Optimal management of an emerging pathogen

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DECLARATION

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Abstract

Emerging pests and pathogens have potentially devastating environmental, ecological and economic consequences, making their early control an imperative. There is an increased interest in designing management strategies that reduce the rate of arrival and spread of a pathogen ('precautionary' management) and are frequently promoted as preferable. However, in practice, management strategies are often only deployed once an outbreak has been detected ('reactionary' management). With the frequency of pest outbreaks likely to increase, a key policy question is how to deploy resources to reduce their potentially irreversible impact.

In this thesis, we create a deterministic bioeconomic model to examine how the optimal level of precautionary management (which is restricted by a budget) changes when the key characteristics of the pest and its economic impact are changed. We show that when a reactive management strategy is unavailable, the optimal level of precautionary management increases as the primary and secondary disease transmission rates and the loss (caused by disease) are increased. This trend still holds when a reactive management strategy is available, except for a small range of loss values (caused by disease), where it is optimal to wait and deploy the reactive strategy only. We show that the optimal management strategy is highly sensitive to small changes in the effectiveness of both precautionary and reactive management.

The deterministic model is modified to include random arrival to find the optimal management strategy and the difference between deterministic and stochastic results. We introduce uncertainty into the deterministic model using the absolute value of a normal distribution and log-normal distribution. We extend the "profit" function by an exponential utility function to

include risk attitude. So, allocation of resources changes when utility function changes and managers become more risk-averse for both precautionary and reactive management strategies. The key policy question is how to reduce the potentially irreversible impact of pathogens on the environment. Biosecurity measures are implemented to reduce (or eliminate) the risk of introducing pathogens from outside the country (or region) both for prevention and reactive measures. These reactive actions are intended to reduce the present and future economic and environmental losses caused by the arrival of pathogens.

Sensitivity analysis highlights that the optimal management strategy is very sensitive to small changes in the effectiveness of both precautionary and reactive management. The optimal control theory is employed to help the decision maker determine the paths of expenditure that minimise the present value of the costs associated with disease over a fixed period. The analysis quantified the uncertainty associated with disease predictions by running multiple simulations with different parameter values. By simulating multiple scenarios and analysing the results, we gain valuable insight into the interaction between the random arrival of the pathogen, the spread of the disease, and the effectiveness of the management. It provided a range of potential disease outcomes, aiding in risk assessment and decision-making under uncertainty. The research affirmed the importance of adopting risk management strategies for farmers and stakeholders to navigate uncertainties in plant and agricultural activities. Using risk management tools indicates an interconnected relationship between different risk management strategies.

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Chapter 1

Introduction and Background

"In the field of observation, chance favours only the prepared mind"

Louis Pasteur, a French chemist and microbiologist, celebrated for his findings of the principles of vaccination, microbial fermentation, and pasteurisation, emphasises that scientific discoveries often occur through systematic observation and inquiry. In scientific research, it is not merely chance that leads to breakthroughs but rather the deliberate, methodical examination of phenomena. Pasteur's statement implies that researchers should actively engage with their subjects of study [105]. In the context of this thesis, this quote is applied to the challenges of emerging plant pathogens. The main goal is to investigate the optimal management of emerging and re-emerging plants pathogen using statistical and mathematical approaches. We begin with a brief introduction to the biology of plant pathogen and current trends in the UK. However, by being prepared through research, surveillance, and response strategies, we can better equip ourselves to manage and mitigate the impact of these pathogens on agriculture and on the natural environment.

1.1 Emerging / Imported Pests and Diseases

Plant pests and diseases are a major threat to UK agriculture [72]. They cause significant crop losses, along with a loss in biodiversity and ecosystem services [30]. Furthermore, due to the

increasing globalisation and changes in climate, which are increasingly putting more pressure on native plant species, we have an increased risk of invasive pathogens [130]. The UK plant health regime has recognised the need to manage these risks for securing plants [147]. Defence mechanisms against pests and diseases should be able to anticipate the potential impact of climate change so as to protect plants from these threats and avoid invasion risks caused by introduction agents [35, 45]. Likewise, the economic implications of plant diseases are also a subject that deserves our attention [130]. It has been established that plant pest breakouts cause huge economic losses due to the impact on ecosystem services that affect human well-being [26, 70]. One recent estimate suggested that it might cost £15 billion over 100 years—of which the first decade alone was to amount to £7.6 billion [62].

In the UK, there are around 120 million mature ash trees that provide essential ecosystem services and habitats [62]. Though exact mortality rates are unknown, native European ash trees should show 'substantial depletion' ranging between 70% and 90% loss [130]. In total, ash supports around 1000 species but with specificity to this tree and nowhere else for 45 of them [62, 130]. It is crucially important to have well-developed surveillance and monitoring schemes at the field level to control plant pests properly through disease management [69, 121]. Such practice will help to detect and implement a quick response as soon pests or diseases are signalled [26, 62]. The Great Britain Plant Health and Biosecurity Strategy is based on a risk-based approach, with those pests and diseases which have the highest potential impact [62, 130].

The UK has implemented strict measures to prevent the introduction and spread of plant pests and diseases [130]. These measures include regulations, regular inspections, and monitoring of plant materials entering the country. The UK government, through the Animal and Plant Health Agency, plays a crucial role in enforcing these measures and ensuring compliance with plant health regulations by conducting inspections at ports and airports, as well as within nurseries and plant-growing facilities [130]. The implementation of the Great Britain Plant Health and Biosecurity Strategy 2014–2019 has been effective in addressing plant pests and diseases in the UK and has led to improved surveillance, early detection, and management

practices [96, 130]. According to the DEFRA 2019 progress report, the strategy contributed to better coordination of plant health surveillance and the development of risk-based inspection regimes [130]. Additionally, the strategy has increased awareness and understanding of plant health issues among the general public through education and citizen science initiatives [130]. Evidence of this can be seen in initiatives such as the 'Observatree' project, a citizen science program launched in collaboration with the Forestry Commission and partners, which has involved volunteers in monitoring tree health across the UK [27]. Furthermore, Defra-supported 'Plant Health Week' campaigns have been instrumental in engaging schools and communities in discussions about biosecurity and pest threats [130]. The integrated plant health management strategy is a holistic approach to managing plant pests and diseases that focuses on prevention, early detection, and control [96, 130]. It combines various strategies, including cultural, biological, and chemical control methods, to effectively manage plant health threats while minimising negative impacts on the environment and human health [79]. Effective management of new plant pathogens is key to maintaining crop health and food safety [20, 30, 133]. More than 750 plant pests and pathogens are on the Plant Health Risk Register of Defra UK [67, 126]). As direct consequences of such disease outbreaks, there are economic losses for the agriculture and forestry business, changes in biodiversity and landscape within affected areas [109, 126].

Global change, such as climate change, is expanding the geographical range of many infectious agents into regions where they were once not endemic [30, 133]. Globalisation enabled the movement of goods and people, leading to a quick distribution of pathogens and pests [43]. The introduction of non-native species to new environments could disturb local ecosystems and facilitate pathogens that might otherwise not be able to effect hosts in a balanced state [43, 146]. Factors like agricultural practices, monoculture farming, and even pesticides can have the unintended consequences of actually creating perfect environments for pathogens to grow [111]. Plant pathogens have the potential to adapt, evolve and overcome plant defences in often unpredictable ways [150]. As they adapt, the established notion of plant-pathogen interactions and, consequently, our capacity to manage their disease

development or transmission are challenged [89]. The globalisation of agriculture also now allows a pathogen outbreak in one part of the world to become a crisis affecting all other parts quickly [18, 100]. The infectious agent can also replicate within or on the plant surface, and can spread from host to host as for human or animal diseases [89, 94].

Timely, accurate detection of pathogens is critical for infection prevention and disease onset [8]. Efficiency and proper assessment of integrated pest and disease management strategies depend considerably on developing rapid, sensitive, and specific diagnostic methods for identifying crop pest diseases [142]. These methods facilitate fast responses and deployments of needed control measures to protect agricultural systems [120, 142]. Methods based on the symptoms and pathogen cultures are traditional; however, issues such as slowness in results or reliability of outcomes arise [118]. Hence, it is important to design better technologies for being able on-the-spot, rapidly and correctly identify pathogens that can be implemented in the field [33, 118].

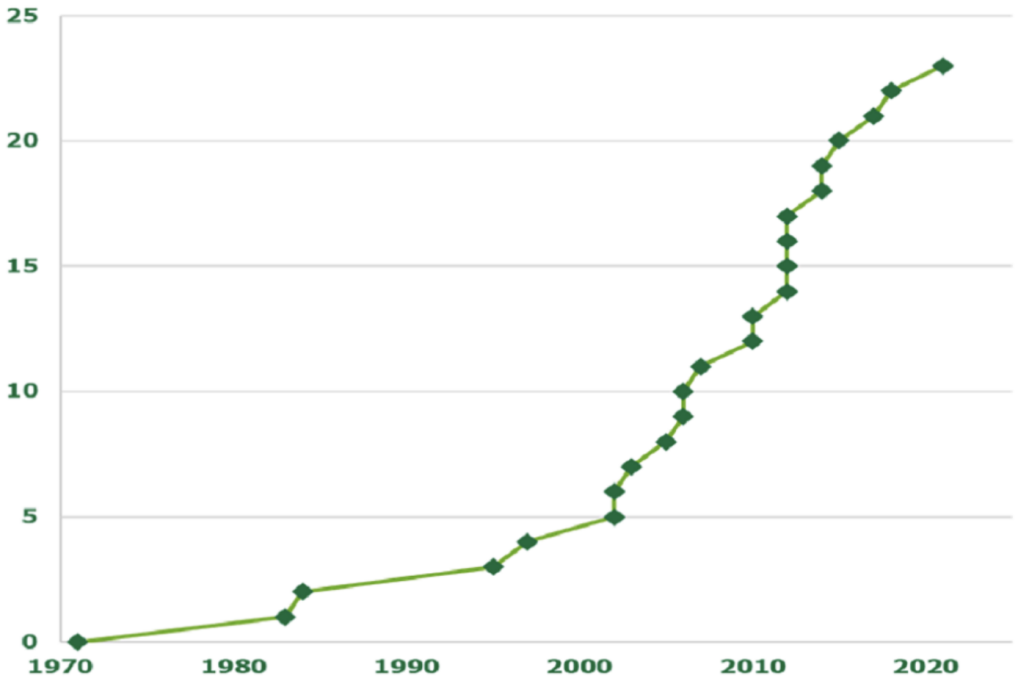

Figure 1.1: Cumulative incidence of first detection of free pest. The cumulative incidence of disease outbreaks in the number of new pests and disease outbreaks (y-axis) affecting plants since 1971 (x-axis). This Figure is from [30].

An annual amount of 4 million tonnes of plants and plant material is imported into the UK, which has risen due to a global trade increase of more than five-fold over the last century, significantly accelerating pathogen and pest transfer [30]. Figure 1.1 shows the cumulative addition of new plant pests and diseases, from Dutch Elm Disease in 1971 to *Phytophthora pluvialis* detected in the UK in 2021 [91]. During the 30 years leading up to 2000, there were sporadic invasions as opposed to the subsequent period, where monthly incidence totals reached a maximum of only 19 [30, 133]. This demonstrates a significant uptrend in the frequency of outbreaks between 2002-2020 (Figure 1.1) [30].

The arrival in the UK of *Hymenoscyphus fraxineus*, an aggressive pathogenic fungal disease (causing 'ash dieback'), triggered naturally from wind-borne spores but also via infected plants taken to market, played a major role and was a pivotal moment for public attention on biosecurity that lead to the publication of first plant biosecurity strategy published by

DEFRA in 2014 [30]. *Ash dieback* is a devastating fungal disease which affects ash trees [30]. While progress has been made in implementing this strategy, *ash dieback* is just one of many escalating threats to plant health, as illustrated by the rising number of new plant pests and diseases detected over the years [30].

The UK has made measurable progress in addressing plant health threats through improved surveillance and response systems, as evidenced by the improved detection and containment of outbreaks such as interceptions of *Xylella fastidiosa* and the national ash dieback action plan [130]. However, ash dieback is only one of a growing number of threats to plant health, as demonstrated by the increasing number of new pests and diseases identified across a range of plant species in recent years [130]. The UK left the EU in 2020, and there is an opportunity to take stock and strengthen frameworks looking at plant biosecurity risk levels but also safeguard a £15.7 billion annual economic environmental and social benefit that plants and trees deliver [30]. This incorporates extensive benefits from carbon sequestration, agriculture and horticulture with forestry, recreational opportunities and potential for biodiversity to landscape amenities, and even mental health well-being with functioning social network values [30]. This puts plants' asset value at circa £350 billion over 100 years, demonstrating the need to acknowledge and protect what is termed as nature's health service from its domination by industry [30].

Global movements of plants and plant materials have increased level of threats to agricultural systems, natural ecosystems and community health by the movement of many pest species [41]. There are many common or widespread plant pests and diseases that, in terms of identification and classification, present significant risks to UK plant health [130]. In the UK, introducing new pests and diseases is a huge burden on local plant production due to increasing examples in agriculture [17]. For example, the caterpillars of *Oak Processionary Moth* (OPM), a defoliating insect is harmful to oak trees and poses serious risks to human health as a cause of allergic reactions. *Thaumetopoea processionea* was first seen in Britain nearly two decades ago and has now colonised multiple areas across several regions [30]. The Oak Processionary Moth has since been found across the UK and is considered a significant

threat to forestry and public health authorities [131].

However, these examples also underscore how trade can move plants and animals in ways that create substantial additional outcomes (e.g., the transport of invasive species and the spread of disease) [132]. The *box tree moth, Cydalima perspectalis*, was first detected in the UK in 2008 [30]. It is a major threat to box trees, commonly used for hedging and topiary [30]. In 2015, a different imported plant pest, the *tomato brown rugose* fruit virus, was reported in the UK [30]. Tomato yellow leaf curl disease is a very harmful viral affliction that affects tomato plants, occasionally causing near-wholly crop yield loss [30]. These cases show how increased biosecurity to prevent the arrival and spread of plant pests and diseases within the UK is critical [30]. Furthermore, global warming can dramatically influence disease risk by increasing environmental temperatures [45, 56, 115].

Controlling pests and pathogens has been derived largely from research to prevent avoidable infections and manage those present [59]. By identifying regional patterns and the prevalence of serotype, the study [59] highlights the importance of robust surveillance systems and early detection in the management of disease spread, principles that are equally relevant in plant health. The emphasis of the paper [59] on international collaboration, data harmonization, and laboratory proficiency underscores the need for similarly coordinated networks in plant health to effectively monitor, report, and respond to outbreaks. In this way, the study reinforces the broader concept of "One Health", showing how methods used to monitor food-borne pathogens in humans and animals can inform precautionary approaches, early warning systems, and tailored interventions to control infection pathways in plants [59]. Only once the infection pathway is known can measures be taken to protect other plants [117]. Good infection control practices can greatly reduce the rate of avoidable infections, thereby preventing and reducing it [135].

Invasive species can be introduced through different trade pathways [3, 38]. These comprise unplanned invasions, where transported plants carry along with the nonnative organisms and are represented in planting materials [3]. Invasions can also occur through the intentional importation of plants that subsequently escape and spread [3]. The value of

performing pest and disease risk assessments is key for determining both the likelihood that a species will arrive or establish and estimating probable damages [38]. Pest and disease risk assessments are essential for excluding, quarantining and controlling invasive species related to specific commodities or pathways [38]. The complexities of managing biosecurity risks in international trade, including the requirement for collaboration and harmonisation between trading partners to establish and enforce good regulations and standards [13, 38]. The role of global initiatives and agreements, such as the International Plant Protection Convention and the Convention on Biological Diversity, in addressing invasive species and trade-related biosecurity concerns [38, 141]. This thesis, therefore, stresses that a multifaceted risk management approach is necessary for these risks, incentive-based tools, and monitoring to identify early impact changes [11]. It emphasizes the need to consider and manage biological invasions as they manifest through international trade flows [38, 65]. Although descriptive evidence shows the increasing risks of emerging and imported pests, analytical modeling provides a framework to evaluate which management strategies are cost-effective. We then turn to these modelling approaches.

1.2 Basic concepts

1.2.1 Invasive Species Management

Control and management of plant diseases caused by emergent pathogens are complex systems at the gateway between biology, economics, and probabilistic expositions [68]. Plant diseases constantly change, and sophisticated solutions require incorporating biological, economic or risk assessment aspects and models developed to predict increasing impacts [68]. Infecting plants with bacteria, fungi, viruses, and other pathogenic microorganisms can be disastrous to agricultural systems worldwide and threaten food security [128]. For many years, the study of these infections has intersected with biology or sought to shed light on pathogen-host interactions [97]. The Susceptible-Infectious (SI) model is one of the basic

epidemiological models that have helped to build a theoretical framework for understanding pathogen transmission within plant populations [81]. These models have helped elucidate the dynamics of infection transmission and the role of factors like plant susceptibility and pathogen virulence [68, 81].

Invasive species seriously threaten biodiversity, ecosystem functioning, human health and economic well-being [53, 110]. The management of invasive species that cause damage to plants has become an important area of research [39]. Invasive species can cause ecological disruption, loss of native species, and negative impacts on agriculture and human health[110]. If an invading pathogen arrives and spreads, the combined benefits flows through the parcel under investigation will be reduced over time [110]. Efforts to unravel the relationships between economic behaviour and pathogen spread have come from work at the interface of ecology, infectious disease epidemiology, and economics in recent years [107].

The introduction of *Ambrosia artemisiifolia* (common ragweed) from North America to Europe offers a compelling example of the broader impacts of invasive species, with direct relevance to the management of invasive plant pathogens [110]. This species has been associated with a notable increase in the duration and severity of hay fever seasons in affected regions, illustrating how biological invasions can have significant public health implications [110]. From a plant health perspective, this case underscores the importance of early detection and rapid response systems in managing the spread of harmful organisms [110]. Monitoring networks, public awareness campaigns, and integrated surveillance, strategies used in the tracking of ragweed pollen, can be applied similarly to identify and manage invasive plant pathogens before they establish and spread [110]. Furthermore, the use of biological control agents, as tested against *Ambrosia artemisiifolia*, provides a model for environmentally sustainable approaches to the management of plant diseases [110]. This example also highlights the role of cross-sectoral collaboration, involving public health, agriculture, and environmental authorities, which is essential for the effective management of the risks of plant pathogens [110].

Pre-invasion management refers to actions taken before a non-native pathogen or pest

has established in a new area, with the goal of preventing introduction or reducing its likelihood or potential impact [64, 83, 123]. Many works mainly rely upon models to study the fundamentals of pre-invasion management strategies for invasive pathogens from economic and epidemiological perspectives [28, 63]. *Palmer, Heard, and Sheppard (2010)* present a seminal bioeconomic model that integrates ecological and economic components to assess the cost-effectiveness of invasive species management strategies [103]. Using *Parthenium hysterophorus* as a case study in Australia, the authors simulate the spread of the weed over time under different control scenarios, including chemical, biological, integrated and delayed interventions [103]. Their model captures both the ecological dynamics of invasion and the economic trade-offs involved in the timing of the intervention and the selection of the intervention method [103]. The findings show that integrated management, particularly early implementation of biological and chemical control, yields the highest net economic returns over time, while delayed action significantly increases long-term costs [103]. This study underscores the value of bioeconomic modelling as a decision support tool, guiding decision-maker toward more cost-effective and sustainable approaches to the control of invasive species [103]. Plant infections have been recognized for some time to have severe economic consequences [136]. In fact, loss of crop yield and lower supply chain reliability means that there are clear economic testimonials to evaluate the consequences associated with plant diseases [136]. Economic models, such as the Crop Disease Economics (CDE) model, help us to perform economic impact assessments of plant infections to assist land manager in decision-making [88]. Unless stated otherwise, the modelling framework takes a social planner perspective, focusing on minimising total economic damages at the system level. However, in some scenarios we consider the perspective of a private landowner, who aims to minimise their own economic losses. Clarifying this distinction is important, as it affects how benefits of prevention and control are evaluated. When reactive management is an available strategy, the first trend remains true with two exceptions: over a narrow range of annual losses due to disease, it is optimal not to act and deploy whatever limited amount of resources you have [58].

The challenge of plant infection is compounded by the inherent stochasticity, which introduces further complexity [46]. If there is no response management strategy, the level of precautionary increases as the primary and secondary disease transmission rates (and diseases losses) increase [10]. For studying infectious disease spread, it is essential to include stochastic models due to the inherent randomness in disease dissemination, and this becomes critical, especially for emerging pathogens that have hardly been studied, such as COVID-19 [134]. Such models should incorporate variations in environmental circumstances, pathogen entry, and host susceptibility [10].

Hanley and Roberts (2019) provide a comprehensive synthesis of the economic consequences of invasive species in terrestrial, aquatic, and agricultural systems, with a strong focus on the valuation challenges involved in cost–benefit analyses of management interventions [54]. The literature focuses on the costs of invasions, such as losses in agricultural productivity, damage to infrastructure, loss of biodiversity, and impacts on ecosystem services management actions, which can also generate positive economic outcomes beyond damage reduction [54]. These include the restoration of ecosystem services (e.g., improved water quality or pollination after removal of invasive plants), employment opportunities linked to control efforts (especially in rural areas), and in some cases, increased recreational or aesthetic value of landscapes after eradication or containment efforts [54]. The review advances the field by calling for more comprehensive economic evaluations that include both avoided costs and co-benefits when evaluating invasive species policy and practice [54].

However, an interdisciplinary article that effectively analyses ecological knowledge for managing invasive species in a socioecological system, such as European red foxes, presents further evidence to combine scientific and community-based forms of understanding [95]. *Moon et al. (2015)* interviewed policy advisors and scientists, each of whom had unique ideas about whether community stakeholder knowledge matters or not [95]. However, *Moon et al. (2015)* recommends adopting a co-management and co-production strategy that legitimises different types of knowledge claims and stakeholder involvements in the policy-making process [95]. This understanding is crucial in managing invasive species (of any kind), especially

in populated areas where socio-political aspects have a greater weight [95]. These results highlight the importance of combining different knowledge to help increase control success rates in invasive plant management [95].

Advanced and accurate diagnostic tools, as well as technologies, are one of the key gaps in the management of emerging pathogens [144]. These include new approaches, such as the use of advanced diagnostics and surveillance systems, as well as improved coordination and communication, which are central to countering emerging pathogens [44]. By incorporating these practices with current management strategies, organisations and healthcare facilities may improve their readiness to combat emerging pathogens [44]. The faster and more accurate detection of pathogens is possible through the use of advanced diagnostic tools, allowing for earlier interventions and better management strategies [73]. Moreover, the tools can better profile the pathogenic features and behaviour, thereby supporting targeted solutions and prevention methodologies [23]. But there can also be negatives to dependence on sophisticated diagnostic tools and technologies [42]. An important limitation may be the expense of acquiring and maintaining these tools, which can present a significant barrier to entry for many healthcare facilities (particularly in resource-limited settings) [21, 90]. Additionally, there might be some issues regarding the accuracy and dependability of certain diagnostic instruments that would result in either a false positive or a false negative [90].

1.2.2 Addressing Uncertainties

Understanding stochastic models in epidemiology is crucial for predicting and managing the spread of emerging pathogens [147]. Stochastic models incorporate natural randomness in disease transmission, incorporating aspects like individual behavior, demographics and environmental fluctuation (across time only) [147]. Stochastic models like these are essential to account for the numerous sources of stochasticity and heterogeneity in disease transmission dynamics, thereby providing more informative risk assessments [5]. A key feature of stochastic models in epidemiology is that they explicitly incorporate individual-level heterogeneity, capturing differences among population members with regard to susceptibility, infectiousness

and patterns of contact [4]. Stochastic models can help to capture these individual level differences, providing important results for prediction of the effects of control measures and interventions under different patterns [25]. However, stochastic models can quantify the probability of establishment and growth in small populations, whereas deterministic models typically predict a constant basic reproduction number and may overlook rare or limited-size events that are not captured by average (mean) behaviour [5]. Furthermore, explicit representation of stochasticity in disease transmission models allows the assessment for the effects under partial observability [147]. This is especially true for emerging pathogens, with sparse data and rapidly evolving epidemiological context making decision-making challenging [31]. Stochastic models can enable identifying robust control measures that are adaptive to potential future states, and in doing so increase the resilience of public health systems against epidemic threats [31].

We believe that this characteristic makes stochastic models valuable tools for dealing with properties inherent in pathogen dynamics and management outcomes [127]. Pathogen spread in the environment and agricultural context can be a variable of uncertain nature, with control measures being only potentially effective during certain periods or over thresholds; these models allow for this by varying regions where different diffusivites are defined [26, 32]. Integrating stochastic models into decision support systems should provide land manager with an idea of the consequences using different management approaches [71]. By better understanding these underlying mechanisms, land manager can evaluate the durability of their solutions and are able to create more resilient strategies that improve the effectiveness as a whole [114].

1.2.3 Risk Attitudes in Pest and Disease Response Strategies

Risk perception and communication are critical yet often underemphasised components in the development and implementation of pathogen response strategies. Although technical models and scientific assessments provide valuable information on disease dynamics and management effectiveness, these responses can become narrowly technocratic, not

taking into account how affected communities understand, react to or trust the proposed interventions [25]. Public resistance or non-compliance may arise not because strategies are biologically unsound, but because they are perceived as socially intrusive, culturally inappropriate, or psychologically alarming. This disconnect between expert-driven policy and community-level acceptance can undermine even the most well-designed interventions. Therefore, integrating risk perception research, stakeholder participation, and effective risk communication into decision making processes is essential to ensure that disease management policies are not only epidemiologically sound, but also socially viable and ethically legitimate. This study aims to address this gap by exploring how perception of risk shapes public responses to threats to plant health and how such insights can be incorporated into adaptive and participatory management frameworks.

Informed by stakeholders and by how they perceive risk taking, land manager can design a variety of communication strategies that also target various stakeholder groups to gain support for the implementation of disease management interventions [6, 26]. Individuals' risk attitude profiles can also help identify and resolve any inherent trade-offs in allocating resources for pathogen response [26]. By knowing the risk preferences of the stakeholders, decision-maker can focus on the relevant stakeholders who most value the response measures in terms of their risk attitudes and then allocate resources accordingly to improve the overall efficacy of the ongoing pathogen control efforts [26, 76]. Individuals or organizations who are afraid of risk will also be more inclined to take proactive measures and invest in disease control interventions that mitigate known risks [25]. This latter finding reinforces the point that cross-disciplinary collaborations between economics and epidemiological disease modelling are a means of advancing our quantitative knowledge on emerging pathogens [5].

Assessment of risk attitudes is an important component of any future strategies implemented to manage and control pathogens [5]. Risk attitudes are critical in the design and execution of pathogen response solutions [5]. This diversity of risk tolerance among stakeholders and decision-maker has significant implications for the choice and design of management interventions [26]. Assessment of willingness to take risks can ensure that suitable response

strategies are consistent with the risk tolerance of stakeholders, thus increasing adoption success rates and compliance [26]. Regular evaluation of risk attitudes of stakeholders and adjustment of response strategies ensure that pathogen response measures remain appropriate and effective [26]. However, doing so can be critical to maintaining relevance among decision-maker in shifting agricultural landscapes [26]. Integrating risk preferences into pathogen response strategies can improve their real-world applicability, stakeholder adoption, and cost-effectiveness by aligning management actions with the behavioural tendencies and decision-making constraints of affected individuals or communities [49]. This approach enhances not only compliance but also promotes biologically and economically sustainable disease control in agriculture [49].

Addressing this global challenge, *Lodge et al. (2016)* discuss the impact of invasive species on ecosystems, economies, and human health and provide information on the development of effective policies and management strategies [85]. *Lodge et al. (2016)* provides a broad review of the policies, risk assessments, and economic considerations related to invasive species [85]. The authors emphasise that the economic impacts of invasive species extend far beyond direct damages to agriculture, fisheries, and infrastructure, encompassing indirect costs such as the loss of ecosystem services and long-term ecological degradation [85]. One of the central arguments of the review is the cost-effectiveness of prevention over reactive control, supported by economic modelling that shows how early intervention can significantly reduce long-term management expenses [85]. The paper also underscores the importance of bioeconomic models that integrate ecological dynamics, uncertainty, human behaviour, and the costs and benefits of different control strategies to support risk-based decision-making [85]. Additionally, *Lodge et al. (2016)* advocate for the use of economic instruments and incentives, such as subsidies, taxes, and liability frameworks, to encourage compliance and shared responsibility among stakeholders [85]. Crucially, they point out that current economic assessments often overlook non-market values, such as biodiversity and cultural heritage, resulting in systematic underinvestment in invasive species control [85]. The authors call for a more comprehensive approach to economic valuation, particularly through the inclusion

of ecosystem services in cost-benefit analyses [85]. Their review reinforces the need for interdisciplinary approaches that combine ecological risk analysis with economic reasoning to guide policy and resource allocation in invasive species management [85].

Most stochastic programs minimise an expectation criterion, which maps each scenario with a probability of occurrence to the expected cost [99]. Research has been conducted that used uncertainty to generate input data in the context of outbreaks of diseases [151]. Rojas et al. 2021 applied a probabilistic decision analysis to evaluate the viability of climate adaptation investments in sweet cherry orchards in Central Chile [116]. Risk management strategies in sweet cherry production have been predicted to produce similar results [116]. Their study, which integrates expert elicitation and Monte Carlo simulation, highlighted regional differences in the expected profitability of installing protective covers [116]. The approach illuminated how investment risk is mediated by local climatic factors such as frost and rainfall patterns. The literature suggests that risk management tools, including decision support systems, can provide farmers with crucial information to manage risks [116, 140]. Concerning the evaluation of risk management tools, despite the fact that [148] acknowledged long ago the usefulness of such tools when combined with expert knowledge to assist plants in making decisions on risk management. Due to the genetic similarity or even identity of many heather plants and frequent fungal infections, heather growers are exposed to high financial risks [119].

The utility function can be used to transform probabilistic estimates of possible outcomes into expected utility values based on a decision-makers level of risk aversion [22]. Individual preferences for risk are expressed by the concavity or convexity of the utility function, ranging from 'risk-taking' to 'risk aversion'. Considering the expected utility analysis, decision-makers can obtain detailed decision support that takes into account the risk preferences [122]. Decision support systems can be integrated with risk perceptions and preferences to increase the usefulness of disease management in agricultural production systems [48]. Economic experts and people in business generally believe that increased risk or increased risk aversion should push them to make better decisions [34]. For instance, in the context of agricultural disease

management, farmers facing uncertainty about invasive pathogens may adopt precautionary measures such as crop diversification, investment in resistant varieties, or early chemical treatments, despite the additional costs [49]. This behaviour aligns with risk-averse preferences and is supported by empirical studies showing that risk-averse farmers are more likely to adopt biosecurity measures to avoid uncertain losses [49]. Similarly, businesses facing supply chain risks often adjust their operations such as sourcing from multiple suppliers or increasing inventory to hedge against potential disruptions, reflecting the strategic adaptation under risk [138].

1.2.4 The Research Methodology

In this study, we explore the relationship between epidemiological, economic and stochastic models in plant management, mainly focusing on the impact of diseases on management decisions. Our analysis aim to assess the direct impact of pest and diseases on plant management and how/when to control to the diseases spread. To achieve this, we consider a range of parameters such as land area, time horizon, and invasion rates, each playing a pivotal role in shaping the dynamics of pest and disease invasion and control. Computer simulations complement analytical models by providing a dynamic framework to test hypotheses and visualise the spread and control of pathogens under various scenarios. However, scenarios in this context refers to the different conditions, assumptions, or intervention strategies that can be simulated to explore how a pathogen might spread or be controlled. For example, computer simulations in the management of plant pathogens will consider how disease might move across farms, regions, trade, and the level of control measure applied at varying times and intensities. These simulations use complex algorithms to mimic real-world behaviours and interactions. One of the key advantages of computer simulations is their ability to incorporate randomness and stochastic events, which are often present in real-world scenarios.

Sensitivity analysis examines the contribution of each predicted parameter in both the deterministic analysis and stochastic process. The benefit of this model is that we can undertake extensive sensitivity analysis to illustrate how different epidemiological and economic

components of the system change the optimal management strategy. When the pathogen arrives and spreads, it reduces the values of these combined benefits flows over time. The primary purpose of this model is to understand how the optimal level of precautionary management (PM) is changed when the economic or epidemiological parameters are changed. The deterministic model was modified to include random arrival using numerical simulation to find the optimal management strategy between the deterministic and stochastic results. We developed a management strategy based on cost transformation into disutility. Considering the social, economic, and environmental aspects of risk aversion, a basic model of the sustainable application is developed. Explores the development of innovative disease surveillance systems, sustainable farming practices, and biotechnological solutions that can empower farmers and land manager to protect plant health and secure our agricultural future [100]. Inadequate management also contributes to increased pesticide use, which can have adverse environmental and human health effects [84].

1.3 Methods and Definitions

In this thesis, we use both deterministic and stochastic model to study the dynamic of plant pathogens. Here, we introduce some major mathematical concepts that we use throughout the thesis in this section.

1.3.1 Deterministic Model

A deterministic model is a mathematical representation of a system or process that assumes the same input will always lead to the same output and does not consider fluctuations or probabilities [24]. A deterministic model can be continues time model (Ordinary Differential Equations, Partial Differential Equations, Delay Differential Equations) or discrete-time models [124]. For instance, *Kermack* and *McKendrick's* seminal compartmental model developed in 1927, which uses a system of ODE, is a classic example of a deterministic model used in epidemiology to study disease spread within populations [149]. The model compartments

people into fixed disease states able to develop new infections at rates estimated from the population and tracks how individuals flow between these compartments [149].

Deterministic models are used to simulate pest-pathogen population dynamics and the performance of control strategies in plant pest disease management [24]. These models rely on well-established biological and environmental relationships, such as the influence of temperature, humidity, and other abiotic factors on pests or pathogens' life cycle and behaviour [26]. By incorporating these relationships into a mathematical framework, deterministic models can be used to forecast the spread and impact of plant pests and diseases, enabling practitioners to develop targeted and effective management strategies [26, 80, 132]. These methods are increasingly being recognized as a platform that allows practitioners an opportunity to develop focused management strategies with maximal effectiveness; such approaches may offer means for developing more precise decision support tools from minimum epidemiological data [26].

Deterministic models have been applied in various aspects of plant pest and disease management, such as predicting the onset of diseases to assessing risk from invasive species introduction [24, 26, 132]. As noted in the literature, these models help to facilitate insect pest management (IPM) by indicating when and where control should best be effected using pesticides, biological control agents or cultural practices [24, 40].

1.3.2 Stochasticity

The concept of stochasticity is central to modelling plant pest and pathogen dynamics because it captures their intrinsic randomness and unpredictability as biological systems [26]. The term "stochastic" is used to describe the random characteristics of events in contrast with deterministic ones because their courses are not fully determined but rather on those that are influenced by many chance factors [12, 26]. Examples of stochasticity in plant pests and diseases include the demographic fluctuations or cycles observed with some pathogens, as well as outbreaks that appear to occur at random [26]. For example, the effects of some abiotic factors are predictably frequent in plant-pest systems, others like climate change, have the

potential to induce other sources of uncertainty in plant-pest dynamics, ultimately resulting in greater year-to-year variability in both incidence and severity of disease [26]. Similarly, *Madden et al.* argue that plant disease epidemics are stochastic events with respect to their timing of appearance, and the level at which they occur can vary significantly depending on chance occurrence [87].

For deterministic, the state of the population at some time t can be predicted with complete accuracy if the prior state of the population at a previous time is known [74]. Such models are useful for large populations, where random effects are averaged out according to the law of large numbers [14]. Deterministic models are helpful when we are studying a big population with a lot of participants, where (after some transform) random effects cancel out on average by the law of large numbers [108]. However, they do not provide accurate results with little numbers since random procedures are described as relations [108]. Stochastic models can account for the inherent randomness in disease transmission, which is especially important in the early stages of an outbreak or in small populations [108]. By incorporating random fluctuations, stochastic models can potentially provide more accurate short-term and long-term epidemic forecasts [108]. These models can help assess the risk of observing a major epidemic outbreak, which is particularly valuable for public health decision-making [108]. Stochastic models can capture a variety of early epidemic growth profiles, ranging from sub-exponential to exponential growth dynamics, which have been observed across different infectious diseases [108].

The importance of stochasticity in plant pest and diseases research is demonstrated by *Garrett et al* on climate change effects to plant pathogens and diseases [26]. This study shows that variability and unpredictability of environmental conditions due to climate change will increase the stochasticity in transmission dynamics, which has very important implications for disease control or mitigation strategies [26]. The consequences of stochasticity on risk assessment and decision-making are also central to plant pests [26]. Stochasticity is a key feature of plant pest and disease dynamics, the consequences of which have wide implications for both our understanding, prediction and management [104].

1.3.3 Precautionary Management (PM) Strategy

Precautionary management (PM) is a management strategy that reduces the probability of the arrival of plant pests and diseases and makes proactive decisions using the best available information to protect against or reduce the risks from pest and disease outbreaks [16, 26, 50]. The PM strategy acknowledges that the dynamics of the trade and the introduction of non-Native Species (NNS) create significant risks to plant health [40]. The strategic framework behind this approach is that it is more efficient and less costly to stop the introduction of pests and diseases at a future time than to control them once they become established [37].

The fundamental principle of this Precautionary Management (PM) strategy is to identify and address the potential threats proactively rather than depend purely on responsive approaches [26]. This involves a full evaluation of the plant production system, any potential pest and disease risks identified and appropriate measures taken to minimise risks before they appear [50]. A precautionary management strategy involves actively surveying the forest, maintaining vigilant biosecurity surveillance to catch threats early, and setting clear tolerance levels for pests and diseases [50]. Together, these steps help reduce both the chance that harmful pests will arrive and the vulnerability of the system to damage. By focusing on early detection and timely action, this approach aims to stop problems before they grow, protecting plant health more effectively [50]. By prioritising prevention over control, this strategy aims to minimise the need for costly and potentially harmful interventions, such as applying pesticides [26]. Precautionary Management forms a foundational part of Integrated Pest Management (IPM), which combines multiple complementary tactics to manage pests and diseases effectively. Although PM focuses on early detection, risk reduction and prevention of pest establishment, IPM integrates these Processionary management with targeted control strategies when needed, in order to optimize economic efficiency and minimize risks to human health [50]. These strategies have been widely promoted to reduce the risks of increasing globalisation of trade and changes in the dynamics of pests and diseases through climate change [26, 40].

1.3.4 Reactionary Management (RM) Strategy

The reactionary management (RM) strategy is a comprehensive approach to addressing plant pests and diseases that highlight a rapid reduction in land benefits once emerging threats is established [40]. The main elements of a reaction management strategy are timely detection and diagnosis, integrated pest management (IPM) methods, and judicious application of disease or pest control means [40]. Early detection is important; it allows quick action before infestations or outbreaks can become widespread and do much damage. This can often be done with tools such as remote sensing and automated monitoring systems to rapidly detect new threats [40].

If a specific pest or disease is noticed, a reactionary management strategy focuses on integrated pest management (IPM) strategies. These actions can involve the use of biological, cultural and chemical control methods as well as precautionary measures [40, 92]. Another important part of this management practice is how control methods are strategically deployed. Plant health professionals need to weigh the pros and cons of different control measures, balancing issues such as environmental considerations (biodegradability), cost-effectiveness (need for high virulence production) and resistance development risk [57]. With a Reactionary Management Strategy, plant health professionals can quickly and appropriately address new threats to plant health when they arise, leading to reduced further loss of crop production or damage to the environment [57].

With the rapid deployment of biological control agents to combat an invasive insect pest through the use of an RM Strategy, plant health professionals can respond swiftly and effectively to plant pest and disease threats, helping to minimise economic losses and protect valuable agricultural and natural resources [26, 112]. Early detection is very important in the management of plant health because it serves two key purposes. On one hand, ongoing surveillance helps catch pests or diseases as soon as they appear, ideally before they have a chance to take hold. On the other hand, once a pest or disease is found, early detection triggers processionary control efforts to stop it from spreading and causing more damage. In this way, early detection acts as a crucial link between processionary control before they

start and reactionary control once they have arrived and causing serious damage, allowing for faster and more effective responses [40]. As global trade and climate change continue to drive the emergence of new plant health threats, the need for effective reactionary management strategies will only become more critical [125, 132, 145].

1.3.5 Previous Epidemiological and Economic Modelling Studies

A substantial body of work has examined the role of epidemiological and economic modelling in understanding the spread and management of invasive pests and plant diseases. Epidemiological models have long provided a framework for exploring the dynamics of pathogen spread and the impact of control interventions [29, 51, 52]. These models typically distinguish between primary infections, introduced from external sources, and secondary infections, which occur through local spread [29, 51]. This distinction has important management implications, precautionary measures reduce the probability of primary introductions, while detection, containment, or eradication strategies target secondary transmission once disease is established [52].

From an economic perspective, much attention has focused on weighing the costs of precautionary actions against the damages or costs of control after establishment [82]. Famously captured this in the phrase 'an ounce of prevention is worth a pound of cure', formalising prevention as a decision under uncertainty [82]. Similarly, Olson and Roy (2006) and Burnett et al. (2006) modelled the trade-offs between precautionary spending and expected damages, showing that prevention is not always optimal if invasion risks are low. Finnoff et al. (2007) and Epanchin-Niell and Hastings (2010) extended this by developing dynamic optimisation approaches, highlighting that early detection and rapid response can be cost-effective alternatives to high levels of pre-border prevention [37, 41].

In the specific context of plant diseases, bioeconomic models have been developed to assess management strategies for pathogens such as citrus canker, sudden oak death, and banana Xanthomonas wilt [98, 139]. These studies integrate epidemiological dynamics with economic decision-making, demonstrating how the timing, intensity, and type of interven-

tions alter both epidemiological outcomes and economic efficiency [98]. However, relatively few explicitly connect prevention as the reduction of the probability of primary introduction with alternative strategies such as early detection or reactive management in a unified framework. This is the gap that the present thesis addresses, by explicitly modelling the trade-off between precautionary investment to reduce introductions and reactive responses after disease establishment, while also incorporating the role of uncertainty and risk preferences of decision makers.

1.3.6 Precautionary management versus reactive control

The management of emerging invasive species, whether pests or pathogens, remains a central challenge in environmental policy and ecological research. A key debate in this field is the relative effectiveness of precautionary management (PM) compared to reactive strategies such as early detection and control. The rationale for PM is straightforward, once an invasive species becomes established, both the ecological and economic damages tend to escalate rapidly, and in many cases these impacts are irreversible, therefore, interventions at an earlier stage can reduce long-term damages and overall welfare losses [37, 82]. These studies emphasise that the value of PM lies not only in lowering the probability of invasion, but also in reducing the uncertainty about future damages [37, 82]. Once a species has spread beyond an initial point of entry, eradication often becomes unfeasible, leaving land managers with only partial and ongoing control options. Thus, the literature positions PM as a strategy that shifts investment to the earliest stages of the invasion process, where interventions can have the greatest long-term impact [37, 82].

The case for reactive strategies rests on the premise that prevention, while effective, is often costly and may not always be economically justified. Olson Roy (2006) argue that when the probability of invasion is low, investing heavily in pre-border precautionary measures could represent an inefficient allocation of resources [102]. Instead, if incursions are rare events, resources might be better spent on systems that enable early detection, rapid response, and targeted eradication once an invasion occurs [102]. Burnett et al. (2006) illustrate this trade-

off with examples from Hawaii, showing that in some contexts, containment and eradication following establishment can be more cost-effective than continuous, high-cost prevention, particularly when species spread can be detected early and eradication success is relatively high [19].

This reasoning has motivated a growing body of bioeconomic modelling that explicitly compares the relative value of prevention versus control. For example, Epanchin-Niell and Hastings (2010) demonstrate that the optimal strategy depends heavily on invasion dynamics, when spread rates are rapid, precautionary management is generally preferred, but under slower spread or limited establishment potential, post-invasion control can dominate as the cost-effective option [19]. Similarly, Finnoff et al. (2007) develop stochastic models that capture the uncertainty inherent in invasions and show how managers face an intertemporal trade-off between 'upfront insurance' (prevention) and 'wait-and-see' (control) strategies [41]. Their findings highlight that the optimal balance depends on factors such as the probability of introduction, the costs of surveillance and eradication, and the ecological consequences of delayed action [41].

The case for reactive strategies is based on the observation that prevention can be expensive, and if the probability of invasion is low, the expected damage may not justify high precautionary expenditure [101, 19]. In such cases, investments in efficient surveillance systems and rapid eradication capacity may provide a more cost-effective solution, allowing resources to be allocated only when invasions occur. The literature has therefore framed the problem as one of trade-offs between pre-border or precautionary investment and post-establishment control [37, 41].

The rationale for PM is grounded in the observation that once an invasive species becomes established, both ecological and economic damages escalate rapidly and are often irreversible [82]. Leung et al. (2002) captured this idea in their widely cited argument that 'an ounce of prevention is worth a pound of cure', demonstrating that investment in prevention can substantially reduce long-term costs by avoiding irreversible ecological impacts [82]. Similarly, Epanchin-Niell and Hastings (2010) developed a dynamic bioeconomic framework showing

that prevention, while costly, can minimise long-term welfare losses by reducing both the likelihood of establishment and the severity of subsequent damages [37]. These studies underline that the value of PM lies not only in lowering invasion probability but also in reducing uncertainty, since once spread occurs, eradication is typically infeasible and control becomes an indefinite commitment [82].

By contrast, a body of literature has highlighted the limitations and costs of PM, particularly when the probability of invasion is low or the costs of precautionary measures are high. Olson and Roy (2006) argued that under these conditions, heavy investment in prevention may not be economically justified, as resources may be wasted on threats that never materialise [101]. Burnett et al. (2006) similarly emphasised that costly pre-border interventions may deliver little net benefit compared with strategies focused on efficient post-border surveillance and rapid eradication capacity [19, 37]. From this perspective, reactive strategies can offer a more cost-effective allocation of resources, since they target actual invasions rather than potential ones, allowing decision-makers to balance prevention with flexibility in responding to realised risks [37].

1.3.7 Economics aspects of Prevention and reaction measures

International trade is a significant driver of biological invasions worldwide [65, 106]. Among these are supply chain disruption, economic loss, and an increased risk of black-market smuggling [36, 65]. The paper by *Springborn et al.* concludes that a holistic, integrated approach is required for managing risks associated with international trade [36]. The authors have identified several key processes driving the facilitation of introductions through trade, such as changing global species distributions, levels or types of traded commodities produced and consumed in the rest of the world (trade-partner effects), evolving technologies for quarantine pest risk analysis and surveillance to protect local biodiversity from invasions [36]. They also illustrate the heterogeneity across imported commodities and trade routes, leading to increased risks about movements from bio-geographic regions of similar climatic conditions [36]. They stress the need to target analyses and management resources to address

how likely and impactful a biological invasion event is [36]. The paper also stresses the case for corresponding strategic trade policies to help contain invasive species [36].

For better management of the risks of invasive species in international trade, the recommendation is to use scaled pest and disease risk assessments to assess the likelihood of a species arriving and establish a potential impact [36, 65]. In the context of animal health, WTO member countries are required to perform a risk assessment before restricting trade with another country, and a similar principle applies in plant health under the WTO Sanitary and Phytosanitary Agreement. In the UK, plant pest risk assessments are typically conducted by Defra's Plant Health Risk Group, with technical input from FERA and other agencies, rather than APHA. For better management of the risks of invasive species associated with international trade, several authors have recommended scaling up pest and disease risk assessments to assess the likelihood of the arrival, establishment, and impact of species [36, 65]. In addition, pre-border biosecurity measures for treatment, such as shipment clearances, have been identified to reduce the risk of non-native species (NNS) introduction [65, 137]. The review points to the complexities associated with biological invasions related to international trade [36, 137]. Using biological invasions *Springborn et al.* explore the bidirectional exchange between trade and introduction of invasive species via international commerce pathways - namely, both import/export processes originating in different countries [36].

1.3.8 Net Present Value (NPV)

Net Present Value (NPV) is a tool for economic/financial decision making because it helps quantify the costs and benefits (including avoided losses) of interventions over time, guiding resource allocation. The application to reducing pest risk in plants focuses on direct monetary flows, such as treatment costs, loss of crop value, environmental impact, and long-term sustainability [40]. NPV calculates the present value of all future cash flows associated with a particular investment or decision, discounted at an appropriate rate [40]. We use NPV to assess the expected monetary cost of different management practices [145] regarding plant pest and disease control. For example, by managing pests effectively, farmers can expect

to earn more over time thanks to fewer infestations and reduced costs for treatments [47]. Studies have shown that NPV can be used to evaluate the profitability of pest control policies, estimate optimal investments in plant biosecurity systems, and assess costs or benefits related to the economic feasibility of specific management tactics [40].

Plant pathogens are well known to diminish the ecological diversity of plant ecosystems and ecosystem services provided by plants[86, 129]. A case study by *Pelini et al. (2021)* evaluating the NPV of invasive plant pests found that prevention greatly outweighed the cost in most scenarios [40]. Another paper emphasised the suitability of NPV for cost-benefit analysis in plant disease control engineering projects [60]. The use of NPV analysis is a significant advancement in the economic evaluation of plant pest and disease management strategies [60]. Managers and engineers in the agricultural sector are concerned with the bottom-line impact of their decisions, and NPV provides a way to quantify the long-term financial outcomes [47].

Using this framework, inputs into an cost-benefit analysis within the crop biosecurity domain include upfront costs of deploying control tactics and expected probability and severity of future pest or disease incursions, respectively, together with estimated economic damage that would be incurred [78]. Uncertainty in these estimated values can be introduced by performing a risk analysis like the Monte Carlo simulation, which results in a more reliable calculation of NPV [47, 78]. There is growing pressure at the federal level for institutions to incorporate NPV in their evaluations [40]. State-level decision-makers should consider adopting it too, as it can help them maximise returns, safeguard plant health, and increase overall productivity [40]. With changes in international trade patterns and the likely shift of the pool of potential invasive pests, there is a case for more dynamic NPV (real-time data systems) assessing to inform biosecurity strategies [40].

1.3.9 Risk aversion and exponential utility functions

Risk-averse agents will assign less value to the activity at each level of expected return because they are more cautious about potential utility outcomes [2]. As a benchmark for this

application in the context of decision-making under risk and uncertainty, we consider the exponential utility function, which has been used intensively since 1989 [148]. For instance, *Abbas et al. (2013)* analysed the individual investor behaviour with a simple but important question: how does a decision-maker's attitude to risk change the value they place on getting extra information? In many applied problems (environmental management, investments, biosecurity, etc.) analysts compute the *value of information* (VOI) assuming a risk-neutral decision-maker [2]. *Abbas et al. (2013)* point out that real people (and organisations) are often risk-averse, and that risk aversion can change not only whether someone pays for monitoring, but also how valuable different types of information are to them [2]. The authors therefore set out to characterise, rigorously, how VOI depends on the decision-maker's utility (risk) function [2]. A study by *Bedoui* and *BenMabrouk (2017)* gave a startup that used the exponential function on asset pricing and risk aversion [9]. The *Baker 2017* study optimised spatial and temporal resource allocation using an exponential utility function for invasive species control [7]. The study found a better understanding of the underlying problem and how to explicitly account for risk aversion and preferences for uncertain outcomes, such as using an exponential utility function in this study, would help develop optimal management strategies [7].

The exponential utility function is a mathematical representation of risk aversion that is commonly used in economic analysis [2]. It is typically expressed as $U(x) = -exp(-ax)$, where $U(x)$ represents the utility or subjective value of an outcome x [2, 9]. This function exhibits several key properties that make it useful for modelling decision-making under uncertainty [9]. First, the exponential utility function exhibits diminishing marginal utility, meaning that as the value of x increases, the increase in utility becomes smaller and smaller. This property reflects risk aversion, as individuals tend to value gains and losses asymmetrically, placing a higher importance on avoiding losses than on pursuing gains [1, 15]. Second, the exponential utility function captures the trade-off between risk and expected return [1]. Risk management strategies, including early detection and rapid response measures are essential to limit the risk of entry for an invasive species, but equally important is the improvement in capabilities at

ports of entry to increase surveillance and detection ability as well as efficiency by developing rapid response protocols that reduce cost while preventing establishment if prevention fails [38].

The exponential utility function was also used in various studies about the bioeconomic impacts of plant pests and diseases [17]. The effects of tree pests and diseases on ecosystem services have been assessed using the exponential utility function [17]. They eventually determined that including the risk aversion in the on-farm model improved (reduced bias) the estimated economic costs of these challenges [17]. There is a growing interest in using the exponential utility function for investigating plant pests and diseases as well as researching other areas through, e.g., economic and asset pricing studies where it has been applied in recent years [7, 17].

1.3.10 The Knowledge Gap

Research and studies on plant health management have been ongoing for many years, but there are still some knowledge gaps and challenges that researchers are working to address [93]. With globalisation and increased trade, new pests and diseases are constantly being introduced to new regions [93]. Research is needed to quickly identify and develop management strategies for these emerging threats before they become widespread and cause significant damage to crops [93].

While both epidemiological and economic models have provided valuable insights into the management of plant pests and diseases, certain limitations remain in how these two perspectives are integrated. Epidemiological studies have been highly effective in describing the dynamics of pathogen establishment and spread, and in identifying where interventions might slow or stop epidemics [51, 29]. Meanwhile, economic studies have clarified the costs and benefits of prevention versus reactive strategies, showing that under some conditions early detection and eradication may be more cost effective than high levels of precautionary investment [82, 101, 19]

However, most of this literature treats prevention and reaction in isolation or assumes that

prevention only reduces damages rather than explicitly reducing the probability of primary introductions. Similarly, few models formally integrate risk preferences or decision-making under uncertainty into the bioeconomic analysis, despite their importance in real-world policy contexts [49]. In the specific case of plant diseases, only a handful of bioeconomic studies have linked epidemiological processes with economic decisions in applied contexts, such as citrus canker, sudden oak death, and banana wilt [98, 139].

This thesis addresses these gaps by developing a unified framework that explicitly connects prevention measures, which reduce the probability of primary introductions, with reactive measures such as early detection and control of secondary spread. Despite this growing body of work, few studies explicitly link prevention as a reduction in primary introduction with early detection and reactive management in a unified framework. By embedding these within both deterministic and stochastic models and incorporating decision-maker risk attitudes, this research contributes to a more realistic and policy relevant understanding of the trade-offs between precautionary and reactive strategies for emerging plant pests and diseases. This is the focus of the present thesis.

1.4 Thesis Outline

The thesis consists of six chapters. Chapter One introduces the research topic, its significance and literature review. This Chapter provides a brief overview of the research's purpose and explains the research problem's relevance and importance. It aims to orient the reader to the research topic, justify its importance, and provide a clear framework for the study's objectives and methodology.

Chapter Two (2) introduced the mathematical model with the detailed economic objective function. This Chapter defines all the parameters and terminology used in this thesis and their baseline values. In this thesis, we created a generalisable, bioeconomic model to examine how to deploy resources across two management strategies to minimise the expected economic costs of an emerging pathogen on a border of a country, which produces a flow of economic

and environmental benefits. The bioeconomic model was used to understand how best to deploy a precautionary management (PM) strategy to reduce the effect of an emerging pathogen. The key policy question is how to reduce the potentially irreversible impact of emerging pathogens on the environment. One of the methods studied in this thesis is to execute precautionary management strategies to reduce the rate of pathogen emergence. This involves implementing biosecurity measures to reduce (or eliminate) the risk of introducing the pathogen from outside the country (or region).

In Chapter Three (3), we established a framework to understand the dynamics of disease spread within plants and the economic implications of various management strategies. Building upon this foundation, we apply reactionary management (RM) and precautionary management (PM) strategies. The aim is to ascertain the optimal level of PM that minimises the present value of costs arising from disease impacts over a fixed time horizon, considering the availability of RM once a pathogen is detected within the forest under investigation. The application of RM strategies once the pathogen has been detected is used to reduce the future rate of spread and damage caused by the disease. These reactive actions are intended to reduce present and future economic and environmental losses caused by the introduction of pathogens within the forest. The numerical analysis shows the outcome of different management strategies in the area of infected border over time.

We introduce uncertainty to the deterministic model created in chapter two (2). Our approach to incorporating uncertainty in pathogen arrival in Chapter Three (3) involves two distinct methods. Firstly, we utilise the absolute value of a normal distribution to introduce random variability into the primary infection rate, P. This method allows us to simulate scenarios where P fluctuates around a mean value, reflecting real-world conditions where transmission rates are not constant. Secondly, we employ a log-normal distribution to model the baseline primary infection rate. This choice is motivated by the log-normal distribution's ability to represent a wide range of possible outcomes, especially when the variable of interest can vary significantly. This chapter aims to find the optimal level of PM that minimises the present value of costs arising from disease impacts over a fixed time horizon with random ar-

rival of the disease rate. We compare the two different stochastic results with the deterministic results.

In Chapter Five (5), we delve deeper into the complexities of decision making under uncertainty by incorporating an exponential utility function into our existing stochastic model. This approach allows us to analyse the expected outcomes and the decision-maker's attitudes towards risk. The Chapter's aim is to minimise the loss from infection through an adapted utility-based objective function and to discern the degree of risk associated with different management strategies. The core of this Chapter revolves around the concept of exponential utility functions, both negative and positive, to capture the risk preferences of decision-makers, typically represented by farmers in our context.

We conclude this thesis with Chapter Six, which summarises our findings and highlights areas for future research.

Chapter 2

Deterministic Model and Application to Precautionary Management Strategy

In this chapter, we introduce the general mathematical model in Section 2.2 and detail the economic objective function in Section 2.3. The parameter and variable descriptions, along with their baseline values, can be seen in 2.4. Section 2.5 presents the results of our sensitivity analysis. Sensitivity analyses were carried out to explore how model outcomes change under different conditions of disease spread and management costs. The results are discussed in Section 2.6, where we give our concluding remarks. All the parameters and variables used in this thesis are present within the text, and can also be found, along with their baseline values, in Section 2.4.

2.1 Introduction

This chapter introduces the general mathematical model in Section 2.2, which outlines the precautionary management strategy (PM). The Precautionary Management (PM) Strategy involves making decisions in anticipation of potential future losses and identifying and mitigating risks before they materialise. This approach is particularly relevant when dealing with scenarios before pathogens are detected, prompting immediate action alongside ongoing

precautionary strategy. The objective is to minimise the present value of the potential future costs associated with the plant pathogens, considering both direct and indirect impacts on the forest's value. We consider the economic implications of PM strategies, providing a basis for understanding their role in forest health and economics. Losses refer to the reduction in yield or forest productivity caused directly by disease. Damages represent the economic valuation of those losses (e.g., monetary impact on timber markets or ecosystem services). Costs, on the contrary, denote the resources invested in prevention, detection, or control measures. Importantly, prevention costs are not 'losses' but deliberate investments that aim to reduce expected damages.

The model captures the costs of deploying PM and the expected losses from disease, both in terms of market and non-market services. Our model presented in Section 2.2 is based on:

- The infected area of land and the economic cost of the disease impact, and
- The cost of the precautionary management and its effectiveness in delaying the arrival of the pest or pathogen.

We employ a Susceptible-Infected (SI) compartmental model, a framework traditionally used in epidemiological studies. Adapting this model to forestry, we aim to capture the dynamics of disease spread across a forested area. The model focuses on key variables such as the size of the area, the rate of disease transmission, and the evolution of the infected zone over time. Crucially, this model also incorporates strategies for disease management and prevention, allowing us to simulate various scenarios and their outcomes. The following section delves into the intricacies of this model, elucidating how it can be effectively applied to understand and mitigate disease spread in forest management.

2.2 General Mathematical Model

The infection dynamics are governed by a Susceptible-Infected (SI) compartmental model. The time horizon (years), $t \in [t_0, T]$ where $t_0 = 0$ and $T = 100$. The pathogen is assumed to

arrive at time $t = 0$ (years) and subsequently spreads through the forest area $L > 0$ (hectares). The area of the forest that is infected at time t is $I(t)$ (hectares), where $I(t) \in [0, L]$. The baseline primary infection rate, $P \geq 0$ (hectares^{-1}), controls when the pathogen is detected in the forest, and the parameter P represents the proportion of the forest/land that is initially infected. We define primary infection as the rate at which new infections arise from an external source of inoculum (e.g. imported or environmental pathogen pressure). It is therefore a flow parameter rather than a timing parameter. The baseline secondary disease transmission rate $\beta \geq 0$ (year^{-1} hectares^{-1}) controls the rate of spread of the pathogen within the forest. The secondary transmission rate (β_s) represents the rate of spread resulting from host-to-host transmission within the forest once infection is established. Together, these rates capture both the risk of initial introduction and the potential for subsequent local amplification of the pathogen.

Precautionary management (PM) decreases the rate of primary infection by δ, where $\delta \in [0, 1]$. For $\delta = 1$, there is no reduction in the primary disease rate, and when $\delta = 0$, the pathogen is stopped from arriving completely. We assume the pathogen is detected when the infected area reaches a detection threshold level, $I_1 > 0$. This occurs at time $t = \tau(\delta)$, which depends on δ, since PM influences disease dynamics.

$$\boxed{\left(\frac{dI}{dt}\right)_A = \beta(L - I(t))(I(t) + \delta P),\ 0 < t \leq \tau(\delta).} \tag{2.1}$$

Equation (2.1) represents the rate of change of infected area *before* detection, where $L - I(t)$ is the area of the forest which is uninfected (susceptible but under surveillance) and the label A indicates that this correspond to the pathogen spread before detection.

We use the method of separation of variables to solve Equation (2.1), with initial condition $I(0) = 0$ meaning that the forest is initially uninfected (susceptible), which is represented as

$$\boxed{I(t)_A = \frac{L\delta P(e^{\beta(L+\delta P)t} - 1)}{L + \delta P e^{\beta(L+\delta P)t}}, 0 < t \leq \tau(\delta).} \tag{2.2}$$

We assume the pest starts to damage trees when 5% of the forest is infected. The time the

pest starts causing damage, t_{05}, satisfies the condition $I(t_{05}) = 0.05L$ and is discussed further in Section 2.4.1. The choice of 5% is an arbitrary starting point and can easily be altered within the model. By solving Equation (2.2) we find;

$$\boxed{t_{05} = \frac{1}{\beta(L+\delta P)} \ln\left(\frac{L(0.05L+\delta P)}{\delta P(L-0.05L)}\right).} \tag{2.3}$$

It is convenient to re-parametrize the rate of secondary spread by introducing the doubling time T_2, i.e., the time for an unchecked growth of the spread of pathogens to double the initial area. We write;

$$\boxed{T_2 = \frac{\ln 2}{\beta}.} \tag{2.4}$$

The doubling time, $T_2 > 0$, is the time for the exponential growth of the spread of the pathogen to double the initial area.

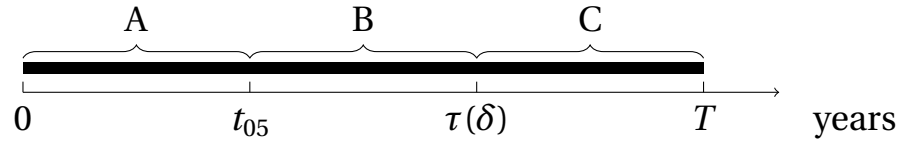

Figure 2.1: Timeline of events, during the interval [0, T].

Consider the timeline in Figure (2.1) as the intervals identified in the model. In this timeline, the PM strategy is applied continuously in intervals A, B, and C, while RM is applied only in interval C. This will help to visualise the three main time-frames in this chapter:

- Interval A: The pathogen arrives at 0 years and subsequently spreads through the forest. PM reduces the rate of pathogen arrival and hence delays the costs incurred by the disease. No loss is associated with this part of the timeline.

- Interval B: The losses start to accrue annually, starting at t_{05} (the time 5% of the forest are infected) until detected at time $\tau(\delta)$.

- Interval C: The pathogen is detected at time $\tau(\delta)$ years. There is a one-off cost of applying the RM to the whole forest when the pathogen is detected. The control needs to be applied annually during interval C.

The effect of PM on the infected area of the forest (through parameter δ) are compared in Figure (2.2). The PM reduces the primary infection by δ. The relationship of an invaded area over time is shown in Figure 2.2(a). The pathogen arrives and spreads according to the graph because PM is not applied ($\delta = 1$). The application of PM only ($\delta = 0.05$ and 0.005) shown in the blue and the red lines delay the appearance of the infection (see Figure 2.2(b)).

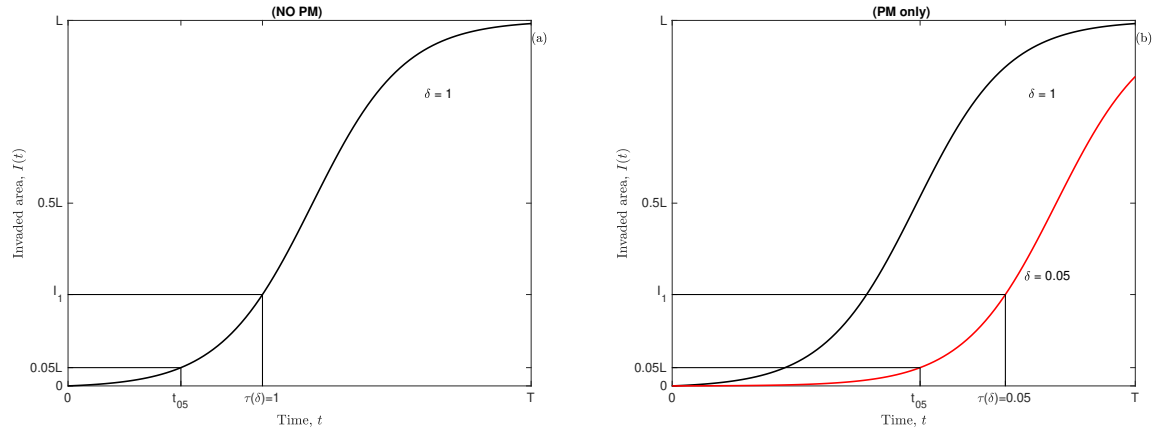

Figure 2.2: The effect of the precautionary management on the infected area of land. The infected area is given by Equation (2.2)(hectares) and shown against time, t (years) when in (a) there is no PM ($\delta = 1$) in black, (b) PM only ($\delta = 0.05$) in red. The time the pest starts causing damage (5% forest infected) is t_{05}, and $\tau(\delta)$ is the time when the detection threshold I_1 is reached.

2.3 Economic Model of Precautionary Management Only

We use an optimisation approach, evaluating management strategies by minimising a defined objective function that incorporates both management costs and disease-induced losses. This approach is used to help the decision maker determine the path of expenditure that minimise the present value of the costs associated with the disease over a fixed interval, $0 \leq t < T$. When

RM is not available (interval A and B in Figure 2.1), the objective function consists of the loss in market and non-market services provided by the land (cost of diseases), and the cost of deploying the PM, as follows.

The loss from the infected area is given by $\rho_I I(t)$ where ρ_I is the annual, per unit area reduction in benefit from the forest (£ hectare^{-1} year^{-1}) and $I(t)$ is the infected area (hectare) at time t. We assume that the loss accrues annually, from interval B (the time where 5% of the area is infected) until interval C (the end time horizon). The reason for setting a non-zero lower threshold where the loss starts to accrue (t_{05}) is because when the primary infection, P, is positive, the pathogen will start to spread from year 0 as the model Equations (2.1) is deterministic. The deterministic model assumes homogeneous mixing, meaning that infection spreads uniformly across the landscape, without spatial clustering. This assumption is appropriate for a baseline model because it allows us to capture the average dynamics of spread and control. Therefore, if the threshold is not set, then the loss caused by the disease will *always* start to accrue at year 0, despite the magnitude of P. By setting a positive lower threshold, we capture the effect of the primary infection when the loss caused by the disease begins to accumulate. Since there is no RM available in this section, the disease will continue to spread across the forest throughout the time horizon according to Equation (2.2).

The cost of deploying the PM throughout the time horizon ($0 \leq t < T$) is $C_p L$, where C_p is the annual per unit area cost of the PM (£ ha^{-1} year^{-1}). The cost of deploying the PM is constrained by the annual per unit area PM budget, Π (£ ha^{-1} year^{-1}), giving the condition $0 \leq C_p \leq \Pi$. The total cost of the PM is assumed to be linearly dependent on the area of the forest. This could represent prophylactic spraying, where the cost depends on the sprayed area. Costs for activities like drone surveillance, on-ground inspections, or satellite monitoring can depend directly on the area being monitored. Larger forest areas require more resources (equipment, personnel, or technology) to cover the entire space adequately. The cost of patrolling scales with area, as larger areas require more personnel, vehicles, or observation points to maintain coverage. The assumption of linear dependence is made for simplicity and to keep the model general since we are not modelling a specific pest or

pathogen. However, other functions could be used instead. For example, PM strategies that stop access to the land area may depend on the perimeter of the forest. PM reduces the rate of primary transmission through δ, (see Figure (2.2)) and we therefore let δ depend on C_p.

The objective function giving the present value of the total costs when RM is not available is given by

$$\boxed{J_p(C_p) = \int_0^T (C_p L e^{-rt})dt + \int_{t_{05}}^T (\rho_I I(t)_A e^{-rt})dt} \tag{2.5}$$

Loss = Cost of PM + Loss if invaded before detection. The first term corresponds to the cost of PM throughout intervals A and B (see Figure 2.1). The second term describes the losses in interval B, i.e. when the pest or pathogen has started to impact but has not been detected. The discount rate is a core concept and an operational tool for valuing time separated costs and benefits. Whether used in public investment appraisal, monetary policy, or financial evaluation, the discount rate serves as a lens through which the future is interpreted and weighed against present needs.

We now need to specify the effectiveness of PM on the reduction of primary disease transmission rate, δ. We assume that the reduction in the primary disease transmission, δ, is proportional to the cost of deploying the PM, C_p, and the greater the expenditure (higher C_p), the larger the reduction in the primary disease transmission (smaller δ). Additionally, we assume that the maximum reduction in primary disease transmission, $\delta = \delta_M$, is achieved when the full PM budget is spent, e.g., $C_p = \Pi$. The parameter δ_M could be interpreted as a measure of the maximum effectiveness of the PM in delaying the time at which the loss caused by disease starts to accrue, e.g., t_{05}. For example, if the PM completely stops the arrival of the pathogen (i.e., it is fully effective), we write $\delta_M = 0$, and when the full budget is spent ($C_p = \Pi$), $\delta = 0$. There are many functional forms that this cost-effort relationship could take. However, for simplicity, we use a linear function of the form;

$$\boxed{\delta = 1 - (1 - \delta_M)\frac{C_p}{\Pi}.} \tag{2.6}$$

The objective function between different numerical activities aimed to minimise the cost

of controlling the losses caused by the pathogen. We carried out a sensitivity analysis to examine the optimal level of C_p that minimises the cost associated with the loss over a fixed time horizon $0 \leq t < T$. The model includes the land manager's objective function, which calculates the the present value of total costs of implementing the PM and the loss caused by the disease over a fixed time horizon. Specifically, we assume that pathogens first arrive at time 0 (t_0) years, start causing damage at t_{05} and are detected at $\tau(\delta)$ (see table 2.1). We distinguish the concept of arrival t_{05} from the "detection" $\tau(\delta)$, the point at which the relevant regulatory authority becomes aware that the pathogen has been detected and is causing damage.

The optimal level of PM, C_p^*, is found by minimising the present value of total costs, Equation (2.5)

$$\boxed{C_p^* = arg \min_{C_p \in [0,\Pi]} J_p(C_p).} \tag{2.7}$$

2.4 Parameter and variable definitions and estimates

Table 2.1: Parameter and variable descriptions, alongside their baseline values.

Parameter	Description	Baseline values	Range of tested values
EPIDEMIOLOGICAL			
t	Time horizon(years), $t \in [t_0, T]$	$t_0 = 0$, $T = 100$	-
L	Area of the forest (hectares)	$L = 1$	-
$I(t)$	Infected area of the forest at time t, $I(t) \in [0, L]$	Equation (2.1)-(**??**)	-
P	Baseline primary infection	$P = 0.005$	$P \in \{0, 0.0005, 0.005, 0.1\}$
β	Baseline secondary disease transmission rate	$\beta = 0.1$	$\beta \in [0, 0.3]$
$1 - \delta_M$	Maximum reduction in primary disease transmission due to PM, $\delta_M \in [0, 1]$	$\delta_M = 0$	$\delta_M \in \{0, 0.05, 0.2, 0.5, 0.8\}$
δ	Reduction in primary disease transmission due to PM	Equation(2.6)	$\delta \in [0, \delta_M]$
I_1	Detection threshold: area of the forest that is infected (hectares) $I_1 \in [0, 1]$	$I_1 = 0.25$	$I_1 \in \{0.1, 0.25, 0.5, 0.8\}$
$\tau(\delta)$	Time the infection is detected and RM is deployed (years)	Equation(**??**)	-
t_{05}	Time the pest start causing damage	Equation(2.3)	-
T_2	Doubling time(years)	Equation(2.4)	$T_2 \in [0, 60]$
ECONOMIC			
ρ_I	Annual per unit area loss due to disease (£ ha^{-1} $year^{-1}$)	-	$\rho_I \in [0, 10000]$
C_p	Annual per unit area cost of PM without RM (£ ha^{-1} $year^{-1}$)	$0 \leq C_p \leq \Pi$	-
Π	Annual per unit area PM budget (£ ha^{-1} $year^{-1}$)	$\Pi = 100$	-
r	Discount rate	$r = 0.03$	-
C_p^*	Optimal cost of PM without RM (£ ha^{-1} $year^{-1}$)	Equation(2.7)	$0 \leq C_p^* \leq \Pi$

2.5 Results

In this section, we present the results of analysing the precautionary management strategy using a deterministic model. Our aim is to identify how precautionary management (PM) performance and cost influence invasion timing, detection, and overall impact, thereby guiding efficient resource allocation. We simulated a range of parameter combinations to capture realistic variability in invasion dynamics and cost managements, and to assess how these differences influence the timing, cost effectiveness, and feasibility of PM strategies.

We first show the relationship between the effect of PM (δ), or cost of PM deployment (C_p), and the time the pest or pathogen starts to affect trees (t_{05}). Understanding these relationships is important because earlier damage onset or delayed detection can significantly affect management feasibility and economic outcomes. Additionally, we highlight the effect of PM investment on the invasion dynamics. In Section 2.5.1, we investigate the optimal management investment strategy, performing a sensitivity analysis to the annual loss rate due to disease (ρ_I), doubling time (T_2), primary infection (P) and maximum reduction in primary transmission due to PM ($(1-\delta_M)$.

In order to understand how parameters affect the model results, we show how the time the pest starts to damage trees, t_{05}, and detection time, $\tau(\delta)$, change with the reduction in the primary disease transmission due to PM, δ, and investment in PM deployment, C_p. This analysis allows us to determine whether increased spending on PM yields proportionally better outcomes, which is crucial for justifying higher investment levels.

Demonstration in Figure 2.3 plots show the impact of the annual loss, ρ_I, on the objective function, $J_p(C_p)$, given by Equation 2.5 and the optimal level of PM, C_p, when the RM strategy is not available. These plots illustrate that changes in the annual loss, ρ_I, will result in a different optimal solution, which depends on the budget (Π). The relationship between the plots in Figure 2.3 can be explained further in Figure (2.6). We do nothing when there is no reduction in the disease transmission rate and invest when we have losses. This was highlighted by dots on the red and purple curves in Figure (2.3). We notice a jump from

investing nothing when there is no reduction in the disease transmission rate to investing everything when the value of the annual loss per hectare of infected land, ρ_I, is increased. The dots on the red and purple curve in Figure (2.3) show the optimal investment level at each value of ρ_I. Figure 2.3(a) shows the optimal level of PM when $\rho_I = 500$ and $\rho_I = 700$, compared with the cost of control. The purple and red dot on Figure 2.3(a) curves indicates the minimum loss when the $\rho_I = 500$ and $\rho_I = 700$, respectively, at $C_p = 0$. Hence, this implies that we do not invest when the optimal solution is zero.

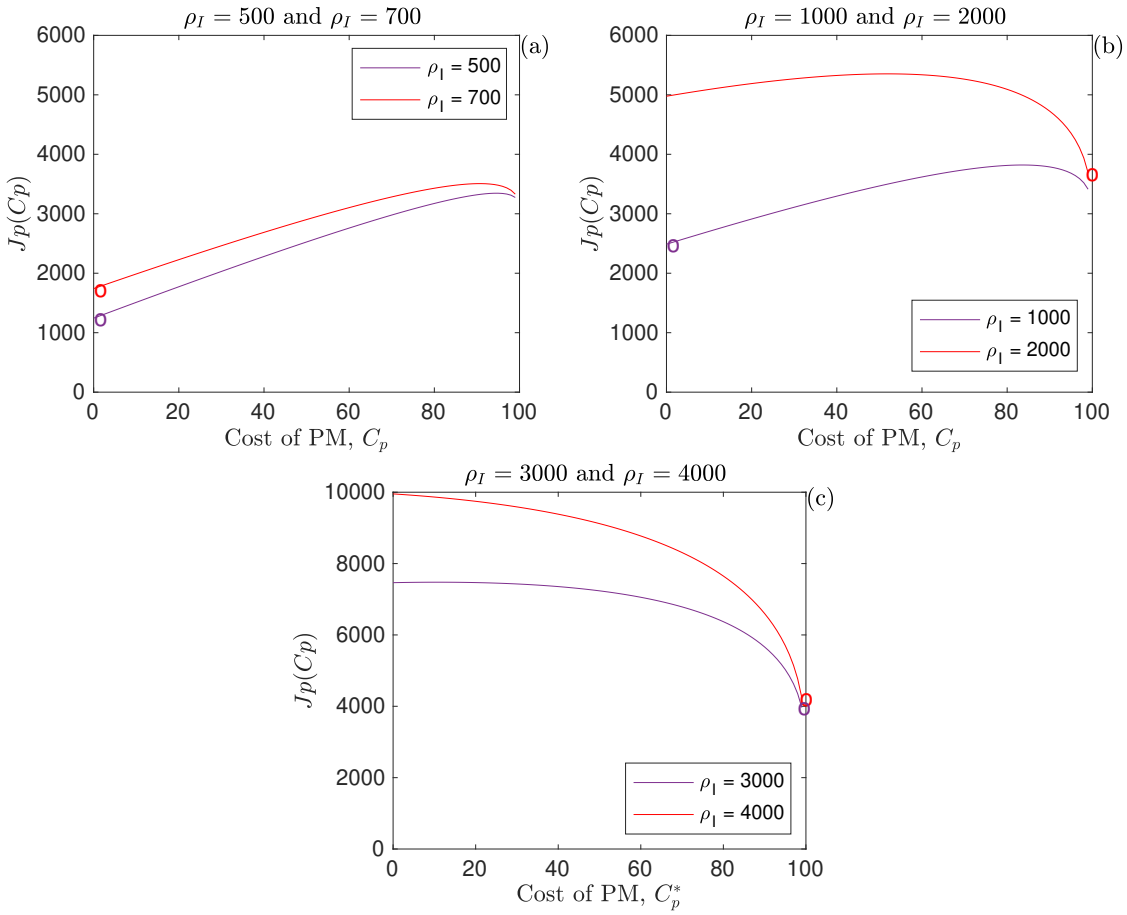

Figure 2.3: Plot of the total loss, $J_p(C_p)$, as a function of C_p for different values of ρ_I given by Equation 2.5 and the level of PM, C_p. Plot (a) shows the optimal solution when $\rho_I = 500$ and $\rho_I = 700$, plot (b) shows the optimal solution when $\rho_I = 1000$ and $\rho_I = 2000$, and plot (c) shows the optimal solution when $\rho_I = 3000$ and $\rho_I = 4000$. The purple curve shows the loss when the value of ρ_I is smaller, while the red curve shows the loss with a higher value of ρ_I in each plot. The baseline primary infection rate, $P = 0.005$ while other parameter values are at their baseline in Table 5.1.

Figure 2.3(b) shows the optimal level of PM when the annual per unit area loss caused by the diseases, $\rho_I = 1000$, and $\rho_I = 2000$, compared with the cost of control. The dot on the purple curve in Figure 2.3(b) indicates minimum loss when the $\rho_I = 1000$ at $C_p = 0$, which means that the optimal solution is to do nothing. The dot on the red curve indicates the minimum loss when the $\rho_I = 2000$ at $C_p = 100$, which means that the optimal solution is to invest the entire budget. Therefore, at some point in Figure 2.3(b), the total loss will

appear on the same level as the maximum budget (see Figure 2.5(b)). Figure 2.3(c) shows the optimal level of PM when $\rho_I = 3000$ and $\rho_I = 4000$. The dot on the purple curve indicates the minimum loss when the $\rho_I = 3000$ at $C_p = 100$, and the dot on the red curve indicates the minimum loss when the $\rho_I = 4000$ at $C_p = 100$. However, this means the optimal solution is to invest the entire budget at a very high loss. Hence, this indicates that a high ρ_I will result in a very high loss. The analysis will help minimise losses and understand the best time not to invest and when to invest.

To illustrate this switch of the optimal investment, we examine the effect of the annual loss, ρ_I, on the elements of the objective function, $J_p(C_p)$, given by Equation (2.5) and the cost of PM in Figure (2.4). The blue line represents the cost of PM throughout Periods A, B, and C (see Figure (2.1)). Simultaneously, the dot on the blue line indicates the point of optimal investment (cost of PM). The red and the purple curve in Figure (2.4) correspond to the smaller values of the annual loss, ρ_I, ranging to the larger value, respectively. However, these curves describe the losses when the pathogen starts to impact the forest. Plot (a) shows the losses and the cost of PM when $\rho = 500$ and $\rho = 700$. Plot (b) shows the losses and the cost of PM when $\rho = 1000$ and $\rho = 2000$. Plot (c) shows the losses and the cost of PM when $\rho = 2500$ and $\rho = 3000$.

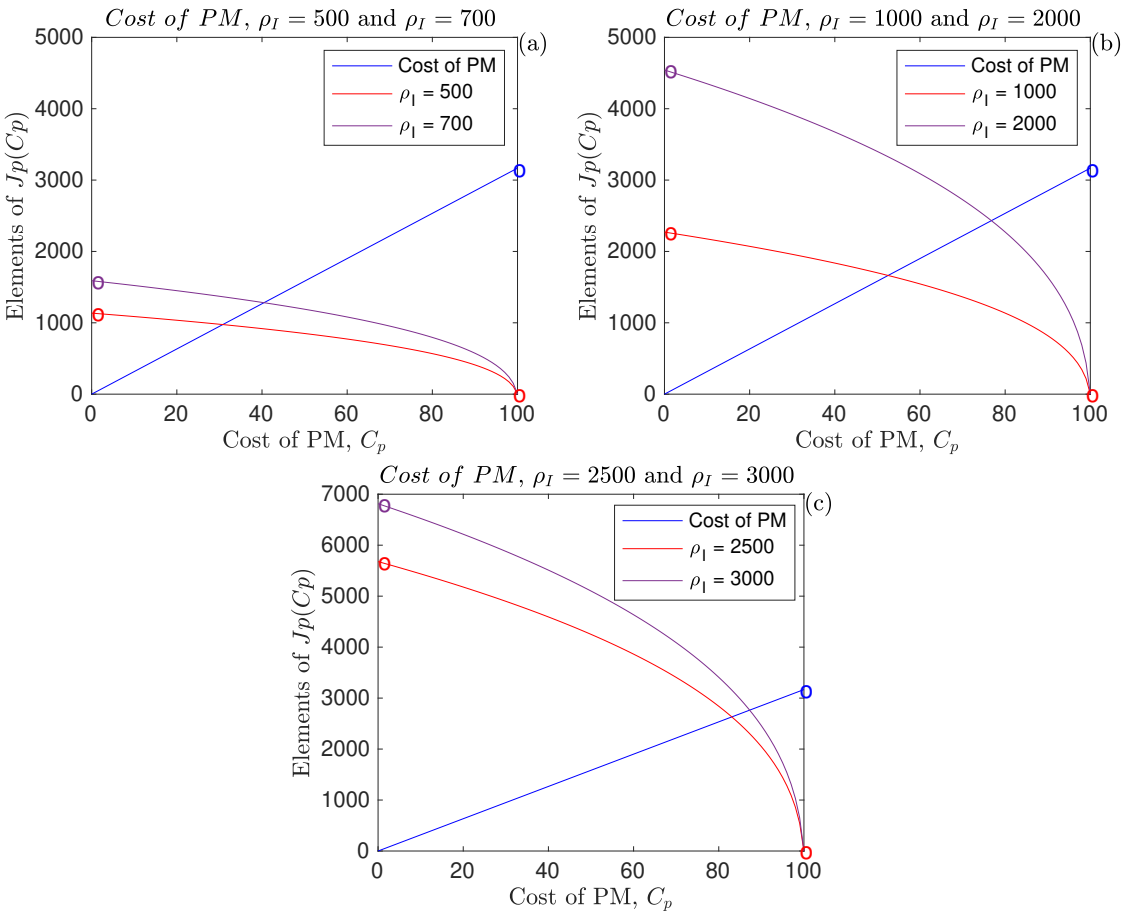

Figure 2.4: Effect of the loss caused by the diseases, ρ_I, on the elements of $J_p(C_p)$ given by Equation 2.5 and the cost of PM. Plot (a) shows the losses and the cost of PM when $\rho_I = 500$ and $\rho_I = 700$. Plot (b) shows the losses and the cost of PM when $\rho_I = 1000$ and $\rho_I = 2000$. Plot (c) shows the losses and the cost of PM when $\rho_I = 2500$ and $\rho_I = 3000$. The blue line indicates the cost of PM, while the red and purple curve indicates different values of ρ_I when the pathogen starts to impact the forest. All the parameter values are at their baseline in Table 5.1.

In Figure (2.4), we have a similar simulation for the objective function, which was carried out using the components of $J_p(C_p)$ with respect to the cost of PM. We examine the elements of the $J_p(C_p)$ to identify how increasing the annual loss, ρ_I, will affect the cost of PM. In Figure 2.4(a), we observe that for smaller values of the annual loss, ρ_I, the curve shows a lower impact on the cost of PM. As the annual loss goes up, at some point, we will reach the point where the loss will be almost the same as the maximum investment. For a very high value of ρ_I in Figure 2.4(b) and (c), we identify that the loss will be higher than the cost of PM. In

Figure 2.4(b), the red curve where $\rho_I = 1000$, the loss is lower than the cost while the purple curve donates a very high loss to compare with the cost of control. Therefore, at some point in Figure 2.4(b), the annual loss and the cost of control will appear on the same level (see Figure 2.5(a)). In Figure 2.4(c), both the red and purple curves exhibit a higher loss than the cost of PM.

These figures demonstrate that, for any value of ρ_I, the behaviour of the annual loss similar. This suggests that the annual loss will continue to increase over some time without the cost of prevention. By performing simulation over a wide range of ρ_I values, we could show that there will be some point where the value of annual loss will be approximately the same level as the cost of prevention. This point will be where the strategy changes from do nothing (the black region in Figure 2.6) to do something (the purple region in Figure 2.6).

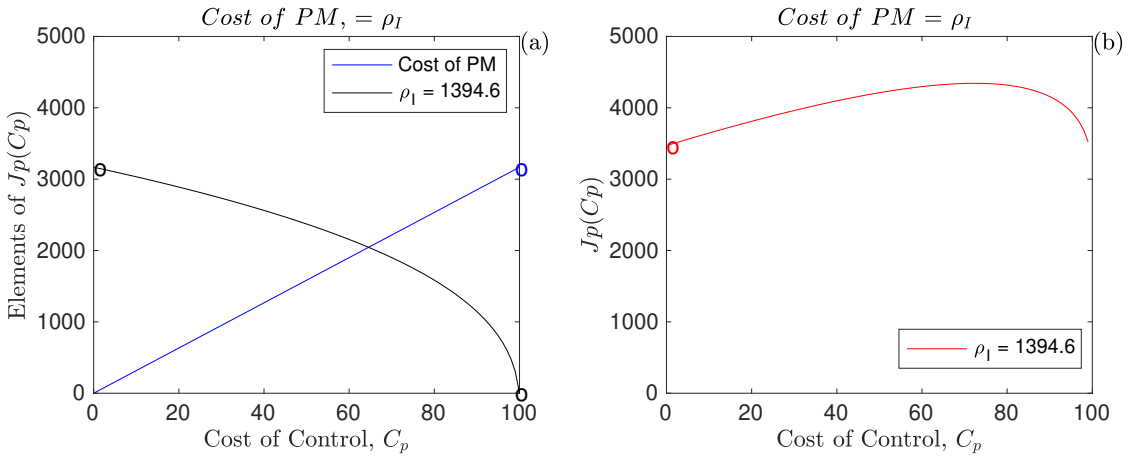

Figure 2.5: Plots with almost the exact value of the annual loss, ρ_I, and the cost of control, C_p, are created when carrying out sensitivity analysis using the key parameters. The blue line shows the cost of PM, the black curve denotes when the annual loss is approximately the same as the maximum investment, and the red curve shows when the total loss is approximately the same as the cost of control. All other parameter values are at their baseline in Table 5.1.

Figure (2.5) shows the sensitivity analysis when the loss and the cost of control, C_p, is approximately the same level. Figure 2.5(a) represents the plot of the element of total loss when the cost of control is approximately the same as the annual loss. However, the blue line indicates the cost of PM, which increases as the element of a total loss, $J_p(C_p)$, increases. In

comparison, Figure 2.5(b) denotes the plot of the total loss (damages and cost of prevention), $J_p(C_p)$, when the annual loss is approximately the same as the maximum investment. When examining the relationship, we observe that when the annual loss $\rho_I = 1394.6$, the cost of control and the annual loss appear on the same level as the total loss. The black curve equally shows that the loss will rapidly drop as the cost increases. The black curve illustrates the annual loss decline as the cost of PM increases, following the point when the loss is equal to the cost of PM. The red curve in Figure 2.5(b) shows the minimum loss when the maximum investment and the annual loss are almost the same level.

$$\rho_I = \frac{C_p L\left(1 - e^{-rt}\right)}{\int_{t_{05}}^{T} \left(I(t)_A e^{-rt}\right) dt} \tag{2.8}$$

2.5.1 Optimal precautionary management (PM) only strategy

When the RM strategy is not available, the optimal management strategy is the optimal level of PM found by Equation (2.7). In this section, we highlight the optimal PM strategy which minimises the present value of costs incurred in managing an invasive species impact on a forest over a specified interval. We find three different types of strategies, given in Table (2.2). We do nothing when loses due to diseases are lower than the cost of PM, Figures 2.3(a) and 2.4(a) : the optimal PM level is zero ($C_P^* = 0$). We name this PM strategy "None". In the figures in this section, parameter spaces where the "None" PM strategy is optimal are coloured black and labelled management strategy $1a$. However, when pathogens arrive and spread but at a rate below the threshold for severe damage, the optimal strategy under a bang–bang framework is to invest a portion of the budget aggressively in prevention to halt further spread before it escalates. Then, the optimal level of PM will be between zero and the full budget ($0 < C_P^* < \Pi$). We name this PM strategy "Partial". In the figures in this section, parameter spaces where the "Partial" PM strategy is optimal are coloured pink and labelled management strategy $2a$. Finally, when the pathogen is spreading very fast or causing significant damage, the optimal level of PM is equal to the budget ($C_P^* = \Pi$), and it is optimal to spend all of the budget. We name this PM strategy "Full". In this case, losses due to disease are much higher

than the cost of PM, Figures 2.3(c) and 2.4(c). In the figures in this section, parameter spaces where the "Full" PM strategy is optimal are coloured purple and labelled management strategy $3a$.

Optimal management strategy	PM (Optimised)
$1a$ (black)	None, $C_P^* = 0$.
$2a$ (pink)	Partial, $0 < C_P^* < \Pi$.
$3a$ (purple)	Full, $C_P^* = \Pi$.

Table 2.2: The management strategies when the RM is not available.

The optimal level of PM, C_p^*, is given by Equation (2.7). We solve this numerically since the minimisation problem has is no analytic solutions. We use the parameter values presented in Table (2.1) for the sensitivity analysis. Firstly, we perform a sensitivity analysis of the optimal PM strategy to the doubling time (T_2), which determines the secondary infection rate (β), and the annual loss rate due to the disease (ρ_I). We then investigate how the optimal management strategy changes when the baseline primary infection rate (P), and the maximum reduction in primary transmission rate (δ_M) are altered.

Sensitivity analysis to the annual loss rate due to disease, ρ_I, and doubling time, T_2.

In this section, we explore the sensitivity of the optimal PM only strategy to the annual loss, ρ_I, and doubling time, T_2. Figure (2.6) shows the optimal PM only strategy for different combinations of the doubling time (T_2) and annual loss rate due to disease (ρ_I). We start by examining the relationship between the zones, as labelled in Table (2.2) and given by Equation (2.7).

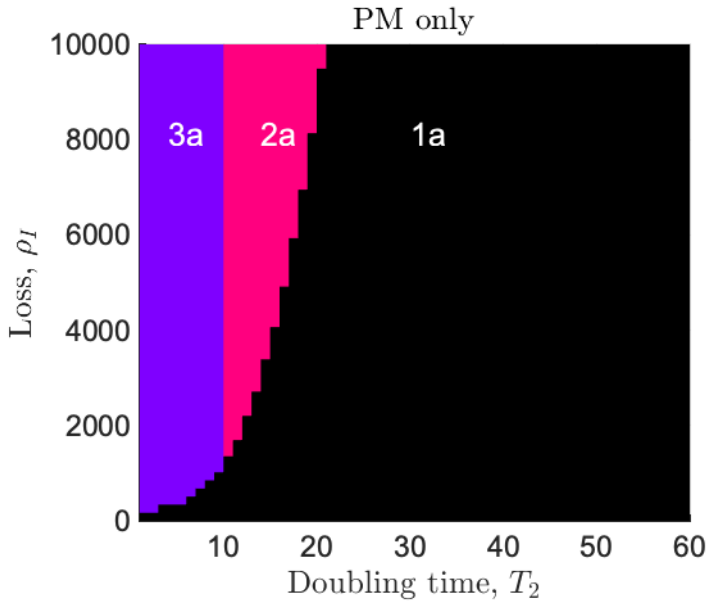

Figure 2.6: Optimal PM only strategy in a doubling time - loss rate due to disease ($T_2 - \rho_1$) parameter space. The doubling time is T_2 and the annual loss rate due to disease is ρ_I. The optimal management strategy is given by Equation 2.7 and described in Table 2.2. This means that the zones in Figure (2.6) show the optimal management strategy against the doubling time, T_2 and the annual loss rate due to disease, ρ_I. The black region (zone 1a) shows where it is optimal not to deploy PM, the pink region (zone 2a) is optimal to deploy partial PM, and the purple region (zone 3a) is optimal to deploy full PM. The baseline primary infection is $P = 0.005$ and $\delta_M = 0$ so that PM can completely stop the pathogen, while other parameter values are at their baseline in Table 2.1.

In Figure (2.6), we observe that zone $1a$ (None) extends from small loss, ρ_I, to a very high loss as the doubling time, T_2, increases. The zone $1a$ region extends to the fast spreading pathogen (small T_2)if the loss is minimal. This means that if the disease is spreading but not causing enough damage (small ρ_I), we do nothing and the PM strategy is not deployed. This is because the total loss caused by the disease is smaller than the cost of deploying the PM, regardless of the time that the loss starts to accrue or how fast the disease transmits within the forest, see Figure 2.3 (a).

The switch from no PM (zone $1a$) to partial PM (zone $2a$) occurs for a middle doubling time as ρ_I increases (Figure, (2.6)). At the medium values of T_2, the start of the damage (t_{05}) can be push so far that it reduces the losses without having to set $Cp = \Pi$, i.e. investing all.

The combination of parameters labelled zone $2a$ represents scenarios with a faster spread and greater impacts from invasive species. Under these parameters, a no action policy produces substantially larger discounted losses; equivalently, the present value of costs rises markedly if we do nothing. Thus, in zone $2a$ the model predicts stronger incentives for active intervention, and under a bang–bang optimal-control formulation the policy will tend to switch to full control once the system crosses the threshold at which the avoided discounted costs justify expending the entire available budget. Therefore, it is optimal to deploy partial PM to delay when the losses start to accrue. However, there is yet no need to deploy the full PM (as in Zone $3a$), as a partial reduction in the primary infection is enough to delay the pathogen arrival and decrease the losses.

Interestingly, the boundary between where partial PM is optimal (zone $2a$) and full PM is optimal (zone $3a$) does not depend on the annual loss rate from the disease (ρ_I), Figure (2.6). In contrast, the boundary between where it is optimal not to deploy PM (zone $1a$) and optimal to deploy partial PM (zone $3a$) depends on the loss rate parameter, ρ_I. An increase in the loss rate (ρ_I) increases the potential total loss caused by the disease. The threshold value of the loss rate due to the disease (ρ_I) that results in it being optimal to deploy partial PM over none is small when the doubling time (T_2) is short. This threshold value in the loss rate (ρ_I) increases as the doubling time increases.

The switch from partial PM (zone $2a$) to full PM (zone $3a$) occurs at a small doubling time (fast spreading pathogen) as the loss, ρ_I increases, (Figure (2.6)). The whole forest would be infected by the end of the time horizon if the full PM is not deployed. This is particularly true when the doubling time, T_2, is low since any benefit of deploying the PM is diminished by the fast rate of disease spread. Shortening the doubling time, T_2, brings forward the time the loss caused by the disease starts to accrue, but deploying the full PM will delay it. Spending the whole PM budget is optimal to prevent the further losses in this case. A fast-spreading invasive species requires immediate intervention due to the increase in loss in a short doubling time, zone $3a$ in Figure (2.6).

Sensitivity analysis when annual loss due to diseases, ρ_I, is equal to cost of control

We carried out a sensitivity analysis to determine what happens when the maximum investment is approximately the same as the damage from the diseases. We start by examining the relationship between the annual loss given by Equation 2.9 and the doubling time, T_2.

The optimal management strategy minimizes the total cost of control (prevention) and the cost associated with the damage over the time horizon T. Based on the objective function giving the total loss in Equation 2.5, the cost of PM is $\int_0^T (C_p L e^{-rt}) dt$ and the losses caused by the diseases before and after detection are given as $\int_{t_{05}}^T (\rho_I I(t)_A e^{-rt}) dt$.

From the numerical simulation, when the maximum feasible investment is approximately equal to the losses from the disease, the results indicate that;

$$\int_{t_{05}}^{T} \left(\rho_I I(t)_A e^{-rt}\right) dt = \int_0^T C_p L e^{-rt} dt$$

Simplifying further, the numerical simulation was carried out to find the annual loss due to diseases, ρ_I, since there is no analytical solution.

$$\rho_I \left(\int_{t_{05}}^{T} \left(I(t)_A e^{-rt}\right) dt \right) = C_p L \left(1 - e^{-rt}\right)$$

$$\rho_I = \frac{C_p L \left(1 - e^{-rt}\right)}{\int_{t_{05}}^{T} \left(I(t)_A e^{-rt}\right) dt} \tag{2.9}$$

However, Equation (2.9) can not be evaluated analytically, so we used numerical methods in Matlab. Figure (2.7) shows the combination of Figure (2.6) and a white curve. This boundary is crucial because it helps us determine when it is optimal to invest in precautionary measures versus when it is more cost-effective to do nothing. The white curve between zone $2a$ and zone $1a$ in Figure (2.7) shows what happens when the annual loss, ρ_I, is at the same level as the cost of control. This depends on doubling time because the lower the doubling time, the higher the losses. The boundary between zones $1a$ and $2a$ is determined by the balance between the investment in disease control and the loss from the disease. In this section, we

provide a way to find this boundary.

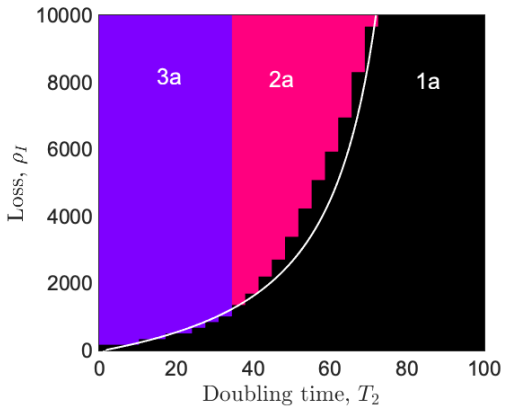

Figure 2.7: Plots of when the loss from the diseases, ρ_I, as given by Equation 2.9, is equal to the cost of control, C_p. We produce these plots using all parameters at their baseline value.

The three regions in Figure (2.7) represent the optimal level of PM when the RM strategy is not available. Zone $1a$ (black region) is optimal not to deploy the PM strategy, so we do nothing. It is optimal not to deploy any precautionary measures because the losses are relatively low, particularly at high doubling times where the pathogen is less infectious and spreads slowly. The Zone $2a$ (pink region) is optimal for deploying partial PM, so the disease presents a moderate threat, so a partial investment in prevention can help mitigate further losses without investing too much resources. The Zone $3a$ (purple region) is optimal for deploying full PM, so we invest to prevent further damage. In Figure (2.7), the white curve corresponds to the boundary between where it is optimal not to deploy PM (zone $1a$) and the other region where we deploy partial and full PM strategies (zone $3a$ and $2a$). At a very high doubling time we do nothing because the pathogen is not infectious. Conversely, for low values of ρ_I we also do nothing, even if the pathogen is highly infectious (low doubling time T_2 and high β). The left-hand side of the white curve represents the invest region, while the right-hand side indicates the region where no PM is used.

Sensitivity analysis to the infection rate, P.

In this section, we analyse how the optimal PM strategy changes when the pathogen is more likely to arrive by considering different values of the primary infection, P. Figure (2.8) shows the optimal PM only strategy for different combinations of the doubling time (T_2) and annual loss rate due to disease (ρ_I), as well as the primary infection (P). Figure 2.8(a) show what happens when the P is reduced to 0.0005, while 2.8(b) assumes P is at the baseline value $P = 0.005$, and is the same as Figure 2.6 above. Then, Figure 2.8(c) shows the effect of the pathogen when P is increased to 0.1.

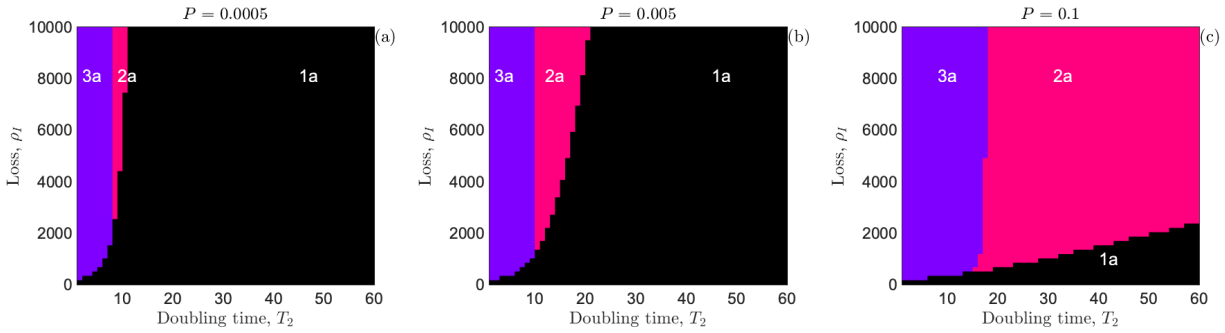

Figure 2.8: Optimal PM only strategy in a T_2-ρ_I parameter space, for different values of the baseline primary infection, P. The doubling time is T_2 and the annual loss rate due to disease is ρ_I. The optimal management strategy is given by Equation 2.7 and described in Table 2.2. The black region shows where it is optimal not to deploy PM, the pink region is optimal to deploy partial PM but the purple region is optimal to deploy full PM. The baseline primary infection, P, is (a) $P = 0.0005$, (b) $P = 0.005$ and (c) $P = 0.1$, while other parameter values are at their baseline in Table 2.1.

We observe that zone $1a$ (None) is reduced as the primary infection, P, increases while zone $2a$ and zone $3a$ grow, Figure 2.8. In particular, the switches from no PM (zone $1a$) to partial PM (zone $2a$), and from partial PM (zone $2a$) to full PM (zone $3a$), occurring as the doubling time (T_2) decreases, happen at smaller values of the loss, ρ_I. Zone $1a$ (do nothing) always starts from a low doubling time and extends to a very high doubling time (T_2) and high loss rate due to disease (ρ_I). However for the majority of doubling time (T_2) and loss rate (ρ_I) combinations in Figure 2.8(a), the total loss caused by the disease is smaller than the cost of deploying the PM regardless of how fast the disease is transmitted within the population.

On the other hand, zone $1a$ is small in Figure 2.8(c) when the primary infection rate is high ($P = 0.1$), so the threshold for the total losses caused by the disease to exceed the cost of PM deployment is reached for a small value of the annual loss rate parameter (ρ_I).

The region where it is optimal to deploy partial PM (zone $2a$) grows as the primary infection, P, increases. The invasive species arrives and starts to spread earlier when the primary infection (P) is higher. Figure 2.8(a) ($P = 0.0005$) shows a small range of partial PM region while Figure 2.8(c) ($P = 0.1$) shows partial PM is optimal even for longer doubling times (T_2) and for lower annual loss rates (ρ_I). Therefore, for a higher P we will deploy partial PM in more case, in order to delay the time the losses start to accrue. Similarly, when the primary infection increases and invasion occurs earlier, full PM is optimal over partial PM for longer doubling times T_2, Figure 2.8(a)(c). An increase in the annual loss rate due to disease (ρ_I) or doubling time (T_2) typically increases the optimal level of PM, and when the primary infection (P) is higher, these effects are magnified.

Sensitivity analysis to the maximum reduction in primary disease transmission rate due to PM, $1-\delta_M$**.**

In this section, we analyse how the optimal PM strategy changes when the effectiveness of PM in delaying the invasion is varied. Figure 2.9 shows the optimal PM only strategy for different combinations of the doubling time (T_2) and annual loss rate due to disease (ρ_I), for different values of the maximum reduction in primary transmission rate due to PM ($1-\delta_M$). The parameter δ_M can be interpreted as a measure of maximum effectiveness of the PM in delaying the time at which the loss caused by disease starts to accrue, Equation 2.6 and Equations 2.1. When the parameter $\delta_M = 0$, it means that the maximum reduction in primary disease transmission rate due to PM is 1, and the PM can completely stop the arrival of the pathogen. As δ_M, increases from 0, the value of δ for $C_p = \Pi$ (full investment) is no longer 0. This means that the PM cannot stop the disease from arriving.

As shown in the previous subsections, it is optimal to deploy some type of PM when the cost of deployment is outweighed by the losses from disease, i.e., if the doubling time

(T_2) is sufficiently short, or the losses due to disease (ρ_I) are high enough. Decreasing the maximum reduction in primary disease transmission rate due to PM, $1-\delta_M$, by increasing the parameter δ_M , makes it less likely that deploying PM is optimal and the $T_2-\rho_1$ (doubling time -loss rate due to disease) parameter space where deploying PM is optimal shrinks, Figure 2.9. When we compare Figure 2.9 (a) to (b) to (c), we see that as PM effectiveness decreases (δ_M increases), the region (zone $1a$) where it is optimal not to deploy PM increases. Specifically, as the effectiveness of PM decreases ($1-\delta_M$ decreases), the threshold value of loss rate due to disease (ρ_I), where above that it is optimal to deploy PM (switch from $1a$ to $2a/3a$), is higher. Similarly, the threshold value on the doubling time, where below that value it is optimal to deploy PM, is lower. Additionally, the region where it is optimal to deploy partial PM (zone $2a$) disappears, and we are more likely to deploy full PM or no PM (all or nothing). The region where it is optimal to deploy full PM (zone $3a$) shrinks as $\delta_M \to 1$. The benefits of deploying PM strategy decrease as PM effectiveness decreases (δ_M increases).

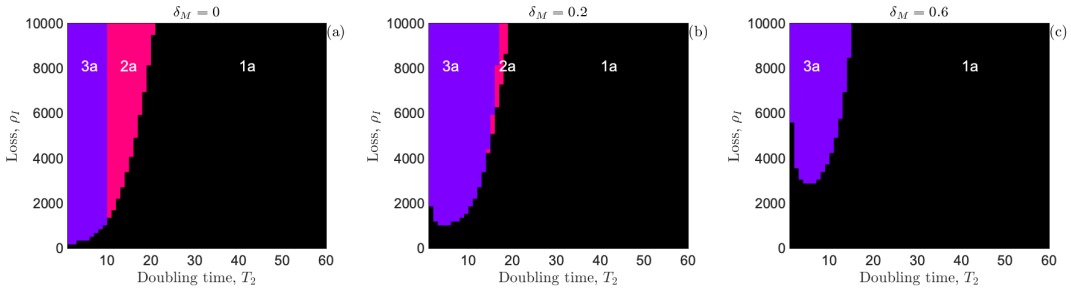

Figure 2.9: Optimal PM only strategy in a T_2-ρ_I parameter space, for different values of the maximum reduction in primary transmission rate due to PM, $1-\delta_M$. The black region shows where it is optimal not to deploy PM. The pink region is where it is optimal to deploy partial PM, but the purple region is where it is optimal to deploy full PM. The maximum reduction in primary transmission rate due to PM ($1-\delta_M$) is varied across the panels: (a) $\delta_M = 0$, (b) $\delta_M = 0.2$, and (c) $\delta_M = 0.6$.

As before, in Figure 2.9(a), we have 3 regions: zone $1a$, zone $2a$ and zone $3a$. Zone $1a$ is no investment, zone $2a$, a partial investment, and zone $3a$, a full investment. Full investment in PM (zone $3a$) is typically optimal when the disease causes significant damage, through a combination of spreading quickly (shorter doubling time, T_2) or a high loss rate due to disease

(larger ρ_I. On the other hand, if the spread is slow (high doubling time, T_2) and causes little damage (lower ρ_I then no investment is more likely to be optimal (zone $1a$). In this case, the disease will not spread within the next 100 years. Hence, there is a trade-off between the doubling time, T_2, infectiousness, and the loss rate due to disease, ρ_I, therefore, the disease will become more infectious as the doubling time decreases. When the losses due to the pest or pathogen will outweigh the cost of PM deployment, we invest to prevent this damage in zone $2a$ and $3a$. The curve between the black region (zone $1a$) and the rest of the zones (zone $2a$ and $3a$) can be identified as a point of interruption. They indicate threshold combinations of the doubling time (T_2) and loss rate due to disease (ρ_I), where increasing the loss rate or shortening the doubling time further would result in it being optimal to deploy some type of PM investment.

2.6 Discussion

In this chapter, we first developed a generalisable bioeconomic model to understand how best to deploy a strategy to reduce the effect of an emerging pathogen. The model assumes the control strategies protect the forest from an invasive pathogen, which would reduce the benefits provided by the forest. Here, we investigate the relationship between epidemiological dynamics of plant diseases and economic decisions that guides forest management practices. This holistic approach provides opportunities to quantify not only the consequences of disease evolution, but also any costs associated with different management decisions to reduce disease impact, building upon *Macpherson et al.* [86], we explore a wide range of parameters essential to various aspects of forestry management, ranging from forest area considerations to the impact and efficacy of pest control measures.

We explore the dynamics of disease spread in a forest area using a Susceptible-Infected compartmental model. The key to our model are parameters such as the forest area (L), the infected area ($I(t)$), and the primary and secondary infection rates (P) and (β). Our model incorporates both precautionary and reactionary management strategies, represented by δ

and ϵ respectively, to simulate the effects of different management approaches on disease dynamics. The solutions to our model equations provide insights into the progression of the disease, from initial infection to the point of detection and subsequent management intervention. Particularly, the model outputs, including the detection time and the time of initial significant damage (t_{05}), are crucial in understanding the timing of intervention strategies. The doubling time (T_2) further offers a perspective on the aggressiveness of the pathogen's spread and is easier to interpet the rate of spread β. These insights are instrumental in shaping effective forest management strategies under the threat of disease invasion.

Having introduced the full model in Section 2.2, we then consider the application of the Precautionary Management, PM, only. Our results illustrate the intricate dynamics between the cost of precautionary management C_p and its effectiveness in curtailing the spread of disease within the forest. The optimal PM level, C_p^*, emerges as a critical decision point that balances economic expenditure against epidemiological control. The model predicts that the cost-effectiveness of PM is directly tied to the financial resources allocated, constrained by the budget. The maximum effectiveness of PM δ_M signifies the potential to delay the onset of significant disease impact, potentially altering the economic landscape of forest management. Lastly, the sensitivity of the time to reach a 5% infection level (t_{05}) and the detection time $\tau(\delta)$ to variations in δ and C_p is particularly revealing. It suggests that the early stages of disease spread are crucial intervention points that can significantly alter the course of the disease trajectory, which is vital for forest managers to consider in their strategic planning.

The gradual increase in the critical value of annual loss ρ_I, as doubling time increases, as illustrated by the white curve, emphasise the importance of timely intervention. If the disease is allowed to spread unchecked, the losses can escalate rapidly, resulting to more aggressive and costly interventions. On the contrary, in scenarios with slower disease progression (higher T_2), the analysis suggests that it may be more advisable to economise the resources and avoid investing in precautionary measures. The white curve in Figure (2.7) is a pivotal indicator that helps balance the costs and benefits of disease management strategies. By identifying the optimal intervals for intervention, this analysis ensures that resources are used efficiently,

either by investing in precautionary measures where necessary or by conserving them when the threat level is low. This approach not only minimises potential losses but also enhances the overall effectiveness of disease control efforts.

A key benefit of our approach is that we can undertake extensive sensitivity analysis to illustrate how different epidemiological and economic components change the optimal management strategy. In general, the results show that when the pest or pathogen will cause an increased loss through earlier arrival, quicker spread, or greater impact, or the effectiveness of PM deployment is increased, there is an increased incentive to deploy a PM strategy. This is especially true when the disease transmits quickly, or the loss caused by pathogens starts accruing early in the time horizon. In these scenarios, we require an increase in the level of PM to counter the present value of the total cost of the invasion. We found that under some conditions, if the annual loss caused by the disease is low and if the rate of disease transmission is low, it may be optimal to do nothing. This result is intuitive since, under these conditions, the cost of deploying any management strategy outweighs the benefits of reduced disease spread.

Our model makes a number of simplifying assumptions to keep the analysis tractable: we treat the host population as uniform and evenly distributed, with constant transmission rates and management effects over time, immediate and sustained benefits from precautionary management, and no random variation in spread or detection. Although this approach allows clear comparisons between scenarios, it omits important complexities such as spatial spread, stochastic events, seasonal variation, changes in host population, and potential delays or declines in management effectiveness. Incorporating these features in future work, for example stochastic model would make the epidemiological component more realistic and help ensure that management recommendations are robust under real world conditions.

Chapter 3

Modelling Uncertainty in Pathogen Arrival

In this chapter, we introduce a stochastic model in section 3.1 which considers variability in the model pathway, where the time of pathogen arrival is uncertain. Section 3.3 presents the results of our uncertainty analysis which consist of a log-normal distribution in section 3.3.1 and the absolute values from a normal distribution in section 3.3.2. We also explore the sensitivity and uncertainty associated with the model, where the sensitivity analysis examines the contribution of each predicted parameter in both the deterministic analysis and the stochastic process. Specifically, we want to investigate whether introducing randomness to the disease transmission model will reduce the rate of pathogen arrival, and hence delay the costs and damage caused by the disease, and hence change the conditions for applying the control measures. In this chapter, we concentrate on the PM strategy and the chapter discussion is in section 3.4.

3.1 Introduction

This chapter focuses on developing a stochastic model derived from our initial deterministic approach, with the aim of capturing the inherent uncertainties in pathogen transmission. A stochastic model integrates random variables in some way. In a stochastic model, the relevant parameters are either modelled as stochastic processes or the stochastic processes

are added to the driving systems. Deterministic models are generally easier to analyse than stochastic models. However, in many cases, stochastic models are more realistic, particularly for problems that involve small numbers. Deterministic models of disease transmission were used in chapter 2 of this thesis to gain insight into the transmission dynamics of infectious diseases. This emphasised the need to analyse the basic assumptions behind infection spread and control models and recognise how these assumptions influence key epidemiological parameter estimates and subsequent epidemic predictions. In order to generate epidemic forecasts that are useful for plant health decision-making, there is a need to modify the deterministic model to include random fluctuation. This will capture the disease transmission rate dynamics by incorporating random arrivals.

In this chapter, we develop the stochastic model, which we shall study in the rest of the chapter. We reviewed and incorporated different mathematical modelling approaches, including log-normal distribution and the absolute values from a normal distribution. A stochastic model takes into account variability in the model pathway, where the time to the next event, or which event will occur next, is uncertain. Utilising the log-normal distribution for this purpose is appropriate when variables are modelled since they can not be negative, and the variables are positively skewed. Our approach to incorporating stochasticity involves two distinct methods. We utilise the absolute values from a normal distribution to introduce random variability into the primary infection, P, hence the arrival times are random. This method allows us to simulate scenarios where P fluctuates around a mean value, reflecting real-world conditions where transmission rates are not constant. This choice is motivated by the log-normal distribution's ability to represent a wide range of possible outcomes, especially in situations where the variable of interest can vary significantly.

The primary objective of this section is to examine how this stochastic element influences the spread of pathogens and the corresponding efficacy of precautionary management (PM) strategies. This analysis is critical in understanding the thresholds and triggers for different PM approaches under the stochastic nature of pathogen spread. The introduction of stochastic elements into our model not only enhances its realism, but also offers a more advanced tool

for forest management planning.

3.2 Model development

The deterministic model was defined in Chapter (2) as Susceptible-Infected compartmental model (see Equation (2.1)). The area of the forest that is infected at time $t$is $I(t)$(hectares), where $I(t) \in [0, L]$. In contrast to Chapter (2), we assume no infection $I(0) = 0$, at the beginning of the time horizon ($t_0 = 0$). The baseline primary infection, P (hectares^{-1}), controls when the pathogen emerges in the forest and the rate of transmission of primary infection by a factor of δ where $\delta \in [0, 1]$. The baseline secondary disease transmission rate β (year^{-1} hectare^{-1}) controls the rate of spread of the pathogen within the forest. We assume the pathogen is detected when the infected area reaches a detection threshold level, I_1. This occurs at time $t = \tau(\delta)$, which depends on the effort δ. Equation (3.1) are given here for convenience,

$$\left(\frac{dI}{dt}\right)_A = \beta(L - I(t))(I(t) + \delta P), \quad 0 < t \leq \tau(\delta). \tag{3.1}$$

The stochastic transmission model has the same objectives as the deterministic model described in chapter (2), to understand how the optimal level of precautionary management (PM) changes when the economic or epidemiological parameters change. Taking into account the initial condition, the equations 3.1 to become;

$$\left(\frac{dI}{dt}\right)_A = \begin{cases} \beta(L - I(t)(I(t) + \delta P), \\ P = \text{random} \end{cases} \tag{3.2}$$

The rate of new infections $\frac{dI}{dt}$ is proportional to the infection pressure $I(t) + \delta P$ and the susceptible area. Equation (3.2) represents the rate of change of infected area *before* detection, where $L - I(t)$ is the forest area that is not infected (susceptible but under surveillance) and the label A denotes the spread of the pathogen before detection. If RM is not available, as considered here, we set $\epsilon = 1$, so the contribution of $I(t)$ to the losses will be different. Here, $N(\mu, \sigma)$ represents a random variable sampled from a normal distribution with mean μ and

standard deviation σ. Taking the absolute value ensures that the parameter remains positive. Assume that $N(\mu,\sigma)$ follows a normal distribution with parameters μ and σ.

3.3 Precautionary management results

This section shows the numerical calculation of the random arrival when the reactive management (RM) strategy is unavailable. After the initial parametrization of a given model in chapter 2, this chapter analysed the sensitivity and uncertainty associated with the model. In general, sensitivity analysis examines the contribution of each predicted parameter in both the deterministic analysis and stochastic process. We examine the difference between the deterministic results in chapter (2) and the stochastic process results computed in this chapter. The colours in Figure (3.3) show the switch from employing full PM (zone 3a, purple) when the doubling time is short to incomplete control (zone 2a, pink) to when losses are high at the medium doubling time and do nothing (zone 1a, black), when the losses are less at a high doubling time. The nature of the colour patterns within Figure (3.3) and the previous chapter (Chapter 2) will lead to a better understanding of the colours and zones in the table (3.1).

Optimal management strategy	PM (Optimised)
1a (black)	None, $C_P^* = 0$.
2a (pink)	Partial, $0 < C_P^* < \Pi$.
3a (purple)	Full, $C_P^* = \Pi$.

Table 3.1: The management strategies when the RM is not available.

The optimal level of PM was solved numerically by introducing random arrival to Equation 2.5 (as given in Chapter (2) above). The numerical solution used in this Chapter was explained in detail in section (3.3) and (3.3.1) while Table (2.1) presented the parameters used for this analysis with the baseline values. We carried out the sensitivity analysis with the

key parameters and different values of variance ($\sigma^2 = 0, 0.00001, 0.0001, 0.001, 0.01, 0.1$). The strategy was shown through the annual loss ρ_1 and against the doubling time, T_2.

3.3.1 Log-normal distribution

The distribution of P values

In this analysis, Figure (3.1) shows the distribution of a log-normal function, which models the probability of arrival spread of pathogens based on different variances. The plots highlight the effect of changing variance on a log-normal distribution, providing information on how variability affects the spread of pathogens. The infected area, I(t), is given by Equation (3.2) with the primary infection, P, computed with log-normal distribution with mean(μ)= 0.005 and the variance (σ^2), the values are (a) variance = 0.00001, (b) variance = 0.0001, (c) variance = 0.001, (d) variance = 0.01 and (e) variance = 0.1. The histogram in Figure (3.1) exhibits a very asymmetric frequency distribution. Data are clustered at lower values of P and progressively decrease as P increases. The tall bar on the left side of the histogram represents a high frequency of data points with low values of P but the right tail stretches to approximately 0.008 at a high variance (see Figure 3.1(a)). There are very few data points with extremely high P values.

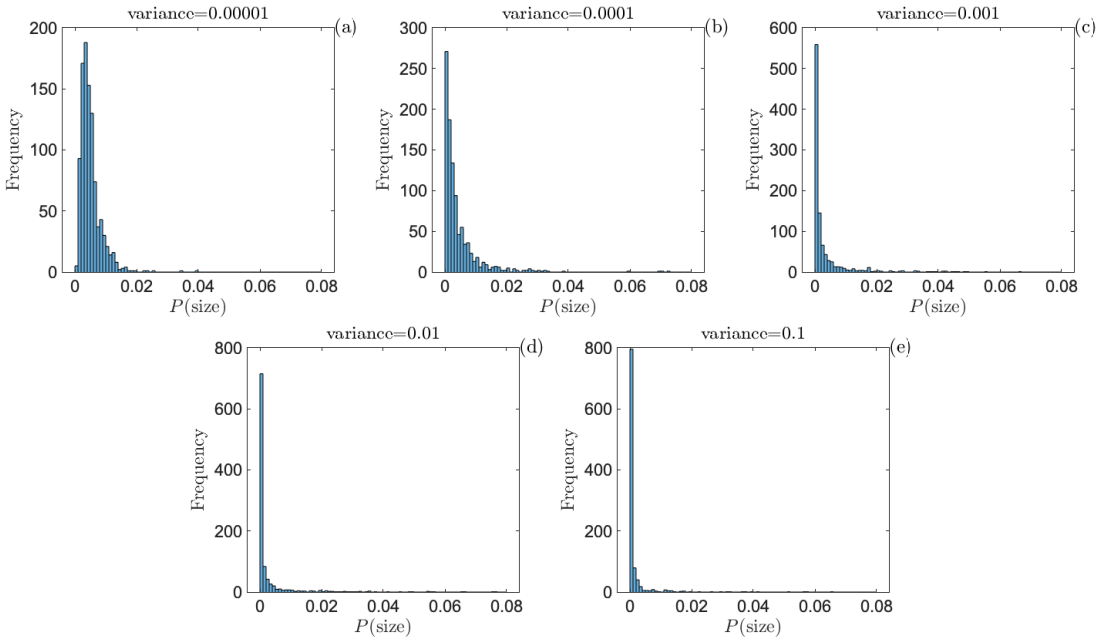

Figure 3.1: Histograms of the log-normal distribution function. The probability of arrival and spread of pathogens was calculated at different variance level: (a) Variance = 0.00001*, (b) Variance* = 0.0001*, (c) Variance* = 0.001 *(d) Variance* = 0.01*, and (e) Variance* = 0.1*. The Figure is showing one thousand (*1000*) values of randomly choosing P.*

The histogram in Figure 3.1(a) illustrates the frequency distribution with a very low variance of 0.00001 using the log-normal function. The distribution shows that there is a high probability of the pathogen spreading with low P value. As the variance increases to 0.0001 in Figure 3.1(b), the shape remains similar, but the peak of the distribution is lower than in Figure 3.1 (a). This suggests a broader range of P that measures the arrival time. With the variance increasing to 0.001, in Figure 3.1 (c), the distribution starts to show a noticeable change. The peak has reduced considerably compared to the first two plots (Figures 3.1(a) and (b)), suggesting more variability in P. In Figure 3.1 (d), when variance is 0.01, the peak has been lowered further. In Figure 3.1(e), with the highest variance value used, the peak of the distribution is lowest observed among the five graphs but the range of P is higher. This shows that with higher variances, the predictability of pathogen arrival decreases. These spikes represent the probable time of pathogen arrival, yet the general trend suggests increased uncertainty. Having the highest variance, which is 0.1, the distribution shows the lowest

amount of spread. These results in Figure (3.1) show valuable information on the behaviour of pathogens at the time of arrival at various levels of variability, emphasising the profound influence that low variance has on predictions and model outcomes. As variance increases from Figure 3.1(a)-(e), the histograms show reduced variabilities in the arrival time of the pathogen, indicating a decrease in predictability of pathogen arrival time.

10 **sample trajectories of** $I(t)$

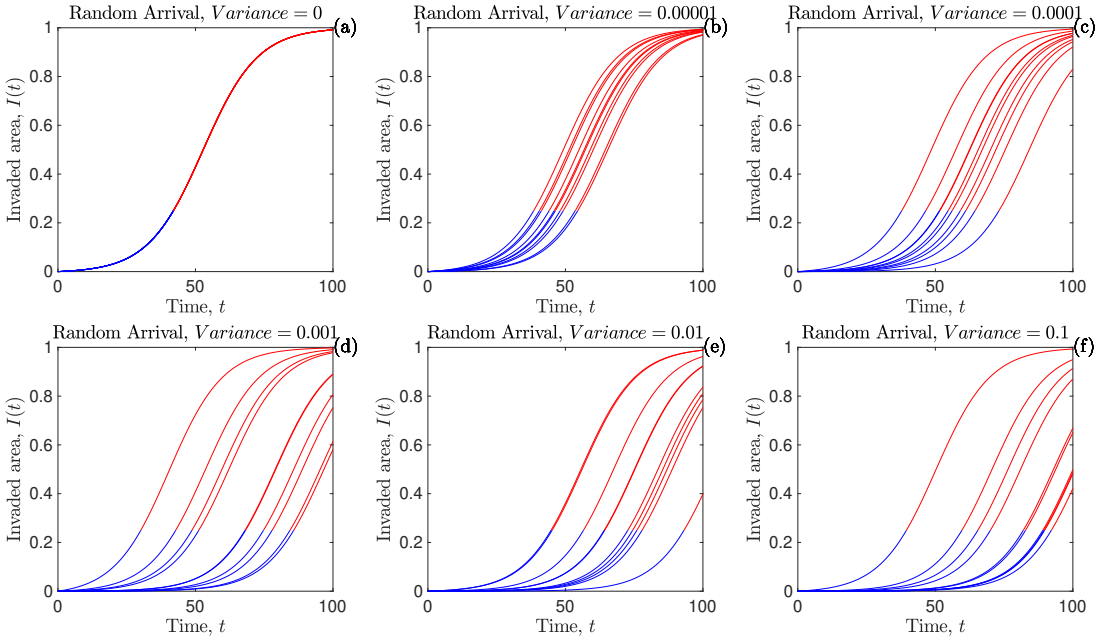

Figure 3.2: The effect of randomly choosing ten (10) values of P from a log-normal distribution. The effect of different values of variance on the area of the infected forest, I(t), with respect to time, t. The infected area, $I(t)$, is given by Equation 3.2 with the primary infection, P, computed with log-normal distribution with mean(μ)= 0.005 and the variance (σ^2) values are (a) variance = 0, (b) variance = 0.00001, (c) variance = 0.0001, (d) variance = 0.001, (e) variance = 0.01, and (f) variance = 0.1. The blue curves represent the detection threshold ($I_1 = 0.25$), while the red part of the curve shows the arrival and spread of the pathogens. All other parameters are at their baseline values in table 2.1.

This section models the progression of an invaded area $I(t)$ over time, where the time evolution of the invasion process is governed by two distinct behaviours before and after a specific time point $\tau(\delta)$. The parameter P is sampled from a log-normal distribution. This indicates

randomness in the model, where different runs will produce slightly different results based on the random value of P. For each value of P, the plots show the growth of the invaded area over time, resulting in multiple overlapping plots that offer insights into the variability and scenarios of the invasion process. The infected area, $I(t)$, is given by Equation (3.2) with the primary infection, P, computed with log-normal distribution with mean (μ) = 0.005 and the variance (σ^2) values are (a) variance = 0, (b) variance = 0.00001, (c) variance = 0.0001, (d) variance = 0.001, e) variance = 0.01, and (f) variance = 0.1. The blue curves represent the detection threshold ($I_1 = 0.25$), while the red part of the curve shows the arrival and spread of the pathogens. All other parameters are at their baseline values in Table (2.1).

The optimal strategy for the stochastic model

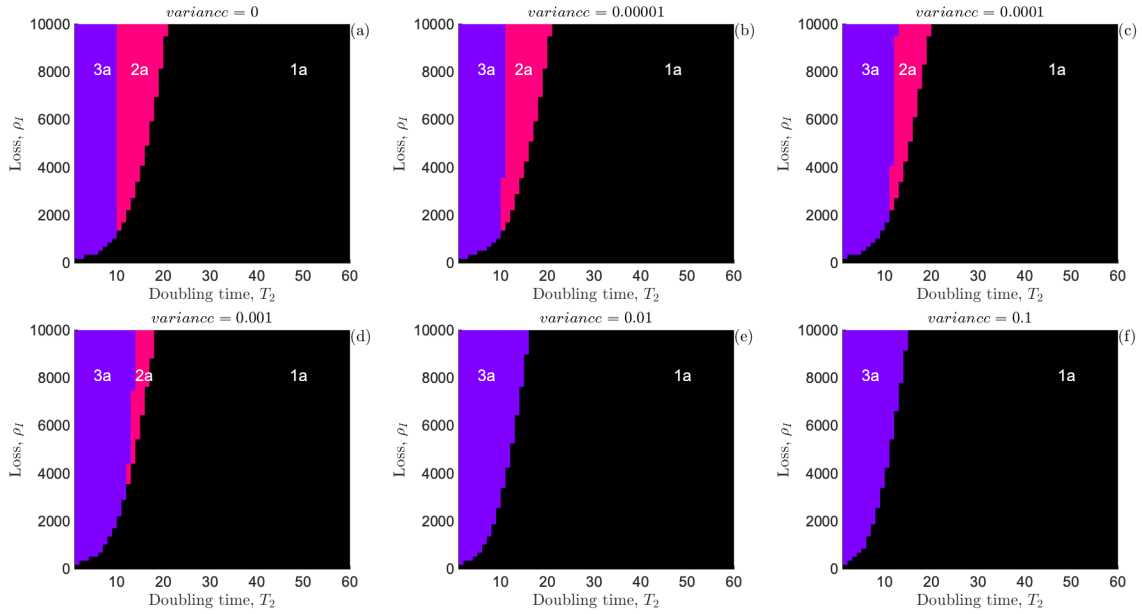

Figure 3.3: Sensitivity analysis to the key parameters when the pathogen arrival is a random process and RM strategy is not available. The probability of arrival was carried out at different variance levels: (a) $Variance = 0$, (b) $Variance = 0.00001$, (c) $Variance = 0.0001$, (d) $Variance = 0.001$ (e) $Variance = 0.01$, and (f) $Variance = 0.1$. The Figure is based on one thousand (1000) values of randomly chosen P. The colours in the figures are detailed in table 3.1. The parameters $\delta_M = 0$, while other parameters are at their baseline values in table 2.1.

Sensitivity analysis in Figure (3.3), shows the effect of random arrival with different values of variance on the invaded area $I(t)$ compared to time, t. The probability of arrival was carried out at different variance (σ^2) level: (a) $\sigma^2 = 0$, (b) $\sigma^2 = 0.00001$, (c) $\sigma^2 = 0.0001$, (d) $\sigma^2 = 0.001$ (e) $\sigma^2 = 0.01$, and (f) $\sigma^2 = 0.1$. The mean ($\mu = 0.005$) is always the same as given in the baseline value (see Table 2.1) while the variance (σ^2) changes from $0 \rightarrow 0.00001 \rightarrow 0.0001 \rightarrow 0.001 \rightarrow 0.01 \rightarrow 0.1$. The regions presented in Figure (3.3) shows the zone $1a$ which means no investment (in black), zone $2a$ which means partial investment (in pink) and zone $3a$ which means full investment (in Purple). The plots in Figure (3.3) demonstrate that as the variance is increasing, the region where the pathogens spread without causing damage shows a slight increase causing zone $2a$ (partial strategy) to disappear at some point. This is as a result of the total loss caused by the pathogens that is lesser than the cost of deploying the PM at a higher variance ($\sigma^2 = 0.01, 0.1$) regardless of how fast the disease transmits within the forest and the time the loss starts to accrue. As variance is increasing ($\sigma^2 \rightarrow 0 \rightarrow 0.00001 \rightarrow 0.0001 \rightarrow 0.001 \rightarrow 0.01 \rightarrow 0.01$), the region where we deploy partial PM strategy decreases and eventually disappears, see Figure 3.3(e) and (f) when variance is increased.

The switch from no PM (zone 1a) to partial PM (zone 2a) occurs at a low doubling time as the loss increases from Figure 3.3(a) to Figure 3.3(d) and disappears in Figures 3.3(e) and 3.3(f). However, the pathogens will arrive and spread at some point if we do nothing to prevent it. So we deploy partial PM to slow down the time the loss will start to accrue. This happens because there is an increase in loss from disease at a small variance, so it is optimal to deploy the PM to counter this loss. The region where we deploy full investment (zone $3a$) increase at a high variance (σ^2). The results in Figure (3.3) show that as the variance is increasing is less preferable to act. This is because Figures (3.3) shows that the pathogen is less likely to arrive and spread at a high variance. Therefore, the log-normal distribution shows that increasing uncertainty will decrease the arrival and spread of the pathogen.

The difference between deterministic and stochastic models using log-normal distribution.

We will now combine the plots from Figures (3.1) - (3.3) to explain how changing the uncertainty in the arrival time (determined by the values P) using log-normal distribution affects the optimal control strategy. This helps us to explain the difference between deterministic and stochastic models. The first plot shows the distribution of P values, the second plot shows 10 sample trajectories of $I(t)$. The optimal strategy for the stochastic model was discussed in the third graph using log-normal distribution with mean 0.005 and different variance values. The deterministic result with the same P value as used in chapter 2 is the fourth plot.

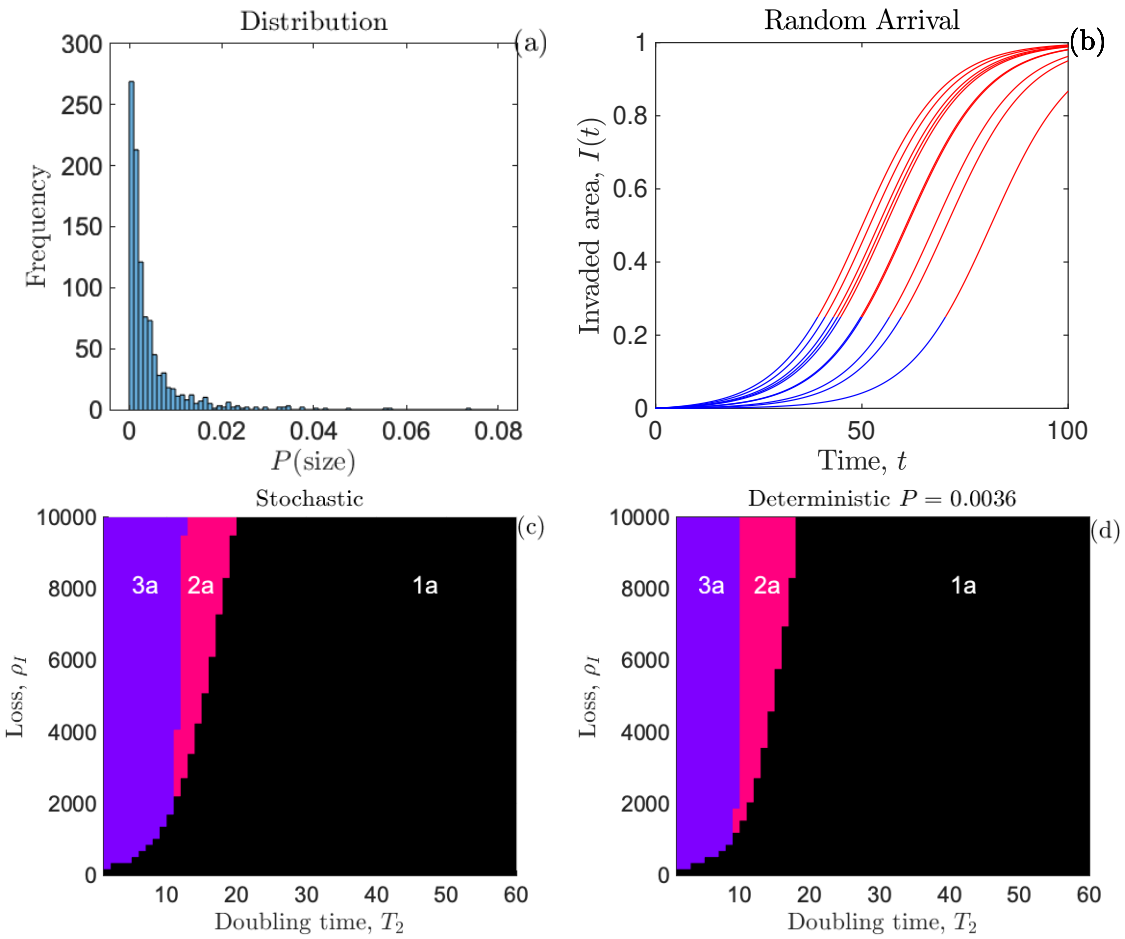

Figure 3.4: Explaining the difference between deterministic and stochastic models. (a) the distribution of P values, (b) 10 sample trajectories of $I(t)$ (blue: before detection, red: after detection), (c) the optimal strategy for the stochastic case, and (d) the deterministic case with same P value using log-normal distribution with mean 0.005 *and variance* 0.0001.

The plots in Figures 3.4(a) and (b) demonstrate that as the variance increases, the region where the pathogens spread without causing damage shows a slight increase. Figure 3.4 (a) shows a high frequency of data with lower values of P but the right tail stretches out to $P = 0.008$. In Figure 3.4 (b), we observe the variability between these 10 trajectories and we notice there are significant differences between the trajectories. Figure 3.4 (b) showing 10 trajectories (10) of randomly chosen P values. Since there is little divergence between the trajectories when the variance is 0.0001, Figure 3.4 (b) is not sensitive to changes in the parameter drown from the distribution. This is because the total loss caused by pathogens

is less than the cost of deploying PM at a higher variance ($\sigma^2 = 0.01, 0.1$) regardless of how fast the disease spreads within the forest and the time the loss. As variance increases, the log-normal distribution of P becomes more skewed: there are more extremely high values of P (early arrival), but this is offset by increase in the number of low values of P (late arrival). This helps us to understand the impact of randomness on the outcome introduced by the log-normal distribution.

Figure 3.4 (c) shows the resulting optimal control strategy for different values of T_2 and ρ_I base on the stochastic model. Figure 3.4 (d) shows the corresponding results for the deterministic model (chapter 2) with P values corresponding to average P in Figure 3.4 (c). We observe that zone 1a starts from a small loss, ρ_I, and extends to a very high loss, as T_2 increases in Figure 3.4(c) similarly to Figure 3.4(d). The switch from no PM to partial PM occurs for the middle doubling time as ρ_I increases in both Figures 3.4(c) and (d). This shows that the pathogens will arrive and spread in zone 2a if we do nothing to prevent it, and we deploy partial PM to delay the time the loss starts to accrue in both the deterministic and the stochastic model. The switch from partial PM to full PM occurs at the low doubling time as the loss increases in both Figures 3.4(c) and (d). The whole forest under investigation will be infected if the full PM is not deployed. Therefore, investing all the resources when it is optimal to deploy full PM will prevent further losses, both for the deterministic model and the stochastic model.

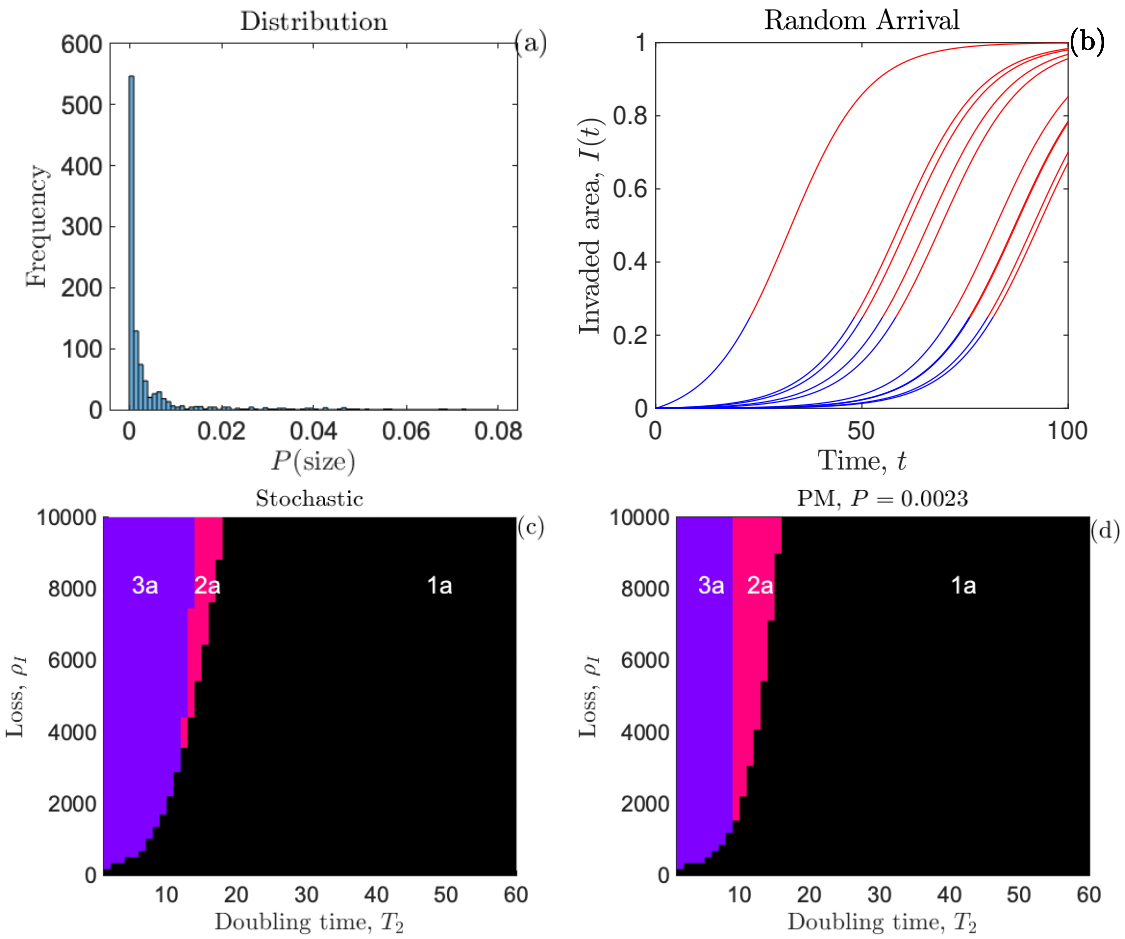

Figure 3.5: Explaining the difference between deterministic and stochastic models. (a) the distribution of P values, (b) 10 sample trajectories of $I(t)$ (blue: before detection, red: after detection), (c) the optimal strategy for the stochastic case, and (d) the deterministic case with same P value using log-normal distribution with mean 0.005 *and variance* 0.001.

As variance increases, Figure (3.5) shows that the pathogen is more likely to arrive both early and late. Therefore, the log-normal distribution shows that increasing uncertainty will increase the arrival and spread of the pathogen. The region where it is optimal to deploy partial PM (zone 2a) reduces as the variance is increased to 0.001, see Figure 3.5(c) compared to Figure 3.5 (d). The region where it is optimal to deploy full PM (zone 3a) increases as the variance is increased to 0.001 in Figure 3.5(c) and compared to Figure 3.5 (d) which the deterministic result. Hence, allocating resources for precautionary measure will help avoid further losses in both the deterministic and stochastic models.

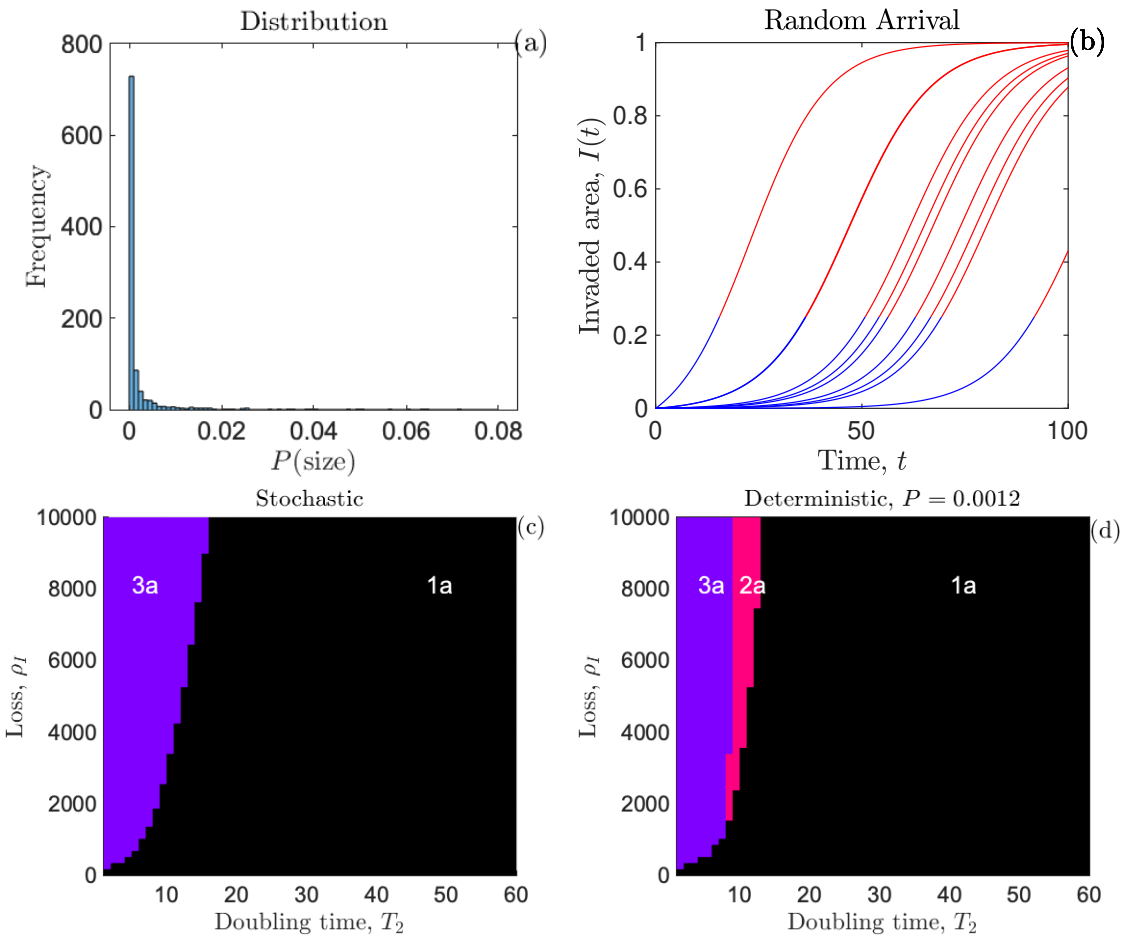

Figure 3.6: Explaining the difference between deterministic and stochastic models. (a) the distribution of P values, (b) 10 *sample trajectories of* $I(t)$ *(blue: before detection, red: after detection), (c) the optimal strategy for the stochastic case, and (d) the deterministic case with same P value using log-normal distribution with mean* 0.005 *and variance* 0.01.

Increasing the variance further reduces the $2a$ zone even further, compared to the corresponding deterministic case. Figure 3.6 (a) shows the spread when variance is 0.01 which suggests that the spread of the P values increases when the variance of the distribution is increased. The histogram shows higher frequencies on the left side of the graph, corresponding to the small P values. While fewer data points have large P values. In Figure 3.6 (b) there is a lot of divergence between trajectories, signifying that the system is highly sensitive to changes in the parameters drawn from the distribution when variance is increased. An increase in the loss increases the potential total loss caused by the disease since the rate at which the

forest will become infected will increase. The Figure 3.6 demonstrates the difference between stochastic and deterministic behaviours in your model, especially with respect to the trajectories of invaded areas and the associated loss when variance is 0.01. The stochastic plot in Figure 3.6 (c) displays two distinct regions, labelled "3a" (in purple) and "1a" (black).

Pathogens can arrive and spread in ecosystems in various ways, however, how we understand and interpret these arrivals can influence our management strategies. The doubling time indicates the rapidity of the pathogen's spread. A shorter doubling time suggests faster spread, leading to greater losses, especially in the initial phases. The stochastic result 3.6 (c) reflects real-world uncertainties, indicating that even with the same conditions, the spread of the pathogen and its resultant loss can vary due to random factors. The deterministic result 3.6 (d) provides a predictable outcome for pathogen spread under a set of specific conditions. The presence of the 2a (pink) region suggests that there are scenarios in which a more controlled or manageable spread can be achieved, leading to reduced losses. In both models, there's an emphasis on the importance of early management. Initial rapid spread (low doubling time) leads to high losses, emphasising the critical nature of early detection and intervention. As the pathogen spread stabilises, losses remain high, indicating the long-term impacts of uncontrolled pathogen spread.

3.3.2 Absolute values from a normal distribution

The distribution of P values

The log-normal distribution describes the situation when there is a large probability that the pathogen arrives late (small P) and a very small probability that it arrives early (large P) although it can arrive very early (very large P). In order to capture a situation with a more even distribution of P, we use the absolute values from a normal distribution. The baseline primary infection, P, is generated from a normal distribution (Gaussian distribution), and then taking the absolute values of the outcome. However, the idea of this approach is to transform the deterministic models into a stochastic process. The variance, σ^2, measures the average degree

to which each number is different from the mean. The variance offers the model flexibility in specifying the shape of the pathogen's arrival. Due to the uncertainty of arrival, we model from the smaller to high variance values; this helps us capture the uncertainty. The infected area, I(t), is given by Equation (3.2) with the primary infection, P, computed with absolute values from a normal distribution with mean(μ)= 0.005 and the variance (σ^2) values are (a) variance = 0.00001, (b) variance = 0.0001, (c) variance = 0.001, (d) variance = 0.01 and (e) variance = 0.1. If $\sigma^2 = 0$, the model default to the deterministic model in Chapter (2). We generate an array of positive random numbers with N vector size. Hence, the model describes the range of possible outcomes given the uncertainty in the input variable.

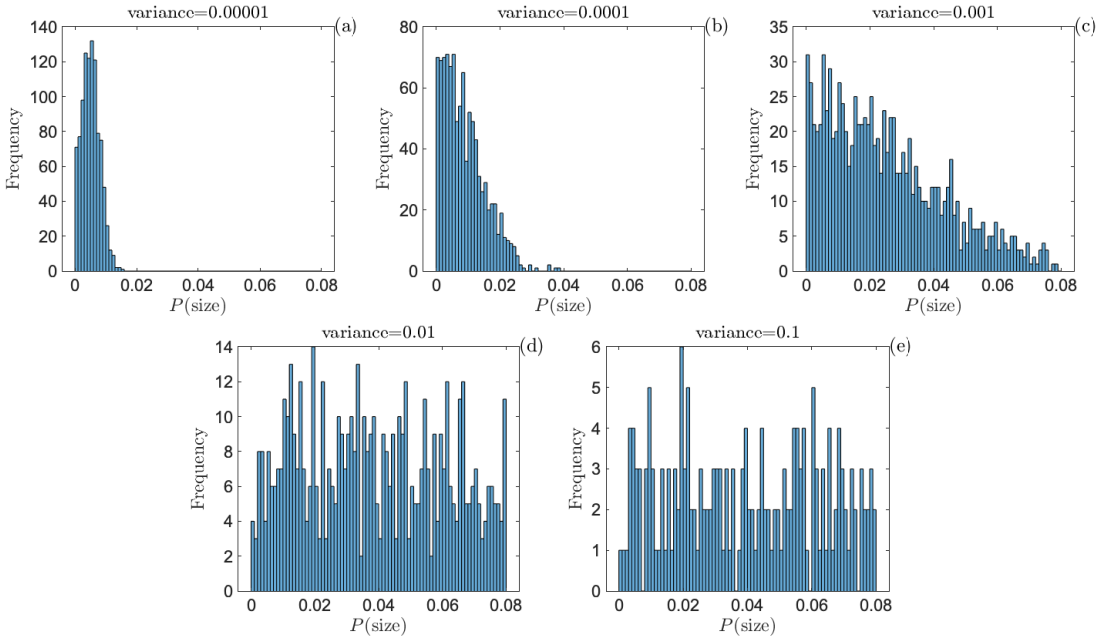

Figure 3.7: Histograms of the absolute values from a normal distribution function. The probability of arrival and spread of pathogens was calculated at different variance level: (a) variance = 0.00001*, (b) variance* = 0.0001*, (c) variance* = 0.001*, (d) variance* = 0.01 *and (e) variance* = 0.1*. The Figure is showing one thousand (1000) values of randomly choosing P.*

The plots in Figure (3.7) present the abs-normal distribution function to model the probability of arrival across different levels of variance. The displayed histograms provide a clear visualisation of how increasing variance affects the frequency distribution of the pathogen sizes. With the inclusion of the absolute values, these distributions avoid negative values,

which could be inapplicable in the context of pathogen arrival time. The progression from low variability in Figure 3.7(a) to high variability in Figure 3.7(e) serves to demonstrate the impact of variance on modelling outcomes. The histogram in Figure 3.7(a) showcases the frequency distribution with a very low variance of 0.00001. With such a low variance, the distribution is narrowly focused, representing a strong predictability in the pathogen arrival time. A noticeable spread can be seen in Figure 3.7(c) compared to the previous two histograms Figure 3.7(a) and (b), suggesting a broader range of potential pathogen arrival time. When variance is 0.001 as shown in Figure 3.7(c), the histogram still presents a wide range of sizes, indicating increased variability. At this level of variance, the distribution is quite spread out with numerous spikes when equal to variance 0.001. These spikes represent the probable time of pathogen arrival, yet the general trend suggests increased uncertainty. Having the highest variance, which is 0.1, the distribution shows the highest amount of spread. These results in Figure (3.7) show valuable information on the behaviour of pathogen arrival time at various levels of variability, emphasising the profound influence that variance has on predictions and model outcomes. As variance increases from Figure 3.7(a)-(e), the histograms show wider spreads, indicating a decrease in predictability of pathogen arrival time.

10 **sample trajectories of** $I(t)$

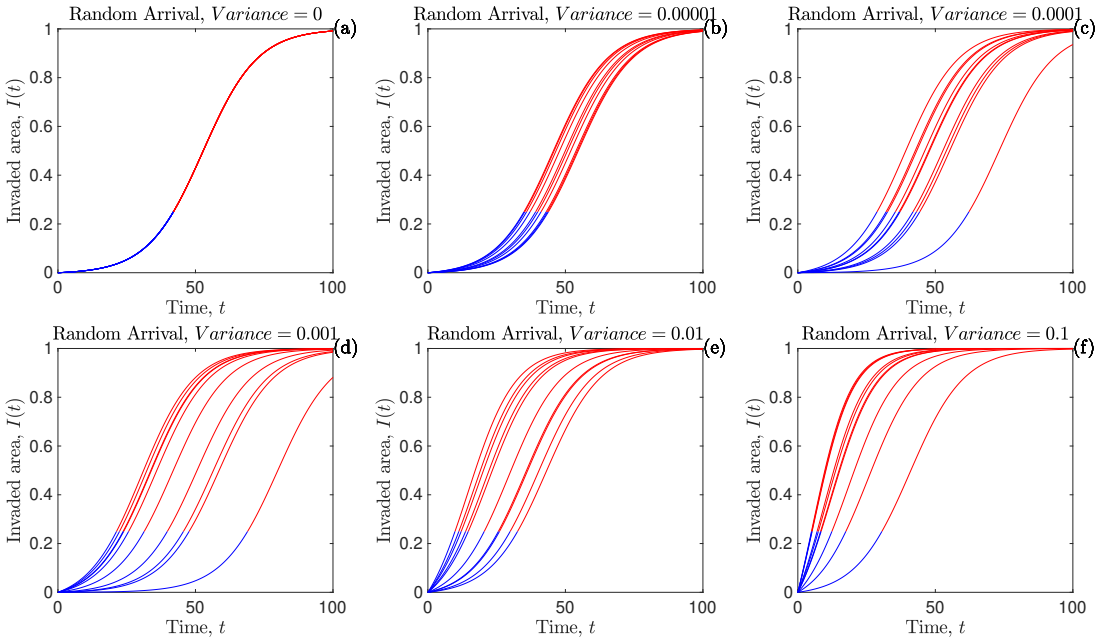

Figure 3.8: The effect of randomly choosing ten (10) *values of P from the absolute of a normal distribution. The effect of different values of variance on the area of the infected forest,* $I(t)$*, with respect to time,* t*. The infected area,* $I(t)$*, is given by Equation 3.2 with the primary infection, P, computed with absolute values from a normal distribution with mean*$(\mu) = 0.005$ *and the variance* (σ^2) *values are; (a)* 0*, (b)*= 0.00001*, (c)* 0.0001*, (d)* 0.001*, (e)*= 0.01*, and (f)* 0.1*. The blue curves represent the detection threshold* $(I_1 = 0.25)$*, while the red part of the curve shows the arrival and spread of the pathogens. All other parameters are at their baseline values in table 2.1.*

We show the effect of random arrival with different variance values on the invaded area $I(t)$ compared to time, t, in Figure (3.8). The mean is always the same, while the variance changes from $0 \rightarrow 0.00001 \rightarrow 0.0001 \rightarrow 0.001 \rightarrow 0.01 \rightarrow 0.1$. Due to the uncertainty of our model, we use this analysis to determine how likely the pathogen is to arrive and how fast the infection spreads within the forest. Figure (3.8) (a) shows how quickly the disease spreads within the forest when variance = 0, which is similar to Figure 2.8 (b) when the primary infection of the baseline, P, is 0.005. Then Figure 3.8 (b) shows how the arrival and spread of the disease within the forest is slightly increased to 0.001, while Figure 3.8(c) shows when the variance is 0.01. The results show that as the variance is increasing, is more preferable to do something to

prevent further spread of the pathogens. This is because Figure (3.8) shows that the pathogen is more likely to arrive and spread earlier with high variance.

The optimal strategy for the stochastic model

In figure (3.9), we present the sensitivity analysis to the key parameters; annual loss, ρ_I and double time, T_2, when the arrival of the pathogen is a random process and the RM strategy is not available. The relationships between the colours (zones) are given in detail in Section (3.3) and Table (3.1). The approach used in the deterministic model (Chapter (2)) and the stochastic model is different, but the results are similar. The effect of different variance values with the stochastic model is shown in Figure (3.9). In contrast, the deterministic results were shown in Figure (2.8) using different values of the primary infection, P. The analysis in Figure (3.9) shows similar results to the sensitivity analysis to the primary infection, P (see Figure (2.8) in Chapter (2).

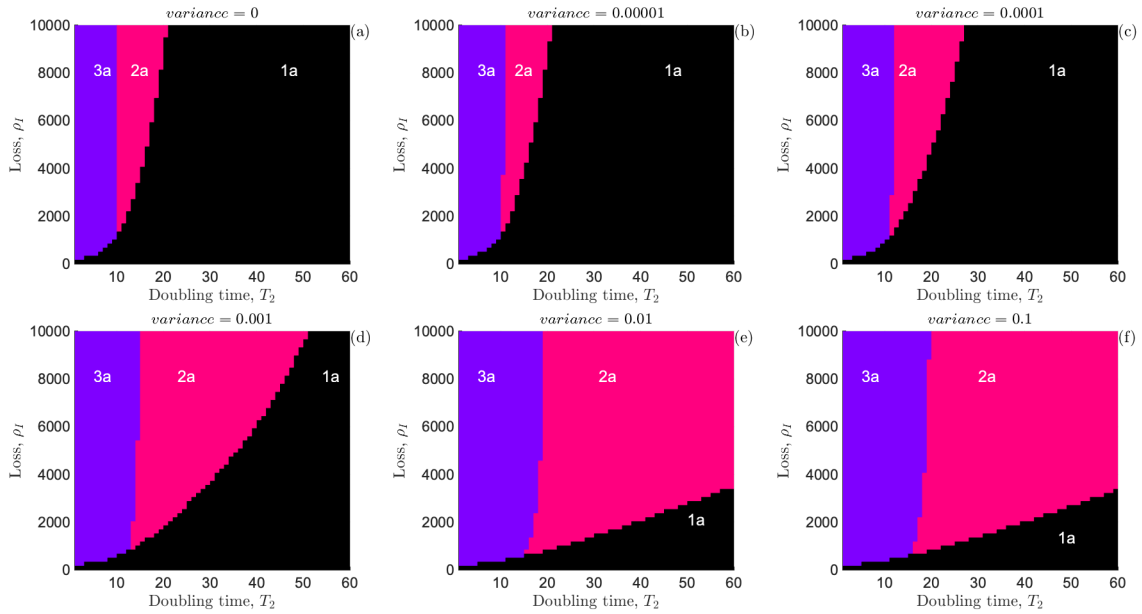

Figure 3.9: Sensitivity analysis to the key parameters when the pathogen arrival is a random process and RM strategy is not available. The probability of arrival was carried out at different variance levels: (a) $Variance = 0$, (b) $Variance = 0.00001$, (c) $Variance = 0.0001$, (d) $Variance = 0.001$ (e) $Variance = 0.01$, and (f) $Variance = 0.1$. The Figure is based on one thousand (1000) values of randomly chosen P. The colours in the figures are detailed in table 5.3. The parameters $\delta_M = 0$, while other parameters are at their baseline values in table 2.1.

Figure (3.9)b and (c) present the arrival and spread of the disease within the forest when the variance increases slightly to 0.001 and 0.01, respectively. Figure (3.9)d shows that the pathogens is more likely to arrive and spread at a higher variance (variance = 0.1). In figure (3.9) d and figure (2.8)c, it can be seen that the higher the variance ($\sigma^2 = 0.1$) and the primary infection, $P = 0.1$, the more likely the pathogen is to arrive and spread. Moreover, this shows that a slight increase in the purple region ($zone3a$) and the pink region ($zone2a$) as the black region ($zone1a$) decreases. The finding in Figure (3.9) suggests that an increase in the loss increases the potential total loss caused by the disease since the rate that the whole forest will become infected will increase. One possible implication of this result is that as the doubling time, T_2 decreases, the optimal level of the PM increase. Taken together, the analysis from Chapter (2), the results suggest that when it is optimal to deploy PM, the optimal level of PM is always enough so that the loss does not start within the time horizon.

The difference between deterministic and stochastic models using abs normal distribution.

We will now combine the graphs from Figures (3.7) - (3.9) to explain how changing the uncertainty in the arrival time using the abs normal distribution affects the optimal control strategy. This helps us to explain the difference between deterministic and stochastic models. The first plot shows the distribution of P values, the second plot shows 10 sample trajectories of $I(t)$. The optimal strategy for the stochastic model was discussed in the third graph using the abs normal distribution with mean 0.005 and different variance values. The deterministic result with the same P value as used in chapter (2) is the fourth plot. This helps us to understand the impact of randomness on the outcome introduced by the abs normal distribution.

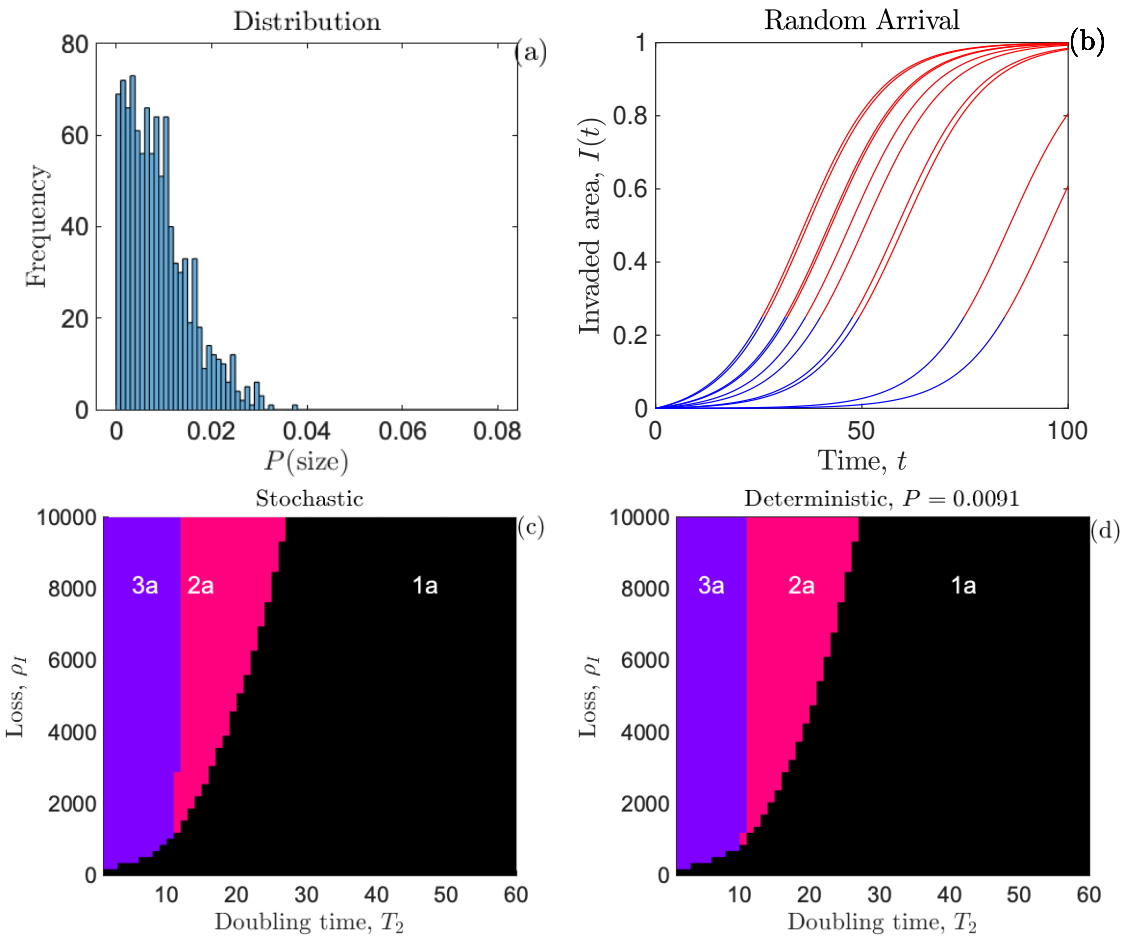

Figure 3.10: Explaining the difference between deterministic and stochastic models. (a) the distribution of P values, (b) 10 *sample trajectories of $I(t)$ (blue: before detection, red: after detection), (c) the optimal strategy for the stochastic case, and (d) the deterministic case with the same P value, using absolute values from a normal distribution with mean* 0.005 *and variance* 0.0001.

The distribution plot in Figure 3.10(a), illustrates the distribution of the parameter P, based on its size. The bars show the frequency distribution of different sizes. The higher bars around the 0.02 mark suggest that the most common P value is around this point. As the size increases towards 0.08, the frequency of these sizes decreases sharply. In Figure Figure 3.10 (b) depicts how the invaded area changes over time, the trajectories show the spread of invasive species over time. The blue lines represent slower spreads, reaching a smaller invaded area even after a long time. The red lines indicate faster and more aggressive

spreads, covering a large area in a short time. Figure 3.10 (c) shows an uncertain behaviour regarding losses incurred due to the spread of a pathogen. High loss incurred even when the spread is slow at a high doubling time. As the speed of spread increases at a lower doubling time, the loss starts to decrease but is still significant. Similarly to Figure 3.10 (c), this one offers a predictable outcome given specific initial conditions or inputs. The regions and their implications remain the same, with zone 1a denoting high losses for slower spreads, zone 2a indicating decreased losses for moderate spreads, and zone 3a indicating high stabilised losses for fast spreads. The distribution in Figure 3.10 (a) suggests that smaller sizes of infected areas are more common. The trajectories in Figure 3.10 (b) emphasise the rapid spread of pathogens over time. The stochastic model in Figure 3.10 (d) highlights the unpredictability in real-world scenarios, where various factors can influence the doubling time and associated losses. The deterministic model in (d) provides a predictable progression of pathogen spread.

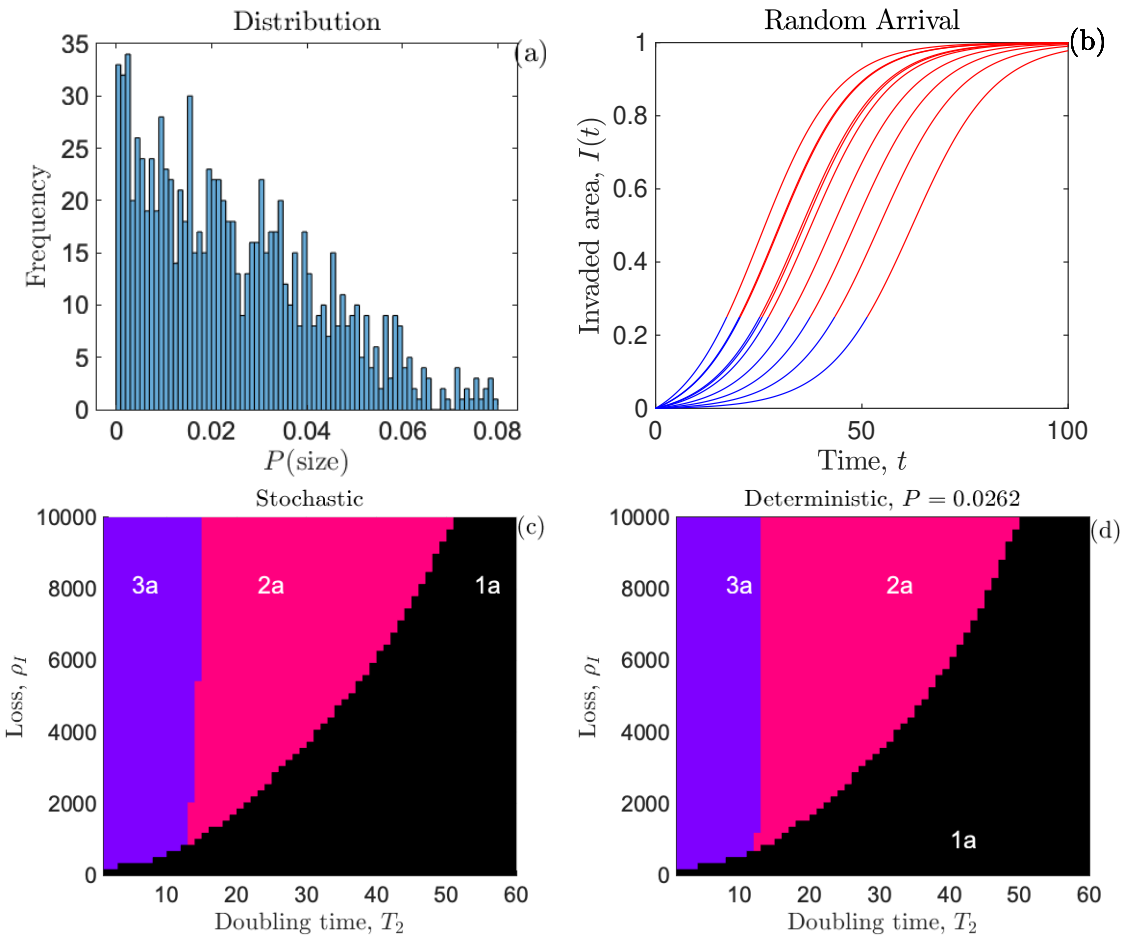

Figure 3.11: Explaining the difference between deterministic and stochastic models. (a) the distribution of P values, (b) 10 *sample trajectories of $I(t)$ (blue: before detection, red: after detection), (c) the optimal strategy for the stochastic case, and (d) the deterministic case with the same P value, using absolute values from a normal distribution with mean* 0.005 *and variance* 0.001.

The distribution in Figure 3.11 (a) appears right-skewed, which indicates a higher frequency of smaller P sizes with a gradual decrease in frequency as the size grows. However, Figure 3.11 (b) shows the invasion dynamics of a pathogen with respect to time. The trajectories denote different variations of pathogen spread. The trajectories display a rapid increase, hinting at exponential growth which are common trend observed in pathogen spread. The stochastic plot in Figure 3.11 (c) underlines the unpredictable nature of real-world scenarios. Even under the same conditions, various unpredictable elements can influence the doubling

time and consequent losses. Figure 3.11 (d) conveys a deterministic model of the spread, whereby the pattern resembles the stochastic model, as it is deterministic, transitions between the regions are more defined.

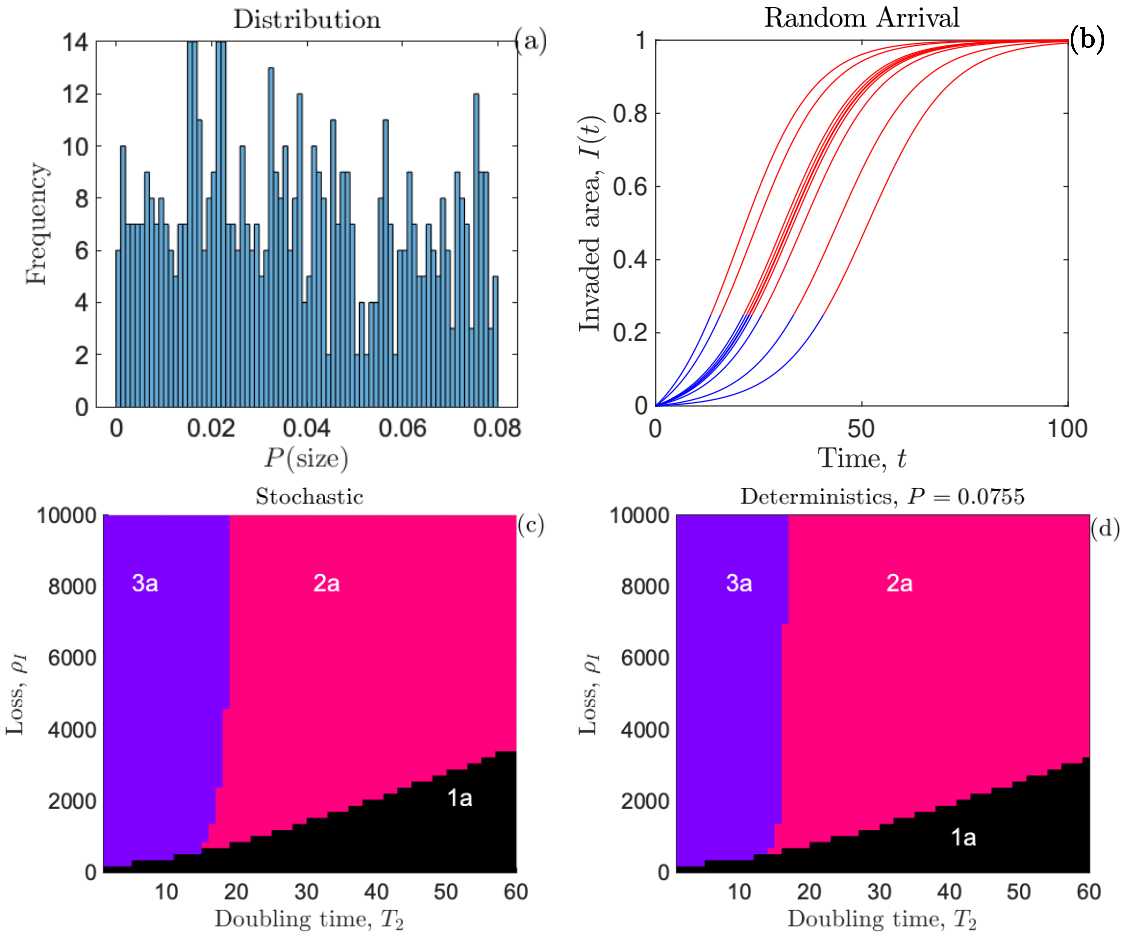

Figure 3.12: Explaining the difference between deterministic and stochastic models. (a) the distribution of P values, (b) 10 *sample trajectories of $I(t)$ (blue: before detection, red: after detection), (c) the optimal strategy for the stochastic case, and (d) the deterministic case with the same P value using absolute values from a normal distribution with mean* 0.005 *and variance* 0.01.

The distribution in Figure 3.12 (b) has a more irregular pattern, suggesting more variability or noise in the data. Given the mentioned variance of 0.01, this indicates a spread around the mean of the normal distribution, implying a moderate level of variability. There are 10 trajectories in Figure 3.12 (b), representing different possible paths of spread. The stochastic

heat map Figure 3.12 (c) emphasises the unpredictability inherent in real-world scenarios, even if initial conditions remain consistent. The variance of 0.01 indicates that the spread of outcomes around the mean is quite limited. The deterministic heat map Figure 3.12 (d), although visually similar to (c), is based on fixed value of P. Given the variance of 0.01 in the stochastic model, outcomes tend to cluster closely around the average, indicating reliable predictability. The colours in the heat maps for both (c) and (d) provide a clear visual representation of the loss levels for various doubling times, offering a user-friendly tool for risk assessment and planning.

3.4 Discussion

The model used in this chapter incorporates randomness into the existing deterministic model (Chapter (2)). Our analysis reveals how introducing randomness the initial proportion infected when the pathogen arrives significantly alters the optimal PM strategy. This analysis is crucial to understanding the limits of the effectiveness of PM in different epidemiological scenarios. Comparing these results with the deterministic model in Chapter (2) highlights the added complexity and realism brought about by stochastic elements. Although the deterministic model offers a baseline understanding, the stochastic model provides insights into the uncertainty inherent in real-world pathogen dynamics. The outcomes are not entirely determined by the initial conditions, but have a random component. A distribution in which the logarithm of the variable follows a normal distribution. This is essentially the magnitude of a value from a normal distribution without regard to its sign. The logarithmic normal distribution is inherently positive. The absolute values from a normal distribution creates a half-bell curve, where values are always non-negative.

We highlight the significance of this variability by performing different types of analysis using the absolute values from a normal distribution and a log-normal distribution. In this chapter, the optimal management strategy for stochastic analysis was performed for the precautionary management strategy. We have four types of plot with different variances

(variance = $0.00001, 0.0001, 0.001$), showing the distribution plot, the trajectories plot, the stochastic plot, and the deterministic plot. The stochastic analysis was performed with annual loss due to infection,ρ, and doubling time , T_2, using different variances. This analysis helps us to compare the stochastic result with the deterministic results. This comparison underscores the importance of considering variability in pathogen transmission rates for effective disease management in forests. Our analysis provides critical insights into how stochastic variations in disease transmission rates can influence the spread of pathogens and the effectiveness of PM strategies. These insights are invaluable for forest management, emphasising the need for flexible and adaptive strategies that can accommodate the inherent uncertainties of natural ecosystems.

We seek to understand how the inherent uncertainties in pathogen behaviour and spread can be modelled and how these uncertainties influence the decision-making process in forest disease management. A key aspect of this analysis is the consideration of randomness in the primary infection, P, which significantly alters the landscape of potential outcomes and strategies. We engage in a detailed examination of how varying levels of uncertainty, represented by different variances in the log-normal distribution of P values, impact the optimal deployment of PM strategies. These tests are crucial in evaluating the effectiveness of different management strategies in various scenarios of uncertainty and risk. By simulating multiple scenarios and analysing the results, we gain valuable insight into the interaction between the random arrival of the pathogen, the spread of the disease, and the effectiveness of the management.

Understanding the distribution is key to predicting extreme events. If the model leans heavily towards a log-normal distribution, there is always a non-zero probability of encountering very large values, which might be crucial for risk management. The absolute values from a normal distribution might be useful for phenomena where direction does not matter (e.g., loss is loss, regardless of the cause or direction of the initial deviation). When working with stochastic models, recognising the underlying distributions is vital. The log-normal distribution emphasises the multiplicative nature of certain phenomena and the ever-present

risk of large but rare events. Meanwhile, the abs normal distribution provides insight into the magnitude of deviations from the mean, without concerning the direction of those deviations. Incorporating these distributions provides a richer and more clear understanding of potential outcomes and risks, helping better decision-making and planning.

Comparing the observed distribution of pathogen arrival events with the expected distribution predicted by a model allows researchers to assess how well the model aligns with real-world data. Comparing these stochastic model results with the deterministic outcomes from Chapter 2, we see that including stochastic elements in our model adds a layer of realism. The deterministic model offers a baseline prediction under controlled conditions, while the stochastic model accounts for the unpredictability inherent in natural systems. As the variance in the primary infection, P, increases, we observe a tendency towards an earlier implementation of PM, as indicated by the shift from the zones of partial control to the full control. This finding is particularly relevant for forest managers, suggesting that a higher degree of uncertainty in disease transmission rates requires more aggressive management interventions.

The analysis in this chapter shows that higher variability in the timing of pathogen arrival has important implications for the optimal management choice. In both parameterisations, an increase in variance reduces the region in which a 'partial' (invest-some) precautionary management strategy is optimal and expands the region where the 'invest-all' strategy dominates. This is intuitively consistent with the behaviour of risk-neutral decision-makers: when the risk of early arrival and large losses increases, it becomes optimal to allocate the full budget to prevention in order to minimise expected (average) losses. The infrequent, very large outbreaks caused by extremely early arrival have only a limited effect on the expected-loss criterion because they still occur with low probability. In the case of the abs-normal distribution, even when the mean arrival time is unchanged, a higher variance shifts mass toward early arrivals and thus expands the 'invest-all' region at the expense of the 'do-nothing' region.

The findings of this chapter underline the importance of incorporating uncertainty into

pathogen arrival models when informing plant health policy. Unlike deterministic approaches, uncertainty-based models capture the variability and unpredictability inherent in the dynamics of real-world diseases. This has direct policy applicability; it suggests that intervention thresholds should be based on ranges rather than fixed values, that resources can be allocated more efficiently by focusing on periods or regions of highest risk, and that management strategies should be adaptive and responsive to new information as uncertainty is reduced. By explicitly recognising uncertainty in decision frameworks, land managers can design more resilient, flexible, and cost-effective strategies to prevent or mitigate the impact of invasive plant diseases.

Under a risk-neutral framework that focuses on expected rather than worst-case losses, higher variability in arrival times strengthens the case for full precautionary investment, not for reduced investment. The key is to strike a balance between being prepared for the worst-case scenarios while also being efficient in the average or most likely scenarios. Incorporating a stochastic element requires the decision-makers to consider not only the immediate costs and benefits but also the range of potential outcomes and their likelihoods. This approach provides a more strong strategy, especially when dealing with unpredictable factors like disease outbreaks. Deterministic models offers initial insights and broad strategies, while stochastic models refine these strategies, considering uncertainties and providing a more meaningful understanding of risks and potential outcomes. This issue is explored in the next chapter.

Chapter 4

Risk Attitude

4.1 Introduction

In this chapter, we delve deeper into the complexities of decision-making under uncertainty by incorporating an exponential disutility function into our existing stochastic model. We introduce the exponential disutility function in section 4.1 and the development of the risk attitude model introduced in section 4.2 combined with the stochastic model. This approach allows us to analyse not only the expected outcomes, but also the land manager's attitudes towards risk. The purpose of the chapter is to minimise infection loss through an adapted disutility-based objective function and to find the degree of risk associated with different management strategies. The core of this chapter revolves around the concept of exponential disutility functions, both negative and positive, to capture the risk preferences of decision maker, represented by land manager in our context. Through this approach, we can better tailor management strategies to align with the risk attitudes of decision maker, ultimately leading to more effective and personalised disease management solutions.

Using exponential disutility functions, we explore how varying degrees of risk tolerance, from risk-averse to risk-seeking behaviours, influence the selection of precautionary management strategies. Utilising a stochastic model enhanced with exponential disutility functions, we examine the decision-making process under different risk attitudes. This approach allows

us to simulate a range of scenarios, from conservative to aggressive disease management, based on the modelled risk preferences. By integrating risk attitudes into disease management models, this chapter contributes to a more refined understanding of decision-making in uncertain environments. The insights gained are not only academically significant but also offer practical guidance for decision maker in crafting more effective and adaptable disease management strategies.

We present the results in section 4.3, and the simulation examines how the optimal level of PM can minimise the cost associated with loss. Sensitivity analysis to the degree of risk aversion when variance = 0, 0.0001 and 0.1 is presented in 4.3.2. We also explore the sensitivity analysis to the risk preference parameter in the precautionary management strategies in Section 4.3.6. Sensitivity analysis was performed to explore how model outcomes change under different conditions of disease spread and management costs. In this chapter, we concentrate on the PM strategy and the chapter discussion is in section 4.4.

4.2 Model development

In this chapter, the negative and positive exponential utility function is used to analyse the risk preferences of the decision maker. Forest managers' risk attitudes and risk management decisions were analysed by using the disutility function. In the study of farmer risk preferences, the negative exponential utility function has been regularly used in agricultural economics [55, 152]. This function is particularly useful because it implies constant absolute risk aversion (CARA), meaning that a farmer's aversion to risk remains consistent regardless of changes in wealth. The function is mathematically expressed as:

$$U_C = \begin{cases} \frac{1-e^{-\lambda C}}{\lambda} & \lambda < 0, \lambda > 0 \\ C & \lambda = 0 \end{cases} \tag{4.1}$$

C is a variable that the decision maker refers to as loss which represents the total cost, including the cost of disease and the cost of control (PM), while λ is a constant that represents the parameter of risk preference. Thus, $\lambda < 0$, corresponds to risk aversion, $\lambda = 0$ to risk neutrality, and $\lambda > 0$ to risk seeking. In other words, λ controls the curvature of the cost function: risk-averse managers ($\lambda < 0$) treat large losses as particularly undesirable and therefore tend to favour more precautionary strategies, whereas risk neutral managers ($\lambda = 0$) simply minimise the average loss.

Formally, the decision-maker's problem is to select the level of precautionary management (PM) that minimises expected disutility:

$$\min_{PM} \mathbb{E}[U(C)] = \min_{PM} \mathbb{E}\left[\frac{1 - e^{-\lambda C}}{\lambda}\right] \tag{4.2}$$

where total cost is given by

$$C = C_{PM} + C_D \tag{4.3}$$

with C_{PM} denoting the cost of prevention investments and C_D the damages from infection. The exponential disutility function ensures that the decision-maker's preferences over risky outcomes depend on both the mean and the variability of these costs.

This formulation captures the idea that decision-makers are risk averse to both prevention costs and infection damages. In practice, committing large sums to prevention is itself uncertain and costly: funds may be wasted if the outbreak does not materialise, budgets are often constrained, and irreversible investment decisions carry political or financial risk. Hence, managers may be reluctant to adopt strategies that involve large or highly variable prevention expenditures.

By contrast, in a model where the decision-maker is assumed to be risk averse only to damages (and risk-neutral toward prevention costs), prevention investments are treated as certain, linear outlays. Such a formulation tends to favour high levels of prevention whenever damages are large or variable, as the decision-maker has no aversion to variability in

prevention spending. However, this fails to reflect observed behaviour, where managers often underinvest in prevention due to the perceived risks of committing resources upfront. Therefore, by treating both C_{PM} and C_D as subject to risk aversion, our model provides a more realistic representation of the decision-making process. It reflects the fact that managers balance the uncertain damages of infection against the equally uncertain (and often politically sensitive) costs of prevention, yielding management strategies that are more consistent with actual behaviour under uncertainty.

The shape of an individual's disutility function determines their level of risk aversion. The sensitivity analysis is done with four different values of variance (Variance = $0, 0.00001, 0.0001, 0.1$). The disutility function was introduced to the stochastic model in chapter 3. These analyses were performed using 1000 random choice values P and six different values of λ ranging from -0.01 to 0.01.

4.3 Results

In this section, some data were produced by introducing a risk attitude to the stochastic model by continuing section 3.3. In Chapter 3 above, uncertainty was introduced to a log-normal distribution to describe the natural variability in the infection dynamics, which shows a decrease in the willingness of the land manager to invest. We include risk attitude by incorporating the exponential disutility function in our model. The simulated data examine how the optimal level of PM can minimise the cost associated with loss.

4.3.1 Relationships between non-linear disutility function and cost

Figure 4.1 shows the relationship between the cost and disutility for different values of λ. The disutility function used in Figure 4.1 shows how decision maker perceive the loss. So, the negative values of $\lambda(-0.01, -0.001, -0.0001)$ will mean risk-averse, while the positive values of $\lambda(0.0001, 0.001, 0.01)$ will mean risk-seeking. The mean feature of risk aversion in Figure 4.1 is that the disutility function shows a convex curve with negative values of λ.

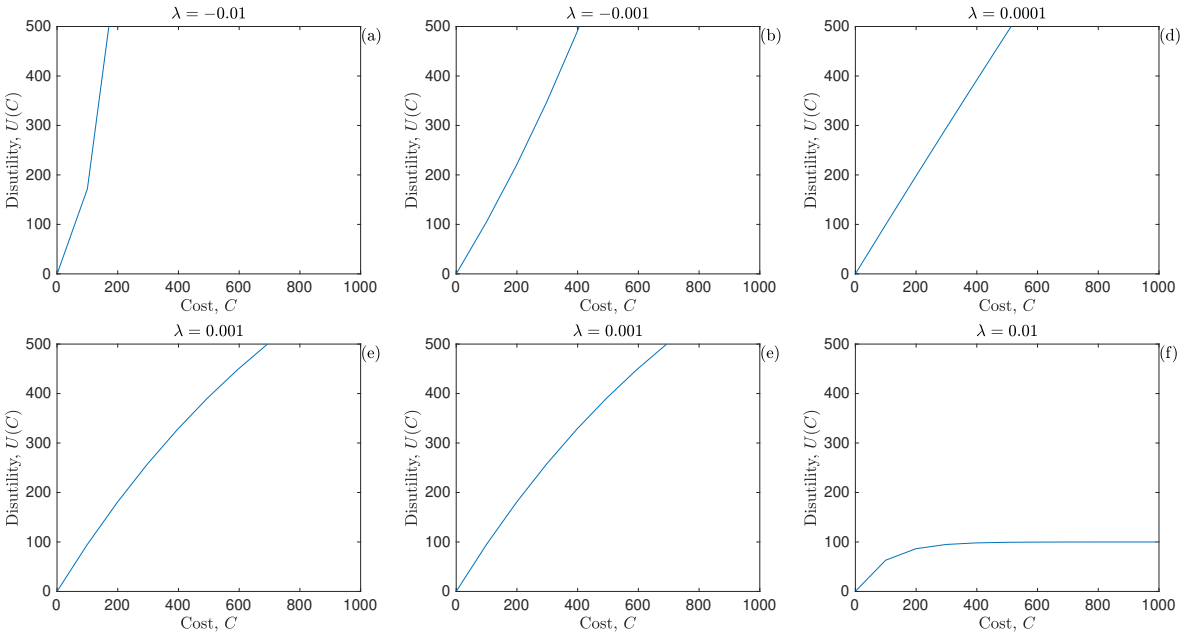

Figure 4.1: Relationships between non-linear disutility function and total cost. This diagram expands upon the established concepts of exponential disutility functions when the risk preference parameter is (a) lambda=-0.01, (b) lambda=-0.001, (c) lambda=-0.0001, (d) lambda=0.0001, (e) lambda=0.001, and (f) lambda=0.01.

In Figure 4.1, it is observed that the more significant the damage, the more it is advisable to invest in control. Figure 4.1(a) shows that we have high disutility at a low cost. However, figure 4.1(f) shows that we have low disutility at a very high cost. Figure 4.1(c,d) shows the disutility function when the value of λ(-0.0001 and 0.0001) slightly decreases and increases above the risk neutral respectively. The key distinguishing feature of risk aversion is that the disutility curve is concave at high levels of damage.

Risk-seeking decision-makers are those who accept greater uncertainty in invasion outcomes, prioritising short-term savings or flexibility over the mitigation of potential high damages. For example: Budget-constrained managers may deliberately underinvest in precautionary measures, accepting the possibility of higher infection costs in the future to preserve scarce resources today. Managers under uncertainty about the arrival of the disease may gamble on the chance that an outbreak will not occur, preferring to risk larger damage later rather than incur certain costs now.

4.3.2 Precautionary Management with disutility Function

The numerical evaluation of exponential disutility functions with random variables using precautionary management is demonstrated in this section. This analysis evaluates the effect of different variances in the area of the infected forest, I(t), over time. Mathematical functions with concave curvatures can be used to describe disutility functions. The level of risk tolerance determines how much risk an investor is willing to take on. The disutility function captures how the forest manager perceives the total loss (i.e., damage from the disease plus the cost of precautionary management). If the disutility function is concave, the manager is risk averse, meaning that large losses are perceived as disproportionately more harmful than small losses. In practical terms, a risk-averse manager would prefer to invest heavily in precautionary measures even when the expected damage is moderate, because they place high value on avoiding the rare but catastrophic outbreaks that result in very large losses. Conversely, if the disutility function is convex, the manager is risk seeking and is therefore more willing to accept the possibility of a large loss in exchange for the chance of incurring little or no cost (e.g., by not investing in precautionary management). In this case, the decision maker prefers to delay or avoid investment unless the expected damage is extremely high, as they are prepared to take the risk that the disease may arrive late or have a limited impact.

Forest managers do not value all losses equally: small losses may be regarded as acceptable or tolerable, whereas larger losses are perceived as increasingly undesirable and therefore receive greater weight in the decision-making process. In this way, minimising the cost of control for different values of λ, we examine how variance changes affect different risk levels (λ). In Chapter 2, Table 2.1 presents the parameters used for this analysis. To test sensitivity to PM, random values of P were used when the variance is $0, 0.0001$ and 0.1, with $\lambda = -0.01, -0.001, -0.0001, 0.0001, 0.001$ and 0.01. Without RM strategy, the optimal management strategy can be determined by Equation (2.7) and Table (4.1). As the optimal level of PM is zero $\left(C_p^{**} = 0\right)$, it is called 'none' (management strategy $1a$, black). When the optimal level of PM is between zero and the full budget $\left(0 < C_p^* < \Pi\right)$ it is labelled 'partial' (management strategy $2a$, pink) . When it is optimal to spend all the PM budget $\left(C_p^* = \Pi\right)$, it is

labelled as the 'full' (management strategy $3a$, purple). The colours show the switch from full PM, zone $3a$ (purple) when the losses are very high, to partial control, zone $2a$ (pink) when the losses are moderately high, to no control, zone $1a$ (black), when the damages are less.

Optimal management strategy	PM (Optimised)
1a (black)	None, $C_p^* = 0$.
2a (pink)	Partial, $0 < C_p^* < \Pi$.
3a (purple)	Full, $C_p^* = \Pi$.

Table 4.1: The management strategies when the RM is not available.

First, we analyse how different risk attitudes influence the optimal level of investment in precautionary management (C_p^*) under two settings: (i) when the time of pest arrival is known with certainty (no variance), and (ii) when there is uncertainty in the arrival time (variance = 0.0001 and 0.01). Secondly, we will study the sensitivity analysis to degree of risk aversion in the precautionary management strategies. The analysis is used to determine how changes in risk aversion affect investment decisions based on changes in the level of risk aversion. It involves varying the value of λ, which represents the investor's level of risk aversion, and examining how the results of the analysis change as a result. This examines the relationship between the three zones shown in Table 4.1 and illustrates how risk affects the choice of disease control. A management strategy is not used in the black zone ($1a$), as the pathogen spreads slowly and has no impact on the forest. The pathogen is partially controlled in the pink zone ($2a$), whereas it is fully controlled in the purple zone ($3a$). As λ increases or decreases, the optimal level of investment in PM changes depending on both the likelihood of pest arrival and the speed of subsequent spread.

4.3.3 Sensitivity analysis to the degree of risk aversion when variance =0

In Figure 4.2, the six plots illustrate what happens to the precautionary management strategy when the reactionary strategy is not available and the disutility function is introduced

with zero variance. The risk preference parameter, λ is changed six times ($\lambda = -0.01, \lambda = -0.001, \lambda = -0.0001, \lambda = 0.0001, \lambda = 0.001,$ and$\lambda = 0.005$) for the same variance value. Figure 4.2(a) shows the control strategy when λ equals -0.01 corresponding to relatively high risk aversion. Similarly, 4.2(b) shows the same colours as 4.2(a) with a slight reduction in the control strategy against damage. Figure 4.2(b) shows the results when λ is -0.001, demonstrating a high control measure. Based on Figure 4.2(c), the outcome shows that zone $2a$ and $3a$ decrease as λ increase while Figure 4.2(d), (e) and (f) shows a similar outcome. The results show the appearance of smaller purple (zone $3a$) and pink (zone $2a$) regions, and more of the black (zone $1a$) region, as explained in Table 4.1. As shown in Figures 4.2(d)(e) and (f), zone $1a$ (do nothing) increases as the risk preference parameter increases, while zone $2a$ and zone $3a$ decrease.

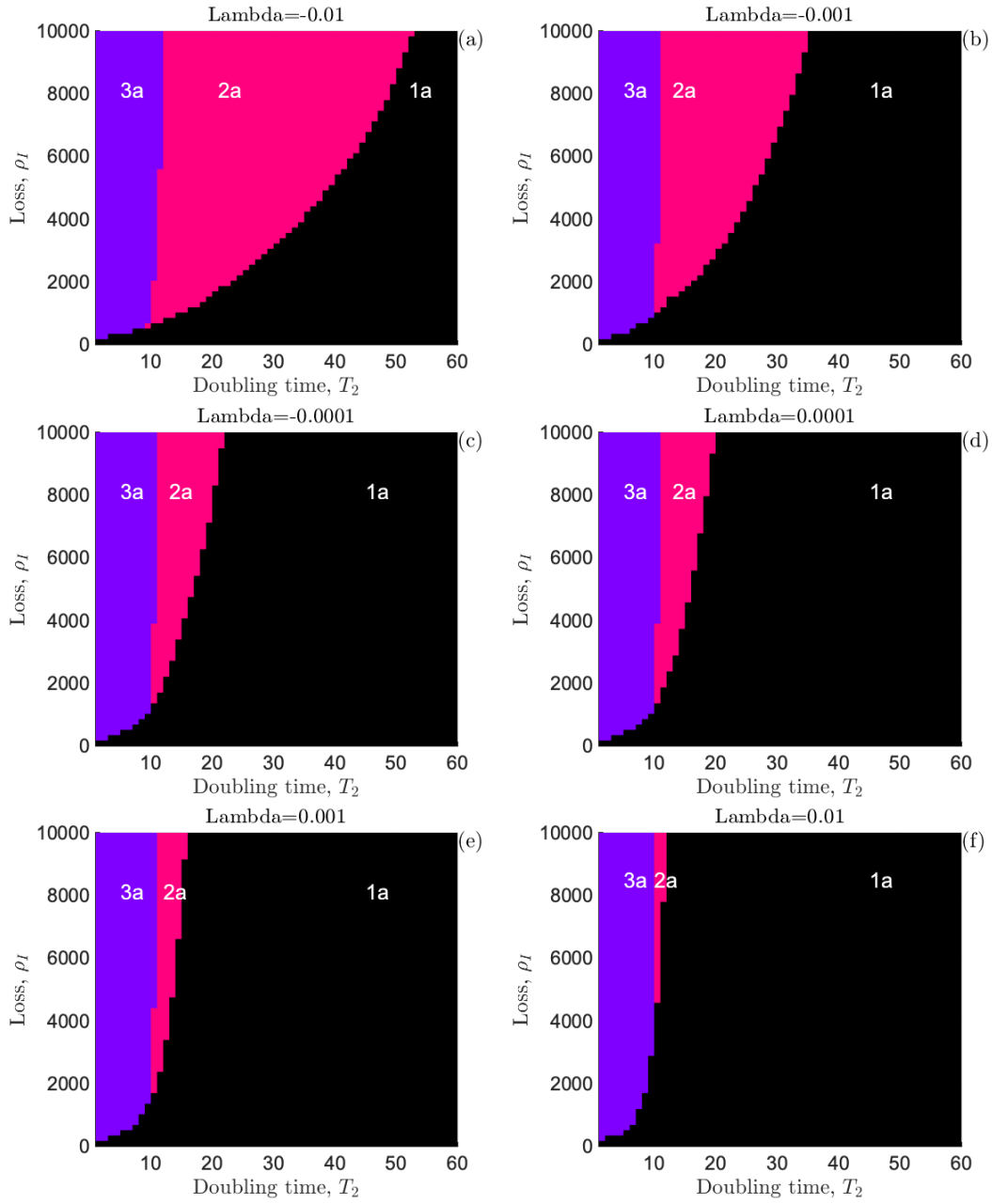

Figure 4.2: Sensitivity analysis to λ when variance =0. The effect of exponential disutility on the optimal management strategy. The colours describes the optimal strategy as shown in table 4.1. The parameters $\delta_M = 0$ and variance =0, while other parameter values are at their baseline values in Table 2.1.

Plots with negative values of λ indicate risk aversion, while plots with positive values of λ indicate risk seeking. Due to the rapid spread of the pathogen, the first two graphs, Figure

4.2 (a) and (b), show regions in which the highest level of investment in the precautionary management strategy is optimal. When $\lambda = -0.0001$ and 0.0001, the result shows neutrality of risk between risk aversion and risk seeking. Figures 4.2 (c) and (d) represent the region with a partial control strategy due to the slow spread of the pathogens, while Figures 4.2 (e) and (f) represent more of the slow arrival of the pathogens. Thus, the shift from high risk aversion to risk neutrality and risk seeking results in a strategy of no control (zone $1a$) becoming dominant.

Zone $1a$ (do nothing) starts on the left side of the doubling time and extends to the right of 20% of doubling time, in Figure 4.2(a)(b). When λ, is negative and the variance is zero, the disease spreads quickly and causes severe damage. As shown in Figure 4.2(a), the total loss caused by the disease is high, so it is essential to implement the PM strategy. However, Figure 4.2(f) shows that the total loss caused by the disease is smaller than the cost of deploying the PM regardless of how fast the disease is transmitted. Furthermore, the region where it is optimal to deploy partial PM (zone $2a$) gradually disappears as λ increases. According to Figure 4.2, the greater the investment, the more likely the pathogen will not arrive and spread. Therefore, the exciting fact about these results relies on the losses due to the presents of pathogens.

The damages from plant infections, as shown in Figure 4.2(d), (e) and (f) reduces as the doubling time move from left to right. Figure 4.2(a) and (b) present a high control strategy because people are more likely to control in regions with lower risk values. The result shows disease progress, the infected plants move from left to right during the doubling time, but the same infected plants reflect the high cost of control as the loss moves from low to high. However, as the damage increases, it can easily be identified because the disutility magnifies the losses.

4.3.4 Sensitivity analysis to the risk preference parameter when variance =0.0001

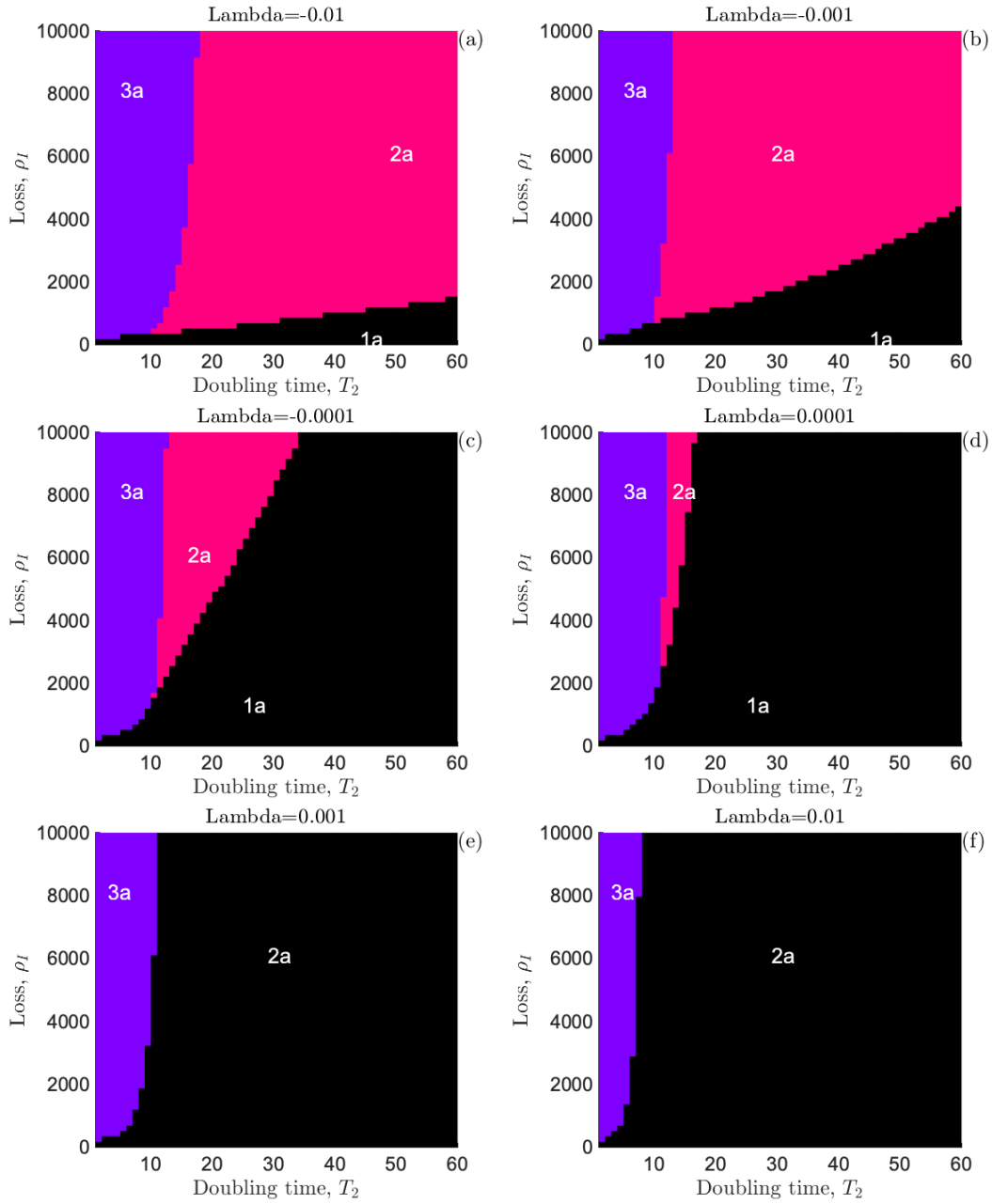

Figure 4.3: Sensitivity analysis to λ, when variance =0.0001. The effect of exponential disutility on the optimal management strategy with random P from a log-normal distribution. The colours describes the optimal strategy as shown in table 4.1. The parameters $\delta_M = 0$ and variance =0.00001, while other parameter values are at their baseline values in Table 2.1.

In Figure 4.3, the six plots illustrate what happens to the precautionary management strategy when the reactionary strategy is unavailable, and the disutility function is introduced with variance = 0.0001. The value of λ is changed six times ($\lambda = -0.01, \lambda = -0.001, \lambda = -0.0001, \lambda = 0.0001, \lambda = 0.001,$ and$\lambda = 0.005$) for the same variance value. The λ is varied over six values ranging from negative (risk-averse) to positive (risk-seeking) to examine how different risk attitudes affect the optimal level of precautionary management under a fixed level of uncertainty of arrival time. The disutility function is used in this analysis to capture the decision-maker's risk attitude with respect to the total cost, defined as the sum of disease-related damages and the cost of precautionary management. When $\lambda = -0.01$ as shown in figure 4.3(a) the black zone with no management strategy will disappear as variance is increased. The pink region with partial control strategy increases in figure 4.3(a), and the purple region also increases, which means that the result shows a high management strategy at a low $\lambda = -0.01$. Therefore, more attention will be paid to the event of high cost.

The result in Figure 4.3(a) is shown in three colours, the black region (zone $1a$) means do nothing, the pink region (zone $2a$) means partial intervention, and the purple region (zone $3a$) means full control. Figure 4.3(a) shows the control strategy when λ equals -0.01, and Figure 4.3(b) when λ is -0.001, demonstrating a high control measure. Based on Figure 4.3(c), we notice that zone $2a$ and zone $3a$ decrease as λ is increased from $\lambda = -0.01 \longrightarrow \lambda = -0.001 \longrightarrow \lambda = -0.0001$. The results in figure 4.3(a), (b) and (c) show an increase in total loss with the disutility function. In Figure 4.3(d), (e) and (f), I have a similar outcome, the results show the appearance of lesser Purple (zone $3a$), pink (zone $2a$) region, and more of the black (zone $1a$) region as explained in Table 4.1. As shown in Figure 4.2(d)(e) and (f), zone $1a$ (do nothing) increases as the risk preference parameter increases, zone $2a$ disappears while zone $3a$ decrease.

Similarly, zone $1a$ starts from the left side of the doubling time and extends to the right of 5% of doubling time, in Figure 4.2(a)(b). When λ is negative and the variance = 0.0001, the disease spreads quickly and causes severe damage. However, the spread of disease reduces when λ is positive. As shown in Figure 4.3(a), the total loss caused by the disease is high, so it

is vital to implement the PM strategy. However, Figure 4.3(f) shows that the total loss caused by the disease is smaller than the cost of deploying the PM regardless of how fast the disease transmits within the forest. Furthermore, the region where it is optimal to deploy partial PM gradually disappears as the λ increases.

Comparing results in Figure 4.2 for the deterministic model with the results in Figure 4.3 with low but finite variance shows that the stochastic model is associated with increased control. The region of parameters ρ_I and T_2 for which it is optimal to do nothing is smaller for the stochastic model, while the zones $2a$ and $3a$ expand. This effect is particularly strong for highly risk-averse attitude ($\lambda < 0$), compare Figure 4.2 (a) (b) (c) with Figure 4.3 (a) (b) (c). This can be associated with more risk-averse decision maker paying more attention to rare but large losses, which can occur in the stochastic case.

4.3.5 Sensitivity analysis to the risk preference parameter when variance =0.1

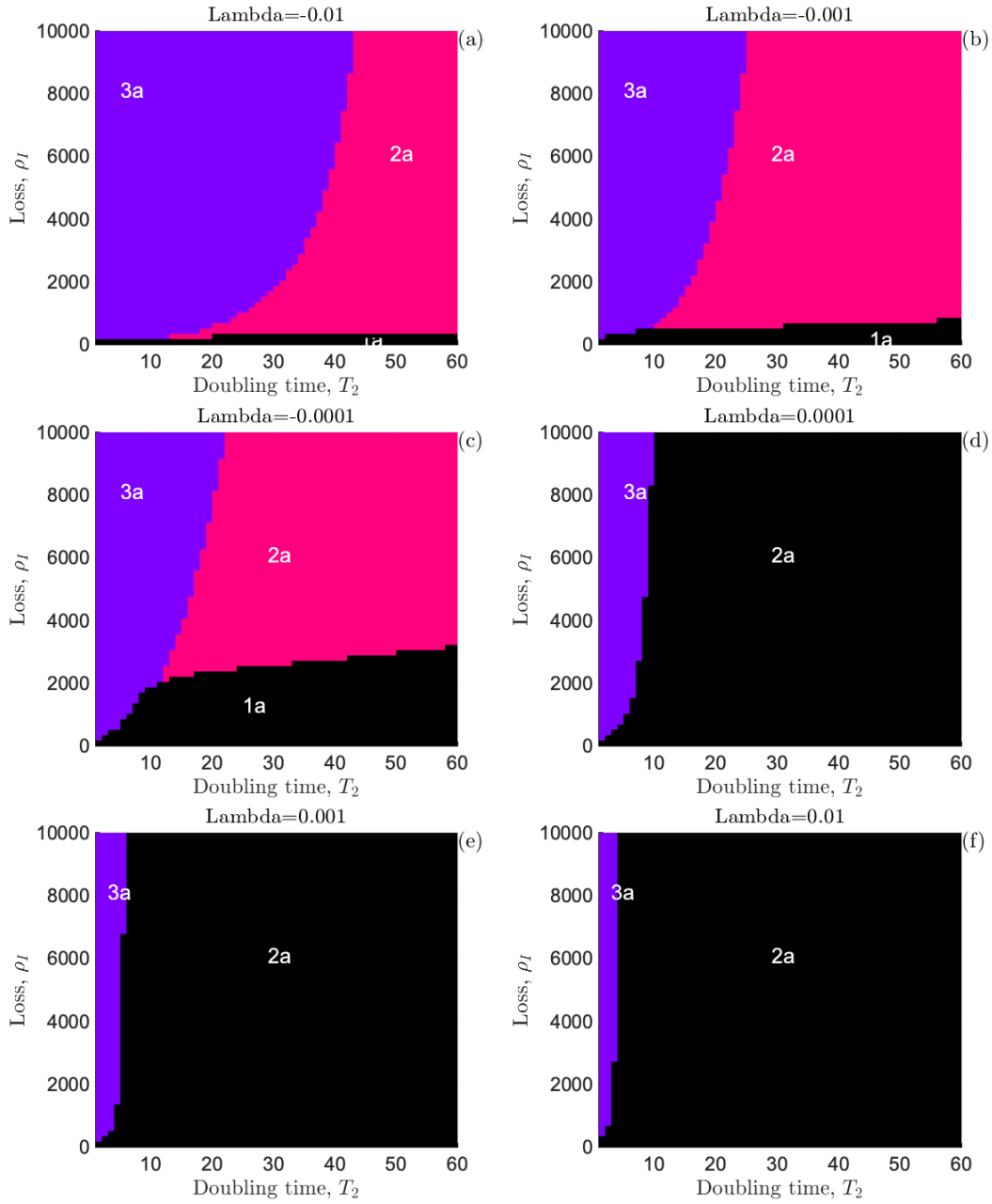

Figure 4.4: Sensitivity analysis to the λ when variance = 0.1. *The effect of exponential disutility on the optimal management strategy with random P from a log-normal distribution. The colours describes the optimal strategy as shown in table 4.1. The parameters $\delta_M = 0$ and variance* = 0.1, *while other parameter values are at their baseline values in Table 2.1.*

In Figure 4.2, the plots illustrate what happens to the precautionary management strategy and the disutility function is introduced with variance = 0.1. The risk preference parameter, λ is changed in the six plots ($\lambda = -0.01, \lambda = -0.001, \lambda = -0.0001, \lambda = 0.0001, \lambda = 0.001,$ and$\lambda = 0.005$) for the same variance value. When $\lambda = -0.1$ as shown in Figure 4.4(a) the black zone without a management strategy will disappear as the variance increases. Therefore, more attention will be paid to the event of high cost. The negative value of risk when $\lambda = -0.01$ and $\lambda = -0.001$ shown in Figure 4.4 (a) and (b) represent the risk attitude, which means more attention to be paid to the cost of control.

The effect of the expansion of zones $2a$ and $3a$ at the cost of zone $1a$ is even more visible when the variance increases to 0.1, Figure 4.4. Similarly to Figures 4.2 and 4.3, this is most visible when λ is negative, which means risk aversion. The parameter region defined by ρ_I and T_2 where no action is optimal becomes smaller, while zones $2a$ and $3a$ expand. This effect is particularly pronounced for a highly risk-averse attitude ($\lambda < 0$), as illustrated by the comparison between Figure 4.2 (a) (b) (c) and Figure 4.3 (a) (b) (c). This behaviour can be attributed to more risk-averse decision maker giving greater consideration to infrequent but significant losses, which are more likely in the stochastic scenario.

4.3.6 Sensitivity analysis to risk preference parameter in the precautionary management strategies

This section dives deeper into how the λ and variance affect the choice of optimal strategy. The analysis shows how the optimal management decisions change when risk attitudes are incorporated into the stochastic model. In this case, the decision-making process reflects not only the probability of different outcomes but also the manager's preference toward risk (i.e. whether they are risk-averse, risk-neutral or risk-seeking). Here, the land manager assigns a value or disutility to each possible outcome, and the goal is to maximise the expected disutility rather than the expected value. The stochastic model significantly impacts management strategies because it means that decisions will be based on more than just the average of

different outcomes.

The computer simulation is used to examine each strategy to describe the result for better understanding. These results will help better understand what is happening in the strategies and why it is happening. In this way, we will clearly understand why we have different strategies with different values of λ.

The simulation results show how the optimal level of precautionary investment changes once the decision-maker's attitude toward the risk of infection losses is incorporated. In this context, the variance refers to the uncertainty in the timing of pest arrival, and a higher variance implies a greater likelihood of very early infection and, therefore, larger potential damages. The parameter λ reflects the risk attitude of the manager (not the level of risk in the system): more negative values of λ indicate stronger risk aversion, and lead to higher precautionary investment, because the manager is willing to incur higher costs to avoid the possibility of extreme losses. In contrast, less negative or positive values of λ correspond to risk-seeking behaviour, where the manager is prepared to accept a greater chance of large infection losses in order to keep precautionary costs low. As a result, even when the variance (i.e. uncertainty in arrival time) is high, risk-seeking managers invest less in prevention, whereas risk-averse managers invest more to reduce the tail risk of severe outbreaks. A higher value of λ implies a higher level of risk aversion, while a lower value of λ implies a lower level of risk aversion. By performing a sensitivity analysis to λ for a given variance, investors can examine the impact of changes in λ on their investment decisions. This allows them to assess the trade-off between risk and return and to make more informed investment decisions.

disutility function plot, when lambda=-0.005

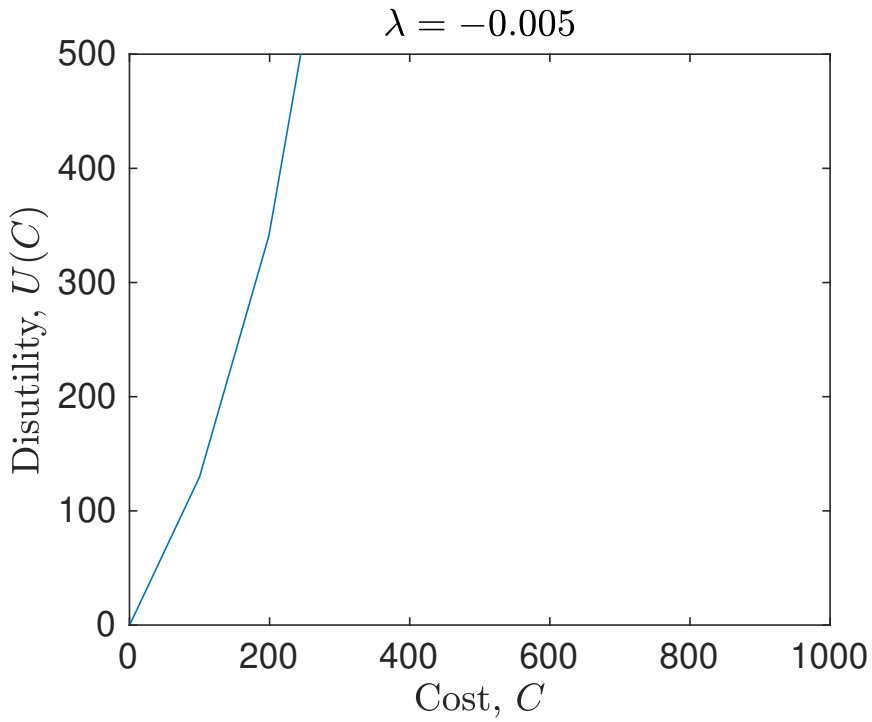

Figure 4.5: Illustration of the disutility function, $U(C)$ and total cost, C. The plot represent the relationships between non-linear disutility function and cost. This diagram expands upon the established concepts of exponential disutility functions when the risk preference parameter is $\lambda = -0.005$

As described in Figure 4.5, the disutility function graph provides insight into the preferences of the decision maker and the level of risk aversion. The non-linear disutility function for $\lambda = -0.005$ and $\sigma = 0.0001$ is shown in Figure 4.5. A disutility function plot is a mathematical representation of a land manager's preferences that curves upward from left to right. In this case, the disutility ranges from 0 to 1000 on the y-axis. Cost is represented on the x-axis, which ranges from 0 to 1000. In this case, $\lambda - 0.005$, which means the investors are risk-averse and prefer a low level of risk. The plot shows that as cost increases, disutility increases even more. As the decision maker minimises the disutility here rather than the cost (as in the previous chapter), strategies with relatively large cost are penalised. Investors become less willing to take on additional risk as the cost increases. Convex non-linear disutility functions are shaped

by the λ, represented by -0.005. The curve becomes less convex as the λ increases. In other words, as the investor becomes more risk-averse, the disutility increases less rapidly.

However, the non-linear disutility function exhibits a convex curve, meaning that the disutility increases as the cost increases. As risk degree becomes more negative, the curve becomes less convex, and the disutility increases less rapidly as an individual becomes risk-averse. The result shows that we do not invest when λ has a lower value. In the context of Figure 4.5, the results show that risk-averse managers tend to invest more in precautionary management in order to avoid the possibility of very large losses, even when the expected (average) reduction in loss is relatively small. In contrast, risk-seeking managers are willing to invest less or even nothing in prevention, as they are prepared to accept a higher probability of a large loss in the hope that the invasion arrives late or causes limited damage. The key distinguishing feature of risk aversion is that the disutility curve is concave at high levels of damages. The relationship between cost and disutility is asymmetric: reductions in cost produce relatively small changes in disutility (i.e. they feel "normal"), whereas increases in cost lead to disproportionately larger increases in disutility.

Sensitivity analysis to risk aversion and disutility, when variance$= 0.0001$ **and lambda**$= -0.005$

This section illustrates the management strategies in which different control strategies were shown with changes in the doubling time, T_2 and loss, ρ_I and how these strategies change with and without the disutility function. The analyses presented side-by-side are the cost plot and the heat-map plots without the disutility function and with the disutility function (see Chapter 3). The results help to interpret why different optimal strategies were obtained for different loss values (ρ_I) and doubling time (T_2) in the heat-map plot. Additionally, more explanation about why different results were obtained is introduced if the model includes both variance and disutility.

These plots explain why different management strategies were used in different places of the T_2 and ρ_I plane and why the results differ if variance and disutility were included. Some

points were chosen from each graph to help explain what happens with the control strategies. Essentially, picking up a combination of T_2 and ρ_I to show how the total cost, which is the combination of the cost of infection and cost of control, changes with changing C_p. After that, the disutility is brought in to see how disutility changes the disease progress curve and observe the minimum of the total cost. The $25th$ value of ρ_I which is 6600 £ ha^{-1} year^{-1}, are picked and it will correspond to different values of T_2 which are 8 years, 12 years and 35 years. The loss ρ_I is fixed to see what happens as doubling time, T_2 changes.

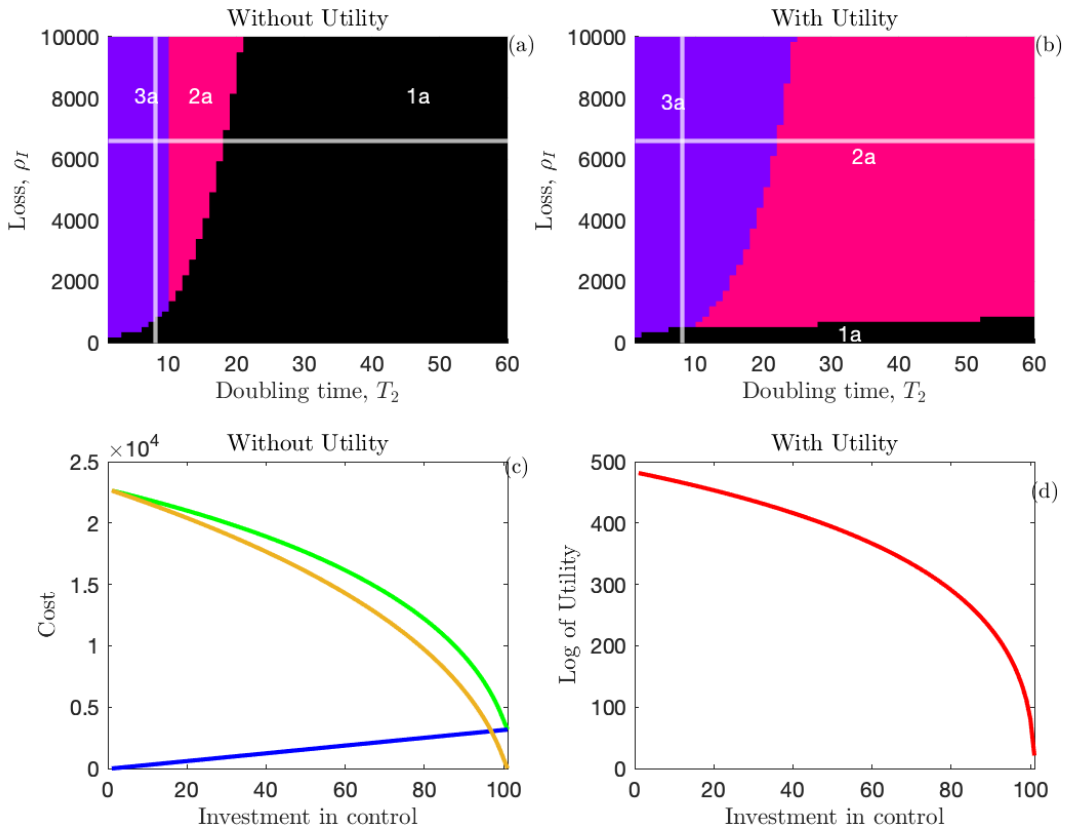

Figure 4.6: The figure illustrates the management strategies when loss, ρ_I value, is 6600 £ ha^{-1} year^{-1}, and doubling time, T_2 value is 8 years with the Variance=0.0001 *and* λ =−0.005. *The heat-map plot shown in Figure 4.6(a) shows the management strategies with variance* =0.0001 *and no disutility. The heat-map plot in Figure 4.6(b) illustrates the management strategies with disutility function. The cost plot is shown in Figure 4.6(c), with the cost of control in blue, the cost of infection in yellow, and the total cost in green. A cost plot with log of disutility function can be seen in Figure 4.6(d). Other parameters values are at their baseline values (See Table 2.1).*

The analysis provides valuable information on the cost and effectiveness of different management strategies to control the spread of a disease and how these strategies are affected by varying levels of risk aversion and without disutility. The heat-map plot presented in Figure 4.6(a) shows the management strategies with variance=0.0001 and without disutility, while Figures 4.6(b) illustrates the result with variance = 0.0001 and the disutility function. When the variance is 0.0001 and λ, is -0.005, the result shows how the optimal strategy depends on the doubling time and loss combination.

On the x-axis in Figure 4.6(a)(b), we have the doubling time, which ranges from 0 to 60 years; for Figures 4.6(d) and (e), we pick 8 years. In Figure 4.6(a)(b), the y-axis represents the potential losses that could occur under different management strategies, ranging from 0 to 10000, we pick ρ_I value 6600 £ ha^{-1} year $^{-1}$. These values were chosen so that the optimal strategy is $3a$ (full control) for both the case without and the case with disutility. The variance value of 0.0001 indicates that the model assumes high accuracy during the simulation. This value represents the uncertainty in the assumptions used in the model. The analysis provides valuable information on the cost and effectiveness of different precautionary management strategies for controlling disease spread, and shows how the optimal level of investment changes under different levels of risk aversion.

The white cross in Figure 4.6(a) represents the specific combination of loss (ρ_I) and doubling time (T_2) chosen for analysis. The horizontal and vertical white lines in Figure 4.6(a) represent the range of values for which optimal management strategies are considered. In this case, the white cross is located in the purple strategy, indicating that investing all resources (purple) is the most effective option for controlling the spread of the disease, given the selected values of loss and doubling time (ρ_I= 6600 £ ha^{-1} year $^{-1}$ and T_2=8 years). The level of uncertainty in the model assumptions is reflected in the degree of accuracy assumed for the simulation (variance= 0.0001). The plot in the upper right of Figure 4.6 (b) shows the three management strategies (no intervention in black, partial intervention in pink and full intervention in purple) with the inclusion of the disutility function and variance = 0.0001. Including the disutility function allows us to account for the decision maker's attitude towards

risk, and therefore to evaluate management strategies not only in terms of their expected losses, but also in terms of how they reduce the likelihood of very large losses.

Similarly to the previous plot in Figure 4.6(a), the white cross in Figure 4.6(b) represents the specific combination of loss (ρ_I) and doubling time (T_2) chosen for this analysis. In this case, the white cross is also located in the purple strategy, indicating that investing all resources (purple) will be the most effective option to control the spread of the disease when ρ_I value is 6600 £ ha^{-1} year $^{-1}$ and 8 years. In Figure 4.6(b), with the inclusion of the disutility function, the optimal strategy is still the purple region (zone $3a$), which represents the full intervention. This indicates that investing all resources in controlling the spread of the disease is the most effective option for minimising both the cost of infection and the cost of control. The inclusion of the disutility function enables a more comprehensive assessment of the risk associated with each management strategy, and confirms that the purple region still represents the optimal level of precautionary investment for controlling disease spread under risk-averse behaviour. Disutility refers to the perceived value of a particular outcome, and its absence indicates that the model is focused solely on minimizing losses. The plot provides a decision-making model that helps identify the most effective management strategies for minimizing losses under specific conditions of loss value and doubling time. By selecting a specific combination of loss and doubling time and analyzing the associated costs of control and infection, the plot provides insights into the optimal management strategies for controlling the spread of a disease in a given context.

The plot shown in Figure 4.6 (c) shows the relationship between the total cost, the cost of infection, and the cost of control as the investment in control changes. The plot provides a baseline for understanding the cost without disutility before introducing disutility in Figure 4.6(d). The green curve's shape and trajectory depend on various factors, such as the cost of control, the cost of infection, the investment in control, and the loss associated with infection (ρ_I) and the doubling time (T_2). The cost of control is represented by the blue line, the cost of infection is represented by the yellow curve, while the green curve represents the total cost. The yellow curve in Figure 4.6(c) represents the cost of infection, which is the cost

associated with the negative consequences of an infection spreading. As the investments in control increase, the cost of infection decreases since more resources can be allocated towards controlling the spread of the infection (Precautionary Measure).

The total cost has a minimum either on the left or on the right or in the middle line shown by the green line. This implies that there are multiple ways to minimize the total cost, depending on the value of the investment in control. The total cost that corresponds to the minimum of the green line in Figure 4.6(c) is approximately 0.55. As at this level of control, pathogen spread is completely arrested, there is no cost (loss) associated with infection. Thus, the total cost (approximately 0.55) is associated fully with control, and control should be applied at maximum (zone $3a$). This means that investing around 0.55 of the available resources into control measures would result in the minimum total cost. This value represents the optimal investment in control measures to minimize the total cost of infection and control. Therefore, if land manager want to minimize the total cost while also effectively controlling the spread of the disease, they should invest maximum possible of C_p, resulting in the cost equal 0.55 (arbitrary units). The optimal strategy in Figure 4.6(a) is the purple region, indicating full intervention, for the selected combination of loss and doubling time. It is important to note that the optimal strategy in Figure 4.6(a) is based on a specific combination of loss and doubling time, whereas the minimum in Figure 4.6(c) is the minimum of the total cost over a range of investment levels. The plot provides insights into the relationship between the cost of control, cost of infection, and total cost and how they change with varying investments and chosen values of loss and doubling time.

Now, moving on to Figure 4.6(d), the log of the disutility function illustrates how risk aversion is incorporated into the decision-making process, by showing how larger losses are increasingly penalised relative to smaller ones. However, since disutility spans a wide range of values, it can be difficult to display the data on a linear scale. The reason for this is that as the values of the variable increase or decrease, the differences between the values become more difficult to discern visually. This can lead to difficulties in interpreting the data and making accurate comparisons. Therefore, a logarithmic scale is used in order to better display the

wide range of values for the variable "disutility" in a clear and meaningful way. The red curve represents the log of disutility, including both the cost of infection and control, along with the level of risk involved. The optimal cost is where the red curve reaches its minimum point, which is the most efficient and cost-effective decision. The plot demonstrates the importance of balancing the direct cost of a decision with the level of risk involved. By finding the optimal cost that minimizes the total cost, land manager's can make efficient and effective choices that take into account both the potential gains and losses associated with a decision.

The results presented in Figure 4.6 show how including a disutility function affects the disease progression curve and the minimum of the total cost. It also shows how the inclusion of a disutility function can affect decision-making around disease control strategies. Overall, the plot provides a visual representation of the total costs and benefits of different management strategies and shows how the inclusion of a disutility function can affect decision-making around disease control strategies. By finding the optimal cost that minimizes the total cost, we can make efficient and effective choices that take into account both the potential gains and losses associated with a decision. In a cost-effectiveness analysis, decision maker are typically interested in identifying the best management strategy that provides the most value for the resources invested. By finding the point on the red curve where the total cost is minimized, land manager can make the most efficient and cost-effective choice. This means that they can achieve the desired level of effectiveness while minimizing the cost of achieving that effectiveness.

The minimum in Figure 4.6(d) is the lowest point on the red curve, which represents the optimal cost that minimizes the total cost while taking into account the level of risk involved in the decision-making process. This point corresponds to the most efficient and cost-effective decision based on the research objective. The red curve in Figure 4.6(d) is different from the green line in Figure 4.6(c) but it points to the same solution, i.e. maximum investment in control. The green line in Figure 4.6(c) represents the total cost without considering the level of risk involved in the decision-making process, while the red curve in Figure 4.6(d) represents the total cost incorporating the log of a disutility function that accounts for the level of risk

involved in the decision-making process. As the investment in control increases, the cost of control also increases, which is expected since more resources will be required to implement and maintain control measures. The plot shows that as the investment in control changes, the costs of control and infection also change, as shown in Chapter 2. It is important to note that the investment is full (100 %) in Figure 4.6(c) but 100% in Figure 4.6(d).

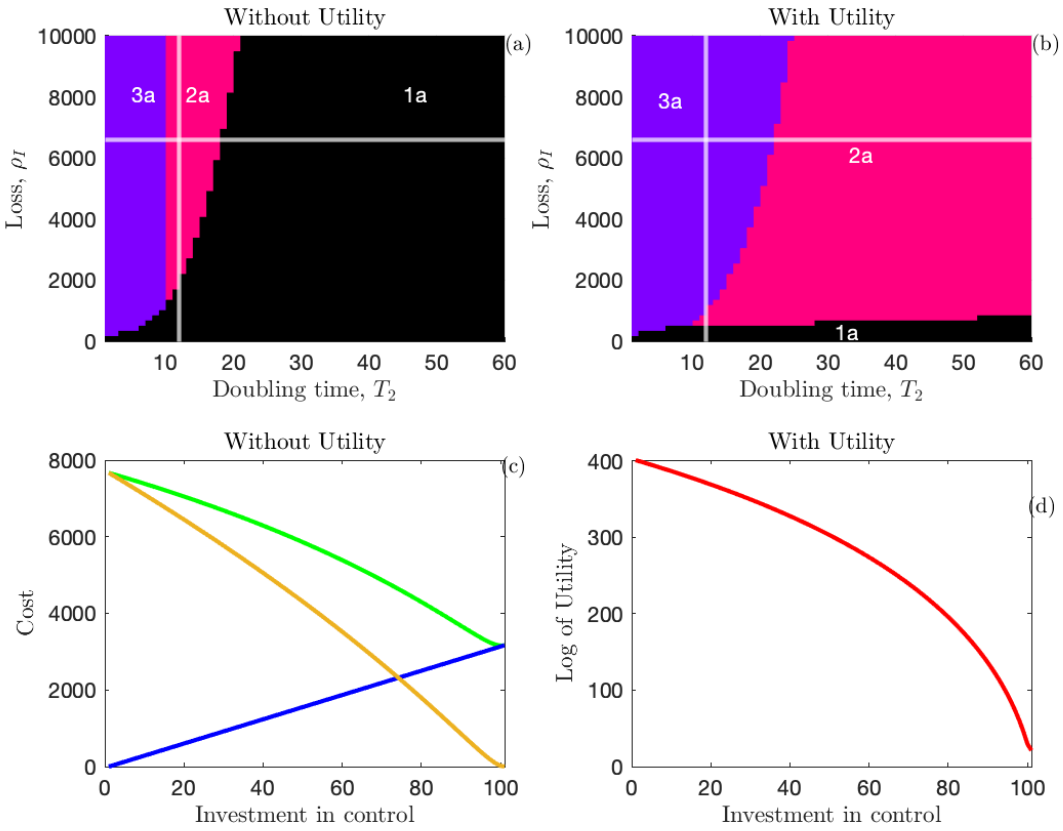

Figure 4.7: The figure illustrates the management strategies when loss, ρ value is 6600 £ ha^{-1} $year^{-1}$, and doubling time, T_2 = 12 years with the Variance=0.0001 *and* λ =−0.005. *The heat-map plot shown in Figure 4.7(a) shows the management strategies with variance*=0.0001 *and no disutility. The heat-map plot in Figure 4.7(b) illustrates the management strategies with disutility function. The cost plot is shown in Figure 4.7(c), with the cost of control in blue, the cost of infection in yellow, and the total cost in green. A cost plot with log of disutility function can be seen in Figure 4.7(d). Other parameters values are at their baseline values (See Table 2.1).*

The cost plot in Figure 4.7(c) shows the relationship between the cost of control, cost

of infection, and total cost. The blue line represents the cost of control, the yellow curve represents the cost of infection, and the green curve represents the total cost. The shape and trajectory of the yellow curve depend on the loss associated with infection (ρ_I) and the doubling time (T_2). The blue line in the plot (Figure 4.7(c)) represents the cost of control, which is the cost associated with the implementation of the precautionary control measure to reduce the spread of infection. The minimum of the green line represents the optimal balance between the cost of control and the cost of infection, where the total cost is minimised.

As the investment in control increases, the cost of control increases, which is expected since more resources will be required to implement and maintain control measures. On the other hand, the cost of infection decreases as the investment in control increases since more resources can be allocated towards controlling the spread of the infection. The green line, which represents the total cost, has a minimum point where the cost of control and the cost of infection are balanced. The minimum point on the green curve represents the optimal investment in control measures that minimises the total cost of infection and control. It is important to note that the investment is partial (95 %) in Figure 4.7(c) but 100% in Figure 4.7(d).

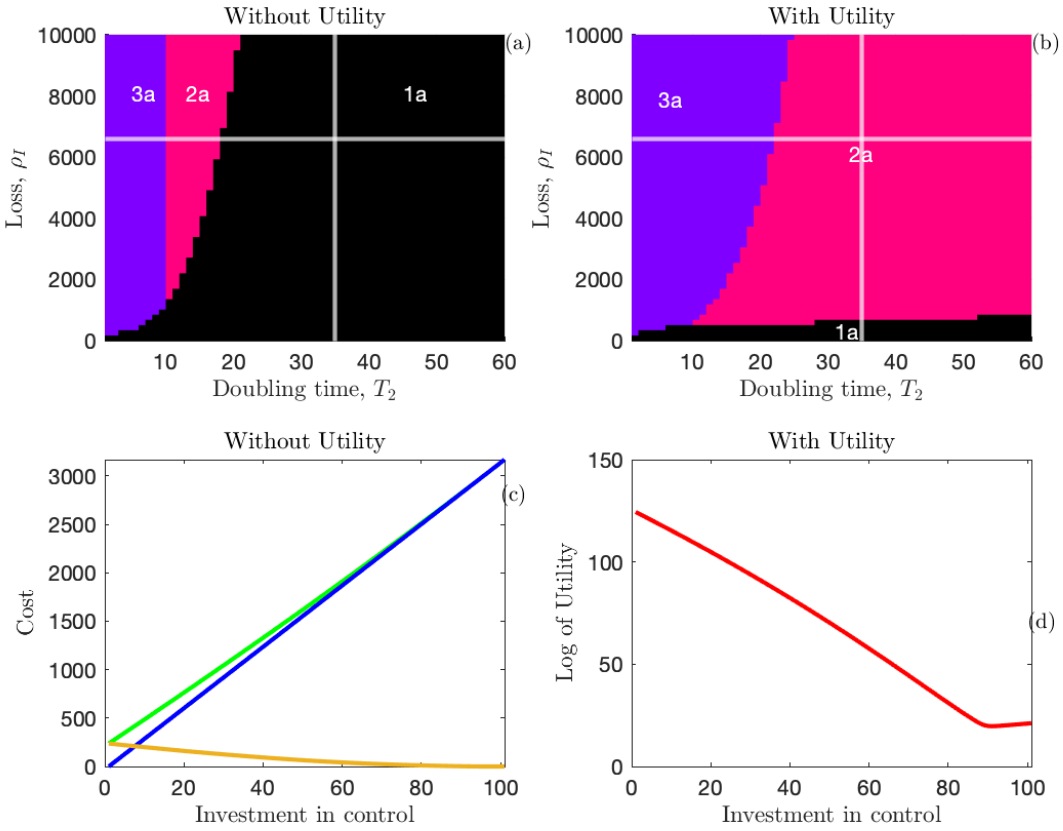

Figure 4.8: The figure illustrates the management strategies when loss, ρ value is 6600 £ ha^{-1} $year^{-1}$, and doubling time, $T_2 = 35$ years with the Variance=0.0001 and $\lambda = -0.005$. The heat-map plot shown in Figure 4.8(a) shows the management strategies with variance=0.0001 and no disutility. The heat-map plot in Figure 4.8(b) illustrates the management strategies with disutility function. The cost plot is shown in Figure 4.8(c), with the cost of control in blue, the cost of infection in yellow, and the total cost in green. A cost plot with log of disutility function can be seen in Figure 4.8(d). Other parameters values are at their baseline values (See Table 2.1).

The plots in Figure 4.8 show the management strategies and the cost analysis for a double time of 35 years. The heat-map plot in Figure 4.8(a) illustrates the management strategies without disutility and with variance=0.0001. The heat-map plot in Figure 4.8(b) shows the management strategies with disutility and variance=0.0001. The cost plot without disutility is presented in Figure 4.8(c), while the cost plot with disutility is shown in Figure 4.8(d). The minimum in Figure 4.8(c) is without (zero) investment and 90% in Figure 4.8(d). This point

corresponds to the most efficient and cost-effective decision based on the research objective. The optimal management strategy changes as the doubling time increases from 8 years to 12 years and to 35 years. In general, as the doubling time increases, it becomes more difficult and expensive to control the spread of the disease. In Figure 4.6, as the doubling time increases from 8 years to 12 years and 35 years, the optimal management strategy shifts from the purple zone to the pink zone, indicating that a less intensive control strategy (i.e., investing fewer resources in control measures) may be more cost-effective.

disutility function plot, when lambda=-0.001

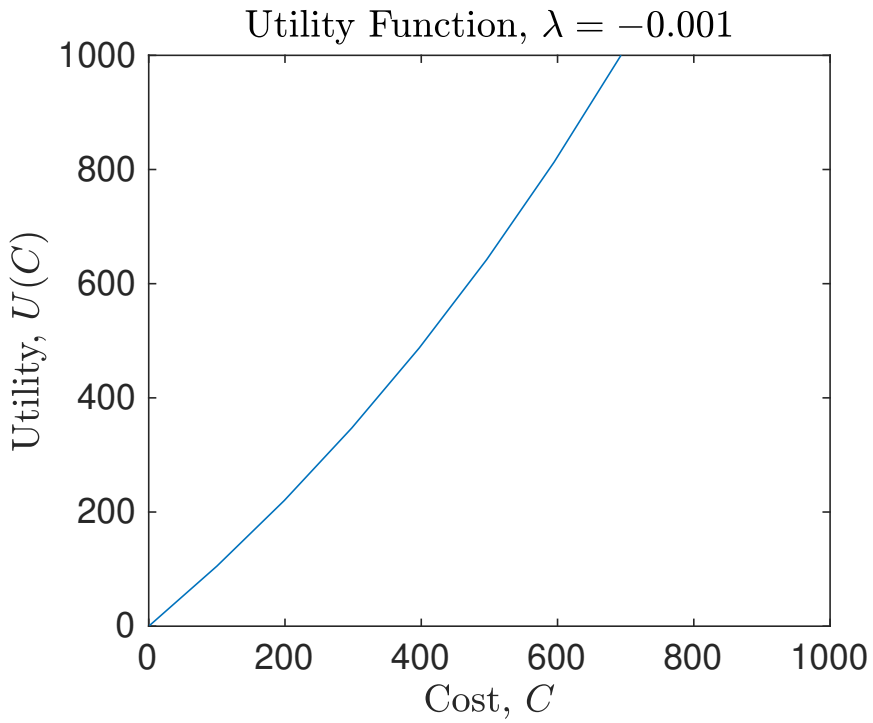

Figure 4.9: Illustration of the disutility function, $U(C)$ and cost, C. The plot represent the relationships between non-linear disutility function and total cost. This diagram expands upon the established concepts of exponential disutility functions when $\lambda = -0.001$.

The plot in Figure 4.9 represents the disutility function when the variance is set to 0.1 and the degree of risk aversion, λ, is -0.001. The plot shows that, as the cost on the x-axis increases, the disutility on the y-axis also increases even more. This reflects the idea that the land

manager derives less additional satisfaction or gain from each unit increase in cost. However, the degree of risk aversion is lower (-0.001) compared to Figure 4.5, indicating a slightly lower level of risk aversion. This suggests that the land manager is less sensitive to changes in cost and exhibits a smaller preference for lower costs. The variance value of 0.1 implies higher uncertainty or variability in the outcomes. It indicates that the decision maker is more tolerant of fluctuations in cost and is willing to accept a certain level of risk. The degree of risk aversion, represented by $\lambda = -0.001$, suggests a very slight risk aversion. The land-manager is somewhat sensitive to changes in cost and exhibits a small preference for lower costs.

Sensitivity analysis to risk aversion and disutility, when variance=0.1 and lambda=-0.001

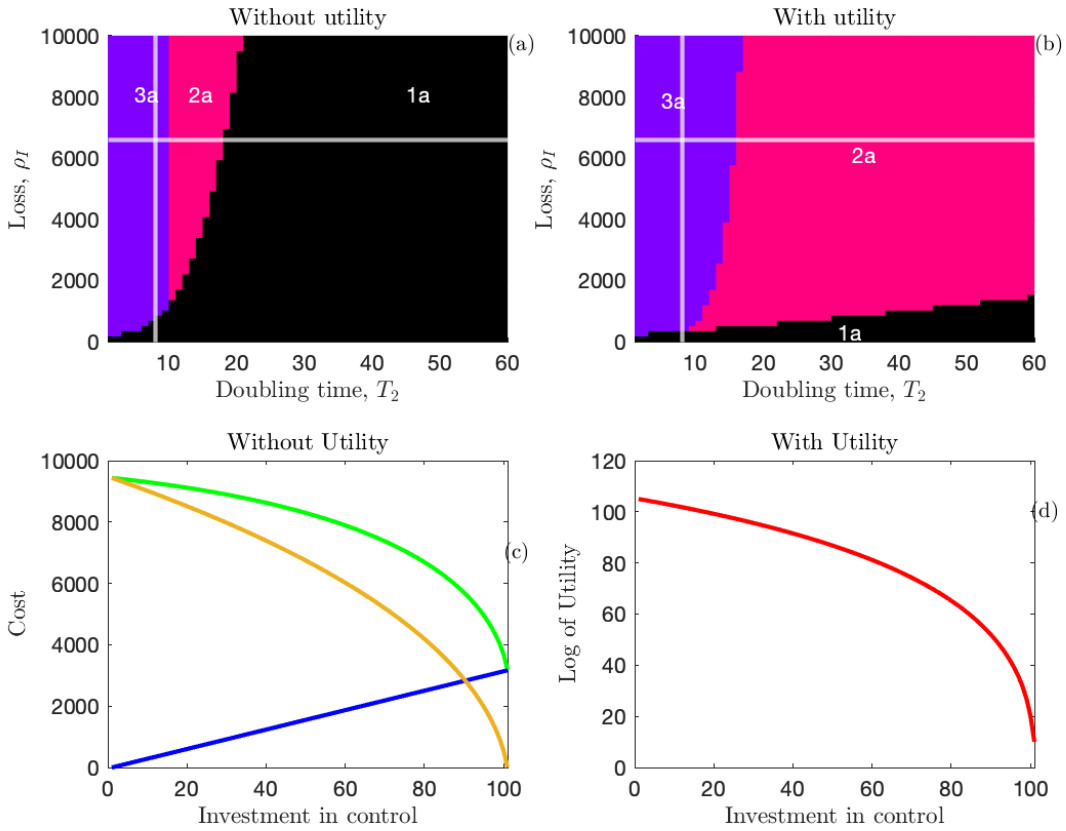

Figure 4.10: The figure illustrates the management strategies when loss, ρ_I value is 6600 £ ha^{-1} $year^{-1}$, and doubling time, T_2 value is 8 years with the Variance=0.1 and λ =−0.001. The heat-map plot shown in Figure 4.10(a) shows the management strategies without disutility function. The heat-map plot in Figure 4.10(b) illustrates the management strategies with disutility function. The cost plot is shown in Figure 4.10(c), with the cost of control in blue, the cost of infection in yellow, and the total cost in green. A cost plot with log of disutility function can be seen in Figure 4.10(d). Other parameters values are at their baseline values (See Table 2.1).

In Figure 4.10(a), we see the heat-map plot of the management strategies with variance=0.1 and without the disutility function, while Figure 4.10(b) shows the management strategies with the disutility function. The white cross in Figure 4.10(a) represents the specific combination of loss and doubling time chosen for analysis. The white cross is located on the purple (zone

3a) strategy, indicating that investing all of the resources (purple) is the most effective option for controlling the spread of the disease, given the selected values of loss and doubling time (ρ_I= 6600 £; ha^{-1}; year $^{-1}$ and T_2=8 years). Figure 4.10(c) shows the cost plot with the cost of control in blue, the cost of infection in yellow, and the total cost in green, without the disutility function. The green curve represents the total cost, which is the sum of the cost of control and the cost of infection. Figure 4.10(d) shows the cost plot with the log of the disutility function included. The red curve represents the log of the disutility function, including both the total cost of infection and control, along with the level of risk involved. The maximum investment point in both Figures 4.10(c) and (d) is 100%. Hence, since decision maker's want to minimize the total cost while also effectively controlling the spread of the disease, they should invest the maximum possible of C_p.

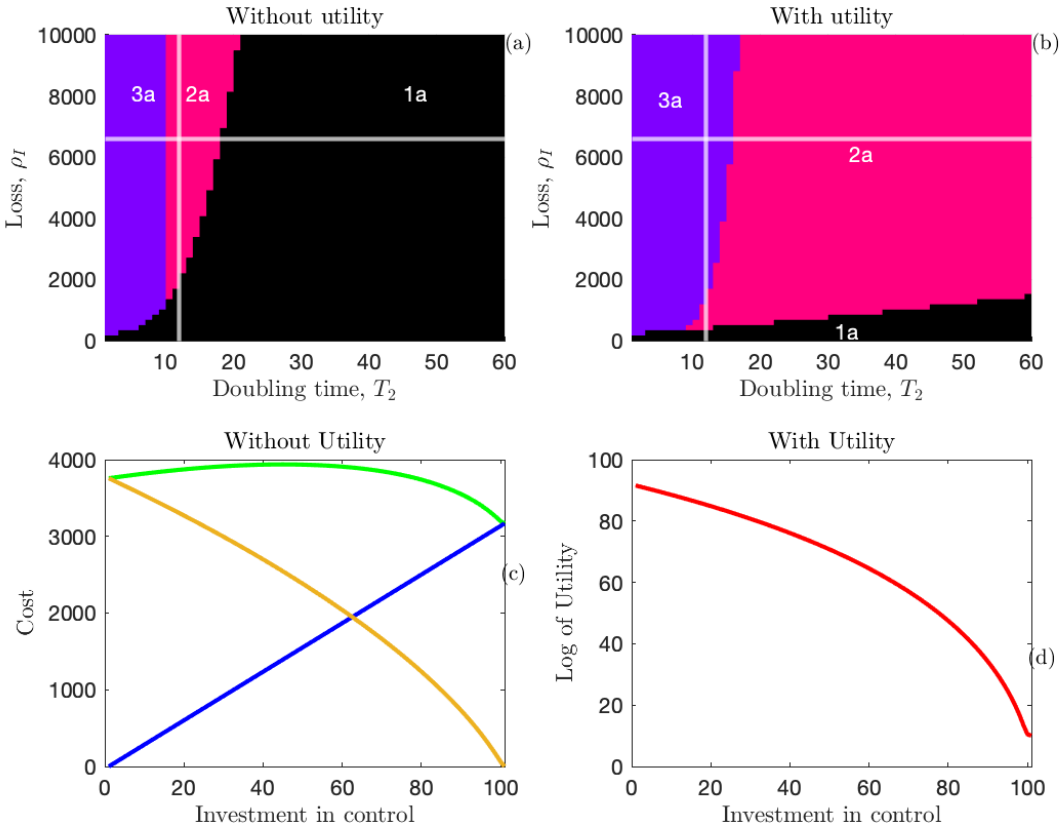

Figure 4.11: The figure illustrates the management strategies when loss, ρ_I value is 6600 £ ha^{-1} $year^{-1}$, and doubling time, T_2 value is 12 years with the Variance=0.1 and λ =−0.001. The heat-map plot shown in Figure 4.11(a) shows the management strategies with variance=0.1 and no disutility. The heat-map plot in Figure 4.11(b) illustrates the management strategies with disutility function. The cost plot is shown in Figure 4.11(c), with the cost of control in blue, the cost of infection in yellow, and the total cost in green. A cost plot with log of disutility function can be seen in Figure 4.11(d). Other parameters values are at their baseline values (See Table 2.1).

The heat-map plot in Figure 4.11(a) shows that the optimal management strategy is less sensitive to loss rate changes compared to doubling time changes. The key parameters used for the analysis include a loss rate of 6600 £; ha^{-1}; $year^{-1}$, a doubling time of 12 years, a variance of 0.1, and $\lambda = -0.001$ while other parameters values are at their baseline values (see Table 2.1). In Figure 4.11(a), the white cross in the graph represents the specific combination of the loss rate and the doubling time chosen for the analysis. In this case, the white cross

is located in the pink zone ($2a$) of the plot, indicating that investing some of the resources could be the most effective option to control the spread of the disease when the loss is 6600 £; ha^{-1}; $year^{-1}$ and doubling time of 12 years. This suggests that when deciding on the optimal management strategy, it may be more important to consider the doubling time of the disease than the loss rate, particularly in cases where the doubling time is large.

Figure 4.11(b) shows the heat-map plot of the optimal management strategies with variance=0.1 and the disutility function. The plot shows that the optimal management strategy is shifted towards investing all resources in control measures compared to Figure 4.11(a), indicating the importance of considering the potential gains and losses associated with different management strategies. Figure 4.11(c) shows the cost plot with the cost of control in blue, the cost of infection in yellow, and the total cost in green without considering the level of risk aversion and disutility. Figure 4.11(d) shows the cost plot with the log of the disutility function. In the plots in Figure 4.11(c) and (d), the investment is the maximum, which is 100%. The plot demonstrates the importance of balancing the direct cost of control and infection with the potential gains and losses associated with different management strategies, determined by the disutility function.

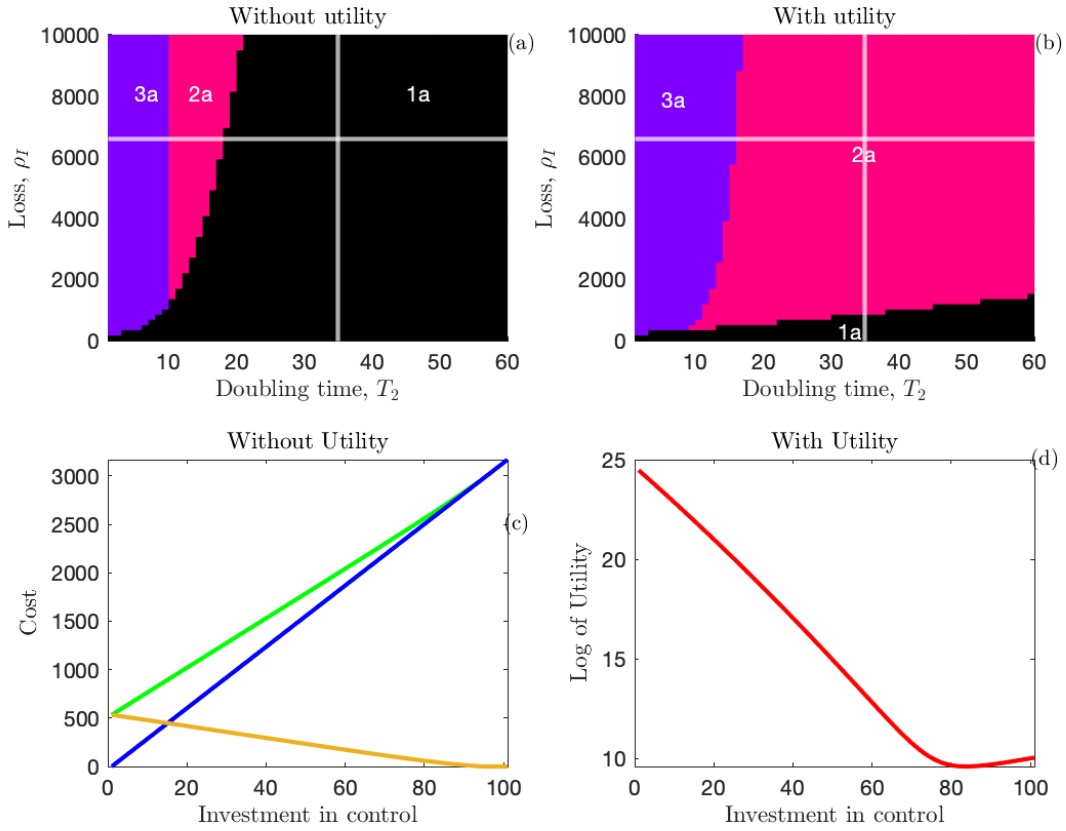

Figure 4.12: The figure illustrates the management strategies when loss, ρ_I value is 6600 £ ha^{-1} $year^{-1}$, and doubling time, T_2 value is 35 years with the Variance=0.1 and λ =−0.001. The heat-map plot shown in Figure 4.12(a) shows the management strategies with variance=0.1 and no disutility. The heat-map plot in Figure 4.12(b) illustrates the management strategies with disutility function. The cost plot is shown in Figure 4.12(c), with the cost of control in blue, the cost of infection in yellow, and the total cost in green. A cost plot with log of disutility function can be seen in Figure 4.12(d). Other parameters values are at their baseline values (See Table 2.1).

Figure 4.12 (a) is a heat map plot that illustrates the optimal management strategies for controlling the spread of disease with the Variance=0.1 and λ =−0.001. The different colours indicate the optimal management strategies for various combinations of control and infection costs. The white cross represents the specific combination of loss and doubling time when the loss, ρ_I, is 6600 £; ha^{-1}; $year^{-1}$, the doubling time, T_2, is 35 years. Figure 4.12 (b) shows the heat map plot for the management strategies with variance=0.1 and the disutility function.

In Figure 4.12 (c), the minimum point on the green line represents the optimal management strategy that balances the direct cost of control and infection with the potential benefits of reducing the spread of infection while also considering the level of risk involved in the decision-making process. In Figure 4.12(d), the red curve in Figure 4.12(d) represents the total cost incorporating the log of the disutility function that accounts for the level of risk involved in the decision-making process. The minimum point on the green curve represents the optimal investment in control measures that minimises the total cost of infection and control. We investment nothing (zero) in Figure 4.7(c) but 80% in Figure 4.7(d). This strategy balances the direct cost of control measures with the potential benefits of reducing the spread of infection, while also considering the level of risk involved in the decision-making process.

4.4 Discussion

In this chapter, we develop a model to explore the impact of risk attitudes on decision-making in disease management, employing a stochastic model integrated with exponential disutility functions. We investigated how the optimal precautionary management strategy changes when costs are evaluated through a disutility function, focusing in particular on decision-making under uncertainty. Our analysis revealed how variations in risk tolerance, as represented by different values of λ, significantly influence the choice of precautionary management (PM) strategies under uncertainty. The chapter effectively highlights how different attitudes towards risk can drastically alter decision-making outcomes. In this context, risk-averse decision-makers (negative values of) prioritise avoiding large infection losses, even if this requires higher precautionary investment. In contrast, risk-seeking decision maker (positive values of) are willing to accept a higher chance of large losses in order to reduce the cost of precautionary management. By contrast, risk-seeking decision-makers (positive values of λ) are willing to accept a higher level of uncertainty in infection outcomes, and therefore a greater chance of severe losses in order to reduce current precautionary expenditure. This dichotomy is crucial in forest disease management, where decisions can have long-term ecological, economic, and social implications.

The disutility function, U_C, is a powerful tool for modelling the subjective value of losses. Using a stochastic model provides a decent, logical, and achievable process for sustainability, which will significantly help decision-making processes. We introduce the risk measure into the objective function to reduce disease transmission. Taking the decision maker's risk attitude into account helps to minimise potential losses and to plan precautionary budgets more effectively under uncertainty during a disease epidemic. Previous studies have used uncertainty to produce input data for disease outbreaks and have estimated recent similar management strategies [113]. Based on these insights, for example, [41] introduce the role of risk preferences in invasive species management decisions and show that higher risk aversion leads to more precautionary investment. Our results are consistent with this pattern, showing

that risk-averse managers allocate higher investment to precautionary management, whereas risk-seeking managers invest less, even under high uncertainty. Their findings reveal that risk-averse managers reduce prevention efforts and amplify control measures. The sensitivity analysis conducted by [41] shows that, even when managers are risk-averse, higher invasion growth rates prompt increased allocation of resources to precautionary measures. Our results show a similar pattern: risk-averse forest managers invest more in precautionary management as the potential rate of disease spread increases.

The analysis focuses on the management strategies to control the spread of the disease, considering different levels of risk aversion and disutility. The goal is to understand how optimal strategies change with varying levels of risk aversion and the inclusion of a disutility function, as well as the effects of changing variance. The analysis begins by exploring the negative value of risk aversion (λ) to investigate the case where caution is prioritised in disease spread control. The analysis demonstrates how different management strategies change with varying risk aversion, disutility, and inclusion of variance. The plots provide insight into the cost and effectiveness of various strategies and help land manager's identify the most optimal approach to controlling the spread of a disease based on specific loss and double-time values.

The graphs of the disutility function illustrate how varying risk preferences influence the perceptions of decision maker about cost and disutility. They provide a visual context to support the discussion of intervention strategies under different risk attitudes. These graphs were instrumental in illustrating the diminishing marginal disutility in the context of increased costs, particularly under varying risk aversions. Our findings underscore the pivotal role of uncertainty in shaping management decisions. As variance increases, indicating greater unpredictability, decision maker naturally gravitate towards more protective strategies. This trend reflects a fundamental aspect of human decision-making under uncertainty, which favours caution in the face of less predictable outcomes. The application of negative and positive values of λ illustrated different risk profiles risk-averse and risk-seeking behaviours. Risk-averse decision maker (negative λ) preferred more aggressive PM strategies, likely driven by a higher aversion to potential losses.

The analysis highlights how management strategies vary with the interplay between potential loss and disease doubling time. Key insights include the trade-offs between total cost, cost of control, and cost of infection, showing that more aggressive interventions are warranted when potential losses are high or disease spreads rapidly. In addition, cost plots with the log of the disutility function were included to show how the inclusion of the disutility function affects the optimal management strategy and the corresponding cost. We observed that the optimal management strategy depends on the specific combination of loss, doubling time, and risk aversion. Depending on the scenario, the optimal strategy may involve investing resources in prevention and control measures, while in other cases, it may be more effective to focus on managing infections. The choice depends on factors such as potential loss, disease spread rate, and risk preferences. Including the disutility function significantly affects the optimal management strategy and the corresponding cost. The disutility function allows the consideration of the potential benefits of different management strategies and the minimisation of losses. Different levels of risk aversion, reflected in the shape of the disutility function, can lead to varying optimal strategies and associated costs. However, the results of this analysis provide valuable information on management strategies to control the spread of plant disease in a farmland setting. The model offers practical guidance for land manager, helping them make informed decisions about where and how to allocate resources. The key messages are that early, precautionary interventions can significantly reduce losses, that strategies should adapt to the speed of disease spread, and that the level of risk aversion influences whether efforts focus more on prevention or on managing infections once they occur.

The optimal strategy may shift from full intervention to partial intervention or from partial intervention to no intervention, depending on the level of risk aversion. The heat map plots indicate the optimal investment in control measures, while the cost plots show the relationship between the cost of control, infection, and total cost. The sensitivity analyses show that risk aversion becomes particularly important when potential losses are high or when the disease spreads rapidly, as it strongly influences the choice of optimal management

strategies. Higher risk aversion leads to more intensive management efforts, while lower risk aversion results in a reduced management strategy. The analyses also show that the disease's variance impacts the optimal strategy, with higher variances leading to increased management efforts. These findings suggest that risk-averse decision makers, such as forest managers and plant traders, tend to prioritise investing in prevention and control measures to avoid large outbreaks of plant and crop infections. These figures provide insights into the optimal management strategies for controlling the spread of the disease under different scenarios, considering costs, potential benefits, and the level of risk aversion. By considering the disutility function and the level of risk, land manager can make more informed choices that balance costs and potential gains while accounting for their risk preferences.

Our result contributes significantly to the understanding of decision-making under risk and uncertainty. It highlights the need for important approaches that consider both the psychological dimensions of risk attitudes and the practical aspects of uncertainty. These insights are particularly relevant today, where decision maker often face complex, high-stakes situations. Although our model provides valuable information, it operates under the assumption of consistent risk preferences, which may only sometimes hold true in dynamic real-world scenarios. Future research could explore models that account for evolving risk attitudes, especially in response to changing environmental or economic conditions. However, this chapter underscores the critical role of risk attitudes in shaping disease management decisions in uncertain circumstances. By integrating these psychological dimensions into stochastic models, we can achieve a more holistic understanding of decision-making processes, paving the way for more effective management strategies in the face of disease outbreaks.

Chapter 5

Precautionary and Reactionary Management Strategy

In this chapter, we use deterministic models to examine the dynamic of disease spread when both precautionary management (PM) and reactionary management (RM) strategies are used. The general mathematical model was introduced in Chapter 2 which describes how the PM strategy protects the forest from invasive pathogens. In Section 5.2, we detail the economic objective function of the reactionary and precautionary management strategies. All the parameters and variables used in this thesis are present within the text, and can also be found, along with their baseline values, in Section 5.3. Section 5.5 presents the results of our sensitivity analysis when the RM strategy is available and optimised. These scenarios help clarify under what conditions prevention (PM) becomes more favourable than reaction. The stochastic result of the precautionary and reactive management strategy is given in Section 5.6. The results are discussed in Section 5.7, where we give our concluding remarks.

5.1 Introduction

Our aim in this chapter is to evaluate how precautionary (PM) and reactionary (RM) management strategies affect overall control costs and losses under different levels of disease

spread and risk aversion. This model serves as both a theoretical framework and a practical tool for resource allocation across the two management strategies (detailed in Chapter 2). Our aim in this chapter is to investigate the economic implications of PM and RM management strategies. The chapter analyses the conditions under which each strategy is effective in reducing the threats posed by emerging pathogens. The reactionary management (RM) strategy is responsive and takes the necessary actions as disease outbreaks unfold. Although more adaptive and flexible, this approach can be more loss prone as it relies on effective response to unanticipated epidemics within a given time frame. These strategies are crucial in guiding organisations and land manager in environments where outcomes are assumed to be predictable based on known variables.

Our analysis focuses on disease spread amongst plants within the forest and examining scenarios where investment can reduce disease losses. We apply optimal control strategy to create a decision-making tool designed to minimise the financial impact of forest diseases under PM and RM management strategies. The model depends on the objective function that includes both the costs of PM deployment, the cost of RM, and the losses due to the impact of the disease on the forest value. The chapter includes sensitivity analyses of key parameters, examining the relationship between annual loss due to diseases, ρ_I, doubling time, T_2, and the sum of costs and losses. These scenarios help clarify under what conditions prevention becomes more favourable than reaction.

We undertake double optimisation to find the optimal PM level and whether RM should be applied. The optimal level of PM, C^*_{pr}, is determined as the level of spending that minimises the net present value of costs while we also consider whether the application of RM decreases the overall cost. This optimisation provides insights into the most economically viable PM and RM strategies, balancing the costs of early intervention against the potential for greater losses if the disease spreads unchecked. One critical aspect we discuss is whether the model is sensitive to parameters, such as the cost of PM and RM and the discount rate. These parameters are pivotal in shaping the optimal strategy, suggesting a balance between economic prudence and the urgency of disease management.

5.2 Economic model of reactionary and precautionary management

When reactionary management (RM) is available, it is applied to the whole forest when the pathogen is detected. The RM reduces the rate of disease transmission by a factor ϵ, where $\epsilon \in [0,1]$: where $\epsilon = 0$ the RM stops additional disease transmission, and when $\epsilon = 1$ the RM has no effect on the disease transmission. Thus, the rate at which the infected area of the forest changes over time is given by

$$\boxed{\left(\frac{dI}{dt}\right)_A = \beta(L - I(t))(I(t) + \delta P),\ 0 < t \leq \tau(\delta).} \tag{5.1}$$

$$\boxed{\left(\frac{dI}{dt}\right)_B = \epsilon\beta(L - I(t))(I(t) + \delta P),\ \tau(\delta) > t.} \tag{5.2}$$

Equation (5.1) represents the rate of change of infected area *before* detection, where $L - I(t)$ is the forest area that is not infected (susceptible but under surveillance) and the label A indicates that this corresponds to the spread of the pathogen before detection. Equation (5.2) represents the rate of change of infected area *after* detection and when the RM strategy has been applied, where the label B indicates that this corresponds to the spread of the pathogen after detection. We assume classical "density-dependent" transmission [77], where the rate of new infections $\frac{dI}{dt}$ is proportional to the infection pressure $I(t) + \delta P$, and the uninfected area $L - I(t)$. If RM is not available, we set $\epsilon = 1$, so Equation (5.1) and Equation (5.2) are the same.

We use the method of separation of variables to solve Equation (2.1), with initial condition $I(0) = 0$ meaning that the forest is initially uninfected (susceptible) and Equation (**??**), with condition $I(\tau(\delta)) = I_1$, as RM is applied when the detection threshold is reached. The infected area of the forest is therefore

$$\boxed{I(t)_A = \frac{L\delta P(e^{\beta(L+\delta P)t} - 1)}{L + \delta P e^{\beta(L+\delta P)t}}, 0 < t \leq \tau(\delta).} \tag{5.3}$$

$$\boxed{I(t)_B = \frac{L(I_1+\delta P)e^{\epsilon\beta(L+\delta P)(t-\tau(\delta))-\delta P(L-I_1)}}{L-I_1+(I_1+\delta P)e^{\epsilon\beta(L+\delta P)(t-\tau(\delta))}},\ \tau(\delta) > t.} \tag{5.4}$$

$$\boxed{I_1 = \frac{LP(e^{(\beta*(L+P)t)}-1)}{L+Pe^{(\beta(L+P)t)}}.} \tag{5.5}$$

The time when the pathogen is detected, $\tau(\delta)$, can be found by substituting $I(\tau(\delta)) = I_1$ into Equation (5.4) and re-arranging to get;

$$\boxed{\tau(\delta) = \frac{1}{\beta(L+\delta P)}\ln\left(\frac{L(I_1+\delta P)}{\delta P(L-I_1)}\right).} \tag{5.6}$$

We assume that the pest starts to damage trees when 5% of the forest is infected. The time the pest starts causing damage, t_{05}, satisfies condition $I(t_{05}) = 0.05L$ and is discussed further in Section 2.4.1. The choice of 5% is an arbitrary starting point and can easily be altered within the model.

The effect of PM (through parameter δ) and RM (through parameter ϵ) on the infected area of the forest are compared in Figure (2.2). The PM reduces the primary infection by δ. The RM is deployed when the infected area reaches the detection threshold I_1, at time $\tau(\delta)$, and the secondary disease transmission rate is reduced by ϵ. The application of RM only ($\delta = 1$ and $\epsilon = 0.5$) shown in the blue and red lines delay the spread of the infection while the infection is stopped when $\epsilon = 0$ (see Figure 5.1(a)). When both PM and RM are applied to the infected land area, PM shifts the disease progress curve to the right, as seen on the blue line, and RM stops the pathogen from spreading once the detection threshold, I_1, is reached, as seen on the red line.

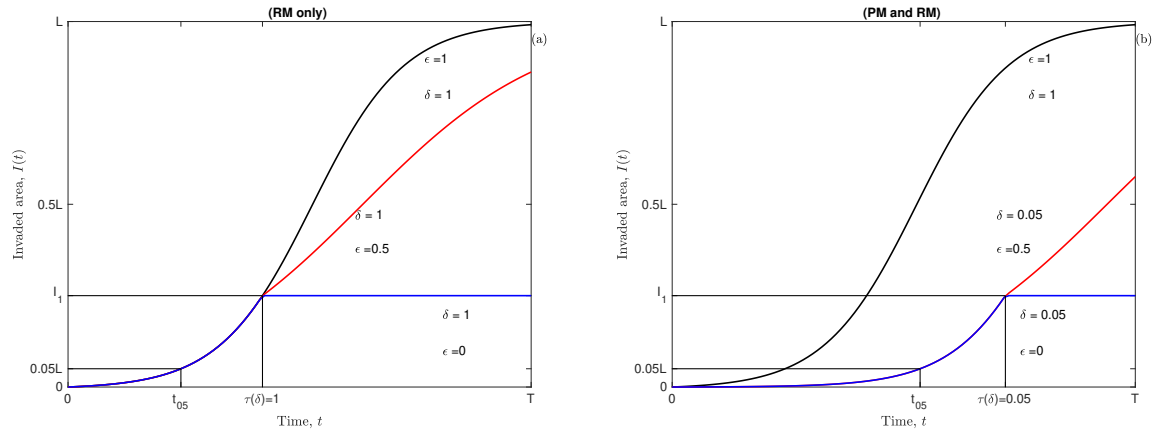

Figure 5.1: The effect of the precautionary management on the infected area of land. The infected area is given by Equation (5.2)(hectares) and shown against time, t (years) when in (a) RM only ($\delta = 1$ and $\epsilon = 0.5$) in red ($\delta = 1$ and $\epsilon = 0$) in blue (b) both PM and RM ($\delta = 0.05$and $\epsilon = 0.5$) in red, $\delta = 0.05$ and $\epsilon = 0$in blue. The time the pest starts causing damage (5% forest infected) is t_{05}, and $\tau(\delta)$ is the time when the detection threshold I_1 is reached.

Assuming that an RM strategy is available, the land manager wants to find the optimal level of PM that minimises the present value of costs associated with the disease over a fixed time horizon, $0 \leq t < T$, since the RM strategy is applied once the pathogen is detected (period C). The cost of PM over the time horizon $0 \leq t < T$ is still $C_p L$.

The loss caused by the disease is given by $\rho_I I(t)$ until the time the infection is detected and the RM is deployed, $t_{05} \leq t < \tau(\delta)$ (period B). There is a one-time cost of applying RM to the whole forest at $\tau(\delta)$ which is $C_r L$, where C_r is the cost per unit area of RM (£$^{-1}$ hectare^{-1}). We consider a situation where the RM is applied to the whole forest, as this could represent a case of not knowing which trees/plants are infected (asymptomatic infection) and hence having to apply treatment to all of them. If the detection threshold drops below the forest area or if there is no asymptomatic infection, then applying RM to the whole forest may be unrealistic. In this case, the land manager may only apply the RM to the known infected area or the known infected area plus a buffer area (a set of land around the known infected area that might be infected). These options are easy to include within this model but we are not considering them here. The benefit of RM is that it reduces the rate of pathogen once it detected (through ϵ in Equation 5.4 and Figure (2.2)), and hence reduces the loss caused by

the disease.

The loss accrued over the remaining time horizon *after* the RM is applied ($\tau(\delta) \leq t < T$) is $\rho_{PI} I(t)$, where ρ_{PI} is the annual, per unit area reduction in economic and environmental benefits after the RM has been applied (henceforth "post-RM loss"; £$^{-1}$ hectare $^{-1}$ year $^{-1}$). We assume that the annual post-RM loss is less than the annual loss incurred before the RM is applied, for example $0 \leq \rho_{PI} \leq \rho_I$. Moreover, when $\rho_{PI} = 0$, the ecological benefits are fully restored after implementing the RM, but when $\rho_{PI} = \rho_I$, the RM has no effect on restoring the ecological benefits (but the RM can still reduce infection transmission as discussed in Section 2.2). When $\rho_{PI} \neq 0$, the RM strategy (for example, a treatment that is not fully effective) only reduces the negative effect of the disease (not mitigates it). In this case, the disease would still be able to transmit (so $\epsilon > 0$ in Equation (5.4)). However, if the RM strategy is to remove infected trees/plants (in order to remove the infection), then despite the infection being removed, there would still be a reduction in the benefit provided by those trees/plants (that were removed). This scenario can be considered in our model by examining the sensitivity to the post-RM loss parameter, ρ_{PI}, and assuming that the removal has stopped future spread (thus $\epsilon = 0$ in Equation (5.4)).

When an RM strategy is available and applied at the detection threshold, the objective function giving the present value of the total cost is

$$J_{pr}(C_{pr}) = \int_0^T (C_p L e^{-rt}) dt + \int_{t_{05}}^{\tau(\delta)} (\rho_I I(t)_A e^{-rt}) dt + C_r L e^{-r\tau(\delta)} + \int_{\tau(\delta)}^T (\rho_{PI} I(t)_B e^{-rt}) dt, \tag{5.7}$$

The first term represents the cost of PM throughout the period A, B and C. The second term corresponds to the loss caused when the pathogen starts to cause damage before detection. The third term described the one-off cost of RM to the whole forest when the losses caused by the pathogen are detected. The last term represents the loss accrued over the remaining time horizon after the RM is applied (post-RM loss).

The optimal level of PM, C^*_{pr}, is given by solving

$$\boxed{C^*_{pr} = arg \min_{C_p \in [0,\Pi]} J_{pr}(C_{pr})} \tag{5.8}$$

subject to Equation (5.4 - 5.3)

Equation (5.8) gives the minimisation problem when RM is $always$ applied to the detection threshold. However, this may not be the optimal solution if the RM is, for example, very expensive or ineffective. Therefore, we undertake double optimisation to find the optimal PM level and whether RM should be applied. The optimal level of PM is given by

$$\boxed{C^* = C^*_p, \text{ if } J_p(C^*_P) \leq J_{pr}(C^*_{pr})} \tag{5.9}$$

Equation (5.9) shows that, when $C^* = C^*_p$ the minimum loss is given by $J_p(C^*_p)$ in Equation (**??**), and it is optimal not to apply the RM.

$$\boxed{C^{**} = C^*_{pr}, \text{ if } J_p(C^*_P) > J_{pr}(C^*_{pr})} \tag{5.10}$$

Whereas, when $C^{**} = C^*_{pr}$ the minimum loss is given by $J_{pr}(C^*_{pr})$ in Equation (**??**), and Equation (5.10) shows that it is optimal to apply RM. Note Equations (5.9) and (5.10) were only used to optimise if RM should be applied, but we do not optimise when RM should be applied or the level of RM that should be applied.

5.3 Parameter and variable definitions and estimates

Table 5.1: Parameter and variable descriptions, alongside their baseline values.

Parameter	Description	Baseline values	Range of tested values
EPIDEMIOLOGICAL			
t	Time horizon(years), $t \in [t_0, T]$	$t_0 = 0$, $T = 100$	-
L	Area of the forest (hectares)	$L = 1$	-
$I(t)$	Infected area of the forest at time t, $I(t) \in [0, L]$	Equation (2.1)-(**??**)	-
P	Baseline primary infection	$P = 0.005$	$P \in \{0, 0.0005, 0.005, 0.1\}$
ϵ	Reduction in secondary disease transmission due to RM, $\in [0,1]$	$\epsilon = 1$	$\epsilon \in \{0, 0.5, 1\}$
t_F	Time taken for F% of the forest to become infected when PM and RM is available and deployed	Equation (5.3)-(5.4)	-
β	Baseline secondary disease transmission rate	$\beta = 0.1$	$\beta \in [0, 0.3]$
$1-\delta_M$	Maximum reduction in primary infection due to PM, $\delta_M \in [0,1]$	$\delta_M = 0$	$\delta_M \in \{0, 0.05, 0.2, 0.5, 0.8\}$
δ	Reduction in primary infection transmission due to PM	Equation(2.6)	$\delta \in [0, \delta_M]$
I_1	Detection threshold: area of the forest that is infected (hectares) $I_1 \in [0,1]$	$I_1 = 0.25$	$I_1 \in \{0.1, 0.25, 0.5, 0.8\}$
$\tau(\delta)$	Time the infection is detected and RM is deployed (years)	Equation(5.6)	-
t_{05}	Time the pest start causing damage	Equation(2.3)	-
ϵ	Reduction in secondary disease transmission due to RM, $\in [0,1]$	$\epsilon = 1$	$\epsilon \in \{0, 0.5, 1\}$
T_2	Doubling time(years)	Equation(**??**)	$T_2 \in [0, 60]$
ECONOMIC			
ρ_I	Annual per unit area loss due to disease (£ ha^{-1} year^{-1})	-	$\rho_I \in [0, 10000]$
ρ_{PI}	Annual per unit area post RM loss due to disease (£ ha^{-1} year^{-1})	$\rho_{PI} = 0$	$\rho_{PI} \in [0, 6000]$
C_p	Annual per unit area cost of PM without RM (£ ha^{-1} year^{-1})	$0 \le C_p \le \Pi$	-
C_r	One-off per unit area cost of RM (£ ha^{-1})	$C_r = 3000$	$C_r = \{0, 3000, 5000\}$
Π	Annual per unit area PM budget (£ ha^{-1} year^{-1})	$\Pi = 100$	-
r	Discount rate	$r = 0.03$	-
C_p^*	Optimal cost of PM without RM (£ ha^{-1} year^{-1})	Equation(**??**)	$0 \le C_p^* \le \Pi$
C_{pr}^*	Optimal cost of PM and RM (£ ha^{-1} year^{-1})	Equation(5.8)	$0 \le C_{pr}^* \le \Pi$
C^*	Optimal cost of PM without optimal RM (£ ha^{-1} year^{-1})	Equation(5.9)	$0 \le C^* \le \Pi$
C^{**}	Optimal cost of PM with optimal RM (£ ha^{-1} year^{-1})	Equation(5.10)	$0 \le C^* \le \Pi$

5.4 Results

When RM is available, the optimal management strategy (the optimal level of PM, C_{pr}^*, C^* and C^{**}) is found by Equations (5.8), (5.9), (5.10). The optimal level C_{pr}^* is when the RM strategy is available and applied at the detection threshold. The optimal level C^* is when we do not apply the RM strategy because the pathogen is not detected within the time horizon $\tau(\delta) > T$. The optimal level C^{**} is when we deploy the RM strategy because the pathogen is not detected within the time frame. Initially, assume that the maximum reduction in the transmission of primary infection due to PM is $\delta_M = 0$. The RM strategy will be "perfect" if it stops the disease from spreading, $\epsilon = 1$, and completely restores the provision of environmental and economic benefits, $\rho_{PI} = 0$. This analysis is similar to that of Figure (2.6) where RM was not

available. The key difference here is that RM is deployed once the pathogen is detected in order to reduce further damages over the remaining planning period. The RM strategy is applied in two ways;

- When the RM strategy is available and optimised (see Table (5.2) and Figure 5.2(b)).
- When the RM strategy is available and always deployed upon detection (see Table (A.1) and Figure A.1(b)).

We carried out a sensitivity analysis on key parameters: doubling time, T_2 depending on the baseline secondary disease transmission rate (β), and the impact on loss ρ_I. In other to simulate the optimal management strategy, we conducted a sensitivity analysis on some other secondary parameters. The investigation was carried out using numerical simulation techniques to find out how the optimal management strategy depends on the following parameters:

- the baseline primary infection (P),
- time horizon (T)
- the one-off cost of the RM (C_r),
- the detection threshold (l_1),
- the maximum reduction in primary infection transmission due to the PM ($1-\delta_M$),
- the reduction in secondary disease transmission due to the RM (ϵ),
- the post-RM loss $\left(\rho_{PI}\right)$.

The notation and baseline values of the parameters are given in Table 5.1.

5.5 When the RM strategy is available and optimised

Now let us assume that the RM strategy is available and optimal. The optimal level of PM, C^* and C^{**}, is found by Equation (5.9) and Equation (5.10) labelled in Table (5.2). The optimal management strategy is the optimal level of PM, C^*_{pr}, and whether it is optimal to deploy RM (Off/On). When it is optimal not to deploy the RM, C^*, then the pathogens do not arrive within the time horizon; therefore, using RM would generate costs without any corresponding benefit and is therefore not cost-effective "Off". When it is optimal to deploy the RM, C^{**}, then the pathogens arrive in the time horizon; hence it is cost-effective; "On". When the PM's optimal level is zero ($C^{**} = 0$), we label this "None" and label the management strategy $1c$ yellow. Furthermore, when the optimal level of PM is equal to the budget ($C^{**} = \Pi$), we label this zone "Full" and label the management strategy $3c$ red.

Optimal management strategy	PM (Optimised)	RM(Off/On)
$1a$ (black)	None, $C^* = 0$	Off, $\tau(\delta) > T$.
$2a$ (pink)	Partial, $0 < C^* < \Pi$	Off, $\tau(\delta) > T$.
$3a$ (purple)	Full, $C^* = \Pi$	Off, $\tau(\delta) > T$.
$1c$ (yellow)	None, $C^{**} = 0$	On, $\tau(\delta) \leq T$.
$3c$ (red)	Full, $C^{**} = \Pi$	On, $\tau(\delta) \leq T$.

Table 5.2: The potential combination of management strategies. considered when the RM is optimal.

When the RM strategy is available and optimal, we perform a double optimisation to find the optimal level of PM and whether RM should be applied. This approach ensures that resources are not allocated to RM when the pathogen does not arrive within the planning horizon, or when the costs of deploying RM exceed the resulting reduction in infection-related damages. In other words, the decision is based on a cost-effectiveness comparison between the cost of implementing RM and the expected reduction in losses (i.e. infection costs) over the remaining planning period. Therefore, this will help to increase the effectiveness of the

RM strategy. Firstly, if the present value of total costs when the RM strategy is not available, $J_p(C_p)$, in Equation (2.5) is *less than or equal to* the present value of the total cost when the RM strategy is available and applied at the detection threshold $J_{pr}(C_{pr})$ in Equation (5.7), it is optimal not to apply the RM. Hence, the optimal level of PM, C^*, given by Equation (5.9) shows that $C^* = C_p^*$ (Equation (5.9) = Equation (5.7)). The analysis shows that the minimum loss will be given by $J_p(C_p)$ (Equation (2.5)); therefore, the loss caused by the disease is smaller than the cost of implementing the RM.

Secondly, if the present value of total costs when the RM strategy is not available, $J_p(C_p)$, in Equation (2.5) is *greater than* the present value of the total cost when the RM strategy is available and applied at the detection threshold $J_{pr}(C_{pr})$ in Equation (5.7), it is optimal to apply the RM. Then, the optimal level of PM, C^{**}, given by Equation (5.10) shows that when $C^{**} = C_{pr}^*$ (Equation (5.10) = Equation (5.8)), it will be optimal to apply the RM. Hence, the minimum loss will be given by $J_{pr}(C_{pr})$ (Equation (5.7)). Therefore, once the disease causes substantial damage, the most effective way to reduce further losses is to deploy the RM strategy.

5.5.1 Sensitivity analysis with respect to the annual loss, ρ_I and the doubling time, T_2

The sensitivity analysis with respect to the annual infection damage ρ_I, and the doubling time, T_2, when the RM strategy is available and optimised, is shown in Figure 5.2. We show in Figure 5.2(a), the case where the RM is not available as shown in Figure (2.6). We have zone $3a$, $2a$ and $1a$ as we have in Figure (5.6), but now zone $1c$ appears in Figure 5.2(b) where RM is available. The two Figures, shows a significant difference when RM is not available 5.2 (a) and when RM is available Figures 5.2 (b). Initially, we assume that the maximum reduction in primary infection transmission due to the PM is $\delta_M = 0$ and the RM is "perfect" it stops the disease from spreading, $\epsilon = 1$, and completely restores the provision environmental and economic benefits, $\rho_{PI} = 0$. This means that RM is assumed to be fully effective in order to

isolate the effect of timing on the decision to deploy RM. The black region (zone $1a$) shows where it is optimal to do nothing. The pink region (zone $2a$) shows where it is optimal to deploy partial PM. The purple region (zone $3a$) shows where it is optimal to deploy full PM. The yellow region (zone $1c$) shows where it is optimal to deploy the RM strategy. The baseline primary infection, $P = 0.005$ and the parameter $\delta_M = 0$.

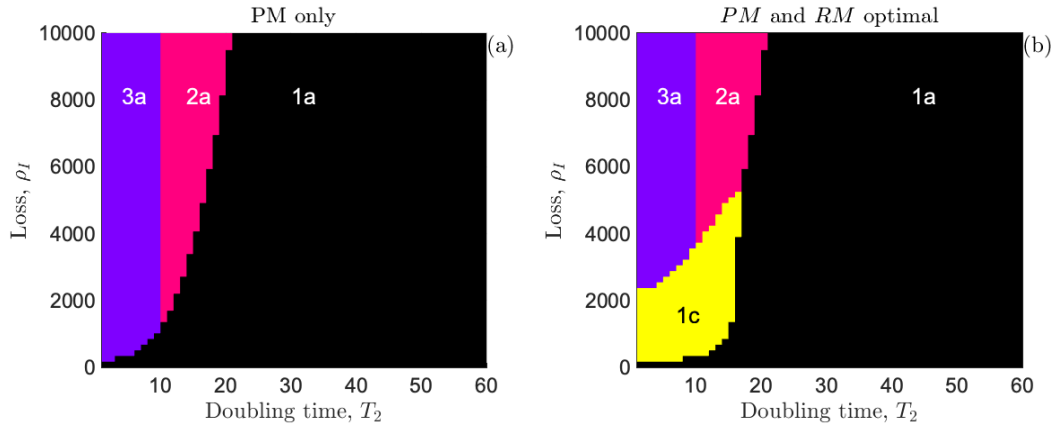

Figure 5.2: Sensitivity analysis to the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by (a) when RM is not available (as given by Equation (2.7) as described in Table 2.2) and (b) when RM is available and always optimised (as given by Equation 5.9 and 5.10 as described in Table 5.2). The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The baseline primary infection, $P = 0.005$ and the parameter $\delta_M = 0$, while other parameter values are at their baseline in Table 5.1.

We use numerical simulation techniques to find out how the optimal management strategy depends on the following parameters: annual loss (ρ_I), and doubling time T_2. For small values of the doubling time, T_2, the optimal strategy depends on the level of annual loss, ρ_I, . When ρ_I, is high, the best approach is to implement full precautionary measures (PM) in zone $3a$, without reactionary measures (RM). Similarly, for the middle values of ρ_I,, full PM is optimal in zone $3a$, again without RM because of the time interval $(\tau(\delta) > T)$. However, when ρ_I, is low, the optimal solution is to implement no PM but to rely on RM in zone $1c$. For the middle values of the double time T_2, the management strategy is also influenced by ρ_I,. The high

and middle levels of ρ_I, call for a partial PM deployment (zone $2a$), with no RM. On the other hand, at low ρ_I,, the best strategy shifts to deploying no PM but using RM (zone $1c$). For large values of T_2 ($T_2 = 80$), regardless of the value of ρ_I,, the optimal approach is to implement no PM and no RM, placing the solution in zone $1a$.

When the annual loss, ρ_I, is relatively small in Figure 5.2, it will be optimal to do nothing. As the annual loss, ρ_I, slightly increase in Figure 5.2(b) the parameter space where it is optimal to apply the RM emerges at a short doubling time (zone 1c). Hence, the loss caused by the disease starts to accrue earlier in the time horizon as the loss increases, the optimal management strategy changes from applying the RM (zone1c) to apply PM (zone3a) in Figures 5.2(b). While, in Figure 5.2(a), as the annual loss, ρ_I, slightly increase the parameter space where it is optimal to apply the Full PM emerges at a short doubling time (zone 3a). These scenarios represent situations where (i) the disease spreads rapidly (short T_2) and causes substantial damage each year, or (ii) the disease spreads more slowly but still generates significant costs over time. However, as annual infection damage increases, losses begin to accrue earlier in the time horizon, and it becomes more cost-effective to invest in PM prior to detection to reduce the impact of a potential outbreak (zone $3a$).

Importantly, at a high doubling time and low annual loss, the optimal management strategy switch from applying RM (zone $1c$) to do nothing (zone $1a$) in Figure 5.2(b). While at a high doubling time and high annual loss, the optimal management strategy switch from applying PM (zone $3a$ and zone $2a$) to do nothing (zone $1a$) in Figure 5.2(b). However, at a high doubling time in Figure 5.2(a), it is optimal to do nothing (zone $1a$) as the annual loss is increasing. Under some conditions when the loss ρ_I increases, the optimal management strategy will switch as the doubling time T_2 decreases and it will be optimal to deploy PM only (see zone $1c \rightarrow 3a \rightarrow 2a$ in Figure 5.2(a). As the annual loss, ρ_I, decreases, the switch in strategies from deploying the RM to deploying the PM occurs at smaller values of T_2 since a higher proportion of the forest is required to be infected before it becomes cost-effective to deploy the PM. When the loss is very small, a region where it is optimal to do nothing arises and is remains independent of T_2 (β) value (zone $1a$ in Figure(5.2), since the cost of deploying

the PM or the RM, is much higher than the total loss caused by the disease. The full PM (zone $3a$) is deployed in a very shallow doubling time when the annual loss is high. The switch from no PM (zone la) to partial PM (zone $2a$) occurs within the middle doubling time at a high annual loss.

The pathogen will arrive and spread in zone $2a$, where it is optimal to deploy partial PM, so we deploy partial PM to counter the loss. This happens because there is an increase in loss from disease, so it is optimal to deploy the PM to counter this loss. Furthermore, the region where it is optimal to deploy the RM once the pathogen is detected is earlier in the time horizon. This means that we do not deploy the RM strategy when the disease does not arrive in the time horizon or is not cost-effective. Importantly, when it is optimal to deploy the RM, the level of PM required is sufficient so that the time the loss begins to accrue is pushed to outside the time horizon (e.g., $t_{05} > T$); thus the RM is not needed. Without the PM or RM strategy, the total loss increases because the effects of the loss are realized earlier. Therefore, waiting to apply the RM is too costly, and it is more cost-effective to deploy the PM annually to prevent the pathogen from arriving.

5.5.2 Impact of investment management strategy costs on invasion dynamics

To understand how parameters affect model results, we show how the time the pest starts to cause damage, t_{05}, and the detection time, $\tau(\delta)$, change with the reduction in the primary infection transmission rate due to PM, δ and the investment in PM deployment, C_p. PM reduces the rate of primary infection by δ. RM is implemented when the infected area reaches the detection threshold I_1 at time $\tau(\delta)$, reducing the secondary disease transmission rate by ϵ. The pathogen is always introduced but may not cause damage if $t_{0.5} > T$, or may cause undetected damage if $\tau(\delta) > T$. Without intervention (ie, $\delta = \epsilon = 1$), the pathogen arrives and spreads as shown in the graph. This result shows that the reduction in primary infection transmission rate, δ, depends on the cost of deploying the PM, C_p, the higher the C_p, the

greater the investment in the PM, the greater the reduction in the primary transmission rate.

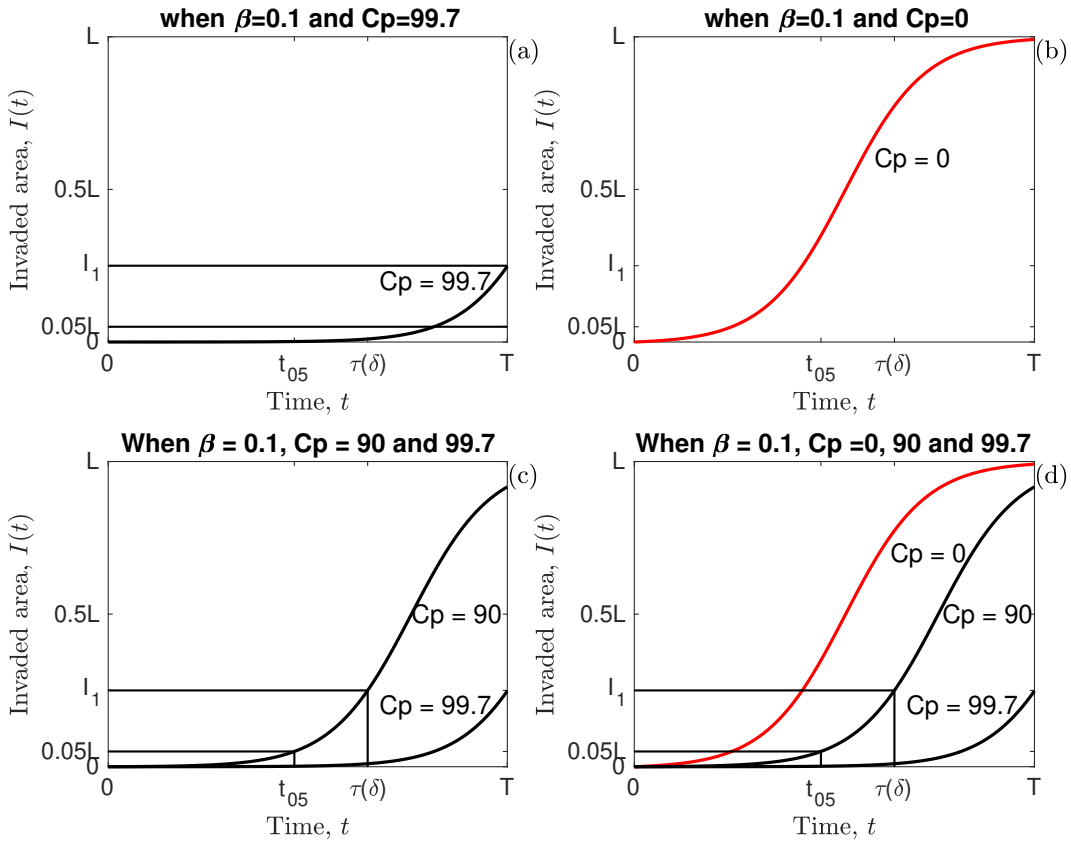

*Figure 5.3: The effect of PM investment management strategies on the area of the infected forest over time. Results were found by solving I(t) in Equation (5.3)-(5.4) and Equation (**??**) for different investments in PM deployment, $C_p = 0, 90, 99.7$. The red colour shows when there will be no reduction in disease transmission when no budget is invested ($C_p = 0$) while black shows when PM or RM is applied and other parameters are as in Table 5.1.*

The outcome of different management strategies in the area of infected forest over time is shown in Figure 5.3. The area of the infected forest is shown in Equation (5.3) and (5.4). PM reduces the rate of primary infection transmission through C_p as we linked C_p to δ in Equation 2.6. When no budget is invested ($C_p = 0$), there will be no reduction in infection transmission, therefore, no PM or RM is applied at this point. When some part of the budget is invested $C_p = 90$, there will be a slight reduction in the transmission of diseases. When the full PM budget is spent $C_p = 99.7$, there will be a more significant reduction in primary

infection transmission.

Figure 5.3 help us to expand more on why the yellow zone $1c$, the purple zone $3a$, the pink zone $2a$ and black zone $1a$ are shown in Figure A.1. The yellow zone $1c$ is a very fast epidemics and this is shown in Figure 5.3, zone $1c$ strategy is implemented if $\tau(\delta) > T$. This happens because the spread of the pathogens is spreading so fast and the only solution will be to apply RM and not PM. The plot in Figure 5.3 demonstrates how C_p affects the reduction in the primary infection transmission rate (δ) and the timing of pest damage initiation ($t_{0.5}$) and detection ($\tau(\delta)$). The results highlight that higher investments in PM ($C_p = 99.7$) significantly reduce primary transmission, while no investment ($C_p = 0$) leads to increased spread of the disease. This analysis emphasizes the importance of budget allocation in determining effective management strategies and their influence on disease transmission dynamics.

5.5.3 Sensitivity analysis with respect to baseline primary infection, P

This section discus the analysis with respect to the baseline primary infection, P against the doubling time, T_2 which depend on β (baseline secondary disease transmission rate), and the annual loss, ρ_I, when the RM strategy is available and always optimised. The plots in Figure (5.4) show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation (5.9) and (5.10) as described in Table (5.2). The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. We use the numerical simulation techniques to find how the optimal management strategy is dependant on the baseline primary infection, P: (a) $P = 0.0005$, (b) $P = 0.005$, (c) $P = 0.1$. All other parameters are at their baseline value, as shown in Table (5.1).

The optimal solution from Figure 5.4(a) when $P = 0.0005$ are as follow:

- For small $T_2(\beta)$, depending on the annual loss, ρ_I:
  - high ρ_I, full and partial PM (zone $3a$ and $2a$) only.
  - middle ρ_I, full PM (zone $3a$) and no RM.
  - low ρ_I, no PM and RM (zone $1c$)
- For middle $T_2(\beta)$, regardless of ρ_I:
  - no PM and no RM (zone $1a$)
- For large $T_2(\beta)$, regardless of ρ_I:
  - no PM and no RM (zone $1a$)

The optimal solution from Figure 5.4(b) when $P = 0.005$ are as follow:

- For small $T_2(\beta)$, depending on the annual loss, ρ_I:
  - high ρ_I, full PM (zone $3a$) and no RM.
  - middle ρ_I,full PM (zone $3a$) and no RM.
  - low ρ_I, no PM and RM (zone $1c$)
- For middle $T_2(\beta)$, depending on the annual loss, ρ_I:
  - high ρ_I, partial PM (zone $2a$) and no RM.
  - middle ρ_I, partial PM (zone $2a$) and no RM.
  - low ρ_I, no PM and RM (zone $1c$)
- For large $T_2(\beta)$, regardless of ρ_I:
  - no PM and no RM (zone $1a$)

The optimal solution from Figure 5.4(c) when $P = 0.1$ are as follow:

- For small $T_2(\beta)$, depending on the annual loss, ρ_I:
  - high ρ_I, full PM (zone $3a$) and no RM.
  - middle ρ_I,full PM (zone $3a$) and no RM.
  - low ρ_I, no PM and RM (zone $1c$)
- For middle $T_2(\beta)$, depending on the annual loss, ρ_I:
  - high ρ_I, partial PM (zone $2a$) and no RM.
  - middle ρ_I, partial PM (zone $2a$) and no RM.
  - low ρ_I, no PM and RM (zone $1c$)
- For large $T_2(\beta)$, depending on the annual loss, ρ_I:
  - high ρ_I, partial PM (zone $2a$) and no RM.
  - middle ρ_I, partial PM (zone $2a$) and no RM.
  - low ρ_I, no PM and no RM (zone $1a$)

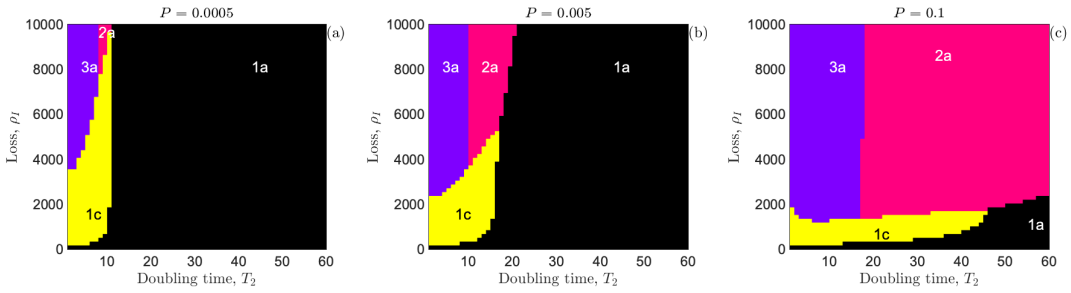

Figure 5.4: Sensitivity analysis to the baseline primary infection, P, when the RM strategy is available and always optimised. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.9 and 5.10 as described in Table 5.2. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The baseline primary infection is: (a) $P = 0.0005$, (b) $P = 0.005$ and (c) $P = 0.1$. All other parameters are at their baseline values in Table 5.1.

We show in Figure 5.4 that as the doubling time, T_2, increases, the optimal level of PM increases, and a region in the parameter space where it is optimal to apply RM only emerges when the loss ρ_I, is relatively small (zone 1c). When the annual loss, ρ_I, is small, the optimal management strategy switches from doing nothing to applying RM (zone 1a $\rightarrow$ 1c in Figures 5.4(b)) when the loss caused by the disease starts to accrue earlier in the time frame or the disease spreads faster. The black region (zone la) shrinks, the region with pink (zone $2a$) grow, the region with purple (zone $3a$) grows and the part with yellow (zone $1c$) disappears gradually as the baseline primary infection rate, P, increases. The optimal management strategy switch from not applying PM and RM (zone $1a$) to applying RM only (zone $1c$) in Figure 5.4(b). This happens because the loss caused by the disease is small, and so waiting to apply the RM and incurring the one-off cost is more beneficial than deploying the PM annually. The increase in loss, ρ_I, shows the switch (zone $1c \rightarrow$ zone $3a \rightarrow$ zone $2a$) in the optimal management strategy as the doubling time, T_2 increases , and it is optimal to deploy only PM in Figure 5.4(b). As the annual loss, ρ_I, decreases, the switch in strategies (from deploying the RM to deploying the PM) occurs at smaller values of the doubling time, T_2, since a higher proportion of the forest is required to be infected before it becomes cost effective to deploy the PM (vs. waiting to apply the RM). When the loss is very small, a region where it is optimal to do nothing arises and is remains independent of T_2 (β) value (zone $1a$ in Figure 5.4, since the cost of deploying the PM or the RM is much higher than the total loss caused by disease. As the baseline primary infection, P, is increasing the time at which the loss starts to accrue is decreased and there is an increase in the region where it is optimal to deploy the PM (zone $2a$ and $3a$ in Figure (5.4).

This analysis aims to detect the likelihood that the pathogen will arrive and the rate of spread of the infection within the forest when the primary infection, P, is altered. Hence, Figures 5.4 (a) and (c) show a significant difference compared to Figure 5.4 (b), where the sensitivity analysis was carried out. However, Figure 5.4(b) is the same as Figure 5.2(b) above. In general, Figure 5.4(a) show what happens when the P is reduced to $P = 0.0005$. While 5.4(b) shows the effect on the arrival and spread of the pathogen when P is at the baseline value ($P = 0.005$). Then, Figure 5.4(c) shows The effect of the pathogen when P is increased

to ($P = 0.1$). We observe that zone $1a$ which is do nothing reduces as the primary infection, P, increases while zone $2a$ and zone $3a$ grow as P increases. Zone $1c$ gradually reduces as the primary infection, P, increases. This trend is seen for different values of P. However, the switch from no PM and no RM (zone $1a$) to RM strategy (zone $1c$) and from RM to partial PM (zone $2a$), and from partial PM (zone $2a$) to full (zone $3a$), occurs from smaller to a higher value of the annual loss, ρ_I, with respect to the doubling time.

Similar to when the RM is unavailable (Figure 2.8), as the baseline primary infection, P, is increased the time at which the loss starts to accrue is decreased, and thus there is a decrease in the region where it is optimal to do nothing (zone 1a), and a subsequent increase in the region where it is optimal to deploy the PM (zone $2a$ and $3a$ in Figure 5.4). Interestingly, under some conditions when the annual loss is, say, $\rho_I = 2500$, the optimal management strategy will switch again as T_2 is decreasing, and it will be optimal to deploy PM only (zone $1c \rightarrow 2a \rightarrow 3a$) in Figures 5.4. Therefore, waiting to apply the RM is too costly, and it is more cost effective to deploy the PM annually to prevent the pathogen from arriving. This is emphasised in Figure 5.4, where the region where it is optimal to wait and apply the RM (zone 1c) disappears when the baseline primary infection is high. However, since the loss caused by disease starts to accrue very early, and the cost of applying the RM is realised earlier too. Considering the research objective, which is to find the optimal level of PM which minimises the present Value costs associated with the disease, this may not be the best solution if the cost of deploying the RM strategy is very expensive.

5.5.4 Sensitivity analysis with respect to time, T

The plots in Figure 5.5 show the sensitivity analysis with respect to time, T, the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy when RM is available and optimised. The optimal management strategy is found by Equation 5.9 and 5.10 as described in Table 5.2. The numerical simulation techniques were used to find the optimal management strategy with different time horizon: (a) $T = 50$, (b) $T = 100$, (c) $T = 150$. This can be seen

in Figures 5.5(a),5.5(b) and 5.5(c) respectively. The black region (zone 1a) shows where it is optimal to do nothing. The pink region (zone 2a) is optimal to deploy partial PM and no RM. The purple region (zone 3a) is optimal to deploy full PM and no RM. The yellow region (zone 1c) shows where it is optimal to deploy RM and no PM.

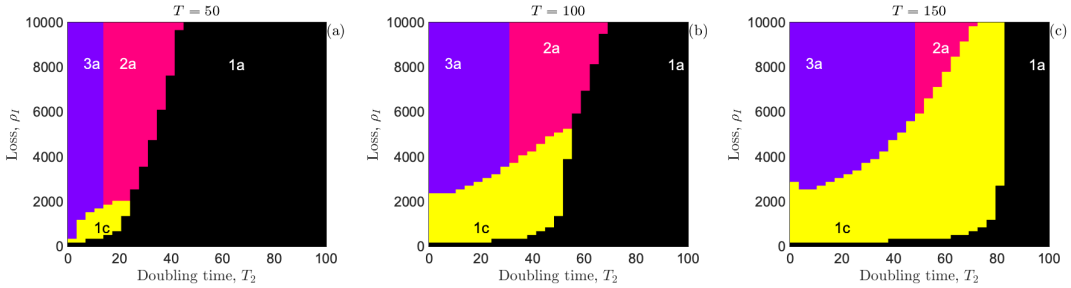

Figure 5.5: Sensitivity analysis to time, T, when RM is available and optimised. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.9 and 5.10 as described in Table 5.2. The black region (zone 1a*) shows where it is optimal to deploy no PM and no RM. The pink region (zone* 2a*) is optimal to deploy partial PM and no RM. The purple region (zone* 3a*) is optimal to deploy full PM and no RM. The yellow region (zone* 1c*) shows where it is optimal to deploy RM and no PM. The analysis was carried out at different time horizon: (a)* $T = 50$*, (b)* $T = 100$ *and (c)* $T = 150$*. All other parameters are at their baseline values in Table 5.1.*

Interestingly, when the time is reduced to $T = 50$, it will be optimal to deploy only the PM strategy. This is as a result of the region where it is optimal to deploy RM will reduce at a very low loss and at short doubling time. Considering the plot in Figure 5.5(a), at a short time horizon, the optimal management strategy tends to the RM not to be deployed. This is as a result of the reduction in the region where it is optimal to deploy the RM strategy. The black region (zone 1a) shows that the annual loss is very small at a high doubling time. When the time, T, is increased to $T = 100$ similar to sensitivity analysis to doubling time and annual loss (Figure A.1), the region in the parameter space where it is optimal to apply the RM only emerges when the loss, ρ_I, is relatively small (zone 1c in Figure 5.5(c)). When the time is increased to $T = 150$, the region where it is (zone 1c) increases from small animal loss, ρ_I, to a very high loss. The region where it is optimal to apply full PM increases as the time is increased. However, the region where it is optimal to apply partial PM gradually disappears

as the time is increased. However, there is a decrease in the region where it is optimal to do nothing (zone $1a$) at high time horizon. Undertaking sensitivity analysis to the time horizon, T, reveals a great change in the optimal management strategy (Figure 5.5). More specifically, an increase in the time horizon, T, increase the potential loss caused by disease since the rate that the whole forest will become infected is increased (See Figure 4.7(c)). Therefore, as time increases the optimal level of the RM increases in other to counter the loss (see the switch in zone $1c$ from Figure 5.5(a) → Figure 5.5(b)→ Figure 5.5(c)). However, the switch from no PM (zone $1a$) to partial PM (zone $2a$) to full PM (zone $3a$) gradually decreases while the zone to deploy the RM strategy increases with time.

5.5.5 Sensitivity analysis with respect to the One-off cost of RM, C_r, and the detection threshold, I_1

Sensitivity analysis with respect to the detection threshold, I_1, and cost of the RM, C_r, when RM is available and optimised. The plots Figure 5.6 show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.9 and 5.10 as described in Table 5.2. The numerical simulation techniques were used to find the optimal management strategy with different values of detection threshold, I_1, and cost of the RM, C_r: (a) $I_1 = 0.1$ and $C_r = 0$, (b) $I_1 = 0.1$ and $C_r = 3000$, (c) $I_1 = 0.1$ and $C_r = 5000$, (d) $I_1 = 0.25$ and $C_r = 0$, (e) $I_1 = 0.25$ and $C_r = 3000$, (f) $I_1 = 0.25$ and $C_r = 5000$, (g) $I_1 = 0.5$ and $C_r = 0$, (h) $I_1 = 0.5$ and $C_r = 3000$, (i) $I_1 = 0.5$ and $C_r = 5000$, (j) $I_1 = 0.8$ and $C_r = 0$, (k) $I_1 = 0.8$ and $C_r = 3000$, (l) $I_1 = 0.8$ and $C_r = 5000$. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The detection threshold is, $I_1 = 0.1, 0.25, 0.5, 0.8$, and cost of the RM is, $C_r = 0, 3000, 5000$ as given on the title of each plot. All other parameters are at their baseline values in Table 5.1.

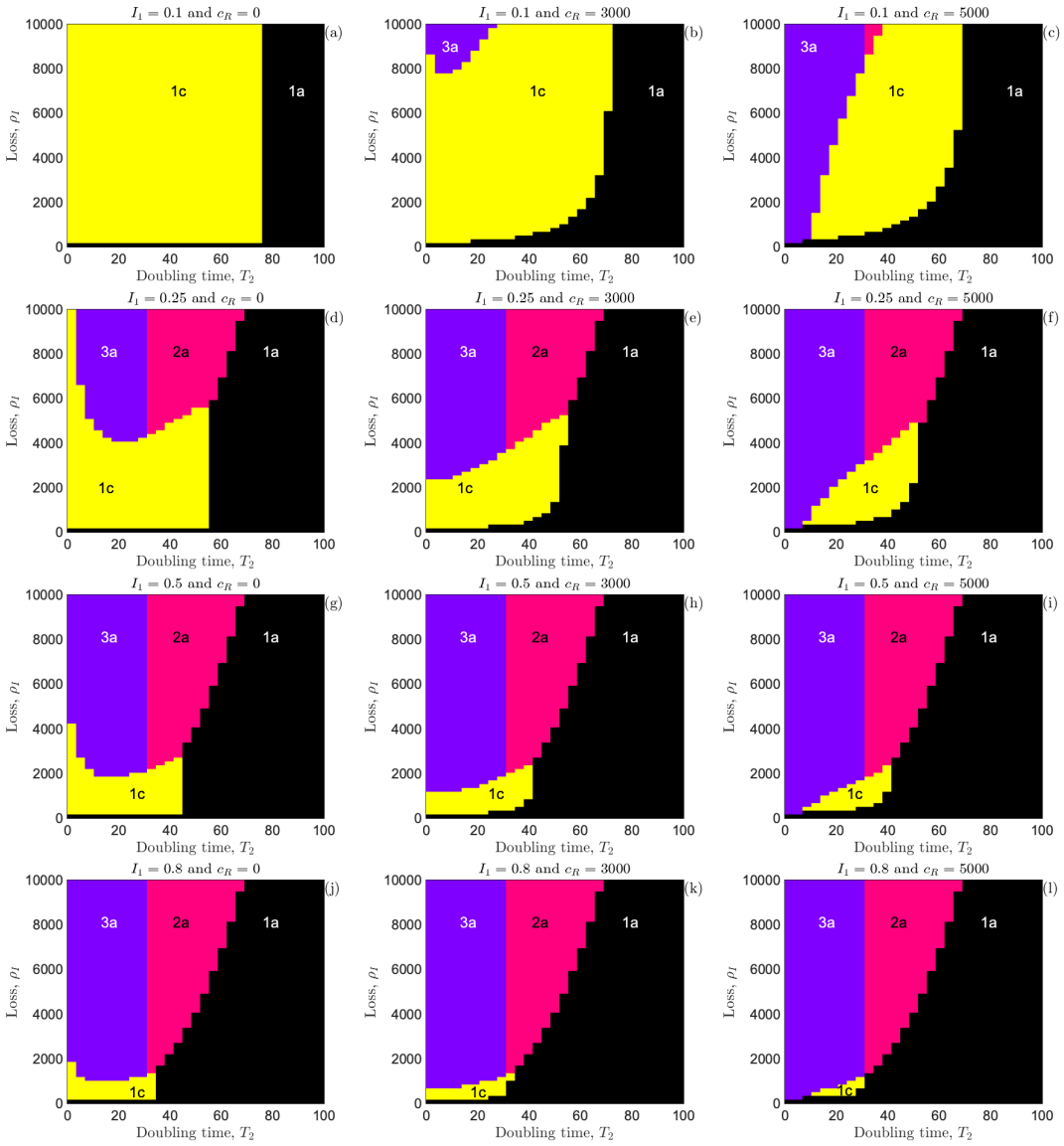

Figure 5.6: Sensitivity analysis to the detection threshold, I_1, and cost of the RM, C_r, when RM is available and optimised. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.9 and 5.10 as described in Table 5.2. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The detection threshold is, $I_1 = 0.1, 0.25, 0.5, 0.8$, and cost of the RM is, $C_r = 0, 3000, 5000$ as given on the title of each plot. All other parameters are at their baseline values in Table 5.1.

when the one-off cost of applying the RM is small (say, $C_r = 0$), the region where it is

optimal to wait and apply the RM is much larger. Increasing the one-off cost of applying the RM, C_r, or the detection threshold, I_1, will decrease the range of T_2 , P and ρ_I values where it is optimal to apply the RM only, and subsequently increase the parameter ranges where it is optimal to deploy the PM, either partially, zone 2b, or fully, zone $3a$ in Figure 5.6. Interestingly, when the one-off cost of applying the RM is small, it is optimal to apply the RM for an increase in ρ_I values as the doubling time, T_2 increases. This is the reverse when C_r is much bigger as seen in comparing Figure 5.6(d) and Figure 5.6(e).

As C_r becomes large the optimal management strategy tends to the strategy when the RM is unavailable (Figure 2.6 in the Chapter 3). A similar effect (a reduction in parameter range where it is optimal to only apply the RM, and a subsequent increase in parameter range where it is optimal to deploy the PM) is seen when the detection threshold, I_1, is increased (figure 5.6). This arises because a pathogen which spreads quickly will be detected quicker, thus the time horizon over which the loss is accrued will be much smaller but the net present cost of the applying the RM is larger (due to discounting). Therefore, when the cost of applying the RM is small the benefit of detecting the disease earlier and applying the RM earlier is larger. This highlights that the cost of waiting and applying the RM, or the ability to detect the pathogen early, will have a significant effect on the optimal management strategy that should be deployed.

Interestingly when $C_r = 0$, the range of values corresponding to the annual loss from disease, ρ_I increases as the doubling time, T_2, increases (Figure 5.6(d), (g), (j)); this is the reverse when C_r is much bigger (Figure 5.6(e), (h)(k)). This occurs because an increase in β means the pathogen is detected earlier in the time horizon, but the cost of applying the RM, C_r, is also realised earlier. When the cost of RM is low, it becomes worthwhile to detect the pathogen as early as possible, because an early deployment of RM can substantially reduce future infection damages at little expense. However, when the cost of applying RM is high, the benefits of early detection become less attractive: although early detection still reduces damages, the present value of the RM cost (once discounted over time) can exceed the corresponding reduction in damages. In this case, it can be more cost-effective to invest

in PM before detection occurs, as PM delays the onset of infection and therefore spreads the cost of RM and infection damages further into the planning horizon. Furthermore, the region where it is optimal to wait and apply the RM once the pathogen is detected is decreased (zone 1c in Figure 5.6). This highlights what time the loss from disease starts to accrue, t_{05}, is earlier in the time horizon it is more cost effective to deploy the PM and stop the pathogen (since $\delta_M = 0$), contrarily to waiting and treating the disease once it is detected (deploy the RM). This is because the closer t_{05} is to the start of the time horizon, t_0, the larger the total loss and the cost of applying the RM.

5.5.6 Sensitivity analysis with respect to the maximum reduction in primary infection due to the PM $(1-\delta_M)$

Sensitivity analysis with respect to the maximum reduction in primary infection transmission rate due to PM when RM is available and optimised. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.9 and 5.10 as described in Table 5.2. The black region (zone $1a$) shows where it is optimal to do nothing. The pink region (zone $2a$) is optimal to deploy partial PM. The purple region (zone $3a$) is optimal to deploy full PM. The yellow region (zone $1c$) shows where it is optimal to deploy RM only and the red region (zone $3c$) is full PM and RM. Parameter values which are different from their baseline values are given in the title of the plots in Figure 5.7, and all other parameters are at their baseline values in Table 5.1. Decreasing the maximum reduction in primary infection transmission due to the PM, $1-\delta_M$ (by increasing the parameter δ_M) means that the pathogen can never fully be prevented from arriving within the time horizon.

The PM and RM strategy are both effective in Figure 5.7(a), where the parameter $\delta_M = 0$, this can be seen from the region where it is optimal to deploy full and partial PM (zone $3a$ and $2a$) and RM region (zone $1c$). Decreasing the maximum reduction in primary infection transmission by only 5% (to $\delta_M = 0.05$), significantly reduces the parameter range where it

is optimal to deploy the PM (zones $2a$ and $3a$ in Figure 5.7(b)) and subsequently increases the parameter range where it is optimal to apply the RM (zone 1c in Figure 5.7(b)). When the maximum reduction in primary transmission from precautionary measures (δ_M) becomes very small (PM is no longer very effective), it is no longer worthwhile to invest in precautionary actions in advance. In such situations, two different policy responses emerge depending on the severity of the disease. If the disease spreads quickly (low T_2) and produces moderate to large damage, it becomes optimal to rely on RM and intervene only after detection, since PM would offer very little benefit. If the disease spreads slowly and the infection damages are very small, then even RM does not justify its cost and the best policy is simply to do nothing (i.e. accept the small damage rather than spending money on control). So the core insight for land manager is that precautionary measures are only justified when (i) they are reasonably effective, and/or (ii) the consequences of uncontrolled spread are high. Once PM becomes ineffective, resources should be shifted either to reactive strategies (if infection damages are significant) or to monitoring only (if infection damages are small).

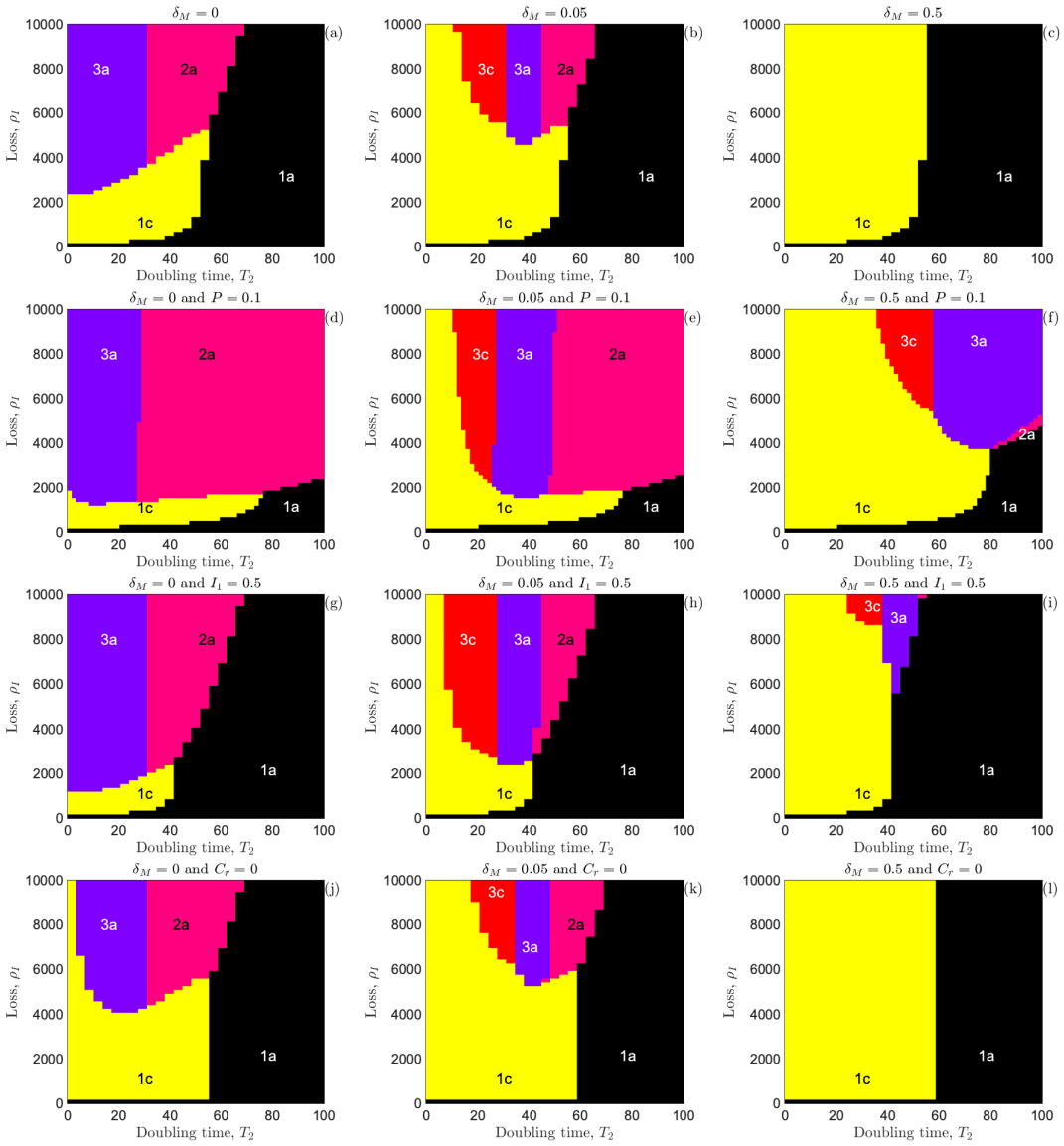

Figure 5.7: Sensitivity analysis to the maximum reduction in primary infection transmission rate due to PM when RM is available and optimised. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.9 and 5.10 as described in Table 5.2. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM and the red region (zone $3c$) is full PM and RM. The maximum reduction in the primary infection transmission due to the PM is (a),(d),(g),(j) $\delta_M = 0$, (b),(e),(h),(k) $\delta_M = 0.05$ and (c),(f),(i),(1) $\delta_M = 0.5$. Parameter values which are different from their baseline values are given in the title, and all other parameters are at their baseline values in Table 5.1.

When the baseline primary infection, P, is increased from the baseline value, which is $P = 0.005$ to $P = 0.1$, there is a reduction in the region where it is optimal to deploy the PM (see Figure 5.7$(d)-(f)$). When the detection threshold, I_1 is increased from the baseline value $I_1 = 0.25$ to $I_1 = 0.5$, the region in which it is most favourable to deploy the PM is reduced (see Figure 5.7$(g)-(i)$). However, when the cost of applying the RM, C_r, is decreased from the baseline value $C_r = 3000$ to $C_r = 0$ and the parameter δ_M is increased from $\delta_M = 0 \rightarrow 0.05 \rightarrow 0.5$ (see Figure 5.7$(j)-(I)$), there is reduction in region where it is optimal to deploy the PM. Moreover, we deploy PM and RM by decreasing the maximum reduction in primary infection transmission due to the PM (increasing δ_M) and increasing the detection threshold (see zone 3c in Figure 5.7(b)(e), (f), (h), (i), (k)). Hence, as $\delta_M \rightarrow 1$, the optimal management strategy generally changes from deploying the PM (zones $2a$ and $3a$) to applying the RM once the pathogen is detected (zone $1c$ in Figure 5.7(d) – (I)).

When the maximum reduction in primary infection transmission is slightly reduced (e.g. $\delta_M = 0.05$), a combination of both the PM and RM is optimal under some (limited) conditions. This generally occurs for pathogens with a shallow doubling time, T_2, and a high loss, ρ_I and when the baseline primary infection, P, is high (Figure 5.7(e)), or when the detection threshold, I_1, is large (Figure 5.7(h)). When P is high, the time that the loss from disease will start to accrue, t_{05}, and be detected, $\tau(\delta)$, will be closer to the start of the time horizon. Although the total loss is accrued over a small time period (e.g. when P is higher, $\tau(\delta) - t_{05}$ is smaller), the present value of the total cost will be significant (due to the cost of applying the RM and discounting). Therefore, a combination of deploying PM to delay the arrival of the pathogen (and the associated loss and cost of applying the RM) and applying the RM to stop the spread and loss is optimal. When I_1 is increased, in the absence of any control, the total loss is accrued over a more extended period (e.g. $\tau(\delta) - t_{05}$ is larger). Deploying the PM will reduce the loss over this period, and applying the RM will stop the spread and loss from $\tau(\delta)$ onwards.

5.5.7 Sensitivity analysis with respect to reduction in secondary disease transmission, ϵ, due to RM and reduced post-RM loss, ρ_{PI}

Sensitivity analysis with respect to the secondary disease transmission, ϵ and the post-RM loss, ρ_{PI}, when RM is available and optimised. The optimal management strategy is found by Equation 5.9 and 5.10 as described in Table 5.2. The plots, Figure 5.8, show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The value of the secondary disease transmission, ϵ are given as $\epsilon = 0, 0.5$ and 1 while the post-RM loss, ρ_{PI}, are given as $\rho_{PI} = 0, 0.5$ and 1. All other parameters are at their baseline value.

When the RM is not fully effective, it means that the RM does not reduce the loss caused by the disease; for example, when the post-RM loss is due to the disease, $\rho_{PI} > 0$,. The RM does not stop the pathogen from spreading further when the reduction in secondary disease transmission rate $\epsilon > 0$. We notice a decrease in the range of parameter values where it is optimal to wait and apply the RM (zone 1c) and an increase in the range of parameter values where it is optimal to deploy the PM (see Figure 5.8). More specifically, as the annual post-RM loss, ρ_{PI}, is increased (e.g. $\rho_{PI} \rightarrow \rho_I$), it becomes optimal to deploy the PM instead of waiting to apply the RM.

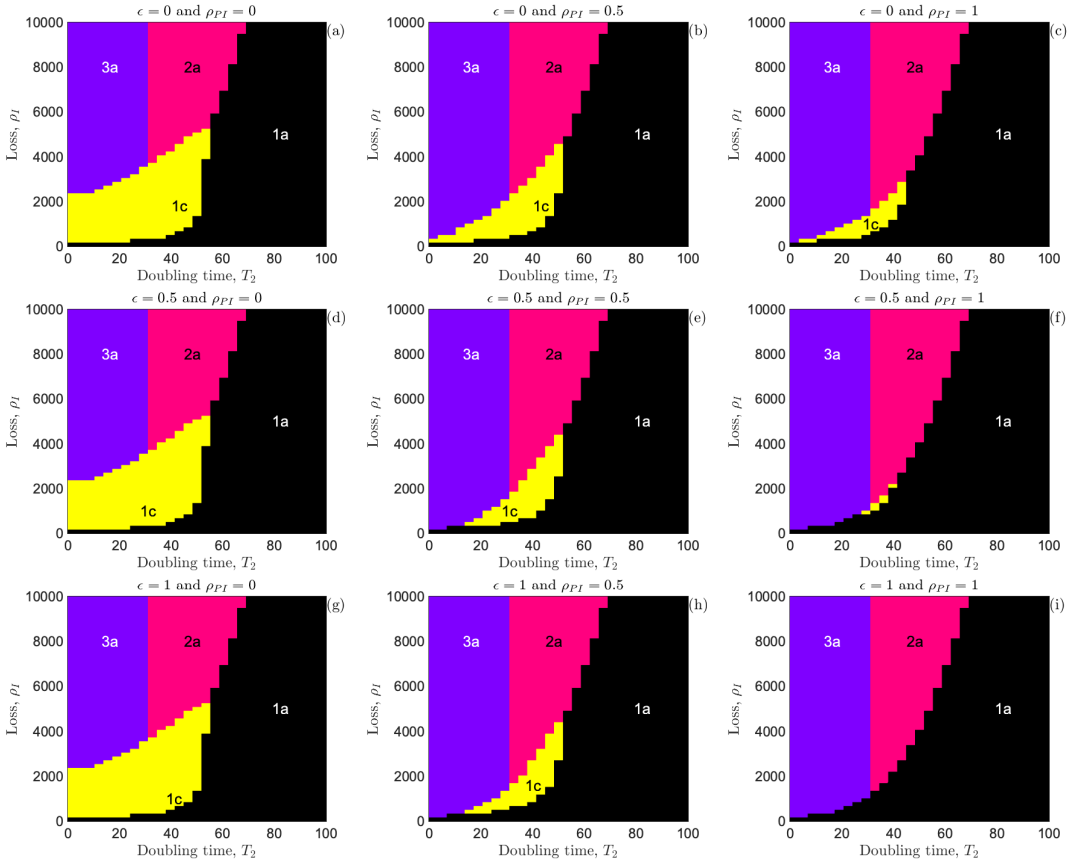

Figure 5.8: Sensitivity analysis to the secondary disease transmission, ϵ and the post-RM loss, ρ_{PI}, when RM is available and optimised. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.9 and 5.10 as described in Table 5.2. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The value of the secondary disease transmission, ϵ and the post-RM loss, ρ_{PI}, is given in the title of each plot. All other parameters are at their baseline value.

As we can see on Figure 5.8$(a)-(c)$ where the post-RM loss, ρ_{PI} is increased from $\rho_{PI} = 0 \rightarrow 0.5 \rightarrow 1$,.The zone $1c$ (yellow region) gradually disappears, which shows that the higher the ρ_{PI}, the more likely we do not require the RM strategy. Interestingly, when the reduction in secondary disease transmission due to the RM, ϵ, is increased, there is no much changes on the region where it is optimal to apply the RM (see zone $1c$ in Figure 5.8$(a),(d),(g)$). This highlights that the effect of the RM on reducing the annual loss post-application is considering

the optimal management strategy; possibly more so than the RM ability to reduce the disease transmission. The optimal management strategy is more sensitive to the effect of RM on reducing annual loss after application, ρ_{PI}, compared to the ability of the RM to reduce disease transmission.

5.6 Stochastic result of the precautionary and reactive management.

When introducing a random element to any management framework, uncertainties will play a crucial role in determining the optimal disease management strategy. Introducing stochasticity into the Precautionary Management (PM) and Reactive Management (RM) strategy significantly affects its design, application, and effectiveness. Here are the main impacts of stochasticity on the RM: The model would need to consider random variables into the existing deterministic values. land manager aims to identify the optimal level of PM that minimises the expected present value of disease-related costs over a fixed time horizon. This objective is pursued with the understanding that there is uncertainty as regards when the pathogen will be detected and that an RM strategy will be implemented probabilistically upon detection. Due to uncertainties, land manager will employ a probabilistic approach in determining whether to apply RM to the entire forest, only to detected infected areas, or to a buffer zone around detected infections. RM is expected to reduce the rate of spread of the pathogen and is also expected to decrease the expected loss of the disease, given the variability.

After deploying RM, the expected loss over the remaining time horizon, the expected yearly post-RM loss, might vary based on the uncertainties and is considered less than or equal to the expected annual loss before RM is applied. In scenarios where the RM strategy reduces the disease's effects but does not eliminate them, there is a probabilistic expectation that the disease could still spread, but at a potentially reduced rate. Given the uncertainties introduced by the stochastic element, the decision-making process would revolve around an expected objective function that computes the expected present value of the total cost, incorporating the random variables or distributions for the respective parameters.

When the RM strategy is available and optional with the primary infection, P, changed to a random element, the optimal management strategy is the optimal level of PM, c^*, c^{**}, and whether it is optimal to implement RM (off / on). We show the optimal disease management

effects required to reduce the present value of the costs incurred in managing the impact of invasive species on the forest over the time horizon. We do nothing when no damage is caused to the forest under investigation. Therefore, we label this "None". The optimal level of PM will be zero ($c^* = 0$ and $c^{**} = 0$), which is the management strategy 1a (black), 1b (dark blue) and 1c(yellow) in Table 5.3. The black region (zone 1a) shows when the optimal level of PM is zero and RM is not available. The pink region (zone 2a) is when the optimal level of PM is between zero and the maximum budget, but RM is not available. The purple region (zone 3a) is when the optimal level of PM is equal to the full budget and RM is not available. The dark blue region (zone 1b) is when the optimal level of PM is zero, RM is available, but not deployed (because the pathogen arrived after the time horizon, $\tau(\delta) > T$). The turquoise region (zone 2b) is when the optimal level of PM is between zero and the maximum budget, but RM is available and is not deployed (because the pathogen arrived after the time horizon, $\tau(\delta) > T$. The yellow region (zone 1c) shows when the optimal level of PM is zero and RM is available and deployed. However, no damage was caused when the pathogen was detected within the time horizon ($\tau(\delta) \leq T$).

Optimal management strategy	PM (optimised)	RM (Off/On)	RM(No/Yes)
1a (black)	None, $c^* = 0$	Off	No.
2a (pink)	Partial, $0 < c^* < \Pi$	Off	No.
3a (purple)	Full, $c^* = \Pi$	Off	No.
1 b (dark blue)	None, $c^{**} = 0$	On	No, $\tau(\delta) > T$.
2b (turquoise)	Partial, $0 < c^{**} < \Pi$	On	No, $\tau(\delta) > T$.
1c (yellow)	None, $c^{**} = 0$	On	Yes, $\tau(\delta) \leq T$.

Table 5.3: The management strategies considered when the RM is optional.

5.6.1 Log-normal distribution

Sensitivity analysis to annual loss, ρ_I, and doubling time, T_2

The log-normal distribution is determined by the parameters μ and σ, which are derived from a given mean and variance. The model then draws 1000 samples from this distribution to represent the possible values of P. By sampling 1000 different values for the primary invasion rate (P) from the log-normal distribution, the model introduces uncertainty in how the invasion might proceed. This is a way to account for the variability and unpredictability of real-world invasion processes. The code runs simulations for each of these sampled values of P. For each P value, the model calculates the time of invasion detection, the time damage begins to accumulate, and the total losses both with and without a reactive management (RM) strategy. These total losses are then averaged across all stochastic realizations of P to determine the expected total loss for a given level of control and other parameters. The stochastic component in this model is introduced by considering the primary invasion rate (P) as a random variable following a log-normal distribution. In doing so, the model captures the inherent uncertainty and variability of invasion processes, providing a more robust prediction of the optimal control strategy.

Numerical simulation is used to evaluate the total losses due to an invasion under two conditions, with and without applying RM and for various levels of PM. The plots in Figure 5.9 analyse the impact of varying the variance in the primary invasion rate (P) on annual loss and doubling time for different management strategies: precautionary management (PM) and reactionary management (RM). The variance values represent the uncertainty in the primary invasion rate. A higher variance indicates a greater range of potential invasion rates, while a lower variance implies more certainty about the rate. The plots display the relationship between the annual loss (ρ_I) on the y-axis and the doubling time (T_2) on the x-axis. Different colour regions represent different outcomes of the precautionary and reactionary management strategies. The regions are labelled ($1a$, 1b, $1c$, $2a$, $2b$ and $3a$) and are associated with different colours, the details of which are provided in table 5.3. The plots are categorised

based on different variance levels, as shown in Figure 5.9: (a) variance = 0 (b) variance = 0.00001 (c) variance = 0.0001 (d) variance = 0.001 (e) variance = 0.01 (f) variance = 0.1. The results shown in Figure 5.9 indicate the rate at which some quantity doubles in size. Higher values indicate a slower doubling in size and lower values indicate faster doubling in size. Higher values indicate a greater annual loss. The plot has been divided into different coloured regions, each representing a different management strategy. This includes zones $1a$, $2a$, and $3a$ (Black, Pink, and Purple, respectively) for a specific PM condition. Reactionary management is shown as zones 1b, 2b (Dark blue, Turquoise) and $1c$ (Yellow) for another condition of PM associated with the RM.

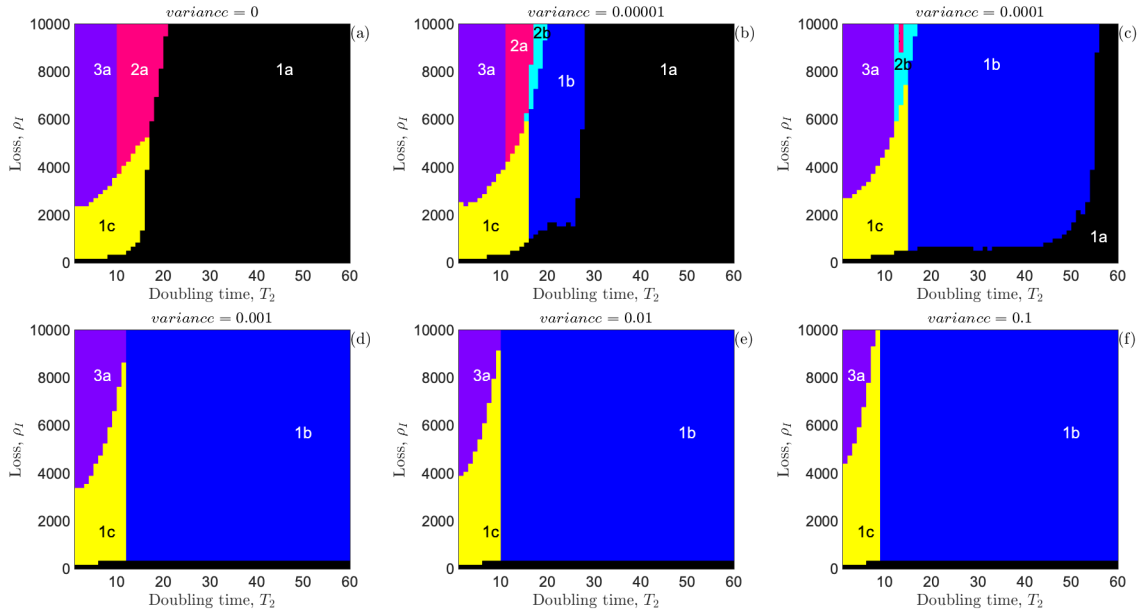

Figure 5.9: Sensitivity analysis to annual loss, ρ_I, and doubling time, T_2, using log-normal distribution on precautionary management and reactionary management strategy. The outcome of the PM and RM as give in Equation 2.2 with default to probability on arrival with time. The probability of arrival was carried out at different variance level in Figure(a-f): (a) $variance = 0$, (b) $variance = 0.00001$, (c) $variance = 0.0001$, (d) $variance = 0.001$, (e) $variance = 0.01$, and (f) $variance = 0.1$. The Figure is showing one thousand (1000) values of randomly choosing P. The colours in the figures are detailed in table 3.1. All other parameters are at their baseline values in tables 2.1.

The plots are separated based on the variance of the log-normal distribution, ranging from 0 to 0.1. Variance measures how spread out the values are from the mean. As variance

increases, there is a noticeable shift in the management strategies' regions. At variance = 0, the strategy $1a$ (black) covers a large part of the plot. However, as the variance increases, its region decreases significantly. The region for $3a$ (Purple) expands as the variance increases from 0 to 0.0001 but then disappears entirely at higher variances. The $1c$ (Yellow) strategy becomes dominant at higher variances. Doubling time, T_2, does not show significant variance-specific trends. However, for larger T_2 (slower doubling rates), the $1a$ and $1b$ strategies become dominant, regardless of the variance. As the annual loss increases, the preferable management strategy changes; for low yearly losses, the $1c$ strategy is preferred, while for moderate losses it shifts to $3a$ or $1b$, depending on the variance. For very high annual losses, the strategy $1a$ becomes dominant. The optimal management strategy varies according to the annual loss and the variance in the log-normal distribution. For land manager, this indicates the need to consider the variability in predictions or measurements when deciding on a management strategy.

The RM model's sensitivity to variance in the log-normal distribution highlights the importance of understanding the inherent variability or uncertainty in the studied processes. Different management strategies become optimal depending on the variance, underlining the necessity of adaptive and flexible policy making. The analysis can guide land manager in selecting the most suitable management strategy based on the expected annual loss and the reliability or variability of the data at hand. From a broad perspective, the plots help decision maker understand how uncertainty in the primary invasion rate (represented by variance) can impact the effectiveness of different management strategies. The plots allow decision maker to make informed decisions on which strategy to adopt based on acceptable levels of annual loss, double time, and the level of uncertainty in the invasion rate.

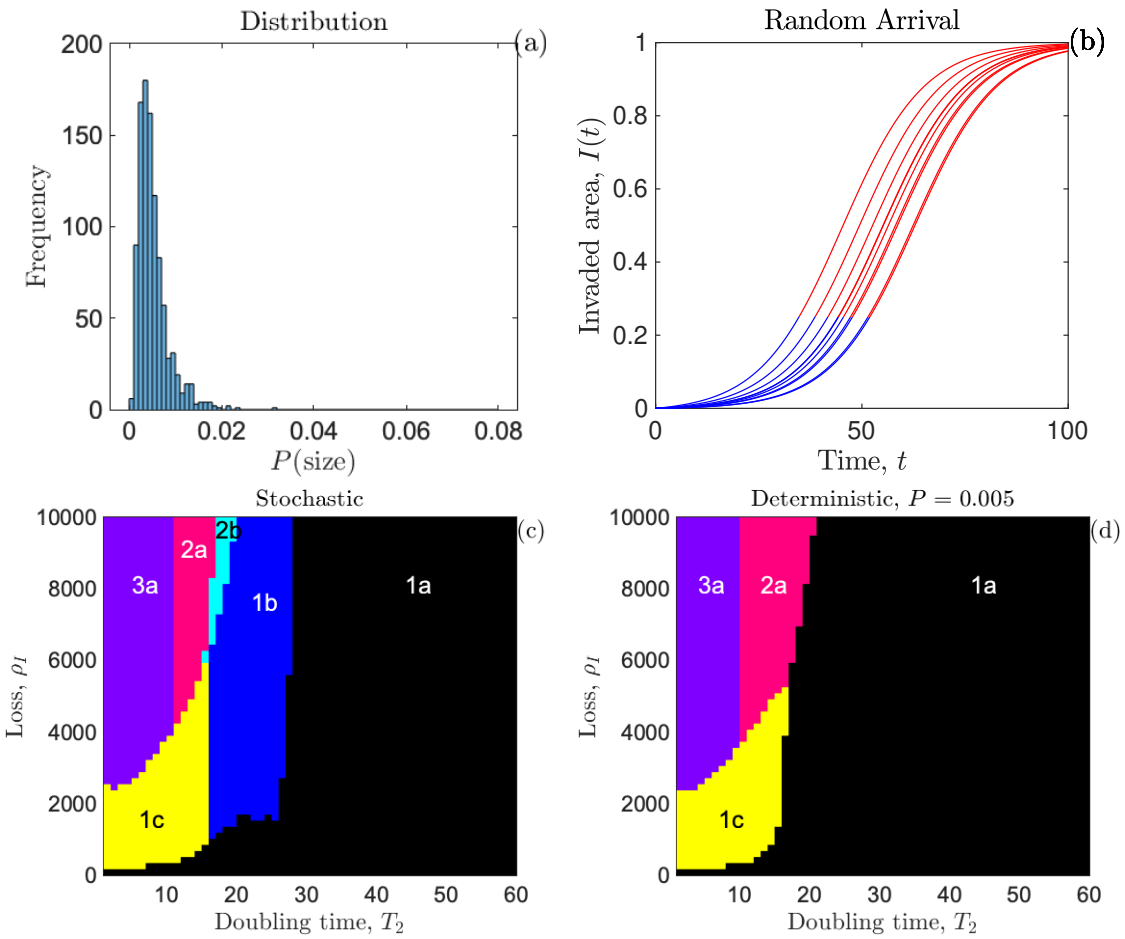

Figure 5.10: Different types of plots with log normal Distribution when the variance is 0.00001 (a) The distribution (b) 10 Trajectories (c) The stochastic plot and (d) The deterministic with the same P value.

The four graphs presented in Figure 5.10 are results of a scientific study on the stochastic process and distributions with a focus on the log-normal distribution with a specified variance of 0.0001 compared to the deterministic graph. Figure 5.10 (a) shows the result, which is the distribution of sizes, revealing the skewness toward smaller values when the variance is 0.0001. The x-axis represents the P size (ranging from 0 to just under 0.08), and the y-axis represents the frequency of occurrences. The data appear to be positively skewed, meaning that there is a tail on the right side of the distribution with more frequent occurrences of smaller sizes and fewer occurrences as the size increases. This plot in Figure 5.10 (b) illustrates ten trajectories

over time. The x-axis represents time t, ranging from 0 to 100, and the y-axis represents the invaded area, $I(t)$, which is a function of time and describes the spread process. Each trajectory represents a path the process takes under random variation. These values were plotted to show how the 'invaded area' changes over time, with different trajectories showing the variability due to randomness. If there were little to no variability, all the trajectories would be similar or overlap significantly, indicating a predictable process. High variability results in a wide spread of potential outcomes, which signifies uncertainty in the invaded area over time, and the variability in trajectories shows how the same process can lead to different outcomes due to random events or fluctuations.

The heat map plot in Figure 5.10 (c) represents the loss function with respect to doubling time on the x-axis (ranging from 0 to 60) and the loss function on the y-axis (ranging from 0 to 10,000). The different colours ($1a$, $1c$, 1b, $2a$, 2b, $3a$) represent different zones of this loss function, indicating regions where certain behaviours are observed in a stochastic model. A process with low variability has a more uniform colouration (less variation in loss across doubling times). However, high variability leads to a more colourful plot with distinct regions, indicating that the loss varies more dramatically with changes in doubling time due to the random elements of the process. This plot in Figure 5.10 (d) is similar to Figure 5.10 (c) but is a deterministic result which suggests that it shows a system without randomness as opposed to the stochastic system in Figure 5.10 (c). The same range of values is used for the axes and similar color coding is seen, although the regions are labelled differently ($1a$, $1c$, $2a$, $3a$), implying different behaviours within this deterministic framework.

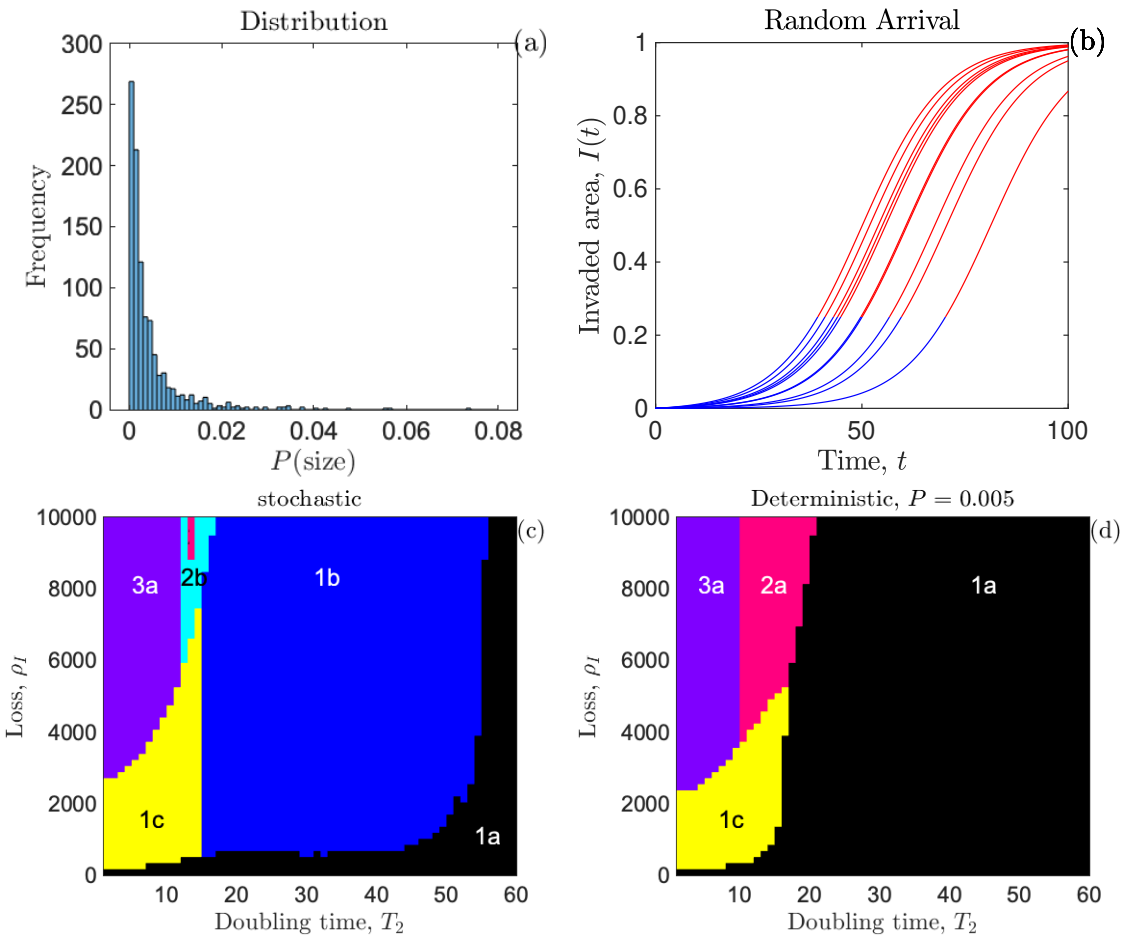

Figure 5.11: Different types of plots with log normal Distribution when the variance is 0.0001 (a) The distribution (b) 10 Trajectories (c) The stochastic plot and (d) The deterministic with the same P value.

The distribution plot in Figure 5.11 (a) is a histogram showing the frequency distribution of P values. It is a representation of a log-normal distribution in which most of the data points are concentrated around a particular size, with a tail extending to the larger P sizes. Most data concentrate around P size of 0.02-0.04 with a peak frequency around 175-200. The distribution has a positive skew as it tails off to the right. Random arrival plot is shown in Figure 5.11 (b), the plot depicts the invaded area as a function of time, with multiple trajectories which illustrate the variation in how quickly an area gets invaded based on different starting conditions or external influences. The y-axis represents the "Invaded area," indicating the

spread of an invasion process over the "Time, t" given on the x-axis. The blue trajectories seem to start strong and then slow down as time progresses, while the red trajectories show a slower start but faster growth over time. In Figure 5.11 (d), the stochastic heat map plot represents different regions ($1a$, $2a$, $2b$, $3a$, 1b, $1c$), which means various management strategies under stochastic conditions. The x-axis indicates the doubling time, T_2, and the y-axis represents the loss, ρ. The deterministic plot in Figure 5.11 (d) is similar to (c), but is fixed at $P = 0.005$, which implies that it is based on fixed parameters or conditions, as opposed to (c), which is stochastic.

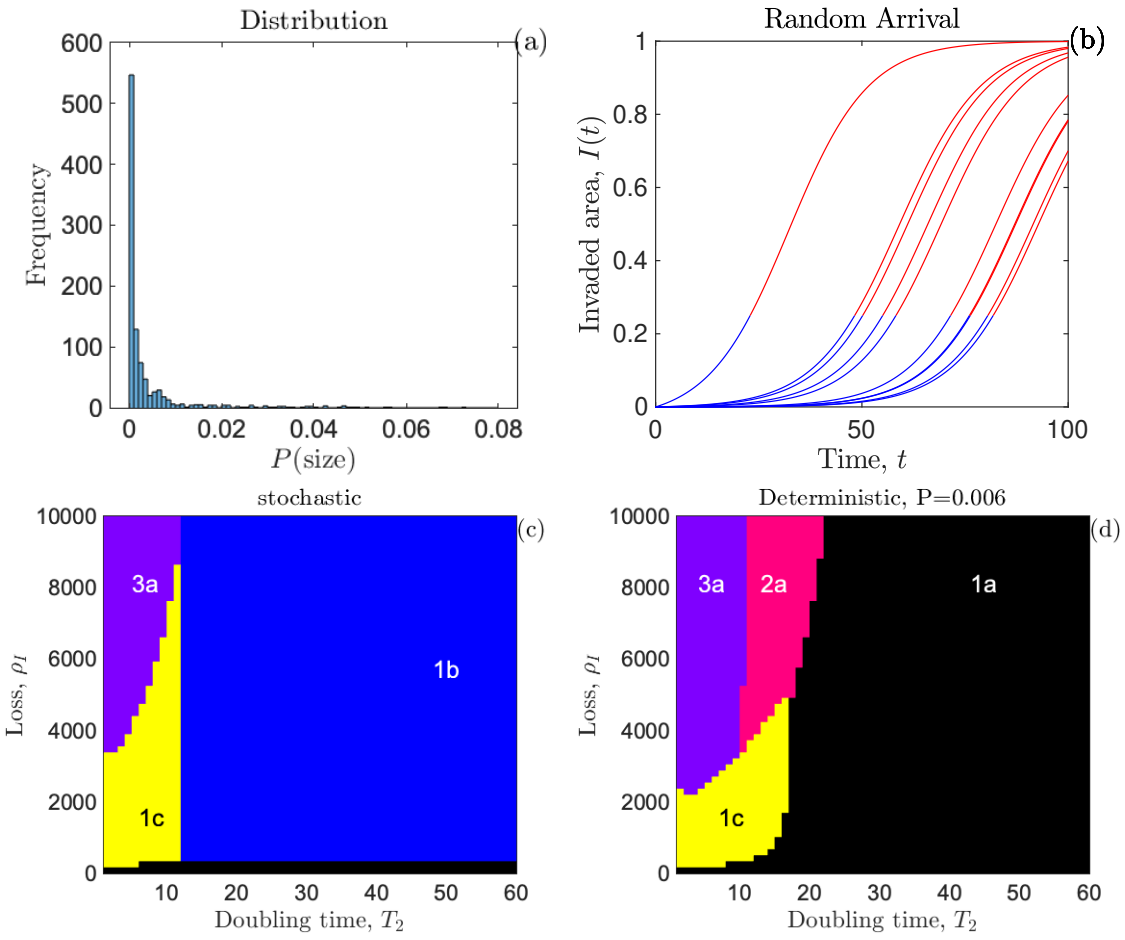

Figure 5.12: Different types of plots with log normal Distribution when the variance is 0.001 (a) The distribution (b) 10 Trajectories (c) The stochastic plot and (d) The deterministic with the same P value.

In Figure 5.12 (a),the x-axis represents the P size of the distribution, whose values range from 0 to 0.08. The y-axis represents the number of occurrences for each size interval. The frequency peaks slightly above 0.02 and decreases sharply as the size increases. The multiple line plots in Figure 5.12 (b) represent the area of the forest that has been invaded over time, ranging from 0 to 100. The proportion of pathogen arrival and spread is shown on the y-axis, with values ranging from 0 to 1. In Figure 5.12 (c), the time it takes for a pathogen to arrive and spread to double is represented on the x-axis. It ranges from 0 to 60, while the loss on the y-axis ranges from 0 to 10000. Depending on the management strategy "1a" as indicated in the provided table (see Table 5.3), black (zone 1a) means No optimal management, RM is off, and RM is not applied ($c^* = 0$). The dark blue strategy shown by "1b" means No optimal management, RM is on, and RM is not applied due to the late infection detection time ($\tau(\delta) > T$). Relates to turquoise colour, strategy "2b", no optimal management, RM is on, but RM is not applied due to the infection detection time being more significant than the time horizon ($\tau(\delta) > T$). The yellow colour represents the strategy "1c", no optimal management, RM is on, and RM is applied because the infection detection time is less significant than the time ($\tau(\delta) \leq T$). By assigning distinct colours to each strategy, the plots allow for a clear and immediate comparison of how each strategy performs under varying conditions. The Deterministic Plot Similar to (c), it represents the time required for some quantity to double. The range is from 0 to 60. Again, similar to (c), it represents some loss, ranging from 0 to 10000. The colour scheme is the same as in the stochastic plot (c), but the regions corresponding to each strategy are different due to the deterministic nature of this plot.

Variability across different variances showcases the importance of understanding and accounting for uncertainties in modelling and decision-making. The different colour-coded regions in each plot suggest different outcomes under the two management strategies (see Table 5.3). Higher variances represent more uncertainty in the invasion rate. Under certain variances, reactionary management is riskier due to unpredictability, while precautionary management could provide more consistent outcomes. The plots can serve as a tool for land manager. Although the plots provide a structured way to understand the outcomes

under different conditions, real-world scenarios might introduce additional complexities not captured in the model. For further exploration, it would be interesting to see how other parameters, not just variance, impact the outcomes. In addition, integrating economic, ecological, or social factors might provide a more comprehensive perspective.

5.6.2 Absolute values from a normal distribution

The absolute values from a normal distribution, often referred to as a half-normal distribution, retains only the positive half of the normal distribution while reflecting the negative half onto the positive side. When the model is used for decision-making, particularly in optimisation and control strategies, the absolute value transformation will impact the selection of optimal strategies. For example, the cost functions are based on deviations from a certain target and these deviations are modelled with a half-normal distribution, the model suggest different strategies for risk management, taking the absolute values from a normal distribution significantly alters its properties and, consequently, any models that incorporate it. When such a transformation is applied within a model, it typically reflects specific constraints or assumptions about the underlying variables and influences the outcomes and interpretations of the model.

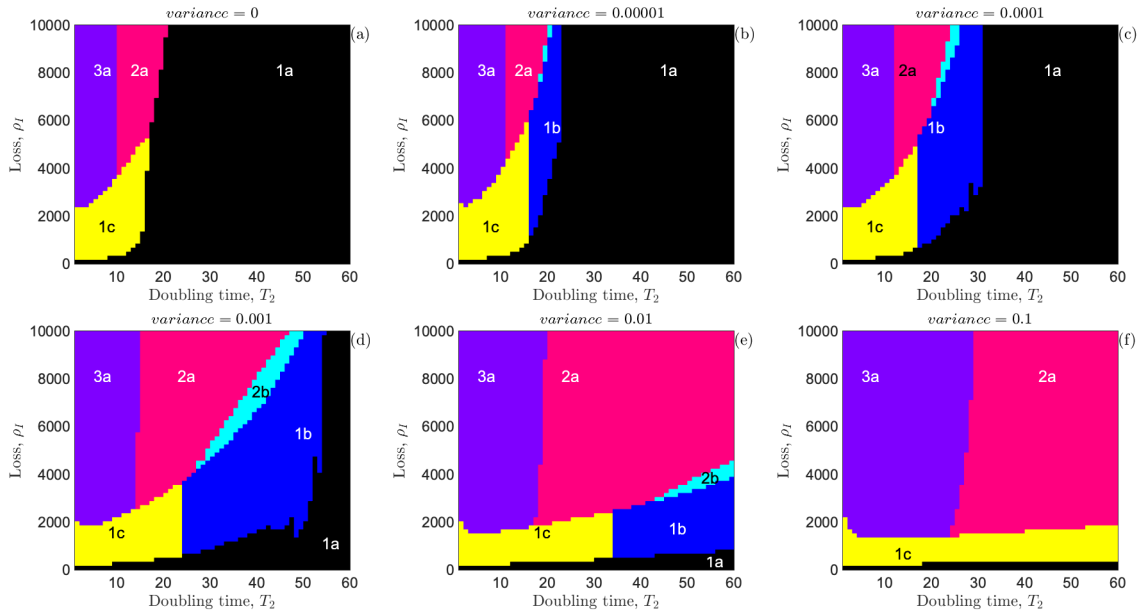

Figure 5.13: Sensitivity analysis to annual loss, ρ_I, and doubling time, T_2, using absolute values from a normal distribution. The outcome of the PM and RM as give in Equation 2.2 with default to probability on arrival with time. The probability of arrival was carried out at different variance level in Figure(a-f): (a) $variance = 0$, (b) $variance = 0.00001$, (c) $variance = 0.0001$, (d) $variance = 0.001$, (e) $variance = 0.01$, and (e) $variance = 0.1$. The Figure is showing one thousand (1000) values of randomly choosing P. The colours in the figures are detailed in table 3.1. All other parameters are at their baseline values in table 2.1 and 2.1.

The plots in Figure 5.13 are a set of sensitivity analysis graphs that show how the annual loss, ρ_I and the doubling time, T_2 are affected by the absolute values from a normally distributed random variable, under different levels of variance. This analysis is relevant to understanding the results of the PM and RM strategies. Doubling time represents the time it takes for a loss to double in magnitude, while annual loss represents the yearly financial losses. Each colour in the plot corresponds to a different management strategy, as detailed in a table 3.1. These strategies range from 'None' (no action taken) to 'Full' (full implementation of PM and RM). The plots (a-f) show how increasing the variance of the normally distributed variable affects the distribution of outcomes in terms of ρ_I and T_2. As variance increases, the spread of the risk outcomes becomes wider, implying greater uncertainty and unpredictability in the potential annual loss. The plots are based on a model that defaults to the probability

of disease arrival over time, which is random and is modelled by a normally distributed variable with absolute value. Figure 5.13 indicates that 1000 random values of P were chosen to generate these graphs, which gives a stochastic modelling aspect to the analysis.

At zero variance, the outcomes are predictable and not spread out, which corresponds to a deterministic model. The black region (1a strategy) shows that is optimal to the expected low loss and the risk evolves slowly at high T_2, making PM and RM unnecessary. As variance increases, the region where this strategy is optimal decreases, indicating less certainty about low risk. The dark blue region (1b strategy) suggests activating RM only after detection of the risk. It is chosen when the loss is moderate, but the time required to double is longer than the critical threshold ($\tau(\delta) > T$). This strategy becomes more prevalent with a slight increase in variance, reflecting a moderate level of uncertainty. The yellow region (1c strategy) RM is applied immediately because the doubling time is within a critical range. This strategy is chosen when the risk evolves rapidly and there is a high chance of it occurring within the time horizon. It is optimal in high-risk, high-urgency scenarios, especially at lower variances. In the pink (2a strategy) and turquoise (2b strategy), these strategies involve applying PM to some extent but not fully. The difference between 2a and 2b is the application of RM; it is off in 2a and on in 2b. This partial approach becomes more optimal as the variance increases, suggesting that, as uncertainty grows, a more cautious and proactive approach is warranted. The purple strategy (zone 3a) involves implementing PM at full capacity, which is chosen when the expected loss is high enough to justify a complete PM investment, but RM is not activated, possibly due to a slower evolution of risk T_2 or a high threshold for activation of RM measures. As the variance increases, the transitions between different strategies become more gradual, and the regions where partial strategies (2a and 2b) are optimal become more prominent. This indicates that as uncertainty increases, there is a shift toward more proactive management strategies.

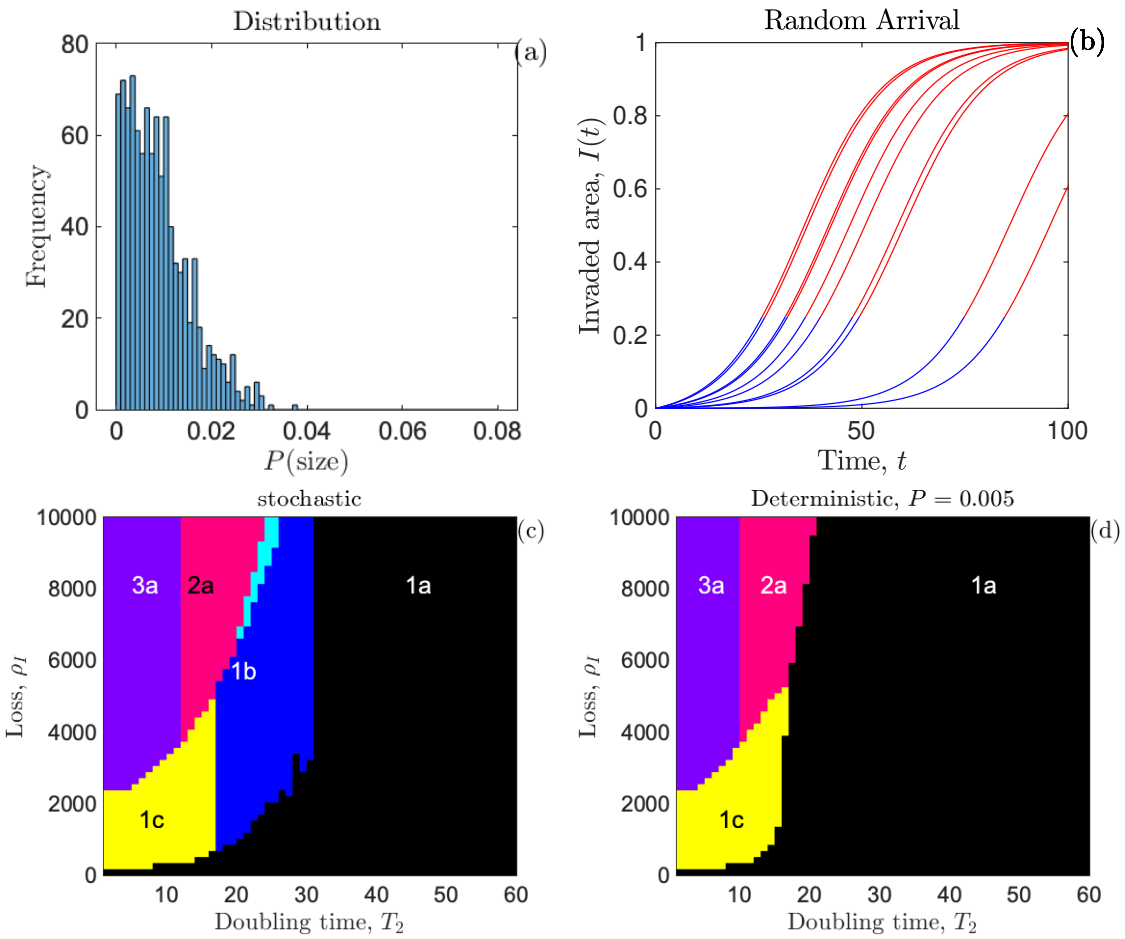

Figure 5.14: Different types of plots with absolute values from a normal distribution when the variance is 0.0001 *(a) The distribution (b)* 10 *Trajectories (c) The stochastic plot and (d) The deterministic with the same P value.*

The four different graphs in Figure 5.14 show the result of PM and RM using the absolute values from a normal distribution when the variance is 0.0001. Each plot provides a different perspective on the distribution and its implications for the model within the context of loss management that involves stochastic processes. The distribution in Figure 5.14(a) is skewed toward the left, indicating that higher P values are less frequent. The graph shown in 5.14(b) has ten individual trajectories representing the progression of an invaded area over time for different realizations of a stochastic process. The trajectories visually represent how the invaded area evolves over time under different stochastic realisations. The heat map plot in

Figure 5.14(c) indicates different management strategies, which are determined based on the combination of annual loss and doubling time under stochastic conditions. However, the plot provides a way to visualise how management strategies will change when accounting for inherent randomness in the system using the absolute values from a normal distribution with variance equal to 0.0001. The deterministic plot in Figure 5.14(d) offers a comparison point to see how strategies would differ in a world without stochastic variability, where the outcomes are predictable and certain.

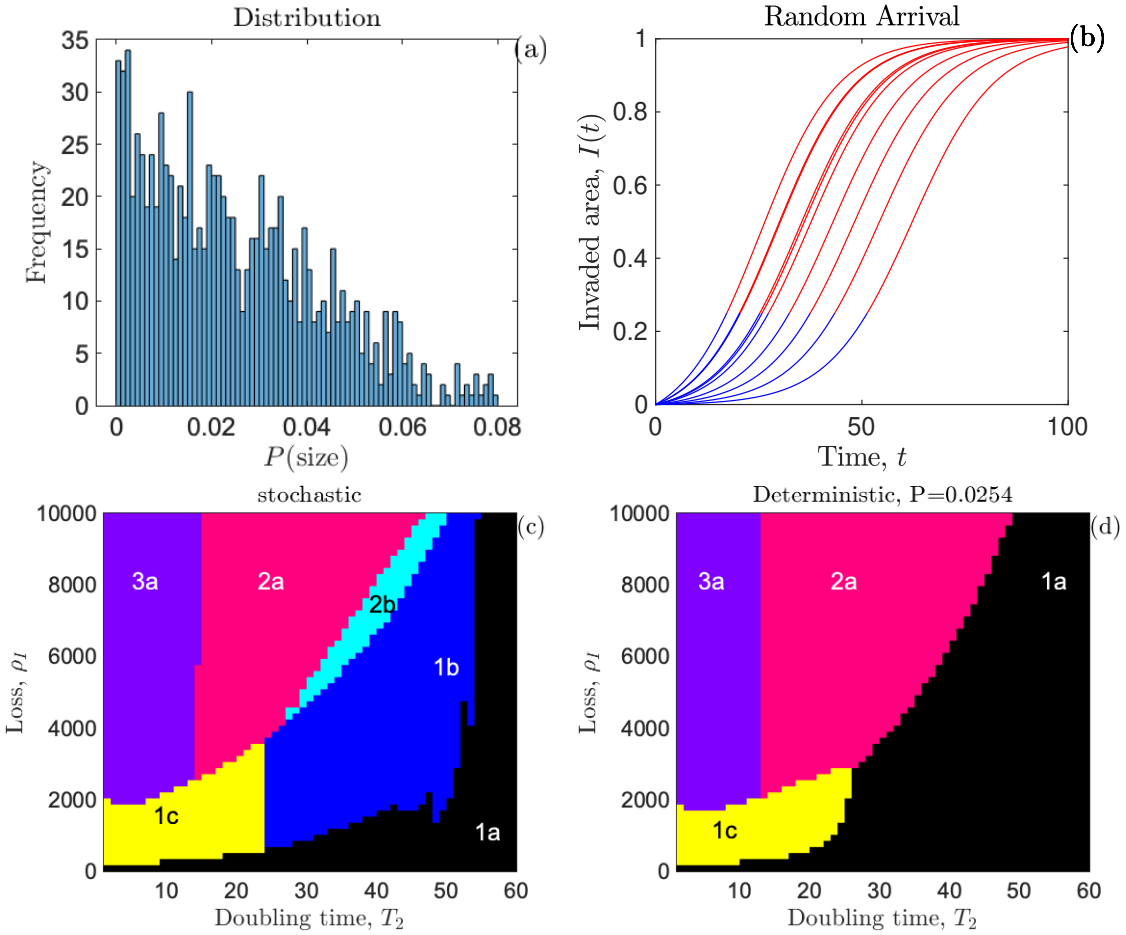

Figure 5.15: Different types of plots with absolute values from a normal distribution when the variance is 0.001 (a) The distribution (b) 10 Trajectories (c) The stochastic plot and (d) The deterministic with the same P value.

The plots in Figure 5.15 describe different types of outcomes with an absolute values from

a normal distribution when the variance is 0.001. The difference between Figure 5.15 and the previous result, Figure 5.14, appears to be in the specific values of probability P and variance, which affects the distribution and the zones of the management strategy resulting.

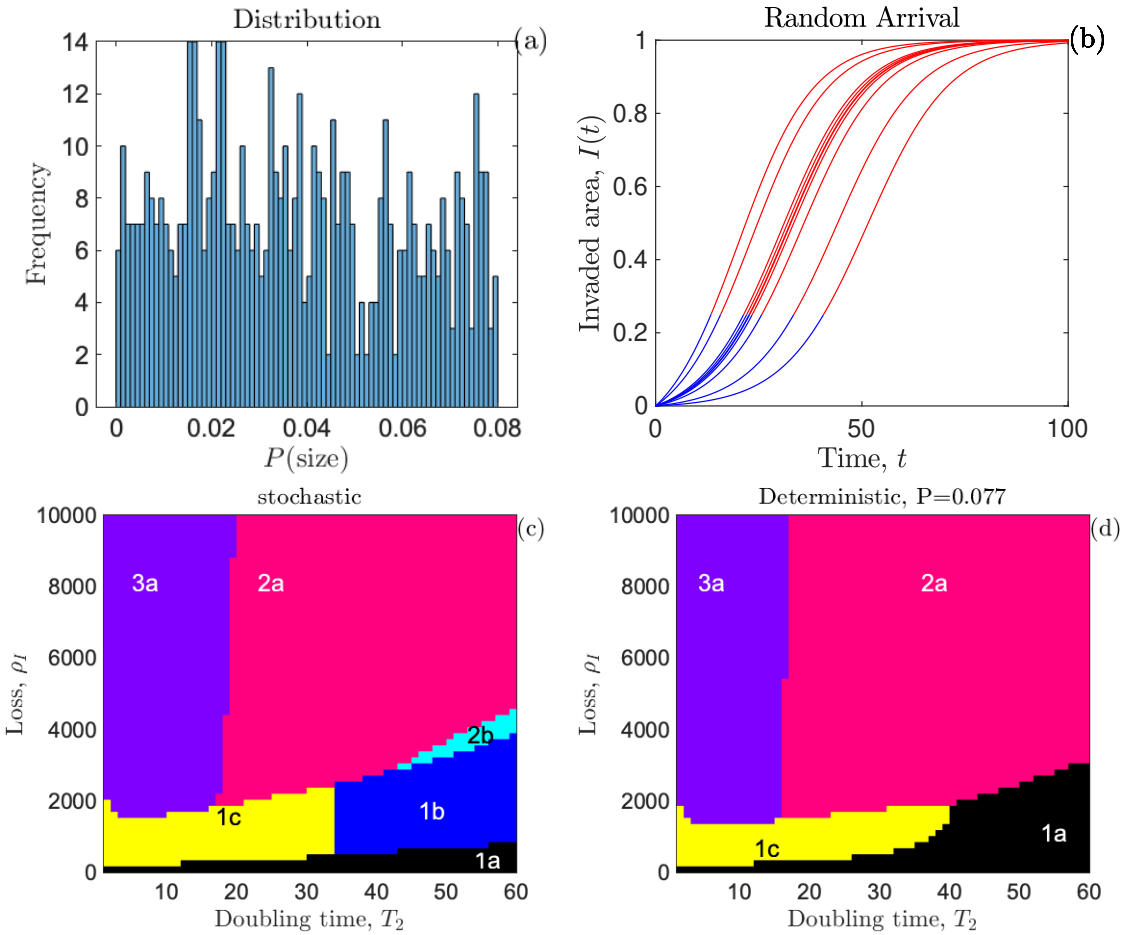

Figure 5.16: Different types of plots with absolute values from a normal distribution when the variance is 0.01 (a) The distribution (b) 10 Trajectories (c) The stochastic plot and (d) The deterministic with the same P value.

In the distribution plot Low variability would result in most data points clustering around a central value (peak), while high variability would spread the data across a wider range of values, which will lead to a flatter distribution or multiple peaks. The colours in Figure 5.16 (c) and (d) represent different management strategies that are appropriate under varying conditions of loss and doubling time. The yellow region in the stochastic plot represents a

strategy where no PM is applied (PM is 'None'), and RM is immediately necessary due to the high probability of the loss occurring within the time horizon. The strategy in yellow indicates urgent action is needed, and the costs of inaction are higher than the costs of implementing RM. The yellow region (zone 1c) in the deterministic plot is smaller compared to the stochastic plot, suggesting that under deterministic assumptions, the conditions requiring immediate RM are fewer or more clearly defined.

The blue region (zone 1b) in Figure 5.16 (c) indicates a strategy where RM is on standby, activated only upon detection of the loss. It occupies a transitional space between immediate action strategies and less active strategies, reflecting a moderate level of risk. The pink region (zone 2a) and turquoise region (zone 2b) in the stochastic plot represent strategies of partial PM, with turquoise (2b) indicating the presence of an RM strategy. The size and position of these zones in the stochastic plot show that there is a range of conditions under which a balanced approach to PM and RM is appropriate. The deterministic plot does not show the turquoise region strategy but the pink region strategy, which imply that when primary infection rates are certain and fixed, a partial approach is not considered optimal. The purple region (zone 3a) signifies a strategy of full PM with no RM applied. It is a proactive approach adopted when the expected loss is significant but manageable without active risk management. In the deterministic plot, the purple zone suggests that when primary infection are fixed and known, investing fully in PM without additional RM is often the best strategy. The implication of the colours in these plots is significant as they visually encode the strategic response to risk based on the severity of the loss and urgency with depends on the doubling time. The presence and size of these coloured zones across both plots highlights how the introduction of variability and uncertainty can dramatically shift the decision-making landscape from clear-cut strategies to more exact ones that require careful consideration of stochastic elements.

5.7 Discussion

We developed a comprehensive model for understanding the dynamics of disease spread and the RM management strategies within forest under investigation while the Chapter 3 presented the mathematical groundwork. The RM strategy is analysed in terms of when it should be deployed (i.e. the optimal timing), while assuming that the level of RM is fixed and applied fully once the infection is detected. This chapter discusses the importance of identifying the optimal point at which to invest in PM, i.e., the level of disease risk or detection threshold that minimises the present value of total disease related costs over the planning horizon. The observations made by [75] reinforce our conclusions, as they reveal an augmented expenditure on precautionary and control management strategies with higher invasion growth rates. In [61, 143], implementing disease control measures and farmers' behaviours influence the health of plants and livestock, depending on the strategies individuals choose to apply during disease outbreaks. Therefore, this study investigated how best to identify the best management strategy for farmers towards the handling of disease outbreaks.

In this chapter, we did sensitivity analysis, supported by numerical simulations, which provide a clear illustration of heat maps, demonstrating the conditions under which the costs of PM align with the losses incurred from the pathogen. Using a numerical approach, we can explore how different levels of infection damage affect overall costs and identify the most cost-effective investment in precautionary management. The analysis carried out in this chapter is to find the optimal management strategy with a focus on loss sensitivity, rho_I, doubling time, T_2, primary infection, P, time, T, one-time cost of RM, C_r, and detection threshold, I_1, the maximum reduction in primary infection transmission rate δ_M, secondary disease transmission, ϵ, due to RM and reduced post-RM loss, ρ_{PI}.

Our findings offer a decision-making framework that can guide land managers in allocating resources for disease control, emphasising the importance of a calculated and responsive approach to PM investment. The analysis shows that the optimal management strategy depends both on the severity of infection and the speed of spread of the disease. When infection

damages are high or the pathogen spreads rapidly, it is cost-effective to deploy RM once detection occurs. In contrast, when infection damages are low or the disease spreads slowly, it may be more efficient to invest in precautionary measures or, in some cases, not to take action. These results highlight the importance of tailoring management strategies to disease dynamics and risk levels. This framework enables us to strategically allocate resources to control invasive pathogens, thereby preserving both the market and non-market benefits derived from the invaded land. A notable advantage of these models lies in their capacity to facilitate extensive sensitivity analyses, shedding light on how diverse epidemiological and economic components influence the optimal management approach. This result is important because it helps to allocate resources efficiently, ensuring that interventions are applied where they are most needed and avoided where they are not cost-effective. It underscores the importance of balancing the cost of control measures against the potential losses, allowing for a more significance and economically sound approach to disease management.

We demonstrate that, generally, when a disease inflicts a substantial annual loss, it proves more economically prudent to deploy a precautionary management strategy rather than relying solely on reactive measures post-pathogen detection. Firstly, we found that when the RM strategy is available and *always* deployed upon detection, the ability to detect the pathogen early will have a significant effect on the optimal management strategy that should be deployed; this benefit is reduced when the cost of applying the RM is high, and so applying the PM to delay disease loss becomes more cost effective. Secondly, when the RM strategy is available and optimised, the model indicates that it is cost-effective to deploy RM only when expected future infection damages exceed the cost of intervention. If the pathogen does not appear within the planning horizon, or if applying RM would cost more than the expected reduction in damages, it is optimal not to implement RM, thereby avoiding unnecessary expenditure. Hence, it is more cost-effective to deploy the PM annually to prevent the pathogen from arriving, especially when waiting to apply the RM is too expensive. This principle holds particularly true for impending pathogen threats that trigger early losses or those with rapid transmission rates. Notably, the elevation of primary and secondary transmission rates ne-

cessitates a corresponding increase in precautionary management efforts, countering the present value of total cost associated with pathogenic invasion.

When RM is always applied at the detection threshold, we considered this may not be the optimal solution if RM is very expensive or ineffective, and whether RM should be applied was carried out by double optimisation. We examines how the PM strategy protects the forest from invasive pathogens, showing that when a disease causes high annual losses, deploying a PM strategy is more cost-effective than waiting to apply RM once the pathogen is detected. Similar to the result in Chapter 2, the results are sensitive to small change as to applying RM to the whole forest will be costly if the detection threshold is at the barest minimum to compare with the area of the forest.

This chapter analysed the economic implications of precautionary (PM) and reactionary management (RM) strategies for invasive forest pathogens. Our results highlight that prevention is usually more cost-effective than reaction. Investing in PM substantially reduces long-term losses compared to relying solely on RM, especially when disease spread is rapid. RM is best understood as a one-off responsive measure that can limit severe outbreaks, but it cannot substitute for sustained PM investment. The availability of RM can reduce overall costs, yet it remains economically viable only when coupled with sufficient prevention. Optimal strategies depend on key parameters. The cost of PM and RM, the discount rate, and the speed of pathogen spread strongly influence which strategy is most effective. These sensitivities show that decision-makers must weigh economic prudence against the risks of delayed response.

Chapter 6

Conclusions

This thesis contributes to the management of infectious diseases, particularly emerging pathogens, by combining ecological dynamics with economic decision-making in a deterministic modelling framework. Unlike many previous studies, which either focus solely on disease spread or on economic optimisation, this work integrates uncertainty into deterministic models to reflect real-world variability in pathogen arrival and spread. By incorporating costs of precautionary (PM) and reactionary (RM) strategies, the analysis provides a quantitative basis for allocating resources effectively, showing how risk preferences and disease characteristics influence the choice and timing of interventions. The significance of this thesis is its holistic approach, considering not just the biological aspects of pathogen dynamics but also the unpredictable elements and economic implications of disease management strategies. The findings from this research are expected to deepen our understanding of how pathogens spread and how different management strategies can be optimised in the face of uncertainty and economic constraints. This leads to a more effective and realistic disease control and prevention framework, which is vital for public health resilience.

The sensitivity analysis carried out in this thesis examines the contribution of each predicted parameter in both the deterministic analysis and the stochastic process. A key contribution of this thesis is the ability to perform extensive sensitivity analyses that systematically explore how variations in both epidemiological (e.g., disease spread rate) and economic (e.g.,

infection damages, control costs) parameters influence the optimal management strategy. When the pathogen arrives and spreads, it reduces the values of these combined benefits flows over time. A PM strategy is assumed to be deployed annually throughout the time horizon and represents a set of precautionary management actions aimed at reducing the risk or rate of pathogen introduction and early spread. Such measures could include border control, quarantine, or prophylactic spraying. The deterministic model was modified to include random arrival using numerical simulation to find the optimal management strategy between the deterministic and stochastic results. Taking into account the social, economic and environmental aspects of risk aversion, a basic model is developed to identify management strategies that are economically and ecologically sustainable over the planning horizon.

There are two main benefits to defining a cost-effort curve for precautionary management as we did here: a maximum budget for the precautionary control measure and the variability in the maximum effectiveness of the management strategies can be explicitly included and examined. According to the literature, the main trade offs in disease management involve balancing effectiveness, cost, and timing of interventions. For example, precautionary strategies (PM) can reduce the risk of outbreaks but often require upfront investment and may be only partially effective. Reactive strategies (RM) act after detection and can reduce damages, but their effectiveness depends on timely detection and the speed of disease spread. Stochastic strategies add flexibility to account for uncertainty but can increase complexity and monitoring costs. In an uncertain context, people gather information before making a decision. Our work shows that early detection and better information about the pathogen can significantly improve decision-making. When RM is available and affordable, early knowledge allows managers to deploy RM more effectively, reducing expected damages. However, if RM is costly or the disease spreads slowly, early detection alone may not justify immediate action, and investing in PM to delay losses can be more cost-effective. In other words, the value of increased knowledge depends on both the cost and effectiveness of available interventions. It is crucial to understand these mechanisms to predict net effects in specific disease systems and to evaluate the generalisability of these patterns.

Integrating economic factors, this model illuminated the cost implications of disease management strategies. It quantified the financial impact of different management interventions, balancing costs against the benefits of disease mitigation. These insights are pivotal for land managers, offering a cost-benefit analysis crucial for budget allocation and prioritising economic sustainability alongside effective disease control. The stochastic model provided a more realistic depiction of disease spread by introducing randomness and variability. This model accounted for the unpredictable nature of pathogen dynamics. The stochastic model's ability to incorporate uncertainty makes it invaluable for preparing more robust and adaptable disease management plans.

Focusing on risk attitudes, this model explored how risk perceptions and tolerance levels affect decision-making in pathogen management. It highlighted the significance of considering human behavioural factors in disease control. Our analysis revealed that varying levels of risk aversion (denoted by λ) and different degrees of variance significantly influence management strategies. In scenarios where decision maker exhibited risk-averse behaviour (negative values of), they tended to invest more in precautionary measures (PM) to prevent potential losses, rather than relying on reactive management (RM) after infection occurs. Conversely, less risk-averse or risk-neutral decision maker were more willing to delay intervention and rely on RM, accepting the possibility of larger future damages in exchange for lower upfront costs. In contrast, risk-seeking tendencies (positive values λ) led to more aggressive management actions. Higher variance, indicative of increased uncertainty, consistently resulted in more cautious approaches across different scenarios. This model is particularly relevant in tailoring management strategies to suit varying risk appetites, which is crucial for ensuring stakeholder and effective implementation of control measures.

These models collectively illustrate a comprehensive picture of plant pathogen management, each building upon the other. The deterministic model simplicity is a stepping stone towards the stochastic and risk models, while the economic model bridges practical financial considerations with theoretical predictions. Together, they provide a holistic approach to understanding and managing plant diseases. Theoretically, this thesis contributes to the existing

body of knowledge by integrating various modelling approaches in plant pathology. It offers actionable insights for developing adaptive, cost-effective, and risk-tolerant management strategies, which are crucial in the current agricultural landscape marked by climatic variability and economic constraints. The success of this model depends on accurately determining the risk preferences of decision maker and correctly estimating parameters such as λ and P. This requires careful interpretation, which can be challenging but is crucial for the models accuracy and applicability. The findings from this model will inform policies by highlighting the importance of considering risk preferences in management strategies. Policies sensitive to these preferences are likely to be more effective and better received by the stakeholders involved.

Understanding these mechanisms is crucial for predicting the net effects of management strategies in specific disease systems and evaluating how generalisable these patterns are. Our analysis highlights the importance of adopting risk management strategies for farmers and stakeholders to navigate uncertainties in plant and agricultural production. The results show an interconnected relationship between different management strategies, where the choice and timing of precautionary (PM) and reactive (RM) measures jointly determine the outcomes [66]. Sensitivity analyses demonstrate that the optimal management strategy is highly responsive to changes in the effectiveness of both PM and RM.

A long-standing question in managing invasive pests and diseases is whether to prioritise precautionary management (PM), such as border controls, quarantine, or prophylactic spraying, or to rely more heavily on reactive strategies like early detection and rapid response. The case for PM is that once an invasion establishes, losses escalate quickly and are often irreversible, so preventing entry or slowing establishment avoids large downstream costs. On the other hand, prevention can be expensive, and when the risk of invasion is low, reactive control may be the more cost-effective option especially if surveillance is efficient and eradication tools are reliable. The results of this thesis help clarify these trade-offs. We show that PM becomes more valuable when damages are high, spread is fast, or reactive control is costly or delayed. Conversely, when invasion risks are lower and response tools are effective, reactive

management is often sufficient. Importantly, the balance is not fixed: even small changes in effectiveness or costs can tip the decision between prevention and reaction. Our integration of risk preferences adds a further dimension risk-averse managers tend to invest more in PM to avoid catastrophic losses, while risk-neutral managers tolerate greater reliance on reactive approaches.

From a policy perspective, these findings suggest that interventions should be tailored to both disease dynamics and risk preferences. Instead, resources should be allocated across both precautionary and reactive measures, with surveillance playing a pivotal role in linking the two. Recognising how risk preferences influence decisions also makes allocation choices more transparent and aligned with stakeholder behaviour. For example, when RM is affordable and effective, early detection and timely deployment can significantly reduce damage. When RM is costly or slow to act, investment in PM becomes more important to delay infection and reduce long-term costs. Land manager can use these insights to prioritise resource allocation, design cost-effective surveillance and intervention programs, and implement flexible management strategies that adjust to disease severity and economic constraints. In general, this framework provides actionable guidance for sustainable disease management planning on a fixed horizon.

6.1 Future Research Directions

Future research can build upon this thesis by further refining the models developed, incorporating more complex variables, and validating them with real-world data. As research in infectious disease management continues to evolve, several avenues warrant further exploration and consideration. One area for future research is the development of innovative diagnostic tools that can quickly and accurately identify infectious pathogens. With the ongoing threat of emerging diseases and antimicrobial resistance, there is a critical need for improved diagnostic capabilities to facilitate timely and targeted interventions. Additionally, ongoing surveillance and monitoring of infectious diseases, coupled with the integration of

advanced data analytics and predictive modelling, can provide valuable insights for the early detection and mitigation of outbreaks.

Furthermore, policy implications in infectious disease management are of utmost importance. Land manager, in collaboration with researchers and public health professionals, must prioritise investments in public health infrastructure, including healthcare systems, laboratory capacities, and vaccination programs. These collaborative efforts at the national and international levels are essential to strengthen preparedness and response mechanisms, as infectious diseases do not adhere to geopolitical boundaries. Additionally, policies that promote antimicrobial stewardship and regulation of antimicrobial use in healthcare and agriculture are crucial in addressing the growing threat of antimicrobial resistance.

The future of infectious disease management hinges on continuous research advancements and evidence-based policy measures to safeguard global health. There is a clear need to develop and maintain systems to support evidence-based practice in wildlife disease management. This implies a fundamental change from what has been standard practice in the past, such that in the future, the outcomes of disease management interventions should be systematically monitored, collated, and made available to others.

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Appendix A

When RM strategy is available and always deployed upon detection

Now let us assume that the RM strategy is available and *always* deployed upon detection, as in Equation (5.8) and Table A.1. The optimal management strategy is the combination of the optimal level of PM, C^*_{pr}, and whether the pathogen is detected within the time horizon and therefore the RM is applied. RM is not applied ("No") if the pathogen is *not* detected within the time horizon ($\tau(\delta) > T$). RM is applied ("Yes") if the pathogen is detected within the time horizon ($\tau(\delta) \leq T$). When the optimal level of PM is zero ($C^*_{pr} = 0$), we label this "None" and label the management strategy $1c$ (yellow). Furthermore, when the optimal level of PM is equal to the budget ($C^*_{pr} = \Pi$), we label this zone "Full" and the management strategy $3c$ (red).

Optimal management strategy	PM (Optimised)	RM(No/Yes)
$1a$ (black)	None, $C_p^* = 0$	No , $\tau(\delta) > T$.
$2a$ (pink)	Partial, $0 < C_p^* < \Pi$	No, $\tau(\delta) > T$.
$3a$ (purple)	Full, $C_p^* = \Pi$	No, $\tau(\delta) > T$.
$1c$ (yellow)	None, $C_{pr}^* = 0$	Yes, $\tau(\delta) \leq T$.
$3c$ (red)	Full, $C_{pr}^* = \Pi$	Yes, $\tau(\delta) \leq T$.

Table A.1: The management strategies considered when the RM is available and always deployed if the pathogen is detected within the time horizon.

Sensitivity analysis was used to find the optimal level of PM, which minimises the present value of the cost associated with the disease over a fixed time horizon. However, the RM strategy is applied once the pathogen is detected within the time horizon ($\tau(\delta) > T$). The numerical simulation technique was used to determine how the optimal management strategy depends on the key parameters. At detection, the one-time cost of RM is applied to the entire forest under investigation.

A.0.1 Sensitivity analysis with respect to the annual loss, ρ_I, and doubling time, T_2

Sensitivity analysis with respect to the annual loss, ρ_I, and doubling time, T_2, when RM is available and always deployed upon detection is shown in Figure (A.1) as labelled in Table (A.1). We show in Figure A.1(a) the case where the RM is unavailable (as given in Figure (2.6)). Similarly to Figure (2.6), when the loss caused by the disease begins to accumulate early in the time horizon or the disease spreads faster, an increase in the level of PM is required to counter this loss. The two plots show a significant difference when RM is not available in Figure A.1(a) and when RM is available in Figure A.1(b).

We assume that the maximum reduction in primary disease transmission due to PM is $\delta_M = 0$ and RM is "perfect", so it stops the disease from spreading, $\epsilon = 1$, and completely

restores the provision of environmental and economic benefits, $\rho_{PI} = 0$. The optimal solutions derived from Figure A.1(b) can be summarised as follows: For small T_2(large β), the strategy varies depending on the annual loss ρ_I. If ρ_I is high (ρ_I=6000 to 10000), the optimal approach is to implement full PM (zone $3a$). This means that the pathogen spreads rapidly and that the associated losses are significant. In this scenario, it is optimal to fully implement preventive measures (PM) to mitigate the impact of the disease.

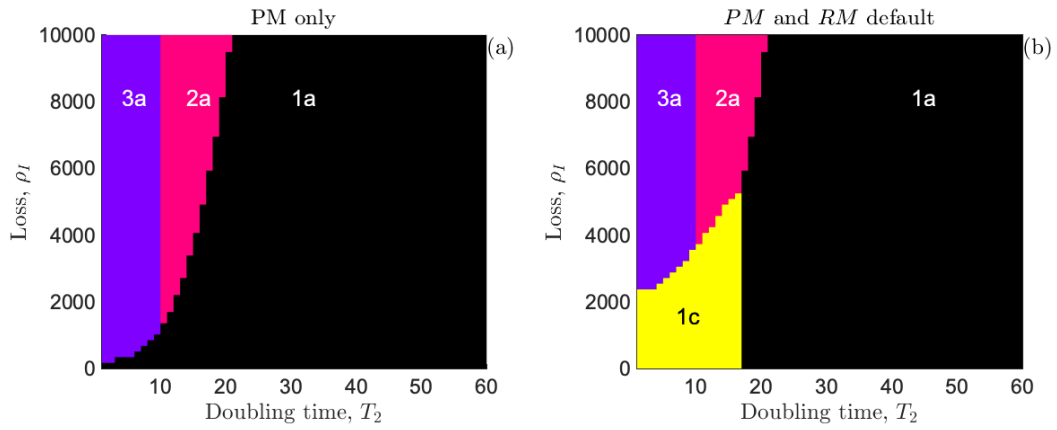

Figure A.1: Sensitivity analysis with respect to the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by (a) when RM is not available (as given by Equation (??) as described in Table 2.2) and (b) when RM is available and always deployed upon detection (as given by Equation (5.8) as described in Table (5.2)). The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The baseline primary disease transmission rate, $P = 0.005$ and the parameter $\delta_M = 0$.

We start by examining the relationship between the zones labelled in Table (A.1) and given by Equation (5.8). The RM is not available as shown in Figure A.1(a), which is the same as Figure (2.6) in Chapter 2 above. When the RM strategy is available and always implemented after detection as in Figure A.1(b). This means that we do not deploy the RM strategy when the disease does not arrive in the time horizon ($\tau(\delta) > T$). However, we deploy the RM strategy when the pathogen is detected within the time horizon ($\tau(\delta) \leq T$). The yellow zone $1c$ is a very fast epidemic, and this is shown in Figure A.1 (b), the zone $1c$ strategy is implemented

if $\tau(\delta) > T$. This happens because the spread of the pathogens is spreading so fast and the only solution will be to apply RM and not PM. In this region, PM is too expensive compared to the loss, but because PM has to be applied throughout the whole time and RM can only be applied once, so zone 1c is that region. In zone 1c, the cost of PM is greater than the loss due to disease, which is greater than the cost of RM.

Therefore, for a pathogen that spreads faster in a short doubling time (zone 1c), it requires immediate intervention due to the increase in loss. However, without the PM or RM strategy, the total loss will increase. Therefore, a one-time cost is required when it is optimal to deploy the RM strategy to prevent further losses. When the annual loss ρ_I, is high, it is always more cost-effective to deploy the PM instead of waiting to treat by applying the RM. It is not optimal to deploy a combination of PM and RM. The reason why it is not optimal to deploy both strategies is because both strategies are completely effective. The PM stops the pathogen from arriving, thus not requiring the RM, and the RM stops any further disease spread and loss.

A.0.2 Sensitivity analysis with respect to baseline primary disease transmission rate, P

Sensitivity analysis of the optimal management strategy with respect to the baseline primary disease transmission rate, P, when the RM is available and always deployed upon detection is seen in Figure (A.2). The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation (5.8) as described in Table (5.2). The numerical simulation techniques were used to find the optimal management strategy when the baseline primary disease transmission rate, P, is given as $P = 0.0005, P = 0.005, P = 0.1$. This can be seen in Figures A.2(a),A.2(b) and A.2(c) respectively.

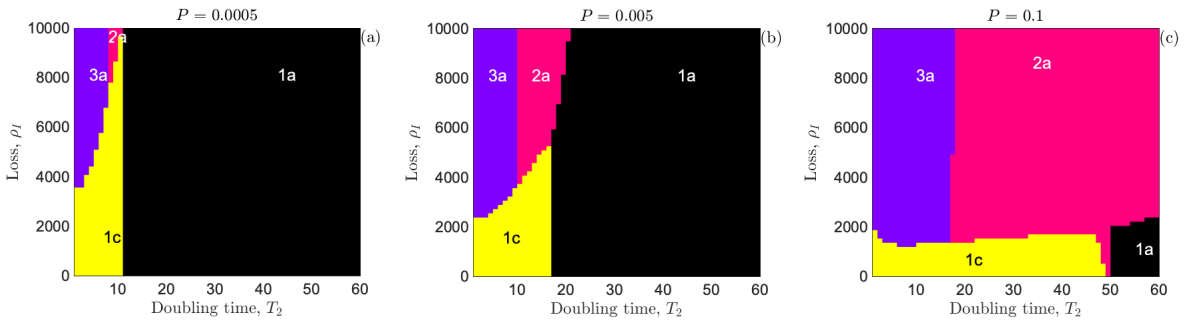

Figure A.2: Sensitivity analysis to the baseline primary disease transmission rate, P, when the RM is available and always deployed upon detection. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.8 as described in Table 5.2. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The baseline primary disease transmission rate is: (a) $P = 0.0005$, (b) $P = 0.005$ and (c) $P = 0.1$. All other parameters are at their baseline values in Table 5.1.

Sensitivity analysis with respect to the primary disease transmission rate, P, is carried out for the effects of the change in the doubling time, T_2, which depends on the secondary disease transmission rate, β, and change in the optimal level of PM. The PM strategy becomes more important than doing nothing or implementing RM. This sensitivity analysis is used to study the effect of how likely the pathogen is to arrive. In general, Figure A.2(a) shows what happens when the baseline primary disease transmission rate, P, is reduced to $P = 0.0005$, while A.2(b) shows the effect on the arrival and spread of the pathogen when P is at the baseline value ($P = 0.005$). Then Figure A.2 (c) shows the effect of the pathogen when P increases to ($P = 0.1$).

Similarly, when RM is not available, Figure (2.8), as the baseline primary disease transmission rate, P, increases, the time it takes for the loss to begin to accumulate, and thus there is a decrease in the region where it is optimal to do nothing (zone 1a), and a subsequent increase in the region where it is optimal to deploy the PM (zone $2a$ and $3a$ in Figure A.2). The black region (zone la) shrinks as the baseline primary disease transmission rate, P, increases ($P = 0.0005 \rightarrow P = 0.005 \rightarrow P = 0.1$ in Figure (A.2)). The pink and purple region (zone $2a$ and zone $3a$) increases as the baseline primary disease transmission rate, P, increases, while the

yellow part (zone $1c$) disappears gradually. This shows that as the baseline primary disease transmission rate P increases, the pathogen arrives earlier but spreads the same. However, as the baseline primary disease transmission rate, P, increases, the time at which the loss begins to accrue is decreasing. The time in which the loss accrues is increasing. There is an increase in the region where it is optimal to deploy the PM (zone $2a$ and $3a$ in Figure (A.2). Increase in the annual loss, ρ_I, shows the switch from deploying the RM strategy (zone $1c$) to deploying PM (zone $3a \rightarrow$ zone $2a$) in the optimal management strategy as T_2 increases. Therefore, it is optimal to deploy RM (Figure A.2) due to the high loss in a short doubling time. However, as a result of the high loss caused by the disease in a short time to double, applying the one-off cost will be cost-effective than deploying PM annually.

A.0.3 Sensitivity analysis with respect to time, T

Sensitivity analysis of the time horizon, T, when RM is available and always deployed upon detection. This is shown in Figure (A.3) together with the annual loss, ρ_I, and the doubling time, T_2. The optimal management strategy is found by Equation (5.9) and (5.10) as described in Table (5.2). The three plots in Figure (A.3), show a significant difference in the results when the time horizon is altered, (a) $T = 50$, (b) $T = 100$, (c) $T = 150$, Figures A.3(a),A.3(b) and A.3(c) respectively.

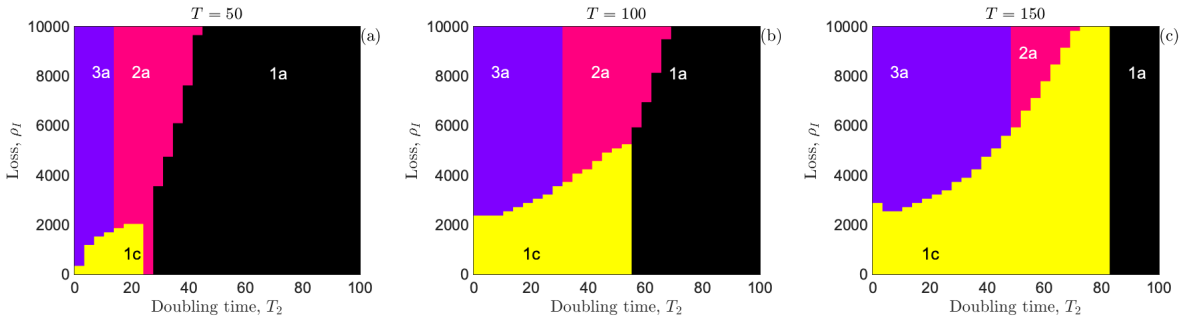

Figure A.3: Sensitivity analysis to time, T, when RM is available and always deployed upon detection. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.8 as described in Table 5.2. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The analysis was carried out at different time horizon: (a) $T = 50$, (b) $T = 100$ and (c) $T = 150$. All other parameters are at their baseline values in Table 5.1.

The sensitivity analysis with respect to the time horizon, T, reveals the effects on the doubling time, T_2, and the annual loss, ρ_I, which increase the optimal level of PM. We show in Figure A.3 that as the doubling time, T_2, increases, the optimal level of PM increases, and the region where it is optimal to deploy RM increases. The yellow region (zone $1c$) grows as the time horizon T, increases as shown in Figure A.3 ($T = 50 \rightarrow T = 100 \rightarrow T = 150$). The black region (zone la) shrinks as time, T, increases ($T = 50 \rightarrow T = 100 \rightarrow T = 150$ in Figure (A.3)). The pink region (zone $2a$) reduces as the time increases to $T = 150$ while the purple region (zone $3a$) increases as the time horizon, T, increases. This shows that the duration of this sensitivity analysis has a great effect on how quickly the pathogen spreads. However, as the time, T, increases, the time at which the loss begins to accrue is increasing and there is an increase in the region where it is optimal to deploy the full PM (zone $3a$) in Figure A.3.

In general, Figure A.3(a) shows what happens when the time, T, is reduced to $T = 50$. While A.3(b) shows the effect on the arrival and spread of the pathogen when the time, T is at the baseline value ($T = 100$). Then, Figure A.3(c) shows The effect of the pathogen when the time T increases to ($T = 150$). Hence, Figures A.3 (a) and (c) show a significant difference

compared to Figure A.3 (b), where the sensitivity analysis of the parameter $T = 100$ was carried out. Since Figure A.3 (b) is the same as Figure A.1(b) above. However, the loss caused by the disease starts to accrue earlier or the disease spreads faster in the early stages of the epidemic, t_0.

In Figure A.3(a), the time is reduced to $T = 50$, and it will be optimal to deploy only the PM strategy. This is because the region where it is optimal to deploy RM will reduce when $T = 50$, at a low loss, and at a short doubling time. The black region (zone $1a$) in Figure A.3(a) shows that we do nothing when the time horizon is small and the annual loss is very small at a high doubling time. When time T, is at the baseline value, $T = 100$ in Figure A.3(b), the region in the parameter space where it is optimal to apply the RM only emerges when the loss, ρ_I, is small (zone $1c$ in Figure A.3(c)). When the time increases to $T = 150$, the region where it is optimal to apply full PM increases as the time increases.

The region where it is optimal to apply partial PM gradually disappears as time increases. However, there is a decrease in the region where it is optimal to do nothing (zone $1a$) at high time horizon. An increase in the time horizon, T, increase the potential loss caused by disease since the rate that the whole forest will become infected is increased (See Figure A.3(c)). Therefore, as time increases, the optimal level of the RM increases in other to counter the loss (see the switch in zone $1c$ from Figure A.3(a) → Figure A.3(b)→ Figure A.3(c)). However, the switch from no PM (zone $1a$) to partial PM (zone $2a$) to full PM (zone $3a$) gradually decreases while the region (zone $1c$) to implement the RM strategy increases over time.

A.0.4 Sensitivity analysis with respect to the one-off cost of RM, C_r, and the detection threshold, I_1

Sensitivity analysis with respect to the detection threshold, I_1, and the cost of the RM, C_r, when the RM is available and always deployed upon detection. The plots in Figure (A.4) highlight the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation (5.8) as described in Table (5.2). The

numerical simulation techniques were used to find the optimal management strategy with different values of detection threshold, I_1, and cost of the RM, C_r: (a) $I_1 = 0.1$ and $C_r = 0$, (b) $I_1 = 0.1$ and $C_r = 3000$, (c) $I_1 = 0.1$ and $C_r = 5000$, (d) $I_1 = 0.25$ and $C_r = 0$, (e) $I_1 = 0.25$ and $C_r = 3000$, (f) $I_1 = 0.25$ and $C_r = 5000$, (g) $I_1 = 0.5$ and $C_r = 0$, (h) $I_1 = 0.5$ and $C_r = 3000$, (i) $I_1 = 0.5$ and $C_r = 5000$, (j) $I_1 = 0.8$ and $C_r = 0$, (k) $I_1 = 0.8$ and $C_r = 3000$, (l) $I_1 = 0.8$ and $C_r = 5000$. This can be seen in Figures (A.4) below, with their baseline values in Table (5.1).

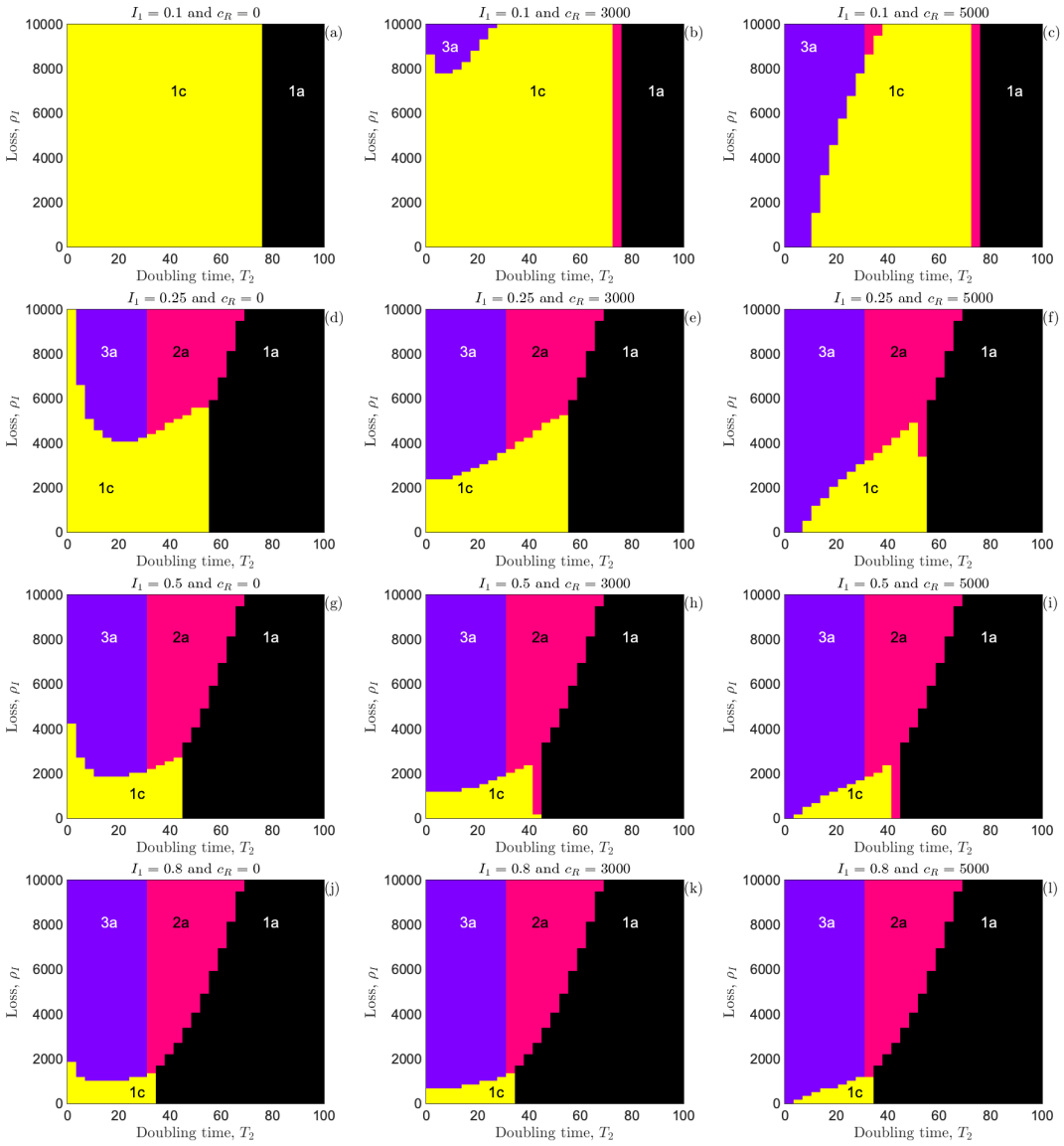

Figure A.4: Sensitivity analysis to the detection threshold, I_1, and cost of the RM, C_r, when RM is available and always deployed upon detection. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.8 as described in Table 5.2. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The detection threshold is, $I_1 = 0.1, 0.25, 0.5, 0.8$, and cost of the RM is, $C_r = 0, 3000, 5000$ as given on the title of each plot. All other parameters are at their baseline values in Table 5.1.

Interestingly, when the one-off cost of applying the RM is zero (say, $C_r = 0$), the region

where it is optimal to wait and apply RM is very big to compare when the one-off cost increases. When the detection threshold $I_1 = 0.1$, and the cost of RM increases progressively ($C_r = 0 \rightarrow C_r = 3000 \rightarrow C_r = 5000$) as we can see in Figure $A.4$(a) to Figure $A.4$(d), the yellow region (zone $1c$) gradually decreases. The black region (zone la) remains the same as the detection threshold, $I_1 = 0.1$, and the cost of RM increases progressively from $C_r = 0 \rightarrow C_r = 3000 \rightarrow C_r = 5000$. The region in which it is optimal to deploy the full PM appears when the one-time cost is $C_r = 5000$.

However, when the one-time cost of applying RM is small, it is optimal to apply the RM for an increase in ρ_I values as the doubling time T_2 increases. This is the reverse when C_r is much larger as seen in comparing Figure $A.4$(d) and Figure $A.4$(e). As C_r increases, the optimal management strategy tends to the strategy when RM is not available (Figure (2.6) in Chapter (2)). A similar effect (a reduction in the parameter range where it is optimal to only apply RM and a subsequent increase in the parameter range where it is optimal to deploy PM) is seen when the detection threshold, I_1, increases (figure $A.4$). This arises because a pathogen that spreads quickly will be detected quicker, thus the time horizon over which the loss is accrued will be much smaller, but the net present cost of the application of the RM is larger (due to discounting). Therefore, when the cost of applying the RM is small, the benefit of detecting the disease earlier and applying the RM earlier is greater. This shows that the ability to detect the pathogen early will have a significant effect on the optimal management strategy to be implemented. However, this benefit is reduced when the cost of applying the RM is large, and so applying the PM to delay the loss from the disease becomes more cost-effective.

A.0.5 Sensitivity analysis with respect to the maximum reduction in primary disease transmission rate due to PM, $1 - \delta_M$

In this section, we analyse how the optimal PM strategy changes when the maximum reduction in primary disease transmission rate due to PM when RM is available and always deployed upon detection. The plots in Figure (A.5) show the effect of different values of δ_M on

annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation (5.9) and Equation (5.10) as described in Table (A.1). The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM and the red region (zone $3c$) is full PM and RM.

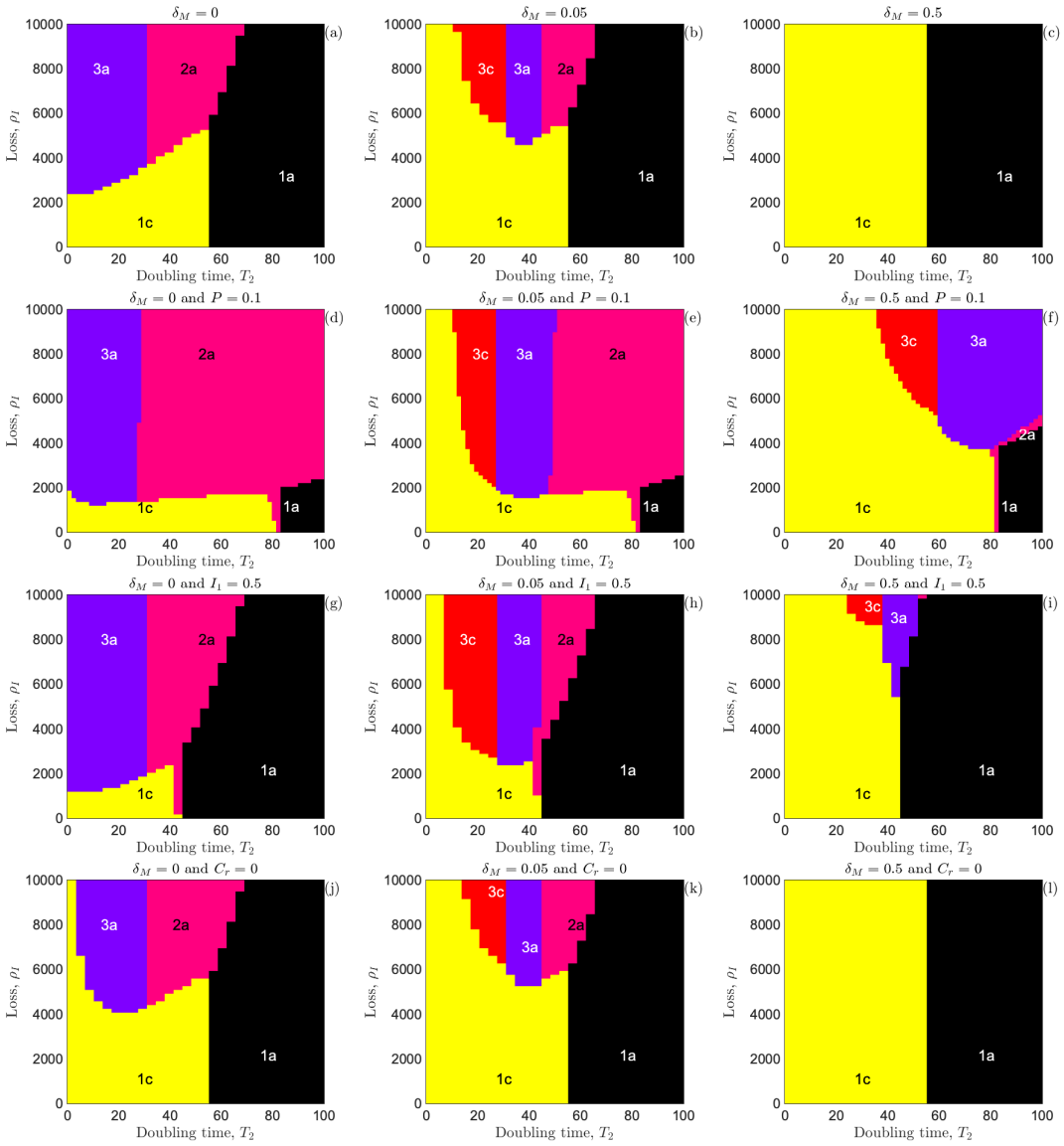

Figure A.5: Sensitivity analysis to the maximum reduction in primary disease transmission rate due to PM, $1-\delta_m$, when RM is available and always deployed upon detection. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.8 as described in Table 5.2. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM, and the red region (zone $3c$) shows full PM and RM. The parameters δ_M, P and I_1 have the following values: $\delta_M = 0, 0.05, 0.5$,$P = 0.005, 0.1$, $I_1 = 0.25, 0.5$ and $C_r = 0$, all other parameters are at their baseline values in Table 5.1.

Numerical simulation techniques were used to find the optimal management strategy with different parameter values δ_M ($\delta_M = 0, 0.05, 0.5$), primary disease transmission rate P ($P = 0.005, 0.1$), and detection threshold, I_1 ($I_1 = 0.25, 0.5$). All other parameters are at their baseline values in Table (5.1). Decreasing the maximum reduction in primary disease transmission due to PM, $1 - \delta_M$ (by increasing the parameter δ_M) means that the pathogen can never be fully prevented from arriving within the time horizon. The parameter δ_M, plays a critical role in determining the optimal management strategy. Focusing on the figures in the first column, which includes Figures A.5 (a), (d), (g) and (j), where they all have the same value of $\delta_M = 0$, Figures A.5(d) has $P = 0.1$, A.5(g) has $I_1 = 0.5$ and A.5, and (j) has $C_r = 0$.

The result shows a larger region where full or partial PM (zones $3a$ and $2a$) is optimal and a smaller region for the RM strategy. Figures in the middle column, which include Figures A.5 (b), (e), (h), and (k), all have the same value of $\delta_M = 0.05$, Figures A.5 (e) have $P = 0.1$, A.5 (h) have $I_1 = 0.5$ and A.5 (k) have $C_r = 0$. We notice a reduction in the region where the full PM strategy is optimal (zone $3a$ shrinks), and an increase in the zones where RM is necessary. Figures in the last column, which include Figures A.5 (c), (f), (i), and (l), all have the same value of $\delta_M = 0.5$, Figures A.5(f) have $P = 0.1$, A.5(i) has $I_1 = 0.5$ and A.5 (l) has $C_r = 0$, all other parameters are at their baseline values in Table 5.1. The regions where PM alone is deployed significantly shrinks and shows meanly the region for the RM strategy (zone $1c$).

Figure A.5 shows the relationship between management strategies and pathogen dynamics, showing how reducing PM effectiveness results in greater challenges to controlling the spread and arrival of pathogens. The analysis reveals the critical balance between the effectiveness of preventive measures (PM) and the need for reactive measures (RM). As PM becomes less effective (increasing δ_M), the model shows a shift toward doing nothing or relying more on reactive strategies (RM). This has significant implications for disease management, suggesting that when preventive measures cannot completely stop the spread of a pathogen, more resources should be allocated to RM. The cost effectiveness of interventions changes with the disease transmission shown through doubling time and the economic impact of the disease shown through annual loss.

A.0.6 Sensitivity analysis with respect to reduction in secondary disease transmission, ϵ, due to RM and reduced post-RM loss, ρ_{PI}

Sensitivity analysis with respect to secondary disease transmission, ϵ, post-RM loss, ρ_{PI}, and doubling time, T_2, when RM is available and always deployed upon detection. The plots in Figure (A.6) show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy when the secondary disease transmission, ϵ, and the post-RM loss, ρ_{PI} are altered. The optimal management strategy is found by Equation (5.8) as described in Table 5.2. The value of secondary disease transmission, ϵ, is given as $\epsilon = 0, 0.5$ and 1 while the post-RM loss, ρ_{PI}, is given as $\rho_{PI} = 0, 0.5$ and 1. We used numerical simulation to find the optimal management strategy with different parameter values, secondary disease transmission, ϵ ($\epsilon = 0, 0.5, 1$) and post-RM loss, ρ_{PI} ($\rho_{PI} = 0, 0.5, 1$). All other parameters are at their baseline values in Table (5.1).

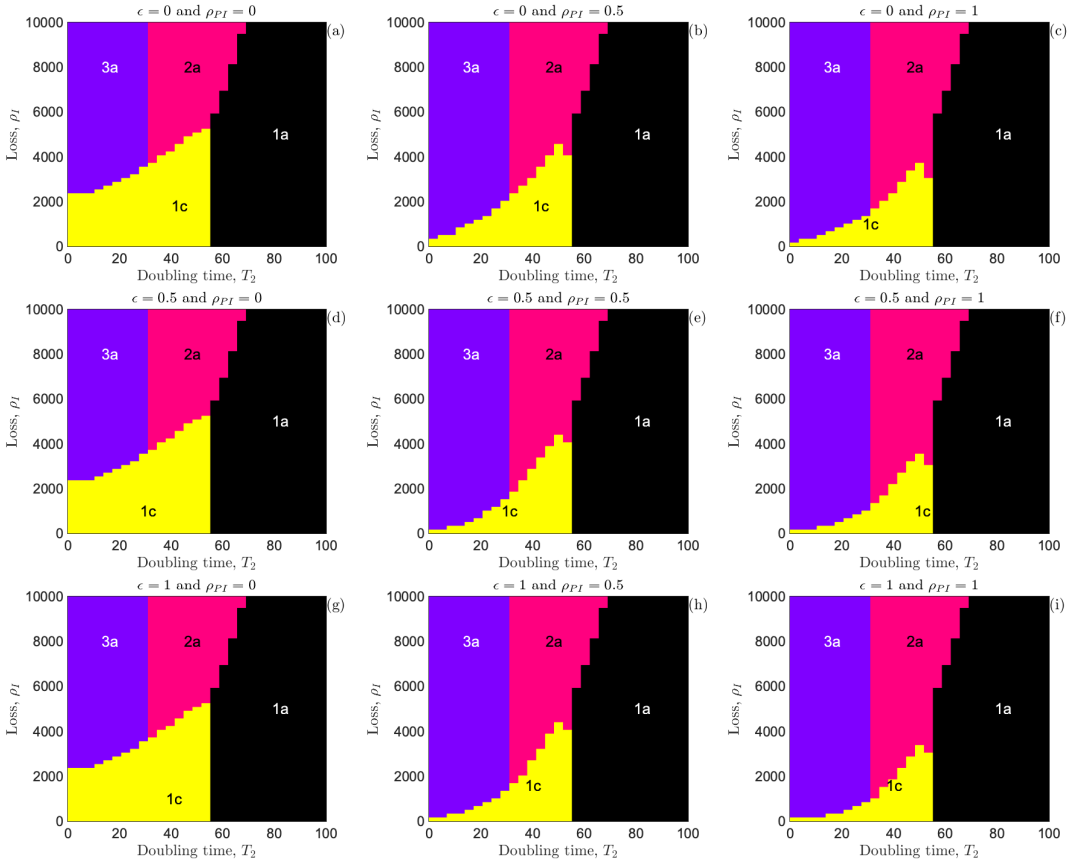

Figure A.6: Sensitivity analysis to the secondary disease transmission, ϵ and the post-RM loss, ρ_{PI}, when RM is available and always deployed upon detection. The plots show the effect of the annual loss, ρ_I, and the doubling time, T_2, on the optimal management strategy. The optimal management strategy is found by Equation 5.8 as described in Table 5.2. The black region (zone $1a$) shows where it is optimal to deploy no PM and no RM. The pink region (zone $2a$) is optimal to deploy partial PM and no RM. The purple region (zone $3a$) is optimal to deploy full PM and no RM. The yellow region (zone $1c$) shows where it is optimal to deploy RM and no PM. The value of the secondary disease transmission, ϵ are $\epsilon = 0, 0.5, 1$ and the post-RM loss, ρ_{PI}, are $\rho_{PI} = 0, 0.5, 1$, all other parameters are at their baseline value.

The first row shows Figures A.6 (a), (b), (c) where $\epsilon = 0$ in all figures, while $\rho_{PI} = 0, 0.5, 1$ respectively. When secondary transmission is zero, the disease does not spread after initial introduction, making RM more effective in containing outbreaks. As post-RM loss, ρ_{PI} gradually increases from 0 to 1 across these three plots, the size of the yellow region (zone $1c$) increases, where RM is deployed without PM, also decreases as ρ_{PI} increases. The second row shows Figures A.6 (d), (e), (f) where the secondary disease transmission rate, $\epsilon = 0.5$ in all the

figures while $\rho_{PI} = 0, 0.5, 1$ respectively. With the secondary transmission rate ($\epsilon = 0.5$), the spread of the disease increases the need for control. This introduces a larger purple region (zone $3a$) where full PM is deployed, where RM is deployed without PM, and also reduces as the post-RM loss ρ_{PI} increases. The last row shows Figures A.6 (g), (h), (i) where $\epsilon = 1$ in all figures, while $\rho_{PI} = 0, 0.5, 1$ respectively. When $\epsilon = 0.5$, there is a full secondary transmission rate, and this implies that once the disease is introduced, it spreads completely unchecked unless it is managed.

Consequently, the plots show a significant increase in the purple zone (zone $3a$), where the full PM is deployed. Even at higher doubling times and losses, PM remains the preferred strategy to prevent further transmission. The plots indicate that the decision to deploy preventive measures (PM) versus reactive measures (RM) is highly dependent on both secondary transmission and post-RM losses. As secondary transmission increases (ϵ), it becomes more optimal to implement full PM strategies to prevent the disease from spreading. However, as post-RM losses ρ_{PI} increase, reliance on RM alone becomes a more common strategy, particularly when secondary transmission is lower.