Journal of Animal Ecology 2007
76, 926–936

The impact of disease on the survival and population growth rate of the Tasmanian devil

SHELLY LACHISH*, MENNA JONES†‡ and HAMISH MCCALLUM†

**School of Integrative Biology, University of Queensland. Brisbane, Qld. 4072, Australia; †School of Zoology, University of Tasmania, Hobart, Tasmania 7001, Australia; and ‡School of Botany and Zoology, Australian National University. Canberra, ACT 0200, Australia*

Summary

1. We investigated the impact of a recently emerged disease, Devil Facial Tumour Disease (DFTD), on the survival and population growth rate of a population of Tasmanian devils, *Sarcophilus harrisii*, on the Freycinet Peninsula in eastern Tasmania.
2. Cormack–Jolly–Seber and multistate mark–recapture models were employed to investigate the impact of DFTD on age- and sex-specific apparent survival and transition rates. Disease impact on population growth rate was investigated using reverse-time mark–recapture models.
3. The arrival of DFTD triggered an immediate and steady decline in apparent survival rates of adults and subadults, the rate of which was predicted well by the increase in disease prevalence in the population over time.
4. Transitions from healthy to diseased state increased with disease prevalence suggesting that the force of infection in the population is increasing and that the epidemic is not subsiding.
5. The arrival of DFTD coincided with a marked, ongoing decline in the population growth rate of the previously stable population, which to date has not been offset by population compensatory responses.

Key-words: devil facial tumour disease, mark–recapture analysis, *Sarcophilus harrisii*, Tasmanian devil, wildlife disease.

Journal of Animal Ecology (2007) **76**, 926–936
doi: 10.1111/j.1365-2656.2007.01272.x

Introduction

Devil Facial Tumour Disease (DFTD) is a recently emerged disease that is now widespread and represents a serious threat to the Tasmanian devil, *Sarcophilus harrisii*, the world's largest extant marsupial carnivore (Hawkins *et al*. 2006). A cancerous disease, DFTD appears to be consistently fatal with no evidence of recovery or natural immunity yet observed (Hawkins *et al*. 2006). Investigations of the aetiology of this disease are still in their infancy and neither the latency period nor the exact transmission mode is known (Loh *et al*. 2006). Recent cytogenetic work has shown that tumours in different devils have identical chromosomal rearrangements, suggesting that DFTD is transmitted from animal to animal by direct transmission of a 'rogue' cell line, probably during social interactions (Pearse & Swift 2006). Only one other known tumour, the sexually transmitted Canine Transmissible Venereal Sarcoma (CTVS) of dogs, is transmitted in a similar manner (Das & Das 2000).

Currently recorded in over 51% of the island of Tasmania, DFTD has resulted in a decline of 41% in the wild devil population, with local declines of up to 83% having been recorded (Hawkins *et al*. 2006). The loss of a vertebrate predator can have wide-ranging negative effects on the rest of the ecosystem (Sih *et al*. 1985; Schmitz, Hamback & Beckerman 2000). Decline in devil numbers may allow the meso-predator release of feral cat populations and the establishment of foxes (Bloomfield, Mooney & Emms 2005; Saunders *et al*. 2006). It is imperative therefore to establish effective management regimes to mitigate the impacts of this disease for this iconic species.

In the absence of a vaccine or treatment, options for managing the disease in wild populations are limited. One of the few possibilities available is removing infected individuals in an attempt to reduce transmission to susceptible animals (McCallum & Jones 2006).

Correspondence: Shelly Lachish, School of Integrative Biology, University of Queensland, St Lucia, Brisbane 4072, Australia. Tel.: +617 3365 2769. Fax: +617 3365 1655. E-mail: s.lachish@uq.edu.au

Monitoring disease dynamics in 'removal' vs. 'control' areas could provide information on how transmission rates vary with population density (Bradshaw, McMahon & Brook 2006). While ideal, evaluating this strategy experimentally in replicated control and treatment populations is logistically and financially demanding. In addition, the benefit to the population of reducing the force of infection needs to be balanced against the reproductive contribution of the individuals removed. Demographic models provide an inexpensive efficient means of assessing the likely outcome of management actions and are therefore necessary to determine the utility of this strategy. These models require field estimates of the vital rates of infected and uninfected animals, together with the force of infection as a function of disease prevalence.

Detecting population impacts of pathogens necessitates either direct monitoring of traits of infected vs. uninfected individuals in diseased populations or, ideally, determining changes in demographic parameters following disease outbreaks in long-term study populations, where data are available from before the start of the epidemic (McCallum 1994). Long-term studies of disease in wild populations are rare (Mutze *et al*. 2002; Williams *et al*. 2002b) with most investigations of disease impacts commonly undertaken retrospectively or opportunistically so that the pathogen is either already endemic or declining in the system before investigation begins (Telfer *et al*. 2002; Hall *et al*. 2006). Several recent long-term studies, however, where disease was detected part-way through the study, have detected negative impacts of pathogens on survival rates and population abundance (Arthur, Ramsey & Efford 2004; Faustino *et al*. 2004; Wilmers *et al*. 2006).

The detection of DFTD in 2001 in a population of Tasmanian devils that had been monitored since 1999 provided a valuable opportunity to examine the impacts of this debilitating disease on demographic parameters. Here we report on the impact of DFTD within an intensively trapped population of individually marked devils on the east coast of Tasmania. Using both traditional mark–recapture models and more recently developed multistate mark–recapture models we investigate the impact of DFTD on age- and sex-specific apparent survival rates and also examine the pattern of variation in infection rates (transition rates from healthy to diseased states) within the population in relation to disease prevalence. Finally, we employ reverse-time mark–recapture models to investigate the effect of DFTD on population growth rate.

Methods

STUDY AREA, TRAPPING METHOD AND DATA COLLECTION

Tasmanian devils were trapped within a 160 km^2 site comprising the entire Freycinet Peninsula (42°03′53″S, 148°17′14″E, Fig. 1) on the east coast of Tasmania.

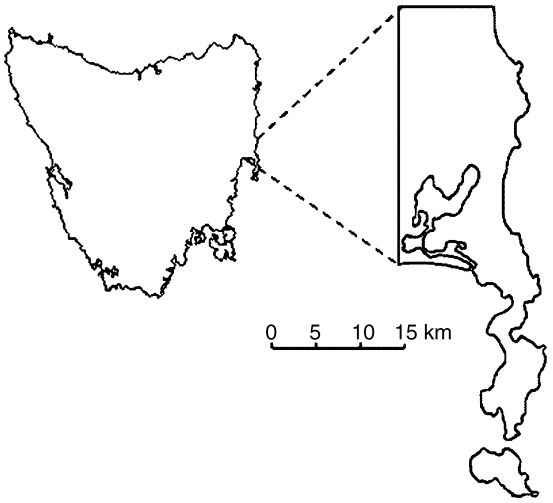

Fig. 1. Map of Tasmania showing location of the Freycinet peninsula study site.

The Peninsula is dominated by rugged granite peaks rising to 600 m. The vegetation consists of dry sclerophyll forests and coastal heath, small areas of wet eucalypt forest and tea-tree swamp with *Eucalyptus amygdalina*, *E. globulus*, *E. ovata*, *Kunzea ambigua*, *Melaleuca* and *Epacris* spp., the dominant species. Land tenure is predominantly national park and Crown Land conservation area, but also includes farmland (sheep/cattle) and private rural/bush block residential land.

The site has been trapped as four contiguous trapping regions (Bicheno, 41 km^2; Northern, 50 km^2; Southern, 40 km^2; Peninsula, 26 km^2) up to four times a year since 1999. Trapping periods were typically seven nights in each region with trap numbers varying slightly (mean trap-nights per 4-week trip = 657 ± 10·7). Traps were placed at strategic sites an average of 2 km apart that were most likely to catch devils (crossroads, creek/road junctions, alongside creeks). In 2004 the fine wire-mesh cage traps were replaced by a newly designed PVC pipe trap (diameter 315 mm × length 875 mm) to reduce the risk of tooth damage and for ease of cleaning to minimize disease transmission. Traps were baited with a variety of meats, and checked daily. Accidental disease transmission was minimized by following a strict protocol, developed by veterinary practitioners involving the sterilization of all equipment and traps with Virkon™ (Antec International, Du Pont Animal Health Solutions, Sudburg, UK), an antibacterial, antiviral, antifungal, DNA denaturing solution and burning sacks and any remaining bait and faeces.

The data set for the analyses in this paper consists of capture histories from 1999 to 2006, obtained in June/July each year, as this was the only consistently sampled period every year. Data from this 4-week winter trapping period were pooled into a single capture occasion per individual. Sampling at this time of year minimized the likelihood of including dispersing juveniles in the data set (the natal dispersal phase occurs post-weaning from December through to March/April; Guiler 1970) and thus introducing bias into the survival estimates.

IDENTIFICATION, AGEING AND DISEASE DETECTION IN TRAPPED DEVILS

Animals were identified via unique ear tattoos (from 1999 to 2003) or via unique microchip transponders (Allflex®, Palmerston North, New Zealand) (from 2004 on). Most individuals in the data set were of known age having been first captured as juveniles. Devils first captured as adults were aged on the basis of skeletal measurements, molar eruption, tooth wear indices and canine overeruption (distance from the dentine-enamel junction to the gum) (Pemberton 1990; M. Jones unpubl. data). This method is precise for ageing devils to 2 years of age. In this study, survival rates were estimated for two age classes only: 1 year olds (subadults) and 2+ year olds (adults). Juveniles and pouch young were not included in analyses.

As there is no pre-clinical diagnostic test for DFTD, detection of the disease is only possible by the visible presentation of tumours. All manifestations of the disease involve the appearance of facial tumours, which are distinctive, often circular and large, ulcerated lesions that occur on the head, neck or inside the mouth. Devils were thoroughly examined both externally and inside the mouth. Although devils were not initially examined with the intensity now used to check for early tumours (initial examination of the mouth was used only for ageing purposes), the disease produces such large, open lesions that even a cursory glance at a trapped animal would raise suspicion of the disease. The health status of an individual was scored on an index from 1 (no apparent DFTD) through 2 and 3 (wounds, inflammations or other irregularities present) to 4 (characteristic DFTD tumours present). Only individuals with definite cases of DFTD (those that scored 4) were included as diseased in these analyses.

MARK–RECAPTURE MODELLING APPROACH

Capture–mark–recapture (CMR) methods were used to estimate age-specific survival rates and to test specific hypotheses of survival patterns and disease infection rates. The standard Cormack–Jolly–Seber (CJS) framework was employed to estimate average survival probabilities of the population as a whole (determined by the relative proportion of diseased vs. healthy individuals and their respective survival rates). Subsequently, a multistate mark–recapture framework was used to estimate 'state-dependent' survival probabilities for diseased vs. healthy individuals as well as to estimate transition probabilities between states. Data were analysed using the program MARK (White & Burnham 1999).

CJS MODELLING FRAMEWORK

The CJS model makes the following assumptions. First, the fates of individual animals and individuals in different cohorts are independent. As devils are solitary animals they are unlikely to have correlated survival rates (Pemberton 1990). Second, marks are not lost or overlooked. Tag loss is negligible with tattoos. Microchip failure/loss has also been negligible in this study as individuals in this study site have all been genotyped with no single genotype has ever been assigned to more than one microchip number (M. Jones unpubl. data). Third, there is no mortality during the trapping period, which is instantaneous relative to the length of the survival interval. In this study, some mortality may have occurred during the trapping period, particularly in diseased animals. As each individual is simply recorded as captured or not captured during the 4-week period, any mortality would contribute to heterogeneity in capture probability. O'Brien, Robert & Tiandry (2005) found that mortality within trapping periods does not significantly bias survival estimates if recapture rates are high (> 0·2). Finally, it is assumed that survival and capture probabilities are equivalent across individuals or individuals within groups, which is normally tested with a goodness of fit, GOF, analysis. The parametric bootstrap procedure available in MARK (White & Burnham 1999) was used to test the GOF of the CJS and to calculate the variance inflation factor $\hat{c}$ (observed deviance/mean deviance from bootstrap replicates), which indicates the degree of overdispersion in the data.

Individual capture histories were grouped by sex and 'marking group' (either 'marked as a subadult' or 'marked as an adult'). We investigated the effect of sex, marking group, time and disease on variation in recapture and apparent survival rates (where apparent survival reflects the probability of surviving and remaining on the trapping grid). We also investigated the effect of trapping effort (total trap-nights) and trap type (wire vs. pipe) on recapture rates. Disease was modelled as the population disease prevalence; the proportion of diseased individuals captured in each sampling period, set to zero for the first two intervals. The population was assumed to be disease free for the first two survival intervals (1999–2000, 2000–01) as no diseased devils were captured during any of the monitoring trips prior to June 2001 (including November 2000, January 2001, April 2001). Survival rates were modelled either as time-dependent, a linear function of disease prevalence or constant over the study period as we had no a priori belief as to how disease would impact on survival rates. To reduce the number of models in the candidate set, modelling of the survival rate of the adults marked-as-adults group was limited to either time- or prevalence-dependent, after a preliminary look at the parameter estimates for this group showed a clear trend over time. Model notation is explained in Table 1.

MULTISTATE MODELLING FRAMEWORK

For the multistate models, capture histories were grouped by sex, and captures for each individual were reassigned to one of three states: 1 = subadults (all

Table 1. Subscripts used in model notation. These subscripts denote the main effects and model structure used in modelling apparent survival rate (ϕ), recapture rate (p), transition rate (ψ) and population growth rate (λ)

Subscript	Description	Parameter type
Sx	Sex effect	ϕ, p
M	Marking group effect	
s	Subadults of group 1 (marked as subadults)	ϕ
a1	Adults of group 1 (marked as subadults)	ϕ
a2	Adults of group 2 (marked as adults)	ϕ
S	Healthy subadults	ϕ
H	Healthy adults	ϕ
D	Diseased adults	ϕ
S > D	Transitions from healthy subadults to diseased adults	ψ
H > D	Transitions from healthy adults to diseased adults	ψ
P	Disease effect (parameter varies with disease prevalence)	ϕ, p, ψ, λ
TE	Trapping effort (total trap/nights)	p
TT	Trap type (either wire mesh or PVC pipe)	p
t	Full time dependence	ϕ, p, ψ, λ
•	Constant rate	ϕ, p, ψ, λ

healthy), 2 = healthy adults or 3 = diseased adults. Subadults must age, so were allowed only one time interval in stratum 1 and were forced to progress either to healthy adults or diseased adults (transitions from $1 \rightarrow 1$ fixed to zero and recapture rate for stratum 1 fixed to zero). Conversely, adults must remain adults (transitions from $2 \rightarrow 1$ and $3 \rightarrow 1$ fixed to zero). Healthy adults could remain healthy adults and diseased adults could remain diseased adults. Diseased adults could not become healthy adults (recovery from DFTD has never been observed) so transitions from $3 \rightarrow 2$ were fixed to zero and recapture rate for stratum 3 was fixed to one (to improve parameter identity issues). Our global model was a reduced parameter model due to sparseness in the data set (we only recorded 36 transitions to diseased states over five intervals). The goodness-of-fit of this reduced parameter model was tested using the median $\hat{c}$ approach (White & Burnham 1999). Patterns of variation in apparent survival rates and transition rates between states were examined in relation to time and disease prevalence while the effect of sex was retained in recapture rate models only (based on the results of the CJS modelling above). In addition to the assumptions of the CJS model, multistate models assume that the probability of an individual making a transition between time i and $i + 1$ depends only on its state at time i (Williams, Conroy & Nichols 2002a). Our current knowledge of the dynamics of this disease (i.e. no observed recovery from DFTD infection and no apparent signs of immunity or resistance to infection by any individual) suggest that this assumption may be a good approximation of the real-world infection process for this disease.

MODEL RANKING PROCESS

Ranking of models in the candidate set was based on small sample size corrected Akaike Information Criteria (AICc, Anderson & Burnham 2002). The relative likelihood of each model in a candidate set was estimated with normalized AICc weights (w_i, or the index of relative plausibility) with the ratio of w_i between any two models indicating the relative proportional support between them. A three-step model ranking process was employed. First, recapture rates were modelled with survival fully parameterized. Then survival and transition rates were modelled using the single best supported recapture model identified in step 1 (based on AICc weight). Finally, the best supported survival and transition rate models were each re-run with all the best supported recapture rate models identified in step 1. Robust parameter estimates were obtained through model averaging, which accounts for model selection uncertainty (Burnham & Anderson 2002).

To examine disease impacts in light of the temporal pattern of infection in the population, changes in the age of infected individuals over time were analysed using a linear regression. The difference between the age of the infected individual and the average age of all other 'at-risk' individuals in the population (all the healthy individuals in the population at the previous time period) was used as the dependent variable to control for concurrent changes in the average age of the whole population.

ESTIMATION OF POPULATION GROWTH RATE (λ) AND POPULATION SIZE

The impact of disease on population rate of change was assessed using reverse-time CMR methods (Pradel models) to estimate the finite rate of change of the adult population (Pradel 1996; Nichols & Hines 2002). In this approach, λ represents the realized population growth rate (the observed change in population size between two time periods) and does not assume constant, ergodic conditions or a stable age distribution. As Pradel models do not allow for age effects, these models were used with a data set consisting of all the adult captures of the individuals in our study. The goodness of fit of the global model (with time variation in all parameters) was tested using the parametric bootstrap method (Cooch & White 2001). Model selection proceeded by modelling recapture rates, then applying constraints to λ, with apparent survival retained as time-dependent in all models. Variation in λ parameters was examined in relation to time and disease prevalence or a constant rate. Model selection proceeded as outlined above with Akaike Information Criteria adjusted for overdispersion (QAICc). Population size each winter was estimated using the POPAN open population models in MARK (White & Burnham 1999), without distinguishing between sexes. Yearly population estimates were derived from models with a constant recapture rate and time-varying survival rates and were obtained for the population as a whole and for the adult component of the population. Young of the year were not included in population estimates.

Table 2. Summary results of the age-structured CJS analysis of apparent survival and recapture rates. Models highlighted in bold are the best supported models in the candidate set. See Table 1 for model notation

	ϕ	p	Delta AICc	AICc weight	No. Par	Deviance
1. Initial modelling of recapture rates	$Sx * M(s_t/a1_t/a2_t)$	Sx * t	9·46	0·00	49	68·08
		Sx + t	2·80	0·09	43	75·49
		TE	3·27	0·07	38	87·46
		P	4·06	0·05	38	88·25
		TT	4·22	0·04	39	83·59
		t	2·59	0·11	42	77·59
		Sx	**0·15**	**0·36**	**37**	**86·61**
		•	**0·00**	**0·39**	**36**	**88·73**
2. Modelling the effect of sex	$Sx * M(s_t/a1_t/a2_t)$	•	20·68	0·00	36	88·73
	$Sx * M * P(s_P/a1_P/a2_P)$		16·57	0·00	14	132·58
	$Sx * M(s_t/a1_t/a2_t)$ [no S * M * t or S * t]		3·32	0·14	25	95·81
	$Sx * M(s_t/a1_t/a2_t)$ [no S * M * t, S * t, S * a]		3·36	0·14	23	100·19
	$M(s_t/a1_t/a2_t)$		**0·00**	**0·72**	**21**	**101·14**
3. Modelling the effect of marking group	$M(s_t/a1_t/a2_t)$	•	**0·00**	**0·99**	**21**	**101·14**
	$s_t/a1_t = a2_t$		8·58	0·01	15	122·49
4. Modelling the effect of disease*	$s_P/a1_t/a2_P$	•	**0·00**	**0·35**	**11**	**113·45**
	$s./a1_t/a2_P$		**0·10**	**0·33**	**10**	**115·62**
	$s_t/a1_t/a2_P$		**0·66**	**0·25**	**16**	**103·63**
5. Re-employ alternate recapture model (p.) from stage 1 with the best supported survival models from stage 4	$s_P/a1_t/a2_P$	•	0·51	0·17	11	113·45
		Sx	0·00	0·22	12	110·86
	$s./a1_t/a2_P$	•	0·61	0·16	10	115·62
		Sx	0·34	0·18	11	113·29
	$s_t/a1_t/a2_P$	•	1·17	0·12	16	103·63
		Sx	1·17	0·12	17	101·51

*All combinations of s ($_t$ or $_P$ or .) & a1 ($_t$ or $_P$ or .) & a2 ($_t$ or $_P$) were run. Only the best supported of those 12 possible combinations are shown here.

Results

We captured 448 devils (206 males, 242 females) during the study, two-thirds of which were first captured on the study site as subadults. DFTD was first detected in June 2001 at the northernmost end of the peninsula, with diseased individuals captured sequentially southward in subsequent years (Fig. 4 in Hawkins *et al*. 2006). There was no difference in age structures between the four trapping regions before disease (log-linear analysis, Δ Dev = 0·380 P = 0·83). A total of 36 diseased (score of 4) individuals were captured during the study. No animal scoring a 4 ever regressed to a lower score. In this study, 5% of females bred as 1 year olds (15 of 242). It is possible that the survival of these females in this year will be more similar to that of other adults due to the cost of reproduction incurred. Classifying these individuals as adults, however, did not alter the results presented below and these individuals were retained in the subadult group.

CJS MODELLING

The parametric bootstrap procedure suggested an acceptable fit of the global model and indicated no overdispersion in the data (1000 bootstrap replicates, P = 0·657; $\hat{c}$ = 1·065). The CJS model, however, does not take into account age-specific variation in survival, which we expected in our data set. We compared the fit of the CJS model with a model that kept recapture rate as sex- and time-dependent (preliminary analyses showed no differences in recapture rates between the marking groups or between age classes) and allowed for full age × marking group × time effects in survival rates. This global model was better supported by the data (AICc weight = 0·92) and became our starting point for fitting subsequent models. A GOF test (1000 bootstrap replicates) showed no indication of lack of fit for this model or overdispersion in the data (P = 0·526; $\hat{c}$ = 1·008).

ENCOUNTER RATE

The model selection procedure showed that constant and sex-dependent recapture rate models were equally well supported by the data (Table 2). There was little support for models in which recapture rates varied with trapping effort or the type of type used. A model that allowed recapture rates to vary with disease prevalence was also not supported by the data (Table 2). Overall recapture rate was high and fairly uniform throughout the study (0·79 ± 0·044, CI = 0·692–0·862) with males having a slightly higher recapture rate than females, though the confidence intervals overlapped (males; 0·824 95% CI = 0·66–0·92 females; 0·767 95% CI = 0·65–0·86 and CI for the logit transformed beta estimate of the effect of sex included zero, 95% CI = –0·35 to 2·07).

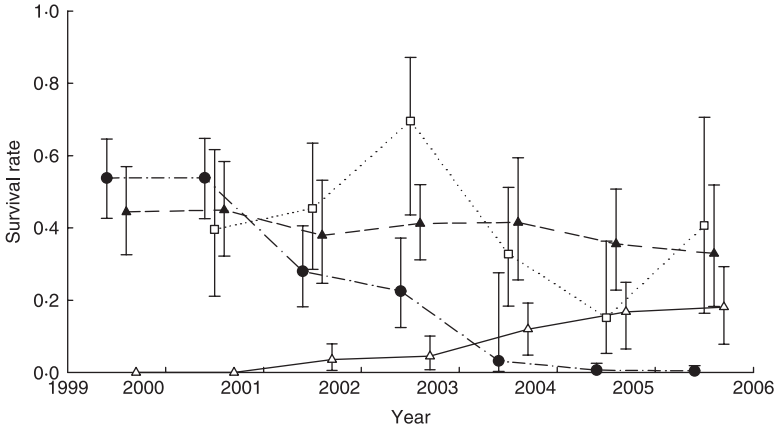

Fig. 2. Model averaged estimates of yearly apparent survival rate of individuals from age-structured Cormack–Jolly–Seber analysis. Estimates are mean ± 95% CI. Filled triangles (▲) and dashed line show subadult survival rate, open squares (□) and dotted line show survival rate of adults marked as subadults and filled circles (●) and dot-dashed line show survival rate of adults marked as adults. Open triangles (△) and solid line show Devil Facial Tumour Disease prevalence at each sampling period (mean ± 95% CI).

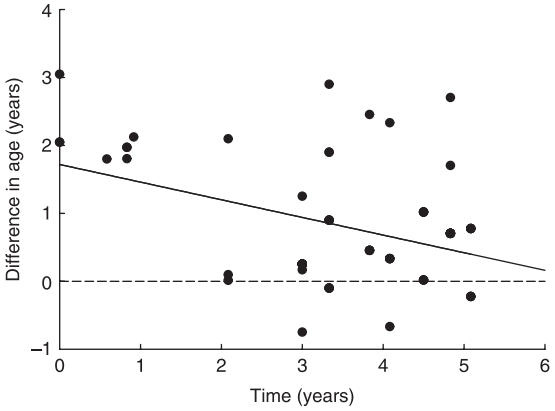

Fig. 3. The difference between the age of diseased individuals (in years) and the average age of healthy individuals in the population (in years) as a function of time since the arrival of Devil Facial Tumour Disease into the population. The solid line is a linear regression. $R^2 = 0{\cdot}126$.

APPARENT SURVIVAL AT THE POPULATION LEVEL

All of the best supported survival models in the candidate set included an effect of marking group and an effect of disease but no sex effect (Table 2). Survival rates of males and females did not differ with time or between age groups. In addition, a model that included an interaction between sex and disease prevalence (i.e. survival rates of males and females impacted by disease in different ways) was not supported by the data (Table 2).

There was strong support for the effect of marking group on apparent survival rates. Immediately following disease arrival the survival rate of adults marked-as-adults began to decline and continued to decline virtually to zero at a rate predicted well by disease prevalence in the population (Table 2, Fig. 2). In contrast, the survival rate of adults marked-as-subadults fluctuated over time, was not related to disease prevalence and appeared to be highly variable (Fig. 2, Table 2). This result suggests that older individuals were the first to be impacted by DFTD, because adults of the marked-as-adults group are, on average, older than the adults of the marked-as-subadults group. Analysis of the age of diseased individuals relative to the average age of healthy individuals shows a significant decrease in the age of diseased devils over time ($F = 13{\cdot}45$, d.f. = 1, 67, $P = 0{\cdot}0004$). Diseased individuals were initially older than the population average (95% confidence interval, 1·18–2·25 years) but after 5 years, this age difference declined (95% confidence interval, 0·10–0·69 years) (Fig. 3).

Apparent survival of subadults, meanwhile, was relatively constant throughout the study period, declining slightly in the latter time periods (Fig. 2). The three models for the apparent survival of subadults (constant over time or varying either with disease prevalence or with time) received equal support (Table 2). While this limits our ability to make inferences, it suggests that the population-level impact of DFTD on apparent survival of subadults has, to date, been comparatively less than that detected for adults.

MULTISTATE CAPTURE–RECAPTURE MODELS

There was no indication of lack of fit or overdispersion in the data for the global multistate model ($\hat{c} = 1{\cdot}007$). The apparent survival of diseased adults was estimated as zero, as no diseased devil was ever recaptured. The apparent survival of both healthy adults and healthy subadults meanwhile was best explained by changes in disease prevalence (Table 3). The survival rate of both groups declined immediately following disease arrival and continued to decline as disease prevalence increased over time (Fig. 4). The relationship between subadult survival rate and disease is evidently stronger in this analysis.

Although the decrease in survival rate was slightly more pronounced for adults than for subadults, the two trajectories were similar and the confidence intervals of the parameter estimates overlapped. In fact, a model with these two rates identical was equally well supported by the data (shown in italics in Table 3). Parameter estimates from this model show that apparent yearly survival rate of healthy individuals in the population declined significantly post disease (confidence interval for the logit transformed beta parameter estimate for the effect of DFTD on survival did not include zero; –4·805, 95% CI –7·94, to –2·28). For clarity and due to the age effects we detected in the age-structured analysis above, we continued modelling using the state-dependent survival rate model (shown in bold in Table 3). Incorporating both alternative survival models in further modelling did not alter the results described below.

Table 3. Summary results of the multistate analysis of apparent survival rates, transition rates and recapture rates. Models highlighted in bold are the best supported models in the candidate set. The model in italics has identical survival rates for both states (see text for details). See Table 1 for model notation

	ϕ	p	ψ	Delta AICc	AICc weight	No. Par	Deviance
1. Initial recapture rate modelling	$S_t/H_t/D.$	Sx	$S > D_t/H > D_t$	**0·00**	**0·57**	**29**	**173·03**
		•		**0·60**	**0·43**	**28**	**175·84**
2. Modelling effect of disease on survival rates	$S_t/H_t/D.$	Sx	$S > D_t/H > D_t$	10·15	0·00	29	175·84
	$S./H_t/D.$			8·98	0·01	23	184·98
	$S_P/H_t/D.$			5·83	0·04	23	181·82
	$S_t/H_P/D.$			5·19	0·06	23	181·20
	$S./H_P/D.$			2·97	0·16	17	191·83
	$S_P/H_P/D.$			**0·00**	**0·73**	**17**	**188·86**
3. Modelling effect of disease on transition rates (with the best supported survival model from stage 2)	$S_P/H_P/D.$	Sx	$S > D_t/H > D_t$	8·71	0·00	17	188·86
			$S > D_P/H > D_t$	6·10	0·02	12	196·76
			$S > D_t/H > D.$	2·64	0·10	12	193·30
			$S > D_t/H > D_P$	2·43	0·12	12	193·10
			$S > D_P/H > D.$	**0·21**	**0·36**	**7**	**201·21**
			$S > D_P/H > D_P$	**0·00**	**0·40**	**7**	**200·99**
	$S_P = H_P/D.$	*Sx*	$S > D_P/H > D_P$	*0·68*	*0·27*	*6*	*202·36*
4. Re-employ alternate recapture rate model (p.) from stage 1 with the best supported models from stage 3	$S_P/H_P/D.$	Sx	$S > D_P/H > D.$	0·21	0·24	7	201·21
		•		1·13	0·15	6	204·17
	$S_P/H_P/D.$	Sx	$S > D_P/H > D_P$	0·00	0·27	7	200·99
		•		0·91	0·17	6	203·96

Table 4. Summary results of the Pradel temporal symmetry analysis of realized population growth rates. Models highlighted in bold are the best supported models in the candidate set. See Table 1 for model notation

	ϕ	p	λ	Delta QAICc	QAICc weight	No. Par	Deviance
1. Initial modelling of recapture rate	t	t	t	5·04	0·044	22	97·55
		•		**0·58**	**0·409**	**15**	**108·49**
		Sx		**0·00**	**0·547**	**16**	**105·71**
2. Modelling effect of disease on realized population growth	t	Sx	t	3·18	0·15	16	105·71
			•	14·58	0·00	10	129·92
			P	**0·00**	**0·72**	**11**	**113·23**
3. Re-employ the alternate recapture rate [p(.)] from stage 1 with the best supported model from stage 2	t	Sx	P	**0·00**	**0·43**	**11**	**113·23**
		•		**0·13**	**0·41**	**10**	**115·47**

DISEASE STATE TRANSITIONS

The best supported models in the candidate set show that transitions of subadults to diseased adults are best explained by disease prevalence, while transitions from healthy to diseased adults have been either constant over time or likewise prevalence related (Table 3). Transitions of healthy adults to diseased adults remained relatively constant, whereas transitions of healthy subadults to diseased adults increased almost exponentially (Fig. 4). Thus, while initially all infections occurred in adults, the majority of new infections appear to be occurring in recently matured adults.

POPULATION GROWTH RATE

A GOF test on the global model showed no lack of fit ($P = 0{\cdot}13$) and slight over dispersion ($\hat{c} = 1{\cdot}16$). Realized population growth rate in this study was calculated from a subset of the data consisting of all the adult captures of individuals in our study ($n = 215$). The estimates of λ reported here, hence refer only to the adult segment of our population. The two best models in the candidate set included variation in λ associated with population disease prevalence (Table 4). Prior to disease arrival the population appears to have been stable (confidence intervals for the first two parameters include 1·0), with an immediate decline in growth rate following the onset of disease (Table 5). The estimates for λ suggest that the adult segment of the population declined immediately following disease arrival and is now approximately halving annually. Indeed, yearly estimates of the adult population size show a clear decline in numbers of adults from 2001 (Fig. 5). Population size estimates for the population as a whole show a similar pattern, though the decline began 1 year later (Fig. 5).

Discussion

IMPACT OF DISEASE ON DEVIL SURVIVAL RATES

The arrival of DFTD at the Freycinet peninsula in June 2001 triggered an immediate and steady decline in

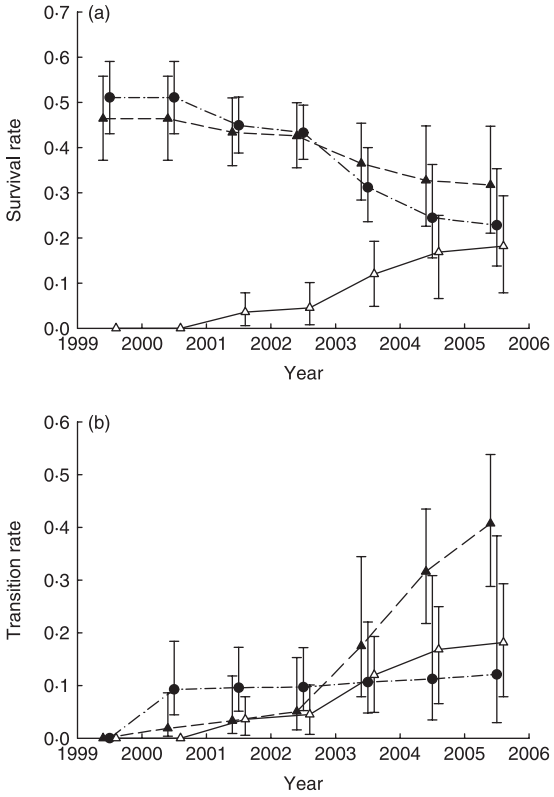

Fig. 4. Model averaged estimates from multistate analysis.(a) Yearly apparent survival rates for healthy individuals. Filled triangles (▲) and dashed line show survival rate of healthy subadults, filled circles (●) and dot-dashed line show survival rate of healthy adults. Open triangles (△) and solid line show Devil Facial Tumour Disease (DFTD) prevalence at each sampling period. In each case, the mean is shown ± 95% CI. (b) Transition rates of healthy individuals to diseased adults. Filled triangles (▲) and dashed line show the transition rate of healthy subadults to diseased adults, filled circles (●) and dot-dashed line show the rate of healthy adults to diseased adults. Open triangles (△) and solid line show DFTD prevalence at each sampling period. In each case, the mean is shown ± 95% CI.

Table 5. Realized population growth rate (λ) estimates from Pradel models (estimates are model averaged means with upper and lower 95% CI given in brackets)

Year	λ
1999–2000	1·001 (0·901, 1·101)
2000–01	1·001 (0·901, 1·101)
2001–02	0·899 (0·818, 0·946)
2002–03	0·874 (0·804, 0·921)
2003–04	0·697 (0·603, 0·777)
2004–05	0·602 (0·483, 0·711)
2005–06	0·579 (0·454, 0·694)

apparent survival rates of adults and subadults in the population, which was predicted well by the increase in disease prevalence in the population over time. DFTD had the most evident impact on the average apparent survival rates of older individuals. Apparent survival of adults marked-as-adults halved in the year following

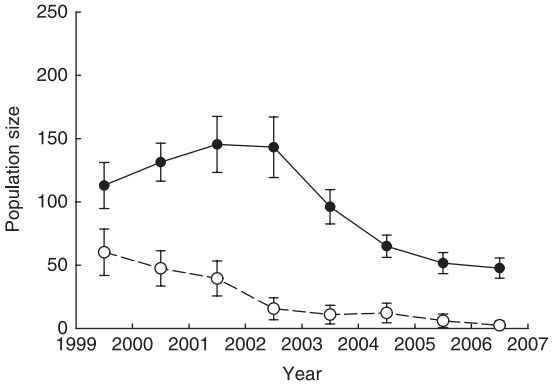

Fig. 5. Closed capture population size estimates of the whole population (filled circles ● and solid line) and the adult population only (open circles ○ and dashed line). Error bars are 95% CI.

the detection of DFTD and continued to decline effectively to zero as DFTD prevalence in the population increased. The variable apparent survival rates of younger adults likely reflects spatial variation in disease impact as it spread southwards down the peninsula, with the survival rate of individuals in some locations remaining unaffected until the disease progressed through the area. The change in survival rates through time, differed with age and marking group in a manner consistent with the observation that DFTD affected older animals first, but inconsistent with the possible action of environmental variables operating similarly across all survival rates.

The finding from the multistate recapture models that DFTD negatively impacted on the apparent survival of subadults in this study was unexpected. Only five diseased subadults have ever been captured in this population, all of which were caught in the final sampling period (July 2006) and all of which were excluded from the analyses presented here. In fact, it was thought that either there was a lengthy latency period associated with DFTD or that subadults were somehow excluded from DFTD infection and thus, that the continued survival of this group might ensure population persistence or act as a buffer to population extinction (Hawkins *et al.* 2006). Although the negative impact of DFTD on this group to date has not been as great as that observed for adults, the relationship between increasing disease prevalence and decreasing subadult survival rates detected in this study has diminished these possibilities.

We found that older individuals were the first in the population to be infected and succumb to DFTD; younger adults followed; and subadults only became infected once the majority of adults had died. This was evidenced by the decline in the age of infected individuals following the detection of DFTD into the population and also by the different pattern of infection rates (transition rates) of adults and subadults in the years following DFTD detection. In view of this, the time frame of this study may have been insufficient to detect

the full effect of DFTD in the population, particularly its impact on subadult survival.

The observation that DFTD is first detected in older individuals is an interesting one. It may simply be related to a lower force of infection due to low prevalence when the disease first enters the population. The average age of infection by a pathogen is inversely related to the force of infection (Grenfell & Anderson 1985). Alternatively, older individuals may be behaviourally more susceptible to acquiring disease, due to increased exposure to opportunities for transmission (Altizer *et al*. 2003). DFTD is believed to be transmitted via biting during feeding or sexual interactions (Pearse & Swift 2006). Thus, the pattern observed here may indicate that older individuals engage in aggressive or sexual encounters more frequently and predominantly with other adults. Older animals may also be more physiologically susceptible to disease due to higher levels of stress-related hormones associated with reproductive activity (Bronson 1989). Males of other dasyurid species are well-known to suffer stress-related immunological dysfunction following breeding (Boonstra 2005). However, we detected no evidence that males were particularly prone to DFTD infection or suffered higher mortality than females.

One important limitation of the mark–recapture methods employed in this study is that estimates of apparent survival do not differentiate between death and permanent emigration. If DFTD has disrupted movement patterns and resulted in increased emigration rates from diseased areas, then our survival rates will be biased low (as we are estimating the combined effects of both processes on survival). Biologically plausible explanations, relating to local population density, resource availability and available mating opportunities exist that could explain either increased or decreased dispersal in diseased areas (Dieckmann, O'Hara & Weisser 1999). Little is known about dispersal patterns or the mechanisms driving dispersal decisions in Tasmanian devils. Although they are likely to follow the normal mammalian (and dasyurid) pattern of predominantly male-biased natal dispersal of newly independent juveniles, an indication of such a pattern in terms of strongly sex-biased recapture rates was not detected in this study.

Multistate analyses allow robust estimates of apparent survival for individuals of different disease states. Apparent survival of diseased devils was effectively zero, because no diseased devils survived the year-long interval between sampling periods, confirming that DFTD is an aggressive, consistently fatal disease. From the regular trapping trips conducted at this study site the longest period between recaptures for a diseased animal was 6 months, while the longest known period between recaptures for any diseased animal from all state-wide disease monitoring efforts is 9 months (Hawkins *et al*. 2006).

As progression to death is so rapid, many of the individuals in the population may have acquired disease and died without this transition ever appearing as 'diseased' in our data set. In addition, those individuals that were captured with DFTD during trapping periods other than the winter period analysed in this study ($n = 36$ in this case) could also not be included in this analysis. These missed infections partly explain the somewhat counter-intuitive result that apparent survival of healthy individuals declined with disease prevalence. An additional possibility is that some asymptomatic devils were misclassified as healthy individuals but were in fact diseased, thereby depressing the apparent survival estimates of the healthy group. Although DFTD tumours are distinctive and thorough examinations for symptomatic signs were undertaken, it is possible that early stage lesions were misdiagnosed. Also, the latent period is unknown but could be as long as 12 months. Some devils were inevitably infected at the time they were trapped but had not yet developed visible tumours. The inevitable outcome of these missed and possibly misdiagnosed infections would be that the negative impacts of disease on apparent survival and infection rates were underestimated in this study.

No differences in the impact of DFTD on males and females were detected. Males and females appear to be equally susceptible to DFTD infection indicating that transmission of DFTD within and between the sexes occurs with equal probability. This study also did not detect any difference in apparent survival rates of males and females. This is in contrast to the only other study of mortality rates in Tasmanian devils, which reported higher mortality rates for males than females (Pemberton 1990). The estimates from that study, however, were obtained using cohort-based approaches with smaller sample sizes than recommended for those methods (Caughley 1977).

IMPACT OF DISEASE PREVALENCE ON TRANSITION RATES

The clear positive relationship found in this study between increasing disease prevalence and the transition rate to the infected class is expected from an infectious disease. Estimating the force of infection of a disease, which is the rate of acquisition of the infection for a susceptible host (Heisey, Joly & Messier 2006), is crucial to understanding infectious disease dynamics. Transition rates obtained from multistate models are a compound measure of the probability of becoming infected (showing symptoms) and surviving to be captured and may be regarded as a conservative indicator of the force of infection in the population. Since the detection of DFTD in this population, the total infection rate (all transitions of healthy individuals to disease states) has followed a steadily increasing trend over time. Moreover, as discussed above, true infection rates are probably considerably higher than is indicated by the estimates obtained here. These observations provide strong evidence that the force of DFTD infection in this population is increasing and that the current epidemic is

not subsiding. Particularly disturbing is the rapid and ongoing increase in the infection rate of subadults. A continuation in the trend observed (Fig. 4) will soon see subadults become more likely to develop into diseased adults than healthy adults. This scenario could constitute a major change to this species' life history (typically multiple breeding in a 5–6-year life span) as diseased devils may not survive long enough to rear a litter successfully (devils become independent at 9 months of age).

IMPACT OF DISEASE ON POPULATION GROWTH RATE

The decrease in estimated survival rates has resulted in a marked ongoing decline in the population growth rate. The population growth rate declined immediately following disease arrival despite disease prevalence being extremely low at this time (only 3·5%) and continued to decline rapidly, reaching a 30% decline after just 3 years. This is extremely relevant for the management of disease-free populations, highlighting the need to implement strategies swiftly following disease detection to prevent population decline. Of particular concern is the decline of almost 50% per year in the adult segment of the population.

Epidemiological theory suggests that a single-host pathogen is unlikely to drive its host to extinction, if disease transmission follows a density-dependent process, because disease maintenance and spread will not be possible when populations are sufficiently reduced (below a population threshold) (McCallum & Dobson 1995; de Castro & Bolker 2005). Indeed, a recent population projection model for devils showed that devils would be unlikely to be driven to extinction by a density-dependent disease (Bradshaw & Brook 2005).

If disease transmission is not density-dependent, however, thresholds for disease maintenance will not occur and population extinction is possible (de Castro & Bolker 2005). Although more field data are required to determine the transmission dynamics of DFTD, data from Mt William National Park in the far north-east of the state, suggest that any threshold population density for DFTD persistence is very low. At this site, DFTD prevalence remains high (33%) despite a reduction in population size from 7·14 individuals per km^2 to just 0·18 individuals per km^2 (unpubl. data). Whether the negative impacts of DFTD on survival detected in this study can be compensated for remains to be investigated. Our results suggest, however, that in the absence of population thresholds for disease maintenance and strong mechanisms of demographic compensation, local population extinction seems likely.

Acknowledgements

We would like to thank Steve Beissinger for invaluable advice with the CMR modelling approach and Dydee Mann and Rebecca Wilson who collected data for this study. Assistance with trapping was given by Campbell Allen, Frances Alberto Conelli, Martin Dallimer, Teresa Diehl, Luke Einoder, Karen Hurley, James Kennedy, Joy McDonald, Helen Otley, David Parer, Chris Spencer, Peggy MacQueen Nina Trikojus, Billy van Utreight, Megan Ward, Reuben Wells, Gab Warren, and thanks to Leon Barmuta who made a large part of this work possible. This manuscript was greatly improved by constructive comments given by Anne Goldizen, Clare Hawkins and four anonymous reviewers. This work was funded by Australian Research Council grants F19905533 (Australian Postdoctoral Fellowship to M. Jones), A00000162 (M. Jones and Andrew Cockburn) and LP0561120 (M. Jones and Hamish McCallum), an Australian National University Faculties Research Grant F02085 (M. Jones and A. Cockburn), the Estate of Winifred Violet Scott, and with contributions from Gunns Tamar Ridge Wines, Cosy Cabins Tasmania, Vanessa Quilliam, Rosalind Wharton, and Launceston Girl Guides. The Tasmanian Department of Primary Industries and Water provided financial assistance and much appreciated logistic support.

References

Altizer, S., Nunn, C.L., Thrall, P.H., Gittleman, J., Antonovics, J., Cunningham, A.A., Dobson, A., Ezenwa, V., Jones, K.E., Pederson, A.B., Poss, M. & Pulliam, J.R.C. (2003) Social organisation and parasite risk in mammals: integrating theory and empirical studies. *Annual Review of Ecology, Evolution and Systematics*, **34**, 517–547.

Anderson, D.R. & Burnham, K.R. (2002) Avoiding pitfalls when using information-theoretic methods. *Journal of Wildlife Management*, **66**, 912–918.

Arthur, A., Ramsey, D. & Efford, M. (2004) Impact of bovine tuberculosis on a population of brushtail possums (Trichosurus vulpecula Kerr) in the Orongorongo Valley, New Zealand. *Wildlife Research*, **31**, 389–395.

Bloomfield, T.E., Mooney, N. & Emms, C. (2005) The red fox in Tasmania; an incursion waiting to happen. *13th Australasian Vertebrate Pest Conference, Te Papa, Wellington, New Zealand*, pp. 299–300. Landcare Research Wellington, New Zealand.

Boonstra, R. (2005) Equipped for life: the adaptive role of the stress axis in male mammals. *Journal of Mammalogy*, **86**, 236–247.

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

Bradshaw, C.J., McMahon, C.R. & Brook, B.W. (2006) The devil in the (demographic) detail. *Frontiers in Ecology and the Environment*, **5**, 235–235.

Bronson, F.H. (1989) *Mammalian Reproductive Biology*. University of Chicago Press, Chicago, IL.

Burnham, K.P. & Anderson, D.R. (2002) *Model Selection and Multimodel Inference: a Practical Information-Theoretic Approach*. Springer-Verlag, New York.

de Castro, F. & Bolker, B. (2005) Mechanisms of disease-induced extinction. *Ecology Letters*, **8**, 117–126.

Caughley, G. (1977) *Analysis of Vertebrate Populations*. The Pitman Press, Bath.

Cooch, E.G. & White, G.C. (2001) *A Gentle Introduction. Program MARK. Analysis of Data from Marked Individuals*, Vol. 2006. Online Publication: www.phidot.org/software/mark/docs/book.

Das, U. & Das, A.K. (2000) Review of canine transmissible venereal sarcoma. *Veterinary Research Communications*, **24**, 545–556.

Dieckmann, U., O'Hara, B. & Weisser, W. (1999) The evolutionary ecology of dispersal. *Trends in Ecology and Evolution*, **14**, 88–90.

Faustino, C.R., Jennelle, C.S., Connolly, V., Davis, A.K., Swarthout, E.C., Dhondt, A.A. & Cooch, E.G. (2004) Mycoplasma gallisepticum infection dynamics in a house finch population: seasonal variation in survival, encounter and transmission rate. *Journal of Animal Ecology*, **73**, 651–669.

Grenfell, B.T. & Anderson, R.M. (1985) The estimation of age-related rates of infection from case notifications and serological data. *Journal of Hygiene*, **95**, 419–436.

Guiler, E.R. (1970) Observations on the Tasmanian devil, *Sarcophilus harrisii* (Marsupialia: Dasyuridae) II: Reproduction, Breeding and growth of pouch young. *Australian Journal of Zoology*, **18**, 63–70.

Hall, A.J., Jepson, P.D., Goodman, S.J. & Harkonen, T. (2006) Phocine distemper virus in the North and European Seas. Data and models, nature and nurture. *Biological Conservation*, **131**, 221–229.

Hawkins, C.E., Baars, C., Hesterman, H., Hocking, G.J., Jones, M.E., Lazenby, B., Mann, D., Mooney, N., Pemberton, D., Pyecroft, S., Restani, M. & Wiersma, J. (2006) Emerging disease and population decline of an island endemic, the Tasmanian devil, *Sarcophilus harrisii*. *Biological Conservation*, **131**, 307–324.

Heisey, D.M., Joly, D.O. & Messier, F. (2006) The fitting of general force-of-infection models to wildlife disease prevalence data. *Ecology*, **87**, 2356–2365.

Loh, R., Bergfeld, J., Hayes, D., O'Hara, A., Pyecroft, S., Raidal, S. & Sharpe, R. (2006) The pathology of devil facial tumor disease (DFTD) in Tasmanian devils (*Sarcophilus harrisii*). *Veterinary Pathology*, **43**, 890–895.

McCallum, H. (1994) Quantifying the impact of disease on threatened species. *Pacific Conservation Biology*, **1**, 107–117.

McCallum, H. & Dobson, A. (1995) Detecting disease and parasite threats to endangered species and ecosystems. *Trends in Ecology and Evolution*, **10**, 190–194.

McCallum, H. & Jones, M. (2006) To lose both would look like carelessness: Tasmanian devil facial tumour disease. *Plos Biology*, **4**, 1671–1674.

Mutze, G., Bird, P., Kovaliski, J., Peacock, D., Jennings, S. & Cooke, B. (2002) Emerging epidemiological patterns in rabbit haemorrhagic disease, its interaction with myxomatosis, and their effects on rabbit populations in South Australia. *Wildlife Research*, **29**, 577–590.

Nichols, J.D. & Hines, J.E. (2002) Approaches for the direct estimation of lambda, and demographic contributions to lambda, using capture-recapture data. *Journal of Applied Statistics*, **29**, 539–568.

O'Brien, S.J., Robert, B. & Tiandry, H. (2005) Consequences of violating the recapture duration assumption of mark–recapture models: a test using simulated and empirical data from an endangered tortoise population. *Journal of Applied Ecology*, **42**, 1096–1104.

Pearse, A.M. & Swift, K. (2006) Transmission of devil facial-tumour disease – an uncanny similarity in the karyotype of these malignant tumours means that they could be infective. *Nature*, **439**, 549–549.

Pemberton, D. (1990) Social organisation and behaviour of the Tasmanian devil, *Sarcophilus harrisii*. PhD, University of Tasmania, Hobart, Tasmania.

Pradel, R. (1996) Utilization of capture-mark-recapture for the study of recruitment and population growth rate. *Biometrics*, **52**, 703–709.

Saunders, G., Lane, C., Harris, S. & Dickman, C.R. (2006) *Foxes in Tasmania: a Report on the Incursion of an Invasive Species*. Invasive Animals Cooperative Research Centre, Canberra, Australia.

Schmitz, O.J., Hamback, P.A. & Beckerman, A.P. (2000) Trophic cascades in terrestrial systems: a review of the effects of carnivore removal on plants. *American Naturalist*, **155**, 141–153.

Sih, A., Crowley, P., McPeek, M., Petrank, J. & Strohmeier, K. (1985) Predation, competition and prey communities: a review of field experiments. *Annual Review of Ecology, Evolution and Systematics*, **16**, 269–311.

Telfer, S., Bennett, M., Bown, K., Cavanagh, R., Crespin, L., Hazel, S., Jones, T. & Begon, M. (2002) The effects of cowpox virus on survival in natural rodent populations: increases and decreases. *Journal of Animal Ecology*, **71**, 558–568.

White, G.C. & Burnham, K.P. (1999) Program MARK: survival estimation from populations of marked animals. *Bird Study*, **46**, 120–138.

Williams, B.K., Conroy, M.J. & Nichols, J.D. (2002a) *Analysis and Management of Animal*. Populations Academic Press, San Diego, CA.

Williams, E.S., Miller, M.W., Kreeger, T.J., Kahn, R.H. & Thorne, E.T. (2002b) Chronic wasting disease of deer and elk: a review with recommendations for management. *Journal of Wildlife Management*, **66**, 551–563.

Wilmers, C.C., Post, E., Peterson, R.O. & Vucetich, J.A. (2006) Predator disease out-break modulates top-down, bottom-up and climatic effects on herbivore population dynamics. *Ecology Letters*, **9**, 383–389.

Received 11 February 2007; accepted 4 May 2007