ECOGRAPHY 28: 181–190, 2005

Disease and the devil: density-dependent epidemiological processes explain historical population fluctuations in the Tasmanian devil

Corey J. A. Bradshaw and Barry W. Brook

Bradshaw, C. J. A. and Brook, B. W. 2005. Disease and the devil: density-dependent epidemiological processes explain historical population fluctuations in the Tasmanian devil. – Ecography 28: 181–190.

Australia's last mega-carnivore marsupial, the Tasmanian devil *Sarcophilus harrisii*, Dasyuridae is endemic to the island state of Tasmania. The recent appearance and rapid spread of a debilitating and usually lethal, cancer-like disease has raised concerns regarding the species' future. We used a demographic matrix modelling approach to evaluate the potential long-term implications of epidemics on this population. Both adult survival and temporally autocorrelated re-occurrence of disease were expressed as a function of female abundance. Large fluctuations in abundance resulted when disease outbreaks were conditioned to be density-dependent; however, this resulted in a low probability of quasi-extinction due to the dissipation of disease transmission at low densities. Epidemic stochasticity alone in an otherwise deterministic model resulted in major population cycles occurring every 77–146 yr, consistent with historical reports. Although epidemics in this species may not result in extinction directly, the contemporary presence of additional mortality sources during periods of low abundance may increase extinction risk.

C. J. A. Bradshaw (corey.bradshaw@cdu.edu.au) and B. W. Brook, Key Centre for Tropical Wildlife Management and Research School of Environmental Studies, Charles Darwin Univ., Darwin, NT 0909, Australia.

With the extinction of the Tasmanian tiger *Thylacinus cynocephalus* early last century (Bulte et al. 2003), the dubious distinction of being Australia's (and the world's) last marsupial mega-carnivore fell to the endemic Tasmanian devil *Sarcophilus harrisii*. The devil is the largest carnivorous marsupial (dasyurid) in Australia (mean adult male weight = 8.7 kg; female = 6.1 kg – Guiler 1978), and relies mainly on scavenging rather than active predation (Buchmann and Guiler 1977, Guiler 1992). Devils are entirely restricted to the island state of Tasmania, although they were once more widespread throughout continental Australia. Eradication from the mainland ca 400 yr ago was most likely due to competition from the introduced dingo *Canis lupus dingo* (ca 4000 yr BP), and the intensification of human settlement (Dawson 1982, Corbett 1995, Johnson and Wroe 2003).

In the late 1990s it was noted that in certain areas of Tasmania where devils occur at high densities, individuals were succumbing to a debilitating disease (Jones et al. 2004). The disease (often called "Devil Facial Tumour Disease" or "DFTD") produces an array of cancerous tumours around the face and neck, leading to death within months of the first symptoms. Although little has been published on the aetiology of the disease, the infectious agent is believed to be a retrovirus (Jones 2003; although recent evidence casts doubt on this assumption – Loh 2004) that only affects adults. The endemic population of devils in Tasmania is thought to have "halved" since the mid-1990s (Jones 2003). No other endemic species appear to be susceptible to the disease (Mooney 2003a,b).

The emergence of this debilitating, high-profile disease in the iconic and taxonomically unique Tasmanian devil

Accepted 2 November 2004

ISSN 0906-7590

has raised serious concerns about the future of the species (Jones 2003, Mooney 2003a, Clausen 2004). However, DFTD is not the first known occurrence of disease in devils. A serious epidemic led to a large population reduction around the turn of last century, with earlier crash/recovery phases also recorded (Guiler 1964, 1992). Although the aetiology of historic epidemics is unknown, the absence of gross pathology reports for historic epidemics as seen with DFTD suggests that aetiologies differed. The population recovered to an estimated high of 130 000–150 000 in the 1990s prior to the outbreak of the most current disease event (Mooney pers. comm.). The rapid decline and recovery of the species within the last century, and perhaps throughout the period of European colonisation, suggest that susceptibility to epidemics (i.e., not necessarily just to DFTD) within this species occurs frequently or even cyclically. Cyclic fluctuations in viral or bacterial infections and associated high mortality have been observed in salamander *Ambystoma tigrinum* and great gerbil *Rhombomys opimus* populations (Davidson et al. 2003, Davis et al. 2004).

Although disease pathology has been addressed historically in the context of veterinary science (Lafferty and Gerber 2002), some attempts to combine epidemiology and population ecology have resulted in a firmer understanding of population dynamics when two or more species interact (Hadeler and Freedman 1989, Beltrami and Carroll 1994, Chattopadhyay et al. 2003, Rohani et al. 2003, Grenfell et al. 2004). For example, Beltrami and Carroll (1994) modelled the role of viral disease in recurrent phytoplankton blooms. Their model was successful in describing how the contamination of algal cells by viruses can serve as a regulatory mechanism in bloom dynamics. Nonetheless, there has been relatively few attempts to combine epidemiology and population models to understand the behaviour of ecological systems (Woodroffe 1999, Lafferty and Gerber 2002). Indeed, the impacts of disease on populations is a relatively neglected topic in ecology, evolution and conservation biology, despite its important link with biodiversity (Harvell et al. 1999) and implications for species management.

Here we assess the potential effects of disease epidemics on the temporal trends in abundance of the Tasmanian devil population since early European colonisation by considering a set of hypothesised relationships between disease and population dynamics. This was achieved using a baseline population projection matrix model (Caswell 2001) incorporating the best demographic data available for the species, and then evaluating the effect of pathogens on changes in abundance by incorporating frequency of occurrence according to either a) an autocorrelated random process or b) a density-feedback mechanism. It was not our intention to model the effects of DFTD specifically on the Tasmanian devil population; rather, our aim was to incorporate a generic, density-dependent disease-producing pathogen based on the observed impact of DFTD. Our results are developed within an evolutionary framework that examines the role of disease in shaping this, and perhaps other, marsupial species life-history characteristics. From these results, we attempt to provide a firmer understanding of the long-term implications of the current outbreak and other possible disease-producing pathogens on the species' future.

Methods

Reconstructing the past

A search of the available literature provided indices of relative population change since European colonisation and spread in the late 18th and early 19th centuries. We constructed a relative population abundance graph from these sources (Fig. 1) that comprised mainly anecdotal and qualitative assessments of abundance (Flynn 1939, Guiler 1964, 1970a, 1982, 1992). It should be emphasised that this graph is not a literal quantification of population size – it simply outlines the approximate relative abundance of devils in Tasmania since European colonisation. At least three rapid declines are evident – one in the early 19th century, one just prior to the turn of the 20th century (Guiler 1992), and the most recent epidemic of DFTD beginning in the late 1990s (Mooney 2003a, b).

Modelling environment

Populations in which individuals differ in their contributions to population growth are structured, and these individuals can be classed by state (e.g. age, size, sex,

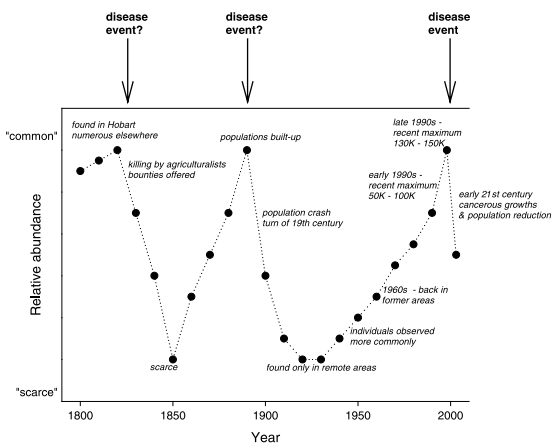

Fig. 1. Reconstructed relative abundance of Tasmanian devils in Tasmania since European colonisation.

stage, etc. – Morris and Doak 2002). In such cases where information is available on variation in vital rates (i.e. rates of birth, growth, maturation, fertility and mortality – Caswell 2001) among states, the principal and most-accessible tool for assessing temporal changes in populations is the population projection matrix model (Morris and Doak 2002). In general, structured models give a more accurate portrayal of population change through time (although see Pfister and Stevens 2003 for growth-related limitations to matrix models), and they can contribute to more targeted management questions because they identify the vital rates and specific states with the greatest influence on the population rate of change (Caswell 2001, Morris and Doak 2002).

Thus, our modelling approach was to begin with deterministic, density-independent Leslie matrix population models (Leslie 1945) that describe the average demography of the Tasmanian devil population. The matrix's dominant eigenvalue (λ_1) indicates the rate of population change through time (Leslie 1945, Caswell 2001). Our first basic assumption was that although population size has fluctuated substantially since European colonisation, it was "stable" over the long term (i.e. 200 yr since colonisation), being regulated by density dependence (as per Bulte et al. 2003 for the Tasmanian tiger). We then examined progressively more complex modelling scenarios that incorporated stochastic or regulatory disease-transmission effects, whilst maintaining the assumption that the devil population has not increased or decreased on average over the course of the last two centuries.

The baseline deterministic matrix models included density-dependent adult survival but no disease. Disease was then imposed by invoking outbreaks randomly throughout a 200-yr Markov chain projection of the population, with no other form of stochasticity incorporated into the projections. This latter model was used to identify which probabilities of disease occurrence (p[disease]) and temporal autocorrelation ([ac] = inter-year disease-event dependency) resulted in long-term population stability on average. Finally, we modelled [ac] as a function of population density in combination with density-dependent adult survival.

Although we were obliged to make assumptions regarding the regulatory mechanisms and the severity of the disease epidemics themselves, we are confident these relatively simple models are more appropriate in this context than complex approaches requiring many parameters unsupported by data (Ginzburg and Jensen 2004), or unregulated models allowing extremely high and unrealistic population sizes (data not presented). Although the data required to derive regulatory functions are often unknown or impossible to collect (Beissinger and Westphal 1998), population projections are often influenced greatly, and rendered more realistic, by the explicit incorporation of regulatory mechanisms (Ginzburg et al. 1990, Chapman et al. 2001).

Devil life history

The devil is an annual breeder with most births occurring between March and May (Hughes 1982). Female devils begin breeding at two years of age (Guiler 1964, 1970b, Pemberton 1990) though one-year-old females have occasionally been observed with pouch young (Pemberton 1990). Devils produce a maximum of four pouch young annually, with a mean of two to three (Guiler 1970b). Pouch young sex ratios vary regionally, but are ca 50:50 on average (Pemberton 1990). Lifespan is relatively short and most females die by the age of six years (Buchmann and Guiler 1977, Guiler 1978).

Population densities vary with habitat, with higher densities (5–12 km^{-2} – Guiler 1970a, Pemberton 1990) in drier to warmer dry sclerophyll forests and adjacent agricultural areas than in wetter rainforest and colder alpine moorlands (<2 km^{-2} – Pemberton 1990). Individuals have large home ranges (>10 km^2), and can travel up to 10 km per night to forage (Pemberton 1990), although they will often return to a central position during the day (Guiler 1964, 1970b). A solitary animal, devils avoid conspecifics (outside of the mating season), but will fight aggressively over carrion (Buchmann and Guiler 1977, Jones 1998). Agonistic interactions often lead to severe facial lacerations that may increase the transmission rate of pathogens between individuals (Guiler 1992, Kabat pers. comm.).

Description of the models

The basic form of the age-classified model was defined using MATLAB software, with six yearly age-classes used to represent the female segment of the population only. Vital rates were s_i = age-specific probability of survival from age i to i+1, m_i = the average age-specific number of pouch young produced per breeding event, x = the pouch young sex ratio and b = the proportion of mothers breeding per breeding event. The matrix entries were derived assuming a birth-pulse, pre-breeding design, with first-year survival incorporated into the fertility coefficients (Caswell 2001).

To calculate the initial population vectors [n_1, n_2, ... n_6], we chose the average, recent "maximum" population size estimated from field and anecdotal records. Here, the population maximum was assumed to equal the average of 130 000–150 000 (i.e. 140 000) individuals estimated to be alive in the 1990s prior to the influence of DFTD (Mooney pers. comm.). This value was then halved to represent the "average" population size since European colonisation. Only females were modelled, so we assumed an equal sex ratio to estimate an "average"

female population size ($\hat{N}_1$) of 35 000. This $\hat{N}_1$ was divided into the age-specific initial population vector using the stable-age distribution (sad) calculated from the matrix producing population increase (see below).

Deterministic projections in the absence of disease

Our first goal was to construct a deterministic model for a growing population in the absence of disease, based on realistic vital rates for Tasmanian devils. First, we used the vital rates calculated for devils in the only complete-life-cycle dataset available (Guiler 1978). Here, survival rates were re-calculated to be expressed as age-specific annual survival. To this end we used a logarithmic regression of the number of individuals versus age to estimate survival for the age-integer intervals (e.g. 1–2 yr, etc.). Survival probabilities (s) used were 0.3983, 0.6278, 0.6524, 0.6220, 0.5285 and 0.2711 for s_0, s_1, ... s_5, respectively. No one-year-old females were allowed to breed, and all females died after breeding in their final year. Pouch sex ratios (x) have been found to vary from 0.48 to 0.58 (proportion female; Guiler 1970b, 1978, Hughes 1982), so we used the average of these values (0.538) in the model. The proportion of breeding females (b) was set at 0.80 (Guiler 1978). Primiparous females (2 yr old) had an average of 2.0 pouch young (m_2), 3–5-yr-old females had 3.6 pouch young (m_3–m_5), and old females (6 yr old) had 2.0 pouch young (Guiler 1978). However, these rates produced a declining population ($\lambda = 0.9044$; see Results), so we updated the adult survival probability (ages 2–6) based on information from Pemberton (1990). Here, adult survival (s_1–s_4) was set at 0.82 (s_5 was kept from the original model). Pemberton (1990) also recorded a single female out of his sample bred at the age of one; therefore, we used the value of 0.061 for $b \times m_1$. For the updated model, all other pouch sex ratio and m_i were identical to those described above.

We chose to modify adult survival as a function of population size because there is recent evidence that the infectious agent responsible for DFTD is affected by devil population density (Mooney 2003b). Indeed, transmission rates, intensity and prevalence of infectious diseases are often higher in larger populations (Anderson and May 1986, Vandermeer and Goldberg 2003) because the spread of a directly transmitted infectious disease agent increases with the density of susceptible hosts (Lafferty and Gerber 2002). In high-density areas DFTD is believed to result in nearly 90% mortality in adults, whereas in low-density areas the mortality rate is closer to 40–50% (Mooney 2003b). Additionally, the rate of infection appears to be high because once symptoms are obvious, the tumours spread rapidly throughout the body and result in death within months (Mooney 2003b).

To incorporate this information into the model we constructed a simple negative feedback function on adult survival as a function of female population size. We used a simple 4-parameter logistic expression of the form:

$$s = y_0 + \frac{a}{1 + \left(\frac{N}{x_0}\right)^b} \quad (1)$$

to modify adult survival probability. Here, s = p[survival], N = population size and y_0, a, b and x_0 are constants.

Our aim was to construct a negative density feedback function on adult female survival that would result in a maximum equilibrium female population of 70 000. We assumed a starting population of half the maximum female population size (35 000) and maximum adult survival (e.g. s_2–s_5) of 0.82 (Pemberton 1990). Limits of the density function and the associated coefficients were found by setting the inflexion point in the logistic function at 70 000 individuals and maximum survival (0.82) at 10% of that population size (7000). To calculate the lower survival probability limit we trialled various values of s that would eventually result in a stable population of 70 000 females within the 200-yr projection interval. Coefficients for the logistic function were derived iteratively using a generalised reduced-gradient nonlinear optimisation algorithm (GRG2, Lasdon and Waren 1978). The logistic density-dependent feedback on adult survival that resulted in a stable female population of 70 000 individuals was:

$$s = 0.6234 + \frac{0.1968}{1 + \left(\frac{N}{70\,000}\right)^{2.9838}} \quad (2)$$

Thus, adult survival (s) varied between 0.6455 when the population was 140 000, and 0.82 when there were 7000 individuals.

Density-dependent survival with density-dependent disease

To re-create the situation of a rapidly spreading epidemic such as DFTD appearing within the population we defined a separate "disease" matrix using putative survival rates recorded during the most-recent disease epidemic (Mooney 2003a, b). Although few data on these mortality rates have been published, estimates of adult survival are thought to be reduced to 0.60 in low-density areas and 0.10 in high-density areas (Mooney 2003a, b). In the deterministic form of this model we used the average of these values (0.35) for adult survival (s_{3-6}). This aspatial model assumes that there is universal mixing and random mating (Jones et al.

2003) of the population and all individuals have the capacity to be infected.

To incorporate density-dependent survival into the disease matrix, we chose to define another logistic function of the same form described above. For this we set $x_0 =$ to half the maximum female population size (35 000), which describes the inflexion point of the logistic curve. The remaining constants were determined using the GRG2 algorithm. Here, we adjusted y_0, a and b by constraining the maximum survival value to 0.60 in low-density years and 0.10 in high-density years (Mooney 2003b). We termed low-density years as those when the population approached 5% of the maximum number of adult females ($0.05 \times 70\,000 = 3500$). High-density years were those where the population approached the maximum number of adult females. Thus, the density-dependent relationship for adult survival in the disease matrix was

$$s = 0.0376 + \frac{0.6530}{1 + \left(\frac{N}{35\,000}\right)^{3.0033}} \quad (3)$$

To model the effect of disease on population dynamics, we used Bierzychudek-style matrix selection (Bierzychudek 1982) where the matrix for maximum population growth rate and the disease matrix above were selected randomly through the projection. Here, the maximum-growth matrix was used to project the population vector until such time that a particular p[disease] (range: 0 to 1) would invoke the disease matrix. Once the disease matrix was invoked, the autocorrelation describing whether the following time step would also invoke the disease matrix was varied from -1 to 1 ([ac]). Thus, we accounted for both positive and negative temporal first-order auto-correlation in the model. This simple Markov chain essentially estimates a first-order autocorrelation between years once the epidemic appears. We then examined the range of p[disease] and [ac] together that resulted in population stability over time. Negative [ac] resulted in consistently positive population growth (see Results), so values of $[ac] < 0$ were not considered further. However, even incorporating a positive [ac] does not capture an important aspect of epidemic behaviour – once a disease occurs it is likely to persist for some time based on the rate of infection within the population. The spread of the disease is therefore also likely to be a function of population density (i.e. denser populations have a higher rate of infection and disease persistence over time-Vandermeer and Goldberg 2003, Davis et al. 2004).

To model the effect of density on the probability that disease epidemics re-occur once established, we used the same form of the 4-parameter logistic expression employed to model the negative feedback of density on adult survival. Again, we set $x_0 =$ to half the maximum female population size and determined the remaining constants using the GRG2 algorithm, but in this case we arbitrarily set the maximum probability of epidemic re-occurrence to 1.0 in high-density years (70 000 females), and 0 in low-density years (3500 females). Thus, the relationship takes the form

$$[ac] = 1.095 + \frac{-1.094}{1 + \left(\frac{N}{35\,000}\right)^{3.3950}} \quad (4)$$

For this model we set the initial auto-correlation at 0.5 and thereafter it was modified by the above logistic function for each time step. The frequency of epidemic occurrence (p[disease]) was drawn from a uniform random distribution from the range estimated above. These values were then incorporated into a 200-yr projection (1000 iterations) to estimate a mean population trend and associated 95% confidence intervals of female abundance over the duration of the projection. We calculated the expected minimum abundance (minimum abundance during a trajectory, averaged over all trajectories – McCarthy and Thompson 2001) as a proportion of the initial population size and calculated its 95% confidence interval over the 1000 iterations. We also applied a 10-yr running mean smoother to a sample run to examine the broad characteristics of population oscillations without minor inter-annual fluctuations.

To estimate the number of major cycles within the projected population trend over the 200-yr time series we applied a fast Fourier transform (FFT – Duhamel and Vetterli 1990) to the output of this model. The oscillations produced by the density feedback functions on adult survival and the temporal auto-correlation of epidemics lent themselves to a time-series analysis approach for discrete data. The absolute value of the Fourier coefficients were produced using the fft command in MATLAB and dividing by half the number of population estimates ($200/2 = 100$). An examination of the first 10 elements in the Fourier series using a semilog_{10} plot of the absolute coefficients versus the frequency distribution (Hz) highlights the major cycles in the original data. For this we excluded the first coefficient because this was almost always the highest value (i.e. frequency of one population cycle per 200 yr), and examined the highest and next-highest coefficients in the first 10 frequencies. These two values provided the most-common major frequencies of population cycling over the projection period. We repeated the above procedure for 1000 iterations of the base model to estimate a mean and confidence interval for the average duration of the major population cycles resulting from density-dependent disease epidemics.

Quasi-extinction threshold

We defined quasi-extinction (Q_t) as the number of years required to reduce the female population to ≤ 50 individuals. The formula used to calculate Q_t was derived by solving the Malthusian model of exponential growth for t (which is equivalent to Q_t):

$$Q_t = \frac{\log_e\left(\frac{\hat{N}_t}{\hat{N}_1}\right)}{\log_e \lambda} \quad (5)$$

where $\log_e \lambda = r$ is the instantaneous rate of exponential population change, $\hat{N}_t$ is the threshold population size (50 individuals) and $\hat{N}_t$ is initial population size (Otway et al. 2004).

Results

Density-dependent survival without disease

The deterministic model using constant survival rates from Guiler (1978) produced a rapidly declining population ($\lambda = 0.9044$). After 25 yr, this resulted in a population that was 8.1% of its original size. Time to quasi-extinction (≤ 50 females) was 65 yr. Updating the vital rates with those estimated by Pemberton (1990) resulted in an increasing population ($\lambda = 1.0409$) so that after 25 yr the population was 2.72 times larger than at time 0. The density-independent nature of this model results in a population that increases without end. The stable-age distribution gave 0.35 juveniles and 0.65 adults. Incorporating density-dependent adult survival into the model resulted in a stable population of 70 000 females after 71 yr (Fig. 2a), with adult survival reaching an asymptote = 0.7224 (Fig. 2c) at population stability (i.e. when $\log_e \lambda = r = 0$; Fig. 2c).

Density-dependent survival with density-dependent disease

A deterministic population projection applying the disease matrix alone resulted in rapid population decline ($\lambda = 0.6595$) and a halving of the population in only 1.7 yr. This rapid population decline results primarily from the low survival rates set for adults (0.35).

Using the full range of probabilities of disease occurrence (p[disease] from 0 to 1) and temporal autocorrelation ([ac] from -1 to 1), $\log_e \lambda$ ranged from -0.0793 to 0.0034 (Fig. 3). The range of these probabilities that resulted in population stability over the 200-yr projection ($\log_e \lambda$ ranging from $0 \pm 5 \times 10^{-4}$) were $0.0256 \leq$ p[disease] ≤ 0.1282, and $0.0000 \leq$ [ac] ≤ 0.8947 (Fig. 3). The replacement of random [ac] range with the more mechanistic density-dependent [ac] function resulted in the probability of quasi-extinction = 0 and in a stable population over the 200-yr projection (Fig. 4a). The expected minimum abundance expressed as a proportion of the sum of the initial population vector was 67.0% (95% confidence interval: 2.2–80.4%). An example run of the oscillation in female abundance without (Fig. 4b) and with (Fig. 4c) a 10-yr running-mean smoother is shown. After 1000 iterations the mean duration of the major population cycle was 146 yr (range 40–200 yr). An examination of the next-highest absolute Fourier coefficients yielded a mean duration of 77 yr (range 33–100 yr) per major population cycle.

Discussion

Historical and anecdotal information on the relative abundance of Tasmanian devils since European colonisation suggests a long-term fluctuating population with at least three major population maxima (Fig. 1; Guiler

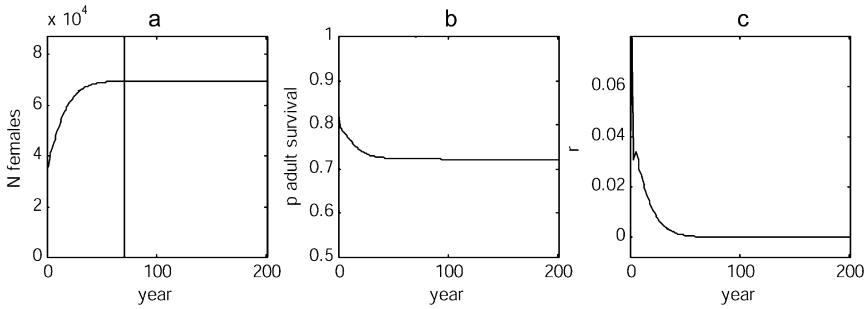

Fig. 2. a) Deterministic matrix projection of the female population of Tasmanian devils with density-dependent adult survival in the absence of disease. The negative density feedback function is set to produce a maximum population size of 70 000 females, with an initial population of 35 000 at stable age distribution. Stability (difference of <10 individuals between years) is achieved after 71 yr. b) Change in adult survival (s) with time under the density-dependent model. c) Change in the instantaneous population growth rate ($\log_e \lambda = r$) with time.

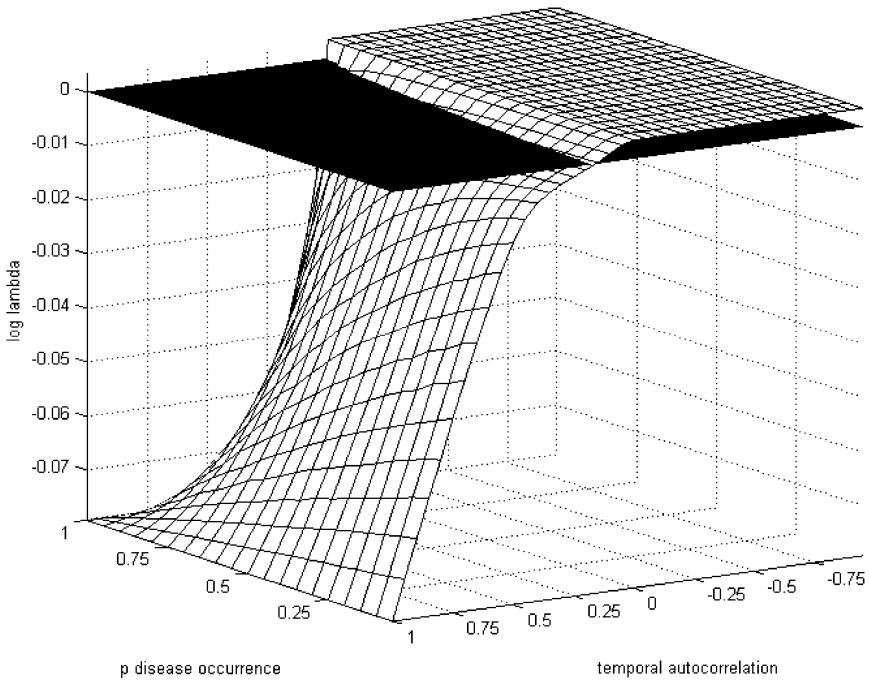

Fig. 3. Population growth rate ($\log_e\lambda = r$) as a function of the frequency of disease occurrence (p[disease] from 0 to 1) and first-order temporal autocorrelation ([ac] from -1 to 1) with density-dependent adult survival (s). Population stability is indicated by the black plane at $\log_e\lambda = r = 0$.

1992). Our disease model predictions agree well with this scenario, despite their simple form and basic assumptions. For instance, we were able to re-create a fluctuating population based solely on density-dependent incidence and severity of disease epidemics within an otherwise deterministic modelling framework, implying disease epidemics represent a valid mechanism for explaining the historical population variation of this endemic island species. There is also recent evidence to suggest that repeated periods of moderate reduction in population size have contributed to the lower relative genetic diversity observed in this dasyurid species (Jones et al. 2004).

The density feedback functions and Markov chain approach resulted in a population that never went extinct during the 200-yr time interval, because when populations are driven to low densities, individuals are less subject to exposure by the pathogen (Lafferty and Gerber 2002). This is likely to be an over-simplification of the population dynamics of this species, because the observed dynamics of any real population normally result from a combination of density-dependent and density-independent processes, the latter often manifesting at low population density in the form of demographic and environmental stochasticity and Allee effects (Strong 1986, Chapman et al. 2001). Other complicating factors that were either impossible to estimate or beyond the scope of our model include the effects of culling by agriculturists in the 19th century, the supplementation of food resources in the form of domestic carcasses once livestock farming became common in Tasmania, and the effects of direct mortality from increasing human populations (e.g. car strikes). Nonetheless, the large historical fluctuations and reports of disease-related mortalities suggest that epidemics may be the driving force in the long-term population trends of this dasyurid scavenger.

These results have important implications for the conservation of this species. Tasmanian devils are extinct in mainland Australia and now only occur on the main

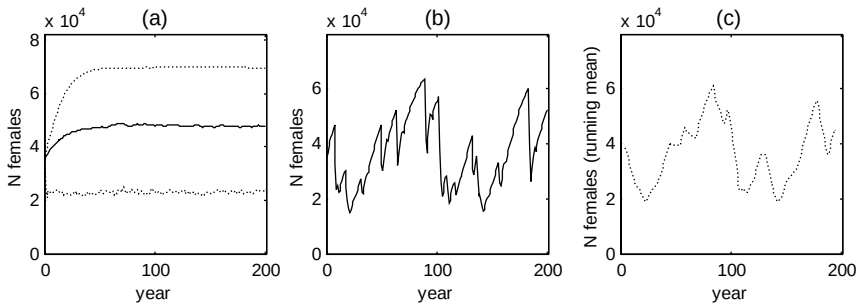

Fig. 4. a) Mean population abundance (solid line) $\pm$ 95% confidence intervals (dotted lines) of the projected population over 200 yr with density-dependent temporal autocorrelation [ac] and density-dependent adult survival (s), b) a sample projection of the population, and c) the sample projection with a 10-yr running-mean smoother.

island of Tasmania. Thus, the restricted distribution and high dispersal capacity of this species (Pemberton 1990, Jones et al. 2004) supports the idea that once epidemics take hold, they are likely to spread rapidly through the population and elicit large declines in numbers within only a few years. As indicated, it is unlikely that historical epidemics were caused by DFTD per se; however, density-related susceptibility to disease appears to drive population cycling. Indeed, captive and wild dasyurids, especially Tasmanian devils, are particularly susceptible to a wide variety of hyperplasias and neoplasias (Booth 1994), catastrophic age-related degeneration (Holz and Little 1995, Cockburn 1997, Oakwood et al. 2001) and many zoonotic parasites and pathogens (Gregory 1976, Obendorf et al. 1990, Georghiou et al. 1992, Andrews et al. 1993). This, in addition to the presence of the pathogen responsible for the latest outbreak of DFTD, support the hypothesis that devils experience frequent outbreaks of disease with the capacity to regulate long-term population size and invoke century-scale oscillations.

Interestingly, our models suggest that the presence of a disease epidemic itself is not necessarily a cause of grave concern, because infectious diseases are unlikely to be agents of extinction due to the density-dependent behaviour of transmission (sensu Dobson and May 1986, Rohani et al. 2003). However, this conclusion depends on the presence or absence of a possible reservoir host that could maintain the pathogen even at low devil densities (Lafferty and Gerber 2002). Additionally, if human contact with devils (e.g. via scientific and other conservation activities) inadvertently increases disease transmission rates or introduces the disease to unaffected sub-populations, the risk of extinction or increased disease persistence may increase. Perhaps a more worrisome outcome of rapid population declines due to disease is the contemporaneous addition of other stochastic factors at low population densities. Even though infectious diseases do not normally cause extinction, large reductions in host density due to a combination of disease and anthropogenic impacts may lead to a greater probability of reproductive failure via Allee effects and inbreeding depression, or the occurrence of random stochastic events such as drought that could act as the ultimate cause of extinction (Lafferty and Gerber 2002).

The recent (1998) introduction and establishment of the European red fox *Vulpes vulpes* in Tasmania (Anon. 2002) could expose devils to a new and efficient competitor and predator. There is some evidence that the presence of foxes may restrict the distribution of other dasyurid marsupials such as the spotted-tail quoll *Dasyurus maculatus* in mainland Australia (Catling and Burt 1997). Devils are Tasmania's main native scavenger, so an expanding fox population coinciding with a period when devil population density is low due to a disease epidemic may increase the probability of the latter's extinction (Mooney 2003b). Indeed, another introduced canid predator, the dingo, has been implicated as a major contributor to the extinction of the Tasmanian devil from the mainland during the late Holocene (Corbett 1995, Johnson and Wroe 2003), although it is arguable that the intensification of human settlement also contributed greatly (Johnson and Wroe 2003). Of course, the competitive interactions between these two species have yet to be examined directly, and are likely to depend on additional extrinsic factors such as environmental variability (e.g. drought years) and the degree of niche partitioning. Future models could consider the addition of density-independent stochastic factors that may reveal an increasing extinction risk when modelled within an epidemiological and competitive framework (Lafferty and Gerber 2002).

Our models are still constrained by the lack of an explicit spatial component incorporating movement of infected individuals from disease-source regions to unaffected areas. Many modelling approaches have been advanced in recent years to simulate the effects of individual dispersal, contact rates and landscape structure on the rate of disease transmission and advance (Barlow 1994, Smith et al. 1997, Rushton et al. 2000, Shirley et al. 2003, Eubank et al. 2004). However, without the corresponding data it would have been difficult, if not impossible, to incorporate this information into our models. Furthermore, disease transmission and susceptibility can vary between the sexes (Perkins et al. 2004) due to the negative effects of testosterone on male immunity, sexual dimorphism and disparity in the degree of aggressive interactions between sexes (e.g. male territorial/carrion disputes). Thus, sex-specific models of disease epidemiology that also incorporate the effects of group size and structure (Smith et al. 2001) on disease transmission may shed more light on the impact of DFTD in Tasmanian devils (Skorping and Helge Jensen 2004).

The interaction between epidemiology and population ecology is an informative avenue of research that can explain much of the variation in both the long-term trends of animal populations and the appearance, persistence and virulence of emerging pathogens (Rohani et al. 2003). Our models highlight how ignoring relatively simple density-influenced disease dynamics in population models could mislead conservation efforts for threatened and endemic species, and point to recurrent, density-linked disease as a potential major driver of long-term population fluctuations in this species. Additionally, the inter-play between immunodynamics, epidemiology and evolutionary biology ("phylodynamics" sensu Grenfell et al. 2004) can aid not only in the understanding of pathogen evolution, but also in host evolution. Thus, viewing high parasite and pathogen prevalence resulting from a scavenging existence

within a phylodynamic framework may help to explain the evolution of rapid degeneration, senescence and pathogen susceptibility in Tasmanian devils and possibly other marsupials.

Acknowledgements – We thank D. Bowman, R. Frankham, M. Hindell, A. Kabat, R. Rose, I. Robertson, K. Warren and P. Whitehead for helpful comments on earlier versions of this manuscript.

References

Anderson, R. M. and May, R. M. 1986. The invasion, persistence and spread of infectious diseases within animal and plant communities. – Philos. Trans. R. Soc. Lond. B 314: 533–568.

Andrews, J. R. H., Ainsworth, R. and Abernethy, D. 1993. *Trichinella pseudospiralis* in man. – Lancet 342: 298–299.

Anon. 2002. The fox in Tasmania. – Dept of Primary Industries, Water and Environment, Hobart, pp. 1–4.

Barlow, N. D. 1994. Predicting the effect of a novel vertebrate biocontrol agent: a model for viral-vectored immunocontraception of New Zealand possums. – J. Appl. Ecol. 31: 454–462.

Beissinger, S. R. and Westphal, M. I. 1998. On the use of demographic models of population viability in endangered species management. – J. Wildl. Manage. 62: 821–841.

Beltrami, E. and Carroll, T. O. 1994. Modelling the role of viral disease in recurrent phytoplankton blooms. – J. Math. Biol. 32: 857–863.

Bierzychudek, P. 1982. The demography of jack-in-the-pulpit, a forest perennial that changes sex. – Ecol. Monogr. 52: 335–351.

Booth, R. 1994. Medicine and husbandry – dasyurids, possums and bats. – Postgraduate Committee in Veterinary Science, Univ. of Sydney, Sydney, pp. 423–434.

Buchmann, O. L. K. and Guiler, E. R. 1977. Behaviour and ecology of the Tasmanian devil, *Sarcophilus harrisii*. – In: Stonehouse, B. and Gilmore, D. (eds), The biology of marsupials. Macmillan, pp. 155–168.

Bulte, E. H., Horan, R. D. and Shogran, J. F. 2003. Is the Tasmanian tiger extinct? A biological-economic re-evaluation. – Ecol. Econ. 45: 271–279.

Caswell, H. 2001. Matrix population models: construction, analysis, and interpretation, 2nd ed. – Sinauer.

Catling, P. C. and Burt, R. J. 1997. Studies of the ground-dwelling mammals of eucalypt forests in north-eastern New South Wales: the species, their abundance and distribution. – Wildl. Res. 24: 1–19.

Chapman, A. P. et al. 2001. Population viability analyses on a cycling population: a cautionary tale. – Biol. Conserv. 97: 61–69.

Chattopadhyay, J., Srinivasu, P. D. N. and Bairagi, N. 2003. Pelicans at risk in Salton Sea – an eco-epidemiological model – II. – Ecol. Modell. 167: 199–211.

Clausen, L. 2004. Sympathy for the devil. – Time Asia, 9 February 2004 No. 5: 48.

Cockburn, A. 1997. Living slow and dying young: senescence in marsupials. – In: Saunders, N. and Hinds, L. (eds), Marsupial biology: recent research, new perspectives. Univ. of New South Wales Press, pp. 163–171.

Corbett, L. 1995. The dingo in Australia and Asia. – Univ. of New South Wales Press.

Davidson, E. W. et al. 2003. Pathogenicity and transmission of chytridiomycosis in tiger salamanders (*Ambystoma tigrinum*). – Copeia 2003: 601–607.

Davis, S. et al. 2004. Predictive thresholds for plague in Kazakhstan. – Science 304: 736–738.

Dawson, L. 1982. Taxonomic status of fossil devils (*Sarcophilus*, Dasyuridae, Marsupialia) from late Quaternary eastern Australian localities. – In: Archer, M. (ed.), Carnivorous marsupials. Royal Zoological Society of New South Wales, pp. 517–525.

Dobson, A. P. and May, R. M. 1986. Patterns of invasions by pathogens and parasites. – In: Mooney, H. A. and Drake, J. A. (eds), Ecology of biological invasions of North American and Hawaii. Springer, pp. 58–76.

Duhamel, P. and Vetterli, M. 1990. Fast Fourier transforms: a tutorial review and a State of the Art. – Signal. Process. 19: 259–299.

Eubank, S. et al. 2004. Modelling disease outbreaks in realistic urban social networks. – Nature 429: 180–184.

Flynn, T. T. 1939. Note on the egg of the rare marsupial, *Sarcophilus*. – Proc. Irish Acad. 1938–39: 16–19.

Georghiou, P. R., Mollee, T. F. and Tilse, M. H. 1992. *Pasteurella multocida* infection after a Tasmanian devil bite. – Clin. Infect. Dis. 14: 1266–1267.

Ginzburg, L. R. and Jensen, C. X. J. 2004. Rules of thumb for judging ecological theories. – Trends Ecol. Evol. 19: 121–126.

Ginzburg, L. R., Ferson, S. and Akçakaya, H. R. 1990. Reconstructability of density dependence and the conservative estimate of extinction risk. – Conserv. Biol. 4: 63–70.

Gregory, G. G. 1976. Role of Tasmanian devil in epizootiology of ovine cysticercosis and echinococcosis. – Aust. Vet. J. 52: 471–476.

Grenfell, B. T. et al. 2004. Unifying the epidemiological and evolutionary dynamics of pathogens. – Science 303: 327–332.

Guiler, E. R. 1964. Tasmanian devils. – Aust. Nat. Hist. 14: 360–362.

Guiler, E. R. 1970a. Observations on the Tasmanian devil, *Sarcophilus harrisii* (Marsupialia: Dasyuridae). I. Numbers, home range, movements and food in two populations. – Aust. J. Zool. 18: 49–62.

Guiler, E. R. 1970b. Observations on the Tasmanian devil, *Sarcophilus harrisii* (Marsupialia: Dasyuridae). II. Reproduction, breeding, and growth of pouch young. – Aust. J. Zool. 18: 63–70.

Guiler, E. R. 1978. Observations on the Tasmanian devil, *Sarcophilus harrisii* (Dasyuridae: Marsupiala) at Granville Harbour, 1966–75. – Pap. Proc. R. Soc. Tas. 112: 161–188.

Guiler, E. R. 1982. Temporal and spatial distribution of the Tasmanian devil, *Sarcophilus harrisii* (Dasyuridae: Marsupialia). – Pap. Proc. R. Soc. Tas. 116: 153–163.

Guiler, E. R. 1992. The Tasmanian devil. – St. David's Park Publ.

Hadeler, K. P. and Freedman, H. I. 1989. Predator-prey populations with parasite infection. – J. Math. Biol. 27: 609–631.

Harvell, C. D. et al. 1999. Emerging marine diseases–climate links and anthropogenic factors. – Science 285: 1505–1510.

Holz, P. H. and Little, P. B. 1995. Degenerative leukoencephalopathy and myelopathy in dasyurids. – J. Wildl. Dis. 31: 509–513.

Hughes, R. L. 1982. Reproduction in the Tasmanian devil *Sarcophilus harrisii* (Dasyuridae. Marsupialia). – In: Archer, M. (ed.), Carnivorous marsupials. Royal Zoological Society of New South Wales, pp. 49–63.

Johnson, C. N. and Wroe, S. 2003. Causes of extinction of vertebrates during the Holocene of mainland Australia: arrival of the dingo, or human impact? – Holocene 13: 941–948.

Jones, M. 2003. Disease decimates the devil. – Wilderness News, Summer 2003, No. 164: 14–15.

Jones, M. E. 1998. The function of vigilance in sympatric marsupial carnivores: the eastern quoll and the Tasmanian devil. – Anim. Behav. 56: 1279–1284.

Jones, M. E. et al. 2003. Microsatellites for the Tasmanian devil (*Sarcophilus laniarius*). – Mol. Ecol. Not. 3: 277–279.

Jones, M. E. et al. 2004. Genetic diversity and population structure of Tasmanian devils, the largest marsupial carnivore. – Mol. Ecol. 13: 2197–2209.
Lafferty, K. D. and Gerber, L. R. 2002. Good medicine for conservation biology: the intersection of epidemiology and conservation theory. – Conserv. Biol. 16: 593–604.
Lasdon, L. S. and Waren, A. D. 1978. Generalized reduced gradient software for linearly and nonlinearly constrained problems. – In: Greenburg, H. J. (ed.), Design and implementation of optimization software. Sijthoff and Noordhoff, pp. 335–362.
Leslie, P. H. 1945. On the uses of matrices in certain population mathematics. – Biometrika 33: 183–212.
Loh, R. 2004. Devil facial tumour disease: devastating the Tasmanian icon. – In: Noods, R. (ed.), Proc. of the 2004 Conference of the Australian Association of Veterinary Conservation Biologists. Australian Association of Veterinary Conservation Biologists, Canberra, pp. 126–127.
McCarthy, M. A. and Thompson, C. 2001. Expected minimum population size as a measure of threat. – Anim. Conserv. 4: 351–355.
Mooney, N. 2003a. Deadly diseases. Tasmanian devils. – Brochure URL <http://www.dpiwe.tas.gov.au/inter.nsf/Attachments/LBUN-5RJ8NJ/$FILE/indesign%20devildis_1.PDF>.
Mooney, N. 2003b. Disease affecting Tasmanian devils. – URL <http://www.dpiwe.tas.gov.au/inter.nsf/WebPages/LBUN-5QF86G?open>.
Morris, W. F. and Doak, D. F. 2002. Quantitative conservation biology. – Sinauer.
Oakwood, M., Bradley, A. J. and Cockburn, A. 2001. Semelparity in a large marsupial. – Proc. R. Soc. Lond. Ser. B 268: 407–411.
Obendorf, D. L. et al. 1990. *Trichinella pseudospiralis* infection in Tasmanian wildlife. – Aust. Vet. J. 67: 108–110.
Otway, N. M., Bradshaw, C. J. A. and Harcourt, R. G. 2004. Estimating the rate of quasi-extinction of the Australian grey nurse shark (*Carcharias taurus*) population using deterministic age- and stage-classified models. – Biol. Conserv. 119: 341–350.
Pemberton, D. 1990. Social organisations in the Tasmanian devil *Sarcophilus harrisii*. – Ph.D. thesis, Univ. of Tasmania, Hobart.
Perkins, S. E. et al. 2004. Empirical evidence for key hosts in persistence of a tick-borne disease. – Int. J. Parasitol. 33: 909–917.
Pfister, C. A. and Stevens, F. R. 2003. Individual variation and environmental stochasticity: implications for matrix model predictions. – Ecology 84: 496–510.
Rohani, P. et al. 2003. Ecological interference between fatal diseases. – Nature 422: 885–888.
Rushton, S. P. et al. 2000. Modelling the spatial dynamics of parapoxvirus disease in red and grey squirrels: a possible cause of the decline in the red squirrel in the UK? – J. Appl. Ecol. 37: 997–1012.
Shirley, M. D. F. et al. 2003. Investigating the spatial dynamics of bovine tuberculosis in badger populations: evaluating an individual-based simulation model. – Ecol. Modell. 167: 139–157.
Skorping, A. and Helge Jensen, K. 2004. Disease dynamics: all caused by males? – Trends Ecol. Evol. 19: 219–220.
Smith, C. C., Cheeseman, C. L. and Clifton-Hadley, R. S. 1997. Modelling the control of bovine tuberculosis in badgers in England: culling and the release of lactating females. – J. Appl. Ecol. 34: 1375–1386.
Smith, C. C. et al. 2001. A model of bovine tuberculosis in the badger *Meles meles*: an evaluation of control strategies. – J. Appl. Ecol. 38: 509–519.
Strong, D. R. 1986. Density-vague population change. – Trends Ecol. Evol. 1: 39–42.
Vandermeer, J. H. and Goldberg, D. E. 2003. Population ecology, First principles. – Princeton Univ. Press.
Woodroffe, R. 1999. Managing disease threats to wild mammals. – Anim. Conserv. 2: 185–193.