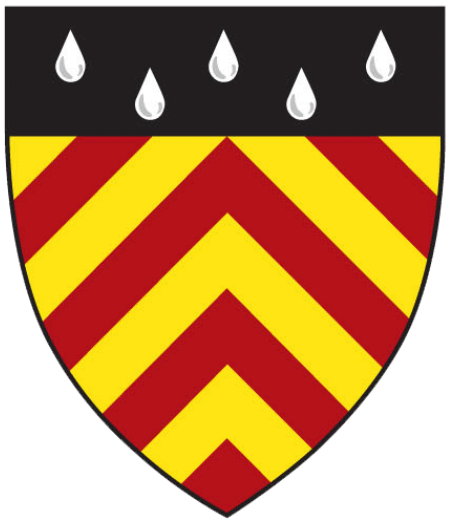

How growers make decisions impacts plant disease control

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Declaration

This dissertation is the the result of my own work and includes nothing which is the outcome of work done in collaboration except as declared in the Preface and specified in the text.

This dissertation is not substantially the same as any that I have submitted, or, is being concurrently submitted for a degree or diploma or other qualification at the University of Cambridge or any other University or similar institution except as declared in the Preface and specified in the text. I further state that no substantial part of my dissertation has already been submitted, or, is being concurrently submitted for any such degree, diploma or other qualification at the University of Cambridge or any other University or similar institution except as declared in the Preface and specified in the text.

This dissertation does not exceed the prescribed word limit of 60,000 words (excluding bibliography, figures and appendices) as specified by the Degree Committee of the Faculty of Biology.

Rachel Emma Murray-Watson
September 2022

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Abstract

Whilst the spread of plant disease depends strongly on biological factors controlling transmission, epidemics clearly also have a human dimension. Disease control depends on decisions made by individual growers, who are in turn influenced by a broad range of factors. Despite this, human behaviour has rarely been included in plant epidemic models. This thesis focusses on addressing this oversight by developing combined epidemic and economic models of disease spread. We use simple continuous-time models of disease spread which we couple with behavioural models which set the management programme of the growers for the next growing season. Our models are rooted in game theory, with growers making strategic decisions based on the expected profitability of different control strategies.

In the first instance, we compare different versions of this behavioural model, which differ in terms of the information used by the growers to assess profitability. We investigate these models in the context of Cassava Brown Streak Disease (CBSD) and its management via the use of clean seed systems (CSS). We find that both the information used by growers to assess profitability and the perception of economic and epidemiological parameters influence long-term participation in the CSS. Over-estimation of infection risk leads to lower participation in the CSS, as growers perceive that paying for the CSS will be futile. Additionally, though the CSS can reduce the burden of disease, and allow a scenario in which all growers control, disease is not eliminated from the system.

For the remainder of this thesis, we use one behavioural model to investigate the deployment of crop that is either resistant or tolerant to Tomato Yellow Leaf Curl Virus (TYLCV). We find that when growers used resistant crop, higher yields were achieved by both controllers and non-controllers, though widespread use of resistant crop was not achieved. The use of tolerant crop reduced the yields for non-controllers, but generally benefited its users, inducing a positive feedback loop that resulted in a high proportion of growers using tolerant crop.

By extending this TYLCV model to allow a three-way choice of tolerant, resistant, and unimproved crop, we see again how growers prefer tolerant crop. However, these responses can be manipulated by changing the cost of each crop type through subsidisation schemes. To do this, we consider the efforts of a “social planner" who moderates the price of crops. We find that subsidising tolerant crop costs the social planner more in subsidies, as its use encour-

ages selfishness and widespread adoption. Subsidising resistant crop, however, increased the use of resistant crop, again enabling higher yields across the community of growers.

Many of our results obtained were robust to spatial and stochastic effects. Some differences arose when growers narrowed their information sources to only consider those growers whose fields are in close proximity to their own, as this allowed assessments of profitability to be based on local disease pressures.

In this thesis, we show how simple models of grower behaviour can be incorporated into both deterministic and spatial-stochastic models of disease spread. Understanding the influence of economic and epidemiological factors, as well as the feedback loops induced by different control mechanisms, on these behaviours can help to promote better outcomes for growers.

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Chapter 1: Introduction

1.1 The global challenge of plant diseases

Global food security relies on the deployment of plant disease control mechanisms. Disease-related yield losses in five staple crops (rice, wheat, maize, potato and soybean, which collectively provide nearly half of the world's calorie intake) vary between 20-30% (Savary et al. (2019)). These losses are not shared evenly; losses are disproportionately concentrated in resource-poor regions such as Sub-Saharan Africa or South East Asia. Such low yields can have severe economic consequences, but also threaten the health and security of millions of people (Savary & Willocquet (2020)). Consequently, much effort has been expended in researching effective control mechanisms for disease.

However, even when available, crop protection mechanisms are not universally adopted. Barriers to adoption are varied, and are likely to be context-dependent. Growers may be concerned by the unreliability of control (Milne et al. (2018), Singerman & Rogers (2020), Rodriguez et al. (2009), Sherman & Gent (2014)), or potential lack of transferability between tests and real-world

scenarios. Another oft-cited reason as to why growers are reluctant to enter into a management scheme is the belief that others would not also do the same, so the grower would effectively be subsidising the disease management efforts of others (Milne et al. (2018), Singerman & Useche (2019)). Growers may be economically limited (Rodriguez et al. (2009), Sherman & Gent (2014)). Access to information is also an important influence over a grower's management decisions (Kaup (2008), Milne et al. (2018), Singerman & Rogers (2020), Rodriguez et al. (2009), Sherman & Gent (2014) Garcia-Figuera et al. (2021)). Understanding why growers choose to partake in control is vital for predicting not only epidemic outcomes, but also to learn how to incentivise participation.

Whilst the motivations behind grower participation have been studied from a sociological perspective, its inclusion in epidemic models of plant disease management has been limited (exceptions include Milne et al. (2016), McQuaid et al. (2017a), Milne et al. (2020), Saikai et al. (2021) and Bate et al. (2021)). Yet growers' participation in voluntary control schemes will have a substantial impact on disease spread. In this thesis, we develop models of plant epidemics that integrate grower behaviour, addressing the oversights discussed above. A primary aim of this work is to assess how not only the inclusion of behaviour, but its means of inclusion, affects model predictions and potential policy recommendations.

1.2 History of plant disease management

The diverse nature of plant diseases, as well as the varied contexts in which they occur, have inspired a broad range of disease control mechanisms. These

vary depending on the type of organism that causes disease (bacteria, fungi, viruses, nematodes *etc.* all have unique physiologies that require targeted control mechanisms), the mechanism of disease spread (for example, whether it is vector-transmitted or soil-borne) and the cultural context of the grower (as they may be limited by regulations, resources, or current local practices). The efficacy of control will also vary depending on local environments, and some may be more dependent on the collective action of a group of growers than others. Broadly, there are four main categories of plant disease control: chemical, biological, cultural and genetic. For the purposes of this thesis, we only focus on cultural or genetic controls.

Cultural controls

There is a long history of using cultural controls to manage plant diseases; as far back as 1660 France, laws required that barberry bushes be removed due to their concurrence with cereal rusts (Katan (2010)). Many different forms of cultural control have been recorded and vary in intensity.

Roguing is a common means of cultural control whereby scouts survey an area of land for signs of infection. Any infected hosts found during these surveys are then removed. This removal can be paired with culling of other hosts within a defined radius and/or with replanting of non-infected hosts. Roguing has been used to manage huanglongbing (*Candidatus* Liberibacter spp) with varying degrees of success (Sisterson & Stenger (2013), Yuan et al. (2021), Graham et al. (2020)). It has also been used to manage tomato yellow leaf curl

virus (TYLCV; Polston et al. (1999), Polston & Lapidot (2007), Ddamulira et al. (2021), Ioannu (1987)), banana bunchy top virus (Allen (1978)) and grapevine leafroll disease (*Grapevine leafroll-associated virus 3*; Almeida et al. (2013))

Crop rotation schemes are common in disease management, as growing non-host species between crops of at-risk species can interrupt pathogen replication cycles or reduce the build-up of toxic substances in the soil (Krupinsky et al. (2002)). Strawberry plants inter-cropped with tomato were less susceptible to fusarium wilt (*Fusarium oxysporum*) than those in non-rotational plots (Fang et al. (2012)). Other common cultural controls include clean seed systems (Vroh et al. (2011), McQuaid et al. (2017b)) and altered planting times (Karungi et al. (2000) and Asante et al. (2001)). Cultural controls may also be specific to a pathosystem: to manage citrus greasy spot (caused by *Zasmidium citri*) it is recommended that growers remove leaf litter from the vicinity of citrus trees, as this acts as a source of inoculum that can cause further infections (Whiteside et al. (1970)).

Genetic controls

Harnessing and manipulating the genetic underpinnings of plant immune responses allows for pathogen-specific disease control. A broad range of such modifications has been made, targeting all aspects of the plant's immune response (van Esse et al. (2020)). Disease resistance is the best-known form of genetic control, and involves the plant host mounting an immune response to

kill the invading pathogen. A wide range of crops have been bred or genetically edited to be resistant or partially resistant to pathogenic organisms (van Esse et al. (2020)).

However, pathogen evolution occurs at a much faster rate than that of the development and deployment of resistant varieties, particularly as there is high selection pressure from monocultures (Consortium et al. (2013), Yang et al. (2021)). "Resistance breakdown", where a pathogen has evolved to evade or otherwise overcome the plant's immune defence, follows. In the 1990s, for example, a new strain of *Puccinia graminis f. sp. tritici* (Ug99), the causative agent of stem rust, was detected that overcame the resistance genes of many wheat varieties that had been in use (Singh et al. (2015), Njau et al. (2010)). The potential for resistance breakdown has encouraged much research into the development of durable disease-resistant varieties (such as by "pyramiding" or "stacking" quantitative resistance traits, requiring the pathogen to overcome multiple plant defence mechanisms) (Mundt (2018)) or co-deploying different resistant varieties (McDonald & Stukenbrock (2016)).

Other forms of genetic improvement include modifying a crop so that it is resistant to herbicides, allowing more selective elimination of weeds (Meissle et al. (2011)). Alternatively, crops may be bred/modified such that they are tolerant to the effects of disease; rather than mounting an immune response that reduces the pathogen burden, the host will instead mitigate the negative consequences of infection (Pagán & García-Arenal (2020)). Historically, the development of disease-tolerant varieties has been slow, partially due to con-

fusion over the distinction between tolerance and partial resistance (Bingham et al. (2009)). Nevertheless, tolerant varieties for a variety of pathogens have now been developed (for example, peppers tolerant to cucumber mosaic virus (Lapidot et al. (1997b)) or identified in already-common varieties (such as cassava landraces tolerant to cassava mosaic virus infection (Allie et al. (2014))). Indeed, given that the benefits of tolerant crop are only felt by an individual grower, and cannot be shared across the landscape by others (i.e. they generate *negative externalities*, discussed below), breeders may actually be incentivised to develop disease-tolerant cultivars in the future. However, this has not been studied to date.

Integrated pest management

Increasingly, to achieve better management outcomes and ensure sustainable use of current control mechanisms, a suite of different controls is employed when faced with disease. Integrated pest management (IPM; more broadly called integrated disease management; McDonald & Stukenbrock (2016)) schemes encourage a holistic approach to crop control: there is no rigid approach to IPM, and individual schemes may involve using a mixture of chemical controls, cultural practices and host genetic resistance. For example, better control of tomato yellow leaf curl virus (TYLCV) was found when a combination of resistant plants, insecticides and reflective mulch (that deterred whitefly vectors) was used (Fonsah et al. (2018)). Similarly, small-holder farmers in Northern India improved cauliflower yields when IPM techniques that included raised

beds, polythene sheets, bio-pesticides and insecticides were adopted (Ahuja et al. (2012), Ahuja et al. (2015)). This mixed-method approach may also slow the emergence of resistance-breaking pathogens, ensuring long-term durability and efficacy of current management practices (McDonald & Stukenbrock (2016)). Overall, IPM schemes are effective at reducing pesticide use whilst maintaining yields, though the need for specialised training or equipment may limit their use (Rezaei et al. (2019)).

1.3 Human behaviour and epidemic models

In recent decades, much attention has been devoted to including aspects of human behaviour into epidemic models of human disease spread (reviewed in Funk et al. (2010) and Chang et al. (2020)). Historically in human disease epidemiology, most models of disease followed the law of mass action where individuals interact with each other at random, axiomatically excluding any human decision-making. Expanding models such that awareness of disease can spread in the population leads to different degrees of susceptibility between groups (Epstein et al. (2008), Kiss et al. (2009), Funk & Jansen (2013), Perra & Vespignani (2013)). Other extensions to the classical susceptible-infected-removed (SIR) models include individual characteristics (such as ignorance, previous experience or risk aversion; Epstein et al. (2008), Vardavas et al. (2010)) and models where individuals change their actions based on game-theoretic evaluations (Bauch & Earn (2004), Karlsson & Rowlett (2020)). Many of these studies focus on vaccination behaviours, though other studies have included social distancing measures (Epstein et al. (2008), Del Valle

et al. (2013)) and protections such as face masks (Karlsson & Rowlett (2020)).

The inclusion of human behaviour into models of plant epidemics has been much more limited. There are a number of qualitative differences between plant and human disease spread that in turn impact how behaviour may be included into plant disease models, the most obvious being that plants cannot make decisions so all behaviours must be enacted by a grower. In plant disease models, therefore, there is a decoupling of those that are infected (plants) and those who bear the consequence of infection (growers, as they may suffer a loss in yield if their plants become infected). This facilitates a gap in knowledge, as growers may be unaware of infection in their field and consequently fail to adjust their behaviour. The spread of disease and the spread of information are naturally separated.

Additionally, though human-mediated transport is possible, plants are largely sessile so some control options (such as changing contact networks, social distancing or vaccinations) have limited utility in plant epidemics, though there are analogous control options available (such as limiting the import/export of plant materials). Even so, there is a range of control options that are not available in human disease management (such as deployment of resistant crop, roguing, use of crop protection chemicals, altering trade networks *etc.*, though again they may have analogues such as vaccination or social isolation). Unlike many options for human disease prevention which may have small economic costs (e.g. receiving a government-subsidised vaccination or wearing a face mask), many of the options for plant disease prevention will place considerable

economic burdens on growers. This, and the distinction between making a decision about one's health and one's livelihood, may change how individuals evaluate risk and make decisions.

Recently, a small number of models of plant disease epidemics have included aspects of the grower's decision-making processes. The behavioural components of the models in Milne et al. (2016), McQuaid et al. (2017a) and Saikai et al. (2021) all take a similar form in which growers have a binary choice of whether or not to control. Their decision rests on the grower's assessment of the profitability of each action and how it compares with their own profit in the previous season (meaning the growers act based on strategic-adaptive expectations (Fenichel & Wang (2013)), discussed in detail below). These models have complex spatial and stochastic components, allowing the inclusion of factors such as trade networks and diverse information sources. Whilst this adds an element of biological realism to the models, it can obscure underlying mechanisms that drive particular behaviours. Simplifying these complex models is therefore a key goal of this work.

Milne et al. (2020) examine co-operative control schemes, using "opinion dynamics" (Xia et al. (2010), Moussaïd (2013), Moussaïd et al. (2013)) to focus on the influence of social factors on the control decisions of growers. In the model, growers consider the control practices of their neighbours, and have a tendency to conform with practices that align with their current beliefs. Bate et al. (2021) use coalition theory (Mesterton-Gibbons et al. (2011)) to model a farmer's voluntary participation in a co-operative control scheme. Individual

farmers want to maximise their own profit, but those who are members of a scheme must consider the impact of their actions on others (which non-participating farmers do not). Bate et al. (2021) find that such schemes can be successful, but require external support (e.g. from governments).

Though each of these models successfully incorporates aspects of grower behaviour into plant disease models, they also have limitations. Those that consider the economics of disease control are largely based on the same form (strategic-adaptive expectations; Milne et al. (2016), McQuaid et al. (2017a), Saikai et al. (2021)) and do not consider the impact this has on model dynamics. The complexity of most of the models, too, precludes any generalisations that would allow for general conclusions to be drawn about the incentives required to encourage control. Finally, in each case growers could only choose between two options, which for many pathosystems in resource-rich settings may be an unrealistic constraint.

1.4 Introduction to game theory

Throughout this work, we use techniques and principles borrowed from game theory, a branch of economics that uses mathematics to study the strategic interaction between individuals (Morris (2012)). Each of these individuals aims to optimise their own outcome, which usually relies on the decisions other individuals make. The core concept underlying game theory is that of a *Nash equilibrium* (Nash Jr (1950), Morris (2012)). A Nash equilibrium is a steady-state solution where the strategy choices of the player mean no individual should rationally deviate from their choice, given that they have full informa-

tion on the strategic choices of others. Nash equilibria are closely related to the concept of an evolutionary stable strategy (ESS; Smith & Price (1973), Thomas (1984)), which has the additional stipulation of non-invasibility (that is, an individual adopting a variant strategy cannot achieve a higher payoff than anyone currently in the population).

Game-theoretic approaches have been used in a wide variety of disciplines, from resource management (Madani (2010), Munro (2009), Meshram et al. (2021)) to cancer treatment (Archetti & Pienta (2019), Wölfl et al. (2022)) and political theory (Austen-Smith & Banks (1998), Duell (2020)). It is particularly prominent in the field of evolutionary biology, where the theory of replicator dynamics has been used to study how a strategy's frequency changes in a population depending on its relative fitness (Taylor & Jonker (1978), Schuster & Sigmund (1983)). In that formalism, in a two-strategy model, the performance of the player's own strategy is compared against that of the average performance of the population. If the payoff is less than that of the population, the focal player changes strategy; if it is higher, they remain with their current strategy.

Game theory in human and veterinary disease models

The nature of the games used in human and veterinary disease modelling is varied (Chang et al. (2020); Table 1.1), though many are based on replicator dynamics. Games can either be repeated ("iterative"), where players can have multiple opportunities to update their information and make decisions (e.g.

Delabouglise & Boni (2020), where farmers make repeat decisions whether to vaccinate their flock of poultry based on current disease prevalence) or non-repeated, where the decision made at the beginning of the game is what is used throughout (e.g. Nixon et al. (2017), where farmers play a game once to determine whether they should invest in prophylactic treatment for sheep scab). The games can be played across the entire population (e.g. Sykes & Rychtáň (2015), where individual pet owners base their vaccination strategy for their pets on the expected payoffs for the entire population), or across networks that connect a subset of nodes (e.g. Fu et al. (2011*a*), in which an individual's decision to vaccinate is based on the payoffs of those in their social network).

Strategies may be self-learned, where individuals have perfect information on the state of the system and base their decisions on this information coupled with their previous history of adopting a strategy (e.g. Mohr et al. (2020), where a farmer's previous experience will inform whether they adopt diagnostic tests for livestock). Alternatively, individuals may learn through "imitator dynamics" (Hofbauer & Sigmund (2003), used in: e.g. Galvani et al. (2007), where individuals base their vaccination decisions on what the most profitable strategy is across the population). The forms of the equations or models based on imitation dynamics bear a close resemblance to replicator dynamics, and both are based on the relative fitness of each strategy averaged across every player using each strategy. The differences lie in which values are compared, as imitation dynamics can allow a number of different "imitation" rules. For

example, imitation dynamics often directly compare the outcomes of two players interacting (and not the average across entire strategies as is the case for replicator dynamics; Hofbauer & Sigmund (2003)). Additionally, imitator dynamics can allow for an "adopt if better" rule, where individuals will always adopt the alternative strategy if it offers a better payoff.

Perhaps the best-studied application of game theory in disease modelling is the "vaccination game", first introduced by Bauch & Earn (2004). In the vaccination game, individuals have a choice of vaccinating or not vaccinating (either themselves, another person or animal). Usually, the individual's decision is based on their weighting of their perceived risk of the vaccine, its cost and the benefit it provides (which can be based on its efficacy, the risk of the disease *etc.*) (Chang et al. (2020)). A simple example of the vaccination game is provided in Box 1, though it has spawned many derivatives that incorporate the extensions described in this section. Vaccination games are typically non-repeated, as the decision to vaccinate occurs either at the beginning of life or the beginning of an epidemic and it is assumed the vaccination will provide coverage over the individual's entire lifetime, though in some cases there may be an annual decision to vaccinate (Delabouglise & Boni (2020)).

Table 1.1: Classification of Game Theoretic Models in Epidemiology
.

Game classification	**Examples**
Iterative	Fukuda et al. (2014), Eksin et al. (2017), Reluga et al. (2006), Bauch & Bhattacharyya (2012), Mohr et al. (2020), Delabouglise & Boni (2020), Poletti et al. (2012), Poletti et al. (2009)
Non-repeated	Bauch (2005), Nixon et al. (2017), Sykes & Rychtáă (2015), Bhattacharyya et al. (2019), Bhattacharyya & Bauch (2011), Fu et al. (2011*a*), d'Onofrio et al. (2007)
Population-level	Bauch (2005), Mohr et al. (2020), Nixon et al. (2017), Sykes & Rychtáă (2015), Galvani et al. (2007), Bhattacharyya & Bauch (2011), Reluga et al. (2006), Bauch & Bhattacharyya (2012), d'Onofrio et al. (2007), Delabouglise & Boni (2020), Poletti et al. (2012), Poletti et al. (2009)
Network-based	Fukuda et al. (2014), Eksin et al. (2017), Bhattacharyya et al. (2019), Fu et al. (2011*a*)
Self-learned	Eksin et al. (2017), Mohr et al. (2020), Nixon et al. (2017), Sykes & Rychtáă (2015), Bhattacharyya et al. (2019), Bhattacharyya & Bauch (2011)
Imitator dynamics	Bauch (2005),Fukuda et al. (2014), Galvani et al. (2007), Bhattacharyya et al. (2019), Fu et al. (2011*a*), Reluga et al. (2006), Bauch & Bhattacharyya (2012), d'Onofrio et al. (2007), Delabouglise & Boni (2020), Poletti et al. (2012), Poletti et al. (2009)

The vaccination game.

Here we summarise Bauch and Earn's canonical "vaccination game", from which many other models of vaccination behaviour have been derived. The game focuses on the vaccination intentions of parents in a population of size N. The parents play a mixed strategy, vaccinating their children with probability P or not vaccinating with probability Q. The current proportion vaccinated is given by p.

When evaluating the expected payoffs for each strategy, parents consider the perceived risk of the vaccination (r_V) and the actual risk of infection $r_\theta(p)$. The risk of infection depends on the current proportion of the population that has been vaccinated, p, and is strictly decreasing as p increases. When the herd immunity threshold, $p' = 1 - \frac{1}{R_0}$ is met, then $r_\theta(p') = 0$ (as disease should be eliminated). Conversely, the risk of infection is at its maximum when $p = 0$ ($r_\theta(0) = \max(r_\theta)$)

The expected payoffs of each strategy are given by:

$$E_P(P, p) = -r_V P - r_\theta(1 - P) \tag{1.1}$$

$$E_Q(Q, p) = -r_V(1 - Q) - r_\theta Q \tag{1.2}$$

The expected benefit of vaccination, then, is given by:

$$\Delta E = E_P - E_Q = (r_\theta - r_V)(P - Q). \tag{1.3}$$

If the perceived risk of vaccination is greater than the actual risk of infection when no-one is vaccinated (i.e. $r_V \geq r_\theta(0)$), then $r_V > r_\theta$ for all values of p. The only way for $\Delta E > 0$ (i.e. for there to be a benefit of switching to the vaccination strategy) is if $P = 0$. This is a Nash equilibrium.

Conversely, if the perceived risk of vaccination is less than the actual risk of infection when no-one is vaccinated (i.e. $r_V \leq r_\theta(0)$), there must be some value of p, p^*, at which $r_V \leq r_\theta(p^*)$. This is because there is an upper bound on p, p' (i.e. the herd immunity threshold), at which point $r_\theta(p') = 0$, so any positive value of r_V must be greater than $r_\theta(p')$. The value p^* must be less than p', so the second Nash equilibrium for this game results in sub-optimal vaccination levels, and disease cannot be eradicated.

Game-theoretic models have also been adapted to include stochastic effects (Fukuda et al. (2014), Eksin et al. (2017), Mohr et al. (2020), Galvani et al. (2007), Fu et al. (2011*a*)).

1.4.1 Game theory and plant disease models

The management of plant disease has inspired an expansive field of study, often focusing primarily on optimal resource use (Ndeffo Mbah & Gilligan (2010), Vyska et al. (2016), Cunniffe et al. (2015b), Mastin et al. (2020), Thompson

et al. (2018), Hilker et al. (2017), Bussell et al. (2019) and Bussell & Cunniffe (2020)). However, few studies focus on the effect of growers' individual decision-making processes on the outcome of a control scheme, instead assuming that all growers will enact control mechanisms as advised.

Whilst many studies make the comparison between disease control decisions and the prisoner's dilemma (Nowak et al. (1994), Richardson et al. (2017), Karlsson & Rowlett (2020)), there are also clear similarities between decisions in plant protection and the snowdrift problem (Milne et al. (2016); also called "chicken" or the "hawk-dove" game). The game's central concept is as follows: there are two drivers who come across a snowdrift that blocks their path. Neither can proceed on their journey, so if neither clears the snowdrift they incur a negative payoff (waiting for the snow to melt). Both drivers may choose to co-operate, contributing some effort to clearing the snow. If they do this, they will split the cost of clearing the snow (as it is cleared more quickly, with less of an investment of time and energy from each participant). Yet if only one of the drivers clears the snowdrift, they get a direct benefit (as they can now proceed on the cleared road) but paid a high energeic cost to do so. The other driver, however, can also proceed on their journey without having endured any of the negative consequences of having to clear the drift. This non-co-operative behaviour is called "free-riding", and is a major deterrent to co-operation. Unlike in the prisoner's dilemma, the incentive to "co-operate" is often enough to ensure that some individuals will adopt that strategy, lowering the risk of exploitation by others.

This has clear analogies to plant disease control. If a disease is present in an area, and no grower enacts any crop protection mechanisms, disease will become widespread and all growers will have a high probability of crop loss. If growers co-operate and coordinate their control practices, it may be possible to eliminate disease (or at least minimise losses) with reduced individual efforts from each grower. However, if one grower engages in control, and another does not, the non-controller can free-ride off the efforts of their neighbour.

However, the use of game theory in crop management models is limited. Milne et al. (2016), McQuaid et al. (2017a) and Saikai et al. (2021) all use two-strategy models where growers assess profitability using "strategic-adaptive" behaviour, comparing a grower's own outcome from the previous season with the potential profits they could earn if they adopted the alternative strategy. The growers then change strategy probabilistically based on the magnitude of the difference between the two quantities. In the Milne et al. (2016) model, growers compare their outcome with what they could expect to earn if they adopted the alternative strategy. The growers can either adopt or abandon *Bacillus thuringiensis* (*Bt*) maize, which has been modified to expresses insecticidal proteins) when faced with European corn borer (ECB; *Ostrinia nubilalis*). The growers are connected in a variety of communication networks, the structure of which will determine the value of the profit of the alternative strategy. Saikai et al. (2021) developed a similar agent-based model also based on the adoption of *Bt* maize. The profit-based decision component is identical to that of Milne, but now growers balance this consideration with the infor-

mation they get from social contacts. Both studies uses stochastic dispersal of ECB spread.

In the McQuaid et al. (2017a) model, as well as basing their decisions to switch strategies on the profits of others, growers had a range of characteristics, such as "contrariness" or "stubbornness", which modified the probabilities of joining a clean seed system (CSS) that distributed disease-free cassava. The growers could either compare their profits with growers across the entire landscape or with a subset of growers. Additionally, growers could be part of a trade network from which they sourced their planting material. It is a spatial model with stochastic disease spread (both in terms of the trade networks and spread of the vector), though the within-field "bulk-up" is deterministic. We use McQuaid et al. (2017a) as a basis for our model in Chapter 2, simplifying the spatial and stochastic components to a continuous-time deterministic model that is more mathematically tractable.

Bate et al. (2021) take a different approach, instead modelling growers' participation in a co-operative control scheme using what they call "coalition games". In coalition games, players must consider both their direct payoffs but also the effect of their actions on others who are also in the scheme. Growers must decide whether to partake in a biosecurity-related assurance scheme in which all members deploy certain biosecurity measures. When making decisions, growers not only consider the direct cost of control, but also the impact of trade restrictions *etc.* that could result from an outbreak of disease. Some growers will have a co-operative mindset, assuming that if they control for

disease, others will reciprocate, whilst others will act only in their own self-interest. The scheme can be supported by the government, which can lower the costs associated with an outbreak for members of the scheme.

In Milne et al. (2020), no consideration is given to the cost of control. Instead, growers' decision-making is based on their perceived risk of infection and belief that area-wide control is effective. These growers are influenced by the opinions of those around them, and growers are more likely to consider the opinions of those in close proximity to themselves.

The dynamics of grower behaviour in response to market pressures (amongst others) can be studied using partial adjustment models, which were originally conceived by Nerlove to study agricultural supply response. In the models, growers estimate the demand from the markets, and match their production accordingly (Nerlove (1958), Askari & Cummings (1977)). That is, they estimate what the "desired" level of production will be, and adjust their current production in line with that estimate (hence the "lag"). The "desired" level of production requires a theory of expectation formation, which can be based on rational expectations (Kennan (1979)).

Conclusions of game theory in plant disease models

Both McQuaid et al. (2017a) and Milne et al. (2016) found that allowing growers access to a wide range of information sources improved engagement in control, though Saikai et al. (2021) found that access to information on other growers' profitability could discourage the use of control. Oscillations in uptake

were also observed in all models. Growers were sensitive to the rewards offered by the control schemes, and McQuaid et al. (2017a) note that if the disease pressure is sufficiently high, growers will likely not control as the rewards will not be sufficient to overcome the coupled burden of the costs of control and high probability of loss due to infection.

As with the previous models we have discussed, Bate et al. (2021) found that growers were very sensitive to the costs of the control scheme, and that stable coalitions could only form once costs were sufficiently low, without which co-operative schemes are likely to fail.

Where growers make comparisons based on profit (Milne et al. (2016), McQuaid et al. (2017a) and Saikai et al. (2021)), all models use the same form of equations. Yet as was discussed earlier, a range of different decision-making rules have been devised in human disease models. These different decision rules based on replicator, imitation, best-response dynamics *etc.* are known to influence model dynamics (Sun & Hilker (2021)), though the exploration of their use and effect in plant disease modelling has not been investigated to date.

1.5 Control mechanisms and externalities

A key factor underpinning the strategic nature of disease control is that the decisions of each individual are interdependent; the choices of one have clear implications for others. In economics, these third-party effects are called "externalities". Externalities can be positive or negative in nature, but are usually

hard to quantify and - crucially - are not considered by the actor undertaking the action. Many disease control schemes generate positive externalities, meaning that the actions reduce the probability of infection for others (Geoffard & Philipson (1997)). For example, vaccinated individuals are less likely to infect other individuals with whom they interact, offering them some protection from disease (Figure 1.1). In this sense, the success of a disease control scheme may be self-limiting: as more individuals control for disease, the less incentive there is to control (because there is a lower prevalence of disease) as they can "free-ride" off the control efforts of others. Such control mechanisms are called "strategic substitutes": their use discourages further adoption. This may induce oscillatory dynamics, with peaks and troughs of both disease prevalence and control uptake (Fisman & Laupland (2009), Epstein et al. (2008), Milne et al. (2016), McQuaid et al. (2017a)).

Alternatively, control mechanisms that generate negative externalities are called "strategic complements". Examples of disease control mechanisms that act as strategic complements include tolerance-based therapies such as anti-inflammatory drugs that reduce the symptoms of illness without actually clearing pathogen load (Hozé et al. (2018)). This could lead to the further spread of disease and increased use of those tolerance-based therapies.

Externalities are a well-documented phenomenon in agricultural systems, with some consideration given to the externalities generated by pesticide application. These externalities can be positive, for example by reducing the need for other growers to spray for pesticides themselves (Zheng & Goodhue

(2021)). However, unintended negative effects are also possible: Grogan & Goodhue (2012) found that growers spraying pesticides in one field killed the natural enemy of a pest of a different crop in neighbouring fields. Similarly, Citrus Health Management Areas (CHMAs, which coordinate pesticide spraying between growers to combat citrus diseases) are more successful if more growers in a region participate, but benefits are still experienced with incomplete grower participation (Singerman et al. (2017))

Resistant crops have similar characteristics to pesticide-treated crops, as they also lower the likelihood a field will act as a reservoir of infection for others. Theoretical studies have shown that only a subset of growers need plant resistant crop for their benefits to be experienced widely across the community, including by growers of non-resistant crop (van den Bosch & Gilligan (2003), Lo Iacono et al. (2013), Vyska et al. (2016)). Indeed, Hutchison et al. (2010) estimate that the majority of the benefits conferred by *Bt*-resistant maize in the United States were experienced by free-riders and not those who invested in the improved crop themselves. Considering the potential externalities of a particular control mechanism can help predict the success of a control scheme, but may also afford those overseeing the scheme more control over its success by enabling them to alter the weighting of externalities through taxes, subsidies, policies, and other incentives.

1.6 Conclusions from game-theoretic epidemic models

Many of these conclusions relate specifically to vaccinations and individual uptake under a voluntary vaccination regime. However, they may be generalised to other control mechanisms that offer sufficiently high protection such as cultivars with a high degree of resistance or pesticides with a high degree of efficacy.

In voluntary vaccination regimes, the proportion of the population vaccinated will always be less than that required for herd immunity.

Under a voluntary vaccination regime, the proportion of the population electing to vaccinate will always be less than that needed for herd immunity (and, therefore, the disease will not be eliminated) (Bauch & Earn (2004),Bauch et al. (2003), Bauch (2005), Sykes & Rychtáă (2015), Galvani et al. (2007)). Similarly, if vaccination is perfectly effective, a steady-state where all individuals vaccinate cannot be reached under a voluntary vaccination scheme. Instead, situations where no individuals vaccinate or an intermediate number vaccinate are achieved. Herd immunity thresholds for vaccine-preventable diseases can be extremely high (e.g. poliomyelitis: 80-86%, measles: 91-94% (Plans-Rubió (2012))) and context-dependent (ebola: 42.2-63% (Gittings & Matson (2016))). Indeed, these sub-optimal vaccination rates may create greater damage than the case where no one vaccinates. It is known that age structure influences the

herd immunity threshold needed for disease eradication (Hoppensteadt (1974), May & Anderson (1984)), and Liu et al. (2012) found that intermediate vaccination coverage can increase the average age of those infected with chickenpox, which is typically associated with worse outcomes.

As vaccines generate positive externalities, they act as strategic substitutes and discourage widespread use. A schematic of the externalities produced by vaccinations is shown in Figure 1.1. These positive externalities induce a negative feedback loop, leading to periodic adoption and abandonment of control, which are accompanied by resurgence of disease (Bauch (2005), McQuaid et al. (2017a), Milne et al. (2016), d'Onofrio et al. (2007)).

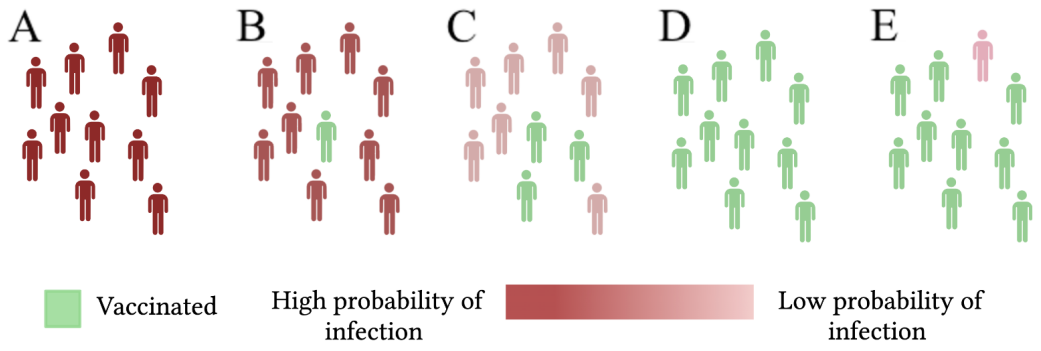

Figure 1.1: **Externalities generated by vaccination.** (A) When no one is vaccinated (green), the probability of infection (dark red) is high. (B) and (C) Once individuals in the population are vaccinated, the probability of infection for others decreases (indicated by the lightened shade of red). (D) If all individuals vaccinate, there is no risk of infection. (E) Individuals "free ride" off the protection provided by other individuals' vaccinations, largely avoiding infection without having to vaccinate themselves. Vaccination decisions are largely irreversible, but such dynamics could apply either when individuals express an intent to vaccinate or when seasonal/periodic vaccination is possible. Created using Biorender.com.

The structure of contact networks influences decision dynamics

Contact networks (both for disease spread and dissemination of information) have a strong influence on an individual's decision dynamics, though the precise impact factors such as connectedness or structuring have is varied and highly dependent on other factors (namely the cost of the control mechanism). Heterogenous networks, where individuals are only in contact with a subset of the population, may lead to opinion clustering (Salathé & Bonhoeffer (2008)). If these opinions are sufficiently sceptical of disease control, disease may still invade an otherwise well-protected community (Eames (2009)).

Milne et al. (2016) found that when growers considered a range of local and population-level information (including information from extension workers), losses due to infestation with European corn borer were minimised, particularly when compared with a scenario where growers only considered local information. Similarly, a high degree of community structure (where connections between nodes are unevenly distributed across the population) can cause many localised epidemics to occur of varying size and duration (Salathé & Bonhoeffer (2008)). Similarly, both Fu et al. (2011*a*) and Zhang et al. (2010) found that individuals with more social contacts are more likely to vaccinate, as they are at a higher risk of infection. Fu et al. (2011*a*) also found that when an individual can only access local information sources, they are less likely to free-ride and pro-social vaccination behaviour becomes more likely.

Cumulatively, models where games are played across a network of indi-

viduals find that nodes with a higher number of connections are more likely to engage in disease control. This correlates with empirical studies, which found that those who communicate with neighbours and/or specialised extension workers may be more likely to participate in area-wide pest management (AWPM) of plant diseases (Garcia-Figuera et al. (2021), Sherman et al. (2019)).

Many different factors influence model outcomes

A range of parameters, from risk perception to cost of the control mechanism, impacts how decisions will be made.

Unsurprisingly, the cost of the control has a strong influence on its uptake. Many studies found that high costs alone were sufficient to deter individuals from controlling for disease (e.g. Bauch & Earn (2004), Liu et al. (2012), Jijón et al. (2017), Delabouglise & Boni (2020), Fu et al. (2011*a*), Brettin et al. (2018)), perhaps necessitating some sort of intervention to encourage uptake. Bate et al. (2021) found that external, government-provided incentives were required to encourage participation in biosecurity schemes aimed at preventing plant disease. The incentives did not need to be direct financial subsidies (they could, for example, be relaxation of trade restrictions for those who are part of the biosecurity scheme) but they did have to increase the payoff to controllers.

Nixon et al. (2017) found that the willingness to pay for control depended on the risk perception of the farmer; if they assessed their flock of sheep to be at high risk of infection, they were willing to pay for diagnostic tests. If

growers perceived a low probability of infection, however, such tests were rarely adopted. Similarly, increased "selection" (analogous to a strong willingness to imitate neighbours) can have a detrimental effect on vaccine uptake, as individuals are more sensitive to the benefits of free-riding (Fu et al. (2011*a*)).

Efforts to educate the population on the risks associated with diseases have the potential to positively influence control uptake (Brettin et al. (2018), McQuaid et al. (2017a)), though the extent of their influence is hard to gauge. Any educational campaigns would primarily act on parameters related to disease-avoidance behaviour or risk perception, potentially lowering the gap between perceived and actual risk (Funk et al. (2010)). In cases where perceived risk has been inflated by exposure to misinformation, educational campaigns would encourage the use of a control mechanism.

Though not based on game-theoretic principles, Milne et al. (2020) used opinion dynamics to model how growers decide to join an AWPM scheme. The model does not consider any economic incentives, but only how the opinion of growers changes through their contact with growers, some of whom may have very different beliefs on the benefits of AWPM. The growers could also receive information through extension agents, who are trained to educate growers on the risks of disease in their area. Growers can prioritise the opinions of those who are already more similar to their own, minimising the impact of encountering a diverse group of growers. The educational information provided by the extension agent had relatively little impact, as did the range of opinions listened to, especially when compared with factors such as the efficacy of con-

trol. However, if control is effective, regular contact with extension workers can bolster participation in AWPM, ensuring better control outcomes.

1.7 Overview of case studies

In this thesis, we focus on two pathosystems and model the uptake of control mechanisms for each. The first case study we consider in Chapter 2 is cassava brown streak disease (CBSD) and its management through clean seed systems. CBSD is caused by either cassava brown streak virus (CBSV, Monger et al. (2001)) or Ugandan CBSV (UCBSV; Patil et al. (2011)), and is widespread across East and Central Africa (Tomlinson et al. (2018)). CBSV and UCBSV are spread by *Bemisia tabaci* (Maruthi et al. (2005)), a whitefly vector, and through vegetative propagation of infected cuttings. As yet, there are no resistant cultivars available (Tomlinson et al. (2018)), though there are some that are tolerant to infection. Instead, clean seed systems (CSSs) have been devised as a means of cultural control. In CSSs, growers are provided with cassava cuttings that are guaranteed to be virus-free, reducing long-range viral spread through the trade of infected cuttings and preventing the introduction of CBSD to new areas. CSSs have been piloted in several countries (Tomlinson et al. (2018)).

In Chapters 3 and 4 we model the management of tomato yellow leaf curl virus (TYLCV) via the deployment of varieties that are either tolerant or resistant to disease. Also transmitted by *B. tabaci*, TYLCV is a major threat to global tomato production and has been detected in nearly seventy countries and territories (www.cabi.orgiscdatasheet55402#toDistributionMaps). Infec-

tion can be devastating, causing up to 100% yield loss (Czosnek & Laterrot (1997)). Consequently, many different management schemes have been devised, including insecticide treatments (Rojas et al. (2018)), quarantining of plants (Polston et al. (1999)), the application of biocontrol agents (Stansly et al. (2004)) and the removal of alternative hosts (Papayiannis et al. (2011)). In Chapters 3 and 4, we model the use of TYLCV-tolerant and -resistant varieties of tomato, as well as the effect of roguing infected fields.

1.7.1 Economic context of the case studies

Cassava is a staple crop in much of sub-Saharan Africa; in Zimbabwe, for example, 30% of the population relies on cassava as a staple part of their diet. Most of the cassava is grown on fields of less than 1 ha in size (Chikoti et al. (2019)).

In the models presented in this thesis, we focus on the subsistence growers who use these smallholdings to grow cassava that they will then consume. The growers will then sell any excess that they have (a practice carried out by around 20% of Zambians). The income of these growers is hard to estimate, but given Zimbabwe's Poverty Assessment Survey of 2011/2012 estimated that 61.3% of rural households earned less that US$100 a year (UNDP, 2013), we assume that the income of the growers we model here will be low. We also assume that all growers will come from the same income bracket (i.e. that there will not be large income disparities between growers).

For tomato production, we assume that a similar system is in place. Tomato

is the third most popular vegetable crop to be grown by smallholder farmers in Zimbabwe, with on average 37.1

In both cases, limited income and resources, alongside a lack of knowledge of the disease and farming practices, means that growers cannot easily access different control mechanisms (particularly expensive chemical controls; (Mukarumbwa et al. (2017))).

1.8 Empirical evidence linking human behaviour and disease management decisions

1.8.1 Experimental studies on health-related behaviours

Collecting data on how individuals behave is challenging, as many experimental settings are necessarily artificial. However, some efforts have been made to develop agent-based participatory simulations of epidemics (Wilensky & Stroup (1999), Maharaj et al. (2011), Delaney et al. (2013)). In Merrill et al. (2019a) and Merrill et al. (2019b), the game's players are in charge of a virtual farm, intending to earn units of virtual money (which are sometimes translated into cash payouts for participants). The growers could either comply with costly biosecurity measures and reduce their risk of infection or ignore protocols and risk infection. Similar simulations have been used to study an individual's response to how information is conveyed (e.g. graphically or linguistically; Merrill et al. (2021)), finding that graphic portrayal of risk levels using simple language was most effective. The approach has obvious limitations, as the

simulations are divorced from reality and therefore may not accurately reflect the decisions an individual would make.

Simulations have been used to study specific disease systems, such as the effects of risk attitudes on social distancing behaviours during an influenza epidemic (Reluga (2010)), or more general systems (such as Chen et al. (2013), which modelled general "disease-prevention" behaviours for a disease that can re-infect hosts). The latter found that individuals responded positively to reduced cost, and more engaged in self-protection behaviour. This interacted with other factors, as the players were more sensitive to information regarding disease prevalence if the cost of the control option was low.

Simulated games need not be virtual. For example, vaccination games can be held in laboratory settings, with players making simultaneous decisions on whether to accept a hypothetical vaccine (Lim & Zhang (2020)). Such studies, though small, support theoretical studies that emphasise the importance of the relatively large benefits of vaccination.

1.8.2 Nudge theory and health behaviours

Nudge theory (Thaler & Sunstein (2008)) posits that changing certain aspects of an individual's environment can influence their decisions in a predictable fashion. The nudges do not limit an individual's range of choices but often alter how information or options are presented to the individual; opt-out schemes for organ donations, for example, are nudges (Thaler & Sunstein (2008)).

Some experimental studies have been carried out investigating the effect of

different nudges on the vaccination intentions of individuals. A meta-analysis (Reñosa et al. (2021)) found that seven categories of nudges had significant positive effects: novel or targeted messaging; direct incentives; reframing the decision (e.g. making vaccinations opt-out); invoking or establishing social norms during communications; using dramatic narratives to evoke emotional responses; communicating messages through a trusted, non-medical source and reframing the outcomes (e.g. communicating the benefits of vaccination, rather than the consequences of being unvaccinated). There are some overlaps in these conclusions with those derived from the simulated games, particularly those related to incentives and the communication of benefits.

Nudge theory has been applied in some agricultural settings. Nudges such as monetary rewards or increased social capital were considered effective at encouraging food safety measures during pork production in Vietnam (Hennessey et al. (2020)). Increased access to price information encouraged farmers in Nigeria to divert more resources toward growing commodities (Belay & Ayalew (2020)). Providing free delivery for fertiliser increased its use amongst Kenyan farmers, boosting yields (Duflo et al. (2011)). However, there are no studies to date on the effect of nudges on the disease decisions made by growers.

1.8.3 Surveys of behaviour changes

Aside from simulated games, surveys are often used to gather information on individuals' responses to the threat of disease. Surveys do suffer from limitations, such as self-selection bias of respondents or sampling bias from surveyors

(Sedgwick (2013)), and results are often context-dependent. Nevertheless, they can provide useful information on trends in behaviour.

Previous surveys have estimated that around 75% of individuals would avoid public transportation during an influenza pandemic (Sadique et al. (2007)), and 60% said that they would restrict shopping trips. During the 2009 H1N1 ("swine flu") epidemic, however, self-reported engagement with disease avoidance behaviours was low (4.9%), though 37.8% engaged in behaviours recommended by the government (such as increased hand-washing). Overall, amongst these participants, anxiety related to swine flu was low (24%), perhaps affecting the degree to which individuals will undertake protective or avoidance behaviours.

The recent outbreak of COVID-19 has provided a wealth of opportunities to study how individuals respond to disease threats. For example, early in the pandemic (June 2020), the vast majority of survey respondents in North America stated they "strictly" followed health guidance disseminated by their local government (Wang et al. (2021)). However, the majority of the respondents were women (who are known to be more likely to engage in health-protection practices; Hibbard & Pope (1983), Pinkhasov et al. (2010)), potentially impacting the conclusions.

The willingness to comply with health advice will vary over time. In Ireland, though initial compliance with government regulations relating to the COVID-19 pandemic was generally high, as time progressed, compliance fell (Kearney et al. (2022)). Generally, those who reported breaking restrictions

did so for work-related travel, indicating that economic factors underpin an individual's ability to comply with regulations.

1.8.4 Conclusions from empirical studies

Across human, veterinary, and plant epidemiology, lack of certainty relating to the risk of infection has been cited as a barrier to adopting control strategies (Huang & Liu (2022), Karafillakis et al. (2019), Roberts et al. (2012) Singerman & Useche (2019), Garcia-Figuera et al. (2021)). Individuals were less likely to engage in disease-avoidance behaviour during the H1N1 pandemic if they were uncertain about the accuracy of the information regarding the epidemic's severity (Rubin et al. (2009)). Similar results were found for the recent COVID-19 pandemic (Wang et al. (2021)). Some evidence suggests that individuals will pay more to reduce uncertainty (Merrill et al. (2019a)).

Cost is an often-cited factor that influences behaviour and is included in many theoretical models. In these models, costs can be pecuniary (e.g. McQuaid et al. (2017b)) or related to the health-related consequences of infection (e.g. Bauch & Earn (2004)). Both simulated games and surveys show a robust behavioural response to these costs. Other studies show that individuals were willing to miss pre-paid flights during the H1N1 pandemic (Fenichel et al. (2013)), representing a "willingness to pay" to avoid infection.

Recent experience with infection can also increase the likelihood of engaging with control (Elbers et al. (2010); Merrill et al. (2019b), Chen et al. (2013)). Related to the idea of "psychological distancing", individuals may

be less likely to take disease-preventing actions if they have not had recent exposure to disease. There is experimental evidence for importance of other factors such as the biosecurity practices of coworkers on the use of control measures (Trinity et al. (2020)). Interestingly, in a meta-analysis of surveys of care givers' decisions to vaccinate their children, many respondents cited herd immunity or considerations of the health of others as a factor, even if they went on to refuse vaccination.

Many of these conclusions fall in line with Protection Motivation Theory (PMT; Floyd et al. (2000)). Under PMT, individuals will undertake health-preserving actions if they perceive the risks of illness to be greater than any cost associated with the avoidance action. The decision hinges on how an individual perceives four factors: the risks posed by illness, their own vulnerability, the efficacy of the potential action and their own ability to undertake such an action (*self-efficacy*). PMT has many similarities with the Health Belief Model (HBM; Janz & Becker (1984)), which again focuses on the underpinnings of an individual's health-related behaviours. The HBM, however, is more limited in scope, only focusing on the value an individual places on their health and the belief that an action will help them to achieve good health. The two theories, however, are largely complementary, and there is evidence supporting both in the studies we have outlined above.

1.9 Aims of this thesis

The overarching aim of this thesis is to develop epi-economic models that integrate aspects of growers' decision-making into models of disease spread. In

particular, we are interested in how assessments of profitability affect a growers' decision to use a control mechanism, and how the externalities generated by other growers affect long-term disease and control outcomes.

In the first instance, we will compare different models of grower behaviour and examine how model formulation affects grower participation in control schemes (Chapter 2). These models differ in terms of the "decision rules" used by the growers, varying whether growers compare solely based on the expected profits of a particular strategy (*rational expectations*) or on a mix of their previous and expected profits (*strategic-adaptive expectations*). To date, a systematic investigation of the effect of the decision rule has not been undertaken, so how they impact the long-term dynamics of decision-making is unknown. In our models, growers will have only two choices - to control or not control. We will examine these model formulations using the management of cassava brown streak disease (CBSD) via clean seed systems (CSS) as a means of control. We investigate the models' response to changes in economic and epidemiological parameters. We also vary the accuracy of the information on these parameters that growers use in their estimates, breaking a common assumption of economic models (that growers have perfect access to information).

For the remainder of the thesis, we use one of the strategic-adaptive formulations developed in Chapter 2. In Chapter 3, we will use this model to examine the effect of deploying crop varieties that are tolerant or resistant to disease, with the specific aim of determining the effect of the externalities generated by each crop type on the uptake of control. Such an investigation has

not been conducted to date. As both tolerant and resistant crops are being deployed as a more sustainable and environmentally-friendly means of disease management, we aim to address this gap in knowledge here. We will again examine the response to changes in economic and epidemiological parameters, this time focussing on the efficacy of each control option, which moderates these externalities. We will carry out these investigations using tomato yellow leaf curl virus (TYLCV) as a case study. Again, in this model growers will only be able to choose between two crop types (either tolerant/unimproved crop or resistant/unimproved crop).

In Chapter 4, we will expand the decision model developed in Chapter 3 to include three different strategy options. In the plant epi-economic literature to date, growers have only been given a binary choice of control strategies. The effect of including a third strategy is therefore unknown. We will use the disease model for TYLCV developed in Chapter 3, giving growers the choice between three crop types: tolerant, resistant, and unimproved. We will investigate how to incentivise an optimal solution to the model using Pareto optimality and Gini coefficients. The former determines the set of model solutions that provide the best outcome for all invested parties (in this case, the growers and a subsidy-provider who controls the price of crop). The latter is a measure of fairness and will indicate if one party's outcome is being prioritised over the other's.

Finally, in Chapter 5, we investigate the effect of adding space and stochasticity into our models. We will develop a spatial-stochastic model for both of

our case studies (clean seed systems and CBSD and improved crop types for TYLCV). We will use the model of CBSD to investigate similar questions to those in Chapter 2 (the effect of parameters and parameter mis-estimation), as well as establish the role of spatial heterogeneity and stochasticity on growers' participation the CSS. We will investigate similar questions for the model of TYLCV in Chapter 2, but additionally will investigate if spatial clustering of strategy choices occurs.To study this, we include networks of growers that vary in scale, limiting the amount of information a grower has access to regarding the profitability of each strategy.

Chapter 2: Models of grower decision-making[1]

2.1 Introduction

Human behaviour is intuitively important in the control of plant infectious disease, as individuals often face choices when adopting infection-limiting behaviours. This behaviour has been widely considered in the context of human (e.g. Bauch et al. (2003), Bauch & Earn (2004), Salathé & Bonhoeffer (2008), d'Onofrio et al. (2011), Cascante-Vega et al. (2022); reviewed in Funk et al. (2010) and Chang et al. (2020)) and animal (e.g. Hidano et al. (2018), Bucini et al. (2019), Delabouglise & Boni (2020), Mohr et al. (2020)) diseases, though there have been fewer studies for plant diseases (with the exception of Milne et al. (2016), McQuaid et al. (2017a), Szyniszewska et al. (2021), Milne et al. (2020), Bate et al. (2021) and Saikai et al. (2021)). Where studies do consider individual's decisions there is much variation in how decision-making is

[1]The work presented in this chapter is based on Murray-Watson *et al.*, 2022.

modelled, although it clearly will affect the outcomes of their decisions.

We assume that decisions regarding disease control are intrinsically linked to disease prevalence. Clearly, they must also depend upon considerations such as the cost of control, the consequences of infection and the perceived risk of becoming infected (Kaup (2008), Sherman & Gent (2014), Garcia-Figuera et al. (2021)). Additionally, and crucially, decisions made by one grower will influence the decisions of another. Voluntary disease control programmes can therefore be viewed as a collective action problem (also termed social dilemmas; Siegal et al. (2009)) as they generate externalities that affect the outcomes of those not involved in the programme. By reducing or eliminating their own chance of infection, growers who control also lower the probability that their neighbours will be infected, thereby generating a positive externality that disincentivises their neighbours' engagement in disease prevention (Geoffard & Philipson (1997)).

Such "free-riding" behaviour is indirectly encouraged when there is higher overall participation in control schemes, as growers are less likely to become infected whenever the proportion controlling is high. Yet if too many growers engage in free-riding behaviour, there will be a resurgence of infections, which may make disease control more likely. These feedback loops are believed to be a major reason why some public health schemes for human disease, such as subsidised vaccination campaigns, have failed to lead to disease eradication (Geoffard & Philipson (1997)). Consequently, the success of voluntary control policies may be self-limiting. Indeed, perceived risk of other growers benefiting

from an individual's control policies are a prominent reason why growers do not participate in control schemes (Kaup (2008), Singerman & Useche (2019)).

2.1.1 Game theory and assessing profitability

In Chapter 1, we outlined how game theory has been used in previous epidemiological models. These models were often based on evolutionary game theory (e.g. Bauch (2005) and Delabouglise & Boni (2020); for non-epidemic uses see: Riechert & Hammerstein (1983), Roca et al. (2009), Hummert et al. (2014), Anderson (1984), Nowak et al. (1994), Pacheco et al. (2014)). In this formalism, in a two-strategy model, the performance of the player's own strategy is compared against that of the average performance of the population. If the payoff is less than that of the population, the focal player changes strategy; if it is higher, they remain with their current strategy. For plant disease, this would involve growers comparing the average profit of all of those using their own control strategy with the average profit over all growers in the population (we call this the "strategy vs population" model). Of course, this assumes, perhaps unrealistically, that growers will base their decision-making upon the profit of all those using the same control strategy.

An alternative means of assessing profitability is used in Milne et al. (2016), McQuaid et al. (2017a) and Saikai et al. (2021) (though the inclusion of grower characteristics in McQuaid et al. (2017a) means it deviates from pure game-theoretic assumptions of rationality as growers may adopt a strategy that will earn them lower profits). In these two-strategy models, growers compare their

own profit with the average profit of those playing the alternative strategy (the strategy that the grower does not currently use; for a controller, this would be non-control and vice versa). We henceforth call this the "grower vs alternative" model. Ostensibly this seems a more likely comparison a grower will make, as it requires less information and focuses more closely on a grower's own performance, rather than all who act similarly to that grower. Other possible models combine elements of both approaches, since growers compare their strategy with the expected profit of the alternative strategy ("strategy vs alternative" model) or compare their own profit with the average of the population ("grower vs population" model). To our knowledge, the "grower vs population" and "grower vs alternative" models have never been compared. A schematic showing each type of comparison is shown in Figure 2.1.

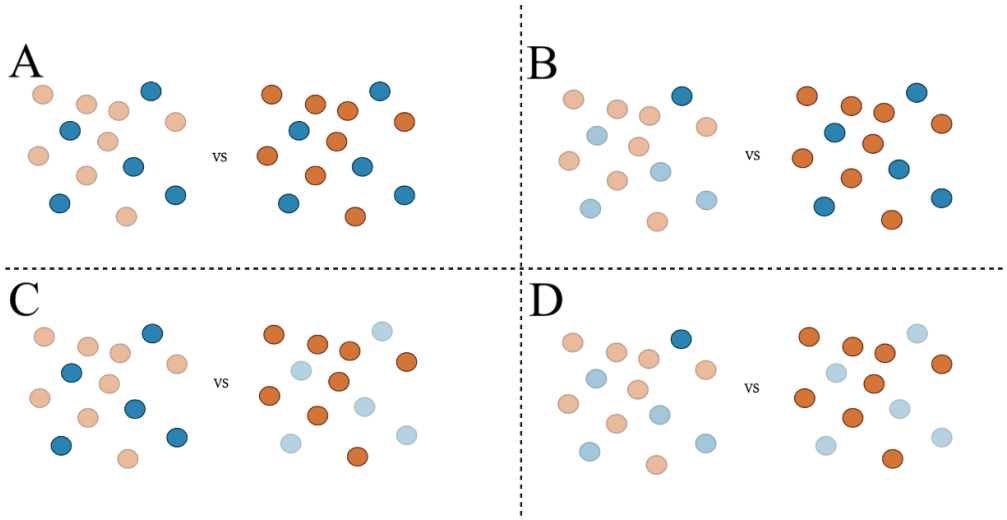

Figure 2.1: **Schematic of different means of comparison.** Each circle represents a grower, and there are two possible strategies: orange or blue. (A) Growers compare the expected value across their entire strategy (blue) with the value across the entire population ("strategy vs population"). (B) Growers compare their outcome from the previous season with the expected value across the entire population ("grower vs population"). (C) Growers compare the expected value across their entire strategy (blue) with the expected value of the alternative strategy (orange) ("strategy vs alternative"). (D) Growers compare their outcome from the previous season with the expected value of the alternative strategy ("grower vs alternative"). (A) and (C) are based on rational expectations, whereas (B) and (D) are based on strategic-adaptive expectations. Created using Biorender.com.

The two model classes ("strategy vs" and "grower vs") can be broadly classified based on the economic concepts of "rational" and "adaptive" expectations, respectively (Mlambo (2012)). If an individual forms their expectations rationally, they assess the information available at any instant and then extrapolate/determine what that means for the future. This has a clear relationship with our "strategy vs" models, which use the current probabilities of infection as a proxy for what the future probabilities will be. Conversely, adaptive expectations are based solely on historical outcomes. Rather than simply being "adaptive", our "grower vs" models can instead be viewed as "strategic-

adaptive" (Fenichel & Wang (2013)), accounting for prior experience whilst still accounting for future events.

Hypothetical scenarios in which each of these model types could potentially apply can be constructed. If a grower is part of an area-wide control scheme (such as the citrus health management areas (CHMAs) for citrus diseases in the United States of America (Council et al. (2010)), or to manage Queensland fruitfly populations in Australia (Florec et al. (2013))) that only includes a subset of the population, they may wish to compare the profits of the scheme with the expected profit of the entire population (the "strategy vs population" comparison). The "strategy vs alternative" comparison may be used if a grower is part of an area-wide control scheme but wishes to join another scheme with a different means of control. If a particular strategy may confer a market advantage to the grower (for example, if pesticide use is widespread, they may wish to stop spraying to then market themselves as "organic"), the grower may compare their outcome with the expected profit of the population ("grower vs population"). Finally, if a grower is using a particular control strategy and is then approached by an extension worker who proposes a different strategy, they may employ the "grower vs alternative" comparison.

2.1.2 Case study: Cassava Brown Streak Disease

To examine the effects of these different models of growers' decision-making in a concrete setting, we use a simple model describing the spread of Cassava Brown Streak Disease (CBSD) based on the model presented in McQuaid et al.

(2017a). Cassava (*Manihot esculenta*) is a staple food in Sub-Saharan Africa, where it is predominantly grown as a subsistence crop. Cassava production is threatened by CBSD, a viral disease caused by either cassava brown streak virus (CBSV, Monger et al. (2001)) or Ugandan CBSV (UCBSV; Patil et al. (2011)). Infection results in necrosis of the stems and tubers, leading to yield losses of up to 70% (Hillocks et al. (2001)). Coupled with its increasing spread from east to west Africa, this positions CBSD as a major challenge to cassava cultivation across the region (Legg et al. (2014)).

Both viruses are transmitted horizontally by a whitefly vector (*Bemisia tabaci*; Maruthi et al. (2005)) and vertically through the replanting of infected stem cuttings. Informal trade of cassava cuttings is widespread amongst growers, though the subtlety of symptoms often means that the practice contributes to vegetative propagation of infected material (Chipeta et al. (2016)) and spread of CBSD between growers (McQuaid et al. (2017b)).

This potential for vertical transmission means that clean seed systems (CSSs) are a plausible CBSD management strategy (Legg et al. (2017)). CSSs disseminate virus-free planting material, thereby avoiding the vegetative propagation of infected material. The success of CSSs will depend on grower behaviour, as participation will be governed by the balance of costs and benefits of the scheme. This, and the combination of trade- and whitefly-mediated pathogen transmission, means that a grower's profit will depend on the action of others.

2.1.3 Aims of this chapter

The overall objective of this chapter is to investigate how including grower behaviour in plant epidemic models affects the outcome of control schemes, and how the factors influencing behaviour can best be manipulated to encourage control uptake. We use our behavioural model alongside the CBSD case study to address the following: (1) How does the formulation of the profit comparison (i.e. "strategy vs" or "grower vs") affect the model and its results, and can a scenario where all growers use the CSS be attained? (2) How does grower participation in the CSS depend on epidemiologically and economically important parameters, and how can high levels of control be encouraged? (3) How does systemic uncertainty in epidemiological parameters affect participation in the CSS? Though the "strategy vs" models are mathematically tractable and therefore allow for the derivation of analytical expressions, our view is that it is less likely that growers will make their assessment of profitability based on every other grower using the same strategy as they are using, rather than just their own outcomes. Thus, after our initial comparisons (Question 1 above), we focus solely upon the "grower vs alternative" model to investigate the effect of parameters and systematic uncertainty on CSS participation (Questions 2 and 3 above).

2.2 Methods

2.2.1 Epidemiological model

Our model (Figure 2.2(A)) is a simplified representation of a CSS. Fields are classified by infection status (susceptible, S, or infected, I), as well as whether their growers currently control, i.e. used certified clean seed the last time the field was planted (subscripts: controller, C, or non-controller, N). We assume each grower cultivates only a single field, and so use the terms "grower" and "field" synonymously. Infection status and control status are binary classifications; we do not model within-field spread of disease, and assume controllers plant only certified virus-free material.

Susceptible fields are vulnerable to horizontal infection (at rate β) due to viruliferous vectors moving from infected fields (fields of class I_C and I_N) (Table 2.2.2). Vertical infection also occurs via infected propagation material. We assume that the propagation material used by controllers is never infected. The probability of a non-controller becoming vertically infected depends jointly on the probability of acquiring infected planting material from an individual infected field (p) and the prevalence of disease at the time of planting $\left(\frac{I_C+I_N}{N}\right)$. The season length ($1/\gamma$, where γ is the rate of harvesting) is independent of the control strategy adopted in, and infection status of, any given field. Since we use a continuous time model, fields are asynchronously harvested and replanted (Madden et al. (2007)), a plausible assumption for cassava cultivation in much of Sub-Saharan Africa (Szyniszewska (2020)).

To integrate behavioural dynamics, we represent the decisions made by growers at the time of planting by *switching terms*. These reflect the probability of growers switching strategy for their following crop, based on their current strategy and the current state of the system, as well as, potentially, whether their previous crop was infected. In general, we distinguish four switching terms:

$$z_{SC} = \mathbb{P}\left(\text{switch from } C \text{ to } N | \text{previous crop remained uninfected } (S)\right), \quad (2.1)$$

$$z_{IC} = \mathbb{P}\left(\text{switch from } C \text{ to } N | \text{previous crop was infected } (I)\right), \quad (2.2)$$

$$z_{SN} = \mathbb{P}\left(\text{switch from } N \text{ to } C | \text{previous crop remained uninfected } (S)\right), \quad (2.3)$$

$$z_{IN} = \mathbb{P}\left(\text{switch from } N \text{ to } C | \text{previous crop was infected } (I)\right). \quad (2.4)$$

As described below, the probabilities encoded in the switching terms depend on growers' assessments of likely profits, and thus depend on the comparisons we assume are made by growers.

Our general model is

$$\frac{dS_C}{dt} = \gamma\theta_C - \beta S_C\left(I_C + I_N\right) - \gamma S_C, \quad (2.5)$$

$$\frac{dI_C}{dt} = \beta S_C\left(I_C + I_N\right) - \gamma I_C, \quad (2.6)$$

$$\frac{dS_N}{dt} = \gamma\theta_N\left(1 - \frac{p(I_C + I_N)}{N}\right) - \beta S_N\left(I_C + I_N\right) - \gamma S_N, \quad (2.7)$$

$$\frac{dI_N}{dt} = \gamma\theta_N\left(\frac{p(I_C + I_N)}{N}\right) + \beta S_N\left(I_C + I_N\right) - \gamma I_N, \quad (2.8)$$

where:

$$\theta_C = S_C(1 - z_{SC}) + I_C(1 - z_{IC}) + S_N z_{SN} + I_N z_{IN} \tag{2.9}$$

$$\theta_N = S_C z_{SC} + I_C z_{IC} + S_N(1 - z_{SN}) + I_N(1 - z_{IN}) \tag{2.10}$$

A schematic if the model is given by in Figure 2.2(A).

2.2.2 Parameterisation

Baseline parameter values for CBSD were taken from previous studies where possible (Table 2.1). The rate of secondary infection, β, was set by matching the results of our model against McQuaid et al. (2017a). In particular, we used the baseline behaviour of that model to motivate the choice that, in the absence of trade-mediated transmission, with 1% of fields initially infected and with no option of controlling for disease, 50% of fields would be infected within 10 seasons. Importantly, here and in the rest of the thesis the term “season" refers to the average length of time a field spends planted before it is harvested (300 days). To only account for the horizontal transmission, the probability of vertical transmission, (p) was set to zero when matching these results.

The value of 60% of fields' potential profit lost due to infection was assumed, to account for both the loss of starch content – which causes up to 40% loss of yield (Ephraim et al. (2015)) – and the reduced market value of infected materials (Hillocks et al. (2001)).

Vertical transmission requires cuttings taken from infected plants to be re-

Table 2.1: Summary of parameter values.

Parameter	Meaning	Value	Reference
$1/\gamma$	Length of the growing season	300 days	McQuaid et al. (2017a); Jeger et al. (2004)
β	Rate of secondary infection	8.93×10^{-6} day^{-1} field^{-1}	Calibrated to McQuaid et al. (2017a)
p	Probability of planting infected cuttings	0.8	Assumed (see main text)
η	Responsiveness of growers	10	Assumed (see main text)
Y	Maximum yield	1	All values scaled relative to yield
L	Loss due to infection	0.6	Hillocks et al. (2001); Ephraim et al. (2015)
ϕ	Cost of control	0.25	McQuaid et al. (2017a)
N	Total number of fields/growers	750	Illustrative
$S_C(0)$	Initial proportion of susceptible controllers	$0.1N$	Illustrative
$I_C(0)$	Initial proportion of infected controllers	0	Illustrative
$S_N(0)$	Initial proportion of susceptible non-controllers	$0.89N$	Illustrative
$I_N(0)$	Initial proportion of infected non-controllers	$0.01N$	Illustrative

planted and cause infection in the next season. The probability of vertical transmission is therefore reduced by selection, in which growers avoid replanting visibly infected stems (van den Bosch et al. (2007)) and by reversion, in which low viral titres mean healthy cuttings can be taken even from infected plants (Gibson & Otim-Nape (1997)), a process that has been acknowledged for a range of virus diseases (Mohammed et al. (2016)). It is difficult to quantify these effects into a single parameter value; we therefore take $p = 0.8$ as a pragmatic but arbitrary default in which vertical transmission at the field scale often, but not always, occurs.

Growers' responses to differences in profit are notoriously difficult to quantify (Milne et al. (2020)), and here we were forced to assume a value for our parameter describing the responsiveness of growers (η). An intuition for our default selection of $\eta = 10$ per unit of profit follows by noting that, using the default costs and losses in the "strategy vs population" model, a controller will have an approximately 80% chance of switching to become a non-controller if there is sufficient infection in the rest of the system such that it is almost certain they will become infected over the next season (i.e. if the probability of infection of a controller, $q_C, \approx 1$; Equation 2.17).

2.2.3 Strategies, outcomes and profits

For simplicity, we assume only a single control strategy is available, the CSS. Thus, there are only two pure strategies available to growers at the beginning of each season: to control for disease, or to not control (Figure 2.2(B)). Within each strategy, there are two outcomes realised at the time of harvesting: the grower's field may remain uninfected or have become infected.

Each strategy $\times$ outcome combination results in a different payoff (Figure 2.2(B)). An uninfected field generates yield Y, with loss of income, L, in

infected fields. Control incurs a cost ϕ. The profit for each outcome is:

$$P_{SN} = \text{Profit for non-controller who remains susceptible} = Y, \tag{2.11}$$

$$P_{IN} = \text{Profit for non-controller who becomes infected} = Y - L, \tag{2.12}$$

$$P_{SC} = \text{Profit for controller who remains susceptible} = Y - \phi, \tag{2.13}$$

$$P_{IC} = \text{Profit for controller who becomes infected} = Y - \phi - L, \tag{2.14}$$

Rational growers will never control if $L < \phi$, since then the cost of clean seed exceeds the loss due to infection. We therefore only consider $L > \phi$, from which it follows that:

$$P_{SN} > P_{SC} > P_{IN} > P_{IC}. \tag{2.15}$$

2.2.4 Risks of infection

We initially focus on the case in which growers can estimate instantaneous risks of infection precisely. In particular, we assume that growers use their current instantaneous probability of infection as an estimate of their instantaneous probability of horizontal infection (p(Horiz)) over the next season:

$$\begin{aligned} p(\text{Horiz}) &= \frac{\text{Instantaneous infection rate}}{\text{Instantaneous infection rate} + \text{Harvesting rate}}, \\ &= \frac{\beta(I_C + I_N)}{\beta(I_C + I_N) + \gamma}. \end{aligned} \tag{2.16}$$

For controllers, clean seed rules out vertical transmission. Equation 2.16 therefore sets a grower's estimate of the probability of infection next season if they

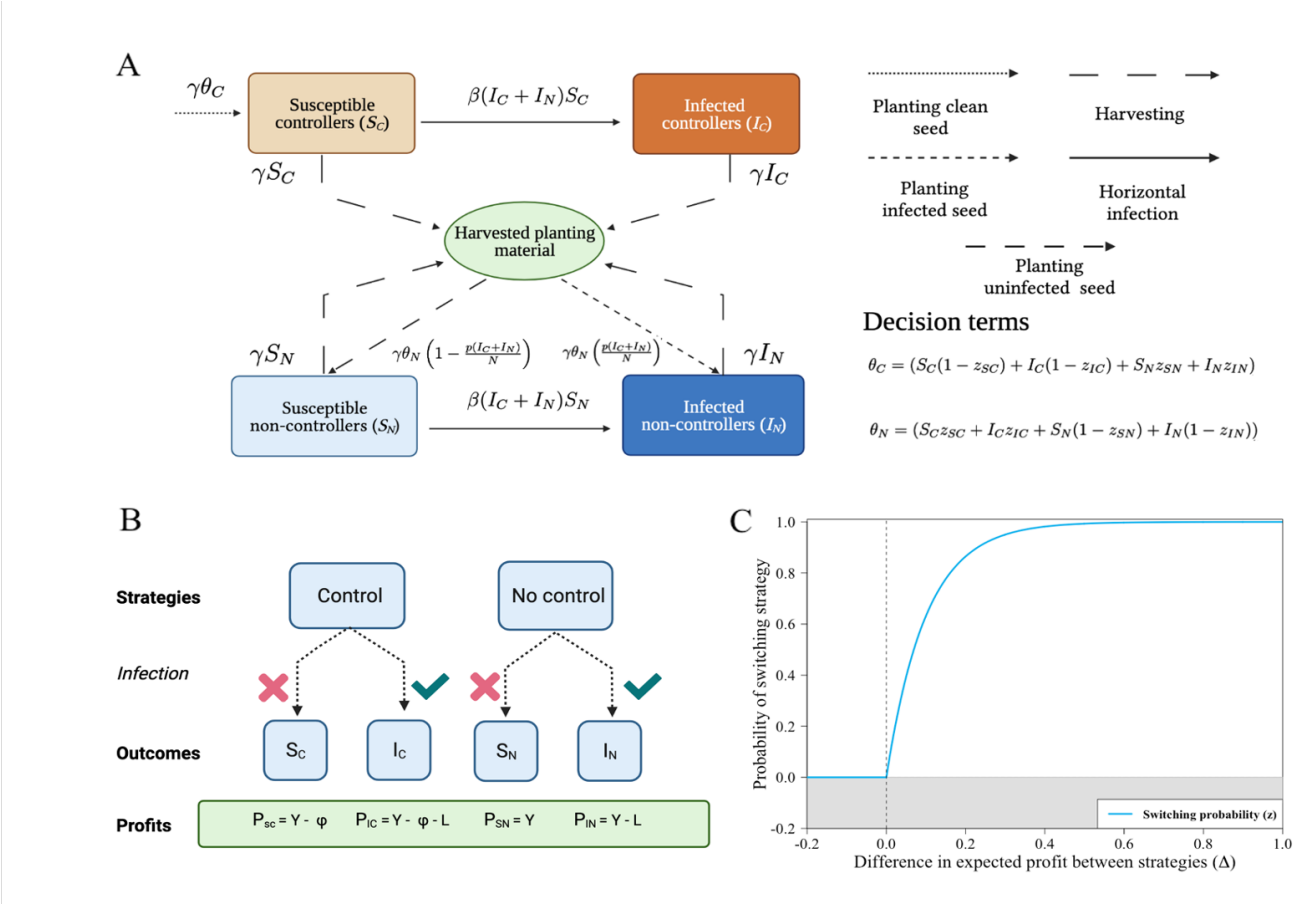

Figure 2.2: **Epidemiological and behavioural models.**(A)The epidemiological model distinguishes growers who control (i.e. plant clean seed) from those who do not. Only non-controllers can become infected via vertical transmission, although all growers are subject to horizontal transmission due to movement of vectors from infected fields (Equations 2.5-2.8). When replanting growers potentially switch strategies. (B) Strategies, outcomes and profits; the latter account for the loss of yield if infected and the cost of participating in the control scheme. (C) Decisions to switch are based on a switching function, parameterising the probability of switching strategy z as a function of Δ as difference in predicted profits. Figures (A)-(B) were created with BioRender.com.

were to choose to control (q_C):

q_C = Grower's estimate of the probability of infection next season if control is adopted,

$$= \frac{\beta(I_C + I_N)}{\beta(I_C + I_N) + \gamma}. \tag{2.17}$$

For non-controllers, vertical infection must also be considered. We again assume growers can estimate the relevant instantaneous probability of vertical transmission ($p_N(\text{Vert})$) with perfect accuracy:

$$\begin{aligned} p_N(\text{Vert}) &= \frac{\text{Probability of vertical transmission}}{\text{Total number of fields}}, \\ &= \frac{p(I_C + I_N)}{N}. \end{aligned} \tag{2.18}$$

A non-controller's instantaneous probability of horizontal infection ($p(\text{Horiz})$) must account for the fact that they may first be infected via vertical transmission, leading to:

$$\begin{aligned} p_N(\text{Horiz}) &= \begin{array}{c}\text{Instantaneous probability} \\ \text{of horizontal transmission}\end{array} \times \left(1 - \begin{array}{c}\text{Instantaneous probability} \\ \text{of vertical transmission}\end{array}\right), \\ &= \left(\frac{\beta(I_C + I_N)}{\beta(I_C + I_N) + \gamma}\right)\left(1 - \frac{p(I_C + I_N)}{N}\right). \end{aligned} \tag{2.19}$$

Combining these gives us the instantaneous probability of infection for a non-

controller (q_N):

q_N = Grower's estimate of the probability of infection next season if control is not adopted,

$$= p_N(\text{Vert}) + p_N(\text{Horiz}),$$
$$= \frac{p(I_C + I_N)}{N} + \left(\frac{\beta(I_C + I_N)}{\beta(I_C + I_N) + \gamma}\right)\left(1 - \frac{p(I_C + I_N)}{N}\right). \quad (2.20)$$

How these probabilities of infection change with increasing disease pressure ($I_N + I_C$) is shown in Appendix A.1 Figure A.1.1(A).

2.2.5 Expected profits

Our behavioural models assume that – at the time of planting – individual growers estimate the expected profit for the next season of adopting each strategy. These depend on the payoffs for each strategy × outcome combination (Equations 2.11-2.14) and the estimated probabilities of infection under each strategy (Equations 2.17 and 2.20). In particular, the estimated expected profit for a controller, P_C, would be

P_C = Grower's estimate of the expected profit next season if control is adopted,

$$= q_C P_{IC} + (1 - q_C) P_{SC},$$
$$= Y - \phi - L\left(\frac{\beta(I_C + I_N)}{\beta(I_C + I_N) + \gamma}\right). \quad (2.21)$$

The corresponding estimate without control, P_N, is

$$\begin{aligned} P_N &= \text{Grower's estimate of the expected profit next season if control is not adopted,} \\ &= q_N P_{IN} + (1 - q_N) P_{SN}, \\ &= Y - L\left(\frac{p(I_C + I_N)}{N} + \left(\frac{\beta(I_C + I_N)}{\beta(I_C + I_N) + \gamma}\right)\left(1 - \frac{p(I_C + I_N)}{N}\right)\right). \end{aligned} \quad (2.22)$$

The expected profit averaged over all growers, P, is

$$\begin{aligned} P &= \text{Grower's estimate of the expected profit next season averaged over the population,} \\ &= P_C\left(\frac{S_C + I_C}{N}\right) + P_N\left(\frac{S_N + I_N}{N}\right). \end{aligned} \quad (2.23)$$

Calculating P therefore requires growers to know the instantaneous proportion of the population controlling. This calculation of P is instantaneous, i.e. based on the state of the system at the time of planting, and provides an estimate for the next season.

Importantly, even though these are the instantaneous profits a grower could expect to earn based on the current risks of infection, they are effectively the expected profit per season of using a particular control strategy.

2.2.6 Behavioural models

We systematically examine different ways to construct the switching terms in Equations 2.5-2.8. Differences between behavioural models turn on how Δ, an expected difference in profit (and thus profitability), is estimated. We consider

four possible comparisons:

- *Strategy vs population.* Growers compare the expected profit for their current strategy with the expected profit across the entire population of growers ($P - P_j$, with $j \in \{C, N\}$).

- *Strategy vs alternative strategy.* Growers compare the expected profit for their current strategy with the expected profit of the strategy which they did not use ("alternative strategy"; $P_i - P_j$, with $i, j \in \{C, N\}$ and $i \neq j$).

- *Grower vs population.* Growers compare their own outcome during the previous season with the expected profit across the entire population of growers ($P - P_G$, where P_G is the grower's profit from the previous season).

- *Grower vs alternative strategy.* Growers compare their own outcome during the previous season with the expected profit of the alternative strategy ($P_i - P_G$, where $i, j \in \{C, N\}$).

All set z, the probability of an individual grower switching strategy, as a function of Δ, via

$$z = \max\left(0, 1 - e^{-\eta\Delta}\right) = \begin{cases} 1 - e^{-\eta\Delta} & \text{if } \Delta \geq 0 \\ 0 & \text{otherwise.} \end{cases} \tag{2.24}$$

Since growers aim to maximise their profit, they only consider switching strategy when the comparison appears to offer a higher payoff (i.e. when $\Delta > 0$;

Table 2.2: **Summary of the information required by growers to carry out each means of comparison.** "Controller prevalence" refers to the proportion of growers who adopted control the last time their field was planted (i.e. $\frac{S_C+I_C}{N}$) and "disease prevalence" is the proportion of growers whose fields are currently infected (i.e. $\frac{I_N+I_C}{N}$). The "✓" refers to when information is required, and "X" when it is not necessary for decision-making.

	Disease prevalence	Controller prevalence	Own outcome
Strategy vs population	✓	✓	X
Strategy vs alternative	✓	X	X
Grower vs population	✓	✓	✓
Grower vs alternative	✓	X	✓

Figure 2.2(C)). The parameter η sets the responsiveness of growers to a unit difference in profit.

A summary of the information required for each means of comparison is provided in Table 2.2.6.

The "strategy vs" models are inspired by evolutionary game theory (Bauch (2005), Delabouglise & Boni (2020)), with Δ depending on the expected profit for the strategy currently adopted. The "grower vs" models are closer to previous usage in plant disease epidemiology (e.g. McQuaid et al. (2017a), Milne et al. (2016) and Saikai et al. (2021)), with the outcome obtained by the grower for their last crop used as the point of comparison. Table 2.2.6 provides a summary of the symbols used in the behavioural model.

Strategy vs population

In the "strategy vs" models, the switching probabilities are decoupled from the outcome during the previous season, i.e. $z_{SC} = z_{IC} = z_C$ and $z_{SN} = z_{IN} = z_N$.

Table 2.3: **Summary of symbols used in defining the behavioural models.** The outcomes $i \in \{S, I\}$ represent – at the end of the season – fields that are (S)usceptible and (I)nfected, respectively. The strategies $j \in \{C, N\}$ correspond to (C)ontrollers (i.e. use clean seed) and (N)on-controllers (i.e. do not use clean seed), respectively.

Symbol	Meaning	Defined in
z_{ij}	Switching probability for grower in infection class $i \in \{S, I\}$ who used strategy $j \in \{C, N\}$	Equations 2.1-2.4
P_{ij}	Profit for grower with outcome $i \in \{S, I\}$ who used strategy $j \in \{C, N\}$	Equations 2.11-2.14
q_j	Estimated probability at time of planting of infection if go onto use strategy $j \in \{C, N\}$	Equations 2.17 & 2.20
$p_N(\text{Vert})$	Estimated probability of vertical infection for non-controllers	Equation 2.18
$p_N(\text{Horiz})$	Estimated probability of horizontal infection for non-controllers	Equation 2.19
P_j	Estimated profit at time of planting if go onto use strategy $j \in \{C, N\}$	Equations 2.21 and 2.22
P	Estimated profit at time of planting averaged over entire population	Equation 2.23
Δ	Estimated difference in expected profit	Equation 2.24
q_β	Perceived value of the rate of horizontal transmission (β)	Equation 2.37
q_I	Perceived number of infected fields	Equation 2.38

The probability of a controller switching to no longer use clean seed is

$$z_{SC} = z_{IC} = z_C = \max\left(0, 1 - e^{-\eta(P - P_C)}\right). \tag{2.25}$$

This probability is only non-zero when the expected profit for a controller is assessed to be smaller than the expected profit over the entire population (i.e. when $P_C < P$). Conversely, the probability of a non-controller switching is

$$z_{SN} = z_{IN} = z_N = \max\left(0, 1 - e^{-\eta(P - P_N)}\right), \tag{2.26}$$

which takes a non-zero value only when $P_N < P$. Since P is a convex combination of P_C and P_N (the two bracketed terms in Equation 2.23 are complementary probabilities), only one of P_C or P_N can be greater than P at any time, meaning z_C and z_N cannot simultaneously be non-zero (although if $P_C = P_N$, i.e. the expected profits are equal, then $P = P_C = P_N$ and so $z_C = z_N = 0$).

How the expected profits and switching terms change with increasing disease pressure ($I_N + I_C$) is shown in Appendix A.1 Figure A.1.1(B)-(C) and Appendix A.1 Figure A.1.1(E)-(F).

Strategy vs alternative strategy

This model is similar to the "strategy vs population" model, but now instead of comparing to the expected profit of the population, P, growers compare to the expected profit of the alternative strategy (i.e. P_C or P_N, as appropriate). The probability of a controller switching is

$$z_{SC} = z_{IC} = z_C = \max\left(0, 1 - e^{-\eta(P_N - P_C)}\right), \tag{2.27}$$

which takes a non-zero value only when $P_N > P_C$, i.e. when non-controllers are expected to be more profitable next season. Conversely, the probability of a non-controller switching is

$$z_{SN} = z_{IN} = z_N = \max\left(0, 1 - e^{-\eta(P_C - P_N)}\right), \tag{2.28}$$

which is non-zero only when $P_C > P_N$. Here it is clear that only one of z_C and z_N can be non-zero at any time, since either $P_C > P_N$ or $P_C < P_N$ (or $P_C = P_N$, in which case no grower would switch since the expected profits of the two strategies are equal, and $z_C = z_N = 0$).

Grower vs population

In the "grower vs" models, decisions are made on the basis of grower's own outcome for the last crop, and so all four switching probabilities can differ. The ordering of profits in Equation 2.15 means that an uninfected non-controller (i.e. class S_N) should never switch strategy, since these "successful free-riders" (Fu et al. (2011*b*)) obtained the highest possible profit for the last crop. Similarly, having obtained the "suckers' pay off", a grower who controlled for disease but was nevertheless infected (i.e. class I_C) will always have a non-zero probability of switching, since they must have obtained a lower than average profit. Whether or not S_C and I_N growers switch strategy depends on their profit relative to the expectation over the whole population, P. The switching terms are therefore

$$z_{SN} = 0, \tag{2.29}$$

$$z_{IN} = \max\left(0, 1 - e^{-\eta(P - P_{IN})}\right), \tag{2.30}$$

$$z_{SC} = \max\left(0, 1 - e^{-\eta(P - P_{SC})}\right), \tag{2.31}$$

$$z_{IC} = 1 - e^{-\eta(P - P_{IC})}. \tag{2.32}$$

Grower vs alternative strategy

This model is similar to the "grower vs population" model, but now growers compare to the expected profit of the alternative strategy (i.e. P_C or P_N) rather than that of the population, P, with

$$z_{SN} = 0, \tag{2.33}$$

$$z_{IN} = \max\left(0, 1 - e^{-\eta(P_C - P_{IN})}\right), \tag{2.34}$$

$$z_{SC} = \max\left(0, 1 - e^{-\eta(P_N - P_{SC})}\right), \tag{2.35}$$

$$z_{IC} = 1 - e^{-\eta(P_N - P_{IC})}. \tag{2.36}$$

2.2.7 Systematic uncertainty

The models as presented thus far presume that all growers can perfectly perceive the risk of infection by knowing the true values of β and the total number of infected individuals (I). However, these quantities must actually be estimated by growers. To account for this, we introduce two new parameters: the perceived rate of horizontal transmission (q_β) and the perceived number of infected fields (q_I). These are given by:

$$q_\beta = \nu_\beta \beta, \tag{2.37}$$

$$q_I = \nu_I (I_N + I_C). \tag{2.38}$$

Therefore ν_β and ν_I scale the values of β and $(I_C + I_N)$ respectively, amounting

to either an over- or underestimate of their values. This modifies their contributions to the estimated profits in Equation 2.21 and Equation 2.22, which become:

$$\tilde{P}_C = Y - \phi - L\left(\frac{q_\beta q_I}{q_\beta q_I + \gamma}\right). \tag{2.39}$$

and

$$\tilde{P}_N = Y - L\left(\frac{pq_I}{N} + \left(\frac{q_\beta q_I}{q_\beta q_I + \gamma}\right)\left(1 - \frac{pq_I}{N}\right)\right). \tag{2.40}$$

Importantly, these changes to how growers perceive the risk of infection do not change the epidemiology of the system, only the behaviour of the growers. If the value of $\nu_I(I_N + I_C) > N$, we assume that growers estimate that all fields in the system are infected and thus set $\nu_I(I_N + I_C) = N$.

2.3 Results

2.3.1 Effect of the formulation of the profit comparison

Characterising model equilibria

The field-scale basic reproduction number in the absence of control (Appendix A.2 Text) is:

$$R_0 = \text{Basic reproduction number} = R_0^H + R_0^V = \frac{\beta N}{\gamma} + p, \tag{2.41}$$

with distinct components corresponding to horizontal (R_0^H) and vertical (R_0^V) transmission (Hamelin et al. (2021)) (Appendix A.3 Text). This applies for both the "strategy vs" and "grower vs" models; below this threshold, there is a disease-free equilibrium (DFE) where there are no infected fields and consequently no controllers (as there is no need to control from disease where there is no risk of infection) (Figure 2.3(A)-(C)). Other, disease-endemic equilibria are possible and depend on the type of comparison being used.

There are up to four different equilibria, defined as:

- **Disease-free equilibrium** at which the disease is not able to spread even when there is no control via clean seed. This is stable for $R_0 < 1$.
- **Control-free, disease-endemic equilibrium** at which the disease is endemic, but it is unprofitable to use the CSS and so no growers control.
- **All-control, disease-endemic equilibrium** at which all growers con-

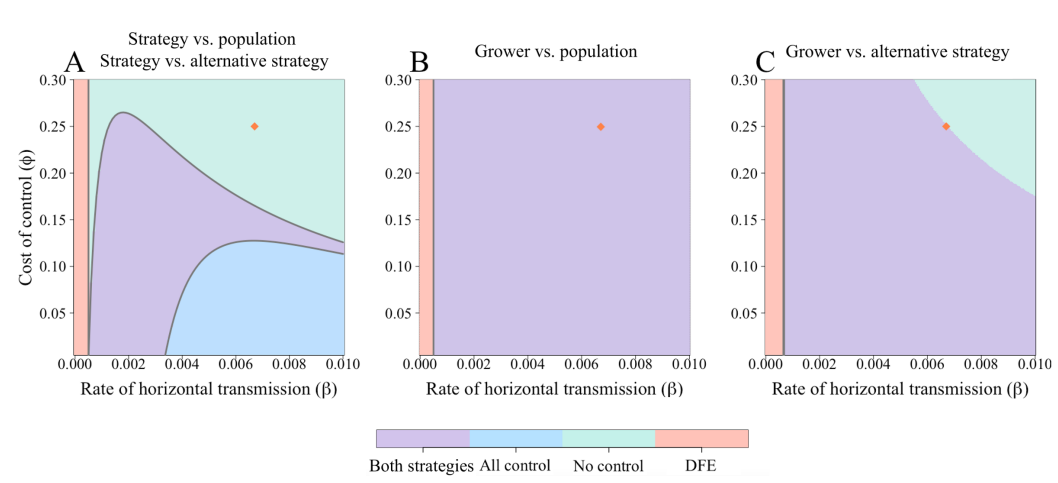

Figure 2.3: **Effect of the rate of horizontal transmission (β) and the cost of control (ϕ) on final equilibrium attained.**(A)The "strategy vs population" and "strategy vs alternative" models have four possible, mutually-exclusive equilibria. For any given parameter set, both of these models will have the same equilibrium values. Which is attained depends both on R_0 and the balance of benefit between controlling and not controlling. Below $R_0 = 1$ (which corresponds to the vertical line at $\beta = 0.0005$ day $^{-1}$), only the disease-free equilibrium (DFE) is possible. Boundaries between equilibria are determined by the stability conditions (Appendix A.3 Text). Also shown in (B) and (C) are the equilibria for the "grower vs" models, though numerical expressions determining these cannot be derived analytically. Parameters are as in Table 2.1; the orange diamonds mark the default values β and ϕ (which, in C, is in the "no control" region). The dynamics for these default values are shown in Figure 2.4. (B) does not show all equilibria achievable for the "grower vs population" model (see Appendix A.3 Figure A.2.1).

trol, but nevertheless disease is still endemic in the system.

- **Two-strategy, disease-endemic equilibrium** at which both disease and control equilibrate at some intermediate level.

Which subset of these outcomes is possible will depend on which model formulation is used.

Parameterisations leading to each of the four equilibria are possible for the 'strategy vs" models, depending on the balance between the rate of horizontal transmission (β) and the cost of control (ϕ) (Figure 2.3(A)). In these models,

coupling of the switching terms allows mathematical analysis (Appendix A.3 Text), and stability conditions can be derived for the single-strategy equilibria. As noted above, although the dynamics in reaching equilibrium can differ, the final state of the system does not depend on whether the "strategy vs population" or "strategy vs alternative strategy" model is considered. In each case, for an equilibrium with both strategies present, the expected payoffs (P_C and P_N) must be equal, otherwise one group of growers will continue to be incentivised to change strategy. For only controllers to be present, $P_C > P_N$ and vice versa for the non-controller equilibria. The differences in dynamics approaching equilibrium arise from the different calculations of Δ (Equations 2.25 - 2.28).

Importantly, the "all control" equilibrium, can be attained in these "strategy vs" models. This equilibrium is due to the imperfect protection provided by the control scheme; if horizontal infection is sufficiently significant, even if all growers control it does not guarantee the system remains disease-free. We note here that the equilibrium attained is independent of responsiveness of growers, η, though it does affect the dynamics approaching equilibrium (Appendix A.1 Figure A.1.1(D)).

The more complex form of the "grower vs" models makes mathematical analysis difficult, as conditions for model equilibria no longer simply depend on the difference in profits between controllers and non-controllers. However, extensive numerical work (Appendix A.3 Text) indicates the equilibrium that is reached again depends only on model parameters (i.e. there is no bistability

between equilibria). In the "grower vs" models, only three of the four equilibria can be attained: the "all control" equilibrium is impossible because growers who controlled but nevertheless became infected always consider switching strategy, as they are earning the lowest possible payoff (P_{IC}, the "sucker's payoff"). As long as there is a non-zero probability of infection (which is necessary for control to be worthwhile) there will always be non-controlling growers. Additionally, for the "grower vs population" model, the "no control" equilibrium is only possible if all non-controllers are infected at equilibrium (Appendix A.3 Text). This requires a very narrow range of parameters, shown in Appendix A.3 Figure A.2.1. Unlike in the "strategy vs" models, where the equilibrium is not dependent on the value of responsiveness of growers, for the "growers vs" models η does affect the final equilibrium values (Appendix A.3 Figure A.3.1).

Default behaviour of models

Using the default parameters, we obtain a "no control" equilibrium when growers employ the "strategy vs population" and "strategy vs alternative" models (Figure 2.4(A)-(B) respectively). At these parameter values, the rate of horizontal transmission and probability of vertical infection are so high that growers are likely to become infected, thus paying the cost of control (ϕ) and the loss due to disease (L). Therefore, control is not worthwhile.

For the "grower vs population" model, around 38% of growers control at equilibrium. For these parameters, any non-controlling grower with an infected

field (I_N) has a non-zero probability of changing strategy as the presence of uninfected non-controllers (S_N) means that they will earn below the expected population profit, P (see Equation 2.30). Thus, there will be controllers at equilibrium. Only when the parameter values mean that there will be no susceptible growers will there be a "no control" equilibrium (Equation A.56).

This is not observed in the "grower vs alternative" model, where no growers control at equilibrium (Figure 2.4(D)). For this model, non-controlling growers with infected fields (I_N) need only earn more than the alternative strategy, so if there is a high number of controllers with infected fields, then I_N growers should not switch strategy (Equation A.55).

2.3.2 Effect and implications of epidemiological and economic parameters on uptake of control

We now continue our investigation using only the "grower vs alternative" model set-up. The idea of the "reflexive producer" means that growers are likely to respond to previous season's outcomes (Kaup (2008)), but still incorporate information about what the likely infection risk is for the next season (Milne et al. (2018), Garcia-Figuera et al. (2021)). We therefore believe that this is a useful approximation of how growers may actually perceive profitability, though we include analysis of the other models in Appendix A.3 Figure A.3.2.

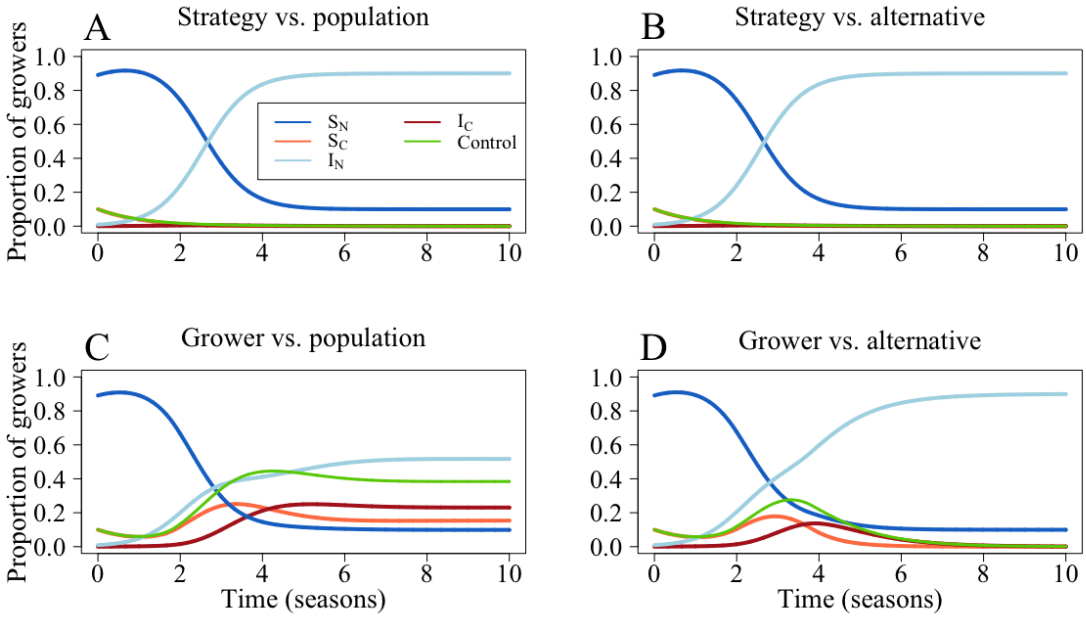

Figure 2.4: **Model dynamics for different means of comparison.** Here, S_N are susceptible non-controllers, I_N infected non-controllers, S_C are susceptible controllers and I_C infected controllers. C represents $S_C + I_C$. In (A), growers compare the expected profit of their strategy with that of the entire population; in (B) they compare the expected profit of their strategy with the expected profit of the alternative strategy. For (C), growers compare their own outcome with expected profit of the population and in (D) they compare their outcome with the expected value of the alternative strategy. In (A), (B) and (D), no growers control for disease at equilibrium, though in (C) around 38% of growers control. Parameters are outlined in Table 2.2.2.

Effect of epidemiological and economic parameters on uptake of control

We investigate the change in uptake of control and proportion of disease fields when the cost of control (ϕ), probability of vertical transmission (p) and loss due to disease (L) are varied (Figure 2.5). Responses to changes in parameter values were often intuitive. For example, at very high costs of control (ϕ), fewer growers controlled for disease (Figure 2.5(A)). Similarly, when the risk of vertical transmission increased, so too did the proportion of growers controlling (Figure 2.5(B)). However, even within these broad trends, there were also non-monotonic responses. We found that at medium-to-low values for cost of control (ϕ) and the rate of horizontal infection (β), high proportions of growers controlled for disease (Figure 2.5(A)). Yet as β increased, the higher probability of infection narrows the range of costs for which a controller will consider participation in the CSS, as the grower will likely have to pay both the cost of control (ϕ) and the loss due to disease (L) (the "sucker's payoff").

A similar principle underpins Figure 2.5(B)-(C). At higher probabilities of horizontal transmission (β), disease pressure increases and controllers are likely to be infected, even if non-controllers are more likely to be infected due to high probabilities of vertical transmission (p). In each case, when there is a higher proportion of controllers at equilibrium, there are fewer infected fields (Figure 2.5(D)-(F)).

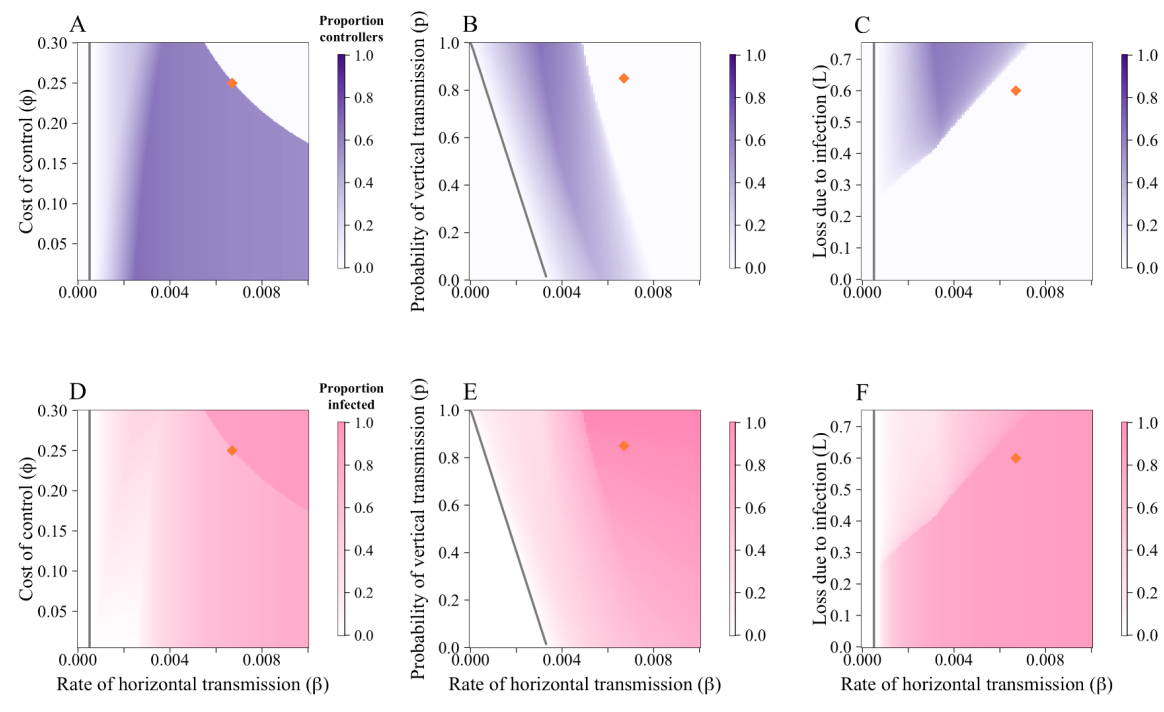

Figure 2.5: **CSS participation and proportion of infected fields are dependent on economic and epidemiological parameters.** Plots show the equilibrium proportion of controllers in response to variations in parameters when comparisons are made using the "grower vs alternative" comparison.(A)Effect of the rate of horizontal transmission (β) and cost of control (ϕ). (B) Effect of the rate of horizontal transmission (β) and probability of receiving infected planting material via trade (p). (C) Effect of the rate of horizontal transmission (β) and loss due to infection (L). At these default parameter values (particularly the high cost of control), both p and L only affect participation in the CSS for a narrow range of β. (D)-(F) show the response of the proportion of infected fields ($(I_N + I_C)/N$) to β, ϕ, p and L respectively. In each case, as control increased, disease decreased. Parameters values are in Table 2.2.2; the orange diamonds mark the default values.

Growers receive the highest profits when $R_0 < 1$ (i.e. there is no disease, so growers cannot incur any yield loss) (Figure 2.6). When disease is endemic to the system (i.e. $R_0 > 1$), both profit and yield decrease with higher costs, losses and probability of infection (corresponding to the higher proportion of infected fields in Figure 2.5(D)-(F)). The lowest yield and profit are obtained when all fields are infected, and at this point nobody controls for disease.

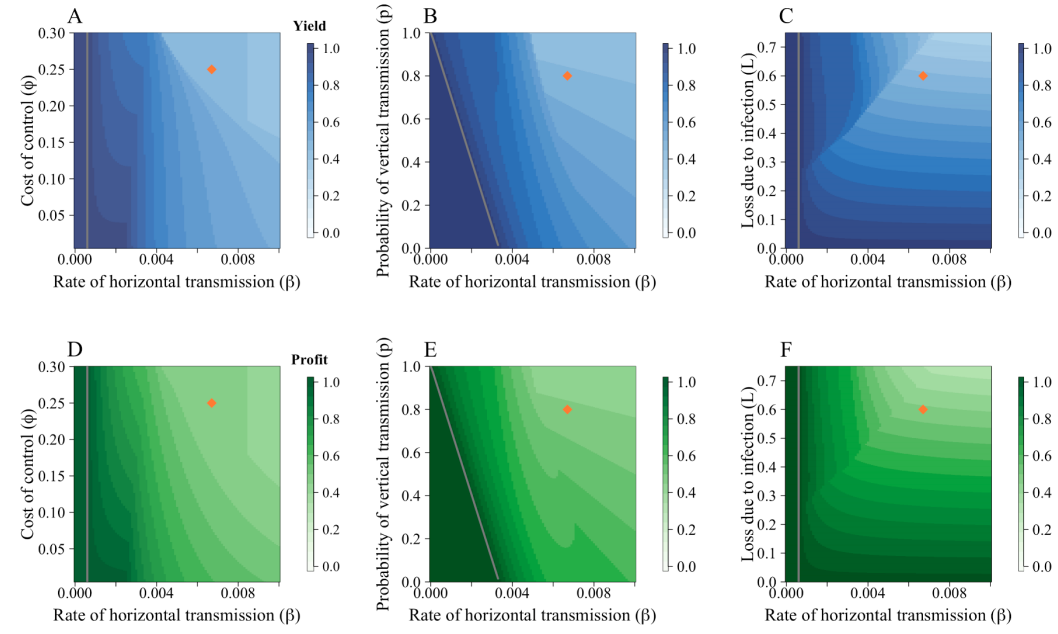

Figure 2.6: **Effect of parameters on yield and profit for the "grower vs alternative" models.**(A)– (C) show the average yield, whilst (D) – (F) show the average profit. For each parameter scan, at low rates of horizontal transmission (β), average yield and profit was higher as very few fields were infected (and thus did not incur the loss due to disease, L).(A)The effect of the cost of control, ϕ, on the yield. As the cost of control increases, fewer growers participate in the CSS and disease incidence increases, leading to lower yields. (B) The effect of the probability of vertical transmission, p. At higher values of p, yield falls as disease prevalence increases. (C) Effect of loss due to disease, L. Intuitively, at higher L, yield falls. Parameters are as in Table 2.2.2; the orange diamonds mark the default values. Patterns were similar across (D) – F, with lower profits observed as ϕ, p and L increased.

Implications for encouraging adoption of control

Growers are responsive to both economic and epidemiological factors. If the cost of the control mechanism is too high, growers will not use the scheme as they do not perceive it to be worthwhile. If the scheme provides too little a benefit over the non-control option (which in this case would mean that the probability of vertical transmission was relatively low), growers will also not use it. Though lower losses are beneficial for profit and yield, they discourage disease control. If losses are sufficiently low (such that $L < \phi$), control would not be a rational choice. It is therefore important to emphasise the importance of subsidies for control schemes (which will have the effect of reducing ϕ) to policy-makers in order to encourage participation. The effect of such a subsidy is shown in Appendix A.3 Figure A.3.4, where the lower cost of control widens the values of the rate of horizontal transmission (β) and the probability of vertical transmission (p) for which control is profitable.

2.3.3 Effect of systematic uncertainty

Even when growers underestimate the rate of horizontal transmission, β, to the extent that they do not believe there to be any risk of horizontal transmission (i.e the relative perception of β is zero ($\nu_\beta = 0$, so the perceived value of β (q_β)$= 0$), some still use the CSS as the probability of vertical transmission is non-zero (Figure 2.7(A)). As ν_β increases, fewer growers use the control

scheme as they perceive they will achieve the "sucker's payoff". The default parameterisation (when $q_\beta = \beta$, i.e. $\nu_\beta = 1$) means that growers should never control. The higher cost of control (ϕ) caused the system to reach a "no control" equilibrium (Figure 2.7(A)).

Unlike when misestimating β, misestimations of I affect perception of both horizontal and vertical transmission. This means there is a less straightforward response to misestimations of I. The kinks in Figure 2.7(B) are caused by changes in the values of the switching terms (Equations 2.33-2.36), which set the probability that a grower managing a field of a particular type will switch strategy.

When the relative perception of I, ν_I, is small and increasing, there are more controllers at equilibrium (Figure 2.7(B)). At low values of ν_I, the non-infected controllers (S_C) growers perceive that the profit for non-controllers is greater than what they have earned ($\tilde{P}_N > P_{SC}$), so they should still consider switching strategy (i.e. $z_{SC} > 0$) (Figure 2.7(C)). As ν_I increases, their perception of $\tilde{P}_N$ falls and fewer S_C growers switch. This causes an increase in infected controllers (I_C), and fewer S_C switch strategy before they become infected. Eventually, $\tilde{P}_N < P_{SC}$ and S_C growers stop switching strategy entirely (around season 4 in Figure 2.7(D)). Many of these controllers will then become infected and switch strategy, leading to lower participation in control with higher ν_I. As ν_I increases, I_N growers are less likely to switch strategy as $\tilde{P}_C$ falls. Eventually, $z_{IN} = 0$ and no I_N growers should switch strategy (Figure 2.7(F)). As all I_C growers should have a non-zero probability of switching

strategy, but no non-controllers should switch, this leads to a "no control" equilibrium.

2.4 Discussion

Surprisingly, few plant disease studies examine how human behaviour alters the dynamics of epidemic models. Here we presented a model that uses game theory to incorporate grower decision-making into a model of CBSD and investigated the effect of economically- and epidemiologically- important parameters on disease spread and the uptake of control by way of a clean seed system. We found that the formulation of the model (whether based on rational or strategic-adaptive expectations) had significant consequences on the possible equilibria that could be attained. The model parameters also influenced outcomes, and interactions between them (particularly the cost of control and the rate of horizontal transmission) had large impacts on growers' participation in the CSS. Finally, the response to misperception of epidemiological quantities was often not intuitive, and could have implications for the communication of risk.

We found that basing decisions on rational (the "strategy vs" models) or strategic-adaptive ("grower vs" models) changed the potential outcomes for the model. Only the "strategy vs" comparisons permitted an "all control" equilibrium, whereas the low payoff for controllers with infected fields (the "sucker's payoff") meant that they will always have a non-zero probability of

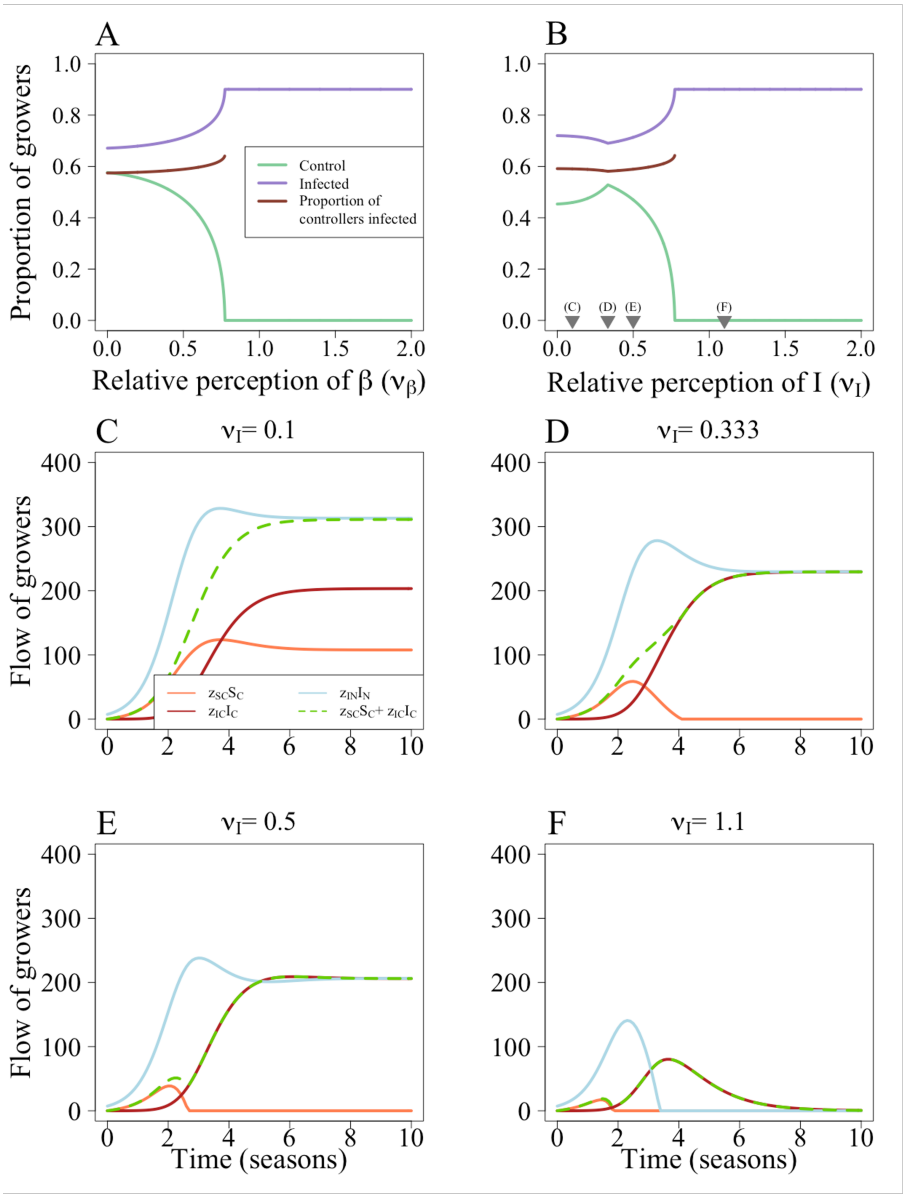

Figure 2.7: **Effect of systematic uncertainty of epidemiological parameters and quantities.** When the perception of β (ν_β) is small there is a higher proportion of controllers (A), which decreases as ν_β increases. In (B), there is initially an increase in the proportion controlling with the perception of I (ν_I). In (B) the number of infected fields levels out once the conditions for a "no control" equilibrium are met. The grey triangles in (B) show the values of ν_I used in (C)-(F). (C) When $\nu_I = 0.1$, S_C growers still perceive that it is profitable to change strategy ($z_{SC} > 0$). (D) At $\nu_I = 0.33$, as the number of infected fields increases, the perceived value of P_N ($\tilde{P}_N$) decreases and $z_{SC} = 0$. However, they continue to get infected and there is an increase in I_C fields. (E) At $\nu_I = 0.5$, growers with S_C fields stop switching strategy earlier in the epidemic. As $\tilde{P}_C$ falls, so does z_{IN}. (F) At $\nu_I = 1.1$, $\tilde{P}_C < \tilde{P}_{IN}$, so $z_{I_N} = 0$. This leads to a "no control" equilibrium. Parameters are as in Table 2.2.2.

switching strategy in the "grower vs" comparisons. This difference in possible equilibria from the models shows that it is important not only to include behaviour in epidemic models, but to consider how it is included based on what quantities are being compared and what information will be available to individual growers, such as the disease pressure, profit of other growers and breakdown of the expected profit for each strategy.

Game theory predicts that high participation in voluntary disease control schemes is difficult to attain, as the success of disease control policies in reducing the probability of infection disincentivises continued use of the scheme (Geoffard & Philipson (1997), Bauch et al. (2003), Bauch (2005); reviewed in Chang et al. (2020)). Indeed, in the case of voluntary vaccinations, at least with perfect immunity, models predict that actual level of vaccination will be less than that needed for herd immunity (Bauch & Earn (2004)). This is because of the *positive externalities* generated by having high levels of vaccination; if the majority of individuals are perfectly protected from infection, then there will be a lower probability of infection for others who are not vaccinated. They can then get much of the benefit of vaccination without themselves incurring any risk or cost. Indeed, the risk of other individuals "free-riding" off an individual's control mechanism may also be a disincentive to control (Milne et al. (2018)).

Yet if vaccines are not completely effective, the reduction in magnitude of positive externalities can lead to increased vaccine uptake until a stable "all vaccinated" equilibrium is attained (Wu et al. (2011)). That is, as the vaccine

no longer confers perfect immunity to the vaccinated, they may then transmit the disease to the unvaccinated. This decreases the benefit of high levels of vaccination in the population to the unvaccinated, and thus encourages them to be engaged in disease prevention themselves. This is more akin to the control system we have considered here, where the CSS only reduces the initial probability of infection and does not provide continued control during the season.

In our model, it was the imperfect protection offered by the CSS that allowed the "all control" equilibrium to be attained in the "strategy vs" models (Figure 2.3(A)). Although the CSS eliminates a grower's chance of receiving infected material via vertical transmission, it offers no protection from infection via the whitefly vector. By comparing the expected payoff of everyone using their strategy, rather than basing decisions on their own outcomes, even growers who themselves received the lowest possible payoff do not necessarily have a positive probability of switching strategy.

In the "strategy vs" models, we found that use of CSS could reduce CBSD spread, but never lead to disease elimination. By considering the stability of the equilibria, we can see that for an "all control" equilibrium, disease must be capable of moving via the whitefly vector alone. Fields that were initially planted with clean seed can therefore become infected, allowing disease to remain endemic in the system. Thus, though increased use of the CSS leads to lower disease prevalence, some degree of disease spread is required for growers to consider the CSS worthwhile. The threshold that determines the benefit of

the CSS is determined by the stability conditions outlined in Appendix A.3, which are broadly determined by the probability of incurring the loss due to disease for both controllers and non-controllers. This result, and associated requirement for horizontal transmission, holds true for the "grower vs" models, too, though analytical expressions cannot be derived to describe the stability of the equilibria.

We assumed that all growers had the same responsiveness (our parameter η). This impacted the final equilibrium value attained in our "grower vs" models (Appendix A.3 Figure A.3.1), but not our "strategy vs" models (Appendix A.1 Figure A.1.1). We have also assumed that growers would always choose the most profitable strategy; if the alternative strategy has a higher expected profit, growers have a non-zero probability of switching (Figure 2.2(C)). As η increases, growers are almost guaranteed to switch strategy. For the "strategy vs models", which are based on rational expectations, this increase in η approaches the assumption of perfect rationality in game theory (Morris (2012)). Both rationality and responsiveness are likely to vary from grower to grower. Their distribution can be described by a "risk attitude" framework, in which individuals fall on a spectrum between risk tolerant and risk averse (Zhang et al. (2014)). Including such heterogeneity in risk perception in future studies would allow for greater understanding of behaviour in uncertain environments.

When conducting our further analysis into the effects of parameters and misperception, we focused on the "grower vs alternative" evaluation, where individual growers compare their own profits with the expected profit of the

alternative strategy. The reasoning behind this was twofold: first, this has been used previously in plant epidemiological literature (Milne et al. (2016), McQuaid et al. (2017a), Saikai et al. (2021)). Secondly, and most importantly, we believe this comparison to be most similar to what real growers might be expected to do (Kaup (2008), Milne et al. (2018), Garcia-Figuera et al. (2021)). It requires a moderate amount of information, as it only relies on the individual knowing their own profit from the previous season and the expected profit of the alternative strategy (Table 2.2.6).

Epidemiological and economic parameters had a substantial impact on the uptake of control (Figure 2.5). When the cost of control (ϕ) was low, more growers used the CSS (Figure 2.5(A) and Appendix A.3 Figure A.3.4). However, even at these low costs of control, if the rate of horizontal transmission was too high, growers would still not use the control scheme as the high probability of infection meant they were likely to incur the dual penalty of yield loss and cost of control. Subsidies, then are likely to be an effective way of encouraging control, particularly as both profits and yields are highest when the cost of control is low (Figure 2.6(A) and 2.6(C)) In practice, the success of subsidies may be limited by a lack of information amongst growers, unwillingness of growers to vary from current practices, or perceived ineffectiveness of control schemes. It is important that the introduction of subsidies control is accompanied by outreach and educational campaigns for growers (Jowett et al. (2022)).

For control to be worthwhile, it must provide sufficient benefit compared to

the alternative non-control strategy. In our case study, the benefit of control is the elimination of vertical transmission for those who use the CSS. Thus, if the probability of vertical transmission is sufficiently low for non-controllers, there may not be enough incentive to control (Figure 2.5(B)). Similarly, if growers do not stand to lose much yield when infected, the cost of control may be a disincentive, particularly if the probability of infection is low (Figure 2.5(C)). Indeed, if the expected yield loss over a season is less than the cost of control, it would never be worthwhile to control. It is therefore important to emphasise the benefits of control to growers, particularly if there is likely to be a significant future increase in disease prevalence that they are not accounting for when they are making their decisions.

Growers' inability to properly perceive epidemiological parameters had varying effects on CSS participation. Underestimating the rate of horizontal transmission, β, increased participation as growers believed that infection via this route was unlikely (Figure 2.7(A)). However, there was still a high probability of vertical transmission so control was worthwhile. As estimates of β increased, control was less favourable and participation decreased. Misestimations of the proportions of infected individuals had a non-monotonic pattern, with the proportion of controllers initially increasing with the perceived number of infected fields ($q_I I$) before falling as more controllers become infected and thus receive the lowest payoff (Figure 2.7(B)).

It is therefore important to understand how growers perceive their probability of infection, as this will influence their participation in control schemes

and subsequent spread of disease. Overestimating (or exaggerating when communicating with growers) the threat can potentially discourage participation due to the perceived inevitability of infection regardless of any actions taken to prevent it. The effect of underestimation depends on the route of disease transmission in question, but control only remains attractive if it is seen as providing sufficient benefits. Again, here every grower has the same level of misperception, but in reality this will be closely linked to an individual grower's attitude towards risk, access to information and previous history of infection, which all will be highly variable between growers.

As well as economic considerations, there are many social factors underpinning the adoption of a new control mechanism. Descriptive norms (what the majority of people are doing, and thus what is deemed socially acceptable; Lazić et al. (2021)) have been found to influence growers' decision to partake in control (Rodriguez et al. (2009)). Relatedly, a grower's social network and ability to access trusted information has a strong impact on management practices. Some growers will treat the advice of "experts" with a degree of scepticism (often due to a perceived conflict of interest or disconnectedness from the "true" needs of growers) (Sherman & Gent (2014)). Other growers have developed a strong dependency on the advice of these experts (Sherman & Gent (2014)). Such contrasting beliefs and degree of trust in knowledge sources, even in adjacent landscapes, highlights the heterogeneous information network used by growers. In all cases, growers placed high value on personal and trans-generational knowledge (Sherman & Gent (2014)). Growers may

also be reluctant to adopt a new technology for fear of failure, or as they contradict a pre-existing ideology (Sherman & Gent (2014)).

Kaup introduces the idea of a "reflexive producer" as a grower that must make decisions by balancing knowledge generated by local growers and knowledge provided by external experts (Kaup (2008)). This is similar to the hypothetical scenarios we previously outlined, particularly that of the "grower vs alternative" scenario where a grower is using a particular control strategy and is then approached by an extension worker that proposes an different strategy. Thus, motivations and considerations of a grower to partake in specific control schemes are highly varied and depend on the market demands, information network available to growers, disease pressure and proximity to previous outbreaks (Garcia-Figuera et al. (2021)).

Though we have not investigated it here, CSS can "bundle" desirable traits by providing CBSD resistant or tolerant planting material, changing the nature of the externalities generated by clean seed use. There are few such varieties available for cassava. Provision of resistant material, though of obvious benefit to the recipient, will also increase the benefit experienced by free riders by reducing their probability of infection. Cassava varieties that are tolerant to CBSD infection will benefit the focal grower, but may increase the disease pressure experienced by other growers and thus incentivise control. These are potentially important distinctions, which we investigate in Chapters 3 and 4.

The inclusion of grower behaviour alters disease dynamics, as the option to participate in a control scheme lowered the probability of infection and has the

potential to increase the yield achieved by the growers. We have found that the means of inclusion of behaviour is also important; the quantities of comparison impact dynamics approaching equilibrium, but also determine the nature of the equilibria that can be achieved. Our investigation has been limited to one case study, but the decision modelling framework laid out in this paper is sufficiently flexible to be incorporated into a range of other disease systems. It would also allow grower behaviour to feed into other types of study, for example those in which control has a spatial element and/or is done reactively in response to detected infection (Cunniffe et al. (2015b), Cunniffe et al. (2016), Hyatt-Twynam et al. (2017), Laranjeira et al. (2020)), or where estimates of parameters become more accurate over time and as disease spreads (Thompson et al. (2018)).

Chapter 3: Tolerant and resistant crop varieties and their influence on grower behaviour

3.1 Introduction

3.1.1 Information from previous chapter

In Chapter 2, we compared different models of grower behaviour to investigate their impact on model outcomes. We found that basing estimates of profitability on rational expectations (our "strategy vs" models) or strategic-adaptive expectations (our "grower vs") models causes large variations in model predictions, which resulted in different long-term outcomes and responses to changes in economic and epidemiological parameters. Here, we use the "grower vs alternative" model from Chapter 2 to investigate the effect of different crop types on grower behaviour.

3.1.2 Tolerance and resistance to disease

Tolerance and resistance represent the two main mechanisms underpinning the genetic control of plant disease, and the differences between the two have clear implications for epidemic management. Resistance traits are associated with a reduction in pathogen burden (Pagán & García-Arenal (2018)), and disease-resistant varieties are consequently less susceptible and/or infectious than unimproved varieties. By contrast, tolerant varieties can be infected and maintain high pathogen burdens, meaning that infected plants can transmit infection at high rates, but their yield remains largely unaffected (Pagán & García-Arenal (2018)). Figure 3.1 shows a simplification of the difference in responses for host fitness and probability of infection for tolerant and resistant crop.

The two traits can be difficult to distinguish during breeding programmes, as both tolerance and resistance characteristics preserve yields when a plant is infected (Rahman et al. (2021)). Partial, or quantitative, resistance where the host does not completely restrict viral load but has a lower yield loss (Marchant et al. (2020), French et al. (2016)), is also more common than complete (qualitative) immunity (Corwin & Kliebenstein (2017)), further complicating the distinction between tolerance and resistance in breeding.

Epidemiological models have long been used to identify strategies that optimise the deployment of resistant crop (reviewed in Rimbaud et al. (2021)). Very often the focus is pathogen evolutionary dynamics and the breakdown

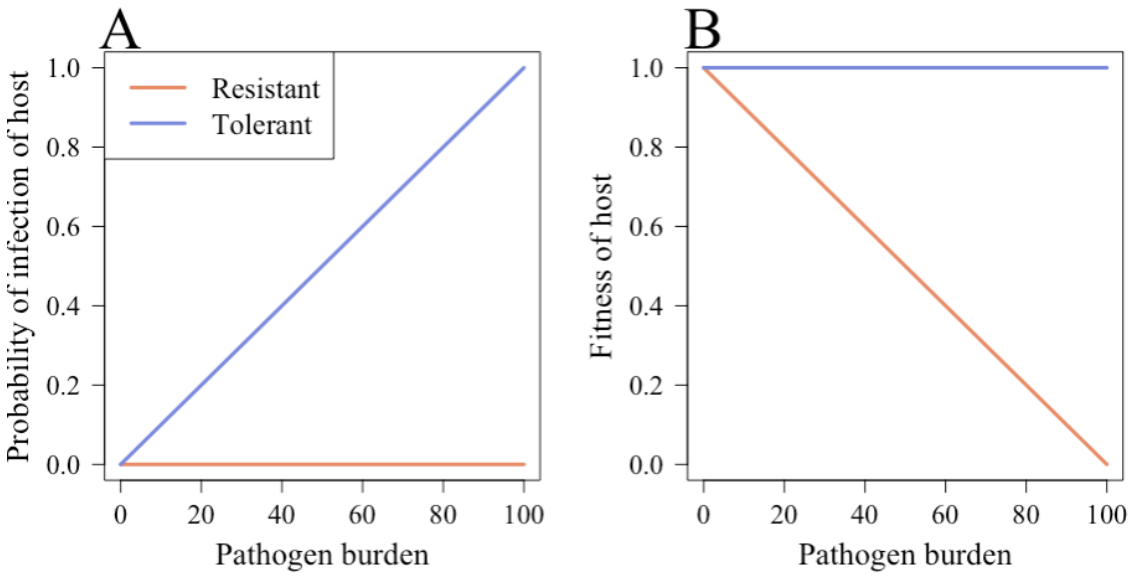

Figure 3.1: **Comparison of (A) a host's probability of infection given a specific pathogen burden in the environment and (B) the fitness of the host upon infection for tolerant and resistant genotypes.** In both cases, we assume complete resistance to and tolerance of disease. A resistant crop's probability of infection is the same (near zero or very low) irrespective of the pathogen burden in the environment, whereas a tolerant crop's probability of infection increases. Once infection occurs, the tolerant crop's fitness remains high, whereas that of resistant crop decreases with increased pathogen burden.

of resistance traits (van den Bosch & Gilligan (2003), Fabre et al. (2012), Watkinson-Powell et al. (2020), Rimbaud et al. (2018)). This builds on a long history of models aiming to explain gene-for-gene polymorphisms in host and pathogen populations, stretching back to theoretical work which is now nearly fifty years old (Leonard (1977)) although is still of current interest (Tellier & Brown (2007), Clin et al. (n.d.), Hamelin et al. (2022)). However, no studies have compared the epidemiological consequences of using tolerant versus resistant crop at the population scale, despite tolerant and resistant crop having significantly distinct effects on other growers. There has also been no consideration of factors incentivising growers to deploy one or other of these possible

disease controls.

Although no studies to date have compared the epidemiological consequences of using tolerant vs resistant crop, recent models have incorporated pathogen evolutionary dynamics and the breakdown of resistance traits. However, in our study, we only consider the epidemiological, not evolutionary, effects of the use of resistant or tolerant crop.

3.1.3 Externalities in disease models

In economics, externalities are the effect of an action by one party on other parties (Gersovitz (2014)). Previous work has shown that in disease management schemes, prevention measures such as vaccinations generate enough *positive externalities* that they disincentivise others from partaking in control (Geoffard & Philipson (1997), Ibuka et al. (2014)). By lowering the infection burden, this type of control increases the probability that those not using the control scheme will be "successful free-riders" (that is, gain the benefit of control without paying any of the costs, Bauch & Bhattacharyya (2012)). In plant epidemiology, resistance traits lower the prevalence of infection in the system as a whole and thus have the same impact as vaccinations by decreasing the probability of infection for non-resistant crop. Previous theoretical studies have shown that not every grower needs to plant resistant crop for the benefits to be felt across a community of growers (van den Bosch & Gilligan (2003), Lo Iacono et al. (2013), Vyska et al. (2016)). This same result applies to other mechanisms of control. Though high participation in citrus health manage-

ment areas (CHMAs; voluntary schemes established in the United States to combat citrus greening via methods such as synchronous spraying of pesticides) is correlated with better outcomes, complete participation is not needed to see improved yield outcomes (Singerman et al. (2017)).

Similarly, the majority of the benefits conferred by *Bt*-resistant maize in the United States were experienced by those who did not themselves use the improved crop and were consequently “free-riding" off the actions of others (Hutchison et al. (2010)). Reducing the degree of resistance could diminish these positive externalities, but ultimately some growers planting resistant varieties still acts as a disincentive to other individuals to practice disease management.

Disease tolerance, by contrast, should have the opposite effect to resistance. Tolerant crops do not restrict pathogen replication (Pagán & García-Arenal (2018), Kause & Ødegård (2012)) and so do not reduce the probability of infection for others. Tolerant crops, however, can sustain such pathogen burdens without the same degree of yield loss as unimproved crops (Pagán & García-Arenal (2018)). As tolerant crops have less noticeable symptoms, they cannot be as effectively removed and/or treated for disease. Thus, growers who use tolerant crops will experience the benefit whilst generating *negative* externalities for those around them by maintaining a high infection pressure (Hozé et al. (2018)). This effect may weaken other disease management efforts, increasing the negative consequences for others (see Earn et al. (2014) for an example in human disease epidemiology where it was found that tolerance-based thera-

pies for chronic infections increase population-level mortality as asymptomatic carriers circulate in the population). Tolerant crops may be asymptomatic and thus have a reduced probability of being visually detected and rogued (removed) in comparison to unimproved crops (Sisterson & Stenger (2018)), again allowing an increase in infection pressure. This incentivises others to use tolerant crops, too, to minimise their losses and could lead to overall higher participation in control schemes.

3.1.4 Case study: Tomato Yellow Leaf Curl Virus

To examine the differing effects of disease tolerance and resistance on the profits and, consequently, the behaviour of growers, we employ *Tomato Yellow Leaf Curl Virus* (TYLCV) as a case study. Tomato (*Lycopersicon esculentum*) is a globally important crop, with over 18.5 million tonnes produced in 2020 alone (Anon (2022)). TYLCV is one of the major viruses affecting tomato production worldwide (Ramos et al. (2019)), and it has been detected from East Asia to Western Europe, the United States of America and Australia. Transmitted by the sweet potato whitefly *Bemisia tabaci* (Pan et al. (2012)), infection leads to Tomato Yellow Leaf Curl Disease (TYLCD), which causes curling of the leaves, chlorosis of young leaves, flower abortion and stunting. Combined, these symptoms can cause up to 100% yield loss. Consequently, much research has been done on developing both tolerant and resistant varieties (Vidavsky & Czosnek (1998), Dhaliwal et al. (2020)), some of which have been deployed (Lapidot et al. (1997a), Riley & Srinivasan (2019)).

Despite the expansive literature, and perhaps reflecting the confusion between tolerance and resistance across crop breeding, there is considerable debate as to what degree of disease resistance has been achieved in tomato breeding. Some argue that though purportedly TYLCV-"resistant" cultivars have been developed, none have complete immunity to infection (Marchant et al. (2020) and Yan et al. (2018), who describe how what they term "resistant lines" are not actually immune to TYLCV, but instead have reduced symptoms). As there is incomplete restriction of viral replication, yield loss is less extreme than in completely susceptible genotypes. Some disease-resistant accessions have been identified in wild relatives of tomato (Yan et al. (2018)) and others have produced what appear to be genuinely disease-resistant varieties of tomato (Vidavsky & Czosnek (1998)), which will possibly lead to the deployment of fully-resistant cultivars in the future.

Additional complications, including the dependence of symptom development on non-genetic factors such as when in its life cycle the host was infected (Levy & Lapidot (2008)), or even which part of the plant was inoculated (Ber et al. (1990)) again make it harder to distinguish between resistance and tolerance. However, even the incomplete restriction of viral replication means that these cultivars can be considered partially resistant, rather than tolerant. To account for the difficulties in establishing whether a cultivar is tolerant or resistant, we consider a range of parameters pertaining to both qualities. These parameters represent a continuum between tolerance and resistance characteristics, allowing us to move smoothly from one parameterisation to another.

3.1.5 Aims of this chapter

We use Tomato Yellow Leaf Curl Virus as a case study to investigate the following questions: (1) How does the average profit of a group of growers change when a fixed proportion of those growers are using crop that is either tolerant or resistant to disease? (2) When growers can choose which type of crop they plant, how do the initial proportion of infectious and controlled fields affect the deployment of tolerant or resistant crop? (3) How does the use of improved crop change depending on whether it is disease-tolerant or resistant?

3.2 Methods

In our model of disease spread amongst a system of tomato fields, there are two available crop varieties: unimproved crop (U, "uncontrolled" or "unimproved") or an improved variety (C, "controlled"). The latter has some degree of tolerance and/or resistance, depending on the scaling of certain epidemiological and economic parameters. We can break down this tolerance/resistance continuum into six parameters (Table 3.2) which relate to how TYLCV is transmitted and the losses sustained when a field is infectious.

We classify fields based on their infectious states (susceptible, S; latently-infected and asymptomatic, E; or infectious, I) and by the variety with which they are planted (subscripts U and C). We do not model within-field disease spread, and assume that the two control strategies are mutually exclusive: a field cannot be planted with both unimproved and improved crop.

Susceptible fields are infected with TYLCV (transmitted by viruliferous

B. tabaci) at rate β or $\delta_\beta\beta$ for unimproved and improved crops, respectively. This choice was a pragmatic one, as it ensured that disease spread over a short period of time. However, to represent the uncertainty associated with this choice, we systematically scan over a range of values of β. Once infected (and provided they are not first harvested), fields remain latently infected (i.e. are asymptomatic and cannot transmit disease) for an average of $1/\epsilon$, days, after which they become infectious. Crop that is resistant to infection has an extended latent period, as symptoms take longer to develop. The latent period is increased by a factor of $\delta_{\epsilon_R} < 1$ (ensuring that $\delta_{\epsilon_R}/\epsilon < 1/\epsilon$). Fields are harvested on average every $1/\gamma$ days, irrespective of their control type or infection status. By restricting within-host viral replication, resistant crop also has a reduced probability of transmitting disease. The relative infectivity of a resistant field is given by δ_σ.

We also add roguing as a control mechanism enacted by all growers irrespective of the crop variety they use. Roguing infected plants is a common practice for management of TYLCV (Ioannu (1987), Ddamulira et al. (2021), Polston & Lapidot (2007), Polston et al. (1999)). In line with our assumption regarding symptom emergence and infectivity, only infectious crops (I_i, $i \in \{U, C\}$) are rogued. Visual scouting for infection occurs at time intervals of Δ.

Tomato is a climacteric fruit, meaning it can ripen once harvested from the plant (Arah et al. (2015)). Growers, therefore, who harvest before "full maturity" call still gain marketable product (Arah et al. (2015)), though there

will be a yield penalty for early harvest as immature fruit have inferior flavours and are more susceptible to damage (Tolasa et al. (2021)). As this penalty is less than the loss due to disease, growers who detect infection in their fields will do better by prematurely harvesting all crop to prevent disease progression (reducing losses by a factor ϕ_R).

The degree of symptom severity will differ between unimproved and improved crop. As such, each crop type will have its own probability of detection (ν and $\delta_\nu\nu$). We presume that when the improved crop has "tolerant" characteristics, the milder symptoms result in a low probability of detection (i.e. $\delta_\nu < 1$; Table 3.2). However, if the improved crop has "resistance" characteristics but nevertheless becomes infected, it is detected with the same probability as the unimproved crop (i.e. $\delta_\nu = 1$). The average length of time to detect an infectious field is therefore given by Δ/ϵ and $\Delta/(\delta_\epsilon\epsilon)$

The emergence of symptoms occurs at a constant rate ($1/\epsilon$ or $1/\delta_\epsilon\epsilon$) and can therefore occur at any time between the previous round of surveys (when the field was asymptomatic) and the following round (when the field is symptomatic). Infectious fields will, on average, be symptomatic for half of this time period ($\Delta/2$).

Unimproved and controlled fields are removed at rates μ_U and μ_C respec-

tively which are given by (Cunniffe et al. (2014)):

$$\mu_U = \frac{1}{\left(\frac{1}{\nu} - \frac{1}{2}\right)\Delta}, \tag{3.1}$$

$$\mu_C = \frac{1}{\left(\frac{1}{\delta_\nu \nu} - \frac{1}{2}\right)\Delta}. \tag{3.2}$$

As we are modelling on the scale of a field, we presume that if a field is rogued, it is then replanted immediately with healthy crop. The epidemiological model is given by:

$$\frac{dS_C}{dt} = \gamma C + \mu_C I_C - \delta_\beta \beta S_C (I_U + \delta_\sigma I_C) - \gamma S_C, \tag{3.3}$$

$$\frac{dE_C}{dt} = \delta_\beta S_C (I_U + \delta_\sigma I_C) - \delta_\epsilon \epsilon E_C - \gamma E_C, \tag{3.4}$$

$$\frac{dI_C}{dt} = \delta_\epsilon \epsilon E_C - \mu_C I_C - \gamma I_C, \tag{3.5}$$

$$\frac{dS_U}{dt} = \gamma U + \mu_U I_U - \beta S_U (I_U + \delta_\sigma I_C) - \gamma S_U, \tag{3.6}$$

$$\frac{dE_U}{dt} = \beta S_U (I_U + \delta_\sigma I_C) - \epsilon E_U - \gamma E_U, \tag{3.7}$$

$$\frac{dI_U}{dt} = \epsilon E_U - \mu_U I_U - \gamma I_U. \tag{3.8}$$

where $C = S_C + E_C + I_C$ and $U = S_U + E_U + I_U$.

The rate of roguing applies to the field level, but when we calculate the profits for each strategy we do so for individual growers. Roguing aims to minimise yield loss; the losses due to disease (L) are thus reduced by a factor $\phi_R < 1$ that represents the relative benefit of harvesting an infected field before the end of the season and thus avoiding the maximum yield loss. We assume that roguing and replanting a rogued field occur instantaneously. The benefit

provided by roguing appears in the profits of those growers who rogued an infectious field.

Parameters for this model are summarised in Table 3.2 and initial conditions outlined in Table 3.2.

Table 3.1: **Parameters related to resistant and tolerant varieties.** The distinction between the two varieties lies in how disease is transmitted and what losses are incurred when a field is infectious.

Parameter	Meaning	Value if resistant	Value if tolerant
δ_β	Relative susceptibility of improved variety	0.5	1
δ_σ	Relative infectivity of improved variety	0.5	1
δ_Y	Relative yield of improved variety	1	1
δ_L	Relative losses due to disease of improved variety	1	0.1
$\frac{1}{\delta_\epsilon}$	Relative latent period for improved crop	0.5	1
δ_ν	Relative probability of detection for improved crop	1	0.1

Table 3.2: **Summary of parameter values.** When the parameter relates to improved crop, the first value is for tolerant crop and the second for resistant.

Parameter	Meaning	Value	Reference
$1/\gamma$	Length of the growing season	120 days	Holt et al. (1999*a*); Rocco & Morabito (2016)
β	Rate of secondary infection	0.055/N day^{-1} field^{-1}	See main text
Δ	Time between roguing	120 days	Illustrative
ν	Probability of detection	1	Illustrative
μ_U	Removal rate (unimproved)	1/60 day^{-1}	Illustrative
μ_C	Removal rate (improved)	1/60 or 1/1140 day^{-1}	Illustrative
$\frac{1}{\epsilon}$	Average latent period	41 days	Holt et al. (1999*b*); Ber et al. (1990)
η	Responsiveness of growers	10	Chapter 2
Y	Maximum yield	1	All values scaled relative to yield
L	Loss due to infection	0.6	Riley & Srinivasan (2019)
ϕ_C	Cost of improved crop	0.1	Fonsah et al. (2018)
ϕ_R	Relative reduction in loss due to roguing	0.7	Illustrative
N	Total number of fields/growers	1	Scaled to 1

Table 3.3: **Default initial conditions.** These are scaled to be proportions (i.e. $N = 1$).

Variable	Meaning	Value
$S_C(0)$	Initial proportion of susceptible controlling fields	$0.1N$
$E_C(0)$	Initial proportion of latently infected controlling fields	$0N$
$I_C(0)$	Initial proportion of infectious controlling fields	$0N$
$S_U(0)$	Initial proportion of susceptible non-controllers	$0.89N$
$E_U(0)$	Initial proportion of latently infected non-controlling fields	$0N$
$I_U(0)$	Initial proportion of infectious non-controllers	$0.01N$

3.2.1 Parameterisation

The rate of horizontal transmission, β, is parameterised such that after 10 seasons without any improved crop or roguing, 60% of fields are infectious. This gives a value of $\beta = 0.055$ day^{-1}. This choice was a pragmatic one, as it ensured that disease spread between fields over a short period of time. However, to represent the uncertainty associated with this choice, we systematically scan over a range of values of β.

To account for the reduced susceptibility of resistant plants, we set the parameter $\delta_\beta = 0.5$ as an illustrative example (so "resistant" plants still have some probability of being infected, as would be the case for quantitative disease resistance French et al. (2016)). Resistant plants are less likely to act as sources of inoculum for whitefly vectors than susceptible plants (Lapidot et al. (2002), Legarrea et al. (2015)). We set the reduced probability of infection from an infectious, resistant field (δ_σ) as 0.5 as an illustrative example of this phenomenon.

The cropping period, γ, is 120 days, in line with tomato cultivation regimes (Holt et al. (1999*a*), Rocco & Morabito (2016)).

Complete crop losses due to TYLCV have been historically reported in many regions (e.g. in the Middle East; Czosnek & Laterrot (1997) and the United States of America; Fonsah et al. (2018)), though certain management practices can alleviate such extreme events. Use of improved cultivars when TYLC is present can increase yield by up to 40% (Vijeth et al. (2018), Riley

& Srinivasan (2019)). Riley & Srinivasan (2019) evaluated tomato yield for a combination of commonly-used control methods. When grown with silver mulch and cyantraniliprole insecticide, a susceptible variety *FL47* had a yield of 47 kg per plot, which we use as a proxy for maximum yield. Without either of those treatments, the yield was 18 kg per plot. The best resistant variety (*Security*) had a yield of 50 kg and 28 kg per plot respectively. However, under our definition, *Security* is better defined as a tolerant variety rather than a resistant one, as it did not completely restrict viral replication. Using these values we can estimate the yield loss to be $\approx 60\%$ for susceptible and resistant cultivars and up to $\approx 45\%$ in tolerant cultivars. As such, $L = 0.6$ and $\delta_L L \leq 0.45$, though both of these parameters can be scanned over to account for environmental and cultivar effects.

The latent period $\left(\frac{1}{\epsilon}\right)$, during which the host plant is infected but not infectious, can be estimated by using the number of days post infection when DNA can be detected. Significant amounts of viral DNA can be detected after 8 days (Ber et al. (1990)), which can be up to a week before symptoms appear. In Holt et al. (1999*b*), a latent period of 13 days was used. Here, we use an intermediate value of $1/\epsilon = 10$ days as the latent period of a single plant. Using the model stipulated in Holt et al. (1999*a*), though where the rate of harvesting and replanting is zero, if we then begin the season with 100% of plants in the E compartment, it takes around 41 days for 95% of these latently-infected plants to become infectious. We use this as our field-scale latent period (i.e. $\frac{1}{\epsilon} = 41$ days).

We assume that symptoms and infectivity develop over roughly the same time scale (as symptoms can develop as early as two weeks after infection (Levy & Lapidot (2008)) and we use an individual-level latent period of 10 days). Thus we assume that the $P_{SC} = P_{EC}$ and $P_{SU} = P_{EU}$.

Economic analysis of tomato production in Georgia, USA estimates that the cost of improved cultivars is approximately 25% that of the total expected profits (Fonsah et al. (2018)), though these profit forecasts included other aspects of disease control. We set the value of the cost of control, ϕ_C, as 0.1 of the total yield as an example of the extra costs control can entail, though our investigations involve a scan over possible costs.

The relative degree of tolerance and resistance will depend on both the unimproved and improved cultivars being compared. To allow for flexibility, the parameters presented in Table 3.2 are used to illustrate the effect of tolerance and resistance, though a range of parameters will be used in our investigations.

Similarly, the probability of symptom detection in an infectious field (ν) will depend on a variety of anthropological, environmental and biological factors. The values presented in Table 3.2 are baseline parameters that can then be varied in our investigations. Importantly, when improved crop is tolerant, we assume that $\delta_\nu \nu < \nu$, whereas for resistant crop we assume $\delta_\nu \nu = \nu$.

For roguing to be worthwhile, it must reduce the potential losses to a grower. Though premature harvest of fruit can incur a yield penalty (between 16-19% for vine-ripened tomatoes, Davis & Gardner (1994)), we presume that

if a grower notices a field is infectious and harvests it before the end of the growing season, it is overall more beneficial and their loss due to disease is reduced by some factor, $\phi_R < 1$. The value of ϕ_R will vary with crop cultivar and environmental conditions; the value presented in Table 3.2 is illustrative though can be varied.

3.2.2 Growers' profits

To determine the benefits provided by each strategy, we estimate the expected profits of a grower using a particular strategy. These are then compared with the grower's profit from the previous season to determine if the grower should consider switching strategy. These profits account for the costs and losses

associated with each crop type and infection outcome and are given by:

$$P_{SU} = \text{Profit for non-controller with a susceptible field} = Y, \tag{3.9}$$

$$P_{EU} = \text{Profit for non-controller with latently-infected field} = Y, \tag{3.10}$$

$$P_{IU_H} = \text{Profit for non-controller with an infected field that was not rogued} = Y - L, \tag{3.11}$$

$$P_{IU_R} = \text{Profit for non-controller with an infected field that was rogued} = Y - \phi_R L, \tag{3.12}$$

$$P_{SC} = \text{Profit for controller with a susceptible field} = Y - \phi_C, \tag{3.13}$$

$$P_{EC} = \text{Profit for controller with latently-infected field} = Y - \phi_C, \tag{3.14}$$

$$P_{IC_H} = \text{Profit for controller with an infected field that was not rogued} = Y - \phi_C - \delta_L L, \tag{3.15}$$

$$P_{IC_R} = \text{Profit for controller with an infected field and that was rogued} = Y - \phi_C - \phi_R \delta_L L. \tag{3.16}$$

The profit for an uninfected or latently-infected field using unimproved crop (P_{SU} or P_{EU}) will always be the maximum achievable profit, as at the time of harvest these growers have avoided paying the cost of control or incurring any losses due to infection. We differentiate between profits of growers with infectious fields by whether or not their field was rogued before harvesting, and thus had a lower yield loss ($\phi_R L$ or $\phi_R \delta_L L$).

As we only consider the case where the costs of control are less than the losses due to disease in unimproved crop ($\phi_C < L$), the relative sizes of the

remaining profits depend on the tolerance/resistance characteristics of the improved crop. Under the default parameterisation provided in Tables 3.2 and 2.2.2, tolerant crop, where $\delta_L L < L$, the order of profits is:

$$P_{SU} = P_{EU} > P_{SC} = P_{EC} > P_{IC_R} > P_{IC_H} > P_{IU_R} > P_{IU_H} \tag{3.17}$$

and for a default resistant background, where $\delta_L L = L$, is:

$$P_{SU} = P_{EU} > P_{SC} = P_{EC} > P_{IU_R} > P_{IU_H} > P_{IC_R} > P_{IC_H} \tag{3.18}$$

A full explanation of these orderings is found in Appendix B.1.

In the case where there is a fixed proportion of growers using each strategy, we can quantify the benefit each strategy provides by calculating the average profits for a grower using each crop type. To do this, we must first define the probability that an infectious field has not been rogued (q_{IU_H} and q_{IC_H} for unimproved and improved fields respectively):

$$q_{IU_H|I_U} = \frac{\gamma}{\gamma + \mu_U}, \tag{3.19}$$

$$q_{IC_H|I_C} = \frac{\gamma}{\gamma + \mu_C}, \tag{3.20}$$

and the field has been rogued is:

$$q_{IU_R|I_U} = \frac{\mu_U}{\gamma + \mu_U}, \tag{3.21}$$

$$q_{IC_R|I_C} = \frac{\mu_C}{\gamma + \mu_C}. \tag{3.22}$$

The average profits for unimproved (P_U) and improved (P_C) crop are given by:

$$P_U = \frac{S_U P_{SU} + E_U P_{EU} + \frac{\gamma}{\mu_U+\gamma} I_U P_{IU_H} + \frac{\mu_U}{\mu_U+\gamma} I_U P_{IU_R}}{U} \tag{3.23}$$

$$P_C = \frac{S_C P_{SC} + E_C P_{EC} + \frac{\gamma}{\mu_C+\gamma} I_C P_{IC_H} + \frac{\mu_C}{\mu_C+\gamma} I_C P_{IC_R}}{C} \tag{3.24}$$

where $U = S_U + E_U + I_U$ and $C = S_C + E_C + I_C$.

3.2.3 Calculating expected profits

In the above, growers were assigned a control strategy at the beginning of the epidemic and could not change to the alternative, irrespective of profitability or grower preference. However, it is expected that growers will instead choose whether to control based on the perceived profitability of control, which will depend on parameters such as the cost of control and also the current risk of infection.

We use the "grower vs alternative" mechanism for decision-making, as set out in McQuaid et al. (2017a), Milne et al. (2016), Saikai et al. (2021) and

Chapter 2. In this behavioural model, growers compare their outcome from the previous season with the expected profit of the alternative strategy (i.e. the strategy that they did not previously adopt), which in turn is based on the instantaneous probability of infection. The probability that they change strategy is then based on the magnitude of the differences between their previous profit and the profit of the alternative strategy.

The probability of horizontal transmission for a grower using unimproved crop (q_U) is given by:

$$\begin{aligned} q_U &= \text{Instantaneous probability of horizontal infection (non-control)}, \\ &= \frac{\text{Instantaneous infection rate}}{\text{Instantaneous infection rate + Harvesting rate}}, \\ &= \frac{\beta(\delta_\sigma I_C + I_U)}{\beta(\delta_\sigma I_C + I_U) + \gamma}, \end{aligned} \tag{3.25}$$

and for a grower using improved crop (q_C) is:

$$\begin{aligned} q_C &= \text{Instantaneous probability of horizontal infection (control)}, \\ &= \frac{\text{Instantaneous infection rate}}{\text{Instantaneous infection rate + Harvesting rate}}, \\ &= \frac{\delta_\beta \beta(\delta_\sigma I_C + I_U)}{\beta(\delta_\sigma I_C + I_U) + \gamma}. \end{aligned} \tag{3.26}$$

We must also consider the probability that, once infected, a grower will be latently infected (E) or infectious (I). The probabilities that a field planted

with unimproved or improved crop will be latently infected at the time of harvest (q_{EU} and q_{EC}) are given by:

$$q_{EU} = q_U \left(\frac{\gamma}{\epsilon + \gamma} \right), \tag{3.27}$$

$$q_{EC} = q_C \left(\frac{\gamma}{\delta_\epsilon \epsilon + \gamma} \right), \tag{3.28}$$

whilst the probabilities the field is infectious (q_{IU} and q_{IC}) are:

$$q_{IU} = q_U \left(\frac{\epsilon}{\epsilon + \gamma} \right), \tag{3.29}$$

$$q_{IC} = q_C \left(\frac{\delta_\epsilon \epsilon}{\delta_\epsilon \epsilon + \gamma} \right). \tag{3.30}$$

Finally, we must consider the probability that a field is infectious and then rogued (q_{IU_R} and q_{IC_R}) before it is harvested:

$$q_{IU_R} = q_{IU} \left(\frac{\mu_U}{\gamma + \mu_U} \right), \tag{3.31}$$

$$q_{IC_R} = q_{IC} \left(\frac{\mu_C}{\gamma + \mu_C} \right), \tag{3.32}$$

or that it is harvested (q_{IU_H} and q_{IC_H}) before being rogued:

$$q_{IU_H} = q_{IU} \left(\frac{\gamma}{\gamma + \mu_U} \right), \tag{3.33}$$

$$q_{IC_H} = q_{IC} \left(\frac{\gamma}{\gamma + \mu_U} \right). \tag{3.34}$$

The expected profits for a non-controller, P_U is therefore given by:

P_U = Grower's estimate of the expected profit next season if control is not adopted,

$$= (1 - q_U)P_{SU} + q_{EU}P_{EU} + q_{IU_H}P_{IU_H} + q_{IU_R}P_{IU_R}, \tag{3.35}$$

and for a controller (P_C) it is:

P_C = Grower's estimate of the expected profit next season if control is adopted,

$$= (1 - q_C)P_{SC} + q_{EC}P_{EC} + q_{IC_H}P_{IC_H} + q_{IC_R}P_{IC_R} \tag{3.36}$$

Using Equations 3.9 - 3.16, Equations 3.35 and 3.36 be further simplified:

$$P_U = Y - q_U\frac{\epsilon}{\epsilon+\gamma}L\left(\frac{\gamma}{\gamma+\mu_U} + \frac{\mu_U}{\mu_U+\gamma}\phi_R\right), \tag{3.37}$$

$$P_C = Y - \phi_C - q_C\frac{\delta_\epsilon\epsilon}{\delta_\epsilon\epsilon+\gamma}\delta_L L\left(\frac{\gamma}{\gamma+\mu_C} + \frac{\mu_C}{\mu_C+\gamma}\phi_R\right). \tag{3.38}$$

3.2.4 Switching terms based on the expected profits

From Equations 3.37 - 3.38, we can determine the probability of a grower of outcome $i \in \{S_U, E_U, I_{U_H}, I_{U_R}, S_C, E_C, I_{C_H}, I_{C_R}\}$ switching into the alternative strategy (Chapter 2) (note: these outcomes differ from the state variables we track in the Equations 3.3 - 3.8, as when calculating profits we track whether a field has been rogued). These "switching terms" compare the difference between the grower's profit from the previous season with the expected profit of the strategy that they did not adopt. These differences are multiplied by a

"responsiveness" parameter, η, which accounts for the responsiveness of growers to differences in profit (Chapter 2). If the expected profit is less than the grower's current profit, the grower should not switch strategy. The payoff for a non-controller who harvests susceptible crop (P_{SU} and, for the default parameterisation, P_{EU} from latently-infected fields) should always be the highest as they do not pay the cost of control or losses due to disease. These growers should therefore never switch strategy. Which is the lowest payoff will depend on the strength of the tolerance or resistance traits in the improved corp, as well as the relative costs (Equations 3.17-3.18; Appendix B.1). If it is tolerant, the loss for tolerant crop will be less than that of unimproved crop ($\delta_L L < L$). Consequently, P_{IU_R} is the lowest payoff. Conversely, if the improved crop is resistant, $\delta_L L = L$ and the cost of control means that P_{IC_R} is the lowest payoff.

The switching terms are given by:

$$z_{SU} = \max\left(0, 1 - e^{-\eta(P_C - P_{SU})}\right), \tag{3.39}$$

$$z_{EU} = \max\left(0, 1 - e^{-\eta(P_C - P_{EU})}\right), \tag{3.40}$$

$$z_{IU_H} = \max\left(0, 1 - e^{-\eta(P_C - P_{IU_H})}\right), \tag{3.41}$$

$$z_{IU_R} = \max\left(0, 1 - e^{-\eta(P_C - P_{IU_R})}\right), \tag{3.42}$$

$$z_{SC} = \max\left(0, 1 - e^{-\eta(P_U - P_{SC})}\right), \tag{3.43}$$

$$z_{EC} = \max\left(0, 1 - e^{-\eta(P_U - P_{EC})}\right), \tag{3.44}$$

$$z_{IC_H} = \max\left(0, 1 - e^{-\eta(P_U - P_{IC_H})}\right). \tag{3.45}$$

$$z_{IC_R} = \max\left(0, 1 - e^{-\eta(P_U - P_{IC_R})}\right). \tag{3.46}$$

Incorporating these into the epidemiological model, we have:

$$\frac{dS_C}{dt} = \gamma\theta_C - \delta_\beta\beta S_C(I_U + \delta_\sigma I_C) + M_C - \gamma S_C, \tag{3.47}$$

$$\frac{dE_C}{dt} = \delta_\beta\beta S_C(I_U + \delta_\sigma I_C) - \delta_\epsilon\epsilon E_C - \gamma E_C, \tag{3.48}$$

$$\frac{dI_C}{dt} = \delta_\epsilon\epsilon E_C - \mu_C I_C - \gamma I_C, \tag{3.49}$$

$$\frac{dS_U}{dt} = \gamma\theta_U - \beta S_U(I_U + \delta_\sigma I_C) + M_U - \gamma S_U, \tag{3.50}$$

$$\frac{dE_U}{dt} = \beta S_U(I_U + \delta_\sigma I_C) - \epsilon E_U - \gamma E_U, \tag{3.51}$$

$$\frac{dI_U}{dt} = \epsilon E_U - \mu_U I_U - \gamma I_U. \tag{3.52}$$

where:

$$\theta_C = (1 - z_{SC})S_C + (1 - z_{EC})E_C + (1 - z_{IC_H})I_C + z_{IU_H}I_U, \tag{3.53}$$

$$\theta_U = S_U + (1 - z_{EU})E_U + (1 - z_{IU_H})I_U + z_{SC}S_C + z_{EC}E_C + z_{IC_H}I_C, \tag{3.54}$$

$$M_C = (1 - z_{IC_R})\mu_C I_C + z_{IU_R}\mu_U I_U, \tag{3.55}$$

$$M_U = z_{IC_R}\mu_C I_C + (1 - z_{IU_R})\mu_U I_U, \tag{3.56}$$

$$N = S_C + E_C + I_C + S_U + E_U + I_U. \tag{3.57}$$

We highlight here that in Equation 3.54, S_U and E_U are not associated with any switching terms as for our parameterisation, the values of $z_S U$ and $z_E U$ are zero. A schematic of the model is provided in Figure 3.2.

Which switching terms are positive or fixed at zero depends on the parameter values and the epidemiological state of the system. Due to the ordering of

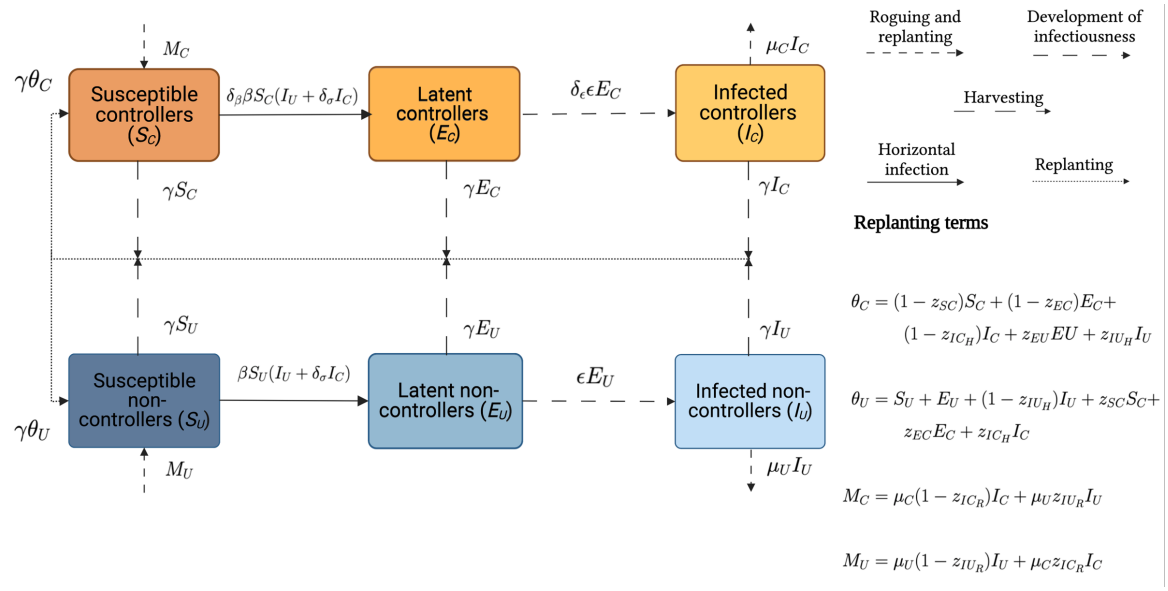

Figure 3.2: **Schematic showing the structure of the model when growers can choose their strategy based on expected profits.** We have two classes of grower, those who use unimproved seed (U) and those who use improved seed (C). This improved seed comes in one of two varieties: tolerant or resistant. The terms θ_C and θ_U are the rates of replanting for harvested improved and unimproved fields, whilst M_C and M_U are rates of replanting for rogued fields (Equations 3.53 - 3.56, with the total replanting rate of rogued fields given by: $M_C + M_U = \mu_C I_C + \mu_U I_U$). Created with BioRender.com

the payoffs, only certain combinations of positive switching terms are possible

(Appendix B.1).

3.3 Results

3.3.1 Externalities of tolerant and resistant crop with fixed proportions of growers

Basic reproduction number, R_0

When there is only one type of crop (improved, which can be either resistant or tolerant, or unimproved), the basic reproduction numbers are as follows:

$$R_{0U} = \frac{\beta\epsilon N}{(\mu_U + \gamma)(\epsilon + \gamma)}, \tag{3.58}$$

$$R_{0C} = \frac{\delta_\beta\beta\delta_\epsilon\epsilon\delta_\sigma N}{(\mu_C + \gamma)(\delta_\epsilon\epsilon + \gamma)}. \tag{3.59}$$

where for R_{0U}, $N = U$ and for R_{0C} $N = C$. In these expressions, $\frac{\epsilon}{(\mu_U+\gamma)(\epsilon+\gamma)}$ and $\frac{\delta_\epsilon\epsilon}{(\mu_C+\gamma)(\epsilon+\gamma)}$ are the probabilities that a field will become infectious before it is harvested for unimproved and controlled fields respectively. The mean time spent in the I_i compartment is $\frac{1}{\mu_i+\gamma}$ (with $i \in \{U, C\}$) and the number of infections given caused by these infectious fields is βU or $\delta_\beta\beta C$ for unimproved or improved respectively. The value of R_{0C} also accounts for the reduced infectivity of infectious improved crop (δ_σ).

We use the next generation matrix method (NGM; Diekmann et al. (2010)) to evaluate R_0 when both crop types are present at the disease-free equilibrium (i.e. $(S_U, E_U, I_U, S_C, E_C, I_C) = (U, 0, 0, C, 0, 0)$) (see Appendix B.2).

R_0 when both crop types are present is given by:

$$\begin{aligned} R_0 &= \frac{U}{N}R_{0U} + \frac{C}{N}R_{0C}, \\ &= \frac{\beta\epsilon U}{(\gamma+\mu_U)(\gamma+\epsilon)} + \frac{\delta_\beta\beta\delta_\epsilon\epsilon\delta_\sigma C}{(\gamma+\mu_C)(\gamma+\epsilon).} \end{aligned} \tag{3.60}$$

This is a combination of Equations 3.58 and 3.59, scaled in proportion with the proportion using each crop type (Gandon (2004), Bosch et al. (2008)).

Effect of changing proportion of improved crop

We first consider the effect of an increased proportion of improved crop on the expected profits of growers of each type. Using the default parameters outlined in Table 3.2, we can see that an increase in the proportion of growers using tolerant crops has little impact on the expected profit of controllers (decreasing profits by ~1%), though reduces those of non-controllers (Figure 3.3(A)). As tolerant crops have a lower probability of being detected ($\delta_\nu\nu = 0.1$) and thus a lower removal rate (μ_U), having more tolerant crop increases the disease pressure as fewer infectious fields are removed via roguing (Figure 3.3(C)).

Conversely, an increase in the proportion of resistant crops provided much greater benefits to non-controllers than to controllers (Figure 3.3(B)). Controllers already had a relatively low probability of infection, so their average profit is already close to the maximum possible ($P_{SC} = 0.85$). Thus, a decrease in the probability of infection due to an increased proportion of growers using resistant crop provides little additional benefit. With a sufficient proportion

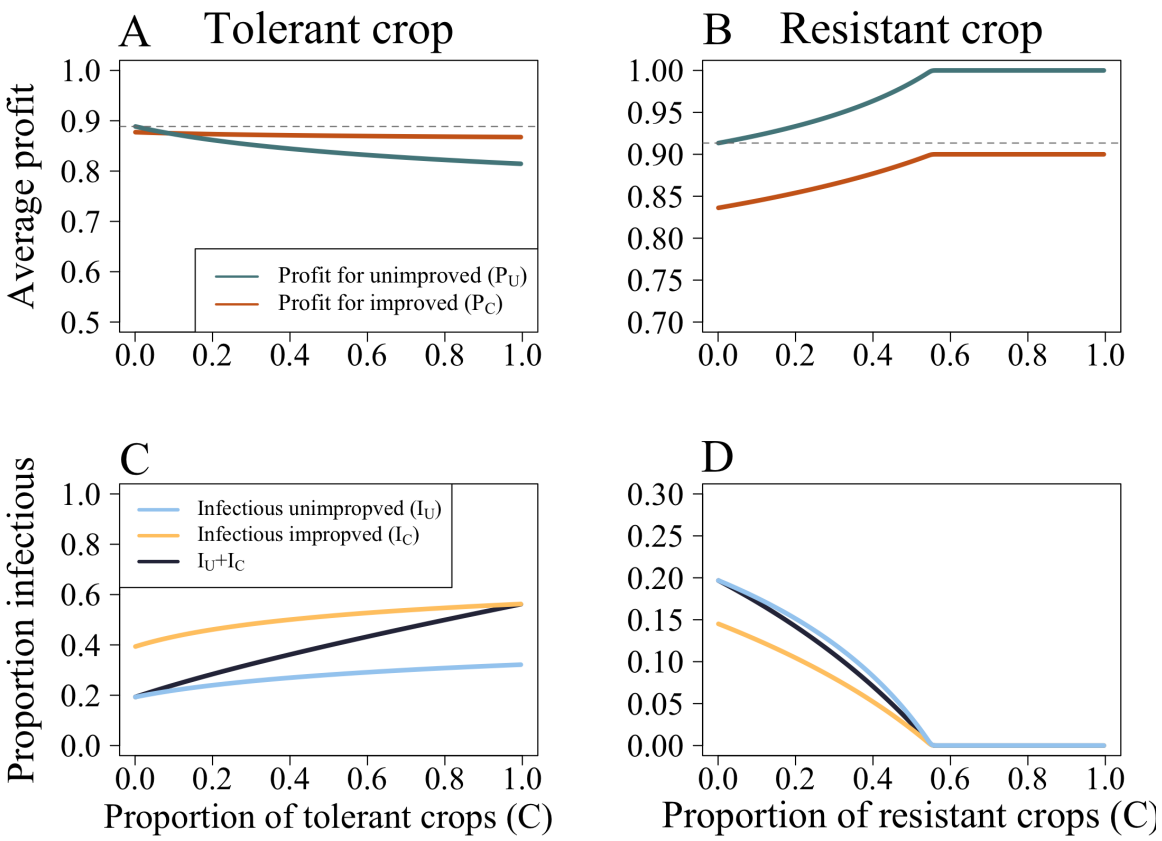

Figure 3.3: **Effect of increasing proportions of improved crop on the average profit of growers using unimproved and improved crop.** (A) and (C) use the tolerant parameterisation whilst (B) and (D) show the same for resistant crops. The two types of improved crop had opposite effects; as the proportion of improved, tolerant crop increased, so too did the amount of infection. This caused a decrease in profits for non-controllers. Conversely, as C increased when the crop was resistant, there was a decrease in the amount of infection and concomitant increases in profits. In both cases, the grey dashed line shows the average profit of a non-controller when there are no controllers ($C = 0$), which can be used to measure the externalities generated by each type of improved crop. Parameters and initial conditions are as in Tables 3.2 and 3.2 respectively.

of resistant crops, disease is eliminated from the system (Figure 3.3(D)). However, in this scenario controllers had to continue paying the cost of control (ϕ_C) even though there is little need for control, so the controllers earn less than the non-controllers.

In both of Figure 3.3(A) and (B), the deviation from the average profits

where there is no improved crop in the system ($C = 0$, indicated by the grey dashed line) can be seen as the magnitude of the externalities generated by each crop type. As an increase in tolerant crop causes P_U to decrease, it generates negative externalities. Conversely, the resistant crop reduces the probability of infection and thus generates positive externalities, increasing P_U. The increase in P_U at higher values of C (from $P_U = 0.91$ when $C = 0$ to $P_U = 1$ when $C = 0.99$) is greater than the corresponding increase in P_C (from from $P_C = 0.836$ when $C = 0$ to $P_C = 0.9$ when $C = 0.99$), indicating that a greater benefit is felt by non-controllers than by controllers.

We have shown the above for only a single set of parameters, but the broad patterns are recapitulated for parameters controlling the effectiveness of the tolerant/resistant crop (namely the probability of detection of improved crop ($\delta_\nu \nu$) and the relative susceptibility of improved crop (δ_β) (Appendix B.3 Figs. B.3.1 and B.3.2)).

3.3.2 Effect of initial conditions on long-term outcomes in the behavioural model

The complexity of the model (in particular, the presence of the switching terms) means that explicit expressions cannot be found for the values of state variables at equilibrium. However, their values and the stability of the equilibria can be evaluated numerically.

Using the NGM method (see Appendix 4), we found the basic reproduction

number for the behavioural model to be:

$$R_0 = \frac{\beta \epsilon N}{(\mu_U + \gamma)(\epsilon + \gamma)}. \tag{3.61}$$

Broadly, this determines the stability of the disease-free equilibrium, as disease can only invade the system once $R_0 > 1$. In some cases, however, bistability is possible and there may be multiple possible equilibria below this threshold depending on the initial conditions (Gumel (2012)).

The long-term outcomes of the model can be divided into one of four types:

- **Disease-free equilibrium:** as $R_0 < 1$, there are no infected fields. As there is no risk of infection, no growers use improved crop (and therefore avoid the cost of control, ϕ_C).
- **"No control" equilibrium:** disease is endemic, but no growers use improved crop.
- **"All control" equilibrium:** disease is endemic and no growers use unimproved crop.
- **Two-strategy equilibrium:** disease is endemic and crops of both varieties are used.

For a given parameter set, it may be that two of these equilibria are locally stable, depending on the initial conditions.

The parameterisation of the model, both in terms of whether the improved crop is tolerant or resistant and its degree of tolerance and resistance, deter-

mines the subset of possible long-term outcomes. When the improved crop was resistant, an "all control" equilibrium was not possible (Figure 3.4(A)). The positive externalities generated by the presence of resistant crop disincentivises non-controllers from using improved crop, as they have a lowered probability of infection without themselves being infected. Thus, as the proportion using resistance reaches increasingly high levels, fewer non-controllers will switch to using the control scheme.

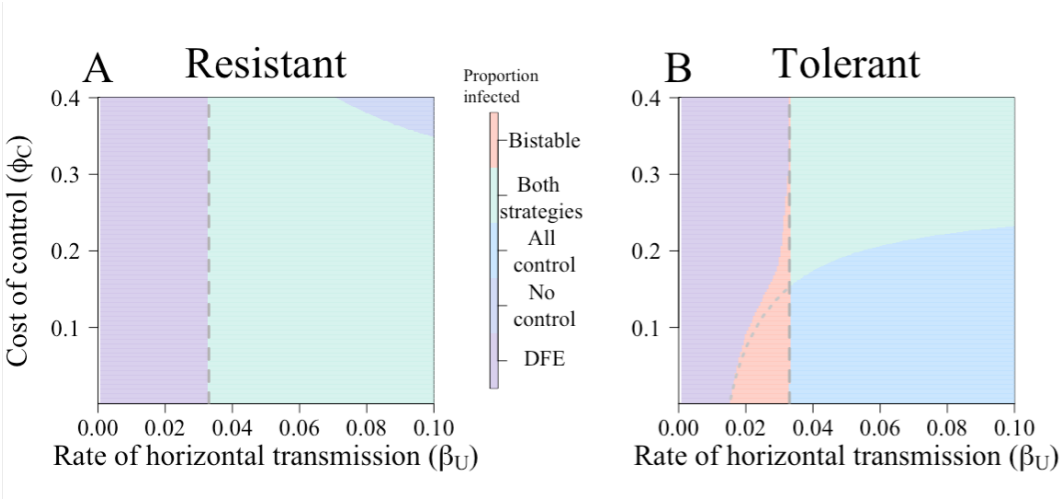

Figure 3.4: **Nature of equilibria attained when improved crop is either tolerant or resistant.** There are five possibilities: a "disease-free equilibrium" (DFE), with only unimproved crop; a disease-endemic, "no control" equilibrium; a disease-endemic, "all control" equilibrium; and an equilibrium where both strategies and disease are present and finally a bistable region of parameter space, where two stable equilibria are possible and so the long-term behaviour of the system depends on the initial conditions (see also Figure 3.5). For both parameterisations $R_0 = 1$ is indicated by the grey vertical dashed line at $\beta = 0.0333$ day $^{-1}$. (A) Equilibria for the resistant crop. Here, only three of the possible equilibria exist: at no point can there be an "all control" equilibrium. Additionally, there is no bistable region. (B) Equilibria for the tolerant crop. Now, at lower costs of control and medium-to-high values of β, an equilibrium where all growers use improved crop is possible. There is a bistable region for $0.015 < \beta < 0.0333$ day^{-1} (depending on the value of the cost, ϕ_C). Below the dotted line in the pink bistable region, there is bistability between the "all control" and DFE; above, there is bistability between the two-strategy equilibrium and the DFE.

When the improved crop was tolerant, however, such an "all control" equilibrium was possible (Figure 3.4(B)). The primary benefit of tolerant crop is that, when infection occurs, there is a lower loss of yield compared to unimproved (or resistant) crop. As there is no reduction in the viral titre, tolerant crops are just as likely to act as sources of infection as unimproved crops. Thus, the benefits of planting tolerant crop are experienced by the grower that plants tolerant crop, so there is no disincentive for other growers to also use it.

We then further investigated the initial conditions that could lead to bistability. Figure 3.5(A) shows the effect of the rate of horizontal transmission (β) on the stability of equilibria. There is a bistable region between $\beta = 0.02$ and 0.0333 day $^{-1}$, and which equilibrium (either the disease-free or all-control equilibrium) is attained will depend on initial conditions. The discontinuous system leads to kinks in the graphs, whose ordering follows that outlined in Appendix B.1.

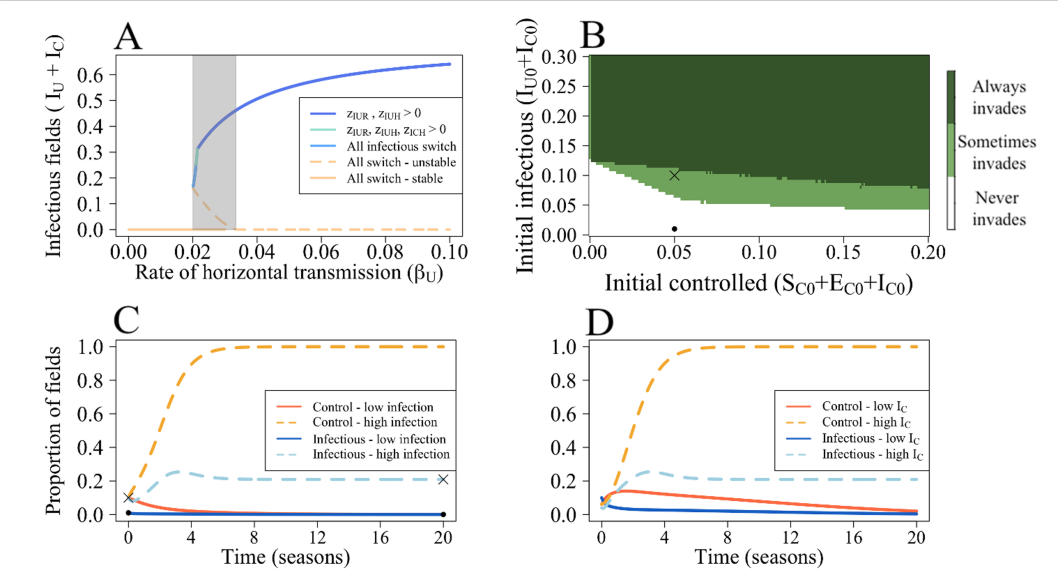

Figure 3.5: **Bifurcation for the tolerant parameterisation.** (A) Between $\beta = 0.02$ and 0.0333 day $^{-1}$ (the grey region), two biologically-meaningful equilibria are possible. Below $\beta = 0.02$ day $^{-1}$, only the disease-free equilibrium (DFE) is stable; above $\beta = 0.0333$ day $^{-1}$ (when $R_0 > 1$), only the disease-endemic equilibrium is. The kinks in the graph are caused by the change in form of the switching terms. (B) The effect of the initial proportion of tolerant fields ($S_{C0} + E_{C0} + I_{C0}$) and infectious fields ($I_{U0} + I_{C0}$) on the persistence of disease at equilibrium. To only account for these factors, $E_{U0} = E_{C0} = 0$. When $S_{C0} + E_{C0} + I_{C0}$ and $I_{U0} + I_{C0}$ are very low, all initial conditions lead to the DFE. As these quantities increase, so too does the probability of disease persistence. The black cross and dot denote the proportions used in (C). (C) When $I_{U0} + I_{C0} = 0.01$ ("low infectious"), disease died out. However, once $I_{U0} + I_{C0} = 0.1$, disease persisted. (D) For the "high infection" parameterisation from (C), we investigated the effect of the proportion of infectious improved crop. When $\frac{I_{C0}}{I_{U0}+I_{C0}} > 0.5$ (as $(S_{U0}, E_{U0}, I_{U0}, S_{C0}, E_{C0}, I_{C0}) = (0.83, 0, 0.03, 0.03, 0, 0.07)$; "high I_C"), disease can persist. However, if $\frac{I_{C0}}{I_{U0}+I_{C0}} < 0.5$ (as $(S_{U0}, E_{U0}, I_{U0}, S_{C0}, E_{C0}, I_{C0}) = (0.83, 0, 0.07, 0.07, 0, 0.03)$; "low I_C"), disease dies out. Other than $\beta = \delta_\beta\beta = 0.0028$ day^{-1} (which is within the bistable region), parameters are as in Table 3.2.

The initial proportions of growers using tolerant crop ($S_{C0} + E_{C0} + I_{C0}$) and infectious fields ($I_U + I_C$) had a large impact on the final proportion of infectious fields. We did this for $\beta = \delta_\beta\beta = 0.0028$ day^{-1}, which is within the bistable region in Figure 3.5(A). At very low levels of both initially controlled

and infectious fields, the system always goes to the disease-free equilibrium (DFE), with no disease persisting (Figure 3.5(B)). As both increase, disease is more likely to invade until both controlled and infectious fields are sufficiently high that the system will always go to a disease-endemic equilibrium.

We compared two sets of initial conditions, which differed in their initial proportion of infectious fields. In Figure 3.5(C), $S_{C0} + E_{C0} + I_{C0} = 0.1$ (i.e. initially 10% of fields are planted with improved crop). In the "high infection" scenario, 10% of fields are infectious; for the "low infection" scenario, this is just 1%. In the former scenario, disease persisted at equilibrium, whilst it died out in the latter.

The ratio of initially-infectious improved and unimproved crop (Figure 3.5(D)) was important in determining disease persistence. If 70% of initially-infected fields were tolerant ($(S_{U0}, E_{U0}, I_{U0}, S_{C0}, E_{C0}, I_{C0}) = (0.83, 0, 0.03, 0.03, 0, 0.07)$; "high I_C") disease persisted as it did in Figure 3.5(C). However, if there were fewer infectious tolerant fields ($(S_{U0}, E_{U0}, I_{U0}, S_{C0}, E_{C0}, I_{C0}) = (0.83, 0, 0.07, 0.07, 0, 0.03)$; "low I_C"), disease died out. Though this is not the only condition for disease persistence when $R_0 < 1$, as at high levels of I_{U0} disease can persist even if there are no I_{C0} fields, these results cumulatively suggest that the presence of infected tolerant crop early in the epidemic can lead to alternative equilibrium being attained. This is possibly driven by the lower probability of detection of tolerant crop ($\delta_\nu \nu = 0.1$), which means that it is not removed quickly once it becomes infectious and allows disease to spread.

3.3.3 Impact of tolerant or resistant crop on grower behaviour

Effect of epidemiological and economic parameters on grower behaviour

We first investigated the impact of changes to the rate of horizontal transmission on the adoption of improved crop. For all values of the rate of horizontal transmission in non-improved crop (β), the proportion of infected fields was higher when the improved crop was tolerant than when it was resistant (Figure 3.6(A) *vs* b). Disease could also invade at a lower value of β (in the bistable region where $R_0 < 1$). The expected profits for both strategies were higher when there was resistant crop (Figure 3.6(C)-(D)), and the non-controllers gained more benefit from others using the resistant crop than from the tolerant crop.

Generally, for both crop varieties, as β increased so too did the proportion of fields controlling. With the tolerant improved crop, this led to a higher proportion of infectious fields ($I_U + I_C$; Figure 3.6(B)), as tolerant crops have a reduced probability of being rogued so there is a higher disease pressure on other fields in the system. Once disease invades, at no point should growers of either strategy that have susceptible or latently-infected fields switch strategy as $P_U < P_{SC,EC}$ and $P_C < P_{SU,EU}$.

The trend is broadly similar for resistant crop. However, as β increases, there is a decrease in the proportion controlling. As P_U approaches $P_{SC,EC}$, fewer growers managing fields of these types should switch strategy. However,

the increase in infection pressure means that more of these fields will become infected, achieving the lowest payoff. Thus, the growers have a non-zero probability of switching strategy.

Once $\beta > 0.067$ day^{-1} (marked by the vertical dashed line in Figure 3.6), the proportion of controllers falls. At this point, the high infection pressure means the expected profits of non-controllers exceed those of susceptible or latently-infected controllers, so they do not switch strategy ($P_U > P_{SC} = P_{EC}$). Yet the fields of these controllers are still likely to get infected as resistance is incomplete, so the growers will incur the double penalty of the cost of control and loss due to disease. Thus, as $P_{IC_H,IC_R} < P_U$ for all values of β, growers managing infected fields with improved crop should always consider switching strategy. As there are now fewer fields planted with resistant crop and the overall disease pressure increases, so too does the number of infectious fields.

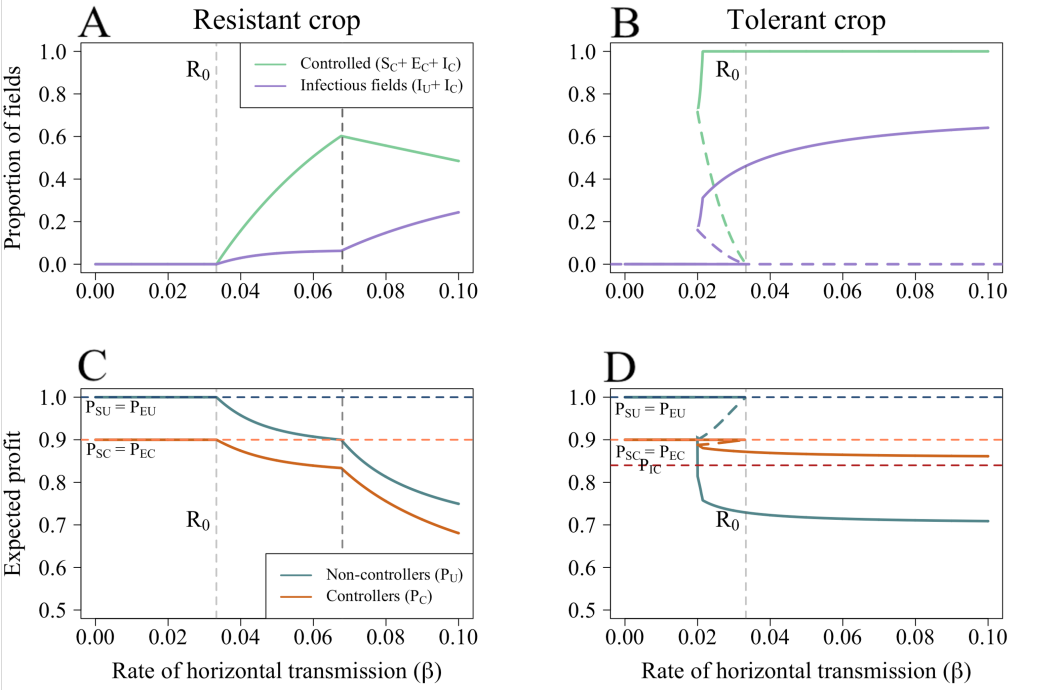

Figure 3.6: **Response to the rate of horizontal transmission in non-improved crops (β).** (A) Change in controllers and infectious fields when the improved crop has resistant characteristics. Generally, with an increase in β comes an increase in C, though at $\beta = 0.067$ day^{-1} there is a decrease and corresponding increase in infectious fields. (B) The change in proportion of growers using the improved crop (C) and infectious fields ($I_U + I_C$) when the improved crop has tolerant characteristics. (C) Change in the expected profit for non-controllers (P_U, Equation 3.35) and controllers (P_C, Equation 3.36) for resistant crop. Until $\beta = 0.067$ day^{-1} , controllers with susceptible or latently-infected crop should switch strategy as $P_U > P_{SC,EC}$. After this point, they should stop switching. As $P_{IC_H,IC_R} < P_U$, these growers should always change strategy, leading to a fall in the proportion of controllers. (D) Change in the expected profit for non-controllers (P_U) and controllers (P_C). For each value of β, growers managing susceptible or latently-infected fields of neither strategy should change (as $P_U < P_{SC,EC}$ and $P_C < P_{SU,EU}$). In (C) and (D), the dashed lines show the outcomes achieved by different classes of growers (given by Equations 3.9 - 3.16), which do not change with changing β. Other than those being varied, parameters and initial conditions are as in Tables 3.2 and 3.2 respectively.

The response to infection rate changed for different values of the cost of control, ϕ_C (Figure 3.7). When the parameterisation was tolerant (Figure 3.7(A)), bistability existed for values of $\phi_C <= 0.3$, though the region where

bistability was possible narrowed as the cost of control increased. When $\phi_C = 0.1$ or 0.2, there existed a scenario where grower behaviour meant that only tolerant crop was possible. Even at very high costs of control ($\phi_C = 0.5$), some growers nevertheless controlled at equilibrium.

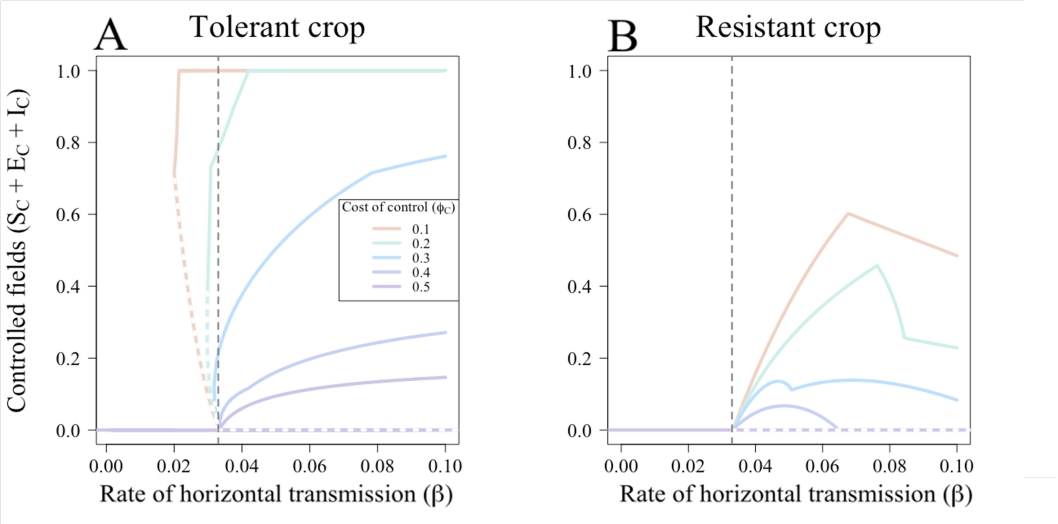

Figure 3.7: **Effect of the cost of control (ϕ_C) on uptake of improved crop.** The grey dashed lines show where $R_0 = 1$), and grey dots show unstable equilibria. (A) When improved crop is tolerant, when $\phi_C \leq 0.3$, bistability is possible. The range of β values for which bistability exists is larger with a lower ϕ_C. Even at very high costs of control ($\phi_C > 0.4$), control persists at equilibrium. (B) When improved crop is resistant, no bistability exists. Only once $R_0 > 1$ can control persist. However, as β gets larger, even at low costs of control fewer growers use resistant crop. Note, the maximum possible value for ϕ_C when the crop is resistant is $\phi_C = 0.4$, otherwise payoff for infected resistant crop, P_{IU_H}, would be negative for the default parameterisation.

When the improved crop is resistant, disease can only invade once $R_0 > 1$, irrespective of the cost of control. Once disease invades, control only persists for a narrow range of β, as above a certain threshold, control is seen as too costly (as growers are more likely to pay the dual penalty of the cost of control and loss due to disease, L_C, thus earning the lowest possible profit, P_{IC_H}). The range for which resistant crop is used at all is narrower for larger values

of ϕ_C. The kinks in this graph (such as in Figure 3.7(A) when $\phi_C = 0.3$ and $\beta = 0.78$ day^{-1} or Figure 3.7(B) when $\phi_C = 0.1$ and $\beta = 0.72$ day^{-1}) are caused by changes in the switching terms, discussed in detail in Appendix 5 Figure B.5.1.

Comparison of profits for tolerant and resistant crop

We now compare the expected profits at equilibrium for controllers and non-controllers when the improved crop is either tolerant or resistant (Figure 3.8, which shows the difference in expected profit for non-controllers (A) and controllers (B) when crop is resistant and when it is tolerant). The expected profits for unimproved, tolerant and resistant crop is shown in Appendix 5 Figure B.5.3. When generating these graphs, we chose initial conditions that would always guarantee a disease-endemic equilibrium in the bistable region to ensure that differences are calculated between comparable equilibria ($I_{U0} + I_{C0} = 0.15, S_{C0} + E_{C0} + I_{C0} = 0.2$, Figure 3.5(B)).

Directly comparing the types of improved crop and their effect on the expected profits, we can see that for non-controllers, the presence of the improved crop in the system is always more beneficial when crop is resistant (Figure 3.8(A)). However, whether tolerant or resistant crop improves controllers' profits depends on parameter values (Figure 3.8(B)). At low-to-medium costs of control (ϕ_C) and transmission rates (β), those growing resistant crop earn slightly more than they would if they used tolerant crop. Both improved crop types cost the same amount (ϕ_C), so it is more advantageous to use resistant

crop when there is already a low probability of infection, and effectively completely avoid incurring the yield loss due to infection, than to be tolerant and risk losing yield (even if the yield loss itself is small).

In both graphs, the increase and subsequent decrease in the benefit of resistance for non-controllers and resistant controllers that begins when $\beta = 0.064$ day $^{-1}$ in Figs. 3.8(A)-(B) is caused by changes in the switching terms (Appendix B.1) and changes in infection pressure.

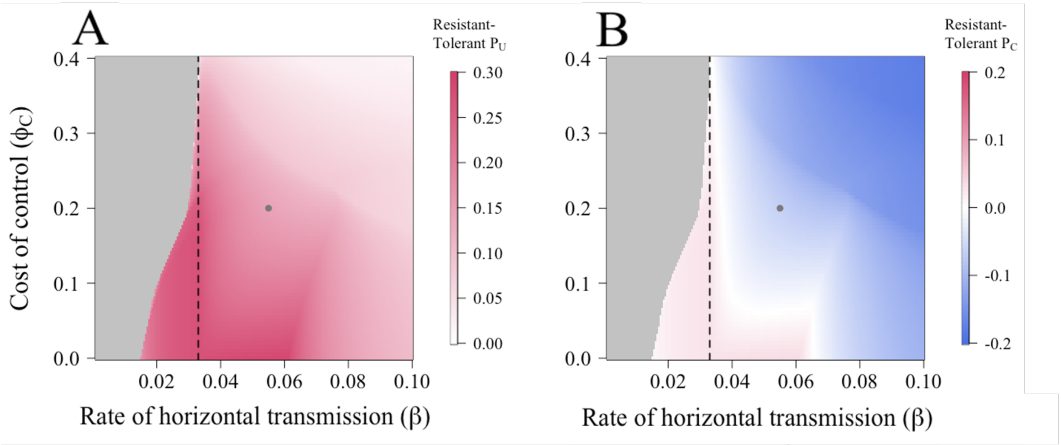

Figure 3.8: **Difference in expected profits when there is resistant crop vs tolerant crop.** (A) The expected profit for unimproved crop and (B) the expected profit for improved crop. The grey dots indicate the default parameterisation and the white section below $R_0 = 1$ (the black dashed line) is where there is no difference in profit due to the lack of improved crop being used. Other than those being varied, parameters and initial conditions are as in Tables 3.2 and 3.2 respectively.

3.4 Discussion

Though tolerance and resistance to disease have been widely researched in plant biology, little thought has been given to the distinct epidemiological consequences of deploying tolerant or resistant varieties upon a community of growers, and none to how this affects the decisions of growers to partake in control. Growers' behaviour more broadly, and its effects on epidemic outcomes, remains largely overlooked in plant disease epidemiology (with some exceptions including Milne et al. (2016), McQuaid et al. (2017a), Szyniszewska et al. (2021), Milne et al. (2020), Bate et al. (2021), Saikai et al. (2021) and Chapter 2). Here we investigated the effect of a fixed proportion of "improved" crop (with either tolerant or resistant characteristics) on growers' profits and subsequently how this affected growers' use of improved crop when given the choice. Though the models we developed can in principle be applied to a broad range of pathosystems, we demonstrate the model using TYLCV as a case study.

As is intuitive from the underlying epidemiology, when a fixed proportion of growers was assigned tolerant crop, there was an increase in the proportion of infectious fields compared to when the improved crop was resistant (Figure 3.3(B) *vs* (D)). However, for the tolerant crop, the negative impact of having more infectious fields was predominantly felt by the growers using unimproved crop, who saw a larger fall in profits than the controllers. This was due to the negative externalities generated by the use of tolerant crop; as tolerant crop

has reduced symptom development, it has a lower rate of removal via roguing. This allows more disease to build in the system, increasing the probability of infection for fields with unimproved crop and consequently reducing the profit of non-controllers.

When there was a constant proportion of growers using each strategy, when the proportion using resistant crop, $C > 0.55$, disease goes extinct (Figure 3.3(D)). Despite the increase in profits experienced by all growers when disease was eliminated via the use of the resistant crop, most of the benefit was experienced by non-controllers, who saw a more substantial increase in profits than the controllers (Figure 3.3(B)). This echoes past studies showing that if some proportion of growers in a landscape do control for disease, the benefits are widely felt (Hutchison et al. (2010),van den Bosch & Gilligan (2003), Lo Iacono et al. (2013)). In our case, growers of resistant crop generated positive externalities, benefiting others whilst incurring a cost themselves. Thus, the resistant crop is more beneficial to growers of both strategies than tolerant crop (which cannot be used to eliminate disease and increase the payoffs of non-controllers).

The nature of these contrasting externalities suggests that, given the choice, relatively few growers should choose to use resistant crop when available as they will gain more benefit when they do not (i.e. they will "free-ride" off the costs incurred by controllers). Similarly, it suggests growers using tolerant crops incentivises others to do so too, as they will have higher yields when infected. To investigate these dynamics, we included growers' behaviour in our disease

spread model, with growers evaluating profitability based on the "grower vs alternative strategy" method described in Chapter 2 (which takes the same form as decision models in Milne et al. (2016), McQuaid et al. (2017a) and Saikai et al. (2021)). In this model, growers compared their own outcome from the previous season (i.e. whether they had a rogued field that had resistant crop, a susceptible field with unimproved crop *etc.*) with the average expected profit of the alternative strategy. The model is based on "strategic-adaptive" expectations, which balances a grower's previous experience against the probability of future events (Fenichel & Wang (2013)).

Once growers' behaviour is introduced, the threshold proportion of growers using resistant crop needed to eliminate disease is never reached, even at high disease pressures (Figure 3.4(A)). This is because of "free-riding"; growers gain more benefit from the protection provided by others using resistant crop than they would if they themselves used resistant crop (in Figure 3.6(C), the expected profit for non-controllers is higher than for controllers ($P_U > P_C$)). Thus, though the use of resistant crop can theoretically lead to disease elimination, when considering the behaviour of growers it is not possible.

When the improved crop was tolerant, however, bistability between disease-free and disease-endemic equilibria was observed when the basic reproduction number (R_0) was less than unity (Figure 3.4(B)). Previous epidemiological models including factors such as imperfect vaccination, risk-structure or re-infection (e.g. Arino et al. (2003), Gumel & Song (2008), summarised in Gumel (2012)), vector dynamics (Garba et al. (2008), Cunniffe et al. (2022)), fungi-

cide application (Castle & Gilligan (2012)) or aspects of individual behaviour (Ajbar et al. (2021), Hadeler & Castillo-Chavez (1995)) have also identified such bistable regions. In our case, changing the rate of horizontal transmission ($\beta = \delta_\beta \beta$ for the tolerant parameterisation) induced this bistability, and whether the system went to a disease-free or disease-endemic equilibrium depended on the initial proportion of infectious fields and initial proportion of controllers (Figure 3.5(B)). Increasing the cost of control reduced the size of the region in parameter space in which bistability was observed (Figure 3.7(A)). If the cost of control is sufficiently high such that tolerant crop is never more profitable than unimproved crop, the bistable region is eliminated. Bistability was not observed when the improved crop had the default resistant parameterisation (Figs. 3.4(A) and 3.7(B)). Thus, the use of tolerant crop may lead to less predictable outcomes at lower values of $\delta_\beta \beta$, as having a $R_0 < 1$ is no longer sufficient to prevent disease spread.

When growers' behaviour was introduced, an "all control" equilibrium, where all growers used improved crop, was attained at low rates of horizontal transmission for the tolerant parameterisation (Figure 3.6(B)). As seen for the fixed proportions, however, this was accompanied by a higher number of infectious fields and correspondingly low expected profit for non-controllers (Figure 3.6(A)). It is these low expected profits for non-controllers that drive the higher participation in control, as the profits for those using tolerant crop remain high irrespective of the infection pressure. The benefits of using tolerant crop are felt privately by those using it, generating negative externalities

for others.

Conversely, such an "all control" equilibrium was not achieved when the improved crop was resistant, since it generates positive externalities for non-controllers (Figs. 3.4(A) and 3.6(B)). This is a product of both how the model was set up and the nature of the externalities produced. As the lowest possible payoff was achieved by growers of resistant crop that did not rogue their fields (P_{IC_H}), any of these growers should always have a non-zero probability of switching strategy ($z_{IC_H} > 0$). Thus, there will always be non-controllers at equilibrium. Additionally, the reduced probability of infection meant that the need to control was reduced, disincentivising growers from switching to the costly control strategy. This conflict between private and social benefits is often observed in epi-economic models and is theorised to be the reason why many vaccination schemes fail to achieve a socially optimal level of vaccination (Brito et al. (1991), Geoffard & Philipson (1997), Sadique (2006)).

For growers who choose to use improved crop, tolerant varieties generally give better outcomes than resistant ones (except in cases where infection is unlikely enough that the probability of incurring the loss due to disease is low) (Figure 3.8(B)). This will generate lower payoffs for non-controllers, who earn higher profits when there are more resistant fields (Figure 3.8(A)). In our model, there was widespread use of control when the tolerant crop was effective at reducing yield loss and not so costly as to discourage control when the probability of infection is low. However, this came at the cost of increasing the level of infection in the system and reducing the profits for non-controllers.

Interestingly, despite the positive feedback loop induced by the use of tolerant crop, a mixed "unimproved and tolerant crop" equilibrium was possible. In this equilibrium, even though the growers of tolerant crop should all switch strategy ($P_U > P_{SC,EC,ICR,ICH}$), the growers of unimproved crop with infectious fields should also switch strategy ($P_C > P_{IUH,IUR}$). This corresponds to the first combination of switching terms outlined in Appendix B.1 ($z_{SC}, z_{EC}, z_{ICR}, z_{ICH}, z_{IUR}, z_{IUH} > 0$). Altering the behavioural rule – for example, to one of the "strategy vs" rules based on rational expectations in Chapter 2 - could change this outcome and prevent such a mixed equilibrium.

Several simplifying assumptions were made during this investigation. Our model was deterministic and did not account for spatial effects, both of which can influence epidemic outcomes. In our behavioural model, we assumed that all growers would have access to the same information regarding disease pressure and the expected profits. In reality, a grower's knowledge of these quantities will be highly dependent on their communication network (Milne et al. (2016)), their trust in expert knowledge (Sherman & Gent (2014)), their experience with previous outbreaks (Garcia-Figuera et al. (2021)) *etc.*. How growers react to differences in profit (represented by our parameter η) will also vary between individual growers, and will impact long-term outcomes (Chapter 2). Growers must balance these information sources with market demands to make their decisions regarding disease control, and become what Kaup terms the "reflexive producer" (Kaup (2008)).

Overall, this study has shown that tolerant and resistant varieties of crop

have different effects on disease outcomes, and provide benefits to different groups of growers (controllers *vs* non-controllers). In particular, even when resistant crop was available, disease was never eliminated from the system (even though it was theoretically possible) as too few growers chose to use the resistant variety. This is an important consideration as previous studies have found optimal cropping ratios for different sets of conditions (e.g. Ohtsuki & Sasaki (2006)); in reality, the strategic decision-making of growers, as well as other factors such as their access to information or risk aversion, may mean that these ratios are never attained. Accounting for these behaviours can help improve future models of control uptake and in turn our understanding of how plant diseases spread.

Chapter 4: Expanding the strategy set: modelling grower behaviour when there are three control options

4.1 Introduction

4.1.1 Information from the previous chapters

In the models presented thus far, growers have faced a binary choice of whether to control for disease. In Chapter 2, growers could either use the clean seed system or re-plant cassava cuttings that were potentially infected with cassava brown streak disease. In Chapter 3, they either used improved crop (which could be tolerant or resistant to tomato yellow leaf curl virus, TYLCV) or conventional crop. Such a narrow choice is unlikely, and in practice growers will instead have a choice of a range of improved varieties or control options with different characteristics efficacies.

In this Chapter, we adapt the game-theoretic models described in Chapter 2 to allow growers an expanded strategy set. The model is to go beyond the previous choice between "control" and "not control" to allow growers to choose between three crop types: two control options, disease-resistant or disease-tolerant crop, or unimproved crop (for other examples of the broad approach see Milne et al. (2016), McQuaid et al. (2017a) and Saikai et al. (2021)). Their decision on which crop type to use will depend on their estimation of how much profit they expect to earn over the next growing season with each crop type, which in turn depends on the prevalence of infection and the proportion of other growers using each control strategy. The models are again based on "strategic adaptive" expectations (Fenichel & Wang (2013)), where growers balance their knowledge of the current state of the system with their experience from the previous season. In particular, growers compare their profit from the previous season with the expected profit over the next season for each of the strategies that they did not use (the "strategy vs alternative" models described in Chapter 3). This allows growers to balance sources of information, acting as "reflexive producers" (Kaup (2008)).

4.1.2 Trade-offs in disease management

We now also consider how we may incentivise growers to act in a particular way by altering the costs of each improved crop type. In reality, those tasked with managing epidemics typically have limited resources which they must distribute efficiently. This often incurs a trade-off that is experienced by multiple

parties with conflicting objectives, and how these trade-offs are managed will depend on the relative priority of each objective. A well-known example of such a trade-off for disease management occurs during vaccination campaigns (e.g. Gavish & Katriel (2022), Yousefpour et al. (2020)). Members of the public often wish to minimise their infection risk, whilst governments may want to optimise the allocation of vaccines to protect the most vulnerable (Gavish & Katriel (2022)). Similarly, in the context of plant epidemics, growers faced with crop disease will want to maximise profits, but with this comes potential environmental damage (Zaffaroni & Bevacqua (2022)) or reduced profitability when there is no risk of infection (Vyska et al. (2016)). The control mechanism adopted by growers may also have negative consequences for others; Grogan & Goodhue (2012) found that when growers sprayed pesticides within their non-citrus fields, the pesticides killed parasitic wasps that are the natural enemy of pests in neighbouring citrus fields. This intensified the reliance of citrus growers on pesticides, increasing their expenditure and environmental impact.

4.1.3 Social Planners

In economics, a social planner is a decision-maker whose goal is to balance the trade-offs that result from a policy action, aiming to maximise the welfare across all parties (Sugden (2013)). Originally conceptualised for welfare economics, the social planner's problem has been used in a range of settings, such as infectious disease management (Gersovitz & Hammer (2005), Toxvaerd (2019), Giannitsarou et al. (2021), Toxvaerd & Rowthorn (2022)), wildlife con-

servation (Johannesen & Skonhoft (2005)) and supply chain management (Lee & Choi (2021)). A key aspect of the social planner is that they account for the externalities associated with certain actions. In economics, externalities are the consequences of an action that are felt by a third party not involved in the action (Gersovitz (2014)). These externalities are often ignored by individuals, who only care about their own outcome, but they are important when considering what is socially optimal.

An alternative approach to this model would be to set it up as a Stackelberg game. In these games, there is a "leader" and a "follower"; the leader takes action knowing that they are being observed by the follower, who will then take action themselves. The leader, knowing that they are being observed, must know how the followers will respond, and take an action that will maximise their payoff. In our case, the leader would be the social planner, who would set the price of the subsidies pre-supposing the response of the growers. If we were to arrange our growers and planners into a hierarchy, the Stackelberg game would be transformed into a Bi-Level Optimisation Problem (Vicente & Calamai (1994)).

4.1.4 Pareto optimaility

The social planner's problem can be solved using Pareto optimality. Under a Pareto-optimal solution, we cannot improve one party's objective without ensuring a worse outcome for another party (Luc (2008)). These solutions are efficient, as they optimise for each outcome, and non-dominated, as, given the

same constraints, they cannot be improved upon by any other solution. There may be many possible solutions for a given problem, which combine to give a Pareto front. Pareto fronts have been calculated to visualise the trade-offs involved in the recent coronavirus pandemic (Yousefpour et al. (2020)), to retrospectively evaluate responses to the 1918 influenza pandemic (Velde (2022)) and to better understand how to optimally allocate healthcare equipment during an epidemic (Donmez et al. (2022)). In plant disease modelling, they have been used to investigate the trade-offs between crop productivity and environmental impacts (Zaffaroni & Bevacqua (2022)). None of the solutions in the Pareto front is inherently better than any other, so the decision-maker must decide which option to use based on additional information.

In Chapter 2, we showed that tolerant and resistant crops caused contrasting outcomes for non-controllers. Planting tolerant crop was often more beneficial for controllers than resistant crop, as it limited the yield loss in the fields of controlling growers if they became infected. However, their reduced symptom expression meant that tolerant crops that did become infected were less likely to be detected, reducing the effectiveness of roguing as a disease control strategy. As tolerant varieties do not restrict pathogen replication, this leads to a build-up of inoculum and high infection levels. This lowered yields for all non-controllers. This directly incentivised use of the improved crop, as growers could expect to earn higher profits when using a tolerant variety than an unimproved variety.

When some growers used improved crop, this also benefited other growers

(Chapter 2). As resistant crops are less susceptible to infection, and limit pathogen replication once infected, the overall disease pressure on other fields is reduced. This increases the profits of non-controllers, as they can gain some of the benefits of resistant varieties without themselves paying the cost of the improved variety (i.e. can *free-ride* off controllers). The consequences of each crop type - the increase and decrease in infection pressure and the subsequent effect on profits of non-controllers - can therefore be considered *externalities* of each control decision. The impact of these externalities on growers' disease management practices when growers can choose between three crop types - unimproved, tolerant or resistant - is unknown.

4.1.5 Subsidies and plant disease management

We consider the perspective of a social planner who has the ability to subsidise these crop types. Historically, the European Union has subsidised the cultivation of processing tomato varieties (Sumner et al. (2001)); here, we study subsidies as a means of promoting particular epidemic outcomes. Subsidies are a means of rewarding/penalising a grower based on the external effects of their actions (i.e. internalising the externalities generated by using a particular practice) (Pretty et al. (2001)). They have a long history in agriculture, from regulating food production to promoting more environmentally-friendly practices such as hedgerow conservation in the UK (Stokstad (2020)) or reduced pesticide use in France (Aubert & Enjolras (2022)). They also have been used to encourage the use of particular crop varieties or control mechanisms (Car-

riere et al. (2020), Holden & Fisher (2015), Zhao et al. (2022), Okechukwu & Kumar (2016)). Here we consider two subsidies: one for resistant crop and one for tolerant crop, either of which is provided to the growers by a "social planner" or centralised body. As, in our model, growers choose between different crop varieties based on their expected profitability, subsidising each crop type incentivises its use. The planner has two objectives: to maximise the average profit across growers and to minimise their own spending on subsidies. We study the options available to the social planner using Pareto optimality.

4.1.6 Aims of this chapter

The work, therefore, addresses three primary questions: 1) What are the long-term outcomes when growers have access to three different crop varieties? 2) How do the responses depend on the efficacy of tolerant and resistant traits? 3) Which subsidisation strategy promotes socially-optimal solutions?

4.2 Methods

4.2.1 Epidemic model with three crop varieties

The following model extends the model detailed in Chapter 3. It describes the spread of *Tomato Yellow Leaf Curl Virus* (TYLCV) through a population of fields, each cultivated by a single grower. The fields can be planted with one of three crop varieties: unimproved crop (U), crop that is tolerant to TYLCV (T) or crop that is resistant to TYLCV (R). We also track each field's infection status; as we do not model within-field spread, fields can either

be susceptible to infection (S), latently infected (E) or infectious (I). The parameterisation of this model is identical to that presented in Chapter 3, with a summary of the parameter values presented in Tables 4.1 and 4.2. State variables are scaled to be a proportion of the total number of fields (i.e. $N = S_U + E_U + I_U + S_T + E_T + I_T + S_R + E_R + I_R = 1$).

Irrespective of the crop type planted in the field, and unless the crop in a field is rogued (see below), the season length ($1/\gamma$) is 120 days (Holt et al. (1999*a*), Rocco & Morabito (2016)). Replanting of any field occurs immediately after it is harvested. Susceptible fields become infected at rate βI day^{-1}, where I is the proportion of infectious fields. Resistant crop, however, reduces the probability of infection per unit time by a factor $\delta_{\beta_R} < 1$. As tolerant crop does not reduce the probability of infection, we presume that $\delta_{\beta_T} = 1$. If fields are infected, they enter the "exposed" compartment where they remain latently infected (i.e. infected but not infectious) for an average of $1/\epsilon$ days . During this time, we assume that they show no symptoms of infection and cannot infect other fields. We assume that resistant crops that become infected have a reduced rate of symptom development, increasing this latent period by a factor of $\delta_{\epsilon_R} < 1$ (ensuring that $\delta_{\epsilon_R}/\epsilon < 1/\epsilon$).

After becoming fully infectious and symptomatic, the ultimate fate of any field is to be either harvested or rogued. Roguing involves surveying fields ("scouting") and removing any visibly-infected plants, and has been used as a means of TYLCV management for decades (Ioannu (1987), Polston et al. (1999), Polston & Lapidot (2007), Ddamulira et al. (2021)). As tomatoes can

be harvested at different levels of maturity and ripen off-vine (Arah et al. (2015)), if there is infection in a field, growers may be better to harvest the entire field to prevent infection spreading. This will reduce the infection-associated yield loss by a factor ϕ_Q such that the losses experienced by a grower who has rogued an infectious field are $\phi_Q L$). We assume scouting occurs at time intervals of Δ days and symptoms are detected with probability ν. Infectious, symptomatic fields (class I_b) are removed as soon as they are detected. As tolerant crops reduce symptom severity, we presume that they have a lower probability of detection (reduced by multiplication by a factor of $\delta_{\nu_T} < 1$) compared to unimproved and resistant crop (where $\delta_{\nu_R} = 1$).

The removal rates of fields, μ_b with $b \in \{U, T, R\}$, which represent such a program of roguing, are given by Cunniffe et al. (2014):

$$\mu_U = \frac{1}{\left(\frac{1}{\nu} - \frac{1}{2}\right)\Delta}, \tag{4.1}$$

$$\mu_T = \frac{1}{\left(\frac{1}{\delta_{\nu_T}\nu} - \frac{1}{2}\right)\Delta}, \tag{4.2}$$

$$\mu_R = \frac{1}{\left(\frac{1}{\delta_{\nu_R}\nu} - \frac{1}{2}\right)\Delta}. \tag{4.3}$$

The epidemiological model is then given by:

$$\frac{dS_U}{dt} = \gamma\theta_U + M_U - \beta S_U(I_U + \delta_{\sigma_T} I_T + \delta_{\sigma_R} I_R) - \gamma S_U, \tag{4.4}$$

$$\frac{dE_U}{dt} = \beta S_U(I_U + \delta_{\sigma_T} I_T + \delta_{\sigma_R} I_R) - \epsilon E_U - \gamma E_U, \tag{4.5}$$

$$\frac{dI_U}{dt} = \epsilon E_U - \mu_U I_U - \gamma I_U, \tag{4.6}$$

$$\frac{dS_T}{dt} = \gamma\theta_T + M_T - \delta_{\beta_T}\beta S_T(I_U + \delta_{\sigma_T} I_T + \delta_{\sigma_R} I_R) - \gamma S_T, \tag{4.7}$$

$$\frac{dE_T}{dt} = \delta_{\beta_T}\beta S_T(I_U + \delta_{\sigma_T} I_T + \delta_{\sigma_R} I_R) - \delta_{\epsilon_T}\epsilon E_T - \gamma E_T, \tag{4.8}$$

$$\frac{dI_T}{dt} = \delta_{\epsilon_T}\epsilon E_T - \mu_T I_T - \gamma I_T, \tag{4.9}$$

$$\frac{dS_T}{dt} = \gamma\theta_R + M_R - \delta_{\beta_R}\beta S_R(I_U + \delta_{\sigma_T} I_T + \delta_{\sigma_R} I_R) - \gamma S_R, \tag{4.10}$$

$$\frac{dE_R}{dt} = \beta S_R(I_U + \delta_{\sigma_T} I_T + \delta_{\sigma_R} I_R) - \delta_{\epsilon_R}\epsilon E_R - \gamma E_R, \tag{4.11}$$

$$\frac{dI_R}{dt} = \delta_{\epsilon_R}\epsilon E_R - \mu_R I_R - \gamma I_R. \tag{4.12}$$

where terms $\gamma\theta_U$, $\gamma\theta_T$ and $\gamma\theta_R$ are the rates of replanting for harvested fields,

whilst M_U, M_R and M_T are rates of replanting for rogued fields:

$$\theta_U = (1 - z_{SU})S_U + (1 - z_{EU})E_U + (1 - z_{I_HU})I_U + z_{STU}S_T + z_{ETU}E_T + z_{I_HTU}I_T +$$
$$z_{SRU}S_R + z_{ERU}E_R + z_{I_HRU}I_R, \tag{4.13}$$
$$\tag{4.14}$$
$$\theta_T = (1 - z_{ST})S_T + (1 - z_{ET})E_T + (1 - z_{I_HT})I_T + z_{SRT}S_R + z_{ERT}E_R + z_{I_HRT}I_R +$$
$$z_{SUT}S_U + z_{EUT}E_U + z_{I_HUT}I_U, \tag{4.15}$$
$$\tag{4.16}$$
$$\theta_R = (1 - z_{SR})S_R + (1 - z_{ER})E_R + (1 - z_{I_HR})I_R + z_{STR}S_T + z_{ETR}E_T + z_{I_HTR}I_T +$$
$$z_{SUR}S_U + z_{EUR}E_U + z_{I_HUR}I_U, \tag{4.17}$$
$$\tag{4.18}$$
$$M_U = (1 - z_{I_RU})\mu_U I_U + z_{I_RTU}\mu_T I_T + z_{I_RRU}\mu_R I_R, \tag{4.19}$$
$$\tag{4.20}$$
$$M_T = z_{I_RUT}\mu_U I_U + (1 - z_{I_RT})\mu_T I_T + z_{I_RRT}\mu_R I_R, \tag{4.21}$$
$$\tag{4.22}$$
$$M_R = z_{I_RUR}\mu_U I_U + z_{I_RTR}\mu_T I_T + (1 - z_{I_RR})\mu_R I_R. \tag{4.23}$$

Note, a distinction is made between the replanting of rogued (I_R) and harvested (I_H) crop. For rogued unimproved fields, $z_{I_RU}\mu_U I_U = z_{I_RUT}\mu_U I_U + z_{I_RUR}\mu_U I_U$ (i.e. the total rate at which rogued unimproved fields switch strategy must be equal to the rate at which they enter either other strategy, with equivalent expressions for tolerant and resistant crop). Similarly, for harvested

unimproved fields, $z_{I_HU}I_U = z_{I_HUT}I_U + z_{I_HUR}I_U$. The exact forms of the *switching terms* (Chapter 2) is described below, including the meaning of the different number of subscripts (Equation 4.36).

Replanting in our model is represented by switching terms (Milne et al. (2016), McQuaid et al. (2017a) and Chapter 2) which are the terms of the form z_{ab} or z_{abc} where $a \in \{S, E, I_H, I_R\}$ and $b, c \in \{U, T, R\}$ (note the additional complexity of our model means there is a change of notation compared to Chapters 2 and 3). The switching terms with three subscripts, z_{abc}, represent the probability of a grower changing strategy from their current strategy b to strategy c based on their outcome, a, in the previous season (note that $b \neq c$). For growers whose fields were susceptible (S) or exposed (E), the outcome depends solely on the epidemiological class of their field at the end of the season, whereas for growers whose fields were infectious, it depends also on whether that infectious field was rogued (subscript R) or harvested (subscript H). The two-subscript version, z_{ab}, is the probability that a grower who harvested field type a_b switches strategy. The forms of these terms are explained in Section 2.4 (below). A schematic of the model is shown in Figure 4.1.

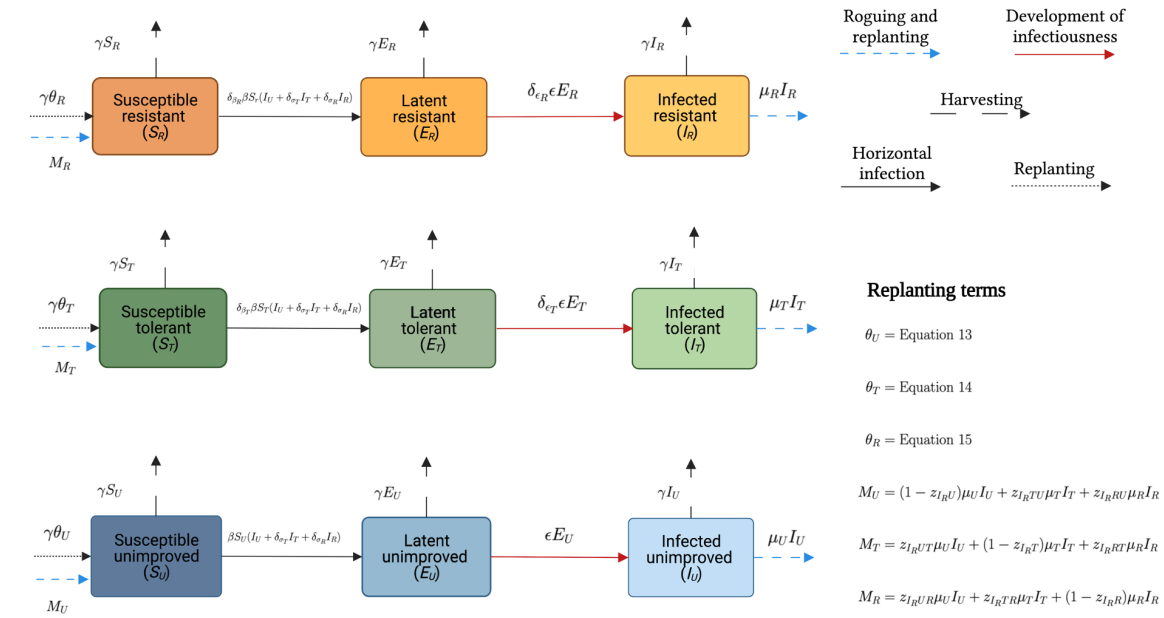

Figure 4.1: **Schematic showing the structure of the model when growers can choose their strategy based on expected profits.** We have three classes of growers; those who use unimproved seed (subscript U), those who use tolerant seed (subscript T), and those that use resistant seed (subscript R). The terms θ_U, θ_T and θ_R are the rates of replanting for harvested fields, whilst M_U, M_R and M_T are rates of replanting for rogued fields (Equations 4.13 - 4.23, with $M_U + M_R + M_T = \mu_U I_U + \mu_R I_R + \mu_T I_T$). Created with BioRender.com

Table 4.1: Parameters related to resistant and tolerant varieties. The distinction between the two varieties lies in how disease is transmitted and what losses are incurred when a field is infectious. Here, b is the strategy of the grower and $b \in \{U, T, R\}$

.

Parameter	Meaning	Value if unimproved	Value if resistant	Value if tolerant
δ_{β_b}	Relative susceptibility	1	0.5	1
δ_{σ_b}	Relative infectivity	1	0.5	1
δ_{Y_b}	Relative yield	1	1	1
δ_{L_b}	Relative losses due to disease	1	1	0.1
$\frac{1}{\delta_{\epsilon_b}}$	Relative latent period	1	0.5	1
δ_{ν_b}	Relative probability of detection	1	1	0.1

Table 4.2: **Summary of parameter values.**

Parameter	Meaning	Value	Reference
$1/\gamma$	Length of the growing season	120 days	Holt et al. (1999*a*); Rocco & Morabito (2016)
β	Rate of secondary infection	0.055/N day^{-1} field^{-1}	See main text
Δ	Time between roguing	120 days	Illustrative
ν	Probability of detection	1	Illustrative
μ_U	Removal rate (unimproved)	1/60 day^{-1}	Illustrative
μ_R	Removal rate (resistant)	1/60 day^{-1}	Illustrative
μ_T	Removal rate (tolerant)	1/1140 day^{-1}	Illustrative
$\frac{1}{\epsilon}$	Average latent period	41 days	Holt et al. (1999*b*); Ber et al. (1990)
η	Responsiveness of growers	10	Chapter 2
Y	Maximum yield	1	All values scaled relative to yield
L	Loss due to infection	0.6	Riley & Srinivasan (2019)
ϕ_T	Cost of tolerant crop	0.1	Fonsah et al. (2018)
ϕ_R	Cost of resistant crop	0.1	Fonsah et al. (2018)
ϕ_Q	Relative reduction in loss due to roguing	0.7	Illustrative
N	Total number of fields/growers	1	Scaled to 1

4.2.2 Growers' profits

The profit a grower earns is based on the control strategy that they previously used and their infection status at the time of harvest. Each crop type ($b \in \{U, T, R\}$) has specific associated costs and yield losses when infected with TYLCV. We note that, in the absence of disease, we assume that all varieties have the same maximum yield, Y.

$$P_{Sb} = \text{Profit for a susceptible field of type } b = Y - \phi_b, \tag{4.24}$$

$$P_{Eb} = \text{Profit for a latently-infected field of type } b = Y - \phi_b, \tag{4.25}$$

$$P_{I_H b} = \text{Profit for an infected field that was not rogued of type } b = Y - \phi_b - \delta_{L_b} L, \tag{4.26}$$

$$P_{I_R b} = \text{Profit an infected field that was rogued of type } b = Y - \phi_b - \phi_Q \delta_{L_b} L, . \tag{4.27}$$

Notably, here, $\phi_U = 0$, since ϕ_b represents the additional cost of using crop type b. For a full enumeration of the outcomes for growers, see Appendix C.1.

For our parameterisation, we assume that latently-infected fields do not lose any yield, so $P_{Sb} = P_{Eb}, b \in \{U, T, R\}$. As growers who use unimproved crop and harvest susceptible or latently-infected fields pay no cost of control and sustain no yield loss, $P_{SU} = P_{EU}$ is always the maximum achievable profit. The relative sizes of the remaining profits will depend on the values of the costs of the improved varieties (ϕ_T and ϕ_R), the infection-induced yield losses ($\delta_{L_L} L$, $\delta_{L_T} L$ and $\delta_{L_R} L$) and the benefit of roguing (ϕ_Q). Under the default parameterisation (Table 4.2), the ordering of the profits is given by:

$$P_{SU} = P_{EU} > P_{ST} = P_{ET} = P_{SR} = P_{ER} > P_{I_R T} > P_{I_H T} > P_{I_R U} > P_{I_H U} > P_{I_R R} > P_{I_H R}. \tag{4.28}$$

4.2.3 Calculating expected profits

The growers' decision of which crop variety to use will depend on how each individual grower's profit from the previous season compares to the expected profit of the alternative crop types. These expected profits are based on the probability of attaining each outcome outlined in Equations 4.24-4.26, which in turn depends on the probability of infection for each crop type. The complete derivation is provided in Appendix C.1; the simplified expressions for the expected profits for unimproved (P_U), tolerant (P_T) and resistant (P_R) are:

$$P_U = Y - q_U \frac{\epsilon}{\epsilon + \gamma} L \left(\frac{\gamma}{\gamma + \mu_U} + \frac{\mu_U}{\mu_U + \gamma} \phi_Q \right), \tag{4.29}$$

$$P_T = \delta_{Y_T} Y - \phi_T - q_T \frac{\delta_{\epsilon_T} \epsilon}{\delta_{\epsilon_T} \epsilon + \gamma} \delta_{L_T} L \left(\frac{\gamma}{\gamma + \mu_T} + \frac{\mu_T}{\mu_T + \gamma} \phi_Q \right), \tag{4.30}$$

$$P_R = \delta_{Y_R} Y - \phi_R - q_R \frac{\delta_{\epsilon_R} \epsilon}{\delta_{\epsilon_R} \epsilon + \gamma} \delta_{L_R} L \left(\frac{\gamma}{\gamma + \mu_R} + \frac{\mu_R}{\mu_R + \gamma} \phi_Q \right). \tag{4.31}$$

4.2.4 Switching terms

The switching terms (Chapter 2) affect the rate at which growers move from their current strategy to one of the alternative strategies.

In previous models (Milne et al. (2016), and McQuaid et al. (2017a), Saikai et al. (2021)), growers only had one alternative strategy with which to compare profits. Now, growers must consider two alternatives to their current strategy. Growers first assess the expected profits of the two alternative strategies and only compare their outcome with the highest expected profit of the two alternatives. If both alternatives have the same expected profit (so, for example,

if the expected payoff for tolerance and resistance are the same ($P_T = P_R$)), and therefore the grower should have the same probability of switching into each strategy, growers chose to compare with the profit associated with the crop type most growers use. This follows from "descriptive norms", where individuals follow what the majority of other people are doing (Cialdini et al. (1990), Sinclair & Agerström (2021), Lazić et al. (2021)). Additionally, there is some evidence that growers will be more likely to participate in control if other growers also participate (Milne et al. (2018)). If both the expected profits and the proportion of growers using each strategy are the same, then half of the growers changing strategy will go to each alternative.

In their general form, the switching terms have the following structure, where a grower with outcome P_{ab}, contemplates switching into strategy c or d, $a \in \{S, E, I_H, I_R\}$ and $b, c, d \in \{U, T, R\}, b \neq c \neq d$.

If $P_c > P_d$ or $P_c = P_d$ and $C > D$ (where C and D are the proportion using strategies c and d), then:

$$z_{abc} = \max(0, 1 - \exp(-\eta(P_c - P_{ab})), \tag{4.32}$$

$$z_{abd} = 0. \tag{4.33}$$

In the case where both the profits and the proportion of growers using the alternative strategy are equal, growers changing strategy will be divided evenly

between the two alternative strategies. So, if $P_c = P_d$ and $C = D$, then:

$$z_{abc} = \frac{\max(0, 1 - \exp(-\eta(P_c - P_{ab}))}{2}, \tag{4.34}$$

$$z_{abd} = \frac{\max(0, 1 - \exp(-\eta(P_c - P_{ab}))}{2}. \tag{4.35}$$

To simplify writing the model, we can then say:

$$z_{ab} = \max(z_{abc}, z_{abd}). \tag{4.36}$$

where z_{ab} is the probability that a grower who harvested field of type a_b leaves strategy b to adopt either strategy c or d.

The full detail of all switching terms is provided in Appendix C.2.

4.2.5 Calculating the Pareto front and Gini coefficients

We use the concept of Pareto efficiency to dually optimise two conflicting objectives. Our first objective is to maximise the average profit of growers at equilibrium, calculated as the yield they achieve less any investment they have made to control for disease (based on the outcomes outlined in Equations 4.24 - 4.27). Our second objective is to minimise the spending of a central planning body which subsidises the cost of tolerant and resistant crops. These subsidies reduce the cost of improved crop every time that it is planted.

If the cost to the planner of the crop type is $\phi_{T,max}$, and the cost paid by growers is ϕ_T then the subsidy to growers for the use of tolerant crop

$\sigma_T = \phi_{T,max} - \phi_T$ (and, equivalently, for resistant crop is $\sigma_R = \phi_{R,max} - \phi_R$). The total cost to the planner is then

$$\tau = \sigma_T T + \sigma_R R \tag{4.37}$$

where $T = S_T + E_T + I_T$ and $R = S_R + E_R + I_R$ (i.e. are the proportions of growers using tolerant and resistant crop, respectively). We use Pareto optimality to establish the optimal allocation of subsidies between tolerant and resistant crop.

We first run the model for different values of ϕ_T and ϕ_R (400 values between 0 and 0.4 for each parameter) to find the equilibrium profit of growers and cost to the planner. We then use these two results as inputs to $get\_frontier()$ function from the KraljicMatrix package in R (Boehmke et al. (2017)), which calculates the Pareto front.

Though the Pareto front shows the optimal outcomes for a given set of parameters, one objective may be more heavily prioritised over the other. To quantify the degree of fairness between outcomes, we calculate the Gini coefficient (Equation 4.38). Originally developed as a measure of income inequality (Gini (1936)), the Gini coefficient calculates the extent to which a solution deviates from total equality between objectives. In that sense, the Gini coefficient is related to fairness but does not consider the optimality of the solution: a scenario with a low Gini coefficient (indicating a high degree of fairness between objectives, with a value of zero indicating perfect equality) may be a

suboptimal solution for one or both objectives. Similarly, in some scenarios, the Gini coefficient may be small due to the number of objectives being considered: if just one individual's objective is perfectly optimised, and all others are ignored, then $G = 1 - 1/n$, where n is the number of objectives. The coefficient has been used to estimate regional inequalities in the risk of veterinary epidemics (Li et al. (2020)), geographical variability in STD incidence (Elliott et al. (2002)) and in ecology to evaluate management programmes and agricultural productivity (Li et al. (2017), Zaffaroni & Bevacqua (2022)). The Gini coefficient for a given scenario i is calculated by (Dorfman (1979), Zaffaroni & Bevacqua (2022)):

$$G_i = \frac{\sum_{j=1}^{n} \sum_{z=1}^{n} |x_{j,i} - x_{z,i}|}{2n \sum_{j=1}^{n} x_{j,i}} \tag{4.38}$$

where n is the number of objectives considered and $x_{j,i}$ and $x_{z,i}$ are the values of the objectives for scenario i.

Calculating the Gini coefficient requires both objectives to be measured on the same scale, so we must aim to maximise or minimise both objectives. Additionally, to simplify the optimization problem, we must ensure that both the cost to the planner and profit of the grower is scaled between 0 and 1. We therefore calculate the relative cost to the planner, τ^* as:

$$\tau^* = \frac{\max(\tau) - \tau_j}{\max(\tau) - \min(\tau)} \tag{4.39}$$

where $\max(\tau)$ and $\min(\tau)$ are the maximum and minimum costs to the planner

that lie along the Pareto front and τ_j is the cost value for a combination of ϕ_R and ϕ_T. This scales the cost to the planner between 0 and 1. Maximising this relative cost, τ^*, is equivalent to minimising the actual cost, τ.

Similarly, we must also rescale the profits so that they are normalised between 0 and 1:

$$P^* = \frac{\text{max(Profit) - Profit}_j}{\text{max(Profit) - min(Profit)}} \tag{4.40}$$

where max(Profit) and min(Profit) are the maximum and minimum profits to growers that lie along the Pareto front, Profit_j is the objective value for a particular combination of ϕ_R and ϕ_T.

We use the Gini coefficient to evaluate how a particular subsidy scheme benefits the planner relative to the growers, giving a measure of the fairness of each scheme. Since we have only two objectives, the Gini coefficient will be between 0 and 0.5.

4.3 Results

4.3.1 Equilibria for the three-strategy model.

Using the next-generation method (NGM, Diekmann et al. (2010); Appendix C.3) we find R_0 for the model to be:

$$R_0 = \frac{\beta\epsilon N}{(\epsilon+\gamma)(\mu_U+\gamma)}. \tag{4.41}$$

At the disease-free equilibrium, only unimproved crop is used by growers. The absence of disease makes control obsolete, so no growers should pay the cost

of control. In Equation 4.41 $\frac{\epsilon}{(\epsilon+\gamma)}$ is the probability that an unimproved field will become infectious before it is harvested and $\frac{1}{\mu_U+\gamma}$ is the mean time in the I_U compartment. The number of infections caused by these infectious fields is βN.

There are eight possible long-term outcomes for the model:

- **Disease-free equilibrium (DFE):** where $R_0 < 1$ and no growers control for disease.
- **"No control" equilibrium:** where disease is endemic but no growers use improved crop ($N = U$).
- **"All tolerant" equilibrium:** where disease is endemic and growers only use tolerant crop ($N = T$).
- **"All resistant" equilibrium:** where disease is endemic and growers only use resistant crop ($N = R$).
- **Three-strategy equilibrium:** where disease is endemic and crops of all three varieties are in use ($N = U + T + R$).
- **"Tolerant and unimproved" equilibrium:** where disease is endemic and growers use either tolerant or unimproved crop ($N = U + T$).
- **"Resistant and unimproved" equilibrium:** where disease is endemic and growers use either tolerant or unimproved crop ($N = U + R$).
- **"Tolerant and resistant" equilibrium:** where disease is endemic and growers use either tolerant or resistant crop ($N = T + R$).

Additionally, the model presented in Chapter 3 allowed for a bistable region when $R_0 < 1$. To prevent this from affecting our results, we always begin the models with the initial conditions outlined in Table 4.3, which guarantees a disease-free equilibrium in the bistable region for the default parameterisation.

Table 4.3: **Default initial conditions.**

Variable	Meaning	Value
$S_U(0)$	Initial proportion of susceptible unimproved fields	0.79
$E_U(0)$	Initial proportion of latently infected unimproved fields	0
$I_U(0)$	Initial proportion of infectious unimproved fields	0.01
$S_T(0)$	Initial proportion of susceptible tolerant fields	0.1
$E_T(0)$	Initial proportion of latently infected tolerant fields	0
$I_T(0)$	Initial proportion of infectious tolerant fields	0
$S_R(0)$	Initial proportion of susceptible resistant fields	0.1
$E_R(0)$	Initial proportion of latently infected resistant fields	0
$I_R(0)$	Initial proportion of infectious resistant fields	0

Parameterisations that lead to the disease-free, all-tolerant, tolerant and resistant, unimproved and resistant, unimproved and tolerant, and three-strategy equilibria are shown in Figure 4.2, in which we vary the rate of horizontal transmission (β) and in (A) the cost of both improved crops (ϕ_R and ϕ_T) and in (B) just ϕ_T (ϕ_R is fixed at 0.06 for demonstrative purposes). However, the externalities generated by both tolerant and resistant crop mean that some of these potential equilibria are only realised within a narrow range of parameter

values (namely the "no control" equilibrium). The "all resistant" equilibrium is not possible, as those using unimproved crop can free-ride off the efforts of those who plant resistant crop (Chapter 3).

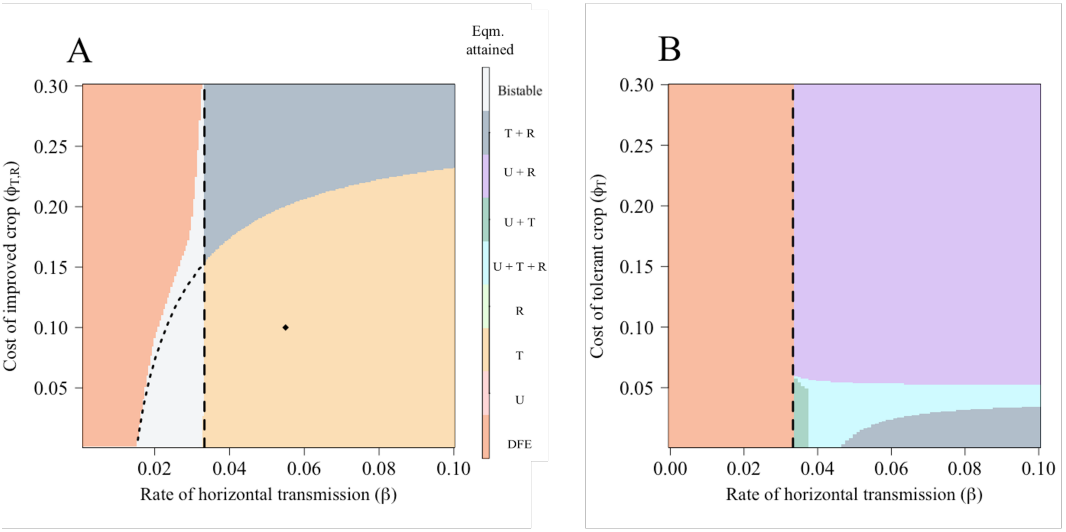

Figure 4.2: **Effect of the cost of tolerant or resistant crop and the rate of horizontal transmission on the equilibrium values.** (A) Uses the default parameterisation, whilst (B) has $\phi_R = 0.06$, $\delta_{\nu_T} = 1$ (as might be the case if disease detection was done via a genetic test), $\delta_{L_T} = 0.3$ and $\delta_{\beta_R} = 0.1$. In both cases, a disease-free equilibrium persists once $R_0 < 1$, though there is a bistable region shown in light grey between the limits of $\beta_U = 0.020$ and 0.0333 day^{-1} in (A). The equilibrium realised within this region is either a mixed tolerant and unimproved crop equilibrium or an all-tolerant equilibrium (indicated by the dotted line). In (B), lowering the costs and losses associated with resistant crop allowed for a mixed equilibrium between resistant and unimproved crop and a three-strategy mixed equilibrium between all three crop types. The dashed vertical lines show $R_0 = 1$.

Which long-term outcomes are possible depends strongly on parameter values, particularly the parameterisation of resistance and tolerance. When the cost of both improved crop varieties varied (Figure 4.2(A)), lower costs incentivised the use of tolerant crop, and as the rate of horizontal transmission (β) increased, more growers used tolerant crop. The high probability of infection meant that growers would be better off using tolerant crop and limiting the

damage due to disease (as, under this parameterisation, the losses in tolerant crop were a tenth of those for resistant or unimproved crop). As the benefits of tolerant crop are felt privately by those who use them, there is more incentive to use these varieties and an "all-tolerant" equilibrium is possible even at relatively high costs of control.

If we keep the cost of resistant crop fixed at a low price ($\phi_R = 0.06$), and parameterise it such that it is significantly less susceptible to infection ($\delta_{\beta_R} = 0.1$), its use is much more widespread (Figure 4.2(B)). At low values of ϕ_T, mixed equilibria with tolerant crop are achieved, particularly at higher values of β when infection becomes more likely to occur. However, under this parameterisation, the tolerant crop is both more easily detected via roguing ($\delta_{\nu_R} = 1$) and is less effective at limiting yield loss ($\delta_L = 0.3$), so there is less of an incentive for growers to use it compared to the parameterisation in Figure 4.2(A). As ϕ_T increases, growers stop using tolerant crop and instead a larger proportion "free-ride" off of the efforts of those that use resistant crop. As resistant crop generates positive externalities for other growers, the fields of the non-controllers have a reduced infection pressure without incurring any costs. Consequently, an "all-resistant" equilibrium is not reached.

The bistable region between $\beta = 0.02$ and 0.0333 day^{-1} in Figure 4.2(A) varies between either the DFE and "all tolerant" equilibria or the DFE and "tolerant and resistant" equilibria depending on parameter values, with the dotted line showing the distinction between these equilibria. As in Chapter 3, a high initial proportion of tolerant crops (or infectious crops) causes the

system to go to the disease-endemic equilibria.

4.3.2 Effect of parameters relating to tolerance and resistance on behaviour

The primary characteristic of tolerant crop is its ability to limit yield loss if infection occurs, so the losses experienced by growers of tolerant crop are lower than those of unimproved or resistant crop ($\delta_{L_T} L < L$). At low values of δ_{L_T} (Figure 4.3), many growers therefore use tolerant crop. As tolerant crop is less likely to be rogued, infection builds up, further incentivising growers to use tolerant crop.

As δ_{L_T} increases, the value of the expected profit of those growing tolerant crop (P_T) falls. For this parameterisation, at $\delta_{L_T} \approx 0.31$ ("X"), the profits of growers who harvested infected tolerant crop are lower than the expected profits of non-controllers ($P_{I_H T} < P_U$), so controllers that have harvested infected crop have a non-zero probability of switching strategy. As the expected profit for unimproved crop is higher than that of resistant crop ($P_U > P_R$), these growers only consider switching to unimproved crop. Similarly, at $\delta_{L_T} \approx 0.405$ ("+"), $P_{I_R T} < P_U$ and growers of tolerant crop who have rogued their fields should also consider switching to unimproved crop. These changes to the switching terms cause sharp changes in the proportion of growers using each strategy.

At the value of $\delta_{L_T} \approx 0.64$ ("O"), growers of tolerant crop who have rogued their infected fields earn less than the expected profit of those using resistant

crop ($P_{I_RT} < P_R$). These growers start switching strategy, causing a decrease in the proportion using tolerant crop and an increase in the expected profits of both tolerant and resistant crop. At $\delta_{L_T} \approx 0.64$ $P_T = P_R$: the model set-up means that growers of unimproved crop who harvested infected fields compare with the profit of whichever strategy has more growers using it, which, in this case, is tolerant crop. Once $\delta_{L_T} > 0.64$ ("O"), $P_T < P_R$ and the growers of infected unimproved crop now consider switching to resistant crop. This causes a sharp increase in those using resistant crop and decrease in proportion of infected fields (Figure 4.3 (A) at $\delta_{L_T} > 0.64$). All expected profits rise with this decrease in infection (Figure 4.3 (B)), though the relative ordering of the profits does not change ($P_U > P_R > P_T$). The response flattens out after $\delta_{L_T} > 0.64$ as no growers are using the tolerant crop, so no growers are affected by the changes in this parameter. The model will therefore run to the same equilibrium irrespective of further changes to δ_{L_T}.

Between the values of $\delta_{L_T} \approx 0.51$ and 0.71 (denoted by the black bar in Figure 4.3 (A)), there is bistability which changes the precise value of δ_{L_T} at which growers stop using tolerant crop ("O"), as a function of the initial conditions of the model (Appendix 3).

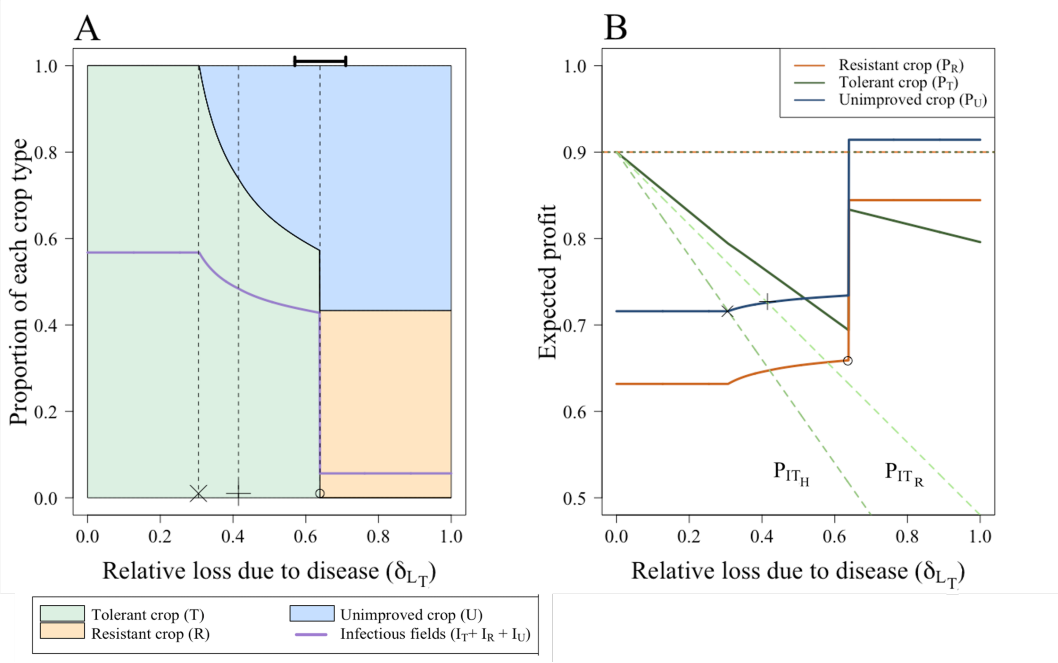

Figure 4.3: **Effect of the relative loss due to infection on the choice of unimproved, tolerant or resistant crop.** (A) The proportion of infectious fields ($I_T + I_R$), tolerant (T) and resistant (R) fields. (B) The expected profit for growers using unimproved, resistant or tolerant crop. The dashed orange and red lines show the profit for susceptible and latently-infected tolerant and resistant fields ($P_{ST} = P_{ET} = P_{SR} = P_{ER} = 0.9$), and the green dashed lines show the profits for a rogued and harvested infected tolerant field. For low values of δ_{L_T} (such that $\delta_{L_T}L << \delta_{L_R}L, \delta_{L_T}L << L$), all growers use the tolerant crop. However, once $\delta_{L_T} > 0.31$, growers who have harvested infected tolerant crop should have a non-zero probability of switching to unimproved crop. Similarly, once $\delta_{L_T} > 0.41$, growers who have rogued tolerant crop should also have a non-zero probability of switching to unimproved crop. Once $\delta_{L_T} > 0.64$ (when $P_T = P_R$), all non-controllers who harvested infected crop should consider switching to resistant crop, rather than tolerant crop. This causes a decrease in those using tolerant crop and an increase in those planting resistant, which also causes a decrease in the proportion of infected fields (A). The black bar in (A) shows the region where bistability affects the switch between equilibria. Save for δ_{L_T}, parameters and initial conditions are as in Table 4.2 and Table 4.3 respectively.

When considering the efficacy of resistant plants, only when the relative susceptibility of resistant plants (δ_{β_R}) is low is there resistant crop at equilibrium. Once the resistant crop is more susceptible to infection, growers prefer to use the tolerant crop and sustain lower losses once infection occurs. For the

parameterisation in Table 4.2, and as δ_{β_R} varies, when $\delta_{\beta_R} = 0.08$, all growers use tolerant crop ("all tolerant" equilibrium), so the responses to changes in δ_{β_R} flatten out as there are no resistant fields with reduced susceptibility. The remaining parameters are unchanged, so the model goes to the same equilibrium as parameter δ_{β_R} is varied.

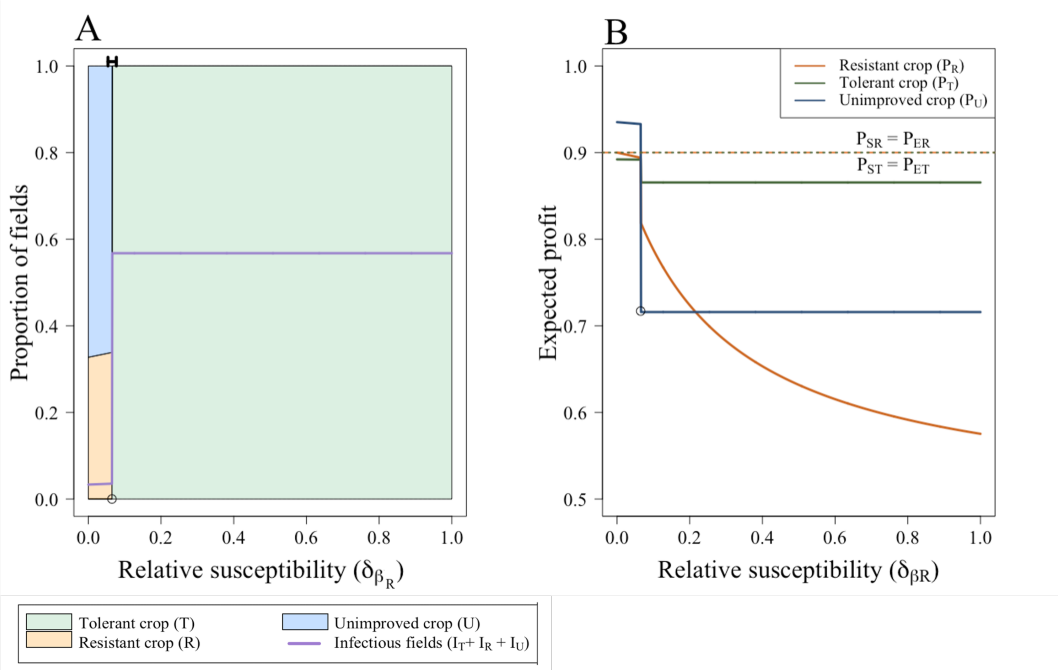

Figure 4.4: **Effect of the relative susceptibility to infection on the choice of unimproved, tolerant or resistant crop.** (A) The proportion of infectious fields ($I_T + I_R + I_U$), tolerant (T) and resistant (R) fields. (B) The expected profit for growers using unimproved, resistant or tolerant crop. The dashed orange and red lines show the profit for susceptible and latently-infected tolerant and resistant fields ($P_{ST} = P_{ET} = P_{SR} = P_{ER} = 0.9$). Most growers use the resistant crop for low values of δ_{β_R}. The remaining growers free-ride off these actions and do not use any improved crop. However, as δ_{β_R} increases, the benefits to growers of resistant crop are lower, and growers start using tolerant crop. This causes an increase in the proportion of infected fields. Save for δ_{β_R}, parameters and initial conditions are as in Table 4.2 and Table 4.3 respectively.

Similarly to Figure 4.3, between the values of $\delta_{\beta_R} = 0.054$ and 0.077 (denoted by the black bar in Figure 4.4), there is bistability which changes the

point at which growers stop using resistant crop and only use tolerant crop (Appendix 3).

4.3.3 Pareto optimality

Here, as an example, we calculate the Pareto front when neither improved crop type is very effective (the relative susceptibility of resistant crop is $\delta_{\beta_R} = 0.5$ and the relative loss due to disease in tolerant crop is $\delta_{L_T} = 0.5$). Figure 4.6 shows the Pareto front when the planner is trying to minimise the cost of the subsidy scheme whilst also maximising the average profit of growers, with both quantities considered at the eventual equilibrium of the model.

We plotted the Pareto front onto a two-way scan of the cost of resistant and tolerant crop (ϕ_R and ϕ_T respectively) to investigate the subsidy regime that produces optimal outcomes (Figure 4.6(A) - (D)). The strategies that guarantee the best outcome for both the growers and the planner are to use any combination of ϕ_R and ϕ_T that discourages widespread use of tolerant crop (Figure 4.6(D), where the Pareto front lies along the edge of the "U + T" and the "U + R" equilibrium). Below the value of $\phi_R = 0.365$, the optimal subsidy schemes ensure a mixed equilibrium of unimproved and resistant crop (Figure 4.6(D)). Above this value (for which the only optimal strategies are $\phi_T = 0.4$, $\phi_R = 0.365$ or $\phi_T = 0.4$, $\phi_R = 0.367$) resistant crop is too expensive and growers instead use tolerant crop, though it is always at relatively low levels (around 10% of fields are planted with tolerant crop, Appendix C.3 Figure C.4.4).

Though the use of tolerant crop earns the growers high profits, the resulting increase in infection pressure induces a positive feedback loop that incentivises other growers to also use tolerant crop. The planner will therefore have to subsidise more growers, so it is ultimately not economical for the planner, and no Pareto-optimal strategy lies in the region where there is high use of tolerant crop. Conversely, as resistant crop provides benefit to growers who do not use it (i.e. "free ride" off the efforts of others), by subsidising a minority of growers to use resistant crop the planner can achieve good profit outcomes averaged over the population as a whole for lower costs.

Though the Pareto front lies along the diagonal, any points in a given row along to the right of the diagonal have the same value (as there is no tolerant crop at equilibrium, so changing the cost of tolerant crop has no effect on the equilibrium achieved; equivalently, all points in a given column above the diagonal have the same value as there is no resistant crop at equilibrium). They are, therefore, all considered to be Pareto optimal.

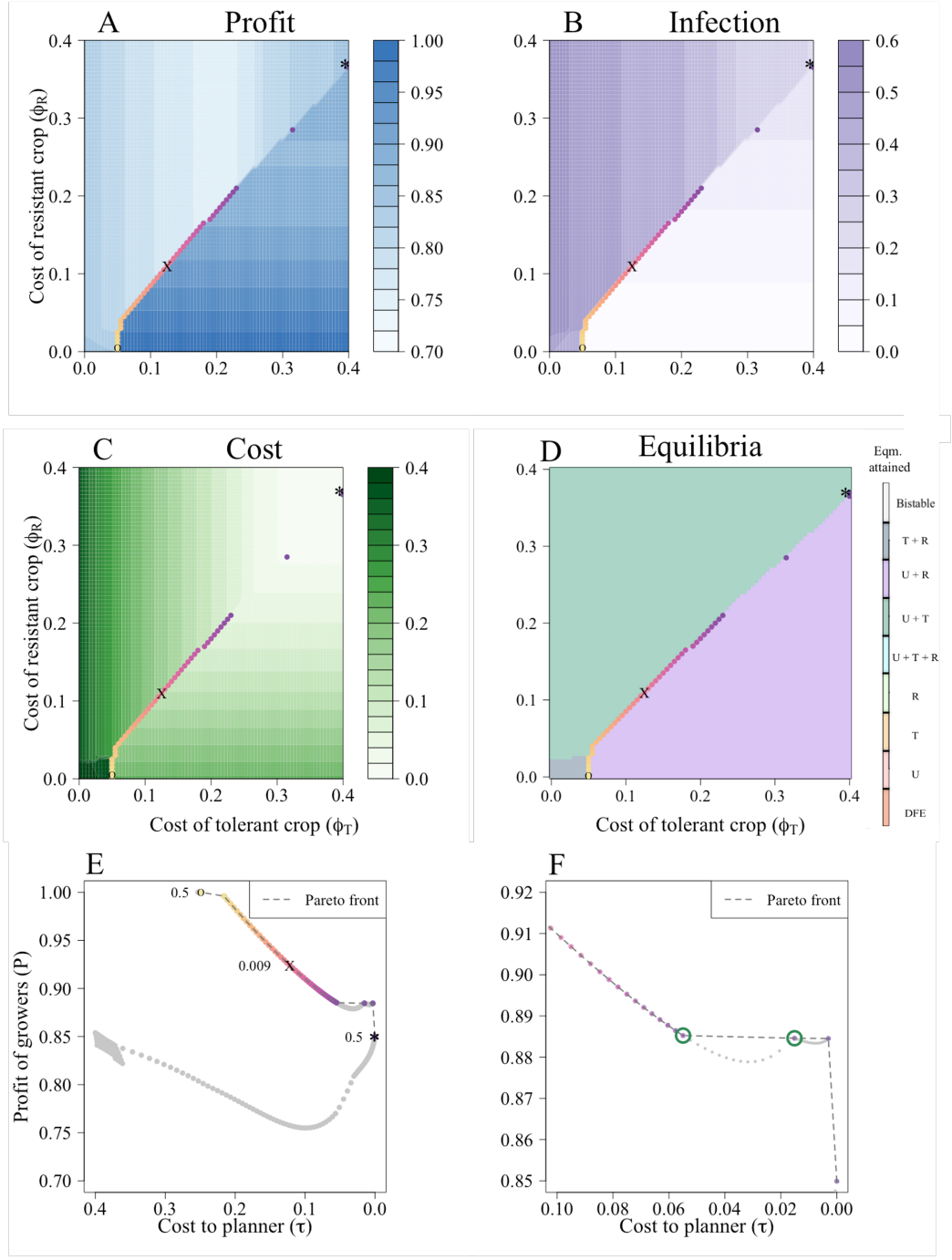

Figure 4.6: **Pareto front when both the tolerant and resistant crop are only moderately effective.** (A) The profit to growers. (B) The level of infection. (C) The cost to the planner (τ, Equation 4.37) and (D) which equilibria are attained. The dots in (A) - (D) represent the combination of ϕ_T and ϕ_R that lie along the Pareto front in (E), and all are parameter combinations that disincentivise the use of tolerant crop. (E) Pareto front when both the tolerant and resistant crop are only moderately effective. The individual grey dots correspond to all pairs of values of (ϕ_T,ϕ_R) considered in our two way scan; the coloured dots are those lying on the Pareto front. The darker the dots, the more the low costs to the planner have been prioritised. The Gini coefficients are shown for the fairest scenario ($G = 0.009$, when $\tau = 0.13$ and $P = 0.93$; "X" on the graphs) and the least fair scenarios ($G = 0.5$; "O" when the profit is prioritised and "*" when the costs to the planner are). (F) Pareto dominance between $\tau = 0.06$ and $\tau = 0.0$, which corresponds to the "break" in the Pareto front in (E). Any solution where $\phi_R < 0.365$, the final equilibrium is an unimproved and resistant crop equilibrium. Otherwise, the equilibrium has unimproved and tolerant crop.

We then plotted the Pareto front for each combination of costs and profits from Figure 4.6(A) and (C) (Figure 4.6(E)). The grey dots are subsidy strategies that are dominated by other strategies that lie on the Pareto front. The Pareto front is broken between $\tau = 0.058$ and $\tau = 0.015$ (indicated by green circles in Figure 4.6(F)). These combinations of costs and profits are dominated by $\tau = 0.015$ and $P = 0.88$, where the profit is at a local maximum. The profits earned by growers when $0.058 > \tau > 0.016$ are all $P < 0.88$, so both growers and planners can achieve better outcomes at $\tau = 0.015$. Between $0.058 > \tau > 0.016$, the subsidisation scheme reduces use of the resistant crop, decreasing profits for the growers as more fields become infected.

The degree of subsidisation (and associated outcome) will depend on the planner's weighting of the relative importance of the cost to the planner and the profit of growers. This can be measured using Gini coefficients. The Gini coefficients are shown for the extremes of equality: the least fair scenarios, with a Gini coefficient of 0.5, lie at either end of the Pareto front. Time courses for these different scenarios are shown in Figure C.4.3 in Appendix C.4.

When the profit to growers is 1.00 and the cost to the planner is 0.25, corresponding to $\phi_R = 0.0$ and $\phi_T = 0.05$ (marked "O" in Figure 4.6), a relatively high proportion of growers use resistant crop ($\approx 49.8\%$, Figure C.4.4(B) in Appendix C.4). At the other end of the front, the profit to the growers is 0.85 and $\tau = 0$. As $\phi_R = 0.4$ and $\phi_T = 0.375$ ((marked "*" in Figure 4.6)), so improved crop is effectively not subsidised. This results in no users of resistant crop and only around 10% of growers use tolerant crop (Figure C.4.3(C) in Appendix C.4), reducing the costs to the planner (this is also the only point on the Pareto front where any

growers use tolerant crop; Figure C.4.4(A) in Appendix C.4).

The fairest scenario ($G = 0.009$, "X") occurs when $\phi_T = 0.12$ and $\phi_R = 0.105$ ($\tau = 0.13$ and $P = 0.93$). At this point, $\approx 40.3\%$ of growers use resistant crop, none use tolerant crop (Figure C.4.4 in Appendix C.4), and only 6% of fields are infected (Figure 4.6 (B)). In this "U + R" equilibrium, the majority of growers can "free ride" off the efforts of those using resistant crop.

4.3.4 Effect of time on the Pareto front

Both the costs to the planner and the profits of growers depend on the proportion of fields of each type in the system. This has a strong temporal component; consequently, the Pareto front may change depending on the time horizon examined. We investigated this temporal effect after 1, 2, 3, 4, and 5 seasons, fixing the cost of resistant crop (ϕ_R) to 0.1 for ease of analysis (that is, in this section, we only consider changes to the cost of tolerant crop, ϕ_T). Importantly, here when we refer to seasons, we refer to the average amount of time a field will be planted before it is harvested (120 days).

At short time points (one or two seasons), nearly every subsidisation scheme lies on the Pareto front (Figure 4.7(A)-(D)). Each of these value of ϕ_T are considered equally efficient, and produce a Pareto-optimal combination of outcomes for both the growers and the planners.

As the epidemic progresses (seasons three, four, and five), only higher values of ϕ_T give Pareto-optimal solutions (Figure 4.7(A)-(D)). The higher cost of tolerant crop disincentivises its use, so fewer growers use it (Figure 4.7(A)). Though there is an increase in the proportion using resistant crop, uptake of resistant crop is

still relatively low (Figure 4.7(B)) due to the ability of non-controllers to free-ride. Overall, the higher values of ϕ_T lowers the costs to the planner, whilst maintaining high profits for the growers (Figure 4.7(C)-(E)).

Yet, between three and four seasons into the epidemic, low values of ϕ_T also permit Pareto-optimal solutions. At these values, relatively few of the growers are using tolerant crop (Figure 4.7(F)) and the costs to the planner are relatively low, whilst the profits to the grower are high (Figure 4.7(C)- (D)).

The Pareto fronts show that, over these shorter time horizons, there is often a small difference in the profits and costs for the optimal cases (Figure 4.7(E)). We again find that the costs of improved crop that discourage the use of tolerant crop are optimal. For each time horizon, the Pareto optimum is achieved at higher values of the cost of tolerant crop to growers (ϕ_T), which discourages the use of tolerant crop. This implies that in the early stage of an epidemic, there is not as severe a trade-off between the optimal outcomes for the planner and the grower. Figure 4.7(F) shows a time course of the proportion of growers using tolerant crop. At low values of the cost of tolerant crop (ϕ_T), which are only part of the Pareto-optimal sets for short time horizons (between one and four seasons), tolerance does not die out. At higher values, tolerance dies out within five seasons. Therefore, whilst lower values of ϕ_T can sometimes provide Pareto-optimal solutions, higher values give optimal outcomes irrespective of the time horizon.

The same results hold for other parameterisations of tolerance and resistance - irrespective of how good or bad the improved crop varieties are, the Pareto optimal strategies are those in which the use of tolerant crop is discouraged.

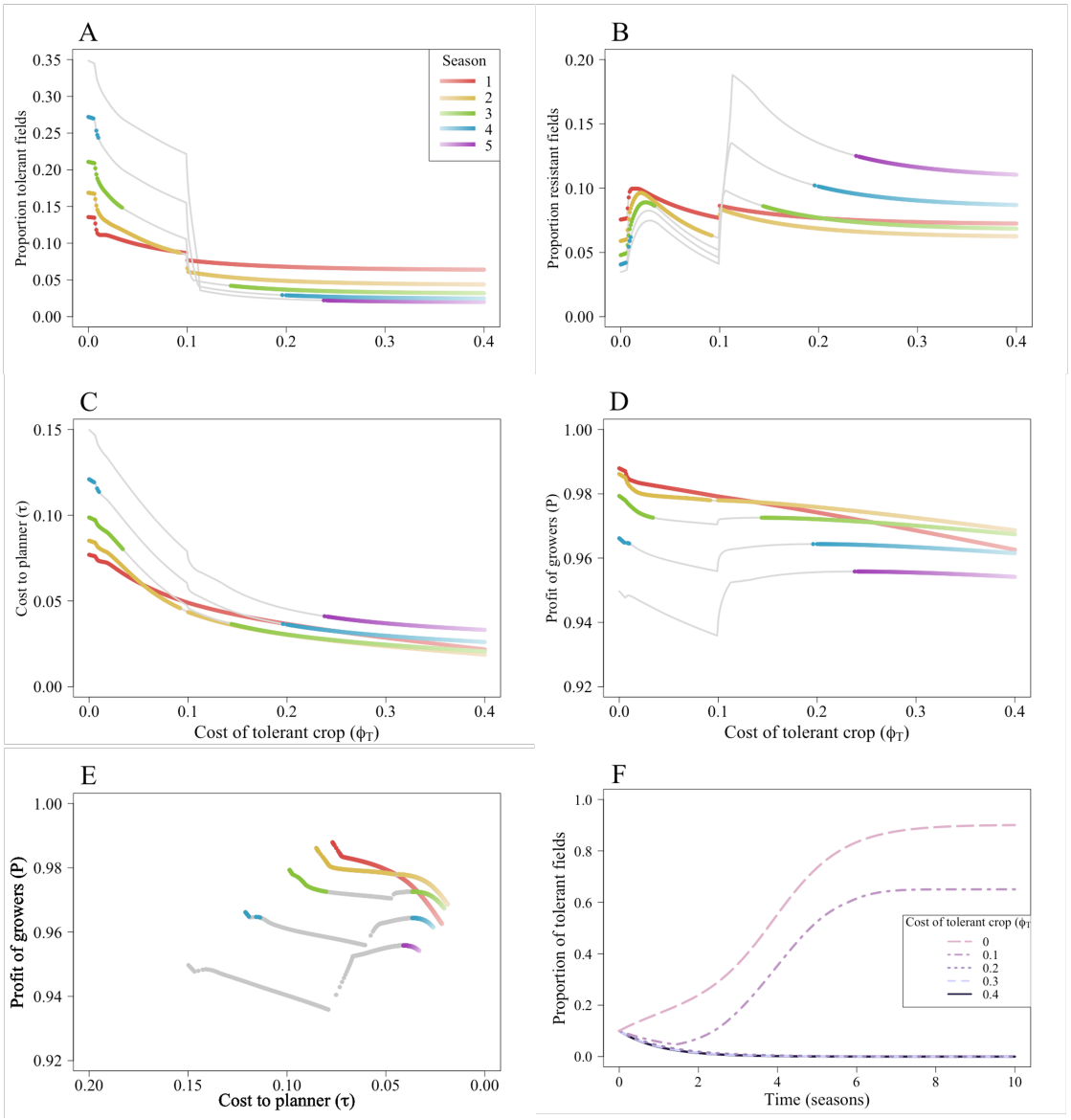

Figure 4.7: **Pareto front at early seasonal time points when both the tolerant and resistant crop are only moderately effective.** (A) The proportion of tolerant fields. (B) The proportion of resistant fields. (C) The cost of the subsidisation scheme to the planner. (D) The profit to the growers. The darker the colour, the more the profit of growers is prioritised over the cost to planners. Generally, the Pareto front lies along values of ϕ_T that reduce the proportion of growers using tolerant crop and increase those using resistant crop. (E) At low time points, all all subsidisation schemes are Pareto-optimal, though as time progresses there are a narrower range of optimal solutions. (F) Once $\phi_T > 0.2$, no growers use tolerant crop after 5 seasons. Other than $\delta_{L_T} = 0.5$ and $\delta_{\beta R} = 0.5$, parameters are as in Table 4.2. Note, in each of these graphs, the cost of resistant crop (ϕ_R) is 0.1.

4.4 Discussion

Human behaviour has increasingly been investigated in the context of plant disease epidemiology (Milne et al. (2016), McQuaid et al. (2017a), Saikai et al. (2021), Milne et al. (2020), Bate et al. (2021)), where models allow growers choose between engaging in disease control or taking no action. The proportion of growers using control depends on the control mechanism itself, with previous studies investigating the uptake of clean seed systems amongst cassava growers (McQuaid et al. (2017a)) or transgenic herbicide-resistant maize (Milne et al. (2016), Saikai et al. (2021)). Uptake will also depend on the relative costs of control and losses due to disease, amongst other factors not investigated here (such as the social connections between growers). However, in each of these cited examples, growers could only choose whether to use control or not, not between different types of control.

In Chapter 3, when investigating the effect of having crop that was either tolerant or resistant to Tomato Yellow Leaf Curl Virus (TYLCV) available to growers (alongside unimproved crop), we found that widespread use of tolerant crop was often achieved. Tolerant crop only benefited those growers using it, and due to the lower rate of removal via roguing, they increased the infection pressure on other growers (generating negative externalities). However, when resistant crop was available, such high levels of adoption were not achieved. Resistant crop protected other, unimproved fields (generating positive externalities), who can *free-ride* off the efforts of those using resistant crop.

These results were corroborated in the work presented here. Even when growers have a choice between three crop types (unimproved, disease-tolerant or -resistant),

the positive feedback loop induced by the use of tolerant crop is sufficient to ensure that growers will overwhelmingly use tolerant crop, even when tolerant crop was relatively ineffective (Figure 4.3(A)). This positive feedback loop means that tolerant crop is a "strategic complement" (a strategy that incentivises others to adopt the strategy as more individuals use that strategy; Hennessy (2008), Murray (2014), Delabouglise & Boni (2020)). Resistant crop, by contrast, is a "strategic substitute": its use discourages others from also using resistant crop, as they can free-ride off of the efforts of others.

With the default costs of each crop type (where the grower paid $\phi_T = \phi_R = 0.1$), only when tolerant crop is very ineffective (sustaining a high relative loss of yield) do some growers consider resistant crop (Figure 4.3(A)). Similarly, if resistant crop is very effective (with a low relative susceptibility), there will be a mixed "resistant and unimproved" crop equilibrium (Figure 4.4). However, due to the free-riding, universal adoption of resistant crop is not achieved, echoing an analogous result in the simpler two strategy model (Chapter 3). Therefore, a mixed equilibrium persists (such as the "unimproved and resistant" equilibrium in Figure 4.4(A)).

We used the concept of Pareto efficient strategies (Luc (2008)) to optimise the dual objectives of ensuring high profits for growers whilst also minimising the cost to social planners. The positive-feedback induced by the use of tolerant crop means that it will have widespread adoption by growers, so those providing subsidies will have to do so for nearly all growers. This increases the cost to the planner (τ), though does increase the profits to the growers (P) (Figure 4.6(E)). However, relatively high profits can also be obtained when resistant crop is subsidised (at least 85% of the maximum theoretical profit, Figure 4.6(E)). Furthermore, as the

adoption of resistant crop is not as widespread as that of tolerant crop, most of the profits can be achieved with less investment from the planner. Therefore, the control strategies on the Pareto front require the planner to provide sufficient subsidies to resistant crop to ensure that it is used whilst never incentivising tolerant crop (Figure 4.6(A)-(D)). Most optimal subsidisation schemes result in a "unimproved and resistant" crop equilibrium (Figure 4.6(D)).

By targeting subsidies at resistant crop, the social planner can exploit the free-riding behaviour of growers not growing improved crop and only subsidise a subset of growers. Targeting subsidies to resistant crop can be seen as a way of internalising some of the positive externalities produced by these growers; the financial benefit of the subsidy is a way for them to experience some of the benefits that they generate for others.

Conversely, not subsidising tolerant crop internalises their negative externalities (Gersovitz (2014)). This strategy was optimal irrespective of the time course over which profits and costs were calculated (Figure 4.7(F)). However, earlier in the epidemic (between 1-4 seasons), subsidisation schemes that resulted in some fields planted with tolerant crop were possible, but only when the costs to the planner were sufficiently low (Figure 4.7).

These models ignore the possibility of virus evolution, which is a major threat to the durability of genetic disease control mechanisms (Gallois et al. (2018), Sett et al. (2022)). Tolerant crops, by allowing pathogen replication, do not exert the same selection pressure (Råberg et al. (2009)) and therefore may provide a more durable protection against yield loss (Bingham et al. (2009), Newton (2016), Zhu et al. (2004)). Resistant crop exerts much stronger selection pressures on the

pathogen, often leading to "resistance breakdown" where the crop's resistant traits become ineffective against infection (García-Arenal & McDonald (2003), Parlevliet (2002)). Over the short term, it may be cheaper for the planner to subsidise the use of resistant crop, but when considering the potential evolution of a resistance-breaking pathogen and the associated costs of developing and disseminating new crop types, tolerant crop may be more favourable.

Other simplifying assumptions were made for this study. We did not include spatial or stochastic effects (Cunniffe et al. (2015b), Hyatt-Twynam et al. (2017), Cunniffe & Gilligan (2020), Fabre et al. (2021)), both of which can influence disease progression and growers' decision-making. We also did not differentiate between the nature or quantity of information available to each grower, both of which will depend on the grower's social and professional network (Milne et al. (2016), Sherman & Gent (2014)) or allow for differing perceptions of risk (our values of responsiveness, η_b, $b \in \{U, T, R\}$ were the same across all growers irrespective of control strategy). A grower's "risk attitude" will be influenced by factors such as their previous experience with disease outbreaks (Garcia-Figuera et al. (2021)) or received knowledge from other growers (Sherman & Gent (2014)) and will impact their control decisions and consequently disease progression (Chapter 2). Growers, then, are ultimately "reflexive producers" (Kaup (2008)), and their control decisions must balance each of these information sources with external factors such as market pressures.

Our model demonstrates how decision models of grower behaviour can be combined with other economic concepts, such as Pareto fronts, to find socially-optimal solutions across conflicting objectives. We found that what was optimal for the

growers (using tolerant crop) led to worse outcomes for the planner, and the social optimum occurred when a subset of the growers used resistant crop. Though we examine these trade-offs for a crop that is either tolerant or resistant to TYLCV, the model form is sufficiently flexible to allow for other pathosystems or control mechanisms.

Chapter 5: Including grower behaviour in spatial-stochastic models [1]

5.1 Introduction

5.1.1 Introduction to spatial modelling

In the previous chapters, we have assumed that the population is well-mixed: all susceptible fields planted with the same crop type have an equal probability of infection, and growers have access to information on the expected profits of all other growers when making their planting decisions. Yet, in reality, the spatial arrangement of fields in a landscape will influence both of these factors, as fields closer to already-infectious fields have a higher probability of infection than those on the other side of the landscape. Similarly, growers may be more likely to interact with those around them, particularly in resource-poor areas where other

[1] Some of the work presented in this chapter is based on Murray-Watson *et al.*, 2022.

communication routes may be lacking. With this in mind, this chapter aims to develop spatial-stochastic analogues of the models presented in Chapters 2 and 3 to investigate the impact of accounting for randomness in pathogen dynamics and the influence of space on the epidemic progression and decisions of growers.

Including spatial heterogeneity in models of plant diseases allows us to create more detailed models that allow for different scales of pathogen dispersal, varying contact networks between growers, and more detailed co-evolutionary processes between plants and pathogens (Burdon & Thrall (1999), Watkinson-Powell et al. (2020)). It can also be used to determine the probability of invasion of a plant pathogen (Mikaberidze et al. (2016), Gilligan & van den Bosch (2008)), which will strongly depend on the arrangement of fields in the landscape. This allows us to move away from the relatively binary $R_0 > 1$ invasion threshold required in most deterministic models (except when there is bistability between a disease-free and disease-endemic equilibrium for $R_0 < 1$).

In particular, spatial models allow for an investigation of certain control policies which organise treatment in space depending on the system's current state. These can be pre-emptive in nature, anticipating which areas may be more at risk of infection and allowing decision-makers to focus management efforts on those areas (Cunniffe et al. (2015b), Gilligan et al. (2007), Meentemeyer et al. (2011), Hyatt-Twynam et al. (2017)). Reactive culling, where all plants within a certain radius of a confirmed infectious plant are removed, has been used in models of huanglongbing (Cunniffe et al. (2015b)) or sudden oak death (Cunniffe et al. (2016)). Such focused control can allow better allocation of resources. Spatial models have also been used to study the optimal deployment of resistance genes (Watkinson-Powell et al.

(2020)) or to compare the efficacy of different management schemes (Ferris et al. (2020)).

5.1.2 Spatial models and changing the source of information

Spatial models can also offer a greater understanding of the social dynamics that underpin growers' decisions on whether to adopt a particular control mechanism. A significant assumption of the previous game-theoretic models is that growers will have access to population-level data regarding infection status, profits and strategies adopted by other growers. This information may, in reality, be essentially impossible to access, particularly in infrastructure-poor regions. Instead, growers may compare with individuals in their vicinity rather than the wider population. This may be through choice, as growers may trust the experiences of those in their community more, or because this is the most readily accessible information. Indeed, in a study of cassava growers in Zambia, neighbours, family and friends were cited as common sources of information regarding cassava diseases and CSS (Szyniszewska et al. (2021)). Radio is also a common source of information, from which growers might receive landscape-level news. Indeed, in Nigeria, radio dramas were found to be a successful means of disseminating information on climate change adaptation to smallholder farmers (Ojedele (2016)). The majority of growers interviewed in Szyniszewska et al. (2021) also said they would consider using a CSS if a small group of their neighbours used it (between two and four other growers). Kaup (2008) also observes, in the context of *Bt* maize in the United States, that growers are more likely to be influenced by local experiences.

These sources of information impact dynamics, as growers limit their pool of

comparison to those experiencing similar infection pressures. If the infectious fields are highly localised, growers would have a better estimate of profitability by only comparing with growers in their area. By comparing with the entire population, they may over- or under-estimate their own probability of infection, resulting in sub-optimal decisions. Over-estimating the probability of infection could deter growers from using control, as they believe they will be infected regardless and pay a dual penalty of the loss due to disease and cost of control. Similarly, under-estimating the infection risk could reduce the probability of growers using control as they perceive it to be unnecessary.

In their model of European Corn Borer (ECB) spread across the landscape, Milne et al. (2016) included different contact networks between growers. These contact networks influenced the nature of the information growers could access on the profitability of *Bt* maize, a genetically-modified crop that produces a pesticide used to manage ECB. Networks were modelled over four scales: local (in which growers can only assess profitability with farms that directly neighbour their own), landscape (where growers have information on profits from all other growers), "Kaup" (where, based on a study of grower behaviour and their access to information regarding *Bt* crops by Kaup (2008), growers have a set probability of being influenced by an agronomic consultant, their neighbour or a university extension officer) and "varying response" (which uses the same probabilities as the "Kaup" network, but includes a reluctance on the part of the grower to switch back to conventional crop after using *Bt* maize the previous season). The losses experienced by growers when they only considered close neighbours were nearly three times those when they considered a broad range of sources. Though the landscape network had lower losses than the neighbourhood network, it also had

much more regular cycles of adoption and abandonment of *Bt* maize. The inclusion of these spatial network dynamics allows a much more in-depth analysis of how information impacts the control decisions of growers.

However, if disease is so prevalent in the local area that infection is almost guaranteed, growers may decide that control is not worthwhile. Therefore, more pronounced "localities" of adoption, or neighbourhoods of controllers and non-controllers, may be seen. McQuaid et al. (2017a) found that increased reliance on information from local groups decreased the overall use of clean seed systems but also reduced the oscillations in uptake observed. This corroborates with Milne et al. (2016)'s observation that there were fewer oscillations in adoption when they were only comparing with closer neighbours. Basing control decisions on local groups may also create an inadvertently coordinated response to either control or not control, as they will all experience the same local disease pressures. However, the emergence of such cooperating clusters could be confounded by the stochastic nature of decisions. Examples from human disease literature suggest that heterogeneous contact networks, where individuals have unequal probabilities of interacting with other individuals, can lead to "clusters" across the network within which individuals will have similar opinions relating to disease control (Salathé & Bonhoeffer (2008), Eames (2009)). This can hinder disease management efforts, as clusters of individuals who are equally sceptical of the utility of control can allow disease to invade.

Spatial modelling does have its constraints and can be severely limited by the availability of data relating to pathogen dispersal, the scale at which control should be enacted (Gilligan et al. (2007)) and the lack of information regarding the

location of fields in the landscape (Cunniffe et al. (2015a)). Even so, approximating these quantities may improve the predictions from mean-field models.

5.1.3 Adding stochastic effects

To add a degree of complexity, we also allow for the effect of randomness by incorporating stochasticity into our spatial models. Whilst deterministic models often allow for mathematical analysis that enable us to make useful predictions of disease dynamics (such as our "strategy vs population" and "strategy vs alternative" models in Chapter 2, for which we could find stability conditions for different equilibria), they ignore the effect of randomness that is pervasive in biological systems (Fabre et al. (2021)). Though less analytically tractable, stochastic models account for these effects and importantly allow for the possibility of disease extinction through random events (which cannot happen in deterministic models once $R_0 > 1$, unless the system is bistable) (Gilligan & van den Bosch (2008)). We use Gillespie's algorithm to simulate trajectories from our stochastic models (Gillespie (1977), Keeling & Rohani (2008)).

5.1.4 Aims of this chapter

Broadly, this chapter aims to develop spatial-stochastic analogues of the deterministic models presented in Chapters 2 and 3. We do this using the "grower vs alternative" behavioural model presented in Chapter 2, where growers compare the profit obtained from their previous harvest with the expected profit of the alternative strategy. This model is based on "strategic-adaptive expectations", where

growers use both their own experience and estimates for the entire population, emulating Kaup's idea of the "reflexive producer" (Kaup (2008)).

For the spatial-stochastic analogue to the model of cassava brown streak disease (CBSD) and clean seed systems (CSS) from Chapter 2, we investigate the following questions: (1) How does the inclusion of spatial and stochastic effects impact the predictions made from the deterministic model? (2) How does participation in the CSS vary according to economic and epidemiological parameters in a spatial-stochastic model? (3) What effect does the inclusion of systematic uncertainty have on growers' participation in the CSS in a spatial-stochastic model?

Using the equivalent model for the uptake of tolerant or resistant crop for the control of tomato yellow leaf curl virus (TYLCV) from Chapter 3, the following questions are addressed: (1) How does the inclusion of spatial and stochastic effects impact the predictions made from the deterministic model? (2) How does varying the scale of the information accessed by the growers impact control uptake? (3) Do clusters of growers with similar management strategies develop?

5.2 Methods

5.2.1 Cassava brown streak disease and clean seed systems

We use an individual-based version of our model from Chapter 2 to test the robustness of our conclusions to spatially explicit and/or stochastic effects. The model outlined here is based on Equations 2.5 - 2.10 in Chapter 2. The model tracks the spread of CBSD across a population of 750 fields by two routes: horizontal transmission via viruliferous whitefly vectors or vertically via trade-mediated vegetative

propagation of infected cassava cuttings. Fields are harvested asynchronously, and replanting occurs at the time of harvest. Where growers source their cassava cuttings - via replanting/trade or from the clean seed system - depends on their estimation of the profit they could make if they used either strategy. As in the deterministic model, we track both the infectious status of each field (susceptible, S, or infected, I) and the control strategy used by the grower (clean seed, C, or non-clean seed, N). Our model therefore tracks the number of individuals in each of four compartments:

- Susceptible fields of non-controllers (S_N),
- Infected fields of non-controllers (I_N),
- Susceptible fields of controllers (S_C) or
- Infected fields of of controllers (E_C).

Host landscape and initial conditions

Disease spreads on a 1,000 Ha square landscape containing $N = 750$ identically-sized fields (a similar density to McQuaid et al. (2017a), with the same number of fields as the deterministic model), placed at uniform random locations. All simulations have the same initial conditions as the deterministic models and are started with 75 growers using clean seed (i.e. 10% of the total population), distributed randomly across the landscape. A random selection of eight non-controllers is set to be initially infected ($\approx$ 1% total population). The location of the fields was kept constant between simulation runs (as the dispersal matrix of the whitefly

vector was pre-calculated), which reduced computation time and allowed for direct comparison of the effect of changes in parameters or initial conditions rather than changes in the landscape set-up. However, we allowed for stochasticity in our initial conditions and the growers who were either initially infected or used the clean seed system differed between simulations.

Parameterisation

The majority of parameters in the spatial model took the same values as those in the simpler non-spatial model (Table 1 in Chapter 2). However, the rate of CBSD transmission via the whitefly vector *B. tabaci* will depend on its ability to spread between fields. Following McQuaid et al. (2017a) and based on mean dispersal distance of *B. tabaci* reported by Byrne et al. (1996) and Byrne (1999), we set the scale parameter of our dispersal kernel to be $\alpha = 150$ m. The inclusion of space and the whitefly dispersal kernel meant that our previous parameterisation of the rate of secondary infection was no longer appropriate. We therefore re-scaled the rate of secondary infection, which we set to $\beta_S = 110$ day^{-1} field^{-1}. Again, this value was chosen to ensure that, on average, 50% of fields would be infected after ten seasons. These values are summarised in Table 5.1.

Dispersal and force of infection

Since horizontal transmission is a consequence of whitefly movement, the probability that a particular infected field leads to infection of a susceptible field depends upon the distance d between them. We model this using an exponential dispersal

Table 5.1: **Summary of parameter values and initial conditions required for the spatial-stochastic model of CBSD spread.** Parameters marked with an asterisk ("*") have the same value as that of the deterministic model in Chapter 2.

Parameter	Meaning	Value	Reference
$1/\gamma$ *	Length of the growing season	300 days	McQuaid et al. (2017a); Jeger et al. (2004)
η *	Responsiveness of growers	10	Assumed (Chapter 2)
Y *	Maximum yield	1	All values scaled relative to yield
L *	Loss due to infection	0.6	Hillocks et al. (2001); Ephraim et al. (2015)
ϕ *	Cost of control	0.25	McQuaid et al. (2017a)
N *	Total number of fields/growers	750	Illustrative
α	Dispersal scale parameter for whitefly	150m	McQuaid et al. (2017a); Byrne (1999)
β_S	Effective rate of secondary infection (spatial)	110 day^{-1} field^{-1}	Calibrated to McQuaid et al. (2017a)
$S_C(0)$ *	Initial proportion of susceptible controllers	$0.1N$	Illustrative
$I_C(0)$ *	Initial proportion of infected controllers	0	Illustrative
$S_N(0)$ *	Initial proportion of susceptible non-controllers	$0.893N$	Illustrative
$I_N(0)$ *	Initial proportion of infected non-controllers	$0.0107N$	Illustrative

kernel

$$K(d, \alpha) = \frac{1}{2\pi\alpha^2} \exp\left(-d/\alpha\right), \tag{5.1}$$

in which α is the dispersal scale (Cunniffe & Gilligan (2020), Fabre et al. (2021)). This sets the force of infection, Γ_k, upon the field belonging to grower k

$$\Gamma_k = \beta_S \sum_{\substack{l \neq k \\ l \text{ is infected}}} K(d_{k,l}, \alpha), \tag{5.2}$$

in which the sum runs over all infected fields (l), $d_{k,l}$ is the distance between fields k and l, and β_S is the effective rate of infection in the spatial version of our model (Table 5.1). The force of infection sets the rate at which grower k becomes infected; it varies in both time and space (since it depends not only on the instantaneous number of infected fields but also on their spatial arrangement).

Model dynamics

Each individual grower is characterised by their current strategy as adopted on planting (i.e. controller or non-controller), as well as their current infection status (i.e. susceptible or infected). Between harvesting and replanting events, dynamics are independent of the strategy adopted. We simulate the model using Gillespie's algorithm (Keeling & Rohani (2008)).

The only event that can affect an infected grower (i.e. class I_C or I_N) is harvesting; this occurs at rate γ. As well as harvesting, susceptible growers (i.e. class S_C or S_N) can also become horizontally infected. This occurs, for grower k, at rate Γ_k (Equation 5.2). As noted above, the force of infection not only depends on time but also takes different values for each grower.

Replanting and grower behaviour

Replanting is assumed to occur immediately after harvesting and potentially leads to changes in both strategy and infection status. Different growers make different assessments of the probability of horizontal infection next season depending on their instantaneous force of infection, with

$$\begin{aligned}\text{Estimated probability of} \\ \text{horizontal infection for grower } k\end{aligned} = \frac{\text{Force of infection on grower } k}{\text{Force of infection on grower } k + \text{Harvesting rate}}, \tag{5.3}$$

$$= \frac{\Gamma_k}{\Gamma_k + \gamma}, \tag{5.4}$$

We assume growers make decisions according to spatial analogues of Equations 17 and 20 in Chapter 2:

$$\begin{aligned} q_C^k = & \begin{array}{c}\text{Grower } k\text{'s estimate of the probability of} \\ \text{infection next season if control is adopted,}\end{array} \\ = & \frac{\Gamma_k}{\Gamma_k + \gamma}, \end{aligned} \tag{5.5}$$

and

$$q_N^k = \begin{array}{c}\text{Grower } k\text{'s estimate of the probability of} \\ \text{infection next season if control is not adopted,}\end{array}$$
$$= \frac{p(I_C + I_N)}{N} + \left(\frac{\Gamma_k}{\Gamma_k + \gamma}\right)\left(1 - \frac{p(I_C + I_N)}{N}\right). \tag{5.6}$$

All other aspects of the behavioural model follow essentially unchanged, although the switching terms now enter the model via a single Bernoulli trial (Grimmett & Welsh (2014)) at the time of planting to determine whether a grower switches strategy. Whether or not non-controllers become vertically infected is also simulated using a single Bernoulli trial at the time of planting (rather than via a systematic alteration to a rate parameter Equations 2.5 - 2.10 in Chapter 2).

Misestimating parameters

We also investigated the misestimation of epidemiological parameters in the spatial model, focusing on the dispersal scale (α) and the rate of horizontal transmission, β_S:

$$q_\alpha = \nu_\alpha \alpha, \tag{5.7}$$

$$q_{\beta_S} = \nu_{\beta_S} \beta_S. \tag{5.8}$$

The exponential dispersal kernel ($\tilde{K}(d, q_\alpha)$) used in growers' decision making is

now given by:

$$\tilde{K}(d, q_\alpha) = \frac{1}{2\pi q_\alpha^2} \exp\left(-d/q_\alpha\right), \tag{5.9}$$

which sets the estimated force of infection, $\tilde{\Gamma}_k$, upon the field belonging to grower k via

$$\tilde{\Gamma}_k = q_{\beta_S} \sum_{\substack{l \neq k \\ l \text{ is infected}}} \tilde{K}(d_{k,l}, q_\alpha). \tag{5.10}$$

5.2.2 Tomato yellow leaf curl virus

There is a significant overlap between the model set-ups for the spatial-stochastic model of CSS and the model presented here of TYLCV and the adoption of tolerant or resistant crops. TYLCV is also spread by *B. tabaci*, so the dispersal of the whitefly across the landscape and resulting force of infection on fields is modelled using Equations 5.1-5.2 above. Growers make decisions at the time of harvest (or when an infectious field has been detected and rogued). They estimate the profitability of the alternative strategy using Equations 5.3 - 5.6 and whether they change strategy is determined by a single Bernoulli trial.

However, TYLCV has a latent period after initial infection, during which the tomato crop is infected but not infectious and does not show any symptoms of disease. In this model, we therefore have three compartments delineating infection status: susceptible (S), latently infected (E) and infectious (I). The two control strategies tracked improved crop (C) or unimproved crop (U); whether the improved crop is tolerant or resistant depends on the parameterisation. Fields can therefore be classed as one of:

- Susceptible fields planted with unimproved crop (S_U),
- Latently-infected fields planted with unimproved crop (E_U),
- Infectious fields planted with unimproved crop (I_U),
- Susceptible fields planted with improved crop (S_C),
- Latently-infected fields planted with improved crop (E_C) or
- Infectious fields planted with improved crop (I_C).

Host landscape and initial conditions

As there are limited data on the density and distribution of tomato growers in Sub-Saharan Africa, we consider the same spatial set-up of growers as in the CBSD model described above. Though tomato is not a subsistence crop, we can imagine that small-scale producers will be similarly spread across the landscape. Additionally, we no longer consider trade of plant materials, both because tomato is not traded between growers in the same way as cassava and because there is limited evidence for the vertical transmission of TYLCV in tomato (Kil et al. (2016), Pérez-Padilla et al. (2020)).

We therefore uniformly distribute 750 small-holder tomato growers on a 1,000 Ha square landscape. We begin all simulations with 10% of growers using improved crop (i.e. 75 growers) and eight fields planted with unimproved crop are infectious ($\approx$ 1% total population). As with the CBSD model, we keep the location of fields in the landscape constant between simulations, though vary which fields are initially planted with each crop type or are infectious.

Parameterisation

Again, most of the parameters take the same values outlined in Table 3.2 in Chapter 3. As TYLCV is also spread by *B. tabaci*, the scale parameter of our dispersal kernel is set to be $\alpha = 150$m (Byrne (1999)). To match the dynamics of the deterministic model without control, we parameterise the spatial model such that if we begin with just 1% of fields being infectious, 60% of fields become fully infectious (I_U) after ten seasons. This requires a value of $\beta = 980$ day^{-1} field^{-1}. These values are summarised in Table 5.2.

Scales of information access

For this model, we also consider the effects of the spatial scale of the information the grower uses to make decisions. At the time of replanting, we assume that a grower will assess the profitability of all those using the alternative strategy within their *comparison group*. We model four scales of comparison: "own" information, where growers compare their profit from the previous season with the expected profit *they* can expect to achieve based on their own probability of infection of the next season; close neighbours ($<$ 100 m, for which growers compare with three neighbours on average), a local group ($< 1,000$m, approximately 200 other growers) or the entire community of growers (Figure 5.1).

Spatial auto-correlation

As growers in close proximity to each other will experience similar disease pressures, they will likely choose similar management practices. We use Moran's I

Table 5.2: **Summary of parameter values and initial conditions required for the spatial-stochastic model of TYLCV.**

Parameter	Meaning	Value	Reference
$1/\gamma$	Length of the growing season	120 days	Holt et al. (1999*a*); Rocco & Morabito (2016)
η	Responsiveness of growers	10	Assumed (see Chapter 2)
Y	Maximum yield	1	All values scaled relative to yield
L	Loss due to infection	0.6	Riley & Srinivasan (2019)
ϕ	Cost of improved crop	0.1	Fonsah et al. (2018)
ϕ_R	Relative reduction in loss due to roguing	0.7	Illustrative
Δ	Time between roguing	120 days	Illustrative
ν	Probability of detection	1	Illustrative
μ_U	Removal rate (unimproved)	1/60 day^{-1}	Illustrative
μ_C	Removal rate (improved)	1/60 or 1/1140 day^{-1}	Illustrative
$\frac{1}{\epsilon}$	Average latent period	41 days	Holt et al. (1999*b*); Ber et al. (1990)
N	Total number of fields/growers	750	Illustrative
α	Dispersal scale parameter for whitefly	150m	Byrne (1999)
β_S	Effective rate of secondary infection (spatial)	970 day^{-1} field^{-1}	Calibrated to Chapter 3
$S_C(0)$	Initial proportion of susceptible controllers	$0.1N$	Illustrative
$I_C(0)$	Initial proportion of infected controllers	0	Illustrative
$S_N(0)$	Initial proportion of susceptible non-controllers	$0.88.93N$	Illustrative
$I_N(0)$	Initial proportion of infected non-controllers	$0.0107N$	Illustrative

(Moran (1950)) to measure the degree of spatial auto-correlation (the similarity of control strategies based on where the fields are in the landscape).

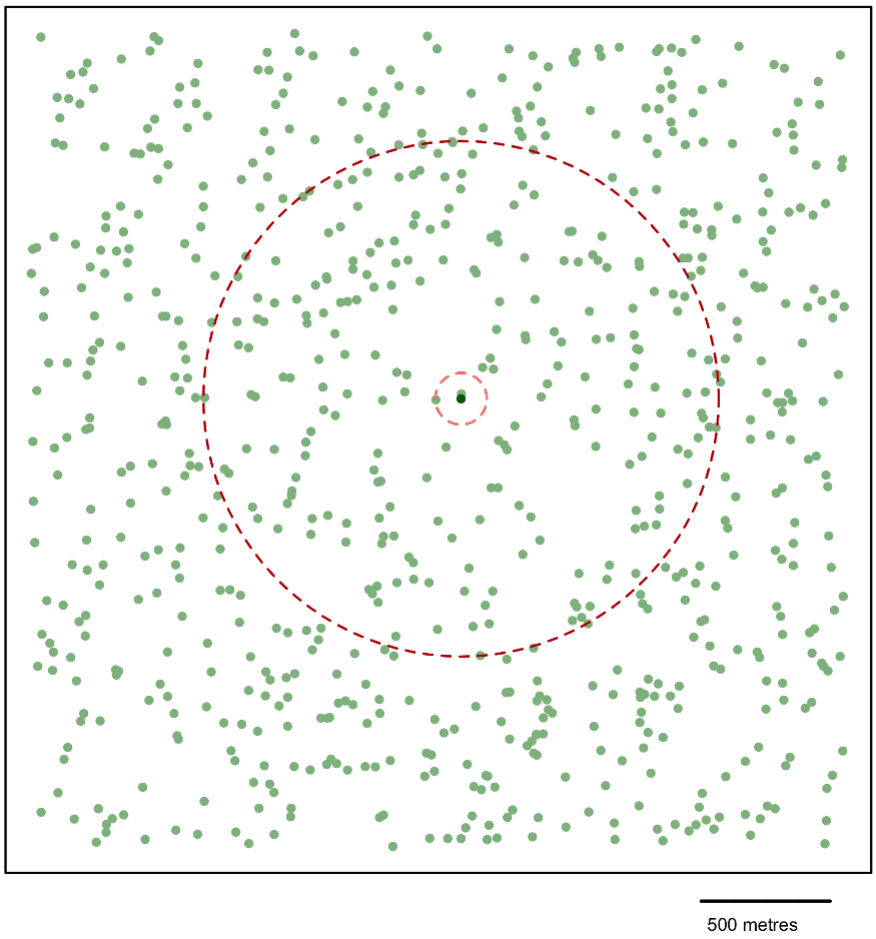

Figure 5.1: **Scales of comparison groups used by the grower when assessing profitability.** The dark green field is the focal grower. The light red circle has a radius of 100m (which, for this focal grower, includes two other growers), whilst the dark red has a radius of 1,000m (which includes 220 other growers). The entire landscape has 750 growers.

To calculate Moran's I, we first calculate a *weight matrix*, which contains the distance-based weights for fields i and j. We investigate clustering within 200m of the focal grower. We do this for several comparison radii (i.e. growers can compare with fields that are within $100, 250, 500, 750, 1000, 1500, 2000, 2500$ and 3000m of the their own). Any field within this radius is given a weight of 1 ($w_{ij} = 1$), and

all other fields get a weight of 0 ($w_{ij} = 0$, including when $i = j$)). The control strategy used by a grower is also given a binary classification (x): fields using improved crop are given a value of 1, and unimproved crop 0. These are then used in the following formula:

$$I = \frac{N}{W} \frac{\sum_{i=1}^{N} \sum_{j=1}^{N} w_{ij}(x_i - \bar{x})(x_j - \bar{x})}{\sum_{i=1}^{N}(x_i - \bar{x})^2} \tag{5.11}$$

where N is the number of fields, W is the sum of the weight matrix (i.e. $W = \sum_{i=1}^{N} \sum_{j=1}^{N} w_{ij}$) and $\bar{x}$ is the mean of x. To calculate Moran's I, we use the *ape* package in *R* (Paradis et al. (2019)). We calculated Moran's I after ten seasons.

5.3 Results

5.3.1 Cassava brown streak disease and clean seed systems

Spatial spread of disease

Figure 5.2 shows the spatial component of disease spread in the spatial-stochastic model, restricting attention to the case when there is only horizontal transmission ($p = 0$). Disease then spreads in a "wavelike" pattern, as expected from the thin-tailed exponential dispersal kernel adopted for whitefly. After 10 seasons, only 50% of fields were infected. As there was no vertical transmission, we also removed "control" from the strategy set of the growers, as there was no benefit to controlling for disease, and the strategy would quickly disappear from the population. Unlike the other models, we also start our epidemic with a cluster of infected fields. Making these changes allows us to focus on the underlying dynamics of the model.

Once we allow for vertical transmission ($p = 1$, so cuttings from infectious fields are guaranteed to transmit CBSD when replanted), disease spreads rapidly across the landscape (Figure 5.3.1). Within five seasons, over 78% of fields are infected.

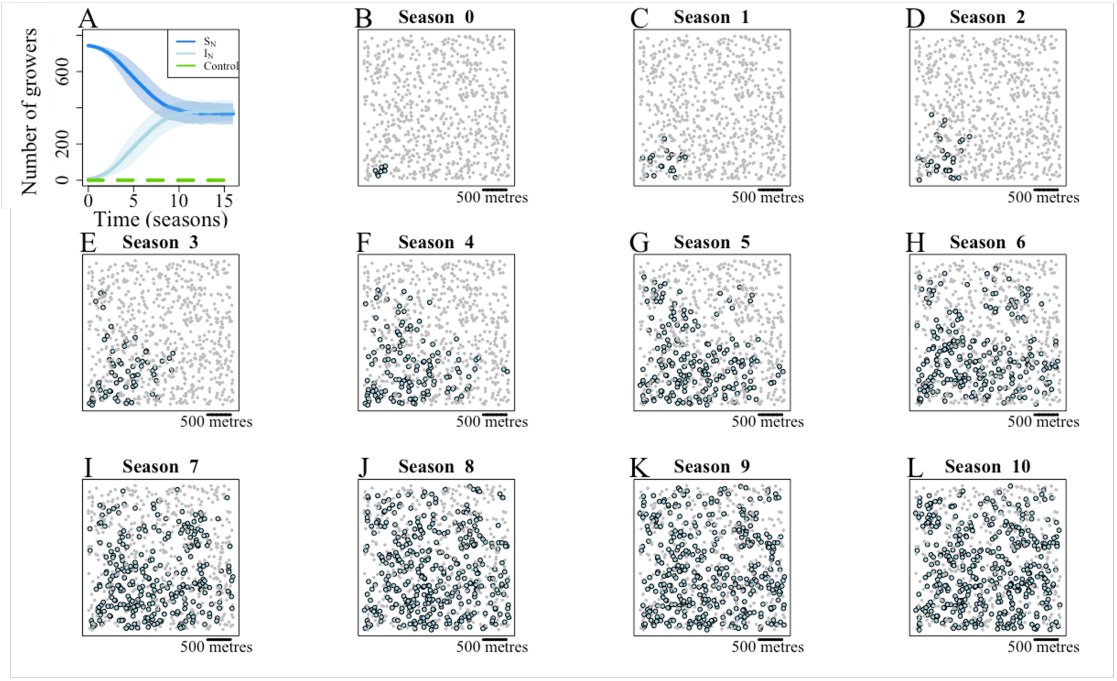

Figure 5.2: **Spatial spread of infection in spatial-stochastic model.** To emphasise the spatial component of infection, vertical transmission has been removed from the model (i.e. $p = 0$). Additionally, we have removed the "control" strategy from the growers as, without vertical transmission, this would have disappeared from the population within five seasons. The light blue dots show infected fields, whilst the grey dots are uninfected fields.

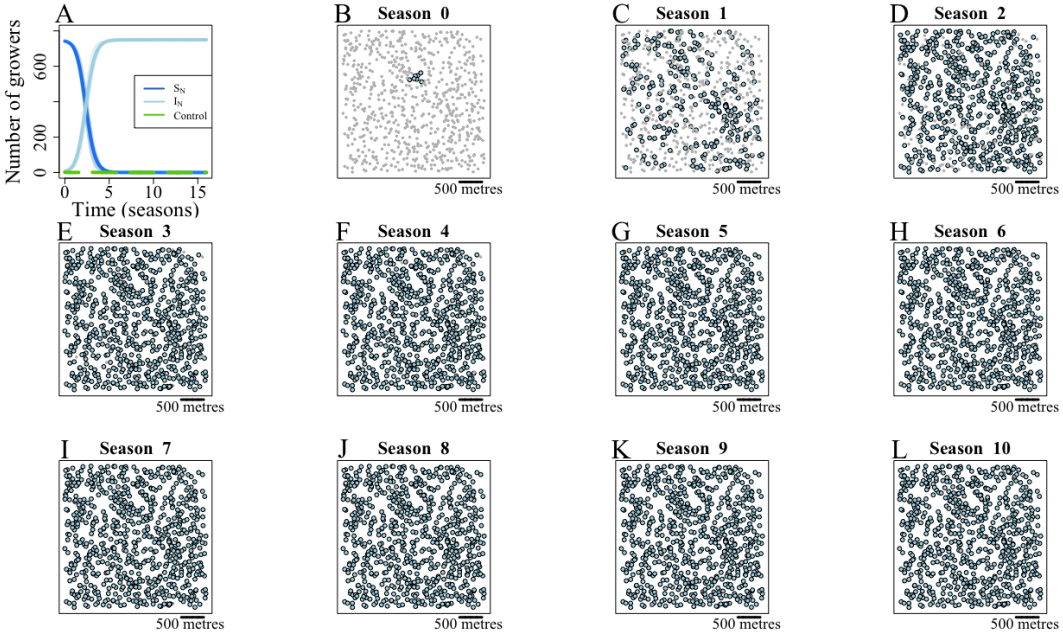

Figure 5.3: **Spatial spread of infection in spatial-stochastic model with vertical transmission.** Here, the probability of vertical transmission is $p = 1$. Additionally, we have removed the "control" strategy from the growers to allow for comparison with Figure 5.2. The light blue dots show infected fields, whilst the grey dots are uninfected fields.

We then investigated how the change in the uptake of the CSS and proportion of infected fields responded to changes in the probability of infection (Figure 5.3.1). When there is no vertical transmission ($p = 0$), there is generally good agreement between the deterministic and stochastic models, though there are fewer controllers in the spatial-stochastic model (17.3% vs 20.2 %) and a higher proportion of infected fields (49.8 % vs 30.1 %; Figure 5.3.1(A) and (B)). However, the model predictions diverge with increases to the probability of vertical transmission, (Figure 5.3.1 (C)); after ten seasons, once $p > 0.35$, less that 3% of growers use the control strategy. This near-complete abandonment of the CSS does not happen at any value of p in the deterministic model within this 10-season time frame (Figure 5.3.1 (D)).

As vertical transmission is not affected by the spatial spread of disease (all growers can trade planting material with all other growers in the landscape), investigations into the clustering of the use of clean seed were not informative, as it was similarly dispersed in the landscape. We therefore limit our investigation of the spatial clustering of control options to the TYLCV model from Chapter 3.

Parameter scans in the spatial-stochastic model

Under the default parameterisation, no grower controls (Figure 5.5(A)), though providing a 50% subsidy encouraged participation in the CSS (Figure 5.5(B)). Disease spreads rapidly between fields due to both the high values of β and ϕ

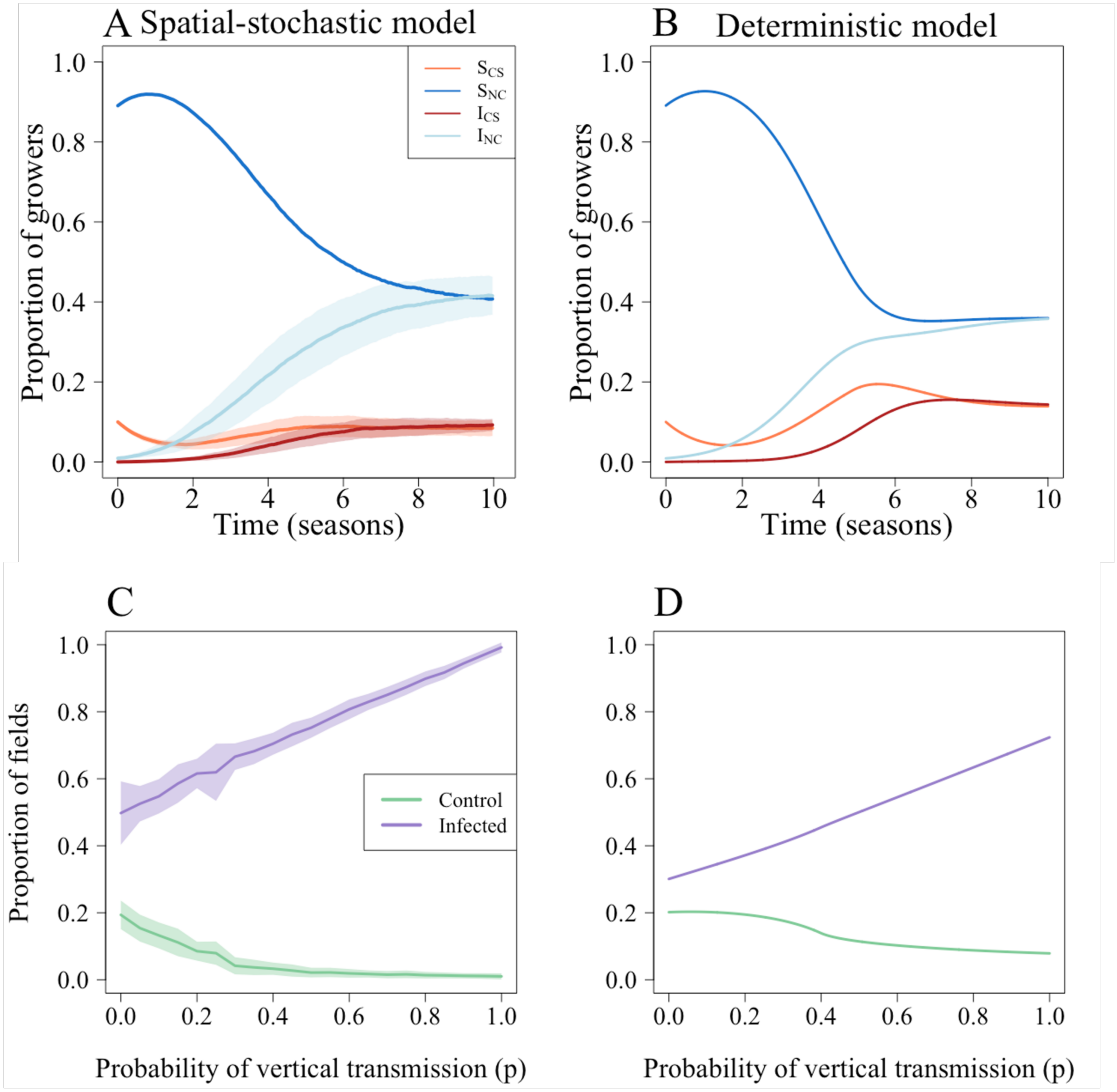

Figure 5.4: **Effect of changing the probability of vertical transmission (p) on the deterministic and stochastic models of CBSD spread.** Epidemic dynamics when there is no vertical transmission in (A) the spatial-stochastic and (B) deterministic models. Though the models broadly agree, there are fewer controllers in the spatial-stochastic model. (C) and (D) show the response of the proportion of controllers and infected fields to changes in the probability of vertical transmission after ten seasons. Growers stop using the CSS at low values of p in the spatial-stochastic model; though it reaches low levels ($< 10\%$) in the deterministic model, use of the CSS persists even at high values of p.

(which discourage control) and the non-spatial aspect of trade, which allows disease to spread far away from its initial source.

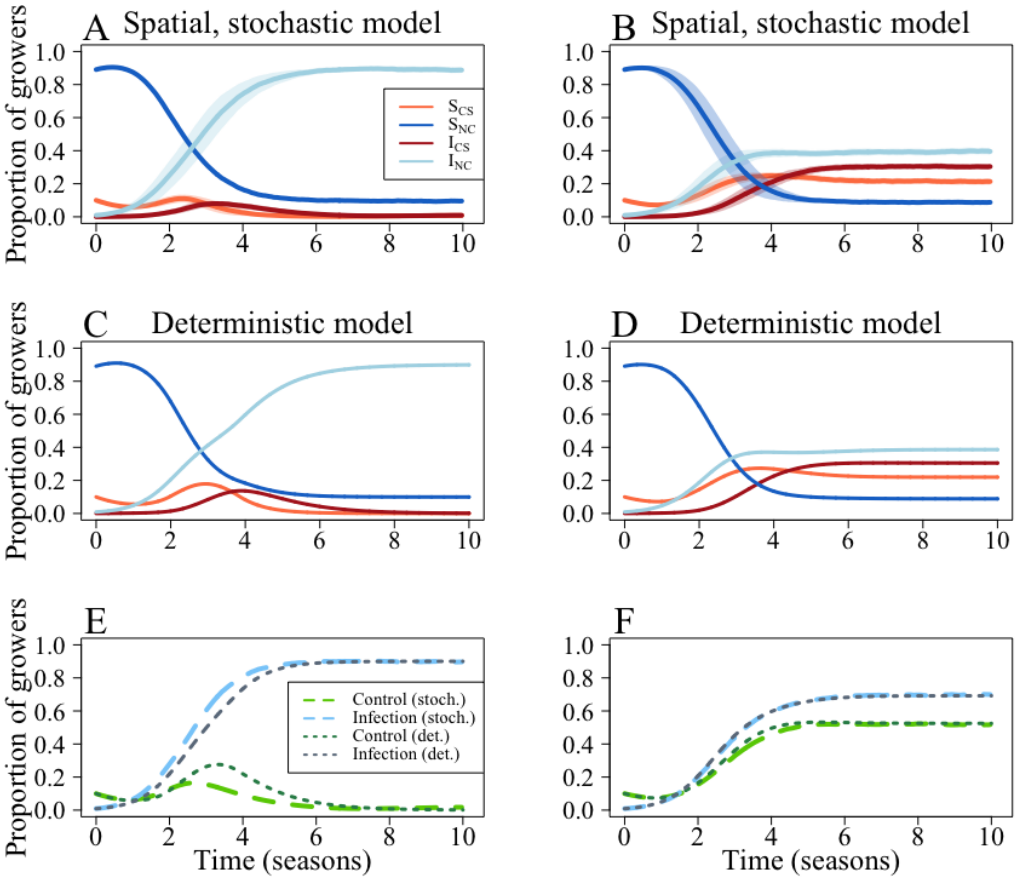

Figure 5.5: **Comparison of the default behaviour of the "grower vs alternative" spatial-stochastic and deterministic models.** (A) Dynamics for the spatial model. As with the deterministic model C, under the default parameterisation, no growers use the CSS after ten seasons. Adding a subsidy in (B) and (D) allows for the two-strategy equilibrium. The figures show the mean for 100 runs of each model, and the error bars show one standard deviation. The equilibrium values and dynamics for spatial (A and C) and non-spatial models (B and D) are very similar in both cases, emphasised in (E) and (F), which show the proportions controlling and infected for the spatial-stochastic ("stoch.") and deterministic ("det.") models. Panel (C) was originally presented in Figure 2.4 (D) in Chapter 2 and is reproduced here for clarity.

For our default parameterisation, and when a 50% subsidy is provided for the cost of control (ϕ), the results of our spatial model are comparable with the deterministic version of the model (Figure 5.6). For equivalent rates of horizontal transmission, the equilibrium values for the deterministic model are similar to the values attained after 50 seasons in the spatial-stochastic model (Figure 5.6 A-B). Disease went extinct in every simulation when $\beta_S = 0$ day^{-1} field^{-1}, and around 4% of the time when $\beta_S = 40$ day^{-1} field^{-1}. Above $\beta_S = 40$ day^{-1} field^{-1}, disease extinction was uncommon (Figure 5.6(A)).

As in Figure 2.7 of the Chapter 2, which shows the analogous result for the non-spatial deterministic model, the kinks in these graphs are a result of susceptible controllers (S_C) growers ceasing to switch strategy (around $\beta_s = 50$ day^{-1} field^{-1} and $\beta = 0.003$ day^{-1} in Figure 5.6(A)-(B)). As the infection pressure increases, these susceptible growers become infected and switch to the non-control strategy, causing an overall decrease in controllers.

In both cases, when the cost of control (ϕ) is varied, at around $\phi = 0.19$, growers should stop using the control strategy (Figure 5.6(C)-(D)). Thus, under these parameterisations, moving to a spatially-explicit stochastic model did not cause the model's outputs to hugely differ from the deterministic model in Chapter 2.

Effect of systematic uncertainty

Growers misestimating the dispersal scale for the whitefly vector (α) has a similar effect to when growers misestimated β in the deterministic model (Figure 2.7 in Chapter 2). As the perceived value of α (q_α, given by $q_\alpha = \nu_\alpha \alpha$, Table 5.1)

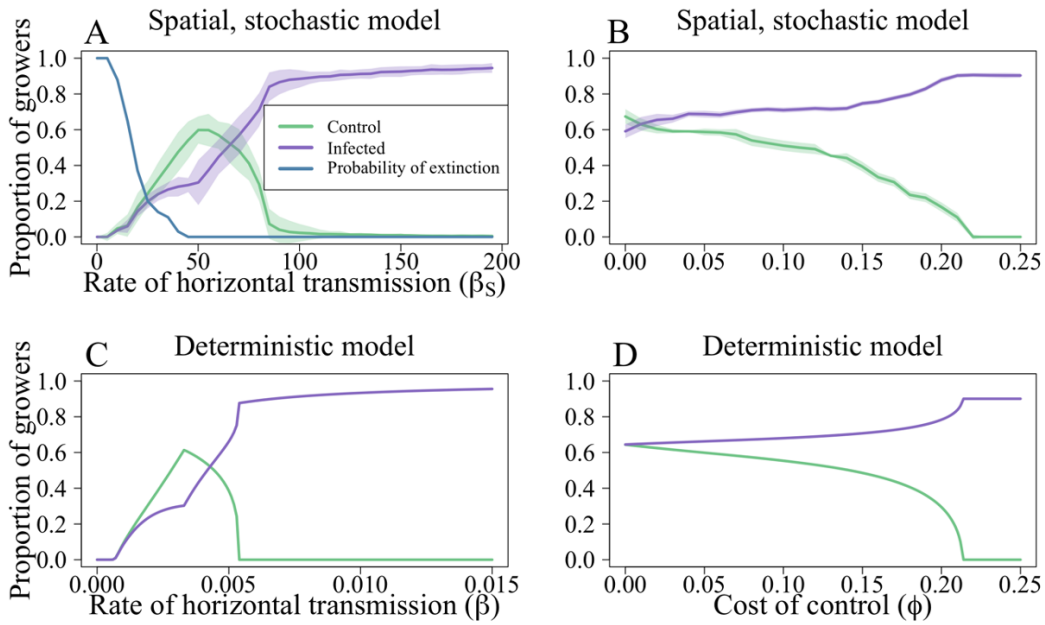

Figure 5.6: **Response to changes in the rate of horizontal transmission and cost of control for the "grower vs alternative" models.** (A) and (C) The proportion of controllers and infected fields after 50 seasons for the spatial-stochastic model and (B) and (D) the equilibrium values of control ($S_C + I_C$) and infection ($I_N + I_C$) for the deterministic model. Aside from the parameters being scanned over, the default parameters are used (Table 5.1 and Table 2.1 in Chapter 2). The results in (A) and (C) closely align with the equilibrium values in the non-spatial model ((B) and (D)), indicating that our results are robust to spatial and stochastic effects. In (A) and (C), the means are calculated over 100 runs, and the error bars show one standard deviation around the mean.

increases, fewer growers participate in the CSS as they estimated that they would be paying the dual penalty of the cost of control and loss due to disease (Figure 5.7).

When growers misestimate the rate of horizontal transmission, β_S, the pattern of CSS use again resembles that of the deterministic model (Figure 5.7(A), compared to the deterministic results in Figure 2.7 in Chapter 2). At low values of the perceived value of β (q_{β_S}, with $q_{\beta_S} = \nu_{\beta_S}\beta_S$, Table 5.1), more growers use

the control scheme as they believe that they are unlikely to be infected. As q_{β_S} increases, growers believe that they are likely to pay the dual penalty of the cost of control, ϕ, and the loss due to infection, L, and therefore abandon the CSS.

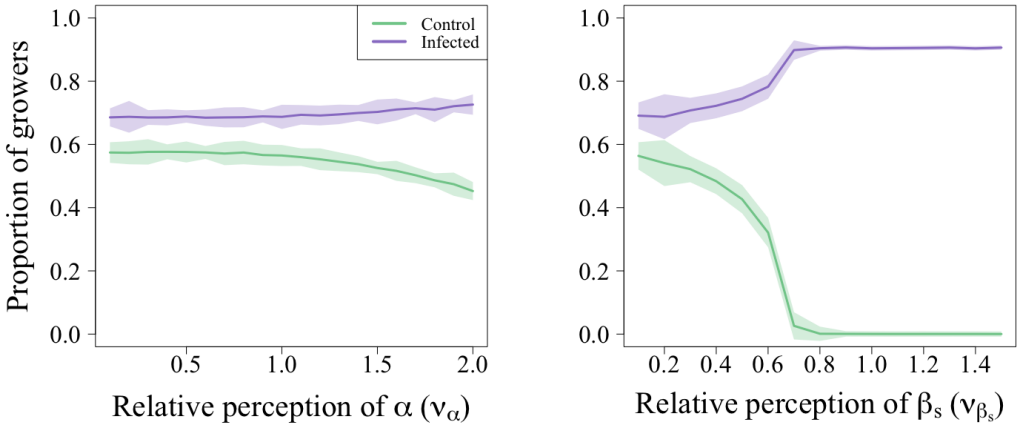

Figure 5.7: **Effect of systematic misestimation in the spatial-stochastic model.** (A) For the default parameters, as the perceptions of the dispersal scale for the whitefly vector (ν_α) increase, fewer growers use the control scheme as they estimate that they would likely end up infected. (B) As perceptions of the rate of horizontal transmission increase (ν_β), fewer growers use the CSS in a pattern that matches that seen in Figure 2.7(A) in Chapter 2. The mean values were calculated over 100 runs, and the error bars show one standard deviation around the mean.

5.3.2 Tomato yellow leaf curl virus

Comparison of deterministic and stochastic models

In the first instance, we compared the deterministic and spatial-stochastic model results for both the tolerant and resistant parameterisations. For the tolerant parameterisation, the steady-state behaviour of the deterministic models with tolerant parameterisation was captured in the stochastic model. However, there were

differences in dynamics approaching equilibrium, with a lower peak in the proportion of fields using tolerant crop in the stochastic model (Figure 5.8(A) and (C)). Nevertheless, in both cases, there was universal adoption of the tolerant crop (Figure 5.8(E)).

Dynamics differed between the deterministic and spatial-stochastic versions of the model when the improved crop was resistant to TYLCV. Though the final values after ten seasons were similar, fewer growers used resistant crop in the stochastic model. This is related to the accuracy of the estimates used by growers to assess the profitability of switching strategy (discussed below; Figure 5.11(A) and (B)).

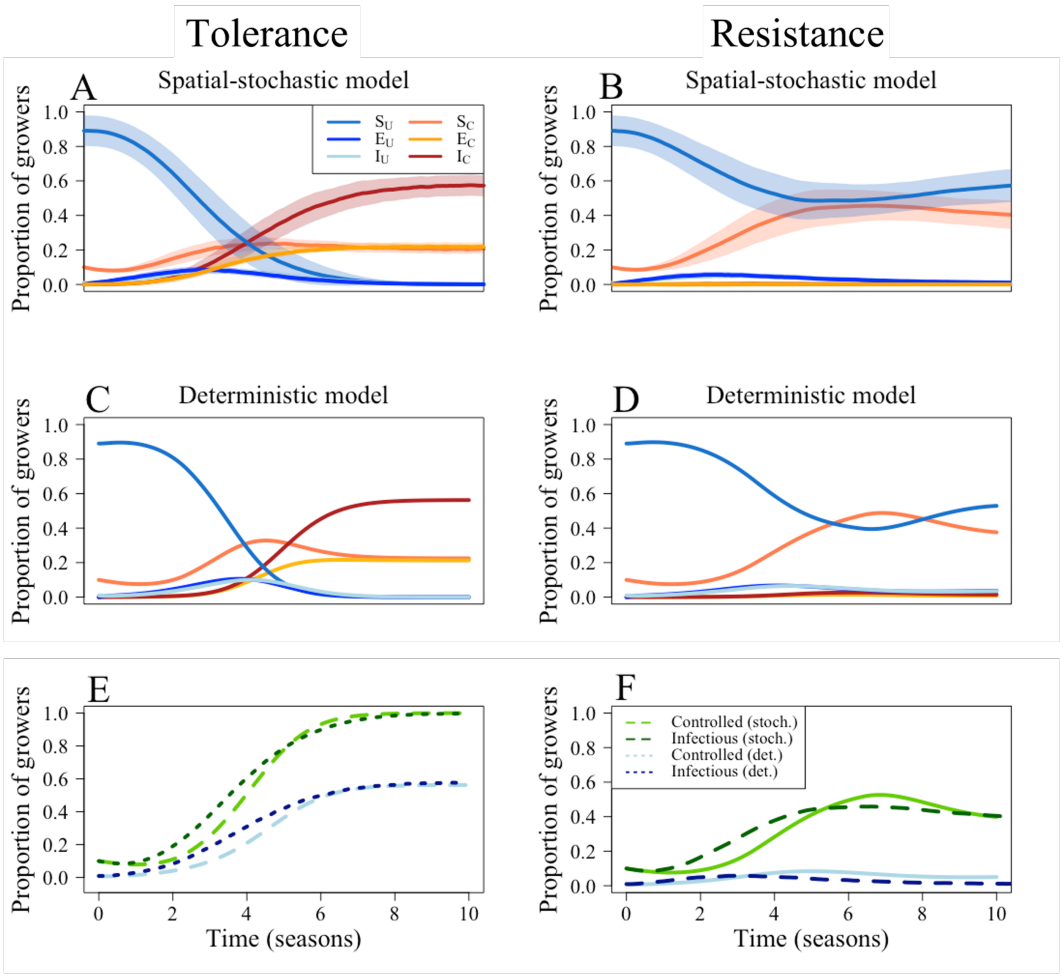

Figure 5.8: **Comparison of the default behaviour of the "grower vs alternative" spatial-stochastic and deterministic models for the tolerant and resistant crop.** (A) Dynamics for the spatial model when improved crop is tolerant. As with the deterministic model (C), there is universal adoption and high levels of infection after ten seasons. (B) Dynamics for the spatial model when improved crop is resistant. Here, the dynamics approaching steady-state differ between the spatial-stochastic and deterministic models, with fewer oscillations in dynamics in the stochastic model. (A) and (B) figures show the mean for 100 runs of each model, and the error bars show one standard deviation. The equilibrium values and dynamics for spatial and non-spatial models are emphasised in (E) and (F), which show the proportions controlling and infected for the spatial-stochastic ("stoch.") and deterministic ("det.") models. In the legend, the subscript "C" denotes whether the crop is tolerant or resistant. For both the tolerant and resistant parameterisations, the steady-state values are the same between the deterministic and stochastic models, though dynamics approaching equilibrium differ.

Effect of changing the parameter values in the spatial-stochastic model

We then investigated the effect of economic and epidemiological parameters on the spatial-stochastic model (Figure 5.9). We do this for two parameters relating to the efficacy of either tolerant or resistant crop (δ_L, which reduces the loss due to infection in tolerant crop, or δ_β, which reduces the susceptibility of resistant crop). Results are shown after ten seasons.

Again, there is good agreement between the spatial-stochastic and deterministic versions of the model where the improved crop is tolerant (Figure 5.9(A) and (C)), though a high value of δ_L leads to lower use of tolerant crop in the spatial-stochastic model. At these values, the benefits conferred by the tolerant crop are not enough to warrant its cost, and as the growers in the spatial-stochastic model are more sensitive to changes in profitability (as they each have an individualised probability of infection), growers abandon tolerant crop quicker in the spatial-stochastic than the deterministic model.

The results for the models where the improved crop is resistant diverge between the deterministic and spatial-stochastic model, though the pattern in the proportion of infected fields is similar. The discrepancy is related to the difference in the growers' estimates of the expected profits and the probability of extinction. After ten seasons in the stochastic model, when δ_β in the majority of simulations (64%), disease goes extinct. However, there is a lag between disease extinction and the cessation of the use of resistant crop by growers; after 50 seasons, growers stop using resistant crop at these low values of δ_{beta} (Appendix D.1).

The use of resistant crop initially increases as δ_β is increased for the deterministic model, as when resistant crop is highly effective, the positive externalities

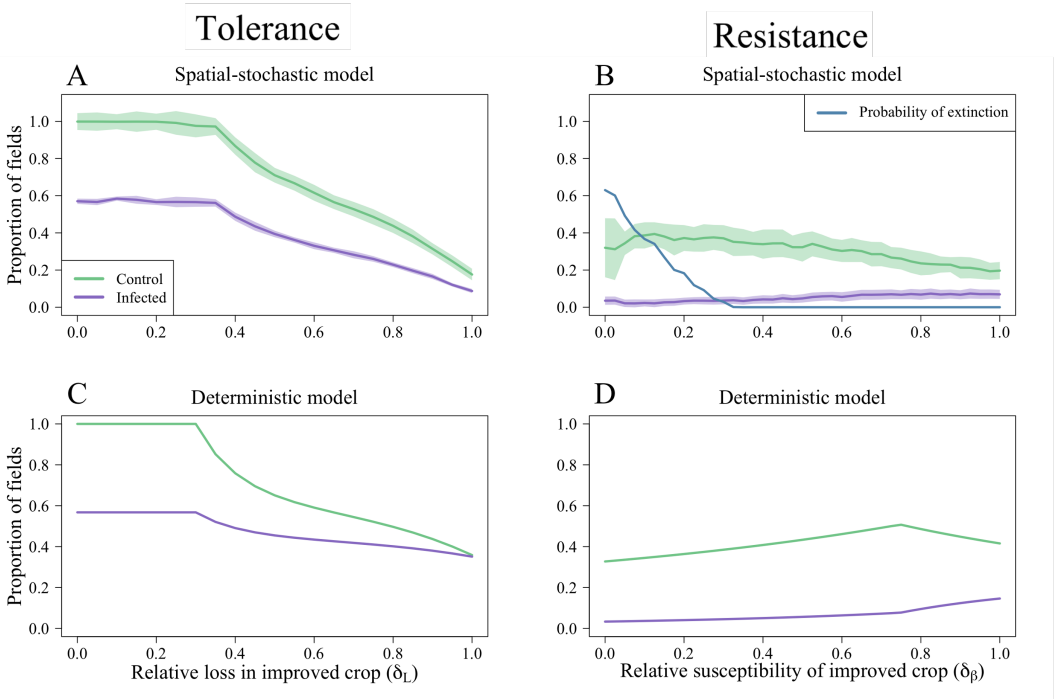

Figure 5.9: **Effect of the change in parameters relating to the effectiveness of tolerance or resistance on the uptake of improved crop.** (A) and (C) are for the tolerant parameterisation, where there is good agreement across values of δ_L (which moderates the losses due to infection in tolerant crop). However, in (B) and (D) though the trend is similar between the spatial-stochastic and deterministic models, more growers use resistant crop at smaller values of δ_β in the spatial-stochastic model; conversely, more growers use resistant crop at higher values of δ_β in the deterministic model. This is due to transient effects, but also the differences in how profits are estimated (Figure 5.10(A) and (B)). Parameters are as in Table 5.2, and initial conditions of the deterministic models are as in Chapter 3 Table 3 (which guarantees the disease-endemic equilibrium is attained in the case of bistability at $\delta_\beta = 0.5$).

it generates are stronger. Therefore, the growers of unimproved crop can benefit without paying for control themselves ("free-ride"). As the resistant crop gets less effective (i.e. δ_{β_L} increases), growers will have to pay to use resistant crop to experience its benefits. Once $\delta_{\beta_L} > 0.79$, however, the use of control is unprofitable and the proportion controlling decreases.

Effect of the radius of comparison

Narrowing the group of growers with whom growers could compare their profits had opposing effects depending on whether the improved crop was tolerant or resistant (Figure 5.10).

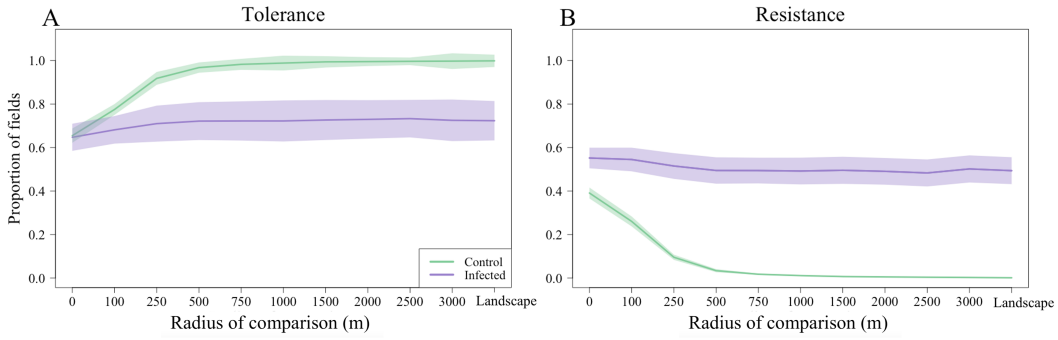

Figure 5.10: **Effect of the spatial scale of the information accessible to growers on the adoption of improved crop and the level of infection.** (A) When improved crop is tolerant, growers are less likely to use it when they have access to a limited range of information. By contrast, when the improved crop is resistant in (B), narrowing the scope of the information by reducing the radius of comparison favours the use of resistant crop.

Growers were most likely to use resistant crop if they only had information about what they did the previous season and compared it to their individual expected profit if they adopted the alternative strategy (i.e. a radius of comparison of "0" m). More broadly, narrowing their pool of comparison, growers only compare outcomes with other growers whose fields are experiencing similar disease pressures. When growers compare their profit with the expected profit of all growers using the alternative strategy, they are also comparing with growers whose fields have vastly different infection pressures exerted upon them. The focal grower, therefore, may over-estimate their risk of infection for the large radii of compari-

son, reducing the perceived benefit of resistant crop and making growers less likely to use it (Figure 5.11(B)). By only comparing with those that are near to them, growers will have a more accurate estimate of their own expected profits (Figure 5.11(B) and (D)).

Conversely, fewer growers use tolerant crop when there is a narrow pool of comparison (Figure 5.10(B)). Tolerant crop only benefits growers if their own field becomes infected, so over-estimating the probability of infection by looking at a broader range of growers incentivises control uptake. Even so, irrespective of the radius of comparison, the estimates for the expected profits were similar for tolerant crop (Figure 5.11(A)). This is in part because, for this parameterisation, there is only a narrow difference between the maximum possible profit achieved with tolerant crop ($P_{SC} = P_{EC} = 0.9$) and the minimum possible profit ($P_{ICH} = 0.84$).

The reduced use of tolerant crop when growers only consider nearby fields is therefore largely because the growers over-estimate the expected profits of using unimproved crop (Figure 5.11(C)). At larger radii of comparison, the estimates fall and become more similar to the "actual" expected profit for each individual field.

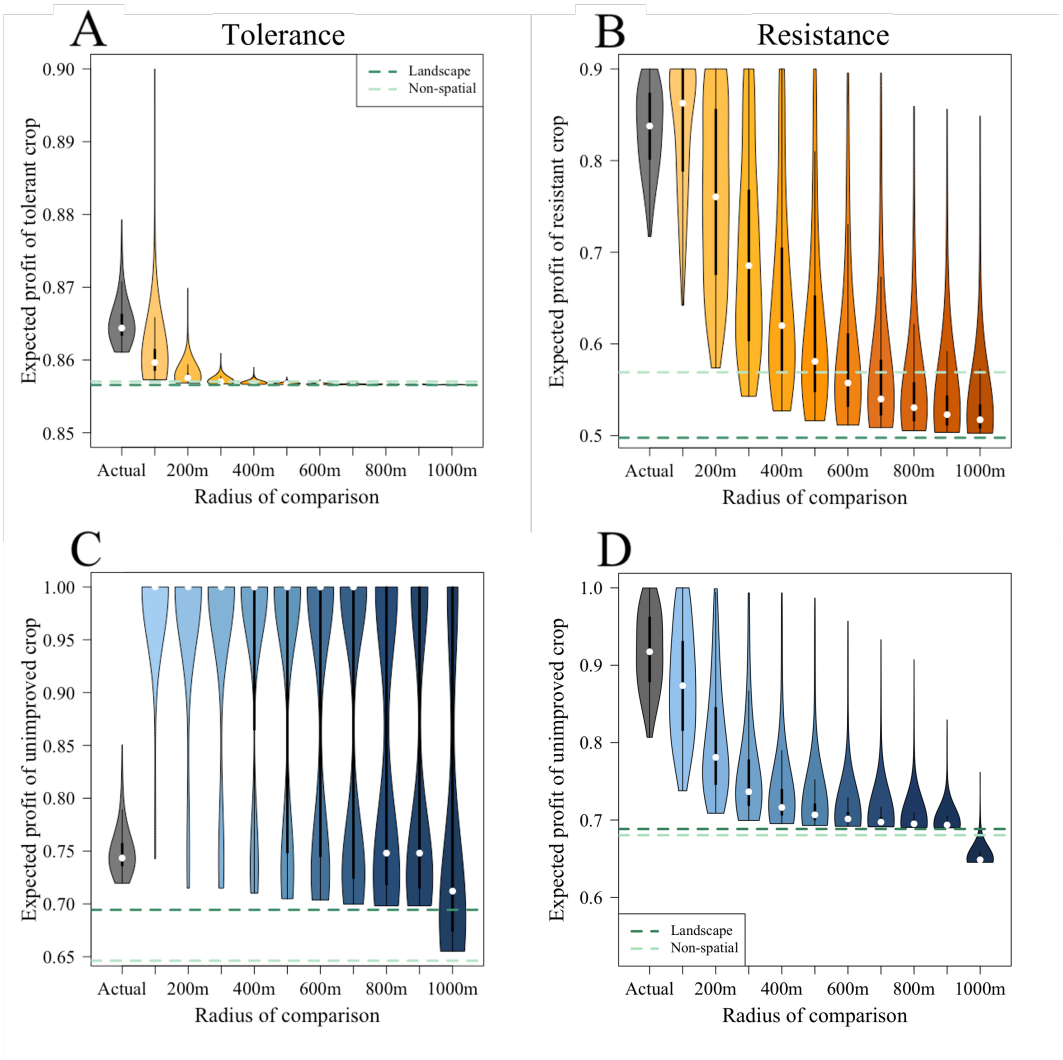

Figure 5.11: **Perception of the expected profits depending on the radius of comparison.**(A) and (C) are when the tolerant parameterisation is used; (B) and (D) the resistant parameterisation. (A) and (C) The estimates are generally closer to the landscape-level estimate and non-spatial estimate when the improved crop is tolerant, though now the landscape-level estimates are less closely aligned with the "actual" profits growers could expect to earn. Growers also over-estimate the expected profit for unimproved crop when they only consider the fields at smaller radii of comparison (C).(B) and (D) The expected profits from the non-spatial (deterministic) model are similar to those for the spatial model for both improved and unimproved crop. Generally, the smaller the radius of comparison, the closer the estimates are to the distribution of the "actual" probabilities of infection experienced by each individual field.

Ensuring accurate information on the local disease pressure encourages sustained use of resistant crop. The same logic does not apply to tolerant crop, as if growers underestimate their disease risk and then pay the dual penalty of infection and the cost of control, the infection penalty is sufficiently low that it does not drastically change the growers' decision to remain using tolerant crop (Figure 5.11(A)). Conversely, if a grower does not use tolerant crop and then is infected, they are much more likely to switch strategy to using tolerant crop the following season.

Spatial clustering of similar control patterns

We evaluated the degree of spatial clustering using Moran's I, a measure of spatial auto-correlation (Moran (1950)). We evaluated the degree of correlation for fields within 200m of the focal growers. There is, therefore, a distinction between the source of information (as growers are evaluating profitability based on growers within 100m, 200m, 300m, 400m, 500m or 600m of their own field) and the scale over which we measure spatial auto-correlation (as we calculate Moran's I based on the similarity in control strategy/infectious status of fields within a 200m radius of the focal field). We do this for after 10 seasons, at which point the epidemic is approximately at steady-state for all radii of comparison.

When the improved crop is tolerant, at lower radii of comparison (100m and 200m), there is initially evidence of dispersion of tolerant fields in the landscape (negative Moran's I). At these radii, the tolerant fields are more spread out in the landscape than expected by chance (so there is no clustering and they are not randomly distributed). There is some clustering of infectious fields at intermediate

radii (200m - 400m); after 10 seasons, at these radii of comparison, the infectious fields will be a mix of tolerant and unimproved crop. As the radii of comparison get larger (> 300m), there is no significant clustering of either tolerant or infectious fields in in the landscape. At this point, the all growers use tolerant crop (Figure 5.10(A)). Therefore, as every field is of the same crop type, there cannot be any clustering of like fields. The majority of these fields will also be infectious, and consequently infection will not be clustered in the landscape.

When the improved crop was resistant (Figure 5.12(B)), there was significant spatial correlation of fields planted with resistant crop across all radii (i.e. fields in close proximity to each other were more likely to be of the same infectious status and have the same control strategy; this is indicated by a positive Moran's I). At smaller radii of comparison, there was also clustering of infectious fields, though these became more randomly distributed in the landscape as the radius of comparison got larger. This is probably due to the decrease in the number of resistant fields (Figure 5.11(B)); with fewer resistant fields, infection becomes more dispersed in the landscape and is not clustered in particular localities.

At lower radii of comparison, growers are only comparing with other fields that are experiencing similar disease pressures. Therefore, if it beneficial for a nearby grower to use resistant crop, it will likely be beneficial for the focal grower, leading to a clustering of resistant fields. As the radius of comparison expands, the growers will begin comparing with those that have different disease pressures, reducing the likelihood that they will perceive resistant crop as beneficial (Figure 5.11(B)).

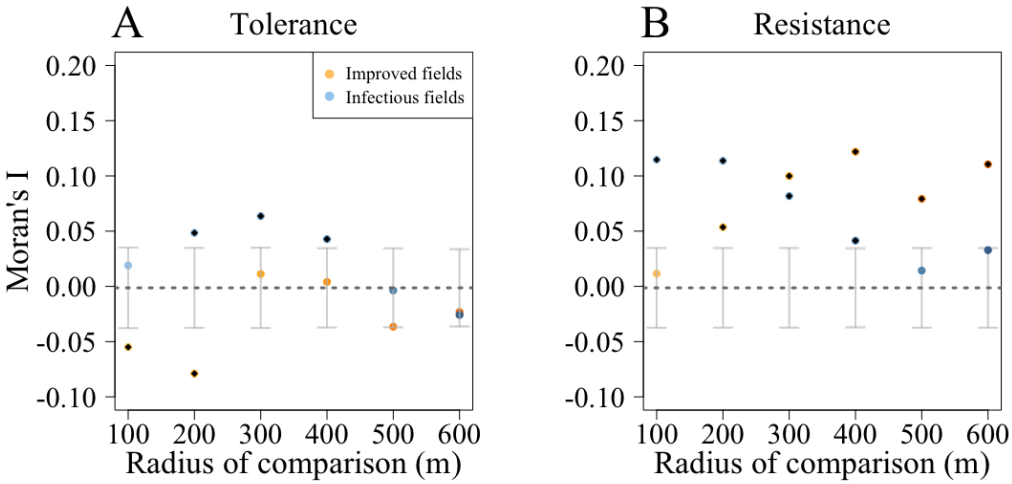

Figure 5.12: **Moran's I after 10 seasons for different radii of comparison.** The black dots indicate values that are statistically significant. (A) At smaller radii of comparison, the tolerant crop is dispersed in the landscape. At 300m, there is no significant correlation in the location of tolerant fields. (B) Across all radii, when the improved crop is resistant, there is significant positive spatial correlation. At small radii, this also applies to infectious fields, though as the radius increases the clustering of infectious crop disappears. The grey bars show the 95% confidence interval around the expected value ($E = -1/(N-1)$). For a sample time course for both resistant and tolerant crop, see Appendix D.1.

5.4 Discussion

5.4.1 CBSD and CSS

The results for the spatial-stochastic model of clean seed systems cassava brown streak disease (CBSD) were comparable with those for the equivalent deterministic

model described in Chapter 2 (Figure 5.6), demonstrating the robustness of our results to stochastic and spatial effects. This was partly due to the non-spatial aspect of trade, as it allowed for transmission across the relatively small landscape considered here (McQuaid et al. (2017b)).

When trade was not included (i.e. $p = 0$) the disease spread across the landscape in a more "wavelike" pattern as expected from the thin-tailed dispersal kernel for the whitefly (Shaw (1995)) (Figure 5.2). However, when trade occurs via a market or central organisation, most transactions occur over a scale larger than our landscape (a square with sides 3.16 km), with around 70 % of transactions occurring over a scale of 10-50 km (Szyniszewska et al. (2021)). For more informal trade settings, with growers interacting with each other, a proximity-based kernel may be more appropriate (McQuaid et al. (2017b), McQuaid et al. (2017a); though both of these were modelled over a larger landscape than ours). However, such exchanges are hard to parameterise as the probability of exchanging planting material is highly variable across settings (varying between 30 - 92.51% over unspecified spatial scales; Ntawuruhunga et al. (2007), Chikoti et al. (2016), Djaha et al. (2018), Houngue et al. (2018) and Teeken et al. (2018)). For this reason, we only investigated the effect of different networks of comparison on the TYLCV model, which does not include any trade-mediated spread of the virus.

As in Chapter 2, we investigated the effect of growers' misperception of epidemiological parameters on their participation in the CSS. Results were similar to those found in Chapter 2; if growers underestimated the probability of infection (either by underestimating the rate of horizontal transmission, β_S, or the whitefly's dispersal scale parameter, α), they were more likely to control. At these low

levels, the growers did not believe there was a high probability that they would receive the "sucker's payoff" and pay the dual cost of control and infection. The high probability of vertical transmission ($p = 0.8$) preserves the perceived benefit of control. As the estimates of both β_S and α increase, the perceived benefit of control decreases and participation in the CSS decreases.

5.4.2 TYLCV model

The robustness of our results from the deterministic models described in Chapter 3 depended on whether growers could choose to use tolerant or resistant crop. There was better agreement between deterministic and stochastic tolerance models than for the equivalent resistance models (both for default behaviour and response to parameters). Though the general patterns were the same, uptake of the resistant crop was generally lower in the spatial-stochastic model (Figures 5.8 and 5.9).

In the spatial-stochastic model, growers' use of highly-effective resistant crop could lead to elimination of TYLCV (Figure 5.9(B)). Though very low levels of disease could be achieved in the deterministic model (Figure 5.9(D)), it could not go extinct. Disease elimination was never possible with tolerant crop, as tolerant traits do not limit the infectivity or susceptibility (Figure 5.9(A) and (C)). The possibility for disease extinction led to discrepancies between the spatial-stochastic and deterministic models when resistant crop was used. Additionally, there were larger differences in the growers' estimates of the expected profits of each strategy (Figure 5.11(A) and (B), "landscape" vs "non-spatial" lines). Ensuring growers have accurate estimates of their probabilities of infection, rather than an average probability of infection obtained from a non-spatial model, can help incentivise the

use of resistant crop as a means of control. Growers only use control when needed, and they can avoid paying unnecessarily for protection based on probabilities of infection of fields on the other side of the landscape.

As this spatial-stochastic model was more sensitive to the effects of space, we investigated whether narrowing the pool of comparison growers used to estimate profits affected the uptake of control. The radius of comparison had opposite effects for each grower type. A smaller radius of comparison (0 - 400m) discouraged the use of tolerant crop (Figure 5.10(A)). At these lower radii, growers overestimated the profitability of unimproved crop (Figure 5.11(C)), encouraging its use. Conversely, when growers can use resistant crop, the landscape-level information (where growers have information on all other growers) under-estimates the profitability resistant crop (Figure 5.11(A)). Consequently, more growers use resistant crop when they only compare with growers in their close vicinity (Figure 5.10(B)). The distinction between these two results is important, as it shows that crop types with different externalities may require different levels of information to encourage their use.

We found no evidence that growers of tolerant crop cluster in the landscape, though there was evidence that growers of resistant crop did (Figure 5.12(A) vs (B)). Tolerant crop provided more benefits to growers and its use was widespread in the landscape irrespective of the radius of comparison used (Figure 5.10(A)); its widespread use therefore precluded any clustering. As the use of resistant crop was less widespread, and its use was strongly influenced by the radius from which the focal grower accessed information, clustering was possible. The clustering of growers of resistant crops is similar to the effects observed in Salathé & Bonhoef-

fer (2008) and Eames (2009), where clusters of individuals with similar opinions regarding vaccination occurred.

5.4.3 General conclusions of developing spatial-stochastic models

Broadly, the conclusions we obtained from the deterministic models used in Chapters 2 and 3 are transferable to the models described here, with notable exceptions being the spatial-stochastic version of the TYLCV model where growers could choose between resistant and tolerant crop. We have shown, then, that the use of simpler, more mathematically-tractable and less computationally-intense models is therefore justified in some situations. In particular, the use of spatial-stochastic models should be considered when a control scheme generates strong positive externalities that disincentivise widespread adoption or that can lead to disease extinction.

There are several limitations to the models we have developed in this chapter. Alongside the general criticisms described in Chapters 2 and 3, here we discuss some explicitly related to these models' spatial and stochastic components. We have not incorporated any environmental stochasticity, which would influence disease spread across the landscape. We have also considered fields of a uniform size that are randomly distributed in the landscape; in reality, the field size will vary and the fields may be clustered together and not evenly dispersed (e.g. around roads or in groups based on the social contacts of the growers).

Additionally, the yield loss for an infected field is constant irrespective of when

the field was infected during the season. Allowing for within-field bulk-up of infection will change the nature of the payoffs and may help incentivise control. This is particularly true in the case of the CSS, as the lack of initial infection from replanting infected cuttings will exacerbate the differences in expected profits between the control strategies. The size of a field, which here we've assumed to be uniform across all growers, will also impact their profits. Finally, once disease goes extinct, it cannot be reintroduced into the system, eliminating the likelihood of control (as growers cannot act irrationally and cannot predict a reintroduction event). These limitations provide ground for future work, discussed in Chapter 6.

Chapter 6: Discussion

Including human behaviour in epidemic models remains rare, and it is a very recent development in the field of plant disease epidemiology. In this thesis, we attempt to address this oversight by modifying existing disease-behaviour models (such as those presented in Milne et al. (2016), McQuaid et al. (2017a) and Saikai et al. (2021)) to simpler, non-spatial models. We develop and test a number of different behavioural models, and then deploy them in different management contexts to test the interactions between disease spread and grower decision-making.

6.1 Summary of thesis

By combining epidemiological and game-theoretic models, we demonstrated how grower decision-making can be included in plant disease models. In Chapter 2, we considered cassava brown streak disease (CBSD) as a case study. CBSD is a viral disease that is widespread across sub-Saharan Africa, and provided a useful case study for investigating behaviour because of how it is transmitted. CBSD has two main routes of transmission: it can be vectored between fields by *B. tabaci*, introducing an inter-dependence between growers as the infection status of their

field will depend on that of neighbouring fields. Additionally, its second transmission route (vertically via the replanting of infected cassava cuttings) introduces a behavioural component, as growers can chose whether or not to trade cuttings and potentially receive infected material.

We used this case study to investigate how the potential increase in profits due to disease management influences participation in clean seed systems (CSS). Our models were rooted in game theory, with growers making strategic decisions based on the expected profitability of different control strategies. We found that each behavioural model ("strategy vs population", "strategy vs alternative", "grower vs population" and "grower vs alternative") led to varying participation in the CSS and, consequently, epidemic outcomes. Each model had a different response to changes in parameter values, and the "all control" equilibrium (where all growers used the CSS) was not achievable for our "grower vs" behavioural models. Though in this chapter we investigated the effects of all four behavioural models, for the majority of the thesis we focused on the "grower vs alternative" model as this had the best experimental and empirical evidence supporting its use (Kaup (2008), Milne et al. (2016), McQuaid et al. (2017a), Saikai et al. (2021)).

By considering systematic misestimation of parameters by growers, we also found that the perception of epidemiological quantities influenced long-term participation in the CSS. Over-estimation of infection risk led to lower participation in the CSS, as growers perceived that paying for the CSS will be futile.

Additionally, even though good disease management would be achieved through the implementation of CSS, and a scenario where all controllers use the CSS was achievable in the "strategy vs" models, CBSD was rarely eliminated from the sys-

tem. This was primarily due to within-season spread mediated by *B. tabaci*, which was not controlled by the CSS. These results were robust to stochastic and spatial effects (Chapter 5). Our work highlighted the importance of including human behaviour in plant disease models, but also the significance of how that behaviour was included.

In Chapters 3 and 4, we considered how the deployment of disease-resistant or -tolerant varieties influenced the disease management strategies of growers. Resistant varieties are less susceptible and, therefore, less likely to act as reservoirs of inoculum, whereas tolerant varieties can be highly susceptible but limit yield loss for those who grow them. To date, the population-scale effects of deploying resistant or tolerant varieties have received little consideration from epidemiologists. The traits are often confounded by crop companies, particularly when considering quantitative resistance and partial tolerance. In Chapter 3, we examined how tolerant and resistant crops had opposing consequences upon the uptake of control using a behavioural model based on strategic-adaptive expectations (the "grower vs alternative" model introduced in Chapter 2). Growers compared last season's profit with an estimate of what could be expected from the alternative crop type ("grower vs alternative"), thereby assessing whether to alter their strategy for the next season. Tolerant crop only benefited growers using it, and because it was not rogued as effectively, its use decreased yields for other growers by allowing for a high build-up of infection. This incentivised widespread use via a positive feedback loop.

Resistant crop provided benefits to controllers and non-controllers, as the reduced population-scale disease pressure led to increased yields for all. However,

this positive externality allowed growers who do not deploy resistant crop to "free-ride" upon the management efforts of others. The results presented in this chapter highlighted how a community of growers responds to the contrasting incentives caused by tolerant and resistant crop varieties and how this led to distinct effects on yields and population-scale deployment.

Then, in Chapter 4, we extended the two-strategy models explored in Chapters 2 and 3 to include a third option. Growers could now choose between three different crop varieties: disease-resistant, disease-tolerant or conventional crop. We also introduced the concept of the "social planner", an individual who could provide subsidies to growers to incentivise or disincentivise the use of particular crops. This, however, introduced conflicting goals: which subsidisation regime maximised growers' profits whilst also minimising the cost to the planner? We solved this problem using Pareto optimisation, and found that growers achieved the highest profits at the lowest cost to the planner when subsidies were provided for resistant crop. Almost no scenarios where the tolerant crop was subsidised led to a Pareto-optimal solution. This was because tolerant crop induces a positive feedback loop, encouraging widespread use (i.e. tolerant crop is a "strategic complement"), though the benefits are only felt privately by each grower. By subsidising resistant crop, however, planners could subsidise only a subset of the population whilst still ensuring that all growers, irrespective of their crop type, achieved high profits. This showed a conflict between what is privately optimal for growers (growing tolerant crop) and what is socially optimal when a planner is considered. However, even within this regime of discouraging tolerant crop, there are a variety of possible optima with different outcomes for the planners and growers. Pareto optimisation cannot determine which of these scenarios is better,

so the social planner will need external information to decide (such as budgetary constraints, the economic and political climate, the importance of the crop *etc.*). Though we have used different crop types, the principles of Pareto optimisation can, in principle, be applied to any control scheme.

Finally, in Chapter 5, we developed spatial-stochastic analogues of the deterministic models described in Chapters 2 and 3. The conclusions drawn from our spatial-stochastic model of the CSS and CBSD were essentially identical to those obtained from the deterministic model in Chapter 2. This was primarily due to our inclusion of trade-mediated vertical transmission, which occurred between all non-controllers with equal probability (and therefore had no spatial aspect). The trade-mediated transmission caused the rapid spread of CBSD in the landscape, overwhelming the whitefly-mediated spread whose rate depended on the distance between infected and susceptible fields. Future work could include network-based trade schemes that would introduce a spatial aspect to CBSD spread via vertical transmission.

However, the results from the spatial-stochastic version of the model presented in Chapter 3 (which modelled the deployment of TYLCV-tolerant or -resistant tomato cultivars) revealed important nuances from the spatial-stochastic context, though differences between the models were primarily due to temporal effects. The positive feedback loop induced by the use of tolerant crop in some fields meant that, for the tolerant parameterisation, results were comparable between the deterministic and spatial-stochastic models. If the tolerant crop was sufficiently cheap and the disease pressure sufficiently high, all growers adopted tolerant crop. The benefits conferred by the tolerant crop meant that even at relatively low

probabilities of infection (where the grower risks unnecessarily paying the cost of control), growers would still achieve higher profits using tolerant crop rather than risk incurring a large yield loss with unimproved crop.

However, the results diverged when we used the resistant parameterisation. The resistant crop only protected from initial infection, with growers sustaining a high yield loss if the resistant crop becomes infected. In the spatial-stochastic model, each field has its own probability of infection. This meant that growers could use local levels of infection to assess when they are likely to be paying the "sucker's payoff" or when the cost of control is justified. Additionally, the spatial-stochastic models allowed for disease extinction. As the resistant crop reduced the effective reproductive number of TYLCV, disease extinction was likely to occur when resistant crop was used. Tolerant crop's inability to reduce disease transmission meant that extinction events were rarer, though not, in principle, impossible.

The complexity of the behavioural model with an expanded strategy set, coupled with time constraints, prevented us from developing a spatial-stochastic analogue of the model presented in Chapter 4, though in principle such a model could be constructed. We imagine, as with the TYLCV models from Chapters 3 and 5 upon which Chapter 4 is based, the results would be broadly similar between the spatial-stochastic and deterministic models. The response to changing the pool of comparison may be comparable with those presented in Chapter 3. As with the resistant parameterisation in Chapter 3, there may be an increased uptake of resistant crop when growers narrow their pool of comparison, and decreased use of tolerant crop.

Though the spatial-stochastic models presented in Chapter 5 were analogues of our deterministic models, as with those deterministic models, the spatial set-up and way in which growers make decisions are sufficiently flexible to be adapted to other pathosystems or control mechanisms.

6.2 Contributions to the literature

Previous models that include grower behaviour have included spatial, stochastic, and network-based components (Milne et al. (2016), McQuaid et al. (2017a), Saikai et al. (2021)). We simplified these complex landscapes in which decisions were being made to a non-spatial deterministic model, allowing us to focus on the behavioural components of our models. These non-spatial models are less computationally intensive and more mathematically tractable, allowing more general conclusions on the interaction between behaviour and epidemic outcomes to be drawn.

We then developed four different decision rules. These decision rules were either based on rational (our "strategy vs" models) or strategic-adaptive (our "grower vs" models) expectations. We then systematically compared these decision rules, determining how they impact grower behaviour and, ultimately, epidemic outcomes. To our knowledge, this is the first time such a comparison has been undertaken for epidemiological modelling. The differences we found resulting from each decision rule highlight that, when including grower behaviour, greater consideration must be given to how decision-making is modelled as this will *a priori* affect outcomes. However, in the absence of richer data - particularly in terms of growers' risk attitudes, access to information, the relative importance of the profits of others and

how they consider the weighting of past outcomes - each of these decision rules remain simplified representations of how growers may make decisions relating to disease management.

Where decision rules of similar forms have been included before, the settings have been complex spatially-explicit models and the studies have not included an in-depth analysis of their behaviour (Milne et al. (2016), McQuaid et al. (2017a), Saikai et al. (2021)). Here, we analysed the response of these switching rules to different parameter values and disease pressures, enabling a greater understanding of the drivers of grower behaviour. This analysis, alongside our work comparing the "strategy vs" and "grower vs" models, will help guide future models incorporating human behaviour and plant disease management to ensure the appropriate decision rules are chosen for a given context. In the absence of data on how growers might behave, the results obtained from each decision rule can provide a range of possible outcomes.

In this thesis, we also considered the effect of the externalities generated by each control mechanism. These externalities have significant repercussions on the success of a control scheme across a community of growers. Positive externalities, such as those generated by resistant crop, disincentivised control and could lead to suboptimal profits for growers.

In the context of tolerant crop, negative externalities, by contrast, actually incentivised the use of control. Although this led to high disease levels, profits improved for growers as their yield loss was limited. Providing subsidies to internalise some of the positive externalities generated by resistant crop can help to recover the growers' profits. Moreover, knowing in advance the nature of the

externalities generated by a particular control mechanism can help predict its uptake and what actions may be needed to ensure its success. Subsidies have been proposed as a means of internalising the externalities generated by vaccinations, which - like resistant crop - generates positive externalities that disincentivise wide uptake (Francis (2004), Ibuka & ichiro Bessho (2015)).

In this thesis, we have also shown including behaviour results in similar dynamics in both deterministic and their analogous spatial-stochastic models. There were some notable differences, however, particularly when considering the case where growers can choose between resistant and unimproved crop. Generally, however, we have shown that the simpler, more tractable deterministic models can make robust predictions about the influence of grower behaviour on disease management.

6.3 Limitations and scope for future work

Despite decades of work in human disease epidemiology, incorporating behaviour into plant disease models is a relatively recent development. Though, in this thesis, we have expanded on existing models, there are also some ways in which the treatment we have adopted here is limited. This leaves a broad range of possibilities that could be incorporated into future models.

6.3.1 Limitations to the game-theoretic approach

There are a number of limitations to the game-theoretic models that are used to model disease-avoidance behaviours (including those used in this thesis). Like many economic models, they assume perfect rationality of the player (Morris (2012)). However, empirical studies have shown that there is often a substan-

tial deviation from the predicted optimal equilibrium state (Kasthurirathna & Piraveenan (2015), Oraby et al. (2014), Camerer (2011), Shim et al. (2012)). One possible explanation is that the players are irrational in their decision making, though they may also be limited by their ability to perceive risk, access to information, or have previous experience that will influence their decisions. The assumption that players are rational has been addressed in some studies by including a parameter that ensures some players will act contrary to what is rationally good (McQuaid et al. (2017a), Poletti et al. (2012), Poletti et al. (2009)). Conversely, individuals may be acting within the limits of "bounded rationality": they believe they are behaving rationally, but are constrained by their own mental capabilities and access to information (Simon (1990), Jones (1999), Tsutsui et al. (2010)). Some models have explicitly included these constraints (Oraby & Bauch (2015), Shi et al. (2019)). Inclusion of these effects on individual decision-making changes the long-term predictions of these models; for example, including bounded rationality in Oraby & Bauch (2015) increases the predicted proportion of individuals who will get vaccinated, though means it is harder to eliminate disease when vaccines are not mandatory. Deviations from perfectly rational behaviour, then, could influence the policy recommendations derived from disease models.

Even if an individual acts within the bounds of rationality, their attitude to risk may also affect their decisions (Buchak & Buchak (2013), Stefánsson & Bradley (2019)). An individual's "risk attitude" describes where they fall on a spectrum between risk-tolerant and risk-averse (Zhang et al. (2014)). In the case of plant disease management, if an individual is risk-tolerant, they do not readily engage in actions that protect themselves from the threat posed by disease. The converse is true for risk-averse individuals; any control actions they undertake are proactive

and aim to prevent any infection from occurring rather than reactively minimise the impact of infection. As an intermediate, there are risk-neutral individuals who neither under- nor overestimate risk but rather adhere to behaviours predicted by assuming perfect rationality.

Risk sensitivity will depend both on circumstance and prior experience (Bucini et al. (2019), Mankad (2016), Hidano et al. (2018), Garcia-Figuera et al. (2021)). For example, even when banana growers perceived there to be a high risk of infection of Panama Tropical Race 4 (*Fusarium oxysporum* f. sp. *cubense (Foc), TR4*), many did not actively engage in preventative measures because they were not highly dependent on banana as their main source of income (Mankad et al. (2019)). Risk attitudes, therefore, do not have a clear impact *a priori*, as their effects depend on whether the individual is more averse to the costs associated with infection or those associated with preventing infection.

We have also assumed homogeneity in terms - amongst other things - of the growers' economic resources, dependence on their crop, access to the control mechanism and willingness to trust the information they receive. In effect, any part of our models that assumes that growers will receive the same information, and act upon it in the same way, is a simplification of reality.

Despite some limited investigations into parameter mis-estimation in Chapters 2 and 5, we also assume that individuals can all interpret epidemiological information to get an exact probability of infection upon which they base their decisions. This is over-simplified on two counts: growers may not be able to access or interpret information relating to disease prevalence, but they also may not use it as the factor upon which they make their decisions. Each of these assumptions could be

improved with additional data collection, or extensions to our spatial-stochastic model, which allow for more individualised behaviours between growers.

Though the field of economic epidemiology connects human behaviour and disease spread, models may temporally disconnect the two processes. In the canonical example of the vaccination game (Bauch & Earn (2004), Box 1 in Introduction), the payoffs for the vaccination and non-vaccination strategies are calculated when the disease dynamic component of the system is at steady-state. The individuals then decide whether to vaccinate, and the disease component of the model is allowed to equilibrate again until the entire system reaches a steady-state. Delabouglise & Boni (2020) and Brettin et al. (2018) similarly ignore transient effects of disease dynamics and the replicator dynamics that govern individual behaviour takes place when the disease is at steady-state. Acknowledging that disease spread and human behaviour may occur on different time scales, Poletti et al. (2009) and Karlsson & Rowlett (2020) include parameters that modulate the relative speed of both dynamics. In our models, the "responsiveness" parameter (η) in some ways acted as a rate parameter, as it set how sensitive growers were to unit differences in profit (which is related to their risk attitude). Our use of η was constrained by the lack of data pertaining to growers' risk attitudes, and for simplicity we assumed a constant value across all simulations and growers. More nuance could be added; for example, growers who use control may have a different degree of responsiveness than non-controllers, or in our spatial-stochastic models each individual grower could have their own value of η.

Other models use seasonal dynamics for disease (e.g. Vardavas et al. (2007) and Fukuda et al. (2014)). In these models, all individuals decide on vaccination

at the beginning of the epidemic season and cannot change their strategy until the beginning of the next epidemic season. Whilst such model structures may apply to crop epidemic systems (and are used in Milne et al. (2016), McQuaid et al. (2017a), Saikai et al. (2021)), where fields can only be planted once, and there are legislative constraints on the timing of chemical controls, the restriction of vaccinations to the very beginning of an epidemic is unlikely in human biology. Future models in human epidemiology could consider the impact of this mismatch in temporal scales and further integrate the two processes. In our model, though we used continuous time, growers' behaviours were similarly limited and their decision-making was restricted to the time of planting (though planting was asynchronous between growers). Moving to discrete time may induce more oscillations in uptake, as was seen in Milne et al. (2016) and McQuaid et al. (2017a).

Additionally, even within the field of plant epidemiology, the seasonal timing does not allow for asynchronous decision-making between individuals. In this thesis, we directly incorporate growers' decisions into disease models, avoiding complications related to the relative scales of decision-making and disease dynamics. Decisions are based on the current disease prevalence and occur at the time of planting without any delay between the decision-making and the action being carried out. Further research should examine the comparative impact of synchronous and asynchronous decision-making between individuals.

In both of our case studies, we were limited by a lack of data relating to our models' economic, behavioural, and epidemiological parameters. In some cases, we could somewhat compensate for this lack of data through extensive scans over parameter space, but others - particularly those parameters pertaining to

the spatial-stochastic models - were approximated from previous literature. For the distribution of fields in the landscape, we approximated the density from McQuaid et al. (2017b) for both our CBSD and TYLCV models and did not vary it between simulations. Yet fields may be clustered around features such as roads or neighbourhoods based on family connections. We also had no data on how information regarding disease risk is communicated between growers; our models effectively assume it is "broadcast" across the landscape, and each grower has an equal likelihood of receiving it. But complex structures are likely to exist, with growers potentially having access to multiple information sources and putting a different emphasis on each (such as in Milne et al. (2016)). Such data were lacking for our study systems. We emphasise here the need for more data to be collected and made available, and echo Cunniffe et al. (2015a) in calling for more data related to the spatial structure of hosts, trade networks, and dispersal kernels of vectors, as well as other factors such costs of different control mechanisms, the yield of infected crops in non-experimental settings and risk attitudes of growers.

6.3.2 Changing the form of the decision rules

There is a fundamental lack of data pertaining to the information growers use to make decisions, the importance they put on different factors, and their response to uncertainty. In this thesis, we examined two broad categories of decision rules, based on either rational or strategic-adaptive expectations, but future work could expand on the assumptions underlying the formulations that we used.

Though our behavioural models were based on principles of game theory, they deviated in form from the classical "replicator" or "imitator" dynamics used in many

previous models linking disease and behaviour (Bauch & Earn (2004), Delabouglise & Boni (2020), Bauch (2005)). Replicator dynamics assumes that each individual of the same class will act in the same way, changing the human behavioural dynamics (Sun & Hilker (2021)) and ultimately affecting the model output. Sun & Hilker (2021) compared replicator dynamics with "best response" dynamics, which are similar in form to our switching terms. They found that best-response dynamics recovered the predictions of replicator dynamics when agents were more rational (increasing the conformity amongst individuals of the same class). We did not construct our models using replicator dynamics due to complications arising from differences in time scales; replicator dynamics would require a new state variable (alongside the epidemiological state variables describing disease dynamics) that tracks the total proportion of growers in the population and how they behave. The speed of dynamics in this "behavioural" compartment would differ from the disease dynamics. However, a comparison between our models using "switching terms" and replicator dynamics could provide further useful information on the effect of the behavioural model's construction on epidemic outcomes.

Future work could include modifications to the decision rules to allow for a relative weighting of future outcomes. Temporal discounting refers to the observed tendency of individuals to prefer immediate rewards to delayed ones, even if the delay increases the value of the reward (Loewenstein & Thaler (1989)). The lack of preventative action recommended to growers whose vineyards have low disease prevalence is posited to be a form of temporal discounting, as it does not consider the potential for severity to increase (Hillis et al. (2016)). In our model, we employ a severe version of temporal discounting, as growers only consider the expected profits of the next season. It is likely that there will be some priority given to

subsequent season's profits, particularly amongst risk-prone growers. Our decision rules could include such a preference, allowing for more complex behaviours.

6.3.3 Extensions to the spatial-stochastic model

In Chapter 5, we conducted a preliminary investigation into the effects of space and stochasticity on the robustness of the conclusions derived from Chapter 2. However, for the majority of the models presented in this thesis, we assumed that the population was well-mixed and neglected to consider any spatial structure. Such structure is likely to impact both the epidemiological and behavioural components of our model. Omitting space in disease models means that disease could theoretically spread between two individuals that would, in reality, be sufficiently far apart to preclude infection (Cunniffe et al. (2015a)). The possibility of disease dying out will have clear implications on disease control, as growers are less likely to use control options if the risk of infection is low (Chapter 2). Though the limited spatial modelling we did include did consider the epidemiological impact, modelling over a larger spatial scale could allow us to explore these dynamics in further depth.

Including spatial heterogeneity would significantly impact how growers make decisions by affecting their access to information. In the majority of models presented in this thesis, we presume that all growers have access to the same degree of knowledge; only in our spatial-stochastic model of TYCLV did we allow for varying access to information. However, there is likely to be a large degree of variability in knowledge between growers based on their relationship with neighbours, access to "expert" information, previous experience with control mechanisms etc. (Kaup

(2008)). Networks of information spread have been modelled in Milne et al. (2016), who found, unsurprisingly, that such networks significantly impacted the speed at which growers responded to disease threats, and growers who considered a range of different information sources minimised their losses. The networks we included in Chapter 5 were simple, based only on the distance between growers' fields. Future work could extend these networks to include external sources of knowledge or a mix of growers whose fields are dispersed across the landscape.

Additionally, in all of our models, we presumed that knowledge pertaining to both economic and epidemiological parameters was the same across growers. However, as with information regarding profitability, this is likely to vary depending on an individual grower's own network.

These spatial extensions also apply to trade networks: in Chapter 1, we presumed that when growers traded cassava cuttings, they used a centralised market, enabling them to trade with all other growers in the landscape. However, when trade occurs via a market or central organisation, most transactions occur over a scale larger than our landscape (a square with sides 3.16 km), with around 70 % of transactions occurring over a scale of 10-50 km (Szyniszewska et al. (2021)). For more informal trade settings, with growers interacting with each other, a proximity-based kernel may be more appropriate (McQuaid et al. (2017b), McQuaid et al. (2017a), though both of these were modelled over a larger landscape than ours). Yet such exchanges are hard to parameterise as the probability of exchanging planting material is highly variable across settings (varying between 30 - 92.51% over unspecified spatial scales; Ntawuruhunga et al. (2007), Chikoti et al. (2016), Djaha et al. (2018), Houngue et al. (2018) and Teeken et al. (2018)). Including

plant trade networks may therefore be limited by the availability of data (Pautasso & Jeger (2014)), but completely ignoring them omits an important means of disease spread.

Spatial settings could also be important for planners, who can introduce targeted subsidies to encourage the use of a control scheme. Plant disease control is often targeted to high-risk areas, where hosts that are likely to be infected are removed (Cunniffe et al. (2015b), Hyatt-Twynam et al. (2017), Fabre et al. (2021)). McQuaid et al. (2017a) found that subsidising clean seed increased participation in clean seed systems, though the effect of clustering these subsidies is uncertain (McQuaid et al. (2017b)). By including spatial structure in our models, we could target subsidies at those most at risk of infection, potentially reducing costs to the subsidy provider.

6.3.4 Co-operative control schemes

In each of our models, growers only consider their own profits and act in their own self-interest. Yet for many plant diseases, individual efforts made by a grower to reduce disease severity on their own land are ineffective due to disease spread from an external source (termed primary infection) (Madden et al. (2007), Laranjeira et al. (2020)). Pathogens and their vectors, then, can be considered as a form of "common property" (Lazarus & Dixon (1984)).

The mobility of vectors, in particular, is an excellent motivator for co-ordinated control efforts (Singerman & Useche (2019)). In many cases, then, co-operation between growers is needed to maximise profits for all growers. It is increasingly common for groups of growers to partake in such co-ordinated schemes (Faust et al.

(2008)). These agreements between growers can be formalised via area-wide pest management (AWPM) schemes. These AWPM schemes consist of co-ordinated control amongst growers in a region against a specific disease and have been used in a wide variety of contexts, from managing grape phylloxera in Europe (Kogan (1986)) to corn boll weevil and European corn borer (ECB) in the US (Faust et al. (2008) and Bell et al. (2012) respectively).

Citrus Health Management Areas (CHMAs) are a specific subset of AWPM schemes established across the United States to combat Huanglongbing (HLB, or citrus greening) (Bassanezi et al. (2013), Jones et al. (2013), Graham et al. (2020)). Growers in these CHMAs co-ordinate insecticide sprays, targeting the Asian citrus psyllid (ACP) that vectors HLB. These schemes provide economic benefits, though participation is still limited (Singerman et al. (2017)). Many growers not participating in the CHMA cite others' lack of participation as a key obstacle to their own participation, as well as the effort co-ordinated sprays would require.

A grower's participation in AWPM also will depend on both the opinions of the grower (Milne et al. (2020)) and the incentives provided that help to internalise the externalities produced by engaging in disease control (Bate et al. (2021)). Combining these with other considerations outlined above can therefore give a more realistic insight into the drivers of growers' decisions to use disease control.

6.3.5 Other extensions

Models are, by definition, abstractions of reality; therefore, we have made many simplifying assumptions that could be rectified in future pieces of work. For sim-

plicity, in both our stochastic and deterministic models we included an average loss of yield. In reality, the yield will depend on how far into the growing season the field was infected and the rate of within-field disease spread (as well as many other factors, such as environmental stochasticity, the variety of crop in question, availability of technology, planting density *etc.*). The inclusion of time-dependent yield loss will change the nature of the payoffs to the growers and could shift the balance of payoffs such that the payoff for an infected field planted with clean seed is no longer the lowest possible payoff (i.e. eliminating the "sucker's payoff"). If this shift were observed, an "all control" equilibrium may now be stable in the "grower vs" models that we have described.

We have only considered economic incentives for grower participation in the control. Realistically, other considerations - such as preference for local varieties, flavour, *etc.* - will play an important role in determining participation. Indeed, surveys of cassava growers in Sub-Saharan Africa have found that such qualitative traits have a greater role in determining a grower's preference than economic traits such as yield (Szyniszewska et al. (2021), Houngue et al. (2018)). These private preferences or perceptions of risk are difficult to assess and could be encapsulated in a "cost of control" parameter that will vary both between growers and the cultural and market context in which the crop is being grown.

Empirical studies have shown that recent exposure to disease positively influence growers' engagement with disease control strategies (Elbers et al. (2010), Merrill et al. (2019b), Chen et al. (2013)). We have not explicitly included this "psychological distancing" in our models, though it is somewhat accounted for in our "grower vs" models (where growers consider their outcome from the previ-

ous season). Explicit consideration would mean that growers would account not only for the season immediately preceding their decision but the outcomes of all previous seasons since the start of the epidemic.

Though we focus here on two case studies - the use of clean seed systems for cassava brown streak disease (CBSD) and the deployment of crop varieties that are tolerant and/or resistant to tomato yellow leaf curl virus - the behavioural component of our model is sufficiently flexible that it could be used in other pathosystems.

6.4 Concluding remarks

During this thesis, we have combined human behaviour with classical epidemiological models, demonstrating the importance of including grower decision-making in plant epidemic models. These behavioural models have used different decision rules, showing that *a priori* assumptions about what information growers will use when making assessments affects model outcomes. Though in some ways rather simplistic, our models capture important features of the factors that influence a grower's decision-making process and are sufficiently flexible in form to be adapted to a range of pathosystems. They also leave scope to include other factors, such as irrationality or risk perception, and our agent-based, spatial-stochastic models can allow for even greater complexity. We have tested our models on different control schemes, demonstrating both their range and the importance of considering the externalities generated by each control option, as this could lead to suboptimal outcomes. With greater attention being placed on the "human" factors that impact disease control, these models are a step towards integrating the fields of behavioural economics, sociology and plant disease epidemiology. It is vital that

more empirical studies are done to understand the complex motivations that underpin plant disease management.

Appendix to Chapter 2

A.1 Underlying behaviour of the "strategy vs" models

To demonstrate the underlying drivers of dynamics in our models, we dissect the behaviour of the "strategy vs population" model in some depth. We first investigated the response of the probability of infection, the expected profits and the probability of switching strategy to the total number of infected fields (Figure A.1.1). Generally, as the number of infected fields increases, so too does the probability of infection for controllers and non-controllers (q_C and q_N respectively) (Figure A.1.1A). However, the increased number of infected fields changes the relative importance of the infection pathways for non-controllers. With an increase in the number of infected fields, the instantaneous probability of horizontal transmission for non-controllers (p_N(Horiz), Equation 2.19), decreases as field infection has already taken place through vertical transmission (p_N(Vert), Equation 2.18) (Figure A.1.1A).

For the default parameterisation (Figure A.1.1(B)), the expected profits for the non-controllers (P_N) are always higher than that of the population, so they should always have a zero probability of switching strategy (Figure A.1.1(C)). Conversely, P_C is always below the population average, and thus they should always have a non-zero probability of switching. This results in an equilibrium where there are no controllers present, as all of them have abandoned using the unprofitable CSS. However, by reducing the cost of clean seed ($\phi = 0.125$, equivalent to a 50% subsidy), at around $I = 411$ fields control becomes more profitable and non-controllers have a non-zero probability of switching strategy (Figure A.1.1(E)). As at this point controllers cannot switch strategy, there is an "all control" equilibrium. The point at which $P_N = P_C$ (i.e. when $I \approx 411$, which is the total number of infected fields in the system and does not describe the number of infected controllers and non-controllers) leads to an equilibrium where both control strategies are present.

The value of η (the responsiveness of growers) has no effect on the equilibrium attained by the growers in the "strategy vs population" models, as the equilibrium is based only on the relative values of P_C, P_N and P, though it did affect the time it took to reach equilibrium (Figure A.1.1(D)). This also holds true for the "strategy vs alternative" model.

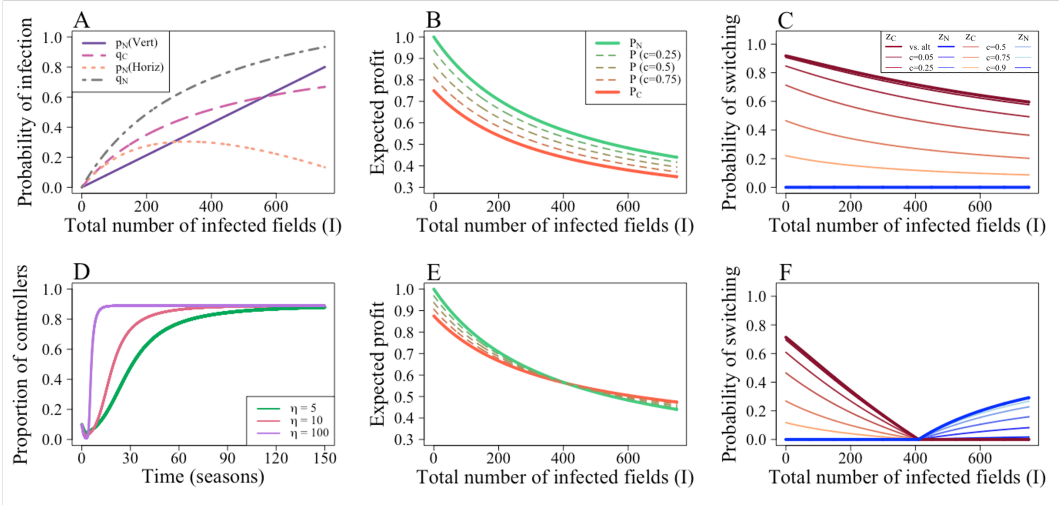

Figure A.1.1: **Probability of infection, responsiveness of growers, expected profits, and switching terms for the "strategy vs population" model.** (A) For the default parameterisation, the probability of infection increases as the number of infected growers increase (q_N and q_C), though the probability non-controllers will be infected via horizontal transmission ($p_{N(\text{Horiz})}$) falls as I increases. (B) The expected profits for controllers, non-controllers and the population for different proportions of controllers in the population (c). For the default parameters, $P_C < P$ for all values of I.(C) Switching probabilities for controllers and non-controllers for different c. As $P_C < P$, controllers should always have a non-zero probability of switching strategy, whereas non-controllers should never start to control.(D) The responsiveness of growers (η) does not affect the final equilibrium values, though it does impact the time it takes to reach equilibrium. (E) shows the expected profits for controllers, non-controllers and the population when the cost of control $\phi, = 0.125$. Now, at high levels of I, it is profitable to control. Where $P_C = P_N$, both strategies will be present at equilibrium. F For $\phi = 0.125$, for low values of I controllers should switch strategy, but as I increases non-controllers should have a non-zero probability of switching into the clean seed system. For this parameter set, the equilibrium value of I is therefore 411, with 349 infected controllers and 63 infected non-controllers.

A.2 Mathematical derivations

A.2.1 The basic reproductive number (R_0) for the "strategy vs" models.

To find the value for R_0, we calculated the next-generation matrix (NGM; Diekmann et al. (2010)). This relies on decomposing a linearised version of the model into two matrices: the first contains the terms relating to disease transmission (matrix J_F) whilst the second has terms relating to non-epidemiological transitions between states (matrix J_V). The NGM, K, is given by $K = J_F J_V^{-1}$ (van den Driessche (2017)). To shorten the notation in what follows, we introduce the following function of state variables:

$$\Gamma = S_C z_{SC} + I_C z_{IC} + S_N(1 - z_{SN}) + I_N(1 - z_{IN}). \tag{A.1}$$

We focus only on the infected compartments in the general model, leading to:

$$\frac{dI_C}{dt} = \beta S_C(I_C + I_N) - \gamma I_C, \tag{A.2}$$

$$\frac{dI_N}{dt} = \gamma\Gamma\left(\frac{p(I_C + I_N)}{N}\right) + \beta S_N(I_C + I_N) - \gamma I_N. \tag{A.3}$$

The disease-free equilibrium (DFE) is given by $(S_C, I_C, S_N, I_N) = (0, 0, N, 0)$. Given that we have restricted the model to just the infected compartments, the DFE can also be written as $(I_C, I_N) = (0, 0)$

The matrix of rates at which new infections occur is

$$F = \begin{bmatrix} \beta S_C(I_C + I_N) \\ \gamma\Gamma\left(\frac{p(I_C+I_N)}{N}\right) + \beta S_N(I_C + I_N) \end{bmatrix}. \tag{A.4}$$

The matrix of rates at which infections are removed is

$$V = \begin{bmatrix} \gamma I_C \\ \gamma I_N \end{bmatrix}. \tag{A.5}$$

The Jacobians for these matrices are:

$$J_F = \begin{bmatrix} \beta S_C & \beta S_C \\ \gamma\Gamma\left(\frac{p}{N}\right) + \gamma\frac{\partial\Gamma}{\partial I_C}\left(\frac{p(I_C+I_N)}{N}\right) + \beta S_N & \gamma\Gamma\left(\frac{p}{N}\right) + \gamma\frac{\partial\Gamma}{\partial I_N}\left(\frac{p(I_C+I_N)}{N}\right) + \beta S_N \end{bmatrix}, \tag{A.6}$$

and

$$J_V = \begin{bmatrix} \gamma & 0 \\ 0 & \gamma \end{bmatrix}. \tag{A.7}$$

We need to evaluate the Jacobians at the DFE. Note that $\gamma\Gamma$ is the net rate at which fields in which there is no control are planted, and so at the DFE, $\Gamma = N$. Note too that, since both are multiplied by $\frac{p(I_C+I_N)}{N}$ (which is zero at the DFE), the partial derivatives and $\frac{\partial\Gamma}{\partial I_C}$ and $\frac{\partial\Gamma}{\partial I_N}$ do not need to be calculated.

At the DFE, the J_F becomes

$$J_F = \begin{bmatrix} 0 & 0 \\ \gamma p + \beta N & \gamma p + \beta N \end{bmatrix}, \tag{A.8}$$

and J_V^{-1} is given by:

$$J_V^{-1} = \begin{bmatrix} 1/\gamma & 0 \\ 0 & 1/\gamma \end{bmatrix}. \tag{A.9}$$

The NGM, $K = J_F J_V^{-1}$, is therefore given by:

$$\begin{bmatrix} 0 & 0 \\ p + \frac{\beta N}{\gamma} & p + \frac{\beta N}{\gamma} \end{bmatrix} \tag{A.10}$$

The dominant eigenvalue for this matrix is $\lambda_1 = \frac{\beta N}{\gamma} + p$, which gives the R_0 for the system. This can be further broken down into distinct components corresponding to horizontal ($R_0^H = \frac{\beta N}{\gamma}$) and vertical ($R_0^V = p$) transmission (Hamelin et al. (2021)).

A.2.2 Stability of equilibria in the "strategy vs population" model.

There are four equilibria, which can be distinguished by the presence of disease and the proportion of growers controlling at equilibrium.

- **Disease-free equilibrium** at which the disease is not able to spread even when there is no control via clean seed

$$(S_C, I_C, S_N, I_N) = (0, 0, N, 0). \tag{A.11}$$

- **Control-free, disease-endemic equilibrium** at which the disease is able to spread in the absence of control, but nevertheless no grower controls

$$(S_C, I_C, S_N, I_N) = \left(0, 0, \frac{\gamma(1-p)}{\beta}, \frac{\beta N - \gamma(1-p)}{\beta}\right). \tag{A.12}$$

- **All-control, disease-endemic equilibrium** at which all growers control, but nevertheless disease is still present in the system

$$(S_C, I_C, S_N, I_N) = \left(\frac{\gamma}{\beta}, \frac{\beta N - \gamma}{\beta}, 0, 0\right) \tag{A.13}$$

- **Two-strategy, disease-endemic equilibrium** at which both disease and control equilibrate at some intermediate level, with

$$S_C = \frac{N(R_0(\gamma pL - \beta\phi N) - \gamma pL)}{\gamma p^2 L}, \tag{A.14}$$

$$I_C = \frac{\beta\phi N^2(R_0(\beta\phi N - \gamma pL) + \gamma pL)}{\gamma p^2 L(\beta\phi N - \gamma pL)}, \tag{A.15}$$

$$S_N = \frac{N(\phi R_0 - pL)(\beta N(\beta\phi N - \gamma pL) + \gamma^2 pL)}{\gamma p^2 L(\beta\phi N - \gamma pL)}, \tag{A.16}$$

$$I_N = \frac{-\phi N R_0(\beta N(\beta\phi N - \gamma Lp) + \gamma^2 Lp)}{\gamma p^2 L(\beta\phi N - \gamma Lp)}. \tag{A.17}$$

We also note that none of Equations A.11, A.12, A.12 or A.14 - A.17 have a dependence on the responsiveness of growers, η. Thus, the equilibrium attained is independent of this parameter, though it does affect the dynamics approaching equilibrium (Appendix 1 Figure A.1.1(D)).

The stability of each of these equilibria will be discussed in turn.

Disease-free equilibrium.

We determined the conditions for stability of each equilibrium in the "strategy vs" models by first evaluating the Jacobian matrix for the system at each possible equilibrium and then determining the eigenvalues for the matrix. The system can be reduced to three state variables, as $N = S_C + I_C + S_N + I_N$. The model therefore

becomes:

$$\frac{dS_C}{dt} = \gamma\left(S_C(1-z_{SC}) + I_C(1-z_{IC}) + (N - S_C - I_C)z_{IN}\right) - \beta S_C\left(I_C + I_N\right) - \gamma S_C, \tag{A.18}$$

$$\frac{dI_C}{dt} = \beta S_C\left(I_C + I_N\right) - \gamma I_C, \tag{A.19}$$

$$\frac{dI_N}{dt} = \gamma\left(S_C z_{SC} + I_C z_{IC} + (N - S_C - I_C)(1 - z_{IN})\right)\left(\frac{p(I_C + I_N)}{N}\right) + \beta(N - S_C - I_C - I_N)\left(I_C + I_N\right) - \gamma I_N. \tag{A.20}$$

where the switching terms are given by:

$$z_{SC} = z_{IC} = \max\left(0, 1 - e^{-\eta(P - P_C)}\right), \tag{A.21}$$

$$z_{SN} = z_{IN} = \max\left(0, 1 - e^{-\eta(P - P_N)}\right). \tag{A.22}$$

The DFE is given by $(S_C, I_C, I_N) = (0, 0, 0)$. The Jacobian matrix evaluated with these values is given by:

$$\begin{bmatrix} -\gamma(1 - \exp(-\eta\phi) + \eta\phi) & \gamma(\exp(-\eta\phi) - \eta\phi) & 0 \\ 0 & -\gamma & 0 \\ 0 & \beta N + \gamma p & \beta N + \gamma p - \gamma \end{bmatrix}$$

From this, we can see the first eigenvalue is $\beta N - \gamma + \gamma p$. The 2×2 matrix that

remains is given by:

$$\begin{bmatrix} -\gamma(1-\exp(-\eta\phi)+\eta\phi) & \gamma(\exp(-\eta\phi)-\eta\phi) \\ 0 & -\gamma \end{bmatrix}$$

This matrix is upper triangular, so the eigenvalues are given by the diagonal elements. We can see that $-\gamma$ and $-\gamma(1-\exp(-\eta\phi)+\phi)$ are the remaining eigenvalues for the matrix.

As $-\gamma$ and $-\gamma(1-\exp(-\eta\phi)+\phi)$ are always negative, the stability of this equilibrium is dependent upon $\beta N-\gamma+\gamma p<0$. This corresponds to the R_0 found using the NGM.

Disease-endemic, all control equilibrium.

The disease-endemic, all control equilibrium is given by: $(S_C, I_C, I_N) = \left(\frac{\gamma}{\beta}, \frac{\beta N-\gamma}{\beta}, 0\right)$. As there are no non-controllers at this equilibrium, $P_C = P$ (Equation 2.23 in the main text).

The Jacobian matrix evaluated using these values is given by:

$$\begin{bmatrix} \frac{\partial \dot{S_C}}{\partial S_C} & \frac{\partial \dot{S_C}}{\partial I_C} & -\gamma \\ \beta N-\gamma & 0 & \gamma \\ \frac{\partial \dot{I_N}}{\partial S_C} & \frac{\partial \dot{I_N}}{\partial I_C} & -\gamma \end{bmatrix} \tag{A.23}$$

with:

$$\frac{\partial \dot{S_C}}{\partial S_C} = \gamma \left(\exp\left(\frac{-\eta(\beta N(\gamma pL - \beta\phi N) - \gamma^2 pL)}{\beta^2 N^2} \right) \right) - \frac{-\eta(\beta N(\gamma pL - \beta\phi N) - \gamma^2 pL)}{\beta^2 N^2} - \beta N \tag{A.24}$$

$$\frac{\partial \dot{S_C}}{\partial I_C} = -\gamma \left(1 - \exp\left(\frac{-\eta(\beta N(\gamma pL - \beta\phi N) - \gamma^2 pL)}{\beta^2 N^2} \right) \right) - \frac{-\eta(\beta N(\gamma pL - \beta\phi N) - \gamma^2 pL)}{\beta^2 N^2} \tag{A.25}$$

$$\frac{\partial \dot{I_N}}{\partial S_C} = \beta N + \gamma - \frac{\gamma p(\beta N - \gamma)}{\beta N} \left(\exp\left(\frac{-\eta(\beta N(\gamma pL - \beta\phi N) - \gamma^2 pL)}{\beta^2 N^2} \right) - \frac{-\eta(\beta N(\gamma pL - \beta\phi N) - \gamma^2 pL)}{\beta^2 N^2} \right) \tag{A.26}$$

$$\frac{\partial \dot{I_N}}{\partial I_C} = \frac{\partial \dot{I_N}}{\partial S_C}. \tag{A.27}$$

We can see that

$$\frac{\partial \dot{S_C}}{\partial S_C} = \frac{\partial \dot{S_C}}{\partial I_C} - \gamma + \beta N. \tag{A.28}$$

The characteristic equation for Matrix A.23 is given by

$$-(\lambda + \gamma) \left(\lambda^2 - \lambda \left(\frac{\partial \dot{S_C}}{\partial I_C} - \gamma + \beta N \right) - (\beta N - \gamma) \left(\frac{\partial \dot{S_C}}{\partial I_C} \right) \right) \tag{A.29}$$

Solving this equation, we find the eigenvalues to be $-\gamma$, $\beta N - \gamma$ and

$$\frac{\partial \dot{S_C}}{\partial I_C} = -\gamma \left(1 - \exp\left(\frac{-\eta(N\beta(\gamma pL - \beta\phi N) - \gamma^2 pL)}{\beta^2 N^2}\right) + \frac{\eta(\beta N(\gamma pL - \beta\phi N) - \gamma^2 pL)}{\beta^2 N^2}\right). \tag{A.30}$$

As $-\gamma$ is always negative, the stability depends on the remaining eigenvalues. The second eigenvalue is always negative for $\frac{\beta N}{\gamma} > 1$, which is also the R_0 for horizontal transmission (R_0^H). Given that this expression can be re-written as:

$$\frac{\partial \dot{S_C}}{\partial I_C} = -\gamma \left(1 - \exp\left(-x\right) + x\right), \tag{A.31}$$

where $x = \frac{\eta(\beta N(\gamma pL - \beta\phi N) - \gamma^2 pL)}{\beta^2 N^2}$, the final eigenvalue is negative once the term in the exponent (x) is negative:

$$\frac{-\eta(\beta N(\beta\phi N - \gamma pL) + \gamma^2 pL)}{\beta N} < 0 \tag{A.32}$$

$$(\beta N(\beta\phi N - \gamma pL) + \gamma^2 pL) < 0 \tag{A.33}$$

$$\frac{\beta N(\beta\phi N - \gamma pL)}{\gamma} < \gamma pL \tag{A.34}$$

$$\gamma pL \left(\frac{\gamma - \beta N}{\beta^2 N^2}\right) > \phi \tag{A.35}$$

$$pL \left(\frac{\gamma - \beta N}{\beta N}\right)\left(\frac{\gamma}{\beta N}\right) > \phi. \tag{A.36}$$

For clarity, this can be re-arranged as follows:

$$\begin{matrix}\text{Cost of}\\ \text{control}\end{matrix} < \begin{matrix}\text{Loss due to}\\ \text{disease}\end{matrix} \times \begin{matrix}\text{Probability of}\\ \text{vertical transmission}\end{matrix} \times \begin{matrix}\text{Proportion of}\\ \text{infected fields}\end{matrix} \times \left(1 - \begin{matrix}\text{Probability of}\\ \text{horizontal transmission}\end{matrix}\right),$$

$$\phi < Lp\left(\frac{\beta N - \gamma}{\beta N}\right)\left(1 - \frac{\beta N - \gamma}{\beta N}\right). \tag{A.37}$$

That is, for the "all control" equilibrium to be stable, $R_0^H > 1$ and the cost of control (ϕ) must be less than the expected losses of infected non-controllers (L).

Disease-endemic, control-free equilibrium.

The disease-endemic, control-free equilibrium is given by: $(S_C, I_C, I_N) = \left(0, 0, \frac{\beta N - \gamma(1-p)}{\beta}\right)$. Additionally, as there are no controllers at this equilibrium, $P_N = P$ (Equation 2.23 in the main text). The entries for the Jacobian matrix evaluated at the disease-endemic, no control equilibrium are given as follows:

$$\begin{bmatrix} \frac{\partial \dot{S_C}}{\partial S_C} & \frac{\partial \dot{S_C}}{\partial I_C} & 0 \\ \gamma(R_0 - 1) & -\gamma & 0 \\ \frac{\partial \dot{I_N}}{\partial S_C}^* & \frac{\partial \dot{I_N}}{\partial I_C}^* & \gamma(R_0 - 1) \end{bmatrix} \tag{A.38}$$

Entries marked with "*" are not written out in full as they are not needed for

further analysis. Remaining entries are given as follows:

$$\frac{\partial \dot{S_C}}{\partial S_C} = \gamma \left(\exp\left(-\frac{\eta(\gamma R_0(\beta\phi N - \gamma pL) + \gamma^2 pL)}{\beta\gamma N R_0} \right) - \frac{\gamma\eta(R_0(\beta\phi N - \gamma pL) + \gamma pL)}{\beta N R_0} - R_0 \right) \tag{A.39}$$

$$\frac{\partial \dot{S_C}}{\partial I_C} = \gamma \left(\exp\left(-\frac{\eta(\gamma R_0(\beta\phi N - \gamma pL) + \gamma^2 pL)}{\beta\gamma N R_0} \right) - \frac{\gamma\eta(R_0(\beta\phi N - \gamma pL) + \gamma pL)}{\beta N R_0} \right) \tag{A.40}$$

from which we can see that

$$\frac{\partial \dot{S_C}}{\partial S_C} = \frac{\partial \dot{S_C}}{\partial I_C} - \gamma R_0. \tag{A.41}$$

From this matrix, we can see that the first eigenvalue is given by $\gamma(R_0 - 1)$. This is negative for $R_0 > 1$.

The remaining eigenvalues can be found by solving the equation:

$$\lambda^2 - a_1\lambda + a_2 = 0 \tag{A.42}$$

where

$$a_1 = \frac{\partial \dot{S_C}}{\partial I_C} - \gamma(R_0 - 1) \tag{A.43}$$

$$a_2 = -\gamma R_0 \left(\frac{\partial \dot{S_C}}{\partial I_C} - \gamma \right). \tag{A.44}$$

Solving this, we find the remaining eigenvalues to be: $-\gamma R_0$, and

$$\gamma\left(\exp\left(-\frac{\eta(\gamma R_0(\beta\phi N-\gamma pL)+\gamma^2 pL)}{\beta\gamma N R_0}\right)-1+\frac{-\eta(R_0((\beta\phi N-\gamma pL))-\gamma\eta pL)}{\beta N}\right). \tag{A.45}$$

Clearly, $-\gamma R_0$ is always negative. The final eigenvalue is negative once

$$1-\exp\left(-\frac{\eta(\gamma R_0(\beta\phi N-\gamma pL)+\gamma^2 pL)}{\beta\gamma N R_0}\right)<\frac{-\eta(R_0((\beta\phi N-\gamma pL))-\gamma\eta pL)}{\beta N} \tag{A.46}$$

That is, the eigenvalue is negative once the term in the exponent is negative. This can be rearranged as follows:

$$Lp\left(\frac{\beta N-\gamma(1-p)}{\beta N}\right)\left(\frac{\gamma}{\beta N+\gamma p}\right)<\phi \tag{A.47}$$

For clarity, this can be re-written in terms of the probability of infection:

$$\begin{array}{ccccccc}\text{Loss due to} & & \text{Probability of} & & \text{Proportion of} & & \\ \text{disease} & \times & \text{vertical transmission} & \times & \text{infected fields} & \times & \left(1-\begin{array}{c}\text{Probability of}\\ \text{horizontal transmission.}\end{array}\right) < \begin{array}{l}\text{Cost o}\\ \text{contro}\end{array}\end{array} \tag{A.48}$$

$$Lp\left(\frac{\beta N-\gamma(1-p)}{\beta N}\right)\left(1-\frac{\beta N-\gamma(1-p)}{\beta N+\gamma p}\right)<\phi. \tag{A.49}$$

Therefore, the control-free, disease-endemic equilibrium is stable for $R_0 > 1$ and when the expected losses due to disease for an infected non-controller are less than the cost of control.

A.2.3 Stability of two-strategy equilibrium in the "strategy vs" models.

Our "strategy vs" models are discontinuous, as the switching terms take different forms depending on the relative values of the expected profits for each strategy and the population. Due to this discontinuity, the stability of the two-strategy equilibrium for the "strategy vs population" model cannot be assessed by linearising around the equilibrium. We instead used a numerical approach to assess the stability of this equilibrium. We first generated a parameter set by sampling parameters from a plausible range (given in Appendix 2 Table A.2.1). We then ran the model for each parameter set and found the equilibrium values for each state variable ($E^* = (S_U^*, I_U^*, S_C^*, I_C^*)$, taken after 500 seasons). Then, for the same parameter set, the model was run with a set of 10,000 different initial conditions again for 500 seasons. The difference between the final equilibrium value for each state variable and E^* was calculated. If the difference was greater than 10^{-8}, it was deemed large enough to conclude that the equilibrium was unstable based on different initial conditions. We repeated this for 5,000 different parameter sets and, in each instance, the equilibrium attained was that expected for the parameter set.

Table A.2.1: table
Range of values used for parameters when evaluating the stability of the "grower vs" models.

Parameter	Meaning	Range of values
$1/\gamma$	Length of the growing season	$1 - 600$ days
β	Rate of secondary infection	$0 - 2 \times 10^{-5}$ day^{-1} field^{-1}
p	Probability of getting infected cuttings	$0 - 1$
η	Responsiveness of growers	$1 - 200$
L	Loss due to infection	$0 - 1$
ϕ	Cost of control	$0 - 1$
q_j^k	Estimated value of q_j for grower k (only differs by grower in the spatial model)	Equations 5.5 and 5.6
q_α	Perceived value of the whitefly dispersal scale parameterα	Equations 5.7
q_{β_S}	Perceived value of horizontal transmission in the stochastic, spatial model (β_S)	Equations 5.8
$S_C(0)$	Initial proportion of susceptible controllers	$0 - N$
$I_C(0)$	Initial proportion of infected controllers	$0 - N$
$S_N(0)$	Initial proportion of susceptible non-controllers	$0 - N$
$I_N(0)$	Initial proportion of infected non-controllers	$0 - N$

A.2.4 Mutual exclusivity of equilibria

For the "no control" equilibrium to be stable, the expected losses due to vertical transmission must be less than the cost of control. That is, from Equation A.49, the following must be true:

$$Lp\left(\frac{\beta N - \gamma(1-p)}{\beta N}\right)\left(1 - \frac{\beta N - \gamma(1-p)}{\beta N + \gamma p}\right) < \phi \tag{A.50}$$

Conversely, for the "all control" equilibrium to be stable, the costs must be less than the expected losses due to vertical transmission. From Equation A.37, this means that:

$$\phi < Lp\left(\frac{\beta N-\gamma}{\beta N}\right)\left(1-\frac{\beta N-\gamma}{\beta N}\right) \tag{A.51}$$

Thus, for both equilibria to be stable, the following condition must be met:

$$Lp\left(\frac{\beta N-\gamma(1-p)}{\beta N}\right)\left(1-\frac{\beta N-\gamma(1-p)}{\beta N+\gamma p}\right) < Lp\left(\frac{\beta N-\gamma}{\beta N}\right)\left(1-\frac{\beta N-\gamma}{\beta N}\right) \tag{A.52}$$

This can be simplified to:

$$\left(\frac{\beta N-\gamma(1-p)}{\beta N+\gamma p}\right) < \left(\frac{\beta N-\gamma}{\beta N}\right), \tag{A.53}$$

which, in turn, leaves us with the condition that $0 < -\gamma^2 p$ must be true. However, as both γ and p are positive parameters, this is not possible and thus both equilibria cannot be simultaneously stable.

A.2.5 Numerical assessment of equilibria in the "grower vs" models.

The more complex form of these models precludes mathematical analysis, as conditions for model equilibria no longer simply depend on the difference in profits between con-

trollers and non-controllers. For these "grower vs" models to reach a two-strategy equilibrium, the flow between the two strategies must be equal, i.e.

$$z_{IN} I_N = z_{SC} S_C + z_{IC} I_C \tag{A.54}$$

Consequently, we evaluated the stability of the possible equilibrium using numerical methods. Using the same method as when assessing the stability of the "two-strategy equilibrium" in the "strategy vs" models, we tested 10,000 sets of initial conditions and ran the model to equilibrium for 5,000 different parameter sets (chosen from a range of the plausible range parameters described in Appendix 2 Table A.2.1). In no case for either of the "grower vs" models was it found that the difference between equilibrium values of the simulated model was larger than our difference threshold of 10^{-8}. We thus concluded that the final equilibrium attained was not dependent on initial conditions.

The forms of the switching terms will differ between the two "grower vs" models, leading to different equilibria for each model (Figure 2.4(C)-(D) in the main text). There are two important differences from the "strategy vs" models:

- **The "all control" equilibrium is impossible** because growers who controlled but nevertheless became infected always consider switching strategy, as they are earning the lowest possible payoff (P_{IC}, the "sucker's payoff"). As long as there is a non-zero probability of infection (which is necessary for control to be worthwhile) there will always be non-controlling growers.

- **For the "grower vs population" model, the "no control" equilibrium is only possible if all non-controllers are infected at equilibrium.** For a "no control" equilibrium, the expected profit of the population must be less than or equal to P_{IN} to prevent I_N growers switching strategies (i.e. $P \leq P_{IN}$, Equation 2.34). At the "no control" equilibrium $P = P_N$ (Equation 2.23), leading to:

$$P = \frac{P_N(S_N + I_N)}{N} \leq P_{IN} \tag{A.55}$$

As, in the "no control" equilibrium, $S_N + I_N = N$, this can be simplified:

$$\begin{aligned} P_N &\leq P_{IN} \\ q_N P_{IN} + (1 - q_N)P_{SN} &\leq P_{IN} \\ (1 - q_N)P_{SN} &\leq P_{IN}(1 - q_N) \\ P_{SN} &\leq P_{IN} \end{aligned} \tag{A.56}$$

The non-zero value of the loss due to disease (L) means that the conclusion presented in Equation A.56 is impossible (see Equation 2.11 & 2.12 in the main text). Therefore, for a "no-control" equilibrium to be possible, all fields must be infected (i.e. $N = I_N$ and $q_N = 1$). In Figure 2.3(B) (main text), a "no control" equilibrium would require higher β and p (Figure A.2.1).

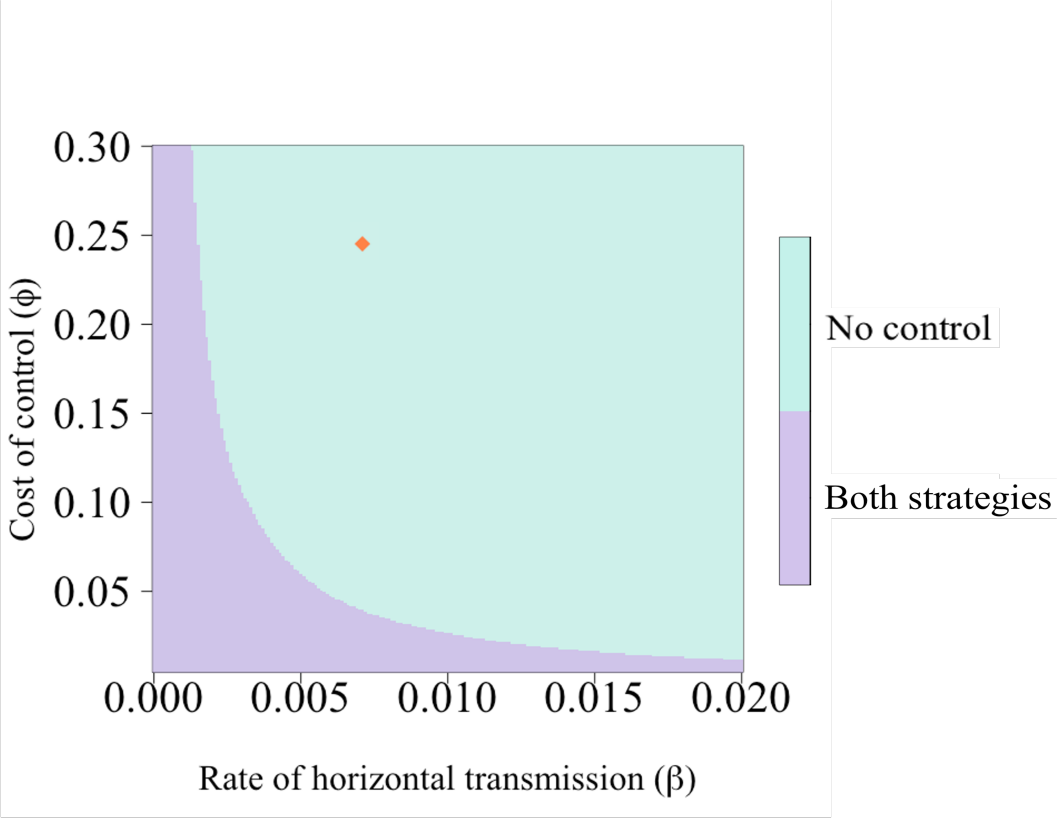

Figure A.2.1: **Possible equilibria for the "grower vs population" model when $p = 1$.** With the higher probability of vertical transmission, the "no control" equilibrium is possible for the "grower vs population" model as there will be no non-infected, non-controlling (S_N) growers at equilibrium (Equation A.56). However, now that $p = 1$, there can never be a disease-free equilibrium for this parameter set (as $R_0 > 1$).

Additionally, unlike in the "strategy vs" models, where the equilibrium is not dependent on the value of responsiveness of growers (η), for the "growers vs" models η does affect the final equilibrium values (see also Appendix 3 Figure A.3.1).

A.3 Supplementary results

A.3.1 Effect of responsiveness (η) on the "grower vs" models.

Unlike in the "strategy vs" models, where the equilibrium values are not dependent on the value of responsiveness of growers, for the "growers vs" models η does affect the final equilibrium values. Appendix 3 Figure A.3.1 shows how the proportion of controllers changes with varying values of horizontal transmission (β) and η.

Interestingly, there is a directionality to the response. Below a certain threshold (which here is $\beta = 0.003301$ day $^{-1}$), an increase in η decreases the proportion controlling. The increase in η means that all growers who have the potential to switch strategy have a higher probability of doing so, and if η is sufficiently high then each switching term approaches 1. This means that there is a decrease in controllers, as all S_C and I_C growers will switch strategy, though only I_N growers will start controlling.

At higher values of β, the increase in η causes an increase in controllers. For these values, z_{sc} eventually falls to zero, and having higher values of η increases the rate at which this happens. As the parameter values permit a two-strategy equilibrium, but the S_C growers are no longer switching strategy, there is an increase in the number of controllers with an increase in η.

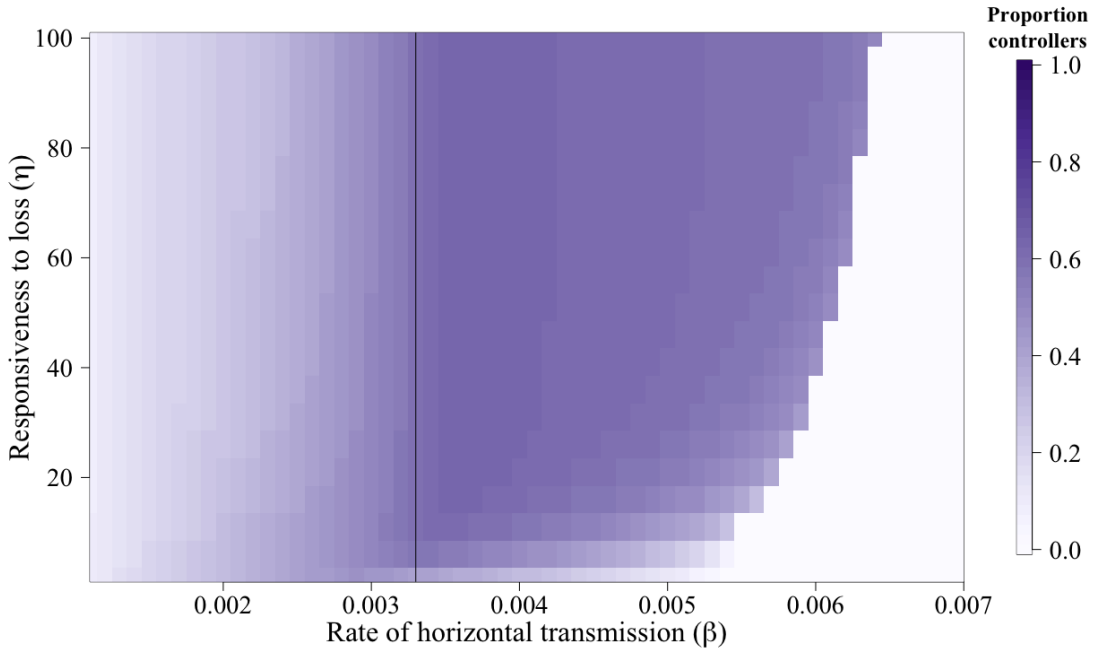

Figure A.3.1: **Effect of changes in responsiveness and horizontal transmission on the proportion of controllers at equilibrium.** Below a certain threshold, there is a decrease in the proportion of controllers with an increase in η, as the higher responsiveness causes more S_C growers to switch strategy. Above this threshold, an increase in η causes an increase in controllers. The parameter values mean that S_C growers no longer change strategy, so they cannot leave the CSS. However, an increase in η means that I_N growers have a higher probability of switching into the CSS. The solid vertical line denotes where this threshold is crossed ($\beta = 0.03301$ day $^{-1}$).

In the "grower vs" models, equilibrium is reached when $z_{SC}S_C + z_{IC}I_C = z_{IN}I_N$ (i.e. the flow of growers out of one strategy matches the flow of growers out of the other) (Figure A.3.2). As the switching terms are in part set by the value of η, for changes in responsiveness then the values of S_C, I_C and I_N at equilibrium must change to ensure the equilibrium condition is still met.

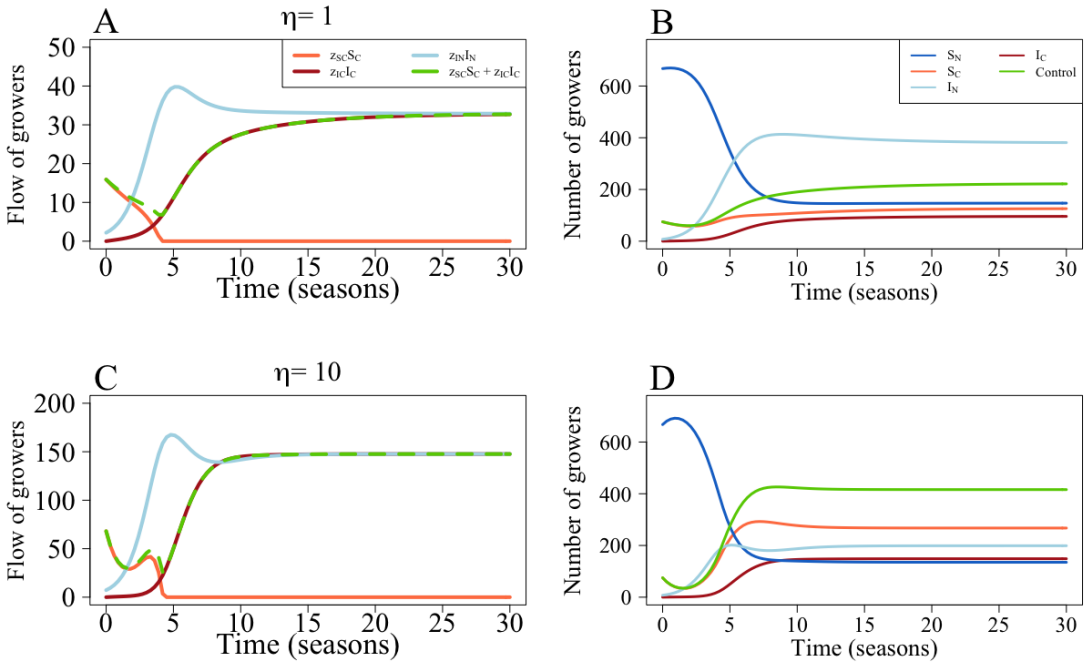

Figure A.3.2: **Effect of responsiveness (η) on the "grower vs alternative" model.** (A) The flow of growers between the non-infected controllers (S_C), infected controllers (I_C) and infected non-controllers (I_N) based on their probability of switching strategy (z_{SC}, z_{IC} and z_{IN} respectively) with $\eta = 1$. (B) Full model dynamics for $\eta = 1$.(C) The flow of growers between S_C, I_C and I_N based on their probability of switching with $\eta = 10$. Note that with lower values of η, there are fewer growers moving between strategies.(D) Full model dynamics for $\eta = 10$. Parameters are as in Table 2.2.2, except for $\beta = 0.004$ day -1, which was used to allow for a disease- and control-endemic equilibrium (Figure 2.3).

A.3.2 Effect of parameters on the "strategy vs" and "grower vs population" models

We will now consider the three other behavioural models (the "strategy vs population", "strategy vs alternative" and "grower vs population") that were not a focus of the main text. Each model formulation responded very differently to changes in parameter values. In all cases, when the rate of horizontal transmission (β) is sufficiently low, no grower should use the CSS (Appendix 3 Figure A.3.2). At medium-to-low values of β, more growers use the CSS, though as β increased, the higher probability of infection narrows the range of costs for which a controller will consider participation in the CSS. The two "strategy vs" models - which had the same equilibria - allowed an "all control" equilibrium at low costs of control (ϕ). This was never possible for the "grower vs population" model, as controllers managing infected fields should always consider switching strategy as they have received the "sucker's payoff".

Overall, the "strategy vs" models saw lower participation in the CSS than the "grower vs" models. The high default value of the cost of control ($\phi = 0.25$) means that growers will participate in the CSS only at very high probabilities of vertical transmission (p) and loss due to disease (L). In the "grower vs" models, however, the non-controllers with infected fields will likely have a lower payoff than the population average, and thus should have a higher probability of switching strategy for a wider range of these parameter values.

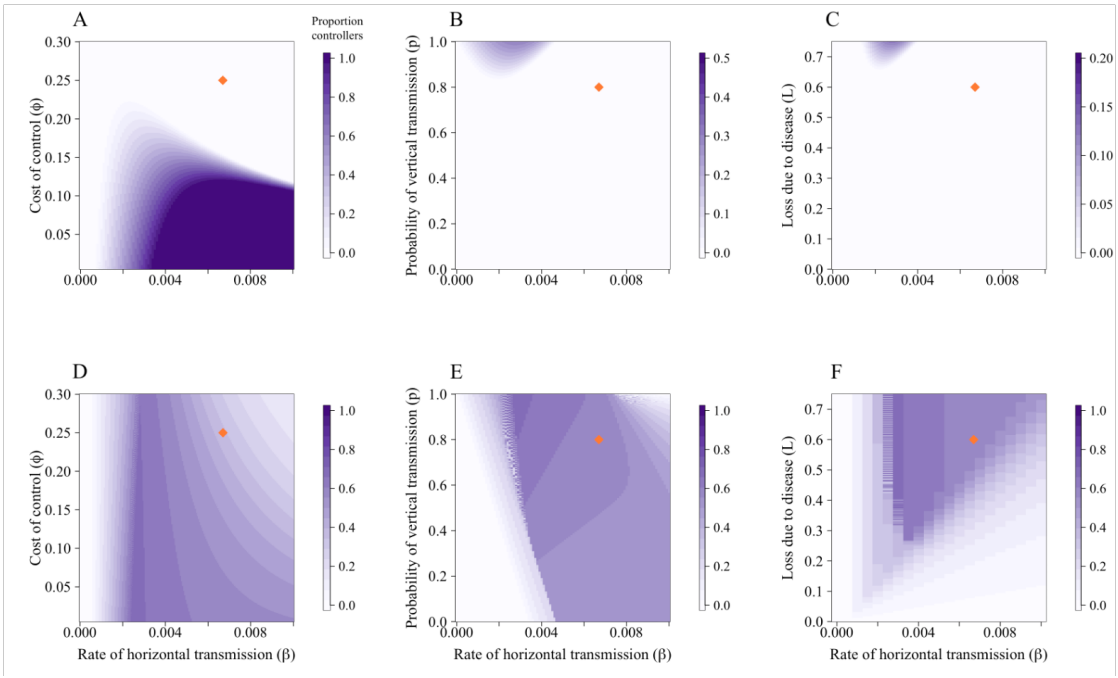

Figure A.3.3: **Effect of parameters on participation in the CSS.** (A)–(C) shows results for the "strategy vs population" and "strategy vs alternative" model formulations;(D)–(F) are for the "grower vs population" model. Parameters are as in Table 2.2.2 in the main text; the orange diamonds mark the default values. (A) and (D) examine the impact of changing the cost of control (ϕ) and the rate of horizontal transmission (β). At low values of β, no-one should control as the probability of infection is sufficiently low that it is not necessary. As β increases, controlling is more beneficial and the cost of control is perceived to be worthwhile. However, as infection becomes more likely, the value of control diminishes so it is not worthwhile to invest in control. In the "strategy vs" comparisons, it is possible to reach an "all control" equilibrium, though this cannot happen for the "grower vs population" model. (B) and (C) At very high probabilities of vertical transmission (p) and loss due to disease (L), growers will participate in the CSS, though only for a narrow range of β. However, for the "grower vs population" models, a much wider range of all parameters allowed for participation. The irregular contours in (E) and (F) are due to oscillations around the equilibrium.

A.3.3 Effect of subsidy on parameter scans

Using the default parameters, there is a narrow range of parameter values for which growers should consider control at equilibrium in the "strategy vs" models (Appendix 3 Figure 2.5D). In the "grower vs" models, though there is a wider range of parameters for which growers control at equilibrium, there are still low levels of participation in the CSS (Appendix 3 Figure 2.5(E)-(F)). Even when the probability of vertical transmission is high, there are few growers using the CSS due to the high cost of participation. However, providing a 50% subsidy such that $\phi = 0.125$ increases both the level of participation and the range of parameter values for which growers control (Appendix 3 Figure A.3.4).

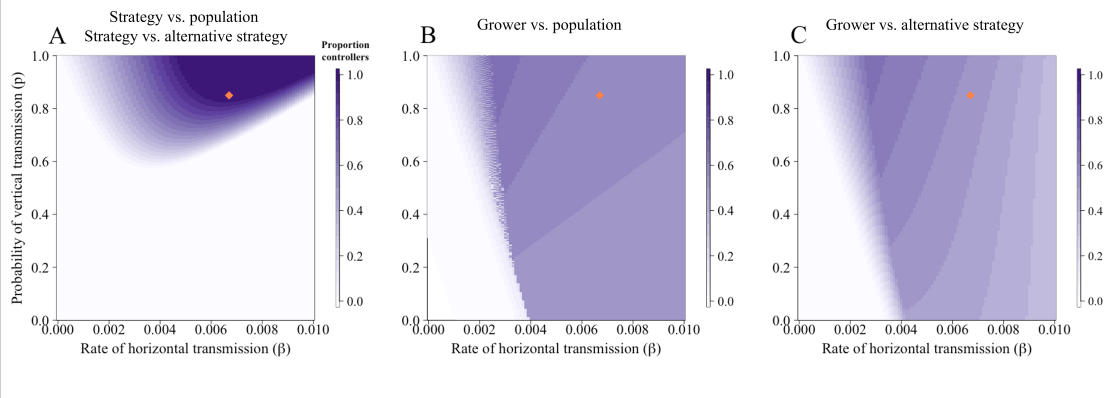

Figure A.3.4: **Effect of changes in the rate of secondary transmission (β) and probability of vertical transmission (p) on the proportion of controllers when $\phi = 0.125$.** Compared to the default value of $\phi = 0.25$, a higher proportion of growers control for a broader range of parameter values.

Appendix to Chapter 3

B.1 Ordering of switching terms

The values of the switching terms are determined by the values of the profits laid out in Equations 3.9-3.16. The ordering will depend on the relative values of three key parameters: the loss due to disease for unimproved crop (L), the loss due to disease for the improved crop ($\delta_L L$) and the cost of control (ϕ_C).

If we assume that $L > \delta_L L$ and $L > (\delta_L L + \phi_C)$ (as for the default tolerant parameterisation), the ordering of the profits is as follows:

$$P_{SU} = P_{EU} > P_{SC} = P_{EC} > P_{ICR} > P_{ICH} > P_{IUR} > P_{IUH} \tag{B.1}$$

There are five possible combinations of values for the switching terms:

- $z_{SC}, z_{EC}, z_{ICR}, z_{ICH}, z_{IUR}, z_{IUH} > 0$
- $z_{ICR}, z_{ICH}, z_{IUR}, z_{IUH} > 0$

- $z_{ICH}, z_{IUR}, z_{IUH} > 0$
- $z_{IUR}, z_{IUH} > 0$
- $z_{IUH} > 0$

For higher costs of tolerant crop, it may be the case that though $L > \delta_L L$, $L < (\delta_L L + \phi_C)$. If the rogued tolerant crop is is more expensive than the rogued unimproved crop (i.e. $\phi_R \delta_L L + \phi_C > \phi_R L$), and the harvested unimproved crop is more expensive than the rogued tolerant crop ($L > \phi_R \delta_L L + \phi_C$) the payoffs and switching terms will be:

$$P_{SU} = P_{EU} > P_{SC} = P_{EC} > P_{IUR} > P_{ICR} > P_{IUH} > P_{ICH} \tag{B.2}$$

- $z_{SC}, z_{EC}, z_{IUR}, z_{ICR}, z_{IUH}, z_{ICH} > 0$
- $z_{IUR}, z_{ICR}, z_{IUH}, z_{ICH} > 0$
- $z_{ICR}, z_{IUH}, z_{ICH} > 0$
- $z_{IUH}, z_{ICH} > 0$
- $z_{ICH} > 0$

Conversely, for $L > \delta_L L$, $L < (\delta_L L + \phi_C)$, if the rogued tolerant crop is more expensive than the rogued unimproved crop (i.e. $\phi_R \delta_L L + \phi_C < \phi_R L$), but the harvested unimproved crop is cheaper than the rogued tolerant crop ($L < \phi_R \delta_L L + \phi_C$), then the payoffs are:

$$P_{SU} = P_{EU} > P_{SC} = P_{EC} > P_{IUR} > P_{IUH} > P_{ICR} > P_{ICH} \tag{B.3}$$

and the switching terms are given by:

- $z_{SC}, z_{EC}, z_{IUR}, z_{IUH}, z_{ICR}, z_{ICH} > 0$
- $z_{IUR}, z_{IUH}, z_{ICR}, z_{ICH} > 0$
- $z_{IUH}, z_{ICR}, z_{ICH} > 0$
- $z_{ICR}, z_{ICH} > 0$
- $z_{ICH} > 0$

For the default resistant parameterisation, $L = \delta_L L$ and $L < (\delta_L L + \phi_C)$. The profits are ordered as:

$$P_{SU} = P_{EU} > P_{SC} = P_{EC} > P_{IUR} > P_{IUH} > P_{ICR} > P_{ICH} \tag{B.4}$$

The following combinations of switching terms are possible:

- $z_{SC}, z_{EC}, z_{IUH}, z_{ICR}, z_{ICH}, z_{IUR} > 0$
- $z_{IUH}, z_{ICR}, z_{ICH}, z_{IUR} > 0$
- $z_{ICR}, z_{ICH}, z_{IUR} > 0$
- $z_{ICH}, z_{IUR} > 0$
- $z_{ICH} > 0$

B.2 Mathematical details of non-behavioural model

We use the NGM method (Diekmann et al. (2010)) to calculate the basic reproduction number when there are two types of crop (improved and unimproved) present at the disease-free equilibrium, but growers cannot change strategy.

We focus only on the infected compartments, which are given by:

$$\frac{dE_C}{dt} = \delta_\beta \beta S_C (I_U + \delta_\sigma I_C) - \delta_\epsilon \epsilon E_C - \gamma E_C, \tag{B.5}$$

$$\frac{dI_C}{dt} = \delta_\epsilon \epsilon E_C - \mu_C I_C - \gamma I_C, \tag{B.6}$$

$$\frac{dE_U}{dt} = \beta S_U (I_U + \delta_\sigma I_C) - \epsilon E_U - \gamma E_U, \tag{B.7}$$

$$\frac{dI_U}{dt} = \epsilon E_U - \mu_U I_U - \gamma I_U. \tag{B.8}$$

We first linearise these equations to give the Jacobian matrix and evaluate it around the disease-free equilibrium. We then decompose this Jacobian matrix into two further matrices: F, which is the matrix of terms relating to disease transmission, and $V = -Q$, where Q is the matrix containing non-epidemiological transition terms. The NGM, K, is then given by FV^{-1} (Diekmann et al. (2010)).

For this system,

$$F = \begin{bmatrix} 0 & \delta_\sigma \delta_\beta \beta C & 0 & \delta_\beta \beta C \\ 0 & 0 & 0 & 0 \\ 0 & \delta_\sigma \beta U & 0 & \beta U \\ 0 & 0 & 0 & 0 \end{bmatrix}, \tag{B.9}$$

and

$$V = \begin{bmatrix} \delta_\epsilon\epsilon + \gamma & 0 & 0 & 0 \\ -\delta_\epsilon\epsilon & \mu_C + \gamma & 0 & 0 \\ 0 & 0 & \epsilon + \gamma & 0 \\ 0 & 0 & -\epsilon & \gamma + \mu_U \end{bmatrix}. \tag{B.10}$$

The inverse of V is given by:

$$V^{-1} = \begin{bmatrix} \frac{1}{(\delta_\epsilon\epsilon+\gamma)} & 0 & 0 & 0 \\ \frac{\delta_\epsilon\epsilon}{(\delta_\epsilon\epsilon+\gamma)(\mu_C+\gamma)} & \frac{1}{\gamma+\mu_C} & 0 & 0 \\ 0 & 0 & \frac{1}{\epsilon+\gamma} & 0 \\ 0 & 0 & \frac{\epsilon}{(\epsilon+\gamma)(\mu_U+\gamma)} & 1/(\mu_U+\gamma) \end{bmatrix}. \tag{B.11}$$

The NGM, $K = FV^{-1}$, is then given by:

$$K = \begin{bmatrix} \frac{\delta_\epsilon\epsilon\delta_\sigma\delta_\beta\beta C}{(\delta_\epsilon\epsilon+\gamma)(\mu_C+\gamma)} & \frac{\delta_\sigma\delta_\beta\beta C}{(\mu_C+\gamma)} & \frac{\epsilon\delta_\beta\beta C}{(\epsilon+\gamma)(\mu_U+\gamma)} & \frac{\delta_\beta\beta C}{(\mu_U+\gamma)} \\ 0 & 0 & 0 & 0 \\ \frac{\delta_\epsilon\epsilon\delta_\sigma\beta U}{(\delta_\epsilon\epsilon+\gamma)(\mu_C+\gamma)} & \frac{\delta_\sigma\beta U}{(\mu_C+\gamma)} & \frac{\epsilon\beta U}{(\epsilon+\gamma)(\mu_U+\gamma)} & \frac{\beta U}{(\mu_U+\gamma)} \\ 0 & 0 & 0 & 0 \end{bmatrix}. \tag{B.12}$$

R_0 is given by the leading eigenvalue of this matrix:

$$R_0 = \frac{\epsilon\beta U}{(\gamma+\mu_U)(\gamma+\epsilon)} + \frac{\delta_\epsilon\epsilon\delta_\sigma\delta_\beta\beta C}{(\gamma+\mu_C)(\gamma+\delta_\epsilon\epsilon)} \tag{B.13}$$

B.3 Supplementary results for non-behavioural model

B.3.1 Parameters relating to tolerance and resistance

A broad range of parameter values relating to tolerant and resistant traits are possible depending on the cultivar and environmental conditions. As illustrative examples of the effect of changing parameters along the tolerance/resistance continuum, we investigate the effects of changing the probability of detection for improved crop ($\delta_\nu \nu$; Figure B.3.1) and relative susceptibility of improved crop (δ_β; Figure B.3.2). In each case, profits were highest for both controllers and non-controllers when the parameterisation approached that of the resistant crop (i.e. a high probability of detection and low relative susceptibility). However, there was little impact on the profits of controllers who grew tolerant crop (Figure B.3.1(C) and Figure B.3.2(C)), as the low loss due to disease for tolerant crop means that the reduced probability of infection conferred by high δ_ν and low δ_β is of little benefit. We note that by lowering the relative susceptibility of tolerant crop, it contravenes the typical definition of tolerance (which does not have a reduced probability of infection). However, in terms of the relative yield loss, this crop type retains some tolerant characteristics and we therefore refer to it as "tolerant" for this result.

Disease elimination was possible under many parameterisations for the resistant crop (due to the lower background infectivity of resistant crop, as well as its lower susceptibility). For the "tolerant crop", when the relative susceptibility was very low, only then was disease elimination possible (Figure B.3.2(A),(C)). This allowed growers to earn the

maximum possible profits.

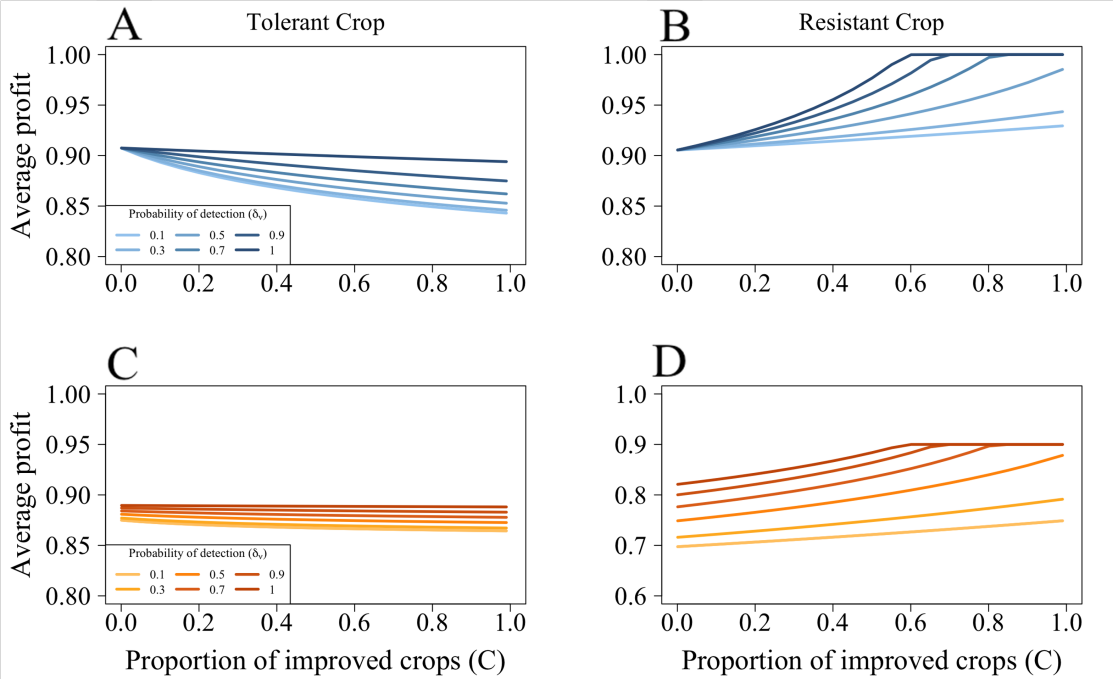

Figure B.3.1: **Change in average profits for tolerant and resistant parameterisation when the probability of detection for improved crop ($\delta_\nu\nu$) is varied.** (A) and (C) show the average profit for unimproved and improved crop respectively for the tolerant parameterisation, whilst (B) and (D) show the same for the resistant parameterisation. In all cases, the highest profits were achieved when $\delta_\nu = 1$ (i.e. infectious crop is always detected). The probability of detection had little impact on the average profit of controllers (C). This is because the probability of detection affects the rate at which infectious plants are removed; the higher the probability of detection, the lower the disease pressure and thus the lower the probability of incurring the loss due to disease. As the loss due to disease is low for tolerant crop, there is little impact on the profits of controllers. Additionally, disease was not eliminated when the crop was tolerant. When the crop was resistant, this also allowed disease elimination to occur at lower proportions of resistant crop ($C = 0.6$ when $\delta_\nu\nu = 1$, (B) and (D)). Other than those scanned over, parameters are as in Table 3.2.

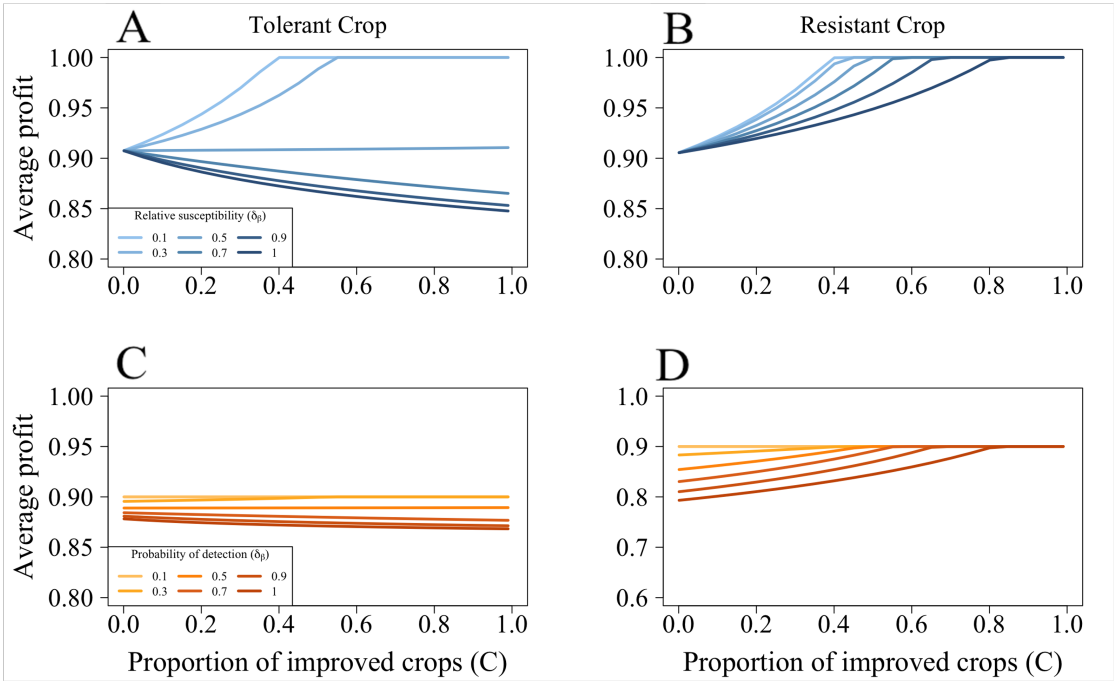

Figure B.3.2: **Change in average profits for tolerant and resistant parameterisation when the relative susceptibility of improved crop (δ_β) is varied.** (A) and (C) show the average profit for unimproved and improved crop respectively for the tolerant parameterisation, whilst (B) and (D) show the same for the resistant parameterisation. We note that by altering the relative susceptibility of tolerant crop, it contravenes the typical definition of tolerance and instead is more akin to quantitative resistance. In all cases, at lower susceptibilities (which means the improved crop is less likely to become infected), profit increases. Indeed, under this parameterisation, disease can go extinct under the tolerant parameterisation (at $C = 0.4$ when $\delta_\beta = 0.1$, (A) and (C)). There is little impact on the average profit for controllers when the improved crop is tolerant (C), as the low loss due to disease in the tolerant parameterisation means that the reduced probability of infection brought about by a lower δ_β has little effect. Disease elimination can occur under all values of δ_β when the improved crop is resistant ((B) and (D)). Even at high values of δ_β, the resistant crop still has a lower relative infectivity (δ_σ) and higher probability of detection (δ_ν) than tolerant crop. Other than those scanned over, parameters are as in Table 3.2.

B.4 Mathematical details of behavioural model

B.4.1 Evaluating stability for behaviour model

To investigate how the initial conditions affected the equilibrium, we conducted a randomisation scan with 10,000 sets of initial conditions and used *nleqslv* (Hasselman & Hasselman (2018)) in R to investigate the number of equilibria attained for each parameter set. The nature of the switching terms means that the system is discontinuous, and the equations will have a different form depending on the values of the state variables. There are ten possible Jacobians, depending on the values of the switching terms (Appendix 1). For each set of equilibrium values found from our randomisation scan, we evaluated the stability of that equilibrium using the appropriate Jacobian matrix.

B.4.2 Basic reproductive number of behavioural model

As the form of the equations differs between the model with fixed proportions and the behavioural model, R_0 must be calculated separately for the behavioural model. For the disease-free equilibrium given by $(S_U, E_U, I_U, S_C, E_C, I_C) = (U, 0, 0, 0, 0, 0)$, all switching terms should be non-zero. As there is no disease, there is no need for control, so no growers should use the control strategy. Additionally, any growers whose fields do become infected will have a lower payoff than the expected payoff of the alternative strategy (as there is no disease), so all non-controllers with infectious fields should switch strategy. The

system of equations is therefore:

$$\frac{dS_C}{dt} = \gamma\theta_C - \delta_\beta\beta S_C(I_U + \delta_\sigma I_C) + M_C - \gamma S_C, \tag{B.1}$$

$$\frac{dE_C}{dt} = \delta_\beta\beta S_C(I_U + \delta_\sigma I_C) - \delta_\epsilon\epsilon E_C - \gamma E_C, \tag{B.2}$$

$$\frac{dI_C}{dt} = \delta_\epsilon\epsilon E_C - \mu_C I_C - \gamma I_C, \tag{B.3}$$

$$\frac{dS_U}{dt} = \gamma\theta_U - \beta S_U(I_U + \delta_\sigma I_C) + M_U - \gamma S_U, \tag{B.4}$$

$$\frac{dE_U}{dt} = \beta S_U(I_U + \delta_\sigma I_C) - \epsilon E_U - \gamma E_U, \tag{B.5}$$

$$\frac{dI_U}{dt} = \epsilon E_U - \mu_U I_U - \gamma I_U. \tag{B.6}$$

where:

$$\theta_C = (1 - z_{SC})S_C + (1 - z_{EC})E_C + (1 - z_{ICH})I_C + z_{IUH}I_U, \tag{B.7}$$

$$\theta_U = S_U + E_U + (1 - z_{IUH})I_U + z_{SC}S_C + z_{EC}E_C + z_{ICH}I_C, \tag{B.8}$$

$$M_C = (1 - z_{ICR})\mu_C I_C + z_{IUR}\mu_U I_U, \tag{B.9}$$

$$M_U = z_{ICR}\mu_C I_C + (1 - z_{IUR})\mu_U I_U, \tag{B.10}$$

$$N = S_C + E_C + I_C + S_U + E_U + I_U. \tag{B.11}$$

Using Equations B.1 - B.6, and the method outlined in Diekmann et al. (2010) and the main text, we find:

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \beta\delta_\sigma N + 0 & 0 & \beta N \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{B.12}$$

$$V = \begin{bmatrix} \gamma + \delta_\epsilon\epsilon & 0 & 0 & 0 \\ -\delta_\epsilon\epsilon & \gamma + \mu_C & 0 & 0 \\ 0 & 0 & \gamma + \epsilon & 0 \\ 0 & 0 & -\epsilon & \gamma + \mu \end{bmatrix} \tag{B.13}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{\gamma+\delta_\epsilon\epsilon} & 0 & 0 & 0 \\ \frac{\delta_\epsilon\epsilon}{(\delta_\epsilon\epsilon+\gamma)(\gamma+\mu_C)} & \frac{1}{\gamma+\mu_C} & 0 & 0 \\ 0 & 0 & \frac{1}{\gamma+\epsilon} & 0 \\ 0 & 0 & \frac{\epsilon}{(\epsilon+\gamma)*(\gamma+\mu)} & \frac{1}{\gamma+\mu} \end{bmatrix} \tag{B.14}$$

The NGM, FV^{-1}, can then be simplified to:

$$K = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ R_{0C} & \frac{\beta\delta_\sigma N}{\gamma+\mu_C} & R_0 & \frac{\beta N}{\gamma+\mu} \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{B.15}$$

where $R_0 = \frac{\beta\epsilon N}{(\mu_U+\gamma)(\epsilon+\gamma)}$ (Equation 3.58) and $R_{0C} = \frac{\delta_\beta\beta\delta_\epsilon\epsilon\delta_\sigma N}{(\mu_C+\gamma)(\delta_\epsilon\epsilon+\gamma)}$ (Equation 3.59). The

eigenvalue of K is given by R_0, so the basic reproduction number is the same regardless of whether behaviour is included in the model, depending only on whether $N = U$ or $N = C$.

B.5 Supplementary results for behavioural model

B.5.1 Underlying behaviour of switching terms for resistant parameterisation with variable cost of control.

The kinks in Figure 3.7b in the main text are caused by changes in the values of the switching terms. As the epidemic progresses, the expected profits for each strategy change. If they fall below the profit for a particular outcome (for example, the profit for a controller with an infected field, P_{IUH}), growers who have earned that outcome switch from having a non-zero probability of switching strategy to never switching. For different values of ϕ_C, this occurs at different values of β (Figure B.5.1).

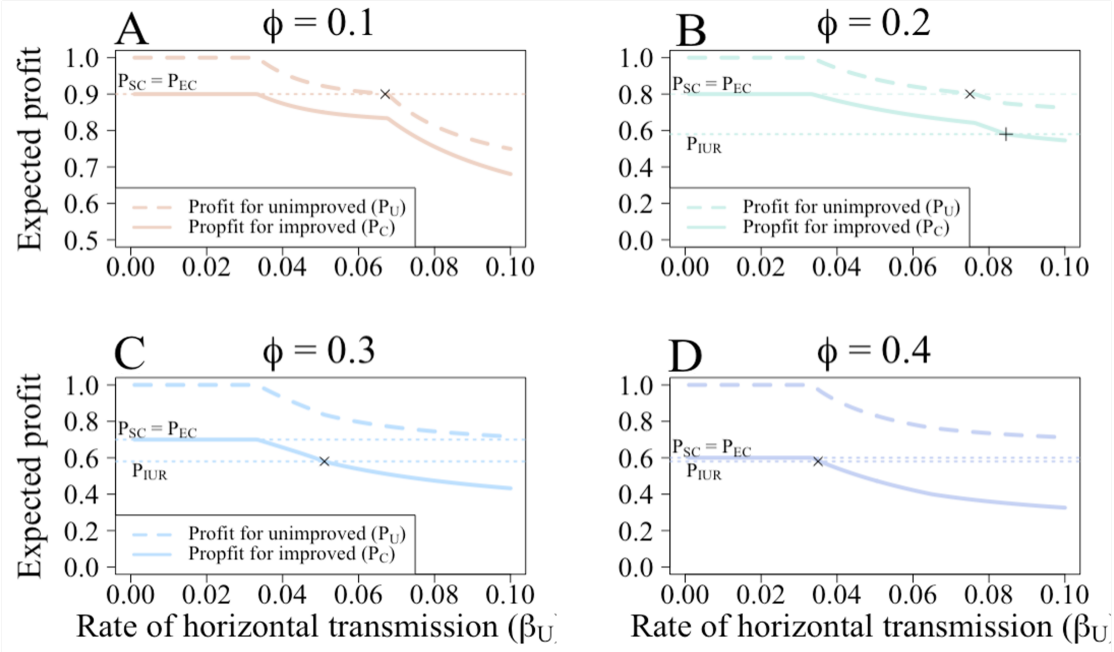

Figure B.5.1: **Change in expected profits with different values of the cost of control (ϕ_C) for resistant parameterisation.** (A) When $\phi_C = 0.1$, the expected profits for non-controllers fall below the expected profits for controllers with susceptible or latently-infected (S_C or E_C) crop at $\beta = 0.068$ day^{-1} ("x"). At this point, those with S_C or E_C fields stop switching strategy. (B) When $\phi_C = 0.2$, S_C and E_C growers stop switching strategy at $\beta = 0.075$ day^{-1} ("x"). At $\beta = 0.0845$ day^{-1} ("+"), non-controllers with infectious fields that have been rogued also stop switching strategy (as $P_C < P_{IUR}$). (C) When $\phi_C = 0.3$, non-controllers with I_{UR} fields stop switching strategy at $\beta = 0.051$ day^{-1} ("x"), and controllers with S_C or E_C fields stop when $\beta = 0.1$ day^{-1}. (D) When $\phi_C = 0.4$, growers with P_{IUR} fields stop switching strategy at $\beta = 0.035$ day^{-1} ("x"). Controllers always have a non-zero probability of switching strategy for this parameter set. Other than those scanned over, parameters are as in Table 3.2.

B.5.2 Expected profits for unimproved, tolerant and resistant crop

The pattern outlined below is the same as that for Figure 3.6 in the main text, though for a two-way scan of the rate of horizontal transmission (β) and the cost of control (ϕ_C).

Irrespective of whether the improved crop was tolerant or resistant, as β increased there was a corresponding increase in the proportion of infectious fields ($I_U + I_C$; Figure B.5.2(A),(B)). This increase occurred more quickly when the improved crop was tolerant, as tolerant crop has the same susceptibility and infectivity as unimproved crop, but is less likely to be detected and removed once infected.

Resistance is incomplete (Table 3.2 in the main text), so fields planted with resistant crop may still be infected. However, the reduced probability of infection means that there are overall lower proportions of infected fields. Participation in control is relatively high for low values of β and ϕ_C, though this decreases as β gets larger (approaches 0.067 day^{-1} in Figure 3.6 in the main text; where it occurs in Figure B.5.2(A),(C) depends on the value of ϕ_C). For these parameter values, P_U approaches $P_{SC,EC}$ (Figure 3.6(A) in the main text), so fewer controllers with susceptible or latently-infected fields should switch strategy. There is still a high infectious pressure, though, so more of these controlled fields will become infected. They will therefore incur the loss due to disease (L_C), resulting in the lowest possible payoff. Expected profits for controllers (and consequently proportion of growers using control) briefly increase when $P_C < P_{IUR}$ (which

occurs at $\beta = 0.067$ day^{-1} in Figure 3.6(A) in the main text). The non-controllers who rogued their fields (achieving P_{IUR}), should no longer switch strategy, and they replant S_U fields. There is still a high probability of infection, however, and many of these non-controlled fields will be infected by the time they are harvested. Some will be rogued before harvesting, preventing some loss of yield. Those that have not rogued achieve a low payoff and switch into the control strategy.

After this point, however, the proportion of controllers falls. The increased infection pressure means that the expected profit of non-controllers is lower than the profit of controllers with susceptible or latently-infected fields ($P_U < P_{SC,EC}$). Controllers who harvest susceptible or latently-infected fields should therefore never consider switching strategy. The high infection pressure, however, means that many of these resistant fields will be infected before they are harvested. As $P_{ICH,ICR} < P_U$ for these values of β, controllers with infectious fields should always switch strategy. As fewer growers control and plant resistant crop, the disease pressure increases and there are more infectious fields.

When the improved crop was tolerant, we chose initial conditions such that there would always be a disease-endemic equilibrium in the bistable region ($I_{U0} + I_{C0} = 0.15, S_{C0} + E_{C0} + I_{C0} = 0.2$, Figure 3.5 in the main text). A high proportion of infectious fields was seen for most parameter combinations, in part due to the lower probability of infectious tolerant fields being removed by roguing. This accompanied a high degree of participation in control, as the low default value of L_C ($= 0.06$) and lower probability of paying the roguing cost favoured tolerant crop. Additionally, once $R_0 > 1$ and the costs

of control < 0.2, an "all-control" equilibrium persists, where $S_C + E_C + I_C = N$.

This "all-control" equilibrium was not seen in the parameterisation where the improved crop was resistant. This is both due to the positive externalities generated by the crop reducing the probability of infection for non-controllers and the structure of the model, which means growers with infectious resistant crop should always have a non-zero probability of switching strategy (as P_{ICR} is the lowest payoff).

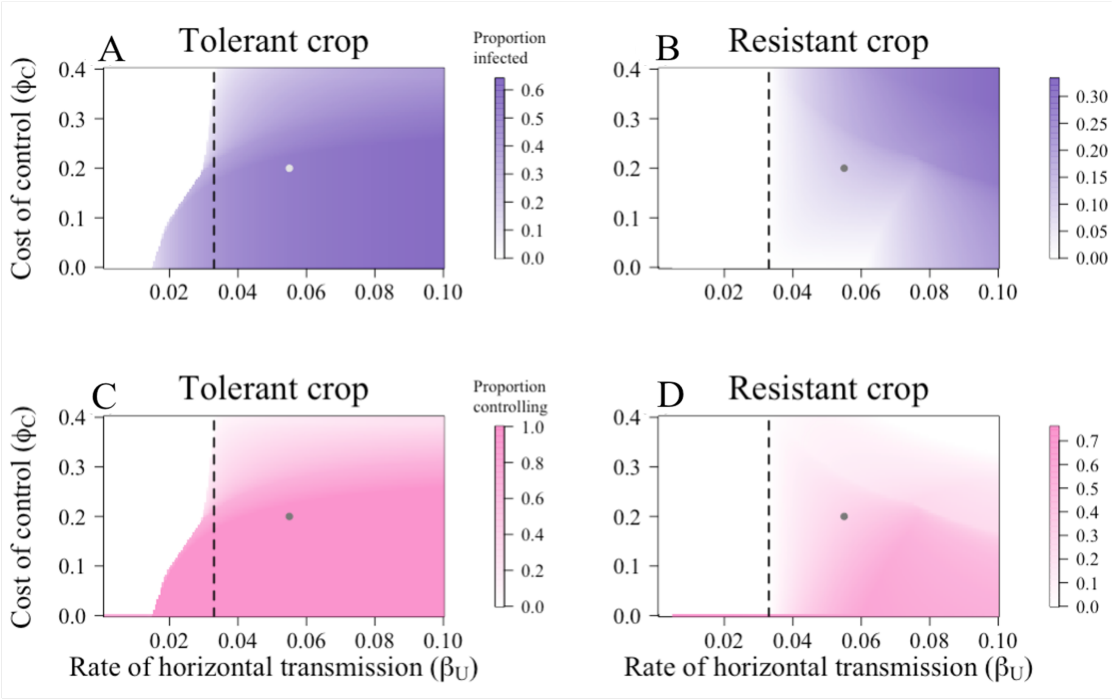

Figure B.5.2: **Response of the number of infectious fields and participation in control to the rate of horizontal transmission in non-improved crops (β) and the cost of control (ϕ_C).** (A) The change in proportion of infectious fields ($I_U + I_C$) and (B) change in participation in control when the improved crop has tolerant characteristics. Equivalent plots for resistant improved crop are shown in (C) and (D). In all graphs, the vertical dashed line at $\beta = 0.0333$ day $^{-1}$ is where $R_0 = 1$. When the crop used by controllers is tolerant to infection, there are high levels of infection and participation in control. Additionally, at low values of ϕ_C, disease can invade when $R_0 < 1$ (A). For resistant crop, there is a much lower proportion of infectious fields and controllers for most values of β. Other than those being varied, parameters and initial conditions are as in Tables 3.2 and 3.2 respectively.

The expected profits of both controllers and non-controllers follow a similar pattern to that of the proportion of infectious fields and controllers (Figure 3.6 in the main text). In both cases, when $R_0 < 1$ the profit of non-controllers (P_U) is equal to that of susceptible/latently-infected non-controllers (except where bistability exists in the

tolerant parameterisation). Once disease invades, profits of both controllers and non-controllers fall. However, for the majority of values of β and ϕ_C, P_U is higher when there is resistant crop than when there is tolerant crop, indicating that non-controllers benefit more from the presence of resistant crop. However, the profits of growers using tolerant crops were generally higher than those using resistant crops, as the benefits generated by tolerant crops were experienced privately by the growers using them.

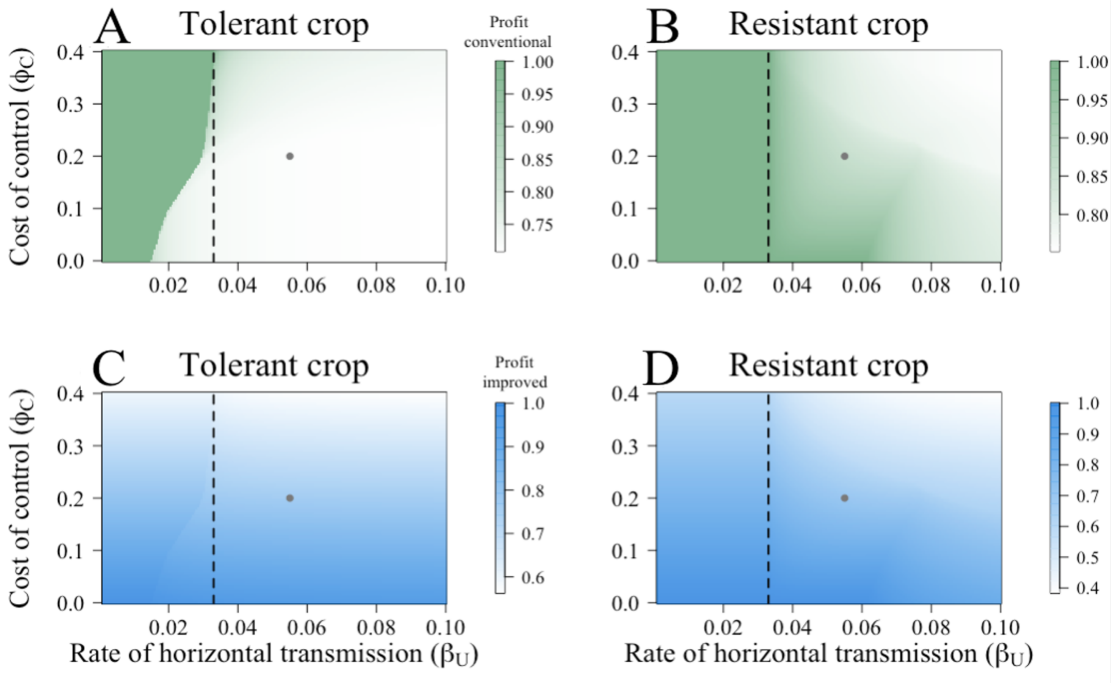

Figure B.5.3: **Response of the expected profits to the rate of horizontal transmission in non-improved crops (β) and the cost of control (ϕ_C).** (A) and (C) are the expected profits for non-controllers (P_U) and controllers (P_C) respectively when the improved crop is tolerant; (B) and (D) show the same for resistant crop. The highest profits for non-controllers are seen when $R_0 < 1$, though P_U is generally higher for those in the resistant-crop scenario than the tolerant crop. Conversely, controllers who used tolerant crop generally had higher profits than those using resistant crop. The grey dots indicate the default parameterisation. Other than those being varied, parameters and initial conditions are as in Tables 3.2 and 3.2 respectively.

Appendix to Chapter 4

C.1 Calculating the expected profit

To calculate the expected profit, we must first calculate the profits associated with each possible outcome a grower could achieve at the time of harvest (or when a field is rogued). These will depend on the control strategy used by the grower (whether they planted tolerant, resistant, or unimproved crop at the beginning of the season), the infectious status of their field and whether an infectious field was rogued.

The profits for each field type are given as follows:

P_{SU} = Profit for non-controller with a susceptible field,

$$= Y, \tag{C.1}$$

P_{EU} = Profit for non-controller with latently-infected field,

$$= Y, \tag{C.2}$$

P_{I_HU} = Profit for non-controller with an infected field that was not rogued,

$$= Y - L, \tag{C.3}$$

P_{I_RU} = Profit for non-controller with an infected field that was rogued,

$$= Y - \phi_Q L, \tag{C.4}$$

P_{ST} = Profit for controller using tolerant crop with a susceptible field,

$$= \delta_{Y_T} Y - \phi_T, \tag{C.5}$$

P_{ET} = Profit for controller using tolerant crop with latently-infected field,

$$= \delta_{Y_T} Y - \phi_T, \tag{C.6}$$

P_{I_HT} = Profit for controller using tolerant crop with an infected field that was not rogued,

$$= \delta_{Y_T} Y - \phi_T - L_T, \tag{C.7}$$

P_{I_RT} = Profit for controller using tolerant crop with an infected field and that was rogued,

$$= \delta_{Y_T} Y - \phi_T - \phi_Q L_T. \tag{C.8}$$

P_{SR} = Profit for controller using resistant crop with a susceptible field,

$$= \delta_{Y_R} Y - \phi_R, \tag{C.9}$$

P_{ER} = Profit for controller using resistant crop with latently-infected field,

$$= \delta_{Y_R} Y - \phi_R, \tag{C.10}$$

P_{I_HR} = Profit for controller using resistant crop with an infected field that was not rogued,

$$= \delta_{Y_R} Y - \phi_R - L_R, \tag{C.11}$$

P_{I_RR} = Profit for controller using resistant crop with an infected field and that was rogued,

$$= \delta_{Y_R} Y - \phi_R - \phi_Q L_R. \tag{C.12}$$

The expected profits for each strategy depend on the probabilities of a grower receiving each of these payoffs.

For each crop variety, the probability of infection (q_j) is given as:

$$\begin{aligned} q_j = & \text{ Instantaneous probability of horizontal infection,} \\ = & \frac{\text{Instantaneous infection rate}}{\text{Instantaneous infection rate + Harvesting rate}}, \end{aligned}$$

so

$$q_U = \frac{\beta(I_U + \delta_{\sigma_T} I_T + \delta_{\sigma_R} I_R)}{\beta(I_U + I_T + \sigma I_U) + \gamma}, \tag{C.13}$$

$$q_T = \frac{\delta_{\beta_T}\beta(I_U + \delta_{\sigma_T} I_T + \delta_{\sigma_R} I_R)}{\delta_{\delta_{\beta_T}\beta}(I_U + I_T + \sigma I_U) + \gamma}, \tag{C.14}$$

$$q_R = \frac{\delta_{\delta_{\beta_R}\beta}(I_U + \delta_{\sigma_T} I_T + \delta_{\sigma_R} I_R)}{\delta_{\delta_{\beta_R}\beta}(I_U + I_T + \sigma I_U) + \gamma}. \tag{C.15}$$

The expected profit, however, will also be dependent on whether a grower's field is harvested whilst latently infected (q_{Eb}) or proceeds to become fully infectious (q_{Ib}). The probability of being harvested whilst latently infected is given as:

$$q_{EU} = \frac{\gamma}{\epsilon + \gamma}, \tag{C.16}$$

$$q_{ET} = \frac{\gamma}{\delta_{\epsilon_T}\epsilon + \gamma}, \tag{C.17}$$

$$q_{ER} = \frac{\gamma}{\delta_{\epsilon_R}\epsilon + \gamma}, \tag{C.18}$$

and the probability of becoming fully infectious before harvest is:

$$q_{IU} = \frac{\epsilon}{\epsilon + \gamma}, \tag{C.19}$$

$$q_{IT} = \frac{\delta_{\epsilon_T}\epsilon}{\delta_{\epsilon_T}\epsilon + \gamma}, \tag{C.20}$$

$$q_{IR} = \frac{\delta_{\epsilon_R}\epsilon}{\delta_{\epsilon_R}\epsilon + \gamma}. \tag{C.21}$$

Fully infectious crops can either be harvested or removed via roguing. The probability that harvesting occurs before roguing ($q_{I_H b}$) is given by:

$$q_{I_H b} = q_{Ib}\left(\frac{\gamma}{\gamma + \mu_b}\right), \tag{C.22}$$

and that it is rogued before harvesting ($q_{I_R b}$) is:

$$q_{I_R b} = q_{Ib}\left(\frac{\mu_b}{\gamma + \mu_b}\right), \tag{C.23}$$

We can then use these probabilities to calculate the expected profit of each strategy:

P_U = Grower's estimate of the expected profit next season if control is not adopted,

$$= (1 - q_U)P_{SU} + q_{EU}P_{EU} + q_{I_HU}P_{I_HU} + q_{I_RU}P_{I_RU}, \tag{C.24}$$

P_T = Grower's estimate of the expected profit next season if tolerant crop is used,

$$= (1 - q_T)P_{ST} + q_{ET}P_{ET} + q_{I_HT}P_{I_HT} + q_{I_RT}P_{I_RT}, \tag{C.25}$$

P_R = Grower's estimate of the expected profit next season if resistant crop is used,

$$= (1 - q_R)P_{SR} + q_{ER}P_{ER} + q_{I_HR}P_{I_HR} + q_{I_RR}P_{I_RR}. \tag{C.26}$$

We can simplify these expressions to:

$$P_U = Y - q_U \frac{\epsilon}{\epsilon + \gamma} L \left(\frac{\gamma}{\gamma + \mu_U} + \frac{\mu_U}{\mu_U + \gamma} \phi_Q \right), \tag{C.27}$$

$$P_T = \delta_{Y_T} Y - \phi_T - q_T \frac{\delta_{\epsilon_T} \epsilon}{\delta_{\epsilon_T} \epsilon + \gamma} \delta_{L_T} L \left(\frac{\gamma}{\gamma + \mu_T} + \frac{\mu_T}{\mu_T + \gamma} \phi_Q \right), \tag{C.28}$$

$$P_R = \delta_{Y_R} Y - \phi_R - q_R \frac{\delta_{\epsilon_R} \epsilon}{\delta_{\epsilon_R} \epsilon + \gamma} \delta_{L_R} L \left(\frac{\gamma}{\gamma + \mu_R} + \frac{\mu_R}{\mu_R + \gamma} \phi_Q \right). \tag{C.29}$$

C.2 Details of switching terms

The switching terms take the general form: a grower with outcome P_{ab}, is switching into strategy c or d, $a \in \{S, E, I_H, I_R\}$ and $b, c, d \in \{U, T, R\}, b \neq c \neq d$. If the profits of both c and d are exactly equal, the grower will switch into whichever strategy has the higher proportion of current users. If both the expected profits and the proportion of growers using each strategy are the same, then half of the growers considering changing strategy will compare with each alternative expected profit.

The full details of all the switching terms are given below. If $P_T < P_R$ or $P_T = P_R$ and $R > T$ (i.e. there are more using resistant than tolerant crop), then

$$z_{SUR} = \max(0, 1 - \exp(-\eta(P_R - P_{SU})), \tag{C.30}$$

$$z_{EUR} = \max(0, 1 - \exp(-\eta(P_R - P_{EU})), \tag{C.31}$$

$$z_{I_HUR} = \max(0, 1 - \exp(-\eta(P_R - P_{I_HU})), \tag{C.32}$$

$$z_{I_RUR} = \max(0, 1 - \exp(-\eta(P_R - P_{I_RU})), \tag{C.33}$$

$$z_{SUT} = 0, \tag{C.34}$$

$$z_{EUT} = 0, \tag{C.35}$$

$$z_{I_HUT} = 0, \tag{C.36}$$

$$z_{I_RUT} = 0. \tag{C.37}$$

If $P_T > P_R$ or $P_T = P_R$ and $R < T$, then

$$z_{SUR} = 0, \tag{C.38}$$

$$z_{EUR} = 0, \tag{C.39}$$

$$z_{I_HUR} = 0, \tag{C.40}$$

$$z_{I_RUR} = 0, \tag{C.41}$$

$$z_{SUT} = \max(0, 1 - \exp(-\eta(P_T - P_{SU})) \tag{C.42}$$

$$z_{EUT} = \max(0, 1 - \exp(-\eta(P_T - P_{EU})) \tag{C.43}$$

$$z_{I_HUT} = \max(0, 1 - \exp(-\eta(P_T - P_{I_HU})) \tag{C.44}$$

$$z_{I_RUT} = \max(0, 1 - \exp(-\eta(P_T - P_{I_RU})). \tag{C.45}$$

If $P_T < P_U$ or $P_T = P_U$ and $U > T$, then

$$z_{SRU} = \max(0, 1 - \exp(-\eta(P_U - P_{SR})), \tag{C.46}$$

$$z_{ERU} = \max(0, 1 - \exp(-\eta(P_U - P_{ER})), \tag{C.47}$$

$$z_{I_H RU} = \max(0, 1 - \exp(-\eta(P_U - P_{I_H R})), \tag{C.48}$$

$$z_{I_R RU} = \max(0, 1 - \exp(-\eta(P_U - P_{I_R R})), \tag{C.49}$$

$$z_{SRT} = 0, \tag{C.50}$$

$$z_{ERT} = 0, \tag{C.51}$$

$$z_{I_H RT} = 0, \tag{C.52}$$

$$z_{I_R RT} = 0. \tag{C.53}$$

If $P_T > P_U$ or $P_T = P_U$ and $U < T$, then

$$z_{SRU} = 0, \tag{C.54}$$

$$z_{ERU} = 0, \tag{C.55}$$

$$z_{I_HRU} = 0, \tag{C.56}$$

$$z_{I_RRU} = 0, \tag{C.57}$$

$$z_{SRT} = \max(0, 1 - \exp(-\eta(P_T - P_{SR})), \tag{C.58}$$

$$z_{ERT} = \max(0, 1 - \exp(-\eta(P_T - P_{ER})), \tag{C.59}$$

$$z_{I_HRT} = \max(0, 1 - \exp(-\eta(P_T - P_{I_HR})), \tag{C.60}$$

$$z_{I_RRT} = \max(0, 1 - \exp(-\eta(P_T - P_{I_RR})). \tag{C.61}$$

If $P_U > P_R$ or $P_U = P_R$ and $R < U$, then

$$z_{STU} = \max(0, 1 - \exp(-\eta(P_U - P_{ST})), \tag{C.62}$$

$$z_{ETU} = \max(0, 1 - \exp(-\eta(P_U - P_{ET})), \tag{C.63}$$

$$z_{I_HTU} = \max(0, 1 - \exp(-\eta(P_U - P_{I_HT})), \tag{C.64}$$

$$z_{I_RTU} = \max(0, 1 - \exp(-\eta(P_U - P_{I_RT})), \tag{C.65}$$

$$z_{STR} = 0, \tag{C.66}$$

$$z_{ETR} = 0, \tag{C.67}$$

$$z_{I_HTR} = 0, \tag{C.68}$$

$$z_{I_RTR} = 0. \tag{C.69}$$

If $P_U < P_R$ or $P_U = P_R$ and $R > U$, then

$$z_{STU} = 0, \tag{C.70}$$

$$z_{ETU} = 0, \tag{C.71}$$

$$z_{I_HTU} = 0, \tag{C.72}$$

$$z_{I_RTU} = 0, \tag{C.73}$$

$$z_{STR} = \max(0, 1 - \exp(-\eta(P_R - P_{ST})), \tag{C.74}$$

$$z_{ETR} = \max(0, 1 - \exp(-\eta(P_R - P_{ET})), \tag{C.75}$$

$$z_{I_HTR} = \max(0, 1 - \exp(-\eta(P_R - P_{I_HT})), \tag{C.76}$$

$$z_{I_RTR} = \max(0, 1 - \exp(-\eta(P_R - P_{I_RT})). \tag{C.77}$$

In the case where both the profits and the proportion of growers using the alternative strategy are equal, growers changing strategy will be divided evenly between the two alternative strategies.

To simplify writing the model, we can then say:

$$z_{SU} = \max(z_{SUT}, z_{SUR}), \tag{C.78}$$

$$z_{EU} = \max(z_{EUT}, z_{EUR}), \tag{C.79}$$

$$z_{I_HU} = \max(z_{I_HUT}, z_{I_HUR}), \tag{C.80}$$

$$z_{I_RU} = \max(z_{I_RUT}, z_{I_RUR}), \tag{C.81}$$

$$z_{ST} = \max(z_{STU}, z_{STR}), \tag{C.82}$$

$$z_{ET} = \max(z_{ETU}, z_{ETR}), \tag{C.83}$$

$$z_{I_HT} = \max(z_{I_HTU}, z_{I_HTR}), \tag{C.84}$$

$$z_{I_RT} = \max(z_{I_RTU}, z_{I_RTR}), \tag{C.85}$$

$$z_{SR} = \max(z_{SRU}, z_{SRT}), \tag{C.86}$$

$$z_{ER} = \max(z_{ERU}, z_{ERT}), \tag{C.87}$$

$$z_{I_HR} = \max(z_{I_HRU}, z_{I_HRT}), \tag{C.88}$$

$$z_{I_RR} = \max(z_{I_RRU}, z_{I_RRT}). \tag{C.89}$$

C.3 Calculating the basic reproduction number for the model

At the disease-free equilibrium, if growers have a choice between strategies they would never chose to pay for control. Thus, in the behavioural model, the disease-free equilibrium is given as: $(S_U, E_U, I_U, S_T, E_T, I_T, S_R, E_R, I_R) = (N, 0, 0, 0, 0, 0, 0, 0, 0)$.

For this model, $F =$

$$F = \begin{bmatrix} 0 & \beta_U N & 0 & \beta_U N & 0 & \sigma\beta_U N \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \tag{C.90}$$

and

$$V = \begin{bmatrix} \epsilon + \gamma & 0 & 0 & 0 & 0 & 0 \\ -\epsilon & \mu_U + \gamma & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta_{\epsilon_T}\epsilon + \gamma & 0 & 0 & 0 \\ 0 & 0 & -\delta_{\epsilon_T}\epsilon & \mu_T + \gamma & 0 & 0 \\ 0 & 0 & 0 & 0 & \delta_{\epsilon_R}\epsilon + \gamma & 0 \\ 0 & 0 & 0 & 0 & -\delta_{\epsilon_R}\epsilon & \mu_R + \gamma \end{bmatrix}. \tag{C.91}$$

The inverse of V is given by:

$$V^{-1} = \begin{bmatrix} \frac{1}{\epsilon+\gamma} & 0 & 0 & 0 & 0 & 0 \\ \frac{\epsilon}{(\epsilon+\gamma)(\mu_U+\gamma)} & \frac{1}{\mu_U+\gamma} & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{\delta_{\epsilon_T}\epsilon+\gamma} & 0 & 0 & 0 \\ 0 & 0 & \frac{\delta_{\epsilon_T}\epsilon}{(\delta_{\epsilon_T}\epsilon+\gamma)(\mu_T+\gamma)} & \frac{1}{\gamma+\mu_T} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{\delta_{\epsilon_R}\epsilon+\gamma} & 0 \\ 0 & 0 & 0 & 0 & \frac{\delta_{\epsilon_R}\epsilon}{(\delta_{\epsilon_R}\epsilon+\gamma)(\gamma+\mu_R)} & \frac{1}{\mu_R+\gamma} \end{bmatrix}. \quad \text{(C.92)}$$

The NGM, $K = FV^{-1}$, is then given by:

$$K = \begin{bmatrix} \frac{\beta_U\epsilon N}{(\epsilon+\gamma)(\mu_U+\gamma)} & \frac{\beta_U N}{\mu_U+\gamma} & \frac{\beta_U\delta_{\epsilon_T}\epsilon N}{(\delta_{\epsilon_T}\epsilon+\gamma)(\mu_T+\gamma)} & \frac{\beta_U N}{\mu_T+\gamma} & \frac{\beta_U\delta_{\epsilon_R}\epsilon\sigma N}{(\delta_{\epsilon_R}\epsilon+\gamma)(\mu_R+\gamma)} & \frac{\beta_U\sigma N}{\mu_R+\gamma} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \quad \text{(C.93)}$$

The leading eigenvalue for this matrix is

$$R_0 = \frac{\beta_U\epsilon N}{(\epsilon+\gamma)(\mu_U+\gamma)}. \quad \text{(C.94)}$$

C.4 Supplementary results

C.4.1 Change in switching points for parameter scans

The underlying bistability of the model means that, for a given set of parameters, different equilibria can be attained depending on the initial conditions. This manifests in our parameter scans over the relative loss due to disease in tolerant crop (δ_{L_T}) and the relative susceptibility in resistant crop (δ_{β_R}) (Figures 4.4 and 4.5 in Chapter 4).

In these parameter scans, the initial conditions influence the parameter value at which the system switches from a mixed "tolerant and unimproved crop" equilibrium to a "resistant and unimproved crop" equilibrium in the δ_{L_T} parameter scan (and vice versa in the δ_{β_R} scan). When the switch occurs depends strongly on the initial proportion of tolerant crop (Figures C.4.2 and C.4.1, which show the "extreme" parameter values for which the switch can occur). Importantly, this does not change the parameter values where growers of infected tolerant crop should switch strategy, as this occurs within the range of δ_{L_T} that is unaffected by bistability (Figure C.4.1 (B) and (D)).

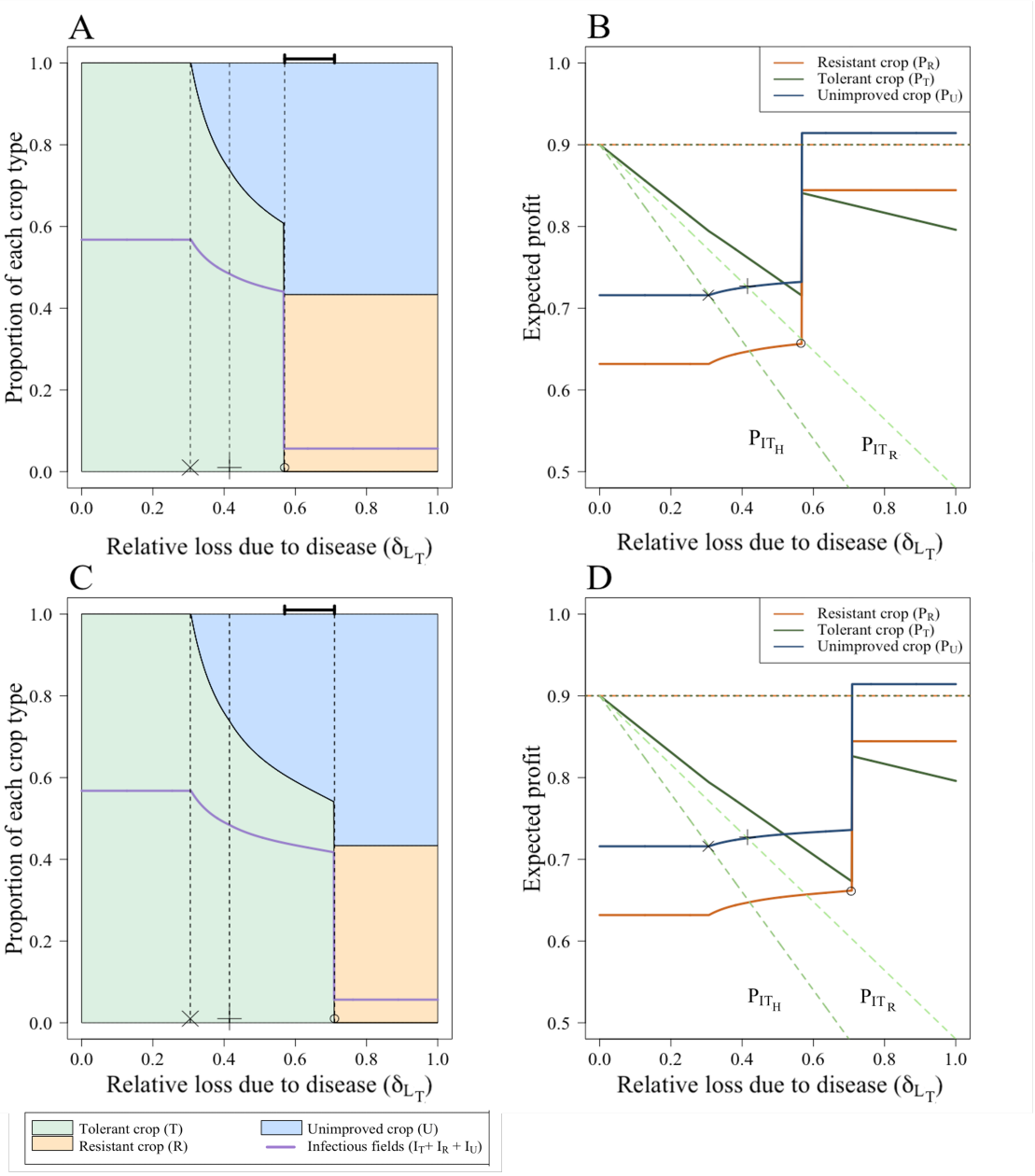

Figure C.4.1: **Parameter scans showing range of δ_{L_T} values during which switch in equilibria can occur.**(A) + (B) When none of the fields are initially planted with tolerant crop ($T_0 = 0$), the switch to the "resistant and unimproved" equilibrium occurs at $\delta_{L_T} = 0.51$ ("O"). (C) + (D) When all of the fields were initially planted with tolerant crop, the switch to the "resistant and unimproved" doesn't occur until $\delta_{L_T} = 0.51$ ("O").

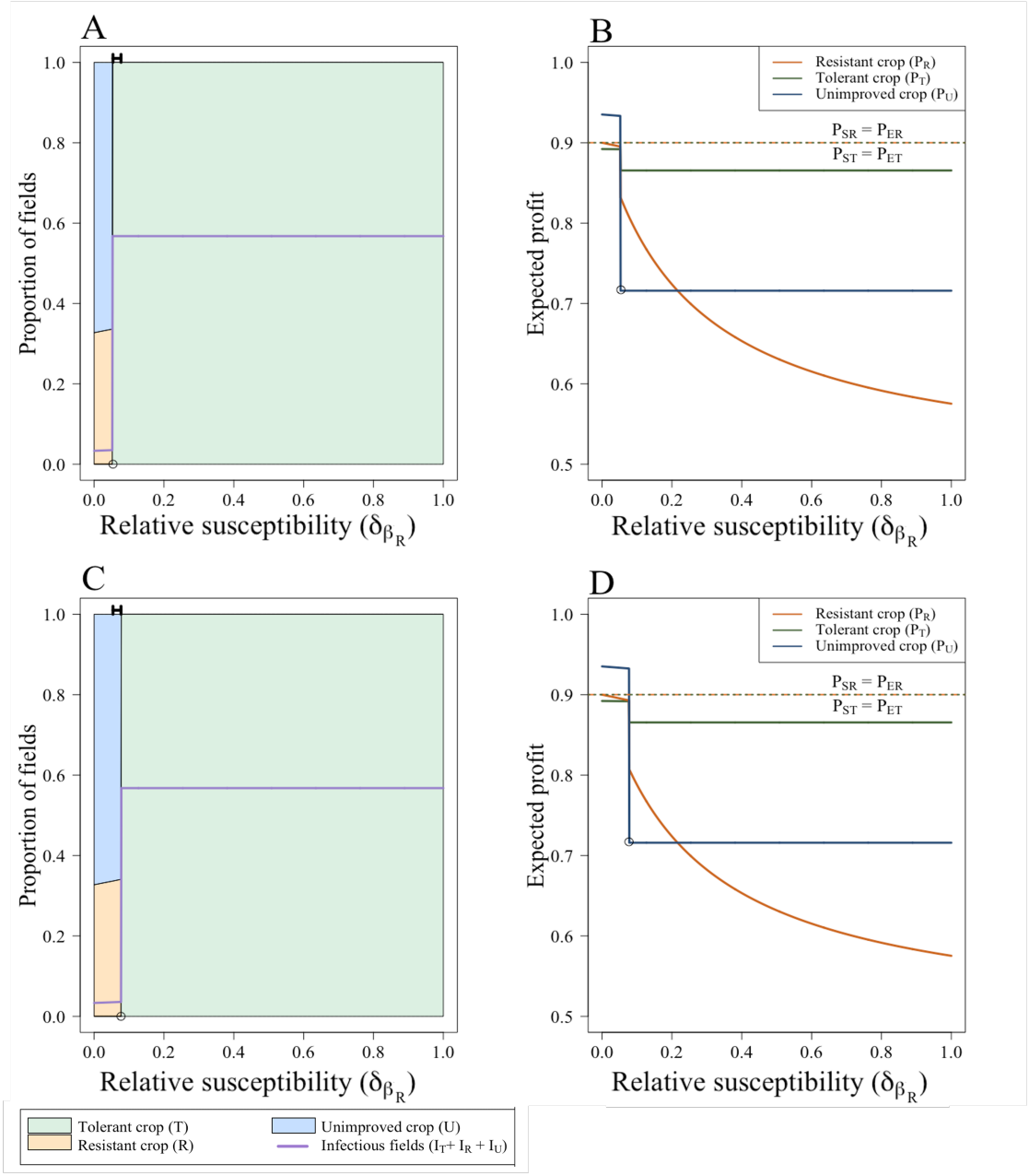

Figure C.4.2: **Parameter scans showing range of δ_{β_T} values during which switch in equilibria can occur.** When none of the fields are initially planted with tolerant crop ($T_0 = 0$), the switch to the "resistant and unimproved crop" equilibrium occurs at a lower value of δ_{β_R}. When all of the fields are initially planted with tolerant crop ($T_0 = 0$; (A)), the switch to the "resistant and unimproved" equilibrium occurs at lower values of δ_{β_R} than if $T_0 = 1$; (B).

C.4.2 Time courses for subsidisation schemes

The Pareto front is bounded by two extremes: one where the profit of growers is maximised ($P = 1$), and the other when the cost to the planner is minimised ($\tau = 0$). Both of these scenarios have a Gini coefficient of 0.5. To prioritise the growers' profits, the cost of both resistant and tolerant crop must be low ($\phi_R = 0$ and $\phi_T = 0.05$). Conversely, to minimise the cost to the planner, both crops must be expensive for the growers ($\phi_R = 0.4$ and $\phi_T = 0.375$).

The fairest scenario ($G = 0.009$) occurs when $\tau = 0.13$ and $P = 0.93$. To achieve this, the cost of tolerant crop (ϕ_T) is 0.12, and the cost of resistant crop (ϕ_R) is 0.105.

Here, we show the dynamics of the model for each scenario. In both the "fairest" scenario and when the profits to the grower are preferred, the subsidisation scheme results in a high proportion using resistant crop and very low levels of disease (Figure C.4.3(A)-(B)). When the costs to the planner are minimised, then no growers use resistant crop, and very few use tolerant crop (which is not subsidised, but still provides a reduced loss in yield if infected).

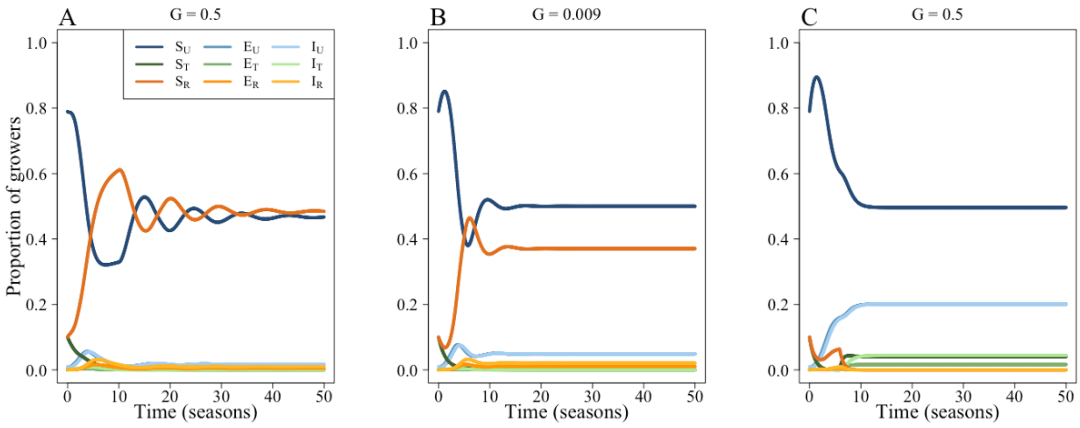

Figure C.4.3: **Time courses for the most- and least-fair subsidisation schemes when the tolerant and resistant crop are inefficient.** (A) Dynamics that maximise the profits to the grower. Here, $\approx 49.8\%$ of growers use resistant crop. (B) The fairest scenario, where both the profits and costs have relatively equal weighting. In this case, $\approx 40.3\%$ use resistant crop. (C) When minimising the cost to the planner is prioritised, $\approx 10\%$ of growers use tolerant crop.

C.4.3 Effect of change in cost of improved crop on the proportion of tolerant or resistant crops

When the cost of resistant (ϕ_R) and tolerant (ϕ_T) crops are varied, there is only a narrow parameter space where both crop types can coexist (Figure 4.6(D) in the main text). For the majority of the parameter space, two distinct regions emerge: one where growers use resistant and unimproved crop, and one where growers use tolerant and unimproved crop. The Pareto front lies within the "resistant and unimproved" equilibrium, save for the solution at $\phi_T = 0.375$ and $\phi_R = 0.4$, where no growers use resistant crop and 10% use tolerant crop.

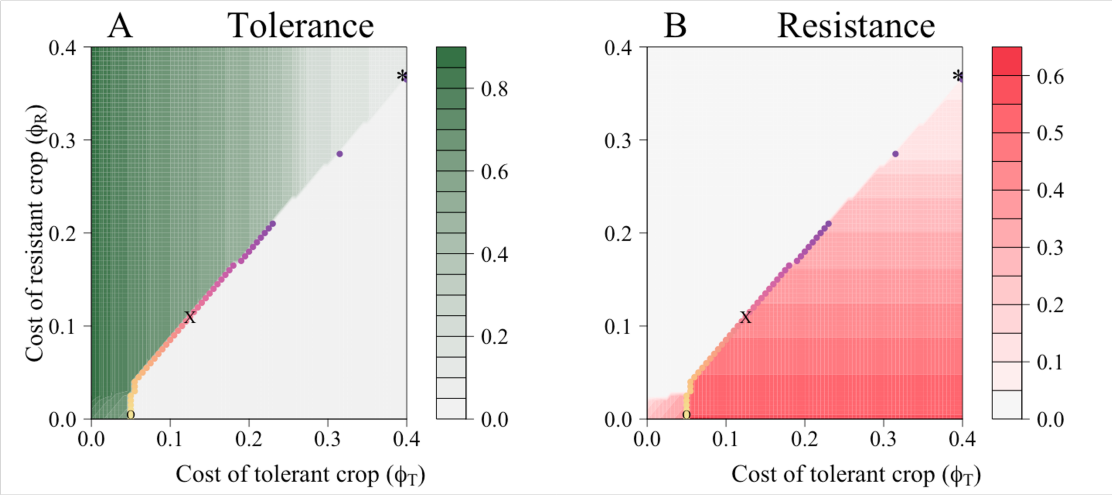

Figure C.4.4: **Effect of change in the price of tolerant (ϕ_T) and resistant (ϕ_R) crop on the proportion of growers using improved crop over time.** Change in uptake of (A) tolerant crop and (B) resistant crop. Along the Pareto front, growers are always in a "resistant and unimproved" equilibrium, except for the point at $\phi_T = 0.375$ and $\phi_R = 0.4$, where no growers use resistant crop and 10% of growers use tolerant crop. The fairest scenario is marked with a "X" (Gini coefficient = 0.009, when $\tau = 0.13$ and $P = 0.93$). The least fair scenarios ($G = 0.5$) are marked with "O" when the profit is prioritised, and "*" when the costs to the planner are.

Appendix to Chapter 5

D.1 Uptake of resistant crop after 50 seasons

The use of highly-effective resistant crop introduces the possibility for disease extinction (as tolerant crop does not restrict TYLCV replication, disease extinction is not promoted). When disease goes extinct, however, there is no longer any use for resistant crop. Yet there is a time lag between the elimination of TYLCV and the abandonment of resistant crop: though there are very few infected fields after 10 seasons, and in a high proportion of the simulations disease goes extinct, $\approx 38\%$ of growers still use resistant crop (Figure 5.9(B) in the main text). After 50 seasons, if the relative susceptibility is very low ($\delta_\beta < 0.050$) TYLCV always goes extinct, and growers stop using resistant crop (Figure C.4.4). Once $\delta_\beta > 0.050$, more growers use resistant crop, though a large proportion remain "free-riders" and continue planting unimproved crop. Once $\delta_\beta > 0.15$, however, uptake of resistant crop falls. As the resistant crop becomes less effective, fewer growers use it as they are more likely to receive the "suckers payoff" and pay the dual

cost of the loss due to disease and cost of resistant crop.

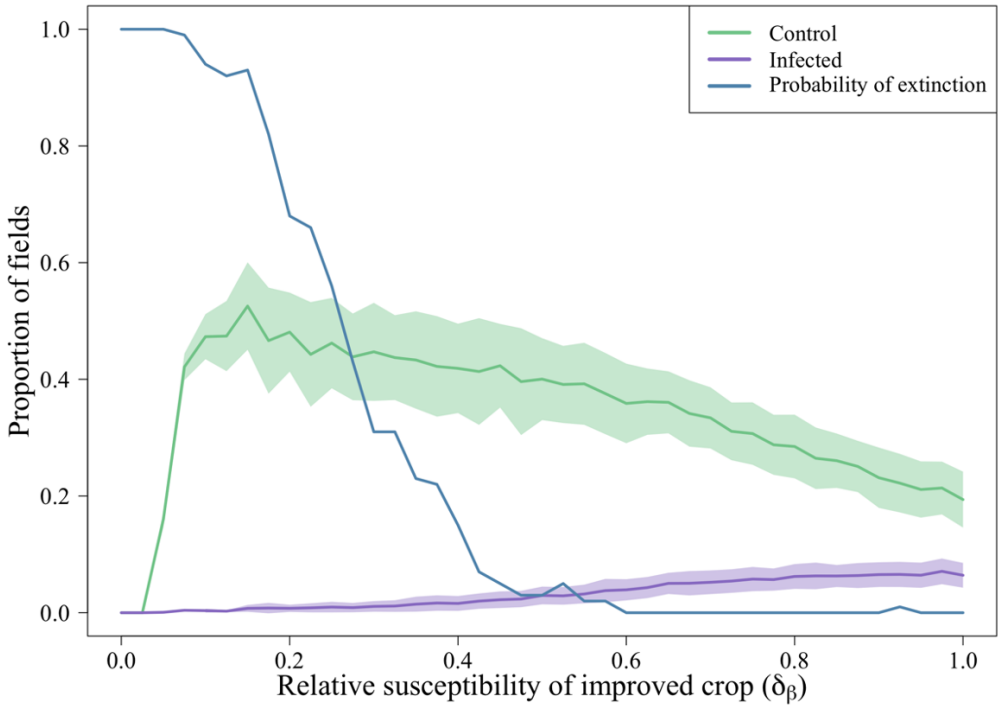

Figure C.4.4: **Effect of the change in the relative susceptibility of resistant crop on its uptake after 50 seasons.** For $\delta_\beta < 0.050$, TYLCV always goes extinct and no growers use resistant crop. Then, as resistant crop becomes more effective (δ_β increases), more growers use resistant crop, though its uptake declines after $\delta_\beta = 0.15$. The reduced efficacy makes resistant crop less worthwhile, so fewer growers use it.

D.2 Clustering of control strategies after 10 seasons

We used Moran's I to evaluate the similarity in control strategy between growers whose fields were in close proximity. Using the parameterisation in Table 5.2, we in-

vestigate the degree of clustering of control strategies and infection after 10 seasons. We measured clustering over 200m (i.e. when calculating Moran's I, we evaluated the similarity in the control and infection status of fields within 200m of the focal grower), though growers could access information from other fields within 100m of their own when assessing profitability.

When growers can only access information on fields within a 100m radius of their own field, there is widespread adoption of tolerant crop (Figure D.1.1). However, there is no evidence of clustering after ten seasons, and indeed the negative Moran's I indicates a degree of dispersion in the locations of like fields in the landscape (Moran's I at season 10: -0.0550, $p \leq 0.05$). As tolerant crop is widespread in the landscape (after 10 seasons 97% of of growers use tolerant crop), this dispersion likely refers to the unimproved crop.

We found that groups of growers using resistant crop were clustered in the landscape (Figure 5.12(B), Moran's I at season 10: 0.0601, standard deviation = 0.0181, $p \leq 0.05$).

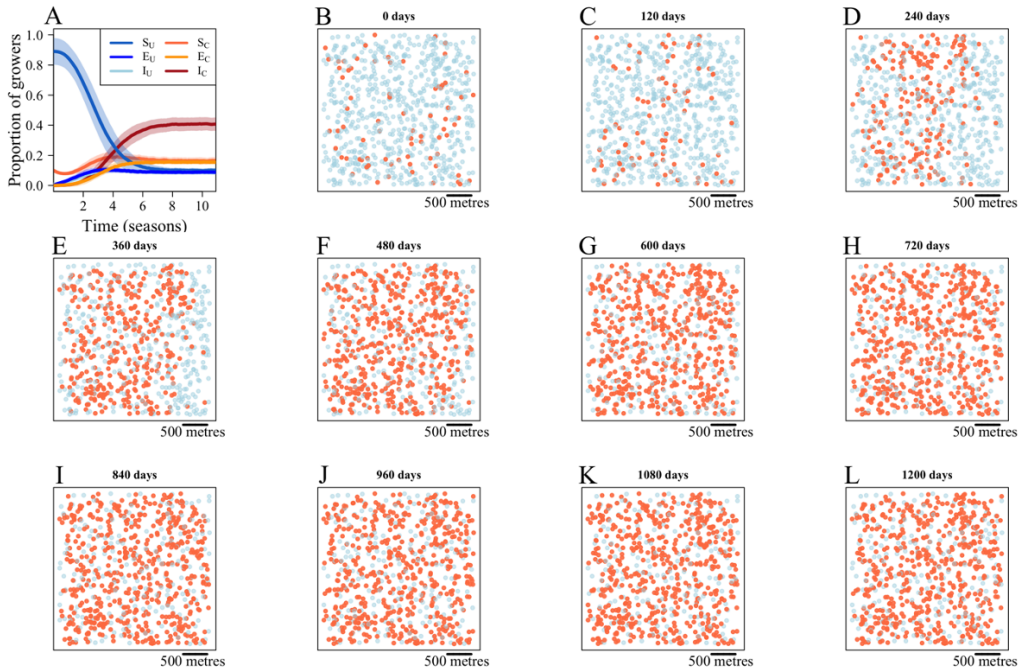

Figure D.1.1: **Adoption of tolerant crop in the landscape when growers can only receive information from growers within 100 m of their field.** Moran's I is 0.0105, $p \leq 0.05$. This indicates a weak positive correlation between the location of fields planted with the same crop type in this landscape.

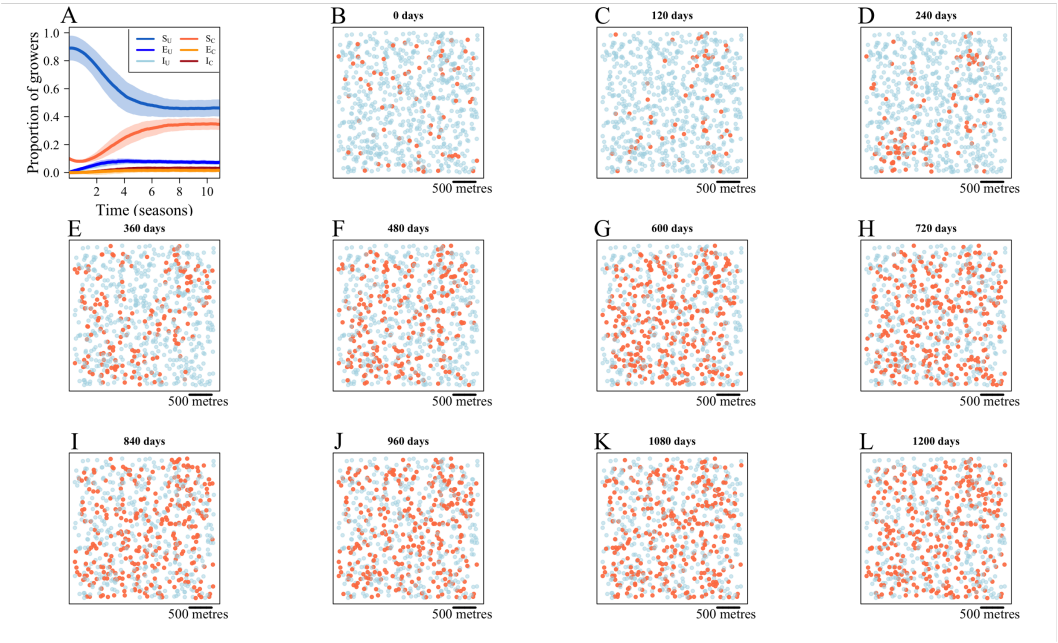

Figure D.1.1: **Adoption of resistant crop in the landscape when growers can only receive information from growers within 100m of their field.** Moran's I for this result is 0.154, standard deviation: 0.0386, $p \leq 0.05$ .This indicates a strong positive correlation between the location of fields planted with resistant crop in this landscape. After ten seasons, 42.3% of growers use resistant crop.

Bibliography

Ahuja, D., Ahuja, U. R., Singh, S. & Singh, N. (2015), 'Comparison of integrated pest management approaches and conventional (non-ipm) practices in late-winter-season cauliflower in Northern India', *Crop Protection* **78**, 232–238.

Ahuja, D., Ahuja, U. R., Srinivas, P., Singh, R., Malik, M., Sharma, P. & Bamawale, O. (2012), 'Development of farmer-led integrated management of major pests of cauliflower cultivated in rainy season in India', *Journal of Agricultural Science* **4**(2), 79.

Ajbar, A., Alqahtani, R. T. & Boumaza, M. (2021), 'Dynamics of an SIR-based COVID-19 model with linear incidence rate, nonlinear removal rate, and public awareness', *Frontiers in Physics* **9**.

URL: *https://www.frontiersin.org/article/10.3389/fphy.2021.634251*

Allen, R. (1978), 'Epidemiological factors influencing the success of roguing for the control of bunchy top disease of bananas in New South Wales', *Australian Journal of Agricultural Research* **29**(3), 535–544.

Allie, F., Pierce, E. J., Okoniewski, M. J. & Rey, C. (2014), 'Transcriptional analysis

of South African cassava mosaic virus-infected susceptible and tolerant landraces of cassava highlights differences in resistance, basal defense and cell wall associated genes during infection', *BMC Genomics* **15**(1), 1–30.

Almeida, R. P., Daane, K. M., Bell, V. A., Blaisdell, G. K., Cooper, M. L., Herrbach, E. & Pietersen, G. (2013), 'Ecology and management of grapevine leafroll disease', *Frontiers in Microbiology* **4**, 94.

Anderson, M. (1984), 'The evolution of eusociality', *Annual Review of Ecology and Systematics* **15**(1), 165–189.

Anon (2022), 'Statistics division of the Food and Agricultural Organization of the United Nations.'.
URL: *https://www.fao.org/faostat/en/*

Arah, I., Amaglo, H., Kumah, E. & Ofori, H. (2015), 'Preharvest and postharvest factors affecting the quality and shelf life of harvested tomatoes: A mini review', *International Journal of Agronomy* **2015**, 1–6.

Archetti, M. & Pienta, K. J. (2019), 'Cooperation among cancer cells: applying game theory to cancer', *Nature Reviews Cancer* **19**(2), 110–117.

Arino, J., Mccluskey, C. & Van Den Driessche, P. (2003), 'Global results for an epidemic model with vaccination that exhibits backward bifurcation', *SIAM Journal on Applied Mathematics* **64**(1), 260–276. cited By 229.

Asante, S., Tamo, M. & Jackai, L. (2001), 'Integrated management of cowpea insect pests using elite cultivars, date of planting and minimum insecticide application', *African Crop Science Journal* **9**(4), 655–665.

Askari, H. & Cummings, J. T. (1977), 'Estimating agricultural supply response with the nerlove model: a survey', *International economic review* pp. 257–292.

Aubert, M. & Enjolras, G. (2022), 'Intensive and extensive impacts of EU subsidies on pesticide expenditures at the farm level', *Journal of Environmental Economics and Policy* **11**(2), 218–234.

Austen-Smith, D. & Banks, J. S. (1998), 'Social choice theory, game theory, and positive political theory', *Annual Review of Political Science* **1**, 259–287.

Bassanezi, R. B., Montesino, L. H., Gimenes-Fernandes, N., Yamamoto, P. T., Gottwald, T. R., Amorim, L. & Filho, A. B. (2013), 'Efficacy of area-wide inoculum reduction and vector control on temporal progress of huanglongbing in young sweet orange plantings', *Plant Disease* **97**(6), 789–796.

Bate, A. M., Jones, G., Kleczkowski, A. & Touza, J. (2021), 'Modelling the effectiveness of collaborative schemes for disease and pest outbreak prevention', *Ecological Modelling* **442**, 109411.
URL: *https://www.sciencedirect.com/science/article/pii/S0304380020304701*

Bauch, C. T. (2005), 'Imitation dynamics predict vaccinating behaviour', *Proceedings of the Royal Society B: Biological Sciences* **272**(1573), 1669–1675.

Bauch, C. T. & Bhattacharyya, S. (2012), 'Evolutionary game theory and social learning can determine how vaccine scares unfold', *PLOS Computational Biology* **8**(4), 1–12.
URL: *https://doi.org/10.1371/journal.pcbi.1002452*

Bauch, C. T. & Earn, D. J. D. (2004), 'Vaccination and the theory of games', *Proceedings of the National Academy of Sciences* **101**(36), 13391–13394.

Bauch, C. T., Galvani, A. P. & Earn, D. J. D. (2003), 'Group interest versus self-interest in smallpox vaccination policy', *Proceedings of the National Academy of Sciences* **100**(18), 10564–10567.
URL: *https://www.pnas.org/content/100/18/10564*

Belay, D. G. & Ayalew, H. (2020), 'Nudging farmers in crop choice using price information: Evidence from Ethiopian commodity exchange', *Agricultural Economics* **51**(5), 793–808.

Bell, J. R., Burkness, E. C., Milne, A. E., Onstad, D. W., Abrahamson, M., Hamilton, K. L. & Hutchison, W. D. (2012), 'Putting the brakes on a cycle: bottom-up effects damp cycle amplitude', *Ecology Letters* **15**(4), 310–318.

Ber, R., Navot, N., Zamir, D., Antignus, Y., Cohen, S. & Czosnek, H. (1990), 'Infection of tomato by the tomato yellow leaf curl virus: susceptibility to infection, symptom development, and accumulation of viral dna', *Archives of Virology* **112**, 169–80.

Bhattacharyya, S. & Bauch, C. T. (2011), '"wait and see" vaccinating behaviour during a pandemic: a game theoretic analysis', *Vaccine* **29**(33), 5519–5525.

Bhattacharyya, S., Vutha, A. & Bauch, C. T. (2019), 'The impact of rare but severe vaccine adverse events on behaviour-disease dynamics: a network model', *Scientific Reports* **9**(1), 1–13.

Bingham, I., Walters, D., Foulkes, M. & Paveley, N. (2009), 'Crop traits and the tolerance of wheat and barley to foliar disease', *Annals of Applied Biology* **154**(2), 159–173.
URL: *https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1744-7348.2008.00291.x*

Boehmke, B. C., Montgomery, R. T., Ogden, J. A. & Freels, J. K. (2017), 'Kraljicmatrix: An r package for implementing the Kraljic Matrix to strategically analyze a firm's purchasing portfolio.', *Journal of Open Source Software* **2**(10), 170.

Bosch, F., McRoberts, N., van den Berg, F. & Madden, L. (2008), 'The basic reproduction number of plant pathogens: Matrix approaches to complex dynamics', *Phytopathology* **98**, 239–49.

Brettin, A., Rossi-Goldthorpe, R., Weishaar, K. & Erovenko, I. V. (2018), 'Ebola could be eradicated through voluntary vaccination', *Royal Society Open Science* **5**(1), 171591.

Brito, D. L., Sheshinski, E. & Intriligator, M. D. (1991), 'Externalities and compulsary vaccinations', *Journal of Public Economics* **45**(1), 69–90.
URL: *https://www.sciencedirect.com/science/article/pii/0047272791900487*

Buchak, L. & Buchak, L. M. (2013), *Risk and rationality*, Oxford University Press.

Bucini, G., Merrill, S. C., Clark, E., Moegenburg, S. M., Zia, A., Koliba, C. J., Wiltshire, S., Trinity, L. & Smith, J. M. (2019), 'Risk attitudes affect livestock biosecurity deci-

sions with ramifications for disease control in a simulated production system', *Frontiers in Veterinary Science* **6**, 196.

Burdon, J. & Thrall, P. (1999), 'Spatial and temporal patterns in coevolving plant and pathogen associations', *The American Naturalist* **153**(S5), S15–S33.

Bussell, E. & Cunniffe, N. (2020), 'Applying optimal control theory to a spatial simulation model of sudden oak death: ongoing surveillance protects tanoak while conserving biodiversity', *Journal of the Royal Society Interface* **17**(165), 20190671.

Bussell, E. H., Dangerfield, C. E., Gilligan, C. A. & Cunniffe, N. J. (2019), 'Applying optimal control theory to complex epidemiological models to inform real-world disease management', *Philosophical Transactions of the Royal Society B: Biological Sciences* **374**(1776), 20180284.

Byrne, D. N. (1999), 'Migration and dispersal by the sweet potato whitefly, *Bemisia tabaci*', *Agricultural and Forest Meteorology* **97**(4), 309–316.
URL: *https://www.sciencedirect.com/science/article/pii/S016819239900074X*

Byrne, D., Rathman, R., Orum, T. & Palumbo, J. (1996), 'Localized migration and dispersal by the sweet potato whitefly, *Bemisia tabaci*', *Oecologia* **105**, 320–328.

Camerer, C. F. (2011), *Behavioral game theory: Experiments in strategic interaction*, Princeton University Press.

Carriere, Y., Brown, Z. S., Downes, S. J., Gujar, G., Epstein, G., Omoto, C., Storer, N. P., Mota-Sanchez, D., Søgaard Jørgensen, P. & Carroll, S. P. (2020), 'Governing evolution:

a socioecological comparison of resistance management for insecticidal transgenic Bt crops among four countries', *Ambio* **49**(1), 1–16.

Cascante-Vega, J., Torres-Florez, S., Cordovez, J. & Santos-Vega, M. (2022), 'How disease risk awareness modulates transmission: coupling infectious disease models with behavioural dynamics', *Royal Society Open Science* **9**(1), 210803.

Castle, M. D. & Gilligan, C. A. (2012), 'An epidemiological framework for modelling fungicide dynamics and control', *PLOS ONE* **7**(8), 1–10.
URL: *https://doi.org/10.1371/journal.pone.0040941*

Chang, S. L., Piraveenan, M., Pattison, P. & Prokopenko, M. (2020), 'Game theoretic modelling of infectious disease dynamics and intervention methods: a review', *Journal of Biological Dynamics* **14**(1), 57–89. PMID: 31996099.
URL: *https://doi.org/10.1080/17513758.2020.1720322*

Chen, F., Griffith, A., Cottrell, A. & Wong, Y.-L. (2013), 'Behavioral responses to epidemics in an online experiment: using virtual diseases to study human behavior', *PLOS ONE* **8**(1), e52814.

Chikoti, P. C., Mulenga, R. M., Tembo, M. & Sseruwagi, P. (2019), 'Cassava mosaic disease: a review of a threat to cassava production in zambia', *Journal of Plant Pathology* **101**(3), 467–477.

Chikoti, P., Melis, R. & Shanahan, P. (2016), 'Farmer's perception of cassava mosaic

disease, preferences and constraints in Lupaula province of Zambia.', *American Journal of Plant Sciences* pp. 1129–1138.

Chipeta, M. M., Shanahan, P., Melis, R., Sibiya, J. & Benesi, I. R. M. (2016), 'Farmers' knowledge of cassava brown streak disease and its management in Malawi', *International Journal of Pest Management* **62**(3), 175–184.
URL: *https://doi.org/10.1080/09670874.2016.1167268*

Cialdini, R., Reno, R. & Kallgren, C. (1990), 'A focus theory of normative conduct: Recycling the concept of norms to reduce littering in public places', *Journal of Personality and Social Psychology* **58**, 1015–1026.

Clin, P., Grognard, F., Andrivon, D., Mailleret, L. & Hamelin, F. M. (n.d.), 'Host mixtures for plant disease control: Benefits from pathogen selection and immune priming', *Evolutionary Applications* **n/a**(n/a).
URL: *https://onlinelibrary.wiley.com/doi/abs/10.1111/eva.13386*

Consortium, R. et al. (2013), 'Heterogeneity of selection and the evolution of resistance', *Trends in Ecology & Evolution* **28**(2), 110–118.

Corwin, J. A. & Kliebenstein, D. J. (2017), 'Quantitative resistance: more than just perception of a pathogen', *The Plant Cell* **29**(4), 655–665.

Council, N. R. et al. (2010), *Strategic planning for the Florida citrus industry: addressing citrus greening disease*, National Academies Press.

Cunniffe, N. J., Cobb, R. C., Meentemeyer, R. K., Rizzo, D. M. & Gilligan, C. A. (2016), 'Modeling when, where, and how to manage a forest epidemic, motivated by sudden oak death in California', *Proceedings of the National Academy of Sciences* **113**(20), 5640–5645.

URL: *https://www.pnas.org/content/113/20/5640*

Cunniffe, N. J. & Gilligan, C. A. (2020), Use of mathematical models to predict epidemics and to optimize disease detection and management, *in* J. B. Ristaino & A. Records, eds, 'Emerging Plant Diseases and Global Food Security', APS Press, chapter 12.

Cunniffe, N. J., Koskella, B., E. Metcalf, C. J., Parnell, S., Gottwald, T. R. & Gilligan, C. A. (2015a), 'Thirteen challenges in modelling plant diseases', *Epidemics* **10**, 6–10. Challenges in Modelling Infectious Disease Dynamics.

URL: *https://www.sciencedirect.com/science/article/pii/S1755436514000309*

Cunniffe, N. J., Laranjeira, F. F., Neri, F. M., DeSimone, R. E. & Gilligan, C. A. (2014), 'Cost-effective control of plant disease when epidemiological knowledge is incomplete: Modelling bahia bark scaling of citrus', *PLOS Computational Biology* **10**(8), 1–14.

URL: *https://doi.org/10.1371/journal.pcbi.1003753*

Cunniffe, N. J., Stutt, R. O. J. H., DeSimone, R. E., Gottwald, T. R. & Gilligan, C. A. (2015b), 'Optimising and communicating options for the control of invasive plant disease when there is epidemiological uncertainty', *PLOS Computational Biology* **11**(4), 1–24.

URL: *https://doi.org/10.1371/journal.pcbi.1004211*

Cunniffe, N. J., Taylor, N. P., Hamelin, F. M. & Jeger, M. J. (2022), 'Epidemiological and ecological consequences of virus manipulation of host and vector in plant virus transmission', *PLOS Computational Biology* **17**(12), 1–41.
URL: *https://doi.org/10.1371/journal.pcbi.1009759*

Czosnek, H. & Laterrot, H. (1997), 'A worldwide survey of tomato yellow leaf curl viruses', *Archives of Virology* **142**, 1391–406.

Davis, J. M. & Gardner, R. G. (1994), 'Harvest maturity affects fruit yield, size, and grade of fresh-market tomato cultivars', *HortSci* **29**(6), 613 – 615.
URL: *https://journals.ashs.org/hortsci/view/journals/hortsci/29/6/article-p613.xml*

Ddamulira, G., Isaac, O., Kiryowa, M., Akullo, R., Ajero, M., Logoose, M., Otim, A., Masika, F., Mundingotto, J., Matovu, M. et al. (2021), 'Practices and constraints of tomato production among smallholder farmers in Uganda', *African Journal of Food, Agriculture, Nutrition and Development* **21**(2), 17560–17580.

Del Valle, S., Mniszewski, S. & Hyman, J. (2013), 'Modeling the impact of behavior changes on the spread of pandemic influenza', pp. 59–77.

Delabouglise, A. & Boni, M. F. (2020), 'Game theory of vaccination and depopulation for managing livestock diseases and zoonoses on small-scale farms', *Epidemics* **30**, 100370.

Delaney, L., Kleczkowski, A., Maharaj, S., Rasmussen, S. & Williams, L. (2013), Reflections on a virtual experiment addressing human behaviour during epidemics., *in* 'SummerSim', p. 33.

Dhaliwal, M. S., Jindal, S. K., Sharma, A. & Prasanna, H. (2020), 'Tomato yellow leaf curl virus disease of tomato and its management through resistance breeding: A review', *The Journal of Horticultural Science and Biotechnology* **95**(4), 425–444.

Diekmann, O., Heesterbeek, J. A. P. & Roberts, M. G. (2010), 'The construction of next-generation matrices for compartmental epidemic models', *Journal of The Royal Society Interface* **7**(47), 873–885.

URL: *https://royalsocietypublishing.org/doi/abs/10.1098/rsif.2009.0386*

Djaha, K. E., Abo, K., Kone, T., Kone, D. & Kone, M. (2018), 'Analysis of the population structure of cassava growers, production systems, and plots' sanitary state in Côte d'Ivoire', *Journal of Animal & Plant Sciences* pp. 5833–5843.

Donmez, Z., Turhan, S., Karsu, O., Kara, B. Y. & Karaşan, O. (2022), 'Fair allocation of personal protective equipment to health centers during early phases of a pandemic', *Computers and Operations Research* **141**, 105690.

URL: *https://www.sciencedirect.com/science/article/pii/S0305054821003853*

Dorfman, R. (1979), 'A formula for the Gini coefficient', *The Review of Economics and Statistics* pp. 146–149.

Duell, D. (2020), 'Follow the majority? how voters coordinate electoral support to secure club goods', *Political Science Research and Methods* pp. 1–17.

Duflo, E., Kremer, M. & Robinson, J. (2011), 'Nudging farmers to use fertilizer: Theory and experimental evidence from Kenya', *American Economic Review* **101**(6), 2350–90.

d'Onofrio, A., Manfredi, P. & Poletti, P. (2011), 'The impact of vaccine side effects on the natural history of immunization programmes: an imitation-game approach', *Journal of Theoretical Biology* **273**(1), 63–71.

d'Onofrio, A., Manfredi, P. & Salinelli, E. (2007), 'Vaccinating behaviour, information, and the dynamics of SIR vaccine preventable diseases', *Theoretical Population Biology* **71**(3), 301–317.

Eames, K. T. (2009), 'Networks of influence and infection: parental choices and childhood disease', *Journal of the Royal Society Interface* **6**(38), 811–814.

Earn, D. J. D., Andrews, P. W. & Bolker, B. M. (2014), 'Population-level effects of suppressing fever', *Proceedings of the Royal Society B: Biological Sciences* **281**(1778), 20132570.
URL: *https://royalsocietypublishing.org/doi/abs/10.1098/rspb.2013.2570*

Eksin, C., Shamma, J. S. & Weitz, J. S. (2017), 'Disease dynamics in a stochastic network game: a little empathy goes a long way in averting outbreaks', *Scientific Reports* **7**(1), 1–13.

Elbers, A., De Koeijer, A., Scolamacchia, F. & Van Rijn, P. (2010), 'Questionnaire survey about the motives of commercial livestock farmers and hobby holders to vaccinate their animals against bluetongue virus serotype 8 in 2008–2009 in the Netherlands', *Vaccine* **28**(13), 2473–2481.

Elliott, L., Blanchard, J., Beaudoin, C., Green, C., Nowicki, D., Matusko, P. & Moses, S.

(2002), 'Geographical variations in the epidemiology of bacterial sexually transmitted infections in Manitoba, Canada', *Sexually Transmitted Infections* **78**(suppl 1), i139–i144.

Ephraim, N., Yona, B., Evans, A., Sharon, A. & Titus, A. (2015), 'Effect of cassava brown streak disease (CBSD) on cassava (Manihot esculenta Crantz) root storage components, starch quantities and starch quality properties', *International Journal of Plant Physiology and Biochemistry* .

Epstein, J. M., Parker, J., Cummings, D. & Hammond, R. A. (2008), 'Coupled contagion dynamics of fear and disease: Mathematical and computational explorations', *PLOS ONE* **3**(12), 1–11.
URL: *https://doi.org/10.1371/journal.pone.0003955*

Fabre, F., Coville, J. & Cunniffe, N. J. (2021), Optimising reactive disease management using spatially explicit models at the landscape scale, *in* P. Scott, R. Strange, L. Korsten & M. L. Gullino, eds, 'Plant Diseases and Food Security in the 21st Century:', Springer.

Fabre, F., Rousseau, E., Mailleret, L. & Moury, B. (2012), 'Durable strategies to deploy plant resistance in agricultural landscapes', *New Phytologist* **193**(4), 1064–1075.

Fang, X., You, M. P. & Barbetti, M. J. (2012), 'Reduced severity and impact of Fusarium wilt on strawberry by manipulation of soil pH, soil organic amendments and crop rotation', *European Journal of Plant Pathology* **134**(3), 619–629.

Faust, R. M., Koul, O., Cuperus, G. & Elliot, N. (2008), 'General introduction to areawide pest management', *Areawide Pest Management: Theory and implementation* pp. 1–14.

Fenichel, E. P., Kuminoff, N. V. & Chowell, G. (2013), 'Skip the trip: air travelers' behavioral responses to pandemic influenza', *PLOS ONE* **8**(3), e58249.

Fenichel, E. P. & Wang, X. (2013), *The Mechanism and Phenomena of Adaptive Human Behavior During an Epidemic and the Role of Information*, Springer New York, New York, NY, pp. 153–168.

Ferris, A. C., Stutt, R. O., Godding, D. & Gilligan, C. A. (2020), 'Computational models to improve surveillance for cassava brown streak disease and minimize yield loss', *PLOS Computational Biology* **16**(7), e1007823.

Fisman, D. N. & Laupland, K. B. (2009), 'The sounds of silence: Public goods, externalities, and the value of infectious disease control programs', *The Canadian Journal of Infectious Diseases &; Medical Microbiology* **20**(2), 39—41.
URL: *https://europepmc.org/articles/PMC2706405*

Florec, V., Sadler, R. J., White, B. & Dominiak, B. C. (2013), 'Choosing the battles: The economics of area wide pest management for Queensland fruit fly', *Food Policy* **38**, 203–213.
URL: *https://www.sciencedirect.com/science/article/pii/S0306919212001212*

Floyd, D. L., Prentice-Dunn, S. & Rogers, R. W. (2000), 'A meta-analysis of research on protection motivation theory', *Journal of Applied Social Psychology* **30**(2), 407–429.

Fonsah, E. G., Chen, Y., Diffie, S., Srinivansan, R. & Riley, D. (2018), 'Economic productivity and profitability analysis for whiteflies and tomato yellow leaf curl virus (TYLCV) management options', *Journal of Agriculture and Environmental Sciences* **6**.

Francis, P. J. (2004), 'Optimal tax/subsidy combinations for the flu season', *Journal of Economic Dynamics and Control* **28**(10), 2037–2054.
URL: *https://www.sciencedirect.com/science/article/pii/S0165188903001830*

French, E., Kim, B.-S. & Iyer-Pascuzzi, A. S. (2016), 'Mechanisms of quantitative disease resistance in plants', *Seminars in Cell & Developmental Biology* **56**, 201–208. X chromosome inactivation Plant immunity.
URL: *https://www.sciencedirect.com/science/article/pii/S1084952116301410*

Fu, F., Rosenbloom, D. I., Wang, L. & Nowak, M. A. (2011*a*), 'Imitation dynamics of vaccination behaviour on social networks', *Proceedings of the Royal Society B: Biological Sciences* **278**(1702), 42–49.

Fu, F., Rosenbloom, D. I., Wang, L. & Nowak, M. A. (2011*b*), 'Imitation dynamics of vaccination behaviour on social networks', *Proceedings of the Royal Society B: Biological Sciences* **278**(1702), 42–49.
URL: *https://royalsocietypublishing.org/doi/abs/10.1098/rspb.2010.1107*

Fukuda, E., Kokubo, S., Tanimoto, J., Wang, Z., Hagishima, A. & Ikegaya, N. (2014), 'Risk assessment for infectious disease and its impact on voluntary vaccination behavior in social networks', *Chaos, Solitons & Fractals* **68**, 1–9.
URL: *https://www.sciencedirect.com/science/article/pii/S0960077914001209*

Funk, S. & Jansen, V. (2013), *The Talk of the Town: Modelling the Spread of Information and Changes in Behaviour*, Springer, New York, pp. 93–102.

Funk, S., Salathé, M. & Jansen, V. A. (2010), 'Modelling the influence of human behaviour on the spread of infectious diseases: a review', *Journal of the Royal Society Interface* **7**(50), 1247–1256.

Gallois, J.-L., Moury, B. & German-Retana, S. (2018), 'Role of the genetic background in resistance to plant viruses', *International Journal of Molecular Sciences* **19**(10), 2856.

Galvani, A. P., Reluga, T. C. & Chapman, G. B. (2007), 'Long-standing influenza vaccination policy is in accord with individual self-interest but not with the utilitarian optimum', *Proceedings of the National Academy of Sciences* **104**(13), 5692–5697.

Gandon, S. (2004), 'Evolution of multihost parasites', *Evolution* **58**(3), 455–469.

Garba, S., Gumel, A. & Abu Bakar, M. (2008), 'Backward bifurcations in dengue transmission dynamics', *Mathematical Biosciences* **215**(1), 11–25.
URL: *https://www.sciencedirect.com/science/article/pii/S0025556408000734*

García-Arenal, F. & McDonald, B. A. (2003), 'An analysis of the durability of resistance to plant viruses', *Phytopathology* **93**(8), 941–952.

Garcia-Figuera, S., Deniston-Sheets, H., Grafton-Cardwell, E., Babcock, B., Lubell, M. & McRoberts, N. (2021), 'Perceived vulnerability and propensity to adopt best management practices for huanglongbing disease of citrus in California', *Phytopathology* **111**(10), 1758–1773. PMID: 33599529.
URL: *https://doi.org/10.1094/PHYTO-12-20-0544-R*

Gavish, N. & Katriel, G. (2022), 'The role of childrens vaccination for COVID-19 Pareto-optimal allocations of vaccines', *PLOS Computational Biology* **18**(2), 1–30.
URL: *https://doi.org/10.1371/journal.pcbi.1009872*

Geoffard, P.-Y. & Philipson, T. (1997), 'Disease eradication: Private versus public vaccination', *The American Economic Review* **87**(1), 222–230.
URL: *http://www.jstor.org/stable/2950864*

Gersovitz, M. (2014), 'Infectious disease externalities', *Encyclopedia of Health Economics* p. 35.

Gersovitz, M. & Hammer, J. S. (2005), 'Tax/subsidy policies toward vector-borne infectious diseases', *Journal of Public Economics* **89**(4), 647–674. Cornell - ISPE Conference on Public Finance and Development.
URL: *https://www.sciencedirect.com/science/article/pii/S0047272704000957*

Giannitsarou, C., Kissler, S. & Toxvaerd, F. (2021), 'Waning immunity and the second wave: Some projections for SARS-CoV-2', *American Economic Review: Insights* **3**(3), 321–38.

Gibson, R. W. & Otim-Nape, G. W. (1997), 'Factors determining recovery and reversion in mosaic-affected African cassava mosaic virus resistant cassava', *Annals of Applied Biology* **131**(2), 259–271.

URL: *https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1744-7348.1997.tb05155.x*

Gillespie, D. T. (1977), 'Exact stochastic simulation of coupled chemical reactions', *The Journal of Physical Chemistry* **81**(25), 2340–2361.

Gilligan, C. A., Truscott, J. E. & Stacey, A. J. (2007), 'Impact of scale on the effectiveness of disease control strategies for epidemics with cryptic infection in a dynamical landscape: an example for a crop disease', *Journal of the Royal Society Interface* **4**(16), 925–934.

Gilligan, C. A. & van den Bosch, F. (2008), 'Epidemiological models for invasion and persistence of pathogens', *Annual Reviews Phytopathology* **46**, 385–418.

Gini, C. (1936), 'On the measure of concentration with special reference to income and statistics', *Colorado College Publication, General Series* **208**(1), 73–79.

Gittings, K. & Matson, K. L. (2016), 'Establishing herd immunity against ebola through vaccination', *Vaccine* **34**(24), 2644–2647.

Graham, J., Gottwald, T. & Setamou, M. (2020), 'Status of huanglongbing (HLB) outbreaks in Florida, California and Texas', *Tropical Plant Pathology* **45**(3), 265–278.

Grimmett, G. & Welsh, D. (2014), *Probability: an introduction*, Oxford University Press.

Grogan, K. A. & Goodhue, R. E. (2012), 'Spatial externalities of pest control decisions in the california citrus industry', *Journal of Agricultural and Resource Economics* pp. 156–179.

Gumel, A. (2012), 'Causes of backward bifurcations in some epidemiological models', *Journal of Mathematical Analysis and Applications* **395**(1), 355–365.
URL: *https://www.sciencedirect.com/science/article/pii/S0022247X12003551*

Gumel, A. & Song, B. (2008), 'Existence of multiple-stable equilibria for a multi-drug-resistant model of mycobacterium tuberculosis', *Mathematical Biosciences and Engineering* **5**(3), 437–455. cited By 25.

Hadeler, K. & Castillo-Chavez, C. (1995), 'A core group model for disease transmission', *Mathematical Biosciences* **128**(1), 41–55.
URL: *https://www.sciencedirect.com/science/article/pii/0025556494000669*

Hamelin, F. M., Bowen, B., Bernhard, P. & Bokil, V. A. (2021), 'Optimal control of plant disease epidemics with clean seed usage.', *Bulletin of mathematical biology* **83 5**, 46.

Hamelin, F., Mammeri, Y., Aigu, Y., Strelkov, S. & Lewis, M. (2022), 'Host diversification may split epidemic spread into two successive fronts advancing at different speeds', *Bulletin of Mathematical Biology* **84**(7), 1–24.

Hasselman, B. & Hasselman, M. B. (2018), 'Package 'nleqslv'', *R package version* **3**(2).

Hennessey, M., Kim, S., Unger, F., Nguyen-Viet, H., Dang-Xuan, S., Nguyen-Thi, T. & Häsler, B. (2020), 'Exploring the potential of using nudges to promote food hygiene in the pork value chain in vietnam', *Preventive Veterinary Medicine* **181**, 105003.

Hennessy, D. A. (2008), 'Biosecurity incentives, network effects, and entry of a rapidly spreading pest', *Ecological Economics* **68**(1), 230–239.
URL: *https://www.sciencedirect.com/science/article/pii/S0921800908001134*

Hibbard, J. H. & Pope, C. R. (1983), 'Gender roles, illness orientation and use of medical services', *Social Science & Medicine* **17**(3), 129–137.

Hidano, A., Enticott, G., Christley, R. M. & Gates, M. C. (2018), 'Modeling dynamic human behavioral changes in animal disease models: Challenges and opportunities for addressing bias', *Frontiers in Veterinary Science* **5**.
URL: *https://www.frontiersin.org/article/10.3389/fvets.2018.00137*

Hilker, F. M., Allen, L. J., Bokil, V. A., Briggs, C. J., Feng, Z., Garrett, K. A., Gross, L. J., Hamelin, F. M., Jeger, M. J., Manore, C. A. et al. (2017), 'Modeling virus coinfection to inform management of maize lethal necrosis in Kenya', *Phytopathology* **107**(10), 1095–1108.

Hillis, V., Lubell, M., Kaplan, J., Doll, D. & Baumgartner, K. (2016), 'The role of pest control advisers in preventative management of grapevine trunk diseases', *Phytopathology* **106**(4), 339–347.

Hillocks, R. J., Raya, M. D., Mtunda, K. & Kiozia, H. (2001), 'Effects of brown streak

virus disease on yield and quality of cassava in Tanzania', *Journal of Phytopathology* **149**(7-8), 389–394.

URL: *https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1439-0434.2001.tb03868.x*

Hofbauer, J. & Sigmund, K. (2003), 'Evolutionary game dynamics', *Bulletin of the American Mathematical Society* **40**(4), 479–519.

Holden, S. T. & Fisher, M. (2015), 'Subsidies promote use of drought tolerant maize varieties despite variable yield performance under smallholder environments in Malawi', *Food Security* **7**(6), 1225–1238.

Holt, J., Colvin, J. & Muniyappa, V. (1999*a*), 'Identifying control strategies for tomato leaf curl virus disease using an epidemiological model', *Journal of Applied Ecology* **36**(5), 625–633.

URL: *https://besjournals.onlinelibrary.wiley.com/doi/abs/10.1046/j.1365-2664.1999.00432.x*

Holt, J., Colvin, J. & Muniyappa, V. (1999*b*), 'Identifying control strategies for tomato leaf curl virus disease using an epidemiological model', *Journal of Applied Ecology* **36**(5), 625–633.

Hoppensteadt, F. (1974), 'An age dependent epidemic model', *Journal of the Franklin Institute* **297**(5), 325–333.

Houngue, J., Pita, J., Cacaï, G., Zandjanakou-Tachin, M., Abidjo, E. A. E. & Ahanhanzo, C. (2018), 'Survey of farmers' knowledge of cassava mosaic disease and their

preferences for cassava cultivars in three agro-ecological zones in Benin.', *Ethnobiology Ethnomedicine* **14**(29).

URL: *https://doi.org/10.1186/s13002-018-0228-5*

Hozé, N., Bonhoeffer, S. & Regoes, R. (2018), 'Assessing the public health impact of tolerance-based therapies with mathematical models', *PLOS Computational Biology* **14**(5), 1–14.

URL: *https://doi.org/10.1371/journal.pcbi.1006119*

Huang, Y. & Liu, W. (2022), 'Promoting COVID-19 vaccination: The interplay of message framing, psychological uncertainty, and public agency as a message source', *Science Communication* **44**(1), 3–29.

Hummert, S., Bohl, K., Basanta, D., Deutsch, A., Werner, S., Theißen, G., Schroeter, A. & Schuster, S. (2014), 'Evolutionary game theory: cells as players', *Molecular BioSystems* **10**(12), 3044–3065.

Hutchison, W. D., Burkness, E. C., Mitchell, P. D., Moon, R. D., Leslie, T. W., Fleischer, S. J., Abrahamson, M., Hamilton, K. L., Steffey, K. L., Gray, M. E., Hellmich, R. L., Kaster, L. V., Hunt, T. E., Wright, R. J., Pecinovsky, K., Rabaey, T. L., Flood, B. R. & Raun, E. S. (2010), 'Areawide suppression of European Corn Borer with Bt maize reaps savings to non-Bt maize growers', *Science* **330**(6001), 222–225.

URL: *https://www.science.org/doi/abs/10.1126/science.1190242*

Hyatt-Twynam, S. R., Parnell, S., Stutt, R. O., Gottwald, T. R., Gilligan, C. A. & Cun-

niffe, N. J. (2017), 'Risk-based management of invading plant disease', *New Phytologist* **214**(3), 1317–1329.

Ibuka, Y. & ichiro Bessho, S. (2015), 'Subsidies for influenza vaccination, vaccination rates, and health outcomes among the elderly in japan', *Japan and the World Economy* **36**, 56–66.

URL: *https://www.sciencedirect.com/science/article/pii/S092214251500033X*

Ibuka, Y., Li, M., Vietri, J., Chapman, G. B. & Galvani, A. P. (2014), 'Free-riding behavior in vaccination decisions: An experimental study', *PLOS ONE* **9**(1), 1–9.

URL: *https://doi.org/10.1371/journal.pone.0087164*

Ioannu, N. (1987), 'Cultural management of tomato yellow leaf curl disease in Cyprus', *Plant Pathology* **36**(3), 367–373.

URL: *https://bsppjournals.onlinelibrary.wiley.com/doi/abs/10.1111/j.1365-3059.1987.tb02245.x*

Janz, N. K. & Becker, M. H. (1984), 'The health belief model: A decade later', *Health Education Quarterly* **11**(1), 1–47.

Jeger, M., Holt, J., van den Bosch, F. & Madden, L. (2004), 'Epidemiology of insect-transmitted plant viruses: modelling disease dynamics and control interventions', *Physiological Entomology* **29**, 291–304.

Jijón, S., Supervie, V. & Breban, R. (2017), 'Prevention of treatable infectious diseases: A game-theoretic approach', *Vaccine* **35**(40), 5339–5345.

Johannesen, A. B. & Skonhoft, A. (2005), 'Tourism, poaching and wildlife conservation: what can integrated conservation and development projects accomplish?', *Resource and Energy Economics* **27**(3), 208–226.
URL: *https://www.sciencedirect.com/science/article/pii/S0928765504000776*

Jones, B. D. (1999), 'Bounded rationality', *Annual Review of Political Science* **2**(1), 297–321.

Jones, M. M., Stansly, P. A. & Russo, J. (2013), Extension model to improve Asian citrus psyllid control in citrus health management areas (CHMAs), *in* 'Proceedings of the Florida State Horticultural Society', Vol. 126, pp. 68–71.

Jowett, K., Milne, A. E., Potts, S. G., Senapathi, D. & Storkey, J. (2022), 'Communicating carabids: Engaging farmers to encourage uptake of integrated pest management.', *Pest Management Science* **n/a**(n/a).
URL: *https://onlinelibrary.wiley.com/doi/abs/10.1002/ps.6878*

Karafillakis, E., Simas, C., Jarrett, C., Verger, P., Peretti-Watel, P., Dib, F., De Angelis, S., Takacs, J., Ali, K. A., Celentano, L. P. et al. (2019), 'HPV vaccination in a context of public mistrust and uncertainty: a systematic literature review of determinants of HPV vaccine hesitancy in Europe', *Human Vaccines & Immunotherapeutics* .

Karlsson, C.-J. & Rowlett, J. (2020), 'Decisions and disease: a mechanism for the evolution of cooperation', *Scientific Reports* **10**(1), 1–9.

Karungi, J., Adipala, E., Ogenga-Latigo, M., Kyamanywa, S. & Oyobo, N. (2000), 'Pest

management in cowpea. part 1. influence of planting time and plant density on cowpea field pests infestation in eastern Uganda', *Crop Protection* **19**(4), 231–236.

Kasthurirathna, D. & Piraveenan, M. (2015), 'Emergence of scale-free characteristics in socio-ecological systems with bounded rationality', *Scientific Reports* **5**(1), 1–16.

Katan, J. (2010), 'Cultural approaches for disease management: present status and future prospects', *Journal of Plant Pathology* pp. S7–S9.

Kaup, B. Z. (2008), 'The reflexive producer: The influence of farmer knowledge upon the use of Bt corn', *Rural Sociology* **73**(1), 62–81.
URL: *https://onlinelibrary.wiley.com/doi/abs/10.1526/003601108783575871*

Kause, A. & Ødegård, J. (2012), 'The genetic analysis of tolerance to infections: a review', *Frontiers in Genetics* **3**, 262.

Kearney, P. M., Spillane, M., Humphries, R., Gannon, A., Stamenic, D., Bhuachalla, C. N., Hoevel, P., Arensman, E., O'Riordain, M., Troya, M. I. et al. (2022), 'Compliance with local travel restrictions and face masks during first phase of COVID-19 pandemic in Ireland: a national survey', *Journal of public health (Oxford, England)* .

Keeling, M. J. & Rohani, P. (2008), *Modeling Infectious Diseases in Humans and Animals*, Princeton University Press.
URL: *http://www.jstor.org/stable/j.ctvcm4gk0*

Kennan, J. (1979), 'The estimation of partial adjustment models with rational expectations', *Econometrica: Journal of the Econometric Society* pp. 1441–1455.

Kil, E.-J., Kim, S., Lee, Y.-J., Byun, H.-S., Park, J., Seo, H., Kim, C.-S., Shim, J.-K., Lee, J.-H., Kim, J.-K., Lee, K.-Y., Choi, H.-S. & Lee, S. (2016), 'Tomato yellow leaf curl virus (TYLCV-IL): A seed-transmissible geminivirus in tomatoes', *Scientific Reports* **6**, 19013.

Kiss, I., Cassell, J., Recker, M. & Simon, P. (2009), 'The impact of information transmission on epidemic outbreaks', *Mathematical Biosciences* **225**, 1–10.

Kogan, M. (1986), Plant defense strategies and host-plant resistance., *in* 'Ecological Theory and Integrated Pest Management Practice', John Wiley and Sons, New York.

Krupinsky, J. M., Bailey, K. L., McMullen, M. P., Gossen, B. D. & Turkington, T. K. (2002), 'Managing plant disease risk in diversified cropping systems', *Agronomy Journal* **94**(2), 198–209.

Lapidot, M., Friedmann, M., Lachman, O., Yehezkel, A., Nahon, S., Cohen, S. & Pilowsky, M. (1997a), 'Comparison of resistance level to tomato yellow leaf curl virus among commercial cultivars and breeding lines', *Plant Disease* **81**(12), 1425–1428.

Lapidot, M., Friedmann, M., Pilowsky, M., Ben-Joseph, R. & Cohen, S. (2002), 'Effect of host plant resistance to tomato yellow leaf curl virus (TYLCV) on virus acquisition and transmission by its whitefly vector', *Phytopathology* **91**, 1209–13.

Lapidot, M., Paran, I., Ben-Joseph, R., Ben-Harush, S., Pilowsky, M., Cohen, S. & Shifriss, C. (1997b), 'Tolerance to cucumber mosaic virus in pepper: development of advanced breeding lines and evaluation of virus level', *Plant Disease* **81**(2), 185–188.

Laranjeira, F. F., Silva, S. X. B., Murray-Watson, R. E., Soares, A. C. F., Santos-Filho, H. P. & Cunniffe, N. J. (2020), 'Spatiotemporal dynamics and modelling support the case for area-wide management of citrus greasy spot in a Brazilian smallholder farming region', *Plant Pathology* **69**(3), 467–483.

URL: *https://bsppjournals.onlinelibrary.wiley.com/doi/abs/10.1111/ppa.13146*

Lazarus, W. F. & Dixon, B. L. (1984), 'Agricultural pests as common property: Control of the corn rootworm', *American Journal of Agricultural Economics* **66**(4), 456–465.

Lazić, A., Kalinova, K. N., Packer, J., Pae, R., Petrović, M. B., Popović, D., Sievert, D. E. C. & Stafford-Johnson, N. (2021), 'Social nudges for vaccination: How communicating herd behaviour influences vaccination intentions', *British Journal of Health Psychology* **26**(4), 1219–1237.

URL: *https://bpspsychub.onlinelibrary.wiley.com/doi/abs/10.1111/bjhp.12556*

Lee, J.-Y. & Choi, S. (2021), 'Supply chain investment and contracting for carbon emissions reduction: A social planner's perspective', *International Journal of Production Economics* **231**, 107873.

URL: *https://www.sciencedirect.com/science/article/pii/S0925527320302322*

Legarrea, S., Barman, A., Marchant, W., Diffie, S. & Srinivasan, R. (2015), 'Temporal effects of a begomovirus infection and host plant resistance on the preference and development of an insect vector, *Bemisia tabaci*, and implications for epidemics', *PLOS ONE* **10**(11), 1–19.

URL: *https://doi.org/10.1371/journal.pone.0142114*

Legg, J., Ndalahwa, M., Yabeja, J., Ndyetabula, I., Bouwmeester, H., Shirima, R. & Mtunda, K. (2017), 'Community phytosanitation to manage cassava brown streak disease', *Virus Research* **241**, 236–253. Plant Virus Epidemiology.

URL: *https://www.sciencedirect.com/science/article/pii/S0168170217301533*

Legg, J., Somado, E., Barker, I., Beach, L., Ceballos, H., Cuellar, W., Elkhoury, W., Gerling, D., Helsen, J., Hershey, C., Jarvis, A., Kulakow, P., Kumar, L., Lorenzen, J., Lynam, J., McMahon, M., Maruthi, G., Miano, D., Mtunda, K., Natwuruhunga, P., Okogbenin, E., Pezo, P., Terry, E., Thiele, G., Thresh, M., Wadsworth, J., Walsh, S., Winter, S., Tohme, J. & Fauquet, C. (2014), 'A global alliance declaring war on cassava viruses in Africa.', *Food Security* **6**, 231–248.

URL: *https://doi.org/10.1007/s12571-014-0340-x*

Leonard, K. J. (1977), 'Selection pressures and plant pathogens', *Annals of the New York Academy of Sciences* **287**(1), 207–222.

URL: *https://nyaspubs.onlinelibrary.wiley.com/doi/abs/10.1111/j.1749-6632.1977.tb34240.x*

Levy, D. & Lapidot, M. (2008), 'Effect of plant age at inoculation on expression of genetic resistance to tomato yellow leaf curl virus', *Archives of Virology* **153**, 171–9.

Li, T., Long, H., Zhang, Y., Tu, S., Ge, D., Li, Y. & Hu, B. (2017), 'Analysis of the spatial mismatch of grain production and farmland resources in China based on the potential crop rotation system', *Land Use Policy* **60**, 26–36.

URL: *https://www.sciencedirect.com/science/article/pii/S0264837716300515*

Li, Y., Sun, L., Zhou, W. & Su, Q. (2020), 'Regional differences in and influencing factors of animal epidemic risk in China', *Frontiers in Veterinary Science* **7**, 520.

Lim, W. & Zhang, P. (2020), 'Herd immunity and a vaccination game: An experimental study', *PLOS ONE* **15**(5), e0232652.

Liu, J., Kochin, B. F., Tekle, Y. I. & Galvani, A. P. (2012), 'Epidemiological game-theory dynamics of chickenpox vaccination in the USA and Israel', *Journal of the Royal Society Interface* **9**(66), 68–76.

Lo Iacono, G., van den Bosch, F. & Gilligan, C. A. (2013), 'Durable resistance to crop pathogens: An epidemiological framework to predict risk under uncertainty', *PLOS Computational Biology* **9**(1), 1–9.
URL: *https://doi.org/10.1371/journal.pcbi.1002870*

Loewenstein, G. & Thaler, R. H. (1989), 'Anomalies: intertemporal choice', *Journal of Economic perspectives* **3**(4), 181–193.

Luc, D. T. (2008), *Pareto Optimality*, Springer New York, New York, NY, pp. 481–515.

Madani, K. (2010), 'Game theory and water resources', *Journal of Hydrology* **381**(3), 225–238.
URL: *https://www.sciencedirect.com/science/article/pii/S0022169409007653*

Madden, L., Hughes, G. & van den Bosch, F. (2007), *The study of plant disease epidemics*, American Phytopathological Society (APS Press), St Paul, MN.

Maharaj, S., McCaldin, T. & Kleczkowski, A. (2011), A participatory simulation model for studying attitudes to infection risk, *in* 'Proceedings of the 2011 Summer Computer Simulation Conference', pp. 8–13.

Mankad, A. (2016), 'Psychological influences on biosecurity control and farmer decision-making. a review', *Agronomy for Sustainable Development* **36**(2), 1–14.

Mankad, A., Zhang, A. & Curnock, M. (2019), 'Motivational drivers of action in response to an environmental biosecurity incursion', *Journal of Environmental Management* **232**, 851–857.

Marchant, W. G., Gautam, S., Hutton, S. F. & Srinivasan, R. (2020), 'Tomato yellow leaf curl virus-resistant and-susceptible tomato genotypes similarly impact the virus population genetics', *Frontiers in Plant Science* **11**.

Maruthi, M. N., Hillocks, R. J., Mtunda, K., Raya, M. D., Muhanna, M., Kiozia, H., Rekha, A. R., Colvin, J. & Thresh, J. M. (2005), 'Transmission of cassava brown streak virus by *Bemisia tabaci* (Gennadius)', *Journal of Phytopathology* **153**(5), 307–312. **URL:** *https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1439-0434.2005.00974.x*

Mastin, A. J., Gottwald, T. R., van den Bosch, F., Cunniffe, N. J. & Parnell, S. (2020), 'Optimising risk-based surveillance for early detection of invasive plant pathogens', *PLOS Biology* **18**(10), e3000863.

May, R. M. & Anderson, R. M. (1984), 'Spatial heterogeneity and the design of immu-

nization programs', *Mathematical Biosciences* **72**(1), 83–111.

URL: *https://www.sciencedirect.com/science/article/pii/0025556484900634*

McDonald, B. A. & Stukenbrock, E. H. (2016), 'Rapid emergence of pathogens in agro-ecosystems: global threats to agricultural sustainability and food security', *Philosophical Transactions of the Royal Society B: Biological Sciences* **371**(1709), 20160026.

McQuaid, C. F., Gilligan, C. A. & van den Bosch, F. (2017a), 'Considering behaviour to ensure the success of a disease control strategy', *Royal Society Open Science* **4**(12), 170721.

URL: *https://royalsocietypublishing.org/doi/abs/10.1098/rsos.170721*

McQuaid, C. F., van den Bosch, F., Szyniszewska, A., Alicai, T., Pariyo, A., Chikoti, P. C. & Gilligan, C. A. (2017b), 'Spatial dynamics and control of a crop pathogen with mixed-mode transmission', *PLOS Computational Biology* **13**(7), 1–18.

URL: *https://doi.org/10.1371/journal.pcbi.1005654*

Meentemeyer, R. K., Cunniffe, N. J., Cook, A. R., Filipe, J. A., Hunter, R. D., Rizzo, D. M. & Gilligan, C. A. (2011), 'Epidemiological modeling of invasion in heterogeneous landscapes: spread of sudden oak death in California (1990–2030)', *Ecosphere* **2**(2), 1–24.

Meissle, M., Romeis, J. & Bigler, F. (2011), 'Bt maize and integrated pest management-a European perspective', *Pest Management Science* **67**(9), 1049–1058.

Merrill, S. C., Koliba, C. J., Moegenburg, S. M., Zia, A., Parker, J., Sellnow, T., Wilt-

shire, S., Bucini, G., Danehy, C. & Smith, J. M. (2019a), 'Decision-making in livestock biosecurity practices amidst environmental and social uncertainty: evidence from an experimental game', *PLOS ONE* **14**(4), e0214500.

Merrill, S. C., Moegenburg, S., Koliba, C. J., Zia, A., Trinity, L., Clark, E., Bucini, G., Wiltshire, S., Sellnow, T., Sellnow, D. & Smith, J. M. (2019b), 'Willingness to comply with biosecurity in livestock facilities: Evidence from experimental simulations', *Frontiers in Veterinary Science* **6**, 156.

Merrill, S. C., Trinity, L., Clark, E. M., Shrum, T. R., Koliba, C. J., Zia, A., Bucini, G., Sellnow, T. L., Sellnow, D. D. & Smith, J. M. (2021), 'Message delivery strategy influences willingness to comply with biosecurity', *Frontiers in Veterinary Science* p. 688.

Meshram, S. G., Adhami, M., Kisi, O., Meshram, C., Duc, P. A. & Khedher, K. M. (2021), 'Identification of critical watershed for soil conservation using game theory-based approaches', *Water Resources Management* **35**(10), 3105–3120.

Mesterton-Gibbons, M., Gavrilets, S., Gravner, J. & Akcay, E. (2011), 'Models of coalition or alliance formation', *Journal of Theoretical Biology* **274**(1), 187–204.

Mikaberidze, A., Mundt, C. C. & Bonhoeffer, S. (2016), 'Invasiveness of plant pathogens depends on the spatial scale of host distribution', *Ecological Applications* **26**(4), 1238–1248.

Milne, A. E., Bell, J. R., Hutchison, W. D., van den Bosch, F., Mitchell, P. D., Crowder,

D., Parnell, S. & Whitmore, A. P. (2016), 'The effect of farmers' decisions on pest control with Bt crops: A billion dollar game of strategy', *PLOS Computational Biology* **11**(12), 1–18.

Milne, A. E., Gottwald, T., Parnell, S. R., Alonso Chavez, V. & van den Bosch, F. (2020), 'What makes or breaks a campaign to stop an invading plant pathogen?', *PLOS Computational Biology* **16**(2), 1–20.
URL: *https://doi.org/10.1371/journal.pcbi.1007570*

Milne, A. E., Teiken, C., Deledalle, F., van den Bosch, F., Gottwald, T. & McRoberts, N. (2018), 'Growers' risk perception and trust in control options for huanglongbing citrus-disease in Florida and California', *Crop Protection* **114**, 177–186.
URL: *https://www.sciencedirect.com/science/article/pii/S0261219418302540*

Mlambo, L. (2012), 'Adaptive and rational expectations hypotheses: Reviewing the critiques', *Journal of Economic Behaviour* .

Mohammed, I. U., Ghosh, S. & Maruthi, M. N. (2016), 'Host and virus effects on reversion in cassava affected by cassava brown streak disease', *Plant Pathology* **65**(4), 593–600.
URL: *https://bsppjournals.onlinelibrary.wiley.com/doi/abs/10.1111/ppa.12458*

Mohr, S., Beard, R., Nisbet, A., Burgess, S., Reeve, R., Denwood, M., Porphyre, T., Zadoks, R. & Matthews, L. (2020), 'Uptake of diagnostic tests by livestock farmers: a stochastic game theory approach.'.
URL: *https://www.frontiersin.org/articles/10.3389/fvets.2020.00036/full*

Monger, W. A., Seal, S., Isaac, A. M. & Foster, G. D. (2001), 'Molecular characterization of the cassava brown streak virus coat protein', *Plant Pathology* **50**(4), 527–534.

Moran, P. A. (1950), 'Notes on continuous stochastic phenomena', *Biometrika* **37**(1/2), 17–23.

Morris, P. (2012), *Introduction to Game Theory*, Universitext, Springer.

Moussaïd, M. (2013), 'Opinion formation and the collective dynamics of risk perception', *PLOS ONE* **8**(12), e84592.

Moussaïd, M., Kämmer, J. E., Analytis, P. P. & Neth, H. (2013), 'Social influence and the collective dynamics of opinion formation', *PLOS ONE* **8**(11), e78433.

Mukarumbwa, P., Mushunje, A., Taruvinga, A., Akinyemi, B. & Ngarava, S. (2017), 'Factors influencing number of post-harvest practices adopted by smallholder vegetable farmers in mashonaland east province of zimbabwe', *International Journal of Development and Sustainability* **6**(11), 1774–1790.

Mundt, C. C. (2018), 'Pyramiding for resistance durability: theory and practice', *Phytopathology* **108**(7), 792–802.

Munro, G. R. (2009), 'Game theory and the development of resource management policy: the case of international fisheries', *Environment and Development Economics* **14**(1), 7–27.

Murray, A. G. (2014), 'A game theory based framework for assessing incentives for local

area collaboration with an application to Scottish salmon farming', *Preventive Veterinary Medicine* **115**(3), 255–262.

URL: *https://www.sciencedirect.com/science/article/pii/S0167587714001184*

Nash Jr, J. F. (1950), 'Equilibrium points in n-person games', *Proceedings of the National Academy of Sciences* **36**(1), 48–49.

Ndeffo Mbah, M. L. & Gilligan, C. A. (2010), 'Balancing detection and eradication for control of epidemics: sudden oak death in mixed-species stands', *PLOS ONE* **5**(9), e12317.

Nerlove, M. (1958), The dynamics of supply: estimation of farmers' response to price, Technical report, Johns Hopkins Press Baltimore.

Newton, A. C. (2016), 'Exploitation of diversity within crops: the key to disease tolerance?', *Frontiers in Plant Science* **7**.

URL: *https://www.frontiersin.org/article/10.3389/fpls.2016.00665*

Nixon, E. J., Vineer, H. R. & Wall, R. (2017), 'Treatment strategies for sheep scab: an economic model of farmer behaviour', *Preventive Veterinary Medicine* **137**, 43–51.

Njau, P. N., Jin, Y., Huerta-Espino, J., Keller, B. & Singh, R. P. (2010), 'Identification and evaluation of sources of resistance to stem rust race Ug99 in wheat', *Plant Disease* **94**(4), 413–419.

Nowak, M. A., Bonhoeffer, S. & May, R. M. (1994), 'Spatial games and the maintenance

of cooperation.', *Proceedings of the National Academy of Sciences* **91**(11), 4877–4881.
URL: *https://www.pnas.org/doi/abs/10.1073/pnas.91.11.4877*

Ntawuruhunga, P., Legg, J., Okidi, J., Okao-Okuja, G., Tadu, G. & Remington, T. (2007), 'Southern Sudan, Equatoria Region, cassava baseline survey technical report', *IITA* .

Ohtsuki, A. & Sasaki, A. (2006), 'Epidemiology and disease-control under gene-for-gene plant–pathogen interaction', *Journal of Theoretical Biology* **238**(4), 780–794.
URL: *https://www.sciencedirect.com/science/article/pii/S0022519305002882*

Ojedele, R. (2016), 'When the drum beats: Radio drama as a vehicle for behavior change in climate change adaptation amongst rural small holder farmers'.

Okechukwu, R. & Kumar, P. L. (2016), 'Releasing disease-resistant varieties of cassava in Africa', *Case Studies of Roots, Tubers and Bananas Seed Systems* pp. 2016–3.
URL: *https://hdl.handle.net/10568/79752*

Oraby, T. & Bauch, C. T. (2015), 'Bounded rationality alters the dynamics of paediatric immunization acceptance', *Scientific Reports* **5**(1), 1–12.

Oraby, T., Thampi, V. & Bauch, C. T. (2014), 'The influence of social norms on the dynamics of vaccinating behaviour for paediatric infectious diseases', *Proceedings of the Royal Society B: Biological Sciences* **281**(1780), 20133172.

Pacheco, J. M., Santos, F. C. & Dingli, D. (2014), 'The ecology of cancer from an evolutionary game theory perspective', *Interface Focus* **4**(4), 20140019.

Pagán, I. & García-Arenal, F. (2020), 'Tolerance of plants to pathogens: a unifying view', *Annual Reviews Phytopathology* **58**, 77–96.

Pagán, I. & García-Arenal, F. (2018), 'Tolerance to plant pathogens: Theory and experimental evidence', *International Journal of Molecular Sciences* **19**.

Pan, H., Chu, D., Yan, W., Su, Q., Liu, B., Wang, S., Wu, Q., Xie, W., Jiao, X., Li, R., Yang, N., Yang, X., Xu, B., Brown, J. K., Zhou, X. & Zhang, Y. (2012), 'Rapid spread of tomato yellow leaf curl virus in China is aided differentially by two invasive whiteflies', *PLOS ONE* **7**(4), 1–9.
URL: *https://doi.org/10.1371/journal.pone.0034817*

Papayiannis, L., Katis, N., Idris, A. & Brown, J. (2011), 'Identification of weed hosts of tomato yellow leaf curl virus in cyprus', *Plant Disease* **95**(2), 120–125.

Paradis, E., Blomberg, S., Bolker, B., Brown, J., Claude, J., Cuong, H. S., Desper, R. & Didier, G. (2019), 'Package 'ape'', *Analyses of phylogenetics and evolution, version* **2**(4), 47.

Parlevliet, J. E. (2002), 'Durability of resistance against fungal, bacterial and viral pathogens; present situation', *Euphytica* **124**(2), 147–156.

Patil, B., Ogwok, E., Wagaba, H., Mohammed, I. U., Yadav, J. S., Bagewadi, B., Taylor, N. J., Kreuze, J. F., Maruthi, M. N., Alicai, T. & Fauquet, C. M. (2011), 'Rnai-mediated resistance to diverse isolates belonging to two virus species involved in cassava brown streak disease', *Molecular Plant Pathology* **12**(1), 31–41.

Pautasso, M. & Jeger, M. J. (2014), 'Network epidemiology and plant trade networks', *AoB Plants* **6**, plu007.

URL: *https://doi.org/10.1093/aobpla/plu007*

Perra, N. & Vespignani, A. (2013), 'Modeling contact and mobility based social response to the spreading of infectious diseases', pp. 103–123.

Pinkhasov, R. M., Wong, J., Kashanian, J., Lee, M., Samadi, D. B., Pinkhasov, M. M. & Shabsigh, R. (2010), 'Are men shortchanged on health? perspective on health care utilization and health risk behavior in men and women in the United States', *International journal of clinical practice* **64**(4), 475–487.

Plans-Rubió, P. (2012), 'Evaluation of the establishment of herd immunity in the population by means of serological surveys and vaccination coverage', *Human Vaccines & Immunotherapeutics* **8**(2), 184–188.

Poletti, P., Ajelli, M. & Merler, S. (2012), 'Risk perception and effectiveness of uncoordinated behavioral responses in an emerging epidemic', *Mathematical Biosciences* **238**(2), 80–89.

URL: *https://www.sciencedirect.com/science/article/pii/S0025556412000892*

Poletti, P., Caprile, B., Ajelli, M., Pugliese, A. & Merler, S. (2009), 'Spontaneous behavioural changes in response to epidemics', *Journal of Theoretical Biology* **260**(1), 31–40.

Polston, J. E. & Lapidot, M. (2007), Management of tomato yellow leaf curl virus: US

and Israel perspectives, *in* 'Tomato yellow leaf curl virus disease', Springer, pp. 251–262.

Polston, J., McGovern, R. & Brown, L. (1999), 'Introduction of tomato yellow leaf curl virus in Florida and implications for the spread of this and other geminiviruses of tomato', *Plant Disease* **83**(11), 984–988.

Pretty, J., Brett, C., Gee, D., Hine, R., Mason, C., Morison, J., Rayment, M., Bijl, G. V. D. & Dobbs, T. (2001), 'Policy challenges and priorities for internalizing the externalities of modern agriculture', *Journal of Environmental Planning and Management* **44**(2), 263–283.

URL: *https://www.tandfonline.com/doi/abs/10.1080/09640560123782*

Pérez-Padilla, V., Fortes, I. M., Romero-Rodríguez, B., Arroyo-Mateos, M., Castillo, A. G., Moyano, C., De León, L. & Moriones, E. (2020), 'Revisiting seed transmission of the type strain of tomato yellow leaf curl virus in tomato plants', *Phytopathology* **110**(1), 121–129. PMID: 31584339.

URL: *https://doi.org/10.1094/PHYTO-07-19-0232-FI*

Råberg, L., Graham, A. L. & Read, A. F. (2009), 'Decomposing health: tolerance and resistance to parasites in animals', *Philosophical Transactions of the Royal Society B: Biological Sciences* **364**(1513), 37–49.

Rahman, M. M., Davies, P., Bansal, U., Pasam, R., Hayden, M. & Trethowan, R. (2021), 'Relationship between resistance and tolerance of crown rot in bread wheat', *Field*

Crops Research **265**, 108106.

URL: *https://www.sciencedirect.com/science/article/pii/S0378429021000526*

Ramos, R. S., Kumar, L., Shabani, F. & Picanço, M. C. (2019), 'Risk of spread of tomato yellow leaf curl virus (TYLCV) in tomato crops under various climate change scenarios', *Agricultural Systems* **173**, 524–535.

URL: *https://www.sciencedirect.com/science/article/pii/S0308521X18305870*

Reluga, T. C. (2010), 'Game theory of social distancing in response to an epidemic', *PLOS Computational Biology* **6**(5), e1000793.

Reluga, T. C., Bauch, C. T. & Galvani, A. P. (2006), 'Evolving public perceptions and stability in vaccine uptake', *Mathematical Biosciences* **204**(2), 185–198.

Reñosa, M. D. C., Landicho, J., Wachinger, J., Dalglish, S. L., Bärnighausen, K., Bärnighausen, T. & McMahon, S. A. (2021), 'Nudging toward vaccination: a systematic review', *BMJ Global Health* **6**(9), e006237.

Rezaei, R., Safa, L., Damalas, C. A. & Ganjkhanloo, M. M. (2019), 'Drivers of farmers' intention to use integrated pest management: integrating theory of planned behavior and norm activation model', *Journal of Environmental Management* **236**, 328–339.

Richardson, E. T., Barrie, M. B., Nutt, C. T., Kelly, J. D., Frankfurter, R., Fallah, M. P. & Farmer, P. E. (2017), 'The ebola suspect's dilemma', *The Lancet Global Health* **5**(3), e254–e256.

Riechert, S. E. & Hammerstein, P. (1983), 'Game theory in the ecological context', *Annual Review of Ecology and Systematics* **14**(1), 377–409.
URL: *https://doi.org/10.1146/annurev.es.14.110183.002113*

Riley, D. & Srinivasan, R. (2019), 'Integrated management of tomato yellow leaf curl virus and its whitefly vector in tomato', *Journal of Economic Entomology* **112**, 1526–1540.

Rimbaud, L., Fabre, F., Papaïx, J., Moury, B., Lannou, C., Barrett, L. G. & Thrall, P. H. (2021), 'Models of plant resistance deployment', *Annual Review of Phytopathology* **59**, 125–152.

Rimbaud, L., Papaïx, J., Rey, J.-F., Barrett, L. G. & Thrall, P. H. (2018), 'Assessing the durability and efficiency of landscape-based strategies to deploy plant resistance to pathogens', *PLOS Computational Biology* **14**(4), e1006067.

Roberts, T. W., Peck, D. E. & Ritten, J. P. (2012), 'Cattle producers' economic incentives for preventing bovine brucellosis under uncertainty', *Preventive Veterinary Medicine* **107**(3), 187–203.
URL: *https://www.sciencedirect.com/science/article/pii/S0167587712002085*

Roca, C. P., Cuesta, J. A. & Sánchez, A. (2009), 'Evolutionary game theory: Temporal and spatial effects beyond replicator dynamics', *Physics of Life Reviews* **6**(4), 208–249.
URL: *https://www.sciencedirect.com/science/article/pii/S1571064509000256*

Rocco, C. D. & Morabito, R. (2016), 'Production and logistics planning in the tomato processing industry: A conceptual scheme and mathematical model', *Computers and*

Electronics in Agriculture **127**, 763–774.

URL: *https://www.sciencedirect.com/science/article/pii/S0168169916305865*

Rodriguez, J. M., Molnar, J. J., Fazio, R. A., Sydnor, E. & Lowe, M. J. (2009), 'Barriers to adoption of sustainable agriculture practices: Change agent perspectives', *Renewable Agriculture and Food Systems* **24**(1), 60–71.

Rojas, M. R., Macedo, M. A., Maliano, M. R., Soto-Aguilar, M., Souza, J. O., Briddon, R. W., Kenyon, L., Rivera Bustamante, R. F., Zerbini, F. M., Adkins, S. et al. (2018), 'World management of geminiviruses', *Annual Review of Phytopathology* **56**, 637–677.

Rubin, G. J., Amlôt, R., Page, L. & Wessely, S. (2009), 'Public perceptions, anxiety, and behaviour change in relation to the swine flu outbreak: cross sectional telephone survey', *BMJ* **339**.

Sadique, M. Z., Edmunds, W. J., Smith, R. D., Meerding, W. J., De Zwart, O., Brug, J. & Beutels, P. (2007), 'Precautionary behavior in response to perceived threat of pandemic influenza', *Emerging Infectious Diseases* **13**(9), 1307.

Sadique, Z. (2006), 'Individual freedom versus collective responsibility: An economic epidemiology perspective', *Emerging Themes in Epidemiology* **3**, 12.

Saikai, Y., Hurley, T. M. & Mitchell, P. D. (2021), 'An agent-based model of insect resistance management and mitigation for Bt maize: a social science perspective', *Pest Management Science* **77**(1), 273–284.

URL: *https://onlinelibrary.wiley.com/doi/abs/10.1002/ps.6016*

Salathé, M. & Bonhoeffer, S. (2008), 'The effect of opinion clustering on disease outbreaks', *Journal of The Royal Society Interface* **5**(29), 1505–1508.

Savary, S. & Willocquet, L. (2020), 'Modeling the impact of crop diseases on global food security', *Annual Review of Phytopathology* **58**(1), 313–341. PMID: 32511041.
URL: *https://doi.org/10.1146/annurev-phyto-010820-012856*

Savary, S., Willocquet, L., Pethybridge, S. J., Esker, P., McRoberts, N. & Nelson, A. (2019), 'The global burden of pathogens and pests on major food crops', *Nature Ecology & Evolution* **3**(3), 430–439.

Schuster, P. & Sigmund, K. (1983), 'Replicator dynamics', *Journal of Theoretical Biology* **100**(3), 533–538.

Sedgwick, P. (2013), 'Questionnaire surveys: sources of bias', *BMJ* **347**.

Sett, S., Prasad, A. & Prasad, M. (2022), 'Resistance genes on the verge of plant–virus interaction', *Trends in Plant Science* (S1360-1385(22)00183-2).
URL: *https://doi.org/10.1016/j.tplants.2022.07.003*

Shaw, M. W. (1995), 'Simulation of population expansion and spatial pattern when individual dispersal distributions do not decline exponentially with distance', *Proceedings of the Royal Society B: Biological Sciences* **259**(1356), 243–248.
URL: *https://royalsocietypublishing.org/doi/abs/10.1098/rspb.1995.0036*

Sherman, J., Burke, J. M. & Gent, D. H. (2019), 'Cooperation and coordination in plant disease management', *Phytopathology* **109**(10), 1720–1731.

Sherman, J. & Gent, D. (2014), 'Concepts of sustainability, motivations for pest management approaches, and implications for communicating change', *Plant Disease* **98**, 1024–1035.

Shi, B., Liu, G., Qiu, H., Wang, Z., Ren, Y. & Chen, D. (2019), 'Exploring voluntary vaccination with bounded rationality through reinforcement learning', *Physica A: Statistical Mechanics and its Applications* **515**, 171–182.
URL: *https://www.sciencedirect.com/science/article/pii/S0378437118312664*

Shim, E., Chapman, G. B., Townsend, J. P. & Galvani, A. P. (2012), 'The influence of altruism on influenza vaccination decisions', *Journal of The Royal Society Interface* **9**(74), 2234–2243.

Siegal, G., Siegal, N. & Bonnie, R. J. (2009), 'An account of collective actions in public health', *American Journal of Public Health* **99**(9), 1583–1587. PMID: 19608946.
URL: *https://doi.org/10.2105/AJPH.2008.152629*

Simon, H. A. (1990), Bounded rationality, *in* 'Utility and Probability', Springer, pp. 15–18.

Sinclair, S. & Agerström, J. (2021), 'Do social norms influence young people's willingness to take the COVID-19 vaccine?', *Health Communication* **0**(0), 1–8. PMID: 34114897.
URL: *https://doi.org/10.1080/10410236.2021.1937832*

Singerman, A., Lence, S. H. & Useche, P. (2017), 'Is area-wide pest management useful? The case of citrus greening', *Applied Economic Perspectives and Policy* **39**(4), 609–

634.

URL: *https://onlinelibrary.wiley.com/doi/abs/10.1093/aepp/ppx030*

Singerman, A. & Rogers, M. (2020), 'The economic challenges of dealing with citrus greening: The case of Florida', *Journal of Integrated Pest Management* **11**.

Singerman, A. & Useche, P. (2019), 'The role of strategic uncertainty in area-wide pest management decisions of Florida citrus growers', *American Journal of Agricultural Economics* **101**(4), 991–1011.

URL: *https://onlinelibrary.wiley.com/doi/abs/10.1093/ajae/aaz006*

Singh, R. P., Hodson, D. P., Jin, Y., Lagudah, E. S., Ayliffe, M. A., Bhavani, S., Rouse, M. N., Pretorius, Z. A., Szabo, L. J., Huerta-Espino, J. et al. (2015), 'Emergence and spread of new races of wheat stem rust fungus: continued threat to food security and prospects of genetic control', *Phytopathology* **105**(7), 872–884.

Sisterson, M. S. & Stenger, D. C. (2013), 'Roguing with replacement in perennial crops: conditions for successful disease management', *Phytopathology* **103**(2), 117–128.

Sisterson, M. S. & Stenger, D. C. (2018), 'Modelling effects of vector acquisition threshold on disease progression in a perennial crop following deployment of a partially resistant variety', *Plant Pathology* **67**(6), 1388–1400.

URL: *https://bsppjournals.onlinelibrary.wiley.com/doi/abs/10.1111/ppa.12833*

Smith, J. & Price, G. R. (1973), 'The logic of animal conflict', *Nature* **246**(5427), 15–18.

Stansly, P. A., Sánchez, P., Rodrıguez, J., Canizares, F., Nieto, A., Leyva, M. L., Fajardo, M., Suarez, V. & Urbaneja, A. (2004), 'Prospects for biological control of *Bemisia tabaci* (Homoptera, Aleyrodidae) in greenhouse tomatoes of southern Spain', *Crop Protection* **23**(8), 701–712.

Stefánsson, H. O. & Bradley, R. (2019), 'What is risk aversion?', *The British Journal for the Philosophy of Science* **70**(1), 77–102.
URL: *https://doi.org/10.1093/bjps/axx035*

Stokstad, E. (2020), 'United Kingdom breaks from EU farm subsidies'.
URL: *DOI: 10.1126/science.367.6476.348*

Sugden, R. (2013), 'The behavioural economist and the social planner: To whom should behavioural welfare economics be addressed?', *Inquiry* **56**(5), 519–538.
URL: *https://doi.org/10.1080/0020174X.2013.806139*

Sumner, D. A., Rickard, B. J. & Hart, D. S. (2001), 'Economic consequences of European Union subsidies for processing tomatoes', *University of California, Agricultural Issues Center, Spring* .

Sun, T. A. & Hilker, F. M. (2021), 'Comparison between best-response dynamics and replicator dynamics in a social-ecological model of lake eutrophication', *Journal of Theoretical Biology* **509**, 110491.
URL: *https://www.sciencedirect.com/science/article/pii/S0022519320303465*

Sykes, D. & Rychtáă, J. (2015), 'A game-theoretic approach to valuating toxoplasmosis

vaccination strategies', *Theoretical Population Biology* **105**, 33–38.

URL: *https://www.sciencedirect.com/science/article/pii/S0040580915000817*

Szyniszewska, A. M. (2020), 'CassavaMap, a fine-resolution disaggregation of cassava production and harvested area in Africa in 2014', *Scientific Data* **7**, 159.

Szyniszewska, A. M., Chikoti, P. C., Tembo, M., Mulenga, R., Gilligan, C. A., van den Bosch, F. & McQuaid, C. F. (2021), 'Cassava planting material movement and grower behaviour in Zambia: implications for disease management', *Phytopathology* **111**(11), 1952– –1962.

Taylor, P. D. & Jonker, L. B. (1978), 'Evolutionary stable strategies and game dynamics', *Mathematical Biosciences* **40**(1-2), 145–156.

Teeken, B., Olaosebikan, O., Haleegoah, J. A. S., Oladejo, E., Madu, T. U., Bello, A., Parkes, E., Egesi, C. N., Kulakow, P., Kirscht, H. & Tufan, H. A. (2018), 'Cassava trait preferences of men and women farmers in Nigeria: Implications for breeding', *Economic Botany* **72**, 263 – 277.

Tellier, A. & Brown, J. (2007), 'Stability of genetic polymorphism in host-parasite interactions', *Proceedings. Biological sciences / The Royal Society* **274**, 809–17.

Thaler, R. & Sunstein, C. (2008), Following the herd, *in* 'Nudge: Improving decisions about health, wealth and happiness', Yale University Press, New Haven, CT, pp. 53–73.

Thomas, B. (1984), 'Evolutionary stability: states and strategies', *Theoretical Population Biology* **26**(1), 49–67.

Thompson, R. N., Gilligan, C. A. & Cunniffe, N. J. (2018), 'Control fast or control smart: When should invading pathogens be controlled?', *PLOS Computational Biology* **14**(2), e1006014.

Tolasa, M., Gedamu, F. & Woldetsadik, K. (2021), 'Impacts of harvesting stages and pre-storage treatments on shelf life and quality of tomato (solanum lycopersicum l.)', *Cogent Food & Agriculture* **7**, 1863620.

Tomlinson, K. R., Bailey, A. M., Alicai, T., Seal, S. & Foster, G. D. (2018), 'Cassava brown streak disease: historical timeline, current knowledge and future prospects', *Molecular Plant Pathology* **19**(5), 1282–1294.

Toxvaerd, F. (2019), 'Rational disinhibition and externalities in prevention', *International Economic Review* **60**(4), 1737–1755.

Toxvaerd, F. & Rowthorn, R. (2022), 'On the management of population immunity', *Journal of Economic Theory* p. 105501.

Trinity, L., Merrill, S. C., Clark, E. M., Koliba, C. J., Zia, A., Bucini, G. & Smith, J. M. (2020), 'Effects of social cues on biosecurity compliance in livestock facilities: evidence from experimental simulations', *Frontiers in Veterinary Science* **7**, 130.

Tsutsui, Y., Benzion, U., Shahrabani, S. & Din, G. Y. (2010), 'A policy to promote influenza vaccination: A behavioral economic approach', *Health Policy* **97**(2), 238–

249.

URL: *https://www.sciencedirect.com/science/article/pii/S0168851010001326*

van den Bosch, F. & Gilligan, C. A. (2003), 'Measures of durability of resistance', *Phytopathology* **93**(5), 616–625. PMID: 18942985.

URL: *https://doi.org/10.1094/PHYTO.2003.93.5.616*

van den Bosch, F., Jeger, M. & Gilligan, C. (2007), 'Disease control and its selection for damaging plant virus strains in vegetatively propagated staple food crops; a theoretical assessment', *Proceedings of the Royal Society B: Biological Sciences* **274**(1606), 11–18.

URL: *https://royalsocietypublishing.org/doi/abs/10.1098/rspb.2006.3715*

van den Driessche, P. (2017), 'Reproduction numbers of infectious disease models', *Infectious Disease Modelling* **2**(3), 288–303.

URL: *https://www.sciencedirect.com/science/article/pii/S2468042717300209*

van Esse, H. P., Reuber, T. L. & van der Does, D. (2020), 'Genetic modification to improve disease resistance in crops', *New Phytologist* **225**(1), 70–86.

Vardavas, R., Breban, R. & Blower, S. (2007), 'Can influenza epidemics be prevented by voluntary vaccination?', *PLOS Computational Biology* **3**(5), e85.

Vardavas, R., Breban, R. & Blower, S. (2010), 'A universal long-term flu vaccine may not prevent severe epidemics', *BMC Research Notes* **3**, 92.

Velde, F. R. (2022), 'A model of economic activity in San Francisco during the 1918 influenza epidemic', *FRB of Chicago Working Paper* .

Vicente, L. N. & Calamai, P. H. (1994), 'Bilevel and multilevel programming: A bibliography review', *Journal of Global optimization* **5**(3), 291–306.

Vidavsky, F. & Czosnek, H. (1998), 'Tomato breeding lines resistant and tolerant to tomato yellow leaf curl virus issued from lycopersicon hirsutum', *Phytopathology* **88**, 910–4.

Vijeth, S., Dhaliwal, M. S., Jindal, S. K. & Sharma, A. (2018), 'Evaluation of tomato hybrids for resistance to leaf curl virus disease and for high-yield production', *Horticulture, Environment, and Biotechnology* **59**, 699–709.

Vroh, B. et al. (2011), 'Clean seed systems: sustainable production and distribution of clean banana and plantain.', *IITA-R4D Review* (6), 38–40.

Vyska, M., Cunniffe, N. & Gilligan, C. (2016), 'Trade-off between disease resistance and crop yield: a landscape-scale mathematical modelling perspective', *Journal of the Royal Society Interface* **13**(123), 20160451.

Wang, D., Marmo-Roman, S., Krase, K. & Phanord, L. (2021), 'Compliance with preventative measures during the COVID-19 pandemic in the USA and Canada: Results from an online survey', *Social Work in Health Care* **60**(3), 240–255.

Watkinson-Powell, B., Gilligan, C. A. & Cunniffe, N. J. (2020), 'When does spatial diversification usefully maximize the durability of crop disease resistance?', *Phytopathology* **110**(11), 1808–1820.

Whiteside, J. et al. (1970), 'Etiology and epidemiology of citrus greasy spot', *Phytopathology* **60**(10), 1409–1414.

Wilensky, U. & Stroup, W. (1999), 'Learning through participatory simulations', *Computer Supported Collaborative Learning (CSCL'99)* .

Wölfl, B., Te Rietmole, H., Salvioli, M., Kaznatcheev, A., Thuijsman, F., Brown, J. S., Burgering, B. & Staňková, K. (2022), 'The contribution of evolutionary game theory to understanding and treating cancer', *Dynamic Games and Applications* **12**(2), 313–342.

Wu, B., Fu, F. & Wang, L. (2011), 'Imperfect vaccine aggravates the long-standing dilemma of voluntary vaccination', *PLOS ONE* **6**(6), 1–7.
URL: *https://doi.org/10.1371/journal.pone.0020577*

Xia, H., Wang, H. & Xuan, Z. (2010), 'Opinion dynamics: Disciplinary origins, recent developments, and a view on future trends', *Phys. Rev* **18**(5), 35–39.

Yan, Z., Pérez-de Castro, A., Díez, M. J., Hutton, S. F., Visser, R. G. F., Wolters, A.-M. A., Bai, Y. & Li, J. (2018), 'Resistance to tomato yellow leaf curl virus in tomato germplasm', *Frontiers in Plant Science* **9**.
URL: *https://www.frontiersin.org/article/10.3389/fpls.2018.01198*

Yang, L.-N., Nkurikiyimfura, O., Pan, Z.-C., Wang, Y.-P., Waheed, A., Chen, R.-S., Burdon, J. J., Sui, Q.-J. & Zhan, J. (2021), 'Plant diversity ameliorates the evolutionary development of fungicide resistance in an agricultural ecosystem', *Journal of Applied Ecology* **58**(11), 2566–2578.

Yousefpour, A., Jahanshahi, H. & Bekiros, S. (2020), 'Optimal policies for control of the novel coronavirus disease (COVID-19) outbreak', *Chaos, Solitons and Fractals* **136**, 109883.
URL: *https://www.sciencedirect.com/science/article/pii/S0960077920302836*

Yuan, X., Chen, C., Bassanezi, R. B., Wu, F., Feng, Z., Shi, D., Li, J., Du, Y., Zhong, L., Zhong, B. et al. (2021), 'Region-wide comprehensive implementation of roguing infected trees, tree replacement, and insecticide applications successfully controls citrus huanglongbing', *Phytopathology* **111**(8), 1361–1368.

Zaffaroni, M. & Bevacqua, D. (2022), 'Maximize crop production and environmental sustainability: Insights from an ecophysiological model of plant-pest interactions and multi-criteria decision analysis', *European Journal of Agronomy* **139**, 126571.
URL: *https://www.sciencedirect.com/science/article/pii/S1161030122001198*

Zhang, H., Zhang, J., Zhou, C., Small, M. & Wang, B. (2010), 'Hub nodes inhibit the outbreak of epidemic under voluntary vaccination', *New Journal of Physics* **12**(2), 023015.

Zhang, R., Brennan, T. J. & Lo, A. W. (2014), 'The origin of risk aversion', *Proceedings of the National Academy of Sciences* **111**(50), 17777–17782.

Zhao, Y., Deng, H., Hu, R. & Xiong, C. (2022), 'Impact of government policies on seed innovation in China', *Agronomy* **12**(4), 917.

Zheng, Y. & Goodhue, R. E. (2021), 'Cross-crop spatial externalities of pesticide use:

Management of lygus bugs in the San Joaquin Valley of California', *2021 Annual Meeting* .

Zhu, L., Smith, C., Fritz, A., Boyko, E. & Flinn, M. (2004), 'Genetic analysis and molecular mapping of a wheat gene conferring tolerance to the greenbug (*Schizaphis graminum Rondani*)', *TAG. Theoretical and Applied Genetics. Theoretische und angewandte Genetik* **109**, 289–93.