Journal of Ecology 2004
92, 675–685

Demography of American chestnut populations: effects of a pathogen and a hyperparasite

ANITA L. DAVELOS and ANDREW M. JAROSZ

Departments of Plant Biology and Plant Pathology, and Ecology, Evolutionary Biology and Behaviour Programme, Michigan State University, East Lansing, MI 48824–1312, USA

Summary

1 Matrix models were used to evaluate the effect of chestnut blight infection on transition probabilities and population growth rates for American chestnuts. Disease-free, epidemic and recovering (i.e. pathogen infected with a double-stranded (ds) RNA hypovirus) populations were compared.

2 Population growth rates (λ) did not differ significantly over time or with disease status. However, predicted stable stage distributions differed between population types, with disease-free and recovering populations more similar to each other than either was to epidemic populations.

3 Survival had the highest proportional contribution to population growth rates as revealed by elasticity analyses. However, reductions in stasis of the largest trees contributed most to reductions in population growth rate when comparing diseased with disease-free populations using LTRE.

4 The presence of hypovirus reduces pathogen virulence, allowing individual American chestnut trees to increase in size. Where dsRNA has spread, chestnut populations in Michigan have attained population dynamics similar to those found in disease-free populations.

5 Matrix models and life table response experiments can be used to detect important pathogen-mediated changes in the dynamics of host populations.

Key-words: biological control, *Cryphonectria parasitica*, *Castanea dentata*, double-stranded RNA, host–pathogen interaction, hypovirus, matrix projection model, pathogen virulence, population size structure

Journal of Ecology (2004) **92**, 675–685

Introduction

Chestnut blight is a classic example of how introduced pathogens can alter host population biology and overall plant community dynamics. The blight pathogen, *Cryphonectria parasitica* (Murrill) Barr, was introduced into the United States from Japan (Milgroom 1995; Milgroom *et al*. 1996) around 1904 (Merkel 1905) and rapidly spread throughout the range of the American chestnut, *Castanea dentata* (Marsh.) Borkh. Blight-infected branches are killed when a canker girdles the stem disrupting phloem transport and cambrial growth. As the pathogen cannot enter the root system, genets survive and new sprouts are produced from the root collar. The epidemic is perpetuated when the sprouts become infected. An intracellular hyperparasite of *C. parasitica* can alter the interaction between chestnuts and blight. In Europe, where chestnut blight attacks the European chestnut, *Castanea sativa* Mill., atypical strains of *C. parasitica* with reduced virulence (termed hypovirulence) and reduced sporulation were often isolated from non-lethal cankers (Grente 1965). These atypical hypovirulent strains were parasitized with a double-stranded (ds) RNA hypovirus (Day *et al*. 1977), which is a member of the Hypoviridae (Hillman *et al*. 1995). Hypovirus infection often reduces canker expansion rates (Anagnostakis & Waggoner 1981), which provides more time for infected branches to produce wound callus tissue that can wall off the infection. If the callus successfully halts canker expansion, branch longevity is increased.

The reappearance of large reproducing chestnut trees, associated with a large proportion of *C. parasitica* isolates being parasitized by dsRNA (Fulbright *et al*. 1983), is currently taken to indicate recovery of

Present address and correspondence: Dr Anita L. Davelos, Department of Biology, The University of Texas-Pan American, 1201 West University Drive, Edinburg, TX 78541, USA (fax +1956 381 3657; e-mail: davelos@panam.edu).

American chestnut populations but this definition may well be inadequate for assessing the potential of dsRNA for restoring American chestnuts in eastern hardwood forests. When large branches are infected, the probability of survivorship is low when dsRNAs are absent, but high when the dsRNA is present in the infecting fungus. In contrast, the mortality of infected small branches (< 2 cm diameter), which is ignored by the current definition, is extremely high and is not affected by the presence of dsRNA (Davelos 1999). Thus, growth and survival of small American chestnuts may not benefit from the presence of dsRNA, and small trees in recovering populations may not therefore be able to grow to reproductive size. Such chestnut populations are not recovering in an ecological sense, because any negative effects of *C. parasitica* epidemics at the level of population growth rates are not ameliorated by the presence of hypovirus. In essence, the reappearance of large chestnut trees does not by itself signal that the long-term persistence of the population is assured. This idea was summarized by Silvertown *et al.* (1996, p. 595): 'To prevent the extinction of [a] population … involve[s] raising the finite rate of population increase to a value larger than 1.' The persistence of a population is not '… achieved simply by prolonging the life of individuals if there is no recruitment…'

We used matrix projection models to determine how pathogens alter host population growth rates. The finite rate of population increase (λ) has been shown to be sensitive to both abiotic and biotic influences. Although growth rates vary with habitat and over space and time for a variety of plant species (e.g. Huenneke & Marks 1987; Menges 1990; Kalisz & McPeek 1992; Horvitz & Schemske 1995; Kephart & Paladino 1997; Golubov *et al*. 1999), matrix models have rarely been used to evaluate the impact of disease on λ of plant populations (Emery 1998), despite the potential for pathogens to affect host population dynamics. Indeed, population size (Antonovics *et al*. 1994; Mihail *et al.* 1998) and population expansion rate (Carlsson & Elmqvist 1992) have been shown to be reduced by infection in natural plant-pathogen systems.

Matrix projection models were used to characterize the fate of individuals within each population, and to determine finite rates of population increase (λ). These measures allowed us to ask whether hypovirus infection of *C. parasitica* leads to ecological recovery of diseased American chestnut stands. If ecological recovery has occurred, we predict that:

1. Transition matrices for recovering populations will resemble those of disease-free more than epidemic populations.

2. Disease-free and recovering populations will have similar population growth rates.

Our objective is to assess quantitatively the effects of *C. parasitica* epidemics and the extent of hypovirus-mediated recovery in growth and survival of individual trees. We also determine whether changes evident at the level of the individual have a major influence on population growth rates of American chestnut in Michigan.

Materials and methods

SPECIES AND STUDY SITES

American chestnut, *C. dentata*, was a dominant overstorey species in hardwood forests of the eastern United States of America prior to the introduction of blight (Day & Monk 1974; Karban 1978; Russell 1987). After the spread of *C. parasitica*, oak (*Quercus* spp.), red maple (*Acer rubrum*) and hickory (*Carya* spp.) became the dominant overstorey tree species (Keever 1953; Stephenson *et al.* 1991). Today, chestnuts continue to be an important understorey species because of sprouts produced by extant tree root systems (Keever 1953; Russell 1987; Stephenson *et al*. 1991). However, infected sprout clusters exhibit reductions in survival and size, particularly when in competition with other hardwoods (Griffin *et al*. 1991; Parker *et al.* 1993). Heiniger & Rigling (1994) postulated that the natural spread of hypovirulence in Europe has led to a decline in the severity of disease and has allowed many stands of European chestnut to recover. Many attempts have been made to introduce hypoviruses as biological control agents of *C. parasitica* in the eastern United States (reviewed in MacDonald & Fulbright 1991), but they have failed to spread and contain the epidemic. Several hypotheses have been proposed to explain this failure, including high diversity of vegetative compatibility groups (Anagnostakis *et al.* 1986), the high susceptibility of American chestnuts to blight, and high competition from other tree species (MacDonald & Fulbright 1991; Heiniger & Rigling 1994).

Naturalized populations of *C. dentata* occur throughout the lower peninsula of Michigan (Brewer 1995). Populations originated from seed or seedlings planted by early settlers of the state. Populations expanded when farms were abandoned, and the state is now a patchwork of American chestnut populations ranging in size from a few individuals to over 1000 adult trees. Blight was first reported in Michigan in the late 1920s (Baxter & Strong 1931), and hypovirus was detected in the late 1970s (Day *et al*. 1977). In some cases hypoviruses have spread naturally, leading to recovery of some chestnut populations (Fulbright *et al*. 1983).

After a preliminary survey of over 20 American chestnut populations in the north-west lower peninsula of Michigan, the six sites with the most similar population size and site characteristics were chosen. However, although two sites, Leelanau (Leelanau County, latitude 45°03′09″, longitude 85°45′34″) and Missaukee Healthy (Missaukee County, 44°25′58″ and 85°09′23″), had escaped the *C. parasitica* epidemic (and were therefore used as disease-free control sites), the pathogen had colonized the other four chestnut populations at some point during the early 1970s to mid-1980s (D. W. Fulbright & T. Williams, personal communication).

Table 1 Stage classes used to describe populations of American chestnut and disease incidence (i.e. proportion of trees infected) by stage in the four populations infected with *Cryphonectria parasitica*

Stage	Size		Category	Disease incidence
	Height (cm)	d.b.h. (cm)		
1	≤ 50		First-year seedlings	0.00
2	≤ 50		Second-year or older seedlings	0.00
3	≤ 50		Disease or herbivore damaged	0.30
4	> 50 and ≤ 100		Small juveniles	0.48
5	> 100	≤ 1	Juveniles	0.47
6		> 1 and ≤ 10	Potentially reproductive	0.73
7		> 10 and ≤ 20	Potentially reproductive	1.00
8		> 20	Potentially reproductive	1.00

DsRNA was known to occur at the Frankfort (Benzie County, 44°38′37″ and 86°13′48″) and County Line (Manistee County, 44°31′00″ and 86°06′25″) sites for about a decade prior to the beginning of the current study, whereas Missaukee Diseased (Missaukee County, 44°26′13″ and 85°26′13″) and Stivers (Manistee County; 44°28′52″ and 85°51′48″) are experiencing severe epidemics of *C. parasitica* without significant invasion by dsRNA.

Cryphonectria parasitica infection occurs most commonly at branch points, where movement creates small wounds that allow the pathogen to enter the tree. Individuals less than 50 cm in height are only rarely infected and disease incidence increases with plant size (Table 1), presumably because of an increase in the number of potential wound entry sites. All large trees (diameter at breast height (d.b.h.) > 10 cm) at both the Stivers and Missaukee Diseased sites were infected. Less than 5% of the cankers at these two sites were infected with dsRNA hypovirus (Davelos 1999) and they were therefore designated as 'epidemic' sites in which the effects of *C. parasitica* infection could be monitored. At the two remaining 'infected' sites, County Line and Frankfort, disease incidence is comparable with that of the two epidemic populations, with all large trees being infected (Table 1), but trees were known to be recovering due to hyperparasite infection. At the beginning of this study > 85% of cankers at the two sites contained dsRNA (Davelos 1999), with distinct types present at County Line and Frankfort (Peever *et al.* 1997). Thus, the six sites form a natural experiment, allowing comparisons among the two disease-free, two epidemic, and two recovering populations to investigate the effects of *C. parasitica* and dsRNA on the demography of American chestnut populations.

POPULATION PROJECTION MATRICES

To calculate demographic parameters for each population, the following matrix model was used:

$$\mathbf{n}(t+1) = \mathbf{A}\mathbf{n}(t)$$

where $\mathbf{n}(t)$ is a vector of the number of individuals in each stage in year t, $\mathbf{n}(t+1)$ is the population vector in the following year, and **A** is the transition matrix for the population.

To estimate the matrix elements required for the model described above, individual trees were censused annually and placed into size-based stages to construct Lefkovitch (1965) stage-based transition matrices. Eight stages were constructed for each population (Table 1). Logistically, it was not possible to sample every small plant (i.e. stages 1–4) within a population. Therefore, eight to 16 permanent 9 × 9 m plots, which represented most microsites, were established in each population. All seedlings and plants less than 100 cm in height were marked and the height was measured in September/October of each year. The aggregate plot data within each population were used to estimate transition probabilities for trees in stages 1 to 4. The $\mathbf{n}(t)$ matrix requires population level estimates of the number of individuals in each size class. Numbers of plants in stages 1 to 4 within each population were therefore scaled from the area of the plots to the total area of the population (Table 2).

Stage 1 included only first-year seedlings, which were easily identified because the seed coat remained attached to the base of the new seedling at the start of the season. Most populations contained a heterogeneous group of plants ≤ 50 cm in height derived both from true seedlings and by size reductions due to *C. parasitica* infection, herbivory or shading. As significant differences in survival probability between true seedlings and other small individuals have been found (Davelos 1999), they were classified as separate stages (stages 2 and 3, respectively, Table 1). Stage boundaries beyond stage 3 were based on plant height for individuals ≤ 100 cm tall and on main stem diameter at breast height (d.b.h.) for individuals > 100 cm tall (Table 1).

All stage 5–8 individuals within a population were surveyed in the fall of each year for survivorship, size and reproduction. Measurements were made on entire genets rather than individual ramets, i.e. the largest stem of multistemmed individuals was measured. Transition probabilities for stages 5–8 were based on a complete census of all individuals within these stages. Size reductions (retrogressions) from large size stages occurred when the largest stem died and all surviving sprouts were smaller in size.

Table 2 Population area, mean fecundity, recruitment, survival, growth and finite rates of population increase of mean transition matrices for six American chestnut populations across four transition periods. Population area is in m^2. Means ± standard errors are reported for fecundity (burr production of reproducing trees) and recruitment (estimated number of first-year seedlings). Survival is the proportion of individuals surviving and growth is the proportion of individuals growing to a larger size class. λ ± standard error for the mean transition matrix of each population is presented

Population	Population area	Fecundity	Recruitment	Survival	Growth	λ ± SE
Missaukee Healthy	6 369 (8)*	78.3 ± 16.2	217.5 ± 110.6	0.88	0.10	0.995 ± 0.05
Leelanau	13 931 (12)	57.6 ± 12.2	122.5 ± 25.2	0.92	0.15	1.020 ± 0.04
County Line	7 344 (16)	54.7 ± 4.3	265.5 ± 92.5	0.95	0.16	1.010 ± 0.04
Frankfort	8 493 (12)	31.1 ± 4.7	180.0 ± 55.6	0.89	0.13	0.968 ± 0.07
Stivers	11 844 (12)	20.6 ± 4.3	60.0 ± 11.0	0.91	0.21	0.979 ± 0.03
Missaukee Diseased	5 063 (8)	59.1 ± 9.0	106.0 ± 56.9	0.92	0.17	1.000 ± 0.05

*The number of 9 × 9 m plots at the site. Plots were used to sample individuals in size classes 1–4.

Reproduction by an individual tree was determined by counting the number of branches with burrs for each tree. Three of these branches were selected and the number of burrs on each was counted. As nearly all burrs contain three seeds (Jaynes 1978; Paillet & Rutter 1989), the mean number of burrs per branch multiplied by the number of branches with burrs was used as the measure of reproduction for an individual. Because there is no seed bank (A.L. Davelos & A.M. Jarosz, unpublished data), the average number of seedlings produced per tree in each size class was calculated to fill in stage 1 (seedling row) of the matrix (Enright & Watson 1991; Caswell 2001). Total seedling production was estimated by scaling the number of seedlings in the plots to the total population area. The average number of seedlings per individual in a stage was determined using the following formula:

$$\frac{(\text{No. of burrs in stage}(t)/\text{no. of burrs for population}(t))\ (\text{estimated no. of seedlings for population}(t+1))}{\text{No. of trees in stage}(t)}$$

where t is time in years.

ANALYSES

Transition matrices

The six populations were compared for four transition periods: 1996–97, 1997–98, 1998–99 and 1999–2000. Some biologically important transitions were not observed in all populations. Individuals were rarely observed to grow from stage 2 to stage 4 and neither epidemic population ever showed forward transition from stage 7 to stage 8, i.e. no growth of trees in the 10–20 cm d.b.h. size class to the greater than 20 cm d.b.h. size class was observed, even though stage 8 individuals were present when the pathogen first invaded. Such missing transition probabilities were estimated by calculating the average probability of that transition across all populations within the period concerned. This method assumes that the null hypothesis (i.e. that the populations do not differ) is correct and estimates of differences among populations are therefore conservative (values for disease-free populations are biased downwards and values for diseased populations are biased upwards). If the transition did not occur in any population (e.g. death in the largest size class), a transition probability was estimated by assuming that the next individual sampled within the stage would make the transition. As stage 2 individuals could not be identified with certainty in 1996, the transition probabilities from 1997 to 1998 were used to estimate stage 2 transition values in the 1996–97 transition matrices.

Population growth rates and stable stage distributions

For each annual transition period, the finite rate of population increase (λ) of the six populations was compared based on transition matrices. This parameter was estimated by assuming that the elements of each transition matrix were constant, and the number of individuals in each stage was calculated iteratively using RAMAS/stage (Ferson 1994) until a stable stage distribution was attained. At this point the finite rate of population increase (λ) was estimated (Caswell 2001). Because finite rates of population increase (λ) for the four transition periods were not independent, profile analysis using multivariate analysis of variance (MANOVA) (PROC GLM; SAS Institute 1988b) was used to compare population growth over time. Profile analysis examines the levels of the main effect (Time × Disease), and the flatness of the response curves (Time) (i.e. whether or not the slopes of the curves differ from zero) (von Ende 2001). The disease status of a population (disease-free, epidemic or recovering) was the main effect included in the model. Pillai's trace is the test statistic reported because it is the most robust to violations of assumptions among the tests for significant differences among populations that differed in disease status (Scheiner 2001).

To examine general differences in λ between the populations, a mean transition matrix was calculated for each population over the four transition periods, which eliminates year-to-year variability (Huenneke & Marks 1987; Caswell 2001). Finite rates of population

increase were determined as above and confidence limits for each λ were calculated following the methods of Ebert (1999). Stable stage distributions were generated using RAMAS/stage (Ferson 1994) for the three mean transition matrices. Expected number of individuals in each stage was compared between population types with the log likelihood ratio, *G* (PROC FREQ; SAS Institute 1988a). This test is more robust than χ^2 for evaluating goodness of fit (Sokal & Rohlf 1981).

Elasticity

Elasticity values ($e_{ij} = a_{ij}/\lambda\ (\delta\lambda/\delta a_{ij})$ where a_{ij} is a value within the **A** matrix) represent the proportional contribution of each transition probability to λ (Caswell 2001). Elasticities within a given transition matrix sum to one, facilitating comparisons among populations (De Kroon *et al*. 1986). Elasticities were calculated for mean transition matrices of each population type (disease-free, recovering and epidemic) over all years (Huenneke & Marks 1987; Caswell 2001). Elasticity values were summed for reproduction, growth and stasis to determine the effects of these demographic parameters on λ for each population type (Hoffmann 1999).

Life table response experiments

Life table response experiments (LTRE) were conducted to examine which stage classes and demographic parameters contribute most to the effects of disease on population growth rate (Hoffmann 1999; Caswell 2001). Following the notation of Caswell (2001), the mean disease-free matrix was used as the reference matrix ($\mathbf{A}^{(r)}$) against which the mean recovering and epidemic matrices ($\mathbf{A}^{(m)}$) were compared. A difference matrix ($\mathbf{D}^{(m)} = \mathbf{A}^{(m)} - \mathbf{A}^{(r)}$) was determined and then a matrix of contributions ($\mathbf{C}^{(m)}$) was calculated, where $c_{ij} = d_{ij}s_{ij}$. Sensitivities, $s_{ij} = \delta\lambda/\delta a_{ij}$, are evaluated for matrix $\mathbf{A}\dagger = (\mathbf{A}^{(m)} + \mathbf{A}^{(r)})/2$. The effect of disease on differences in λ can be determined by examining the contributions of growth, survival and reproduction for each stage and overall contributions of each vital rate to differences in λ can be calculated by summing contributions across stages (Caswell 2001).

Results

TRANSITION MATRICES

Averaged across the four transition periods, the populations varied in fecundity (burr production), recruitment (number of first-year seedlings) and growth (the proportion of individuals moving to a larger size class) (Table 2). Recruitment was highly variable among years within each population, while mean survival fell within a fairly narrow range for all populations. Among the mean matrices for each population, finite rates of population increase did not differ significantly and ranged from a high of 1.020 for Leelanau (a disease-free population) to a low of 0.9675 for Frankfort (a recovering population) (Table 2). The confidence limits for the estimates of λ included values above and below 1 for all populations.

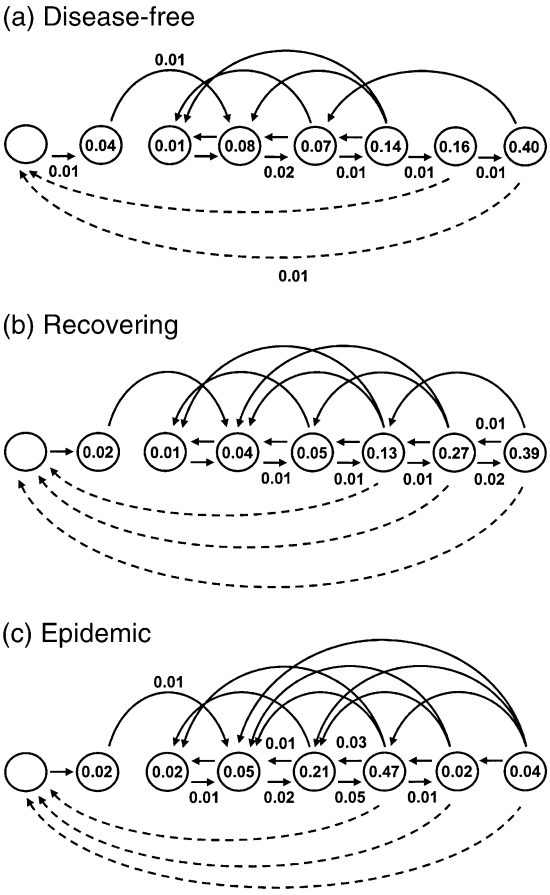

Fig. 1 Life cycles for (a) disease-free, (b) recovering and (c) epidemic populations based on mean transition matrices. Stage classes are represented by circles with stage 1 at the left and proceeding to stage 8 at the right. Elasticity values for stasis (probability of remaining in a stage from year to year) are presented in the circles. Forward arrows represent growth to a larger size class (elasticity values below arrows); reverse arrows represent retrogressions (i.e. plants becoming smaller in size) (elasticity values above arrows); dashed arrows represent fecundity (elasticity values below arrows). Arrows without numbers have elasticity values < 0.01.

Mean transition matrices for disease-free, recovering and epidemic populations are presented in Table 3 and life cycle graphs are shown in Fig. 1 (matrices for individual populations are presented in Appendix S1 in Supplementary Material). The direct effects of disease were most evident in the two largest size classes. No retrogressions (reductions in size) were observed for stage 7 or 8 trees in either disease-free population, with the exception of a single stage 8 tree at Missaukee Healthy during 1999–2000 that was struck by lightning. In contrast, some stage 7 and 8 trees retrogressed in size during each transition period for both epidemic and recovering populations. The severity of size reductions was greatest for epidemic populations, with an average of 30% of stage 7 trees and 10% of stage 8 trees being

Table 3 Mean transition matrices for three types (disease-free, recovering and epidemic) of American chestnut populations. Stages are described in Table 1. Italicized probabilities (the diagonals) represent stasis or remaining in a given stage from year to year. Probabilities below the diagonal represent growth and probabilities above the diagonal represent reductions in size (retrogressions). Probabilities within a column do not always sum to one due to mortality of individuals within a stage. Transition probabilities followed by a † are estimated, see text for details

Stage in $n+1$	Population type	Stage in Year n							
		1	2	3	4	5	6	7	8
1	Disease-free						0.001	0.03	7.74
	Recovering						0.016	0.78	2.43
	Epidemic						0.02	0.32	3.69
2	Disease-free	0.56	*0.80*						
	Recovering	0.71	*0.81*						
	Epidemic	0.62	*0.77*						
3	Disease-free			*0.80*	0.05	0.04	0.02		
	Recovering			*0.82*	0.10	0.04	0.01		
	Epidemic			*0.73*	0.06	0.02	0.005		
4	Disease-free		0.04†	0.05	*0.84*	0.07	0.03	–	–
	Recovering		0.02	0.07	*0.78*	0.09	0.02	0.002	–
	Epidemic		0.09	0.18	*0.66*	0.03	0.009	0.01	0.004
5	Disease-free				0.10	*0.81*	0.02	–	0.008
	Recovering				0.06	*0.78*	0.06	0.005	–
	Epidemic				0.25	*0.79*	0.07	0.04	0.01
6	Disease-free					0.08	*0.92*	–	–
	Recovering					0.09	*0.89*	0.04	0.005
	Epidemic					0.15	*0.90*	0.25	0.08
7	Disease-free						0.01	*0.95*	–
	Recovering						0.02	*0.91*	0.05
	Epidemic						0.01	*0.66*	0.01
8	Disease-free							0.05	*0.987*†
	Recovering							0.04	*0.944*†
	Epidemic							0.04†	*0.889*
Mortality	Disease-free	0.44	0.16	0.15	0.01	0	0	0	0.005
	Recovering	0.29	0.17	0.11	0.06	0	0	0.003	0.001
	Epidemic	0.38	0.14	0.09	0.03	0.01	0.006	0	0.007

reduced in size (Table 3). In particular, the proportion of stage 7 and 8 trees retrogressing was extremely high at the Stivers site in 1996–97 and 1998–99, with up to 95% of stage 7 trees and 40% of stage 8 trees moving to smaller size classes. Further evidence of the ability of *C. parasitica* to limit tree growth could be seen in epidemic populations, where no stage 7 tree grew to stage 8 during the study. This finding contrasted with the growth pattern seen in disease-free and recovering populations, where stage 7–8 transitions occurred sporadically over the four transition periods.

The only consistent pattern for trees in stages 2 to 5 was a higher probability of growth to a larger size class in the epidemic populations (Table 3). Increased growth of small trees in the epidemic populations may be an indirect effect of the pathogen through increased light levels at the forest floor due to the dieback of canopy trees infected by *C. parasitica* (A.L. Davelos & A.M. Jarosz, unpublished data). Finally, there were no consistent patterns for stage 6 trees among populations. However, the cumulative probability of retrogressing was nearly always greater than growth probabilities for stage 6 trees in all populations, with retrogressions being greater than growth for stage 6 individuals in 21 out of 24 matrices.

Disease also altered reproduction within epidemic and recovering populations, where stage 7 and 6 trees often flowered and set seed in addition to stage 8 trees. With rare exceptions, only stage 8 trees reproduced in healthy populations. Surprisingly, the rate of seedling production did not display any consistent trends, except that stage 8 trees at Missaukee Healthy were the most prolific in three out of four years.

POPULATION GROWTH AND STABLE STAGE DISTRIBUTIONS

There was surprisingly little variability in estimates of the finite rate of population increase (λ) among populations (Table 4). In the repeated measures analysis, there was no effect due to time ($F_{3,1} = 5.18$, $P = 0.31$) or disease status ($F_{3,2} = 7.41$, $P = 0.12$), although the average population growth rate for healthy populations (average $\lambda = 1.008$) was slightly above one and values

Table 4 Finite rate of population increase (λ) for six American chestnut populations. Observed values are based on transition matrices presented in the Appendix S1. Average of the observed values of λ for each population are presented

	Populations					
	Disease-free		Recovering		Epidemic	
	Missaukee Healthy	Leelanau	County Line	Frankfort	Stivers	Missaukee Diseased
1996–97 Observed λ	1.009	1.007	0.993	0.994	0.982	0.985
1997–98 Observed λ	0.994	1.012	1.028	0.994	1.014	0.997
1998–99 Observed λ	1.017	1.024	1.004	0.995	0.995	1.001
1999–2000 Observed λ	0.998	1.005	1.008	0.977	1.006	0.995
Average λ	1.005	1.012	1.008	0.990	0.999	0.995

for recovering (0.999) and epidemic (0.997) were slightly below one. The average population growth rate for populations within a disease status also did not differ ($F_{3,15} = 2.67$, $P = 0.08$). However, the two recovering populations ranked very differently: County Line had the second highest average λ, while Frankfort had the lowest (Table 4). Frankfort was the only site where λ never exceeded one. The lack of effect of disease on population growth rate is likely to be the result of infection reducing the size of individuals, but not killing them.

Chestnut blight does have a marked effect on the structure of populations (Fig. 2). The predicted stable stage distribution for epidemic populations was significantly different from that of disease-free populations ($\chi^2 = 991.47$, d.f. = 7, $P < 0.001$). At stable stage, disease-free populations are expected to be dominated by stage 2 trees, with stage 1 and 4 trees also being relatively important, while epidemic populations are expected to be dominated by stage 5 and 6 trees. Although predicted stable stage distributions for recovering and disease-free populations differed significantly ($\chi^2 = 115.45$, d.f. = 7, $P < 0.001$), recovering populations recapture much of the structure predicted for disease-free populations, with stage 2 trees being most frequent (Fig. 2). However, stage 1 and 4 trees are expected to be less frequent in recovering populations than in disease-free populations and stage 7 and 8 trees more common.

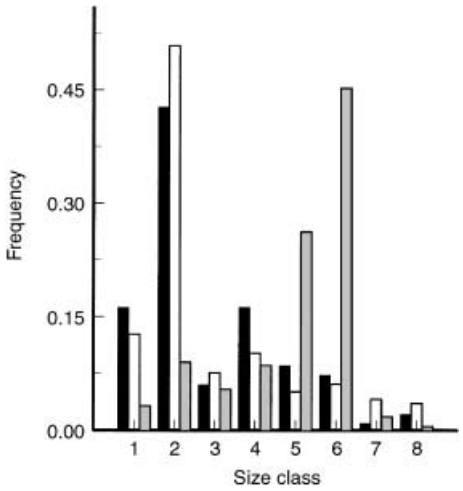

Fig. 2 Frequency of each stage class at the stable stage distribution of mean transition matrices for disease-free (black bars), recovering (open bars) and epidemic (grey bars) American chestnut populations.

ELASTICITY

Elasticity analysis of the mean transition matrices for the three population types indicated that stasis had an overwhelming influence on population growth rates across all population types (Table 5). In contrast, elasticity values for growth, reproduction and retrogression were less than one-tenth the values for stasis. However, the specific stage classes that contributed most to population growth differed among the population types. In disease-free populations, stasis within stages 6, 7 and 8 had the largest elasticity values and

Table 5 Elasticity values and contributions to reductions in population growth rate (λ) due to disease in recovering and epidemic populations of American chestnut for demographic parameters based on mean transition matrices of three American chestnut population types. Survival is the combined effects of stasis, growth and retrogression. Σ is the sum of survival and reproduction. The sum for the contributions to reductions in λ is the amount that λ for disease-free populations is reduced in recovering and epidemic populations

		Stasis	Growth	Retrogression	Survival	Reproduction	Σ
Elasticity	Disease-free	0.903	0.076	0.009	0.988	0.012	1.0
	Recovering	0.911	0.057	0.028	0.996	0.004	1.0
	Epidemic	0.834	0.104	0.056	0.994	0.006	1.0
Contributions to differences in λ	Recovering	−0.0371	−0.0046	0.0209	−0.0208	−0.0070	−0.0278
	Epidemic	−0.0721	0.0541	0.0078	−0.0102	−0.0051	−0.0153

accounted for greater than 60% of the contribution to λ (Fig. 1a). In contrast, for epidemic populations, stasis of stage 7 and 8 trees contributed little to population growth, while stasis of stage 5 and 6 trees contributed the most to population growth. The pattern of elasticity values in the recovering populations resembled that of disease-free populations, with stasis of stage 6, 7 and 8 trees making an overwhelming contribution to population growth rate. Retrogressions were much more common in epidemic (Table 3, Fig. 1c) and recovering populations (Table 3, Fig. 1b) than disease-free populations (Table 3, Fig. 1a), but the net detrimental effect of retrogressions on population growth rates was relatively minor for all populations. Indeed, the only retrogressions with elasticity values ≥ 0.01 were the transitions from stage 6 to 5 and from stage 5 to 4 in the epidemic populations (Fig. 1c), and from stage 8 to 7 in the recovering populations (Fig. 1b). Thus, the main effects of the pathogen are evident in the size structure of the population (Fig. 2), rather than in growth rates (Tables 2 and 4).

LIFE TABLE RESPONSE EXPERIMENTS

For both recovering and epidemic populations, the observed reduction in λ and the reduction predicted by LTRE analysis differed by less than 1%. Disease altered reproduction, survival and stasis, all of which contributed to reductions in population growth rates (Table 5). For both recovering and epidemic populations, the largest reduction in λ was caused by the effect of disease on stasis. Indeed, over 65% of the reductions in population growth in diseased populations were accounted for by reductions in stasis. Further, in diseased populations, retrogression had positive effects on differences in population growth between diseased and disease-free populations. This counterintuitive effect results from the greater probability of retrogression in diseased populations. For differences in λ between epidemic and disease-free populations, growth had a positive effect. As stated earlier, this effect is probably an indirect result of disease caused by increased light levels in the forest understorey after the dieback of overstorey trees. Given the ability of chestnuts to survive by producing sprouts from the root collar, it is not surprising that reductions in survival did not contribute much to reductions in λ.

The contribution of reductions in stasis to the reduction in λ was mainly the result of the effect of disease on stage 7 and 8 trees for both recovering and epidemic populations (Fig. 3). Together with the concomitant positive effect of retrogression for stage 7 and 8 trees, these results suggest that retrogressions (reductions in size) of stage 7 and 8 trees had a large impact on the negative contribution of growth to λ in diseased American chestnut populations, while the increased growth of smaller trees (stages 2–5) due to higher light levels at the forest floor in epidemic populations had a consistently positive effect on λ.

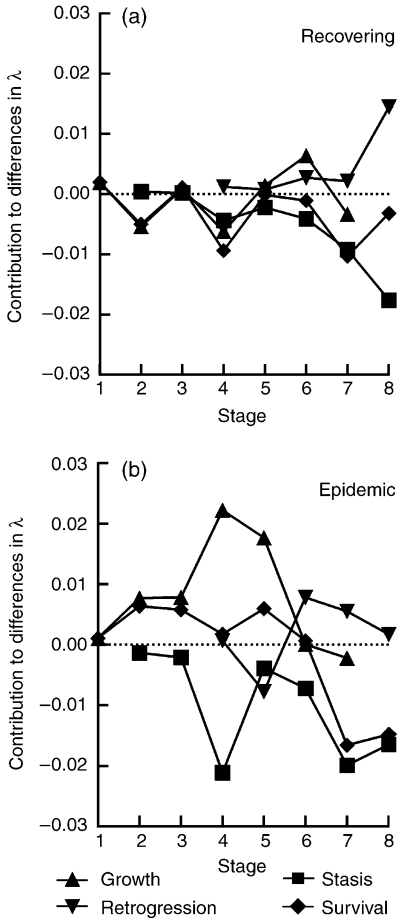

Fig. 3 The contributions of growth, retrogression, stasis and survival to reductions in λ of American chestnuts due to disease. The contributions were obtained from the analysis of life table response experiments (LTRE) comparing the mean disease-free population matrix with the mean recovering population matrix and with the mean epidemic population matrix.

Discussion

It has been suggested that pathogens can affect many aspects of the ecological and evolutionary dynamics of plant populations, including alterations in population size (Burdon 1987). However, very few studies have documented that disease epidemics actually reduce population size or growth rates (Carlsson & Elmqvist 1992; Antonovics *et al*. 1994; Emery 1998; Mihail *et al*. 1998). We found that *C. parasitica* epidemics had almost no influence on the growth rate of American chestnut populations, despite very high disease incidence among the largest trees in these populations. The constancy in population growth rates is explained largely by the fact that infections debilitate a tree, and only rarely cause death. Mortality of the largest trees (stages 6–8) increases by only 6% in epidemic populations, but this drop in survival is compensated by decreased mortality of smaller trees (stages 2–5) (Table 3). Thus, the large overall contribution of survival to population growth rates is essentially unchanged in epidemic populations (Table 5). The altered pattern of

mortality combined with changed growth patterns of infected trees has an immense effect on which trees are contributing most to population growth. Whereas survival of large trees in stages 7 and 8 contributes > 50% to population growth in disease-free populations, these two size classes contribute only 6% in epidemic populations (Fig. 1). The major effect of epidemics is to alter transition probabilities of large trees, resulting in significant changes in the predicted stable size structure of populations (Fig. 2). Further, reductions in stasis and an increase in retrogression of the largest trees contribute most to reductions in population growth rates for diseased populations (Fig. 3). For American chestnuts, the major epidemic following the introduction of *C. parasitica* into the United States has resulted in a rapid change in the species' place in eastern hardwood forests. Although still dominant numerically, chestnuts are now restricted to the forest understorey (Keever 1953; Russell 1987; Stephenson *et al*. 1991).

Invasion of dsRNAs into the pathogen population had a positive influence on the population dynamics of *Castanea dentata*. Chestnuts at recovering sites exhibited dynamics that were unique but, in many important ways, these recovering populations resembled disease-free populations more closely than epidemic populations. The contribution of transition elements to population growth rates was very similar to that seen in disease-free populations (Fig. 1). The three highest proportional contributions to population growth were made by survival of trees in stages 6, 7 and 8 in both disease-free and recovering populations, which differed from the pattern exhibited by the epidemic populations. The transition from stage 7 to 8 was re-established at both recovering sites, and the transition from stage 6 to 7 was enhanced at one recovering site, County Line. As a consequence of dsRNA invasion, the predicted stable stage distribution for recovering populations more closely resembled that predicted for the disease-free populations (Fig. 2).

Several scientists have proposed that dsRNA can be used as a biological control agent to dampen the effects of *C. parasitica* infections and restore *C. dentata* to its former position of dominance in the eastern hardwood forest (Van Alfen *et al*. 1975; MacDonald & Fulbright 1991; Nuss 1992). Our data indicate that dsRNAs can have a positive influence on the population structure of *C. dentata*. In recovering populations, the transition matrices do change in a manner indicating that the effects of disease are reduced, and the predicted stable stage distribution regains much of the character of disease-free populations. However, our inability to differentiate population growth rates across the three population types limits our ability to state conclusively that infected chestnut populations with dsRNA have recovered in an ecological sense. While dsRNAs had a net positive effect in general on large trees in recovering sites, stems continued to be girdled and killed by the blight, causing stage 7 and 8 trees occasionally to retrogress to smaller size classes. These retrogressions reduce the probability that trees contribute to population growth through stasis, as indicated by the negative contribution of stasis to population growth for stage 7 and 8 trees in diseased populations (Fig. 3). This inability to attain maximal growth may not affect *C. dentata* in Michigan where forests are rather open and shading of medium sized trees is uncommon. Indeed, the positive contribution of the growth of small trees to population growth rates in epidemic populations revealed by LTRE supports this idea (Fig. 3). However, the inability to achieve maximal growth rates may be a problem within the main portion of the species' range where several aggressive codominant tree species may effectively suppress recovering American chestnut trees. Suppression by competing species has been shown to be important in the eastern United States, where periodic cutting of competing hardwoods increases growth of infected *C. dentata* (Griffin *et al*. 1991).

Another potential problem of using dsRNAs for biological control is that dsRNAs may evolve to reduce their effect on *C. parasitica*. A theoretical model of this system suggests that selection will favour dsRNAs that minimize their negative effect on the pathogen (Taylor *et al*. 1998). DsRNAs are known to differ in their effects (W.L. MacDonald & M. Double, personal communication), and some may actually be commensalistic with the pathogen (Enebak *et al.* 1994). Thus, the effectiveness of dsRNAs as a biological control may diminish through time.

As the dsRNA hyperparasite is completely intracellular, its invasion into a *C. parasitica* population mimics phenotypically an evolutionary reduction in virulence. There is active debate about how changes in virulence should effect host populations (e.g. Bull 1994). While it seems reasonable to assume that reduced virulence should benefit host population dynamics, some theoretical work suggests that this need not happen in all cases (Lenski & May 1994). Our data suggest that reduced pathogen virulence alters the ecology of the *C. parasitica–C. dentata* interaction in a way that allows significant host recovery.

Matrix models have been used to examine population viability and identify at-risk populations for rare and endangered species (e.g. Menges 1990; Silvertown *et al*. 1993; Ratsirarson *et al.* 1996; Kephart & Paladino 1997). Our results indicate that demographic parameters, such as population growth rates and summed elasticity values, may fail to detect important changes among populations. For long-lived species, population growth rate estimates based on single year changes in population size may not be sensitive enough to detect important changes in population dynamics that would be evident if change were measured over a period of a decade. This may be particularly true for pathogens or other stresses that do not cause major changes in the life span of extant individuals. In contrast, factors that reduce the survival of individuals from long-lived species, such as harvesting, may have profound impacts on population persistence (e.g. Chaloupka 2002;

Freckleton *et al*. 2003). We find that life table response experiments (LTRE), that can identify changes in growth or survival for particular size classes, may indicate important changes in a population that are not apparent in some population-level parameters. The LTRE approach may offer further benefits when modelling the effects of biological control efforts because it can offer insight on the best methods for deploying the biocontrol agent in natural and agricultural systems.

Acknowledgements

We thank the many people who spent long hours helping us with our yearly censuses. In particular we wish to acknowledge Jessica Cook, Dennis Fulbright, Ngoc Kieu, Richard Leschen, Emily Lyons, Erin Mason, Jennifer Schaupp, David Taube and a host of 'volunteer' graduate and undergraduate students. Their assistance and advice made this work possible. We thank Bill Hoffmann for generous assistance with the LTRE analyses. Our work was supported by NSF grants DEB-9623416 and DEB-9509034, and by a graduate student research grant from Michigan State University's Ecology, Evolutionary Biology and Behaviour program. We extend our gratitude to the owners of the properties where we conducted our research for allowing us access to their land.

Supplementary material

The following material is available from
http://www.blackwellpublishing.com/products/journals/suppmat/JEC/JEC907/JEC907sm.htm

Appendix S1 Transition matrices for six American chestnut populations.

References

Anagnostakis, S.L., Hau, B. & Kranz, J. (1986) Diversity of vegetative compatibility groups of *Cryphonectria parasitica* in Connecticut and Europe. *Plant Disease*, **70**, 536–538.

Anagnostakis, S.L. & Waggoner, P.E. (1981) Hypovirulence, vegetative incompatibility, and the growth of cankers of chestnut blight. *Phytopathology*, **71**, 1198–1202.

Antonovics, J., Thrall, P., Jarosz, A. & Stratton, D. (1994) Ecological genetics of metapopulations: the *Silene-Ustilago* plant-pathogen system. *Ecological Genetics* (ed. L.A. Real), pp. 146–170. Princeton University Press, Princeton, New Jersey.

Baxter, D.V. & Strong, F.C. (1931) Chestnut blight in Michigan. *Michigan State College Agricultural Experiment Station Circular Bulletin*, **135**, 1–18.

Brewer, L.G. (1995) Ecology of survival and recovery from blight in American chestnut trees (*Castanea dentata* (Marsh.) Borkh.) in Michigan. *Bulletin of the Torrey Botanical Club*, **122**, 40–57.

Bull, J.J. (1994) Perspective virulence. *Evolution*, **48**, 1423–1437.

Burdon, J.J. (1987) *Diseases and Plant Population Biology*. Cambridge University Press, Cambridge.

Carlsson, U. & Elmqvist, T. (1992) Epidemiology of anthersmut disease (*Microbotryum violaceum*) and numeric regulation of populations of *Silene dioica*. *Oecologia*, **90**, 509–517.

Caswell, H. (2001) *Matrix Population Models*, 2nd edn. Sinauer Associates, Sunderland, Massachusetts.

Chaloupka, M. (2002) Stochastic simulation modeling of southern Great Barrier Reef green turtle population dynamics. *Ecological Modelling*, **148**, 79–109.

Davelos, A.L. (1999) *Double-stranded RNA mediated recovery of American chestnut populations: a demographic analysis*. PhD thesis, Michigan State University, East Lansing, Michigan.

Day, P.R., Dodds, J.A., Elliston, J.E., Jaynes, R.A. & Anagnostakis, S.L. (1977) Double-stranded RNA in *Endothia parasitica*. *Phytopathology*, **67**, 1393–1396.

Day, F.P. & Monk, C.D. (1974) Vegetation patterns on a southern Appalachian watershed. *Ecology*, **55**, 1064–1074.

De Kroon, H.J., Plaisier, A., van Groenendael, J. & Caswell, H. (1986) Elasticity: the relative contribution of demographic parameters to population growth rate. *Ecology*, **67**, 1427–1431.

Ebert, T.A. (1999) *Plant and Animal Populations: Methods in Demography*. Academic Press, San Diego.

Emery, K.M. (1998) *Population dynamics of birdsfoot trefoil in relation to disease and microclimate*. PhD thesis, University of Missouri, Columbia, Missouri.

von Ende, C.N. (2001) Repeated-measures analysis: growth and other time-dependent measures. *Design and Analysis of Ecological Experiments*, 2nd edn (eds S.M. Scheiner & J. Gurevitch), pp. 134–157. Oxford University Press, New York.

Enebak, S.A., MacDonald, W.L. & Hillman, B.I. (1994) Effect of dsRNA associated with isolates of *Cryphonectria parasitica* from the central Appalachians and their relatedness to other dsRNAs from North America and Europe. *Phytopathology*, **84**, 528–534.

Enright, N.J. & Watson, A.D. (1991) A matrix population model analysis for the tropical tree, *Araucaria cunninghamii*. *Australian Journal of Ecology*, **16**, 507–520.

Ferson, S. (1994) *RAMAS/Stage: Generalized Stage-Based Modeling for Population Dynamics*. Applied Biomathematics, Setauket, New York.

Freckleton, R.P., Matos, D.M.S., Bovi, M.L.A. & Watkinson, A.R. (2003) Predicting the impacts of harvesting using structured population models: the importance of density-dependence and timing of harvest for a tropical palm tree. *Journal of Applied Ecology*, **40**, 846–858.

Fulbright, D.W., Weidlich, W.H., Haufler, K.Z., Thomas, C.S. & Paul, C.P. (1983) Chestnut blight and recovering American chestnut trees in Michigan. *Canadian Journal of Botany*, **61**, 3164–3171.

Golubov, J., Mandujano, M.D., Franco, M., Montana, C., Eguiarte, L.E. & Lopez-Portillo, J. (1999) Demography of the invasive woody perennial *Prosopis glandulosa* (honey mesquite). *Journal of Ecology*, **87**, 955–962.

Grente, J. (1965) Les formes hypovirulentes d'*Endothia parasitica* et les espoirs de lutte contre le chancre du châtaignier. *Académie d'Agriculture de France, Extrait du Proces-verbal de la Séance*, **51**, 1033–1037.

Griffin, G.J., Smith, H.C., Dietz, A. & Elkins, J.R. (1991) Importance of hardwood competition to American chestnut survival, growth, and blight development in forest clearcuts. *Canadian Journal of Botany*, **69**, 1804–1809.

Heiniger, U. & Rigling, D. (1994) Biological control of chestnut blight in Europe. *Annual Review of Phytopathology*, **32**, 581–599.

Hillman, B.I., Fulbright, D.W., Nuss, D.L. & van Alfen, N.K. (1995) Hypoviridae. *Sixth Report of the International Committee for the Taxonomy of Viruses* (eds F.A. Murphy, C.M. Fauquet, D.H.L. Bishop, S.A. Ghabrial, A.W. Jarvis, G.P. Martelli *et al*.), pp. 261–264. Springer Verlag, New York.

Hoffmann, W.A. (1999) Fire and population dynamics of woody plants in a neotropical savanna: matrix model projections. *Ecology*, **80**, 1354–1369.

Horvitz, C.C. & Schemske, D.W. (1995) Spatiotemporal variation in demographic transitions of a tropical understory herb: projection matrix analysis. *Ecological Monographs*, **65**, 155–192.

Huenneke, L.F. & Marks, P.L. (1987) Stem dynamics of the shrub *Alnus incana* ssp. *rugosa*: transition matrix models. *Ecology*, **68**, 1234–1242.

Jaynes, R.A. (1978) Selecting and breeding blight resistant chestnut trees. *Proceedings of the American Chestnut Symposium* (eds W.L. MacDonald, F.C. Cech, J. Luchok & C. Smith), pp. 4–6. West Virginia University Press, Morgantown, West Virginia.

Kalisz, S. & McPeek, M.A. (1992) Demography of an age-structured annual: resampled projection matrices, elasticity analyses, and seed bank effects. *Ecology*, **73**, 1082–1093.

Karban, R. (1978) Changes in an oak-chestnut forest since the chestnut blight. *Castanea*, **43**, 221–228.

Keever, C. (1953) Present composition of some stands of the former oak-chestnut forest in the southern Blue Ridge Mountains. *Ecology*, **34**, 44–54.

Kephart, S.R. & Paladino, C. (1997) Demographic change and microhabitat variability in a grassland endemic, *Silene douglasii* var. *oraria* (Caryophyllaceae). *American Journal of Botany*, **84**, 179–189.

Lefkovitch, L.P. (1965) The study of population growth in organisms grouped by stage. *Biometrics*, **21**, 1–18.

Lenski, R.E. & May, R.M. (1994) The evolution of virulence in parasites and pathogens: reconciliation between two competing hypotheses. *Journal of Theoretical Biology*, **169**, 253–265.

MacDonald, W.L. & Fulbright, D.W. (1991) Biological control of chestnut blight: use and limitations of transmissible hypovirulence. *Plant Disease*, **75**, 656–661.

Menges, E.S. (1990) Population viability analysis for an endangered plant. *Conservation Biology*, **4**, 52–62.

Merkel, H.W. (1905) A deadly fungus on the American chestnut. *10th Annual Report of the New York Zoological Society*. New York Zoological Society, Bronx, New York.

Mihail, J.D., Alexander, H.M. & Taylor, S.J. (1998) Interactions between root-infecting fungi and plant density in an annual legume, *Kummerowia stipulacea*. *Journal of Ecology*, **86**, 739–748.

Milgroom, M.G. (1995) Population biology of the chestnut blight fungus, *Cryphonectria parasitica*. *Canadian Journal of Botany* (Suppl. 1), **73**, S311–S319.

Milgroom, M.G., Kerong, W., Yang, Z., Lipari, S.E. & Shigeru, K. (1996) Intercontinental population structure of the chestnut blight fungus. *Mycologia*, **88**, 179–190.

Nuss, D.L. (1992) Biological control of chestnut blight: an example of virus-mediated attenuation of fungal pathogenesis. *Microbiological Reviews*, **56**, 561–576.

Paillet, F.L. & Rutter, P.A. (1989) Replacement of native oak and hickory tree species by the introduced American chestnut (*Castanea dentata*) in southwestern Wisconsin. *Canadian Journal of Botany*, **67**, 3457–3469.

Parker, G.G., Hill, S.M. & Kuehnel, L.A. (1993) Decline of understory American chestnut (*Castanea dentata*) in a southern Appalachian forest. *Canadian Journal of Forestry Research*, **23**, 259–265.

Peever, T.L., Liu, Y.-C. & Milgroom, M.G. (1997) Diversity of hypoviruses and other double-stranded RNAs in *Cryphonectria parasitica* in North America. *Phytopathology*, **87**, 1026–1033.

Ratsirarson, J., Silander, J.A. Jr & Richard, A.F. (1996) Conservation and management of a threatened Madagascar palm species, *Neodypsis decaryi*, Jumelle. *Conservation Biology*, **10**, 40–52.

Russell, E.W.B. (1987) Pre-blight distribution of *Castanea dentata* (Marsh.) Borkh. *Bulletin of the Torrey Botanical Club*, **114**, 183–190.

SAS Institute (1988a) *SAS Procedures Guide*, release 6.03. SAS Institute, Cary, North Carolina.

SAS Institute (1988b) *SAS/STAT User's Guide*, release 6.03. SAS Institute, Cary, North Carolina.

Scheiner, S.M. (2001) MANOVA: multiple response variables and multispecies interactions. *Design and Analysis of Ecological Experiments*, 2nd edn (eds S.M. Scheiner & J. Gurevitch), pp. 99–115. Oxford University Press, New York.

Silvertown, J., Franco, M. & Menges, E. (1996) Interpretation of elasticity matrices as an aid to the management of plant populations for conservation. *Conservation Biology*, **10**, 591–597.

Silvertown, J., Franco, M., Pisanty, I. & Mendoza, A. (1993) Comparative plant demography – relative importance of life-cycle components to the finite rate of increase in woody and herbaceous perennials. *Journal of Ecology*, **81**, 465–476.

Sokal, R.R. & Rohlf, F.J. (1981) *Biometry*, 2nd edn. W.H. Freeman, New York.

Stephenson, S.L., Adams, H.S. & Lipford, M. (1991) The present distribution of chestnut in the upland forest communities of Virginia. *Bulletin of the Torrey Botanical Club*, **118**, 24–32.

Taylor, D.R., Jarosz, A.M., Lenski, R.E. & Fulbright, D.W. (1998) The acquisition of hypovirulence in host-parasite systems with three trophic levels. *American Naturalist*, **151**, 343–355.

Van Alfen, N.K., Jaynes, R.A., Anagnostakis, S.L. & Day, P.R. (1975) Chestnut blight: biological control by transmissible hypovirulence in *Endothia parasitica*. *Science*, **189**, 890–891.

von Ende, C.N. (2001) Repeated-measures analysis: Growth and other time-dependent measures. *Design and Analysis of Ecological Experiments*, 2nd edn (eds S.M. Scheiner & J. Gurevitch), pp. 134–157. Oxford University Press, New York.

Received 8 March 2003
revision accepted 6 May 2004
Handling Editor: Hans de Kroon