-Dombois, 1987). This synchronous senescence may play an important role in the system. For example, in dying stands of *Metrosideros* in Hawai'i there was much more regeneration than in comparable healthier stands (Jacobi, Gerrish & Mueller-Dombois, 1983).

The forest dynamics and regeneration patterns of many of New Zealand podocarps appear to be dependent on development beneath a canopy of large, dying hardwoods (Beveridge, 1973). Beveridge outlines a six stage cycle of *regeneration* for New Zealand forests based on Pureora while possum numbers were very low, and deer had only been affecting the forest for 10-15 years (fig 2.2). Stage 1 requires windfall of a large podocarp to open a canopy gap, under which a colony of (usually) *Dicksonia squarrosa* develops and inhibits recruitment of all trees on the ground (stage 2). The tree ferns provide sites for epiphytic establishment of (usually) kamahi (stage 3), which eventually suppress the tree ferns and later provide perching for (usually) kereru (*Hemiphaga novaeseelandiae* Gmelin) (*stage* 4). Stage 5 *sees* recruitment of podocarp seedlings from bird-dispersed seeds, and as the kamahi canopy dies the podocarp saplings develop (stage 6). It may take 200-300 years from time of windfall of a podocarp to establishment of a new podocarp pole stage.

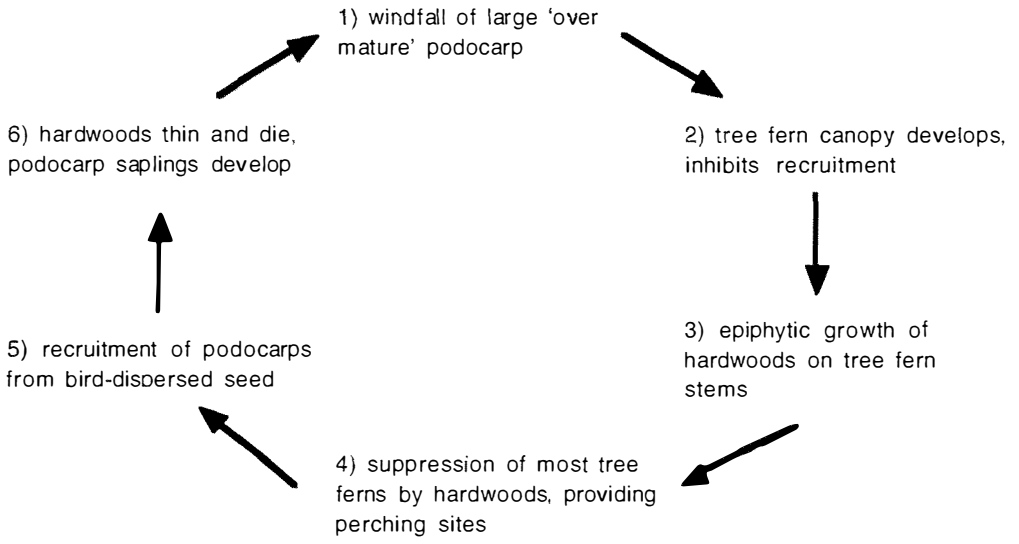


Figure 2: *Generalised model of a regeneration cycle for a North Island forest in the absence of exogenous factors (from Beveridge, 1973).*

The cycle may be disrupted at any stage by more widespread disturbances. For example, if any step were disrupted by a fire, the cycle would be reset to stage 1, and only the scale of the pattern should be affected; scale is determined by the nature of the disturbance (Ogden, Lusk & Steel, 1993). However, if a disturbance occurs regularly enough (such as anthropogenic fires) or affects recruitment (as do many introduced herbivores) the cycle may break altogether and lead to a different vegetation type, such as pasture or scrub.

AIMS

Kamahi dieback has been reported in Tongariro National Park and has been found to be occurring at a high rate in some parts of the Park. This study investigates the rate at which dieback is occurring overall, to assess whether kamahi may be declining in this area. It will assess whether this dieback is caused by natural or anthropogenic factors, as the ecological significance of each is quite different.

A sample of kamahi sites, chosen haphazardly from a range of kamahi habitats, were studied to assess the health of kamahi present. The null hypothesis was adopted, that kamahi in the area is not undergoing decline, and any mortality observed is as a result of natural causes. Sites were compared for a number of different characters, which may impact on kamahi health, and regeneration of kamahi at each location was assessed, to indicate the likelihood of replacement of any dead trees by kamahi.

The health of kamahi trees at these locations was measured and compared to indices of some factors that are suggested to cause dieback. Some were measured indirectly, such as *Sporothrix* where the prevalence of its vector, pinhole borer (*Platypus* spp.), was the index. Others were measured as directly as possible, for example, browsing. Some variables' measures may indicate any of a number of causes, such as wilt, where for example, water stress will induce wilt; *Sporothrix* may induce water stress by blocking sapwood; drought may reduce available water; water-logging may lead to root rot and leaf wilt, *et cetera*. Health was measured directly (health and dead terminal shoots) and indirectly, such as epicormic growth (suggested by Leutert (1988) to be a response to stress in rata) or reproduction. The indirect measures were used to determine how appropriate these more easily measured variables are to predict health, possibly allowing a reduction in sampling time if they can be used to assess health of a tree.

METHOD

The survey of kamahi health in Tongariro National Park was undertaken during the summer of 1995/96, with the first sites, on Hauhungatahi, scored on 1st December 1995, and the last completed by the end of March 1996.

SITE SELECTION

Atkinson's vegetation map (Atkinson, 1981) was used to determine where kamahi is found and the range of habitats it occupies within the Park. Sites are positioned haphazardly within these areas, and include as representative a portion of kamahi habitats as possible, although areas suspected to contain dieback were sampled more intensively. The location of sites is shown in figure 3.1 (appendix A lists locations of sites). Each site is at least 20 metres x 5 metres, and contains ten or more kamahi trees, with 'tree' defined as having a diameter at breast height (DBH) equal to or greater than 10 centimetres. All trees within this area are included, and if the area contained fewer than ten trees, it was expanded to include ten.

SITE SURVEY

Details of the 'physiognomy' or physical character were recorded for each site. Altitude (in metres above sea level) was determined from maps; slope and aspect (in degrees) were measured at the site. Level of exposure is on a scale of 0 to 2, where 0 is not exposed, for example in a gully, and 2 is very exposed, particularly on ridges. Canopy closure was scored as 0 if the canopy is completely closed, and 2 if very open (approximately corresponding to less than 50 % canopy cover). Drainage is on a scale of 1 to 5, with 1 free draining and usually on a slope, and 5 if water is able to 'pond'.

To describe the character of kamahi, the approximate average canopy height (in metres) and density of kamahi (per 100 square metres) at each site was recorded.

Five circular sub-plots 3 metres in diameter were studied intensively at each site for the occurrence of kamahi regeneration and possum pellets. These plots are located at five metre intervals the length of the site, and ten paces perpendicular to the mid-line in alternate directions. Kamahi regeneration was split into three classes: seedlings less than 50 centimetres in height; saplings taller than 50 centimetres but less than 2 metres in height; and saplings taller than 2 metres but with a diameter at breast height less than 10 centimetres. All kamahi within the sub-plots in each of these classes were counted. For possums, each single or discrete pile of possum pellet(s) within the sub-plot was recorded. The number of pellet piles at each site

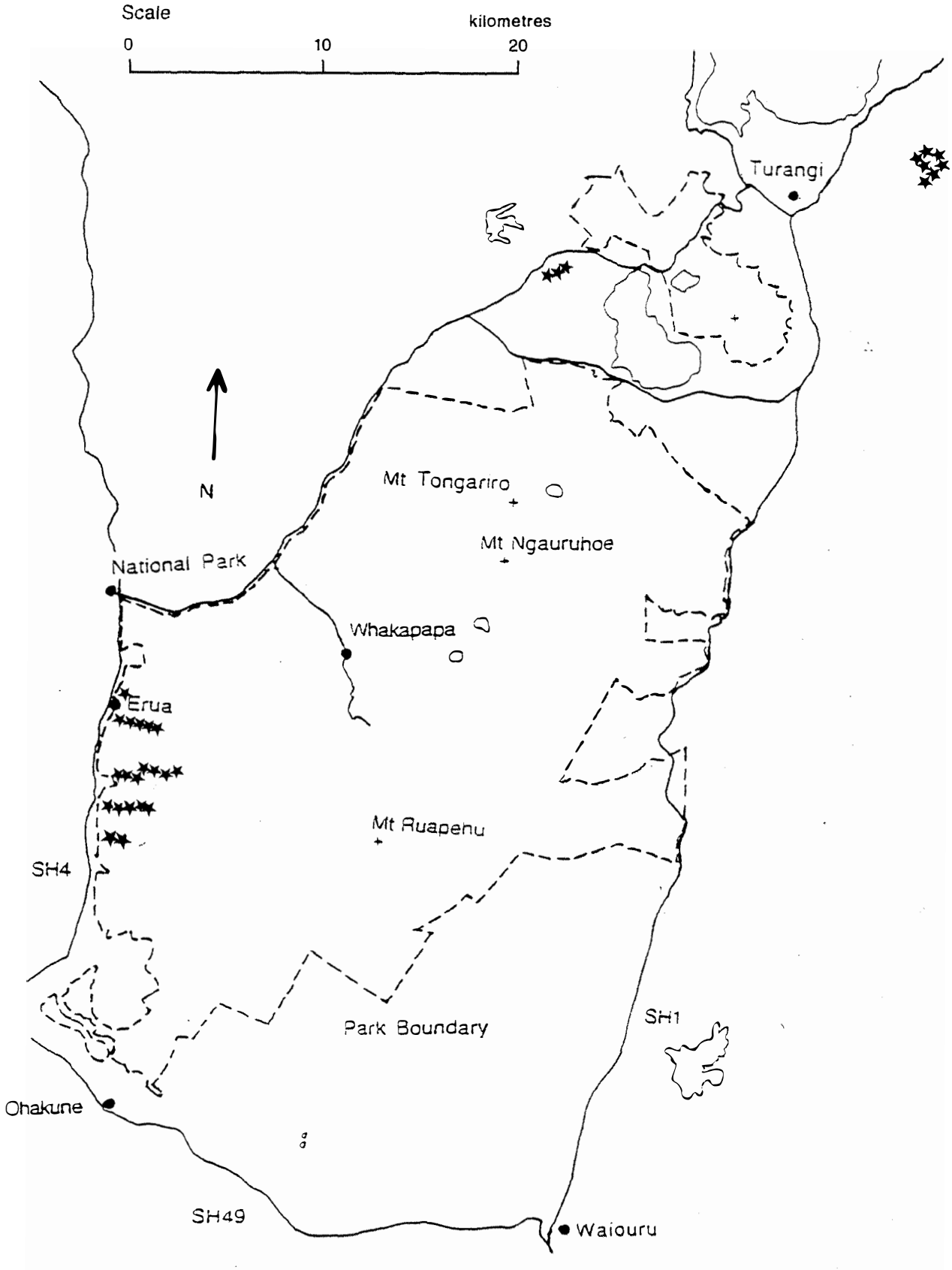


Fig 3.1 *Very approximate location of sites (✱) surveyed.*

indicates level of recent possum *usage*, not population or density (Pekelharing & Reynolds, 1983).

The percentage canopy cover and percentage ground cover were estimated. 'Canopy' is defined as being the uppermost *layer* of plant crowns (c.*f.* emergents), and cover as the proportion of the ground covered by a vegetation layer when projected vertically downwards (Atkinson, Jenkins & Druce, 1968). Ground cover is the proportion covered by the lower most vegetation layer (approximately less than 1 m high). All canopy and ground species occupying more than 10 percent of that layer were recorded.

TREE SURVEY

Characters

Within each site, diameter at breast height was measured for each kamahi tree, and its height estimated. Status in the canopy was recorded: the tree is considered canopy status, and given a score of 1, if half or more of its crown is exposed to direct radiation from the sky; it is considered sub-canopy, with a status score of 0, if less than half its canopy was exposed (from Atkinson, 1962).

Health

The health of a tree was recorded on a scale of 0 to 4 separately for the upper and lower crown, with 0 indicating death of that layer, and 4 indicating no visible dieback. This was a direct measure of tree health, and was compared to the following to assess their suitability as health indicators.

Crown, or foliage, density was measured using a scale provided by Manaaki Whenua - Landcare Research (fig 3.2 in Ch 2). The proportion of dead terminal shoots was recorded on a scale from 0 (none) through 1 (<10%), 2 (11-30%), 3 (31-60%), 4 (>60%) to 5 (all dead). The amounts of flowers and of seed pods were recorded separately on a scale of 0 to 4, relating to none, rare, occasional, common and abundant. Adventitious sprouts, or epicormic shoots, often develop on the trunks of trees that have sustained branch dieback (Sinclair & Hudler, 1988). The amount of epicormic shoots on each kamahi was recorded as 0 if none, 1 if a few (less than five), and 2 if there are many.

Potential causes of dieback

The extent of scratching on the trunk, typical of possums climbing, was scored from 0 (none) to 3, where the bark is worn by a run, with 1 being less than three scratches, and 2 being more. Scraping on the trunk typically caused by possum gnawing was scored from 0 (none) to 2, meaning bark is heavily stripped.

The amount of pinhole borer holes on the trunk of each tree was scored as 0 (none), 1 (rare), 2 (occasional), 3 (common) or 4 (more than 15 holes 100 cm^{-2}).

Damage to the foliage, by possum and insect browsing, and wilting, is recorded on a linear scale from 0 to 4, corresponding to the percent affected (0=none, 1<25%, 2=25 to 50%, 3=50 to 75%, 4>75%) separately for the upper and lower crown. Twig breakage, referring to loss of part of a twig, was scored on the same scale.

ANALYSIS

Univariate statistics of data were calculated by SAS (SAS Institute, 1995), correlation coefficients calculated using SYSTAT (Systat Inc, 1992). Principal components were analysed by JMP (SAS Institute, 1994) using correlations.

Site descriptors

Much of the site data could be analysed without modification, although some variables were converted to more meaningful scores (table 3.1). Aspect is divided into north-

Table 3.1 *List of site variables analysed. Explanation of dieback variables in text. Where no derivation is included, variable is analysed untransformed.*

Variable	Derivation
Altitude (m asl)	
Slope (°)	
North-South	cosine(aspect)
East-West	sine(aspect)
Exposure	
Canopy closure	
Drainage	
Canopy height (m)	
Kamahi density (per 100 m^{-2})	
Ground cover (%)	
Canopy cover (%)	
Seedlings	sum of 5 sub-plots
Small saplings	sum of 5 sub-plots
Large saplings	sum of 5 sub-plots
Possum pellets	sum of 5 sub-plots
Mean dieback	(sum dieback)/(number of trees)
Unhealthy trees	proportion of trees with dieback 4-5
Dead trees	proportion of trees with dieback 5

south and east-west components by taking the cosine and the sine of aspect respectively. The number of seedlings for each sub-plot within a site is pooled to give one total per site, as are data for small and large saplings, and possum pellets. A mean dieback value per site is obtained by meaning the dieback scores for individual trees (see section below). The proportion of trees belonging to unhealthy dieback classes (*i.e.* with a score of 4 or 5 as described below) is the 'unhealthy trees' variable, while 'dead trees' is the proportion of dead kamahi in the site.

Relationships between all site characters, and between each of the dieback variables and each site character were analysed by correlations.

Principal components of site character variables were extracted, for all variables excluding those relating to the amount of kamahi dieback at the site (so components are not influenced by observed levels of dieback at each site). Correlations were then calculated between each of the four main components and the dieback variables, to determine how well the measured site characters predict dieback.

Tree data

Many of the variables analysed were unmodified from those measured. Others are combinations or functions of the original data (table 3.2). The health scores for upper and lower crown were summed. They were then transformed to dieback class with a health score of 8 corresponding to dieback score of 0, health of 6 or 7 to dieback of 1, health of 3, 4 or 5 to dieback 2, health 1 or 2 to dieback 3, and 0 health to dieback 4. Reproduction is the greater of the values for flower or for seedpod scores, to account for variation in time of sampling. Trunk damage is the sum of scratch and scrape

Table 3.2 *List of tree variables analysed. Where no derivation is included, variable is analysed untransformed.*

	Variable	Derivation
Tree character	DBH (cm)	
	Height (m)	
	Canopy status	
	Stems	
Dieback	Dieback	
Dieback indicators	Dead terminal shoots	
	Crown density (%)	
	Reproduction	maximum value from flowers and seedpods
	Epicormics	
Damage to Trunk	Trunk damage	sum of scratch + scrape scores
	Borer	
Damage to Foliage	Possum	sum of possum damage scores for upper and lower canopy
	Insect	sum of insect damage scores for upper and lower canopy
	Wilt	sum of wilt scores for upper and lower canopy
	Breakage	

scores. For each of possum damage and insect damage, and wilt, variables are the sum of the scores for the top and lower crown.

Correlations between each of the tree variables were calculated with all trees included, only live trees, and only dead trees. This distinguishes between the relationships that may be driven by dead trees, which tended to have very different scores on some variables. Of particular interest were the correlations between dieback and each of the possible indicators of dieback (dead shoots, crown density, reproduction and epicormics). Also of interest are the relationship between dieback and all damage measures, and between dieback and the four tree character scores.

The principal components of all variables, excluding dieback and dead terminal shoots, were analysed for all trees with scores for all variables. Eight dead trees had to be omitted as they had no foliage to score for foliage damage types. This was repeated on data containing live trees only, and dead trees only. Dieback and dead terminal shoots scores were omitted to allow comparison between these and the principal components at a later stage. When analysing the dead trees, foliage damage scores (possum and insect browse, and wilt) were excluded, as most dead trees had no foliage, although those that did were scored for foliage damage. Correlations between the first four components and dieback class and dead terminal shoots were calculated to assess how well the measured variables predict level of dieback of a tree.

RESULTS

SITES

Sites range in altitude, from 650 to 1000 m above sea level (tab 4.1), though most are below 850 m asl. Slope varied widely between sites, with a minimum of 0° and a maximum of 40°, but most were on a light to medium slope (<20°); sites tended to face south and west. Exposure and canopy closure tended to be 'average', while drainage was generally very good. Canopy height ranged from 4 m to 16 m, but tended to be between 11 and 14; kamahi density was usually close to 10 trees per 100 m^2. Ground cover varied greatly between sites, from 5 % to 95 %, and with a consequently large standard deviation, whereas canopy cover tended to be higher, ranging from 30 % to 90 %, but usually between 60 % and 70 %. The average number of seedlings per site is strongly skewed by a few sites with many seedlings, there was generally less than 20 per site (median=4.5); likewise the mean numbers of each size of saplings per site have large standard deviations. The mode for possum pellets per site was 0, but again, this was highly variable. The mean amount of dieback of each tree per site was close to 1, which is very little dieback; at one site where 4 dead trees were present, the mean was 2.3, indicating medium to high levels of dieback. There was on average less than one dead kamahi tree per ten trees, with the maximum being four; when very

Table 4.1 *Distribution of site data. Mean and standard deviation to four decimal places. * Denotes average is median rather than mean.*

Variable	N	Average	Std deviation	Minimum	Maximum
Altitude (m asl)	30	820	83.3	650	1000
Slope (°)	30	11	10	0	40
N-S	30	-0.1580	0.7286	-1	1
E-W	30	-0.1717	0.6798	-1	1
Exposure	30	1*	0.64	0	2
Canopy closure	30	1*	0.56	0	2
Drainage	30	1*	0.93	1	5
Canopy height (m)	30	13	2.78	4	16
Kamahi density (100 m^{-2})	30	9.3	1.9	4	13
Ground cover (%)	30	52	32	5	95
Canopy cover (%)	30	66	15	30	90
Seedlings	30	40.2	94	0	488
Small saplings	30	3.1	5	0	20
Large saplings	30	1.0	1.8	0	9
Possum pellets	30	0.6	1.5	0	7
Mean dieback	30	1.2	0.56	0.2	2.3
Dead kamahi	30	0.04	0.097	0	0.4
Dying kamahi	30	0.065	0.120	0	0.4

Relationship	*r*-value	*P*-value
Canopy cover * ground cover	-0.5137	0.0037
Small saplings * ground cover	0.5628	0.0012
Dead kamahi * small saplings	0.5966	0.0005

Table 4.2 *Significant correlations between site variables (*r>*0.5,* P<*0.005, 29 d.f.).*

unhealthy trees are added, the mean is slightly higher, but still less than one to ten trees, and with the same maximum of four.

Of the correlations tested (Appendix B), ground cover was negatively related to canopy cover, and positively to the index of small saplings, which was the only variable significantly correlated to dead kamahi (table 4.2). Excluding site 20, the outlier (fig 4.1), this relationship is still marginally significant (*r*=0.43, *P*=0.02); if also excluding site 19, the relationship is not significant (*P*=0.7). Most sites have no dead kamahi trees (24/30), and half of those also have no small saplings (12/24). Of the six sites with any dead kamahi, three have no small saplings.

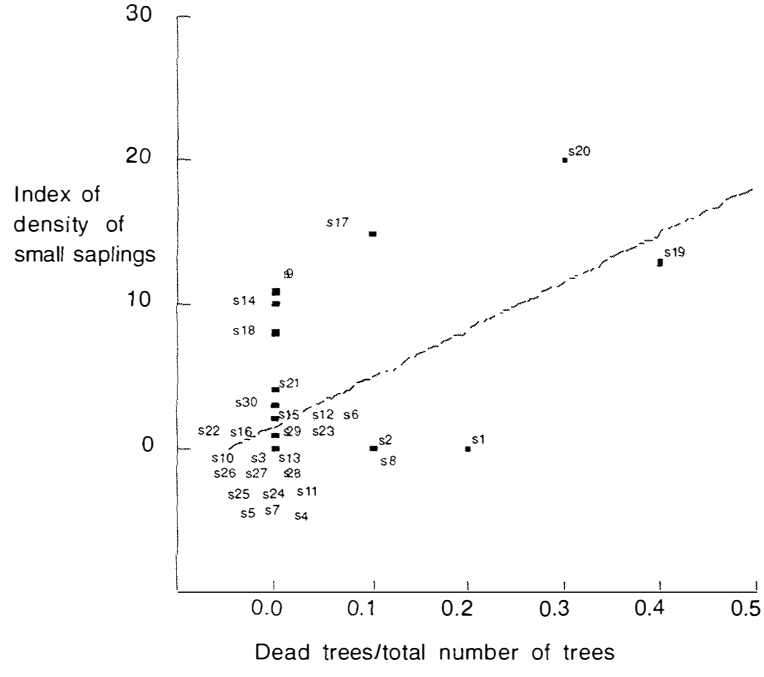


Fig 4.1 *Relationship between the proportion of dead kamahi trees at each site, and the number of small saplings counted in sub-plots at that site.* r=*0.58,* P =*0.0005.*

The two longest principal components of the site character analysis explained 17.88 % of the data variance on the first component and 17.30 % on the second (fig 4.2). The first component is most influenced by the variables ground cover and small saplings in a positive direction and canopy cover in a negative. The second component is most influenced by canopy height, E-W aspect, exposure and density, all positively. The third longest component explains a further 15.19 % of the variance, and is most influenced by drainage (negative), and canopy closure and slope (positive). No other component explains more than 10 % additional variance.

Component 1 is weakly correlated to mean dieback (r=0.38, P=0.04) (fig 4.3). No other correlations between dieback variables and the three longest principal components are statistically significant.

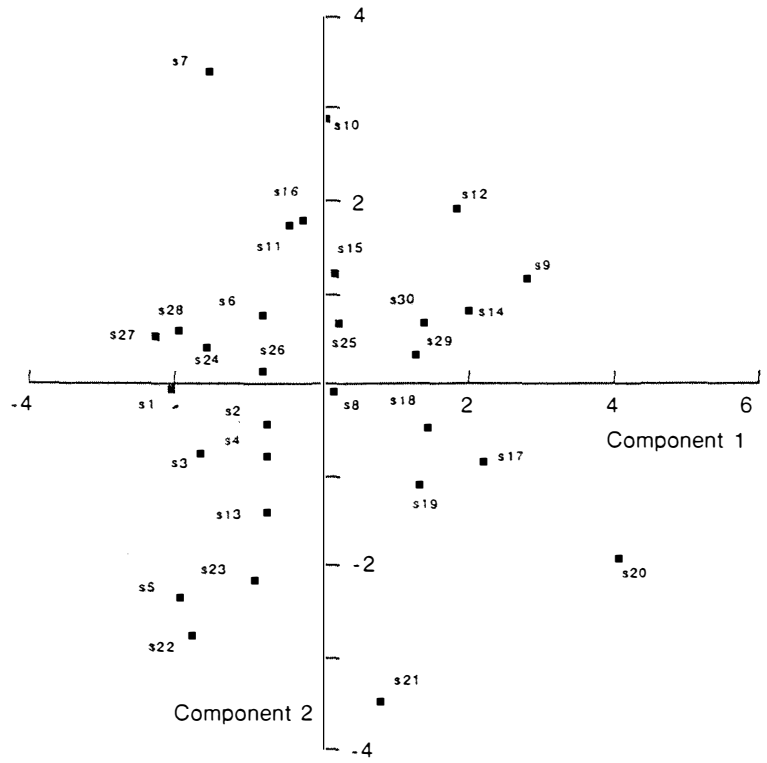


Fig 4.2 *Location of sites on first two principal components of site character data. Component 1 explains 17.9 % of variance, component 2 a further 17.3 %.*

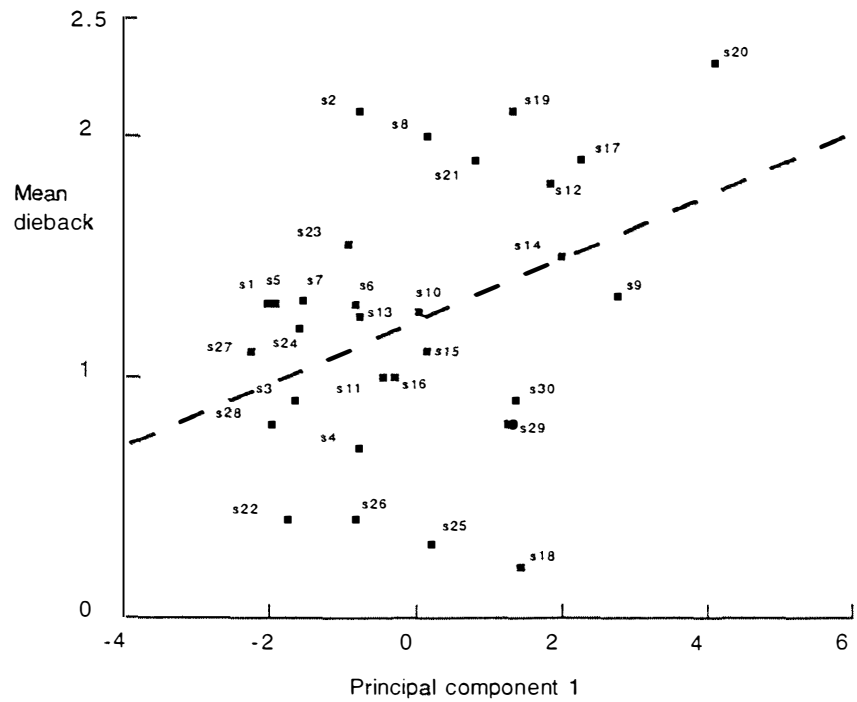


Fig 4.3 *Relationship between sites' mean dieback and score on first principal component of site character data. Dashed line is linear regression (r=0.38, P=0.04).*

TREES

When live and dead trees are compared, many scores are quite different (table 4.3). Although dead trees have a larger mean girth, they have a much lower maximum girth than live trees. The number of stems, height and canopy status of both sets are

Table 4.3 *Distribution of tree variables. ˙ Denotes average is median rather than mean.*

	Variable	N	Average	Standard deviation	Minimum	Maximum
All trees	DBH	305	25.92	12.807	10	78.6
	Stems	305	1.4	0.86	1	7
	Height	305	11	2.8	4	19
	Status	305	1˙	0.4	0	1
	Dieback	305	1˙	0.9	0	4
	Dead shoots	305	2˙	0.9	0	5
	Crown density	305	62	19.9	0	95
	Reproduction	305	2˙	1.1	0	4
	Epicormics	305	0˙	0.7	0	2
	Trunk damage	305	0˙	0.7	0	4
	Borer	305	0˙	0.9	0	4
	Possum browse	296	0˙	0.7	0	4
	Insect browse	296	2˙	0.5	0	3
	Foliage wilt	296	1˙	1.0	0	8
	Breakage	305	1˙	0.5	0	4
Live trees	DBH	293	25.70	12.859	10	79
	Stems	293	1.4	0.87	1	7
	Height	293	11	2.8	4	19
	Status	293	1˙	0.5	0	1
	Dieback	293	1˙	0.7	0	3
	Dead shoots	293	2˙	0.7	0	4
	Crown density	293	64	17.1	15	95
	Reproduction	293	2˙	1.1	0	4
	Epicormics	293	0˙	0.7	0	2
	Trunk damage	293	0˙	0.7	0	4
	Borer	293	0˙	0.7	0	4
	Possum browse	293	0˙	0.7	0	4
	Insect browse	293	2˙	0.5	0	3
	Foliage wilt	293	1˙	0.9	0	8
	Breakage	293	1˙	0.3	0	2
Dead trees	DBH	12	31.25	10.567	10	47
	Stems	12	1.2	0.58	1	3
	Height	12	11	3.9	4	15
	Status	12	1˙	0.5	0	1
	Dead shoots	12	5˙	0.5	4	5
	Crown density	12	9	6.8	0	15
	Reproduction	12	0˙	0.3	0	1
	Epicormics	12	0˙	0.3	0	1
	Trunk damage	12	0.5˙	0.5	0	1
	Borer	12	3˙	1.1	1	4
	Possum browse	3	0	0	0	0
	Insect browse	3	0	0	0	0
	Foliage wilt	3	4˙	2.3	4	8
	Breakage	12	3˙	0.9	1	4

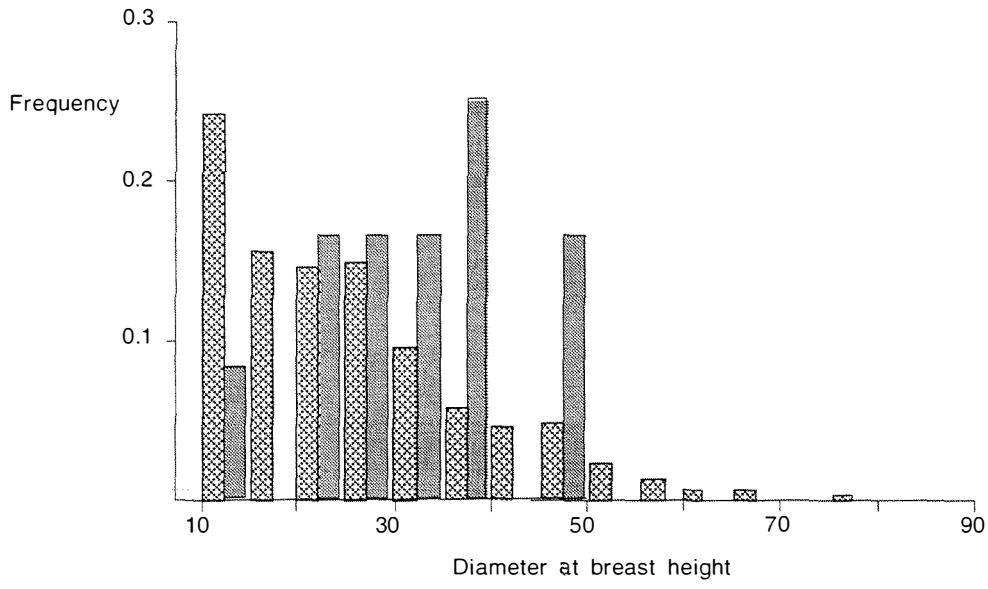


Fig 4.4 *Frequency distributions of live and dead trees; hatched bars represent live trees (N=293), grey bars are dead trees (N=12).*

comparable. As would be expected, dead trees had more dead terminal shoots, and lower crown densities than live trees. Live trees tended to have low to medium levels of reproduction, while dead trees had very little or no reproductive effort. There was much more variation in the amount of trunk damage on live trees, with dead trees scoring only low values. Pinhole borer were much more common on dead trees than live. Only three dead trees had enough foliage to receive a meaningful score for possum and insect browse (all scored none for both) and foliage wilt, which ranged from 4 to 8, and had a correspondingly high standard variation. Twig breakage tended to be much more common on dead trees than live.

There are many more significant correlations between variables when all trees are included than in either live trees or dead trees (table 4.4). The few dead trees are having a large effect on these relationships, as shown when live trees are analysed by themselves (Appendix C, D & E). When dead trees are analysed by themselves sample sizes are too small for the relationships present to be significant. All sets share the correlation between height and diameter, and both sets where dieback can be included have a strong relationship of both crown density and dead terminal shoots with dieback. In addition, the dead trees show a relationship between trunk damage and both borer and diameter.

Table 4.4 *Correlations of tree variables. All tree and live tree relationships were considered significant only if* r>*0.5 regardless of* P *value. Dead tree correlates were included if* P<*0.05. This excluded most variables with sample=3.*

	Relationship	*r*-value	N
All trees	Height * DBH	0.5438	305
	Crown density * dieback	-0.6867	305
	Borer * dieback	0.5474	305
	Borer * crown density	-0.5051	305
	Breakage * dieback	0.5888	305
	Breakage * crown density	-0.5663	305
	Breakage * borer	0.5711	305
	Dead terminal shoots * dieback	0.7907	305
	Dead terminal shoots * crown density	-0.6408	305
	Dead terminal shoots * borer	0.5385	305
	Dead terminal shoots * breakage	0.6401	305
Live trees	Height * DBH	0.5361	293
	Crown density * dieback	-0.5383	293
	Dead terminal shoots * dieback	0.6716	293
Dead trees	Height * DBH	0.8664	12
	Trunk damage * DBH	0.6256	12
	Borer * trunk damage	-0.6247	12
	Wilt * status	-1	3

Level of dieback exhibited bears little relation to amount of possum browse observed (fig 4.5). Dieback also occurs indiscriminately of DBH (fig 4.6). But presence of pinhole borer relates to high levels of dieback (r=0.55) (fig 4.7). Note when there is no dieback (dieback=0), there is never more than very few borer holes; and, although all dead trees (dieback=4) had pinhole borer present, most of the dying trees (dieback=3) did not.

Principal component 1 of the data containing all trees except the eight dead trees omitted due to missing data (fig 4.8a), is most influenced by pinhole borer, breakage and DBH (positive relationship), and crown density (negative), and explains 18.3 % of the variance. Component 2 is most influenced by height, DBH, canopy status and reproduction (positive), and explains a further 14.2 % of the variance. Trees with little to no dieback (dieback < 2) tend to be to the left of zero, and have similar distributions (fig 4.9a). Trees with medium dieback (dieback = 2) are positioned rather evenly over both axes (fig 4.9a,b), with some to the far right. Unhealthy trees (dieback = 3) tend to be in the lower centre-right, and dead trees (dieback = 4) are positioned in the bottom right of the graph.

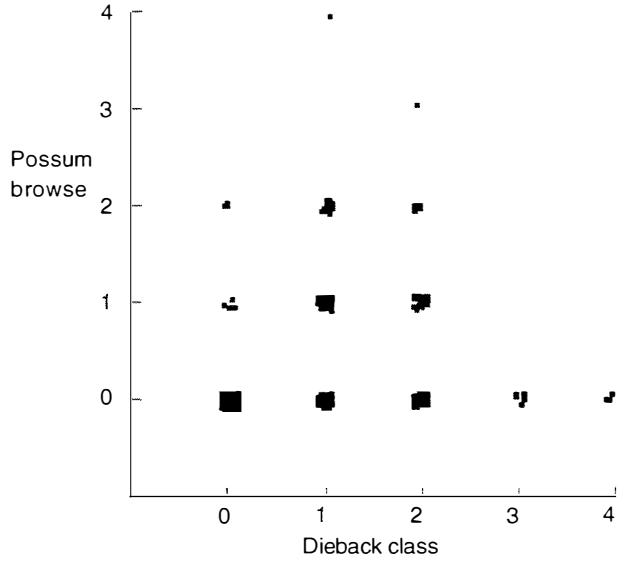
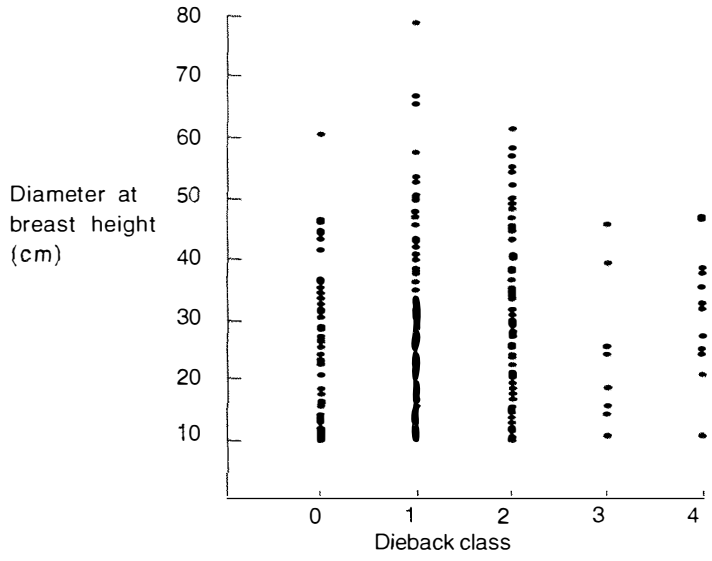


Figure 4.5
Relationship between observed level of possum browse (0=none, 4>75%) and dieback class (0=no dieback, 4=dead tree) of trees with foliage (n=296; r=0.025). Multiple data points are jittered to indicate frequency.

Figure 4.6
Diameter at breast height of trees in each dieback class. N=305; 0 relates to no dieback, 4 to a dead tree (r=0.12).



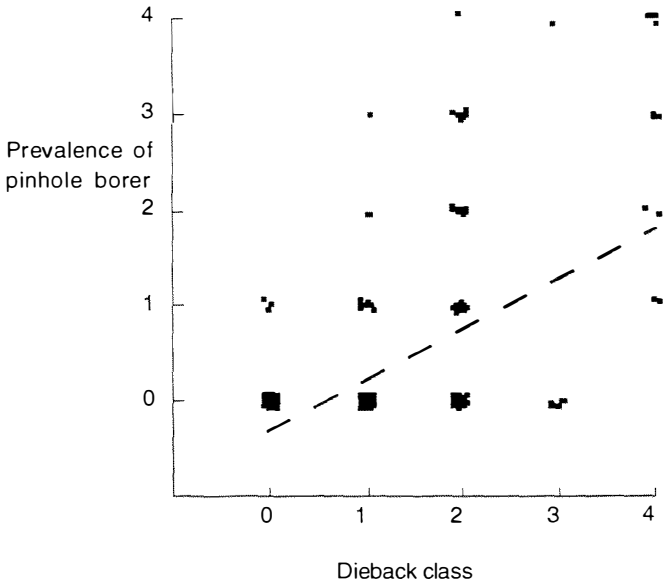


Figure 4.7
Relationship between pinhole borer presence (0=absent, 4=>15 holes/cm^2) and dieback class (0=none, 4=dead tree), N=305. Dashed line corresponds to linear regression (r=0.55), data points 'jittered' to indicate frequency.

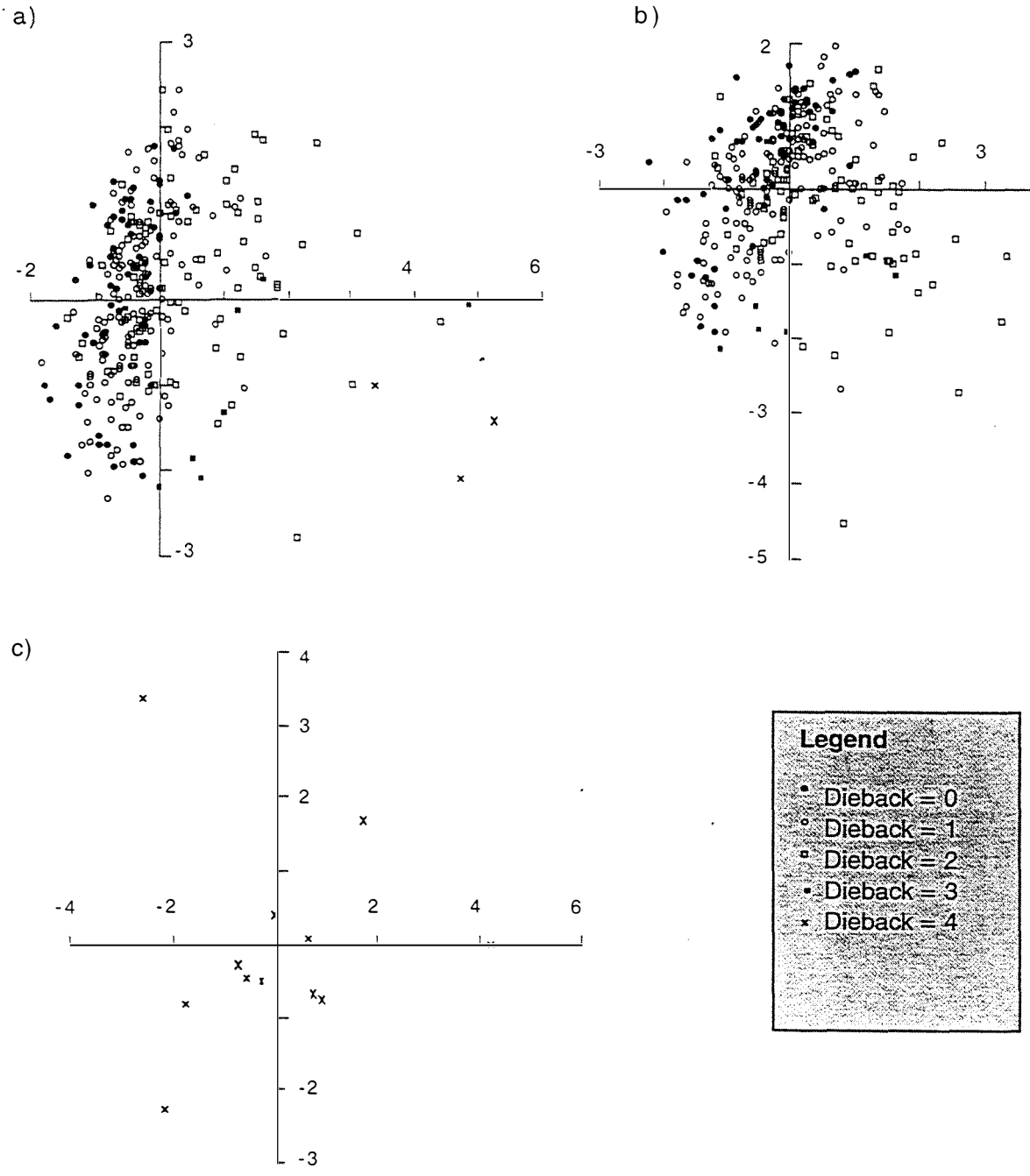


Figure 4.8 *Location of trees on first two principal components of tree data. a) includes all trees and explains 18.3 % of the variance on the first axis and a further 14.2 % on the second. Eight dead trees are not included due to missing data. b) includes only live trees and explains 17.4 % of variance on the first axis, and 12.8 % on the second. c) includes only dead trees and excludes variables 'possum', 'insect' and 'wilt'. The first axis explains 35.1 % of variance, the second 19.8 %.*

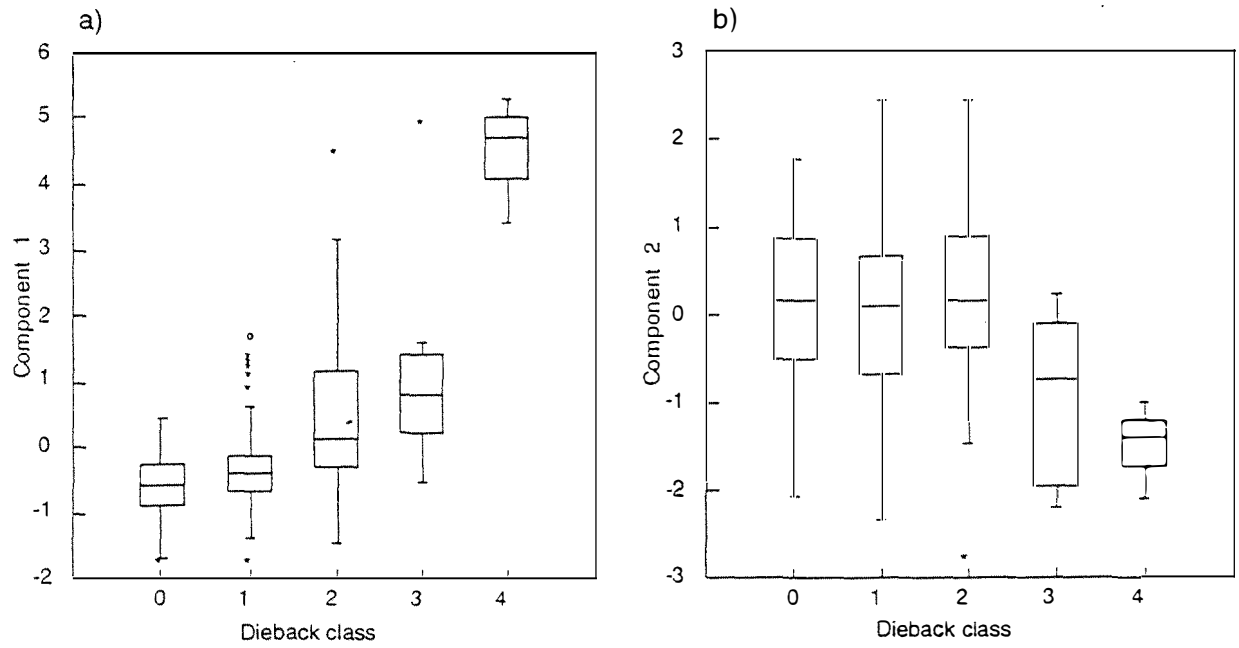


Fig 4.9 *Box plots of position of each dieback class on principal components 1 and 2 of all trees. Edges of boxes correspond to upper and lower quartiles (interior of box = Hspread), horizontal line represents median value, whiskers represent range of values within 1.5 Hspreads of box edge. Stars represent outliers, circles extreme outliers (outside 3 Hspreads of box edge). a) is principal component 1, b) component 2.*

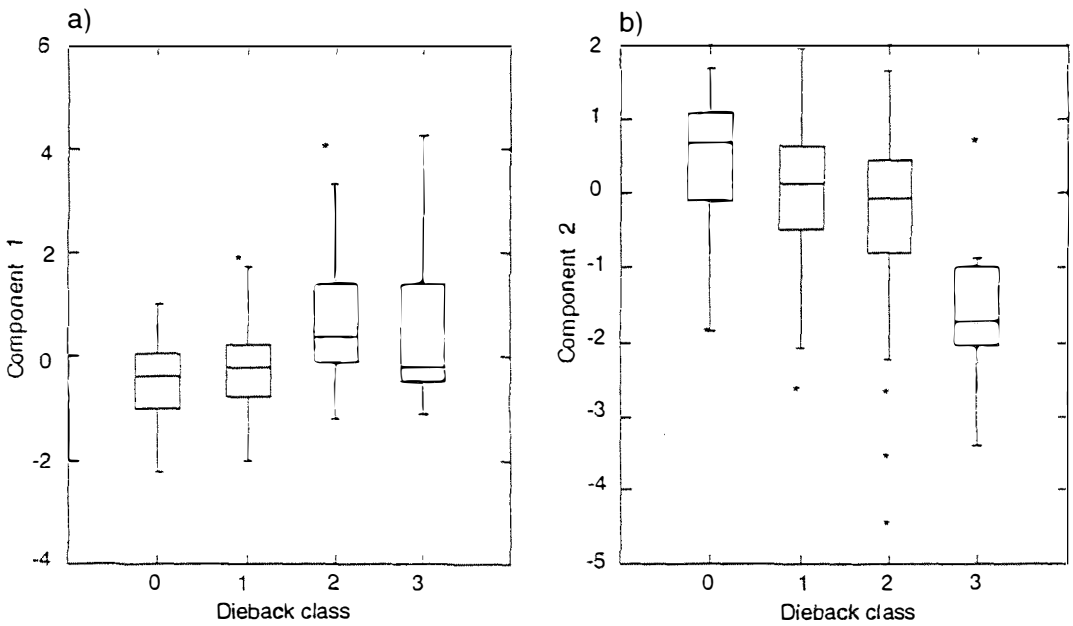


Fig 4.10 *Box plots of position of each dieback class on principal components 1 and 2 of live trees only. Edges of boxes correspond to upper and lower quartiles (interior of box = Hspread), horizontal line represents median value, whiskers represent range of values within 1.5 Hspreads of box edge. Stars represent outliers, circles extreme outliers (outside 3 Hspreads of box edge). a) is principal component 1, b) component 2.*

The first component of the analysis of live trees (fig 4.8b), explaining 17.4 % variance, is strongly influenced by DBH, and also by pinhole borer, height, breakage and canopy status (all positive). The second component is most influenced by reproduction, status and crown density (positive). Trees with little to no dieback (dieback<1) tend to be in the upper-left half of the graph, with high scores on component 2, and relatively low scores on component 1 (fig 4.10), whereas trees with medium dieback (dieback=2) stretch the graph down, and tend to be to the right of zero. Unhealthy trees (dieback=3) are all in the lower portion of the graph, and close to, or right of, zero.

In the analysis of dead trees, the variates possum and insect browse, and wilt are excluded due to missing data. Component 1 (fig 4.8c) explains 35.1 % of variance, and is most influenced by trunk damage, DBH and height (all negative). Component 2 explains 19.8 % further variance and is strongly influenced by breakage and number of stems (positive).

When principal components are compared to the dieback scores, only component 1 from the set with both live and dead trees is related to dieback (r=0.5398) (fig 4.11). Live trees (dieback<4) do not achieve significant correlations between dieback and the longest components.

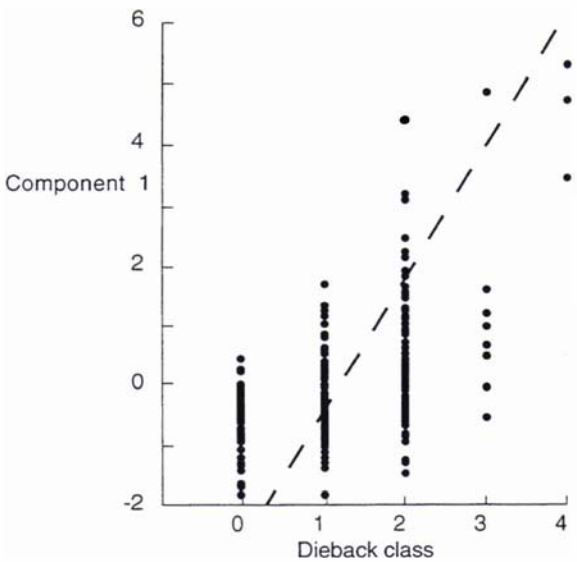


Fig 4.11 *Relationship between dieback class (0=no dieback, 4=dead) and principal component 1 from all trees (r=0.54, N=296).*

DISCUSSION

Kamahi dieback in Tongariro National Park, its possible causes, and implications, is a contentious public issue. The survey of sites investigated factors that may predispose a stand to dieback, such as exposure and drainage. Sites covered a wide range of kamahi habitats, including its upper and lower altitudinal limits in the Park, a range of slopes and aspects, high and low exposure and canopy closure levels, and with good and poor drainage. Mean scores for dieback of all the trees at each site were compared to the site factors to determine their effect. Any significant correlations between these site factors and level of dieback would indicate a possible predisposing cause, such as, for example, range contraction if sites at high altitudes are experiencing high levels of dieback. Dieback scores were also compared to measures of factors that may cause dieback in each tree, indicating both the frequency at which dieback is occurring, and possible correlates of dieback. The latter may serve as predictors for more rapid survey and monitoring techniques.

SITE RELATIONSHIPS TO DIEBACK

None of the site factors suggested as possible causes of dieback, *i.e.* exposure, canopy closure or drainage, which may indicate susceptibility to drought stress, predicted level of dieback. Altitude, slope and aspect, which measure some of the ecological range of kamahi, fail to indicate any pattern. Density of kamahi, which was one measure of kamahi dominance in the canopy, was also unrelated to level of dieback.

There is no evidence from this study supporting lack of canopy closure or range contraction as explanations for kamahi dieback in the Park.

It is extremely hard to estimate possum population size and density reliably from pellet counts, partly due to different disappearance rates of pellets (Batcheler, Darwin & Pracy, 1967). Number of possum pellets was therefore not expected to relate closely to dieback, and doesn't. Also, if possum populations have reached a peak and had time to decline, low possum numbers may currently be found in stands destroyed by recent (*i.e.* 10-15 years before present) browsing (Pekelharing, 1979).

The number of small saplings (height < 2m) counted at each site, one measure of reproduction, is positively correlated to the proportion of kamahi trees that are dead at that site. Small saplings are present at many sites with no dead kamahi, but tend to occur in higher numbers where there are dead trees. This relationship is largely dependent on the two sites with greater than 30 % of kamahi dead where there are

many small saplings; omitting these sites from the analysis excludes 1/3 of sites with any dead trees, and all sites with more than 20 % of trees dead. Not surprisingly, the relationship is no longer statistically significant. None of the other measures of reproduction at each site (*i.e.* seedlings and larger saplings) were significantly related to any measures of kamahi health. It may be that seedlings are present regardless of canopy health, but will only reach sapling stage (>50 cm height in this study) if released by a thinning canopy. This largely agrees with the conclusion of Ogden (1991) that kamahi is well suited to establish quickly after a gap in the canopy forms. The lack of relationship at a larger sapling stage (> 2m in height) may be due to the length of time to attain this height, allowing the canopy to close.

When the principal components of the site data are analysed, sites do not cluster according to level of dieback. There is only a weak relationship between mean dieback at a site and principal component 1, which is due to the effect of small saplings on this axis. None of the site variables measured are capable of predicting patterns of kamahi dieback in the study area.

TREE RELATIONSHIPS TO DIEBACK

Results from tree data show similar patterns: none of the suggested causal factors seem to be impacting on the level of dieback in trees in any predictive way. Also, of the secondary, or indirect, measures of health (crown density, reproduction and epicormic growth), only crown density was found to predict dieback class well. The only studies relating these factors to tree health are from relatively unrelated species, such as heavy epicormic growth observed in stressed rata (Brockie, 1992), and high levels of reproduction in cabbage tree before death (Simpson, 1993).

The level of possum browse observed in foliage was not found to relate to the health (by any measure) of a tree. As discussed previously, this may be due to lack of persistence of damaged leaves, although little possum sign was observed anywhere in the Park during this study. Further, in the Orongorongo Valley, Fitzgerald (1976) noted that kamahi buds and flowers disappear soon after their production, and suggested possums to be responsible; in Tongariro National Park, seedpods from flowers persist until the following year (pers.obs.). If Fitzgerald's results are widely applicable, this implies low possum density. Trunk damage was also unrelated to level of dieback. This study provides no evidence to suggest possums are killing kamahi in the areas studied.

There are significant correlations between presence of pinhole borer, the indirect measure of *Sporothrix* presence in a tree, and both dieback class and dead

terminal shoots, the two most direct measures of tree health. This could indicate that *Sporothrix* is adversely affecting tree health, particularly as very few healthy trees (dieback<2) have any borer holes present. However, when dead trees, usually with many holes, are removed from the analysis, the correlation is no longer significant; also only one tree near death (dieback=3) had holes present. This suggests pinhole borer are tending to attack trees in poorer health, rather than causing the reduction in health, and agrees with the conclusions of Hosking (1993b; 1993a).

Diameter at Breast Height (DBH) of a tree was found to be unrelated to the level of dieback. The twelve dead trees were of a range of diameters, with none very large (greater than 50 cm) and few small (less than 20 cm). These trees have not died because they are old. This result seems to fit the natural population dynamics model, suggested by Harcombe (1987), that 'natural' tree death occurs independently of age and describes a reverse J-curve for tree mortality. Coleman (1980) also found live and dead kamahi in Westland had similar age distributions, concluding that age was not a factor in kamahi death.

Evidence further suggesting that the observed dieback may be due to natural causes is the frequency at which it was observed. Of the 305 trees included in this study, 12, or approximately 4 %, were standing dead; a further 8, or 2.7 % of live trees, were near death. Trees that did not remain standing after death are not included in this study. Assuming the true mortality rate is somewhere near (probably between) these values, and a canopy persistence of around twenty years for standing dead trees, this mortality does not appear to be excessive (Rose, Pekelharing & Platt, 1992), particularly as kamahi may live for up to 400 years (Lusk, 1989). Data from this study are inappropriate for speculation on expected regeneration, growth or death rates, as this requires longer-term studies (Harcombe, 1987). It is an area largely neglected for non-economic species, such as kamahi, which is why research is currently lacking.

CONCLUSION

From these results there is no evidence with which to discard the hypothesis that kamahi in Tongariro National Park is in a healthy state, and that any observed mortality is as a result of natural causes. Results from an earlier study on Hauhungatahi (Druitt, 1985) also found that kamahi was in a healthy state based on size-class distribution. Kamahi is the most common canopy species in the areas studied, and the reported widespread decline may be a product of perception, with all dead trees presumed to be kamahi, and no consideration given to the number of healthy kamahi. This has also been suggested for Westland, where kamahi is a dominant species and possums were again the main suspects (Coleman *et al*., 1980). Coleman *et al*. (1980) suggest that although many kamahi trees are dying, they may not make up a significant proportion of this very prevalent species. Much of the observed dieback in Tongariro National Park that can be confidently identified as being kamahi, seems to be localised, particularly along forest margins (refer figs 5.1 and 5.2 of Ch 2b) which are often near public roads. Even areas that appear from the outside to be completely dead, from inside there may be few dead trees: site 8 of this survey (on the ridge north of Erua) is a patch of kamahi that has been used an example of the high level of kamahi dieback occurring in the Park; however, no dead or dying kamahi were recorded there in this survey, and regeneration was extremely high. This patch is clearly getting shorter (*Pers. obs*.), but doesn't appear to be dying (for kamahi at least). This may mean that kamahi dieback is both unimportant for the forest in general, and deceptively apparent.

Another possibility is this study simply failed to find evidence because tree decline is a complex problem. A single visit to a site provides no indication of the direction of change in crown health (Innes, 1993); any tree may be deteriorating to death, or recovering. Identifying causes after death has occurred is often impossible (Clarkson, 1993); this is why trees in a range of health states were studied, in an attempt to find significant correlations between health and any causal factor. However, correlation does not imply causation. Positive identification of causes of tree death requires repeat sampling and/or experimentation.

With the current lack of knowledge of 'normal' population dynamics, it is very difficult to distinguish between these two possibilities: that kamahi dieback in Tongariro National Park occurs on a small scale, and at a natural rate, and is due to largely natural, but complex causes; or that this study failed to find evidence of kamahi

decline and its causes due to the complexity of the problem, and the necessarily small-scale short-term approach taken.

This survey did find patches of high mortality that warrant further investigation into causes of death, which has been undertaken in the second survey (chapter 2b).

REFERENCES

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APPENDIX A

List of site locations for survey; distance from forest edge and altitude are approximations (grid references from Infomap 262-6).

Site	Location	Approximate grid reference	Distance from forest edge (m)	Altitude
1	South of Erua	717216	40	800
2	South of Erua	717216	300	850
3	South of Erua	717216	550	900
4	South of Erua	717216	800	950
5	South of Erua	718216	1050	1000
6	Te Ponanga Saddle	747240	150	700
7	Te Ponanga Saddle	747240	400	700
8	North of Erua	717217	40	800
9	Makatote Gorge (N ridge)	718213	80	820
10	Te Ponanga Saddle	747240	650	670
11	Makatote Gorge (N ridge)	718214	400	850
12	Makatote Gorge (N ridge)	719214	650	850
13	Makatote Gorge (N ridge)	720214	2500	900
14	Makatote Gorge (N ridge)	720214	2000	900
15	Makatote Gorge (N ridge)	719214	1500	850
16	Makatote Gorge (N ridge)	719214	1000	850
17	North of Manganuiateao	716211	15	800
18	North of Manganuiateao	716211	250	840
19	North of Manganuiateao	717211	500	860
20	North of Manganuiateao	717211	750	880
21	North of Manganuiateao	717211	1000	890
22	Pokaka	716210	15	820
23	Pokaka	716210	300	840
24	Kiko Road	769238	500	760
25	Kiko Road	770238	1000	760
26	Kiko Road	770238	1200	860
27	Kiko Road	770238	1000	780
28	Kiko Road	769238	700	820
29	Kiko Road	769238	500	660
30	Kiko Road	769238	800	650

APPENDIX B

Correlation matrix of site variables analysed. See text for explanation of variables.

alt = altitude

slp = slope

ns = North-South aspect

ew = East-West aspect

exp = exposure

cls = canopy closure

drg = drainage

cht = canopy height

dsty = density of kamahi

GdC = ground cover

CyC = canopy cover

Sds = seedlings

SmlS = small saplings

LrgS = large saplings

PP = possum pellets

xdbk = mean dieback

DdWr = proportion of dead kamahi

DgWr = proportion of dead and dying kamahi

Correlations

Variable	alt	slp	ns	ew	exp	cls	drg	cht	dsty	GdC	CyC	Sds	SmlS	LrgS	PP	xdbk	DdWr	DgWr
alt	1.000	0.062	-0.055	-0.492	0.060	0.092	0.114	-0.302	-0.362	-0.135	-0.044	0.014	0.147	-0.035	0.280	0.147	0.122	0.126
slp	0.062	1.000	0.064	0.000	0.363	0.184	-0.403	0.206	-0.148	-0.329	-0.110	-0.060	-0.356	-0.350	0.134	-0.083	-0.041	-0.020
ns	-0.055	0.064	1.000	0.054	-0.315	0.061	-0.066	-0.138	-0.245	-0.165	0.165	-0.138	0.108	-0.184	0.110	-0.167	0.297	0.231
ew	-0.492	0.000	0.054	1.000	0.247	-0.185	-0.009	0.403	0.328	0 023	-0.052	-0.213	-0.254	0.019	-0.214	-0.192	-0.264	-0.272
exp	0.060	0.363	-0.315	0.247	1.000	-0.090	-0.335	0.457	0.258	-0.078	-0.075	-0.054	-0.313	0.068	0.065	-0.106	-0.267	-0.149
cls	0.092	0.184	0.061	-0.185	-0.090	1.000	-0.373	-0.096	-0.262	0.398	-0.492	-0.152	0.250	0.117	0.108	0.018	0.090	0.137
drg	0.114	-0.403	-0.066	-0.009	-0.335	-0.373	1.000	-0.165	0.015	-0.200	0.428	-0.062	0.037	0.302	-0.079	0.035	0.122	0.006
cht	-0.302	0.206	-0.138	0.403	0.457	-0.096	-0.165	1.000	0.262	0.060	-0.184	-0.219	-0.123	0.092	0.061	-0.249	-0.095	-0.172
dsty	-0.362	-0.148	-0.245	0.328	0.258	-0.262	0.015	0.262	1.000	0.409	-0.103	0.087	0.155	-0.073	-0.274	0.065	0.034	-0.078
GdC	-0.135	-0.329	-0.165	0.023	-0.078	0.398	-0.200	0.060	0.409	1.000	-0.514	0.098	0.563	0.236	-0.109	0.177	0.208	0.188
CyC	-0.044	-0.110	0.165	-0.052	-0.075	-0.492	0.428	-0.184	-0.103	-0.514	1.000	-0.162	-0.363	0.031	0.092	-0.346	-0.263	-0.225
Sds	0.014	-0.060	-0.138	-0.213	-0.054	-0.152	-0.062	-0.219	0.087	0.098	-0.162	1.000	0.263	0.109	-0.112	0.421	0.300	0.202
SmlS	0.147	-0.356	0.108	-0.254	-0.313	0.250	0 037	-0.123	0.155	0.563	-0.363	0.263	1.000	0.346	-0.105	0.459	0.597	0.410
LrgS	-0.035	-0.350	-0.184	0.019	0.068	0.117	0 302	0.092	-0.073	0 236	0.031	0.109	0.346	1.000	-0.213	0.055	-0.147	-0.147
PP	0.280	0.134	0.110	-0.214	0.065	0.108	-0.079	0.061	-0.274	-0.109	0.092	-0.112	-0.105	-0.213	1.000	0.121	0.209	0.121
xdbk	0.147	-0.083	-0.167	-0.192	-0.106	0.018	0.035	-0.249	0.065	0.177	-0.346	0.421	0.459	0.055	0.121	1.000	0.582	0.709
DdWr	0.122	-0.041	0.297	-0.264	-0.267	0.090	0.122	-0.095	0.034	0.208	-0.263	0.300	0.597	-0.147	0.209	0.582	1.000	0.809
DgWr	0.126	-0.020	0.231	-0.272	-0.149	0.137	0.006	-0.172	-0.078	0.188	-0.225	0.202	0.410	-0.147	0.121	0.709	0.809	1.000

APPENDIX C

Correlation matrix of tree variables analysed, including all trees. See text for explanation of variables.

Dbk = dieback

DBH = diameter at breast height

Stm = number of stems

CD = crown density

Rep = reproduction

Eps = epicormic shoots

TrD = trunk damage

PosD = possum damage

PHB = pinhole borer

Inst = insect damage

Wilt = wilt

Bkg = twig breakage

Dts = dead terminal shoots

Correlations

Variable	Dbk	DBH	Stm	Ht	St	CD	Rep	Eps	TrD	PosD	PHB	Inst	Wilt	Bkg	Dts
Dbk	1.000	0.098	0.142	-0.009	0.110	-0.583	-0.001	0.022	-0.002	0.025	0.406	-0.265	0.298	0.427	0.702
DBH	0.098	1.000	0.187	0.539	0.325	-0.033	0.182	0.029	0.116	0.112	0.236	-0.063	0.204	0.158	0.141
Stm	0.142	0.187	1.000	0.087	0.103	-0.047	0.101	0.162	0.032	0.089	0.110	0.055	0.038	0.037	0.095
Ht	-0.009	0.539	0.087	1.000	0 276	-0.017	0.083	-0.026	0.111	0.060	0.032	-0.078	-0.005	0.039	0.065
St	0.110	0.325	0.103	0.276	1.000	-0.027	0.309	-0.154	-0.119	-0.081	0.097	0.093	0.079	0.105	0.089
CD	-0.583	-0.033	-0.047	-0.017	-0.027	1.000	0.105	-0.039	0.082	0.050	-0.365	0.275	-0.169	-0.415	-0.509
Rep	-0.001	0.182	0.101	0.083	0.309	0.105	1.000	0.022	-0.127	-0.128	-0.050	0.077	-0.111	-0.023	-0.096
Eps	0.022	0.029	0.162	-0.026	-0.154	-0.039	0.022	1.000	0.118	0.096	0.051	0.086	-0.084	-0.076	-0.013
TrD	-0.002	0.116	0.032	0.111	-0.119	0.082	-0.127	0.118	1.000	0.365	-0.024	0.022	0.028	-0.048	0.083
PosD	0.025	0.112	0.089	0.060	-0.081	0.050	-0.128	0.096	0.365	1.000	0.030	0.024	0.007	0.004	0.097
PHB	0.406	0.236	0.110	0.032	0.097	-0.365	-0.050	0.051	-0.024	0.030	1.000	-0.214	0.398	0.423	0.377
Inst	-0.265	-0.063	0.055	-0.078	0.093	0.275	0.077	0.086	0.022	0.024	-0.214	1.000	-0.152	-0.137	-0.189
Wilt	0.298	0.204	0.038	-0.005	0.079	-0.169	-0.111	-0.084	0.028	0.007	0.398	-0.152	1.000	0.275	0.319
Bkg	0.427	0.158	0.037	0.039	0.105	-0.415	-0.023	-0.076	-0.048	0.004	0.423	-0.137	0.275	1.000	0.480
Dts	0.702	0.141	0.095	0.065	0.089	-0.509	-0.096	-0.013	0.083	0.097	0.377	-0.189	0.319	0.480	1.000

APPENDIX D

Correlation matrix of tree variables analysed, for live trees only. See text for explanation of variables.

Dbk = dieback

DBH = diameter at breast height

Stm = number of stems

CD = crown density

Rep = reproduction

Eps = epicormic shoots

TrD = trunk damage

PosD = possum damage

PHB = pinhole borer

Inst = insect damage

Wilt = wilt

Bkg = twig breakage

Dts = dead terminal shoots

Correlations

Variable	Dbk	DBH	Stm	Ht	St	CD	Rep	Eps	TrD	PosD	PHB	Inst	Wilt	Bkg	Dts
Dbk	1.000	0.092	0.173	-0.013	0.131	-0.538	0.039	0.036	0.008	0.049	0.325	-0.157	0.187	0.356	0.672
DBH	0.092	1.000	0.190	0.537	0.334	-0.024	0.190	0.034	0.115	0.114	0.252	-0.054	0.203	0.176	0.137
Stm	0.173	0.190	1.000	0.088	0.103	-0.063	0.097	0.161	0.031	0.086	0.139	0.040	0.063	0.062	0.114
Ht	-0.013	0.537	0.088	1.000	0.291	-0.015	0.090	-0.019	0.107	0.061	0.050	-0.080	-0.029	0.069	0.065
St	0.131	0.334	0.103	0.291	1.000	-0.037	0.307	-0.161	-0.114	-0.083	0.094	0.089	0.125	0.095	0.102
CD	-0.538	-0.024	-0.063	-0.015	-0.037	1.000	0.080	-0.050	0.078	0.036	-0.303	0.197	-0.072	-0.368	-0.467
Rep	0.039	0.190	0.097	0.090	0.307	0.080	1.000	0.016	-0.129	-0.135	-0.019	0.043	-0.074	0.010	-0.070
Eps	0.036	0.034	0.161	-0.019	-0.161	-0.050	0.016	1.000	0.120	0.095	0.061	0.080	-0.074	-0.083	-0.005
TrD	0.008	0.115	0.031	0.107	-0.114	0.078	-0.129	0.120	1.000	0.365	-0.003	0.014	0.029	-0.023	0.094
PosD	0.049	0.114	0.086	0.061	-0.083	0.036	-0.135	0.095	0.365	1.000	0.054	0.005	0.031	0.027	0.118
PHB	0.325	0.252	0.139	0.050	0.094	-0.303	-0.019	0.061	-0.003	0.054	1.000	-0.105	0.356	0.298	0.316
Inst	-0.157	-0.054	0.040	-0.080	0.089	0.197	0.043	0.080	0.014	0.005	-0.105	1.000	-0.018	-0.011	-0.101
Wilt	0.187	0.203	0.063	-0.029	0.125	-0.072	-0.074	-0.074	0.029	0.031	0.356	-0.018	1.000	0.231	0.242
Bkg	0.356	0.176	0.062	0.069	0.095	-0.368	0.010	-0.083	-0.023	0.027	0.298	-0.011	0.231	1.000	0.443
Dts	0.672	0.137	0.114	0.065	0.102	-0.467	-0.070	-0.005	0.094	0.118	0.316	-0.101	0.242	0.443	1.000

APPENDIX E

Correlation matrix of tree variables analysed for dead trees only. See text for explanation of variables.

Dbk = dieback

DBH = diameter at breast height

Stm = number of stems

CD = crown density

Rep = reproduction

Eps = epicormic shoots

TrD = trunk damage

PosD = possum damage

PHB = pinhole borer

Inst = insect damage

Wilt = wilt

Bkg = twig breakage

Dts = dead terminal shoots

Correlations

Variable	Dbk	DBH	Stm	Ht	St	CD	Rep	Eps	TrD	PosD	PHB	Inst	Wilt	Bkg	Dts
Dbk	1.000	•	•	•	•	•	•	•	•	•	•	•	•	•	•
DBH	•	1.000	•	0.995	-0.720	•	-0.961	-0.961	0.720	•	-0.720	•	0.720	-0.720	•
Stm	•	•	1.000	•	•	•	•	•	•	•	•	•	•	•	•
Ht	•	0.995	•	1.000	-0.786	•	-0.929	-0.929	0.786	•	-0.786	•	0.786	-0.786	•
St	•	-0.720	•	-0.786	1.000	•	0.500	0.500	-1.000	•	1.000	•	-1.000	1.000	•
CD	•	•	•	•	•	1.000	•	•	•	•	•	•	•	•	•
Rep	•	-0.961	•	-0.929	0.500	•	1.000	1.000	-0.500	•	0.500	•	-0.500	0.500	•
Eps	•	-0.961	•	-0.929	0.500	•	1.000	1.000	-0.500	•	0.500	•	-0.500	0.500	•
TrD	•	0.720	•	0.786	-1.000	•	-0.500	-0.500	1.000	•	-1.000	•	1.000	-1.000	•
PosD	•	•	•	•	•	•	•	•	•	1.000	•	•	•	•	•
PHB	•	-0.720	•	-0.786	1.000	•	0.500	0.500	-1.000	•	1.000	•	-1.000	1.000	•
Inst	•	•	•	•	•	•	•	•	•	•	•	1.000	•	•	•
Wilt	•	0.720	•	0.786	-1.000	•	-0.500	-0.500	1.000	•	-1.000	•	1.000	-1.000	•
Bkg	•	-0.720	•	-0.786	1.000	•	0.500	0.500	-1.000	•	1.000	•	-1.000	1.000	•
Dts	•	•	•	•	•	•	•	•	•	•	•	•	•	•	1.000

Chapter 2b: Causes of kamahi ill-health in localised areas of high tree mortality in Tongariro National Park

ABSTRACT

Concern has been raised over the health of kamahi in Tongariro National Park; in particular, dead trees appear to have become a common component of the forest canopy on the western side of the Park. In an attempt to isolate the causes of this apparent dieback problem, this study focuses on the western face of Hauhungatahi where dieback is most visible, and uses the Decline-Disease Theory of forest dieback as a model for a survey. Thirty quadrats 20 m x 10 m have been located systematically on three randomly placed and oriented transects. The health of kamahi trees within these quadrats has been scored, and these scores have been compared to measures of factors possibly affecting health. None of the suggested predisposing, triggering or hastening factors were consistently related to kamahi health; however, older stems were more likely to be dead than younger stems, and pinhole borer was very common on dead stems, and very uncommon on live stems of any health class. Nor did principal components analysis provide any further answers. These results suggest that age predisposes kamahi stems to dieback, with older kamahi stems more prone, and that *Sporothrix*/pinhole borer tend to invade already dead or dying trees. No factor likely to be causing any decline in health was identified from this study. The implications of these results and the direction for future research is discussed.

INTRODUCTION

Tongariro National Park, in the Central Volcanic Plateau of the North Island (fig 2 chapter 1) is a World Heritage area, which status recognises and protects nationally and internationally significant vegetation and landscape features. The Park encompasses over 80 000 ha, and ranges in altitude from around 600 m above sea level (a.s.l.) near Rotoaira, to nearly 2800 m at the summit of Ruapehu. Within this are a number of ecologically important and sensitive areas. Vegetation in the Park can be broadly grouped as mixed beech forest (*Nothofagus* spp.) and podocarp/mixed-hardwood forest in the south and west, scrub/tussockland in the north and east, and sub-alpine vegetation at higher altitudes (Atkinson, 1981). Recently there have been reports of extensive dieback in forested areas of the Park; concern has been raised, for example, over the state of beech, kaikawaka (*Libocedrus bidwillii*) and kamahi (*Weinmannia racemosa*). This last species is the least studied presently, and forms the focus of this study.

Kamahi is one of New Zealand's most common canopy species and probably associates with every other tree species occurring south of its northern-most limit, Auckland (Wardle, 1966). Its altitudinal range in Tongariro National Park extends from the lowest point in the Park (600 m above sea level) to approximately 1100 m, where it gives way to more strictly montane canopy species such as kaikawaka and beech (Atkinson, 1981). Kamahi has abundant, small wind-dispersed seeds, and achieves maximum growth in the sapling stage, suggesting it requires high light conditions for germination and development (Lusk, 1989). It also has fast growth rates relative to other New Zealand canopy species (Ogden, Fordham, Pilkington & Serra, 1991).

On the slopes of Hauhungatahi, on the western side of the Park, kamahi is one of the forest dominants and dead trees are an important component of the forest (figs 2.1 and 2.2). In nearby areas of similar kamahi dominated forest types, for example the southern slopes of Ruapehu, dead trees appear rare. Even on the western slopes of Hauhungatahi, where the dead trees are most common, the pattern appears complex: stands which are looking extremely moribund or completely dead may be within one hundred metres of apparently healthy stands.

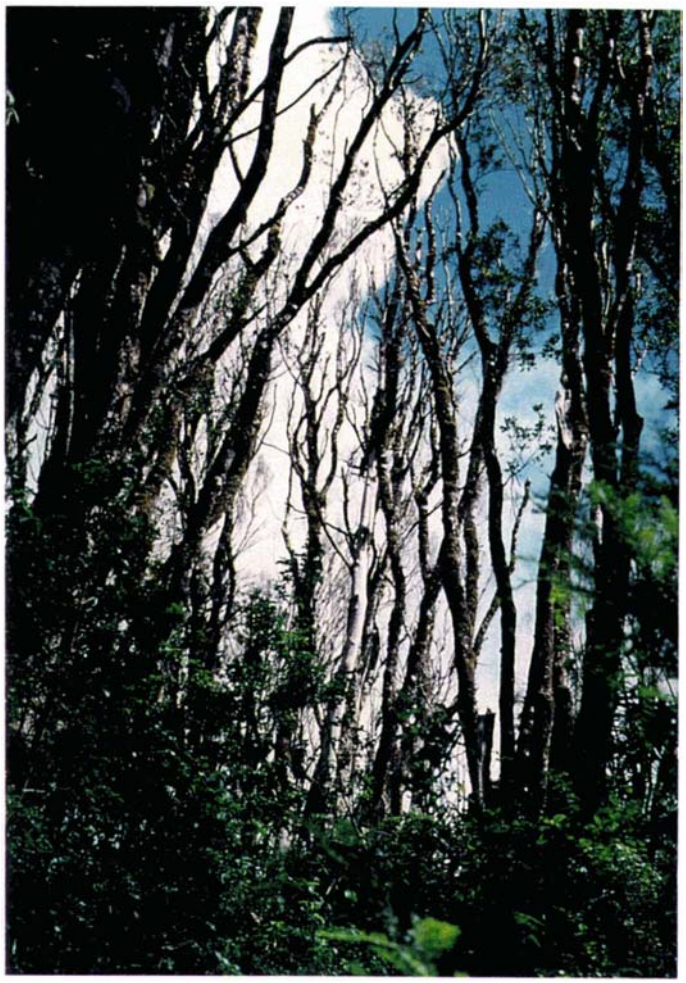

Fig 2.1 (right) *Moribund stand of kamahi in Tongariro National Park (photo G.Rapson).*

Fig 2.2 (below) *Moribund patch of kamahi in Erua State Forest (15 km of Tongariro National Park boundary).*

As defined in the previous chapter, the term 'dieback' refers to a multi-factor cause of tree death (Houston, 1992). This makes understanding the dieback difficult, as the factor that ultimately causes death may be a long way along a process of decreasing health (Franklin, Shugart & Harmon, 1987). 'Decline' refers not to individual trees, but is a stand phenomenon, where so many of a species are under-going dieback that the population is declining locally.

As the processes influencing live vegetation patterns are complex and poorly understood (Stewart & Veblen, 1982), it is no surprise that the processes affecting which trees die within a natural community are also complex and poorly understood (Franklin *et al.*, 1987). It is extremely difficult to distinguish natural forest changes from those largely due to anthropogenic causes (Jane & Green, 1983), and for many cases the exhibition of ill-health at crown level, which triggers investigation, is a later stage in a process which is already largely over (Landmann, 1993). Also, if only dying trees are looked at, it is possible to mistake the distribution of unhealthy trees for an expression of the factors leading to their ill-health, when it may actually be a pattern inherent to the ecology of that species (Acker, Harmon, Spies & McKee, 1996). In addition, decline of a species in different areas may be due to completely different causes; this can cause controversy and confusion (Manion, 1991). Popular reports about dieback can further confuse the issue by attempting to oversimplify the situation, overstating our understanding, and ignoring death as a natural event (Sinclair & Hudler, 1988).

CAUSES OF KAMAHI DIEBACK

Possible causes of kamahi decline have been widely researched, and more widely speculated upon (Leutert, 1988).

Browsing by the repeatedly-introduced brushtail possum (*Trichosurus vulpecula* Kerr) has come to be the generally accepted cause of any decline in natural vegetation or of tree death in New Zealand (for example Kean & Pracy, 1953; Veblen & Stewart, 1980; Pekelharing & Batcheler, 1990; Clarkson & Clarkson, 1995). Kamahi is known to be a preferred food of possums in many areas (e.g. Mason, 1958; Fitzgerald, 1976; Pekelharing, 1979), and possums have therefore been the prime suspects in kamahi decline in other areas of the country (for example, Wellington (Fitzgerald, 1976) , South Westland (Fitzgerald & Wardle, 1979) and Taranaki (Leutert, 1988)). Batcheler (1983) cites the repeated coincidence of increased possum densities with decreased canopy health in rata-kamahi forest all over New Zealand, as evidence of their guilt, while Rose *et al.* (1992) observe that the

geographic variation of this forest decline reflects patterns of possum establishment. Holloway (1973, p. 127) even goes so far as naming possums 'the number one problem animal' for the health of rata-kamahi forests. Kean (1953) expected that, wherever possums persist, all their highly preferred foods, including kamahi, would become locally extinct. Most of the evidence associating possums with kamahi decline is as prolifically reported as it is circumstantial (Leutert, 1988).

Natural causes have also been suggested for some kamahi decline. The canopy may be opened by various natural occurrences, such as breakage due to windthrow or heavy snowfall, or by possums. Opening of the canopy may then lead to death of many trees, by altering the micro-climate and increasing exposure (Payton, 1988). This may be a factor important in later stages of decline.

Drought may increase mortality in a forest, or lead to stand collapse (Green & Jane, 1983; Jane & Green, 1985; Jane & Green, 1986). The effect of drought will be heightened if it coincides with spring flush (Hosking, 1986). Some of the mortality that has been blamed on introduced animals in the past may be due to drought (Atkinson & Greenwood, 1972; Grant, 1984), and perhaps other natural factors.

A fungus related to Dutch Elm Disease and Oak Wilt (*Sporothrix*) is often associated with kamahi death (Payton, 1989). This association has sometimes led to the conclusions that *Sporothrix* is driving the decline, or alternatively, that *Platypus*, the insect vectors of *Sporothrix*, prefer moribund trees (Hosking & Hutcheson, 1988). Hosking (1993b) comments that organisms indigenous to an area, and with a long history there, are likely to have 'co-evolved' with their host, and are thus unlikely to cause a widespread decline of that host.

High levels of insect herbivory have been suggested as contributing to kamahi dieback, particularly in the North Island (Rogers & Leathwick, 1997).

Climate change has been suggested as a possible cause driving dieback. This is reasonably untestable with our current lack of understanding of vegetation changes (Grant, 1984).

Poor soil nutrient status may inhibit recovery from a period of ill-health caused by other factors (Jane & Green, 1983).

It has also been suggested that kamahi dieback is a completely natural process driven by natural population dynamics (Mosley, 1978; Veblen & Stewart, 1982). There are many theories of natural population dynamics in trees (as discussed by Harcombe, 1987), and the pattern may be complex. However, if natural population dynamics are the cause, dieback should be restricted in scale, with no risk of extinctions.

The decline disease theory of stand-level dieback has been widely applied to New Zealand forest declines (in particular Stewart, 1989). As discussed in the previous chapter, this is a three factor model of forest decline: trees must be predisposed or susceptible to dieback; dieback must be precipitated, or initiated; and further factors may hasten or accelerate decline (Houston, 1974; Mueller-Dombois, 1988; Manion, 1991). Stewart (1989:243) comments that "all known examples of dieback in New Zealand *Nothofagus* spp., *Metrosideros* spp., and beech/hardwood forests can be explained using this three-factor framework". It is now extremely common in investigations of forest decline to approach the problem using this theory.

AIMS AND APPROACH

This study focuses on an area of Tongariro National Park where kamahi dieback has been most marked. Random selection of co-ordinates for sites within the study area is ideal for statistical rigour, but often logistically difficult in a forest environment due to the prohibitive time commitment involved (Payton, Pekelharing & Frampton, 1997b). A compromise is systematic sampling on transects randomly located at the forest margin and allocated random orientations into the area to be sampled (Druitt, 1985). Although not strictly random, this design allows samples to be treated statistically as if they are random (Hurlbert, 1984). All kamahi trees within each quadrat are included in the study, to show the pattern of healthy as well as unhealthy kamahi trees (Acker *et al.*, 1996); details that may affect the level of kamahi regeneration at each quadrat (ground cover and deer browse on kamahi) have also been recorded.

Measures of health

Crown density and proportion of dead terminal shoots are recorded as measures of stem health. Crown density is most appropriate for studying changes in health of a stem over time, while proportion of dead terminal shoots is a more direct measure of stem health. The numbers of trees that are dead or very unhealthy in each quadrat, as well as the mean score for dead terminal shoots at that quadrat, have been used to provide measures of stand health. A multitude of dieback variables has been included because little is known of the nature of dieback, or how to measure it; these different ways of measuring dieback should allow every opportunity for meaningful relationships to be detected.

Possible causes of dieback

The decline disease model categorizes factors potentially involved in kamahi dieback as those most likely to predispose certain trees to die, those that may trigger a decline in health that may lead to death, or those that hasten death in trees already declining. By comparing each of the factors studied to a measurement of stem or stand health, it may be possible to determine the role each is playing. If any of the factors suggested to predispose trees to dieback are consistently associated with stands or stems in poor health, and not usually with stands in good health, then those factors are likely to be contributing to the pattern.

Most of the factors suggested as potential 'triggering' or causal factors may instead be hastening factors, which may be confused with the causal factor, or lead the investigation away from the proximal cause (Stewart & Veblen, 1983). In some cases a particular factor may cause dieback, while in another case it may merely hasten death in an unhealthy tree, whereas it is usually clear that predisposing factors are not causing death - they have usually been associated with a stand over the whole lifetime of the trees involved. If a factor is consistently associated with dead or dying trees, but not with more healthy trees, it is likely to be a hastening factor. Causal factors are more likely to be associated with stems in declining health. Thus, it is the relationships between each factor studied and measurements of health that are the most relevant to this study.

Predisposing factors

Many of the factors mentioned previously as possibly contributing to kamahi dieback are 'predisposing' factors, which potential importance has been largely overlooked until the last ten years (Stewart & Rose, 1988).

- Many authors have suggested age affects susceptibility to dieback (for example Stewart & Rose, 1988; Payton, 1987). In this study, stem age is measured indirectly by diameter at breast height (DBH), rather than directly by tree coring, which is more invasive and much more time-consuming. Lusk (1989) found a very tight relationship ($y=48.38+5.53x-0.058x^2+0.0004x^3$, r = 0.87, $P<0.0001$, N=200, y=age, x=DBH) between DBH and age from sections of kamahi taken at approximately 650-700 m a.s.l. on the south-western side of the Park, an area very close to that studied here, but at a slightly lower and more limited altitudinal range.
- Stewart and Rose (1988) comment that young stands are less susceptible to dieback, while stands with a high proportion of old trees are more susceptible. In the

present study, quadrats have been put into age categories based on the size distribution of trees within them in order to investigate the effect of stand age.

- Stand density can predispose trees to dieback, as competition is higher in denser stands (Peet & Christensen, 1987). Conversely, high stem density implies the stand is recently established.
- In the Southern Ruahine Range, Rogers and Leathwick (1997) found slope, aspect and altitude could predispose forest to poor health. Stands with slopes greater than 20°, and stands with western (and to a lesser extent northern) aspects were more susceptible to dieback, while mid-altitude forests were less susceptible. Each of these factors has been measured directly in the present study.
- Cowan's (1997) study of rata dieback in the Orongorongo Valley found exposure and distance from forest edge were important; in the present study, exposure is measured on a subjective scale, while distance from forest edge is measured directly.
- Landsberg and Gillieson (1995) comment that soil drainage can affect nutrient levels and tree health, and Akashi and Mueller-Dombois (1995) found drainage to be the key factor in Hawaiian rain-forest dieback; in the present study, a subjective scale is used to compare drainage at each quadrat.
- Percentage of kamahi in the canopy is estimated in the present study to determine if more or less monocultural stands are predisposed to dieback.
- Whether a stem represents a portion of a multi-stemmed tree, or a single stemmed tree was recorded and is included to determine if either may predispose kamahi trees to dieback.

Triggering factors

In kamahi dieback, possum browse is usually considered to be the trigger factor (e.g. most recently Clarkson & Clarkson, 1995; Payton, Forester, Frampton & Thomas, 1997a). However, insects can also be trigger factors (Landsberg & Gillieson, 1995). Relative intensity of possum browse and of insect browse on each tree can be estimated, but not absolute amount of foliage affected (Leutert, 1988), due to the unknown persistence of damaged leaves relative to undamaged (Meads, 1976), and the difficulty of observing leaves totally lost to browsing. If, as Meads (1976) suspects, leaves damaged by browsing are abscised at a higher rate than undamaged leaves, they will be present in lower numbers than were actually damaged. Also, browsing is most damaging on essential tissues such as meristem; however, it is most easily measured on foliage, where there is considerable tolerance to browse (Franklin *et al.*, 1987). In

the present study, to overcome the problem of estimating absolute browse, a scale is used for comparing the relative intensities of possum browse and insect browse in the crown of each stem.

Pathogenic fungi such as *Sporothrix* (Payton, 1989) may also trigger dieback. Rather than establishing *Sporothrix* presence directly, which would involve considerable damage to a tree and usually involves taking a section, *Sporothrix* presence is measured here indirectly, through presence or absence of its vector's entry holes on the stem.

Accelerating factors

Accelerating factors can also be very important in dieback; the importance of these secondary factors is highlighted by Rogers *et al*. (1997) who found that in the Southern Ruahine Range, canopy collapses have been due to possum browse, but once started, collapse continues irrespective of levels of possum defoliation. Payton (1988) had previously examined the effect of canopy closure, a measure of stand collapse, in Westland rata-kamahi forest, and found where possum browsing resulted in canopy opening, exposed leaf bunches continued to deteriorate, even in the absence of further browsing. Rogers *et al.* (1997) suggest mechanical damage from wind in canopies opened by possums may hasten collapse, while Payton (1988) comments that trees already opened by browse were browsed to much lower levels than intact trees, and suggests the increased light affects palatability of foliage. Payton (1988) also found that level of canopy closure was only important in stands that had already undergone natural thinning (i.e. mature stands). In the present study, quadrats' canopies are classed as either open or closed, and the percentage of canopy cover estimated.

Rogers *et al*. (1997) suggested outbreaks of defoliating insects attracted by damaged trees (also Agyeman & Safo, 1997; Payton, 1987) contributed to the canopy collapse; insect browse is measured in the present study as described above. Fungal (Agyeman & Safo, 1997), or more specifically *Sporothrix* (Payton, 1987) attacks, may hasten dieback; *Sporothrix* presence is assumed from presence of *Platypus*, as described above. Possum browse may also be a hastening factor, rather than or as well as a trigger factor (Stewart & Veblen, 1983); for example, canopy mortality may lead to a 'richer' understorey, which may attract possums; estimation of possum browse in the present study is outlined above.

Statistical Approach

In studies of dieback, multiple regression is often considered to be the best way to establish which of a number of variables is most likely to be affecting tree health (for example Manion, 1991; Fisher, 1997; Rogers & Leathwick, 1997). However, multiple regression may be inappropriate if 'independent' variables are highly correlated (Sokal & Rohlf, 1995), as they almost certainly are in the present study, and are likely to be in most surveys. For this reason, predictive models have been rejected in this study in favour of more exploratory statistics, in particular correlations and Principal Components Analysis (PCA), where results are easier to analyze and interpret with confidence.

Manion (1991) comments that if dieback is due to decline disease, there should be weak correlations between measures of stem health and other variables - if any correlations are strong, then those factors are likely to be the only ones involved in dieback. In this study, correlations are calculated between stem or stand health and all other variables. Correlations with $r > 0.7$ (i.e. $r^2 > 0.49$) are considered large, while those with $r > 0.5$ ($r^2 > 0.25$) are considered weak but significant (Chatfield & Collins, 1980).

On site data, where 15 variables are included, some of which are likely to be *correlated* to *each* other, principal components have been calculated. Many more variables were collected about sites than stems, where only four of the variables measured were likely to be involved in tree health. For this reason, correlations were considered sufficient for determining whether any relationships exist. Principal components analysis effectively summarises strongly correlated variables into one or more components, which explain more of the variation of the data than any of the variables of which it is (or they are) composed. This reduction in dimensions more easily allows data to be visualised. In addition, if the first few components explain much of the variation in the data, then they can be treated as summaries of the data set, and further analysis can be carried out on them. For example, relationships between measures of health and the components that correlate most strongly with the variables of interest can be examined (Chatfield & Collins, 1980).

METHOD

The survey of kamahi in areas of dieback was undertaken during the summer of 1997/1998.

SITE SELECTION

All 30 quadrats are on the western face of Hauhungatahi, on one of three transects laid by the Department of Conservation (fig 3.1, Appendix B). Transects are numbered from 2 to 4 to coincide with Department of Conservation data. The centre of the first quadrat on each transect is 100 metres from the randomly located transect origin at a randomly allocated direction into the forest. The centres of subsequent quadrats are located at 100 metre intervals (ground distance) on that orientation. Each quadrat is 20 m x 10 m, with the long side perpendicular to the transect.

SURVEY

Site character

Details of the physiognomy of each quadrat were recorded. Slope and aspect were measured on site; altitude and distance from the nearest forest edge were later determined from topographical maps. Level of exposure is on a scale of 0 to 2, where 0 is not exposed and 2 is very exposed, particularly on ridges. Canopy closure was scored as 0 if open, and 1 if closed. Drainage is on a scale of 1 to 5, with 1 free draining (usually on a slope) and 5 if water is able to 'pond'. Number of kamahi stems and number of stems of all species at each quadrat are from Department of Conservation (unpub.) data. The height of the canopy was estimated, as were percentage ground cover (vegetation less than 1 metre high), percentage canopy cover, and percentage of kamahi in the canopy. Stand age categories have been established based on frequency distributions of diameter at breast height of all trees in each plot (Appendix B).

Tree health

Within each quadrat, all kamahi stems at least 3 centimetres diameter at breast height (DBH) are included, and DBH of each is recorded. If two or more stems originate from one root bole, they are scored separately, but recorded as stems (1), while single-stemmed trees are scored as trees (2). Crown, or foliage, density was measured where possible, using a scale provided by Manaaki Whenua (fig 3.2). If crowns overlapped to an extent that limits could not be determined, no score was recorded.

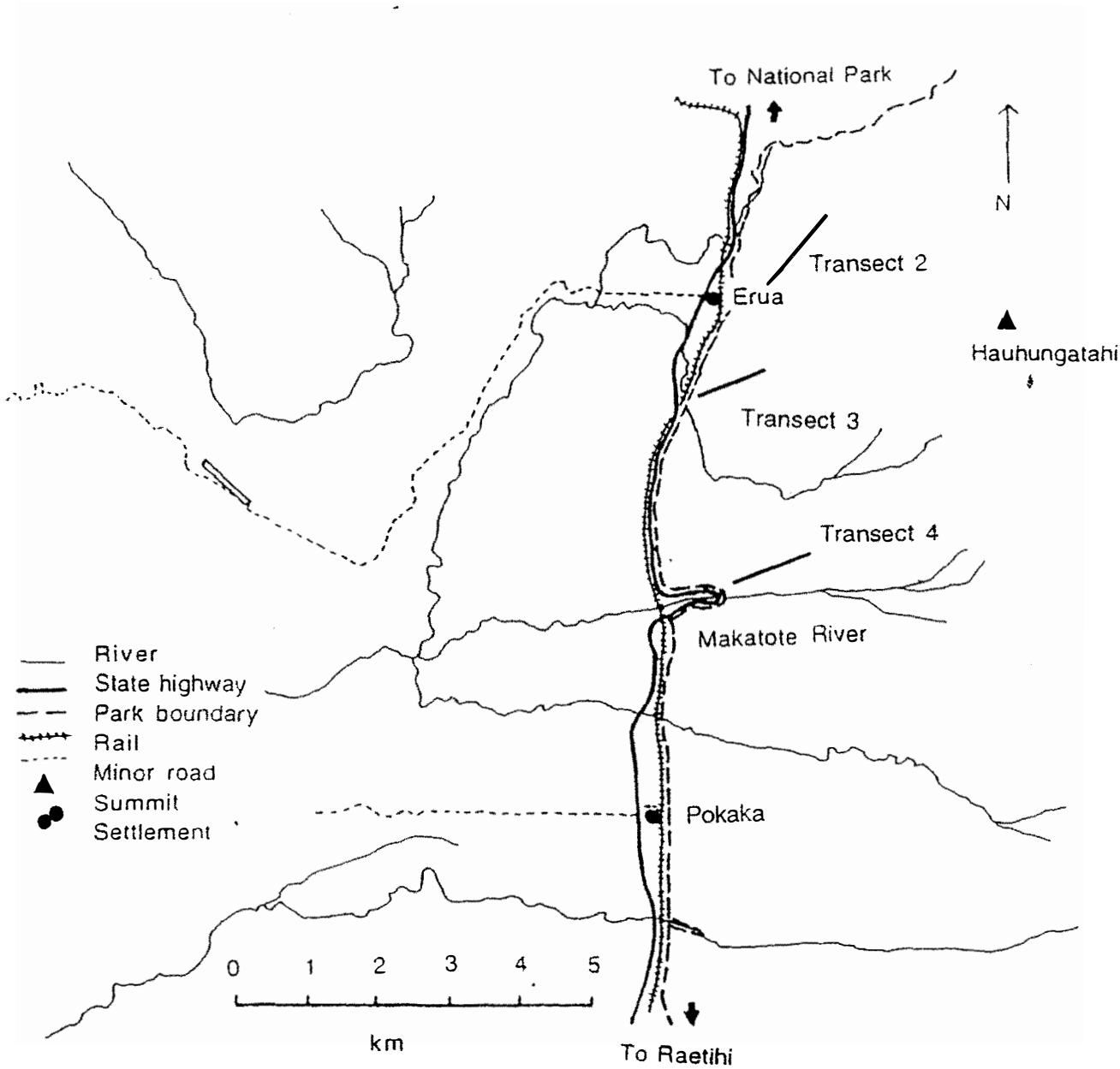

Fig 3.1 *Location of transects surveyed.*

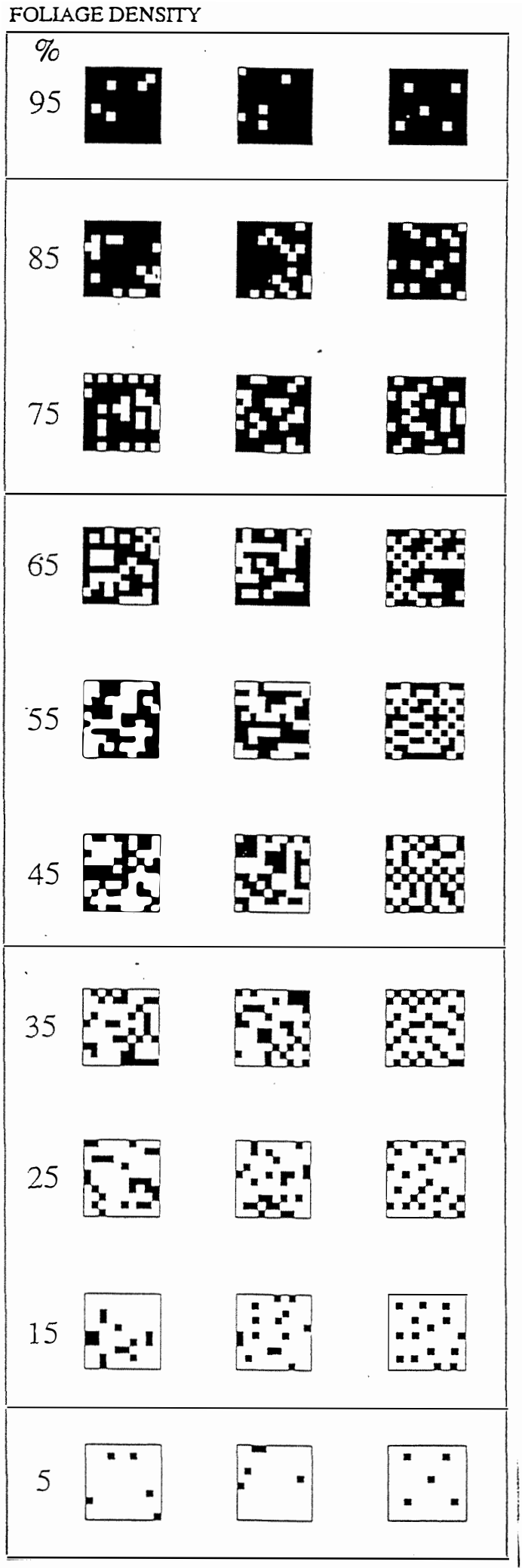

Fig 3.2 *Reference model for scoring percentage crown density (from Manaaki Whenua)*

Stems were scored for possum browsed, insect browsed, and deer browsed leaves (where leaves are present at a height accessible to deer, i.e. less than 2 metres) as a proportion of all leaves (table 3.1), or in the case of deer browse, as a proportion of all accessible leaves. Again, for some trees this was not possible as their leaves could not be distinguished from those of surrounding trees.

Table 3.1 *Scale used for scoring possum-, insect- and deer-browse in stems. Where there were no live leaves, or leaves from the stem to be scored could not be distinguished from those of other stems, the stems were scored as x.*

Score	Level of damage	Proportion of leaves browsed
0	nil	0
1	light	1-25 %
2	moderate	26-50 %
3	heavy	51-75 %
4	severe	76-100%
x	unable to estimate	

Presence or absence of pinhole borer on the trunk was recorded.

The proportion of dead terminal shoots was estimated on a linear scale from 0-6 (table 3.2). Where the stem being scored was dead, there was a further score for approximate time since death (table 3.2).

Table 3.2 *Scoring of stem health. All stems were scored for dead terminal shoots; dead stems were also scored for time since death. Where the crown from the stem to be scored could not be distinguished from other stems it was not scored.*

Score	Proportion of dead terminal shoots	
0	0	
1	1-19 %	
2	20-39 %	
3	40-59 %	
4	60-79 %	
5	80-99 %	
6	100 % stem appears dead	
	Time since death	**State of stem**
1	very recent	very fine twigs present, still has dead leaves
2	recent	fine twigs
3	dead	main branches, some twigs
4	long dead	large branches only, stem may be broken
5	very long dead	rotten stem

ANALYSIS

Univariate statistics were analysed by Systat (Systat Inc, 1992); all other statistical analysis of data were conducted by JMP (SAS Institute, 1994).

Site character

Most of the variables describing the character of each quadrat could be analysed without modification; however some variables needed manipulation to enable meaningful analysis (table 3.3). Aspect has been divided into north-south and east-west components by taking the cosine and the sine of the aspect (in radians) respectively. As mentioned previously, classes for stand age were determined from the slopes of their frequency histograms for diameter at breast height. Density of stems of all species and density of kamahi stems are calculated from the total number of each in the quadrat. 'Dead stems' refers to the density of dead stems of any species in the quadrat, 'Dead kamahi stems' to the density of dead kamahi stems in each quadrat, while 'Dying kamahi stems' to the density of kamahi stems with a dead terminal shoot score of 5 (i.e. with 80-99 % of shoots dead, as described below). The proportion of stems that are dead at each quadrat for all species, and for kamahi stems only, are given in percentages, as is the proportion of kamahi *stems* that are dying. The variable

Table 3.3 *List of site variables analysed (explanation of dieback variables is in text).*

Variable	Explanation
Altitude	m asl
Slope	°
N-S	cosine (aspect)
E-W	sine (aspect)
Exposure	in text
Closure	in text
Drainage	in text
Canopy height	m
Distance from forest edge	m
Ground cover	%
Canopy cover	%
Kamahi cover	%
Stand age	in text
Total stems	100 m^{-2}
Kamahi stems	100 m^{-2}
Dead stems	100 m^{-2}
Dead kamahi stems	100 m^{-2}
Dying kamahi stems	(kamahi stems with dead shoots score=5) 100 m^{-2}
Average kamahi health	mean dead shoot score
Percent dead stems	
Percent dead kamahi	
Percent dying kamahi	

'Average kamahi health' is taken from the mean scores of dead terminal shoot for kamahi stems at each quadrat.

Relationships between all site characters, and between each of the dieback variables and each site character were analysed by correlation.

Principal components of site character variables were extracted, for all variables excluding height, because the lack of a tree canopy at quadrat 5 on transect 3 would cause it to be omitted from PCA if height was included. Variables relating to the amount of dieback at each quadrat were also excluded so components were not influenced by observed levels of dieback at each quadrat. Correlations were calculated between each of the three main components and each of the dieback variables, to determine whether the combined force of all site characters could predict the level of dieback at a quadrat.

Tree health

Most of the variables analysed were unmodified from those measured (table 3.4).

Correlations between each of the tree variables were calculated with all trees included, again with only live trees, and again with only dead trees. This should help determine if any of the relationships are being driven largely by the few dead trees.

Table 3.4 *List of stem variables analysed. Where no explanation is included, the variable is analysed as explained in text.*

Variable	Explanation
Diameter at Breast Height	cm
Tree/stem	stem=1; tree=2
Crown density	%
Possum browse	
Insect browse	
Deer browse	
Borer	
Dead terminal shoots	
Death	

RESULTS

SITES

Transect two contains eleven quadrats, transect three contains nine, and transect four is ten quadrats in length. Canopy height could not be recorded at quadrat 5 on transect 3 (Appendix B), as there was no tree canopy formed. Values could not be calculated for dying kamahi stems and average kamahi health at eight quadrats, because dead terminal shoots scores were not collected at those quadrats.

Quadrats range in altitude from 770 m above sea level (m asl) to 940 m asl (table 4.1), and from approximately 75 m from the forest edge to 1100 m from the edge. On transect two all quadrats are between 100 m and 500 m from the forest margin, on transect three between 75 m and 750 m, and on transect four quadrats are between approximately 200 m and 1100 m. Ten quadrats have no slope, six have a slope of 5° or less, and the slopes of the remaining 14 quadrats are distributed normally between 10° and 40°; this led to the disparity between mean and median for slope (skewness = 0.71). Of the 20 quadrats with any slope, most have a southerly aspect, with only five northerly facing; all quadrats are west facing.

Table 4.1 *Summary of site data. There was one quadrat where no canopy was formed, so it has no score for canopy height; dead shoot data were not collected from stems at 8 quadrats. No standard deviation was given for stand age as the scores are not on any linear scale.*

Variable	N	Mean	Median	Std deviation	Minimum	Maximum
Altitude (m)	30	846.5	855	41.1	770	940
Slope (°)	30	12.4	3.5	14.2	0	40
N-S	30	-0.2063	-0.3420	0.4907	-0.8660	0.7661
E-W	30	-0.8430	-0.8660	0.1380	-0.9848	0
Exposure	30	0.3	0	0.45	0	1
Closure	30	0.6	1	0.5	0	1
Drainage	30	2	1	1.6	1	5
Canopy height (m)	29	14.2	15	3.45	2	18
Distance (m)	30	464	400	257	75	1100
Ground cover (%)	30	37	40	22	10	80
Canopy cover (%)	30	52	52.5	20.95	5	85
Kamahi cover (%)	30	38	50	28.25	1	80
Stand age	30	4	4.5	NA	1	9
Density stems (m^{-2})	30	31.8	24.25	19.81	10.5	85.5
Density kamahi stems (m^{-2})	30	13.3	12.75	7.9	0.5	32.5
Dead stems (m^{-2})	30	3.58	2.25	3.84	0	15.5
Dead kamahi stems (m^{-2})	30	1.88	1	2.03	0	7
Dying kamahi stems (m^{-2})	22	1.0	0.5	1.1	0	4.5
Average kamahi health	22	2.33	2.36	0.735	1	4.1
Percent dead stems	30	10.3	9.7	8.1	0	33.75
Percent dead kamahi	30	13.3	9.1	13.9	0	57.9
Percent dying kamahi	22	10.0	3.8	20.7	0	100

Most quadrats were not exposed (8 of the thirty were), and most had a closed canopy (12 open). Drainage was generally very good, although six quadrats had very poor drainage. Quadrat 3 on transect 3 had a canopy height of 2 m; the remaining quadrats ranged from 9 m to 18 m, and distribution is strongly skewed to taller canopies.

Percentage ground cover and canopy cover varied greatly between quadrats. Ground cover is slightly skewed towards lower values (skewness 0.24), while canopy cover is skewed towards higher values (skewness -0.50). Kamahi cover ranges from 1 % to 80 %, with 9 quadrats from 1 % to 10 %, but most from 50 % to 80 %. The distribution of stand age is extremely normal, with the exception of the nine sites in class 1. Density of stems ranged from 10.5 to 85.5 per 100 m^2, but only six quadrats had a density greater than 50 stems per 100 m^2. Density of kamahi stems ranged from 0.5 to 32.5 per 100 m^2, with only five quadrats having a density greater than 20 stems per 100 m^2.

The density of dead stems (any species) at each quadrat varied from 0 to 15.5 per 100 m^2, but at most quadrats (24) was less than 5 per 100 m^2; data separating dead trees into species are not available except for kamahi. Dead kamahi stems at each quadrat ranged from 0 to 7 per 100 m^2, with 24 quadrats at 2.5 or less per 100 m^2. The density of dying kamahi was from 0 to 4.5 per 100 m^2 at each quadrat for which there is a value; 15 of those 22 quadrats have values of 1 or less. The value at each quadrat for average kamahi (from scores for dead terminal shoots on each tree) ranges from 1 to 4.1. The percent of stems that are dead ranges from 0 to 33.75 %, with only three quadrats having greater than 20 % dead. The percent of kamahi stems that are dead ranges from 0 to 57.9 %, with six quadrats over 20 %. The percent of kamahi stems that are dying ranges from 0 to 100 %; of the 22 quadrats with values, 21 are under 15 %.

Of the correlations tested (Appendix C), a number are significant (table 4.2). Both north-south and east-west components of aspect are negatively related to slope, while exposure is positively related to slope.

Drainage is negatively related to canopy height. Distance from forest edge has a predictably strong positive relationship with altitude. Predictably, canopy cover is higher with closed canopies; it also has significant negative relationships with drainage and ground cover. The density of stems is negatively related to slope and stand age. The relationship between stem density and density of kamahi stems is strongly positive.

Table 4.2 *Significant correlations between site variables (where* |r|*>0.5, and* P*<0.005). Correlations with* P*>0.005 but* |r|*>0.5 are included in italics.*

	Relationship	d.f.	*r*-value	*P*-value
Site character variables	NS x slope	29	-0.53	0.0028
	EW x slope	29	-0.60	0.0006
	Exposure x Slope	29	0.65	0.0001
	Canopy height x Drainage	28	-0.58	0.0010
	Distance x Altitude	29	0.70	<0.0001
	Canopy cover x Closure	29	0.70	<0.0001
	Canopy cover x Drainage	29	-0.54	0.0021
	Canopy cover x Ground cover	29	-0.64	0.0001
	Stem density x Slope	29	-0.55	0.0015
	Stem density x Stand age	29	-0.58	0.0008
	Kamahi density x Stem density	29	0.75	<0.0001
Dieback variables	*Kamahi average x EW*	*21*	*-0.59*	*0.0207*
	% dead kamahi x Ground cover	29	0.50	0.0045
	% dying kamahi x Drainage	21	0.59	0.0040
	% dying kamahi x Canopy height	20	-0.78	<0.0001

The percentage of dead kamahi stems is positively related to percentage ground cover. The percentage of kamahi stems that are dying is positively related to drainage, and negatively related to canopy height; both of these correlations are reliant on one quadrat, quadrat 3 of transect 3 with 100% of its kamahi stems (of which there is one) dying (fig 4.1), and neither are significant if that quadrat is excluded.

The principal component analysis of the site variables, excluding height and dieback variables, explained 28.30 % of variance on the first component, 20.88 % on the second (fig 4.2) and 15.59 % on the third; none of the other components explain more than 10 % additional variance. The first component is very strongly influenced in a negative direction by the slope recorded at each quadrat, and in a positive direction by stem density; the east-west component of slope also is closely related in a positive direction, while exposure is related negatively (table 4.3). The second component is most influenced by canopy cover and closure in a negative direction and drainage and ground cover and distance from the forest margin in a positive direction. The third component is most influenced by altitude, percentage kamahi cover and distance from forest margin (all positive).

The only significant correlation between any of these three components summarising aspects of the site dataand the measures of stand health, is the density of dead stems of any species and principal component 1 (table 4.4). The next nearest to significant correlations exist between the density of dying kamahi and the percent of dead kamahi stems at each quadrat, and score on principal component 2 (r=0.44 and 0.45 respectively; fig 4.3).

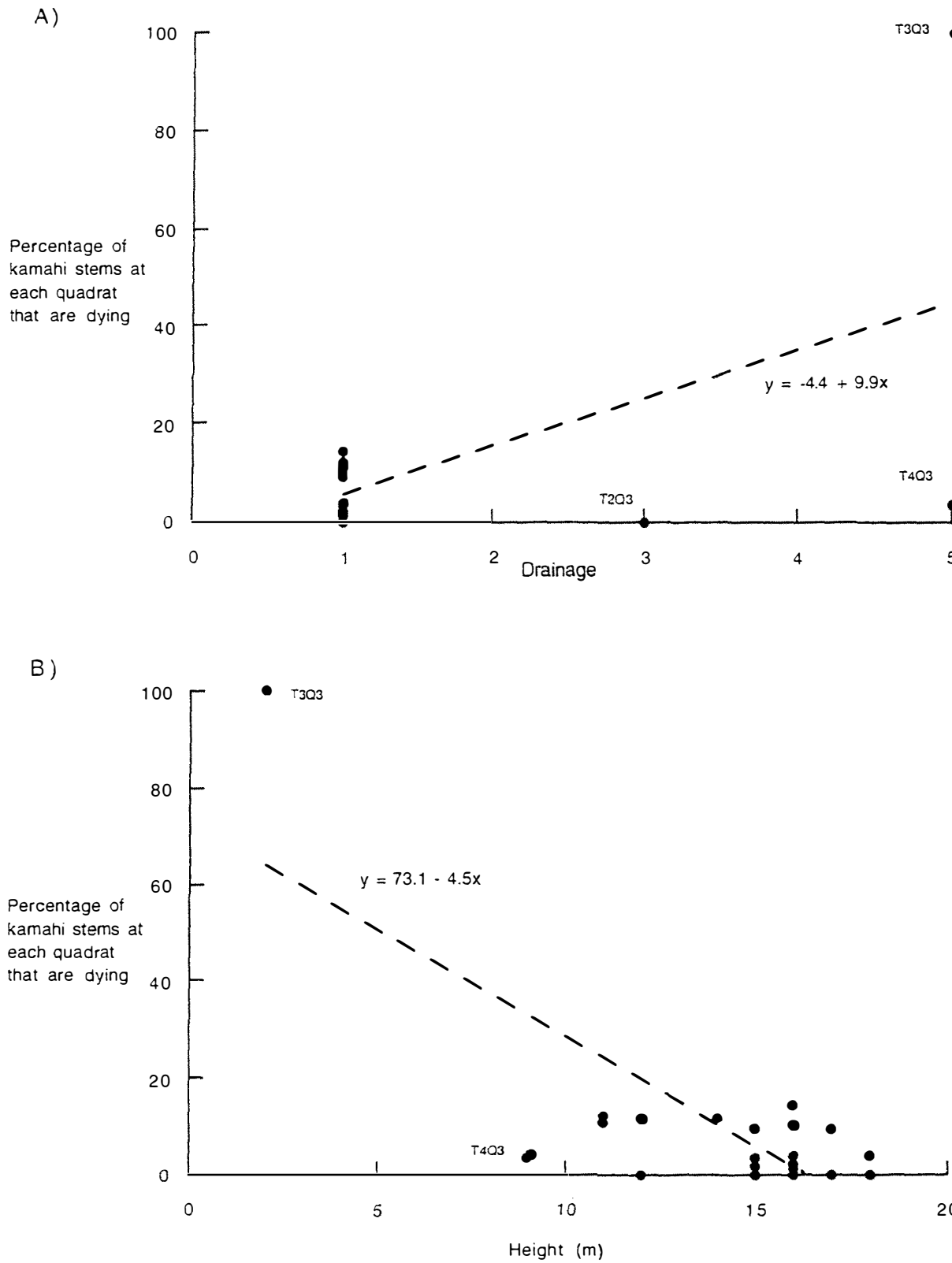

Fig 4.1 *A) Relationship between the percentage of kamahi stems dying and drainage score for each quadrat. r=0.59. B) Relationship between the percentage of kamahi stems dying and canopy height at each quadrat. r=-0.78. Outliers for each are labeled with transect (T) and quadrat (Q) number; dashed lines indicate linear regression.*

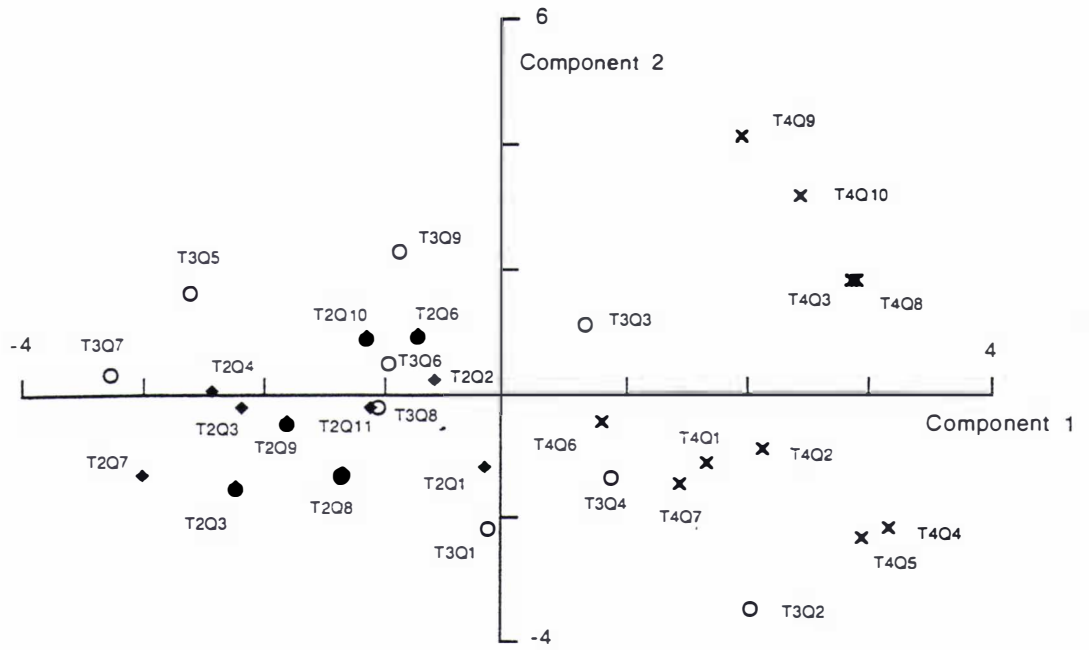

Fig 4.2 *Location of quadrats on first two principal components of site character data. Diamonds are quadrats on transect 2, open circles on transect 3, and crosses on transect 4. Component 1 explains 28.3 % of variance, component 2 a further 20.9 %*

Table 4.3 *Component loadings on first three principal components of site data.*

Variable	Component 1	Component 2	Component 3
Altitude	-0.08	0.38	0.85
Slope	-0.80	0.09	0.40
NS	0.60	-0.16	0.12
EW	0.63	0.31	-0.20
Exposure	-0.69	-0.05	0.08
Closure	0.04	-0.72	0.38
Drainage	0.37	0.73	-0.03
Distance	0.28	0.58	0.62
Ground cover	-0.05	0.65	0.02
Canopy cover	-0.13	-0.81	0.35
Kamahi cover	0.51	-0.07	0.74
Stand age	-0.65	0.17	0.11
Density	0.82	-0.13	-0.04
Kamahi density	0.71	-0.37	0.12

Table 4.4 *Correlations between site dieback variables and principal components 1 and 2 of site data.*

Variable	Component 1	Component 2	Component 3
Dead stems (all species)	0.5736	0.1209	0.1170
Density dead kamahi	0.3519	0.1442	-0.0103
Dnesity dying kamahi	0.2976	-0.4410	-0.0268
Average kamahi health	-0.0876	-0.1658	-0.0745
Dead stems (%)	0.2274	0.3078	0.3079
Dead kamahi (%)	0.1654	0.4471	0.0834
Dying kamahi (%)	0.0929	0.2369	-0.3861

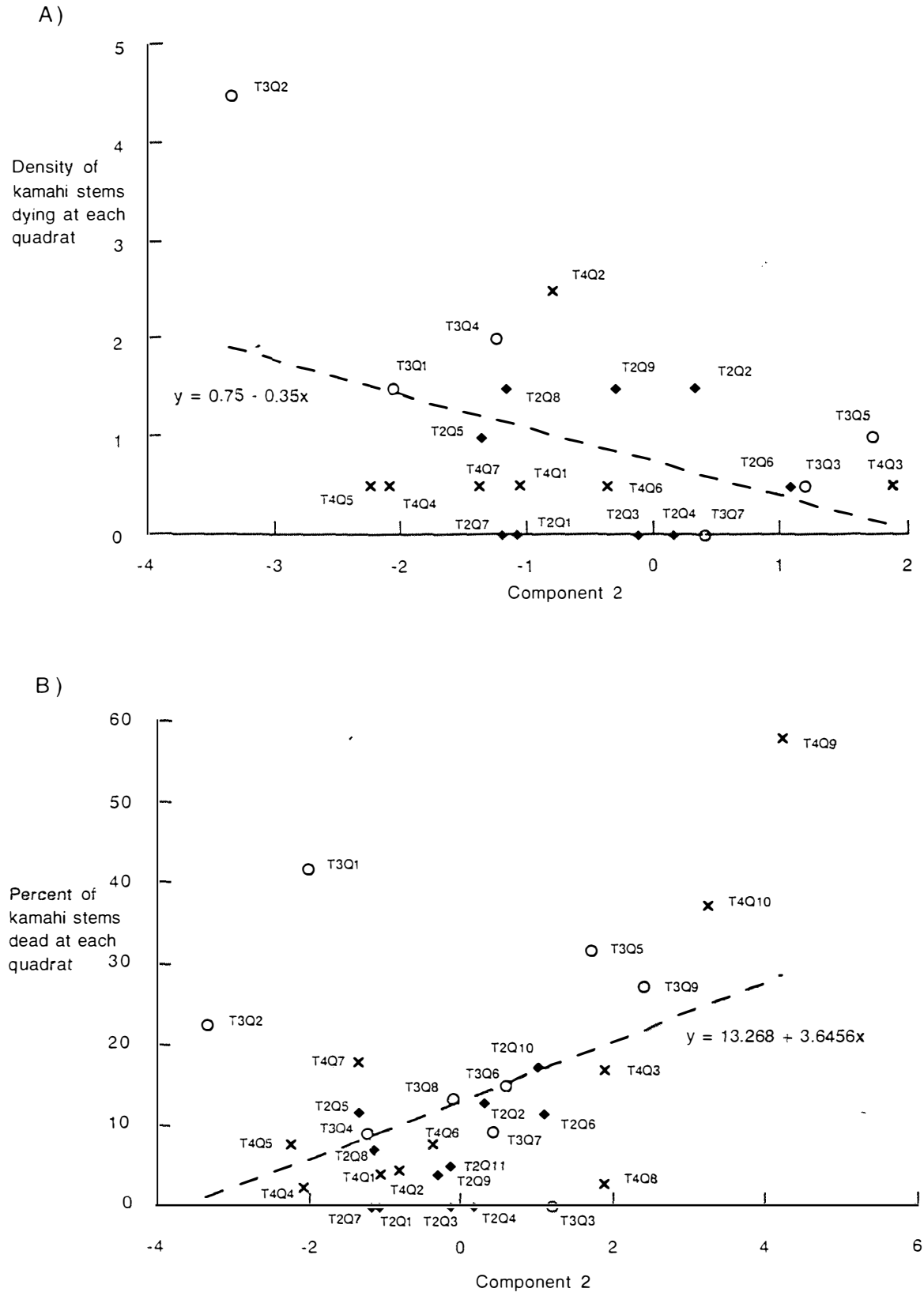

Fig 4.3 *A) Relationship between score on principal component 2 of site character data and A) the density of kamahi stems dying (per 100 m²) at each quadrat (r=-0.44), and B) the percentage of kamahi stems that are dead at each quadrat (r=0.45). Dashed line is linear regression; diamonds are quadrats on transect 2, open circles on transect 3 and crosses on transect 4.*

TREES

The scores for live and dead stems have been separated to compare univariate statistics. 800 kamahi stems were scored in total, with 115 of those dead and the remaining 675 alive. When the distributions are compared, few variables appear similar between live and dead stems; however, the lack of scores for dead stems on some variables has meant it is impossible to prove statistically that they are different from live stems (table 4.5). The DBH of dead stems is significantly higher than that of live stems (t=2.90, P=0.004), with a slightly larger minimum DBH and a much higher maximum (fig 4.4). Both live and dead stems have a much higher frequency at smaller stem sizes, with live stems having a much higher proportion of very small stems (3-13 cm DBH); the shape of the distribution for live stems (with a much larger sample size) describes a decay curve more closely than that of dead stems. For both live and dead trees, most of the units scored represent stems, rather than trees. Crown density is around zero percent for most dead stems (dead leaves may persist), while for live stems the mean is 44%; this was highly significantly different (t=627.1, P<0.0001).

Table 4.5 *Summary of stem data. No standard deviation or mean given for tree/stem or death variables as these data are nominal. See text for explanations of differing counts (N). Asterisks indicate significant differences between live and dead stems calculated by student* t-*test (* $P<0.01$, ** $P<0.001$, *** $P<0.0001$).

	Variable	N	Mean	Median	Std dev	Minimum	Maximum
All stems	DBH*	782	13.88	9.5	13.074	3	103
(N=800)	Tree/stem	796	NA	1	NA	1	2
	Crown density***	674	36	40	23.4	0	85
	Possum browse	631	0.3	0	0.55	0	3
	Insect browse	649	2	2	0.75	0	4
	Deer	382	0.7	0	1.0	0	4
	Borer***	792	0.2	0	0.4	0	1
	Dead shoots	651	3	2	1.9	0	6
	death	71	3	3	1.3	1	5
Live stems	DBH*	672	13.18	9.4	12.146	3	90.4
(N=685)	Tree/stem	683	NA	1	NA	1	2
	Crown density***	565	43	45	18.8	0	85
	Possum browse	622	0.3	0	0.55	0	3
	Insect browse	640	2	2	0.7	0	4
	Deer browse	372	0.7	0	1.0	0	4
	Borer***	682	0.1	0	0.3	0	1
	Dead shoots	536	2	1	1.3	0	5
Dead stems	DBH*	110	18.13	12.35	17.184	3.1	103
(N=115)	Tree/stem	113	NA	1	NA	1	2
	Crown density***	109	0.4	0	2.1	0	15
	Possum browse	9	0	0	0	0	0
	Insect browse	9	1	1	1.0	0	3
	Deer browse	10	0.8	0	1.3	0	4
	Borer***	110	0.8	1	0.4	0	1
	death	71		3		1	5

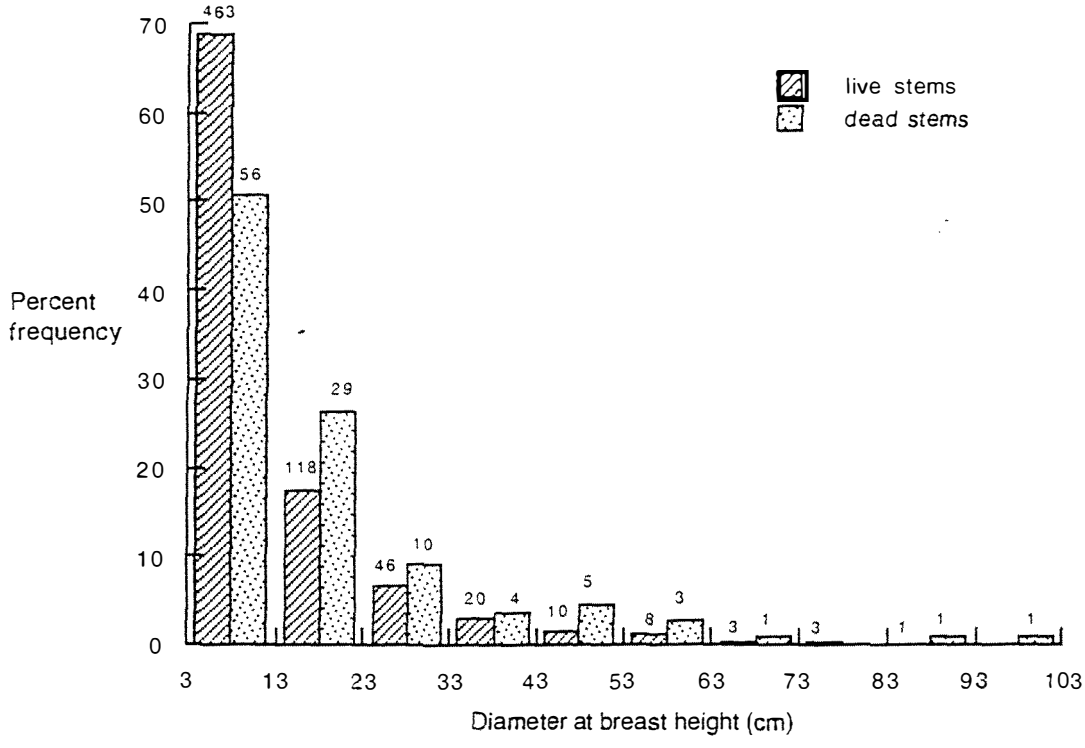

Fig 4.4 *Frequency by percentage of diameter at breast height for live and dead stems of all quadrats. Actual numbers of stems in each size class are given above bars; total number of trees included is 782, with 672 live and 110 dead.*

The level of possum browse ranges from 0-3 (representing 0% and 51-75 % respectively) on live stems, with the median at 0, while for the nine dead stems where possum browse could be scored (i.e. with any dead foliage still attached), none was recorded. Insect browse ranges from 0-4 (4 represents 76-100 % of leaves browsed) on live stems, with the median at 2 (representing 26-50 % of leaves browsed), while for the nine dead stems that could be scored it ranged from 0-3 with the median at 1 (1-25 % of leaves browsed). Ten dead stems could be scored for deer browse; they and the live stems scored from 0-4, with a median of 0; live stems had greater variation around the mean. Most live stems were recorded with no sign of borer, while most dead stems had borer present (t=137.7, P<0.0001). The level of dead terminal shoots recorded for live stems ranged from 0-5 (representing 0 % and 80-99% respectively), with a median of 1 (representing 1-19 %), while all dead stems scored 6 (100 % of shoots appear dead). For the dead stems where an indication of time since death was recorded, the scores ranged from 1-5 with the median at 3 (where main branches and some twigs are still present).

There are several significant correlations when data for all stems are analysed (table 4.6). Crown density tends to be lower on stems with borer present. The score for dead shoots is very closely negatively related to crown density. A high score for dead shoots is associated with presence of borer. When data for live and dead stems are analysed separately, most of these correlations are no longer statistically significant, indicating that outliers from the dead stem data may be driving the relationships. For live stems the only relationship that remained significant is that between the percentage crown density and the score for dead shoots. For dead stems the only significant relationship was between deer browse and crown density.

Table 4.6 *Significant correlations between stem variables (where* $|r|>0.5$ *and* $P<0.005$*). All correlations with possum browse have been excluded for the dead stem group because all nine dead stems where a score was recorded had 0 possum browse.*

	Relationship	d.f.	r-value	P-value
All stems	Borer x crown density	666	-0.56	<0.0001
	Dead shoots x crown density	534	-0.86	<0.0001
	Dead shoots x borer	644	0.64	<0.0001
Live stems	Dead shoots x crown density	425	-0.68	<0.0001
Dead stems	Deer browse x crown density	8	0.86	0.0033

DISCUSSION

Dead and dying trees are a common part of New Zealand forests (Jane & Green, 1983), and have been for some time (Holloway, 1957). Because kamahi is such an important canopy species, its dieback is often very visible (figs 5.1 and 5.2). When this occurs in national parks, public pressure for action (and occasionally explanations) tends to ensure an official response (Hosking, 1993a). This study of kamahi in Tongariro National Park focuses on the areas where dieback is visibly apparent, and uses the 'decline disease' theory of dieback to attempt to narrow down the possible causes.

The survey covers 30 quadrats on three transects within this area, amounting to 6000 square metres. Quadrats include a range of altitudes within the limits of kamahi distribution in this area, from near the forest edge to well within the forest, a wide range of slopes, a range of the westerly aspects, medium and low exposure levels, high and low canopy closure and cover as measures in this study, good to poor drainage, a wide range of canopy heights, stand ages and kamahi cover. Mean scores for dieback at each quadrat, and numbers and proportions of dead and dying kamahi stems were compared to these site factors to determine their effect. In most cases, significant correlations would indicate the more likely predisposing factors, but for some, such as canopy closure, may also indicate a possible hastening factor.

The 800 kamahi stems included in the study ranged widely in diameter at breast height (and therefore probably in age), in level of insect or possum predation, and some were attacked by pinhole borer while others were not. Significant correlations between any of these variables and either of the measures of stem health (crown density or dead terminal shoots) would indicate likely triggering or hastening factors.

Results from stem data show that dieback is affecting a reasonable proportion of kamahi stems in this study with approximately 14 % dead, and a further 6.7 % dying. Very few data are available to indicate the proportion of dead and dying trees that would be expected in healthy forests. Coleman *et al.* (1980) found mortality rates of 0 % to 29.5 % for kamahi in Westland depending on the forest type. Skipworth (1981) studying Mountain Beech (*Nothofagus solandri* var. *cliffortioides*) in West Ruapehu found mortality rates from 7 % to greater than 50 %. However, both these studies were investigating mortality because of a perceived problem, so although the observed incidence of standing dead stems in the present study falls within their ranges of values, it is not necessarily normal.

Fig 5.1 *margin of forest at base of Hauhungatahi. Note the grey dead trees (photo G.Rapson).*

Fig 5.2 *Forest at edge of airstrip, Erua Rd. Note the patch of dead kamahi in the canopy.*

PREDISPOSING FACTORS

There were few significant relationships between any of the suggested predisposing factors and any measure of stem or stand health.

There was no correlation between health and diameter at breast height (a measure of age), but there was a significant difference in the distributions of diameter at breast height for live and dead stems, with dead stems having a significantly larger DBH, and live stems much more likely to be in the smallest size class than dead stems. Dead stems were over represented in the second smallest size class compared with live stems. The general shape of the frequency distribution curve, particularly for live stems, agrees closely with that shown by Lusk and Smith (1998) for kamahi; their study, including 943 kamahi stems, gave kamahi distribution a perfect reverse-J decay curve for size frequency. The small sample size of dead stems for which DBH was measured (N=110) makes it difficult to comment on the different size distributions, and the gaps in the frequency distribution suggest that a larger sample size would be necessary for testing hypotheses with any confidence. However, sample size is only likely to be affecting resolution in the rarer large size classes, while the general shape of the curve in the lower size classes, which are adequately represented here, should remain largely the same. The smallest size class (less than 13 cm DBH) may represent stems before competition becomes so important in thinning out young stems (Peet & Christensen, 1987) in these stands, while the second size class (over 13 cm up to and including 23 cm DBH) may represent the age where competition severely thins the cohort. Harcombe (1987) predicts that in healthy forest, mortality should be evenly distributed among larger size classes, and this prediction appears to be met in this study with the above caveat of sample size: for dead stems large size classes are only represented by a single stem. Coleman *et al.* (1980) in their study of forest mortality on Mt Bryan O'Lynn, Westland, found no significant difference in size for live and dead kamahi stems (N=1169). They excluded small stems (< 20 cm DBH) from their analysis; when data from the present study are treated similarly the sample size becomes very small (29 dead trees, 111 live) and the difference between DBH of dead and live stems is no longer significant (mean dead = 40, mean live = 35; F=2.5, P>0.05). Franklin (1987) suggested this to be normal, commenting that although there may be no real limit to how long a tree may live, reduced growth and vigour may decrease its ability to resist or recover from stresses. These results also agree with Harcombe (1987) and Peet *et al.* (1987) in showing that most of the dead stems are young, and stems are represented at similar rates in both the live and dead frequency

distributions, *i.e.* they die at a rate proportional to that at which they occur in the overall population.

There is no evidence from this study that stand age plays any role in kamahi dieback, *i.e.* all measures of health at quadrats varied independently of stand age. Stewart and Rose (1988), in highlighting the importance of stand age in the pattern of kamahi dieback, state that in Westland there is no evidence of dead rata-kamahi stands dominated by small even-sized stems (< 35 cm DBH); they suggest that these stands are less susceptible due to the seral species present and high plant vigour. None of the stands examined in the quadrats of the present study could be considered dead. The quadrat with far and away the greatest percent of kamahi stems dead was quadrat 9 on transect 4 (57.9 % kamahi dead); at this quadrat, three kamahi stems were greater than 30 cm DBH, with the largest 35.7 cm.

Stand density also does not appear to be involved in kamahi mortality in this study. Stand density is largely another measure of stand age.

This study found no effect of slope or altitude. Rogers and Leathwick (1997) found stands with slope > 20° were far more susceptible to forest collapse. In the present study, eight quadrats had slopes greater than 20°, and the percentage of kamahi stems that were dead in these ranged from 0 - 32 %. While 32 % was one of the highest percentages of dead kamahi, the greatest slope of the three quadrats with more dieback was 2°. They also found forests in the upper montane-subalpine zone were more susceptible to collapse; all the quadrats in the present study are within a relatively narrow upper lowland-lower montane altitudinal range. Higher levels of dieback were just as likely to occur at the lower limits of this range, as they were at the higher limits.

There was a barely significant correlation between the east-west component of aspect and the average kamahi health at a quadrat: more westerly sites were found to have better health. This result was also found by Rogers and Leathwick (1997), who for their area in the Southern Ruahine Range, postulate that it is due to the greater warmth, but lower humidity on western aspects. In the present study of the western slopes of Hauhungatahi, the least westerly aspect recorded was 210°. The more westerly aspects are likely to receive sun for a greater part of the day than any deviations from west, allowing greater photosynthetic potential. As Rogers and Leathwick (1997) suggest, this may enable stands to be more resilient to any adversity.

Neither exposure nor distance from forest margin (with nearly all but 2 sites within 1 kilometre, and most less than 500 m, from the edge) had any effect on kamahi

health in the present study. In Payton's (1988) study of rata-kamahi forest in Westland, he found that trees were most susceptible to dieback at exposed sites at the edge of the forest. In the present study, the site closest to the forest edge (transect 3 quadrat 1, 75 m from edge) does have a very high percentage of dead kamahi stems (41.9 %), but the site with the highest level of kamahi mortality (transect 4 quadrat 9) is 1 kilometre from the forest edge. While not highly exposed, many of the plots on transect 4 are a relatively great distance from the edge of the forest, but relatively close to the Makatote Gorge (only a few hundred metres). This study includes no sites very close to the forest edge, but from the road which runs parallel to the western edge of the forest, it does appear that trees within approximately 10 m of the edge are very unhealthy. Even so, our data do not indicate a higher susceptibility to dieback at sites closer to the forest margin.

There was a significant relationship between drainage score and percent of kamahi stems that were dying at each quadrat; however this relationship depended on one anomalous site, where kamahi was clearly not at its best (transect 3 quadrat 3, with a single dying kamahi).

Neither percent of the canopy that is kamahi nor whether trees are multi- or single-stemmed affected the health *of* kamahi in this study.

A strong correlation was found between canopy height and percent of kamahi stems dying. Again this relationship was dependent on the one anomalous site, and when this was removed from the analysis, there was no relationship.

The principal component analysis, which summarises the site character data into a few variables explaining most of the variation, offers no further insight. Quadrats do not cluster according to health when they are mapped on the two longest components. There is a nearly significant relationship between the number of kamahi stems dying at a quadrat and its score on component 2. Again this relationship is dependent on one quadrat with a much greater density of dying kamahi stems; removing this site from the analysis shows the variation to be entirely random. There is another nearly significant relationship between the percent mortality of kamahi stems and score on component 2 for each quadrat, which is dependent on the quadrat with the highest score for percent dead kamahi, and without which is nowhere near significant. Thus, principal components are also unable to establish any possible factors involved in kamahi dieback from the data collected in this study.

TRIGGERING AND ACCELERATING FACTORS

These two classes of dieback factors are being discussed together, as most accelerating factors are also capable of triggering dieback in different circumstances, and *vice versa*. Again results from the present study offer no clear answers.

There is no relationship between recorded levels of possum browse and either measure of stem health used in this study (crown density or dead terminal shoot class). Department of Conservation (unpub.) data gives the trap catch rates for possums on transect 2 as 20.0 %, transect 3 as 3.4 %, and transect 4 as 11.8 %. Transect 2, with the lowest incidence of dead kamahi stems (8.5 %), clearly has the highest recorded rate of possum catch, while transect 3 has the lowest recorded incidence of possums, and the highest rate of dead kamahi stems (22.6 %). While these results can easily be explained as possums building up in numbers, eating an area to death and moving on to a new healthy patch, and thus having high densities at healthy areas (which they are about to annihilate) and low densities at unhealthy areas (which they have already destroyed), there is clearly no evidence from this study to resort to such speculation. Interestingly, the level of possum browse recorded and trap catch rates for possums on the transects are not related. There are three likely explanations: trap catch rates are a poor estimation of possum density or usage in an area, as suggested by Batcheler *et al.* (1967); our method for estimating levels of possum browse in the canopy is poor (for any or all of the reasons given in the introduction to this chapter); or, possum density is only one factor in the level of possum browse on kamahi in this area, other factors may be of enough importance to disrupt the relationship. Although there are many reasons why this study may have missed finding a positive result for possums and kamahi mortality, the fact is that there is no evidence from the present study to suggest that possums are causing kamahi dieback, or accelerating any dieback caused by other factors. Many studies of dieback in other New Zealand forests have found possums are an important factor in mortality of some species, often including kamahi, in those forests. In some of these studies, evidence has been largely circumstantial (as discussed by Leutert, 1988) and few have focused on kamahi.

There is no relationship between insect browse and stem health in the present study. In New Zealand, insects have generally only been suggested as accelerating factors in tree mortality (e.g. Payton, 1987); however there is no evidence from the present study to suggest this for kamahi in Tongariro National Park.

There is clear evidence supporting the hypothesis that *Sporothrix* is a contributing factor to kamahi mortality. When all stems are included in analysis, pinhole borer, the vector for *Sporothrix* fungus, is significantly more likely to be

present when crown density is low, and when level of dead shoots is high. When only live stems are included, these relationships are no longer significant. When only dead stems are looked at, these relationships are again not significant; however, this is because dead stems all have a dead shoot score of 6, and have very low crown densities (i.e. only 4 % of dead stems have a crown density greater than 1 %), while 82 % of dead stems contain borer. That this relationship breaks down when dead and live stems are analysed separately, combined with most dead stems containing borer, strongly implies pinhole borer or *Sporothrix* are hastening factors rather than actually causing any decline in health. This adds support to those authors who have suggested *Sporothrix* as a symptom rather than a cause of declining health (particularly Hosking, 1993a; 1993b).

Canopy closure, as measured in this study, was not found to be related to kamahi health. Several authors have found that it may play a role in dieback (mainly Payton, 1988; Rogers & Leathwick, 1997), but with the few quadrats in this study with very high levels of kamahi dieback, it is unlikely even if a relationship existed that it would be found.

CONCLUSIONS

The only factor this study has any evidence to suggest as predisposing kamahi stems to dieback, is age, as measured by stem diameter at breast height, with older stems more susceptible than younger stems. Even for this factor, the evidence is not compelling, and further investigation is necessary to confirm its importance . This study provides further support to the hypothesis that *Platypus* tend to invade trees that are in poor health, and any *Sporothrix* infestation only serves to amplify the decline in health. This study fails to find any link between site factors and tree health. It also fails to find any 'triggering' or causal factor of kamahi dieback. There is certainly no evidence from this survey to suggest possums are having an impact on the health of kamahi in this study area.

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APPENDIX A

APPROXIMATE LOCATION OF TRANSECTS

Grid references from Infomap 262-6

Transect	Origin	Magnetic bearing	Transect length
2	717217	30°	1100 m
3	717216	60°	900 m
4	718213	55°	1000 m

APPENDIX B

Method used for allocating Stand Age classes. This method assumes that diameter at breast height is closely related to stem age (see text), and that this relationship is fairly constant over the altitudinal range of this study (approximately 170 m). The use of broad size classes should lessen any effect of altitude.

1. Frequency histograms are plotted of diameters at breast height of all kamahi stems in each quadrat using broad size classes (30 cm).
2. Linear regression lines are fitted through the frequency values including up to the largest size class represented at that quadrat (*i.e.* not including any empty size classes unless there are also larger sizes represented).
3. In this study, There were eight quadrats where stems from only one size class were represented (and therefore no regression line could be plotted). In these cases all stems were in the 3-30 cm size class, and those quadrats could therefore be allocated to the youngest stand age class (class 1).
4. Slopes of the regression lines were plotted on a stripe graph, and the natural groupings determined by eye (natural groupings are where values tend to fall into discrete 'clumps', or where a value is considered quite distinct from other data).
5. Nine groups were determined, and these were numbered from 1 (youngest) to 9 (oldest); younger age classes were much more strongly represented.
6. The quadrat with the greatest slope was so far removed from the next greatest slope that it was decided to include that quadrat with the youngest stand ages.

Chapter 2: Discussion of the two surveys

Understanding the extent of any mortality is critical to applying the models of dieback, and for this a broad scale survey is required. The broad scale survey of kamahi mortality in Tongariro National Park concluded that although there were patches of seemingly high kamahi mortality, overall it appeared to be in a healthy state, with 4 % of kamahi trees dead. For a more detailed examination of the demographics of kamahi mortality and the likely causes, a finer scale approach focussing on areas of high mortality is necessary. The second survey, in an area where approximately 14 % of kamahi stems were dead, found that older stems may be more susceptible to dieback, but could not identify a proximal cause.

These surveys differ substantially in their overall presentation of the extent and severity of kamahi dieback in the Park. This could be due to the extreme patchiness of the mortality, but even where sites from the two surveys were close, results were often disparate. In the broad scale survey of kamahi health (chapter 2a), the two sites with the highest levels of dieback were approximately 500 m and 700 m from where the Manga-nui-o-te-ao River leaves the forest (with 40 % and 30 % of their ten kamahi trees (not stems) dead respectively). These sites are several kilometres further south than any of the quadrats in the survey of dieback areas (chapter 2b). Another site (chapter 2a) had 20 % of its 10 ten kamahi dead, and was located near the forest margin approximately 150 m south of Erua; this was very close to the origin of transect 2 of the survey focusing on areas of dieback, where 0 % of (16) kamahi stems were recorded as dead at the quadrat closest to the forest edge. Of the thirty sites included in the broad scale survey, 13 are within the area of apparently high dieback targeted in the chapter 2 survey. Of these 13 sites, only three had any dead kamahi present: two had 1 (of ten) dead trees, and one had 2 (of ten), and there was a total 3 % of kamahi trees dead. On transect 2 of the chapter 2b survey, which is close to 6 of the sites of the broad scale survey (with 7 % of trees dead), 8.5 % of kamahi stems are dead. On transect 3, which is close to 5 of the sites of the broad scale survey (with 6 % of trees dead), 23 % of kamahi stems are dead. On transect 4, close to 7 sites in chapter 2a (with 1 % of trees dead), 12 % of stems are dead. It appears to be random chance that the sites in chapter 2a which are near transect 2 of chapter 2b recorded similar levels of mortality. These data suggest that mortality is very 'patchy' in Tongariro Park and surrounding areas.

The different levels of dieback recorded in the two surveys could be due to any or several of a number of factors. There is a difference in the way death was recorded

in these two surveys which would certainly lead to a higher incidence of kamahi mortality in chapter 2b than chapter 2a. The survey of areas of high mortality (chapter 2b) scored and recorded each stem of multi-stemmed kamahi individually, whereas the broad-scale survey scored all stems of a tree together as a single tree. If one or more stems of a multi-stemmed tree were alive, that tree would be scored as living in chapter 2a (regardless of how many stems were dead); while in chapter 2b, dead stems would be recorded as dead, and live as alive. Unfortunately, from the data that were (or were not) collected in each of these surveys, there is no way to correct for this difference, or to predict the severity of its effect, except that sites with few multi-stemmed trees or with a lower stem frequency of dieback should be less affected by this methodological difference, than sites with many multi-stemmed trees or higher levels of dieback.

The other difference in methodology that would have affected the frequency at which dieback was recorded, is the different minimum diameter at breast height for each survey. The survey of areas of high mortality (chapter 2b) included stems to a minimum of 3 cm DBH, whereas the broad-scale survey only includes trees with a DBH greater than 10 cm per stem. When stems with a DBH less than 10 cm are excluded from the chapter 2b data, the percentage of kamahi stems that are dead remains largely the same as when all stems are included for transects 2 and 4, but on transect 3, 34 % of kamahi stems greater than 10 cm in diameter at breast height are dead. Clearly this does not explain the difference in frequency of kamahi death recorded in the two surveys, but it does further highlight transect 3 as having high stem mortality.

Another possible explanation for the difference in rates of dieback recorded in each survey is the patchiness of that dieback. For example, transects 2 and 3 of the chapter 2b survey recorded very different rates of dieback (8.5 % and 23 % respectively) despite being located only approximately 1000 m from each other. Even within transects dieback was extremely variable: on transect 4, quadrat 8 recorded 3 % of stems dead, whereas quadrat 9 (100 m from quadrat 8) recorded 58 % of stems dead. With this degree of variability in mortality levels, it is possible that one survey has missed healthy patches, or 'hit' a disproportionate number of unhealthy patches, or that the other survey struck a disproportionate number of healthy patches and missed unhealthy ones.

Therefore the different levels of dieback recorded in each survey certainly reflects the different methodology used in each survey, with the method used in chapter 2b *guaranteed* to find higher rates of mortality in a large survey, and may also reflect the patchiness of the dieback, with transect 2 (and the first 8 quadrats of transect 4)

of chapter 2b and all but one or two sites of chapter 2a missing the patches of heavy mortality (or transect 3 chapter 2b missing the patches of healthy kamahi).

There is evidence of localised high levels of kamahi mortality, but overall kamahi appears to be extremely healthy.

Chapter 3: An experimental approach to determine effects of possums, pinhole borer, and *Sporothrix* on kamahi health

ABSTRACT

In order to test the possiblity that possum browse, attack by pinhole borer, or infection by *Sporothrix* fungus may be causing the kamahi dieback observed in Tongariro National Park, their impact was examined in an experiment conducted in nearby Erua State Forest. Trees were banded to exclude possums, had holes drilled in them to simulate pinhole borer attack, and had either *Sporothrix* fungus introduced or were inoculated with fungicide. The effects were monitored by measures of litter fall and observations of health and damage in each tree. Data are summarised by principal components, which have been tested for treatment effects. Cluster analysis has been performed on data to assess whether samples are grouped by treatment.

Insect browse was common in all trees irrespective of treatment, possum browse was rare. The only treatment that analysis could detect was whether trees were healthy or unhealthy, which was predetermined at the outset of the experiment. None of the treatments had any effect on the health of trees in this experiment. It is concluded that there is no evidence to suggest that possums, pinhole borer or *Sporothrix* are important in determining the health of trees in this area.

INTRODUCTION

The western side of Tongariro National Park is largely forested, and at lower altitudes, particularly less than 1000 metres above sea level (m asl), the forests are dominated by kamahi (*Weinmannia racemosa*) associations (Atkinson, 1981; Atkinson, 1985). Dead trees are a common feature of these forests, and concern has been raised over the health of kamahi in this area, particularly because at forest edges, which are highly visible, there is often a strip of dead or extremely unthrifty kamahi. The prime suspects in this observed dieback are the Australian brushtail possum (*Trichosurus vulpecula* Kerr), and a *Sporothrix* species fungus (Deuteromycotina: Hyphomycetes) with *Platypus* spp. Herbst pinhole borer (Coleoptera: Platypodidae) as its vector.

POSSUMS

Possums (Marsupialia: Phalangeridae) are small-ish mammals, averaging around 2-3 kilograms in their native Australia (Statham, 1995). Statham (1995) studied possums in Tasmania, where they are indigenous and widely distributed through forest, agricultural and urban areas, reaching highest densities in dry sclerophyll forests, and woodlands near improved agricultural land. There, the availability of improved pasture has allowed possum populations to increase over the last few decades, and herbs, especially clover, were found to be the preferred food group.

After much effort, possums were successfully introduced to New Zealand. They now number around an estimated 70 million (Livingstone & Nelson, 1993) and utilise a wide range of habitats (Coleman, Green & Polson, 1985). Unmodified New Zealand 'bush' is probably not their ideal habitat, and possums seem to prefer exposed ridges, allowing easier traveling and a drier microclimate (Leutert, 1988). Higher densities also occur where the lower vegetation layers have been reduced, providing easier access to food and nesting (McKelvey, 1959; Cunningham, 1979). It is at these higher densities that possums tend to cause most problems, in part due to their feeding habits. Possums' diets are diverse, largely reflecting the diversity of their habitats (Coleman *et al.*, 1985). Mason (1958) commented that there is also much variation in food preference between localities (confirmed by Coleman, Gillman & Green, 1980). However, possums tend to feed selectively within each locality (Livingstone & Nelson, 1993), concentrating their efforts on particular species, and often on individual plants, while available.

Possum impact is reported to be most severe in podocarp/mixed hardwood forest associations (McKelvey, 1959; Leutert, 1988), particularly those dominated by rata and kamahi (Fitzgerald & Wardle, 1979). This is considered to be due to the

preponderance in these associations of seral shrub hardwoods, which are highly palatable to possums (Coleman *et al.*, 1980; Stewart & Rose, 1988). Coleman *et al.* (1985), studying the diet of possums on a pasture-alpine gradient, found that 88 % of foliage eaten came from woody species, and 69 % of this from three canopy species, kamahi, southern rata (*Metrosideros umbellata*) and mahoe (*Melicytus ramiflorus*). Kean (1953) speculated that the effects of possum browse are masked by their strong preference for particular species, such that local extinctions of these species may occur, but the forest could remain healthy-looking. Local extinctions have occurred with highly preferred species such as fuchsias and mistletoes (Livingstone & Nelson, 1993). The effect of possums overall is complicated by the variation in diet preference found in different areas.

Cowan (1990) notes that the partial replacement in the diet of foliage by fruits is associated with the build up of fat reserves in late summer and autumn. Studies have found evidence of many fruits in faeces seasonally, but were not able to estimate the proportion of total diet these represent (Coleman *et al.*, 1985; Cowan, 1990). Fitzgerald and Wardle (1979) comment that although fruits, seeds and invertebrates are eaten in small quantities overall, due to the quality of these foods, they probably reduce the total amount of food ingested.

POSSUMS AND KAMAHI DIEBACK

Despite the low crude protein value for kamahi foliage of less than 6 % (*c.f.* pigeonwood at 17 %, or grasses at higher still (Fitzgerald, 1976)) kamahi is generally a preferred food species of possums (e.g., Fitzgerald, 1976; Pekelharing, 1979). In the Orongorongo valley, kamahi and rata made up 60 % of the foliage eaten by possums (proportion of these species in the canopy of the areas studied is not discussed) (Fitzgerald, 1976). Fitzgerald and Wardle (1979) found kamahi to be one of the main species eaten in South Westland, where visible deterioration of kamahi was occurring. In Wardle's (1974) study in (northern) Westland, possum use was greatest in kamahi associations, which were also most susceptible to possum damage. And Cunningham (1979) blames possums for the large scale deterioration of kamahi and other canopy dominants in the Ruahine Range. However, Coleman *et al.* (1985) found that although 38 % of all foliage eaten in their Westland study was from kamahi, kamahi made up 46 % of the forest basal area, suggesting it was not selected for.

Much of the evidence linking possums to dieback is circumstantial, based on the correlation between mortality and possum 'infestation' (Leutert, 1988). It is equally

possible that possums occur in higher numbers at sites susceptible to dieback from other causes. For example, in Australia, forests adjacent to pasture are capable of sustaining higher densities of possums due to the availability of food such as clover (Statham, 1995). Conversely, possums might feed preferentially on stressed trees if the stress, for example insect defoliation, resulted in lower concentrations of secondary compounds affecting palatability (Payton, 1983). Superimposed on this dearth of causal evidence is our lack of knowledge of baseline mortality rates in unmodified New Zealand forests (Rose, Pekelharing & Platt, 1992).

SPOROTHRIX AND PINHOLE BORER

Platypus attacks have been recorded for a taxonomically diverse range of trees in New Zealand, from the monocotyledon ti (*Cordyline* spp.) (Grehan & Nixon, 1978), through gymnosperms such as the exotic *Pinus*, and angiosperms, such as beech (*Nothofagus* spp.) (Milligan, 1972) and kamahi (Payton, 1989).

Milligan (1972) describes the behaviour of the three species of pinhole borer associated with beech. A male makes a radial entry into a live tree, and releases pheromones, which attract females and/or males of the same species. The release of these 'aggregating' pheromones after successful entry to the tree may further concentrate attack on selected trees (Payton, 1989). Milligan (1974) recorded maximum density of holes as approximately 13 per 100 cm^2.

Copulation occurs at the tunnel entrance, and the female continues to bore radially into the tree while the male ejects the frass (this often being the most visibly obvious sign of infestation, fig 2.1). The female burrows through a right-angle until the tunnel is tangential near the sapwood/heartwood boundary; at the end of this tunnel the first batch of eggs are laid in mid-summer. The female retreats to the curved portion of the tunnel and bores another radial branch in the opposite direction where she lays the second batch of eggs in late summer. Eggs hatch over winter, and larvae spend approximately a year tunneling in the wood, cultivating and feeding on yeast and fungi (Milligan, 1979).

Milligan (1972) found a dark stain spreading from *Platypus* spp. tunnels in beech. Such staining has also been found associated with pinhole borer in ti and kamahi (Grehan & Nixon, 1978; Payton, 1989 respectively). The staining was found to be caused by the presence of a pathogenic fungus, the conidial stage of which was identified as *Sporothrix* species (P. Gadgil cited in Faulds, 1973).

Fig 2.1 *Bark of tree infested with pinhole borer, note lighter coloured frass ejected from tunnels on darker bark (photo G.Rapson).*

Sporothrix seems to prefer drier wood, becoming established initially in the drier, innermost sapwood or heartwood, and then spreading radially to the limits imposed by wood moisture and defensive reactions of outer sapwood (Milligan, 1974). Faulds (1973) observed that stained sapwood seemed to dry out more quickly than unstained wood at cut surfaces, due to water transport being impeded. Faulds (1977) concluded that *Sporothrix* kills trees by interrupting the xylem, and thus water flow to the crown; this is typical of vascular wilt pathogens such as Dutch Elm Disease (*Ceratocystis ulmi*).

Pinhole borer is well suited as the vector for *Sporothrix* fungus: although pinhole borer are not restricted to unhealthy trees (Milligan, 1972), moisture stress, in particular, tends to predispose trees to attack. *Sporothrix* requires entry to the transition zone of the wood, which is where pinhole borer tend to concentrate (Milligan, 1972); and, although *Sporothrix* is quite capable of invading live tree tissue (Faulds, 1973), a stressed tree is less able to overcome the infection, and pinhole borer tend to attack stressed trees. Milligan (1974) speculated that the aggregating hormones of *Platypus* also add to the success of *Sporothrix* attack, increasing the intensity of attack by insect, and thus by fungus.

Wardle and Allen (1983) found that in beech the pinhole borer was often, if not usually, associated with dieback; and, in all aforementioned species where *Sporothrix* impacts have been studied, it has been found that successful infection will kill even healthy trees (Faulds, 1977; Payton, 1989). The key to success of the fungus seems to be water stress, as noted by Milligan (1974) who found that trees only lightly or abortively attacked by pinhole borer succumbed to the fungal pathogen when a drought occurred the following summer; and Faulds (1977) speculated that drought turned sub-lethal inoculations into lethal during his experiments.

SPOROTHRIX, PINHOLE BORER AND KAMAHI DIEBACK

Questions have been raised over the inferred causality in the relationship between death of kamahi and presence of pinhole borer implying presence of *Sporothrix*. Payton (1989) undertook a series of experiments in Westland to test the relationship. He induced pinhole borer attack by stacking dead wood infested with pinhole borer around six apparently healthy kamahi trees; this produced maximum densities of infestation of 1.5 to 2.5 holes per 10 cm^2, well below the maximum level noted by Milligan (1974). All but the largest tree died, and *Sporothrix* was isolated from all five when felled; when *Sporothrix* was added to a further 10 apparently healthy trees, all but one died within 16.5 months, and all of the control trees remained healthy.

These experiments were based more or less on work focusing on *Sporothrix*, pinhole borer and beech mortality by Faulds (1973; 1977) who found much the same relationship.

AIMS

Observational and comparative data using 'natural experiments' provide only weak, correlative, inferences. The scale and effort required of field experiments often precludes true replication, causing statistical implications not relevant in tighter controlled laboratory- or glasshouse-type experiments (Hurlbert, 1984); field experiments do, however, allow true controls, knowledge of initial, pre-treatment, conditions and manipulation of the variables of interest (Gurevitch & Collins, 1994). In investigating a complex, and probably multi-factor phenomenon such as dieback, field experimentation is the only technique that can provide replicable conclusions of causation (Manion & Lachance, 1992).

A multi-factorial field experiment to investigate the relationship between possums, pinhole borer and *Sporothrix* impacting on kamahi health, was conducted in Erua State Forest, which is predominantly rata/kamahi forest. Trees in either apparently healthy and apparently unhealthy states were used. Possums were excluded from trees, and allowed access to others. Pinhole borer attack was simulated, as much as possible, by drilling holes in some trees, and not in others. In half the trees where holes were drilled, holes were filled with sterile distilled water; the remainder were inoculated with *Sporothrix*. In trees where the health was suspected to be affected by *Sporothrix* already, half were inoculated with a fungicide, in an attempt to eliminate the fungus, while the remainder were inoculated with water. Health was measured by visual assessment.

STUDY SITE

The study site is approximately 15 kilometres west of Erua settlement (fig 3.1). It is to the west of Tongariro National Park and is situated in the Erua State Forest, close to a road servicing two farms. The forest was cut-over in the early part of this century. The site was chosen due to the quantity of healthy and unhealthy trees within a small area of bush. This was ideal, as this experiment requires 50 trees in similar environmental conditions. Being outside the National Park allowed experimentation of a more destructive nature than would otherwise be possible. The study site is divided into two sub-sites, "healthy" and "unhealthy". The main tree species is kamahi mixed with other broadleaves. Many of the kamahi trees are apparently of similar age, possibly due to synchronous establishment following a major disturbance.

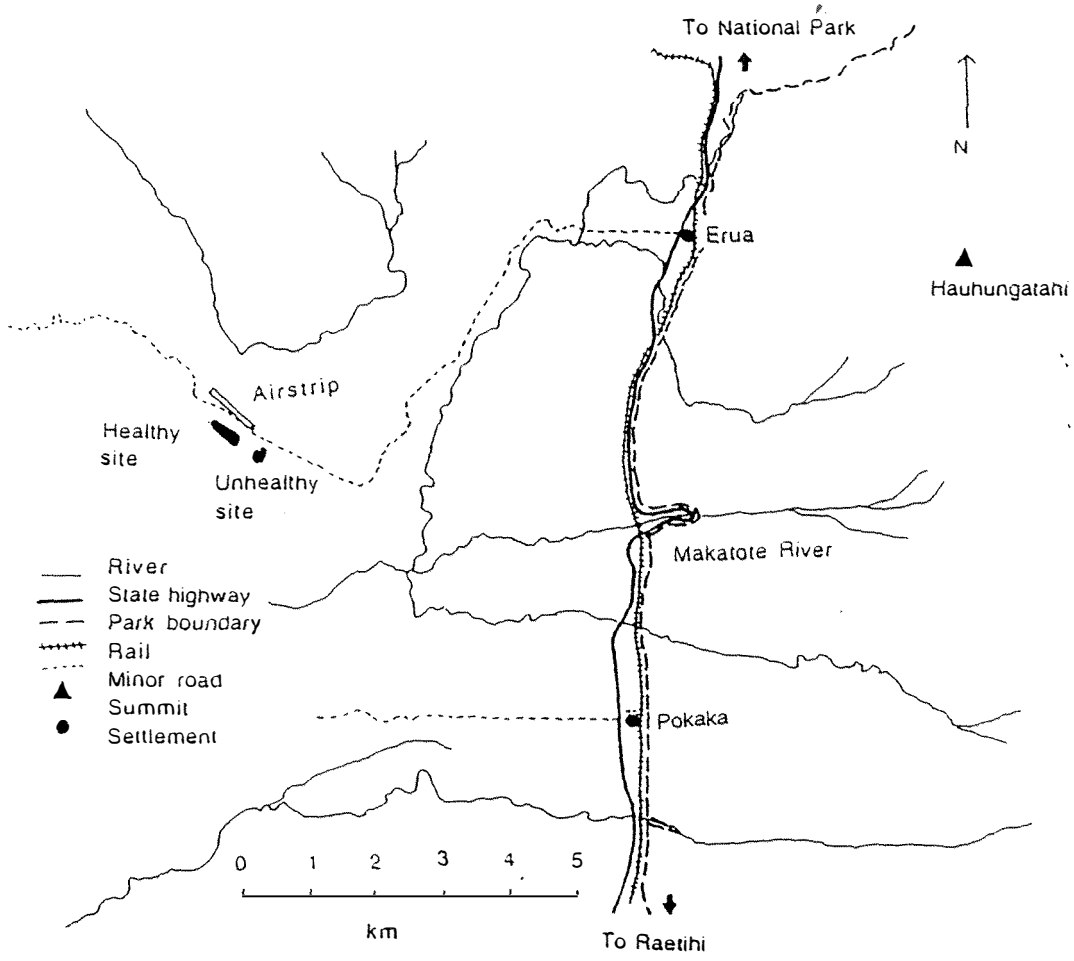


Fig 3.1 *Location of study site off Erua Road.*

METHOD

Thirty apparently healthy kamahi were chosen within the healthy sub-site, and twenty apparently unhealthy within the unhealthy site. Trees were haphazardly allocated to remaining treatments, with five replicates per treatment. The treatments were:

Healthy

a) unbanded, no holes, no fungus

b) unbanded, holes, no fungus

c) unbanded, holes, fungus

d) banded, no holes, no fungus

e) banded, holes, no fungus

f) banded, holes, fungus

Unhealthy

g) unbanded, no fungicide

h) unbanded, fungicide

i) banded, no fungicide

j) banded, fungicide.

HEALTHY SUB-SITE

Banding

Banding of the healthy site was undertaken on September 10 and 11, 1994. Aluminium 'Possum Guard' of the dimensions 0.44 mm thick x 590 mm wide was used for all trees. The circumference of the tree at approximately 1.5 m from the ground was measured (diameter at breast height or DBH), and the appropriate length of aluminium was then wrapped around the trunk and attached with galvanised nails. Where the trees had multiple stems, each stem was banded individually, with each band on the tree at approximately the same height to dissuade possums from jumping to stems from others banded higher up. Care was taken to ensure all trees whose canopies overlapped with banded trees, or from which a possum may gain access to a banded tree, were either themselves banded or tied-back to limit this access (fig 4.1 and 2).

Fig 4.1 *Kamahi tree banded with aluminium possum guard. Note funnel-shaped litter fall trap in middle foreground.*

Fig 4.2 *Study site with many trees banded to exclude possums, and litter traps positioned to collect any litter falling from the canopy.*

Fungus

The fungus, *Sporothrix*, was grown from specimens obtained from W. Faulds at New Zealand Forest Research Institute. The fungus was grown in a medium of 1.5 g KH_2PO_4, 1 g $MgSO_4.7H_2O$, 25 g glucose, 2 g asparagine, 2 g mycological peptone, and 2 g yeast extract, dissolved in 1 litre of sterile distilled water in mixed shake culture in six 250 ml flasks at 17°C for six days, from 25th July, 1994 (as described by Faulds, 1973). The mycelia were centrifuged out of the medium, washed with sterile distilled water, resuspended in 4 litres sterile distilled water and stored in sterile McCartney bottles and sealed flasks before use in the field the following day.

Holes and Inoculation

A thirty centimetre wide band was marked on the stem(s) of the tree to receive holes, at a minimum height of approximately 1.3 m, and to a maximum of approximately 1.7 m high. This was found to be the most comfortable height range for drilling, and coincides with the area pinhole borer usually inhabit. All mosses and many other epiphytes were removed from this band by hand or a scrubbing brush. Holes were drilled randomly within the marked area at a density of 17 holes per 100 cm^2 (Faulds, 1977). Holes are oriented slightly downward into the tree (to prevent inoculum dripping out) from a horizontal plane and approximately perpendicular to the outside of the tree (fig 4.3). Depth of holes was dependent on DBH of stem, and never exceeded 1/3 of this. After each hole was drilled, approximately 1-2 ml of inoculum (water or fungus, dependent on the treatment) was injected into the hole. The only difference between the 'holes, no fungus' treatment, and the 'holes, fungus' treatment is whether the tree is inoculated with sterile distilled water, or the suspension of fungus in sterile distilled water. The hole was then sealed with a thick layer of petroleum jelly.

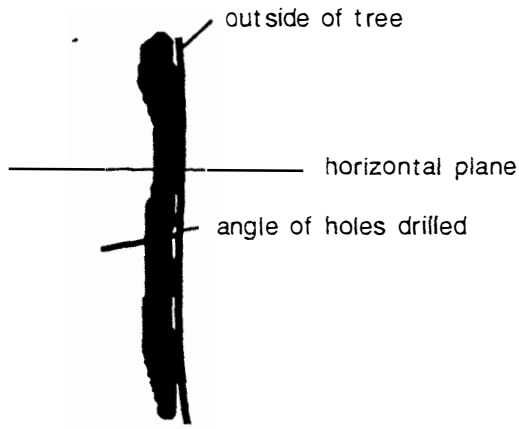


Fig 4.3 *Tangential cross section of tree showing angle of holes relative to horizontal.*

UNHEALTHY SUB-SITE

Banding

Ten trees were selected at random and banded as described above.

Fungicide inoculation

'Tecto', a systemic fungicide with 450 g/L suspension of thiobendazole (TBZ), was applied as high volume-low concentration injections. This is similar to the fungicide method for control of Dutch Elm Disease (Greig, 1990), and recommended quantities of doses and concentrations for injection into trees are published.

The fungicide was diluted on site to approximately 0.45 % active ingredient thiobendazole (TBZ) using water from a nearby stream, and applied at a rate of 10 L inoculum per 10 cm stem diameter. This is a slightly higher concentration than standard, but considered safe for the tree from the conclusions of Greig (1990); the dose is the same as that recommended. The higher concentration was used because the uptake of solution (in control trees, receiving only water) was observed to be considerably slower than described by Greig (1990).

The solution was applied internally using a pressure flow system (adapted from Lanier, 1987; and Greig, 1990) (fig 4.4). Three metres of 14 mm polythene irrigation tubing is connected to a 20 L reservoir and a 'T-piece', which connects to two 1 m lengths of 14 mm tubing. Nine 20 cm lengths of 4 mm irrigation tubing are inserted into each of these at 10 cm intervals. Holes are drilled at 10 cm intervals around the stem(s) of the tree, into which the smaller tubes are jammed, with any unused portion of the hoses clamped. The reservoir is raised to a height allowing the solution to be forced into the tree by gravity. This may take between 1 and over 10 hours, depending on the amount of water and speed of uptake. After application, holes were sealed with a thick layer of petroleum jelly.

Control trees were treated in the same manner with appropriate amounts of water.

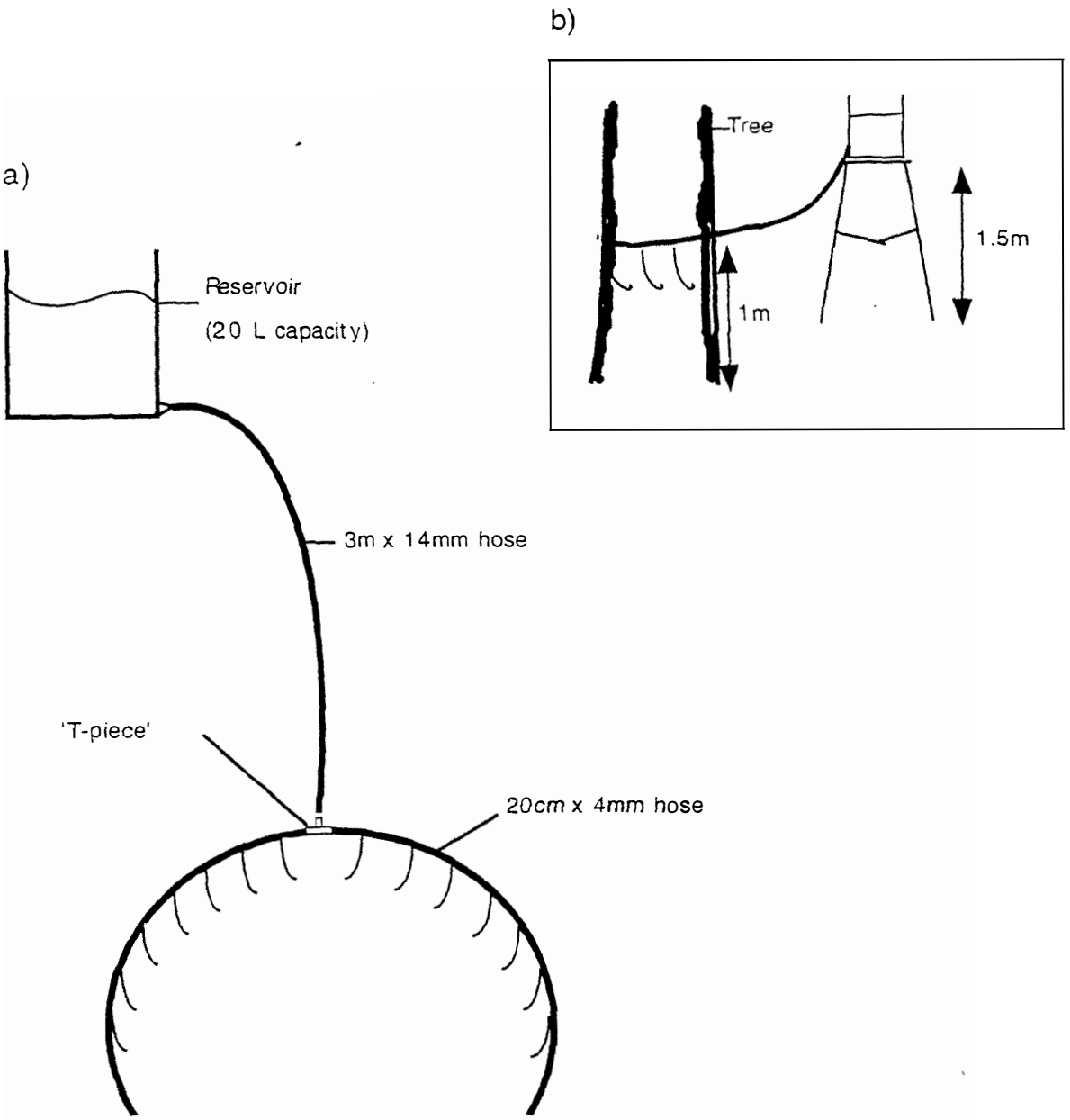


Figure 4.4. *Schematic diagram of fungicide treatment: a) layout of constructed apparatus; b) orientation of apparatus in field showing position of tree and related equipment.*

MONITORING

Leaf litter traps

Eighteen funnel-shaped leaf-litter traps, 60 cm diameter (0.283 m^2 surface area), were obtained from New Zealand Forest Research Institute, Rotorua. One of these was positioned under each of the crowns of three randomly selected healthy trees in each treatment (fig 4.1 and 2); under the crown of each of the other healthy trees two buckets, 25 cm diameter (0.1 m^2 total surface area), were placed, also to 'trap' leaf fall. In the unhealthy sub-site, two 35 cm buckets (0.19 m^2 total surface area) were positioned under each tree. These leaf litter traps were emptied once a month, from December 1994 to April 1996 for healthy trees (excluding May 1995 due to number of lost samples; duration = 17 months) and from June 1995 to April 1996 for unhealthy trees (duration = 11 months).

After collection, samples were frozen until sorted. Before sorting, samples were rinsed through a 2 mm mesh sieve to remove heavily decomposed litter and various detritus impeding easy sorting, such as volcanic ash, pollen, and small seeds and flowers. Collections were sorted into kamahi leaves, buds and twigs, where positive identification was possible, as well as possum and rodent pellets, which were easily distinguished if samples were collected in dry-ish weather, and 'other', where litter was either not kamahi, or could not be positively identified as kamahi (table 4.1). Kamahi leaves were further sorted into undamaged, insect or possum damage types. Insect and possum damage were distinguished by close examination of the leaf margin, and comparison with photographs of possum and insect damaged leaves (Forest Research Institute, pers.com., Meads, 1976). Undamaged leaves were those with less than approximately 5 % of any sort of damage. Damaged leaves were divided into some damage (approximately less than 40 %) and lots of damage (approximately more than 40 %), by rapid visual assessment.

Table 4.1 *Categories of leaf litter collected in litter traps. See text for details.*

Kamahi					
	Leaves			buds	twigs
	Undamaged	Insect some/lots	Possum some/lots		
Other					
	Possum pellets	Rodent pellets	Unidentified		

After sorting, kamahi leaves in each category were counted. All samples were then dried at 98 °C for 3 days and weighed.

Observational data

Scores for observations of details of each tree were recorded each month, weather permitting (table 4.2). In poor weather observations of canopy could not be made.

Productive effort was estimated under the categories 'flush', or new growth typically lighter green in colour, 'flower' and 'bud', relating to presence of racemes, 'seed pods', either from the current or previous seasons, and 'epicormic shoots' growing from the base of the tree. These were scored on a scale of 0, meaning none, through to 4, indicating *a* heavy "crop".

All observational litter fall estimates, damage scores and levels of dead terminal shoots are on a non-linear scale of 0 to 5: 0 indicates no loss or damage to that category, 1 is less than 10 % affected, 2 is 10-25 %, 3 equals 26-75 %, 4 indicates more than 75 % affected, and 5 means loss or damage to all parts scored.

Litter fall was estimated for foliage, flowers and seed pods.

Five categories of damage were assessed: trunk scraping by ungulates, foliage browsing by possums and by insects, foliage wilt, and twig breakage.

The proportion of dead terminal shoots was estimated on the same scale.

Table 4.2 *Observational variables scored. Production, litter fall, damage and dead terminal shoots are estimated (see text for details).*

Production	Litter fall	Damage	Health
Flush	Foliage	Trunk scraping	Dead terminal shoots
Flower	Bud	Possum browse	
Bud	Seed pods	Insect browse	
Seed pods		Foliage wilt	
Epicormic shoots			

ANALYSIS

All counts and weights are per m^2. Data were analysed in seventeen sets, in four groups.

All treatments

1) containing both counts and weights in each of the litter categories per day per m^2;

2) containing the proportions from number of kamahi leaves in each damage class;

3) containing the leaf counts in each damage class per day per m^2;

4) containing the proportions of total biomass in each litter category;

5) containing the proportion of total kamahi biomass in each category of kamahi litter;
6) containing litter biomass in each category per day per m^2;

Healthy site

7) containing the proportions of number of kamahi leaves in each damage class for healthy trees;
8) containing leaf counts in each damage class per day per m^2 for healthy trees;
9) containing the proportions of total biomass in each litter category for healthy trees;
10) containing the proportion of total kamahi biomass in each category of kamahi litter for healthy trees;
11) containing litter biomass in each category per day per m^2 for healthy trees;

Unhealthy site

12) containing the proportion of number of kamahi leaves in each damage class for unhealthy trees;
13) containing leaf counts in each damage class per day per m^2 for unhealthy trees;
14) containing the proportion of total biomass in each litter category for unhealthy trees;
15) containing the proportion of total kamahi biomass in each category of kamahi litter for unhealthy trees;
16) containing litter biomass in each category per day per m^2 for unhealthy trees;

Observational data

17) containing scores from observation categories for all trees at each sample period.

Due to the large number of variables and data sets, data were summarised by extracting the principal components (using correlations) of each set. The first two principal components of each data set were used for analysis, rather than the individual variables from each. These two components were examined for the effect of treatments using analyses of variance (ANOVA) by JMP (SAS Institute, 1994).

Another data exploration technique is used to determine if samples from within each treatment are similar: cluster analysis with city-block distance measure and flexible sorting with beta= -0.25 were performed, and dendrograms were truncated at the level of 8 groupings. All multivariate analysis was performed by software provided by J.B.Wilson (*pers.com.*).

RESULTS

Litter samples generally contained a high proportion of leaves with no damage or some insect damage, with very small amounts of possum damaged leaves (table 5.2). Variation was low between treatments for all categories of litter, except that much more litter was collected under healthy treatments than unhealthy.

ALL TREATMENTS

Counts and weights per day per m^2

Principal components analysis of the data set containing weights of litter in all categories and numbers of leaves in each kamahi damage class, per day per m^2, including all trees, explains 39.6 % of variance on the longest axis, and a further 13.1 % on the second (table 5.1). The first component is most influenced by number of leaves with some insect damage (r=-0.87), number of undamaged leaves (r=-0.86) and weight of leaves with some insect damage (r=-0.86). The second component is most influenced by both weights and numbers of leaves with any possum damage (some damage weight r=-0.53, lots of damage weight r=-0.63; some damage counts r=-0.55, lots of damage counts r=-0.61). Analysis of variance on these components found a significant difference between the healthy and unhealthy sites (P<0.001) on both axes, and no other effects (table 5.3).

Table 5.1 *Percent of variance explained by first two components of principal component analyses of each data set.*

Data type	Data set	Variables	Component1	Component2
All treatments	all data (day per m^2)	15	39.6	13.1
	leaf counts (proportions)	5	31.7	25.2
	leaf counts (day per m^2)	5	59.9	20.8
	weight (proportion)	10	24.2	12.8
	kamahi weight (proportion)	5	34.4	24.1
	weight (day per m^2)	10	31.5	12.3
Healthy treatments	counts (proportions)	5	32.1	27.6
	counts (day per m^2)	5	59.9	20.8
	weight (proportion)	10	23.5	13.5
	kamahi weight (proportion)	5	36.3	26.5
	weight (day per m^2)	10	27.6	13.4
Unhealthy treatments	counts (proportions)	5	30.9	24.2
	counts (day per m^2)	5	42.2	21.1
	weight (proportion)	10	20.6	13.0
	kamahi weight (proportion)	5	32.9	23.2
	weight (day per m^2)	10	21.0	13.9
All treatments	Observational	13	41.9	13.4

Table 5.1 *Mean values per m^2 for counts and weights in each litter category under each treatment.*

		All	Health		Banding		Holes		Fungus		Fungicide	
			high	low	yes	no	yes	no	yes	no	yes	no
Undamaged	count	1.6	2.0	0.6	1.7	1.5	2.0	2.1	2.1	2.0	0.6	0.6
	weight	0.18	0.2	0.06	0.2	0.2	0.2	0.2	0.2	0.2	0.06	0.06
Some insect	count	1.9	2.6	0.5	2.0	1.8	2.6	2.6	2.6	2.5	0.5	0.5
	weight	0.2	0.3	0.05	0.2	0.2	0.3	0.3	0.3	0.3	0.05	0.06
Some possum	count	0.09	0.1	0.01	0.08	0.1	0.1	0.1	0.1	0.1	0.02	0.008
	weight	0.009	0.01	0.0009	0.007	0.01	0.01	0.01	0.01	0.01	0.001	0.0004
Lots insect	count	0.9	1.2	0.2	0.9	0.9	1.2	1.3	1.1	1.2	0.2	0.2
	weight	0.05	0.07	0.01	0.05	0.05	0.06	0.07	0.06	0.07	0.01	0.01
Lots possum	count	0.1	0.2	0.008	0.09	0.2	0.2	0.2	0.2	0.2	0.01	0.005
	weight	0.005	0.007	0.0002	0.003	0.006	0.006	0.007	0.006	0.006	0.0003	0.0001
Buds	weight	0.05	0.08	0.0091	0.06	0.05	0.07	0.09	0.07	0.07	0.009	0.01
Twigs	weight	0.08	0.1	0.06	0.09	0.08	0.1	0.09	0.09	0.1	0.06	0.06
Possum pellets	weight	0.004	0.004	0.002	0.001	0.006	0.006	0.0008	0.01	0.003	0.002	0.002
Rat pellets	weight	0.0002	0.0002	<0.0001	<0.0001	0.0003	0.0003	0.0001	0.0001	0.0004	0.0001	<0.0001
Unidentified	weight	0.5	0.5	0.4	0.4	0.5	0.6	0.5	0.5	0.6	0.4	0.3

Table 5.3 P *values from analyses of variance of principal components of the data sets containing all treatments.*

	Data set	Health	Banding	Holes	Fungus	Fungicide
Component 1	all (day per m^2)	<0.0001	0.3	0.6	1.0	0.5
	counts (proportions)	<0.0001	0.9	0.7	0.05	0.5
	counts (day per m^2)	<0.0001	0.2	0.5	0.8	0.6
	weight (proportion)	<0.0001	0.002	0.5	0.01	0.7
	kamahi weight (proportion)	<0.0001	0.6	0.8	0.2	0.1
	weight (day per m^2)	<0.0001	0.6	0.7	0.1	0.2
Component 2	all (day per m^2)	<0.0001	0.1	0.5	0.9	0.6
	counts (proportions)	<0.0001	0.1	0.02	0.3	0.6
	counts (day per m^2)	<0.0001	0.04	0.4	0.9	0.8
	weight (proportion)	0.006	0.1	0.2	0.7	0.5
	kamahi weight (proportion)	0.5	0.06	0.05	0.5	0.08
	weight (day per m^2)	<0.0001	0.4	0.6	0.7	0.6

Cluster analysis of these data does not distinguish well between treatments (fig 5.1), although most trees from the unhealthy site are grouped together (group C, and group (A,B),C). Samples are grouped better by the date of collection, with most winter samples (from July to November) in group (A,B),C.

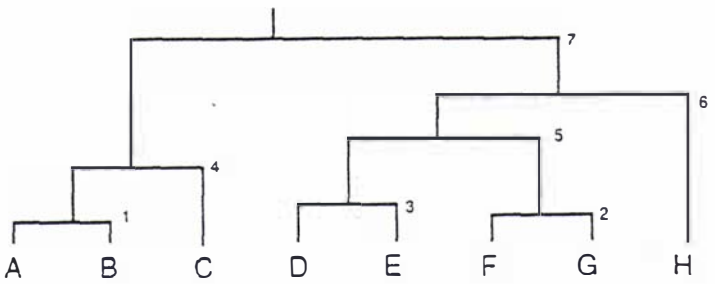
Proportions of counts

Principal component analysis of the proportions of number of kamahi leaves in each damage class for all trees explains 31.7 % of variance on the first component, and a further 25.2 % on the second (table 5.2). The first component is very strongly affected by the proportion of undamaged leaves in each sample (r=0.95), and also by the proportion with some insect damage (r=-0.73). Component 2 is related to the proportion of leaves with some insect damage (r=0.60), and the proportion with lots of insect damage (r=-0.63). ANOVAs of these components found a significant difference between healthy and unhealthy sites on both components (P<0.001, table 5.3).

Cluster analysis of these data does not distinguish well between treatments or sample period (Appendix I).

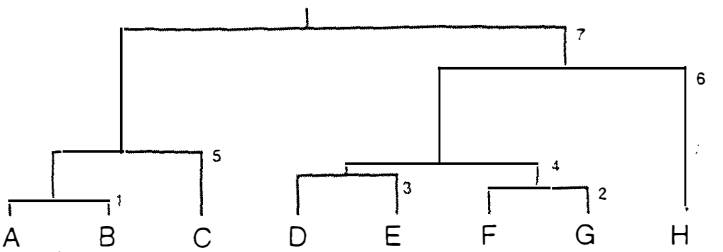
Counts per day per m^2

Principal component analysis of the number of leaves per day per m^2 in each damage class, including all trees, explains 59.9 % of variation on the first component, and 20.8 % on the second (table 5.1). The first component is most strongly influenced by the number of leaves with some insect damage (r=-0.88), and of undamaged leaves (r=-0.88), although all damage classes were highly correlated to this component.



Treatment:	A	33	9	15	15	19	5	1	1
	B	24	11	24	25	11	4	1	0
	C	46	5	14	19	15	1	0	0
	D	28	9	18	17	15	8	3	0
	E	29	3	17	14	28	6	3	0
	F	24	4	18	15	30	6	3	0
	G	13	5	80	0	2	0	0	0
	H	13	5	80	0	2	0	0	0
	I	18	5	76	0	0	0	0	0
	J	9	7	80	2	0	0	0	0
Date:	12/94	43	3	7	27	20	0	0	0
	1/95	17	3	3	33	17	27	0	0
	2/95	14	14	0	46	18	7	0	0
	3/95	20	7	10	47	10	7	0	0
	4/95	43	0	13	13	30	0	0	0
	6/95	34	0	50	6	8	0	0	0
	7/95	37	0	61	0	2	0	0	0
	8/95	24	0	76	2	0	0	0	0
	9/95	20	4	76	0	0	0	0	0
	10/95	4	56	26	0	12	0	2	0
	11/95	32	0	64	2	2	0	0	0
	12/95	32	0	22	30	8	6	0	0
	1/96	24	2	18	12	20	12	10	2
	2/96	14	8	32	10	30	4	2	0
	3/96	14	2	45	8	29	0	2	0
	4/96	31	0	43	0	25	2	0	0

Fig 5.1 *Dendrogram showing relationship between each tree at each sample period for both counts and weights of litter in each category per day per m², for all trees; numbers refer to degree of relatedness. Tables under dendrogram refer to percent from each treatment or sample period (date) in each of the terminal branches of the dendrogram. For explanation of treatments see text.*



		A	B	C	D	E	F	G	H
Treatment:	A	28	19	15	23	5	8	1	1
	B	14	20	28	24	5	9	1	0
	C	33	24	9	23	8	5	0	0
	D	24	18	22	16	4	10	6	0
	E	18	20	13	29	9	8	4	0
	F	25	9	19	21	14	8	5	0
	G	2	13	84	2	0	0	0	0
	H	11	4	85	0	0	0	0	0
	I	13	9	78	0	0	0	0	0
	J	2	9	87	2	0	0	0	0
Date:	12/94	45	3	3	28	14	7	0	0
	1/95	17	3	3	31	7	28	10	0
	2/95	10	17	3	63	0	7	0	0
	3/95	17	7	10	38	7	21	0	0
	4/95	37	7	10	30	13	3	0	0
	6/95	22	18	50	6	4	0	0	0
	7/95	20	28	50	2	0	0	0	0
	8/95	14	12	72	2	0	0	0	0
	9/95	8	12	80	0	0	0	0	0
	10/95	6	34	50	4	4	2	0	0
	11/95	14	22	60	4	0	0	0	0
	12/95	16	18	24	18	4	18	2	0
	1/96	18	10	20	20	6	8	16	2
	2/96	14	18	26	18	18	4	2	0
	3/96	18	6	44	18	10	2	2	0
	4/96	30	12	40	16	0	2	0	0

Fig 5.2 *Dendrogram showing relationship between each tree at each sample period, from rate of kamahi leaf counts per day per m² in each damage class for all trees, numbers refer to degree of relatedness. Tables under dendrogram refer to percent from each treatment or sample period (date) in each of the terminal branches of the dendrogram. For explanation of treatments see text.*

Component 2 is related to the number of leaves with any possum damage (some damage r=-0.58, lots of damage r=-0.65). ANOVAs of these components found a significant difference between the healthy and unhealthy sites on both components (P<0.001, table 5.3).

Cluster analysis of these data groups most trees from the unhealthy site (fig 5.2 group C), but distinguishes poorly between treatments within this site, or within the healthy site. Samples are grouped better by date of collection, with nearly all winter samples (July to November) in group (A,B),C, and most of those in group C; group D contains most samples from February 1995 (Appendix I).

Proportion of weight

Principal component analysis of the proportion of biomass in each category of litter for all trees explains 24.2 % of variance on the first component and another 12.8 % on the second (table 5.1). The first component is very closely related to the proportion of biomass in the unidentified category (r=-0.91) and to the proportion of leaves with some insect damage (r=0.77). The second component is influenced most by the proportion of kamahi twigs (r=-0.88). ANOVAs of these components found component 1 is significantly affected by initial health of trees (P<0.001) and banding (P=0.002); there is also a significant effect of initial tree health on component 2 (P=0.006, table 5.3).

Treatments are fairly evenly distributed between groupings from cluster analysis (Appendix I), except 60-70 % of trees from each unhealthy treatment are in group (G,H). Nor are samples grouped by date of collection, although most samples from December 1994 and January 1995 are in group ((A,B),C),D.

Proportion of kamahi component

Principal component analysis of the proportion of kamahi leaf biomass in each class of leaf damage for all trees explains 34.4 % of variation on the first component and a further 24.1 % on the second (table 5.1). The first component is very closely related to the proportion of undamaged leaf biomass in each sample (r=-0.95), and to the proportion of biomass of leaves with some insect damage (r=0.88), the second component is most affected by proportion of weight of leaves with lots of insect damage (r=-0.73). ANOVAs of these components show initial health class has a significant effect on component 1 (P<0.001, table 5.3).

Cluster analysis does not distinguish between treatments or dates in this data set.

Weight per day per m^2

Principal component analysis of the biomass of litter in each category per day per m^2 for all trees explains 31.5 % of variation on the first component and 12.3 % on the second (table 5.1). The first component is most influenced by weight of leaves with some insect damage (r=-0.85), undamaged leaves (r=-0.82) and leaves with lots of insect damage (r=-0.79); Component 2 is most influenced by weight of kamahi twigs (r=0.81). ANOVAs on these components found initial tree health had a significant effect on both (P<0.001) (table 5.3).

Cluster analysis of these data does not group samples by treatments, except most samples of trees in the unhealthy treatments are in group H (Appendix I). Samples are grouped well by date of collection, with 90 % of December 1994 and most of spring (February, March, April) 1995 samples in group (A,B), most of winter (June, July, August, September) and November 1995 in group H, and most of October 1995 in group (F,G).

HEALTHY TREATMENTS

Proportion of counts

Principal component analysis of the proportions of number of kamahi leaves in each damage class for healthy treatments explains 32.1 % of variance on the first component, and a further 27.6 % on the second (table 5.1). Component 1 is most affected by leaves with some insect damage (r=-0.83) and undamaged leaves (r=0.81). Component 2 is most affected by leaves with lots of insect damage (r=-0.67) and with lots of possum damage (r=-0.59) and undamaged leaves (r=0.55). ANOVAs of these components show drilling holes (P=0.02) and inoculating with *Sporothrix* fungus (P=0.04) have small significant effects on component 2 (table 5.4).

Cluster analysis of these data does not distinguish between treatments or dates.

Table 5.4 P *values from analyses of variance of principal components of the data sets containing healthy treatments.*

	Data set	Banding	Holes	Fungus
Component 1	counts proportions	0.8	0.8	0.07
	counts per day per m^2	0.4	0.5	0.8
	weight proportion	0.09	0.7	0.01
	kamahi weight proportion	0.8	0.7	0.3
	weight per day per m^2	0.4	0.6	0.1
Component 2	counts proportions	0.1	0.02	0.04
	counts per day per m^2	0.09	0.4	0.9
	weight proportion	0.9	0.3	0.6
	kamahi weight proportion	0.04	0.09	0.2
	weight per day per m^2	0.5	0.6	0.8

Counts per day per m^2

Principal component analysis of the number of leaves per day per m^2 in each damage class for healthy treatments explains 59.9 % of variation on the first component, and a further 20.8 % on the second (table 5.1). The first component is most influenced by leaves with some insect damage (r=-0.88), undamaged leaves (r=-0.88) and leaves with lots of insect damage (r=-0.81); component 2 is most influenced by leaves with lots of possum damage (r=-0.60) and with some insect damage (r=-0.58). ANOVAs of these components find no significant effects (table 5.4).

Cluster analysis of these data does not distinguish between treatments (Appendix I); groups A and C contain most samples for all treatments. Group C contains most winter samples (July, August, September, October, November), group (A,B) contains most April 1996 samples, and group (A,B),C contains most April and June 1995 samples.

Proportion of weight

Principal component analysis of the proportion of biomass in each category of litter for healthy trees explains 23.5 % of variation on component 1, and another 13.5 % on Component 2 (table 5.1). Component 1 is most influenced by proportion of unidentified biomass (r=-0.85), biomass of leaves with some insect damage (r=0.76) and undamaged leaves (r=0.71); component 2 is most related to proportion of kamahi twig biomass (r=-0.78). ANOVAs of these components found inoculation with fungus has a significant effect on component 1 (P=0.01) (table 5.4).

Cluster analysis of these data does not group samples by treatment or date, although most samples collected in October are in group F (Appendix I).

Proportion of kamahi component

Principal component analysis of the proportion of kamahi leaf biomass in each class of leaf damage for healthy treatments explains 36.3 % of variance on component 1, and a further 26.5 % on component 2 (table 5.1). Component 1 is highly influenced by the proportion of weight of leaves with some insect damage (r=-0.95) and undamaged leaves (r=0.92); component 2 is most influenced by the proportion of leaves with lots of insect damage (r=-0.72) and lots of possum damage (r=-0.67). ANOVAs of these components found no significant effects (table 5.4).

Cluster analysis of these data does not distinguish between treatments or date of collection.

Weight per day per m^2

Principal component analysis of the biomass of litter in each category per day per m^2 for healthy treatments explains 27.6 % of variance on component 1, and 13.4 % on component 2 (table 5.1). Component 1 is most influenced by the weight of leaves with some insect damage (r=-0.82), no damage (r=-0.82) and lots of insect damage (r=-0.74). Component 2 is most influenced by the weight of kamahi twigs (r=-0.79). ANOVAs of these components found no significant effects (table 5.4).

Cluster analysis of these data groups most samples in all treatments into groups A, B and C. Most of the samples from December, January, February, March, April and June are in group (A,B); samples from July, August, September and November are in group C, and samples from October are in group (G,H) (Appendix I).

UNHEALTHY TREATMENTS

Proportion of counts

Principal component analysis of the proportions of number of kamahi leaves in each damage class for unhealthy treatments explains 30.9 % of variance on component 1, and 24.17 % on component 2 (table 5.1). Component 1 is most influenced by proportion of number of undamaged leaves (r=-0.91) and leaves with some insect damage (r=0.82); component 2 is most influenced by the proportion of leaves with lots of insect damage (r=-0.95). ANOVAs of these components found no significant effects (table 5.5).

Table 5.5 P *values from analyses of variance of principal components of the data sets containing unhealthy treatments.*

	Data set	Banding	Fungicide
Component 1	counts proportions	0.5	0.2
	counts per day per m^2	0.2	0.7
	weight proportion	0.07	0.6
	kamahi weight proportion	0.8	0.7
	weight per day per m^2	0.7	0.8
Component 2	counts proportions	0.8	0.3
	counts per day per m^2	1.0	0.8
	weight proportion	0.9	0.6
	kamahi weight proportion	0.7	0.03
	weight per day per m^2	0.8	0.9

Cluster analysis does not distinguish between treatments, and only groups December 1995 and January 1996 by collection date (Appendix I).

Counts per day per m^2

Principal component analysis of the number of leaves per day per m^2 in each damage class for unhealthy trees, explains 42.2 % of variance on component 1 and a further 21.1 % on component 2 (table 5.1). Component 1 is influenced by number of leaves with some insect damage (r=-0.88), no damage (r=-0.84) and lots of insect damage (r=-0.76); component 2 is most influenced by number of leaves with lots of possum damage (r=-0.75) and some possum damage (r=0.70). ANOVAs of these components found no significant effects (table 5.5).

Cluster analysis of these data does not distinguish between treatments or date of collection.

Proportion of weight

Principal component analysis of the proportion of biomass in each category of litter for unhealthy treatments explains 20.6 % of variance on component 1 and another 13.0 % on component 2 (table 5.1). Component 1 is most affected by proportion of unidentified biomass (r=-0.94), component 2 is most influenced by proportion of kamahi twig biomass (r=-0.94). ANOVAs of these components found no significant effects (table 5.5).

Cluster analysis of these data does not distinguish between treatments or date of collection, with most samples from all treatments and dates in group (G,H), except October 1995, where 55 % of samples are in group E (Appendix I).

Proportion of kamahi weight

Principal component analysis of the proportion of kamahi leaf biomass in each class of leaf damage for unhealthy trees explains 32.9 % of variance on component1 and a further 23.2 % on component 2 (table 5.1). Component 1 is influenced by proportion of biomass of undamaged leaves (r=-0.92) and leaves with some insect damage (r=0.86); component 2 is most influenced by proportion of biomass of leaves with lots of insect damage (r=0.93). ANOVAs of these components found no significant effects.

Cluster analysis of these data does not distinguish between treatments (Appendix I). Group B contains 70 % of samples from January 1996, and group (C,D) 70 % from December 1995.

Weight per day per m^2

Principal component analysis of the biomass of litter in each category per day per m^2 for unhealthy treatments explains 21.0 % of variance on component 1, and a further 13.9 % on component 2 (table 5.1). Component 1 is most influenced by weight of leaves with some insect damage (r=-0.85) and with no damage (r=-0.78), component 2 is influenced by weight of buds (r=-0.66), twigs (r=-0.64) and leaves with some possum damage (r=-0.59). ANOVAs of these components found no significant effects.

Cluster analysis of these data does not distinguish between treatments with most of all treatments in group (A,B) (Appendix I). Most samples collected between June and September 1995 are in group (A,B); group (F,G),H contains most samples from October 1995; group (A,B) contains most samples from November and December 1995, and April 1996.

OBSERVATIONAL DATA

Principal component analysis of observational data explains 41.9 % of variance on component 1, and a further 13.44 % on component 2 (table 5.1). Component 1 is most influenced by scores for loss of buds (r=-0.91), damage to trunk (r=-0.89) and buds (r=-0.83); component 2 is influenced by scores for dead terminal shoots (r=0.68), seed pods (r=-0.58) and twig breakage (r=0.55). ANOVAs of these components found health has a significant effect on component 1 and component 2 (P<0.001) (table 5.6).

Table 5.6 P *values from analyses of variance of principal components of the observational data.*

	Health	Banding	Holes	Fungus	Fungicide
Component 1	<0.0001	1.0	0.2	0.7	0.6
Component 2	<0.0001	0.1	0.8	0.4	0.6

Cluster analysis distinguishes poorly between treatments, with dead trees tending to be in groups (D,E) or H (Appendix I). Samples are grouped by date of scoring, with group (A,B),C containing samples from October 1994 to January 1995, C containing February 1995 samples, group (D,E) with May 1995, D containing June to November 1995, group (F,G),H containing December 1995 and February 1996, and (F,H) with March 1996.

DISCUSSION

Possums, and the *Platypus* spp./*Sporothrix* association have been highlighted as the most likely causes, or prime factors, in decline of kamahi. However, Veblen and Stewart (1982) caution against the prevailing tendency to associate any widespread tree death with introduced animals, and Hosking (1986) comments that insects and disease should be viewed as symptoms of ill-health, rather than causes. This experiment tests for effects of four manipulated factors on the health of trees that were initially either healthy or unhealthy. The duration of treatments on healthy trees was 17 months and on unhealthy trees 11 months. Data were manipulated and analysed in several different ways in order to extract as much information about the treatments as possible. It was anticipated that due to the short period of this study, in comparison to the life scale of a kamahi tree, effects may be small (Gurevitch & Collins, 1994).

Data collected from litter samples showed consistently high proportions of leaves were either undamaged or had some insect damage. Over all treatments, the weights and counts for leaves damaged by insects were much higher than for those damaged by possums. Even in unbanded trees, signs of possum damage in litter were rare; however, there were uncommon but regular (52 incidents of a possible 700) occurrences of possum faeces (pellets) in samples; these pellets occurred randomly irrespective of tree banding, indicating that possums were present in all trees, and banding may not have been effective. Rat pellets also appeared occaisionally in samples (17 times), and this was much more likely to occur if the tree was unbanded.

Data in each set were summarised using principal components to lessen the number of analyses needed to determine effects of treatments. Analysis of variance was used to determine differences between each treatment and its control. The only consistent and strong effect found, was of initial tree health (which had been selected at the outset); from all data sets the only other effect significant (to $P<0.01$) was banding of trees on component 2 of the data with proportions of biomass in all litter categories of all trees.

From the results of these ANOVAs, it is clear that this experiment found no evidence of a change in initial health state under any treatments. Banding of trees was expected to cause an increase in the health of trees at the unhealthy site, but this did not occur. These trees maintained their low level of health despite their reduced availability to possums. It was expected that infecting trees with *Sporothrix* would reduce the vigour of healthy trees, but again this did not occur.

Cluster analyses of data sets did not group samples into treatments. Again the only treatment that grouped data was initial health of canopy, which was not a manipulated variable. Date of sample collection had more of an effect on grouping, with some seasonal effect being seen. This effect has not been tested further as seasonal variation in kamahi production is peripheral to this thesis.

These results contrast with most similar published research.

Most published studies linking effects of possums on tree health are descriptive or anecdotal. However, Meads (1976) observed possum browsing on very unhealthy northern rata (*Metrosideros robusta*) in the Orongorongo Valley. When some of these trees were banded to exclude possums, recovery was rapid and dramatic, with visible results after one summer, while other trees continued to decline. No recovery of unhealthy trees following banding was visible in the present experiment, nor was it indicated from the data collected. This strongly suggests that possums are not impacting on the health of kamahi in this area, and they are not currently maintaining the poor state of kamahi in the unhealthy site.

W.Faulds undertook a series of experiments to determine the cause of beech dieback (Faulds, 1973; Faulds, 1977). He introduced *Sporothrix* and other fungi to trees, to investigate their effect (Faulds, 1977) and found only 2 out of 15 trees inoculated with *Sporothrix* survived longer than 13 months, and both of these had died after 40 months. Only one other tree died, having been inoculated with *Endomycopsis platypodis* (a *Platypus* spp. ambrosia fungus). *Sporothrix* was also isolated from this tree after death. Payton (1983; 1988; 1989) initiated a series of experiments investigating the factors involved in rata-kamahi dieback in Westland. He introduced *Sporothrix* to kamahi at sites with a healthy or an unhealthy canopy (Payton, 1989). All except the largest kamahi had died 16.5 months after inoculation with fungus, while trees inoculated with sterile water remained healthy, regardless of site. Time from inoculation to death was found to relate to stem size. The results from their experiments indicate that, had the inoculation with fungus caused infection and disease, the duration of this experiment was long enough to see the effects. However, inoculation with fungus caused no deaths in the present study, nor was any loss of vigour indicated from the data collected. Payton (1989) doesn't comment on rainfall at time of his experiments, but Faulds' (Faulds, 1973; Faulds, 1977) experiments coincided with periods of low rainfall, and moisture stress is accepted to increase the toxicity of *Sporothrix*. The present study was in an area with relatively consistent and high rainfall, during a period of warm, wet weather (*Pers.obs.*).

The lack of fungicide effect was expected. Greig (1990) advises that best results are achieved when trees are separated from other trees of the same species and there are no diseased trees in the same area, when the disease is at an early stage and is limited to terminal branches; also, that it is too difficult to attempt fungicidal control if there is infection carried over from the previous year or if the tree is severely diseased. Fungicide has only been used for control of tree diseases in an urban environment, and is recommended to be used in conjunction with pruning of infected branches (Greig, 1990; Lanier, 1987). Lanier(1987) conducted experiments on treating Dutch Elm Disease in American elms, and found that even massive injection of fungicide in trees with root-grafted infections did not succeed, and although fungicide injection without pruning succeeded in 76 % of trees with infections from the current year, it was only successful in 33 % of trees with infections from the previous year. If the trees in the unhealthy site of the present experiment are infected with *Sporothrix* (and many, if not all, do have sign of borer), it is likely that the infections are not recent.

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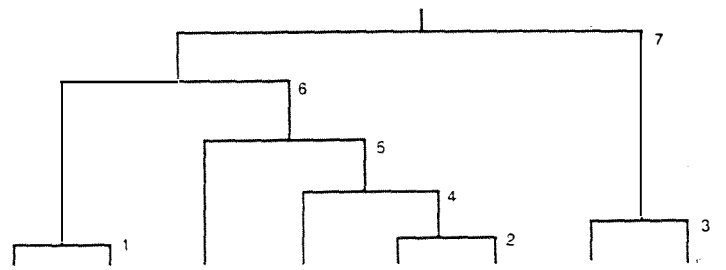
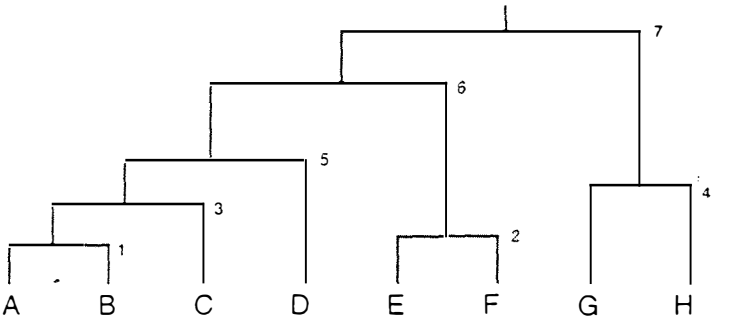
APPENDIX I

Dendrograms showing relationships between samples from each tree at each sample period. Dendrograms are labeled with their data set. Numbers refer to degree of relatedness. Tables under dendrogram refer to percent from each treatment or sample period (date) in each of the terminal branches of the dendrogram. For explanation of treatments and data sets, see text.



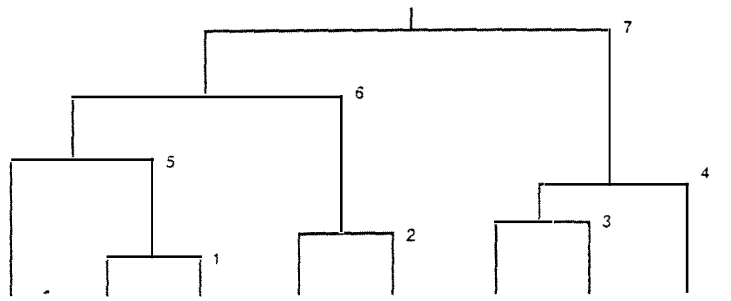
		A	B	C	D	E	F	G	H
Treatment:	A	18	1	31	29	3	0	18	1
	B	16	0	29	38	5	3	10	0
	C	21	0	28	26	6	3	16	0
	D	21	1	31	33	3	0	11	0
	E	14	1	44	16	5	1	18	1
	F	9	0	34	19	5	0	34	0
	G	6	13	18	13	13	7	22	9
	H	4	0	7	0	16	5	62	6
	I	6	0	15	7	16	5	45	6
	J	13	7	9	18	13	2	31	7
Date:	12/94	20	0	20	23	3	7	27	0
	1/95	7	0	40	37	3	0	13	0
	2/95	37	0	47	17	0	0	0	0
	3/95	7	0	47	43	3	0	0	0
	4/95	13	0	40	27	3	0	17	0
	6/95	14	2	22	26	10	6	16	4
	7/95	6	0	24	38	4	2	24	2
	8/95	14	8	8	26	12	2	26	4
	9/95	10	2	12	24	10	4	30	8
	10/95	32	4	8	14	12	4	24	2
	11/95	10	2	8	32	20	4	20	4
	12/95	16	0	36	22	10	0	16	0
	1/96	0	0	42	6	8	0	42	2
	2/96	14	4	30	10	0	0	42	0
	3/96	8	4	32	4	4	2	40	6
	4/96	14	2	30	8	8	4	32	2

a) Proportion of total number of kamahi leaves in each damage class (all treatments)



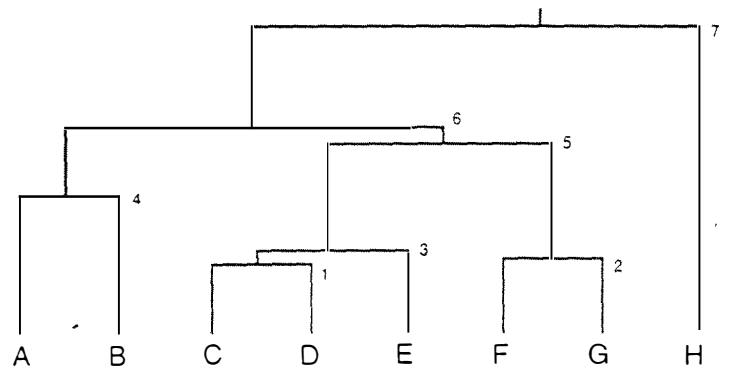
Treatment:	A	3	14	19	21	13	4	21	6
	B	0	13	30	11	5	4	18	20
	C	1	9	36	21	14	1	14	4
	D	1	15	24	23	8	4	18	9
	E	3	15	24	24	11	1	15	8
	F	0	30	28	15	6	4	14	4
	G	2	2	13	6	5	7	24	42
	H	4	2	2	4	4	5	36	44
	I	9	0	9	9	5	5	25	36
	J	13	2	13	4	5	5	20	38
Date:	12/94	3	27	20	33	0	0	17	0
	1/95	0	33	17	40	0	0	3	7
	2/95	0	13	43	10	0	0	20	13
	3/95	0	20	27	20	0	0	33	0
	4/95	0	23	33	17	10	0	13	3
	6/95	6	4	30	24	6	0	20	10
	7/95	2	12	16	20	2	0	24	24
	8/95	4	10	18	12	14	4	18	20
	9/95	8	2	22	12	2	0	14	40
	10/95	0	0	2	0	32	50	8	8
	11/95	6	2	14	24	6	0	16	32
	12/95	0	4	34	14	0	0	28	20
	1/96	2	20	26	14	4	0	22	12
	2/96	8	6	16	4	26	0	22	18
	3/96	2	12	22	0	6	0	30	28
	4/96	2	16	12	12	8	0	20	30

b) Proportion of total biomass in each litter category for all trees



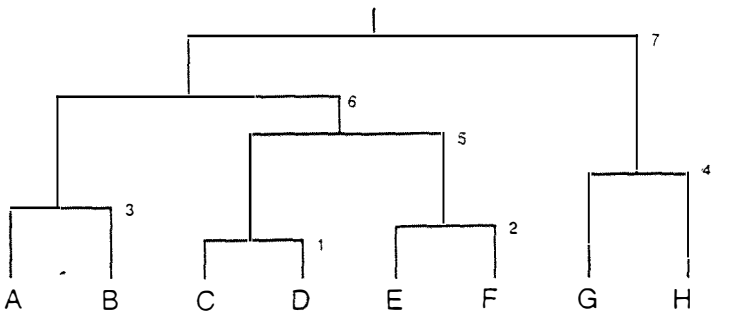
Treatment:	A	1	5	0	13	33	33	15	1
	B	3	4	0	10	31	24	18	11
	C	3	3	0	15	33	25	14	9
	D	1	6	0	9	31	21	25	6
	E	3	1	0	15	39	26	9	8
	F	0	1	0	16	49	22	9	2
	G	7	18	5	25	11	13	9	11
	H	24	2	4	26	24	11	0	9
	I	16	4	5	29	15	7	15	9
	J	29	7	2	11	15	24	9	4
Date:	12/94	3	7	0	13	30	33	7	7
	1/95	0	3	0	10	47	30	3	7
	2/95	0	7	0	3	43	17	27	3
	3/95	0	0	0	0	53	23	20	3
	4/95	0	3	0	20	40	17	13	7
	6/95	6	4	6	10	26	22	16	10
	7/95	8	0	2	22	22	20	20	6
	8/95	12	12	2	12	20	16	14	12
	9/95	16	0	4	16	8	28	18	10
	10/95	10	12	0	12	26	2	22	16
	11/95	8	8	0	10	10	38	10	16
	12/95	0	0	0	14	40	34	12	0
	1/96	6	0	0	32	36	24	2	0
	2/96	12	8	0	18	42	16	4	0
	3/96	10	4	2	26	34	14	6	4
	4/96	10	6	2	24	24	16	12	6

c) Proportion of total kamahi biomass in each litter category (all treatments)



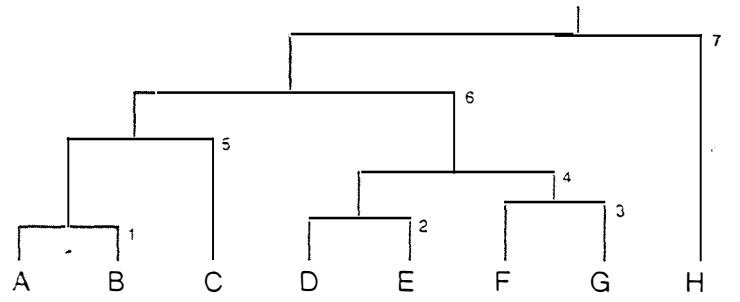
Treatment:	A	34	16	6	1	1	9	1	30
	B	28	26	13	0	4	3	3	25
	C	41	15	5	0	3	6	0	30
	D	30	16	6	0	1	6	1	38
	E	34	19	10	0	0	3	3	32
	F	41	11	4	0	1	4	4	35
	G	2	13	0	0	0	5	2	78
	H	2	27	0	0	0	5	0	65
	I	5	5	0	0	0	5	0	84
	J	2	15	0	0	0	7	2	75
Date:	12/94	76	14	7	0	0	0	0	3
	1/95	52	0	31	0	7	0	0	10
	2/95	53	27	0	0	3	0	0	17
	3/95	48	38	3	0	0	0	0	10
	4/95	53	13	3	0	0	3	0	27
	6/95	22	8	2	0	0	0	0	68
	7/95	8	6	0	0	0	0	0	86
	8/95	4	8	0	0	0	2	0	86
	9/95	0	12	0	0	0	2	0	86
	10/95	0	12	0	0	0	48	20	20
	11/95	10	8	0	0	0	2	0	80
	12/95	26	22	12	0	6	0	0	34
	1/96	32	18	18	2	4	0	0	26
	2/96	30	26	8	0	0	12	0	24
	3/96	20	38	2	0	0	2	2	36
	4/96	16	20	2	0	0	4	0	48

d) biomass per day per m_2 in each litter category (all treatments)



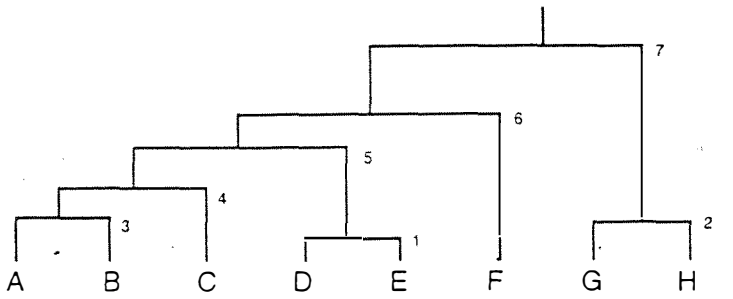
Treatment:	A	21	13	26	9	4	6	16	5
	B	29	10	18	9	4	1	19	11
	C	30	8	15	13	4	3	26	3
	D	26	19	21	8	3	1	18	5
	E	24	4	30	20	4	3	10	6
	F	21	3	20	33	3	1	19	1
Date:	12/94	20	13	13	20	3	0	23	7
	1/95	27	0	33	7	0	0	27	7
	2/95	47	10	23	7	0	0	10	3
	3/95	33	0	33	7	0	0	20	7
	4/95	20	10	27	17	3	0	17	7
	6/95	23	10	20	13	7	0	23	3
	7/95	17	13	27	7	0	3	23	10
	8/95	33	13	7	20	3	3	13	7
	9/95	20	13	13	13	0	10	20	10
	10/95	37	30	7	3	10	3	10	0
	11/95	17	3	10	10	3	3	37	17
	12/95	17	10	27	3	0	3	37	3
	1/96	13	0	47	27	0	0	13	0
	2/96	40	3	13	23	7	3	7	3
	3/96	20	7	30	27	7	7	3	0
	4/96	20	10	17	37	10	3	3	0

e) proportion of total number of kamahi leaves in each damage class (healthy treatments)



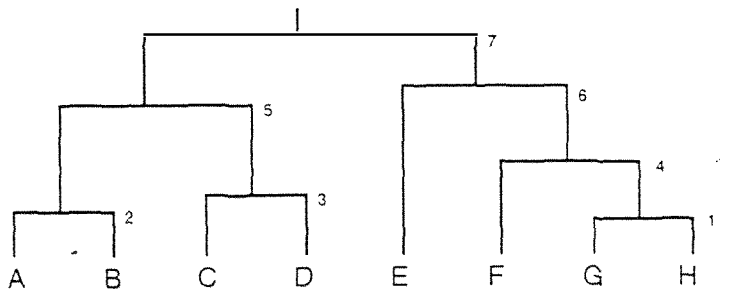
		A	B	C	D	E	F	G	H
Treatment:	A	28	13	34	10	5	8	1	1
	B	13	8	48	18	5	9	1	0
	C	28	10	35	15	8	5	0	0
	D	16	11	42	10	4	10	6	0
	E	32	3	33	13	9	8	4	0
	F	31	9	28	6	14	8	5	0
Date:	12/94	28	17	10	24	14	7	0	0
	1/95	17	10	7	21	7	28	10	0
	2/95	30	0	23	40	0	7	0	0
	3/95	7	14	17	34	7	21	0	0
	4/95	40	13	20	10	13	3	0	0
	6/95	23	17	50	3	7	0	0	0
	7/95	20	13	67	0	0	0	0	0
	8/95	17	7	73	3	0	0	0	0
	9/95	10	3	87	0	0	0	0	0
	10/95	13	0	77	0	7	3	0	0
	11/95	10	17	73	0	0	0	0	0
	12/95	10	13	13	23	7	30	3	0
	1/96	30	0	10	7	10	13	27	3
	2/96	30	0	13	17	30	7	3	0
	3/96	43	3	20	10	17	3	3	0
	4/96	63	13	20	0	0	3	0	0

f) kamahi leaf counts per day per m_2 in each damage class (healthy treatments)



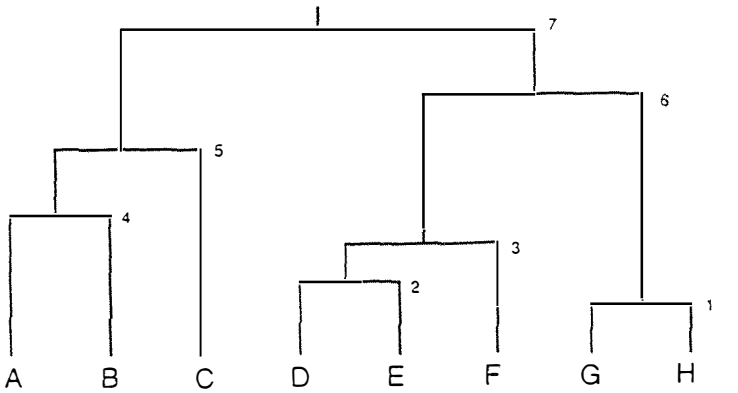
Treatment:	A	8	9	10	19	11	11	26	6
	B	9	21	6	8	9	6	18	24
	C	5	34	10	11	9	6	21	4
	D	6	10	14	13	18	9	21	10
	E	10	16	8	18	13	6	23	8
	F	9	15	9	19	16	8	21	4
Date:	12/94	7	13	7	23	30	0	20	0
	1/95	10	17	0	30	33	0	3	7
	2/95	3	37	0	10	7	0	30	13
	3/95	7	20	3	23	7	0	33	7
	4/95	13	30	3	7	17	3	20	7
	6/95	3	13	37	7	7	7	20	7
	7/95	7	10	13	20	23	0	20	7
	8/95	10	13	13	10	13	3	30	7
	9/95	3	7	33	7	13	3	7	27
	10/95	0	3	0	0	0	80	10	7
	11/95	0	27	0	20	13	3	23	13
	12/95	0	3	23	20	3	0	33	17
	1/96	23	20	7	23	7	0	17	3
	2/96	10	37	0	7	13	10	17	7
	3/96	17	23	0	0	3	7	33	17
	4/96	10	7	10	23	10	7	30	3

g) proportion of total biomass of sample in each category (healthy treatments)



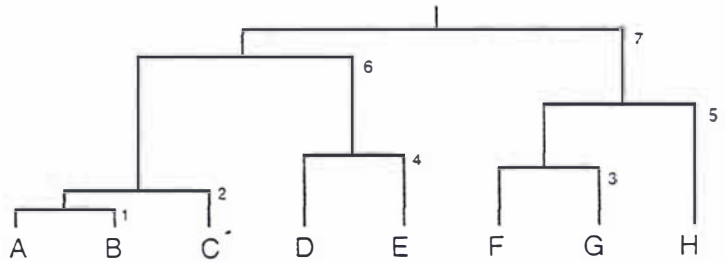
Treatment:	A	9	4	20	18	10	13	24	4
	B	9	3	14	19	8	16	15	18
	C	14	0	20	15	10	19	11	11
	D	9	3	8	25	15	18	13	11
	E	15	1	14	26	4	11	19	10
	F	15	0	13	38	3	13	14	6
Date:	12/94	10	0	20	17	10	27	7	10
	1/95	17	0	7	33	3	13	17	10
	2/95	0	0	13	33	20	20	10	3
	3/95	0	0	7	47	7	10	17	13
	4/95	17	0	10	33	3	13	10	13
	6/95	10	7	13	27	3	13	20	7
	7/95	10	0	10	17	3	27	20	13
	8/95	13	10	20	13	10	7	13	13
	9/95	13	7	10	3	7	17	23	20
	10/95	7	3	20	10	33	13	0	13
	11/95	7	0	7	0	7	27	23	30
	12/95	3	0	13	33	3	20	23	3
	1/96	20	0	0	47	3	10	20	0
	2/96	13	0	47	10	7	7	13	3
	3/96	27	0	23	23	0	7	17	3
	4/96	20	0	13	27	10	7	20	3

h) proportion of total kamahi biomass in each litter category (healthy treatments)



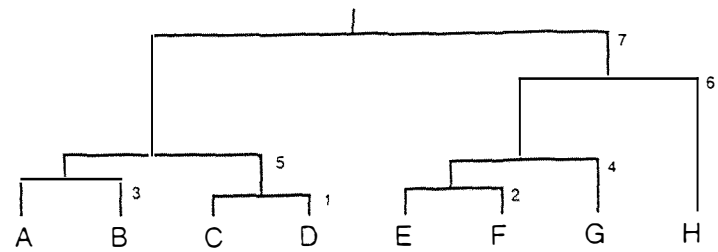
Treatment:	A	31	30	21	4	1	3	9	1
	B	24	39	16	13	0	4	3	3
	C	39	25	24	5	0	3	5	0
	D	23	34	29	6	0	1	6	1
	E	36	30	20	6	0	1	3	4
	F	48	23	20	4	0	0	3	4
Date:	12/94	67	20	10	3	0	0	0	0
	1/95	50	3	17	23	0	7	0	0
	2/95	47	47	3	0	0	3	0	0
	3/95	43	40	13	3	0	0	0	0
	4/95	63	33	3	0	0	0	0	0
	6/95	43	27	27	3	0	0	0	0
	7/95	13	17	70	0	0	0	0	0
	8/95	10	20	70	0	0	0	0	0
	9/95	3	33	57	0	0	3	3	0
	10/95	3	17	3	0	0	0	50	27
	11/95	17	23	57	0	0	0	3	0
	12/95	23	50	0	17	0	10	0	0
	1/96	33	27	0	30	3	3	0	3
	2/96	33	37	3	13	0	3	10	0
	3/96	40	53	0	3	0	0	0	3
	4/96	43	33	13	3	0	0	7	0

i) biomass per day per m_2 in each litter category (healthy treatments)



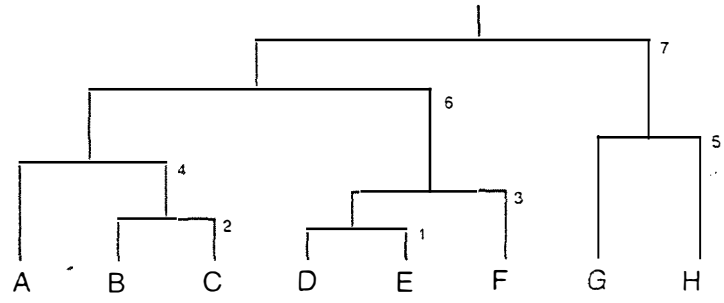
Treatment:	G	9	5	9	27	20	9	15	5
	H	9	31	0	25	25	4	0	5
	I	5	25	0	31	20	5	7	5
	J	7	15	9	38	16	2	9	4
Date:	6/95	10	5	5	30	25	15	10	0
	7/95	10	20	0	30	25	5	10	0
	8/95	10	20	10	25	5	5	10	15
	9/95	15	15	5	15	25	10	15	0
	10/95	5	15	10	15	20	5	5	25
	11/95	10	15	0	20	35	5	10	5
	12/95	0	10	0	65	20	0	5	0
	1/96	5	25	0	50	20	0	0	0
	2/96	0	35	10	40	10	0	5	0
	3/96	15	25	5	30	15	5	0	5
	4/96	5	25	5	15	25	5	15	5

j) proport ion of t ot al number of kamahi leaves in each damage class (unhealthy t reatment s)



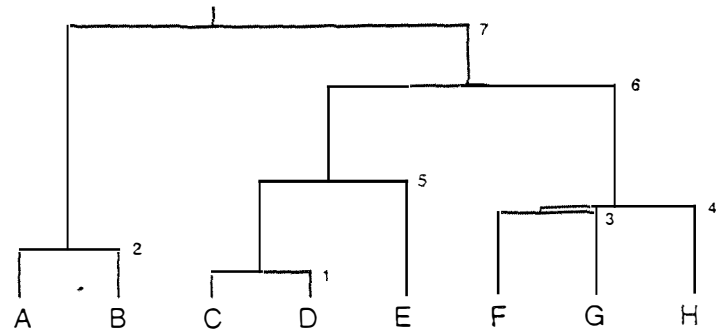
		A	B	C	D	E	F	G	H
Treatment:	G	35	22	22	5	2	13	0	2
	H	20	33	14	7	11	4	11	0
	I	24	13	24	13	7	9	9	2
	J	27	18	29	4	4	5	9	4
Date:	6/95	40	10	45	0	5	0	0	0
	7/95	40	20	20	0	0	5	15	0
	8/95	40	30	20	0	0	5	5	0
	9/95	40	40	15	5	0	0	0	0
	10/95	40	25	25	5	0	0	5	0
	11/95	45	10	30	0	5	0	10	0
	12/95	5	5	25	0	10	40	5	10
	1/96	0	5	10	15	30	15	15	10
	2/96	0	25	10	20	10	15	20	0
	3/96	10	45	25	10	0	5	5	0
	4/96	30	20	20	25	5	0	0	0

k) kamahi leaves in each damage class per day per m_2 (unhealthy treatments)



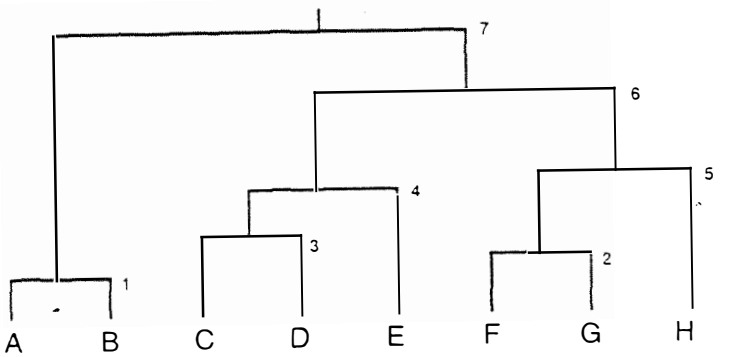
		A	B	C	D	E	F	G	H
Treatment:	G	0	16	2	6	5	2	44	25
	H	4	7	0	0	5	0	40	44
	I	7	13	4	4	5	0	35	33
	J	11	15	4	6	4	0	40	22
Date:	6/95	15	25	20	0	0	0	15	25
	7/95	5	5	0	0	0	0	50	40
	8/95	5	20	5	15	0	0	40	15
	9/95	10	0	0	0	0	0	65	25
	10/95	0	0	0	5	55	5	5	30
	11/95	5	5	0	0	0	0	60	30
	12/95	0	40	0	0	0	0	20	40
	1/96	5	30	0	0	0	0	25	40
	2/96	15	0	0	20	0	0	35	30
	3/96	0	10	0	0	0	0	50	40
	4/96	0	5	0	0	0	0	70	25

I) proportion of total biomass in each litter category (unhealthy treatments)



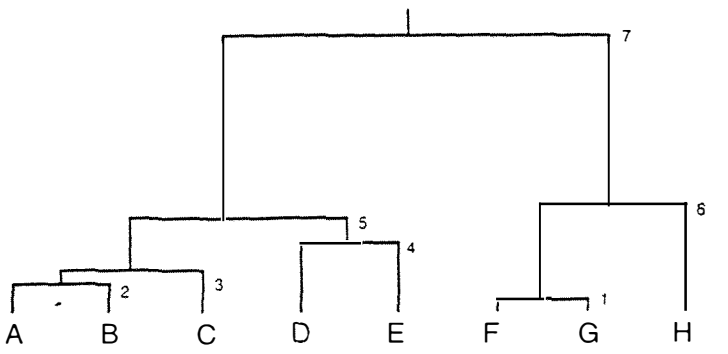
Treatment:	G	5	29	16	9	13	7	5	15
	H	9	40	9	25	9	2	4	2
	I	7	38	13	16	11	7	5	2
	J	13	27	29	16	4	7	2	2
Date:	6/95	15	10	25	10	20	5	15	0
	7/95	15	45	15	15	0	5	5	0
	8/95	15	15	15	10	20	10	5	10
	9/95	15	30	20	5	5	15	10	0
	10/95	5	30	10	15	25	5	0	10
	11/95	5	25	20	20	15	10	0	5
	12/95	0	30	30	40	0	0	0	0
	1/96	0	70	15	15	0	0	0	0
	2/96	0	50	10	25	0	0	0	15
	3/96	15	25	15	20	5	5	5	10
	4/96	10	40	10	10	10	10	5	5

m) proportion of total kamahi biomass in each litter category (unhealthy treatments)



		A	B	C	D	E	F	G	H
Treatment:	G	55	16	7	9	5	5	0	2
	H	31	22	25	15	2	5	0	0
	I	36	33	5	16	0	7	2	0
	J	38	31	9	7	5	5	2	2
Date:	6/95	80	20	0	0	0	0	0	0
	7/95	50	35	10	5	0	0	0	0
	8/95	65	15	5	5	0	10	0	0
	9/95	65	15	5	10	5	0	0	0
	10/95	25	5	5	5	0	40	10	10
	11/95	35	40	15	10	0	0	0	0
	12/95	5	50	5	35	5	0	0	0
	1/96	5	35	20	30	10	0	0	0
	2/96	20	20	30	10	5	15	0	0
	3/96	40	25	25	5	5	0	0	0
	4/96	50	20	10	15	5	0	0	0

n) biomass per day per m_2 in each litter category (unhealthy treatments)



Treatment:	A	28	8	22	17	0	7	18	0
	B	20	7	25	23	0	12	13	0
	C	23	7	22	23	0	10	15	0
	D	17	12	22	25	0	10	15	0
	E	17	3	33	22	0	17	8	0
	F	15	13	25	22	0	10	15	0
	G	9	0	0	24	33	12	0	21
	H	7	0	0	37	20	10	0	27
	I	3	0	0	55	10	23	0	10
	J	10	0	0	43	7	17	0	23
Date:	10/94	37	23	40	0	0	0	0	0
	12/94	53	10	37	0	0	0	0	0
	1/95	10	53	37	0	0	0	0	0
	2/95	10	7	83	0	0	0	0	0
	4/95	40	2	16	30	12	0	0	0
	5/95	10	2	12	60	16	0	0	0
	6/95	6	0	21	71	3	0	0	0
	8/95	30	0	8	54	8	0	0	0
	11/95	12	0	10	64	6	4	0	4
	12/95	0	0	0	0	0	34	42	24
	2/96	0	0	0	0	0	34	44	22
	3/96	0	0	0	0	0	73	27	0

o) observational data (all treatments)

Chapter 4: Regeneration under a healthy or thinning kamahi canopy, and the effect of excluding large animals

ABSTRACT

The effect of kamahi canopy health, and of larger vertebrate herbivores on regeneration was investigated in a patch of kamahi in Erua State Forest. Plots that were under a healthy or an unhealthy canopy, and that were enclosed in chicken-mesh to exclude larger herbivores or open, were compared. All seedlings taller than 8 cm were recorded and identified at three sample periods, at approximately two year intervals. Data were analysed using principal components on both density of seedlings per plot, and proportion of seedlings in each plot. Analyses of variance were conducted on number of species and number of seedlings per plot, and on number of species and seedlings lost and gained between each sample period.

Plots tended to increase in number of seedlings present. Canopy health had an effect on composition of the communities of seedlings present, with composition more strictly controlled under an unhealthy canopy, and more variable under a thinning canopy; canopy health did not affect density of seedlings. Excluding browsers caused an increase in the density of seedlings per plot, but did not affect community composition. These results suggest that a healthy canopy exerts constraints on the species that will successfully regenerate beneath it, while an unhealthy canopy does not. Likely factors are shade tolerance and the exclusion of wind-borne seeds from further afield by an intact canopy. It is also clear that introduced herbivores are limiting the level of regeneration occurring at this site.

INTRODUCTION

REGENERATION

Little attention has been paid until recently to the role of stand dynamics and regeneration in relation to canopy dieback. Yet, the implications of any dieback may be very different if replacement is good, rather than if the canopy species are not regenerating. Mueller-Dombois (1988) lists three types of stand-level dieback suggested from studies of New Zealand beech (*Nothofagus* spp.) forests by Hosking (1986; Hosking & Hutcheson, 1986). Replacement dieback refers to decline with adequate regeneration; displacement dieback to where there is a re-establishment problem for that species, but dieback is restricted to older trees; and stand-reduction to where both old and young trees are declining.

An increasing number of studies are suggesting that some canopy tree species require a thinning canopy for regeneration (for example Beveridge, 1973; Arentz, 1983; Jacobi, Gerrish & Mueller-Dombois, 1983: in NZ podocarps, Papua New Guinea Nothofagus spp. and Hawai'ian Metrosideros respectively). Bartlett (1984) comments that forests should be looked at as a mosaic of stands resulting from different micro-climate conditions relating to the 'gap' or patch they occupy. This mosaic may relate to the three types of stand level dieback referred to above. Peet (1987) (also Mueller-Dombois, 1987) sees the final stage of forest development as a mosaic of patches of various sizes and ages which contains all stages of succession. These studies predict that the species regenerating under unhealthy canopies will be different from those under healthy canopies, and often that regeneration will be much less under a healthy canopy.

Kamahi is the dominant canopy species in much of the forest in and around Tongariro National Park, and appears to be prone to stand-level dieback. Beveridge (1973) felt this dieback of kamahi is important for regeneration in New Zealand podocarp/hardwood forests, and so natural, allowing release of seedlings, particularly of podocarp species.

ANIMALS

Regeneration in New Zealand forests is also affected by introduced animals, particularly ungulate browsers, such as deer (*Cervus* spp.) and goats (*Capra hircus*), and possums (*Trichosurus vulpecula* Kerr). The effect of rodents is largely unknown.

Effects of introduced animals in New Zealand forests have initiated a huge amount of research and debate. As early as 1959, Holloway (1959) warned that New

Zealand's forests contain many species that appear susceptible to browsing by introduced mammals, particularly ungulates, and that there will be few species able to tolerate long-term grazing and soil compaction. Veblen and Stewart (1982) comment that the impact of introduced browsing and grazing mammals on New Zealand vegetation is widely perceived as an ecological disaster. They also note that study of these impacts illustrates the difficulty in distinguishing animal-induced changes from other causes.

Kean (1959) described red deer (*C. elaphus*) as the most aggressive coloniser of dense New Zealand bush, and predicted a depauperation of forests they inhabit. He rated possums conjointly with deer as capable of inducing forest collapse. McKelvey (1959) lists cases of forest degradation and possible effects of deer and goats. Wardle (1973) found that deer had huge effects on some plant associations in his study area (Sth Westland), and predicted that continued use by deer would result in regeneration failure at some sites.

From these studies, it can be concluded that vertebrate browsers have the potential to be a major problem for the forests of Tongariro National Park.

AIMS

This study examines regeneration in an area of kamahi showing differing levels of canopy thinning. It aims to investigate the relationship between canopy health and the 'communities' of seedlings, as well as densities of seedlings. The null hypothesis is that regeneration will be the same under a closed or a thinning canopy. This project will also study the effect on regeneration of excluding large animals, with the null hypothesis that there will be no effect.

The experiment adopts a multi-factorial approach, to reduce the amount of replication necessary for statistical analysis. Eight plots will be split between healthy and thinning canopies, and will be enclosed or open, and sampled at three time periods, with two 'replicate plots' in each treatment. Strictly speaking there is no true replication, as all plots are within one patch of kamahi. This is a common and necessary aspect of many ecological field experiments (Gurevitch & Collins, 1994; Hurlbert, 1984).

STUDY SITE

The study site is approximately 15 kilometres west of Erua settlement (fig 3.1 previous chapter). The site is within an area of forest that was cut-over early this century. The main canopy species is kamahi, and other broadleaves, such as putaputaweta (*Carpodetus serratus*) and maire (*Nestegis* sp.) are common. The lower canopy is dominated by, for example, coprosmas, pate (*Schefflera digitata*) and tree ferns. Climbers such as bush-lawyer (*Rubus cissoides*), clematis and supplejack (*Ripogonum scandens*) are common. The healthy plots are coincident with the healthy sub-site of the experiment in chapter 3, and the unhealthy plots with the unhealthy sub-site. All plots are within 100 m of the edge of the bush, which is delimited by a road. Due to this proximity to a road, densities of larger mammals, such as possum, deer and goats, have been kept low by hunters and trappers (C.Speedy, *pers.com.*), but these animals are still present. Location of plots within these sites was haphazard, but based on the requirement that a 5 m x 5 m chicken mesh enclosure could be built, regardless of whether the plot was to be enclosed.

METHOD

ENCLOSURE PLOTS

During winter 1994, four 'enclosure plots' were constructed, approximately 4 m x 4 m (table 4.1) and 1 m high, surrounded and covered (*i.e.* roofed-over) by chicken mesh (*ca.* 2 mm aperture, figures 4.1 and 4.2). Two of these are under the healthy canopy, and the other two are in the unhealthy site.

The following summer a further two plots were marked out by pegs in each of the sites. These were left open, and are the un-enclosed or control plots (table 4.1).

Table 4.1 *Area of each plot. Standard area is area divided by area of the largest plot (plot 8 = 24.7 m²). All counts are multiplied by this value to give density per standard plot(at the third sample period (t2), markers for plots 2 and 3 had been removed, and the area sampled is slightly different).*

Plot	Health of canopy	Enclosed or open	Area (m^2)	Area t2 (m^2)	Standard area multiplier	Standard area multiplier t2
1	healthy	open	19.8	19.8	0.802	0.802
2	healthy	enclosed	20.6	20.3	0.831	0.823
3	healthy	enclosed	20.1	21.8	0.812	0.880
4	healthy	open	21.5	21.5	0.870	0.870
5	unhealthy	enclosed	14.3	14.3	0.580	0.580
6	unhealthy	enclosed	15.4	15.4	0.621	0.621
7	unhealthy	open	16.4	16.4	0.664	0.664
8	unhealthy	open	24.7	14.7	1	1

RECORDING

Plots were first scored in early March 1995. The species of all seedlings greater than 8 cm and less than 1 m in height within each plot was recorded, as well as height. This height limit was imposed for three reasons: to avoid the problems associated with finding every seedling in a plot, to avoid the problem of identifying very small seedlings (which may have few or no leaves present), and because deer and goat typically do not target plants smaller than approximately 10 cm (C. Speedy, pers.com.). All seedlings above this height were numbered and tagged.

The second recording was taken in early March, 1997. If a seedling was tagged, its number was recorded, as well as species, height and number of leaves (if applicable). Searches were made for all missing tags, and some fallen ones were recovered. All other seedlings within the height range also had their details recorded.

Fig 4.1 *An exclosure plot under the healthy canopy (photo G.Rapson).*

Fig 4.2 *An exclosure plot under the unhealthy canopy (photo G.Rapson)*

A final recording was taken in late November 1998. At this time, two of the enclosures had been removed, and the location of the plots had to be estimated by the presence of holes in the ground.

ANALYSIS

Plots were different areas so total counts per standard plot, which is the largest area of the 8 plots (table 4.1), were calculated. The square root transformation of counts of species and seedlings per standard plot was used for comparison between plots, to limit the influence of very high numbers of seedlings in a few plots.

Principal components were analysed by JMP (SAS Institute, 1994) for the proportion of seedlings of each species at each plot, and the number of each species at each plot.

Analyses of variance (ANOVA) were conducted using JMP (SAS Institute, 1994). ANOVAs were calculated for number of species and total seedling density per plot; with time, health of canopy and open or enclosed as the effects. ANOVAs were calculated for change in number of species and change in number of seedlings per plot, with canopy health and presence of enclosure as the effects. ANOVAs were calculated for proportions of seedlings of each species at each plot, and density of each species at each plot.

RESULTS

DATA EXPLORATION

Relocation of tags on live seedlings was approximately 69 % over the study (table 5.1). At the second recording (t1), one tag was recovered from a dead seedling in each of plots 1 and 2, and four from plot 3 (11.1 %, 3.2 % and 8.7 % of originally tagged seedlings respectively). At the third recording, another tag was recovered from a dead seedling at plot 1, three at plot 2, two at plot three, seven at plot 5, and one at each of plots 6 and 8.

Table 5.1 *Percentage of tagged seedlings recovered (dead or alive) at each plot and tagged seedlings lost or recovered dead. Tag recovery is the percent of tags present at the beginning of a sample period which were recorded at the end; tags lost or dead are those tags which are not recovered, or are recovered from a dead plant. Plot 6 had more tagged seedlings recovered at t2 than 1.*

Plot (open)	Recovery t0-t1	Recovery t1-t2	Recovery t0-t2	Lost or dead (t0-t1)	Lost or dead (t1-t2)	Lost or dead (t0-t2)
1	77.8	71.4	55.6	33.3	42.9	66.7
4	85.7	91.7	78.6	14.3	8.3	21.4
7	52.6	70	36.8	47.4	30	63.2
8	52.9	100	52.9	47.1	11.1	52.9
total	67.3	83.3	56.0	35.5	23.1	51.0
(enclosed)						
2	87.1	88.9	77.4	16.1	22.2	35.5
3	93.5	81.4	76.1	15.2	23.3	37.0
5	93.4	87.3	81.6	6.6	22.5	27.6
6	77.4	120.8	93.5	22.6	-16.7	9.7
total	87.9	94.6	82.2	15.1	12.8	27.4
Overall total	77.6	88.9	69.1	25.3	18.0	39.2

Plots varied in the number of species and seedlings present at the first sampling time (table 5.3), and density of seedlings of each species per standard plot (table 5.2). Number of species and number of seedlings increased at all plots between first and second samples; number of species increased at all plots except plot 2, and at all except plots 2, 3 and 7 number of seedlings increased between second and third samples. The slight decrease in species density observed in plot 2 was due to a change in plot size, not number of species.

Table 5.2 *Density of species per standard plot (24.7 m²). Plots are labelled by number and sample time (a=time 0, b=time 1, c=time 2). Plots 1-4 are under a healthy canopy, plots 5-8 under a thinning canopy; plots 1,4,7 and 8 are open, plots 2,3,5 and 6 enclosed.*

	Plots																					
Species	**1 a**	**1 b**	**1 c**	**2 a**	**2 b**	**2 c**	**3 a**	**3 b**	**3 c**	**4 a**	**4 b**	**4 c**	**5 a**	**5 b**	**5 c**	**6 a**	**6 b**	**6 c**	**7 a**	**7 b**	**7 c**	**8 a**	**8 b**	**8 c**
undetermined				0.8	0.8														0.7					
Alseuosmia pusilla			1.6	2.5	2.5	1.6	11.4	18.7	20.2	3.5	3.5	4.3	2.3	4.1	2.9	2.5	1.9	2.5	2.0	1.3	1.3	1	1	4
wineberry			0.8		0.8	1.6							0.6									1		1
lawa				1.7	3.3	3.3	8.1	12.1	15.0	0.9	0.9	2.6	0.6	0.6	1.2	0.6	0.6	1.2						
putaputaweta			0.8	4.2	7.5	5.8			0.9				1.7	1.7	3.5	0.6		0.6					5	2
clematis			1.6						0.9						4.6		0.6	3.7	1.3	0.7	0.7			1
Coprosma arborea												0.9												
C. australis													0.6	0.6	1.2	0.6	0.6	0.6						
C. foetidissima																			0.7	0.7	0.7			
C. grandifolia							1.6																	
C. lucida														1.2										
C. rhamnoides					1.7	0.8								1.2						0.7				
C. robusta					11.6	0.8		12.2			1.7				0.6									
C. tenuifolia	2.4	5.6		1.7	9.1	17.3		69.0	48.4	0.9	3.5	6.1	2.3	12.2	18.5		6.2	18.6	2.7	8.6	3.3	1	4	4
rimu													0.6	3.5	0.6									
Fuchsia						0.8																		
broadleaf	0.8	1.6	0.8		0.8	0.8		0.8					4.1	11.0	13.3	1.2	3.7	8.7			2.0	2	2	2
pigeonwood		0.8		0.8	0.8		0.8				0.9	0.9	1.2	5.8	8.1	2.5	1.9	2.5				1	1	
hinau							0.8	4.9	2.6				0.6	1.7	1.7		1.9	0.6					1	
maire	0.8	2.4	1.6	1.7	3.3	4.9	2.4	11.4	1.8	1.7	2.6	0.9	4.6	12.2	18.5	1.2	1.9	1.9			1.3	3	1	1
mahoe		0.8			1.7		5.7	3.2	1.8	1.7	2.6	3.5	2.3	6.4	9.3									1
Metrosideros perforata																				0.7				
toro							0.8	1.6	1.8				2.9	8.7	11.6	3.1	3.1	3.1					1	1
miro		0.8				1.6	3.2	21.1	15.0	0.9	0.9	0.9	7.0	16.8	17.4	2.5	3.1	3.7	2.0	2.0	2.0	1	1	1
five-finger						0.8							1.2	1.7	1.7									
Pseudowintera axillaris								2.4																
P. colorata			1.6				0.8	0.8	5.3			0.9	2.3	2.9	5.8	0.6	0.6	0.6	0.7			3	2	2
lancewood				1.7	0.8	2.5	0.8	1.6	1.8	0.9	3.5	3.5	8.1	19.7	52.7	2.5	3.7	5.0	2.7	1.3	2.0			2
Pseudopanax simplex					0.8									2.3										
rata	0.8	2.4	3.2		2.5	3.2			0.9	0.9	3.5	4.3			1.2			0.6			2.0			2
supplejack		4.0	6.4			0.8		0.8	0.9						0.6									
bushlawyer				0.8	0.8	0.8																		
pate	2.4	4.0	4.8	10.0	13.3	9.9	0.8	0.8	0.9	0.9	1.7	2.6			0.6									
kamahi						0.8			0.9		1.7	6.1	1.2	6.4	24.3	1.2	0.6	0.6		0.7	1.3	2	2	8

Table 5.3 *Change in species and seedling densities per standard site over the three sampling times. t0 is initial sampling, t1 is second sample time and t2 is third sample time. ∂1 is the* increase *in density at t1 since t0, ∂2 is the increase in density between t1 and t2, total increase is increase in density between t0 and t2.*

Site	Species/ site (t0)	Species/ site (t1)	Species/ site (t2)	Increase in species (∂1)	Increase in species (∂2)	Total increase
1	4.0	7.2	8.0	80 %	11.1 %	100 %
2	8.3	14.1	14.0	70 %	-1.1 %	68.2 %
3	9.7	12.2	14.1	25 %	15.6 %	44.5 %
4	7.8	10.4	11.3	33.3 %	8.3 %	44.4 %
5	10.4	11.6	12.8	11 %	10 %	22.2 %
6	7.5	8.7	9.9	16.7 %	14.3 %	33.3 %
7	5.3	6.0	6.6	12.5 %	11.1 %	25 %
8	10	12	15	20 %	25 %	50 %

	Seedlings/ site (t0)	Seedlings/ site (t1)	Seedlings/ site (t2)	Increase in seedlings (∂1)	Increase in seedlings (∂2)	Total increase
1	7.2	22.4	23.2	211 %	3.6 %	222 %
2	25.8	62.4	58.4	142 %	-6.4 %	127 %
3	37.4	161.6	118.8	333 %	-26.5 %	218 %
4	12.2	27.0	37.4	121 %	38.7 %	207 %
5	44.0	120.6	200.0	174 %	65.9 %	354 %
6	19.3	30.4	54.7	58 %	79.6 %	184 %
7	12.6	16.6	16.6	32 %	0 %	32 %
8	17	24	35	41 %	45.6 %	106 %

The principal components analysis of proportions of species at each plot at each sample time explains 15.7 % of variance on the first axis and a further 12.4 % on the second (fig 5.1), the third component explaining a further 11.3 %. Component 1 is most influenced by proportions of toro (*Myrsine salicina*) and *Coprosma australis*, and also by miro (*Prumnopitys ferruginea*) (all positively) and pigeonwood (in a negative direction) (table 5.4). Component 2 is most influenced by *Coprosma foetidissima* (in a negative direction) and maire (positively). Component 1 discriminates between healthy and unhealthy canopies, with all plots under healthy canopies to the left of the origin, and all under unhealthy canopies to the right (means: healthy = -1.64, thinning = 1.64) except plot 7 at the second sample and plot 8 at the third. Plots 5 and 6, under an unhealthy canopy and enclosed, remain close together and a distinct pair at all sampling times. The two open plots in the unhealthy site are quite distinct from each other at all times on the second component, but very close on the first. Neither component distinguishes much change in plot three over time. Plots 1, 2 from the healthy site are very close together, while plot 4 is somewhat separated.

The principal components analysis of densities of species per standard plot explains 29.6 % of variance on the first component, and a further 15.3 % on the second (fig 5.2), the third component explaining a further 13.8 %. Component 1 is

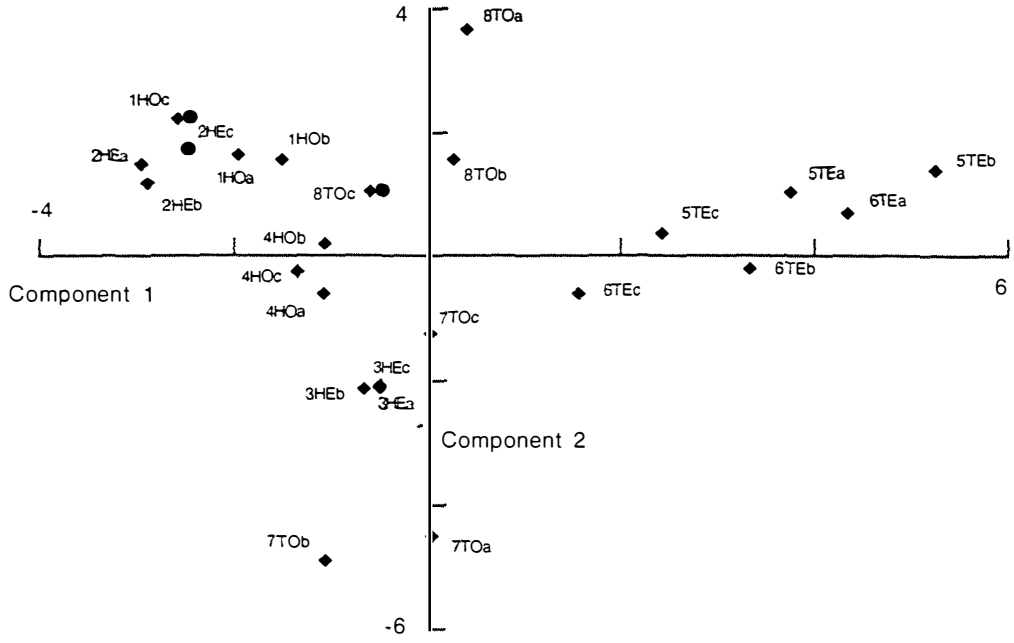


Fig 5.1 *First two components of principal component analysis of proportion of each species at each plot at each sample time. Numbers refer to plots, H=healthy T=thinning canopy, O=open E=enclosed, a=time 0, b=time 1, c=time 2.*

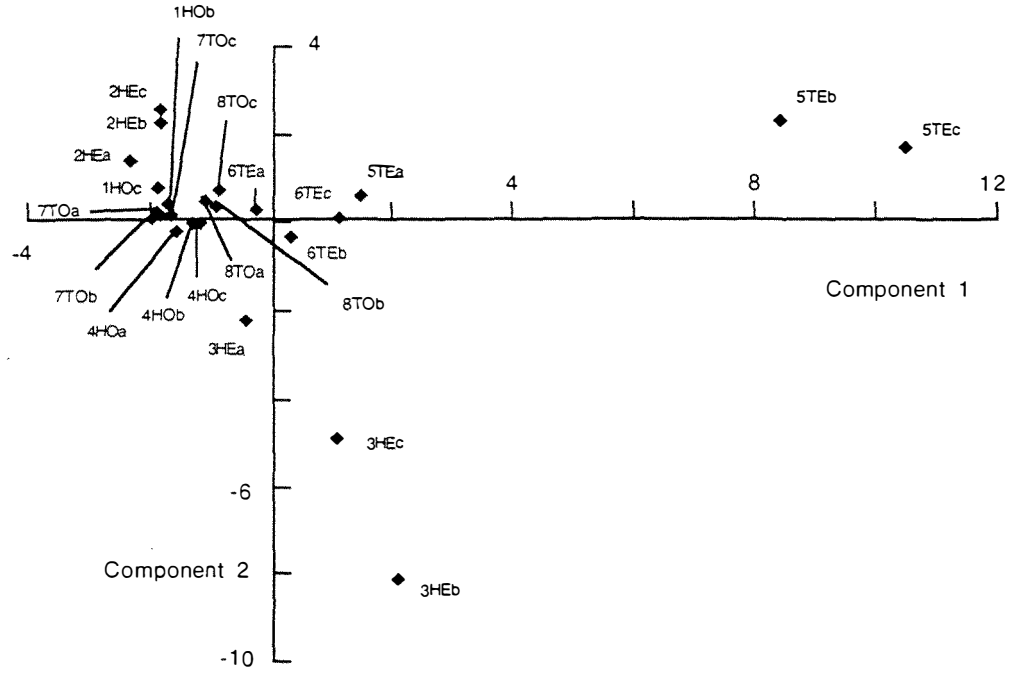


Fig 5.2 *First two components of principal component analysis of densities of each species at each plot at each sample time. Numbers refer to plots, H=healthy T=thinning canopy, O=open E=enclosed, a=time 0, b=time 1, c=time 2.*

Table 5.4 *Component loadings on first two principal components of species' proportions at each plot, and of density of species at each plot.*

Species	Proportions		Densities	
	Component 1	Component 2	Component 1	Component 2
undetermined	-0.19	-0.32	-0.25	0.24
Alseuosmia pusilla	-0.11	-0.48	0.21	**-0.87**
wineberry	-0.18	**0.54**	-0.21	0.35
tawa	-0.21	-0.28	0.12	**-0.80**
putaputaweta	-0.24	0.38	0.08	0.44
clematis	-0.00	-0.45	**0.51**	0.11
Coprosma arborea	-0.12	-0.03	-0.08	0.00
C. australis	0.74	0.08	**0.78**	0.23
C. foetidissima	-0.05	**-0.69**	-0.23	0.04
C. grandifolia	-0.05	-0.22	-0.03	-0.21
C. lucida	0.48	0.14	**0.56**	0.22
C. rhamnoides	-0.20	-0.28	0.12	0.38
C. robusta	-0.31	0.04	0.02	-0.39
C. tenuifolia	-0.22	**-0.51**	0.34	**-0.76**
rimu	**0.61**	0.18	**0.69**	0.26
Fuchsia	-0.23	0.18	-0.12	0.24
broadleaf	**0.53**	0.40	**0.88**	0.29
pigeonwood	**0.65**	0.33	**0.90**	0.31
hinau	0.39	-0.11	**0.54**	**-0.75**
maire	0.11	**0.63**	**0.89**	-0.06
mahoe	-0.02	-0.12	**0.81**	-0.08
Metrosideros perforata	-0.10	**-0.51**	-0.13	0.02
toro	**0.85**	0.11	**0.97**	0.14
miro	**0.69**	**-0.58**	**0.81**	**-0.51**
five-finger	0.45	0.23	**0.83**	0.35
Pseudowintera axillaris	-0.06	-0.22	0.14	**-0.76**
P. colorata	0.20	0.35	**0.71**	-0.12
lancewood	**0.63**	-0.27	**0.88**	0.23
Pseudopanax simplex	0.25	0.18	**0.50**	0.29
rata	**-0.50**	0.25	-0.25	0.25
supplejack	-0.30	0.28	-0.11	-0.01
bushlawyer	-0.43	0.24	-0.25	0.36
pate	**-0.66**	**0.55**	-0.35	0.37
kamahi	0.18	0.21	**0.76**	0.24

most influenced by numbers of toro and pigeonwood, and by maire, horoeka (*Pseudopanax crassifolium*) and broadleaf (*Griselinia littoralis)* (all positively) (table 5.4). Component 2 is most influenced by *Alseuosmia pusilla* and tawa (*Beilschmiedia tawa*), and also by *Coprosma tenuifolia*, *Pseudowintera axillaris*, and hinau (*Elaeocarpus dentatus*) (all negatively). Plots 3 and 5, the plots with the most seedlings, and the greatest increases in seedling density over time, are distinct from the remainder of plots at all sample periods. All other plots are relatively closely grouped. Component 1 is strongly related to the density of seedlings at the plot (r=0.83).

ANALYSIS

Analysis of variance of the density of species per standard plot found no significant effects from any of the treatments (table 5.5). Density of seedlings (number per

		Species	Seedlings
Effect	*DF*	*P*	*P*
Time	2	0.08	**0.04**
Health	1	0.74	0.88
Time*health	2	0.71	0.69
Enclosure	1	0.05	**0.001**
Time*enclosure	2	0.99	0.44
health*enclosure	1	0.28	0.94
time*health*enclosure	2	0.99	0.72
Model	11	0,39	0.06
Error	12		

Table 5.5 *Analysis of variance of density (sq.rt. transformed) of species and seedlings per standardised plot. Significant effects are in bold.*

standard plot) differed significantly with time (*P*=0.04) and enclosure (*P*=0.001), with fewest seedlings at time 0 and most at time 2 (means: t0=4.5 (seedlings per standard plot), t1=7.0, t2=7.6), and more in enclosed plots (E) than open (O) (means: O=4.5, E=8.3). ANOVAs of gains of species and gains of seedlings over time found time (*P*=0.04) and time*canopy (*P*=0.04) had significant effects (table 5.6), with greater gains at time 1 (species means: t1=34, t2=12; seedlings means: t1=139, t2=25), and the greatest and least gains under the healthy canopy (species means: t1=52, t2=9; seedlings means: t1=202, t2=2). ANOVAs on gains made over the entire sampling period found no significant effects. Enclosure affected loss of tagged seedlings (*P*=0.01, table 5.7), with more lost from open sites than enclosed (means: O=25, E=9); this effect was strengthened slightly when analysing the number of tags lost overall (means: O=44, E=18). When dead tagged seedlings are included there are no significant effects.

Table 5.6 *Analysis of variance for percentage of species and seedlings gained per standardised plot per sampling period, and over the whole study (overall); significant effects in bold.*

			Species	Seedlings
	Effect	*DF*	*P*	*P*
per period	Time	1	**0.04**	**0.006**
	Health	1	0.12	0.23
	Time*health	1	**0.04**	**0.02**
	Enclosure	1	0.58	0.23
	Time*enclosure	1	0.93	0.30
	health*enclosure	1	0.93	0.46
	time*health*enclosure	1	0.77	0.54
	Model	7	0.13	0,04
	Error	8		
overall	Health	1	0.13	0.66
	Enclosure	1	0.48	0,20
	health*enclosure	1	0.86	0.08
	Model	3	0.36	0,18
	Error	4		

Table 5.7 *Analysis of variance of percentage of tagged seedlings lost per plot, and tagged seedlings lost plus those recovered from dead plants; significant effects are in bold.*

			Lost	Lost or dead
	Effect	***DF***	***P***	***P***
per period	Time	1	0.13	0.36
	Health	1	0.68	0.94
	Time*health	1	0.07	0.16
	Enclosure	1	**0.04**	0.08
	Time*enclosure	1	0.51	0.52
	health*enclosure	1	0.18	0.23
	time*health*enclosure	1	0.76	0.76
	Model	7	0.13	0.33
	Error	8		
overall	Health	1	0.50	0.89
	Enclosure	1	**0.03**	0.13
	health*enclosure	1	0.10	0.27
	Model	3	0.06	0.30
	Error	4		

When the proportion of seedlings from each species per plot is analysed separately, most species appear to vary independently of sampling time and treatment (table 5.8). Health of canopy had an effect on more species than whether a plot is enclosed (9 were affected by canopy health, 4 by enclosure). Under a healthy canopy, a higher proportion of seedlings were tawa ($P<0.01$), *Coprosma robusta* ($P=0.02$), miro ($P=0.02$) and rata ($P<0.01$), while a higher proportion of seedlings under the thinning canopy were *C. australis* ($P<0.01$), broadleaf ($P<0.01$), toro ($P<0.01$), miro ($P=0.04$) and horoeka ($P=0.02$). Enclosed plots were likely to have a higher proportion of tawa ($P=0.02$), *C. australis* ($P<0.01$) and toro ($P<0.01$) seedlings, and a lower proportion of rata ($P<0.01$). There is a significant interaction between canopy health and enclosure for *C. australis* ($P<0.01$), toro ($P<0.01$) and rata ($P<0.01$). *C. robusta* was likely to make a higher proportion of a plot at the second sample period, and there was a significant interaction between health and time ($P=0.1$). The proportion of rata in plots increased over time ($P<0.01$).

When density of each species per plot is analysed, means differed significantly over time for clematis ($P<0.01$), *C. robusta* ($P<0.01$) and rata ($P=0.04$), with densities of clematis and rata increasing over time, and *C. robusta* having its highest density at the second sample time (table 5.9). Canopy health had significant effects on tawa ($P=0.02$), *C. robusta* ($P<0.01$), rata ($P<0.01$) and pate ($P=0.04$), all having higher densities in plots under a healthy canopy; clematis ($P<0.01$), *C. australis* ($P<0.01$), broadleaf ($P<0.01$), pigeonwood ($P=0.02$), and toro ($P=0.01$) had higher densities under a thinning canopy. There was a significant interaction between time

Table 5.8 *Significant results from ANOVAs of each species' proportion of seedlings at each plot. Health refers to canopy health, H=healthy canopy T=thinning canopy; enclosure refers to presence (E) or absence (O) of an enclosure.*

Species	Effect	*P*	Means
tawa	Health	0.006	H=0.06, T=0.008
	Enclosure	0.02	O=0.01, E=0.06
Coprosma australis	Health	0.004	H=0, T=0.007
	Enclosure	0.004	O=0, E=0.007
	Health*enclosure	0.004	H=0, O=0, TE=0.01
Coprosma robusta	Health	0.02	H=0.03, T=0.0002
	Time	0.02	t0=0, t1=0.04, t2=0.002
	Health*time	0.01	t0,Tt1=0, Ht1=0.08, Ht2=0.004, Tt2=0.0007
Griselinia littoralis	Health	0.009	H=0.02, T=0.08
toro	Health	0.0005	H=0.004, T=0.05
	Enclosure	0.001	O=0.006, E=0.05
	Health*enclosure	0.005	HO=0, HE=0.008, TO=0.01, TE=0.09
miro	Health	0.02	H=0.04, T=0.1
horoeka	Health	0.02	H=0.04, T=0.1
rata	Health	0.0001	H=0.07, T=0.02
	Enclosure	<0.0001	O=0.07, E=0.01
	Health*enclosure	0.002	HO=0.1, HE=0.02, TO=0.03, TE=0.003
	Time	0.003	t0=0.02, t1=0.04, t2=0.07
pate	Health	0.03	H=0.1, T=0.03

and health of canopy for density of clematis (P=0.02) with the lowest densities at the first and second sample times under a healthy canopy, and the greatest density at the third time under the thinning canopy; *C. robusta* had lower densities at the first sample, and highest at the second sample under a healthy canopy (P<0.01). Presence of an enclosure increased the observed densities of tawa (P=0.02), *C. australis* (P<0.01), *C. robusta* (P<0.01), *C. tenuifolia* (P=0.04), broadleaf (P<0.01), pigeonwood (P<0.01), hinau (P=0.03), maire (P=0.04), toro (P<0.01), miro (P=0.03) and rata (P=0.02). There are significant interactions between health and enclosure for clematis (P=0.04), *C. australis* (P<0.01), broadleaf (P=0.04), pigeonwood (P=0.02) and toro (P=0.03) with higher density in enclosed plots under unhealthy canopy; *C. robusta* has higher density in enclosed plots under a healthy canopy (P<0.01). There was a significant interaction between time and enclosure for clematis, where the highest densities were recorded at the third recording in enclosed plots (P<0.01); *C. robusta* recorded higher densities in enclosed plots at the second sample time (P<0.01). Both clematis (P<0.01) and *C. robusta* (P<0.01) also had significant interactions between time, health and enclosure, these effects are each driven by a pair of high densities, for clematis at the final recording, under the

thinning and in enclosed plots, and for *C. robusta* at the second sampling under a healthy canopy and also enclosed.

Table 5.9 *Significant results from ANOVAs of density of species at each plot. Health refers to canopy health, H=healthy T=thinning; E and O to presence or absence of enclosure, t0=initial sample, t1=second some, t2=third sample.*

Species	Effect	*P*	Means
tawa	Health	0.02	H=4, T=0.4
	Enclosure	0.02	O=0.4, E=4
clematis	Time	0.0002	t0=0.2, t1=0.2, t2=1.6
	Health	0.002	H=0.2, T=1.1
	Time*health	0.02	Ht0,Ht1=0, Ht2=0.6, Tt0=0.3, Tt1=0.3, Tt2=2.5
	Time*enclosure	0.01	Ot0=0.3, Ot1=0.2, Ot2=0.8, Et0=0, Et1=0.2, Et2=2.3
	Health*enclosure	0.04	HO=0.3, HE=0.1, TO=0.6, TE=1.5
	Time*health*enclosure	0.003	Ht0,Ht1,TEt0=0, Tt1=0.3, HEt2=0.4, TOt0=0.7, Ot2=0.8, TEt2=4.2
Coprosma australis	Health	<0.0001	H=0, T=0.3
	Enclosure	<0.0001	O=0, E=0.3
	Health*enclosure	<0.0001	HO,HE,TO=0, TE=0.7
C. robusta	Time	<0.0001	t0=0, t1=3, t2=0.2
	Health	<0.0001	H=2.2 T=0.04
	Enclosure	<0.0001	O=0.1, E=2.1
	Time*health	<0.0001	t0,Tt1=0, Ht1=6.4, Ht2=0.2, Tt2=0.1
	Time*enclosure	<0.0001	Ot2,t0=0, Ot1=0.4, Et1=6.0, Et2=0.4
	Health*enclosure	<0.0001	HO=0.3, HE=4.1, TO=0, TE=0.1
	Time*health*enclosure	<0.0001	t0,HOt2,TO,TEt1=0, HOt1=0.9, HEt1=12.0, HEt2=0.4, TEt2=0.3
C. tenuifolia	Enclosure	0.04	O=3.5, E=17.0
broadleaf	Health	0.0007	H=0.5, T=4.2
	Enclosure	0.005	O=0.9, E=3.7
	Health*enclosure	0.04	HO=0.5, HE=0.4, TO=1.3, TE=7.0
pigeonwood	Health	0.02	H=0.4, T=2.0
	Enclosure	0.02	O=0.4, T=2.0
	Health*enclosure	0.02	H=0.4, TO=O.3, TE=3.7
hinau	Enclosure	0.03	O=0.08, E=1.2
maire	Enclosure	0.04	O=1.4, E=5.5
toro	Health	0.01	H=0.4, T=2.9
	Enclosure	0.007	O=0.2, E=3.1
	Health*enclosure	0.03	HO=0, HE=0.7, TO=0.3, TE=5.4
miro	Enclosure	0.03	O=1.0, T=7.6
rata	Health	0.001	H=1.8, T=0.5
	Enclosure	0.02	O=1.6, E=0.7
	Time	0.001	t0=0.2, t1=1.0, t2=2.2
pate	Health	0.04	H=4.3, T=0.7

DISCUSSION

There is a paucity of research on the nature of regeneration under a healthy relatively closed kamahi canopy, compared to that under an unhealthy, more open, canopy. This study addresses that question, while at the same time, investigating the effect of excluding all animals wider than 1 cm. Introduced animals that are likely to be present in this area are rats and mice, goats and deer, and possums. All have been implicated as causing a reduction in regeneration in New Zealand forest (for example, Cunningham, 1979; Campbell, 1990; Brockie, 1992; Veblen & Stewart, 1982).

An artifact of this experiment was that the mesh roof of the enclosure plots trapped leaves and branches, to the extent that humus was forming in places (fig 6.1); the decrease in light under this was substantial, at least 30 %, but somewhat patchy, and will have affected the plants in these enclosures. Further compounding this, build up of litter was much less on plots under the thinning canopy. This may affect comparisons between open and enclosed plots, and also between enclosed plots under healthy and thinning canopies. Seed fall to areas inside enclosures under a healthy canopy may be significantly reduced by seeds trapped in litter, while an enclosed plot with less litter may provide perching for birds, increasing seed fall. Plants under reduced light may etiolate, and some seedlings in enclosures at the healthy site did appear to have unusually large leaves (*pers.obs.*). There may be an increase in mortality due to competition for light and space, or protection from smothering by falling litter may decrease mortality of seedlings (Brockie, 1992).

A final caution in interpreting these results is the removal of the enclosure from plots 2 and 3 sometime (less than 3 months) prior to the third sample.

RESULTS

Attrition of tagged seedlings was low, with recovery higher than expected from open enclosures (greater than 50 % over 3.5 years), and loss of tags or recovery from dead seedlings was low across treatments (less than 40 %). The trend to greater density of seedlings and species at all plots over time was also unexpected. Only plots 2 and 3 experienced a decrease in seedling densities, this occurring at the third sample period when both their enclosures had been recently removed, and plants may have been heavily affected by the removal process or by their new accessibility to herbivores. Under a thinning canopy, seedling densities may be expected to increase in response to increased light; however, this increase occurred under both healthy and unhealthy canopies. There has been a series of years with high reproductive effort by some species in the Park (1994/5 and 1995/6 pers.obs.), and perhaps seedlings

Fig 6.1 *Exclosure plot with litter build up on roof (top photo), and after litter is removed (lower photo) (photos G.Rapson).*

originating from that time have passed the 8 cm threshold for this study since the first measures.

Canopy health was not found to affect densities of species or seedlings present, but over time the densities of seedlings increased significantly, and enclosed plots had significantly more seedlings than open plots at all times (including the initial sampling period). The percentages of species and seedlings gained from each sample time to the next were significantly reduced over time, and time and health interacted with the healthy canopy recording huge gains from time 1(t=0) to time 2 (t=1), and very small gains from time 2 (t=1) to time 3 (t=2).

Loss of tagged seedlings may indicate mortality, particularly in enclosed sites, where there is less opportunity for a tag to be knocked off by wind, animals, etc.; however, the only significant effect was presence or absence of enclosure, and this would be expected for the aforementioned reasons. The lack of significance when tags from dead seedlings are included further suggests losses are not related to mortality.

The principal component analysis of proportions of species at each plot, shows all plots under a healthy canopy tend to contain similar proportions of species, or similar communities, with plot 3 somewhat separate. This is not unexpected as they are all situated near to each other. Plots from the unhealthy site are not as closely grouped by these components, despite all being in close proximity (in real space), *i.e.* they are more variable in composition. The two unhealthy open plots are very dissimilar from each other and all other plots. Plots 5 and 6, both enclosed and under the thinning canopy, were closely related at the first sample, very different by the second, and more closely at the third. A high, but expected, degree of auto-correlation is shown between each plot at each sample time: the community present at each sample will be influenced by that initially present. Results from this analysis suggest that a closed canopy may impose a much stronger influence on the community regenerating beneath it, than a thinning canopy. This may be for a number of reasons: the lower light levels in a healthy may preclude many species from germinating or competing successfully; local seed saturation may be greater under a healthy canopy causing high competition, or if there is lower seed saturation from local sources under a thinning canopy, seeds from more distant parents may be more successful.

The principal component analysis of density of each species per standardised plot does not distinguish between those plots under healthy and those under unhealthy canopies. The first component relates closely to density of all seedlings per plot, so plots 3 and 5, with very high densities of seedlings at time 1 and 2, are outliers. The

influence of total density of seedlings in this analysis is strong enough to override any more interesting trends that may be present.

The proportions of tawa, *Coprosma australis*, *C. robusta*, broadleaf, toro, miro, rata and pate seedlings in the 'communities' of plots were influenced by health of the canopy, but only the proportion of tawa, *C. australis*, toro and rata were affected by enclosure plots (of these toro is known to be unpalatable to deer). Because healthy canopies and unhealthy canopies were spatially separated and there was no true replication (*i.e.* all plots under a healthy canopy are in one area and all under a thinning canopy are in another), it is impossible to assess whether the effect of canopy health on the 'community' regenerating beneath is merely reflecting differences in the canopy 'community' or some other site related factor, or whether canopy health, for example through its effect on light levels at the forest floor, has direct implications for the regeneration success of different species. The higher proportions of tawa, *C. australis* and toro in enclosed plots, and rata in open plots, are more likely to reflect differences in species ecology, for example susceptibility to browsing or ability to compete in crowded conditions.

When density is considered, several species are affected by time, enclosure and canopy health. Results appear strongly influenced by the lack of some species from most sites, for example, *C. robusta* or clematis which are each present at high numbers in two enclosed plots, the former under a healthy canopy at time 1, the latter under a thinning canopy at time 2. This may indicate the influence of browsing on these species, which is most likely to be by ungulates (Brockie, 1992), or the impact of seed predation by rodents in open sites. Clematis, *C. robusta* and rata increased in density over time. Tawa, *C. robusta* , rata, and pate did better under the healthy canopy; clematis, *C. australis*, broadleaf, pigeonwood and toro all did better under the thinning canopy. Tawa, *C. australis*, *C. robusta* , *C. tenuifolia*, broadleaf, pigeonwood, hinau, maire, toro, miro and rata have higher densities in enclosures.

Although there is no difference in density of seedlings or number of species under healthy or unhealthy canopies, there is a difference in the communities found there, with the proportions of some species higher under the healthy canopy, and others under the thinning canopy.

There is clearly an effect on density of seedlings when all large animals are excluded. That excluding herbivores will benefit plants is intrinsically obvious. The main vertebrate herbivores currently in New Zealand forest are introduced, and are the usual suspects in any vegetation community change (Veblen & Stewart, 1982).

However, the indigenous fauna of New Zealand included several species of browsers (*i.e.* moa), and it has been suggested that introduced herbivores replace these, if somewhat crudely (McSaveny & Whitehouse, 1988). This study confirms the effect of non-insect browsers, even at low densities, on regeneration; in the absence of large herbivores, more seedlings will develop. It found no evidence of effects on community structure. In areas with higher densities of mammalian browsers this effect will be more dramatic. Allen *et al.* (1984) caution against the generalisation of results from single experiments, as exclosures only remove animals at one point in time, and the combination of factors impacting on communities at that time may not occur again, at that site or elsewhere.

COMPARISON WITH LITERATURE

Many of the data on the impact of introduced browsing animals on New Zealand forests is contained in unpublished New Zealand Forest Service reports (Allen *et al.*, 1984). Little has been formally recorded in refereed publications.

Brockie (1992) describes results from two longer term studies on the effects of excluding goats and deer, but not possums or hares, by fencing (to a height of *ca.* 2 m) a small area of vegetation. Neither of these studies contained any replication, so any inferences from them are suspect. The first plot suffered loss of most species due to erosion and probably browsing by hares and possums. The second showed an increase in kawakawa, *Coprosma grandifolia* and *Geniostoma rupestre* inside the plot compared to outside. Of these, only *C. grandifolia* is present at this site, and then only in low numbers.

Allen *et al.* (1984) report results from 17 ungulate exclosure plots in or near Urewera National Park within the range of deer (particularly red deer) and pigs (*Sus scrofa*), but probably not goats. Exclosures were fenced as above, varying in area from 60 to 100 m^2, and situated in a range of forest types, generally at less than 20° slope, and between 300-400 m asl. The authors found significantly more tree and shrub species between 15 cm and 1.4 m in height inside enclosures than outside, but no differences for seedlings less than 15 cm tall, and the same pattern when seedling density is compared between exclosure plots and controls. When data from the present study are divided into the above height classes, they generally fit this pattern, but density of seedlings less than 15 cm in height is also significantly higher in enclosed plots (table 6.1). With the addition of time as a factor, both number of species (species richness) and density of seedlings between 8 cm and 15 cm tall are increasingly significantly over time. Few of the species studied by Allen *et al.* (1984)

Table 6.1 *ANOVA of species richness and seedling density (both sq.rt. transformed) at two height classes, for comparison with Allen* et al. (1984). *Health refers to canopy health (H=healthy, T=thinning), Enclosure to absence (O) and presence (E) of enclosure, and Time to sample period 1 (t1) or 2 (t2).*

Effect		*P*	means
Health	Species richness <15cm	*n.s.*	H=2.3, T=2.4
	Seedling density <15cm	*n.s*	H=4.3, T=4.1
	Species richness >15cm	*n.s.*	H=2.7, T=2.7
	Seedling density >15 cm	*n.s.*	H=4.5, T=4.6
Enclosure	Species richness <15cm	*n.s.*	O=2.3, E=2.4
	Seedling density <15cm	**0.03**	**O=3.4, E=5.1**
	Species richness >15cm	**0.001**	**O=2.3, E=3.1**
	Seedling density >15cm	**<0.001**	**O=2.9, E=6.3**
Time	**Species richness <15cm**	**0.005**	**t0=1.6, t1=2.6, t2=2.8**
	Seedling density <15cm	**<0.003**	**t0=2.1, t1=5.1, t2=5.4**
	Species richness >15cm	*n.s.*	t0=2.5, t1=2.8, t2=2.9
	Seedling density >15 cm	*n.s.*	t0=3.7, t1=4.7, t2=5.3

showing significant changes in density due to exclusion of ungulates (all increased in density) are found in the present study.

From these results, it is clear that canopy health has a substantial effect on regeneration beneath it, with regeneration under a closed canopy much less variable than under an unhealthy canopy. Vertebrate browsing also has an effect on regeneration, with a higher density of seedlings allowed to develop where browsers are reduced. Canopy health affects community structure, and browsing affects population densities. However there is still a dearth of statistically sound research looking at regeneration in New Zealand forests. Until the largely anecdotal, or descriptive, body of information is backed up with empirical and experimental evidence, predictions on the effect of either canopy health or ungulates cannot be made with confidence.

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Chapter 5: Discussion

The aim of this study was to look at the dieback of kamahi (*Weinmannia racemosa*, Cunoniaceae) in Tongariro National Park, to assess whether this species is in decline, to suggest causes of the dieback, and for examining and comparing models of forest dieback.

INVESTIGATIONS INTO KAMAHI DIEBACK

Chapter 1 of this thesis introduced the concept of dieback, and some general models that explain the dieback process, or place it in its ecological context. Dieback is seen as a chain-reaction, either driven largely by factors outside the system, that are disrupting the 'balance' (Houston, 1974), or as a natural and integral part of that system, allowing reproduction (Mueller-Dombois, 1983; 1986). The ecological significance of dieback to forests dominated by species with particular life history strategies is noted. There is currently a lot of interest in human impacts on the environment, and the implication of this, in relation to forests, has been a realisation that trees are dying. Public pressure has led to managers initiating research that often approaches dieback as a problem, rather than investigating whether it has a role in the system. Kamahi dieback in Tongariro National Park was chosen as being an issue in need of research.

A wide ranging survey was conducted to assess the extent of the kamahi dieback phenomenon (chapter 2a). A haphazard but generally representative sample of kamahi habitats and sites around Tongariro National Park were studied. Health was compared to a number of possible causes of dieback that, from the literature, were suspected to be important in kamahi dieback elsewhere in New Zealand. Unhealthy kamahi trees were extremely rare: approximately 4 % of trees from this survey were standing dead, and a further 2.7 % were near death. It was found that health of kamahi was independent of any of the factors examined. For example, presence of possum pellets (an index of possum usage), diameter at breast height (indicating age), and level of attack by pinhole borer (an indication of *Sporothrix* infection), had no consistent relationship with kamahi health in this study. However, the proportion of unhealthy kamahi in the canopy was found to influence the number of small kamahi saplings (> 50cm, < 2m tall) present (an index of kamahi regeneration at each site), with more small saplings under an unhealthy canopy. It was concluded that kamahi is not in a state of decline in this area, and that regeneration of kamahi is largely dependent on any

dieback that does occur. However, there were patches where a high percentage of kamahi were dead or extremely unhealthy.

An area where a seemingly high proportion of kamahi trees appeared to be in ill-health is the western side of Hauhungatahi. A survey of randomly located and aligned transects in this area was undertaken in an attempt to determine the causes of dieback using the three factor 'decline-disease' model of forest dieback (chapter 2b). Factors that have been suggested as predisposing kamahi stems to die, triggering a decline in health or accelerating that decline were compared to measures of stem and stand health to determine their effect. 14 % of trees included were standing dead, and a further 7 % were extremely unhealthy (> 80% of shoots dead). There was no evidence that any of the factors included in this study, such as possum browse, insect browse, or attack by *Platypus* spp. pinhole borer carrying the *Sporothrix* fungus, were triggering dieback. There was some evidence that age may predispose trees to death, with dead trees more likely to be older, although age of itself was clearly not causing death. There was evidence that *Sporothrix* was at most an accelerating (or contributing) factor, as its most likely vector was found fairly consistently in dead trees, but seldom in trees in any other health category.

It was expected that 'triggering' factors would be difficult to determine from surveys; and, were we able to find correlations between suspected triggering factors and tree health, causation would still need to be established. Therefore a more manipulative approach was necessary. An experiment was designed to establish whether dieback could be induced in naturally occurring healthy kamahi, and whether it could be halted in naturally occurring unhealthy kamahi. This experiment was conducted in a patch of kamahi in Erua State Forest, adjacent to Tongariro National Park, to determine the effect of possums, pinhole borer and *Sporothrix* fungus on kamahi in a healthy stand and in a stand exhibiting considerable dieback (chapter 3). Possums were excluded from some trees at both healthy and unhealthy sites; borer attack was simulated at high but naturally occurring levels on some trees in the healthy stand, and *Sporothrix* was introduced into half of these, while some trees in the unhealthy stand were treated with fungicide to eliminate any existing *Sporothrix* infection. None of these treatments had any observable effect on either healthy or unhealthy trees: healthy trees remained healthy, and unhealthy trees remained unhealthy over the 15 - 19 months (depending on treatment) of the experiment, regardless of treatment. This suggests that none of these factors are impacting on the health of kamahi in these stands at this time and treatment scale, and that some other factor may be determining the pattern of dieback.

In conjunction with the previous work, chapter 4 examines the impact of canopy health and vertebrate browsers (none indigenous) on regeneration in a stand of kamahi in Erua State Forest. Plots were selected at a site with a healthy, closed kamahi canopy, and at a site experiencing a high level of dieback, with an unhealthy, open kamahi canopy. At each site some plots were fully enclosed in mesh to exclude all vertebrate browsers, while others were left open. Canopy health did not affect the density of seedlings or species richness of communities beneath it. However, a healthy canopy imposes a strong influence on the type of community regenerating beneath it (regardless of browsing), with all plots under the healthy canopy having very similar species present in similar proportions. Communities regenerating in plots under the unhealthy canopy were much more variable. Excluding browsers from plots allowed a dramatic increase in the density of seedlings in plots under both the healthy and unhealthy canopy, but not of diversity of seedlings.

From these studies, there is no evidence that possum browse, insect browse or pinhole borer/*Sporothrix* are causing kamahi mortality in Tongariro National Park; further, there is no evidence that pinhole borer/*Sporothrix* can cause dieback in healthy trees in this area, or that excluding possums or *Sporothrix* will improve the health of moribund trees in areas with high kamahi mortality. Other studies have been able to show that *Sporothrix* can kill even healthy kamahi (Payton, 1989), and that treatment with fungicide can often halt the decline in health caused by a similar fungus (Dutch Elm Disease) (Lanier, 1987; Greig, 1990). Where there has been good evidence of possums' role in increased tree mortality, there has sometimes been limited response to excluding possums (through poisoning them); Payton (1997) found some heavily defoliated trees recovered after a reduction in possum density, but no general increase in canopy health in the short term. Therefore, although it could have been expected that if *Sporothrix* is important in causing kamahi decline in the area, then the *Sporothrix* treatment of chapter 3 would cause a reduction in tree health, and the fungicide treatment an increase in health; it may have been optimistic to expect that excluding possums would cause an increase in tree health of unhealthy trees, even if possum browse had caused a decline in health.

There is some evidence from the present studies to suggest that in areas where tree mortality is common, age may predispose kamahi to dieback. There is much support for the theory that *Sporothrix* is a factor that hastens any decline in health, but does not cause poor health in kamahi.

There is evidence that some species regenerate better under an unhealthy kamahi canopy than under a healthy closed canopy, and that small kamahi saplings seem

to be more prevalent under a canopy with more dead kamahi trees. Browsing by introduced vertebrates was also found to have an effect on levels of regeneration.

In terms of kamahi in Tongariro National Park, this thesis found dieback to be heavy in patches, but occurring at a low level in the overall region, with regeneration occurring at reducued levels due to browsing. Where dieback was occurring, the proximal cause was not apparent, but larger trees appear more susceptible, and unhealthy trees are more susceptible to attack by pinhole borer.

KAMAHI ECOLOGY

Data from this thesis largely confirm what was already thought or known about kamahi. The maximum height of kamahi recorded in this study was 19 m (considerably less than the maximum of 25 m reported by Salmon, 1980) and the largest stem recorded was 103 cm DBH (again less than the recorded maximum of 120 cm Salmon, 1980). Most trees in this study were single stemmed; however, many had multiple stems (26 % of trees in chapter 5, percentages are not available for chapter 2).

No site in this study was beyond the recorded altitudinal range of kamahi (1100 m above sea level Wardle, 1966). Results from this study may also suggest that kamahi is intolerant of poorly drained sites, with the 'swampy' quadrat 3 on transect 3 of the second chapter being by far the worst drained site, and having a single dying kamahi present (which tree is short in height (less than 10 m), has a small diameter (less than 20 cm DBH), and may or may not have ever looked healthy).

Evidence from this study indicates that kamahi has an all-sized population structure, with a reverse-J shaped frequency distribution of stem DBH. This confirms the result of Lusk and Smith (1998), and supports his and Ogden *et al.*'s (1991) thesis, that kamahi has continual recruitment, and can be thought of as a gap specialist.

Beveridge (1973) speculated that kamahi dieback may be important in the forest system, particularly for regeneration of podocarps. The only data in this thesis that provide any evidence to support the proposed role of kamahi in podocarp regeneration, is that rimu (*Dacrydium cupressinum*) was only found at one site, which was under an unhealthy canopy, in the study of regeneration. Miro (*Prumnopitys ferruginea*) was the only other podocarp present in that study, and it was unaffected by health of canopy. However, there is some evidence that level of kamahi dieback at a site impacts on regeneration of kamahi, with more saplings where more kamahi trees are dying (chapter 2a). This provides support to Lusk *et al.*'s (1998; Ogden *et al.*, 1991) conclusion that kamahi is a gap coloniser.

Because of its prevalence in many New Zealand forests, kamahi is a very visible tree that exhibits obvious dieback. It does not appear to be overly susceptible to exposure, drainage, highly sloping sites, or aspect. Kamahi trees do not appear to 'die of old age', but are likely to be killed at any age. Kamahi is capable of regenerating from seedling banks, seeds, and vegetatively from fallen trees.

UNDERSTANDING THE DIEBACK PROCESS

Nothing is a problem unless we perceive it as such (Ziegler, 1988). Currently there is widespread concern about the existence and effects of global warming. For forest ecologists, this is focusing the need for better knowledge of the processes and dynamics of our forest ecosystems (Manion & Lachance, 1992). In addition, as western society becomes more affluent, conservation issues are capturing the public's attention. Forest conditions that in the past were ignored, or considered normal, are now seen as symbols of the all-pervasive and destructive influence of people on our environment (Kandler, 1992).

The historic disinterest in forests with no obvious economic value has led to a dearth of information regarding the processes in those systems. Forest health has usually been studied either in forests that are never allowed to senesce before they are harvested, or as an aside to some other issue (Jane & Green, 1983). For example, early studies in New Zealand forests were in response to increased erosion in headwaters, causing concern for the 'protection value' of those forests (e.g. Dale & James, 1977; McKelvey, 1959). However, times have changed, and native forests are accepted as having, if not intrinsic value, then at least, aesthetic or scientific values. Now, when the public notices patches of unhealthy looking trees, much pressure is placed on the Department of Conservation in New Zealand (and overseas' equivalents), to provide explanations, and quick remedies (Mueller-Dombois, 1983).

There is not yet a general acceptance of stand-level dieback as an important component in natural processes. Current thinking still sees widespread dieback as indicating a problem, and especially an anthropogenically induced problem. Even when stand-level dieback and widespread catastrophes are part of the natural succession or dynamic of an area, there is a problem when these forests are scaled down to relatively small chunks of a naturally huge system, such as in reserves (Mueller-Dombois, 1987), particularly as these protected areas are often on mountains and other 'marginal' land unsuitable for economic use (Haemmerli & Schlaepfer, 1993). The patch affected by dieback under natural forest conditions may exceed the size of the now protected area, and such areas may be more prone to dieback due to problems of

instability and exposure. Unless a reserve is large enough to include all successional stages of the system, it will be vulnerable to declining condition. The need for fire management in reserves of limited size may also exacerbate problems by upsetting natural processes and increasing the prevalence of standing dead trees (Savage, 1997).

Eucalypt (*Eucalyptus*) dieback occurs in Australia, and though associated with heavy defoliation by insects, produces an inexplicably patchy pattern (Landsberg, 1990a; Landsberg, 1990b; Landsberg, 1990c). In Tasmania, eucalypt dieback was found to be associated with increased densities of possums, which, although indigenous to the area, had achieved well-above natural densities in the proximity of enhanced pastures (Statham, 1995). Widespread, and locally heavy Myrtle (*Nothofagus cunninghamii*) dieback occurs in Tasmania (Elliott, Kile, Candy & Ratkowsky, 1987; Kile & Walker, 1987), with the symptoms appearing very similar to kamahi dieback. It was initially thought that the trees were succumbing to *Chalara australis* infection (a fungus very similar to and possibly in the same genus as *Sporothrix*), and that the fungus was introduced via *Platypus subgranosus.* It has since been shown that although *C. australis* is causing the wilt disease, it was present prior to attack by *P. subgranosus*, and is probably spread by root-grafting (Kile & Hall, 1988; Packham, 1994). *Nothofagus* dieback on Mt Giluwe, Papua New Guinea, was initially thought to be due to attack by a root pathogen that had recently invaded the area, but is now thought to be a natural phenomenon resulting from synchronous establishment and senescence of cohorts (Arentz, 1983); moreover, dieback is thought to be necessary for regeneration of *Nothofagus* in the area. An analogous example is 'ohi'a (*Metrosideros polymorpha*) dieback in Hawai'i; the cause was initially thought to be a disease which could have eliminated much of this forest type, but is now known to be a periodically recurring phenomenon, allowing regeneration (Stemmermann, 1983; Mueller-Dombois, 1986).

The above examples from the Pacific region highlight the focus on natural phenomena and other pests or pathogens in that area. In areas of Europe and North America, where forest dieback is considered an immense problem, air pollutants are traditionally the focus, even though the symptoms of dieback may be the same as in the Pacific region (Hinrichsen, 1987). Huettl (1993) comments that the needle yellowing often seen in pines in Europe has been blamed on acid rain, but the exact same symptoms are interpreted as magnesium deficiency in *Pinus* plantations in New Zealand. In Switzerland, the 'inexplicable forest damage' observed in the 1980s has not escalated, but such high proportions of trees are exhibiting defoliation that it is still considered important (Haemmerli & Schlaepfer, 1993). Haemmerli and

Schlaepfer (1993) go on to comment that present day surveys (damage inventories and defoliation indices) do little to address the question of whether this is a problem, as there are no comparable data from earlier times to act as reference points. Innes (1993), however, notes that European surveys have continued for long enough to show patterns of fluctuating health, and provide evidence rebutting the theories of Waldsterben presented for the above European examples. Kandler (1992) compared photographs of German forest stands taken in the 1930s with recent photographs of the same stands, and found evidence of fluctuation of crown conditions, but not of a deterioration in general forest condition. Moreover, using the same forest damage inventories as are being used to assess health of some species, he assessed the 1930s photographs, and found 'almost none' of the trees of those species would now be classed as healthy, with less than 10 % foliage loss (Kandler, 1992). These stands, that were considered healthy 60 years ago, would have had no exposure to the high levels of pollution that are being blamed for current defoliation, and yet by today's standards would have been assessed as showing decline. Both Hunter (1993) and Haemmerli and Schlaepfer (1993) suggest that it is the cycle of nutrient availabilities through each stand's development that may cause some of Europe's stand-level dieback.

Until the most recent decades, each case of observed dieback was examined as a specific problem, with no extrapolation of conclusions to other cases of dieback. The above examples show the differing views of dieback around the world, and the way these views are changing as information is gained and shared.

IMPLICATIONS FOR THEORIES OF DIEBACK

Models can be a useful way of summarising or simplifying complex systems, and the ability of dieback models to do this was assessed for kamahi dieback. When the key points of the different models of dieback are compared (table 1), it becomes clear that

Table 1 *Comparison of key points of kamahi dieback in Tongariro National Park, with succession and dieback models. Predisposing, triggering and hastening factors are only applicable to dieback models and not the model of succession. ('yes' = necessary to the model)*

	General model	Decline disease	Cohort senescence	Succession	Kamahi
Predisposing factors		Yes	Yes		Yes
Triggering factors	Yes	Yes	Yes		Yes
Hastening factors	Yes	Yes	Yes		Yes
Stand-level		Yes	Yes	Yes	Yes
Cohorts important			Yes	Yes	(unknown)
Catastrophic disturbance			Yes		Yes
Mosaic pattern			Yes	Yes	(unknown)
Regeneration important			Yes	Yes	Yes
Invasion important			Yes		Yes
Stand age important			Yes		(maybe)
Dieback is a problem	Yes	Yes			(maybe not)

the main differences are in specificity. The general model is most general, the cohort senescence model most specific. The cohort model takes the key elements of the decline disease model, and combines them with a model of succession.

The information we have about kamahi dieback in Tongariro National Park can be included in this comparison. It contains all the elements of the general and the decline disease models, with the exception of whether or not it is a problem - in other areas (such as Westland rata-kamahi forests, where a much greater extent of the forest is affected, Stewart & Rose, 1988) kamahi decline is certainly a problem; in Tongariro National Park this has not yet been established. Of the 23 sites in the broad-scale survey of kamahi health that were inside the Park, only 4 % of trees were dead, suggesting that dieback may not be a problem for maintaining kamahi in a state of good health in the overall Park. If this is so, the general and decline disease models of dieback can be rejected.

Kamahi dieback contains all the key elements of the cohort senescence model. The results also fit Beveridge's (1973) model of succession, which sees kamahi dieback as an integral part of the forest system and its successional processes. Kamahi provide cover for seedlings, and when canopy dieback occurs, these seedlings are released. However, the existence of the mosaic pattern of development and health, and the presence of spatially discrete cohorts of kamahi, has not been established. Obviously these two points are crucial to the cohort senescence model, but none of the research in this thesis addresses how they apply to kamahi in Tongariro National Park. In order to answer these questions, a survey of kamahi health is necessary, including all areas of the Park where kamahi is present (not just where dieback is apparent), and at a scale that may identify changes in health over sometimes small areas - the most efficient method would be through use of aerial photographs taken at a height where it is possible to identify species of tree (Rogers & Leathwick, 1997).

It appears from this comparison that the successional model best describes what we currently know about kamahi dieback in Tongariro National Park; although it is necessary to investigate the importance of cohorts and the presence of a mosaic pattern of health, as described above.

The Mueller-Dombois (1983; 1986) model of cohort senescence with the resulting mosaic of stands, also seems to fit kamahi dieback. However, if 'senescence' is defined by an 'over-mature' life-stage, there are problems with fitting this model to kamahi in Tongariro National Park: data from this thesis do not find that older trees or stands were more likely to be dead, although data do indicate that dead trees were more likely to be older than live trees.

Houston's (1984) general model of dieback may not apply to kamahi dieback in this area, as it implies a disorder in the forest system (Mueller-Dombois, 1983). Clearly, the levels of dieback recorded for kamahi in Tongariro National Park show a localised phenomenon occurring on the western slopes of Hauhungatahi south of Erua (chapter 2b), with the health of kamahi in the overall area looking good (chapter 2a).

For the same reason, the decline disease model of dieback may not explain kamahi dieback in Tongariro National Park; however, the data from chapter 2b do largely fit the decline-disease model of dieback, with age suggested (albeit tentatively) as a predisposing factor, *Sporothrix* as an accelerating factor, and an unknown initiating factor.

Despite its apparent applicability and widespread use in explaining dieback, when the decline disease theory is examined, it appears to be able to explain, not just 'all known examples of dieback in New Zealand *Nothofagus* spp., *Metrosideros* spp., and beech/hardwood forests' (Stewart, 1989: 243), but all known examples of death, animal or plant, accidental or pathological. From the tree on a windy ridge (= predisposing factor) that gets blown over in a storm (triggering factor), and the truncated trunk attacked by boring insects searching for brood material (hastening factors), to the drunk motorist who hits a tree and bleeds to death, to the classic example of forest decline, *any* death can fit this model. It is clear from this that the three factor theory does not explain forest dieback, but rather *describes* the process of death, any death. And although this theory is treated as a model of disease, which implies a problem in the system (Mueller-Dombois, 1983), it describes death when this is part of a natural system just as well.

The theory undoubtedly provides a useful framework for investigating dieback phenomena: it highlights that in all deaths there are factors that lead to one individual being more susceptible to stress than other individuals, or at one time rather than another; that there are factors which cause a decline in health, and other factors that 'finish off' the individual. But it does not address questions such as whether the dieback is occurring at natural levels, or if it is endangering the forest integrity. For this, a number of the other models discussed in chapter 1 are necessary. These models can be combined and a framework constructed for ascertaining how the dieback fits into the natural forest system, and the extent and potential severity of any dieback (fig 1). The resulting model will not only indicate any causes of dieback, but will also determine whether the dieback poses a problem for the continued integrity of the forest system.

Model of Dieback Management

This model highlights the importance of understanding the stand dynamics and succession in the forest of concern (Mueller-Dombois, 1983), and the necessity for good quality research and information on 'healthy' forests. To flow through the model, at point a), someone notices that trees are dying, and reports this to the appropriate authority. That authority then has to decide if any action is necessary. The first stage (question 1 in fig 1) in determining if anything needs to be done is finding out if the deaths are unusual, or whether they are simply a part of the natural succession of the species concerned. This requires knowledge of the species' successional process (from literature), and knowledge of the patterns of healthy and unhealthy trees, and regeneration of the species in the area (from surveys). It is important to study the wider area at this stage, and not to focus on small areas where dieback may be focused, because succession does not operate on a small scale in forest systems (Mueller-Dombois, 1983; Beveridge, 1973).

If the patterns of mortality do not fit the known patterns of succession for those species (for example, if only young trees are affected, or if dieback is occurring at a stand level, where only individual deaths had previously been recorded for the species), or data on either or both of the patterns is not available, the manager should then try to answer question 2. The cohort senescence model is outlined by Mueller-Dombois (1983; 1986) and requires an environment with a catastrophic disturbance regime (where catastrophic is defined as large-scale), a species able to take advantage of the disturbance, and the time between disturbances to be longer than the generation turn-over of the species (kamahi in Tongariro National Park would fit these criteria). To fit this model, dieback should have a patchwork pattern based on site suitability, with successive cohorts smaller and more out of phase with each other until reset by catastrophic disturbance. The key factor in determining the patches of sites undergoing dieback (by affecting development of stands) is likely to be edaphic, as on a patch (or small) scale, this factor more than any other determines the rate of development at a site - patches at later stages of development should be more susceptible to stress (Steel, 1989; Agyeman & Safo, 1997).

If the situation does not meet the criteria for the cohort senescence model, or data from research do not fit the model, or are unavailable, then the rate of mortality should be determined (question 3). If there has been an increase in mortality rates of the species of concern, then the dieback may be a problem. If data are unavailable to answer this question, then in the interest of risk aversion, it should be assumed that dieback may be a problem (point b).

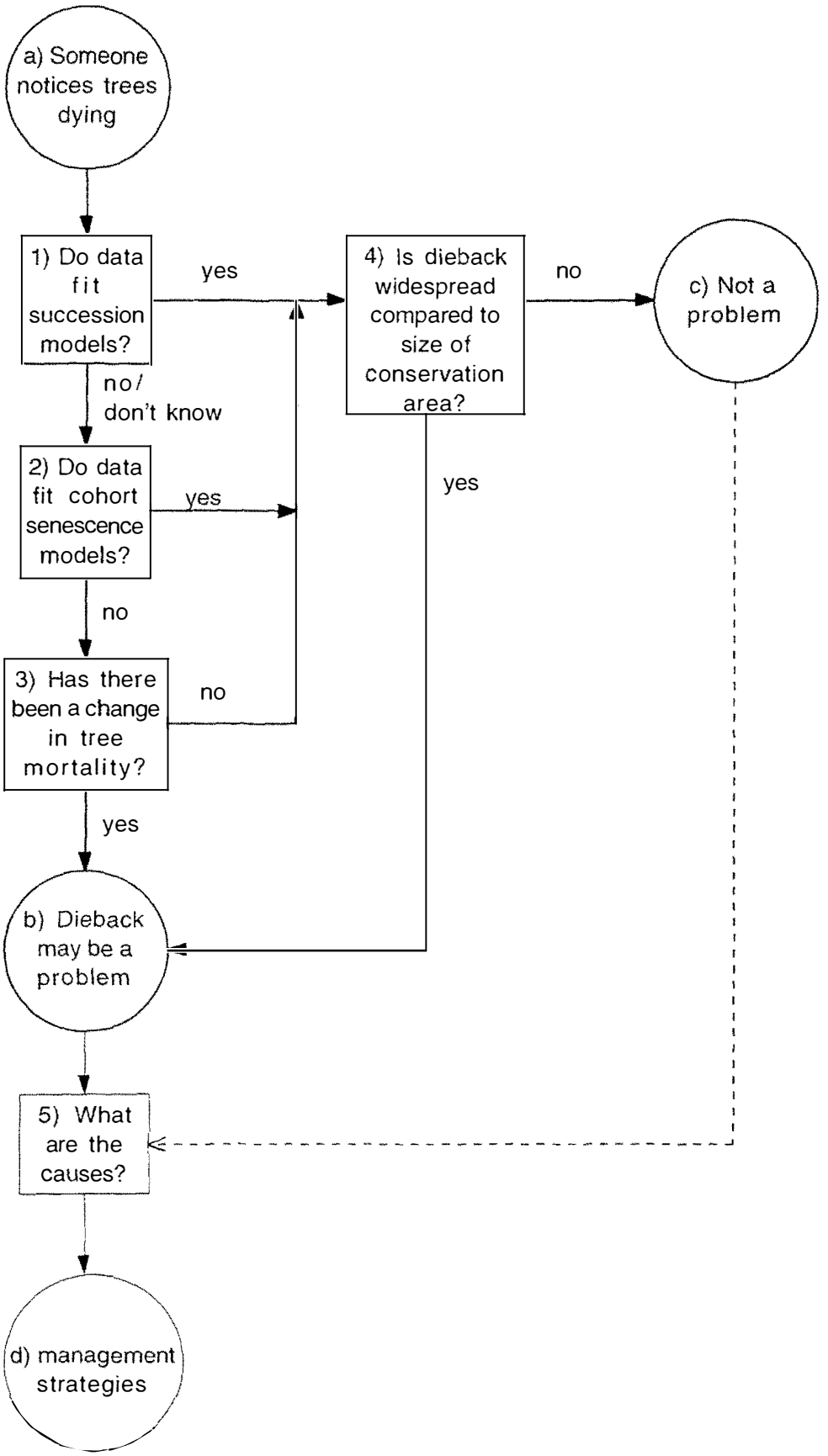

Fig 1 *Model for investigating forest dieback. Circles indicate temporary endpoints, squares indicate research questions. Solid lines indicate fixed paths, dotted lines indicate possible path (for example, if interested).*

If research shows that data do fit the model of succession for the species of concern, or the cohort senescence model, or there has been no increase in tree mortality, then the dieback is likely to be a completely natural process for those species. As long as the forest area is large enough to accommodate this process (given that most forests have been severely reduced in area), the dieback is unlikely to cause any problem (point c). However, if the dieback occurs over a large area in a forest of reduced size (as most parks and reserves are), then it is likely to be a problem, whether or not it is a natural occurrence.

If this model indicates that dieback may be a problem, then the next step is obviously determining causes (question 5). The most effective way of achieving this will be a long term study (Innes, 1993; Fisher, 1997) looking at all factors that may be involved, including edaphic and climatic factors. The three-factor decline-disease model provides a useful framework for such a study, with different expectations for the relationships between predisposing, triggering and accelerating factors, and tree health. Predisposing factors are generally not capable of killing a tree, and should always be associated with trees or stands that are dead or in poor health, and not so strongly associated with healthy stands - any correlations should be weak. Triggering factors are extremely difficult to discover from single visit studies, as the factor that initiates dieback may no longer be present when the tree is dead (Landmann, 1993). Correlations between accelerating factors and tree health may not be significant because they do not necessarily affect unhealthy trees, but they will be strongly associated with dead trees. If long term studies are possible, these relationships will be more easily determined. Once these studies have determined the most important factors in dieback, experimentation is necessary to prove causation (Manion, 1991; Power & Ashmore, 1996).

When causes of dieback have been determined, appropriate strategies for control can be devised (point d). In many cases, management will decide action is necessary before attempts have been made to answer all questions. These actions will be based on no more than best guesses, and there is just as great a chance that resources will be wasted fighting factors that are in fact of little importance in determining health of the species concerned, and more importantly wasting the time that is so precious that it led to this premature action, as there is a chance that the actions will successfully increase the health of the forest, or halt the decline.

If action *must* be taken before the model is implemented, then as much information as possible should be collected to give managers a fighting chance. A list of all possible causes of dieback should be compiled, based on literature, observation and

creative thinking. Any factors on this list that the manager cannot realistically control (for example, most predisposing factors, drought, and in some cases exotic animals if the conservation area is vast and few resources available) should then be disregarded. If money becomes available for research, their role can be investigated at a less pressing stage. Any factors that are *clearly* unlikely to be killing the species of concern in the area (for example, if dieback has been observed for the last 20 years, but possums have only invaded in the last decade), can also be eliminated. The resulting list will probably include a handful or less of the most likely causes that a manager might be able to affect. Unless an unlimited amount of resources is available, at this stage a survey must be conducted in an effort to eliminate any or all of the remaining factors, and the extent of the dieback should be estimated in some way. If all factors that are within control of the manager can be eliminated, then the management strategy will be frustratingly simple - hope that one day resources and information will be available to do more than watch the forest die. If only one or two factors could not be eliminated, then ideally these would be studied further; if this is not possible, then control should be initiated, with adequate monitoring of forest health before and during control to assess its success. Care needs to be taken to avoid self-fulfilling monitoring strategies; this may require the use of control areas and the inclusion of healthy and unhealthy areas, as monitoring only the unhealthy areas increases the likelihood of finding an improvement in health regardless of treatment, especially if dieback is a natural part of the system.

CONCLUSIONS FOR KAMAHI IN TONGARIRO NATIONAL PARK

Implications from this study are encouraging. Despite the appearance of seemingly large numbers of dead and dying trees at forest margins in Tongariro National Park, and small patches where dieback is affecting a large proportion of trees, results from this study suggest that, as far as kamahi is concerned, the forests are in a healthy state, and dead trees are only a minor proportion of the total population. These results do not dispute that widespread dieback or decline of kamahi is happening in other areas, nor that other canopy species in the Park may be in a better or worse state of health. They do indicate that dieback of kamahi in this region is not occurring at an abnormally high, or unsustainable, rate over the area where kamahi occurs, despite the existence in the Park of smaller areas where dieback is occurring at high rates.

Nor do these results dispute that factors such as possums or *Sporothrix* infection can cause dieback of trees or stands of trees; rather, they indicate that in Tongariro National Park, these factors are not important in determining the pattern of

kamahi dieback. The forests of Tongariro National Park have lower densities of possums (4-6 possums per hectare, C.Speedy *pers.com.*) compared to other forested areas where possums are impacting (e.g. ca. 8 possums per hectare in the Orongorongo Valley, Wellington, Brockie, 1992). Little sign of possums was encountered while undertaking this thesis; there was certainly not the visible possum damage seen in areas such as the West Coast (pers. obs.). This may be the reason possums are not impacting on kamahi in Tongariro National Park. The effect of *Sporothrix* on trees is enhanced by moisture stress (Faulds, 1977). The areas of kamahi in Tongariro National Park are on the western and southern sides, where precipitation is both consistent and high (mean annual rainfall at nearby met. stations is 2850 mm (Chateau), 2620 mm (Ohakune)). This may be the reason *Sporothrix* had such a minor impact on the health of trees in this study. There is also the possibility that, like *Chalara australis*, *Sporothrix* is not only spread by *Platypus* spp., but may be capable of infecting a tree through roots. While this would not affect the findings of the experiment (chapter 3), it would mean that neither of the surveys were capable of detecting its presence.

However, regeneration is being maintained at a lower level than would occur in the absence of vertebrate browsers. Whether this is impacting on the composition of the forest at present, or will impact on the future forest composition, remains to be seen (Campbell, 1990).

MANAGEMENT IMPLICATIONS

This thesis raises a number of issues for management of kamahi in Tongariro National Park:

1. TREATMENT OF DIEBACK

Attempts at managing patches of dying kamahi, for example, by banding for possums or fungiciding for *Sporothrix*, will be expensive and time-consuming, and are unlikely to be successful.

2. MAINTENANCE OF POSSUM DENSITY

Reducing density of possums in the Park is unlikely to have much effect on the health of kamahi, as they do not appear to be exerting much influence over kamahi health; however, this may be due to currently low densities of possums in the Park. If possum densities increase, possums may begin to affect the health of kamahi. With this in mind, it is advisable that possum densities be maintained at (or below) the present densities.

3. DISSEMINATION OF INFORMATION TO PUBLIC

The Department of Conservation manages New Zealand's national parks on behalf of the public of New Zealand. When an apparently high density of dead trees is visible from a main highway, as is the case with the western side of Tongariro National Park, members of the public are bound to become concerned (see also Mueller-Dombois, 1983). Information regarding the nature of kamahi dieback and the scale of its occurrence should be made available to the public, in order to assuage these concerns.

4. RESEARCH

When visible dieback of the dominant canopy species of an area occurs, there is a tendency to assume, first, that there is a problem, and second, that there is a simple solution. It is important to first identify whether this dieback is likely to be abnormal or problematic, before trying to isolate possible causes. This will involve research and, ideally, long term monitoring. Monitoring is the key to increasing our understanding of forest processes, and without an understanding of these, it is very difficult to make appropriate management decisions regarding our forests. The difference in the two surveys highlights the importance of where and how research is conducted. Experimentation is the key to determining the causal factors in any forest dieback.

FUTURE RESEARCH

This thesis has highlighted many areas where research is necessary. As discussed above, it is crucial to begin collecting data on 'normal' forests and rates of mortality, otherwise the first three questions of the Dieback Management model may be unanswerable. Unfortunately, in the present funding regime where the few pennies available for conservation work are needed for ensuring public safety and crisis management, it is unlikely that such research will be well funded. And such 'descriptive' baseline studies are no longer fashionable. Chapter 2b of this thesis contains data that form part of a long term study initiated by the Department of Conservation in 1997. While this already has potentially great consequences for New Zealand forest ecological science, if it could be expanded to include more of the Park (where dieback is not so apparent), it would be even more useful. It is only through the use of temporal comparisons that the effect of factors suggested to be causal (such as possums) will be known with anything approaching certainty (Fisher, 1997).

Aerial surveys can be useful for providing an indication of the extent and pattern of any dieback. Problems may arise when the trees being surveyed are lower canopy (as for kamahi), and for myrtle wilt disease in Tasmania, preliminary trials of aerial surveys found they did not provide accurate information with only a proportion of the most recent deaths detected (Elliott *et al.*, 1987). The Department of Conservation has conducted an aerial survey of kamahi dieback covering the area included in chapter 2b; if the technique is found to be accurate for kamahi, it is an obvious approach.

As well as investigating the large scale patterns of dieback, it may be useful to investigate the fine scale pattern of tree health. Elliott *et al* (1987) looked at nearest neighbour distances to quantify the degree of 'clumpedness' of trees suffering myrtle wilt. They found a significant clumping effect, which is indicative of diseases.

The direction of research into forest decline has changed recently. There has been increasing interest in studies that have focused attention below ground rather than in the canopy. Waring (1987) comments that demand for nutrients in a forest may be greater than that returned to the soil by normal leaf fall, root turnover, and decomposition. This may be exacerbated by the removal of timber or herbivores' bodies. Landsberg and Gillieson (1995) suggest that trees may be more resilient where soil nutrient levels are high. Liu and Tyree (1997; also Liu, Ellsworth & Tyree, 1997) suggest that acidic soils with low soil cation availability may be the underlying stress in sugar maple decline. This confirmed the earlier findings of Ouimet *et al.* (1996). Power and Ashmore (1996) found healthy northern beech trees had higher proportions of live mycorrhizal roots than

unhealthy trees, and that there were higher concentrations of calcium, magnesium and potassium and lower aluminium/calcium ratios under healthy trees. Agyeman and Safo (1997) predict that plant nutrition is one of the most important factors influencing tree dieback.

So far in New Zealand there have been few studies of the effect of soil nutrient levels on forest health. Clarkson and Clarkson (1995) looked at soil development in their study of vegetation changes on Mt Tarawera; however, theirs is a study of succession rather than health. Green and Jane (1983) and Hosking and Hutcheson (1986) look at the effects of soil moisture levels, but not nutrients. Clearly, this is an area of research that is lacking in the New Zealand context.

Climate is of course another area where much research is needed. It has been found to either predispose sites to dieback (e.g. Fisher, 1997; Auclair, Lill & Rivenga, 1996; Grant, 1963) and/or to initiate dieback (e.g. Auclair *et al*., 1996; Grant, 1963; Landmann, 1993). If times are known for the origins of declines in stand health, these can be compared to climate data, to investigate the existance of any relationships. However, it is very difficult to find meaningful patterns (Akashi & Mueller-Dombois, 1995), and should perhaps be left until other avenues have been explored.

Because of the stress forest managers place on the role of possums in kamahi dieback, it would be useful to expand our knowledge of this relationship. Experiments with adequate controls could be conducted to investigate the effect on forest or kamahi health of poisoning possums. Defoliation experiments could be conducted to investigate at what level of defoliation a tree will decline in health, both temporarily and irreversibly. The results from the defoliation experiment could be compared to an analysis of possum stomach contents from an area of known vegetation. The stomach contents could indicate whether the species of concern in that area was being attacked by possums to the level indicated by the defoliation experiment as likely to impact on tree health.

The relationship between *Chalara australis* and *Sporothrix* needs clarifying. With the inconsistency of fungal classifications, it is not inconceivable that these two fungi are of the same genus. Certainly, symptoms of their infection in trees are virtually identical.

This thesis shows that if forest mortality is examined without bias, the mortality may be found to be less important than expected, as well as less easily interpreted. Forest dieback is still a problematic issue for managers and scientists alike, but rigorous methods can be illuminating.

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