THE UNIVERSITY OF
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QUANTIFICATION OF THE RISK OF PHYTOPHTHORA DIEBACK IN THE GREATER BLUE MOUNTAINS WORLD HERITAGE AREA

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Declaration of Originality

This dissertation presents the results of research conducted at the Royal Botanic Gardens, Sydney, of the Botanic Gardens and Domain Trust and the Faulty of Agriculture and Environment, The University of Sydney.

The content is, to the best of my knowledge, original and contains no work formally published or written by another person except where dully acknowledged. This thesis does not include any material that has been submitted or accepted to any institution for a degree of diploma.

Signed,

Zoe-Joy Newby

Abstract

Biological invasions exert great pressure on natural ecosystems and conservation areas, the latter of which have been established to conserve biodiversity. The presence of invasive species in natural ecosystems disrupts evolutionary processes, alters species abundance and can potentially lead to extinction (Mack *et al.,* 2000; Crowl *et al.,* 2008). When an invasive species is the cause of plant disease, the potential for that pathogen to survive in a new environment and the expectation of the impacts it may cause, can be estimated from locations where it already occurs. Understanding the dynamics of disease is important for management and research alike, and will hopefully make way for a proactive rather than reactive response.

Disease in natural Australian ecosystems caused by the invasive species *Phytophthora cinnamomi* has been recognised for nearly 100 years (Newhook and Podger, 1972); its devastating impacts have lead to the disease syndrome, Phytophthora dieback, being classified as a Key Threatening Process by the Australian Federal Government (Commonwealth of Australia, 2005). Yet, the assessment of potential disease establishment, that is, disease risk, is limited. This remains true for the globally significant Greater Blue Mountains World Heritage Area (GBMWHA) in New South Wales, a centre of plant and animal conservation. Not only is the understanding of the pathogen distribution limited, so too is knowledge of the potential impacts on flora and the influence climate change may have on disease expression. Management of Phytophthora dieback in the GBMWHA is made increasingly complex by the rugged and remote nature of much of the World Heritage Area, as well as competing demands from tourism, recreation and the impacts of fire and other introduced species. This study aims to address some of these complexities by establishing the suitability of the GBMWHA to *P. cinnamomi*, its current distribution and the potential for disease. Additionally, with the difficulty of accessing much of the GBMWHA and the risk of disease transmission in mind, an alternate approach to disease identification is trialed.

The first task of this project, was concerned with understanding the potential distribution of *P. cinnamomi* within the GBMWHA using mechanistic modelling and information on the pathogen's ecology. Most of the GBMHWA was found to be suitable, leading to the acceptance of the first hypothesis that the climatic and topographic conditions of the GBMWHA are conducive to *P. cinnamomi* establishment. The most conducive areas were characterised by high soil wetness, high rainfall and moderate temperatures, while the areas least conducive were conversely hotter and drier. Although

the model appeared to overpredict into areas the pathogen was not found, increasing distribution risk was associated with increasing isolations, possibly indicating that the pathogen is yet to reach its potential niche.

The modelled distribution of *P. cinnamomi* was then used to inform a field investigation to determine the actual distribution in the GBMWHA and assess the impact of the pathogen on vegetation communities and individuals. As an invasive species, the distribution of *P. cinnamomi* was hypothesised to be primarily found in locations with high anthropogenic activity; however it was isolated extensively from remote areas, leading to the rejection of this hypothesis. Disease was never the less expected, albeit sporadic, as per disease expression in other vegetation communities in New South Wales (Arentz, 1974; Walsh *et al.,* 2006; Howard, 2008). Heathland communities that often have a higher incidence of disease (McDougall and Summerell, 2003), had a high rate of pathogen isolation, as well as clear indications of disease in the GBMWHA. Additionally, freshwater wetlands, many of which are endangered ecological communities under Commonwealth and State legislation, had a high rate of pathogen isolation also.

The results collected during the field work were then utilised to assess the risk of Phytophthora dieback occurring in the GBMWHA within the context of the disease triangle. The distribution of *P. cinnamomi* was combined with models of over 130 individual host species to produce a spatially explicit model, quantifying the risk of disease. That a large portion of the GBMWHA is at risk of Phytophthora dieback was not the case, and as such this hypothesis was rejected. Although much of the World Heritage Area had a least some level of risk, greatest risk was associated with a few small areas that occurred at higher elevations with suitable rainfall and temperature conditions. Unfortunately, many of these locations were associated with high levels of tourism and recreation, highlighting the potential for anthropogenic dispersal of *P. cinnamomi* into, around and out of the GBMWHA.

Disease itself has a temporal element which cannot be quantified in one set of field results and as disease spreads the results become outdated quickly (O'Gara *et al.,* 2005). Field-based assessments of disease are expensive and time consuming, and in area as vast and rugged as the GBMWHA, difficult and potentially dangerous. Real-time information on the impacts of disease are therefore needed by land managers to efficiently deploy management strategies (O'Gara *et al.,* 2005). Remote sensing offers an alternative means of assessment not requiring site entry. Vegetation condition can be assessed remotely in all manner of plant systems including the detection and quantification of disease. As such, it was hypothesised here that infection caused by *P. cinnamomi* could be detected from

remotely-sensed reflectance and distinguished from spectral changes caused by water stress. To test this theory, five commonly occurring species within the GBMWHA were infected with *P. cinnamomi* and their foliar responses were monitored over several months. *Phytophthora cinnamomi* infection was detected by assessing water content and vegetation indices, and when data dimensionality was reduced using principal component analysis. The response of individual species to *P. cinnamomi* was, however, variable and difficult to identify once water stress had become severe. *Phytophthora cinnamomi* infection did not appear to invoke a unique spectral response, and this hypothesis was rejected. However, infection was detectible in some species outside the visible range suggesting the potential for remote sensing to identify presymptomatic disease or disease in asymptomatic plants infected with *P. cinnamomi.*

The results of this research will improve the management of *P. cinnamomi* in the GBMWHA. Prioritisation of management strategies will be supplemented with the understanding of which areas are at greatest risk of disease, which areas can potentially be protected from infection and what activities are associated with disease. Such information is useful to not only land managers, but other park users including locals, tourists and recreationalists.

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And finally to my creator, God, the one who gives me strength and knows the plans intended me for; thank you for your graciousness and love.

I wish to dedicate this work to my mother, Cheryl June Newby – we did it.

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List of Abbreviations and Units

1d	First derivative
AC	*Angophora costata*
ALA	Atlas of Living Australia
ANOVA	Analysis of variance
aREP	Area under the red edge peak
ARI	Anthocyanin reflective index
ASCII	American Standard Code for Information Interchange
AUC	Area under the ROC curve
BIOCLIM	Bioclimatic Prediction and Modelling System
BMAD	Bell Minor-associated dieback
BMNP	Blue Mountains National Park
C	control
˚C	Degrees Celsius
cm	centimetres
CMA	Catchment Management Authority
CR	Conservation reserve (Jenolan Karst CR)
d.a.i.	Days after inoculation
DECC	NSW department of Climate Change (now OEH)
DECCW	NSW department of Climate Change and water (now OEH)
DEM	Digital Elevation Model
DWO	Distance Weighted Overlay
EA	Environment Australia
ENVI	Environment for visualising images
EPPO	European and Mediterranean Plant Protection Organisation
FC	Field capacity
GBMWHA	Greater Blue Mountains World Heritage Area
g	grams
GDA94	Geodetic Datum of Australia, 1994
GIS	Geographical Information System
HNCMA	Hawkesbury Nepean Catchment Management Authority
GPS	Global positioning system
HRS	Hyperspectral remote sensing
HSD	Honest Significant Difference
ID	identification
kg	kilograms
km	kilometres
kPa	kilopascals
MGA56	Map Grid Australia, Zone 56
m	metres
Maxent	Maximum Entropy
MIR	Middle infrared
ml	millilitres
min	minute
mmpa	Millilitres per annum
msl	Metres above sea level
NDVI	Normalised difference vegetative index
NIR	Near infrared
nm	nanometres

NP	National Park
NPWS	NSW National Parks and Wildlife Service
NSW	New South Wales
OEH	Office of Environment and Heritage
P	inoculated
PC	Principal component
PCA	Principal component analysis
P/L	Proprietary limited
PSM	Phytophthora selective media
QLD	Queensland
RES	Red edge slope
ROC	Receiver Operator Curve
SD	Standard deviation
SWIR	Short-wave infrared
VIC	Victoria
v/v	Volume per volume
μ	mean
UK	United Kingdom
USA	United States of America
UV	Ultra violet
WA	Western Australia
WHA	World heritage area
WS	Water stress
WSP	Water stress and inoculated
%	percent
~	approximately
≈	approximately equal to
˚	degrees (slope)

List of Species and Authorities

Acacia dealbata Link
Acacia melanoxylon R, Br.
Acacia myrtifolia (Sm.) Willd
Acacia oxycedrus Sieber ex DC
Acacia paradoxa DC
Acacia parramattensis Tindale
Acacia suaveolens (Sm.) Willd
Acacia terminalis (Salisb.) J.F.Macbr.
Acrotriche serrulata (Labill.) R.Br.
Allocasuarina littoralis (Salisb.) L.A.S.Johnson
Allocasuarina paludosa (Sieber ex Spreng.) L.A.S.Johnson
Allocasuarina verticillata (Lam.) L.A.S.Johnson
Amperea xiphoclada (Spreng.) Druce
Angophora costata (Gaertn.) Britten
Anisopogon avenaceus R.Br.
Aotus ericoides (Vent.) Don
Astroloma humifusum (Cav.) R.Br.
Banksia ericifolia L.f.
Banksia integrifolia L.f.
Banksia marginate Cav.
Banksia paludosa R.Br.
Banksia serrata L.f.
Banksia spinulosa var collina (R.Br.) A.S.George
Banksia cunninghamii Sieber ex Rchb
Bauera rubioides Andrews
Bossiaea obcordata (Vent.) Druce
Bossiaea prostrate R.Br.
Brachyloma daphnoides (Sm.) Benth.
Burchardia umbellate R.Br.
Callitris rhomboidea R.Br. ex A.Rich. & Rich.
Calytrix tetragona Labill.
Cassytha glabella R.Br.
Cheilanthes austrotenuifolia H.M.Quirk & T.C.Chambers
Correa reflexa (Labill.) Vent.
Corymbia gummifera (Gaertn.) K.D.Hill & L.A.S.Johnson
Corymbia maculate (Hook.) K.D.Hill & L.A.S.Johnson
Cryptandra ericoides Sm.
Daviesia latifolia R.Br.
Daviesia ulicifolia Andrews
Dianella revoluta R.Br.
Dillwynia glaberrima Sm.
Dillwynia phylicoides A.Cunn.
Dillwynia sericea A.Cunn.
Dodonaea viscose Jacq.
Eucalyptus botryoides Sm.
Elaeocarpus holopetalus F.Muell.
Entolasia stricta (R.Br.) Hughes
Epacris microphylla R.Br.

Epacris obtusifolia Sm.
Epacris paludosa R.Br.
Eucalyptus camaldulensis Dehnh.
Eucalyptus consideniana Maiden
Eucalyptus dalrymplenana Maiden
Eucalyptus dives Schauer
Eucalyptus fastigata H.Deane & Maiden
Eucalyptus globoidea Blakely
Eucalyptus macrorhyncha F.Muell. ex Benth.
Eucalyptus radiata Sieber ex DC.
Eucalyptus rossii R.T.Baker & H.G.Sm.
Eucalyptus saligna Sm.
Eucalyptus sideroxylon Woolls
Eucalyptus sieberi L.A.S.Johnson
Eucalyptus smithii R.T.Baker
Eucalyptus tereticornis Sm.
Eucalyptus viminalis Labill.
Gleichenia dicarpa R.Br.
Gonocarpus teucrioides DC.
Goodenia hederacea s.l. Sm.
Gymnoschoenus sphaerocephalus (R.Br.) Hook.f.
Hakea dactyloides (Gaertn.) Cav.
Hibbertia riparia (R.Br. ex DC.) Hoogland
Hovea linearis (Sm.) R.Br.
Isopogon anemonifolius (Salisb.) Knight
Isopogon fletcheri F.Muell.
Lambertia formosa Sm.
Lepidosperma laterale R.Br.
Lepidosperma longitudinal Labill.
Lepidosperma urophorum N.A.Wakef.
Leptospermum continentale Joy Thomps.
Leptospermum trinervium (Sm.) Joy Thomps.
Lepyrodia scariosa R.Br.
Leucopogon ericoides (Sm.) R.Br.
Leucopogon esquamatus (R.Br.) Benth.
Leucopogon lanceolatus (Sm.) R.Br.
Leucopogon virgatus (Labill.) R.Br.
Lobelia gibbosa Labill.
Lomanadra confertifolia (F.M.Bailey) Fahn
Lomandra longifolia Labill. var. longifolia
Lomandra oblique (Thunb.) J.F.Macbr.
Lycopodium deuterodensum Herter
Macrozamia communis L.A.S.Johnson
Melaleuca squamea Labill.
Melaluca styphelioides Sm.
Monotoca elliptica (Sm.) R.Br.
Nematolepis squamea (Labill.) Paul G.Wilson
Patersonia glabrata R.Br.
Persoonia levis (Cav.) Domin
Persoonia linearis Andrews
Petrophile canescens A.Cunn. ex R.Br.
Petrophile pulchella (Schrad. & J.C.Wendl.) R.Br.

Petrophile sessilis Sieber ex Schult. & Schult.f.
Phytophthora cinnamomi R. D. Rands
Pimelea linifolia s.l. Sm.
Platylobium formosum Sm.
Platysace lanceolata (Labill.) Druce
Poa sieberiana Vickery
Polyscias murrayi (F.Muell.) Harms
Pteridium esculentum (G.Forst.) Cockayne
Pterostylis concinna R.Br.
Ptilothrix deusta (R.Br.) K.L.Wilson
Pultenaea retusa Sm.
Pultenaea scabra R.Br.
Schoenus imberbis R.Br.
Selaginella uliginosa (Labill.) Spring
Sprengelia incarnate Sm.
Stylidium graminifolium s. l. Sw. ex Willd.
Tetraria capillaries (F.Muell.) J.M.Black
Tetrarrhena juncea R.Br.
Tetratheca glandulosa Sm.
Themeda triandra Forssk.
Tricoryne elatior R.Br.
Xanthorrhoea australis R.Br.
Xanthorrhoea glauca subsp. Glauca D.J.Bedford
Xanthosia atkinsoniana F.Muell.
Ziera covenyi J.A.Armstr.

Chapter 1 General Introduction

1.1 The basis of successful disease management

Successful management of disease caused by the plant pathogen *Phytophthora cinnamomi* in natural ecosystems is dependent upon understanding the relationships between the pathogen, host and environment. Disease caused by species of *Phytophthora* also affect agriculture and horticulture around the world and are one of the most devastating groups of plant pathogens known to man (Erwin and Ribeiro, 1996). In the globally significant Greater Blue Mountains World Heritage Area (GBMWHA), information on the distribution of *Phytophthora* and its impacts on plants and ecosystems within the area is limited. Environmental degradation caused specifically by *P. cinnamomi* is a potential threat to the criteria for which World Heritage status was granted. Under the World Heritage Convention, it is the responsibility of land managers to protect the quality of these World Heritage properties. Should they fail, World Heritage status may be lost. Management efforts in the GBMWHA, as with other natural environments in Australia and around the world, are thus flawed by the lack of site-specific information available on *P. cinnamomi*, which can potentially undermine any actions taken. For land managers to manage disease appropriately, that is, in a timely and proactive manner, an understanding of the risk posed *by P. cinnamomi* to specific areas needs to be developed with consideration of the pathogen's distribution, the distributions of hosts, and the environment in which disease is considered a potential threat.

1.2 Assessing the risk of disease

Invasive species and diseases are the second largest cause of extinction globally, second only to humans (Crowl *et al.,* 2008). Disease has detrimental impacts on human health and welfare as well as ecosystem function and stability (Crowl *et al.,* 2008; Yang, 2006). Globalisation is increasing the spread and frequency of disease, and changing the way disease is expressed (Crowl *et al.,* 2008; Yang, 2006). Climate change additionally influences disease expression, both in Australia and around the world (Chakraborty *et al.,* 1998; Chakraborty *et al.,* 2000; Crowl *et al.,* 2008). These impacts can be reduced via disease mitigation and prevention which in turn depends upon well-informed management decisions (Yang, 2006). Successful disease management relies on assessing disease risk.

Risk is defined in the Standards Australian/Standards New Zealand as "effect of uncertainty on objectives" and notes that it is often expressed in terms of the potential of an event occurring, which may include the consequences and likelihood of that event (2009). Diseases are a risk to natural ecosystems because they influence their functionality and diversity (Holdenrieder *et al.,* 2004). They may directly impact native species or allow a competitive advantage to other species unaffected by disease (Mack *et al.,* 2000). In so doing, disease can modify species richness and abundance and influence landscape patterns by way of altering fire regimes, water quality and biochemical cycling (Mack *et al.,* 2000; Holdenrieder *et al.,* 2004; Crowl *et al.,* 2008). Effective disease management depends upon understanding the interactions between the pathogen and other factors that influence ecosystem balance (Crowl *et al.,* 2008). The assessment of environmental disease risk is essential for understanding the epidemic potential of exotic, new, and emerging diseases (Yang, 2006).

Moskowitz and Bunn (1987) noted that "virtually all important decisions involve uncertainty and risk." Management of risk is an ongoing process defined by cultural practice and organisational structure, and should form part of holistic management, as risk does not just result in detrimental effects but also in potential (Standards Australia/Standards New Zealand, 2009).

The process of understanding the nature and perceived level of risk forms the basis of risk analysis. It provides the knowledge for which risk can be evaluated and treated, and as such, forms only one part in the broader process of risk assessment (Standards Australia/Standards New Zealand, 2009). The risk assessment process can be broken down into any number of steps; this includes risk identification, analysis of possible scenarios and their associated impacts, assessment of the probability or likelihood of the scenarios actually happening, and prioritisation of management actions accordingly. Successful risk assessment will lead to the most appropriate and cost effective risk treatment strategies (Standards Australia/Standards New Zealand, 2009).

Yang (2006) provides a diagram of the disease risk assessment process and notes that information is stored in a database, transferred to a GIS (Geographical Information Systems) which is then used for disease prediction (Figure 1.1). Computers have revolutionized the way in which disease is assessed (Yang *et al.,* 1991; Graham *et al.,* 2004) and have allowed us to simulate elements of epidemiology such as rates and extent of spread, epidemic and progress curves, inoculum loads throughout the disease cycle, as well as evaluate the accuracy of simulations (Contreras-Medina *et al.,* 2009).

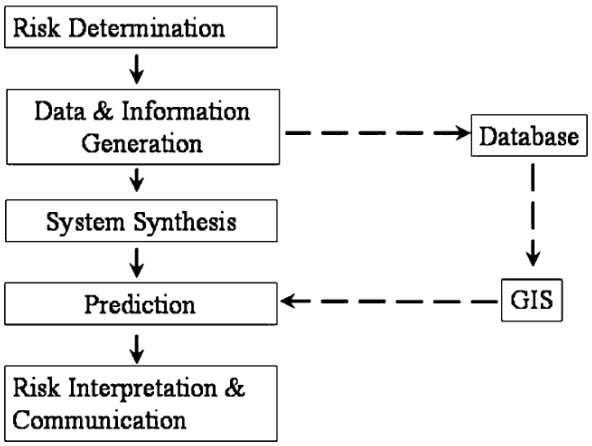


Figure 1.1 The disease risk assessment process (Yang, 2006, pp 29).

GIS incorporates various landscape elements derived from multi-source data, allowing for mapping and correlative analysis between environmental factors and disease (Plantegenest *et al.,* 2007). So called modelling increases our understanding of disease progression in relation to environmental variables (Graham *et al.,* 2004), aids in the characterisation and prediction of the spatial patterns of disease spread (Ristaino and Gumpertz, 2000; Plantegenest *et al.,* 2007), provides insight into the potential impacts of disease in areas it is not known to occur (Jacquez, 2000), allows for spatial-risk prioritisation, and assesses the effectiveness of control measures (Ristaino and Gumpertz, 2000).

The work outlined here is concerned with management of disease risk in natural environments. It does not complete the entire risk management process, but seeks to address the stage of risk assessment. Outlined below is an introduction to the pathogen, *Phytophthora cinnamomi*, the environment in which it is found, how it is detected, and the means by which disease caused by this pathogen can be modelled. Additionally, a description of the natural environment in which disease risk is being addressed, i.e., the Greater Blue Mountains World Heritage Area, is included.

1.3 *Phytophthora cinnamomi*: a cause of disease in natural ecosystems in Australia

1.3.1 Biology of *P. cinnamomi*

As their name suggests, species of *Phytophthora* are 'plant destroyers' causing millions of dollars of damage in natural and production systems around the world annually (Zentmyer, 1980; Erwin and Ribeiro, 1996; Cahill *et al.,* 2008). Described as fungal-like water moulds, of the more than 100 species formally described, most are associated with plant disease (Kroon *et al.,* 2012). Hosts include both agricultural and horticultural crops,

plantation species and native vegetation. Nearly one hundred years has passed since *P. cinnamomi* was first described as the cause of disease in Sumatra in 1922 by R. D. Rands, by which stage its distribution in subtropical areas globally was well underway (Weste and Taylor, 1971; Zentmyer, 1980; Gerrettson-Cornell, 1989).

While still not completely reviewed, the *Phytophthoras* are classified within the Kingdom Straminipile, Phylum Oomycota and the Family Pythiaceae (Ribeiro, 2013). Most species are soil-borne and cause disease of plant root systems; however some species such *as P. infestans, P. palmivora* and *P. ramorum* cause disease on aerial plant parts. The host range of each species may be limited to a few, or, like *P. cinnamomi*, several thousand (Erwin and Ribeiro, 1996; Shearer *et al.,* 2007). Their success as plant pathogens stems from the ability to reproduce asexually, short generation times and a motile reproductive spore (the zoospore) that can actively seek out susceptible hosts (Erwin and Ribeiro, 1996).

Phytophthora cinnamomi can additionally spread via hyphal extension, or root-to-root contact. If environmental conditions are not appropriate, the vegetative phase (hyphae) may be reduced while the pathogen may remain dormant for many years as chlamydospores, that are resistant to desiccation and microbial attack (Mircetich and Zentmyer, 1966; Weste and Vithanage, 1978).

1.3.2 History of *P. cinnamomi* in Australia

Phytophthora cinnamomi has a long history of disease in Australian agricultural and horticultural industries. The earliest report of disease associated with *P. cinnamomi* is suggested to date back to the 1880s when an epidemic of wilt and top rot broke out in Queensland pineapple crops (Simmonds, 1929; Irwin *et al.,* 1995). Disease was reported on *Salix alba* in the 1930s in Werribee outside Melbourne, Victoria, although it was not until much later that *P. cinnamomi* was recognised as the cause (McLennan *et al.,* 1973). Today *P. cinnamomi* remains a significant pathogen and continues to cause extensive losses in the Australian pastoral, ornamental and horticultural industries annually (Cahill, 1993).

Disease associated with *P. cinnamomi* in natural ecosystems has, however, led to a greater awareness of the pathogen, due initially to its impacts within the forestry industry but also because of its impacts on biodiversity and ecosystem function. Disease affecting native vegetation was first recognised in 1948 in New South Wales and reported in 1956 (Fraser, L., 1956 cited in Newhook and Podger, 1972); however by this stage *P. cinnamomi* had been causing disease in Western Australian forests since the 1920s and Victorian forests since the 1930s (Podger and Batini, 1971; Weste and Taylor, 1971; Marks *et al.,* 1972). The need to identify the cause of disease in Western Australian Jarrah forests due to

losses experienced in the forestry industry led Frank Podger to identify *P. cinnamomi* as the causal agent in the 1960s (Podger, 1965 cited in Podger, 1972). Similar symptoms were recognised in the Victorian forestry industry and *P. cinnamomi* was again isolated (Marks *et al.,* 1972). Still today, dieback associated with *P. cinnamomi* remains an issue in plantation industries (Carnegie, 2007) and is suggested to be the cause of 25% of the decline expressed in the genus *Eucalyptus* in Australia (Jurskis, 2005). Disease has continued to impact natural ecosystems in Western Australia and Victoria, and has also been isolated extensively along the New South Wales coast especially in the last decade (McDougall *et al.,* 2003; Daniel *et al.,* 2006; Walsh *et al.,* 2006; Howard, 2008; Suddaby, 2008a). During the 1970s, *P. cinnamomi* was isolated from natural vegetation in South Australia (Lee and Wicks, 1977), Queensland (Pratt *et al.,* 1972; Brown, 1976) and Tasmania (Pratt *et al.,* 1972; Podger and Brown, 1989). Disease caused by *P. cinnamomi* continues to impact natural ecosystems today. It has been recently suggested to be a related cause of two of Australia's ten most vulnerable ecosystems (Laurance *et al.,* 2011) and associated with the demise of nearly 25% of some of Australia's most threatened plants (Australian Network for Plant Conservation, 2012). Management of Phytophthora dieback is expected to cost the Australian economy $1.6 billion over a period of ten years (dieback.org.au, 2013).

1.3.3 Symptoms of disease

A host of a pathogen is any entity that is able to be infected by that pathogen, allowing it to obtain nutrients (Agrios, 2005). This does not mean that the host will actually show symptoms of infection and if it does not, it is considered an asymptomatic host or a carrier (Oxford, 2008). When a susceptible host is infected with *P. cinnamomi*, primary symptoms can develop quickly. Root lesions develop first (Dawson and Weste, 1984) and the suppression of root growth can begin within 10 hours (Marks *et al.,* 1972). Changes in the level of Abscisic Acid at the infection site, influencing the plant-pathogen interaction, occur within 6 hours of infection (Cahill and Ward, 1989; Cahill *et al.,* 1993). Water uptake, glucose metabolism and thus respiration are retarded within 12 hours (Dawson and Weste, 1984), leading to secondary symptoms of leaf necrosis and death within two weeks (Weste and Taylor, 1971). Necrotic tissue may also occur on the collar and stem (Podger and Brown, 1989) and in some species, leaf colour changes, wilt and chlorosis may also occur (Weste and Taylor, 1971; Dawson and Weste, 1984; Podger and Brown, 1989; Newell, 1998; Laidlaw and Wilson, 2003). As *P. cinnamomi* typically attacks the fine feeder roots compromising the hosts ability to absorb water, infected plants often appear wilted (Newhook and Podger, 1972). Over extensive periods of infection, reductions occur in leaf

size (Marks *et al.,* 1972) and overall plant growth (Podger and Brown, 1989). Species that are considered field resistant will often have a reduced root system, but appear to produce roots faster than the pathogen can destroy them (Marks *et al.,* 1972), and as such are considered an asymptomatic host of *P. cinnamomi.* Recent work in Western Australia has shown that asymptomatic hosts remain in infested sites in which the majority of susceptible species have been lost, allowing the pathogen to survive in plant material (Crone *et al.,* 2013a). The presence of haustoria in some of these plants and others tested under controlled conditions, suggest *P. cinnamomi* behaves as a biotroph in some hosts (Crone *et al.,* 2013a; Crone *et al.,* 2013b). Depending on the size, age and susceptibility of the host, death may occur suddenly (Weste and Taylor, 1971; Marks *et al.,* 1972), be protracted over several years (Weste and Marks, 1987), or, in the case of asymptomatic hosts, it may not occur at all.

1.3.4 Aetiology of Phytophthora dieback

Although *P. cinnamomi* can be isolated from an extensive range of environments, disease does not always occur (Erwin and Ribeiro, 1996). This results from environmental suitability for the pathogen and the different responses of plant hosts leading to a variable expression of disease across Australia. In the northern regions, with temperate and tropical conditions, rainfall is more even throughout the year, or, increases coincide with increases in temperature. As such, dieback expression caused by a lack of water in warmer climates does not readily occur. Disease in these areas is typically associated with other confounding factors, resulting in 'patch' death and frequent asymptomatic hosts (McDougall and Summerell, 2003). Additionally, the level of inoculum in the soil is inconsistent (Gadek and Worboys, 2003; Gillieson *et al.,* unpublished), and there appears to be greater resistance amongst the native species (McCredie *et al.,* 1985; Suddaby *et al.,* 2008). In the southern regions, dieback is expressed more broadly, such that there is clear delineation between vegetation through which the pathogen has spread and that which it has not yet reached (Weste and Ruppin, 1977; Cahill *et al.,* 2008). Disease outbreaks are closely controlled by seasonality. The Mediterranean climate of the south with higher rainfall periods in spring and autumn, encourages pathogen spread and infection, while plant stress induced by hot, dry summers triggers dieback and plant death (Weste and Ruppin, 1977; Weste and Marks, 1987).

Dieback typically occurs in heathlands, open forest, woodlands (Weste and Marks, 1987) and in some cases rainforest (Newell, 1998; Gadek and Worboys, 2003) (Figure 1.2). Symptom expression of water stress at the individual plant level leads to holistic expression

of drought stress within the community (Marks *et al.,* 1972; Weste and Marks, 1987). The most susceptible species tend to be found in the understorey and are the species that are lost first (Marks *et al.,* 1972; Newell, 1998). In Victorian woodland communities as much as 50% of the understorey is lost within 6-8 months of infection (Weste, 1974), while in Western Australia, reductions of 30-40% have been reported (McDougall *et al.,* 2005). Overstorey species may survive much longer, except in the circumstance that additional stress, such as drought or water-logging, occurs (Weste and Taylor, 1971; Newhook and Podger, 1972). The loss of overstorey species has detrimental impacts on canopy structure; their loss signals broad-scale changes in community composition and function (Duncan and Keane, 1996), such as a reduction in leaf litter and an increase in the presence of bare ground leading to erosion (Wills, 1993; Brown *et al.,* 2002; McDougall *et al.,* 2002b; McDougall *et al.,* 2005). In Victorian woodlands the loss of susceptible Eucalypts combined with reduced growth rates and species recruitment, decreases biomass production by one third (Kennedy and Weste, 1986). This has implications for nutrient cycling, soil fertility and stability, and plant health. As the structure of the upper parts of an infected ecosystem begins to change, so too does the lower part in which ground covers continue to die due to their susceptibility (Duncan and Keane, 1996; Newell, 1998; McDougall *et al.,* 2002b), or as a secondary result from losing overhead protection (McDougall *et al.,* 2002b; McDougall *et al.,* 2005). Opportunistic grasses, sedges and weeds increase in prevalence often due to a greater resistance to disease (Weste, 1974; Wills, 1992; Newell, 1998). The resulting ecosystem has a vastly changed structure and distribution of species. While the abundance and diversity of some families is reduced (Weste, 1974; Wills, 1992; Newell, 1998; Laidlaw and Wilson, 2003), others will increase (Brown *et al.,* 2002). Recruitment of susceptible species is impeded (McDougall *et al.,* 2002b), and some species are lost from the community entirely (Podger and Brown, 1989). Changes in species composition reflect the pathogens decreasing distribution in the soil (Podger and Brown, 1989) and both collectively reflect the length of time since initial infection (Weste *et al.,* 1973).

The impacts of Phytophthora dieback within natural ecosystems are not limited to those experienced by plants. Faunal communities affected by loss of habitat and food supply include invertebrates, numerous mammals and birds (Wilson *et al.,* 1990; Wills, 1992).

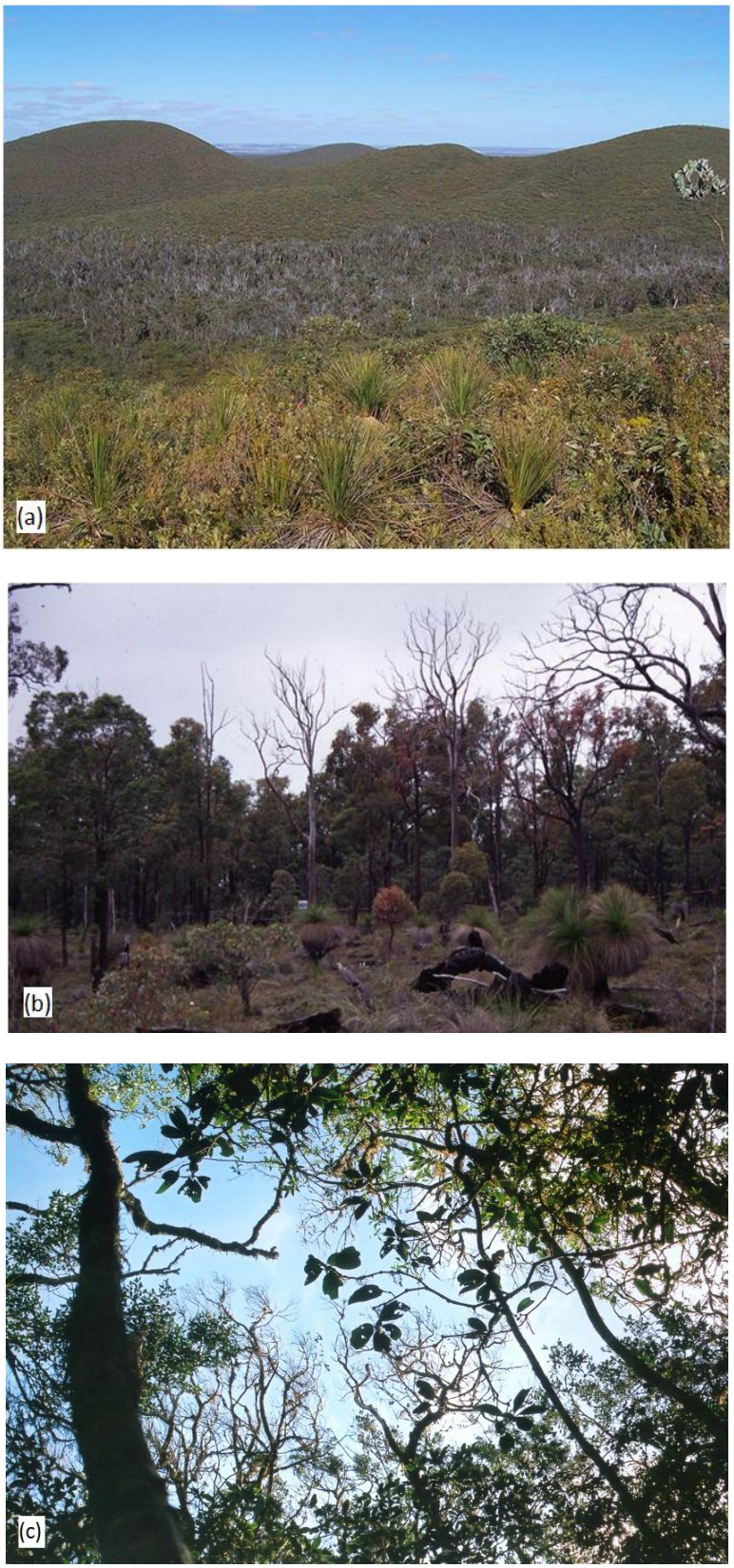


Figure 1.2 Phytophthora dieback in different ecosystems around Australia. (a) Dieback in a heathland of the Stirling Ranges National Park in Western Australia (Gnangarra , 2006) (b) Dieback in a Jarrah Woodland community in Western Australia (E. Hansen, forestpathology.org) and (c) dieback in a Queensland rainforest (Worboys, 2006).

1.3.5 Plant hosts

Phytophthora cinnamomi has an extensive host range, yet its ability to cause disease is variable. This variability is a function of natural genetic resistance, the pathogenicity of the individual isolate and the environment in which disease is occurring (Cahill *et al.,* 2008). As such, the response of individuals of a single host species may be variable within the one population. Reports of the susceptibility of a species are often contradictory, which may result from species being studied in different environments. For example two species of *Banksia, B. integrifolia* and *B. serrata,* have had their susceptibility reported on five separate occasions. Initially both species were described by Weste and Marks (1974) as highly susceptible, with 80% of the natural population at a diseased site in Victoria lost. Again in laboratory experiments they were found to be susceptible (Hinch and Weste, 1979). However this was contradicted two years later when a glasshouse trial conducted in Hawaii led to both species being classified as resistant (Cho, 1981). They were again found to be resistant in 1985, (McCredie *et al.,* 1985), but in 2007 both species were identified as susceptible (Newby, 2007), even more so than the highly susceptible *Pinus radiata* which has been shown to die within eight weeks (Butcher *et al.,* 1984; Ali *et al.,* 1999). The variability amongst these results highlights the difficulty in determining species susceptibility due to their different responses when tested under different environmental conditions. Although it is useful for land managers to have this sort of information, results must be treated cautiously as the relationships established in a glasshouse or laboratory will be complicated by the many interactions occurring in natural environments. Likewise, the unique interactions occurring in one ecosystem will be different to that which occurs in another. Although susceptibility varies at the genus level making predictions about species potential susceptibility unreliable (Shearer *et al.,* 2004), there are a number of genera that appear to have a large proportion of susceptible species. This includes, but is not limited to many individuals in Fabaceae, Epacridaceae, Proteaceae, and Myrtaceae (Newhook and Podger, 1972).

A comprehensive list of native plant hosts found within Australia has been included in the appendices of O'Gara *et al.,* (2005)(McDougall, 2005). From this list and other literature, 130 species that grow in the Greater Blue Mountains World Heritage Area (GBMWHA) have been identified as hosts (Appendix 8.1). Out of this list, at least four species have conservation status. Other species identified as hosts also form part of endangered ecological communities or are depended upon by fauna for their habitat and survival. Invariably, out of the hundreds of species endemic to the GBMWHA, many more will be hosts of *P. cinnamomi*.

1.3.6 Phytophthora dieback – a Key Threatening Process

With increasing reports of disease and ecosystem degradation occurring across Australian national parks and conservation areas, especially in Western Australia, Victoria and Tasmania, the need to tackle Phytophthora dieback from a national approach became evident. As a result, dieback in natural ecosystems caused by *Phytophthora cinnamomi* was classified as a Key Threatening Process under the Commonwealth Environmental Protection and Biodiversity Act, 1999. As part of this process a Threat Abatement Plan was developed which included best practice management guidelines (O'Gara *et al.,* 2005). Within this document, New South Wales (NSW) was identified as a state in which minimal information on the incidence and spread of disease was available. Similarly, in NSW, disease caused by *P. cinnamomi* was also listed as a Key Threatening Process (NSW *Threatened Species Conservation Act, 1995*). Under this legislation, a Statement of Intent was developed by the NSW Department of Climate Change (2008), which highlighted the need to address this information shortage. Expectations of disease and the way in which it is currently managed in NSW, is largely based on the experience of other states, however, as indicated, environmental differences limit the relevance of these studies to NSW.

1.4 The disease environment and its influence on the pathogen's biology

Phytophthora dieback is generally considered a disease of Mediterranean climates favoured by high soil moisture in spring and hot temperatures in summer (Weste and Marks, 1987; Weste, 1994); however the pathogen has been isolated in a range of environmental conditions and habitats around Australia. These include additionally, sub-alpine regions of New South Wales (McDougall *et al.,* 2003), and tropical rainforests of Queensland (Brown, 1976). Although these environments represent a range of climates, the most conducive conditions for the pathogen have been characterised from field studies, laboratory experiments and correlative analysis. The key factors appear to relate to rainfall, temperature and soil characteristics such as texture, pH and microbial activity. The activity of the pathogen, and thus the severity of disease, is closely correlated with environmental conditions. These basic conditions surrounding pathogen proliferation are summarised below (Table 1.1).

Table 1.1 Optimal conditions of *P. cinnamomi* establishment.

Variable	Optima	Reference
Temperature	21-24˚C (*in vitro*), 24-25˚C (in soil)	Byrt and Grant, 1979; Nesbitt *et al.,* 1979
Rainfall	>600 mmpa	O'Gara et al., 2005
Soil pH	6.8	Byrt and Grant, 1979
Soil type	Variety	Podger and Brown, 1989; Gillieson *et al.,* unpublished
Soil texture	Clay	Sterne et al., 1977
Topography	Drains, gullies, depressions, flat ground, north aspect	Podger and Brown, 1989; Shearer and Dillon, 1996b; Laidlaw and Wilson, 2003
Water table	Shallow	Shearer and Dillon, 1996b

Temperature is key to disease because of its influence on *P. cinnamomi* physiology. As listed above, the optimal temperatures for *P. cinnamomi* growth is between 21-24˚C when tested in culture (Byrt and Grant, 1979) and 24-25˚C when tested in soil (Nesbitt *et al.,* 1979). Higher or lower temperatures reduced the rate of growth and sporogenesis (Figure 1.3). The lower and upper limits of *P. cinnamomi* detection from the soil environment are 4˚C and 36˚C, respectively (Macdonald and Duniway, 1978; Nesbitt *et al.,* 1979; Bowers *et al.,* 1990). Development is halted outside of these limits but prolonged exposure results in death (Macdonald and Duniway, 1978; Nesbitt *et al.,* 1979).

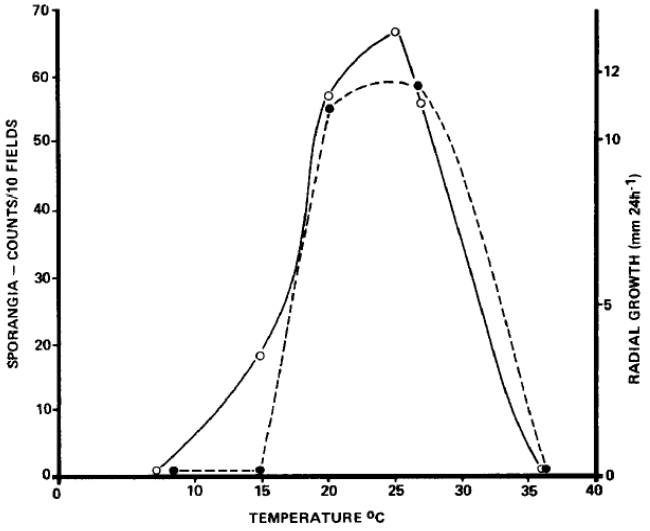


Fig. 3. The effect of temperature on the growth —○— and sporangial production —●— of *P. cinnamomi.*

Figure 1.3 The effect of temperature on the growth and sporangial production of *P. cinnamomi* in soil (Nesbitt *et al*., 1979, pp 139).

The optimal matric potential for the growth of *P. cinnamomi* occurs at field capacity (FC) or just below (0 to -1,000 kPa), and is inhibited completely at -4,000 kPa (Malajczuk and Theodorou, 1979; Nesbitt *et al.,* 1979). Sporangial production is maximised at FC and by half FC it is non-existent (Nesbitt *et al.,* 1979). Interestingly, saturation (twice FC) has been shown to slow sporangial production (Nesbitt *et al.,* 1979; Weste and Vithanage, 1979) likely due to the creation of anerobic condtions. The release of zoospores is more tightly controlled by matric potential such that it is maximised between -100 to -300 kPa, and decreases by -360 kPa (Gisi and Zentmyer, 1980). If matric potential decreases below -1,000 kPa, the pathogen will alternatively produce chlamydospores (Malajczuk and Theodorou, 1979). Cyclic patterns of wetting and drying encourage disease (Weste and Vithanage, 1979; Wilcox and Mircetich, 1979; Bowers *et al.,* 1990), although sustained saturation can suppress the pathogen and halt disease (Bowers *et al.,* 1990).

Soil texture influences disease due to its effect on water potential and aeration favouring either the pathogen and/or the disease. Increased aeration associated with sandy soils enhances zoospore production (Byrt and Grant, 1979); however disease incidence is higher in clay soils and typically worse where drainage is poor (Newhook and Podger, 1972; Sterne *et al.,* 1977; Weste and Marks, 1987).

Phytophthora cinnamomi is better suited to acidic soils with the optimal range extending from pH 5.2 to 6.8; growth is suppressed as soils become more basic. (Byrt and Grant, 1979; Falcon *et al.,* 1984). Continued growth has been recorded in soil as low as pH 3.3; however in such conditions, sporangial production is not evident (Blaker and Macdonald, 1983).

The successful growth and development of *P. cinnamomi* is closely controlled by these environmental conditions and their direct impact on *P. cinnamomi* physiology. The expression of disease however will often occur in conditions that are not ideal for the pathogen. It is during hotter and drier periods that disease is often expressed as plants begin to experience water stress.

The expression of disease is also influenced by interactions with the host, and interactions with other soil microbes that are competitive or antagonistic towards *P. cinnamomi*. Generally considered a poor saprophyte (Erwin and Ribeiro, 1996) and an uncompetitive microorganism, *P. cinnamomi* remains quiescent in biologically fertile soils (Broadbent and Baker, 1974; Meyer and Linderman, 1986). Elevated microbial activity within soil has been associated with hyphal and sporangial lysis, yet *P. cinnamomi* does not grow as well in sterile soil, suggesting the presence of other microbes acts as a stimulant (Weste and Vithanage, 1979). The reduction in growth of *P. cinnamomi* beyond 24˚C is

suggested to result from microbial interactions (Weste and Vithanage, 1979) where microbial hyphal lysis is maximised in soil at 27˚C (Nesbitt *et al.,* 1979). Suppression of *P. cinnamomi* in the soil by other microbes is a key component of disease management in agricultural and horticultural systems. For example, the 'Ashburner System' is used to control disease in avocados by applying appropriate mulches and improving draining, increasing aeration and stimulating the growth of antagonists (Broadley, 1992, cited in Drenth and Guest, 2004). The suppression of disease in environments from which the pathogen is readily isolated can be a direct result of soil microflora (Broadbent and Baker, 1974). In natural environments antagonism occurs in the soil, but possibly also in infected plant roots (Marks and Smith, 1981). Therefore antagonism does not just reduce the incidence of disease by reducing the pathogen population, but also reduces disease directly. The failure of disease expression is additionally linked with good drainage and high soil organic matter (Broadbent and Baker, 1974). Ecosystems with higher and more even rainfall, having more dense vegetation, have higher levels of soil organic matter and greater biological fertility. These ecosystems are likely to have a lower pathogen population and less expression of disease (Weste and Marks, 1987). As seasonality changes, or in the event of unusual weather conditions, each of these relationships can change and in turn alter the pathogen population and the expression of disease.

1.5 Detection of disease

1.5.1 Pathogen and disease detection

Detection of *P. cinnamomi* usually occurs in one of two ways. The first method depends upon the collection of soil or plant material from the area of interest. Soil samples are collected from, or near, vegetation communities that may or may not be expressing disease symptoms. Samples are then returned to a laboratory where the pathogen is isolated in a number of possible ways. Soil is 'baited' with a susceptible host thereby attracting *P. cinnamomi* to the bait tissue if present (Erwin and Ribeiro, 1996). Water is tested in a smiliar manner, except that the bait tissue is place directly into the water, with the pathogen then isolated from the bait (eg, Huberlie *et al.,* 2013). These methods will not always successfully isolate the pathogen, leading to 'false negatives' (Pryce *et al.,* 2002; Davison and Tay, 2005; O'Brien *et al.,* 2009). There is much uncertainity surrounding the relaibability of negative results and just how much soil, water or plant material needs to be tested before an area can be considered pathogen-free (Pryce *et al.,* 2002; Davison and Tay, 2005). Additionally, these processes are lengthy and expensive and require particular

equipment and facilities as well as specific user training in pathogen identification (Drenth *et al.,* 2006; O'Brien *et al.,* 2009).

Molecular techniques may alternatively be applied to extract *Phytophthora* DNA present in the soil sample (e.g. Drenth *et al.,* 2006). Again this approach is expensive, requires equipment and facilities and expertise, and does not distinguish between living and dead organisms, nor pathogenic and non-pathogenic isolates. Such an approach, although not as labour-intensive as soil baiting, will only be appropriate in certain situations with specific management outcomes.

Alternatively, a number of immunodetection assays have been developed to detect *P. cinnamomi* from either a soil-water extract or plant material (Macdonald *et al.,* 1990; Cahill and Hardham, 1994). These can produce instantaneous results with just the test kit and no expertise required by the user to interpret the outcome. The reliability of these tests in terms of their ability to detect different species and create reproducible results is, however, questionable (O'Brien *et al.,* 2009).

Disease identification depends completely upon the expertise of a trained person to 'interpret' Phytophthora dieback based on the presence of disease in 'indicator' species, i.e. a judgement on the margins between diseased and infected areas is made based on the distribution of dieback symptoms in plants that are known to be highly susceptible (CALM, 2001; O'Gara *et al.,* 2005). There are not necessarily any plant or soil samples tested to validate this interpretation. This process of interpretation usually occurs from the ground. There are, however, examples of the application of Phytophthora dieback interpretation from aerial and satellite imagery, especially in areas where *P. cinnamomi* is already known to occur (McDougall *et al.,* 2002a; Bluett *et al.,* 2003; Gadek and Worboys, 2003; Wilson *et al.,* 2012). The obvious risk of interpretation is that an incorrect conclusion may be made, as results are subjective and depend upon the skill of the interpreter. Dieback observed in plants may result from causes other than disease such as natural senescence or drought, or disease, if present, may result from another pathogen. Sufficient training and collection of plant and soil samples can reduce the chance of misinterpretation. Unlike baiting, ground-based interpretation requires no equipment and the process can be quick and easy to complete (Bock *et al.,* 2010).

1.5.2 Remote sensing of plant health

The last 30 years has seen rapid developments in the ability to assess plant health and functionality from remote platforms. Using either aerial techniques or satellite based

platforms and visible or thematic imagery, characterisation of plant health may be completed from the canopy to the global level. Functional processes such as evapotranspiration, photosynthesis, stomatal conductance and respiration may all be quantified using remote sensing techniques (Treitz and Howarth, 1999). Plant productivity indicators such as light use efficiency (Nicols *et al.,* 2000), Leaf Area Index (LAI)(Tucker, 1979), and biomass production (Running and Coughlan, 1988) have all been determined using remote sensing across a variety of spatial scales and vegetation types. Additionally, individual nutrimental and biological components of plants can be quantified, such as chlorophyll content (Gitelson and Merzlyak, 1997), nitrogen (Yoder and Pettigrew-Crosby, 1995) and carbon content (Huber *et al.,* 2008), as well as water and pigment content (Blackburn, 2007; Huber *et al.,* 2008). An understanding of these characteristics combined with the ability to observe physical changes, stress in plants may be identified, and in some cases a cause assigned.

Numerous tools are available for assessing plant health and physiology by way of assessing information acquired remotely. Remotely-sensed assessments themselves may be conducted by observation of changes in spectra or determined from spectral indices (Treitz and Howarth, 1999). Examples include the Normalised Difference Vegetative Index, NDVI (Tucker, 1979), the red edge, RE (Horler *et al.,* 1983), and the LAI (Tucker, 1979), all of which can be utilised to assess canopy conductance, colour and density. Changes in leaf moisture content will effect leaf architecture, resulting in a different spectral reflectance (Jackson and Ezra, 1985). In this way NDVI can be used to assess water stress (Sellers, 1985; Penuelas *et al.,* 1993). Changes in the position of the RE are indicative of physiological or chemical changes in the plant and result from reductions in photosynthesis, loss of chlorophyll (loss of green), as well as the production of anthocyanin and phenolics (increase in reds, purples and blues) (Seager *et al.,* 2005). LAI can be determined by assessing changes in absorbance and reflectance data and gives an indication of plant photosynthesis, transpiration and evapotranspiration (Davishzadeh *et al.,* 2009).

The quantification of vegetation 'states' from remotely sensed information may be completed either directly from the spectral signature, require a transformation or normalisation process, or incorporation with other data sources or modelling formulae (Figure 1.4). One example of direct extraction is the determination of water stress which, when well characterised from the target organism, can be observed directly from remotely sensed data (Jackson and Ezra, 1985). Other indicators are derived from ratios of spectra, which allow for normalisation of data and the removal of atmospheric effects and noise. The most common of these is the NDVI (Tucker, 1979), which expresses absorption of green

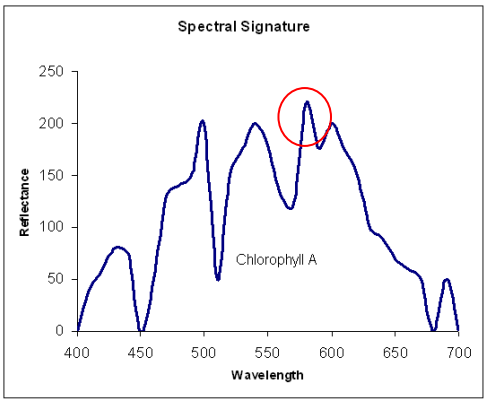
light against that of red light and gives an indication of plant 'greenness' and productivity. Also known as band ratio analysis, these techniques allow for quick characterisation of plant physiology and may be completed at the most rudimentary level of thematic data such as the freely available LandSat imagery.

Other indices are calculated from derivatives of spectra, which highlight otherwise unseen absorption features. For example, quantification of chlorophyll is maximised from the 2nd and 4th derivative functions of original absorption spectra (Butler and Hopkins, 1970; Curren *et al.,* 1991).

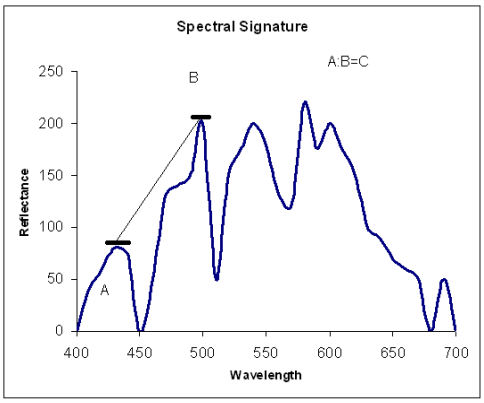
Image spectra may also be combined with additional data sets, such as climatic information, or incorporated into models to generate productivity indicators. For example, plant net primary production can be estimated from remotely sensed data, climatic data and soil data (Coops *et al.,* 1998). In this case the climate data was acquired from global climate models and the soil information gathered from the field. Evapotranspiration rates can also be estimated using remotely sensed data and ground data such that surface temperature, net radiation and soil heat flux are derived from LandSat imagery, while air temperature and air humidity are obtained from ground data (Boegh *et al.,* 2002). Information gathered from remotely sensed assessments of plant health can be used directly in research and management or incorporated into broader ecological studies, such as the effect of land use on plant health, the impacts of climate change on plant productivity or the potential impacts of disease on species distributions.

1.6 Species distributions and disease modelling

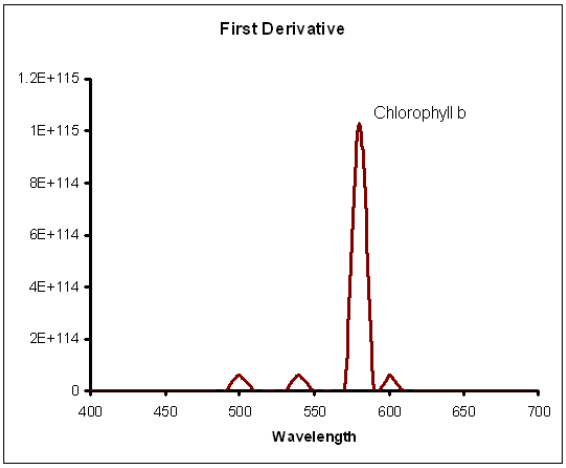
Species distribution models explore how the environment influences the distribution of a species. There are two general types of such models: i) process, mechanistic or niche models and ii) correlative, statistical or empirical models (Jeschke and Strayer, 2008). In the first instance the relationship between the species distribution and the environment is known, in the second it is not, instead, species records are required to determine the relationship (Kearney, 2006; Phillips *et al.,* 2008; Elith and Leathwick, 2009).



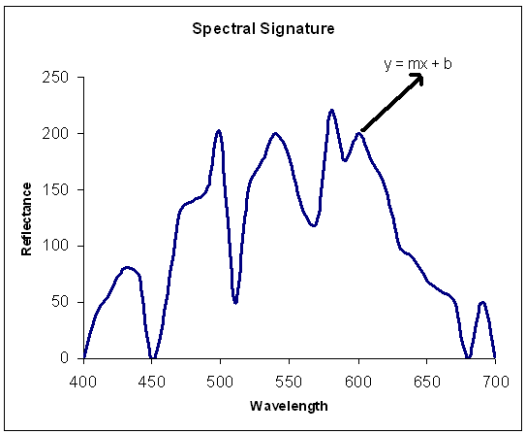
a) Direct

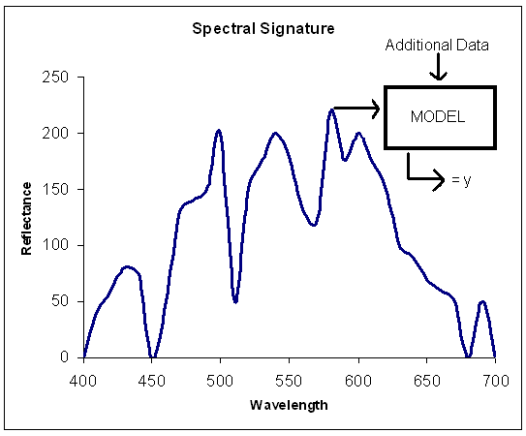


b) Ratio analysis



c) Data transformation





d) Data incorporation, standard formulae, modelling

Figure 1.4 Techniques for the characterisation of plant functionality from reflectance data by a) direct extraction, b) ratio band analysis, c) spectral transformation or d) wavelength incorporation into formulae or modelling.

There are several advantages in a statistically-based approach, first and foremost, the incorporation of actual sample records (Guisan and Zimmermann, 2000; Elith and Leathwick, 2009). Secondly, the model will more effectively identify complex patterns in the landscape that might not be explained in a mechanistic model (Kelly *et al.,* 2007). Thirdly, the model will not over predict into areas uninhabitable due to impacts such as physical boundaries or competition (Kearney, 2006; Phillips *et al.,* 2008). These impacts delineate the realised niche within the fundamental niche (Hutchinson, 1957; Kearney, 2006), a theoretical space containing all habitats suitable for species establishment. In reality, imapcts that suppress establishment are inadvertently incorporated when using sample data (Elith and Leathwick, 2009). This, however, in itself can become a drawback if the species has only been observed occupying part of the realised niche, resulting in underpredictions in the model. Extensive sampling and extrapolation (or interpolation) within the bounds of the variables' range can reduce this underprediction. Fourthly, plasticity often expressed in the phenotype of invasive species can result in a different range expression between the native and invaded area (Jeschke and Strayer, 2008). Such changes would not be detected in a mechanistic model defined by the native environment, but could be corrected for by a statistical model if species records in the new area are available. And finally, a statistical approach allows for better model selection (beyond accounting for autocorrelation), i.e., variable importance can be considered objectively and reconsidered through the process of model construction. The user can identify the model of best fit by removing correlated variables and redundant information, generating the simplest and most accurate model available from the inputs. By modelling the realised niche with the most relevant information, effort lost in the management of areas erroneously overpredicted by the model can be avoided (Vaclavik *et al.,* 2010).

Mechanistic models differ in that they use what is known of the species ecology to model their potential distribution in a new space. Such rule-based models are not prerequisite to current distribution, are unaffected should the phenomena of interest not have reached its full range (potential niche), are easily transferable and can be dynamically adjusted as our understanding of species ecology changes (Guisan and Zimmermann, 2000; Guisan and Thuiller, 2005; Barry and Elith, 2006).

This ability to predict the distribution of an organism is especially useful when dealing with introduced species for which minimal information on the current distribution is known or exotic species that have not been introduced but are potentially suited to an area (Elith and Leathwick, 2009). Recently a global risk model of *P. ramorum* establishement was developed and identified many countries in which *P. ramorum*

currently does not exsist (Ireland *et al.,* 2013). Australia is one such country however, it is believed that many of our native plants may be susceptible (Ireland *et al.,* 2012) and therefore, by having an understanding of which parts of the landscape are most vulnerable, land managers can implement preventative measures as necessary. Similar risk models for *P. cinnamomi* have been established in few cases around Australia and in fact the world. Perhaps the earliest example of a *Phytophthora* risk model is of a rule-based model, in which the distribution of *P. cinnamomi* was predicted in southern Europe (Brasier and Scott, 1994; Brasier, 1996). Survival limits were gathered from literature and were used to construct a CLIMEX model, the results of which were anecdotally compared back to the occurrence of disease in the Mediterranean climates of Europe and Australia.

Once model construction is complete, the outputs can then be used by land managers and researchers to work more effectively within the landscape. For researchers, not only does it allow the identification of the exact habitat constructs that are most suitable to the species of interest, but also identifies the parts of the landscape that need more attentive investigations. As with researchers, land managers have the benefit of seeing which parts of the landscape need more attention, for example which areas control measure must be applied, areas where preventive measures may still be applicable, which areas need additional monitoring and how effective any current management strategies may have already been. Of course models are still just estimations of reality and carry an innate level of error; caution must be taken when considering their outputs both in terms of accuracy and precision, and what we actually know to be true. However, they can aid decisions on the order and priority research and management is to be conducted. In short, modelling species distributions allows for educated and constructive spatial risk prioritisation.

1.7 The Greater Blue Mountains World Heritage Area (GBMWHA)

Situated 60 km from Sydney, the GBMWHA is comprised of eight adjoining reserves spanning approximately 1.2 million hectares, making it the largest protected area in New South Wales (Figure 1.5). It was designated a World Heritage Area in November 2000 as an area of i) *in-situ* conservation, and ii) ongoing biological process. The GBMWHA presents on the international stage as a centre of geological and ecological diversity and a place of natural beauty with thousands of years of cultural associations.

The geology of the GBMWHA has formed over millennia, developing from metamorphic processes, the retraction of an inland sea, followed by thousands of years of weathering. The resulting landscape contains a mix of sandstone and shale, cliffs, gorges, plateaux and pagodas generating a myriad of microclimates and environmental gradients that tightly mosaic the wilderness landscape (Pickard and Jacobs, 1984; Keith and Benson, 1988; Keith and Myerscough, 1993; NPWS and Environment Australia, 1998).

The geodiversity of the GBWMHA landscape supports endemism and diversity that is amongst the highest in the world (Keith and Benson, 1988; James, 1994; Department of Environment and Climate Change, 2009). Vegetation communities include rainforests, eucalyptus tall open forests, open forests, woodlands, heathlands and sedge swamps (Keith and Benson, 1988). The GBMWHA supports approximately 1% of the worlds vascular plants in 152 families and 474 genera. Nearly 50% of the 114 endemic plant species are endangered or rare, including the iconic Wollemi pine (*Wollemi nobilis*) and the Blue Mountains pine (*Pherosphaera fitzgeraldii*), both of which occur in only a few sites within the GBMWHA (Smith, 1981; Jones *et al.,* 1995; Hill, 1997). The region is also well known for its diversity and abundance of Eucalypt vegetation, of which there are over 100 species creating one of three major scleromorphic ecosystems in the world (NPWS and Environment Australia, 1998).

Diversity and endemism is not only reflected in the floral communities but also the faunal with one third of all Australian bird species (265) inhabiting the area as well as 400 vertebrate taxa, 63 reptiles, 30 frog species and an estimate population of 4000 moth and butterfly species (Lepidoptera)(NPWS and Environment Australia, 1998). Amongst these inhabitants are approximately 120 rare and threatened species.

This vast array of plant and animal species set against a diverse landscape leads to a 'natural beauty' that hold strong cultural associations. For at least 14,000 years, Aboriginals in three language groups have inhabited the GBMWHA and created nearly 15,000 cultural sites including many paintings and engravings. This encapsulation of the landscape combined with that of European culture is responsible for the renowned understanding of the beauty of the GBMWHA. Cultural associations within the landscape have led to over 80 years of conservation values established within the community, fostering recreation, inspiration, and science (National Parks and Wildlife Service and Environment Australia, 1998).

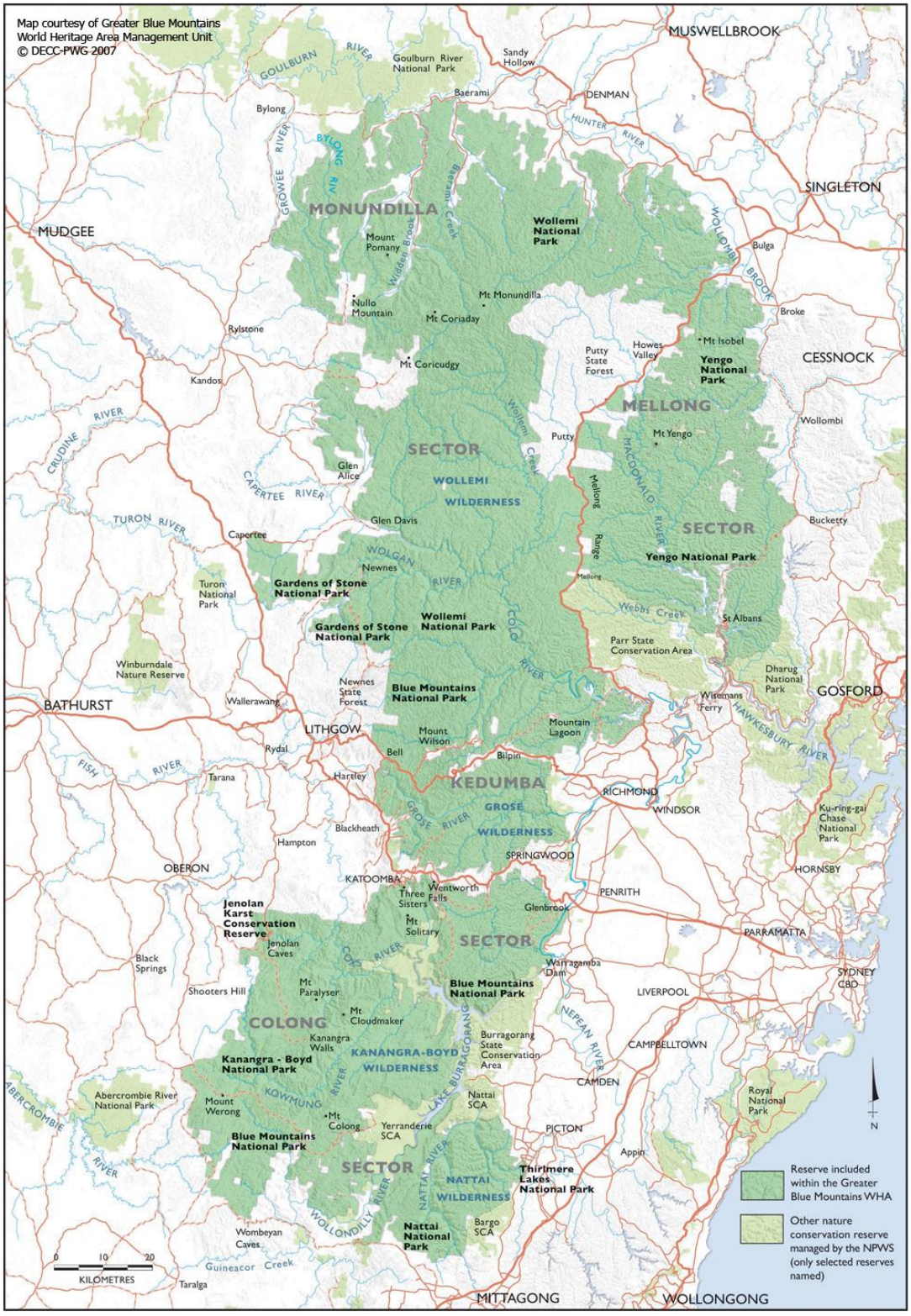


Figure 1.5 Greater Blue Mountains World Heritage Area and adjoining reserves. (Department of Environment, Climate Change and Water, 2009).

1.8 Quantification of Phytophthora dieback risk in the GBMWHA

Dieback within the GBMWHA has not been formally documented, but there is evidence of its occurrence (e.g. McQueen, unpublished, BMADWG, 2010). Two suggested causes of dieback include Bell minor dieback and Phytophthora dieback, both of which are Key Threatening Processes. In neither case has the extent of dieback within the GBMWHA been quantified. Areas of dieback are found throughout the GBMWHA, and may result from a single or combination of causes such as water logging, natural death, climatic stress, (e.g. drought), competition (succession and allelopathy), pollution, fire, nutrient stress, insect pests, and native or exotic microorganisms (Sterne *et al.,* 1977; Jurskis, 2005; Carnegie, 2007).

The climatic and environmental conditions of the GBMWHA are often optimal for the growth of *P. cinnamomi* both in terms of temperature and rainfall. Given that the area is popular for recreational activities that are associated with the dissemination of *Phytophthora*, the GBMHWA is also predisposed to the spread of disease. There is ample evidence of the susceptibility of species inhabiting the GBMWHA based on research published from other regions of Australia (Appendix 8.1), indicating that both the hosts and the environment required for disease are present. All that would remain for disease to occur would be the establishment of *P. cinnamomi* itself.

In 2008 the Hawkesbury Nepean Catchment Management Authority (HNCMA) in association with The Royal Botanic Gardens and Domain Trust, completed a survey of the occurrence of *Phytophthora* throughout the HNCMA area (Suddaby, 2008a, Figure 1.6). Much of the HNCMA area covers parklands of the GBMWHA (Figure 1.7) and hence has given a preliminary understanding of the distribution of *Phytophthora*. Isolations were made right across the HNCMA area, with approximately 1 in 2 samples positive indicating that *Phytophthora* is widespread but not ubiquitous. These results indicate that there should be ample opportunity to suppress further anthropogenic spread if appropriate management actions are taken. However, consideration must still be given to the reliability of negative results and the possibility that *P. cinnamomi* does occur in areas where sampling failed to detect the pathogen. With this in mind, the first steps in the management process must include an assessment of the risk of disease and an understanding of the pathogens distribution across the extent of the GBMWHA.

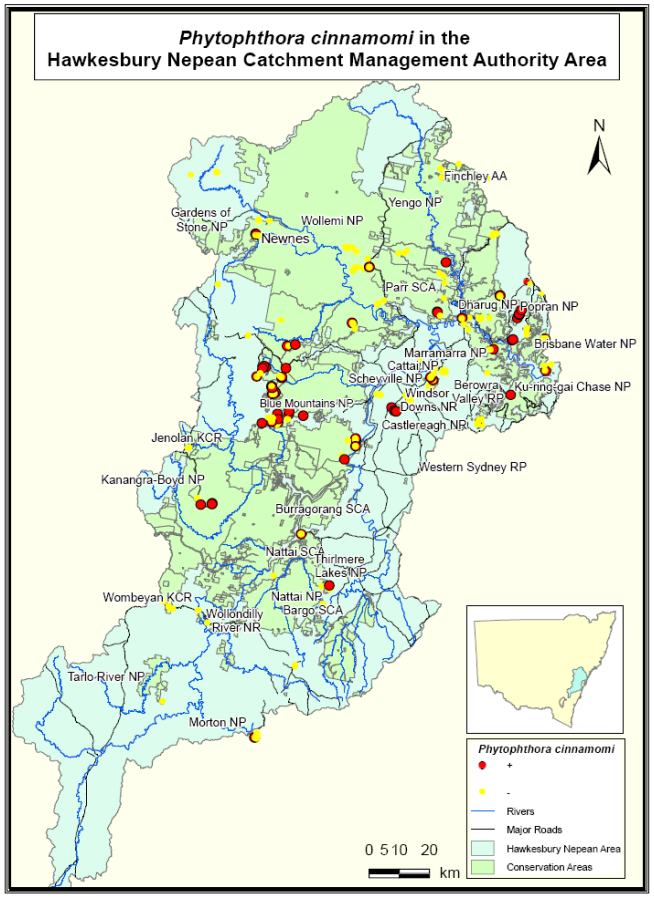


Figure 1.6 Distribution of *P. cinnamomi* in the Hawkesbury Nepean Catchment (Suddaby, 2008a).

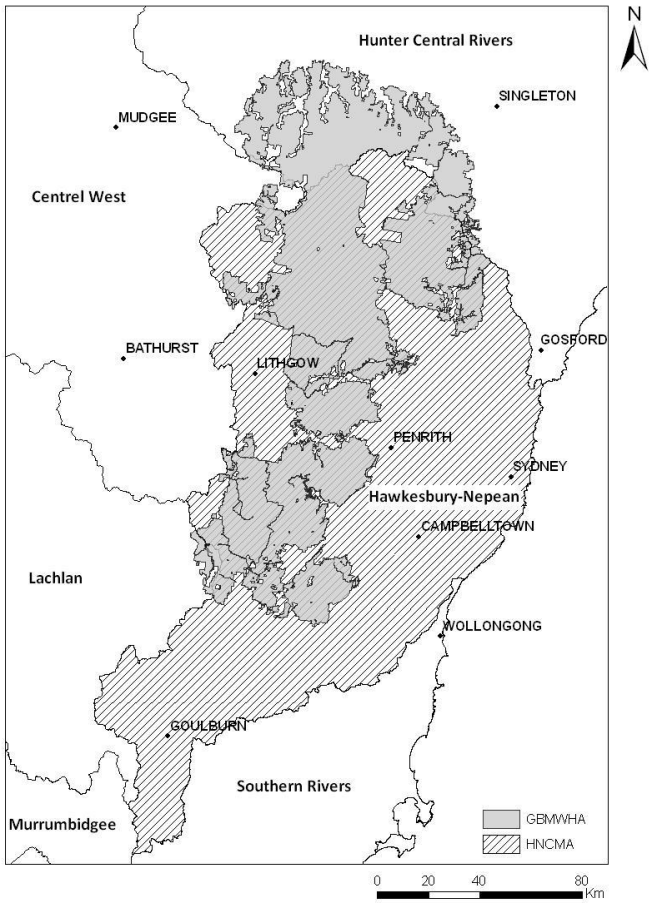


Figure 1.7 Location of the GBMWHA and the HNCMA. Surrounding catchments and selected locations have been shown.

1.9 Aims of this research

The work outlined here seeks to identify which parts of the GBMWHA are at risk of Phytophthora dieback. Chapter 2 addresses the identification of which parts of the landscape *P. cinnamomi* is most likely to be found. As the distribution of *P. cinnamomi* was unknown for the extent of the GBMWHA at the time of model construction, a mechanistic model was developed. Following from this model, a field sampling campaign addressed the immediate shortage of information on the distribution *P. cinnamomi* (Chapter 3). This allowed for the assessment of model performance in Chapter 2 as well as the characterisation of environmental variables associated with the pathogens distribution. The sampling results were then used to build a statistical model of *P. cinnamomis* distribution but added to this was a host component, thus producing a model of disease risk (Chapter 4). And finally, in an effort to identify a safer, more cost-efficient and effective method of *P. cinnamomi* detection, changes in hyperspectral leaf reflectance associated with infection were assessed in a glasshouse trial (Chapter 5). The outcomes of this research have significant implication for the way in which *P. cinnamomi* is detected and managed both in the GBMWHA and natural ecosystems around Australia.

Chapter 2 An expert-driven risk model of *Phytophthora cinnamomi* for the Greater Blue Mountains World Heritage Area

2.1 Introduction

The dearth of information on the distribution of *Phytophthora cinnamomi* in NSW makes it challenging for land managers responsible for disease mitigation. Nevertheless, land managers are responsible for quantifying the impact of disease caused by *P. cinnamomi* (Phytophthora dieback), treating infested locations and reducing the opportunity for further infection. Best practice management of Phytophthora dieback includes, foremost, the prevention of disease spread, a task necessitating an understanding of the most conducive parts of the landscape, i.e. the areas at highest risk of infection.

As an exotic pathogen probably introduced into Australia around the 1880s (Simmonds, 1929; Newhook and Podger, 1972) it is obvious by the extensive distribution of *P. cinnamomi* that it is capable of spreading under poor hygiene and quarantine practice as well as failure to control vectors which move it unimaginable lengths. Recognition of disease spread is hampered by the pathogens patchy distribution and inconspicuous behaviour in NSW vegetation communities (Pratt *et al.,* 1973; McDougall and Summerell, 2003), where infected but asymptomatic hosts may live indefinitely, with the pathogen undetected.

There are a small number of studies on the distribution of *P. cinnamomi* in NSW that have been initiated by the occurrence of symptomatic vegetation. The earliest example is of work conducted by Dr Lillian Fraser of the NSW Department of Primary Industries in the 1950s. She found *P. cinnamomi* to be the cause of disease in native plants in the Royal National Park (Fraser, L, 1956 cited in Podger and Ashton, 1970 and Pratt *et al.,* 1972). Many years past before *P. cinnamomi* was identified again causing disease in bushland on Black Mountain outside Canberra (Pratt *et al.,* 1972). Other studies have reported dieback widely distributed across NSW (Howard, 2008), but specifically at Mt Imlay (McDougall and Summerell, 2003), the Far South Coast State Forest (David Guest, Pers. comm.), south-eastern NSW (Pratt *et al.,* 1973), Barrington Tops National Park (McDougall *et al.,* 2003; Howard, 2008), the Gondwana Rainforest World Heritage Area (Anon., 2012), Sydney Harbour National Park (Daniel *et al.,* 2006; Howard, 2008) and parts of the Sydney and Hawkesbury Nepean Catchments (Suddaby, 2008a). Some of the latter coincide with

transects of the GBMWHA, an area that has likely been suffering the effects of Phytophthora dieback for many years, given its long history of European activity dating back to the early 1800s.

The 2008 study conducted by the The Royal Botanic Gardens and Domain Trust (Suddaby, 2008a) strategically sampled along major walking tracks and tourists areas, and gave the first indication of the distribution of *P. cinnamomi* in the GBMWHA. Nothing further was to become of the results until 2010 when a Commonwealth Caring for our Country grant was acquired by the NSW National Parks and Wildlife Service (NPWS) to address the management of *Phytophthora* in the GBMWHA. The NPWS project, although separate, has been conducted in conjunction with the work outlined here.

The first priority of this project was to assess disease risk as per best practice management (O'Gara *et al.,* 2005). As with any disease survey, some kind of logical sampling strategy was required to avoid haphazard sampling and bias, as well as ensuring full geographic coverage, while minimising cost, time, and risk (Guisan and Thuiller, 2005). A model of the potential distribution of *P. cinnamomi* was constructed which was followed with a field survey (Chapter 3) allowing for the assessment of model performance. As there was limited information on the pathogens distribution, an expert-driven (mechanistic) model was required to facilitate the sampling and risk assessment process.

The Distance Weighted Overlay technique (DWO) is one example of a expert-driven rule-based modelling approach that requires no prior information on the species distribution. The DWO approach is used commonly in investigation of phenomena across the geographical landscape, for example, land use suitability (Fleischer *et al.,* 1998; Panagopoulos *et al.,* 2006; Ahmadi *et al.,* 2010; Erden and Coskun, 2010), environmental degradation (Kitsiou and Karydis, 2000; Xu *et al.,* 2001), or disaster mitigation including flood prediction (Chau and Yang, 1992), landslide potential (Yalcin and Bulut, 2007; Gemitzi *et al.,* 2011), pyroclastic flow risk (Alberico *et al.,* 2008), disease predictions (Kolivras, 2006; Fleming *et al.,* 2007), or fire severity (Atkinson *et al.,* 2010). All that is required to construct a DWO is an understanding of the way the subject behaves under different environmental conditions, geographic layers of the area of interest that relate to this behaviour, and a means by which the layers can be modified and combined, namely a geographical information system (GIS) (Guisan and Zimmermann, 2000). The output is a geographic image highlighting parts of the landscape most to least applicable to the subject. As our understanding of the subject changes, or as the environment itself changes, so too can the model change.

Since the 2000s much *Phytophthora* modelling work has been concerned with *P. ramorum* in the USA and Europe. In the early days of *P. ramorum* research, expert-driven models were utilised as little information on the pathogen's distribution was available to provided managers with the information they required to build statistically-based models. Meentemeyer *et al.* (2004) provided the first example of an expert-driven model applied to the distribution of *P. ramorum* in California. Information on host susceptibility, pathogen reproduction and transmission, as well as key climatic layers were used to construct a DWO. Areas of high infection risk that had not been sampled for disease were identified in the northern portion of California, as well as localities of moderate risk infection 150km to the nearest known outbreak. Venette and Cohen (2006) produced a similar model using climatic suitability as an indication of disease risk for the whole of the USA. Their final model used only information on temperature and soil moisture to predict risk. Finally, Kelly *et al.* (2007), compared five different techniques for the development of a *P. ramorum* risk model for the USA including an expert-driven model and four statistical models. Each of the five models tested by Kelly *et al.* (2007) performed well and produced similar responses; however, the model constructed using the Support Vector Machine performed the best with 96% prediction accuracy. The authors concluded that the expert-driven approach was too simple to explain the complexity of the interaction between *P. ramorum* and the environment, leading to its poorer performance. Clearly modelling benefits from information on where the species occurs in the area of interest, which allows for the construction of statistically-based models. More recently, Keith *et al.* (2012) used knowledge of the landscape and distribution of *P. cinnamomi* to construct a risk model for the Royal National Park. Instead of considering just the ecology of *P. cinnamomi* to develop their model, the authors used information pertinent to the area such as the composition of vegetation groups and soil type. Once sampling results are available, this type of approach is applicable.

This chapter outlines the construction of a model which produces an image of the areas most suitable to *P. cinnamomi* across the GBWMHA, in other words an image of *P. cinnamomi* risk. Using the expert-driven DWO technique, the model is constructed with a series of geographic and climatic variables that reflect both abiotic and biotic factors known to influence the establishment and spread of *P. cinnamomi*. The results show the areas that are at risk of infection by *P. cinnamomi* based on their environmental suitability and their proximity to current infestations. The outcomes have subsequently been used to devise a logical and comprehensive field survey which is outlined in Chapter 3. The results from the

survey have been used to retrospectively assess model accuracy and model limitations allowing for the discussion of model adjustments and improvements.

2.2 Methods

A GIS approach was used to model the interaction between *P. cinnamomi* and the environment within the GBMWHA including a background zone encompassing all areas 100 km beyond the reserve boundary (Figure 2.1). Selecting an appropriate background is important because models developed with a background area to small tend to over predict incorrectly, while models developed with a background to big loose spatial detail, tend to highlight the importance of a select number of variables and inflate the test statistic(s) (VanDerWal *et al.,* 2009). After studying the effect of background size on 12 Australian species, VanDerWal *et al.* (2009) identified that a buffer of 200km was ideal. Their assessment of buffer suitability was based on expert opinion of the known species distribution, which was not possible here. As such, 100km was deemed a suitable buffer as it reduces the likelihood of 'edge effects' without extrapolating unnecessarily into areas where the pathogen would clearly not exist such as the drier inland areas of NSW and the Pacific Ocean. Inputs governing survival and influencing pathogen spread were both biotic and abiotic and can generally be considered to reflect local geography, climate and anthropogenic activity within the GBMWHA. The distribution information from the Royal Botanic Gardens survey (Suddaby, 2008a) was included in the analysis.

2.2.1 Model inputs

2.2.1.1 Geographic layers

The geographical variables used in the model were slope, wetness and Euclidian distance of water bodies. The slope of each cell was calculated as a ratio of the rise over run which in turn were derived from a 25 m digital elevation model (DEM). Wetness or the anticipated dampness of a cell was determined for each using the DEM and the flow direction function in ArcMap 9.3 (Economic and Social Research Institute, California). This function identifies which direction a cell will flow by identifying its steepest down slope neighbour. The Euclidian distance function in ArcMap was also used to identify the shortest (straight line) distance between water bodies and the boundary of the study area. The DEM and water bodies layer were both acquired from the Office of Environment and Heritage, NSW Government (OEH; OEH Data Broker, data.broker@environment.nsw.gov.au), with the DEM being constructed from historical topographic map data using 10m or 20m contours.

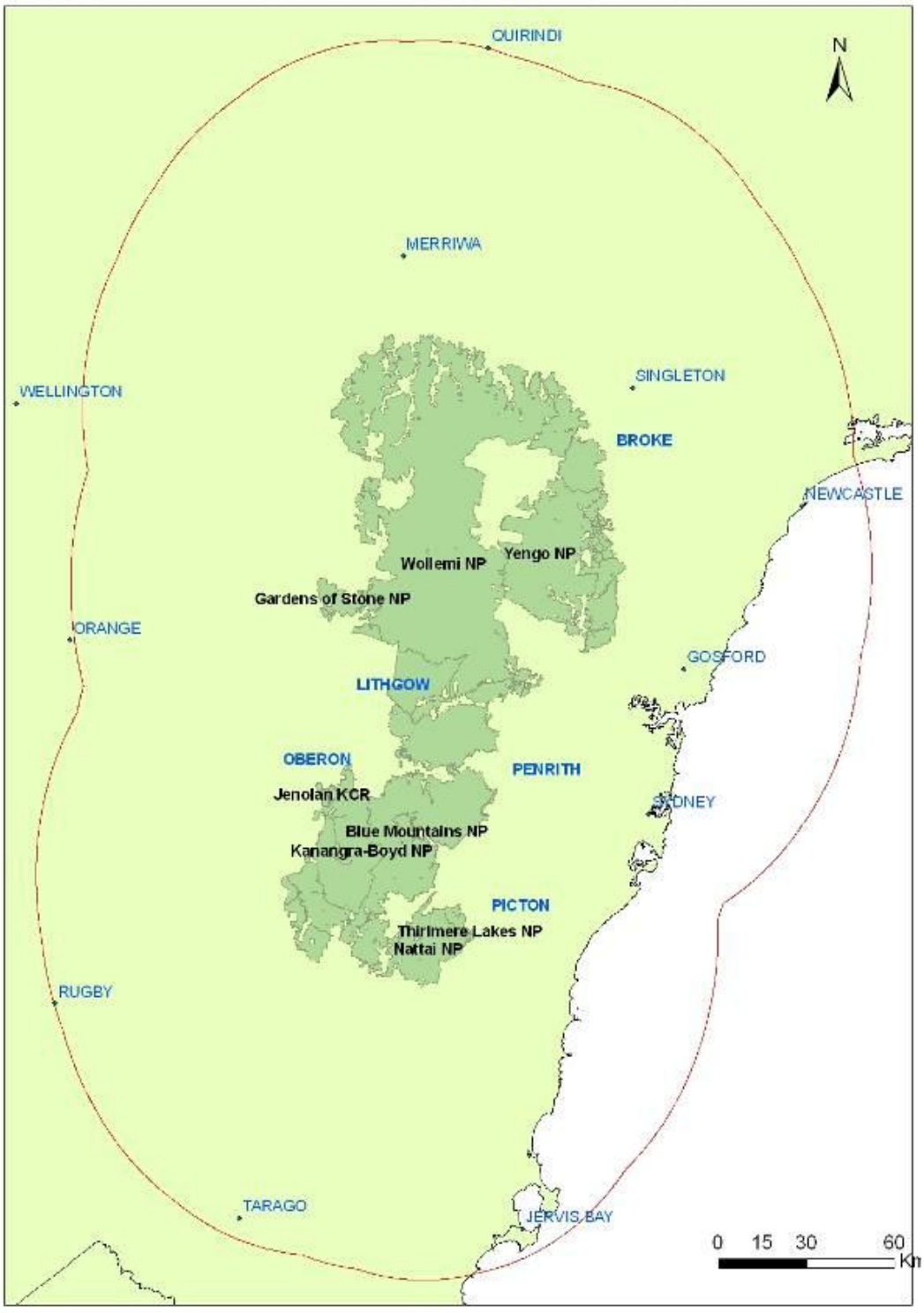


Figure 2.1 GBMWHA 100 km buffer. The buffer is indicated by the red line surrounding the GBMWHA (darker green) and was calculated using the ArcMap buffer function. Localities (blue) proximal to the buffer and the GBMWHA are indicated.

2.2.1.2 Climatic Layers

Phytophthora cinnamomi is best suited to a minimum of 600 mm rainfall per annum (mmpa); however, it can survive at 400 mmpa (O' Gara *et al.,* 2005). The growth of *P. cinnamomi* has been closely assessed under laboratory conditions in which it was shown to be ideally suited to 21-24˚C (Byrt and Grant, 1979), and will not grow below 4˚C or above 35˚C (Marks *et al.,* 1975; Phillips and Weste, 1985). Climatic variables of annual rainfall, maximum temperature of the warmest period (maximum temperature of the warmest week) and minimum temperature of the coldest period (minimum temperature of the coldest week) were calculated for the current study in the BIOCLIM package version 5.2 (Hutchinson, 2004) using the 25 m DEM. BIOCLIM, version 5.2 uses information from metrological stations to interpolate climate data from 1961-1990, and produce continuous climatic surfaces for any area of interest.

The distribution of *P. cinnamomi* is likely to be closely controlled by micro-climatic variations in the landscape which cannot be effectively assessed using a 25m, macroclimatic resolution. However, microclimatic data is not available for the entire GBMWHA, and therefore BIOCLIM layers have been used as acceptable alternative as is seen in modelling of plants and animals around the world.

2.2.1.3 Anthropogenic layers

The spread of *P. cinnamomi* as a result of human activities across Australia is well documented (O'Gara *et al.,* 2005). To incorporate this spread, a series of layers were used to reflect the factors which are most likely to spread disease, namely transport corridors and the presence of people. Layers for built-up areas and road networks were acquired from the OEH and partitioned into walking track, unsealed and sealed roads. These layers will be collectively referred to as roads hereon.

2.2.2 Model development

All feature class layers were converted to rasters using the Spatial Analyst toolkit in ArcMap. Rasterised layers were converted where necessary to GDA94, Map Grid Zone 56 in ArcMap using the AGD_1996_To_GDA_1994_11_NTv2 transformation. Layers were clipped to a rectangular area equal to the greatest distance of a 100 km buffer around the GBMWHA, then transformed to ASCII format and imported into Idrisi Kilimanjaro (Clark Labs, Massachusetts, USA).

The distance function in Idrisi was applied to roads, water bodies and disease localities (taken from Suddaby *et al.,* 2008a; Figure 1.6), such that the distance increased from the origin. Distance functions were created to reflect decreasing probability of spread away from roads and built-up areas, and decreasing soil moisture which was constructed using the water bodies layer. The use of a distance function for the roads and builtup areas are inclined to carry inherent bias, however, they have been included to reflect the spread of *P. cinnamomi* associated within anthropogenic activity. The Fuzzy function in Idrisi was then applied to all layers to rescale each into degrees of appropriateness based on the optimal conditions required for *P. cinnamomi* growth. Membership values between 0 (non-member) and 1 (full membership) were assigned to each cell within the layer. After each of the layers had been rescaled, the final stage of model development was to weigh and combine the layers. As per Meentemeyer *et al.* (2004), the model can be summarised as the sum of the weighted layers divided by the number of weights.

The assignment of variable weights (Table 2.1) was made within the context of the literature; weights were set to reflect the relative importance of environmental variables required for *P. cinnamomi* establish in Australia and other parts of the world. The highest weight was given to that of the current known distribution, as an initial point of introduction is required for spread, hence uninfested areas adjacent to infested areas present the greatest risk. Following this, climatic variables that influence the survival of *P. cinnamomi* as well as the highest means of spread, that is, the unsealed road network and walking paths were given the second level weight. These layers dictate whether or not *P. cinnamomi* will survive if it reaches a new location and the easiest and most likely means by which it will spread. The third level weight was related to less important or slower methods of spread (sealed roads and slope), and proliferation of *P. cinnamomi* in an environment (wetness). In this way, the expert approach of model development accounts for both natural and human-mediated spread, but places more emphasis on the latter.

Table 2.1 Fuzzy function description and weighting of utilised layers.

Layer	Fuzzy Function and Shape [A]	Membership Limits [B]	Relative Weight
Built up areas	Exclusion	1 = 1, 0 = 0	-1
Water bodies	Exclusion		-1
Water bodies, distance	S, MD	1 = 0 m; 0 = max distance	0.058
Tracks	S, MD		0.118
Roads	S, MD		0.058
Unsealed roads	S, MD		0.118
Previous detections	S, MD		0.176
Slope		1 = max slope; 0 = min slope	0.058
Wetness	S, MI	1 = max wetness; 0 = min wetness	0.058
Annual rainfall	S, MI	0 = 0mm; 1 ≥ 600 mm	0.118
Temp max warmest period	S, Sy	0 = 0 and maximum temperature, 1= 21-24˚C	0.118
Temp min coolest period	S, Sy		0.118

[A] shape of the fuzzy curve applied: S, sigmoidial; MD, monotonically decreasing; MI, monotonically increasing; Sy, symmetric. [B] 1= full member, 0 = non-member.

Boolean maps (with values of 0 or 1 for absence and presence respectively) of built-up areas and water bodies were generated before exporting all layers back to ArcMap for the final assemblage. The Raster Calculator (ArcMap) was used to add each risk surface together, and the Boolean maps were then subtracted to exclude areas were *P. cinnamomi* would not occur within the GBMWHA. The final map, on a scale of 0-1, gave predicted probabilities of establishment risk to each cell within the context of all surrounding cells in the rectangular area for which modelling was completed.

2.2.3 Model testing

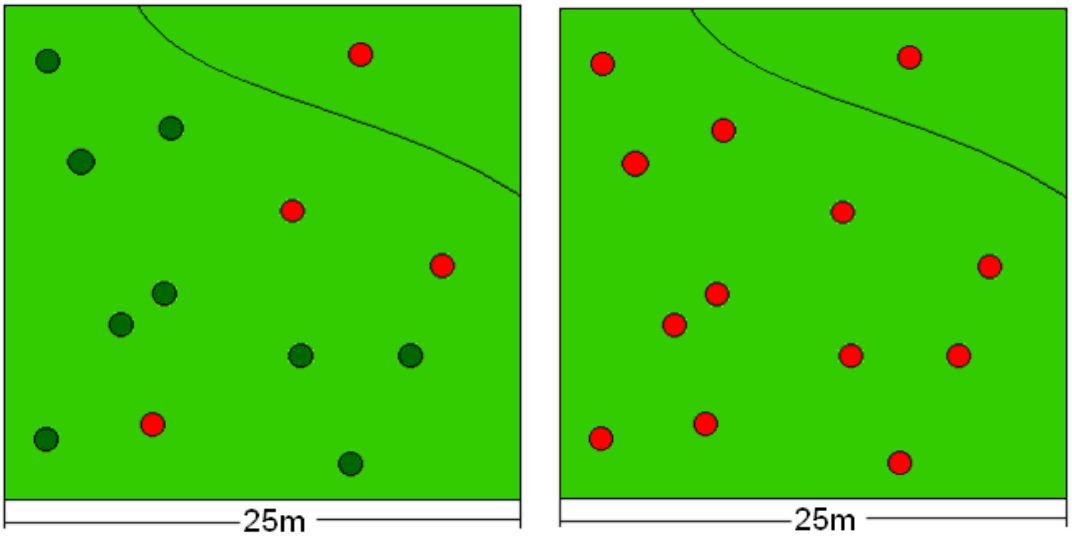
The model was subsequently used to construct a sampling strategy, the details of which are outlined in Chapter 3. From the final risk map, the sampling area was defined as all land within 500 m of roads, tracks and paths. The range of risk values remaining in the sampling area was divided into five strata with increasing risk (ie, stratum 1 = lowest risk, to stratum 5 = highest risk). The area remaining in each stratum was used to calculate the minimum number of randomly allocated samples required to achieve 95% confidence and 5% error in the sampling results (Krejcie and Morgan, 1970). By utilising a stratified random sample, full coverage of environmental variables could be gained providing the most useful

result for subsequent construction of statistical models (Guisan and Zimmermann, 2000). A Chi-squared analysis was used to assess model accuracy by way of determining observed and expected numbers of positive results across the range of predicted probabilities within the GBMWHA. The lower probability cut-off of each stratum was used as the expected value as any result with a predicted probability greater than or equal to that value would be included in that stratum.

The Receiver Operator Curve (ROC) was constructed in R (R Development Core Team., 2010) using the ROCR package (Sing *et al.,* 2005). The Area Under the ROC Curve (AUC) was determined to assess whether the predicted risk reflected the rate of positive and negative detections and if the rate of prediction was better than random. The binomial ROC test was performed twice, once with all other *Phytophthora* species classified as negative and once with them classified as positive. This assessed the models ability to predict the distribution of *P. cinnamomi* in the former case and *Phytophthora* in the latter.

2.2.4 Data manipulations and adjustments

Several attempts were made to improve model accuracy by way of data and design adjustments. Firstly, because the number of positive detections was highest in the fourth stratum, it was combined with the fifth stratum in an attempt to boost the rate of detection in the most suitable class. This was done to force the highest rate of detection in the highest risk stratum. Secondly, a decreasing weight was applied to the number of negatives in each stratum such that negative samples in the first stratum (lowest risk) were multiplied by 0.9, the second by 0.8 and so on up to the highest stratum which had a multiplier of 0.5, effectively halving the number of negative samples. As the suitability of land increased, it was argued that the chances of false negatives also increased. The third adjustment made was to the positive detections. Sample results were inspected within the GIS and wherever negative results were recorded alongside positive results in any single 25 m cell, the negative results were reclassified to positive (Figure 2.2) as *P. cinnamomi* often has a patchy distribution and can be difficult to isolate from the soil (Podger and Ashton, 1970; Weste and Taylor, 1971; Weste and Kennedy, 1997; Brown *et al.,* 2002; Pryce *et al.,* 2002; McDougall *et al.,* 2003). Fourthly, The bin ranges were also adjusted so that each stratum had the same number of samples. In each case, Chi-squared analysis was re-run to assess for model improvements.



a) b)

Figure 2.2 Data adjustments for spatial proximity. a) A theoretical example of the original sampling results of 15 samples taken in one 25 m x 25 m cell showing negative samples in green and positive samples in red, and b) showing the same cell after reclassification of the negative samples due to their proximity to positive samples.

2.3 Results

2.3.1 Suitability distribution

The final model of the risk of *P. cinnamomi* infection within the GBMHWA is in Figure 2.3. The model predicted an intricate pattern of suitable locations following the distribution of geographic and climatic variables across the study area. The entire scene was marginally suitable with a minimum predicted probability of 0.4, and the highest at 0.88, which occurred outside the GBMWHA. The general area calculated to be most suitable to *P. cinnamomi* was centred on the Blue Mountains NP, and covered the majority of the southern, middle and northern sections. Approximately fifteen locations within the Blue Mountains NP were identified as most suitable. These 'hot spots' were all within 200 m of a road, close to a previous detection, close to a water source (and therefore had increased soil wetness), and often at the base of canyons or waterfalls with large encompassing cliff faces. Such locations included Beauchamp Falls (Blackheath), Arethusa Falls (Leura), Wentworth Falls and Kanangra Falls (Figure 2.4). The suitability level at these locations was higher than anywhere else in the GBMWHA with the highest calculated probability at 0.86

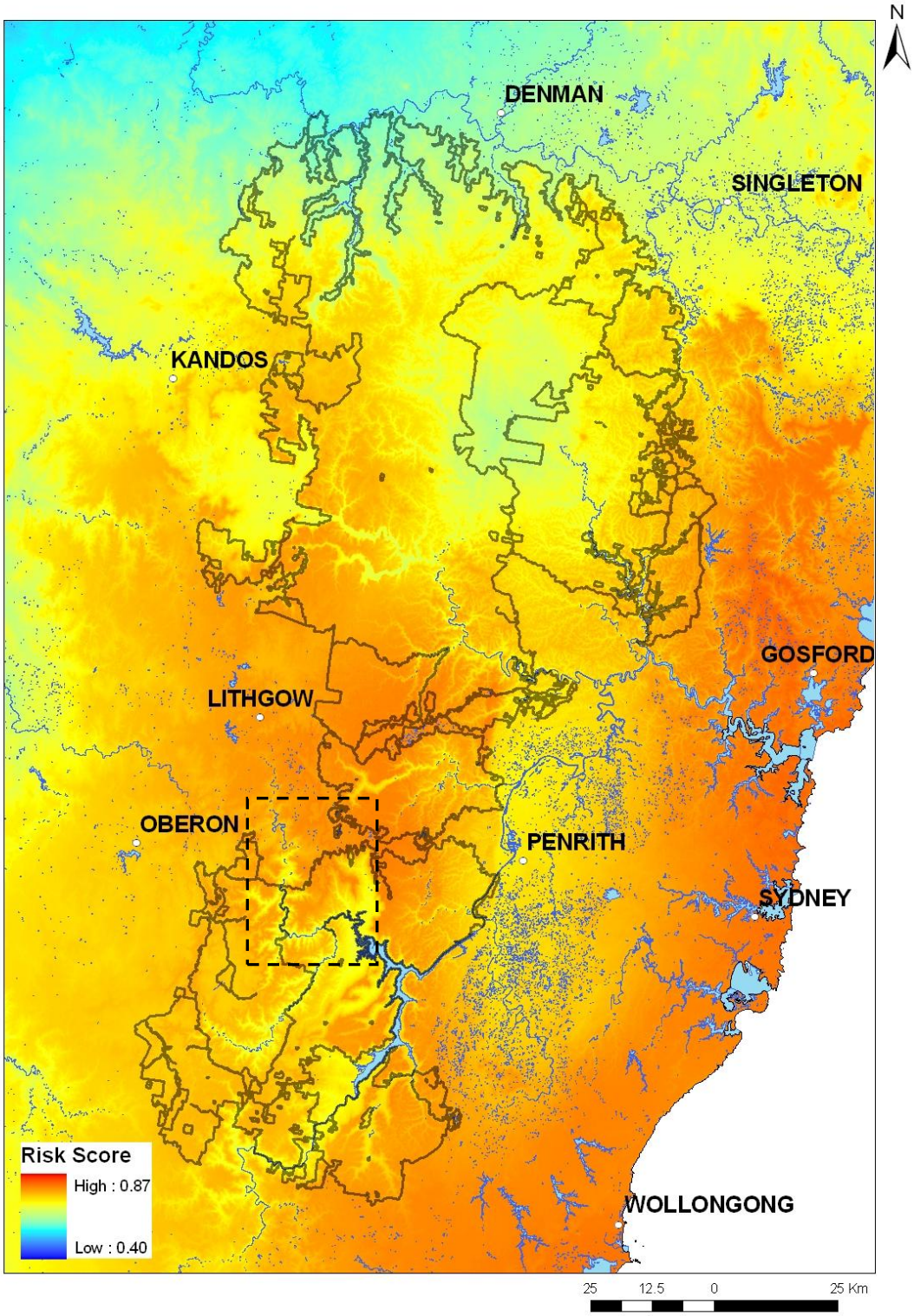
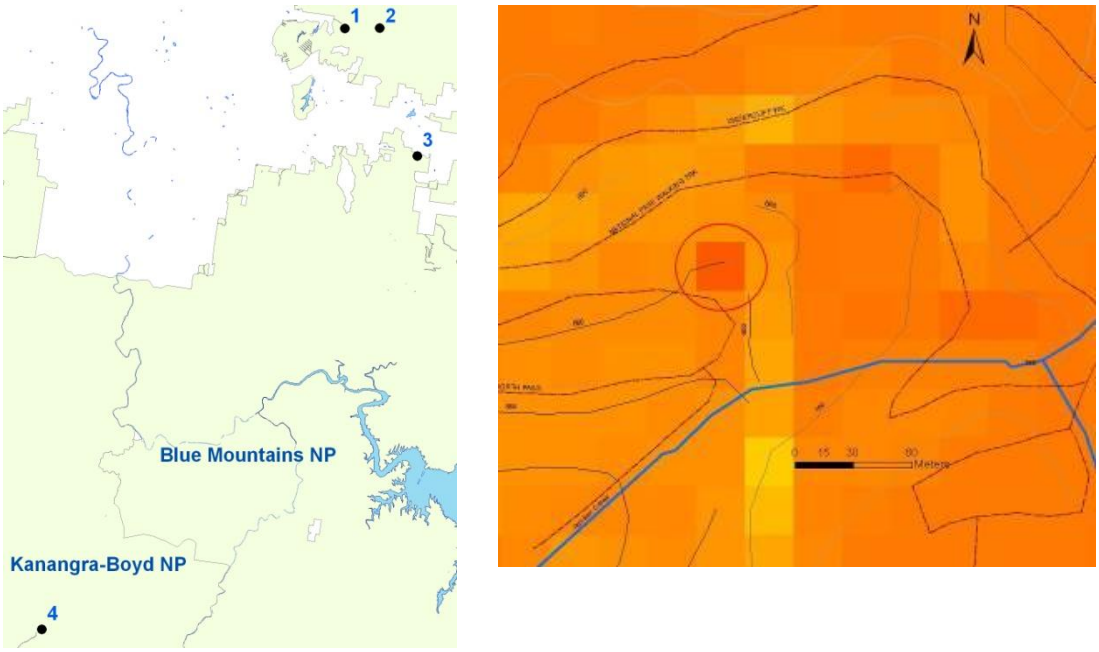


Figure 2.3 GBMWHA Phytophthora Risk Assessment map. This image shows the final risk model in which red indicates higher risk locations through to blue which indicates lower risk locations. The GBMWHA boundary is indicated by the grey line. The inset provides reference for Figure 2.4.



a) b)

Figure 2.4 Examples of high risk locations. The high risk locations were distributed between Kanangra-Boyd NP, Blue Mountains NP and Wollemi NP, examples of which can be seen in a) and inset in Figure 2.3 including (1) Beauchamp Falls, (2) Arethusa Falls, (3) Wentworth Falls, and (4) Kanangra Falls. b) shows the typical structure of a high risk location, using Wentworth Falls as an example. The 'hot spot' cell in is circled in red. The shape of the walking tracks (black lines) and contours (grey lines, 20 m) illustrated the encompassing nature of the hollow in which Wentworth Falls occurs. The close proximity of Jamieson Creek (blue) is also evident.

The area determined by the model to have the least risk was the northern most section of the Wollemi NP. The Wollemi NP also had the largest range of predicted probabilities with values occurring from the first to the fourth stratum.

2.3.2 The representation of risk across the reserves of the GBMWHA

Risk scores were distributed heterogeneously across the GBMWHA which was reflected in the distribution of the 5 risk strata (Table 2.2). Stratum 5 represented only 3.7 km^2 or 0.001% of all lands of the GBMWHA, the majority of which was found in the Blue Mountains NP. The reminder was found in the Gardens of Stone NP and Kanangra-Boyd NP. Stratum 4 was the predominate stratum representing just over 50% of all GBMWHA lands and was the only one to feature in all of the reserves. Stratum 4 was most common in the

Blue Mountains NP, unlike stratum 3 which was more common in the Wollemi NP and represented 44% of the whole GBMWHA. Stratum 3 was found in all reserves except Thirlmere Lakes NP.

Collectively Stratum 3 and 4 covered nearly 95% of all of the GBMWHA, indicating that 95% of the GBMWHA had a risk score between 0.75-0.85. Stratum 2 represented just below 6% coverage and only featured in Yengo NP and Wollemi NP, while Stratum 1, at 0.03% only occurred in a very small pocket in the most north-western section of the Wollemi NP. Although a 20% variation occurred in the risk scores across all five strata, *P. cinnamomi* was successfully isolated from all (Chapter 3).

Table 2.2 Stratum area of each reserve and percentage contribution of each stratum in the GBMWHA.

	Stratum Area (km^2)				
Reserve	**1**	**2**	**3**	**4**	**5**
Blue Mountains NP	0	0	536.451	2 144.765	0.042
Wollemi NP	3.739	579.402	2 834.184	1 602.347	0
Yengo NP	0	28.962	1 028.715	618.043	0
Natti NP	0	0	104.721	389.823	0
Kanangra-Boyd NP	0	0	162.305	532.054	0.02
Jenolan Karsk CR	0	0	0.143	30.786	0
Gardens of Stone NP	0	0	33.894	117.406	0.001
Thirlmere Lakes NP	0	0	0	6.619	0
Total area	3.739	608.364	4 700.416	5 441.847	0.062
(% of GBMWHA)	(0.03)	(5.66)	(43.71)	(50.6)	(0.001)

The higher risk scores associated with Stratum 3 and 4 were reflected in their higher positive detection rates for the entire GBMWHA (18.2% and 24.85% respectively) and within individual reserves (Table 2.3). There was a clear increase in the rate of detection between the second and third stratum (2.75% and 18.2%, respectively, Figure 2.5). The pattern of increasing detection with increasing risk, however, was not followed in

the fifth stratum where the number of positive samples identified decreased.

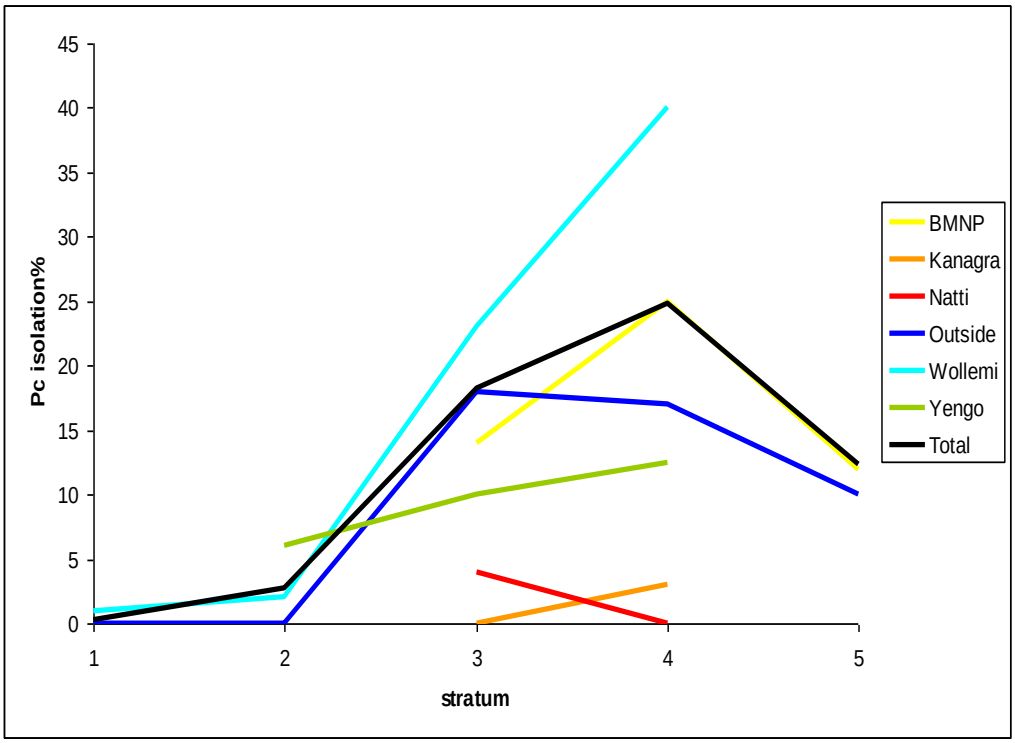


Figure 2.5 Detection rates for each reserve across the strata. Gardens of Stone NP, Jenolan Karsk CR and Thirlmere Lakes NP have not been included as samples were only collected from one stratum.

Table 2.3 Soil sampling results across each stratum and reserve. This table indicates the distribution of all detections for each reserve in each stratum.

RESERVE	RESULT [B]	Stratum [A] 1	2	3	4	5	Grand Total
	neg	-	-	55	288	77	420
	Pc	-	-	11	120	12	143
	P. spp	-	-	9	58	5	72
Blue Mountains	total	-	-	75	466	94	635
NP	*% Pc*	-	-	*14.67*	*25.75*	*12.77*	*22.52*
	neg	-	-	-	7	-	7
	Pc	-	-	-	1	-	1
	P. spp	-	-	-	-	-	-
Gardens of	total	-	-	-	8	-	8
Stone NP	*% Pc*	-	-	-	*12.5*	-	*12.5*
	neg	-	-	-	5	-	5
	Pc	-	-	-	-	-	-
Jenolan-Karsk	*P. spp*	-	-	-	-	-	-
CR	total	-	-	-	5	-	5
	% Pc	-	-	-	-	-	-
	neg	-	-	1	53	-	54
	Pc	-	-	-	2	-	2
	P. spp	-	-	-	7	-	7
Kanagra-Boyd	total	-	-	1	62	-	63
NP	*% Pc*	-	-	-	*3.23*	-	*3.17*
	neg	-	-	24	23	-	47
	Pc	-	-	1	-	-	1
	P. spp	-	-	5	1	-	6
	total	-	-	30	24	-	54
Natti NP	*% Pc*	-	-	*3.33*	-	-	*1.85*
	neg	-	-	-	1	-	1
	Pc	-	-	-	-	-	-
	P. spp	-	-	-	-	-	-
Thirlmere Lakes	total	-	-	-	1	-	1
NP	*% Pc*	-	-	-	-	-	-
	neg	349	245	187	87	-	868
	Pc	1	6	64	72	-	143
	P. spp	19	23	26	17	-	85
	total	369	274	277	176	-	1 096
Wollemi NP	*% Pc*	*0.27*	*2.19*	*23.10*	*40.91*	-	*13.05*
	neg	-	41	67	39	-	147
	Pc	-	3	8	6	-	17
	P. spp	-	2	4	3	-	9
	total	-	46	79	48	-	173
Yengo NP	*% Pc*	-	*6.52*	*10.13*	*12.50*	-	*9.83*
	neg	2	7	26	47	20	102
	Pc	-	-	7	12	3	22
	P. spp	-	-	5	8	5	18
Outside the	total	2	7	38	67	28	142
GBMWHA	*% Pc*	-	-	*18.42*	*17.91*	*10.71*	*15.49*
Grand Total		371	327	500	857	122	2 177

[A] Samples could not be collected from strata in every reserve as the range of probabilities calculated did not cover all five strata, or were outside the sampling area. As a result, for example, no samples were collected from stratum 1 or 2 in the Blue Mountains NP. [B] (*Pc*) indicates the isolations for *P. cinnamomi.*

Model testing 2.3.3

Chi-squared analysis revealed that the sampling outcomes were quite different to those of the model. The lower limit of each stratum was used to calculate the expected positive detection rate for each of the five strata. This, when compared to the actual detection rates, indicated that there were significant differences between the expected outcomes of the model and those achieved in the survey (Table 2.4). Even when the survey results were modified, significant differences still remained in each analysis. This included combining the fourth and fifth stratum, applying an increasing weight to the value of positives in each stratum, and spatially adjusting the results due to proximity to a positive sample. One non-significant difference was identified between the observed and excepted outcomes in the fifth stratum of the spatially adjusted data set.

Table 2.4 Chi-squared analysis of the sample data (Chapter 3) and the modelled data in its original and adjusted forms.

			χ^2 value [A]			
Stratum	Lower limit [B]	Upper Limit [C]	Original Data	4 & 5 combined	Negative Weighting	Spatially Adjusted
1	0.65	0.699	579.45	579.45	469.38	579.45
2	0.7	0.749	553.17	553.17	355.33	553.17
3	0.75	0.799	589.07	589.07	276.3	589.07
4	0.8	0.849	1 045.35	977	355.83	740.27
5	0.85	0.64	398.18	579.45	109.57	2.09*

The observed values were the sampling results presented in Chapter 3 and the expected values were those calculated in the model. [A] indicates the χ^2 value calculated for each stratum from each of the 4 Chi-squared analyses. The χ^2 test statistic for all was 3.84 with 1 degree of freedom at $\alpha = 0.05$. [B] the lower limit (risk score) of each stratum which was used as the expected value in the analysis. [C] the upper limit of each stratum. * indicated non-significant difference. The full analysis can been found in Appendix 8.3.

Aside from adjusting the raw data, an attempt to improve model outcomes by changing the bin ranges was also performed (Error! Reference source not found.). This created five equidistant bins (i.e., the range of risk values covered by each was equal) and produced a more even sample allocation across the five strata. This still resulted in significant differences between the observed and expected outcomes of the model. The increase in the positive detection rate between the second and third stratum remained (5.99%-16.48% , respectively).

As the ROC calculation can only be conducted on binomial data, the results were partitioned into a binomial response. In the case that a *Phytophthora* species other than *P. cinnamomi* was isolated, the result was initially included in the analysis as a negative

sample. This gave an AUC of 0.67 and reflects the ability of the model to predict the distribution of *P. cinnamomi*. In the second instance, *Phytophthora* species was included as positives, thereby testing the ability of the model to predict the distribution of *Phytophthora, sensu lato*. The AUC increased to 0.77. These results indicate that the model performed better than a random prediction.

Table 2.5 Modified bin ranges of equal width with recalculated detection rates.

Stratum	New lower limit	New upper limit	New *P. cinnamomi* positive rate, %
1	0.696	0.728	0.22
2	0.728	0.76	5.99
3	0.76	0.792	16.48
4	0.792	0.824	24.63
5	0.824	0.856	21.27

2.4 Discussion

Based on the model outcomes, the GBMWHA is a highly conducive environment for *P. cinnamomi* with 95% of the GBMWHA having a suitability score between 0.75-0.85. The Blue Mountains NP appears at greatest risk from *P. cinnamomi*, while the northern sections of the Wollemi NP are at the least risk in comparison to the whole World Heritage Area. The results verify that the rate of *P. cinnamomi* detection increases across the five risk strata suggesting that the model is able to predict the relationship between the distribution of *P. cinnamomi* and the environment. This efficacy of the model was supported in the result of the ROC analysis.

The final model produces a relative, static map of the risk of the GBMWHA for *P. cinnamomi*. The results are relative because one cell is compared to its neighbour and the whole scene; risk would be different if the region of interest was elsewhere. The results are static as they reflect conditions at the time of model construction. These conditions, however, have been calculated using annual averages and therefore do not represent changing risk from one season to the next, but longer term risk. The map itself is geographically referenced, thereby allowing the quantification of risk at specific locations within the GBMWHA.

Increasing risk predicted by the model was reflected in an increasing rate of *P. cinnamomi* detection across the strata indicating the model was able to predict the pattern

of detection across the GBMWHA. The detection frequency of *P. cinnamomi* also tended to increase in subsequent strata across each reserve.

The exception to this was a decrease in the detection of *P. cinnamomi* in Natti NP and a decrease in the detection rate in the Blue Mountains NP between the fourth and fifth stratum. The rapid increase in the total detection rate between the second and third stratum for the whole survey is an artefact of the detection rate in the Wollemi NP and areas outside of the GBMWHA, but indicates that the environment becomes increasingly more conducive between the risk values of 0.7 and 0.75. Without regression analysis it is impossible to determine which of the variables included in the model are responsible for this increase, but it is likely to prove informative in producing a statistically based model of the distribution of *P. cinnamomi* in the GBMWHA.

The reserve with the highest predicted risk was the Blue Mountains NP. This result reflects the highly conducive environment in the Blue Mountains NP in which temperature and rainfall conditions are ideal for *P. cinnamomi*, transport vectors abound and there is already evidence of *P. cinnamomi* within the area. Conversely, the least risk occurred in the northern most sections of the Wollemi NP. These areas are comparatively drier and warmer, with less human influence and no known occurrence of *P. cinnamomi* at the time of model construction. This does not indicate that the northern section of Wollemi NP does not contain the necessary environment to suit *P. cinnamomi*, (indeed, *P. cinnamomi* has been isolated in the vicinity of the Wollemi pine site, E. C. Y. Liew, pers. comm.). It simply means that, relatively speaking, the Blue Mountains NP is more suitable for the establishment of *P. cinnamomi*. The model also predicted higher risk for the Natti NP in which only one positive sample were identified. This does not necessarily indicate that the model prediction is incorrect; it could indicate that the pathogen has not yet spread to this area or that the sampling effort needs to be expanded for model validation purposes. This raises the issues of whether or not the information on the known distribution of *P. cinnamomi* should have been included as it biased the model toward those areas that already contained *P. cinnamomi*. It was, however, included to maximise the chance of isolating *P. cinnamomi* while conducting the field survey. Re-running the model without the 2008 sampling results would allow for the detection of any bias towards those areas already known to contain *P. cinnamomi*. Unfortunately the version of Idrisis used to contrast the model is no longer available meaning that both models would need to be reconstructed in an alternate program before any comparison could be made.

The assignment of variable weight was done objectively in the context of the literature. The highest weight, given to that of current disease localities, was perceived as

most important as an initial point of introduction is required for spread, and uninfested areas adjacent to infested areas present the greatest risk (Pratt and Heather, 1973; Weste, 1974). Following this, climatic variables (temperature and rainfall) that control the survival of *P. cinnamomi* as well as the means of spread, (unsealed road network and walking paths) were given the second level risk weight. These are the layers that dictate whether or not *P. cinnamomi* will survive if it reaches a new location and the easiest and most likely means by which it will spread (Marks *et al.,* 1972; Pratt and Heather, 1973; Weste, 1974; Podger and Brown, 1989; Peters and Weste, 1997). Sealed roads, as with all other vehicular corridors were scaled to reflect decreasing risk with distance from the origin. By giving a higher weight to these variables, greater emphasis is placed on anthropogenic activity which spreads *Phytophthora* further and faster than it can spread naturally (Podger and Brown, 1989; O'Gara *et al.,* 2005). The third level weights are related to comparatively less important methods of spread (sealed roads and slope) and survival (wetness). Slope, was rescaled such that the risk increased with increasing slope, as *P. cinnamomi* moves faster down steeper slopes (Weste and Ruppin, 1975). A similar reclassification method was used for the wetness variable, as increasing wetness leads to increasing suitability, as higher soil moisture is more conducive to establishment and spread (Weste and Marks, 1987; Duncan and Keane, 1996; Laidlaw and Wilson, 2003). Rainfall variables are linked with this, i.e., as rainfall increases, so will soil moisture. These 'third-level' variables give an indication of long term survival and proliferation of *P. cinnamomi*. The three successive levels could be seen to reflect the invasion process as establishment, spread, and persistence of *P. cinnamomi*.

As a static model, the prediction of distribution risk assumes *P. cinnamomi* has reached equilibrium (Guisan and Zimmermann, 2000). As an introduced organism this is not likely to be the case, which must be taken into consideration when viewing risk predictions (Elith and Leathwick, 2009). It also assumes that environmental variables, such as soil types, vegetation types, and ground covers, are suitable. *Phytophthora cinnamomi* is favoured by poorly draining soils with a higher sand content (Marks *et al.,* 1972; Weste and Marks, 1987) and is better suited to a slightly acid pH (Falcon *et al.,* 1984; Erwin and Ribeiro, 1996); however none of these were given consideration during model construction. This keeps the model simple and transferable and ensures only universal variables are maintained should it be applied to another space.

A model of the risk of *P. cinnamomi* in the Royal NP NSW, was recently developed based on the known distribution of *P. cinnamomi*, and the distribution of soil and vegetation classes (Keith *et al.,* 2012). Such specific variables make the model less

transferable to other areas. Additionally, the model was still dependent on an understanding of the distribution of *P. cinnamomi* across the Royal NP, and therefore could be considered biased towards areas with higher levels of sampling. The authors admit that the samples did not cover the full range of variables and therefore regression analysis was likely hampered by this. By constructing the model using ecological principles known to be true, a model cannot be biased because the range of each variable has been sampled.

The overall approach of the model construction in this chapter is therefore mechanistic (Guisan and Zimmermann, 2000; Buckley *et al.,* 2010). The predicted distribution is determined by the physiology of the organism irrespective of the environment it has been recorded in. Not only does the model reflect the current distribution of *P. cinnamomi* in environmental constructs it currently inhabits, it also identifies new environments that allow survival and reproduction, thus providing an image of the pathogens fundamental niche (Grinnell, 1917; Whittaker *et al.,* 1973; Guisan and Zimmermann, 2000). Ultimately the model is quite simple to construct and can be easily transferred to other areas, making it ideal for land managers who wish to investigate the possibility of the occurrence of *P. cinnamomi* with little or no information on its current distribution. Put simply, the model allows for spatial risk prioritisation with a known level of certainty.

Regardless of the ability to model the distribution of a species, an even more important question still remains. Supposing the model tells us that location A is more environmentally suitable than location B; they are, however, both susceptible. What is perhaps more important is a consideration of the level of damage applicable to that location. According to the disease triangle concept, disease is a function of a suitable environment, a viable pathogen and a susceptible host. If there are greater numbers of host that are more susceptible at Location B, disease will be worse. The host distribution is more difficult to predict. Because *P. cinnamomi* has potentially over 3,000 host species in Australia (Shearer *et al.,* 2004; O'Gara *et al.,* 2005), of which over 130 inhabit the GBMWHA (Appendix 8.1), it is argued that wherever plants could be found, *P. cinnamomi* would likely find a host. Therefore the addition of a vegetation layer would be redundant as the GBMWHA is predominantly vegetated. Additionally, there were no standardised or consistent vegetation layers for the GBMWHA at the time of model construction. Inconsistent coverage of vegetation is not suitable for modelling. Determining the distribution of an appropriate climatic envelope is likely to be more powerful than modelling the distribution of susceptible hosts (Kelly *et al.,* 2007). Consideration however, still needs to be given to the distribution of host, especially for those that may be highly

susceptible or have conservation status. The management of such species can be considered in conjunction with the pathogen distribution model.

Aside from the incorporation of host information, numerous other predictions of *Phytophthora* distributions illustrate ways in which this model could be improved. Land use has previously been incorporated (Meentemeyer *et al.,* 2004), but this is perhaps not applicable to a national park. Other variables used effectively include altitude (Wilson *et al.,* 2000), elevation and a sun index (Wilson *et al.,* 2003), aspect (Marcais *et al.,* 2004), soil pH (Vettraino *et al.,* 2005), drought and heat stress (Venette and Cohen, 2006) and soil type (Keith *et al.,* 2012). Although some of these variables were available at the time of model construction, they were not incorporated as the most simple models are often the most effective (Elith and Leathwick, 2009). Nevertheless, some of this information was partially included in the model, such as the relationship between aspect and soil wetness and the effects of rainfall and temperature on drought and heat stress. Confusion remains, however, as some of these variables may make a substantial contribution in one study, but make no contribution in another. Therefore, model selection is necessary and possible once detection information is available.

Under the original model, significant difference occurred across each of the five strata between the predicated probability (expected values) and the rates of detection achieved in the field (observed values). One explanation for this may simply be that *P. cinnamomi* has not yet reached equilibrium within the environment of the GBMWHA (Hutchinson, 1959; Elith and Leathwick, 2009) meaning that it has not spread to all inhabitable environments. When sample results were spatially adjusted to reflect a higher degree of spread within cells, no significant difference was detected in the fifth stratum, supporting this notion and demonstrating that the difference between the modelled distribution and the current distribution is equal to potential spread. The total percentage detection recorded for each stratum followed that of the predicted model (i.e., the observed and expected data had a similar slope), except between the fourth and fifth strata. The fifth stratum represented the highest risk areas and included most of the 'hot spots'. If the soil in these locations is permanently waterlogged (and therefore effectively anaerobic), this may prevent the survival of plants and/or *P. cinnamomi*. Numerous studies indicate that water logging favours disease (Weste and Taylor, 1971; Broadbent and Baker, 1974; Davison *et al.,* 1994; Erwin and Ribeiro, 1996; Laidlaw and Wilson, 2003; Hardham, 2005) with minimal indication that the prolonged presence of excess water is detrimental (Nesbitt *et al.,* 1979). The combination of very low winter temperatures as well as the addition of extended saturation can, however, suppress sporangial production and enhance hyphal

lysis (Weste and Vithanage, 1979). As the information currently available is contradictory, it is not possible to conclude why detection decreased in these areas. *Phytophthora cinnamomi* is often recognised as having a patchy distribution in the soil (Podger and Ashton, 1970; Weste and Taylor, 1971; Weste and Kennedy, 1997;Brown *et al.,* 2002; Pryce *et al.,* 2002; Gadek and Worboys, 2003; McDougall *et al.,* 2003). The reason for the disparity between the predicted risk and that rate of isolation may also be due to false negative sampling results in which, although present, *P. cinnamomi* was not successfully isolated (Pryce *et al.,* 2009; Davidson and Tay, 2005).

According to the ROC analysis the model performed relatively well; however, attempts were made to improve the model outcomes and strengthen its predictive probability. The approaches used included altering the sampling results or the arrangement of samples within the strata. In the case of the latter, the fourth and fifth strata were combined and bin ranges were redistributed to create five equidistant bins. Significant difference still remained. Differential weights were also applied to the negatives in each stratum to reduce their number and increase the influence of the positive samples on the detection rate. In this case it is argued that as predicted probability increased, the chance of false negatives also increased. Even so, this made no further improvements to the model performance. The final attempt to adjust the data to improve model outcomes was with the spatial adjustments. These reclassified samples were in some cases a few meters away from positive samples, and given enough time, *P. cinnamomi* would likely spread between them. The second justification for this was that *P. cinnamomi* often has a patchy distribution (Podger and Ashton, 1970; Weste and Taylor, 1971; Weste and Kennedy, 1997; Brown *et al.,* 2002; Pryce *et al.,* 2002), including NSW (McDougall *et al.,* 2003), and therefore any number of negative and positive detections may be isolated from one location which would ultimately be classified as an infested site and managed accordingly.

The AUC was improved by the way the data was categorised to run the ROC analysis. Initially the data was classified such that the negative results equalled a negative detections, *P. cinnamomi* equalled a positive and all other *Phytophthora* species equalled a negative. This still produced a model better than random (0.67). However, when the other *Phytophthora* species were reclassified to positive, the AUC improved by 10 points. This suggested that the model was not only effective at predicting the distribution of *P. cinnamomi* but *Phytophthora* species more generally. Although there is much variation in the growth conditions required for the presently known *Phytophthora* species, it makes sense that the model performed better including these species as they were collected from the GBMWHA and are therefore obviously suited to the environment. Although

construction of this model has not been concerned about risk associated with *Phytophthora* species other than *P. cinnamomi*, their detection warrants further investigation as all formally described species are plant pathogenic often with multiple hosts (Erwin and Ribeiro, 1996; Kroon *et al.,* 2012). Regardless of the adjustments made to either the input data or the way in which the analysis was constructed, the model prediction is better than a random prediction and therefore still has the capacity to explore *P. cinnamomi* risk.

This model has already proved helpful to land managers of the GBMWHA by allowing them to identify areas of risk within their jurisdiction (R. Harris, NSW NPWS, pers. comm.). The identification of Natti NP as a higher risk area with only one positive detection highlights the need for management strategies to keep *P. cinnamomi* out of the area. The model can also be utilised to identify those areas at higher risk within individual management areas of the GBMWHA. For example, the identification of high risk trails that should not be traversed in wet weather, or reducing the risk of possible cross contamination from high risk trails proximal to those that are lower risk. The model will continue to be used to support prevention and treatment management strategies across the GBMWHA.

The model presented is simple and easily transferable. It uses expert information to make an interpretation of the potential distribution of *P. cinnamomi* within the GBMWHA. The product is a map that clearly identifies parts of the GBMWHA with greater environmental suitability and therefore greater risk of infection. The model has not required a complex statistical analysis, but only geographic layers classified according to species ecology. Although there is much room for improvement, the model performed better than a random prediction and was, on the whole, able to identify an increasing probability of risk that was supported by an increasing rate of detection in field results.

Chapter 3 The distribution of *Phytophthora* across the Greater Blue Mountains World Heritage Area.

3.1 Introduction

Understanding the distribution and imapct of *Phytophthora cinnamomi* in an ecosystems is required to determine the order in which management options must be implemented, and to ensure their success (O'Gara *et al.,* 2005). To minimise ecosystem degradation caused by *P. cinnamomi* in the GBMWHA, land managers need more information to deal with disease in a cost effective and timely manner. A survey conducted in 2008 (Suddaby, 2008a) is the only available information on the distribution of *P. cinnamomi* within the GBMWHA. These results are informative in terms of identifying the distribution of *P. cinnamomi* in some of the highly visited areas, but are by no means comprehensive in terms of either geographic or environmental coverage. Additionally, the impacts of *P. cinnamomi* on species or ecosystems within the area was not comprehensively reported. This is limiting for all aspects of management and research.

In vegetation communities highly susceptible to Phytophthora dieback, disease may result in destruction of ecosystem structure and composition, loss of biodiversity, and loss of habitat for those animals which depend upon it (Commonwealth of Australia, 2005). Environmental degradation associated with dieback caused by *P. cinnamomi* is a direct threat to the criteria for which the GBMWHA was listed; specifically *in-situ* conservation and ongoing biological process. As a plant pathogen with perhaps as many as 3,000 native host species in Australia, it is not surprising that we find as many as 130 host species growing within the GBMWHA (Appendix 8.1) across a wide range of vegetation types. There may be many more susceptible species that have not yet been described. Out of these 130 species, at least five are currently vulnerable or endangered, or form part of an Endangered Ecological Community. Under Commonwealth legislation (Environment Protection and Biodiversity Conservation Act, 1999) both the National Parks and Wildlife Service and the Sydney Catchment Management Authority are responsible for investigating the impact of *P. cinnamomi* on these plants within the GBMWHA. Additionally, they are responsible for ensuring the values for which World Heritage status was granted are maintained. *Phytophthora cinnamomi* is present and causing disease within the GBMWHA (Suddaby, 2008a) but the extent of spread or impact on biota remains largely unknown.

In NSW dieback has been identified in many of our National Parks including Mt Imlay (McDougall and Summerell, 2003), The Royal NP (Walsh *et al.,* 2006), Sydney Harbour NP (Daniel *et al.,* 2006; Howard, 2008), Barrington Tops NP (National Parks and Wildlife Service, 2010), Werrikimbi NP (Howard, 2008), Dorrigo NP (Howard, 2008; Anon., 2012), New England, and Cunnawarra National Parks (Anon., 2012). In many of these cases, investigations into the distribution of *P. cinnamomi* have occurred after the appearance of symptoms, that is, after the pathogen has already started to affect ecosystem function.

The extensive distribution of *P. cinnamomi* along the NSW coast from the Victorian border to the Queensland border suggests it has long been established and likely been reintroduced multiple times (Weste, 1975a; Weste and Marks, 1987; Howard, 2008). Frank Podger suggested that while South Africa and Indonesia were Dutch colonies, nursery material was gathered from Indonesia and Papua New Guinea where *P. cinnamomi* likely has its origins (Dobrowolski *et al.,* 2003). Infected nursery material was then transported to South Africa where it joined international trade routes and thus may have been transported to Australia with the earlier settlers (D. Guest, pers. comm.). The Blue Mountains themselves were being explored from the late 1700s not long after initial European settlement and were finally crossed in 1813. Within two years the Great Western Highway was constructed and became the major transport corridor between the Sydney Basin and the Western Plains – the agricultural food bowl that saved the colony (Spriggs, 1962). From then, explorers and pioneers moved north following the routes of the Putty and Great Northern Roads into the realms of the Hunter Valley, and south in the Burragorang Valley and Goulburn, constantly searching for new land and new ways to cross the Mountains (Spriggs, 1962). Railroads followed in the 1860s, all the while mining, logging and agriculture grew utilising the many valuable resources found within the GBMWHA (Spriggs, 1962; Bayley, 1980). Additionally, the Blue Mountains became a popular tourist location by those seeking to see its landscapes and "breathe its healing air" (Spriggs, 1962). These activities we recognise today as having the capacity to spread *Phytophthora* via the movement of contaminated soil, gravel or plant material. Given 200 years of European activity within the GBMWHA, *P. cinnamomi* has possibly been in the area for many decades. With the first confirmed identification of *P. cinnamomi* affecting native vegetation within the Sydney region in 1948 (Fraser, L., 1956, cited in Newhook and Podger, 1972), it is certainly possible that *P. cinnamomi* has been present in the GBMWHA for at least the last 70 years.

It appears however that Phytophthora dieback has subsequently gone unnoticed or that its impacts on the vegetation of the GBMWHA are not as severe as those expressed in

other parts of Australia. This is common in NSW where asymptomatic plants allow the spread of *P. cinnamomi* to go unnoticed (Pratt *et al.,* 1973; McDougall *et al.,* 2003). The confounding factor influencing the expression of disease is likely to be climate, such that the Mediterranean climates of southern Western Australia, South Australia and Victoria with winter dominant rain and hot dry summers, encourages both the pathogen and the expression of disease. In temperate climates such as NSW where rainfall and temperature are more even throughout the year, the environment is less conducive to dieback as water stress which triggers the secondary disease symptoms is less likely to occur (O'Gara *et al.,* 2005).

As a result of investigations into *P. cinnamomi* in NSW, important findings have been made and management options have been prioritised accordingly. Dieback was first reported in the Barrington Tops National Park in the 1990s (National Parks and Wildlife Service, 2010). Subsequent soil sampling revealed that death in a number of species was likely to be caused by *P. cinnamomi* (McDougall *et al.,* 2003). A quarantine area was established within the park to reduce additional spread by the public and hygiene apparatus were installed to prevent NPWS staff from inadvertently spreading the pathogen (National Parks and Wildlife Service, 2010). In the Wollemi NP, the endangered Wollemi Pine (*Wollemia nobilis*) identified as susceptible to *P. cinnamomi* (Bullock *et al.,* 2000) was found displaying dieback symptoms in the wild in 2005. Soil sampling revealed *P. cinnamomi* had been introduced into the previously *Phytophthora*-free area. Subsequently a monitoring program was established, and chemical control was utilised to suppress pathogen spread (D. Crust, pers. comm.; NSW Department of Environment and Conservation, 2006).

Phytophthora cinnamomi was also isolated from soil surrounding the endangered *Eucalyptus imlayesis* at Mt Imlay NP in southern NSW (McDougall and Summerell, 2003). It was recognised that susceptibility trials were required to determine if *P. cinnamomi* had a role in plant death, and that *ex-situ* conservation was essential for their survival (James and McDougall, 2007; Environment Australia, 2008). And, finally after surveys were conducted in the Royal NP in 2001, it was determined that controlling hygiene would be of little use in the park as *P. cinnamomi* was so widely dispersed. Instead a long-term monitoring program was recommended to investigate the disappearance of Waratahs (*Telopea speciosissima*) possibly as a result of *P. cinnamomi* infection (Walsh *et al.,* 2006). The outcomes of each of these surveys were different. Even though *P. cinnamomi* was identified in each, hygiene was not always feasible and indeed quarantine would not be either. Some hosts were suited to chemical treatment while others would possibly only survive through *ex-situ*

conservation efforts. The distribution of *P. cinnamomi*, the conservation and health status of the plants, and the extent of disease dictate appropriate, site specific, management outcomes. These can only be determined once the pathogen distribution is better understood.

Information on the distribution of *P. cinnamomi* also has important outcomes for research, which then feeds back into management. Once data has been acquired, it can be used to determine which environmental constructs control the distribution of disease. This information can be used to build risk models of habitat suitability, enabling preventative measures to halt *Phytophthora* dissemination. Where *P. cinnamomi* is not isolated from diseased vegetation, additional research is required to identify the cause. As mentioned in the above cases, following soil surveys, research into the susceptibility of individual species, establishment of ongoing monitoring trials of plant health or investigations into the secondary impacts on border ecosystem function may be warranted.

The initiation of research and management begins with an understanding of the pathogens distribution (or the acknowledgement of a lack of understanding) and an understanding of the (potential or realised) impacts a pathogen has. This chapter reports investigations into the occurrence of *P. cinnamomi* across the GBMWHA, and begins to address the specific impacts *P. cinnamomi* is having on individual species and ecological communities. The data collected also enables the testing of the pathogen distribution model in Chapter 2, and the construction of a disease distribution model in Chapter 4, both of which would not have been possible in its absence. Environmental outcomes are discussed as are implication for management and additional research priorities.

3.2 Methods

3.2.1 Sampling strategy

As outlined in the previous chapter, an expert-driven risk assessment of the probability of *P. cinnamomi* distribution was developed for the GBMWHA. (Chapter 2, Figure 2.2). Once the risk model was established, it was then used to devise a sampling strategy to investigate the distribution of *P. cinnamomi* across the GBMWHA.

The ‘sampling area’ was determined as all land within 500 m of roads (Figure 3.1). This was simply the area within the GBMWHA that was deemed reasonably accessible within the time frame given for sampling and the resources made available. The sampling area was then used to clip the risk assessment, and the area remaining within each of the five risk strata was calculated using ArcMap 9.3 (Table 3.1)(for further explanation on the stratification process refer to Chapter 2). The calculated area was then used to determine

the number of samples required to achieve 95% confidence in the results with 5% error using the method of Krejcie and Daily (1970) where individuals were substituted with metres squared. Random points were allocated to each stratum using ArcMap (Table 3.1), resulting in a stratified random design containing 1,915 sample locations (Figure 3.2).

Table 3.1 Representation of each stratum within the sampling area and the number of samples to be collected from each.

Strata	Area (Km^2)	Number of samples
1	0.529	384
2	46.611	384
3	783.296	384
4	1,703.413	384
5	0.030	379
total	**2,533.879**	**1,915**

3.2.2 Sample collection

Soil sampling was conducted between November 2010 to May 2011, and September 2011 to April 2012. These two sampling seasons were chosen to ensure that samples were collected while soil temperatures were above the minimum isolation temperature of 10-12˚C (Weste and Ruppin, 1977), and when rainfall and temperature conditions became ideal for pathogen activity, maximising the likelihood of isolation.

3.2.3 Sample collection and *Phytophthora* baiting.

Sample points were loaded onto a Garmin Oregon 550 GPS which was used to identify them in the field. Where the exact sample location could not be reached, soil was collected as close to as possible, or the sample was repositioned to a proximal location within the same risk class. Once the sampling location had been reached, a surface sterilised trowel was used to remove the humic layer and sample the soil and root material to a depth of 10 cm. Three to four subsamples were collected over about a one metre square, each being combined into a labelled, sealable plastic bag. While the samples were being collected, the GPS was set to average its location and this value was then assigned as the location of sample collection. This helped to ensure that coordinates were recorded as accurately as possible. Once all the subsamples were collected the bag was sealed and the trowel was surface sterilised with 70% (v/v) methylated spirits until no more soil particles remained. Photos were also taken at locations exhibiting symptoms of dieback using a digital camera and field notes describing such characteristics were made.

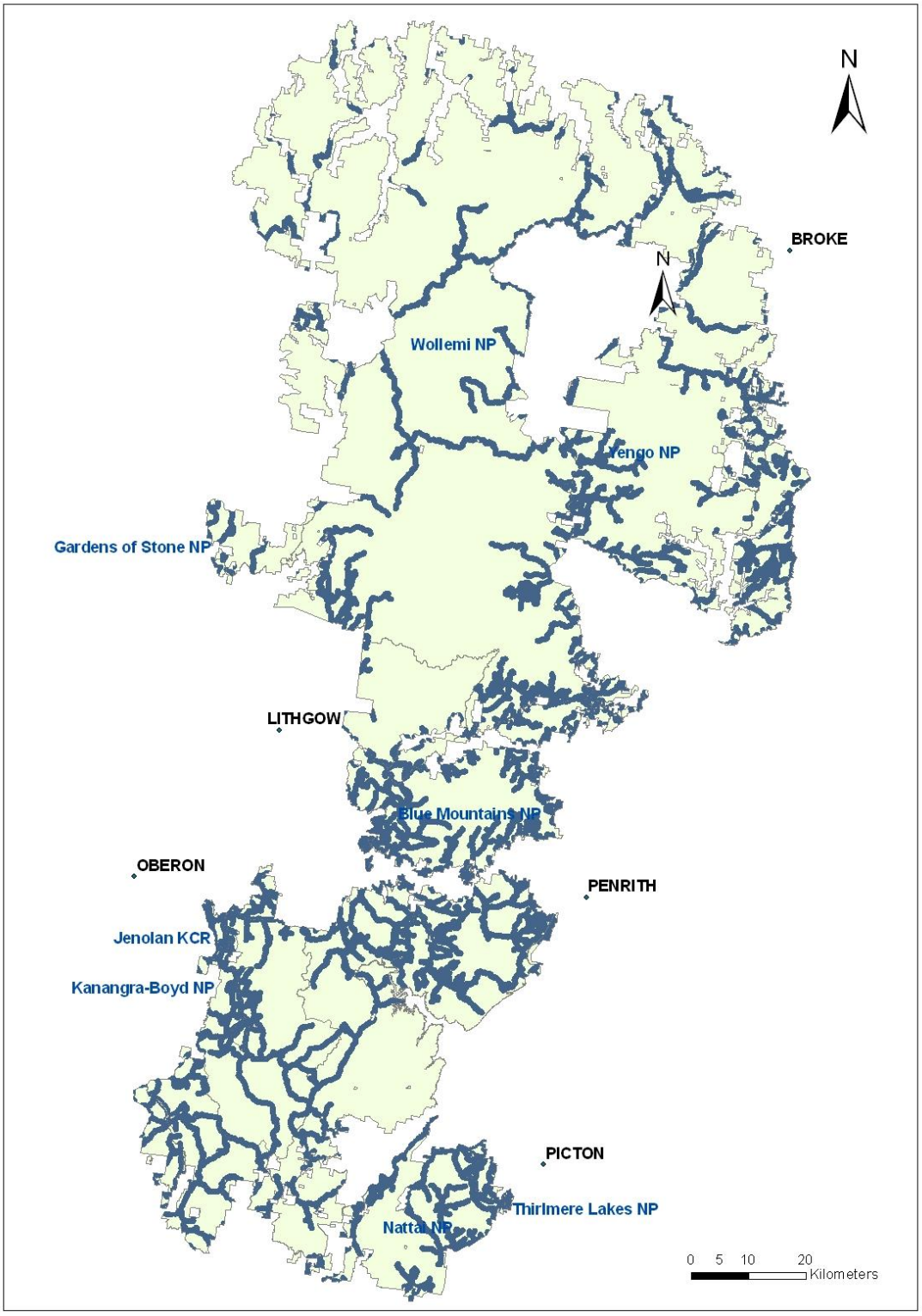


Figure 3.1 Sampling area within the GBMWHA. The area marked out in blue is all lands within 500m of a road, track or path, and was designated the sampling area from which soil samples would be collected.

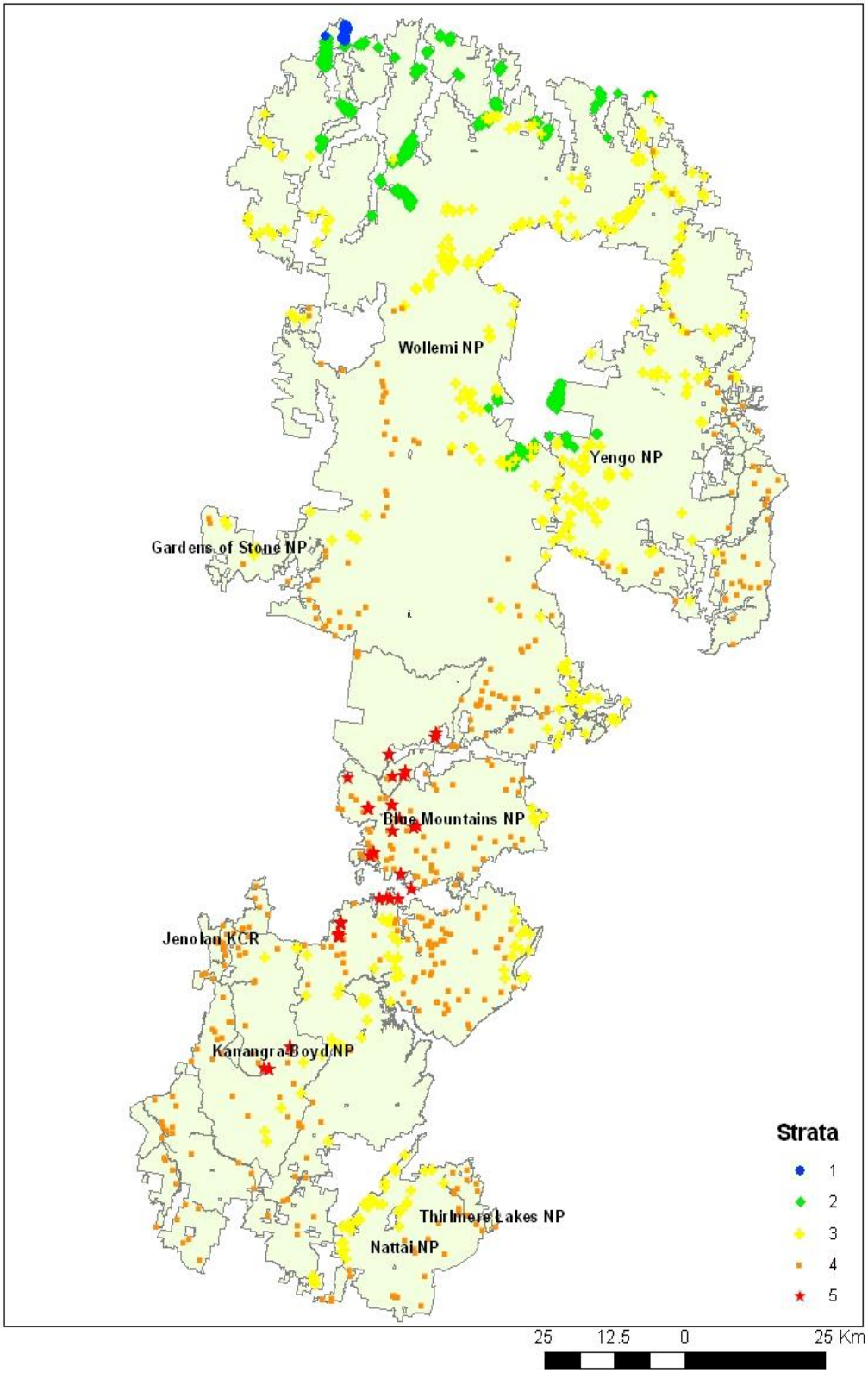


Figure 3.2 Sampling scheme for the distribution *P. cinnamomi* in the GBMWHA. Each point represents a single sample location. Different shape and colours combinations show samples to be collected within the different strata, for example blue • indicates the samples to be collected from the first (lowest risk) stratum and so on.

After the first season of sampling (2010-2011), the allocation of samples across the five strata and across the environmental variables included in the model (Chapter 2) were investigated. It was found that samples from the highest risk category (Stratum 5) had often not been reached and instead the samples had been collected from the fourth stratum. To acquire an appropriate number of samples from cells with a risk value above 0.85, an additional set of samples was randomly allocated to the high risk 'hot spots' identified within the model. Some of these sites occurred outside of the GBMWHA but were still within a short distance to the park boundary and were at least joined by a common waterway. The presence of *P. cinnamomi* neighbouring the park poses as a significant risk due to the potential movement of contaminated soil and water. These additional sampling locations were deemed necessary to ensure the effective evaluation of the expert driven model and the subsequent construction of a statistically-based model. It was also found that the remaining locations were from environments that had not yet been sampled, and therefore coverage of all variables should be achieved on completion of the survey.

3.2.4 *Phytophthora* baiting and identification

Soil samples were maintained at room temperature until they could be processed at the Plant Disease Diagnostic Unit at the Royal Botanic Gardens in Sydney. The lupin baiting method of Chee and Newhook (1965) was used with some minor modifications. Each soil sample was homogenised in the sealed plastic bag then approximately 150 g was equally divided among three 3 plastic cups. Distilled water was added to each cup at a ratio of 1:4, making a slurry. Each cup was then baited with four, 2-day-old New Zealand Blue Lupin (*Lupinus angustifolius*) seedlings (Rocklea Seeds, Sassafras, Tasmania) and observed microscopically for the presence of sporangia after five and seven days. On the seventh day, the lupins were replaced with a new set in a modified form of double baiting which can improve isolation results (Jeffers and Aldwinckle, 1987; Davison and Tay, 2005).

Whenever characteristic pear-shaped sporangia were observed, this was recorded as a result positive for the genus *Phytophthora*. In such cases, or when necrotic root tissue was present, four to five 1 cm pieces of lupin tissue were removed, surfaced sterilised in 70% (v/v) ethanol for 30 seconds and then plated on Phytophthora Selective Media (Appendix 8.2). Plates were then stored at 25˚C in the dark for a minimum of five days before they were removed and inspected. The presence of *P. cinnamomi* was determined by the branching of sporangia, the presence of numerous chlamydospores and coralloid hyphae in culture as per Erwin and Ribeiro (1996). All other *Phytophthora* species were categorised to

the genus level only. All *Phytophthora* cultures were then hyphal tipped to produce single isolates for long term storage. These isolates as well as all of the soil have been stored at the Plant Disease Diagnostic Unit at the Royal Botanic Gardens, Sydney.

3.2.5 Statistical analysis

Clustering of the sampling results across the GBMWHA was investigated in ArcMap. Moran's I (Spatial Analyst) was used to determine the distance at which the Z score first decreased, ie, where the level of spatial autocorrelation started to decrease. This distance was then used to test for clustering using Getis-ord, Gi* (Spatial Statistics), using the search method of Zone of Indifference allowing for some flexibility in the search neighbourhood. Data were partitioned to identify statistically significant clusters.

The relationship between the distribution of *Phytophthora* and minimum temperature, maximum temperature, annual rainfall, slope and road distance was assessed using Analysis of Variance (ANOVA). Each of these layers has been outlined in Chapter 2. The value of each of these variables at each sampling location was determined in ArcMap and then transferred to R (R Core Team, 2012) for statistical analysis. ANOVA and Tukey's HSD was used to identify where significant difference occurred between the environment that *P. cinnamomi, Phytophthora* species, or a negative result were obtained. Chi-squared analysis was also employed in R to investigate if there were significance differences in the rate of isolation of *P. cinnamomi* and *Phytophthora* species from different vegetation formations in the GBMWHA.

3.3 Results

3.3.1 Distribution of *Phytophthora* across the GBMWHA

By the end of the sampling program 2,177 samples had been collected (Figure 3.3) from which 329 isolates of *P. cinnamomi* and 197 isolates of *Phytophthora* were obtained. Although not all of the 1,915 sites from the original sampling strategy were successfully sampled, numerous other samples were contributed by staff at the NPWS, by volunteers and trained members of the public. Of particular note were the samples collected in April of 2012 on the 'Root Rot Trot' expedition, in which 16 volunteers collected 273 soil samples from the most remote section of the Wollemi National Park. This expedition was specifically

established to collect data from a largely unsampled section of the GBMWHA and assist in model validation and improvements.

Phytophthora cinnamomi was successfully isolated from all reserves except Jenolan Karst CR and Thirlmere Lakes NP during the surveys. Both the Blue Mountains NP and the Wollemi NP yielded 143 positive samples representing a 22.5% isolation rate from 635 samples collected in the Blue Mountains NP, compared to 12.5% from 1,069 samples collected from the Wollemi NP. Seventeen positive samples were collected from Yengo NP, two from Kanangra-Boyd NP, and one from Natti and Gardens of Stone NP each. Out of 142 samples collected off-park, 22 were positive. The sample allocation and distribution of positive results across each of the reserves is shown in Figure 3.4. The overall isolation rate for the whole survey was approximately 15%.

In addition to *P. cinnamomi*, 9% of sites were infected with *Phytophthora* species across the whole survey with a constant 5-10% isolation from each reserve. A large number of unidentified *Phytophthora* species was collected from the Wollemi NP (85 isolations, 9% isolation rate) and the Blue Mountains NP (72 isolations, 11% isolation rate). *Phytophthora* species were also collected from Yengo NP (9 isolations), Kanangra-Boyd NP (7 isolations), Natti NP (6 isolations) and 18 isolations from samples collected outside the GBMWHA.

The ratio of isolation of *Phytophthora* species between the reserves was fairly similar to the ratio of allocated samples (Figure 3.4), except for the Blue Mountains NP from which approximately one quarter of all samples for the whole survey were taken, yet nearly half of all the *Phytophthora* isolates were found.

The results of the Moran's I indicated that spatial autocorrelation between samples began to decrease after 840 m. Of the 1,980 sample results used in the Getis-ord Gi* analysis (excluding the *P. spp*), 724 samples occurred in statistically significant clusters (P = 0.05). Hot clusters (ie, where *P. cinnamomi* was likely to occur) where found mostly in the Wollemi and Blue Mountains NP, with a few located in the Yengo and Kanangra Boyd NP. The statistically significant hot clusters included 312 samples of which *P. cinnamomi* was not isolated from 108 sites. Cold clusters were identified in the northern most section of Wollemi NP and one in the centre of the Blue Mountains NP. These included 412 samples from which only two sites yield *P. cinnamomi*.

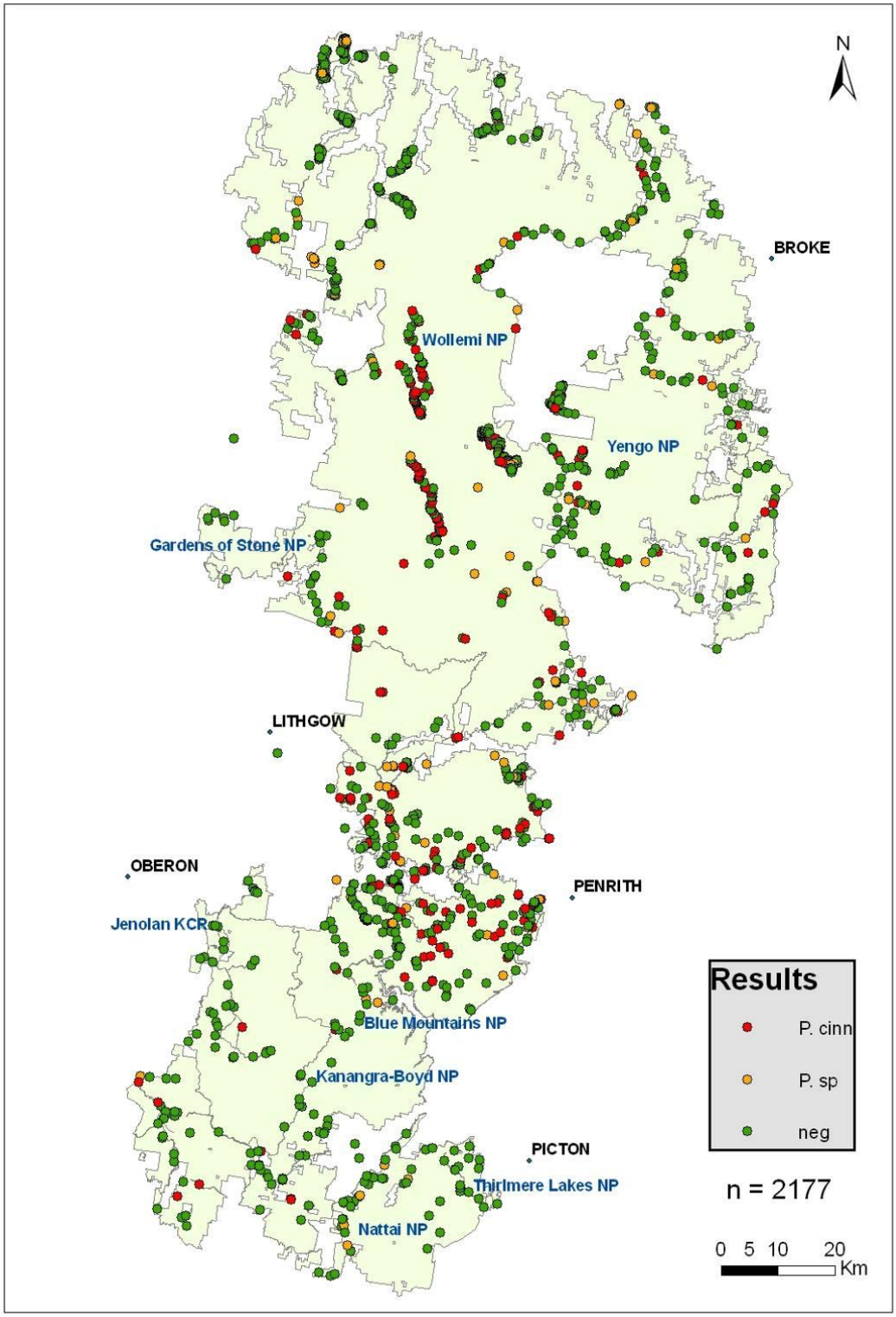


Figure 3.3 Distribution of *Phytophthora* across the GBMWHA determined from samples collected between 2010 and 2012. Results indicate where samples have been collected across the GBMWHA and are negative (green), positive for *P. cinnamomi* (red) or another *Phytophthora* species (yellow).

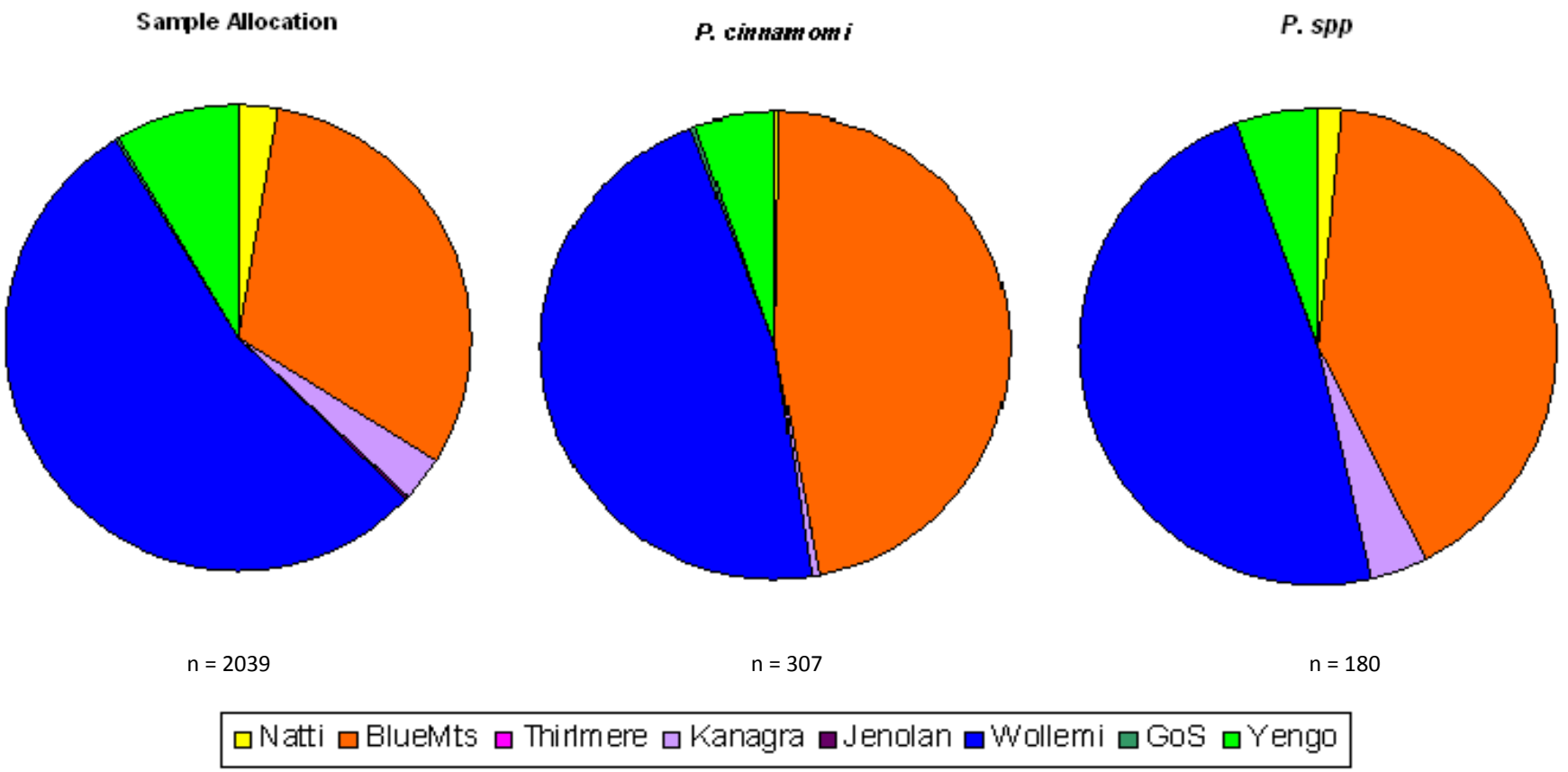


Figure 3.4 Sample allocation and isolation results across each reserve. The graph on the left illustrates the proportional distribution of samples across each reserve, while the graphs in the middle and on the right show the proportional distribution of samples positive for *P. cinnamomi* and other *Phytophthora* species respectively. The isolation graphs shows that most positives soil samples came from the Wollemi NP and the Blue Mountains NP, however these also had the highest number of samples collected from them, that is, the rate of isolation was proportional to the number of samples collected and was thus fairly constant.

Positive and negative samples were intermixed throughout the survey. It was not uncommon to find three positive samples mixed among ten negative samples along the one track. In such cases *P. cinnamomi* was not limited to the start of the track, but anywhere along the transect. If an area was sampled, *P. cinnamomi* was found mixed among the negative samples and in the case of the high risk locations, could be found within meters of negative results. Additionally, results were mixed regardless of whether or not a track or trail was publicly accessible to cars, restricted to foot traffic, or entered via private property.

Phytophthora cinnamomi was frequently isolated from tracks and trails that were accessible to the public and less frequently from those that were restricted. For example, samples collected from the north-western section of Wollemi NP including Yarrawa Trail, Gallic Trail and Perimeter Trail (Figure 3.5) returned no samples positive for *P. cinnamomi*. Access to the GBMWHA via these areas is through private property, or on foot. This is contrast to areas such as Bulga Trail and Hunter Main Range on the north-eastern side of Wollemi NP (Figure 3.5) from where *P. cinnamomi* was frequently isolated. These areas are publicly accessible and are frequently used by four-wheel drivers and trail bike riders alike. Natti NP is not publicly accessible, except on foot and only on selected trails. Only one site contained *P. cinnamomi*, which was found on previously cleared land, on the West-4-D (W4D) trail that leads to sheds and accommodation for field staff (Figure 3.6).

Positive isolations were also readily returned from highly frequented areas. This was especially true for the Blue Mountains NP were popular tracks and trails at Blackheath, Katoomba, Leura (Figure 3.7) and Wentworth Falls all had high isolation rates. *Phytophthora cinnamomi* was also isolated from camp grounds, such those on Mt Solitary (Figure 3.8). *Phytophthora cinnamomi* was isolated from the full range of road distances, the mean of which was significantly different ($p = 0.000$) from those of *Phytophthora* species and negative samples which were not significantly different from each other ($p = 0.776$). As the sampling area was restricted to 500 m of roads, most samples fell within this window, however samples were collected as far as 10km from the nearest road during the Root Rot Trot. Negative results tended to occurred within 400 m of a road, but 75% of *P. cinnamomi* isolations occurred within 3.25 km of a road (Figure 3.9).

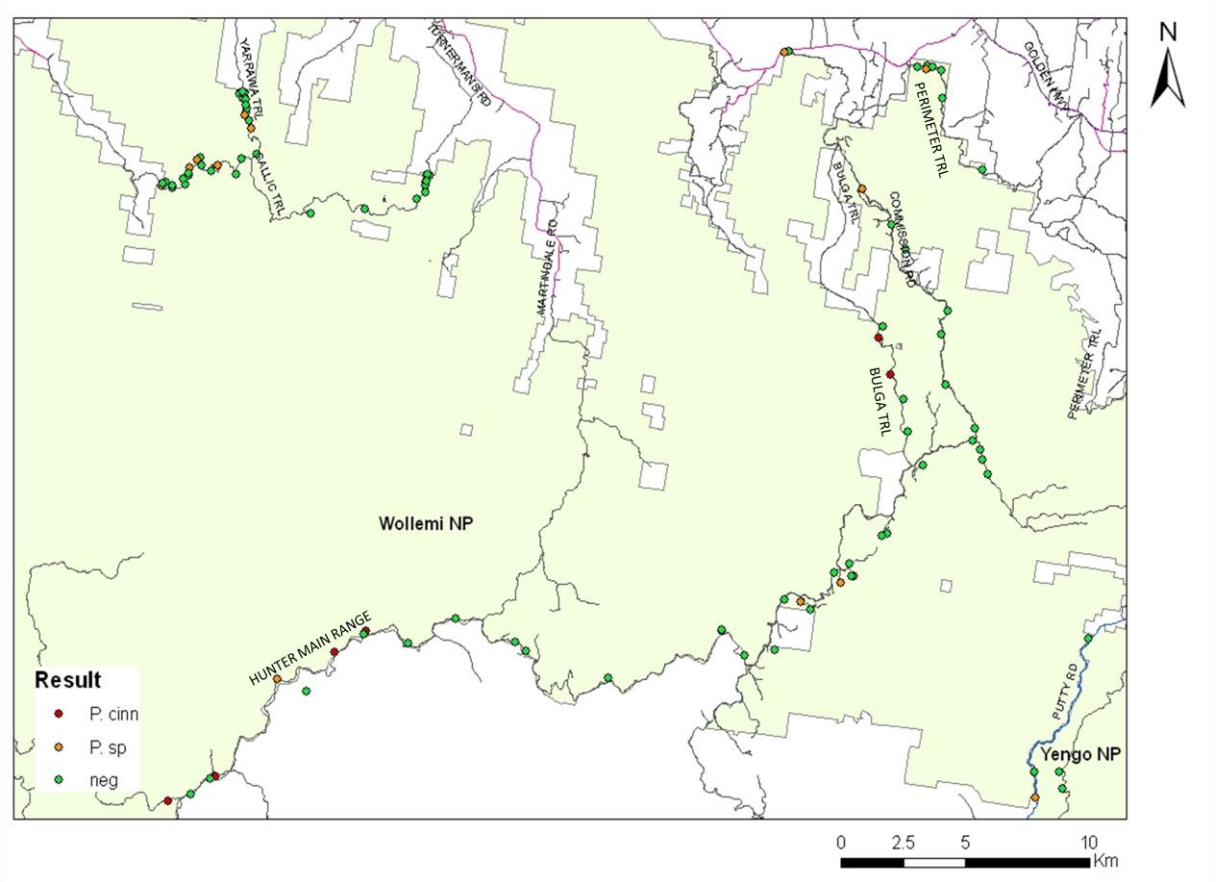


Figure 3.5 Sampling results along public and private access corridors through the Wollemi NP. Gallic, Yarrawa and Perimeter Trails each of which is private, returned no samples positive for *P. cinnamomi*, however it was isolated from public trails in this example including Bulga Rd and Hunter Main Range.

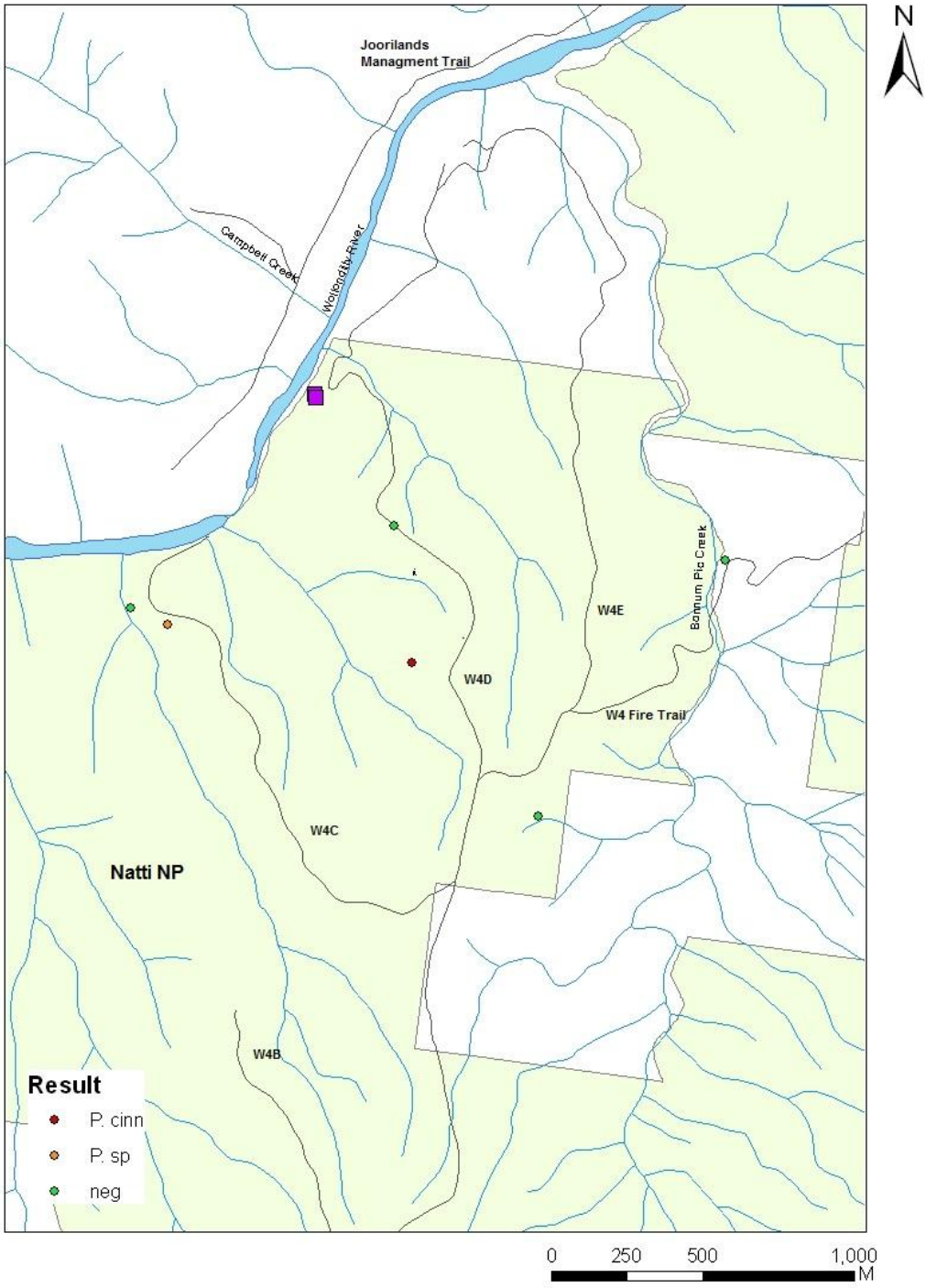


Figure 3.6 Location of the positive isolation of *Phytophthora cinnamomi* made in the Natti NP. The single positive result collected from Natti, occurred on the W4D Trail which had a number of sheds (purple squares) and staff accommodation at its end.

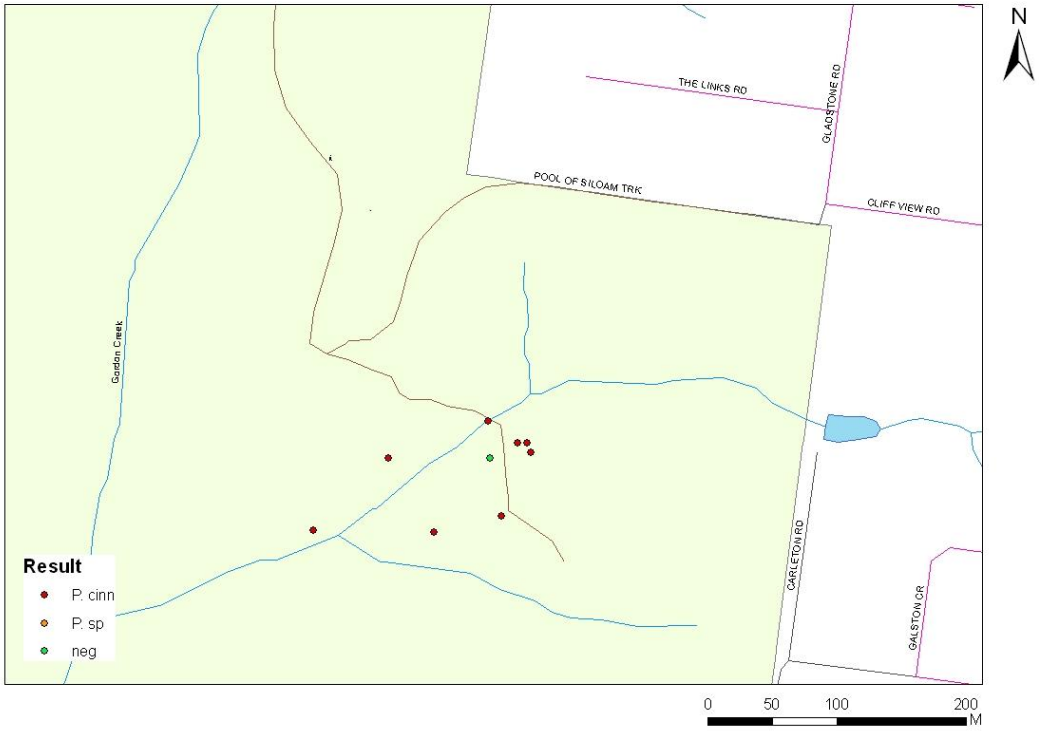


Figure 3.7 Samples collected near the Pool of Siloam Track, Blue Mountains NP. From the nine samples collected at this location, *P. cinnamomi* was isolated from eight.

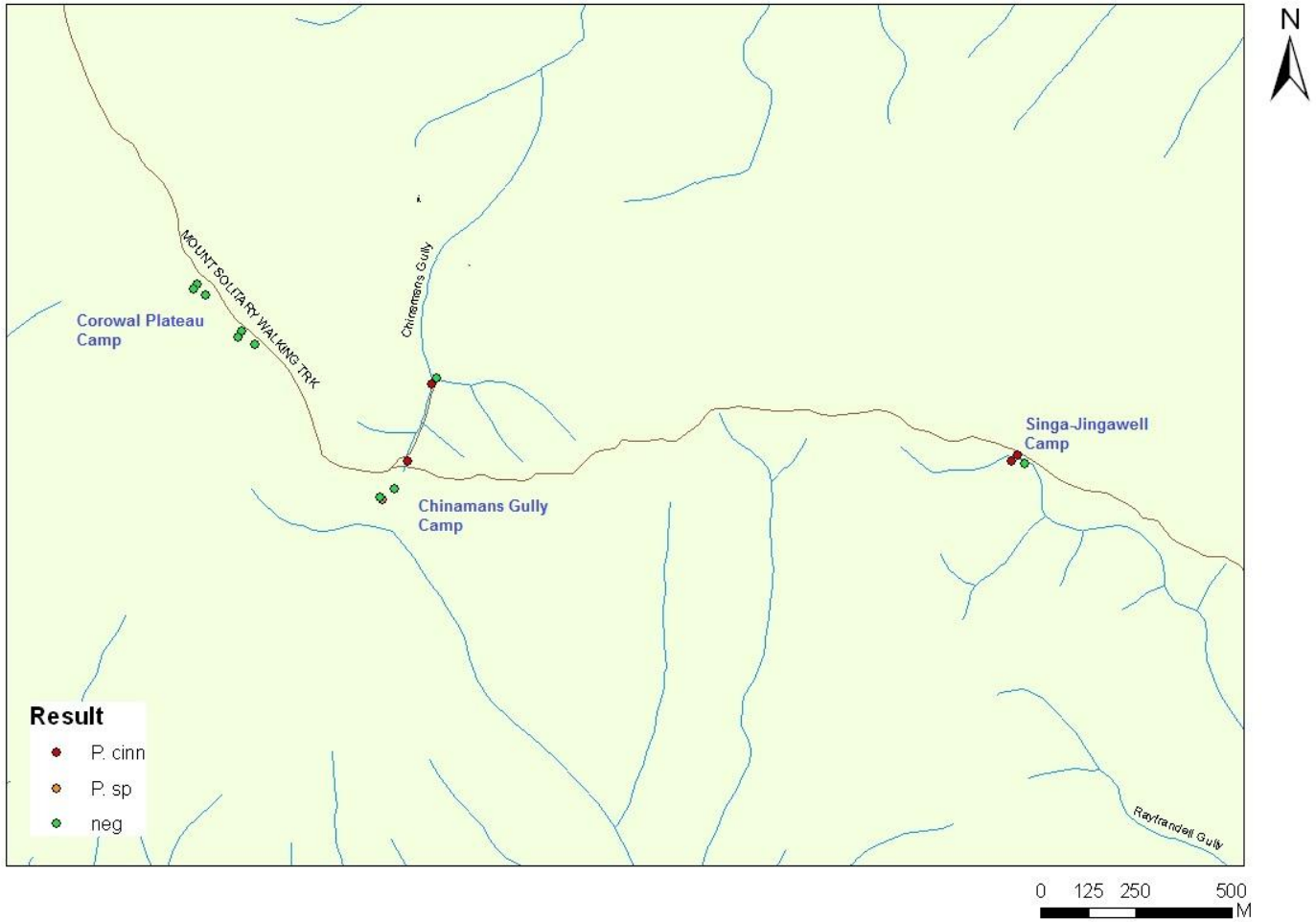


Figure 3.8 Soil sampling locations at the Camp Grounds on top of Mt Solitary, Blue Mountains NP. *Phytophthora cinnamomi* was isolated from both Chinamans Gully camp site and Singa-Jingawell camp sites.

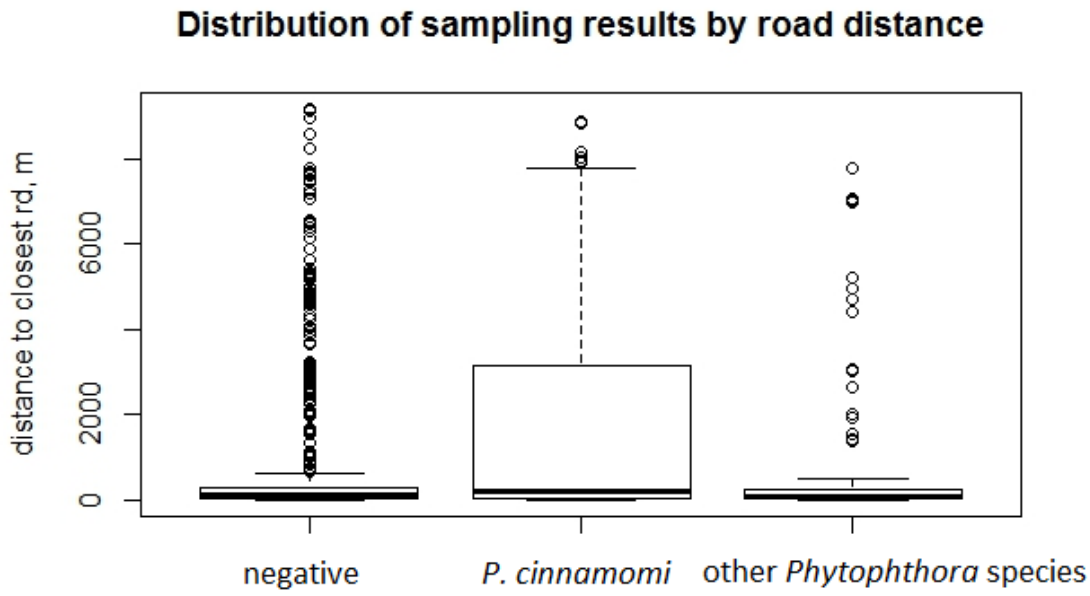


Figure 3.9 Sampling results with respect to distance from roads. Positive and negative result were acquired from as far as 10 km from the nearest road, however, *P. cinnamomi* isolations were collected from a larger range of distances from roads.

The samples collected on the Root Rot Trot were among the most remote of any samples collected over the two seasons. Deep within the Wollemi Wilderness, the four collection teams were helicoptered into the area and delivered to old fire trails or isolated ridges, from where they then spent five days hiking through the wilderness collecting up to 80 soil samples per team. Individually, the samples collected during the trip achieved a 40% isolation rate which was higher than the average isolation rate for both the Wollemi and Blue Mountains NP as well as the overall average for the whole survey. These samples were selectively taken from disease locations instead of randomly and therefore can be expected to have a higher rate of *P. cinnamomi* isolations. This also means that the rate of isolation cannot be statistically compared to the rate of isolation for the rest of the survey because the sampling method biased the results. These soil samples, however, demonstrated an intermixed pattern of positive and negative isolation along each route that didn't appear to be associated with any specific environmental variable such as slope, wetness or distance to roads.

3.3.2 Isolation of *Phytophthora* from specific environments

The distribution of *P. cinnamomi* and other *Phytophthora* species was compared to a series of environmental variables to address which might be influencing its spread. A series of regional vegetation surveys have recently been amalgamated to generate a consistent layer of 'vegetation formations' across the GBMWHA (Hammil and Tasker, 2010) using the vegetation classification system established by Keith (2004). Each specific formation is characterised largely by its structure and the structure of dominant taxa, however individual plant species will occur more frequently in specific formations. Nine vegetation formations have been identified across the GBMWHA which include sclerophyllous types, rainforest, heathlands, wetlands, woodlands and grasslands. This information was used to assess if *P. cinnamomi* was common in specific environments (Table 3.2). Chi-squared analysis revealed that *P. cinnamomi* was isolated at significantly different rates from each of the nine vegetation classes (χ^2 = 166.63, df=24, $p < 0.05$). *Phytophthora cinnamomi* was isolated from 33% of samples taken from the Heathlands formation. This included areas such as Mt Banks and Mt Hay in the Blue Mountains NP. In the Freshwater Wetland the isolation rate of *P. cinnamomi* was 28.6%. Most samples were taken from the Dry Sclerophyll Forest (Shrubby subformation) (1,407 samples), which had an isolation rate of 18%. Grassy Woodlands and Cleared lands had isolation rates of 0.9% and 0.5% respectively. These results were different to the isolation of *Phytophthora* species

which were found in 35% of samples collected from the Freshwater Wetlands and 17% from the Wet Sclerophyll Forest (Grassy subform).

Temperature significantly influenced the isolation frequencies of *P. cinnamomi, Phytophthora* species, and negative results (p = 0.000), the means of which were also significantly different from each other. *Phytophthora cinnamomi* was associated with specific temperature maxima and minima such that it was more likely to be isolated from cooler sites in the GBMWHA. The median temperature of *P. cinnamomi* isolations occurred at a minimum (minimum temperature of the coldest period) of 13˚C, and maximum (maximum temperature of the warmest period) of 26˚C. For other *Phytophthora* species these values were 14˚C minimum and 27˚C maximum (Figure 3.10).

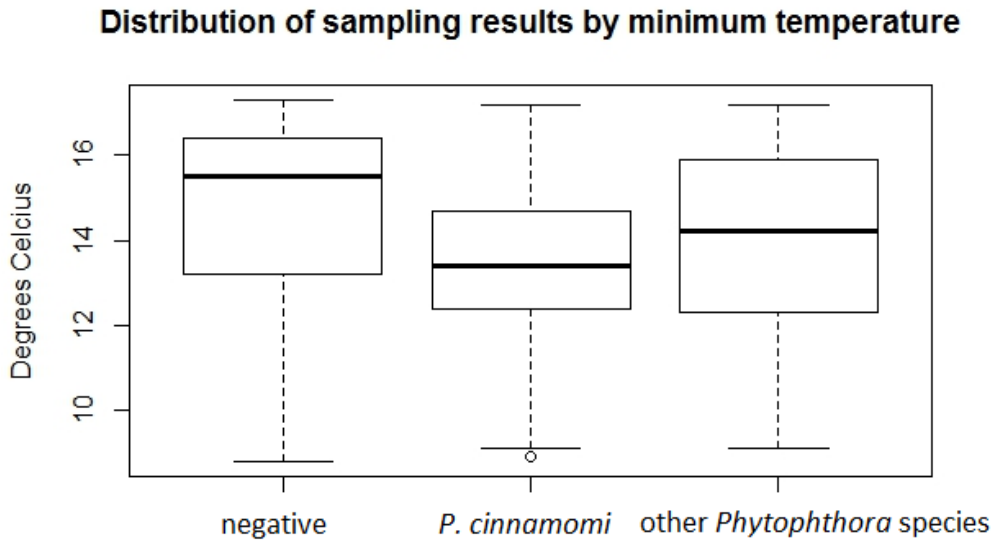
Annual rainfall also influenced the distribution of *Phytophthora* across the GBMWHA (p = 0.000), such that positive samples occurred in higher rainfall areas. Each interaction was also significantly different (p = 0.000) indicating that *P. cinnamomi, Phytophthora* species, and negative sites occurred at different ranges of annual rainfall. Samples were collected from within 600 to 1,400 mm per annum (mmpa) with fifty percent of the *P. cinnamomi* tending from the range of 850 mmpa to 1,200 mmpa and 750mmpa to 1,200 mmpa for other *Phytophthora* species (Figure 3.11).

Phytophthora cinnamomi was isolated from the same range of slopes that *Phytophthora* species and negative samples were attained from (p = 0.197). Most samples were collected from gradient of about 1:4 (approximately 15˚), but they were also collected on slopes as steep as 4:1 (approximately 78˚), with 75% of the data falling on a 1:1 grade (45˚) or less (Figure 3.11).

Table 3.2 Distribution of *Phytophthora* across vegetation formations.

Vegetation Formation	neg	*P. cinn*.	*Phytophthora* species	Grand Total	Isolation rate per veg formation, *P. cinn*.	Isolation rate per veg formation, *Phytophthora* species
Cleared Land	176	1	16	193	**0.5**	8.3
Dry Sclerophyll (Shrubby)	1,027	265	115	1,407	18.8	8.2
Dry Sclerophyll (Shrubby/Grass)	155	3	16	174	1.7	9.2
Forested Wetlands	16	3	4	23	13.0	17.4
Freshwater Wetlands	10	8	10	28	28.6	**35.7**
Grassy Woodlands	107	1	5	113	0.9	**4.4**
Heathlands	34	22	10	66	**33.3**	15.2
rainforest	18	2	1	21	9.5	4.8
Wet Sclerophyll (Grassy)	32	1	7	40	2.5	17.5
Wet Sclerophyll (Shrubby)	62	19	12	93	20.4	12.9
(Unknown)	14	4	1	19	21.1	5.3
Grand Total	1,651	329	197	2,177		

The vegetation formation present at each sample location was determined from the vegetation maps produced by (Hammil and Tasker, 2010). Highest and lowest isolation rates have been indicated in bold text.



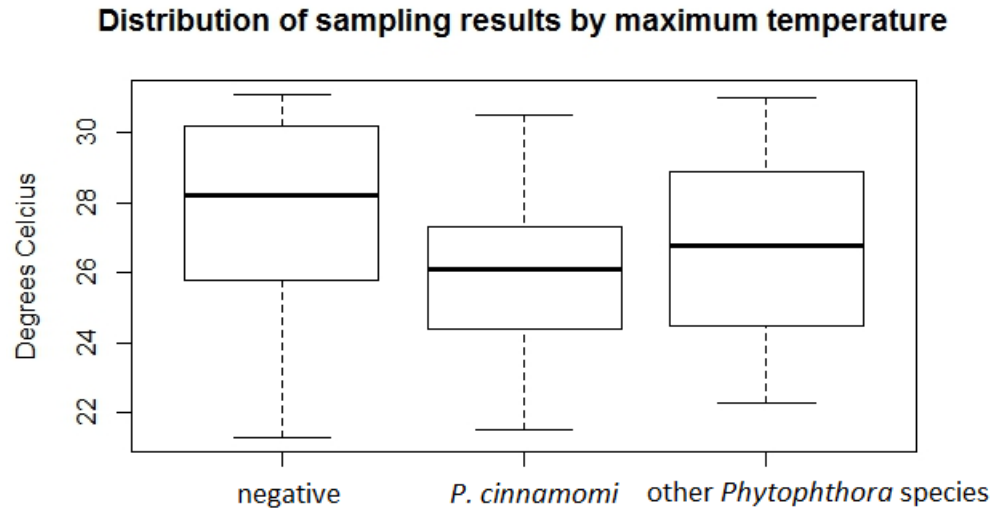


Figure 3.10 Box plots of isolation results as a function of minimum or maximum temperature. The box plots indicate that *Phytophthora* was more readily isolated, but not limited to, samples that were taken from locations with cooler temperatures.

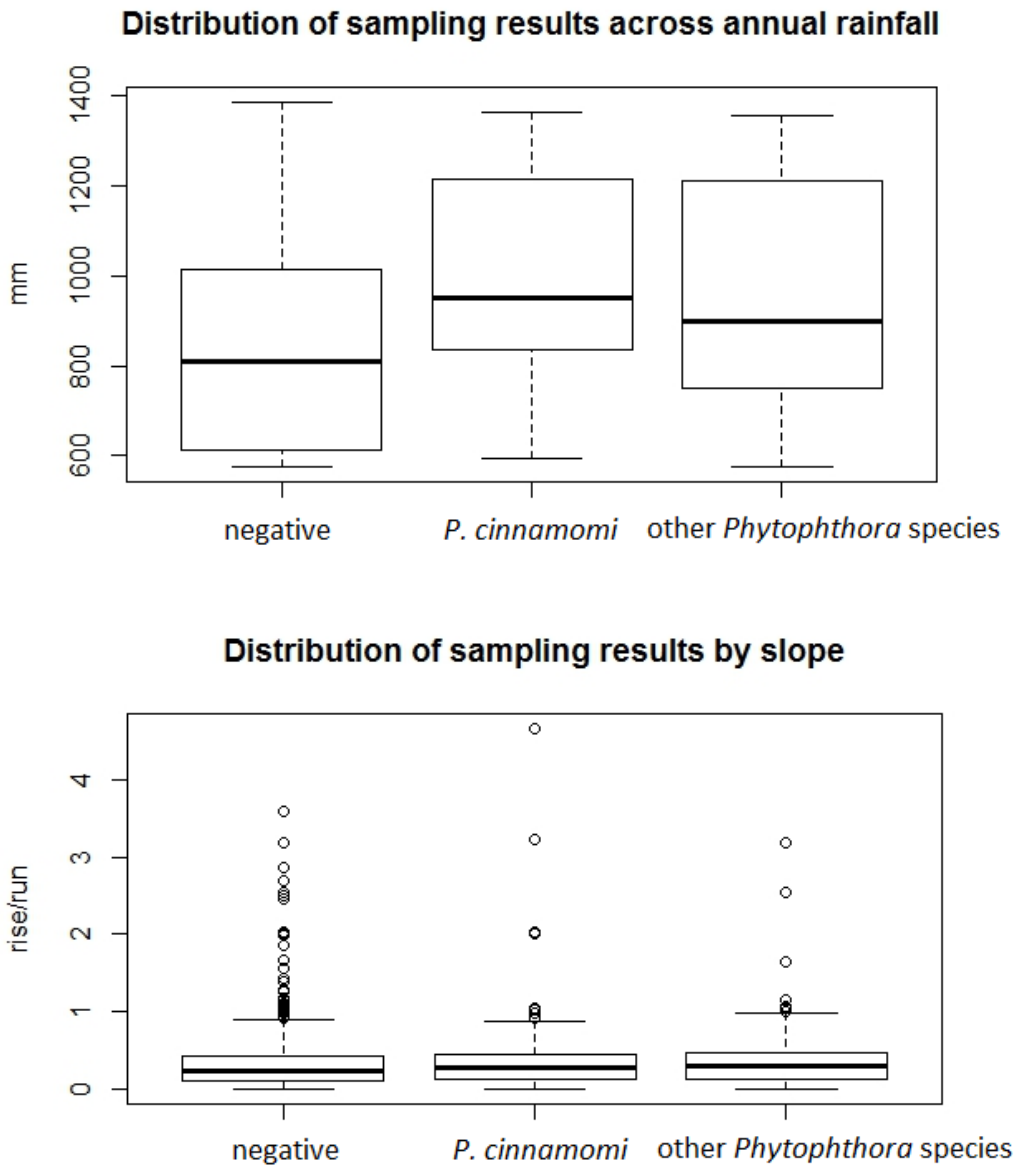


Figure 3.11 Distribution of sampling results across annual rainfall and slope. *Phytophthora* was more frequently isolated from locations with higher rainfall. Positive and negative samples were collected at similar rates from the full range of slopes sampled.

3.3.3 Isolation from risk areas

As discussed in the previous chapter, *P. cinnamomi* was isolated from each of the five risk strata, and was isolated at an increasing rate as the prediction of risk increased except in the fifth stratum. Areas that had over 85% risk and above occurred in one to four cell locations, that is, areas of approximately 625 m^2 to 2,500 m^2. Either *P. cinnamomi* or other *Phytophthora* species were isolated from each high risk location sampled, except Wentworth Falls.

3.3.4 Evidence of Phytophthora dieback in the field

The observed occurrence of Phytophthora dieback in the field was sporadic. Dieback was mostly observed in the Dry Sclerophyll (Shrubby) vegetation formation, where it was seen in both the undergrowth (Figure 3.12) and canopy (Figure 3.13). Dieback, if evident, was often cryptic and might only present as a slight reduction in the canopy density, with the loss of 2-3 individual plants. Such patches ranged in size from an area of approximately 10 m^2 to 200 m^2. Clear boundaries between what might be diseased and infected vegetation were difficult to interpret as differences in the density of ground cover and understorey were gradual or unaffected. In few cases, the health of vegetation could be seen to change in a matter of metres.

The occurrence of dieback was not limited to locations close to human activity. It was also identified in remote areas of the Blue Mountains and Wollemi NPs. Dieback was more obvious and more frequently associated with positive isolations in the Heathlands vegetation formation such as those found on Mt Banks and Mt Hay in the Blue Mountains NP. It could be observed that all individuals of specific species were affected or species that are known to be highly susceptible to *P. cinnamomi* were found dead alongside unaffected individuals (Figure 3.14). When dieback was observed, a variety of different habits were symptomatic from ground covers right through to dominant canopy species. The families in which plants were typically affected included Myrtaceae, Proteaceae, Epacridaceae, Xanthorrhoeaceae, and Fabaceae. These and other individuals identified showing symptoms of dieback have been recorded in Table 3.3 and Figure 3.15 - 3.18. Where possible, plant samples were collected for tissue isolation, however *P. cinnamomi* was infrequently recovered.

Figure 3.12 Examples of suspected Phytophthora dieback in the understorey of Dry Sclerophyll Forests within the GBMWHA. Dieback can be seen in each of these locations from where *P. cinnamomi* was isolated. In each case, dead plants are intermixed with living and much of the canopy remains unaffected. The top right photo was taken by Diedree Noss.

Figure 3.13 Evidence of Phytophthora dieback in the canopy of Dry Sclerophyll (Shrubby) vegetation formation. *Phytophthora cinnamomi* was isolated from samples that came from both of these sites. The left photo was taken by Diedree Noss.

Figure 3.14 Expressions of Phytophthora dieback in heathland vegetation communities. The image on the left is from a site *P. cinnamomi* was isolated from at Mt Banks, Blue Mountains NP. Each individual of the one species is seen dying here. This is contrast to death of some individuals alongside living ones as is seen in the Epacrids in the right image taken at Mt Hay, Blue Mountains NP.

Table 3.3 Plants suspected to be suffering Phytophthora dieback with the GBMWHA.

Family	Species	*P. cinnamomi* isolated?
CASUARINACEAE	*Allocasuarina nana*	Not tested
	Allocasuarina torulosa	Not tested
CYPERACEAE	*Caustis flexuosa*	Not tested
EPACRIDACEAE	*Epacris microphylla*	Yes
	Leucopogon esquamatus	Yes
	Sprengelia incarnata	No
	Styphelia nuerophylla	Yes
FABACEAE	*Dillwynia sericea*	Not tested
	Phyllota phylicoides	Not tested
	Pultenaea scabra	Not tested
	Pultenaea tuberculata	Not tested
GOODENIACEAE	*Goodenia sp*	No
MYRTACEAE	*Angophora costata*	Not tested
	Corymbia gummifera	Not tested
	Eucalyptus piperita	Not tested
	Eucalyptus sieberi	Not tested
	Leptospermum trinervium	Not tested
PROTEACEAE	*Banksia serrata*	Not tested
	Banksia spinulosa	Not tested
	Grevillea buxifolia ssp phylicoides	Not tested
	Hakea dactyloides	Not tested
	Isopogon anemonifolius	Not tested
	Persoonia sp	Yes
RHAMNACEAE	*Cryptandra ericoides*	Yes
SELAGINELLACEAE	*Selaginella uliginosa*	No
XANTHORRHOEACEAE	*Xanthorrhoea sp.*	Not tested
	Xanthorrhoea sp. Broad leaf	Not tested
ZAMIACEAE	*Macrozamia sp*	Not tested

These plants have shown typical symptoms of dieback in locations where *P. cinnamomi* was isolated from the soil. *Phytophthora cinnamomi* has not been isolated from many of the plants listed and therefore their symptoms cannot be directly attributed to *P. cinnamomi*.

Epacris microphylla

Leucopogon esquamatus

Sprengelia incarnata

Figure 3.15 Dieback observed in species of Epacridaceae in the GBMWHA.

Phylotta phylicoides

Pultena scarbra

Pultena tuberculata

Figure 3.16 Dieback observed in species of Fabaceae in the GBMWHA.

Banksia spinulosa

Grevillia buxifolia

Hakea dactyloides

Isopogon anemonifolius

Figure 3.17 Dieback observed in species of Proteaceae in the GBMWHA.

Caustics flexuosa (Photo: Deidree Noss)

Xanthorrhoea sp – narrow leaf

Xanthorrhoea sp – broad leaf (Photo: Ian Brown)

Figure 3.18 Dieback observed in species of Cyperaceae and Xanthorrhoeaceae in the GBMWHA.

Dieback could also be observed in areas that had previously or were still being disturbed. Means of disturbance in the GBMWHA included the presence of roads or walking tracks, areas from which vegetation had been removed, or where rubbish was present (Figure 3.19). Although the cause of death of individuals at these locations was not directly assessed, *P. cinnamomi* was isolated from each site and in some cases was isolated from the opportunistic plant samples collected during the survey.

Other types of disturbance that may have been impacting the expression of disease included the involvement of other pest and pathogens. Although not directly tested, it appeared that *Armillaria* infection was present at some dieback sites while at others Bell Miners could be heard, indicating the likelihood of Bell-minor associated psyllid dieback.

Figure 3.19 Examples of different types of disturbance that were found on sites displaying dieback in the Blue Mountains NP. Dieback was observed along sides roads as is seen in a) along Victoria Falls Rd. It could also be found along walking tracks such as Cliff Top Track, b). The Kent Street Fire trail Bullaburra, c), shows evidence of tree removal or logging and at Lapstone, an old metal water tank seen in the vicinity of dying trees d). *Phytophthora cinnamomi* was isolated from all of these locations.

3.4 Discussion

Phytophthora cinnamomi has been found right across the geographic extent of the GBMWHA, and found to be causing disease in several locations. It ranges from the north of the Wollemi NP to the southern end of Blue Mountains NP, from Yengo NP and Natti NP in the east across to Garden of Stone NP in the west. *Phytophthora cinnamomi* has been isolated readily from the boundary between the reserves and suburban areas, as well as in the most isolated and inaccessible parts of the Park. The only reserves from which *P. cinnamomi* was not isolated was Jenolan-Karst CR and Thirlmere Lakes NP. It was, however, isolated from Thirlmere Lakes during the survey previously conducted by the Royal Botanic Gardens (Suddaby, 2008a). These results must still be taken cautiously as the soil baiting technique used here does not always successfully isolate the pathogen leading to a potential underestimation of the distribution of *P. cinnamomi*. The potential for false negative results is evident in the occurrence of numerous statistically significant hot spots from which *P. cinnamomi* was not isolated. The distribution of *P. cinnamomi* within the GBMWHA is widespread, and likely to have a patchy distribution throughout the soil. Clustering of samples also indicates that a much larger portion of the GBMWHA is suitable to *P. cinnamomi* than that which is not. Such a wide distribution of *P. cinnamomi* is not surprising given its range along the east coast of Australia and the period of European activity within the area dating back 200 years.

From the samples collected, *P. cinnamomi* was only present in approximately 15%. This isolation rate is not surprising in NSW where the isolation of *P. cinnamomi* in climatically suitable environments is known to be sporadic due to low inoculum levels (Walsh *et al.,* 2006; Howard, 2008), possibly as a result of microbial competition in the soil (Marks *et al.,* 1975). There was however areas where the isolation of *P. cinnamomi* increased above the survey average. This included highly frequented parts of the GBMWHA such as the short walking trails in the upper Mountains and publicly accessible roads throughout the park. The Blue Mountains NP is the most highly visited reserve within the GBMWHA and this has inevitably aided in nearly doubling the isolation rate when compared to the average for the whole WHA. It seems certain that *P. cinnamomi* is being spread largely by human activity.

The identification of a 40% isolation rate in the Wollemi Wilderness was quite concerning as it was hoped this might be one part of the GBMWHA that would remain free of the impacts of Phytophthora dieback. As the most remote part of the GBMWHA, the

extensive distribution of *P. cinnamomi* across the ridges and gullies of the Wollemi Wilderness was quite puzzling. Although not frequently entered today, the opportunity for humans to have introduced *P. cinnamomi* into this part of the park with logging and mining activity dates back many decades. Evidence of stray cattle, rock cairns, and fire pits were identified during the remote trips, suggesting that people have entered into these parts of the park in the past and still do today. Those that do enter most frequently may in fact be management staff on activities such as fire fighting, weed and feral animal control. Although staff are well aware of the need to maintain good hygiene, it has been suggested that staff may inadvertently spread *P. cinnamomi* during scientific research, or during fire fighting activities when hygiene becomes a secondary priority (C. Baker, NSW NPSW, Pers. comm.). Such activities occur anywhere within the park, which may explain why *P. cinnamomi* was isolated extensively from the Wollemi Wilderness. The occurrence of *P. cinnamomi* in the Natti NP on the W4D fire trail is possibly also a direct consequence of staff activities as the area is not accessible to the public at all, but is used by staff to reach sheds and accommodation at the end of the trail.

Aside from the natural spread of *P. cinnamomi* and that caused by humans, feral animals likely have a role in the movement of infested soil also. Feral pigs have been associated with the spread of *Phytophthora* in Queensland due to their rooting and wallowing behaviour (Brown, 1976). Feral pigs occur at a high density within the Kanangra-Boyd NP and the southern sections of the Blue Mountains NP, and a medium to low density across all other reserves (Department of Environment and Climate Change NSW, 2007; Department of Environment and Conservation NSW, 2008). Although *P. cinnamomi* does not appear to be widely distributed in the southern section of the GBMWHA, feral pigs have the potential to spread *P. cinnamomi* more extensively. Additional feral animals that may have a contributory role in the spread of *P. cinnamomi* that are found within the WHA include cattle, deer, wild dogs and foxes, goats, horses and rabbits (Department of Environment and Climate Change NSW, 2007; Department of Environment and Conservation NSW, 2008). Each of these animals causes soil and vegetation disturbance, potentially resulting in the spread of *P. cinnamomi* or the creation of additional plant stress, exacerbating the disease.

Evidence of dieback symptoms existed throughout the GBMWHA, but was especially clear in the Wollemi Wilderness. Dieback sites were frequently identified and the range of habits affected included the herbaceous layer, the understorey and the canopy. The impact of *P. cinnamomi* in the Wollemi Wilderness was often severe in comparison to the majority of symptoms observed in the rest of GBMWHA. Typically, plants highly

susceptible to *P. cinnamomi* will be killed early in the invasion process leaving only those with greater resistance behind (Weste, 2003), resulting in a less overt expression of disease in an individual site. As a result, the rate of isolation decreases with the length of the infestation (McDougall *et al.,* 2001; Weste, 2003). Thus, frequently observed dieback in combination with a comparatively higher isolation frequency suggest that *P. cinnamomi* has likely to have been introduced into the Wollemi Wilderness at a date later then the majority of the GBMWHA.

Phytophthora dieback was identified in localities right across the GBMWHA, however positive isolations of *Phytophthora* were frequently associated with healthy vegetation rather than diseased. There may be several reasons for this including a suppressive nature of the soil, field resistance in many hosts resulting in asymptomatic infection, or environmental conditions which are conducive to the pathogen but not necessarily to disease. Microbial inhibition of *P. cinnamomi* resulting in a lack of disease has been observed in both natural and agricultural environments in Australia (Broadbent and Baker, 1974; Weste and Marks, 1987). If the soils of the GBMWHA are in fact inhibitory, *P. cinnamomi* may be inclined to spread via root-to-root contact which can result in a mosaic of dieback (Weste and Marks, 1987). This insidious behaviour of *P. cinnamomi* in NSW has been observed repeatedly (Arentz, 1974; Walsh *et al.,* 2006; Howard, 2008), and is anticipated from eastern Australian vegetation communities.

The survival of *P. cinnamomi* as a biotroph has recently been demonstrated in a number of Western Australian species, many of which had no or limited indication of disease (Crone *et al.,* 2013a; Crone *et al.,* 2013b). A lack of dieback observed in the GBMWHA from areas in which *P. cinnamomi* has been isolated may be for similar reasons. Inspection of plants would be required to establish if such a relationship is occurring. Additionally, in NSW, native species are suggested to have a greater resistance to disease (McCredie *et al.,* 1985; Suddaby *et al.,* 2008), which may also explain a lack of evidence of disease.

Perhaps the most likely reason for the lack of disease expression, however, is that although environmental conditions of the GBMWHA are suitable for the establishment of *P. cinnamomi*, or hot and dry summers which often triggers disease (Weste and Vithanage, 1978; Weste and Marks, 1987), are absent, or not severe enough to cause stress.

The impacts of *P. cinnamomi* that were observed within the GBMWHA were strongest in plants of the herbaceous layer which typically includes species of Epacridance, Fabaceae, Myrtace and Proteaceae. These same plant families have been found affected in The Royal National Park (Walsh *et al.,* 2006) and other parts of Australia (Newhook and

Podger, 1972; Weste, 1994). Many of the plants found to be affected were herbaceous vegetation in the heathland complexes of the upper mountains where evidence of disease coincided with a high rate of positive isolations. These fragile communities occur on exposed ridges with skeletal, low nutrient, sandy soil, have a high wind and fire exposure and are frequently inundated following heavy and/or continuous rainfall. Ironically, these harsh conditions make heathland communities highly diverse and an important food source for many small herbivores (Hammil and Tasker, 2010). The occurrence of Phytophthora dieback in heathlands is distinct such that dead plants can be seen to occur frequently amongst the living and disease margins, not normally observed in NSW are evident (McDougall *et al.,* 2003). Unlike the remote areas, the heathland communities such as Mt Banks and Mt Hay are frequently visited. As a result, the introduction of *P. cinnamomi* into these areas may have occurred many decades ago. The pathogen appears to be quite active, which suggests that infection is more recent (Weste, 2003). A likely cause may be that many of the susceptible herbaceous plants have the ability to set seeds before they succumb to disease, so dieback occurs frequently with each new generation sustaining the pathogen as well as their own population. With a constant supply of highly susceptible hosts and an ability to survive intermittent periods without them, Phytophthora dieback could go on killing herbaceous plants and creating blatant dieback in heathland communities indefinitely.

Disease expression in the Dry Sclerophyll vegetation formation was markedly different, and more typical of the response of NSW vegetation. Without close inspection, dieback in the dry sclerophyll forests of the GBMWHA, although widely distributed, could go unnoticed. Slight reductions in canopy density and branch death as well as two to three dead individuals were often the only telltale sign that *P. cinnamomi* was present. This is quite unusual in that it is the dry sclerophyll vegetation that is typically most affected by *P. cinnamomi* in Australia, where Mediterranean climates dominate (Weste and Marks, 1987). Periodic rainfall throughout the year, may be enough to stave off the impacts of dieback resulting in asymptomatic host (Weste and Marks, 1987). Sites that were clearly affected ranged in size from approximately 10-100 m^2, and the margin between healthy and diseased vegetation was inconspicuous. The only time dieback appeared more obvious was when disturbance was present, such as along the edge of roads, or where possible soil contamination has occurred, the latter of which has been shown to exacerbate disease (Scarlett *et al.,* 2012). The identification of minimal impact on the most dominant vegetation community of the GBMWHA is encouraging as it suggests a natural resilience in the dry sclerophyll forests, or perhaps as previously reported, greater resilience in the

vegetation of eastern Australia in general (Suddaby *et al.,* 2008). Disease expression in the GBMWHA is markedly different to the occurrence of disease in native ecosystems in other parts of Australia such as Victoria, Tasmania and southern Western Australia where 50-75% of plants may be lost (Weste and Marks, 1987). Although less devastating disease is reported here, it does not mean that land managers should be any less concerned about the potential impact of *P. cinnamomi* as there is much we don't yet understand such as the potential impact of climate change or the association between Phytophthora dieback and other pests and diseases.

Destruction of trees in a manner similar to that caused by *Armillaria* infection was observed at times in the field. *Armillaria lutenobubalina*, known to be highly pathogenic on Eucalyptus in Australia (Podger *et al.,* 1978), has been observed infecting trees in areas where *P. cinnamomi* was suspected to be the causing decline on the Swan Coastal Plain in Western Australia (Shearer and Dillon, 1996). Unfortunately, once the authors isolated *A. lutenobubalina*, no further investigation was reported of the possible co-occurrence of *P. cinnamomi*, only that *P. cinnamomi* was isolated from disease sites in surrounding areas. Plants co-infected with *Armillaria mellea* and *P. cinnamomi* are known to suffer greater symptoms then that which would be caused by either pathogen in isolation (Marcais *et al.,* 2011). Taking both of these reports in to account, it may be possible that a disease complex develops between *A. lutenobubalina* and *P. cinnamomi* which should be investigated further. Additionally, Bell Miners (*Manoria melanophrys*) were often heard at sites presenting dieback in the canopy, suggesting that Bell Miner Associated Dieback (BMAD) may have a role in canopy destruction. Their possible association, has not yet been investigated. Whether *Armillaria* or BMAD are complicating Phytophthora dieback in the GBMWHA needs further investigation. Both could be considered to weaken the susceptible hosts in the first instance, collectively form a more destructive disease complex or be the cause of their final demise.

It is clear from the survey data that *P. cinnamomi* occurs in the GBMWHA in environments that are well know to sustain the pathogen across Australia. Both rainfall and temperature influence the distribution of *P. cinnamomi* in the GBMWHA. *Phytophthora cinnamomi* is reported to occur in areas where annual rainfall exceeds 600 mmpa, but it can be found as low as 400 mmpa (O'Gara *et al.,* 2005). The minimum rainfall required for isolation in the GBMWHA was 600 mmpa, however it was infrequently isolated until rainfall reach at least 850 mmpa. This relationship was not as strong in terms of temperature, as *P. cinnamomi* was isolated from a similar range of samples as those that returned a negative result. The distribution of rainfall and temperature in the GBMWHA are such that the

lowest rainfall is received in the winter months when temperature would be considered too low for pathogen establishment. In summer, however, temperatures are ideal, but rainfall is highest, meaning, that although conditions are conducive to pathogen spread and establishment, water stress that leads to dieback is less likely to occur.

Unlike rainfall and temperature, slope did not make a significant contribution to the distribution of *P. cinnamomi*. This is in contrast to distribution of *P. cinnamomi* in other areas of Australia (Wilson *et al.,* 2000; Keith *et al.,* 2012). The finding that *P. cinnamomi* is more likely to be found closer to roads suggests that *P. cinnamomi* has been introduced into the GBMWHA and is being spread via the road network. Other species of *Phytophthora* were also found close to roads highlighting the potential for not just *P. cinnamomi* but other species of *Phytophthora that* may also cause disease, to be spread along the road network. This is an important factor that needs to be dealt with in the management of Phytophthora dieback in the GBMWHA. These findings will also assist in the selection of environmental information to be included in future modelling work (Chapter 4).

The environmental limits that *P. cinnamomi* was significantly correlated with were slightly different to the other *Phytophthora* species that were isolated during the survey. Regardless of the reserves they were collected from, a consistent 5-10% isolation rate occurred across the GBMWHA. This suggests one of two things: that some of these species may be native and have adapted to a wide range of climatic and environmental conditions, or that, like *P. cinnamomi*, these species may have been introduced and have spread extensively across the GBMWHA also. The *Phytophthora* species were isolated from slightly warmer sites with 50% of isolations occurring between 14-27˚C, and at a slightly lower rainfall window of 750-1,000 mmpa. Again this suggests some of these species may be native as they have been isolated from locations with warmer and drier conditions typical of Australia. This is contrast to the tropical and wet origins of many *Phytophthora* species. A recent study exploring the molecular diversity of isolates of *Phytophthora* collected in native ecosystems in Western Australian identified nine undescribed taxa and nine recognised species (Burgess *et al.,* 2009). Although the origin of the undescribed species remains unknown, the authors suggest that at least one may be native to Western Australia highlighting the role that Australia has had in the evolution of *Phytophthora*. Further morphological identification and molecular analysis of the isolates collected here will be required to characterise the isolated *Phytophthora* species and explore their phylogenetic relationship within the genus. Caution must also be taken in terms of their management as they, like *P. cinnamomi,* may be introduced and causes of disease.

This information can now be used by land managers and researchers to target those parts of the landscape that appear most likely to sustain *P. cinnamomi*. This is especially true for the heathland vegetation communities that appear to be highly susceptible to the impacts of Phytophthora dieback. Special attention should be paid to these communities where management actions could include signage that alerts Park users to the presence of *P. cinnamomi* in these area advising that they need to be careful not to spread infested soil or cause any undue stress. Community awareness and education can coincide with staff education and training which is especially important here given the extensive opportunity for staff to spread *P. cinnamomi* during routine activities. Managers might also consider installing raised platforms such as those on the Penguin Walking Track in Sydney Harbour NP to cover sections of track that are known to frequently become water logged. This will reduce the spread of infested soil likely to have a high inoculum load (Weste and Marks, 1987). Monitoring the health and population size of vegetation communities and affected individuals should also be investigated to see what longer term impacts *P. cinnamomi* has on these areas, and if *ex-situ* conservation may be required should plant populations be dedining. Chemical control may not be a viable option even if it is shown to assist the health of specific plants, as many of the impacted areas form along ridge tops from where chemicals may leach into water ways or enter other sensitive vegetation communities. The application of chemicals would need to be considered very carefully. The role feral animals play in the spread of *P. cinnamomi* within the GBMWHA should also be investigated, as specific species may be responsible warranting an appropriate expansion of vermin control.

This is the first comprehensive survey of the distribution of *P. cinnamomi* across the GBMWHA ever conducted. It provides land managers with information on the position of *P. cinnamomi* and an insight into the impacts Phytophthora dieback is having on ecosystems. The results provide insight into the possible means by which *P. cinnamomi* is being spread about the park, the environmental conditions associated with its occurrence and the plausibility of management options under the current set of circumstances.

Chapter 4 A statistically-based risk model of disease caused by *P. cinnamomi* in the GBMWHA

4.1 Introduction

Biological invasion exerts great pressure on communities and ecosystems. Regardless of whether they are animals, plants, or microorganisms, invasive species can impact on the conservation of threatened species, reduce agricultural productivity, disrupt ecosystem processes and function, and have detrimental impacts on human health (Mack *et al.,* 2000; Crowl *et al.,* 2008). Global losses in agricultural production associated with invasive species are around 30%, however, without existing mitigation strategies, these losses may be in the order of 70% of total production (Oerke and Dehne, 2004). Profit losses are substantial due to the high cost of mitigation strategies; estimated to be $120 billion annually in the US (Crowl *et al.,* 2008; Vurro *et al.,* 2010). In natural environments, invasion can disrupt evolutionary processes, alter species niche dynamics, alter species abundances and dominance hierarchies, and ultimately cause extinction (Cronk and Fuller, 1995; Rhymer and Simberloff, 1996; Crowl *et al.,* 2008). It is clear that proactive prevention is preferable to reactive control, eradication, and remediation wherever possible (Mack *et al.,* 2000), but prevention requires detailed knowledge of the invasion process and is contingent upon the identification of potential invasion sites and the likelihood of impacts to vulnerable ecosystems (Reichard and Hamilton., 1997; Holdenrieder *et al.,* 2004). The concept of invasion risk, and its inherent uncertainty, is therefore at the heart of invasion prevention (Yang, 2006). Quantification and contextualisation of the risk of invasion is a vital tool in prevention strategies, requiring both traditional methods of observation and also novel approaches that identify the way that invasive species interact with their environment (Crowl *et al.,* 2008).

The difficulties in identifying invasion risk are most obvious when the invasive species is a pathogen. Disease resulting from invasive pathogens is a global problem that typically manifests differently from animal and plant invasion. Disease expression is usefully contextualised within the construct of the disease triangle; a basic concept of plant pathology that explains how disease is a function of i) a virulent pathogen, ii) a susceptible host, and iii) a conducive environment in which the pathogen and host coincide (Agrios, 2005). Changes in any one of these three entities will result in a different expression of disease favouring either the pathogen or the host. By establishing the relationship between

the pathogen, host and environment, it is then possible to estimate disease risk and simulate what might happen under different management or climatic scenarios (Ostfeld *et al.,* 2005; Yang, 2006; Jeschke and Strayer, 2008). This process is complex, and many models of disease risk struggle to quantify how the landscape influences the host-pathogen interaction (Plantegenest *et al.,* 2007), or fail to consider the distribution of hosts. A model that is constructed to explain the relationship between a host and a pathogen is only an epidemiological model, while a model that explains the distribution of a pathogen within an environment without incorporating information on the host(s) is only a species distribution model. Bringing these together represents a considerable but important challenge.

For example, a recent study on the likelihood of plant disease within North America modelled transmission risk from the distribution of the four most abundantly grown and susceptible agricultural crops (Margosian *et al.,* 2009). The authors concluded that the results could be utilised in policy development and planning of crop rotations to avert disease. Problematically, no consideration was given to role environmental variation may play on the ability of a pathogen to reach and survive at new locations across such a large geographic area. Similarly, the global distribution of Pitch Canker disease has recently been modelled from climatic data alone, with recommendations of continued strict quarantine measures for four regions identified as being potentially suited to the disease (Ganley *et al.,* 2009). There is no indication of consideration being given to whether or not the host, *Pinus spp.*, grows in the four identified regions. Similarly, a recent model of the potential distribution of *Puccinin psidii* (Eucalyptus Rust) within Australia was constructed by incorporating the way in which various climatic factors influence the host-pathogen relationship (Booth and Jovanovic, 2012). Although the model was presented as a distribution of areas vulnerable to pathogen spread, no consideration was given to the host distribution. As *P. psidii* is an obligate biotroph, hosts are essential for its ability to survive and spread regardless of environmental suitability. Approaches that estimate disease risk using the full spectrum of knowledge from the disease triangle are needed to enhance management and mitigation.

A suitable starting place is the pathogen *Phytophthora cinnamomi*, a plant pathogen of worldwide significance and included in the top 100 global plant diseases (Global Invasive Species Database, 2013). Believed to have originated in south-east Asia (Brasier, 1992) or South Africa (Linde *et al.,* 1997), *P. cinnamomi* is now found throughout Europe, Asia, Africa, America and Oceania and is defined by the European and Mediterranean Plant Protection Organisation (EPPO) as a quarantine pest (EPPO/CABI, 1997). Phytophthora dieback (the syndrome of disease caused by this pathogen in natural

ecosystems) is of great concern in Australia and was classified as a Key Threatening Process in 1999 (Commonwealth of Australia, 2005). The Mediterranean climate of much of Australia means there are suitable environments for the establishment of *P. cinnamomi* in Tasmania, the south western corner of Western Australia, around the northern, eastern and south-eastern coast line, and inland to the 400 mmpa (millilitres per annum) rainfall limit (O'Gara *et al.,* 2005). Importantly, with host range estimates approaching 3,000 species (Shearer *et al.,* 2004), the potential for the spread of the disease is considerable. Already, disease is devastating conservation areas that have been established to preserve biodiversity, such as the Stirling Ranges and Fitzgerald River National Parks of Western Australia (Wills, 1992; Barrett *et al.,* 2008; Shearer and Crane, 2011), the Grampians, Kinglake and Wilson Promontory National Parks of Victoria (Weste, 1974), the Narawntapu and Rocky Cape National Parks of Tasmania (Parks and Wildlife Service, 2000; Schahinger *et al.,* 2003), and the Sydney Harbour, Barrington Tops and Royal National Parks of New South Wales (McDougall *et al.,* 2003; Daniel *et al.,* 2006; Walsh *et al.,* 2006).

Successfully management of Phytophthora dieback does not solely depends upon an understanding of the pathogens distribution (O'Gara *et al.,* 2005), but also on the distribution of susceptible hosts; both of which should be expressed as functions of their environment. Yet the *Phytophthora* literature lacks examples of risk assessments incorporating all three elements of the disease triangle, possibly because these elements are considered separately, or one is assumed to be suitable while the other two are modelled. Disease risk, expressed as the potential distribution of *P. austrocedrae* in *Austrocedrus chilensis* forests in Patagonia was recently modelled without considering the distribution of *A. chilensis* itself (La Manna *et al.,* 2012). The distribution of *Rhododendron ponticum* has also been modelled as means of predicting the distribution of *P. ramorum* and *P. kernoviae* in the UK (Purse *et al.,* 2013). Although the model incorporated localities of inoculum sources, such as nurseries and gardens, no consideration was given to environmental influences on the pathogen distribution. In both of these cases, predictions of potential disease were likely to be exaggerated because unsuitable environments remained in the final model. In contrast, host distributions have been incorporated into spatial models of *P. ramorum* in the US (Kelly *et al.,* 2007) and *P. cinnamomi* in Australia (Keith *et al.,* 2012). In both cases, the susceptibility of individual hosts was incorporated, but disease risk was assessed for vegetation communities rather than individual species. While this may improve generalisations and applicability to on-ground management, the wide range of susceptibility among species may not be fully accommodated by this approach. Perhaps the best example of a *Phytophthora* model that incorporates the host,

pathogen and environment was developed by Vaclavik *et al.* (2010). They combined modelled distributions of individual species with a modelled pathogen distribution, however their approach to modelling individual hosts has only a reasonable ability to predict the distribution of vegetation accurately (Ohmann and Gregory, 2002) indicating there is opportunity to improve model performance.

To date, there are no models of the distribution of *P. cinnamomi* that incorporate host information at the species level which properly account for the disease triangle. In this chapter, a model of the Phytophthora dieback was developed for a globally significant conservation region in Australia, the Greater Blue Mountains World Heritage Area (GBMWHA). The aim was to determine whether a robust model of disease risk for *P. cinnamomi* could be developed and to evaluate whether a changing climate may alter the pathogens distribution. Data collected during the field survey (Chapter 3) and species location records were used to statistically model the distribution of *P. cinnamomi* and host susceptibly within the GBMWHA before combining them into a spatially explicit risk model of Phytophthora dieback. The sensitivity of the risk model to changing environmental conditions on the modelled pathogen distribution was also addressed.

4.2 Methods

The model of Phytophthora dieback risk was constructed by combining two separate models: the distribution of hosts and the distribution of *P. cinnamomi*. Both models were developed using Maxent and then compiled in ArcMap 9.3 (ESRI, California).

4.2.1 *Phytophthora cinnamomi* distribution

4.2.1.1 Layer selection and standardisation

A series of climatic, topographic and anthropogenic layers were obtained or constructed for model development (Table 4.1). A Digital Elevation Model (DEM), was obtained from the Office of Environment and Heritage, as well as layers for built-up areas, water bodies and the road network (OEH; OEH Data Broker, data.broker@environment.nsw.gov.au) As the distribution of *P. cinnamomi* is associated with human activity (O'Gara *et al.,* 2005), the Euclidian distance function in ArcMap was applied to the built-up areas layer to generate a risk surface representing decreasing risk away from the source. The roads layer was converted using the Kernel Density function with a 10km search radius. This output reflects the relative concentration of roads not just

their proximity. A slope layer was developed using the DEM to assess the association between *P. cinnamomi* and topographical steepness due to pathogen spread (Weste and Marks, 1987). *Phytophthora cinnamomi* requires wet soil for reproduction and spread (Weste and Ruppin, 1977) and water bodies themselves also disperse *Phytophthora* spores (La Manna *et al.,* 2012; Huberli *et al.,* 2013). The water-bodies layer was converted using the Euclidian Distance function such that the edge of a water body represented maximum risk, and as soil moisture decreased with distance, so too did risk. A layer reflecting soil wetness was created using the DEM and the Flow Direction function in ArcMap to reflect the topographic effect on soil moisture. Soil type has also been shown to influence the distribution of *P. cinnamomi* (Podger and Brown, 1989), however, the soil types collected during the survey did not represent all of the soil types present in the scene and therefore, the effect (if any) of soil type on the distribution of *P. cinnamomi* in the GBMWHA could not be addressed directly. Instead, soil texture, by way of topsoil percent clay was used. *Phytophthora cinnamomi* prefers sandy soil because of increased aeration and a larger the pore size allowing for zoospore dispersal (Byrt and Grant, 1979). Additionally, sandy soils have lower organic matter and thus lower microbial activity, that is known to suppress the occurrence of *P. cinnamomi* in soil (Podger and Baker, 1974; Meyer and Linderman, 1986). The percent clay layer (Henderson *et al.,* 2001; available from ASRIS: Australian Soil Resource Information System, www.anra.gov.au) was converted from a categorical variable into a continuous variable using the mean estimated clay content of each soil texture grade (McDonald *et al.,* 1990) and bilinear interpolation in ArcMap. Climatic variables of annual rainfall, rainfall seasonality, maximum mean temperature of the warmest period and minimum mean temperature of the coldest period were developed with the BIOCLIM package (Hutchinson, 2004 version 5.2) using the DEM. These variables were included following the occurrence of *P. cinnamomi* in specific climates (Marks *et al.,* 1975; Byrt and Grant, 1979; Phillips and Weste, 1985; O'Gara *et al.,* 2005). All layers were projected in GDA94 Map Grid Australia Zone 56 and where necessary, re-sampled to 25m using Inverse Distance Weighting. Finally, all the layers were standardised such that they had a mean of zero and a standard deviation of one.

4.2.1.2 Maximum entropy model of *P. cinnamomi* distribution

The correlation between layers was assessed using ArcMap using the correlation matrix function. Each of the layers was converted to ASCII format and then exported to Maxent v 3.3.3a. Default settings were maintained for the analysis and 25% of the data was reserved for testing. The first model run contained all the above variables except annual

rainfall and maximum temperature as they were each highly correlated with another variable. From the output, any variable with less than a 5% contribution (i.e. no statistically significant contribution) to the model outcome was removed and the model was re-run as per Keith *et al.* (2012). Also, where layers were highly correlated, they were interchanged with their opposing layer to identify the layer with better predictive power. Multiple variable combinations were trialed until the model was as simple as possible without comprising model performance (as determined by the AUC). The final model of the distribution of *P. cinnamomi* in the GBMWHA was completed using the layers for annual rainfall, minimum temperature and clay topsoil percentage.

Table 4.1 Summary of layers utilised in disease risk model development, their conversion and source.

Layer	Conversion	Data Source
Built-up areas	Euclidian distance	OEH
Subsoil clay %	Bilinear interpolation	ASRIS
Topsoil clay %	Bilinear interpolation	ASRIS
Rain annual	none	Bioclim
Rain seasonality	none	Bioclim
Slope	Slope function	DEM
Max. temp. of the warmest period	none	Bioclim
Min. temp. of the coolest period	none	Bioclim
Water bodies	Euclidian distance	OEH
Wetness	flow direction function	DEM
Roads	Kernel Density	OEH

4.2.2 Host distribution and susceptibility

The distribution of host susceptibilities was constructed in a similar way to that of the distribution of *P. cinnamomi*.

4.2.2.1 Host selection and data acquisition

At present there are approximately 130 species known to be susceptible to *P. cinnamomi* growing in the GBMWHA (Chapter 1, Appendix 8.1). The distribution of 124 of these species was modelled in Maxent using records obtained from the Atlas of Living Australia database (ALA, www.ala.org.au). Data contained in the ALA database has been collected in a standardised manner with all species in a survey plot being identified. Because of this, the accuracy of records on ALA is maintained to a high quality. The ALA records also include the spatial accuracy of each species listing. Data was downloaded from ALA and inspected. Where too few recorded existed, species information was withheld due

to conservation status (e.g. Wollemi Pine, *Wollemia nobilis*) or spatial accuracy was greater than 100m, these sightings were excluded from the analysis. The species included in the host model have been outlined in Appendix 8.1.

4.2.2.2 Maximum entropy model of susceptible host distributions

The distribution of each species was then modelled in Maxent using the layers outlined in section 4.2.1.1, however only minimum temperature of the coldest period, annual rainfall, topsoil clay percentage and soil wetness were used following a process of model selection. The default settings in Maxent were maintained and 25% of each dataset was reserved for testing. Each model was inspected and exported to ArcMap for projection. Finally, all layers were combined using the Raster Calculator to produce a map of the distribution of susceptible host within the GBMWHA.

4.2.2.3 Model of distribution of host scores

The host species distributions were also classified and combined to reflect the distribution of host susceptibility. Each of the modelled distributions was multiplied by the species susceptibility score (as outlined in the Appendix 8.1). Each of these outputs was then combine using the Raster Calculator, the result of which produced an image of the distribution of the level of host susceptibility across the GBMWHA, that is, the summed value of each species probability of distribution multiplied by its susceptibility score.

4.2.3 Model of Phytophthora dieback disease risk for the GBMWHA

The final stage of disease risk model development was the amalgamation of the distribution maps of both *P. cinnamomi* and the susceptible hosts. Using the Raster Calculator, the two layers were multiplied to produce an image of disease risk in the GBMWHA based on the co-occurrence of the pathogen and hosts.

4.2.4 Climatic sensitivity of the modelled distribution *P. cinnamomi*

To inspect the sensitivity of the *P. cinnamomi* distribution model to an altered climatic system, the model was re-run having made small adjustments to the rainfall and temperature inputs. In all, an additional 125 models were compiled in Maxent representing a matrix of rainfall and temperature combinations. The minimum temperature was increase by 5°C in 1 degree increments and rainfall averages were adjusted to range from a 10% decreased to 10% increase by 1% increments. All Maxent settings were left as before, and

clay topsoil was maintained, unadjusted. The model outputs were projected in ArcMap and each raster layer was clipped to the approximate boarder of the GBMWHA. The sum of the probabilities remaining within the GBMWHA was then determined for each rainfall and temperature combination. The final dataset was then exported to R (R Core Team, 2012) for compilation into a sensitivity map for easy visualisation.

4.3 Results

The modelled distribution of *P. cinnamomi* risk identified that the GBMWHA is a highly conducive environment. Approximately one third of the GBMWHA has a minimum suitability score of 0.5. Hosts were most abundant at the higher altitudes of the GBMWHA following a north-south direction. Based on the combined probability of the occurrence of *P. cinnamomi* and susceptible hosts, areas of high disease risk straddled the Great Dividing Range and any high altitude areas with the highest risk occurring in the centre of the Blue Mountains NP. Changes to the inputs of the *P. cinnamomi* distribution model revealed the model was more sensitive to changes in rainfall then temperature such that the summed probabilities increased or decreased in unison with rainfall.

4.3.1 Prediction of *P. cinnamomi* distribution across the GBMWHA

The final model of the most suitable areas for *P. cinnamomi* in the GBMWHA, identified that most of the WHA is conducive to the pathogen (Figure 4.1; Appendix 8.4). The final model performed well with an AUC of 0.937 for the training data and 0.927 for the test data. Annual rainfall made the largest contribution to the model with 39.1%, which was closely followed by the minimum temperature of the coldest period (33.4%) and topsoil clay (27.4%). The logistic probability outputs of the *P. cinnamomi* distribution indicates it is most likely to be found where annual rainfall is 1,300 mmpa, temperature minimum are between 11.5°C and 13.5°C and the topsoil clay percentage is between approximately 6-8%. Away from these limits, suitability decreased. *Phytophthora cinnamomi* was not predicted to occur where rainfall was less than 550 mmpa, the minimum temperature of the coldest period did not drop below 18 °C and the top soil clay content exceeded 37%.

The area predicted to be most conducive was centred around the middle of the Blue Mountains NP and the southern section of the Wollemi NP. The northern bound included the Wollangambe Wilderness, then south to the lands surrounding Mt Wilson and Mt Irvine, in the northern section of the Blue Mountains NP. This continued to the head

lands of the Grose Valley and down the Mt Hay Range. The western boundary stretched from Mt Victoria down through Blackheath to Katoomba then along Narrow Neck and Mt Solitary. The eastern edge of the higher risk locations followed Lawson Ridge and continued south onto Kings Tableland ending at the headland of McMahons Lookout (Figure 4.2). The areas that appear to be least conducive to *P. cinnamomi* were the most northern portion of the Wollemi NP and the south-western corner of Kanangra-Boyd NP. Less conducive areas occurred sporadically over the park and typically followed low lying areas.

4.3.2 Distribution of susceptible host and host scores across the GBMWHA

The models of host distributions performed well with an average training AUC of 0.89 (standard deviation = 0.04) and testing AUC of 0.88 (standard deviation = 0.04), the lowest training AUC being 0.798. The minimum number of species records used to construct a single model was 15. The statistical outputs of the species models have been tabulated and presented in the Appendix 8.5. According to the combined suitability models the greatest frequency of susceptible host occurs along the western side of the GBMWHA in a north-south direction. A high frequency of hosts also occurs in the northern region of the GBMWHA where the Wollemi NP runs parallel to the Goulbourn River NP, and on the lower, eastern side of Yengo NP (Figure 4.3).

Incorporation of a susceptibility rating did not alter the distribution of host scores as most species included had only low levels of susceptibility. The areas with the greatest occurrence of high risk species were outside the GBMWHA at the south-western corner of Kanangra-Boyd NP and north-west of Jenolan-Karst CR.

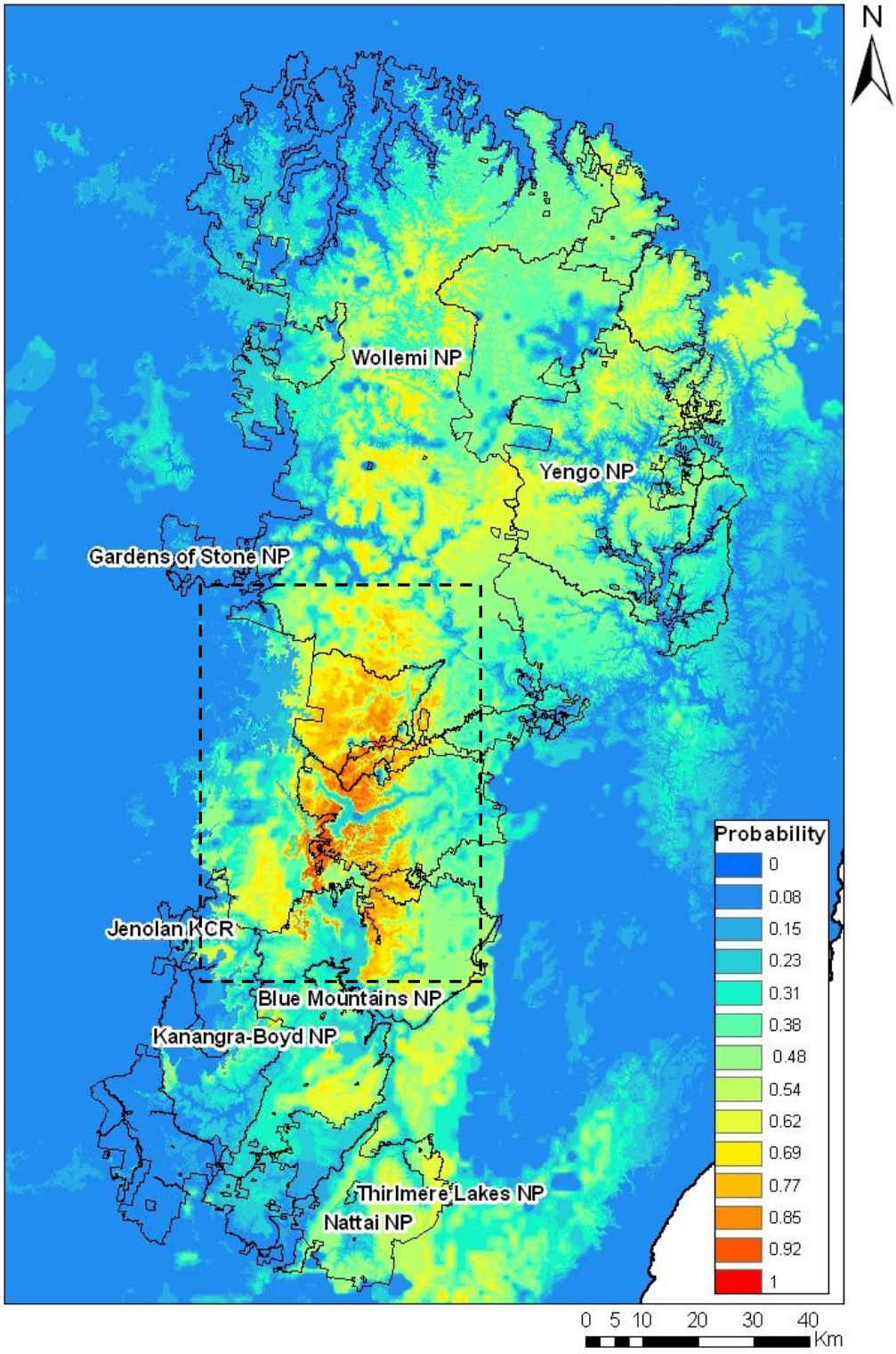


Figure 4.1 Predicted suitability score of *P. cinnamomi* in the GBMWHA. Dashed area is shown in detail in Figure 4.2.

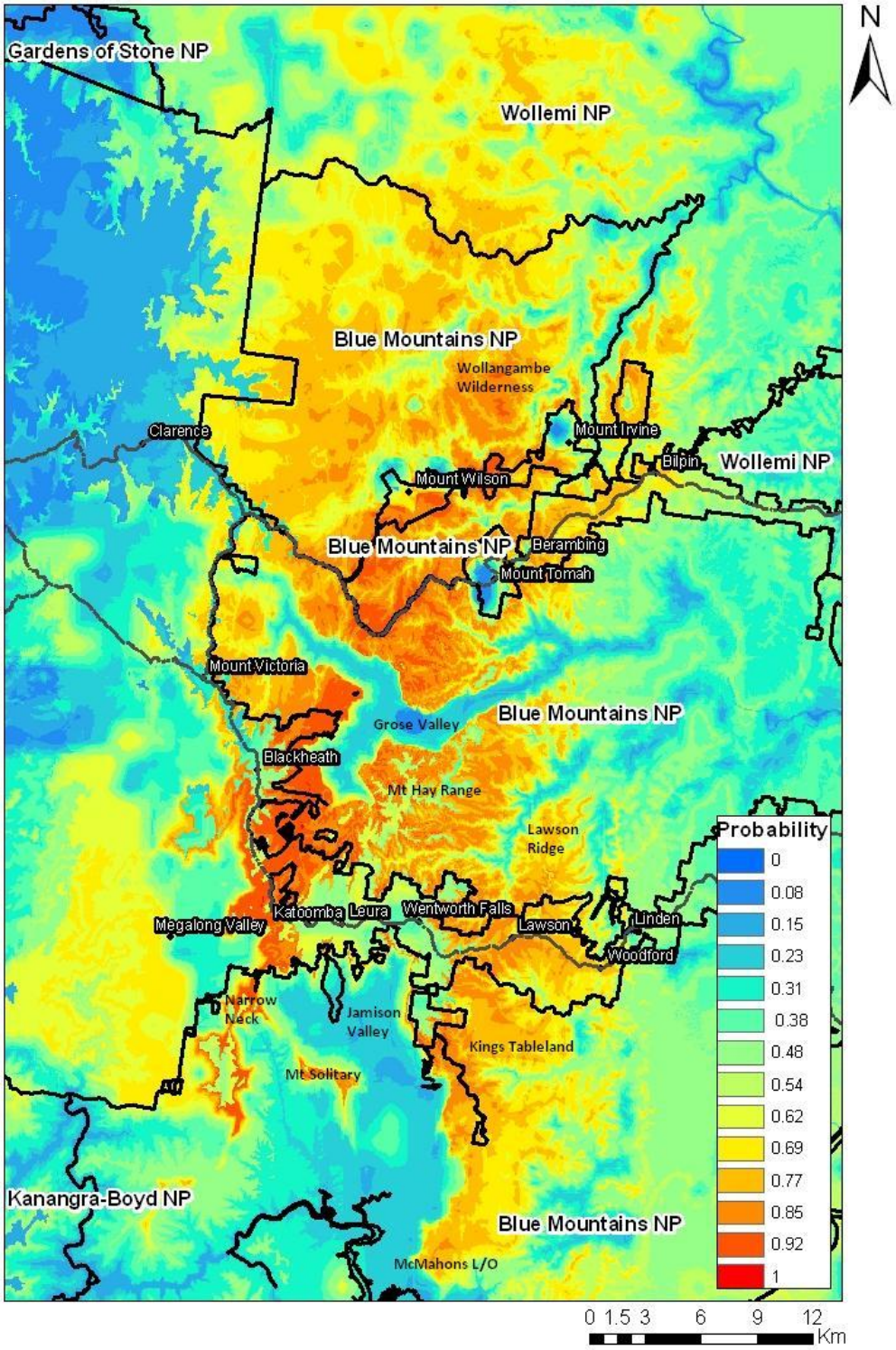


Figure 4.2 Area of highest *P. cinnamomi* suitabilty within the GBMWHA.

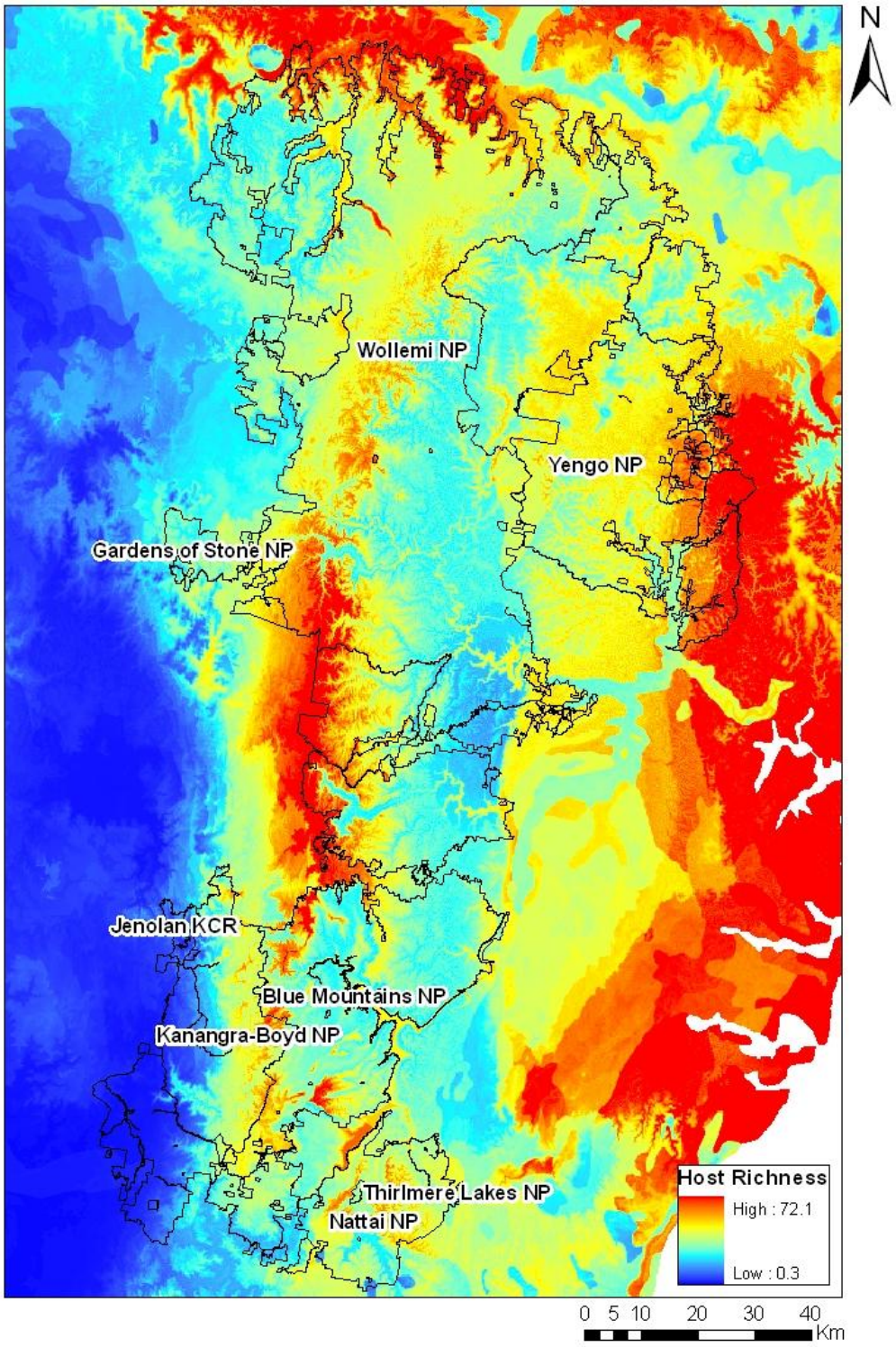


Figure 4.3 Cumulative suitability score of *P. cinnamomi*-host distributions.

4.3.3 Phytophthora dieback disease risk of the GBMWHA

As the final disease risk model was completed by combining the host distribution model with the *P. cinnamomi* distribution model, the distribution of disease risk reflects both of the inputs (Figure 4.4). The area of greatest risk followed the western boundary of the Blue Mountains NP from the intersection of the Wollemi and Blue Mountains NPs and continued south through Mt Victoria, Blackheath, Katoomba and down onto Narrow Neck (Figure 4.5). The areas of lowest risk were generally the most western parts of the GBMWHA including the south-western corner of the Blue Mountains NP (below Kanangra-Boyd NP), the west side of Kanangra Boyd NP and Jenolan-Karst CR, Gardens of Stone NP and the north-western corner of the Wollemi NP.

4.3.4 Influence of climate change on *P. cinnamomi* distribution

The modelled distribution of *P. cinnamomi* changed with adjustments made to either/or annual rainfall and minimum temperature (Figure 4.6). When no change was made to either, the sum of the probabilities in the GBMWHA was approximately 4.1 $x10^7$. Changes to rainfall had greater influence on the model where increasing rainfall increased the sum of probabilities and decreasing rainfall, decreased the sum of the probabilities. With no change in temperature, these values were 4.7 $x10^7$ for a 10% increase and 2.7 $x10^7$ for a 10% decrease. Increasing temperature, however, reduced the summed probabilities. When rainfall was left unchanged and temperature was increased by 5°C, the summed probabilities decreased by 3.5 $x10^6$. The reduction in the sum of probabilities associated with increasing temperature was smaller if rainfall was decreased and larger when rainfall was increased. When rainfall was decreased by 10%, the difference between the summed probabilities of the 0°C and 5°C models was 2.1 $x10^6$, and when rainfall was increased by 10% the difference between the 0°C and 5°C models was 3.9 $x10^6$ almost halving the summed probabilities across the 20% rainfall range. The reduction in the sum of probabilities associated with decreasing rainfall became larger as the temperature increased. The reduction in summed probabilities caused by temperature could be offset, in part, by rainfall such that increases in temperature combined with specific increases in rainfall maintained the sum of probabilities as though no changes had been made to the climate system at all. The effects of changing the rainfall and temperature inputs of the *P. cinnamomi* prediction model have been display visually in Figure 4.6.

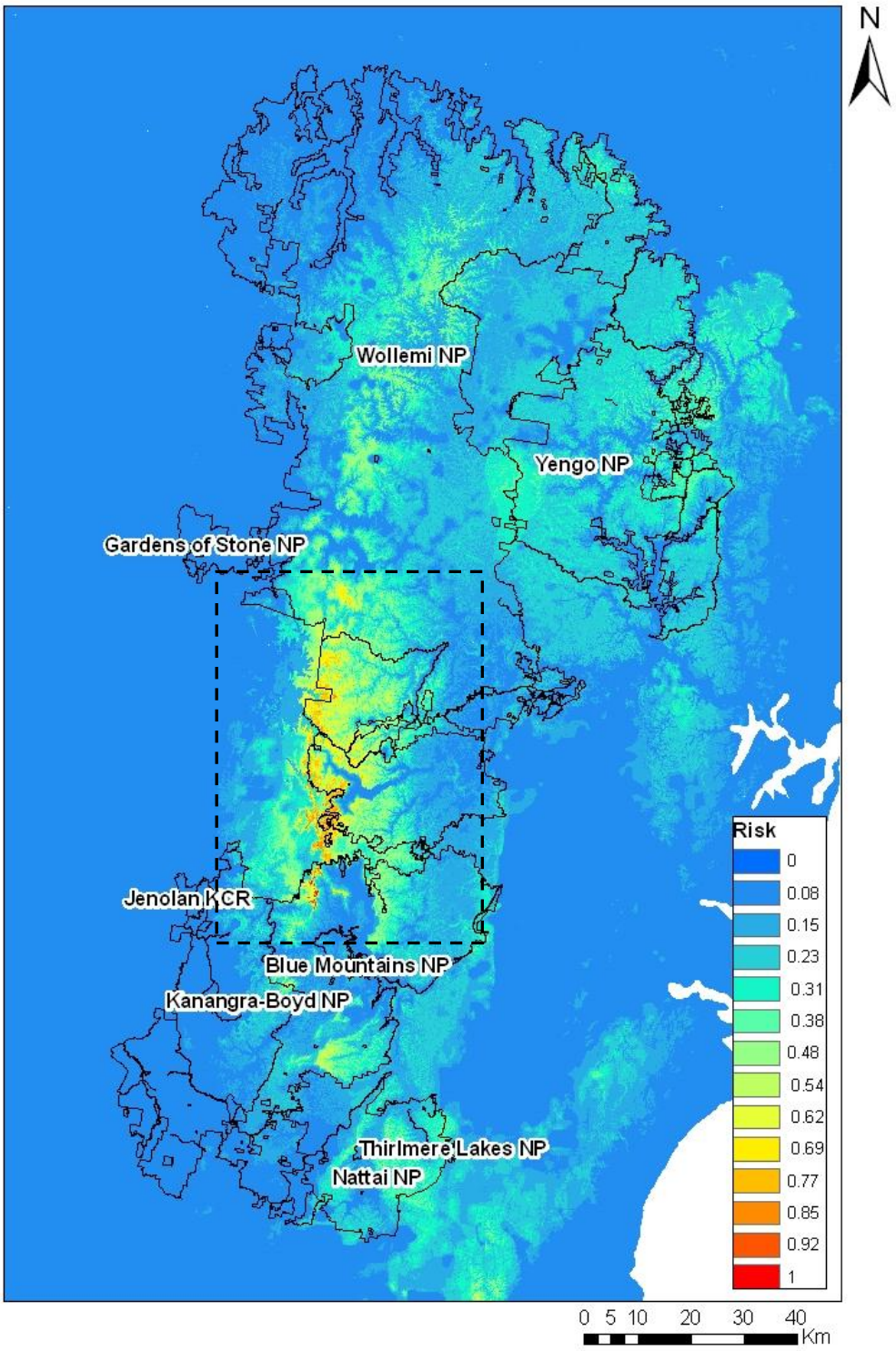


Figure 4.4 Model of predicted risk of Phytophthora dieback occurring within the GBMWHA. Dashed area is shown in detail in Figure 4.5.

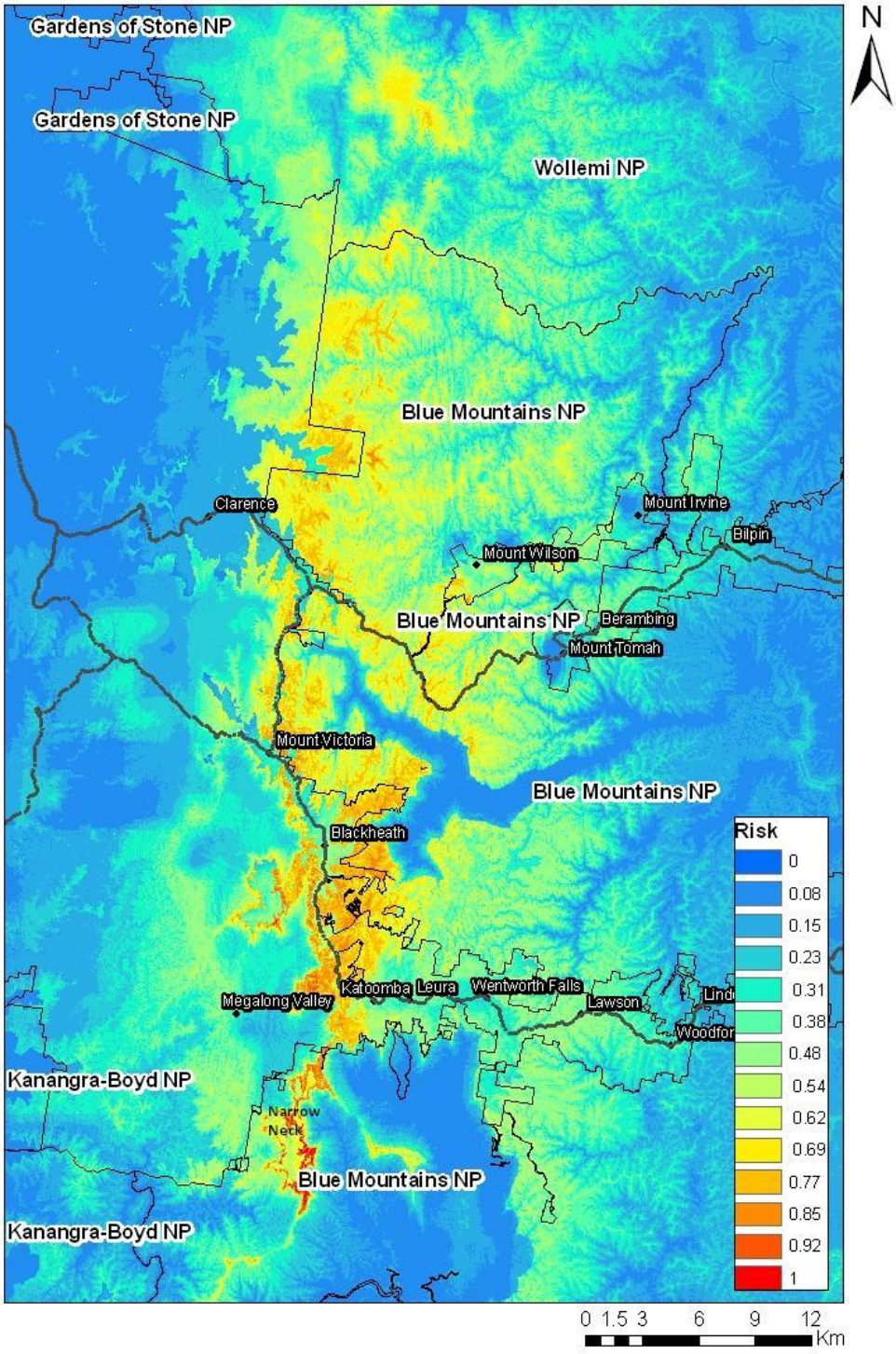


Figure 4.5 High risk areas of Phytophthora dieback as predicted for the GBMWHA.

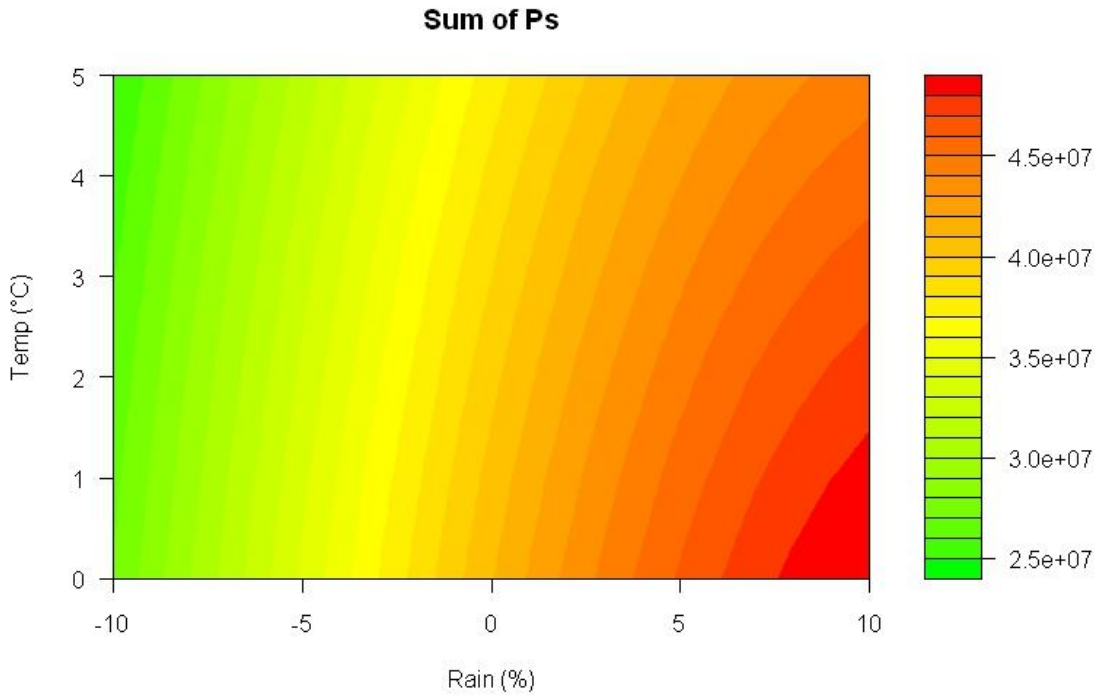


Figure 4.6 Variation in the sum of predicted probabilities of the distribution of *P. cinnamomi* in the GBMWHA associated with a changing climatic environment.

4.4 Discussion

The highest risk of Phytophthora dieback calculated in the model occurred in a small corridor on the western side of the GBMWHA spaning the length of the central region of the Blue Mountains NP. This area contains some of the most renowned locations of the GBMWHA such as the Three Sisters at Echo Point, Wentworth Falls and the Grose Valley. Degradation to the natural landscape caused by Phytophthora dieback could reduce this intrinsic value for which that area is acclaimed, with potential impacts on tourism and the local community. Perhaps even more importantly, Phytophthora dieback has the potential to cause irreversible damage to ecosystem function within this part of the GBMWHA, leading to habitat destruction and species extinction. The risk of disease is greatest in this area because, according to the model, *P. cinnamomi* is ideally suited to the environmental conditions of the upper parts of the mountains and there is a greater frequency of host species in the area. Evidence of dieback observed in the field (Chapter 3), supports the findings that Phytophthora dieback has a high probability of occurrence in this area.

The areas with lowest disease risk also occurred on the western side of the GBMWHA at the southern and northern limits. At the southern end, in Kanangra-Boyd NP, few hosts are predicted to be present, yet in the north, in Wollemi NP, this is not the case. Lower disease risk in the Wollemi NP is instead associated with a lower predicted probability for the occurrence of *P. cinnamomi*. In this case, should the environment change, it may become more suitable to *P. cinnamomi* increasing the likelihood of disease.

Considering the ecology of *P. cinnamomi*, the outcomes of the model seems sensible. The final model was constructed using rainfall, temperature and soil texture, each of which has been associated with the successful invasion of *P. cinnamomi* around the world. As a soil-inhabiting organism that requires water for spore dispersal and survival (Erwin and Ribeiro, 1996), it follows that the model should predict the distribution of *P. cinnamomi* to only occur in rainfall areas of ≥550 mmpa which is close to the reported lower annual rainfall limit of 600 mmpa (O'Gara *et al.,* 2005). Additionally, *P. cinnamomi* requires specific temperatures for survival (Marks *et al.,* 1975; Byrt and Grant, 1979; Phillips and Weste, 1985). *Phytophthora cinnamomi* was not isolated from areas where the minimum temperature of the coldest period did not drop below 18°C. Although *P. cinnamomi* survives to temperatures as low as 10°C in soil (Weste and Ruppin, 1977), this value is likely to reflect areas where the maximum temperatures exceed the upper limit of *P. cinnamomi* survival of 35°C (Marks *et al.,* 1975; Phillips and Weste, 1985).

The influence of soil texture on the occurrence of *P. cinnamomi* has not be investigate as extensively as the influence of rainfall and temperature, however, the proportion of clay in the soil is relevant to *P. cinnamomi*s distribution due to its effect on soil-water relations. *Phytophthora cinnamomi* advances through the soil profile with motile spores called zoospores. To move unobstructed through a soil profile, pore spaces need to be 50-140 µm in diameter (Allen and Newhook, 1974). As clay particles are 2 µm, increasing clay content will reduce soil pore size suppressing zoospore dissemination. Conversely, as sand content increases, water leaves the soil profile quickly, and again zoospores are prevented from spreading due to a lack of water. These relationships have been accounted for in the prediction of *P. cinnamomi* distribution in which the proportion of clay in the soil has an upper and lower limit.

It appears from the model that *P. cinnamomi* is only occurring at higher altitudes within the GBMWHA. This is clear from the high probability of *P. cinnamomi* predicted along the ridges and tablelands of the GBMWHA, while there is a low prediction in the valleys, or where the height of the GBMWHA drops down the flanks of the Great Dividing Range. Although altitude was not included as a variable during model construction, it has been effectively accounted for due to its effect on temperature. Altitude has made significant contributions to the distribution of *P. cinnamomi* in at least two other models of the distribution of *P. cinnamomi* (Wilson *et al.,* 2003; Moreira and Martins, 2005), however in both cases, the effect has been negatively correlated such that the probability of *P. cinnamomi* decreased with altitude. However, at Barrington Tops NP in NSW, *P. cinnamomi* is observed occurring at higher altitudes (McDougall *et al.,* 2003), and again in the Queensland wet tropics (Gadek and Worboys, 2003). These differences are likely to be location-specific requirements, where altitude is behaving as a surrogate for temperature which is ultimately the factor dictating habitat suitability.

Poor contribution associated with the other variables incorporated in the modelling process was unexpected especially in the circumstance that they had been useful in other studies. Slope has previously been reported to make a significant contribution to the model of *P. cinnamomi* in the Royal National Park (Keith *et al.,* 2012) and influence its distribution in the Eastern Otway Ranges in Victoria (Wilson *et al.,* 2000), yet it made no significant contribution to the modelled distribution of *P. cinnamomi* in the GBMWHA. Soil wetness and distance to water bodies made no significant contribution either. Corcobado *et al.* (2013) recently demonstrated how neither slope or soil moisture associated with proximity to water bodies causes any lesser or greater disease in Oak trees (*Queruc ilex*) infected with *P. cinnamomi,* in Spain. Locations close to streams had higher pathogen activity resulting in

greater disease. Up-slope areas lose water more quickly leading to earlier presentation of water stress. In both cases, disease results. Why then *P. cinnamomi* seems to be associated with ridges and not valleys in the GBMWHA remains unexplained, but it may be as a result of microclimatic variations between ridges and valleys or a higher sand content on ridges compared to higher colluvial clay deposits on valley floors again influencing soil-water relations.

The failure of the anthropogenic layers to provide any contribution to the distribution of *P. cinnamomi* is interesting. As an introduced species, and as found in other models of *Phytophthora* distribution (Cushman and Meentemeyer, 2008; Meentemeyer *et al.,* 2008; Harwood *et al.,* 2009; Xu *et al.,* 2009; Chadfield and Pautasso, 2012; Thompson *et al.,* 2013), variables associated with human activity make a contribution, often substantial, to model performance. An explanation for this may be that *P. cinnamomi* has been present in the GBMWHA long enough to have reached somewhat of an equilibrium state in which its distribution is moderated primarily by the environment.

Incorporating the host distribution in the assessment of Phytophthora dieback risk was a key part of the model as it formed the third element of the disease triangle. In so doing, high risk locations were refined even further to an area 40 km long and at most 10 km wide centred on the Blue Mountains NP. Similar to other studies (Keith *et al.,* 2012; Purse *et al.,* 2013; Thompson *et al.,* 2013), a level of host susceptibility was considered in the assessment of risk, an approach which is reported to be quite novel but effective (Purse *et al.,* 2013). Incorporating host susceptibility in this case however, made little difference to the outcomes of predicted disease risk (and therefore the final model of Phytophthora dieback disease risk was constructed using the host distribution layer). Many of the species modelled here have a lower susceptibility and therefore increases in risk associated with increased susceptibility occurred infrequently. Also, species that were coded with a higher susceptibility score had a lower probability of prediction thereby cancelling out their proportional risk. Species with a high susceptibility and a high predicted probability of occurrence were either in areas with low *P. cinnamomi* probability or outside of the GBMWHA. Since there is limited information on the susceptibility of species to *P. cinnamomi* in NSW, the assessment of the host element of Phytophthora dieback has been limited to that of the host distribution only.

Keith *et al.* (2012) faced this same problem and chose instead to estimate host susceptibility based on taxonomic relationships. This is suggested by some to be plausible (Cahill, 2008) however, there is large variations in the susceptibility of several genera such as *Banksia* (Cho, 1983), *Eucalyptus* (Podger and Batini, 1971, Tippett *et al.,* 1985; Suddaby,

2008b), *Lambertia* (Shearer *et al.,* 2007; Suddaby *et al.,* 2008), *Pultenaea* (Barker and Wardlaw, 1995; Suddaby, 2008b), *Epacris*, and *Acacia* indicating that species susceptibility is not consistent at the genus level (Shearer *et al.,* 2004). Additionally, there tends to be differences between geographic regions such that eastern Australian species tend to be more resistant to *P. cinnamomi* infection compared to Western Australian species (Blowes *et al.,* 1982; McCredie *et al.,* 1985; Suddaby *et al.,* 2008). Generalising species susceptibility can lead to over- or underpredictions of the susceptibility of vegetation creating erroneous models. For this reason, only species for which susceptibility had been reported were used and in the event that more than one level of susceptibility was reported, a composite value was assigned. Had the predicted distribution of these species been different or their susceptibilities varied more, outcomes of the host score model would have been quite different and likely to have had a stronger influence on the Phytophthora dieback risk model.

The final variables included in the model of *P. cinnamomi*s distribution follow the aetiology of Phytophthora dieback. As a result, areas predicted by this model to contain the pathogen coincide, in several cases, with locations *P. cinnamomi* is already known to occur. This includes the Royal NP (Walsh *et al.,* 2006), Barrington Tops NP (McDougall *et al.,* 2003), Dhaural NP (Suddaby, 2008a), Sydney Harbour NP (Daniel *et al.,* 2006), and along the south coast of NSW from which *P. cinnamomi* has been isolated at numerous locations. This indicates that the model performs well to predict the distribution of *P. cinnamomi* over an area larger than just the GBMWHA.

Variation in the climatic system of the *P. cinnamomi* model influenced the predicted distribution of the pathogen especially with regard to annual rainfall. Because of the influence of rainfall and temperature on the survival of *P. cinnamomi*, this outcome is not unexpected. Alterations to the distribution associated with each variable individually were expected, however, the interaction between them was not. The GBMWHA is climatically cooler than other places of *P. cinnamomi* distribution around the world especially in the upper Blue Mountains NP where *P. cinnamomi* has the highest level of predicted probability; increases in temperature would be expected to simply move averages closer to the preferred temperature range. Changes to temperature however may be reducing the prediction of probability in other areas of that park where the temperature averages are already approaching the upper limit of *P. cinnamomi* suitability. The aim of this assessment was not to investigate the potential impact of climate change on the distribution of Phytophthora dieback, as there are numerous interactions to be explained that are beyond the scope of this study, but simply to explore how rainfall and temperature

influence the modelled *P. cinnamomi* distribution. None the less, having a better understanding of how pathogen risk may differ in above or below average years is still informative and useful for management as it may indicate where disease risk is likely to increase at such times.

Temperature and rainfall changes made to the model have given a brief insight into what might be expected during shifts in climatic averages such as those predicted for climate change. It is generally accepted that climate change will alter the distribution of many species and in so doing be either adventitious or disadventitious for invasive species (Chakraborty *et al.,* 2000). Range expansions have been predicted for *P. cinnamomi* in Europe (Brasier, 1996; Bergot *et al.,* 2004) due to an increase in temperature minimums. In France, the incidence of *P. cinnamomi* is expected to increases because of a reduction in cold stress (Desprez-Loustau *et al.,* 2007). The potential influence of precipitation changes is much less understood. The impacts of climate change on Phytophthora dieback is, at present, largely unknown.

In Chapter 2, the utility of a mechanistic model was addressed to predict the distribution of *P. cinnamomi* in the GBMWHA. At the time, information on the distribution of *P. cinnamomi* across the GBMWHA was limited and by no means comprehensive in either geographical range or variable representation. Here however, the results of the field survey (Chapter 3) have been used to assess environmental suitability of the GBMWHA to *P. cinnamomi* taking advantage of a statistically-based approach. This has greatly benefited the model for several reasons. Firstly, the process of model selection has allowed for the removal of spatial autocorrelation, which reduces model accuracy, (Vaclavik *et al.,* 2012), and simplified the model as much as possible. This included the exclusion of anthropogenic layers highlights the possibility that *P. cinnamomi* is progressing to a naturalised state in the GBMWHA which has significant implications for management. Secondly, the interaction between temperature and rainfall could not have been identified using a mechanisitic model (Elith *et al.,* 2006). This interaction became obvious when each of these layers was changed revealing that, although increasing temperatures caused a reduction in the predicted distribution of *P. cinnamomi*, this reduction could be offset partially, by increasing rainfall.

The use of Maxent for this statistically-based approach was also quite useful. Not only does Maxent assist in the process of model selection by reporting variable contribution, but perhaps its greatest advantage for this study was in the use of pseudo-absences data. *Phytophthora cinnamomi* is difficult to isolate from soil, so much so that Pryce *et al.* (2002) suggests that for a disease site in Queensland to be rendered pathogen free with 95%

confidence, approximately 1kg of soil would need to be tested. In Western Australia this amount increases to 40 sample at approximately 350g each or 14kg of soil (Davison and Tay, 2005)! Not surprisingly, researchers accept the occurrence of 'false negatives', knowing that the only putative result is a positive. For this reason, the 'negative' results collected during the survey were doubtful and therefore should not be utilised in distribution modelling (La Manna *et al.,* 2012). Maxent generates random background points from the scene and uses these in model calibration (Phillips *et al.,* 2006). Purse *et al.* (2013) also found that the most effective way to incorporate the background points was to allow them to be selected for the whole scene instead of just the area studied or the areas accessible for model testing. This was also the case here where model performance was best when absence data were selected from the whole scene (data not shown).

Maxent also has the advantage that it assess model performance automatically. The final model of *P. cinnamomi* distribution with an training AUC of 0.937 and testing AUC of 0.927, according to Swets (1988), is excellent. The model also performed better than others produced in Maxent including the distribution of *P. cinnamomi* (Keith *et al.,* 2012), and *P. austrocedrae* (La Manna *et al.,* 2012). The combined models of host distribution also had a 'good' AUC value (Swets, 1988) indicating strong predictive performance. By combining two well performing and ecologically sound models based on extensive sampling data, the resulting prediction of Phytophthora dieback risk for the GBMWHA accounts for all elements of the disease triangle, based on the current understanding of the pathogens distribution.

Highlighting the areas where disease is most likely to occur is required for effective management. The high risk areas coincide with much of the upper mountains tourist activity including areas such as Blackheath, Katoomba, Echo Point, Leura and Wentworth Falls. Efforts to prevent *P. cinnamomi* entering and exiting the GBMWHA should be made to stop further introductions of *Phytophthora* or dispersal of *P. cinnamomi* to areas outside the GBMWHA . This may include sealing dirt roads such as Glenraphael Drive along Narrow neck or incorporating foot baths at track heads such as Wentworth Falls, Leura, Katoomba, Echo Point and Blackheath. Education in these locations would also be worthwhile especially given their high volume of visitor activity.

Investigations into where species with conservation status occur that coincide with locations predicted to be highly conducive to the occurrence of *P. cinnamomi* should also be a management priority. Additional sampling would be pertinent in these areas, and species health should be assessed with appropriate treatments applied.

In terms of management of *P. cinnamomi* for the GBMWHA, access restriction is not likely to be an option for two reasons: *P. cinnamomi* is widely distributed within the GBMWHA, and many of the areas that are predicted to be highly susceptible are popular tourist areas with multiple access points. Instead proactive spread prevention and education should be utilised. Temporary site restrictions (e.g., during high rainfall events) may be an option for specific sites if considered worthwhile.

Our understanding of the susceptibility of NSW plant species to *P. cinnamomi* infection is limited. As mentioned here, this has limited model development, but it also limits effective management of areas with highly susceptible species assemblages or susceptible individual with high conservation status. Keith *et al.* (2012) suggested using the expression of disease as a surrogate for species susceptibility which could then be used to model the distribution of the pathogen. *Phytophthora cinnamomi* is but one cause of dieback and as recently reported in WA, when susceptible host have all but been removed, *P. cinnamomi* can go on living in asymptomatic hosts (Crone *et al.,* 2013a). While so little is understood of Phytophthora dieback aetiology in NSW, incorporation of species susceptibility into assessments of disease risk should continue to be utilised in established host-pathogen interactions. To this, our understanding of the disease triangle of Phytophthora dieback will be benefited by further research especially in terms of the environmental component. Such areas could investigate the effect altitude has on *P. cinnamomis* distribution and how climate change is going to effect disease risk.

The potential risk of Phytophthora dieback in the GBMWHA has been modelled in this chapter using a combination of host and pathogen distributions. The model outcomes are strong and reflect what it understood of the behaviour of *P. cinnamomi* in natural environments. These results allow land managers to focus their attention to where disease is likely to be worse allowing for more efficient use of time and funds. Although only a small area is predicted to be at high risk of Phytophthora dieback, it coincides with some of the most renowned and valuable parts of the GBMWHA. These findings warrant further investigation to the specific impacts of Phytophthora dieback on the ecosystem functions in the GBMWHA.

Chapter 5 Hyperspectral leaf response of plants inoculated with *Phytophthora cinnamomi*

5.1 Introduction

Dieback associated with the water mould *P. cinnamomi* (Phytophthora dieback) has been occurring in native vegetation communities around Australia for over sixty years (Weste, 1994). The effects of Phytophthora dieback are expressed across a wide range of plant species with varying habits, growing in a range of environments. In severe cases 75% of the understorey and 100% of the canopy may be lost (Kennedy and Weste, 1986; Weste and Marks, 1987; Weste *et al.,* 2002; Weste, 2003), leading to ecosystem change and habitat destruction. *Phytophthora cinnamomi* is an introduced pathogen that is widespread across Australia (Newhook and Podger, 1972), however it is continually being identified as the cause of disease in areas not previously known to be infected including the Greater Blue Mountains World Heritage Area (GBMWHA) the largest conservation reserve in the state of New South Wales (Suddaby, 2008a).

Detection of disease depends upon the direct isolation of the pathogen or the presumption of disease based on the appearance of typical symptoms. In the first instance, samples of diseased plants are collected, or soil is collected from their vicinity. In either case, the material is returned to a laboratory for pathogen detection via a number of different methods (see O'Brien *et al.,* 2009 for a review). This process is lengthy, expensive, requires specific facilities and can lead to false negatives where the pathogen, although present, in not successfully isolated (Pryce *et al.,* 2002; Davison and Tay, 2005; O'Brien *et al.,* 2009). Alternatively, disease is identified remotely; either based on symptom presentation in 'indictor' species as seen from the ground, or as general dieback observed in aerial photography or satellite imagery from above. In the case of the latter, there is often a history of Phytophthora dieback in the area. Remote detection can lead to a misdiagnosis, but it can be a quicker, safer and a more efficient way to identify disease (Bock *et al.,* 2010) or at least identify areas that need further investigation.

Excluding the use of remote imagery, the identification of disease caused by *P. cinnamomi* is reliant upon someone gaining access to the area in question; either for interpretation or sample collection. This is problematic for areas that are hard to reach, and may be dangerous, remote or difficult to access. Site access also increases the chances of spreading *P. cinnamomi* inadvertently. A second drawback of current methodologies is that

the results quickly become outdated as the pathogen spreads (O'Gara *et al.,* 2005). Interpretation of remotely sensed imagery can avert both of these problems, however it is limited in its ability to identify non-severally affected vegetation or dieback in understorey vegetation (O'Gara *et al.,* 2005). The effect of *P. cinnamomi* infection is variable between individuals plants, between species, between geographic areas, and where additional stressors influence the expression of disease. Here, in New South Wales, infection is often asymptomatic (Pratt *et al.,* 1973; McDougall and Summerell, 2003), thus any attempt to interpret disease based on symptom expression would likely underestimate the pathogens-presences.

More sophisticated methods of disease detection via remote sensing may offer an appropriate alternative that is less expensive, real-time and ideal for areas that are geographically restricted (Jackson, 1986; Bock *et al.,* 2010). Remote sensing occurs when information is obtained on an object by viewing it without making contact with the objects surface. Remotely sensed assessments may be made from satellite imagery right down to hand-held probes that do not touch the object but measure energy emitted from its surface. Routine applications of remote sensing technologies in plant ecology include, for example, assessments of water stress, leaf area, evapotranspiration, chlorophyll content, foliar chemical composition, light use efficiency and productivity (Treitz and Howarth, 1999). The application of remote sensing to detect and quantify plant stress and plant disease has been demonstrated extensively since the early 1980s (reviewed in Jackson, 1986; Treitz and Howarth, 1999), however the last decade has seen increasing interest in the use of hyperspectral remote sensing (HRS) which appears superior in its ability to detect, delineate and quantify plant disease (Bock *et al.,* 2010). As the name suggests the acquired spectrum is divided into, typically, hundreds of single bands of reflectance providing high spectral resolution. In general, the wavelength measured extend from 350 nm to 1,000 nm or 2,500 nm often in nominal 1nm increments. This covers not just the visible wavelengths (400-700 nm), but ultra violet (350-400 nm, UV), near infrared (700-1,300 nm, NIR), and shortwave infrared (1,300-2,500 nm, SWIR). Examples of the use of hyperspectral reflectance and hyperspectral imagery to assess disease have been outlined in Table 5.1, including experiments associated with the detection of disease caused by *Phytophthora*.

More recent advances in hyperspectral remote sensing have shown that 'presymptomatic' stress detection is possible, i.e., stress can be detected before it is seen with the naked eye. This has been illustrated for three fungal diseases of Beetroot (Rumpf *et al.,* 2010) and Apple Scab (Delalieux *et al.,* 2009). The ability to identify disease before it

is visible would be particularly useful for the detection of infection caused by *P. cinnamomi* due to the visually asymptomatic response of some hosts.

Table 5.1 Examples of studies in which Hyperspectral Remote Sensing has been used to quantify disease caused by a variety of different microorganisms in a variety of different plant types.

Resource	Pathogen type	Reference
Cereals	fungi	(Bauriegel et al., 2011) (Cao et al., 2013) (Devadas et al., 2009) (Mahlein et al., 2013)
	virus	(Yang, 2010)
Annual crops	fungi	(Mahlein et al., 2012) (Reynolds et al., 2012) (Rumpf et al., 2010) (Yang et al., 2010)
	water mould [A]	(Ray et al., 2011) (Yusuf and He, 2011) (Zhang et al., 2003)
	virus	(Grisham et al., 2010)
Perennial crops	fungi	(Delalieux et al., 2009) (Sankaran et al., 2013)
	water mould [A]	(Pozdnyakova et al., 2002)
	virus	(Naidu et al., 2009)
Plantations	fungi	(Coops et al., 2003) (Shafri et al., 2011)
Natural environment	water mould [A]	(Pu et al., 2008b)

[A] Each of the examples of a water mould are for a study conducted on a disease caused by a species of *Phytophthora*.

The primary symptoms of *P. cinnamomi* infection include lesion development and necrosis of the root system, however it is the secondary symptoms of wilt, chlorosis and necrosis in the aerial parts of the plant that allow for the detection of disease. Within two hours of root infection, the membrane of root cells start to become permeable leading to electrolyte leakage, which is followed by an increase in cellular respiration (Cahill and Weste, 1983). This decreases leaf water potential, evapotranspiration, and the concentration of xylem Abscisic Acid (Maurel *et al.,* 2004). As the pathogen continues to move through a susceptible host, it destroys vascular tissue leading to additional cell wall hydrolysis further retarding hydraulic conductivity and ultimately leading to wilt (Weste, 1975b; Dawson and Weste, 1984). In some species, phloem discolouration may occur due

to polyphenol production and oxidation (Weste and Marks, 1987) and for those individuals that survive, there are significant reductions in biomass as well as decreased foliar carbon, nitrogen and potassium (Maurel *et al.,* 2001). Each of these factors can influence the colour and internal structure of the infected host; these are changes that may be detectable via HRS.

In this chapter, the effect of *P. cinnamomi* infection on hyperspectral leaf response is investigated. Although *P. cinnamomi* infects roots, assessments are made on leaves as this does not require the uprooting of plants; an approach which is achievable with remote sensing. This approach also investigates the possibility of using HRS to assess a root disease via foliar symptoms of which there are few examples (Yang *et al.,* 2010; Reynolds *et al.,* 2012). If disease of roots can be successfully detected from leaves, this may enable the subsequent detection of *P. cinnamomi* infection from aerial or satellite hyperspectral imagery. Detection may possibly depend upon the ability to delineate between water stress following *P. cinnamomi* infection and water stress caused by unavailable water. For this reason, each host tested is exposed to water stress. Additionally, due to variability in the host response as well as the effect of the defence response on leaf reflectance (Gitelson *et al.,* 2001; Coops *et al.,* 2004), hosts with a variety of susceptibilities have been included in the trial.

5.2 Methods

5.2.1 Plant material and glasshouse setup

Five species with a wide distribution across the GBMWHA where selected for the trial, each having a different host response to *P. cinnamomi* as reported in O'Gara *et al.* (2005) and other literature (Table 5.2). Different growth habits were also chosen to reflect differences in structural units of vegetation communities. *Angophora costata* and *Eucalyptus piperita* are both dominant overstorey species, while *Banksia serrata* occurs in the understorey and *Dianella revoluta and Lomandra longifolia* occur readily as ground cover.

Seedlings approximately six months old were obtained from the DPI Forest Nursery's, Cumberland, (West Pennant Hills, NSW) and Downes Nursery (Theresa Park, NSW). Seedlings were repotted into 1.5 L plastic pots with Debco Native Premium Potting Mix (Debco P/L, Berkshire Park, NSW) with a test tube placed around the root zone to facilitate inoculation.

The trial was completed at the University of Sydney, Bosch Glasshouse and began in June of 2012 when plants were around 15 months old. The temperature of the glasshouse was maintained at 20-27°C for a 12 hour day and 16-20°C at night. A pressurised watering system was installed providing 5 minutes of water via variable dripper (Pope P/L, Beverly, SA) to each pot at 6am and 1 pm every day.

Table 5.2 Species susceptibility ratings (host status) as reported in O'Gara (2005) and other references.

Species	Habit	Host status	References
A. costata	Tree	(MS) [A]	O'Gara *et al.,* 2005; Newby, 2007; OEH, 2011
B. serrata	Tree	S	Cho, 1983
D. revoluta	Grass	S	Shearer and Dillon, 1996
E. piperita	Tree	(FR) [A], (MS) [A]	O'Gara *et al.,* 2005; Newby, 2007
L. longifolia	Grass	FR	O'Gara *et al.,* 2005

[A] Host status recorded in brackets have not been published but are suspected to be thus based on field experiments and/or unpublished data.

5.2.2 Inoculation

A sand-bran inoculum (Aryantha *et al.,* 2000) containing propagating sand, wheat bran (soaked for 24 hrs in distilled water) and a millet seed mix (soaked for 48 hrs in distilled water) at a rate of 2:3:6 respectively, was prepared. The inoculum was pack loosely into 400 ml tissue culture jars (with a final volume of approximately 300 ml) and autoclaved 3 times for 25 minutes each.

Four isolates of *P. cinnamomi* collected from disease locations during the field work (Chapter 3) were used including "Glen12", "CTTT2", "Tree 3" and "1,067". Isolates were grown on Phytophthora Selective Media (PSM) for 7-10 days in the dark at 25°C, then transferred to half the jars of sterilised sand bran (20, 1 cm^2 pieces per 300 ml). The remaining jars were reserved for the uninoculated treatments. Jars were sealed, shaken lightly and place in the dark for three weeks with intermittent shaking.

Plants were randomly allocated to one of four treatment groups: control, water stressed, inoculated, water stressed + inoculated (combination treatment) with a minimum of ten replicates in each. For inoculation, the sand bran was homogenised in a sterilised tray, the test tube was removed and the space filled with the inoculated sand bran resulting

in about 30 mls of inoculum being added to each pot. Sterilised, uninoculated sand bran was added to the uninoculated pots in the same manner. Inoculated pots were physically separated before all pots were flooded to aid the spread of inoculum through the potting mix. Also at this time the variable drippers were adjusted such that non-water stressed plants received approximately 110 ml of water per minute while water stressed plants received approximately 50 ml/min.

5.2.3 Leaf reflectance measurements

Absolute reflectance of leaves was measured with the Field Spec 3 spectroradiometer fitted with a contact probe with an integrated halogen light-source, and the spectra recorded by the RS^3 Spectral Acquisition Program (ADS Inc., Boulder, Colorado, USA). To complete the measurements, a minimum of three leaves were selected randomly and stacked (Blackburn, 1999) over a black object (spectrally-flat slate), with the probe carefully placed on top of the leaves ensuring they remained stacked and no gaps occurred in the centre or at the edge of the field of view. Two measurements were taken from a different stack of leaves on each plant. Each reflectance measurement was the average of 40 individual reflectance measurements captured by the probe. Measurements were made from a reflectance standard (approximately 99% reflectance, Spectralon, Labsphere, Sutton, New Hampshire, USA) and calibrated with a dark reference (closed shutter) after every fifth plant. Measurements of reflectance were taken three times on a fortnightly basis before inoculation, and then conducted weekly following inoculation for nine weeks. This was followed by an additional six weeks of fortnightly measurements taking the total period of inoculation to fifteen weeks. Measurements were taken at the same time on each measurement date and as plants were individually marked they could be assessed in the same order each time.

5.2.4 Processing of Plants

During the trial, plants that were severely wilted were removed and the pathogen was reisolated by plating out surface sterilised pieces of root tissue onto PSM. Upon completion of the trial, sections of root tissue of all remaining plants were plated out onto PSM to verify the presence/absence of the pathogen.

5.2.5 Spectral transformation and data analysis

To consider the effects of *P. cinnamomi* infection on host plants, a number of different features within each spectra were analysed. This included an analysis of changes in water features, changes in vegetative indices that are used routinely in the assessment of plant stress, and general changes in reflectance in the UV-NIR region (Figure 5.1). Due to large amount of data collect during the trial and the slow onset of symptoms, data was only analysed from fortnightly intervals. This gave a total of nine collection dates including one before inoculation. Analysis was completed on untransformed data as the transformations trialed gave limited improvement to the data.

5.2.5.1 Analysis of water features

Spectra were loaded into ENVI (Exelis Visual Information Solution, Boulder, USA) and visually inspected for the location of water features. These features are so called because they quantify the amount of molecular water present in foliar tissue. Four water features were defined from the reflectance spectra and were as follows:. 1): 1,021-1,352 nm, 2): 1,227-1,761 nm, 3): 1,614-1,856 nm and 4): 1,797-2,258 nm (Figure 5.1). The depth and area of these features was then determined. This was done by first removing the continuum from each spectrum using a hull-quotients procedure (Clark and Roush, 1984). An automated feature extraction procedure was then used to extract depth and area. Although depth and area are correlated, both were determined as depth is effected by extraneous information in the spectral curve (noise) while asymmetry will alter the area of features of equal depth and thus both area and depth were analysed. One-way analysis of variance (ANOVA) was completed in R version 2.15.1 (R Core Team, 2012) on each water feature with a single treatment of four levels (control, water stress, inoculation, water stress + inoculation). Tukey's Honest Significant Difference (HSD) was used to separate treatment means. Each species was assessed individually, on each sample date with either the area or depth as the response variate.

5.2.5.2 Vegetation indices

A series of vegetation indices were calculated from the hyperspectral data to assess reflectance in the Visible Near-Infrared (VNIR) region as well as identify water stress and changes to anthocyanin production (Figure 5.1). The first derivative spectrum was also calculated and this was used to characterise the red edge slope and the area under the red edge peak. The indices and their method of calculation have been outlined in Table 5.3. One-way ANOVA and Tukey's HSD were used to assess differences between means.

Table 5.3 Vegetation indices used to assess changes in reflectance

Vegetation indices	Equation [A]	References
Green (reflectance in green region) [B]	μ (530-550)	
Red (reflectance in red region) [B]	μ (650-680)	
NIR (reflectance in near infrared region) [B]	μ (780-890)	
NDVI (normalised difference vegetative index	(NIR- Red) / (NIR + Red)	(Tucker, 1979)
RES (Red Edge Slope) [C]	Max 1d(Red)	(Elvidge and Chen, 1995; Cao *et al.,* 2013)
aREP (area of the Red Edge Peak) [C]	sum 1d(680-760)	(Filella and Penuelas, 1994)
ARI (Anthocyanin Reflectance Index)	(1/550)-(1/700)	(Gitelson et al., 2001)

[A] These calculations are based upon reflectance at the specified wavelength. [B] These are not indices as such but are regions within the spectrum that can change during plant stress (Jackson, 1986). [C] These calculations were made on the first derivative spectra also indicated by '1d' in the associated equation.

5.2.5.3 Principal Component Analysis

Reflectance in the region of 350-900 nm (UV-NIR, Figure 5.1) was used to assess changes in reflectance as a result of the water stress and inoculation. Data was analysed in Primer 6 version 6.1.13 (Primer-E, Ivybridge, UK) in both a raw and normalised format (mean of zero and a standard deviation of one). Principal component analysis (PCA) is suited to highly correlated variables such as hyperspectral reflectance and is used to reduce the dimensionality of data, that is, it reduces the number of variables required to explain the distribution of the data. In the case of hyperspectral data, PCA can identify which wavelengths contain the information that contribute to the variability required to separate samples into respective treatment groups. For the analysis, individual wavelengths were set as (independent) variables, with the spectral responses as the (dependant) samples (2 per plant). The four treatments were set as factors and used to interpret treatment separation in the projected data set.

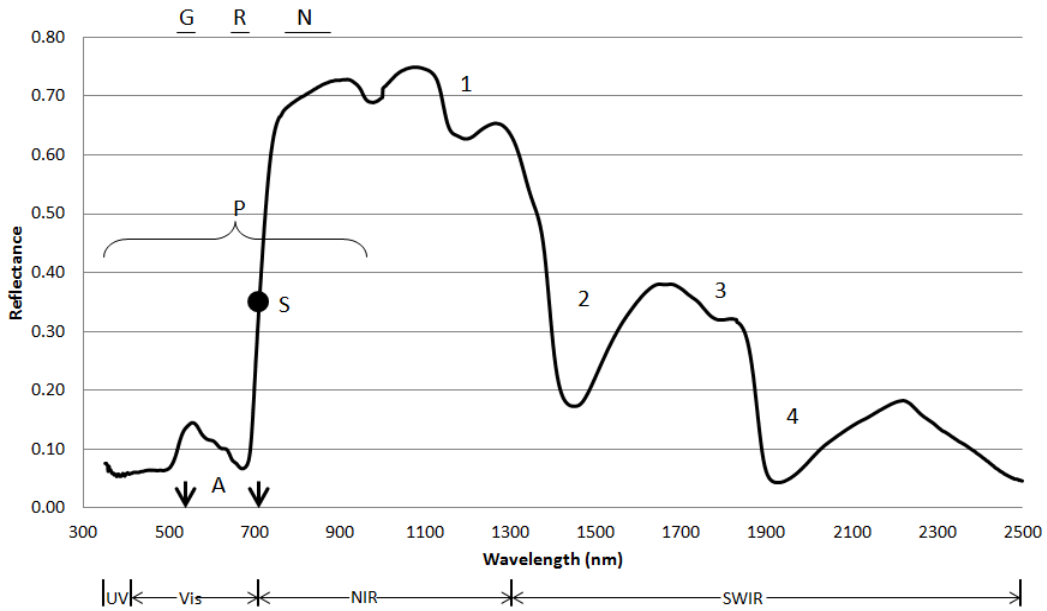


Figure 5.1 Spectral regions used in the hyperspectral analysis. An example leaf reflectance spectra is shown in the solid black line. The bars at (G), (R) and (N) represent the approximate locations of the green, red and NIR regions respectively, while the absorption features labelled (1)-(4) indicate the location of the four water features. The bracket (P) shows the approximate region of wavelengths used for the PCA. The point (S) shows the approximate location of the red edge inflection point (ie where the slope of the line starts decreasing) which, in the first derivative, was used to characterise the red edge slope. The arrows at (A) indicate the wavelengths used to calculate the ARI.

5.3 Results

Analysis of the water features and vegetation indicies idenetified many significant difference between treatements and over the suration of the trail. Each species had a unique response to incoculation and water stress which was evident via the visual observations and in their hyperspectral response as detected by the ANOVA (Appendix 8.6, 8.7) and the PCA.

5.3.1 Species response to inoculation and water stress

No plant death occurred in the inoculation treatment during the 15 weeks of the trial, however individual plants in either water stressed treatment died within 6 weeks (Table 5.4). Symptoms of *P. cinnamomi* infection became evident in individuals of both inoculated groups as the trial progressed.

Table 5.4 Number of individual plants that died during the trial in each treatment.

Species	Control	Water Stressed (WS)	Inoculated (P)	WS+P
A. costata	0/12	9/16	0/13	10/15
B. serrata	0/13	8/11	0/13	7/13
D. revoluta	0/10	0/10	0/10	1/10
E. piperita	1/11	7/14	0/13	6/11
L. longifolia	0/10	4/10	0/10	3/10

The number of dead plants have been expressed as a fraction of the total number of individuals in each treatment.

Water stress became apparent in *A. costata* within the first month of the trial, however the response to *P. cinnamomi* infection did not appear until 64 days after infection (d.a.i.) at which time individual leaves appeared chlorotic in the inoculated treatments. Shortly after this, at 78 d.a.i., water stress was quite severe in both water stress treatments and several individuals had already died (Table 5.2). In the final week of measurements (106 d.a.i.), the leaves of several *A. costata* plants had discoloured turning red and brown, while leaf and tip necrosis was also prevalent (Figure 5.2).

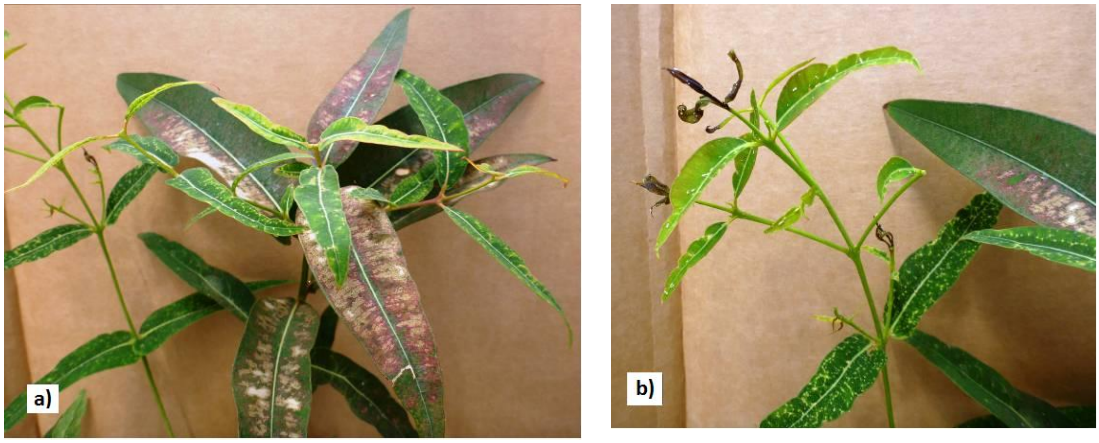


Figure 5.2 Leaf discolouration and tip dieback in *A. costata* infected with *P. cinnamomi*. (a) leaf discolouration, chlorosis and necrosis and (b) shoot necrosis as observed in the individual plant AC 38 from which *P. cinnamomi* was isolated at the end of the trial.

There was no clear indication of infection in *B. serrata* at all during the trial. Many of the inoculated plants appeared chlorotic and later discoloured, but these same features were seen in uninoculated plants also (Figure 5.3). Water stress, however, did become apparent with leaves desiccating and becoming necrotic. A number of water stressed plants died before the conclusion of the trial (Table 5.4, Figure 5.3).

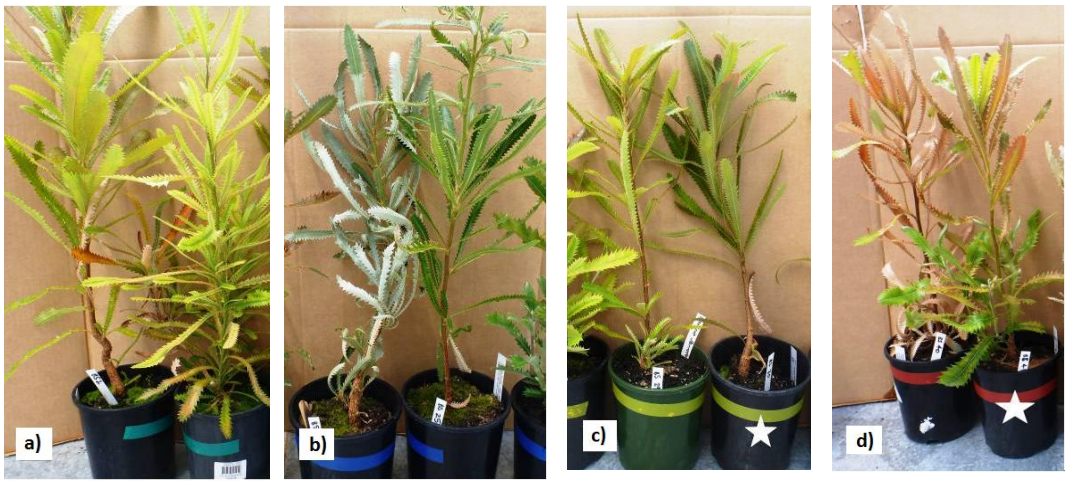


Figure 5.3 Visual comparison of four treatments on *B. serrata* 106 d.a.i. Leaf chlorosis and discolouration was evident in control plants (a) inoculated plants (c) and in the combination treatment (d). Leaf desiccation was evident in the water stressed plants (b) and in the combination treatment (not seen here). White stars in (c) and (d) indicate plants from which *P. cinnamomi* was re-isolated at the end of the trial.

Dianella revoluta also gave no clear visual indication of *P. cinnamomi* infection during the trial (Figure 5.4). Water stress began occurring midway through the trial resulting in leaf desiccation and the formation of necrotic lesions. These lesions were evident in all treatments except the control indicating their symptoms may have resulted from either water stress or inoculation.

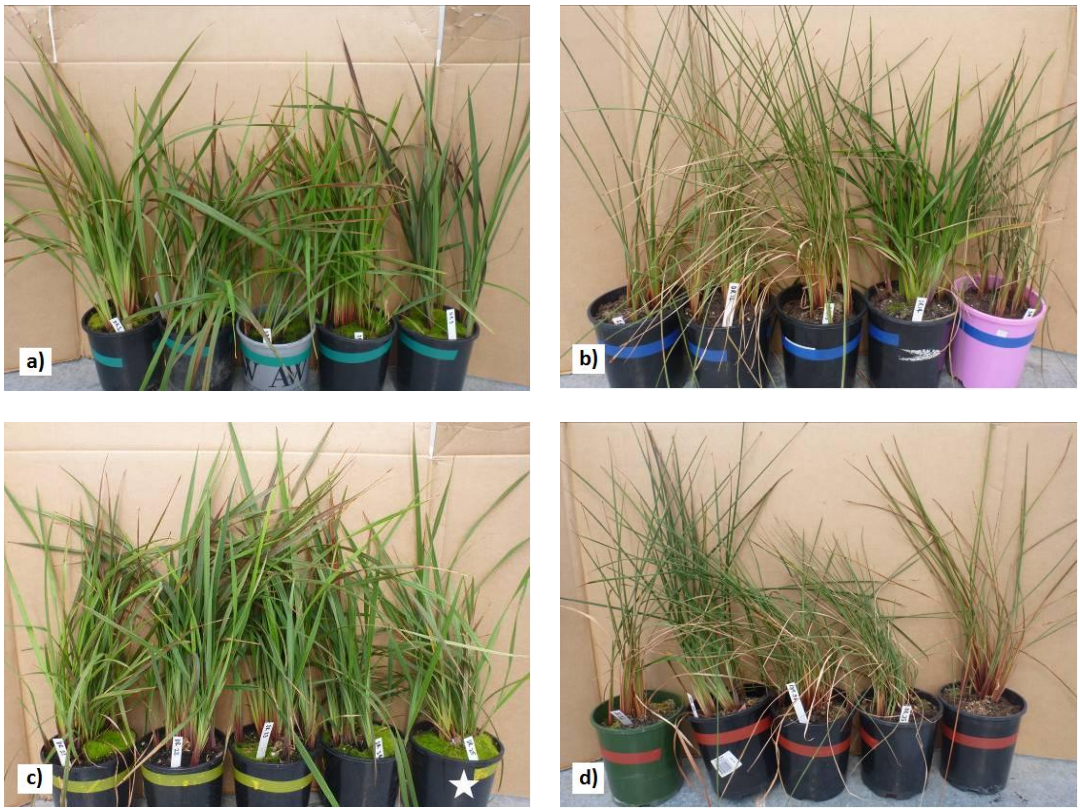


Figure 5.4 Symptoms of water stress in *D. revoluta* 106 d.a.i. Wilting was evident in the water stressed plants (b) and in the water stress and inoculated plants (d). There were no obvious differences between the inoculated plants (c) and the controls (a). White star in (c) indicates a plant from which *P. cinnamomi* was successfully isolated at the end of the trial.

Water stress started to become evident in *E. piperita* one month into the trial. Individuals of *E. piperita* were the first to shown signs of infection with new growth on some individuals appearing chlorotic 36 d.a.i (Figure 5.5). This was followed by leaf necrosis on inoculated plants in both the inoculated and combination treatments 50 d.a.i. (Figure 5.5). By 64 d.a.i., many leaves on *E. piperita* plants had died although they remained attached to the stem. Several plants exposed to water stress had died by this time (Table 5.4). Towards the end of the trial at 92 d.a.i., inoculated plants of *E. piperita* were smaller and less vegetated than control plants, often leaves had abscised and shoots of severally affected plants had also become necrotic (Figure 5.6).

Figure 5.5 Symptoms of *P. cinnamomi* infection and water stress in *E. piperita* 50 d.a.i. (a) *E. piperita* seedlings in the inoculated treatment group with black arrows indicating chlorotic tissue; (b) seedlings in the combination treatment group with arrows indicating necrotic leaves. White stars indicate plants from which *P. cinnamomi* was re-isolated.

Figure 5.6 Symptoms of *P. cinnamomi* infection and water stress in *E. piperita* 92 d.a.i. (a) *E. piperita* plants in the control group; (b) plants in the inoculated treatment group with arrows indicating chlorotic leaves and tip necrosis; (c) plants in the combination treatment (water stressed + inoculated). Arrows indicate tip and leaf necrosis, and leaf abscission. It can be seen that plants in the inoculated treatment group are shorter (when compared to the background of the photos) and have less foliage than the control group. White stars in (b) and (c) indicate plants from which *P. cinnamomi* was re-isolated.

Lomandra longifolia showed no sign of infection at all during the trial (Figure 5.7). Water stress however did cause wilting which was evident 78 d.a.i. Several water stressed plants died during the trial (Table 5.4).

Figure 5.7 Symptoms of water stress in *L. longifolia* 106 d.a.i. Wilting was evident in the water stressed plants (b) and in the water stress and inoculated plants (d). There were no obvious differences between the inoculated plants (c) when compared to the control (a).

5.3.2 Isolation results

Phytophthora cinnamomi was infrequently re-isolated and in the case of *L. longifolia* not isolated at all. Despite efforts to keep inoculated and uninoculated plants separate and strict hygiene during the measurement of reflectance, several plants in the uninoculated treatments had been infected with *P. cinnamomi* either before or during the trial. The total number of positive isolated from each treatment is outlined in Table 5.5.

Table 5.5 Positive isolation percentage for *P. cinnamomi* from each species and treatment.

Species	Control %	Water Stressed (WS) %	Inoculated (P) %	WS+P %
A. costata	50	12.5	61.5	6.6
B. serrata	0	0	7.6	7.6
D. revoluta	10	0	10	10
E. piperita	27	7	46	36

5.3.3 Hyperspectral leaf response

Water stress and inoculation were both found to alter the water features, vegetation indices and the PCA. Changes in these measures as a result of infection appeared earlier in the trial, but were generally subtle. Once water stress began to occur, it became the dominant variable influencing changes in the spectral regions analysed. Although plants were randomly allocated to treatment groups on the day of inoculation, there were a number of significant differences between treatments observed. The responses of individual species to the stressors was quite variable which impacted the statistical outcomes of many of the ANOVAs.

5.3.3.1 The effect of inoculation and water stress in species water features

Absorption feature area and depth, correlates of available water, were both influenced by inoculation and water stress. Inoculation was generally observed to initially increase the depth and area of water features while water stress decreased them. The combination treatment generally decreased feature depth and area, but was then either slightly higher than the water stress treatment indicating a correction, or slightly lower indicating a cumulative decrease in reflectance caused by both stressors together. As the trial continued, species response to stress varied, resulting in increasing or decreasing feature characteristics.

Increases in feature size in *A. costata* cause by *P. cinnamomi* infection were present at 78, 92 and 106 d.a.i. alongside decreases caused by water stress (Figure 5.8). By 92 d.a.i., only the water stress treatments were significantly different from the controls (Figure 5.8). The water stress treatments were generally different from the control for the remainder of the trial in all water features except 1,614 nm, however a large amount of variability in the response of individual plants to the pathogen and water stress led to differences being non-significant (Figure 5.8).

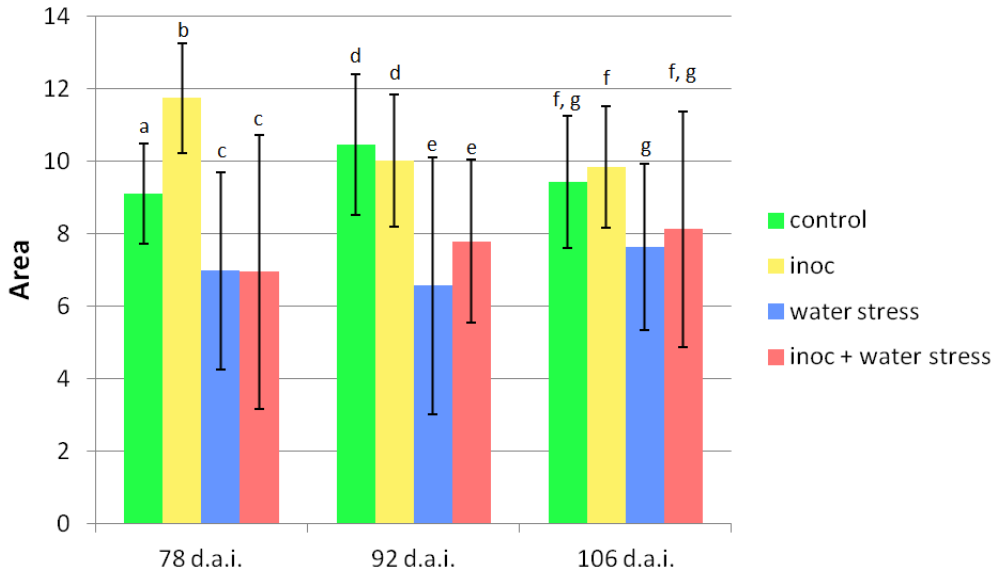


Figure 5.8 Average area (+/- 1 standard deviation, SD) of the water feature at 1,021 nm in *A. costata* at 78, 92 and 106 d.a.i. Columns with different letters above them indicate significantly different treatment means ($P < 0.05$) as determined by Tukey's HSD. For example, at 78 d.a.i., the two treatments labelled (c) are statistically the same, while the treatments labelled (a) and (b) are statistically different from each other and from (c).

An increase in the size of water features in *B. serrata* due to *P. cinnamomi* infection was observed 6 d.a.i. In the first water feature (1,027 nm), water stress also increased feature size and thus the three stress treatments were only significantly different from the control and not from each other. Feature size subsequently decreased in the inoculated *B. serrata* plants while it increased further in the combination treatment 50 d.a.i. in the first water feature (1,021 nm). No other significant effects were observed in *B. serrata* until 78 d.a.i. at which time significant differences were detected in three of the four water features. At this point, changes in feature size caused by *P. cinnamomi* could not be delineated from the water stressed treatments. Water stress continued to decrease depth and area in each of the water features (Figure 5.9). At the end of the trial, the mean values of feature area were similar, except in the combination treatment which was significantly different from the non-water stressed treatments (Figure 5.9).

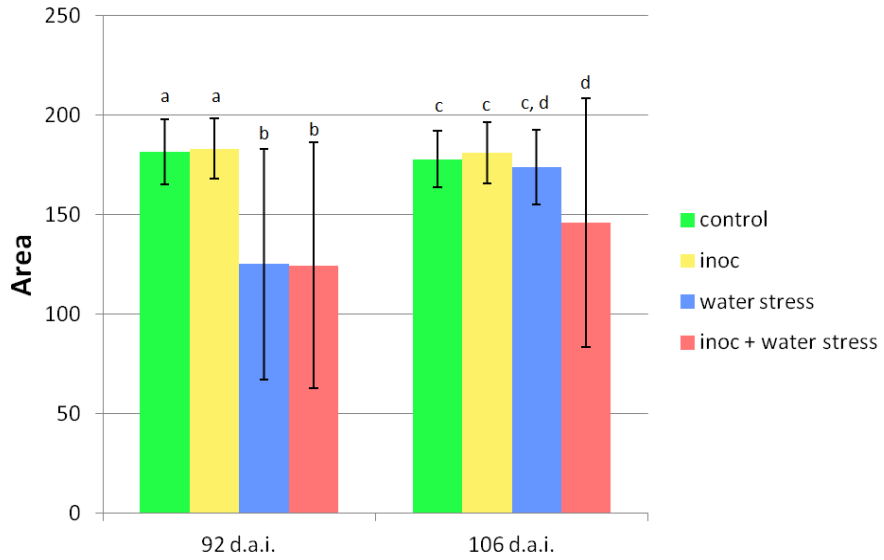


Figure 5.9 Average area (+/- 1 SD) of the 1,797 nm water features in *B. serrata* in response to water stress and infection at 92 and 106 d.a.i. Columns with different letters above them indicate significantly different treatment means ($P < 0.05$).

An increase in the size of water features associated with *P. cinnamomi* infection was observed in *D. revoluta* 6 d.a.i., however this differences was not significant. Water stress also increased the size of the four water features, even more so in the combination treatment (Figure 5.10) which was often significantly different from the control throughout the trial. Inoculation decreased the size of the second (1,227 nm) and fourth features (1,797 nm) 36 d.a.i. (Figure 5.10), but increased it in the other two, in which case treatment means were statistically the same as the control. Water stress became the dominant feature 50 d.a.i. decreasing feature size in both of the water stress treatments. By 78 d.a.i., inoculation increased feature size, while the combination treatment decreased it (Figure 5.10). For the remainder of the trial, water stress significantly decreased the size of all four water features.

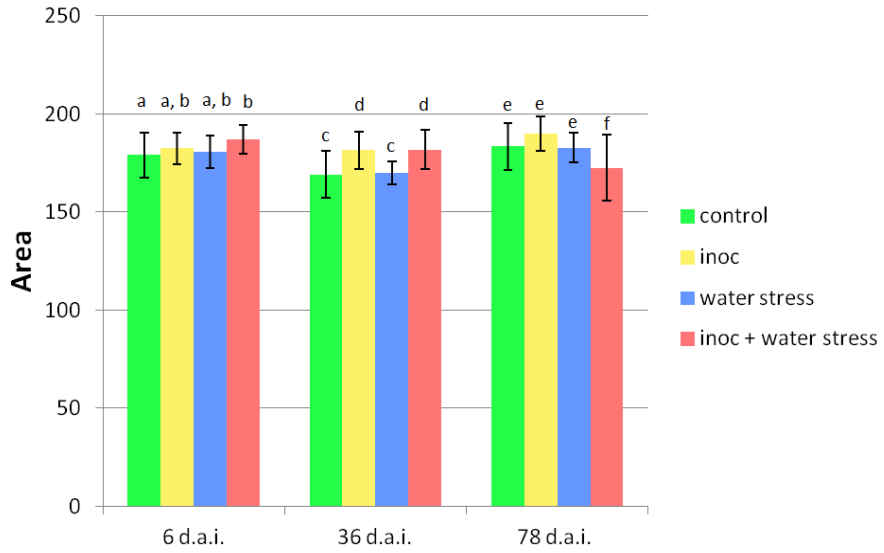


Figure 5.10 Average area (+/- 1 SD) of the 1,797 nm water feature detected in *D. revoluta* associated with water stress and infection at 6, 36 and 78 d.a.i. Columns with different letters above them indicate significantly different treatment means ($P < 0.05$).

Plants of *E. piperita* followed a similar pattern or changing reflectance over time as was seen in *D. revoluta*. Within the first month of the trial inoculation had caused a significant increase in reflectance, however neither water stress treatment changed reflectance. By 50 d.a.i. however, water stress was detectable in the first and second water features (1,021 nm, 1,227 nm) but was still not significantly different from the control (Figure 5.11). Toward the end of the trial, again water stress became the dominant treatment in all features creating a significant difference in reflectance when compared to the control (Figure 5.11). Inoculation increased or decreased reflectance when compared to the control in the second and third feature (1,227 nm, 1,614 nm) however these individual differences were not significant.

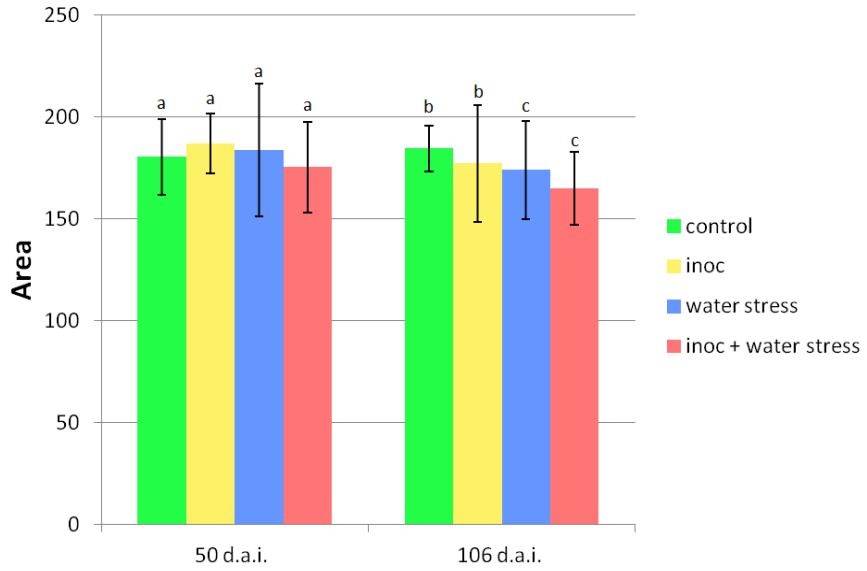


Figure 5.11 Average area (+/- 1 SD) of the 1,227 nm water feature detected in *E. piperita* associated with inoculation and water stress at 50 and 106 d.a.i. Columns with different letters above them indicate significantly different treatment means ($P < 0.05$).

The response of *L. longifolia* to water stress and infection was similar again to the other plants tested in that inoculation initially increased the size of the water features in the first month of the trial. After this water stress became the dominant treatment and was significantly different from the control in most features. At the conclusion of the trial, water stress was still the dominant feature, while infection caused slight but insignificant decreases in the area of the first and second water features (1,021 nm, 1,227 nm) which had a cumulative effect between the two stressors (Figure 5.12).

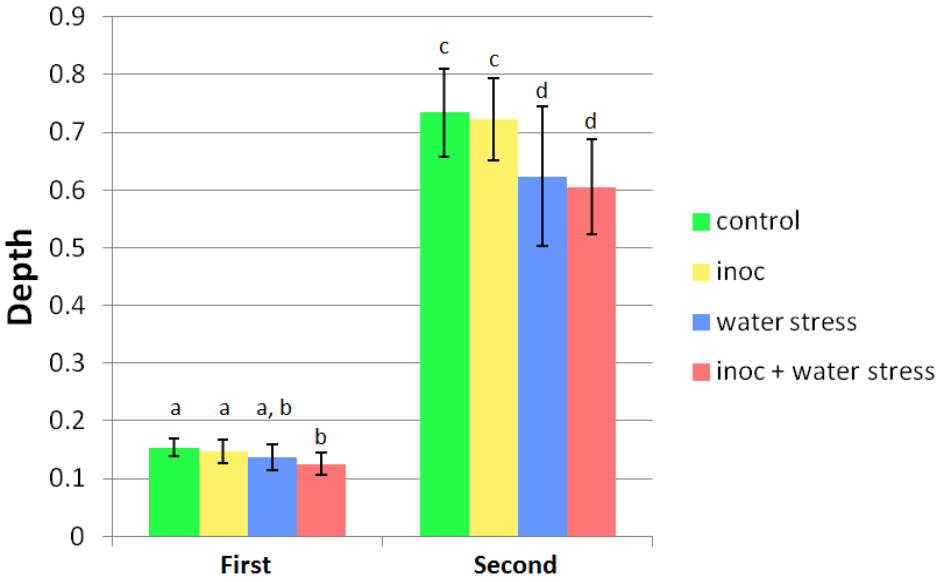


Figure 5.12 Average depth (+/- 1 SD) of the first and second water feature of *L. longifolia* associated with inoculation and water stress at 106 d.a.i. Columns with different letters above them indicate significantly different treatment means ($P < 0.05$).

5.3.3.2 The effect of inoculation and water stress in species vegetation indices

The vegetation indices used to assess changes in reflectance caused by either water stress or *P. cinnamomi* infection were able to identify significant interactions. Changes in vegetation indices were variable for the different species and on different measurement dates. Reflectance in the green and red region was useful for detecting infection however the response was species specific. The RES and the aREP were most useful for the detection of *P. cinnamomi* infection. Significant differences were evident in both of these indices by the third month of the trial. The ARI was useful for separating out the combination treatment while NDVI was useful in the separation of both water stress treatments.

Reflectance in the coloured region of A. costata also gave an indication of treatment effects. A cumulative and significant effect of the two stressors in the combination treatment was evident in green reflectance of 78 d.a.i. (Figure 5.13). By 92 d.a.i., increases in red reflectance associated with the combination treatment were however no longer significantly different from the control (Figure 5.13). This was also the case for the RES. By the end of the trial, the four treatments were evident in the ARI, but differences were not significant.

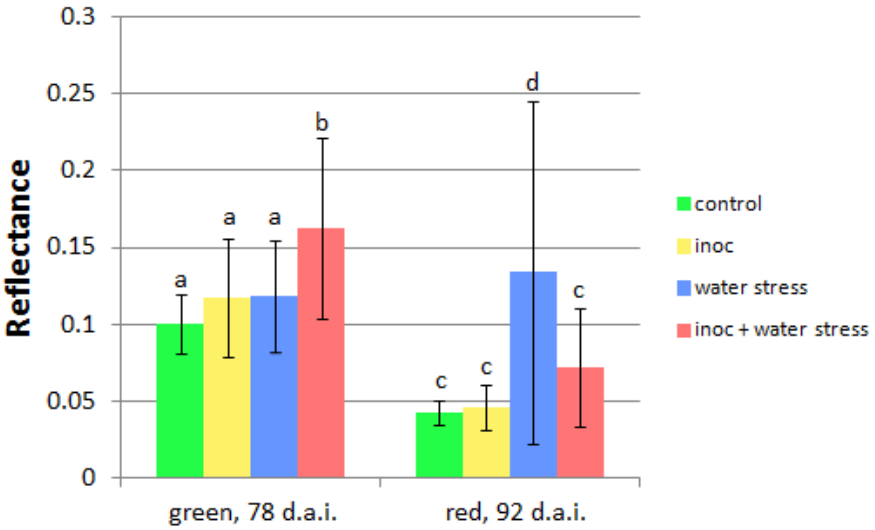


Figure 5.13 Average (+/- 1 SD) green reflectance at 78 d.a.i. and red reflectance at 92 d.a.i. of *A. costata* associated with inoculation and water stress. Columns with different letters above them indicate significantly different treatment means ($P < 0.05$).

The effect of inoculation and water stress on *B. serrata* was not obvious until later in the trial 92 d.a.i. Inoculation caused a clear, but non-significant increase in green

reflectance (Figure 5.14) while water stress was evident in the aREP (Figure 5.14). A cumulative effects of the two stressors was evident in NDVI (Figure 5.14) however both water stress treatments were statistically the same, as was the control with the inoculated treatments. Unlike *A. costata,* water stress and inoculation reduced the ARI in *B. serrata* at the conclusion of the trial, however, only the water stress treatments were significantly different from the control but were not significantly different from each other.

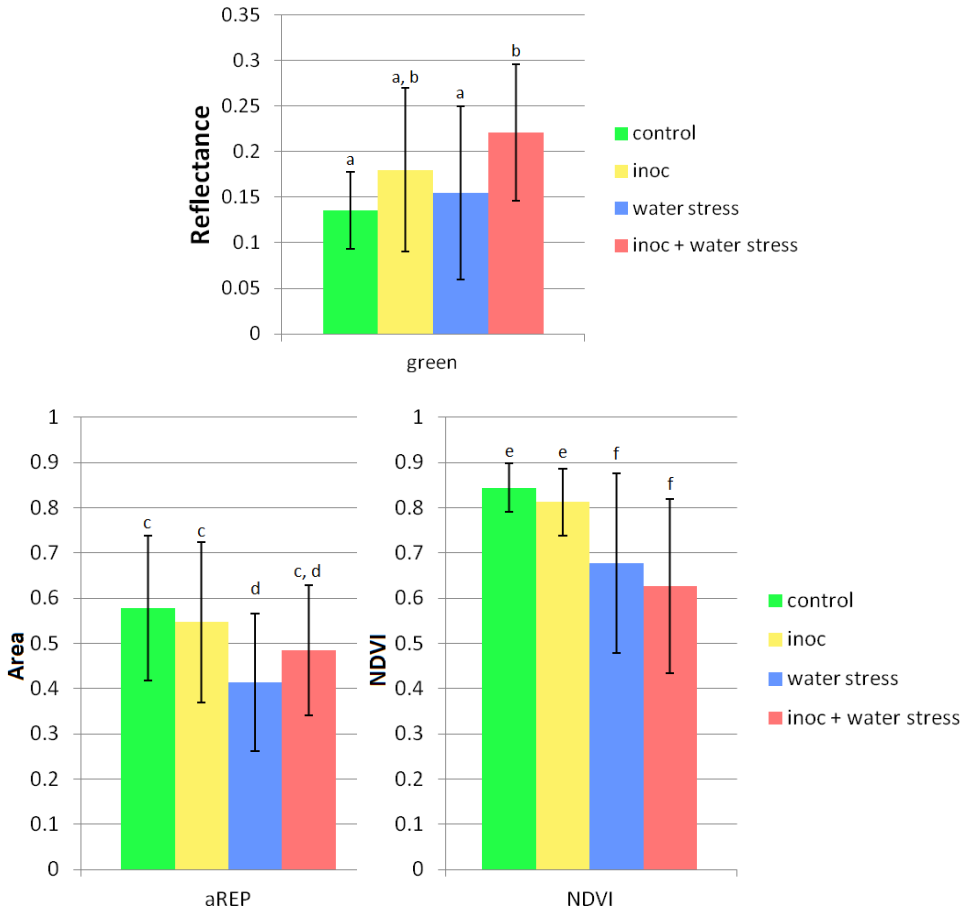


Figure 5.14 Average (+/- 1 SD) green reflectance, aREP, and NDVI in *B. serrata* associated with inoculation and water stress at 92 d.a.i. Columns with different letters above them indicate significantly different treatment means ($P < 0.05$).

The effect of inoculation on *D. revoluta* was first detected in the vegetation indices 78 d.a.i. at which time the RES of the inoculated plants was significantly different from the other three treatments. Water stress was also detectable from the RES (Figure 5.15), however the difference was not significant. The combination treatment was evident also at this time in the ARI in which plants exposed to water stress and inoculation together had a significantly higher mean anthocyanin reflectance compared to the other three treatments

(Figure 5.15). By the end of the trial, water stress again became the dominant feature and any differences between means caused by inoculation were no longer significant.

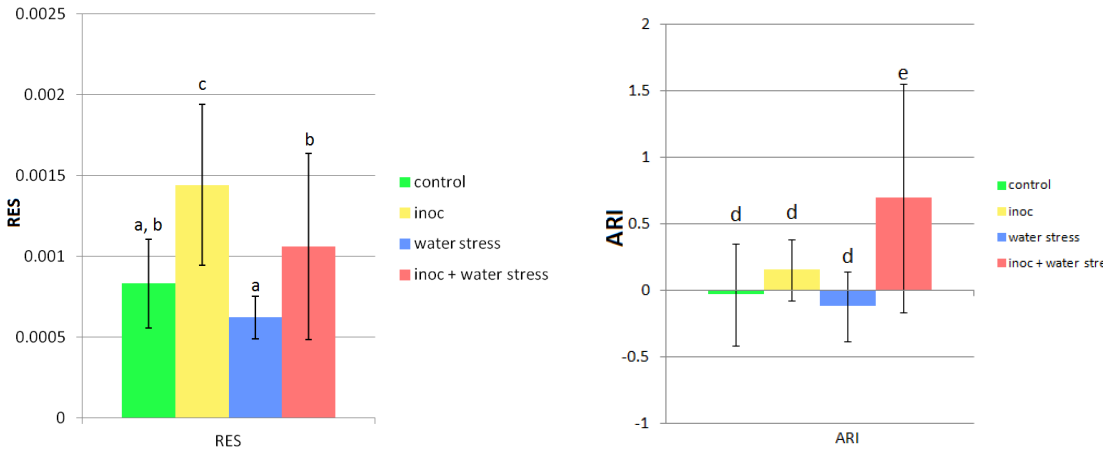


Figure 5.15 Average (+/- 1 SD) RES and ARI of *D. revoluta* associated with inoculation and water stress at 78 d.a.i. Columns with different letters above them indicate significantly different treatment means ($P < 0.05$).

Changes to leaf reflectance of *E. piperita* as a result of water stress and/or inoculation were typically not significant. The response of individuals to stress in the trial was highly variable and not consistent between measurement dates. Towards the end of the trial (92 d.a.i.), plants inoculated with *P. cinnamomi* had higher green reflectance, but where not significantly different from the control. However, plants in the combination treatment had lower green reflectance but they were not significantly different from the control (Figure 5.16). Reflectance in the red region increased above the control in the water stressed and in the inoculated treatment, however the combination treatment was not significantly different from the control (Figure 5.16).

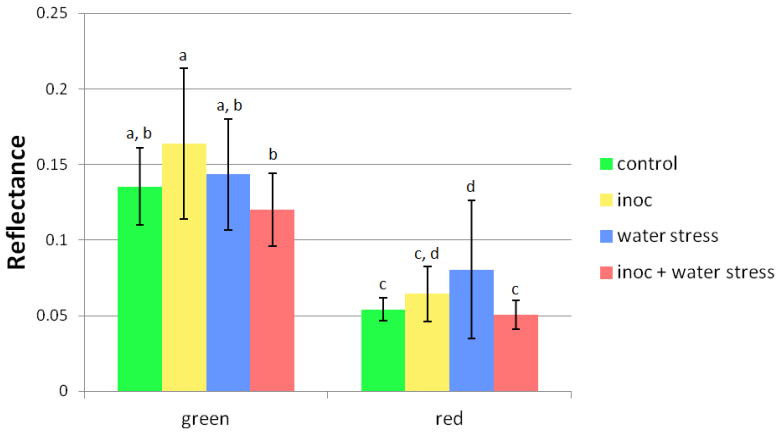


Figure 5.16 Average (+/- 1 SD) green and red reflectance of *E. piperita* associated with inoculation and water stress at 92 d.a.i. Columns with different letters above them indicate significantly different treatment means ($P < 0.05$).

Evidence of disease and water stress was detectable via green reflectance and the RES of *L. longifolia* 64 d.a.i. such that infection increased both, while water stress decreased them. The combination treatment, however, was not significantly different from the control (Figure 5.17). By 78 d.a.i. water stress was evident in the RES, but the combination treatment was not significantly different to the control (Figure 5.17). Infection was not as clear in any of the indices 92 d.a.i. as water stress became dominant being particularly evident in the ARI and NDVI. The combination treatment could be separated from the other three treatments via green reflectance however non-water stressed inoculated plants were not significantly different from the control.

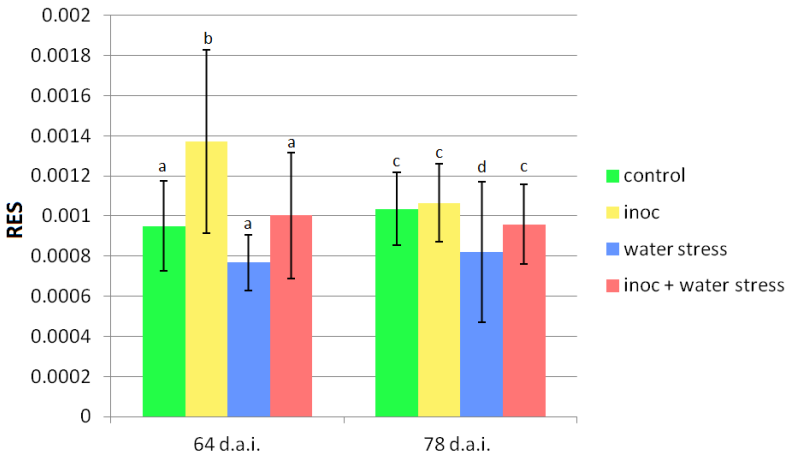


Figure 5.17 Average RES (+/- 1 SD) at 64 and 78 d.a.i. of *L. longifolia* associated with inoculation and water stress. Columns with different letters above them indicate significantly different treatment means ($P < 0.05$).

5.3.3.3 Changes in reflectance detected via PCA.

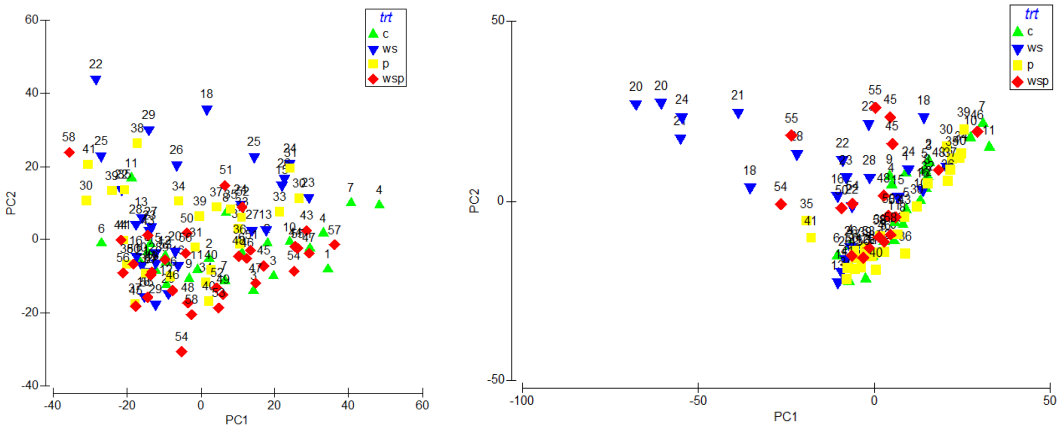
Separation of treatments from one another was detected in the PCA for each of the species with varying degrees of delineation. Separation between treatments on the PCA plot was evident as early as 6 d.a.i. in absolute reflectance and the first derivative. The separation of treatments became more obvious as the trial continued with at least some separation identified in each of the species 92 d.a.i. Separation was still evident at the conclusion of the trial 106 d.a.i., however due to plant death in the water stress treatments, delineation of samples from the different treatments was not as effective. More than 95% of the dimensionality of samples could be explained in three principal components (Table 5.6). The greatest variable contribution in the first and second component was associated with the red region of the spectrum while the third came from the UV, visible or NIR regions (Table 5.6).

Table 5.6 The ten spectral bands that gave the highest degree of treatment separation in each species as identified via Principal Component Analysis, using first derivative data, 92 d.a.i.

Species	PC1	PC2	PC3	Total variance explained, %
A. costata	718-727 (70.6)	730-739 (22.1)	517-526 (3.7)	96.5
B. serrata	709-718 (59.4)	688-697 (31.4)	424-433 (5.7)	96.6
D. revoluta	718-727 (64)	734-743 (30.2)	753-762 (3.8)	98
E. piperita	711-720 (71.3)	689-698 (19.1)	385-394 (96.3)	96.3
L. longifolia	726-735 (60.1)	693-702 (32.7)	515-524, 724 (4.2)	97.1

Principal components (PC) are expressed as a range of wavelengths measured in nanometres. Brackets under each spectral range indicate the percentage contribution that each principal component made to the analysis of that species. The total variance explained indicates what proportion of the input data is explained in the three components listed in the table for each species.

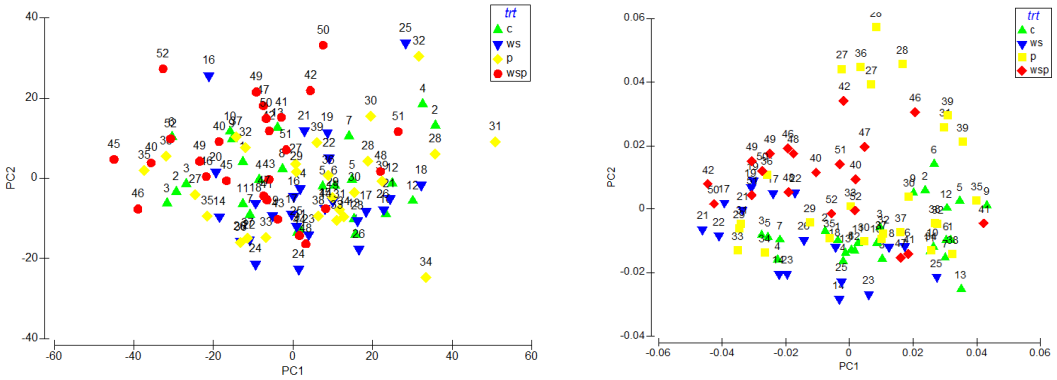
Treatment separation in the normalised reflectance data of *A. costata* became evident 64 d.a.i at which point the water stress and inoculation treatments were beginning to separate from the control and combination treatment (Figure 5.18). The inoculated treatment merged back toward these 92 d.a.i. leaving the water stress samples separating out on their own (Figure 5.18).



64 d.a.i. 92 d.a.i.

Figure 5.18 Arrangement of *A. costata* samples in multidimensional space follow principal component analysis. The greater the distance between the samples, the greater the variation between them. The analysis in this case has been completed on normalised reflectance data at 64 and 92 d.a.i. The numbers above the samples indicate the individual plant ID. Samples are in pairs as reflectance was measured twice. The key indicates the analysis factors (treatments) as follows: c: control; ws: water stress; p: inoculated; wsp: water stress + inoculation.

Banksia serrata was one of the earlier plants to show treatment separation in the PCA 6 d.a.i. at which point several plants in the inoculated treatment group started to separate from the other samples based on absolute reflectance (Figure 5.19). Towards the end of the trial (92 d.a.i.), several inoculated individuals had separated completely on the PCA plot from all other treatments when considering the first derivative data, while the combination treatment had begun to group, and the water stress plants were congregating, although they remained mixed among individuals of other treatments (Figure 5.19).



6 d.a.i. 92 dai

Figure 5.19 Arrangement of *B. serrata* samples on the PCA plot. The plot at 6 d.a.i. is of normalised absolute reflectance, while the plot at 92 d.a.i. is of the first derivative spectra. The numbers above the samples indicate the individual plant ID. Samples are in pairs as reflectance was measured twice. The key indicates factors as follows: c: control; ws: water stress; p: inoculated; wsp: water stress + inoculation.

The effect of inoculation and water stress on *D. revoluta* became apparent 6 d.a.i. at which time the treatment groups were starting to congregate in the first derivative spectra, although all samples were still intermixed. Later in the trial, 92 d.a.i., separation of both inoculation treatments was evident in a number of samples on two perpendicular axes. The control, and the water stress samples grouped, but appeared to be following the inoculated and combination treatments respectively (Figure 5.20). By the conclusion of the trial the water stressed samples had moved onto a common axis with samples in the combination treatment, while the control samples remained partially mixed with the inoculated samples.

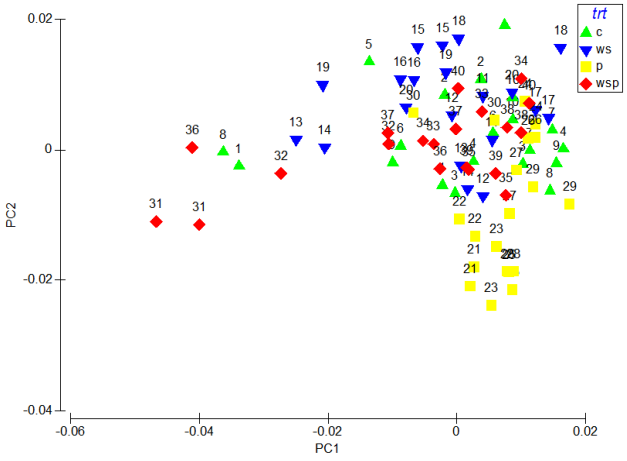
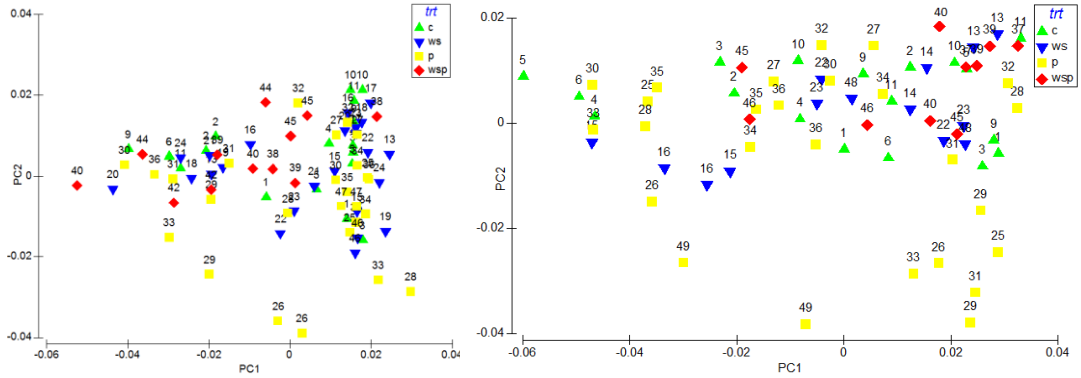


Figure 5.20 Arrangement of *D. revoluta* samples on the PCA plot using the first derivative spectra at 92 d.a.i. The numbers above samples indicate the individual plant ID. Samples are in pairs as reflectance was measured twice. The key indicates factors as follows: c: control; ws: water stress; p: inoculated; wsp: water stress + inoculation.

Separation of treatments was slower in *E. piperita* in which it was not evident until 64 d.a.i. in the first (Figure 5.21) and second derivative spectra. Although many sample remained intermixed, the majority of inoculated plants were moving along a single axis with a number separating into a group (26, 28, 29, 33). Samples in the combination treatment appeared to be moving along a perpendicular axis. The majority of the control treatments remained in a group with the water stressed treatments being intermixed. This same patterning was evident 92 d.a.i. however all samples had separated further from each other (Figure 5.21). At the conclusion of the trial, there were few samples remaining in the water stressed treatment and those that did were intermixed.



64 d.a.i. 92 d.a.i.

Figure 5.21 Arrangement of the *E. piperita* samples on the PCA plot using the first derivative spectra 64 and 92 d.a.i. The numbers above samples indicate the individual plant ID. Samples are in pairs as reflectance was measured twice. The key indicates factors as follows: c: control; ws: water stress; p: inoculated; wsp: water stress +

Lomandra longifolia showed the least amount of treatment separation in the PCA. Separation of any kind was not evident until 92 d.a.i. at which time the combination treatment had started to separate from the other three treatments being furthest away from the control. This was evident in the reflectance spectra and the first and second derivatives. Three plants in the combination treatment (33, 36, 37) and one plant in the water stressed treatment (19) had formed a separate group away from the other samples (Figure 5.22), suggesting some separation on the basis of water stress.

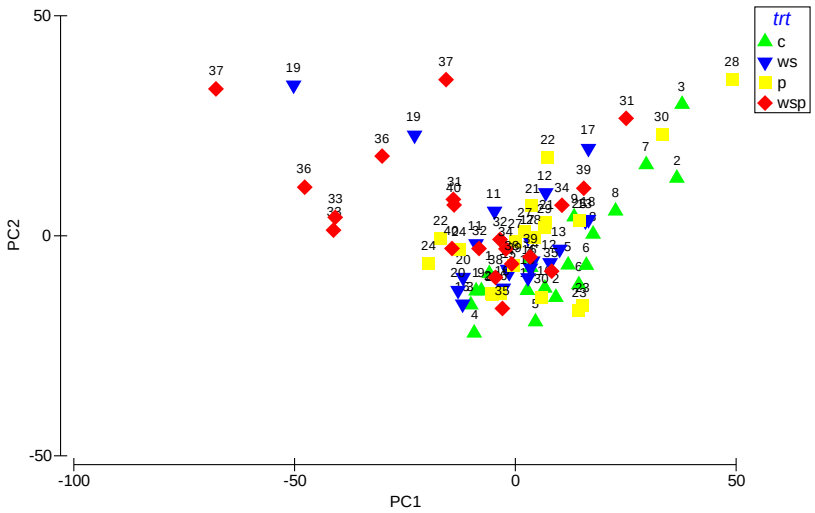


Figure 5.22 Arrangement of the *L. longifolia* samples on the PCA plot using normalised reflectance data 92 d.a.i. The numbers above samples indicate the individual plant id. Samples are in pairs as reflectance was measured twice. The key indicates factors as follows: c: control; ws: water stress; p: inoculated; wsp: water stress + inoculation.

5.4 Discussion

Detection of water stress and *P. cinnamomi* infection was possible, in some cases, from hyperspectral reflectance by analysing water features, vegetation indices and narrow band reflectance reduced via PCA. Disease was detected in four of the five species; *L. longifolia* being the exception. Individual species responses were unique with respect to both water stress and inoculation. Additionally, the visual symptoms of stress varied as did the changes in reflectance. The species responses varied over time as both disease and water stress progressed.

Changes in water features indicative of water stress and inoculation were found, with individual features alluding to the different treatments. The affect of inoculation was evident in the first (1,021 nm) and second (1,227 nm) water features in which inoculation decreased feature size. The third water feature (1,614 nm) was useful in identifying the inoculated treatments however differences were often not significant. The fourth water feature (1,797 nm) showed the most variation in the five species and indicated an increase in feature size associated with inoculation and decrease in water stress, the latter of which caused a greater change in reflectance in this region.

The ability to identify water stress via these treatments is not surprising given that they are intended to qualify leaf water content. The use of water features to assess disease is quite novel yet seemingly logical here given that *P. cinnamomi* infection causes water stress. Pu *et al,*(2008a) provides a brief insight into how disease may be detected by water

features suggesting that, the so called 'minor' water features (as a pose to the major water features centred at 1,450 nm and 1,940 nm), are less sensitive to changes in relative water content but relate to plant health in a greater sense. It was also noted by Pu *et al,* (2008a) that infection of Oak leaves with *P. ramorum* resulted in an initial decrease in reflectance (resulting in an increase in feature size) prior to a decrease, an effect which was observed here. Even when water stress decreased reflectance in the earlier stages of the trial, a slight increase in reflectance was often visible in the combination treatments.

Disease and water stress were also detectable in the vegetation indices with the most consistent changes in reflectance seen in the RES which detected inoculation and the ARI which detected waters stress. With just these two indices it was possible to see all four treatments separately in *B. serrata* and *E. piperita* 92 d.a.i. Treatments were not all significantly different, which may have been due to the number of replicates used in the trial. Consideration of another feature such as NDVI would allow for the separation of the four treatments. Anthocyanins produced in leaves in response to environmental stress can be detected at 550 nm (Gitelson *et al.,* 2001). Therefore an increase in the ARI as a result of stress was expected and allowed for the identification of water stress in all five species. Slight and additional increases in the ARI were evident in the water stress treatment and combination treatment respectively, highlighting the ability of the ARI to delineate both. The ARI has previously been found useful to detect rust in wheat leaves, and, when used in conjunction with a secondary indices can delineate between yellow rust, stem rust and leaf rust of wheat (Devadas *et al.,* 2009). Three different diseases in Sugar Beet have also been detected with the ARI although not delineated from each other (Mahlein *et al.,* 2013).

Numerous indices associated with the red edge have proven useful in the identification and quantification of disease. Here, disease was associated with increased reflectance in the red-NIR region and, thus, increased the RES in all five species, although these increases were not always significantly different from the control. Increasing slope of the red edge has been associated with infection of Sugar Beet roots by *Rhizoctonia solani* (Reynolds *et al.,* 2012), correlated with the severity of Powdery Mildew on Wheat leaves caused by *Blumeria graminis* (Cao *et al.,* 2013) and used to detect bacterial leaf blight of rice caused by *Xanthomonas oryzae* pv. *oryzae* (Yang, 2010).

The green region of the spectrum also gave clear and significant indications of disease and water stress in the five species, however the response of each species was different making it difficult to characterise the impact of *P. cinnamomi* infection within this region. Increases in green reflectance were pronounced for species with greater susceptibility 92 d.a.i. such as *B. serrata* and *D. revoluta* but not detectable in the field

resistant species *L. longifolia*. Increasing green reflectance caused by stress such as disease results from a breakdown of chlorophyll (Barrett and Curtis, 1992). Single wavelengths in the green region have been found useful in the identification of other diseases including 540 nm for the detection of P*. ramorum* infection in potatoes leaves (Ray *et al.,* 2011), 560 nm for the detection of Citrus Greening (Huanglongbing) (Sankaran *et al.,* 2013), and 495 nm and 496 nm for the detection of Ganoderma infection in Oil Palms (Shafri *et al.,* 2011). Spectral changes within the green region have been used to indentify Fusarium Head Blight of Wheat (550-560 nm)(Bauriegel *et al.,* 2011), as well as the violet-blue-green region (400-500 nm) and green-yellow region (500-590 nm) to identify Yellow Leaf Virus in Sugarcane (Grisham *et al.,* 2010).

Numerous studies have found red reflectance to be useful in the detection and quantification of disease, however here the red region was dominated by the effects of water stress. Any increases in red associated with disease were not significant 92 d.a.i. except for *D. revoluta* in which case it was the combination treatment that was significantly different. An overall increase in reflectance in the visible range caused by disease was evident and consist with numerous other studies (Zhang *et al.,* 2003; Yang, 2010; Reynolds *et al.,* 2012; Mahlein *et al.,* 2013). Interestingly, infection of Tobacco roots by *P. nicotianae* is reported to decrease visible reflectance (Yusuf and He, 2011), a finding the author was able to validate with several studies highlighting disease-specific variation in the visible region.

Reflectance in the NIR region was only found to be significantly different between treatments five times during the trial and only once a the end of the study 106 d.a.i. This was surprising as NIR reflectance is often the most useful spectral region for disease detection. Mahlein *et al.* (2012; 2013) was able to delineate between Cercospora Leaf Spot, rust and Powdery Mildew on Sugar Beet based on NIR reflectance when considered with respect to the visible region. Yusuf and He (2011) found NIR reflectance continued to decrease as disease became more severe while Yang (2010) found the NIR, short-wave infrared and mid-infrared regions were most sensitive to changes caused by disease. Yang *et al.* (2010) also found the NIR region quite useful in the detection of Cotton root rot, suggesting that the NIR region is suitable for the detection of root infection. Although the NIR region is quite useful in the detection of stress and disease, it is likely to only be useful in time series analysis such that the magnitude of change in NIR reflectance can be determined and then used to separate healthy and diseased vegetation.

Although the NIR region was not useful as a vegetation indices, it along with the red region, was found useful for separating treatments in the PCA. The PCA was able to account

for more than 95% of the variation in the spectral data in three principal components. The first was defined by the upper half of the red region, that is, 700-740 nm the region known as the red edge. The second component was more variable among species and ranged from 688 to 743 nm placing it just inside the NIR region. The third component was different for each species and included the UV region for treatment separation in *E. piperita*, the blue region for *B. serrata*, the green region for *A. costata* and *L. longifolia* with an additional band in the red region, and the NIR region for *D. revoluta*. Seeing there was no obvious response in *L. longifolia* to disease it follows that the area on which the PCA could delineate was on the red and green regions that respond to water stress. At the end of the trial minimal evidence of disease remained detectable in *A. costata* as water stress had become the dominant influence on spectral reflectance. Thus treatment separation in the third component is based on increased green reflectance following chlorophyll reduction resulting from water stress. Although changes in *E. piperita* resulting from disease were visually evident, changes in the UV region detected by the PCA would not have been. Both *B. serrata* and *D. revoluta* gave no clear visual indication of disease throughout the trial. Thus the effect of disease in the NIR region that allowed for treatment separation were unseen, yet changes in the blue (visual) region of *B. serrata* were not visually evident. The ability of the human eye to detect changes in the blue region is limited and lost in wavelengths shorter than 400 nm (Vos, 1978). The maximum difference in reflectance of the treatment means of the blue bands contributing to the third principal component was 0.04, a difference that is likely to be too small for the human eye to detect. This highlights the potential of hyperspectral detection of *P. cinnamomi* infection to interpret disease in earlier stages of development or in what 'appear' to be an asymptomatic hosts which are in fact responding to disease in the UV or NIR regions.

The use of PCA in the interpretation of hyperspectral data for the purpose of disease detection, is not widely applied, yet it appears far superior in its ability to detect disease. Bauriegel *et al.* (2011) used PCA to identify spectral changes in Wheat infected with Fusarium head blight and classified hyperspectral images identifying healthy ears with 100% accuracy and diseased ears with 94% accuracy. In a similar manner, Yang *et al.* (2010) detected Cotton Root Rot using PCA and hyperspectral imagery with a minimum 96% accuracy, while Yusuf and He (2011) also successfully applied PCA to separate healthy and diseased Tobacco infected with *P. nicotianae*.

It has been shown here that infection caused by *P. cinnamomi* can be identified from hyperspectral imagery using appropriate vegetation indices, water features and PCA. Disease, although it affects the roots of plants, could be identified via foliar analysis.

However, the response was species-specific and changed over time as disease progressed. There was no one particular wavelength or indices that could identify *P. cinnamomi* infection unanimously throughout the trial. Additionally, it appears from the response of *L. longifolia* that quantification of disease is more difficult in resistant hosts. Whether this is due to infection having failed completely, or simply that the interaction between the pathogen and host is kept to a minimum is unclear as the pathogen was never re-isolated from any of the inoculated *L. longifolia* plants. *Phytophthora cinnamomi* can be difficult to isolate from plants especially when the tissue is plated (Huberli *et al.,* 2000; O'Brien *et al.,* 2009). This was evident here, yet some plants from which it was not reisolated were seen to behave in a similar way to those from which it was reisolated. This illustrates the potential of hyperspectral reflectance to identify disease in a non-destructive manner.

It was also clear by the end of the trial that water stress had a stronger influence on reflectance often drowning out the response associated with disease. Determining plant relative water content using an index such as NDVI may alleviate this problem or at least confirm whether or not plant water content is suitable to allow an assessment of infection.

This approach to *P. cinnamomi* disease detection does show potential for application in a natural or agricultural environment. It would however likely dependent upon *a priori* knowledge of the pathogens presence and distribution. Using this information the progression of disease could then be followed in susceptible hosts. For this to occur, the spectral response of more hosts to *P. cinnamomi* infection would be required, especially in those species that form a dominant part of an ecosystem. Even if hyperspectral analysis did not reveal *P. cinnamomi* infection as such, it would likely assist in the identification of stressed or disease vegetation warranting further investigation from a field-based approach. Within the context of the GBMWHA, this could mean the analysis of hyperspectral imagery of areas with high conservation value or where the pathogen is already known to exist, allowing monitoring programs of disease spread and plant health to be established.

The response of the individual plants tested here was quite variable and further work is required to more accurately describe the effect of *P. cinnamomi* infection on spectral reflectance. Additionally, although significant wavelengths have been identified via hyperspectral analysis, the application of disease detection in the field may only require multispectral analysis assuming similar regions in the UV, visible and NIR regions were available. Hyperspectral analysis however likely offers a new and effective way to monitor and manage Phytophthora dieback in the natural ecosystems of Australia.

Chapter 6 General Discussion

The research presented in this thesis has attempted to quantify the risk of Phytophthora dieback in the GBMWHA. I found that *P. cinnamomi* is suited to many of the ecosystems included in the GBMWHA, that Phytophthora dieback is significantly affecting vegetation health there, and that there are extensive parts of the landscape where the risk of Phytophthora dieback is high. Additionally, I developed a new method for detecting Phytophthora dieback that could potentially be utilised in remote and inaccessible areas.

Dieback and vegetation decline caused by *P. cinnamomi* represent a direct threat to the criteria upon which World Heritage status depends. At the initiation of this project, there was little information available on the distribution of *P. cinnamomi* in the GBMWHA, making it difficult for land managers to assess the threat and develop appropriate management strategies.

An expert-driven (mechanical) modelling approach was used to quantify the likelihood that *P. cinnamomi* would establish in different parts of the GBMWHA (Chapter 2), a task that was limited by the lack of distribution information currently available. The model was kept as simple as possible to ensure that risk assessments could be confidently extrapolated to areas where no physical sampling information was available. The findings of this model indicate that much of the GBMWHA is suitable for pathogen establishment and thus the first hypothesis was accepted. Areas that where modelled with greater risk were centred on the Blue Mountains NP, the area that is central to most tourist and community activity within the GBMWHA.

The findings of the model were then used to guide a field survey (Chapter 3) that allowed for the retrospective evaluation of the distribution prediction. The initial assessment of model performance indicated the model had greatly over-predicted the distribution of *P. cinnamomi*, as the actual rate of isolation was lower than predicted. This, however, may have several explanations outside the parameters of the model. Firstly, the largely clonal population of *P. cinnamomi* in Australia suggests it is an introduced species (Dobrowolski *et al*., 2003), with generally only one of the two known mating types found in Australia (Old *et al*., 1984, 1988). Extensive disease across Australia also suggests that native vegetation has not co-evolved with *P. cinnamomi* (Howard, 2008), supporting the hypothesis that *P. cinnamomi* has been introduced. As a result, *P. cinnamomi* is unlikely to have reached equilibrium in the landscape (Elith and Leathwick, 2009), and is likely to move toward a dynamic equilibrium state with the population continually responding to changes in the environment. As a result, *P. cinnamomi* will not have established into all the areas it

can potentially exist within the GBMWHA. Secondly, *P. cinnamomi* typically has a patchy distribution at fine scales in the soil (Ashton, 1970; Weste and Taylor, 1971; Weste and Kennedy, 1997; Brown *et al.,* 2002; Podger and; Pryce *et al.,* 2002; McDougall *et al.,* 2003) and as a result, it can easily be missed during sampling. This was clearly seen in some of the high risk 'hot spots' in which multiple samples within a confined area gave a mixture of both positive and negative results. According to Pryce *et al.* (2002) and Davidson and Tay (2005) several kilograms of soil requiring the removal of over one square metre of topsoil may need to be tested before a site can be rendered pathogen free with 95% confidence. This would cause significant logistical problems, and cause unacceptable disturbance to sampling sites.

Finally, the model did not take into account physical boundaries and restrictions that prevent *P. cinnamomi* from establishing in sites that are environmentally suitable. This was illustrated in the Natti NP, most of which is a restricted reserve accessed only by management staff, thereby limiting the risk of anthropogenic introduction of the pathogen. On the basis of environmental parameters used in the model, Natti NP was identified at risk of infection, however only one positive sample was recorded in my sampling. *Phytophthora cinnamomi* is known to be present in the Natti NP from previous studies (Suddaby, 2008a), which again highlights the limitations of sampling strategies and soil baiting to ensure accurate pathogen detection.

Discussion of appropriate methods for the detection of *P. cinnamomi* in natural environments, and the suitability of sample strategies for studying environmental issues is lacking (Zhang and Zhang, 2012). There are four general approaches to sampling strategies which include simple random sampling, systematic sampling, stratified sampling, or Latin Hypercube Sampling (Minasny and McBratney, 2006; Falk *et al.,* 2011; Zhang and Zhang, 2012). The third and fourth methods use ancillary information to construct a sampling model ensuring effective coverage of environmental variables (Minasny and McBratney, 2006; Falk *et al.,* 2011). The method selected will depend on the allocated time, the budget and the specific data request (Domburg *et al.,* 1997), and should have the goal of maximising sample representativeness while minimising the number of samples required (Zhang and Zhang, 2012). The stratified random scheme used here, a model-based approach, is likely to be more efficient than a simple random sample or a systematic sample (Zhang and Zhang, 2012), the latter of which would have been unsuitable for this research given the size and nature of the GBMWHA. Using a model-based approach (i.e., a strategy based on ancillary information) is better if prediction is to be completed with the sampling results (Minasny and McBratney, 2006), which was the case here. The samples acquired

through the stratification process, covered the full range of environmental variables subsequently utilised in the construction of the model in Chapter 4.

The collection of samples during the Root Rot Trot, however, was slightly different in that, diseased vegetation was specifically targeted. Such a strategy could still be considered stratified (i.e., collecting from either diseased or healthy vegetation), and defined more accurately as judgemental sampling (Zhang and Zhang, 2012). The result was an increase in the isolation of *P. cinnamomi* from 15% for the overall survey average, to 40% for the samples collected on this trip. This does not indicate that *P. cinnamomi* is more abundant in this part of the GBMWHA, simply, that targeting diseased vegetation, in this case, increased the rate of isolation. Interestingly, Pryce *et al.* (2002), noted that rates of isolation were equal in diseased and asymptomatic sites in Queensland when samples were collected randomly. The frequency of isolations of *P. cinnamomi* at disease-free sites in Queensland may indicate that the environment at these particular locations is favourable to the pathogen but not to the expression of disease. Further to this, disturbance to plant root systems caused by wild pigs in the area likely created plant stress resulting in symptom expression. Such disturbance was not observed during the Root Rot Trot, supporting the notion that the area was in the early stage of infection. Pryce (2002) also noted that at 1,300 m^2, the distribution of *P. cinnamomi* was uniform, suggesting, a sampling unit smaller than this would be required to detect the spatial pattern of *P. cinnamomis* distribution. For the GBMWHA, this sampling rate represents nearly eight million samples, a task that would never be completed, and as such, modelling the distribution of *P. cinnamomi* is the only realistic option.

Phytophthora cinnamomi was found across a range of geographic and climate environments in the GBMWHA, in close proximity to urban areas as well as in the most remote parts of the Park (Chapter 3). The pathogen was recovered more frequently around urban areas supporting the conclusions from previous research that *P. cinnamomi* is typically spread via anthropogenic means (Weste and Taylor, 1971; Pratt *et al.,* 1972; Hardham, 2005; Howard, 2008). The extensive isolation of *P. cinnamomi* in wilderness areas, however, was not expected, as many of these areas are remote and public access has been restricted for nearly 40 years. Although the diversity and abundance of species was not addressed directly, dieback observed in highly susceptible species suggests that *P. cinnamomi* has been introduced into this area more recently as highly susceptible species tend to be affected and lost from an ecosystem first and species diversity is decreased by the infestation (Marks *et al.,* 1972; Newell, 1998; Weste, 2003; McDougall *et al.,* 2005).

Disease was not always evident from where the pathogen was isolated; in fact it was mostly associated with healthy vegetation. This again suggests that *P. cinnamomi* has been established in some parts of the GBMWHA for many decades as the highly susceptible species have been lost leaving species that are resistant to disease or remain asymptomatic until additional stress induces symptoms (Marks *et al.,* 1972; Newell, 1998; Weste, 2003). As recently demonstrated in Western Australia, it is also possible the plants remain asymptomatic during infection in which *P. cinnamomi* behaves as a biotroph (Crone *et al.,* 2013a; Crone *et al.,* 2013b), suggesting that such plants can harbour the pathogen for the duration of their life. As vegetation appears to be at different stages of infection throughout the GBMHWA, it is quite probable that *P. cinnamomi* has been introduced multiple times (Weste, 2003; Howard, 2008). The extenisve distribution of the other *Phytopthora* species in the GBMWHA also supports the possiblitly of multipul introductions of *Phytopthora* throughout the Park.

A lack of disease expression can result from a number of other causes including greater resistance amongst hosts which is recognised in NSW species (McCredie *et al.,* 1985; Suddaby *et al.,* 2008). Also, as discussed above, the pathogen may have a patchy distribution in the soil, and as such, potential hosts may escape infection. The reasons for inconsistent levels of inoculum in the soil are not well understood, but likely relate to small scale variations in host tissue, soil water and texture, as well as the uneven distribution of organic matter and soil microbes. Antagonism and competition between soil microbes and *P. cinnamomi* is known to influence the distribution of the pathogen (Broadbent and Baker, 1974; Weste and Vithanage, 1978) as well as the expression of disease (Broadbent and Baker, 1974; Marks and Smith, 1981).

As reported elsewhere in NSW, many host plants, although infected, showed no indication of disease (McDougall and Summerell, 2003). Although *P. cinnamomi* is typically thought of as a pathogen, it has recently been demonstrated behaving as a biotroph in a number of herbaceous species, and as such, plants remain alive (Crone *et al.,* 2013a; Crone *et al.,* 2013b). Few of the studied plants showed symptoms of infection, in which *P. cinnamomi* was able to produce haustoria and a number of survival and reproductive structures that were able to germinate within the plant. Such behaviour of *P. cinnamomi* in infected hosts would make detection and management of the pathogen difficult.

Aside from greater host resistance to the pathogen, lack of disease expression may result from climatic factors. This is perhaps the most likely explanation for the lack of evidence of Phytophthora dieback occurring in the GBMWHA. Although much of the area is suitable to pathogen establishment, prolonged hot and dry summer conditions that usually

induce disease in winter-dominant Mediterranean climates (Weste and Ruppin, 1977; Weste and Marks, 1987; Weste, 1994) are less likely to occur in the wetter summers of the warm temperate climate of the GBMWHA. The highest period of rainfall occurs in summer months meaning water stress is less likely to occur, and the dryer months of winter are likely to be too cold for the pathogen to remain active (Weste and Vithanage, 1978; Nesbitt *et al.,* 1979). Dieback, when it was observed, appeared frequently along ridge tops where vegetation is more prone to water stress as soils are likely to dry out quickly both because they are shallow and because water will drain from them to lower areas. Such areas included the heathland communities of the upper mountains.

Within the heathlands, there are many species from families that tend to be more susceptible to *P. cinnamomi* infection in other parts of Australia including Proteaceae, Epacridaceae, Xanthorrhoeaceae, and Fabaceae (Newhook and Podger, 1972). These heathland communities include, or are associated with, at least three ecological communities with threatened or endangered conservation status, highlighting that these ecosystems are already at risk of being permanently damaged or lost. Disease was prevalent in the heathland communities sampled, many of which have been a favourite tourist destination for decades. Again active and extensive disease may result from the pathogen being introduced recently, or alternatively, these herbaceous plants are able to set seed before succumbing to infection. In the latter case, the cycle of dieback may continue indefinitely as each new generation of plants continues to sustain the pathogen population.

Although the vegetation in most areas from which the pathogen was isolated would have been considered healthy, site disturbance was observed in several locations exhibiting disease symptoms. Disturbance has been linked with disease expression elsewhere (Podger and Brown, 1989; Gadek and Worboys, 2003; Scarlett *et al.,* 2012) and results from contamination, logging, fire or other pests and diseases.

Although disturbance was associated with disease and the pathogen was isolated extensively from urban areas, anthropogenic layers did not make significant contributions to the construction of the Phytophthora dieback model (Chapter 4). This possibly indicates that *P. cinnamomi* has reached somewhat of an equilibrium state within urban landscapes. Given that European exploration of the GBMWHA began 200 years ago, and dieback was first observed near Sydney in the 1940s (Fraser, L., 1956, cited in Newhook and Podger, 1972), there has been ample opportunity for the pathogen to spread naturally, or be introduced extensively across the individual reserves of the GBMWHA. *Phytophthora cinnamomi* is widely distributed in the Royal National Park, south of Sydney (Walsh *et al.,*

2006) and as such, its distribution in the soil is now likely to be determined by small-scale changes in climatic factors and soil characteristics. Because *P. cinnamomi* has been found widely distributed within the GBMWHA, anthropogenic factors are still relevant, however, climatic and topographic factors likely have more effect on its survival and continued spread. Anthropogenic introduction of new species or new isolates of *Phytophthora* pose a much more significant risk to the sustained ecosystem function of the GBMWHA.

The distribution of *P. cinnamomi* predicted by the statistical approach (Chapter 4) was similar to that produced by the mechanistic approach (Chapter 2), in that the high risk areas were centred on the Blue Mountains NP. As discussed, the mechanistic model appears to have over-predicted the distribution of *P. cinnamomi* and respectively, it is possible that the statistical model has under-predicted the distribution. This is possible because the areas in which *P. cinnamomi* has been successfully isolated does not necessarily reflect all areas suitable to the pathogen (Elith and Leathwick, 2009) and because it may not have been successfully isolated at all (Pryce *et al.,* 2002; Davison and Tay, 2005). Additionally, introduced species behave differently in new environments, adapting to the new conditions in which they would previously not have been found (Jeschke and Strayer, 2008). As a result, there is a clear advantage in having both models as the actual distribution of *P. cinnamomi* is likely to occur somewhere between the two.

The predicted distribution of Phytophthora dieback, including the addition of the host distributions (Chapter 4), was obviously even more restricted then the predicted distribution of *P. cinnamomi*. Again, the central region of the GBMWHA was highlighted as the area most at risk of disease not just because of its environmental suitability to the pathogen, but also due to a greater presence of host species.

The predicted disease distribution, however, did not necessarily reflect cases of dieback observed in the field. In the natural environment, there are other intricacies that influence the occurrence of disease that has not been accounted for in the model. There are two possible reasons as to why disease is not evident; *P. cinnamomi* is widespread, but dieback is limited due to the environment or, simply, *P. cinnamomi* is not widespread. As discussed, the two key factors that are likely to suppress the expression of disease when the pathogen is present include, suppression by other soil microbes (Broadbent and Baker, 1974), and climates in which prolonged drought stress does not occur (Weste and Vithanage, 1978; Weste and Marks, 1987). This does not mean that Phytophthora dieback will not eventuate in the GBMWHA. If *P. cinnamomi* is able to spread into uninfested locations, disease may eventuate, or if the environment changes, disease expression may alter also.

By adjusting the rainfall and temperature inputs, it was shown that the incidence of disease potential changed. These changes made to the prediction of *P. cinnamomi* distribution were not intended to reflect the potential impacts of climate change on Phytophthora dieback. There are many other complexities that would need to be considered to do this, such as the changing distribution of host and non-uniform changes in the climate predicted for NSW (Department of Environment Climate Change and Water NSW., 2010), creating rainfall and temperature gradients across the GBMWHA. However, the adjustments made to the prediction provide insight into how the distribution of *P. cinnamomi* may be effected in a changing environment. As such, increasing temperature and decreasing rainfall reduced the predicted distribution of *P. cinnamomi* suggesting these changes would be detrimental to the pathogen. However, under these conditions, an increase in outbreaks of Phytophthora dieback may occur as drought stress increases.

As demonstrated in Chapter 4, sampling results can improve the accuracy of species distribution models (Elith and Leathwick, 2009), however the acquisition of such data is challenging, expensive, and as discussed above, does not always reflect the actual distribution of the target organism due to sampling and analytical error (Zhang and Zhang, 2012). Additionally, physical sampling results quickly become outdated (O'Gara *et al.,* 2005) and as a consequence, many more hours and funds would be required to keep them up-to-date. This highlights some of the main issues of assessing the distribution of *P. cinnamomi* and Phytophthora dieback from a field-based, soil-based perspective. In an attempt to find a more economical, safer and real-time alternative, a method of remote detection of disease was developed. The application of hyperspectral detection of disease has been demonstrated in a number of plant-pathogen interactions (Bock *et al.,* 2010), however, it was unable to reliably identify *P. cinnamomi* infection here. Hyperspectral analysis detects water stress that could result from several causes, including the secondary symptoms of dieback. There was no unique spectral shift that could be associated with *P. cinnamomi* infection, however, changes in regions that have been associated with stress and disease were evident and allowed for the separation of treatments in some of the species tested. Additionally, with the use of principal component analysis, minute spectral changes and changes outside of the visible range where evident, suggesting disease detection is possible in presymptomatic or asymptotic hosts. This would be particularly useful in areas that lack obvious symptoms of disease such as the GBMWHA.

The co-occurrence of water stress in infected plants made it difficult to specifically identify disease, suggesting the *P. cinnamomi* infection cannot be distinguished from water stress. This, however, may only become an issue in areas where drought stress leads to the

expression of wilt. As dieback was not often evident in the GBMWHA, hyperspectral identification of infection may still be possible. Given that the distribution of *P. cinnamomi* is now more clearly understood, remote sensing offers a possible alternative to allow for the monitoring of infection and disease, however these relationships would need more clarification.

The clearer understanding of *P. cinnamomi* distribution in the GBMWHA resulting from the work presented in this thesis enables land managers to develop proactive strategies to limit the damage caused by dieback. The areas within the park that are most likely to harbour the pathogen have been identified and these can be targeted with efforts to reduce pathogen impacts. Although *P. cinnamomi* appears relatively widespread, the maintenance of hygiene is still important as new introductions of *P. cinnamomi* may bring in more virulent isolates, or other species that also cause plant disease. Strategies for areas with high pathogen risk may include the placement of foot baths at track heads, or track closure during periods of high rainfall. Full restrictions of access to tracks is unrealistic in the GBMWHA as many tourists enter the area simply for the walking and hiking opportunities. Relocation of tracks to lower risk areas would reduce the risk of pathogen spread, however, the new route may not pass the attraction that the track has been positioned towards making them less favourable to tourists and the community.

Education of the community and management staff should continue to be a priority. As the major park users, they need to be aware of the risk of *P. cinnamomi* and its impacts on plant abundance and diversity, dependant fauna, water and nutrient cycling, erosion and loss in aesthetic value. Additionally, they need to understand that it is not just to the GBMWHA that may be impacted, but anywhere susceptible species occur. Awareness of their own personal responsibility in reducing the spread of disease and preventing site disturbance to ensure the ongoing health of natural ecosystems susceptible to Phytophthora dieback should be fostered. Systems for community education and awareness have been established in other area such as the Penguin Walking Track in the Sydney Harbour National Park in NSW (David Guest, Pers. comm.), and other states of Australia (O'Gara *et al.,* 2005). These systems would be easily transferable to the GBMWHA.

Where dieback has been observed occurring, both hygiene and education should be a priority (O'Gara *et al.,* 2005). This includes the heathland communities of the upper mountains in which Phytophthora dieback appeared to affect a number of species. Research efforts also need to be focused here to elucidate the role of *P. cinnamomi* in the dieback of heathland species and determine if *ex-situ* conservation or possibly chemical treatment are needed in these areas, as in the Wollemi NP (David Crust, Pers. comm.;

O'Gara *et al.,* 2005). *Phytophthora cinnamomi* may also be placing additional stress on the endangered ecological communities of the upper mountains and this ought to be investigated also.

The reason for the lack of visible dieback occurring in areas where *P. cinnamomi* has been isolated should be explored more. Soil suppression, genetic resistance in plant hosts, and biotrophic behaviour of *P. cinnamomi* are all areas of research that may lead to new methods of disease control for both the GBMWHA and other ecosystems affected by dieback. Additionally, the effect of seasonality, i.e., the interplay between temperature, rainfall and symptom expression should be investigated, increasing knowledge of the behaviour of *P. cinnamomi* in NSW ecosystems. Approaches could continue using modelling, or field and glasshouse-based techniques.

Remote sensing of *P. cinnamomi* infection should continue to be pursued. If we continue to use the methods of disease identification and treatment we are using now, we will continue to get the same results (Cahill *et al.,* 2008). Plant pathology and landscape epidemiology needs to more extensively utilise the new opportunities available with remote sensing and modelling platforms (Holdenrieder *et al.,* 2004; Plantegenest *et al.,* 2007). Climate change and invasive species may alter ecosystems faster than traditional methods of analysis can keep up.

The consensus of the two *P. cinnamomi* models (Chapters 2 and 4) and the field results (Chapter 3) indicate that the GBMWHA is environmental suitable to *P. cinnamomi* and host known to be susceptible to disease are distributed throughout. The elements of the disease triangle used to quantify disease, indicate the GBMWHA is at risk. Yet, fortunately, Phytophthora dieback doesn't appear to be devastating ecosystems as it does in the Mediterranean climates of Australia. The reasons appear complex and need further investigation. The quantification of Phytophthora dieback via hyperspectral remote sensing, although not proven here conclusively, does appear to offer a new means of disease detection improving the efficacy of proactive disease management.

Chapter 7: References

Agrios, G., 2005. *Plant Patholgy,* California, Elsevier Academic Press.

Ahmadi, M., Zade, P. S., Pouryani, S. B. M., Gilak, S. & Sgem, 2010. Site selection of waste storage locations with FUZZY logic and analytic hierarchy process in a GIS framework, case study: Si-sakht town in Dena City, Kohgilouye o Boyer Ahmad Province, Iran. *10th International Multidisciplinary Scientific Geoconference: Sgem 2010, Vol Ii.* Sofia: Int Scientific Conference Sgem.

Alberico, I., Lirer, L., Petrosino, P. & Scandone, R., 2008. Volcanic hazard and risk assessment from pyroclastic flows at Ischia island (southern Italy). *Journal of Volcanology and Geothermal Research,* 171, 118-136.

Ali, Z., Smith, I. & Guest, D. I., 1999. Effect of potassium phosphonate on root rot of *Pinus radiata* caused by *Phytophthora cinnamomi*. *Australasian Plant Pathology,* 28**,** 120-125.

Allen, R. N. & Newhook, F. J., 1974. Suppression by Ethanol of Spontaneous turning activity in Zoospores of *Phytophthora-cinnamomi*. *Transactions of the British Mycological Society,* 63**,** 383-385.

Anon., 2012. Sampling and Mapping of *Phytophthora cinnamaomi* in the Gondwana rainforests of eastern Australia. The University of Sydney.

Arentz, F., 1974. *Studies on Phytophthora cinnamomi.* PhD Thesis, Australian National Universtiy.

Armstrong, J. A., 2002. Zieria (Rutaceae): a systematic and evolutionary study. *Australian Systematic Botany,* 15**,** 277-463.

Aryantha, I. P., Cross, R. & Guest, D. I., 2000. Suppression of *Phytophthora cinnamomi* in potting mixes amended with uncomposted and composted animal manures. *Phytopathology,* 90**,** 775-782.

Atkinson, D., Chladil, M., Janssen, V. & Lucieer, A., 2010. Implementation of quantitative bushfire risk analysis in a GIS environment. *International Journal of Wildland Fire,* 19, 649-658.

Australian Network for Plant Conservation., 2012. *21 of Australia's most threatened plants* [Online]. Available: http://www.anpc.asn.au/21threatenedplants.html [Accessed 19/7/13].

Baker, M. & Corringham, R., 2004. *Native Plants of the Blue Mountains,* Winmalee, Bower Bird Books.

Barker, P. C. J. & Wardlaw, T. J., 1995. Susceptibility of selected Tasmanian rare plants to *Phytophthora-cinnamomi*. *Australian Journal of Botany,* 43**,** 379-386.

Barrett, E. C. & Curtis, L. F., 1992. *Introduction to environmental remote sensing,* London, Chapman & Hall.

Barrett, S., Shearer, S. L., Crane, C. E. & cochrane, A., 2008. An extinction-risk assessment tool for flora threatened by *Phytophthora cinnamomi*. *Australian Journal of Botany,* 56**,** 477-486.

Barry, S. & Elith, J., 2006. Error and uncertainty in habitat models. *Journal of Applied Ecology,* 43**,** 413-423.

Bauriegel, E., Giebel, A., Geyer, M., Schmidt, U. & Herppich, W. B., 2011. Early detection of Fusarium infection in wheat using hyper-spectral imaging. *Computers and Electronics in Agriculture,* 75**,** 304-312.

Bayley, W., 1980. *Blue Mountains Railways,* Bulli, NSW, Austrail Publications.

Bell Miner Assoicated Dieback Working Group, 2004. Bell Minor Associated Dieback Strategy.

Benson, D., 1979. Native Vegetation of Corridor-Wolgan River to Putty Road. Royal Botanic Gardens Sydney.

Benson, D., 1992. The Natural Vegetation of the Penrith 1:100 000 map sheet. *Cunninghamia,* 2**,** 541-596.

Benson, D., Howell, J. & Mcdougall, L., 1996. *Native Plant Species in the Hawkesbury-Nepean Catchment,* Sydney, Royal Botanic Gardens.

Benson, D. & Keith, D. 1990. The Natural Vegetation of the Wallerawang 1:100 000 map sheet. *Cunninghamia,* 2**,** 305-335.

Bergot, M., cloppet, E., Perarnaud, V., Deque, M., Marcais, B. & Desprez-Loustau, M. L., 2004. Simulation of potential range expansion of oak disease caused by *Phytophthora cinnamomi* under climate change. *Global Change Biology,* 10**,** 1539-1552.

Blackburn, G. A., 1999. Relationships between spectral reflectance and pigment concentrations in stacks of deciduous broadleaves. *Remote Sensing of Environment,* 70**,** 224-237.

Blackburn, G. A., 2007. Hyperspectral remote sensing of plant pigments. *Journal of Experimental Botany,* 58**,** 855-867.

Blaker, N. S. & Macdonald, J. D., 1983. Influence of container medium ph on sporangium formation, zoospore release, and infection of rhododendron by P*hytophthora-cinnamomi*. *Plant Disease,* 67**,** 259-263.

Blowes, W. M., Heather, W. A., Malajczuk, N. & Shea, S. R., 1982. The Distribution of *Phytophthora cinnamomi* Rands at Two Sites in Southern Western Australia and at Durras in South-eastern New South Wales. *Australian Journal of Botany,* 30**,** 139-145.

Bluett, V., Weste, G. & Cahill, D., 2003. Distribution of disease caused by *Phytophthora cinnamomi* in Wilsons Promontory National Park and potential for further impact. *Australasian Plant Pathology,* 32**,** 479-491.

Bock, C. H., Poole, G. H., Parker, P. E. & Gottwald, T. R., 2010. Plant Disease Severity Estimated Visually, by Digital Photography and Image Analysis, and by Hyperspectral Imaging. *Critical Reviews in Plant Sciences,* 29**,** 59-107.

Boegh, E., Soegaard, H. & Thomsen, A., 2002. Evaluating evapotranspiration rates and surface conditions using Landsat TM to estimate atmospheric resistance and surface resistance. *Remote Sensing of Environment,* 79**,** 329-343.

Booth, T. H. & Jovanovic, T., 2012. Assessing vulnerable areas for *Puccinia psidii* (eucalyptus rust) in Australia. *Australasian Plant Pathology,* 41**,** 425-429.

Bowers, J. H., Papavizas, G. C. & Johnston, S. A., 1990. Effect of soil-temperature and soil-water matric potential on the survival of *phytophthora-capsici* in natural soil. *Plant Disease,* 74**,** 771-777.

Brasier, C. M., 1992. Evolutionary biology of *Phytophthora* Part I: genetic system, sexuality and the question of variation. *Annual Review of Phytopathology,* 30**,** 153-171.

Brasier, C. M., 1996. *Phytophthora cinnamomi* and oak decline in southern Europe. Environmental constraints including climate change. *Annual Science Forum,* 53**,** 347 - 358.

Brasier, C. M. & Scott, J., 1994. European oak declines and global warming: a theoretical assessment with special reference to the activity of *Phytophthora cinnamomi*. *EPPO Bulletin,* 24**,** 221-232.

Broadbent, P. & Baker, K. F., 1974. Behavior of *Phytophthora-cinnamomi* in soils suppressive and conductive to root-rot. *Australian Journal of Agricultural Research,* 25**,** 121-137.

Brown, B. N., 1976. *Phytophthora cinnamomi* assoicated with patch death in tropical rain forests in Queensland. *Plant Pathology Society Newsletter,* 5**,** 1-4.

Brown, M. J., Balmer, J. & Podger, F. D., 2002. Vegetation change over 20 years at Bathurst Harbour, Tasmania. *Australian Journal of Botany,* 50**,** 499-510.

Bullock, S., Summerell, B. A. & Gunn, L., 2000. Pathogens of the Wollemi Pine (*Wollemia nobilis*). *Australasian Plant Pathology,* 29**,** 211-214.

Butcher, T. B., Stukely, M. J. C. & Chester, G. W., 1984. Genetic Variation in Resistance of *Pinus radiata* to *Phytophthora cinnamomi*. *Forest Ecology and Management,* 8**,** 197-220.

Butler, W. L. & Hopkins, D. W., 1970. Higher derivative analysis of complex absorption spectra. *Photochemistry and Photobiology,* 12**,** 439-450.

Buckley, L. B., Urban, M. C., Angilletta, M. J., Crozier, L. G., Rissler, L. J. & Sears, M. W., 2010. Can mechanism inform species' distribution models? *Ecology Letters,* 13, 1041-1054.

Burgess, T. I., Webster, J. L., Ciampini, J. A., White, D., Hardy, G. E. S. & Stukely, M. J. C., 2009. Re-evaluation of *Phytophthora* Species Isolated During 30 Years of Vegetation Health Surveys in Western Australia Using Molecular Techniques. *Plant Disease,* 93**,** 215-223.

Byrt, P. & Grant, B. R., 1979. Some conditions governing zoospore production in axenic cultures of *Phytophthora-cinnamomi* rands. *Australian Journal of Botany,* 27**,** 103-115.

Cahill, D., 1993. Review of *Phytopthora* diseases in Australia : a report. RIRDC, Barton, ACT.

Cahill, D. M. & Hardham, A. R., 1994. A dipstick immunoassay for the specific detection of *Phytophthora-cinnamomi* in soils. *Phytopathology,* 84**,** 1284-1292.

Cahill, D., Morris, P. F. & Ward, E. W. B., 1993. Influence of metalaxyl on abscisic acid levels in soybean hypocotyls infected with *Phytophthora sojae. Physiological and Molecular Plant Pathology,* 42**,** 109-121.

Cahill, D. M., Rookes, J. E., Wilson, B. A., Gibson, L. & Mcdougall, K. L., 2008. *Phytophthora cinnamomi* and Australia's biodiversity: impacts, predictions and progress towards control. *Australian Journal of Botany,* 56**,** 279-310.

Cahill, D. & Ward, E. W. B., 1989. Rapid and localised changes in abscisic acid concerntrations in soybean in interactions with *Phytophthora megasperma* f. sp. glycinea or after treatment with elicitors. *Physiological and Molecular Plant Pathology,* 35**,** 4583-4593.

Cahill, D. & Weste, G., 1983. Changes in respiration of seedling roots inoculated with *Phytophthora-cinnamomi. Phytopathologische Zeitschrift-Journal of Phytopathology,* 106**,** 51-62.

Calm, 2001. *Phytophthora cinnamomi and disease caused by it. Volume 2- Interpreter's Guidelines for Detection, Diagnosis and Mapping.* Department of Conservation and Land Management, Government of Western Australia.

Cameron Mcnamara Consultants, 1988. Jenolan Caves Reserve Draft Plan of Managment. Vol 1. Tourisum Commission of New South Wales and Crown Lands Office.

Cao, X., Luo, Y., Zhou, Y. & Cheng, D., 2013. Detection of powdery mildew in two winter wheat cultivars using canopy hyperspectral reflectance. *Crop Protection,* 45**,** 124-131.

Carnegie, A. J., 2007. Forest health condition in New South Wales, Australia, 1996-2005. II. Fungal damage recorded in eucalypt plantations during forest health surveys and their management. *Australasian Plant Pathology,* 36**,** 225-239.

Chadfield, B. V. & Pautasso, M., 2012. *Phytophthora ramorum* in England and Wales; which environmental variables predict county disease incidence? *Forest Pathology,* 42**,** 150-159.

Chakraborty, S., Murray, G. M., Magarey, P. A., Yonow, T., O'brien, R. G., Croft, B. J., Barbetti, M. J., Sivasithamparam, K., Old, K. M., Dudzinski, M. J., Sutherst, R. W., Penrose, L. J., Archer, C. & Emmett, R. W., 1998. Potential impact of climate change on plant diseases of economic significance to Australia. *Australasian Plant Pathology,* 27**,** 15-35.

Chakraborty, S., Tiedemann, A. V. & Teng, P. S., 2000. Climate change: potential impact on plant diseases. *Environmental Pollution,* 108**,** 317-326.

Chau, K. W. & Yang, W. W., 1992. A knowledge-based expert system for unsteady open channel flow. *Engineering Applications of Artificial Intelligence,* 5, 425-430.

Chee, K.-H. & Newhook, F. J., 1965. Improved methods for use in studies on *Phytophthora cinnamomi* Rands and other *Phytophthora* species. *New Zealand Journal of Agricultural Research,* 8**,** 88-95.

Cho, J. J., 1981. Phytophthora root-rot of Banksia - host range and chemical control. *Plant Disease,* 65**,** 830-833.

Clark, R. N. & Roush, T. L., 1984. Reflectance spectroscopy - quantitative-analysis techniques for remote-sensing applications. *Journal of Geophysical Research,* 89**,** 6329-6340.

Commonwealth Of Australia, 2005. Environment Protection and Biodiversity Conservation Act 1999. Canberra: Commonwealth Government Printer.

Contreras-Medina, L. M., Torres-Pacheco, I., Guevara-Gonzalez, R. G., Romero-Troncoso, R. J., Terol-Villalobos, I. R. & Osornio-Rios, R. A., 2009. Mathematical modeling tendencies in plant pathology. *African Journal of Biotechnology,* 8**,** 7399-7408.

Coops, N., Stanford, M., Old, K., Dudzinski, M., Culvenor, D. & Stone, C., 2003. Assessment of Dothistroma needle blight of Pinus radiata using airborne hyperspectral imagery. *Phytopathology,* 93**,** 1524-1532.

Coops, N. C., Stone, C., Culvenor, D. S. & Chisholm, L., 2004. Assessment of crown condition in Eucalypt vegetation by remotely sensed optical indices. *Journal of Environmental Quality,* 33**,** 956-964.

Coops, N., Warning, R. H. & Landsberg, J. J., 1998. Assessing forest productivity in Australia and New Zealand using a physiologically-based model driven with averaged monthly weather data and satellite-derived estimates of canopy photosythetic capacity. *Forest Ecology and Management,* 104**,** 113-127.

Corcobado, T., Cubera, E., Moreno, G. & Solla, A., 2013. *Quercus ilex* forests are influenced by annual variations in water table, soil watr deficit and fine root loss casued by *Phytophthora cinnamomi. Agricultural and Forest Meterorology,* 169**,** 92-99.

Crone, M., Mccomb, J., O'brien, P. A. & Hardy, G. E. S., 2013a. Assessment of Australian native annual/herbaceous perennial plant species as asymptomatic or symptomatic hosts of *Phytophthora cinnamomi* under controlled conditions. *Forest Pathology,* 43**,** 245-251.

Crone, M., Mccomb, J. A., O'brien, P. A. & Hardy, G. E. S., 2013b. Survival of *Phytophthora cinnamomi* as oospores, stromata, and thick-walled chlamydospores in roots of symptomatic and asymptomatic annual and herbaceous perennial plant species. *Fungal Biology,* 117**,** 112-123.

Cronk, Q. C. B. & Fuller, J. L., 1995. *Plant invaders,* London, Chapman and Hall.

Crowl, T. A., Crist, T. O., Parmenter, R. R., Belovsky, G. & Lugo, A. E., 2008. The spread of invasive species and infectious disease as drivers of ecosystem change. *Frontiers in Ecology and the Environment,* 6**,** 238-246.

Curren, P. J., Dungan, J. L., Macler, B. A. & Plummer, S. E., 1991. The effect of a red-edge pigment on the relationship between red-edge and chlorophyll concentration. *Remote Sensing of Environment,* 35**,** 69-75.

Cushman, J. H. & Meentemeyer, R. K., 2008. Multi-scale patterns of human activity and the incidence of an exotic forest pathogen. *Journal of Ecology,* 96**,** 766-776.

Daniel, R., Taylor, J. & Guest, D., 2006. Distribution and occurrence of *Phytophthora cinnamomi* at Middle Head and North Head, Sydney Harbour. *Australasian Plant Pathology,* 35**,** 569-571.

Darvishzadeh, R., Atzberger, C., Skidmore, A. K. & Abkar, A. A., 2009 Leaf Area Index derivation from hyperspectral vegetation indicies and the red edge position. *International Journal of Remote Sensing*. 30, 6199-6218.

Davison, E. M., Stukely, M. J. C., Crane, C. E. & Tay, F. C. S., 1994. Invasion of phloem and xylem of woody stems and roots of *Eucalyptus-marginata* and *Pinus-radiata* by *Phytophthora-cinnamomi*. *Phytopathology,* 84, 335-340.

Davison, E. M. & Tay, F. C. S., 2005. How many soil samples are needed to show that *Phytophthora* is absent from sites in the south-west of Western Australia? *Australasian Plant Pathology,* 34**,** 293-297.

Dawson, P. & Weste, G., 1984. Impact of root infection by *Phytophthora-cinnamomi* on the water relations of 2 *Eucalyptus* species that differ in susceptibility. *Phytopathology,* 74**,** 486-490.

Delalieux, S., Auwerkerken, A., Verstraeten, W., Somers, B., Valcke, R., Lhermitte, S., Keulemans, J. & Coppin, P., 2009. Hyperspectral Reflectance and Fluorescence Imaging to Detect Scab Induced Stress in Apple Leaves. *Remote Sensing,* 1**,** 858-874.

Department of Environment and Climate Change, 2009. Greater Blue Mountains World Heritage Area Strategic Plan. Sydney.

Department of Environment and Climate Change, 2008. The Native Vegetation of Yengo and Parr Reserves and Surrounds. *In:* Department of Environment and Climate Change Nsw (ed.). Hurstville.

Department of Environment Climate Change and Water NSW., 2010. NSW Climate Impact Profile: The impacts of climate change on the biophysical environments of New South Wales. Sydney.

Department of Environment and Conservation, 2004. The Native Vegetation of the Natti and Bargo Reserves. Unpublished Report. *In:* Department of Environment and Conservation NSW (ed.). Hurstville.

Department of Environment and Conservation, 2006. The vegetation of the Western Blue Mountains. Unpublished reported funded by the Hawkesbury Nepean Catchment Managment Authority. *In:* Department of Environment and Conservation Nsw (ed.). Hurstville.

Department of Environment and Conservation Nsw, 2008. Sydney South Region Pest Management Strategy 2008-2011. Sydney, NSW.

Desprez-Loustau, M. L., Robin, C., Reynaud, G., Deque, M., Badeau, V., Pioc, D., Husson, C. & Marcis, B., 2007. Simulating the effects of a climate-change scenario on the geographical range and activity of forest-pathogenic fungi. *Canadian Journal of Plant Pathology,* 29**,** 101-120.

Devadas, R., Lamb, D. W., Simpfendorfer, S. & Backhouse, D., 2009. Evaluating ten spectral vegetation indices for identifying rust infection in individual wheat leaves. *Precision Agriculture,* 10**,** 459-470.

Dieback.Org.Au., 2013. *What is Phytophthora dieback* [Online]. Available: http://www.dieback.org.au/go/what-is-dieback/what-is-phytophthora-dieback [Accessed 15/7/13].

Dobrowolski, M. P., Tommerup, I. C., Shearer, B. L. & O'brien, P. A., 2003. Three Clonal Lineages of *Phytophthora cinnamomi* in Australia Revealed by Microsatellites. *Ecology and Population Biology,* 83**,** 695-704.

Domburg, P., De Gruijter, J. J. & Van Beek, P., 1997. Designing efficient soil survey schemes with a knowledge-based system using dynamic programming. *Geoderma,* 75**,** 183-201.

Drenth, A. & Guest, D., 2004. Principals of Phytophthora Disease Management. *In:* Drenth, A. & Guest, D. (eds.) *Diversity and Management of Phytophthora in Southeast Asia. ACIAR Monograph 114.*

Drenth, A., Wagels, G., Smith, B., Sendall, B., O'dwyer, C., Irvine, G. & Irwin, J. A. G., 2006. Development of a DNA-based method for detection and identification of *Phytophthora* species. *Australasian Plant Pathology,* 35**,** 147-159.

Duncan, M. J. & Keane, P. J., 1996. Vegetation changes associated with *Phytophthora cinnamomi* and its decline under *Xanthorrhoea australis* in Kinglake National Park, Victoria. *Australian Journal of Botany,* 44**,** 355-369.

Elith, J., Graham, C. H., Anderson, R. P., Dudik, M., Ferrier, S., Guisan, A., Hijmans, R. J., Huettmann, F., Leathwick, J. R., Lehmann, A., Li, J., Lohmann, L. G., Loiselle, B. A., Manion, G., Moritz, C., Nakamura, M., Nakazawa, Y., Overton, J. M., Peterson, A. T., Phillips, S. J., Richardson, K., Scachetti-Pereira, R., Schapire, R. E., Soberon, J., Williams, S., Wisz, M. S. & Zimmermann, N. E., 2006. Novel methods improve prediction of species' distributions from occurrence data. *Ecography,* 29**,** 129-151.

Elith, J. & Leathwick, J. R., 2009. Species Distribution Models: Ecological Explanation and Prediction Across Space and Time. *Annual Review of Ecology Evolution and Systematics.*

Elvidge, C. D. & Chen, Z. K., 1995. Comparison of broad-band and narrow-band red and near-infrared vegetation indexes. *Remote Sensing of Environment,* 54**,** 38-48.

Environment Australia, 2008. Approved Conservation Advice for *Eucalyptus imlayensis* (Imlay Mallee).

Erden, T. & Coskun, M. Z., 2010. Multi-criteria site selection for fire services: the interaction with analytic hierarchy process and geographic information systems. *Natural Hazards and Earth System Sciences,* 10, 2127-2134.

Erwin & Ribeiro, 1996. *Phytopthora Diseases Worldwide,* St Paul, MN, APS Press.

Falcon, M. F., Fox, R. L. & Trujillo, E. E., 1984. Interactions of soil-ph, nutrients and moisture on Phytophthora root-rot of avocado. *Plant and Soil,* 81**,** 165-176.

Falk, M. G., Denham, R. J. & Mengersen, K. L., 2011. Spatially stratified sampling using auxiliary information for geostatistical mapping. *Environmental and Ecological Statistics,* 18.

Filella, I. & Penuelas, J., 1994. The red edge position and shape as indicators of plant chlorophyll content, biomass and hydric status. *International Journal of Remote Sensing,* 15**,** 1459-1470.

Fleischer, P., Bowles, F. A. & Richardson, M. D., 1998. Identification of potential sites for deep-ocean waste isolation with a geographic site-selection model. *Journal of Marine Systems,* 14, 241-271.

Fleming, G., Van Der Merwe, M. & Mcferren, G., 2007. Fuzzy expert systems and GIS for cholera health risk prediction in southern Africa. *Environmental Modelling & Software,* 22, 442-448.

Ford, A., 1990. Flora List of the Bare Rock Bluff area, including rainforest in Wollemi Creek.

Gadek, P. A. & Worboys, S., 2003. Rainforest Dieback Mapping and Assessment: *Phytophthora* species diversity and impacts of dieback in rainforest canopies.: Centre for Tropical Rainfores Ecology and Managment, Rainforest CRC, Cairns.

Ganley, R. J., Watt, M. S., Manning, L. & Iturritxa, E., 2009. A global climatic risk assessment of pitch canker disease. *Canadian Journal of Forest Research-Revue Canadienne De Recherche Forestiere,* 39**,** 2246-2256.

Gemitzi, A., Falalakis, G., Eskioglou, P. & Petalas, C., 2011. Evaluating landslide susceptibility using environmental factors, fuzzy membership functions and GIS. *Global Nest Journal,* 13, 28-40.

Gerrettson-Cornell, L., 1989. *A compendium and classification of the species of the genus Phytophthora de Bary by the canons of the traditional taxonomy.* Wood Technology and Forest Research Division Forestry Commision of New South Wales, Sydney.

Gillieson, D., Landsberg, J. J., Gadek, P. A. & Edwards, W. unpublished. Dieback Mapping and assessment in rainforests of tropical North Queensland. Available: www.tesag.jcu.edu.au/staf/davidg/rainforest_dieback2.htm [Accessed 16/3/2007].

Gisi, U. & Zentmyer, G. A., 1980. Mechanism of zoospore release in *Phytophthora* and *Pythium*. *Experimental Mycology,* 4**,** 362-377.

Gitelson, A. A. & Merzlyak, M. N., 1997. Remote estimation of chlorophyll content in higher plant leaves. *International Journal of Remote Sensing,* 18**,** 2691-2697.

Gitelson, A. A., Merzlyak, M. N. & Chivkunova, O. B., 2001. Optical properties and nondestructive estimation of anthocyanin content in plant leaves. *Photochemistry and Photobiology,* 74**,** 38-45.

Global Invasive Species Database., 2013. *Phytophthora cinnamomi* [Online]. Available: /http://www.issg.org/database/species/impact_info.asp?si=143&lang=EN [Accessed].

Graham, A. J., Atkinson, P. M. & Danson, F. M., 2004. Spatial analysis for epidemiology. *Acta Tropica,* 91**,** 219-225.

Grinnell, J., 1917. Field tests of theories concerning distributional control. *American Nature,* 51, 115-128.

Grisham, M. P., Johnson, R. M. & Zimba, P. V., 2010. Detecting Sugarcane yellow leaf virus infection in asymptomatic leaves with hyperspectral remote sensing and associated leaf pigment changes. *Journal of Virological Methods,* 167**,** 140-145.

Guisan, A. & Thuiller, W., 2005. Predicting species distribution: offering more than simple habitat models. *Ecology Letters,* 8**,** 993-1009.

Guisan, A. & Zimmermann, N. E., 2000. Predictive habitat distribution models in ecology. *Ecological Modelling,* 135**,** 147-186.

Hammil, K. & Tasker, L., 2010. *Vegetation, Fire and Climate Change in the Greater Blue Mountains World Heritge Area,* Sydney, Department of Environment Climate Change and Water NSW.

Harwood, T. D., Xu, X., Pautasso, M., Jeger, M. J. & Shaw, M. W., 2009. Epidemiological risk assessment using linked network and grid based modelling: *Phytophthora ramorum* and *Phytophthora kernoviae* in the UK. *Ecological Modelling,* 220**,** 3353-3361.

Hill, K., 1996. The Wollemi pine: Discovering a living fossil. *Nature & Resources,* 32**,** 20-25.

Hill, K., 1997. Architecture of the Wollemi Pine (*Wollemia nobilis*, Aracariaceae), a unique combination of model and reiteration. *Australian Journal of Botany,* 45**,** 817-826.

Hinch, J. & Weste, G., 1979. Behavior of *Phytophthora-cinnamomi* zoospores on roots of Australian forest species. *Australian Journal of Botany,* 27**,** 679-691.

Holdenrieder, O., Pautasso, M., Weisberg, P. J. & Lonsdale, D., 2004. Tree diseases and landscape processes: the challenge of landscape pathology. *Trends in Ecology & Evolution,* 19**,** 446-452.

Horler, D. N. H., Dockray, M. & Barber, J., 1983. The redge edge of plant leaf refelctance. *International Journal of Remote Sensing,* 4**,** 273-288.

Howard, C. G., 2008. *A contemporary study of the genetic variation of Phytophthora cinnamomi recovered from natural ecosystems of New South Wales.* PhD, The University of Sydney.

Huber, S., Kneubuhler, M., Psomas, A., Itten, K. & Zimmermann, N. E., 2008. Estimating foliar biochemistry from hyperspectral data in mixed forest canopy. *Forest Ecology and Management,* 256.

Huberli, D., Hardy, G. E. S., White, D., Williams, N. & Burgess, T. I., 2013. Fishing for Phytophthora from Western Australia's waterways: a distribution and diversity survey. *Australasian Plant Pathology,* 42**,** 251-260.

Huberli, D., Tommerup, I. C. & Hardy, G. E. S., 2000. False-negative isolations or absence of lesions may cause mis-diagnosis of diseased plants infected with *Phytophthora cinnamomi*. *Australasian Plant Pathology,* 29**,** 164-169.

Hutchinson, G. E., 1957. Concluding Remarks. *Cold Spring Harbour Symposia on Quantitative Biology*. 22, 415-427.

Hutchinson, G. E. 1959., Homage to Santa Rosalia, or why are there so many kinds of animals? *American Nature,* 93, 145-149.

Hutchinson, M. F., 2004. ANUCLIM 4.3. Canberra: Fenner School of Environment and Society, Australian National University.

Ireland, K. B., Hardy, G. E. S. J & Kriticos, D. J., 2013 Combining Inferential and Deductive Approaches to Estimate the Potential Geographic Range of the Invasive Plant Pathogen, *Phytophthora ramorum*. *PLoS ONE*. 8, e63508

Ireland, K. B., Huberli, D., Dell, B., Smith, I. W., Rizzo, D. M. & Hardy, G., 2012. Potential susceptibility of Australian flora to a NA2 isolate of *Phytophthora ramorum* and pathogen sporulation potential. *Forest Pathology,* 42**,** 305-320.

Irwin, J. A. G., Cahill, D. M. & Drenth, A., 1995. Phytophthora in Australia. *Australian Journal of Agricultural Research,* 46**,** 1311-1337.

Jackson, R. D., 1986. Remote-sensing of biotic and abiotic plant stress. *Annual Review of Phytopathology,* 24**,** 265-287.

Jackson, R. D. & Ezra, C. E., 1985. Spectral response of cotton to suddenly induced water stress. *International Journal of Remote Sensing,* 6**,** 177-185.

Jacquez, G. M., 2000. Spatial analysis in epidemiology: Nascent science or a failure of GIS? *Journal of Geographical Systems,* 2**,** 91-97.

James, T., 1994. An assessment of the World Heritage Area Values of the Blue Mountains and Surrounding Plateaus. Royal Botanic Gardens, Sydney.

James, E. A. & Mcdougall, K. L., 2007. Extent of clonality, genetic diversity and decline in the endangered mallee Eucalyptus imlayensis. *Australian Journal of Botany,* 55**,** 548-553.

Jeffers, S. N. & Aldwinckle, H. S., 1987. Enhancing detection of *Phytophthora-cactorum* in naturally infested soil. *Phytopathology,* 77**,** 1475-1482.

Jenolan Caves Reserve Trust, 1999. Wombeyan Karst Conservation Reserve Plan of Managment.

Jeschke, J. M. & Strayer, D. L., 2008. Usefulness of bioclimatic models for studying climate change and invasive species. *Year in Ecology and Conservation Biology 2008,* 1134**,** 1-24.

Jones, W. G., Hill, K. & Allen, J. M., 1995. *Wollemia nobilis*, a new living Australian genus and species in the Araucariaceae. *Telopea,* 3**,** 173-176.

Jurskis, V., 2005. Eucalypt decline in Australia, and a general concept of tree decline and dieback. *Forest Ecology and Management,* 215**,** 1-20.

Kearney, M., 2006. Habitat, environment and niche: what are we modelling? *Oikos,* 115**,** 186-191.

Keith, D., 2004. *Ocean Shores to Desert Dunes : the Native Vegetation of New South Wales and the ACT,* Sydney, Department of Environment and Conservation (NSW).

Keith, D. & Benson, D., 1988. The natural vegetation of the Katoomba map sheet. *Cunninghamia,* 2.

Keith, D., A., Mcdonald, K. L., Simpson, C., C. & Walsh, J., L., 2012. Spatial analysis of risks posed by the root rot pathgen *Phytopthora cinnamomi:* implications for disease managment. *Proceedings of the Linnean Society of New South Wales,* 134, B147-B179.

Keith, D. & Myerscough, P. J., 1993. Floristic and soil relations of upland swamp vegetation near Sydney. *Australian Journal of Ecology,* 18**,** 325-344.

Kelly, M., Guo, Q., Liu, D. & Shaari, D., 2007. Modeling the risk for a new invasive forest disease in the United States: An evaluation of five environmental niche models. *Computers Environment and Urban Systems,* 31**,** 689-710.

Kennedy, J. & Weste, G., 1986. Vegetation changes associated with invasion by *Phytophthora-cinnamomi* on monitored sites in the Grampians, western Victoria. *Australian Journal of Botany,* 34**,** 251-279.

Kitsiou, D. & Karydis, M., 2000. Categorical mapping of marine eutrophication based on ecological indices. *Science of the Total Environment,* 255, 113-127.

Kolivras, K. N., 2006. Mosquito habitat and dengue risk potential in Hawaii: A conceptual framework and GIS application. *Professional Geographer,* 58, 139-154.

Krejcie, R. V. & Morgan, D. W., 1970. Determining sample size for research activities. *Educational and Psychological Measurement,* 30, 607-&.

Kroon, L. P. N. M., Brouwer, H., De Cook, A. W. A. M. & Govers, F,. 2012. The genus *Phytophthora* Anno 2012. *Phytopathology,* 102**,** 348-364.

Laidlaw, W. S. & Wilson, B. A., 2003. Floristic and structural characteristics of a coastal heathland exhibiting symptoms of *Phytophthora cinnamomi* infestation in the eastern Otway Ranges, Victoria. *Australian Journal of Botany,* 51**,** 283-293.

La Manna, L., Matteucci, S. D. & Kitzberger, T., 2012. Modelling Phytophthora disease risk in Austrocedrus chilensis forests of Patagonia. *European Journal of Forest Research,* 131**,** 323-337.

Laurance, W. F., Dell, B., Turton, S. M., Lawes, M. J., Hutley, L. B., Mccallum, H., Dale, P., Bird, M., Hardy, G., Prideaux, G., Gawne, B., Mcmahon, C. R., Yu, R., Hero, J.-M., Schwarzkop, L., Krockenberger, A., Douglas, M., Silvester, E., Mahony, M., Vella, K., Saikia, U., Wahren, C.-H., Xu, Z., Smith, B. & Cocklin, C., 2011. The 10 Australian ecosystems most vulnerable to tipping points. *Biological Conservation,* 144**,** 1472-1480.

Lee, T. C. & Wicks, T. J., 1977. *Phytophthora cinnamomi* in native vegetation in South Australia. *APPS Newsletter,* 6**,** 22-23.

Linde, C., Drenth, A., Kemp, G. H. J., Wingfield, M. J. & von Broembsen, S. L., 1997. Population structure of *Phytophthora cinnamomi* in South Africa. *Phytopathology,* 87**,** 822-827.

Macdonald, J. D. & Duniway, J. M., 1978. Influence of the matric and osmotic components of water potential on zoospore discharge in *Phytophthora*. *Phytopathology,* 68**,** 751-757.

Macdonald, J. D., Stites, J. & Kabashima, J., 1990. Comparison of serological and culture plate methods for detecting species of *phytophthora, pythium,* and *rhizoctonia* in ornamental plants. *Plant Disease,* 74**,** 655-659.

Mack, R. N., Simberloff, D., Lonsdale, W. M., Evans, H., Clout, M. & Bazzaz, F. A., 2000. Biotic invasions: Causes, epidemiology, global consequences, and control. *Ecological Applications,* 10**,** 689-710.

Mahlein, A.-K., Rumpf, T., Welke, P., Dehne, H.-W., Plumer, L., Steiner, U. & Oerke, E.-C., 2013. Development of specral indicies for detecting and identifiying plant diseases. *Remote Sensing of Environment,* 128**,** 21-30.

Mahlein, A.-K., Steiner, U., Hilllnhutter, C., Dehne, H.-W. & Oerke, E.-C., 2012. Hyperspectral imaging for small-scale analysis of symptoms casued by different sugar beet diseases. *Plant Methods,* 8**,** 1-13.

Malajczuk, N. & Theodorou, C., 1979. Influence of water potential on growth and cultural-characteristics of *Phytophthora-cinnamomi*. *Transactions of the British Mycological Society,* 72**,** 15-18.

Marcais, B., Bergot, M., Perarnaud, V., Levy, A. & Desprez-Loustau, M. L., 2004. Prediction and mapping of the impact of winter temperature on the development of *Phytophthora cinnamomi*-induced cankers on red and pedunculate oak in France. *Phytopathology,* 94, 826-831.

Margosian, M. L., Garrett, K. A., Hutchinson, J. M. S. & With, K. A., 2009. Connectivity of the American Agricultural Landscape: Assessing the National Risk of Crop Pest and Disease Spread. *Bioscience,* 59**,** 141-151.

Marks, G. C., Kassaby, F. Y. & Fagg, P. C., 1975. Variation in population levels of *Phytopthora cinnamaomi* in Eucalyptus forest soils in Eastern Victoria. *Australian Journal of Botany,* 22, 345-49.

Marks, G. C., Kassaby, F. Y. & Reynolds, S. T., 1972. Die-back in mixed hardwood forests of eastern Victoria - preliminary report. *Australian Journal of Botany,* 20**,** 141-&.

Marks, G. C. & Smith, I. W., 1981. Factors influencing Suppression of Root Rot of *Eucalyptus sieberi* and *E. obliqua* caused by *Phytophthora cinnamomi. Australian Journal of Botany,* 29**,** 483-495.

Marryott-Brown, K. & Willis, D., 1993. Rare and Endagered Plants of Yengo National Park and Adjacent Areas. National Parks and Wildlife Service, NSW.

Maurel, M., Robin, C., Capron, G. & Desprez-Loustau, M. L., 2001. Effects of root damage associated with *Phytophthora cinnamomi* on water relations, biomass accumulation, mineral nutrition and vulnerability to water deficit of five oak and chestnut species. *Forest Pathology,* 31**,** 353-369.

Maurel, M., Robin, C., Simonneau, T., Loustau, D., Dreyer, E. & Desprez-Loustau, M. L., 2004. Stomatal conductance and root-to-shoot signalling in chestnut saplings exposed to *Phytophthora cinnamomi* or partial soil drying. *Functional Plant Biology,* 31**,** 41-51.

McCredie, T. A., Dixon, K. W. & Sivasithamparam, K., 1985. Variability in the resistance of *Banksia* species to *Phytophthora-cinnamomi* Rands. *Australian Journal of Botany,* 33**,** 629-637.

McDougall, K. L., 2005. The response of Native Australian Plant Species to *Phytophthora cinnamomi. Managment of Phytopthora cinnamomi for Biodiversity Conservation in Australia.* Centre for Phytophthora Science and Managment, Murdoch University, Western Australia.

McDougall, K. L., Hardy, G. E. S. & Hobbs, R. J., 2001. Additions to the host range of *Phytophthora cinnamomi* in the jarrah (Eucalyptus marginata) forest of Western Australia. *Australian Journal of Botany,* 49**,** 193-198.

McDougall, K. L., Hardy, G. E. S. & Hobbs, R. J., 2002. Distribution of *Phytophthora cinnamomi* in the northern jarrah (*Eucalyptus marginata*) forest of Western Australia in relation to dieback age and topography. *Australian Journal of Botany,* 50**,** 107-114.

McDougall, K. L., Hobbs, R. J. & Hardy, G. E. S., 2002. Vegetation of *Phytophthora cinnamomi*-infested and adjoining uninfested sites in the northern jarrah (*Eucalyptus marginata*) forest of Western Australia. *Australian Journal of Botany,* 50**,** 277-288.

McDougall, K. L., Hobbs, R. J. & Hardy, G. E. S., 2005. Distribution of understorey species in forest affected by *Phytophthora cinnamomi* in south-western Western Australia. *Australian Journal of Botany,* 53**,** 813-819.

McDougall, K. L. & Summerell, B. A., 2003. The impact of *Phytophthora cinnamomi* on the flora and vegetation of New South Wales-a re-appraisal. *In:* Mccomb, J., Hardy, G. E. S. & Tommerup, I. C. (eds.) *2nd international IUFRO working party* Albany Western Australia: Murdoch University Print: Perth.

McDougall, K. L., Summerell, B. A., Coburn, D. & Newton, M., 2003. *Phytophthora cinnamomi* causing disease in subalpine vegetation in New South Wales. *Australasian Plant Pathology,* 32**,** 113-115.

McLennan, E., Weste, G. & Parbery, D., 1973. The Earliest Known Record of *Phytophthora cinnamomi* in Victoria. *Australian Plant Pathology,* 2**,** 30-31.

Meentemeyer, R. K., rank, N. E., Anacker, B. L., Rizzo, D. M. & Cushman, J. H., 2008. Influence of land-cover change on the spread of an invasive forest pathogen. *Ecological Applications,* 18**,** 159-171.

Meentemeyer, R., Rizzo, D., Mark, W. & Lotz, E., 2004. Mapping the risk of establishment and spread of sudden oak death in California. *Forest Ecology and Management,* 200, 195-214.

Meyer, J. R. & Linderman, R. G., 1986. Selective influence on populations of rhizosphere or rhizoplane bacteria and actinomycetes by mycorrhizas formed by glomus-fasciculatum. *Soil Biology & Biochemistry,* 18**,** 191-196.

Minasny, B. & Mcbratney, A. B., 2006. A conditioned Laitn hypercube method for sampling in the presence of ancillary information. *Computers and Geoscience,* 32**,** 1378-1388.

Mircetich, S. M. & Zentmyer, G. A., 1966. Production of oospores and chlamydospores of *Phytophthora cinnamomi* in roots and soil. *Phytopathology,* 56**,** 1076-1078.

Moreira, A. C. & Martins, J. M. S., 2005. Influence of site factors on the impact of *Phytophthora cinnamomi* in cork oak stands in Portugal. *Forest Pathology,* 35**,** 145-162.

Moskowitz, H. & Bunn, D., 1987. Decision and risk analysis. *European Journal of Operational Research,* 28**,** 247-260.

Naidu, A. R., Perry, E. M., Pierceb, F. J. & Mekurina, T., 2009. The potential of spectral reflectance technique for the detection of Grapevine leafroll associated virus-3 in two red-berried wine grape cultivars. *Computers and Electronics in Agriculture,* 66**,** 38-45.

National Parks and Wildlife Service, 1997. Thirlmere Lakes National Park New Plan of Managment.

National Parks and Wildlife Service, 2001. The Natti Reserve Plan of Managment.

National Parks and Wildlife Service, 2010. Barrington Tops National Park, Mount Royal National Park, and Barrington Tops State Conservation Area plan of management. *In:* Department of Environment and Climate Change (NSW) (ed.). Sydney.

Nesbitt, H. J., Malajczuk, N. & Glenn, A. R., 1979. Effect of soil-moisture and temperature on the survival of *Phytophthora-cinnamomi* Rands in soil. *Soil Biology & Biochemistry,* 11**,** 137-140.

Newby, Z.-J., 2007. *Preserving Sydney's Heritage: Utilising phosphonate to protect endemic species susceptible to infection caused by Phytophthora cinnamomi.* Honours Thesis - Bachelor of Science in Agriculture, The University of Sydney.

Newell, G. R., 1998. Characterization of vegetation in an Australian open forest community affected by cinnamon fungus (*Phytophthora cinnamomi*): implications for faunal habitat quality. *Plant Ecology,* 137**,** 55-70.

Newhook, F. J. & Podger, F. D., 1972. Role of *Phytophthora cinnamomi* in Australian and New Zealand forests. *Annual Review of Phytopathology,* 10**,** 299-&.

Nicols, C. J., Hummrich, K. F., Black, T. A., Javis, P. G., Walthall, C. L., Grace, J. & Hall, F. G., 2000. Remote sensing of photosynthetic-light-use efficiency of boreal forest. *Agricultural and Forest Meterorology,* 101**,** 131-142.

NPWS & Environment Australia, 1998. The Greater Blue Mountains World Heritage Nomination. Unpublished report.

NSW Department of Environment and Climate Change, 2008. Statment of Intent 1: Infection of native plants by *Phytophthora cinnamomi*. Sydney, NSW, Australia.

NSW Department of Environment and Conservation, 2006. Wollemi Pine (*Wollemia nobilis*) Recovery Plan. *In:* Nsw Department of Environment and Conservation (ed.). Hurstville NSW.

O'Brien, P. A., Williams, N. & Hardy, G. E. S., 2009. Detecting Phytophthora. *Critical Reviews in Microbiology,* 35**,** 169-181.

Oerke, E. C. & Dehne, H. W., 2004. Safeguarding production - losses in major crops and the role of crop protection. *Crop Protection,* 23**,** 275-285.

Office of Environment and Heritage., 2011. *Infection of native plants by Phytophthora cinnamomi - key threatening process listing: NSW Scientific Committee - final determination* [Online]. Available:

http://www.environment.nsw.gov.au/determinations/PhytophthoraKTPListing.htm [Accessed 4/6/13].

Office of Environment and Heritage., 2005. *Threatened Speices database - Fletcher's Drumsticks* [Online]. Available: http://www.environment.nsw.gov.au/threatenedspeciesapp/profile.aspx?id=10440 [Accessed 13/7/12].

Office of Environment and Heritage., 2011. *Infection of native plants by Phytophthora cinnamomi - key threatening process listing: NSW Scientific Committee - final determination* [Online]. Available: http://www.environment.nsw.gov.au/determinations/PhytophthoraKTPListing.htm [Accessed 4/6/13].

O'Gara, E., Howard, K., Wilson, B. A. & Hardy, G. E. S., 2005. Managment of *Phytopthora cinnamomi* for Biodiversity Conservation in Australia. Centre for Phytophthora Science and Managment, Murdoch University, Western Australia.

Ohmann, J. L. & Gregory, M. J., 2002. Predictive mapping of forest composition and structure with direct gradient analysis and nearest-neighbor imputation in coastal Oregon, USA. *Canadian Journal of Forest Research-Revue Canadienne De Recherche Forestiere,* 32**,** 725-741.

Old, K. M., Dudzinshki, M. J., Bell, J. C., 1988. Isozyme variability in field populations of *Phytophthora cinnamomi* in Australia. *Australian Journal of Botany*. 36 355-360.

Old, K. M., Moran, G. F., Bell, J. C., 1984. Isozyme variability amoung isolates of *Phytophthora cinnamomi* from Australia and Papua New Guniea. *Canadian Journal of Botany*. 62, 2016-2022.

Ostfeld, R. S., Glass, G. E. & Keesing, F., 2005. Spatial epidemiology: an emerging (or re-emerging) discipline. *Trends in Ecology & Evolution,* 20**,** 328-336.

Oxford, 2008. Dictionary of Biology, 6th ed. Oxford: Oxford University Press.

Panagopoulos, T., Jesus, J., Antunes, M. D. C. & Beltrao, J., 2006. Analysis of spatial interpolation for optimising management of a salinized field cultivated with lettuce. *European Journal of Agronomy,* 24, 1-10.

Parks and Wildlife Service, 2000. Narawntapu National Park, Hawley Nature Reserve Management Plan. *In:* Department of Primary Industries Water and Environment Tasmania (ed.).

Penuelas, J., Filella, I., Biel, C., Serrano, L. & Save, R. ,1993. The reflectance at the 950-970 region as an indication of plant water stress. *International Journal of Remote Sensing,* 14.

Peters, D. & Weste, G., 1997. The impact of *Phytophthora cinnamomi* on six rare native tree and shrub species in the Brisbane Ranges, Victoria. *Australian Journal of Botany,* 45, 975-995.

Phillips, B. L., Chipperfield, J. D. & Kearney, M. R., 2008. The toad ahead: challenges of modelling the range and spread of an invasive species. *Wildlife Research,* 35**,** 222-234.

Phillips, D. & Weste, G., 1985. Growth Rates of four Austrlian isolates of *Phytophthora cinnamomi* in relation to temprature. . *Transactions of the British Mycological Society,* 84, 183-85.

Phillips, S. J., Anderson, R. P. & Schapire, R. E., 2006. Maximum entropy modeling of species geographic distributions. *Ecological Modelling,* 190**,** 231-259.

Pickard, J. & Jacobs, S. W. L., 1984. Vegetation patterns on the Sassafras Plateau, New South Wales *In:* Young, R. W. & Nanson, G. C. (eds.) *Topics in Australian sandstone environments.* Wollongong: Department of Geography, University of Wollongong.

Plantegenest, M., Le May, C. & Fabre, F., 2007. Landscape epidemiology of plant diseases. *Journal of the Royal Society Interface,* 4**,** 963-972.

Podger, F. D., 1972. *Phytophthora cinnamomi,* a casue of lethal disease in indigenous plant communitites in Western Australia. *Phytopathology,* 62**,** 672-678.

Podger, F. D., 1973. The role of Phythophthora cinnamomi in dieback diseases of Australian Eucalypt forests. *In:* BRUEHL, G. W. (ed.) *Biology and control of soil-borne plant pathogens. .* The American Pathological Society, St Paul, MN,.

Podger, F. D. & Ashton, D. H., 1970. *Phytophthora-cinnamomi* in dying vegetation on the Brisbane Ranges Victoria. *Australian Forest Research,* 4**,** 33-36.

Podger, F. D. & Batini, F., 1971. Susceptibility to *Phytophthora-cinnamomi* root rot of 36 species of Eucalyptus. *Australian Forest Research,* 5**,** 9-20.

Podger, F. D. & Brown, M. J., 1989. Vegetation damage caused by *Phytophthora-cinnamomi* on disturbed sites in temperate rain-forest in western Tasmania. *Australian Journal of Botany,* 37**,** 443-480.

Podger, F. D., Kile, G. A., Waitling, R. & Fryer, J., 1978. Spread and effects of *Armillaria luteobubalina* sp. nov. in an Australian *Eucalyptus regans* plantation. *Transactions of the British Mycological Society,* 71**,** 77-87.

Pozdnyakova, L., Oudemans, P. V., Hughes, M. G. & Gimenez, D., 2002. Estimation of spatial and spectral properties of phytophthora root rot and its effects on cranberry yield. *Computers and Electronics in Agriculture,* 37**,** 57-70.

Pratt, B. H. & Heather, W. A., 1973. Origin and distribution of *Phytophthora-cinnamomi* rands in Australian native plant communities and significance of its association with particular plant species. *Australian Journal of Biological Sciences,* 26, 559-573.

Pratt, B. H., Heather, W. A., Sedgley, J. H. & Shepherd, C. J., 1972. *Phytophthora cinnamaomi* Rands in natural vegetation. *Australian Plant Pathology Society Newsletter,* 1**,** 13.

Pratt, B. H., Heather, W. A. & Shepherd, C. J., 1973. Recovery of *Phytophthora-cinnamomi* from native vegetation in a remote area of New-South-Wales. *Transactions of the British Mycological Society,* 60, 197-204.

Pryce, J., Edwards, W. & Gadek, P. A., 2002. Distribution of *Phytophthora cinnamomi* at different spatial scales: When can a negative result be considered positively? *Austral Ecology,* 27**,** 459-462.

Pu, R., Kelly, M., Chen, Q. & Gong, P., 2008a. Spectroscopic determination of health levels in coast live oak (*Queruc agrifolia*) leaves. *Geocarto Interantional,* 23**,** 3-20.

Pu, R. L., Kelly, M., Anderson, G. L. & Gong, P., 2008b. Using CASI hyperspectral imagery to detect mortality and vegetation stress associated with a new hardwood forest disease. *Photogrammetric Engineering and Remote Sensing,* 74**,** 65-75.

Purse, B. V., Graeser, P., Searle, K., Edwards, C. & Harris, C., 2013. Challenges in predicting invasive reservoir hosts of emerging pathogens: mapping Rhododendron ponticum as a foliar host for *Phytophthora ramorum* and *Phytophthora kernoviae* in the UK. *Biological Invasions,* 15**,** 529-545.

R Development Core Team., 2010. R: A language and environement for statistical computing. Vienna, Austria: R Foundation for Statistical Computing.

Ray, S., Jain, N., Arora, R., Chavan, S. & Panigrapy, 2011. Utility of Hyperspectral Data for Potato Late Blight Disease Dectection. *Journal of the Indian Society of Remote Sensing,* 39**,** 161-169.

Reichard, S. & Hamilton., W., 1997 Predicting invasions of woody plants introduced into North America. *Conservation Biology* 11**,** 193 - 203.

Reynolds, G. J., Windels, C. E., Macrae, I. V. & Laguette, S., 2012. Remote Sensing for Assessing Rhizoctonia Crown and Root Rot Severity in Sugar Beet. *Plant Disease,* 96**,** 497-505.

Rhymer, J. & Simberloff., D., 1996. Extinction by hybridization and introgression. *Annual Review of Ecology and Systematics* 27**,** 83-109.

Ribeiro, O. K., 2013. A historical Perspective on *Phytophthora*. *In:* Lamour, K. (ed.) *Phytophthora A Global Perspective.* London, UK: CABI International.

Ristaino, J. B. & Gumpertz, M. L., 2000. New frontiers in the study of dispersal and spatial analysis of epidemics caused by species in the genus Phytophthora. *Annual Review of Phytopathology,* 38**,** 541

Rumpf, T., Mahlein, A.-K., Steiner, U., Oerke, E.-C., Dehne, H.-W. & Plumer, L., 2010. Early detection and classification of plant diseases with Support Vector Machines based on hyperspectral reflectance. *Computers and Electronics in Agriculture,* 74**,** 91-99.

Running, S. W. & Coughlan, J. C., 1988. A general model of forest ecosystem porcess for regional applications: I. Hydrologic balance, canopy gas exchange and primary production. *Ecological Modelling,* 42**,** 125-154.

Sankaran, S., Maja, J., Buchanon, S. & Ehsani, R., 2013. Huanglongbing (Citrus Greening) Detection Using Visible, Near Infrared and Thermal Imaging Techniques. *Sensors,* 13**,** 2117-2130.

Scarlett, K., Guest, D. & Daniel, R., 2012. Elevated soil nitrogen increases the severity of dieback due to *Phytophthora cinnamomi Australasian Plant Pathology,* 42**,** 155-162.

Schahinger, R., Rudman T. & Wardlaw, T. J., 2003. Conservation of Tasmanian Plant Species & Communities threatened by *Phytophthora cinnamomi*. Strategic Regional Plan for Tasmania. Technical Report 03/03, . *In:* Nature Conservation Branch Department of Primary Industries Water and Environment Hobart (ed.).

Seager, S., Turner, E. L. Schafer, J. & Ford, E. B., 2005 Vegetation's red edge: A possible spectroscopic biosignature of extraterrestrial plants. *Astrobiology*, 5, 372-390

Sellers, P. J., 1985. Canopy Reflectance, Photosynthesis and Transpiration. *International Journal of Remote Sensing,* 6**,** 1335-1372.

Shafri, H. Z. M., Anuar, M. I., Seman, I. A. & Noor, N. M., 2011. Spectral discrimination of healthy and Ganoderma-infected oil palms from hyperspectral data. *International Journal of Remote Sensing,* 32**,** 7111-7129.

Shearer, B. L. & Crane, C. E., 2011. Habitat suitability of soils from a topographic gradient across the Fitzgerald River National Park for invasion by *Phytophthora cinnamomi*. *Australasian Plant Pathology,* 40**,** 168-179.

Shearer, B. L., Crane, C. E., Barrett, S. & Cochrane, A., 2007. *Phytophthora cinnamomi* invasion, a major threatening process to conservation of flora diversity in the South-west Botanical Province of Western Australia. *Australian Journal of Botany,* 55**,** 225-238.

Shearer, B. L., Crane, C. E. & Cochrane, A., 2004. Quantification of the susceptibility of the native flora of the South-West Botanical Province, Western Australia, to *Phytophthora cinnamomi*. *Australian Journal of Botany,* 52**,** 435-443.

Shearer, B. L. & Dillon, M., 1996. Impact and disease centre characteristics of *Phytophthora cinnamomi* infestations of Banksia woodlands on the Swan Coastal Plain, Western Australia. *Australian Journal of Botany,* 44**,** 79-90.

Shearer, B. L. & Dillon, M., 1996b. Susceptibility of plant species in Banksia woodlands on the Swan Coastal Plain, Western Australia, to infection by *Phytophthora cinnamomi*. *Australian Journal of Botany,* 44**,** 433-445.

Simmonds, J. H., 1929. Disease of Pineapples. *Queensland Agricultural Journal,* 32**,** 398-405.

Sing, T., Sander, O., Beerenwinkel, N. & Lengauer, T., 2005. ROCR: visualizing classifier performance in R. *Bioinformatics* 21, 3940-3941

Smith, J., 1981. The distribution and conservtion status of a rare conifer, *Microstrobes fitzgeraldii* (Podocarpaceae). *Cunninghamia,* 1**,** 125-128.

Spriggs, P., 1962. *Our Blue Mountains Yesterdays,* Leaura, Blue Mountains , NSW, P. W. Spriggs.

Standards Australia/Standards New Zealand, 2009. Risk Managment- Principals and guidlines. Jointly published by Standards Australia, Sydney and Standards New Zealand, Wellington.

Stephens, J., 2000. A collection of the Native Flora at the Crags in the Natti Valley. Unpublised Report.

Sterne, R. E., Zentmyer, G. A. & Kaufmann, M. R., 1977. Influence of matric potential, soil texture, and soil amendment on root disease caused by *Phytophthora-cinnamomi*. *Phytopathology,* 67**,** 1495-1500.

Suddaby, T., 2008a. Survey of the distribution of *Phytophthora cinnamomi* in bushland of the Hawkesbury Nepean Catchment Management Authority Area. The Royal Botanic Gardens Sydney.

Suddaby, T., 2008b. Survey of the distribution of *Phytophthora cinnamomi* in bushland of the Sydney Metropolitan Catchment Management Authority area. The Royal Botanic Garden Sydney.

Suddaby, T., Alhussaen, K., Daniel, R. & Guest, D., 2008. Phosphonate alters the defence responses of Lambertia species challenged by *Phytophthora cinnamomi*. *Australian Journal of Botany,* 56**,** 550-556.

Swets, K. A., 1988. Measuring the accuracy of diagnostic systems. *Science,* 240**,** 1285-1293.

Thompson, S., Levin, S. & Rodriguez-Iturbe, L., 2013. Linking Plant Disease Risk and Precipitation Drivers: A Dynamical Systems Framework. *American Naturalist,* 181**,** E1-E16.

Tippett, J. T., Hill, T. C. & Shearer, B. L., 1985. Resistance of *Eucalyptus* spp to invasion by *Phytophthora-cinnamomi*. *Australian Journal of Botany,* 33**,** 409-418.

Treitz, P. M. & Howarth, P. J,. 1999. Hyperspectral remote sensing for estimating biophysical parameters of forest ecosystems. *Progress in Physical Geography,* 23**,** 359-390.

Tucker, C. J., 1979. Red and photographic infrared linear combination for monitoring vegetation. *Remote Sensing of Environment,* 8**,** 127-150.

Vaclavik, T., Kanaskie, A., Hansen, E. M., Ohmann, J. L. & Meentemeyer, R. K., 2010. Predicting potential and actual distribution of sudden oak death in Oregon: Prioritizing landscape contexts for early detection and eradication of disease outbreaks. *Forest Ecology and Management,* 260**,** 1026-1035.

Vaclavik, T., Kupfer, J. A. & meentemeyer, R. K., 2012. Accounting for multi-scale spatial autocorrelation improves performance of invasive species distribution modelling (iSDM). *Journal of Biogeography,* 39**,** 42-55.

VanDerWal, J., Shoo, L., P., Graham, C. & Williams, S. E., 2009. Selecting pseudo-absence data for presence-only distribution modeling: How far should you stray from what you know? *Ecological Modelling.* 220, 589-594

Venette, R. C. & Cohen, S. D., 2006. Potential climatic suitability for establishment of *Phytophthora ramorum* within the contiguous United States. *Forest Ecology and Management,* 231, 18-26.

Vettraino, A. M., Morel, O., Perlerou, C., Robin, C., Diamandis, S. & Vannini, A., 2005. Occurrence and distribution of *Phytophthora* species in European chestnut stands, and their association with Ink Disease and crown decline. *European Journal of Plant Pathology,* 111, 169-180.

Vos, J. J., 1978. Colorimetric and photometric properties of a 2-deg fundamental observer. *Color Research and Application, ,* 3**,** 125-128.

Vurro, M., Bonciani, B. & Vannacci, G., 2010. Emerging infectious diseases of crop plants in developing countries: impact on agriculture and socio-economic consequences. *Food Security,* 2**,** 113-132.

Walsh, J. L., Keith, D. A., Mcdougall, K. L., Summerell, B. A. & Whelan, R. J., 2006. Phytophthora Root Rot: Assessing the potential threat to Australia's oldest national park. *Ecological Management & Restoration,* 7**,** 55-60.

Weste, G., 1974. *Phytophthora-cinnamomi* - cause of severe disease in certain native communities in Victoria. *Australian Journal of Botany,* 22**,** 1-8.

Weste, G., 1975a. Distribution of *Phytophthora-cinnamomi* within a national park, Wilsons-Promontory, Victoria. *Australian Journal of Botany,* 23**,** 67-76.

Weste, G., 1975b. Pathogenicity of *Phytophthora-cinnamomi* towards *Nothofagus-cunninghamii*. *Australian Journal of Botany,* 23**,** 277-283.

Weste, G., 1994. Impact of *Phytophthora* species on native vegetation of Australia and Papua New Guinea. *Australasian Plant Pathology,* 23**,** 190-209.

Weste, G., 2003. The dieback cycle in Victorian forests: a 30-year study of changes caused by *Phytophthora cinnamomi* in Victorian open forests, woodlands and heathlands. *Australasian Plant Pathology,* 32**,** 247-256.

Weste, G., Brown, K., Kennedy, J. & Walshe, T., 2002. *Phytophthora cinnamomi* infestation - a 24-year study of vegetation change in forests and woodlands of the Grampians, Western Victoria. *Australian Journal of Botany,* 50**,** 247-274.

Weste, G., Cooke, D. & Taylor, P., 1973. Invasion of native forest by *Phytophthora-cinnamomi* .2. post-infection vegetation patterns, regeneration, decline in inoculum, and attempted control. *Australian Journal of Botany,* 21**,** 13-29.

Weste, G. & Kennedy, J., 1997. Regeneration of susceptible native species following a decline of *Phytophthora cinnamomi* over a period of 20 years on defined plots in the Grampians, western Victoria. *Australian Journal of Botany,* 45, 167-190.

Weste, G. & Marks, G. C., 1974. Distribution of *Phytophthora-cinnamomi* in Victoria. *Transactions of the British Mycological Society,* 63**,** 559-&.

Weste, G. & Marks, G. C., 1987. The biology of *Phytophthora-cinnamomi* in Australasian forests. *Annual Review of Phytopathology,* 25**,** 207-229.

Weste, G. & Ruppin, P., 1977. *Phytophthora-cinnamomi* - population-densities in forest soils. *Australian Journal of Botany,* 25**,** 461-475.

Weste, G. & Vithanage, K., 1978. Seasonal variation on numbers of chlamydospores in Victorian forest soils infected wtih *Phytophthora cinnamomi*. *Australian Journal of Botany,* 26**,** 657-662.

Weste, G. & Vithanage, K., 1979. Production of sporangia by *Phytophthora-cinnamomi* in forest soils. *Australian Journal of Botany,* 27**,** 693-701.

Weste, G. M. & Taylor, P., 1971. Invasion of native forest by *Phytophthora-cinnamomi* .1. Brisbane ranges, Victoria. *Australian Journal of Botany,* 19**,** 281

Whittaker, R. H., Levin, S. A. & Root, R. B. 1973., Niche, Habitat, and Ecotope. *The American Naturalist* 107, 321-338.

Wilcox, W. & Mircetich, S. M. 1979., Iinfluence of different levels of soil-moisture on Phytophthora root-rot and crown rot of mahaleb cherry rootstock. *Phytopathology,* 69**,** 1049-1049.

Wills, R. T. 1992., The ecological impact of *Phytophthora-cinnamomi* in the Stirling-Range-National-Park, Western-Australia. *Australian Journal of Ecology,* 18**,** 145-159.

Wilson, B. A., Aberton, J. & Cahill, D. M. 2000., Relationships between site factors and distribution of *Phytophthora cinnamomi* in the Eastern Otway Ranges, Victoria. *Australian Journal of Botany,* 48**,** 247-260.

Wilson, B. A., Lewis, A. & Aberton, J. 2003., Spatial model for predicting the presence of cinnamon fungus (*Phytophthora cinnamomi*) in sclerophyll vegetation communities in south-eastern Australia. *Austral Ecology,* 28, 108-115.

Wilson, B. A., Robertson, D., Moloney, D. J., Newell, G. R. & Laidlaw, W. S. 1990. Factors affecting small mammal distribution and abundance in the Eastern Otway Ranges, Victoria. *Proceedings of the Ecological Society of Australia,* 16**,** 379-396.

Wilson, B. A., Zdunic, K., Kinloch, J. & Behn, G., 2012. Use of remote sensing to map occurrence and spread of *Phytophthora cinnamomi* in Banksia woodlands on the Gnangara Groundwater System, Western Australia. *Australian Journal of Botany,* 60**,** 495-505.

Worboys, S. 2006., Rainfrorest Dieback Mapping and Assessment. 2004 Monitoring Report Including as Assessment of Dieback in Higt Altitude Rainforests.: Cooperative Research Centre for Tropical Rainforest Ecology and Mangament. Rainforest CRC, Cairns.

Xu, F. L., Tao, S., Dawson, R. W. & Li, B. G. 2001., A GIS-based method of lake eutrophication assessment. *Ecological Modelling,* 144, 231-244.

XU, X. M., HARWOOD, T. D., PAUTASSO, M. & JEGER, M. J. 2009., Spatio-temporal analysis of an invasive plant pathogen (*Phytophthora ramorum*) in England and Wales. *Ecography,* 32**,** 504-516.

Yalcin, A. & Bulut, F., 2007. Landslide susceptibility mapping using GIS and digital photogrammetric techniques: a case study from Ardesen (NE-Turkey). *Natural Hazards,* 41, 201-226.

Yang, C.-M., 2010. Assessment of the severity of bacterial leaf blight in rice using canopy hyperspctral reflectance. *Precision Agriculture,* 11**,** 61-81.

Yang, C. H., Everitt, J. H. & Fernandez, C. J., 2010. Comparison of airborne multispectral and hyperspectral imagery for mapping cotton root rot. *Biosystems Engineering,* 107**,** 131-139.

Yang, X. B., 2006. Framework development in plant disease risk assessment and its application. *European Journal of Plant Pathology,* 115**,** 25-34.

Yang, X. B., Dowler, W. M. & Royer, M. H., 1991. Assessing the risk and potential impact of an exotic plant-disease. *Plant Disease,* 75**,** 976-982.

Yoder, B. J. & Pettigrew-Crosby, R. E., 1995. Predicting nitrogen and chlorophyll content and concerntrations from reflectance spectra (400-2500 nm) at leaf and canopy scales. *Remote Sensing of Environment,* 53**,** 199-211.

Yusuf, B. L. & He, Y., 2011. Application of hyperspectral imaging sensor to differentiate between the moisture and reflectance of healthy and infected tobacco leaves. *African Journal of Agricultural Research,* 6**,** 6267-6280.

Zhang, J. & Zhang, C., 2012. Sampling and sampling strategies for environmental analysis. *International Journal of Environmental Analytical Chemistry,* 92**,** 466-478.

Zhang, M., Qin, Z., Liu, X. & Ustin, S., 2003. Detection of stress in tomatoes induced by late blight disease in California, USA, using hyperspectral remote sensing. *International Journal of Applied Earth Observation and Geoinformation,* 4**,** 295-310.

Zentmyer, G. A., 1980. *Phytophthora cinnamomi and the disease it casues*, American Pathological Society, St Paul, Minn.

Chapter 8: Appendices

Appendix 8.1

Host of *Phytophthora cinnamomi* growing in the GBMWHA as reported by McDougall (2005) in O'Gara *et al.* (2005) and other literature.

Species	*Status* [A]	Distribution reference [B]	Host score [C]	Susceptibility reference [D, E]
Acacia dealbata		13	1	
Acacia melanoxylon		13, 14	1	
Acacia myrtifolia		11, 14	3.5	
Acacia oxycedrus		11	3	
Acacia paradoxa		1, 3, 14	3	
Acacia parramattensis		12, 14	2	7
Acacia suaveolens		1, 3, 9, 11, 14	2	
Acacia terminalis	*E*	12, 14	1	3
Acrotriche serrulata		6, 9, 13	3	
Allocasuarina littoralis		3, 6, 9, 11, 13, 14	3	
Allocasuarina paludosa		14	4	
Allocasuarina verticillata		13	1	
Amperea xiphoclada		3, 6, 9 , 14	2	
Angophora costata		12, 13, 14	2	3
Anisopogon avenaceus		13, 14	1	
Aotus ericoides		9	4	
Astroloma humifusum		6, 13	4	
Banksia ericifolia		3, 6, 11, 14	3	
Banksia integrifolia		6	4	3
Banksia marginata		3, 6, 11	3	
Banksia paludosa		6	3	
Banksia serrata		3, 6, 9, 11, 14	4	3
Banksia spinulosa var collina		13	3.5	field observation
Banksia cunninghamii		3, 9	3	
Bauera rubioides		6, 11, 13, 13	2	
Bossiaea obcordata		3, 6, 9, 11, 13, 14	2	
Bossiaea prostrata		13, 14	4	
Brachyloma daphnoides		1, 3, 6, 11, 14	3	
Burchardia umbellata		13, 14	1	
Callitris rhomboidea		13	1	
Calytrix tetragona		1, 3, 6, 9	3	
Cassytha glabella		13, 14	1	
Cheilanthes austrotenuifolia		14	1	
Correa reflexa		1, 3, 6, 9, 13, 14	3	
Corymbia gummifera		6, 7, 10, 11, 13, 14	3	
Corymbia maculata		6	1	
*Cryptandra ericoides**		13	4	field observation and isolation
Daviesia latifolia		3, 6, 11, 13, 14	3	
Daviesia ulicifolia		1, 3, 6, 9, 11, 13, 14	3	
Dianella revoluta		1, 6, 13, 14	4	
Dillwynia glaberrima		6	4.5	
Dillwynia phylicoides		6, 13, 14	3	
Dillwynia sericea		3, 13	4	

Dodonaea viscosa		6, 13, 14	1	
Eucalyptus botryoides		6	2	3
Elaeocarpus holopetalus		Plantnet [#]	2	
Entolasia stricta		13, 14	1	
Epacris microphylla		12, 13, 14	5	field observation and isolation
Epacris obtusifolia		6, 11, 13, 14	3	
Epacris paludosa		6, 11, 13	5	
Eucalyptus camaldulensis	*E*	4	1	
Eucalyptus consideniana		6, 13	3	
Eucalyptus dalrymplenana		13	1	
Eucalyptus dives		2, 3, 6, 13	3	
Eucalyptus fastigata		2, 6, 13	4	
Eucalyptus globoidea		3, 8, 9 ,13, 14	3	
Eucalyptus macrorhyncha		3, 6, 13	3	
*Eucalyptus oblique**		13	3	
*Eucalyptus pauciflora**		13	1	
Eucalyptus radiata		2, 3, 6, 13, 14	3	
Eucalyptus rossii		13	2.5	7
Eucalyptus saligna		13	2	7
Eucalyptus sideroxylon		13, 14	5	2, 6, 8
Eucalyptus sieberi		2, 8, 9, 11, 13	2.5	
Eucalyptus smithii		13, 14	5	
Eucalyptus tereticornis		12, 13, 14	2	3
Eucalyptus viminalis		13, 14	1	
Gleichenia dicarpa		3, 6, 11, 14	3	
Gonocarpus teucrioides		13	1	
Goodenia hederacea s.l.		3, 9, 11, 13	3	
Gymnoschoenus sphaerocephalus		13	1	
Hakea dactyloides		1, 3, 9, 11, 14	3	
Hibbertia riparia		11, 13	3	
Hovea linearis		3, 6, 13, 14	3	
Isopogon anemonifolius		3, 6, 9, 11, 13	3	
Isopogon fletcheri	*V*	6	3	4
Lambertia formosa		6, 11, 13, 14	3	
Lepidosperma laterale		13, 14	1	
Lepidosperma longitudinale		13	1	
Lepidosperma urophorum		13, 14	1	
Leptospermum continentale		6, 9, 13, 14	3	
Leptospermum trinervium		6, 9, 11, 13	2	
Lepyrodia scariosa		13, 14	1	
Leucopogon ericoides		13, 14	4.5	
Leucopogon esquamatus		6, 13	4	
Leucopogon lanceolatus		13, 14	1	
Leucopogon virgatus		3, 13	3	
*Lindsaea linearis**		13	1	
Lobelia gibbosa		6, 4	1	
Lomanadra confertifolia		13, 14	1	
Lomandra longifolia		12 ,13	1	
Lomandra obliqua		14	1	
Lycopodium deuterodensum		3, 6, 13	3	
Macrozamia communis		6, 11, 13	3	
Melaleuca squamea		6, 13	5	
Melaluca styphelioides		13, 14	1	3
Monotoca elliptica		6, 9, 13	3	
Nematolepis squamea		13, 14	5	
Patersonia glabrata		1, 6, 9, 13, 14	3	
Persoonia levis		13, 14	1	

Persoonia linearis		13, 14	1	
Petrophile canescens		13	2	7
Petrophile pulchella		13, 14	2	7
Petrophile sessilis		14	2	7
Pimelea linifolia s.l.		1, 3, 9, 11, 13, 14	2	
Platylobium formosum		13	4.5	
Platysace lanceolata		12, 14	1	
Poa sieberiana		13, 14	1	
Polyscias murrayi		3	3	
Pteridium esculentum		13	1	
Pterostylis concinna		9	3	
Ptilothrix deusta		13, 14	1	
*Pultenaea daphnoides**		11, 14	5	
Pultenaea retusa		13	2	7
Pultenaea scabra		3, 6, 9, 11, 13	3	
Schoenus imberbis		13, 14	1	
Selaginella uliginosa		13, 14	4	
Sprengelia incarnata		6, 13	3	field observation and isolation
Stylidium graminifolium s. l.		6, 11, 13, 14	4	
Tetraria capillaris		13	1	
Tetrarrhena juncea		13	1	
Tetratheca glandulosa	*V*	5, 6	3	5
*Thelymitra pauciflora**		13	1	
Themeda triandra		11	3	
Tricoryne elatior		14	1	
*Wollemi nobilis**	*E*	16	4	1
Xanthorrhoea australis		13	4.5	
Xanthorrhoea glauca subsp. glauca		6	5	
Xanthosia atkinsoniana		13	1	
Ziera covenyi	*E*	17	3	5

[A] Conservation status defined as E: endangered, V: vulnerable

[B] Distribution references

1 (Benson, 1979)
2 (Cameron McNamara Consultants, 1988)
3 (Ford, 1990)
4 (DECC, 2008)
5 (Marryott-Brown and Willis, 1993)
6 (Benson *et al.,* 1996)
7 (National Parks and Wildlife Service, 1997)
8 (Jenolan Caves Reserve Trust, 1999)
9 (Stephens, 2000)
10 (National Parks and Wildlife Service, 2001)
11 (Baker and Corringham, 2004)
12 (Benson, 1992)
13 (DEC, 2006)
14 (DEC, 2004)
15 (Benson and Keith, 1990)
16 (Hill, 1996)
17 (Armstrong, 2002)

[C] Host score as per O'Gara *et al.* (2005) and other literature. 1: field resistant; 2: low susceptibility; 3: moderate susceptibility; 4: susceptible; 5: highly susceptible. Where two or more susceptibility ratings are given, these have been averaged. These values were applied to the construction of the host susceptibility scores in Chapter 5.

[D] Susceptibility reference other than O'Gara *et al.* (2005).

1 Bullock *et al.,* 2000
2 Marks *et al.,* 1972
3 Newby, 2007
4 Office of Environment and Heritage, 2005
5 Office of Environment and Heritage, 2011
6 Podger, 1973
7 Suddaby, 2008b
8 Weste *et al.,* 1973

[E] species which have their susceptibility reference indicated as 'field observation' or 'field observation and isolation' were observed repeated dying in areas from which *P. cinnamomi* was isolated during the field work. *Phytophthora cinnamomi* was been isolated from a number of these species also.

* Indicates species not included in modelling in Chapter 5 due to less than 30 species records in ALA.

[#] Species distribution as recorded on Plantnet: www.plantnet.rbgsyd.nsw.gov.au

Appendix 8.2 Phytophthora selective medium (PSM)

20 mls carrot puree
80 mls potato puree (recipes below)
20 g agar

Make up to 1 L with distilled water
Autoclave

When cooled to 60˚C, add 3.75 mL Hymexazol in water
400 mL Pimaricin
3.75 – 6.25 mL Rifampicin

Wrap the plates in plastic wrap and store them in the fridge out of the light. Discard after a month.

A. CARROT PUREE - 400 g carrots washed and diced, autoclaved 10 mins in 400 mL distilled water. Puree the mix, then add an additional 500 mL water. This can be measured out and frozen in plastic containers until needed.

B. POTATO PUREE - Dice 200 g potato and boil in 500 mL tap water until tender. Filter through 2 layers of cheesecloth and make up to a total of 800 mL with additional water. Store as above.

C. HYMEXAZOL STOCK SOLUTION - Add 0.3 g pure Hymexazol to 20 mL sterile water.

D. PIMARICIN - Pimaricin can be added directly to the molten agar. Shake well before dispensing. Store wrapped in foil in the fridge.

E. RIFAMPICIN STOCK – Add 0.15 g Rifampicin to 20 mL methanol in a clean McCartney bottle. Add 3.75 mL to 1000mL agar if plates are to be used straight away. If to be stored, add 6.5 mL.

Appendix 8.3 Chi-squared analysis of modelling results, Chapter 2.

	Risk Assessment values		expected values (%)	
strata	lower	upper	positive	negative
1	0.65	0.699	0.65	0.35
2	0.7	0.749	0.7	0.3
3	0.75	0.799	0.75	0.25
4	0.8	0.849	0.8	0.2
5	0.85	0.864	0.85	0.15

Raw Data

results				
strata	neg	P. spp	p. cinn	totals
1	351	19	1	371
2	293	25	9	327
3	360	49	91	500
4	550	94	213	857
5	97	10	15	122
	1651	197	329	2177

	neg	pos	X^2 test statistic		X^2 value @0.05, 1df
1	376.6448	202.8087	579.4535	sd	3.84
2	387.2172	165.9502	553.1675	sd	
3	441.8	147.2667	589.0667	sd	
4	836.2775	209.0694	1045.347	sd	
5	338.453	59.727	398.18	sd	

Stratum 4 and 5 combined

	neg	P. spp	p. cinn	total
1	351	19	1	371
2	293	25	9	327
3	360	49	91	500
4	587	104	288	979

	neg	pos	X^2 test statistic		X^2 value @0.05, 1df
1	376.6448	202.8087	579.4535	Sd	3.84
2	387.2172	165.9502	553.1675	Sd	
3	441.8	147.2667	589.0667	Sd	
4	781.6008	195.4002	977.001	sd	

Adjust data according to spatial proximity to positive samples

strata	neg	P. spp	p. cinn	totals
1	351	19	1	371
2	293	25	9	327
3	360	49	91	500
4	490	93	274	857
5	24	10	88	122
	1651	197	329	2177

	neg	pos	X^2 test statistic		X^2 value @0.05, 1df
1	376.6448	202.8087	579.4535	sd	3.84
2	387.2172	165.9502	553.1675	sd	
3	441.8	147.2667	589.0667	sd	
4	592.2168	148.0542	740.271	sd	
5	1.77541	0.313308	2.088717	nsd	

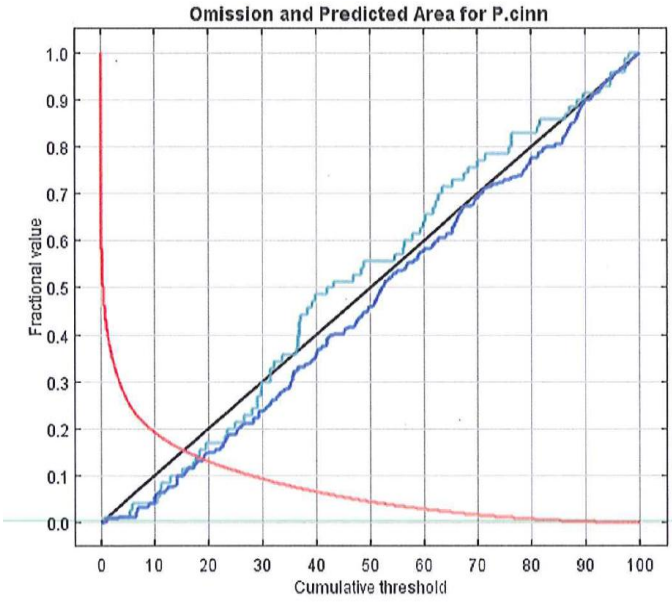
Appendix 8.4 Maxent output of *P. cinnamomi* distribution model

Maxent model for *P. cinnamomi*

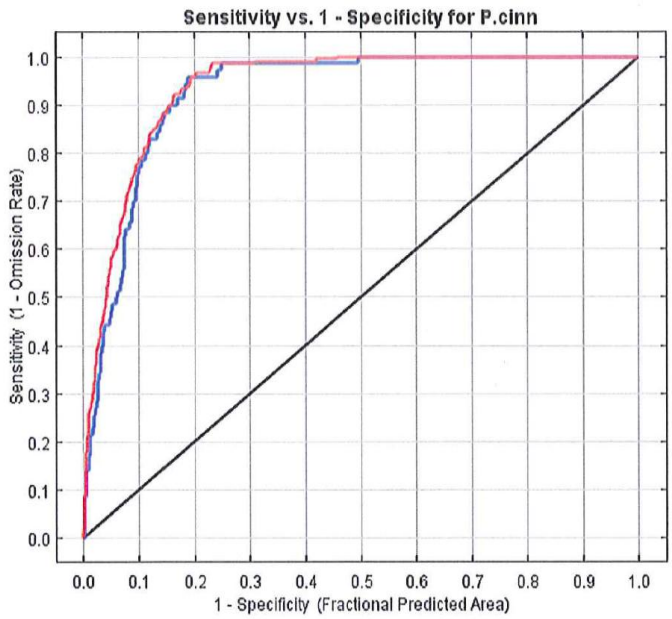
This page contains some analysis of the Maxent model for P.cinn, created Thu Dec 20 11:07:31 EST 2012 using Maxent version 3.3.3a. If you would like to do further analyses, the raw data used here is linked to at the end of this page.

Analysis of omission/commission

The following picture shows the omission rate and predicted area as a function of the cumulative threshold. The omission rate is is calculated both on the training presence records, and (if test data are used) on the test records. The omission rate should be close to the predicted omission, because of the definition of the cumulative threshold.



The next picture is the receiver operating characteristic (ROC) curve for the same data. Note that the specificity is defined using predicted area, rather than true commission (see the paper by Phillips, Anderson and Schapire cited on the help page for discussion of what this means). This implies that the maximum achievable AUC is less than 1. If test data is drawn from the Maxent distribution itself, then the maximum possible test AUC would be 0.922 rather than 1; in practice the test AUC may exceed this bound.



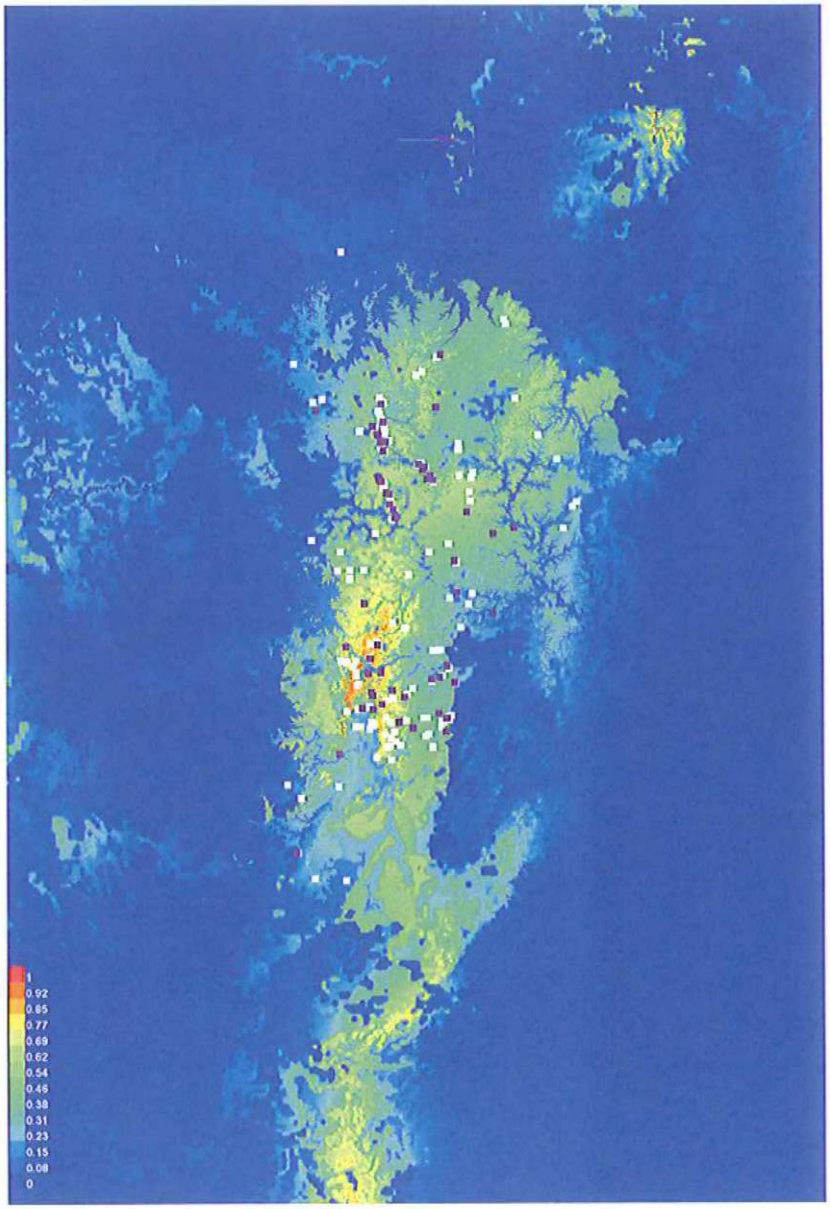
Some common thresholds and corresponding omission rates are as follows. If test data are available, binomial probabilities are calculated exactly if the number of test samples is at most 25, otherwise using a normal approximation to the binomial. These are 1-sided p-values for the null hypothesis that test points are predicted no better than by a random prediction with the same fractional predicted area. The "Balance" threshold minimizes 6 * training omission rate + .04 * cumulative threshold + 1.6 * fractional predicted area.

Cumulative threshold	Logistic threshold	Description	Fractional predicted area	Training omission rate	Test omission rate	P-value
1.000	0.022	Fixed cumulative value 1	0.418	0.009	0.014	2.898E-22
5.000	0.088	Fixed cumulative value 5	0.259	0.014	0.014	4.559E-44
10.000	0.181	Fixed cumulative value 10	0.193	0.057	0.043	2.767E-59
0.627	0.014	Minimum training presence	0.459	0.000	0.014	4.353E-19
15.290	0.253	10 percentile training presence	0.155	0.100	0.114	2.009E-64

18.956	0.298	Equal training sensitivity and specificity	0.136	0.137	0.157	0E0
9.902	0.180	Maximum training sensitivity plus specificity	0.194	0.043	0.043	5.427E-59
17.610	0.285	Equal test sensitivity and specificity	0.142	0.128	0.143	0E0
10.528	0.188	Maximum test sensitivity plus specificity	0.188	0.062	0.043	4.32E-61
3.128	0.056	Balance training omission, predicted area and threshold value	0.309	0.009	0.014	9.025E-35
9.419	0.171	Equate entropy of thresholded and original distributions	0.198	0.043	0.043	2.552E-57

Pictures of the model

This is a representation of the Maxent model for P.cinn. Warmer colors show areas with better predicted conditions. White dots show the presence locations used for training, while violet dots show test locations. Click on the image for a full-size version.

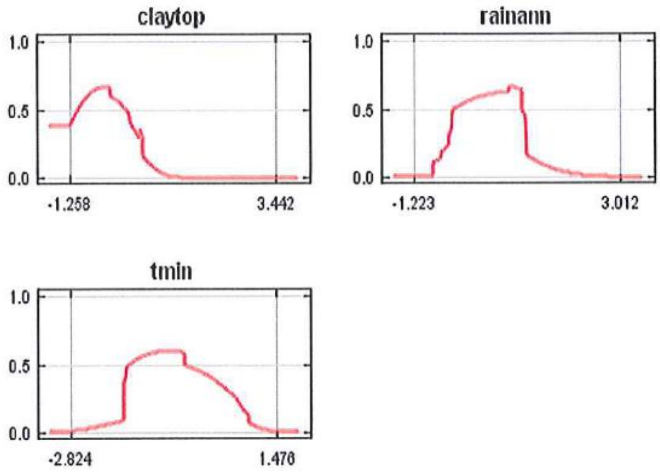


(A link to the Explain tool was not made for this model. The model uses product features, while the Explain tool can only be used for additive models.)

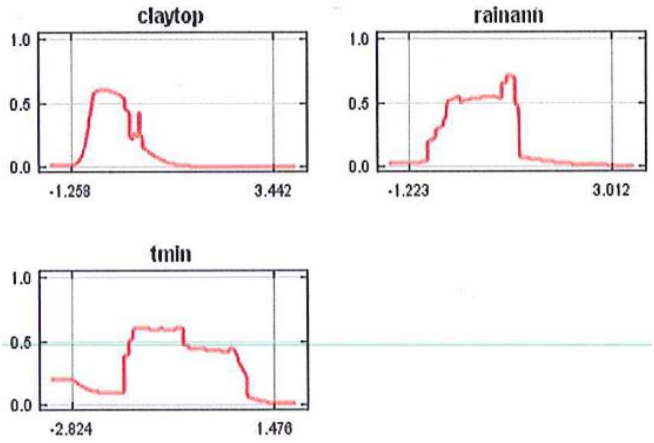
Response curves

These curves show how each environmental variable affects the Maxent prediction. The curves show how the logistic prediction changes as each environmental variable is varied, keeping all other environmental variables at their average sample value. Click on a response curve to see a larger version. Note that the curves can be hard to interpret if you have strongly correlated variables, as the model may depend on the correlations in ways that are not evident in the curves. In other words, the curves show the marginal effect of changing

exactly one variable, whereas the model may take advantage of sets of variables changing together.



In contrast to the above marginal response curves, each of the following curves represents a different model, namely, a Maxent model created using only the corresponding variable. These plots reflect the dependence of predicted suitability both on the selected variable and on dependencies induced by correlations between the selected variable and other variables. They may be easier to interpret if there are strong correlations between variables.



Analysis of variable contributions

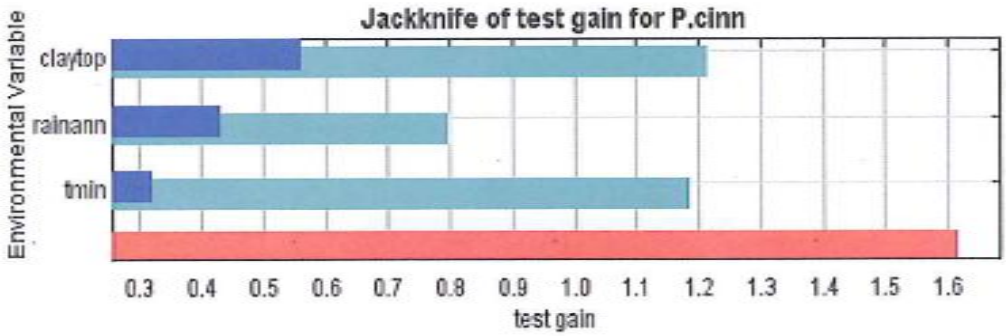
The following table gives estimates of relative contributions of the environmental variables to the Maxent model. To determine the first estimate, in each iteration of the training algorithm, the increase in regularized gain is added to the contribution of the corresponding variable, or subtracted from it if the change to the absolute value of lambda is negative. For the second estimate, for each environmental variable in turn, the values of that variable on training presence and background data are randomly permuted. The model

is reevaluated on the permuted data, and the resulting drop in training AUC is shown in the table, normalized to percentages. As with the variable jackknife, variable contributions should be interpreted with caution when the predictor variables are correlated.

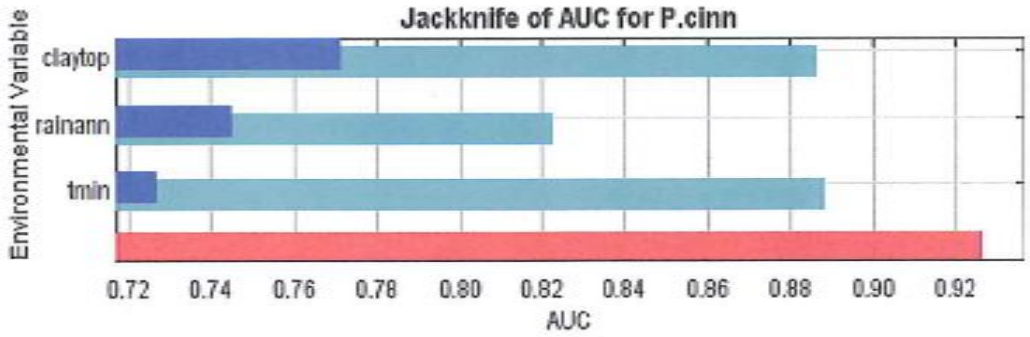
Variable	Percent contribution	Permutation importance
rainann	39.1	49.8
tmin	33.4	29.7
claytop	27.4	20.5

The following picture shows the results of the jackknife test of variable importance. The environmental variable with highest gain when used in isolation is claytop, which therefore appears to have the most useful information by itself. The environmental variable that decreases the gain the most when it is omitted is rainann, which therefore appears to have the most information that isn't present in the other variables.

The next picture shows the same jackknife test, using test gain instead of training gain. Note that conclusions about which variables are most important can change, now that we're looking at test data.



Lastly, we have the same jackknife test, using AUC on test data.



Raw data outputs and control parameters

The data used in the above analysis is contained in the next links. Please see the Help button for more information on these.
The model applied to the training environmental layers
The coefficients of the model
The omission and predicted area for varying cumulative and raw thresholds
The prediction strength at the training and (optionally) test presence sites
Results for all species modeled in the same Maxent run, with summary statistics and (optionally) jackknife results

Regularized training gain is 1.658, training AUC is 0.937, unregularized training gain is 1.797.
Unregularized test gain is 1.617.
Test AUC is 0.927, standard deviation is 0.009 (calculated as in DeLong, DeLong & Clarke-Pearson 1988, equation 2).
Algorithm terminated after 500 iterations (11 seconds).

The follow settings were used during the run:
211 presence records used for training, 70 for testing.
10207 points used to determine the Maxent distribution (background points and presence points).
Environmental layers used (all continuous): claytop rainann tmin
Regularization values: linear/quadratic/product: 0.050, categorical: 0.250, threshold: 1.000, hinge: 0.500
Feature types used: product linear quadratic hinge threshold
responsecurves: true
jackknife: true
randomtestpoints: 25

Appendix 8.5 Summary of Maxent statistics from species models

	AUC		Varible contribution				Gain				samples		
Species	Train	Test	rain	tmin	soil	wet	reg train	unreg train	unreg test	stnd div	train	test	b.ground
Acacia dealbata	0.888	0.867	43.3	51.9	5	1.8	1.336	1.501	1.215	0.032	120	39	10116
Acacia melanoxylon	0.849	0.819	41.8	34.5	18	5.7	0.729	0.906	0.753	0.022	275	91	10272
Acacia myrtifolia	0.877	0.877	68.1	17.8	12.6	1.4	0.946	1.079	1.065	0.009	604	201	10571
Acacia oxycedrus	0.962	0.94	51.8	26.9	20	1.3	2.035	2.319	1.806	0.009	200	66	10185
Acacia paradoxa	0.867	0.862	43.6	37.3	15.6	3.6	0.893	1.1011	0.988	0.01	712	237	10681
Acacia parramattensis	0.822	0.78	58.1	11.2	29.2	1.6	0.613	0.801	0.564	0.031	104	34	10100
Acacia suaveolens	0.867	0.871	57.7	23.3	18.2	0.8	0.926	1.009	0.986	0.008	919	306	10875
Acacia terminalis	0.857	0.855	52.4	24.4	22.7	0.4	0.845	0.949	0.855	0.008	1035	345	10989
Acrotriche serrulata	0.956	0.981	20.3	22.3	56.3	1.2	1.708	2.245	2.817	0.012	16	5	10016
Allocasuarina littoralis	0.841	0.826	39.3	16.4	43.1	1.2	0.774	0.834	0.753	0.008	1114	371	11053
Allocasuarina paludosa	0.935	0.869	35.5	34.5	20.4	9.6	1.323	1.757	1.206	0.041	59	19	10058
Allocasuarina verticillata	0.855	0.82	68.3	11.6	8.7	11.4	0.621	0.839	0.658	0.039	60	20	10059
Amperea xiphoclada	0.865	0.869	45.1	28.2	26	0.7	0.832	1.002	1.041	0.013	391	130	10374
Angophora costata	0.865	0.853	53.4	10.8	34.6	1.2	0.934	1.004	0.911	0.007	1269	423	11175
Anisopogon avenaceus	0.894	0.875	59.6	15.1	24.2	1.2	1.109	1.246	1.086	0.012	408	135	10387
Aotus ericoides	0.878	0.863	52	38.6	8.5	0.8	0.925	1.129	1.002	0.014	225	75	10220
Astroloma humifusum	0.861	0.788	39.7	20.6	32	7.7	0.792	0.972	0.599	0.05	192	63	10182
Banksia ericifolia	0.927	0.926	43.8	43	10.6	2.7	1.573	1.722	1.704	0.009	421	140	10370
Banksia integrifolia	0.936	0.936	20.8	64.4	14.4	0.4	1.695	1.83	1.834	0.013	135	45	10122
Banksia marginata	0.886	0.897	40.5	28.9	29.2	1.5	0.963	1.165	1.257	0.013	288	96	10272
Banksia paludosa	0.909	0.867	32.2	22.5	40.5	4.8	1.339	1.608	1.223	0.034	76	25	10072

	AUC		Varible contribution				Gain				samples		
Species	Train	Test	rain	tmin	soil	wet	reg train	unreg train	unreg test	stnd div	train	test	b.ground
Banksia spinulosa var cuningahmii	0.921	0.946	22.7	57.8	19.2	0.4	1.452	1.708	2.071	0.01	134	44	10129
Banksia cunninghamii	0.959	0.944	34.2	50.6	15	0.2	2.235	2.438	2.313	0.02	87	29	10086
Banksia serrata	0.885	0.874	53.7	22.1	22.7	1.4	1.066	1.162	1.08	0.008	800	266	10741
Bauera rubioides	0.922	0.894	51.6	28.1	20	0.3	1.408	1.579	1.305	0.012	389	129	10364
Bossiaea obcordata	0.862	0.84	54.9	26.7	16.5	1.9	0.821	1.002	0.883	0.012	483	160	10462
Bossiaea prostrata	0.886	0.914	16.9	50.8	21.8	10.5	1.046	1.289	1.95	0.022	88	29	10083
Brachyloma daphnoides	0.852	0.796	39.3	32.2	24.8	3.7	0.73	0.908	0.61	0.019	312	103	10286
Burchardia umbellata	0.891	0.903	62.7	22.8	8.2	6.3	1.002	1.197	1.277	0.014	169	56	10162
Callitris rhomboidea	0.909	0.896	48.4	25.7	15.3	0.6	1.086	1.436	1.326	0.02	114	38	10111
Calytrix tetragona	0.866	0.866	40	26	31.6	2.4	0.896	1.021	1.011	0.014	367	122	10344
Cassytha glabella	0.893	0.899	56.9	24.2	17.1	1.8	1.13	1.216	1.238	0.008	633	210	10590
Cheilanthes austrotenuifolia	0.909	0.875	41.3	43.7	13.6	1.4	1.236	1.459	1.227	0.0369	90	29	10084
Corymbia gumiferia	0.851	0.855	66.9	20.2	11.8	1.2	0.836	0.914	0.925	0.007	1272	424	11200
Corymbia maculata	0.896	0.894	8.7	79.7	10.6	0.9	1.212	1.259	1.247	0.006	840	280	10771
Correa reflexa	0.851	0.84	44.2	41.7	13.4	0.7	0.908	1.075	0.933	0.02	218	72	10204
Daviesia latifolia	0.94	0.884	31.1	54.7	13.5	0.6	1.69	1.994	1.386	0.038	87	29	10087
Daviesia ulicifolia	0.875	0.877	30.1	56.1	12.4	1.3	1.005	1.08	1.088	0.009	675	224	10647
Dianella revoluta	0.853	0.846	20.7	64.3	13.8	1.1	0.862	0.942	0.884	0.011	605	201	10570
Dillwynia glaberrima	0.936	0.945	25.5	30.2	37.3	7	1.578	1.808	1.994	0.015	99	33	10090
Dillwynia phylicoides	0.934	0.904	31.1	55.6	10.9	2.3	1.636	1.861	1.495	0.027	97	32	10092
Dillwynia sericea	0.886	0.856	51.1	11.2	32.2	5.5	0.871	1.243	1.058	0.026	112	37	10108
Dodonaea viscosa	0.833	0.788	44	36.8	16.4	2.9	0.702	0.825	0.588	0.019	300	100	10298
Elaeocarpus holopetalus	0.991	0.976	29.7	49.8	20	0.5	3.393	3.694	3.029	0.013	42	14	10041
Entolasia stricta	0.834	0.828	52.5	33.6	12.8	1.1	0.77	0.815	0.768	0.007	1590	529	11516

	AUC		Varible contribution				Gain				samples		
Species	Train	Test	rain	tmin	soil	wet	reg train	unreg train	unreg test	stnd div	train	test	b.ground
Epacris obtusifolia	0.919	0.93	46	6.6	42.7	2.7	1.443	1.612	1.623	0.008	295	98	10277
Epacris paludosa	0.939	0.838	31.3	42.6	25.3	0.8	1.763	2.101	1.006	0.035	126	41	10124
Epacris microphylla	0.877	0.871	47.8	12.5	37.9	1.8	0.987	1.121	1.027	0.009	607	202	10562
Eucalyptus botryoides	0.944	0.926	25.7	63.7	9.4	1.2	1.782	1.923	1.641	0.011	207	69	10196
Eucalyptus camaldulensis	0.931	0.928	10.8	65.5	22.4	1.2	1.764	1.816	1.796	0.003	1668	556	11585
Eucalyptus consideniana	0.907	0.849	46.2	22	25.7	6.1	1.026	1.414	0.894	0.023	118	39	10118
Eucalyptus dalrymplenana	0.963	0.978	32.3	66.1	1.2	0.6	2.414	2.59	2.84	0.005	87	29	10087
Eucalyptus dives	0.956	0.946	32	55.1	10.1	2.8	1.928	2.199	2.074	0.019	110	36	10109
Eucalyptus fastigata	0.918	0.898	23.8	43.4	30.2	2.6	1.472	1.706	1.312	0.026	108	35	10107
Eucalyptus globoidea	0.836	0.832	69	16.5	11.1	3.5	0.719	0.82	0.799	0.011	633	210	10604
Eucalyptus macrorhyncha	0.96	0.936	51.9	35.6	10	2.5	1.512	2.025	1.765	0.026	24	8	10024
Eucalyptus radiata	0.94	0.941	38.1	59	3.9	1.9	1.806	1.961	1.837	0.009	196	65	10195
Eucalyptus rossii	0.939	0.898	45.7	34.7	19	0.6	1.489	1.854	1.313	0.019	113	37	10109
Eucalyptus saligna	0.88	0.872	45.9	44.9	8	1.3	1.012	1.122	1.035	0.011	464	154	10442
Eucalyptus sideroxylon	0.918	0.897	14.5	67.4	7.3	10.8	1.416	1.716	1.507	0.023	125	41	10120
Eucalyptus sieberi	0.855	0.842	24.9	17.1	35.7	0.4	0.806	0.927	0.881	0.011	585	194	10567
Eucalyptus smithii	0.946	0.937	50.3	36.5	11.9	1.2	1.495	1.922	1.882	0.018	121	40	10118
Eucalyptus tereticornis	0.852	0.845	11.4	75.5	11.5	1.7	0.886	0.923	0.887	0.009	921	306	10872
Eucalyptus viminalis	0.876	0.883	33	43.6	19.9	3.5	0.994	1.166	1.189	0.021	196	65	10195
Gleichenia dicarpa	0.898	0.907	49.3	19.9	30.1	0.8	1.081	1.246	1.342	0.01	460	153	10437
Gonocarpus teucrioides	0.864	0.875	59.2	24.8	15.5	0.5	0.891	0.986	1.051	0.009	669	223	10640
Goodenia hederacea s.l.	0.858	0.846	23.8	51.7	18.8	5.7	0.855	0.956	0.879	0.012	438	146	10414
Gymnoschoenus sphaerocephalus	0.938	0.906	32.8	16.7	47.2	3.2	1.395	1.695	1.605	0.034	81	26	10079
Hakea dactyloides	0.853	0.821	47.7	24.9	26.3	1.1	0.809	0.917	0.726	0.01	795	265	10746

	AUC		Varible contribution				Gain				samples		
Species	Train	Test	rain	tmin	soil	wet	reg train	unreg train	unreg test	stnd div	train	test	b.ground
Hibbertia riparia	0.878	0.87	55.8	25.5	13.2	5.5	0.923	1.142	1.116	0.027	140	46	10133
Hovea linearis	0.868	0.882	38	44.5	16.5	1	0.889	1.015	1.111	0.01	493	164	10470
Isopogon anemonifolius	0.852	0.837	56.7	20.2	20.8	2.3	0.795	0.897	0.833	0.01	801	267	10771
Isopogon fletcheri	0.995	0.996	44.9	51.2	3.5	0.4	3.659	4.303	4.103	0.001	21	7	10021
Lambertia formosa	0.864	0.86	66.7	17.4	14.9	1	0.887	0.978	0.969	0.008	963	321	10909
Leptospermum continentale	0.885	0.924	38.1	27.7	26.5	7.7	0.871	1.189	1.577	0.015	120	39	10115
Lepidiosperma laterale	0.82	0.827	50	40.6	8.9	0.5	0.649	0.716	0.748	0.008	1253	417	11208
Lepidosperma longitudinale	0.894	0.913	35.6	18	39.1	7.3	1.047	1.344	1.441	0.043	37	12	10032
Lepyrodia scariosa	0.898	0.903	55.9	13.6	28	2.4	1.167	1.294	1.331	0.008	636	211	10587
Leptospermum trinervium	0.834	0.831	62.3	18.7	18	1	0.722	0.8	0.769	0.007	1304	434	11251
Lepidosperma urophorum	0.83	0.784	39.9	19.5	36.5	4.1	0.62	0.821	0.583	0.04	65	21	10064
Leucopogon ericoides	0.878	0.876	47.3	29.1	21.4	2.1	0.922	1.107	1.086	0.013	345	115	10328
Leucopogon esquamatus	0.924	0.907	58.6	6.8	33.5	1.1	1.46	1.708	1.396	0.017	162	53	10151
Leucopogon lanceolatus	0.837	0.829	56.7	21.4	21.8	0.1	0.669	0.784	0.782	0.013	522	173	10514
Leucopogon virgatus	0.856	0.827	36.4	33.2	23.9	6.5	0.803	1.054	0.762	0.029	144	47	10138
Lobelia gibbosa	0.862	0.841	35.1	22.5	33.2	9.3	0.793	1.203	0.807	0.046	30	9	10029
Lomanadra confertifolia	0.84	0.817	42.1	39.6	17.3	1	0.767	0.861	0.748	0.015	349	116	10339
Lomandra longifolia	0.804	0.802	42.8	44.3	12.5	0.5	0.607	0.646	0.652	0.007	1800	599	11715
Lomandra obliqua	0.851	0.846	62.6	19.4	17.1	0.9	0.816	0.907	0.882	0.009	917	305	10878
Lycopodium deuterodensum	0.908	0.898	47.3	45.3	7.1	0.2	1.47	1.716	1.336	0.022	126	42	10125
Macrozamia communis	0.919	0.912	24.3	66.7	7.8	1.2	1.492	1.642	1.55	0.014	216	72	10212
Melaleuca squamea	0.961	0.96	31.8	28.8	37.2	2.1	1.815	2.5	2.462	0.027	30	9	10030
Melaluca styphelioides	0.895	0.848	19.4	72.9	6.7	1.1	1.167	1.261	0.886	0.014	377	125	10358
Monotoca elliptica	0.944	0.942	21.2	51.5	26.5	0.9	1.885	2.109	2.02	0.014	189	62	10174

	AUC		Varible contribution				Gain				samples		
Species	Train	Test	rain	tmin	soil	wet	reg train	unreg train	unreg test	stnd div	train	test	b.ground
Nematolepis squamea	0.876	0.803	72.2	11.2	13.5	2.7	0.842	1.093	0.606	0.041	60	20	10056
Patersonia glabrata	0.902	0.882	44.3	41.3	13.8	0.6	1.2	1.337	1.144	0.012	396	132	10383
Persoonia levis	0.859	0.858	63.6	19.7	15.9	0.8	0.855	0.95	0.947	0.008	955	318	10909
Persoonia linearis	0.798	0.782	49.8	31.1	18.6	0.5	0.59	0.632	0.586	0.008	1228	409	11176
Petrophile canescens	0.951	0.924	28.6	49.2	12.8	9.5	1.943	2.355	1.759	0.04	33	10	10032
Petrophile pulchella	0.894	0.88	60.3	12.6	24.6	2.5	1.119	1.241	1.15	0.009	750	250	10695
Petrophile sessilis	0.887	0.835	51.2	5.8	38.5	4.6	1.051	1.34	0.936	0.038	99	32	10093
Pimelea linifolia s.l.	0.835	0.822	56.8	27.2	14.4	1.6	0.731	0.799	0.732	0.008	1236	411	11184
Platylobium formosum	0.924	0.906	40.9	48.5	10.6	0.1	1.405	1.581	1.422	0.012	305	101	10297
Platysace lanceolata	0.849	0.839	46.6	27	23.9	2.5	0.795	0.915	0.83	0.012	493	164	10464
Poa sieberiana	0.868	0.805	38.9	49.6	9	2.6	1.012	1.179	0.75	0.023	263	87	10260
Polyscias murrayi	0.867	0.87	52.2	19.8	27.6	0.4	0.845	1.063	1.106	0.036	45	14	10042
Pteridium esculentum	0.946	0.928	10.8	37.6	51.5	0	2.557	2.802	1.982	0.044	51	16	10050
Pterostylis concinna	0.821	0.809	45.1	42.7	12	0.2	0.666	0.762	0.668	0.008	1529	509	11466
Ptilothrix deusta	0.898	0.909	56.9	12.6	27.4	3.1	1.213	1.357	1.449	0.011	384	127	10344
Pultenaea retusa	0.902	0.885	36.3	42.1	14.1	7.6	1.195	1.398	1.268	0.019	171	57	10169
Pultenaea scabra	0.865	0.849	65.2	11.8	21.8	1.2	0.969	1.103	0.968	0.018	274	91	10266
Schoenus imberbis	0.936	0.906	35.5	36.1	27.5	0.9	1.602	1.784	1.369	0.01	333	110	10315
Selaginella uliginosa	0.902	0.867	62.7	16	19.6	1.6	1.088	1.3	1.021	0.016	189	62	10183
Sprengelia incarnata	0.923	0.892	51.3	11.3	34.5	2.9	1.423	1.632	1.25	0.015	234	77	10209
Stylidium graminifolium s. l.	0.842	0.834	49.3	34	14	2.8	0.723	0.857	0.821	0.014	361	120	10349
Tetraria capillaris	0.901	0.899	63.8	8.6	25	2.6	1.055	1.321	1.238	0.023	78	25	10074
Tetratheca glandulosa	0.941	0.94	20.8	54.9	22.7	1.6	1.782	1.855	1.845	0.004	790	263	10721
Tetrarrhena juncea	0.935	0.913	59	20	17.9	3	1.533	1.813	1.524	0.016	142	47	10132

	AUC		Varible contribution				Gain				samples		
Species	Train	Test	rain	tmin	soil	wet	reg train	unreg train	unreg test	stnd div	train	test	b.ground
Themeda triandra	0.835	0.927	17.6	71.7	8.4	2.3	0.778	0.814	0.772	0.008	1291	430	11244
Tricoryne elatior	0.909	0.903	1.7	77.7	11.4	9.2	1.284	1.407	1.344	0.014	203	67	10190
Xanthosia atkinsoniana	0.9	0.944	44.7	20.7	28.8	5.8	1.121	1.51	1.873	0.014	61	20	10061
Xanthorrhoea australis	0.911	0.824	1.3	0.4	97.5	0.9	1.458	1.945	1.36	0.129	12	3	10012
Xanthorrhoea glauca	0.854	0.956	4.9	0	80.4	14.7	0.434	0.711	1.204	0.008	13	4	10013
Ziera covenyi	0.997	0.998	38.6	54	3.3	4.1	4.636	4.954	5.263	0	41	13	10041
Sum	**0.891**	**0.8789**	**41.946**	**33.73**	**21.4**	**2.672**	**1.2116**	**1.4103098**	**1.2850163**	**0.01784**	**410**	**136.2**	**10389.63**
Standard deviation	**0.042**	**0.0487**											

Appendix 8.6: ANOVA output for water features.

Treatments are as follows: c: control; w: water stress; p:inoculation; wsp: combination treatment.

Angophora costata

		Area	Depth
28/06/12	1021	> **WF1021_ACarea<- aov(area ~ trt, data=WF1021_AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 59.21 19.735 9.65 1.03e-05 Residuals 111 227.00 2.045 > **TukeyHSD(WF1021_ACarea)** diff lwr upr p adj p-c 1.0774972 0.01907861 2.1359158 0.0443459 ws-c 0.9517411 -0.05531183 1.9587941 0.0711479 wsp-c 2.0937736 1.06724663 3.1203006 0.0000032 ws-p -0.1257561 -1.08728383 0.8357717 0.9862699 wsp-p 1.0162764 0.03437131 1.9981815 0.0395318 wsp-ws 1.1420325 0.21572617 2.0683388 0.0091132	**> WF1021_ACdepth<- aov(depth ~ trt, data=WF1021_AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.007125 0.0023751 9.528 1.19e-05 Residuals 111 0.027671 0.0002493 > **TukeyHSD(WF1021_ACdepth)** diff lwr upr p adj p-c 0.010976147 -0.0007096585 0.02266195 0.0737647 ws-c 0.010099435 -0.0010192519 0.02121812 0.0890904 wsp-c 0.022881442 0.0115477454 0.03421514 0.0000041 ws-p -0.000876712 -0.0114927639 0.00973934 0.9964497 wsp-p 0.011905295 0.0010642600 0.02274633 0.0253788 wsp-ws 0.012782006 0.0025548281 0.02300918 0.0079554
	1227	**> WF1227_ACarea<- aov(area ~ trt, data=WF1227_AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 465 154.92 1.776 0.156 Residuals 111 9683 87.23	> **WF1227_ACdepth<- aov(depth ~ trt, data=WF1227_AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00498 0.001661 1.588 0.196 Residuals 111 0.11603 0.001045
	1614	**> WF1614_ACarea<- aov(area ~ trt, data=WF1614_AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.738 0.2460 1.062 0.368 Residuals 111 25.716 0.2317	> **WF1614_ACdepth<- aov(depth ~ trt, data=WF1614_AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000041 1.355e-05 0.44 0.725 Residuals 111 0.003420 3.081e-05
	1797	> **WF1797_ACdepth<- aov(depth ~ trt, data=WF1797_AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00179 0.0005973 1.998 0.118 Residuals 111 0.03318 0.0002989	**> WF1797_ACarea<- aov(area ~ trt, data=WF1797_AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 294 98.10 1.682 0.175 Residuals 111 6475 58.33
17/07/12	1021	**> wf1021acarea<- aov(area ~ trt, data=wf1021ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 7.74 2.579 1.288 0.282 Residuals 108 216.15 2.001	**> wf1021acdepth<- aov(depth ~ trt, data=wf1021ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000692 0.0002306 0.994 0.399 Residuals 108 0.025064 0.0002321
	1227	> **wf1227aca<- aov(area ~ trt, data=wf1227ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 369 122.88 1.403 0.246 Residuals 108 9458 87.57	> **wf1227acdepth<- aov(depth ~ trt, data=wf1227ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00412 0.001373 1.339 0.266 Residuals 108 0.11073 0.001025

	1614	> **wf1614acarea<- aov(area ~ trt, data=wf1614ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 2.52 0.8387 2.756 0.046 Residuals 108 32.87 0.3044 > **TukeyHSD(wf1614acarea)** diff lwr upr p adj p-c 0.01609991 -0.39141348 0.42361330 0.9996053 ws-c -0.30551139 -0.69425439 0.08323161 0.1761392 wsp-c 0.04982951 -0.34442785 0.44408687 0.9875557 ws-p -0.32161130 -0.70171449 0.05849188 0.1275978 wsp-p 0.03372960 -0.35201148 0.41947068 0.9957887 wsp-ws 0.35534091 -0.01051444 0.72119625 0.0602729	> **wf1614acd<- aov(depth ~ trt, data=wf1614ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000306 1.018e-04 3.388 0.0207 Residuals 108 0.003247 3.007e-05 **TukeyHSD(wf1614acarea)** diff lwr upr p adj p-c 8.581124e-05 -0.0039644255 0.0041360479 0.9999390 ws-c -3.054272e-03 -0.0069179520 0.0008094071 0.1719998 wsp-c 1.153915e-03 -0.0027645711 0.0050724014 0.8684971 ws-p -3.140084e-03 -0.0069178930 0.0006377256 0.1385422 wsp-p 1.068104e-03 -0.0027657398 0.0049019477 0.8860917 wsp-ws 4.208188e-03 0.0005719863 0.0078443890 0.0164210
	1797	> **wf1614acarea<- aov(area ~ trt, data=wf1614ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 2.52 0.8387 2.756 0.046 Residuals 108 32.87 0.3044 > **TukeyHSD(wf1614acarea)** diff lwr upr p adj p-c 0.01609991 -0.39141348 0.42361330 0.9996053 ws-c -0.30551139 -0.69425439 0.08323161 0.1761392 wsp-c 0.04982951 -0.34442785 0.44408687 0.9875557 ws-p -0.32161130 -0.70171449 0.05849188 0.1275978 wsp-p 0.03372960 -0.35201148 0.41947068 0.9957887 wsp-ws 0.35534091 -0.01051444 0.72119625 0.0602729	> **wf1797acdepth<- aov(depth ~ trt, data=wf1797ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00229 0.0007617 1.466 0.228 Residuals 108 0.05610 0.0005194
16/08/12	1021	> **ac1021a<- aov(area ~ trt, data=wf1021ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 16.7 5.577 1.69 0.173 Residuals 108 356.4 3.300	> **ac1021d<- aov(depth ~ trt, data=wf1021ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00196 0.0006544 1.982 0.121 Residuals 108 0.03566 0.0003302
	1227	> **ac1227a<- aov(area ~ trt, data=wf1227ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 939 313.1 1.416 0.242 Residuals 108 23886 221.2	> **ac1227d<- aov(depth ~ trt, data=wf1227ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0199 0.006639 1.543 0.208 Residuals 108 0.4648 0.004304
	1614	> **ac1614a<- aov(area ~ trt, data=wf1614ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.38 1.4605 2.496 0.0637 Residuals 108 63.19 0.5851	> **ac1614d<- aov(depth ~ trt, data=wf1614ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000090 3.016e-05 0.556 0.645 Residuals 108 0.005854 5.421e-05
	1797	> **ac1797a<- aov(area ~ trt, data=wf1797ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 1 824 824.4 2.729 0.108 Residuals 35 10574 302.1	> **ac1797d<- aov(depth ~ trt, data=wf1797ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 1 0.000074 0.0000743 0.157 0.694 Residuals 35 0.016527 0.0004722
30/08/12	1021	> **ac1021a<- aov(area ~ trt, data=wf1021ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 17.9 5.977 1.846 0.143 Residuals 106 343.2 3.238	> **ac1021d<- aov(depth ~ trt, data=wf1021ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00234 0.0007805 2.088 0.106 Residuals 106 0.03963 0.0003738
	1227	> **ac1227a<- aov(area ~ trt, data=wf1227ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 2626 875.4 2.881 0.0392 Residuals 110 33428 303.9 > **TukeyHSD(ac1227a)** diff lwr upr p adj p-c 5.538931 -7.111542 18.18940 0.6642903 ws-c -7.373654 -19.653788 4.90648 0.4020048 wsp-c 1.219767 -11.234563 13.67410 0.9941180 ws-p -12.912585 -24.680820 -1.14435 0.0255553 wsp-p -4.319164 -16.269058 7.63073 0.7817990 wsp-ws 8.593421 -2.963708 20.15055 0.2176283	> **ac1227d<- aov(depth ~ trt, data=wf1227ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0513 0.017112 2.68 0.0505 Residuals 110 0.7024 0.006385

27/09/12	1614	> **ac1614a<- aov(area ~ trt, data=wf1614ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 15.05 5.018 8.452 2.41e-05 Residuals 224 133.00 0.594 > **TukeyHSD(ac1614a)** diff lwr upr p adj p-c 0.3907146 -0.001601766 0.78303088 0.0513857 ws-c 0.1381426 -0.242688770 0.51897403 0.7839213 wsp-c -0.3137305 -0.699964080 0.07250298 0.1554414 ws-p -0.2525719 -0.617528312 0.11238445 0.2801872 wsp-p -0.7044451 -1.075035106 -0.33385510 0.0000099 wsp-ws -0.4518732 -0.810282725 -0.09346363 0.0069284	> **ac1614d<- aov(depth ~ trt, data=wf1614ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.001253 0.0004177 7.923 4.8e-05 Residuals 224 0.011808 0.0000527 > **TukeyHSD(ac1614d)** diff lwr upr p adj p-c 4.536615e-03 0.0008400765 0.008233152 0.0091540 ws-c -9.179921e-05 -0.0036801223 0.003496524 0.9998955 wsp-c -1.771129e-03 -0.0054103530 0.001868095 0.5895377 ws-p -4.628414e-03 -0.0080671570 -0.001189670 0.0033086 wsp-p -6.307744e-03 -0.0097995688 -0.002815918 0.0000299 wsp-ws -1.679330e-03 -0.0050563867 0.001697727 0.5720621
	1797	> **ac1797a<- aov(area ~ trt, data=wf1797ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 4169 1389.8 2.101 0.104 Residuals 110 72768 661.5	> **ac1797d<- aov(depth ~ trt, data=wf1797ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0309 0.010290 1.53 0.211 Residuals 110 0.7397 0.006724
13/09/12	1021	> **ac1021a<- aov(area ~ trt, data=wf1021ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.77 0.2555 0.097 0.961 Residuals 104 273.20 2.6269	> **ac1021d<- aov(depth ~ trt, data=wf1021ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000051 1.692e-05 0.058 0.981 Residuals 104 0.030240 2.908e-04
	1227	> **ac1227a<- aov(area ~ trt, data=wf1227ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 552 183.9 0.949 0.42 Residuals 104 20145 193.7	> **ac1227d<- aov(depth ~ trt, data=wf1227ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0153 0.005107 1.353 0.261 Residuals 104 0.3925 0.003774
	1614	> **ac1614a<- aov(area ~ trt, data=wf1614ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 10.16 3.387 1.116 0.346 Residuals 104 315.71 3.036	> **ac1614d<- aov(depth ~ trt, data=wf1614ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00262 0.0008750 2.296 0.0821 . Residuals 104 0.03963 0.0003811
	1797	> **ac1797a<- aov(area ~ trt, data=wf1797ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 2434 811.4 2.311 0.0805 . Residuals 104 36518 351.1	> **ac1797d<- aov(depth ~ trt, data=wf1797ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0236 0.007878 1.839 0.145 Residuals 104 0.4455 0.004283
27/09/12	1021	> **ac1021a<- aov(area ~ trt, data=wf1021ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 418.9 139.62 20.32 1.94e-10 Residuals 104 714.7 6.87 > **TukeyHSD(ac1021a)** diff lwr upr p adj p-c 2.62787791 0.6903225 4.5654333 0.0033001 ws-c -2.13607644 -4.0401269 -0.2320259 0.0214079 wsp-c -2.16944995 -4.0439785 -0.2949214 0.0164483 ws-p -4.76395435 -6.6281548 -2.8997539 0.0000000 wsp-p -4.79732787 -6.6313651 -2.9632907 0.0000000 wsp-ws -0.03337351 -1.8319784 1.7652314 0.9999589	> **ac1021d<- aov(depth ~ trt, data=wf1021ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.04768 0.015895 18.57 1.01e-09 Residuals 104 0.08902 0.000856 > **TukeyHSD(ac1021d)** diff lwr upr p adj p-c 0.0273575467 0.005733742 0.048981352 0.0070695 ws-c -0.0234905180 -0.044740396 -0.002240640 0.0241250 wsp-c -0.0236300753 -0.044550478 -0.002709672 0.0202355 ws-p -0.0508480647 -0.071653203 -0.030042927 0.0000000 wsp-p -0.0509876221 -0.071456127 -0.030519117 0.0000000 wsp-ws -0.0001395574 -0.020212626 0.019933511 0.9999978
	1227	> **ac1227a<- aov(area ~ trt, data=wf1227ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 52712 17571 18.76 8.43e-10 Residuals 104 97408 937 > **TukeyHSD(ac1227a)** diff lwr upr p adj p-c 13.89698 -8.722918 36.51688 0.3807959 ws-c -32.33057 -54.559314 -10.10182 0.0013871 wsp-c -38.99490 -60.878992 -17.11080 0.0000567 ws-p -46.22755 -67.991070 -24.46403 0.0000013 wsp-p -52.89188 -74.303260 -31.48050 0.0000000 wsp-ws -6.66433 -27.662060 14.33340 0.8407291	> **ac1227d<- aov(depth ~ trt, data=wf1227ac)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.091 0.3637 17.57 2.65e-09 Residuals 104 2.153 0.0207 > **TukeyHSD(ac1227d)** diff lwr upr p adj p-c 0.04616701 -0.06016715 0.15250116 0.6696592 ws-c -0.15234365 -0.25683904 -0.04784827 0.0013444 wsp-c -0.19218077 -0.29505597 -0.08930556 0.0000228 ws-p -0.19851066 -0.30081906 -0.09620227 0.0000104 wsp-p -0.23834778 -0.33900079 -0.13769476 0.0000001 wsp-ws -0.03983711 -0.13854558 0.05887136 0.7182102

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1614

```
> ac1614a<- aov(area ~ trt, data=wf1614ac)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3  13.55   4.518     1.8  0.152
Residuals   108 271.11   2.510
```

```
> ac1614d<- aov(depth ~ trt, data=wf1614ac)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.002071 0.0006903   3.444 0.0193
Residuals   108 0.021648 0.0002004
> TukeyHSD(ac1614d)
                diff          lwr          upr     p adj
p-c     0.006236862 -0.004040096  0.016513820 0.3922270
ws-c   -0.005386359 -0.015503975  0.004731257 0.5088177
wsp-c  -0.002114730 -0.012232345  0.008002886 0.9475946
ws-p   -0.011623221 -0.021331045 -0.001915396 0.0120869
wsp-p  -0.008351591 -0.018059415  0.001356233 0.1178317
wsp-ws  0.003271629 -0.006267350  0.012810609 0.8074576
```

1797

```
> ac1797a<- aov(area ~ trt, data=wf1797ac)
> summary(ac1797a)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 104799   34933   17.49 2.89e-09
Residuals   104 207757    1998
> TukeyHSD(ac1797a)
                diff        lwr       upr     p adj
p-c        7.629936  -25.40474  40.66461 0.9308821
ws-c     -46.978340  -79.44177 -14.51491 0.0014823
wsp-c    -65.830301  -97.79039 -33.87021 0.0000028
ws-p     -54.608276  -86.39227 -22.82428 0.0001094
wsp-p    -73.460237 -104.72996 -42.19051 0.0000001
wsp-ws   -18.851962  -49.51758  11.81366 0.3802259
```

```
> ac1797d<- aov(depth ~ trt, data=wf1797ac)
> summary(ac1797d)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.8848 0.29492   14.76 4.45e-08
Residuals   104 2.0777 0.01998
> TukeyHSD(ac1797d)
                diff         lwr         upr     p adj
p-c      0.01757771 -0.08688987  0.12204528 0.9715060
ws-c    -0.12944282 -0.23210390 -0.02678174 0.0073195
wsp-c   -0.19792094 -0.29899029 -0.09685160 0.0000086
ws-p    -0.14702053 -0.24753301 -0.04650805 0.0012869
wsp-p   -0.21549865 -0.31438481 -0.11661249 0.0000007
wsp-ws  -0.06847812 -0.16545388  0.02849763 0.2589762
```

1021

```
ac1021a<- aov(area ~ trt, data=wf1021ac)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  232.6   77.52   12.86 5.13e-07
Residuals    86  518.6    6.03
> TukeyHSD(ac1021a)
                diff        lwr        upr     p adj
p-c     -0.4608997 -2.282117  1.3603180 0.9106454
ws-c    -3.9020936 -5.801120 -2.0030667 0.0000037
wsp-c   -2.6761952 -4.682292 -0.6700983 0.0041140
ws-p    -3.4411938 -5.304961 -1.5774262 0.0000335
wsp-p   -2.2152955 -4.188048 -0.2425432 0.0214008
wsp-ws   1.2258983 -0.818905  3.2707017 0.4006568
```

```
> ac1021d<- aov(depth ~ trt, data=wf1021ac)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.02912 0.009706   13.73 2.13e-07
Residuals    86 0.06077 0.000707
> TukeyHSD(ac1021d)
                diff          lwr          upr     p adj
p-c     -0.004062932 -0.023778249  0.015652385 0.9489962
ws-c    -0.043392353 -0.063949982 -0.022834724 0.0000020
wsp-c   -0.028864846 -0.050581546 -0.007148146 0.0042845
ws-p    -0.039329421 -0.059505356 -0.019153486 0.0000115
wsp-p   -0.024801914 -0.046157646 -0.003446181 0.0161283
wsp-ws   0.014527507 -0.007608203  0.036663218 0.3199842
```

1227

```
> ac1227a<- aov(area ~ trt, data=wf1227ac)
> summary(ac1227a)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  39186   13062    17.1 8.53e-09
Residuals    86  65692     764
> TukeyHSD(ac1227a)
                diff         lwr         upr     p adj
p-c       0.3553959 -20.141907  20.852699 0.9999661
ws-c    -49.4588396 -70.831863 -28.085816 0.0000002
wsp-c   -25.3018472 -47.879915  -2.723779 0.0217619
ws-p    -49.8142355 -70.790426 -28.838045 0.0000001
wsp-p   -25.6572431 -47.860026  -3.454460 0.0168385
wsp-ws   24.1569924   1.143294  47.170691 0.0358658
```

```
> ac1227d<- aov(depth ~ trt, data=wf1227ac)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.8736 0.29120   16.79 1.13e-08
Residuals    86 1.4912 0.01734
> TukeyHSD(ac1227d)

                diff         lwr          upr     p adj
p-c      0.005805391 -0.09185199  0.103462773 0.9986440
ws-c    -0.232673732 -0.33450340 -0.130844065 0.0000003
wsp-c   -0.110224547 -0.21779553 -0.002653564 0.0424743
ws-p    -0.238479123 -0.33841812 -0.138540130 0.0000001
wsp-p   -0.116029938 -0.22181291 -0.010246963 0.0258093
wsp-ws   0.122449185  0.01280269  0.232095684 0.0223804
```

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1614

```
> ac1614a<- aov(area ~ trt, data=wf1614ac)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   7.27   2.423   0.862  0.464
Residuals    90 252.87   2.810
> TukeyHSD(ac1614a)
               diff        lwr       upr     p adj
p-c     0.67125877 -0.5493211 1.8918386 0.4782699
ws-c    0.57810451 -0.6885510 1.8447601 0.6317607
wsp-c   0.21455253 -1.1535918 1.5826969 0.9765189
ws-p   -0.09315425 -1.3137341 1.1274256 0.9971565
wsp-p  -0.45670623 -1.7823072 0.8688947 0.8038556
wsp-ws -0.36355198 -1.7316963 1.0045924 0.8985260
```

```
> ac1614d<- aov(depth ~ trt, data=wf1614ac)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00310 0.0010334   3.773 0.0133
Residuals    90 0.02465 0.0002739
> TukeyHSD(ac1614d)
                diff          lwr          upr     p adj
p-c    -0.005368482 -0.017419503  0.006682540 0.6496505
ws-c   -0.015657417 -0.028163353 -0.003151482 0.0079816
wsp-c  -0.008800336 -0.022308291  0.004707618 0.3269272
ws-p   -0.010288936 -0.022339957  0.001762085 0.1217051
wsp-p  -0.003431855 -0.016519769  0.009656060 0.9020353
wsp-ws  0.006857081 -0.006650873  0.020365036 0.5471691
```

1797

```
> ac1797a<- aov(area ~ trt, data=wf1797ac)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  88376   29459   17.03 9.09e-09
Residuals    86 148753    1730
> TukeyHSD(ac1797a)
                diff         lwr        upr     p adj
p-c       1.023938  -29.820322  31.868199 0.9997623
ws-c    -74.349661 -106.511702 -42.187619 0.0000002
wsp-c   -36.028311  -70.003699  -2.052923 0.0333146
ws-p    -75.373599 -106.938487 -43.808710 0.0000001
wsp-p   -37.052250  -70.462910  -3.641589 0.0236677
wsp-ws  38.321349    3.690427  72.952271 0.0240837
```

```
> ac1797d<- aov(depth ~ trt, data=wf1797ac)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.6446 0.21486   14.59 9.19e-08
Residuals    86 1.2665 0.01473
> TukeyHSD(ac1797d)
                diff         lwr         upr     p adj
p-c     0.004417659 -0.08558312  0.094418436 0.9992344
ws-c   -0.201642912 -0.29548886 -0.107796968 0.0000013
wsp-c  -0.084886442 -0.18402357  0.014250684 0.1198834
ws-p   -0.206060571 -0.29816408 -0.113957066 0.0000005
wsp-p  -0.089304102 -0.18679340  0.008185201 0.0846884
wsp-ws  0.116756469  0.01570655  0.217806385 0.0168577
```

1021

```
> ac1021a<- aov(area ~ trt, data=wf1021ac)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3  32.75  10.916    3.15  0.032
Residuals    56 194.08   3.466
> TukeyHSD(ac1021a)
               diff        lwr       upr     p adj
p-c     0.4156693 -0.9797069  1.8110455 0.8592412
ws-c   -1.7854190 -3.7978592  0.2270211 0.0992133
wsp-c  -1.2979067 -4.9258848  2.3300714 0.7795261
ws-p   -2.2010883 -4.1940842 -0.2080925 0.0249216
wsp-p  -1.7135760 -5.3308045  1.9036525 0.5953967
wsp-ws  0.4875123 -3.4095613  4.3845859 0.9873302
```

```
> ac1021d<- aov(depth ~ trt, data=wf1021ac)
             Df   Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.004211 0.001404    4.14 0.0101
Residuals    56 0.018986 0.000339
> TukeyHSD(ac1021d)
                diff         lwr          upr     p adj
p-c     0.004395941 -0.009405281  0.018197162 0.8334982
ws-c   -0.020482701 -0.040387106 -0.000578296 0.0414291
wsp-c  -0.015146230 -0.051029405  0.020736946 0.6802649
ws-p   -0.024878642 -0.044590729 -0.005166555 0.0078830
wsp-p  -0.019542170 -0.055319025  0.016234685 0.4764138
wsp-ws  0.005336472 -0.033208242  0.043881185 0.9829895
```

1227

```
> ac1227a<- aov(area ~ trt, data=wf1227ac)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   1892   630.6   3.798  0.015
Residuals    56   9298   166.0
> TukeyHSD(ac1227a)
               diff        lwr       upr     p adj
p-c       4.712447  -4.945569 14.370463 0.5718202
ws-c    -12.839023 -26.768012  1.089966 0.0810612
wsp-c     1.441534 -23.669308 26.552375 0.9987333
ws-p    -17.551470 -31.345876 -3.757064 0.0072867
wsp-p    -3.270913 -28.307352 21.765526 0.9856242
wsp-ws   14.280557 -12.692814 41.253927 0.5035144
```

```
> ac1227d<- aov(depth ~ trt, data=wf1227ac)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.03003 0.010011   3.281 0.0274
Residuals    56 0.17084 0.003051
> TukeyHSD(ac1227d)
                diff         lwr         upr     p adj
p-c     0.025439300 -0.01596035  0.066838953 0.3720266
ws-c   -0.042565974 -0.10227340  0.017141454 0.2449524
wsp-c   0.021406695 -0.08623240  0.129045791 0.9522941
ws-p   -0.068005275 -0.12713581 -0.008874744 0.0180652
wsp-p  -0.004032606 -0.11135277  0.103287559 0.9996429
wsp-ws  0.063972669 -0.05165027  0.179595605 0.4651491
```

1614

```
> ac1614a<- aov(area ~ trt, data=wf1614ac)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   4.13   1.378   0.686  0.564
Residuals    58 116.44   2.007
```

```
> ac1614d<- aov(depth ~ trt, data=wf1614ac)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00075 0.0002500   1.341   0.27
Residuals    58 0.01081 0.0001864
```

	1797	> ac1797a<- aov(area ~ trt, data=wf1797ac) Df Sum Sq Mean Sq F value Pr(>F) trt 3 5710 1903.5 3.283 0.0273 Residuals 56 32468 579.8 > TukeyHSD(ac1797a) diff lwr upr p adj p-c 15.483938 -2.563973 33.5318486 0.1170591 ws-c -10.111906 -36.140973 15.9171615 0.7334465 wsp-c 20.684509 -26.240059 67.6090770 0.6498044 ws-p -25.595843 -51.373416 0.1817291 0.0522982 wsp-p 5.200571 -41.584961 51.9861030 0.9910305 wsp-ws 30.796415 -19.608657 81.2014862 0.3771039	> ac1797d<- aov(depth ~ trt, data=wf1797ac) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0398 0.01327 2.279 0.0893 Residuals 56 0.3259 0.00582

Banksia serrata

		Area	Depth
	1021	> bs1021a<- aov(area ~ trt, data=wf1021bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 26.98 8.994 2.086 0.112 Residuals 60 258.69 4.311	> bs1021d<- aov(depth ~ trt, data=wf1021bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.002809 0.0009364 2.266 0.0899 Residuals 60 0.024788 0.0004131
	1227	> bs1227a<- aov(area ~ trt, data=wf1227bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 2525 841.7 3.489 0.021 Residuals 60 14474 241.2 > TukeyHSD(bs1227a) diff lwr upr p adj p-c 2.8518383 -10.18910 15.892776 0.9383380 ws-c 0.8687864 -12.17215 13.909724 0.9980400 wsp-c -20.1741570 -38.76262 -1.585692 0.0283476 ws-p -1.9830519 -16.49367 12.527564 0.9837251 wsp-p -23.0259953 -42.67346 -3.378529 0.0153308 wsp-ws -21.0429434 -40.69041 -1.395477 0.0312564	> bs1227d<- aov(depth ~ trt, data=wf1227bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.05216 0.017386 3.978 0.0119 Residuals 60 0.26225 0.004371 > TukeyHSD(bs1227d) diff lwr upr p adj p-c 1.446308e-02 -0.04104790 0.069974060 0.9010964 ws-c 7.353346e-05 -0.05543745 0.055584515 1.0000000 wsp-c -9.163731e-02 -0.17076229 -0.012512326 0.0169518 ws-p -1.438955e-02 -0.07615646 0.047377373 0.9267157 wsp-p -1.061004e-01 -0.18973319 -0.022467582 0.0074070 wsp-ws -9.171084e-02 -0.17534364 -0.008078037 0.0262223
	1614	> bs1614a<- aov(area ~ trt, data=wf1614bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.61 0.2041 0.112 0.953 Residuals 60 109.29 1.8214	> bs1614d<- aov(depth ~ trt, data=wf1614bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00027 0.0000910 0.104 0.958 Residuals 60 0.05264 0.0008774
28/06/12	1797	> bs1797a<- aov(area ~ trt, data=wf1797bs) > summary(bs1797a) Df Sum Sq Mean Sq F value Pr(>F) trt 3 5867 1955.7 3.521 0.0203 Residuals 60 33331 555.5 > TukeyHSD(bs1797a) diff lwr upr p adj p-c 3.199289 -16.59059 22.989165 0.9735980 ws-c -3.986511 -23.77639 15.803365 0.9508635 wsp-c -31.881750 -60.09011 -3.673394 0.0207078 ws-p -7.185800 -29.20594 14.834341 0.8241373 wsp-p -35.081039 -64.89645 -5.265627 0.0148181 wsp-ws -27.895239 -57.71065 1.920173 0.0747197	> bs1797d<- aov(depth ~ trt, data=wf1797bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.02025 0.006751 1.365 0.262 Residuals 60 0.29682 0.004947

17/07/12	1021	> **wf1021bsarea<- aov(area ~ trt, data=wf1021bs)** > summary(wf1021bsarea) Df Sum Sq Mean Sq F value Pr(>F) trt 3 53.31 17.770 9.687 1.14e-05 Residuals 100 183.45 1.834 > **TukeyHSD(wf1021bsarea)** diff lwr upr p adj p-c 1.9594798 0.9779928 2.94096683 0.0000059 ws-c 1.0648701 0.0833831 2.04635714 0.0279681 wsp-c 1.4176243 0.4361373 2.39911131 0.0015337 ws-p -0.8946097 -1.8760967 0.08687734 0.0872719 wsp-p -0.5418555 -1.5233425 0.43963150 0.4761583 wsp-ws 0.3527542 -0.6287329 1.33424118 0.7839202	**> wf1021bsdepth<- aov(depth ~ trt, data=wf1021bs)** > summary(wf1021bsdepth) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.005199 0.0017330 8.713 3.44e-05 Residuals 100 0.019891 0.0001989 > **TukeyHSD(wf1021epdepth)** diff lwr upr p adj p-c -0.0041974250 -0.02097330 0.01257845 0.9136465 ws-c -0.0032329285 -0.02000881 0.01354295 0.9578774 wsp-c -0.0027924580 -0.02025335 0.01466843 0.9751920 ws-p 0.0009644965 -0.01509718 0.01702618 0.9986087 wsp-p 0.0014049670 -0.01537091 0.01818085 0.9962595 wsp-ws 0.0004404705 -0.01633541 0.01721635 0.9998828
	1227	> **wf1227bsa<- aov(area ~ trt, data=wf1227bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 527 175.67 4.23 0.00737 Residuals 100 4153 41.53 **> TukeyHSD(wf1227bsa)** diff lwr upr p adj p-c 6.038377 1.368301 10.708453 0.0056544 ws-c 2.379482 -2.290594 7.049558 0.5453752 wsp-c 4.340558 -0.329518 9.010634 0.0782693 ws-p -3.658895 -8.328972 1.011181 0.1779606 wsp-p -1.697819 -6.367895 2.972257 0.7779765 wsp-ws 1.961076 -2.709000 6.631152 0.6920896	> **wf1227bsdepth<- aov(depth ~ trt, data=wf1227bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00398 0.0013265 2.733 0.0477 Residuals 100 0.04853 0.0004853 > **TukeyHSD(wf1227bsdepth)** diff lwr upr p adj p-c 0.014013230 -0.001951174 0.029977635 0.1064389 ws-c 0.001897262 -0.014067143 0.017861666 0.9895609 wsp-c 0.012371730 -0.003592674 0.028336135 0.1858428 ws-p -0.012115969 -0.028080373 0.003848435 0.2013369 wsp-p -0.001641500 -0.017605904 0.014322904 0.9931760 wsp-ws 0.010474469 -0.005489935 0.026438873 0.3217483
	1614	> **wf1614bsarea<- aov(area ~ trt, data=wf1614bs)** **> summary(wf1614bsarea)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.802 0.6008 2.54 0.0607 Residuals 100 23.654 0.2365	> **wf1614bsd<- aov(depth ~ trt, data=wf1614bs)** > summary(wf1614bsd) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0001879 6.262e-05 2.794 0.0442 Residuals 100 0.0022410 2.241e-05 **> TukeyHSD(wf1614bsd)** diff lwr upr p adj p-c 0.0037072508 0.0002768454 0.007137656 0.0288233 ws-c 0.0025463494 -0.0008840559 0.005976755 0.2183752 wsp-c 0.0022958609 -0.0011345444 0.005726266 0.3043669 ws-p -0.0011609013 -0.0045913067 0.002269504 0.8130549 wsp-p -0.0014113898 -0.0048417952 0.002019015 0.7055283 wsp-ws -0.0002504885 -0.0036808938 0.003179917 0.9975222
	1797	**> wf1797bsarea<- aov(area ~ trt, data=wf1797bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 110 36.78 0.519 0.67 Residuals 100 7088 70.88	**> wf1797bsdepth<- aov(depth ~ trt, data=wf1797bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00217 0.0007242 0.801 0.496 Residuals 100 0.09038 0.0009038
16/08/12	1021	> **bs1021a<- aov(area ~ trt, data=wf1021bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.83 1.610 1.188 0.318 Residuals 100 135.50 1.355	> **bs1021d<- aov(depth ~ trt, data=wf1021bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000448 0.0001492 1.065 0.367 Residuals 100 0.014007 0.0001401
	1227	> **bs1227a<- aov(area ~ trt, data=wf1227bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 290 96.68 1.691 0.174 Residuals 100 5718 57.18	> **bs1227d<- aov(depth ~ trt, data=wf1227bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00423 0.0014111 1.711 0.17 Residuals 100 0.08249 0.0008249

30/08/12	1614	> **bs1614a<- aov(area ~ trt, data=wf1614bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 2.483 0.8277 2.978 0.0351 Residuals 100 27.792 0.2779 > **TukeyHSD(bs1614a)** diff lwr upr p adj p-c 0.38847625 -0.00141954 0.77837204 0.0512095 ws-c 0.05111007 -0.34789502 0.45011517 0.9870063 wsp-c 0.26523592 -0.09843466 0.62890650 0.2323013 ws-p -0.33736618 -0.74391976 0.06918741 0.1393158 wsp-p -0.12324033 -0.49517721 0.24869655 0.8224748 wsp-ws 0.21412584 -0.16734942 0.59560111 0.4614614	> **bs1614d<- aov(depth ~ trt, data=wf1614bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000481 1.604e-04 4.118 0.00846 Residuals 100 0.003896 3.896e-05 > **TukeyHSD(bs1614d)** diff lwr upr p adj p-c 0.0044265334 -0.0001897016 0.0090427684 0.0651647 ws-c -0.0001319794 -0.0048560655 0.0045921067 0.9998596 wsp-c 0.0041138133 -0.0001919240 0.0084195507 0.0666097 ws-p -0.0045585128 -0.0093719705 0.0002549449 0.0702050 wsp-p -0.0003127201 -0.0047163276 0.0040908874 0.9977191 wsp-ws 0.0042457927 -0.0002707461 0.0087623316 0.0733177
	1797	> **bs1797a<- aov(area ~ trt, data=wf1797bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1092 364.2 2.249 0.0872 Residuals 100 16190 161.9	> **bs1797d<- aov(depth ~ trt, data=wf1797bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01805 0.006016 2.95 0.0364 Residuals 100 0.20393 0.002039 > **TukeyHSD(bs1797d)** diff lwr upr p adj p-c 0.010172583 -0.0232263736 0.04357154 0.8561946 ws-c -0.004387693 -0.0385669645 0.02979158 0.9869236 wsp-c 0.028451942 -0.0027005313 0.05960442 0.0863182 ws-p -0.014560276 -0.0493861607 0.02026561 0.6950084 wsp-p 0.018279359 -0.0135812159 0.05013993 0.4419352 wsp-ws 0.032839635 0.0001619901 0.06551728 0.0483925
	1021	> **bs1021a<- aov(area ~ trt, data=wf1021bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 37.21 12.404 5.821 0.00105 Residuals 99 210.96 2.131 > **TukeyHSD(bs1021a)** diff lwr upr p adj p-c 0.7489718 -0.3195538 1.8174974 0.2647607 ws-c 0.2267530 -0.8417726 1.2952785 0.9451058 wsp-c 1.5645409 0.4960154 2.6330665 0.0012856 ws-p -0.5222188 -1.5802168 0.5357791 0.5715356 wsp-p 0.8155691 -0.2424288 1.8735671 0.1896651 wsp-ws 1.3377880 0.2797900 2.3957859 0.0071473	> **bs1021d<- aov(depth ~ trt, data=wf1021bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.003074 0.0010248 5.461 0.00163 Residuals 99 0.018579 0.0001877 > **TukeyHSD(bs1021d)** diff lwr upr p adj p-c 0.008608015 -0.001419525 0.01863555 0.1188425 ws-c 0.007275141 -0.002752399 0.01730268 0.2364395 wsp-c 0.015472808 0.005445269 0.02550035 0.0006208 ws-p -0.001332874 -0.011261618 0.00859587 0.9850979 wsp-p 0.006864794 -0.003063950 0.01679354 0.2763025 wsp-ws 0.008197667 -0.001731076 0.01812641 0.1424694
	1227	> **bs1227a<- aov(area ~ trt, data=wf1227bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1116 372.0 1.647 0.183 Residuals 99 22353 225.8	> **bs1227d<- aov(depth ~ trt, data=wf1227bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0155 0.005158 1.207 0.311 Residuals 99 0.4229 0.004272
	1614	> **bs1614a<- aov(area ~ trt, data=wf1614bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 26.6 8.855 2.44 0.0655 Residuals 202 733.0 3.629	> **bs1614d<- aov(depth ~ trt, data=wf1614bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00604 0.0020128 2.811 0.0406 Residuals 202 0.14466 0.0007161 > **TukeyHSD(bs1614d)** diff lwr upr p adj p-c 0.012297410 -0.0014336486 0.026028469 0.0968654 ws-c 0.004552944 -0.0091781147 0.018284003 0.8259145 wsp-c 0.013008193 -0.0007228656 0.026739252 0.0704592 ws-p -0.007744466 -0.0213402402 0.005851308 0.4541275 wsp-p 0.000710783 -0.0128849911 0.014306557 0.9991107 wsp-ws 0.008455249 -0.0051405249 0.022051023 0.3746885
	1797	> **bs1797a<- aov(area ~ trt, data=wf1797bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 3125 1041.7 1.561 0.204 Residuals 99 66064 667.3	> **bs1797d<- aov(depth ~ trt, data=wf1797bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0454 0.015150 1.559 0.204 Residuals 99 0.9621 0.009718

13/09/12	1021	> bs1021a<- aov(area ~ trt, data=wf1021bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 6.9 2.299 1.017 0.389 Residuals 100 226.2 2.262	> bs1021d<- aov(depth ~ trt, data=wf1021bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000506 0.0001686 0.955 0.417 Residuals 100 0.017647 0.0001765
	1227	> bs1227a<- aov(area ~ trt, data=wf1227bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 457 152.3 1.074 0.364 Residuals 100 14176 141.8	> bs1227d<- aov(depth ~ trt, data=wf1227bs) > summary(bs1227d) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0081 0.002687 0.737 0.532 Residuals 100 0.3647 0.003647
	1614	> bs1614a<- aov(area ~ trt, data=wf1614bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.67 1.558 0.522 0.668 Residuals 100 298.53 2.985	> bs1614d<- aov(depth ~ trt, data=wf1614bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0050 0.001653 0.379 0.769 Residuals 100 0.4365 0.004365
	1797	> bs1797a<- aov(area ~ trt, data=wf1797bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 504 167.9 0.346 0.792 Residuals 100 48513 485.1	> bs1797d<- aov(depth ~ trt, data=wf1797bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0121 0.004046 1.034 0.381 Residuals 100 0.3915 0.003915
27/09/12	1021	> bs1021a<- aov(area ~ trt, data=wf1021bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 67.6 22.528 4.857 0.00339 Residuals 100 463.8 4.638 > TukeyHSD(bs1021a) diff lwr upr p adj p-c 1.4171289 -0.1434303 2.9776881 0.0890648 ws-c -0.8364393 -2.3969986 0.7241199 0.5020798 wsp-c 0.2651401 -1.2954192 1.8256993 0.9706459 ws-p -2.2535682 -3.8141275 -0.6930090 0.0015377 wsp-p -1.1519888 -2.7125481 0.4085704 0.2227169 wsp-ws 1.1015794 -0.4589798 2.6621386 0.2589705	> bs1021d<- aov(depth ~ trt, data=wf1021bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00527 0.0017550 3.509 0.0181 Residuals 100 0.05002 0.0005002 > TukeyHSD(bs1021d) diff lwr upr p adj p-c 0.011814322 -0.004391870 0.028020513 0.2326632 ws-c -0.008113977 -0.024320168 0.008092215 0.5599591 wsp-c -0.000250491 -0.016456682 0.015955700 0.9999762 ws-p -0.019928298 -0.036134490 -0.003722107 0.0094319 wsp-p -0.012064813 -0.028271004 0.004141379 0.2161012 wsp-ws 0.007863486 -0.008342706 0.024069677 0.5855024
	1227	> bs1227a<- aov(area ~ trt, data=wf1227bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 7330 2443.4 5.293 0.00199 Residuals 100 46158 461.6 > TukeyHSD(bs1227a) diff lwr upr p adj p-c 3.372276 -12.19641 18.940961 0.9419349 ws-c -18.639436 -34.20812 -3.070750 0.0121568 wsp-c -6.136230 -21.70492 9.432456 0.7324194 ws-p -22.011712 -37.58040 -6.443026 0.0020130 wsp-p -9.508506 -25.07719 6.060180 0.3857148 wsp-ws 12.503206 -3.06548 28.071892 0.1607352	> bs1227d<- aov(depth ~ trt, data=wf1227bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1434 0.04779 4.685 0.00419 Residuals 100 1.0200 0.01020 > TukeyHSD(bs1227d) diff lwr upr p adj p-c 0.0009057581 -0.0722813 0.07409282 0.9999878 ws-c -0.0902128031 -0.1633999 -0.01702574 0.0092132 wsp-c -0.0364204396 -0.1096075 0.03676662 0.5649731 ws-p -0.0911185612 -0.1643056 -0.01793150 0.0083487 wsp-p -0.0373261977 -0.1105133 0.03586086 0.5445617 wsp-ws 0.0537923635 -0.0193947 0.12697942 0.2261381
	1614	> bs1614a<- aov(area ~ trt, data=wf1614bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 9.52 3.174 1.43 0.239 Residuals 100 221.99 2.220	> bs1614d<- aov(depth ~ trt, data=wf1614bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00326 0.0010882 1.611 0.192 Residuals 100 0.06755 0.0006755
	1797	> bs1797a<- aov(area ~ trt, data=wf1797bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 10174 3391 2.902 0.0386 Residuals 100 116844 1168 > TukeyHSD(bs1797a) diff lwr upr p adj p-c -1.442416 -26.21276 23.32792379 0.9987369 ws-c -24.706777 -49.47712 0.06356294 0.0508499 wsp-c -11.712016 -36.48236 13.05832402 0.6059776 ws-p -23.264361 -48.03470 1.50597871 0.0737054 wsp-p -10.269600 -35.03994 14.50073979 0.7005467 wsp-ws 12.994761 -11.77558 37.76510064 0.5206080	> bs1797d<- aov(depth ~ trt, data=wf1797bs) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0665 0.02218 1.918 0.131 Residuals 100 1.1563 0.01156

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1021

```
> bs1021a<- aov(area ~ trt, data=wf1021bs)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  236.6   78.85   16.28 1.83e-08
Residuals    86  416.6    4.84
> TukeyHSD(bs1021a)
               diff        lwr        upr     p adj
p-c    -0.5256180 -2.124998  1.0737625 0.8248285
ws-c   -3.2696821 -5.037864 -1.5015004 0.0000326
wsp-c  -3.7200698 -5.435214 -2.0049253 0.0000011
ws-p   -2.7440641 -4.512246 -0.9758825 0.0006018
wsp-p  -3.1944518 -4.909596 -1.4793073 0.0000284
wsp-ws -0.4503877 -2.323932  1.4231568 0.9221771
```

```
> bs1021d<- aov(depth ~ trt, data=wf1021bs)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.02540 0.008467   16.05 2.27e-08
Residuals    86 0.04537 0.000528
> TukeyHSD(bs1021d)
                diff         lwr         upr     p adj
p-c    -0.003961550 -0.02065106  0.01272796 0.9248004
ws-c   -0.032658790 -0.05110974 -0.01420784 0.0000729
wsp-c  -0.038342765 -0.05624027 -0.02044526 0.0000014
ws-p   -0.028697240 -0.04714819 -0.01024629 0.0005830
wsp-p  -0.034381216 -0.05227872 -0.01648371 0.0000154
wsp-ws -0.005683976 -0.02523438  0.01386643 0.8713829
```

1227

```
> bs1227a<- aov(area ~ trt, data=wf1227bs)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3  35313   11771   18.32 2.8e-09
Residuals    86  55253     642
> TukeyHSD(bs1227a)
               diff        lwr       upr     p adj
p-c       1.049985 -17.36863  19.46860 0.9988033
ws-c    -37.975079 -58.33762 -17.61254 0.0000277
wsp-c   -40.920112 -60.67187 -21.16835 0.0000031
ws-p    -39.025064 -59.38760 -18.66252 0.0000162
wsp-p   -41.970097 -61.72186 -22.21834 0.0000017
wsp-ws   -2.945033 -24.52094  18.63088 0.9842276
```

```
> bs1227d<- aov(depth ~ trt, data=wf1227bs)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.7271 0.24237   17.91 4.07e-09
Residuals    86 1.1640 0.01353
> TukeyHSD(bs1227d)
                diff         lwr         upr     p adj
p-c       0.006924532 -0.07761229  0.09146135 0.9964824
ws-c    -0.173967710 -0.26742668 -0.08050874 0.0000287
wsp-c   -0.182312391 -0.27296803 -0.09165675 0.0000059
ws-p    -0.180892241 -0.27435121 -0.08743327 0.0000132
wsp-p   -0.189236922 -0.27989256 -0.09858129 0.0000026
wsp-ws  -0.008344681 -0.10737271  0.09068334 0.9961742
```

1614

```
> bs1614a<- aov(area ~ trt, data=wf1614bs)
> summary(bs1614a)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   24.1   8.046    1.63  0.188
Residuals    86  424.5   4.936
```

```
> bs1614d<- aov(depth ~ trt, data=wf1614bs)
> summary(bs1614d)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00208 0.0006947    1.55  0.208
Residuals    86 0.03855 0.0004483
```

1797

```
> bs1797a<- aov(area ~ trt, data=wf1797bs)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  72768   24256   14.68 8.42e-08
Residuals    86 142105    1652
> TukeyHSD(bs1797a)
                diff       lwr       upr     p adj
p-c       1.5032858 -28.03482  31.04139 0.9991470
ws-c    -56.5301744 -89.18578 -23.87457 0.0001076
wsp-c   -57.0542469 -88.73033 -25.37816 0.0000532
ws-p    -58.0334602 -90.68906 -25.37786 0.0000679
wsp-p   -58.5575327 -90.23362 -26.88145 0.0000328
wsp-ws   -0.5240725 -35.12557  34.07742 0.9999774
```

```
> bs1797d<- aov(depth ~ trt, data=wf1797bs)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.3512 0.11708   10.52 5.78e-06
Residuals    86 0.9569 0.01113
> TukeyHSD(bs1797d)
                diff         lwr         upr     p adj
p-c       0.001692411 -0.07495877  0.07834359 0.9999300
ws-c    -0.134610648 -0.21935171 -0.04986958 0.0004275
wsp-c   -0.116478997 -0.19867823 -0.03427977 0.0020267
ws-p    -0.136303060 -0.22104412 -0.05156200 0.0003540
wsp-p   -0.118171408 -0.20037064 -0.03597218 0.0016921
wsp-ws   0.018131651 -0.07165899  0.10792229 0.9518030
```

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1021

```
> bs1021a<- aov(area ~ trt, data=wf1021bs)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3  26.98   8.994   2.086  0.112
Residuals    60 258.69   4.311
```

```
> bs1021d<- aov(depth ~ trt, data=wf1021bs)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.002809 0.0009364   2.266 0.0899
Residuals    60 0.024788 0.0004131
```

1227

```
> bs1227a<- aov(area ~ trt, data=wf1227bs)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   2525   841.7   3.489  0.021
Residuals    60  14474   241.2
> TukeyHSD(bs1227a)
               diff        lwr       upr     p adj
p-c       2.8518383 -10.18910 15.892776 0.9383380
ws-c      0.8687864 -12.17215 13.909724 0.9980400
wsp-c   -20.1741570 -38.76262 -1.585692 0.0283476
ws-p     -1.9830519 -16.49367 12.527564 0.9837251
wsp-p   -23.0259953 -42.67346 -3.378529 0.0153308
wsp-ws  -21.0429434 -40.69041 -1.395477 0.0312564
```

```
> bs1227d<- aov(depth ~ trt, data=wf1227bs)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.05216 0.017386   3.978 0.0119
Residuals    60 0.26225 0.004371
> TukeyHSD(bs1227d)
                diff         lwr          upr     p adj
p-c      1.446308e-02 -0.04104790  0.069974060 0.9010964
ws-c     7.353346e-05 -0.05543745  0.055584515 1.0000000
wsp-c   -9.163731e-02 -0.17076229 -0.012512326 0.0169518
ws-p    -1.438955e-02 -0.07615646  0.047377373 0.9267157
wsp-p   -1.061004e-01 -0.18973319 -0.022467582 0.0074070
wsp-ws  -9.171084e-02 -0.17534364 -0.008078037 0.0262223
```

	1614	> **bs1614a<- aov(area ~ trt, data=wf1614bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.61 0.2041 0.112 0.953 Residuals 60 109.29 1.8214	> **bs1614d<- aov(depth ~ trt, data=wf1614bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00027 0.0000910 0.104 0.958 Residuals 60 0.05264 0.0008774
	1797	> **bs1797a<- aov(area ~ trt, data=wf1797bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 5867 1955.7 3.521 0.0203 Residuals 60 33331 555.5 **> TukeyHSD(bs1797a)** diff lwr upr p adj p-c 3.199289 -16.59059 22.989165 0.9735980 ws-c -3.986511 -23.77639 15.803365 0.9508635 wsp-c -31.881750 -60.09011 -3.673394 0.0207078 ws-p -7.185800 -29.20594 14.834341 0.8241373 wsp-p -35.081039 -64.89645 -5.265627 0.0148181 wsp-ws -27.895239 -57.71065 1.920173 0.0747197	> **bs1797d<- aov(depth ~ trt, data=wf1797bs)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.02025 0.006751 1.365 0.262 Residuals 60 0.29682 0.004947

Dianella revoluta

		Area	Depth
28/06/12	1021	> **dr1021a<- aov(area ~ trt, data=wf1021dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 67.97 22.657 10.29 9.64e-06 Residuals 74 162.87 2.201 > **TukeyHSD(dr1021a)** diff lwr upr p adj p-c -0.2379296 -1.4710333 0.99517397 0.9571421 ws-c -2.2251718 -3.4582754 -0.99206814 0.0000583 wsp-c -1.5932405 -2.8601340 -0.32634698 0.0078200 ws-p -1.9872421 -3.2203457 -0.75413849 0.0003707 wsp-p -1.3553109 -2.6222044 -0.08841733 0.0313343 wsp-ws 0.6319312 -0.6349623 1.89882478 0.5589132	**> dr1021d<- aov(depth ~ trt, data=wf1021dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.008479 0.0028265 11.5 2.84e-06 Residuals 74 0.018184 0.0002457 > **TukeyHSD(dr1021d)** diff lwr upr p adj p-c -0.001841144 -0.014870259 0.011187972 0.9823790 ws-c -0.023883736 -0.036912851 -0.010854621 0.0000439 wsp-c -0.018683102 -0.032069245 -0.005296959 0.0025287 ws-p -0.022042592 -0.035071707 -0.009013477 0.0001741 wsp-p -0.016841959 -0.030228102 -0.003455815 0.0077852 wsp-ws 0.005200634 -0.008185509 0.018586777 0.7376291
	1227	> **dr1227a<- aov(area ~ trt, data=wf1227dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 22018 7339 24.07 5.63e-11 Residuals 74 22562 305 > **TukeyHSD(dr1227a)** diff lwr upr p adj p-c 0.708968 -13.80420 15.22213 0.9992370 ws-c -34.749028 -49.26219 -20.23586 0.0000001 wsp-c -31.426643 -46.33750 -16.51578 0.0000026 ws-p -35.457996 -49.97116 -20.94483 0.0000001 wsp-p -32.135611 -47.04647 -17.22475 0.0000016 wsp-ws 3.322385 -11.58847 18.23324 0.936114	> **dr1227d<- aov(depth ~ trt, data=wf1227dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.4259 0.1420 20 1.35e-09 Residuals 74 0.5252 0.0071 > **TukeyHSD(dr1227d)** diff lwr upr p adj p-c 0.002582771 -0.06743957 0.07260512 0.9996706 ws-c -0.154123382 -0.22414573 -0.08410104 0.0000010 wsp-c -0.137097122 -0.20903824 -0.06515600 0.0000212 ws-p -0.156706153 -0.22672850 -0.08668381 0.0000006 wsp-p -0.139679893 -0.21162101 -0.06773877 0.0000147 wsp-ws 0.017026260 -0.05491486 0.08896738 0.9246997
	1614	> **dr1614a<- aov(area ~ trt, data=wf1614dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 8.81 2.936 5.15 0.00274 Residuals 74 42.18 0.570 > **TukeyHSD(dr1614a)** diff lwr upr p adj p-c 0.2315567 -0.3959592 0.85907274 0.7669203 ws-c -0.2745762 -0.9020922 0.35293976 0.6599034 wsp-c -0.6856012 -1.3303126 -0.04088981 0.0327367 ws-p -0.5061330 -1.1336490 0.12138301 0.1563165 wsp-p -0.9171579 -1.5618693 -0.27244656 0.0020105 wsp-ws -0.4110250 -1.0557364 0.23368642 0.3437461	> **dr1614d<- aov(depth ~ trt, data=wf1614dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000432 0.0001442 1.067 0.368 Residuals 74 0.009998 0.0001351

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1797

```
> dr1797a<- aov(area ~ trt, data=wf1797dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  33364   11121   16.64 2.32e-08
Residuals    74  49447     668
> TukeyHSD(dr1797a)
               diff        lwr       upr     p adj
p-c       2.223249 -19.26208  23.70858 0.9929078
ws-c    -41.627584 -63.11291 -20.14226 0.0000153
wsp-c   -38.572441 -60.64652 -16.49836 0.0001019
ws-p    -43.850833 -65.33616 -22.36550 0.0000053
wsp-p   -40.795690 -62.86977 -18.72161 0.0000378
wsp-ws    3.055143 -19.01893  25.12922 0.9834080
```

```
> dr1797d<- aov(depth ~ trt, data=wf1797dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.3375 0.11250    9.72 1.75e-05
Residuals    74 0.8565 0.01157
> TukeyHSD(dr1797d)
                diff         lwr         upr     p adj
p-c      0.01055448 -0.07886651  0.09997548 0.9895650
ws-c    -0.13400394 -0.22342494 -0.04458295 0.0010352
wsp-c   -0.11607726 -0.20794860 -0.02420592 0.0074662
ws-p    -0.14455843 -0.23397942 -0.05513743 0.0003538
wsp-p   -0.12663174 -0.21850308 -0.03476040 0.0029273
wsp-ws   0.01792669 -0.07394466  0.10979803 0.9557738
```

1021

```
> wf1021drarea<- aov(area ~ trt, data=wf1021dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  51.43   17.14   10.21 1.03e-05
Residuals    75 125.97    1.68
> TukeyHSD(wf1021drarea)
               diff        lwr       upr     p adj
p-c       0.85015242 -0.2407788 1.9410837 0.1800716
ws-c     -0.03778592 -1.1287172 1.0531454 0.9997274
wsp-c     1.94062182  0.8496905 3.0315531 0.0000742
ws-p     -0.88793834 -1.9647925 0.1889158 0.1421004
wsp-p     1.09046940  0.0136152 2.1673236 0.0460347
wsp-ws    1.97840774  0.9015536 3.0552619 0.0000416
```

```
> wf1021drdepth<- aov(depth ~ trt, data=wf1021dr)
             Df   Sum Sq   Mean Sq F value   Pr(>F)
trt           3 0.005623 0.0018742   9.777 1.61e-05
Residuals    75 0.014377 0.0001917
> TukeyHSD(wf1021drdepth)
                 diff          lwr          upr     p adj
p-c       0.008115646 -0.003539156 0.019770447 0.2676953
ws-c     -0.002357806 -0.014012608 0.009296995 0.9511265
wsp-c     0.019151892  0.007497090 0.030806693 0.0002736
ws-p     -0.010473452 -0.021977863 0.001030959 0.0873547
wsp-p     0.011036246 -0.000468165 0.022540657 0.0648122
wsp-ws    0.021509698  0.010005287 0.033014109 0.0000300
```

1227

```
> wf1227dra<- aov(area ~ trt, data=wf1227dr)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3    440  146.69   2.563  0.061
Residuals    75   4292   57.23
```

```
> wf1227drdepth<- aov(depth ~ trt, data=wf1227dr)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00418 0.0013938    1.94   0.13
Residuals    75 0.05387 0.0007183
```

1614

```
> wf1614drarea<- aov(area ~ trt, data=wf1614dr)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3  1.331  0.4436   3.425 0.0214
Residuals    75  9.714  0.1295
> TukeyHSD(wf1614drarea)
               diff         lwr       upr     p adj
p-c       0.20448376 -0.09845873 0.5074262 0.2940590
ws-c      0.05763083 -0.24531166 0.3605733 0.9588522
wsp-c     0.33347340  0.03053091 0.6364159 0.0252261
ws-p     -0.14685293 -0.44588631 0.1521805 0.5718817
wsp-p     0.12898964 -0.17004374 0.4280230 0.6701729
wsp-ws    0.27584257 -0.02319082 0.5748760 0.0812697
```

```
> wf1614drd<- aov(depth ~ trt, data=wf1614dr)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.0003485 1.162e-04   3.327 0.0241
Residuals    75 0.0026185 3.491e-05
> TukeyHSD(wf1614drd)
                 diff          lwr          upr     p adj
p-c       0.0015015525 -0.0034722563 0.006475361 0.8572964
ws-c      0.0008252962 -0.0041485126 0.005799105 0.9720658
wsp-c     0.0054640546  0.0004902458 0.010437863 0.0256241
ws-p     -0.0006762563 -0.0055858843 0.004233372 0.9836537
wsp-p     0.0039625020 -0.0009471259 0.008872130 0.1559640
wsp-ws    0.0046387584 -0.0002708695 0.009548386 0.0709073
```

1797

```
> wf1797drarea<- aov(area ~ trt, data=wf1797dr)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3    697   232.2   2.917 0.0396
Residuals    75   5970    79.6
> TukeyHSD(wf1797drarea)
               diff        lwr       upr     p adj
p-c       3.522456 -3.9879531 11.032865 0.6084387
ws-c      1.720892 -5.7895176  9.231301 0.9311012
wsp-c     7.985320  0.4749109 15.495729 0.0327693
ws-p     -1.801564 -9.2150611  5.611932 0.9191946
wsp-p     4.462864 -2.9506326 11.876361 0.3951233
wsp-ws    6.264428 -1.1490681 13.677925 0.1271126
```

```
> wf1797drdepth<- aov(depth ~ trt, data=wf1797dr)
             Df  Sum Sq  Mean Sq F value  Pr(>F)
trt           3 0.01267 0.004223   4.336 0.00713
Residuals    75 0.07304 0.000974
> TukeyHSD(wf1797drdepth)
                 diff          lwr         upr     p adj
p-c       0.024460628 -0.001808969 0.050730220 0.0771059
ws-c      0.013631313 -0.012638284 0.039900913 0.5260275
wsp-c     0.034285404  0.008015807 0.060555500 0.0053409
ws-p     -0.010829315 -0.036759935 0.015101310 0.6922453
wsp-p     0.009824776 -0.016105844 0.035755400 0.7523888
wsp-ws    0.020654091 -0.005276529 0.046584710 0.1648326
```

16/08/12	1021	> **dr1021a<- aov(area ~ trt, data=wf1021dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 22.88 7.626 5.339 0.00216 Residuals 76 108.54 1.428 > **TukeyHSD(dr1021a)** diff lwr upr p adj p-c -0.9708057 -1.9635182 0.02190687 0.0576305 ws-c -1.2787367 -2.2714492 -0.28602418 0.0061125 wsp-c -0.1690180 -1.1617305 0.82369452 0.9699653 ws-p -0.3079310 -1.3006436 0.68478147 0.8472931 wsp-p 0.8017877 -0.1909249 1.79450017 0.1555578 wsp-ws 1.1097187 0.1170062 2.10243122 0.0223241	> **dr1021d<- aov(depth ~ trt, data=wf1021dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.002682 0.0008939 5.768 0.00131 Residuals 76 0.011778 0.0001550 > **TukeyHSD(dr1021d)** diff lwr upr p adj p-c -0.010569212 -0.020910040 -0.000228385 0.0432493 ws-c -0.014235052 -0.024575880 -0.003894225 0.0029526 wsp-c -0.002515232 -0.012856060 0.007825595 0.9190654 ws-p -0.003665840 -0.014006667 0.006674987 0.7882184 wsp-p 0.008053980 -0.002286847 0.018394807 0.1805628 wsp-ws 0.011719820 0.001378993 0.022060647 0.0199647
	1227	> **dr1227a<- aov(area ~ trt, data=wf1227dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 745 248.41 3.618 0.0168 Residuals 76 5218 68.65 > **TukeyHSD(dr1227a)** diff lwr upr p adj p-c 0.005306 -6.877399 6.8880114 1.0000000 ws-c -5.977318 -12.860023 0.9053874 0.1115115 wsp-c 2.239670 -4.643035 9.1223754 0.8279370 ws-p -5.982624 -12.865329 0.9000814 0.1110242 wsp-p 2.234364 -4.648341 9.1170694 0.8289423 wsp-ws 8.216988 1.334283 15.0996934 0.0127499	> **dr1227d<- aov(depth ~ trt, data=wf1227dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00993 0.003309 3.796 0.0136 Residuals 76 0.06626 0.000872 > **TukeyHSD(dr1227d)** diff lwr upr p adj p-c 0.006885679 -0.017641216 0.031412575 0.8816809 ws-c -0.016388069 -0.040914964 0.008138827 0.3029660 wsp-c 0.013585853 -0.010941042 0.038112748 0.4695647 ws-p -0.023273748 -0.047800643 0.001253147 0.0691563 wsp-p 0.006700173 -0.017826722 0.031227069 0.8898151 wsp-ws 0.029973922 0.005447026 0.054500817 0.0102775
	1614	> **dr1614a<- aov(area ~ trt, data=wf1614dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.528 0.5095 2.807 0.0453 Residuals 76 13.796 0.1815 > **TukeyHSD(dr1614a)** diff lwr upr p adj p-c -0.17053972 -0.52445305 0.1833736 0.5873593 ws-c -0.17951968 -0.53343301 0.1743936 0.5454023 wsp-c 0.15645934 -0.19745398 0.5103727 0.6529225 ws-p -0.00897996 -0.36289329 0.3449334 0.9998928 wsp-p 0.32699906 -0.02691426 0.6809124 0.0805565 wsp-ws 0.33597902 -0.01793430 0.6898924 0.0689773	> **dr1614d<- aov(depth ~ trt, data=wf1614dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0003174 1.058e-04 3.212 0.0276 Residuals 76 0.0025038 3.294e-05 > **TukeyHSD(dr1614d)** diff lwr upr p adj p-c -0.0004570366 -0.0052248300 0.004310757 0.9943502 ws-c -0.0020847292 -0.0068525226 0.002683064 0.6607953 wsp-c 0.0033908187 -0.0013769747 0.008158612 0.2503870 ws-p -0.0016276925 -0.0063954859 0.003140101 0.8065267 wsp-p 0.0038478553 -0.0009199380 0.008615649 0.1560684 wsp-ws 0.0054755479 0.0007077545 0.010243341 0.0178840
	1797	> **dr1797a<- aov(area ~ trt, data=wf1797dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 2927 975.6 10.44 7.89e-06 Residuals 76 7102 93.4 > **TukeyHSD(dr1797a)** diff lwr upr p adj p-c 12.2190280 4.189240 20.248816 0.0008330 ws-c 0.7372475 -7.292541 8.767036 0.9950256 wsp-c 12.6808100 4.651022 20.710598 0.0004933 ws-p -11.4817805 -19.511569 -3.451992 0.0018734 wsp-p 0.4617820 -7.568006 8.491570 0.9987607 wsp-ws 11.9435625 3.913774 19.973351 0.0011320	> **dr1797d<- aov(depth ~ trt, data=wf1797dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.04812 0.016041 17.27 1.2e-08 Residuals 76 0.07059 0.000929 > **TukeyHSD(dr1797d)** diff lwr upr p adj p-c 0.0540815740 0.02876518 0.07939797 0.0000018 ws-c 0.0119516995 -0.01336470 0.03726810 0.6035413 wsp-c 0.0545057265 0.02918933 0.07982212 0.0000015 ws-p -0.0421298745 -0.06744627 -0.01681348 0.0002228 wsp-p 0.0004241525 -0.02489224 0.02574055 0.9999691 wsp-ws 0.0425540270 0.01723763 0.06787042 0.0001900
30/08/12	1021	> **dr1021a<- aov(area ~ trt, data=wf1021dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 13.37 4.457 2.25 0.0893 Residuals 76 150.56 1.981	> **dr1021d<- aov(depth ~ trt, data=wf1021dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.001171 0.0003905 1.872 0.141 Residuals 76 0.015848 0.0002085
	1227	> **dr1227a<- aov(area ~ trt, data=wf1227dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 156 51.91 0.841 0.475 Residuals 76 4689 61.70	> **dr1227d<- aov(depth ~ trt, data=wf1227dr)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00205 0.0006848 0.899 0.446 Residuals 76 0.05787 0.0007614

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1614

```
> dr1614a<- aov(area ~ trt, data=wf1614dr)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3  1.041  0.3468   2.046   0.11
Residuals   156 26.449  0.1695
```

```
> dr1614d<- aov(depth ~ trt, data=wf1614dr)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.000197 6.554e-05   2.163 0.0946
Residuals   156 0.004726 3.030e-05
```

1797

```
> dr1797a<- aov(area ~ trt, data=wf1797dr)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3    845  281.74   4.009 0.0105
Residuals    76   5341   70.28
> TukeyHSD(dr1797a)
               diff         lwr       upr     p adj
p-c     0.7469215  -6.2167881  7.710631 0.9921341
ws-c    8.2222655   1.2585559 15.185975 0.0140756
wsp-c   4.0932700  -2.8704396 11.056980 0.4166976
ws-p    7.4753440   0.5116344 14.439054 0.0305261
wsp-p   3.3463485  -3.6173611 10.310058 0.5895576
wsp-ws -4.1289955 -11.0927051  2.834714 0.4089100
```

```
 dr1797d<- aov(depth ~ trt, data=wf1797dr)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.01939 0.006464    7.48 0.000188
Residuals    76 0.06568 0.000864
> TukeyHSD(dr1797d)
               diff         lwr         upr     p adj
p-c     0.01562637 -0.008792306 0.040045051 0.3407661
ws-c    0.04264404  0.018225361 0.067062719 0.0001009
wsp-c   0.02660198  0.002183305 0.051020662 0.0273161
ws-p    0.02701767  0.002598989 0.051436346 0.0242222
wsp-p   0.01097561 -0.013443068 0.035394290 0.6408254
wsp-ws -0.01604206 -0.040460735 0.008376622 0.3176720
```

1021

```
> dr1021a<- aov(area ~ trt, data=wf1021dr)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3  34.08  11.359   5.909 0.00111
Residuals    76 146.09   1.922
> TukeyHSD(dr1021a)
               diff        lwr        upr     p adj
p-c    -0.7064633 -1.8581557  0.4452291 0.3784722
ws-c   -1.6605528 -2.8122452 -0.5088604 0.0016892
wsp-c  -1.4370359 -2.5887283 -0.2853435 0.0084184
ws-p   -0.9540895 -2.1057819  0.1976029 0.1392352
wsp-p  -0.7305726 -1.8822650  0.4211198 0.3485406
wsp-ws  0.2235169 -0.9281755  1.3752093 0.9565188
```

```
> dr1021d<- aov(depth ~ trt, data=wf1021dr)
             Df   Sum Sq   Mean Sq F value  Pr(>F)
trt           3 0.003095 0.0010317   5.367 0.00209
Residuals    76 0.014611 0.0001922
> TukeyHSD(dr1021d)
                diff          lwr          upr     p adj
p-c    -0.007275282 -0.018792653  0.004242089 0.3522851
ws-c   -0.015968298 -0.027485668 -0.004450927 0.0027166
wsp-c  -0.013740404 -0.025257775 -0.002223033 0.0128317
ws-p   -0.008693016 -0.020210386  0.002824355 0.2036014
wsp-p  -0.006465122 -0.017982493  0.005052249 0.4577936
wsp-ws  0.002227894 -0.009289477  0.013745264 0.9569190
```

1227

```
> dr1227a<- aov(area ~ trt, data=wf1227dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3   1668   556.1   7.176 0.000264
Residuals    76   5890    77.5
> TukeyHSD(dr1227a)

               diff        lwr       upr     p adj
p-c     -6.753038 -14.065568  0.559492 0.0807823
ws-c    -7.445275 -14.757805 -0.132745 0.0443901
wsp-c  -12.862863 -20.175394 -5.550333 0.0000892
ws-p    -0.692237  -8.004767  6.620293 0.9945555
wsp-p   -6.109825 -13.422356  1.202705 0.1339600
wsp-ws  -5.417588 -12.730119  1.894942 0.2178198
```

```
> dr1227d<- aov(depth ~ trt, data=wf1227dr)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.02515 0.008385   6.465 0.000589
Residuals    76 0.09857 0.001297
> TukeyHSD(dr1227d)

                diff         lwr          upr     p adj
p-c    -0.027638739 -0.05755400  0.002276524 0.0805815
ws-c   -0.022596767 -0.05251203  0.007318496 0.2030183
wsp-c  -0.049899060 -0.07981432 -0.019983797 0.0002148
ws-p    0.005041972 -0.02487329  0.034957235 0.9708201
wsp-p  -0.022260321 -0.05217558  0.007654942 0.2144393
wsp-ws -0.027302293 -0.05721756  0.002612971 0.0862074
```

1614

```
> dr1614a<- aov(area ~ trt, data=wf1614dr)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3  0.454  0.1515   0.393  0.758
Residuals    76 29.284  0.3853
```

```
> dr1614d<- aov(depth ~ trt, data=wf1614dr)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.0001902 6.338e-05   1.525  0.215
Residuals    76 0.0031592 4.157e-05
```

1797

```
> dr1797a<- aov(area ~ trt, data=wf1797dr)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3   3366  1122.0   6.851 0.00038
Residuals    76  12447   163.8
> TukeyHSD(dr1797a)
               diff         lwr       upr     p adj
p-c     -8.490010 -19.120409  2.140388 0.1630870
ws-c    -4.211561 -14.841960  6.418838 0.7261665
wsp-c  -17.513175 -28.143574 -6.882777 0.0002610
ws-p     4.278449  -6.351949 14.908848 0.7163740
wsp-p   -9.023165 -19.653564  1.607234 0.1245163
wsp-ws -13.301614 -23.932013 -2.671216 0.0081891
```

```
> dr1797d<- aov(depth ~ trt, data=wf1797dr)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.03172 0.010574   3.768  0.014
Residuals    76 0.21326 0.002806
> TukeyHSD(dr1797d)
                diff          lwr          upr     p adj
p-c    -0.02504016 -0.06904228  1.896197e-02 0.4456391
ws-c   -0.00800238 -0.05200451  3.599975e-02 0.9637877
wsp-c  -0.05198618 -0.09598830 -7.984048e-03 0.0140002
ws-p    0.01703778 -0.02696435  6.103990e-02 0.7399659
wsp-p  -0.02694602 -0.07094815  1.705611e-02 0.3799813
wsp-ws -0.04398380 -0.08798592  1.833245e-05 0.0501356
```

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1021

```
> dr1021a<- aov(area ~ trt, data=wf1021dr)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3   22.5   7.500   5.594 0.00161
Residuals    76  101.9   1.341
> TukeyHSD(dr1021a)
               diff        lwr         upr     p adj
p-c    -0.5869422 -1.548757  0.37487287 0.3831096
ws-c   -1.0206042 -1.982419 -0.05878913 0.0332375
wsp-c  -1.4304947 -2.392310 -0.46867958 0.0011332
ws-p   -0.4336620 -1.395477  0.52815312 0.6385237
wsp-p  -0.8435524 -1.805368  0.11826267 0.1061778
wsp-ws -0.4098904 -1.371706  0.55192467 0.6787449
```

```
> dr1021d<- aov(depth ~ trt, data=wf1021dr)
             Df   Sum Sq   Mean Sq F value  Pr(>F)
trt           3 0.002414 0.0008048   5.594 0.00161
Residuals    76 0.010934 0.0001439
> TukeyHSD(dr1021d)
                diff         lwr           upr     p adj
p-c    -0.004156284 -0.01411993  0.0058073641 0.6931981
ws-c   -0.009979511 -0.01994316 -0.0000158634 0.0494847
wsp-c  -0.014404974 -0.02436862 -0.0044413259 0.0016326
ws-p   -0.005823228 -0.01578688  0.0041404206 0.4218206
wsp-p  -0.010248690 -0.02021234 -0.0002850419 0.0414132
wsp-ws -0.004425463 -0.01438911  0.0055381856 0.6495293
```

1227

```
> dr1227a<- aov(area ~ trt, data=wf1227dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3   3387  1129.1    13.2 5.05e-07
Residuals    76   6501    85.5
> TukeyHSD(dr1227a)

               diff        lwr         upr     p adj
p-c       1.603607  -6.078966  9.28617998 0.9467350
ws-c     -7.060786 -14.743359  0.62178698 0.0829621
wsp-c   -14.824658 -22.507230 -7.14208452 0.0000161
ws-p     -8.664393 -16.346966 -0.98182002 0.0207839
wsp-p   -16.428265 -24.110837 -8.74569152 0.0000018
wsp-ws   -7.763871 -15.446444 -0.08129852 0.0466599
```

```
> dr1227d<- aov(depth ~ trt, data=wf1227dr)
             Df  Sum Sq  Mean Sq F value  Pr(>F)
trt           3 0.04872 0.016240   12.09 1.5e-06
Residuals    76 0.10212 0.001344
> TukeyHSD(dr1227d)

                diff         lwr           upr     p adj
p-c      0.01264280 -0.01780603  0.0430916287 0.6962722
ws-c    -0.01809657 -0.04854540  0.0123522602 0.4068006
wsp-c   -0.05292003 -0.08336886 -0.0224711973 0.0001095
ws-p    -0.03073937 -0.06118820 -0.0002905393 0.0469795
wsp-p   -0.06556283 -0.09601166 -0.0351139968 0.0000015
wsp-ws  -0.03482346 -0.06527229 -0.0043746283 0.0185200
```

1614

```
> dr1614a<- aov(area ~ trt, data=wf1614dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  6.752  2.2506   15.08 8.62e-08
Residuals    76 11.345  0.1493
> TukeyHSD(dr1614a)

               diff        lwr         upr     p adj
p-c       0.6376607  0.3167256  0.95859580 0.0000089
ws-c      0.1274188 -0.1935163  0.44835393 0.7248605
wsp-c    -0.1276865 -0.4486216  0.19324857 0.7235661
ws-p     -0.5102419 -0.8311770 -0.18930676 0.0004472
wsp-p    -0.7653472 -1.0862823 -0.44441212 0.0000001
wsp-ws   -0.2551054 -0.5760405  0.06582975 0.1663156
```

```
> dr1614d<- aov(depth ~ trt, data=wf1614dr)
             Df    Sum Sq   Mean Sq F value  Pr(>F)
trt           3 0.0003958 1.320e-04   4.159 0.00878
Residuals    76 0.0024114 3.173e-05
> TukeyHSD(dr1614d)
                 diff          lwr          upr     p adj
p-c       0.003852215 -0.0008267709  0.008531202 0.1431371
ws-c      0.001392591 -0.0032863953  0.006071577 0.8624787
wsp-c    -0.002282086 -0.0069610724  0.002396900 0.5776516
ws-p     -0.002459624 -0.0071386107  0.002219362 0.5151521
wsp-p    -0.006134302 -0.0108132878 -0.001455315 0.0050799
wsp-ws   -0.003674677 -0.0083536634  0.001004309 0.1746770
```

1797

```
> dr1797a<- aov(area ~ trt, data=wf1797dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3   3032  1010.8   7.245 0.000244
Residuals    76  10603   139.5
> TukeyHSD(dr1797a)
               diff        lwr         upr     p adj
p-c       6.4541415  -3.357486 16.2657685 0.3165466
ws-c     -0.6675545 -10.479182  9.1440725 0.9979551
wsp-c   -10.7558425 -20.567470 -0.9442155 0.0260362
ws-p     -7.1216960 -16.933323  2.6899310 0.2339260
wsp-p   -17.2099840 -27.021611 -7.3983570 0.0000936
wsp-ws  -10.0882880 -19.899915 -0.2766610 0.0415268
```

```
> dr1797d<- aov(depth ~ trt, data=wf1797dr)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.02534 0.008446    6.07 0.000926
Residuals    76 0.10575 0.001392
> TukeyHSD(dr1797d)
                diff          lwr           upr     p adj
p-c      0.03095314 -3.314867e-05  6.193943e-02 0.0503488
ws-c     0.01333593 -1.765036e-02  4.432222e-02 0.6719381
wsp-c   -0.01758444 -4.857073e-02  1.340185e-02 0.4481076
ws-p    -0.01761721 -4.860350e-02  1.336908e-02 0.4464506
wsp-p   -0.04853758 -7.952387e-02 -1.755129e-02 0.0005549
wsp-ws  -0.03092037 -6.190666e-02  6.591667e-05 0.0506957
```

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1021

```
> dr1021a<- aov(area ~ trt, data=wf1021dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  51.76  17.252   8.929 3.86e-05
Residuals    76 146.85   1.932
> TukeyHSD(dr1021a)
               diff        lwr        upr     p adj
p-c    -0.7690618 -1.9237176  0.3855940 0.3057090
ws-c   -2.1190611 -3.2737169 -0.9644053 0.0000419
wsp-c  -1.5806561 -2.7353119 -0.4260003 0.0031485
ws-p   -1.3499993 -2.5046551 -0.1953435 0.0153443
wsp-p  -0.8115943 -1.9662501  0.3430615 0.2600460
wsp-ws  0.5384050 -0.6162508  1.6930609 0.6131349
```

```
> dr1021d<- aov(depth ~ trt, data=wf1021dr)
             Df   Sum Sq   Mean Sq F value   Pr(>F)
trt           3 0.005816 0.0019387   10.68 6.16e-06
Residuals    76 0.013794 0.0001815
> TukeyHSD(dr1021d)
                diff         lwr           upr     p adj
p-c    -0.006031311 -0.01722209  0.0051594626 0.4935782
ws-c   -0.021011155 -0.03220193 -0.0098203811 0.0000274
wsp-c  -0.017710551 -0.02890133 -0.0065197769 0.0004782
ws-p   -0.014979844 -0.02617062 -0.0037890696 0.0040533
wsp-p  -0.011679240 -0.02287001 -0.0004884654 0.0374356
wsp-ws  0.003300604 -0.00789017  0.0144913784 0.8656281
```

1227

```
> dr1227a<- aov(area ~ trt, data=wf1227dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3   7862    2621   19.55 1.72e-09 ***
Residuals    76  10188     134
> TukeyHSD(dr1227a)
               diff        lwr        upr     p adj
p-c      1.6638210  -7.953556  11.281198 0.9685684
ws-c   -18.5352100 -28.152587  -8.917833 0.0000165
wsp-c  -19.3667277 -28.984105  -9.749351 0.0000067
ws-p   -20.1990310 -29.816408 -10.581654 0.0000027
wsp-p  -21.0305487 -30.647926 -11.413172 0.0000011
wsp-ws  -0.8315177 -10.448895   8.785859 0.9958355
```

```
> dr1227d<- aov(depth ~ trt, data=wf1227dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.1279 0.04264    16.6 2.17e-08 ***
Residuals    76 0.1952 0.00257
> TukeyHSD(dr1227d)
                diff         lwr          upr     p adj
p-c     0.01009336 -0.03200652  0.05219325 0.9221565
ws-c   -0.06897123 -0.11107112 -0.02687134 0.0002846
wsp-c  -0.07954728 -0.12164717 -0.03744739 0.0000243
ws-p   -0.07906459 -0.12116448 -0.03696470 0.0000272
wsp-p  -0.08964064 -0.13174053 -0.04754076 0.0000020
wsp-ws -0.01057605 -0.05267594  0.03152383 0.9117390
```

1614

```
> dr1614a<- aov(area ~ trt, data=wf1614dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  4.121  1.3737   6.453 0.000597
Residuals    76 16.178  0.2129
> TukeyHSD(dr1614a)
               diff        lwr        upr     p adj
p-c     0.2777171 -0.1055307  0.66096490 0.2352479
ws-c   -0.3438301 -0.7270779  0.03941770 0.0944889
wsp-c  -0.1494226 -0.5326704  0.23382519 0.7358661
ws-p   -0.6215472 -1.0047950 -0.23829942 0.0003322
wsp-p  -0.4271397 -0.8103875 -0.04389193 0.0228643
wsp-ws  0.1944075 -0.1888403  0.57765527 0.5453673
```

```
> dr1614d<- aov(depth ~ trt, data=wf1614dr)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.0003201 1.067e-04   3.058 0.0333
Residuals    76 0.0026517 3.489e-05
> TukeyHSD(dr1614d)
                diff          lwr          upr     p adj
p-c     0.0024385363 -0.002468035  0.0073451073 0.5623231
ws-c   -0.0032010495 -0.008107621  0.0017055215 0.3237668
wsp-c  -0.0003252625 -0.005231834  0.0045813085 0.9981073
ws-p   -0.0056395858 -0.010546157 -0.0007330147 0.0177599
wsp-p  -0.0027637988 -0.007670370  0.0021427723 0.4547190
wsp-ws  0.0028757870 -0.002030784  0.0077823580 0.4192779
```

1797

```
> dr1797a<- aov(area ~ trt, data=wf1797dr)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3   9032  3010.7   11.57 2.49e-06
Residuals    76  19767   260.1
> TukeyHSD(dr1797a)
               diff        lwr        upr     p adj
p-c      6.809985  -6.586642  20.206611 0.5435996
ws-c   -14.422644 -27.819271  -1.026018 0.0298735
wsp-c  -19.528967 -32.925593  -6.132341 0.0014702
ws-p   -21.232629 -34.629255  -7.836003 0.0004680
wsp-p  -26.338952 -39.735578 -12.942325 0.0000111
wsp-ws  -5.106323 -18.502949   8.290304 0.7491250
```

```
> dr1797d<- aov(depth ~ trt, data=wf1797dr)
             Df Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.0592 0.019733   7.044 0.000306
Residuals    76 0.2129 0.002801
> TukeyHSD(dr1797d)
                diff         lwr          upr     p adj
p-c     0.02929836 -0.01466662  0.073263339 0.3052547
ws-c   -0.02530087 -0.06926585  0.018664108 0.4356495
wsp-c  -0.04289079 -0.08685577  0.001074185 0.0585043
ws-p   -0.05459923 -0.09856421 -0.010634253 0.0088145
wsp-p  -0.07218915 -0.11615413 -0.028224176 0.0002748
wsp-ws -0.01758992 -0.06155490  0.026375056 0.7200948
```

25/10/12	1021	> dr1021a<- aov(area ~ trt, data=wf1021dr) Df Sum Sq Mean Sq F value Pr(>F) trt 3 67.97 22.657 10.29 9.64e-06 Residuals 74 162.87 2.201 > TukeyHSD(dr1021a) diff lwr upr p adj p-c -0.2379296 -1.4710333 0.99517397 0.9571421 ws-c -2.2251718 -3.4582754 -0.99206814 0.0000583 wsp-c -1.5932405 -2.8601340 -0.32634698 0.0078200 ws-p -1.9872421 -3.2203457 -0.75413849 0.0003707 wsp-p -1.3553109 -2.6222044 -0.08841733 0.0313343 wsp-ws 0.6319312 -0.6349623 1.89882478 0.5589132	> dr1021d<- aov(depth ~ trt, data=wf1021dr) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.008479 0.0028265 11.5 2.84e-06 Residuals 74 0.018184 0.0002457 > TukeyHSD(dr1021d) diff lwr upr p adj p-c -0.001841144 -0.014870259 0.011187972 0.9823790 ws-c -0.023883736 -0.036912851 -0.010854621 0.0000439 wsp-c -0.018683102 -0.032069245 -0.005296959 0.0025287 ws-p -0.022042592 -0.035071707 -0.009013477 0.0001741 wsp-p -0.016841959 -0.030228102 -0.003455815 0.0077852 wsp-ws 0.005200634 -0.008185509 0.018586777 0.7376291
	1227	> dr1227a<- aov(area ~ trt, data=wf1227dr) Df Sum Sq Mean Sq F value Pr(>F) trt 3 22018 7339 24.07 5.63e-11 Residuals 74 22562 305 > TukeyHSD(dr1227a) diff lwr upr p adj p-c 0.708968 -13.80420 15.22213 0.9992370 ws-c -34.749028 -49.26219 -20.23586 0.0000001 wsp-c -31.426643 -46.33750 -16.51578 0.0000026 ws-p -35.457996 -49.97116 -20.94483 0.0000001 wsp-p -32.135611 -47.04647 -17.22475 0.0000016 wsp-ws 3.322385 -11.58847 18.23324 0.9361140	> dr1227d<- aov(depth ~ trt, data=wf1227dr) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.4259 0.1420 20 1.35e-09 Residuals 74 0.5252 0.0071 > TukeyHSD(dr1227d) diff lwr upr p adj p-c 0.002582771 -0.06743957 0.07260512 0.9996706 ws-c -0.154123382 -0.22414573 -0.08410104 0.0000010 wsp-c -0.137097122 -0.20903824 -0.06515600 0.0000212 ws-p -0.156706153 -0.22672850 -0.08668381 0.0000006 wsp-p -0.139679893 -0.21162101 -0.06773877 0.0000147 wsp-ws 0.017026260 -0.05491486 0.08896738 0.9246997
	1614	> dr1614a<- aov(area ~ trt, data=wf1614dr) Df Sum Sq Mean Sq F value Pr(>F) trt 3 8.81 2.936 5.15 0.00274 Residuals 74 42.18 0.570 > TukeyHSD(dr1614a) diff lwr upr p adj p-c 0.2315567 -0.3959592 0.85907274 0.7669203 ws-c -0.2745762 -0.9020922 0.35293976 0.6599034 wsp-c -0.6856012 -1.3303126 -0.04088981 0.0327367 ws-p -0.5061330 -1.1336490 0.12138301 0.1563165 wsp-p -0.9171579 -1.5618693 -0.27244656 0.0020105 wsp-ws -0.4110250 -1.0557364 0.23368642 0.3437461	> dr1614d<- aov(depth ~ trt, data=wf1614dr) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000432 0.0001442 1.067 0.368 Residuals 74 0.009998 0.0001351
	1797	> dr1797a<- aov(area ~ trt, data=wf1797dr) Df Sum Sq Mean Sq F value Pr(>F) trt 3 33364 11121 16.64 2.32e-08 Residuals 74 49447 668 > TukeyHSD(dr1797a diff lwr upr p adj p-c 2.223249 -19.26208 23.70858 0.9929078 ws-c -41.627584 -63.11291 -20.14226 0.0000153 wsp-c -38.572441 -60.64652 -16.49836 0.0001019 ws-p -43.850833 -65.33616 -22.36550 0.0000053 wsp-p -40.795690 -62.86977 -18.72161 0.0000378 wsp-ws 3.055143 -19.01893 25.12922 0.983408	> dr1797d<- aov(depth ~ trt, data=wf1797dr) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.3375 0.11250 9.72 1.75e-05 *** Residuals 74 0.8565 0.01157 > TukeyHSD(dr1797d) diff lwr upr p adj p-c 0.01055448 -0.07886651 0.09997548 0.9895650 ws-c -0.13400394 -0.22342494 -0.04458295 0.0010352 wsp-c -0.11607726 -0.20794860 -0.02420592 0.0074662 ws-p -0.14455843 -0.23397942 -0.05513743 0.0003538 wsp-p -0.12663174 -0.21850308 -0.03476040 0.0029273 wsp-ws 0.01792669 -0.07394466 0.10979803 0.9557738

Eucalyptus piperita

		Area	Depth

28/06/12	1021	**> ep1021a<- aov(area ~ trt, data=wf1021ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 53.62 17.873 5.414 0.00264 Residuals 50 165.07 3.301 **> TukeyHSD(ep1021a)** diff lwr upr p adj p-c 0.06146245 -1.419163 1.54208756 0.9995129 ws-c -2.32264364 -4.598975 -0.04631249 0.0438973 wsp-c -2.80282626 -5.472061 -0.13359139 0.0361621 ws-p -2.38410609 -4.571134 -0.19707787 0.0276508 wsp-p -2.86428872 -5.457785 -0.27079228 0.0250785 wsp-ws -0.48018263 -3.597177 2.63681217 0.9765835	**> ep1021d<- aov(depth ~ trt, data=wf1021ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.007363 0.0024545 5.643 0.00207 Residuals 50 0.021747 0.0004349 **> TukeyHSD(ep1021d)** diff lwr upr p adj p-c -0.0000613903 -0.01705582 0.016933035 0.9999997 ws-c -0.0288904284 -0.05501787 -0.002762991 0.0248491 wsp-c -0.0318044840 -0.06244162 -0.001167348 0.0391150 ws-p -0.0288290381 -0.05393147 -0.003726609 0.0184552 wsp-p -0.0317430937 -0.06151091 -0.001975274 0.0324506 wsp-ws -0.0029140556 -0.03869052 0.032862411 0.9963707
	1227	> **ep1227a<- aov(area ~ trt, data=wf1227ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 4835 1611.6 7.966 0.000194 Residuals 50 10115 202.3 > **TukeyHSD(ep1227a)** diff lwr upr p adj p-c 0.3479289 -11.24198 11.937834 0.9998154 ws-c -21.3024163 -39.12088 -3.483955 0.0131726 wsp-c -27.7827586 -48.67676 -6.888760 0.0047962 ws-p -21.6503452 -38.76977 -4.530921 0.0078945 wsp-p -28.1306875 -48.43183 -7.829548 0.0030863 wsp-ws -6.4803423 -30.87928 17.918591 0.894304	> **ep1227d<- aov(depth ~ trt, data=wf1227ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.06006 0.020022 7.454 0.000321 Residuals 50 0.13430 0.002686 > **TukeyHSD(ep1227d)** diff lwr upr p adj p-c 0.0005569345 -0.04167544 0.04278931 0.9999843 ws-c -0.0769881533 -0.14191672 -0.01205959 0.0141406 wsp-c -0.0966666117 -0.17280211 -0.02053112 0.0076020 ws-p -0.0775450878 -0.13992644 -0.01516374 0.0092816 wsp-p -0.0972235462 -0.17119873 -0.02324836 0.0054056 wsp-ws -0.0196784583 -0.10858556 0.06922864 0.9351829
	1614	> **ep1614a<- aov(area ~ trt, data=wf1614ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.789 0.5963 1.417 0.249 Residuals 48 20.199 0.4208	> **ep1614d<- aov(depth ~ trt, data=wf1614ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.002054 0.0006846 7.85 0.000231 Residuals 48 0.004186 0.0000872 > **TukeyHSD(ep1614d)** diff lwr upr p adj p-c -0.001235321 -0.008799648 0.0063290069 0.9722043 ws-c -0.020925795 -0.034598295 -0.0072532959 0.0009644 wsp-c -0.014455294 -0.028127794 -0.0007827946 0.0345199 ws-p -0.019690475 -0.033074594 -0.0063063555 0.0015733 wsp-p -0.013219973 -0.026604093 0.0001641457 0.0540128 wsp-ws 0.006470501 -0.011103708 0.0240447108 0.7615029
	1797	> **ep1797a<- aov(area ~ trt, data=wf1797ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1537 512.5 0.962 0.418 Residuals 50 26647 532.9	> **ep1797d<- aov(depth ~ trt, data=wf1797ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00125 0.0004152 0.259 0.855 Residuals 50 0.08021 0.0016043
17/07/12	1021	> **wf1021eparea<- aov(area ~ trt, data=wf1021ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 3.2 1.075 0.263 0.852 Residuals 92 376.6 4.093	**> wf1021epdepth<- aov(depth ~ trt, data=wf1021ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00023 0.0000753 0.154 0.927 Residuals 92 0.04506 0.0004898
	1227	> **wf1227epa<- aov(area ~ trt, data=wf1227ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 56 18.58 0.089 0.966 Residuals 92 19146 208.11	> **wf1227epdepth<- aov(depth ~ trt, data=wf1227ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00249 0.0008285 0.402 0.752 Residuals 92 0.18962 0.0020611
	1614	**> wf1614eparea<- aov(area ~ trt, data=wf1614ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.709 0.2362 0.917 0.436 Residuals 92 23.699 0.2576	**> wf1614epd<- aov(depth ~ trt, data=wf1614ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000044 1.460e-05 0.268 0.848 Residuals 92 0.005016 5.452e-05
	1797	**> wf1797eparea<- aov(area ~ trt, data=wf1797ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 131 43.77 0.508 0.678 Residuals 92 7925 86.14	> **wf1797epdepth<- aov(depth ~ trt, data=wf1797ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00221 0.0007364 1.661 0.181 Residuals 92 0.04080 0.0004434

16/08/12

1021

```
> ep1021a<- aov(area ~ trt, data=wf1021ep)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3  110.7   36.90   5.051 0.0028
Residuals    90  657.5    7.31
> TukeyHSD(ep1021a)
               diff         lwr        upr     p adj
p-c     2.008294 -0.04124675  4.0578352 0.0569736
ws-c    0.475437 -1.57410403  2.5249780 0.9295357
wsp-c  -1.018616 -3.20452668  1.1672937 0.6159744
ws-p   -1.532857 -3.49514339  0.4294288 0.1795076
wsp-p  -3.026911 -5.13122816 -0.9225933 0.0016602
wsp-ws -1.494053 -3.59837088  0.6102640 0.2533157
```

```
> ep1021d<- aov(depth ~ trt, data=wf1021ep)
             Df  Sum Sq  Mean Sq F value  Pr(>F)
trt           3 0.01458 0.004861   5.563 0.00151
Residuals    90 0.07864 0.000874
> TukeyHSD(ep1021d)
                diff          lwr          upr     p adj
p-c     0.020956065 -0.001459217  0.043371347 0.0755780
ws-c    0.003845627 -0.018569656  0.026260909 0.9696404
wsp-c  -0.014384700 -0.038291415  0.009522016 0.3979493
ws-p   -0.017110438 -0.038571437  0.004350560 0.1651957
wsp-p  -0.035340765 -0.058355122 -0.012326407 0.0006882
wsp-ws -0.018230326 -0.041244684  0.004784031 0.1696748
```

1227

```
> ep1227a<- aov(area ~ trt, data=wf1227ep)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3  15705    5235   5.834 0.00109
Residuals    90  80754     897
> TukeyHSD(ep1227a)
               diff        lwr        upr     p adj
p-c     17.013534  -5.700858  39.727926 0.2106747
ws-c    -2.162428 -24.876820  20.551964 0.9945279
wsp-c  -20.074544 -44.300271   4.151183 0.1398053
ws-p   -19.175962 -40.923336   2.571413 0.1037739
wsp-p  -37.088078 -60.409539 -13.766617 0.0004120
wsp-ws -17.912116 -41.233577   5.409345 0.1917897
```

```
> ep1227d<- aov(depth ~ trt, data=wf1227ep)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3 0.2675 0.08918   5.375 0.00189
Residuals    90 1.4932 0.01659
> TukeyHSD(ep1227d)
                diff         lwr         upr     p adj
p-c     0.063855120 -0.03381934  0.16152958 0.3238744
ws-c   -0.008281502 -0.10595596  0.08939296 0.9961152
wsp-c  -0.089733854 -0.19390723  0.01443952 0.1166038
ws-p   -0.072136622 -0.16565280  0.02137955 0.1886152
wsp-p  -0.153588974 -0.25387390 -0.05330405 0.0007146
wsp-ws -0.081452352 -0.18173728  0.01883258 0.1527127
```

1614

```
> ep1614a<- aov(area ~ trt, data=wf1614ep)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   1.25  0.4179   0.802  0.496
Residuals    90  46.89  0.5210
```

```
> ep1614d<- aov(depth ~ trt, data=wf1614ep)
             Df    Sum Sq    Mean Sq F value   Pr(>F)
trt           3 0.001878 0.0006262    7.57 0.000143
Residuals    90 0.007444 0.0000827
> TukeyHSD(ep1614d)
                diff          lwr          upr     p adj
p-c     0.004993568 -0.001902829  1.188996e-02 0.2373132
ws-c   -0.003322415 -0.010218812  3.573982e-03 0.5899202
wsp-c  -0.007256443 -0.014611701  9.881581e-05 0.0545688
ws-p   -0.008315983 -0.014918780 -1.713185e-03 0.0075196
wsp-p  -0.012250011 -0.019330722 -5.169300e-03 0.0001056
wsp-ws -0.003934028 -0.011014739  3.146683e-03 0.4692516
```

1797

```
> ep1797a<- aov(area ~ trt, data=wf1797ep)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3  21840    7280   4.093 0.00906
Residuals    88 156529    1779
> TukeyHSD(ep1797a)
               diff       lwr        upr     p adj
p-c     12.004965 -19.99001  43.999944 0.7596965
ws-c    -4.768778 -37.36905  27.831491 0.9807557
wsp-c  -31.250551 -65.37436   2.873260 0.0848473
ws-p   -16.773743 -48.03828  14.490789 0.4996326
wsp-p  -43.255516 -76.10560 -10.405433 0.0047308
wsp-ws -26.481774 -59.92167   6.958125 0.1697358
```

```
> ep1797d<- aov(depth ~ trt, data=wf1797ep)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3 0.0927 0.03089   2.706 0.0501
Residuals    88 1.0043 0.01141
```

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1021

```
> ep1021a<- aov(area ~ trt, data=wf1021ep)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3  21.71   7.237   2.786 0.0464
Residuals    76 197.43   2.598
> TukeyHSD(ep1021a)
              diff         lwr       upr     p adj
p-c     1.4062669  0.1080919 2.7044419 0.0285167
ws-c    0.6514592 -0.6686584 1.9715767 0.5681259
wsp-c   0.6365759 -0.9412664 2.2144181 0.7148498
ws-p   -0.7548077 -1.9532650 0.4436495 0.3549122
wsp-p  -0.7696910 -2.2472483 0.7078663 0.5229238
wsp-ws -0.0148833 -1.5117559 1.4819893 0.9999935
```

```
> ep1021d<- aov(depth ~ trt, data=wf1021ep)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.002378 0.0007926    2.62 0.0568
Residuals    76 0.022991 0.0003025
```

1227

```
> ep1227a<- aov(area ~ trt, data=wf1227ep)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3    844   281.2   2.665 0.0542
Residuals    72   7597   105.5
```

```
> ep1227d<- aov(depth ~ trt, data=wf1227ep)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.01202 0.004008   2.911 0.0402
Residuals    72 0.09911 0.001377
> TukeyHSD(ep1227d)
               diff         lwr         upr     p adj
p-c     0.0200333294 -0.01039258 0.050459235 0.3150438
ws-c    0.0202792658 -0.01073369 0.051292220 0.3210744
wsp-c  -0.0113983756 -0.04776429 0.024967537 0.8427538
ws-p    0.0002459364 -0.02855607 0.029047940 0.9999959
wsp-p  -0.0314317050 -0.06593144 0.003068029 0.0869186
wsp-ws -0.0316776414 -0.06669620 0.003340915 0.0903173
```

1614

```
> ep1614a<- aov(area ~ trt, data=wf1614ep)
              Df Sum Sq Mean Sq F value Pr(>F)
trt            3  12.35   4.118   3.089  0.029
Residuals    148 197.28   1.333
> TukeyHSD(ep1614a)
               diff          lwr       upr     p adj
p-c     0.20056497 -0.460873722 0.8620037 0.8598487
ws-c    0.09760022 -0.576600521 0.7718010 0.9817919
wsp-c   0.86368980  0.073119347 1.6542602 0.0262748
ws-p   -0.10296475 -0.729100901 0.5231714 0.9737210
wsp-p   0.66312483 -0.086876157 1.4131258 0.1032586
wsp-ws  0.76608957  0.004809747 1.5273694 0.0479352
```

```
> ep1614d<- aov(depth ~ trt, data=wf1614ep)
              Df  Sum Sq   Mean Sq F value   Pr(>F)
trt            3 0.00542 0.0018079   5.732 0.000974
Residuals    148 0.04668 0.0003154
> TukeyHSD(ep1614d)
                diff          lwr           upr     p adj
p-c    -1.221427e-02 -0.022388824 -2.039709e-03 0.0115773
ws-c   -1.582839e-02 -0.026199257 -5.457518e-03 0.0006508
wsp-c  -1.217635e-02 -0.024337275 -1.543062e-05 0.0495794
ws-p   -3.614121e-03 -0.013245639  6.017396e-03 0.7638603
wsp-p   3.791333e-05 -0.011498951  1.157478e-02 0.9999998
wsp-ws  3.652035e-03 -0.008058326  1.536239e-02 0.8494895
```

1797

```
> ep1797a<- aov(area ~ trt, data=wf1797ep)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   1209   402.9   0.751  0.525
Residuals    72  38639   536.6
```

```
> ep1797d<- aov(depth ~ trt, data=wf1797ep)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.03134 0.010446   3.007 0.0358
Residuals    72 0.25011 0.003474
> TukeyHSD(ep1797d)
               diff         lwr         upr     p adj
p-c     0.02678604 -0.021547002 0.075119089 0.4682738
ws-c    0.04243253 -0.006833069 0.091698134 0.1159219
wsp-c  -0.01169298 -0.069462016 0.046076061 0.9509146
ws-p    0.01564649 -0.030106908 0.061399886 0.8051456
wsp-p  -0.03847902 -0.093283543 0.016325501 0.2603886
wsp-ws -0.05412551 -0.109754207 0.001503187 0.0594480
```

13/09/12	1021	**> ep1021a<- aov(area ~ trt, data=wf1021ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 55.23 18.41 6.304 0.000708 Residuals 76 221.96 2.92 **> TukeyHSD(dr1021a)** diff lwr upr p adj p-c -0.7064633 -1.8581557 0.4452291 0.3784722 ws-c -1.6605528 -2.8122452 -0.5088604 0.0016892 wsp-c -1.4370359 -2.5887283 -0.2853435 0.0084184 ws-p -0.9540895 -2.1057819 0.1976029 0.1392352 wsp-p -0.7305726 -1.8822650 0.4211198 0.3485406 wsp-ws 0.2235169 -0.9281755 1.3752093 0.9565188	**> ep1021d<- aov(depth ~ trt, data=wf1021ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.006098 0.0020328 5.841 0.00121 Residuals 76 0.026452 0.0003481 > **TukeyHSD(dr1021d)** diff lwr upr p adj p-c -0.007275282 -0.018792653 0.004242089 0.3522851 ws-c -0.015968298 -0.027485668 -0.004450927 0.0027166 wsp-c -0.013740404 -0.025257775 -0.002223033 0.0128317 ws-p -0.008693016 -0.020210386 0.002824355 0.2036014 wsp-p -0.006465122 -0.017982493 0.005052249 0.4577936 wsp-ws 0.002227894 -0.009289477 0.013745264 0.9569190
	1227	> **ep1227a<- aov(area ~ trt, data=wf1227ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 2846 948.7 3.421 0.0214 Residuals 76 21076 277.3 **> TukeyHSD(ep1227a)** diff lwr upr p adj p-c 11.206930 -2.205652 24.619511 0.1339415 ws-c 1.418733 -12.220557 15.058022 0.9928127 wsp-c -5.550948 -21.853016 10.751121 0.8077426 ws-p -9.788197 -22.170507 2.594113 0.1701260 wsp-p -16.757877 -32.023814 -1.491940 0.0257629 wsp-ws -6.969680 -22.435181 8.495821 0.6388830	**> ep1227d<- aov(depth ~ trt, data=wf1227ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0401 0.01338 3.119 0.0309 Residuals 76 0.3261 0.00429 **> TukeyHSD(ep1227d)** diff lwr upr p adj p-c 0.035760969 -0.01699531 0.088517244 0.2905884 ws-c 0.001574509 -0.05207348 0.055222502 0.9998342 wsp-c -0.030707449 -0.09482907 0.033414167 0.5922852 ws-p -0.034186460 -0.08289032 0.014517404 0.2611820 wsp-p -0.066468418 -0.12651457 -0.006422263 0.0241327 wsp-ws -0.032281958 -0.09311306 0.028549148 0.5069921
	1614	**> ep1614a<- aov(area ~ trt, data=wf1614ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 16.1 5.367 0.532 0.662 Residuals 76 766.4 10.084	**> ep1614d<- aov(depth ~ trt, data=wf1614ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00438 0.001459 1.043 0.379 Residuals 76 0.10629 0.001399
	1797	**> ep1797a<- aov(area ~ trt, data=wf1797ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 3405 1134.9 1.961 0.127 Residuals 76 43984 578.7	**> ep1797d<- aov(depth ~ trt, data=wf1797ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0229 0.007627 0.965 0.414 Residuals 76 0.6004 0.007901
27/09/12	1021	**> ep1021a<- aov(area ~ trt, data=wf1021ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 328.5 109.49 18.11 4.65e-09 Residuals 80 483.8 6.05 **> TukeyHSD(ep1021a)** diff lwr upr p adj p-c -0.07912551 -2.057560 1.899309 0.9995829 ws-c -4.35163785 -6.363513 -2.339762 0.0000013 wsp-c -3.33658341 -5.553562 -1.119605 0.0009496 ws-p -4.27251235 -6.098976 -2.446049 0.0000002 wsp-p -3.25745790 -5.307658 -1.207258 0.0004404 wsp-ws 1.01505444 -1.067435 3.097543 0.5789294	**> ep1021d<- aov(depth ~ trt, data=wf1021ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.04191 0.013969 19.3 1.65e-09 Residuals 80 0.05791 0.000724 **> TukeyHSD(ep1021d)** diff lwr upr p adj p-c 0.001631344 -0.02001413 0.02327682 0.9972387 ws-c -0.047732597 -0.06974393 -0.02572126 0.0000012 wsp-c -0.035977301 -0.06023261 -0.01172199 0.0011538 ws-p -0.049363941 -0.06934674 -0.02938114 0.0000000 wsp-p -0.037608645 -0.06003928 -0.01517801 0.0001916 wsp-ws 0.011755296 -0.01102860 0.03453919 0.5318543
	1227	> **ep1227a<- aov(area ~ trt, data=wf1227ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 42951 14317 19.86 1.03e-09 Residuals 80 57681 721 > **TukeyHSD(ep1227a)** diff lwr upr p adj p-c 1.786952 -19.816141 23.39004 0.9963607 ws-c -49.116194 -71.084436 -27.14795 0.0000006 wsp-c -33.838673 -58.046496 -9.63085 0.0024350 ws-p -50.903146 -70.846823 -30.95947 0.0000000 wsp-p -35.625624 -58.012346 -13.23890 0.0004302 wsp-ws 15.277522 -7.461769 38.01681 0.2987964	> **ep1227d<- aov(depth ~ trt, data=wf1227ep)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.7382 0.24608 16.12 2.75e-08 Residuals 80 1.2211 0.01526 > **TukeyHSD(ep1227d)** diff lwr upr p adj p-c 0.0007202562 -0.09867567 0.10011618 0.9999975 ws-c -0.2088633167 -0.30993929 -0.10778734 0.0000036 wsp-c -0.1403663437 -0.25174664 -0.02898605 0.0075803 ws-p -0.2095835728 -0.30134452 -0.11782263 0.0000003 wsp-p -0.1410865999 -0.24408800 -0.03808520 0.0030918 wsp-ws 0.0684969729 -0.03612660 0.17312055 0.3213005

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1614

```
> ep1614a<- aov(area ~ trt, data=wf1614ep)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   2.81   0.936   0.227  0.877
Residuals    76 312.85   4.116
```

```
> ep1614d<- aov(depth ~ trt, data=wf1614ep)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.006362 0.0021208   5.919 0.0011
Residuals    76 0.027230 0.0003583
> TukeyHSD(ep1614d)
                  diff          lwr          upr     p adj
p-c     0.0081082200 -0.007395062  0.023611502 0.5195219
ws-c   -0.0118950636 -0.027697472  0.003907345 0.2056416
wsp-c  -0.0127145717 -0.029798357  0.004369214 0.2142991
ws-p   -0.0200032836 -0.034679119 -0.005327448 0.0033089
wsp-p  -0.0208227917 -0.036870214 -0.004775370 0.0056647
wsp-ws -0.0008195081 -0.017156096  0.015517080 0.9991757
```

1797

```
> ep1797a<- aov(area ~ trt, data=wf1797ep)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  53554   17851   11.29 2.97e-06
Residuals    80 126492    1581
> TukeyHSD(ep1797a)
                diff        lwr         upr     p adj
p-c     -3.557919 -35.54917  28.4333296 0.9912837
ws-c   -59.266544 -91.79853 -26.7345599 0.0000458
wsp-c  -36.644043 -72.49254  -0.7955433 0.0432034
ws-p   -55.708625 -85.24250 -26.1747465 0.0000238
wsp-p  -33.086125 -66.23782   0.0655717 0.0506481
wsp-ws  22.622500 -11.05130  56.2963027 0.2988511
```

```
> ep1797d<- aov(depth ~ trt, data=wf1797ep)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.3044 0.10148   7.875 0.000114
Residuals    80 1.0308 0.01289
> TukeyHSD(ep1797d)
                 diff        lwr         upr     p adj
p-c    -0.02451103 -0.1158362  0.06681416 0.8951328
ws-c   -0.15212561 -0.2449944 -0.05925679 0.0002772
wsp-c  -0.08026065 -0.1825971  0.02207581 0.1759692
ws-p   -0.12761458 -0.2119247 -0.04330443 0.0008785
wsp-p  -0.05574962 -0.1503875  0.03888829 0.4154688
wsp-ws  0.07186496 -0.0242634  0.16799332 0.2112685
```

1021

```
> ep1021a<- aov(area ~ trt, data=wf1021ep)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  191.3   63.77   12.32 1.85e-06
Residuals    64  331.2    5.18
> TukeyHSD(ep1021a)
                diff        lwr        upr     p adj
p-c    -0.3816756 -2.221663  1.4583118 0.9469815
ws-c   -4.2057173 -6.344104 -2.0673310 0.0000137
wsp-c  -2.7740340 -5.140794 -0.4072741 0.0152279
ws-p   -3.8240417 -5.813298 -1.8347854 0.0000212
wsp-p  -2.3923584 -4.625293 -0.1594237 0.0311157
wsp-ws  1.4316833 -1.052897  3.9162636 0.431718
```

```
> ep1021d<- aov(depth ~ trt, data=wf1021ep)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.02232 0.007440   11.95 2.62e-06
Residuals    64 0.03985 0.000623
> TukeyHSD(ep1021d)
                  diff         lwr          upr     p adj
p-c    -0.002597725 -0.02278084  0.0175853931 0.9864056
ws-c   -0.045064919 -0.06852122 -0.0216086156 0.0000215
wsp-c  -0.027820168 -0.05378154 -0.0018587983 0.0310727
ws-p   -0.042467194 -0.06428767 -0.0206467216 0.0000168
wsp-p  -0.025222444 -0.04971586 -0.0007290242 0.0411698
wsp-ws  0.017244750 -0.01000901  0.0444985115 0.3483274
```

1227

```
> ep1227a<- aov(area ~ trt, data=wf1227ep)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  15769    5256   12.98 1.01e-06
Residuals    64  25926     405
> TukeyHSD(ep1227a)
                 diff        lwr        upr     p adj
p-c      0.09417521 -16.184741  16.373092 0.9999987
ws-c   -38.45177577 -57.370716 -19.532836 0.0000071
wsp-c  -10.79788456 -31.737314  10.141545 0.5286252
ws-p   -38.54595098 -56.145494 -20.946408 0.0000014
wsp-p  -10.89205977 -30.647497   8.863378 0.4708135
wsp-ws  27.65389121   5.672069  49.635713 0.0079576
```

```
> ep1227d<- aov(depth ~ trt, data=wf1227ep)
>            Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  0.240 0.08001   11.26 5.05e-06
Residuals    64  0.455 0.00711
> TukeyHSD(ep1227d)
                  diff         lwr         upr     p adj
p-c     0.003554412 -0.06464230  0.07175112 0.9990627
ws-c   -0.147606939 -0.22686341 -0.06835047 0.0000381
wsp-c  -0.019208704 -0.10692954  0.06851214 0.9384374
ws-p   -0.151161352 -0.22489052 -0.07743219 0.0000060
wsp-p  -0.022763117 -0.10552390  0.05999767 0.8865345
wsp-ws  0.128398235  0.03631054  0.22048593 0.0026622
```

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1614

```
> ep1614a<- aov(area ~ trt, data=wf1614ep)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   6.82   2.274    1.08  0.364
Residuals    60 126.31   2.105
```

```
> ep1614d<- aov(depth ~ trt, data=wf1614ep)
             Df   Sum Sq   Mean Sq F value   Pr(>F)
trt           3 0.004674 0.0015580   8.412 9.42e-05
Residuals    60 0.011113 0.0001852
> TukeyHSD(ep1614d)
              diff          lwr           upr     p adj
p-c     0.008569515 -0.002644207  0.0197832382 0.1923734
ws-c   -0.012472633 -0.025875595  0.0009303293 0.0770139
wsp-c  -0.010396234 -0.024580596  0.0037881284 0.2238706
ws-p   -0.021042148 -0.033757315 -0.0083269820 0.0002834
wsp-p  -0.018965749 -0.032502077 -0.0054294207 0.0025658
wsp-ws  0.002076399 -0.013322432  0.0174752307 0.9843460
```

1797

```
> ep1797a<- aov(area ~ trt, data=wf1797ep)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3  14802    4934   4.702 0.00498
Residuals    64  67159    1049
> TukeyHSD(ep1797a)
               diff        lwr         upr     p adj
p-c       6.309034 -19.891786 32.50985437 0.9202739
ws-c    -30.502117 -60.952039 -0.05219392 0.0494503
wsp-c    10.526122 -23.175766 44.22801113 0.8429569
ws-p    -36.811151 -65.137511 -8.48479014 0.0057480
wsp-p     4.217088 -27.579173 36.01334932 0.9851661
wsp-ws   41.028239   5.648626 76.40785222 0.0166773
```

```
> ep1797d<- aov(depth ~ trt, data=wf1797ep)
             Df Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.0548 0.018267   2.588 0.0606
Residuals    64 0.4517 0.007058
```

1021

```
> ep1021a<- aov(area ~ trt, data=wf1021ep)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3  53.62  17.873   5.414 0.00264
Residuals    50 165.07   3.301
> TukeyHSD(ep1021a)
               diff        lwr         upr     p adj
p-c      0.06146245 -1.419163  1.54208756 0.9995129
ws-c    -2.32264364 -4.598975 -0.04631249 0.0438973
wsp-c   -2.80282626 -5.472061 -0.13359139 0.0361621
ws-p    -2.38410609 -4.571134 -0.19707787 0.0276508
wsp-p   -2.86428872 -5.457785 -0.27079228 0.0250785
wsp-ws  -0.48018263 -3.597177  2.63681217 0.9765835
```

```
> ep1021d<- aov(depth ~ trt, data=wf1021ep)
             Df   Sum Sq   Mean Sq F value  Pr(>F)
trt           3 0.007363 0.0024545   5.643 0.00207
Residuals    50 0.021747 0.0004349
> TukeyHSD(ep1021d)
               diff          lwr          upr     p adj
p-c    -0.0000613903 -0.01705582  0.016933035 0.9999997
ws-c   -0.0288904284 -0.05501787 -0.002762991 0.0248491
wsp-c  -0.0318044840 -0.06244162 -0.001167348 0.0391150
ws-p   -0.0288290381 -0.05393147 -0.003726609 0.0184552
wsp-p  -0.0317430937 -0.06151091 -0.001975274 0.0324506
wsp-ws -0.0029140556 -0.03869052  0.032862411 0.9963707
```

1227

```
> ep1227a<- aov(area ~ trt, data=wf1227ep)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3   4835  1611.6   7.966 0.000194
Residuals    50  10115   202.3
> TukeyHSD(ep1227a)
               diff        lwr       upr     p adj
p-c       0.3479289 -11.24198 11.937834 0.9998154
ws-c    -21.3024163 -39.12088 -3.483955 0.0131726
wsp-c   -27.7827586 -48.67676 -6.888760 0.0047962
ws-p    -21.6503452 -38.76977 -4.530921 0.0078945
wsp-p   -28.1306875 -48.43183 -7.829548 0.0030863
wsp-ws   -6.4803423 -30.87928 17.918591 0.8943040
```

```
> ep1227d<- aov(depth ~ trt, data=wf1227ep)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.06006 0.020022   7.454 0.000321
Residuals    50 0.13430 0.002686
> TukeyHSD(ep1227d)
               diff          lwr         upr     p adj
p-c      0.0005569345 -0.04167544  0.04278931 0.9999843
ws-c    -0.0769881533 -0.14191672 -0.01205959 0.0141406
wsp-c   -0.0966666117 -0.17280211 -0.02053112 0.0076020
ws-p    -0.0775450878 -0.13992644 -0.01516374 0.0092816
wsp-p   -0.0972235462 -0.17119873 -0.02324836 0.0054056
wsp-ws  -0.0196784583 -0.10858556  0.06922864 0.9351829
```

	1614	> ep1614a<- aov(area ~ trt, data=wf1614ep) Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.789 0.5963 1.417 0.249 Residuals 48 20.199 0.4208	> ep1614d<- aov(depth ~ trt, data=wf1614ep) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.002054 0.0006846 7.85 0.000231 Residuals 48 0.004186 0.0000872 > TukeyHSD(ep1614d) diff lwr upr p adj p-c -0.001235321 -0.008799648 0.0063290069 0.9722043 ws-c -0.020925795 -0.034598295 -0.0072532959 0.0009644 wsp-c -0.014455294 -0.028127794 -0.0007827946 0.0345199 ws-p -0.019690475 -0.033074594 -0.0063063555 0.0015733 wsp-p -0.013219973 -0.026604093 0.0001641457 0.0540128 wsp-ws 0.006470501 -0.011103708 0.0240447108 0.7615029
	1797	> ep1797a<- aov(area ~ trt, data=wf1797ep) Df Sum Sq Mean Sq F value Pr(>F) trt 3 1537 512.5 0.962 0.418 Residuals 50 26647 532.9	> ep1797d<- aov(depth ~ trt, data=wf1797ep) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00125 0.0004152 0.259 0.855 Residuals 50 0.08021 0.0016043

Lomandra longifolia

		Area	Depth
28/06/12	1021	> l11021a<- aov(area ~ trt, data=wf10211l) Df Sum Sq Mean Sq F value Pr(>F) trt 3 53.24 17.746 5.495 0.00211 Residuals 60 193.75 3.229 > TukeyHSD(l11021a) diff lwr upr p adj p-c -0.6129416 -2.114582 0.8886991 0.7038008 ws-c -1.2249254 -3.064052 0.6142014 0.3025890 wsp-c -2.4667683 -4.121496 -0.8120409 0.0012065 ws-p -0.6119838 -2.451111 1.2271429 0.8155381 wsp-p -1.8538267 -3.508554 -0.1990993 0.0222114 wsp-ws -1.2418429 -3.207952 0.7242662 0.3488630	> l11021d<- aov(depth ~ trt, data=wf10211l) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.007403 0.0024677 6.878 0.000466 Residuals 60 0.021526 0.0003588 > TukeyHSD(l11021d) diff lwr upr p adj p-c -0.006632732 -0.02246055 0.009195087 0.6864361 ws-c -0.016551971 -0.03593701 0.002833068 0.1200748 wsp-c -0.028513443 -0.04595485 -0.011072037 0.0003398 ws-p -0.009919240 -0.02930428 0.009465800 0.5339346 wsp-p -0.021880712 -0.03932212 -0.004439305 0.0082584 wsp-ws -0.011961472 -0.03268495 0.008762007 0.4290606
	1227	> l11227a<- aov(area ~ trt, data=wf12271l) Df Sum Sq Mean Sq F value Pr(>F) trt 3 13255 4418 13.28 9.41e-07 Residuals 60 19967 333 > TukeyHSD(l11227a) diff lwr upr p adj p-c -4.691001 -19.93495 10.552946 0.8480042 ws-c -27.076646 -45.74659 -8.406700 0.0017019 wsp-c -34.575118 -51.37313 -17.777107 0.0000061 ws-p -22.385645 -41.05559 -3.715699 0.0125598 wsp-p -29.884117 -46.68213 -13.086106 0.0000903 wsp-ws -7.498472 -27.45748 12.460540 0.7540802	> l11227d<- aov(depth ~ trt, data=wf12271l) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.2032 0.06774 9.527 3.08e-05 Residuals 60 0.4266 0.00711 > TukeyHSD(l11227d) diff lwr upr p adj p-c -0.01268925 -0.08315048 0.05777198 0.9641164 ws-c -0.11064378 -0.19694081 -0.02434675 0.0066709 wsp-c -0.12955326 -0.20719776 -0.05190877 0.0002504 ws-p -0.09795453 -0.18425156 -0.01165750 0.0200037 wsp-p -0.11686401 -0.19450850 -0.03921951 0.0010662 wsp-ws -0.01890948 -0.11116488 0.07334593 0.9484350
	1614	> l11614a<- aov(area ~ trt, data=wf16141l) Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.03 1.3429 1.851 0.148 Residuals 60 43.54 0.7256	> l11614d<- aov(depth ~ trt, data=wf16141l) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000555 0.0001850 1.359 0.264 Residuals 60 0.008172 0.0001362

17/07/12	1797	> lll797a<- aov(area ~ trt, data=wf179711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 9913 3304 4.562 0.00605 Residuals 60 43460 724 > TukeyHSD(lll797a) diff lwr upr p adj p-c -3.448635 -25.93850 19.0412269 0.9773085 ws-c -26.274285 -53.81863 1.2700579 0.0668248 wsp-c -27.955630 -52.73825 -3.1730094 0.0210322 ws-p -22.825650 -50.36999 4.7186930 0.1377910 wsp-p -24.506995 -49.28962 0.2756257 0.0536826 wsp-ws -1.681345 -31.12749 27.7647962 0.9987620	> lll797d<- aov(depth ~ trt, data=wf179711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0675 0.02249 1.53 0.216 Residuals 60 0.8823 0.01470 > TukeyHSD(lll797d) diff lwr upr p adj p-c -0.007407881 -0.1087423 0.09392653 0.9974167 ws-c -0.076512075 -0.2006209 0.04759673 0.3703551 wsp-c -0.065612558 -0.1772776 0.04605252 0.4131652 ws-p -0.069104194 -0.1932130 0.05500461 0.4609396 wsp-p -0.058204677 -0.1698698 0.05346041 0.5182683 wsp-ws 0.010899517 -0.1217784 0.14357741 0.9963481
	1021	> wf1021llarea<- aov(area ~ trt, data=wf102111) Df Sum Sq Mean Sq F value Pr(>F) trt 3 5.3 1.766 1.045 0.378 Residuals 76 128.4 1.690	> wf1021lldepth<- aov(depth ~ trt, data=wf102111) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.001201 0.0004002 2.046 0.114 Residuals 76 0.014863 0.0001956
	1227	> wf1227lla<- aov(area ~ trt, data=wf122711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 146.6 48.85 1.652 0.184 Residuals 76 2246.8 29.56	> wf1227lldepth<- aov(depth ~ trt, data=wf122711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00256 0.0008543 2.042 0.115 Residuals 76 0.03179 0.0004183
	1614	> wf1614llarea<- aov(area ~ trt, data=wf161411) Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.167 0.3891 3.15 0.0298 Residuals 76 9.389 0.1235 > TukeyHSD(wf1614llarea) diff lwr upr p p-c 0.14454127 -0.14741628 0.4364988 0.5654945 ws-c -0.19197504 -0.48393259 0.09998250 0.3168932 wsp-c 0.02686824 -0.26508931 0.31882579 0.9949913 ws-p -0.33651631 -0.62847386 -0.04455877 0.017343 wsp-p -0.11767303 -0.40963058 0.17428452 0.715473 wsp-ws 0.21884328 -0.07311426 0.51080083 0.2088402	> wf1614lldepth<- aov(depth ~ trt, data=wf161411) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0001694 5.646e-05 2.916 0.0396 Residuals 76 0.0014713 1.936e-05 > TukeyHSD(wf1614lldepth) diff lwr upr p adj p-c 0.0004250764 -0.003229820 4.079973e-03 0.9900258 ws-c -0.0033293964 -0.006984293 3.255003e-04 0.0870920 wsp-c -0.0011086703 -0.004763567 2.546226e-03 0.8556681 ws-p -0.0037544728 -0.007409369 -9.957601e-05 0.0417913 wsp-p -0.0015337467 -0.005188643 2.121150e-03 0.6892112 wsp-ws 0.0022207260 -0.001434171 5.875623e-03 0.3869957
	1797	> wf1797llarea<- aov(area ~ trt, data=wf179711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 420 139.97 2.099 0.107 Residuals 76 5067 66.67	> wf1797lldepth<- aov(depth ~ trt, data=wf179711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00914 0.0030451 3.295 0.025 Residuals 76 0.07024 0.0009242 > TukeyHSD(wf1797lldepth) diff lwr upr p adj p-c -0.029289704 -0.054542541 -0.004036866 0.0164426 ws-c -0.008888412 -0.034141249 0.016364425 0.7917948 wsp-c -0.015490022 -0.040742860 0.009762815 0.3784953 ws-p 0.020401292 -0.004851546 0.045654129 0.1553876 wsp-p 0.013799681 -0.011453156 0.039052518 0.4814874 wsp-ws -0.006601611 -0.031854448 0.018651227 0.9018682
16/08/12	1021	> lll021a<- aov(area ~ trt, data=wf102111) Df Sum Sq Mean Sq F value Pr(>F) trt 3 13.31 4.437 2.302 0.0839 Residuals 76 146.50 1.928	> lll021d<- aov(depth ~ trt, data=wf102111) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.001385 0.0004616 2.341 0.0799 Residuals 76 0.014983 0.0001971
	1227	> lll227a<- aov(area ~ trt, data=wf122711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 425 141.65 2.587 0.0592 Residuals 76 4162 54.76	> lll227d<- aov(depth ~ trt, data=wf122711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00396 0.0013193 2.461 0.0691 Residuals 76 0.04075 0.0005361

	1614	> lll614a<- aov(area ~ trt, data=wf161411) Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.110 0.3701 2.952 0.0379 Residuals 76 9.529 0.1254 > TukeyHSD(lll614a) diff lwr upr p p-c 0.30119157 0.007058711 0.59532444 0.0427037 ws-c 0.06078217 -0.233350694 0.35491503 0.9481978 wsp-c 0.20050795 -0.093624914 0.49464081 0.2857677 ws-p -0.24040941 -0.534542269 0.05372346 0.1477749 wsp-p -0.10068363 -0.394816489 0.19344924 0.8052724 wsp-w 0.13972578 -0.154407084 0.43385864 0.5986601	> lll614d<- aov(depth ~ trt, data=wf161411) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0001166 3.885e-05 1.537 0.212 Residuals 76 0.0019206 2.527e-05
	1797	> ll797a<- aov(area ~ trt, data=wf179711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 115 38.31 0.585 0.627 Residuals 76 4980 65.53	> ll797d<- aov(depth ~ trt, data=wf179711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00040 0.0001322 0.149 0.93 Residuals 76 0.06729 0.0008853
30/08/12	1021	> lll021a<- aov(area ~ trt, data=wf102111) Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.33 1.442 1.093 0.357 Residuals 76 100.22 1.319	> lll021d<- aov(depth ~ trt, data=wf102111) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000316 0.0001052 0.729 0.538 Residuals 76 0.010957 0.0001442
	1227	> lll227a<- aov(area ~ trt, data=wf122711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 59.2 19.72 0.689 0.562 Residuals 76 2174.9 28.62	> lll227d<- aov(depth ~ trt, data=wf122711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000626 0.0002085 0.58 0.63 Residuals 76 0.027320 0.0003595
	1614	> lll614d<- aov(depth ~ trt, data=wf161411) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000034 1.130e-05 0.503 0.681 Residuals 156 0.003503 2.246e-05	> lll614a<- aov(area ~ trt, data=wf161411) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.46 0.1535 0.901 0.442 Residuals 156 26.59 0.1704
	1797	> lll797a<- aov(area ~ trt, data=wf179711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 83.7 27.89 0.778 0.51 Residuals 76 2723.7 35.84	> lll797d<- aov(depth ~ trt, data=wf179711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000759 0.0002530 0.857 0.467 Residuals 76 0.022429 0.0002951
13/09/12	1021	> lll021a<- aov(area ~ trt, data=wf102111) Df Sum Sq Mean Sq F value Pr(>F) trt 3 7.01 2.338 0.971 0.411 Residuals 76 182.92 2.407	> lll021d<- aov(depth ~ trt, data=wf102111) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000835 0.0002782 1.054 0.374 Residuals 76 0.020065 0.0002640
	1227	> lll227a<- aov(area ~ trt, data=wf122711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 105 35.05 0.277 0.842 Residuals 76 9627 126.67	> lll227d<- aov(depth ~ trt, data=wf122711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00178 0.0005948 0.3 0.825 Residuals 76 0.15046 0.0019798
	1614	> lll614a<- aov(area ~ trt, data=wf161411) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.825 0.2749 1.532 0.213 Residuals 76 13.632 0.1794	> lll614d<- aov(depth ~ trt, data=wf161411) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0000446 1.488e-05 0.418 0.741 Residuals 76 0.0027042 3.558e-05
	1797	> lll797a<- aov(area ~ trt, data=wf179711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 164 54.63 0.274 0.844 Residuals 76 15169 199.59	> lll797d<- aov(depth ~ trt, data=wf179711) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00417 0.001389 0.439 0.726 Residuals 76 0.24066 0.003167
27/09/12	1021	> lll021a<- aov(area ~ trt, data=wf102111) Df Sum Sq Mean Sq F value Pr(>F) trt 3 12.51 4.170 1.95 0.129 Residuals 76 162.51 2.138	> lll021d<- aov(depth ~ trt, data=wf102111) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.001686 0.0005618 2.369 0.0772 Residuals 76 0.018023 0.0002372

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1227

```
> lll227a<- aov(area ~ trt, data=wf122711)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3   4582  1527.2      17 1.53e-08
Residuals    76   6828    89.8
> TukeyHSD(lll227a)
               diff        lwr       upr     p adj
p-c     -3.869196 -11.742823  4.004430 0.5715573
ws-c   -17.861169 -25.734796 -9.987542 0.0000004
wsp-c  -15.609100 -23.482727 -7.735474 0.0000093
ws-p   -13.991972 -21.865599 -6.118346 0.0000747
wsp-p  -11.739904 -19.613531 -3.866277 0.0010960
wsp-ws   2.252068  -5.621558 10.125695 0.8758218
```

```
> lll227d<- aov(depth ~ trt, data=wf122711)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.06529 0.021762   18.88 3.01e-09
Residuals    76 0.08761 0.001153
> TukeyHSD(lll227d)
                diff         lwr         upr     p adj
p-c    -0.013575605 -0.04177820  0.01462699 0.5882012
ws-c   -0.065934691 -0.09413728 -0.03773210 0.0000002
wsp-c  -0.059967295 -0.08816989 -0.03176470 0.0000021
ws-p   -0.052359086 -0.08056168 -0.02415650 0.0000339
wsp-p  -0.046391690 -0.07459428 -0.01818910 0.0002672
wsp-ws  0.005967396 -0.02223519  0.03416999 0.9446913
```

1614

```
> lll614a<- aov(area ~ trt, data=wf161411)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3  3.347  1.1156   5.754 0.00133
Residuals    76 14.736  0.1939
> TukeyHSD(lll614a)
                diff        lwr         upr     p adj
p-c    -0.08312284 -0.4488920  0.28264633 0.9326917
ws-c   -0.51362460 -0.8793938 -0.14785543 0.0023356
wsp-c  -0.34188514 -0.7076543  0.02388403 0.0754678
ws-p   -0.43050176 -0.7962709 -0.06473259 0.0144770
wsp-p  -0.25876230 -0.6245315  0.10700687 0.2547143
wsp-ws  0.17173946 -0.1940297  0.53750863 0.6077813
```

```
> lll614d<- aov(depth ~ trt, data=wf161411)
             Df    Sum Sq   Mean Sq F value   Pr(>F)
trt           3 0.0005938 1.979e-04   9.251 2.74e-05
Residuals    76 0.0016260 2.139e-05
> TukeyHSD(lll614d)
                 diff          lwr           upr     p adj
p-c    -0.0019425422 -0.005784741  1.899657e-03 0.5481412
ws-c   -0.0058660835 -0.009708282 -2.023885e-03 0.0007960
wsp-c  -0.0065743446 -0.010416544 -2.732146e-03 0.0001421
ws-p   -0.0039235413 -0.007765740 -8.134229e-05 0.0435124
wsp-p  -0.0046318024 -0.008474001 -7.896034e-04 0.0116702
wsp-ws -0.0007082612 -0.004550460  3.133938e-03 0.9623795
```

1797

```
> lll797a<- aov(area ~ trt, data=wf179711)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3   3688  1229.3   19.59 1.66e-09
Residuals    76   4770    62.8
> TukeyHSD(lll797a)
               diff        lwr       upr     p adj
p-c     -3.335930  -9.916422  3.244561 0.5458960
ws-c   -15.423757 -22.004249 -8.843266 0.0000002
wsp-c  -14.633863 -21.214355 -8.053372 0.0000007
ws-p   -12.087827 -18.668319 -5.507335 0.0000412
wsp-p  -11.297933 -17.878425 -4.717441 0.0001344
wsp-ws   0.789894  -5.790598  7.370386 0.9890601
```

```
> lll797d<- aov(depth ~ trt, data=wf179711)
             Df  Sum Sq  Mean Sq F value  Pr(>F)
trt           3 0.01368 0.004560    7.68 0.00015
Residuals    76 0.04512 0.000594
> TukeyHSD(lll797d)
                diff         lwr           upr     p adj
p-c    -0.013315837 -0.03355658  0.0069249018 0.3164569
ws-c   -0.033378532 -0.05361927 -0.0131377922 0.0002570
wsp-c  -0.028178209 -0.04841895 -0.0079374697 0.0025885
ws-p   -0.020062694 -0.04030343  0.0001780453 0.0529309
wsp-p  -0.014862372 -0.03510311  0.0053783678 0.2247827
wsp-ws  0.005200323 -0.01504042  0.0254410618 0.9062857
```

1021

```
> lll021a<- aov(area ~ trt, data=wf102111)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3  70.64  23.548   7.887 0.00012
Residuals    76 226.90   2.986
> TukeyHSD(lll021a)
               diff        lwr       upr     p adj
p-c    -0.9090387 -2.344328  0.52625078 0.3499393
ws-c   -2.3743118 -3.809601 -0.93902232 0.0002448
wsp-c  -2.0338808 -3.469170 -0.59859129 0.0020927
ws-p   -1.4652731 -2.900563 -0.02998362 0.0435934
wsp-p  -1.1248421 -2.560132  0.31044741 0.1761597
wsp-ws  0.3404310 -1.094858  1.77572051 0.9243864
```

```
> lll021d<- aov(depth ~ trt, data=wf102111)
             Df   Sum Sq   Mean Sq F value   Pr(>F)
trt           3 0.008383 0.0027942   8.626 5.35e-05
Residuals    76 0.024617 0.0003239
> TukeyHSD(lll021d)
                diff         lwr          upr     p adj
p-c    -0.008473497 -0.02342346  0.006476462 0.4492060
ws-c   -0.025194445 -0.04014440 -0.010244486 0.0001822
wsp-c  -0.022201982 -0.03715194 -0.007252023 0.0011555
ws-p   -0.016720948 -0.03167091 -0.001770989 0.0222289
wsp-p  -0.013728485 -0.02867844  0.001221474 0.0833462
wsp-ws  0.002992463 -0.01195750  0.017942422 0.9525961
```

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1227

```
> lll227a<- aov(area ~ trt, data=wf122711)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  17122    5707   27.29 4.41e-12
Residuals    76  15894     209
> TukeyHSD(lll227a)
               diff        lwr        upr     p adj
p-c     -3.898269 -15.91068   8.114137 0.8290911
ws-c   -32.386823 -44.39923 -20.374416 0.0000000
wsp-c  -29.639217 -41.65162 -17.626810 0.0000000
ws-p   -28.488554 -40.50096 -16.476147 0.0000001
wsp-p  -25.740947 -37.75335 -13.728540 0.0000017
wsp-ws   2.747606  -9.26480  14.760013 0.9314917
```

```
> lll227d<- aov(depth ~ trt, data=wf122711)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.2411 0.08036   24.34 3.85e-11
Residuals    76 0.2509 0.00330
> TukeyHSD(lll227d)
                diff         lwr         upr     p adj
p-c    -0.015753148 -0.06348139  0.03197509 0.8218141
ws-c   -0.119379261 -0.16710750 -0.07165102 0.0000000
wsp-c  -0.114717670 -0.16244591 -0.06698943 0.0000001
ws-p   -0.103626112 -0.15135435 -0.05589787 0.0000013
wsp-p  -0.098964521 -0.14669276 -0.05123628 0.0000036
wsp-ws  0.004661591 -0.04306665  0.05238983 0.9940296
```

1614

```
> lll614a<- aov(area ~ trt, data=wf161411)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3   9.16  3.0532   6.157 0.000838
Residuals    76  37.69  0.4959
> TukeyHSD(lll614a)
               diff        lwr       upr     p adj
p-c     0.1371684 -0.4477733 0.7221100 0.9266868
ws-c   -0.7384694 -1.3234111 -0.1535278 0.0074984
wsp-c  -0.3387568 -0.9236984 0.2461848 0.4299986
ws-p   -0.8756378 -1.4605794 -0.2906962 0.0010397
wsp-p  -0.4759251 -1.0608668 0.1090165 0.1507323
wsp-ws  0.3997127 -0.1852290 0.9846543 0.2837161
```

```
> lll614d<- aov(depth ~ trt, data=wf161411)
             Df   Sum Sq   Mean Sq F value  Pr(>F)
trt           3 0.000769 2.562e-04   4.109 0.00932
Residuals    76 0.004740 6.236e-05
> TukeyHSD(lll614d)
                diff          lwr          upr     p adj
p-c     0.0007717492 -0.005788085  0.007331584 0.9896845
ws-c   -0.0071776808 -0.013737515 -0.000617847 0.0264147
wsp-c  -0.0021054477 -0.008665282  0.004454387 0.8336982
ws-p   -0.0079494300 -0.014509264 -0.001389596 0.0111195
wsp-p  -0.0028771969 -0.009437031  0.003682637 0.6585890
wsp-ws  0.0050722331 -0.001487601  0.011632067 0.1857694
```

1797

```
> lll797a<- aov(area ~ trt, data=wf179711)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  14057    4686   16.98 1.55e-08
Residuals    76  20974     276
> TukeyHSD(lll797a)
               diff        lwr       upr     p adj
p-c     -6.743007 -20.54236   7.056346 0.5761355
ws-c   -28.863821 -42.66317 -15.064468 0.0000030
wsp-c  -30.012766 -43.81212 -16.213412 0.0000012
ws-p   -22.120814 -35.92017  -8.321461 0.0003958
wsp-p  -23.269759 -37.06911  -9.470405 0.0001804
wsp-ws  -1.148944 -14.94830  12.650409 0.9962754
```

```
> lll797d<- aov(depth ~ trt, data=wf179711)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.05797 0.019324   12.48 1.02e-06
Residuals    76 0.11772 0.001549
> TukeyHSD(lll797d)
                diff         lwr          upr     p adj
p-c    -0.018229547 -0.05092154  0.014462449 0.4636853
ws-c   -0.057893836 -0.09058583 -0.025201839 0.0000794
wsp-c  -0.064468039 -0.09716004 -0.031776042 0.0000104
ws-p   -0.039664289 -0.07235629 -0.006972292 0.0109977
wsp-p  -0.046238492 -0.07893049 -0.013546495 0.0021415
wsp-ws -0.006574203 -0.03926620  0.026117794 0.9519793
```

1021

```
> lll021a<- aov(area ~ trt, data=wf102111)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3  53.24  17.746   5.495 0.00211
Residuals    60 193.75   3.229
> TukeyHSD(lll021a)
               diff        lwr        upr     p adj
p-c    -0.6129416 -2.114582  0.8886991 0.7038008
ws-c   -1.2249254 -3.064052  0.6142014 0.3025890
wsp-c  -2.4667683 -4.121496 -0.8120409 0.0012065
ws-p   -0.6119838 -2.451111  1.2271429 0.8155381
wsp-p  -1.8538267 -3.508554 -0.1990993 0.0222114
wsp-ws -1.2418429 -3.207952  0.7242662 0.3488630
```

```
> lll021d<- aov(depth ~ trt, data=wf102111)
             Df   Sum Sq   Mean Sq F value   Pr(>F)
trt           3 0.007403 0.0024677   6.878 0.000466
Residuals    60 0.021526 0.0003588
> TukeyHSD(lll021d)
                diff         lwr          upr     p adj
p-c    -0.006632732 -0.02246055  0.009195087 0.6864361
ws-c   -0.016551971 -0.03593701  0.002833068 0.1200748
wsp-c  -0.028513443 -0.04595485 -0.011072037 0.0003398
ws-p   -0.009919240 -0.02930428  0.009465800 0.5339346
wsp-p  -0.021880712 -0.03932212 -0.004439305 0.0082584
wsp-ws -0.011961472 -0.03268495  0.008762007 0.4290606
```

1227

```
> ll1227a<- aov(area ~ trt, data=wf122711)
            Df Sum Sq Mean Sq F value   Pr(>F)
trt          3  13255    4418   13.28 9.41e-07
Residuals   60  19967     333
> TukeyHSD(ll1227a)
              diff       lwr        upr     p adj
p-c     -4.691001 -19.93495  10.552946 0.8480042
ws-c   -27.076646 -45.74659  -8.406700 0.0017019
wsp-c  -34.575118 -51.37313 -17.777107 0.0000061
ws-p   -22.385645 -41.05559  -3.715699 0.0125598
wsp-p  -29.884117 -46.68213 -13.086106 0.0000903
wsp-ws  -7.498472 -27.45748  12.460540 0.7540802
```

```
> ll1227d<- aov(depth ~ trt, data=wf122711)
            Df Sum Sq Mean Sq F value   Pr(>F)
trt          3 0.2032 0.06774   9.527 3.08e-05
Residuals   60 0.4266 0.00711
> TukeyHSD(ll1227d)
               diff         lwr         upr     p adj
p-c    -0.01268925 -0.08315048  0.05777198 0.9641164
ws-c   -0.11064378 -0.19694081 -0.02434675 0.0066709
wsp-c  -0.12955326 -0.20719776 -0.05190877 0.0002504
ws-p   -0.09795453 -0.18425156 -0.01165750 0.0200037
wsp-p  -0.11686401 -0.19450850 -0.03921951 0.0010662
wsp-ws -0.01890948 -0.11116488  0.07334593 0.9484350
```

1614

```
> ll1614a<- aov(area ~ trt, data=wf161411)
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3   4.03  1.3429   1.851  0.148
Residuals   60  43.54  0.7256
```

```
> ll1614d<- aov(depth ~ trt, data=wf161411)
            Df   Sum Sq   Mean Sq F value Pr(>F)
trt          3 0.000555 0.0001850   1.359  0.264
Residuals   60 0.008172 0.0001362
```

1797

```
> ll1797a<- aov(area ~ trt, data=wf179711)
            Df Sum Sq Mean Sq F value  Pr(>F)
trt          3   9913    3304   4.562 0.00605
Residuals   60  43460     724
> TukeyHSD(ll1797a)
              diff       lwr        upr     p adj
p-c     -3.448635 -25.93850 19.0412269 0.9773085
ws-c   -26.274285 -53.81863  1.2700579 0.0668248
wsp-c  -27.955630 -52.73825 -3.1730094 0.0210322
ws-p   -22.825650 -50.36999  4.7186930 0.1377910
wsp-p  -24.506995 -49.28962  0.2756257 0.0536826
wsp-ws  -1.681345 -31.12749 27.7647962 0.9987620
```

```
> ll1797d<- aov(depth ~ trt, data=wf179711)
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3 0.0675 0.02249    1.53  0.216
Residuals   60 0.8823 0.01470
> TukeyHSD(ll1797d)
               diff         lwr         upr     p adj
p-c    -0.007407881 -0.1087423 0.09392653 0.9974167
ws-c   -0.076512075 -0.2006209 0.04759673 0.3703551
wsp-c  -0.065612558 -0.1772776 0.04605252 0.4131652
ws-p   -0.069104194 -0.1932130 0.05500461 0.4609396
wsp-p  -0.058204677 -0.1698698 0.05346041 0.5182683
wsp-ws  0.010899517 -0.1217784 0.14357741 0.9963481
```

Appendix 8.7: ANOVA output for vegetation indicies.

Treatments are as follows: c: control; w: water stress; p:inoculation; wsp: combination treatment.

Angophora costata

acred

28/06/12

```
> acred<- aov(Red ~ trt, data=AC)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.000175 5.818e-05   1.162  0.328
Residuals   107 0.005357 5.007e-05
```

17/07/12

```
> acred<- aov(red ~ trt, data=AC)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.000627 2.089e-04   2.418 0.0701
Residuals   112 0.009680 8.643e-05
```

1/08/12

```
> acred<- aov(red ~ trt, data=AC)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.001175 0.0003917   2.035  0.113
Residuals   108 0.020788 0.0001925
```

16/08/12

```
> acred<- aov(red~ trt, data=AC)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00048 0.0001589   0.414  0.743
Residuals   108 0.04140 0.0003833
```

30/08/12

```
> acred<- aov(red ~ trt, data=AC)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.01286 0.004285   2.985 0.0345
Residuals   106 0.15218 0.001436
> TukeyHSD(acred)
                 diff          lwr          upr     p adj
p-c     0.0036677637 -0.024328530 0.0316640570 0.9861697
ws-c    0.0253938328 -0.001691767 0.0524794326 0.0745204
wsp-c   0.0003472666 -0.026738333 0.0274328664 0.9999864
ws-p    0.0217260691 -0.004774460 0.0482265978 0.1472716
wsp-p  -0.0033204971 -0.029821026 0.0231800316 0.9878542
wsp-ws -0.0250465662 -0.050583115 0.0004899822 0.0566726
```

13/09/12

```
> acred<- aov(red ~ trt, data=AC)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.003347 0.0011157   5.196 0.0022
Residuals   104 0.022330 0.0002147
> TukeyHSD(acred)
                 diff          lwr          upr     p
p-c     0.00552304 -0.005521633  0.016567731 0.5614137
ws-c    0.01242446  0.001781546  0.023067390 0.0152436
wsp-c  -0.00136302 -0.011694380  0.008968328 0.9858703
ws-p    0.00690141 -0.003741503  0.017544341 0.3324945
wsp-p  -0.00688607 -0.017217429  0.003445279 0.3082988
wsp-ws -0.01378749 -0.023688184 -0.003886804 0.0024083
```

27/09/12

```
> acred<- aov(red ~ trt, data=AC)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.04221 0.014069   8.979 2.42e-05
Residuals   104 0.16296 0.001567
> TukeyHSD(acred)
                 diff          lwr         upr     p adj
p-c     0.001329407 -0.027927861 0.03058668 0.9993991
ws-c    0.022671022 -0.006080319 0.05142236 0.1735993
wsp-c   0.047910843  0.019605286 0.07621640 0.0001416
ws-p    0.021341615 -0.006807987 0.04949122 0.2023498
wsp-p   0.046581436  0.018887302 0.07427557 0.0001575
wsp-ws  0.025239821 -0.001919283 0.05239892 0.0783639
```

11/10/12

```
> acred<- aov(red ~ trt, data=AC)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.1218 0.04062   11.96 1.28e-06
Residuals    86 0.2920 0.00340
> TukeyHSD(acred)
                 diff          lwr          upr     p adj
p-c     0.002938449 -0.04027679  0.04615369 0.9979767
ws-c    0.090793576  0.04573202  0.13585513 0.0000057
wsp-c   0.029174420 -0.01842778  0.07677662 0.3808591
ws-p    0.087855128  0.04363023  0.13208002 0.0000077
wsp-p   0.026235971 -0.02057500  0.07304694 0.4609258
wsp-ws -0.061619157 -0.11013981 -0.01309851 0.0069507
```

25/10/12

```
> acred<- aov(red ~ trt, data=AC)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.000466 0.0001554   0.329  0.805
Residuals    56 0.026474 0.0004728
```

green

28/06/12

```
> acgreen<- aov(Green ~ trt, data=AC)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00078 0.0002584   0.515  0.673
Residuals   107 0.05372 0.0005020
```

17/07/12

```
> acgrn<- aov(green ~ trt, data=AC)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00170 0.0005656    0.94  0.424
Residuals   112 0.06738 0.0006016
```

1/08/12

```
> acgrn<- aov(green ~ trt, data=AC)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00222 0.0007401   1.292  0.281
Residuals   108 0.06188 0.0005730
```

16/08/12

```
> acgrn<- aov(green~ trt, data=AC)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.00578 0.001928   1.904  0.133
Residuals   108 0.10933 0.001012
```

30/08/12

```
> acgrn<- aov(green ~ trt, data=AC)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.00717 0.002391   2.069  0.109
Residuals   106 0.12249 0.001156
```

13/09/12

```
> acgrn<- aov(green ~ trt, data=AC)
             Df  Sum Sq   Mean Sq F value  Pr(>F)
trt           3 0.00690 0.0022989   4.126 0.00828
Residuals   104 0.05794 0.0005571
> TukeyHSD(acgrn)
                 diff           lwr         upr     p
p-c    0.020304207  0.0025132300 0.038095183 0.0185504
ws-c   0.016419815 -0.0007239987 0.033563629 0.0656566
wsp-c  0.004996437 -0.0116454974 0.021638372 0.8615979
ws-p  -0.003884391 -0.0210282053 0.013259423 0.9344149
wsp-p -0.015307769 -0.0319497039 0.001334166 0.0830369
wsp-ws-0.011423378 -0.0273715910 0.004524835 0.2471999
```

27/09/12

```
> acgrn<- aov(green ~ trt, data=AC)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.05863 0.019542   11.31 1.75e-06
Residuals   104 0.17966 0.001727
> TukeyHSD(acgrn)
                 diff         lwr        upr     p adj
p-c    0.0172890614 -0.01343042 0.04800854 0.4595381
ws-c   0.0182464042 -0.01194186 0.04843467 0.3955023
wsp-c  0.0619388699  0.03221867 0.09165907 0.0000021
ws-p   0.0009573428 -0.02859911 0.03051380 0.9997818
wsp-p  0.0446498085  0.01557158 0.07372803 0.0006556
wsp-ws 0.0436924657  0.01517601 0.07220892 0.0006764
```

11/10/12

```
> acgrn<- aov(green ~ trt, data=AC)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.07032 0.023441   12.24 9.59e-07
Residuals    86 0.16469 0.001915
> TukeyHSD(acgrn)
                 diff          lwr        upr     p adj
p-c     0.009558129 -0.0228960304 0.04201229 0.8670468
ws-c    0.069676578  0.0358358576 0.10351730 0.0000035
wsp-c   0.043771451  0.0080227373 0.07952017 0.0099668
ws-p    0.060118450  0.0269060503 0.09333085 0.0000486
wsp-p   0.034213323 -0.0009411884 0.06936783 0.0594335
wsp-ws -0.025905127 -0.0623435905 0.01053334 0.2518433
```

25/10/12

```
> acgrn<- aov(green ~ trt, data=AC)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00318 0.0010604   1.237  0.305
Residuals    56 0.04801 0.0008573
```

NIR

28/06/12

```
> acnir<- aov(NIR ~ trt, data=AC)
             Df  Sum Sq  Mean Sq F value  Pr(>F)
trt           3 0.02753 0.009178   4.085 0.00866
Residuals   107 0.24037 0.002246
> TukeyHSD(acnir)
                 diff          lwr        upr     p adj
p-c     0.032747361 -0.002661939 0.06815666 0.0805858
ws-c    0.030699737 -0.003115492 0.06451497 0.0892854
wsp-c   0.045064809  0.010781457 0.07934816 0.0046878
ws-p   -0.002047625 -0.034708079 0.03061283 0.9984318
wsp-p   0.012317448 -0.020827444 0.04546234 0.7668241
wsp-ws  0.014365072 -0.017071133 0.04580128 0.6328417
```

17/07/12

```
> acnir<- aov(NIR ~ trt, data=AC)
             Df Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.0268 0.008936   2.584 0.0568
Residuals   112 0.3873 0.003458
```

	1/08/12	16/08/12
	> acnir<- aov(NIR ~ trt, data=AC) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.184 0.06137 1.846 0.143 Residuals 108 3.590 0.03324	**> acnir<- aov(NIR~ trt, data=AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.134 0.04453 1.383 0.252 Residuals 108 3.478 0.03221
	30/08/12	13/09/12
	> acnir<- aov(NIR ~ trt, data=AC) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0718 0.02394 0.858 0.465 Residuals 106 2.9568 0.02789	**> acnir<- aov(NIR ~ trt, data=AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1127 0.03758 1.46 0.23 Residuals 104 2.6773 0.02574
	27/09/12	11/10/12
	> acnir<- aov(NIR ~ trt, data=AC) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0339 0.01131 0.523 0.668 Residuals 104 2.2498 0.02163	**> acnir<- aov(NIR ~ trt, data=AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0427 0.01424 0.564 0.64 Residuals 86 2.1729 0.02527
	25/10/12	
	> acnir<- aov(NIR ~ trt, data=AC) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.2338 0.07793 3.429 0.023 Residuals 56 1.2726 0.02272 **> TukeyHSD(acnir)** diff lwr upr p adj p-c 0.12425454 0.01126427 0.2372448 0.0257492 ws-c 0.07909483 -0.08386205 0.2420517 0.5760648 wsp-c 0.20985595 -0.08391875 0.5036306 0.2433401 ws-p -0.04515970 -0.20654209 0.1162227 0.8801012 wsp-p 0.08560141 -0.20730284 0.3785057 0.8659294 wsp-ws 0.13076111 -0.18480353 0.4463258 0.6926925	
NDVI	28/06/12	17/07/12
	> acndvi<- aov(NDVI ~ trt, data=AC) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000351 0.0001170 0.413 0.744 Residuals 107 0.030343 0.0002836	**> acndvi<- aov(NDVI ~ trt, data=AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00361 0.0012043 2.043 0.112 Residuals 112 0.06603 0.0005895
	1/08/12	16/08/12
	> acndvi<- aov(NDVI ~ trt, data=AC) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0036 0.001200 0.305 0.821 Residuals 108 0.4246 0.003931	**> acndvi<- aov(NDVI~ trt, data=AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0143 0.004778 1.16 0.329 Residuals 108 0.4448 0.004119
	30/08/12	13/09/12
	> acndvi<- aov(NDVI ~ trt, data=AC) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0330 0.010993 1.45 0.232 Residuals 106 0.8036 0.007581	**> acndvi<- aov(NDVI ~ trt, data=AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00799 0.002662 1.112 0.348 Residuals 104 0.24905 0.002395
	27/09/12	11/10/12

```
> acndvi<- aov(NDVI ~ trt, data=AC)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.2471 0.08237   9.808 9.39e-06
Residuals   104 0.8735 0.00840
> TukeyHSD(acndvi)
                diff         lwr          upr     p adj
p-c    -0.001299072 -0.06903502  0.066436875 0.9999546
ws-c   -0.055525716 -0.12209035  0.011038915 0.1361841
wsp-c  -0.114769962 -0.18030252 -0.049237403 0.0000778
ws-p   -0.054226644 -0.11939814  0.010944851 0.1377594
wsp-p  -0.113470890 -0.17758789 -0.049353888 0.0000644
wsp-ws -0.059244245 -0.12212255  0.003634063 0.0723800
```

```
> acndvi<- aov(NDVI ~ trt, data=AC)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3 0.4931 0.16437   12.77 5.6e-07
Residuals    86 1.1071 0.01287
> TukeyHSD(acndvi)
                diff          lwr          upr     p ad
p-c    -0.0006193923 -0.084766011  0.08352723 0.9999974
ws-c   -0.1780362950 -0.265777969 -0.09029462 0.0000049
wsp-c  -0.0817411625 -0.174429850  0.01094752 0.1035853
ws-p   -0.1774169027 -0.263529475 -0.09130433 0.0000035
wsp-p  -0.0811217702 -0.172269818  0.01002628 0.0988153
wsp-ws  0.0962951325  0.001818074  0.19077219 0.0440395
```

25/10/12

```
> acndvi<- aov(NDVI ~ trt, data=AC)
             Df Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.0344 0.011480   1.277  0.291
Residuals    56 0.5033 0.008988
```

28/06/12

```
> acrep<- aov(REP ~ trt, data=AC)
              Df    Sum Sq   Mean Sq F value Pr(>F)
trt            3 2.710e-07 9.042e-08    1.43  0.238
Residuals    107 6.766e-06 6.323e-08
```

17/07/12

```
> acrep<- aov(REP ~ trt, data=AC)
              Df    Sum Sq   Mean Sq F value Pr(>F)
trt            3 2.970e-07 9.900e-08    0.53  0.663
Residuals    112 2.092e-05 1.868e-07
```

1/08/12

```
> acrep<- aov(REP ~ trt, data=AC)
              Df    Sum Sq   Mean Sq F value Pr(>F)
trt            3 1.630e-07 5.418e-08   0.362  0.781
Residuals    108 1.616e-05 1.496e-07
```

16/08/12

```
> acrep<- aov(REP~ trt, data=AC)
              Df   Sum Sq   Mean Sq F value Pr(>F)
trt            3 2.60e-07 8.676e-08   0.501  0.682
Residuals    108 1.87e-05 1.731e-07
```

30/08/12

```
> acrep<- aov(REP ~ trt, data=AC)
              Df    Sum Sq   Mean Sq F value Pr(>F)
trt            3 2.199e-06 7.332e-07   2.533 0.0609
Residuals    106 3.069e-05 2.895e-07
```

13/09/12

```
> acrep<- aov(REP ~ trt, data=AC)
              Df    Sum Sq   Mean Sq F value Pr(>F)
trt            3 1.118e-06 3.727e-07   1.941  0.128
Residuals    104 1.997e-05 1.920e-07
```

27/09/12

```
> acrep<- aov(REP ~ trt, data=AC)
              Df    Sum Sq   Mean Sq F value Pr(>F)
trt            3 5.640e-06 1.879e-06   3.752 0.0132
Residuals    104 5.207e-05 5.007e-07
> TukeyHSD(acrep)
                 diff          lwr          upr     p adj
p-c     1.015555e-04 -4.214322e-04 0.0006245431 0.9572502
ws-c    9.547677e-05 -4.184672e-04 0.0006094207 0.9622591
wsp-c   5.707890e-04  6.481360e-05 0.0010767643 0.0204493
ws-p   -6.078701e-06 -5.092663e-04 0.0004971089 0.9999887
wsp-p   4.692335e-04 -2.581239e-05 0.0009642794 0.0698744
wsp-ws  4.753122e-04 -1.016976e-05 0.0009607941 0.0573159
```

11/10/12

```
> acrep<- aov(REP ~ trt, data=AC)
> summary(acrep)
              Df    Sum Sq   Mean Sq F value   Pr(>F)
trt            3 1.918e-05 6.395e-06   9.795 1.26e-05
Residuals     86 5.615e-05 6.530e-07
> TukeyHSD(acrep)
                 diff           lwr          upr     p
p-c    0.0003260098 -2.732468e-04 0.0009252663 0.487186
ws-c   0.0011454747  5.206157e-04 0.0017703337 0.00008
wsp-c  0.0009469092  2.868196e-04 0.001606998 0.001739
ws-p   0.0008194649  2.062077e-04 0.001432722 0.0040385
wsp-p  0.0006208994 -2.821838e-05 0.001270017 0.0660496
wsp-ws0.0001985655 -8.713911e-04 0.0004742601 0.866339
```

RES

25/10/12

```
> acrep<- aov(REP ~ trt, data=AC)
             Df    Sum Sq   Mean Sq F value   Pr(>F)
trt           3 6.877e-06 2.292e-06   6.234 0.000996
Residuals    56 2.059e-05 3.677e-07
> TukeyHSD(acrep)
                 diff           lwr          upr     p adj
p-c     0.0004524057 -2.125651e-06 0.0009069371 0.0515145
ws-c    0.0006109082 -4.462630e-05 0.0012664426 0.0762758
wsp-c   0.0016033224  4.215408e-04 0.0027851040 0.0037577
ws-p    0.0001585024 -4.906982e-04 0.0008077031 0.9163412
wsp-p   0.0011509167 -2.736330e-05 0.0023291967 0.0579176
wsp-ws  0.0009924142 -2.770228e-04 0.0022618513 0.1755415
```

aREP

28/06/12

```
> acrep<- aov(REP ~ trt, data=AC)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 2.710e-07 9.042e-08    1.43  0.238
Residuals   107 6.766e-06 6.323e-08
```

17/07/12

```
> acarep<- aov(aREP ~ trt, data=AC)
             Df Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.0196 0.006528   1.776  0.156
Residuals   112 0.4116 0.003675
```

1/08/12

```
> acaREP<- aov(aREP ~ trt, data=AC)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3  0.143 0.04761   1.604  0.193
Residuals   108  3.206 0.02968
```

16/08/12

```
> acarep<- aov(aREP~ trt, data=AC)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3 0.1294 0.04314   1.521  0.213
Residuals   108 3.0625 0.02836
```

30/08/12

```
> acarep<- aov(aREP ~ trt, data=AC)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3 0.0378 0.01260   0.468  0.706
Residuals   106 2.8558 0.02694
```

13/09/12

```
> acaREP<- aov(aREP ~ trt, data=AC)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3 0.0638 0.02127   0.946  0.421
Residuals   104 2.3383 0.02248
```

27/09/12

```
> acarep<- aov(aREP ~ trt, data=AC)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3 0.1974 0.06582   2.906 0.0383
Residuals   104 2.3558 0.02265
> TukeyHSD(acarep)
                 diff         lwr          upr     p adj
p-c     0.02884491 -0.08239478  0.140084606 0.9055745
ws-c   -0.04635252 -0.15566862  0.062963575 0.6859590
wsp-c  -0.08129053 -0.18891170  0.026330636 0.2051699
ws-p   -0.07519743 -0.18222564  0.031830779 0.2631716
wsp-p  -0.11013545 -0.21543191 -0.004838981 0.0366996
wsp-ws -0.03493801 -0.13820023  0.068324201 0.8134506
```

11/10/12

```
> acarep<- aov(aREP ~ trt, data=AC)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3 0.1784 0.05947   2.252 0.0881
Residuals    86 2.2710 0.02641
```

25/10/12

```
> acarep<- aov(aREP ~ trt, data=AC)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3 0.1798 0.05992   2.553 0.0646
Residuals    56 1.3141 0.02347
```

ARI

28/06/12

```
> acari<- aov(ARI ~ trt, data=AC)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3  0.886  0.2953   1.165  0.327
Residuals   107 27.124  0.2535
```

17/07/12

```
> acari<- aov(ARI ~ trt, data=AC)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 1.060e+13 3.533e+12   1.541  0.208
Residuals   112 2.567e+14 2.292e+12
```

1/08/12

```
> acari<- aov(ARI ~ trt, data=AC)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   1.03  0.3432   0.185  0.906
Residuals   108 200.39  1.8555
```

16/08/12

```
> acari<- aov(ARI~ trt, data=AC)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 1.296e+12 4.320e+11   1.265   0.29
Residuals   108 3.688e+13 3.415e+11
```

	30/08/12	13/09/12
	> acari<- aov(ARI ~ trt, data=AC) Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.24 1.413 0.547 0.651 Residuals 106 273.77 2.583	**> acARI<- aov(ARI ~ trt, data=AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.23 0.411 0.216 0.885 Residuals 104 197.64 1.900
	27/09/12	11/10/12
	> acari<- aov(ARI ~ trt, data=AC) Df Sum Sq Mean Sq F value Pr(>F) trt 3 9.36 3.121 1.896 0.135 Residuals 104 171.20 1.646	**> acari<- aov(ARI ~ trt, data=AC)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 5.29 1.764 0.6 0.616 Residuals 86 252.58 2.937
	25/10/12	
	> acari<- aov(ARI ~ trt, data=AC) Df Sum Sq Mean Sq F value Pr(>F) trt 3 79.3 26.438 4.454 0.00709 Residuals 56 332.4 5.936 **> TukeyHSD(acari)** diff lwr upr p adj p-c 2.3927182 0.5666205 4.218816 0.0054284 ws-c 1.8945678 -0.7390677 4.528203 0.2377405 wsp-c 2.9339006 -1.8139534 7.681754 0.3670428 ws-p -0.4981504 -3.1063396 2.110039 0.9573916 wsp-p 0.5411824 -4.1926038 5.274969 0.9902612 wsp-ws 1.0393328 -4.0606804 6.139346 0.9489413	

Banksia serrata

red	28/06/12	17/07/12
	> bsred<- aov(Red ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.002482 0.0008273 7.285 0.000183 Residuals 99 0.011244 0.0001136 **> TukeyHSD(bsred)** diff lwr upr p adj p-c -0.0006613613 -0.008462114 0.007139391 0.9961382 ws-c -0.0008600534 -0.008660806 0.006940699 0.9916172 wsp-c 0.0107630764 0.002962324 0.018563829 0.0027186 ws-p -0.0001986920 -0.007922588 0.007525204 0.9998903 wsp-p 0.0114244377 0.003700542 0.019148334 0.0011227 wsp-ws 0.0116231297 0.003899234 0.019347026 0.0008865	**> bsred<- aov(red ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000242 8.059e-05 0.949 0.42 Residuals 100 0.008491 8.491e-05
	1/08/12	16/08/12
	> bsred<- aov(red ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00225 0.0007512 0.946 0.421 Residuals 100 0.07939 0.0007939	**> bsred<- aov(red~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000574 0.0001913 1.681 0.176 Residuals 100 0.011383 0.0001138
	30/08/12	13/09/12
	> bsred<- aov(red ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000919 0.0003064 1.764 0.159 Residuals 99 0.017194 0.0001737	**> bsred<- aov(red ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0035 0.001168 2.827 0.0424 Residuals 100 0.0413 0.000413 **> TukeyHSD(bsred)** diff lwr upr p adj p-c -0.003525384 -0.018252678 0.011201911 0.9236782 ws-c -0.011416385 -0.026143680 0.003310909 0.1856284 wsp-c 0.004429041 -0.010298253 0.019156336 0.8607640 ws-p -0.007891001 -0.022618296 0.006836293 0.5023701 wsp-p 0.007954425 -0.006772869 0.022681720 0.4953956

		wsp-ws 0.015845427 0.001118132 0.030572721 0.0298122
	27/09/12	11/10/12
	> bsred<- aov(red ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0231 0.007694 2.165 0.0968 Residuals 100 0.3553 0.003553	> bsred<- aov(red ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.2180 0.07266 11.71 1.65e-06 Residuals 86 0.5334 0.00620 > TukeyHSD(bsred) diff lwr upr p adj p-c 0.009100254 -0.048128728 0.06632924 0.9754949 ws-c 0.069399933 0.006130913 0.13266895 0.0258030 wsp-c 0.122852132 0.061480886 0.18422338 0.0000065 ws-p 0.060299679 -0.002969341 0.12356870 0.0674952 wsp-p 0.113751879 0.052380632 0.17512312 0.0000312 wsp-ws 0.053452200 -0.013586911 0.12049131 0.1649166
	25/10/12	
	> bsred<- aov(red ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00339 0.001130 0.891 0.451 Residuals 60 0.07610 0.001268	
	28/06/12	17/07/12
	> bsgrn<- aov(Green ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00602 0.0020081 3.436 0.0198 Residuals 99 0.05786 0.0005844 > TukeyHSD(bsgrn) diff lwr upr p adj p-c -0.005634369 -0.023330245 0.012061507 0.8391127 ws-c -0.014605811 -0.032301687 0.003090065 0.1426783 wsp-c 0.006094273 -0.011601603 0.023790149 0.8048543 ws-p -0.008971443 -0.026492971 0.008550085 0.5411301 wsp-p 0.011728641 -0.005792887 0.029250170 0.3041247 wsp-ws 0.020700084 0.003178556 0.038221612 0.0137400	> bsgrn<- aov(green ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00506 0.0016851 2.596 0.0566 Residuals 100 0.06491 0.0006491
	1/08/12	16/08/12
	> bsgrn<- aov(green ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00865 0.002883 1.943 0.128 Residuals 100 0.14843 0.001484	> bsgrn<- aov(green~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00933 0.0031099 3.47 0.019 Residuals 100 0.08963 0.0008963 > TukeyHSD(bsgrn) diff lwr upr p ad p-c -0.016765791 -0.038460727 0.004929145 0.1878803 ws-c -0.024913627 -0.046608563 -0.003218691 0.0176267 wsp-c -0.006953910 -0.028648846 0.014741026 0.8364826 ws-p -0.008147837 -0.029842773 0.013547099 0.7604270 wsp-p 0.009811881 -0.011883055 0.031506817 0.6397967 wsp-ws 0.017959718 -0.003735218 0.039654654 0.1408318
	30/08/12	13/09/12
	> bsrgn<- aov(green ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01516 0.005054 6.37 0.000542 Residuals 99 0.07854 0.000793 > TukeyHSD(bsrgn) diff lwr upr p ad p-c -0.0003343426 -0.020951949 2.028326e-02 0.9999725 ws-c -0.0208179764 -0.041435583 -2.003700e-04 0.0468916 wsp-c 0.0129148869 -0.007702719 3.353249e-02 0.3628692 ws-p -0.0204836337 -0.040898106 -6.916146e-05 0.0488970	> bsgrn<- aov(green ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.03721 0.012404 6.073 0.00077 Residuals 100 0.20425 0.002042 > TukeyHSD(bsgrn) diff lwr upr p ad p-c -0.007978047 -0.04072781 2.477172e-02 0.9199780 ws-c -0.040652906 -0.07340267 -7.903142e-03 0.0085983 wsp-c 0.009693515 -0.02305625 4.244328e-02 0.8663168 ws-p -0.032674859 -0.06542462 7.490515e-05 0.0507569

green

	wsp-p 0.0132492295 -0.007165243 3.366370e-02 0.3312629 wsp-ws 0.0337328633 0.013318391 5.414734e-02 0.0002165	wsp-p 0.017671562 -0.01507820 5.042133e-02 0.4962379 wsp-ws 0.050346421 0.01759666 8.309618e-02 0.0006529
	27/09/12	11/10/12
	> bsgrn<- aov(green ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0039 0.001296 0.332 0.802 Residuals 100 0.3899 0.003899	**> bsgrn<- aov(green ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0903 0.030103 5.12 0.00263 Residuals 86 0.5056 0.005879 **> TukeyHSD(bsgrn)** diff lwr upr p adj p-c 0.04459227 -0.011124682 0.10030923 0.1623486 ws-c 0.01942089 -0.042176517 0.08101831 0.8419768 wsp-c 0.08587417 0.026124396 0.14562395 0.0016980 ws-p -0.02517138 -0.086768790 0.03642603 0.7082513 wsp-p 0.04128190 -0.018467877 0.10103168 0.2756474 wsp-ws 0.06645328 0.001185385 0.13172117 0.0443574
	25/10/12	
	> bsgrn<- aov(green ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00583 0.001945 0.689 0.562 Residuals 60 0.16938 0.002823	
	28/06/12	17/07/12
	> bsnir<- aov(NIR ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0057 0.001895 0.421 0.739 Residuals 99 0.4460 0.004505	**> bsnir<- aov(NIR ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0580 0.019324 4.369 0.0062 Residuals 100 0.4423 0.004423 **> TukeyHSD(bsnir)** diff lwr upr p adj p-c 0.066678542 0.01848443 0.11487265 0.0026255 ws-c 0.034613444 -0.01358066 0.08280755 0.2447169 wsp-c 0.031043519 -0.01715059 0.07923763 0.3380490 ws-p -0.032065099 -0.08025921 0.01612901 0.3095095 wsp-p -0.035635023 -0.08382913 0.01255909 0.2214222 wsp-ws -0.003569925 -0.05176403 0.04462418 0.9974144
	1/08/12	16/08/12
	> bsnir<- aov(NIR ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.030 0.00996 0.297 0.828 Residuals 100 3.357 0.03357	**> bsnir<- aov(NIR~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.044 0.01482 0.404 0.75 Residuals 100 3.669 0.03669
	30/08/12	13/09/12
	> bsnir<- aov(NIR ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.086 0.02866 0.918 0.435 Residuals 99 3.092 0.03123	**> bsnir<- aov(NIR ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1363 0.04543 1.658 0.181 Residuals 100 2.7408 0.02741
	27/09/12	11/10/12
	> bsNIR<- aov(NIR ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1822 0.06073 1.939 0.128 Residuals 100 3.1317 0.03132	**> bsnir<- aov(NIR ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.209 0.06965 2.54 0.0617 Residuals 86 2.358 0.02742
	25/10/12	
	> bsnir<- aov(NIR ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1464 0.04880 1.244 0.302 Residuals 60 2.3532 0.03922	
N_NIR	28/06/12	17/07/12

RES

```
> bsndvi<- aov(NDVI ~ trt, data=BS)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.01191 0.003971   6.729 0.000353
Residuals    99 0.05843 0.000590
> TukeyHSD(bsndvi)
                 diff         lwr          upr     p adj
p-c     0.0005318615 -0.01725045  0.018314175 0.9998276
ws-c    0.0030311182 -0.01475120  0.020813432 0.9703551
wsp-c  -0.0234138824 -0.04119620 -0.005631569 0.0046506
ws-p    0.0024992568 -0.01510786  0.020106371 0.9824805
wsp-p  -0.0239457438 -0.04155286 -0.006338630 0.0032230
wsp-ws -0.0264450006 -0.04405211 -0.008837887 0.0009103
```

1/08/12

```
> bsndvi<- aov(NDVI ~ trt, data=BS)
             Df Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.0059 0.001975   0.348  0.791
Residuals   100 0.5682 0.005682
```

30/08/12

```
> bsndvi<- aov(NDVI ~ trt, data=BS)
             Df Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.0229 0.007643    0.59  0.623
Residuals    99 1.2833 0.012963
```

27/09/12

```
> bsNDVI<- aov(NDVI ~ trt, data=BS)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3 0.0788 0.02625   2.003  0.118
Residuals   100 1.3105 0.01311
```

25/10/12

```
> bsndvi<- aov(NDVI ~ trt, data=BS)
             Df Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.0076 0.002535   0.149   0.93
Residuals    60 1.0236 0.017060
```

28/06/12

```
> bsrep<- aov(REP ~ trt, data=BS)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 7.080e-07 2.362e-07   3.539 0.0174
Residuals    99 6.606e-06 6.673e-08
> TukeyHSD(bsrep)
                  diff           lwr          upr     p adj
p-c    -1.214328e-05 -2.012255e-04 1.769389e-04 0.9983079
ws-c   -1.860457e-04 -3.751280e-04 3.036460e-06 0.0555221
wsp-c   2.078148e-05 -1.683007e-04 2.098637e-04 0.9916941
ws-p   -1.739025e-04 -3.611217e-04 1.331682e-05 0.0785316
wsp-p   3.292477e-05 -1.542945e-04 2.201441e-04 0.9676020
wsp-ws  2.068272e-04  1.960795e-05 3.940465e-04 0.0242860
```

1/08/12

```
> bsndvi<- aov(NDVI ~ trt, data=BS)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.00022 7.41e-05   0.173  0.915
Residuals   100 0.04290 4.29e-04
```

16/08/12

```
> bsnvi<- aov(NDVI~ trt, data=BS)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.01696 0.005654   2.497 0.0641
Residuals   100 0.22640 0.002264
```

13/09/12

```
> bsndvi<- aov(NDVI ~ trt, data=BS)
             Df Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.0289 0.009637    0.98  0.405
Residuals   100 0.9832 0.009832
```

11/10/12

```
> bsndvi<- aov(NDVI ~ trt, data=BS)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.7298 0.24325   13.14 3.87e-07
Residuals    86 1.5925 0.01852
> TukeyHSD(bsndvi)
                 diff        lwr         upr     p adj
p-c    -0.03219663 -0.1310794  0.06668610 0.8287939
ws-c   -0.16714442 -0.2764634 -0.05782545 0.0007438
wsp-c  -0.21752815 -0.3235681 -0.11148823 0.0000038
ws-p   -0.13494779 -0.2442668 -0.02562882 0.0092152
wsp-p  -0.18533152 -0.2913714 -0.07929160 0.0000911
wsp-ws -0.05038373 -0.1662168  0.06544937 0.6661770
```

17/07/12

```
> bsrep<- aov(REP ~ trt, data=BS)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 9.010e-07 3.004e-07   2.934 0.0371
Residuals   100 1.024e-05 1.024e-07
> TukeyHSD(bsrep)
                  diff           lwr          upr     p
p-c    8.45819e-05 -1.47302e-04  3.16467e-04 0.7762373
ws-c  -1.73544e-04 -4.05429e-04  5.83406e-05 0.2120017
wsp-c -3.61210e-05 -2.68006e-04  1.95764e-04 0.9771057
ws-p  -2.58126e-04 -4.90011e-04 -2.62412e-05 0.0228411
wsp-p -1.20703e-04 -3.525881-04  1.11182e-04 0.5272858
wsp-ws 1.37423e-04 -9.44617e-05  3.69308e-04 0.4128201
```

16/08/12

	> bsrep<- aov(REP ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.810e-07 1.603e-07 1.012 0.391 Residuals 100 1.585e-05 1.584e-07	**> bsrep<- aov(REP~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.614e-06 5.379e-07 3.458 0.0193 Residuals 100 1.555e-05 1.555e-07 **> TukeyHSD(bsrep)** diff lwr upr p adj p-c -9.20706e-05 -0.000377862 1.937206e-04 0.8344051 ws-c -2.49841e-04 -0.000535632 3.594962e-05 0.1086518 wsp-c 8.64681e-05 -0.000199323 3.722594e-04 0.8586077 ws-p -1.57771e-04 -0.000443562 1.280203e-04 0.4762007 wsp-p 1.78538e-04 -0.000107252 4.643301e-04 0.3653855 wsp-ws 3.36309e-04 0.000050518 6.221010e-04 0.0142267
	30/08/12	13/09/12
	> bsrep<- aov(REP ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 3.294e-06 1.098e-06 6.3 0.000589 Residuals 99 1.725e-05 1.743e-07 **> TukeyHSD(bsrep)** diff lwr upr p adj p-c -1.764637e-05 -3.232217e-04 2.879290e-04 0.9987672 ws-c -2.763716e-04 -5.819470e-04 2.920373e-05 0.0910155 wsp-c 2.261801e-04 -7.939527e-05 5.317555e-04 0.2205371 ws-p -2.587253e-04 -5.612900e-04 4.383943e-05 0.1210925 wsp-p 2.438265e-04 -5.873824e-05 5.463912e-04 0.1583663 wsp-ws 5.025517e-04 1.999870e-04 8.051164e-04 0.0001990	**> bsrep<- aov(REP ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 9.240e-06 3.080e-06 5.274 0.00203 Residuals 100 5.839e-05 5.839e-07 **> TukeyHSD(bsrep)** diff lwr upr p p-c -0.000108764 -0.000662510 4.44981e-04 0.9557692 ws-c -0.000578078 -0.001131824 -2.43323e-05 0.0372273 wsp-c 0.000242385 -0.000311360 7.96131e-04 0.6634975 ws-p -0.000469313 -0.001023059 8.44320e-05 0.1263706 wsp-p 0.000351149 -0.000202596 9.04895e-04 0.3519896 wsp-ws 0.000820463 0.000266717 1.37421e-03 0.0010937
	27/09/12	11/10/12
	> bsREP<- aov(REP ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 3.250e-06 1.082e-06 1.794 0.153 Residuals 100 6.035e-05 6.035e-07	**> bsrep<- aov(REP ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.004e-05 1.335e-05 10.95 3.69e-06 Residuals 86 1.049e-04 1.220e-06 **> TukeyHSD(bsrep)** diff lwr upr p adj p-c 9.21970e-04 0.00011950 0.0017244397 0.0176923 ws-c -9.47284e-05 -0.00098189 0.0007924343 0.992304 wsp-c 1.58235e-03 0.00072180 0.0024429063 0.0000363 ws-p -1.01669e-03 -0.00190386 -0.0001295365 0.0180764 wsp-p 6.60383e-04 -0.00020016 0.0015209356 0.1921319 wsp-ws 1.67708e-03 0.00073705 0.0026171100 0.0000633
	25/10/12	
	> bsrep<- aov(REP ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 6.820e-06 2.274e-06 1.959 0.13 Residuals 60 6.964e-05 1.161e-06	
aR	28/06/12	17/07/12

	> bsarep<- aov(aREP ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0033 0.001107 0.287 0.834 Residuals 99 0.3815 0.003853	**> bsarep<- aov(aREP ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0479 0.015952 4.186 0.00777 Residuals 100 0.3811 0.003811 **> TukeyHSD(bsarep)** diff lwr upr p adj p-c 0.06061018 0.01587734 0.10534302 0.0033602 ws-c 0.03091025 -0.01382258 0.07564309 0.2768883 wsp-c 0.02831938 -0.01641345 0.07305222 0.3534804 ws-p -0.02969993 -0.07443276 0.01503291 0.3113406 wsp-p -0.03229080 -0.07702363 0.01244204 0.2405951 wsp-ws -0.00259087 -0.04732371 0.04214197 0.9987570
	1/08/12	16/08/12
	> bsarep<- aov(aREP ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0205 0.006818 0.222 0.881 Residuals 100 3.0689 0.030689	**> bsarep<- aov(aREP~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.049 0.01646 0.51 0.676 Residuals 100 3.225 0.03225
	30/08/12	13/09/12
	> bsarep<- aov(aREP ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0734 0.02447 0.886 0.451 Residuals 99 2.7353 0.02763	**> bsarep<- aov(aREP ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.101 0.03367 1.369 0.257 Residuals 100 2.459 0.02459
	27/09/12	11/10/12
	> bsaREP<- aov(aREP ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1577 0.05257 1.783 0.155 Residuals 100 2.9477 0.02948	**> bsarep<- aov(aREP ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.3369 0.11231 4.364 0.00655 Residuals 86 2.2132 0.02573 **> TukeyHSD(bsarep)** diff lwr upr p adj p-c -0.03201633 -0.14858653 0.084553873 0.8890454 ws-c -0.16575506 -0.29462827 -0.036881853 0.0060975 wsp-c -0.09360736 -0.21861498 0.031400254 0.2105639 ws-p -0.13373873 -0.26261194 -0.004865523 0.0388955 wsp-p -0.06159103 -0.18659865 0.063416584 0.5712227 wsp-ws 0.07214770 -0.06440484 0.208700238 0.5125847
	25/10/12	
	> bsarep<- aov(aREP ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0618 0.02059 0.567 0.639 Residuals 60 2.1776 0.03629	
ARI	28/06/12	17/07/12
	> bsari<- aov(ARI ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.649 0.21644 5.984 0.000862 Residuals 99 3.581 0.03617 **> TukeyHSD(bsari)** diff lwr upr p adj p-c -0.004312379 -0.14352437 0.134899613 0.9998085 ws-c -0.137161364 -0.27637336 0.002050627 0.0550464 wsp-c 0.083595018 -0.05561697 0.222807009 0.4008591 ws-p -0.132848985 -0.27068940 0.004991426 0.0631763 wsp-p 0.087907397 -0.04993301 0.225747808 0.3468192 wsp-ws 0.220756382 0.08291597 0.358596793 0.0003555	**> bsari<- aov(ARI ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 8.148e+09 2.716e+09 2.29 0.0829 Residuals 100 1.186e+11 1.186e+09
	1/08/12	16/08/12

	> bsari<- aov(ARI ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.42 1.4742 1.868 0.14 Residuals 100 78.92 0.7892	**> bsari<- aov(ARI~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.568e+10 5.227e+09 0.872 0.458 Residuals 100 5.992e+11 5.992e+09
	30/08/12	13/09/12
	> bsari<- aov(ARI ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 26.1 8.700 1.826 0.147 Residuals 99 471.8 4.766	**> bsARI<- aov(ARI ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.75 0.5847 1.74 0.164 Residuals 100 33.60 0.3360
	27/09/12	11/10/12
	> bsari<- aov(ARI ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 2.34 0.7784 1.271 0.288 Residuals 100 61.24 0.6124	**> bsari<- aov(ARI ~ trt, data=BS)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 5.87 1.9556 4.648 0.00464 Residuals 86 36.18 0.4207 **> TukeyHSD(bsari)** diff lwr upr p adj p-c -0.1576861 -0.62902792 0.3136558 0.8169952 ws-c 0.1590256 -0.36206246 0.6801136 0.8544272 wsp-c 0.5370569 0.03159912 1.0425148 0.0328368 ws-p 0.3167116 -0.20437638 0.8377996 0.3883475 wsp-p 0.6947430 0.18928520 1.2002008 0.0029249 wsp-ws 0.3780314 -0.17410736 0.9301701 0.2833535
	25/10/12	
	> bsari<- aov(ARI ~ trt, data=BS) Df Sum Sq Mean Sq F value Pr(>F) trt 3 28.87 9.624 3.391 0.0236 Residuals 60 170.31 2.839 **> TukeyHSD(bsari)** diff lwr upr p adj p-c 0.5561370 -0.8584972 1.9707713 0.7274238 ws-c -1.2518214 -2.6664557 0.1628129 0.1007602 wsp-c -0.6181853 -2.6345955 1.3982249 0.8494305 ws-p -1.8079585 -3.3820182 -0.2338987 0.0181594 wsp-p -1.1743224 -3.3056093 0.9569646 0.4701730 wsp-ws 0.6336361 -1.4976508 2.7649230 0.8606895	

Dianella revoluta

red	28/06/12	17/07/12
	> drred<- aov(Red ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.002277 7.59e-04 13.26 4.74e-07 Residuals 76 0.004349 5.72e-05 **> TukeyHSD(drred)** diff lwr upr p adj p-c 9.007777e-03 0.002724045 0.015291509 0.0018158 ws-c -2.842468e-03 -0.009126200 0.003441265 0.6361021 wsp-c 9.107410e-03 0.002823678 0.015391142 0.0015820 ws-p -1.185024e-02 -0.018133977 -0.005566512 0.0000252 wsp-p 9.963275e-05 -0.006184100 0.006383365 0.9999738 wsp-ws 1.194988e-02 0.005666145 0.018233610 0.0000214	**> drred<- aov(red ~ trt, data=DR)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.000207 6.891e-05 0.703 0.553 Residuals 75 0.007349 9.798e-05
	1/08/12	16/08/12
	> drred<- aov(red ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.001276 0.0004253 1.126 0.344 Residuals 76 0.028711 0.0003778	**> drred<- aov(red~ trt, data=DR)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.003300 0.0010998 8.437 6.56e-05 Residuals 76 0.009907 0.0001304

		> TukeyHSD(drred) diff lwr upr p ad p-c -0.015009147 -0.024493145 -0.005525148 0.0004783 ws-c -0.011062630 -0.020546628 -0.001578632 0.0156585 wsp-c -0.016341120 -0.025825119 -0.006857122 0.0001266 ws-p 0.003946517 -0.005537482 0.013430515 0.6948252 wsp-p -0.001331974 -0.010815972 0.008152024 0.9827235 wsp-ws-0.005278490 -0.014762488 0.004205508 0.4653472
	30/08/12	13/09/12
	> drred<- aov(red ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.003382 0.0011274 11.31 3.25e-06 Residuals 76 0.007577 0.0000997 > TukeyHSD(drred) diff lwr upr p adj p-c 0.006046669 -0.002247168 0.0143405050 0.2304150 ws-c -0.012008793 -0.020302629 -0.0037149562 0.0016021 wsp-c -0.001494646 -0.009788482 0.0067991903 0.9647100 ws-p -0.018055461 -0.026349298 -0.0097616248 0.0000012 wsp-p -0.007541315 -0.015835151 0.0007525216 0.0879636 wsp-ws 0.010514146 0.002220310 0.0188079828 0.0071938	> drred<- aov(red ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.004925 0.0016417 3.972 0.011 Residuals 76 0.031408 0.0004133 > TukeyHSD(drred) diff lwr upr p adj p-c 0.009367787 -0.007518823 0.026254396 0.4682434 ws-c -0.002760822 -0.019647432 0.014125788 0.9732470 wsp-c 0.017053787 0.000167177 0.033940396 0.0468693 ws-p -0.012128609 -0.029015219 0.004758001 0.2423776 wsp-p 0.007686000 -0.009200610 0.024572610 0.6314987 wsp-ws 0.019814609 0.002927999 0.036701219 0.0148700
	27/09/12	11/10/12
	> drred<- aov(red ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00453 0.0015099 5.056 0.00302 Residuals 76 0.02269 0.0002986 > TukeyHSD(drred) diff lwr upr p adj p-c 0.006994153 -0.0073601268 0.021348433 0.5784444 ws-c -0.005998795 -0.0203530743 0.008355485 0.6919777 wsp-c 0.014087502 -0.0002667782 0.028441781 0.0563642 ws-p -0.012992948 -0.0273472273 0.001361332 0.0901315 wsp-p 0.007093349 -0.0072609311 0.021447628 0.5670060 wsp-ws 0.020086296 0.0057320164 0.034440576 0.0024354	> drred<- aov(red ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00831 0.0027690 6.216 0.000783 Residuals 76 0.03386 0.0004455 > TukeyHSD(drred) diff lwr upr p adj p-c 0.0045154056 -0.013016956 0.02204777 0.9056798 ws-c -0.0007282666 -0.018260628 0.01680409 0.9995310 wsp-c 0.0243335799 0.006801219 0.04186594 0.0026830 ws-p -0.0052436722 -0.022776034 0.01228869 0.8607542 wsp-p 0.0198181743 0.002285813 0.03735054 0.0204000 wsp-ws 0.0250618465 0.007529485 0.04259421 0.0018804
	25/10/12	
	> drred<- aov(red ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.03102 0.01034 3.787 0.0138 Residuals 74 0.20203 0.00273 > TukeyHSD(drred) diff lwr upr p adj p-c 0.002272003 -0.0411575385 0.04570154 0.9990637 ws-c 0.046687934 0.0032583927 0.09011748 0.0302210 wsp-c 0.031764265 -0.0128553476 0.07638388 0.2493204 ws-p 0.044415931 0.0009863898 0.08784547 0.0430784 wsp-p 0.029492262 -0.0151273505 0.07411187 0.3120358 wsp-ws -0.014923669 -0.0595432817 0.02969594 0.8156616	
green	28/06/12	17/07/12
	> drgrn<- aov(Green ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.005404 0.0018014 5.148 0.00271 Residuals 76 0.026595 0.0003499 > TukeyHSD(drgrn) diff lwr upr p adj p-c 0.010893586 -0.0046451558 0.026432328 0.2622050 ws-c -0.011893566 -0.0274323082 0.003645175 0.1931768 wsp-c 0.003392062 -0.0121466801 0.018930804 0.9397186	> drgrn<- aov(green ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00067 0.0002245 0.348 0.791 Residuals 75 0.04837 0.0006450

```
ws-p   -0.022787152 -0.0383258942 -0.007248411 0.0013619
wsp-p  -0.007501524 -0.0230402661  0.008037218 0.5858787
wsp-ws  0.015285628 -0.0002531137  0.030824370 0.0555423
```

1/08/12

```
> drgrn<- aov(green ~ trt, data=DR)
            Df  Sum Sq   Mean Sq F value Pr(>F)
trt          3 0.00113 0.0003773   0.395  0.757
Residuals   76 0.07252 0.0009543
```

16/08/12

```
> drgrn<- aov(green~ trt, data=DR)
            Df  Sum Sq   Mean Sq F value Pr(>F)
trt          3 0.00629 0.0020957   2.219 0.0928
Residuals   76 0.07178 0.0009445
```

30/08/12

```
> drrgn<- aov(green ~ trt, data=DR)
            Df  Sum Sq  Mean Sq F value   Pr(>F)
trt          3 0.01391 0.004637   6.147 0.000847
Residuals   76 0.05732 0.000754
> TukeyHSD(drrgn)
              diff          lwr          upr     p adj
p-c     0.023843882  0.001031153  0.046656611 0.0370448
ws-c   -0.012797323 -0.035610052  0.010015406 0.4583682
wsp-c   0.005947562 -0.016865167  0.028760290 0.9025709
ws-p   -0.036641205 -0.059453934 -0.013828476 0.0003844
wsp-p  -0.017896320 -0.040709049  0.004916408 0.1754552
wsp-ws  0.018744885 -0.004067844  0.041557613 0.1443852
```

13/09/12

```
> drgrn<- aov(green ~ trt, data=DR)
            Df  Sum Sq  Mean Sq F value  Pr(>F)
trt          3 0.00977 0.003256   5.823 0.00123
Residuals   76 0.04250 0.000559
> TukeyHSD(drgrn)
              diff          lwr          upr     p ad
p-c     0.010516081 -0.009127380  0.030159541 0.4994151
ws-c   -0.018019825 -0.037663286  0.001623635 0.0838230
wsp-c   0.007290144 -0.012353317  0.026933604 0.7641182
ws-p   -0.028535906 -0.048179367 -0.008892445 0.0015367
wsp-p  -0.003225937 -0.022869398  0.016417524 0.9729029
wsp-ws  0.025309969  0.005666508  0.044953430 0.0060957
```

27/09/12

```
> drgrn<- aov(green ~ trt, data=DR)
            Df  Sum Sq  Mean Sq F value   Pr(>F)
trt          3 0.04114 0.013713   16.67 2.04e-08
Residuals   76 0.06253 0.000823
> TukeyHSD(drgrn)
              diff          lwr          upr     p adj
p-c     0.045250839  0.021424906  0.069076773 0.0000220
ws-c   -0.016158245 -0.039984178  0.007667688 0.2901766
wsp-c   0.003343023 -0.020482910  0.027168956 0.9827712
ws-p   -0.061409084 -0.085235018 -0.037583151 0.0000000
wsp-p  -0.041907816 -0.065733750 -0.018081883 0.0000893
wsp-ws  0.019501268 -0.004324665  0.043327201 0.1468782
```

11/10/12

```
> drgrn<- aov(green ~ trt, data=DR)
            Df  Sum Sq  Mean Sq F value   Pr(>F)
trt          3 0.02386 0.007953   11.44 2.84e-06
Residuals   76 0.05282 0.000695
> TukeyHSD(drgrn)
              diff          lwr          upr     p ad
p-c     0.034845072  0.012945850  0.056744293 0.0004418
ws-c   -0.012121394 -0.034020615  0.009777828 0.4702157
wsp-c   0.005268961 -0.016630261  0.027168183 0.9214067
ws-p   -0.046966465 -0.068865687 -0.025067244 0.0000017
wsp-p  -0.029576111 -0.051475333 -0.007676889 0.0036708
wsp-ws  0.017390354 -0.004508867  0.039289576 0.1669831
```

25/10/12

```
> drgrn<- aov(green ~ trt, data=DR)
            Df Sum Sq  Mean Sq F value Pr(>F)
trt          3 0.0043 0.001435   0.493  0.688
Residuals   74 0.2153 0.002909
```

NIR

28/06/12

```
> drnir<- aov(NIR ~ trt, data=DR)
            Df  Sum Sq  Mean Sq F value Pr(>F)
trt          3 0.01736 0.005787    2.46 0.0691
Residuals   76 0.17879 0.002352
```

17/07/12

```
> drnir<- aov(NIR ~ trt, data=DR)
            Df  Sum Sq  Mean Sq F value   Pr(>F)
trt          3 0.05875 0.019584   10.35 8.84e-06
Residuals   75 0.14185 0.001891
> TukeyHSD(drnir)
              diff          lwr         upr     p adj
p-c     0.060219047  0.0240824498 0.096355645 0.0002198
ws-c    0.031944904 -0.0046640879 0.068553895 0.1088712
wsp-c   0.069464802  0.0333282044 0.105601400 0.0000176
ws-p   -0.028274144 -0.0648831353 0.008334848 0.1865157
wsp-p   0.009245755 -0.0268908430 0.045382352 0.9072406
wsp-ws  0.037519898  0.0009109069 0.074128890 0.0424582
```

1/08/12

```
> drnir<- aov(NIR ~ trt, data=DR)
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3 0.0938 0.03127   1.508  0.219
```

16/08/12

```
> drnir<- aov(NIR~ trt, data=DR)
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3 0.0722 0.02408   0.955  0.418
```

	Residuals 76 1.5757 0.02073	Residuals 76 1.9154 0.02520
	30/08/12	13/09/12
	> drnir<- aov(NIR ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0834 0.02781 2.313 0.0827 Residuals 76 0.9138 0.01202	**> drnir<- aov(NIR ~ trt, data=DR)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0318 0.01060 1.051 0.375 Residuals 76 0.7659 0.01008
	27/09/12	11/10/12
	> drNIR<- aov(NIR ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0325 0.010821 1.764 0.161 Residuals 76 0.4662 0.006135	**> drnir<- aov(NIR ~ trt, data=DR)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0415 0.01384 1.337 0.269 Residuals 76 0.7872 0.01036
	25/10/12	
	> drnir<- aov(NIR ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.021 0.006989 0.317 0.813 Residuals 74 1.633 0.022073	
NDVI	28/06/12	17/07/12
	> drndvi<- aov(NDVI ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.008616 0.0028719 11.86 1.87e-06 Residuals 76 0.018397 0.0002421 **> TukeyHSD(drndvi)** diff lwr upr p adj p-c -0.016636725 -0.029560576 -0.003712874 0.0061536 ws-c 0.005499990 -0.007423861 0.018423841 0.6797019 wsp-c -0.018548662 -0.031472513 -0.005624811 0.0017889 ws-p 0.022136715 0.009212864 0.035060566 0.0001397 wsp-p -0.001911937 -0.014835788 0.011011914 0.9799231 wsp-ws -0.024048652 -0.036972503 -0.011124801 0.0000324	**> drndvi<- aov(NDVI ~ trt, data=DR)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00414 0.0013803 3.22 0.0274 Residuals 75 0.03215 0.0004286 **> TukeyHSD(drndvi)** diff lwr upr p ad p-c 0.0172194504 1.726343e-05 0.03442164 0.0496749 ws-c 0.0115649320 -5.862130e-03 0.02899199 0.3087251 wsp-c 0.0179985065 7.963195e-04 0.03520069 0.0367800 ws-p -0.0056545183 -2.308158e-02 0.01177254 0.8290320 wsp-p 0.0007790561 -1.642313e-02 0.01798124 0.9993921 wsp-ws0.0064335744 -1.099349e-02 0.02386064 0.7668379
	1/08/12	16/08/12
	> drndvi<- aov(NDVI ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01133 0.003778 1.25 0.298 Residuals 76 0.22976 0.003023	**> drndvi<- aov(NDVI~ trt, data=DR)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00757 0.0025237 3.624 0.0167 Residuals 76 0.05292 0.0006964 **> TukeyHSD(drndvi)** diff lwr upr p adj p-c 0.0233605148 0.001440274 0.045280755 0.0322076 ws-c 0.0109394369 -0.010980804 0.032859678 0.5589137 wsp-c 0.0232846039 0.001364363 0.045204845 0.0329833 ws-p -0.0124210779 -0.034341319 0.009499163 0.4494287 wsp-p -0.0000759109 -0.021996152 0.021844330 0.9999997 wsp-ws 0.0123451670 -0.009575074 0.034265408 0.4548757
	30/08/12	13/09/12

```
> drndvi<- aov(NDVI ~ trt, data=DR)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.01125 0.003749   6.895 0.000361
Residuals    76 0.04132 0.000544
> TukeyHSD(drndvi)
                diff          lwr          upr     p adj
p-c    -0.004512041 -0.023881080  0.014856998 0.9279941
ws-c    0.026192692  0.006823653  0.045561731 0.0036181
wsp-c   0.003124180 -0.016244859  0.022493219 0.9742613
ws-p    0.030704733  0.011335694  0.050073772 0.0004666
wsp-p   0.007636221 -0.011732818  0.027005260 0.7291539
wsp-ws -0.023068512 -0.042437551 -0.003699473 0.0130287
```

```
> drndvi<- aov(NDVI ~ trt, data=DR)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.02582 0.008605   3.029 0.0345
Residuals    76 0.21587 0.002840
> TukeyHSD(drndvi)
                diff          lwr          upr     p adj
p-c    -0.024064576 -0.06833573 0.020206577 0.4861295
ws-c    0.001304729 -0.04296642 0.045575881 0.9998321
wsp-c  -0.041891423 -0.08616258 0.002379729 0.0703036
ws-p    0.025369304 -0.01890185 0.069640457 0.4394324
wsp-p  -0.017826848 -0.06209800 0.026444305 0.7160596
wsp-ws -0.043196152 -0.08746730 0.001075001 0.0584483
```

27/09/12

```
> drNDVI<- aov(NDVI ~ trt, data=DR)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.02892 0.009641   6.169 0.000827
Residuals    76 0.11878 0.001563
> TukeyHSD(drNDVI)
                diff          lwr          upr     p adj
p-c    -0.01122116 -4.406010e-02  0.021617793 0.8061022
ws-c    0.02157586 -1.126309e-02  0.054414807 0.3175896
wsp-c  -0.03100479 -6.384374e-02  0.001834159 0.0712202
ws-p    0.03279701 -4.193419e-05  0.065635963 0.0504166
wsp-p  -0.01978363 -5.262258e-02  0.013055314 0.3946383
wsp-ws -0.05258065 -8.541960e-02 -0.019741700 0.0004027
```

```
> drndvi<- aov(NDVI ~ trt, data=DR)
             Df  Sum Sq  Mean Sq F value   Pr(>F)
trt           3 0.05325 0.017749   6.374 0.000654
Residuals    76 0.21164 0.002785
> TukeyHSD(drndvi)
                diff          lwr          upr     p adj
p-c    -0.002328939 -0.04616402  0.04150615 0.9990215
ws-c    0.004252572 -0.03958251  0.04808766 0.9941472
wsp-c  -0.058688907 -0.10252399 -0.01485382 0.0040443
ws-p    0.006581511 -0.03725357  0.05041660 0.9790489
wsp-p  -0.056359969 -0.10019505 -0.01252488 0.0062311
wsp-ws -0.062941480 -0.10677656 -0.01910640 0.0017790
```

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```
> drndvi<- aov(NDVI ~ trt, data=DR)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3 0.1973 0.06578   5.036 0.00313
Residuals    74 0.9666 0.01306
> TukeyHSD(drndvi)
                diff         lwr         upr     p adj
p-c     0.002970278 -0.09202309  0.09796364 0.9997991
ws-c   -0.110905432 -0.20589880 -0.01591207 0.0155668
wsp-c  -0.081615098 -0.17921150  0.01598131 0.1333128
ws-p   -0.113875710 -0.20886907 -0.01888235 0.0123141
wsp-p  -0.084585376 -0.18218178  0.01301103 0.1125473
wsp-ws  0.029290334 -0.06830607  0.12688674 0.8593055
```

RES

28/06/12

```
> drrep<- aov(REP ~ trt, data=DR)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 3.778e-07 1.259e-07   3.962 0.0111
Residuals    76 2.416e-06 3.179e-08
> TukeyHSD(drrep)
                 diff           lwr          upr     p adj
p-c    -9.441360e-05 -2.425139e-04 5.368666e-05 0.3441455
ws-c    8.434125e-05 -6.375901e-05 2.324415e-04 0.4449811
wsp-c   5.879220e-05 -8.930806e-05 2.068925e-04 0.7249349
ws-p    1.787549e-04  3.065459e-05 3.268551e-04 0.0115394
wsp-p   1.532058e-04  5.105543e-06 3.013061e-04 0.0398203
wsp-ws -2.554905e-05 -1.736493e-04 1.225512e-04 0.9688206
```

17/07/12

```
> drrep<- aov(REP ~ trt, data=DR)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 2.30e-08 7.720e-09   0.109  0.955
Residuals    75 5.32e-06 7.094e-08
```

1/08/12

```
> drrep<- aov(REP ~ trt, data=DR)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 2.240e-07 7.462e-08   0.629  0.598
Residuals    76 9.013e-06 1.186e-07
```

16/08/12

```
> drrep<- aov(REP~ trt, data=DR)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 4.150e-07 1.382e-07   1.153  0.333
Residuals    76 9.105e-06 1.198e-07
```

aR

30/08/12

```
> drrep<- aov(REP ~ trt, data=DR)
             Df    Sum Sq   Mean Sq F value   Pr(>F)
trt           3 2.206e-06 7.354e-07   8.966 3.71e-05
Residuals    76 6.233e-06 8.200e-08
> TukeyHSD(drrep)
                  diff           lwr           upr     p
adj
p-c    0.0003737044  1.358149e-04  6.115938e-04 0.0005325
ws-c  -0.0000595221 -2.974115e-04  1.783673e-04 0.9126799
wsp-c  0.0001011642 -1.367253e-04  3.390536e-04 0.6802039
ws-p  -0.0004332265 -6.711159e-04 -1.953370e-04 0.0000483
wsp-p -0.0002725402 -5.104296e-04 -3.465077e-05 0.0182527
wsp-ws 0.0001606862 -7.720318e-05  3.985757e-04 0.2936094
```

13/09/12

```
> drrep<- aov(REP ~ trt, data=DR)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 6.690e-07 2.232e-07   3.191 0.0283
Residuals    76 5.315e-06 6.994e-08
> TukeyHSD(drrep)
                  diff           lwr           upr     p
adj
p-c    0.00002122 -1.98454e-04  2.409008e-04 0.9942198
ws-c  -0.00021268 -4.32367e-04  6.990753e-06 0.0613074
wsp-c -0.00006068 -2.80365e-04  1.589932e-04 0.8865473
ws-p  -0.00023391 -4.53589e-04 -1.423095e-05 0.0324131
wsp-p -0.00008190 -3.01586e-04  1.377715e-04 0.7615601
wsp-ws 0.00015200 -6.76766e-05  3.716815e-04 0.2731546
```

27/09/12

```
> drREP<- aov(REP ~ trt, data=DR)
             Df    Sum Sq   Mean Sq F value  Pr(>F)
trt           3 7.415e-06 2.472e-06   14.72 1.2e-07
Residuals    76 1.276e-05 1.679e-07
> TukeyHSD(drREP)
                  diff           lwr           upr     p
adj
p-c    0.0006113458  2.709351e-04  9.517565e-04 0.0000620
ws-c  -0.0002099077 -5.503184e-04  1.305030e-04 0.3737914
wsp-c  0.0002279941 -1.124166e-04  5.684049e-04 0.3009068
ws-p  -0.0008212535 -1.161664e-03 -4.808428e-04 0.0000001
wsp-p -0.0003833517 -7.237624e-04 -4.294092e-05 0.0210335
wsp-ws 0.0004379018  9.749112e-05  7.783126e-04 0.0061980
```

11/10/12

```
> drrep<- aov(REP ~ trt, data=DR)
             Df    Sum Sq   Mean Sq F value   Pr(>F)
trt           3 4.292e-06 1.431e-06   13.06 5.75e-07
Residuals    76 8.323e-06 1.095e-07
> TukeyHSD(drrep)
                  diff           lwr           upr     p
adj
p-c    5.1129e-04  2.36415e-04  7.861815e-04 0.0000327
ws-c  -9.9019e-05 -3.73903e-04  1.758634e-04 0.7800264
wsp-c  1.4221e-04 -1.32670e-04  4.170960e-04 0.5287663
ws-p  -6.1031e-04 -8.85201e-04 -3.354348e-04 0.0000007
wsp-p -3.6908e-04 -6.43968e-04 -9.420227e-05 0.0039179
wsp-ws 2.4123e-04 -3.36507e-05  5.161158e-04 0.1058496
```

25/10/12

```
> drrep<- aov(REP ~ trt, data=DR)
             Df    Sum Sq   Mean Sq F value   Pr(>F)
trt           3 3.378e-06 1.126e-06   6.585 0.000526
Residuals    74 1.265e-05 1.710e-07
> TukeyHSD(drrep)
                  diff           lwr           upr     p
adj
p-c    0.0001924572 -0.0001512526  5.361670e-04 0.4595920
ws-c  -0.0003531854 -0.0006968952 -9.475554e-06 0.0417114
wsp-c -0.0002043109 -0.0005574392  1.488174e-04 0.4304575
ws-p  -0.0005456426 -0.0008893524 -2.019328e-04 0.0004630
wsp-p -0.0003967681 -0.0007498964 -4.363980e-05 0.0214544
wsp-ws 0.0001488745 -0.0002042538  5.020028e-04 0.6857184
```

28/06/12

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	> drarep<- aov(aREP ~ trt, data=DR) > summary(drarep) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00835 0.002783 1.581 0.201 Residuals 76 0.13381 0.001761	**> drarep<- aov(aREP ~ trt, data=DR)** > summary(drarep) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.04614 0.01538 9.736 1.68e-05 Residuals 75 0.11848 0.00158 --- Signif. codes: 0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1 > TukeyHSD(drarep) Tukey multiple comparisons of means 95% family-wise confidence level Fit: aov(formula = aREP ~ trt, data = DR) $trt diff lwr upr p adj p-c 0.05508494 0.022060088 0.08810979 0.0002166 ws-c 0.03179738 -0.001659185 0.06525395 0.0685038 wsp-c 0.06118885 0.028163998 0.09421370 0.0000356 ws-p -0.02328756 -0.056744123 0.01016901 0.2680331 wsp-p 0.00610391 -0.026920941 0.03912876 0.9620614 wsp-ws 0.02939147 -0.004065100 0.06284803 0.1052565
	1/08/12	16/08/12
	> drarep<- aov(aREP ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0924 0.03079 1.67 0.181 Residuals 76 1.4012 0.01844	**> drarep<- aov(aREP~ trt, data=DR)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0428 0.01428 0.696 0.557 Residuals 76 1.5599 0.02053
	30/08/12	13/09/12
	> drarep<- aov(aREP ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0542 0.018063 1.829 0.149 Residuals 76 0.7506 0.009876	**> drarep<- aov(aREP ~ trt, data=DR)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0234 0.007814 0.789 0.504 Residuals 76 0.7527 0.009904
	27/09/12	11/10/12
	> draREP<- aov(aREP ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0527 0.017568 3.344 0.0235 Residuals 76 0.3993 0.005254 **> TukeyHSD(draREP)** diff lwr upr p adj p-c 0.03582414 -0.02438790 0.096036176 0.4058370 ws-c 0.04873274 -0.01147930 0.108944778 0.1541860 wsp-c -0.01440181 -0.07461385 0.045810229 0.9226498 ws-p 0.01290860 -0.04730344 0.073120642 0.9426486 wsp-p -0.05022595 -0.11043799 0.009986092 0.1349702 wsp-ws -0.06313455 -0.12334659 -0.002922511 0.0362169	**> drarep<- aov(aREP ~ trt, data=DR)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1008 0.03360 3.251 0.0263 Residuals 76 0.7854 0.01033 **> TukeyHSD(drarep)** diff lwr upr p adj p-c 0.057029069 -0.02741262 0.14147076 0.2937362 ws-c -0.009175165 -0.09361685 0.07526652 0.9918292 wsp-c -0.041395889 -0.12583758 0.04304580 0.5735134 ws-p -0.066204235 -0.15064592 0.01823745 0.1758728 wsp-p -0.098424958 -0.18286665 -0.01398327 0.0157583 wsp-ws -0.032220723 -0.11666241 0.05222096 0.7485082
	25/10/12	
	> drarep<- aov(aREP ~ trt, data=DR) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1892 0.06306 2.722 0.0504 Residuals 74 1.7143 0.02317	
AR	28/06/12	17/07/12

> drari<- aov(ARI ~ trt, data=DR)

```
            Df Sum Sq Mean Sq F value   Pr(>F)
trt          3  2.264  0.7547    7.88 0.000121
Residuals   76  7.279  0.0958
```

> TukeyHSD(drari)

```
              diff          lwr          upr     p adj
p-c     0.4596568  0.202586448  0.71672719 0.0000670
ws-c    0.1478807 -0.109189644  0.40495110 0.4359970
wsp-c   0.2666510  0.009580647  0.52372139 0.0390802
ws-p   -0.3117761 -0.568846464 -0.05470572 0.0110372
wsp-p  -0.1930058 -0.450076173  0.06406457 0.2076058
wsp-ws  0.1187703 -0.138300080  0.37584066 0.6201943
```

1/08/12

> drari<- aov(ARI ~ trt, data=DR)

```
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3  2.764  0.9212   3.078 0.0325
Residuals   76 22.747  0.2993
```

> TukeyHSD(drari)

```
               diff        lwr         upr     p adj
p-c     0.30286184 -0.1515810  0.75730463 0.3051972
ws-c   -0.02616776 -0.4806106  0.42827503 0.9987560
wsp-c  -0.21608664 -0.6705294  0.23835616 0.5979071
ws-p   -0.32902960 -0.7834724  0.12541320 0.2359238
wsp-p  -0.51894848 -0.9733913 -0.06450568 0.0187550
wsp-ws -0.18991888 -0.6443617  0.26452392 0.6919680
```

30/08/12

> drari<- aov(ARI ~ trt, data=DR)

```
            Df Sum Sq Mean Sq F value  Pr(>F)
trt          3  5.301  1.7669   5.424 0.00196
Residuals   76 24.756  0.3257
```

> TukeyHSD(drari)

```
  Tukey multiple comparisons of means
              diff         lwr        upr     p adj
p-c     0.3597802 -0.11431196  0.8338723 0.1995302
ws-c   -0.3176633 -0.79175544  0.1564288 0.3005414
wsp-c   0.2309055 -0.24318658  0.7049976 0.5787826
ws-p   -0.6774435 -1.15153559 -0.2033514 0.0018891
wsp-p  -0.1288746 -0.60296674  0.3452175 0.8912276
wsp-ws  0.5485689  0.07447674  1.0226610 0.0167818
```

27/09/12

> drARI<- aov(ARI ~ trt, data=DR)

```
            Df Sum Sq Mean Sq F value   Pr(>F)
trt          3  7.966  2.6554   10.52 7.28e-06
Residuals   76 19.187  0.2525
```

> TukeyHSD(drARI)

```
              diff        lwr       upr     p adj
p-c     0.18590654 -0.2314697 0.6032828 0.6474748
ws-c   -0.08847215 -0.5058484 0.3289041 0.9444149
wsp-c   0.72441794  0.3070416 1.1417942 0.0001120
ws-p   -0.27437869 -0.6917550 0.1429976 0.3170993
wsp-p   0.53851139  0.1211351 0.9558877 0.0060100
wsp-ws  0.81289008  0.3955138 1.2302664 0.0000134
```

25/10/12

> drari<- aov(ARI ~ trt, data=DR)

```
            Df    Sum Sq   Mean Sq F value Pr(>F)
trt          3 5.492e+09 1.831e+09   0.558  0.645
Residuals   75 2.463e+11 3.284e+09
```

16/08/12

> drari<- aov(ARI~ trt, data=DR)

```
            Df    Sum Sq   Mean Sq F value Pr(>F)
trt          3 2.441e+10 8.136e+09   1.242    0.3
Residuals   76 4.978e+11 6.550e+09
```

13/09/12

> drari<- aov(ARI ~ trt, data=DR)

```
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3  0.423  0.1409   0.411  0.746
Residuals   76 26.054  0.3428
```

11/10/12

> drari<- aov(ARI ~ trt, data=DR)

```
            Df Sum Sq Mean Sq F value   Pr(>F)
trt          3  10.37   3.455   7.776 0.000135
Residuals   76  33.77   0.444
```

> TukeyHSD(drari)

```
               diff        lwr       upr     p adj
p-c    -0.04992227 -0.6036456 0.5038011 0.9952862
ws-c    0.12700280 -0.4267206 0.6807262 0.9309752
wsp-c   0.84351605  0.2897927 1.3972394 0.0008208
ws-p    0.17692507 -0.3767983 0.7306484 0.8355482
wsp-p   0.89343832  0.3397150 1.4471617 0.0003590
wsp-ws  0.71651324  0.1627899 1.2702366 0.0058309
```

```
> drari<- aov(ARI ~ trt, data=DR)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   7.16  2.3878   3.075 0.0328
Residuals    74  57.46  0.7765
> TukeyHSD(drari)
               diff         lwr       upr     p adj
p-c     0.02122175 -0.71117367 0.7536172 0.9998401
ws-c    0.52009885 -0.21229657 1.2524943 0.2513371
wsp-c   0.69722359 -0.05524118 1.4496884 0.0792436
ws-p    0.49887710 -0.23351833 1.2312725 0.2861322
wsp-p   0.67600183 -0.07646294 1.4284666 0.0937447
wsp-ws  0.17712474 -0.57534003 0.9295895 0.9257956
```

Eucalyptus piperita

red

28/06/12

```
> epred<- aov(Red ~ trt, data=EP)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.000404 1.346e-04   2.331 0.0794
Residuals    92 0.005315 5.777e-05
```

17/07/12

```
> epred<- aov(red ~ trt, data=EP)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.000425 1.417e-04   1.752  0.162
Residuals    88 0.007118 8.089e-05
```

1/08/12

```
> epred<- aov(red ~ trt, data=EP)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.00582 0.001941   1.813   0.15
Residuals    90 0.09634 0.001071
```

16/08/12

```
> epred<- aov(red~ trt, data=EP)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.00984 0.003282   2.221 0.0912
Residuals    90 0.13300 0.001478
```

30/08/12

```
> epred<- aov(red ~ trt, data=EP)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.001323 0.0004410   2.249 0.0893
Residuals    77 0.015097 0.0001961
```

13/09/12

```
> epred<- aov(red ~ trt, data=EP)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00041 0.0001362   0.186  0.905
Residuals    76 0.05551 0.0007304
```

27/09/12

```
> epred<- aov(red ~ trt, data=EP)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.01027 0.003425   2.965  0.037
Residuals    80 0.09239 0.001155
> TukeyHSD(epred)
               diff          lwr        upr     p adj
p-c     0.009015873 -0.018325425 0.03635717 0.8227118
ws-c    0.029516322  0.001712884 0.05731976 0.0330915
wsp-c   0.018856329 -0.011781567 0.04949423 0.3761813
ws-p    0.020500449 -0.004740661 0.04574156 0.1521609
wsp-p   0.009840456 -0.018492619 0.03817353 0.7988763
wsp-ws -0.010659993 -0.039439285 0.01811930 0.7657730
```

11/10/12

```
> epred<- aov(red ~ trt, data=EP)
             Df  Sum Sq   Mean Sq F value  Pr(>F)
trt           3 0.00736 0.0024518   4.201 0.00891
Residuals    64 0.03735 0.0005835
> TukeyHSD(epred)
               diff          lwr          upr     p ad
p-c     0.010354125 -0.009184332  0.029892581 0.5053112
ws-c    0.026451199  0.003744105  0.049158293 0.0160526
wsp-c -0.003597904 -0.028730052  0.021534244 0.9814901
ws-p    0.016097074 -0.005026439  0.037220588 0.1950259
wsp-p -0.013952029 -0.037663114  0.009759056 0.4130422
wsp-ws-0.030049103 -0.056432363 -0.003665844 0.0193743
```

25/10/12

```
> epred<- aov(red ~ trt, data=EP)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.000525 0.0001749   0.491   0.69
Residuals    50 0.017805 0.0003561
```

green

28/06/12

```
> epgrn<- aov(Green ~ trt, data=EP)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00173 0.0005754   1.356  0.261
Residuals    92 0.03905 0.0004244
```

17/07/12

```
> epgrn<- aov(green ~ trt, data=EP)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00117 0.0003901   0.893  0.448
Residuals    88 0.03843 0.0004367
```

1/08/12

```
> epgrn<- aov(green ~ trt, data=EP)
```

16/08/12

```
> epgrn<- aov(green~ trt, data=EP)
```

	Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00386 0.001286 0.668 0.574 Residuals 90 0.17335 0.001926	Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00052 0.0001738 0.112 0.953 Residuals 90 0.13950 0.0015499
	30/08/12	13/09/12
	> epgrn<- aov(green ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00527 0.0017579 2.116 0.105 Residuals 77 0.06397 0.0008308	**> epgrn<- aov(green ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01606 0.005355 3.639 0.0164 Residuals 76 0.11182 0.001471 **> TukeyHSD(epgrn)** diff lwr upr p adj p-c 0.020689515 -0.01020521 0.051584241 0.3010151 ws-c 0.001566503 -0.02985042 0.032983430 0.9991905 wsp-c -0.022448977 -0.05999939 0.015101433 0.4015233 ws-p -0.019123012 -0.04764460 0.009398572 0.2999886 wsp-p -0.043138492 -0.07830226 -0.007974722 0.0099109 wsp-ws -0.024015480 -0.05963893 0.011607967 0.2952860
	27/09/12	11/10/12
	> epgrn<- aov(green ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01484 0.004947 2.032 0.116 Residuals 80 0.19473 0.002434	**> epgrn<- aov(green ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01721 0.005735 3.843 0.0136 Residuals 64 0.09551 0.001492 **> TukeyHSD(epgrn)** diff lwr upr p ad p-c 0.028627483 -0.002618045 0.059873011 0.0840642 ws-c 0.008229951 -0.028082801 0.044542704 0.9323560 wsp-c -0.015201508 -0.055392360 0.024989345 0.7512288 ws-p -0.020397531 -0.054177852 0.013382789 0.3899456 wsp-p -0.043828990 -0.081747306 -0.005910675 0.0171424 wsp-ws-0.023431459 -0.065623065 0.018760147 0.4644278
	25/10/12	
	> epgrn<- aov(green ~ trt, data=EP) > summary(epgrn) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00772 0.002572 1.903 0.141 Residuals 50 0.06759 0.001352	
NIR	28/06/12	17/07/12
	> epnir<- aov(NIR ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01256 0.004188 2.081 0.108 Residuals 92 0.18518 0.002013	**> epnir<- aov(NIR ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.03268 0.010893 3.519 0.0183 Residuals 88 0.27240 0.003095 **> TukeyHSD(epnir)** diff lwr upr p ad p-c -0.044367997 -0.086960537 -0.001775457 0.0378624 ws-c -0.006479725 -0.049930442 0.036970993 0.9796546 wsp-c -0.035460009 -0.079390859 0.008470842 0.1567299 ws-p 0.037888272 -0.004208874 0.079985417 0.0932143 wsp-p 0.008907988 -0.033684552 0.051500528 0.9469344 wsp-ws-0.028980284 -0.072431001 0.014470434 0.3061325
	1/08/12	16/08/12
	> epnir<- aov(NIR ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.043 0.01433 0.41 0.746 Residuals 90 3.143 0.03492	**> epnir<- aov(NIR~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.055 0.01825 0.483 0.695 Residuals 90 3.401 0.03779
	30/08/12	13/09/12
	> epnir<- aov(NIR ~ trt, data=EP)	**> epnir<- aov(NIR ~ trt, data=EP)**

	Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1039 0.03465 1.425 0.242 Residuals 77 1.8717 0.02431	Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0955 0.03185 1.236 0.303 Residuals 76 1.9583 0.02577
	27/09/12	11/10/12
	> epNIR<- aov(NIR ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1493 0.04976 1.598 0.196 Residuals 80 2.4908 0.03113	**> epnir<- aov(NIR ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1757 0.05856 2.053 0.115 Residuals 64 1.8252 0.02852
	25/10/12	
	> epnir<- aov(NIR ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0088 0.002938 0.11 0.954 Residuals 50 1.3401 0.026801	
NDVI	28/06/12	17/07/12
	> epndvi<- aov(NDVI ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00170 0.0005676 1.251 0.296 Residuals 92 0.04174 0.0004537	**> epndvi<- aov(NDVI ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00104 0.0003460 0.612 0.609 Residuals 88 0.04979 0.0005658
	1/08/12	16/08/12
	> epndvi<- aov(NDVI ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0147 0.004896 0.967 0.412 Residuals 90 0.4557 0.005063	**> epndvi<- aov(NDVI~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.075 0.02501 2.168 0.0973 Residuals 90 1.039 0.01154
	30/08/12	13/09/12
	> epndvi<- aov(NDVI ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01198 0.003993 1.235 0.303 Residuals 77 0.24905 0.003234	**> epndvi<- aov(NDVI ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0103 0.003429 0.636 0.594 Residuals 76 0.4099 0.005393
	27/09/12	11/10/12
	> epNDVI<- aov(NDVI ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1232 0.04108 6.239 0.000734 Residuals 80 0.5267 0.00658 **> TukeyHSD(epNDVI)** diff lwr upr p adj p-c -0.03386580 -0.09914732 0.03141573 0.5272309 ws-c -0.10456355 -0.17094849 -0.03817860 0.0005004 wsp-c -0.05064245 -0.12379510 0.02251020 0.2732598 ws-p -0.07069775 -0.13096475 -0.01043075 0.0148485 wsp-p -0.01677666 -0.08442619 0.05087288 0.9150088 wsp-ws 0.05392109 -0.01479386 0.12263604 0.1755873	**> epndvi<- aov(NDVI ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0497 0.016578 2.194 0.0973 Residuals 64 0.4836 0.007556
	25/10/12	
	> epndvi<- aov(NDVI ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00444 0.001479 0.241 0.868 Residuals 50 0.30741 0.006148	
RES	28/06/12	17/07/12
	> eprep<- aov(REP ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 8.700e-08 2.901e-08 0.373 0.772 Residuals 92 7.148e-06 7.770e-08	**> eprep<- aov(REP ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 3.740e-07 1.245e-07 1.089 0.358 Residuals 88 1.006e-05 1.143e-07
	1/08/12	16/08/12

	> eprep<- aov(REP ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.029e-06 3.431e-07 1.5 0.22 Residuals 90 2.059e-05 2.287e-07	**> eprep<- aov(REP~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.110e-06 3.700e-07 1.232 0.303 Residuals 90 2.702e-05 3.002e-07
	30/08/12	13/09/12
	> eprep<- aov(REP ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 2.62e-07 8.723e-08 0.487 0.693 Residuals 77 1.38e-05 1.792e-07	**> eprep<- aov(REP ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 5.437e-06 1.812e-06 4.662 0.00481 Residuals 76 2.954e-05 3.887e-07 **> TukeyHSD(eprep)** diff lwr upr p p-c 5.96828e-04 9.46767e-05 1.09898e-03 0.0132726 ws-c 1.52188e-04 -3.58451e-04 6.62827e-04 0.8619970 wsp-c -3.49251e-05 -6.45256e-04 5.75405e-04 0.9987790 ws-p -4.44640e-04 -9.08219e-04 1.89394e-05 0.0648872 wsp-p -6.31753e-04 -1.20329e-03 -6.02143e-05 0.0244088 wsp-ws -1.87113e-04 -7.66124e-04 3.91897e-04 0.8308572
	27/09/12	11/10/12
	> epREP<- aov(REP ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 2.870e-06 9.574e-07 1.993 0.122 Residuals 80 3.844e-05 4.804e-07	**> eprep<- aov(REP ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.830e-06 1.609e-06 2.598 0.0599 Residuals 64 3.962e-05 6.191e-07
	25/10/12	
	> eprep<- aov(REP ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.730e-06 1.576e-06 2.352 0.0833 Residuals 50 3.349e-05 6.699e-07 **> TukeyHSD(eprep)** diff lwr upr p adj p-c 6.017469e-04 -6.518466e-05 0.0012686784 0.0905820 ws-c 3.417422e-05 -9.911745e-04 0.0010595229 0.9997475 wsp-c 1.835189e-05 -1.183976e-03 0.0012206798 0.9999757 ws-p -5.675727e-04 -1.552696e-03 0.0004175505 0.4269611 wsp-p -5.833950e-04 -1.751607e-03 0.0005848174 0.5503140 wsp-ws -1.582233e-05 -1.419839e-03 0.0013881942 0.9999902	
aREP	28/06/12	17/07/12
	> eparep<- aov(aREP ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0082 0.002732 1.214 0.309 Residuals 92 0.2070 0.002250	**> eparep<- aov(aREP ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.02436 0.008121 2.325 0.0803 Residuals 88 0.30740 0.003493
	1/08/12	16/08/12
	> eparep<- aov(aREP ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0203 0.006779 0.233 0.873 Residuals 90 2.6131 0.029035	**> eparep<- aov(aREP~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0747 0.02489 0.714 0.546 Residuals 90 3.1360 0.03484
	30/08/12	13/09/12
	> eparep<- aov(aREP ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1101 0.03670 1.644 0.186 Residuals 77 1.7193 0.02233	**> eparep<- aov(aREP ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1218 0.04061 1.699 0.174 Residuals 76 1.8165 0.02390
	27/09/12	11/10/12

	> epaREP<- aov(aREP ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.3236 0.10787 4.027 0.0101 Residuals 80 2.1427 0.02678 **> TukeyHSD(epaREP)** diff lwr upr p adj p-c -0.03674769 -0.16841578 0.094920396 0.8838311 ws-c -0.16248417 -0.29637779 -0.028590546 0.0109204 wsp-c -0.06058336 -0.20812694 0.086960229 0.7042498 ws-p -0.12573648 -0.24729064 -0.004182315 0.0398153 wsp-p -0.02383566 -0.16027987 0.112608543 0.9678015 wsp-ws 0.10190081 -0.03669225 0.240493878 0.2241848	**> eparep<- aov(aREP ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1111 0.03702 1.265 0.294 Residuals 64 1.8733 0.02927
	25/10/12	
	> eparep<- aov(aREP ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0107 0.003573 0.142 0.935 Residuals 50 1.2622 0.025245	
ARI	28/06/12	17/07/12
	> epari<- aov(ARI ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 1.14 0.3802 0.643 0.589 Residuals 92 54.39 0.5912	**> EPari<- aov(ARI ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 7.814e+10 2.605e+10 3.187 0.0276 Residuals 88 7.192e+11 8.172e+09 **> TukeyHSD(EPari)** diff lwr upr p adj p-c -6451.5479 -75657.314 62754.218 0.9948495 ws-c -71641.6331 -142241.795 -1041.471 0.0453687 wsp-c -7225.3781 -78605.676 64154.920 0.9934334 ws-p -65190.0852 -133590.919 3210.748 0.0675163 wsp-p -773.8303 -69979.597 68431.936 0.9999909 wsp-ws 64416.2549 -6183.907 135016.417 0.0865908
	1/08/12	16/08/12
	> epari<- aov(ARI ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.23 0.0763 0.051 0.985 Residuals 90 135.70 1.5078	**> epari<- aov(ARI~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 2.341e+09 7.804e+08 0.572 0.635 Residuals 90 1.228e+11 1.365e+09
	30/08/12	13/09/12
	> epari<- aov(ARI ~ trt, data=EP) Df Sum Sq Mean Sq F value Pr(>F) trt 3 7.97 2.6571 3.548 0.0183 Residuals 77 57.67 0.7489 **> TukeyHSD(epari)** diff lwr upr p adj p-c 0.39875305 -0.2927699 1.0902759 0.4340549 ws-c 0.45337923 -0.2552209 1.1619793 0.3410874 wsp-c 1.04966366 0.2027245 1.8966028 0.0089755 ws-p 0.05462618 -0.5829264 0.6921788 0.9959495 wsp-p 0.65091061 -0.1375468 1.4393680 0.1414773 wsp-ws 0.59628442 -0.2071926 1.3997614 0.2166077	**> epari<- aov(ARI ~ trt, data=EP)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 10.19 3.398 2.704 0.0513 Residuals 76 95.49 1.256
	27/09/12	11/10/12

```
> epARI<- aov(ARI ~ trt, data=EP)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   5.43   1.811   1.648  0.185
Residuals    80  87.88   1.099
```

```
> epari<- aov(ARI ~ trt, data=EP)
             Df Sum Sq Mean Sq F value  Pr(>F)
trt           3  22.35   7.450   4.216 0.00876
Residuals    64 113.08   1.767
> TukeyHSD(epari)
                 diff         lwr       upr     p adj
p-c     0.19538589 -0.87974618 1.270518 0.9633864
ws-c    1.29821274  0.04872177 2.547704 0.0387236
wsp-c   1.31245046 -0.07048263 2.695384 0.0689235
ws-p    1.10282685 -0.05952526 2.265179 0.0690274
wsp-p   1.11706457 -0.18767244 2.421802 0.1188314
wsp-ws  0.01423772 -1.43753960 1.466015 0.9999937
```

25/10/12

```
> epari<- aov(ARI ~ trt, data=EP)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3   2.84  0.9473   0.625  0.602
Residuals    50  75.75  1.5151
```

Lomandra longifolia

28/06/12

```
> llred<- aov(Red ~ trt, data=LL)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.001149 0.0003830   2.782 0.0466
Residuals    76 0.010463 0.0001377
> TukeyHSD(llred)
                 diff          lwr         upr     p adj
p-c     0.010373302  0.0006266883 0.020119915 0.0325209
ws-c    0.004001094 -0.0057455192 0.013747707 0.7037324
wsp-c   0.002890576 -0.0068560375 0.012637189 0.8637187
ws-p   -0.006372207 -0.0161188208 0.003374406 0.3219061
wsp-p  -0.007482726 -0.0172293391 0.002263888 0.1909629
wsp-ws -0.001110518 -0.0108571316 0.008636095 0.9906082
```

17/07/12

```
> llred<- aov(red ~ trt, data=LL)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.000252 8.403e-05   0.579   0.63
Residuals    76 0.011021 1.450e-04
```

1/08/12

```
> llred<- aov(red ~ trt, data=LL)
             Df  Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.00098 0.0003272   0.383  0.765
Residuals    76 0.06490 0.0008540
```

16/08/12

```
> llred<- aov(red~ trt, data=LL)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.000402 0.0001341   1.078  0.364
Residuals    76 0.009456 0.0001244
```

30/08/12

```
> llred<- aov(red ~ trt, data=LL)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.000813 2.712e-04   3.359 0.0231
Residuals    76 0.006136 8.073e-05
> TujeyHSD(llred)
                 diff          lwr         upr     p adj
p-c    -6.067008e-05 -0.007621824 0.007500483 0.9999966
ws-c   -6.405815e-03 -0.013869401 0.001057772 0.1181078
wsp-c   2.029816e-03 -0.005344382 0.009404015 0.8876046
ws-p   -6.345145e-03 -0.013906298 0.001216009 0.1313116
wsp-p   2.090486e-03 -0.005382447 0.009563420 0.8827720
wsp-ws  8.435631e-03  0.001061432 0.015809830 0.0184839
```

13/09/12

```
> llred<- aov(red ~ trt, data=LL)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.001604 0.0005346   1.518  0.217
Residuals    76 0.026764 0.0003522
```

27/09/12

```
> llred<- aov(red ~ trt, data=LL)
             Df   Sum Sq   Mean Sq F value Pr(>F)
trt           3 0.002047 0.0006823    1.78  0.158
Residuals    76 0.029124 0.0003832
```

11/10/12

```
> llred<- aov(red ~ trt, data=LL)
             Df  Sum Sq  Mean Sq F value  Pr(>F)
trt           3 0.03886 0.012952   7.961 0.00011
Residuals    76 0.12364 0.001627
```

red

		> TukeyHSD(llred) diff lwr upr p adj p-c 0.005929568 -0.027575129 0.03943426 0.9664744 ws-c 0.030347125 -0.003157572 0.06385182 0.0898144 wsp-c 0.055687534 0.022182837 0.08919223 0.0002272 ws-p 0.024417557 -0.009087139 0.05792225 0.2307147 wsp-p 0.049757966 0.016253270 0.08326266 0.0011553 wsp-ws 0.025340409 -0.008164287 0.05884511 0.2020587
	25/10/12	
	> llred<- aov(red ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01848 0.006160 2.481 0.0696 Residuals 60 0.14895 0.002483	
green	28/06/12	17/07/12
	> llgrn<- aov(Green ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00238 0.0007931 1.467 0.23 Residuals 76 0.04109 0.0005407	> llgrn<- aov(green ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00172 0.0005720 0.62 0.604 Residuals 76 0.07013 0.0009228
	1/08/12	16/08/12
	> llgrn<- aov(green ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00886 0.002954 1.64 0.187 Residuals 76 0.13691 0.001801	> llgrn<- aov(green~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00378 0.0012600 1.968 0.126 Residuals 76 0.04866 0.0006403
	30/08/12	13/09/12
	> llgrn<- aov(green ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00754 0.0025132 5.297 0.00227 Residuals 76 0.03606 0.0004745 > TukeyHSD(llgrn) diff lwr upr p adj p-c 0.0029464915 -0.015383852 0.021276835 0.9745134 ws-c -0.0204965280 -0.038590341 -0.002402715 0.0200462 wsp-c 0.0024127829 -0.015464329 0.020289895 0.9846062 ws-p -0.0234430195 -0.041773363 -0.005112676 0.0065799 wsp-p -0.0005337086 -0.018650181 0.017582764 0.9998323 wsp-ws 0.0229093109 0.005032199 0.040786423 0.0064462	> llgrn<- aov(green ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.02469 0.008231 9.02 3.51e-05 Residuals 76 0.06936 0.000913 > TukeyHSD(llgrn) diff lwr upr p ad p-c 0.021491132 -0.003602465 0.046584729 0.1193303 ws-c -0.027735118 -0.052828715 -0.002641521 0.0244240 wsp-c -0.006779765 -0.031873362 0.018313832 0.8929571 ws-p -0.049226250 -0.074319847 -0.024132653 0.0000116 wsp-p -0.028270897 -0.053364493 -0.003177300 0.0209618 wsp-ws 0.020955353 -0.004138244 0.046048950 0.1342826
	27/09/12	11/10/12
	> llgrn<- aov(green ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00458 0.0015258 3.257 0.0261 Residuals 76 0.03561 0.0004685 > TukeyHSD(llgrn) diff lwr upr p adj p-c 0.010094523 -0.007885610 0.028074655 0.4576516 ws-c -0.011144167 -0.029124299 0.006835965 0.3692021 wsp-c 0.001699062 -0.016281070 0.019679195 0.9945840 ws-p -0.021238690 -0.039218822 -0.003258557 0.0140229 wsp-p -0.008395460 -0.026375593 0.009584672 0.6120786 wsp-ws 0.012843229 -0.005136903 0.030823361 0.2468320	> llgrn<- aov(green ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.02761 0.009204 5.524 0.00174 Residuals 76 0.12661 0.001666 > TukeyHSD(llgrn) diff lwr upr p adj p-c 0.014345100 -0.019559429 0.04824963 0.6836658 ws-c 0.015435699 -0.018468831 0.04934023 0.6313079 wsp-c 0.050458681 0.016554152 0.08436321 0.0011235 ws-p 0.001090599 -0.032813931 0.03499513 0.9997818 wsp-p 0.036113581 0.002209051 0.07001811 0.0323298 wsp-ws 0.035022982 0.001118453 0.06892751 0.0402185
	25/10/12	
	> llgrn<- aov(green ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01094 0.003646 1.237 0.304 Residuals 60 0.17678 0.002946	
NIR_green	28/06/12	17/07/12
	> llnir<- aov(NIR ~ trt, data=LL)	> llnir<- aov(NIR ~ trt, data=LL)

	Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00979 0.003263 0.998 0.398 Residuals 76 0.24840 0.003268	Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01281 0.004269 2.432 0.0716 Residuals 76 0.13342 0.001755
	1/08/12	16/08/12
	> llnir<- aov(NIR ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1515 0.05049 1.413 0.246 Residuals 76 2.7160 0.03574	**> llnir<- aov(NIR~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0288 0.00959 0.528 0.664 Residuals 76 1.3793 0.01815
	30/08/12	13/09/12
	> llnir<- aov(NIR ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0350 0.011678 1.494 0.223 Residuals 76 0.5941 0.007817	**> llnir<- aov(NIR ~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0660 0.02201 2.139 0.102 Residuals 76 0.7821 0.01029
	27/09/12	11/10/12
	> llnir<- aov(NIR ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0252 0.008391 1.443 0.237 Residuals 76 0.4421 0.005817	**> llnir<- aov(NIR ~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0134 0.004456 0.522 0.669 Residuals 76 0.6491 0.008541
	25/10/12	
	> llnir<- aov(NIR ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1734 0.05779 2.368 0.0796 Residuals 60 1.4644 0.02441	
NDVI	28/06/12	17/07/12
	> llndvi<- aov(NDVI ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00886 0.0029539 3.743 0.0145 Residuals 76 0.05997 0.0007891 **> TukeyHSD(llndvi)** diff lwr upr p adj p-c -0.0274770002 -0.050811433 -0.004142568 0.0144172 ws-c -0.0055541473 -0.028888580 0.017780285 0.9236614 wsp-c -0.0057273299 -0.029061762 0.017607102 0.9170690 ws-p 0.0219228530 -0.001411579 0.045257285 0.0732814 wsp-p 0.0217496703 -0.001584762 0.045084103 0.0766814 wsp-ws -0.0001731826 -0.023507615 0.023161250 0.9999973	**> llndvi<- aov(NDVI ~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00267 0.0008902 1.207 0.313 Residuals 76 0.05604 0.0007374
	1/08/12	16/08/12
	> llndvi<- aov(NDVI ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0106 0.003519 0.536 0.659 Residuals 76 0.4992 0.006568	**> llndvi<- aov(NDVI~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00025 0.0000833 0.078 0.972 Residuals 76 0.08078 0.0010629
	30/08/12	13/09/12
	> llndvi<- aov(NDVI ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00269 0.0008980 1.905 0.136 Residuals 76 0.03582 0.0004713	**> llndvi<- aov(NDVI ~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00503 0.001675 0.502 0.682 Residuals 76 0.25349 0.003335
	27/09/12	11/10/12

RES

```
> llndvi<- aov(NDVI ~ trt, data=LL)
             Df  Sum Sq  Mean Sq F value Pr(>F)
trt           3 0.01329 0.004431   2.946 0.0382
Residuals    76 0.11429 0.001504
> TukeyHSD(llndvi)
                  diff         lwr          upr     p adj
p-c    -0.014868775 -0.04708110  0.017343547 0.6209006
ws-c   -0.034803020 -0.06701534 -0.002590698 0.0290868
wsp-c  -0.025097476 -0.05730980  0.007114846 0.1803120
ws-p   -0.019934245 -0.05214657  0.012278077 0.3705951
wsp-p  -0.010228701 -0.04244102  0.021983621 0.8380727
wsp-ws  0.009705543 -0.02250678  0.041917865 0.8581176
```

```
> llndvi<- aov(NDVI ~ trt, data=LL)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3 0.1761 0.05870   8.308 7.55e-05
Residuals    76 0.5370 0.00707
> TukeyHSD(llndvi)
                  diff         lwr          upr     p adj
p-c    -0.01767954 -0.08750111  0.052142025 0.9098518
ws-c   -0.06530063 -0.13512219  0.004520938 0.0752156
wsp-c  -0.12092115 -0.19074272 -0.051099584 0.0001162
ws-p   -0.04762109 -0.11744265  0.022200479 0.2853331
wsp-p  -0.10324161 -0.17306318 -0.033420044 0.0012233
wsp-ws -0.05562052 -0.12544209  0.014201044 0.1648309
```

25/10/12

```
> llndvi<- aov(NDVI ~ trt, data=LL)
             Df Sum Sq Mean Sq F value Pr(>F)
trt           3 0.1596 0.05319   3.415 0.0229
Residuals    60 0.9345 0.01558
> TukeyHSD(llndvi)
                  diff        lwr         upr     p adj
p-c    -0.024385684 -0.1286756 0.079904270 0.9259793
ws-c   -0.115435893 -0.2431645 0.012292694 0.0903208
wsp-c  -0.111898851 -0.2268208 0.003023082 0.0590853
ws-p   -0.091050209 -0.2187788 0.036678379 0.2459018
wsp-p  -0.087513167 -0.2024351 0.027408766 0.1949590
wsp-ws  0.003537042 -0.1330106 0.140084645 0.9998836
```

28/06/12

```
> llrep<- aov(REP ~ trt, data=LL)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 1.180e-07 3.931e-08   0.816  0.489
Residuals    76 3.659e-06 4.815e-08
```

17/07/12

```
> llrep<- aov(REP ~ trt, data=LL)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 2.810e-07 9.364e-08   0.816  0.489
Residuals    76 8.725e-06 1.148e-07
```

1/08/12

```
> llrep<- aov(REP ~ trt, data=LL)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 1.347e-06 4.489e-07   2.862 0.0423
Residuals    76 1.192e-05 1.569e-07
> TukeyHSD(llrep)
                  diff           lwr           upr     p adj
p-c     0.0000196574 -0.0003093392 3.486540e-04 0.9986111
ws-c   -0.0002900304 -0.0006190271 3.896619e-05 0.1034546
wsp-c  -0.0001881135 -0.0005171101 1.408831e-04 0.4414021
ws-p   -0.0003096879 -0.0006386845 1.930879e-05 0.0724744
wsp-p  -0.0002077709 -0.0005367675 1.212257e-04 0.3524964
wsp-ws  0.0001019170 -0.0002270797 4.309136e-04 0.8478008
```

16/08/12

```
> llrep<- aov(REP~ trt, data=LL)
             Df    Sum Sq   Mean Sq F value Pr(>F)
trt           3 5.200e-07 1.733e-07   2.631 0.0561
Residuals    76 5.007e-06 6.588e-08
```

30/08/12

```
> llrep<- aov(REP ~ trt, data=LL)
             Df    Sum Sq   Mean Sq F value   Pr(>F)
trt           3 9.050e-07 3.017e-07   6.735 0.000433
Residuals    76 3.404e-06 4.479e-08
> TukeyHSD(llrep)
                  diff           lwr           upr     p ad
p-c    2.109374e-05 -1.569980e-04  1.991855e-04 0.9894791
ws-c  -2.297336e-04 -4.055273e-04 -5.393984e-05 0.0052560
wsp-c  2.367285e-05 -1.500155e-04  1.973612e-04 0.9841618
ws-p  -2.508273e-04 -4.289191e-04 -7.273553e-05 0.0022535
wsp-p  2.579105e-06 -1.734348e-04  1.785930e-04 0.9999793
wsp-ws 2.534064e-04  7.971808e-05  4.270947e-04 0.0014546
```

13/09/12

```
> llrep<- aov(REP ~ trt, data=LL)
             Df    Sum Sq   Mean Sq F value   Pr(>F)
trt           3 3.873e-06 1.291e-06   13.75 2.99e-07
Residuals    76 7.138e-06 9.390e-08
> TukeyHSD(llrep)
                  diff           lwr           upr     p
p-c    4.2276e-04  1.68195e-04  6.773296e-04 0.0002302
ws-c  -1.8311e-04 -4.37684e-04  7.144986e-05 0.2411542
wsp-c  5.2607e-05 -2.01959e-04  3.071745e-04 0.9481937
ws-p  -6.0587e-04 -8.60446e-04 -3.513127e-04 0.0000001
wsp-p -3.7015e-04 -6.24722e-04 -1.155880e-04 0.0015185
wsp-ws 2.3572e-04 -1.88424e-05  4.902917e-04 0.0795743
```

	27/09/12	11/10/12
	> llrep<- aov(REP ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 7.170e-07 2.390e-07 4.093 0.0095 Residuals 76 4.438e-06 5.839e-08 **> TukeyHSD(llrep)** diff lwr upr p ad p-c 0.0000292883 -1.714341e-04 2.300107e-04 0.9807055 ws-c -0.0002152947 -4.160171e-04 -1.457234e-05 0.0307110 wsp-c -0.0000770615 -2.777839e-04 1.236609e-04 0.7449576 ws-p -0.0002445830 -4.453054e-04 -4.386064e-05 0.0105639 wsp-p -0.0001063498 -3.070722e-04 9.437256e-05 0.5083694 wsp-ws 0.0001382332 -6.248916e-05 3.389556e-04 0.2771214	**> llrep<- aov(REP ~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 4.292e-06 1.43e-06 5.298 0.00227 Residuals 76 2.052e-05 2.70e-07 **> TukeyHSD(llrep)** diff lwr upr p p-c 1.44401e-04 -2.87236e-04 0.0005760400 0.8158290 ws-c 5.73422e-05 -3.74296e-04 0.0004889807 0.9852971 wsp-c 5.88788e-04 1.57150e-04 0.0010204273 0.0032794 ws-p -8.70593e-05 -5.18697e-04 0.0003445791 0.9515789 wsp-p 4.44387e-04 1.27488e-05 0.0008760257 0.0411576 wsp-ws 5.31446e-04 9.98082e-05 0.0009630851 0.0095758
	25/10/12	
	> llrep<- aov(REP ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 2.673e-06 8.910e-07 2.499 0.0681 Residuals 60 2.140e-05 3.566e-07	
aREP	28/06/12	17/07/12
	> llarep<- aov(aREP ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.01242 0.004139 1.67 0.181 Residuals 76 0.18835 0.002478	**> llarep<- aov(aREP ~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.00918 0.003060 1.941 0.13 Residuals 76 0.11982 0.001577
	1/08/12	16/08/12
	> llarep<- aov(aREP ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.1386 0.04621 1.48 0.227 Residuals 76 2.3738 0.03123	**> llarep<- aov(aREP~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0232 0.00774 0.511 0.676 Residuals 76 1.1515 0.01515
	30/08/12	13/09/12
	> llarep<- aov(aREP ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0305 0.010160 1.567 0.204 Residuals 76 0.4929 0.006485	**> llarep<- aov(aREP ~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0638 0.021278 2.229 0.0917 Residuals 76 0.7255 0.009547
	27/09/12	11/10/12
	> llarep<- aov(aREP ~ trt, data=LL) Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0666 0.022204 4.37 0.00682 Residuals 76 0.3862 0.005081 **> TukeyHSD(llarep)** diff lwr upr p adj p-c -0.007779603 -0.06699308 0.051433880 0.9857638 ws-c -0.074230963 -0.13344445 -0.015017481 0.0080403 wsp-c -0.028873196 -0.08808668 0.030340286 0.5778487 ws-p -0.066451360 -0.12566484 -0.007237878 0.0216346 wsp-p -0.021093593 -0.08030708 0.038119889 0.7857574 wsp-ws 0.045357767 -0.01385572 0.104571249 0.1926098	**> llarep<- aov(aREP ~ trt, data=LL)** Df Sum Sq Mean Sq F value Pr(>F) trt 3 0.0366 0.01218 1.189 0.32 Residuals 76 0.7788 0.01025
	25/10/12	

```
> llarep<- aov(aREP ~ trt, data=LL)
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3 0.2391 0.07970   3.338 0.0251
Residuals   60 1.4325 0.02387
> TukeyHSD(llarep)
                 diff         lwr          upr     p adj
p-c     8.230196e-02 -0.04681527  0.211419184 0.3408108
ws-c   -6.571151e-02 -0.22384717  0.092424147 0.6920759
wsp-c  -6.575142e-02 -0.20803167  0.076528837 0.6159983
ws-p   -1.480135e-01 -0.30614913  0.010122188 0.0745424
wsp-p  -1.480534e-01 -0.29033363 -0.005773123 0.0383579
wsp-ws -3.990397e-05 -0.16909403  0.169014226 1.0000000
```

ARI

28/06/12

```
> llari<- aov(ARI ~ trt, data=LL)
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3 0.3225 0.10749    2.67 0.0535
Residuals   76 3.0600 0.04026
```

17/07/12

```
> llari<- aov(ARI ~ trt, data=LL)
            Df    Sum Sq  Mean Sq F value Pr(>F)
trt          3 4.320e+08 1.44e+08   1.036  0.382
Residuals   76 1.056e+10 1.39e+08
```

1/08/12

```
> llari<- aov(ARI ~ trt, data=LL)
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3  0.777   0.259   0.668  0.575
Residuals   76 29.492   0.388
```

16/08/12

```
> llari<- aov(ARI~ trt, data=LL)
            Df    Sum Sq   Mean Sq F value Pr(>F)
trt          3 3.455e+10 1.152e+10   0.978  0.408
Residuals   76 8.952e+11 1.178e+10
```

30/08/12

```
> llari<- aov(ARI ~ trt, data=LL)
            Df Sum Sq Mean Sq F value  Pr(>F)
trt          3  1.586  0.5288   4.078 0.00967
Residuals   76  9.854  0.1297
> TukeyHSD(llari)
                 diff         lwr        upr     p adj
p-c    -0.04053432 -0.34355612 0.26248747 0.9849996
ws-c   -0.27584371 -0.57495538 0.02326796 0.0814076
wsp-c   0.10653863 -0.18899074 0.40206799 0.7796286
ws-p   -0.23530938 -0.53833117 0.06771241 0.1826872
wsp-p   0.14707295 -0.15241331 0.44655921 0.5721004
wsp-ws  0.38238233  0.08685297 0.67791170 0.0058357
```

13/09/12

```
> llari<- aov(ARI ~ trt, data=LL)
> summary(llari)
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3  0.721  0.2403   1.298  0.281
Residuals   76 14.075  0.1852
```

27/09/12

```
> llari<- aov(ARI ~ trt, data=LL)
            Df Sum Sq Mean Sq F value Pr(>F)
trt          3  1.152  0.3839   2.499 0.0659
Residuals   76 11.676  0.1536
```

11/10/12

```
> llari<- aov(ARI ~ trt, data=LL)
            Df Sum Sq Mean Sq F value   Pr(>F)
trt          3  13.30   4.433   18.56 3.94e-09
Residuals   76  18.15   0.239
> TukeyHSD(llari)
                 diff         lwr       upr     p adj
p-c     0.1497585 -0.2561833 0.5557003 0.7673714
ws-c    0.7453284  0.3393866 1.1512702 0.0000416
wsp-c   0.9854625  0.5795207 1.3914043 0.0000001
ws-p    0.5955699  0.1896281 1.0015117 0.0013540
wsp-p   0.8357040  0.4297623 1.2416458 0.0000042
wsp-ws  0.2401341 -0.1658077 0.6460759 0.4110022
```

25/10/12

```
> llari<- aov(ARI ~ trt, data=LL)
             Df Sum Sq Mean Sq F value   Pr(>F)
trt           3  23.24   7.746   9.434 3.38e-05
Residuals    60  49.27   0.821
> TukeyHSD(llari)
              diff        lwr      upr     p adj
p-c     0.5728397 -0.1843650 1.330044 0.1997992
ws-c    1.0755439  0.1481613 2.002927 0.0167529
wsp-c   1.6143903  0.7799914 2.448789 0.0000205
ws-p    0.5027042 -0.4246784 1.430087 0.4844656
wsp-p   1.0415507  0.2071517 1.875950 0.0086643
wsp-ws  0.5388464 -0.4525673 1.530260 0.4821419
```