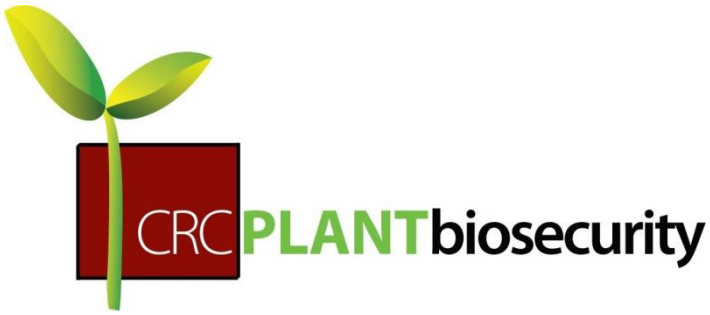

Cooperative Research Centre
for National Plant Biosecurity

Final Report

CRC10162

Communicating Uncertainty in Biosecurity Adaption

Authors

David Cook, Shuang Liu, Jean-Philippe Aurambout, Oscar Villalta, Darren Kriticos, Jacky Edwards, Art Diggle, Abu-Baker Siddique, Michael Hurley and Kim Lowell

30 May 2012

Project Leader contact details:

Name: Dr. David Cook
Address: Department of Agriculture and Food Western Australia, PO Box 1231, Bunbury WA 6231
Phone: +61 8 9780 6179
Fax: +61 8 9780 6136
Email: david.cook@agric.wa.gov.au

CRCNPB contact details:

Cooperative Research Centre for National Plant Biosecurity
LPO Box 5012
Bruce ACT 5012

Phone: +61 (0)2 6201 2882
Fax: +61 (0)2 6201 5067
Email: info@crcplantbiosecurity.com.au
Web: www.crcplantbiosecurity.com.au

Table of contents

Tables

Figures

1. Executive Summary

The Communicating Uncertainties in Biosecurity Adaption (CRC10162) project, dubbed CUBA, was intended to improve the uptake of uncertain information about Emergency Plant Pest (EPP) incursions using a mixture of maps-based (or spatial) incursion simulation models and non-spatial statistical models.

The project had two key stakeholders, Apple and Pear Australia Ltd. (APAL) and the Australian Banana Grower's Council (ABGC). Each stakeholder had slightly different needs in terms of modelling uncertain EPP impacts on their industries, so the project tailored outputs according to their requests. In short, the ABGC did not require a group-based spatial incursion management tool, but did require a detailed economic impact assessment model for several pathogens of concern to the industry. APAL required a spatial incursion management device with a group interface to help manage future incursions, but did not require a non-spatial statistical model. The CUBA researchers therefore prepared both spatial and non-spatial incursion simulation models to cater for these needs. The underlying structure of each of the models is generic, enabling them to be easily adapted to simulate a wide range of EPP incursions on single or multiple host industries.

The spatial models were designed to place decision-making groups psychologically 'near' to an incursion event. They were designed to be used in interactive, multi-disciplinary "war game" workshops in which they can be integrated into a structured decision-making process to refine incursion response protocols. Decision-making groups can use the models to generate virtual realities in which EPP incursions take place and have to be managed over multiple time periods. Strategies adopted by the group can take the form of eradication, slow-the-spread (or containment), live-with-it (i.e. minimal response), or combinations of each.

When decision-making groups come to decision points where they must evaluate the information about an outbreak and establish what their goals and priorities for the next phase of the response are, they can use the structured decision-making (SDM) process we have developed to complement the spatial bioeconomic model. In the SDM process, group members can express their opinions about the key priorities of incursion management (be they economic, social or environmental in nature) using keypad devices which are fed back to a computer and aggregated. The group can then either follow a most preferred management strategy, or they can disaggregate the results to determine any need for additional information if they are collectively unhappy with the automated response recommendation. Spatial models and SDM were successfully trialled in an interactive gaming experiment in April 2012 to simulate an incursion of fire blight in the Goulburn Valley, Victoria.

The non-spatial models developed for the ABGC were used to generate detailed impact assessments for five key plant pathogens. These included:

Banana bunchy top virus (*Babuvirus*, Nanoviridae) - Banana bunchy top virus (BBTV) is established in Australia, but is targeted for eradication from banana growing regions of Queensland and northern New South Wales. We developed a partial budgeting approach using a stratified diffusion spread model to simulate the likely benefits of exclusion of this virus from commercial banana plantations over time relative to a nil management scenario in which no surveillance or containment activities take places. We predict the exclusion benefits of the disease will avoid $15.9-27.0 million in annual losses for the banana industry.

Black Sigatoka (*Mycosphaerella fijiensis* (Morelet)) - *M. fijiensis* has been eradicated from Australia relatively recently and strict quarantine measures are still in place to protect against its reintroduction. But, the damage that could be caused to the Australian banana industry is potentially huge. We provide quantitative estimates of these potential damages and discuss the implications for Australia's acceptable level of protection. We find that if there were no quarantine restrictions, expected producer losses to the disease exceed $200 million. With quarantine measures in place annual expected damages over a 20 year period are still substantial at just under $100 million.

Moko disease (*Ralstonia solanacearum* – race 2) - This disease is found throughout many parts of the world where bananas are cultivated and has proven a serious biosecurity threat as there are no treatments known to be effective against it other than destroying infected plants. We find that if there were no phytosanitary measures in place against imported bananas, expected producer losses to *R. solanacearum* could amount to approximately $100 million per year after 20 years. However, there is a lot of uncertainty in our predictions as there is a relatively high likelihood of successful eradication upon detection provided this takes place very early in the invasion process.

Panama disease (*Fusarium oxysporum* f.sp. *cubense*) tropical race 4 (*Foc* TR4) - *Foc* TR4 is a serious soil-borne disease considered to be one of the most severe threats facing the banana industry worldwide. This race was discovered in the Northern Territory in the late 1990s and has remained under strict quarantine management. All control techniques for this strain have proved unsuccessful, meaning that once a plantation becomes infected with this disease, further spread can only be achieved by the destruction of infected plants. In 20 years' time, we estimate the impact of the disease could exceed $45 million per year.

Yellow Sigatoka (*M. musicola*) - The Queensland State government imposes standards for de-leafing to minimise the risk of *M. musicola* spread and impact within six banana pest quarantine. Of these, the Northern Banana Pest Quarantine Area (NBPQA) is the most significant, encompassing over 80 per cent of the State's banana production. Previous regulations have imposed an obligation on owners of banana plants within the NBPQA to remove leaves from plants with visible *M. musicola* symptoms on more than 15 per cent of any leaf during the wet season. Recently, this leaf disease threshold has been lowered to five per cent. We estimate that over a 30-year period, the average net benefit this reduced threshold will generate for the banana industry in the NBPQA will only be of the order of $1.4 million per year.

By the end of this diverse and complex project, we successfully demonstrated the potential for maps-based incursion models to be used to communicate complex suites of information to industry and government stakeholders. We showed how these models can be used in conjunction with a structured decision making (SDM) approach to refine invasion response plans. We also demonstrated the explanatory power of more traditional, statistics-based economic impact assessments in communicating the potential significance of EPPs over long periods of time (e.g. 20-30 years). These assessments can be of great strategic significance in setting broad research agendas and funding priorities when site-specific details of possible future incursions are not relevant.

Recommendations:

1. Structured decision making encompassing deliberative multi-criteria evaluation should be considered a relevant framework for making incursion response decisions.

2. Maps-based incursion simulation models should be further developed and employed in the refinement of incursion response plans.

3. Research should be conducted to investigate the feasibility of integrating maps-based bio-economic incursion management models with surveillance and field diagnostic technologies to form an incursion response platform.

4. Traditional economic analyses intended for circulation and future use by diverse groups of decision-makers should be designed to be as functional and flexible as possible to cater for this diversity.

2. Aims and objectives

The three objectives of the research project were as follows:

1. Synthesise research outcomes from the entire suite of Program 1 projects to form a flexible and user-friendly decision support tool for industry and regulators that is the culmination of risk related research supported by the CRCNPB.
2. Collaboration between CRC participants and non-participants to develop a framework for biosecurity planning that includes expert consultation, visual technologies, bioeconomic modelling and decision-facilitation techniques.
3. Interactive software tool to facilitate risk mitigation decisions according to climate suitability, the economic significance of hosts and non-market impacts (such as environmental or social effects of invasion).

3. Key findings

3.1. Methodological Review

Recent research and development of decision-support tools to help biosecurity decision makers to make complex investment decisions has relied on the use of Monte Carlo simulation models together with group-based multi-criteria decision analysis (MCDA) (Cook *et al.* 2010b; Cook *et al.* 2009a; Cook *et al.* 2009b; Cook and Proctor 2007; Hurley *et al.* 2010). One of the strengths of the MCDA approach is the transparent communication of uncertainty to decision-making juries through figures and statistics[1]. This is important when, as is often the case in invasive species response policy, decisions are characterised by profound scientific uncertainty and even ignorance about the behaviour of invasive species in environments where they have not been previously observed. In addition to scientific uncertainties, there can also be a great deal of strategic and political uncertainty surrounding risk management decisions.

The MDCA approach put forward in Cook *et al.* (2009a) and Cook *et al.* (2010b) has sufficient flexibility to deal with the changing context of decisions, allowing scientific, economic and social analysts to tailor information to the circumstances of a risk management decision. It can be used to prioritise species by industry or region; to prioritise risk mitigating investments (such as species specific R&D projects or integrated pest management activities); or to determine appropriate management strategies post-invasion (i.e. benefit cost analysis or cost effectiveness analyses).

As effective as MCDA approaches are as transfer vehicles for complex invasive species information, to date they have been limited to non-spatial decision contexts due to a lack of specificity in Monte Carlo simulation models. Where host environments are largely homogenous this does not pose a problem, but in agriculturally and environmentally diverse regions the spatial characteristics of invasive species impacts can be highly varied, and important in a risk management context.

This is particularly true when considering the intertemporal effects of invasive species across different landscapes. Improving the spatio-temporal element of MCDA techniques used in biosecurity can be achieved through the use of maps to communicate information, in addition to traditional statistical indicators. These may be used as both output devices to express predictions and uncertainties, as well as input devices to capture scientific expert judgement in cases of high uncertainty about a species and its relationship with a host.

When an event occurs in the 'here and now' decision-makers tend to have a lot of information about it, and therefore think of it in concrete, low-level (i.e. intricate detail) terms. But, when an event like a pest incursion is further removed from direct experience (i.e. is more distant into the future), decision makers have less available and reliable information about it, leading to the formation of a more abstract and schematic representation of the event (Trope *et al.* 2007). Moreover, words and statistics carry the essence of the referent event, whereas pictures are concrete representations that carry the properties of an invasion event in full detail (Liberman *et al.* 2002; Liberman and Trope 1998). Therefore, when a decision-making group is psychologically 'near' to an event, pictorial representations of it are more effective decision aids than words and statistics (Förster *et al.* 2004).

[1] For a thorough review of MCDA methods see Cook *et al.* (2010b).

In this section we briefly review approaches from the modelling and ecology literature and described methods and techniques that may be useful in developing visual information tools for use in group-based MCDA for invasive species risk management decisions. Projecting this population and impact information on to maps familiar to the group will enhance the uptake of this information by placing decision-makers psychologically closer to incursion events, but the process of doing so is complex. In this section, we discuss some basic steps that should be followed in order to place MCDA participants psychologically close to incursion events. These include including choosing model scale, clarifying the area of interest for the MCDA and choosing the form of population model to use to project population distribution and abundance on to maps. We discuss a range of group decision-making applications of visual, maps-based approaches and identified some of the tools used.

3.1.1. Introduction

3.1.1.1. Definition of invasive species

Numerous terms have been used around biological invasions, including 'non-indigenous', 'non-native', 'alien', 'exotic', 'invasive', 'noxious', 'nuisance', and 'weed'. This proliferation of terms has caused considerable confusion and misuse of existing terminology. The term 'invasive' in particular has been problematic as ecologists typically use it in reference to species which spread quickly and/or widely beyond the location of initial establishment, whereas in policy and legal documents it tends to imply negative effects caused to human beings even though invasiveness of a species does not necessarily predict its impact (Ricciardi and Cohen 2007).

For the purpose of this review invasive species was defined as a species that does not naturally occur in a specific area and whose introduction does or is likely to cause economic or environmental harm or harm to human health. Throughout the review we use the words 'impacts' or 'effects' without necessarily suggesting a negative connotation. We note in passing that most existing economic analyses focus on negative impacts of invasive species.

3.1.1.2. Scope of the literature review

In this review we report on the use of visual devices in group-based, interactive decision making settings and suggest tools and methods that can be used to maximise the effectiveness of visual inputs in to MCDA.

3.1.1.3. Approach used

We look at different applications from the literature, concentrating on examples involving invasive species research. We draw out features and techniques that can improve visual information quality received by decision-making groups with the aim of improving invasive species risk management decisions. In structuring the review, we cite several of the recommendations put forward in Hirzel and Le Lay (2008) for the production of meaningful species habitat suitability maps, and expand them to provide insights into the effective use of maps in group-based MCDA offering support and examples from the literature along the way.

3.1.2. Predictive model scale selection

3.1.2.1. Population models

In the context of invasive species, projecting the potential spread and impact of newly-introduced species requires the use of a population spread model. Since the seminal work of Fisher (1937) and Skellam (1951) ongoing attention has been devoted to the development of species spread models in ecology as a means of either understanding how organisms spread, developing new modelling techniques or predicting their spread rates (reviewed in Hastings *et al.* 2005; Higgins and Richardson 1996). This form of modelling has also identified the role of different spread pathways (Robinet *et al.* 2009) and valued the adoption of a strategic control zone to slow the spread of EPP (Buckley *et al.* 2005; Sharov 2004; Sharov and Liebhold 1998).

Given this substantial body of work exists, it is somewhat surprising that there appear to be relatively few attempts to build spread models with a view to more effective management of invasive species at a regional scale (but see Buckley, Brockerhoff *et al.* 2005; Fox *et al.* 2009; Higgins *et al.* 2000). This may be due to several persistent problems plaguing dynamic spread modelling. Firstly, there is a propensity for spread models to occupy inevitably all of the available habitat space. This results from the exponential process that spread models attempt to represent, the reproduction and dispersal of a population distribution within a finite environmental resource, in this case space. In addition, the outcome of each temporal step results in repeating divergence between replications. That is, the species distribution at a particular time step is based on the stochastic events of every previous time step. A third problem concerns the lack of proper validation opportunities with which to engender confidence in the approach.

Since there is scarce literature to draw from that explicitly sets out the process decision-makers should follow when attempting to use spatio-temporal risk mapping tools, it is prudent to start with a few of the basic steps before moving into model design and use.

3.1.2.2. Scale

To maximise the effectiveness of maps or pictorial representation of invasive species risks, the first essential step is to use the correct spatial dimension (Pitt *et al.* 2009a). The economic, environmental and social risks posed by invasive species are complex with interactions at various scales due to different entry pathways, establishment and spread vectors (Yemshanov *et al.* 2009). Indeed, Gibson and Austin (1996) assert that since they are so complex, deterministic models may be most appropriate for representing the spread of epidemics over large spatial scales.

Predicting species abundance and distribution at coarser scales can be achieved through habitat suitability modelling. Species niche models can and have been applied to assess species invasion risks (e.g. Kriticos *et al.* 2007; Sutherst and Maywald 2005), and climate change impacts on species potential ranges (Stephens *et al.* 2007) (the latter being transferable in space or time (Randin *et al.* 2006))[2]. Jarvis and Baker (2001), for example, use a process-based insect phenology model running at a daily time step for 30 years over 1km grid squares to predict the possible effects of the Colorado potato beetle in England and

[2] At present, climate-based niche modelling techniques typically employ gridded climate datasets of moderate spatial resolution (0.5 degree), although biosecurity decision-makers continually seek greater spatial precision in the risk map products (Kriticos and Leriche 2010).

Wales. Sutherst *et al.* (2007) discuss sources of change in plant pest distributions over time under climate change scenarios, and likely effects over time.

It is also possible to use species assemblage to infer likely future distributions of invasive species over large scales using self organising map analysis, which is a type of artificial neural network. This technique uses worldwide species associations to determine which species have the highest likelihood of establishing in a particular region (Paini *et al.* 2010; Worner and Gevrey 2006). Gevrey and Worner (2006) a worldwide species distribution data from CABI/EPPO (2003) to predict the likelihood of two pest species, the Mediterranean fruit fly (*Ceratitis capitata*) and gypsy moth (*Lymantria dispar*), becoming established in New Zealand in any given year. It is important to note that self organising map analysis does not specifically map spread over time, but instead calculates the likelihood that a species will become established in a certain region given its presence or absence in comparable regions around the world.

At finer scales (e.g. regional or sub-regional), species spread models which represent populations as collections of discrete individuals rather than as a continuum may be more appropriate than habitat suitability models or artificial neural network analysis. At these more refined scales, stochastic spatio-temporal epidemiological models enable decision-makers to have the randomness inherent in real biological systems represented to them in model form[3]. However, it should be borne in minds that the model design chosen by an analyst informing a risk management decision may be very influential on the choice made by decision makers. For instance, control strategies can be highly sensitive to the particular form of stochastic model selected (Gibson and Austin 1996).

3.1.2.3. Explanatory variables

Having identified an appropriate modelling scale or resolution to represent invasive species risks to a decision-making group, the particular geographical area to be considered by the group must be established. This may be a small sub-set of the area simulated by probability models, or it may involve the entire area. In instances where the study area selection is simply dictated by the resource allocation decision that needs to be made, the choice of what area is considered is relatively straightforward. Where this is not clear, expert testimony and stakeholder knowledge may be required to refine the appropriate or preferred area.

Once the study area has been clearly identified, the relative abundance of available information on that area must be determined. Guisan and Zimmerman (2000) outline four main sources of environmental information that ideal for the purpose of characterising the study area: (1) field surveys or observational studies; (2) printed or digitized maps; (3) remote sensing data (numerical aerial photographs and satellite images), and; (4) maps obtained from GIS-based modelling procedures. In relation to the management of species populations, delineation of the study area depends on the data sampling plan and whether difficult-to-detect individuals or groups are present, be they newly established invasive pests, nearly eradicated pests, or displaced species.

Venette *et al.* (2002) review the literature related to the detection of rare individuals in order to improve management. They suggest that sampling for rare species should follow the

[3] The use of individual-based, spatio-temporal stochastic models is not new. Mollison (1977), for instance, uses models scaled at the level of the individual to predict the spatial spread of a population or epidemic. In these models each population member produces offspring according to a Poisson process with the displacement between offspring and parent drawn from a probability distribution, known as the *contact distribution*.

biology of that particular organism but also the principles governing the power of a sampling strategy. They recommend the use of the fundamentals of probability theory as a foundation for any sampling or monitoring program, with consideration of the level of inference that can be drawn from these samples, especially when resources are limited. Specific approaches include binomial, beta-binomial, and hypergeometric-based sampling strategies for quarantine inspections for exotic pests.

Since, in most cases, resources devoted to surveys are small relative to the area possibly affected, there are methods that can be employed to maximise the value of the information available about the chosen study area. For instance, Carpenter *et al.* (1993) predict bettong distributions using DOMAIN which is a range-standardized, point-to-point similarity metric that quantifies the similarity between two sites. This method performs well using presence only data and is sufficiently flexible for use in sampling survey design, reserve selection and potential mapping of rare and common species. Guisan and Zimmerman (2000) suggest the use of the Digital Elevation Model (DEM) in species distribution modelling and mapping as it spawns other environmental variables such as aslope and aspect[4].

However, climatic variables are also of central importance and constitute important information that must be garnered about a study area. Sutherst and Bourne (2009) find that regression models are unable to explain different seasonal patterns across latitudes and longitudes due to selective independent variables in their study context. This variable selection issue can be partially overcome by using factors such as annual average temperatures and rainfall or moisture indices, but fall short in considering biologically relevant combinations of suitable temperature and moisture, extreme conditions of different durations or by using different modelling methods (Sutherst and Bourne 2009). Population distribution range densities are determined by many variables that interact in complex ways through space and time. Recent studies have highlighted influences of heterogeneous temperature, population demographics, community interactions (e.g. keystone species), biogeographic differences and anthropogenic effects (Sagarin *et al.* 2006). Jarvis and Baker (2001) focused on the assessment phase of pest risk analysis and in particular aspects relating to the likelihood of a pest becoming established in a country after arrival based on the host temperature during its developmental period.

3.1.3. Comparison of modelling approaches to ensure relevant selection

3.1.3.1. Explanatory variables

Invasive species distribution can be modelled using a large variety of deterministic methods. Included in these methods are Generalized Linear Models (GLMs), ordination and classification methods, Bayesian models, locally weighted approaches (e.g. GAM), environmental envelopes or even combinations of these models (Guisan and Zimmermann 2000). Table 1 (p. 11), adapted from Guisan and Zimmermann (2000), provides a summary of these and other species distribution modelling methods. Barry and Elith (2006) suggest the use of flexible regression techniques such as BIOCLIM, Distance-based models, and various regression techniques. Selection of an appropriate method should not depend solely

[4] In this paper, Guisan and Zimmerman (2000) clearly distinguish between spatial resolution and map accuracy. Map accuracy can be tested by determining the errors of mapped entities or gradients. For example, a DEM and its basic derivatives – slope, aspect, topographic position and curvature – may be the most accurate maps available, but will not necessarily have the highest predictive potential.

on statistical considerations but should also consider the shape and nature of the species' response. Regression-based techniques such as GAM, Multivariate Adaptive Regression Splines (MARS), Boosted Regression Trees (BRTs) and maximum entropy modelling offer better performance than GLMs due to flexibility in response curves (Barry and Elith 2006). One advantageous feature of more rarely used models such as BRT, Maximum Entropy (MAXENT), and MARS is that they all share a high level of flexibility in fitting complex responses (Elith *et al.* 2006).

To better enable the use of historic and available occurrence data (presence data alone) Elith *et al.* (2006) compare 16 modelling methods over 226 species from six regions of the world. Presence-only data is then used to fit models, and independent presence-absence data to evaluate the predictions. They then make a comparison between common models such as GAMs, Genetic Algorithm for Rule Set Production (GARP) and BIOCLIM, and more rarely applied techniques such as BRT, MAXENT, GDM and MARS, to model species' distributions. Interestingly, they find that the novel methods consistently outperform the more established methods.

Leathwick *et al.* (2005) also incorporates MARS, a technique that uses piece-wise linear segments to describe non-linear relationships between species and environmental variables. Analysis results are imported into a Geographic Information System. Guisan and Harrell (2000) show how models based on ordinal data, which is common in ecology, perform just as well as logistic regression for presence/absence and abundance predictions for plants. Models include the Proportional Odds, the Continuation Ratio and the Stereotype models. Aspinall (1992) used a predictive spatial distribution model for deer in Scotland based on Bayes theorem. The uniqueness of the papers' approach lies in the use of a combination of different data sets to predict a single data set. Guisan *et al.* (1998) analyse and predict correlations between alpine plant species distribution and environmental variables using two types of GLMs in Switzerland. The first model uses a binomial GLM with only the mean annual temperature, while the second uses a logistic model restricted to areas within temperature range so that ordinal abundance data can be adjusted. Both models are mapped using GIS. The stratified modelling approach is concluded to better fit the variability within the optimal altitudinal zone for the species. As the model does not include areas outside of the species range, the prediction of new areas, as required in invasive pest modelling, may not be well adapted to this technique.

Biomapper is a GIS and statistical tool designed to build habitat suitability models and maps for different species of animal or plant (Hirzel *et al.* 2002). It is based on the Ecological Niche Factor Analysis that computes HS models without absence data and that explain the ecological distribution of the species. The extracted factors are totally uncorrelated but have biological signification. This first factor is the marginality factor, which describes how far the species optimum is from the mean habitat in the study area. The specialisation factors are sorted by decreasing amount of explained variance. They describe how specialised the species is by reference to the available range of habitat in the study area. Therefore, only a few of the first factors explain the major part of the whole information.

Araújo and New (2007) advocate the use of multiple models within an ensemble forecasting framework and described alternative approaches to the analysis of bioclimatic ensembles, including bounding box, consensus and probabilistic techniques. An ensemble is an idealization consisting of system copies, considered all at once, each of which represents a possible state that the real system might assume at some specified time. Multiple copies are simulated across more than one set of initial conditions, model classes, parameters and boundary conditions (predictors in a statistical model e.g. climate variables). Each combination is one possible state of the system being forecasted. Multiple simulations using

different parameter values enable parameter uncertainty to be assessed. Araújo and New (2007) use different model classes including polynomials and smoothing splines of different orders in general linear or additive models, nodes in classification and regression trees, hidden layers in neural nets, and various forms of process-based models. Model types included Artificial neural networks, Bagging trees, Boosted additive trees, GARP, and MAXENT.

Table 1. Modelling techniques/tools used to predict invasive species distributions.

Modelling technique	Type of predictions	Description	Type of response variable	Capability for treatment of Uncertainty	Spatial	Reference
BIOCLIM	Probabilistic	Envelope model- Climate pattern-matching with minimum bounding rectangle (MBR)	P	No	Capability to inform GIS	Elith J, *et al.* 2006; Barry S, Elith J. 2006
Classification tree	Class Multinomial	General statistical procedure for defining set membership based upon environmental correlates	PA	Yes	Capability to inform GIS	Guisan A, Zimmermann NE. 2000; Araújo and New 2007
GARP	Probabilistic	rule sets from genetic algorithms - Generates environment-description rules using machine-learning techniques	PA	No	Capability to inform GIS	Guisan A, Zimmermann NE. 2000; Peterson AT, Vieglais DA. 2001; Elith J, *et al.* 2006; Araújo and New 2007
GAM	Probabilistic	regression: generalised additive model	PA	Yes	Capability to inform GIS	Richardson DM, Thuiller W. 2007; Elith J, *et al.* 2006
GLM	Probabilistic	regression; generalised linear model	PA relative abundance, Individual counts, species richness	Yes	Capability to inform GIS	Guisan and Theurillat, 2000; Vincent and Haworth, 1983
MARS	Probabilistic	regression; multivariate adaptive regression splines	PA	Yes	Capability to inform GIS	Leathwick *et al.* 2005
MAXENT	Probabilistic	maximum entropy Probabilistic machine learning technique based on the distribution of maximum entropy	PA	No	Capability to inform GIS	Phillips *et al.* 2006; Araújo and New 2007
maximum-likelihood classification	Probabilistic	based on two principles of normal distribution of cells in the multidimensional space and Bayes' theorem.	Qualitative (categorical, nominal)	considers both the variances and covariances of the class signatures	Capability to inform GIS	Frank, 1988
Bayes formula	Probabilistic Binomial	shows the relation between one conditional probability and its inverse	PA	Uncertainty analysis	Capability to inform GIS	Aspinall, 1992; Brzeziecki *et al.* 1993
Artificial Neural Networks (ANN)	Classification	General modelling technique based on machine learning	PA	Rare or often only point estimates however Bayesian techniques possible	Capability to inform GIS	Gevrey and Worner 2006
CLIMEX	Probabilistic	Match climates function Climate pattern-matching procedure generates an index of climatic similarity	PA	Sensitivity Analysis	Built-in	Sutherst RW, Bourne AS. 2009
DOMAIN	measure of multivariate distances	Climate pattern-matching using a point-to-point similarity index	P	variable sensitivity	Built-in	Carpenter *et al.* 1993
BIOMAPPER - ENFA (Ecological Niche Factor Analysis)	Probabilistic	Computes suitability functions by comparing the species distributions in ecogeographical variables space with that of the whole set of cells using a multivariate approach	P	No	Built-in	Hirzel *et al.* 2000
NAPPFAST	Probabilistic	Online templates for phenology, infection, and empirical models and a climate-matching tool	PA	Identifies biases and uncertainty ranges at fixed levels of risk using Percent Absolute Difference (PAD) analysis.	Built-in	Magarey *et al.* 2007

3.1.3.2. Climate matching and envelopes

Climate matching is a common technique used to predict where exotic species could occur if establishment in a new region is successful. Richardson and Thuiller (2007), for example, use nonparametric niche-based modelling (i.e. a generalized additive model - GAM) calibrated on the current distribution of each South African biome to map regions of the world that are climatically similar to South African biomes. They determine climate matched countries and biomes in order to evaluate potential invasive plant distributions in South Africa. GAM is used to relate the biome distributions to the four selected bioclimatic variables. The model is calibrated using a random sample of the data and using Akaike Information Criterion (AIC).

Matched climatic conditions do not, however, perfectly explain where a species could occur. The extent and distribution of invasive species are influenced by interactions between environmental conditions such as climate and anthropogenic factors. Hence, when using an approach like GAM exceptions occur in fragments of other biomes, riparian zones, and areas that were planted by humans (Richardson and Thuiller 2007).

Nevertheless, climate matching still serves an important role in providing screening information that can act as a starting point in the modelling procedure and make the decision-making process more objective. Additional factors need to be considered alongside climate such as the roles of competition or mutual symbiosis in defining actual invasive potential (Richardson and Thuiller 2007). This is evident in Hartley *et al.* (2006) who note that Argentine ant (*Linepithema humile*) is often competitively dominant against other ant species, and can adapt to wide variety of novel hosts, despite a lack of co-evolutionary history. Climate can alter the state of these interactions, and therefore still exerts a sizeable influence on invasive ant abundance and distribution.

3.1.3.3. Stochastic modelling

To capture and characterise the uncertainty inherent in invasive species spread over time, a stochastic simulation model may be appropriate. This approach has not been as widely employed as deterministic approaches, but several important studies focusing on invasive species issues can be cited. Yemshanov *et al.* (2009) use a spatial stochastic simulation to quantify pest risks and uncertainties. Rafoss (2003) develop a method to predict the establishment and spread of a bacterial disease of potato. The study uses a stochastic simulation in GIS to combine environmental variables and simulate dissemination behaviour of the pest. This paper attempts to define the size of an area affected by an introduction of the disease to a given new region. The stochastic model specifies specific land types (e.g., potato cropping areas) and treated entry as a random event.

The study contained in Pitt *et al.* (2009a) models *L. humile* spread using a spatially explicit stochastic simulation model of dispersal within a GIS framework to recreate the historical spread of the insect in New Zealand. Probabilistic maps are used to simulate local and human-assisted spread to identify areas at risk of infestation. These model predictions are compared to a uniform radial spread model in terms of its ability to explain the historical data. Their results indicate that the uniform spread model performs optimally early in the invasion process, but the simulation model is more successful in the latter stages of the simulation. This finding is used to highlight the potential for different search strategies to be effective at different stages in an invasion when attempting to optimize detection.

The Pitt *et al.* (2009a) study uses raster maps to represent population distributions and open source software - Python and C, within the open-source GIS GRASS (Geographic Resources

Analysis Support System). The model is based on a raster map for each year to represent either the presence or absence of the species in a raster cell. The software developed by the author is titled MDiG and presents an open and standardized platform for species dispersal simulation. The results display probability distributions of possible future spread scenarios for the species. Rather than making specific conclusions about where the species will have established and at what time, the results indicate a relative likelihood of establishment across the landscape.

There are several distinct advantages of the MDiG modelling approach put forward in Pitt *et al.* (2009a). Firstly, it allows replicates of model runs, keeps track of all the maps, and can merge into an average map for each time step. Secondly, MDiG captures different means of spread including long distance, shaped neighbourhoods and local contiguous. Local spread via budding and jump dispersal facilitated by human transport can be simulated using the model. Potential spread rates are influenced by dispersal kernel shapes that describe the distance that propagules travel and Allee effects that can limit spread rates and constrain population fronts that otherwise are predicted to accelerate indefinitely. A budding spread rate of 150 metres per year for regions where habitat and climate are not limiting (raster resolution of 150 metres). The dispersal model links to the habitat suitability layer to dictate survival and controls the probability that an occupied cell might become extinct. The suitability layer is created by expert knowledge about the suitability of various land cover types for persistence of populations of this species.

Similar flexibility in application can be found in the NetLogo platform. Netlogo is a multi-agent programmable modelling platform designed in the Logo programming language to enable quick and easy authoring of models. Net Logo's 'patches' and 'turtles' structure was particularly suited to the problem of simulating EPP movements via multiple dispersal mechanisms. Stochastic jump dispersal via intermediate host transmission and human-aided spread can be accommodated. Moreover, Net Logo's ease of use and capacity to automatically display spread as it occurs and allow dynamic inputs of parameters during model runs lends itself to use in DMCE-style workshops where stakeholder-defined real time 'what-if' scenarios can be run, facilitating interaction and engagement in the problem. The availability of GIS extensions also allow the use of raster datasets to display EPP spread across real landscapes, thus adding realism users.

While stochastic modelling is preferable in group-decision making requiring the full extent of uncertainty to be made known to decision-makers, it is noted that it is also possible to use a simpler deterministic modelling approach to minimise complexity. However, there are dangers associated with this approach. For instance, Mayer *et al.* (1993) compare deterministic and stochastic models of screwworm fly (*Cochliomyia hominivorax*) incursion into Australia. They conclude that the main discrepancies between the models occur at the fringes of the expanding infestation, with the deterministic model under-predicting population densities. Essentially, the deterministic model fails to detect the small proportion of the population at the front line of the incursion, while the stochastic model does not. Modellers of systems that encompass extreme events and distributions should consider this difference in model selection.

3.1.4. Evaluate the predictions – power and variance

Simulating invasive species impacts over time and projecting them on to maps is invariably a complex exercise involving a lot of biological and ecological uncertainty. It is therefore very important that controls are put in place to avoid the misinterpretations of spread and impact that have the potential to mislead stakeholders. For this reason, independent evaluation of

invasive species risk models is needed to avoid flawed results being used to inform decisions, or model results being extrapolated inappropriately.

Several studies highlight the need to evaluate model predictions. Sutherst and Bourne (2009), for instance, compare logistic regression and CLIMEX models in predicting range extensions of the non-equilibrium distribution of the livestock tick in Africa. They find that logistic regression better describes the spatial data but displays inferior performance to CLIMEX in predicting range extensions. They therefore question the effectiveness of descriptive, statistical models (i.e. logistic regression) alone to predict changes in species ranges. Peterson and Vieglais (2001) use the GARP modelling method for ex post (i.e. after the invasion event) projection of models onto new landscapes. Peterson *et al.* (2008) advise that absence data should not be employed in evaluating model quality in niche model applications. This is because ecological niche models are often based on species presence information alone due to a lack of absence information. Even if absence data is available, it is often restricted to current distributional area (Peterson, Papes *et al.* 2008).

The Receiver Operating Characteristic (ROC) curve provides one option for model evaluation when employing invasive pest risk models as decision-support tools. The ROC curve is a commonly applied approach to evaluating predictive distribution models that avoids subjectivity in the threshold selection for evaluated probabilities by summarizing model performance over all possible thresholds. However, Lobo *et al.* (2008) questions the reliability of the ROC curve and cautions against its use for several reasons, including: (a) ROC ignores the predicted probability and the goodness-of-fit of the model; (b) it considers model performance in probability levels across the ROC curve which could be irrelevant to the evaluation; (c) it weights false positive and the false negative errors equally; and (d) it does not give information about the spatial distribution of model errors. Lobo *et al.* (2008) make these criticisms based on comparison among models of different species. Of course, species differ in home range sizes and these problems may not be relevant for model comparison for single species. For example, in regards to criticism (c) a modified ROC can be used that substitutes absence data for proportion of area considered to be presence (Peterson, Papes *et al.* 2008; Phillips *et al.* 2006). Petersen *et al.* (2008) recommend a modified ROC procedure that disposes of absence data, instead using x-axis values as the proportion of the overall area predicted as present, rather than using commission errors based on the aforementioned issues of absences.

3.1.5. Provide a map of prediction confidence with levels of uncertainty

3.1.5.1. Indicate where the model is applied, interpolated and extrapolated

It is important when using visual devices like maps and figures in group-based MCDA to be as open and transparent as possible in regards to the uncertainty inherent within it. Venette *et al.* (2010) highlight the need for substantial improvement in visual decision-support model documentation, communication of uncertainty, data accessibility, human behaviour (i.e. agriculture interactions) and improved training. It is important not to portray a false sense of accuracy to decision-makers by concealing what may or may not be captured by a species impact map, or the model behind this map.

With this in mind, Sutherst and Bourne (2009) recommend statistical models combined with GIS for interpolating sample data to fill in missing values. However, for extrapolating beyond the data sets, as is necessary with species invasions or climate change scenarios, a different approach is called for using a tool like CLIMEX. Rather than trying to achieve a precise description of the distribution (i.e. using regression), CLIMEX interrogates the data

understand critical climatic conditions for a species (Sutherst and Bourne 2009). Barry and Elith (2006) consider the sources of errors in species habitat models. They divided them into two main classes: (i) error resulting from data deficiencies, and (ii) error introduced by the specification of the model. Common data errors include missing covariates and samples of species' occurrences that are small, biased or that lack absences. Almost all models examined in Barry and Elith (2006) contain missing covariates, which introduces significant spatial correlation in the errors of the analysis. Aspinall (1992) create error bounds by using random subsets of the data in a bootstrapping type method. Errors are modified within the GIS by changing from 50m pixel to 1km grid square resolution. The key message of the paper is that by analysing the errors, the model results can be interpreted more appropriately.

Several papers have reviewed uncertainty methods available for spatial distribution modelling. Elith *et al.* (2002) review the aspects of uncertainty and methods that are relevant to habitat maps developed with logistic regression. They address the problems of user, model, and random and systematic errors and suggest methods for developing realistic confidence intervals in relation to decision-making. Regan *et al.* (2003) analyse treatments of uncertainty in a variety of population models. The authors define uncertainty as ignorance about parameter values (e.g. measurement error and systematic error). Risk models include an analysis of variability and parameter uncertainty to give the most comprehensive and flexible endpoint. The paper looks at different risk assessment models at the population level and the relevant sources of uncertainty, and identifies *which* modelling techniques have *what* level of uncertainty treatment (see Table 1, "Capability for treatment of Uncertainty" column, p. 10).

Pitt *et al.* (2009a) attempt to tackle uncertainty in stochastic models by random sampling from the spread kernel and survival module probability distributions. Hartley *et al.* (2006) develop a novel method to test for uncertainty in spatial predictions specifically for invasive pest distribution models. Their approach uses a multi-model inference to generate confidence intervals that incorporate both the uncertainty involved in model selection as well as the error associated with model fitting. Using *L. humile* as a case-study, the uncertainty analysis is used to determine that not only is the ant most likely to occur at a 7-14°C mean daily temperature in midwinter, but also an important extreme value at the maximum daily temperatures during the hottest month averages 19–30°C. The approach quantifies the costs of making false negatives vs. false positives in order to connect modelling to decision-making[5].

Methods also exist that aim to make the best decision in the face of extreme uncertainty. Moilanen *et al.* (2006) apply information-gap decision theory to develop uncertainty analysis methods for reserve selection in order to seek a solution that is robust in achieving a given conservation target, despite uncertainty in the data. Information-gap theory uses 'distribution discounting', in which the conservation value is penalized by an error measure termed *accuracy of statistical prediction*. Information-gap theory can accommodate non-statistical uncertainties such as the subjective choice of candidate variables and the structural assumptions embedded in spatial analysis to account for unknown levels of potentially-extreme uncertainty. The trade-off between predicted probability (i.e. in the case of Moilanen *et al.* (2006), conservation priority value) and the certainty of the prediction may

[5] Hartley *et al.* (2006) quantify false negatives by evaluating the unnecessary effort that is expended in border surveillance and response to an incursion against a species that could never establish. This ignores the possibility of a single surveillance procedure designed for one species detecting multiple species. Social and ecological costs that would be incurred in the event of a successful invasion also need to be considered.

lead to different decisions that reflect the planner's attitude towards risk. Choosing sites that have lower conservation values with more certainty reflects aversion to risk.

3.1.5.2. Consider species habitat and home range

Ecological habitat and species home range are essential in distribution modelling, be it deterministic or stochastic, and can serve as a practical sensibility test for risk maps derived from probability models. As mentioned previously, climatic considerations are a large component of habitat suitability. However, additional ecological variables also garner consideration, and in some cases there may be a great deal of uncertainty as to how these variables will impact distribution patterns.

Peterson and Vieglais (2001) provide an example of predicting invasions by projecting the ecological model onto landscapes that are likely to be invaded. They use a web interface to apply the derived rule set manually to a parallel set of coverages specifically for the test region of special interest. An alternative and more practical approach is to develop the ecological model on a set of coverages that extend across both the native and the potentially invaded regions.

In some instances homogenous habitats can be assumed. For large, broad-acre agricultural regions this may be appropriate, but for more diverse landscapes into which an invasive species may be introduced the spatial heterogeneity must be considered. For complex spatial environments, metapopulation models (e.g. Hanski *et al.* 2000) or stochastic patch occupancy models (e.g. Moilanen 2004) may be appropriate.

3.1.6. Reclassify predictions into robust, meaningful, and honest values for policy makers and the public

Pest risk maps can be powerful visual communication tools to describe aspects of an incursion (Venette, Kriticos *et al.* 2010). They enable decision-makers to receive a wealth of information relatively quickly, and to visualise the threat posed by invasive species. Numerous spatial decision support tools for workshop environments have been developed and applied with success. A summary of these applications is contained in Table 2, below. The decision problems to which they are applied are varied, but illustrate the general applicability of mapping techniques group-based decisions. We outline some of the techniques and applications in more detail below.

The Multi-Criteria Analysis Shell for Spatial Decision Support (MCAS-S) is a software tool produced by the Australian Department of Agriculture, Fisheries and Forestry (DAFF) Bureau of Rural Sciences. MCAS-S is a spatial decision-support tool designed for application in real-time stakeholder workshops, where it helps participants visually link mapped information to a Multi-Criteria Decision Analysis (MCDA) decision making framework (Lesslie *et al.* 2008). MCAS-S can be used with issues of various scales and resolutions, and does not require GIS programming knowledge by the user. User-friendly features of MCAS-S include the capability for a decision-making group to view, classify and combine different types of mapped information in an interactive, real-time setting. MCAS-S can also produce statistical reports for specific regions quickly and simply.

Table 2. Multi-criteria decision analysis spatial tools: a selected list of GIS-based and standalone software-based applications for natural resource management issues (Lesslie *et al.* 2008).

Software/analysis	Application	Reference
1. GIS-based applications		
IDRISI (®Clark University) GIS-based MCA	Earthquake hazards; crop suitability; soil erosion in Ethiopia	Ceballos-Silva and Lopez-Blanco (2003); Dragan *et al.* (2003)
ASSESS (A System for SElecting Suitable Sites) written in *ArcInfo* AML (®ESRI)	Radioactive waste repository; soil conditions; catchment condition	Veitch and Bowyer (1996); Bui, (1999); Walker *et al.* (2002b)
ArcView (®ESRI) GIS-based MCA Planning tool	Urban land use	Pettit and Pullar (1999); Dai *et al.* (2001)
ILWIS GIS	Nature conservation value of agricultural land	Geneletti (2007)
MapInfo (®) GIS-based DSS	Urban transport policies	Arampatzis *et al.* (2004)
Spatially-explicit sensitivity analysis framework for decision making	Invasive plant pest management	Roura-Pascual *et al.* (2010)
2. Hybrid applications		
SIMLAND – cellular automata, MCA and GIS written in C and using *ArcInfo* GIS	Land use change	Wu (1998)
HERO (Heuristic multi-objective optimisation) combined with GIS, AHP and Bayesian analysis	Forest planning; habitat suitability	Kangas *et al.* (2000); Store and Kangas (2003); Store and Jokimaki (2001)
3. Stand-alone software		
LMAS – Land Management Advice System	Spatial expert system	Cuddy *et al.* (1990)
MULINO-DSS (MULti-sectoral, INtegrated and Operational DSS) combines simulation models, mapping and MCA	Water resources	Giupponi *et al.* (2004)
IWM – decision support system for Management of Industrial Wastes	Industrial waste	Manniezzo *et al.* (1998)
GSA (Global Sensitivity Analysis) in *SimLab* (Software for Uncertainty and Sensitivity Analysis)	Hazardous waste disposal	Gomez-Delgado and Tarantola (2006)
MCAS-S - Multi-Criteria Analysis Shell for Spatial Decision Support	biodiversity and salinity mitigation trade-offs in revegetation	Lesslie *et al.* (2008)
CommunityViz planning software and the Placeways suite of GIS offerings provide a real-time interactive environment of 3-D visuals, intelligent maps and dynamic analysis tools.	Economic options for rural areas, urban planning, conservation planning	Placeways, LLC Ltd.

CommunityViz® (Placeways LLC, Boulder, Colorado) is another software package that facilitates decisions in a workshop environment and can bring in pest risk maps in a user-friendly manner. The software serves as an extension to ArcGIS (ESRI) in order to create an interactive decision-making platform. The software is designed to inform decisions concerning alternative futures (scenarios) by analysing decision effects, and can create three-dimensional (3D) map outputs. CommunityViz is designed for real-time workshop communication. Some aspects of using this software package are user-friendly so that an inexperienced operator can utilise them, while others are more sophisticated and require knowledge of GIS.

A framework for deciding among options, in the form of static priority maps is developed for the management of woody invasive alien plants in South Africa in Roura-Pascual *et al.*

(2010). The framework features a spatially-explicit sensitivity analysis. The authors use a combination of analytical hierarchy process, Earth mover's distance, Shannon Diversity index and Akaike's Information Criteria to determine the best management option based on sensitivities among methods. Unlike MCAS-S and CommunityViz, the majority of the analysis in Roura-Pascual *et al.* (2010) is completed 'behind the scenes' by an analyst. However, the models are linked to the decision problem and can therefore be used as part of a decision-making group workshop by presenting clear, meaningful maps. Roura-Pascual *et al.* (2010) includes criteria related to management history, fire risk, and the age, identity, density and spread of invasive plants. Each factor has a weight associated with it that reflected its relative importance in prioritizing areas for management. The authors change the weights using three types of sensitivity analysis and assess the effect of these changes on the spatial structure of the resulting priority maps in three different management regions. Model outcomes are not considered as discrete elements by evaluating rank order when changing the decision criteria, but instead spatial configuration is evaluated spatially explicitly using distance measures. By determining the importance of criteria in shaping priority maps, the sensitivity analysis framework enables the identification of necessary criteria to produce outcomes matching pre-selected management objectives. This is crucial for cost-effective management, as acquisition and curation of data is expensive.

3.1.7. Summary

Section 3.1 has reviewed a cross section of the modelling and ecology literature and described methods and techniques that may be useful in developing visual information tools for use in group-based MCDA for invasive species risk management decisions. These decisions might involve the prioritisation of species by industry or region, the prioritisation of pest and disease entry pathways, or choosing the most desirable pest management strategies post-invasion. In all of these decisions, an invasive species population, spread and distribution model is useful in helping the decision-making group to appreciate the idiosyncrasies of individual invasive species, and to respond to these threats more effectively. Projecting this population and impact information on to maps familiar to the group will enhance the uptake of this information by placing decision-makers psychologically closer to incursion events, but the process of doing so is complex. In this section, we have discussed some of the basic steps that should be followed, including choosing model scale, clarifying the area of interest for the MCDA and choosing the form of population model to use to project population distribution and abundance on to maps. We have highlighted methods that can be used to evaluate the strength of model predictions and communicate this to decision-makers through maps. We have also discussed a range of group decision-making applications of maps-based approaches, and identified some of the tools used.

This background knowledge will be extremely beneficial in the design and use of state-of-the-art maps-based tools to help Australian plant industries to better manage the biosecurity threats facing their industries. We will draw upon this knowledge in section 3.3, but prior to this we review the literature on MCDA with an emphasis on group-based deliberative processes in section 3.2. These approaches provide a vehicle for the transfer of knowledge between technical support (i.e. invasive species modellers, risk assessors, etc.) and those charged with the responsibility of using the information provided to them to make decisions that may or may not be reversible.

3.2. Deliberative Multi-Criteria Evaluation

3.2.1. Introduction

In this section we review the literature on group-based solutions to multi-faceted resource allocation problems like biosecurity. While one of our project goals involves the creation of a spatial tool for use in incursion management, we must provide some guidance on how this is to be incorporated into decision making. That is, we need to identify a very practical, structured way to employ spatial bioeconomic models to make response decisions (e.g. do we eradicate an outbreak, contain or slow it, or do we take actions to live with it for perpetuity?) clearly, decisively and rapidly.

The prevention and management of EPPs regularly involves two fundamental problems. Firstly, risk management decisions frequently involve trade-offs between complex and often competing environmental, social and economic objectives with potential positive or negative consequences for different social groups. Secondly, understanding of these risks is often marked by profound uncertainty (Gregory *et al.* 2006). When combined, these challenges too often become excuses for maintaining the *status quo* instead of considering alternatives that might result in net social welfare gains (Liu *et al.* 2010). The risks frequently concern multiple stakeholders, each with their own perspectives and priorities for preventing an undesirable species from establishing, and for managing its impacts once it has established. In addition, a high level of uncertainty prevails concerning each step in the invasion process, and about how human actions can alter the process of invasion. Risk analysts faced with evaluating the risks of future invasions often have little information on the likelihood that a species will arrive, establish and spread in a new environment, and on the potential impacts should this occur. This is particularly true when the potential consequences of invasion are of a long-term and large-scale nature (Strayer 2009; Strayer *et al.* 2006).

The high level of uncertainty is in part explained by the fact that the limited amount of data we collect about invasions is not reliably representative (Franklin *et al.* 2008). Two reasons may explain this problem of under-representation: (1) only a small proportion of EPP spread and cause harm (Mack *et al.* 2000), and (2) biological invasions frequently involve novelty (Williamson 1999). Yet, numerous studies have shown that the impacts of this small group of EPPs could be irreversible and tremendous (Millennium Ecosystem Assessment 2005; Pimentel *et al.* 2005).

Due to these low-likelihood, high-novelty and high-impact characteristics, it has been argued that EPP risks are difficult to handle within a conventional risk management framework (Horan *et al.* 2002; Simberloff 2005). In this paper, we argue that the conventional model has limited use in managing EPP risk for at least two reasons. Separation of risk assessment and management disrupts essential connections between the social values at stake in risk management and the scientific research involved in gauging the likely impacts of management actions, leaving the risk management decisions to be made in the wake of political pressures that reflect competing views on the proper tradeoffs among competing values (Maguire 2004). Furthermore, the severe uncertainty associated with the scientific analysis tends to be insufficiently communicated (Valle *et al.* 2009). This lack of communication may result in overconfident decisions at one extreme; at the other extreme, it could lead to a crisis-driven or 'fire-fighting' approach (Shea *et al.* 2002) to EPP risk management, characterized by inaction before incursion happens, and potentially damaging over-reaction when incursion does occur.

One new decision-support tool that overcomes the two limitations of the conventional model by taking into account social values and uncertainty is Deliberative Multi-Criteria Evaluation

(DMCE) (Figure 1). DMCE seeks to combine the advantages of Multi-Criteria Decision Analysis (MCDA) in providing analytical structure to assess multi-dimensional objectives with the benefits of stakeholder participation (Proctor and Drechsler 2006). Compared to MCDA without a public involvement component, DMCE provides an opportunity for diverse stakeholder views to be explicitly incorporated within the decision-making process (Rauschmayer and Wittmer 2006). In addition, the DMCE can also function as a platform for risk communication, whereby scientists, stakeholders and decision-makers can interact and discuss the uncertainties associated with biological invasions. Thus, DMCE injects scientific rigor and transparency into the decision-making process of risk management by providing an analytical structure for social complexity and by integrating risk assessment and risk communication.

The DMCE method has been applied in the natural resource management arena as a decision-aid tool (Bojorquez-Tapia *et al.* 2005; Hajkowicz and Collins 2007), but it has only recently been used to assist EPP decision-making (Cook and Proctor 2007; Hurley *et al.* 2010; Liu *et al.* 2009; Liu, Proctor *et al.* 2010). In this section, we situate our methodology within the risk management and science studies literature, addressing the limitations of the conventional decision-making model and proposing to use the DMCE as a new framework for managing the risks of biological invasion. We also detail the challenges of social complexity and profound uncertainty in EPP management and explain how the DMCE can be applied to tackle them.

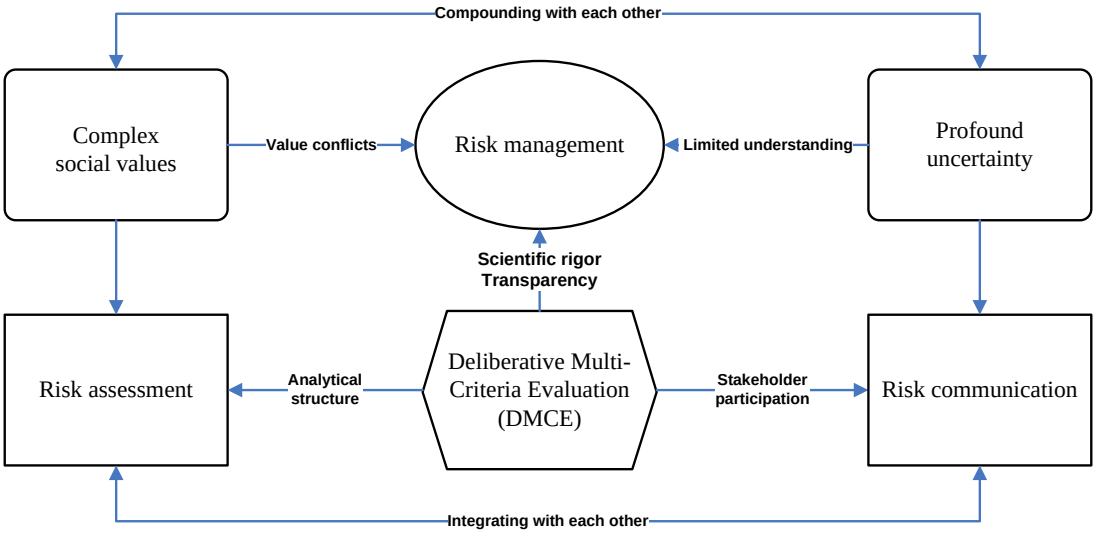

Figure 1. Using DMCE to tackle the dual-challenges of complex social values and profound uncertainty in managing biological invasions.

3.2.2. Limitations of the conventional model of risk management in handling social complexity and profound uncertainty

Risk assessment is the process of evaluating the probability of introduction and spread of an invader and the magnitude of the associated potential consequences. Conventionally, it is separated from risk management. The rationale for this separation is that the former is supposed to be a strictly scientific and value-free process, whilst the latter falls into the political domain, where diverse social values can come into play. The two processes are also different in terms of their final outcomes. A risk assessment derives *risk*, a product of the likelihood of an event and its potential consequences. The goal of risk management, by

comparison, is to identify *acceptable risk* (Fischhoff *et al.* 1981) and policy actions that manage these risks appropriately (Hummel *et al.* 2009).

This separation of risk assessment and management disrupts essential connections between the social values at stake in invasive risk management and the scientific research involved in predicting the likely impacts of management actions (Maguire 2004). As a result, the risk assessment may fail to address stakeholders' major concerns because it is increasingly clear that a quantitative expert view may be different from the views of the public at large (Waage and Mumford 2008). In addition, the uncertainty associated with the scientific analysis could be ignored by, or insufficiently communicated to, the decision-makers, leaving risk management decisions to be made in the wake of political pressures that reflect competing views.

Uncertainty has many meanings and different disciplines have their own ways to classify and manage uncertainty. For the purposes of this report, the term *risk* designates situations when possible outcomes and their probabilities are both known (e.g. throwing a dice or tossing a coin). By contrast, *uncertainty* refers to situations when we only know the possible outcomes but not the probabilities of these outcomes. For example, successful EPP establishment is positively related to propagule pressure but quantification of the probability of establishment is still a challenge for most taxa (Kolar and Lodge 2001).

Risk and uncertainty are not synonymous. Yet, one of the hallmarks of risk assessment is the probability model, where uncertainty is treated as a state that can in principle be known through objective or subjective probability distributions. An implicit assumption for such probability-based models, whether it is a Bayesian net risk assessment or a cost benefit/effectiveness analysis, is that the quality of background knowledge is sufficiently high to justify such an approximation. However, this is often not true in the case of predicting unprecedented events such as climate change (Millner *et al.* 2010) and biological invasions (Gren 2008).

Indeed, although probability-based approaches are powerful for studying simple and static systems, they are not considered adequate for complex socio-ecological systems with unforeseen or unknown future outcomes (Walker *et al.* 2002a). The choice of treating a future event as either risky or uncertain largely depends on the novelty contained in the system (Brouwer and De Blois 2008). When the system contains little or no novelty, probability approaches may be sufficient. When the degree of novelty increases, however, probability approaches may not be sufficient to predict and manage future events.

We are often faced with a high level of novelty concerning invasive species, where uncertainty or even *ignorance* (when we do not even know the range of possible outcomes) is the norm (Horan, Perrings *et al.* 2002; Williamson 1999). Even for the same species, there are many examples where it causes quite different impacts on ecosystem processes at different sites or at different times (Ehrenfeld 2010).

The uncertainty and ignorance has to be accounted for and presented to those making policy decisions. A deliberation process has been proposed for such a purpose so that risk analysts, stakeholders, and decision-makers can interact (Rodriguez-Labajos *et al.* 2009).

3.2.3. Analysing complex social values in managing invasion risks with Deliberative Multi-Criteria Evaluation

3.2.3.1. An overview of complex social values associated with biological invasions

The potential and actual impacts of biological invasions are many and varied. They may be *direct* or *indirect* (i.e. mediated through effects on other species or through an ecosystem) and may affect *market* (e.g. food, fuel, trade access) or *non-market* (e.g. ecosystem services, aesthetic enjoyment, and existence value of native species) goods and services of invaded systems. Hence, there are usually *economic, social* (e.g. human health) and *environmental* dimensions of invasions to consider (Cook and Proctor 2007; Larson *et al.* 2011). It follows that invasive species simultaneously generate multiple impacts on different social sectors.

Ideally, a risk management decision will succeed in balancing public benefits and undesirable costs to potentially affected parties, but in reality this may be difficult to achieve for potential EPP because the risks have higher uncertainty. There can even be disagreement over the magnitude of the likely impacts caused by the most high-profile invasions (Parker *et al.* 1999). Economic evaluations of biological invasions, for instance, tend to focus on direct or market impacts, while indirect and non-market impacts are often ignored or neglected because of difficulties in deriving appropriate estimates (Born *et al.* 2005). Even when such appropriate values are sought, an 'appropriate' value may vary depending on which stakeholder is asked. Different stakeholders with different agendas and priorities among the competing objectives can perceive involuntary risks very differently (Simberloff *et al.* 2005). For example, a proposal to cultivate a potentially invasive weed for the production of biofuels will benefit the prospective farmers but concern ecologists (Davis *et al.* 2010; Meyerson 2008). From this perspective, environmental decision-making is akin to conflict analysis characterized by ecological, economic and socio-political value judgments of different stakeholders.

3.2.3.2. DMCE as a decision-aid to analyze complex social values

Decision scientists argue that good decision-making requires facts, values, and a process for their integration (Gregory, Failing *et al.* 2006; Renn 1999). To accommodate diverse value judgments, public involvement in environmental decision making has become a standard practice (Wilson 2008). In the area of environmental risk management, a hybrid analytical-deliberative process has emerged, of which DMCE is an example. The hybrid approach integrates quantitative risk assessment with participatory approaches that seek to incorporate a wide range of scientific expertise, local knowledge, and diverse values through a new form of science-citizen interaction (Beierle 2002; Renn 1999; US National Research Council 1996).

Several drivers are responsible for shaping this hybridized approach to risk management decision-making. First, participatory theory and deliberative democracy assert that individuals have a right to influence decisions that relate to their welfare (Dryzek 2000). Second, the integration of diverse social values into decision-making processes has multiple benefits, including increased acceptability and strengthened trust in risk decisions (Stirling 2006). Finally, risk assessment, which was believed to be completely objective, inevitably reflects tacit yet dominant cultural values and identities and is thus not a value-free process (Slovic 1999; Wynne 1992). The key question, is not whether subjective elements should still be considered in a decision-making process - they are part of it anyway; but how they should be articulated and incorporated *via a formal and structured analysis* (Keeney *et al.* 1993). With this in mind, decision support analysts need tools to integrate technical

expertise, regulatory requirements, and public values. DMCE is one such tool that allows structured decision-making that engages multiple groups in a decision-oriented discourse incorporating both facts and values (Liu, Proctor *et al.* 2010).

The DMCE method combines the facilitation, interaction, and consensus-building features of citizens' jury processes with the structuring and integration features of traditional MCDA (Proctor and Drechsler 2006). It has been developed for more effective engagement of multiple stakeholders in the decision-making process, as opposed to a single decision maker.

The citizens' jury involves around ten to twenty participants being charged with the responsibility of constituent representation and decision-making (Proctor and Drechsler 2006). The group is guided by an independent facilitator who ensures that participants have equal opportunity to express their views and the process is able to follow a suitable course to achieve outcomes. The jury is encouraged to use expert witnesses, technical analyses, and anecdotal information to form individual opinions. Time is then devoted to information clarification and group discussion, in which group opinions are revealed and modified using an interactive computer software package (e.g. MCAT/Multi-Criteria Analysis Tools) (Marinoni *et al.* 2009). These modified group opinions sometimes indicate increased agreement among participants, which is potentially a very important feature of DMCE when used in a policy-making context (Redpath *et al.* 2004; Webb and Raffaelli 2008).

A detailed description of the DMCE process can be found in Proctor and Drechsler (2006), and is summarized in Figure 2. The essential steps involved include the following. First, the jury is selected while ensuring fair representation of the various stakeholder groups. This jury refines the overall goal of the DMCE procedure, the decision criteria, and policy options to be considered. Experts create an Impact Matrix (IM) to capture the estimated impacts of each policy option relative to the individual criteria, against which each jury member assigns weights reflecting its relative importance. Once the criteria weights and IM have been determined, a deliberative process is carried out with the aid of the facilitator and interactive computer software. For each iteration, the software reveals both individual and group preferences, thus providing a vehicle for negotiation and consensus building. Sensitivity analysis is used to demonstrate the effect of scientific uncertainty on the robustness of the rank order of different policy options as a final aid to the making of a consensus decision.

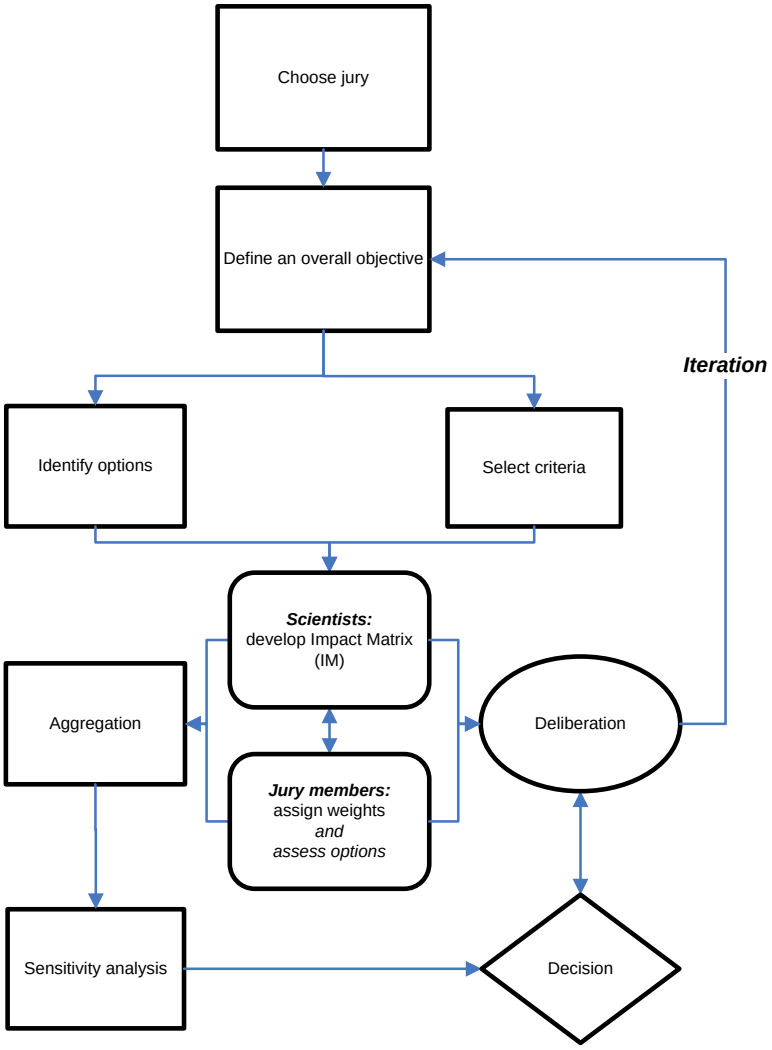

Figure 2. Flowchart of DMCE procedure (adapted from Proctor and Drechsler (Proctor and Drechsler 2003)).

3.2.4. Communicating uncertainty in biological invasion decision making with Deliberative Multi-Criteria Evaluation

3.2.4.1 Uncertainty in biological invasions

Biological invasions are notoriously difficult to predict (Williamson 1999). For most species, we currently have very limited knowledge regarding whether a species will establish in a new environment and the impacts that it might cause (Simberloff 2006).

Although the work on identifying future invaders and predicting their likely sites of invasion are of immense scientific and practical interest, such efforts have often been inconclusive (Mack *et al.* 2000). There are no universally reliable procedures for identifying the invasive potential of an organism. Stochastic effects and their spatial distribution co-determine whether a species becomes invasive (Pyšek and Richardson 2010). An EPP could remain

innocuous in its new environment for decades or longer, then undergo a rapid population explosion to become a raging pest[6] (Groves 2006). On the other hand, occasionally populations of established EPP could undergo a spontaneous decline, sometimes all the way to local extinction (Simberloff and Gibbons 2004).

EPP impacts are also idiosyncratic and often unpredictable (Mack, Simberloff *et al.* 2000). The same species may causes quite different impacts on ecosystem processes at different sites or at different times (Ehrenfeld 2010). For an EPP that is established in a new environment, our ability to estimate their impacts in different dimensions also varies. Economic (e.g. on agriculture) and social (e.g. on health) impacts are relatively easier to assess and quantify because they are more easily perceived and are immediately reported by stakeholders (Vila *et al.* 2010). In contrast, the severe level of uncertainty in estimating environmental impact results from the long-term and large-scale nature (Strayer 2009; Strayer, Eviner *et al.* 2006).

Biological invasion poses a serious challenge to risk analysts (Simberloff and Alexander 1998). Risk assessment of biological invasions requires consideration of the probability of each step in the invasion process, including entry, establishment, spread, and impact creation (Biosecurity Australia 2006; Cook *et al.* 2007b). For many organisms, we know next to nothing to quantify these steps. Even in strictly controlled experimental conditions, endogenously generated variance in spread rate could be remarkably high, which indicates inherent limits to predictability (Melbourne and Hastings 2009). It is not difficult to understand why little effort has historically been aimed at quantifying biological invasions in risk assessment (Andersen *et al.* 2004; Bossenbroek *et al.* 2005). To date, most risk assessment protocols, such as the widely adopted weed risk assessment in Australia (Gordon *et al.* 2008; Pheloung *et al.* 1999), are based on expert opinion and qualitative assessment, and not on rigorously quantitative statistics[7].

There is no doubt that great progress has been made on developing risk assessment for managing invasive species (Crowl *et al.* 2008; Pyšek and Richardson 2010). Due to data limitations, however, improved techniques *alone* will not necessarily enhance predictability. Only a small proportion of introduced species become invaders (Pyšek and Richardson 2010). The chance for an imported plant becoming a weed in Australia, for instance, ranges from 0.007 per cent to 17 per cent, with a central tendency of two per cent (Smith *et al.* 1999). This low probability means there are relatively few data points with which to study biological invasions and any existing information may not be representative (Franklin, Sisson *et al.* 2008). Additionally, most researchers work on invasive species with imminent or realized impacts because of funding availability (Pysek *et al.* 2008).

Prudent decision-making requires tools that are explicit about uncertainty and management options that are both precautionary and adaptive (Doak *et al.* 2008). Yet such a recommended strategy is hardly the norm in today's practice (Simberloff 2005). A common feature of many risk assessment models is that computation of risk probabilities are carried out *without* an uncertainty analysis (Benke *et al.* 2011). We believe the key to a solution is a new decision-making model that explicitly takes into account the uncertainty associated with the results in EPP risk assessment.

[6] The phenomenon might be explained by ongoing propagule pressure, which aids an established EPP to spread by introducing genetic variation adaptive for new habitats (Simberloff 2009).

[7] Quantative approaches for EPP risk assessment do exist (Kolar and Lodge 2002), but they are exceptions rather than a norm.

3.2.4.2. DMCE as a platform to communicate profound uncertainty

One of the most important explanations for the gap between science and policy is scientific uncertainty: scientists are familiar with uncertainty, yet the public and policymakers often accept scientific projections as certain. A management decision that assumes risk assessment results are certain, when in fact they are not, can result in unexpected or undesirable outcomes (Peterson *et al.* 2003). In fact, the consideration of uncertainty may lead to a different decision in managing environmental risks (Burgman *et al.* 1999; Regan *et al.* 2005). Horan *et al.* (2002), for instance, argue that decision models based on standard economic theory have limited value when neither the range of potential impacts nor the possibility of these impacts is known for EPP management. They develop a model where policymakers cease maximizing their utility and became uncertainty-averse instead. As a result, it becomes optimal to devote more resources to confronting high-impact events even if the probability is considered low.

Environmental policy is believed to be most effective if scientific uncertainty is incorporated into a rigorous framework as information for hypothesis building, experimentation, and decision making (Bradshaw and Borchers 2000). The frequently high level of uncertainty associated with biological invasions suggests that any quantitative model should be treated skeptically, and methods of communicating uncertainty should be applied (Franklin, Sisson *et al.* 2008). DMCE is a technique that could be used to reduce decision-makers' and stakeholders' level of discomfort with uncertainty.

An advantage of DMCE is that the deliberation process offers a unique opportunity for risk communication, the process that supplies lay people with the information they need to make informed, independent judgments about risks (Morgan *et al.* 1992). During deliberation, discussions could be geared towards what is known and what is not known, particularly the assumptions framing and embedded in the scientific knowledge of EPP risk assessment. This is because not only the quality of information built into the risk assessments is very important, the ability of stakeholders and decision-makers to interpret and use this information is also critical (Gregory, Failing *et al.* 2006).

In addition to the uncertainty resulting from knowledge gaps (termed 'epistemic uncertainty'), uncertainty also arises from under-specific, ambiguous, and vague use of our natural language (termed 'linguistic uncertainty') (Regan *et al.* 2002). Though often overlooked in risk management, this latter type of uncertainty may be particularly pervasive in language-based settings where the same term is interpreted differently by participants, resulting in misunderstanding and arbitrary disagreement (Carey and Burgman 2008; Webb and Raffaelli 2008). One familiar example is the potentially confusing set of terms developed around biological invasions (Lodge *et al.* 2006) (e.g. exotic, alien, and invasive). Effective communication can prevent needless misunderstandings amongst jury members, so that they can focus discourse on the most critical information concerning risk (Fischhoff 1995). The DMCE approach helps alleviate the negative impacts of linguistic uncertainty (Liu, Proctor *et al.* 2010).

3.2.5. Case studies of applying the DMCE in managing EPP risks

Following Maguire (2004), we classify EPP risk management decisions into two categories: (1) decisions about potential EPP before they arrive in a certain country or region, and (2) decisions about response actions to EPPs after they have arrived. In short, EPP risk management could be either pre-border or post-border. We provide a published case study for each of these situations (Cook and Proctor 2007; Liu, Proctor *et al.* 2010). The focus of the pre-border study is to use the DMCE as a decision-aid to analyze complex social values,

and that of the post-border study is to use the DMCE as a platform to communicate uncertainty.

3.2.5.1. DMCE-facilitated decision-making on pre-border prioritization

The application of DMCE in EPP prioritization was first explored in a workshop in Perth, Western Australia (WA) in November 2005 (Cook and Proctor 2007). Decision-makers were asked to establish ten priority species with a wide variety of impacts, ranging from species that are predominantly of agricultural significance, to those with substantial environmental or social implications. The decision-making group comprised representatives from government, industry, and community groups that might be affected in the event of an EPP incursion.

During the DMCE workshop, the participants were asked to indicate the relative importance of each criterion in comparison to other criteria in a set (Figure 3). They each distributed 100 points among the 10 criteria, and the same weighting process was carried out twice in total. Between the two rounds, the DMCE process involved asking participants to try to reach a consensus on criteria weights in an effort to reduce ranking variation and more clearly identify priority species. Those criteria for which weights differed most significantly were discussed first, with jury members who had expressed the most extreme maximum and minimum weights for each criterion asked to defend their choices. During this review process, jurors could reflect on their choices and those of other jury members and adjust their weights if they felt it was necessary. This revision process continued until participants were no longer willing to adjust their weightings.

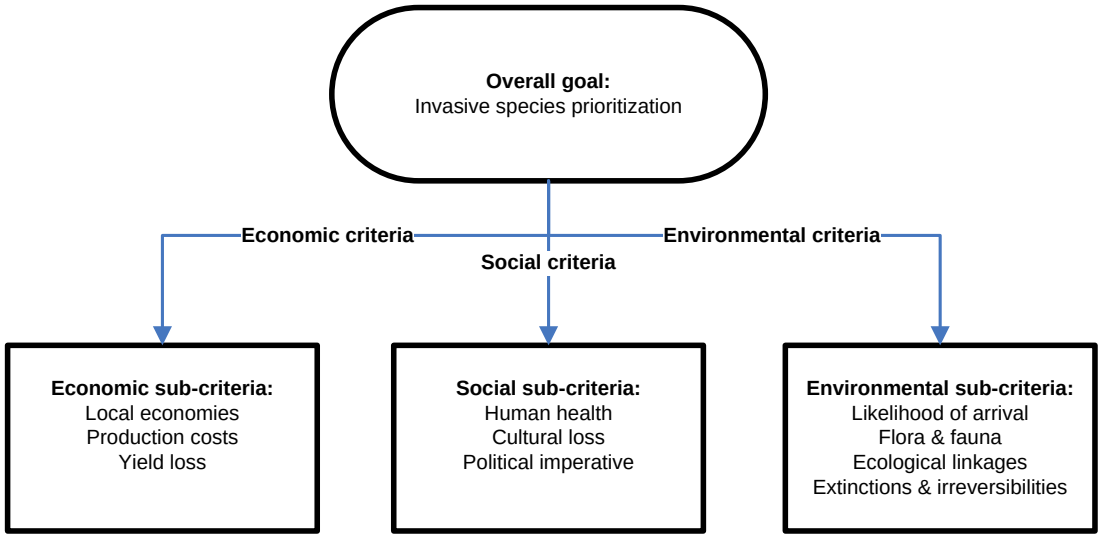

Figure 3. The set of 10 criteria used in the pre-border DMCE study (Cook and Proctor 2007).

The result of the round one weighting showed that juror opinions of criteria importance differ considerably, particularly in relation to production costs, yield loss, human health, local economies, and extinctions and irreversibilities. Some disagreement over the criteria weights was resolved though deliberation, including likelihood of arrival, human health, local

economies, extinction and irreversibilities. Although these changes were relatively minor, the discussion generated in the deliberation was revealing and informative to many of the jurors.

Based on the result of round two weighting, the prioritization results showed species of high environmental and social significance, such as guava rust (*Puccinia psidii*), which was absent from Australia, and red imported fire ant (*Solenopsis invicta*), which was present in only a small area were ranked higher than those of a predominately agricultural significance. At the time of the workshop, however, there was little importance assigned to or funding allocated to either of these species in Australia. By comparison, better known pest species such as the Queensland fruit fly (*Bactrocera tryoni*) have traditionally attracted more attention, reflecting their potential high impact on horticultural industries. This difference suggested that the way in which funds are allocated might need to be reconsidered.

In addition to the lessons learned regarding the current methods of allocating funding, this trial case also demonstrated that much more time and effort was needed to come to terms with some of the crucial trade-offs involved in certain management procedures, collect more detailed data relevant to the concerns of the decision makers and for this information to be digested by participants as well as to provide a truly iterative procedure as more information became available and more discussions and deliberations performed. Ideally, the process would be run over many months with workshops being held during this time at regular intervals. As a result of this trial study, the Australian corporate research centre of national plant biosecurity, Horticulture Australia Ltd. And the Rural Industrial Research and Development Corporation have initiated a joint project designed to further explore the role of DMCE in resource allocation decisions.

3.3.5.2. DMCE-facilitated decision-making on post-border response actions

There are few studies that have evaluated the risks associated with different management policies in response to invasions. Without this information, policy-makers cannot make informed decisions about how best to manage incursions, which can lead to the EPP being given a lower priority than other concerns (Bossenbroek, McNulty *et al.* 2005). To address this lack of knowledge, a DMCE was conducted with an overall goal of choosing among three regulatory actions for managing European House Borer (*Hylotrupes bajulus* Linnaeus) (Liu, Proctor *et al.* 2010), 'one of the world's most destructive pests of seasoned softwood timber' (Australian Department of Agriculture 2005).

A high level of uncertainty exists in terms of how fast the *H*. *bajulus* could spread and even whether the Borer is able to survive in roofing timbers in summer. Following Regan *et al.* (2002), Liu *et al.* (2010) distinguish between epistemic and linguistic uncertainty. In the *H*. bajulus case, Liu *et al.* (2010) *preserved* and explicitly accounted for epistemic uncertainty with a fuzzy set approach. At the same time, they attempted to *eliminate* linguistic uncertainty, to ensure any change in preference was not the result of people using words differently to each other or inexactly.

Conventional (i.e. non-fuzzy) MCDA approaches typically assume that all information can be expressed as accurate values. This assumption is often not met in the real world where imprecise and vague information regarding our knowledge of the state of a system or human preferences in making trade-off decisions can only be represented qualitatively, and in this case application of the fuzzy set approach is justified. This approach can incorporate uncertainty in both the impact scores (i.e. value of each criterion for a particular management option, often provided by experts) and criteria weights (i.e. preferences about relative importance of each criterion, provided by stakeholders in a DMCE process), and in

the Liu *et al.* (2010) study, the uncertainties in these two dimensions were explicitly addressed using the fuzzy set method.

In order to eliminate the linguistic uncertainty, the deliberation after the first round of weighting was dedicated to IM ratification. This experience revealed how divergence in preferences could be caused by factors other than preference differences *per se*. For instance, the jury realized that doing nothing to manage EHB could mean either 'leave it completely alone' or 'eradication only, without forcing the industry to carry out any timber treatment'. The differences in understanding towards this management option led to differences in the weights assigned to the sub-criteria of 'administrative cost' in round one.

In total, three rounds of weighting were conducted to elicit both the jury's initial preferences and the preference changes that occurred after IM ratification and further deliberation rounds. Figure 4 showed the extent of weightings changes by round across the sub-criteria. These are expressed in percentage form, and individual criteria are grouped together along the horizontal axis.

The IM ratification process between round one and two triggered changes in both IM and criteria weights, and the combined effect led to a change in the group's preference ranking for the three management options. Alterations were certainly made to criteria weights between rounds two and three (Figure 4), but these changes alone were not sufficient to produce a shift in the ranking of management options.

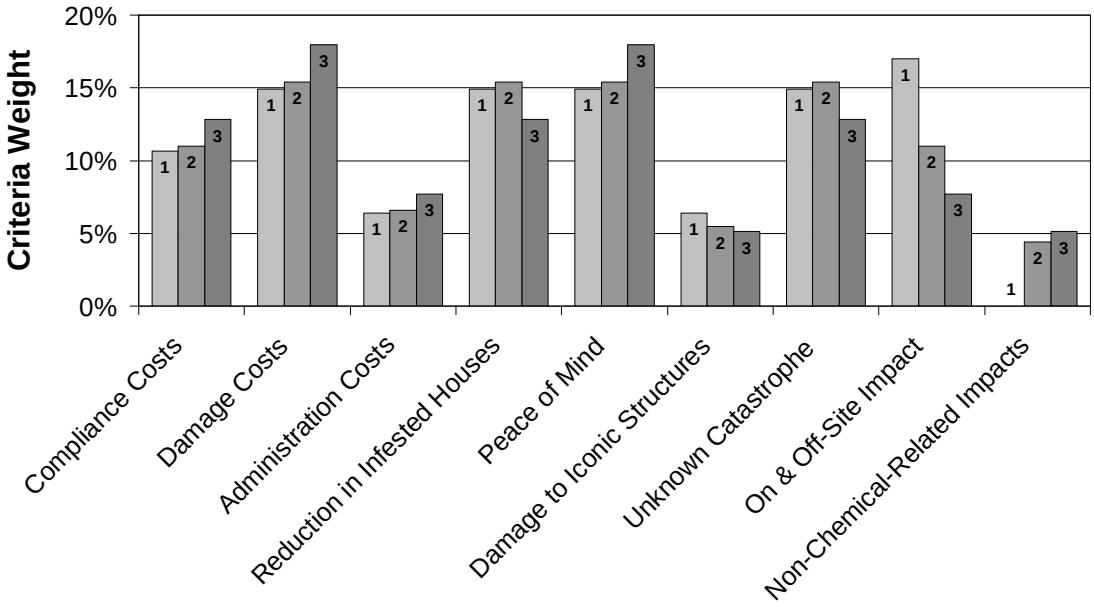

Figure 4. Change in the jury's sub-criteria weights by round in the post-border DMCE study (Liu *et al.* 2010).

The most important lesson learned from the post-border study was the potentially critical role of linguistic uncertainty in EPP risk management; a change in preference could result from the difference in people's understanding about the same terminology rather than their preferences *per se*. Resolving the linguistic disagreement is an important step, yet it has

received little attention in the literature (Carey and Burgman 2008). As our case shows, the DMCE offers a great opportunity to detect and eliminate linguistic uncertainty via group discussion and social learning. On the other hand, the fuzzy set approach may compound different types of uncertainties and introduce under-specificity, although it is more direct and intuitive compared to the probabilistic approach. How to communicate uncertainty effectively in a process of group decision-making warrants further investigation.

3.2.6. Summary

System-based approaches for managing risks pose a significant challenge. As Haimes reiterates, "to the extent that risk analysis is precise and simple, it is not real. To the extent that risk analysis is real and complex, it is not precise (Haimes 2009)." However, public officials and community stakeholders charged with the responsibility of making EPP risk management decisions on a regular basis do not necessarily share this view. Even in the age of post-normal science (Funtowicz and Ravetz 1993), we often hear demands for 'value-free' analyses and see probability-based estimates of incursion risk without sufficient discussion of true uncertainty. As a result, diverse social values are banished, and wide margins of error in risk assessment are neglected. This conventional model leaves EPP risk decisions to be made in the wake of political pressure and the crisis atmosphere of incursion (see Mackenzie and Larson 2010 for an example).

As a new decision-aid tool, DMCE injects scientific rigor and transparency in the decision-making process by providing an analytical structure for social complexity and by providing a platform for risk communication in which scientists, stakeholders and decision-makers can interact and discuss the uncertainty associated with biological invasions. It has been argued, that people tend to rely on a limited number of 'heuristic principles' to help them simplify the process of judgment (Kahneman and Knetsch 1992). Without the help of an analytical tool, decision-making tends to suffer from problems such as omitting important criteria and fixing opinions based on insufficient information. Based on the principles of multi-attribute utility theory (Keeney and Raiffa 1993), DMCE solves these problems by formally structuring a decision in terms of multiple criteria and policy options (Lahdelma *et al.* 2000; Gregory and Failing 2003; Failing *et al.* 2007; Gregory and Long 2009). The integration of risk assessment and risk communication has multiple benefits, such as increasing the policy relevance of risk assessment, gathering more diverse and context-specific bodies of local knowledge from stakeholders, exposing and debating the conditional social assumptions embedded in the scientific knowledge (Stirling 2006), and providing an opportunity to proactively prepare the ground for policy changes (Penning-Rowsell *et al.* 2006). A decision based on such an integrated process will gain more public trust and credibility (Fischhoff 1995).

By no means do we wish to promote the DMCE technique as a panacea. There are a number of challenging issues to address when applying the DMCE in decision-facilitation for EPP risk. These include how a jury should be chosen, which can directly affect decision outcomes (Cook and Proctor, 2007). It may be argued that information based on a DMCE should not be used as the only source of preference information because it will inevitably represent the voice of more active and opinionated jury members (Lahelma *et al.* 2000). In addition, a jury member unfamiliar with the deliberative process may encounter difficulty in participating and interacting with experts (Renn 2003), while a jury member familiar with the process may be prone to strategic misrepresentation of preferences. As in the case of valuation exercises of environmental economics, the DMCE process is also subject to the perils of information bias and 'groupthink' (Ajzen *et al.* 1996; Janis 1982). Recent progress in psychological and behavioral research can shed light on solving these issues (Carlsson 2010; Kerr and Tindale 2004). Last, stakeholder involvement requires investment in extra time, but

this may not always be feasible at the crisis atmosphere of incursions and there is a need to develop rapid participatory methods (Mackenzie and Larson 2010).

We also have no intention to propose replacing technical tools such as risk assessment and cost-benefit analysis with DMCE. On the contrary, we believe these tools could be integrated into the DMCE framework. For example, a cost-benefit-ratio may be used as one of the criteria regarding the desirability of different policy options for EPP management. We do argue that technical tools, as powerful as they can be, cannot *completely* solve environmental problems. This is because environmental decisions are 'political' as well as scientific and resolving environmental problems requires addressing the values of the public (Beierle 2002; Sarewitz 2004). We believe this statement is particularly true when there is profound uncertainty in our scientific understanding. "What to do in the face of uncertainty is a policy question, not a scientific question (Goldston 2008)."

Under this new model of DMCE-facilitated EPP risk management, scholars of biological invasion and risk analysts take the role of integrating their research results into the decision-making process. They fulfill this role by providing expert testimony in the DMCE process and by communicating not only their research but also the uncertainty associated with their results to the decision-makers. Essentially, this new decision-making model fits into a more democratic paradigm that conceptualizes scientists as part of society, working with others to solve problems together (Larson 2007; Norton 1998; Pielke 2007; Robertson and Hull 2003). At the same time, the DMCE offers scientists an interactive platform where their work will be critically discussed and clearly interpreted to the end-users.

We put forward DMCE as a promising model for managing risks in the face of complex social values and profound uncertainty. In this paper, we have focused primarily on the uses of DMCE for the risk management of biological invasions. But the same technique can be used in other environmental risk management decision-making contexts, particularly when those risks have low-probability, high novelty, and high impacts (e.g. flood, earthquake, infectious diseases, and abandoned hazardous waste dump). Applied over time, we believe the methodology will be able to trigger active adaptive management, as it offers an opportunity for deliberative and transparent decision-making based on social learning (Cook *et al.* 2010c; Penning-Rowsell, Johnson *et al.* 2006; Shea, Possingham *et al.* 2002).

3.3. A spatial EPP incursion simulation model

3.3.1. Introduction

Loss of area freedom from plant pests and diseases can potentially have serious implications for plant industries. Industries established around host crops can be affected as a result of EPPs (1) directly decreasing production and/or killing plant hosts, (2) increasing management costs, thereby decreasing farmer revenue, (3) restricting market access by closing domestic and export market routes to regions that are free from these pests.

This issue is particularly important for Australia, where many of the competitive advantages enjoyed by its plant industries reside in the country's pest free status. This allows production and export to national and international markets at relatively low costs. However, in an increasingly interconnected world where goods and people can cross continents in a matter of hours, incursions of agricultural pests and disease pose a serious threat.

In this context, it is particularly important for plant protection agencies to be able to quantify the economic impact associated with the potential incursion of a particular pest or pathogen. It is also critical for them to identify when and where to apply eradication or management strategies if a pathogen does make its way into the country. However, the cost of eradication activities can quickly become high, both for the affected industry and for government agencies in charge incident management. The success of eradication campaigns may also be limited and containment measures may fail. It is therefore essential for biosecurity incursion managers to be able to make an informed decision based on technical feasibility and cost effectiveness when deciding to eradicate, contain or live with a particular EPP.

This section describes the development of an interactive, spatially explicit, bio-economic EPP incursion management model designed to: (1) simulate the spread of an EPP across a landscape and help affected parties from industry and government better understand the complexity and dynamics of spread, as well as the potential economic impact on host industries at the landscape scale; (2) engage with biosecurity managers and assist them to interactively test management strategies or current contingency plans and assess the economic viability of eradication or management measures via the exploration of a range of incursion scenarios. The underlying assumption for the development of this model was that its use, through the equivalent of 'live fire exercises', may promote a better understanding of EPP spread and behaviour across particular landscapes and ultimately facilitate enhanced biosecurity preparedness.

The model outlined in the following sub-sections is described using a topical case study. We use the model to explore the challenges facing stakeholders and risk managers in the event that fire blight (*Erwinia amylovora*) was found to be present in the Goulburn Valley region in Victoria, Australia.

3.3.2. The fire-blight model

A detailed description of fire blight disease, caused by the bacterium *E. amylovora* , appears in both Appendix 1 and section 3.4.2.1, but we provide a brief summary here with sufficient detail to clearly demonstrate the application of the spatial modelling techniques outlined in this section. *E. amylovora* principally affects plants of the *Rosaceae* family (CABI and EPPO 1997). It can cause considerable damage by strongly decreasing yield (in extreme cases nullifying them) and also damaging or killing the host tree. It is of particular economic significance for the apple (*Malus domestica*) and pear (*Pyrus communis*) orchard industries.

The disease can be so destructive that it has led to the abandon of pear cultivation in some States of the USA (CABI and EPPO 1997).

Native to North America, *E. amylovora* is now found across South America, Europe, the Middle East and New Zealand (Merriman 2002). Although an isolated case was previously reported in the Royal Botanic Gardens of Melbourne in 1997 (Rodoni *et al.* 1999), it was successfully eradicated and Australia is still considered free of this pathogen, where it listed as a high priority quarantine pest.

Fire blight development is favoured at temperatures between 21 and 28°C combined with high humidity and rainfall (Merriman 2002). Typical symptoms are bent, withered young shoots (referred to as 'shepherds crooks'), brown to black blight of flower clusters, shoot and leaves, as well as sunken cankers on branches from which bacterial ooze is sometimes present (Merriman 2002). Overwintering in infected host plants, *E. amylovora* is spread by splash dispersal in droplets of rain or irrigation water over short distances and in the spring by insects that carry bacteria from blossom to blossom across longer distances. Honey bees, in particular, have been reported to transport bacteria over several kilometres (Merriman 2002). Migrating birds have also been considered to be able to carry the bacterium over longer distances (CABI and EPPO 1997).

Fire blight is considered a very serious threat in Australia. Merriman (2002) estimated its introduction to Victoria's Goulburn Valley could cause over $20 million damage to the apple and pear industry. Pear producers are likely to suffer the most significant losses, with approximately 80 per cent of the nation's pear production taking place in this region.

Several bio-economic models simulating the economic consequences of pathogen incursions in Australia have already been developed for several plant industries (Cook *et al.* 2011a; Cook *et al.* 2007a; Liu *et al.* 2011). These models incorporated detailed life history characteristics of pathogens and their hosts, as well as the effect and costs of management strategies, but they were inherently non-spatial. They provided summary values at the country scale but were limited by their inability to take into consideration spatially explicit processes such as the dispersal pattern of exotic pathogens across a landscape of orchards and the likely implementation of management actions at a local level.

The objective of our research was to enhance the performance of process- based models through the development of a generic spatially explicit and interactive decision facilitation tool. During facilitated meetings, this tool would allow stakeholders to explore various scenarios of incursion at the landscape scale and estimate the management costs and effectiveness of different management scenarios.

Using a maps-based interface, our aim was to allow stakeholders to control where, when and what level of management they could apply to control simulated incursions in the landscape. The tool would also allow stakeholders to visualise the outcomes of the simulated management scenario in an environment easy to explore and understand.

Specifically, the features required for the development of our decision facilitation tool were:

1. That it possessed basic functionalities to read-in, display and export data in a spatial format.
2. That it modelled dynamic biological and economical processes in a spatially explicit manner.
3. That it simulated disease dispersal across landscapes.

4. That it be interactive to allow 'on the fly' strategy development through the dynamic specifications of parameters such as origin of disease outbreaks, dispersal distance of disease vectors and the choice of management strategies.

3.3.3. Choice of a modelling environment

To identify the most suitable platform for the development of our spatially explicit model, we scoped the potential of four modelling environments based on findings of Methodological Review of section 3.1. These were:

a. A combination of STELLA and the Spatial Modelling Environment (Maxwell and Costanza 1997), as described by Aurambout *et al.* (2009) and Bendor *et al.* (Aurambout *et al.* 2009; BenDor *et al.* 2006)
b. STELLA 9.1.4 Spatial map (see Chichakly (2009))
c. NetLogo (Wilensky 1999), and
d. Modular Dispersal in GIS (MDig) (see Pitt *et al.* 2009b).

The selection of software was chosen based on capacity to provide the requested features. The outcomes of this model comparison are presented in Table 3.

Table 3. Functionality comparison across modelling platforms.

Platform	**Criteria 1** Possesses basic functionalities to read-in, display and export data in a spatial format.	**Criteria 2** Models dynamic biological and economical processes in a spatially explicit manner.	**Criteria 3** Simulates disease dispersal across landscapes.	**Criteria 4** Allows interactive model steering in "war game" strategy development.
SME/STELLA combination	Yes	Yes	Yes* long distance dispersal problematic	No
STELLA Spatial map	No	Yes	Yes* very difficult to implement	No
NetLogo	Yes	Yes	Yes	Yes
MDig	Yes	Yes* Addition of custom equation not tested.	Yes	No

This rapid comparison led to the selection of NetLogo as the preferred environment for the development of our bio-economic *E. amylovora* model.

Our choice of NetLogo 5.0 over other agent based modelling (ABM) environments such as MASON, Swarm or Repast is supported by several agent based model reviews (Berryman 2008; Castle 2006; Railsback *et al.* 2006), where NetLogo was consistently identified as the most appropriate for the circumstances like those faced in the CUBA project. Net Logo's 'patches' and 'turtles' structure was particularly suited to our problem, which required us to be able to simulate bee movements through apple and pear orchards. Its lack of network

causality, as identified by Berryman (2008) was of no consequence as we only made use of very simple agent behaviours.

Net Logo's ease of use and capacity to automatically display agents and allow dynamic inputs of parameters during model runs was essential to our goal, as the model was to be used in stakeholder workshops and run in real time in what-if scenarios defined by the workshop audience. The availability of GIS extensions (not present in 2006 during Castle (2006) study) to load and export spatial data also allowed us to make use of raster datasets and display the spread of *E. amylovora* across a real existing landscape. This was particularly important in that it provided a valuable sense of realism for stakeholders.

3.3.4. Structure of the developed model

The interactive decision facilitation tool simulates biological and economical processes associated with the spread, management and impact of *E. amylovora* incursions in apple and pear orchards in a region of the Goulburn Valley consisting of about 6,000 hectares of orchard area (SPC cannery data). The model operates on a weekly time step and takes into consideration both local (i.e. occurring in a specific geographical patch) and spatially dynamic processes (i.e. dispersal). Local processes are modelled for a square patch of orchard habitat (i.e. not for individual trees) of a size chosen by the model user.

The processes simulated at a patch level for apple and pear orchards include:

- Tree phenology: via the occurrence of flowering
- Tree growth: tree maturity and fruit production potential
- Infection: the presence, detection and impact of *E. amylovora* on fruit yield
- Cost calculation: based on the incursion management strategy chosen and the financial impact of the disease.

The process of spread of *E. amylovora* between orchard patches, vectored by pollinating bees, is simulated by the use of agents referred to as turtles or breeds in NetLogo. These dispersing agents are 'sprouted' from infected orchard patches during the flowering season and stochastically dispersed to neighbouring orchard patches. Orchard patches reached by these dispersing agents become infected.

Processes related to the management of simulated incursions can be selected by users (i.e. creation of a quarantine zone, eradication or live with it strategies) and activated either manually or upon detection of the disease in infected orchard patches.

All values used for the model variables or parameters and assumptions are theoretical values selected from published literature or expert information and represent averages.

3.3.4.1. State variables and scales

Patch-related variables are variables that are calculated for all or a subset of specific patches. A list and description of these variables is provided in Table 4.

Table 4. Patch specific variables.

Variable	Description
grid-value	Integer value read from the input raster dataset.
Tree_Species	String: "apple", "pear" or ""
Tree_Maturity	Integer number corresponding to the age of the apple or pear tree (in year) if they are present
Tree_potential_Yield	Net revenue value calculated as a function of the orchard age and its maximum yield. In $ per hectare
Tree_potential_Yield _FB	Net revenue value for a tree infected with *E. amylovora*. Value calculated as a function of the orchard age and its maximum yield ($/ha)
Flowering	0 or 1 value to specify whether flowering is occurring or not
Pruning	0 or 1 value to specify whether pruning is occurring or not
Infection_Status	0 or 1 value to the patch if infected by *E. amylovora* or not.
Detection_Status	0 or 1 value to specify whether the *E. amylovora* infection has been detected or not.
Degree_of_Infection	Integer identifying the number of weeks since infection occurred.
Quarantine_Status	0 or 1 value to specify whether the patch is in a quarantine zone or not.
Detection-probability	Weekly probability to detect a infection
Time_since_detection	Integer accumulating the number of weeks since detection occurred
Time_since_quarantine	Number of weeks since the patch entered the quarantine area
Cost_to_patch	Cost of management activities for the patch.
Quarantine_cost_to_patch	Cost associated with quarantine activities for the patch
Maturity_initialise	Random value between 0 and 100 used for the initiation of patch tree maturity.
Density	Density of planting: High or Low

Global variables are variables or constants with values independent of a particular agent or location and which can be read by any agent or patch in the model. A list and description of these variables is presented in Table 5, below.

Note that the *E. amylovora* model did not make use of agent (NetLogo breeds) specific variables since we only required their location in the modelled space.

Table 5. Global variables.

Variable	Description
raster-dataset	String identifying the location of the .asc file used in the map interface and to initialise orchard locations [constant].
Output	Name of the .asc output file created for each time step (facultative)
Week	Week counter (incremented every time step from 1 to 4)
Month	Month counter (incremented every 4 weeks from 1 to 12)
Year	Year counter (incremented every 12 months).
Region_Infection_Status	Value summarising the infection status across all orchard cells (0 no infection detected, 1 at least one infection detected).
Species_List_Flowering_season	List variable containing the following information [(1) Orchard type, [(2) flowering starting month, (3) flowering starting week (4) flowering end month, (5) flowering end week], [(6) pruning starting month, (7) pruning starting week (8) pruning end month, (9) pruning end week], (10) land-use code specific to the orchard type and (11) colour to be used to identify the type or orchard in the map interface.
Detection_values	List variable containing the following information [(1) probability of detection of *E. amylovora* during flowering, (2) probability to detect *E. amylovora* during shoot strike (3) probability to detect *E. amylovora* during pruning, and (4) probability to detect *E. amylovora* in a quarantine area] [constant].
Time_since_first_detection	Variable continually incremented, every time step, once a detection has occurred anywhere in the map of interest.
Weekly_quarantine_surveillance_cost	Coast applied to each cell in the quarantine area [constant].
Total_ORC_cost	Total cost paid to the farmer for the destruction of the trees on the patch.
Total_quarantine_cost	Total cost associated with the application of quarantine measures scaled for the patch.
Total_detections	Total number of patches where *E. amylovora* was detected
Total_destruction_area	Total area of patches destroyed (ha)
Total_cost	Sum of all ORC costs and quarantine costs across all cells.
Total_detections	Total number of cells where *E. amylovora* detection has occurred (incremental only).
Shoot_strike_detection_month	Month during which a typical shoot strike can be identified (assumed to be identical for both apple and pears) [constant].
I, j, x, y	Variables used to increment looping procedures.
Cell_size	Approximate surface area of an orchard patch (based on the land-use map) [constant]
Quarantine_radius	User defined radius used to define a circular quarantine zone around a selected patch
Bee_dispersal_radius	User defined value corresponding to the maximum dispersal distance a bee can reach in one flight from one tree to another.
Destruction_radius	User defined used to define an circular destruction zone around a selected patch.
Number_of_crops	Total number of orchard types considered (by default 2: apple and pears) [constant].
Economic_return_FB	Sum of the yield (growth margin) of all infected and non-infected orchard patches.
Economic return no FB	Sum of the yield (growth margin) of all orchard patches, assuming no patch is affected by *E. amylovora*. This corresponds to the maximum growth margin that could have been achieved in the absence of *E. amylovora*.
OCR list HD	List of variables corresponding to the owner reimbursement costs paid in compensation by the government for destruction of an apple or pear tree grown in a High density system (variable as a function of tree maturity) [constant] [(1) cost paid for a tree of 1 year old or less, (2) cost paid for a tree of 2 years old, (3) cost paid for a tree of 3 to 5 years old, (4) cost paid for a tree of 5 to 13 year old, (5) cost paid for a tree of more than 13 years old].
OCR list LD	List of variables corresponding to the owner reimbursement costs paid in compensation by the government for destruction of an apple or pear tree grown in a low density system (variable as a function of tree maturity) [constant] [(1) cost paid for a tree of less than 3 years old, (2) cost paid for a tree of 4 to 7 years old, (3) cost paid for a tree of 7 to 13 year old, (4) cost paid for a tree of more than 13 year old].
Tree per ha	Number of trees per ha based on patch tree density [constant]
Bees dispersing	Number of bees dispersing from an infected patch (calculated randomly based on the number of bees).
Number of bees	User defined maximum number of disease carrying bees capable of spreading to outside patches during a week at flowering
Bees dispersal distance	Distance used to disperse bees, calculated stochastically based on the bee dispersal radius variable.
Maximum_Yield_Apple	Maximum growth margin expected for a fully matured producing apple tree not affected by *E. amylovora* [constant].
Maximum_Yield_Apple_FB	Maximum growth margin expected for a fully matured producing apple tree affected by *E. amylovora* (taking into account the cost of extra pruning and anti biotic spray).
Maximum_Yield_Pear	Maximum growth margin expected for a fully matured producing pear tree not affected by *E. amylovora*.
Maximum_Yield_Pear_FB	Maximum growth margin expected for a fully matured producing pear tree affected by *E. amylovora* (taking into account the cost of extra pruning and anti biotic spray).
Total_revenue_loss	Cumulative sum of Economic_return_no_FB - Economic_return_FB
Degree_of_Infection_threshold	Length of time in weeks necessary for a newly infected orchard patch to become infective.
High density value	Array of the proportion of high density planting for Apple and Pear in percentage [constant] [(1) proportion of high density apple plantings, (2) proportion of high density planting for pear].
Productivity_loss_FB	Percentage value corresponding to the expected loss in gross margin associated with the presence of *E. amylovora*.

3.3.4.2. Process overview and scheduling

The *E. amylovora* model is composed of two main procedures: (1) an initialisation procedure which defines the initial conditions and constants used in the subsequent run and (2) a run or 'go' procedure run iteratively at each time step of the model simulation. Both initialisation and go procedures are controlled via a graphic user interface where model parameters can be changed and the disease spread can be viewed via a dynamic map interface.

Graphical user interface

The *E. amylovora* spatial decision facilitation tool was designed to allow the development of interactive 'war game' simulations of *E. amylovora* incursions. Its structure is composed of a map interface, a reporting section and a parameter/action section (Figure 5).

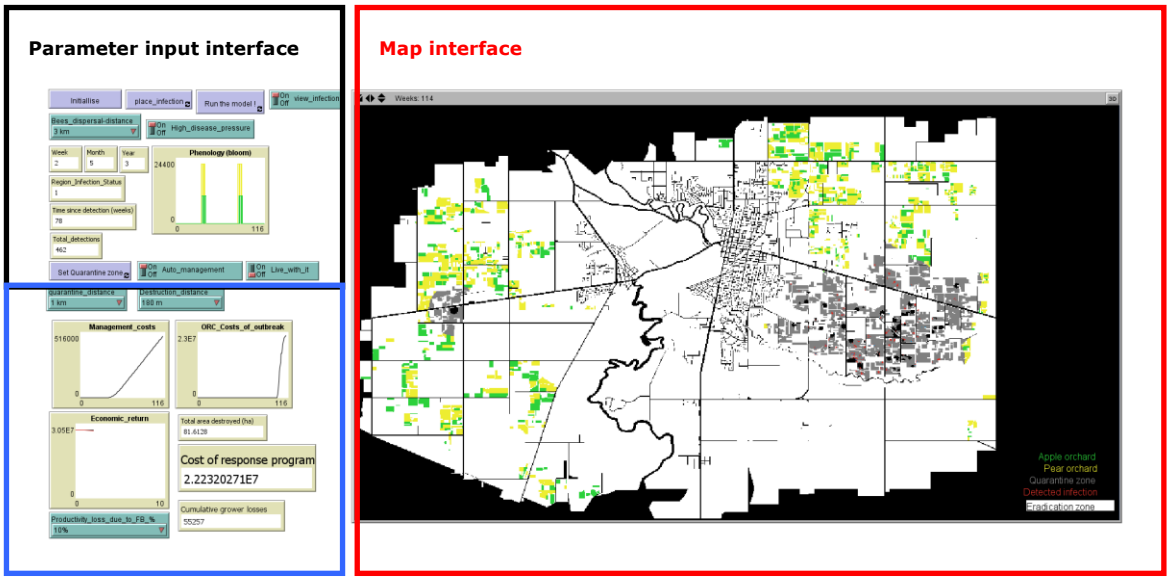

Figure 5. Interface of the spatial interactive decision facilitation tool.

The map interface allows users to visualise the selected area of interest by displaying a land-use map where orchards are highlighted over a white background. Apple orchards are displayed in green and pear orchards in yellow. Users can also directly interact with the spatial interface to initialise *E. amylovora* infections at specific locations or specify quarantine areas and destruction zones.

The parameter interface allows users to specify and change model parameters at any time during the course of a simulation, to define and apply management strategies and to pause or start model runs.

The reporting interface allows user to visualise temporal changes in variables of interest via graphic and numeric reporters. Finally, a data export option allows weekly map outputs to be saved in GIS format for later analysis.

Initialization procedure

The initialisation procedure is used prior to the run of simulations and performs the following actions:

(1) The initialisation and calculation of model 'constant' global variables based on the user defined model parameters (see **Error! Reference source not found.**).

(2) The definition of the map interface (i.e. NetLogo world) coordinate system and the attribution and display of tree species (i.e. apple, pear or 'empty') to each patch as a function of the selected land-use map.

(3) The attribution, for each apple or pear patch, of initial tree maturity (maturity initialise) and density based on the following conditional statements:

Statement 1

$$IF\ RD(100) \geq 20\ THEN\ Tm = 5 + RD(7)\ ELSE$$

$$IF\ RD(100) < 5\ THEM\ Tm = 1\ ELSE$$

$$IF\ RD(100) < 10\ THEN\ Tm = 2\ ELSE$$

$$IF\ RD(100) < 15\ THEN\ Tm = 3 + RD(3) ELSE$$

$$IF\ RD(100) < 20\ THEN\ Tm = 13 + RD(3)$$

Where RD(X) is a random number between zero and X generated once per iteration for each patch, Tm is Tree maturity. The use of this equation assured that 80 per cent of the orchard patches were initiated at a maturity of five to 12 years old (corresponding to fully productive trees) while the remaining 20 per cent were uniformly spread across the ages of one, two, three to five and 13 to 15 years old. These proportions are in accordance with that maintained in production apple and pear orchards.

Statement 2

$$IF\ RD(100) \leq HDV\ THEN\ Density = \text{"high"}\ ELSE\ Density = \text{"}low\text{"}$$

Where HDV is the proportion of high density planting in the orchard region considered.

The initialisation procedure is followed by an 'orchard infection' step requiring user action. The model does not take into considerations processes that can lead to an initial *E. amylovora* infection in the region of interest, such as the movement of bee hives carrying the bacteria, or the planting of infected material or pruning with infected equipment. Rather, initial infections are started by a mouse-click action when the 'place_infection' button is activated. Orchard patches selected by the user from the map interface then become infected.

Go procedure

Upon specification by the user of an initial infection, the go procedure is executed iteratively until stopped by the user or once the simulation counter (tick) reaches a predefined value.

This procedure is composed of several sub-procedures for which detailed equations can be found in Appendix 2. For the purpose of clarity, this section only described the most important sub-procedures in the sequential order in which they are iterated in the model.

Distance conversion sub-procedure:

This procedure converts all 'distance derived' model parameters to an equivalent 'patch number' by dividing their value (in metre or kilometre) by the patch size.

Temporal increment sub-procedure:

This procedure increments and resets the week, month and year variables based on the following assumptions. A week is equivalent to one time step, a month is incremented every four weeks and a year is incremented every 12 months.

A similar procedure also increments, within each patch, the tree maturity variable every first week of January (i.e. adding to the initial value defined in the initialisation procedure). For previously infected patches, the 'degree of infection' is incremented every week.

Orchard flowering sub-procedure:

This procedure calculates, for each orchard type (apple or pear), the occurrence of flowering and pruning events (zero or one) as a function of the week and month variables based on the information on flowering and pruning starting and ending dates provided as part of the Species_List_Flowering_season input variable (see Table 5).

Yield calculation procedure:

This procedure, run on the first week of each year, calculates the potential yield (gross margin without *E. amylovora*) of an apple or pear patch as a function of tree maturity, tree density and the maximum potential yield of the orchard (defined by users for apple or pear). Commercial apple or pear trees typically produce little or no fruit before reaching three to four years of age, depending on the planting density system. From five to seven years until 13 years old, the trees are at their most productive. Production begins to decline from 14 years onwards at different rates, depending on the planting system. This yield cycle is captured in the model by **Error! Reference source not found.**, which in turn is described y the following conditional equations.

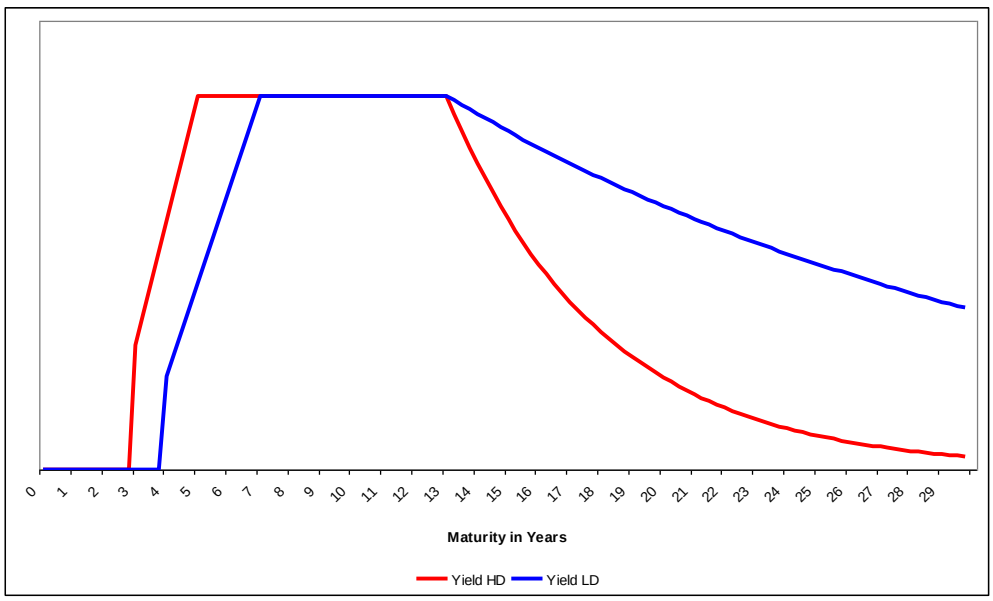

Figure 6. Yield calculation as a function of tree maturity and tree planting density (Low density LD and high density HD).

For high density plantations:

$$IF\ Mt < 3\ THEN\ \text{TpY}\ =\ 0\ \text{ELSE}$$

$$\text{IF } 3 \leq \text{Mt} \leq 5\ THEM\ TpY = \frac{MxY}{3} * Mt - (MxY * \frac{2}{3})\ ELSE$$

$$IF\ 5 < Mt \leq 13\ THEN\ TpY = MxY\ ELSE$$

$$TpY = MxY * e(-(Mt - 13)/5)$$

For low density plantations:

$$IF\ Mt < 4\ THEN\ TpY = 0\ ELSE$$

$$IF\quad 4 \leq Mt \leq 7\ THEN \frac{MxY}{4} * Mt - \left(MxY * \frac{3}{4}\right) ELSE$$

$$IF\ Mt \leq 13\ THEN\ TpY = MxY\ ELSE$$

$$TpY =\ \ MxY * e(-\frac{Mt - 13}{20})$$

Where TpY is the tree potential yield, Mt is the tree maturity of the patch and MxY is the maximum yield of a fully productive tree (with or without *E. amylovora* depending on the patch infection status).

For simplification purposes, we simulate the impact of *E. amylovora* on orchard yield as a fixed percentage loss, which can be adjusted by the user. It is acknowledged that this uniform percentage loss across all tree ages might be an underestimation if young trees are more severely affected by *E. amylovora* than mature trees.

Bees sprouting and dispersal sub-procedure:

Honey bees are one of the principal dispersal vectors of *E. amylovora*. Our model simulates the spread of *E. amylovora* from infected to non infected orchard patches via the use of bee agents (breeds). These bee agents represent insects that can successfully 'pick up' *E. amylovora* from infected blossoms and transport them outside their patch of origin and successfully infect a blossom in another orchard patch.

The bee agents are created or 'sprouted' from infected orchards at each time step during the flowering period. Each agent is then stochastically provided with a dispersal angle (from zero to 360°) and distance (from zero metres to the bee dispersal distance value provided by the user) and spread to neighbouring patches.

Upon 'landing' in a neighbouring patch the bee agent changes the status of the patch to infected if the patch is an orchard in flower, or takes no action if the patch is not an orchard or not in flower. The agent is then subsequently killed.

The rate at which *E. amylovora* can infect susceptible plants is highly dependent on climatic conditions. Wet warm springs allow the bacteria to develop quickly and produce a large amount of inoculums, while drier cooler conditions are less favourable for spread. We

incorporated a 'high disease pressure' trigger to account for effect of inter-annual variability on inoculum availability and bacterium development speed.

Under low disease pressure (switch off), bee agents are only sprouted from orchards which have been infected for more than four weeks and the number of bees sprouted is stochastically selected from zero to one fifth of the user defined number of bees.

Under high disease pressure (switch on), bees are sprouted from any infected orchards (to account for the faster development rate of inoculum) and the number of bees sprouted is stochastically selected from zero to the user defined number of bees (to account for the presence of more inoculum).

Our model can only accommodate an integer number of bee agents. To address cases where the number of bees dispersing would be less than one, we used the following conditional statement:

$$\text{IF NB} < 1 \; THEN$$

$$\text{IF RD}(100) \leq \text{BD} * 100 \text{ THEN Bsprt} = 1$$

$$\text{ELSE Bsprt} = \text{NB}$$

Where NB is the number of bees, RD(100) is a random number between zero and 100 generated for each patch and Bsprt is the number of bees being sprouted in an orchard patch.

Dispersal by rain sub-procedure:

This procedure runs during the flowering period, simulating short distance dispersal that can occur as a result of rain or hail events. It allows flowering orchard patches infected for more than 10 weeks to stochastically infect a directly adjacent flowering orchard patch.

Although pruning as a source of disease spread was not directly simulated by our model, it can be incorporated via the graphic user interface by manual placement of a new infection.

Infection detection sub-procedure:

E. amylovora infection in apple and pear produces symptoms that can be detected by orchardists, scouts or disease specialists during routine disease and pest monitoring activities or as part of infection quarantine measures. Disease detection in infected orchards is affected by the visibility of the symptoms and by the amount of time spent by orchardists 'looking for it'. Our model defines, for infected patches, three different *E. amylovora* identification probabilities, depending on the time of year: (i) at flowering, (ii) shoot strike and (iii) during the tree pruning period.

Shoot strike corresponds to the period (assumed to occur around November in Victoria for modelling purposes) when the typical fire-blight 'shepherds crooks' are most visible, but they can also be spotted at other times.

A fourth identification probability of 50 per cent per year is also attributed if the orchard patch under consideration is located within a quarantine zone (see disease management procedure).

Disease detection is calculated as a zero or one variable for each patch of infected but not yet detected orchard, based on the following conditional statement:

$$\text{IF RD}(100) < DP\ THEN\ DsT = 1$$

Where RD(100) is a random two digit float number between zero and 100 generated for each patch, DP is the *E. amylovora* detection probability based on the time of the year, and DsT is the patch detection status.

Disease management sub-procedure:

Two different mutually exclusive management strategies can be selected by the model user:
(i) Eradication and containment, or
(ii) Live with it.

In eradication and containment mode, the initial detection of infection in an orchard patch triggers two separate processes:

1. The creation of an 'eradication zone' around the detected infected patch with a radius equal to the user defined destruction distance variable, where all apple or pear trees are removed. This changes the status of the patch to 'empty'.
2. The establishment of a quarantine zone around the patch where infection was detected with a radius equal to the user defined quarantine distance variable. The establishment of the quarantine zone is followed by removal of bee hives (i.e. bee agents are stopped from sprouting from infected orchard patches) and by an enhanced surveillance intensity. This increases the probability of detecting infected patches within the quarantine zone.

Both eradication and quarantine zones are assumed to be established a week after infection detection (simulating the turn-around time for positive diagnosis from infected samples). Four weeks (or time steps) after the quarantine zone is applied, the probability of detection increases for that week and the first week of the first month thereafter. This simulates recurrent survey events.

We acknowledge that the revised contingency plan for an *E. amylovora* response specifies that the restricted area should be based on considerations such as terrain, orchard distribution, irrigation practices and wind patterns (Merriman 2002). However, for simplification and functionality, the quarantine areas in our model are defined as fixed diameter circles around detected infections.

In a 'live with it' scenario, no quarantine or eradication zones are applied around detected locations. If a quarantine zone was applied prior to the activation of the 'live with it' switch, it is removed, thus restoring bee movement.

Orchardists are assumed to focus their efforts on actively managing their orchards to control the disease by pruning infected plant limbs with *E. amylovora* cankers and protecting flowers from infection by spraying antibiotics during bloom. This assumes these products will be made available to Victorian orchardists if required. Such management activities are assumed to decrease inoculums by up to 80 percent and are incorporated into the model by decreasing the number of bee agents sprouted from infected patches by this proportion.

Management strategies and settings can be changed during the course of simulations, thereby providing users with the option to alter their strategy based on the observed effectiveness from the map interface.

Economic costs and losses calculation sub-procedure:

This procedure calculates the management costs and economic losses associated with *E. amylovora* infestations for the whole study area. Two types of costs are taken into consideration, depending on the type of management strategies selected by the user:

(i) The cost of eradication and containment response program – typically borne by government institutions, such as State government departments of agriculture, and

(ii) The cost to growers – linked to productivity loss and higher management costs.

The cost of the response program is calculated as the cumulative sum of (a) the Owner Reimbursement Costs (ORC) (i.e. the amount of money paid by the government to orchardists in compensation for the destruction of their trees), and (b) the cost associated with the establishment of quarantine zones. ORC costs are calculated once for each patch of orchard destroyed as a result of an eradication program. These costs, illustrated in Figure 7, vary as a function of the tree species, their age and planting density within each patch. The quarantine costs are calculated every time step for every patch located with a quarantine zone, and account for the cost of: (i) sending a team of expert pathologists to look for the disease, (ii) testing the samples in the laboratory to confirm the pathogen, (iii) removing bee hives, and (iv) managing the incident and engaging the public and the press. In Victoria, these costs are estimated to be approximately $6 per hectare per week (see the model application section for more detail). Loss of fruit yield due to the removal of bee hives was not taken into consideration.

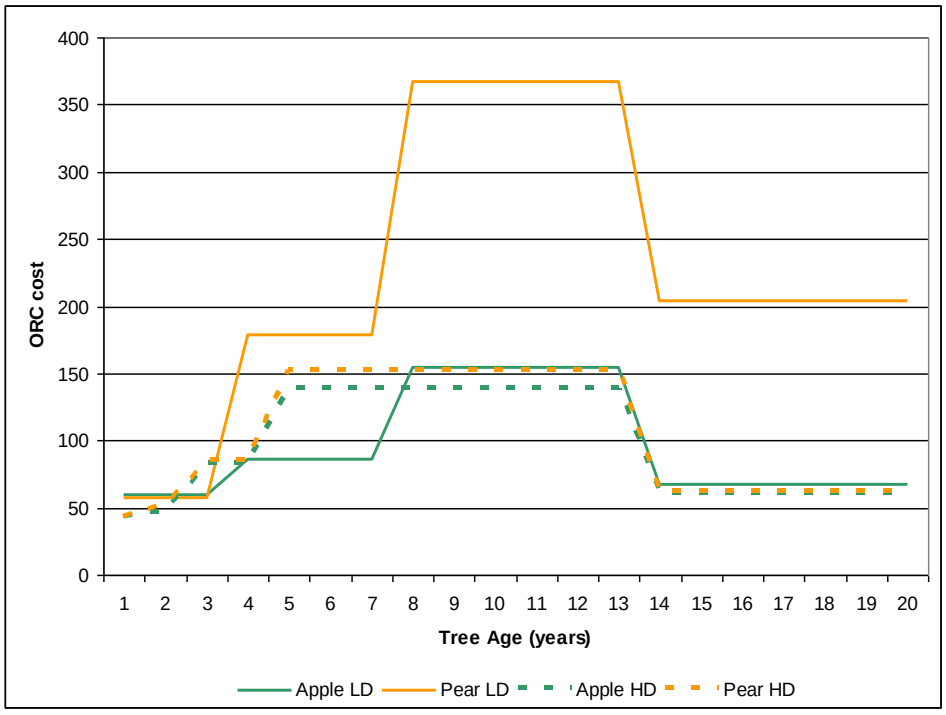

Figure 7. ORC costs as a function of tree age, species and planting density (Low density LD or high density HD).

The cumulated cost to growers represents the difference between the yield (growth margin) that orchardists could have obtained in the absence of *E. amylovora* and the yield they actually obtained with *E. amylovora*. It is calculated once annually for the study area based on the following formula:

$$CLG = \sum_{t=0}^{s} \left(\sum_{j=0}^{n} Y(j) - \sum_{k=0}^{m(t)} (Y(k) * Pl\_FB) \right)$$

Where CLG is the cumulative cost to growers; s is the number of time steps in the simulation, Y(j) is the yield (growth margin) for an orchard patch j, n is the total number of apple and pear orchard, m(t) is the number of orchards patches infected a time t, Y(k) is the yield for a *E. amylovora* infected orchard patch k and Pl_FB is the productivity loss associated with *E. amylovora*.

3.3.5. Example Model application

The *E. amylovora* model was designed to be flexible and applied to different geographical regions, and potentially different exotic diseases and pests. It allows users to investigate, through the user interface, a range of possible spread and management scenarios. The section below describes an example application of the *E. amylovora* model to the Goulburn valley in Victoria.

3.3.5.1. Study region

The Goulburn Valley contains approximately 6,000 hectares of apple and pear orchards and is responsible for 80 per cent of the Australian apple and pear production. We applied the *E. amylovora* management decision support system to a subset of this region, located in the vicinity of the town of Shepparton (illustrated in Figure 8) covering an area of 2,300 hectares of pear and 1,200 hectares of apple orchards.

The land-use data input for the model was derived from the Victorian Land Use Information System (VLUIS) (Morse-McNabb 2011) on which a more detailed apple and pear orchard layer was overlaid (obtained from Victorian Department of Primary Industries). This dataset did not contain information about the presence of potential alternative hosts of *E. amylovora*. Although it must be acknowledged that some alternative hosts undoubtedly exist in the region, no GIS data on their location could be sourced for this exercise.

The obtained vector dataset was converted to raster format using ESRI ArcGIS 10 and exported as an .asc file that can be read directly into NetLogo, using the GIS extension. The NetLogo GIS extension does not currently support projection systems specific to Australia or Victoria and we used the World Geodesic System 1984 coordinate system to create our .asc dataset.

The created dataset consisted of a grid of 926 × 499 cells of a resolution of 0.000325 degree. However, since our model makes use of metric units for the definition of dispersal distances and quarantine zones, we approximated our cell size to 32 metres (covering a total area of 1,024 square metres) for the study site. This approximation of geographic coordinates to a projected system necessarily causes a degree of inaccuracy. For our study site, we estimate our specification of cell size led to a five per cent underestimation of the actual orchard area present in the region. While we are conscious of this source of inaccuracy, we believe it is sufficiently small to not significantly affect the overall model outcomes. Future applications utilising the NetLogo GIS extension will most likely resolve this issue.

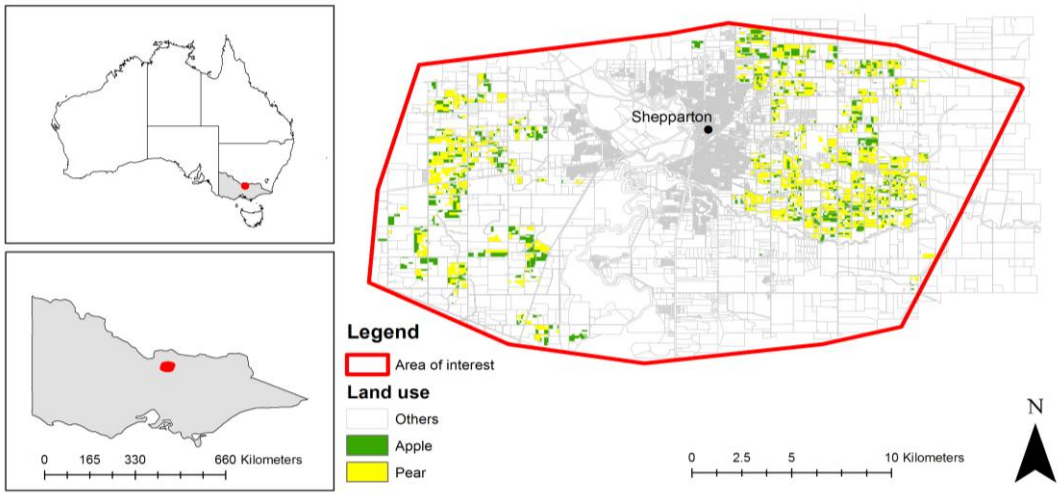

Figure 8. Focus area for the spatial incursion simulation model.

The 926 × 499 grid file was imported into NetLogo using the GIS extension to create a world of 926 × 499 patches using a bottom left corner location of origin and no horizontal or vertical wrap.

3.3.5.2. Model input parameters

The *E. amylovora* model was applied to the Goulburn Valley sub-region defined above and made use of parameters selected to match the attributes specific to its conditions (see Table 6, below). The flowering season was assumed to occur from the second week of September until the second week of October for apple and from the first week of September until the 1st week of October for pear. Pruning was assumed to take place for both apple and pear from the 1st week of May until the 4th week of July. Shoot strike symptoms were assumed to be most visible during the whole month of November but this can be modified as required.

The probability of identifying *E. amylovora* in infected patches at different times of the year was selected based on the author's best guess to:

- 0.1 per cent per week during the flowering period
- 1 per cent per week during shoot strike
- 0.2 per cent per week during pruning
- 50 per cent per detection event in quarantine areas.

The corresponding cumulated annual probabilities of finding an infected patch are shown in Figure 9.

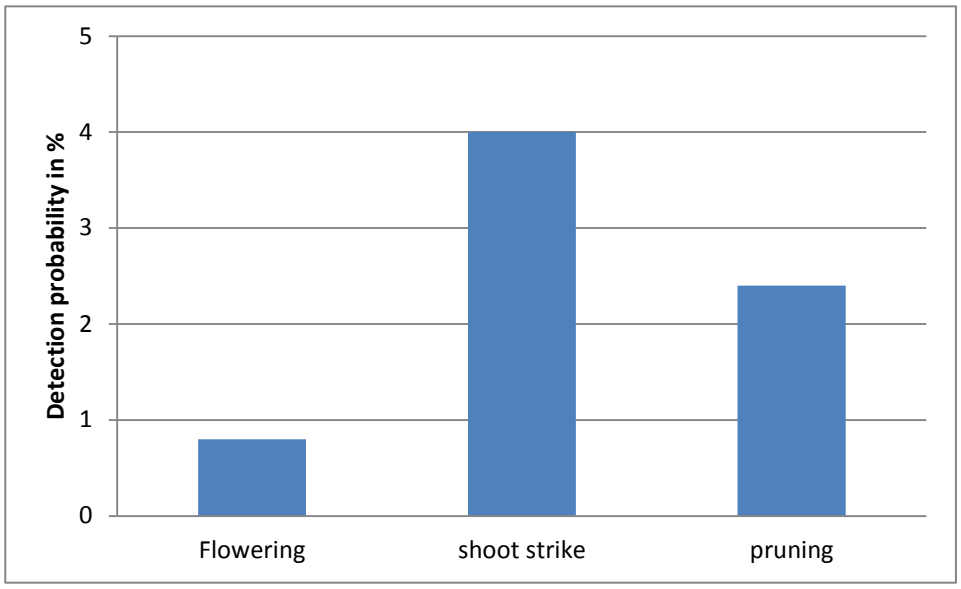

Figure 9. Cumulative annual probability of fire blight detection for infected patches.

Bees were assumed to be able to disperse *E. amylovora* across a maximum distance of three kilometres (Merriman 2002).

Eradication measures, if selected in the model interface, were assumed to follow the guidelines from the revised contingency plan for *E. amylovora* (Merriman 2002). These recommend the creation of a quarantine area of at least three kilometres, restricting the movement of fruit and other plant material from the infected property and increasing disease surveillance. They also recommend the elimination of the *E. amylovora* source by the isolation of honey bees (i.e. no honey bee movement from inside the quarantine area) and the destruction and disposal of infected and suspect trees within a buffer zone (i.e. a 60 metre buffer was used in our model).

The linearised weekly costs of quarantine surveillance in the region were calculated based on the following formula:

$$\frac{\frac{20475(\$/week)}{700000(m2/week)}}{Cellsize\text{^}2(m2/cell)} \div 48(week/year) = 0.624\$/week/32*32m.$$

Where $20,475 is the cost of sending an inspection team of three people into the orchards to actively look for the disease for a week, plus associated costs of incursion management, bee hive removal and communication to the public; 700,000 square metres is the area of orchard that the team can survey in a week (Sosnowski *et al*. 2010).

Table 6. Fire blight model parameters used for the Goulburn Valley application.

Variable	Value
Number of bees	10
Bees dispersal distance	3 km
Quarantine distance	3 km
Eradication zone	60 m
Cell size	32 m
Number of crops	2
Species list flowering season	[["Apple" [9 2 10 2] [5 1 7 4] 6 red] ["Pear" [9 1 10 1] [5 1 7 4] 7 green]]
Detection values	[0.1 1 0.2 50]
Shoot_strike_detection_month	11
Weekly_quarantine_surveillance_cost	0.624 $ / patch
Maximum yield (growth revenue) Apple	12000 $ / ha
Maximum yield (growth revenue) pear	10000 $ / ha
Costs to grower accumulated/year date	1st week of February
Percentage high density values G. Valley	[10% (apple), 2% (pear)]
ORC list HD	[43 47 83 139 61] for apple [43 52 58 152 62] for pear from (Dumont and Boissy 1999)
ORC list LD	[60 86 154 67] for apple [57 179 367 204] for pear from (Dumont and Boissy 1999)
Averages trees per ha [low density, high density]	900 LD 2500 HD

In this particular application of the model, the maturity incrementation module was de-activated so that all orchards remained of constant age during the model run. Therefore, *E. amylovora* was the only factor influencing total apple and pear yield at the scale of the study region.

3.3.5.3. Scenarios investigated:

We investigated the response of our model across the study region to four different scenarios:

- Scenario 1: Live with it management option under low disease pressure;
- Scenario 2: Live with it management option under high disease pressure;
- Scenario 3: Eradication management option with low disease pressure;
- Scenario 4: Eradication management option with high disease pressure.

Each scenario started from the same initial conditions, with a single infected patch located in the bottom left side of the study region (see Figure 10, where the infected area appears in

blue), and ran for a 10-year period. To account for the stochasticity inherent in the model, each scenario was run for 30 iterations. While these scenarios do not represent an exhaustive investigation of the behaviour of the model, they hint at its potential use as a planning tool.

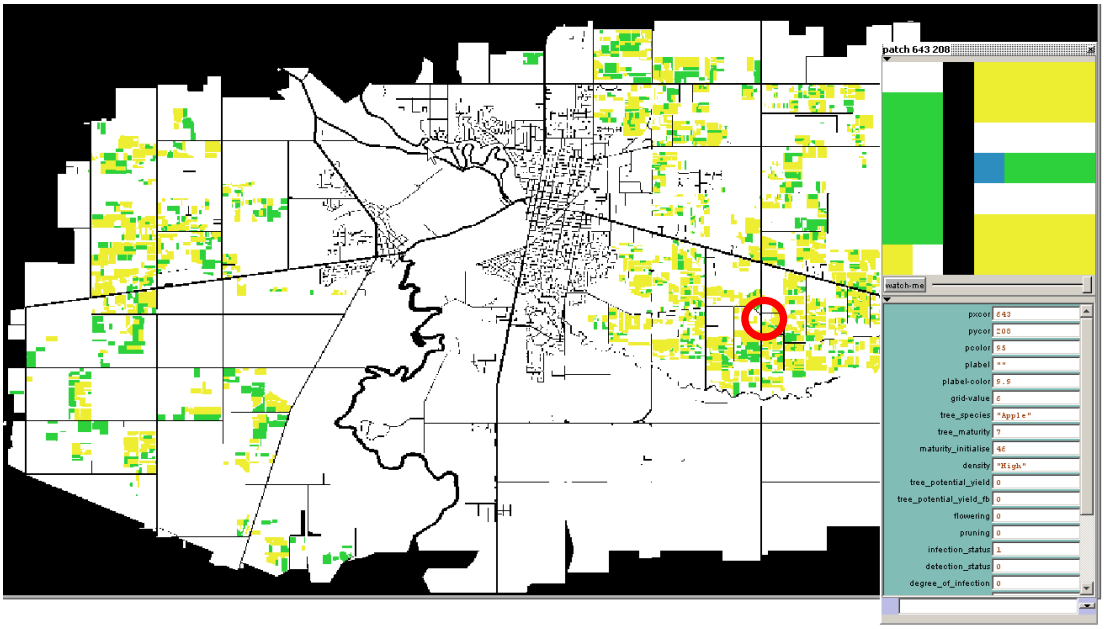

Figure 10. Starting conditions for scenarios.

3.3.5.4. Stakeholder engagement

One of the principal benefits of the model, and one the primary reasons we chose NetLogo as a modelling platform (as explained above) lies in its ability to provide an interactive platform to allow the exploration of various scenarios for managing EPPs across an agricultural landscape. To evaluate potential users' response, the *E. amylovora* model was presented during a hands-on workshop to a panel of over 20 stakeholders, comprising of disease specialists, apple and pear industry representatives and biosecurity incident management specialists. Stakeholders were given the opportunity to interact with the *E. amylovora* model via the use of touch screen interfaces (see Figure 11, below). During two successive sessions, groups of four to six individuals explored a variety of scenarios, defining the location of initial infestations, disease pressure and testing the success of various management strategies to contain or eradicate the disease within a set budget.

Figure 11. Interactive touch screen interface to the fire blight model used during stakeholder engagement.

3.3.5.5. Results

Example outputs from the model as a result of each *E. amylovora* infection scenario are presented in Figure 12. Here, green indicates apple orchards, yellow indicates pear orchards, red indicates detected infection and grey indicates quarantine zones. As expected, across all runs the high disease pressure conditions led to a wider spread of the infection relative to low disease pressure conditions.

In all scenarios, the infestation remained limited to the right side of the map. This behaviour can be explained by the nature of our dataset where the gap between orchards on the east side and the west side of the map was wider than the three kilometre flight range of the bees carrying the bacteria.

In reality, there are many alternative *E. amylovora* hosts such as non-commercial apple and pear trees and ornamental hosts growing in home gardens and on flood plains, as well as native bees, which would allow the disease to spread across this gap. This data was not available to allow evaluation of *E. amylovora* spread across the region.

The use of eradication measures achieved a containment of *E. amylovora* compared to live with it strategies. Both eradication scenarios achieved 100 per cent successful eradication of *E. amylovora* across all model iterations. These results suggest that the use of a quarantine zone of radius larger or equal to the range of the bacteria dispersal by bees, combined with an exhaustive search of infected plants within the quarantine zone and extension of the quarantine area to newly found infection points or foci, could represent a successful eradication strategy. These results therefore support recommendations by the current revised *E. amylovora* contingency plan for Victoria.

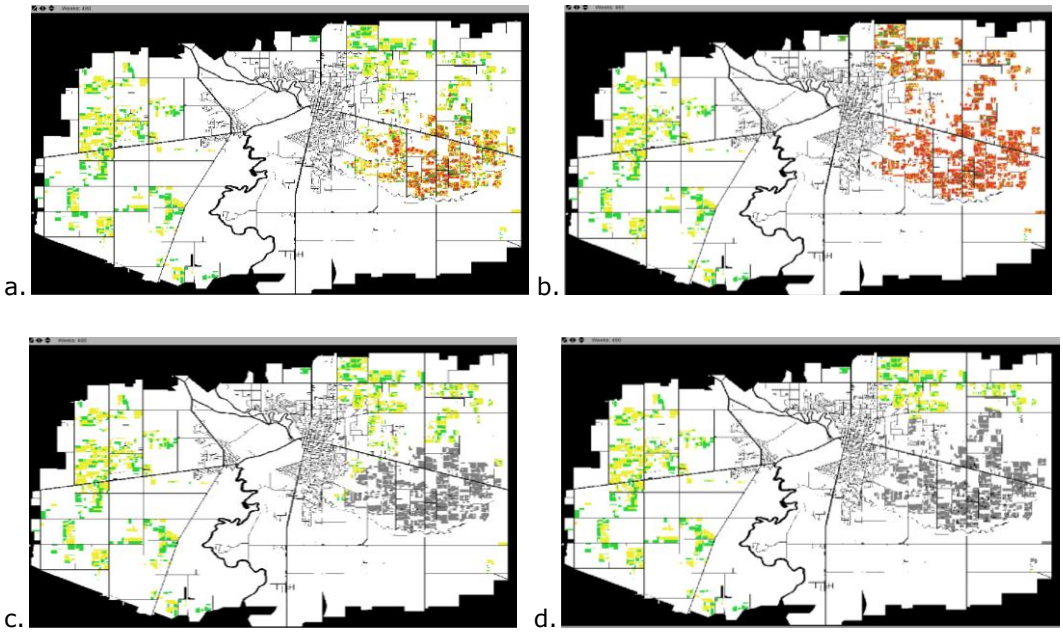

Figure 12. Illustration of model outputs of 10 year runs for: (a) scenario 1; (b) scenario 2; (c) scenario 3, and; (d) scenario 4.

The implementation of an effective eradication program is usually dependant on the availability of funds and cost-benefit considerations, which are determined prior to or during the course of the eradication campaign. Figure 13 presents the results of cumulative grower losses for both 'live with it' scenarios and the costs of simulated response programs for both eradication scenarios across 30 runs.

We observed that for 10-year runs under a low disease pressure, the live with it strategy was marginally more cost effective compared to the eradication strategy as indicated in simulation outputs. Again, costs presented in the model outputs include the cost of response actions (including ORC and maintenance of quarantine zones) as well as grower cost and revenue changes.

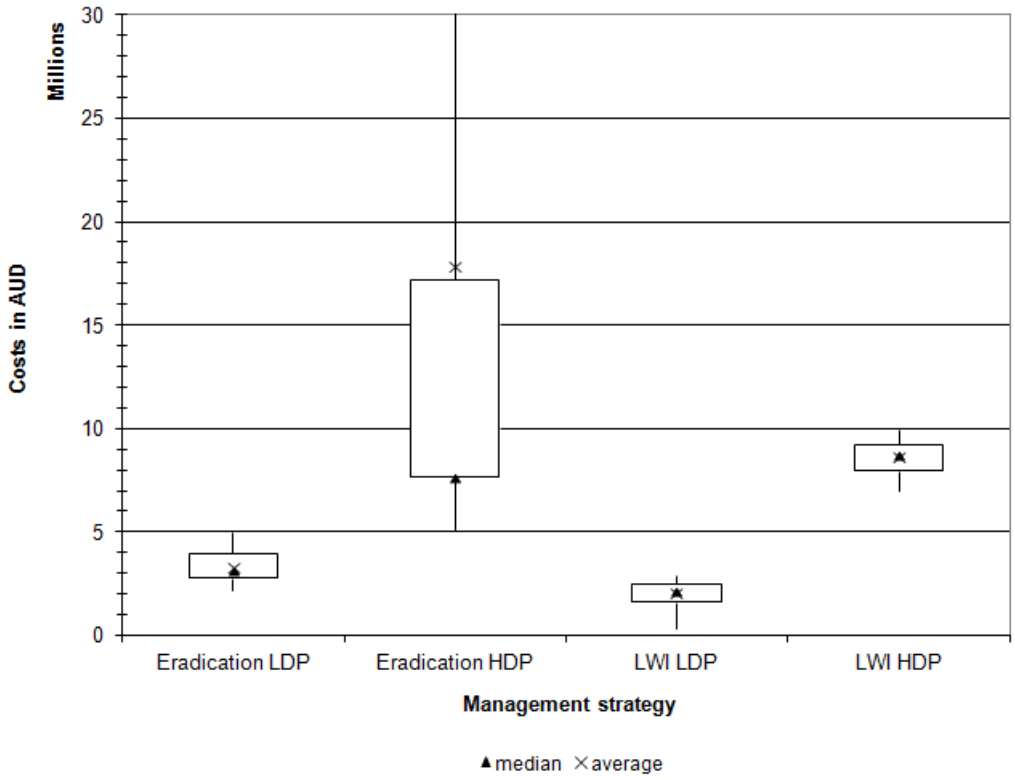

Figure 13. Cost outcomes of 30 model simulations per scenario for Eradication and Live With It (LWI) strategies with Low Disease Pressure (LDP) and High Disease Pressure (HDP), where costs include ORC, maintenance of quarantine zones and grower cost and revenue changes.

The management situation appeared more complicated under high disease pressure. While average cumulative costs to growers under the live with it strategy were much lower than the costs of eradication, the median cost of eradication was lower than that of the live with it cumulative costs to growers. This trend, also observed in simulations using lower dispersal distances and fewer bee densities (although this data is not shown here) indicates that eradication should be the preferred option to the live with it. Under high disease pressure, eradication appeared, in most cases, the most cost effective way to manage a potential *E. amylovora* incursion. However, the enormous variability in eradication costs observed under high disease pressure conditions (i.e. exceeding $130 million in our simulations, which for reasons of scale has not been shown in Figure 13) also indicate that a one rule approach to an *E. amylovora* incursion may not be appropriate to manage all cases.

While the results presented in Figure 13 provide an indication of the potential outcomes associated with different management strategies for one specific infection scenario, we believe that different management parameters, such as smaller quarantine zones, different landscape structures or location of disease initiation will lead to different outcomes. Our model, therefore, does not provide a best answer for all cases, but rather forms a platform on which to investigate scenarios and aid decision makers prepare for and estimate the potential economic outcomes of an *E. amylovora* incursion.

3.3.5.6. Response from stakeholders:

The stakeholder workshop, allowing users to interact with the model and test different incursion and management scenarios, received very positive feedback. The use of the touch screen medium complementing the model graphic user interface was particularly valuable for

encouraging engagement and promoting group investigations and discussions of *E. amylovora* management scenarios.

Some biological assumptions within the model were questioned by stakeholders. In particular, concerns were raised about the flying patterns and behaviour of bees, the lack of alternative *E. amylovora* hosts data and absence of feral beehive data. However, it is important to point out that these issues were recognised by users as they became more comfortable with the model. Moreover, assumptions were questioned as to what impact they would have on the infection spread process and response costs. So, the model itself provided a valuable context through which questions could be framed in terms of their potential implications for management decisions.

3.3.6. Discussion and avenues for future research

We have developed an interactive spatially explicit bio-economic *E. amylovora* incursion management model and demonstrated its potential in the study of the spread and management costs of incursion scenarios.

The framework of the model was designed to be flexible and can be easily modified to simulate spread and economic impact of other pest threats considered to be high biosecurity risks for the apple and pear industry.

The current model, however, is still relatively simple and its potential use as a management tool for a real *E. amylovora* incursion might require further improvements. Possible additions include:

a) The current model does not take alternative *E. amylovora* hosts into consideration. Since *E. amylovora* is known to infect 129 plant species across 37 genera of the *Rosaceae* (Merriman 2002), and the only detection in Australia to date occurred in an ornamental host, it is very likely that our results underestimate the potential rate of spread across locations and provide a conservative estimation of its potential spread. This issue could easily be resolved in the model but would require significant efforts in the collection of GIS field data on the presence and seasonality of these alternative hosts. In the absence of this data, sensitivity tests could reveal how responsive the model outputs are to changes in host abundance and distribution.

b) The model currently only accounts for commercial bees and would need to be modified to incorporate the presence of wild bees. This would also require the collection of additional field data. Once again, in the absence of this data and in a real incursion management situation, sensitivity analysis could be performed to reveal the impact changes in *E. amylovora* spread by bees have on model outputs.

c) Although our costing information was based on published and expert opinion, it did not take into consideration the indirect costs of an incursion such as the impact on domestic fruit trading (i.e. if a cold storage facility is located within a quarantine zone for example) and therefore underestimates the overall potential economical impact on a local region.

d) The establishment of quarantine zones as part of eradication strategies accounted for the removal of bee hives and grower compensation (i.e. ORC), but not the impacts on pollination services and loss of markets in the quarantine zone.

e) A range of model parameters such as the dispersal distance and numbers of bees spreading the disease and the probability of detecting infected patches were based on

the authors' best guess and a full model calibration and validation, based on data collected in the field or during previous incursions (such as the *E. amylovora* incursion in the Po Valley in Italy in 1994) would be necessary.

f) This model was designed to be applied at the local scale and therefore does not take into consideration larger scale economic implications of *E. amylovora* infections such as the costs associated with loss of market access. However in a real incursion management strategy, such costs would also need to be considered.

g) Our model does not directly include climatic conditions (currently approximated via a high/low disease pressure switch). However, these have been suggested as essential for controlling the spread of *E. amylovora* (Calzolari *et al.* 1999). Consequently the capacity to incorporate climate variables (daily weather sequences) or maps to simulate 'optimal' spread conditions would be valuable to allow 'real-time' disease spread forecasting simulations.

The CUBA project has demonstrated the potential and value of an interactive spatially explicit disease spread model. We believe further development could greatly enhance the potential of such a tool in the area of model performance and complexity, and output visualisation / user experience.

The current model architecture is based on NetLogo and is limited in the size and spatial resolution of landscapes it can accommodate. Future work could include the implementation of the NetLogo model into Repast Symphony (as highlighted by Railsback *et al.* (2006)) to allow it to support threading of the program to run in parallel and allow sensitivity analysis or runs over larger area with a larger number of more complex agents. An upgraded model like this could also support the implementation of 'smart' quarantine areas, incorporating factors such as topography, wind patterns and landscape structure, as recommended by the contingency plan.

The development of a digital globe (such as Google Earth) based interface where live steering of the model would be possible could greatly enhance user experience. It could provide an additional layer of contextualising information (see Figure 14) and provide a more interactive and user friendly data viewing environment.

a. b.

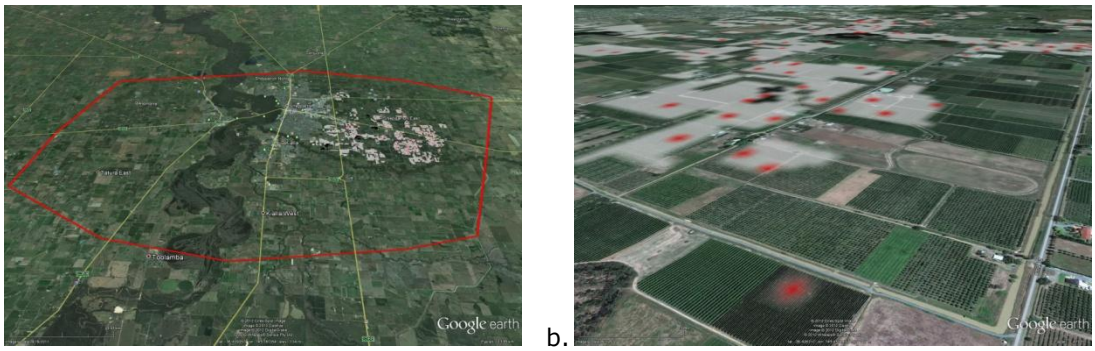

Figure 14. Visualisation of the *E. amylovora* model outputs as a time series imbedded in Google Earth at (a) regional scale and (b) orchard scale.

The use of such an environment could also pave the way to direct linking of field data uploaded by growers via iPhone or tablet device into the model, allowing the forecasting of infection spread and costs in real-time to facilitate real-life incursion management.

3.4. Identifying bio-physical processes for developing an incursion management model

3.4.1 Introduction

This chapter briefly reviews the epidemiology and management of five exotic pest threats for the Australian apple and pear industry in light of modelling framework developed in section 3.3. We investigate some of the main challenges in applying the spatial model to the diseases fire blight (*E. Amylovora*, used to demonstrate the model in the previous section) and European canker of apples (*Neonectria ditissima),* the insect pests Oriental fruit fly (*Bactrocera dorsalis*) and Rosy apple aphid (*Dysaphis plantaginea*)*,* and the mite parasite of European honey bees, *Varroa destructor* (dubbed the Varroa mite). We identify the key biotic and abiotic factors involved in the incursion and spread of these five exotic pest threats to support development of bio-economic models to assess incursion management strategies in the largest fruit producing region (Goulburn Valley) of Australia. We also review phytosanitary measures used for eradication and containment using *E. amylovora* and the Queensland fruit fly as case studies, for inclusion in the model to facilitate assessment of different incursion management responses. This information is supplemented by the threat data sheets provided in Appendix 1.

The apple and pear industry biosecurity plan has included the bacterium *E. amylovora*, the cause of *E. amylovora*, the fungal pathogen *N. ditissima* (previously *Nectria galligena*) the cause of European canker of apples, and the Rosy apple aphid (*D. plantaginea*) as pest threats of high biosecurity priority (Anonymous 2010). The Oriental fruit fly (*B. dorsalis*) and the Varroa mite (*V. destructor)*, a parasite of European honey bees, are also considered important exotic pest threats to the pome fruit industry. *E. amylovora* is considered the most economically threatening of these exotic pests due to its ability to spread rapidly and high economical impact as experienced during the 1997 incursion at the Royal Botanic Gardens of Melbourne (Jock *et al.* 2000; Rodoni *et al.* 1999).

The ability to predict the potential cost of eradicating *E. amylovora* and other exotic pests, or estimating the on-going costs associated with its containment or management if they become established in Australia, can provide useful information for policy analysis, decision making and better inform affected stake holders (Cook *et al.* 2010b; Rodoni *et al.* 2004). The potential economic impact of a hypothetical *E. amylovora* outbreak in Australia's largest pome fruit growing district (Goulburn Valley, Victoria), was estimated using a dynamic multi-regional computer program (Horridge *et al*. 2003; Rodoni *et al.* 2006). This study provided a useful estimate of potential indirect economic costs to industry from an incursion of *E. amylovora* at a regional level.

There has also been interest in using bio-economic models as tools for policy analysis to better understand the impact of potential incursions of exotic pests and effectiveness of eradication approaches in Australian Agriculture. For instance, Cook *et al.* (2010b) used a bio-economic impact simulation model to compare economic importance and impacts of EPPs including *E. amylovora* in Australian horticulture. Elliston *et al.* (2005) used a bio-economic (incursion management) model with a spatial component to track the spread of disease, with grids representing individual paddocks, to investigate the impact of potential exotic pest and disease incursions using Karnal bunt of wheat as a case study. Bio-economic models attempt to capture the interaction between the biophysical (agro-ecological) and socio-economic processes and are therefore very useful tools for assessing the impact of alternative policies on natural resources base and human activity (Brown 2000). This type of modelling can also reveal problems in preparedness and response strategies to improve contingency planning for EPPs such as *E. amylovora*.

3.4.2. Epidemiology and management

3.4.2.1. Fire blight

Infection process

Some of the information that follows draws from section 3.3.2, which outlined the example of *E. amylovora* used as a case study to present the spatial modelling approach developed in the CUBA project. However, for the structural integrity of this report it is repeated here and grouped with similar information about the other EPPs of interest.

E. amylovora bacteria overwinter in cankers on woody branches or infected tissue from the previous season (Beer 1990; Roberts *et al.* 1998). The growth rate of bacteria is influenced by temperature. In the spring, as temperatures increase, the pathogen multiplies in the cankers and cankers begin to exudate bacterial ooze that serves as the primary inoculum. The inoculum is carried via insects such as bees or rain to open flowers (Beer 1990; Norelli *et al.* 2003). The bacteria can grow epiphytically on leaves, shoots and flowers without causing any apparent disease. Infection of host plants is mostly by entry of bacteria through flowers. After colonizing the stigma, the bacteria build up to a critical population size and then move to the nectaries or wounds with the aid of water (Thomson 1986; Pusey 2000; Vanneste and Eden-Green 2000). Infection can spread internally to other parts of the tree, including other blossoms, fruit spurs, twigs, branches, and leaves. After infection, the bacteria grow internally in the plant causing cell death, which is evident as wilting and blackening of the infected tissues. Once the symptoms are visible, there is no effective chemical treatment for *E. amylovora*. To minimise further spread the grower must prune branches at least 12 inches below the visible symptoms. Infections of blossoms and shoots can spread internally to the rootstock and kill the tree (Van der Zwet and Beer 1991).

Secondary infections occur through late blossoms or through wounds caused by pruning, wind abrasion, and hail and insect injury (Beer 1990; Roberts *et al.* 1998). During summer, severe infection may occur on shoots, leaves and fruits following a climatic event such as hail storms which produces wounds on the plant surface.

Host range

E. amylovora is now present in 30 European countries, North America, some Asian countries, New Zealand and many others (CABI 1997; Roberts *et al.* 1998). Host plants considered for survey include plants which are commonly recorded as naturally occurring hosts of *E. amylovora* and those for which there are occasional records of natural infection. These are described in detail in the contingency plan for *E. amylovora* (Merriman 2002). *E. amylovora* is especially destructive to cultivars of apple (*Malus*) and pear (*Pyrus*) but can infect also quince (*Cydonia*) and loquat (*Eriobotrya*). Important ornamental hosts include cotoneaster (*Cotoneaster*), hawthorn (*Crataegus*), firethorn (*Pyracantha*), and mountain ash (*Sorbus*). Rootstocks M 9 and M 26 are highly susceptible to *E. amylovora*. In Australia, ornamental hosts are present in home gardens, city parks, country towns and within fruit production regions. In Victoria, the flowering period for important pear cultivars (William Bon Chretien and Packham) starts in early September, usually a week or two earlier than apple cultivars, and ends in early October. The flowering period for most apple cultivars starts in mid September and ends in mid-to-late October.

Dispersal, occurrence and losses

Short range dispersal of *E. amylovora* by natural means has been reported to include bees, wind-blown rain, rain, insects, and aerosols (Beer 1990; Roberts *et al.* 1998). Other climatic

factors such as frost, hail and thunderstorms can spread and increase the severity of infections. Long range dispersal is mainly by spread via infested nursery stock. The ability of *E. amylovora* to survive in bee hives for a limited period has been demonstrated (de Wael *et al.* 1990). However, the transfer of inoculum from the hives to flowers is considered unlikely and there is no evidence the bacteria overwinters in hives. For more comprehensive reviews of *E. amylovora* biology and spread see reviews by Roberts *et al.* (1998, 2008) and the diagnostic protocols for *E. amylovora* (www.padil.gov.au).

While aspects of the infection and dispersal processes are clear, field observations where *E. amylovora* is a problem indicate that *E. amylovora* is more problematic in some seasons than others and varies greatly from one orchard to another in any specific year (Smith and Pusey 2010). While outbreaks can be sporadic in occurrence, they often result in significant loss of trees, yield and orchard profitability as demonstrated by severe outbreaks of *E. amylovora* in the USA and attempts to eradicate or contain the spread of *E. amylovora* in Europe. In 1998, apple and pear growers in Washington and northern Oregon suffered substantial losses (estimated at $US 68 million) due to severe *E. amylovora* outbreaks (McManus and Stockwell 2000). In the Po Valley, Italy, approximately 500,000 pear trees have been destroyed since 1997 to attempt to eradicate *E. amylovora* (Calzolari *et al.* 1999). A large number of pear, apple and quince trees (580,000) were destroyed in Romania between 1993 and 1997, and apple and pear trees (340,000) in Croatia since 1995 in an effort to halt the spread of *E. amylovora* (Cvjetkovic *et al.* 1999, Severin *et al.* 1999). Although *E. amylovora* is erratic in occurrence mostly due to weather variability, several factors associated with current orchard management practices may increase the vulnerability to *E. amylovora* resulting in more frequent and devastating disease outbreaks (McManus and Stockwell 2000). These factors include orchard density, tree sizes, susceptibility of cultivars grown and training systems. The combination of susceptible cultivars using popular size-controlling susceptible rootstocks (M 9 and M 26) for achieving high tree densities further complicates the susceptibility issue. Other factors such as possible variation in cultivar flower susceptibility remain uncertain. Recent work suggests that the bacteria are able to colonise susceptible pear plants through roots via soil irrigation water.

Disease forecasting

The growth rate of *E. amylovora* is influenced by temperature. Ideal conditions for infection, disease development and spread of the pathogen are wet or humid weather with daytime temperatures in the range of 18 – 30°C and night temperatures above 15°C (Beer 1990; Roberts *et al.* 1998). In some countries affected by *E. amylovora* in Europe, spring is often dry and cold and therefore the risk of *E. amylovora* is low. However, in summer rainfall and temperatures can be high, with maximum above 20°C which are conducive for *E. amylovora* development (Sletten and Melboe 2004). Many infection events can occur during the growing season when suitable weather conditions are met. In New Zealand, for example, *E. amylovora* infection can occur during the bloom period if unusually warm temperatures coincide with wet weather (Beresford pers. comm. 2012). A preliminary analysis using 10 years weather data and the *E. amylovora* model Cougarblight (Smith and Pusey 2010) also indicate that these conditions can occur during the bloom period and after bloom (mid-October-November) when rat-tail flowers hang around in the Goulburn Valley, Victoria (unpublished). Nevertheless, disease risk in a given orchard depends also on the spatial proximity of flowers to cankers from the previous year. Infection risk forecasting is therefore a vital preventative tool for timing the application of treatments to control *E. amylovora* in a seasonal basis in countries where the disease is endemic. If cankers are not present in the orchard, then the risk of infection is low. The days of high risk of infection are identified using weather data from meteorological stations. On the day or the day before the infection risk is identified the warnings are issued to initiate appropriate controls.

There are three main systems available (Maryblight, BIS95, Cougarblight) to predict *E. amylovora* infection risk, based on predicting pathogen incubation period and when symptoms may be visible. In Maryblight (developed at Maryland USA), four conditions must be met in order for infection to be possible: open flowers; at least 110 degree-hours with an average temperature above 18.3°C; existence of wetness event above 0.25 millimetres during the current day or 2.5 millimetres during the previous day; average temperature above 15.6°C (Stener and Lightner 1996). In BIS95 (developed in England), wet spreading happens when three conditions are met: at least 17 days accumulated (degree days) where the maximum temperature was above 18°C; average temperature above or equal to 15°C; existence of wetness above three millimetres (Billing 1996). Dry spreading occurs when two conditions are met: at least 17 days accumulated where the maximum temperature was above 18°C and maximal temperature above or equal to 27°C or average temperature above or equal to 20°C. In Cougarblight (developed for apple and pears in the Pacific Northwest USA), the basic components and assumptions are: orchard *E. amylovora* history; flower life/colony growth; bacterial growth rate based on average pathogen growth rate per 24 hours, divided by 24 to give an hourly growth, then multiplied by 1000 to make the model numbers easier to use; wetting as trigger for infection (Smith and Pusey 2010). A current Excel version of this model is available at http://www.ncw.wsu.edu/treefruit.

New Zealand has operated a weather-based *E. amylovora* warning system since 1990. The models used are Maryblight and Cougarblight. Cougarblight is the standard because it is simpler to interpret and gives the same results as Maryblight. Cougarblight accumulates hourly air temperature readings for the current day and previous three days. If wetness occurs on the current day, *E. amylovora* risk is reported in four categories: low, moderate, high and severe.

Control

Blossom blight control is critical because successful infection enables the bacteria to multiply and spread internally in trees, allowing build up of pathogen populations and inoculum for further infections (Beer 1990). Blossom blight is managed with anti-bacterial treatments including antibiotics and biological control agents applied to protect flower tissues against infection. Antibiotics are effective when the bacteria are multiplying on the surface of flowers before infection. Streptomycin is the most effective antibiotic as it kills the bacteria (McManus and Jones 1994; McManus and Stockwell 2000). Oxytetracycline is less effective than streptomycin because it only suppresses the growth of bacteria. The efficacy of other antimicrobial products including biological control agents is lower than streptomycin. Pre-bloom copper sprays can decrease populations of the bacteria at canker margins but copper is not generally used after bloom due to its phytotoxic effect on fruit. The antibiotic streptomycin has been the main control for *E. amylovora* in the US, however, intensive use of streptomycin during bloom, and later in the season for shoot blight control, has led to the development of streptomycin resistance (Chiou and Jones 1993). A maximum of two applications per season are permitted during flowering in EU countries under strict official control (Nemeth 2004). In New Zealand, some growers use streptomycin or diluted copper sprays against *E. amylovora* during the bloom period but *E. amylovora* is mostly managed with pruning and weather monitoring to identify periods of infection risk (Beresford pers. comm. 2012).

Pruning and destroying infected materials is the most important sanitation practice for *E. amylovora* control. Fire blight cankers and blighted shoots are mostly removed and burned during winter pruning. While all pear cultivars are considered susceptible to *E. amylovora*, some apple cultivars are reported to have genetic resistance to *E. amylovora* (McManus and Stockwell 2000). However, many popular varieties in high demand such as Gala, Pink Lady and Braeburn are susceptible to *E. amylovora* (Van der Zwet and Beer 1991).

3.4.2.2. European canker of apples (Neonectria ditissima)

European canker, caused by *Neonectria ditissima* (formally *Nectria galligena*) is another economically important disease of apples in many European countries, California, Chile and New Zealand. The disease causes cankers on twigs and branches reducing yield and tree vigour but also causes fruit rots at the calyx end in some countries (Latorre *et al.* 1999; Swinburne, 1975). *N. ditissima* has a wide host range which includes more than 60 tree and shrub species from 20 genera including pear (*Pyrus* spp.) and many important amenity trees as reported by Edwards *et al.* (2006) review for Australia. The fungus can enter its host through wounds caused by pruning, insect feeding, winter injury and invasion by other pathogens. Infection also occurs through leaf scars in autumn, which is weather dependent (Grove 1990; Xu and Butt 1994).

The disease is prevalent in commercial apple and pear orchards from most temperate growing regions of the world (Swinburne 1975). In Chile, severe outbreaks can occur after cool and rainy weather conditions during leaf fall throughout March-July with disease incidence varying from year to year depending on weather conditions. For instance, disease incidence ranged from 0.01 per cent to 48.3 per cent on one-year-old twigs in the same orchard in dry and wet seasons respectively (reported in Latorre *et al.* 2002). Both conidia and ascospores may cause infection. In the main production regions of Chile and California conidia are more important for infections (Grove 1990, Lolas and Latorre 1996) whereas in European countries ascospores predominate (Swinburne 1975). Ascospores, produced in red perithecia in cankers, appear mainly in the spring and have been seen in southern Chile at lower latitude 36-42° south (Latorre *et al.* 2002).

Production and release of spores is largely climate dependent, and is most common in spring and autumn. However, spore production and infection of host tissue can occur at any time of the year as long as there is sufficient moisture and temperature is above 5°C. Temperature and free moisture duration are important factors in the infection process of leaf scars of apples by conidia in autumn (Dubin and English 1975; Latorre *et al.* 2002). In an *in vitro* study by Latorre *et al.* (2002), **c**onidia germinated from 6°C to 32°C with the optimum between 20°C and 25°C which is in agreement with work by Dubin and English (1975). However, the germination rate of ascospores was observed to be faster than conidia, suggesting that European canker could be more aggressive in areas where ascospores are produced during leaf fall. Free moisture is also essential for production of sporodochia and conidia and for release of conidia (Lortie 1964; Swinburne 1975). Conidia are dispersed by moist wind currents and rain splash and in some cases carried by insects to susceptible tissue (Houston 1994). Ascospores can be dispersed by rainsplash but are generally considered to be aerially dispersed. Ascospores therefore are capable of long range dispersal, while conidia for short distance dissemination. It is widely accepted that leaf scars become progressively resistant with time and the requirement for wetness duration for infection may increase accordingly (Dubin and English 1975).

Sanitation alone (i.e. pruning out cankers and destruction) is not sufficient to control this disease, with chemical treatments often needed to avoid severe damage. Copper compounds alternated with synthetic fungicides are widely used during leaf fall (Lolas and Latorre 1997). Protective fungicide treatments applied during leaf fall have reduced infection in the next season confirming that leaf scars are important infection points for *N. ditissima* (Lolas and Latorre 1997; Cooke *et al.* 1993). An incursion of European canker in Tasmania in 1954 was successfully eradicated through tree removal and drastic pruning (Ransom 1997). This disease can become established in other regions of Australia where susceptible hosts are available and climate conditions are conducive for disease development (Edwards *et al.* 2006).

3.4.2.3 Rosy apple aphid and Oriental fruit fly

The rosy apple aphid (RAA), *Dysaphis plantaginea*, and the Oriental fruit fly (OFF), *Bactrocera dorsalis* are two other economically important exotic pest threats for the Australian apple and pear and other industries.

RAA, a worldwide problem for apple growers, can decrease fruit setting (Blommers *et al.* 2004), resulting in over 30 per cent yield losses when not controlled (De Berardinis *et al.* 1994). Its reproduction involves both sexual and asexual pathways (Blommers *et al.* 2004). RAA completes its life cycle on two successive host plants. From early autumn to late spring the apple tree is the primary woody host and during summer the winged adult female also feeds on narrow and broad leaf plantain and dock (*Plantago* spp.), a herbaceous host plant. Apple is the preferred host but they also can feed on pear and hawthorn. The need for secondary hosts is unlikely to be a constraint in Australia. Plant stages affected are vegetative and flowering stages. There are two short periods for control: early spring when recently hatched fundatrices (pathenogenetic viviparous female aphids) are not yet protected by curled foliage (Brown and Mathews 2007) and in autumn before mating and overwintering egg laying (Kehrli and Wyss 2001). The pest can spread short distances by natural means such as flight (winged aphids) or wind. Transport of infested fruit or plant material can result in long range dispersal.

OFF is one of the most destructive pest insects of tropical and subtropical fruits and vegetables. *B. dorsalis* has been recorded from more than 150 fruit and vegetables, including apple, with avocado, mango and papaya the most commonly attacked. Damage of fruit can be up to 100 per cent of unprotected fruit. For instance on Mango (*Mangifera indica*), it causes losses up to 80 per cent or higher in unprotected fruit (Verghese and Jayanthi 2004). Potential distribution in Australia will include tropical and sub-tropical where host fruit are grown, particularly along the Queensland and northern New South Wales coast, Northern Territory and north-west Western Australia. In India, *B. dorsalis* survives on its alternative host guava (*Psidium guajava*) during the mango off-season (mid-August to March), therefore it is able to complete several generations within a year (Verghese and Jayanthi 2004). Constant monitoring of fruit fly populations and accurate forecasting of incidence forms part of an effective management strategy. The strategy includes sanitation, male annihilation traps, along with need-based insecticidal cover sprays during fruit maturity, the stage most vulnerable to attack by ovipositing gravid females (Jayanthi and Verghese 2011).

Apart from weather factors such as temperature, rain, wind speed, and humidity, many biotic variables such as host plants can exert influence on aphid and fruit fly population dynamics. Therefore like many crop pests, aphid and fruit fly population sizes and severity of attack vary among years (Mumford and Norton 1984). This variation must be taken into account when developing an incursion management model to assess response strategies for these pests in Australia. Adult flies can disperse over long distances through flight, while the transport of larvae in infested fruit can result in global movement.

3.4.2.4. Varroa bee mite (Varroa destructor)

The Varroa bee mite, *Varroa destructor*, is considered the most serious pest of the European honey bee (*Apis mellifera*), the principal pollinator in many crop production systems and the main producer of honey worldwide (Rosenkranz *et al.* 2010). Within a short time, *V. destructor* has spread almost worldwide except in Australia. It is estimated that without treatment, most of the honey bee colonies in temperate climates would collapse within a two to three year period (Rosenkranz *et al.* 2010). After introduction into bee hives, Varroa mites invade brood cells, where offspring produced by female mites feed on the developing host

bee causing weakness or death. On emergence from the brood cell, juvenile Varroa mites attach to the body of the host bee causing further harm.

Dispersal of this mite is by natural movement activities of European honey bees, such as drifting (transfer of small number of bees between colonies) and fission (when a portion of adult bees leave established colonies to create new ones) facilitating short-distance (local) spread of Varroa. In addition, local spread of Varroa occurs via 'robbing' (distance suggested more than one kilometre), where honey and mites are transferred from hive to hive via robber bees. Spread of Varroa over long distances occurs primarily as a result of beekeeper activities. After the first infestation of a new honey bee colony, Varroa mites are able to build up high populations within a few years (Rosenkranz *et al.* 2010). The mite population growth is highly variable and depends on the interaction between host and the parasite and ambient factors such as climate and nectar flow. Mortality of entire colonies ('hive collapse') can occur after a variable period when the level of Varroa infestation within the colony is high and control measures are not applied (Shimanuki *et al.* 1994).

An economic impact study of the Varroa incursion in Auckland, New Zealand, estimated that it would cost the national economy NZ $400 to $900 million over a 35-year period, primarily as a result of adverse effects on the production of Kiwi fruit and the growth of improved pastures (Anonymous 2000). An eradication policy was deemed feasible only if the infested area could be accurately defined and depopulated of domestic and feral hives and if other aspects of the epidemiology of the mite could be determined with greater certainty. Most apple and pears growers in the Goulburn Valley use one to three bee hives per hectare to achieve good pollination during flowering, especially in areas where the population of native bees is low such as away from rivers. The effect of an incursion by Varroa in the Goulburn Valley will greatly affect pollination levels and thus yields but this would depend on the level of Varroa infestation of bee hives. The exact impact of Varroa on native populations of bees in the Goulburn Valley is not known.

A spatial epidemiology study of the Varroa incursion in the North Island of New Zealand estimated the possible rate of local spread of Varroa under local conditions which in turn allowed policy decisions regarding Varroa management to be more clearly informed to stake holders (Stevenson *et al.* 2005). The study indicated that the possible maximum rate of spread of Varroa based on data available from the incursion point was in the order of 12 kilometres per year (i.e. an interquartile range 10-15 kilometre). This provided a potential surveillance zone around a new identified incursion point for every 12-month period following the date of the high risk movement of Varroa into an area known to be free of disease.

3.4.2.5. Queensland fruit fly

The Queensland fruit fly (*Bactrocera tryoni*) is a native Australian species with the ability to infest a wide range of host fruit species including stone fruit, grapes, citrus, pome fruit (apples and pears) and tomatoes. In response to many outbreaks in Victoria, including the Goulburn Valley, the Department of Primary Industries of Victoria manages this pest through an area-wide pest management program which delivers coordinated pest response and surveillance programs across all production and urban regions according to a national code of practice (DPI Victoria 2009, 2010). The Queensland fruit fly reduces fruit quality so the fruit is not marketable. The area-wide management program allows producers and exporters to consign fruit under area freedom certification to gain access to markets. The program uses international standards for phytosanitary measures (ISPM 26, ISPM 29 and ISPM 30) as a basis for establishing a area-wide management program for fruit flies. This is managed in accordance with a nationally agreed code of practice that describes surveillance, control, diagnostic and reporting requirements for fruit fly management in pest free areas.

The fruit fly is monitored using permanent traps positioned on a one kilometre grid in horticultural production areas and a 400 metre grid in urban centres (DPI Victoria 2009, 2010). If five or more fruit flies are detected in a single trap within 14 days, a 15 kilometre outbreak suspension zone is declared and area freedom status is lost. Once the suspension zone is declared, an eradication program begins, which includes chemical control and mating disruption measures. The flight range of the fruit fly is not known exactly but it is assumed that it can fly beyond the two to three kilometre ranged reported for bees (Hossain pers. comm.). The population size is limited by climate and breeding sites, becoming less active during winter. The females lay eggs five days after mating. Adult female flies can lay several hundred eggs in their life time and they live for several months. If left uncontrolled (depending on the temperature) several generations of fruit flies can overlap creating a large population of fruit flies in an outbreak area (O'Loughlin *et al.* 1984).

3.4.3. Eradication of fire blight and European canker

3.4.3.1. Fire blight and European canker in Australia

In 1997, an outbreak of *E. amylovora* occurred at the Royal Botanic Gardens of Melbourne, Victoria (Jock *et al.* 2000; Rodoni *et al.* 1999). Response actions taken over three years resulted in the containment and eradication of the disease (Rodoni *et al.* 2002). Although this outbreak was detected outside a fruit-growing area or nursery, it was estimated that this incident cost the Australian pome and nursery industries approximately $20 million in lost revenue (Rodoni *et al.* 2006). In Australia, imports of host plant material have been highly regulated under the quarantine act and this has prevented so far the entry of the bacterium into Australia. However, the risk of an incursion and establishment of *E. amylovora* is still very high, especially with weather conditions becoming increasingly favourable for disease development (e.g. warmer and wetter springs). In New Zealand, *E. amylovora* was first recorded in 1919 (Cockayne 1919). It causes noticeable damage once every five to 10 years at a regional level and individual orchard blocks (Beresford pers. comm. 2012). Infection occurs during the bloom period if unusually warm temperatures coincide with wet weather. The last substantial outbreak occurred in Hawke's Bay in 1998. The pome fruit industry in New Zealand manages the *E. amylovora* problem by pruning out cankers, copper sprays and weather monitoring to identify infection periods to schedule antibiotic sprays during bloom. European pears are considered more susceptible than apples but they flower earlier when temperatures are cooler and may escape infection.

Pathogens that infect the internal parts of the host (systemic) such as *E. amylovora* require complete removal of host for elimination. Removal and destruction of host plants infected by this bacterium in the Royal Botanical Gardens of Melbourne (RBGM) resulted in successful eradication from Victoria, Australia (Rodoni *et al.* 1999, 2002). Complete removal and destruction of whole apple trees and orchards also led to the eradication of apple scab (*Venturia inaequalis*) in WA (Cass Smith *et al.* 1948). However, apple scab has been detected again and is now recognised as established and no longer included in the apple IRA as a pest of concern for WA only. The apple pathogen survives during winter mainly as pseudothecia (i.e. sexual stage) in infected leaves on the orchard floor (Villalta *et al.* 2000). Ascospores released from overwintered leaves are the main source of inoculum for primary infections in spring but conidia on infected buds and twig infections can also provide inoculum for infections. Short and long (i.e. local) range dispersal is achieved by ascospores carried to tree canopies by wind currents during flowering and periods of abundant vegetative growth. Short range dispersal occurs via conidia spread within trees by rain splash. Long range dispersal is achieved by movement of infected fruit, leaves and wood. Although the apple scab pathogen is not considered to be systemic, complete destruction of

whole trees and orchards affected was probably necessary to eradicate the disease in WA due to the various mechanisms of spread and difficulty in locating all sources of inoculum.

Wood pathogens that don't spread systemically such as *N. ditissima,* are probably likely candidates for eradication by drastic pruning. Drastic pruning was used successfully to eradicate European canker (*N. ditissima*) from apple orchards in Tasmania in 1958 (Ransom *et al.* 1997). An extensive eradication program was carried out for many years resulting in area freedom declared in 1991. At the time of the incursion, no estimate was made of the cost of this incursion to Australia.

3.4.3.2. Recent Fire blight eradication programs in Europe

Norway

An eradication and containment program has been successful in limiting the spread of *E. amylovora* in Norway after removal of infected plants and highly susceptible plants from infested areas (Sletten 1990; Sletten and Melboe 2004). The first outbreak of *E. amylovora* was detected in 1986 on ornamentals, in particular Cotoneaster (*Cotoneaster bullatus* and *C. salicifolius*) in Rogalandon, south west coast of Norway. Diseased plants were found in private gardens, around public buildings, in recreational grounds, along roads and in rural areas. There were no commercial fruit trees but nurseries in the district where the outbreak occurred. A quarantine area of about 700 kilometres was established for ten years around the focus of infection. New infections of *E. amylovora* were subsequently detected in this area in about 2000 locations during 1986-1993, with decreases observed from 1990 on-wards. The disease is restricted to an area of about 1,500 km^2 in the Rogaland and Hordaland counties on the West coast, which have a total area of about 25,000 km^2. The disease has remained within the restricted area, with some spread to nearby areas mainly due to uncontrolled movement of beehives, and has not moved into important fruit-growing regions or nurseries. New outbreaks were also linked to occurrence of favourable weather, unusually warm spring and summer with rain or high humidity for disease development. Hawthorn is important as a source of inoculum in Norway due to early flowering and infection and difficulty in monitoring plants of this genus.

Exactly how *E. amylovora* was introduced into Norway is not known. Transmission by wind or birds from other European countries is unlikely because of the very long distance to areas where *E. amylovora* is present. Illegal importation of diseased ornamentals is one possibility. Some of the phytosanitary measures introduced to stop the spread of the disease included destruction of susceptible hosts around orchards and nurseries and restriction of beehive movement. Production and sale of Cotoneaster plants is prohibited through the country. Buffer zones of 500 metres free from the most susceptible hosts were established around fruit orchards and nurseries. Managed beehives in the quarantine zone were only allowed to be moved to areas free from *E. amylovora* hosts. Successful containment of *E. amylovora* in Norway was due to an effective eradication campaign which included reduction of hosts and thus inoculum for disease spread and because the disease did not enter commercial orchards or nurseries. These and other drastic measures could not have been accomplished without a public awareness campaign and systematic surveillance of quarantine areas and other regions of Norway (Sletten and Melboe 2004).

Italy

Complete removal of pear trees infected by *E. amylovora* did not eradicate *E. amylovora* in Italy (Calzolari *et al.* 1999; Finelli *et al.* 2004). Before the incursion, import of *E. amylovora* host plants from countries known to have the disease were restricted and subjected to quarantine regulations (Finelli *et al.* 2004). Despite this, *E. amylovora* was found for the first

time in Puglia in southern Italy in 1990. Subsequently, a national monitoring program was established to determine the extent of the *E. amylovora* incursion. In 1994, the first outbreak was found in Emilia-Romagna, the most important pear growing region of Italy. In 1997, a severe epidemic spread throughout this region and other first cases were reported in two bordering regions (Veneto and Lombardia). At 36,000 hectares, the Po Valley, located in the Emilia-Romagna and Veneto regions, is considered the largest pear growing region of Europe, and indeed the world. Pears were seen to be very susceptible to *E. amylovora* and during the epidemics of 1997 and 1998 more than one million pear plants were destroyed by *E. amylovora* (Finelli *et al.* 2004).

Since 1998 the spread of *E. amylovora* has stabilized due to regulations established to control *E. amylovora* including monitoring to contain or delayed the spread of disease in orchards, destruction of ornamental hosts infected, protection of nursery production and regulation of the movement of beehives. Two regional 'protected areas' were set up where production of *E. amylovora* hosts (fruit trees) are allowed for planting. The risk of long-distance spread by movement of bee-hives is managed by restricting their movement from infested to pest-free areas during the flowering period of the main host plants. The impact of *E. amylovora* has been greatest on pears, with cases of *Crataegus* (hawthorns) also reported. On pear, few primary infections of flowers are reported, but shoot infections and secondary flower infections are common. Other host plants such as *Malus* spp. (apple), *Pyracantha* spp. (firethorn) and *Cotoneaster* spp. (cotoneaster) are rarely infected.

In the Emilia-Romagna region, regional monitoring is carried out by teams of two to three trained people each, initially surveying the areas around the outbreaks and then the full growing areas of pears. Buffer zones are set around important nursery production. Information from surveys at provincial level is fed into a geographical information system (FitoGIS). In FitoGIS all nurseries, buffer zones, outbreaks and monitoring points are mapped manually or by GPS. This system facilitates the management of *E. amylovora* and has useful elements to study the spread of disease in different environments. In the Veneto region, all pear orchards are inspected in an area 20 kilometres wide and 240 kilometres long.

Slovenia, Czech Republic, Austria, Slovakia and Bulgaria

Eradication efforts were also unsuccessful in Slovenia (Knapic *et al.* 2004), Bulgaria (Dimitrova and Andreev 2004), Slovakia (Sivicek 2004) and the Czech Republic (Behalova 2004).

In Slovenia, *E. amylovora* was detected for the first time in 2001 on three trees, including one old pear tree at one (Naklo) out of 791 locations surveyed all over the country. In 2002 and 2003, the disease was found in another location (Skofja) within a 15 kilometre radius from Naklo. Host plants showing *E. amylovora* symptoms plants were destroyed. During the 2003 epidemic, 73 new foci were recorded in a 15 kilometre radius from the first focus found in 2002, mostly as a result of blossom infection. By the end of the growing season, a further 111 foci were found in Gorenjska and 23 foci scattered in other regions. Plants infected confirmed by testing included *Malus* spp. (50 per cent), *Pyrus communis* (21 per cent), *Cydonia* spp. or quince (15 per cent), *Cotoneaster* spp. (10 per cent), *Pyracantha* spp. (two per cent), *Crataegus* spp. (one per cent) and *Chaenomeles* or flowering quinces (one per cent). Typical symptoms of *E. amylovora* were observed in plants of *Sorbus*, *Mespilus* and *Pyrus pyrifolia* but these gave a negative test results. Weather conditions have been favourable for the formation of bacterial exudates on infected fruit, with shoot infections observed at the end of spring. Despite strict phytosanitary measures implemented after the discovery of first outbreak, the bacterium spread to an entire region (Gorenjska), with further spread reported in eastern and southern Slovenia. A total of 93,809 nursery plants,

36,743 apple and pear trees in commercial orchards and another 2,760 apple, pear and Cydonia trees were destroyed to attempt to eradicate *E. amylovora*, (Knapic *et al.* 2004). The experience in Slovenia suggests that infections were undetected in orchards during surveys. Spread of the bacterium was caused probably by favourable weather conditions for disease development, availability of inoculum and bee-hive movement, resulting in flower infection during the growing season.

In the Czech Republic, *E. amylovora* has been spreading since 1986 in the direction of the predominant westerly winds and spread slowed down probably by the Bohemian-Moravian highlands (Behalova 2004). It was found originally at four locations in Prague's city parks and Cotoneaster plantations imported from the GDR. Surveying are conducted in spring only if conditions are favourable for disease development. Summer and autumn surveys are conducted only if there is a possibility of 'late infection' through secondary flowering of pear. Winter surveys focus on host plants showing *E. amylovora* symptoms. Surveys are conducted only once in non-infested areas. Survey of nurseries for registered growers is carried out twice a year (i.e. in summer and autumn).

In Austria, several *Cotoneaster salicifolius* plants were found infected in 1993 in the most westerly part, close to the German border. Since then there has been an increase in *E. amylovora* outbreaks and spread from the west to the east. Implementation of control measures has reduced the incidence of *E. amylovora* and probably slowed down its spread for several years (Keck 2004). However, the use of eradication measures (i.e. pruning and burning) and regulation of beehive movements were not successful in eradicating the disease. These measures have not been either effective in preventing new disease outbreaks.

Despite the phytosanitary measures undertaken by the Slovakian and Bulgarian NPPOs, they have not succeeded in preventing the spread of *E. amylovora*. In Bulgaria, the bacterium was first detected on quince in 1989 which became epidemics during 1995/1997 due to favourable weather for disease development. The main hosts found frequently infected were pear, quince, apple and Cotoneaster. Thirteen years after the first confirmed case of *E. amylovora*, fire blight is distributed all over the country. In Slovakia *E. amylovora* was found first on pear in 2003. After the first outbreak, the disease has been found in 194 out of 384 samples taken from gardens, public parks and wild hosts, with only 13 samples taken from commercial high density orchards testing positive for *E. amylovora*. Hosts infected included *Cydonia*, *Malus, Pyrus, Crataegus, Pyracantha, Coteneaster, Chaenomeles* and *Mespilus.*

In summary, reasons for failures to eradicate *E. amylovora* from many European countries include wide distribution of very susceptible hosts in homes and public land and wild hosts which provide a source of inoculum for disease spread and inability to detect infections in host plants during surveys due to weather conditions unfavourable for disease development.

3.4.3.3. Phytosanitary and eradication measures

The current strategy for eradication of *E. amylovora* therefore relies on the removal and destruction (i.e. burning and/or burial) of affected and potentially affected host plants. Successful elimination depends on multiple biological factors, including interactions of the pathogen with its hosts, the presence of alternative hosts and geography and environmental conditions. Due to the complex nature of eradication programs it is difficult to identify specific reasons for success and failures with *E. amylovora* eradication worldwide.

In EU countries, phytosanitary regulations for the control of *E. amylovora* in the EU include preventive and containment measures applied when isolated infection premises (foci) are

found and in infected regions, protected zones and protected sectors in infested regions according to phytosanitary guidelines (EU 2000; OEPP/EPPO 1992). Protected zones are administrative regions (or districts) where EPPs (e.g. *E. amylovora*) have never been detected. With *E. amylovora*, for instance, these zones are set up to prevent the introduction of fire blight or slow its spread into commercial orchards, nurseries and countryside. The purpose is to produce and export planting material from nurseries inside protected zones to other protected zones and outside these zones. Surveys are conducted to verify *E. amylovora* is not present in the proposed protected zones. Protected sectors are established within infested regions to produce healthy plants (e.g. fruit trees) for planting. The territory of each sector is at least 50 km^2. It consists of a disease free zone where compulsory measures are applied to maintain hosts free of diseases and a buffer zone with two sectors: a) all areas within 250 metres of the nursery boundary in which no host plants are present and b) all areas within two kilometres of the nursery boundary where official phytosanitary measures are undertaken. In Bulgaria, maintaining the protected sectors has been difficult because of the difficulty in keeping hosts free from disease within nurseries as well as in the surrounding buffer zones (Dimitrova and Andreev 2004).

In Europe, if suspected samples give a positive result for *E. amylovora*, emergency phytosanitary measures taken include establishment of a quarantine zone (250 metres) in the vicinity of the infected property (IP). Hosts susceptible to the bacterium are not allowed in this zone and movement of plants outside this zone is also prohibited. In Austria, for instance, a three to five kilometre zone is established around *E. amylovora* IPs where movement of beehives and host plants is prohibited. Permits are required for the movement of certified host plants for planting if a nursery is located within the quarantine zone.

In orchard areas, the size of a quarantine area (QA) is 500 and 1,000 metres in non-infested and infested areas, respectively (OEPP/EPPO 1992). In a quarantine area it is prohibited to establish new plant nurseries. Approved plant protection products must be used against *E. amylovora* in nurseries or orchards. Other emergency phytosanitary measures in QA include the removal and destruction of infected branches and whole trees and other host plants, and a ban on beehive movement within QA may also be implemented. Emergency measures are withdrawn if there is no further occurrence of *E. amylovora* in the following two or three growing seasons in the QA after infected plants or their parts were removed or destroyed. Surveys are also conducted in gardens, public parks, and wild host plants. In infested areas, surveys are carried out in July/August and September/October by trained people. Suspected plants are sent to laboratories for testing (EPPO Standard PM 3/40). Results are sent to the inspector and grower and if positive sent to quarantine officers. The decision to destroy infected plant material is taken on the basis of a positive lab result. All infected plants are destroyed including roots. Plants without symptoms in the vicinity of infected plants are treated with an appropriate approved product.

3.4.3.4. Fire blight incursion response in Australia

Contingency plan

A revised contingency plan for *E. amylovora* was published in 2002 (Merrimam 2002). It states that 'if identified early enough, it may be possible to eradicate or eliminate the causal bacterium from Australia'. It also states that 'if this is not possible then containment is recommended to restrict the spread of fire blight'. The first option will allow industry to avoid significant economic costs due to imposition of trade barriers linked to phytosanitary regulations. It will also prevent the expense of controlling it if established and yield losses and indirect effects on allied industries. The plan has two components: guidelines for awareness and preparedness and for response with planned methods of eradication and

containment which are implemented after an incursion. These methods include survey and diagnosis, identification and destruction of affected plants, control of vectors of *E. amylovora*, identifying and protecting unaffected areas, and follow-up monitoring. The recommendations and prescribed actions included in the revised contingency plan are intended only as a guide to assist in decision making.

Planned response actions

Response actions are described under the categories of eradication and containment following the positive diagnosis of *E. amylovora*. Eradication is the application of phytosanitary measures to 'eliminate the pest from an area which means that it can no longer be detected by recommended methods of survey and diagnosis'. Containment is 'the application of phytosanitary measures in and around an infested area to prevent spread of a pest'. The contingency plan recommends that in the event of an incursion by an economically important pest or pathogen, five steps should be implemented immediately following a positive diagnosis:

1. Survey and diagnosis of affected areas to map the extent of the disease.
2. Control strategies including roguing and destruction of affected plants.
3. Application of selected treatments for the control of the disease.
4. Monitoring to check disease status of previously affected areas after treatments have been applied.
5. Monitoring and surveillance to confirm on-going area freedom status for affected areas.

These are nationally agreed protocols which also conform to international standards for survey, diagnosis and control, and criteria for establishment of pest free area status. Recommended survey and sampling methods after detection of *E. amylovora* are described in detail in the contingency plan (Merriman 2002). In brief, two types of survey are required: for 'contact premises' in control areas which surround the outbreak site and surveys outside restricted and control areas to check for additional outbreaks which are the basis for future confirmation of pest free area status. Surveys should conform to the international standards for phytosanitary measures (ISPM No. 6, 1997; ISPM No. 4, 1995) adopted by the International Plant Protection Convention (IPPC, www.ippc.int). Target areas for survey are sites where hosts plants of *E. amylovora* can be found and include orchards, home gardens, park and public lands, including sides of roads and railways. Information on flowering patterns for host plants is also required to assist with surveys.

A trigger for the establishment of an official quarantine action is either the appearance of 'classic' symptoms of fire blight or consistent positive results from selected diagnostic tests. Diagnostic protocols are described in detail in the revised contingency plan (Merriman 2002) and in the Padil tool box (http://www.padil.gov.au/pbt). The main purpose of the diagnostic protocol is to confirm the identity of *E. amylovora* in first samples from a suspect outbreak of *E. amylovora*, and in subsequent samples from national surveys to define the extent of the outbreak.

After *E. amylovora* has been confirmed by tests, the next step is to 'advise the parties involved and if plants are flowering, advise the owners of managed hives explaining the need to quarantine them by containment or isolation and arrange for application of approved product to remove the flowers'. Organise immediate surveys by qualified plant pathologists and quarantine officers to define the extent of plants showing similar symptoms and map using GPS systems. If a suspected specimen returns a positive test (i.e. usually after four to six days) a quarantine zone of up to two kilometres or three kilometres (if plants are flowering) from the edge of the known affected area can be imposed. The survey within a

quarantine zone differ from others in that 'all hosts plants are systematically examined, including their growth stage particularly when flowering'. Also recorded are the locations of 'all managed hives and feral nests of honeybees'.

The requirements for survey beyond the restricted and control areas are described in the plan. These include inspection of all plants, application of insecticides every week for three weeks and an approved anti-bacterial treatment (e.g. antibiotic) if plants are flowering and survey of each contact premises (CP) including the sentinel plants if possible every three days for a further three weeks to confirm freedom from *E. amylovora*, which may have to be extended if weather has been unfavourable for disease development. In nurseries all hosts plants are to be inspected. In affected areas, growers will need to consider the introduction of additional measures to minimise the risk of introduction and establishment of *E. amylovora*. These are described in the plan and include regular inspection of orchards, control movement of people, and application of approved treatments. Recommendations for the treatment and removal of infected and suspected plants and actions for IPs and quarantine of managed hives and treatments of nests of feral honeybees are described in the plan.

The plan recommends a biometric approach to achieve a 99 per cent level of confidence for detecting *E. amylovora* during surveys in orchards. In urban areas and parks the plan recommends sampling strategies based on a grid system the intensity of which is determined by the quarantine status of the survey area. As demonstrated by the incursion at the RBG in Melbourne, quarantine bans will be imposed on interstate and international movement of hosts plants and fruit until confirmation of boundaries of both affected and pest free areas. The procedure to establish pest free areas are described in the plan. In Australia, the Government agencies would work with industry in the establishment and audit of pest free areas.

3.4.4. Considerations for the development of a spatial model

For a maps-based bio-economic model to be more effective in evaluating incursion response options to exotic pest threats at the orchard, locality or regional scale, such as a landscape of orchards in the Goulburn Valley of Victoria, they must have spatial (i.e. mapping) capability to properly study the likely rate of spread and economic impact. This capability is also extremely important for a more realistic evaluation of planned and alternative management options to maximise the chances of successful eradication and resources available. This in turn can allow policy decisions regarding their eradication and management to be more clearly communicated to stakeholders and better allocation of resources.

This section has identified the key biotic and abiotic factors involved in the incursion and spread of five exotic pest threats for the Australian apple and pear industry (*E. amylovora*, *N. ditissima, B. dorsalis, D. plantagine* and *Varroa destructor*) to be considered when developing a bio-economic model to assess spread and management strategies for their incursion (see Table 7, below). It also reviewed the phytosanitary measures used for eradication and containment using *E. amylovora* and Queensland fruit fly as case studies, which can be included in a model to assess different incursion management responses.

Climatic conditions in the Goulburn Valley and other regions of Victoria, where susceptible fruit hosts and alternative hosts occur, are suitable for both *E. amylovora* and *N. ditissima* pathogens to establish (Edwards *et al.* 2006). In a region where pome fruit orchards with highly susceptible hosts such as pears are densely located such as the Goulburn Valley, the changes of eradicating European canker, if acted quickly, may be greater than for *E. amylovora* due to *N. ditissima* potentially lower dispersal range (conidia and ascospores)

compared to the bacteria *E. amylovora* which can be dispersed locally by bees up to three kilometres (see Table 7).

In addition, *N. ditissima*, a wood parasite may be eradicated with drastic pruning as demonstrated in Tasmania compared to *E. amylovora*, a systemic pathogen, which requires complete removal of infected hosts. The first outbreak of fire blight was successfully eradicated from the Royal Botanic Gardens of Melbourne, Australia. This was probably achieved because it occurred outside a fruit-growing area or nursery. However, it was estimated that this incursion cost approximately $20 million in quarantine and eradication expenses and lost revenue to the Australian pome and nursery industries.

E. amylovora has been successfully contained in Norway also because it occurred outside a fruit-growing region and the implementation of strict phytosanitary regulations has limited its spread. In other EU countries, however, despite the implementation of phytosanitary and eradication measures, *E. amylovora* was not eradicated. Unsuccessful eradication or containment has been attributed to many factors including the presence of a wide range of hosts, difficulty in detecting the disease during surveys due to unfavourable weather conditions for disease development and inability to restrict the movement of vectors such as bees. Information on the locations of alternative hosts and native bees would be needed to properly assess incursion management responses in the Goulburn Valley, Victoria.

Several strategies have been recommended and published in the fire blight contingency plan to attempt eradicate or contain the disease if an incursion occurs again in Australia. The preparedness and response strategies recommended have not been properly evaluated under different simulated scenarios to determine their effectiveness. This evaluation is extremely important considering that a rapid response to an exotic pest incursion is required in order to limit the extent of the incursion and to maximise the changes of successful eradication.

This is also extremely important in the event of an incursion by an exotic pest that has the ability to disperse over a large area such as the oriental fruit fly (*B. dorsalis)* and the Queensland fruit fly requiring a wider quarantine zone (25 kilometre) to that suggested for *E. amylovora*. The Queensland fruit fly has the ability to produce many generations in a year, if conditions for reproduction are right and susceptible hosts are available in rural and urban areas. In contrast, the exotic pest Oriental fruit fly survives in alternative hosts such as guava trees in addition to mango its main host and other hosts in places like India and could thus survive and establish well in many sub-tropical and tropical regions of Australia where these hosts occur. More information is required to determine the presence and distribution of alternative hosts for this pest in the Goulburn Valley to allow proper assessment of incursion response for this pest. On the other hand, the Rosy apple aphid will be able to establish and cause damage to bloom return (i.e. fruit setting) in the Goulburn Valley as this pest uses the weedy host *Plantago* spp. as an alternative host.

Varroa mite is capable of significantly reducing the population of bees within a two to three year period without treatment. An incursion of Varroa mite will have a direct economic impact on the bee honey industry and on pollination services for the pome fruit industry in areas where populations of native bees may not be sufficient for pollination of fruit trees and this will affect overall productivity. Evaluation of eradication strategies for Varroa mite with a bio-economic model would require modelling the dispersal of this mite by natural movement of European bees and its effect on bee populations and pollination. For this, accurate information on the location of domestic and feral hives and their contribution to pollination will be required.

The direct costs under 'eradication or containment' and 'live with it' scenarios for incursions by these five exotic pest threats in the Goulburn Valley will be different. For instance, drastic pruning should be considered for European canker whereas *E. amylovora*, as systemic pathogen, would require complete removal of trees. Treatment of fruit will be also different for diseases and pests.

Table 7. Key biotic and abiotic factors involved in the spread and incidence of five exotic pest threats for the Australian apple and pear industry and their potential productivity impact.

EPP	Incursion time[1]	Agent[2]	Host entry point[3]	Short dispersal[4]	Long (local) dispersal[5]	Incidence [6]	Range yield reduction[7]
E. amylovora	Bloom (early Sep to mid October)	bacteria	flower infection	bees, insects, rain, pruning etc	bees (2-3 km)	weather dependent (temp. and rain)	5% – 25% gross margin reduction due to loss trees or branches
N. ditissima	autumn-winter (leaf fall and pruning)	spores	leaf scars and pruning	conidia	ascospores (100 – 500m)	weather dependent (temp. and moisture duration)	5% – 25% gross margin reduction due to loss of branches
D. plantaginea	Bloom and vegetative stages	aphids	flowers and leaves	wingless aphids	winged aphids (100 - 1000 m)	population dynamics influenced by biotic and abiotic factors	decrease bloom and fruit setting by 30% - 50%
B. dorsalis	Fruit development	flies	fruit	flies	flies (1 -5 km)	population dynamics influenced by biotic and abiotic factors	Yield reduction at harvest up to 80%
V. destructor	N/A	Mite+bee	affects pollination over time	Varroa-infested bee	Varroa-infested bee (2-3 km during flowering)	depends on host-parasite interactions	5% – 25% gross margin reduction due to pollination

[1] Crop stage when host is most susceptible or incursion most likely to be detected (e.g. during monitoring of other insects); others discussed in review.

[2] Agent or vector spreading the disease and insect pest or parasite. For *B. dorsalis* development from egg to adult assumed to be 16 days.

[3] Other discussed in review. For Varroa mite, orchardist use 1-3 bee hives per ha in areas where populations of native bee low.

[4] Spread only within and between trees, others discussed in review. A range of biotic and abiotic factors influence inoculum production of pathogens and population dynamics of insect/mite pests and this will affect the rate of spread.

[5] Spread between fruit blocks and orchards, others discussed in review. A range of biotic and abiotic factors influence inoculum production of pathogens and population dynamics of insect/mite pests and this will affect the rate of spread. It is assumed that when wigless aphids reach a high population they can develop into winged aphids capable of spreading from block to block like the Oriental fruit moth and Codling moth. It is also assumed that adult flies are capable of flying up to 4-5 km like the Qld fruit fly. Managed bees are known to travel up to 2 km to collect pollen and 3 km if food sources are limited.

[6] Yearly conditions for disease/pest outbreaks variable; bacteria and spore production and thus disease spread influenced by temperature and moisture so in years with high disease pressure potential for spread is greater. Population dynamics of *B. dorsalis* and *D. plantaginea* affected by biotic and abiotic factors; Varroa mite by host parasite interactions.

[7] Potential loss of productivity without control.

3.5. The non-Spatial Model used to Assess Threats to the Banana Industry

3.5.1. Introduction

Comprehensive bioeconomic decision support frameworks are increasingly needed to assist the banana industry to manage present and future biosecurity risks. Benefit cost analysis is a highly effective means of communicating expected net returns from investment decisions to diverse groups of stakeholders. For biosecurity economists, it can provide a valuable means to convey a raft of technical economic and scientific information via metrics that are easily understood by risk managers. Given the needs of the banana industry and the aims of the CRC10162 project, we adopted a flexible analytical approach that serviced the demands of our client industries in the most appropriate manner. In the case of the banana industry, this meant revisiting non-spatial statistical models developed as part of CRC10010 (*Enhanced Risk Analysis Tools*) project.

Bananas are an important crop throughout the world, particularly in developing countries where their importance as a food crop is only surpassed by rice, wheat and maize (Food and Agriculture Organization 2010; Henderson *et al.* 2006; Heslop-Harrison and Schwarzacher 2007). More than 120 countries produce bananas, with world production estimated to be in excess of 100 million tonnes (Food and Agriculture Organization 2010). Australia contributes less than 0.5 per cent of global production (Food and Agriculture Organization 2010), but banana cultivation makes a sizeable contribution to regional economies across northern Australia. In 2010, the States of Queensland, New South Wales, the Northern Territory and Western Australia produced a combined total of 301,450 tonnes of bananas with a gross value of $492.2 million (ABS 2011).

All commercially grown cultivars of banana have evolved as a result of intra-specific and inter-specific hybridisation, parthenocarpy and triploidy, involving the two wild diploid species *Musa acuminata* and *Musa balbisiana* (Henderson, Pattemore *et al.* 2006; Simmonds 1966). Selection of high-yielding *Musa* clones and current agronomic practices in large-scale monoculture plantations has given rise to the occurrence of a wide range of pests and diseases (Henderson, Pattemore *et al.* 2006; Ploetz 2000).

Of these, the CRC10162 project team was asked to apply statistical impact simulation models to five of current concern to the Australian banana industry. These included:

1. Banana bunchy top virus (*Babuvirus*, Nanoviridae)
2. Black Sigatoka (*Mycosphaerella fijiensis* (Morelet))
3. Moko Disease (*Ralstonia solanacearum* – race 2)
4. Panama Disease (*Fusarium oxysporum* f.sp. *cubense*)
5. Yellow Sigatoka (*Mycosphaerella musicola*)

The specific information requested by the industry regarding these diseases is different for each. Therefore, to coherently report the analytical methodologies refined and applied to separate pathogens requires them to be grouped separately in the following sections. Hence, the reporting structure differs from the apple and pear biosecurity threats reported in previous sections. Each case study is written up as a separate analysis. This means that there is some repetition of information, particularly regarding methods.

3.5.2. An assessment of the benefits of banana bunchy top virus exclusion from commercial banana plantations in Australia

3.5.2.1. Introduction

Banana bunchy top disease caused by banana bunchy top virus (BBTV) (*Babuvirus*, Nanoviridae) is one of the most economically important diseases of bananas in many production regions, including Asia, Africa and the South Pacific (Dale 1987; Hooks *et al.* 2008). It causes stunted growth and infected plants rarely produce a bunch (Dale 1987). BBTV is transmitted by the banana aphid (*Pentalonia nigronervosa*), as well as through infected plant suckers and other plant tissues used in banana propagation (Hooks, Wright *et al.* 2008; Magee 1927). See Appendix 1 for detailed information on the virus.

BBTV has been present in eastern Australia since the early 1900s. Its severity was clearly demonstrated in the 1920s when approximately 90 per cent of the Queensland and New South Wales banana crops were destroyed (CABI/EPPO 2003). This prompted State government initiatives to contain BBTV through eradication of infected plants and controls on the movement of planting material from affected areas, which led to a gradual recovery of the banana industry. In 1993, a five-year Banana Plant Health Improvement Project was initiated by the industry aimed at eradicating BBTV from Australia (Thomas and Dietzgen 1996). Despite achieving substantial reductions in the prevalence of the virus, outright eradication was not achieved by the end of this period.

In this section, we examine a similar policy termed *exclusion*, which aims to remove the disease from banana producing regions and maintain their area freedom from the virus over time. We use computer-simulated economic impact scenarios to determine the likely net benefits of BBTV exclusion from commercial banana production areas. We use a partial budgeting approach in conjunction with a stratified diffusion model to estimate BBTV prevalence and control responses under a nil management and a commercial exclusion scenario over time. We then compare these scenarios and calculate a likely financial return to the banana industry from adopting an exclusion strategy.

3.5.2.2. Methods

We assume that the current presence of BBTV is eliminated from Australia commercial banana plantations and concentrate on events that might subsequently transpire. As such, we treat local eradication of future incursions in banana growing areas as an investment alternative to a nil management approach with respect to BBTV management. We assume that the Australian banana industry is represented by a single planning body (i.e. the ABGC, http://www.abgc.org.au/) determining appropriate biosecurity investment strategies. Predicted investment paths are defined as a function of expected yield and input cost changes (and hence profitability) from investing in BBTV exclusion relative to a nil management approach.

We make the assumption that the planning body will choose to invest in BBTV exclusion in region (i.e. State or Territory) *i* in time step (i.e. year) *t* if it is expected to reduce grower losses by a greater amount than additional costs. The dichotomous adoption variable, α_t, which takes on the value of one if the central planner invests in exclusion across *n* regions in year *t* and zero otherwise, is defined as:

$$\alpha_t = \begin{Bmatrix} 1 \text{ if } \sum_{i=1}^{n} d_{it} \geq \sum_{i=1}^{n} c_{it} \\ 0 \text{ if } \sum_{i=1}^{n} d_{it} < \sum_{i=1}^{n} c_{it} \end{Bmatrix} \quad (1)$$

where d_{it} is the total difference in predicted cost increments induced by BBTV between the exclusion and nil management policy options in region *i* in time *t*, and c_{it} is the total cost of implementing an exclusion strategy in region *i* in time *t*. We focus on the estimation of $\sum_{i=1}^{n} d_{it}$ to determine how large $\sum_{i=1}^{n} c_{it}$ would need to be before α_t assumes a value of zero.

The current pre-border biosecurity strategy for addressing the threat of exotic banana pathogens includes the use of strict phytosanitary measures on traded bananas, which lower the probability of BBTV re-entering an area after via imported fruit. Indeed, these measures are so strict that they effectively mean prominent banana exporting countries such as the Philippines cannot land product in Australia at a sufficiently low price to be competitive on the domestic market for fresh bananas. Post-border biosecurity measures include monitoring through disease surveillance, robust detection and rapid response to incursions.

If, as a result of these post-border measures, a BBTV incursion in a commercial banana production area is detected early enough, there may be a strong likelihood of local eradication through plant removal and destruction. Hence, the value of d_{it} is influenced by local eradication costs and probability of eradication success. This probability of success is assumed to decline negative exponentially at a rate of $e^{0.15A_{it}}$, where A_{it} is the area infected with BBTV in region *i* year *t* weighted by the probability of infection and density of infection. If an outbreak is not detected early enough, a longer term management strategy is required to minimise BBTV impacts using insect control technologies and lethal chemical treatments for infected plants.

Algebraically, we expressed d_t as:

$$d_{it} = \begin{Bmatrix} E_{it}A_{it} \text{ if } A_{it} \leq A_{it}^{\text{erad}} \\ Y_{it}P_tA_{it} + V_{it}A_{it} \text{ if } A_{it} > A_{it}^{\text{erad}} \end{Bmatrix} \quad (2)$$

where: E_{it} is the cost of eradication per hectare in region *i* in year *t*; A_{it}, as stated above, is the area infected with BBTV in region *i* year *t* weighted by the probability of infection and density of infection; A_{it}^{erad} is the maximum technically feasible area of eradication in region *i* in year *t*; Y_{it} is the mean change in yield resulting from the control of insect vectors and treatment of infected plants in region *i* in year *t*; P_t is the prevailing domestic price for bananas in year *t*; and V_{it} is the increase in variable cost of production per hectare induced by BBTV on-plantation management methods in region *i* in year *t*.

A_{it} is inclusive of BBTV re-entry and establishment probabilities (denoted p^{ent} and p^{est}, respectively), and therefore represents the area predicted to be in need of additional management effort (i.e. beyond normal plantation management activities) due to BBTV infection in region *i* in year *t*. A Markov chain process, described in Hinchy and Fisher (1991), is used to change p^{ent} and p^{est} over time according to a vector of transitional probabilities. These transitional probabilities describe the likelihood of moving from one virus state to another. p^{ent} and p^{est} are combined to form a probability of invasion, p_i:

$$p_i = p^{\text{ent}} \times p^{\text{est}} \text{ where } 0 < p_i < 1. \quad (3)$$

To describe the movement of BBTV post-establishment in multiple regions we use a stratified diffusion model combining both short and long distance dispersal processes (Hengeveld 1989). It is derived from the reaction diffusion models originally developed by Fisher (1937) which have been shown to provide a reasonable approximation of the spread of a diverse range of organisms (Cook *et al.* 2011b; Dwyer 1992; Holmes 1993; McCann *et al.* 2000; Okubo and Levin 2002). These models assert that an invasion diffusing from a point source will eventually reach a constant asymptotic radial spread rate of $2\sqrt{r_i D_{ij}}$ in all directions, where r_i describes a growth factor for BBTV per year in region *i* (assumed constant over all infected sites) and D_{ij} is a diffusion coefficient for an infected site *j* in region *i* (assumed constant over time) (Cook, Carrasco *et al.* 2011b; Hengeweld 1989; Lewis 1997; Shigesada and Kawasaki 1997). Hence, we assume that the original infection (i.e. the first of a probable series of sites, *j*) takes place in a homogenous environment in region *i* and expands by a diffusive process such that area infected at time *t*, a_{ijt}, can be predicted by:

$$a_{ijt} = p_i\left[\pi\left(2t\sqrt{r_i D_{ij}}\right)^2\right] = p_i\left(4D_{ij}\pi r_i t^2\right). \quad (4)$$

For practical purposes, an estimate of D_{ij} can be derived from the mean dispersal distance ($\bar{\delta}_{ij}$) of the pathogen at an infection site, where $D_{ij} = \frac{2\left(\bar{\delta}_{ij}\right)^2}{\pi t}$ (Andow *et al.* 1990; Cook *et al.* 2010a; Cook *et al.* 2011c). $\bar{\delta}_{ij}$ is the site-specific average distance (in metres) over which dispersal events leading to infection occur. By assuming D_{ij} is constant across all sites *j* we ignore demographic stochasticity and consequent non-uniform invasion (Cook, Fraser *et al.* 2011c).

The density of BBTV infection within a_{ijt} influences the control measures required to counter the effects of infection, and thus partially determines the value of A_{it}. We assume that in each site *j* in region *i* affected, the infection density, N_{ijt}, grows over time period *t* following a logistic growth curve until the carrying capacity of the environment, K_{ij}, is reached:

$$N_{ijt} = \frac{K_{ij} N_{ij}^{\min} e^{r_i t}}{K_{ij} + N_{ij}^{\min}(e^{r_i t} - 1)}. \quad (5)$$

Here, $N_{ij}^{\min}$ is the size of the original influx at site *j* in region *i* and r_i is the intrinsic rate of density increase in region *i* (assumed to be the same as the intrinsic rate of population increase) (Cook, Fraser *et al.* 2011c).

In addition to a_{ijt} and N_{ijt}, the size of A_{it} depends on the number of nascent foci (see Moody and Mack (1988) – these are *satellite* infection sites) in year *t*, s_{it}, which can take on a maximum value of $s_i^{\max}$ in any year. These sites result from events external to the outbreak itself, such as weather phenomena, animal or human behaviour, which periodically jump the expanding infection beyond the infection front (Cook, Fraser *et al.* 2011c). We use a logistic equation to generate changes in s_{it} as an outbreak continues:

$$s_{it} = \frac{s_i^{\max} s_i^{\min} e^{\mu_i t}}{s_i^{\max} + s_i^{\min}(e^{\mu_i t} - 1)} \quad (6)$$

where μ_i is the intrinsic rate of new foci generation in region *i* (assumed constant over time) and $s_i^{\min}$ is the minimum number of satellite sites generated in region *i*.

Given equations (4)-(6), we can express A_{it} as:

$$A_{it} = \sum_{j=1}^{m} \left(a_{ijt} N_{ijt}\right)^{s_{it}} \text{ where } 0 \le A_{it} \le A_i^{\max}. \quad (7)$$

The total benefit to the central planner of adopting an exclusion policy for BBTV in year *t*, B_t^{BBTV}, can be expressed as:

$$B_t^{\text{BBTV}} = \sum_{i=1}^{n} d_{it} \alpha_t. \quad (8)$$

In the following section we estimate $\sum_{i=1}^{n} d_{it}$ using multiple BBTV re-entry and spread scenarios for Australia's banana growing regions over a 30-year period. These include grower areas of coastal Queensland, the north coast of New South Wales, parts of Western Australia and the Northern Territory (i.e. n = 4) (see Table 1). Where there is uncertainty surrounding parameter values, they are specified within the model as distributions and a Latin hypercube sampling algorithm used to sample from each distribution. In each of 10,000 model iterations one value is sampled from the cumulative distribution function so that sampled parameter values are weighted according to their probability of occurrence. The model calculations are then performed using this set of parameters.

Table 8 provides banana production information for each region used in the analysis. It also contains region-specific BBTV (re-)entry and (re-)establishment probabilities. Given

the continued stringent SPS measures against imported bananas, the probability of entry into new areas beyond the historical distribution of BBTV (i.e. Northern Territory and Western Australia) is regarded as very low: within the range 1.0×10^{-3} to 5.0×10^{-2} (Cook 2003). In areas where the virus has been present (i.e. Queensland and New South Wales), the likelihood of re-entry was arbitrarily assumed to be low: within the range of 5.0×10^{-2} to 0.3. The probability of establishment upon entry was assumed to be moderate in all regions: within the range of 0.3 to 0.7 (Cook 2003).

Table 8. Australian banana production statistics by region.

State	Area (ha) [a]	Production volume (MT) [a]	Average yield (T/ha) [a]	Value produced ($'000,000) [a]	Probability of entry, p^{ent}
Queensland	10,083	279.09	27.68	448.3	Uniform(0.3,0.7)
New South Wales	1,057	10.75	10.17	17.7	Uniform(0.3,0.7)
Western Australia	200	5.64	28.19	15.1	Uniform(1.0×10^{-6}, 1.0×10^{-3})
Northern Territory	203	5.98	29.46	11.1	Uniform(1.0×10^{-6}, 1.0×10^{-3})

[a] ABS (ABS 2011).

Details of all other parameter distributions appear in Table 9, below. Note that *i*, *j* and *t* subscripts are omitted in Tables 8 and 9 since, with the exception of p^{ent} and insecticide and application cost, parameter specification does not change over spatial or temporal ranges. Table notes provide details where a spatial variation is assumed.

Table 9. Parameter values for the BBTV model.

Parameters	Nil management
Probability of establishment, p^{est}. [a]	2.6×10^{-4} to 1.3×10^{-1}
Detection probability.	Binomial(1.0, 0.6)
Probability of successful eradication in a single time step given an infected area, A.	$e^{0.15\times A}$
Population diffusion coefficient, D (m^2/yr). [a,b]	Pert(0,2.5×10^3, 5.0×10^3)
Minimum area infected immediately upon entry, A^{min} (m^2).	1.0×10^3
Maximum area infected, A^{max} (m^2). [c]	1.2×10^8
Intrinsic rate of infection and density increase, r(yr^{-1}). [a]	Pert(0.10,0.15,0.20)
Minimum infection density, N^{min} (#/m^2).	1.0×10^{-4}
Maximum infection density, K (#/m^2). [a]	Pert(100,550,1000)
Minimum number of satellite sites generated in a single time step, S^{min} (#).	0
Maximum number of satellite sites generated in a single time step, S^{max} (#). [a]	Pert(10,5,10)
Intrinsic rate of new foci generation per unit area of infection, μ (#/m^2). [a]	Pert(1.0×10^{-6},3.0×10^{-6},5.0×10^{-6})
Discount rate (%).	5
Supply elasticity. [d]	Uniform(0.2,0.8)
Demand elasticity. [d]	Uniform(-1.1,-1.0)
Prevailing market price for bananas in the first time step ($/T). [c]	1,900
Maximum area considered for eradication (ha).	400
Cost of eradication, E ($/ha). [e]	Pert(1.0×10^4,1.5×10^4,2.0×10^4)
Increased insecticide and application cost ($/ha). [f]	130
Yield reduction despite control, Y (%).	Pert(0.0,2.5,5.0)

[a] Specified with reference to Cook (2003) and Waage *et al.* (2005) using distributions defined in Biosecurity Australia (2001); [b] Derived from Sapoukhina *et al.* (2010); [c] ABS (2011), Note 1ha = 10 000m^2 ; [d] Ulubasoglu *et al.* (2011); [e] Assumes zero compensation following banana plant removal, average density of planting of 2 000 stems/ha and removal, transport, destruction and chemical costs amounting to $20 per tree. This is inclusive of labour (team of three at $50/hr per person), bulldozing equipment ($100/hr at 20 hours per hectare), truck hire ($75/hr), incendiaries ($60/ha for green waste) and creation of a circular chemical buffer zone approximately 5 hectares in diameter around previously infected sites. Chemical used is assumed to be dithane (applied at a rate of 3kg/ha or $25/ha) and oil (applied at 3L/ha or $10/ha) at fortnightly intervals rotated with propiconazole (applied at a rate of 0.3L/ha or $5/ha). Assume 2 additional dithane treatments are required and 4 propiconazole treatments (and therefore 6 additional oil treatments), each taking 1 hour per hectare to apply; [f] Assumes: (i) labour costs of $50/ha (i.e. 1 application × 1hr/ha × $50/hr); (ii) 75mL of chemical solution is used per banana plant per treatment costing $10 per litre (e.g. dimethoate diluted to 75mL/100L (Cook 2003)) (i.e. approx. $15/ha); and (iii) two additional chemical treatments will provide sufficient suppression of banana aphid.

3.5.2.3. Results

Despite exclusion from commercial production areas being assumed to have been achieved at the outset of the analysis, our assumptions are such that re-establishment is likely to occur at some point or multiple points over the estimation period. The model simulates these re-establishment events as a Poisson process where BBTV successfully re-establishes in Queensland and New South Wales on an average of one year in six, and in Western Australia and the Northern Territory one year in 50. Therefore, the resultant expected spread area values under the exclusion and nil management scenarios calculated from the 10,000 iterations of the model are positive. However, as Figure 15 reveals, the extent of expected spread under an exclusion or active containment program is substantially below that of a nil management policy. These projections have been aggregated across all production regions to produce Figure 15.

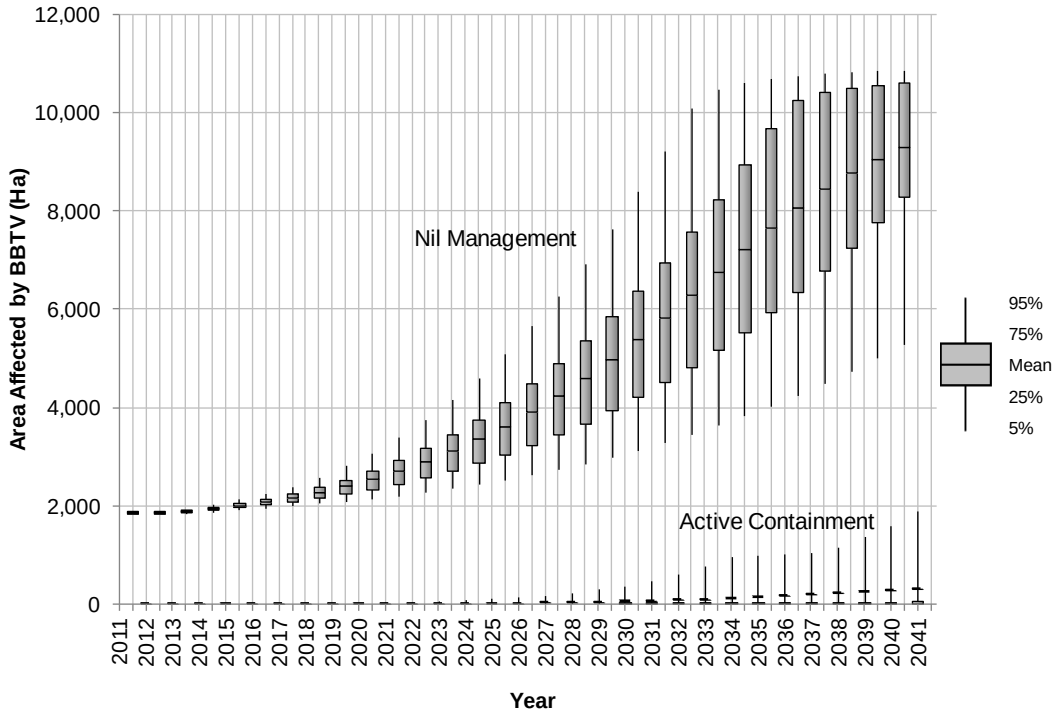

Figure 15. Likely spread of BBTV over time with and without an active containment policy.

The present value of benefits accruing from the exclusion of BBTV from commercial plantations is estimated by the model to average $18.9 million per year over 20 years across banana producing regions (i.e. $\sum_{i=1}^{n} d_{it} = \$1.89 \times 10^{7}$). Recall from equations (1) and (8), this represents the threshold level of $\sum_{i=1}^{n} c_{it}$ beyond which the central planning body will choose not to invest in an exclusion strategy as an alternative to a nil management strategy (i.e. $\alpha_t = 0$). The standard deviation of the distribution of average annual

biosecurity benefits is $3.5 million and skewness -1.8 (i.e. the distribution is skewed left such that the left tail is long compared to the right tail).

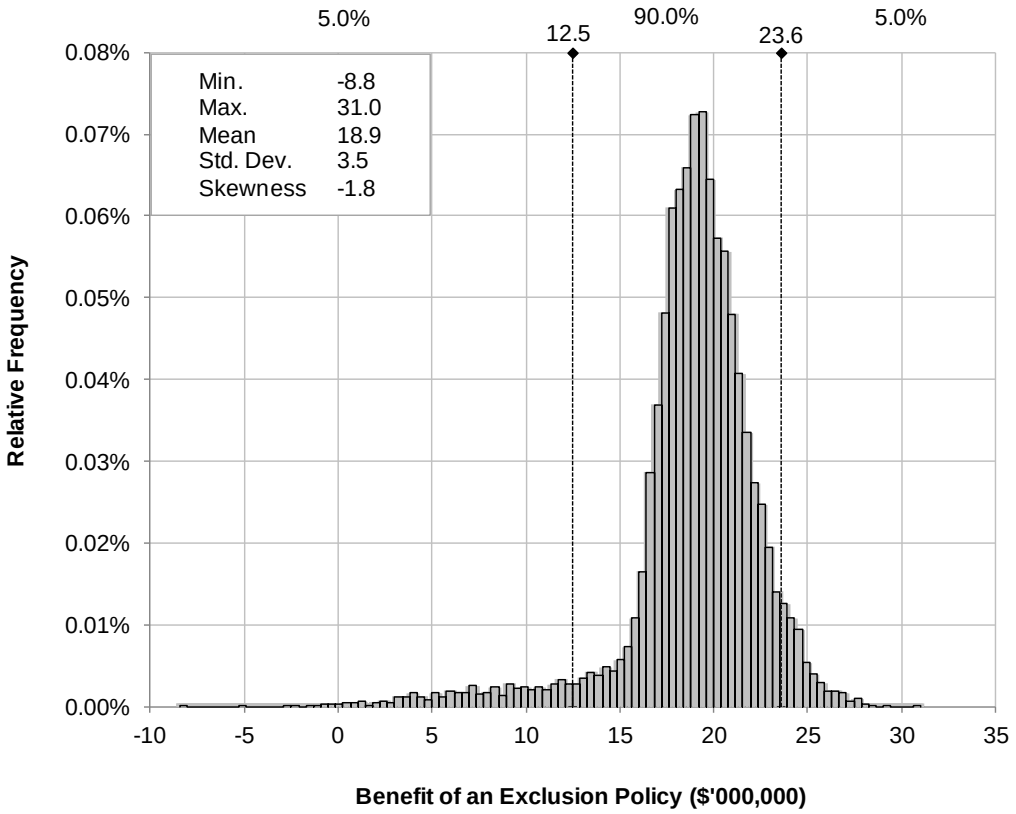

Figure 16. Expected annual benefit of a BBTV exclusion policy over 20 years.

Given current average banana yields, our estimated value of $\sum_{i=1}^{n} d_{it}$ is equivalent to an annual avoidance of losses in national banana production harvest volume of 11.6 thousand tonnes per year. While Figure 16 shows benefits over a 20-year period, Figure 17 illustrates how these annual exclusion benefits are expected to change over time as the expected difference in BBTV prevalence between the exclusion and nil management scenarios increases the further into the future we project. Here, the mean benefit of BBTV exclusion predicted by the model is plotted with 10 per cent and 50 per cent confidence intervals. All projected benefits are discounted at 5 per cent per annum.

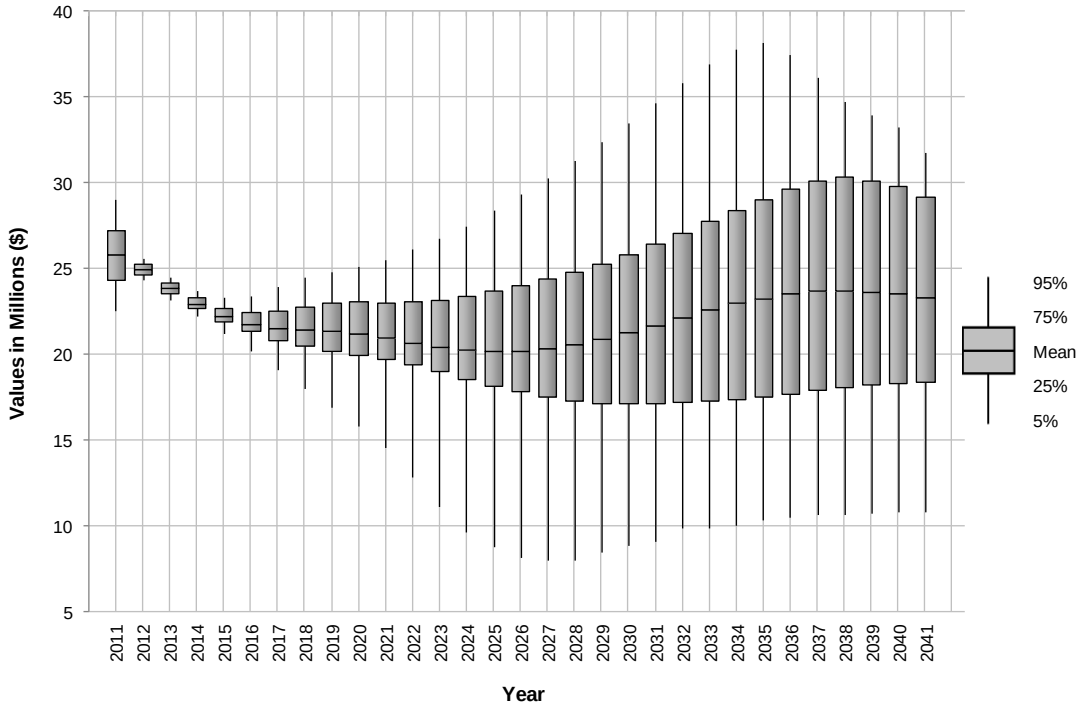

Figure 17. Expected annual benefit of BBTV exclusion over time.

In view of the uncertainty surrounding many of the parameters used to describe the BBTV (re)infection and spread process, the sensitivity of the change in expected exclusion benefits to the key assumptions of the model must be tested. Parameters were sampled from a uniform distribution with a maximum (minimum) of +50 per cent (-50 per cent) of the original values entered in to the model using Monte Carlo simulation. The Spearman's rank correlation coefficients relating the sampled model parameter values and the change in $\sum_{i=1}^{n} d_{it}$ were then calculated. The results are presented in Figure 18.

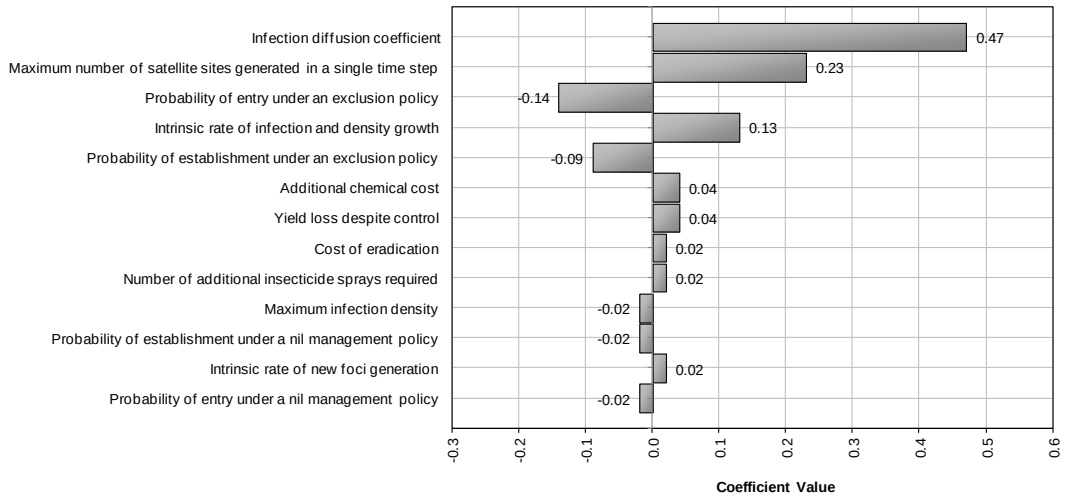

Figure 18. Sensitivity analysis for the BBTV model.

The sensitivity tests indicate that the model is highly responsive to changes in five of the parameters listed in Tables 8 and 9 (13 of which are shown in Figure 18). These parameters and their correlation with predicted $\sum_{i=1}^{n} d_{it}$ are the infection diffusion coefficient (0.47), the maximum number of satellite sites generated in a single time step (0.23), the probability of entry under an exclusion policy (-0.14), the intrinsic rate of infection and density growth (0.13) and the probability of establishment under an exclusion policy (-0.09).

To indicate how high the probability of BBTV entry and establishment under an exclusion strategy must be to produce a result where the central planner is indifferent between the exclusion and nil management options (i.e. $\sum_{i=1}^{n} d_{it} = \$0$) requires the model to be aggregated across all States and Territories. If we consider the sum of all banana growing areas in Australia as one susceptible host block, the probability of BBTV entry and establishment under an exclusion strategy that would lead to expected costs in both policy scenarios to be equivalent is approximately 0.75. This requires a re-entry and establishment event to occur in a commercial plantation in three of every four years.

3.5.2.4. Discussion

Our results are indicative of the potentially large benefits of investing in active containment of BBTV. Based on the model outlined in the Methods section, it is shown in the Results section that excluding the virus from commercial production areas is likely to produce a net benefit over time provided the annual costs of doing so do not exceed $18.9 million.

The sensitivity analysis reveals a high sensitivity of this result to changes in several biological parameters that can be influenced by post-border biosecurity policies. Indeed, four of the five most sensitive model parameters fall into this category, including the infection diffusion coefficient, the maximum number of satellite sites generated in a single time step, the intrinsic rate of infection and density growth and the probability of establishment under an active containment strategy aimed to exclude BBTV from production areas. Strategies that encourage plantation monitoring and disclosure of detection information could have the effect of lowering each of these parameters, thus increasing the likely returns of an active containment strategy over time.

The cost of achieving complete BBTV exclusion from commercial banana growing regions is not known, but the eradication of the fungal pathogen black Sigatoka (*M. fijiensis* (Morelet)) from north Queensland between 2001 and 2003 provides at least some indication of what the possible BBTV exclusion cost might be. *M. fijiensis* was detected in 2001 in the Tully area, the major banana-growing region of Australia. Although past detections of the fungus in far north Queensland were eradicated with similar tactics to those we have suggested for local BBTV eradication (i.e. destruction of infected plants), a programme of intensive de-leafing was employed to remove the majority of inoculum from plants in the Tully outbreak (Peterson *et al.* 2005; Sosnowski *et al.* 2009). This was followed by intensive fungicide treatment applied to plants weekly in rotation for a period of 6 months after de-leafing. In total, the eradication cost was A$17 million (Sosnowski, Fletcher *et al.* 2009).

If this figure can be considered broadly representative of a relatively small scale eradication program, let us hypothetically assume that the exclusion of BBTV might involve

a cost more than three times this amount. Even if exclusion costs from commercial production areas are as high as $60 million and it takes a full five years to remove the virus completely, our results indicate that returns to the industry would be highly favourable. A benefit cost analysis performed using our estimated value would produce a benefit cost ratio of 1.6:1.0 (i.e. every $1.00 spent on eradicating the disease returns $1.60 worth of benefit to the industry). It is possible, indeed likely, that exclusion of BBTV from the main production areas can be achieved at substantially lower cost. If this is the case and exclusion is achieved, the returns on investment will be significantly higher.

Future extension of the model developed in this section could include the consideration of flow-on effects of BBTV to the regional and national economies using a general equilibrium model (Wittwer *et al.* 2005). While the importance of potential costs of non-market (e.g. environmental costs due to the use of pesticides) and indirect market impacts (e.g. reduced purchases of inputs after an industry is affected by an invasive species) of BBTV are acknowledged, they have not been included in the model due to high levels of uncertainty in the data. If the environmental costs of the use of, for instance, pesticides to control BBTV insect vectors were to be included, the benefits of exclusion over time would probably increase. Using a general equilibrium model or using an ecosystem services approach may improve the investigative power of the analysis, but would impose a cost in terms of the increased need for information to run the models effectively.

3.5.3. An assessment of the potential economic impact of black Sigatoka on the Australian banana industry

3.5.3.1. Introduction

Risk perception is difficult to reconcile, particularly in relation to invasive species incursion events. The Australian banana industry has often been portrayed as having a pessimistic view of risk related to pests and diseases potentially introduced by trade leading to the imposition of highly-restrictive phytosanitary measures on imports, to the detriment of domestic banana consumers (James and Anderson 1998; Javelosa and Schmitz 2006). But, comparatively few studies have focused on the pests and diseases of concern to the industry and how the welfare of producers is likely to change over time as incursions spread across growing regions. When the dynamics of possible incursions have been considered, the maintenance of trade barriers has been shown to be justified, at least in the short term, to allow domestic banana producers time to adjust to post-trade production environment (Leroux and Maclaren 2011). However, to date a comprehensive epidemiological model has not been used to support or refute these findings.

In Australia, the States of Queensland, New South Wales, the Northern Territory and Western Australia currently produce over 300,000 tonnes of bananas per year with a gross value of over $490 million (ABS 2011). As Table 8 shows, Queensland contributes by far the largest share of production. In 2001, the harmful disease black Sigatoka (caused by the fungus *Mycosphaerella fijiensis*) was detected in the Tully area of Queensland, the most prominent banana-growing region in Australia. This outbreak was successfully eradicated by the removal and destruction of infected plants and a program of intensive de-leafing to remove the majority of inoculum from plants in the Tully outbreak (Peterson, Grice *et al.* 2005; Sosnowski, Fletcher *et al.* 2009). This was followed up by intensive fungicide treatments applied to plants in the affected area weekly in rotation for a period of six months after de-leafing. In total, the eradication cost of this relatively small outbreak was Aus$17 million (Sosnowski, Fletcher *et al.* 2009).

Had the Tully outbreak not been eradicated, *M. fijiensis* would have presented growers with a major challenge, as indeed it has throughout the banana-growing world. If the disease is not controlled, it causes premature death of large areas of the plant's leaf surface resulting in reduced photosynthetic area. In severe cases fruit does not mature at all, while in less severe outbreaks the size of bunches and individual fingers are reduced (Marín *et al.* 2003). In addition, fruit has a tendency to ripen prematurely, has an abnormal flavour or smell and is prone to chilling injury during transport and ripening (Marín, Romero *et al.* 2003; Mobambo *et al.* 1993).

The closely-related disease yellow Sigatoka (*M. musicola*) is naturalised and actively controlled in Australia through intensive fungicide treatments and diseased leaf removal (Henderson, Pattemore *et al.* 2006). If *M. fijiensis* were to also be introduced to major banana plantations in the future and eradication was not successful, the frequency of chemical treatments and de-leafing are expected to increase substantially, causing a severe contraction of Australia's domestic banana supply.

Until the Philippines requested formal access to the Australian market for fresh bananas in 2000, Australia maintained a ban on banana imports to guard against pests and diseases like *M. fijiensis* from entering the country on imported fruit. The resultant import risk analysis finalised and released by Biosecurity Australia some eight years later found *M. fijiensis* (by then eradicated from the Tully region), was the most serious of 21 exotic pests and diseases associated with bananas present in the Philippines. It concluded that

this disease presented a level of biosecurity risk beyond Australia's acceptable level of protection (ALOP), necessitating that any imports should satisfy a very strict set of pre-export procedures (Biosecurity Australia 2008). The ALOP is a locus of arrival probabilities and incursion impacts with a unique product representing the maximum tolerable level of biosecurity risk associated with imports before a refusal is made to a market entry request.

The requirements placed on prospective banana imports following the request from the Philippines effectively meant foreign producers could not land fruit in Australia below a domestic market equilibrium price (Leroux and Maclaren 2011). Probabilistically, this does not mean that the risk of importing a disease like *M. fijiensis* was reduced to zero. But, it was reduced to a very low level consistent with the country's ALOP.

In this section, we construct a dynamic partial equilibrium model to estimate what this ALOP means in terms of potential consequences for the Australia banana industry over time, and how this differs from an unrestricted trade risk. The model estimates likely shifts in the domestic supply function for bananas in different time periods via a stratified diffusion model of disease spread. In presenting our findings, we address some of the ambiguity inherent in the use of the ALOP as a metric for evaluating trade decisions.

3.5.3.2. Methods

Assume the Australian domestic market for the bananas is characterised by a downward sloping demand curve, $f(q)$, and an upward sloping domestic supply curve, $g(q)$. The demand curve plots the amount of bananas consumers will purchase at different prices. The higher the prevailing market price, the lower the quantity demanded, and hence the downward slope to $f(q)$. Similarly, the domestic supply curve plots how much of the fruit will be offered for sale by local producers at different prices. This curve slopes upwards since more bananas are offered for sale at higher prices. The price corresponding to the point at which the demand and supply curves intersect, p_0, represents the prevailing market price. This situation and is depicted in Figure 19.

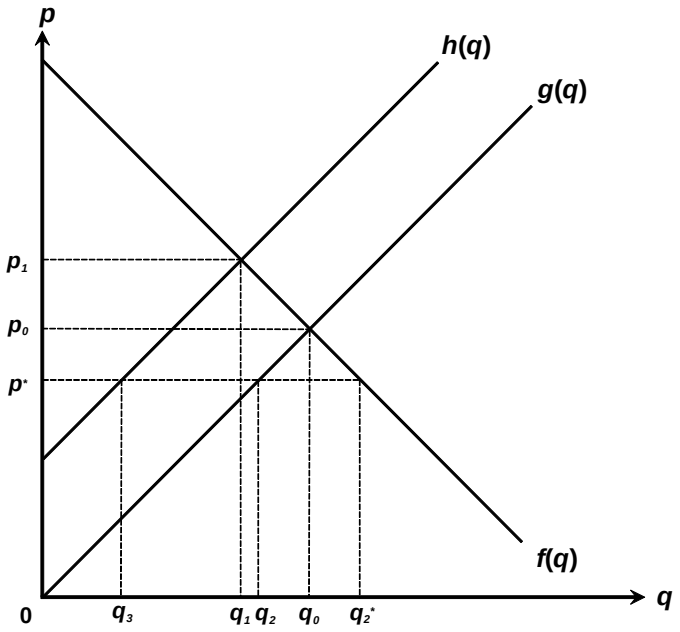

Figure 19. Impact of a plant pest on domestic producer surplus.

Foreign producers to the market are required to undertake a series of phytosanitary treatments to prevent the transfer of harmful pests and diseases to Australia via traded bananas. These treatments have a cost, *y*. As these costs are currently high enough to prevent overseas suppliers from supplying any product to the market at all, the market price adjusts according to the ebbs and flows of domestic supply. However, if the pre-import measures were less costly, foreign suppliers would exert competitive pressure on domestic suppliers, placing downward pressure on prices. We will discuss this prospect further below.

At present, *M. fijiensis* remains exotic to Australia, but has a positive probability of introduction, *z*. If circumstances were to conspire against Australian banana growers and the fungus was introduced into major production areas it would impose extra costs per unit of production on local suppliers. The domestic supply curve would therefore shift inwards as a lower quantity of bananas would be offered for sale at a given price. The increased production costs would relate to increased chemical inputs, labour and machinery costs growers are forced to pay to control *M. fijiensis* in their banana plantations. Assume these increased costs would be such that the supply curve would contract to the new supply curve $h(q)$ in Figure 19. There might also be additional costs to consider if the outbreak of the pest triggers a government or joint industry and government response to try to eradicate or contain the outbreak. We describe specific incursion response assumptions used in the model below, but for the moment simply denote these costs *c*.

Formally, the domestic losses that would result from a *M. fijiensis* outbreak can be estimated using a partial equilibrium model as the total expected change in *producer surplus* brought about the induced negative supply shift, plus *c*. Producer surplus is defined as net revenue earned by a producer from the sale of a good at a price above the minimum acceptable price they would have been willing to sell for before having to leave the market. In terms of Figure 19, this is represented by the area below prevailing market price line (i.e. p_0 if there are no imports) and above the supply curve.

The probability of producers facing either the disease-free supply curve $g(q)$ or the with-disease supply curve $h(q)$ depends on the probability of *M. fijiensis* arriving, *z*. In all likelihood, *z* is an increasing function of the quantity of imported bananas (call it q^*) from foreign sources where the fungus might be established, and a decreasing function of the phytosanitary measures imported fruit is subjected to prior to importation with cost *t* (i.e. $z(q^*, y)$). The entry probability z^* represents the level of importation risk deemed acceptable by biosecurity agencies and institutions. In Australia's case, this corresponds to a probability between 0.1 per cent and 5.0 per cent (Biosecurity Australia 2001).

As a starting point, where the level phytosanitary measures is high and the volume of imported product is negligible (i.e. corresponding to entry probability z_0), domestic producers face the supply schedule $g(q)$ and provide the total supply p_0 to the domestic market at a price p_0. If a *M. fijiensis* incursion were to occur (despite there being a severely restricted trade pathway) the supply curve will shift inwards to $h(q)$ and the new equilibrium price will rise to p_1, at which q_1 will be demanded by consumers. Note that even when no trade takes place $0 < z_0 < 1$ (Cook and Fraser 2008).

If the incursion response mounted upon detection (i.e. with cost *c*) of an outbreak fails to eradicate *M. fijiensis*, we assume this will lead to a great deal of international pressure for Australia to relax import requirements for bananas. Less stringent phytosanitary measures would allow foreign producers to exert downward pressure on the price of fresh bananas. Returning to Figure 19, if area freedom is lost and imported bananas are permitted into Australia the prevailing market price will fall to a level like p^* below the previous market equilibrium price p_0. Here, domestic banana growers will remain suppliers to the domestic market, but will supply a lower quantity, q_3. So, they will face both a falling price and an increase in their production costs relative to international competitors (i.e. due to the added expense of *M. fijiensis* management activities).

Using the conceptual framework of Figure 19, the expected Total Cost to Producers (TC^{P}) associated with quarantine requirements producing a *M. fijiensis* entry probability of z_0 can be determined by:

$$\mathrm{TC}^{\mathrm{P}}_{z_0} = z_0.\left[\left(p_0 - \int_0^{q_0} g(q)\right).dq - \left(p^* - \int_0^{q_3} h(q)\right).dq + c\right] \qquad (9)$$

Equation 9 states that given a high level of biosecurity effort where the probability of a pest incursion z_0 (corresponding to the ALOP), $\mathrm{TC}^{\mathrm{P}}_{z_0}$ is equal to the expected difference between the producer surplus if the volume of imports is negligible and no outbreak occurs and the producer surplus if an outbreak occurs and phytosanitary measures are relaxed, plus the expected cost of incursion response. $\mathrm{TC}^{\mathrm{P}}_{z_0}$ represents the expected total producer cost under a *quarantine-restricted trade* scenario.

For comparison, let us also consider a situation in which there are no impediments to trade. If under a new biosecurity policy all phytosanitary measures currently placed on imported bananas were removed and the prevailing market price falls to p^*, domestic producers will remain suppliers to the domestic market, but they will supply a lower quantity, q_2. However, the probability of *M. fijiensis* arriving (i.e. *z*) via the trade pathway provided by $q_2^* - q_2$ imports increases relative to a quarantine-restricted trade scenario. To simplify the effects of uncertainty, let us assume a deterministic change in *z* results from relaxing the intensity of phytosanitary measures imposed on imported bananas from a high level (corresponding to entry probability z_0) to a lower level corresponding to entry probability z^* (i.e. $z^* > z_0$). If an incursion does result and the supply curve shifts

inwards to $h(q)$, the quantity supplied by domestic producers will further be reduced to q_3 as their production costs increase relative to their international competitors.

Under this situation, the estimation of TC^{P} becomes:

$$\mathrm{TC}^{\mathrm{P}}_{z^*} = z^*.\left[\left(p^* - \int_0^{q_2} g(q)\right).dq - \left(p^* - \int_0^{q_3} h(q)\right).dq + c\right] \qquad (10)$$

Equation (10) states that given a low level of biosecurity effort where the probability of a *M. fijiensis* incursion is z^*, $\mathrm{TC}^{\mathrm{P}}_{z^*}$ is equal to the expected difference between the producer surplus if no outbreak occurs and the producer surplus if an outbreak occurs, plus the expected cost of incursion response. $\mathrm{TC}^{\mathrm{P}}_{z^*}$ is the expected total producer cost under an *unrestricted trade* scenario.

We note that our analysis might also include changes in domestic consumers' welfare as the market price changes according to the level of international competition and domestic pest area freedom status. However, we ignore consumer welfare in this discussion. Indeed, many economic analyses that have formed part of international risk assessments of market access requests from one international supplier to another have been completed on the basis of potential producer surplus losses alone. For a review and a discussion of how this has been influenced by the wording of the Agreement of on the Application of Sanitary and Phytosanitary Measures, see Cook (2008) and Cook *et al.* (2011b).

To estimate the potential shift in the supply curve and value of $\mathrm{TC}^{\mathrm{P}}_{z_0}$ and $\mathrm{TC}^{\mathrm{P}}_{z^*}$ over time, we use a biological model to simulate the arrival, spread and impact of *M. fijiensis* in Australian banana plantations over a thirty year time period. This model is then combined with a measure of the marginal damage cost of invasion and control costs (including eradication attempts).

Banana growing areas are grouped by State in the model, and are denoted *i*. Table 8 provides details of each by area, yield and value of banana crops. *M. fijiensis* arrival events in these regions are generated using unrestricted entry and establishment probabilities (denoted z^{ent} and z^{est}, respectively), stated in Biosecurity Australia (2008). A Markov chain process, described in Hinchy and Fisher (Hinchy and Fisher 1991), is used to change z^{ent} and z^{est} over time according to a vector of transitional probabilities. These transitional probabilities describe the likelihood of moving from one disease state to another. z^{ent} and z^{est} are combined to form a probability of invasion for a specific banana-growing region *i*, z_i:

$$z_i = z^{\mathrm{ent}} \times z^{\mathrm{est}} \text{ where } 0 < z_i < 1. \qquad (11)$$

A stratified diffusion model combining both short and long distance dispersal processes is used to predict the area potentially affected by *M. fijiensis* post-establishment in each region *i* in time period *t*, A_{it}. Parameter estimates for this model appear in Table 10, and are explained below.

The model is derived from the reaction diffusion models originally developed by Fisher (1937) which have been shown to provide a reasonable approximation of the spread of a diverse range of organisms (Cook, Carrasco *et al.* 2011b; Dwyer 1992; Holmes 1993; McCann, Hastings *et al.* 2000; Okubo and Levin 2002). These models assert that an invasion diffusing from a point source will eventually reach a constant asymptotic radial spread rate of $2\sqrt{r_i D_{ij}}$ in all directions, where r_i describes a growth factor for *M. fijiensis* per year in region *i* (assumed constant over all infected sites) and D_{ij} is a diffusion coefficient for an infected site *j* in region *i* (assumed constant over time) (Cook, Carrasco *et al.* 2011b; Hengeveld 1989; Lewis 1997; Shigesada and Kawasaki 1997). Hence, we assume that the original infection (i.e. the first of a probable series of sites, *j*) takes place in a homogenous environment in region *i* and expands by a diffusive process such that area infected at time *t*, a_{ijt}, can be predicted by:

$$a_{ijt} = z_i\left[\pi\left(2t\sqrt{r_i D}_{ij}\right)^2\right] = z_i\left(4D_{ij}\pi r_i t^2\right). \qquad (12)$$

For practical purposes, an estimate of D_{ij} can be derived from the mean dispersal distance ($\bar{\delta}_{ij}$) of the pathogen at an infection site, where $D_{ij} = \frac{2\left(\bar{\delta}_{ij}\right)^2}{\pi t}$ (Andow, Kareiva *et al.* 1990; Cook, Long *et al.* 2010a; Cook, Fraser *et al.* 2011c). $\bar{\delta}_{ij}$ is the site-specific average distance (in metres) over which dispersal events leading to infection occur. By assuming D_{ij} is constant across all sites *j* we ignore demographic stochasticity and consequent non-uniform invasion.

The density of *M. fijiensis* infection within a_{ijt} influences the control measures required to counter the effects of infection, and thus partially determines the value of A_{it}. We assume that in each site *j* in region *i* affected, the infection density, N_{ijt}, grows over time period *t* following a logistic growth curve until the carrying capacity of the host environment, K_{ij}, is reached:

$$N_{ijt} = \frac{K_{ij} N_{ij}^{\min} e^{r_i t}}{K_{ij} + N_{ij}^{\min}(e^{r_i t} - 1)}. \qquad (13)$$

Table 10. Parameter values for the black Sigatoka model.

Description	Values
Probability of entry and establishment in an unrestricted trade setting, z^*. [a]	0.68
Probability of entry and establishment in a quarantine-restricted trade setting, z_0. [a]	Uniform(1.0×10^{-3},5.0×10^{-2})
Detection probability.	Binomial(1.0,0.6)
Population diffusion coefficient, D (m^2/yr). [b]	Pert(1.0×10^4,1.5×10^4,2.0×10^4)
Minimum area infected immediately upon entry, A^{min} (m^2).	1.0×10^3
Maximum area infected, A^{max} (m^2). [c]	1.4×10^8
Intrinsic rate of infection and density increase, r(yr^{-1}). [d]	Pert(0.20,0.35,0.50)
Minimum infection density, N^{min} (#/m^2).	1.0×10^{-4}
Maximum infection density, K (#/m^2). [d]	Pert(100,550,1000)
Minimum number of satellite sites generated in a single time step, S^{min} (#).	0
Maximum number of satellite sites generated in a single time step, S^{max} (#). [d]	Pert(50,60,70)
Intrinsic rate of new foci generation per unit area of infection, μ (#/m^2). [d]	Pert(1.0×10^{-2},3.0×10^{-2},5.0×10^{-2})
Discount rate (%).	5
Supply elasticity. [e]	Uniform(0.2,0.8)
Demand elasticity. [e]	Uniform(-1.1,-1.0)
Prevailing market price of bananas in the first time step ($/T). [c]	1,900
Fall in domestic price of bananas following loss of area freedom status and relaxation of trade restrictions, $p_0 - p^*$ (%).	Pert(40,50,60)
Maximum area considered for eradication, A^{erad} (ha).	Pert(300,400,500)
Cost of eradication, c ($/ha). [g]	Pert(1.0×10^4,1.5×10^4,2.0×10^4)
Negative exponential rate of decline for eradication success probability with respect to area affected	Pert(0.1,0.15,0.2)
Increased fungicide application and de-leafing costs ($/ha). [h]	700-2120
Yield reduction despite control (%).	Pert(0.0,2.5,5.0)

[a] Biosecurity Australia (2008); [b] Derived from Sapoukhina *et al.* (2010); [c] ABS (2011), Note 1ha = 10 000m^2; [d] Specified with reference to Cook (2003) and Waage *et al.* (2005) using distributions defined in Biosecurity Australia (2001); [e] Ulubasoglu *et al.* (2011); [f] James and Anderson (1998); [g] Assumes average density of planting of 2,000 stems/ha and removal, transport, destruction and chemical costs amounting to $20 per tree. This is inclusive of labour (team of three at $50/hr per person), bulldozing equipment ($100/hr at 20 hours per hectare), truck hire ($75/hr), incendiaries ($60/ha for green waste) and creation of a circular chemical buffer zone approximately 5 hectares in diameter around previously infected sites. Chemical used is assumed to be dithane (applied at a rate of 3kg/ha or $25/ha) and oil (applied at 3L/ha or $10/ha) at fortnightly intervals rotated with propiconazole (applied at a rate of 0.3L/ha or $5/ha). Assume 2 additional dithane treatments are required and 4 propiconazole treatments (and therefore 6 additional oil treatments), each taking 1 hour per hectare to apply; [h] Generally, control of *M. musicola* involves applications of dithane (@ 3kg/ha or $25/ha) and oil (@ 3L/ha or $10/ha) at regular intervals during wet periods (Allen *et al.* 1992). Control of *M. fijiensis* requires the frequency of application to be increased. In addition it may be desirable for growers to rotate the use of dithane and oil with propiconazole (@ 0.3L/ha or $25/ha). Assume four additional dithane treatments are required and eight propiconazole treatments (and therefore 12 additional oil treatments) in tropical areas and an additional two dithane treatments and four propiconazole treatments (hence six additional oil treatments) in sub-tropical areas. Further assume aerial application costs of $25/ha. Also assume an additional 10 de-leafing periods at a cost of $140/ha each. In areas that have not been affected by *M. fijiensis*, assume an additional 5 de-leafing periods are necessary as a precautionary measure after the disease has been detected in other areas at a cost of $140/ha each.

Here, $N_{ij}^{\min}$ is the size of the original infection at site *j* in region *i* and r_i is the intrinsic rate of density increase in region *i* (assumed to be the same as the intrinsic rate of infection increase) (Cook, Fraser *et al.* 2011c).

In addition to a_{ijt} and N_{ijt}, the size of A_{it} depends on the number of nascent foci or *satellite* infection sites in year *t*, s_{it}, which can take on a maximum value of $s_i^{\max}$ in any year (Moody and Mack 1988). These sites result from events external to the initial outbreak itself, such as weather phenomena, animal or human behaviour, which periodically jump the expanding infection beyond the infection front (Cook, Fraser *et al.* 2011c). We use a logistic equation to generate changes in s_{it} as an outbreak continues:

$$s_{it} = \frac{s_i^{\max} s_i^{\min} e^{\mu_i t}}{s_i^{\max} + s_i^{\min}(e^{\mu_i t} - 1)} \qquad (14)$$

where μ_i is the intrinsic rate of new foci generation in region *i* (assumed constant over time) and $s_i^{\min}$ is the minimum number of satellite sites generated in region *i*.

Given equations (12)-(14), we can express A_{it} as:

$$A_{it} = \sum_{j=1}^{m} \left(a_{ijt} N_{ijt}\right)^{s_{it}} \text{ where } 0 \le A_{it} \le A_i^{\max} \qquad (15)$$

.

In terms of preventing naturalisation, eradication is the only government incursion response activity simulated in the model. It involves the complete removal of infected trees and the creation of intensive buffer zones (using fungicide treatments) and de-leafing around infected sites. *M. fijiensis* is a listed species under the Emergency Plant Pest Response Deed (EPPRD) (PHA 2005) which states that in the event of an incursion a pre-agreed cost sharing arrangement for eradication is activated. Listed species fall in to one of four cost sharing categories relating to their potential impacts on public and private resources. The category chosen dictates an appropriate split of eradication funding between government and private funding sources. Currently, *M. fijiensis* is classified as a category two species, indicating a 20 per cent private and 80 per cent government/public funding contribution (PHA 2005).

We assume that eradication is immediately commenced once the banana industry and government have been alerted to the presence of *M. fijiensis* in Australia. The detection that triggers the EPPRD is, on average, assumed to occur in 60 per cent of incursion events simulated by the model using a binomial distribution (i.e. binomial(1.0,0.6)). The probability that the eradication attempt will successfully remove an *M. fijiensis* incursion is arbitrarily assumed to decline negative exponentially at an average rate of $e^{0.15A_{it}}$, where A_{it} is the area infected with the fungus in region *i* in year *t* (see Table 2). If this does not occur before infection has spread to a pre-defined maximum area, A^{erad}, which we have arbitrarily assumed is between 300ha and 500ha, the eradication attempt is aborted.

If detection does occur sufficiently early, eradication entails the complete removal of all infected plants and the creation of a chemical buffer zone around the area where the

infection occurred. Infected plants are removed from quarantined properties for incineration. After infected plants have been destroyed the area is immediately re-planted to bananas and effectively re-enters production two years from the time of re-planting. In present value terms, the cost of removing and disposing of infected plants, replanting and waiting for commercially viable bunches is between $10,000 and $20,000 per hectare. See Table 2 for details.

When detection does not occur early in an outbreak or when an eradication attempt fails to prevent infection reaching A^{erad}, eradication is aborted. This does not mean that *M. fijiensis* now spreads unimpeded within the virtual world of the model since we assume fungicide treatments and diseased leaf removal activities currently used for *M. musicola* control can be adapted to *M. fijiensis* control. However, this will add to growing costs considerably as the frequency of these activities increase and are not guaranteed to be 100 per cent effective. Yield losses despite control are estimated to average around 5 per cent per annum (represented in the model as Pert(0.0,0.5,0.1)). In addition, we assume when eradication fails phytosanitary measures imposed on imported bananas are relaxed, exerting downward pressure on the domestic price, as explained above.

The spread of *M. fijiensis* is connected dynamically with the costs of eradication and on-plantation control by simply multiplying the area infested by a constant marginal damage cost (or an average damage cost). For outbreaks involving less than A_{erad}, area is multiplied by eradication and replanting costs (see notes below Table 10). When infection spreads beyond A^{erad} the remaining area is multiplied by an average on-plantation disease management costs. By summing the production losses over each time step and assuming fixed costs are zero, we estimate $\mathrm{TC}^{\mathrm{P}}_{z_0}$ and $\mathrm{TC}^{\mathrm{P}}_{z^*}$ over a thirty year period.

3.5.3.3. Results

M. fijiensis is assumed to be absent from Australia at the beginning of both the quarantine-restricted trade scenario and the unrestricted trade scenario. We assume entry and establishment is likely to occur at some point or multiple points over the estimation period. Therefore, the resultant expected spread area values calculated from 10,000 iterations of the model are positive in both scenarios. But, as the timing of incursions across the temporal range simulated in the model is stochastic, there is a large spread of possible spread scenarios in both the quarantine-restricted and unrestricted trade scenarios. These projections have been aggregated across all production regions to produce Figure 20.

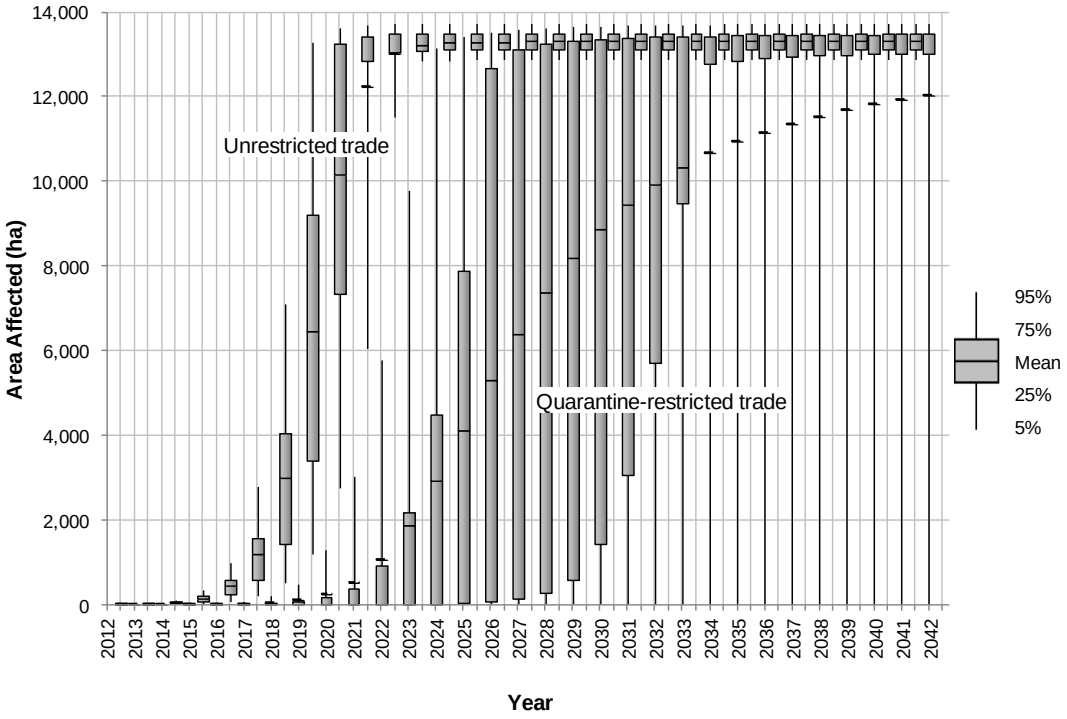

Figure 20. Expected area of commercial banana plantations affected by black Sigatoka in Australia under an unrestricted trade and quarantine-restricted trade scenario.

Figure 21 illustrates how the resultant $\mathrm{TC}^{\mathrm{P}}_{z_0}$ and $\mathrm{TC}^{\mathrm{P}}_{z^*}$ are expected to change over a 30-year period. Here, the mean values of $\mathrm{TC}^{\mathrm{P}}_{z_0}$ and $\mathrm{TC}^{\mathrm{P}}_{z^*}$ predicted by the model are plotted with 10 per cent and 50 per cent confidence intervals. All projected benefits are discounted at 5 per cent per annum. To reiterate, unrestricted trade maximizes the likelihood of the fungus entering Australia on imported fruit. Quarantine-restricted trade imposes pre-entry requirements on imported fruit up to the point where the risks of entry, establishment, spread and consequences correspond with Australia's appropriate level of protection.

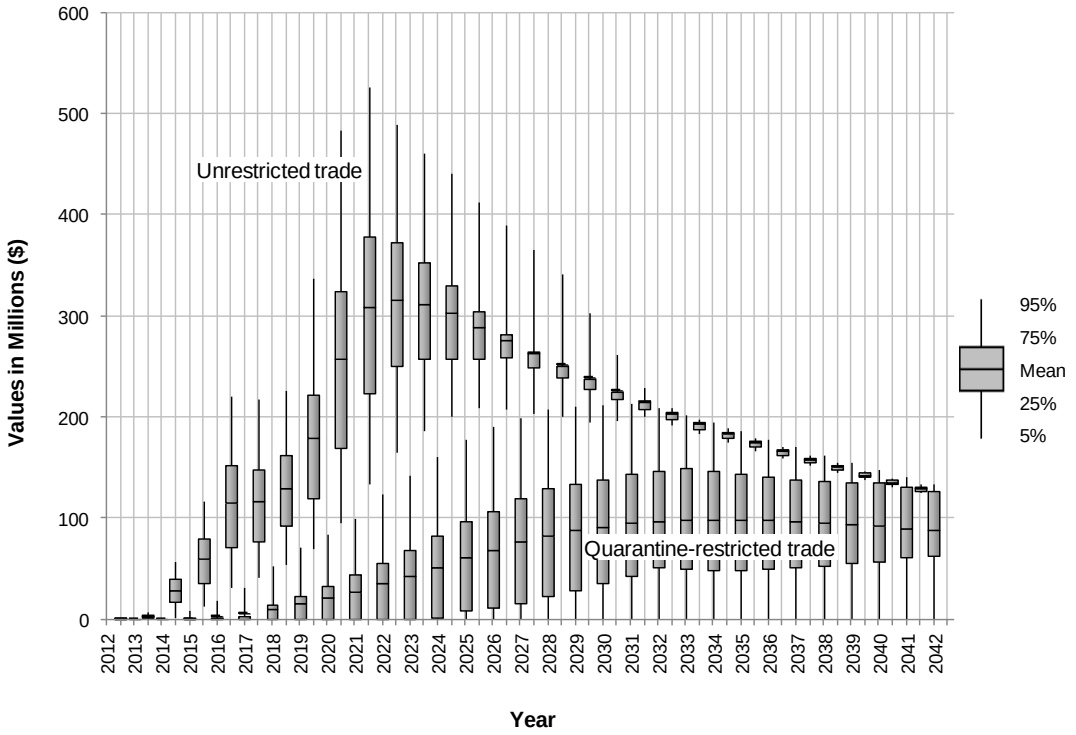

Figure 21. Predicted industry losses from black Sigatoka in Australia under an unrestricted trade and quarantine-restricted trade setting.

In view of the uncertainty surrounding many of the parameters used to describe the *M. fijiensis* infection and spread process, the sensitivity of the change in TC^{P} to the key assumptions of the model must be tested to gauge the robustness of our predictions. We were particularly interested in the sensitivity of results for the quarantine-restricted trade scenario, which represents Australia's current policy with respect to imported bananas. Parameters were sampled from a uniform distribution with a maximum (minimum) of +50 per cent (-50 per cent) of the original values entered in to the model using Monte Carlo simulation. The Spearman's rank correlation coefficients relating the sampled model parameter values and the change in $\mathrm{TC}^{\mathrm{P}}_{z_0}$ were then calculated. The results appear in Figure 22.

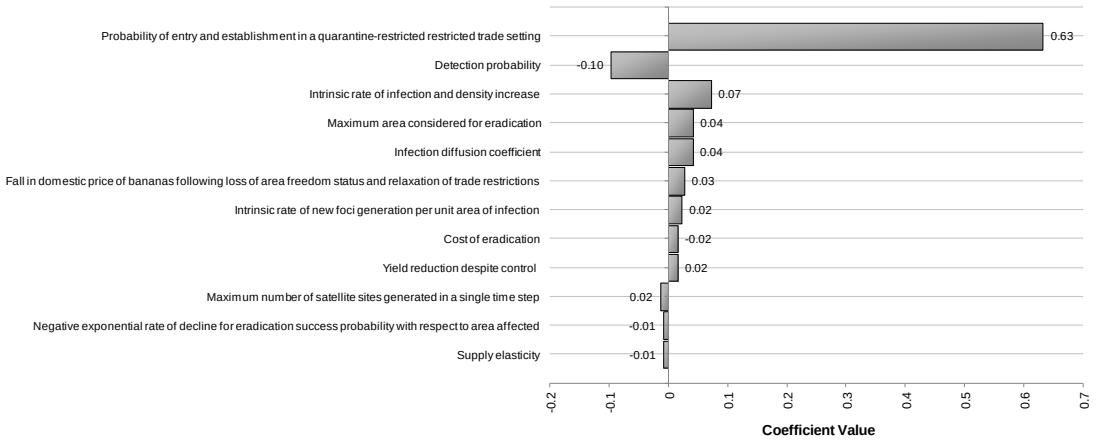

Figure 22. Sensitivity analysis for the black Sigatoka model.

The sensitivity tests indicate that the model is highly responsive to the probability of entry and establishment in a quarantine-restricted trade setting (0.63). Results are also responsive, although to a much lesser extent, to the detection probability (-0.10), the intrinsic rate of infection and density increase (0.07), the maximum area considered for eradication (0.04) and the infection diffusion coefficient (0.04).

3.5.3.4. Discussion

Our results indicate the potentially large costs that would affect the Australian banana industry if *M. fijiensis* was to become established. Even under a quarantine-restricted trade situation where strict phytosanitary measures prevent the likelihood of incursion from exceeding a very low level, the average annual loss to producers ($\mathrm{TC}^{\mathrm{P}}_{z_0}$) is expected to exceed $60 million. This is a quantitative representation of Australia's ALOP with respect to bananas.

Our estimate of $\mathrm{TC}^{\mathrm{P}}_{z_0}$ compares to expected losses of over $180 million if all quarantine restrictions on bananas imported to Australia are removed (i.e. $\mathrm{TC}^{\mathrm{P}}_{z^*}$). This means that the increase in the present value of increased producer costs predicted to result from a relaxation of phytosanitary measures from their current levels (i.e. $\Delta\mathrm{TC}^{\mathrm{P}} = \mathrm{TC}^{\mathrm{P}}_{z^*} - \mathrm{TC}^{\mathrm{P}}_{z_0}$) is estimated to average over $125 million per year over 30 years across banana producing regions of Australia. The standard deviation of the distribution of $\Delta\mathrm{TC}^{\mathrm{P}}$ is $39 million and skewness -0.4 (i.e. the distribution is skewed slightly left such that the left tail is long compared to the right tail). This increase in expected producer costs equates to approximately one third of the current gross value of the banana industry in Australia.

The two of the five most sensitive model parameters in determining the total producer costs under the quarantine-restricted and unrestricted trade scenarios, the intrinsic rate of infection and density increase and the infection diffusion coefficient, cannot be influenced by policy. This makes the robustness of our predictions difficult to ascertain in the absence of targeted scientific information.

Detection probability is also relatively important in determining the total producer costs over time and can be influenced by policy decisions. If the proficiency of surveillance officers and growers is very high following the Tully *M. fijiensis* outbreak, we would expect total producer costs to be smaller than those we have predicted. While we have assumed in our calculations that the probability of an infection being detected is high (60 per cent), this is a subjective estimate. Given the similarities between *M. musicola* and *M. fijiensis*, it may be that the latter is much more difficult to detect and our estimates of total producer costs are biased downwards. However, the sensitivity analysis of Figure 4 suggests there may be considerable gains from strategies that encourage plantation monitoring and disclosure of detection information. Whether the costs of such strategies would outweigh the benefits requires a separate analysis focuses specifically on surveillance.

We have also demonstrated the relative importance the maximum area considered for eradication in determining total producer costs from plant disease. In reality, this is partially a technical, partially an economic and largely a socio-political issue. Area will certainly play a part in the technical feasibility and likely net returns of eradication, but will be considered next to a raft of other factors related to potential social impacts. The manner in which quarantine authorities approach eradication can have a significant impact on the trauma inflicted on individual growers and local economies. Their resilience to plant disease crises will be dependent upon the community's unique geographic, economic, and social profile, degree of social cohesion, community leadership and history of overcoming crises (Barclay 2005).

Although the consequences for the wellbeing of communities and regional economies are potentially the most severe of all the impacts of a disease like *M. fijiensis*, they remain the most difficult to quantify. At the individual and family level, the social impacts of the disease and the incursion response could include strains on family relationships induced by feelings of personal responsibility, isolation, loss of reputation, identity and dignity. At the community level the impacts could range from a breakdown of normal community activities in the midst of quarantine and movement restrictions, to the changes in interpersonal relationships affecting the longer term cohesion of the community (NSWDOCS 2000). These effects could be further exacerbated other long-term challenges faced by banana-growing communities such as urbanisation, labour shortages and extreme weather events.

The impacts on the community are not reflected in our estimates of total producer costs, and highlight a potential problem of relying solely on producer surplus as a measure of social welfare in biosecurity assessments. We acknowledge that additional information is required to supplement the economic damage estimates we have presented to form appropriate policies in relation to *M. fijiensis* exclusion.

3.5.4. An assessment of the potential economic impact of Moko disease on the Australian banana industry

3.5.4.1. Introduction

In this section, we use the same dynamic partial equilibrium model outlined in the previous section to estimate potential consequences for the Australia banana industry associated with Moko disease, and how this differs from an unrestricted trade risk.

The majority if the industry in Australia enjoys area freedom status from many of the world's major banana pests and diseases, including the bacterial diseases Moko and banana blood disease. These harmful diseases are caused by different strains of the same bacterium, *Ralstonia solanacearum* – race 2. Although there are many similarities between the symptoms and epidemiology of Moko and blood disease, the causal bacteria show distinctive phenotypic and genetic differences (Fegan 2005). General symptoms include the youngest leaves becoming yellow-green in colour and eventually collapsing. Soon after this the remaining leaves also collapse. Fruit may turn yellow and the peel may split, and pulp will show a firm brown rot that becomes grey (Stansbury 2000). *R. solanacearum* is initially spread through root contact with infected plants. After infection, the bacteria are carried through the internal tissue and can be transmitted by contaminated cutting tools during pruning, animal hooves and irrigation water (Brown 1998; Stansbury 2000). Insects may also transmit *R. solanacearum* since bacteria can ooze from buds. Worldwide, *R. solanacearum* is found throughout Africa, Central and South America, the Caribbean and Asia, and BBD is only found in parts of Asia. There are no treatments known to be effective against the disease other than destroying infected plants (Stansbury 2000).

3.5.4.2. Methods

The method of analysis is the same as described in section 3.5.3.2, and we refer readers to this section for details. However, an important difference in the assumptions is to be noted. In the simulation model for *R. solanacearum*, we assume that a loss of area freedom status will not lead to a relaxation of phytosanitary measures imposed on imported bananas. We subjectively assume that while extremely serious, the disease does not have the same stigma attached to it as *M. fijiensis*. We therefore assume that naturalisation does not lead to increased international competition and downward pressure on the domestic price of bananas. The parameter values used in the model to simulate Moko disease appear in Table 11.

Table 11. Parameter values for the Moko disease model.

Description	Values
Probability of entry and establishment in an unrestricted trade setting, z^*. [a]	0.16
Probability of entry and establishment in a quarantine-restricted trade setting, z_0. [a]	Uniform(1.0×10^{-3},5.0×10^{-2})
Detection probability.	Binomial(1.0,0.6)
Population diffusion coefficient, D (m^2/yr). [b]	Pert(1.0×10^4,1.5×10^4,2.0×10^4)
Minimum area infected immediately upon entry, A^{min} (m^2).	1.0×10^3
Maximum area infected, A^{max} (m^2). [c]	1.4×10^8
Intrinsic rate of infection and density increase, r(yr^{-1}). [d]	Pert (0.10,0.15,0.20)
Minimum infection density, N^{min} (#/m^2).	1.0×10^{-4}
Maximum infection density, K (#/m^2). [d]	Pert(100,550,1000)
Minimum number of satellite sites generated in a single time step, S^{min} (#).	0
Maximum number of satellite sites generated in a single time step, S^{max} (#). [d]	Pert(10,15,20)
Intrinsic rate of new foci generation per unit area of infection, μ (#/m^2). [d]	Pert(1.0×10^{-2},3.0×10^{-2},5.0×10^{-2})
Discount rate (%).	5
Supply elasticity. [e]	Uniform(0.2,0.8)
Demand elasticity. [e]	Uniform(-1.1,-1.0)
Prevailing market price of bananas in the first time step ($/T). [c]	1,900
Fall in domestic price of bananas following loss of area freedom status and relaxation of trade restrictions, $p_0 - p^*$ (%).	0
Maximum area considered for eradication, A^{erad} (ha).	Pert(300,400,500)
Cost of eradication, c ($/ha). [g]	Pert(1.0×10^4,1.5×10^4,2.0×10^4)
Negative exponential rate of decline for eradication success probability with respect to area affected	Pert(0.10,0.15,0.20)
Increased insecticide application costs ($/ha). [h]	130
Yield reduction despite control (%).	Pert(10,30,50)

[a] Biosecurity Australia (2008); [b] Specified with reference to Waage *et al.* (2005) using distributions defined in Biosecurity Australia (2001); [c] ABS (2011), Note 1ha = 10 000m^2; [d] Specified with reference to Cook (2003) and Waage *et al.* (2005) using distributions defined in Biosecurity Australia (2001); [e] Ulubasoglu *et al.* (2011); [f] James and Anderson (1998); [g] Assumes average density of planting of 2,000 stems/ha and removal, transport, destruction and chemical costs amounting to $20 per tree. This is inclusive of labour (team of three at $50/hr per person), bulldozing equipment ($100/hr at 20 hours per hectare), truck hire ($75/hr), incendiaries ($60/ha for green waste) and creation of a circular chemical buffer zone approximately 5 hectares in diameter around previously infected sites. Chemical used is assumed to be dithane (applied at a rate of 3kg/ha or $25/ha) and oil (applied at 3L/ha or $10/ha) at fortnightly intervals rotated with propiconazole (applied at a rate of 0.3L/ha or $5/ha). Assume 2 additional dithane treatments are required and 4 propiconazole treatments (and therefore 6 additional oil treatments), each taking 1 hour per hectare to apply; [h] Assumes: (i) labour costs of $50/ha (i.e. 1 application × 1hr/ha × $50/hr); (ii) 75mL of chemical solution is used per banana plant per treatment costing $10 per litre (e.g. dimethoate diluted to 75mL/100L (Cook 2003)) (i.e. approx. $15/ha); and (iii) two additional chemical treatments will provide sufficient suppression of potential insect vectors.

3.5.4.3. Results

R. solanacearum is assumed to be absent from Australia at the beginning of both the quarantine-restricted trade scenario and the unrestricted trade scenario. We assume entry and establishment is likely to occur at some point or multiple points over the estimation period. Therefore, the resultant expected spread area values calculated from 10,000 iterations of the model are positive in both scenarios. These projections have been aggregated across all production regions to produce Figure 23.

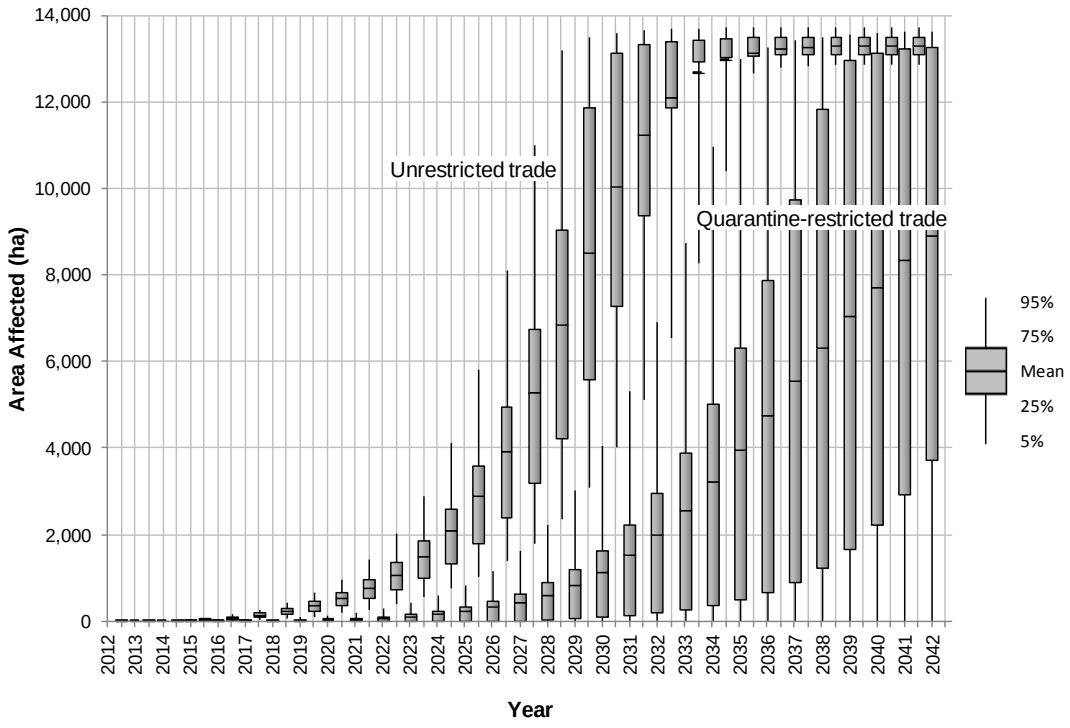

Figure 23. Expected area of commercial banana plantations affected by Moko disease in Australia under an unrestricted trade and quarantine-restricted trade scenario.

Figure 24 illustrates how the resultant $\mathrm{TC}^{\mathrm{P}}_{z_0}$ and $\mathrm{TC}^{\mathrm{P}}_{z^*}$ (i.e. see equations (9) and (10), respectively) are expected to change over a 30-year period. Here, the mean values of $\mathrm{TC}^{\mathrm{P}}_{z_0}$ and $\mathrm{TC}^{\mathrm{P}}_{z^*}$ predicted by the model are plotted with 10 per cent and 50 per cent confidence intervals. All projected benefits are discounted at 5 per cent per annum. To reiterate, unrestricted trade maximizes the likelihood of the fungus entering Australia on imported fruit. Quarantine-restricted trade imposes pre-entry requirements on imported fruit up to the point where the risks of entry, establishment, spread and consequences correspond with Australia's appropriate level of protection.

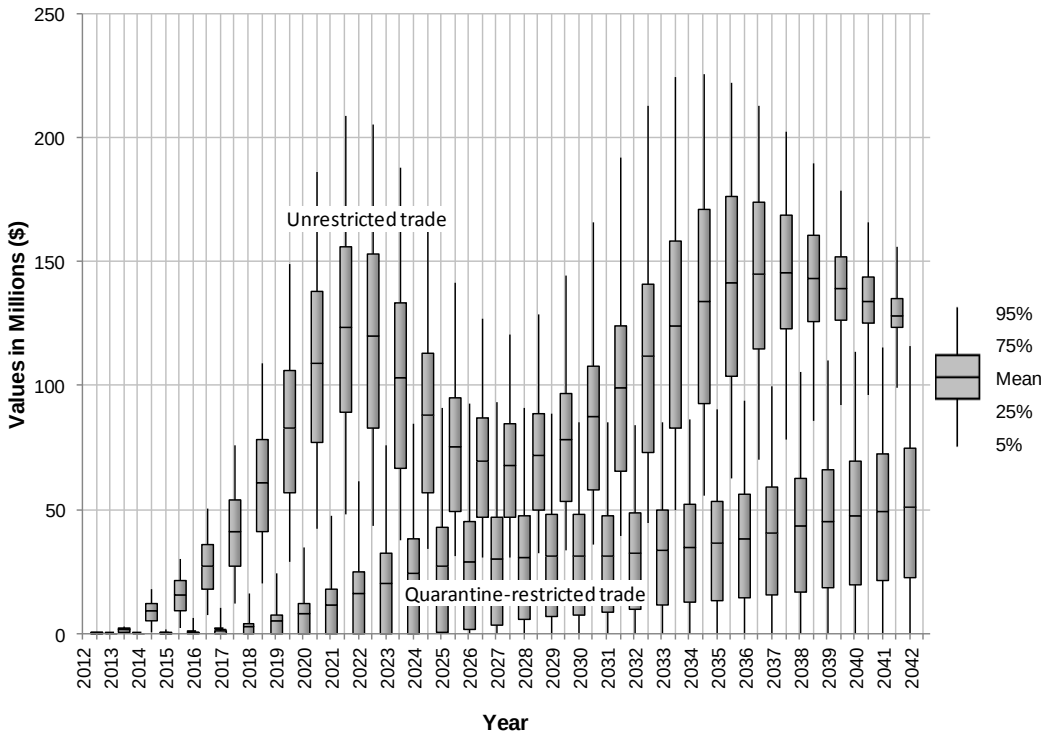

Figure 24. Predicted industry losses from Moko disease in Australia under an unrestricted trade and quarantine-restricted trade setting.

It is interesting to note that in the unrestricted trade scenario a bimodal distribution of expected costs is produced. The first peak corresponds to early attempts at eradication following incursions. Given that *R. solanacearum* spreads relatively slowly of its own accord (i.e. where sound hygiene practices and insect control measures are in place), the model predicts a high degree of success at eradication before the threshold level is reached. Generally, this is expected to occur between time steps zero (i.e. the year 2012) and 13 (i.e. 2025). This eradication comes at considerable cost. In later time steps eradication attempts have generally been aborted, and the distribution of costs reverts back to a curve proportional to the area curve of Figure 23. However, note the erosive effects of the discount rate at these relatively distant time steps.

In view of the uncertainty surrounding many of the parameters used to describe the *R. solanacearum* infection and spread process, the sensitivity of the change in TC^{P} to the key assumptions of the model must be tested to gauge the robustness of our predictions. We were particularly interested in the sensitivity of results for the quarantine-restricted trade scenario, which represents Australia's current policy with respect to imported bananas. Parameters were sampled from a uniform distribution with a maximum (minimum) of +50 per cent (-50 per cent) of the original values entered in to the model using Monte Carlo simulation. The Spearman's rank correlation coefficients relating the sampled model parameter values and the change in $\mathrm{TC}^{\mathrm{P}}_{z_0}$ were then calculated. The results appear in Figure 25.

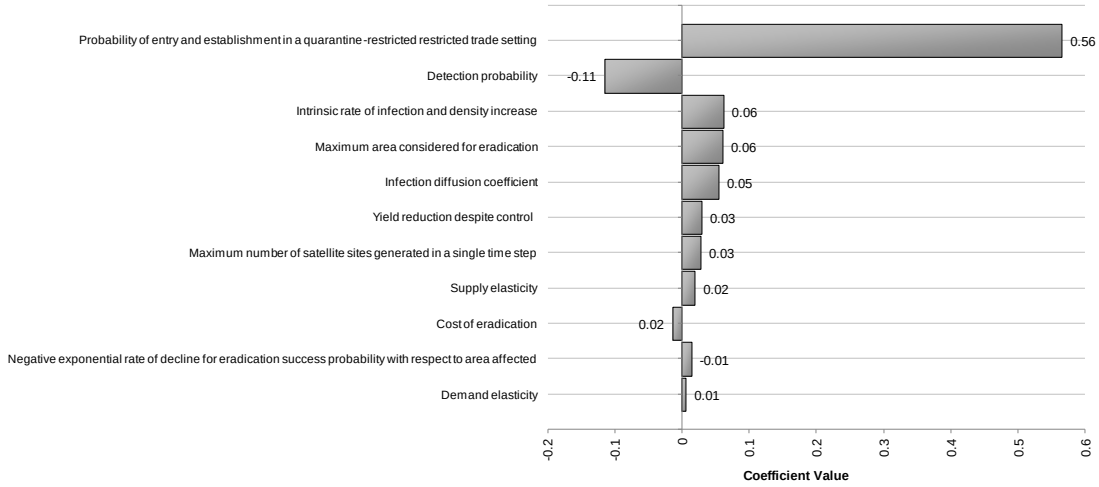

Figure 25. Sensitivity analysis for the Moko disease model.

The sensitivity tests indicate that the model is highly responsive to the probability of entry and establishment in a quarantine-restricted trade scenario (0.56). Results are also sensitive to changes in four of the parameters listed in Table 11, although to a much lesser extent. These parameters and their correlation with predicted $\mathrm{TC}^{\mathrm{P}}_{z_0}$ are detection probability (-0.11), the intrinsic rate of infection and density increase (0.06), the maximum area considered for eradication (0.06) and the infection diffusion coefficient (0.05).

3.5.4.4. Discussion

Our analysis indicates that the Australian banana industry will suffer substantial losses over time if *R. solanacearum* was to become established, but there is a great deal of uncertainty about these potential impacts. This is particularly the case in the unrestricted trade scenario we have simulated. Under a quarantine-restricted trade situation where strict phytosanitary measures prevent the likelihood of incursion from exceeding a very low level, the average annual loss to producers ($\mathrm{TC}^{\mathrm{P}}_{z_0}$) is expected to exceed $25.6 million.

Our estimate of $\mathrm{TC}^{\mathrm{P}}_{z_0}$ compares to expected losses of $89.6 million if all quarantine restrictions on bananas imported to Australia are removed (i.e. $\mathrm{TC}^{\mathrm{P}}_{z^*}$). This is an average value across the 30-year time period over which the model predicted future impacts, and as illustrated in Figure 10 this is a dubious measure of impact given that the distribution from which it is calculated is bimodal. To give some indication of the uncertainty in expected losses to *R. solanacearum*, standard deviation is $19.5 million. Nevertheless, if we use the average value of predicted impact as a guide, it implies that if we were to move from a quarantine-restricted to an unrestricted trade setting, the increase in the present value of producer costs predicted from *R. solanacearum* introductions (i.e.

$\Delta TC^P = TC^P_{z^*} - TC^P_{z_0}$) is approximately $63.0 million per year. The standard deviation of the distribution of ΔTC^P is $20.9 million and skewness 0.28 (i.e. the distribution is skewed slightly right such that the right tail is long compared to the left tail). This increase in expected producer costs equates to 13 per cent of the current gross value of the banana industry in Australia.

The two of the five most sensitive model parameters in determining the total producer costs under the quarantine-restricted and unrestricted trade scenarios, the intrinsic rate of infection and density increase and the infection diffusion coefficient, cannot be influenced by policy. This makes the robustness of our predictions difficult to ascertain in the absence of targeted scientific information.

However, by far the most sensitive parameter is the probability of entry and establishment in a quarantine-restricted trade scenario, which certainly is dictated by biosecurity policy. The sensitivity analysis above indicates that relatively minor fluctuations (either up or down) in this value have large implications for potential future losses to *R. solanacearum* over time, at least as we have constructed our model.

3.5.5. An assessment of the potential economic impact of Panama disease (tropical race 4) on the Australian banana industry

3.5.5.1. Introduction

Panama disease is a serious wilt disease caused by the fungus, *Fusarium oxysporum* f.sp. *cubense* (*Foc*). This soil-borne disease causes severe dieback and wilt of banana plants, and can persist in the soil for decades. The disease was first discovered in Australia in 1876 but became well known following its appearance in Central America in about 1890 (Ploetz 1994). It destroyed around 40,000 hectares of bananas over a 50 year period in Central and Southern America and worldwide is considered to be one of the most severe threats facing the banana industry (Stover 1972).

Strains of *Foc* have been divided into four physiological 'races' based on differential pathogenicity, each of which affect different banana varieties. Race 1 is common in the Northern Rivers in New South Wales, Queensland's Sunshine Coast and Brisbane. It is also present in Coffs Harbour, Woolgoolga, Bundaberg and Mareeba, although less common (Newley 2010). A strain of *Foc* appearing closest to race 1 has also found to be affecting Cavendish bananas at Carnarvon, Western Australia (Shivas *et al.* 1995). Race 1 affects ladyfinger, ducasse and plantain bananas. Race 2 affects cooking varieties such as Bluggoe, while Race 3 only affects some species of Heliconia. Neither of these races is of economic significance to Australia.

Race 4 poses the greatest threat to the Australian banana industry since this form of the disease attacks Cavendish varieties. Race 4 has been shown to consist of two 'sub races', sub-tropical race 4 and tropical race 4. Tropical race 4, henceforth denoted *Foc* TR4, has the ability to attack unstressed plants, making it particularly dangerous to commercial plantations. This race has caused substantial damage on Cavendish varieties in Malaysia and Indonesia and was discovered on a property at Berry Springs in the Northern Territory in 1996 (Hennessy *et al.* 2005). It has since spread to commercial plantings at Lambell's Lagoon, Middle Point and Wanderrie Road and is currently restricted to those areas only. It is not been found in Darwin or Palmerston or other rural areas (Walduck and Daly 2006). In September 2011 a suspected *Foc* TR4 outbreak was detected on one plantation in north Queensland, but this has since been diagnosed as false Panama disease disorder caused by plant stress, rather than a pathogen.

Sub-tropical race 4 is somewhat less damaging, as plants are usually only attacked when in stressed conditions. This race is present in southern Queensland and northern New South Wales, and also in Taiwan, South Africa and China (Cook 2003; McKirdy and Brown 1999).

Foc TR4 is of primary interest in this assessment. All control techniques for this strain have proved unsuccessful. Once a plantation becomes infected with this disease, prevention of spread can only be achieved by the destruction of infected plants, maintenance of a buffer zone of healthy plants around them and long term fallow of affected land. Given the ability of *Foc* TR4 to persist in the soil removed trees cannot be replaced once an infection has been detected.

Should the disease ever be more widely established in Australia, quantifying its impact is an essential step in developing effective practices and policy for management. Here, as above, we use a stochastic bioeconomic model that enables the economic impact of the disease to be estimated based on relatively poorly specified ecological and economic

parameters. We take account of the presence of *Foc* TR4 in the Northern Territory and predict the economic impact of the disease throughout the Australian industry over time.

3.5.5.2. Methods

The stochastic simulation model used in this assessment determines total *expected* (or probability-weighted) damage from *Foc* TR4 over a 30-year period. Uncertain or variable parameters are specified as probability distributions, and 10,000 model iterations are run using values randomly sampled across the range of each distribution using a Latin hypercube sampling algorithm.

The model of infection spread treats each Australian State as a separate region. Table 8 provides details of all Australian banana producing regions. *Foc* TR4 is considered established in the Northern Territory with a small number of properties currently affected. All other production regions are assumed to be free of the disease. An incursion event in any of these regions is the result of two distinct occurrences, arrival and establishment. The probability of a successful outbreak, or the transition between a 'with disease' (call it event *a*) and 'without disease' (event *b*) state is described as a regular Markov process such that the probability of event *a* occurring in any given time period will reduce to a constant value after several periods. Each element of the transition matrix $\boldsymbol{Z} = \begin{pmatrix} z_{aa} & z_{ab} \\ z_{ba} & z_{bb} \end{pmatrix}$, where *a* defines the row and *b* the column, provides an indication of the invasibility of the ecosystem concerned (Perrings 1998). We use deterministic transitional probabilities, with z_{ab} specified as the initial arrival probability, and z_{aa} by an initial establishment probability. The remaining elements are $z_{ba} = (1 - z_{aa})$ and $z_{bb} = (1 - z_{ab})$.

If we denote the probabilities of the events *a* and *b* occurring at any time *t* by $z_a(t)$ and $z_b(t)$, respectively, the probability of *a* occurring in time step $t+1$ given that *b* has occurred in time step *t* can be expressed as:

$$z_a(t+1) = \sum_b z_{ab} z_b(t). \qquad (16)$$

If $z(t)$ is a column vector with elements $z_a(t)$ and $z_b(t)$, we can use the transition matrix to express equation (16) as:

$$z_a(t+1) = Zz(t). \qquad (17)$$

By applying this previous equation repeatedly, we obtain:

$$z(t) = Z^t z(0). \qquad (18)$$

If our Markov chain is regular the vector $z(t)$ will converge to a unique vector z as *t* increases (Hinchy and Fisher 1991; Moran 1984)[8]. Independent of the state of the world in time step *t*, we can accurately predict the probability of being in either state *a* or *b* after several time periods, $t+n$. Hence, the probability of event *a* occurring in any given time period will reduce to a constant value after several time steps. Since we are only concerned with event *a* (i.e. *Foc* TR4 occurrence) in a given region (*i*), we denote $z_a(t)$ as z_i.

If the *Foc* TR4 arrives in a new production region we assume it will become naturalized over time. No large country or region has successfully removed the disease once it has become established, so we are therefore compelled to assume this would also be the case in the Australian context. This is not to say there will be no action taken on behalf of plantation owners to protect their banana operation from *Foc* TR4. But, we assume that this will not be aimed at eradication. Instead, a containment strategy is most likely to be employed due to the relatively limited means by which the disease can spread via natural dispersal mechanisms.

After *Foc* TR4 arrives in any region *i*, the total damage banana producers in this region experience because of the disease in time period *t* (d_{it}) is estimated by:

$$d_{it} = Y_{it} P_t A_{it} + V_{it} A_{it} \tag{19}$$

where: Y_{it} is the mean change in yield resulting from infection (assumed 100 per cent) in region *i* in year *t*; P_t is the prevailing domestic price for bananas in year *t*; V_{it} is the increase in variable cost of production per hectare induced by *Foc* TR4 on-plantation management methods in region *i* in year *t*; and A_{it} is the area infected with *Foc* TR4 in region *i* year *t* weighted by the probability of infection (i.e. z_i, from above) and density of infection.

A stratified diffusion model combining both short and long distance dispersal processes is used to predict the area potentially affected by *Foc* TR4 post-establishment in each region *i* in time period *t*, A_{it}. Parameter estimates for this model appear in Table 12, and are explained below. We hasten to point out that despite the destructive potential of *Foc* TR4, a great deal of uncertainty surrounds the principal mode of disease spread. As a soil borne pathogen, the disturbance and movement of soil on machinery and equipment is the most apparent means of spread from affected to non-affected areas. The efficacy of spread via water splash and wind-borne dust has not been researched. In additional, dispersal via insect vectors (e.g. Banana weevil borer (*Cosmopolites sordidus*)) has not been discounted. It follows that for the purposes of this analysis, some parameters have been specified using relatively broad ranges.

[8] The initial probabilities attached to events *a* and *b* will be dependent on the effectiveness of quarantine and surveillance policies in place at the outset of the analysis. Changes to these policies will alter these probabilities as the likelihood of pre- and post-border detection changes (Hinchy and Fisher 1991). Analyses of policy effectiveness are easily accommodated using this framework, but are not undertaken here.

Table 12. Parameter values for the Panama disease (tropical race 4) model.

Description	Values
Probability of entry and establishment in production regions (i.e. States) previously unaffected, z_{ab}. [a]	Uniform(0.0,1.0×10^{-6})
Detection probability in areas previously unaffected.	Binomial(1.0,0.6)
Population diffusion coefficient, D (m^2/yr). [b]	Pert(1.0×10^{4},1.5×10^{4},2.0×10^{4})
Area currently infected (ha)	Pert(5,10,15)
Minimum area infected immediately upon entry, A^{min} (m^2).	1.0×10^{3}
Maximum area infected, A^{max} (m^2). [c]	1.4×10^{8}
Intrinsic rate of infection and density increase, r(yr^{-1}). [d]	Pert (0.10,0.15,0.20)
Minimum infection density, N^{min} (#/m^2).	1.0×10^{-4}
Maximum infection density, K (#/m^2). [d]	Pert(100,550,1000)
Minimum number of satellite sites generated in a single time step, S^{min} (#).	0
Maximum number of satellite sites generated in a single time step, S^{max} (#). [d]	Pert(0.0,2.5,5.0)
Intrinsic rate of new foci generation per unit area of infection, μ (#/m^2). [d]	Pert(1.0×10^{-2},3.0×10^{-2},5.0×10^{-2})
Discount rate (%).	5
Supply elasticity. [e]	Uniform(0.2,0.8)
Demand elasticity. [e]	Uniform(-1.1,-1.0)
Prevailing market price of bananas in the first time step ($/T). [c]	1,900
Maximum area considered for eradication, A^{erad} (ha).	0
Treatment costs upon detection ($/ha). [f]	Pert(8.0×10^{3},9.0×10^{3},1.0×10^{4})
Yield reduction despite control (%). [g]	100

[a] Biosecurity Australia (2008); [b] Derived from Sapoukhina *et al.* (2010); [c] ABS (2011), Note 1ha = 10 000m^2; [d] Specified with reference to Cook (2003) and Waage *et al.* (2005) using distributions defined in Biosecurity Australia (2001); [e] Ulubasoglu *et al.* (2011); [f] Assumes average density of planting of 2,000 stems/ha and removal, transport, destruction and chemical costs amounting to approximately $4.50 per tree. This is inclusive of labour (team of three at $50/hr per person), bulldozing equipment ($100/hr at 20 hours per hectare), truck hire ($75/hr) and incendiaries ($60/ha for green waste). No chemical buffer zone is deemed necessary as the pathogen is soil borne; [g] Over 95 per cent of bananas grown in Australia are of the Cavendish variety which is susceptible to *Foc* TR4 (Hennessy, Walduck *et al.* 2005). If a plantation becomes infected with the disease it can not be controlled using fungicides, nor can it be eradicated from the soil using fumigants. Research conducted in the Northern Territory has identified a number of resistant varieties, but this stage none are suitable as immediate replacements for the Cavendish variety (Daly and Walduck 2006). Hence, after infection yield loss is assumed to be 100 per cent.

The model is derived from the reaction diffusion models originally developed by Fisher (1937) which have been shown to provide a reasonable approximation of the spread of a diverse range of organisms (Cook, Carrasco *et al.* 2011b; Dwyer 1992; Holmes 1993; McCann, Hastings *et al.* 2000; Okubo and Levin 2002). These models assert that an invasion diffusing from a point source will eventually reach a constant asymptotic radial spread rate of $2\sqrt{r_i D_{ij}}$ in all directions, where r_i describes a growth factor for *Foc* TR4 per year in region *i* (assumed constant over all infected sites) and D_{ij} is a diffusion coefficient for an infected site *j* in region *i* (assumed constant over time) (Cook, Carrasco *et al.* 2011b; Hengeveld 1989; Lewis 1997; Shigesada and Kawasaki 1997). Hence, we assume that the original infection (i.e. the first of a probable series of sites, *j*) takes place in a homogenous environment in region *i* and expands by a diffusive process such that area infected at time *t*, a_{ijt}, can be predicted by:

$$a_{ijt} = z_i\left[\pi\left(2t\sqrt{r_i D}_{ij}\right)^2\right] = z_i\left(4D_{ij}\pi r_i t^2\right). \quad (20)$$

By assuming D_{ij} is constant across all sites *j* we ignore demographic stochasticity and consequent non-uniform invasion.

The density of *Foc* TR4 infection within a_{ijt} influences the control measures required to counter the effects of infection, and thus partially determines the value of A_{it}. We assume that in each site *j* in region *i* affected, the infection density, N_{ijt}, grows over time period *t* following a logistic growth curve until the carrying capacity of the host environment, K_{ij}, is reached:

$$N_{ijt} = \frac{K_{ij} N_{ij}^{\min} e^{r_i t}}{K_{ij} + N_{ij}^{\min}(e^{r_i t} - 1)}. \quad (21)$$

Here, $N_{ij}^{\min}$ is the size of the original infection at site *j* in region *i* and r_i is the intrinsic rate of density increase in region *i* (assumed to be the same as the intrinsic rate of infection increase) (Cook, Fraser *et al.* 2011c).

In addition to a_{ijt} and N_{ijt}, the size of A_{it} depends on the number of nascent foci or *satellite* infection sites in year *t*, s_{it}, which can take on a maximum value of $s_i^{\max}$ in any year (Moody and Mack 1988). These sites result from events external to the initial outbreak itself, such as weather phenomena, animal or human behaviour, which periodically jump the expanding infection beyond the infection front (Cook, Fraser *et al.* 2011c). We use a logistic equation to generate changes in s_{it} as an outbreak continues:

$$s_{it} = \frac{s_i^{\max} s_i^{\min} e^{\mu_i t}}{s_i^{\max} + s_i^{\min}(e^{\mu_i t} - 1)} \quad (22)$$

where μ_i is the intrinsic rate of new foci generation in region *i* (assumed constant over time) and $s_i^{\min}$ is the minimum number of satellite sites generated in region *i*.

Given equations (20)-(22), we can express A_{it} as:

$$A_{it} = \sum_{j=1}^{m} \left(a_{ijt} N_{ijt}\right)^{s_{it}} \text{ where } 0 \le A_{it} \le A_i^{\max} \ . \tag{23}$$

Spread area, infection density and the number of sites are combined with the probability of entry and establishment in an expression of probability-weighted, or expected damage over time. Assuming a discount rate δ, the present value of expected damage after τ time periods (TC^{P}) is:

$$\mathrm{TC}^{\mathrm{P}} = \sum_{t=1}^{\tau} \left(1-\delta\right)^{-t} . \sum_{i=1}^{n} d_{it} \ . \tag{24}$$

This expression provides us with a probability weighted estimate of invasion-induced producer losses over time, and therefore provides an indication of the economic significance of *Foc* TR4 over time. It is *not* a measure of what damage will be inflicted by a species if it is introduced to a region tomorrow. Rather, it provides a measure of expected damage taking into account uncertainty in the time of arrival and change in abundance and distribution of infection over time after arrival.

3.5.5.3. Results

Foc TR4 is assumed to be absent from all production regions other than the Northern Territory at the beginning of time period one. We assume entry and establishment in Queensland, New South Wales and Western Australia is likely to occur at some point or multiple points over the estimation period. Therefore, the resultant expected spread area values calculated from 10,000 iterations of the model are positive. These projections have been aggregated across all production regions to produce Figure 26. Spread is predicted to be relatively slow, but by the end of the estimation period *Foc* TR4 is estimated to affect 2,500 hectares of commercial banana plantations.

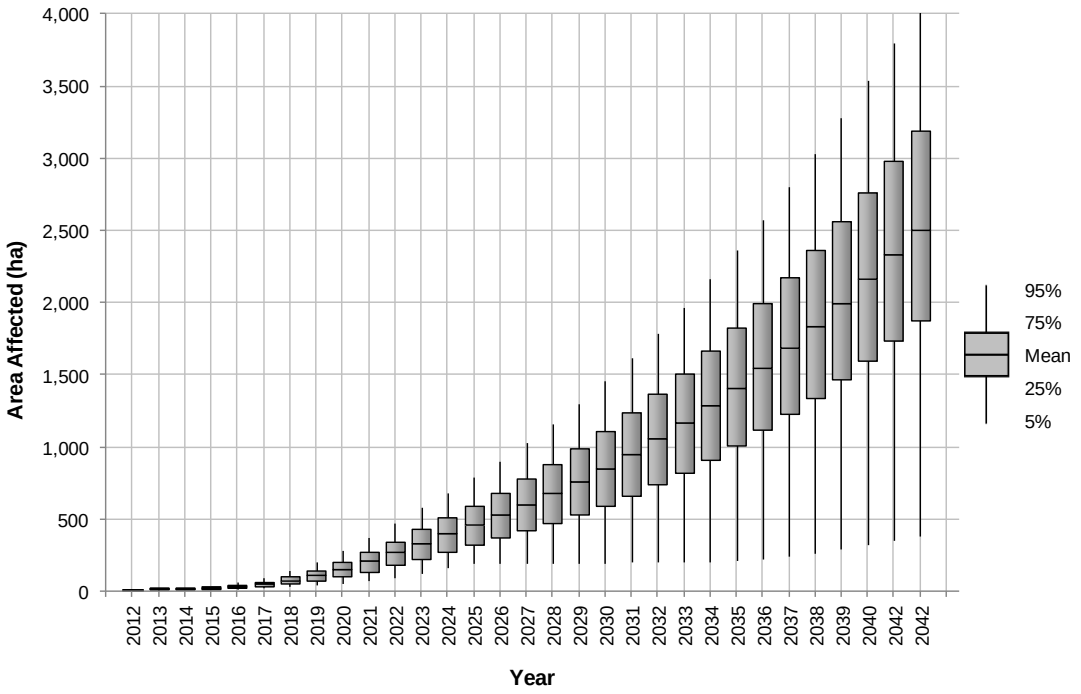

Figure 26. Expected area of commercial banana plantations affected by Panama disease in Australia.

Figure 27 illustrates how the resultant TC^P (i.e. see equation (24)) is expected to change over the 30-year period of the simulation. Here, the mean values of TC^P predicted by the model in each year is plotted with 10 per cent and 50 per cent confidence intervals. All projected benefits are discounted at 5 per cent per annum. By the 30th year, TC^P is expected to average just under $75.5 million per year.

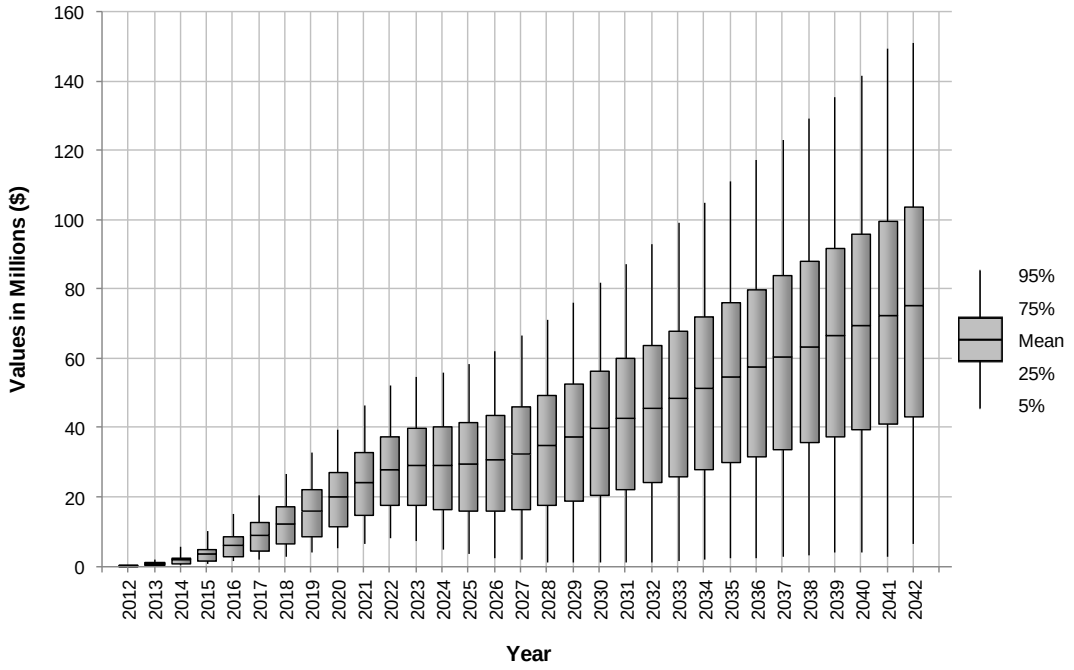

Figure 27. Predicted industry losses from Panama disease in Australia.

Given that *Foc* TR4 spreads relatively slowly of its own accord, overall cost increments are expected to be relatively modest from year to year.

3.5.5.4. Discussion

The present value of the losses incurred by the banana industry could exceed $75 million per year over the 30 year period we have projected possible industry losses. However, as Figure 27 shows, the confidence intervals around our estimate are broad owing to the large amount of uncertainty surrounding the disease. In all likelihood, *Foc* TR4 can be contained by fencing off affected areas, injecting infected plants with herbicides, and restricting movement of infected planting stock and spore bearing material, as illustrated by experiences in the Northern Territory. This implies that if or when an outbreak does occur, containment procedures by government and/or individual growers could substantially reduce the impact on industry by preventing or delaying further spread. Successful eradication appears unlikely, as the pathogen is likely to persist in the soil for an almost indefinite period, even after all banana plants have been destroyed. Permanent containment is probably a more realistic goal in the event of an incursion (McElwee 2000).

In view of the uncertainty surrounding many of the parameters used to describe the *Foc* TR4 infection and spread process, the sensitivity of the change in TC^{P} to the key assumptions of the model must be tested to gauge the robustness of predictions. Parameters were sampled from a uniform distribution with a maximum (minimum) of +50 per cent (-50 per cent) of the original values entered in to the model using Monte Carlo simulation. The Spearman's rank correlation coefficients relating the sampled model parameter values and the change in TC^{P} were then calculated. The results appear in Figure 28.

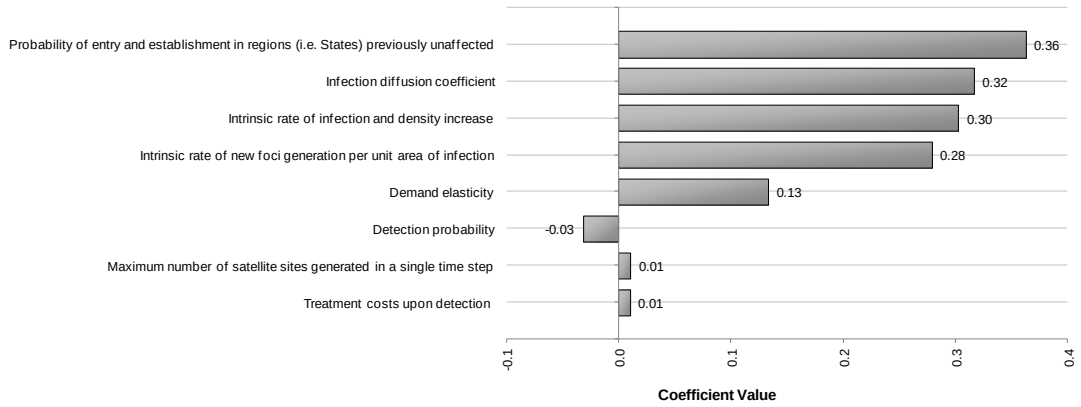

Figure 28. Sensitivity analysis for the Panama disease model.

The sensitivity tests indicate that the model is highly responsive to five parameters. These include the probability of entry and establishment in regions (i.e. States) previously unaffected (with a Spearman's rank correlation coefficient of 0.36), the infection diffusion coefficient (0.32), the intrinsic rate of infection and density increase (0.30), the intrinsic rate of new foci generation per unit area of infection (0.28) and the demand elasticity (0.13).

The sensitivity of the supply elasticity is to be expected given that Australia has *Foc* TR4 present, and its further spread is unlikely to cause any relaxation of phytosanitary requirements on imported produce. This means that as banana plants are infected they cannot be substituted for by imports, which in turn means that the domestic price of bananas is likely to rise in response to disease spread. The degree to which this will occur is determined by the elasticity of demand for bananas which indicates the effects of changes in different variables that determine the quantity of bananas demanded by consumers. One of these is price. If the price-elasticity of demand is relatively high, a disease induced contraction of supply to the domestic market will lead to a relatively large reduction in demand as the price rises, and vice versa. We have specified demand elasticity using the Ulubasoglu *et al.* (2011), which is the most recent and comprehensive study available containing demand elasticity estimates for different food products in Australia.

Given that the remaining four sensitive parameters (i.e. probability of entry and establishment in regions (i.e. States) previously unaffected, infection diffusion coefficient, intrinsic rate of infection and density increase and the intrinsic rate of new foci generation per unit area of infection) are scientific in nature and we have very little published scientific research on which to base our assumptions, it is extremely difficult to comment on the robustness of our predictions. However, the parameter estimates we have used have been specified with reference to other peer-reviewed studies using similar predictive models that have included soil-borne plant pathogens, including Cook *et al.* (2006), Waage *et al.* (2005) and Cook (2003). Our parameter estimates are within very similar ranges to those used in these studies, but beyond this subjective comparison we are unable to comment further on the robustness of our analysis.

We therefore stress the need for further research on *Foc* TR4. The fact that a staple food for many developing county populations around the world could potentially be destroyed by a disease researchers know so little about it is of definite concern. Even if the spread of the disease can be slowed if it is detected early and appropriate plantation hygiene and quarantine measures are put in place, the lack of resistant cultivars still makes *Foc* TR4 an extremely serious biosecurity threat.

3.5.6. An assessment of the benefits of yellow Sigatoka control in the Queensland Northern Banana Pest Quarantine Area

3.5.6.1. Introduction

The Sigatoka disease complex affects bananas in many countries. As previously mentioned in section 3.5.6.1, the disease yellow Sigatoka (*Mycosphaerelia musicola*) is naturalised and actively controlled in Australia through intensive fungicide treatments and diseased leaf removal (Henderson, Pattemore *et al.* 2006). Although less virulent than black Sigatoka (*M. fijiensis*), *M. musicola* also imposes costs on affected banana growers, particularly in the highly productive Innisfail-Tully areas of north Queensland. These costs consist mainly of de-leafing expenses.

State government-imposed standards for de-leafing to minimise the risk of *M. musicola* spread and impact are in place in Queensland. Here, the Plant Protection Regulation 2002 was put in place under the Plant Protection Act 1989, and defined six banana pest quarantine areas (Queensland *Plant Protection Regulation 2002*). Of these, the Northern Banana Pest Quarantine Area (NBPQA) is the most significant, encompassing over 80 per cent of the State's banana production. This imposed an obligation on the owner of land in a pest quarantine area to treat every banana plant by removing every leaf from the plant that has visible symptoms of *M. musicola* (and another endemic disease, banana leaf speckle (*M. musae*)) on more than 15 per cent of any leaf at any time between 1 November and 31 May or on more than 30 per cent of any leaf at any time between 1 June and 31 October.

An amendment to the Plant Protection Regulation 2002, the Plant Protection Amendment Regulation (No. 4) 2003, was subsequently put in place in response to concerns that the de-leafing standards initially imposed were too high. In particular, during wet season conditions in the NBPQA the 15 per cent de-leafing threshold was deemed insufficient to prevent *M. musicola* and *M. musae* from spreading. Moreover, the 30 per cent action level in the dry season was thought to be far too high for wet weather conditions highly conducive to disease spread (Queensland *Plant Protection Amendment Regulation (No. 4) 2003*). The Amendment imposed a threshold of 5 per cent throughout the year in the NBPQA.

As deleterious as these amended regulations appear to be in terms of the foliage carried by commercial banana plants, the impact on production volume is expected to be minimal. During their life, individual banana plants may produce 30 or more leaves, which is surplus to their phosynthetic needs. The oldest leaves are shed at a rate of approximately 1 leaf every 10-12 days so that when the fruit bunch emerges from the top of the pseudostem the plant has an average of 15 leaves. After the bunch shoots no new leaves are produced. The oldest leaves of the plant continue to fall until, at harvest, 6-8 leaves remain (Ostmark 1974).

While the incidence of leaf disease is expected to be reduced if stricter thresholds are implemented and maintained over time, additional costs to banana growers in the NBPQA will apply. These include substantial increases in chemical treatment and application costs in addition to more rigorous de-leafing cycles. In this section we provide an estimate of the likely change in net returns to the banana industry in the NBPQA from adopting the 5 per cent de-leafing threshold.

3.5.6.2. Methods

The stochastic simulation model used in this assessment determines total *expected* (or probability-weighted) damage from *M. musicola* in the NBPQA over a 30-year period under both a 15 per cent and a 5 per cent de-leafing threshold. Uncertain or variable parameters are specified as probability distributions, and 10,000 model iterations are run using values randomly sampled across the range of each distribution using a Latin hypercube sampling algorithm.

The total damage banana producers in the NAPQA experience because of the disease in time period *t* (d_t) is estimated by:

$$d_t = Y_t P_t A_t + V_t A_t \tag{25}$$

where: Y_t is the mean change in yield resulting from infection (assumed 100 per cent) year *t*; P_t is the prevailing domestic price for bananas in year *t*; V_t is the increase in variable cost of production per hectare induced by *M. musicola* on-plantation management methods in year *t*; and A_t is the area infected with *M. musicola* in year *t*.

A stratified diffusion model combining both short and long distance dispersal processes is used to predict the area potentially affected by *M. musicola* in time period *t*, A_t . Parameter estimates for this model appear in Table 13, and are explained below.

The model is derived from the reaction diffusion models originally developed by Fisher (1937) which have been shown to provide a reasonable approximation of the spread of a diverse range of organisms (Cook, Carrasco *et al.* 2011b; Dwyer 1992; Holmes 1993; McCann, Hastings *et al.* 2000; Okubo and Levin 2002). These models assert that an invasion diffusing from a point source will eventually reach a constant asymptotic radial spread rate of $2\sqrt{rD_j}$ in all directions, where r describes a growth factor for *M. musicola* per year in the NBPQA (assumed constant over all infected sites) and D_j is a diffusion coefficient for an infected site *j* in the NBPQA (assumed constant over time) (Cook, Carrasco *et al.* 2011b; Hengeveld 1989; Lewis 1997; Shigesada and Kawasaki 1997). Hence, we assume that the original infection (i.e. the first of a probable series of sites, *j*) takes place in a homogenous environment in the NBPQA and expands by a diffusive process such that area infected at time *t*, a_{jt} , can be predicted by:

$$a_{jt} = \pi\left(2t\sqrt{rD_j}\right)^2 = 4D_j\pi rt^2 . \tag{26}$$

By assuming D_j is constant across all sites *j* we ignore demographic stochasticity and consequent non-uniform invasion. Since the two control strategies we are considering (i.e. 5 per cent and 15 per cent de-leafing regulations) are very similar, many of the parameters remain unchanged in both scenarios. But, D is assumed to be lower under the 5 per cent de-leafing threshold due to increased chemical suppression limiting local dispersal opportunities for the disease.

Table 13. Parameter values for the yellow Sigatoka model.

Description	15% de-leafing threshold	5% de-leafing threshold
Detection probability (%).	100	100
Population diffusion coefficient, D (m^2/yr). [a]	Pert(2.0×10^3,3.5×10^3,5.0×10^3)	Pert(0.0,1.0×10^2,2.0×10^2)
Percentage of total NBPQA plantation area infected in the first time step (%). [b]	Pert(0.0,1.5,3.0)	Pert(0,2,4)
Minimum area infected, A^{min} (m^2).	1.0×10^3	1.0×10^3
Maximum area infected, A^{max} (m^2). [c]	9.8×10^7	9.8×10^7
Intrinsic rate of infection and density increase, r(yr^{-1}). [a]	Pert (0.00,0.01,0.02)	Pert (0.00,0.01,0.02)
Minimum infection density, N^{min} (#/m^2).	1.0×10^{-4}	1.0×10^{-4}
Maximum infection density, K (#/m^2). [a]	Pert(100,550,1000)	Pert(100,550,1000)
Minimum number of satellite sites generated in a single time step, S^{min} (#).	1	1
Maximum number of satellite sites generated in a single time step, S^{max} (#). [a]	Pert(0,5,10)	Pert(0,5,10)
Intrinsic rate of new foci generation per unit area of infection, μ (#/m^2). [a]	Pert(1.0×10^{-2},3.0×10^{-2},5.0×10^{-2})	Pert(1.0×10^{-2},3.0×10^{-2},5.0×10^{-2})
Discount rate (%).	5	5
Supply elasticity. [d]	Uniform(0.2,0.8)	Uniform(0.2,0.8)
Demand elasticity. [d]	Uniform(-1.1,-1.0)	Uniform(-1.1,-1.0)
Prevailing market price of bananas in the first time step ($/T). [c]	1,900	1,900
Maximum area considered for eradication, A^{erad} (ha).	0	0
Treatment costs upon detection - chemical ($/ha). [f]	Pert(8.0×10^3,1.1×10^4,1.3×10^4)	Pert(1.6×10^4,5.0×10^4,6.6×10^4)
Treatment costs upon detection – de-leafing ($/ha). [g]	Pert(1.4×10^3,2.1×10^3,2.8×10^3)	Pert(2.1×10^3,3.1×10^3,3.2×10^3)
Yield reduction despite control (%).	Pert(0.0,2.5,5.0)	Pert(0.0,0.5,1.0)

[a] Specified with reference to Cook (2003) and Waage *et al.* (2005); [b] Derived from Peterson *et al.* (2005); [c] ABS (2011), Note 1ha = 10 000m^2; [d] Ulubasoglu *et al.* (2011); [e] Assumes: (*i*) average density of planting of 2,000 stems/ha and removal, (*ii*) control of *M. musicola* in the NBPQA involves applications of dithane (at 3kg/ha or $21.60/ha) and oil (at 3L/ha or $8.85/ha) at weekly intervals during the wet season (Cook 2003), (*iii*) it is desirable for growers to rotate the use of dithane and oil with propiconazole (at 0.3L/ha or $22.20/ha) to manage resistance (Cook 2003), (*iv*) 15–25 cycles of fungicides are used for control of *M. musicola* in the NBPQA to comply with a 15% de-leafing threshold, (*v*) an additional 5-10 spray cycles are needed to comply with a 5% de-leafing threshold; [g] De-leafing plantations to control *M. musicola* to a 15% threshold occurs up to 15 times per season. Assume an additional 5-10 de-leafing cycles are necessary to achieve a 5% threshold at a cost of $140/ha each.

The density of *M. musicola* infection within a_{jt} influences the control measures required to counter the effects of infection, and thus partially determines the value of A_t. We assume that within each site *j* affected, the infection density, N_{jt}, grows over time period *t* following a logistic growth curve until the carrying capacity of the host environment, K_j, is reached:

$$N_{jt} = \frac{K_j N_j^{\min} e^{rt}}{K_j + N_j^{\min}(e^{rt} - 1)}. \quad (27)$$

Here, $N_j^{\min}$ is the size of the original infection at site *j* and r is the intrinsic rate of density increase (assumed to be the same as the intrinsic rate of infection increase) (Cook, Fraser *et al.* 2011c).

In addition to a_{jt} and N_{jt}, the size of A_t depends on the number of nascent foci or *satellite* infection sites in year *t*, s_t, which can take on a maximum value of $s^{\max}$ in any year (Moody and Mack 1988). These sites result from events external to the initial outbreak itself, such as weather phenomena, animal or human behaviour, which periodically jump the expanding infection beyond the infection front (Cook, Fraser *et al.* 2011c). We use a logistic equation to generate changes in s_t as an outbreak continues:

$$s_t = \frac{s^{\max} s^{\min} e^{\mu t}}{s^{\max} + s^{\min}(e^{\mu t} - 1)} \quad (28)$$

where μ is the intrinsic rate of new foci generation (assumed constant over time) and $s^{\min}$ is the minimum number of satellite sites generated.

Given equations (24)-(28), we can express A_t as:

$$A_t = \sum_{j=1}^{m} \left(a_{jt} N_{jt}\right)^{s_t} \text{ where } 0 \le A_t \le A^{\max}. \quad (29)$$

Spread area, infection density and the number of sites are combined with the probability of entry and establishment in an expression of probability-weighted, or expected damage over time. Assuming a discount rate δ, the present value of expected damage after τ time periods (TC^{P}) is:

$$\mathrm{TC}^{\mathrm{P}} = \sum_{t=1}^{\tau} (1-\delta)^{-t} . d_t. \quad (30)$$

This expression provides us with an estimate of invasion-induced producer losses over time, and therefore provides an indication of the economic significance of *M. musicola* over time given a de-leafing protocol. If we denote the total expected damage under a 15 per cent and a 5 per cent de-leafing protocol $\mathrm{TC}^{\mathrm{P}}_{15\%}$ and $\mathrm{TC}^{\mathrm{P}}_{5\%}$ a, respectively, we can

determine the likely change in expected damage (ΔTC^P) from adopting the 5 per cent protocol as:

$$\Delta TC^P = \Delta TC^P_{15\%} - \Delta TC^P_{5\%}. \quad (31)$$

If indeed the 5 per cent de-leafing protocol is the most effective at reducing *M. musicola* prevalence and impact over time, we would expect $\Delta TC^P > 1$.

3.5.6.3. Results

M. musicola is assumed to be present within the NBPQA at the beginning of time period one. Therefore, the resultant expected spread area values calculated from 10,000 iterations of the model are positive, as revealed by Figure 29. Spread is predicted to be very slow in both the 5 per cent and 15 per cent de-leafing protocol scenarios due to the effectiveness of chemical and de-leafing treatments applied simultaneously.

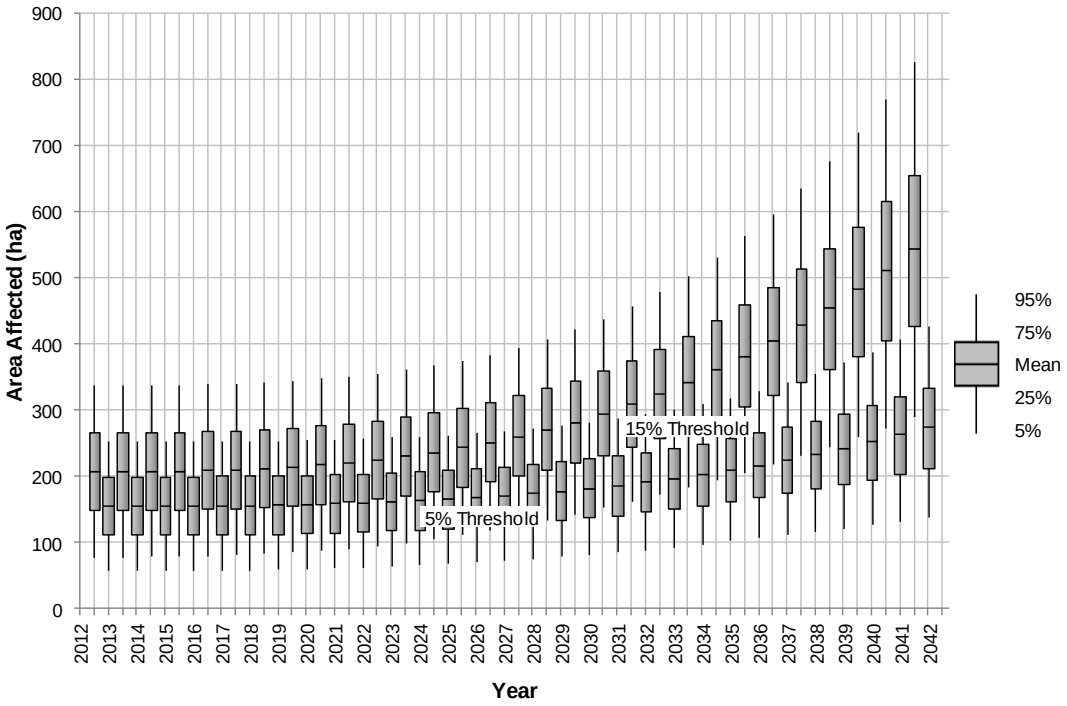

Figure 29. Expected area of commercial banana plantations affected by yellow Sigatoka in Australia under different management guidelines.

Figure 30 illustrates how the resultant $TC^P_{15\%}$ and $TC^P_{5\%}$ (i.e. see equations (30)-(31)) are expected to change over the 30-year period of the simulation. Here, the mean values of $TC^P_{15\%}$ and $TC^P_{5\%}$ predicted by the model in each year are plotted with 10 per cent and 50 per cent confidence intervals. All projected benefits are discounted at 5 per cent per annum. By the 30th year, $TC^P_{15\%}$ is expected to average just under $30 million per year, and $TC^P_{5\%}$ just under $15 million per year.

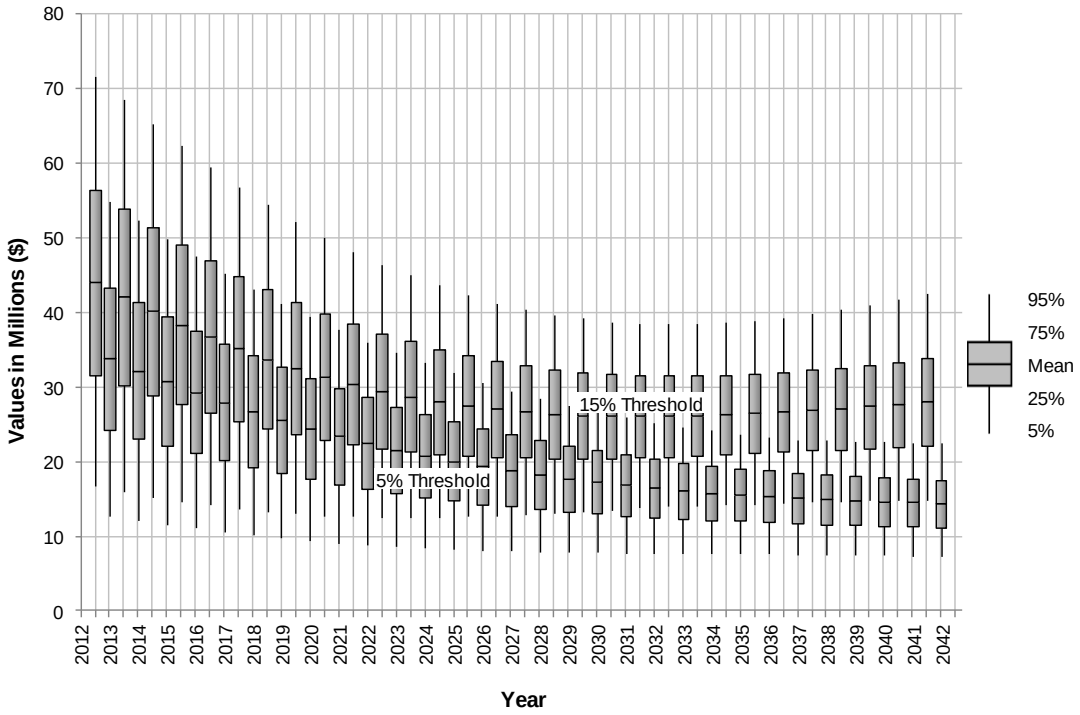

Figure 30. Predicted industry losses from yellow Sigatoka in Australia under different management guidelines.

Note that despite the area affected by the disease remaining relatively constant in both control scenarios, the erosive effects of the discount rate lead to a gradual decline in average annual industry damage.

Figure 31 illustrates how the difference between $\mathrm{TC}^{\mathrm{P}}_{15\%}$ and $\mathrm{TC}^{\mathrm{P}}_{5\%}$ (i.e. $\Delta\mathrm{TC}^{\mathrm{P}}$ in equation (31)) is expected to change over time, and therefore the relative merit in the banana industry choosing a 5 per cent de-leafing protocol over a 15 per cent protocol in the NBPQA.

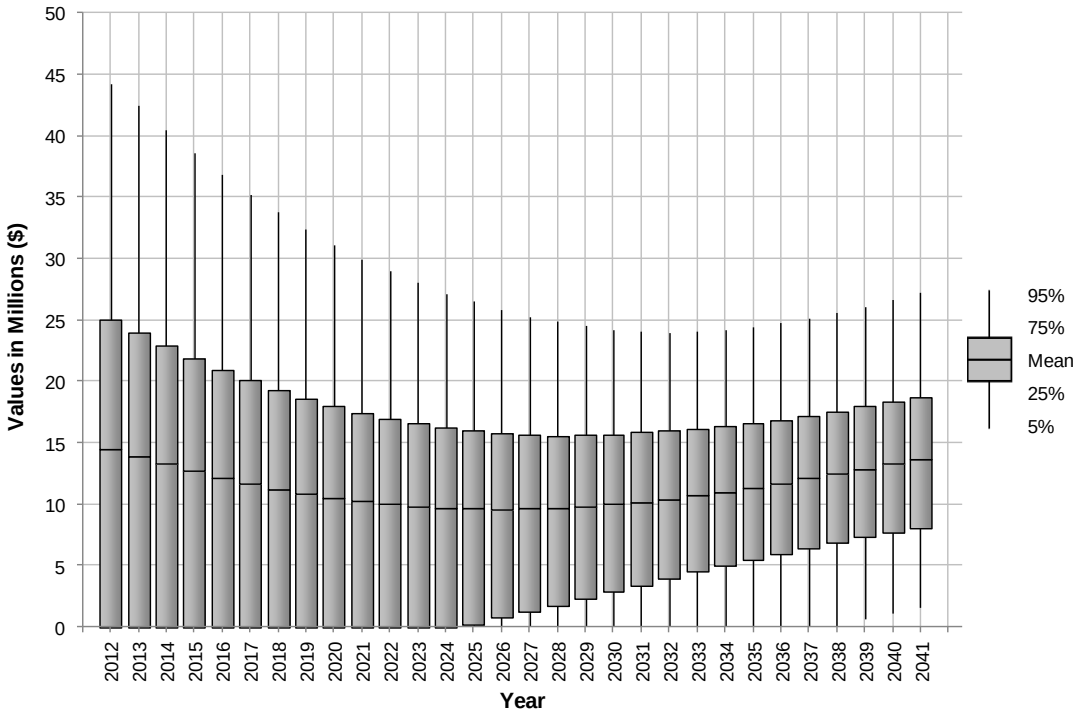

Figure 31. Predicted gross benefit of adopting a 5 per cent de-leafing threshold for yellow Sigatoka suppression in the NBPQA relative to a 15 per cent protocol.

3.5.6.4. Discussion

Queensland *Plant Protection Amendment Regulation (No. 4) {, 2003 #796}* includes a benefit cost analysis of the change in disease threshold in the NBPQA which reveals little about the flow of producer benefits over time. All figures quoted in the analysis are *net* figures, inclusive of both the production benefits of disease control and the costs of fungicide applications and de-leafing. It is estimated that the net impact of the 5 per cent disease threshold will be -$50,000 in the first year, -$20,000 in the second year, $0 in the third year, $200,000 in the fourth year and $400,000 in subsequent years (Queensland *Plant Protection Amendment Regulation (No. 4) 2003*). Although these net returns are arbitrarily stated to include changes in yield and fruit quality and reduced disease control costs once the lower disease threshold is achieved, no details are provided as to their derivation.

The analysis provided in this section allows a more detailed estimation of costs and benefits over time. The notes accompanying Table 13 above indicate that we have used a series of technical assumptions about the way grower behaviour is likely to change with a 5 per cent disease threshold compared to a 15 per cent threshold. Specifically, we assume:

1. An average density of planting of 2,000 stems per hectare and removal.
2. Control of *M. musicola* in the NBPQA involves applications of dithane (at 3 kilograms per hectare or $21.60 per hectare) and oil (at 3 litres per hectare or $8.85 per hectare) at weekly intervals during the wet season (Cook 2003).

3. Growers rotate the use of dithane and oil with propiconazole (at 0.3 Litres per hectare or $22.20 per hectare) to manage resistance (Cook 2003).
4. 15 to 25 cycles of fungicides are used for control of *M. musicola* in the NBPQA to comply with a 15 per cent de-leafing threshold, and an additional 5 to10 spray cycles are needed to comply with a 5 per cent de-leafing threshold.
5. De-leafing plantations to control *M. musicola* to a 15 per cent threshold occurs up to 15 times per season, and an additional 5 to 10 de-leafing periods are necessary to achieve a 5 per cent threshold at a cost of $140.00 per hectare each.

Extrapolating across the entire NBPQA, aggregated costs are provided in Table 14.

Table 14. Predicted cost of adopting a 5 per cent de-leafing threshold for yellow Sigatoka suppression in the NBPQA relative to a 15 per cent protocol aggregated across the region.

Description	15% de-leafing threshold (A)	5% de-leafing threshold (B)	B-A
Chemical treatment costs ($ million)	115.4	146.1	31.3
De-leafing costs ($ million)	19.6	32.0	12.5
Total ($ million)	134.9	178.7	43.8

Note that the costs indicated in Table 14 are total costs estimated across the whole NBPQA attributable to compliance with the leaf disease thresholds in a single year. It follows that the right hand column labelled B-A represents the increase in chemical and de-leafing costs imposed by a lower threshold of 5 per cent. By comparing the present value (i.e. the discounted, or 'real') value of these annual cost increments to the predicted benefits derived from Figure 31, we can estimate the likely change in *net* returns (i.e. benefits minus costs) to the NBPQA from adopting this lower standard. The comparison is summarised in Figure 32, below.

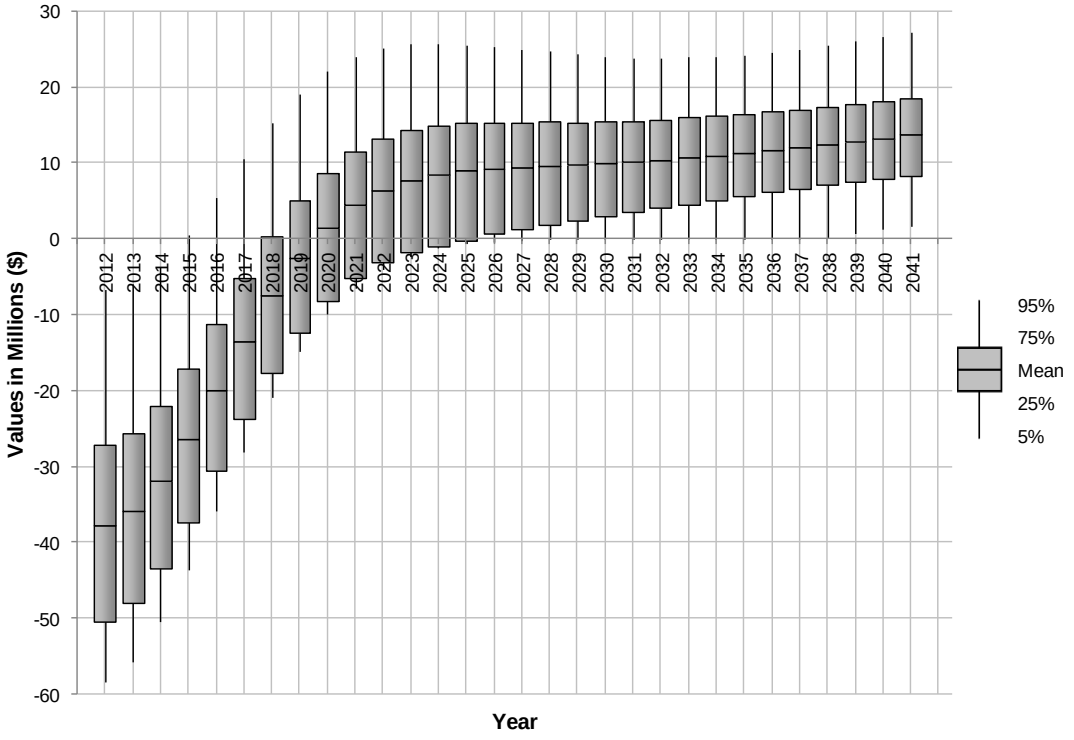

Figure 32. Predicted net benefit of adopting a 5 per cent de-leafing threshold for yellow Sigatoka suppression in the NBPQA relative to a 15 per cent protocol.

Figure 32 shows the full extent of uncertainty surrounding possible net returns to the region. Initially, due to the increased cost of compliance to the 5 per cent leaf disease threshold, net costs (i.e. a surplus of costs over benefits) are likely to result in the short term. However, after a period of time (between seven and 14 years) the benefits generated by lower *M. musicola* prevalence and impact begin to outweigh compliance costs. By the end of the simulation period, net benefits are likely to be over $13 million per annum.

On average, over the entire 30 years of the simulation, the mean net benefit to the banana industry in the NBPQA of adopting the lower leaf disease threshold is estimated to be $1.4 million. Considering this is spread over approximately 10,400 hectares of bananas, the impact of the change in disease thresholds appears to be marginal. If we calculate average net returns over a 20-year period, we find that a net cost of the order of -$3.4 million per annum is likely to result. As Figure 32 clearly shows, this is due to the large net costs concentrated in the early years of adopting the new threshold. The further forward in time we project, the larger the likely returns to the banana industry of imposing the stricter leaf disease threshold.

3.5.7. Summary

The non-spatial models developed for the ABGC have been used to generate detailed impact assessment for five key plant pathogens, each requiring subtly different modelling approaches.

BBTV is established in Australia, but is targeted for eradication from banana growing regions of Queensland and northern New South Wales. We develop a partial budgeting approach using a stratified diffusion spread model to simulate the likely benefits of exclusion of this virus from commercial banana plantations over time relative to a nil management scenario in which no surveillance or containment activities take places. Using Monte Carlo simulation to generate a range of possible future incursion scenarios, we predict the exclusion benefits of the disease will avoid Aus$15.9-27.0 million in annual losses for the banana industry. For these exclusion benefits to be reduced to zero would require a bunchy top re-establishment event in commercial banana plantations three years in every four.

M. fijiensis has been eradicated from Australia relatively recently and strict quarantine measures are still in place to protect against its reintroduction. These measures make it prohibitively expensive for foreign suppliers to land product in Australia. Strict though these import requirements are and small though the risk of reintroduction may be, the potential damage that could be caused to the Australian banana industry is potentially huge. We provide quantitative estimates of these potential damages and discuss the implications for Australia's acceptable level of protection using the example of black Sigatoka *M. fijiensis*. We find that if there were no quarantine restrictions, expected producer losses to the disease exceed $200 million. With quarantine measures in place annual expected damages over a 20 year period are still substantial at just under $100 million.

The same dynamic partial equilibrium model is used to estimate potential economic impact of *R. solanacearum* on the Australian banana industry. This disease is found throughout many parts of the world where bananas are cultivated, and has proven a serious biosecurity threat as there are no treatments known to be effective against it other than destroying infected plants. We find that if there were no phytosanitary measures in place against imported bananas, expected producer losses to *R. solanacearum* could amount to approximately $100 million per year after 20 years. However, there is a lot of uncertainty in our predictions as there is a relatively high likelihood of successful eradication upon detection provided this takes place very early in the invasion process. With quarantine measures in place annual expected damages over the same period remain large at around $30 million.

Foc TR4 is a serious soil-borne disease considered to be one of the most severe threats facing the banana industry worldwide. Unlike other races of Panama disease, *Foc* TR4 has the ability to attack healthy, unstressed plants, making it particularly dangerous to commercial plantations. This race was discovered in the Northern Territory in the late 1990s and has remained under strict quarantine management. All control techniques for this strain have proved unsuccessful, meaning that once a plantation becomes infected with this disease, further spread can only be achieved by the destruction of infected plants. In 20 years time, we estimate the impact of the disease could exceed $45 million per year.

The Queensland State government imposes standards for de-leafing to minimise the risk of *M. musicola* spread and impact, and we have applied our modelling framework to estimate the net benefit that might be gained from lowering these standards. Of the six banana

pest quarantine areas specified, the NBPQA is the most significant in terms of banana production. Previous regulations imposed obligations on owners of banana plants within this area to remove leaves from plants with visible *M. musicola* symptoms on more than 15 per cent of any leaf during the wet season. Recently, this leaf disease threshold has been lowered to 5 per cent. We estimate that over a 30-year period, the average net benefit this reduced threshold will generate for the banana industry in the NBPQA will only be of the order of $1.4 million. Considering this is spread over approximately 10,400 hectares of bananas, the impact of the change in disease thresholds appears to be marginal.

4. Implications for stakeholders

Risk perception is difficult to reconcile, particularly in relation to invasive species incursion events with potentially devastating consequences. The Australian apple and pear industry and banana industry have often been portrayed as having a pessimistic view of risk related to pests and diseases potentially introduced by trade. But, few studies have focused on the pests and diseases of concern to the industry and how the welfare of producers is likely to change over time if and when incursions spread across growing regions.

This project has developed technologies to help both the apple and pear industry and the banana industry to estimate changes in grower welfare induced by pest and disease incursions, and how the impacts on the industry might be best managed.

One of the techniques developed in the project was a maps-based (or spatial) incursion simulation model. This combines computer simulation models of outbreak scenarios that are projected on to interactive maps to produce a 'war games' tool. Industry and government specialists are able to look in detail at incursion scenarios and refine response plans in the virtual world of the animated map where errors are costless. The lessons they learn can then be transferred to real incursion situations in the future. The platform developed is very flexible, and can be modified to depict virtually any EPP incursion affecting any plant industry host(s).

A series of intensive workshops with small groups of industry representatives and invasive species specialists have been conducted to refine a series of incursion scenarios. We have then constructed the computer simulation models around these scenarios to adequately capture the intricacies of pest and disease outbreaks and management considerations. A final workshop was held in Melbourne on April 19-20 in which one of the examples, *E. amylovora*, was showcased to industry and government representatives. This involved an interactive war game component with the simulation model map taking centre stage.

A second tool developed in the project to help the banana industry assess the likely producer losses from plant pathogens threatening the viability of their industry involved non-spatial bioeconomic models. These models can be used to provide highly detailed technical *assessments* of invasive species risks. Separate models were constructed for the following diseases:

1. Banana bunchy top virus – a disease caused by the virus *Pentalonia nigronervosa* which is already established in Australia.
2. Moko disease – an exotic bacterial wilt caused by *Ralstonia solanacearum*.
3. Black Sigatoka – an exotic leaf spot disease caused by the fungus *Mycosphaerella fijiensis*.

4. Panama disease – a serious wilt disease established in some parts of Australia caused by the fungus *Fusarium oxysporum*.
5. Yellow Sigatoka – a leaf spot disease established in Australia caused by the fungus *Mycosphaerelia musicola*.

As their output is based on quantitative measures of economic impact, these models can provide industry with valuable information to use in negotiations regarding future incursion management and imposing phytosanitary restrictions on potential disease entry pathways.

5. Recommendations

1. **Structured decision making encompassing deliberative multi-criteria evaluation should be considered a relevant framework for making incursion response decisions.**

   This project has demonstrated that group-based decision facilitation techniques that provide a structural framework for making invasive species management decisions require on-going research, refinement and communication. Technologies such as keypad devices and interactive decision-making software continue to be developed and, with appropriate training and application, could greatly enhance decision-support processes for biosecurity risk managers.

2. **Maps-based incursion simulation models should be further developed and employed in the refinement of incursion response plans.**

   When a decision-making group is psychologically 'near' to an event, pictorial representations of it are more effective decision aids than words and statistics. Technologies produced by this project can be used in practical biosecurity risk management decisions through the use visual, map based incursion simulation models. These models can be used to simulate incursion events before they actually occur so that risk managers can practice and refine response protocols. Being maps-based, the outputs of these simulation models are relatively easy to interpret and contextualise, particularly when the decision-making group is familiar with the map onto which incursion information is projected.

3. **Research should be conducted to investigate the feasibility of integrating maps-based bio-economic incursion management models with surveillance and field diagnostic technologies to form an incursion response platform.**

   Technologies such as hand-held diagnostic tools and smart traps may one day be integrated with spatial bio-economic models that map incursions and indicate current and possible future economic impacts. This could form a platform around which management decisions could be formulated, tested and implemented by risk managers.

4. **Traditional economic analyses intended for circulation and future use by diverse groups of decision-makers should be designed to be as functional and flexible as possible to cater for this diversity.**

   By building sufficient flexibility in to tools such as the spatial and non-spatial bioeconomic models presented in this report, the uptake of results by industry, government and community stakeholders can be greatly enhanced. This flexibility may be in terms of interactive displays and user-friendliness, a variety of model input and output styles and formats, or the willingness of tool designers to sit down with decision-makers and explain idiosyncrasies of their particular analytical tools. This has the effect of enabling decision-makers to champion information like quantitative model results, and to use them to make more informed decisions and choices about EPP risk management. It also provides researchers and designers of analytical tools valuable feedback to continually improve the interface between models and groups of decision-makers.

6. Abbreviations/glossary

ABBREVIATION	FULL TITLE
ABGC	Australian Banana Grower's Council
ABM	Agent Based Modelling
ABS	Australian Bureau of Statistics
AIC	Akaike Information Criterion
ALOP	Acceptable Level Of Protection
APAL	Apple and Pear Australia Ltd.
ArcGIS	A geographic information system used to create and use maps and to compile geographic data
BBTV	Banana Bunchy Top Virus
B. dorsalis	Oriental fruit fly (*Bactrocera dorsalis* (Hendel))
BIOCLIM	Bioclimatic analysis and prediction system
Biomapper	A maps-based and statistical tool used to construct habitat suitability models and maps for different organisms
BRT	Boosted Regression Tree
CABI	Centre for Agricultural Bioscience Information
CLIMEX	A computer-based system which enables the prediction of an organism's potential relative abundance and distribution around the world using biological data and observations on geographical distribution.
CommunityViz	A software package that facilitates decisions in a workshop environment
CP	Contact Premises
CRCNPB	Cooperative Research Centre for National Plant Biosecurity
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CUBA	Communicating Uncertainties in Biosecurity Adaption
DAFWA	Department of Agriculture and Food, Western Australia
DEM	Digital Elevation Model
DMCE	Deliberative Multi-Criteria Evaluation
DOMAIN	A range-standardized, point-to-point similarity metric that quantifies the similarity between two sites
DPI Vic.	Department of Primary Industries, Victoria
D. plantaginea	Rosy apple aphid (*Dysaphis plantaginea* Pass.)
EHB	European House Borer
EPP	Emergency Plant Pest
EPPO	European and Mediterranean Plant Protection Organization

ERAT	Enhanced Risk Analysis Tools
E. amylovora	Fire blight (*Erwinia amylovora*)
EU	European Union
Foc TR4	Panama disease (*Fusarium oxysporum* f.sp. *cubense*) Tropical Race 4
GARP	Genetic Algorithm for Rule Set Production
GAM	Generalized additive model
GDM	Generalized dissimilarity modelling
GIS	Geographic Information System
GLM	Generalized Linear Model
GPS	Global Positioning System
GRASS	Geographic Resources Analysis Support System
HAL	Horticulture Australia Ltd.
HD	High Density
HDP	High Disease Pressure
IM	Impact Matrix
IP	Infected Property
LD	Low Density
LDP	Low Disease Pressure
LWI	Live With It
MARS	Multivariate Adaptive Regression Splines
MAXENT	Maximum Entropy
MCAS-S	Multi-Criteria Analysis Shell for Spatial Decision Support
MCDA	Multi-Criteria Decision Analysis
MDiG	Modular Dispersal in GIS
M. fijiensis	Black Sigatoka (*Mycosphaerella fijiensis* (Morelet))
M. musicola	Yellow Sigatoka (*Mycosphaerella musicola*)
NBPQA	Northern Banana Pest Quarantine Area
NetLogo	A multi-agent programmable modelling platform designed in the Logo programming language to enable quick and easy authoring of models
N. galligena	European canker (*Nectria galligena*)
OFF	Oriental Fruit Fly
ORC	Owner Reimbursement Costs
PHA	Plant Health Australia
QA	Quarantine Area
RBGM	Royal Botanical Gardens of Melbourne
RAA	Rosy Apple Aphid

ROC	Receiver Operating Characteristic
R. solanacearum	Moko disease (*Ralstonia solanacearum* – Race 2)
SDM	Structured Decision Making
SPS	Sanitary and Phyto-Sanitary
TC^P	Total Cost to Producers
WTO	World Trade Organization
V. destructor	Varroa bee mite (*Varroa destructor*)

7. Plain English website summary

CRC project no:	CRC10162.
Project title:	Communicating Uncertainty in Biosecurity Adaption.
Project leader:	Dr David C. Cook.
Project team:	Dr. Shuang Liu, Dr. Jean-Philippe Aurambout, Dr. Oscar N. Villalta, Dr. Darren J. Kriticos, Mr. Michael Hurley, Asc. Prof. Kim E. Lowell, Dr. Abu-Baker Siddique, Dr. Art Diggle and Dr. Jacqueline Edwards.
Research outcomes:	Our research has demonstrated the usefulness of two broad analytical tools: maps-based incursion response tools, and; statistically-based economic impact models. We have successfully demonstrated the potential for maps-based incursion models to be used to communicate complex suites of information to industry and government stakeholders. These models can be used in conjunction with decision-trees and multi-criteria analysis to form a structured decision making (SDM) approach to refining invasion response plans. This process can be facilitated by using the maps-based models to simulate different invasion scenarios which a decision-making group must then manage in a 'war-gaming' experiment. In this way, response tactics, logistics and flexibility can be tested before an invasion event. When a real event does take place, the lessons learned in the war game experience can be put into practice. We have also demonstrated the explanatory power of more traditional, statistics-based economic impact assessments in communicating the potential significance of EPPs over long periods of time (e.g. 20-30 years). These assessments can be of great strategic significance in setting broad research agendas and funding priorities when site-specific details of possible future incursions are not relevant.
Research implications:	The research carried out by the CUBA project team implies that important steps could be taken to refine incursion response strategies, and that the techniques currently exist to facilitate this. Moreover, with careful planning, a range of complex information about invasive species impacts, uncertain information and expert testimony can be utilised in a structured and sequential way to facilitate management decisions that are mutually acceptable to all stakeholders within a decision-making group. Technologies such as keypad devices and interactive decision-making software continue to be developed that make capturing and processing decision-maker thoughts and preferences quick and easy. With appropriate application and training for prospective users, these technologies could greatly enhance decision-support processes for biosecurity risk managers in the future. In this project, we have demonstrated that group-based decision facilitation techniques that provide a structural framework for making

	invasive species management decisions can be very effective. They can facilitate negotiation and sharing of knowledge between stakeholders, and add an important element of transparency. This is particularly true when decision-support frameworks can be built around maps-based incursion projection technologies. When a decision-making group is placed psychologically 'near' to an event, pictorial representations of it are more effective decision aids than words and statistics. Spatial, maps-based models can be used to simulate incursion events before they actually occur so that risk managers can practice and refine response protocols. Being maps-based, the outputs of these simulation models are relatively easy to interpret and contextualise, particularly when the decision-making group is familiar with the map onto which incursion information is projected. Spatial models may one day form the basis of 'live' control centre operations where a real incursion is tracked as it occurs. Technologies such as hand-held diagnostic tools and smart traps may one day be integrated with these spatial models to update them as information becomes available. This could potentially form a platform around which management decisions could be quickly formulated and tested by projecting EPP abundance and distribution before they are put into practice.
Research publications:	1. Carrasco, L.R., Cook, D., Baker, R., MacLeod, A., Knight, J.D. and Mumford, J.D. (2012) Towards the integration of spread and economic impacts of biological invasions in a landscape of learning and imitating agents. *Ecological Economics* **76**, 95-103. 2. Cook, D.C., Carrasco, L.R., Paini, D.R., Fraser, R.W. (2011) Estimating the social welfare effects of New Zealand apple Imports. *Australian Journal of Agricultural and Resource Economics* **55**, 1-22. 3. Cook, D.C., Liu, S., Edwards, J., Villalta, O.N., Aurambout, J-P., Kriticos, D.J., Drenth, A. and De Barro, P.J. (in press) Predicting the Benefits of Banana Bunchy Top Virus Exclusion from Commercial Plantations in Australia. *PLoS ONE*. 4. Cook, D.C., Fraser, R.W., Waage, J.K. and Thomas, M.B. (2011) Prioritising biosecurity investment between agricultural and environmental systems. *Journal of Consumer Protection and Food Safety* **6**, 3-13. 5. Liu, S., Hurley, M., Lowell, K.E., Siddique, A-B., Diggle, A. and Cook, D.C. (2011) An integrated decision-support approach in prioritizing risks of non-indigenous species in the face of high uncertainty. *Ecological Economics* **70**, 1924-1930. 6. Liu, S., Sheppard, A., Kriticos, D. and Cook, D.C. (2011) Incorporating uncertainty and social values in managing invasive alien species: a deliberative multi-criteria evaluation approach. *Biological Invasions* **13**, 2323-2337.

Acknowledgements:	The Communicating Uncertainty in Biosecurity Adaption project team would like to acknowledge the fantastic support we have received from Apple and Pear Australia Ltd. and the Australian Banana Grower's Council. We are very grateful for the valuable role participants in our Expert Reference Group workshops played in shaping the project and the spatial incursion management model. Thank you also to all who participated in the final project workshop held in Melbourne on the 19-20 April 2012, including Annie Farrow, Jesse Reader and Sarah Kulman (Apple and Pear Australia Ltd.), Michael Crisera (Fruit Growers Victoria), Lucy Gregg (Fruit Growers Tasmania), Susan Finger (Victorian Farmer's Federation), Simon Mills (SPC Ardmona Ltd.), Steven Field, Olivia Montecillo, Brendan Rodoni, Bill Washington, Martin Mebalds, Chin Gouk and David Williams (DPI Victoria), Anne Mooney (DPI New South Wales), Kim James, Brad Wells and Brad Mills (Horticulture Australia Ltd.), Timothy Coutts and Rose Hockham (Department of Agriculture, Fisheries and Forestry), Stephen Dibley (Plant Health Australia), Jim Cullen (CRCNPB), Andre Drenth (University of Queensland), Peter Merriman (Peter Merriman Consulting), Fiona Macbeth (Blackwood and Kemp Pty. Ltd.). We would also like to thank Paul De Barro, Andy Sheppard, Robyn Noel, Dean Paini, David Adams, Stephanie von Gavel and Rieks van Klinken (CSIRO Ecosystem Sciences), Simon McKirdy, David Eagling, Scott Baxter, John Austen, Max Knobel and Andrew Crichton (CRCNPB), Richard Maxwell (DPI Victoria), David Peasley (David Peasley Horticultural Services), John Thomas (University of Queensland), Jim Pekin (Australian Banana Grower's Council), Rob Fraser (University of Kent), Roman Carrasco (National University of Singapore), Jeff Waage (London International Development Centre), Matt Thomas (Penn. State University), Richard Baker and Alan MacLeod (Food and Environment Research Agency), John Mumford and John Knight (Imperial College London), Rachel Meldrum (CRCNPB and Northern Territory Department of Resources), Andrew Tomkins (Northern Territory Department of Resources) and Victoria El Zein (Park Royal Hotel) for their valuable help at different times during the project and in preparing publications.

8. References

ABS (2011) Fruit and nuts - year ended 30 June. In 'Agricultural Commodities, Australia'. (Australian Bureau of Statistics: Canberra)

Ajzen I, Brown TC, Rosenthal LH (1996) Information bias in contingent valuation: Effects of personal relevance, quality of information, and motivational orientation. *Journal of Environmental Economics and Management* 30, 43-57.

Allen R, Akehurst A, Ireland G (1992) Surveys of spraying practices for banana leaf disease control in New South Wales, and an assessment of disease control based on leaf retention at harvest in July. *Australian Journal of Experimental Agriculture* 32, 211-216.

Andersen MC, Adams H, Hope B, Powell M (2004) Risk assessment for invasive species. *Risk Analysis* 24, 787-793.

Andow DA, Kareiva P, Levin SA, Okubo A (1990) Spread of invading organisms. *Landscape Ecology* 4, 177-188.

Anonymous 2000. Government response to the incursion of Varroa mite (*Varroa jacobsoni*). Office of the New Zealand Minister for Biosecurity, Wellington, 19 June.

Anonymous 2010. Industry Biosecurity Plan for the Apple and Pear Industry. Plant Health Australia.

Arampatzis G, Kiranoudis CT, Scaloubacas P, Assimacopoulos D (2004) A GISbased decision support system for planning urban transportation policies. *European Journal of Operational Research* 15, 465-475.

Araújo MB, New M (2007) Ensemble forecasting of species distributions. *Trends in Ecology and Evolution* 22, 42-47.

Aspinall R (1992) An inductive modelling procedure based on Bayes' theorem for analysis of pattern in spatial data. *International Journal of Geographical Information Science* 6, 105-121.

Aurambout JP, finlay KJ, Luck J, Beattie A (2009) A concept model to estimate the potential distribution of the Asiatic citrus psyllid (Diaphorina citri Kuwayama) in Australia under climate change—A means for assessing biosecurity risk. Ecological Modeling 220, 2512-2524.

Australian Department of Agriculture Fisheries and Forestry (2005) Field guide to exotic pests and diseases: European House Borer. In.

Barclay E (2005) 'Local community preparedness for an Emergency Animal Disease outbreak.' Australian Government Rural Industries Research and Development Corporation, Canberra.

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

Beer SV (1990). Fire Blight. P61-63. In: A.L. Jones, H.S. Aldwinckle (ed.), Compendium of Apple and Pear Diseases. APS Press.

Behalova M (2004). Experience with fireblight in the Czech Republic. OEPP/EPPO Bulletin 34, 365-367.

Beierle TC (2002) The quality of stakeholder-based decisions. *Risk Analysis* 22, 739-749.

BenDor TK, Metcalf SS, Fontenot LE, Sangunett B, Hannon B (2006) Modeling te spread of teh Emerald Ash Borer. *Ecological Modelling* 197, 221-236.

Benke KK, Steel JL, Weiss JE (2011) Risk assessment models for invasive species: uncertainty in rankings from multi-criteria analysis. *Biological Invasions* 13, 239-253.

Berryman M (2008) Review of Software Platforms for Agent Based Models. In. (Ed. ADo Defence) pp. 19. (Land Operation Division DSTO Defence Science and Technology Organisation)

Billing, E (1996) BIS95, an improved approach to fire blight risk assessment. *Acta Horticulturae* 411, 121-126.

Biosecurity Australia (2001) 'Guidelines for Import Risk Analysis.' Agriculture, Fisheries and Forestry Australia/Biosecurity Australia, Canberra.

Biosecurity Australia (2006) 'Final import risk analysis report for apples from New Zealand, Part A.' Biosecurity Australia, Canberra.

Biosecurity Australia (2008) 'Final Import Risk Analysis Report for the Importation of Cavendish Bananas from the Philippines, Part A.' (Biosecurity Australia: Canberra)

Blommers LHM, Helsen HHM, Vaal FWNM (2004) Life history data of the rosy apple aphid *Dysaphis plantaginea* (pass.) (Homopt., Aphididae) on plantain and as migrant to apple. *Journal Pest Science* 77, 155-163.

Bojorquez-Tapia LA, Sanchez-Colon S, Martinez AF (2005) Building consensus in environmental impact assessment through multicriteria modeling and sensitivity analysis. *Environmental Management* 36, 469-481.

Born W, Rauschmayer F, Brauer I (2005) Economic evaluation of biological invasions - a survey. *Ecological Economics* 55, 321-336.

Bossenbroek JM, McNulty J, Keller RP (2005) Can ecologists heat up the discussion on invasive species risk? *Risk Analysis* 25, 1595-1597.

Brouwer R, De Blois C (2008) Integrated modelling of risk and uncertainty underlying the cost and effectiveness of water quality measures. *Environmental Modelling and Software* 23, 922-937.

Brown AGP (1998) 'Pest Risk Analysis of Musa Species for the Northern Australia Quarantine Stratergy - A Report Prepared for the Australian Quarantine and Inspection Service.' Australian Quarantine and Inspection Service, Canberra.

Brown D (2000) A review of bio-economic models. CAFSNRM Program. (www.citeseerx.ist.psu.edu)

Brown MW, Mathews CR (2007) Conservation biological control of rosy apple aphid, *Dysaphis plantaginea* (Passerini), in Easyern North America. *Environmental Entomology* 36, 1131-1139.

Buckley YM, Brockerhoff E, Langer L, Ledgard N, North H, Rees M (2005) Slowing down a pine invasion despite uncertainty in demography and dispersal. *Journal of Applied Ecology* 42, 1020-1030.

Bui E (Ed.) (1999) 'A soil information strategy for the Murray-Darling Basin (MDBSIS). Report to Murray Darling Basin Commission, Project D5038.' (CSIRO: Canberra)

Burgman MA, Keith DA, Walshe TV (1999) Uncertainty in comparative risk analysis for threatened Australian plant species. *Risk Analysis* 19, 585-598.

CABI, EPPO (1997) Data sheets on Quarantine Pests: *Erwinia amylovora*. In 'http://www.eppo.int/QUARANTINE/bacteria/Erwinia_amylovora/ERWIAM_ds.pdf'. (Ed. EaMPP Organization).

CABI. http://www.cabicompendium.org/cpc/datasheet.asp

CABI/EPPO (2003) Crop Protection Compendium - Global Module. In. (CAB International: Cayman Islands)

Calzolari A, Finelli F, Mazzoli GL (1999) A severe unforseen outbreak of fire blight in the Emilia-Romagna region. *Acta Horticulturae* 489, 171-489.

Calzolari A, Finlay KJ, Mazzoli GL (1999) A sever unforeseen outbreak of fire blight in the Emilia-Romagna region. *Acta Horticulrae* 489, 171-176.

Carey JM, Burgman MA (2008) Linguistic uncertainty in qualitative risk analysis and how to minimize it. *annals of the New York Academy of Sciences* 1128, 13-17.

Carlsson F (2010) Design of Stated Preference Surveys: Is There More to Learn from Behavioral Economics? *Environmental and Resource Economics* 46, 167-177.

Carpenter G, Gillison AN, Winter J (1993) DOMAIN: a flexible modelling procedure for mapping potential distributions of plants and animals. *Biodiversity and Conservation* 2, 667-680.

Cass Smith WP, Harvey HL, Goss O (1948) Apple scab outbreaks season 1947-48, with special reference to the introduction of the disease by infected bud on imported nursery stock. *Western Australian Journal of Agriculture* 25, 129-135.

Castle C (2006) Principles and concepts of agent-based modelling for developing geospatial simulations. *UCL Working Papers Series* 110, 1-60.

Ceballos-Silva A, Lopez-Blanco J (2003) Evaluating biophysical variables to identify suitable areas for oat in Central Mexico: a multi-criteria and GIS approach. *Ecosystems and Environment* 95, 371-377.

Chichakly K (2009) Spatial Modeling with isee Spatial Map. In 'Making connections isee systems blog'. (Ed. I Systems). (isee systems)

Chiou CS, Jones AL (1993) Nucleotide sequence analysis of a transposon (TN5393) carrying streptomycin resistance genes in *Erwinia amylovora* and other gram-negative bacteria. *Journal of Bacteriology* 175:732-740.

Cockayne AH (1919) Fire blight, a serious disease of fruit trees. *New Zealand Journal of Agriculture* 20, 156-157.

Cook D, Carrasco L, Paini D, Fraser R (2011a) Estimating the social welfare effects of New Zealand apple imports. *The Australian Journal of Agricultural and Resource Economics*, 1-22.

Cook D, Long G, Possingham H, Failing L, Burgman M, Gregory R, Estevez R, Walshe T (2010a) 'Potential methods and tools for estimating biosecurity consequences for primary production, amenity, and the environment.' Australian Centre of Excellence for Risk Analysis, Melbourne.

Cook DC (2003) 'Prioritising Exotic Pest Threats to Western Australian Plant Industries.' Government of Western Australia - Department of Agriculture, Bunbury.

Cook DC (2008) Benefit cost analysis of an import access request. *Food Policy* 33, 277–285.

Cook DC, Carrasco LR, Paini DR, Fraser RW (2011b) Estimating the social welfare effects of New Zealand apple Imports. *Australian Journal of Agricultural and Resource Economics* 55, 1-22.

Cook DC, Fraser RW (2008) Trade and Invasive Species Risk Mitigation: Reconciling WTO Compliance with Maximising the Gains from Trade. *Food Policy* 33, 176–184.

Cook DC, Fraser RW, Paini DR, Warden AC, Lonsdale WM, Barro PJD (2011c) Biosecurity and yield improvement technologies are strategic complements in the fight against food insecurity. *PLoS ONE* 6, e26084.

Cook DC, Hurley M, Liu S, Siddique A-BM, Lowell KE, Diggle A (2010b) 'Final Report CRC10010 Enhanced Risk Analysis Tools.' Cooperative Research Centre for National Plant Biosecurity, Canberra.

Cook DC, Liu S, Hurley M, Siddique A-BM, Diggle A, Lowell KE (2009a) 'Enhanced Biosecurity Planning Tools: Combining Incursion Simulation Tools with Deliberative Decision Facilitation Methods to Prioritise Invasive Species.' Rural Industries Research and Development Corporation, Canberra.

Cook DC, Liu S, Murphy B, Lonsdale WM (2010c) Adaptive Approaches to Biosecurity Governance. *Risk Analysis* 30, 1303-1314.

Cook DC, Liu S, Proctor WL (2009b) '0803 *Deliberative Methods for Assessing Utilities* Final Project Report.' Australian Centre of Excelence for Risk Analysis, Melbourne.

Cook DC, Proctor WL (2007) Assessing the threat of exotic plant pests. *Ecological Economics* 63, 594-604.

Cook DC, Thomas MB, Cunningham SA, Anderson DL, De Barro PJ (2007b) Predicting the economic impact of an invasive species on an ecosystem service. *Ecological Applications* 17, 1832–1840.

Cook DC, Waage JK, Mumford JD, Fraser RW, Wilby A (2006) 'The benefits of potato ring rot exclusion from the United Kingdom.' Department of Trade and Industry, Office of Science and Innovation, Technical Report T8.11, London.

Cooke LR, Watters BS, Brown AE (1993) The effect of fungicide sprays on the incidence of apple canker (*Nectria galligena*) in Bramley's seedling. *Plant Pathology* 42, 432-442.

Crowl TA, Crist TO, Parmenter RR, Belovsky G, Lugo AE (2008) The spread of invasive species and infectious disease as drivers of ecosystem change. *Frontiers in Ecology and the Environment* 6, 238-246.

Cuddy SM, Laut P, Davis JR, Whigham PA, Goodspeed J, Duell T (1990) Modelling the environmental effects of training on a major Australian army base. *Mathematics and Computers in Simulation* 32, 83-88.

Cvjetkovic B, Halupecki E, Spoljaric J (1999) The occurrence and control of fire blight in Croatia. *Acta Horticulturea* 489,71-73.

Dai FC, Lee CF, Zhang XH (2001) GIS-based geo-environmental evaluation for urban land-use planning: a case study. *Engineering Geology* 61, 257-271.

Dale JL (1987) Banana bunchy top: an economically important tropical plant virus disease. *Advances in Virus Research* 33, 301-325.

Daly A, Walduck G (2006) '*Fusarium* Wilt of Bananas (Panama Disease) (*Fusarium oxysporum* f. sp. *cubense*), Agnote No. I51.' (Department of Primary Industries, Fisheries and Mines: Darwin)

Davis AS, Cousens RD, Hill J, Mack RN, Simberloff D, Raghu S (2010) Screening bioenergy feedstock crops to mitigate invasion risk. *Frontiers In Ecology And The Environment* 8, 533-539.

De Berardinis E, Baronio P, Baumgartner J (1994) The effect of aphid (*Dysaphis plantaginea* Pass., Hom., Aphididae) feeding apple fruit growth. *Ecological Modelling* 72, 115-127.

De Wael L, de Greef M, van Laere O (1990) The honeybee fireblight relation. *Apiacta* 25, 111-117.

Department of Primary Industries Victoria (2009) Queensland fruit fly, Melbourne.

Department of Primary Industries Victoria (2010) Benefit-cost analysis of options for managing Queensland fruit fly in Victoria. Evalation series No. 11.

Dimitrova E, Andreev L (2004) Fireblight situation in Bulgaria and measures undertaken by the NPPO. OEPP/EPPO Bulletin 34, 343-345.

Doak DF, Estes JA, Halpern BS, Jacob U, Lindberg DR, Lovvorn J, Monson DH, Tinker MT, Williams TM, Wootton JT, Carroll I, Emmerson M, Micheli F, Novak M (2008) Understanding and predicting ecological dynamics: Are major surprises inevitable? *Ecology* 89, 952-961.

Dragan M, Feoli E, Fernetti M, Zerihun W (2003) Application of a spatial decision support system (SDSS) to reduce soil erosion in northern Ethiopia. *Environmental Modelling and Software* 18, 861-868.

Dryzek J (2000) 'Deliberative democracy and beyond: liberals, critics and contestations.' (Oxford University Press)

Dubin HJ, English H (1975) Effects of temperature, relative humidity, and desiccation on germination of *Nectria galligena* conidia. *Mycologia* 67, 83-88.

Dumont B, Boissy A (1999) Relations sociales et comportement alimentaire au pÃ¢turage. *Productions Animales* 12, 3-10.

Dwyer G (1992) On the spatial spread of insect pathogens - theory and experiment. *Ecology* 73, 479-494.

Edwards J, Villata O, Powney R (2006) Investigate the current state of knowledge worldwide regarding *Neonectria galligena*. Horticulture Australia report AP05029.

Ehrenfeld JG (2010) Ecosystem Consequences of Biological Invasions. *Annual Review Of Ecology, Evolution, And Systematics* 41, 59-80.

Elith J, Burgman MA, Regan HM (2002) Mapping epistemic uncertainties and vague concepts in predictions of species distribution. *Ecological Modelling* 157, 313-329.

Elith J, Graham CH*,* Anderson RP, Dudík M, Ferrier S, Guisan A, Hijmans RJ, Huettmann F, Leathwick JR, Lehmann A, Li J, Lohmann LG, Loiselle BA, Manion G, Moritz C, Nakamura M, Nakazawa Y, Overton JMcCM, Townsend Peterson A, Phillips SJ, Richardson K, Scachetti-Pereira R, Schapire RE, Soberón J, Williams S, Wisz MS, Zimmermann NE (2006) Novel methods improve prediction of species' distributions from occurrence data. *Ecography* 29, 129-151.

Elliston L, Hinde R, Yainshet A (2005) Plant disease incursion management. P. Davidson *et al.* (Eds): MABS 2005, LNAI 3415, pp. 225-235.

Fegan M (2005) Bacterial wilt diseases of banana: evolution and ecology. In 'Bacterial wilt disease and the *Ralstonia solanacearum* species complex'. (Eds C Allen, P Prior and AC Hayward) pp. 379-386. (APS Press: St. Paul)

Finelli F, Contessi A, Calzolari A, Saccardi A, Zecchin G, Vettorazzo M (2004) Fireblight in the Po valley (Italy): the monitoring programmes of Emilia-Romagna and Veneto regions. OEPP/EPPO Bulletin 34, 331-334.

Fischhoff B (1995) Risk Perception and Communication Unplugged - 20 Years of Process. *Risk Analysis* 15, 137-145.

Fischhoff B, Litchtenstein S, Slovic P, Derby SL, Keeney RL (1981) 'Acceptable risk.' (Cambridge University Press: Cambridge)

Fisher RA (1937) The wave of advance of advantageous genes. *Annual Eugenics* 7, 353-369.

Food and Agriculture Organization (2010). In 'FAOSTAT Database'. (Food and Agriculture Organization of the United Nations: Rome)

Förster J, Friedman RS, Liberman N (2004) Temporal construal effects on abstract and concrete thinking: Consequences for insight and creative cognition. *Journal of Personality and Social Psychology* 87, 177–189.

Fox JC, Buckley YM, Panetta FD, Bourgoin J, Pullar D (2009) Surveillance protocols for management of invasive plants: modelling Chilean needle grass (Nassella neesiana) in Australia. *Diversity and Distributions* 15, 577-589.

Franklin J, Sisson SA, Burgman MA, Martin JK (2008) Evaluating extreme risks in invasion ecology: learning from banking compliance. *Diversity and Distributions* 14, 581-591.

Funtowicz SO, Ravetz JR (1993) Science for the post-normal age. *Futures* 25, 739-755.

Geneletti D (2007) An approach based on spatial multi-criteria analysis to map the nature conservation value of agricultural land. *Journal of Environmental Management* 83, 228-235.

Gevrey M, Worner SP (2006) Prediction of Global Distribution of Insect Pest Species in Relation to Climate by Using an Ecological Informatics Method. *Journal of Economic Entomology* 99, 979-986.

Gibson GJ, Austin EJ (1996) Fitting and testing spatio-temporal stochastic models with application in plant epidemiology. *Plant Pathology* 45, 172-184.

Giupponi C, Mysiak J, Fassio A, Cogan V (2004) MULINO-DSS: a computer tool for sustainable use of water resources at the catchment scale. *Mathematics and Computers in Simulation* 64, 13-24.

Goldston D (2008) Hazy reasoning behind clean air. *Nature* 452, 519-519.

Gomez-Delgado M, Tarantola S (2006) GLOBAL sensitivity analysis, GIS and multi-criteria evaluation for a sustainable planning of hazardous waste disposal site in Spain. *International Journal of Geographical Information Science* 20, 449-466.

Gordon DR, Onderdonk DA, Fox AM, Stocker RK (2008) Consistent accuracy of the Australian weed risk assessment system across varied geographies. *Diversity and Distributions* 14, 234-242.

Gregory R, Failing L, Ohlson D, McDaniels TL (2006) Some pitfalls of an overemphasis on science in environmental risk management decisions. *Journal of Risk Research* 9, 717-735.

Gren IM (2008) Economics of alien invasive species management - choices of targets and policies. *Boreal Environment Research* 13, 17-32.

Grove, G.G. (1990) Nectria canker. Compendium of Apple Diseases. 35-36 pp.

Groves RH (2006) Are some weeds sleeping? Some concepts and reasons. *Euphytica* 148, 111-120.

Guisan A, Harrell FE (2000) Ordinal Response Regression Models in Ecology. *Journal of Vegetation Science* 11, 617-626.

Guisan A, Theurillat JP, Kienast F (1998) Predicting the Potential Distribution of Plant Species in an Alpine Environment. *Journal of Vegetation Science* 9, 65-74.

Guisan A, Zimmermann NE (2000) Predictive habitat distribution models in ecology Ecological Modelling. *Ecological Modelling* 135, 147-186.

Haimes YY (2009) On the Complex Definition of Risk: A Systems-Based Approach. *Risk Analysis* 29, 1647-1654.

Hajkowicz S, Collins K (2007) A review of multiple criteria analysis for water resource planning and management. *Water Resource Management* 21, 1553-1566.

Hanski I, Alho J, Moilanen A (2000) Estimating the parameters of survival and migration of individuals in metapopulations. *Ecology* 81, 239-251.

Hartley S, Harris R, Lester PJ (2006) Quantifying uncertainty in the potential distribution of an invasive species: climate and the Argentine ant. Ecology Letters. *Ecology Letters* 9, 1068-1079.

Hastings A, Cuddington K, Davies KF, Dugaw CJ, Elmendorf S, Freestone A, Harrison S, Holland M, Lambrinos J, Malvadkar U, Melbourne BA, Moore K, Taylor C, Thomson D (2005) The spatial spread of invasions: new developments in theory and evidence. *Ecology Letters* 8, 91-101.

Henderson J, Pattemore JA, Porchun SC, Hayden HL, Brunschot SV, Grice KRE, Peterson RA, Thomas-Hall SR, Aitken EAB (2006) Black Sigatoka disease: new technologies to strengthen eradication strategies in Australia. *Australasian Plant Pathology* 35, 181-193.

Hengeveld B (1989) 'Dynamics of Biological Invasions.' (Chapman and Hall: London)

Hengeweld R (1989) 'The Dynamics of Biological Invasions.' (Chapman and Hall: London)

Hennessy C, Walduck G, Daly A, Padovan A (2005) Weed hosts of Fusarium oxysporum f. sp. cubense tropical race 4 in northern Australia. *Australasian Plant Pathology* 34, 115-117.

Heslop-Harrison JS, Schwarzacher T (2007) Domestication, genomics and the future for banana. *Annals of Botany* 100, 1073–1084.

Higgins SI, Richardson DM (1996) A review of models of alien plant spread. *Ecological Modelling* 87, 249-265.

Higgins SI, Richardson DM, Cowling RM (2000) Using a dynamic landscape model for planning the management of alien plant invasions. *Ecological Applications* 10, 1833-1848.

Hinchy MD, Fisher BS (1991) 'A Cost-Benefit Analysis of Quarantine ' (Australian Bureau of Agricultural and Resource Economics: Canberra)

Hirzel AH, Hausser J, Chessel D, Perrin N (2002) Ecological-niche factor analysis: how to compute habitat-suitability maps without absence data? *Ecology.* 83, 2027-2036.

Hirzel AH, LeLay G (2008) Habitat suitability modelling and niche theory. *Journal of Applied Ecology* 45, 1372-1381.

Holmes EE (1993) Are diffusion-models too simple - a comparison with telegraph models of invasion. *American Naturalist* 142, 779-795.

Hooks CRR, Wright MG, Kabasawa DS, Manandhar R, Almeida RPP (2008) Effect of banana bunchy top virus infection on morphology and growth characteristics of banana. *Annals of Applied Biology* 153, 1-9.

Horan RD, Perrings C, Lupi F, Bulte EH (2002) Biological pollution prevention strategies under ignorance: The case of invasive species. *American Journal of Agricultural Economics* 84, 1303-1310.

Hummel S, Donovan GH, Spies TA, Hemstrom MA (2009) Conserving biodiversity using risk management: hoax or hope. *Frontiers In Ecology And The Environment* 7, 103-109.

Hurley MV, Lowell KE, Cook DC, Liu SA, Siddique AB, Diggle A (2010) Prioritizing Biosecurity Risks Using a Participatory Decision-Making Tool. *Human and Ecological Risk Assessment* 16, 1379-1394.

James S, Anderson K (1998) On the Need for More Economic Assessment of Quarantine Policies. *The Australian Journal of Agricultural and Resource Economics,* 42, 425-444.

Janis I (1982) 'Groupthink.' (Houghton Mifflin: Boston)

Jarvis CH, Baker RHA (2001) Risk Assessment for Nonindigenous Pests: 1. Mapping the Outputs of Phenology Models to Assess the Likelihood of Establishment. *Diversity and Distributions* 7, 223-235.

Javelosa J, Schmitz A (2006) Costs and Benefits of a WTO Dispute: Philippine Bananas and the Australian Market. *The Estey Centre Journal for International Law and Trade Policy* 7, 58-83.

Jayanthi PDK, Verghese A (2011) Host-plant phenology and weather based forecasting models for population prediction of the oriental fruit fly, *Bactrocera dorsalis* Hendel. *Crop Protection* 30, 1557-1562.

Jock S, Rodoni B, Gillings M, Kim WS, Copes C, Merriman P, Geider K (2000) Screening of ornamental plants from the botanic gardens of Melbourne and Adelaide for the occurrence of *Erwinia amylovora*. *Australasian Plant Pathology* 29, 120-128.

Kahneman D, Knetsch J (1992) Valuing public goods: the purchase of moral satisfaction. *Journal of Environmental Economics and Management* 22, 57-70.

Kangas J, R RS, Leskinen P, Mehtätalo L (2000) Improving the quality of landscape ecological forest planning by utilising advanced decision-support tools. *Forest Ecology and Management* 132, 157-171.

Keck M (2004) Ten years of experience with fireblight in Austria. *OEPP/EPPO Bulletin* 34, 347-349.

Keeney RL, H. R, Meyer RF (1993) 'Decisions with multiple objectives: preferences and value tradeoffs.' (Cambridge University Press: Cambridge, UK.)

Keeney RL, Raiffa H (1993) 'Decisions with multiple objectives-preferences and value tradeoffs.' (Cambridge University Press: Cambridge and New York)

Kehrli P, Wyss E (2001) Effect of augmentative releases of the coccinellid, *Adalia bipunctata*, and of insecticide treatments in autumn on the spring population of aphids of the genus *Dysaphis* in apple orchards. *Entomologia Experimentalis et Applicata* 99, 245-252.

Kerr NL, Tindale RS (2004) Group performance and decision making. *Annual Review of Psychology* 55, 623-655.

Knapic V, Potocnik A, Skerlavaj V, Brecl A (2004) First outbreak of fireblight in Slovenia. *OEPP/EPPO Bulletin* 34, 351-356.

Kolar CS, Lodge DM (2001) Progress in invasion biology: predicting invaders. *Trends in Ecology and Evolution* 16, 199-204.

Kolar CS, Lodge DM (2002) Ecological predictions and risk assessment for alien fishes in North America. *Science* 298, 1233-1236.

Kriticos DJ, Leriche A (2010) The effects of climate data precision on fitting and projecting species niche models. *Ecography* 33, 115-127.

Kriticos DJ, Potter KJ, Alexander N, Gibb AR, Suckling DM (2007) Using a pheromone lure survey to establish the native and potential distribution of an invasive Lepidopteran, *Uraba lugens*. *Journal of Applied Ecology* 44, 853-863.

Larson BMH (2007) An alien approach to invasive species: objectivity and society in invasion biology. *Biological Invasions* 9, 947-956.

Larson DL, Phillips-Mao L, Quiram G, Sharpe L, Stark R, Sugita S, Weiler A (2011) A framework for sustainable invasive species management Environmental, social, and economic objectives. *Journal of Environmental Management* 92, 14-22.

Latorre BA, Rioja ME, Lillo C, 1999. El pronostico de enfermedades: II Cancro Europeo del manzano. ACONEX (Chile) 65, 18-22.

Latorre BA, Rioja ME, Lillo C, Munoz M (2002) The effect of temperature and wetness duration on infection and a warning system for European canker (*Nectria galligena*) of apple in Chile. *Crop Protection* 21, 285-291.

Leathwick JR, Rowe D, Richardson J, Elith J, Hastie T (2005) Using multivariate adaptive regression splines to predict the distributions of New Zealand's freshwater diadromous fish. *Freshwater Biology* 50, 2034-2052.

Leroux A, Maclaren D (2011) The optimal time to remove quarantine bans under uncertainty: the case of Australian bananas. *The Economic Record* 87, 140-152.

Lesslie RG, Hill MJ, Hill P, Cresswell HP, Dawson S (2008) The Application of a Simple Spatial Multi-Criteria Analysis Shell to Natural Resource Management Decision Making. In 'Landscape Analysis and Visualisation:Spatial Models for Natural Resource Management and Planning'. (Eds C Pettit, W Cartwright, I Bishop, K Lowell, D Pullar and D Duncan) pp. 73-96. (Springer: Berlin)

Lewis MA (1997) Variability, patchiness, and jump dispersal in the spread of an invading population. In 'Spatial Ecology: The Role of Space in Population Dynamics and Interspecific Interactions'. (Eds D Tilman and P Kareiva) pp. 46-74. (Princeton University Press: New Jersey)

Liberman N, Sagristano M, Trope Y (2002) The effect of temporal distance on level of construal. *Journal of Experimental Social Psychology* 38, 523–535.

Liberman N, Trope Y (1998) The role of feasibility and desirability considerations in near and distant future decisions: A test of temporal construal theory. *Journal of Personality and Social Psychology* 75, 5-18.

Liu S, Cook D, Diggle A, Siddique A-B, Hurley M, Lowell K (2009) Using Dynamic Ecological-Economic Modeling to Facilitate Deliberative Multicriteria Evaluation (DMCE) in Quantifying and Communicating Bio-invasion Uncertainty In '18th World IMACS Congress and MODSIM09 International Congress on Modelling and Simulation. '. Cairns, Australia. (Eds RS Anderssen, RD Braddock and LTH Newham) pp. 4333-4338. (Modelling and Simulation Society of Australia and New Zealand and International Association for Mathematics and Computers in Simulation.)

Liu S, Hurley M, Lowell KE, Siddique A-BM, Diggle A, Cook DC (2011) An integrated decision-support approach in prioritizing risks of non-indigenous species in the face of high uncertainty. *Ecological Economics*, 1924-1930.

Liu S, Proctor W, Cook D (2010) Using an integrated fuzzy set and deliberative multi-criteria evaluation approach to facilitate decision-making in invasive species management. *Ecological Economics* 69, 2374-2382.

Lobo JM, Jiménez-Valverde A, Real R (2008) AUC: a misleading measure of the performance of predictive distribution models. *Global Ecology and Biogeography* 17, 145-151.

Lodge DM, Williams S, MacIsaac HJ, Hayes KR, Leung B, Reichard S, Mack RN, Moyle PB, Smith M, Andow DA, Carlton JT, McMichael A (2006) Biological invasions: Recommendations for US policy and management. *Ecological Applications* 16, 2035-2054.

Lorti M (1964) Pathogenesis in cankers caused by *Nectria galligena*. *Phytopathology* 54, 261-263.

Mack RN, Simberloff D, Lonsdale WM, Evans H, Clout M, Bazzaz FA (2000) Biotic invasions: Causes, epidemiology, global consequences, and control. *Ecological Applications* 10, 689-710.

Mackenzie BF, Larson BMH (2010) Participation Under Time Constraints: Landowner Perceptions of Rapid Response to the Emerald Ash Borer. *Society and Natural Resources* 23, 1013-1022.

Magee CJP (1927) Investigation on the bunchy top disease of the banana. *Council for Scientific and Industrial Research Bulletin* 30, 1-64.

Maguire LA (2004) What can decision analysis do for invasive species management? *Risk Analysis* 24, 859-868.

Maniezzo V, Mendes I, Paruccini M (1998) Decision support for siting problems. *Decision Support Systems* 23, 273-284.

Marín DH, Romero RA, Guzmán M, Sutton TB (2003) Black Sigatoka: an increasing threat to banana cultivation. *Plant Disease* 87, 208-222

Marinoni O, Higgins A, Hajkowicz S, Collins K (2009) The multiple criteria analysis tool (MCAT): A new software tool to support environmental investment decision making. *Environmental Modelling and Software* 24, 153-164.

Maxwell T, Costanza R (1997) A language for modular spatio-temporal simulation. *Ecological Modelling* 103, 105-113.

Mayer DG, Atzenih MG, Butler DG (1993) Spatial Dispersal of Exotic Pests the Importance of Extreme Values. *Agricultural Systems* 43, 133-144.

McCann K, Hastings A, Harrison S, Wilson W (2000) Population outbreaks in a discrete world. *Theoretical Population Biology* 57, 97-108.

McElwee H (2000) 'Potential Economic Impact of Panama Disease (*Fusarium oxysporum* f.sp. *cubense*) Tropical Race 4 in Western Australia.' Government of Western Australia - Department of Agriculture, Bunbury.

McKirdy S, Brown A (1999) 'Fusarium wilt of bananas - The threat posed to the WA industry by the arrival of Tropical race 4 in Darwin.' Agriculture Western Australia, South Perth.

McManus P, Stockwell V (2000) Antibiotics for plant disease control: silver bullets or rusty sabers?. APSnet Features (www.apsnet.org/publications).

McManus PS, Jones AL (1994) Epidemiology and genetic analysis of streptomycin-resistant *Erwinia amylovora* from Michigan and evaluation of oxytetracycline for control. *Phytopathology* 84, 627-633.

Melbourne BA, Hastings A (2009) Highly Variable Spread Rates in Replicated Biological Invasions: Fundamental Limits to Predictability. *Science* 325, 1536-1539.

Merriman P (2002) 'Revised contingency plan for fire blight / compiled and edited by Peter Merriman.' (Horticulture Australia: Sydney)

Merriman P (2002) Revised contingency plan for fire blight. Horticulture Australia Project AP98070.

Meyerson LA (2008) Biosecurity, biofuels, and biodiversity. *Frontiers in Ecology and the Environment* 6, 291-291.

Millennium Ecosystem Assessment (2005) 'Ecosystems and human well being: biodiversity synthesis.' World Resources Institute, Washington, D.C.

Millner A, Dietz S, Heal G (2010) Ambiguity and climate policy. In 'NBER Working Paper No. w16050 '.

Mobambo KN, Gauhl F, Vuylsteke D, Ortiz R, Pasberg-Gauhl C, Swennen R (1993) Yield loss in plantain from black sigatoka leaf spot and field performance of resistant hybrids. *Field Crops Research* 35, 35-42.

Moilanen A (2004) SPOMSIM: software for stochastic patch occupancy models of metapopulation dynamics. *Ecological Modelling* 179, 533-550.

Moilanen A, Wintle BA, Elith J, Burgman M (2006) Uncertainty analysis for regional-scale reserve selection. *Conservation Biology*, 1688-1697.

Mollison D (1977) Spatial Contact Models for Ecological and Epidemic Spread. *Journal of the Royal Statistical Society. Series B (Methodological)* 39, 283-326.

Moody ME, Mack RN (1988) Controlling the spread of plant invasions: the importance of nascent foci. *Journal of Applied Ecology* 25, 1009-1021.

Moran P (1984) 'An Introduction to Probability Theory.' (Clarendon Press: Oxford)

Morgan MG, Fischhoff B, Bostrom A, Lave L, Atman CJ (1992) Communicating Risk to the Public. *Environmental Science and Technology* 26, 2048- 2056.

Morse-McNabb E (2011) The Victorian Land Use Information System (VLUIS): A new method for creating land use data for Victoria, Australia. In 'Surveying and Spatial Sciences Biennial Conference'. Wellington, New Zealand pp. 155-167

Mumford JM, Norton GA (1984) Economics of decision making in pest management. *Annual Review of Entomology* 29, 157-174.

Nemeth J (2004) Practice of applying streptomycin to control fire blight in Hungary. *OEPP/EPPO Bulletin* 34, 381-382.

Newley P (2010) Panama disease in bananas. *Primefacts* 1029, 1-2.

Norelli JL, Jones AL, Aldwinckle HS (2003) Fire blight management in the twenty-first century: using new technologies to enhance host resistance in apple. *Plant Disease* 87, 756-765.

Norton BG (1998) Improving ecological communication: The role of ecologists in environmental policy formation. *Ecological Applications* 8, 350-364.

NSWDOCS (2000) 'Assessment of the needs of the mangrove mountain community following the outbreak of the Newcastle Chicken Disease in April 1999.' New South Wales Department of Community Services, Sydney.

O'Loughlin GT, East RA, Meats A (1984) Survival, Development rates and Generation Times of Queensland fruit fly, *Dacus tryoni*, in marginally favourable climate: Experiments in Victoria. *Australian Journal of Zoology* 32, 353-361.

OEPP/EPPO 1992. EPPO Standadrs PM 3/40 *Erwinia amylovora:* sampling and test methods. Bulletin OEPP/EPPO 22, 229-231.

Okubo A, Levin SA (2002) 'Diffusion and ecological problems: modern perspectives.' (Springer: New York)

Ostmark HE (1974) Economic Insect Pests of Bananas. *Annual Review of Entomology* 19, 161-176.

Paini DR, Worner SP, Cook DC, Barro PJD, Thomas MB (2010) Using a self-organizing map to predict invasive species: sensitivity to data errors and a comparison with expert opinion. *Journal of Applied Ecology* 47, 290–298.

Parker IM, Simberloff D, Parker IM, Simberloff D, Lonsdale WM, Goodell K, Wonham M, Kareiva PM,Williamson MH, Von Holle B, Moyle PB, Byers JE, Goldwasser L (1999) Impact: Toward a Framework for Understanding the Ecological Effects of Invaders. *Biological Invasions* 1, 3-19.

Penning-Rowsell E, Johnson C, Tunstall S (2006) 'Signals' from pre-crisis discourse: Lessons from UK flooding for global environmental policy change? *Global Environmental Change-Human and Policy Dimensions* 16, 323-339.

Perrings C (1998) Resilience in the dynamic of economy-environment systems. *Environmental and Resource Economics* 11, 503-520.

Peterson AT, Papes M, Soberon J (2008) Rethinking receiver operating characteristic analysis applications in ecological niche modelling. *Ecological Modelling* 213, 63-72.

Peterson AT, Vieglais DA (2001) Predicting Species Invasions Using Ecological Niche Modeling: New Approaches from Bioinformatics Attack a Pressing Problem. *BioScience* 51, 363-372.

Peterson GD, Carpenter SR, Brock WA (2003) Uncertainty and the management of multistate ecosystems: An apparently rational route to collapse. *Ecology* 84, 1403-1411.

Peterson R, Grice K, Goebel R (2005) Eradication of black leaf streak disease from banana growing areas in Australia. *InfoMusa* 14, 7-10.

Pettit C, Pullar D (1999) An integrated planning tool based upon multiple criteria evaluation of spatial information. *Computers, Environment and Urban Systems* 23, 339-357.

PHA (2005) 'Government and Plant Industry Cost Sharing Deed in Respect of Emergency Plant Pest Responses.' (Plant Health Australia: Canberra)

Pheloung PC, Williams PA, Halloy SR (1999) A weed risk assessment model for use as a biosecurity tool evaluating plant introductions. *Journal of Environmental Management* 57, 239-251.

Phillips SJ, Anderson RP, Schapire RE (2006) Maximum entropy modeling of species geographic distributions. *Ecological Modelling* 190, 231–259.

Pielke RA, Jr. (2007) 'The Honest Broker: Making Sense of Science in Policy and Politics.' (Cambridge University Press: Cambridge)

Pimentel D, Zuniga R, Morrison D (2005) Update on the environmental and economic costs associated with alien-invasive species in the United States. *Ecological Economics* 52, 273-288.

Pitt JPW, Worner SP, Suarez AV (2009a) Predicting Argentine ant spread over the heterogeneous landscape using a spatially explicit stochastic model. *Ecological Applications* 19, 1176-1186.

Pitt JPW, Worner SP, Suarez AV (2009b) Predicting Argentine ant spread over the heterogeneous landscape using a spatially explicit stochastic model. *Ecological Applications* 19, 1176-1186.

Plant Protection Amendment Regulation (No. 4) (2003) No. 303, 2003 (Queensland)

Plant Protection Regulation (2003) (Queensland)

Ploetz RC (1994) Panama disease: Return of the first banana menace. *International Journal of Pest Management* 40, 326-336.

Ploetz RC (2000) Black Sigatoka. *Pesticide Outlook* 11, 19-23.

Proctor W, Drechsler M (2003) Deliberative Multi-criteria Evaluation. In 'Environment and Planning: Government and Policy'. (Pion: London)

Proctor W, Drechsler M (2006) Deliberative multicriteria evaluation. *Environment and Planning C-Government and Policy* 24, 169-190.

Pusey, PL (2000) The role of water in epiphytic colonization and infection of pomaceous flowers by *Erwinia amylovora*. *Phytopathology* 90, 1352-1357.

Pyšek P, Richardson DM (2010) Invasive species, environmental change and management, and health. *Annual Review of Environment and Resources* 35, 25-55.

Pysek P, Richardson DM, Pergil J, Jarosik V, Sixtova Z, Weber E (2008) Geographical and taxonomic biases in invasion ecology. *Trends in Ecology and Evolution* 23, 237-244.

Rafoss T (2003) Spatial stochastic simulation offers potential as a quantitative method for pest risk analysis. *Risk Analysis* 23, 651-661.

Railsback SF, Lytinen SL, Jackson SK (2006) Agent-based simulation platforms: Review and development recommendations. *Simulation* 82, 609-623.

Randin CF, Dirnböck T, Dullinger S, Zimmermann NE, Zappa M, Guisan A (2006) Are niche-based species distribution models transferable in space? *Journal of Biogeography* 33, 1689-1703.

Ransom LM (1997) The eradication of *Nectria galligena* from apple trees in Tasmania, 1954 to 1991. *Australasian Plant Pathology* 26, 121-125.

Rauschmayer F, Wittmer H (2006) Evaluating deliberative and analytical methods for the resolution of environmental conflicts. *Land Use Policy* 23, 108-122.

Redpath SA, Arroyo BE, Leckie EM, Bacon P, Bayfield N, Gutierrez RJ, Thirgood SJ (2004) Using decision modeling with stakeholders to reduce human-wildlife conflict: a Raptor-Grouse case study. *Conservation Biology* 18, 350-359.

Regan HM, Akçakaya HR, Ferson S, Root KV, Carroll S, Ginzburg LR (2003) Treatments of Uncertainty and Variability in Ecological Risk Assessment of Single-Species Populations. *Human and Ecological Risk Assessment* 9, 889-906.

Regan HM, Ben-Haim Y, Langford B, Wilson WG, Lundberg P, Andelman SJ, Burgman MA (2005) Robust decision-making under severe uncertainty for conservation management. *Ecological Applications* 15, 1471-1477.

Regan HM, Colyvan M, Burgman MA (2002) A taxonomy and treatment of uncertainty for ecology and conservation biology. *Ecological Applications* 12, 618-628.

Renn O (1999) A model for an analytic-deliberative process in risk management. *Environmental Science and Technology* 33, 3049-3055.

Renn O (2003) The challenge of integrating deliveration and expertise. In 'Risk analysis and society: an interdisciplinary characterization of the field'. (Eds T McDaniels and MJ Small) pp. 289-366. (Cambridge University Press: Cambridge, UK.)

Ricciardi A, Cohen J (2007) The invasiveness of an introduced species does not predict its impact. *Biological Invasions* 9, 309-315.

Richardson DM, Thuiller W (2007) Home away from home - objective mapping of high-risk source areas for plant introductions. *Diversity and Distributions* 13, 299-312.

Roberts RG, Sawyer AJ (2008) An updated pest risk assessment for spread of *Erwinia amylovora* and fire blight via commercial apple fruit. *Crop Protection* 27, 362-368.

Roberts RG, Hale CN, van der Zwet T, Miller CE, Redlin SC (1998) The potential for spread of *Erwinia amylovora* and fire blight via commercial apple fruit; a critical review and risk assessment. *Crop Protection* 17, 19-28.

Robertson DP, Hull RB (2003) Public ecology: an environmental science and policy for global society. *Environmental Science and Policy* 6, 399-410.

Robinet C, Roques A, Pan H, Fang G, Ye J, Zhang Y, Sun J (2009) Role of human-mediated dispersal in the spread of the pinewood nematode in China. *PLoS ONE* 4, e4646.

Rodoni B, Gardner R, Giles R, Cole M, Wimalajeewa S, van der Zwet T (2002) National surveys did not detect *Erwinia amylovora* on host plants in Australia. *Acta Horticulturae* 590, 39-45.

Rodoni B, Kinsella M, Gardner R, Gillings M, Geider K (1999) Detection of *Erwinia amylovora*, the causal agent of fire blight, in the Royal Botanic gardens, Melbourne, Australia. *Acta Horticulturae* 489, 169-170.

Rodoni B, Merriman P, McKirdy SJ, Wittwer G (2006) Costs associated with fire blight incursion management and predicted costs of future incursions. *Acta Horticulturae* 704, 55-61.

Rodriguez-Labajos B, Binimelis R, Monterroso I (2009) Multi-level driving forces of biological invasions. *Ecological Economics* 69, 63-75.

Rosenkranz P, Aumeier P, Ziegelmann B (2010) Biology and control of Varroa destructor. *Journal of Invertebrate Pathology* 103, S96-S119.

Roura-Pascual N, Krug RM, Richardson DM, Hui C (2010) Spatially-explicit sensitivity analysis for conservation management: exploring the influence of decisions in invasive alien plant management. *Diversity and Distributions* 16, 426-438.

Sagarin RD, Gaines SD, Gaylord B (2006) Moving beyond assumptions to understand abundance distributions across the ranges of species. *Trends in Ecology and Evolution* 21, 524-530.

Sapoukhina N, Tyutyunov Y, Sache I, Arditi R (2010) Spatially mixed crops to control the stratified dispersal of airborne fungal diseases. *Ecological Modelling* 221, 2793-2800.

Sarewitz D (2004) How science makes environmental controversies worse. *Environmental Science and Policy* 7, 385-403.

Severin V, Constantinescu F, Jianu F (1999) Appearance, expansion, and chemical control of fire blight (*Erwinia amylovora*) in Romania. *Acta Horticulturae* 489, 80-84.

Sharov AA (2004) Bioeconomics of managing the spread of exotic pest species with barrier zones. *Risk Analysis* 24, 879-892.

Sharov AA, Liebhold AM (1998) Model of slowing the spread of gypsy moth (Lepidoptera : Lymantriidae) with a barrier zone. *Ecological Applications* 8, 1170-1179.

Shea K, Possingham HP, Murdoch WW, Roush R (2002) Active adaptive management in insect pest and weed control: Intervention with a plan for learning. *Ecological Applications* 12, 927-936.

Shigesada N, Kawasaki K (1997) 'Biological Invasions: Theory and Practice.' (Oxford University Press: Oxford)

Shimanuki H, Calderone N, Knox D (1994) Parasite mite syndrome: the symptoms. *American Bee Journal* 134, 827-828.

Shivas RG, Wood PM, Darcy MW, Peg KG (1995) First Record of *Fusarium oxysporum* f. sp. *cubense* on Cavendish Bananas in Western Australia. *Australasian Plant Pathology* 24, 38-43.

Simberloff D (2005) The politics of assessing risk for biological invasions: the USA as a case study. *Trends in Ecology and Evolution* 20, 216-222.

Simberloff D (2006) Risk assessments, blacklists, and white lists for introduced species: are predictions good enough to be useful? *Agricultural and Resource Economics Review* 35, 1-10.

Simberloff D (2009) The Role of Propagule Pressure in Biological Invasions. *Annual Review of Ecology Evolution and Systematics* 40, 81-102.

Simberloff D, Alexander M (1998) Assessing risks to ecological systems from biological introductions. In 'Handbook of environmental risk assessment and management'. (Ed. P Calow) pp. 147-176. (Blackwell Science: Oxford)

Simberloff D, Gibbons L (2004) Now you see them, now you don't - population crashes of established introduced species. *Biological Invasions* 6, 161-172.

Simberloff D, Parker IM, Windle PN (2005) Introduced species policy, management, and future research needs. *Frontiers in Ecology and the Environment* 3, 12-20.

Simmonds NW (1966) 'Bananas.' (Longman: London)

Sivicek P (2004) Results of the detection survey for fireblight in Slovakia. *OEPP/EPPO Bulletin* 34, 357-359.

Skellam JG (1951) Random dispersal in theoretical populations. *Biometrika* 38, 196-218.

Sletten A, 1990. Fire blight in Norway. Acta Horticulturae 273, 37-40.

Sletten A, Melboe NS (2004) Experiences with the control of fire blight in Norway during 1986/2003. *OEPP/EPPO Bulletin* 34, 361-363.

Slovic P (1999) Trust, emotion, sex, politics, and science: Surveying the risk-assessment battlefield (Reprinted from Environment, ethics, and behavior, pg 277-313, 1997). *Risk Analysis* 19, 689-701.

Smith CS, Lonsdale WM, Fortune J (1999) When to Ignore Advice: Invasion Predictions and Decision Theory. *Biological Invasions* 1, 89-96.

Smith TJ, Pusey PL (2010) CougarBlight 2010, a significant update of the CougarBlight fire blight infection risk model. *Acta Horticulturae* 896, 331-336.

Sosnowski MR, Fletcher JD, Daly AM, Rodoni BC, Viljanen-Rollinson SLH (2009) Techniques for the treatment, removal and disposal of host material during programmes for plant pathogen eradication. *Plant Pathology* 58, 621-635.

Stansbury C (2000) 'HortGuard Threat Data Sheet for the Banana Industry in WA - Moko, Blood, Bugtok and Tapurok Diseases.' Government of Western Australia, Bunbury.

Stener PW, Lightner GW (1996) MARYBLYT 4.3, a predictive program for forecsting fire blight disease in apples and pears. Gempler. Inc. PO Box 270, Mt. Hebron, WI 53572.

Stephens AEA, Kriticos DJ, Leriche A (2007) The current and future potential geographic distribution of the Oriental fruit fly, *Bactrocera dorsalis*, (Diptera: Tephritidae). *Bulletin of Entomological Research* 97, 369-378.

Stevenson MA, Benard H, Bolger P, Morris RS (2005) Spatial epidemiology of the Asian honey bee mite (*Varroa destructor*) in the North Island of New Zealand. *Preventive Veterinary Medicine* 71, 241-252.

Stirling A (2006) Analysis, participation and power: justification and closure in participatory multi-criteria analysis. *Land Use Policy* 23, 95-107.

Store R, Jokimaki J (2003) A GIS-based multi-scale approach to habitat suitability modelling. *Ecological Modelling* 169, 1-15.

Store R, Kangas J (2001) Integrating spatial multi-criteria evaluation and expert knowledge for GIS-based habitat suitability modelling. *Landscape and Urban Planning* 55, 79-93.

Stover RH (1972) 'Banana, Plantain and Abaca Diseases.' (Commonwealth Mycological Institute: Kew)

Strayer DL (2009) Twenty years of zebra mussels: lessons from the mollusk that made headlines. *Frontiers in Ecology and the Environment* 7, 135-141.

Strayer DL, Eviner VT, Jeschke JM, Pace ML (2006) Understanding the long-term effects of species invasions. *Trends in Ecology & Evolution* 21, 645-651.

Sutherst RW, Baker RHA, Coakley SM, Harrington R, Kriticos DJ, Scherm H (2007) Pests Under Global Change - Meeting Your Future Landlords? In 'Terrestrial Ecosystems in a Changing World'. (Eds JG Canadell, DE Pataki and LF Pitelka) pp. 211-223. (Springer: Berlin)

Sutherst RW, Bourne AS (2009) Modelling non-equilibrium distributions of invasive species: a tale of two modelling paradigms. *Biological Invasions* 11, 1231-1237.

Sutherst RW, Maywald G (2005) A Climate Model of the Red Imported Fire Ant, *Solenopsis invicta* Buren (Hymenoptera: Formicidae): Implications for Invasion of New Regions, Particularly Oceania. *Environmental Entomology* 34, 317-335.

Swinburne TR (1975) European canker of apple (*Nectria galligena*). *Review of Plant Pathology*, 54(10):787-799.

Thomas JE, Dietzgen RG (1996) 'Development of detection methods for banana bunch top virus with monoclonal antibodies.' (Horticultural Research and Development Corporation: Gordon, NSW)

Thomson, SV (1986) The role of the stigma in fire blight infections. *Phytopathology* 76, 476-482.

Trope Y, Liberman N, Wakslak C (2007) Construal Levels and Psychological Distance: Effects on Representation, Prediction, Evaluation, and Behavior Construal Level Theory *Journal of Consumer Psychology* 17, 83–95.

Ulubasoglu M, Mallick D, Wadud M, Hone P, Haszler H (2011) 'How Price Affects the Demand for Food in Australia - An analysis of domestic demand elasticities for rural marketing and policy.' Rural Industries Research and Development Corporation, 11/062, Canberra.

US National Research Council (1996) 'Understanding risk: informating decisions in a democratic society ' (Nationa Academy Press: Washington, DC.)

Valle D, Staudhammer CL, Cropper WP, van Gardingen PR (2009) The importance of multimodel projections to assess uncertainty in projections from simulation models. *Ecological Applications* 19, 1680-1692.

Van der Zwet T, Beer SV (1991) Fire blight: Its nature, prevention, and control – a practical guide to integrated disease management. US Department of Agriculture Information Bulletin No. 631, 34 pp.

Vanneste JL, Eden-Green S (2000) Migration of Erwinia amylovora in hosts plant tissues. p. 73-84. In: J.L. Vanneste (ed.), Fire Blight: the disease and its causative agent, *Erwinia amylovora*. CABI Publishing, Wallingford, UK.

Veitch SM, Bowyer JK (1996) ASSESS: A System for Selecting Suitable Sites. In 'Raster Imagery in Geographic Information Systems'. (Eds S Morain and S Lopez-Baros) pp. 495. (OnWord Press: Santa Fe)

Venette RC, Kriticos DJ, Magarey RD, Koch FH, Baker RHA, Worner SP, Gómez Raboteaux NN, McKenney DW, Dobesberger EJ, Yemshanov D, De Barro PJ, Hutchison WD, Fowler G, KalarisTM, Pedlar J (2010) Pest risk maps for invasive alien species: a roadmap for improvement. *Bioscience* 80, 349-362.

Venette RC, Moon RD, Hutchison WD (2002) Strategies and statistics of sampling for rare individuals. *Annual Review Entomology* 47, 143-174.

Verghese A, Tandon PL, Stonehouse J (2004) Economic evaluation of integrated management of the oriental fruit fly, *Bactrocera dorsalis* (Diptera: Tephritidae) in mango in India. *Crop Protection* 23, 61-63.

Vilà M, Basnou C, Pyšek P, Josefsson M, Genovesi P, Gollasch S, Nentwig W, Olenin S, Roques A, Roy D, Hulme PE, DAISIE partners (2010) How well do we understand the impacts of alien species on ecosystem services? A pan-European, cross-taxa assessment. *Frontiers in Ecology and the Environment* 8, 135-144.

Villalta ON, Washington WS, Rimmington GM, MacHardy WE (2001) Environmental factors influencing maturation and release of ascospores of *Venturia pirina* in Victoria, Australia. *Australian Journal of Agricultural Research* 52, 825-837.

Waage JK, Fraser RW, Mumford JD, Cook DC, Wilby A (2005) 'A New Agenda for Biosecurity ' Department for Food, Environment and Rural Affairs, London.

Waage JK, Mumford JD (2008) Agricultural biosecurity. *Philosophical Transactions of the Royal Society B-Biological Sciences* 363, 863-876.

Walduck G, Daly A (2006) 'Panama Disease in Bananas, Information Sheet IS27.' (Department of Primary Industries, Fisheries and Mines: Darwin)

Walker B, Carpenter S, Anderies J, Abel N, Cumming G, Janssen M, Lebel L, Norberg J, Peterson GD, Pritchard R (2002a) Resilience management in social-ecological systems: a working hypothesis for a participatory approach. *Conservation Ecology* 6.

Walker J, Veitch S, Dowling T, Braaten R, Guppy L, Herron N (2002b) 'Assessment of catchment condition.' CSIRO, Canberra.

Webb TJ, Raffaelli D (2008) Conversations in conservation: revealing and dealing with language differences in environmental conflicts. *Journal of Applied Ecology* 45, 1198-1204.

Wilensky U (1999) NetLogo. In. (Center for Connected Learning and Computer-Based Modeling, Northwestern University. Evanston, IL)

Williamson M (1999) Invasions. *Ecography* 22, 5-12.

Wilson RS (2008) Balancing Emotion and Cognition: a Case for Decision Aiding in Conservation Efforts. *Conservation Biology* 22, 1452-1460.

Wittwer G, McKirdy S, Wilson R (2005) Regional economic impacts of a plant disease incursion using a general equilibrium approach. *The Australian Journal of Agricultural and Resource Economics* 49, 75-89.

Worner SP, Gevrey M (2006) Modelling global insect pest species assemblages to determine risk of invasion. *Journal of Applied Ecology* 43, 858–867.

Wu F (1998) SimLand: a prototype to simulate land conversion through the integrated GIS and CA with AHP-derived transition rules. *International Journal of Geographical Information Science* 12, 63-82.

Wynne B (1992) Uncertainty and Environmental Learning - Reconceiving Science and Policy in the Preventive Paradigm. *Global Environmental Change-Human and Policy Dimensions* 2, 111-127.

Xu XM, Butt DJ (1994) The biology and epidemiology of *Nectria galligena* and an infection warning system. *Norwegian Journal Agricultural Science* Sup. 17, 317-324.

Yemshanov D, Koch FH, McKenney DW, Downing MC, Sapio F (2009) Mapping Invasive Species Risks with Stochastic Models: A Cross-Border United States-Canada Application for Sirex noctilio Fabricius. *Risk Analysis* 29, 868-864.

Appendix 1: Threat Data Sheets

THREAT DATA

Banana Bunchy Top

(virus)

Source: http://www.issg.org/database/image.asp?ii=1132&ic=e

CRC10162

Communicating Uncertainty in Biosecurity Adaption

CRC PLANT biosecurity

Banana Bunchy Top

(*Banana bunchy top virus – BBTV*)

Banana bunchy top is one of the most damaging diseases of banana worldwide and is caused by the banana bunchy top virus (BBTV). Infected banana plants are stunted and produce small, deformed fruits. In advanced stages of the disease, plants do not produce any fruit. Infected banana plants are useless and serve only as a source of the virus. A tiny insect called the banana aphid spreads the disease by carrying the virus to healthy plants after feeding on infected plants. Banana bunchy top virus is a regulated disease under active quarantine in Australia.

Host Range: BBTV occurs in *Musa* (including banana, abaca, plantain and ornamental bananas) and *Ensete* in the family Musaceae. Although there are some reports of monocot hosts in related families, evidence is conflicting, and Musaceae are generally considered the only hosts (reviewed by Thomas and Iskra-Caruana 1999). Hosts reported for the banana aphid include various species in the families Musaceae (*Musa, Heliconia*), Araceae (*Dieffenbachia*) and Zingiberaceae (*Elettaria*), many of which are not hosts of BBTV.

Distribution: BBTV is widespread in Southeast Asia, the Philippines, Taiwan, most of the South Pacific islands, and parts of India and Africa (Figure 1). In Hawaii, BBTV was first observed in 1989 and is now widely established. In Australia, the virus was introduced with imported banana plants in early 1900s and devastated the south east Queensland banana industry in the 1920s. Today, BBTV is restricted to northern New South Wales and southern Queensland. Quarantine measures are in force to prevent its introduction to northern Queensland and other states (Biosecurity Queensland 2009, http://www.dpi.qld.gov.au/4790_8393.htm).

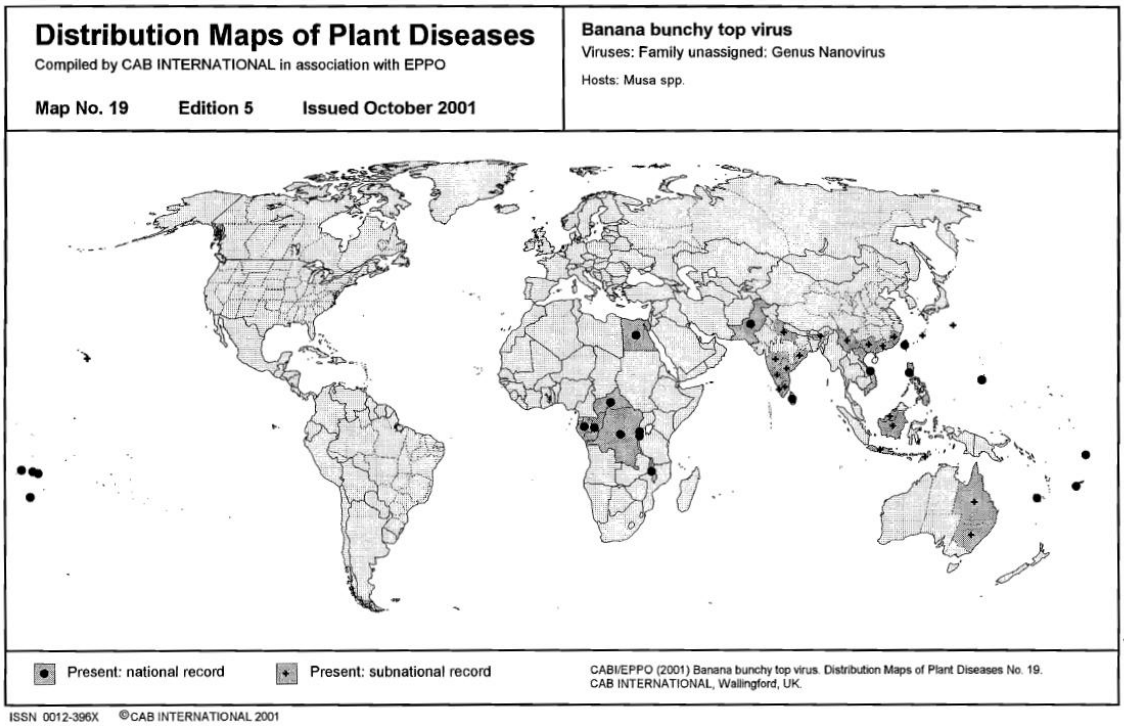

Figure 1. Geographic distribution map for Banana bunchy top virus (2001)

Biology and Ecology: Banana bunchy top virus (BBTV**)** is a plant pathogenic virus of the family Nanoviridae. BBTV is spread by the banana aphid (*Pentalonia nigronervosa*), which acquires the virus after at least four (but usually about 18) hours of feeding on an infected plant. The aphid can retain the virus through its adult life, for a period of 15-20 days. During this time, the aphid can transmit the virus to a healthy banana plant by feeding on it, possibly for as little as 15 minutes but more typically for about two hours.

Banana bunchy top is systemic. Following aphid inoculation, symptoms generally do not appear until a further two or more leaves have been produced. This period can vary between 19 days in summer to 125 days in winter. Suckers produced on an infected stool generally develop symptoms before reaching maturity. BBTV is efficiently disseminated in conventional planting material.

Banana aphids have a worldwide distribution with a host range that includes other species in the Musaceae. On banana plants in New South Wales, aphids are found at the base of the pseudostem at soil level and for several centimetres below the soil surface, beneath the outer leaf sheaths and on newly emerging suckers. Aphid numbers decrease during periods of drought. Transmission efficiency by aphids varies from 46 to 67% and the virus is more efficiently acquired by nymphs than by adults.

Symptoms: The characteristic symptom is that of short erect leaves remaining bunched up, with yellow fringes (Figure 2). This bunchy top symptom is usually most visible on young plants and can be more subtle on older banana plants. Other characteristic symptoms are dark green, dot-dash flecks running along leaf veins, called "Morse code" (Figure 3), and hooking down along the midrib, called "J-hooks" (Figure 4). Dark green streaks run vertically down the leaf sheath into the pseudostem of the banana plant. New emerging leaves are progressively shorter, narrower and more erect. The stools fail to produce fruit.

Figure 2. Bananas infected with bunchy top virus have leaves that are bunched up, narrow, stiff, upright, and with yellow and irregular or wavy leaf margins. (Diseased plant: left;

healthy plant: right). Photo sourced from the University of Hawaii and the Hawaii Department of Agriculture website.

Figure 3. Morse coding of banana bunchy top virus (Photo: Ron Heu, Survey Entomologist - Hawaii Department of Agriculture)

Figure 4. Green J-hooks occur where the flat part of the banana leaf (the blade or lamina) meets the mid rib. Photo sourced from the University of Hawaii and the Hawaii Department of Agriculture website.

The banana aphids are black (Figure 5) and live in colonies often tended by ants. Their preferred feeding sites are young tender tissues such unfurled young leaves (Figure 6) and under petioles.

Figure 5. Banana aphid. Source: http://www.agnet.org/library/tn/2001001/

Figure 6. Colony of banana aphids on young leaf tissue. Source of photo - http://www.ctahr.hawaii.edu/bbtd/aphid_colonies.asp

Affected plant stages: All growth stages of plant

Affected plant parts: Whole plant.

Affected Industries: Banana industry.

Resistant plant varieties: All banana cultivars are susceptible.

Disease movement and Dispersal: BBTV is transmitted locally in a persistent, circulative manner by the banana aphid (*Pentalonia nigronervosa).* Distribution over long distances occurs by the movement of infected vegetative planting material such as suckers, corms, and tissue-cultured plantlets. BBTV is not soil-borne and is unlikely to be spread on cutting tools. Studies of disease outbreaks in commercial banana plantations found that the average distance of secondary spread of the disease by aphids was only 17 metres. Nearly two-thirds of new infections were within 20 metres of the nearest source of infection and 99 per cent were within 86 metres (Allen 1978, 1987). Wild patches of bananas can be an important source of BBTV.

Disease Impact: BBT is the most serious viral disease of bananas and plantains. Affected plants do not produce fruit and this leads to significant production loss. Devastating epidemics occurred in the past in Fiji and in Australia. Quarantine measures are currently the most effective means of control.

Disease Management: Affected plants must be destroyed. Control depends on prompt detection and destruction of infected stools, and effective banana aphid control. There are strict quarantine restrictions to prevent movement of contaminated planting material. Effective disease management relies on the use of uninfected planting material and intensive eradication schemes.

Quarantine Risk: Bunchy top virus is a regulated pest in Queensland, Australia (ref. http://www2.dpi.qld.gov.au/health/4203.html). Bunchy top virus has not been eradicated from southern Queensland, and the banana industry and the Queensland Government are keen to ensure the disease is kept out of north Queensland.

Key strategies for bunchy top control are:

- Gradual eradication from south-east Queensland and northern New South Wales by maintaining pressure on infested areas.
- Use of tissue-cultured, uninfected planting material wherever possible.
- Development of sensitive detection tests.
- Contributions to international research on alternative hosts and strains of the virus.
- Development of contingency plans for dealing with infection if bunchy top occurs in north Queensland.

Acknowledgements: The information was obtained from a variety of sources, particularly the Banana Bunchy Top Virus web pages of the University of Hawaii, Biosecurity Queensland, and the Global Invasive Species Database.

References

Allen RN (1978). Spread of bunchy top disease in established banana plantations. Australian Journal of Agricultural Research, 29(6):1223-1233

Allen RN (1987). Further studies on epidemiological factors influencing control of banana bunchy top disease and evaluation of control measures by computer simulation. Australian Journal of Agricultural Research, 38(2):373-382.

Thomas JE, Iskra-Carvana ML, Jones DR (1994). Banana bunchy top disease, Musa Disease Fact Sheet No. 4. Montpellier, France: INIBAP.

Thomas JE, Iskra-Caruana ML (1999). Bunchy Top. In: Jones DR, ed. Diseases of Banana, Abacá and Enset. Wallingford, UK: CAB International, 241-253.

http://www.ctahr.hawaii.edu/bbtd/

Global Invasive Species Database: http://www.issg.org/database/species/ecology.asp?si=141&fr=1&sts

http://www2.dpi.qld.gov.au/health/4203.html

http://www.dpi.qld.gov.au/4790_8393.htm

http://www.oisat.org/pests/diseases/viral/banana_bunchy_top_virus.html

http://www.new-ag.info/focus/focusItem.php?a=667

http://www.spc.int/pps/PDF%20PALs/PAL%2002_Banana%20Bunchy%20Top%20reprint%202005.pdf

THREAT DATA

Banana – Black Sigatoka (*Mycosphaerella fijiensis*)

Source: http://www.agripinoy.net/wp-content/uploads/2009/04/1_black_leaf_streak_sigatoka_leaf_bllight_1.jpg

CRC10162

Communicating Uncertainty in Biosecurity Adaption

CRC PLANTbiosecurity

Black Sigatoka

(*Mycosphaerella fijiensis*)

Black Sigatoka (BS), also known as black leaf streak, is caused by the fungus *Mycosphaerell fijiensis* and was first reported in 1963 in the Sigatoka valley in Fiji. BS is considered the most damaging and costly disease of banana. The disease reduces yield up to 50% and control measures account for 27% of total production costs. High rainfall and humidity suitable for banana cultivation favours BS development and spread. The economic impact of BS is dependent on climatic conditions, host varieties, cultural practices and other factors.

Host Range: *Mycosphaerella fijiensis* has only been recorded on *Musa* species (banana). *Musa* species and hybrids vary in their levels of resistance to this pathogen. The most widely grown commercial varieties in Australia are Cavendish, which is very susceptible, and Lady Finger, which is susceptible.

Distribution: *Mycosphaerella fijiensis* is found throughout the world's tropical banana growing regions (Figure 1) with the exception of Australia. Black Sigatoka, at the time known as black leaf streak, was first noticed as a new disease in Fiji in 1963, and subsequent surveys of the Oceanic region from 1964-1967 showed that it was already well established in the Pacific region by that time. Examination of herbarium specimens demonstrated that the pathogen was already present in Hawaii in 1958, in Papua New Guinea in 1957 and in Taiwan as early as 1927 (Jones 2002).

The disease now occurs throughout tropical regions of Africa, central and South America. The distribution throughout Asia is unclear, but it is confirmed as being present in southern China, Vietnam, Thailand, Taiwan, Singapore and parts of Malaysia and Indonesia.

Currently, mainland Australia is a designated 'black Sigatoka free' region. There have been nine incursions of the disease in North Queensland since 1981, all successfully eradicated; in Cape York (1981, 1984, 1999), Pascoe River (1991, 1998), the Bloomfield River area (1993), Weipa (1995), Daintree (1997) and the most recent in the Tully Valley in 2001 (Figure 2). Pest Free Area status was declared on the 20th December 2004. The history of black Sigatoga in Australia is well described by Henderson and Grice (2009) on the PaDIL Plant Biosecurity Toolbox website http://www.padil.gov.au/pbt.

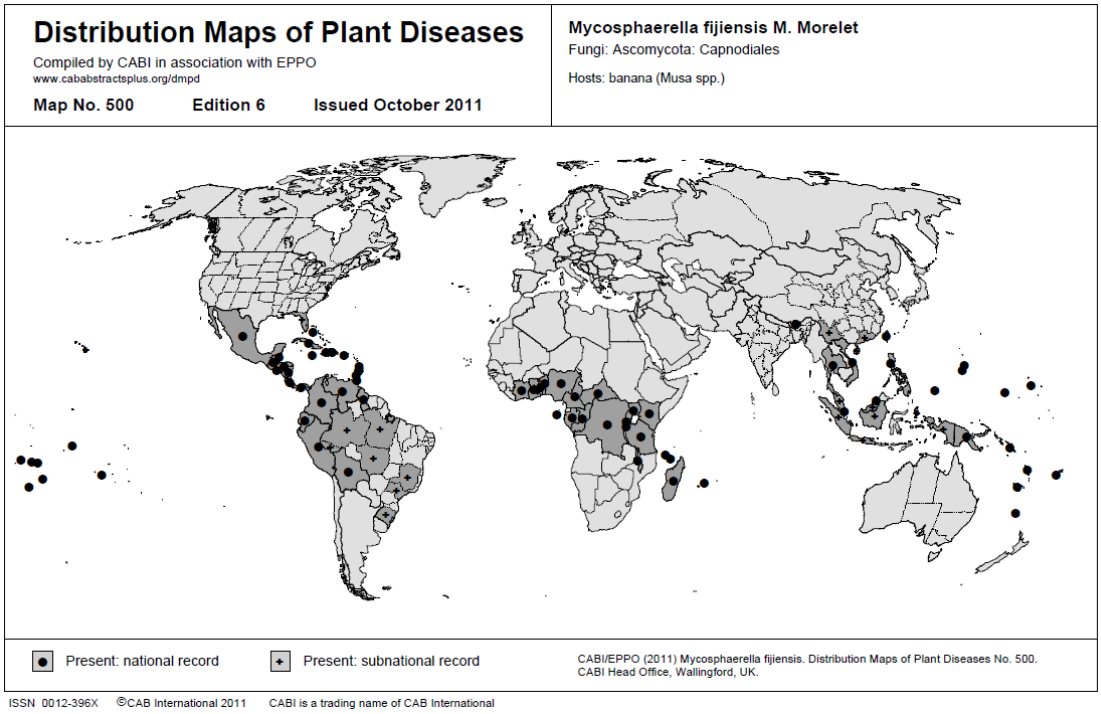

Figure 1. Distribution map for *Mycosphaerella fijiensis* (Black Sigatoka). CAB International October 2011.

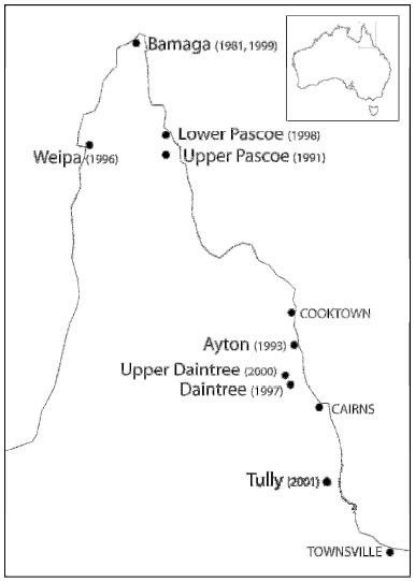

Figure 2: Map of Queensland showing the locations of all *Mycosphaerella fijiensis* incursions on mainland Australia since 1981. All incursions were eradicated by the Queensland Department of Employment, Economic Development and Innovation (DEEDI).

Biology and Life Cycle: The causal organism of black Sigatoka is the fungus *Mycosphaerella fijiensis.* The fungus produces two types of spores: asexual spores called conidia and sexual spores called ascospores. The ascospores are the main dispersal agent

and are produced in fruiting bodies embedded in the leaf spots of infected leaves. Ascospores are ejected from the fruiting bodies during wet weather and drift upwards in wind currents, depositing on the undersides of the banana leaves, generally on the terminal ends resulting in characteristic leaf tip infection. The cigar leaves are the most susceptible. Subsequent discharge of ascospores continues for several months, and diseased leaves fallen to the ground can continue to discharge ascospores until the material is fully disintegrated. The asexual spores, or conidia, are also produced in fruiting bodies in infected leaf spots and can initiate new infections. Both conidia and ascospores are dispersed within banana blocks by rain splash, but ascospores can be ejected into wind currents and therefore carried further.

If the leaf surface is wet or if the humidity is very high, the spores germinate within 2-3 hours at the optimum temperature of 27°C. The germ tubes grow over the leaf surface until they find leaf stomata (breathing holes) through which they enter the leaf. This can take 2-3 days. The incubation period (i.e. time between infection and symptom appearance) varies depending on weather conditions and host susceptibility. Under favourable conditions on a susceptible host, the first symptoms are apparent within 10 to 14 days, but in the dry season, the incubation period can be extended to 35 days.

Although ascospores are sensitive to UV radiation, it was believed that they can travel several hundred kilometres in wind currents. However, recent molecular studies of the genetic make-up of pathogen populations occurring in Australia suggest that dispersal is limited to approximately 50 metres, and long distance movement of the pathogen into Australia was much more likely due to transport of infected material by humans rather than by wind.

The latent period (i.e. time from infection to production of conidia) is determined by weather conditions and host susceptibility. Variations from 25 days (during the rainy season) to 70 days (during the dry season) have been recorded in Costa Rica.

Symptoms: BS causes large necrotic lesions on the leaves of the banana plant and early drop (collapse) of the entire leaf (Figure 4). This results in slower filling of fingers, reduced yields and premature ripening.

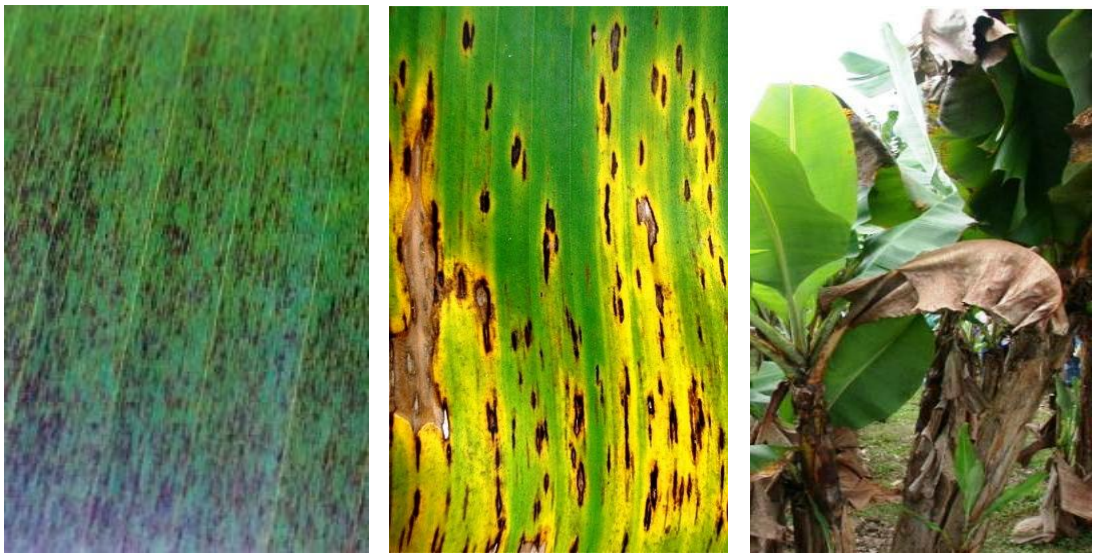

Figure 4. Black sigatoka disease symptoms on banana leaves. Note: Initial, middle and late stages of the symptoms (from left).
Source:http://www.padil.gov.au/pbt/index.php?q=node/13&pbtID=166

There are six recognised stages in symptom development (Fouré 1987; Meredith and Lawrence 1969). A brief description of each stage follows:

Stage 1: Initially, tiny specks < 0.25 mm and white to yellowish in colour that quickly turn a reddish brown, appear on the abaxial surface (underside) of the leaf laminar. This first stage is also known as the 'initial speck stage').

Stage 2: The tiny reddish brown specks elongate and widen, becoming streaks approximately 2mm X <1 mm. This stage is also referred to as the 'initial streak stage'. The streaks are more clearly visible on the abaxial surface of the leaf laminar than the adaxial surface (upper side) of the leaf. Conidia may be present (Figure 5(a)).

Figure 5(a): Stage 2 symptoms or 'First Streak Stage'. Conidia may be present at this stage. Note that Stage 1 symptoms (initial speck stage) are barely visible at <0.25mm. (Image sourced from Plant Biosecurity Toolbox, PaDIL)

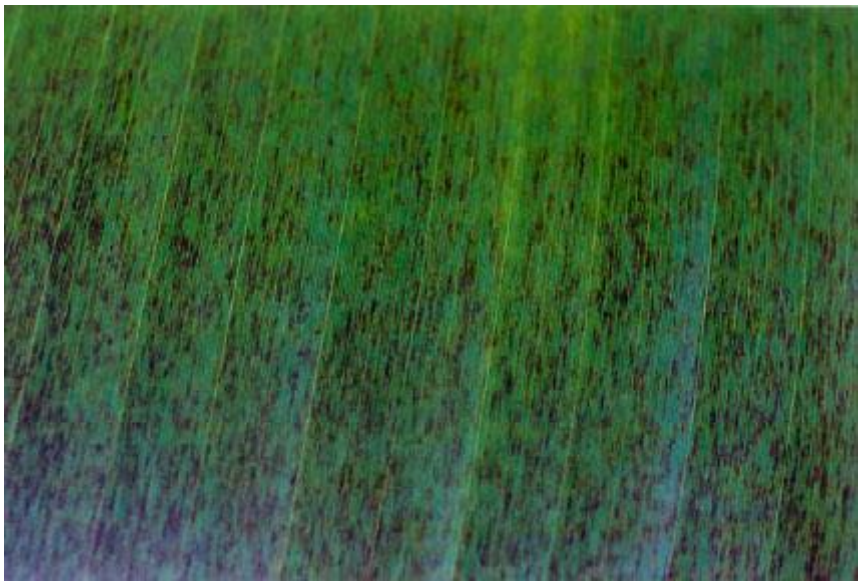

Figure 5(b): Stage 3 symptoms or 'Second Streak Stage'. Streaks are now almost black. Conidia are present. (Image and sourced from Plant Biosecurity Toolbox, PaDIL)

Stage 3: The streaks continue to expand in size and change colour to a very dark brown, almost black, colour. This is also referred to as the 'second streak stage'. Where infection is heavy, the streaks overlap to give a black appearance to large areas of the leaf. The streaks are clearly visible from the adaxial side of the leaf. Conidia are present at this stage (Figure 5(b)).

Stage 4: The streaks continue to enlarge and become more elliptical in shape as it broadens and a water-soaked border may develop around the edges. This stage is known as the 'first spot stage' (Figure 5(c)).

Figure 5(c): Stage 4 symptoms or 'First Spot Stage'. The streaks are becoming more elliptical and have a watersoaked border. (Image sourced from Plant Biosecurity Toolbox, PaDIL)

Stage 5: This stage also known as the 'second spot stage' is characterised by the central region of the spot becoming slightly depressed. The water soaked border may develop a yellow halo around it. Where infection is heavy, large areas of leaf tissue collapses. (Figure 5(d)).

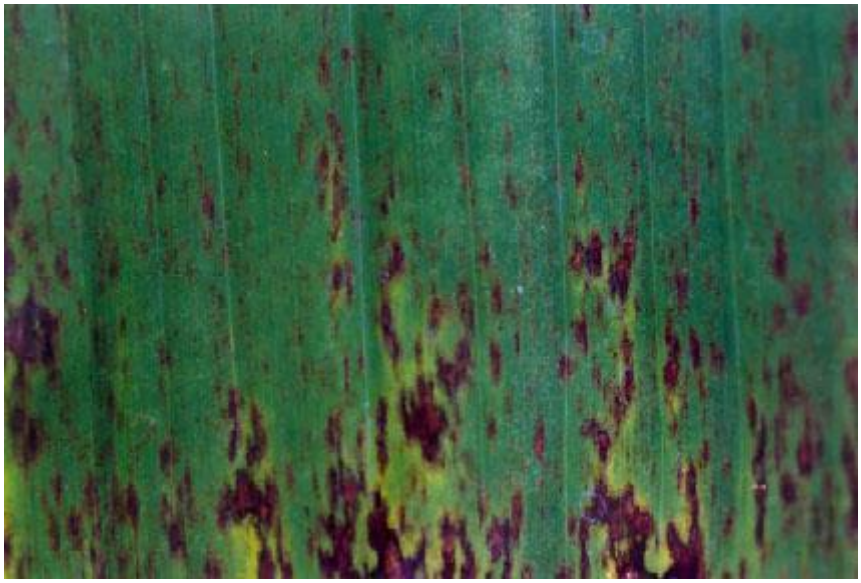

Figure 5(d): Stage 5 symptoms or 'Second Spot Stage'. Note the blackening in the centre of the spots. The watersoaked border begins to develop a yellow halo. (Image sourced from Plant Biosecurity Toolbox, PaDIL)

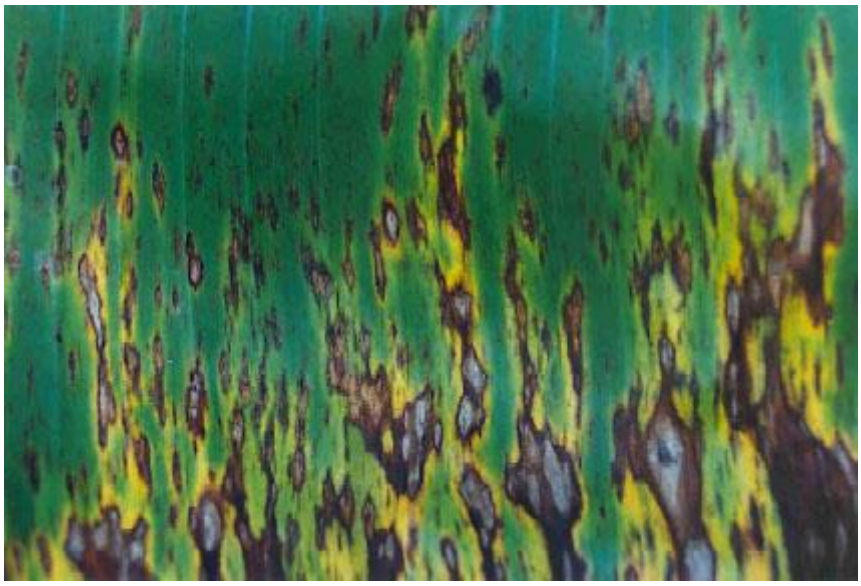

Figure 5(e): Stage 5 & 6 'Third Mature Spot' symptoms. Multiple lesion stages are present. Note the pale grey centres of the Stage 6 lesions. (Image sourced from Plant Biosecurity Toolbox, PaDIL)

Stage 6: The final stage, also referred to as the 'third spot stage', is when the centre of each spot becomes dry and pale grey to beige in colour. Fruiting bodies and ascospores are present in stage 6 lesions. Surrounding each of the spots is a distinctive black border. Where infection is heavy the large areas of the leaf become necrotic. The spots remain visible even after the death and desiccation of the leaf due to the dark border encircling each of the individual spots (Figure 5(e)).

Although similar, there are distinctive differences in the symptoms of black and yellow Sigatoka. Table 1 summarises the stages of both Sigatoka diseases.

Table 1. Summary of the different lesion stages associated with yellow and black Sigatoka leaf spot of bananas.

Lesion Stage	**Yellow Sigatoka**	**Black Sigatoka**
Stage 1	Very small light green dot or dash up to 1 mm long	Small pigmented spot of white or yellow, similar to yellow Sigatoka stage 1
Stage 2	Light green streak several millimetres long	Brown streak, visible on underside of leaf, later visible on leaf upper surface as yellow streak; colour changes progressively to brown, then black on upper leaf surface

Stage 3	An elongated rusty brown spot with an poorly defined border	Enlarged stage 2, streaks become longer
Stage 4	A mature spot with a dark brown sunken centre; often surrounded by a yellow halo, conidiophores and conidia are produced at this stage	Appears on leaf underside as brown spot, as a black spot on upper leaf surface
Stage 5	Spot has developed a grey, dried out centre and a peripheral black ring which is evident even after the leaf has dried out	Elliptical spot is totally black on the underside of the leaf, surrounded by a yellow halo
Stage 6		Centre of spot dries out, turns grey and is surrounded by a well-defined margin and a bright yellow halo

Affected plant stages: All growth stages are affected.

Affected plant parts: Leaves.

Affected Industries: Banana industry.

Resistant cultivars: *Musa* species and subspecies vary in their levels of resistance to *M. fijiensis*, but all commercial cultivars grown in Australia are susceptible.

Disease Movement and Dispersal: *M. fijiensis* is dispersed within banana blocks by rain splash of conidia. Movement between blocks is possible through the aerial spread of ascospores ejected from the perithecia. Ascospores are the main method of dispersal of *M. fijiensis* (Stover and Dickson 1976).

Long distance spread may also be via the wind dispersal of ascospores. The short time that ejected ascospores can survive UV irradiation suggests that the distance viable ascospores are dispersed by this method will also be affected by the amount of cloud cover and the distance travelled through the night (Parnell *et al.* 1998). In many cases long distance movement, especially intercontinental movement, of the pathogen is thought to be more likely due to the direct transportation of germplasm from an infected area to a new region (Rivas *et al.* 2004).

In case of international movement, infected planting material and leaves (often used as packing materials in developing world) are thought to be responsible for BS spread. The disease outbreak in Florida (USA) and northern parts of Australia almost certainly resulted

from the importation and or accidental introduction of infected plant materials by local growers. Long distance movement of banana leaves and planting materials is viewed as a quarantine risk.

Disease Impact: Black Sigatoka causes large necrotic lesions on the leaves of the banana plant and early drop (collapse) of the entire leaf. The resulting loss of photosynthetic capacity leads to slower filling of fingers, reduced yields and finger size and premature ripening of fingers. Field losses vary from 30-50% depending on the climatic conditions (Gauhl *et al.* 2000; Stover 1983) and are presently 5-10% in even well-managed plantations with good control strategies (http://www.padil.gov.au/pbt). In subsistence crops of plantain, yield loss has been estimated to be up to 33% during the first crop cycle and up to 76% in the second (Mobambo *et al.* 1996).

Disease Management: BS is a difficult and expensive disease to control. In commercial export plantations in the USA, BS is controlled by frequent aerial fungicide application but fungicide resistance is common. Cultural practices such as removing affected leaves, good drainage, and sufficient spacing help manage the disease, but these practices are labour intensive.

Quarantine Risk: Quarantine measures are in place in Australia to prevent spread of BS from the Torres Strait region to other areas (Jones, 1990).

Overall consequence: In 2008, Biosecurity Australia determined that the overall consequence to Australia of black sigatoka was deemed to be moderate. http://www.daff.gov.au/ba/ira/final-plant/banana-philippines

Acknowledgements: The information was obtained from the PaDIL Plant Biosecurity Toolbox Black Sigatoka pages, edited by Juliane Henderson: http://www.padil.gov.au/pbt. Contributors: Sharon Van Brunschot, André Drenth, Kathy Grice, Juliane Henderson, Julie Pattemore, Ron Peterson, Susan Porchun.

References

Gauhl F, Pasberg-Gauhl C, Jones DR (2000) Black leaf streak: Disease cycle and epidemiology. In 'Diseases of Banana, Abacá and Enset'. (Ed. DR Jones) pp. 56- 62. (CABI Publishing: Wallingford, UK)

Jones DR (2002) The distribution and importance of the *Mycosphaerella* leaf spot diseases of banana. In '*Mycosphaerella* leaf spot diseases of bananas: present status and outlook'. San José, Costa Rica. (Eds L Jacome, P Lepoivre, DH Marin, R Ortiz, RA Romero, JV Escalant) pp. 25-41. (INIBAP)

Jones DR, 1990. Black Sigatoka - a threat to Australia. In: Fullerton RA, Stover RH, eds. Sigatoka Leaf Spot Diseases of Bananas, Proceedings of an International Workshop held at San José, Costa Rica, March 28-April 1, 1989. Montferrier-sur-Lez, France: INIBAP, 38-46.

Henderson J, Grice K (2009) Black Sigatoka. Plant Biosecurity Toolbox, PADIL, http://www.padil.gov.au/pbt.

Mobambo KN, Gauhl F, Swennen R, Pasberg-Gauhl C (1996) Assessment of the cropping cycle effects on black leaf streak severity and yield decline of plantain and plantain hybrids. International Journal of Pest Management 42, 1-8.

Parnell M, Burt PJA, Wilson K, 1998. The influence of exposure to ultraviolet radiation in simulated sunlight on ascospores causing Black Sigatoka disease of banana and plantain. International Journal of Biometeorology, 42(1):22-27.

Rivas GG, Zapater M-F, Abadie C, Carlier J (2004) Founder effects and stochastic dispersal at the continental scale of the fungal pathogen of bananas *Mycosphaerella fijiensis*. Molecular Ecology 13, 471-482

Stover RH, Dickson JD (1976) Banana leaf spot caused by *Mycosphaerella musicola* and *M. fijiensis* var.*deformis*: a comparison of the first Central American epidemics. FAO Plant Protection Bulletin 24, 36-42.

Stover RH (1983) The effect of temperature on ascospore germinative tube growth of *Mycosphaerella musicola* and *Mycosphaerella fijiensis* var. *difformis*. Fruits 38, 625-628.

http://www.cabicompendium.org/cpc/report_select.asp?CCODE=MYCOFI

http://www.apsnet.org/online/feature/banana/Top.html

http://www.agripinoy.net/wpcontent/uploads/2009/04/1_black_leaf_streak_sigatoka_leaf_blight_1.jpg

THREAT DATA

Moko Disease and Banana Blood Disease

(*Ralstonia solanacearum*)

Source: http://www.actahort.org/members/showpdf?session=27431

CRC10162

Communicating Uncertainty in Biosecurity Adaption

CRC PLANT biosecurity

Moko disease and Banana blood disease (BBD)

(*Ralstonia solanacearum*)

Moko disease and banana blood disease are bacterial wilt diseases of banana, both caused by the bacterium *Ralstonia solanacearum*. The symptoms and epidemiology of the two diseases are very similar, but the causal agents are different. *Ralstonia solanacearum* is a species complex that consists of four phylotypes (genetic groups), with exceptional diversity amongst strains from different hosts and geographical origins. Blood disease is caused by a strain from phylotype IV and Moko disease by Race 2 from phylotype II. These two pathogens can be differentiated by their biological and biochemical properties. Race 2 occurs mainly in tropical areas from South and Central America causing moko and in the Philippines, causing bugtok disease. According to Fegan (2005), bugtok, which is only known in the Philippines, and moko are the same disease. Banana blood disease (BBD) was first reported in Indonesia in 1905 and remains confined to Indonesia. The name blood disease was originally adopted because droplets of a thick milky white, yellow or red-brown liquid often ooze out of the vascular tissues of infected plants at cut surfaces.

No resistant banana cultivars and effective control measures are known for either disease. Both diseases are absent from Australia.

Host Range: Cultivated and wild species of banana (*Musa*) are primary hosts of these pathogens. Phylotype IV (BBD) affects cultivars of both AAA and ABB genomic groups. Race 2 (Moko) also infects *Heliconia* species.

Distribution: Moko disease was first reported in Trinidad in the late 1890's from an outbreak that caused severe losses of Moko cooking bananas, hence the name of the disease. Moko disease is endemic to Central and South America, and has also been recorded from Africa and south east Asia (Figure 1).

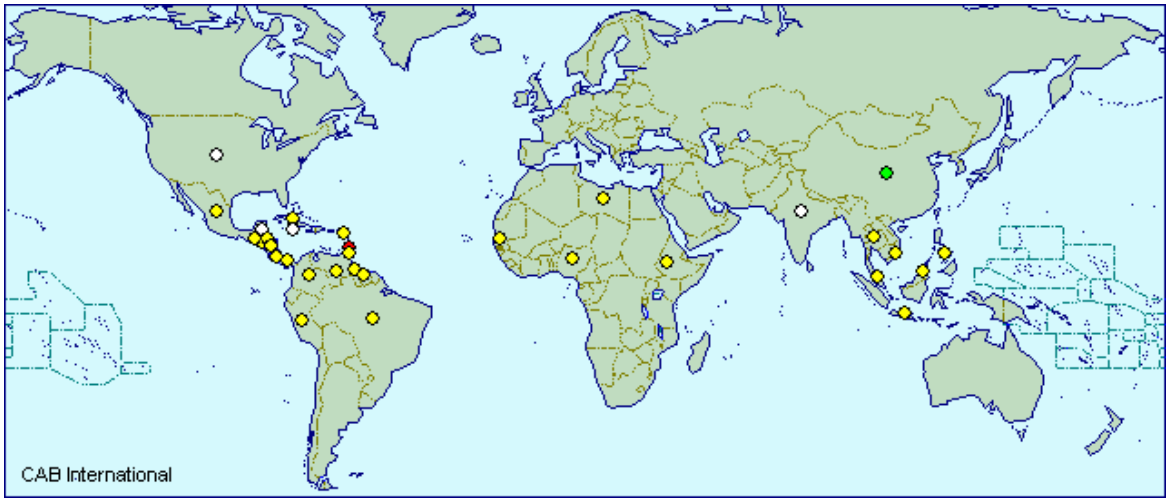

Figure 1. Distribution map for *Ralstonia solanacearum race 2* (moko disease)
For further details of the map see following reference in Crop Protection Compendium (CPC) – Moko disease Datasheet. Ref.
http://www.cabicompendium.org/cpc/datasheet.asp?CCODE=PSDMS2

BBD was first reported in Sulawesi, Indonesia, in 1905 but was not found in other parts of Indonesia until 1980. During the 1980s the disease spread first to Java, then Sumatra, Kalimantan, Bali and other islands (Buddenhagen 2009; Figure 2).

.

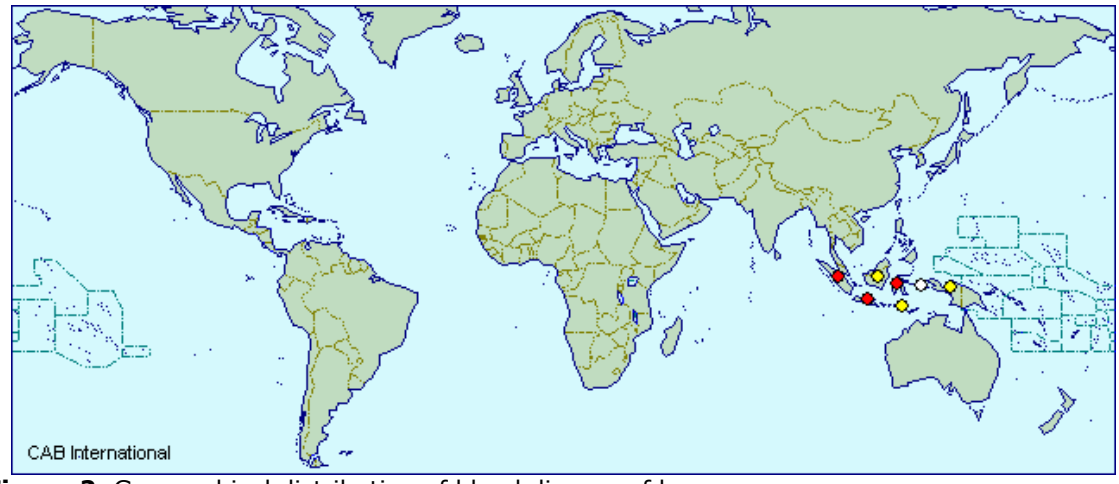

Figure 2. Geographical distribution of blood disease of banana.
http://www.cabicompendium.org/cpc/datasheet.asp?CCODE=RALSSP&COUNTRY=0

Biology and Ecology:

R. solanacearum is a gram negative soilborne bacterium that invades the host plant through the root system, colonising the xylem vessels resulting in wilting. Gäuman (1921, 1923) found that the bacteria can survive for over a year in soil infested by decaying diseased plant tissues and can infect the banana plant through wounds on suckers, pseudostem and fruits. All plant parts are affected and the bacterium can persist and remain infective for at least one year in soil contaminated by diseased plant remnants. Infected fruits can remain symptomless for several weeks, and may be marketed and subsequently discarded by the consumer. Bacteria are also readily spread by insects (bees and wasps) visiting the male banana flowers, in daughter suckers and on pruning knives.

Symptoms:

The symptoms of moko disease and banana blood disease (BDB) cannot be distinguished from each other. Although similar to Panama disease caused by *Fusarium oxysporum* f. sp. *cubense*, they can be distinguished from Panama disease by the presence of fruit symptoms and bacterial ooze.

Fruit rot and fruit stalk discoloration as well as wilting or blackened regrowth suckers are characteristic symptoms. On young plants, wilt can progress rapidly, taking a week or less from the initial symptoms to the collapse of the plant. Light to dark brown vascular discoloration occurs in the pseudostem, rhizome and in sheaths of the leaves (Figure 3). Bacterial ooze may exude as droplets from the cut surface of vascular tissues, mainly in the peduncle or pseudostem (Figure 4). Fruit can be smaller and the fruit pulp can show a firm brown or gray rot.

The sequence of symptoms depends on the route of infection and the ecotype of bacterial strain. If the infection occurs via the roots and rhizomes, yellowing and wilting of the oldest leaves will occur first. Fully expanded leaves of plants of all ages turn yellow then gradually become dry and necrotic. In mature plants, the base of the petiole collapses and the wilted

leaves hang down around the pseudostem (Figure 5). The youngest leaves stop growing and develop whitish and later necrotic panels in the lamina. Eventually, the entire plant is infected and collapses. Internally, vascular bundles exhibit a reddish-brown discoloration which may extend throughout the plant or remain confined to the central stem. Under moist conditions, cut vascular tissues exude droplets of bacterial ooze from white to reddish-brown or black colour.

Bacterial ooze can occur in the male inflorescence, especially from flower and bract scars in those genotypes that shed flowers and bracts, and the disease can be transmitted by insects visiting these flowers. This is thought to be one reason why certain ABB cultivars are much more susceptible to flower infection than other bananas (Davis *et al.* 2000). In this case, the symptoms occur initially in the flowers bud and peduncles, which become blackened and shrivelled. An additional symptom may occur on ABB cultivars. Instead of successively abscising, many bracts on the male buds remain on the peduncle, giving a clumped appearance. The bacteria spread to the fruit causing a reddish dry rot of the pulp. Afterwards the bacteria move down into the pseudostem towards the suckers. As the disease progresses all leaves became gradually yellow and necrotic (Stover & Espinoza 1992), then wilt, collapse and hang down. Red to brown necrotic marks are seen towards the centre of the pseudostem and/or peduncle when cut transversely. Daughter suckers may show general wilting, but infection is not always systemic and healthy suckers are sometimes produced (Eden-Green 1994). Some strains cause less severe symptoms. (Buddenhagen 1961, 1994, Stover 1972, Thwaites *et al.* 2000).

A very common symptom is a red brown dry shrivelled pulp in unripe fruits that look outwardly green and healthy (Figure 6). After some time, this is seen in every fruit of the bunch. The external symptoms usually develop at the beginning of ripening, when the fruits turn yellow or brown, collapse and decay (Gäuman 1921, 1923). Gäuman also observed the fruit flesh may be gradually dissolved and the cavity thus formed is filled to the base of the fruit with slimy, brownish-red fluid containing innumerable bacteria. The fruits finally collapse and decay into a rotten mass.

Figure 3. Red-brown vascular discolouration of Moko and banana blood diseases; longitudinal and cross-sections**.**

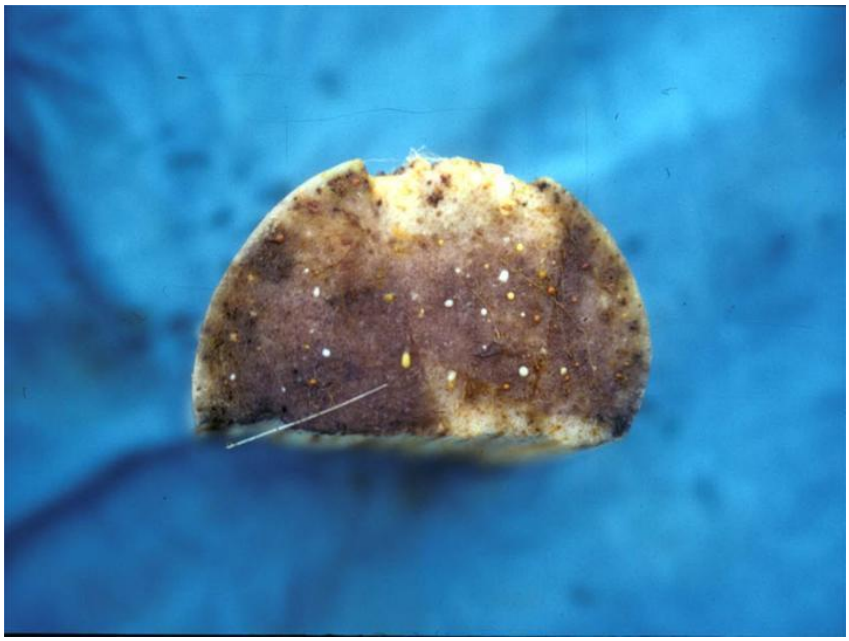

Figure 4. Bacterial ooze from cut surface of vascular tissue in banana infected with *Ralstonia solanacearum.*

Figure 5. Mature bananas dying of Moko disease. Wilted leaves hang down around the pseudostem. These symptoms are the same for banana blood disease.

Figure 6. Internal symptoms of Moko and banana blood disease in healthy-looking unripe fruit.

Source: http://www.padil.gov.au/img.aspx?id=3871&s=s

Affected plant stages: Flowering, fruiting and vegetative growing stages

Affected plant parts: Roots, leaves, stems, fruit, inflorescences, and whole plant.

Affected Industries: Banana industry.

Resistant plant variety: Currently, no information is available on resistant banana cultivars.

Disease Movement and Dispersal: The bacterium is soil-borne and capable of dispersing through contaminated soils and run-off water. It is known to be transmitted by insects, spreading 25 kilometres per annum in some areas in Indonesia. Spread by infected suckers and by symptomless infected banana fruit are important pathways into new areas.

Within a plantation the bacterium may move through an interconnecting network of roots and in surface water. It can also be spread in contaminated soil attached to farm machinery, implements, vehicles and on animals and workers feet. The pathogen can move long distances (national and international) through infected planting materials like rhizomes/suckers that usually do not exhibit any visible symptoms. Therefore, long distance movement of planting materials should be considered a quarantine risk.

Disease Impact: When present, these diseases are a major constraint to banana production as there are no effective control measures. Moko disease affects all plant parts in both growth

and production stages and losses of up to 80% have been reported in South and Central America and the Caribbean. Moko disease, or bugtok, in the Philippines is a potential threat and concern to Australian banana producers and quarantine authorities. Banana blood disease has been referred to as "the most serious threat to banana production in Indonesia" (Subijanto, 1990). Local reports from Indonesia (cited by Supriadi 2005) refer to national losses to the disease of 36% in 1991, losses of 64% in southern Lampung in 1997, and 100% losses in Lombok and Sumbawa, where the pisang kapok (ABB-type) banana was preferred. Widespread BBD in Indonesia is a potential threat to Australian banana producers and quarantine authorities.

Disease Management: There are no effective control methods once a plantation is infected. Field sanitation procedures and good cultural practices such as removal of infected plant material, disinfection of all tools and machinery can reduce the risk of spread of the disease.

Quarantine and exclusion procedures are effective in controlling the disease by restricting the movement of corms, suckers and soil that could be carrying the bacterium from infested to clean areas. The use of micropropagated planting material should be encouraged as this, if managed correctly, should be free from contamination by the pathogen. Plants derived from tissue culture and planted in soil where bananas have not been previously grown should remain free of BBD for a considerable period.

Quarantine Risk: Long distance movement of symptomless propagating material and fruit pose a quarantine risk.

Economic Impact: Yield losses are estimated to vary between 36 to 100%.

Environmental impact: Australia has several native species of Musa which may be adversely affected by the introduction of Moko and banana blood diseases.

Overall consequence: In 2008, Biosecurity Australia determined that the overall consequence to Australia of Moko disease was deemed to be high. http://www.daff.gov.au/ba/ira/final-plant/banana-philippines

Acknowledgements

The information was obtained from the PaDIL Plant Biosecurity Toolbox Banana Blood Disease and Moko Disease pages, written by RI Davis, JR Liberato and L. Gasparotto.

References

Davis RI & Liberato JR (2006) Banana blood disease (Blood disease bacterium) Updated on 7/8/2010 3:08:54 PM Available online: PaDIL - http://www.padil.gov.au.

Buddenhagen IW (1961) Bacterial wilt of bananas: history and known distribution. *Tropical agriculture*, Trinidad, **38**: 107-121.

Buddenhagen IW (1994) Moko disease. in: Ploetz RC, Zentmyer GA, Nishijima WT, Rohrbach KG, Ohr HD (eds.) Compendium of tropical fruit disease. St. Paul, APS Press. p.15-16.

Buddenhagen IW (2009). Blood bacterial wilt of banana: history, field biology and solution. Chronica Horticulturae 49 (2).

Davis RI, Fegan M, Tjahjono B and Rahamma S (2000). DISEASE NOTES OR NEW RECORDS - An outbreak of blood disease of banana in Irian Jaya, Indonesia. *Australasian Plant Pathology 29: 152*

Davis RI & Liberato JR (2007) Banana blood disease *(Blood disease bacterium)* Pest and Diseases Image Library. Updated on 12/22/2007 11:33:20 AM. Available online: http://www.padil.gov.au

Fegan M (2005) Bacterial wilt diseases of banana: evolution and ecology. In Allen C, Prior P & Hayward AC (eds). Bacterial wilt disease and the *Ralstonia solanacearum* species complex. (APS Press: St. Paul) p.379-386.

Gäumann E (1921) Onderzoekingen over de bloedziekte der bananen op Celebes I. (Investigations into the blood disease of bananas on Celebes Island) *Mededeelingen van het Instituut voor Plantenziekten* No. 50, 47p. in: *Review of Applied Mycology* **1**: 225-227, 1922.

Gäumann E (1923) Onderzoekingen over de bloedziekte der bananen op Celebes II (Investigations on the blood disease of banana in Celebes II). *Mededeelingen van het Instituut voor Plantenziekten* No. 59, 45p. in: *Review of Applied Mycology* **3**: 344-346, 1924.

Liberato JR & Gasparotto L (2006) Moko disease of banana (Ralstonia solanacearum) Updated on 7/8/2010 3:04:13 PM Available online: PaDIL - http://www.padil.gov.au

Stover RH (1972) Banana, plantain and abaca diseases. CAB: Kew, England.

Stover RH and Espinoza A (1992) -Blood disease of bananas in Sulawesi. *Fruits* 47: 61 1-613.

Subijanto, 1990. Country paper report on banana and plantain - Indonesia. In: Valmayor RV, ed. Banana and Plantain R&D in Asia and the Pacific. Proceedings of a Regional Consultation on Banana and Plantain R&D Networking, 20-24 November 1989, Manila and Davao. Montpellier, France: International Network for the Improvement of Banana and Plantain, 49-69.

Supriadi, 2005. Present status of blood disease in Indonesia. In: APS Press, eds. Proceedings of the Third International Bacterial Wilt Symposium, South Africa, February 2002, USA: American Phytopathological Society Press, in press.

Thwaites R, Eden-Green SJ, Black R (2000) Diseases caused by bacteria. In *Diseases of Banana, Abacá and Enset*, D.R. Jones (ed.), pp. 213-239. Wallingford, UK: CABI Publishing.

http://www.actahort.org/members/showpdf?session=27431

http://www.cabicompendium.org/cpc/map.asp?CCODE=RALSSP

http://www.padil.gov.au/img.aspx?id=3871&s=s

http://www.actahort.org/members/showpdf?session=27431

http://www.cabicompendium.org/cpc/datasheet.asp?CCODE=RALSSP&COUNTRY=0

http://www.daff.gov.au/ba/ira/final-plant/banana-philippines

THREAT DATA

Panama disease

(*Fusarium oxysporum* f.sp. *cubense*)

Source: http://www.the-scientist.com/news/display/54710/

CRC10162

Com inty in n

CRC PLANTbiosecurity

Panama disease

(*Fusarium oxysporum f. sp. cubense*)

Panama disease of banana, also known as *Fusarium* wilt, is a fungal disease caused by *Fusarium oxysporum* f. sp. *cubense* (Foc). The disease first became epidemic in Panama in 1890 and devastated banana industries in Central America and Caribbean regions in the 1950s and 1960s. The fungus (Foc) invades the plant root system and blocks the water transport system, causing the plant to wilt. Foc is a soil-borne fungus and can remain in soil for more than 30 years. Once a plantation is infested, there is no remedy except growing resistant varieties. Foc is spread by movement of infected plants and field soil. Based on host susceptibility, there are 4 races of Foc (e.g. race 1, race 2, race 3 and race 4) affecting cultivars responsible for more than 80% of the world's banana production. Race 4 is the most aggressive race with the widest host range, and is considered the largest threat to Australian banana production as Cavendish, the basis of the industry, is very susceptible.

Host Range: *Fusarium oxysporum* f. sp. *cubense* infects banana (genus *Musa*) and relatives from the genus *Heliconia* (Jones 2000). Banana cultivars vary in their susceptibility to the four characterised races of the pathogen.

Distribution: The first record of Fusarium wilt was in Australia in 1874 (Bancroft 1876) and the disease has since been found in all banana-growing regions of the world, except for some of the countries bordering the Mediterranean Sea (Fig. 1).

Cavendish cultivars remain the banana varieties of international trade. However, these cultivars are not resistant to all strains of *Foc*. The 'subtropical race 4' strain of *Foc* causes losses of Cavendish cultivars in the subtropical regions of the Canary Islands, South Africa, Australia and Taiwan (Stover 1990). More importantly, in the tropical commercial and subsistence production regions of the Philippines, Indonesia, Taiwan, Malaysia, and in the southern provinces of China, a new strain of *Foc* designated 'tropical race 4' has caused widespread devastation (INIBAP 2006). Alarmingly, the disease is continuing to spread in these areas. Several incursions of this pathogen have also been recorded in Australia (Northern Territory); however these outbreaks have all been contained and have not reached the commercial growing regions situated on the east-coast of the country (Walduck 2002).

Figure 1. World map showing the global distribution of Fusarium wilt of banana. Diagram courtesy of the CRCTPP (Sue McKell).

Fusarium wilt in Australia. The history and distribution of Fusarium wilt in Australia has been well documented (Gerlach *et al.* 2000; Moore *et al.* 2001; Pegg *et al.* 1996). All four races of *Foc* are present in Australia. Races 1 & 2 of *Foc* have been found affecting banana in both northern and southern Queensland, and northern New South Wales (and race 1 has been found in Western Australia). The subtropical Australian banana industry is severely constrained by race 1 of *Foc*, which is predominantly based on the production of the highly susceptible Lady finger variety. Race 3, a pathogen of *Heliconia* spp., has been found in the Northern Territory (Gerlach *et al.* 2000). Subtropical race 4 is found affecting Cavendish cultivars, as well as race 1 and race 2 susceptible cultivars in northern New South Wales and southern Queensland. Isolated outbreaks of tropical race 4 have occurred in the Northern Territory; the first outbreak occurred on a commercial plantation in Darwin in 1997, and the disease has since spread to other commercial plantations in the area (Walduck 2002). Strict quarantine measures are in place to limit the spread of tropical race 4 to other banana producing areas, particularly to the major Cavendish production areas of north Queensland. Tropical race 4 of *Foc* is defined as a high priority pathogen and is targeted in the surveillance programs of the Northern Australia Quarantine Strategy (NAQS) and Northwatch, and the Biosecurity Workgroup of the Queensland Department of Employment, Economic Development and Innovation (DEEDI). The tropical race 4 strain of Fusarium wilt is regarded as one of the most serious threats to banana production in Australia as there are no disease resistant replacements available for Cavendish (Gerlach *et al.* 2000).

Biology and Ecology: The disease cycle of *Foc* begins with the entry of the pathogen into a banana root tip. Substances produced by the banana root tip stimulate germination of Foc resting spores, the chlamydospores, in the soil. Hyphae (fungal threads) grow from the chlamydospores and infect the lateral roots, progressing to invade the xylem vessels. Most initial infections are usually stopped in the xylem by the vascular occluding responses of the host, which include the formation of gels, tyloses and the collapse of vessels, but in susceptible cultivars, some of these infections become established in the xylem and advance ahead of these defence mechanisms. Tiny spores called microconidia are formed in the xylem vessels and spread through the vascular system of the plant, streaming to new sites where they germinate. Hyphae grow and infect cells at this new site, thus repeating the cycle. In

resistant varieties, the initial pathogen-induced occlusion reaction is further enhanced by the production of phenolic compounds, which lignifies these obstructions and limits the pathogen to the infected vessels; no further colonisation of the xylem vessels occurs (Beckman 1969; Beckman 1987; Beckman 1990; Beckman and Keller 1977; Beckman and Talboys 1981; VanderMolen *et al.* 1977).

Symptoms: Fusarium wilt is a typical vascular disease causing disruption of water translocation, systemic foliar symptoms and plant collapse (Jeger *et al.* 1995). Internal symptoms are characterised by reddish-brown discolouration of the vascular tissue. External symptoms are characterised by a yellowing of the leaf margins of older leaves, the collapse of leaves at the petiole and the splitting of the pseudostem base (Figure. 2). Disease progression results in the collapse of the crown and pseudostem, and ultimately plant death (Stover 1962).

Banana suckers that are less than four months old do not develop visible symptoms of Fusarium wilt. The lack of visible symptoms on suckers has assisted in the movement of the pathogen to new regions through the movement of these asymptomatic suckers to new areas as planting material. The fruit of the banana plant does not show any specific disease symptoms.

Figure 2. Disease symptoms of tropical race 4 affecting Cavendish clones at the Coastal Plains Banana Quarantine Station, Northern Territory: (a) Banana plant showing typical symptoms of Fusarium wilt, yellowing, necrosis and collapse of leaves (notice that leaves forms a skirt around the based of the plant). (b) Cross section of pseudostem showing the dramatic vascular discolouration. Photographs courtesy of the CRCTPP (Dr Juliane Henderson).

Affected plant stages: Vegetative growing stage

Affected plant parts: Roots, leaves, stems and whole plant.

Affected Industries: Banana industry.

Cultivar susceptibility:

- Race 1 infects Lady finger, Sugar and Ducasse bananas but not Cavendish bananas.
- Race 2 infects Bluggoe and Blue Java bananas, but not other banana varieties.
- Race 3 infects only *Heliconia* species, not bananas.
- Race 4 infects nearly all varieties of bananas, including the main commercial Cavendish variety.

Disease Movement and Dispersal: *Foc* is most commonly spread by the movement of infected planting material, rhizomes and suckers (and the attached soil), to new uninfected areas. This infected planting material often does not exhibit symptoms of Fusarium wilt (is asymptomatic). The pathogen can also be effectively spread by the movement of soil, running water, and farm machinery and implements. Once a site is infected, the pathogen can persist in the soil as chlamydospores for more than 30 years (Stover 1962; Waite and Dunlap 1953). Also, it is likely that *Foc* can survive non-pathogenically on alternative hosts, such as weed species (Hennessey *et al.*2005).

Disease Impact: Fusarium wilt has had a particularly destructive history in the evolution of international banana trade. In the period 1890-1960, some 40,000 hectares of the susceptible banana cultivar Gros Michel (grown for export) were destroyed or abandoned in Central and South America and the Caribbean because of race 1 of *Foc*. In terms of crop destruction, Fusarium wilt then ranked alongside the foremost devastating plant diseases such as wheat rust and potato late blight (Carefoot and Sprott 1969). Export industries were forced to replace the susceptible Gros Michel variety with Cavendish cultivars, which remains resistant to race 1 of *Foc* (Stover 1990).

Tropical race 4 of *Foc* affects banana cultivars that comprise 80% of the world's banana production, including the important Cavendish and plantain subgroups (Ploetz 2005). The tropical race 4 strain of *Foc* could cause significant damage to the major world export production areas if introduced into Ecuador, Central America and Colombia, which are based on Cavendish cultivars. As it stands, the tropical race 4 strain poses a very real threat to the multi-billion dollar global banana trade, and the food security of millions of subsistence farmers (Ploetz 2005). Furthermore, the Cavendish variety may risk the very same fate as Gros Michel, the cultivar it replaced nearly 50 years ago to control race 1 of *Foc*.

In Australia, tropical race 4 of *Foc* poses an immediate threat to the commercial production areas in the Northern Territory, and to major production areas based along the east coast of Australia. Current disease management strategies are centred on: (a) the prevention of the movement of *Foc* into disease free areas (particularly the movement of tropical race 4), and (b) the early detection and containment of Fusarium wilt outbreaks. Rapid and accurate detection and diagnosis of the pathogen underpins the successful implementation of these management strategies.

Disease Management: There is no satisfactory method to control Panama disease caused by Foc TR 4. Chemical control, flood fallowing, crop rotation and the use of organic amendments have not been effective in managing the disease (Ploetz and Pegg, 1999). The only effective means of control is the use of resistant cultivars, but none are commercially available for Foc TR4.

Quarantine and exclusion procedures are effective in controlling the disease by restricting the movement of corms, suckers and soil that could be carrying the fungus from infested to clean areas. The use of micropropagated planting material should be encouraged as this, if managed correctly, should be free from contamination by the pathogen. Plants derived from tissue culture and planted in soil where bananas have not been previously grown should remain free of *Fusarium* wilt for a considerable period.

Quarantine Risk: Foc spores mainly disperse through surface run-off water, contaminated soil and machinery. Quarantine measures are in place in Australia to prevent spread of Foc TR4 from the infested regions to other areas.

Acknowledgements

The information was obtained from the PaDIL Plant Biosecurity Toolbox Fusarium Wilt pages, edited by Sharon Van Brunschot: http://old.padil.gov.au/pbt/

Contributors: André Drenth, Lisa Gulino, Juliane Henderson, Natalie Moore, Wayne O'Neill, Julie Pattemore, Ken Pegg, Suzy Perry (nee Bentley), Susan Porchun, Linda Smith, Sharon Van Brunschot, Kendle Wilkinson (nee Gerlach)

References

Bancroft J (1876) 'Report of the board appointed to enquire into the cause of disease affecting livestock and plants.' (Votes and Proceedings 1877: Queensland)

Beckman CH (1969) Plasticizing of walls and gel induction in banana root vessels infected with *Fusarium oxysporum*. *Phytopathology* **59**, 1477-1483.

Beckman CH (1987) 'The Nature of Wilt Diseases of Plants.' (APS Press: St Paul, Minnesota)

Beckman CH (1990) Host responses to the pathogen. In 'Fusarium Wilt of Banana'. (Ed. RC Ploetz) pp. 93-105. (APS Press: St Paul, Minnesota).

Beckman CH, Keller JL (1977) Vessels do end! *Phytopathology* 52, 954-956.

Beckman CH, Talboys PW (1981) Anatomy of resistance. In 'Fungal Wilt Diseases of Plants'. (Eds ME Mace, AA Bell and CH Beckman) pp. 487-521. (Academic Press: New York, USA)

Carefoot GL, Sprott ER (1969) 'Famine on the Wind.' (Angus and Robertson: London).

Gerlach KS, Bentley S, Moore NY, Pegg K, G., Aitken EAB (2000) Characterisation of Australian isolates of *Fusarium oxysporum* f. sp. *cubense* by DNA fingerprinting analysis. *Australian Journal of Agricultural Research* **51**, 945-953.

Hennessey C, Walduck G, Daly A, Padovan A (2005) Weed hosts of *Fusarium oxysporum* f.sp. *cubense* tropical race 4 in northern Australia. *Australasian Plant Pathology* **34**, 115-117.

INIBAP (2006) Panama disease: a renewed threat in Asia. In. (Bioversity International: Online http://bananas.bioversityinternational.org/content/view/87/74/lang,en/)

Jeger MJ, Eden-Green S, Thresh JM, Johanson A, Waller JM, Browne AE (1995) Banana diseases. In 'Bananas and Plantains'. (Ed. S Gowen) pp. 337-343. (Chapman and Hall: London).

Jones DR (2000) 'Diseases of Banana, Abaca and Enset.' (CAB International: Wallingford, Oxon, UK)

Moore NY, Pegg K, G., Buddenhagen IW, Bentley S (2001) Fusarium wilt of banana: a diverse clonal pathogen of a domesticated clonal host. In 'Fusarium'. (Eds BA Summerell, JF Leslie, D Backhouse, WL Bryden and LW Burgess). (The American Phytopathological Society: St. Paul)

Pegg K, G., Moore NY, Bentley S (1996) Fusarium wilt of banana in Australia: a review. *Australian Journal of Agricultural Research* **47**, 637-650.

Ploetz RC (2005) Panama disease, an old enemy rears its ugly head: Parts 1 &2. In. (Plant Health Progress, APSnet: Online doi:10.1094/PHP-2005-1221-01-RV).

Ploetz RC, Pegg K, G. (1999) Fusarium Wilt. In 'Diseases of Banana, Abaca and Enset.' (Ed. DR Jones) pp. 143-159. (CAB International Publishing: Wallingford).

Stover RH (1962) 'Fusarial Wilt (Panama disease) of bananas and other Musa species.' (Commonwealth Mycological Institute: Kew, Surrey, England).

Stover RH (1990) Fusarium wilt of banana: some history and current status of the disease. In 'Fusarium Wilt of Banana'. (Ed. RC Ploetz) pp. 1-7. (APS Press: St Paul, MN).

VanderMolen GE, Beckman CH, Rodehorst E (1977) Vascular gelation: a general response phenomenon following infection. *Physiological and Plant Pathology* **11**, 95-110.
Waite BH, Dunlap VC (1953) Preliminary host range studies with *Fusarium oxysporum* f.sp. *cubense*. US Department of Agriculture Disease Reporter 37, 79-80.
Walduck G (2002) New varieties and quarantine help solve Panama riddle. *Australian Bananas* **15**, 16-17.
http://old.padil.gov.au/pbt

THREAT DATA

Yellow Sigatoka

(*Mycosphaerella musicola*)

Source: http://visualsunlimited.photoshelter.com/image/I0000InyfgZJUB5Y

CRC10162

Communicating Uncertainty in Biosecurity Adaption

CRC PLANT biosecurity

Yellow Sigatoka

(*Mycosphaerella musicola*)

Yellow Sigatoka is caused by the fungal pathogen *Mycosphaerella musicola* which is closely related to *M. fijiensis,* the cause of black Sigatoka. Yellow Sigatoka is one of the most serious leaf diseases of banana, although it is less damaging than black Sigatoka. Average yield losses by yellow Sigatoka are 25 - 30%, and control measures account for about 14% of total banana production costs. High rainfall and humidity suitable for banana cultivation favour disease development and spread. Yellow Sigatoka is present in Australia.

Host Range: Currently the only known hosts of *M. fijiensis* and *M. musicola* are *Musa* spp. These species and subspecies all vary in their levels of resistance to *M. fijiensis* and *M. musicola.* There is one report in the literature of *M. musicola* having been isolated from leaf spots on a *Heliconia* species in Venezuela (Madiz *et al.* 1991).

Distribution: Yellow Sigatoka was first reported in Java in 1902 and later in the Sigatoka valley in Fiji. The pathogen is considered to have a worldwide distribution (Figure 1). *M. musicola* also causes serious yield losses in regions not affected by black Sigatoka. As this pathogen can proliferate at lower temperatures and lower relative humidity, *M. musicola* is more widespread than *M.fijiensis*. Yellow Sigatoka is often the dominant disease at higher altitudes (>1 200 metres) although it appears that *M. fijiensis* is becoming more adapted to higher altitudes and is gradually replacing *M. musicola* in these regions (Carlier *et al.* 2000a). It has not however been reported in the banana growing regions of the Canary Islands, Egypt and Israel (Jones 2000) and its exact distribution through Asia is still unclear.

In Australia, *M. musicola* is endemic throughout all banana growing regions in Queensland and northern New South Wales, the Kimberleys of Western Australia, and it has also been detected in banana growing regions in the Northern Territory.

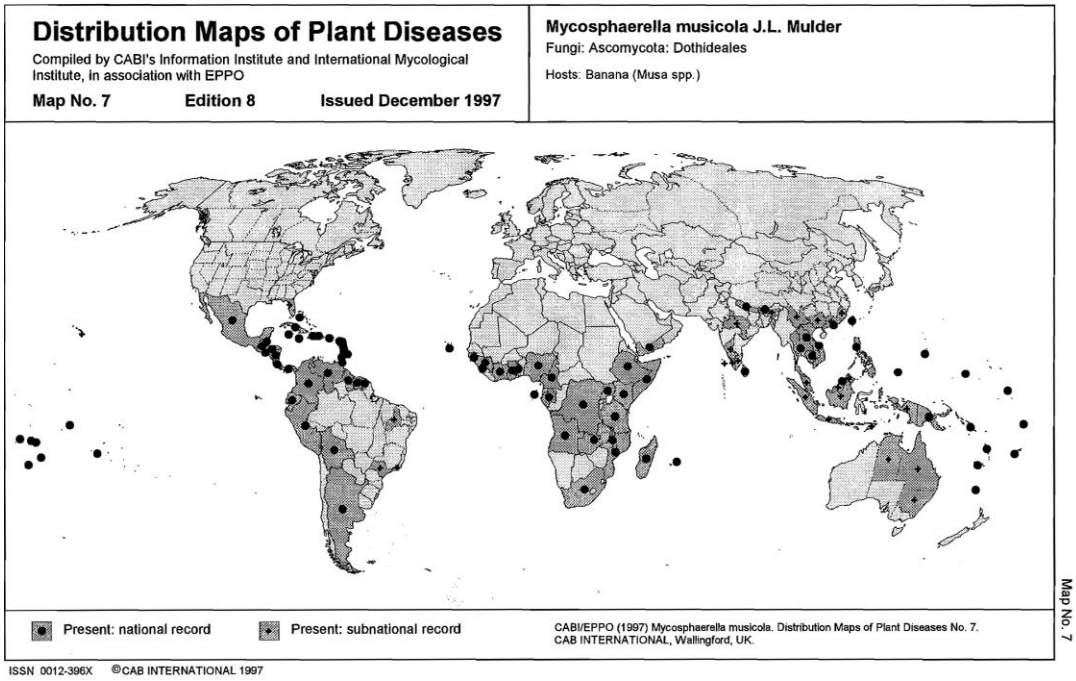

Figure 1. Distribution map for *Mycosphaerella musicola*, causal agent of yellow sigatoka.

Biology and Ecology: Most infections of *M. musicola* begin with spores being deposited on the susceptible cigar leaf of the banana plant. The fungus produces two types of spores: asexual spores called conidia and sexual spores called ascospores. Spores will germinate within 2-3 hours of being deposited on the leaf surface if there is a water film present or if the humidity is very high. The optimal temperature for germination of *M. musicola* conidia is between 25-29ºC (cf. 27ºC for *M. fijiensis)* and for ascospores it is between 25-26ºC. The germ tube then grows over the leaf surface for 4-6 days before penetrating the leaf via stomata (Meredith 1970; Stover 1980). A distinctive line spotting pattern of infection is produced when the source of inoculum is conidia dislodged by rain splashes. These run down the inside of the cigar leaf cylinder contacting the lower point of the cylinder resulting in a line of infection. The deposition of ascospores by wind currents is generally on the terminal end of these leaves resulting in a distinctive leaf tip infection (Meredith 1970; Stover 1972).

The disease cycle for both *M. fijiensis* and *M. musicola* is similar with only minor differences. As *M. fijiensis* produces considerably less conidia and for a shorter period of time than *M. musicola,* ascospores are the main dispersal agent for this pathogen (Stover 1980). Both conidia and ascospores are important for dispersal of *M. musicola* (Stover 1971). For both pathogens, ascospores are involved in the movement of the pathogen over longer distances rather than conidia. Overall the disease cycle is much slower for *M. musicola* than for *M. fijiensis* due to longer time required to complete the life cycle. Inoculation studies conducted in Honduras demonstrated that spotting associated with *M. fijiensis* infections appeared 8-10 days faster than that associated with *M. musicola* infections. Ascospore maturation time is also longer at 4 weeks for *M. musicola* compared with 2 weeks for *M.fijiensis* (Stover 1980). Generally, it has been observed, the optimal conditions for *M. fijiensis* are those where there is, on average, higher temperatures and higher relative humidity.

Both *M. musicola* and *M. fijiensis* are dispersed within banana blocks by rain splash of conidia. Movement between blocks is possible through the aerial spread of ascospores ejected from fruiting bodies produced in the diseased tissues. Due to the larger amount of

conidia produced by *M. musicola* than by *M. fijiensis,* conidia are considered the main means of spread for *M. musicola* while ascospores are the main method of dispersal of *M. fijiensis* (Stover and Dickson 1976).

Symptoms:

Figure 2: (a) Banana plant infected with *Mycosphaerella musicola*. Note later stage lesions are always present in the lower leaves which are older while the newer leaves show the earlier stage symptoms. Symptom development can be used in conjunction with other tools to assist with diagnosis. The photographs in **(b)** and **(c)** show advanced lesions on leaves. (Images sourced from Plant Biosecurity Toolbox, PaDIL)

Yellow Sigatoka disease is similar to black Sigatoka, with five recognised stages. There are, however, some distinguishing diagnostic features for yellow Sigatoka. The disease ultimately has the same effect on yields as black Sigatoka, although yellow Sigatoka disease development is slower, enabling it to be controlled through deleafing and the use of fungicides.

Yellow Sigatoka can be differentiated from Black Sigatoka at the early stages of lesion development (Stages 1 and 2) on visual symptoms. There have been several descriptions of the development of individual lesions of Sigatoka disease over the years which are well summarised in Meredith (1970). Brun's description (Brun 1958) of five stages follows:

Figure 3(a): Stage 1 lesions of yellow Sigatoka characterised by the light green dots and dashes which are about 1 mm in length. (Image sourced from Plant Biosecurity Toolbox, PaDIL)

Stage 1: This stage is characterised by the appearance of very small light green dots or dashes of approximately 1 mm in length. (Figure 3(a)).

Figure 3(b): Lesions associated with Stage 2a (early) of yellow Sigatoka. Note light green streaks which are the characteristic lesions at this stage. (Image sourced from Plant Biosecurity Toolbox, PaDIL)

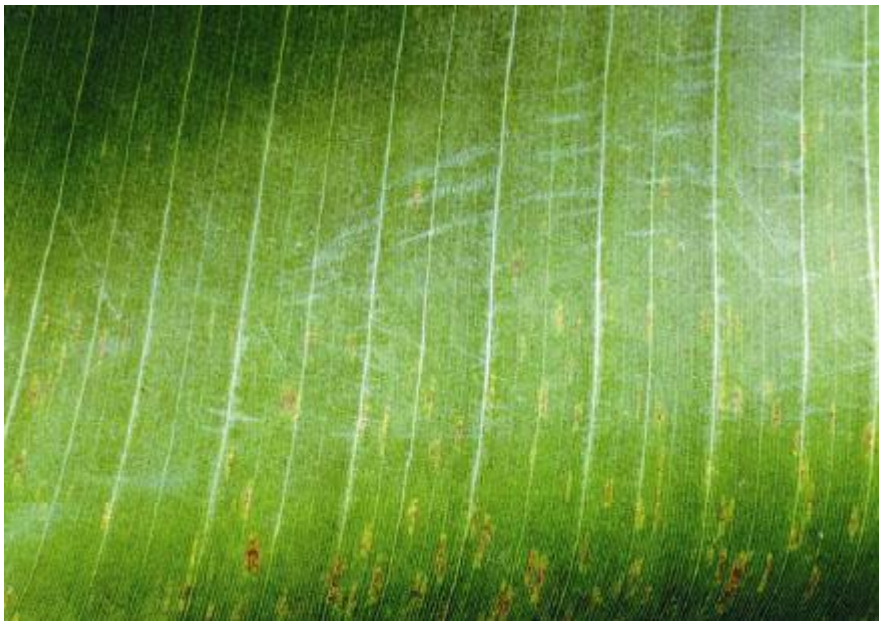

Figure 3(c): Lesions associated with Stage 2b (late) of yellow Sigatoka. Note the change in colour of the streaks from light green to rusty brown. (Image sourced from Plant Biosecurity Toolbox, PaDIL)

Stage 2: The small dot or dash of Stage 1 elongates into a light green streak several millimetres long. (Figure 3(b)&(c))

Figure 3(d): Stage 3 symptoms associated with yellow Sigatoka. Note that the streaks from stage 2 have now elongated and widened. (Image sourced from Plant Biosecurity Toolbox, PaDIL)

Stage 3: At this stage there is a change in the colour of the streak to a rusty brown. The streak becomes elongated and widens slightly. The border of the streak is ill defined. (Figure 3(d)).

Stage 4: The streak becomes more elliptical and is a definite spot with a sunken dark brown centre. It is often surrounded by a yellow halo. At this stage the conidia are produced. (Figure 3(e))

Stage 5: The final stage has a grey dried out centre and an obvious black margin. This black margin can still be seen even after the leaf has dried out. (Figure 3(f))

The stages of both yellow and black Sigatoka are summarised in Table 1.

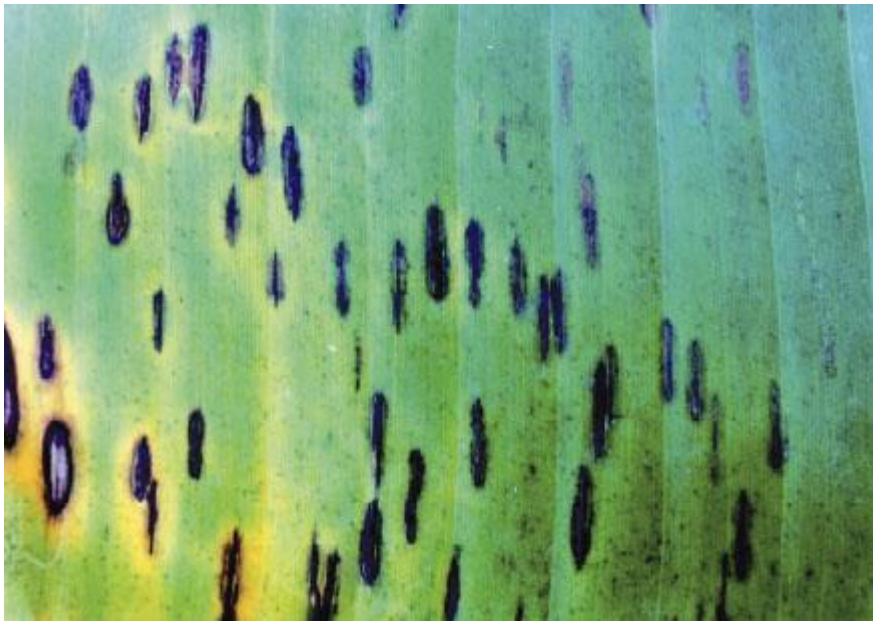

Figure 3(e): Stage 4 symptoms associated with yellow Sigatoka. Note that the Stage 3 streaks have now become spots. Conidia may be present from this stage. (Image sourced from Plant Biosecurity Toolbox, PaDIL)

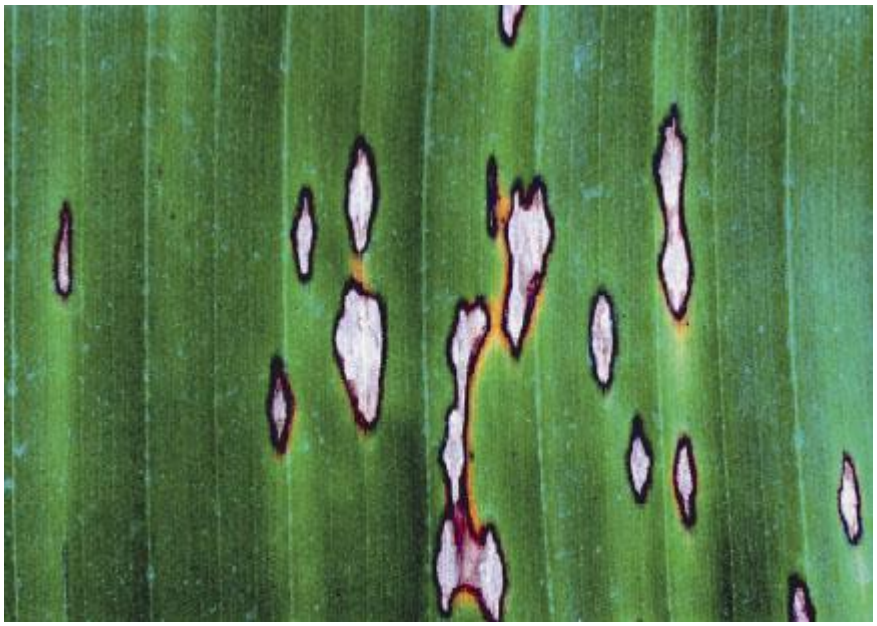

Figure 3(f): Stage 5 symptoms associated with yellow Sigatoka. The final stage has a grey dried out centre and an obvious black margin. This black margin can still be seen even after the leaf has dried out. (Image sourced from Plant Biosecurity Toolbox, PaDIL)

Table 1. Summary of the different lesion stages associated with yellow and black Sigatoka leaf spot of bananas.

Lesion Stage	**Yellow Sigatoka**	**Black Sigatoka**
Stage 1	Very small light green dot or dash up to 1 mm long	Small pigmented spot of white or yellow, similar to yellow Sigatoka stage 1
Stage 2	Light green streak several millimetres long	Brown streak, visible on underside of leaf, later visible on leaf upper surface as yellow streak; colour changes progressively to brown, then black on upper leaf surface
Stage 3	An elongated rusty brown spot with an poorly defined border	Enlarged stage 2, streaks become longer
Stage 4	A mature spot with a dark brown sunken centre; often surrounded by a yellow halo, conidiophores and conidia are produced at this stage	Appears on leaf underside as brown spot, as a black spot on upper leaf surface

Stage 5	Spot has developed a grey, dried out centre and a peripheral black ring which is evident even after the leaf has dried out	Elliptical spot is totally black on the underside of the leaf, surrounded by a yellow halo
Stage 6		Centre of spot dries out, turns grey and is surrounded by a well-defined margin and a bright yellow halo

Affected plant stages: All growing stages affected.

Affected plant parts: Leaves.

Affected Industries: Banana industry.

Disease Movement and Dispersal: Stover (1962) hypothesised the mode of spread of *M. musicola* worldwide. Working from disease records, Stover proposed that *M. musicola* was moved from Java, where it was first described in 1902, to Fiji on banana leaf material used as packing material in shipping containers. *M. musicola* was first identified in Fiji in 1913 (Massee 1914). From here, Stover proposed that the pathogen moved to the east coast of Australia on the prevailing winds in around 1924. At this time there was a disease epidemic in the Fijian banana plantations of the Sigatoka Valley which was causing inoculum levels to be exceptionally high. Once in Australia the disease quickly spread throughout banana plantations, many of which had been left unmanaged due to the severe banana bunchy top disease (BBTD) epidemic.

In Australia, *M. musicola* was found to have spread to the banana growing regions of New South Wales by 1927 (Simmonds 1928). *M. musicola* is now endemic throughout all banana growing regions in Queensland and northern New South Wales. In Western Australia it was first detected in Kununurra in 1990 although it is thought to have been present for some time before this first report (Shivas and Kesavan 1992). It is now identified as a common pathogen to the banana growing regions of the Kimberleys. The pathogen has also been detected in banana growing regions in the Northern Territory.

M. musicola is dispersed within banana blocks by rain splash of conidia. Movement between blocks is possible through the aerial spread of ascospores ejected from the fruiting bodies (Stover and Dickson 1976). In the case of international movement, infected planting material and leaves (often used as packing materials in developing world) are responsible for the disease spread. Long distance movement of banana leaves and planting materials pose a quarantine risk.

Disease Impact: Cavendish is the most important commercial banana cultivar and all Cavendish cultivars (AAA genomic group) are susceptible to yellow Sigatoka. If left uncontrolled, the disease can become very destructive in the subtropics. The disease is controlled with mixtures of fungicides and mineral oil, combined with hygiene measures.

Acknowledgements: The information was obtained from the PaDIL Plant Biosecurity Toolbox Black Sigatoka pages, edited by Juliane Henderson: http://www.padil.gov.au/pbt. Contributors: Sharon Van Brunschot, André Drenth, Kathy Grice, Juliane Henderson, Julie Pattemore, Ron Peterson, Susan Porchun.

References

Brun J (1958) Etude sur l'action des fongicides huileux dans la luttre contre la cercoporiose. Note prélinimaire. Fruits D'outre Mer 13, 3-14.

Carlier J, Fouré E, Gauhl F, Jones DR, Lepoivre P, Mourichon X, Pasberg-Gauhl C, Romero RA (2000a) Black Leaf Streak. In 'Diseases of Banana, Abaca and Enset.' (Ed. DR Jones) pp. 37-79. (CABI Publishing: Wallingford)

D.R. Jones (ed) (2000) Diseases of Banana, Abacá and Enset. CABI Publishing, New York.

Madiz RÇ, Smits B. G, Noguera R (1991) Principales hongos patogenos que afectan alguns especies ornamentales del genero Heliconia. Agronomia Tropical 41, 265-274.

Massee G (1914) Fungi Exotica:XVIII. Bulletin of Miscellaneous Information, Kew Gardens 4, 159.

Meredith DS (1970) 'Banana leaf spot disease(Sigatoka) caused by *Mycosphaerella musicola* Leach.' (Commonwealth Mycological Institute, Kew, Surrey, England)

Shivas R, Kesavan V (1992) Yellow Sigatoka of Bananas. Farm Notes, Department of Agriculture Western Australia 49.

Simmonds NW (1928) Diseases of banana in Queensland. Queensland Agricultural Journal 30, 438-454.

Stover RH (1962) Intercontinental spread of banana leaf spot (*Mycosphaerella musicola*). Tropical Agriculture (Trinidad) 29, 327-338.

Stover RH (1971) Leaf spot of bananas caused by *Mycosphaerella musicola*: Role of conidia in epidemiology. Phytopathology 60, 856-860.

Stover RH (1972) 'Banana, Plantain and Abacá Diseases.' (Commonwealth Mycological Institute (CAB): Kew, England)

Stover RH. (1980). Sigatoka leaf spot diseases of bananas and plantains. Plant Disease 64:750-756.

Stover RH, Dickson JD (1976) Banana leaf spot caused by *Mycosphaerella musicola* and *M. fijiensis* var.*deformis*: a comparison of the first Central American epidemics. FAO Plant Protection Bulletin 24, 36-42.

http://www.padil.gov.au/pbt

http://www.apsnet.org/online/feature/banana/Top.html

http://visualsunlimited.photoshelter.com/image/I0000InyfgZJUB5Y

http://www.cabicompendium.org/cpc/report_select.asp?CCODE=MYCOMU

http://www.oisat.org/pests/diseases/fungal/sigatoka_leaf_spot.html

http://www2.dpi.qld.gov.au/horticulture/5247.html

http://bananas.bioversityinternational.org/files/files/pdf/publications/disease8_en.pdf

http://www.apsnet.org/education/Lessonsplantpath/BlackSigatoka/symptom.htm

THREAT DATA

Fire blight

(*Erwinia amylovora*)

Source: http://extension.missouri.edu/publications/DisplayPub.aspx?P=G6020

CRC10162

Communicating Uncertainty in Biosecurity Adaption

CRC PLANT biosecurity

Fire blight

(*Erwinia amylovora*)

Fire blight is a bacterial disease caused by *Erwinia amylovora* affecting many species within the Rosaceae family. Among these, apple and pear are the most affected and economically important horticultural plants. The disease was first recorded in the USA in 1794 and since then has spread all around the world. Fire blight kills blossoms, shoots, limbs and entire trees leading to losses both in fruit production and orchard productivity. Australia is currently free from *E. amylovora.*

Distribution: Europe (Albania, Austria, Belgium, Bosnia and Herzegovina, Bulgaria, Croatia, Cyprus, Czech Republic, Czechoslovakia [former -] Denmark, France, Germany, Greece, Hungary, Ireland, Italy, Luxembourg, Macedonia, Moldova, Netherlands, Norway, Poland, Romania, Spain, Sweden, Switzerland, United Kingdom, Yugoslavia), Asia (Armenia, Iran, Israel, Jordan, Lebanon, Turkey), Egypt, Bermuda, Canada, Guatemala, Mexico, USA, New Zealand (Figure 1).

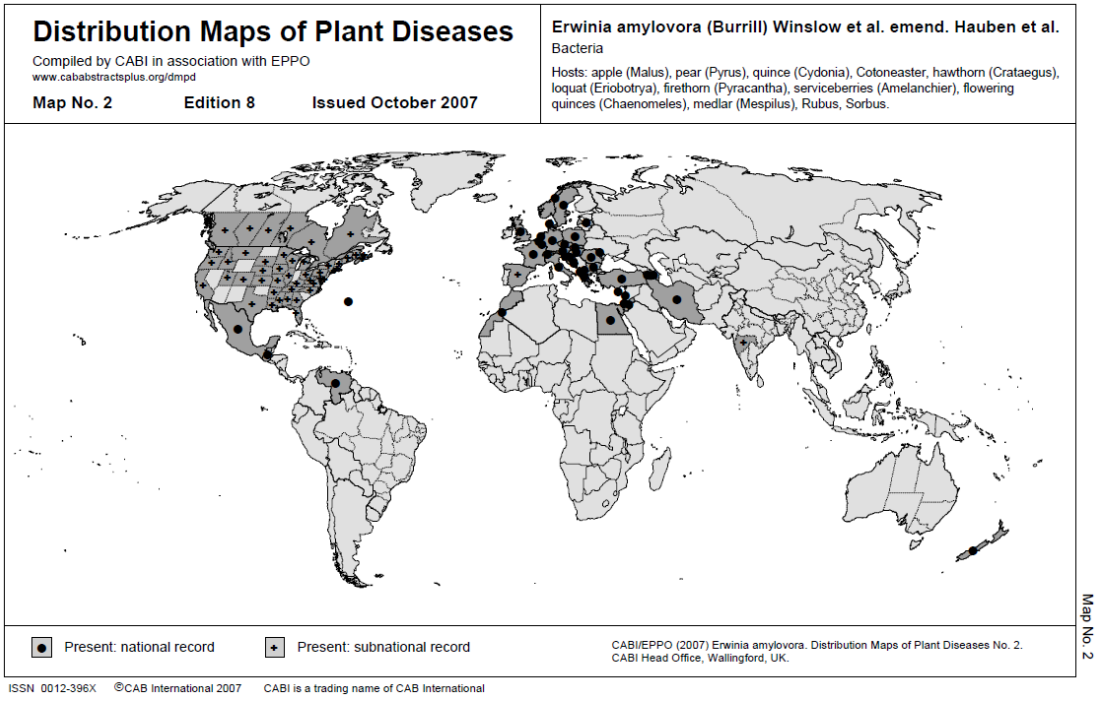

Figure 1. Worlwide distribution map of *Erwinia amylovora*, 2007.

Fire blight was first recorded in 1794 on apples in New York and has since spread to most apple growing areas of the world. In 1997 there was a report of fire blight in the Melbourne Royal Botanic Gardens. This detection of the pathogen was successfully eradicated and Australia is considered free of the disease.

Host range: *Malus* (ornamental species apple), *Pyrus* (pears), *Crataegus* (hawthorns), *Cotoneaster*, *Cydonia*, *Eriobotrya*, *Pyracantha* (Firethorn), *Amelanchier* (serviceberries), *Mespilus*, *Chaenomeles* (flowering quinces), *Rubus* (blackberry, raspberry), *Sorbus*.

Hosts of economic and epidemiological significance are *Cotoneaster* spp. (cotoneaster), *Crataegus* spp. (hawthorn), *Cydonia* spp. (quince), *Eriobotrya japonica* (loquat), *Malus* spp. (apple), *Pyracantha* spp. (firethorn) and *Pyrus* spp. (pear).

Biology and Ecology: Figure 2 illustrates the key steps in the disease cycle of fire blight caused by *E. amylovora*. This bacterial pathogen overwinters almost exclusively along the margins of living bark tissues of overwintering cankers formed during the previous season in spurs, twigs and branches. The overwintering cankers with ill-defined margins are likely to provide inoculum in the spring as trees come out of dormancy. Under warm and humid conditions some of these cankers become active and exude bacteria-laden ooze that acts as the primary inoculum. Overwintering cankers are clearly visible on stems and large limbs but cankers on twigs and smaller limbs are not easily distinguished. The smaller cankers, especially those around cuts made in the previous season to remove blighted limbs, are also important sources of inoculum. Bacteria may also move into the orchard from neighbouring infection sites, including ornamental and wild hosts.

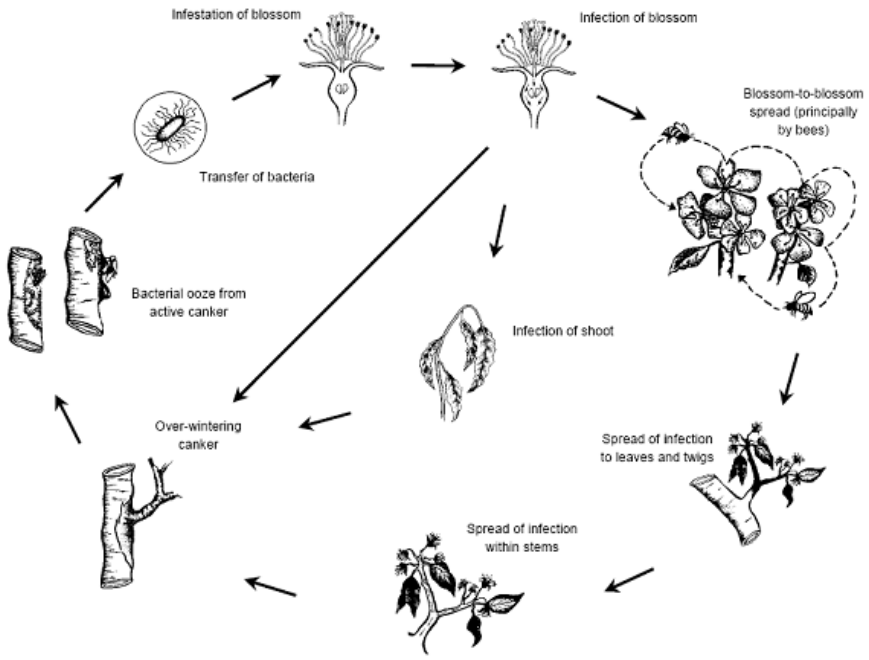

Figure 2. Simplified disease cycle of fire blight. Diagram sourced from the Revised Draft IRA Report for Importation of Apples from New Zealand (2004), Australian Government Department of Agriculture, Fisheries and Forestry.

The bacteria are spread by insects, rain, wind or wind-driven rain (as aerosols) to open blossoms, succulent shoot tips and tender leaves, where infection may occur. Open blossoms are the most susceptible tissues on the apple tree. Bacteria deposited on the

stigma surface of blossoms multiply to very high numbers but usually do not cause disease; it is then in an epiphytic phase. The bacteria are spread from blossom to blossom by pollinating insects (mainly bees). Rain and dew wash the bacteria into the base of the flowers where they gain entry through natural openings and wounds, resulting in infection under conditions of warm temperatures and high humidity. Infected blossoms eventually die. The spurs bearing the dead blossom clusters are retained on the tree and persist into the winter. Under suitable conditions, bacteria multiply rapidly and move through succulent host tissues. Ideal conditions for infection, disease development and spread of the pathogen are wet or humid weather with daytime temperatures in the range of 18 - 30^{o}C and night temperatures above 15°C.

In order to continue the disease cycle, *E. amylovora* must survive the winter on dormant host plants. *E. amylovora* survives almost exclusively in living bark tissues along the margins of overwintering (hold-over) cankers on hosts that have been infected in previous growing seasons (Brooks, 1926; Miller, 1929; Rosen, 1933; Parker, 1936; Schroth *et al.*, 1974; Eden-Green and Billing, 1974; Beer and Norelli, 1977). Usually only a small proportion of cankers formed in the current season become active overwintering cankers and produce visible ooze the following year (Brooks, 1926; Miller, 1929; van der Zwet, 1969). Estimates of the proportion of active overwintering cankers are reported to vary from 2-46% (Miller, 1929; Tullis, 1929) to 2-11% (Brooks, 1926; Rosen, 1929; Pierstorff, 1931; Goodman, 1954; van der Zwet, 1969).

Symptoms: The name fire blight aptly describes the characteristic scorched appearance of leaves and stem ends. Badly infected trees look as if they have been hit by a blowtorch (Figure 3).

Figure 3. Foliar symptoms of fire blight on apple. © Department of Primary Industries, Victoria, Australia.

The first signs of fire blight are blackened blossoms or fruit clusters and contorted branch tips, which are bent over like a 'shepherd's crook'. Infected blossoms and new shoots die and discolour suddenly, turning grey-green, brown or black (Figure 4).

Figure 4. Shoot tip blight on apple. © Department of Primary Industries, Victoria, Australia.

Cankers develop on branches and twigs following invasion of the tissues. Initially these cankers are reddish, but progressively they become brown and then black (Figure 5).

Figure 5. Stem canker on apples. © Department of Primary Industries, Victoria, Australia.

A characteristic sign of the pathogen is ooze or watery exudate that appears from infected plant parts, especially under humid conditions (Figure 6).

Figure 6. Bacterial ooze on apple shoot due to fire blight infection. © Department of Primary Industries, Victoria, Australia.

Early infected fruits remain very small and appear shriveled and dark but are firmly attached to the cluster base. Those infected as a consequence of progressive infection of branches are less shrivelled and discoloured. Those infected following injury by hail or insects often develop red, brown, or black lesions. Infected fruits may also exude ooze that appears clear or milky turning red to brown with time, and shiny and glassy when dry.

Affected plant stages: All

Affected plant parts: Leaves, stems, flowers and fruit (CABI, 2003)

Affected Industries: Apple and Pear, honey.

Disease movement and Dispersal: Bacteria may be spread by visiting insects, rain, wind and pruning tools. Long-range spread of the pathogen occurs by infected or infested nursery or propagative material.

Disease Impact: The *Erwinia amylovora* incursion in the Royal Botanic Gardens, Melbourne (RBGM) in autumn 1997 cost the Australian pome fruit and nursery industries an estimated A$20 million in lost revenue. The cost of the national orchard and urban surveys, eradication programs, diagnostics, and media management was estimated at A$2.2 million and involved some 250 people. As a result of the E. amylovora incursion in the RBGM, an imposition of interstate trade on the movement of host plants and related produce was enforced. These restrictions cost the Victorian pome fruit and nursery industries around A$7 million in lost sales and depressed prices. International trade was also suspended in some instances and the Tasmanian industry application for access to the Japanese apple market was delayed for two years and cost an estimated A$10 million in lost sales. The economic impact of a fire blight outbreak in Australia's largest pome fruit growing district (Goulburn Valley, Victoria) was calculated using a dynamic multi-regional computable general equilibrium program of Australia called TERM (The Enormous Regional Model). Two separate scenarios were considered. In the first scenario an outbreak with 30% yield losses is eradicated in five years and results in losses of A$260 million. In the second scenario an outbreak is not eradicated and pome fruit output in the Goulburn Valley declines by 50% for pears and 20% for apples and results in losses of A$870 million in net present value (Rodoni *et al.* 2006).

In addition to the impacts on the Pome Industry, an outbreak of Fire blight would also impact on commercial honey production. Honeybees are considered to be an important insect vector for the disease and outbreaks could result in quarantining of hives that are located in the vicinity of an outbreak (see BeeGuard plan).

Entry potential: Rating = High. Pathogen was previously detected in Australia in an area isolated from commercial orchards and was eradicated.

Establishment potential: Rating = High. Conducive to climatic conditions.

Spread potential following establishment: Rating = High. May be confused with symptoms of other pathogens. Can exist and not show symptoms.

Economic Impact: Rating = High. Can be economically devastating (CABI, 2002).

Overall risk: Rating = High

Acknowledgements. The information for this sheet was sourced from Plant Health Australia's Plant Biosecurity Toolbox and the National Apple and Pear Industry Biosecurity Plan Appendix 2: Pest Risk Review (authored by Clare Duncan, 2004).

References

Beer, S.V. and Norelli, J.L. (1977) Fire blight epidemiology: factors affecting release of *Erwinia amylovora* by cankers. *Phytopathology.* **67** : 1119-1125 pp.

Brooks, A.N. (1926) Studies of the epidemiology and control of fire blight of apple. *Phytopthology.* **16** : 665-696 pp.

Eden-Green, S.J. and Billing, E. (1974) Fire blight. *Review of Plant Pathology.* **53** : 353-365 pp.

Goodman, R.N. (1954) Apple fruits a source of overwintering fire blight inoculum. *Plant Disease Reporter.* **38** : 414 pp.

http://www.cabicompendium.org/cpc/datasheet.asp?CCODE=ERWIAM&COUNTRY=0

http://www.planthealthaustralia.com.au/pidd-docs/090%20-%20Fire%20blight%20PRR%20-%202005.pdf

Miller, P.W. (1929) Studies of fire blight of apple in Wisconsin. *Journal of Agriculture Research.* **39** : 579 621 pp.

Parker, K.G. (1936) Fire blight: overwintering, dissemination, and control of the pathogen. *New York College of Agricultural Experiment Station Memoirs.* **193** : 1-42 pp.

Pierstorff, A.L. (1931) Studies on the fire blight organism, *Bacillus amylovorus*.

Revised Draft IRA Report for Importation of Apples from New Zealand (2004)

Rodoni, B. C.; Merriman, P. R.; McKirdy, S. J.; Wittwer, G. (2006) Costs associated with fire blight incursion management and predicted costs of future incursions. Proceedings of the Xth International Workshop on Fire Blight, Bologna, Italy, 5-9 July 2004. Acta Horticulturae. 704, 55-61.

Rosen, H.R. (1929) The life history of the fire blight pathogen, Bacillus amylovorus, as related to the means of overwintering dissemination. *Arkansas Agricultural Experiment Station Bulletin.* **244** : 96 pp.

Rosen, H.R. (1933) Further Studies on the overwintering and dissemination of the fire blight pathogen. *Arkansas Agricultural Experiment Station Bulletin.* **283**

Schroth, M.N., Thomson, S.V., Hildebrand, D.C. and Moller, W.J. (1974) Epidemiology and control of fire blight. *Annual Review of Phytopathology.* **12** : 389-412 pp.

Tullis, E.C. (1929) Studies on the overwintering and modes of infection of the fire blight organism. *Michigan Agriculture Experiment Station Technical Bulletin.* **97** : 1-32 pp.

van der Zwet, T. (1969) Study of fire blight cankers and associated bacteria in pear. *Phytopathology.* **59**: 607-613 pp.

THREAT DATA

European canker

(*Neonectria ditissima*)

Photo: Jacqueline Edwards

CRC10162

Communicating Uncertainty in Biosecurity Adaption

CRC PLANT biosecurity

European canker

(*Neonectria ditissima*)

Neonectria ditissima (previously known as *Neonectria galligena* and *Nectria galligena*) is a fungal pathogen of pome fruit and many species of hardwood forest trees worldwide. It causes economically important diseases such as European canker on apple and beech bark canker on beech. Cankers develop on the woody tissues, girdling and killing branches and, occasionally, the whole tree. The pathogen was present in apple orchards in Spreyton, Tasmania, from about 1954 but was eradicated by 1991. Australia is currently free of the disease.

European canker is present in almost all pome-producing regions of the world and losses are greatest in humid climates of northern Europe, north west America and southern Chile. Young and old trees can be affected 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). *N. ditissima* also causes eye-rot of apple fruit, which may develop in the orchard or during storage, with losses of up to 80% of the apple crop reported in Europe and America (Berrie *et al.* 2000).

Host range: *N. ditissima* has a wide host range, being recorded on more than 60 tree and shrub species from 20 genera, including apple, pear, walnut, maple, elm, birch and many more. It is prevalent in commercial apple (*Malus* spp.) and pear (*Pyrus* spp.) orchards from most temperate growing regions of the world (Langrell 2002), and beech forests in North America. 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.

Distribution: *N. ditissima* has been recorded on hosts from climates ranging from sub-arctic (Iceland, Sweden, Canada), temperate (Europe, USA, Chile), arid (Syria, Saudi Arabia, Afghanistan) to tropical (Java, Florida).

Geographic distribution: Asia (Afghanistan, China, India, Indonesia, Iran, Iraq, Japan, Korea, Lebanon, Saudi Arabia, Syria), Canada, Europe (Austria, Belgium, Bulgaria, Denmark, Estonia, Faroe Islands, France, Germany, Greece, Hungary, Iceland, Ireland, Italy, Lithuania, Macedonia, Netherlands, Norway, Poland, Portugal, Romania, Russian Federation, Slovakia, Spain, Sweden, Switzerland, Ukraine, United Kingdom, Yugoslavia), Mexico, New Zealand, South Africa, South America (Argentina, Chile, Uruguay), USA.

Note that the pathogen has been eradicated from Tasmania in Australia (Ransom, 1997). Its presence and establishment in South Africa is also being re-examined (Carstens *et al.* 2010).

Biology and Ecology: *Neonectria ditissima* overwinters as mycelium in twigs and callus tissue of cankers, growing slowly while its host is dormant, or as red/orange perithecia (sexual fruiting structures) in cankered wood (Figure 1). Spore production is initiated during periods of cool, wet weather. On younger cankers asexual conidia are generally produced in cream-coloured sporodochia in spring/early summer (Figure 2). It is unusual for the bright-red to orange perithecia to be formed in the first year following infection. On older cankers, ascospores (sexual spores produced in the perithecia) and conidia are produced, both of which can cause infection. 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. *Neonectria ditissima* perithecia *in situ* on apple twig (J. Edwards).

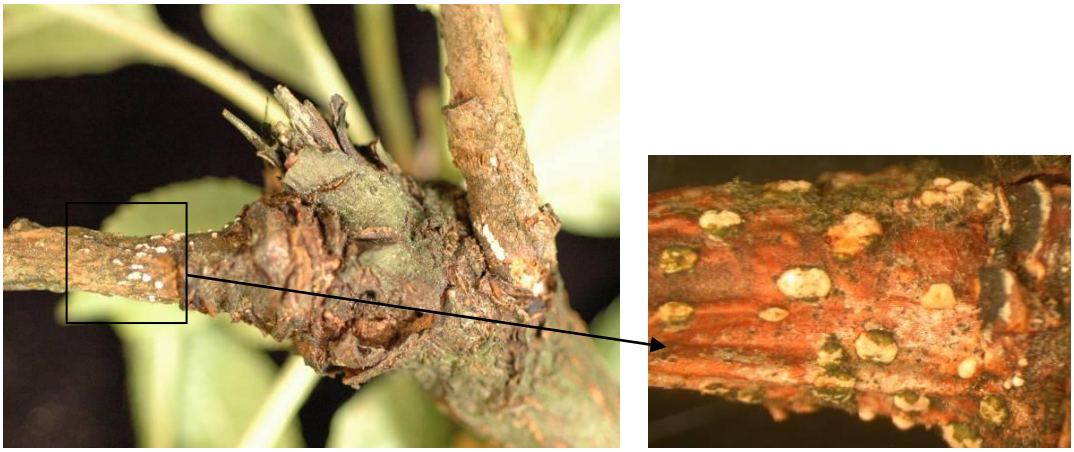

Figure 2. *Neonectria ditissima* asexual spore production *in situ* on apple twig (J. Edwards).

Conidia are dispersed by moist wind currents and rain splash, and in some cases carried by insects to susceptible tissue (Houston 1994). Release of ascospores is dependent on rainfall

quantity and duration of wet periods. Ascospores can be dispersed by rainsplash but are generally regarded to be aerially disseminated. 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.

The fungus enters its host through leaf scars or wounds caused by pruning, insect feeding, winter injury or invasion by other pathogens. 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.

Infection of the fruit occurs on the tree through open calyxes, lenticels, scab lesions or insect wounds, and may remain latent (i.e. symptomless), developing post-harvest into storage rots (Dewey *et al.* 1995).

Symptoms: European canker mainly affects woody tissue (twigs, branches and trunks), and also produces fruit rots.

Apple – wood symptoms. The first sign of canker is a reddish brown spot around a leaf scar, spur or pruning wound or as small, inconspicuous dark depressions on young stems in spring and early summer, leading to twig death and spur death (Figure 3). If not pruned out, the disease can take over and kill the tree.

Figure 3. Leaf scar infection resulting in death of whole twig; apple cv. Royal Gala; Waikato, NZ. (J. Edwards)

Young developing cankers appear as reddish brown lesions (Figure 4). 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. As cankers enlarge, they often girdle infected twigs and branches, killing them.

Figure 4. Young canker on apple cv Braeburn, Waikato, NZ. (J. Edwards)

Cankers on the main stem of older trees reduce the vigour and the value or productivity of the tree (Figure 5). Decreases in water conductivity due to distorted and narrow xylem, especially on branches and stems with numerous or large cankers, result in smaller fruit. These trees are also subject to wind breakage. Pruning to control European canker eventually results in misshapen, hard-to-manage orchard trees.

Figure 5. Cankers developed from infected pruning wounds; apple cv. Red Delicious; County Armagh, Northern Ireland. (J. Edwards)

The disease is most noticeable when the apple tree is in full foliage, as the dead twigs and spurs stand out. Twig death can be traced back to a canker girdling the stem if the cause is *N. ditissima*. Symptoms can be confused with woolly aphid damage which kills buds, resulting in swollen dead cankers, or fireblight, which causes twig death without cankers, or other canker diseases. The characteristic red perithecia, if present, distinguish European canker from these others.

In young apple trees infected by *N. ditissima* at the propagation phase, the disease has been shown to remain latent for up to 3 years (McCracken *et al.* 2003), expressing as poor growth, early senescence and dieback of the young tree.

Apple – fruit symptoms

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. Rotting of fruit can occur while the fruit is still on the tree and such fruit becomes mummified.

Depending on the variety, symptoms of fruit infection are generally not observed until shortly before or after harvest, or in storage. The most obvious symptom of fruit infection is a brown rot (known as eye rot) characterised by circular, sunken necrotic areas on the surface of the fruit that develops before harvest (Figure 6). Others, particularly varieties with an open sinus, may develop a core rot that is difficult to detect without cutting open the fruit (Figure 7) and may remain latent for many months in long term cold storage. Rot

generally develops at the calyx end of the fruit and is a darker brown than rots produced by *Penicillium* species. Internally, the rotted tissue is soft and may have a striated appearance. Fruit rot caused by *N. ditissima* cannot be diagnosed from visual symptoms alone and should always be confirmed by other means.

Figure 6. Eye rot on apple cv. Red Delicious, Northern Ireland (J. Edwards).

Figure 7. Core rot in apple cv. Golden Delicious showing striations in the rotted tissue, Northern Ireland (J. Edwards).

Symptoms on hosts other than apple. *Neonectria ditissima* also causes cankers on other woody hosts. There are two types of canker: open and closed. Open cankers are a series of

concentric calluses resulting in the characteristic target-like cankers (Figure 8). Open cankers are formed when conditions are favourable for the host and the fungus is slow growing.

Figure 8. Open target canker on birch (*Betula* species), Michigan, USA. (Robert L. Anderson, USDA Forest Service, Bugwood.org).

Closed cankers occur when the conditions favour *N. ditissima,* allowing rapid growth of the fungus. Closed cankers are more irregular than the open cankers and are covered by dead bark. The outer bark is rough and cracked but does not fall off for several years in these cankers (Figure 9).

Figure 9. Closed canker on Asian pear (*Pyrus pyrifolia*) trunk. Photo: Bengt Boysen.

Affected plant stages: All

Affected plant parts: Twigs, branches, trunks, fruit

Affected Industries: Pome fruit, walnut, timber.

Resistant plant variety: Apple cultivars differ in their susceptibility to European canker. The Delicious cultivars are highly susceptible eg Red Delicious, Golden Delicious and Royal Gala, which has a Red Delicious parent. In these cultivars, the symptoms start in buds and twigs, and progress into large branches, trunks, etc., killing the tree if not regularly pruned out. Cultivar Braeburn is moderately susceptible, and the disease remains relatively confined to killing spurs. Cultivar Granny Smith is moderately resistant. The infection is contained; killing small twigs but does not move into larger branches. Pear is generally considered more resistant than apple.

Disease movement and Dispersal: Internationally, the fungus is spread via vegetative propagation plant material, rooted plants (Howard *et al.* 1974, McCracken *et al.* 2003) and fruit (CPC 2005). Therefore, in Australia, entry of the pathogen is most likely to be discovered through suspicious rots occurring in imported fruit or suspicious symptoms such as twig dieback observed on hosts in the field. Once established within an orchard, disease is predominantly spread during wet, windy weather by ascospores released into the wind, or conidia carried in water rivulets on the bark of the infected trees. Insects and contaminated pruning tools may also spread disease.

Quarantine Risk: Plant Health Australia lists this pathogen as a high priority pest of apple, pear and cherry.

Entry Potential: Rating = High. Infection can be latent on fruit (Pest Risk Review For European Canker 2005).

Establishment Potential: Rating = High. Wide range of primary and secondary hosts (Pest Risk Review For European Canker 2005).

Spread Potential following Establishment: Rating = High. Has spread since detection in New Zealand (Pest Risk Review For European Canker 2005).

Economic impact: Rating = Medium to High. European canker is one of the most economically damaging diseases of apple in Europe, North America and South America. In Europe, there are reports that the severity of epidemics is increasing. However, the losses are very difficult to quantify as they occur at all stages of production, from the tree nursery to the fruit store. Cankers on branches and stems can necessitate tree replacement ranging from 10% of trees to whole plantations. In Northern Ireland storage losses for fruit of the Bramley's seeding variety varied from 3-60% depending on the type of storage about half of these rots being attributed to *Neonectria ditissima*. Bramley's seedling is more a cooking variety where losses before harvest are generally negligible, rots appearing only after storage. In France, 0.5% and 2% of stored apples of varieties Reinettes du Mans and Rinettes blanches du Canada respectively rotted in storage due to Nectria. Economic damage to host species used for timber, through reduction in both quality and quantity of marketable logs, particularly in North America, has been reported but there is no estimates of the magnitude of this loss. The absence of reports of major losses attributable to European canker in pear plantations implies that it is of less importance on this crop than on apple. (Pest Risk Review For European Canker 2005).

Overall risk: Moderate to high (Pest Risk Review For European Canker 2005).

Acknowledgements. This material was previously written as part of Edwards J, Villalta O, Powney R. (2006) and the National Diagnostic Protocol for Detection of *Neonectria ditissima* (European canker) (Edwards, in review), and the pest risk assessment information was from the Pest Risk Review For European Canker (2005) Plant Health Australia.

References

Berrie, A., Barbara, D., Locke, T. and McCracken, A. (2000) Using molecular biology to study the epidemiology of canker in apple orchards. *The Apple and Pear Research Council (APRC) News* 24: 7-8.

Carstens, E., van Niekerk, J.M., Smit, W.A., Fourie, P.H. (2010). Resolving the status of *Neonectria ditissima* in South Africa. *Australasian Plant Pathology* 39: 43-45.

CPC (2005) 'CAB international crop protection compendium' (Commonwealth Agricultural Bureau International; Wallingford, UK)

Dewey, F.M., Li, R., Swinburne, T. (1995). A monoclonal antibody immunoassay for the detection of *Nectria galligena* in apple fruit and woody tissues. *Bulletin OEPP/EPPO Bulletin* 25, 65 – 73.

Edwards J, Villalta O, Powney R. (2006), . Investigate the current state of knowledge worldwide regarding Neonectria galligena. DPI Victoria Final Report to Horticulture Australia AP05029,

Grove, G.G. (1990) Nectria canker. *Compendium of Apple Diseases*. 35-36 pp.

Houston, D.R. (1994). Major new tree disease epidemics: beech bark disease. *Annual Review of Phytopathology* 32: 75-87.

Howard, B.H. Skeen, D.S. Coles, J.S. (1974). The effects of different grafting methods upon the development of 1-year old nursery apples trees. *Journal of Horticultural Science* 49, 287-295.

Langrell, S.R.H. (2002) Molecular detection of *Neonectria galligena* (syn. *Nectria galligena*). *Mycological Research*, 106(3): 280 - 292.

Lovelidge B. (1995) Solving the apple canker mystery. *Grower*, 123(5):23-25.

McCracken, A.R., Berrie, A., Barbara, D.J., Locke, T., Cooke, L.R., Phelps, K., Swinburne, T.R., Brown, A.E., Ellerker, B., Langrell, S.R.H. (2003). Relative significance of nursery infections and orchard inoculum in the development and spread of apple canker (*Nectria galligena*) in young orchards. Plant Pathology. 52: 553-566.

Ransom, L.M. (1997) The eradication of *Nectria galligena* from apple trees in Tasmania, 1954 to 1991. *Australasian Plant Pathology*, 26: 121 - 125.

Swinburne TR, 1975. European canker of apple (Nectria galligena). Review of Plant Pathology, 54(10):787-799.

http://www.planthealthaustralia.com.au/pidd-docs/157%20-%20European%20canker%20PRR%20-%202005.pdf

THREAT DATA

Oriental fruit fly

(*Bactrocera dorsalis*)

http://en.wikipedia.org/wiki/Image:Bactrocera_dorsalis.jpg

CRC10162

Communicating Uncertainty in Biosecurity Adaption

CRC PLANT biosecurity

Oriental fruit fly

(*Bactrocera dorsalis*)

Oriental fruit fly (*Bactrocera dorsalis)* is a tropical species and widespread in tropical Asia. *B. dorsalis* is a serious pest of a wide variety of unrelated fruit crops and is one of the most destructive fruit fly pests of Asia and the Pacific region.

Distribution: The oriental fruit fly is widespread throughout much of Pakistan, India, Sri Lanka, Sikkim, Myanmar, Indonesia (Celebes, Borneo, Sumatra, Java), Malaya, Thailand, Cambodia, Indochina (Laos, Vietnam), southern China, Taiwan, Philippine Islands, Ryukyu Islands (including Okinawa), Micronesia, Mariana Islands (Guam, Rota, Saipan, Tinian), Bonin Islands, and Hawaiian Islands. It has been introduced to Palau, Hawaii, Nauru and Tahiti, and has been eradicated from southern Japan (Ryukyu Is) and Mauritius.

Potential distribution in Australia: All tropical and subtropical fruit growing areas, particularly along the Queensland and northern NSW coast, Northern Territory and north-west Western Australia.

Host range: The oriental fruit fly has been recorded from more than 150 fruit and vegetables, including apple, citrus, guava, mango, papaya, avocado, banana, loquat, tomato, surinam cherry, rose-apple, passion fruit, persimmon, pineapple, peach, pear, apricot, fig, and coffee. Avocado, mango, and papaya are the most commonly attacked.

Biology and Ecology: Development from egg to adult under summer conditions takes about 16 days. The mature larva emerges from the fruit, drops to the ground, and forms a tan to dark brown puparium. Pupation occurs in the soil. About nine days are required for attainment of sexual maturity after the adult fly emerges. The developmental periods may be extended considerably by cool weather. Under optimum conditions, a female can lay more than 3,000 eggs during her lifetime, but under field conditions from 1,200 to 1,500 eggs per female is considered to be the usual production. Apparently, ripe fruit are preferred for oviposition, but immature ones may be attacked also.

Symptoms: The adult oriental fruit fly (Figure 1) is approximately 6 to 8 mm long, or slightly larger than the common housefly, with a narrow brown band along the edge of its wings. The thorax (middle body part) is dark with two prominent, yellow stripes on top and yellow marks on each side. The abdomen is yellowish with a black T-shaped mark. The female has a serrated-tip ovipositor (tube extending from the back end or underneath), which penetrates the host fruit or vegetable and deposits eggs inside.

In some fruit, external oviposition marks are visible (Figure 2). Larvae that hatch from the eggs that are deposited into the fruit feed on the flesh. Associated yeasts and bacteria hasten the decomposition of the fruit. Ripe fruit are more susceptible to attack than unripened and immature ones.

Figure 1. Adult oriental fruit flies on fruit surface. Department of Plant Industry Archive, Florida Department of Agriculture and Consumer Services, Bugwood.org

Figure 2. Adult fly on fruit surface; ovipositor damage evident. Merle Shepard, Gerald R.Carner, and P.A.C Ooi, Insects and their Natural Enemies Associated with Vegetables and Soybean in Southeast Asia, Bugwood.org

Affected plant stages: Fruiting stage and post-harvest

Affected plant parts: Fruit

Affected Industries: High priority pest of Apple and Pear, Avocado, Citrus, Summerfruit, Tropical Fruit

Pest movement and Dispersal: Adult flies can disperse over long distances through flight, while the transport of larvae in infested fruit can result in global movement, giving these flies an extreme risk rating.

Disease Impact: Oriental fruit fly is considered one of the most devastating pests of fruit in areas where it occurs. Damage levels can be up to 100% of unprotected fruit.

Entry potential: Rating = High. Able to be transported in infested fruit.

Establishment potential: Rating = High. Established in many countries, including Tahiti since 1996 and Mauritius from 1996-1997.

Spread potential following establishment: Rating = High

Economic impact: Rating = High. *Bactrocera dorsalis* is one of the most destructive pest insects of tropical and subtropical fruits and vegetables.

Environmental impact: Rating = High

Overall risk: Rating = High

Acknowledgements: The information for this sheet was sourced from Plant Health Australia's National Apple and Pear Industry Biosecurity Plan Appendix 2: Pest Risk Review (compiled February 2006) and Fact Sheet, and the PaDIL Plant Biosecurity Toolbox Webpages authored by Geoff Waite.

References

http://www.planthealthaustralia.com.au/pidd-docs/Oriental%20fruit%20fly%20FS.pdf

http://www.planthealthaustralia.com.au/pidd-docs/021%20-%20Oriental%20fruit%20fly%20PRR%20-%202005.pdf

http://old.padil.gov.au/pbt/index.php?q=node/46&pbtID=155

THREAT DATA

Rosy apple aphid

(*Dysaphis plantaginea*)

(Ref. http://www.invasive.org/browse/detail.cfm?imgnum=1326213)

CRC10162

Communicating Uncertainty in Biosecurity Adaption

CRC PLANT biosecurity

Rosy apple aphid

(*Dysaphis plantaginea*)

Rosy apple aphid, *Dysaphis plantaginea*, is the most destructive of the five aphid species that feed on apple. In severe infestations, up to 50% of the fruit crop can be damaged. It also feeds on pear and hawthorn. Rosy apple aphid has been a major pest of apple in North America since the end of the 19th century, but is currently not present in Australia.

Distribution: Rosy apple aphid is native to Europe where it is widespread throughout. It was introduced into north America in 1870 where it has become an established pest. EUROPE (excl. USSR), Austria, Belgium, Britain, Bulgaria, Czechoslovakia, Denmark, Finland, France, Germany, West Greece, Hungary, Ireland, Italy, Netherlands, Norway, Poland, Portugal, Sicily, Spain, Sweden, Switzerland, ASIA (excl. USSR), Afghanistan, Cyprus, Iran, Israel, Japan, Korea, Lebanon, Nepal, Pakistan, Taiwan, Turkey, USSR, AFRICA, Egypt, Ethiopia, Libya, Morocco, Tunisia, NORTH AMERICA, Canada, USA (CABI 1981). The pest is unknown in Australia, New Zealand and Oceania, and Central and South America.

Host range: Apple is the preferred host for rosy apple aphids, but they can also feed on pear and hawthorn. The winged adult female also feeds on narrow- and broad-leaf plantain and dock: *Plantago lanceolata, P. major* and *P. rugelii.*

Affected Plant Stages: Vegetative and flowering stages.

Affected Plant Parts: Foliage, fruit spurs, flowers and developing fruit.

Biology and Ecology: Rosy apple aphids overwinter in the egg stage. In the autumn, the females lay 0.5 mm long oval yellow eggs in crevices in the bark of larger branches. The eggs darken over one to two weeks to shiny black and are impossible to differentiate from those of other apple aphids. In spring, the eggs hatch over a two week period while the buds are in the silver-tip to half-inch green stage. The individuals which hatch from the eggs are all wingless females. They pass through five nymphal instars (stages) and range in length from 0.4-2.0 mm; as they grow, the aphids change in colour from dark green to rosy purple (see cover picture). The last instar is the mature stem mother which produces live young without being fertilised by a male. Each female produces an average of 185 offspring, which leads to rapid buildup of large populations. Nymphs cluster around each mother to the extent that infested leaves may be covered by more than one layer of aphids.

One generation is completed in two to three weeks. Adult aphids in a colony are generally wingless until crowded conditions induce the formation of winged individuals that can disperse to new hosts. The wingless adults are rosy brown or purple in colour and are covered in a greyish-white wax coating.The winged adults are 2.0-2.5 mm in length and are brownish-green and black in colour. The winged aphids often fly to a different plant species which is called the secondary host. Rosy apple aphids may remain on apple throughout the

summer, but usually move to narrow-leaf plantain or dock in early-summer. By mid-summer, most of the aphids have left the apple trees. Reproduction without mating continues on secondary hosts (plantain, dock) until late-summer or autumn when the winged females fly back to the apple trees. They are darker than the migrants that left the tree in spring. These returning females lay eggs, from which males also develop. The males mate with the females, which then deposit eggs on the bark.

Symptoms: The body of this aphid has a waxy coating and usually a slight purplish or rosy tinge (Figure 1); hence the name. Young aphids congregate closely around the mother, and in some cases, the colonies are made up of more than one layer of aphids.

Figure 1. Rosy apple aphids.

Curling and twisting of leaves and young shoots is characteristic. The rosy apple aphid injects a toxin with its saliva that causes the leaves to curl (Figure 2) and the fruit to be distorted (Figure 3). It also stunts new growth and causes sooty mold to develop on fruit and leaves. Often these damaged leaves turn bright crimson in color. Relatively low numbers of rosy apple aphids can cause considerable damage.

Fig.1.Leaf curling and distortion caused by rosy apple aphid. Fig. 2. Fruit and leaves distorted by rosy apple aphid (ref.http://www.ipm.ucdavis.edu/PMG/r4301511.html)

Entry Potential: Rating = LOW. It is unlikely to be associated with fruit. It could be associated with budwood contaminated with overwintering eggs. Previous risk analyses rate entry by aphids of this life habit on fruit as low.

Establishment Potential: Rating = LOW. This pest has a host range with a number of species with home-garden members but the likelihood of it finding a suitable host at the right stage in its life cycle is low.

Spread potential after establishment: Rating = MEDIUM-HIGH. This pest is likely to spread readily by natural means, such as flight or wind as are other aphids. The need for its secondary host to be present is unlikely to be a constraint in Australia.

Economic Impact: Rating = HIGH. Based on American and Canadian experience it is a serious pest or apples in particular, difficult to control and with significant damage potential.

Environmental impact: Rating: Negligible due to its restricted host range.

Conclusion: Overall risk rating: LOW

Acknowledgements: The information for this sheet was sourced from Plant Health Australia's National Apple and Pear Industry Biosecurity Plan Appendix 2: Pest Risk Review (prepared by Michael Jefferies, 2006) and Fact Sheet (compiled by Suzy Perry), and the Ontario Ministry of Agriculture, Food and Rural Affairs Publication 310, Integrated Pest Management for Apples, website last updated Sept 27, 2011.

References

CABI, 1981. Distribution Maps of Pests. Series A, No. 429. Wallingford, UK: CAB International.

Plant Health Australia (2006) National Apple and Pear Industry Biosecurity Plan Pest Risk Review, prepared by Michael Jefferies. http://www.planthealthaustralia.com.au/pidd-docs/085%20-%20Rosy%20apple%20aphid%20PRR%20-%202005.pdf

Plant Health Australia Rosy Apple Aphid Fact Sheet, prepared by Suzy Perry. http://www.planthealthaustralia.com.au/pidd-docs/Rosy%20apple%20aphid%20FS.pdf

http://www.omafra.gov.on.ca/english/crops/facts/rosyaph.htm

THREAT DATA

Varroa mite

(*Varroa destructor, Varroa jacobsoni*)

Photo by Kika De La Garza Subtropical Agricultural Research Center Weslaco, Texas, USA (PD-USGov-USDA-ARS)

CRC10162

Communicating Uncertainty in Biosecurity Adaption

CRC PLANT biosecurity

Varroa mite

(*Varroa destructor*)

Varroa mites are parasites of bees and are the most serious pest of honey bees worldwide. Varroa infects honey bees in every major beekeeping area of the world, except Australia. There is currently a combined government and industry effort to keep them out of Australia. If Varroa were to become established in Australia our healthy population of feral honey bees, and the pollination services they provide, could be reduced by 90-100%. The effects would be significant for apiarists, who would face higher costs to manage their hives, and producers of crops such as almonds, apples, cherries, sunflower and canola that rely on pollination from bees. It is estimated that Varroa mite could cost Australian plant industries between $21.3 million and $50.3 million per year over thirty years (Source: CSIRO Submission no. 33, p. 10, to the House of Representatives Standing Committee Inquiry into the Future Development of the Australian Honeybee Industry).

Distribution: Varroa mites are native to Asia and have adapted to living on domestic European honeybees. They have become established in most beekeeping regions of the world (Figure 1). Varroa is not present in Australia, but has established in our near neighbours - New Zealand and Papua New Guinea.

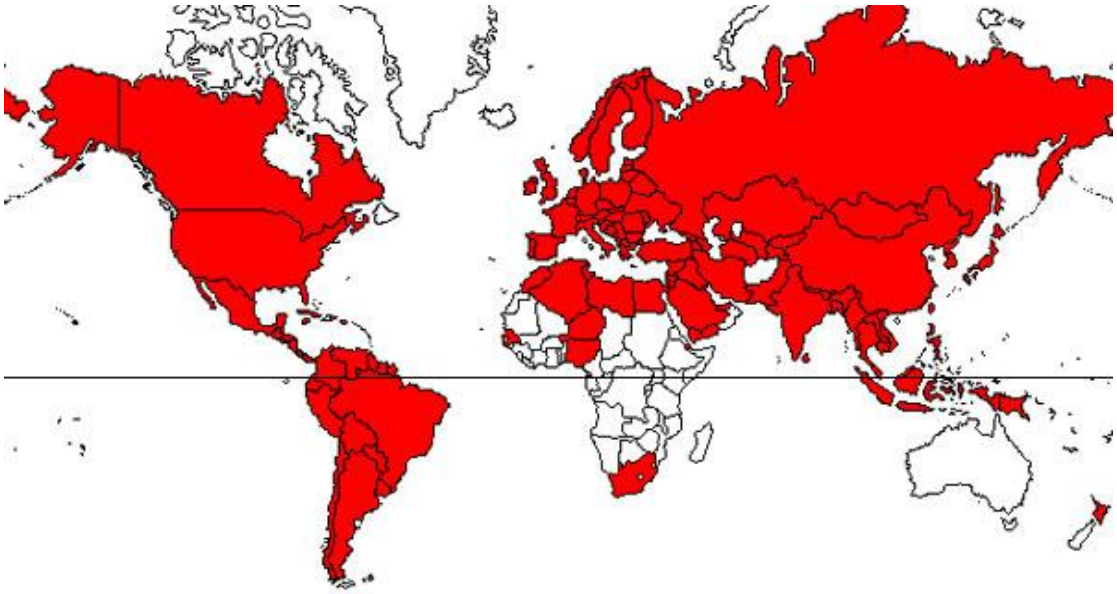

Figure 1. Current varroa mite distribution - 2010. Red areas indicate establishment of *Varroa destructor*. (Source: University of Florida Featured Creatures)

Range: Varroa mites include a group of species, including *V. destructor, V. jacobsoni, V. underwoodi, V. rindereri* and un-named species. It was believed, up until recently, that only *V. destructor* posed a threat to managing European honey bees, *Apis mellifera*. Varroa have evolved with *Apis cerana* (Asian honey bees). The impact of varroa on Asian honey bees is not lethal. The varroa mites normally breed on Asian honey bee drone brood with minimal impact on the Asian honey bee colony. Dr Denis Anderson (CSIRO, Canberra) in a 2000 publication, stated that some varroa were reproducing on honey bees, while other varroa were not. He was able to identify specific varroa that could breed on honey bees and these were named by him as *V. destructor*. This cross-species infestation of *V. destructor* on honey bees probably started around 50 years ago. *V. jacobsoni* is a mite infesting Asian bees throughout Papua New Guinea and Indonesia. Unfortunately, there is now evidence that suggests *V. jacobsoni* is reproducing on European honey bees. Thus, any varroa incursion into Australia has the potential to create major management problems for beekeepers.

Biology and Ecology: Varroa can only reproduce on bee brood. No brood equals no mite breeding. Mites find drone honey bee brood significantly more attractive to breed in than worker brood. Approximately 4 out of 5 mites will enter drone brood if given a choice.

The female mite enters the brood cell of an advanced larva just before the cell is capped by nurse bees. The mite sinks itself into the larval food at the bottom of the cell and emerges once the brood cell is fully capped. She will then move onto the developing bee larvae/pupae and feed on the haemolymph (bees blood). The mother mite may lay up to six eggs at intervals of about 30 hours. The first egg develops into a male mite and the rest are female.

Mite development from egg to adult takes about 8–10 days. The first mite (male) mates with the female mites as they mature. On average, 1.5 daughter mites emerge from a worker cell and 2.5 daughter mites emerge from a drone cell along with the mother mite. The male and undeveloped female mites die inside the cell.

The mother mite and her daughters are then capable of crawling back into adjoining brood cells to complete the reproduction life cycle again. Once the population of mites has increased substantially, it is possible for several mites to be in the one brood cell. There can be 24–30 breeding cycles for the mites in a year. It is believed that female mites will breed up to three times.

Mite numbers increase slowly within a hive. It may not be until the fourth year of infestation that numbers are sufficiently high for honey bee larvae to be parasitised by several females. When this occurs, newly emerged adult bees with deformed wings, legs and abdomens may be found at the hive entrance.

Symptoms: When mites are in low numbers in a colony of honey bees they are difficult to detect. Individual mites are easily identifiable with the naked eye. They look like small brown sesame seeds with eight legs. The females are flat and about 1.1 mm long and 1.7 mm across. Adult males are smaller and are yellowish-white. Both sexes have eight legs. The eggs are 0.5 mm long, milky-coloured and at first rounded. Females of *Varroa jacobsoni* are smaller than females of *V. destructor*, being about 1.0 mm long and 1.5 mm wide.

Unfortunately mites are very good at concealing themselves on adult honey bees (Figure 2). It is generally agreed that to observe adult mites on adult honey bees is very difficult and totally unreliable as a diagnostic tool.

In spring and summer when breeding conditions are ideal most colonies rear large numbers of drones. Occasionally drone brood comb is built between the top bars of combs and the queen excluder. When inspecting a colony and removing the queen excluder, developing brood pupae and larvae can be exposed. The presence of mites feeding on the drone brood is very obvious, as the brown sesame seed-sized mite feeding on the white drone pupae is very distinct (Figure 3).

If you are not deliberately monitoring for mites, the colony is likely to collapse before you are aware of the presence of mites. A colony can appear to be populous with healthy looking brood one week and be all but extinct the following week. In this case the brood pattern is irregular and may look similar to that observed with brood diseases. However, a sample of 'infected' larvae sent to the laboratory for diagnosis is unlikely to be positive for European foulbrood or American foulbrood. This condition has been termed 'parasitic mite syndrome', or PMS.

Figure 2: Varroa mites on a European honeybee and honeybee pupa

Figure 3. Drone brood infested with a varroa mite

Affected Industries: High priority pest of commercial honey industry and all open pollinated crops

Pest movement and Dispersal: Adult mites are quite capable of living for more than five days without the presence of honey bees. This means that the transport of hives, used beekeeping equipment and queen bees by beekeepers is a very effective means of spread. In Australia, the spread of varroa is expected to be fast over long distances because of the migratory nature of the beekeeping industry.

Drone bees drift from hive to hive and even between apiaries. They are certainly able to move varroa mites around. Foraging worker bees will come in contact with other bees when visiting blossom for nectar and pollen. Mites are very agile and quick in moving and can transfer between bees in passing.

Impact: If left untreated in a honey bee colony, varroa mites will kill it. All feral and untreated bee colonies will eventually die. This necessitates very careful management from a beekeeper's perspective to detect and treat mites as and when their population increases to critical levels. There is a significant cost in materials and labour involved in varroa management. There is also the likelihood of the chemicals used for such purposes leaving residues of one form or another in the beeswax and honey.

The most obvious threat is to Australia's bee and honey industries. The most significant impact will be the death of all untreated honey bee colonies across the landscape. The

Varroa mite would decimate Australia's feral bee population and cause a rapid increase in demand for pollination services. This will seriously reduce the positive impact of honey bees in the environment of pollinating a range of horticultural, broadacre crop and pastoral plants.

However, the major part of the cost of Varroa would probably be felt not by the honeybee industry but by other industries with crops that rely on honeybees for pollination, including almonds, avocadoes, cotton, stone fruits, pome fruit, melons and pumpkins. The value of honey bees as pollinators is considered to be extremely important and it is estimated that Varroa mite could cost Australian plant industries between $21.3 million and $50.3 million per year over thirty years (Cook *et al.* 2007).

Varroa mites were discovered in New Zealand in 2000 and have already had a major economic impact, with significant control costs and losses of bees, hives, honey production, crop yields and export revenue.

Entry, spread and establishment: Entry into Australia would likely be in the form of a feral European or Asian honeybee swarm on cargo arriving at an Australian seaport. If varroa entered eastern Australia, they would be capable of spreading throughout Queensland, New South Wales and Victoria within five years.

Varroa is a notifiable disease in all States and Territories. Notification is required by law. Early recognition of varroa is one of the most important factors influencing the chance of controlling the disease and reducing its economic and social impact on the whole community.

Aknowledgements: The information for this document was extracted from Fact Sheets produced by NSW DPI, Victoria DPI and Biosecurity Queensland, and the Department of Agriculture, Fisheries and Forestry website.

Information Sources:

AG1183. Varroa - An Exotic Parasitic Mite of Honey Bees. Russell Goodman. July 2010. http://www.dpi.vic.gov.au/agriculture/pests-diseases-and-weeds/pest-insects/varroa

Cook DC, Thomas MB, Cunningham SA, Anderson DL, De Barro PJ (2007) Predicting the economic impact of an invasive species on an ecosystem service. *Ecological Applications* 17, 1832–1840.

NSW DPI Primefact 861. Varroa Mites. Doug Somerville. January 2009. http://www.dpi.nsw.gov.au/__data/assets/pdf_file/0006/268026/Varroa-mites.pdf

http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/varroa-mite. Sept 2009.

Queensland Government Primary Industries and Fisheries. Varroa mites. Jessica Thompson, Sept 2011. http://www.dpi.qld.gov.au/4790_13505.htm

University of Florida Featured Creatures. http://entnemdept.ufl.edu/creatures/misc/bees/varroa_mite.htm

Appendix 2: NetLogo model equations.

```
extensions [ gis ]

globals [ raster-dataset output

  Week Month Year

  Region_Infection_Status

  Species_List_Flowering_season

  Detection_values Time_since_first_detection

  Weekly_quarantine_surveillance_cost Total_OCR_cost Total_quarantine_cost
Total_detections Total_destruction_area Total_cost

  Shoot_strike_detection_month

  i j x y

  Cell_size Quarantine_radius Bee_dispersal_radius Destruction_radius

  Number_of_crops

  Economic_return_FB

  Economic_return_no_FB

  OCR_list_LD OCR_list_HD

  tree_per_ha

  Bees_dispersing Bees_dispersal_distance

  Maximum_Yield_Apple Maximum_Yield_Apple_FB Maximum_Yield_Pear
Maximum_Yield_Pear_FB

  Total_revenue_loss

  Degree_of_Infection_threshold

  High_density_values

  Productivity_loss_FB

  ]


patches-own [grid-value

  Tree_Species

  Tree_Maturity Maturity_initialise

  Density   ; "low" or "high"

  Tree_potential_Yield Tree_potential_Yield_FB

  Flowering Pruning

  Infection_Status

  Detection_Status

  Degree_of_Infection
```

```
  Quarantine_Status
  Detection-probability
  Time_since_detection Time_since_quarantine
  Cost_to_patch
  Quarantine_cost_to_patch ]

breed [bees bee]
breed [infection_detectives infection_detective]

;*************************************************************************
*********
;***************************Setup
*****************************************
;*************************************************************************
*********

; this cleans up layers and defines projections
to setup
  clear-turtles
  clear-patches
  clear-drawing
  clear-all-plots
  clear-output
  ;; (for this model to work with NetLogo's new plotting features,
  ;; __clear-all-and-reset-ticks should be replaced with clear-all at
  ;; the beginning of your setup procedure and reset-ticks at the end
  ;; of the procedure.)
  __clear-all-and-reset-ticks
  set Cell_size 32
  set Number_of_crops 2
  Set Week 0
  Set Month 0
  Set Year  0
  set Time_since_first_detection 0
  Set Species_List_Flowering_season [["Apple" [9 2 10 2] [5 1 7 4] 6 lime] ["Pear" [9 1 10
1] [5 1 7 4] 7 yellow]];the first value is the name of the crop and the next values is
[start_month start_week and end_month, end_week of flowering season] [ pruning] then
LU and color.
```

```
  set High_density_values [10 2]  ; define the proportion of high density pklanting on apple and pear [Apple density pear density]

  set Detection_values [0.1 1 0.2 50] ;first is the dfetection probability during flowring then at the shoot strike month then at the end season and then during quarantine.

  set Shoot_strike_detection_month 11

  set Weekly_quarantine_surveillance_cost (20475 / (700000 / Cell_size ^ 2)) / 48  ; $ per week in surveillance costs

  set OCR_list_HD [[43 47 83 139 61] [43 52 58 152 62]]

  set OCR_list_LD [[60 86 154 67] [57 179 367 204]]    ; cost given by government for destruction per tree as a function of tree age [[apple][pear]] in high density planting

;  for high density: OCR_list [[43 47 83 139 61] [43 52 58 152 62]]

;  for low density  OCR_list [[60 86 154 67] [57 179 367 204]]


  set tree_per_ha [900 2500]  ;  for low density 900 trees / ha for high density 2500 trees/ha

  set Maximum_Yield_Apple (12000 / 10000 * Cell_size ^ 2)   ; previous value 16587.55

 ; set Maximum_Yield_Apple_FB (10800 / 10000 * Cell_size ^ 2) ; previous value 11494.40

  set Maximum_Yield_Pear (10000 / 10000 * Cell_size ^ 2)   ; previous value 29654.13

;  set Maximum_Yield_Pear_FB (9000 / 10000 * Cell_size ^ 2)   ; previous value 23142.95


  do-plots



  ; Note that setting the coordinate system here is optional, as long as all of your datasets use the same coordinate system.

  gis:load-coordinate-system (word "C:/Program Files/NetLogo 5.0/models/Code Examples/GIS/data/" projection ".prj")

  ; Load all of our datasets

  set raster-dataset gis:load-dataset input-raster

  ; Set the world envelope to the union of all of our dataset's envelopes

  gis:set-world-envelope (gis:envelope-union-of (gis:envelope-of Raster-dataset))

  plant-trees

end



;*************************************************************

; ******************** get values from raster into patches *********
```

```
;*****************************************************************
to plant-trees ; this procedure get values from the raster and place them in the patches
  gis:apply-raster raster-dataset grid-value
  ask patches [set Tree_Species ""]
  ask patches with [grid-value > 0 ][set pcolor white ]
  ask patches with [grid-value = 0] [set pcolor 102]
  set i 0
  while [i < Number_of_crops]
  [ask patches  with [grid-value = item 3 item i Species_List_Flowering_season] [if random 100 < 100
      [set Tree_Species (item 0 item i Species_List_Flowering_season) set pcolor (item 4 item i Species_List_Flowering_season)
        set Maturity_initialise random 100 ifelse Maturity_initialise >= 20 [set Tree_Maturity 5 + random 7] [ifelse Maturity_initialise < 5 [set Tree_Maturity 1]
          [ifelse Maturity_initialise < 10 [set Tree_Maturity 2] [ifelse Maturity_initialise < 15 [set Tree_Maturity (3 + random 3)] [if Maturity_initialise < 20 [set Tree_Maturity (13 + random 3)]]]]]
       ifelse  random 100 <= item i High_density_values [set Density "High"] [set Density "Low"]]]

     ; set maturity age so 80% is more then 5 years old.       use 27 for %
  set i i + 1 ]
end


;*****************************************************************
; ******************** Unit calculations ************************
;*****************************************************************

to calculated_distances    ; Calculate distances from the text choosers defined in the interface.
  set Quarantine_radius int((read-from-string(remove "km" quarantine_distance)) * 1000 / Cell_size)
  set Bee_dispersal_radius int((read-from-string(remove "km" Bees_dispersal-distance)) * 1000 / Cell_size)
  set Destruction_radius int((read-from-string(remove "m" Destruction_distance)) / Cell_size)
  ; calculate losses
  if Live_with_it = TRUE [set Productivity_loss_due_to_FB_% "10%"]
```

```
  set Productivity_loss_FB int(read-from-string(remove "%"
Productivity_loss_due_to_FB_%))

  set Maximum_Yield_Apple_FB Maximum_Yield_Apple * (1 - (Productivity_loss_FB / 100))
; previous value 11494.40

  set Maximum_Yield_Pear_FB Maximum_Yield_Pear * (1 - (Productivity_loss_FB / 100))  ;
previous value 23142.95




end



;*****************************************************************
; ******************** temporal processes ************************
;*****************************************************************


to Calculate_Week_Month_and_Year

  ifelse Week < 4 [set Week Week + 1] [Set Week 1]

  if Week = 1 [set Month Month + 1]

  if Month > 12 [Set Month 1]

  if Month = 1 and Week = 1 [set Year Year + 1]


end


to Calculate_flowering   ; calculated flowering and pruning season.

  set i 0

  while [i < Number_of_crops]

  [ask patches  with [Tree_Species = item 0 item i Species_List_Flowering_season] [ifelse
(Month = (item 0 item 1 item (i) Species_List_Flowering_season) and Week >= (item 1
item 1 item (i) Species_List_Flowering_season) or Month > (item 0 item 1 item (i)
Species_List_Flowering_season)) and (Month = (item 2 item 1 item (i)
Species_List_Flowering_season) and Week <= (item 3 item 1 item (i)
Species_List_Flowering_season) or Month < (item 2 item 1 item (i)
Species_List_Flowering_season))

          [set Flowering 1] [set Flowering 0]]

  ask patches  with [Tree_Species = item 0 item i Species_List_Flowering_season] [ifelse
(Month = (item 0 item 2 item (i) Species_List_Flowering_season) and Week >= (item 1
item 2 item (i) Species_List_Flowering_season) or Month > (item 0 item 2 item (i)
Species_List_Flowering_season)) and (Month = (item 2 item 2 item (i)
```

```
Species_List_Flowering_season) and Week <= (item 3 item 2 item (i)
Species_List_Flowering_season) or Month < (item 2 item 2 item (i)
Species_List_Flowering_season))
          [set Pruning 1] [set Pruning 0]]
 set i i + 1 ]
end

to Calculate_maturity
 ; ask patches with  [not empty? Tree_Species] [ if Month = 1 and Week = 1 [ set
Tree_Maturity Tree_Maturity + 1]]
end


to Grow_infection
 ask patches with [Infection_Status = 1] [ set Degree_of_Infection Degree_of_Infection +
1]
 ;ask patches [if Degree_of_Infection > 100 [Kill_tree]]
 rain_spread_infection
 ask patches with [Time_since_quarantine >= 1] [set Time_since_quarantine
Time_since_quarantine + 1]

end

to Calculate_yield
 ;for Apples HD
 ask patches with [Tree_Species = "Apple" and Density = "High"] [ifelse Tree_Maturity <
3 [set Tree_potential_Yield 0] [ ifelse Tree_Maturity >= 3 and Tree_Maturity <= 5 [set
Tree_potential_Yield (Maximum_Yield_Apple / 3 * Tree_Maturity - (Maximum_Yield_Apple
* 2 / 3))] [ ifelse Tree_maturity <= 13 [set Tree_potential_Yield Maximum_Yield_Apple]
[set Tree_potential_Yield (Maximum_Yield_Apple * exp (- (Tree_Maturity - 13) / 5))] ]]]
 ask patches with [Tree_Species = "Apple"  and Density = "High" and Infection_status =
1] [ifelse Tree_Maturity < 3 [set Tree_potential_Yield_FB 0] [ ifelse Tree_Maturity >= 3
and Tree_Maturity <= 5 [set Tree_potential_Yield_FB (Maximum_Yield_Apple_FB / 3 *
Tree_Maturity - (Maximum_Yield_Apple_FB * 2 / 3))] [ ifelse Tree_maturity <= 13 [set
Tree_potential_Yield_FB Maximum_Yield_Apple_FB] [set Tree_potential_Yield_FB
(Maximum_Yield_Apple_FB * exp (- (Tree_Maturity - 13) / 5))] ]]]
; for pears HD
 ask patches with [Tree_Species = "Pear" and Density = "High"] [ifelse Tree_Maturity < 3
[set Tree_potential_Yield 0] [ ifelse Tree_Maturity >= 3 and Tree_Maturity <= 5 [set
Tree_potential_Yield (Maximum_Yield_Pear / 3 * Tree_Maturity - (Maximum_Yield_Pear *
2 / 3))] [ ifelse Tree_maturity <= 13 [set Tree_potential_Yield Maximum_Yield_Pear] [set
Tree_potential_Yield (Maximum_Yield_Pear * exp (- (Tree_Maturity - 13) / 5))] ]]]
```

```
  ask patches with [Tree_Species = "Pear" and Density = "High" and Infection_status = 1]
[ifelse Tree_Maturity < 3 [set Tree_potential_Yield_FB 0] [ ifelse Tree_Maturity >= 3 and
Tree_Maturity <= 5 [set Tree_potential_Yield_FB (Maximum_Yield_Pear_FB / 3 *
Tree_Maturity - (Maximum_Yield_Pear_FB * 2 / 3))] [ ifelse Tree_maturity <= 13 [set
Tree_potential_Yield_FB Maximum_Yield_Pear_FB] [set Tree_potential_Yield_FB
(Maximum_Yield_Pear_FB * exp (- (Tree_Maturity - 13) / 5))] ]]]


  ;for Apples Low D

  ask patches with [Tree_Species = "Apple" and Density = "Low"] [ifelse Tree_Maturity < 4
[set Tree_potential_Yield 0] [ ifelse Tree_Maturity >= 4 and Tree_Maturity <= 7 [set
Tree_potential_Yield (Maximum_Yield_Apple / 4 * Tree_Maturity - (Maximum_Yield_Apple
* 3 / 4))] [ ifelse Tree_maturity <= 13 [set Tree_potential_Yield Maximum_Yield_Apple]
[set Tree_potential_Yield (Maximum_Yield_Apple * exp (- (Tree_Maturity - 13) / 20))] ]]]

  ask patches with [Tree_Species = "Apple"  and Density = "Low" and Infection_status =
1] [ifelse Tree_Maturity < 4 [set Tree_potential_Yield_FB 0] [ ifelse Tree_Maturity >= 4
and Tree_Maturity <= 7 [set Tree_potential_Yield_FB (Maximum_Yield_Apple_FB / 4 *
Tree_Maturity - (Maximum_Yield_Apple_FB * 3 / 4))] [ ifelse Tree_maturity <= 13 [set
Tree_potential_Yield_FB Maximum_Yield_Apple_FB] [set Tree_potential_Yield_FB
(Maximum_Yield_Apple_FB * exp (- (Tree_Maturity - 13) / 20))] ]]]

; for pears Low D

  ask patches with [Tree_Species = "Pear" and Density = "Low"] [ifelse Tree_Maturity < 4
[set Tree_potential_Yield 0] [ ifelse Tree_Maturity >= 4 and Tree_Maturity <= 7 [set
Tree_potential_Yield (Maximum_Yield_Pear / 4 * Tree_Maturity - (Maximum_Yield_Pear *
3 / 4))] [ ifelse Tree_maturity <= 13 [set Tree_potential_Yield Maximum_Yield_Pear] [set
Tree_potential_Yield (Maximum_Yield_Pear * exp (- (Tree_Maturity - 13) / 20))] ]]]

  ask patches with [Tree_Species = "Pear" and Density = "Low" and Infection_status = 1]
[ifelse Tree_Maturity < 4 [set Tree_potential_Yield_FB 0] [ ifelse Tree_Maturity >= 4 and
Tree_Maturity <= 7 [set Tree_potential_Yield_FB (Maximum_Yield_Pear_FB / 4 *
Tree_Maturity - (Maximum_Yield_Pear_FB * 3 / 4))] [ ifelse Tree_maturity <= 13 [set
Tree_potential_Yield_FB Maximum_Yield_Pear_FB] [set Tree_potential_Yield_FB
(Maximum_Yield_Pear_FB * exp (- (Tree_Maturity - 13) / 20))] ]]]



end



;*****************************************************************
; ******************** Action procedures ************************
;*****************************************************************


to Kill_tree

  ; calculate cost of killing tree first

  if Tree_Species = item 0 item 0 Species_List_Flowering_season [

   ifelse Density = "High" [
```

```
  ifelse Tree_Maturity <= 1 [ set Cost_to_patch  Cost_to_patch + ((item 0 item 0
OCR_list_HD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  ifelse Tree_Maturity = 2 [ set Cost_to_patch  Cost_to_patch + ((item 1 item 0
OCR_list_HD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  ifelse Tree_Maturity > 2 and Tree_Maturity <= 5 [ set Cost_to_patch  Cost_to_patch +
((item 2 item 0 OCR_list_HD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  ifelse Tree_Maturity > 5 and Tree_Maturity <= 13 [ set Cost_to_patch  Cost_to_patch +
((item 3 item 0 OCR_list_HD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  if Tree_Maturity > 13 [ set Cost_to_patch  Cost_to_patch + ((item 4 item 0
OCR_list_HD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]]]]] ]

   [

  ifelse Tree_Maturity < 4 [ set Cost_to_patch  Cost_to_patch + ((item 0 item 0
OCR_list_LD) * item 0 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  ifelse Tree_Maturity >= 4 and Tree_Maturity <= 6 [ set Cost_to_patch  Cost_to_patch +
((item 1 item 0 OCR_list_LD) * item 0 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  ifelse Tree_Maturity >= 7 and Tree_Maturity <= 13 [ set Cost_to_patch  Cost_to_patch
+ ((item 2 item 0 OCR_list_LD) * item 0 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  if Tree_Maturity > 13 [ set Cost_to_patch  Cost_to_patch + ((item 2 item 0 OCR_list_LD)
* item 0 tree_per_ha / 10000 * Cell_size ^ 2)]]]] ]

     ]


  if Tree_Species = item 0 item 1 Species_List_Flowering_season [

    ifelse Density = "High" [

  ifelse Tree_Maturity <= 1 [ set Cost_to_patch  Cost_to_patch + ((item 0 item 1
OCR_list_HD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  ifelse Tree_Maturity = 2 [ set Cost_to_patch  Cost_to_patch + ((item 1 item 1
OCR_list_HD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  ifelse Tree_Maturity > 2 and Tree_Maturity <= 5 [ set Cost_to_patch  Cost_to_patch +
((item 2 item 1 OCR_list_HD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  ifelse Tree_Maturity > 5 and Tree_Maturity <= 13 [ set Cost_to_patch  Cost_to_patch +
((item 3 item 1 OCR_list_HD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  if Tree_Maturity > 13 [ set Cost_to_patch  Cost_to_patch + ((item 4 item 1
OCR_list_HD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]]]]] ]

  [

  ifelse Tree_Maturity < 4 [ set Cost_to_patch  Cost_to_patch + ((item 0 item 1
OCR_list_LD) * item 0 tree_per_ha / 10000 * Cell_size ^ 2)]   [

    ifelse Tree_Maturity >= 4 and Tree_Maturity <= 6 [ set Cost_to_patch  Cost_to_patch
+ ((item 1 item 0 OCR_list_LD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  ifelse Tree_Maturity >= 7 and Tree_Maturity <= 13 [ set Cost_to_patch  Cost_to_patch
+ ((item 2 item 0 OCR_list_LD) * item 1 tree_per_ha / 10000 * Cell_size ^ 2)]   [

  if Tree_Maturity > 13 [ set Cost_to_patch  Cost_to_patch + ((item 3 item 1 OCR_list_LD)
* item 0 tree_per_ha / 10000 * Cell_size ^ 2)]]]] ]
```

```
  ]
  set Tree_Species ""
  set Infection_Status 2
  set Degree_of_Infection 0
  set Tree_Maturity 0
  set pcolor black
  ask turtles in-radius 9 [die]
end

to Infect_tree
  ifelse view_infection = false [if not empty? Tree_Species [ set Infection_Status 1 ]]
                          [if not empty? Tree_Species [   set pcolor sky    set Infection_Status
1]]
end




to place_infection
;; place a agent where the user says to
  if (mouse-down?)
    [ask patch mouse-xcor mouse-ycor
       [ Infect_tree]   ; infect the chosen location.
         display
    ]
end

to Creat_and_move_bees
  calculated_distances
  set Bees_dispersing Number_of_bees
  set Bees_dispersal_distance Bee_dispersal_radius
  set Degree_of_Infection_threshold 4   ; by default we use a low infection pressure
allowing one set of stepping stones dispersal event per year (one jump)


  ifelse High_disease_pressure = true [set Bees_dispersing (Bees_dispersing * 1) set
Degree_of_Infection_threshold 0] [set Bees_dispersing Bees_dispersing / 5]  ; if perfect
weather conditions we double the spread
  if Live_with_it = true [set Bees_dispersing (Bees_dispersing * 0.2)]
```

```
  ;if Live_with_it = true [ifelse (Bees_dispersing * 0.2) >= 1 [set Bees_dispersing
int(Bees_dispersing * 0.2)][if random 100 <= (Bees_dispersing * 0.2) * 100  [set
Bees_dispersing 1] ]]   ; if leave with it 80% less bees disperse.

  ask patches with [Flowering = 1 and Infection_Status = 1 and Quarantine_Status = 0
and Degree_of_Infection >= Degree_of_Infection_threshold ]

  [ ifelse Bees_dispersing < 1 [if random 100 <= (Bees_dispersing * 100) [sprout-bees 1]]
[sprout-bees random (Bees_dispersing + 1)]      ]

   ask bees[

    ;pen-down

    right random 360

    forward random Bees_dispersal_distance

    ]

end



to rain_spread_infection    ; diffuse infection around existing pathces to simulate rain

  ask patches [if Degree_of_Infection > 10 and  Flowering = 1[

      set i (random 3) - 1       set j (random 3) - 1  ask patch-at i j [Infect_tree]]]


end



to Bees_infect_trees

ask bees[ if Flowering = 1 [Infect_tree]

  die]


end



to Emphasize_detection

  ask patches with [Detection_Status = 1 and Time_since_detection < 1 and not empty?
Tree_Species]  [sprout 1 [set color red set shape "circle" set size 5] ]

end



to define_detection_probability

  if Quarantine_Status = 1 and (Month = 1 and Week = 1 and Time_since_quarantine >=
24) or Time_since_quarantine = 4 [set Detection-probability item 3 Detection_values]
```

```
  if Month = Shoot_strike_detection_month [set Detection-probability item 1
Detection_values]

  if Pruning = 1 [set Detection-probability item 2 Detection_values]

  if Flowering = 1 [set Detection-probability item 0 Detection_values]


end




to detect_infection    ; Detection time between detection and application of quarantine is 1
week

  if Auto_management = true [

   ask patches with [Infection_Status = 1 and Detection_Status = 1 and
Time_since_detection = 1 ][ sprout-infection_detectives 1
Create_quarantine_ground_zero] ]

   ask patches with [Infection_Status = 1 and Detection_Status = 1] [set
Time_since_detection Time_since_detection + 1 ]  ; it takes 1 week to detection to be
confirmed.

      ; detection probability should vary depending on the time of the year and weather the
area is in quarantine or not.

   ask patches with [Infection_Status = 1 and Detection_Status = 0]
[define_detection_probability if (random 10000) / 100 < Detection-probability [set
Detection_Status 1 set pcolor red]]  ; this allows us to have less then 1% probability.


end


to check_region_infection_status

 if count patches with [Detection_Status = 1] > 0 [set Region_Infection_Status 1]

 if Region_Infection_Status = 1 [set Time_since_first_detection Time_since_first_detection
+ 1]


end


;*************************************************************

;************** Quarantine management ***************************

;*************************************************************


to Create_quarantine_manual  ; we want to create a cicrle around the user click.

;; place a agent where the user says to

  if (mouse-down?)
```

```
  [set x mouse-xcor set y mouse-ycor calculated_distances

  ask patches [ if (distancexy x y) <= Quarantine_radius and Tree_Maturity > 0 [set
Quarantine_Status 1 set Time_since_quarantine 1 if Detection_Status = 0 [set pcolor
4.5]]] ]

                  if Destruction_radius > 0 [ask patches    [ if (distancexy x y) <=
Destruction_radius and Tree_Maturity > 0  [Kill_tree] ]]  display

  ; [ask patches [ if (distancexy mouse-xcor mouse-ycor) < 10 [set pcolor green]]    ]


end


to Create_quarantine_ground_zero  ; we want to create a cicrle around the infection site

calculated_distances

ask infection_detectives [ ask patches in-radius Quarantine_radius [if Tree_Maturity > 0
[set Quarantine_Status 1 set Time_since_quarantine 1 if Detection_Status = 0 [set pcolor
4.5]]] ]   ; this creates the quarantine area

ifelse Destruction_radius = 0 [ask infection_detectives [die]] [ask infection_detectives [
ask patches in-radius Destruction_radius [if Tree_Maturity > 0 [Kill_tree]] die]]   ; this
creates ground zero


end


to Clean_quarantine

if Live_with_it = true [ask patches with [Quarantine_Status = 1]

  [ifelse Detection_Status = 1 and not empty? Tree_Species [set pcolor red]

  [if Tree_Species = "Apple" [set Quarantine_Status 0 set pcolor (item 4 item 0
Species_List_Flowering_season)]

   if Tree_Species = "Pear" [set Quarantine_Status 0 set pcolor (item 4 item 1
Species_List_Flowering_season)]

]]]


end



;*************************************************************

;************** Calculate costs *********************************

;*************************************************************


to Calculate_costs
```

```
if Live_with_it = false [ask patches with [Quarantine_Status = 1 and not empty? Tree_Species] [set Quarantine_cost_to_patch Quarantine_cost_to_patch + Weekly_quarantine_surveillance_cost]]

set Total_OCR_cost (sum [Cost_to_patch] of patches)

set Total_quarantine_cost (sum [Quarantine_cost_to_patch] of patches)

set Total_detections (count patches with [detection_status = 1])      ; calculate the number of cell where the disease was detected

set Total_destruction_area (count patches with [tree_species = "" and Quarantine_Status = 1 ]) * Cell_size ^ 2 / 10000   ; calcuate surfaces kills since start in ha

set Total_cost (Total_OCR_cost + Total_quarantine_cost)

if month = 2 and week = 1 [

set Economic_return_no_FB  (sum [Tree_potential_Yield] of patches with [not empty? Tree_Species])

;if Live_with_it = true

set Economic_return_FB  (sum [Tree_potential_Yield_FB] of patches with [Infection_Status = 1 and not empty? Tree_Species]) + sum [Tree_potential_Yield] of patches with [Infection_Status = 0 and not empty? Tree_Species]

set Total_revenue_loss Total_revenue_loss - Economic_return_FB + Economic_return_no_FB

  ]

end



;*************************************************************

;************** Export graphics *********************************

;*************************************************************

to do-plots

  set-current-plot "Phenology (bloom)"

  set-current-plot-pen "Pears"

  plot count patches with [Tree_species = "Pear" and Flowering = 1]

  set-current-plot-pen "Apples"

  plot count patches with [Tree_species = "Apple" and Flowering = 1]

end

to do_cost_plot

  set-current-plot "ORC_Costs_of_outbreak"
```

```
  set-current-plot-pen "Cost"
  plot Total_OCR_cost
end

to do_economic_return_plot
 if month = 2 and week = 1 [
 set-current-plot "Economic_return"
 set-current-plot-pen "Annual_Return_no_FB"
 plot Economic_return_no_FB
 set-current-plot-pen "Annual_Return_with_FB"
 plot Economic_return_FB]
end

to do_incusrion_management_cost_plot
 set-current-plot "Management_costs"
 set-current-plot-pen "Cost of incustion"
 plot Total_quarantine_cost
end



;*************************************************************************
;******************** Store output data in raster (asc)*****************
;*************************************************************************

to store-data
  ask patches [set grid-value (Infection_Status + Detection_Status * 10 + 100 *
Quarantine_Status)]
   ; write the dataset to ascii
   set output (word "D:/CUBA_netlogo/SMEScriptsCuba/NetLogo_outputs2/vic" ticks ".asc")
   ;set output (word "D:/CUBA_netlogo/SMEScriptsCuba/vic" ticks ".asc")
   gis:store-dataset gis:patch-dataset grid-value output
end

;*************************************************************************
******************
;****************************Active modelling component
**********************************.
```

```
;**************************************************************************
******************

to go
  if ticks >= 480 [stop]
  if Live_with_it = true [set Auto_management false]
  Clean_quarantine
  Calculate_Week_Month_and_Year
  Calculate_maturity
  Calculate_flowering
  if Month = 1 and Week = 1 [Calculate_yield]
  Grow_infection
  Creat_and_move_bees
  Bees_infect_trees
  detect_infection
  ;if Auto_management = false [ask turtles [die]]
  ask turtles [die]
  if Auto_management = false [Emphasize_detection]
  check_region_infection_status
  Calculate_costs
  do-plots do_cost_plot do_economic_return_plot do_incusrion_management_cost_plot
  if Export_output = true [store-data]
  tick
end
```