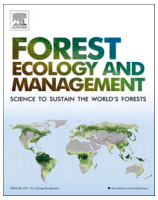
Forest Ecology and Management 566 (2024) 122098

Contents lists available at ScienceDirect

Forest Ecology and Management

journal homepage: www.elsevier.com/locate/foreco



Evaluating the impact of an invasive pathogen on tree population decline: An evidence based modelling approach

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ARTICLE INFO

Keywords:
Invasive forest pathogen
Ash dieback
European ash
Hymenoscyphus fraxineus
Compartmental model
Population dynamics
Epidemiological dynamics

ABSTRACT

An upsurge of invasive forest pathogens (IFPs) has been causing widespread damage to forest ecosystems worldwide. Modelling future forest loss caused by IFPs is challenging, as it requires a sophisticated understanding of the pathogen-hosts-surrounding interactions. We developed a complexity-appropriate model using an evidence-based approach to predict the decline of the European ash (*Fraxinus excelsior* L.) population caused by ash dieback (*Hymenoscyphus fraxineus*) in a British deciduous woodland. Our model predicts that (1) the ash population will decline by ~26 % in the next 10 year; (2) an ±10 % relative error in mortality survey would cause a ~8 % bias in 10-year population decline; and (3) a 5 % increase in resistant trees would save ~3 % population over 10 years. Our research demonstrates the merit of systematic reviews in balancing model complexity against generalisation. By scaling up the methodology to other IFPs, it is possible to forecast forest health with various management scenarios.

1. Introduction

Forest ecosystems cover ~42 million km^2 of Earth accounting for ~30 % of the global land surface (Bonan, 2008; Keenan et al., 2015). Over the past few decades, there has been a steep increase in emerging infectious diseases (EIDs) across the world's forested regions (Eriksson et al., 2019; Ghelardini et al., 2017; Lovett et al., 2016; Nahrung and Carnegie, 2020; Santini et al., 2012; Xu et al., 2006). The sudden rise in these diseases is closely linked to invasive forest pathogens (IFPs), which are primarily introduced by human vectors through intercontinental traffic and trade (Aukema et al., 2011; Ghelardini et al., 2017; Ramsfield et al., 2016; Roy et al., 2014; Stenlid et al., 2016). Once established, these pathogens disperse rapidly through wind, water, and animal vectors, having substantial effects on forest ecosystems (Ghelardini et al., 2017). In many cases, forest EIDs result in serious mortality events, e.g. in the case of Dutch elm disease (*Ophiostoma novo-ulmi*), sudden oak death (*Phytophthora ramorum*), and chestnut blight (*Cryphonectria parasitica*) (Loehle et al., 2022). The most devastating forest epidemics are often caused by fungal or fungal-like infections (Ghelardini et al., 2017; Santini et al., 2012), the substantial rise of which is likely to alter the structure and composition of forests worldwide.

Modelling future forest losses caused by EIDs is a powerful forecasting tool, and may aid the prioritisation of effective management (Sturrock et al., 2011). At the local or landscape scale, forest epidemiological models are used as a forecasting tool for evaluating disease-control strategies (Chavez et al., 2015; Cunniffe et al., 2016; Jeger, 2004). They also help to improve targets for data collection and surveillance (Chavez et al., 2015). At the broader scales, key epidemic parameters can be estimated from epidemiological models for different sites and different host-pathogen systems (Lessler et al., 2016). Subsequently, comparisons between these parameters can be used to explore associations with several covariates, ultimately identifying potential variables that help control the disease.

Developing an 'appropriate' and useful epidemiological model is, however, challenging. The outcomes of forest epidemics depend on complex interactions within the environment-host-pathogen disease triangle (La Porta et al., 2008; Nnadi and Carter, 2021; Ramsfield et al., 2016; Santini and Ghelardini, 2015; Simler-Williamson et al., 2019; Sturrock et al., 2011). A simple model with homogeneous assumptions could not capture these complexities. Environmental variables, especially temperature and water availability, often influence both host and pathogen life cycles simultaneously. In Europe, for example, the

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https://doi.org/10.1016/j.foreco.2024.122098
Received 25 March 2024; Received in revised form 14 June 2024; Accepted 15 June 2024
Available online 20 June 2024

elevated winter precipitation may raise the water tables to kill roots, making the host trees vulnerable to pathogen growth, reproduction, and infection (La Porta et al., 2008; Sturrock et al., 2011). Meanwhile, fungal pathogens may benefit from a humid condition, as water is essential for their survival, sporulation, and dispersal (La Porta et al., 2008). With an increase in rainfall, viable spores are able to disperse through wind-blown rain or rain splash (Davidson et al., 2002). This results in greater inoculum pressure of the disease (Peterson et al., 2015). Temperature also has a diverse range of effects on the phyto-physiological conditions of the host trees. Hot weather may facilitate host stress under prolonged summer drought, thus increasing host susceptibility to root pathogen, wound and sapwood colonisers (Kliejunas et al., 2009; Sturrock et al., 2011). In some cases, however, hot summers suppress the spread of diseases. *Cronartium ribicola*, the causing agent of white pine blister rust, typically germinates and causes infections between 0 and 20°C (Van Arsdel, 1965). Temperature above 35°C is lethal for its mycelium to survive in the leaf (Van Arsdel, 1972).

Given the complex nature of EID systems, a key challenge for modellers lies in the trade-off between incorporating more details and keeping more generality. For policy use, an effective forecasting model should be straightforward and applicable across various scenarios. The current paper aims to predict the extent of tree host population decline, using ash dieback, caused by an invasive fungus *Hymenoscyphus fraxineus*, as a case study. The fungal pathogen originates from East Asia (Enderle et al., 2019; Nielsen et al., 2017) and was brought to Europe primarily through the trade of infected wood (Husson et al., 2012; Solheim and Hietala, 2017). Since its first detection in Poland in 1992 (Przybył, 2002), *H. fraxineus* spread rapidly to most of the European countries (Solheim and Hietala, 2017), colonising the entire native distribution range of the host tree (Gross et al., 2013). The European ash tree (*Fraxinus excelsior* L.) is native throughout Europe, covering 64 % of the continent's land area (Thomas, 2016). The emergence of ash dieback has caused large-scale mortality rates of ash trees throughout its range (Dobrowolska et al., 2011; Downie, 2017; Enderle et al., 2019; McKinney et al., 2014; Pautasso et al., 2013; Skovsgaard et al., 2017). The extent of ash population decline and its impact remains uncertain, although a pan European study suggests a maximal 70 % mortality (Coker et al., 2018).

In this study, we present a model that will (1) forecast the ash population decline rate in the short term (i.e. 10 and 15 years since the first mortality); (2) report the uncertainty of the forecasts caused by data errors; (3) assess how alternative scenarios (e.g. presence of resistant hosts) may affect mortality rates. Our model is systematically justified by an evidence-based modelling framework, which allows us to determine the most appropriate level of model complexity. This methodology is scalable to other ecological communities, making our paper a pioneering study that investigates how ecological models, crafted through systematic review, can effectively bridge the gap between scientific evidence and practical management applications.

2. Materials and methods

2.1. Site description

Our study site is Wytham Woods, Oxfordshire, Britain (51°46' N, 1°19' W). The woodland is situated ~5 km northwest of the city of Oxford, owned and managed by the University of Oxford since 1942. Ash trees have long been a part of the woods and were widely planted by the University between 1950 and 1962 (Kirby, 2020). During the late 20th century, ash spread rapidly and generally became one of the most abundant tree species in the woods (Kirby, 2020), despite a high grazing pressure during the 1980s and 1990s (Kirby, 2020; Savill et al., 2010). In 2017, ash dieback disease reached the woods, followed by substantial damage to the ash canopy. Much of the woodland has been assigned to minimal intervention management strategies which enables the exertion of selection pressure by the pathogen in the ash population with varying host genetic resistance (Kirby, 2020).

2.2. Experimental design and data gathering

Vegetation surveys have taken place in 164 "Dawkins" plots (10 m x 10 m) since the 1970s (survey years: 1974, 1991, 1999, 2012, and 2018) creating a network of long-term monitoring plots across Wytham Woods (Dawkins and Field, 1978). After the detection of ash dieback in Wytham, signs of the disease and canopy decline were recorded in 268 ash trees across the plot network.

In 2020, a series of 10 plots (40 m x 40 m) were set up to monitor the multitrophic impact of ash dieback in Wytham Woods. Half of the plots were established in ash dominated sites and half in sites dominated by beech (Fagus sylvatica), sycamore (Acer pseudoplatanus), and oak (Quercus robur), representing future scenarios when ash has been replaced by other species. A tree census was conducted annually between 2020 and 2023 where the species and diameter of all trees above 1 cm in each plot were recorded. In each plot material from six litter traps were gathered monthly and categorised into leaves, seeds, and twigs which were weighed to the closest two decimals. The seeds were further identified to species.

2.3. Systematic review

We established a qualitative understanding of the disease to aid the conceptualising of the model. This was achieved by a systematic review of 100 peer reviewed papers that had surveyed ash dieback, followed by mapping host-pathogen-environment interactions through a Causal Loop Diagram (CLD). A CLD represents a system by nodes and connections, where nodes represent variables in the system, and connections are cause-and-effect relationships (hereafter referred to as 'causal influence') (Barbrook-Johnson and Penn, 2022). On 21st May 2023, we performed searches in Web of Science Core Collection and Scopus with the search terms TOPIC = ("Fraxinus excelsior" OR "F. excelsior" OR "common ash" OR "European ash") AND ("H. pseudoalbidus" OR "H. fraxineus" OR Hymenoscyphus OR Chalara OR "C. fraxinea" OR "ash dieback"), which yielded 386 papers (duplicates removed) (Supplementary Table S1). Papers without abstracts or those with irrelevant data were removed, resulting in 100 research papers and four reviews. For research papers, the abstracts were analysed and causal influences among environmental, host- and pathogen-related variables were extracted. For review papers, full text analyses were conducted with the same purpose. As a result, 26 variables (e.g. summer temperature, soil moisture, honey fungus) and 42 causal influences were mapped into a CLD (Supplementary Table S2, Figure S1).

Each causal influence was thereafter categorised into either 'strong' or 'weak' according to past findings. A causal influence is 'strong' if numerous studies support it (e.g. nine papers described a positive effect of soil moisture on ash dieback severity), or the effect of the causal influence is quantified to be large (e.g. a 45 % decrease of basal area increment caused by ash dieback reported by Metzler et al. (2012)). Otherwise, the causal influence is considered to be 'weak' (see detailed justifications in Supplementary Table S2). This feature is visualised by the strength of connections in the CLD (Supplementary Figure S1).

2.4. Model description

A list of the causal influences was integrated into the epidemiological model. These causal influences were (1) 'strong', (2) had a direct impact on ash dieback dynamics, and (3) had corresponding data available for model calibration. Seven causal influences adhered to the selection criteria and were considered in the model (Supplementary Table S3).

In the model, we divided the ash population into four classes: susceptible sapling (S_{sap}), infectious sapling (I_{sap}), susceptible adult (S_{adu}), and infectious adult (I_{adu}). S_{sap}, I_{sap}, S_{adu}, and I_{adu} denote the number of

ash trees in each class. Susceptible individuals are prone to disease, but are yet to be infected. Once infected, the individuals transit into the infectious class, namely they are able to spread the fungus. Some diseases have latent periods, when individuals are infected but are not infectious. However, once an ash tree is infected, the fungus reproduces spores quickly in the following spring, leading to its fast-spreading nature. Therefore, we assumed that infected individuals skip the latent period and become infectious directly. Saplings are assumed to become adults at a constant rate (the growth rate g). But they also experience a natural mortality rate μ, mostly from light competition and deer browsing. The model is formulated as follows (a detailed description can be found in Method S1):

$$\frac{dS_{sap}}{dt} = \nu S_{adu} + \nu(1-q)I_{adu} - \beta S_{sap}(I_{sap} + I_{adu}) - (\mu + g)S_{sap}, \quad (1)$$

$$\frac{dI_{sap}}{dt} = \beta S_{sap}(I_{sap} + I_{adu}) - (\mu + g(1-\varphi))I_{sap} - \rho_{sap}I_{sap}[-\delta], \quad (2)$$

$$\frac{dS_{adu}}{dt} = gS_{sap} - \beta S_{adu}(I_{sap} + I_{adu}), \quad (3)$$

$$\frac{dI_{adu}}{dt} = \beta S_{adu}(I_{sap} + I_{adu}) + g(1-\varphi)I_{sap} - \rho_{adu}I_{adu}[-\delta], \quad (4)$$

For model parameters, the meaning, unit, and estimations are summarised in Table 1.

2.5. Prediction, validation, and sensitivity analysis

To predict the future decline of ash population, we simulated the model with an initial population of 268 trees and a sapling ratio of 0.069, as observed in the disease incidence records. It started with a single infectious individual, and updated the population size of all classes every time step (= 0.01 yr). The time step was kept as a small value, following the convention of simulating differential equations, to ensure the continuity of the predicted trajectory. The predicted result should be interpreted with a yearly time scale.

Validation was performed to ensure the model accuracy using data from regions that have already experienced a long period of mortality due to ash dieback. The datasets were adopted from Matisone et al. (2021), Timmermann et al. (2017), and Klesse et al. (2021), respectively. Following the same methodology as that at Wytham Woods, the key parameter ρ_{adu} was estimated from maximum mortality observed at an early stage of the disease. Then, the observed long-term population decline time series were compared with model predictions to assess if the model reproduced tree population decline curves that were similar to the observed curve at each site.

To assess how data uncertainty influences predictions, we conducted a sensitivity analysis by systematically varying model parameters and observing the resulting changes in predicted population decline. In each case, we held all other model parameters constant and assumed a relative error in estimating a single parameter. The relative error ranges were between 1 % and 10 %. With the hypothetical errors, we re-ran the model again and calculated the proportion of population decline in 10 years and 15 years from 2021, the time when mortality was assumed to begin. As for the transmission rate per capita β, because its value is obtained by achieving best alignment between observed and predicted disease incidence rate (Table 1; Supplementary Figure S4), we introduced the relative errors of the disease incidence data rather than the value of β itself.Another source of uncertainty is the proportion of resistant ash trees. In this study, we assumed three levels of genetic resistance (i.e. fully susceptible, intermediate resistant, and resistant) following Evans (2019). The heritability of genetic resistance was modelled by transition probabilities among individuals of different resistance levels. A full description of the tree resistance model is described in Supplementary Method S2. From the forest management perspective, removal of diseased trees may help population recovery as

Table 1
A summary of model parameters and their estimation strategies.

Symbol	Meaning	Unit	Estimation and related causal influence
ν	regeneration rate	yr^{-1}	0.712. Estimated from sapling-adult ratio observed in 1975 (Kirby, 2020), where sapling was defined as trees with a d.b.h. < 10 cm. *'ash dieback → seedling recruitment (–)'*
q	reduction of regeneration caused by the disease (%)	/	35 %. Estimated by seed productivity loss from 2021–2022 in Wytham Woods (see Supplementary Figure S2). *'ash dieback → seedling recruitment (–)'*
β	transmission rate per capita	$(yr^{-1})^{-1}$	0.0174. Calibrated by disease records in 2018. We ran the model with different values of β to search for best alignment between observed and predicted disease incidence rate (Supplementary Figure S4).
μ	natural mortality rate*	yr^{-1}	0.643. Estimated from the difference in sapling-adult ratio observed in 1975 and 2018 (Kirby, 2020). This difference reflected the pressure from increased conspecific competition and potential deer browsing. 'canopy closure (low light) → sapling growth (–)' *'browsing → seedling recruitment (–)'*
g	growth rate	yr^{-1}	1/25. Generally, ash trees reach maturity after 25 years (Hein, 2003) *'ash dieback → sapling growth (–)'*
ρ_{sap}	disease-induced mortality rate for saplings	yr^{-1}	0.0877. Firstly, the ratio ρ_{sap}/ρ_{adu} was estimated from maximum mortality rates of ash woodlands and saplings, synthesised by Coker et al. (2018). Then, ρ_{sap} was calculated by multiplying ρ_{adu} with the ratio. *'ash dieback → survival (–)'*
ρ_{adu}	disease-induced mortality rate for adults	yr^{-1}	0.0495. Estimated from the average annual mortality rate (AMR) during 2021–2022 in Wytham Woods (Supplementary Figure S3). *'ash dieback → survival (–)'*
δ	delayed parameter**	yr	4. The disease arrived at Wytham Woods in 2017. In 2021–2022, a moderate level of ash mortalities was observed (Supplementary Figure S3). This low level of mortality can be seen as the initial phase of the logistic mortality curve, as introduced by Coker et al. 2018. *'ash dieback → survival (–)'*
φ	reduction of growth caused by the disease (%)	/	26 % (Metzler et al., 2012) *'ash dieback → sapling growth (–)'*

* *natural mortality rate:* mortality caused by light competition, damage from deer or whatever reasons. Details can be found on Section 2.2.

** *delayed parameter:* mortality generally occurs after a few years of infection. Therefore, ash mortality in the present year is assumed to depend on the number of infectious individuals in δ years ago. The latter is denoted by $I_{sap}[-\delta]$, $I_{adu}[-\delta]$.

it promotes crossings between resistant parents. We therefore modelled the effect of removing a small proportion (5 %) of diseased adults on the dynamics of resistant trees, and compared the modelling results with a 'no intervention' scenario. Throughout the study, all simulations were performed in R 4.2.0 (R Core Team, 2022) using ecode package (Wu, 2024).

3. Results

3.1. Projected population decline

Overall, the model predicted a population decline of 25.9 % over the period of 2021–2030, with a 25.0 % reduction in adult trees and a 27.2 % decrease in saplings (Fig. 1). We further extended the prediction to 15 years, obtaining an overall decline of 38.6 % (from 2021–2035), with adult and sapling populations declining by 37.9 % and 39.6 %, respectively.As expected, the susceptible individuals dropped rapidly. Our model predicted that all susceptible adults transition to infectious individuals within two years (~1.9 yr). Although susceptible saplings also experienced a sharp decline, their population stabilised at 15 individuals within three years (~2.4 yr) of exposure to the disease.

The model was then benchmarked against long-term time series of tree mortality observed in three typical sites. The results showed a general alignment between observed and predicted curves (Fig. 2), although the Latvian site (Matisone et al., 2021) exhibited a slower increase in cumulative mortality rate than the predicted values after 15 years' exposure to the disease. On the other hand, the Swiss site (Klesse et al. 2021) exhibited a slightly higher rate of increased tree mortality after 10 year's exposure to the disease, but the mortality dynamics before that point was captured by the model.

3.2. Sensitivity analysis

The biggest prediction uncertainty stems from errors in mortality surveys. Notably, we observed an uncertain range of 8.0 % in the 10-year population decline ratio (and 11.1 % for the 15-year ratio) caused by a relative error of ±10 % in ρ_{adu}. For many other estimates, such as uncertainties in ash growth measurements, seed rain surveys, and natural mortality calculations, the predicted decline ratio exhibited moderate sensitivity. Specifically, a ±10 % change of parameters g, ν, and μ caused variations of 3.4 %, 3.3 %, and 3.1 % in the 10-year decline ratio (4.2 %, 4.2 %, and 3.9 % for the 15-year ratio). Following these, uncertainty from δ led to 3.0 % variations for the 10-year period and 1.8 % for the 15-year period. The estimation for reduction in reproduction q resulted in a 2.1 % change for the 10-year period and 2.6 % for the 15-year period. Uncertainties related to disease-induced reduction in growth and sapling mortality appeared to have much less effects (1.0 %, 1.3 % for ϕ) and (1.0 %, 1.0 % for ρ_{sap}).

Surprisingly, our study revealed that disease incidence estimates had little influence on mortality predictions (Fig. 3). A ±10 % uncertainty in disease incidence ratio led to mere 0.2 % changes in 10-year decline rate and 0.2 % for the 15-year decline rate. Therefore, β appeared to be of little importance for estimation.

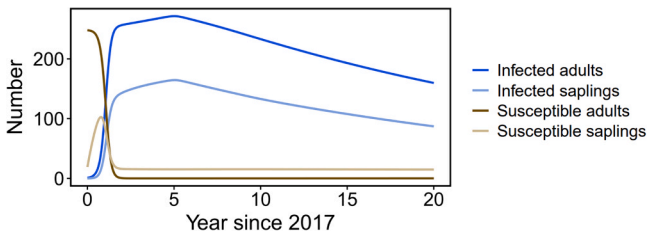

Fig. 1. Predicted epidemiological dynamics of ash dieback disease in Wytham Woods. The simulation started with one infectious individual in 2017. All model parameters estimated or calibrated based on past records.

A small proportion of resistant trees could effectively buffer against the disease (Fig. 4). On average, with every 5 % increase in resistant trees, we observed a ~3 % increase in tree survival over a 10-year period and a 4.4 % increase over 15 years. Interestingly, as the proportion of initial resistant individuals increased, the efficiency in expanding resistant populations rose at first, but then dropped after 15 % of initially resistant individuals. With 40 % initially resistant individuals, however, the ratio of resistant individuals only increased by 3.5 % from 2021–2025, and by 4.8 % from 2021–2035. From a forest management perspective, removal of diseased trees could further help the expansion of resistant populations (Supplementary Figure S5). If 5 % of diseased trees are removed, the increase in the proportion of resistant trees could reach 17 %, compared with 8 % under a 'no intervention' scenario.

4. Discussion

4.1. European ash decline

The extent of population decline of the European ash tree in the future is largely unknown. This poses challenges to estimate extinction risk of ash-obligate species (Hultberg et al., 2020), and makes targeted conservation efforts difficult (Mitchell et al., 2014). Our study presents an evidence-based modelling framework that demonstrates how epidemic survey data can be converted into future forecasts. We predicted a 10-year ash population decline of 25.9 % and a 15-year decline of 38.6 % in Wytham Woods (Oxford, Britain). The model was benchmarked against mortality data from three typical sites that experienced a long history of disease. Our prediction is more optimistic compared to a data synthesis of 36 woodland surveys by Coker et al., 2018, but slightly pessimistic compared with estimated survival probability curve based on large-scale ICP Forests survey (a total of 407 plots across 27 countries with the presence of European ash or narrow-leafed ash) (George et al., 2022). In the review by Coker et al., 2018, a logistic curve was used to estimate the proportion of ash population decline since the exposure to ash dieback. According to the curve for woodland surveys, on average, a decline of ~20 % would be observed within only five years since first death. The decline rate would further reach ~50 % in another five years before stabilising at ~60 %. Our predictions demonstrated a less severe situation in Wytham Woods. We caution that different predictions are rooted in different assumptions. In our model, key assumptions are: firstly, that the population decline rate was assumed to be proportional with infectious individuals. Secondly, that an infected tree succumbed to death after several years of infection, causing a delayed effect that was described by parameter δ. Thirdly, that disease transmission was rapid. These assumptions added to the conclusion that the maximum rate of population decline can occur rapidly after the first death. Therefore, the observed annual mortality rate (AMR) of 4.95 % during 2021–2022 in Wytham Woods was assumed to have already approached the maximal mortality. This value theoretically yields a 5-year population mortality rate of 24.75 %, which is larger than Coker's estimates. However, as the population becomes smaller, the mortality slows down, leading to a lower population decline rate. In addition, the population decline is also compensated by few recruitments. Hence, the net population decline is smaller than cumulative mortality, which is further much smaller than the maximum AMR multiplied by year. This leads to a prediction of a mild decline in population size even though mortality starts with an AMR of 4.95 %.

Our predictions generally concur with estimations from a large-scale ICP Forests survey (George et al., 2022). The ICP Forest survey spanned 1987–2020, and recorded defoliation and mortality of ash populations in 407 plots throughout continental Europe. Assuming non-simultaneous infection in different countries, George et al., (2022) found an overall survival probability of 70 % after 20 years of exposure to the disease (i.e. ~15 years since the survival probability curve began to drop). We estimated a slightly higher rate of 15-year population decline. However, the observed AMR in Wytham Woods around

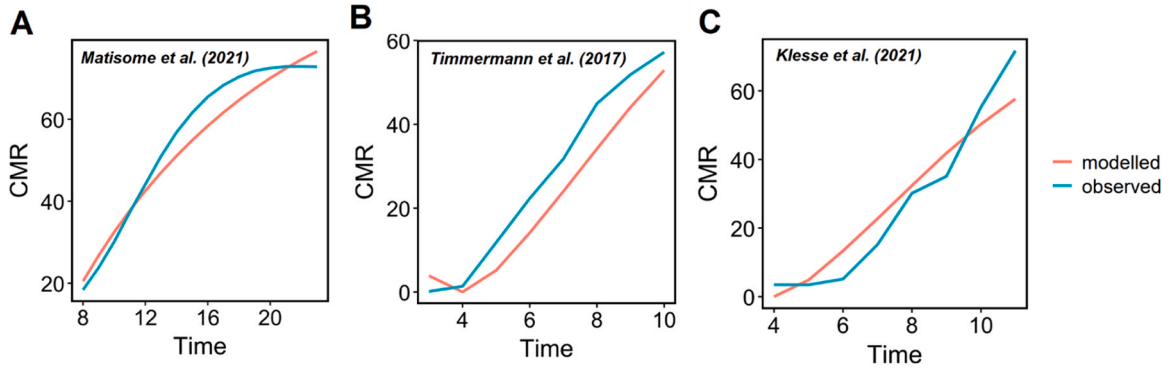

Fig. 2. Model Validation using data from regions with long disease history and long-term tree mortality records. The *x*-axis represents the year since ash dieback arrives at the site, and the *y*-axis represents the cumulative mortality rate (CMR, %), the proportion of population removed by disease compared to pre-disease baseline. Different panels show modelled and observed mortality curves in typical sites located in different countries, (A) Latvia (Matisone et al. 2021); (B) Norway (Timmermann et al. 2017); (C) Switzerland (Klesse et al. 2021).

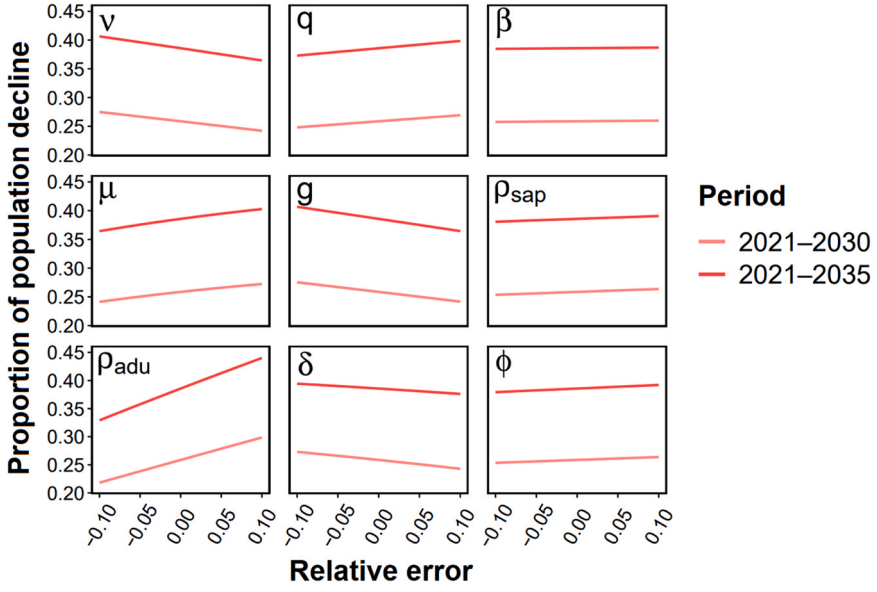

Fig. 3. Sensitivity of population decline forecasting. Each panel showed the predictions of population decline under a 0~±10 % change of the corresponding model parameter while keeping other parameters constant. A negative relative error means to decrease the value of the original estimate. For transmission rate β, the relative error was introduced on disease incidence data, rather than the parameter β itself, because β was calibrated rather than being directly estimated by data. For other parameters, the relative error was directly introduced on the parameter itself to approximate data uncertainty.

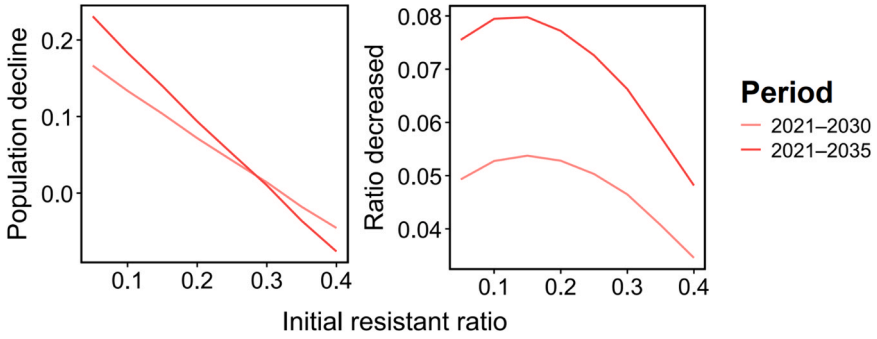

Fig. 4. Sensitivity of predicted population decline to the proportion of resistant trees (i.e. resistant ratio). Different scenarios were used for simulations, with an initial resistance ratio of 5–40 % and an increased step of 5 %. Population decline and the change in resistant ratio were recorded for the period 2021–2030 and 2021–2035. Negative population decline rate indicates that population would increase even with the presence of ash dieback disease.

2021–2022 (with ash dieback first confirmed in 2017) is also higher than George et al.'s estimate at around year 5 of infection (close to 0). Therefore, our scenario began with higher mortality and, unsurprisingly, predicted an additional 8.57 % (= 38.57 % – 30 %) decline in population compared to George et al.'s scenario.

We also predicted that the number of susceptible saplings are kept at a low level (Fig. 1). The infected saplings also decreased steadily as the disease progressed. This aligns with the observations at Wytham Woods that many large saplings and young trees of 2–5 m died of ash dieback (Kirby, 2020). In forest stands of long disease history, there may even be a complete lack of saplings. In south-eastern Norway, over 80 % of the youngest trees in five monitoring plots died from 2009–2016 (Timmermann et al., 2017).

4.2. Limitations and data uncertainty

The decline of the ash population due to the disease is caused by a mixture of external variables, leading to substantial variations across different sites. Coker et al., (2018) reported a maximal mortality of ~70 % across 36 woodland surveys, while extreme cases with no mortality were also observed (e.g. Drenkhan et al., 2017; Giongo et al., 2017; Timmermann et al., 2017), even with a 10-year exposure to the disease (Drenkhan et al., 2017). Similarly, George et al. (2022) identified southern Scandinavia and north-eastern Europe as two mortality hotspots, while other regions exhibited relatively less ash mortality. High disease severity is often correlated with high summer rainfall (George et al., 2022; Marçais et al., 2023), mild summer temperature (Grosdidier et al., 2018; Grosdidier et al., 2020; Marçais et al., 2023; Pušpure et al., 2017), moderate alkaline soils (Turczański et al., 2021), dense crown canopy (Enderle et al., 2018; Grosdidier et al., 2020; Klesse et al., 2021; Matisone et al., 2021), presence of water bodies (Grosdidier et al., 2020; Laubray et al., 2023), and hitchhiking by secondary opportunistic pathogens such as *Armillaria* spp. (Bakys et al., 2011; Enderle et al., 2013; Lenz et al., 2016; Davydenko et al., 2019). For accurate forecasts, a model should balance the inclusion of more variables and preserving its generality to find calibration data. In this paper, we provided a possible strategy to solve the dilemma by combining qualitative and quantiative approaches in process-based modelling. Instead of developing models directly, we first established a qualitative understanding of the host-pathogen-environment system by conducting a systematic review, followed by mapping interactions among system variables in a CLD. By modelling critical interactions, which was supported by a large number of research findings or past quantitative estimates, we ensured that our model captured the most important complexity and was relatively easy to calibrate.A comprehensive understanding of how environmental conditions affect disease outcomes requires careful assessment of their impact on the pathogen life cycle, including the formation of apothecia, sporulation, mycelium survival, and rachis infection (Marçais et al., 2023; Laubray et al., 2023). For instance, tree density is often considered to enhance disease severity, but this relationship seems complicated in ash dieback. Some studies postulated that higher tree density promotes massive inoculum of *H. fraxineus*, which is a required condition for disease transmission and thus significant dieback (e.g. Grosdidier et al., 2020). However, with major crown loss, less leaf material is available for the fungus to overwinter and reproduce (Laubray et al., 2023). We choose to ignore this feedback loop in our model, given a general lack of census data on the pathogen life cycle. Additionally, in Wytham Woods, we did not find significantly higher mortality in ash dominated sites compared to non-ash dominated sites (Supplementary Figure S3). Therefore, these details were unnecessary and were left out to avoid false predictions.

Given that ash population decline is highly variable, it is important to assess whether data uncertainty would change prediction outcomes. Our results showed that a ±10 % change of adult mortality estimates ρ_{adu} lead to notable prediction bias (an uncertain range of 8.1 % for 10-year population decline and 11.1 % for 15-year decline). Since many surveys included hundreds of ash trees with less than a hundred deaths (Coker et al., 2018), we caution that overlooking a few mortalities would cause biases. For instance, a survey with a population size $n = 300$ and a true death number $n_D = 50$ would cause a 5.5 % bias in 15-year decline projections if five deaths were not observed. When the mortality number is high (e.g., $n_D = 203$ for Rosenvald et al., 2015), the bias drops dramatically. Therefore, we recommend that projections should be made for a whole woodland rather than a single plot. Models calibrated with data when $n_D < 5$ may be highly unreliable, since miscounting can easily cause a ~20 % prediction bias.

Though the remaining model parameters seem less crucial, it is important to caution that this conclusion assumes a ±10 % change for each model parameter. A few decades ago ash populations in Wytham Woods experienced rapid expansion; however, in the 1990s, ash seedlings struggled to survive due to high deer pressure (Kirby, 2020). The recent ash dieback disease also discourages ash regeneration, potentially benefiting its competitors like beech (*Fagus sylvatica*) and sycamore (*Acer pseudoplatanus*) (Thomas, 2016). Previous modelling research in Wytham suggested that sycamore could be a potential successor in the next 100 years (Needham et al., 2016). Without competition from ash seedlings, sycamore may thrive with high recruitments under an open canopy that was previously occupied by ash (Needham et al., 2016). This competition effect, however, is difficult to integrate into our model. In this case, the reproduction rate μ can therefore be set lower to provide more conservative estimates.

Finally, the present study only focuses on tree mortalities. Ash dieback also causes substantial ecological impacts on living ash, such as crown defoliation and productivity loss (Broome and Mitchell, 2017). These variables will further threaten ash-associated biodiversity (Hultberg et al., 2020) which are worth modelling to support ash management and policy decisions around ash dieback.

4.3. Broader uses

The present paper serves as a paradigm of how epidemiological modelling can be carefully used to generate predictions. Firstly, with a well calibrated model, diverse scenarios can be explored to obtain potential disease outcomes. This approach has also been widely used in previous modelling studies. For example, Macpherson et al. (2018) optimised timber forest rotation lengths to maximise net present value. With a blend of epidemiological compartmental models and a Faustmann optimal rotation length model, they were able to assess the net benefits of different harvesting strategies across varying disease scenarios. In the context of ash dieback disease, as previously noted, the modelling work by Needham et al. (2016) anticipated a gradual replacement of ash trees by sycamores. Although ash dieback had not been detected at that time, they explored scenarios with different ash population decline rates in the following 10 years, and made projections over the next 100 years.

From management perspective, an area of uncertainty revolves around the effect of a small fraction of resistant ash trees on population recovery trajectory (Evans, 2019; Plumb et al., 2019). This is important to make choices between ash breeding programmes and natural regeneration. To address this uncertainty, we examined various scenarios with different hypothetical proportions of resistant individuals. Our findings revealed that a small resistant population could effectively buffer against the population decline, although resistant trees may still support pathogen sporulation and new infections (Nielsen et al., 2017; Marcais et al., 2023), a process that was assumed in our model. Maintaining 15 % of resistant individuals could maximise the capacity of increasing resistant genotypes. We caution, however, that the extent that our model presents the reality of tree mating systems may affect the prediction outcomes. In many sites, it seems common that diseased ash trees flower heavily prior to death, which would perhaps add the chance of seedling establishment of fully susceptible trees, although it is unclear whether these seedlings could survive the disease. These processes are

currently under-recorded, and are thus difficult to incorporate into the model. In addition, at Wytham Woods, ash dieback had been detected four years earlier than the observation year, meaning that the epidemic is still at its early stage, and not all trees would have contracted the disease at the moment. It remains to see the actual effect of the pathogen on seed production when trees are severely impaired. From the point of forest management, removal of diseased ash trees could promote the crossings of resistant ash, thus further increasing the proportion of resistant genotypes. We modelled the effect of constantly removing 5 % of diseased adult ash trees, and found that the increase of resistant genotypes could rise to 17 % compared with 8 % in non-intervention scenarios. This highlights the possibility of this silvicultural strategy in disease control and forest recovery. We caution, however, that tree removal must be considered carefully as it might cause losses of specialist species and decomposable deadwoods, leading to unintended ecological consequences (Dahlsjö, 2023).

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Secondly, modelling helps to assess the impact of data uncertainty on prediction outcomes. By employing process-level sensitivity analysis, it becomes possible to transparently navigate how errors propagate through simulations (Brugnach, 2005). This not only offers insights into the appropriate use of the model but also prioritises further data collection for accurate prediction. Our results highlighted the importance of obtaining high quality data from disease mortality surveys. A relative error of ±10 % can result in ~8 % in 10-year predictions and ~11 % for 15 years. Furthermore, we emphasised that data errors may be amplified in predictions with longer timeframes, as evidenced by a wider uncertainty range in 15-year predictions compared to those for 10 years. This led us to question the validity of long-term predictions, such as those spanning 100 years, if sensitivity were not reported transparently.

Thirdly, our model can be scaled up to map the severity of ash dieback across Europe, and even synthesise global risks from IFPs. Despite heterogeneities in different environment-host-pathogen systems, our model possesses the essential parameters to understand forest epidemiological dynamics. These parameters can be easily applied to describe other IFPs. The ecological indicators used in this study, including the 10-year population decline rate, are also scalable for other diseases. Therefore, this framework can be extended to facilitate comparative studies across IFPs, developing comprehensive disease ratings. This may promote our understanding of plant epidemiological dynamics and inform global forestry regulations.

5. Conclusions

The surge of infectious forest diseases has been causing large-scale mortality events. Forecasting future loss of forest is challenging, as it requires detailed understanding of pathogens, hosts, and their interactions with the environment. However, a fully mechanistic model may lose its data fitting ability, leading to unreliable predictions. Our study developed an evidence-based modelling framework, which leveraged diverse sources of existing evidence, and enabled us to carefully create a complexity-appropriate model for accurate predictions. We showed that: (1) the ash population in Wytham Woods would decline by 25.9 % in the next 10 years and 38.6 % in the next 15 years.; (2) predictive population decline is strongly influenced by data errors in mortality surveys. An ±10 % relative error could introduce biases of 8.05 % in the 10-year population decline rate and 11.1 % for the 15-year decline rate; (3) resistant trees can benefit population recovery, but the impact is gradual. A 5 % increase in initial resistant trees resulted in a 3 % change in both population decline over a 10-year period. Our research serves as a case for how a systematically constructed model informs forest disease management through scenario building and process-level sensitivity analysis. The model holds promise for application to other forest pathogens, potentially enabling large-scale comparative studies to inform global invasive pathogen management.

Statement of authorship

HW designed and implemented the project, as well as data analysis. HW wrote the manuscript with significant contributions from CALD and advice from YM. CALD provided the field disease records.

CRediT authorship contribution statement

Yadvinder Malhi: Supervision. **Cecilia A. L. Dahlsjö:** Writing – review & editing, Supervision, Project administration. **Haoran Wu:** Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

Acknowledgement

This work is supported by the School of Geography and the Environment at the University of Oxford for MSc Biodiversity Conservation and Management degree thesis. We thank Dr Keith Kirby (University of Oxford) for providing disease records in Wytham Woods, and giving valuable suggestions on project framing and thesis writing. We also thank Dr Thomas Harwood (University of Oxford), Dr John Morris (University of Exeter), Dr Ruth Mitchell (The James Hutton Institute), Dr Emma Goldberg (Natural England), Dr Sean McMahon (Smithsonian Environmental Research Center), Dr Jessica Needham (Lawrence Berkeley National Laboratory), Dr Gillian Petrokofsky (Oxford Long-Term Ecology Laboratory) for useful advice on project framing and methodologies.

Statement of authorship

HW designed and implemented the project, as well as data analysis. HW wrote the manuscript with significant contributions from CALD and advice from YM. CALD provided the field disease records.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.foreco.2024.122098.

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