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Demography, disease and the devil: life-history changes in a disease-affected population of Tasmanian devils (*Sarcophilus harrisii*)

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Summary

1. Examining the demographic responses of populations to disease epidemics and the nature of compensatory responses to perturbation from epidemics is critical to our understanding of the processes affecting population dynamics and our ability to conserve threatened species. Such knowledge is currently available for few systems.

2. We examined changes to the demography and life-history traits of a population of Tasmanian devils (*Sarcophilus harrisii*) following the arrival of a debilitating infectious disease, devil facial tumour disease (DFTD), and investigated the population's ability to compensate for the severe population perturbation caused by this epizootic.

3. There was a significant change to the age structure following the arrival of DFTD to the Freycinet Peninsula. This shift to a younger population was caused by the loss of older individuals from the population as a direct consequence of DFTD-driven declines in adult survival rates.

4. Offspring sex ratios of disease mothers were more female biased than those of healthy mothers, indicating that devils may facultatively adjust offspring sex ratios in response to disease-induced changes in maternal condition.

5. We detected evidence of reproductive compensation in response to disease impacts via a reduction in the age of sexual maturity of females (an increase in precocial breeding) over time.

6. The strength of this compensatory response appeared to be limited by factors that constrain the ability of individuals to reach a critical size for sexual maturity in their first year, because of the time limit dictated by the annual breeding season.

7. The ongoing devastating impacts of this disease for adult survival and the apparent reliance of precocial breeding on rapid early growth provide the opportunity for evolution to favour of this new life-history pattern, highlighting the potential for novel infectious diseases to be strong selective forces on life-history evolution.

Key-words: age structure, demographic compensation, Devil Facial Tumour Disease, sex ratio biases, Tasmanian devil

Introduction

Understanding the relationship between life-history patterns, population dynamics and population regulation has been a central issue of population ecology for the better half of a century (Cole 1954; Sinclair 1989). Despite this, the consequences of infectious diseases and disease-induced changes to host life histories for population dynamics have only recently begun to receive attention (Choisy & Rohani 2006; Woodroffe *et al.* 2008).

Infectious diseases that reduce population numbers or densities can cause density-dependent changes in host population dynamics as a result of changes to the life-history traits of individuals in the population (Fowler 1981). Demographic responses to perturbations that cause a reduction in population size include increased reproductive rates, earlier onset of sexual maturity, increased survival of some population classes, or increased recruitment rates (Coulson *et al.* 2004). These compensatory changes in life-history traits can confer

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animal populations with a remarkable capacity to recover from perturbation and have rescued populations from the brink of extinction (for example, black-footed ferrets Grenier, McDonald & Buskirk 2007). However, while compensatory responses to changes in population abundance and density are known for long-lived species (Pistorius *et al.* 2001; Hadley *et al.* 2006) and have been extensively studied in species subject to harvesting (Coulson *et al.* 2004; Choisy & Rohani 2006, Milner, Nilsen & Andreassen 2007), such responses to disease epidemics remain largely unexplored (Loison, Gaillard & Jullien 1996; Mutze *et al.* 2002).

Moreover, knowledge of the demographic changes that stem from disease epidemics is essential not only for understanding the processes that affect population dynamics but also for effectively managing diseased populations. Since infectious diseases are potentially important drivers of local population extinctions (Haydon, Laurenson & Sillero-Zubiri 2002; Gerber *et al.* 2005), management actions are often needed to mitigate the detrimental impacts of disease, particularly for species of conservation concern. Understanding the impact of a pathogen on host population dynamics and host life histories is necessary to predict population responses to disease control measures (McCallum, Barlow & Hone 2002).

Devil facial tumour disease (DFTD) is a recently emerged infectious cancer that is now widespread and represents a serious threat to the Tasmanian devil *Sarcophilus harrisii*, (Hawkins *et al.* 2006). This fatal cancer is transmitted from animal to animal as an allograft (a tissue graft of the actual tumour cells) during social interactions (Siddle *et al.* 2007). Estimates of disease spread, indicating that DFTD will cover the entirety of the devils' range in as little as 5 years time, in combination with models predicting population declines to extinction within 15 years of disease arrival, equate to an unacceptable risk of extinction for the wild devil population (McCallum *et al.* 2007).

Recently, Jones *et al*. (2008) examined changes in life history in five Tasmanian devil populations affected by the facial tumour disease, reporting consistent changes to population age structure and a decline in the age of sexual maturity of females across all five populations. Their study was based on snapshots of 1 or 2 years data 'before' and 'after' an unknown disease arrival time at each site, as this is the only information available at multiple sites. Here we report the results of a demographic study conducted at one of the five sites, the Freycinet Peninsula on the east coast of Tasmania. This site has been intensively trapped since 1999 with disease first appearing in 2001. The arrival of DFTD in the Freycinet Peninsula population triggered an immediate and steady decline in the survival rates of adults and sub-adults, and resulted in a substantial reduction to population growth rate and an ongoing decline in population abundance (Lachish, Jones & McCallum 2007). This unique data set allows us to investigate thoroughly the demographic and life-history changes following disease arrival, accounting for initial demographic variability within a population before disease arrival and subsequently monitoring changes in life-history parameters with disease progression.

In this study, our objectives were (i) to determine the nature of disease impacts on population demographic parameters and life-history traits, (ii) to obtain robust estimates of the vital rates of diseased and nondiseased individuals for use in models to aid management actions, and (iii) to determine the population's ability to respond to low population densities and to compensate for the detrimental impacts of DFTD.

Methods

TASMANIAN DEVIL LIFE HISTORY

The largest of the extant carnivorous marsupials (Dasyuridae), Tasmanian devils are sexually dimorphic with males growing larger and heavier (average 10 kg) than females (average 7 kg). Like all dasyurids, devils have relatively short life spans, living up to 6 years in the wild (Lee & Cockburn 1985). Devils are synchronous, annual breeders and females produce a maximum of four young per year with most births occurring from March to April (Pemberton 1990). Before the emergence of DFTD, females became sexually mature and began breeding at 2 years of age, with only a handful of records of 1-year-old females breeding (Jones *et al.* 2008). Devils occupy large (range 10–20 km^2) home ranges, which frequently overlap with those of several other individuals (Pemberton 1990).

STUDY AREA AND TRAPPING METHOD

Tasmanian devils were trapped within the 160 km^2 Freycinet Peninsula on the east coast of Tasmania (Fig. 1). For a detailed description of vegetation communities within the peninsula, see Lachish *et al*. (2007). The entire peninsula was trapped up to four times a year since 1999, with trapping periods timed to coincide with key stages in the breeding cycle: autumn (March/April), early pouch young; winter (June/July), late pouch young; spring (September/October), females lactating with young in dens; summer (December/January), dependent young emerging from dens. Trapping protocols and procedures taken to minimize accidental disease transmission are detailed in Lachish *et al*. (2007).

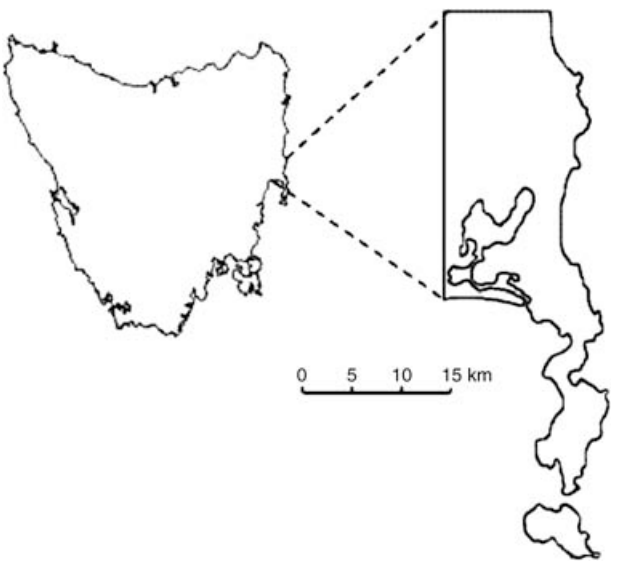

Fig. 1. Map of Tasmania showing location of the Freycinet Peninsula study site.

IDENTIFICATION AND AGEING OF TRAPPED DEVILS

Animals were identified via unique ear tattoos (from 1999 to 2003) or via unique microchip transponders (Allflex New Zealand, 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 over-eruption (distance from the dentine-enamel junction to the gum) (Pemberton 1990, M. Jones, unpublished data). This method is precise for ageing devils to 2 years of age but less reliable for older devils. Nonetheless, it is possible to differentiate mature individuals (3 years) from old animals (4-, 5- and 6-year-olds). As only 7% (42/609) of devils in our data set were estimated to be older than 2 years of age on their first capture, errors associated with ageing animals are likely to be minimal. To further limit errors in ageing, no attempt was made to distinguish 4-, 5- and 6-year-olds (all classed as 4 years and older).

DISEASE ON THE FREYCINET PENINSULA AND DISEASE DETECTION IN TRAPPED DEVILS

DFTD was first detected in June 2001 at the northernmost end of the peninsula, with diseased individuals captured sequentially southward in subsequent years (see Fig. 4A, Hawkins *et al.* 2006). Detection of DFTD is only possible by the visible presentation of tumours. All manifestations of the disease involve facial tumours that are distinctive, globular lesions, which are easily detected during thorough examination of the head region (see Lachish *et al.* 2007 for complete disease detection methods). 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.

ESTIMATION OF DEMOGRAPHIC PARAMETERS AND DATA ANALYSES

To explore overall changes in life-history parameters over time, we employed a model selection approach. A candidate set of plausible models (see details below) was chosen a priori to explain variation in the parameter of interest over time. The models were ranked on the basis of Akaike information criteria (AIC) with the model with the lowest AIC deemed the most parsimonious, 'best', model supported by the data (Burnham & Anderson 2002). Models with differences in AIC values of less than two were considered equivalent in their ability to describe the data, while differences in AIC values of greater than two indicated that one model was considerably better supported by the data (Burnham & Anderson 2002). Normalized AIC weights provide the relative likelihood of each model in the candidate set (Burnham & Anderson 2002). In other analyses that directly addressed specific hypotheses, a traditional, significance-testing approach was employed.

Population age and sex structure

We explored changes in age structure and sex ratios based on individuals trapped during the winter (June/July) trapping period each year from 1999 to 2007, as this was the only consistently sampled period every year and was after the dispersal phase of juveniles (Pemberton 1990). A generalized linear model with Poisson error was first used to determine if the overall age structure of the population changed throughout the study. Linear models were then used to examine changes to the mean age class (the mean age of individuals assigned to three age classes: 1-year-olds, 2-year-olds and 3-year-olds and older) of the population over time. This variable was used instead of mean age to reduce possible bias associated with the greater degree of uncertainty in ageing older individuals at the start of the study.

Changes to the mean age class over time were analysed with respect to population disease status, a binary variable being 'healthy' for the first two time periods (no diseased devils were captured during any of the monitoring trips before June 2001) and 'diseased' thereafter, and one of three time surrogates: 'year', a categorical variable modelling year-to-year variation in mean age class; 'trend', a variable modelling a linear change in mean age class over time; or 'disease prevalence', a variable that allowed mean age class to vary according to estimated population disease prevalence (the proportion of diseased individuals captured in each sampling period). This is an unbiased measure of disease prevalence as analyses show no significant difference in the capture probabilities of diseased and healthy devils at this population (H. McCallum, M. Jones, C. Hawkins, R. Hamede, S. Lachish, D. Sinn, N. Beeton & B. Lazenby, unpublished). To account for the possibility of a nonlinear relationship between changes in mean age class and time (see Results), we also included a quadratic term ($trend^2$) as an explanatory variable and fitted a segmented regression model (with two segments) to the data. These models were compared to a null model of no change in the mean age class over time resulting in a candidate set of nine models.

Changes to the population sex ratio over time were examined using logistic models (with a logit link function and binomial error) with age class (1-year-olds and adults: individuals aged 2 years or older), population disease status and time as explanatory variables (and time again modelled as either 'year', 'trend' or 'disease prevalence'). With the inclusion of a null model, this resulted in a candidate set of 14 models.

Proportion of females breeding

The proportion of females breeding each year was determined from all captures of females in a given year. A female was considered to have bred in a given year if she was captured either with pouch young or with lactating teats from one mating season (March/April) to the next. Lactating mammaries are enormously swollen and easily differentiated from their non-active state. Changes to the proportion of females breeding each year were analysed using logistic models with age class (1-year-olds and adults), population disease status and the three time surrogates ('year', 'trend' or 'disease prevalence') as explanatory variables. With the inclusion of a null model, this resulted in a candidate set of 14 models. Changes to the proportion of adults and 1-year-olds breeding were also analysed in relation to population disease status, the three time surrogates and a null model. A quadratic term ($trend^2$) was included to model a nonlinear change in the proportion of 1-year-olds breeding over time. A Fisher's exact test comparing the proportion of diseased versus healthy females that bred throughout the study was used to specifically examine the influence of DFTD infection status on the likelihood of breeding. This analysis was only possible for adult females as too few 1-year-old females were captured with DFTD.

In order to identify factors contributing to changes in the proportion of 1-year-olds breeding, we compared the body size measurements of 1-year-old breeders to 1-year-old nonbreeders and to adult breeders. Head width (the widest distance across the zygomatic arches), body weight and maximal tail width (indicative of body condition,

as many dasyurids store fat in their tails) were used as body size metrics and were analysed using ANOVAs followed by Tukey's post-hoc tests to examine pairwise differences among groups. When these analyses revealed a relationship between body size and 1-year-old breeding (see results), we specifically examined the likelihood that an individual bred given its body size using a logistic regression model with age class (in this case to 4 years and older) and maximum head width or body weight as explanatory variables.

Growth rate estimates

To determine whether observed changes in the proportion of 1-year-olds breeding were influenced by growth rates, we obtained yearly growth rate estimates for 1-year-olds using a nonlinear mixed effect model with either a Gompertz or a von Bertalanffy growth curve, sex and year as fixed effects and 'individual' as the random effect (using the 'nlme' function in R version 2·5·0). We used growth interval data (change in size over a time period) for 1-year-olds only. The transition between age classes was assumed to occur in March and any growth measurements from intervals spanning this period [e.g. from April of 1 year (age 1) to July of the next year (age 2)] were not included. Hence, only growth measurements from an individual's second year of life were included and thus, each individual only contributed to the growth rate estimate in a single year. Maximum head width was the metric used as it is subject to less measurement error than the other size indices and the asymptotes (separate asymptotes for males and females) were fixed to be constant across years. From this model, we extracted growth rate coefficients in each year for each 1-year-old individual and analysed the change in male and female growth rates over time using linear regressions. Our data set consisted of 97 females and 100 males from 1999 to 2007 (no estimate was available for 2003 as only one trapping trip was undertaken in June of that year).

Litter size and pouch young sex ratios

Litter size estimates were determined from the number of 'active' teats of females captured either lactating or with pouch young attached. This represents the maximum number of young carried by a female since attachment of a neonate is required for a teat to become active and teats remain active for the remainder of the breeding season, even after late detachment or loss of the young. Changes in litter size were analysed in relation to population disease status and time ('year', 'trend' or 'disease prevalence') using linear models to examine changes in mean litter size over time and logistic models to examine changes to the proportion of litters containing four young each year. With the inclusion of null models, this resulted in a candidate set of five models for each response variable. Pouch young sex ratios were determined only from litters where all the pouch young were sexed (sexing very small pouch young is not possible as they still possess an undifferentiated phallus). Logistic models were used to examine changes in pouch young sex ratios in relation to population disease status, time ('year', 'trend' or 'disease prevalence') and a null model.

Finally, mixed models were used to specifically examine the effect of age, experience (primiparous versus multiparous litters; for females with multiple captures and known breeding histories) and a mother's disease status (healthy or diseased) on both litter size (linear mixed model) and pouch young sex ratios (generalized linear mixed model with logit link and binomial error) with 'mother' included as the random effect. All statistical analyses were performed in R version 2·5·0 (R Development Core Team 2007).

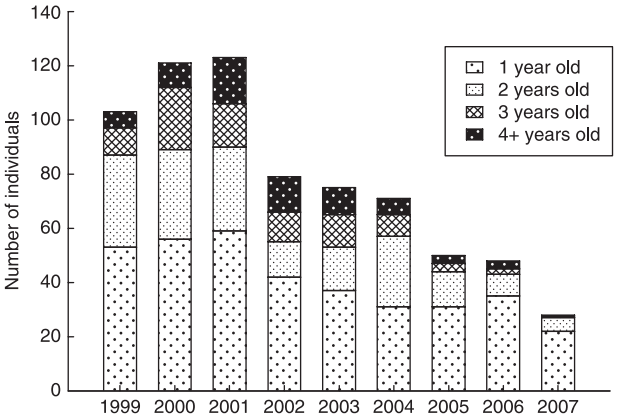

Fig. 2. Number of individual Tasmanian devils of each age class captured during the winter (June/July) field trip each year in the Freycinet Peninsula.

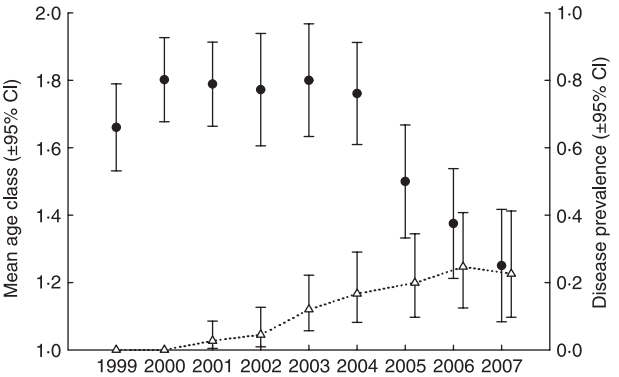

Fig. 3. Changes to the mean age class of the Freycinet population of Tasmanian devils over time (closed circles, •). Estimates are means ±95% CI. Open triangles (Δ) and dashed line shows DFTD prevalence at each sampling period (±95% CI).

Results

We captured 609 devils (308 males, 301 females) during the study, 80% of which were first captured on the study site as 1-year-olds. A total of 93 individuals (44 males and 49 females) were captured with signs of DFTD.

AGE STRUCTURE CHANGES

The age structure of the population changed significantly over the course of this study (Δdev for removal of age class × year interaction = 61·22, Δd.f. = 24, $P < 0{\cdot}001$). Fig. 2 shows that before disease arrival, adults comprised more than half (51–53%) of the individuals captured, with the oldest age class accounting for between 7% and 14% of individuals. By 2007, however, adults comprised just 20% of the sample, with just one individual older than 2 years of age captured (Fig. 2). Inspection of Fig. 3 shows that the mean age of a trapped individual in the population declined by almost 30% over the course of the study. The decline in mean age, however, was not apparent until 2004, 3 years after disease arrival, and there was a slight difference in the mean age between the two

Table 1. Results of model selection based on AIC for (a) linear models explaining changes in the mean age class over time and (b) logistic models explaining changes in the population sex ratio over time of the Freycinet population of Tasmanian devils

Competing models	k	AIC	ΔAIC	w_i
a. Mean age				
Trend^2	3	1664·33	0	0·50
Prevalence + trend^2	4	1666·09	1·76	0·21
Segmented regression	4	1666·43	2·10	0·17
Prevalence + trend	3	1667·99	3·65	0·08
Prevalence	2	1669·53	5·20	0·04
Year	9	1674·42	10·09	0·00
Trend	2	1676·68	12·35	0·00
Disease status	2	1686·07	21·74	0·00
Null	1	1686·17	21·83	0·00
b. Population sex ratios				
Null	1	82·37	0·00	0·32
Disease status	2	84·48	2·10	0·11
Prevalence	2	84·50	2·12	0·11
Trend	2	84·55	2·18	0·11
Age class	2	84·57	2·20	0·11
Age class∗prevalence	5	86·05	3·67	0·05
Age class + disease status	4	86·18	3·80	0·05
Age class∗trend	5	86·18	3·80	0·05
Age class + prevalence	4	86·33	3·96	0·04
Age class + trend	4	86·35	3·97	0·04
Age class∗disease status	5	87·63	5·26	0·02
Year	9	97·02	14·65	0·00
Age class + year	11	99·01	16·64	0·00
Age class∗year	19	107·39	25·02	0·00

Age class = adults or 1-year-olds; * main effects and interactions; k = number of parameters; w_i = model weight.

pre-disease years (Fig. 3). Consequently, the best supported models for the change in mean age class were those supporting a nonlinear change in this variable over time (the models with quadratic terms and the segmented regression model) with models including the effect of disease prevalence receiving around 30% of the weight (Table 1a). This suggests that disease played a role in the decline in mean age but that disease prevalence alone was not enough to explain the observed changes to the age structure of the population. Individuals trapped with signs of disease were on average older [2·27 ± (95% CI) 0·68 years] than healthy individuals (1·80 ± 0·28 years, $t = 3{\cdot}13$, d.f. = 49·95, $P = 0{\cdot}003$) supporting earlier findings that disease predominantly affects older individuals (Lachish *et al.* 2007).

POPULATION SEX RATIOS

The overall sex ratio of the population was not significantly different to parity ($X^2 = 1{\cdot}54$, d.f. = 1, $P = 0{\cdot}21$). Models including age class as a variable received little support indicating that sex ratios did not differ between adults and 1-year-olds. There was little evidence that population sex ratios changed throughout the study with the null model receiving three times the weight of any of the competing models (Table 1b).

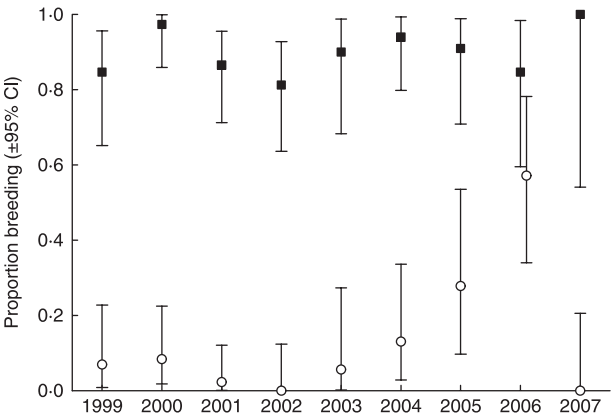

Fig. 4. The proportion of females breeding in the Freycinet population of Tasmanian devils each year ±95% CI. Solid squares (■) show the proportion of adult females that bred each year. Open circles (○) show the proportion of 1-year-old females that bred each year.

PROPORTION OF FEMALES BREEDING

The proportion of adult females breeding varied between 81% and 100% and was consistently high throughout the study (Fig. 4). Conversely, only a minority of all 1-year-old females bred during the study with a marked increase in the proportion of 1-year-olds breeding (precocial breeding) from 2004 to 2006 (Fig. 4). There was no indication that disease affected the likelihood of breeding among adult females (Fisher's exact test for odds of breeding by diseased versus healthy females, 0·96; 95% CI 0·29–4·03).

The best-supported model explaining changes in the proportion of females breeding over time included an interaction between age class and year, indicating differences between adults and 1-year-olds in the pattern of change in this life-history trait over time (Table 2a). While the proportion of adult females breeding did not appear to change throughout the study with the null model the best-supported model by the data, there was evident variation in the extent of precocial breeding over time, with the model including year-to-year variation receiving almost all the weight in the candidate set (Table 2b). Model selection showed that the observed increase in precocial breeding to 2006 was best described either as a quadratic function or as a function of increasing disease prevalence in the population, with models including an effect of disease prevalence receiving almost 50% of the weight (Table 2c). The absence of precocial breeders in 2007 prompted us to examine other factors that may influence precocial breeding in devils.

All size metrics used in the analysis of body size differences were significantly related to an individual's status as 1-year-old breeder, 1-year-old nonbreeder or adult breeder [head width; $F = 120{\cdot}02_{(2,292)}$, $P < 0{\cdot}001$, weight; $F = 112{\cdot}45_{(2,292)}$, $P < 0{\cdot}001$, tail width; $F = 108{\cdot}29_{(2,292)}$, $P < 0{\cdot}001$]. One-year-old breeders had significantly larger head widths [mean (± 95% CI) = 101·32 mm (1·17)] than 1-year-old nonbreeders [96·38 mm (0·811); mean difference (95% CI) = 4·92 mm (2·31–7·52)] and those captured were on average heavier [6·00 kg (0·34)] than 1-year-old nonbreeders [5·53 kg (0·15);

Table 2. Results of model selection based on AIC for competing logistic model explaining changes in the proportion of females breeding over time in the Freycinet population of Tasmanian devils

Competing models	k	AIC	ΔAIC	w_i
a. Adults and 1-year-olds				
Age class*year	19	76·79	0·00	0·70
Age class + year	12	78·57	1·78	0·29
Age class*prevalence	5	85·11	8·31	0·01
Age class + prevalence	4	87·05	10·26	0·00
Age class*trend	5	88·67	11·88	0·00
Age class + trend	4	91·28	14·48	0·00
Age class	2	100·13	23·34	0·00
Age class + disease status	4	102·04	25·25	0·00
Age class*disease status	5	102·04	25·25	0·00
Prevalence	2	415·56	338·76	0·00
Trend	2	415·79	338·99	0·00
Null	1	416·64	339·85	0·00
Disease status	2	418·59	341·79	0·00
Year	9	446·36	369·57	0·00
b. Adults only				
Null	1	32·36	0·00	0·53
Disease status	2	34·61	2·25	0·17
Prevalence	2	34·86	2·50	0·15
Trend	2	34·97	2·61	0·14
Year	9	39·91	7·55	0·01
1-year-olds only				
Year	9	36·88	0·00	0·99
Prevalence	2	51·04	14·16	0·01
Trend	2	54·50	17·62	0·00
Null	1	65·25	28·37	0·00
Disease status	2	68·32	31·44	0·00
c. 1-year-olds only excluding 2007				
Trend^2	3	28·41	0·00	0·57
Prevalence	2	30·15	1·74	0·25
Prevalence + trend^2	4	30·98	2·57	0·16
Year	8	34·88	6·47	0·02
Trend	2	37·25	8·84	0·01
Disease status	2	63·58	35·17	0·00
Null	1	63·67	35·26	0·00

Age class = adults and 1-year-olds; *main effects and interactions; k = number of parameters; w_i = model weight.

mean difference 0·48 kg (–0·21–1·17)]. There was no difference between these two groups in tail widths [mean tail width of 1-year-old breeders = 30·26 mm (0·93); 1-year-old nonbreeders = 30·12 mm (0·51); mean difference 0·14 mm (–2·13–2·41)]. Results of the logistic regression showed a significant interaction between size (maximum head width) and age class in relation to the probability of breeding (Δdeviance for removal of head width by age class interaction = 11·34, Δd.f. = 3, $P = 0·01$; a similar pattern was obtained with body weight). Size was significantly related to the likelihood of breeding for 1-year-olds, but not for any other age class (Table 3). Indeed, 1-year-olds present in years when no precocial breeders were recorded were significantly smaller than the average 1-year-old in other years (e.g. two sample t-test for size difference of 2007 1-year-olds versus 1-year-olds in all other years: head width; $t = 2·43$, d.f. = 13·8, $P = 0·039$; weight, $t = 4·21$, d.f. = 15·02, $P < 0·001$). These results suggest that there is a certain critical size that females need to reach in order to be able to breed as 1-year-olds.

Table 3. Results of logistic regression models showing the significance of body size (maximum head width) for the probability of breeding in female Tasmanian devils of different age classes (given as the change in deviance for the removal of 'head width' from the model) at the Freycinet Peninsula

Age class	Δdeviance	Δd.f.	P
1-year-olds	9·78	1	0·002
2-year-olds	0·23	1	0·621
3-year-olds	0·19	1	0·187
4-year-olds and older	2·96	1	0·085

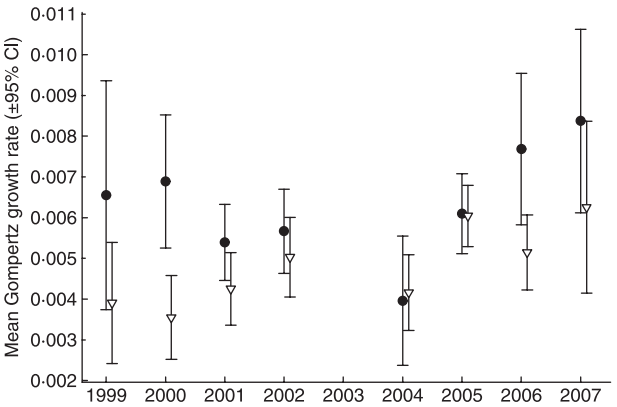

Fig. 5 Growth rates of 1-year-old Tasmanian devils in the Freycinet population over time. Estimates are means ± 95% CI for the Gompertz growth rate parameter '*a*', which demotes the rate at which an individual approaches its asymptote. Solid squares (■) show growth rate estimates for 1-year-old females. Open circles (○) show growth rate estimates for 1-year-old males.

Our data suggest that the increase in precocial breeding observed was mediated by the ability of individuals to grow faster in a diseased (and thus lower density) population. We found a significant increase in the growth rate of 1-year-old males in the population over time [$F = 70·3_{(1,98)}$, $P < 0·001$, $R^2 = 0·41$; Fig. 5] suggesting that the observed population decline reduced competition for resources and led to increased resource availability. Although, there was no consistent trend in the growth rate of 1-year-old females overall [$F = 1·9_{(1,95)}$, $P = 0·17$, $R^2 = 0·009$; Fig. 5], omitting 2004 data (the growth rate estimate for females appears unusually low in this year and also dips among males) resulted in a significant increase in the growth rates of females over time [$F = 9·78_{(1,86)}$, $P = 0·002$, $R^2 = 0·09$] and an even stronger trend among males [$F = 107·8_{(1,86)}$, $P < 0·001$, $R^2 = 0·55$]. However, as we are unable to identify a biologically plausible reason for the low estimate in 2004 (this was not a drought year nor were any of our trapping or measuring methodologies different), this result must be interpreted with reserve. The choice of growth curve model used (Gompertz vs. von Bertalanffy) did not change the patterns observed and only results obtained with the Gompertz growth curve are presented here.

Table 4. Results of model selection based on AIC for (a) linear models explaining changes in the mean litter size over time, (b) logistic models explaining changes in the proportion of litters of four young over time, and (c) logistic models explaining changes in pouch young sex ratios over time in the Freycinet population of Tasmanian devils

Competing models	k	AIC	ΔAIC	w_i
a. Mean litter size				
Null	1	349·19	0·00	1·00
Prevalence	2	396·15	46·96	0·00
Trend	2	396·17	46·98	0·00
Disease status	2	396·18	46·99	0·00
Year	9	401·33	52·14	0·00
b. Proportion of litters with four young				
Null	1	37·63	0·00	0·53
Prevalence	2	39·76	2·13	0·18
Trend	2	39·96	2·33	0·17
Disease status	2	40·60	2·97	0·12
Year	9	48·93	11·30	0·00
c. Pouch young sex ratios				
Null	1	422·05	0·00	0·51
Prevalence	2	424·24	2·19	0·17
Disease status	2	424·35	2·30	0·16
Trend	2	424·55	2·50	0·15
Year	9	433·14	11·09	0·00

k = number of parameters; w_i = model weight.

LITTER SIZE AND POUCH YOUNG SEX RATIOS

Mean litter size among breeding females was 3·42 (± 95% CI 0·1) young per mother with 66% of all mothers carrying litters containing four young (average per year ranged from 54–78%). Pouch young sex ratios were not significantly different from parity ($X^2 = 2·26$, d.f. = 1, $P = 0·13$). There was no evidence to suggest a change in any of these variables over time with the null model the best-supported models in each of the candidate model sets (Table 4a–c).

There was no difference in the litter size of healthy versus diseased mothers (mean litter size for disease mothers = 3·40 (95% CI 3·05–3·75); healthy mothers = 3·42 [95% CI 3·32–3·54; $F = 0·007_{(1,43)}$, $P = 0·98$]. Pouch young sex ratios, however, did vary according to the disease status of the mother (GLMM, Z = 2·25, $P = 0·024$) with diseased mothers producing almost twice as many daughters as did healthy mothers, although the confidence interval was large (Fisher's exact test for odds of producing females, 1·98; 95% CI 1·06–3·76).

Discussion

The arrival of DFTD to the Freycinet Peninsula resulted in a substantial change to the age structure of the population, caused by the loss of older individuals. This shift to a younger population age structure is a result of disease-driven declines in adult survival rates of Tasmanian devils (Lachish *et al.* 2007) and represents a direct population level consequence of disease impacts on individuals. That the decline in mean age class was not wholly explained by disease prevalence and did not begin immediately upon disease arrival (despite adult survival rate declining at this time, Lachish *et al.* 2007), is likely to be a consequence of spatial variation in disease impacts associated with disease spread. Since DFTD reached the southernmost tip of the 30-km long Freycinet Peninsula approximately 5 years after its initial detection (Hawkins *et al.* 2006; McCallum *et al.* 2007), older individuals would have been able to survive and persist in unaffected areas for several years before disease spread to those areas. The initial increase in the mean age of the population may be a result of a high recruitment or immigration pulse before the commencement of the study.

The loss of adults from a population can pose serious threats to its persistence by reducing the breeding potential of the population either as a consequence of lowered reproductive success or recruitment rates (Coltman *et al.* 2003) or as a result of breakdowns to social or dominance hierarchies and networks (Rogers *et al.* 1998). If, however, there is a concurrent reduction in population abundance, which reduces competition for normally limited resources, then these potential impacts may be mitigated by compensatory changes in demographic parameters, such as an earlier onset of maturity or increased fecundity (Coulson *et al.* 2004).

We observed an increase in the proportion of 1-year-old females breeding as disease progressed through the population, providing evidence of reproductive compensation in Tasmanian devil populations in response to disease-driven declines in adult survival rates and population abundance. In contrast, neither litter size nor reproduction by adult females showed evidence of compensatory responses to population decline following disease arrival. Since a very high proportion of adult females bred each year (often only the old, senescent, females did not breed), and since Tasmanian devils produce super-numerary young (females give birth to more than 20 neonates, Hughes 1982), it seems there is limited scope for compensatory increases in these reproductive traits, as selection has already maximized females' investment in these life-history parameters. For 'fast-living' species, such as the Tasmanian devil, that mature early and have high fecundity rates, the decrease in generation time resulting from the earlier onset of sexual maturity, will be key to maximizing the reproductive output of the population and its population growth rate (Oli & Dobson 2003). The plasticity observed in the age of first reproduction in Tasmanian devils and the capacity for this life-history trait to respond to changes in population density potentially represent an important mechanism for mitigating population perturbations.

The earlier onset of maturity in Tasmanian devils breeding in the diseased population appeared to be facilitated by more rapid growth in response to reduced population density, via reduced food competition. It is widely recognized that increases in food availability and consequent nutritional status of individuals' result in increased growth rates of individuals (see Altmann & Alberts 2005 and references therein) which can also accelerate sexual maturity (Bercovitch & Strum 1993). In this study, we detected a significant increase in the growth rates of 1-year-old males over time (with suggestion of

a similar trend in females), thus demonstrating that growth rates did increase as the population declined, most likely in response to increased food availability. Although, it is difficult to ascertain the extent of density-dependent effects on vital rates before disease arrival, models showing that the population growth rate of the Freycinet population was stable pre-disease suggest that the population was initially close to carrying capacity (Lachish *et al.* 2007). Life-history theory predicts that resource limitation at higher population densities will more adversely affect body growth in the larger sex (Hedrick & Temeles 1989).

The finding that female growth rates were not as strongly influenced by changes in population abundance indicates that the ability to breed as a 1-year-old (precocial breeding) is influenced by more than just population density. In fact, our data suggest that, in addition to density-dependence, the age of first reproduction in Tasmanian devils is determined by the ability of individuals to attain a critical size for sexual maturity in their first year. This is indicated by the extremely strong relationship between body size and the probability of 1-year-olds breeding, the absence of precocial breeders in 2007 when, although population density was at its lowest, 1-year-olds were significantly smaller than average, together with the lack of strong support for models relating precocial breeding to either trend or disease prevalence (as indicators of changing population density).

Since Tasmanian devils are seasonal breeders (Pemberton 1990), there will likely be a time limit by which a critical size for maturity must be reached for breeding to occur in a given season. Consequently, the timing of reproduction will have a great effect on the likelihood that an individual will be large enough to breed in its first year. Devils exhibit a seasonal synchronous peak in reproduction but the breeding season is actually quite broad (births occur from February through June, Pemberton 1990), resulting in a potential disparity in offspring size of up to 20 weeks growth. In addition, 1-year-old breeders do not come into oestrus until late April/May (H. Hesterman, S. Jones & M. Jones, unpublished), presumably the earliest time since weaning in January at which they are able to attain a critical size and sexual maturity. Thus, it seems that some individuals, the offspring of late breeders and particularly 1-year-old breeders, will be less likely to attain the critical size necessary to breed in their first year. Although this conclusion remains to be tested, it may partly explain why we did not record precocial breeding in 2007: most of these individuals would have been the offspring of precocial breeders [more than 50% (12/23) of breeders captured in 2006 were 1 year old] and thus born relatively late in the season. This constraint on the probability that individuals will be large enough to breed in their first year will severely limit the strength of this reproductive compensatory response to declines in population abundance.

In addition to the timing of reproduction, maternal effects, in which the mother's phenotype shapes her offspring's phenotype independent of any genetic effects (Bernardo 1996), may also influence the capacity of females to reach a critical body size and thus, their ability to breed in their first year. Of particular interest is the effect a female's disease status might have on the condition (phenotype) of her offspring. If, as seems likely, the offspring of diseased mothers are adversely affected by her condition (assuming she survives long enough to raise them), then this might also explain the absence of precocial breeding in 2007; since almost half (11/23) the females breeding in 2006 [and 82% (9/11) of adult females breeding in this year] were diseased. We currently lack the necessary data to examine the consequence of maternal disease status for offspring condition and this effect remains to be investigated. Nonetheless, this would appear to further constrain the likelihood that individuals breed as 1-year-olds.

A key result of this study was that diseased females had significantly more female-biased litters than did healthy females, suggesting that DFTD infection leads to sex allocation biases in Tasmanian devils. The theory of adaptive sex allocation states that in polygynous mammals that exhibit reproductive skew, mothers in poor physical condition, or breeding in poor environmental conditions, are expected to invest more in daughters than in sons, since the costs of producing and rearing quality sons are much greater than for producing quality daughters (Trivers & Willard 1973). In humans, studies have shown that diseases (including cancer) can lead to female-biased offspring sex ratios (James 2002). We are not aware, however, of any other study in a nonhuman system that shows that an infectious disease can alter maternal sex allocation or investment in offspring. If the change in offspring sex ratio that accompanied disease infection in this study occurs throughout all disease-affected devil populations, it would provide novel evidence that adaptive, facultative adjustment of offspring sex ratio occurs in response to disease-induced changes in maternal condition in a polygynous mammal. Mechanisms facilitating offspring sex ratio manipulation are still the source of speculation and largely unknown (Johnson & Ritchie 2002; Pike 2005), although studies highlight the role of stress-related disruptions to endocrine function due to environmental perturbation (Pike & Petrie 2006) or the physiological challenge of a disease (James 2001). The possibility that DFTD infection alters hormone levels in devils subsequently affecting pouch young sex ratios clearly warrants further investigation.

A facultative adjustment to female-biased pouch young sex ratios, if propagated through the population, could potentially enhance population growth and assist population recovery because theoretically, the most productive populations are those with a female-biased sex ratio (Caughley 1977). Diseased females, however, may not survive to rear their offspring successfully. It therefore seems doubtful that there will be long-term consequences for the population dynamics or persistence of this population as a result of the change in this life-history parameter.

This study found that an epidemic of an infectious disease resulted in major changes to both population age structure and life-history parameters of Tasmanian devils. Reproductive compensation occurred in response to disease impacts via an increase in precocial breeding. The strength of this compensatory response, however, was shown to be limited by factors

such as the timing of reproduction and maternal effects, which constrain the ability of offspring to reach a critical size for maturity within the time frame dictated by the annual breeding season. Constraints on the ability of individuals to breed as 1-year-olds mean that most individuals in this population will have only one opportunity to breed in their lifetime, at 2 years of age. The average age of diseased individuals in this population is 2·27 (95% CI 1·99–2·56) years. Since individuals rarely survive for more than 6 months with disease (Lachish *et al.* 2007) and juveniles only gain independence at 9 months of age, these adults will be unlikely to rear that one litter successfully and thus reproductive success and recruitment will be low.

With precocial breeding reliant on rapid growth in the first year, the opportunity for selection to favour fast-growing individuals that are able to breed earlier is evident, provided there is a heritable basis for rapid early growth in devils. The Freycinet population of devils now exhibits many of the conditions necessary for selection to favour early reproduction (Charnov & Schaffer 1973; Jones *et al.* 2008), with substantial reductions in adult survival rates (Lachish *et al.* 2007) and higher disease prevalence among adults than young individuals (H. McCallum, M. Jones, C. Hawkins, R. Hamede, S. Lachish, D. Sinn, N. Beeton & B. Lazenby, unpublished). The possibility that evolution will drive a shift towards this new life-history pattern in Tasmanian devils potentially increases the likelihood of future population persistence and supports a growing body of evidence indicating that infectious diseases may be strong selective forces for life-history variation and evolution in a range of taxa (Altizer, Harvell & Friedle 2003; Todd 2007; Bolker *et al.* 2008).

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