Research

Learning from history, predicting the future: the UK Dutch elm disease outbreak in relation to contemporary tree disease threats

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Expanding international trade and increased transportation are heavily implicated in the growing threat posed by invasive pathogens to biodiversity and landscapes. With trees and woodland in the UK now facing threats from a number of disease systems, this paper looks to historical experience with the Dutch elm disease (DED) epidemic of the 1970s to see what can be learned about an outbreak and attempts to prevent, manage and control it. The paper draws on an interdisciplinary investigation into the history, biology and policy of the epidemic. It presents a reconstruction based on a spatial modelling exercise underpinned by archival research and interviews with individuals involved in the attempted management of the epidemic at the time. The paper explores what, if anything, might have been done to contain the outbreak and discusses the wider lessons for plant protection. Reading across to present-day biosecurity concerns, the paper looks at the current outbreak of ramorum blight in the UK and presents an analysis of the unfolding epidemiology and policy of this more recent, and potentially very serious, disease outbreak. The paper concludes by reflecting on the continuing contemporary relevance of the DED experience at an important juncture in the evolution of plant protection policy.

Keywords: tree disease epidemics; Dutch elm disease; Phytophthora; biosecurity; biodiversity; epidemiological modelling

1. INTRODUCTION

The biosecurity threat from invasive pathogens to native plant communities, trees and woodland is now internationally recognized. Overwhelmingly, the main risk drivers are the expanding trade in plants and plant products, together with growing transportation [1–4]. Biologically complex and often emerging as disease threats because of an ability to coevolve with new hosts and to rapidly exploit the environments with which they come into contact, tree disease pathogens pose both a scientific and management challenge. While advances in epidemiological modelling techniques mean that the predictive science of tree disease epidemics is now much improved, many of the organisms that currently threaten trees and woodland (for instance, the Phytophthora fungus to be discussed below) were unknown to science before they were identified as a threat [5]. The knowledge base about a pathogen and its biological and epidemiological properties typically develops on the basis of laboratory research and field observation as a disease outbreak unfolds. This means that identifying risk before an organism has become established in a country or jurisdiction, essential if precautionary action is to be justified, is fraught with difficulty. Once established, effective management of a pathogen is similarly problematic. Disease development can be slow at first but if a sufficient density of susceptible hosts is present, infective inoculum may subsequently build up very rapidly, the disease eventually breaking out into the classic exponential phase that characterizes all epidemics. In human terms, effective management (either to contain the outbreak or slow its spread) is often complicated by the presence of large numbers of affected stakeholders, the difficulty of establishing clear lines of institutional and cost-sharing responsibilities, a clash of private and public interests and scientific uncertainty as to the possible consequences. In addition, establishing a public interest justification for intervention may often be difficult, even when important public goods such as biodiversity are at risk.

On the other hand, tree disease epidemics are not new and policymakers and those who advise them have various historical precedents on which they can draw to derive management lessons, seek to avoid...
past mistakes and attempt to lengthen institutional memory. Previous tree disease outbreaks have been documented and commented on. In the USA for instance, the chestnut blight (Cryphonectria parasitica) epidemic of the early twentieth century, which killed over 300 million native trees and profoundly altered the landscape of the eastern seaboard of the United States, is a frequently invoked point of historical reference in that country when present-day disease threats are under discussion (see, for instance [6,7]). In the UK, the Dutch elm disease (DED) outbreak of the 1970s, responsible for the death of some 30 million trees, has been the subject of various retrospective commentaries and expressions of regret [8,9], as well as an ongoing and very productive programme of plant pathology research. However, this landmark outbreak has yet to be systematically analysed in a policy-relevant manner, either as an empirical reference point on the dynamics of disease spread and its responsiveness to management, or as a source of learning experiences on the management of tree disease outbreaks. Indeed, despite its profound impact on the UK rural landscape and biodiversity, no official enquiry was ever conducted into the causes of the epidemic or into the adequacy or otherwise of the government response.

Our starting point for this paper is the contention that the DED epidemic, along with its biology, epidemiology, economic and institutional framing, deserves to be re-examined in relation to emerging threats to the health of trees, woodland and other habitat in the UK. The paper draws on an interdisciplinary investigation that used epidemiological modelling, informed by archival research and interviews with key informants, in order both to simulate the outbreak and to explore the sensitivity of outcomes to different management scenarios. The paper begins by presenting this reconstruction of the DED epidemic and the key deductions that can be drawn about the disease system and its degree of susceptibility to management.

The discussion then proceeds to explore some counterfactuals before examining the legacy of DED and the lessons from history. A final section reflects on the contemporary relevance of DED given a proliferating disease risk to trees and woodland from pathogens such as Phytophthora ramorum (inaccurately but popularly known as ‘sudden oak death’). We discuss the implications of a parallel predictive modelling exercise of so-called ‘ramorum blight’, similarly informed by an analysis of the recent and likely management options for this pathogen. The paper concludes by assessing similarities and differences with DED and the prospects for further major tree disease epidemics in the UK.

2. RECONSTRUCTING THE UK DUTCH ELM DISEASE EPIDEMIC

It began slowly at first, as all great epidemics tend to do. In 1965, the Forestry Commission Advisory Service at Alice Holt began to receive letters and telephone calls from members of the public describing die-back in elms. Elm (mainly Ulmus procera, Ulmus minor and Ulmus glabra) were important trees in the UK landscape, particularly the English elm, U. procera, widely planted as a hedgerow tree from the sixteenth century onwards but eventually to become one of the defining features of the lowland rural landscape after extensive planting and establishment during the parliamentary enclosures of the eighteenth century [10]. Our own estimates suggest a baseline population of 30 million elm trees of all species distributed as shown in figure 1a, with over half the elms concentrated in southern England [11]. By 1970, reports of elm death in various settings had increased dramatically, with hundreds of letters from gardeners, estate managers, farmers and from people making observations during walks and visits [12]. As one of our interviewees, a plant pathologist at Alice Holt, put it: ‘we started getting these reports of something funny happening because there were trees dying and people complaining about it...Originally we thought it was just an upsurge, but that didn’t happen, it just continued at a pace’ (interview with David Burdekin 2008). DED was not new to the UK and, as a fungal pathogen (Ophiostoma ulmi), was first recorded in the late 1920s, having arrived from mainland Europe, where it had become established throughout France, Germany, Holland and Belgium. This previous outbreak was limited in scope, however, with mortality rates rarely exceeding 20 per cent. The conventional wisdom, set down authoritatively by the Forestry Commission’s then Chief Pathologist, Tom Peace [13], regarded DED as an endemic, but manageable, tree disease problem that would periodically recur but that did not justify extensive and costly intervention.

With the benefit of hindsight, it can be seen that the volume and geographical spread of the reports coming into Alice Holt from 1969 onwards could have suggested that something had changed, both in terms

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of the rate of mortality and the speed with which the disease was spreading from the initial sites of infection around the ports of Avonmouth and London and around the areas of usage at Chatham. Later analysis would confirm that the pathogen responsible was a more virulent form of the already established organism, imported into the UK from North America on a consignment of rock elm (Ulmus thomasii) [14]. By the time of the first comprehensive Forestry Commission survey of elms conducted in 1971 [15], 700,000 of the estimated 18 million elms in southern England were dead or dying and a further 1.65 million exhibited evidence of disease. These figures were revised upwards as the decade progressed and by 1980 there were few mature elms remaining in the UK. The loss of over 30 million trees provoked a widespread sense of public loss as well as (judging from our interviews with key personnel involved in the outbreak) various expressions of professional regret among the scientists based at Alice Holt. Critics argued that the Forestry Commission had ‘done too little, too late’ [9,16] and hinted that the authorities should have been better coordinated and quicker to act once the disease had broken out. In a New Scientist editorial, John Tinker, for instance, argued that the compulsory felling policy put in place by the Commission was under-resourced and abandoned too early and that more should have been done to contain the spread of the disease by restricting movements of diseased timber [16].

In an effort to integrate an understanding of the interrelated biology, epidemiology and management response of the DED outbreak, our reconstruction explored the temporal and spatial dynamics of the epidemic across Great Britain. The English hedgerow outbreak has been modelled before, notably by Swinton & Gilligan [17], but without a spatial or management component, and treating hedgerow elm as an epidemiologically isolated system. This earlier study, while valuable as a contribution to understanding how outcomes were a function of very high transmission and lethality rates, is necessarily limited by its assumptions. Our spatial model [11] treats transmission as a spatial process that affects all trees. The distribution of all British elm species in woodland, hedgerow and urban areas was mapped (based on surveys both before and during the epidemic, combined with modern raster datasets) and differences in susceptibility between species treated using a host suitability index. Critically, the model allows local authority management regimes to be simulated, which permits the incorporation of policy scenarios derived from archival material and interviews. Management is thus viewed as an integral component of the pathosystem, able to affect the outcome of the modelled epidemic.

Figure 1b–e shows the rapid progress of the epidemic from the initial points of infection through to its nationwide impact. After 1981, the disease continued (and still continues) to propagate through the rest of Great Britain, slowing down owing to sub-optimal conditions and reduced host density, and returning to previously infected areas following elm regeneration. In biological terms, the epidemic follows a classic disease spread trajectory: building slowly during an extended lag phase (our analysis suggests an earlier than previously estimated date of introduction of 1962—see below); before breaking out into an exponential phase of disease spread; then tailing off as the host population is locally eliminated. More than this, however, our integrated biological, human and institutional reconstruction suggests that the spread of the disease was accompanied by a series of threshold events and decisions that are just as important in understanding the overall trajectory of the outbreak.3

Our archival research and interviews suggest that the human response to the outbreak can be periodized into four key phases: Mounting Public Concern, Official Recognition, Attempted Management and Aftermath. This narrative begins with the extended period of Mounting Public Concern described above. At that stage, the disease is beginning to break out from the initial infection points and reports from the public comment on the rapidity with which the disease appears to be spreading and on the sudden death of large numbers of mature trees. By 1969, local authorities in southern England and the midlands are looking to the Forestry Commission to take action as the lead authority responsible for plant health in relation to trees; a case for central government action is beginning to be assembled. Convinced that this is merely another flare-up of an old problem, the Forestry Commission is initially sceptical of the need for costly intervention. Eventually, in late 1970, following further pressure from local authorities, the Commission concedes the case for a voluntary programme of sanitation felling. During this phase of Official Recognition, the Forestry Commission as the competent authority focuses on raising awareness among stakeholders (it convenes a meeting of national government departments and local authorities) and offering advice to individuals on how best to deal with the disease through its pamphlet ‘The Dying Elms’ [18]. While conceding that the outbreak is more serious than anything previously seen (the disease is by this stage widespread throughout southern England and the midlands), the record of meetings and written exchanges between plant pathologists and senior management in the Forestry Commission suggests continued reluctance to abandon the conventional view of DED as a manageable disease system in the UK [12].

Following further evidence of the much greater than expected pathogenicity of the outbreak and mounting public pressure for something to be done, the Forestry Commission decides that new legislative powers are needed to enable a more systematic site inspection and felling programme to be implemented. The summer of 1971 signals the beginning of what we label the 'Attempted Management' phase of the outbreak, commencing with negotiations between the Forestry Commission, MAFF and the Treasury about who should bear the anticipated costs of such a programme. A critical decision is taken that local authorities should be the enforcing authorities and would bear the burden of the costs that sanitation felling would entail. Under the Dutch Elm Disease Control Order (1971), county and borough councils
are empowered to enter land, inspect trees and to take action to ensure that diseased trees are felled. However, in the absence of any significant subvention of Treasury funds (just £250,000 was made available from central Government) and with limited personnel available to carry out the inspections, implementation is patchy and some local councils rule out DED control at this early stage on cost grounds alone. Controversially, the Order is revoked on the grounds of its unworkability in late 1971. Nevertheless, confirmation that the current outbreak is due to a new and much more virulent form of the DED pathogen (O. novo-ulmi) in March 1972 [14] is followed by publication of the Forestry Commission’s DED survey in September 1972 [15]. This confirms extensive elm death and triggers renewed concern within the Forestry Commission about the effectiveness of the Government’s response. A case is made for a regional cordon sanitaire across southern England to prevent further northward spread but is dismissed on grounds of cost. Further measures designed to contain the northward spread of the outbreak by controlling the extensive movements of diseased timber that were by this stage thought to be accelerating disease spread receive greater support. The DED (Restriction on Movement of Diseased Elm) Order 1974 comes into force in May 1974 designed to ban movements of timber unless it has been debarked and sprayed with insecticide. At the same time, the DED (Local Authorities) Order is re-invoked in order to enable councils in areas that are still disease free to undertake sanitation felling on a more preventative basis than before. None of these interventions is effective and by 1975 everyone involved accepts that the epidemic will inevitably run its course. The immediate legacy is a landscape of dead trees throughout the country and agencies like the Countryside Commission begin to agitate for a clear-up and replanting campaign. In this long Aftermath of the epidemic, there are expressions of professional regret and public loss but, as we have already observed, no official enquiry beyond a Parliamentary Ombudsman enquiry into the administrative effectiveness of the authorities involved [19]. This phase is arguably still in process today, given continuing concern and public debate about how best to protect the remaining small pockets of elm that have survived in places such as Brighton and Hove [12].

Given the still developing nature of plant health legislation and plant pathology research and low public awareness of biosecurity threats, it is unclear what sort of useful deductions scientists and policymakers at the time could have drawn from the experience. It is arguable, for instance, that the scientists’ reluctance to abandon the conventional view of DED as a containable threat to tree health made it easier for policymakers to avoid taking action early in the outbreak. The conventional wisdom enshrined in the work of Peace [13], conducted over many decades, appeared to be the final word on the subject of DED. The community of tree pathologists based at Alice Holt was slow to question this paradigm in the face of conflicting evidence. As Clive Brasier, a senior plant pathologist, acknowledged in interview with us ‘it was very difficult to avoid being influenced by Peace because he was very authoritative: he was a fine scientist and a tremendous observer’ (interview with Brasier [5]). While many of the people involved now regret not identifying the new, more aggressive pathogen sooner, assessing the risk posed by tree pathogens remains very difficult. The DED outbreak occurred on the cusp of new developments in plant pathology research and in the use of taxonomic tools, but the body of DED research on which scientists drew was essentially observational and not orientated towards systematic risk assessment for policy purposes. Policymakers too might have had occasion to reflect on institutional failures whereby agencies such as the Forestry Commission were not given the resources or administrative authority by government to take charge of the outbreak at an earlier stage. They might also have reflected on the ineffectiveness of legislative responses (the succession of DED orders that were the headline response to the outbreak) when these were not linked to any programme for implementation. The lack of Treasury commitment to centralized funding, confusion regarding liability for cost-sharing on the ground and the critical decision to devolve responsibility on to under-resourced local authorities all contributed to the poor management of the outbreak. Even so, would a better coordinated and resourced national DED strategy have actually made a difference? The paper now turns to consider some counterfactuals.

3. SOME DUTCH ELM DISEASE COUNTERFACTUALS

A central objective of our modelling work was to seek to answer this last question by reproducing the epidemic in spatial terms and then exploring whether short- and long-term outcomes would have been affected by earlier and more aggressive interventions compared with those actually followed. The model used [11] was a 1 km² resolution spatial application of the SEIS model [20], treating both the disease fungus (O. novo-ulmi) and the bark beetle vectors (Scolytus spp.) as a single pathogen. Spread between populations of infectious and uninfected, live and dead trees within each grid cell was driven by a radial dispersal kernel capturing both human and natural dispersal. Known introduction points and timber movements were explicitly simulated. Management was simulated with detection and reaction parameters on both a grid cell and local authority basis, drawing on both historical records and interviews with ongoing practitioners. Each local authority could shift policy from that of monitoring to intensive felling when infected trees were detected. In the simulation of actual management, a practical level of monitoring was maintained prior to the detection of the epidemic, and subsequent sanitation felling was maintained for a maximum of 3 years (by which time in most cases both live trees and funding were running out), but was only applied after official recognition in 1970. The counterfactual scenario assumed that funds and manpower were not limited, enabling instant and
indefinite sanitation felling following detection of diseased trees.

The extreme case is shown in figure 2–f, where intensive monitoring and sanitation felling are put in place prior to the fungus arriving in the country, shown relative to the simulated historical spread (figure 2a–c), for a single introduction point at Avonmouth. It shows that, even with significantly greater rates of removal of diseased or dead trees from infected areas than was actually achieved, the epidemic continues to spread, owing to the difficulty of detecting all infectious live trees, some of which may be largely asymptomatic.

Finally, the longer term simulations shown in figure 3 suggest that both these management scenarios and a no intervention policy converge to a similar eventual outcome, regardless of the timing or extent of intervention during the course of the outbreak. Possible restrictions on timber movements were also investigated. However, while these prevent the formation of new disease foci north of the Forestry Commission survey area, in particular slowing the northwards progress, local dispersal through beetle migration and unregulated firewood movement ensures that the epidemic stays ahead of any management, as shown in figure 2.

The rather bleak conclusion from this exercise is that the biological properties of DED—the acute pathogenicity of the fungus, its relationship with the elm bark beetle, the feedback between the presence of dead trees and the beetle population and the very wide dispersal of the fungus that was possible owing both to the movement of beetles and the transportation of diseased timber—mean that effective control was very unlikely once the disease had become established. The model indicates an introduction date as early as November 1962 (compared with previous estimates of late 1964–1966), long before DED was recognized as a new problem, let alone regarded seriously enough to justify government action. Port inspection and quarantining arrangements at the time were unlikely to have been sufficient to have prevented the entry of the disease into the UK. The moral for our times is that prevention is better than cure and in the 40 years since DED national plant health authorities have put in place a series of measures designed to identify biosecurity risks associated with different trades and pathways of entry, to improve port inspections and to prevent or regulate entry where a risk has been identified [21]. Precautionary action has thus become one of the watchwords of international biosecurity policy. Yet, while the UK was one of the early promoters of an internationally coordinated approach to biosecurity, its membership of the European Single Market, combined with a growing volume of agricultural and horticultural trade with other member states, has meant that biosecurity risks have increased in recent years [5]. The formation of the Single Market required the abolition of national barriers to entry in favour of a harmonized system of standards, risk assessment, plant passports and inspection regimes, but also placed the onus on the plant protection authorities to justify measures that would otherwise be regarded as restrictions on the free movement of goods and material. As MacLeod et al. [21] observe, there are inconsistencies in the EU’s Plant Health Directive and weaknesses in the plant passporting system that allow large trees, shrubs and other plant material to be transported across the EU and between very different biogeographical regions provided relatively minimal biosecurity standards have been met. Brasier [5], among others, criticizes both the
4. THE CONTINUING CONTEMPORARY RELEVANCE OF DUTCH ELM DISEASE

The result is that trees and woodland in the UK are arguably under greater threat today from invasive pathogens than ever before [3,4]. The sudden oak death pathogen, *P. ramorum* (*Pr*), is thought to have first arrived in the UK as a single introduction, probably on infected nursery stock originating from within the EU [22]. The pathogen itself is thought to have emerged in southeast Asia and entered international trade pathways on exotic horticultural plants of various sorts. It had previously been identified in the USA in 2001, where an epidemic of what would there be called ‘sudden oak death’ has infected millions of oak and tan oaks [23]. In 2002, it was recognized by the EU as one of the most significant quarantine pathogens within its jurisdiction [22]. A fungal pathogen with an ever-widening host range, the disease spreads via long-lived spores that are produced in large numbers and then disseminated through the natural environment via multiple pathways, including rain splash and wind-blown leaves, via watecourses, on the footware of countryside visitors and through animal movements. From an original outbreak in Cornwall in southwest England first identified in 2002, the disease has expanded throughout the southwest, into southern Wales and western Scotland. In parallel with DED, much of the long-distance spread has been driven by human-mediated movement of infected plants. In the UK, the pathogen was initially typically found in woodland wherever its principal British host species, *Rhododendron ponticum*, was present as an understorey. *Rhododendron* (and other shrubs, such as magnolias, where this is not present) is usually the first site of infection, before the disease spreads to susceptible trees such as beech, ash, sweet chestnut, evergreen oaks and Douglas fir. Critically, sporulation, allowing disease propagation, has until recently been restricted to shrubs.

Once identified as a risk to plant health, the UK authorities moved swiftly to put in place measures designed to contain the outbreak and prevent further imports of infected material. Annual surveys of nursery stock were initiated, with a policy of destroying all infected plants found within 2 km of where infections are discovered. Meanwhile, an Emergency Programme was established in southwest England to inspect woodland gardens and semi-managed or unmanaged woodland and to destroy areas of *R. ponticum* where infection is found [24]. In 2009, following a full science and policy review conducted in consultation with stakeholders [24], a new programme was established with increased funding with the goal of ‘reducing the innoculum to epidemiologically insignificant levels’ through a more extensive programme of *R. ponticum* clearance and better containment and eradication measures in infected gardens and nursery sites [25].

Despite this ambitious aim, plant health authorities face a considerable aim in containing a disease that is now established in the UK. In England and Wales, there had been over 900 reports of *Pr* by June 2009 [26], with infections increasing in number and geographical spread. Some 30 outbreaks of a symptomatically similar pathogen, *Phytophthora kernoviae* (*Pk*), have also been notified, chiefly in southwest England. For many species, including *R. ponticum*, *Pk* is a more aggressive pathogen. Containment of both *Pr* and *Pk* is difficult for a number of reasons. First, the disease system is biologically complex and hard to diagnose. While mortality rates for most trees are lower than for DED, infection periods are much longer and may be asymptomatic, suggesting that the pathogen can be present but undetected for long periods. Visually healthy plants in woodland gardens in the southwest, for instance, may be producing very large volumes of spores that continue to infect other plants. Equally, as Brasier [5] has pointed out, port inspections based purely on visual inspection may still be admitting infected material, particularly in the substrate of trees and ornamental plants. Second, and perhaps most worryingly, the host range is very wide and appears to be expanding.

In a manner strongly reminiscent of DED, the authorities are dealing with an epidemic with unpredictable characteristics (in this case, an expanding host range, as opposed to a new species). Hence, while initial infections were confined to ornamental shrubs such as *Rhododendron* and the stems of trees growing close to these foliar hosts, in 2009, the pathogen was discovered on *Vaccinium myrtillus*, a core species component of both the upland and lowland heathland habitats for which the UK is internationally renowned, and an understorey plant in many woodlands. Given the susceptibility of other ericaceous species, particularly *Calluna vulgaris*, and the sporulation potential of *V. myrtillus*, there is now a recognized threat to...
Figure 4. The changing face of *P. ramorum* infection in England and Wales, showing approximate locations of confirmed (2010) infections (red) on *L. kaempferi*, and suspected new infections (purple), mapped against the (2009) extent of ongoing semi-natural infections (green). *Larix kaempferi* infections occurring in previously infected areas are not mapped. The locations of four National Parks with a strong heathland component are marked to highlight the increased threat brought about by the new host. Sources: [29–32].

heathlands [11]. In summer 2009, the pathogen had further been confirmed on Japanese larch, *Larix kaempferi*, a commercial forestry tree, which is a sporulating host. Douglas fir (*Pseudotsuga menziesii*) and Western hemlock (*Tsuga heterophylla*) trees are also succumbing when in close proximity. While symptoms have not been observed in the field on *Larix decidua*, it has tested positive in inoculation studies [27,28]. Aerial photography of plantations in the summer of 2010 has revealed potential infections, many of which have tested positive, across areas of the southwest of England and Wales previously believed to be free of infection (figure 4). Importantly, this step change in the epidemic exacerbates the threat to heathlands, given both their proximity to many plantations and the higher rates of spore production on *L. kaempferi* compared with other hosts. At the time of writing (September 2010), almost 2000 ha of forest had been placed under statutory notice [26].

The disease now straddles both the public and private domains, with potentially very serious implications for the UK’s horticultural heritage of woodland gardens, the biodiversity of its semi-natural environments (in terms of the implications for woodland and heathland) and commercial forestry interests (in terms of the potential impact on an important commercial forestry tree). While the initial institutional response may have been relatively adroit [25], the subsequent spread of the disease and its proliferating host range have severely tested the ability of the plant health authorities to respond quickly to a shifting risk profile. Indeed, the disease has arguably already passed over the threshold of being a containable tree disease. The grave threat to heathland species was identified as early as 2006 [33], for instance, yet the response to this extension of the host range has been slow. Meanwhile, the management response in woodland gardens, now seen as key sites of infection and sources of spread, has been patchy, with some owners and managers resisting attempts to remove infected specimens and contain infected sites [25]. A modelling exercise designed by the authors to test the likely effectiveness of the new control programme [20] revised the spatial model of *Pr* spread to reflect improved understanding of the sporulation of *V. myrtillus* and the distributions of *V. myrtillus* and *R. ponticum*. We compared the outcomes of the existing policy approaches and the proposed new measures to control *R. ponticum* but concluded that the continuing absence of measures to contain the heathland epidemic, together with the already wide geographical spread of the *R. ponticum* infection, means that the disease is unlikely to be contained at this stage in the outbreak. Given the recent increase in the known infected area in the UK owing to the widespread infection of Japanese larch reported above, disease spread into the wider environment has already reached the extent of spread predicted for *V. myrtillus* and *R. ponticum*. The area now threatened by *P. ramorum* alone is so great that even were effective management solutions available for all the landscapes at risk, implementation is likely to be prohibitively expensive. This is reflected by the recent move from the manual inspection of sites to the use of aerial photography with ground truthing.

5. DISCUSSION AND CONCLUSIONS

In a recent review of the biosecurity threat to native plant communities and woodland in the UK, MacLeod *et al.* [21] comment on the lack of public debate and advocacy on the issue, particularly when compared with the dominant narrative of climate change. This state of affairs is curious given that tree disease epidemics are known to have profound consequences for biodiversity, landscapes and even ecosystem function. Moreover, past epidemics have been widely documented and their landscape and biodiversity consequences in countries like the UK are often the most widely experienced cases of sudden and irreversible environmental change in living memory. With an apparent proliferation of the threat from invasive pathogens owing particularly to the growing volume and changing technologies of international trade and the resulting opening up of new invasion pathways, we have argued that this historical experience can be a useful point of reference, both in demonstrating the possible implications of future epidemics and as a source of learning experiences for forest pathologists, plant health professionals and biosecurity policymakers. History is currently an under-used resource in public policy circles [34], but historically informed analysis can be helpful in shedding light on current problems and hence in lengthening institutional memory and public recall [35]. In seeking to reconstruct the 1970s DED outbreak in the UK, we acknowledge that the availability today of more powerful predictive tools like epidemiological modelling, together
with the hindsight available to the scientists we interviewed, mean that the lessons that can be drawn at this distance in time are different from those that were deduced (albeit without very great official interest) in the immediate aftermath of the epidemic. Posing ‘what if’ questions is still a valid exercise because it allows for a sensitivity testing of epidemic outcomes to present-day policy pathways and management options and thus for a read across to current tree disease threats and unfolding epidemics.

The conclusion from our DED analysis is that biology trumped policy at a very early stage in the outbreak. Policymakers underestimated the pathogenicity of the new DED pathogen (ironically because the scientists advising them were so heavily influenced by previous experience of a less virulent form of DED in the UK), and this arguably delayed and deflected the official response. Poor management and institutional failures followed, but our analysis suggests that eventual DED outcomes would have been little different, even with a more rapid, and better coordinated and resourced programme of sanitation felling. This is because, once established, the pathogenicity of the disease system was sufficiently acute to make containment impossible. Prevention, on the other hand, would have required action to restrict imports of diseased material at a very early date, based on a full risk assessment of the new disease system as it had been developing in North America during the early 1960s. This is an even less likely scenario, given levels of awareness of biosecurity threats, the still developing nature of international biosecurity procedures, protocols and information sharing and the nascent status of precautionary action as a policy principle. Yet, the cardinal need to take preventative action, even to the extent of a much greater regulation of the international trade in plants and plant products, is the key lesson to be drawn from DED. The subsequent international evolution of protective biosecurity measures, designed to prevent introductions as well as establishment [4], goes some, though crucially not all, of the way towards this goal by establishing the precautionary principle in national and international biosecurity policy.

The story of the current outbreak of Pr andPk in the UK suggests that effective prevention is nevertheless still very difficult to achieve, particularly in relation to the weakly regulated horticultural trade in the context of an EU Single Market. Now established in the UK owing to a recognized (and easily anticipated) breach of biosecurity within the European nursery trade, the biosecurity challenge currently confronting policymakers is illustrative of deeper weaknesses in the system. The outbreak can already be seen as a biosecurity failure to the extent that the disease was able to enter the UK. It follows a series of warnings from experts about the laxity of port inspection and quarantining procedures within the European Single Market [5]. Despite important biological differences with DED, there are growing parallels between the two outbreaks in terms of the subsequent difficulties of attempted management post-establishment, and possibly even eventual outcomes. Whereas DED was beyond effective management because of its biological ability to spread very rapidly across a given host range and thus kill many trees of the same genus, the Pr andPk disease system is proving equally uncontainable, despite its slower spread, owing to a capacity to extend across an expanding host range. Present-day policymakers have had access to a much more quickly assembled set of susceptibility studies about the disease, yet have been similarly unable to contain the resulting proliferation of infection sites and disease situations. With the disease now being reported in commercial forestry trees, as well as affecting important heathland habitats, Pr/Pk seems likely to join DED as one of the most serious tree disease epidemics in recent decades.

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ENDNOTES

1 The key institutional players in this narrative were the Forestry Commission, the government agency responsible for plant health in relation to trees and woodland under the 1967 Plant Health Act, the Ministry of Agriculture, Fisheries and Food (MAFF), the competent authority responsible for agricultural and horticultural material and the Department of the Environment (DOE), responsible for countryside protection and biodiversity conservation. Local authorities in England, Wales and Scotland would also have an important role following a decision to devolve responsibility for site inspections and sanitation felling to local councils. 2 Usually understood as a quarantine line, created by clear-felling trees within a designated zone on the leading edge of the epidemic in order to slow its spread via known vectors.

3 Tom Peace was a key figure in the early development of DED research. Peace was Chief Research Officer at Alice Holt in the early 1960s and drew on extensive observational fieldwork of earlier (but less virulent) DED outbreaks in his influential monograph ‘The Status and Development of Elm Disease in Britain’. This was the first point of reference for the Forestry Commission’s pathologists struggling to make sense of the new outbreak.

4 The Department for Food, Environment and Rural Affairs (Defra—the successor ministry to MAFF) and the Forestry Commission (under its Plant Health Service) share responsibility for managing the disease in England, while the Scottish Government and the Welsh Assembly Government share responsibility with the Forestry Commission in their respective jurisdictions.

REFERENCES


1847 C. Potter et al. Learning from history


