DEPARTMENT OF
PRIMARY INDUSTRIES

Investigate the current state of knowledge worldwide regarding *Neonectria galligena*

Final Report
Horticulture Australia AP05029

(November 2006)

Edwards *et al.*
Primary Industries Research Victoria, Knoxfield Centre

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Horticulture Australia Project No: AP05029
Project Leader: Dr Jacqueline Edwards
Contact Details: Department of Primary Industries,
Primary Industries Research Victoria, Knoxfield Centre
Private Bag 15, Ferntree Gully DC, Victoria 3156
Tel: (03) 92109222
Fax: (03) 9800 3521
Email: jacky.edwards@dpi.vic.gov.au
Project Team: Dr Oscar Villalta, Rachel Powney
Address: Department of Primary Industries,
Primary Industries Research Victoria, Knoxfield Centre
Private Bag 15, Ferntree Gully DC, Victoria 3156

Purpose of project:

The purpose of this project was to investigate European Canker of apple: current knowledge and the role of latent infections of *N. galligena* in fruit and rainfall for disease spread and its potential impact in Australia.

Report completed: November 2006.

Acknowledgments:
The researchers gratefully acknowledge the financial support for this project from Horticulture Australia Limited (HAL), Apple and Pear Australia Ltd (APAL), the Federal Government and the Department of Primary Industries, Primary Industries Research Victoria.

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Media Summary

This project conducted a comprehensive literature review into the pathogen *Neonectria galligena,* which causes European canker in apples, and undertook a predictive modelling study using the software packages, CLIMATE and CLIMEX, to identify regions in Australia with environmental conditions favourable for establishment and spread of the pathogen.

European canker is one of the most important diseases of pome fruit and many species of hardwood forest trees worldwide. Cankers develop on the woody tissues, girdling and killing branches and, occasionally, the whole tree. The disease was found on apple in Tasmania in 1958 and an extensive eradication program was carried out, resulting in area freedom declared in 1991. At the time, no estimate was made of the cost of this incursion to Australia. However, a realistic comparison can be made with the costs of eradication and surveillance associated with the fireblight outbreak in Victoria in 1997, which was estimated to be A$20 million.

The main findings of this project are:

- *N. galligena* has been recorded on more than 60 plant species from 20 genera and from climates ranging from sub-arctic (Iceland, Sweden, Canada), temperate (Europe, USA, Chile), arid (Syria, Saudia Arabia, Afghanistan) to tropical (Java, Florida).
- It has the potential to establish in many parts of Australia, particularly the southern and eastern coastal regions. Industries such as apple, pear, walnut, loquat ornamentals and the nurseries that supply these industries could all be adversely affected.
- There is no data on the susceptibility or otherwise of Australian native flora. The pathogen has been recorded on three species of New Zealand native flora and it is unknown what the source of the infection was.
- There is no data to support or refute whether latent infection in fruit of New Zealand apple varieties is a potential pathway of *N. galligena* into Australia. Latent periods in fruit of these varieties have not been studied. There is no information on the role of latent infection in fruit as a source of inoculum, although mummified fruit are a known inoculum source.

A visit in September 2006 to the Applied Plant Science Division, Department of Agriculture and Rural Development, Belfast, Northern Ireland, established dialogue between the research group led by Dr Alistair McCracken and the team at DPI Victoria. Negotiations are underway to conduct a simulated storage experiment in Northern Ireland to investigate incidence of latent infection of *N. galligena* in the dessert variety, Royal Gala.

Technical Summary

The purpose of this project was to investigate the current state of knowledge regarding European canker of apple and other diseases caused by *Neonectria galligena,* undertake predictive modelling to determine regions of Australia where the pathogen can establish, and identify other industries that might be adversely affected by an incursion. Two stages of the project have been completed (a literature review and a modelling study) and are reported in this document, and a third stage (a simulated storage trial in the UK) is currently under negotiation.

Over 900 references, along with Biosecurity Australia's import risk analysis reports (IRA 2004, 2005), websites and available internal industry documents were reviewed, the important points of which are summarised in the following paragraphs.

Latent infection in fruit depends on time of infection, storage conditions, and production of the antimicrobial compound, benzoic acid, by the fruit. Very little is known about latent infection of fruit in varieties other than Bramley Seedling. It has been assumed that dessert apples with low acidity will have no latent period and rot will be apparent at harvest. However, there have been reports of latent infection in several dessert varieties, particularly if infection occurs close to harvest. The latency periods of varieties grown in New Zealand has not been studied.

There is no method for detecting symptomless infections, particularly latent infection in fruit. This seriously limits the development of strategies to mitigate against entry of *N. galligena* via imported fruit.

Annual rainfall above 1000 mm is not a sufficiently accurate indicator of disease risk. High incidences of fruit infection have been reported in Loughgall, Northern Ireland, where annual rainfall is 791 mm. Duration and frequency of wet events conducive for *N. galligena* infection prior to harvest are more important than annual rainfall. In regions with dry summers such as California, fruit infection is less common. However if harvest is delayed, autumn rains allow fruit infections to occur pre-harvest.

Predictive modelling using the computer programs CLIMATE and CLIMEX identified that many regions of Australia provide a suitable environment for establishment and spread of *N. galligena*. Although assumed to prefer temperate climates, the known distribution of *N. galligena* worldwide ranges from the sub-arctic (Iceland, Sweden, Canada), through arid (Saudia Arabia, Syria, Afghanistan) to the tropics (Java, Florida). The fungus has a very wide host range (more than 60 species in 20 genera), including New Zealand native flora, indicating that it is highly adaptable. Currently there is no available information on the susceptibility of Australian native flora to *N. galligena*. Apple is the most susceptible host of *N. galligena* but European pear, Asian pear, walnut, loquat and many other tree species commonly grown in Australia are also susceptible. The predicted distribution indicates that pear and walnut production regions would be at risk of losses from this pathogen, as would ornamental and nursery industries.

As a result of a visit to the UK in September 2006, negotiations are being held with a team in Northern Ireland to compare the incidence of latent infection in fruit of Bramley Seedling, a culinary variety, with Royal Gala, a dessert variety commonly grown in Australia and New Zealand.

Introduction

European canker is one of the most important diseases of pome fruit and many species of hardwood forest trees worldwide. Cankers develop on the woody tissues, girdling and killing branches and, occasionally, the whole tree. The disease was found on apple in Tasmania in 1958 and an extensive eradication program was carried out, resulting in area freedom declared in 1991. At the time, no estimate was made of the cost of this incursion to Australia. However, a realistic comparison can be made with the costs of eradication and surveillance associated with the fireblight outbreak in Victoria in 1997, which was estimated to be A$20 million (Rodoni *et al.* 2004).

European canker is caused by the fungal pathogen *Neonectria galligena* (anamorph *Cylindrocarpon heteronema*). Long distance spread is known to be via infected planting material. In addition, however, apple fruit can harbour latent (ie symptomless) infections that later develop into storage rots. Plant Health Australia (PHA) and Apple and Fruit Australia Ltd (APAL) consider this pathogen a high priority threat.

An import risk analysis (IRA) for the export of fresh apple fruit from New Zealand to Australia was carried out by Biosecurity Australia (Draft IRA 2004; Revised draft IRA 2005). These IRA reports included a review and an import risk analysis for the disease European canker which is present in apple-producing regions of New Zealand. The IRA concluded that the import risk of European canker could be managed to an acceptable level below Australia's appropriate level of protection (ALOP) by sourcing apples for export from orchards free of disease symptoms. The IRA proposes a combination of mitigation measures that it believes will reduce the risk of fruit infected with *N. galligena* being exported to Australia, to a very low, or negligible level, thus satisfying ALOP.

The literature review in the IRA report was comprehensive and covered aspects of the epidemiology, distribution, infection criteria, host range and control of European canker (*N. galligena*). This information was used to systematically and methodically analyse the risk of *N. galligena* entering Australia in apple fruit exported from New Zealand and to estimate theoretical probabilities for incursion of *N. galligena* and establishment and spread of the disease in Australia.

This current HAL funded project (AP05029) was undertaken to re-examine the state of knowledge worldwide regarding European canker of apples and perennial canker of woody and forest plants, caused by *N. galligena*, concentrating on parts of the IRA that were based on limited information. There were three such areas that needed further investigation:

(1) the potential for importation of apple fruit with latent (symptomless) infections that would not have been expressed at harvest or during distribution,

(2) the view that fruit infection is not a problem in regions with annual rainfall below 1000 mm

(3) whether other industries and host plants could be negatively affected by an incursion of *N. galligena* into Australia.

This project does not examine the conclusions in the risk import analysis of the IRA reports but comments on areas that require further experimentation or analysis. The objectives were to investigate (a) the current state of knowledge worldwide regarding *Neonectria galligena*, (b) the potential for *N. galligena* to impact on additional

industries such as timber, ornamentals and on native flora, and (c) the role of latent infection of *N. galligena* in apple tissues as an entry pathway into Australia.

Materials and Methods

The project was divided into three stages.

Stage 1 was a review of current literature to identify any gaps in the available information on the epidemiology of *N. galligena* and in particular, the role of latent infection of fruit for disease dispersal. A literature search used the following parameters "European canker or *Nectria galligena*" and the databases searched were AGRICOLA, CAB abstracts, Current Contents, Informat and Scopus. The searches produced over 900 references that were subsequently reviewed along with Biosecurity Australia's import risk analysis reports (IRA 2004, 2005), and available internal industry documents. The review examined the complete known host range of the pathogen by accessing worldwide herbarium records to determine whether the introduction of *N. galligena* into Australia may affect native and amenity flora and industries other than pome fruit. Websites were also visited and examined for their usefulness. Key word searches using 'Google' resulted in 31,900 hits for 'Nectria galligena', 2,210 hits for 'Neonectria galligena' and 253,000 hits for 'European canker'. These numbers indicate the seriousness of this pathogen and the diseases it causes around the world.

Stage 1 also included a visit in September 2006 to the UK to meet with Drs Alistair McCracken, Louise Cooke and Averill Brown at the Applied Plant Science Division of the Department of Agriculture and Rural Development, Northern Ireland, and Barton Farms at Wisbech, England. The purpose of the visit was to see first-hand the impact of European canker on apple production and to discuss Stage 3: a simulated storage trial in the UK to determine whether symptomless infection of fruit has the potential to be a pathway for *N. galligena* into Australia.

Stage 2 was a predictive modelling study using climate matching and simulation software (CLIMATE and CLIMEX, respectively) to identify regions of Australia with climatic conditions suitable for the establishment of *N. galligena*. These regions were then compared with distribution maps of susceptible host species in Australia to identify regions at most risk. More detailed methodology is covered in the relevant section in this report.

Stage 3 has yet to be completed. It was not part of the 2005-06 project application, but was to conduct a simulation experiment at an offshore location identified in Stage 1 to determine whether latent infection of fruit can survive the supply chain and therefore be an entry pathway for *N. galligena* into Australia. This stage is still being discussed with Dr McCracken in Northern Ireland. The proposed methodology is to source Bramley Seedling and Royal Gala fruit from a current trial site where canker is known to occur, store for several weeks in modifed atmosphere cold storage, then determine the percentage of fruit per cultivar with core rots attributable to *N. galligena*.

In addition, several people and organisations have been consulted as part of this project. We gratefully acknowledge their assistance and advice:

Dr. Averill Brown, Applied Plant Science Division, Department of Agriculture and Rural Development, Belfast, Northern Ireland

Dr. Louise Cooke, Applied Plant Science Division, Department of Agriculture and Rural Development, Belfast, Northern Ireland

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Ian Pascoe, Consultant Mycologist, Victoria

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Mark Whattam, Australian Quarantine Inspection Service, Victoria

Results and Discussion

STAGE 1 – Literature review of past and current knowledge of European canker and *Neonectria galligena* worldwide

European canker of apples (*Neonectria galligena*)

European canker, caused by *Neonectria galligena*, is one the most important diseases of apple (*Malus domestica*) worldwide. The disease is present in almost all apple-producing regions of the world and losses to this disease are greatest in humid climates of northern Europe, north west America and southern Chile (Braun 1997, Biggs 1985, Booth 1998, Latorre *et al.* 2002, Swinburne 1975, Zeller and Owens 1921). European canker affects trunks, branches and twigs of trees, causing cankers, killing branches and occasionally the whole tree (Swinburne 1975). Young and old trees can be affected by *N. galligena* in apple orchards and loss of young trees due to European canker has been reported to range from 1-10% (Berrie *et al.* 2000, Lovelidge 1995), sometimes requiring replanting of the whole orchard (Grove 1990). Although rare, leaf and petiole infections of both apple and pear have been reported (Ogawa and English 1991, Wormald 1955). *N. galligena* also causes fruit infection of apple and pear (Swinburne 1971b, Dillon-Weston 1926). Losses of up to 80% of the apple crop have been reported in Europe and America (Berrie *et al.* 2000, Butler 1949, Latorre *et al.* 2002, McCartney 1967, Bondoux 1967, Swinburne 1974, Swinburne 1975, Wormald 1955).

Neonectria galligena also causes cankers in a wide range of hardwood trees including *Populus* and *Almus* which are used as windbreaks and in hedgerows around apple orchards in Europe (Swinburne 1975, Booth 1998). *N. galligena* is also a pathogen of several genera of hardwood species in North America (Lortie 1969). The importance of alternative hosts in the development of apple canker was reviewed by Flack and Swinburne (1977). The taxonomy of *N. galligena* and differences between the pathogenicity of spore types on *Malus* and hardwood trees has been investigated (Swinburne 1975, Flack and Swinburne 1977). The description of the morphological and cultural characteristics of the genus *Neonectria* has been described in several papers (Swinburne 1975, Lortie 1962, Lortie 1964). *N. galligena* exists in several strains differing in cultural characteristics, but they appear to be largely non-specific in their pathogenicity (Ng and Roberts 1974, Flack and Swinburne 1977). There are several publications with information on the epidemiology, distribution, host range and control of perennial canker on woody plants (Agrios 1997, Anagnostakis and Ferrandino 1988, Barnard *et al.* 1988, Flack and Swinburne 1977, Ng and Roberts 1974, Sakamoto 2004).

Epidemiology of European canker

There are several publications from overseas with information on the epidemiology, distribution, host range and control of European canker of apples (Latorre *et al.* 2002; McCarney 1967; Swinburne 1975). However, there is a lack of recent information on the epidemiology of European canker in New Zealand (Brook and Clarke 1975). The life cycle of *N. galligena* and mechanisms of spore dispersal as well as infection criteria, sources of infection and infection pathways have been described in many publications (Agrios 1997; Dubin and English 1975; Latorre *et al.* 2002; McCracken *et al.* 2003a; Swinburne 1975). Control methods for this disease are also reported (Berri 1992; Berri *et al.* 1997a; Cooke 1999; Swinburne 1975). Complete removal of cankers is accepted to be the best method for control of *N. galligena*. Destruction of infected wood is critical as spore production can continue for over 2 years on excised wood left on the orchard floor (Saure 1962). Various

chemicals have been formulated as paints for application as eradicants of cankers (Swinburne 1975; McCracken and Cooke 1985).

Life cycle of *Neonectria galligena*

Neonectria galligena overwinters as mycelium in twigs and callus tissue of cankers, growing slowly while its host is dormant or as perithecia in cankered wood (Agrios 1997). Spore production is initiated during periods of cool, wet weather. On younger cankers asexual conidia are generally produced in spring/early summer (Swinburne 1975). It is unusual for perithecia (sexual fruiting structures) to be formed in the first year following infection (Saure 1962). On older cankers, ascospores (sexual spores produced in the perithecia) and conidia are produced, both of which can cause infection (Swinburne 1975). Production and release of spores is largely climate dependant, and is most common in spring and autumn. However, spore production and infection of host tissue can occur at any time of year as long as there is sufficient moisture and the temperature is above 5°C.

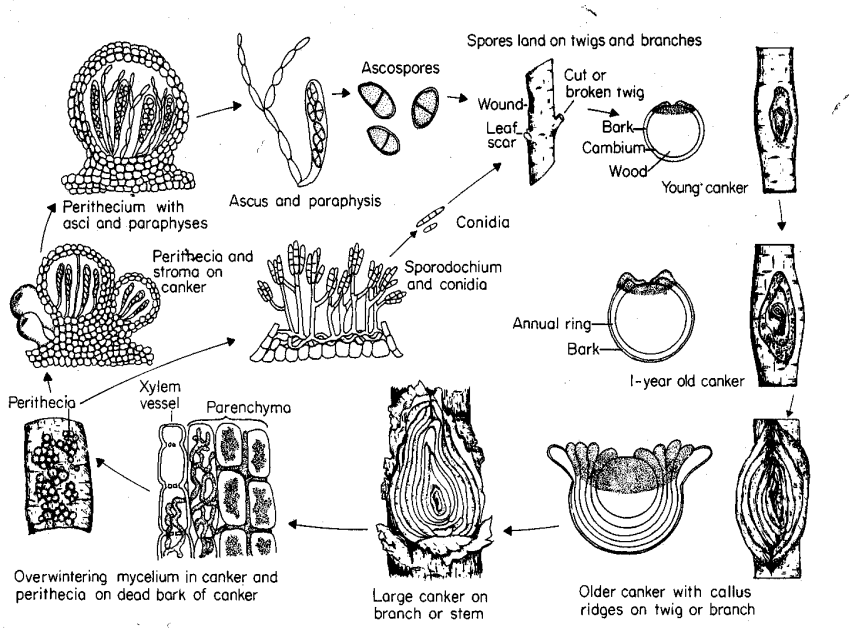

Figure 1 Disease cycle of *Neonectria* canker caused by *Neonectria galligena* (Agrios, G. N. Plant Pathology Fourth Edition, Academic Press Ltd, London 1997)

Conidia are dispersed by moist wind currents and rain splash (Swinburne 1971a, Swinburne 1975) and in some cases carried by insects to susceptible tissue (Houston 1994). With stormy weather, spores carried in rain drops by wind have been reported to travel distances of up to 125 m (Mulder 1966). Release of ascospores is dependent on rainfall quantity and duration of wet periods (Lortie and Kuntz 1963, Swinburne 1971a). There are reported differences in the patterns of seasonal discharge of ascospores in European countries (Swinburne 1971a, Bulit 1957, Swinburne 1975). In Northern Ireland for example, ascospore production was found to peak in spring and early summer, with a second peak in autumn (Swinburne 1971a). Ascospores can be dispersed by rainsplash but are generally regarded to be aerially disseminated (Swinburne 1975). The airborne ascospores are capable of long distance dispersal, while the conidia serve to spread the disease short distances and to intensify the disease in trees that are already infected.

Photo 1. Conidia of *Neonectria galligena*.
http://www.botany.hawaii.edu/

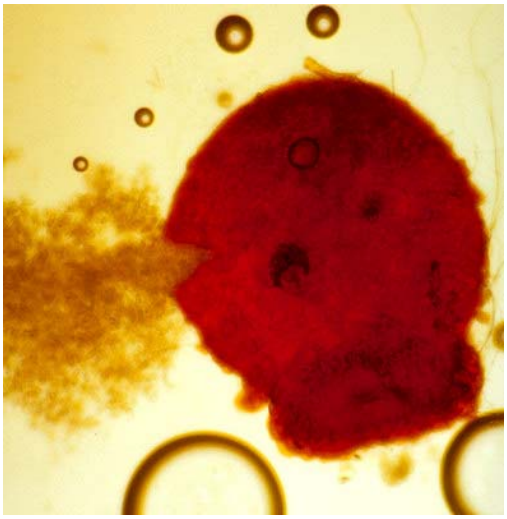

Photo 2. A perithecium (sexual fruiting body) of *Neonectria galligena* from bark canker tissue
http://www.botany.hawaii.edu/

Photo 3. Ascospores of *Neonectria galligena*.
http://plante-doktor.dk/frugttraekraeft.htm

Infection is influenced by temperature. In vitro studies showed that the optimum temperature for germination of conidia is between 20-25°C, with minimum and maximum limits of 6°C and 32°C, respectively (Latorre *et al.* 2002). Latorre *et al.* (2002) also noted that at 20°C, ascospores germinated 2.3 times more rapidly than conidia. Varying climate therefore not only has an effect on spore production and dispersal, but also has an effect on the importance of ascospores versus conidia as a source of inoculum. Conidia are the most important form of inoculum in California and Chile (Grove 1990; Lolas and Latorre 1996), and were the only spore type observed in Tasmania (Ransom 1997). In the UK, ascospores are the major cause of new infection (Lortie 1964; Swinburne 1975). Munson's (1939) observations on ascospore discharge and the susceptibility of freshly exposed leaf scars led to the assumption that autumn leaf fall is the most critical period for infection in the UK. The effect of temperature and wetness duration on spore infection for European canker of apple and perennial canker of woody plants is discussed in detail in the modelling section of this report.

The fungus enters its host through leaf scars or wounds caused by pruning, insect feeding, winter injury or invasion by other pathogens (Swinburne *et al.* 1975). Frost and crotch cracks are also common sites of entry. Plants that are stressed by cold, drought, mechanical injuries or other disease are especially susceptible. Infections may be worse in autumn and winter when the host plant is dormant and wound recovery is weaker than in the growing season. *N. galligena* can also infect apple and pears via the calyx, lenticels and wounds (Swinburne 1971b).

Control

Effective control of European canker relies upon physical removal of infected material and prevention of new infections. Reduction of inoculum (conidia) on wood has been achieved with fungicide sprays applied during key periods of the year (Swinburne *et al.* 1975). The effect of apple scab fungicide programs on control of European canker of apples has been widely studied (Cooke 1999, Swinburne 1975). Spring-summer fungicide programs, applied for the control of apple scab, reduced numbers of new cankers on young apple trees cv. Bramley's Seedling by between 65% and 76% compared with the untreated control (Cooke 1999). Autumn application of copper oxychloride at 5% and 50% leaf-fall further reduced the number of new cankers. Spring-summer fungicide programs that included benzimidazole fungicides (Cooke 1999) also reduced the number of fruit rots in storage. Xu and Butt (1996) reported that curative fungicide sprays were ineffective in preventing canker development on potted apple trees with pruning cuts when applied 48 or 36 h after inoculation with *N. galligena*. The only effective control measure for established infections is removal by pruning.

Symptoms
Woody tissue

The formation of cankers on woody tissue is the most obvious symptom of *Neonectria galligena.* In the early stages of infection however, no external symptoms may be visible. This initial infection often occurs in autumn with visible disease symptoms developing in the spring and summer (Munson 1939). However, in young apple trees infected by *N. galligena* at the propagation phase, the disease has been shown to remain latent for up to 3 years (McCracken *et al.* 2003b). Young developing cankers appear as reddish brown lesions. These lesions soon elongate into elliptical, sunken areas with the necrotic tissue inside appearing water-soaked. Young cankers are often not noticed until other symptoms develop.

The first easily visible signs of *N. galligena* are small cream-coloured fungal fruiting bodies (sporodochia) in the young canker and the development of callus tissue.

N. galligena invades the healthy tissue around the canker and the plant responds with the formation of new closely packed ridges of callus. Necrotic outer bark, phloem and cambial cells at the margins of the canker eventually break away exposing a ring of callus tissue. In cankers of Manchurian Ash in Japan, this tissue was composed mainly of irregular axial parenchyma cells and fewer, narrow, distorted xylem vessels in comparison to healthy tissue (Sakamoto 2004). The alternating fungal growth and callus ridge formation, which continues over subsequent years, results in the characteristic target-like cankers (Figure 2). This kind of canker is referred to as an open canker. Open cankers are formed when conditions are favourable for the host and the fungus is slow growing (Agrios 1997).

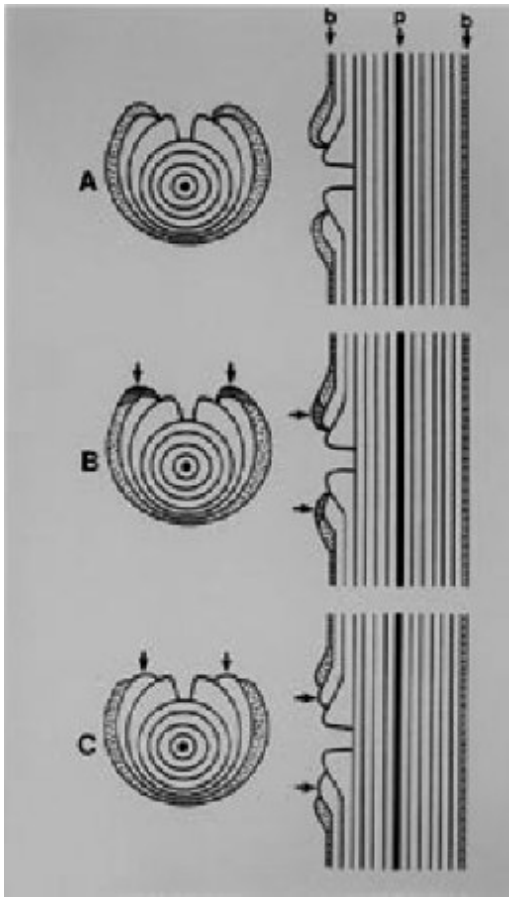

Figure 2. Diagrams of the formation of the target-like canker on woody tissue (Sakamoto 2004).

A: Wide growth increments of the xylem and phloem are formed simultaneously.
B: The edges of the phloem become necrotic (arrows).
C: The edges of the wide growth zones of the xylem become exposed and visible (arrows).

Closed cankers occur when the conditions favour the growth of *N. galligena,* allowing rapid growth of the fungus. Successive zones of callus tissue are further apart and do not fall off (Agrios 1997). The outer bark is rough and cracked but does not fall off for several years in these cankers.

As cankers enlarge, they girdle infected twigs and branches, causing death of the shoot or branch. Cankers on the main stem of older trees reduce the vigour and the value or productivity of the tree. These trees are also subject to wind breakage. A decrease in water conductivity in early spring due to distorted and narrow xylem, especially on branches and stems with numerous or large cankers, is one of the reasons for poor vigour or dieback of seriously affected trees (Sakamoto 2004).

N. galligena overwinters as perithecia or mycelium in the canker tissue. The easily identifiable perithecia are small, round, red granulations that occur in the folds of 3 – 4 year old cankers.

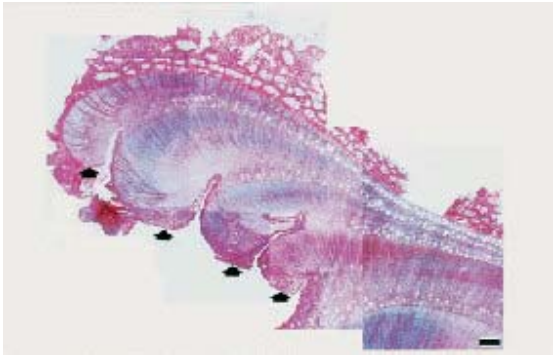

Photo 4 Transverse view of a canker in the woody tissue of Manchurian Ash. Arrows indicate the concentric rings of the target-like structure (Sakamoto 2004).

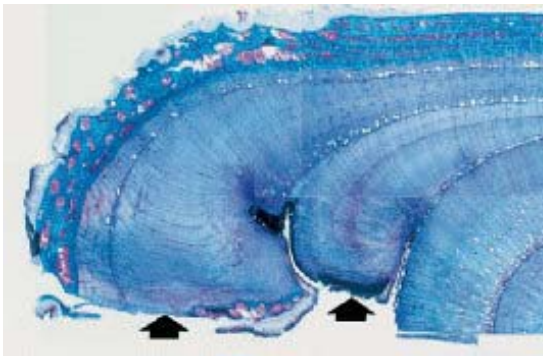

Photo 5 Transverse view of the canker in the woody tissue of Manchurian Ash approximately two years after inoculation. Arrows indicate the concentric rings of the target-like structure (Sakamoto 2004).

Fruit

Depending on the apple variety, symptoms of fruit infection are generally not observed until shortly before or after harvest (Salmon and Wormald 1915) or after storage (Swinburne 1971b). The most obvious symptom of *N. galligena* fruit infection is a brown rot characterised by circular, sunken necrotic areas on the surface of the fruit. If infection has occurred at the calyx end, the developing rot is called 'eye rot'. Lenticel infection and core rot also occur (Ogawa and English 1991). Core rots are common in open sinus varieties of apples and are undetectable until the fruit is cut open. The symptoms are indistinguishable from those caused by other rot pathogens and the causal organism can only be confirmed once grown onto culture media (McCracken, pers. comm. 2006). Internally, the rotted tissue is soft and has a striated appearance (Agrios 1997). White or yellow conidial masses may form on the rotted areas during the later stages of fruit infection (Agrios 1997; Jones and Aldwinkle 1997). This is the imperfect stage, *Cylindrocarpon heteronema,* of the fungus. Infected fruit left on the tree or the orchard floor can become mummified, forming perithecia during the following winter that provide a source of inoculum whenever conditions are conducive for ascospore release (Dillon-Western 1927, Wormald 1955, Ogawa and English 1991).

Symptoms on apple

Photo 6. Canker on apple caused by *Nectria galligena*. (Courtesy A. L. Jones).http://www.apsnet.org/Education/IntroPlantPath/Topics/plantdisease/text/fig

Photo 8. Eye rot in storage caused by *N. galligena*
http://www.bayercropscience.de/de/pf/diagnose_center/online_diagnose/index.asp?ID=0&detail=true&ID_DIAG=861

Photo 10. European Canker in crotch of limb (*Nectria galligena Bres.)*
http://www.nsapples.com/images/photoid/eurocrnk.jpg

Photo 7. Nectria Canker (European Canker) Dead branch beyond the swollen canker. (Photo by Jay Pscheidt, 1992) .http://plant-disease.ippc.orst.edu

Photo 9. Nectria canker girdling branch.
http://plante-doktor.dk/frugttraekraeft.htm

Photo 11. Distal end of branch dying due to girdling by *Nectria* canker (*Nectria galligena*)
http://plante-doktor.dk/frugttraekraeft.htm

Photo 12. Infection of crotch and main stem of apple trees by *Neonectria galligena.*
http://www.pometet.kvl.dk/Aeblehistorier/Aeblesygdomme.htm

Photo 13. Branch girdled by canker (*Neonectria galligena*)
http://www.bongerdgrooteveen.nl/Home.htm

Photo 14. Apple with nectria canker (*Neonectria galligena)*
http://www.science.oregonstate.edu/bpp/Plant_Clinic/images/apple_nectria.htm

Photo 15. Apple with nectria canker (*Neonectria galligena)*
http://www.science.oregonstate.edu/bpp/Plant_Clinic/images/apple_nectria.htm

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Photo 17. Nectria canker on an Empire tree. http://www.nysaes.cornell.edu/ent/scaffolds/2004/040719F3.html

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Photo 19. Close up of perithecia (sexual fruiting structures) of *N. galligena*. http://plante-doktor.dk/frugttraekraeft.htm

Symptoms on other woody hosts

Photo 20. Nectria canker on a black walnut trunk, caused by *N. galligena*. www.apsnet.org/online/Archive/1998/pdcvr15.htm

Photo 21. Fruit tree canker caused by *Neonectria galligena* http://www.bayercropscience.de/de/pf/diagnose_center/online_diagnose/index.asp?ID=0&detail=true&ID_DIAG=855

Photo 22. Nectria canker on a black walnut trunk, caused by *Neonectria galligena*. www.pfc.forestry.ca/.../Canker/canker7_e.html

Photo 23. Nectria canker caused by *Neonectria galligena*.
http://www.forestryimages.org/browse/subthumb.cfm?sub=570&start=1

Photo 24. *Neonectria galligena* in a tree trunk in Germany
http://fets3.freetranslation.com/?Url=http%3A%2F%2Fwww.aufm-berg.de%2FAktuelles%2Fhauptteil_aktuelles.html&Language=German%2FEnglish&Sequence=core

Photo 25. *Neonectria galligena*
www.cientic.com/tema_fungo_img5.html

Photo 26. Nectria canker on honey locust. Photo: Robert Blanchette
http://www.extension.umn.edu/yardandgarden/ygbriefs/p-nectriahoneylocust.html

Photo 27. Nectria canker on oak.
http://www.digitalarborist.com/pubs/oakpests/images/diseases/canker64.jpg

Photo 28. Target canker on a paper birch tree is caused by *Neonectria galligena*
http://www.treedictionary.com/DICT2003/hardtoget/ntb29/pg_51-75/index.html

Photo 29. Nectria Canker on maple caused by *Neonectria galligena.*
http://www.extension.umn.edu/projects/yardandgarden/diagnostics/maplenectria-pict.html

Photo 30. Nectria canker on birch in the United States caused by *N. galligena.*
http://www.forestryimages.org/browse/detail.cfm?imgnum=1198002

Photo 31. Bark in crown of West Indian mahogany (*Swietenia mahogoni*) infected by *N. galligena*. http://www.forestryimages.org/browse/detail.cfm?imgnum=4825027

Photo 32. Cankers caused by *N. galligena.* http://www.fao.org/docrep/007/y5041e/y5041e09.htm

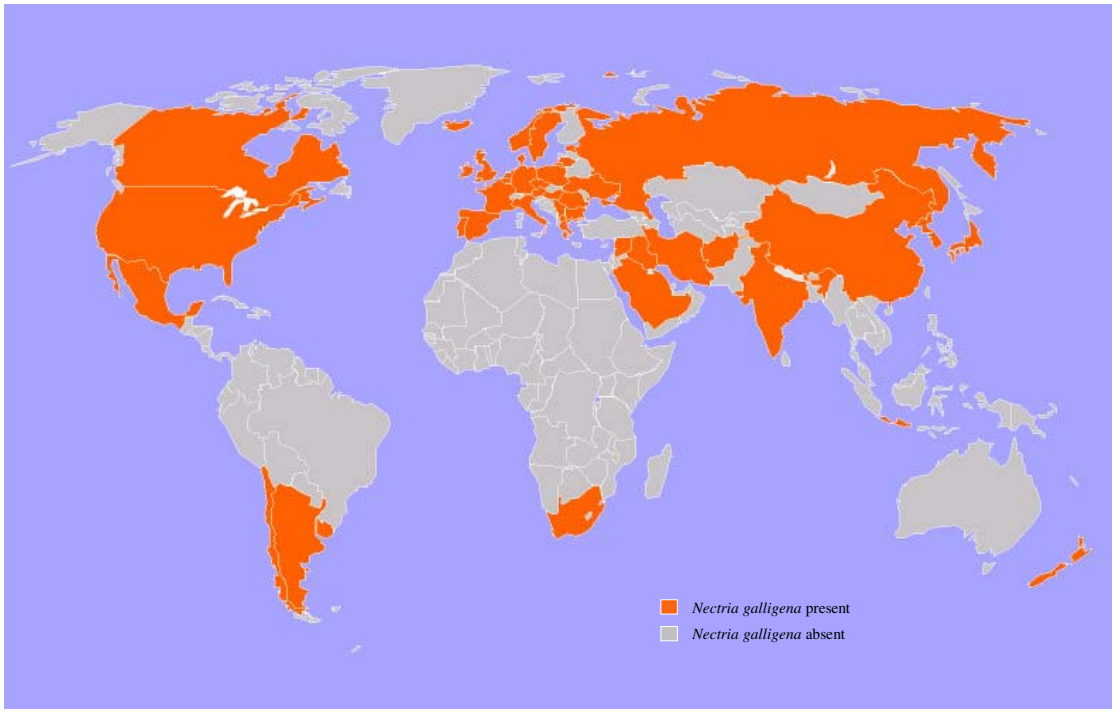
Host range and distribution of *N. galligena* worldwide

N. galligena has been recorded on more than 60 tree and shrub species from over 20 genera (Flack and Swinburne 1977) in all continents except Antartica (CABI). The IRA review (2005) indicated that *N. galligena* needs a moist temperate environment, in particular annual rainfall higher than 1000 mm, to cause disease. However, herbarium records of *N. galligena* show that, when all hosts are considered, the pathogen has been reported from very diverse environments, ranging from sub-artic Iceland and Canada to tropical Java, and also in hot dry countries such as Iran, Syria and Saudia Arabia (CABI).

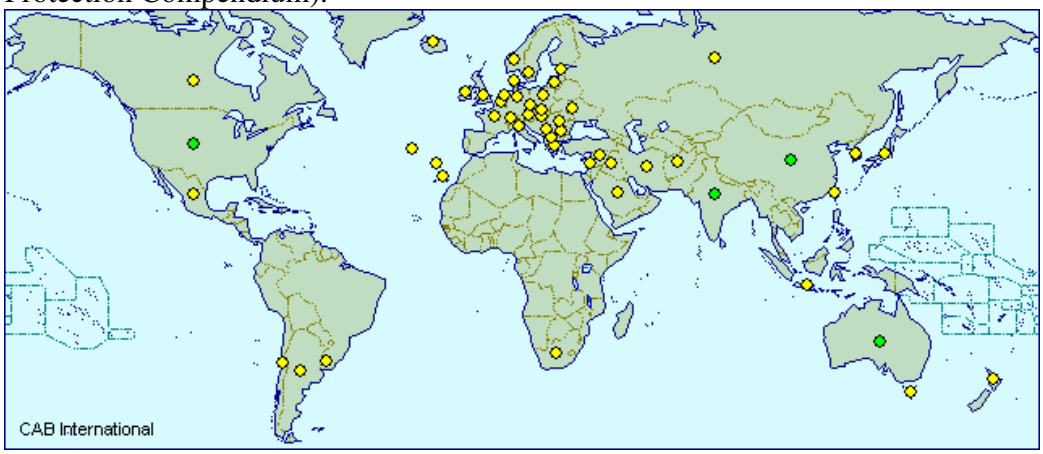
Apple (*Malus domestica)* is the most susceptible host for *N. galligena* but cultivars differ in susceptibility (Braun 1997, McCracken pers. comm). Other hosts of horticultural importance include European pear (*Pyrus communis*), Asian pear (*Pyrus pyrifolia*), loquat (*Eriobotrya japonica*) and English walnut (*Juglans regia*) (Zeller and Owens 1921, Zeller 1926, Wormald 1942, Grand 1985). *N. galligena* also causes perennial cankers on a range of forest trees and shrubs worldwide (Braun 1997, Flack and Swinburne 1977, Plante *et al.* 2002), including many commonly found in windbreaks and gardens around orchards such as hawthorn (*Crataegus* spp), oak (*Quercus* spp), birch (*Betula* spp), elm (*Ulmus* spp) and maple (*Acer* spp). Other woody plants susceptible to *N. galligena* include crab apple (*Malus* spp), beech (*Fagus* spp), ash (*Fraxinus* spp), mountain ash (*Sorbus aucuparia*), walnut (*Juglans* spp), poplar (*Populus* spp), willow (*Salix* spp), eastern redbud (*Cercis canadensis*) and mahogany (*Swietenia mahagoni*) (Anagnostakis and Ferrandino 1988, Barnard *et al.* 1988, Ng and Roberts 1974).

In New Zealand, *N. galligena* has been isolated from the following hosts: *Coprosma areolata*, *C. lucida*, *Eriobotrya japonica*, *Malus pumila*, *M. sylvestris*, *M. domestica*, *Pyrus communis*, *P. pyrifolia* and *Sophora microphylla* (http://nzfungi.landcareresearch.co.nz/). Of these, *C. areolata, C. lucida* and *S. microphylla*, known as Karamu and Kowhai, are endemic to New Zealand, demonstrating that *N. galligena* can move from apple onto the surrounding native flora. Host information is not always available for herbaria specimens, however, and in the case of specimen PDD 31890, collected in 1973 in Auckland, the record does not give a host but states that the fungus was associated with an insect.

N. galligena causes economic losses to timber industries due to cankers reducing log quality and value (Plante *et al.* 2002). For example, in North America the pathogen is associated with beech (*Fagus grandifolia*) bark disease, a disease complex initiated by the beech scale insect, *Cryptoccocus fagisuga,* which predisposes *F. grandifolia* to invasion by *N. galligena* and the related *N. coccinea* var. *faginata*, causing a diffuse canker on beech trees (Houston 1994, Plante *et al.* 2002). *N. galligena* has also been isolated from stem galls on *Cercis canadensis*, wood lesions on *Swietenia mahagoni*, and trunk cankers on *Quercus laurifolia* and *Acer rubrum* in Florida (Barnard *et al*. 1988).



Map 1. Worldwide distribution of *Neonectria galligena*. (Information sourced from the Crop Protection Compendium).



Map 2 Countries where *Neonectria galligena* has been recorded. (on-line Crop Protection Compendium, CAB International).

Table 1. Worldwide records of *N. galligena* (Crop Protection Compendium, CABI)

Country	Province	Reference
Asia		
Afghanistan		CMI, 1985
China		
	Taiwan	CMI, 1985
India		
	Himachal Pradesh	CMI, 1985
Indonesia		
	Java	CMI, 1985
Iran		CMI, 1985
Iraq		CMI, 1985
Japan		CMI, 1985

Korea, Republic of		CMI, 1985
Lebanon		CMI, 1985
Saudi Arabia		Ramadani & Aggab, 1993
Syria		CMI, 1985
Europe		
Austria		CMI, 1985
Belgium		CMI, 1985
Bulgaria		CMI, 1985
Czechoslovakia (former -)		CMI, 1985
Denmark		CMI, 1985
Estonia		CMI, 1985
Faroe Islands		CMI, 1985
France		CMI, 1985
Germany		CMI, 1985
Greece		CMI, 1985
Hungary		CMI, 1985
Iceland		CMI, 1985
Ireland		CMI, 1985
Italy		CMI, 1985
Lithuania		CMI, 1985
Macedonia		CMI, 1985
Netherlands		CMI, 1985
Norway		CMI, 1985
Poland		CMI, 1985
Portugal		de Sousa & Avelar, 1988
	Azores	Gardner & Hodges, 1990
	Madeira	Gardner & Hodges, 1990
Romania		CMI, 1985
Russian Federation		CMI, 1985
Serbia and Montenegro		Susuri, 1988
Slovakia		Stafancik *et al.* 1996
Spain		CMI, 1985
	Canary Islands	Gardner & Hodges, 1990
Sweden		CMI, 1985
Switzerland		CMI, 1985
Ukraine		CMI, 1985
United Kingdom		CMI, 1985
Africa		
South Africa		CMI, 1985
North America		
Canada		CAB Abstracts
	British Columbia	CMI, 1985
	New Brunswick	CMI, 1985
	Nova Scotia	CMI, 1985
	Ontario	CMI, 1985
	Prince Edward Island	CMI, 1985
	Quebec	CMI, 1985
Mexico		CMI, 1985
USA		
	California	CMI, 1985
	Connecticut	CMI, 1985

	Florida	Barnard *et al.* 1988
	Illinois	CMI, 1985
	Indiana	CMI, 1985
	Maine	CMI, 1985
	Maryland	CMI, 1985
	Massachusetts	CMI, 1985
	Michigan	Thomas & Hart, 1986
	Minnesota	CMI, 1985
	Mississippi	CMI, 1985
	New Hampshire	CMI, 1985
	New York	CMI, 1985
	North Carolina	CMI, 1985
	North Dakota	CMI, 1985
	Oregon	CMI, 1985
	Pennsylvania	CMI, 1985
	Rhode Island	CMI, 1985
	South Dakota	CMI, 1985
	Vermont	Houston, 1994
	Virginia	CMI, 1985
	Washington	CMI, 1985
	West Virginia	CMI, 1985
South America		
Argentina		CMI, 1985
Chile		CMI, 1985
Uruguay		CMI, 1985
Oceania		
Australia		
	Tasmania – now eradicated	CMI, 1985 Ransom 1997
New Zealand		CMI, 1985

Host specificity

Host specialisation studies have noted differences in both host range and symptoms produced by isolates of *N. galligena* recovered from different hosts (Barnard *et al.* 1988, Flack and Swinburne 1977). In Europe, Flack and Swinburne (1977) in cross-inoculation experiments showed that *N. galligena* isolated from typical apple cankers on Bramley's Seedling induced lesions on poplar, hawthorn and beech trees but not on ash or sycamore trees. Isolates from lesions on ash trees induced cankers on apple, hawthorn, poplar and beech but the lesions on apple trees were atypical. Flack and Swinburne (1977) found that although isolates from ash were able to induce cankers on Bramley's Seedling, the symptoms were morphological different from those caused by isolates from apple, which were not pathogenic to ash. Poplar, hawthorn and beech trees are commonly used as windbreaks and in hedgerows around orchards, providing external sources of inoculum for infection of apples trees (Flack and Swinburne 1977). In Florida, Barnard *et al.* (1988) reported little evidence of host specificity among four isolates of *N. galligena* isolated from four tree hosts (*Cercis canadensis*, *Swietenia mahagoni*, *Quercus laurifolia* and *Acer rubrum*).

Distribution of potential hosts of *N. galligena* in Australia

There is a broad distribution of potential hosts recorded as susceptible to *N. galligena* across Australia (Maps 3-7). These include fruit crops (specifically *Malus* spp and *Pyrus* spp.), walnut (*Juglans* spp), ornamentals and other woody plants commonly grown in gardens and parkland across Australia. Trees include oak (*Quercus* spp), birch (*Betula* spp), elm (*Ulmus* spp), maple (*Acer* spp), crab apple (*Malus* spp), beech (*Fagus* sp), ash (*Fraxinus* sp), mountain ash (*Sorbus aucuparia*), poplar (*Populus* sp), hawthorn (*Crataegus* spp) and willow (*Salix alba* and *S. purpurea*)(see distribution maps).

Juglans regia (English walnut), a recorded host of *N. galligena* (Grand 1985), is grown commercially in Australia (Map 5). The Australian walnut industry is a small but fast growing industry with current annual walnut production approximately 500 tonnes (in shell) with a farm gate value of 2.5 million dollars annually (Kenez (AWIA) pers comm.).

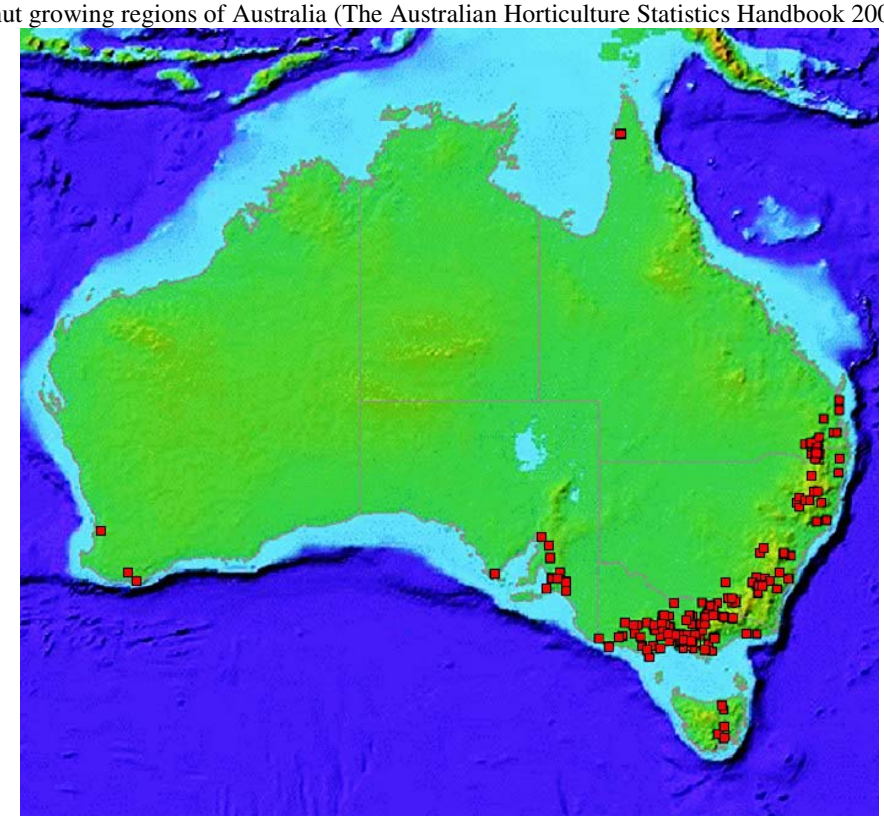
The State of Victoria prides itself on being the Garden State, and avenues of mature elm trees are a major tourist attraction in the city of Melbourne. Since the outbreak of Dutch Elm disease in Europe and North America (a disease not present in Australia), Melbourne's elms are thought to be the most significant in the world. The Melbourne City Council has valued the 6500 amenity elm trees at $10,000 each, and the IRA report (2004) concluded that an outbreak of European canker could be highly significant at the local level.

N. galligena was not found on any Australian native plants prior to its eradication in Tasmania (Ransom 1997). However, given that the broad host range includes the endemic New Zealand flora Karamu (*Coprosma lucida*), *Coprosma areolata* and Kowhai (*Sophora microphylla*), it is likely that at least some species of Australian native and amenity plants will be susceptible to *N. galligena.* The New Zealand specimens of *N. galligena* on the native hosts were collected from the Coromandel Peninsula (http://nzfungi.landcareresearch.co.nz/html/data_collections.asp?), away from commercial apple production regions, indicating that the pathogen is capable of spreading into bushland if the environmental conditions are favourable and suitable hosts are present. Herbarium records (Australian Plant Pest Database) show that many endemic genera, including *Eucalyptus, Grevillea, Banksia*, *Macadamia, Melaleuca, Nothofagus* and *Waratah,* have species that are susceptible to other *Nectria* species currently in Australia.

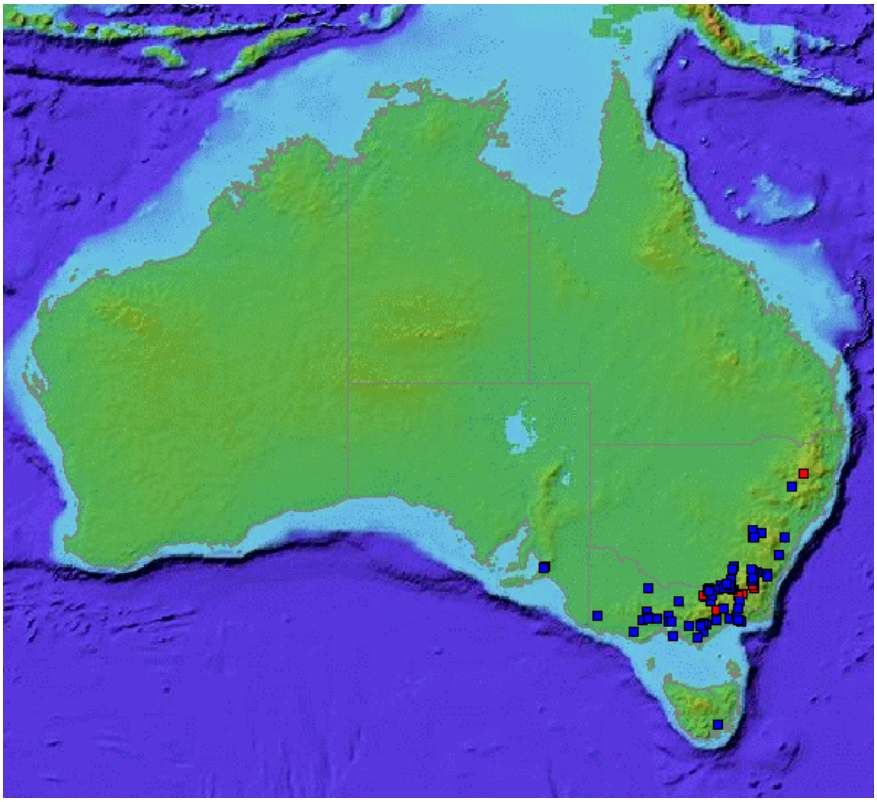
Map 3. Apple growing regions of Australia (The Australian Horticulture Statistics Handbook 2004).

Map 4. Pear growing regions of Australia (The Australian Horticulture Statistics Handbook 2004).

Map 5. Walnut growing regions of Australia (The Australian Horticulture Statistics Handbook 2004).



Map 6. Distribution of *Acer* spp, *Betula* spp, *Malus* spp, *Quercus* spp, *Ulmus* spp, *Fagus* sp, *Fraxinus* sp, *Populus* sp, *Sorbus aucuparia* and *Crataegus* sp across Australia. http://www.rbg.vic.gov.au/cgi-bin/avhpublic/avh.cgi



Map 7. Distribution of *Salix alba* (blue) and *Salix purpurea* (red) across Australia.
http://www.rbg.vic.gov.au/cgi-bin/avhpublic/avh.cgi

The role of nursery and orchard infections in disease spread

There is evidence for the spread of *N. galligena* from nurseries to new orchards and between orchards. Infection can be spread during grafting via conidia contaminating the budwood (Howard *et al.* 1974) and via symptomless infected planting material (Brown *et al.* 1994, Berri *et al.* 2000, Lovelidge 2003, McCracken *et al.* 2003a, McCracken *et al.* 2003b). The Millenium project (McCracken *et al.* 2003b) investigated the importance of nursery infection versus disease spread from adjacent infected orchards in subsequent canker development. Approximately 5% of infection in young orchards could be associated with infected planting material, but under high disease pressure inoculum movement from infected orchards was more important than nursery infection for disease spread. Infection occurring during the grafting stage of propagation remained latent in young apple trees for up to 4 years, and was found to be an important mechanism for the spread of disease between countries and growing regions (McCracken *et al.* 2003b). The New Zealand Pipfruit IFP fact sheet 9 indicated that infection spreads to new orchards from nearby infected trees and from nursery infection.

Disease resistance

Infection and subsequent canker development in the orchard are influenced by many factors: climate, cultivar, rootstock, soil type and agronomic practices such as pruning and fertiliser regimes (Swinburne 1975). *N. galligena* spreads within and between orchards by means of spores (asexual conidia and sexual ascospores) dispersed during rainy and windy weather conditions (Swinburne 1975). Spores infect through natural wounds such as leaf scars or artificial wounds such as pruning cuts. Infection can take place all year round. In Ireland, autumn leaf fall is believed to be the main time of tree infection resulting in dieback of young shoots in the following spring and summer. All apple cultivars appear to be susceptible to canker to some degree but some tolerance to the wood canker phase of *N. galligena* has been noted in some cultivars (Grove 1990). Important apple varieties such as Golden Delicious, Jonathan, Rome Beauty, Cox's Orange Pippin, McIntosh, Red Delicious are listed as susceptible (Grove 1990). Nichols and Wilson (1956) reported that apple varieties most commonly affected by the wood canker phase of European canker in California were Gravenstein and red sports of Delicious. Braun (1997) found 19.6% of trees (8132 apple trees surveyed) were infected with *N. galligena* in Kings County, Nova Scotia. The incidence of European canker was greatest on cvs. Red Delicious, McIntosh, Northern Spy and Idared (>30%) and significantly lower on cvs. Golden Russett and Gloster (<10%). In a controlled study in Holland, Weg *et al.* (1992) reported that Cox's Orange Pippin and James Grieve developed smaller cankers than Golden Delicious, Elstar, Summerred, Jonathan and Lombart's Calville. In another controlled study, Groenwold *et al.* (1994) found that of 41 varieties and 17 selections tested for resistance to canker (*N. galligena*) in the greenhouse, only Jonathan, Ellis Bitter, Lombarts Calville, Clozeau and Golden Delicious were classed as weakly susceptible, the rest being either moderately or highly susceptible.

The role of latent infection in fruit for disease spread
Issues raised in the IRA report

The IRA report (2005) analysed the possibility that picked fruit could carry symptomless (ie latent) infections of *N. galligena*. The New Zealand Pipfruit IFP manual (Fact Sheet 9) states that *N. galligena* does cause fruit rot but that this is not usually a problem in New Zealand. The IRA report concluded that under New Zealand conditions, although fruit is rarely infected, when fruit infection does occur, rot develops rapidly and the affected fruit would either not be harvested or would be culled out during the grading process. However, the report conceded that if latent infections were present, they would not be detectable along the supply

chain. An estimate of 0.0069% probability was given for importation of fruit with latent infections from the total proposed number of apples imported from New Zealand annually, translating into 69 out of every 10,000 apples.

The specific issues raised in the IRA report around the potential for importation of apple fruit with latent (symptomless) infections not expressed at harvest or during distribution include:

- That European canker is mainly a disease of branches and twigs.
- That regular fungicide sprays for apple scab would reduce the risk of fruit infection.
- That the apple varieties involved would be dessert varieties and therefore express disease symptoms at or before harvest and fruit would be discarded.
- Whether contamination of clean fruit could occur during processing in the packing house.
- Whether packhouse procedures would eliminate latent and superficial infections.

The section below reviews the state of knowledge regarding the process of fruit infection and latency periods of apple.

The process of fruit infection in the field

The mechanisms of fruit infection in the field were studied extensively several decades ago in Europe (Bondoux and Bulit 1959, Swinburne 1971a, Swinburne 1975). In general, fruit infection occurs on the tree when spores are dislodged from cankers and land on fruit. The spores infect through openings such as the calyx, sinus, lenticels, scab lesions and wounds caused by insects. The incidence of fruit infection depends on the amount of sporulation occurring on tree cankers and on weather conditions (Bondoux and Bulit 1959, Swinburne 1975). Rotting of fruit can occur while the fruit is still on the tree, such fruit becomes mummified and perithecia (sexual fruiting structures) are formed over winter. These become a source of inoculum by releasing ascospores in spring. The earliest record of fruit rotting is of dessert varieties in Europe during the 1930's (Swinburne 1964). Fruit infection of some apple varieties, such as Cox's Orange Pippin, results in fruit rotting ('eye rot') before harvest. Others, particularly those with an open sinus, may develop a core rot that is difficult to detect without cutting open the fruit (McCracken, pers. comm. 2006). The culinary cultivar, Bramley's Seedling, may remain symptomless for many months in long term cold storage before rots become evident, despite high levels of the wood canker phase of *N. galligena* in the orchard (Swinburne 1975, Cooke 1999).

Latent infection in fruit

There are many reports of latent infection in apple fruit (Bondoux and Bulit 1959, Brown and Swinburne 1971, Snowdon 1990, Swinburne 1973, Swinburne 1971a, Swinburne 1975, Saindrenan and Bompeix 1982, Nobel and Drysdale 1983, Seng *et al.* 1985). The latency period differs between apple varieties and has been linked to the production of antifungal compounds in infected fruit (Brown and Swinburne 1971, Swinburne and Cartwright 1973, Saindrenan and Bompeix 1982, Nobel and Drysdale 1983, Seng *et al.* 1985). Swinburne (1971a) was one of the first to observe that benzoic acid played a key role in latent infection. Benzoic acid inhibits growth of *N. galligena* in the acid conditions of unripe apple fruit, but as acidity decreases and sugar content increases during ripening, the fungus is able to metabolise benzoic acid into less toxic substances, and eventually into CO_2 (Swinburne 1973). Therefore, at lower acidity the fungus resumes growth, rotting the fruit. Saindrenan and Bompeix (1982) studied the levels of benzoic acid production in immature apples of different cultivars in

response to infection by *N. galligena*. The range of benzoic acid produced varied from 3.3 mg/100g tissue in *cv*. Winston to 74.6 mg/100mg tissue in *cv*. Scarlett. Cooking apples such as Bramley's Seedling produce high levels of benzoic acid, whereas dessert apples and ripe fruit produce less. Fruit infection remains latent in Bramley's Seedling for up to 3-7 months storage (Bondoux and Bulit 1959, Swinburne 1971a, Swinburne 1975). Concentrations of CO_2 greater than 2% in storage progressively reduce benzoic acid levels in infected fruit (Swinburne 1974).

Production of benzoic acid and the resultant latency periods for *N. galligena* in apples commonly grown in New Zealand and Australia such as Royal Gala, Pacific Rose, Braeburn, Granny Smith and Fuji have not been studied.

Losses in storage due to latent infection of fruit

There have been several estimates of loss caused by *N. galligena* during storage. In Northern Ireland, losses in culinary cultivar Bramley's Seedling varied from 3-60% depending on the type of storage (Swinburne 1964, Swinburne 1970). In California, losses of 10-60% were reported for cultivars Delicious, Golden Delicious, Jonathan, Rome Beauty and Tomkins King (McCartney 1967). In central Europe, Puia *et al.* (2003) reported that Neonectria rots were identified in Jonathan but not Golden Delicious fruit stored for five months. In France, 0.5% and 2% of stored Reinettes du mans and Reinettes blanches du Canada fruit developed rots attributable to *N. galligena* (Bondoux and Bulit 1959).

When and how the fruit was initially infected, the amount of inoculum and the depth of the inoculum entry point all affect the development of fruit rot in storage (Swinburne 1971a, Brown pers comm. 2006). However, the longer fruit is held in storage, the more time is available for rots to develop. A survey of storage rots in the UK undertaken by Berrie (1997) demonstrated that losses due to all fruit rots (*Botrytis, Phytophthora, Monilinia, Neonectria, Gloeosporium* and *Penicillium*) increased with the length of time in storage. Losses in Cox's Orange Pippin were 0.5 – 9.8% in samples marketed in December and 0.7 – 17.0% in samples marketed in March. Losses specifically due to *N. galligena* were more prominent in the late-stored samples (March). Orchard site was also a contributing factor to the variation (Berrie 1997).

Potential for infection in the packing house

The IRA report analysed the potential sources and mechanisms for contamination of clean fruit during picking and transport to the packing house, the likelihood of contamination during processing and of *N. galligena* surviving routine processing procedures. The IRA report concluded that:

- The estimate of risk calculated allows for some fruit to be contaminated in the packing house but also recognises that conditions in most areas of New Zealand during the harvesting season are not favourable for spore production.
- The estimate of risk takes into account that internal or latent infections are unlikely to be visible and none of the processes in the packing house are likely to substantially reduce these infections.
- The estimate of risk calculated allows for the presence of a small number of spores in the packing process that could contaminate fruit.

The report concluded that the leaves of apples are not infected by *N. galligena*, infected twigs will have been removed during the grading process, and loose spores are unlikely to be present in sufficient quantities or remain viable long enough to contaminate fruit in the packing house.

It is unlikely that conditions in storage (temperature) would be conducive for contamination and subsequent infection of fruit. However, it is disputable that leaf infections do not occur and are therefore not a contamination issue. Infected leaves and petioles of apple and pear have been observed in the UK (Wormald 1955) and in California (Ogawa and English 1991), and this type of material is commonly found in bulk bins of fruit.

Infected fruit as a source of inoculum

Mummified infected fruit have been documented as a source of inoculum (Dillon-Western 1927, Wormald 1955, Ogawa and English 1991), but little research has been conducted to determine their importance in disease spread. Spores have been associated with insects (Houston 1994, Agrios 1997, http://nzfungi.landcareresearch.co.nz/html/data_collections.asp?), but no research has been conducted to determine whether the spores came from infected fruit or wood cankers. The question of whether discarded rotten apples or apple cores can be a source of inoculum has not been addressed. Can infection be transferred from infected fruit to apple seed? Weed apples grown from discarded apple cores are common, particularly along roadsides, yet we could not find any reference as to whether such trees are infected if the seed originated from an infected apple. Three New Zealand native species growing on the Coromandel Peninsula are known be hosts of *N. galligena*. Where was the source of inoculum for these plants? To date, research efforts have concentrated on wood cankers as the source of inoculum and information regarding the importance or otherwise of fruit as a source of incoculum are lacking.

Detecting infections in host plants

Currently there are no reliable, robust methods for detecting *Neonectria galligena* infections in symptomless wood or fruit tissue (McCracken 2003b, McCracken pers comm.).

Several techniques have been used to detect *N. galligena* in cankers. Anagnostakis and Ferrandino (1988) used Granny Smith apples as baits to isolate pure cultures of *N. galligena* from canker lesions on sweet birch. *N. galligena* was detected in xylem tissue using immunofluorescent techniques (Dewey *et al.* 1995). PCR methods have been developed which allow the molecular detection of *N. galligena* in lignified woody tissue (Langrell and Barbara 2001, Langrell 2002).

Molecular techniques have been used to differentiate genotypes of *N. galligena* isolated from apple tree cankers (Berrie *et al.* 2000, McCracken *et al.* 2003a). Genotypic differences between isolates collected from orchards in Northern Ireland and England showed that the populations were different between the geographic areas (McCracken *et al.* 2003a), and were useful to track the source of infections in young apple trees (Brown *et al.* 1994). Plant *et al.* (2002) used RAPD and ribosomal DNA (18S rDNA) polymorphisms to distinguish genetic variability in North American populations of *N. galligena* and *N. coccinea* var. *faginata* in forest trees.

To date, none of these tests have been developed for use in detecting latent infection in fruit or for high throughput purposes as would be required for random testing of imported produce. All have been designed for identification of the pathogen from symptomatic tissue, and with the exception of the method described by Langrell and Barbara (2001), rely on isolation of the pathogen into culture media as a first step.

The effect of rainfall on disease spread and incidence
Issues analysed in the IRA report in relation to spread of *N. galligena*

The IRA report (2005) analysed the possibility of infected fruit providing an entry pathway for *N. galligena* into Australia. The main issues included probability of importation of the pathogen, exposure to susceptible hosts at various utility points, and the probability of establishment and spread of disease. Estimates of partial probabilities of entry, establishment and spread for utility points and susceptible host plants were combined to give an annual probability of entry, establishment and spread of 0.33%. The IRA report considered the effect that rainfall intensity and distribution would have on disease incidence (wood and fruit) and spread at the point of sourcing apples for export (New Zealand) and at the proposed destination point (Australia). European canker is reported to occur in all major apple-growing regions of New Zealand (http://nzfungi.landcareresearch.com.nz/images/maps). These regions include Whangarei, Auckland, Gisborne, Hawke's Bay, Waikato, Bay of Plenty and Nelson. The New Zealand Pipfruit IFP manual (fact sheet 9) states that European canker of apples is only a problem in areas where rainfall is >1000 mm, particularly Auckland and Waikato and periodically Nelson. The IRA report uses the amount of annual rainfall as the criterion to identify apple-growing regions of New Zealand where European canker (wood and fruit infections) is unlikely to be a problem. The use of annual rainfall for identifying regions where the potential risk of fruit infections by *N. galligena* could be low or where environmental conditions may not favour disease establishment is debatable. The potential risk of disease establishment and fruit infections depends on the amount of rainfall and more specifically wetness periods occurring during periods when the host is most susceptible to infections by *N. galligena*. There are many examples of localities with annual rainfall less than 1000 mm, yet European canker is a serious disease (refer to following sections).

Effect of rainfall on disease spread

Neonectria galligena sporulation, dispersal and infection are favoured by mild and wet conditions (Swinburne 1975). It is generally accepted that *N. galligena* spreads from tree to tree via airborne (ascospores) and splash-dispersed (conidia) spores. Rain is necessary for the dispersal of ascospores and conidia from wood cankers and provides the moisture needed for germination of spores on wood and fruit (Bondoux and Bulit 1959, McCartney 1967, Swinburne 1975, Taylor and Byrde 1954). Where rain occurs throughout the year, conidia are present at all times (Bullit 1957, Swinburne 1975). In drier climates such as California, they are produced during the wetter months of autumn and winter (Ogawa and English 1991). Ascospores are released from perithecia (fruiting structures) after rain. Once again, this varies according to the climate of a particular region and in England, France and Oregon, USA, ascospores are discharged all year round and are therefore an important source of inoculum, whereas in California they play a very minor role in disease spread (Ogawa and English 1991).

Infection occurs through an entry point such as a wound or natural opening. These can be bud-scale scars and other damage points during spring-summer and leaf scars in autumn (Swinburne 1975). In climatic conditions conducive to the formation and dissemination of conidia, inoculum from surrounding infected orchards is reported to be the primary source of *N. galligena* (Swinburne 1975, McCracken *et al.* 2003b). Aerial spread is therefore an essential element of the epidemiology of *N. galligena* (Swinburne 1975). Rainsplash and runoff spreads the pathogen within infected trees, and it is common to see secondary cankers developing in the crotch of branches below a primary canker higher in the tree. Primary cankers can also develop in young trees as a result of infections in the nursery (McCracken *et al.* 2003b).

Effect of rainfall on disease incidence worldwide

European canker is an important disease of apples in the UK and in particular in wetter regions of Northern Ireland. In these regions, spores and potential infection sites are available almost all seasons of the year. Incidence of fruit infection is high. Across the world, reports vary with regard to the season considered as the most important for wood infection. In areas with high rainfall such as in Northern Ireland, it was reported that about 75% of wood cankers resulted from infections in spring and summer and 25% at leaf fall from autumn infections (Swinburne *et al.* 1975). Cankers arising from spring and summer infections are apparent in autumn. The recommendation in the IRA (2005) for orchard inspection is mid-winter, after leaf fall. According to Alistair McCracken (pers. comm. 2006), the best time for detecting European canker is in the middle of the summer growing season, as shoot dieback due to small cankers is clearly visible. After leaf fall it is much more difficult to find cankers on twigs, small branches and in the higher reaches of the tree.

Disease incidence and severity are greater in high vigour trees growing in soils with high water-holding capacity (Swinburne 1975). In the UK during 1991-1993, Berrie (1997) demonstrated that 55.5% of the disease development in Cox's Orange Pippin (both fruit rot and wood canker) could be correlated with the amount of September rainfall. In the same study, during 1994, a year particularly conducive to fruit rots, up to 279 mm of rain and 31 infection periods were recorded between July and harvest (Berrie 1997). Therefore the amount of rainfall and, more importantly, the number of infection periods during fruit ripening are more important than annual rainfall in determining the potential risk of disease development.

The Millenium project (McCracken *et al.* 2003b) examined the relative importance of nursery infections and orchard inoculum in the development and spread of European canker in UK apple orchards. Three sites were used: Loughgall in Northern Ireland, East Malling in Kent and Rosemaund in West Midlands, England. The 30-year annual mean rainfall of these sites is 791, 653 and 702mm respectively. Three years after planting, almost all trees at Loughgall had developed cankers, 10% at East Malling and 6% at Rosemaund. The difference in canker development could be correlated with the source of the planting material, proximity of old orchards near the sites and local climatic conditions (McCracken *et al.* 2003b). The results also clearly demonstrate that annual rainfall is not a good indicator of disease.

In regions of California and the Pacific Northwest of the USA, fruit infection is rare (Dubin and English 1974, Nichols and Wilson 1956, McCartney 1967). California's rainfall quantity and frequency is more suitable for wood canker infections because rainfall is more frequent during the dormant season from leaf fall to bud break (Wilson 1966). Fruit infection is associated with delayed harvesting, when autumn rain induces infection (Ogawa and English 1991). For example, in 1965, heavy rain in late summer in the apple growing region of Sonora County, California, caused a severe outbreak of fruit infection visible two weeks after the rain (McCartney 1967). Losses were reported to range from 1 to 10% of the crop but were difficult to quantify because fruit fell from trees. Cultivars affected included Delicious, Golden Delicious, Jonathan, Rome Beauty, and Tompkins King (McCartney 1967). The initial rotting began only at the calyx end of the fruit but fruit infection has also been observed at the stem end in France (Bondoux and Bulit 1959, Swinburne 1964) and in scab infection sites (Butler and Jones 1949).

Rainfall conditions in Australia

In Australia, there are apple-growing regions with annual rainfall ranging from 900 to 1300 mm (http://www.bom.gov.au/silo/) such as Batlow (980 mm), Adelaide Hills (1188 mm), Bilpin (1300 mm) and Orange (941 mm). In regions such as the Goulburn Valley of Victoria, although annual rainfall is less than 900 mm, there are generally 5-12 wet periods that meet the criteria for apple scab infection periods during the pome fruit growing season (Villalta *et al.* 2001, Villalta *et al.* 2002). The requirements for apple scab infection periods are similar to those of European canker, and it is not uncommon for late infection periods to occur near harvest resulting in scab development months later in storage (Washington pers. comm. 2006). In wet years, a larger number (up to 20) of infection periods can occur which results in outbreaks of apple and pear scab and brown rot of stonefruit (Villalta *et al.* 2002, Holmes pers comm.). In these regions, rainfall can also occur during autumn and winter coinciding with leaf fall and pruning periods. *N. galligena* was able to establish in Spreyton Tasmania, which has an annual rainfall of 982 mm (Quoiba, near Spreyton) (Ransom 1997, http://www.bom.gov.au/silo/). Irrigation practices in some orchards and nurseries can create microclimatic conditions favourable for development of diseases such as apple and pear scab, and potentially European canker if *N. galligena* enters Australia.

From the above, it is clearly evident that disease prevalence, incidence and spread is not closely related to mean annual rainfall, but to the frequency and duration of infection periods at times when the host is susceptible to infection. As previously discussed when considering the full host range of the pathogen, *N. galligena* has been recorded from a wide range of climatic conditions ranging from sub-arctic to tropical.

STAGE 2 – Predictive modelling of the potential distribution of *Neonectria galligena* in Australia using the computer programs CLIMATE and CLIMEX

The fungal pathogen, *Neonectria galligena*, causes European canker, an economically important disease of apple and pear around the world. Symptoms include wood cankers that can girdle branches and trunks, and fruit rots (Swinburne 1975). The same pathogen also causes perennial wood cankers and economic loss on other woody hosts, such as forest trees for timber production.

In the event of an incursion, it is important to know which regions of Australia could provide a suitable environment for the establishment of *N. galligena* based on its growth requirements. For this reason, the primary objective of this study was to evaluate the suitability of Australian climates for the establishment of diseases caused by *N. galligena.* For this purpose, the climate matching model CLIMATE and the simulation model CLIMEX® (CSIRO, Victoria, Australia) were used to determine the suitability of regions of Australia for the establishment of *N. galligena*. The CLIMATE model uses climatic conditions or a 'climate profile' from regions where the species has been recorded to identify regions with similar climatic conditions (climate matching) where the species is absent. CLIMEX® can also be used for matching climates but has additional functions for simulation modelling of species distributions. The CLIMEX® model allows the prediction of the potential geographical distribution of a species using its observed geographical distribution and species growth requirements (Sutherst and Maywald 1985). It was developed for the specific purpose of determining where insect species exotic to Australia might be able to establish if they were introduced (Sutherst and Maywald 1985). In the past decade, plant pathologists have used CLIMEX to help determine where exotic pathogens might establish and cause disease. Such examples include *Puccinia psidii* / Eucalyptus rust (Booth *et al.* 2000a), *Cylindrocladium quinqueseptatum* / leaf blight of Eucalyptus (Booth *et al.* 2000b) *Magnaporthe grisea* / rice blast disease (Lanoiselet *et al.* 2002), hop powdery and downy mildews (Pethybridge *et al.* 2003) and *Guignardia citricarpa* / Citrus black spot (Paul *et al.* 2005).

The epidemiology of European canker of apples and the perennial canker diseases of forest trees has been discussed in the earlier section of this report. The fungus produces two spore types: asexual conidia and sexual ascospores. Spores are dispersed mainly by rain splash and wind (Swinburne 1975). In Chile and California, conidial infection is more important than infections by ascospores (Grove 1990, Lolas and Latorre 1996). In European countries ascosporic inoculum appears to be more important for infections (Lortie 1964, Swinburne 1975). Symptoms of new infections usually appear at bud break with the severity dependent on rainfall in spring and autumn (Grove 1990, Ogawa and English 1991). Fruit infections occur usually at the calyx end (Grove 1990, Latorre *et al.* 1999, Swinburne 1975). In USA, *N. galligena* causes bark (perennial canker) diseases on a range of hardwood trees in regions of Connecticut, Massachusetts and New Hampshire (Plante *et al.* 2002). It also causes losses to the timber industry in Canada.

Temperature and duration of wetness are important for germination of spores of *N. galligena* and disease development. *N. galligena* can grow in culture over a wide range of temperatures ranging from 2°C to 30°C (Munson 1939, Butler 1949). The optimum temperatures for disease development occur between 20°C to 25°C (Latorre *et al.* 2002, Grove 1990, Swinburne 1975). These conditions are common during spring and autumn in temperate and subtropical regions of the world. Controlled environment studies by Latorre *et al.* (2002) demonstrated that the optimum temperature for conidial germination was between 20°C and 25°C, with lower and upper limits of 6°C and 32°C, respectively. Ascospore germination was low at 5°C and rapidly increased as temperature increased from 5°C to 20°C. A minimum of 2

hours of wetness duration was required at the optimum temperature (20°C) for infection, with longer wetness duration required at lower temperatures. No infection was induced at 5°C regardless of the duration of wetness periods.

In the field, disease incidence varies significantly from season to season depending on climatic conditions. Latorre *et al.* (2002) reported canker incidences ranging from 0.01% to 48.3% on one year old twigs taken from the same orchard in dry and wet seasons respectively. High incidences of canker in apple orchards in Ireland and the UK have been correlated to cool and rainy weather conditions during leaf fall (McCracken *et al.* 2003). In California, Dubin and English (1974) reported that several days of free moisture were required to obtain high levels of infection. Recent outbreaks of disease in New Zealand were attributed to unusually wet springs and autumns (IRA report, 2004).

Prediction using the CLIMATE model
Method

CLIMATE is a software program designed to predict the distribution of an organism based on climate preferences. The program was developed from concepts contained in the BioClim Prediction system of J. R. Busby (Bureau of Flora and Fauna, Canberra) and CLIMEX (Maywald G. F. and Sutherst R. W., CSIRO). CLIMATE uses temperature and rainfall data from a set of geographical locations to construct a climate profile. This is used to indicate geographical regions that are contained within the boundaries of the profile. Typically the profile is based on the locations where an entity, such as a pathogen species, is known to occur naturally.

The world database used to produce climate profiles is a worldwide collection of locations, each with a complete set of meteorological data (monthly temperature and rainfall). CLIMATE uses meteorological data obtained from locations selected by the user to generate 16 derived climate parameters. The parameters are used to generate a climate profile suitable for a percentile, statistical or Euclidean distance analysis. The 16 parameters are identical to those defined in the BioClim system:

- Mean annual temperature
- Minimum temperature of coolest month
- Maximum temperature of warmest month
- Average temperature range
- Mean temperature of coolest quarter
- Mean temperature of warmest quarter
- Mean temperature of wettest quarter
- Mean temperature of driest quarter
- Average annual rainfall
- Rainfall of wettest month
- Rainfall of driest month
- CV monthly rainfall
- Rainfall of wettest quarter
- Rainfall of driest quarter

- Rainfall of coolest quarter
- Rainfall of warmest quarter

In this analysis, the world database was used to extract locations where *N. galligena* is prevalent, based on latitude and longitude of places where disease has occurred (Table 2). In addition to the specific locations mentioned in Table 2, Japan, all of the British Isles, North Island of NZ and north east USA were included.

For our purposes, a cumulative analysis was used in which every input location was matched individually using the Euclidean distance method. With this preference, the best match is used for output. For each of the 16 climate parameters, the deviation of the input value and the reference output point is calculated (no averaging is done). The Euclidean distance is a sum of the squares of these deviations. Matches are based on an arbitrary 0-100 scale (100 = poor match; 0 = excellent match).

Using the "calculate matches" option, a summary table of the climate profile was output from the cumulative analysis (Table 3). These parameters were then compared to meteorological data from Australia and an output file of matching locations was generated, specifying the degree of the match. This prediction data was then displayed on a map using the high-resolution preference.

Table 2. Locations used to generate the CLIMATE profile

SITE	LAT/LONG	REFERENCE
Australia – Tasmania, Spreyton	41.23S 146.35E	(Ransom 1997)
Austria – Niederdonau, Seehof Bei Lunz	48°24'N 15°23'E	The U.S. National Fungus Collections (BPI)
Austria – Steiermark, Schoeckl-Gruppe	47°1'N 15°26'E	The U.S. National Fungus Collections (BPI)
Canada – Nova Scotia. Amherst	45°50'N 62°12'W	The U.S. National Fungus Collections (BPI)
Canada – Ontario. Guelph	43°33'N 80°15'W	The U.S. National Fungus Collections (BPI)
Chile – Chillan	36°34'S 72° 7' 0"W	(Lattore *et al.* 2002)
Chile – Valdivia	39°48'S 73° 13' W	(Lattore *et al.* 2002)
East Germany – Brandenburg	52°25'N 12°32'E	The U.S. National Fungus Collections (BPI)
England - Kent, East Malling	0°25'E, 51°19'N	(Mc Cracken *et al.* 2003)
England – Hereford, Rosemaund	2°38'W, 52°7'N	(Mc Cracken *et al.* 2003)
Germany – Bayern, Regierungsbezirk Oberbayern, München city,	48°9'N 11°35'E	The U.S. National Fungus Collections (BPI)
Netherlands – Baarn	52°14'N 5°20'E	The U.S. National Fungus Collections (BPI)
Netherlands – Nr. Utrecht, Holland	52°7'N 5°9'E	The U.S. National Fungus Collections (BPI)
New Zealand – Auckland	36°51'S 174°46'E	http://nzfungi.landcareresearch.co.nz
New Zealand – Nelson	41°17'S 173°17'E	http://nzfungi.landcareresearch.co.nz
New Zealand – Taranaki	39°18'S 174°21'E	http://nzfungi.landcareresearch.co.nz
New Zealand –Bay of Plenty	38°11'S 176°46'E	http://nzfungi.landcareresearch.co.nz
New Zealand –Coromandel	36°46'S 175°30'E	http://nzfungi.landcareresearch.co.nz
New Zealand –Northland	35°30'S 173°59'E	http://nzfungi.landcareresearch.co.nz
New Zealand –Waikato	38°35'S 175°27'E	http://nzfungi.landcareresearch.co.nz
Northern Ireland – Armagh	54°34 N 6°63'W	BMS Fungal Records Database (FRDBI)

Northern Ireland – Hillsborough	54°46N 6°08'W	BMS Fungal Records Database (FRDBI)
Northern Ireland – Horticulture and Plant Breeding Station	54°19'N 6°35'W	(Mc Cracken *et al.* 2003)
Republic of Ireland – Kerry	52°03'N 9°62'W	BMS Fungal Records Database (FRDBI)
Romania – Transylvania, Distr. Alba	46°6'N 23°36'E	The U.S. National Fungus Collections (BPI)
Scotland – Benmore, near Dunoon, Argyllshire. Elevation 150 Feet		The U.S. National Fungus Collections (BPI)
Sweden – Gavle, Lovudden	60°42'N 17°12'E	The U.S. National Fungus Collections (BPI)
Sweden – Stockholm	59°20'N 18°5'E	The U.S. National Fungus Collections (BPI)
Switzerland – Locarno	46°12'N 8°49'E	The U.S. National Fungus Collections (BPI)
USA – Maine. Bar Harbor	46°50'N 67°55'W	The U.S. National Fungus Collections (BPI)
USA – California, Sonoma County, Sebastapol-Graton	38°42'N 122°85'W	(McCartney 1967)
USA – California. Crescent City, del Norte Co.	41°46'N 124°13'W	The U.S. National Fungus Collections (BPI)
USA – Connecticut. Cobalt	41°34'N 72°31'W	The U.S. National Fungus Collections (BPI)
USA – Connecticut. Watertown	41°37'N 73°9'W	The U.S. National Fungus Collections (BPI)
USA – Georgia. Blairsville	34°53'N 83°56'W	The U.S. National Fungus Collections (BPI)
USA – Hawaii. Poli, Oahu	21°27'N 157°59'W	The U.S. National Fungus Collections (BPI)
USA – Maryland. Oakland	38°52'N 76°55'W	The U.S. National Fungus Collections (BPI)
USA – Massachusetts. Hamilton	42°39'N 70°50'W	The U.S. National Fungus Collections (BPI)
USA – Michigan. Munising	46°24'N 86°39'W	The U.S. National Fungus Collections (BPI)
USA – New Hampshire. Black Brook Trail	43°51'N 71°42'W	The U.S. National Fungus Collections (BPI)
USA – New Jersey. Morristown	40°49'N 74°30'W	The U.S. National Fungus Collections (BPI)
USA – New York. Arnot Forest, near Ithaca, Tompkins County	42°27'N 76°30'W	The U.S. National Fungus Collections (BPI)
USA – New York. Newcomb	43°59'N 74°9'W	The U.S. National Fungus Collections (BPI)
USA – North Carolina. John Rock. Near Pisgah Forest	35°33'N 79°52'W	The U.S. National Fungus Collections (BPI)
USA – Oregon. Corvallis	44°35'N 123°16'W	The U.S. National Fungus Collections (BPI)
USA – Pennsylvania. Allegheny National Forest	40°27'N 80°1'W	The U.S. National Fungus Collections (BPI)
USA – Pennsylvania. Taylor	41°24'N 75°43'W	The U.S. National Fungus Collections (BPI)
USA – Vermont. Peru	43°13'N 72°53'W	The U.S. National Fungus Collections (BPI)
USA – Virginia. Vienna	38°54'N 77°15'W	The U.S. National Fungus

		Collections (BPI)
USA – West Virginia. Hillsboro	39°12'N 77°43'W	The U.S. National Fungus Collections (BPI)
USA – Wisconsin. Blue Mounds	43°1'N 89°50'W	The U.S. National Fungus Collections (BPI)

Results

The locations used to produce the climate profile for *N. galligena* are shown in Map 8. These are locations where *N. galligena* is known to consistently cause disease in pome fruit and timber. The summary table generated for the climate profile is shown in Table 3. The climate matching capability of CLIMATE generated a map of Australia showing regions that matched the profile and where *N. galligena* could potentially establish if a suitable host was available (Map 9).

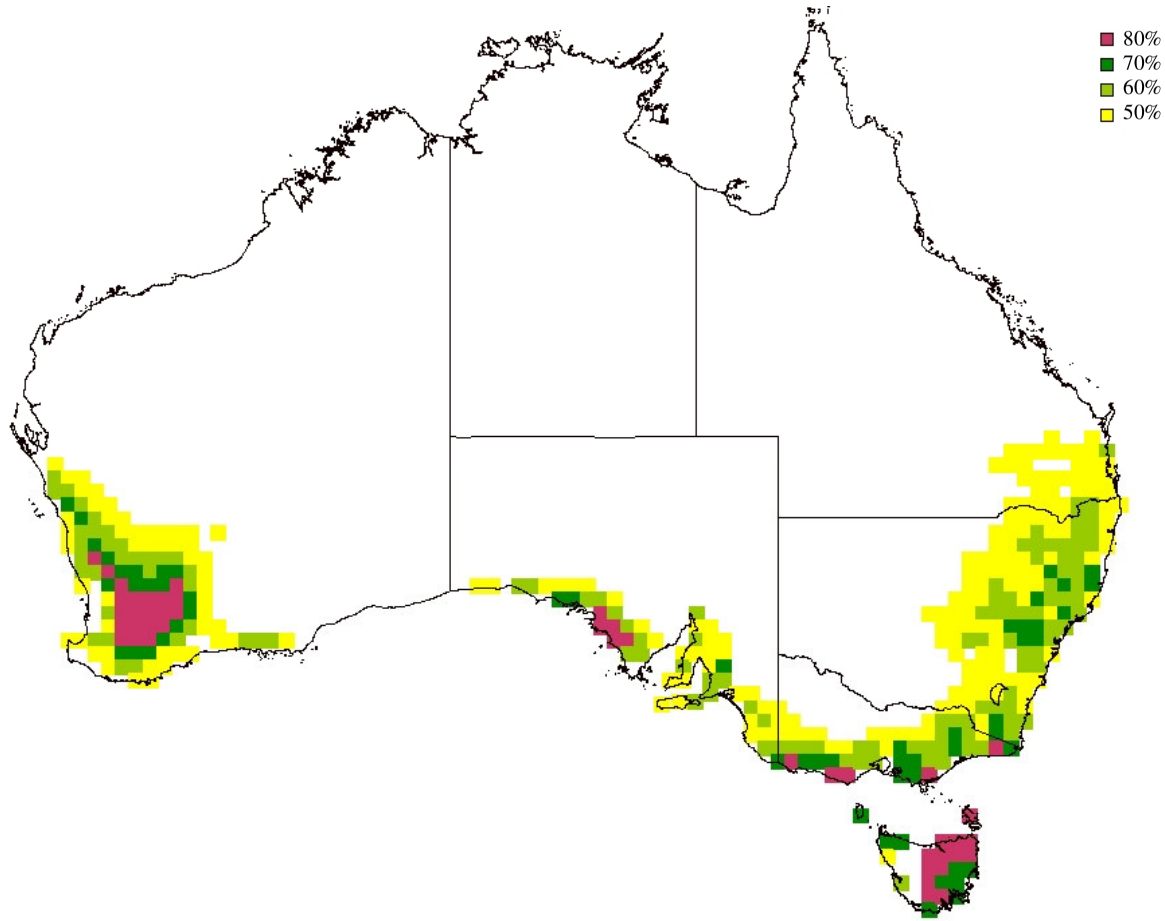
Table 3. Summary table of the CLIMATE profile for *N. galligena*

16 variables in analysis.
854 location records analysed.
Cumulative method used. Closest Euclidean match used.

	Statistics					
	Mean	St. dev.	Skew	Kurtosis	Min	Max
1 Mean annual temp	10.37	2.86	-0.44	8.19	-6.20	23.90
2 Min temp. cool month	-2.63	4.56	-0.64	3.61	-17.20	19.40
3 Max temp. warm month	24.98	4.66	-0.55	3.77	3.30	33.20
4 Average temp range	27.62	7.30	-0.14	1.91	8.90	42.80
5 Mean temp. cool quarter	1.72	3.84	-0.56	4.29	-12.60	22.20
6 Mean temp. warm quarter	19.05	3.93	-0.24	4.02	0.40	26.90
7 Mean temp. wet quarter	14.12	7.46	-0.45	2.33	-8.10	26.10
8 Mean temp. dry quarter	6.51	6.01	0.16	3.08	-11.90	24.60
9 Average annual rainfall	999.63	422.42	2.21	10.46	366.00	4157.00
10 Rainfall wet month	122.11	66.73	2.49	11.76	52.00	631.00
11 Rainfall dry month	52.94	21.09	1.49	8.17	0.00	185.00
12 CV monthly rainfall	25.10	12.56	1.37	5.52	4.50	101.00
13 Rainfall wet quarter	325.12	166.67	2.52	12.57	145.00	1672.00
14 Rainfall dry quarter	178.28	66.44	1.54	8.22	0.00	597.00
15 Rainfall cool quarter	219.93	110.18	3.70	22.62	51.00	1169.00
16 Rainfall warm quarter	289.25	147.78	2.40	13.90	0.00	1648.00

Australian prediction: 427 matching locations from 2798 comparisons
204 locations within 50 % of the mean.
123 locations within 40 % of the mean.
56 locations within 30 % of the mean.
44 locations within 20 % of the mean.

Map 8. Map output of locations used to produce the CLIMATE profile for *N. galligena.*



Map 9. The potential distribution of *N. galligena* in Australia based on the CLIMATE profile. 80% = locations within 20% of the mean climate profile, 70% = locations within 30% of the mean climate profile, etc.

(b) Prediction using the CLIMEX model

The CLIMEX model integrates the weekly response of a population to climate into a series of annual indices (Sutherst and Maywald 1985, 1999). Responses to temperature and moisture are combined into a weekly population growth index for the species. Weekly soil moisture is calculated using a hydrological model from rainfall and estimated evaporation. Annual temperature and moisture indices summarise the response of the species to temperature and moisture. Responses to detrimental conditions are calculated by a series of stress indices that estimate the effect of extremes of climatic conditions (hot, cold, dry and wet). The growth and stress indices are combined into an ecoclimatic index (EI). This index is scaled from 0 to 100 and indicates the suitability of a given location to support a permanent population of the target species. An EI value close to 0 indicates that a location is not favourable for the long-term survival of the species and a value of 100 is only possible under constant and ideal conditions such as provided in an incubator. EI values greater than 30 are considered very favourable for population growth and persistence (Sutherst *et al.* 2005). This study used the 'Compare Locations' function of CLIMEX, which compares the relative potential for growth of a species in different localities and allows prediction of the potential geographic distribution of that species based on its climatic requirements (Sutherst and Maywald 1985, 1999).

Information on the distribution of *N. galligena* worldwide was obtained from literature, fungal databases (eg CAB International) and web sites. The meteorological database in CLIMEX® has values for monthly long-term (30 year) average maximum and minimum temperatures, rainfall and relative humidity for 3092 locations worldwide.

Method

The values of the parameters for use in the simulation model were based on specific epidemiological information available for *N. galligena* and modified to fit the current known distribution of *N. galligena* worldwide. The parameters therefore reflect the climatic requirements for growth of *N. galligena* and establishment and persistence of diseases caused by this pathogen. The values for temperature, moisture (rainfall and RH) and stress indices (cold, warm, dry and wet) describe the response of the species to climate. The parameters were adjusted until a close visual match occurred between the simulated and the known geographical distribution of *N. galligena* (Sutherst and Maywald, 1985). When we were satisfied that the match was as accurate as possible, the parameters (Table 4) were used to predict the potential geographical distribution of *N. galligena* in Australia. A second simulation was run using the irrigation option of CLIMEX, with top-up irrigation was set at 4 mm/day during September, October, November and December, based on the water requirements of apple crops in the Goulburn Valley, Victoria (O'Connell and Goodwin 2003).

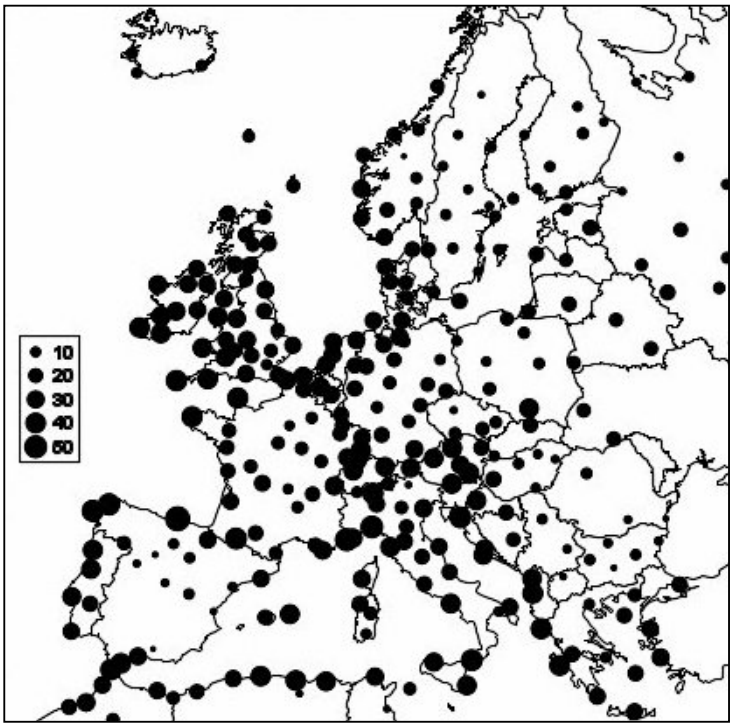
Results

The CLIMEX template for temperate species was used as a basis for the *N. galligena* species profile for the simulation model. Temperature, moisture and stress indices were modified according to information published on the growth requirements of the pathogen. These values were then fine-tuned by visually matching the simulated distribution of the pathogen to the known distribution in various countries around the world (eg Maps 10 - 13), based on records published in the CAB International on-line database (refer to Table 1 in earlier section). The simulation model was run to identify regions in Australia where climatic conditions are favourable for *N. galligena* growth. Many locations around the southern and eastern coastline of Australia are suitable, particularly if irrigation is also considered (Map 14).

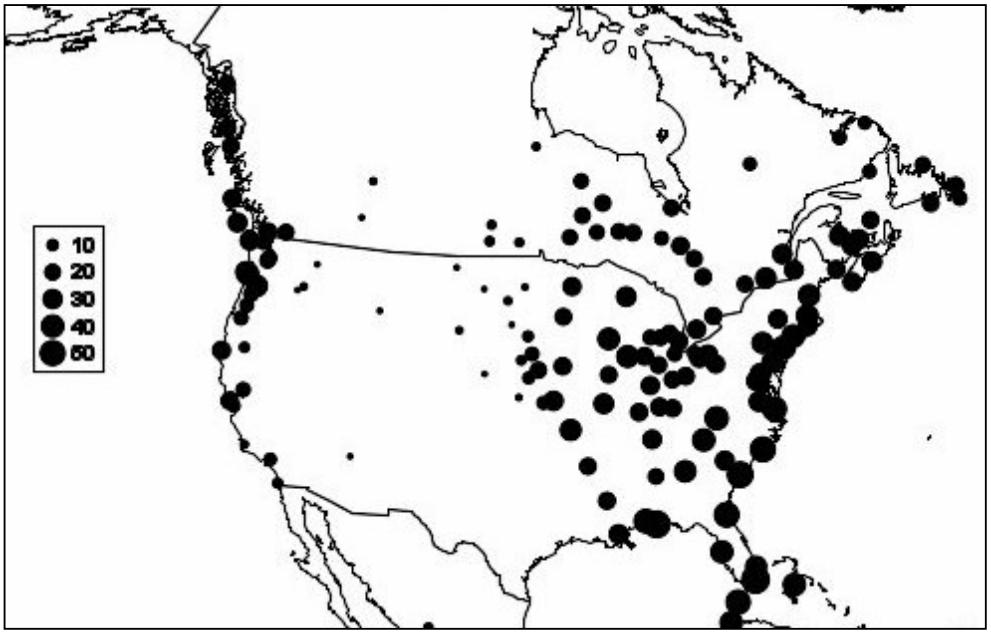
Table 4. CLIMEX parameter values used to best fit the known worldwide distribution of *Neonectria galligena*.

Parameter description	Value
Soil Moisture	
Soil moisture capacity	100
Evapotranspiration coefficient	0.8
Temperature Index	
Lower threshold of temperature for population growth (DV0)	5
Lower optimal temperature for population growth (DV1)	20
Upper optimal temperature for population growth (DV2)	25
Upper threshold of temperature for population growth (DV3)	32
Moisture Index	
Lower threshold of soil moisture (SM0)	0.3
Lower limit of optimal range of soil temperature (SM1)	0.7
Upper limit of optimal range of soil temperature (SM2)	2.0
Upper threshold of soil moisture (SM3)	3.52
Light index – not used	
Cold stress – not used	
Dry stress – not used	
Wet stress – not used	
Heat stress	
Heat stress temperature threshold C (TTHS) (above which heat stress accumulates)	32
Heat stress temperature rate (THHS)	0.1
Heat stress degree-day threshold C (DTHS)	0
Heat stress degree-day rate (DHHS)	0
Hot-wet stress interaction	
Hot-wet temperature threshold	32
Hot-wet moisture threshold	0.8
Hot-wet stress rate	0.05

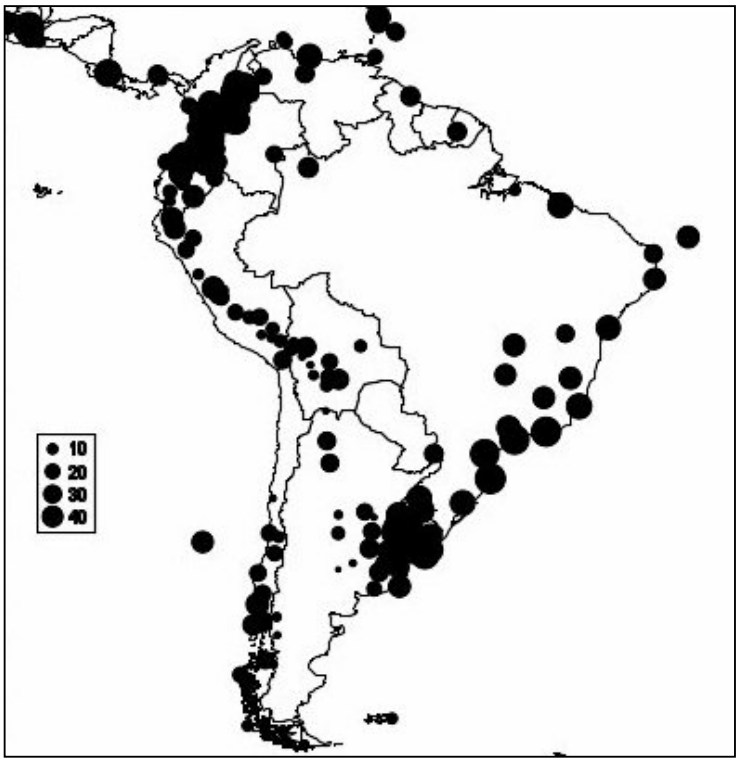
Map 10. Locations in Europe where *Neonectria galligena* is established or has the potential to establish. The dots represent ecoclimatic indices as determined by CLIMEX.



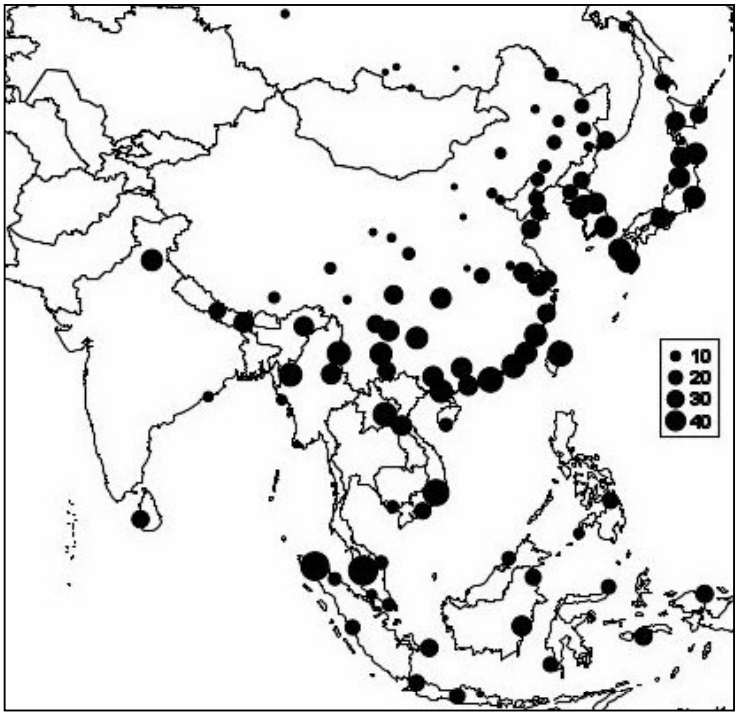
Map 11. Locations in North America where *Neonectria galligena* is established or has the potential to establish. The dots represent ecoclimatic indices as determined by CLIMEX.



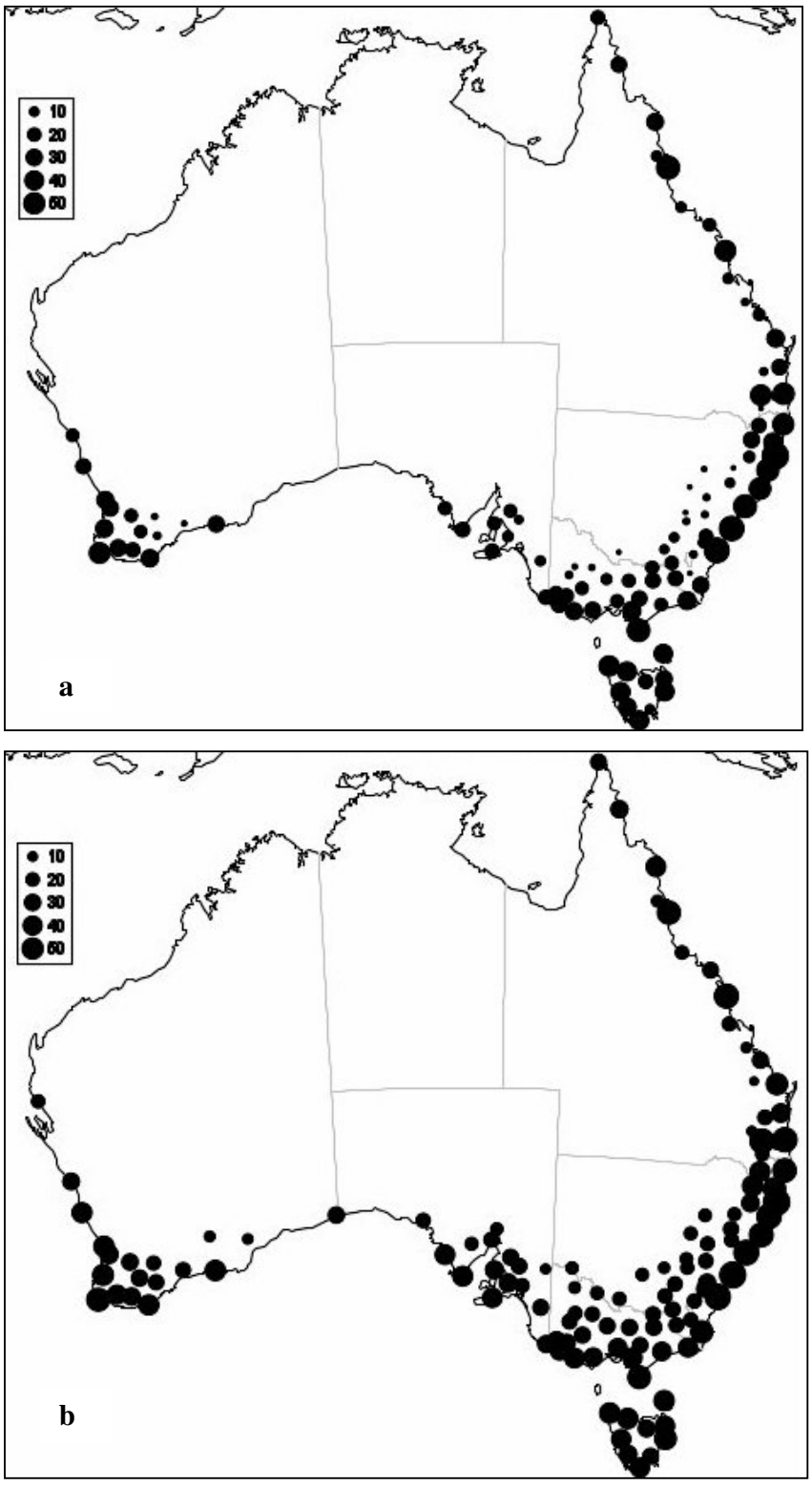
Map 12. Locations in South America where *Neonectria galligena* is established or has the potential to establish. The dots represent ecoclimatic indices as determined by CLIMEX.



Map 13. Locations in Asia where *Neonectria galligena* is established or has the potential to establish. The dots represent ecoclimatic indices as determined by CLIMEX.



Map 14. Locations in Australia where *Neonectria galligena* has the potential to establish (a) without irrigation, (b) with irrigation. The dots represent ecoclimatic indices as determined by CLIMEX.



As explained above, the various environmental indices (temperature, moisture, etc) are combined to give an ecoclimatic index (EI) value, which rates the suitability of a locality for establishment and persistence of the species. The EI values for the occurrence of *N. galligena* could be broadly categorised as follows: EI≤10, climate unfavourable for the persistence of the species; 11≤EI≤20, marginally suitable for disease development; EI≥21, favourable for disease development, with EI≥25 as highly favourable for disease development (based on Paul *et al.* 2005). Table 5 shows the locations within Australia that have an EI value above zero. Out of 228 locations in Australia, 53 had EI values ≥21, and of these 43 were ≥25, highly favourable for disease development.

Table 5. Locations in Australia, with eco-climatic indices (EI) greater than zero, as determined by the simulation model, CLIMEX, using the species profile defined in Table 4.

Location	EI	Location	EI	Location	EI
New South Wales					
Albury	19	Dubbo	2	Sydney	68
Armidale	14	Forbes	2	Tamworth	2
Bathurst	5	Glen Innes	29	Taralga	21
Bega	30	Goulburn	12	Taree	52
Canberra (ACT)	6	Grafton	40	Tenterfield	24
Coff's Harbour	78	Kempsey	55	Thredbo	25
Cooma	2	Lismore	50	Tumbarumba	20
Coonabarabran	3	Mudgee	5	Wagga	9
Cootamundra	10	Murrurundi	10	Williamtown	59
Cowra	5	Muswellbrook	1	Wyalong	1
Deniliquin	2	Nowra	70		
Queensland					
Amberley	5	Innisfail	60	St Lawrence	12
Bowen	18	Kingaroy	7	Thursday Island	21
Brisbane	51	Lockhart River	27	Toowoomba	47
Bundaberg	35	Mackay	48	Townsville	12
Cooktown	32	Mareeba	12	Warwick	2
Gladstone	16	Monto	1		
Gympie	27	Rockhampton	6		
South Australia					
Adelaide	13	Keith	11	Mount Gambier	23
Clare	18	Kimba	1	Port Lincoln	24
Elliston	19	Kingscote	24		
Eudunda	8	Maitland	19		
Tasmania					
Bicheno	40	Hobart	9	St Helens	30
Burnie	40	Launceston	23	Strathgordon	35
Flinders Island	38	Queenstown	41		
Hastings	41	Redpa	46		
Victoria					
Ararat	18	Charlton	4	Melbourne	19
Bairnsdale	18	Colac	29	Warracknabeal	3
Bendigo	13	Euroa	19	Warragul	38
Bright	25	Hamilton	23	Warrnambool	33

Cann River	36	Heywood	31	Wilsons Promontory	58
Casterton	22	Horsham	6	Woods Point	26
Western Australia					
Albany	34	Geraldton	18	Narrogin	18
Augusta	48	Jurien	26	Ongerup	6
Bunbury	35	Katanning	17	Perth	33
Esperance	29	Lake Grace	4	Ravensthorpe	3
Frankland	23	Manjimup	31	Serpentine	31
Rest of World					
Auckland, NZ	66	Aldergrove, N.Ireland	29	Southampton, UK	29
Portland, Maine, USA	36	San Franscisco, USA	24	Amsterdam, The Netherlands	31

Discussion

The two models, CLIMATE and CLIMEX, produced slightly different predictions for the potential distribution of *N. galligena* in Australia. The CLIMATE model was asked to find localities in Australia with climatic conditions that matched those of locations in the world where disease caused by this pathogen is frequent. By comparison, the CLIMEX model used information about the environmental requirements for survival and growth of *N. galligena*, based on records of all the locations where it is known to occur combined with data from laboratory studies. Taking all records into consideration, *N. galligena* appears to be a very adaptable organism. It has been found in cold, sub-arctic regions such as Iceland, Sweden and Nova Scotia; in tropical regions such as Java, Florida, New Orleans, and also in arid regions such as Saudia Arabia, Syria and Afghanistan. Therefore the potential distribution predicted by CLIMEX was more extensive than that of CLIMATE and included some coastal regions of sub-tropical Australia that were omitted from the latter model.

Neither model takes into consideration whether a suitable host is present or not. The predictions of both models include regions where important horticultural crops such as apple, pear and walnut are grown (see Maps 3, 4 and 5) and also many susceptible tree species (Maps 6 and 7). Apple growing regions (Map 3) near locations with an EI > 21 (ie favourable for disease development, Table 5) are Perth Hills (Perth EI = 33), Manjimup (EI = 31), Bilpin (Sydney EI = 68), Gippsland (Warragul = 38), Tamar Valley (Launceston EI = 23) and Huon Valley (Hastings EI = 41). Pear-growing regions (Map 4) favourable for disease development are Perth Hills (Perth EI = 33), Donnybrook (Manjimup EI = 31) and Huon Valley (Hastings EI = 41). Walnut-growing regions (Map 5) favourable for disease development are south of Perth (Perth EI = 33), Blue Mountains and Bowral (between Sydney EI = 68 and Williamtown EI = 59), Gippsland (Warragul = 38), Bright (EI = 25), Devonport (Burnie EI = 40) and East Coast of Tasmania (Bicheno EI = 40). In addition, *N. galligena* has a known host range of over 60 species, many of which are common throughout the regions indicated to be favourable by both models (Maps 6, 7 and 14), and has demonstrated the ability to 'jump' onto New Zealand native flora. Several Australian genera are susceptible to endemic species of *Nectria,* so it is quite conceivable that native flora will also be able to host this pathogen.

If *N. galligena* enters and establishes in Australia, the cost of pome fruit production would increase significantly. The only effective control for European canker is pruning and removal of affected limbs, which is expensive, time-consuming and disruptive of canopy architecture systems. The emerging walnut industry in Australia would also be at risk. Walnut production is being established in the same regions as pome fruit and two-thirds of the trees are still to come into full production (http://www.walnut.net.au/public/walnut_production_Adem). The main commercially grown species, *Juglans regia*, and the most popular rootstock, *J. nigra*,

are both susceptible hosts (CABI). Nurseries supplying the pome and nut industries would be particularly at risk because infection can be spread in symptomless planting material. A comparable situation has occurred in nurseries supplying the viticulture industry in the case of Petri disease, caused by the fungus *Phaeomoniella chlamydospora*. The disease is spread in symptomless young vines, and several nurseries in the USA and Australia were involved in litigation for unknowingly supplying infected planting material (Morton 1995, Gubler, pers. comm, Waite, pers comm). The impact on the ornamentals and allied industry is difficult to estimate, but many popular garden trees such as *Acer, Betula* and *Quercus* species are susceptible.

STAGE 3 – Simulation experiment to demonstrate whether symptomless infection of fruit has the potential to be a pathway for *N. galligena* into Australia

This stage is still under negotiation. During Stage 1, discussions with colleagues plus information from the literature review indicated that Dr. Alistair McCracken, Applied Plant Science Division, Department of Agriculture and Rural Development, Belfast, Northern Ireland, was best placed to conduct a simulated storage trial. Email contact was established, and in September 2006, Dr Edwards travelled to Belfast to meet with him. She visited the research station near Armagh, was shown around the facilities and taken to nearby trial sites and commercial orchards to see European canker first-hand. Discussions were held with Drs Alistair McCracken, Louise Cooke and Averill Brown. Drs McCracken and Cooke are actively involved in research into European canker, and have a current trial investigating fungicide control of the disease. Dr Brown is now retired, but was actively involved in the research conducted by Swinburne during the 1970s. Her PhD research was responsible for identifying that benzoic acid played a key role in latency in fruit. Dr Edwards also visited the apple-growing region of Wisbech, Norfolk, and was able to talk with apple grower Roger Manning of Barton Farms, and observe European canker on the cultivar Braeburn in the field.

Drs McCracken and Louise Cooke are willing to collaborate. One of the major gaps in knowledge identified in Stage 1 is the lack of information regarding latency periods in fruit of cultivars grown in New Zealand. From the discussions with Dr Brown , it was clear that naturally-infected fruit must be used. If fruit is artificially inoculated, symptoms usually develop straight away bypassing any latent stage. In Northern Ireland, a single variety, Bramley Seedling, is grown, although the pollinators can be many varieties. It was decided that to ensure that the experiment takes place this season, the best approach is to source fruit from their current trial, which is comparing the response of tree infection in Royal Gala and Bramley Seedling to various treatments. In this way, the incidence of latent infection in fruit of a common New Zealand variety (Royal Gala) can be compared with that of Bramley Seedling (high benzoic acid content). Since returning to Australia, Dr Edwards is continuing negotiations with Dr McCracken by email, and is waiting for Dr McCracken to finalise experimental design and estimate the cost. As soon as she has this information, she will discuss the proposal with APAL and HAL.

Information gained from the visit include:

- *N. galligena* commonly causes core rot in fruit, particularly in open sinus varieties (see Photo 38). During the visit, core rot was observed in Braeburn at Wisbech, and Bramley Seedling and Golden Delicious in Northern Ireland. This type of infection is not usually visible, but is detected when the fruit is cut open (see Photo 39). Several pathogens can cause the same symptom, so identification relies on traditional methods of isolation and examination by microscopy.
- The pollinators used in the UK are common dessert varieties such as Royal Gala, Jonathan, Golden Delicious, Red Delicious, all of which are susceptible to *N. galligena*. As a consequence, European canker symptoms are first observed in the pollinators and they become the foci of disease in the orchard.
- There were cankers on many of the hedgerow plants around the orchards in Armagh.
- Dr McCracken voiced the opinion that a single inspection after leaf fall was not sufficient to detect all infections. Larger cankers would be visible, but twig cankers would be missed. He demonstrated how shoot dieback symptoms can be used to find twig cankers and small cankers on branches (see Photo 40) when the tree is in full leaf.

Symptoms of European canker in Northern Ireland, September 2006.

Photo 33. Severe symptoms of dieback including leaf reddening, poor growth and early senescence caused by *N. galligena* on Royal Gala. Photo: J Edwards

Photo 34. Canker on the trunk of Royal Gala caused by *N. galligena* Photo: J Edwards

Photo 35. Canker on the trunk of Royal Gala caused by *N. galligena.* Photo: J Edwards

Photo 36. Canker on the trunk and branch girdling caused by *N. galligena* of Royal Gala. Photo: J. Edwards

Photo 37. Numerous cankers on a branch of Royal Gala caused by *N. galligena* Photo: J. Edwards

Photo 38. The open calyx of some apple varieties make them more susceptible to latent infection by *N. galligena*. Photo: J. Edwards

Photo 39: Core rot of Golden Delicious possibly caused by *N. galligena*. Photos: J. Edwards

Photo 40: Twig dieback caused by European canker demonstrating that infections can be found when the tree is in full leaf. Photo: J. Edwards

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Technology Transfer

Due to the sensitive nature of this subject, technology transfer was not considered appropriate. The predictive model study is currently being prepared into a manuscript for submission to a suitable scientific journal.

Recommendations

This study has identified that:

- Data is lacking regarding latent periods in fruit of cultivars grown in New Zealand for export. Latent infection in apple fruit that developed into rot during or post-storage has been reported for several dessert varieties such as Jonathan (Puia et al. 2003), Reinettes du mans and Reinettes blanches du Canada (Bondoux and Bulit 1959), Cox's Orange Pippin (Berrie 1997).
- Data is lacking to confirm or reject the hypothesis that disease can be spread in infected fruit. Mummified fruit is known to be a source of inoculum. Insects have been associated with *N. galligena* (**http://nzfungi.lancareresearch.co.nz** specimen PDD 31890). It is unknown whether weed apple trees grown from discarded cores of infected fruit are infected or not.
- A single annual inspection during the dormant period after leaf fall is unlikely to detect all infections (McCracken, pers. comm. 2006).
- Leaf and petiole infections of pome fruit have been reported (Wormald 1955, Ogawa and English 1997), but there is no information as to whether they can provide a source of inoculum.
- Incidence and severity of fruit infection is not correlated with annual rainfall.
- *Neonectria galligena* is highly adaptable. The pathogen has been recorded from over 60 host species from 20 genera, and from climatic regions ranging from sub-arctic (Iceland, Sweden, Nova Scotia), arid (Syria, Saudia Arabia, Afghanistan) to tropical (Java, Florida, New Orleans).
- Predictive modelling demonstrated that many regions of Australia would provide environmental conditions favourable for disease establishment.
- Industries that would be adversely affected by an incursion of *N. galligena* are apple, pear, walnut, loquat and ornamental. Nurseries supplying these industries would also be affected.
- There is no information regarding the susceptibility of Australian native flora. Three species of New Zealand native flora are recorded as hosts. It is not known how they became infected as the records are from the Coromandel Peninsula, not from apple production regions.

It is also recommended that Stage 3 be allowed to progress through to completion to provide data on latent infection of fruit in a variety commonly grown in Australia and New Zealand.