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Abstract

Diversification of the tree species composition of production forests is a frequently advocated strategy to increase resilience to pests and diseases, however there is a lack of a general framework to analyse the impact of economic and biological conditions on the optimal planting strategy in the presence of tree disease. To meet this need we use a novel bioeconomic model to quantitatively assess the effect of tree disease on the optimal planting proportion of two tree species. We find that diversifying the species composition can reduce the loss from disease even when the benefit from the resistant species is small. However this key result is sensitive to a pathogen's characteristics (probability of arrival, time of arrival, rate of spread of infection) and the costs (damage of the disease to the susceptible species and reduced benefit of planting the resistant species). This study provides an exemplar framework which can be used to help understand the effect of a pathogen on forest management strategies.

1 Introduction

Tree pest and pathogen outbreaks can have negative economic and environmental impacts especially when large areas of forest are affected (Pimentel et al., 2005; Ayres and Lombardero, 2000). Once a pest or pathogen has established there are relatively few treatments which help diseased trees to recover, therefore any reactive strategy

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tends to focus on controlling the outbreak (often this is preventing or reducing the spread to other forest areas). On the other hand, anticipatory (proactive) strategies have been proposed to reduce the initial susceptibility of forests to an outbreak, and/or to reduce the impact of disease on the trees once a pest or pathogen has arrived (Quine et al., 2015; Jactel et al., 2005, 2009; Wainhouse, 2004). In this study, a mathematical model is used to examine one such strategy, and in particular address the question: how does disease effect the optimal planting strategy for choices over alternative species?

The literature examining the effect of diversification of the tree species composition of forests on timber and non-timber outputs is ever expanding; however the range of ecological impacts are difficult to disentangle and explicitly define (Jactel et al., 2009). The type of forest and the objective(s) of the forest owner or social planner will influence the economic and ecological outcomes of diversifying. In this paper, the focus is narrowed by considering a plantation where the manager is interested in productivity of timber only. Plantation forests are commercially important since they contribute a large proportion of timber to the world markets. They often consist of a single species monoculture chosen for growth or other properties, but are potentially vulnerable to a pest or pathogen of that tree species. For example, over the last century eucalypts have been grown in non-native plantations in large areas of the southern hemisphere. Their fast growth rate, and separation from their natural enemies has made them an economically important species in South America, South Africa, and more recently South and East Asia (Wingfield et al., 2008). However, the increase in arrival of pests and pathogens, such as Cryphonectria canker caused by the fungus *Cryphonectria cubensis* (Wingfield, 2003), are beginning to have a negative affect (Wingfield et al., 2008). The European spruce bark beetle (*Ips typographus*), which is not currently believed to be in the UK, has been shown to have a greater effect on stands with higher proportions of spruce trees Wermelinger (2004). Due to the high proportion of Sitka spruce monocultures in the UK, a contingency plan (`http://www.forestry.gov.uk/pdf/Ips-typographus_contingency-plan.pdf/ $FILE/Ips-typographus_contingency-plan.pdf`) has been created in case the beetle is found.

With the arrival of novel pathogens, and their subsequent damage, increasing worldwide (Freer-Smith and Webber, 2015; Aukema et al., 2010), and with world trade continuing to provide fresh biosecurity challenges (Brasier, 2008), strategies to reduce their impact on plantations are of great importance. Species diversification is one such strategy. The main argument for diversifying the tree species composition of production forests is the "insurance hypothesis" since, at the forest level, planting more than one species spreads the risk (Loreau et al., 2001; Pautasso et al., 2005), by reducing the initial susceptibility, and/or reducing the impact if a pest/pathogen does arrive particularly as many are species or genus specific in their impact. Modelling in Sweden has shown that there is a reduction in the risk of damage from *Heterobasidion annosum* when spruce stands are mixed with pine (Thor et al., 2005), moreover transmission rates of *Armillaria* spp. were found to reduce with increased tree diversity by (Gerlach et al., 1997). Haas et al. (2011) used field data and Bayesian hierarchical models to show that sites with higher species diversity have a reduced disease risk of *Phytophthora ramorum* in California, and

the experiments of Hantsch et al. (2014) showed that local tree diversity can decrease the level of fungal pathogen infestations of *Tilia cordata* and *Quercus petraea*. More recently, Guyot et al. (2016) sampled a network of forest plots spanning several countries, and showed a positive relationship between tree species richness and resistance to insect pests. They argued that these "findings confirm the greater potential of mixed forests to face future biotic disturbances in a changing world" (Guyot et al., 2016).

In this paper, we explore how the optimal planting strategy of two tree species, which minimises the present value of economic loss, changes due to disease. To do this, we create a bio-economic model, where there is a probability of a pathogen arriving and reducing the timber value of one of the species planted. This model is not based on a specific host-pathogen system, rather our general approach facilitates a better understanding of the qualitative effects that pathogen characteristics can have on the optimal planting strategy. Moreover, the model can be calibrated for specific forest types and specific diseases.

It is assumed that a forest manager has the option of planting two tree species (species A or species B or both), over a fixed rotation period (note: we consider the effects on optimal rotation for a single species in Macpherson et al. (2015, 2016)). In the first part of this paper, it is assumed that the difference in economic returns between both species, is realised at the end of the rotation, where the timber from un-infected species A returns a higher profit than the timber from species B. This may occur through the trees of species A growing faster than trees of species B, or species A having a higher timber price than species B. Initially the cost of establishment, and cost and benefit of the silvicultural operations used throughout the rotation, are assumed to be the same for both species (this assumption is relaxed later). Under these conditions (without disease), the optimal planting strategy will be to plant species A only (since species B has a smaller timber benefit). However, species A is susceptible to a new pathogen which will lower the timber benefit, whilst species B remains resistant to the pathogen. If the pathogen, and hence disease, arrives during the rotation and species A has been planted, then the forest manager is faced with a loss through reduced benefit of infected trees. If species B has been planted then a loss from switching from species A to species B will be realised independent of the arrival of the pathogen. If both species are planted, then a combination of losses may occur. The optimal planting strategy, more specifically how much of each species to plant, is the strategy which minimises the expected economic loss.

The mathematical framework for this optimisation problem consists of an objective function that calculates the present value loss of planting both species, when compared with the 'ideal situation' of a monoculture of trees of species A remaining un-infected. The potential loss due to planting trees of species A will depend on a number of factors: the probability of arrival of the pathogen and occurrence of disease; when the pathogen arrives within the rotation; how fast the pathogen spreads throughout the forest; and the effect of the disease on the timber benefit (through increased harvesting costs, reduced growth or quality of the timber). Thus the objective function depends on the area of infected trees (of species A) at the end of the rotation which is described by a Susceptible-Infected epidemiological compartmental model.

How fast the pathogen spreads throughout the forest will largely depend on the contact rate (for example, of spores), the probability that contact is with a tree of species A, and the probability that the tree is susceptible to disease. This formulation will depend on the spatial arrangement of the trees within the forest, since the probability that contact is made with a tree of species A, will likely be different if species A is planted in a monoculture block, or in an intimate mixture with species B. Whilst we do not explicitly define space in the model, we demonstrate how the pathogen transmission term is constructed for both a monoculture and an intimately mixed forest. Exploring both these cases is important, since the majority of the existing evidence reported above shows a positive effect of tree species diversity (on reducing the effect of disease) when the species in the forest plots are intimately mixed.

The final part of this paper examines the case when the timber benefit from species A and B are the same, but the cost of establishment of species B is greater than species A. This can represent the planting of two genotypes within a species, where one is higher yielding but more susceptible to disease, and the second is lower yielding but more resistant. Often greater resistance is at a cost of lower yield, but another likely scenario is that there is a higher cost of planting stock of a more resistant species or genotype. While the combination of these scenarios, where the more resistant species B has both a higher cost of planting stock and lower timber value, is not explicitly investigated here, the outcome of such a scenario can be deduced using the other qualitative results that are shown.

The three research questions that this paper addresses are: (1) what is the optimal planting strategy when species A returns a higher timber benefit than species B, but species A is susceptible to a new disease, whereas species B is not; (2) how do different bio-economic conditions alter the optimal planting strategy; and (3) how does the optimal planting strategy change when the timber value of uninfected trees of species A and species B is the same, but the cost of planting species B is greater than species A? Examining these questions for a range of bio-economic parameter sets allows the optimal planting strategies to be found for different 'types' of diseases.

The layout of the paper is as follows. A short literature review on the use of bioeconomic models used to analyse the effect of pests and pathogens on forest management strategies is given in Section 2. The economic and epidemiological components of the model are derived in Section 3, and the results are given in Section 4. In Section 5 the model is re-arranged so that the cost of establishment is different between both species (and the timber benefit is the same). A discussion in Section 6 is followed by a brief conclusion of the key results found in this paper in Section 7 .

2 Bioeconomic modelling of the effect of pests and pathogens on forest management strategies

Changing forest strategies in response to a pest or pathogen threat often has major economic consequences (Wainhouse, 2004). For example, there will likely be a cost of changing the strategy but, if successful, after a pest or pathogen arrives, the forest output (timber and/or non-timber) may be maintained at a higher level, and thus there will be a benefit (compared with 'doing nothing different'). The decision maker therefore has to weigh-up the costs and benefits of changing the strategy, with the risk of the pest or pathogens arriving, and their predicted effect on the forest.

Mathematical modelling has been used to examine these effects 'in silico'. Models can help to analyse and compare the effect of a pest or pathogen on the relative success of alternative management strategies under different economic and biological conditions. This section highlights some of the bioeconomic models that have been developed to analyse: forest management strategies in the presence of a pest or pathogen; invasion–specific management strategies such as surveillance or control; and the effect of mixed species composition in the presence of other abiotic and biotic risks. (Note, that the difference between the first and second point is that the first assumes that a *change* in a management strategy occurs (i.e. these strategies occur when there is no risk of an incursion), whereas 'invasion specific' strategies are deployed specifically to target management of a pest or pathogen risk.)

There are many forest management strategies, whose success may be affected by a pest or pathogen incursion. Jactel et al. (2009) highlighted the effect of a range of forestry practices on biotic and abiotic hazards in a synthesis. Strategies identified to affect the likelihood of an outbreak, and susceptibility of forests to pathogens and pests, included thinning and pruning, tree species composition and density of plantings. Using knowledge from practitioners and experts Quine et al. (2015) recommended 33 strategies as potentially relevant to combat a single disease, Dothistroma Needle Blight, in just one country, the UK. Bioeconomic models, can be used to aid the exploration of the effect that disease can have on this multiplicity of alternative strategies that would be very time consuming to individually test empirically. However, despite their benefits, bioeconomic models are still underutilised in examining how the optimality of strategies changes in the presence of disease. An example of the insight bioeconomic models can give has been demonstrated for *Heterobasidion annosum*, an economically important pathogen on conifers. *H. annosum* is endemic in Europe and spreads through spores which colonise on freshly cut conifer stumps, causing timber deterioration and thus a reduction in the commercial value. Several models have been used to simulate the spread of the pathogen, at a tree and forest level, and the subsequent timber decay (Seifert, 2007; Pukkala et al., 2005). Moreover, bioeconomic models (which combine both the pathogen dynamics and the economics), have been used to examine the effect of management strategies, such as thinning and chemical stump treatment (which can be applied after tree cutting like thinning), on the reduction

of the pathogen spread and economic damage (Wang et al., 2015; Thor et al., 2006; Möykkynen and Miina, 2002). Bioeconomic models have also been used to assess the effect of a pathogen on the optimal rotation length of forests (Macpherson et al., 2015, 2016). The authors adapted a classical Faustmann model so that it depended on the rate of pathogen spread (through a Susceptible-Infected epidemiological model). This optimal control framework showed that the optimal rotation length (the forest age at which net present value of the forest is maximised) of a plantation forest is generally shortened when the damage from disease reduces the timber benefit (Macpherson et al., 2015). When a forest manager considers both the timber and non-timber benefits (included in the model through a green payment), and the damage from disease reduces the timber benefit only, the optimal rotation length is restored; when the damage reduces both the timber and the non-timber benefit, the optimal rotation length is reduced (Macpherson et al., 2016).

Other bioeconomic models which address tree disease management strategies that aim to reduce the impact of an invasion, focus on questions about optimal surveillance (Epanchin-Niell et al., 2013), and control (Thompson et al., 2016; Mbah and Gilligan, 2010, 2011; Sims et al., 2010; Thor et al., 2006). Epanchin-Niell et al. (2013) created a mechanistic bioeconomic model to examine the cost-efficiency of a trap-based pest surveillance program for multiple, simultaneous novel invasions at a landscape scale. In their model, multiple pests arrive, spread and cause damages to urban and plantation forests, but upon detection eradication can be attempted at a cost (dependent on the area of the invasive species population). Earlier detection can lead to a greater chance of eradication, and reduction in the future damage and costs. The authors use a case study of wood borer and bark beetles in New Zealand to parameterise their model, and found the optimal surveillance program, which minimised the total net present value of expected future costs (surveillance, invasion damages, and control costs), required very high investment in surveillance (about 10,000 traps in each year of the 30-year surveillance program). This strategy reduced the costs by 39% compared with no surveillance (Epanchin-Niell et al., 2013); moreover in general they found that, the cost, even at a low level of surveillance, was offset by the economic benefits of surveillance. Another example of a bioeconomic model informing on control is Sims et al. (2010). The authors used a bioeconomic model to examine passive, localised, and centralised timber harvesting strategies to maximise a household utility function (which included both produced goods, like timber, and the quality of the forest, such as recreation, amenity values, and ecosystem services) of a lodgepole pine forest with a mountain pine beetle (MPB) outbreak. The baseline strategy of passive management involves no control or harvesting, whereas the localised and centralised strategies involve the forest manager harvesting adult and salvage (dead) trees dependent on the household preference, stock of trees and the MPB population. The difference between these strategies is that localised management optimally treats the outbreak as exogenous (the harvest decisions respond to an outbreak and take future outbreaks as given), whereas centralised management optimally recognises the endogenous nature of MPB (the harvest decisions consider future outbreaks and future tree mortality). The authors found that centralised forest management substantially reduced the size of the

outbreaks and risk of future outbreaks when compared with passive and localized management (which actually increased the risk and severity of epidemics).

Models have been used to assess the general risk of a catastrophic event on mixed species forests (Griess and Knoke, 2013; Neuner et al., 2013; Roessiger et al., 2013; Knoke et al., 2005; Knoke and Seifert, 2008). The majority of these papers use portfolio theory to establish the expected financial return and risk of investment in a forest. For example, Knoke et al. (2005) evaluated mixed *vs.* single species management of Norway spruce and European beech (for a mixed species forest, the species were planted in separate blocks). The expected financial return (net present value of all future net revenue flows) and the risk of the investment was calculated by using Monte Carlo simulations. Planting a mixed forest reduced the profitability due to the lower value of beech compared with Norway spruce). However, increasing the risk of planting spruce (through an increased risk of the occurrence of a natural hazard), reduced the return from spruce, made planting a mixture more profitable and reduced the overall risk of the portfolio (Knoke et al., 2005). In a follow-up study, Knoke and Seifert (2008) used a bioeconomic model to examine the financial return and risk of two different forest types of the same two species – a pure beech forest and a mixture (planted in smaller rectangular blocks of 25×40 m). The authors used data from existing studies on forest productivity, timber quality and resistance to the hazard (a polynomial survival probability function, based on storm damage data) to inform the model. Again, Monte Carlo simulations, under site conditions and risks typical of southern Germany, were used to simulate the financial risk and return for varying proportions of species mixtures. The main results showed that a mixture decreased the financial risk and increased the return when all the tested ecological factors were included (Knoke and Seifert, 2008).

The difference between a pest or pathogen outbreak and other abiotic risks, such as fire or storms, can be significant (for example, the time scale over which the event occurs, the symptoms and whether it leaves salvageable timber), and therefore we argue that a separate study is required to examine the effect of a pathogen on the success of forest management strategies differing in tree species diversification. Moreover, a lot of previous studies (Thompson et al., 2016; Mbah and Gilligan, 2011; Sims et al., 2010; Thor et al., 2006) concentrate on specific host-pathogen systems which can be necessary when addressing strategies to combat single pathogen species. However, much benefit can be gained by developing and analysing qualitative models that highlight the interaction of a general pest or pathogen with the management strategy, and allow the sensitivity to biological and economic parameters to be investigated. This also has the advantage of identifying which parameters are important when considering a specific host-pathogen system, and so help prioritise data gathering.

3 Model framework

First, we list some terminology used throughout this paper. The total area of the forest managed is referred to as the 'plot '. A 'monoculture' refers to a planting strategy where only one species is planted in the plot, whilst a 'mixture' refers to the case when two species are planted intimately, with no spatial aggregation of the trees of each species throughout the plot.

In this section, we consider a case where species A returns a higher timber benefit than species B, but species A is susceptible to a new disease, whereas species B is not. The model is formulated in two parts. The first derives the minimisation problem for two scenarios (Section 3.1), and the second creates a Susceptible-Infected (SI) epidemiological model (Section 3.2).

3.1 Economic model

This section is divided into three parts. The first assumes that the forest manager is restricted to planting a monoculture; thus the question becomes *which* species should be planted (Section (3.1.1). The second part creates the minimisation problem for planting a mixture of both species in the plot (Section 3.1.2). The cost of establishment is initially assumed to be the same for both species. Similarly the silvicultural practices, which are implemented throughout the rotation, are the same and obtain the same results. This Section concludes by showing how the timber volume at the end of the rotation is derived (Section 3.1.3).

3.1.1 Planting a single species monoculture

First, assume that the forest manager can only plant a monoculture of species A *or* species B. The net benefit of timber from a plot of area L is a product of the price of timber per cubic metre, p, and the timber volume at the end of the rotation, $Lf(T)$. The difference between planting the two species is realised through the timber benefit obtained at the end of the rotation (the rotation length is fixed to $T = 40$ years for both species). If disease arrives, and species A has been planted, then the benefit from the timber is reduced through either the timber quality (for example, due to staining or rot causing loss of mechanical integrity), slower timber growth, greater costs of harvesting, or through lower price due to market saturation with that species. To include this in the model, the term $\tilde{L}(T)$ is used to represent the effect of disease on timber from species A at the end of the rotation (explained in detail in Section 3.2).

If species A is planted and the disease arrives then there is a loss (when compared with the net benefit obtained from uninfected trees of species A). This can be written as

$$pf(T)Le^{-rT} - pf(T)\tilde{L}(T)e^{-rT} \tag{1}$$

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where the future benefit is discounted, with rate r, to give the present value. Alternatively, if species B is planted, a reduced timber benefit occurs through a slower growth rate or reduced timber value (when compared with the net benefit from uninfected trees of species A). This loss is

$$pf(T)Le^{-rT} - R_P pf(T)Le^{-rT} \tag{2}$$

where $R_P \in [0,1]$ is the value of timber from species B relative to timber from uninfected trees of species A.

Factorising $pf(T)e^{-rT}$ from Equations (1) and (2) the loss from disease (through planting species A) is

$$\Delta_A = L - \tilde{L}(T), \tag{3}$$

and the loss from switching to species B is

$$\Delta_B = L(1 - R_P). \tag{4}$$

The forest manager chooses which species to plant (the 'optimal planting strategy'), by finding which species produces the minimum loss, namely

$$J = \min[\Delta_A, \Delta_B]. \tag{5}$$

Uncertainty in the arrival of the pathogen will affect the expected loss due to disease. The probability that a pathogen will arrive at the plot during the rotation is $P \in [0,1]$ (for the purpose of this paper, the forest manager is assumed to have full knowledge of this probability and to be risk neutral). The expected loss from planting species A will then be

$$E[\Delta_A] = P\left(L - \tilde{L}(T)\right) \tag{6}$$

since the loss when a pathogen does not arrive, with probability $1 - P$, is zero. The loss of planting species B remains unchanged (Equation (4)). The optimal planting strategy in Equation (5) is therefore updated to be

$$J = \min\left[E[\Delta_A], \Delta_B\right]. \tag{7}$$

3.1.2 Planting a two species mixture

In this section, the minimisation problem for planting a mixture of two species is given. The area of the plot remains fixed at L hectares, and the parameter $\delta \in [0,1]$ controls the fraction of species B that is planted in the mixture. Therefore, the area occupied by trees of species A is $L_A = (1 - \delta)L$, and the area occupied by trees of species B is $L_B = \delta L$. The expected loss from planting a mixture, compared with planting only species A and

the trees remaining uninfected, is given by

$$Ppf(t)\left(L - (\tilde{L}(T,\delta) + R_P L_B)\right)e^{-rT} + (1-P)pf(t)\left(L - (L_A + R_P L_B)\right)e^{-rT} \tag{8}$$

which can be simplified to

$$E[\Delta_{AB}] = P\left(L_A - \tilde{L}(T,\delta)\right) + L_B\left(1 - R_P\right). \tag{9}$$

The pathogen arrives and disease occurs with probability P which reduces the net benefit of trees from species A. The term $\tilde{L}(T,\delta)$ is again used to represent the effect of the disease on the area of species A (more on this in Section 3.2). If any of species B is planted, a loss will always be incurred when compared with planting only species A and the trees remaining uninfected. The optimal planting strategy is found by minimising $E[\Delta_{AB}]$ in Equation (9) subject to δ.

3.1.3   Timber volume

The net benefit at the end of the rotation is dependent on the volume of timber, which in this paper is $f(T) = 579.9$ m^3 ha^{-1}. This value is taken from the Forest Yield model which is developed by the UK government agency Forest Research, and used to estimate the average timber volume per tree and density of trees (number per hectare) over time (Forestry Commission, 2016). Yield class 14 of *Picea sitchensis* (Sitka spruce) is chosen as species A since it is the dominant conifer species grown in Scotland and elsewhere in the British uplands (Forestry Commission, 2011). The timber volume for species B is not fitted since parameter R_P allows the timber volume growth of species B (and/or the price of timber of species B) to be scaled relative to species A. This permits flexibility within the model since analysis of sensitivity to R_P can be explored.

3.2   Epidemiological System

$P \in [0,1]$ is the probability that a specific pathogen is introduced to the plot. If there is a disease outbreak then a SI epidemiological model shows how the area occupied by infected trees of species A changes throughout time. The area occupied by species A, L_A, consists of the area of trees (of species A) that are susceptible to disease (i.e. not infected), $S_A(t)$, and those that are infected, $I_A(t)$, at time t (so $L_A = S_A(t) + I_A(t)$). All trees of species A are initially susceptible to infection, giving $S_A(0) = L_A$. If there is a disease outbreak, then this occurs via some primary infection rate, ϵ (through, for example, the arrival of inoculum on the wind or insect vectors). In this model, there is no interaction between the probability of the pathogen arrival, P, and the time at which the pathogen arrives, ϵ. Typically, models have one parameter controlling the probability of the disease arriving at a certain time within the rotation. Here, parameters P and ϵ keep the probability of arrival and time of arrival separate, allowing additional sensitivity analysis. Once the pathogen has arrived in

the plot, there is a secondary infection rate which represents the spread of infection throughout the forest.

To illustrate the secondary infection term (henceforth 'transmission') we start with the assumption that the rate at which a single infected tree 'converts' a susceptible tree to an infected tree is

$$g_1(L) \times g_2 \left(\frac{L_A}{L} \right) \times g_3 \left(\frac{S_A}{L_A} \right) \tag{10}$$

where $g_1(L)$ is the contact rate between the infected tree and any other tree in the plot, $g_2(L_A/L)$ is the probability that the contact is with a tree of species A, and $g_3(S_A/L_A)$ is the probability that the contact is with a susceptible tree. The contact can be thought of as occurring through the spores spreading throughout the plot, or spread by the growth of the pathogen through the tree root network. Contact may occur at different spatial scales dependent on the dispersal range – but this will be proportional to the total area of the plot, L (not the area of species A). The probability that the contact is with a tree of species A will depend on how the tree species are arranged. For example, if the species are arranged as a monoculture, then the contact will always be with species A. However, if the two species are mixed intimately with no spatial correlations, then the probability of contact with a tree of species A will be proportional to the number (area) of trees of species A planted within the plot. This gives

$$g_2 \left(\frac{L_A}{L} \right) = \begin{cases} 1 & \text{if a monoculture} \\ \frac{L_A}{L} & \text{if a mixture.} \end{cases} \tag{11}$$

Finally, the probability that contact is made with a susceptible tree is proportional to the number (area) of susceptible trees of species A (S_A/L_A).

Therefore, for a mixture of two species, the rate of 'converting' a susceptible area to an infected area using Equation (10) is

$$\left(g_1(L) \times \frac{L_A}{L} \times \frac{S_A}{L_A} \right) \times I_A = \beta S_A I_A \tag{12}$$

since $g_2(L_A/L) = L_A/L$ from Equation (11) and β is the transmission rate. The rate of change of area of infected trees over time is

$$\frac{dI_A}{dt} = \beta \left(L_A - I_A(t) \right) \left(I_A + \epsilon \right) \tag{13}$$

since $S_A(t) = L_A - I_A(t)$. (This is the same as classical 'density-dependent' transmission, where the force of infection increases with the area of infected trees – since the contact rate with species A is increased.) Using the initial conditions $(I_A(0) = 0)$ this is solved to give

$$I_A(t) = \frac{\epsilon \left(e^{(L_A+\epsilon)\beta t} - 1 \right)}{\frac{\epsilon}{L_A} e^{(L_A+\epsilon)\beta t} + 1}. \tag{14}$$

When a monoculture of species A is planted, Equation (10) can be used to derive the rate of change of area of infected trees. The indices are now dropped since only species A is planted in area L, and this gives

$$\frac{dI}{dt} = \beta(L - I(t))(I(t) + \epsilon) \tag{15}$$

where $g_2(L/L) = 1$ and $g_3(S/L) = S(t)/L$. The area of infected trees can then be found using the initial condition $I(0) = 0$, which gives

$$I(t) = \frac{\epsilon \left(e^{(L+\epsilon)\beta t} - 1\right)}{\frac{\epsilon}{L}e^{(L+\epsilon)\beta t} + 1}. \tag{16}$$

Figure 1 (a) shows the area of infected trees over time for Equation (16), for three primary infection rates, ϵ, used in this study.

Under the restrictions of planting a monoculture (Section 3.1.1), the pathogen transmission occurs across the whole plot of fixed size $L_A = L$ (when species A is planted). However, when a mixture is considered (Section 3.1.2), the area of species A is changed to find the optimal δ (the fraction of area occupied by species B). Varying the area of species A will clearly have an economic impact (through an increased loss from planting more species B, but also through reducing the speed that infection progresses through the population of species A due to the reduced probability of pathogen contact between infected trees of species A and conspecific trees (Equation (11)). This can be seen by finding the time for a fraction of species A (θ) to become infected, which is

$$t_\theta = \frac{1}{\beta(L_A + \epsilon)} \ln\left(\frac{\theta L_A + \epsilon}{\epsilon(1 - \theta)}\right). \tag{17}$$

Figure 1 (b) shows the time taken for 95% of species A to become infected ($\theta = 0.95$) against the proportion of species A planted ($1 - \delta$) for three primary infection rates. Increasing the area of species A (increasing $1 - \delta$, or increasing the probability of pathogen contact between trees of species A, Equation (11)), decreases the time taken for the disease to spread throughout the population of species A. For example, a pathogen which arrives early in the rotation will take approximately 44 years for 95% of 1 ha of species A ($1 - \delta = 1$) to become infected, but the time taken to infect 95% of the occupied area is more than quadrupled when the area of species A is reduced to 0.1 ha ($1 - \delta = 0.1$; the black curve in Figure 1 (b)). This interaction will clearly have important implications when finding the optimal planting strategy for two tree species.

Disease affects the timber benefit obtained from infected trees. The function $\tilde{L}(t, \delta) = S_A(t) + \rho I_A(t)$ (or $\tilde{L}(t) = S(t) + \rho I(t)$ for a monoculture of species A) captures the effect of disease on the area of trees of species A at time t. The parameter $\rho \in [0, 1]$ measures the effect of the disease on the timber value, so that when $\rho = 0$, the timber from diseased trees has no value, alternatively when $\rho = 1$ there is no difference in value between timber from infected and uninfected trees. This function is used to calculate the expected loss from planting species A, which therefore depends on the rates of primary infection (ϵ) and secondary transmission

within species A (β), and the effect of the disease on the timber value (ρ). For the main part of this paper we set $\rho = 0$, so that infected trees have no timber value. This assumption is relaxed in Section 4.3 where analysis of sensitivity to ρ is explored.

4   Results

The first application of this model is to find the optimal planting strategy when species A returns a higher timber benefit than species B, but species A is susceptible to a new disease, whereas species B is not. In this section the optimal planting strategy is shown under these conditions when the forest manager plants a monoculture (Section 4.1), and then a mixture (Section 4.2). Analysis of sensitivity to ρ is then carried out (Section 4.3).

4.1   Monoculture

The optimal planting strategy is found by determining which species gives the minimum loss (Equation (5)) when $\rho = 0$. The top row of Figure 2 shows the optimal planting strategy against the transmission rate (β; x-axis) and timber value of species B relative to species A (R_P; y-axis), for different primary infection rates (ϵ). As the transmission rate is increased, there is an increase in the R_P range where it is optimal to plant trees of species B, since the loss from disease, Δ_A, is increased (Equation (3)), and the cost of planting species B remains the same (Equation (4)). Once the transmission rate reaches a level such that all trees in the plot become infected by the end of the rotation ($I(T) = L$), it will always be optimal to plant species B independent of the difference in timber value. As the primary infection rate increases, the range of R_P where it is optimal to plant species B increases for smaller transmission rates (shown by the boundary of the white parameter space moving towards the left with the increasing values of epsilon in Figure 2). Again, this is due to the increase in the economic loss due to the disease.

When there is a lower probability of pathogen arrival, the expected loss from planting species A decreases. This is seen in Figure 2 where the region in the parameter space in which it is optimal to plant species A (shaded black) increases as P decreases between the rows. Increasing the primary infection rate, ϵ, increases the parameter space in which it is optimal for species B to be planted across all of the values of P in Figure 2. However, this effect diminishes with lower values of P so it can still be optimal to plant species A for high pathogen transmission values (especially for low values of R_P). This occurs because the probability of economic loss due to the disease being realised is reduced with a lower probability of pathogen arrival.

Finding an equation for the boundary between the two planting strategies is achieved by setting both losses in Equation (7) as equal. This gives the relative timber value of species B (R_P^B) in terms of the pathogen’s

transmission characteristics. It can be expressed as

$$R_P^B = 1 - P\frac{I(T)}{L} \tag{18}$$

since $\tilde{L}(T) = L - I(T)$ when $\rho = 0$. As the pathogen transmission rate increases, then $R_P^B \to 1 - P$ since $I(T) \to L$. This is shown in Figure 2 as the R_P value of the boundary between the two species tends to $1 - P$. Once the primary infection rate and/or the transmission is large enough for the disease to spread throughout the whole plot by the end of the rotation ($I(T) = L$), the optimal planting strategy is predominantly determined by the probability of arrival (P) and the timber value of species B (relative to that of species A; R_P).

4.2 Mixture

The optimal planting strategy for two species is found by minimising the loss due to planting a mixture relative to a monoculture of the species providing the largest benefit (with the zero probability of pathogen arrival). This can be found by differentiating Equation (9) with respect to δ, which gives CHECK THIS

$$\frac{dE[\Delta_{AB}]}{d\delta} = P\frac{d\tilde{L}}{d\delta} - L\left(P + R_P - 1\right)) \tag{19}$$

where

$$\frac{d\tilde{L}}{d\delta} = -L - \frac{(\rho - 1)L\epsilon\beta Te^{((1-\delta)L+\epsilon)\beta T}\left(\frac{2\epsilon}{(1-\delta)L}e^{((1-\delta)L+\epsilon)\beta T} + 1 + \frac{1}{(1-\delta)L}\right)}{\left(\frac{\epsilon}{(1-\delta)L}e^{((1-\delta)L+\epsilon)\beta T} + 1\right)^2} \tag{20}$$

when $\rho = 0$. Unfortunately, δ cannot be found explicitly from Equation (20), however we can proceed using numerical optimisation.

The optimal planting strategy is plotted in Figure 3 against the transmission rate (β) and the timber value of species B relative to that of species A (R_P). The greyscale gives the optimal planting proportion of species B (δ): it is optimal to plant a monoculture of only species A in the black region of the parameter space and of species B in the white region, and the grey region indicates the conditions under which it is optimal to plant a mixture of the two species.

When the disease arrives late in the rotation (small primary infection rate, ϵ, left-hand column in Figure 3), it will always be optimal to plant a proportion of species A, and for a large region of the parameter space it is optimal to plant a mixture. A lower probability of disease arrival increases the region of the parameter space where it is optimal to plant only species A (black). As the primary infection rate increases (centre and right-hand columns of Figure 3), the region in the parameter space where it is optimal to plant species A (either as a monoculture or in a mixture) decreases, and a region where it is optimal to plant a monoculture of species B emerges (white). Again, this occurs because the loss due to disease is increased as the primary infection rate

(and/or the transmission rate) increases, whereas planting a higher proportion of species B reduces the overall loss in Equation (9).

It is interesting that when comparing Figure 3 with the planting strategy boundary between the two species when planting monocultures (the white line given by Equation (18) shown in Figure 2), the region where it is optimal to plant a mixture (grey) extends into the parameter space where it was optimal to plant a monoculture of species B (to the right of R_P^B), much more than it does into the parameter space where it was optimal to plant a monoculture of species A (to the left of R_P^B). Moreover, a region where it is optimal to plant a mixture for very small values of R_P and medium transmission emerges.

To explain this behaviour, the optimal planting strategy is explored in further detail for the case where $P = 0.75$ and $\epsilon = 0.0175$ in Figure 4. The expected loss due to planting a mixture relative to a monoculture of species A in Equation (9) is shown as the black lines in Figure 4 (b) and the proportion of species A that is infected by the end of the rotation is shown as the grey lines when they are plotted against the optimal planting proportion of species B in the mixture (δ) for the four points highlighted in Figure 4 (a). At point 1, pathogen transmission is slow and only a small proportion of species A is infected by the end of the rotation (top plot in Figure 4 (b)). The expected loss from planting a monoculture of species A is therefore small (relative to the loss from planting any trees of species B) and a monoculture of species A is optimal. Decreasing the probability of pathogen arrival doesn't affect the optimality of this solution since it will act to reduce the expected loss from disease (Figure 3).

As the transmission rate increases, the expected loss due to disease from planting species A increases (and the loss from planting species B stays the same). It therefore becomes optimal to plant a mixture of both species since, (i) timber from species B has a higher value than infected timber from species A (timber from infected trees is assumed to be worth nothing in this scenario ($\rho = 0$)), and (ii) planting species B reduces the rate of transmission to uninfected trees of species A. To expand on the second reason: as the area occupied by the trees of species A in the intimate mixture with species B decreases the rate of contact decreases (Equation (11) and Figure 1 (b)), and so more uninfected – and higher value – trees of species A are available by the end of the rotation. The second plot in Figure 4 (b) shows that an increasing proportion of species B in the mixture (δ) steadily decreases the proportion of infected trees of species A (grey), and initially decreases the expected loss (black) from planting a mixture due to the higher proportion of trees of species B that remain uninfected due to the effects of mixing with species B. However, once the optimal δ is achieved (at $\delta \approx 0.4$), the expected loss increases since the loss from planting more species B is greater than the benefit from reduced spread of infection to trees of species A. This provides the reason why it may be optimal to plant a mixture despite the very low values assumed in this scenario for timber from species B (in the region of the parameter space below the white R_P^B boundary in Figure 4 (a) where for a monoculture forest it would be optimal to plant only species A).

As the transmission rate is increased further, the spread of infection through the population of species A

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occurs faster. A region of the parameter space where it is optimal to plant a high proportion of species B in the mixture emerges for high values of timber of species B (R_P). The optimal planting proportion changes to a mixture as R_P is decreased (point 3), since planting a mixture reduces the spread of infection throughout species A (due to reduced probability of contact with species A, Equation 11)), but only when the area occupied by trees of species A is very small (due to a higher transmission rate in this scenario). This can be seen for points 3 and 4 in Figure 4 (b) where all the trees of species A are infected by the end of the rotation (grey line) when the proportion of the area occupied by trees of species A is approximately greater than 0.5 ($\delta < 0.5$). When the proportion of the area occupied by trees of species A is smaller than this ($\delta > 0.5$), there is a reduction in transmission and thus in the proportion of the population of species A that is infected. Decreasing R_P further means that the loss from planting species B is now high, and thus there is a reduced benefit due to planting a mixture, and it therefore becomes optimal to plant a monoculture of species A since there is a probability that the loss from disease may not be realised (Equation (9). This region is denoted by point 4 in Figure 4 (a), and it can be seen that the upper boundary of this (black) region approaches the boundary $R_P^B = 1 - P$ of the region of the parameter space where for a monoculture forest it would be optimal to plant only species A (white curve). In fact, it will be optimal to plant a monoculture of species A when

$$E[\Delta_A] < E[\Delta_{AB}]. \tag{21}$$

Re-arranging this gives

$$R_P^B = 1 - P\frac{I_A(T) - I(T)}{L - L_A}, \tag{22}$$

and when $\beta \to \infty$ then $R_P^B \to 1 - P$ as before. This means that the probability of disease arrival and the timber value of species B relative to species A are both key factors in determining the optimal planting strategy when the infection will spread throughout the area of forest before the end of the rotation (if it arrives).

4.3 Sensitivity to the revenue from the timber of infected trees relative to uninfected trees

In the previous section the infected timber from species A was assumed to have no value ($\rho = 0$). In this section the sensitivity of the optimal planting strategy to changes in ρ are qualitatively examined in Figure 5 which shows the optimal planting strategy when the values of ρ varied between the columns, and values of P varied between the rows (the primary infection rate is fixed). When the pathogen is certain to arrive (top row in Figure 5), decreasing ρ reduces the parameter space in which it is optimal to plant a monoculture of species A. This is not surprising since the loss from disease increases as ρ decreases, and therefore a greater proportion of species B is required to offset the loss (by reducing the spread of infection between trees of species A). As the

probability of pathogen arrival (P) is decreased, the region in the parameter space where is it optimal to plant only species A increases (independently of the value of ρ).

When the primary infection rate (ϵ) is increased (results are not shown here), the optimal planting strategy's sensitivity to ρ remains qualitatively similar to the results shown in Figure 5: decreasing the value of infected timber (ρ) reduces the region in the parameter space where it is optimal to plant only species A. However, increasing ϵ decreases the region in the parameter space where it is optimal to plant a mixture, and a region emerges where it is optimal to plant a monoculture of species B (this is similar to the behaviour in Figure 3 when ϵ is increased).

When the primary infection and/or transmission rate are large enough so that the disease will spread through all the trees of species A by the end of the rotation, whether it is optimal to plant only species A depends on the probability of pathogen arrival (P), the timber value of species B (R_P), and the value of infected timber (ρ). The boundary of the region in the parameter space in which it is optimal to plant only species A can be found using Equation (21) to give

$$R_P^B = 1 - \frac{P}{L_B} \left(L_B - (\tilde{L}(T) - \tilde{L}(T, \delta)) \right). \tag{23}$$

This shows that, the white boundary curve in Figure 5 is dependent on ρ (since there will always be some benefit of timber from infected trees when $\rho \neq 0$). Reducing the timber benefit from infected trees, will increase the value of R_P^B required for only species A to be planted.

5   Difference in cost of establishment

In the previous section, trees of species B are assumed to have the same establishment cost, but reduced timber value, compared with trees of species A. The case where the cost of establishment is different between the two species is now examined (when $\rho = 0$). Assume that $R_P = 1$, which means that the relative price of timber from species B is the same as species A, and introduce a coefficient R_C which scales the cost of establishment of species B compared with species A (where $R_C \geq 1$). The cost of establishment for species A and species B is cL_A and $R_C c L_B$ respectively, where c is the cost per hectare of planting species A (Table 1).

First examine the case under the constraint of planting a monoculture. The loss from planting species B is

$$\Delta'_B = cL(R_C - 1), \tag{24}$$

and the loss due to disease from planting species A is

$$E[\Delta'_A] = Ppf(T) \left(L - \tilde{L}(T) \right) e^{-rT} \tag{25}$$

where P is the probability of arrival. The optimal planting strategy is chosen by finding the minimum loss, namely

$$J' = \min[E[\Delta'_A], \Delta'_B] \tag{26}$$

subject to $\tilde{L}(T) = L - I(T) + \rho I(T)$ where the area occupied by infected trees of species A is given by Equation (16). The optimal planting strategy, when $\rho = 0$, is shown in Figure 6 against the transmission rate and relative cost of establishment for species B (R_C). For slowly transmitting pathogens, it will be optimal to plant species A for a wide range of R_C values. As the transmission rate is increased, the loss from species A becomes greater than the loss from species B, thus it becomes optimal to plant species B. Once the primary infection and/or transmission rates are such that the infection spreads throughout the plot by the end of the rotation, the optimal planting strategy then depends on the probability of pathogen arrival (P) and R_C. For example, if a pathogen is certain to arrive then it is optimal to plant species B over a larger range of R_C values (top row in Figure 6), when compared with a pathogen which is unlikely to arrive (bottom row in Figure 6). Interestingly, when the loss due to planting species B occurs at the beginning of the rotation (through its higher establishment costs), the pattern in Figure 6 is similar to the reflected pattern in Figure 2, which is presenting a scenario where the loss due to planting species B occurs at the end of the rotation (through its lower timber value). The boundary between planting species A and B, which is found by setting both losses equal to each other, is

$$R_C^{B'} = 1 + P\left(\frac{pf(T)e^{-rT}}{cL}I(T)\right). \tag{27}$$

Thus, when the pathogen transmission rate increases, $I(T) \to L$ and $R_C^{B'} = 1 + Ppf(T)e^{-rT}/c$.

Finally, the case where the forest manager minimises the loss from planting a mixture of the species is given by

$$E[\Delta'_{AB}] = Ppf(T)\left(L_A - \tilde{L}(T, \delta)\right)e^{-rT} + cL_B(R_C - 1), \tag{28}$$

where $L_A = (1 - \delta)L$ and $L_B = \delta L$. The optimal planting strategy (which is not shown here) shows similar results to those presented in Section 4.2. For slow transmission, the loss from disease is at a minimum, and so it will be optimal to plant only species A. As the rate of pathogen transmission increases, the loss from disease increases; however planting a mixture will reduce the probability of contact with species A, (Equation 11)), and thus transmission, and so an area where it is optimal to plant a mixture emerges. Increasing the transmission rate further means that the infection will spread throughout the population of species A by the end of the rotation, and so the region in the parameter space where is it optimal to plant a mixture reduces, and eventually it will become optimal to plant a monoculture of species B or of species A depending on the probability of disease arrival P, and cost of establishment for species B. Decreasing the probability of pathogen arrival increases the area where it is optimal to plant species A.

6 Discussion

Our bioeconomic model shows that diversifying tree species composition can reduce the impact of a pathogen on a forest, and that this effect is dependent on the pathogen's characteristics (probability of arrival, time of arrival, and rate of spread of infection) and the costs (damage of the disease to the susceptible species and reduced benefit of planting the resistant species). Reduction of damage by a pathogen is just one reason why tree species diversification in forests is currently being advocated, other benefits include, but are not limited to: improved overall biomass through mixture overyielding (Smith et al., 2013); improved market resilience, using, for example, portfolio theory (Neuner et al., 2013; Roessiger et al., 2013; Knoke and Seifert, 2008); decreased wind throw or storm damage (Felton et al., 2016; Jactel et al., 2009; Schütz et al., 2006; Knoke et al., 2005); aiding adaption to a changing climate (Felton et al., 2016; Pawson et al., 2013); and reducing pest population sizes and damage (Griess and Knoke, 2011; Jactel et al., 2005; Jactel and Brockerhoff, 2007). However, it is often difficult to generalise about the benefits of diversification as individual studies concentrate on different specific systems. This is in part recognised by the number of papers with conflicting findings, which suggests that there may not be benefits to planting mixtures and, in some circumstances, there may be negative effects. For example, Griess and Knoke (2011) discussed that tree species with similar ecological niches will not produce a greater yield when planted together (since they may be competing for similar resources). Felton et al. (2016) highlighted that fire risk could actually increase for some mixtures if, for example, shade levels were altered such that understorey vegetation is promoted (which can act as a fuel). Moreover, whilst species mixtures have been shown to reduce pest outbreaks though mechanisms such as associational resistance (Jactel et al., 2009; Tahvanainen and Root, 1972), there are many other studies which argue that increasing the number of tree species, may facilitate invasion from more generalist herbivores (Koricheva et al., 2006). Often outcomes are dependent on the mixture selected and the productivity of the site (Felton et al., 2016), this highlights the importance of both primary empirical studies and meta-analyses of their results when trying to understand the effect of diversification on the forest system. Bioeconomic models like the one presented here can also add to this important discussion as they (1) provide a broader perspective on how different biological and economic characteristics qualitatively affect the optimal planting strategy, and (2) provide a flexible (and extendable) framework so that the optimal planting strategy for a specific host-pathogen system can be examined (often using data from empirical studies).

In the present study's models we made only one of the two tree species as susceptible to a pathogen. In reality, and due to the timescale of the rotation, both species may be susceptible to different pathogens or indeed to the same pathogen if there is some evolutionary change (such as for *Phytophthora ramorum*; Appiah et al. (2004)). Our model can be extended to include this by altering the epidemiological system and the objective function appropriately. However, we suggest that much caution is needed since it is not clear what effect the two

tree species being susceptible to separate pathogens would have on the pathogen transmission of each species (and possibly between species if the same pathogen can infect both). This complexity is highlighted in the following discussion regarding the pathogen transmission term used in this study.

The effect of how to characterise the pathogen transmission term within compartmental models has been widely debated within epidemiological modelling (Begon et al., 2002; McCallum et al., 2001). In this instance, our model uses a pathogen transmission term which is derived using the contact rate, the probability of contact with a tree species that is susceptible to the pathogen, and the probability that the tree which belongs to the susceptible species is susceptible to the pathogen (i.e. not infected). In our study, it is assumed that the probability of spores contacting a susceptible tree is proportional to the fraction of the trees in the forest which are susceptible to a pathogen (i.e. L_A/L). Whilst we do not specify the specific spatial arrangement of the tree species in the forest within this paper, the 'density-dependent' transmission term used can represent a forest where trees are intimately mixed. Changing the spatial arrangement of the forest significantly, may affect the probability of a pathogen spore contacting a tree of a conspecific species, and thus alter the disease transmission term. For example, if the two species are planted in two blocks, then the probability of contact between conspecific trees may be higher; in fact it may even be one. In turn this would alter the pathogen transmission term since $g_2(L_A/L) = 1$ (Equation (11)), and the commonly used 'frequency-dependent' transmission term is derived, giving

$$\frac{dI_A}{dt} = \beta \left(L_A - I_A(t) \right) \left(\frac{I_A}{L_A} + \epsilon \right). \tag{29}$$

Analysis of this bioeconomic model using Equation (29) reveals that it will never be economically optimal to plant a mixture (since no reduction in the probability of contact, and thus spread of infection, is gained by planting two species).

This highlights three important points. Firstly, more evidence of how tree pathogens spread within multi-species forests is crucial for understanding how this affects management strategies like tree species composition. Secondly, careful construction and interpretation of bioeconomic models is essential. Finally, how do different spatial arrangements within a forest change the optimal planting strategy in the presence of a tree pathogen? This final point would require further study using a bioeconomic model at the individual tree-level in order to incorporate the detail required. Moreover, if the spatial structure was representative of arrangements used in practice, then costs (and benefits) of species composition could be included in more detail. We ignore the potential for increased costs of planting a mixture (through, for example, a difference in combined timber yield or an increased cost of extraction), since this adds an unnecessary complexity, and is likely to depend on the species and their arrangement within the forest. However, extending the model framework we present here to be spatially-explicit allows (or examining the effect of) this detail to be included and would be an important contribution to the literature, with direct relevance to forestry practice.

At the other end of the spatial scale, if mixtures reduce the spread of infection within a forest, then this may also reduce the spread between forests since there is less infection pressure being emitted by, say, spores. This is an important question at the landscape scale since reduction in the spread between forests could 'buy time', which may reduce the overall damage by allowing trees to grow more before being infected, and so increasing the economic benefit of salvageable timber. The effect of mixtures has important policy implications since advice, incentive mechanisms or even regulations could be altered in favour of tree species mixtures not only to reduce damage within individual forests, but also to reduce the spread of tree diseases at a landscape and even regional level. This study only addresses effects of a pathogen on timber benefits within a single forest. To understand the effect of diversifying at a landscape scale, a bioeconomic model could be used to examine how different species mixtures affect the disease spread on a network of connected forests. This could be analysed from either the perspective of an individual forest manager (who manages a single forest on the network by minimising their expected loss as in this study), or from a social planner perspective (where the objective is to minimise the expected loss across all forests at a landscape level).

A further consideration is non-timber benefits produced by forests. In this study we have excluded these by concentrating on plantations managed for the dominant purpose of timber production. However, it is acknowledged that there are a range of non-timber outputs associated with plantation forestry, such as carbon sequestration, water regulation and habitat provision (Bauhus et al., 2010). Diversification of tree species composition is commonly linked to increasing the range of ecosystem services provided (Gamfeldt et al., 2013), however tree pathogens can often have an adverse effect on these (Pimentel et al., 2005). Quantifying the non-timber benefits, and the effect that the interaction of diversification and tree pathogens have on them, is likely to be difficult. However, bioeconomic models could be used to explore a range of effects on timber and non-timber benefits, and how these change the optimal planting strategy. Sensitivity analysis to the non-timber benefits would provide a useful comparison of how the optimal strategy for a plantation forest managed only for timber benefits compares with a multi-output forest. One way of examining this, could be to extend the objective function presented here to include a non-timber term which is dependent on the number of species planted and also the effect the disease has on the non-timber benefit. (Another possibility is for the non-timber benefits to be linked to the social planner model mentioned above since the provision of non-timber benefits is often dependent on the connectivity of forests, for example habitat for wildlife corridors (Lookingbill et al., 2010; Dobson et al., 1999).)

7   Conclusions

We develop a novel approach using a bioeconomic model to assess quantitatively the effect of tree disease on optimal planting strategy for tree species mixtures. To find the optimal planting proportion of two trees species

– of which one is resistant to disease and the other susceptible – we minimise the damage of disease (to the timber benefit) and the reduction in benefit caused by increasing the proportion of a second tree species that has a lower timber benefit, or greater establishment costs, compared with the first susceptible tree species (in the absence of infection).

A key result of this paper, is that we found that the risk and damage of disease can alter the optimal planting proportion. If the forest manager perceives that the risk of a pathogen arriving is zero, they will only plant the species which has the highest net benefit (due either to its higher timber benefit or lower establishment costs). If the forest manager wishes to plant such a single species monoculture where there a risk of arrival of the pathogen, the rate of primary infection and pathogen transmission increases the probability of switching to planting only the resistant tree species despite the lower net benefit (Figure 2 and 6). The probability of pathogen arrival also affects which species it is optimal to plant: as the probability decreases, the benefit of planting the susceptible species is greater since the expected damage due to disease is reduced (Figure 2 and 6).

When the forest manager has the option of planting a mixture of both tree species, the optimal planting proportion is dependent on the probability of pathogen arrival, the time at which the pathogen arrives, the rate of pathogen transmission, the effect of disease on the timber value, and the reduced benefit of planting the resistant species (through either its lower timber benefit or higher establishment costs compared to the susceptible tree species, in the absence of infection). For a pathogen which arrives early or has a small rate of transmission, the optimal planting strategy is to plant a monoculture of the susceptible species since the damage caused by the disease is small (Figure 3). As the rate of pathogen transmission increases, it becomes optimal to plant a mixture of both species, predominately because introducing the resistant species will reduce the probability that the pathogen will infect a tree which is susceptible to disease (Equation (10). This is a key result: planting a tree species mixture will increase the overall net benefit even if the benefit from the disease resistance of the second species is small (Figure 3 and 4). Increasing the transmission rate again will reduce the benefit of planting a mixture and it will be optimal to plant only the resistant species. However, a decrease in the probability of the pathogen's arrival will reduce the expected loss due to the disease, and so it may be optimal to plant only the susceptible species (dependent on the difference in benefit between the resistant and uninfected, susceptible species (Figure 3)). Reducing the effect that disease has on the timber benefit of the susceptible species will increase the proportion of the susceptible species that it is optimal to plant (Figure 5).

In the final part of this study we examined the case where the difference between uninfected, susceptible tree species and resistant tree species occurs at the beginning or end of the rotation (through a difference in establishment costs or timber benefit respectively). We found that the sensitivity of the optimal planting strategy to the different disease characteristics behaved similarly. This showed that the qualitative changes in the optimal planting strategy is independent of whether the difference between the two species occurs at the beginning or end of the rotation; however we have not examined the effect of this difference on the value of the

net benefit of the optimal solution. One extension to this model would be to examine the case where the resistant species is more expensive to establish *and* has a reduced timber value (compared with the uninfected, susceptible species). Moreover, it is interesting to note that when the difference between the uninfected, susceptible tree species and resistant tree species occurs at the end, the optimal planting strategy is not dependent on the discount rate (Equation (9)). However, when the difference occurs at the beginning (due to the difference in establishment costs), then the optimal planting strategy may be dependent on the discount rate (Equation (28)). Increasing the discount rate will decrease the expected timber benefit (from both species), but the effect on the optimal planting strategy is not clear, thus sensitivity of the results to the discount factor should a future research priority.

Most previous modelling/statistical work on this topic is for specific host-pathogen systems and uses data from the field (Guyot et al., 2016; Hantsch et al., 2014; Haas et al., 2011; Thor et al., 2005; Gerlach et al., 1997). Therefore, this paper makes a step-change advance on existing capacity to assess the effect of diversification of production forests with respect of emerging pathogens through a general framework to analyse the impact of economic and biological conditions on the optimal planting strategy in the presence of tree disease. This flexible model framework can also be parameterised (and extended) to represent a specific host-pathogen system which would allow the optimal planting strategy to be examined for threats of new pathogens.

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Table 1: The parameter definitions and baseline values used in this paper.

Parameter	Definition	Baseline value
ECONOMIC		
c	Establishment cost of species A	$c = $ £1920 ha^{-1} *
p	Price of timber from species A	$p = $ £18.24 m^{-3} **
R_c	Cost of establishment of species B (relative to species A)	$R_c \in [13]$
R_p	Price of timber of species B (relative to species A)	$R_p \in [0, 1]$
r	Discount rate	$r = 0.03$
ECOLOGY		
L	Total area of forest	$L = 1$ ha
δ	The planting proportion of species B	
L_i	Area of trees from species $i \in [A, B]$	$L_B = \delta L$ and
		$L_A = (1 - \delta)L$
$f(T)$	Timber volume growth (m^3 ha^{-1})	$f(T) = 579.9$ m^3 ha^{-1}
EPIDEMILOGY		
P	Probability of pathogen arrival	$P \in [0, 1]$
ϵ	Primary infection rate	$\epsilon = 0.00033, 0.0175, 0.13$
β	Secondary infection rate	$\beta = 0.1$
ρ	Revenue from the timber of infected trees relative to uninfected trees	$\rho = 0$
$\tilde{L}(T, \delta)$	Effect of disease on the forest area of species A at time T	$\tilde{L}(T, \delta) = S(T) + \rho I(T)$

*The net cost of planting is taken to be zero on the basis that the gross cost is the same as the Scottish government subsidy payments available for Woodland Creation operation on 24th March 2016 (in the form of an initial planting payment; `https://www.ruralpayments.org/publicsite/futures/topics/all-schemes/forestry-grant-scheme/woodland-creation/`).

** The price of timber is the average standing price (per cubic metre overbark) taken from Coniferous Standing Sales Price Index on 30th September 2015 for Great Britain (`http://www.forestry.gov.uk/forestry/INFD-7M2DJR`).
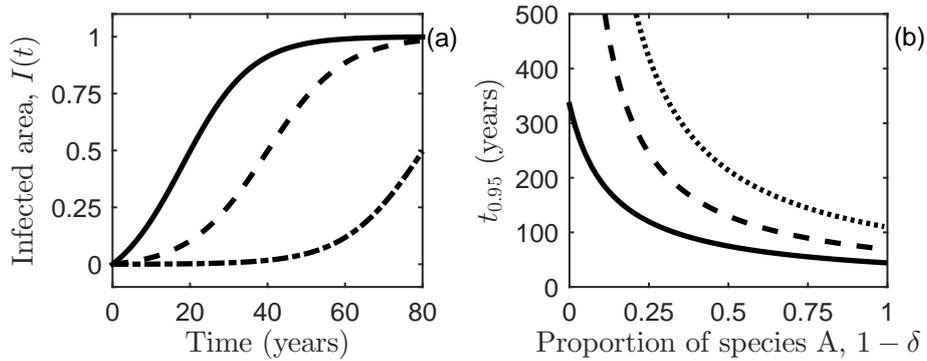

Figure 1: Area occupied by infected trees over time and the time taken for 95% of species A to become infected. In (a) the area of infected trees from Equation (16) is shown over time (When $L = 1$ha). In (b) the time taken for 95% of species A to become infected (t_θ where $\theta = 0.95$ in Equation (17)) is shown against the proportion of species A, $1 - \delta$. In both figures the pathogen transmission is $\beta = 0.1$, and the primary infection rate is $\epsilon = 0.13$ (solid), $\epsilon = 0.0175$ (dashed) and $\epsilon = 0.00033$ (dotted).
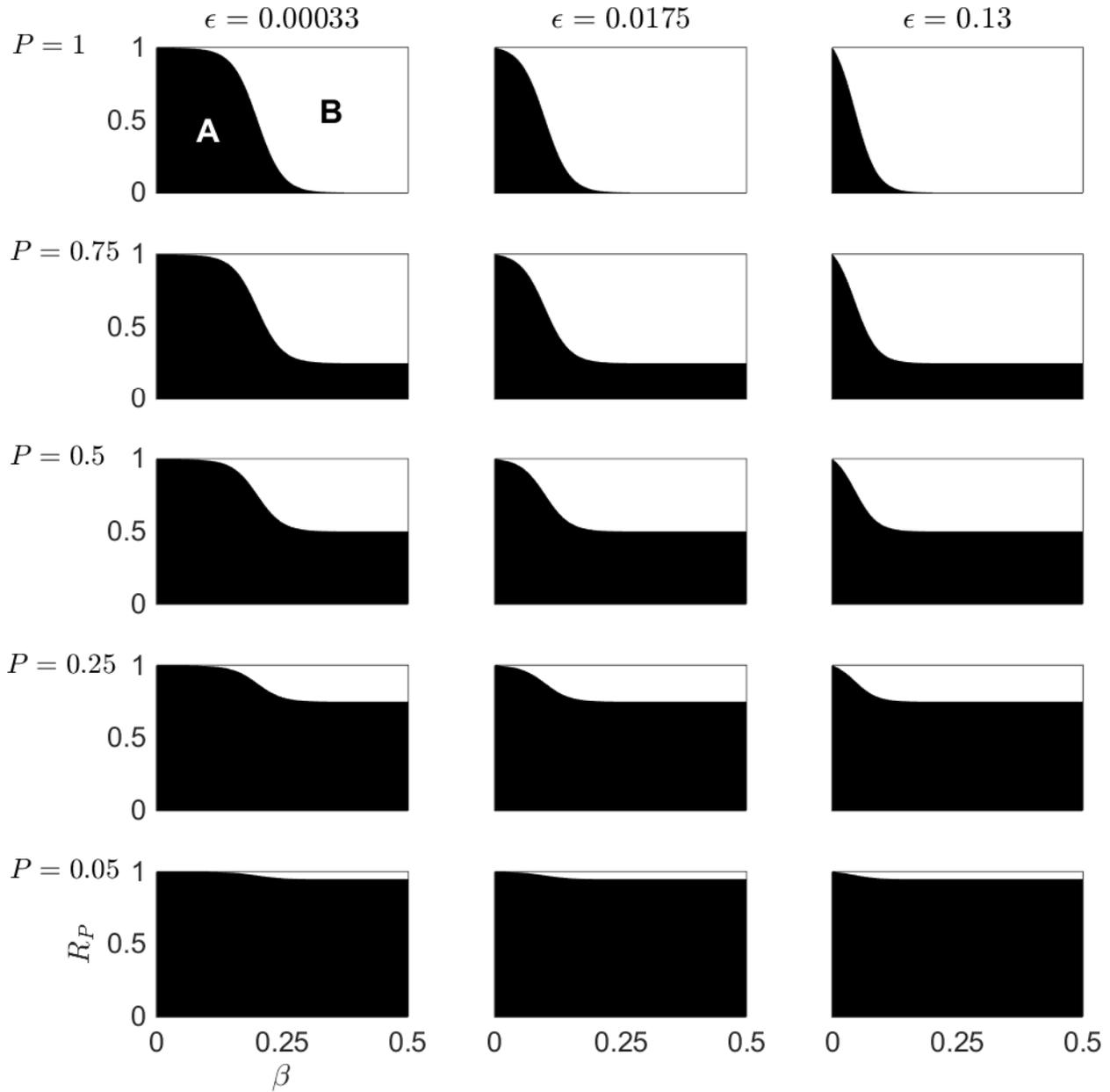
Figure 2: The optimal planting strategy for a monoculture when the timber of species A has a higher value then of species B. The optimal planting strategy for a risk neutral manager is found by minimising the expected loss due to planting either species A or species B (Equation (7)) in a $\beta - R_P$ parameter space (pathogen transmission *vs.* the timber value of species B relative to species A). The primary infection rate, ϵ, is altered between each column, and the probability of pathogen arrival, P is altered between each row. Only a monoculture can be planted: all species A (black area) or all species B (white area). Parameter values are given in Table 1.
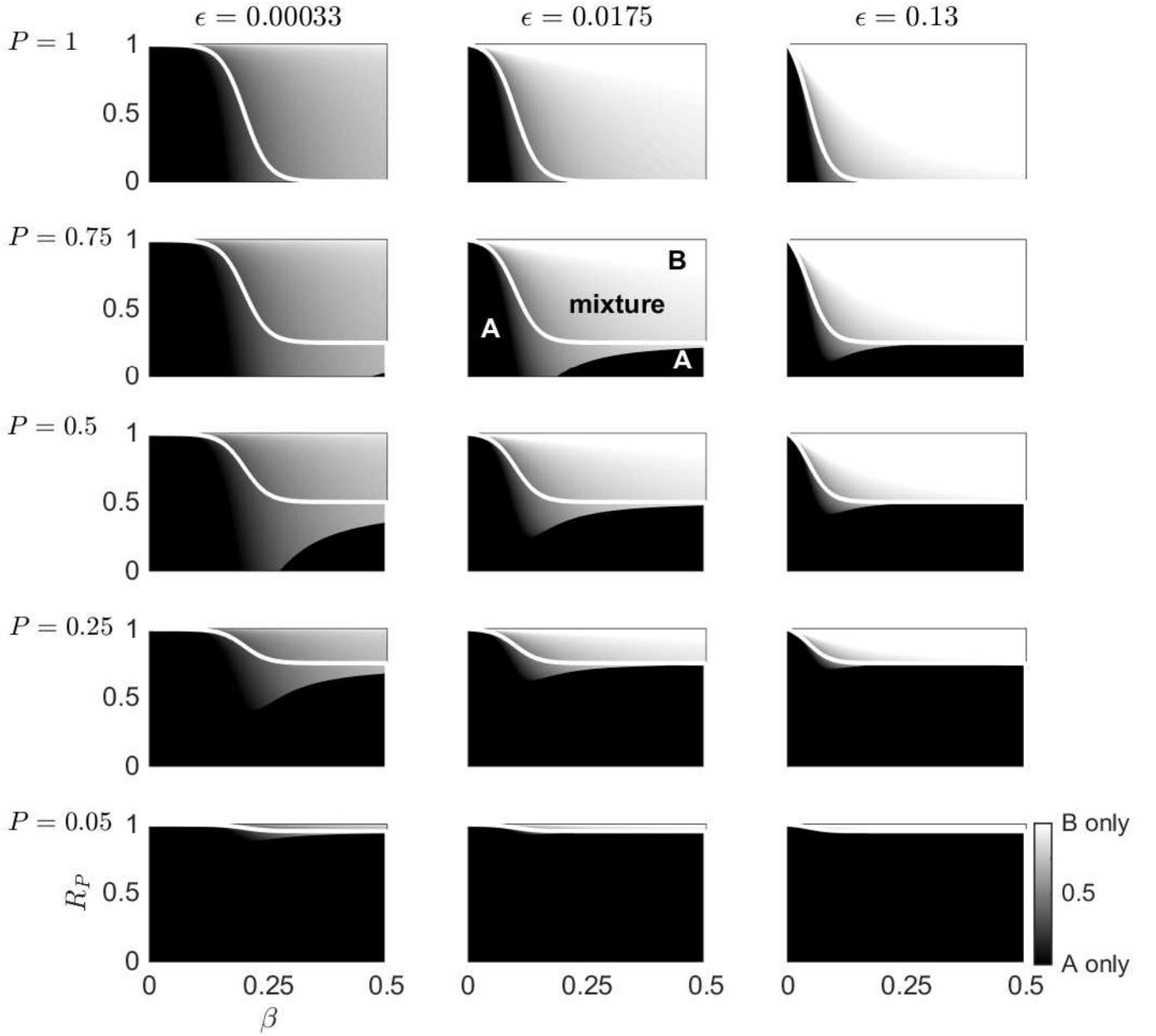
Figure 3: The optimal planting strategy for a mixture when timber of species A has a higher value then of species B. The optimal planting strategy for a risk neutral manager is found by minimising the expected loss (Equation (9)) in a $\beta - R_P$ parameter space (pathogen transmission *vs.* the timber value of species B relative to species A). The primary infection rate, ϵ, is altered between each column, and the probability of pathogen arrival, P is altered between each row. The grey scale (bottom right) shows δ, the optimal planting proportion: a monoculture of species A (black), of species B (white) or a mixture of A and B (gradations of grey). The white line indicates the switch in planting strategy when only a monoculture is allowed (i.e. the border between the black and white parameter spaces in Figure 2). Parameter values are given in Table 1.
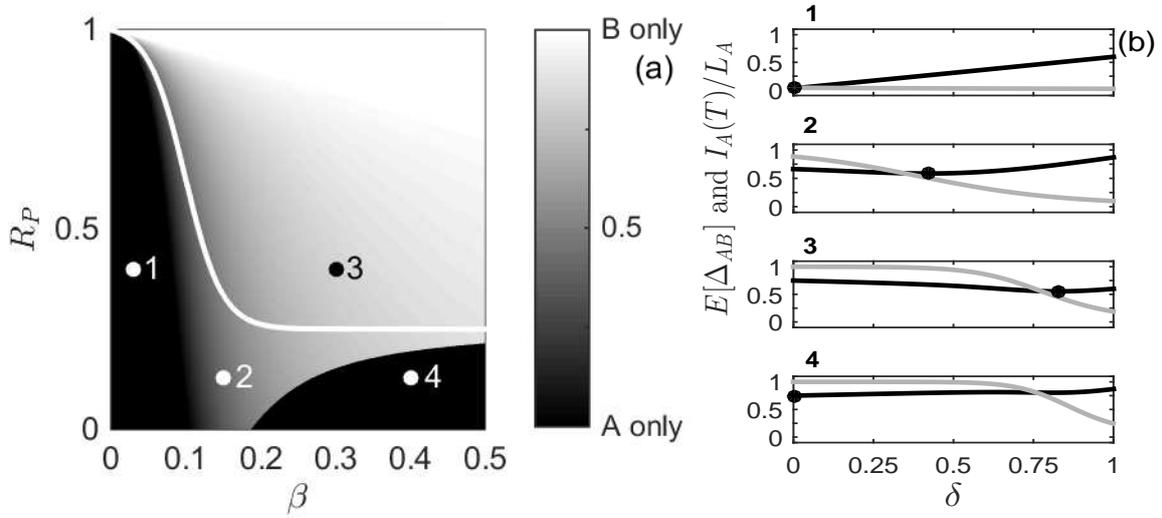
Figure 4: The proportion of species A that is infected and the expected economic loss at the end of the rotation against the proportion of species B. In (a) the optimal planting strategy is found by minimising the expected loss in Equation (9) and plotted in a $\beta - R_P$ parameter space (pathogen transmission *vs.* the timber value from species B relative to species A) for probability of disease arrival $P = 0.75$ and primary infection rate $\epsilon = 0.0175$. The grey scale shows δ, the optimal planting proportion (shade bar at the right): a monoculture of species A (black), of species B (white) or a mixture of A and B (gradations of grey). The white line indicates the switch in planting strategy when only a monoculture is allowed (i.e. the border between the black and white parameter space in Figure 2). In (b) the expected loss due to planting a mixture ($E[\Delta_{AB}]$ in Equation (9); black), and the proportion of species A that is infected ($I_A(T)/L_A$ from Equation (14); grey) are shown against δ for the four selected regions in the parameter space in (a). The black dot on the $E[\Delta_{AB}]$ curves indicates the minimum expected loss, and so gives the optimal value of δ (when $\delta = 0$, only plant species A only, and when $\delta = 1$ only plant species B). Parameter values are given in Table 1.
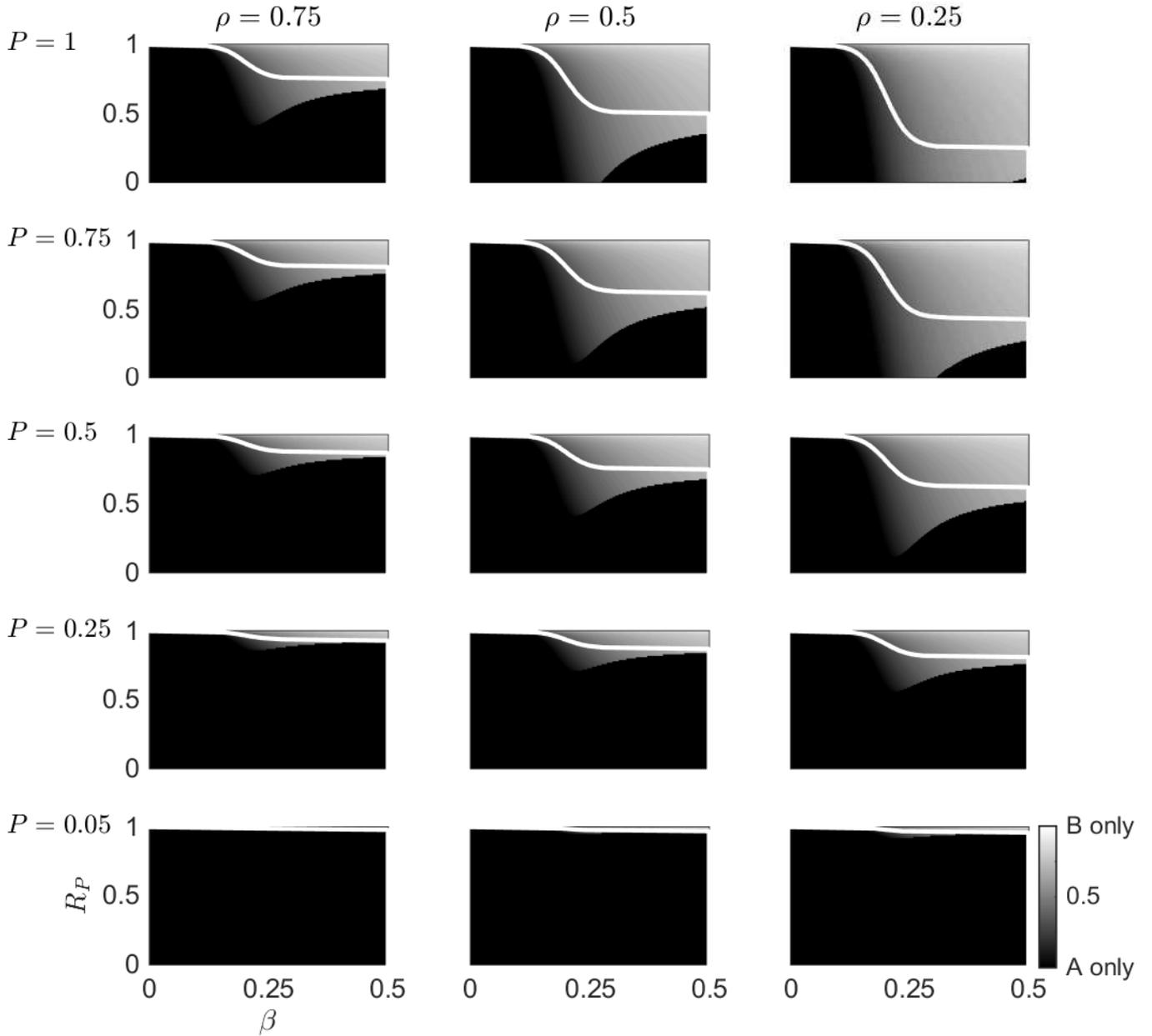
Figure 5: Sensitivity of the optimal planting strategy to changes in the revenue of timber from infected trees (relative to uninfected trees), ρ, when timber of species A has a higher value than of species B. The optimal planting strategy for a risk neutral manager is found by minimising the expected loss (Equation (9)) in a $\beta - R_P$ parameter space (the pathogen transmission $vs.$ the timber value of species B relative to species A). The primary infection rate is $\epsilon = 0.00033$. The reduced value of timber from infected trees of species A, ρ, is altered between each column, and the probability of pathogen arrival, P, is altered between each row. The grey scale (bottom right) shows δ, the optimal planting proportion: a monoculture of species A (black), of species B (white) or a mixture of A and B (gradations of grey). The white line indicates the switch in planting strategy when only a monoculture is allowed. Parameter values are given in Table 1.
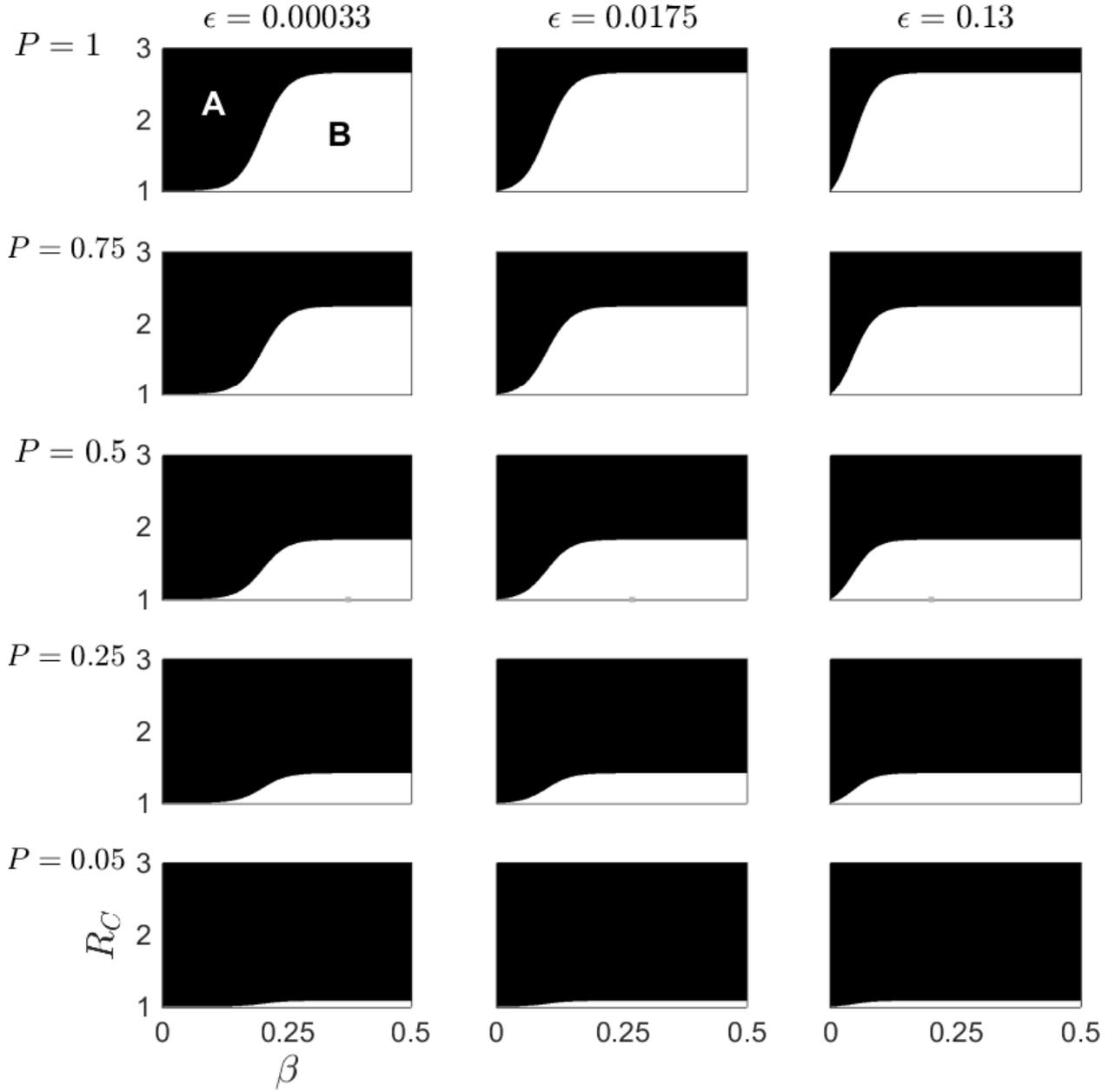
Figure 6: The optimal planting strategy for a monoculture when species B has a higher cost of establishment than species A. The optimal planting strategy for a risk neutral manager is found by minimising the expected loss (Equation (26)) in a $\beta - R_C$ parameter space (pathogen transmission *vs.* the establishment cost of species B relative to species A). The primary infection rate, ϵ, is altered between each column, and the probability of pathogen arrival, P, is altered between each row. Only a monoculture can be planted: all species A (black area) or all species B (white area). Parameter values are given in Table 1.