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Dutch Elm Disease and the Future of the Elm in the U.K.: A Quantitative Analysis

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Dutch elm disease and the future of the elm in the U.K.: a quantitative analysis

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SUMMARY

We consider the future of the elm in the United Kingdom by analysing a simple model for the spread of Dutch elm disease. We show that this model, using biologically plausible parameterizations, is capable of mirroring observed data over short timescales such as a decade, but also demonstrates, over century-long timescales, a wide variety of outcomes ranging from pathogen extinction to substantial elm loss.

This complexity of outcome from a simple model suggests that a greatly improved understanding of the basic population dynamics of elm and of the epidemiology of the causal fungus is necessary to analyse the future of the elm, the effect of spatial or genetic heterogeneity, or the impact of human interventions.

We also use the model to discuss briefly the qualitative evolutionary impact of the combined saprophytic and parasitic stages of the fungal lifecycle.

1. INTRODUCTION

Questions about the long term future of the elm and its pathogens are not new: in 1862 a *Times* editorial noted the sickly state of park elms and recorded the prediction by a 'prophet of dendrology that elms will be extinct in England before another century has elapsed' (Anonymous 1862; Rackham 1986). A century later, the elm was surviving as a population of tens of millions of mature trees: we may never know the exact nature of the disease that threatened them. But by the end of the 1960s (Gibbs & Brasier 1973) came the first British cases of a Dutch elm disease (DED) epidemic which was to kill most of the elm in southern England, most notably the English elm *Ulmus procera*. Although the trees were killed, many root systems survived, and large numbers of suckers are now being produced and maturing into adults once more susceptible to infection (Greig 1994).

Many questions about the long term future of host and pathogen were raised in a scientific form over 15 years ago by Brasier & Gibbs (1978) and Brasier (1983*b*, 1987). Will elm populations recover? What are the evolutionary pressures on the elm, the pathogenic fungus, and the other organisms involved? We re-examine some of these questions using simple deterministic mathematical models and argue that useful and necessary insight can be found with the aid of these methods, which have proved extremely useful in medical epidemiology (Anderson & May 1991; Scott & Smith 1994), but which have as yet found limited use in botanical epidemiology (for example, Brassett & Gilligan 1988; Chan & Jeger 1994; Gilligan 1994, 1995). Previous epidemiological analyses of the Dutch elm epidemic (Gibbs 1978*a*, *b*) have collated data on the temporal spread of disease in the form of Vanderplank's *r* (Vanderplank 1963) which is a useful comparative tool but, as we have argued elsewhere (Swinton & Anderson 1995), is difficult to interpret in the context of a situation with regrowth of hosts after infection. The approach we take here is close to that of Anderson & May (1986) who were particularly interested in the competition between different pathogen species. Another quantitative approach was outlined by Ipiniza Carmona & Gil Sánchez (1987) who constructed both a catastrophe theoretic model and a compartmental one for the spread of the disease in Spain.

In this paper we begin the task of applying quantitative methods to the situation in England. We concentrate on this region because of the relatively good quality of epidemiological data available, and the knowledge of transmission biology accumulated, thanks to the authoritative work of T. Peace, J. Gibbs, C. Brasier, J. Webber and their colleagues at the Forestry Commission (Gibbs *et al.* 1994).

2. EPIDEMIOLOGY OF DUTCH ELM DISEASE

The biology of Dutch elm disease has been intensively studied and this is not the place for a full review. We refer the reader to the papers of Gibbs (1978*a*, *b*) for more information and references on the national and international epidemiology of the disease before 1978, and Brasier (1986) on the population

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biology of the fungus. Webber & Brasier (1984) describe its transmission biology, and the conference proceedings edited by Sticklen & Sherald (1993) report on research at the cellular and molecular level. Comprehensive bibliographies have been compiled by Laut *et al.* (1979) and Chapman (1990). Here we give a necessarily abbreviated outline of those features of the disease particularly salient from an epidemiologist's point of view. Richens (1983) describes the known population biology of the elm itself in more detail.

There have been two pandemics of Dutch elm disease this century, each with substantial impact on elm populations (Gibbs 1978*b*; Brasier 1990). It is thought that the first, caused by the fungus *Ophiostoma ulmi* (Buism.) Nannf, appeared in northwest Europe before 1920, and reached Britain in the next decade. In Europe, although not North America, both the number of affected trees and the severity of symptoms appeared to decline from the late 1930s (Peace 1960). The second, more catastrophic pandemic, which is believed to have originated in the 1940s and reached Britain by 1970, is caused by the more aggressive pathogen, now recognized as a separate species, *Ophiostoma novo-ulmi* Brasier (Brasier 1991).

The lifecycle of the fungus is relatively well known, although with a number of significant lacunae (Webber & Brasier 1984). When the xylem of a healthy tree is infected, a severe wilt ensues. The bark of the dead or dying tree provides breeding material for the bark beetle (usually *Scolytus scolytus* in the U.K.) which acts as a vector. Fungus is introduced into the bark, either by spore-carrying beetles or by transmission from the xylem. It remains unclear which of these routes is the most significant (Webber & Brasier 1984). Growth of the fungus in the bark leads to the production of spores in the pupal chambers and emerging beetles carry these spores. Infested beetles feed on healthy trees and xylem infection may occur via feeding wounds. Once dead trees are no longer 'fresh' (Peace 1960), they become unsuitable for beetle breeding. Transmission is also known to occur through the root grafts (Verrall & Graham 1935) and common root systems of elms (Peace 1960).

There is evidence for a viral-like infection of the fungus which may be responsible for a decrease in disease pathogenicity (Brasier 1983*a*). It has been speculated that this 'd-factor' played a role in tbe decline of the first pandemic, and it has been proposed as a mechanism for biological control (Webber 1993).

3. METHODS

(*a*) *Model structure*

In this paper we consider national prevalence data, and so we begin by representing the healthy elm population by X, the number of individual non-woodland trees of all elm species present in our unit area, which we take to be the maximal survey area defined by the Forestry Commission in their 1972 Survey discussed below.

These healthy trees die, due either to 'natural' (i.e. non DED) mortality or to xylem DED infection. There is no transmission from the xylem into the bark (in this model) so this mortality produces a number, Y, of dead trees with bark suitable for beetle breeding, but as yet uncolonized by fungus. We are particularly interested in the competition between the two species of fungus, *O. ulmi* and *O. novo-ulmi*, and we include a representation of each in the model. The two fungal species compete to infect the beetle breeding grounds; the numbers of dead elm infected by the non-aggressive and aggressive species are Z_u and Z_{nu} respectively. Dead elm bark, whether infected or not, soon becomes unsuitable for further beetle breeding. Note that X, Y, Z_u, and Z_{nu} all have dimensions 'number of trees per unit area'. It is perhaps worth emphasising the slightly unusual structure of this model: only those trees which have already died can be bark-infected, although the presence of the pathogen does contribute to xylem infection (not directly counted in the model) which in turn causes additional mortality.

To describe the population dynamics of this model we need to describe how transitions between each of these compartments occur. A schematic outline of the model is shown in figure 1.

For the population biology of the elm we make the simple assumption that non-DED mortality occurs at a constant rate μ. We assume that regeneration occurs only when the removal of an adult has taken place, modelled by a density dependent *per capita* growth term $r(1-X/K)$. Here K is a representation of the carrying capacity of the region. On the basis that young trees, although susceptible to infection, are too small to provide breeding grounds for the beetle vector (Greig 1994), we introduce a sapling class W of juvenile trees of this type. We assume that trees leave this class at a rate m. Once adult, the individual risk of xylem infection is given by the force of infection λ_x. Trees die, either due to such infection or to other causes of mortality, and enter the Y class when they are suitable for colonization by beetles and become colonized by beetles carrying the non-aggressive species and the aggressive species at the rates λ_u and λ_{nu}, respectively. All trees of types Y, Z_u and Z_{nu} are removed from the system, by becoming unsuitable for beetle breeding, at a rate γ. Note that competition between the two fungal species is entirely in the competition for breeding material between beetles carrying each species. Finally we need to define the transmission processes by defining the various forces of infection λ.

(*b*) *Xylem infection*

Infection and death of healthy trees, due to each fungal species, is taken to occur at a rate proportional to the density of vectors present, V, and the fraction of those vectors carrying each fungal species:

$$\lambda_x = \phi_u V \frac{Z_u}{Y+Z_u+Z_{nu}} + \phi_{nu} V \frac{Z_{nu}}{Y+Z_u+Z_{nu}}. \qquad (1)$$

But now we assume that the vector density is directly controlled by the density of breeding material: $V \propto Y+Z_u+Z_{nu}$. Some support for this assumption comes from the opinion of Peace (1960) that 'it is almost certain, on circumstantial evidence', that a reduction

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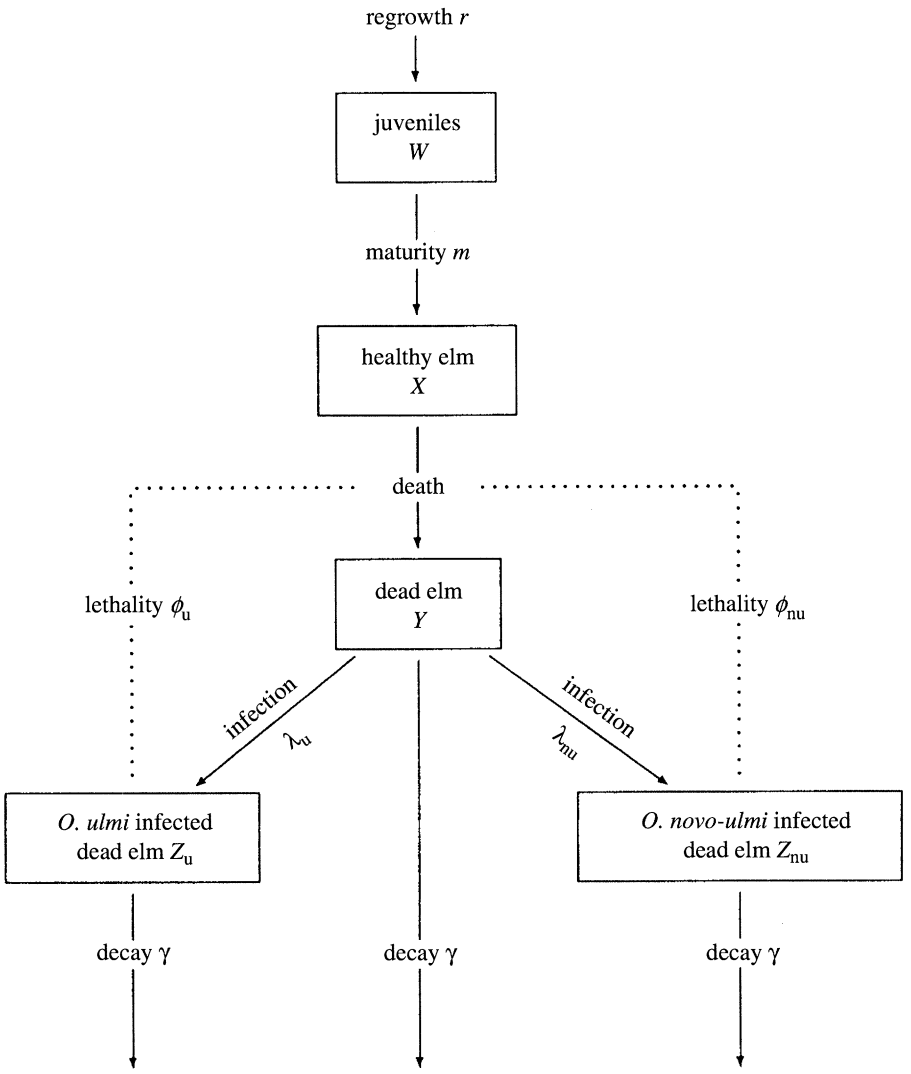

Figure 1. Flow diagram of Dutch elm disease model. Flow of elm through the different compartments indicated by solid lines, influence of pathogen partly indicated by dotted lines.

in the number of infected trees fell in concert with the number of beetles. So, after a rescaling of the parameters ϕ, we have simply,

$$\lambda_x = \phi_u Z_u + \phi_{nu} Z_{nu}, \quad (2)$$

where ϕ represents the lethality of each species.

(*c*) *Saprophytic colonization*

The rate at which bark is colonized by a species of fungus is the sum of the rate at which new individual trees are colonized and the rate at which growth occurs through existing ones. For the non-aggressive species we have:

$$\lambda_u = \beta_u V[Z_u/(Y+Z_u+Z_{nu})] + r_u Z_u, \quad (3)$$

where β represents the rate at which beetles infect the bark of new trees and r_u the rate at which the fungus spreads within an existing tree or root system. Conceptually, this conflates two different kinds of infection events: infection of a new tree, which might be thought of as a discrete, all-or-nothing event; and spread of infection within a tree, a more continuous process. However, the deterministic differential equation formalism allows us to represent both processes simultaneously: under this mass-action approximation two dead trees acting as breeding sites which are 50% infected are the same as one breeding site which is 100% infected. More practically, it is not possible to distinguish these two processes from the aggregate epidemiological data: indeed, with the same assumption as before that beetle densities are directly proportional to breeding material we can simply write (again after a recaling of β),

$$\lambda_u = \beta_u Z_u, \quad (4)$$

with a similar expression for the aggressive species. Note that the rate at which the fungus spreads within a tree is now subsumed by the rescaling into the aggregate transmission parameter β.

With these assumptions about transmission biology, the equations of the model are now:

$$\dot{W} = rX(1-X/K) - mW \quad (5)$$

$$\dot{X} = mW - \mu X - (\phi_u Z_u + \phi_{nu} Z_{nu}) X \quad (6)$$

Table 1. *Parameter meanings and values used in the simulations*

parameter	meaning	typical value
r	intrinsic elm replacement rate	0.05 a^{-1}*
K	carrying capacity	15×10^6
m	rate at which saplings become large enough for bark beetle colonisation	0.1 a^{-1}
μ	mortality rate of non DED infected elm	0.01 a^{-1}
ϕ_u	lethality of non-aggressive species	variable
ϕ_{nu}	lethality of aggressive species	variable
β_u	transmissibility of non-aggressive species	variable
β_{nu}	transmissibility of aggressive species	variable
γ	rate at which bark becomes unsuitable for beetle breeding	1.0 a^{-1}

* a^{-1} = year^{-1}.

Table 2. *Summary of variable meanings*

variable	meaning
W	sapling elms too small to allow beetle breeding
X	healthy adult elms
Y	xylem infected and other dead and dying elms
Z_u	dead elms colonized by *O. ulmi*
Z_{nu}	dead elms colonized by *O. novo-ulmi*

$$\dot{Y} = (\phi_u Z_u + \phi_{nu} Z_{nu} + \mu) X - (\beta_u Z_u + \beta_{nu} Z_{nu}) Y - \gamma Y \quad (7)$$

$$\dot{Z}_u = \beta_u Z_u Y - \gamma Z_u \quad (8)$$

$$\dot{Z}_{nu} = \beta_{nu} Z_{nu} Y - \gamma Z_{nu}. \quad (9)$$

(*d*) *Model parameters*

The parameters used in the model were described in the previous section. Here we record the numerical values which were used in the simulation runs, and the rationale for their choice.

The intrinsic elm regeneration rate r is taken (arbitrarily, in the absence of published data to our knowledge) to be 0.05, corresponding to a typical replacement period of 20 years in the absence of other mature elm. The carrying capacity is taken to be $K = 15 \times 10^6$. This is so that, combined with the estimates for r and μ the model yields a preinfection count of healthy trees equal to 12×10^6, chosen for consistency with the estimate made by Gibbs & Howell (1974) of the number of U.K. non-woodland elm before the last major epidemic.

The rate m at which seedlings or root suckers mature into trees large enough to be colonized by bark beetles is taken to be 0.1 per year. Greig (1994) stated that after regeneration beginning in 1977, 'any suckers killed in the 1980s were generally too small', so we take a mean maturation period of 10 years. We take the mean lifespan of an elm in the absence of DED infection to be 100 years, so that $\mu = 0.01$ per year. We assume that breeding material becomes unusable after approximately one year and so we take $\gamma = 1.0$ per year. These values are summarized in table 1, and the variables used are given in table 2.

4. DATA

We take data on the prevalence of infection in the U.K. from the Forestry Commission's extensive surveys which took place each year from 1971 to 1978 with the exception of 1977. In a previous epidemiological analysis by Gibbs (1978*a*), consistency was improved by restricting those data used to only those sites actually surveyed every year. However this analysis was carried out before the results of the 1978 survey were available. This is an important survey year because it is the first to show evidence that the rate of spread of the epidemic is slowing down (as predicted by epidemiological theory), and thus to allow conclusions to be drawn about the eventual level of infection. Accordingly, we reanalysed the data using additional information published elsewhere (Gibbs & Howell 1972, 1974; Gibbs 1975, 1977*b*, 1979) in a different form. The scale of the survey and the detail of the published data changed from year to year. To minimize sampling uncertainty, we restrict ourselves here to the dataset on non-woodland (i.e. hedgerow and urban) elm classified into healthy, dying and recently dead, or long-dead trees. These data are shown in table 3, and we refer to it as the 'maximal region' survey dataset as it uses information from as many survey plots as possible, and extrapolates to give estimates of the total number of trees within the survey area. Note the large discrepancy between years 1971 and 1972; the later survey was considered by Gibbs to be more reliable. By contrast, the 'minimal region' dataset given by Gibbs (1978*a*) is more consistent in that it is derived only from sites which were surveyed every year between 1971 and 1977, but covers a smaller region, gives absolute numbers of trees surveyed, and does not include information on 1978. Only elms over 6 m tall were recorded in the survey, and so we attempt to compare these data with the model outputs for the non-juvenile classes.

5. RESULTS

(*a*) *Model runs*

Given the model definition and initial parameter estimates, numerical simulations were carried out using the packages Stella (High Performance Systems Inc.) and Mathematica (Wolfram Research Inc.). To make baseline estimates of the key transmission parameters we took the element of published data most closely related to our model variables, the number of healthy trees, and used this to calibrate the model through a least squares fit, shown in figure 2.

Although there is a good visual fit with this model, problems remain. It is clear that the best fit to one variable alone significantly underestimates another. Interpretation is complicated by the difficulty of comparing the aggregate Forestry Commission classifiction for dead and dying trees with the '$Y + Z_u + Z_{nu}$' compartments and this, together with our neglect of

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Table 3. *Forestry Commission survey data*

(Published data from 1974 onwards aggregates classes D and E. Millions of non-woodland elm; n.d. denotes data not reported.)

year		weak infection	strong infection		recently dead	long dead	
FC class	total	C	D	D+E	E	F	source
1971	10.81	0.99	0.31		0.19	0.14	(Gibbs & Howell 1972)
1972	14.17	1.10	0.74		0.50	0.34	(Gibbs & Howell 1974)
1973	14.10	1.24	0.99		0.86	0.62	(Gibbs & Howell 1974)
1974	13.25	1.41		1.90		1.25	(Gibbs 1975)
1975	12.68	1.08		3.22		1.66	(Gibbs 1976)
1976	11.83	1.40		2.66		3.22	(Gibbs 1977*a*)
1977	no survey						
1978	10.0	n.d.		2.4		4.3	(Gibbs 1979)

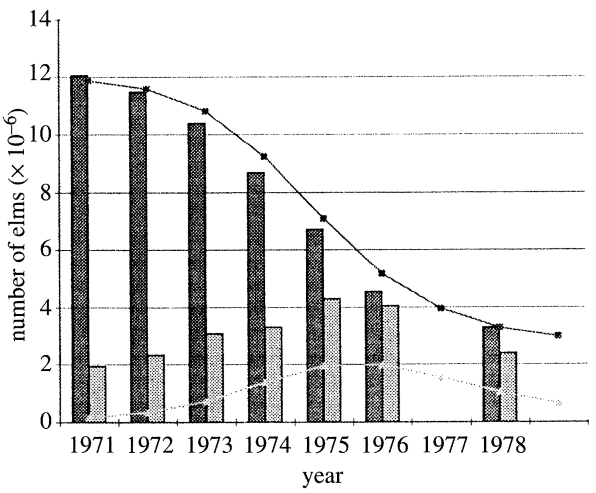

Figure 2. Numbers of healthy or weakly infected elm, and numbers of severely infected, dying or dead elm as given in table 3 and a least squares best fit model output using parameter values in table 1 and allowing β, ϕ, K and the time of introduction to vary, while keeping introductory level of $Z_{nu} = 1$. Nonaggressive species not present ($Z_u = 0$). Fit shown: $\beta = 24.406\ a^{-1} \times 10^{-6}$, $\phi = 0.164573\ a^{-1} \times 10^{-6}$, $K = 14.9414 \times 10^6$, introduction time 1964.94. Because of survey problems in 1971 discussed in the text, this year was not used as a data point in computing the fit. For comparison, the data point shown for 1971 has been scaled so total number of trees estimated in all categories is the same as for 1972. Dark-shaded area: maximal region survey data healthy trees; light-shaded area: maximal region survey data infected, dead and dying trees; dark line: model fit healthy trees; faint line: model fit dead and dying trees.

mortality due to the non-aggressive species may well account for a systematic underrepresentation, but nevertheless the trend is well mirrored by the model. However, there are a number of uncertainties in the parameter estimates, notably in the assumptions about elm demography, and until these estimates can be improved there seems little point in attempting to improve the fit of the model through adjustments in an ever higher dimensional parameter space. The principal value of this exercise is to give order of magnitude estimates for the key transmission parameters. Armed with the knowledge that the model predicts the basic qualitative behaviour of the epidemic in the short term, but is not yet capable of fine quantitative calibration, we turn to the long term predictions of the model and explore how these depend on the most uncertain parameters.

Figures 3, 4 and 5 show some typical model outputs over the timescale of a century, for nine different combinations of low, medium and high values of β and ϕ. In the simulation of this deterministic continuous model at certain parameter values, some population sizes may become extremely small, in particular the number of infecteds in the trough after an epidemic. The relation between the size of this trough and the probability of fade-out remains unclear (Nåsell 1995); for illustration we set $Z_{nu} = 1$ as a logical cutoff at which extinction might occur and record in the legend the years at which this happens. The simulations in figure 3 are carried out for the aggressive species present alone ($Z_u = 0$). Note that the low transmission cases all correspond to parameter values for which we predict that small levels of initial infection cannot persist (the basic reproductive number R_0 defined in the next section is less than unity). In the low lethality, low transmission case of figure 3, the number of healthy trees is indeed barely affected, but in the low transmission cases of figures 4 and 5 there are long transients before infection is eventually removed. Paradoxically, it is in the least pathogenic case figure 3, in which the infection persists for longest, because it does not provoke a trough of infection within which it can be rapidly extinguished. The intermediate lethality cases of figure 4 are those most closely mimicking the observed timescale of U.K. elm loss, but show considerable disparity in long term outcome: low transmission leads to replacement of elm losses; the medium transmission case appears to behave in the same way for more than half a century until infection reappears in a cyclic manner; while the high transmission case relatively quickly settles down to an endemic level of infection in which the healthy elm population is maintained at around a third of its pre-disease level. Note that although the resurgence in the medium transmission case occurs more than 40 years after the original introduction, the level of infection has not fallen below the, admittedly arbitrary, cutoff in the meantime. The final trio of runs, corresponding to higher values for the lethality parameter ϕ, display

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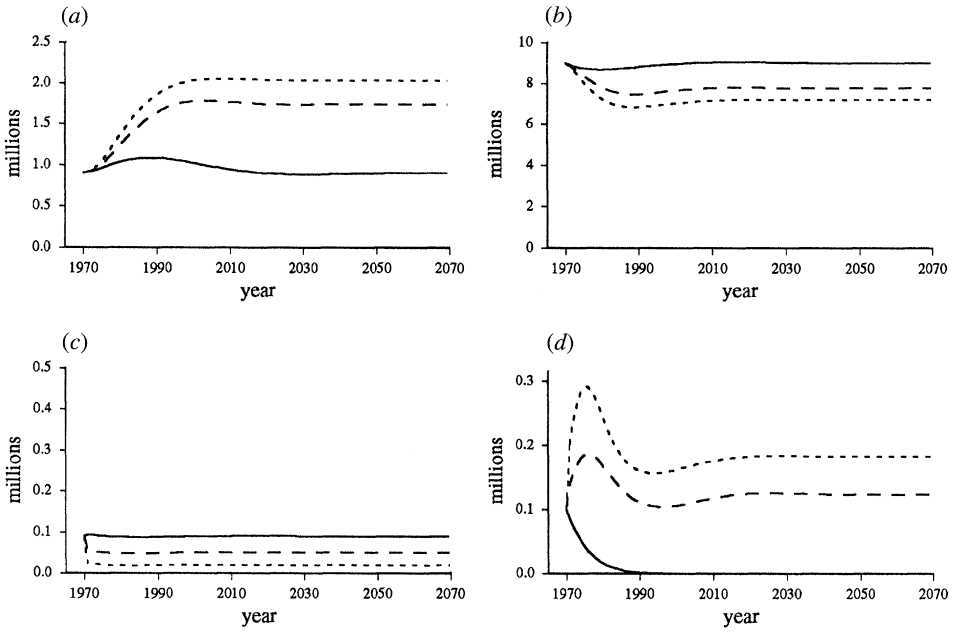

Figure 3. Long term model outcomes for different transmission and lethality rates. (*a*) Saplings; (*b*) healthy trees; (*c*) dead trees; (*d*) infected trees. Parameter values $r = 0.1\ a^{-1} \times 10^{-6}$, $\mu = 0.01\ a^{-1}$, $K = 10 \times 10^6$, $\gamma = 1\ a^{-1}$, initial infection 0.1×10^{-6}, introduction time 1970.0. Runs were carried out without truncation for small numbers; if infection was assumed to be extinguished when infected elm populations fell below 1, then extinction of the pathogen would occur in the low transmission case at time 2027. Non-aggressive species not present. This figure shows outcomes at a low lethality rate ($\phi = 0.1\ a^{-1} \times 10^{-6}$) and at low ($\beta = 9.0\ a^{-1} \times 10^{-6}$, solid line), medium ($\beta = 20.0\ a^{-1} \times 10^{-6}$, dashed line), and high ($\beta = 50.0\ a^{-1} \times 10^{-6}$, dotted line) transmission rates.

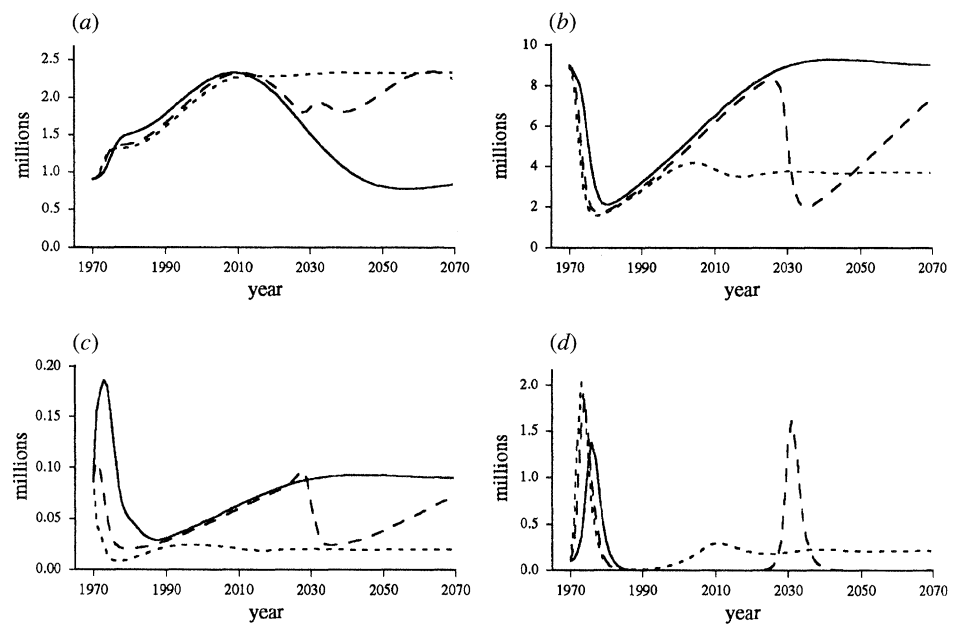

Figure 4. Long term model outcomes in the medium lethality case, for low (solid line), medium (dashed line) and high (dotted line) transmission. (*a*) Saplings; (*b*) healthy trees; (*c*) dead trees; (*d*) infected trees. Parameters as figure 3 except that lethality $\phi = 0.25\ a^{-1} \times 10^{-6}$. Infected numbers fall below one in year 2001 in the low transmission case.

much faster elm eradication than has been observed in practice at a national scale, and all lead to rapid extinction of the pathogen population within the first decade. The small number effects this corresponds to in the untruncated models shown in the figure lead to very wide variations in the long term solutions, a demonstration of the sensitivity of the disease free state to perturbations. Even when extinction does not occur, the precise time at which infection reemerges is no doubt sensitive to the extremely small levels of infection attained at the bottom of the imediate post-epidemic trough: more work needs to be done on understanding the dynamics of this system when local extinction is important, along the lines of recent work on the spatial dynamics of infections such as measles (Grenfell *et al.* 1994; Rand *et al.* 1995).

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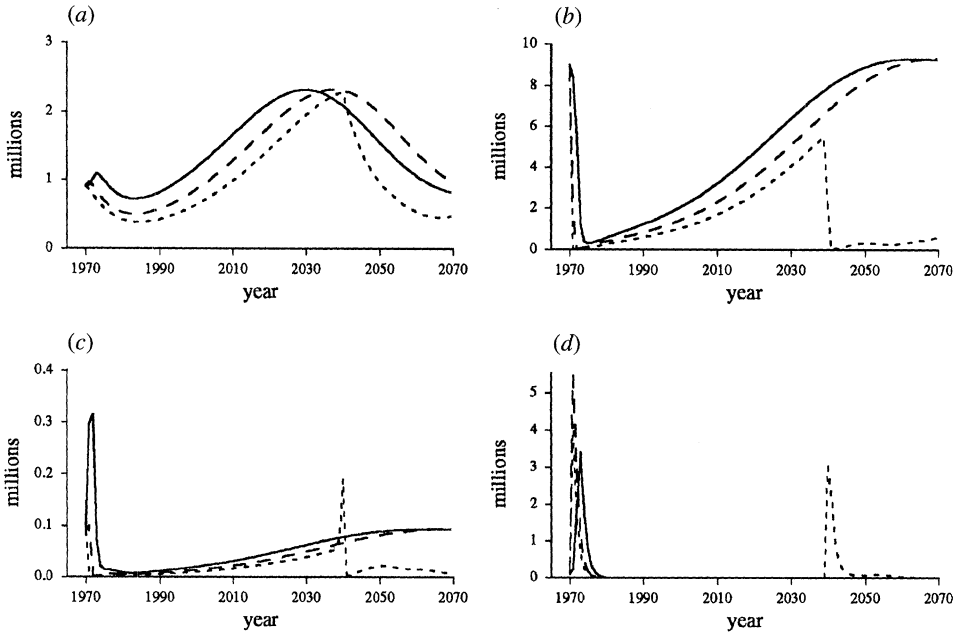

Figure 5. Long term model outcomes in the high lethality case, for low (solid line), medium (dashed line) and high (dotted line) transmission. (*a*) Saplings; (*b*) healthy trees; (*c*) dead trees; (*d*) infected trees. Parameter as figure 3 except that lethality $\phi = 1.0\ a^{-1} \times 10^{-6}$ (low and medium transmission) and $\phi = 4.0\ a^{-1} \times 10^{-6}$ (high transmission). Infected numbers fall below one in year 1989, 1989 and 1991 in each respective case.

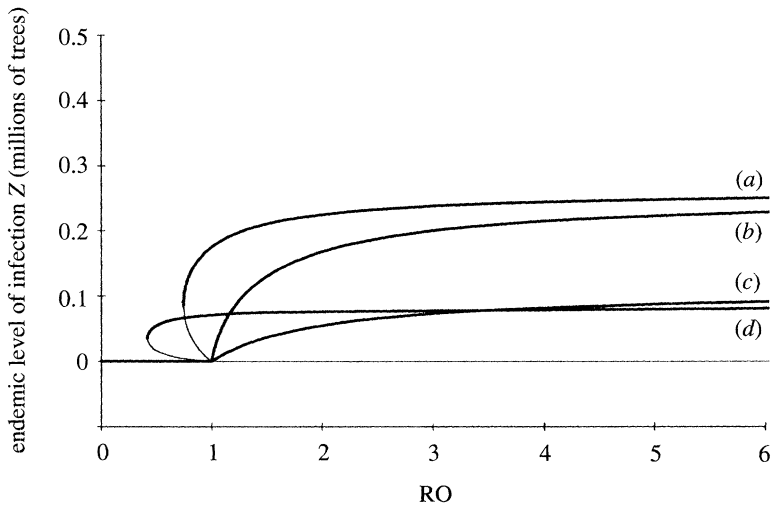

Figure 6. Graph of endemic level of infection (Z_{nu}) as a function of the basic reproduction number R_0 computed from equation (11) in the text with parameter values $\gamma = 1\ a^{-1}$, $\mu = 0.01\ a^{-1}$, $r = 0.1\ a^{-1}$, $K = 11.11 \times 10^6$, and with varying lethality parameter $\phi_{nu} = \phi\ a^{-1} \times 10^{-6}$. (*a*) $\phi = 0.2$; (*b*) $\phi = 0.1$; (*c*) $\phi = 0.01$; (*d*) $\phi = 1.0$. Solid lines represent stable levels of endemic infection, thin lines represent levels of endemic infection unstable to small perturbations.

(*b*) *Model properties*

The model described in eq (5)–(9) has an analytical structure which is of some interest to the interpretation of our results. Consider first the system with only one species present, for example the aggressive species *O. novo-ulmi*. Then we may define, either heuristically or analytically, a basic reproductive number R_0, which is given by

$$R_{0nu} = (\beta_{nu}/\gamma)\, Y^\circ, \qquad (10)$$

where $X^\circ = K(1-\mu/r)$ and $Y^\circ = (\mu/\gamma)\, X^\circ$ are the numbers of healthy and dying trees in the absence of any Dutch elm infestation. (A heuristic definition would observe that one colonized tree would create new colonizations at a rate $\beta_{nu}\, Y^\circ$ and persist for a time $1/\gamma$, whereas an analytical definition would consider the stability properties of the evolution operator associated with eq (5)–(9) linearized at the disease-free equilibrium.)

Note that this basic reproductive number is independent of the pathogenicity ϕ. Setting the model

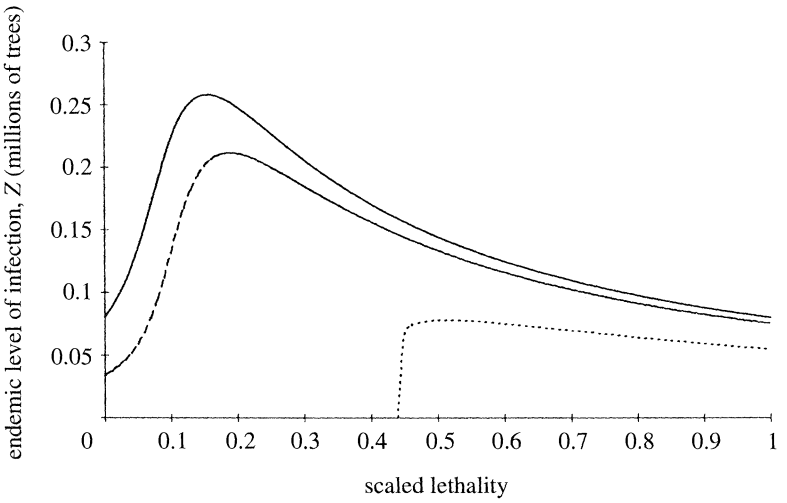

Figure 7. Graph of endemic level of infection Z_{nu} as a function of the lethality parameter $\phi_{nu} = \phi\ a^{-1} \times 10^{-6}$, computed from equation (11) in the text with other parameter values as in figure 6 and with $R_0 = 5$ (solid line), 1.5 (dashed line) and 0.5 (dotted line).

equations to zero, to correspond to an equilibrium, and eliminating variables, allows us to express the level of endemic infection Z_{nu} as a quadratic function of this basic reproductive number. Specifically, we find:

$$\phi_{nu}^2 Z_{nu}^2 - [\phi_{nu}(r-2\mu) - r\gamma/K]\, Z_{nu} - \mu(r-\mu)\,(1-1/R_0) = 0. \quad (11)$$

Given this function, we can plot how the level of endemic infection present depends on R_0 (figure 6).

For low levels of lethality (curve (*a*) in figure 6), there is a standard epidemiological threshold. The endemic level of infection (solid line) is seen to be zero when $R_0 < 1$: the infection cannot persist and the system settles down to a disease-free equilibrium which is stable in the sense that small amounts of incoming infection will not be magnified. When $R_0 > 1$, on the other hand, such inocula will be magnified over time, and so the disease free state is no longer stable. This is reflected in the existence of an endemic equilibrium at which host and pathogen coexist (although at lower pathogen levels than would be found at the peak of an epidemic): this can be shown to be stable for values of R_0 near to unity and is typically stable for all values of $R_0 > 1$. As the lethality parameter ϕ is increased, however, a more complex transition occurs as R_0 increases.

It is a consequence of the fungal lifecycle that infection acts to promote transmission by increasing beetle numbers. This means that even if the disease is incapable of invading a wholly healthy population, it might persist in an otherwise identical population which was already diseased. Thus we have an ecological Allee effect (Begon *et al.* 1986) in curves (*b*), (*c*) and (*d*) of figure 6.

In these situations, when $R_0 > 1$ the situation is broadly similar to what it was before. But when R_0 is closer to and less than unity, it is simultaneously possible for the infection to persist in the population or to go extinct. The outcome in a particular case would depend on the level of infection present initially.

A related question is how the level of pathogenicity affects the level of endemic infection. The same equation for the level of endemic infection (11) can be used to plot it against lethality ϕ, keeping R_0 constant. Figure 7 shows that a fungus which is of intermediate lethality will maximize its prevalence level within the population compared to one of low or high lethality. If we accept this prevalence level as related to evolutionary fitness then we predict that the fungus will evolve to a state of intermediate virulence (Levin *et al.* 1982).

However this picture is further complicated by the interaction between the different species. It is simple to show that the model does not allow the two species to coexist at equilibrium. In this model each species is characterised by two key epidemiological parameters: R_0 (or equivalently transmission, β) and lethality ϕ. Stability analyses of the equilibrium states can be used to demonstrate that competitive exclusion (Bremermann & Thieme 1989) will always act to exclude the species with the lower R_0, whatever the relative lethalities. Thus the model predicts, as did the simpler model of Anderson & May (1986), the replacement of one species by the other (figure 8) as suggested by Brasier a decade ago (Brasier 1983*b*) and observed in the U.K., Europe and the U.S.A. (Houston 1985, 1991; Brasier 1988). Note that at these parameter values it does not support the hypothesis of Brasier (1983*b*) of a transient increase in the number of trees infected by the non-aggressive species.

In principle, a species which was aggressive enough at colonising beetle breeding grounds when they existed (high R_0 or high β) need not cause any additional mortality at all ($\phi = 0$) to healthy trees to outcompete a less transmissible but more pathogenic species. This has obvious, and welcome, implications for the design of biological control programmes. It would be straightforward to extend the model to include a 'd-factor' independently transmitted within one species, but the general principle is already clear: to be successful, such a factor must be highly transmissible but need not be

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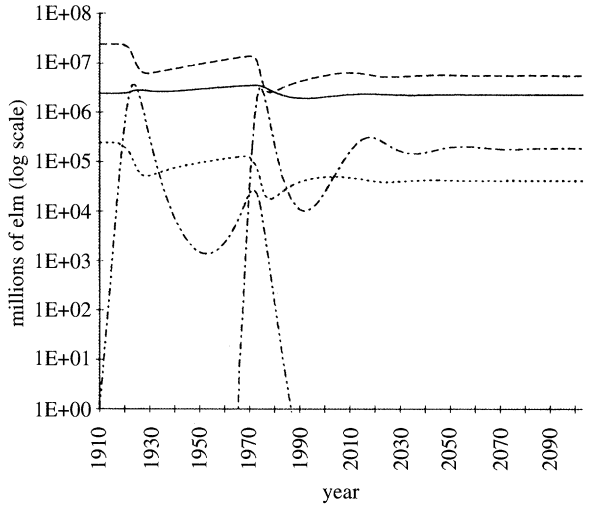

Figure 8. An example of competitive exclusion. Parameter values as figure 2 but with the non-aggressive species introduced in year 1915 with $\phi_u = 0.08\ a^{-1} \times 10^{-6}$ and $\beta_u = 10.0\ a^{-1} \times 10^{-6}$. Solid line = juveniles; dashed line = healthy adults; dotted line = dead trees; dash-dot line = *O. novo-ulmi* infected dead trees; dash-dot-dot-dash line = *O. ulmi* infected dead trees.

highly pathogenic, despite the importance of the pathogenic phase of the pathogen in determining endemic levels of infection.

One interesting scenario, albeit not one occurring in the biologically relevant parameter regions estimated above, is that two species might both have an $R_0 < 1$, but the one with the lower R_0 may have a lethality, ϕ, large enough to allow it to persist through the mechanism described above. If the second species had a lower lethality but a higher R_0 then it would be possible for it initially to displace the first species through competitive exclusion and then in turn go to extinction. The resulting state, with no infection present would then be stable against the introduction of small inocula of either species, and the introduction of one species would thus have caused the permanent extinction of both.

6. DISCUSSION

(*a*) *Epidemiological implications*

We have shown that a simple model is capable of mirroring the broad short term trends observed in the U.K. Dutch elm disease epidemic, including the exclusion of the less aggressive species by the more aggressive *O. novo-ulmi*. Even though we estimate this species to have a basic reproductive ratio greater than unity, we observe that, in some cases, extinction occurs due to stochastic fadeout in the trough following an epidemic. Even in the absence of such fadeout, long transients occur before the system reaches a range of possible equilibria.

We do not pretend that our simple model begins to include all of the important biological complexities of Dutch elm disease. Many different model structures and parameterizations would lead to predictions following the same qualitative structure, and it is not surprising that it is possible to find parameter combinations which mimic the limited observed data. What is important is that no extra mechanisms, such as evolution to reduced virulence or infection by a hyperparasite, are essential to explain these data. Moreover the simulations make it plain that even in the absence of these complications the long term quantitative future of the elm is not predictable on the basis of our current epidemiological knowledge.

However we believe that this situation can be substantially improved. Improved knowledge of the natural history of the elm and in particular its demographic properties would be extremely useful. Although much is known about the structure of elm populations and the behavioural differences between taxa, we are not aware of any published data on intrinsic growth and death rates to be expected, either on average or by taxa; and it is such data which are essential to the epidemiology of the infection, governed as it is in the long term by the supply of susceptibles. Also, the Forestry Commission surveys recorded prevalence at a relatively small spatial scale. Analysis of these existing data at such scales should allow much better estimation of the crucial transmission parameters. Data have also been recorded (Greig 1994) on the regeneration of plots after infection: these data are clearly crucial to the eradication question. This model's framework can also be tested against historical data on the temporal spread of other Dutch elm epidemics both in recent times (Went 1954; Peace 1960) and possibly against the highly time-resolved data now available from pollen record on the mid-Holocene elm decline (Peglar 1993; Peglar & Birks 1993), although the relation of this decline to Dutch elm disease is controversial (Rackham 1986; Brasier 1990).

Although more data are rarely unwelcome, more model complexity is a different matter. Although the list of potentially relevant biological complexities is long we single out spatial heterogeneity as a factor particularly likely to influence the results. The model assumes that an elm in Kent is as likely to receive infection from a tree in Somerset as one in the next field. Metapopulation theory suggests that this global mixing approximation is particularly likely to break down when the local dynamics consists of near extinctions followed by recovery, as in this case (Gilpin & Hanski 1991). With such a system, local stochasticity may promote fadeout of infection and contribute to the extinction of the pathogen. It will be possible to validate a model incorporating such effects by using the spatially explicit Forestry Commission data mentioned above. The very nature of threshold behaviour remains unclear in spatially extended systems in general, especially those found in botanical epidemiology and much work remains to be done in this area. Such a model would also aid the exploration of questions about the speed and nature of the spatial spread of infection from an initial focus: another type of question on which there exists theoretical insight (Zadoks & van den Bosch 1994) and empirical data (Gibbs & Howell 1974). A further complexity arises when we consider the diversity of elm genetics. Species identification in elm populations is hard (Richens

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1983), but a broad distinction can be made in the U.K. between wych elm *Ulmus glabra* and types of smooth-leaved elm *U. minor* which includes English elm, *U. procera* (Gibbs *et al.* 1994). These elms differ in their spatial distribution (Gibbs & Howell 1972) and susceptibility to infection (Gibbs *et al.* 1994). The trees occur as both woodland and hedgerow and amenity trees; each of these functional classes is likely to have a different type of contact structure leading to a complex metapopulation. Fortunately, the Forestry Commission spatial data includes a classification of this kind which will enable us to infer a plausible patch structure.

Our results would be also affected if we were to change our assumptions about the fungal lifecycle. Many of our assumptions, for example that there is no transmission from the xylem (parasitic) phase to the saprotrophic phase, that all the species involved have no relevant genetic diversity, that the fungus only spreads through this vector and not by root systems, are demonstrably false (Webber & Gibbs 1989). In principle, all of these complexities could be included in the model. However in the absence of specific data to parameterize such effects the value of doing so would be limited as long as basic transmission parameters also have to be estimated from the same limited dataset. We have made broad assumptions about the demography of elm populations: in particular, assuming that regeneration occurs only when an adult has been removed is tantamount to assuming that the population is at an equilibrium level before the emergence of infection. Future research, based on pollen records and historical data, may yield more precise numerical trends but these trends are likely to be negligible in the short term dynamics of infection, while adding once more to the considerable uncertainty about long term effects.

The model is also sensitive to choices of scale and unit. Spatial scale is discussed above, but choosing the individual tree as the basic unit deserves some comment. This ignores the complexity arising from the usual means of reproduction of *Ulmus procera* through clonal root suckers, and also does not allow us to represent the differential ability of fungal species to colonize within a single tree. Nevertheless, the disadvantages of this are outweighed by the fact that the individual tree is a natural item for enumeration and so any theoretical predictions need to be based around this unit.

An interesting insight from the mathematical analysis of the basic reproductive ratio is that the ability of the pathogen to invade a wholly healthy tree population is apparently independent of the degree to which infection causes mortality, even though such mortality is in reality essential to provide enough breeding material to allow the vector population to grow. This apparent paradox derives from the fact that the invasion criterion deals with what happens when extremely small levels of infection are present, and then non disease mortality is the significant provider of breeding material. The interactions between two different species sharing these transmission factors are not yet fully understood and pose an interesting challenge to theoreticians.

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