Life-history change in disease-ravaged Tasmanian devil populations

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In this article, we investigate changes in life history in Tasmanian devil populations affected by the facial tumor disease. We analyzed data from five study sites where individually marked devil populations have been studied both before and after the arrival of the disease. Changes in precocial sexual maturity are documented in relation to changes in population age structure and consequent lifetime number of breeding events that result from the disease-induced increase in adult mortality.

Results

Disease has had a highly significant effect on the age structure of the devil populations at all five sites ($\chi^2 = -5.52, df = 1, P < 0.05$).

Author contributions: M.E.J. designed research; M.E.J., R.H., C.H., H.H., S.L., D.M., and D.P. performed research; M.E.J. and H.M. analyzed data; and M.E.J. and A.C. wrote the paper.

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0.001), with a much higher proportion of older (3+ years) animals present in the population before the disease (log odds ratio of being old vs. young before and after disease = –1.774, 95% C.I. –2.428 to –1.257; Fig. 3a). The final model included only disease (fixed effect) and between-year variation within site (random effect). There were no consistent differences between sexes (removal of sex term from model: $\chi^2 = 2.728$, df = 1, $P = 0.099$) or among sites (removal of site term from model: $\chi^2 = 7.663$, df = 4, $P = 0.105$).

Before the disease emergence at Freycinet, a significantly greater proportion of females (21 of a total of 29) produced more than one litter in their lifetime than after the disease spread across the site (6 of 17; Fisher’s exact test, $P = 0.028$; Fig. 4). Precocial breeding by 1-year-old females increased substantially after disease emergence (log odds ratio of 1-year-old breeding vs. not breeding before and after disease = –3.279, 95% C.I. –5.147 to –1.728; $\chi^2 = –4.057$, df = 1, $P < 0.001$; Fig. 3b). Before the disease, between zero (at three sites) and 12.5% (year 2000 at Freycinet) of 1-year-old females bred. After the disease spread, precocial breeding increased on average 16-fold to between 13.3% (Mt. William, 2004) and 83.3% (also Mt. William, 2006), except at Little Swanport, where it remained at zero. The final model included only disease (fixed effect) and between-year variation within site (random effect). There were no statistically significant differences attributable to site (removal of site term from model: $\chi^2 = 7.437$, df = 4, $P = 0.115$).

**Discussion**

The arrival of DFTD on the Freycinet Peninsula in 2001 triggered an immediate and steady decline in survival rates (16% of at least 60% compared with former levels (10), resulting in a population comprised almost entirely of animals <3 years of age. Before the disease, the modal female began seasonal breeding at age 2 and produced a litter annually for 3 years, with senescence and death occurring in her fifth or sixth year (M.E.J., unpublished work) (17, 18). Females now generally have one breeding
opportunity and may not survive long enough to rear that litter. Hence, they are now largely semelparous.

Comparable changes in age structure have occurred at four additional sites where data before and after disease invasion were collected. At Mt. William National Park, where the disease was first recognized 10 years ago, the population has declined by an estimated 89% following arrival of disease (10).

Substantial increases in precocial breeding by 1-year-old females occurred at four of the five sites. A cause for the lack of increase in precocial breeding at the Little Swanport site after disease arrival cannot be determined given the snapshot nature of the data and may be due to chance. Habitat is equivalent to other sites. We lack the data to test whether the disease status of breeding females the previous year affected growth and survival of the 1-year-old cohort of potential precocial breeders. Tasmanian devils have shown their capacity to respond to this disease-induced increased adult mortality with precocious sexual maturity. A mechanism that might facilitate early breeding is more rapid growth in response to reduced population density and probably reduced food competition (4). A reduction in interference competition from older, more dominant females could also play a role, although it is unlikely to produce such dramatic results. Most adult devils breed in any one year (S.L., H.M., and M.E.J., unpublished work), even small, subordinate 2-year-old females. In addition, before DFTD emergence, records of precocial breeding were rare, and records were restricted to sites with good soil and plentiful prey, suggesting that resource levels may play a role.

Life-history theory predicts that selection for early reproduction will occur (6). Demographic responses can, over time, evolve into new, genetically mediated life-history parameters (1). Tasmanian devils are the largest member of a marsupial clade famous for intense investment in early reproduction at the expense of longevity, exemplified by the repeated evolution of semelparity (20). Devils share with semelparous carnivorous marsupials (20) several traits that may predispose them to an emergent shift in breeding toward precociality when semelparity is imposed. They breed few times and reproduction is costly. Over the mating season, males experience 25% weight loss (M.E.J., unpublished work) (18), anemia (M.E.J., unpublished work), and often a reduction in immunocompetence (B. Burton and G. Woods, unpublished work). Devils are thus an excellent model species with an interesting new disease, which behaves like a parasite in that it is a piece of devil that propagates itself to another devil, in which to pursue questions of adaptation.

Rapid evolution on ecological time scales is now widely recognized in natural ecosystems (21), including resistance to parasites (22). There is accumulating evidence that adaptation to strong directional selection can contribute to the persistence of populations (23). Whereas the presence of genetic variation in populations needed for traits such as age at maturation is well established (24–27), of emerging interest is the role of phenotypic plasticity in adaptive responses. If a particular form of phenotypic plasticity enhances an organism’s probability of surviving directional selection at the population level, it may lead to an adaptive evolutionary response (28). A pertinent example that demonstrates life-history change under directional selection is increased mortality under harvesting pressure inducing early maturation in cod (2). Although such evolutionary responses are predicted, and demographic responses in age at maturity to
harvesting pressure are documented (29), there is little empirical evidence for this phenomenon in mammals (27).

Here, we have a persistent situation of almost complete mortality of individuals in their first year of adult life from a new disease that is likely to become a permanent feature of devil biology. Phenotypic plasticity in life-history traits in Tasmanian devils is evident in the variation in precocial breeding among populations and years before the emergence of DFTD. Any ability in devils to increase lifetime reproductive output beyond one litter, or indeed to rear a single litter to independence before death from cancer, should also enhance the fitness of those individuals. Although MHC gene diversity is very low in Tasmanian devils in eastern Tasmania (13), this is not universal across either the genome or the island (e.g., neutral microsatellite loci) (30), and there is some diversity on which selection can operate. At this stage, all we have evidence for is a phenotypic response. We suggest that DFTD is a novel, strong selective agent for life-history change that might lead to rapid adaptation toward a population genetically and demographically more robust to the effects of DFTD.

To our knowledge, this is the first known case of infectious disease leading to increased early reproduction in a mammal. The persistence of this disease and ongoing population decline is of profound conservation concern for a unique endemic carnivore (10). Although the ability to switch to precocious reproduction offers some prospect for persistence and recovery, the prognosis for this iconic species remains uncertain.

Methods
We used data from five study sites that broadly represent the current geographic extent of the disease and where populations were trapped and individually marked both before and after the emergence of DFTD (Fig. 2). A 160-km² site on the Freycinet peninsula on the east coast of Tasmania was continually monitored from 1999 to 2007. The disease was first detected at the northern end of the peninsula in July 2001 and progressively spread southwards down the peninsula. Devil populations over the entire geographic area used in these analyses were affected by 2005. We compared “before disease” data from 2000 and 2001 (n = 110:129 males:females; n = 65 1-year-old females) only with “after” data from 2005 and 2006 (n = 45:48 males:females; n = 33 1-year-old females) because data from the intervening years included parts of the study site that were diseased combined with areas where the disease had not yet spread. At Mt. William National Park in northeast Tasmania, where the disease signs were first detected in 1996 (9), data from 2004 to 2006 (n = 39:35 males:females; n = 28 1-year-old females) was compared with that from an earlier study in 1983–1985 (n = 87:51 males:females; n = 42 1-year-old females) (18). Postdisease trapping data collected in 2006 were compared with 1999 data (30) for two other sites in southeast Tasmania: Little Swanport, which was in the early stages of disease invasion in 1999 (before: n = 17:13 males:females; n = 13 1-year-old females; after: n = 21:11, n = 8), and Pavileena, where the disease was first detected in 2002 (before: n = 13:16 males:females; n = 5 1-year-old females; after: n = 15:7, n = 7). Wisedale in northern Tasmania is near the current disease front and has been monitored to coveacute;epidemiological parameters since the early stages of disease invasion in 2006. Data from 2006 and 2007 are presented (before: n = 21:33 males:females; n = 9 1-year-old females; after: n = 30:18, n = 14).

Populations were monitored by trapping conducted over 7 (Freycinet) or 10 (all other sites) consecutive nights at each study site, using specially designed carnivore traps baited with meat. Wire-cage traps were used until 2004, and PVC-pipe traps were used thereafter. After the disease emerged, biosecurity protocols were used to reduce the chance of the trapping program contributing to the spread of the disease. Devils were gently emptied into sacks, individually marked with ear tattoos (before 2004) or microchips (from 2004 to 2007), weighed and measured, and assessed for age and reproductive condition. Age was estimated by using a combination of molar tooth eruption, molar and canine tooth wear, canine over-eruption, body weight, and general appearance (M.E.J., unpublished work). The reproductive condition of females was assessed upon inspection of the pouch for pouch young or active lactation (enlarged teats and mammary glands). Field trapping of devils was approved by the Animal Ethics Committees of the University of Tasmania, the Australian National University, the University of Queensland, and the Tasmanian Department of Primary Industry and Water. These are established ethics committees which have an undertaking that the provisions of the Australian Code of Practice for the Care and Use of Animals for Scientific Purposes, referred to in the Statement of Animal Experimentation, will be observed.

Most female devils reach sexual maturity at the age of 2 and few live beyond 5 years of age in the wild (M.E.J., unpublished work). Breeding is seasonal, with the majority of births in March and April (31). The population was thus divided into young animals, defined as independent 1- and 2-year-olds before March of the year they turn 3, and devils older than 2. The aging techniques used are definitive to the age of 2 years, which is beyond the age of precocial breeding (14 months), and reliable to the age of 3, after which individual variance in tooth wear, and consequently over-eruption, reduces precision. To analyze the effect of DFTD on the proportion of females that breed more than once in their lifetime, we used only data from Freycinet, where detailed information on life history, including entire individual lifespans, has been collected since 1999 (before:after n = 29:12). For this analysis, reproductive events over the entire lifespan of individual females were examined for females that were present in 2000, 2001, 2005, and 2006. All analyses were performed by using R version 2.5.0. The age structure and precocial breeding data were analyzed by using an REML-based generalized linear mixed model (lmer in R package lme4) with binomial error and a logistic link, using disease status and site as fixed effects and year within site as a random effect. The models were simplified by removing terms along lines recommended by Crawley (2005). The effects of removing terms are reported as χ² distributed likelihood ratios comparing models, and the effects of the fixed effects on the response variable are reported as Wald statistics (estimate divided by standard error). An mcmcreampling procedure using 1,000 iterations was used to provide confidence intervals for the effect of the disease, the effect of the log odds of being young rather than old, and the effect of breeding at age 1 or not before and after the disease. Mixed models were used because of significant residual variation associated with the year within site term when this term was included as a fixed effect in an entirely fixed-effects model. Lack of any two- or three-way interactions in the fixed-effects models also confirmed that no important inter-actions had been neglected. The proportion of all known adult females producing more than one litter in their lifetime before and after the disease on the Freycinet peninsula was analyzed by using Fisher’s exact test.

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![Fig. 4. Change in the proportion of all known adult females producing more than one litter in their lifetime before and after invasion of DFTD at Freycinet in eastern Tasmania.](image-url)
University Faculties Research Grants Scheme (F02085 to M.E.J. and A.C.), and the Tasmanian Government Save the Tasmanian Devil Program.

31. Hesterman H (2007) Reproductive physiology of the Tasmanian devil (Sarcophilus harrisii) and spotted-tailed quoll (Dasyurus maculatus). PhD thesis (Univ of Tasmania, Hobart).