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Effects of juvenile and adult diet on ageing and reproductive effort of male and female black field crickets, *Teleogryllus commodus*

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Summary

1. How and when resources are allocated to reproduction is expected to differ between the sexes, potentially generating differences in how males and females age. For this reason, acquisition of resources should be an important determinant of both age-dependent reproductive effort and of deteriorative ageing (i.e. senescence).

2. We used black field crickets, *Teleogryllus commodus*, to test whether differences in diet quality of juveniles and adults determine sex-specific resource allocation to reproduction and whether there are any subsequent effects on ageing.

3. We show that ageing does not depend on nymph diet. There was, however, a significant difference in the rates of actuarial ageing for males and females. Females showed reproductive ageing, whereas male reproductive effort plateaus or continues to increase with age.

4. These results highlight the link between diet, reproduction and ageing and show that differences in resource utilization between the sexes can lead to different patterns of ageing. This is likely to have profound effects on how life-histories evolve in the sexes.

Key-words: age-dependent mortality, life-history, reproductive ageing, senescence, sexual conflict

Introduction

Life-history theory is based on the trade-offs that arise as limited resources are allocated to current reproduction, somatic maintenance and other fitness components (Houle 1991; Stearns 1992; Roff 2001). These trade-offs are also likely to be responsible for ageing. The trade-off between somatic maintenance and reproduction is the basis for the 'disposable soma' theory of senescence (Kirkwood 1977; Kirkwood & Rose 1991). This states that when there are extrinsic sources of mortality it is better to invest some resources into traits that increase current reproductive success even if this makes somatic maintenance less efficient. The underlying argument is that it is more advantageous to invest a unit of resources into reproduction than maintenance if the average cost of the resulting somatic decline is less than the average benefit from an increased reproductive rate. When extrinsic mortality rate is very high, very few individuals actually pay a cost and die due to somatic decline because most succumb earlier to extrinsic sources of mortality. In contrast, all individuals benefit from elevated reproductive output early in life. More generally, traits that elevate reproductive success early in life will be favoured by selection even if there is a trade-off so that they reduce fitness later in life (the 'antagonistic pleiotropy' theory of ageing; Williams 1957). The exact amount invested into current success will, of course, partly depend on the rate of extrinsic mortality. A difference is that harmful mutations that act later in life are less strongly selected against than those acting early in life, and therefore can accumulate over the course of evolution (the 'mutation accumulation' theory of ageing; Medawar 1952). The effects of accumulated deleterious alleles should, in theory, not be sex-specific.

Life-history theory predicts the optimal strategy for allocating resources among traits as an individual ages, but this is expected to differ between the sexes (Wedell *et al.* 2006). Females typically produce resource-rich gametes but are not required to invest heavily in attracting mates (Arnqvist & Nilsson 2000), whereas the opposite tends to be true for males (Simmons 2001). This fundamental sexual difference is expected to generate evolutionary conflict between the sexes in their optimal reproductive strategies (Parker 2006)

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and there is ample empirical evidence for sexual differences in the costs of reproduction (Kotiaho & Simmons 2003; Promislow 2003; Fedorka *et al.* 2004). In principle, these factors could simply generate a consistent difference in the proportion of resources allocated to reproductive effort by each sex at all ages. However, when trade-offs between current and future reproduction are combined with age-specific selection this is likely to result in sexual differences in how investment patterns change with age. Ultimately, different patterns of investment might explain the widely observed sexual differences in life span and senescence (Carey *et al*. 1995; Owens 2002; Fox *et al.* 2003). Surprisingly, the extent to which sex differences in ageing are due to differences in age-dependent reproductive effort remains a relatively unexplored question in evolutionary biology.

Reproductive effort is often condition-dependent because it depends on the acquisition and allocation of available resources (Van Noordwijk & Dejong 1986; Reznick *et al.* 2000; Hunt *et al*. 2004a). Consequently, adult diet should have an important effect on life-history schedules and this has been well-documented in many species (Tatar & Carey 1995; Chapman & Partridge 1996; Hunt *et al*. 2004b; Romanyukha *et al*. 2004; Hunt *et al*. 2006; Johnston *et al*. 2006). Of particular importance is the effect that dietary restriction has on patterns of ageing in many animal taxa, ranging from *Caenorhabditis elegans* (Gardner *et al.* 2004; Lee *et al*. 2006) and *Drosophila melanogaster* (Mair *et al*. 2003; Magwere *et al.* 2004) to crickets (Hunt *et al*. 2004b; Maklakov *et al*. 2008) and mice (Weindruch *et al*. 1986; Dhahbi *et al*. 2004). In general, reduced dietary intake is associated with greater longevity and lower fecundity in females (but see Johnston *et al*. 2006), but a reverse pattern has been shown in males (Hunt *et al*. 2004b). Whether male and female ageing is influenced by diet in a similar way remains an open empirical question (but see Magwere *et al*. 2004).

The effects of diet are unlikely to be independent of age. There is, for example, a large literature showing that limited access to resources early in life can have irreversible detrimental effects on adult performance (Metcalfe & Monaghan 2001). In many cases, after periods of poor nutrition, animals can 'catch up' with the size of conspecifics that had good quality nutrition, by accelerating their growth (Metcalfe & Monaghan 2001). But this compensatory growth is often costly, although this is only evident in the long-term (Metcalfe & Monaghan 2003; Stoks *et al.* 2006). Nymph diet has been shown to affect traits expressed during adulthood in several arthropod species (Gray & Eckhardt 2001; Kaspi *et al*. 2002; Boggs & Freeman 2005). In contrast, there are often no detectable long-term effects of temporary starvation or food shortages for adults (Mair *et al*. 2003).

The black field cricket, *Teleogryllus commodus* (see Fig. 1), has proved an exceptionally useful organism for studying the relationship between age-dependent reproductive effort and ageing because it is possible to measure the most important components of both male and female reproductive effort. In contrast in most species it is a major technical challenge to measure male reproductive effort. In *T. commodus*, producing

Fig. 1. A male (bottom) and a female (top) black field cricket, *Teleogryllus commodus*.

advertisement calls (or calling effort) is the most costly form of male reproductive effort (Hunt *et al*. 2004b) and is subject to intense sexual selection (Bentsen *et al*. 2006). Both male calling effort and female egg production can be accurately quantified in the laboratory using a multi-channel event recorder (Hunt *et al*. 2004b) and by counting eggs, respectively (Hunt *et al*. 2006; Zajitschek *et al*. 2007).

In *T. commodus* there is a complex relationship between male reproductive effort and diet. Males fed a nutrient-rich diet that was characterized by high protein content from birth onward start to call at a younger age and produce more calls over their lifetime than those fed a nutrient-poor diet with low protein content. This is true despite males on a nutrient-rich diet having shorter life spans than those on a nutrient-poor diet (Hunt *et al*. 2004b). The possibility that higher quality males (based on the assumption that higher nutrient diets increase net fitness) can be more attractive (i.e. call more) but show reduced longevity was predicted by several recent theoretical models (Hansen & Price 1995; Kokko *et al*. 2002). While a recent study has looked in more detail at the relationship between longevity and reproductive effort, using diets with a range of different ratios of protein to carbohydrate content (Maklakov *et al*. 2008), to date it is unclear whether the effects observed in both males and females depend on the diet during the nymph stage, or can be solely attributable to the adult diet. There is, for example, a large literature on condition-dependence showing that adult diet alone can affect the expression of sexually selected traits, especially behavioural ones like advertisement calling (Aluja *et al.* 2001; Holzer *et al.* 2003).

In this study, we examine whether the nutrient content of nymph and adult diet affects the intensity of male sexual signalling, female fecundity and both sexes' longevity. Age-dependent declines in reproductive effort and survival rates are both important signatures of ageing (hereafter called reproductive and actuarial ageing respectively), so we also tested whether they are affected by nymph and adult diet and how they differ between the sexes. We manipulated diet

quality in a two-way factorial design experiment, testing the effect of high and low amounts of nutrients fed to nymphs and to adults of *T. commodus*.

Materials and methods

EXPERIMENTAL DESIGN

To manipulate the resource acquisition of crickets we created two diets that were characterized by low and high levels of a range of nutrients. These two diets were selected as previous work on *T. commodus* showed that they have substantial effects on morphology, life-history traits, behaviour, levels of age-dependent sexual advertisement and longevity (Hunt *et al*. 2004b; Hunt *et al*. 2006). The nutrient-poor diet consisted of 50% fish food (Pisces Enterprises, 45% protein, 10% fat, 17·34 MJ kg^{-1} gross energy) and 50% oatmeal (Farmland, 10·5% protein, 8% fat, 16 MJ kg^{-1} gross energy), whereas the high-nutrient diet contained 100% fish food. Oatmeal and fish food were ground, mixed with water and formed into pellets (121 ± 2 mg) by putting the mixture into a perspex mould and drying it for 12 h at 60 °C. The most substantial difference between the two diets is their protein content, however, fish food and oatmeal differ in other respects (e.g. vitamins and minerals, oils of marine and vegetable origin). This means that we did not exclusively manipulate protein content *per se*. Therefore we refer to the two diets as nutrient-rich (H) and nutrient-poor (L) respectively.

We manipulated diet with a two-way factorial design. One hundred randomly mated females from our Smith's Lake (NSW, Australia) laboratory stock cultures were isolated in individual plastic containers (5 × 5 × 5 cm) with a Petridish containing moist cotton wool for oviposition. A total of 591 cricket nymphs were then collected within 24 h of hatching. Nymphs were randomly assigned to the low (L) or the high-nutrient diet (H), and raised individually in clear plastic containers (5 × 5 × 5 cm) that contained a piece of egg carton for shelter, a plastic water bottle plugged with cotton wool and three food pellets. Each week containers were cleaned and provided with fresh water and food and we noted nymph survival or death in the intervening period.

From the fifth larval instar onwards, we checked daily for eclosion to adulthood. On the day of eclosion, body weight and pronotum width were measured using a high precision electronic balance (Mettler-Toledo, model AG135) and an eyepiece graticule in a binocular microscope (Leica MZ5), respectively. Animals were then assigned an adult diet. We either maintained the nymph diet (LL or HH) or changed to the alternate diet (LH or HL). Adults were maintained in an identical manner to nymphs, with the containers cleaned and fresh food and water added weekly. Adult survival was checked daily.

MALE REPRODUCTIVE EFFORT

We quantified male reproductive effort as the amount of time the male spent broadcasting his sexual advertisement call. Each male's calling effort was measured every 5 days until his death, starting at 4 days post-eclosion. Calling effort was measured using an electronic event recorder, equipped with 64 channels connected to individual microphones mounted in the lid of the cricket's container. This device sampled each of the 64 channels (representing 64 individual males) 10 times per second and recorded whether or not the male called. Full details of this recording device are provided in the supplement of Hunt *et al*. (2004b). All males had their nightly calling effort recorded from 6 pm to 9 am. In total 250 males successfully eclosed, but six died before we could measure their calling effort.

FEMALE REPRODUCTIVE EFFORT

We quantified female reproductive effort as lifetime egg production. Females were given the opportunity to mate with a random stock male 7 days after eclosion by adding a male to their container overnight (6 pm–9 am). Female *T. commodus* receive no nuptial gifts. They can potentially receive and store enough sperm for a sufficient lifetime sperm supply from one mating (Bussière *et al*. 2006). However, like in many other gryllid crickets, the recruitment of sperm for fertilizations will depend on both the relative abundance and viability of stored sperm (Sakaluk & Eggert 1996; Garcia-Gonzalez & Simmons 2005). Therefore, to ensure that females were not sperm limited and did not have a lack of viable sperm in storage, a new male was provided for mating every seventh day. We gave each female a Petri dish filled with moist sand for 5 days after each mating to oviposit. Eggs were collected by swirling the samples in water to separate eggs from sand. They were then counted. In total 249 females successfully eclosed and four died before their first mating.

STATISTICAL ANALYSIS

To analyse differences in life span between treatments, we used analyses of variance and Cox regression survival analysis (COXREG procedure, SPSS 16). Confidence intervals for median longevity were bootstrapped using PopTools v2·6·2 (Hood 2004) by randomly drawing 80% of the original sample (with replacement) in 1000 permutations and calculating the median longevity. The intervals correspond to the upper and lower 2·5% of the range of values in these redrawn subsamples.

To analyse age-specific mortality rates, we fitted four commonly used statistical models (Gompertz, Gompertz-Makeham, Logistic, Logistic-Makeham) to the mortality data of each treatment group using maximum likelihood implemented in WinModest (Pletcher 1999). These analyses were performed separately for each sex. We excluded very late age groups with less than five survivors. The best model was selected using log-likelihood ratio tests. It is possible to use log-likelihood ratio tests here, because the four candidate models are hierarchical and nested, that is, the most complex – the Logistic-Makeham model contains parameters that, when set to zero, reduce the model to the less complex remaining three models respectively. For seven of the eight sex-diet groups, the Gompertz model provided the best fit. The logistic model provided a marginally better fit for one group (log-likelihood ratio test between Gompertz and Logistic model for HH males, $P = 0{\cdot}047$). We therefore estimated baseline mortality and any increase in age-specific mortality with age for all groups with the Gompertz model, given by

$$\mu_x = \alpha e^{\beta x}$$

where x is age, μ_x is an estimate of the hazard rate h_x, which is a measure of age-dependent mortality in any specific age interval that is not influenced by individuals dying in the intervals preceding or following this interval. The parameter α is interpreted as the age-independent baseline mortality. Parameter β estimates the age-dependent increase in mortality rate, and is our estimate of senescence expressed as the change in mortality rates over time. To test whether α or β differed between two groups, we constrained two models to have either the same α or the same β, and then compared the resulting log-likelihood with the unconstrained model using log-likelihood ratio tests. The 95% confidence intervals for the estimate of the Gompertz parameter β were calculated in WinModest (Pletcher 1999).

Table 1. MANOVA for development time, and weight and size at eclosion including both sexes (a) and separate ANOVAs for males (b) and females (c)

(a) Both sexes	Pillai's trace		$F_{3,493}$	P
Sex	0·094		17·128	< 0·001
Nymph diet	0·028		4·735	0·003
Sex × Nymph diet	0·016		2·624	0·050
(b) Males	Low nutrient	High nutrient	$F_{1,248}$	
Development time (day)	61·81 ± 0·75	60·18 ± 0·49	3·276	0·071
Weight at eclosion (mg)	656·95 ± 11·37	714·32 ± 9·72	14·621	<0·001
Size at eclosion (mm)	6·85 ± 0·05	6·95 ± 0·06	1·489	0·224
(c) Females			$F_{1,247}$	
Development time (day)	57·89 ± 0·74	57·71 ± 0·55	0·038	0·845
Weight at eclosion (mg)	630·09 ± 11·83	643·08 ± 9·68	0·730	0·394
Size at eclosion (mm)	6·65 ± 0·04	6·70 ± 0·03	0·932	0·335

Means ± SE: variable means ± standard errors of the means for low nutrient and high nutrient nymph diet treatments.

To analyse data on male and female age-dependent reproductive effort (calling effort and fecundity) that were not normally distributed we used generalized estimation equations (GEE) with a negative binomial error structure and link function, and a first-order auto-regressive correlation structure that specifies the correlation of repeated measures within each individual (GENLIN procedure; SPSS 16). The few crickets (6 males, 4 females) that died before their first measurement of reproductive effort were excluded from our analyses. Separately for each sex, we started fitting full models with reproductive effort as the response variable, and nymph diet, adult diet, age at measurement and life span, and their quadratic values as independent variables. We also included all possible two- and three-way interaction terms between these variables (except for interactions between the quadratic covariate terms), making the models completely factorial. For model selection, we used an information-criterion based on quasi-likelihood (QICu, Pan 2001), resulting in smaller values of QICu for more parsimonious models, that is, compensating for the complexity of models. For a graphic presentation of the relationship of reproductive effort with treatment, age and life span, we fitted nonparametric thin-plate splines using the TPS function in the fields package in R 2·7·2 (R Development Core Team 2007).

For each male, lifetime reproductive effort was the sum of all measurements of nightly calling effort taken during his entire life. It was $\log_e + 1$ transformed to ensure normality. For each female, lifetime reproductive effort was the total number of eggs she laid. We tested the effects of diet treatment on lifetime reproductive effort separately for each sex using ANCOVA with nymph and adult diet treatment and their interaction as fixed effects, and life span as a covariate. To test for equal slopes among treatment groups, we included interactions between life span and the fixed factors and their interaction term. Non-significant interactions with life span were excluded from a model before it was run again. Unless otherwise stated summary statistics are presented as mean ± SE.

Table 2. Cox regression survival analyses: complete full factorial model (a) and effects of nymph diet and adult diet in males and females (b, c)

	d.f.	χ^2/Wald*	P
(a) Both sexes			
Overall model	7	232·605	<0·001
Sex	1	4·545	0·033
Nymph diet	1	0·004	0·951
Adult diet	1	55·551	<0·001
Sex × Nymph diet	1	0·1	0·996
Sex × Adult diet	1	0·001	<0·001
Nymph diet × Adult diet	1	42·261	0·751
Sex × Nymph diet × Adult diet	1	1·436	0·231
(b) Males			
Overall model	3	4·074	0·254
Nymph diet	1	0·031	0·859
Adult diet	1	2·326	0·127
Nymph diet × Adult diet	1	1·948	0·163
(c) Females			
Overall model	3	119·248	<0·001
Nymph diet	1	0·011	0·917
Adult diet	1	62·379	<0·001
Nymph diet × Adult diet	1	0·179	0·672

*χ^2 for overall model, Wald's for separate variables.

Results

NYMPH SURVIVAL, DEVELOPMENT TIME AND ADULT MORPHOLOGY

Nymph diet did not significantly affect survival to adult eclosion ($\chi^2_1 = 0{\cdot}176$, $P = 0{\cdot}675$; percentage of individuals that died: LL + LH = 16·84%, HH + HL = 14·29%).

Males fed a high-nutrient diet as nymphs weighed significantly more at eclosion than those fed a low-nutrient diet (Table 1). There was no effect of diet on female weight at eclosion. There were no significant effects of nymph diet on development time and pronotum width for either sex (Table 1). On average, females eclosed earlier (sex-differences: low-nutrient: 3·92 days, high-nutrient: 2·47 days) into adulthood than males. Males were larger (sex-differences: low-nutrient: 0·19 mm, high-nutrient: 0·24 mm) and heavier (sex-differences: low-nutrient: 26·86 mg, high-nutrient: 71·24 mg) as newly eclosed adults than females.

ADULT LONGEVITY

Survival rates

We tested whether adult longevity differed between the sexes and how it was affected by diet using a Cox regression survival analysis. There was no effect of nymph diet on life span for either sex. There was, however, a significant effect of adult diet on longevity that differed between the sexes (Table 2). There

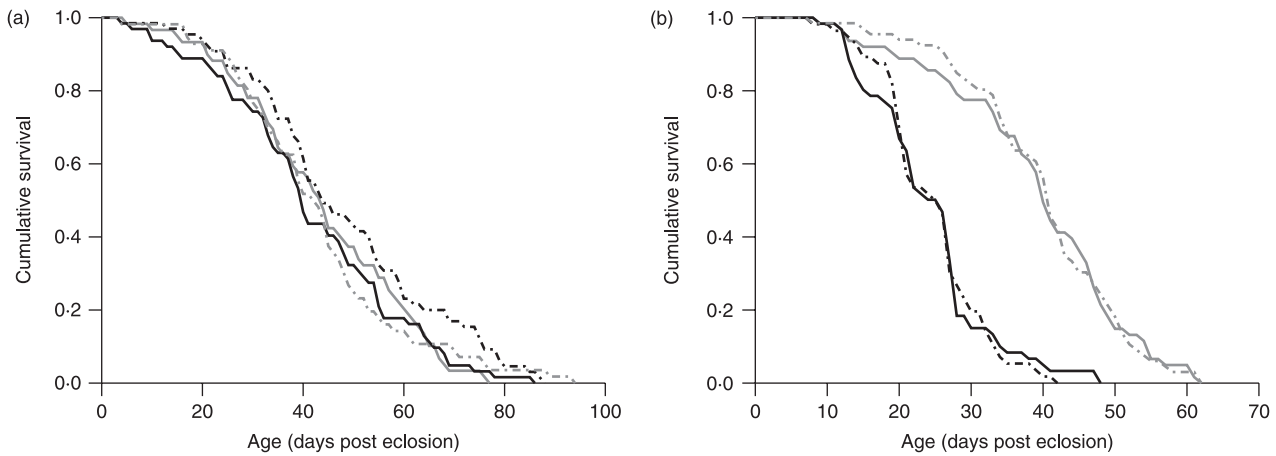

Fig. 2. Survival curves for males (a), and females (b). Treatment groups: LL (solid, grey), HH (solid, black), LH (dashed, black), HL (dashed, grey).

Table 3. Pairwise tests of differences in mortality rates between diet treatments and between the sexes

	Treatments			Gompertz parameters and *P* values					
Comparisons	1	2	Sex	α (1)	α (2)	P_α	β (1)	β (2)	P_β
Overall diet	LL	HH	m	0·003	0·004		0·066	0·058	
	LL	HH	f	0·001	0·003		0·105	0·152	*
Adult diet	LL	LH	m	0·003	0·003		0·066	0·053	
	LL	LH	f	0·001	0·002		0·105	0·181	**
	HH	HL	m	0·004	0·003		0·058	0·069	
	HH	HL	f	0·003	0·001	**	0·152	0·122	
Nymph diet	LL	HL	m	0·003	0·003		0·066	0·069	
	LL	HL	f	0·001	0·001		0·105	0·122	
	HH	LH	m	0·004	0·003		0·058	0·053	
	HH	LH	f	0·003	0·002		0·152	0·181	
Between sexes	LL(m)	LL(f)	mf	0·003	0·001		0·066	0·105	**
	HH(m)	HH(f)	mf	0·004	0·003		0·058	0·152	**
	LH(m)	LH(f)	mf	0·003	0·002		0·053	0·181	**
	HL(m)	HL(f)	mf	0·003	0·001	**	0·069	0·122	**

Gompertz-parameter α (baseline mortality), and β (the rate of ageing) were calculated and used in significance tests with a precision of five decimal places. Values in the table were rounded to three decimal places for presentation.
Treatment numbers 1 and 2 identify the specific treatment and its α and β, e.g. in the first row, α(1) = 0·003 is the estimated α for LL males.
m : male, f : female, mf : comparison between male and female.
Stars depict pairs of parameters with significantly different parameter values: * $P < 0{\cdot}05$, ** $P < 0{\cdot}01$. *P*-values are from log-likelihood ratio tests (See *Methods*).

was no interaction between nymph and adult diet. To further examine how adult diet affected each sex, we performed separate analyses for males and females. Adult diet did not affect male longevity (Table 2; Fig. 2a; male life span: LL + HL: 42·29 ± 1·6 days, HH + LH: 42·08 ± 1·78 days), whereas females on a low-nutrient diet lived significantly longer than those on a high-nutrient diet (Table 2; Fig. 2b; female life span: LL + HL: 37·78 ± 0·72 days, HH + LH: 23·47 ± 1·10 days).

Age-specific mortality rates

We compared the effects of overall diet (LL vs. HH), nymph diet (LL vs. HL, HH vs. LH) and adult diet (LL vs. LH, HH vs. HL) on the age-dependent mortality rate, given by the Gompertz parameter β, running separate analyses for each sex. Males showed no difference in β between the treatments (Table 3). Females showed a more rapid age-dependent increase in mortality rate when they had high-nutrient food as adults, but this was only significant when tested against LL females, with a similar trend in the same direction in the comparison against HH females (Table 3). The age-independent baseline mortality rate, estimated by α, differed in only one of 10 within-sex comparisons (HH vs. HL females). In general, there was no strong and consistent effect of diet on age-specific or baseline mortality.

To compare patterns of senescence between males and females, we tested whether there was a difference in β between males and females within diet treatment groups. In all four treatments β was significantly higher in females than in males

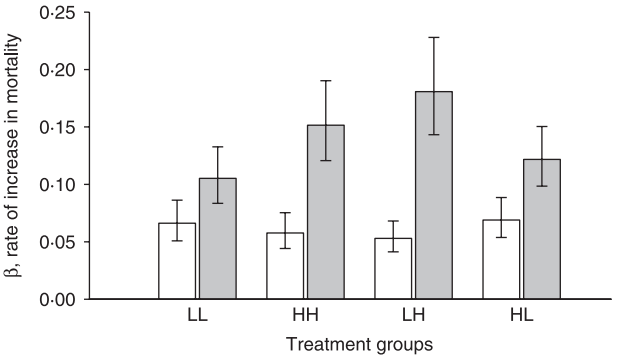

Fig. 3. Differences in the Gompertz age-dependent mortality, β, between males (white bars), and females (grey bars). Error bars show 95% confidence intervals of the estimated parameter β.

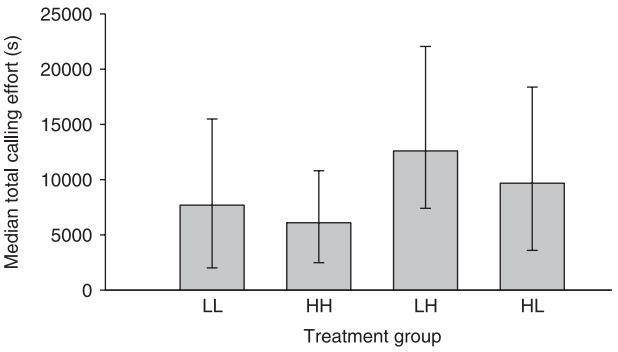

Fig. 4. Total male calling effort in each of the four diet treatments. Values presented as the median ± 95% bootstrapped confidence interval of the median.

(Table 3; Fig. 3), meaning that females aged much faster than males. The baseline mortality rate of males was higher in all four comparisons, but only significantly so in one comparison (HL diet).

Reproductive effort

In males there was a significant interaction between nymph diet and adult diet that affected total calling effort ($F_{1,239} = 4{\cdot}254$, $P = 0{\cdot}040$). To understand which treatment combinations were driving this interaction, we used *post hoc* tests, excluding the covariate life span from the model, to examine differences between the four treatment groups. Tukey's HSD revealed that the only significant pairwise differences between treatments were due to greater calling effort by LH than HH males ($P = 0{\cdot}025$) and HL than HH males ($P = 0{\cdot}039$). LH, HL and HH males did not differ from LL males ($P = 0{\cdot}734$, $P = 0{\cdot}774$, $P = 0{\cdot}311$ respectively; Fig. 4). Life span had a significant effect on total calling effort ($F_{1,239} = 166{\cdot}353$, $P < 0{\cdot}001$), but interactions between life span and diet were not significant. This means that males that lived longer called more during their lifetime, but the slope of the relationship between diet treatment and lifetime calling effort was independent of life span. The best-fit model of age-dependent calling effort included a positive linear (Wald $\chi^2 = 36{\cdot}502$, $P < 0{\cdot}001$) and a negative quadratic age term (Wald $\chi^2 = 20{\cdot}090$, $P < 0{\cdot}001$) (see Model 19 in Table S1 in Supporting Information). Calling effort increased with age and the rate of this increase did not differ between treatments. Fig. 5a shows that the negative quadratic coefficient does not result in a senescent decrease in calling effort, but that calling effort increases at a lower rate, compared to calling effort in early life, or plateaus at later ages.

In females, life span ($F_{1,240} = 278{\cdot}932$, $P < 0{\cdot}001$) and the interaction between nymph diet and life span ($F_{1,240} = 4{\cdot}507$, $P < 0{\cdot}035$) were significant predictors of total lifetime number of eggs laid. To examine this relationship further, we regressed lifetime egg number on life span, separately for each low- (pooled LL and LH) and high-nutrient nymph diet treatment (pooled HH and HL). We found a slightly steeper increase in

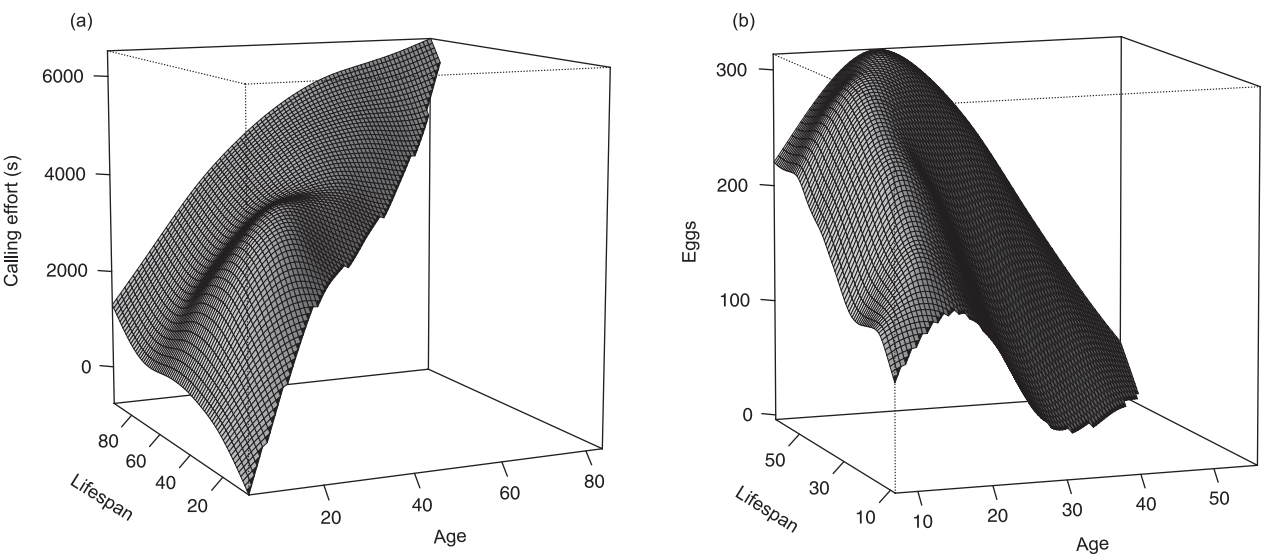

Fig. 5. The effect of life span and age on (a) male calling effort and (b) female fecundity. Data is pooled across all treatment groups.

lifetime egg number with increasing life span in females on high-nutrient nymph diet (standardized regression coefficient for low-nutrient nymph diet, $\beta_{low} = 0{\cdot}775$; high-nutrient nymph diet, $\beta_{high} = 0{\cdot}817$). The best-fit model of age-dependent reproductive effort in females included a positive linear (Wald $\chi^2 = 53{\cdot}699$, $P < 0{\cdot}001$) and a negative quadratic age term (Wald $\chi^2 = 62{\cdot}258$, $P < 0{\cdot}001$), and life span (Wald $\chi^2 = 53{\cdot}615$, $P < 0{\cdot}001$) (Model 19 in Table S2 in Supporting Information). Including treatment group in the models did not provide a better fit, suggesting that, as with males, diet had no substantial effect on the pattern of age-dependent fecundity (Table S2 in Supporting Information). In contrast to males, the visualization of age-dependent fecundity indicates that the negative quadratic coefficient of age stands for a decline in reproductive effort by old females (Fig. 5b). The effect of life span is likely to be caused by an increase in fecundity at early ages over a slightly longer period of time in females that lived longer (Fig. 5b).

Discussion

Consuming a low-nutrient diet as an adult extended longevity in female *T. commodus*. We found no evidence for such an effect in males. In contrast, nymph diet had no major effect on nymph or adult survival patterns or on reproductive ageing in males and females, but longer-lived females on a high-nutrient nymph diet laid more eggs during their lifetime than shorter-lived females. This suggests that animals on low-nutrient diet as nymphs exhibited no compensatory feeding or at least not enough to offset the cost of it in terms of long-term effects on reproduction. Females, but not males, showed a clear signature of reproductive senescence. Females also aged more rapidly than males in terms of increasing mortality rate with age, but both these effects on reproductive effort and mortality rate were independent of the diet that they were fed. Taken collectively, our results lead to three important conclusions. First, general patterns of ageing are likely to differ substantially between the sexes. Second, the effects of nutrient content of diet on ageing differ between the sexes. Third, these effects can be accounted for almost exclusively by adult diet, so that the effects of pre-adulthood diet can largely be ignored.

AGEING IS LIKELY TO BE SEX-DEPENDENT

In many animal species, females live longer than males (Promislow & Harvey 1990; Owens 2002; Fox *et al*. 2004), although in a few, males are known to outlive females (e.g. McCulloch & Gems 2003). Sexual differences in ageing rates are expected to be widespread, if not ubiquitous, because the costs of reproduction differ between the sexes (Chapman *et al*. 1995; Johnstone & Keller 2000; Partridge *et al.* 2005). Here we show that reproducing female crickets not only died sooner than males, but also show a more rapid increase in mortality with age, which is the signature of more rapid actuarial ageing (Charlesworth 1994). This sexual difference was consistent across all four experimental diet treatments.

The experimental conditions in our study may have imposed greater reproductive costs on females than males. Females were mated every 7 days by pairing them with a male cricket over night, leading to costs of being courted and of mating (Cordts & Partridge 1996; Yanagi & Miyatake 2003), of male harassment (Bussiere *et al*. 2006) and of egg production (Martin & Hosken 2004). In contrast, experimental males never encountered a female and therefore did not produce a courtship call or experience potentially costly matings. Long-range advertisement calls which we measured in this experiment are still more likely to be energetically more costly than much shorter and quieter courtship calls and spermatophore production. In addition, while females mated on average only four times in their lives, males are likely to have called every night during their lives. Interestingly, in a previous experiment (Zajitschek *et al*. 2007) female *T. commodus* that were mated using the same protocol but only twice in their life, lived on average 7 days shorter than virgin females (F. Zajitschek, unpublished data). This suggests that the reduced female life span that we report here may, in part, be due to a cost of frequent mating. The same pattern of a greater age-dependent increase in mortality rate for females was also found in a previous experiment comparing virgin females and males. In that study, females had a longer life span than males (i.e. due to a lower baseline mortality rate; Hunt *et al*. 2006), so the greater age-dependent increase in mortality in females seems to be independent of actual life span and might be a general difference between the sexes that persists across a range of different social and dietary environments.

Female crickets, unlike males, also showed signs of reproductive senescence. Both actuarial and reproductive ageing are likely to reflect a trade-off between investment into reproduction and somatic maintenance, but they probably act in different ways: through immediate costs of reproduction causing reproductive ageing, and through an accumulation of these physiological effects and of genetic damage over time resulting in actuarial ageing (Mair *et al*. 2003; Bonduriansky & Brassil 2005).

THE EFFECTS OF ADULT DIET ON AGEING ARE SEX-DEPENDENT

Adult females on a nutrient-rich diet lived approximately 14 days shorter and therefore laid (49%) fewer eggs over their lifetime than those fed the nutrient-poor diet. We expected the nutrient-rich diet that we fed crickets here to be of better quality than the nutrient-poor diet. In fact, when fed during adulthood, it actually had no direct effect on female lifetime fecundity, rather a negative indirect effect mediated through decreased longevity, with no evidence for a trade-off between reproduction and longevity. This might be due to the negative physiological effects of a high protein diet, as shown in cockroaches (Hamilton *et al.* 1990), or may suggest that, at the very least, females on nutrient-poor diet are able to compensate for the lower protein content in their diet by increasing their feeding rate (see Simpson *et al*. 2002), resulting in a protein intake that might still be lower than the one of

females on a high nutrient diet with higher protein content. Nevertheless, the greater longevity of females on a nutrient-poor diet is consistent with the common finding that dietary restriction (of calories or protein) can slow the ageing process (Doubal & Klemera 1999; Shanley & Kirkwood 2000; Mitteldorf 2001; Speakman *et al*. 2002; Kirkwood 2005). The non-existent direct effect of diet on fecundity in females indicates, however, that the effects of dietary restriction in *T. commodus* are not a consequence of suppressed reproduction, as has been postulated in *D. melanogaster* (Partridge & Andrews 1985; Rauser *et al.* 2004) and in Mediterranean fruit flies (*Ceratitis capitata*) (Carey *et al*. 1998; but see Mair *et al*. 2004; Kaeberlein *et al*. 2006).

In contrast to females, diet treatment did not influence male longevity, mortality rates or age-dependent calling effort. The effect of diet on ageing is sex-specific in *T. commodus*, as has been shown before for the effect of diet on longevity and reproduction (Maklakov *et al*. 2008). This finding is consistent with evidence from *D. melanogaster* that males and females have different optimal diets for longevity when maintained as adults on diets that differ in levels of food (yeast and sugar) restriction (Magwere *et al*. 2004).

In a previous experiment on *T. commodus* (Hunt *et al*. 2004b), males fed a high protein diet over their entire lifetime died sooner than those fed a medium or low protein diet, with high, medium and low protein levels in the respective diets being similar to our study. This dietary effect might be due to greater calling effort earlier in life, because this was negatively correlated with male longevity for all three diets (Hunt *et al*. 2004b). In the present study, total calling effort differed among the four diet treatments, but this pattern was difficult to interpret, as HH males called less than LH and HL males, but LL males were not different from the other three groups. We also did not find the same effects of diet on male longevity reported by Hunt *et al*. (2004b). The reasons for this difference in results are currently unclear. It is not due to switching diets at adult eclosion, as there was no difference for the pairwise comparison between the HH and LL diets which are identical to the High and Low protein diets used by Hunt *et al*. (2004b). We also used animals from the same wild population as Hunt *et al*.'s (2004b) (Smiths Lake, NSW, Australia).

The recent findings of Maklakov *et al*. (2008) are similar to the present results when we only take the known concentration of protein in our diets into account: comparing our low (*c.* 28% protein) and high (*c.* 45% protein) diets, the difference in life span in males that we would expect to find according to Maklakov *et al*.'s findings, is much smaller than the difference in females.

DIETARY EFFECTS ARE NOT COMPLETELY RESTRICTED TO ADULTHOOD

Evidence from studies on insects suggests that there is often an immediate change in mortality rates when adult diet is altered (Chippindale *et al*. 1993; Tatar & Carey 1995; Good & Tatar 2001; Mair *et al*. 2003). In contrast, accumulating evidence suggests that diet manipulation during the larval stage has little effect on senescence, instead it affects the acute risk of dying rather than the long-term accumulation of defects that lead to senescence (Tu & Tatar 2003; Partridge *et al.* 2005; but see Jacot *et al*. 2005). Our study is the first in *T. commodus* that manipulates nymph diet in addition to adult diet, using an experimental design that allows to separate the effects of these two treatments. The present results provide further evidence in support of this trend, with nymph diet having little influence on patterns of senescence.

Males reared on a high protein diet as nymphs were larger at eclosion, but this was the only carry over effect of nymph diet into adulthood in males. However, larger males did not show an increase in total calling effort or experience greater longevity. A qualitatively similar pattern was found in the field cricket, *Gryllus texensis*, with nymph diet affecting body weight at eclosion but having no effects on the structure of courtship calls (Gray & Eckhardt 2001). Likewise, *G. campestris* penultimate instar nymphs fed more food were both larger and heavier at eclosion compared to nymphs maintained on a restricted food intake but they did not call more or, with the exception of carrier frequency, produce structurally different calls (Scheuber *et al.* 2003b). In general, the importance of adult body size in determining fitness for male crickets is unresolved. It increases calling activity (Simmons 1988) and pairing success (Zuk 1988; Simmons 1995) in some species, but not others (Zuk 1988; Simmons & Zuk 1992). In contrast, adult diet appears to have a more uniform effect on fitness, with improved diet increasing calling effort (Holzer *et al*. 2003; Scheuber *et al.* 2003a; Hunt *et al*. 2004b; Hedrick 2005; Maklakov *et al*. 2008). Collectively, these studies suggest that adult diet has a greater impact on fitness than male body size across field cricket species.

Which diet female nymphs were fed had an interesting, even though not large effect on the correlation between life span and lifetime fecundity. The later females died, the more eggs they laid during their lifetime, regardless of diet treatment. However, females on high-nutrient diet as nymphs laid more eggs with increasing life span, compared to females on low-nutrient nymph diet. This suggests a more beneficial long-term effect of high-nutrient diet when taken up as a nymph, although maybe not in the form of nutrient reserves in the body, as there was no weight difference at eclosion between diet treatments (Table 1). Alternatively, it could indicate a long-term negative effect of compensatory feeding and 'catch up' growth before eclosion into adulthood (Metcalfe & Monaghan 2003).

Conclusions

In conclusion, we demonstrate that males and females of the field cricket *T. commodus* differ in their patterns of actuarial and reproductive senescence under the experimental conditions imposed on them. Most interestingly, there is no evidence that older males show a decrease in calling effort, whereas females show reproductive senescence and lay fewer eggs in all diet treatments as they age. This suggests that

senescent effects are more important to female than male life-history in this species. Nymph diet did not contribute to the observed patterns of ageing or age-dependent reproductive effort, so future research on *T. commodus* should focus more heavily on the effects of adult diet in this species.

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Supporting information

Additional Supporting Information may be found in the online version of this article:

Table S1. Model selection in males.

Table S2. Model selection in females.

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