Evol Biol (2011) 38:182–196
DOI 10.1007/s11692-011-9116-9

RESEARCH ARTICLE

Rapid Evolution of Lifespan in a Novel Environment: Sex-Specific Responses and Underlying Genetic Architecture

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Received: 13 January 2011 / Accepted: 7 March 2011 / Published online: 23 March 2011

Abstract Animal lifespans can vary substantially among closely related species and even among conspecific populations, but it is often difficult to identify environmental and genetic factors producing such variation. We used experimental evolution to examine how transfer to a novel environment affects adult lifespan and rates of senescence in a seed-feeding beetle. Three replicate lines of *Callosobruchus maculatus* (F.) were switched to a new host plant (cowpea), and each evolved shorter adult lifespans compared to a line maintained on the ancestral host (mung bean). However, the evolution of lifespan differed between the sexes; female lifespan was reduced by ~11% in all cowpea replicates, whereas male lifespan decreased by an average of only 5.6% and the magnitude of the reduction varied among replicates. Reduced lifespan in lines switched to cowpea mirrored the shorter lifespan observed in a separate population chronically associated with cowpea. We then performed crosses between the mung bean and cowpea lines to estimate the genetic architecture underlying the rapid evolution of a shorter lifespan on cowpea. Dominance (overdominance) contributed substantially to the difference between the cowpea and mung bean lines for female lifespan but not for male lifespan. However, details of the genetic architecture varied among the three replicate crosses, so that the convergent evolution of shorter female lifespan in the different cowpea lines did not arise from identical allelic substitutions. Our study demonstrates that insect lifespan can be predictably modified by a switch to a novel host plant, that both the magnitude of this response and its underlying genetic architecture can be sex-specific, and that convergent evolution of a complex trait such as lifespan can arise from different genetic mechanisms.

Keywords Aging · Experimental evolution · Longevity · Seed beetle · Selection experiment · Senescence · Life span

Electronic supplementary material The online version of this article (doi:10.1007/s11692-011-9116-9) contains supplementary material, which is available to authorized users.

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Introduction

Convergent evolution occurs commonly in nature, often in response to similar ecological conditions (Wood et al. 2005; Christin et al. 2010). Though cases of convergent evolution may be easy to recognize, one cannot usually isolate the particular selective pressures that account for the convergence. Moreover, even when phenotypic evolution appears to be predictable, it is difficult to determine whether parallel phenotypic changes reflect parallel genetic mechanisms (Christin et al. 2010; Nadeau and Jiggins 2010). Recent studies have shown that, for traits affected by one or a few genes, homoplasy can arise from similar or even identical sequence changes (e.g., Li et al. 2010; Yokoyama and Radlwimmer 2001), probably because there are strong biases in the likelihood of different amino acid replacements suitable for the emergence of the convergent phenotype (Christin et al. 2010). However, for more

complex traits, such as those affected by many genes and genotype-by-environment interactions, there may be multiple genetic pathways by which the same phenotypes can evolve (Bult and Lynch 1996; Wood et al. 2005; Arendt and Reznick 2008; Stern and Orgogozo 2008). Experimental evolution studies, in which replicate populations are allowed to adapt to controlled environmental conditions, provide a means by which we can test whether common genetic pathways underlie repeatable phenotypic evolution of complex traits (Chippindale 2006). In this study, we use natural selection experiments to examine genetic and environmental factors underlying the evolution of adult lifespan.

Lifespan and patterns of senescence can vary substantially within and among species (Wilson et al. 2008). Research in the last decade has produced many insights into the genetics and physiology underlying this variation (Finch and Tanzi 1997; Tatar 2001; Ricklefs 2008), but we still have a poor understanding of the specific sources of selection producing it (Monaghan et al. 2008). In natural populations, lifespan can evolve as a target of selection or as a correlated response to selection on other fitness components. Numerous interspecific comparisons have identified suites of life history and physiological traits that are likely to coevolve with lifespan (e.g., Austad 1997; Møller 2007, 2008; Blumstein and Møller 2008; Jones et al. 2008; McCoy and Gillooly 2008; see review in Ricklefs 2008). However, few studies have been able to attribute variation in lifespan to a specific ecological context (e.g., Reznick et al. 2004).

Selection experiments allow us to directly assess how selection might influence the evolution of senescence. Artificial selection can simulate forms of selection believed to occur in nature (e.g., selection for delayed reproduction), and has revealed a wide diversity of direct and correlated responses to manipulation of lifespan (e.g., Tucić et al. 1997, 1998; Chippindale 2006; Ackermann et al. 2007). By manipulating ecological conditions rather than specific characters (Fry 2003; Chippindale 2006), experimental evolution studies can be used to estimate both the repeatability of evolutionary responses and the effects of specific environmental variables on those evolutionary responses. For example, quasi-natural selection experiments have demonstrated that lifespan and rates of senescence (the rate of increase of mortality with age) can evolve in response to such factors as mating rate (Maklakov et al. 2007).

Selection experiments that manipulate key environmental factors can also be used to test whether lifespan is likely to evolve in a predictable and convergent manner, i.e., if multiple populations shifted to a novel environment converge toward patterns of aging observed in populations already adapted to this environment. Crosses between different lines can then establish the degree to which such parallel phenotypic evolution reflects parallel genetic evolution. Lifespan appears to be influenced by a rather complex genetic architecture that involves many loci (Wilson et al. 2006; Curran and Ruvkun 2007) with both additive and non-additive effects (Mackay et al. 2005; Seslija and Tucic 2008). Consequently, there may be a diversity of potential genetic and developmental pathways by which populations can converge toward similar mean lifespans and patterns of senescence in similar environments.

We examined the evolution of adult lifespan and patterns of adult mortality (shapes of mortality curves) in replicate lines of a seed-feeding beetle that had been shifted to a novel host plant. *Callosobruchus maculatus* (F.) has become a model organism for the study of senescence because of the ease with which it can be reared in the laboratory and because laboratory conditions approximate its "natural" environment. Beetles develop inside dry legume seeds and have likely been associated with human seed stores for thousands of generations (Tuda et al. 2006). Recent studies revealed substantial variation in adult lifespan and rates of senescence among populations adapted to different host species (Fox et al. 2004a, b) or experiencing different levels of sexual conflict (Maklakov et al. 2007).

We investigated the genetic architecture underlying convergent evolution of lifespan and rates of senescence in Asian beetle lines that had been switched from mung bean [*V. radiata* (L.) Wilczek] to cowpea [*Vigna unguiculata* (L.) Walp.] (Messina 2004a, b). By crossing each cowpea-adapted line with the mung bean-adapted line from which it had been derived, we could examine the degree to which the evolution of shorter lifespan on the new host was mediated by parallel genetic mechanisms in each replicate (Fox et al. 2009). We also crossed the Asian-origin mung bean line with an African line chronically associated with cowpea. We could thus compare the genetic architecture underlying a recent, experimentally induced divergence with the architecture underlying longstanding differences between populations associated with different hosts (Fox et al. 2004b).

Methods

Natural History and Study Populations

Callosobruchus maculatus (F.) is a cosmopolitan pest of grain legumes (Fabaceae), particularly beans of the genus *Vigna*. Females cement their eggs to the surface of host seeds and hatching larvae burrow into the seed directly under the egg. Larval development and pupation are completed entirely within a single seed. Female adults

mate and begin to lay eggs within hours of emerging from the seed. They are facultatively aphagous, and require neither food nor water in the adult stage. In storage and laboratory conditions, beetles are capital breeders; they primarily use larval resources for adult somatic maintenance and reproduction (Messina and Slade 1999).

In previous studies we examined the inheritance of differences in adult lifespan between populations that had been collected from and maintained on different legume hosts. The South India (SI) population was collected in 1979 from infested pods of mung bean and the closely related black gram [*Vigna mungo* (L.) Hepper] in Tirunelveli, India (Mitchell 1991). The Burkina Faso (BF) population was collected in 1989 from infested pods of cowpea in Ouagadougou, Burkina Faso (Messina 1993). These populations differ in body size, lifetime fecundity, egg dispersion, oviposition preference, and adult longevity. Despite long-term maintenance in the laboratory, each population maintains substantial genetic variation in adult lifespan (Fox et al. 2003a, 2004a).

Experimental Evolution Study

The natural selection experiment that provided the lines used in this study is described in detail in Messina (2004a, b) and Messina and Karren (2003). Prior to initiation of the experiment, the beetle populations had been maintained in laboratory growth chambers on cowpea (BF) or mung bean (SI) seeds at >1,000 adults per generation for >100 generations (BF) or >200 generations (SI), and thus were likely to have approached genetic equilibrium with respect to the standard rearing conditions (Harshman and Hoffmann 2000).

From the original SI population adapted to mung bean (henceforth = SI-mung), we established three independent lines (with >2,000 newly emerged adults per line) on cowpea. 'California blackeye' cowpeas are nearly three times heavier than mung beans (mean seed mass, cowpea = 189 mg, mung = 66 mg; $N = 100$ seeds each). Coefficients of variation for seed mass were similar between hosts: 15.7% for cowpeas, 14.8% for mung beans. Replicate SI lines maintained on cowpea will henceforth be referred to as SI-cowpea replicates A, B and C. Subsequent generations of the SI-cowpea lines were maintained in the same way as the SI-mung and BF populations, i.e., by adding 1,500–2,500 adults to ~750 g of seeds in each generation. Adults were collected in the middle of their emergence period (28–32 days after the start of the previous generation) to minimize inadvertent selection on development time or other traits (such as body size) that are genetically correlated with development time (Møller et al. 1990; Šešlija and Tucić 2003).

Line cross Experimental Design

Our crosses were among three sources of beetles: the SI population maintained on mung bean (SI-mung), the three replicate SI lines shifted to cowpea (SI-cowpea), and the BF population that had been chronically associated with cowpea. When the SI-cowpea lines had spent 117 generations on cowpea, we performed four types of crosses. One set of crosses was between the SI-mung and BF populations. These crosses, which were replicated three times, allowed us to quantify the genetic architecture underlying differences between the two populations that were adapted to different hosts and known to exhibit different mean lifespans. The second set of crosses was between each of the three SI-cowpea lines and the SI-mung line. These three crosses used the same design as the SI-mung × BF cross, and were performed simultaneously in the same growth chamber. Because the three SI-cowpea lines were independently evolving populations, the three SI-cowpea × SI-mung crosses represent true replicates of potential differentiation from the ancestral population. However, the three replicates of the SI-mung × BF cross were arbitrarily defined when we initiated the line crosses because we needed a different set of SI-mung beetles to be crossed to each of the three SI-cowpea lines (i.e., these are three replicate crosses between a single pair of lines, SI-mung × BF, and are not crosses between three distinct pairs of lines). We treated these arbitrary replicate SI-mung and BF lines as statistical replicates ($N = 3$ for each) in the ANOVA for mean lifespan because the three SI-cowpea lines represented true replicates and could not be pooled. For the demographic and genetic analyses, however, we used a single, pooled data set to represent the SI-mung and BF populations (see below).

We describe the crossing design between two generalized populations, which we will call P_1 and P_2 (the two parental lines). These represent both the crosses between SI-mung and BF and the crosses between SI-mung and the three replicate SI-cowpea (replicates A, B, or C). P_1 and P_2 beetles of generation 0 were mated to produce F_1 progeny in generation 1. Thus, in generation 1 we have four crosses, the two parentals (P_1 and P_2) and reciprocal hybrids (F_1 and F_1R) between these parental lines (Fig. 1). These generation 1 beetles were then crossed to produce F_2, backcross (B_1 and B_2) and new F_1 progeny in generation 2. By this final generation, we produced 14 distinct crosses (Fig. 1): two purebreds (P_1 and P_2), two reciprocal F_1 crosses (F_1 and F_{1R}), two reciprocal F_2 crosses (F_1♀ × F_1 ♂, F_{1R} ♀ × F_{1R} ♂), four backcrosses to P_1 (P_1 ♀ × F_1 ♂, P_1 ♀ × F_{1R} ♂, F_1 ♀ × P_1 ♂, F_{1R} ♀ × P_1 ♂) and four backcrosses to P_2 (P_2 ♀ × F_1 ♂, P_2 ♀ × F_{1R} ♂, F_1 ♀ × P_2 ♂, F_{1R} ♀ × P_2 ♂)(see also Table S1 in the supplemental material). Beetles from all crosses were scored simultaneously in this final

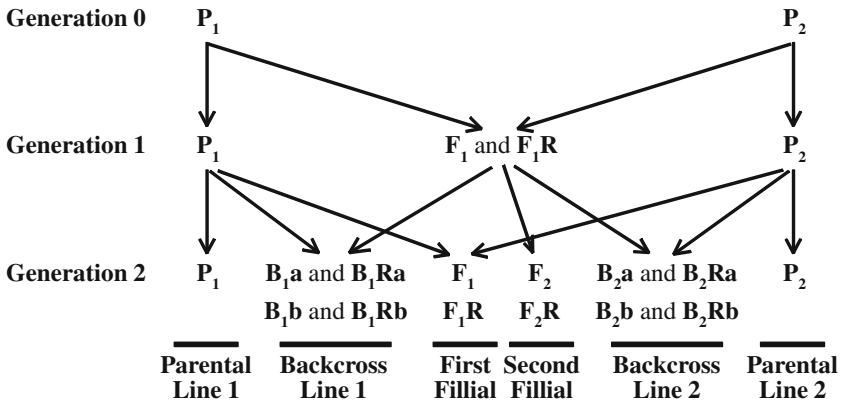

Fig. 1 The crossing design created four types of crosses in generation two and 14 crosses in generation three. *R* refers to the reciprocal cross (e.g., $F_1 = P_1$ ♀ × P_2 ♂ and $F_1R = P_2$ ♀ × P_1 ♂). The *a* and *b* in the *backcrosses* indicate whether the cross was between a *parental line* and the *F_1 line* (*a*) or a *parental line* and the *F_1R line* (*b*). See table S1 in the supplemental material for details of each cross

generation (see Bieri and Kawecki 2003; Fox et al. 2004b, c). Reciprocal crosses allowed us to test for maternal-genetic, Y-chromosome, and cytoplasmic effects on line-cross means. To remove any host-associated non-genetic effects, all three generations were reared on mung bean. Mung bean is generally a very good host for beetles of both the BF and SI populations (Stillwell et al. 2007; Fox and Stillwell 2009), and there is no evidence that the SI-cowpea lines are poorly adapted to mung relative to the SI-mung lines from which they were derived (Messina 2004a). Previous studies demonstrated that rearing host did not affect patterns of genetic variation in lifespan within these same parental populations (Fox et al. 2004a) or the composite genetic effects underlying the difference in lifespan between the SI-mung and BF populations (Fox et al. 2004).

We established a minimum of 10 mating pairs for each of the 14 crosses within each of the four cross types. Because cytoplasmic and Y-chromosome effects were not significant in any type of cross, we pooled reciprocal crosses that differed only in these effects. This reduced the number of crosses from 14 to 9, and yielded a minimum of 25 pairs per cross. Matings were performed in 35-mm Petri dishes containing ~35 mung bean seeds, with one mated pair per dish. Females were allowed to mate and oviposit for 48 h, after which the parents were discarded. Seeds bearing a single egg were isolated in 35 mm Petri dishes (one egg per dish) and reared to adult emergence. Thus, all offspring larvae developed without competition, and emerging adults were unmated and of known parentage. Our study thus focuses on the lifespan of virgin beetles, and we have no data on reproduction or reproductive lifespan in these populations. Mating and reproduction affect mean lifespan and rates of senescence of both males and females (Messina and Fry 2003), and may affect the genetics underlying variation in adult lifespan, but including effects of reproduction is beyond the scope of the current study.

All offspring developed at 25°C and 15:9 light:dark in a single growth chamber. The positions of dishes in the chamber were rotated daily. Lifespans of emerged beetles were measured simultaneously on all cross-types in the final generation of the experiment. Dishes were checked for emerging beetles twice per day. Each emerged adult was isolated in an empty 35-mm Petri dish. Lifespan was determined by checking each dish twice per day until a beetle died. Because all test beetles descended from three previous generations on a common host (mung bean), we removed any host-associated, non-genetic maternal effects on adult lifespan (Fox et al. 2004a). In the final (test) generation, we created an average of 124 families per cross type for the SI-mung × BF crosses, and scored adult lifespan for a total of 1,501 female and 1,488 male offspring. For the SI-mung × SI-cowpea crosses we created an average of 27 families per cross type per replicate and scored adult lifespan for a total of 1,250 female and 1,364 male offspring.

Mortality Rates

Age-specific mortality rate was estimated as $u(t) = -\ln[P(t)]$, where $P(t)$ is probability of surviving from the beginning of age t to the beginning of age $t + 1$. As in previous studies (Fox and Moya-Laraño 2003; Fox et al. 2003a, b, 2004a) mortality rates of *C. maculatus* populations were best described by a logistic model of the form:

$$u(t) = \frac{ae^{bt}}{\left[1 + \left(\frac{as}{b}\right)(e^{bt} - 1)\right]}$$

where $u(t)$ is the estimated age-specific hazard rate at age t, a is the intercept of the relationship $u(t) = ae^{bt}$ (often referred to as the initial or "extrinsic" mortality rate), b is the rate of exponential increase in mortality at young ages, and s describes the deceleration in mortality with increasing age (Pletcher 1999; Vaupel 1990). This model is similar to a Gompertz mortality model except that it includes s to account for the slowing of the increase in mortality rate with age (Pletcher and Curtsinger 1998). When $s = 0$, the logistic model reduces to the Gompertz model.

Parameters of the logistic mortality model (a, b, and s) were estimated using the maximum likelihood estimation procedure of WinModest (Pletcher 1999). We used the log-likelihood-ratio test of WinModest to determine whether individual parameter estimates differed significantly between lines. This test compares the log-likelihood [$L(v)$] of models for which parameter estimates are constrained to be equal in the two models with the log-likelihood of the models in which parameter estimates are unconstrained. Twice the difference between the maximum log-likelihood estimates of the constrained and unconstrained models is distributed as a χ^2 random variable with one degree of freedom. Statistical comparisons of mortality rates require large sample sizes. Because we found no differences in mean lifespan among the three SI-cowpea replicates, we pooled these life tables for these analyses.

Genetic Analysis

Composite genetic effects on line means were estimated using the genetic model of Kearsey and Pooni (1996), which has the parameterization described in the Supplemental Material and uses the expected mean of F_∞ offspring as a point of reference. We tested goodness of fit to genetic models using the weighted residual sums of squares (Bradshaw and Holzapfel 2000), as described in the Supplemental Material. A significant χ^2 value indicates that the fitted model was inadequate to explain the observed line cross means.

Details of our model selection procedure are described in the Supplemental Material and in Fox et al. (2004a, b, 2009). We used Akaike's Information Criterion to find the most parsimonious model (following Bieri and Kawecki 2003; see Burnham and Anderson 1998, 2004). This technique chooses a model that is the best compromise between the amount of variance explained and the number of parameters in the model. The model with the lowest AIC is most parsimonious, where $AIC = -2\ \ln(L) + 2\ K$, L is the log-likelihood of the model given the data, and K is the number of fitted parameters. Bieri and Kawecki (2003) showed that $AIC = RSS_w + 2\ K +$ constant. The constant is the same for all models and therefore need not be calculated to compare models.

It is possible that the most parsimonious model includes parameters that contribute little, such that removing the parameter would not significantly decrease the fit of the model. We used a likelihood-ratio test to determine whether the removal of individual terms significantly reduced the fit of the model (Lynch and Walsh 1998). The degree of reduced fit of the model is estimated as $\Lambda = RSS_{w(\text{reduced model})} - RSS_{w(\text{full model})}$. The parameter Λ is χ^2-distributed at large sample sizes, with degrees of freedom equal to the difference in the number of parameters in the two models.

A primary goal of this study was to compare composite genetic effects among the different types of crosses and between the sexes. To do this, we first fit a common model to each type of cross, or between sexes within a cross, i.e., a model including all effects that were significant in either cross/sex. We then compared the estimated composite genetic effects using a Wald Chi-Square test (see Supplemental Material; Fox et al. 2004a, b, 2009). We corrected for differences in mean lifespan between lines by dividing the composite genetic effects by the difference between parental lines, so that composite effects were a proportion of the difference between parental lines (see the Supplemental Material for details).

Results

Evolution of Adult Lifespan During Adaptation to Cowpea

Overall, there was substantial variation in adult lifespan among the SI-mung, SI-cowpea and BF lines (Fig. 2; $F_{2,6} = 5.8$; $P = 0.04$ in an ANOVA with replicate nested

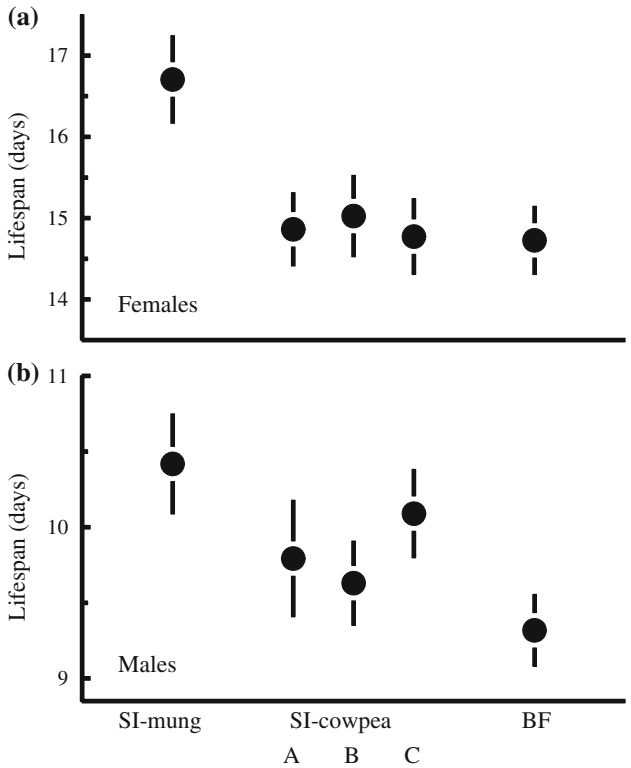

Fig. 2 Mean adult lifespan (±SEM) of *females* (**a**) and *males* (**b**) in the *SI-mung*, *SI-cowpea*, and *BF lines*. *SI-mung* represents *SI lines* maintained on the ancestral host (mung bean), whereas *SI-cowpea* represents lines switched to a new host (cowpea) 116 generations prior to this study. Cowpea is the ancestral host of the *BF lines*

within line). We therefore used linear contrasts (planned comparisons) to dissect the pattern of lifespan variation among lines.

Lifespan Differences Between Populations Chronically Associated with Mung Bean Versus Cowpea

SI-mung beetles lived longer as adults than the cowpea-adapted BF beetles, regardless of sex (linear contrasts; $F > 6.0$; $P < 0.02$ for both sexes). Although the magnitude of the absolute difference between SI-mung and BF differed between the sexes (a difference of 2.0 days for females vs. only 1.1 days for males), proportional differences between populations were similar for the two sexes. That is, BF lifespans were 11% shorter than those of SI beetles, regardless of sex.

Evolution of Lifespan in Response to the Host Shift

Adaptation to cowpea caused a decrease in adult lifespan; the mean lifespan of SI-cowpea beetles was significantly shorter than that of the SI-mung population from which they were derived (Fig. 2; $P < 0.02$ for each linear contrast between an SI-cowpea replicate and SI-mung). However, the magnitude of lifespan evolution in the three SI-cowpea replicates differed between males and females. We therefore present separate linear contrasts for each sex.

The adult lifespan of SI-cowpea females converged on that of the cowpea-adapted BF females; none of the SI-cowpea replicates was significantly different from the BF line (linear contrasts, $P > 0.60$ for each replicate), whereas females of all three replicates died earlier than the SI-mung females (replicates A, B, and C had 11, 10 and 11% shorter adult lifespans). None of the SI-cowpea replicates differed from each other with respect to female lifespan ($P > 0.23$ for each contrast). Thus, the adult lifespan of female SI beetles evolved to be shorter in response to the switch to cowpea and converged on the lifespan of beetles chronically associated with cowpea (rank order of female lifespan: SI-mung > SI-cowpea = BF for all replicates).

The evolution of male lifespan was more complex. Overall, the lifespan of SI-cowpea males was intermediate between the lifespans of the SI-mung and BF populations, but there was significant variation among replicates. SI-cowpea males lived 6, 8 and 3% shorter lives than SI-mung males (Replicates A, B, and C, respectively; Fig. 2). In linear contrasts, replicates A and B differed significantly from the SI-mung, but replicate C did not. The SI-cowpea replicates also lived 5, 3 and 8% longer than BF beetles; replicate C differed significantly from the BF line, but replicates A and B did not. Thus, the pattern of male lifespan evolution depended on replicate, with a rank order of SI-mung > SI-cowpea = BF for replicates A and B, and different rank order, SI-mung = SI-cowpea > BF, for replicate C.

Evolution of Mortality Curves

Mortality Curves of Populations Chronically Associated with Mung Bean Versus Cowpea

A comparison of the overall shape of the mortality curves [$u(t)$] between the SI-mung and BF populations was consistent with the ANOVA results for lifespan; mortality curves differed substantially between the two populations for both females ($\chi^2_1 = 19.8$; $P < 0.001$) and males ($\chi^2_1 = 28.4$; $P < 0.001$). Compared to BF females, SI-mung females had a slightly higher baseline mortality rate (a; $\chi^2_1 = 4.7$; $P = 0.03$) but a significantly lower rate of increase in the mortality rate (b; Figs. 3a, 4a; $\chi^2_1 = 5.6$; $P = 0.02$). Similarly, SI-mung males had a lower rate of increase in the mortality rate (b) than did BF males, but the difference was only marginally significant (Figs. 3b, 4b; $\chi^2_1 = 3.7$; $P = 0.05$). SI-mung and BF males did not differ in baseline mortality rate (a; $\chi^2_1 = 0.99$; $P = 0.32$).

Evolution of the Mortality Curve in Response to the Host Shift

Females—The difference in female lifespan between the SI-mung and SI-cowpea was also evident in the shape of their adult mortality curves ($\chi^2_1 = 17.2$; $P < 0.001$).

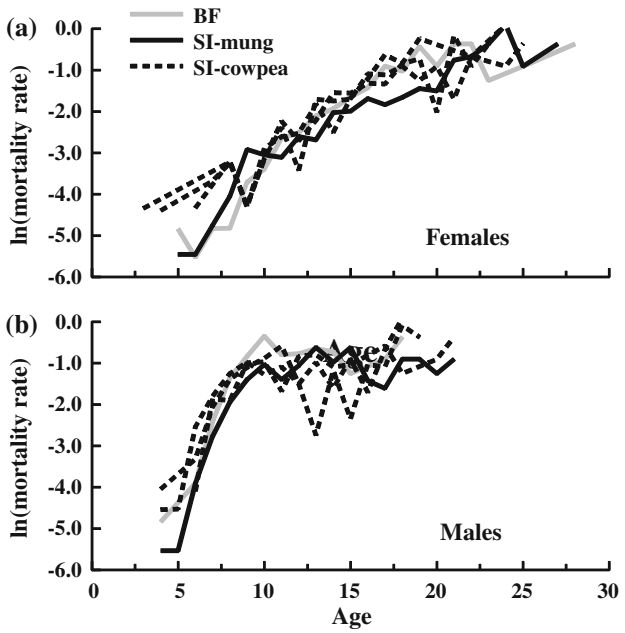

Fig. 3 Age-specific mortality, log[$u(t)$], plotted versus age for *females* (**a**) and *males* (**b**) in the *SI-mung*, *SI-cowpea*, and *BF lines*. See legend of Fig. 2 for details

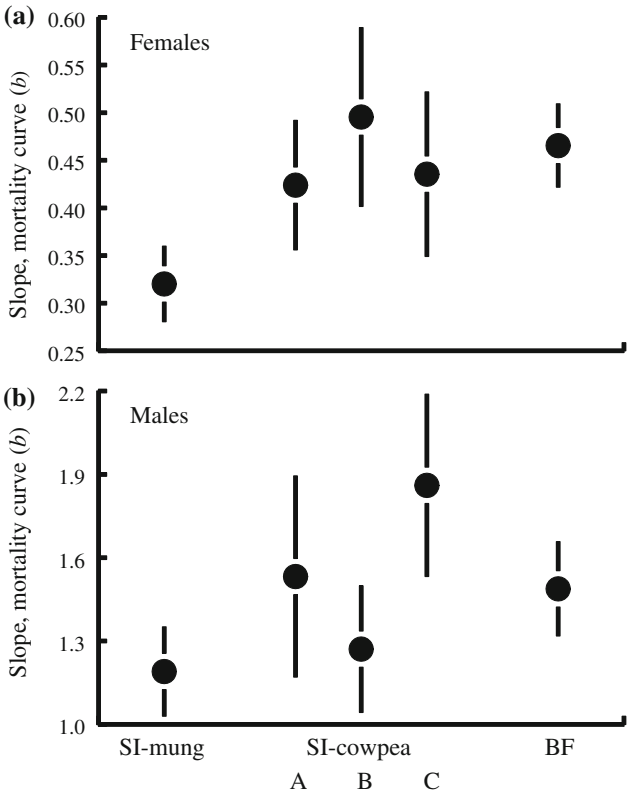

Fig. 4 The rate of change (b) of the age-specific mortality rate of *females* (**a**) and *males* (**b**) in the *SI-mung*, *SI-cowpea*, and *BF lines*. See legend of Fig. 2 for details

SI-cowpea lines evolved a significantly increased slope (b) relative to the ancestral population (Figs. 3a, 4a; $\chi^2_1 = 4.2$; $P = 0.04$). However, there was no detectable change in baseline mortality rate; estimates of a are all lower for SI-cowpea than for SI-mung, but the standard errors are large and none of the differences were significant (Table 1). In addition to diverging from SI-mung females, the SI-cowpea replicates also converged on similarly shaped mortality curves as the BF population; mortality curves of the aggregate SI-cowpea replicates and the BF population were not significantly different ($\chi^2_1 = 2.3$; $P = 0.13$), and none of the individual parameters of the mortality curves differed between the SI-cowpea and BF lines ($\chi^2_1 < 0.40$; $P > 0.52$).

Males—The divergence of male lifespan between the SI-mung and SI-cowpea lines was mirrored in the significant difference in the shapes of their mortality curves ($\chi^2_1 = 18.6$; $P < 0.001$ in a comparison of full mortality models, with all parameters constrained to be identical). The trend was toward a shorter lifespan of SI-cowpea males because of a higher slope (b) of their mortality curve (Figs. 3b, 4b), but neither a ($\chi^2_1 = 0.19$; $P = 0.66$) nor b ($\chi^2_1 = 1.2$; $P = 0.28$) differed significantly between the SI-cowpea and SI-mung lines. The mortality curves of SI-cowpea males also differed significantly from the BF mortality curve ($\chi^2_1 = 21.4$; $P < 0.001$ in a comparison of the full models). These results suggest that, contrary to the result for females, the male SI-cowpea mortality curves had not completely converged on that of the BF line. However, none of the individual model parameters differed significantly between the SI-cowpea and BF populations (Figs. 3b, 4b; $\chi^2_1 < 1.2$; $P > 0.28$ for both a and b).

Genetic Architecture Underlying Population Differences

Populations Chronically Associated with Mung Bean Versus Cowpea (SI-Mung × BF Cross)

The genetic architecture underlying the difference in lifespan between the SI and BF populations was significantly different for females than for males (Wald $\chi^2_3 = 253.4$;

Table 1 Parameter values for the logistic mortality model, $u(t) = ae^{bt}/[1 + (as/b)(e^{bt}-1)]$, for females of the SI-mung, SI-cowpea and BF populations of *Callosobruchus maculatus*

	Parameter estimates (95% confidence intervals)		
	a ($\times 10^{-3}$)[1]	b[2]	s[3]
SI-mung	1.46 (0.62–3.64)	0.320 (0.251–0.481)	0.589 (0.254–1.370)
SI-cowpea replicate A	0.71 (0.16–3.22)	0.424 (0.309–0.581)	0.855 (0.386–1.892)
SI-cowpea replicate B	0.32 (0.04–2.39)	0.495 (0.342–0.718)	1.192 (0.533–2.666)
SI-cowpea replicate C	0.59 (0.09–3.69)	0.435 (0.296–0.641)	0.859 (0.313–2.356)
BF	0.34 (0.12–0.94)	0.465 (0.386–0.561)	0.841 (0.513–1.381)
Likelihood ratio test	$SI_m > SI_c = BF$	$SI_m < SI_c = BF$	$SI_m = SI_c = BF$

Male parameter values for the SI-cowpea were generally intermediate between values for SI-mung and BF but none of the individual parameters (a, b, or s) differed significantly from either the SI-mung or BF

[1] a is the initial mortality rate

[2] b is the rate of exponential increase in mortality at young ages

[3] s describes the degree of deceleration in mortality with increasing age

Table 2 Most parsimonious models (chosen by AIC) and estimated composite genetic effects contributing to differences in adult lifespan in crosses between *Callosobruchus maculatus* populations

	SI-mung	SI-mung × SI-cowpea		
	× BF	Replicate A	Replicate B	Replicate C
Female lifespan				
μ_0	15.89 ± 0.32	15.75 ± 0.32	8.31 ± 2.38	16.10 ± 0.21
α	1.14 ± 0.31	0.75 ± 0.31	0.84 ± 0.33	1.19 ± 0.32
δ	1.54 ± 0.58	2.11 ± 0.56	22.78 ± 5.81	–
α^2	–	–	7.62 ± 2.35	–
$\alpha\delta$	–	–	–	–
δ^2	–	–	−12.20 ± 3.61	–
m_α	–	–	–	–
m_δ	–	–	–	–
χ^2	3.17 *ns*	1.81 *ns*	0.27 *ns*	3.58 *ns*
Male lifespan				
μ_0	10.06 ± 0.13	10.05 ± 0.24	10.27 ± 0.13	10.20 ± 0.15
α	–	0.41 ± 0.24	0.53 ± 0.19	–
δ	–	0.91 ± 0.49	–	–
α^2	–	–	–	–
$\alpha\delta$	–	–	–	–
δ^2	–	–	–	–
m_α	0.48 ± 0.15	–	–	–
m_δ	–	–	–	−0.61 ± 0.30[a]
χ^2	3.88 *ns*	4.67 *ns*	7.51 *ns*	2.62 *ns*

SI-mung refers to the South India population adapted to its ancestral host (mung bean). *SI-cowpea* is the South India population after it has adapted to cowpea seeds. *BF* is the Burkina Faso population that is adapted to cowpea seeds

χ^2, goodness of fit of the model to the real data (including only for those model terms shown)—a low χ^2 indicates a better fit; *ns* indicates that the model adequately explains the data. Model parameters: μ_0, mean; α, additive; δ, dominance; α^2, additive-additive epistasis; $\alpha\delta$, additive-dominance epistasis; δ^2, dominance-dominance epistasis

[a] A parameter in the most parsimonious model deletion of which did not significantly reduce the fit of the model to the observed line means (see methods)

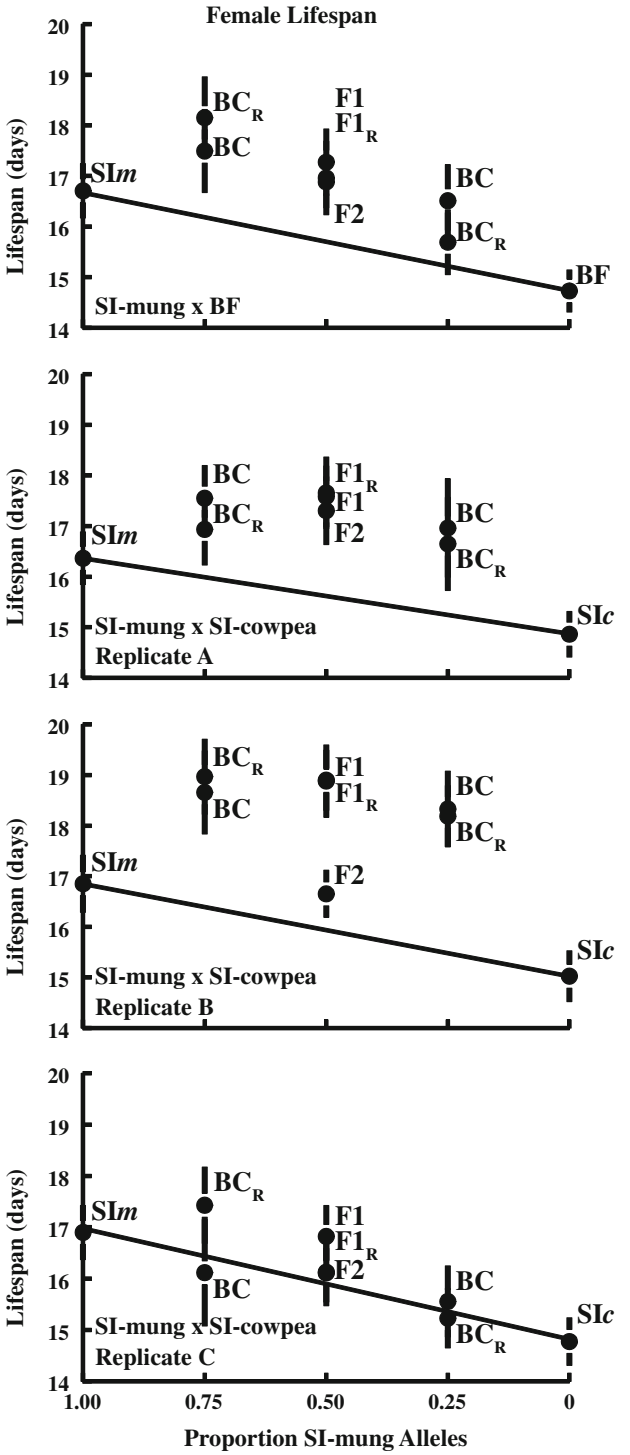

Fig. 5 Mean adult lifespan (±SE) of parental and hybrid females according to the proportion of genes from the *SI-mung line* (SIm). Separate crosses were performed between the *SI-mung line* and each of three replicate *SI-cowpea lines* (SIc)

$P < 0.001$). As we found in a previous study of only the SI-mung and BF populations (Fox et al. 2004), the AIC best-fit model for explaining the distribution of hybrid means for female lifespan included dominance (the $\mu_0 + \alpha + \delta$ model; Table 2; Fig. 5). This model was adequate to explain the distribution of hybrid female lifespan; the non-significant χ^2 indicates that observed line-cross means did not differ significantly from line-cross means predicted by the most parsimonious model. Dropping dominance from the model significantly reduced the fit to the data ($\chi^2_1 = 5.2$; $P = 0.02$).

In contrast, dominance was not present in the AIC most parsimonious model explaining the means for male lifespan. Consistent with our previous study (Fox et al. 2004), we detected a large additive-genetic maternal effect (m_α) on male lifespan (Fig. 6); the AIC best-fit model included only the overall mean plus additive-genetic maternal effect

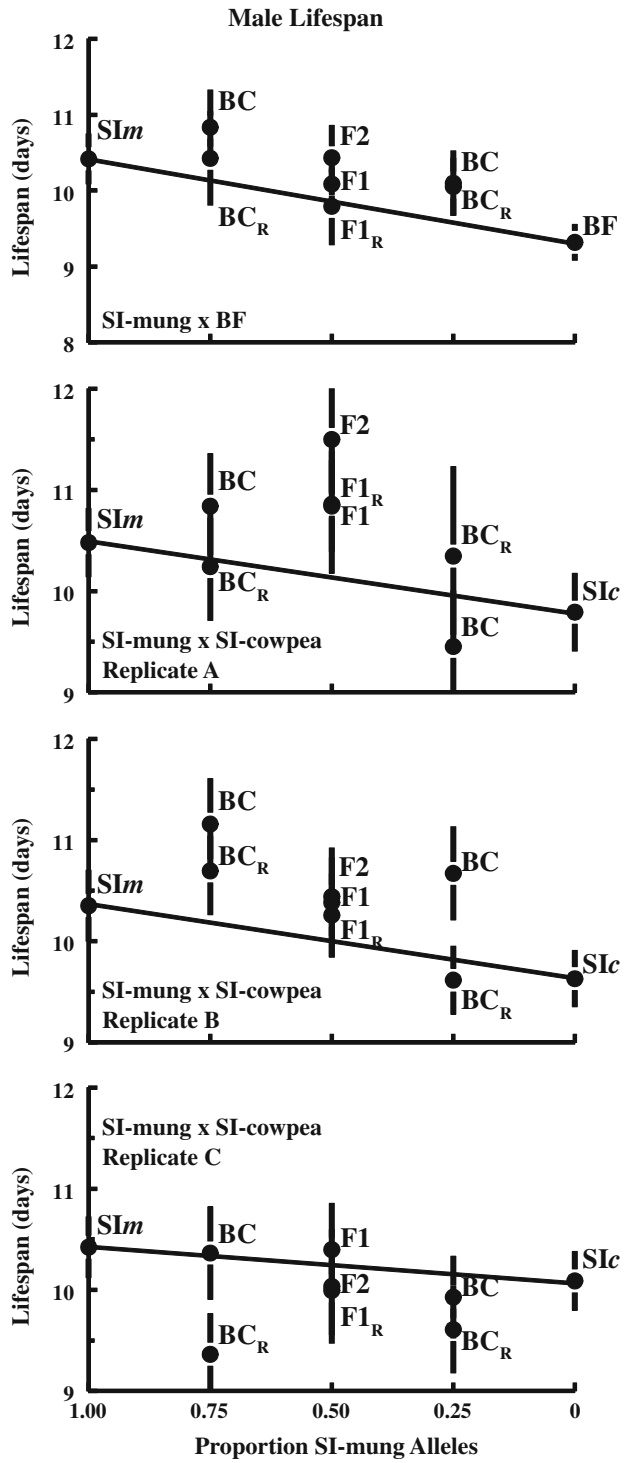

Fig. 6 Mean adult lifespan (±SE) of parental and hybrid males according to the proportion of genes from the *SI-mung line* (SIm). Separate crosses were performed between the *SI-mung line* and each of three replicate *SI-cowpea lines* (SIc)

($\mu_0 + m_\alpha$) (Table 2). Including additive composite genetic effects (α) in this model did not improve the fit to the data, and m_α was greater in magnitude than α in this expanded model. Thus, additive-genetic maternal effects most affected the distribution of hybrid means for male lifespan—females (mothers) possessing more SI-mung alleles produce male offspring that live longer whereas females possessing more BF alleles produce sons with shorter lifespans.

Lines Maintained on Mung Bean versus shifted to cowpea (SI-Mung × SI-Cowpea Crosses)

The genetic architecture underlying evolved differences in male lifespan between the SI-cowpea lines and the SI-mung lines differed statistically from that for female lifespan for two of the replicates (replicates B and C; Wald χ^2 pair-wise test between the sexes; $\chi^2_4 = 57.9$; $P < 0.001$ and $\chi^2_2 = 7.4$; $P = 0.02$ for replicates B and C, respectively; Table 2) but not for the third replicate (replicate A, $\chi^2_2 = 0.56$; $P = 0.76$; Table 2) (note that the number of degrees of freedom varies among crosses because the number of terms in the best-fit model varied; see the Methods).

The genetic architecture underlying evolved differences in female lifespan between the SI-cowpea lines and the SI-mung line varied significantly among the three replicates of the SI-cowpea lines (Wald χ^2 pairwise comparisons between replicates; $P < 0.02$ for each comparison, replicate A vs. B, B vs. C and A vs. C). Dominance contributed significantly to the genetic architecture underlying the difference in female lifespan between the SI-mung line and SI-cowpea replicates A and B, but not to the difference between the SI-mung line and SI-cowpea replicate C (Table 2; Fig. 5). If dominance was added to the model for replicate C, δ was non-significant. Epistasis (both α^2 and δ^2) contributed significantly to the lifespan difference between the SI-mung line and replicate B of the SI-cowpea lines (Table 2; Fig. 5), but there was no detectable epistasis for the lifespan differences between the SI-mung line and SI-cowpea replicates A or C.

The AIC best-fit model explaining hybrid means for male lifespan included different composite genetic effects for each cross between the SI-mung line and the three SI-cowpea replicates (Fig. 6; Table 2). Most interesting is that dominance was significant in only one of the crosses (that involving Replicate A) and a maternal effect (m_δ) was significant in only the cross involving Replicate C. Dropping δ from the best-fit model for the Replicate A cross significantly reduced the fit of the genetic model to the data, but dropping m_δ from the best-fit model for the Replicate C cross did not significantly reduce the fit of the data to the model (Table 2). Despite the differences in which terms were included in the AIC best-fit models for the three SI-cowpea replicates, an overall comparison of models indicated that the genetic architecture underlying the difference in male lifespan between the SI-cowpea lines

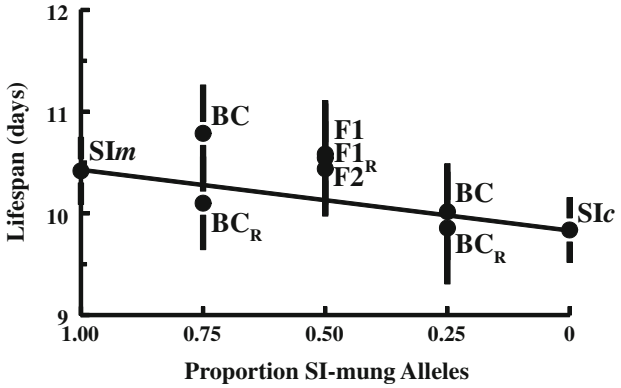

Fig. 7 Consensus genetic architecture for males of the *SI-mung* × *SI-cowpea cross*. Mean adult lifespan (±SE) of parental and hybrid males pooling replicates A, B and C

and the SI-mung population did not differ significantly among the three replicates (pairwise Wald χ^2 tests; $P > 0.20$). We thus pooled males from all three replicates to estimate a consensus genetic architecture (Fig. 7); the AIC best-fit model for the pooled replicates included only the overall mean plus the additive-genetic composite genetic effect; i.e., the consensus architecture for males was $\mu_0 + \alpha$.

Discussion

Geographic populations of *C. maculatus* are associated with several different legume hosts, and are known to vary in adult lifespan and patterns of senescence. Our results lead to three main conclusions. First, we demonstrate that mean lifespan and the rate of senescence (the rate of change in the mortality rate with age) evolved in response to an experimental host shift; the switch from mung bean to cowpea led to the repeatable and convergent evolution of a shorter adult lifespan and a higher slope of the adult mortality curve. The direction of evolution matched predictions based on comparisons between natural populations adapted to the different hosts (Fox et al. 2004a). Second, the magnitude of the evolutionary response depended on sex; female lifespan declined ~11% with adaptation to cowpea seeds, whereas male lifespan declined by only 3–8% (and varied among replicates). Finally, the genetic architecture underlying the evolved differences between the ancestral (mung bean-adapted) and derived (cowpea-shifted) populations varied among replicate experimental populations and differed between the sexes. For female lifespan, the effect of dominance was substantial in two crosses, but dominance was detectable (and small) in only one cross for male lifespan. Conversely, maternal effects were significant in at least one cross for male lifespan, but not in any cross for female lifespan. Thus, convergent phenotypic evolution was produced by different underlying genetic evolution among lines, though some features of the emergent genetic architecture could be predicted from comparisons between natural populations adapted to these hosts (Fox et al. 2004a). In particular, crosses between both natural populations and experimental lines indicated dominance affecting female lifespan, and maternal effects playing a role in male lifespan.

Convergent Evolution of Adult Lifespan and the Shape of the Mortality Curve

We observed genetically-based evolution of shorter adult lifespan in lines of *C. maculatus* switched from mung bean to cowpea. Previous studies have shown that the diet or rearing host experienced by larvae affects adult lifespan, rates of senescence, and even the genetic architecture underlying differences between long- and short-lived lines (Seslija and Tucic 2008). For example, *C. maculatus* reared on cowpea are known to have shorter adult lifespans than beetles reared on mung bean (Fox et al. 2004a, b), but these previously demonstrated effects are the result of phenotypic plasticity rather than adaptation to the rearing host. Our study demonstrates that shorter lifespan evolves in concert with adaptation to cowpea. Because all of our lines were reared on mung for three generations (one generation before the start of the experiment plus two generations of crosses) at a controlled density of one egg per seed, our results are not confounded by parental or rearing-host effects.

The experimental populations used in this study (SI and BF) have been shown to maintain heritable variation for lifespan despite their long-term persistence in the laboratory (Fox et al. 2003b, 2004a, 2006; Bilde et al. 2009). It is therefore not surprising that lifespan evolves following a switch to a new environment. But why might adaptation to cowpea favor a shorter lifespan? It is possible that the switch from mung bean to cowpea changes the female reproductive schedule, and that this change influences selection on lifespan. Cowpea seeds are larger than mung beans and support more larvae per seed, such that there is less selection for female avoidance of occupied seeds during oviposition on cowpea relative to mung bean (Tuda and Iwasa 1998; Messina and Karren 2003). If finding and inspecting hosts takes more time in populations adapted to small seeds, chronic association with small hosts may mimic selection for delayed reproduction, which in turn can lead to the evolution of increased lifespan (Rose et al. 2008). Decreased lifespan may evolve in our cowpea lines because shifting to this larger host selects for an accelerated reproductive schedule, which causally leads to a shorter adult life through antagonistic pleiotropy, or because a plastic change in the reproductive schedule on

larger seeds (more eggs laid early in life) relaxes selection against late-acting deleterious alleles that reduce lifespan. Although we have no data on reproductive schedule of females in the SI-cowpea lines, plastic or evolved, separate studies have shown that female reproductive schedules have not evolved to be particularly different between the SI-mung and BF populations when all females lay eggs on a common host (C. W. Fox, unpublished data). We therefore think it unlikely that reproductive schedule has evolved in SI-cowpea lines, but cannot test the hypothesis that relaxed selection associated with plastic changes in egg-laying schedule may have led to the accumulation of deleterious late-acting alleles in populations associated with the larger host.

Alternatively, reduced lifespan may simply be a correlated response to selection on other traits that were modified following the shift to cowpea. For example, body size also evolved to be substantially different between SI-cowpea and SI-mung populations (body size is smaller in the SI-cowpea lines; Messina 2004b). Body size was not measured in the current experiment (because of the large sample sizes needed in line crosses) but, in previous studies of these same populations, body size failed to explain much of the variation in lifespan within or between populations (Fox et al. 2004a, b). The host shift may also affect the optimal balance between life history traits that genetically covary with lifespan. For example, a shift to cowpea may favor a shift in the allocation of resources between egg size and number. The difference in larval competition strategy on large versus small hosts leads to relaxed selection on larval size (and thus egg size) on the larger host, and allows the evolution of higher fecundity (per unit body mass) on cowpea, which may lead to correlated evolution of lifespan because both egg size and number are genetically correlated with lifespan (Møller et al. 1989; Fox 1994).

Adult lifespan is commonly sexually dimorphic in animals, with females typically living longer than males (Fox et al. 2003b; Bonduriansky et al. 2008). These sex-differences in lifespan commonly vary among populations within a species (Gotthard et al. 2000; Promislow and Haselkorn 2002; Teriokhin et al. 2004). We found that adaptation to cowpea affected male and female adult lifespan differently; female lifespan evolved more than did male lifespan. Because we compared populations only after 117 generations following the shift to cowpea, we cannot determine whether this difference occurred because females evolved more quickly than males (e.g., males have not yet reached evolutionary equilibrium) or because the magnitude of change at selective equilibrium is truly greater for females. We suspect the latter explanation is correct because our populations maintain substantial genetic variation for lifespan (Fox et al. 2004a) and a variety of other traits measured in this experiment (body size, oviposition preference, and egg dispersion) converged toward the mean phenotype of the BF population, which has been chronically associated with cowpea (Messina and Karren 2003; Messina 2004a, b). We thus suspect that our experimental lines have reached evolutionary equilibrium for most behavioral and life history traits.

It remains unclear, however, why adaptation to cowpea should affect male lifespan less than female lifespan. One possibility is that the host shift changes patterns of sexual selection, which can have different effects on the evolution of male versus female lifespan (Promislow 2003; Hall et al. 2009). Sexual conflict has been shown to have large effects on female adult lifespan in *C. maculatus* (Crudgington and Siva-Jothy 2000; Edvardsson and Tregenza 2005). For example, manipulation of mating rate (polygamy vs. monogamy) resulted in substantial evolution of lifespan (of proportionally greater magnitude than observed here) in just 35 generations (Maklakov et al. 2007). However, subsequent experiments by Maklakov and colleagues that have attempted to tease apart the effects of natural versus sexual selection on the evolution of lifespan and the mortality rate have found that natural selection (e.g., selection favoring early versus late reproduction) affects the evolution of lifespan much more than does sexual selection (Maklakov and Fricke 2009; Maklakov et al. 2009).

The reduction of female lifespan following the shift to cowpea was due primarily to an increase in the slope of mortality curve, b, which converged on the slope observed in the cowpea-adapted BF population. This means that beetles shifted to cowpea evolved an increased rate of aging relative to beetles adapted to mung bean. The evolution of a higher b was also suggested for males in all three SI-cowpea replicates, but large standard errors on the parameter estimates prevented us from concluding that b evolved in males. Rapid evolution of the rate of aging in females is inconsistent with the general observation of Promislow and Tatar (1998) that mutations affecting adult survival act additively on the log of the instantaneous mortality rate [$\log(u(t)$] rather than on the age-specific survival rate, and that rapidly evolved changes in lifespan should consequently lead to proportional changes in the mortality rate (i.e., affect the intercept but not the slope mortality curve). Our results are also in contrast with a large study of *Drosophila* that found that variation in lifespan among populations within species was primarily due to variance in the baseline mortality rate, a, whereas variation among species was generally due to variance in both the slope and intercept (Promislow and Haselkorn 2002). However, other studies have shown that the rate of aging can vary significantly among conspecific populations of *Drosophila* (Sambucetti et al. 2005) and other insects (e.g., Gotthard et al. 2000; Fox et al. 2004a) and can evolve quickly in response to an environmental manipulation

(Linnen et al. 2001; Reznick et al. 2004; Maklakov et al. 2007). It therefore may be difficult to reach any general conclusions about which mortality-curve parameters are most likely to respond to changing ecological conditions.

Genetic Architecture Underlying Convergent Lifespan Evolution

The degree to which convergent phenotypic evolution of complex traits is due to parallel, and thus predictable, genetic mechanisms is generally unknown (Wood et al. 2005; Arendt and Reznick 2008). Our study shows that the precise genetic architecture underlying the convergent evolution of shorter female lifespan in response to a host shift differed among the three experimental evolution replicates. Alternative paths of evolutionary change should probably be expected for a trait such as lifespan, since it is affected by many genes and some genetic effects are non-additive. Lifespan is also strongly influenced by genotype-by-environment interactions (Seslija and Tucic 2008). For highly polygenic traits, convergent phenotypic evolution may be accomplished through different genetic mechanisms (Bieri and Kawecki 2003; Hoekstra and Nachman 2003; Stern and Orgogozo 2008), even in cases where experimental lines subjected to quasi-natural selection were all recently derived from the same ancestral stock (Zhang and Kumar 1997; Wood et al. 2005; Arendt and Reznick 2008). The parallel phenotypic responses of the three SI-cowpea replicates in this study may have been mediated by changes at a few loci or many loci (Shao et al. 2008), but clearly did not arise from the same allelic substitutions. In a companion study, we found that convergent changes in oviposition behavior in the three SI-cowpea lines were similarly produced by somewhat divergent genetic mechanisms (Fox et al. 2009).

Despite the difference in genetic architecture underlying the evolution of shorter female lifespan of the cowpea-shifted lines, significant dominance was detected in two of the three SI-cowpea × SI-mung crosses. Dominance was similarly detected for female lifespan in the cross between the two geographic populations, SI-mung × BF. The repeated detection of dominance suggests that long-life alleles are typically dominant over short-life alleles in female *C. maculatus*, even if parallel declines in female lifespan involved different numbers and types of loci in different replicates. It also suggests that non-additive genetic architectures can underlie recently evolved differences as populations adapt to different environments, with evidence of overdominance even in crosses between recently diverged populations. The influence of significant amounts of non-additivity on population divergence has generally been assumed to require much longer periods of differentiation (though this constraint may be more applicable to epistasis than to dominance *per se*). Non-additivity is also thought to play a bigger role during founder events and episodes of genetic drift (Bradshaw and Holzapfel 2000), with responses to selection largely due to additive-genetic effects (Lynch and Walsh 1998). However, recent studies suggest that non-additive effects may play a significant role in adaptive population differentiation (Carroll et al. 2001) and may contribute to non-adaptive differentiation much earlier than previously thought (Demuth and Wade 2007).

Although evidence of dominance for longer lifespans was obtained for females, no such effect was observed among males. Dominance (δ) was detected for male lifespan in only one cross, and the magnitude of δ in this cross was small relative to the estimates for females. This sex difference in the contribution of dominance in the SI-cowpea × SI-mung cross was also noted in the SI-mung × BF cross here and in a previous study (Fox et al. 2004). Within-population inbreeding studies similarly detect significant dominance affecting female, but not male, lifespan (Fox et al. 2006), though the magnitude of the sex-difference depends on the environment (e.g., temperature; Fox and Stillwell 2009). A few studies of *Drosophila melanogaster* lifespan have also detected QTLs exhibiting dominance, and some of these alleles appear to have sex-specific and environment-specific effects (Leips and Mackay 2000; Leips and Mackay 2002). Although the current study does not examine specific genes underlying differences in lifespan, our results extend these previous studies by demonstrating that the evolution of adult lifespan in a new environment can be sex-specific, that the genetic architecture underlying rapid evolutionary responses can be similarly sex-specific, and in particular that this sex-specific difference in the influence of dominance on differences in adult lifespan is a repeatable feature of the evolution of *C. maculatus* lifespan.

Because populations of *C. maculatus* consistently maintain heritable variation in a wide variety of fitness traits (Kawecki 1995), they are ideal subjects for examining the evolution of behavioral, physiological, and life-history traits following quasi-natural selection in the laboratory (Wasserman and Futuyma 1981; Tuda and Iwasa 1998; Messina and Karren 2003; Fricke and Arnqvist 2007; Maklakov et al. 2007). By including multiple lines that simultaneously adapt to the same novel challenges, these experiments can provide insights into whether the modification of complex, quantitative traits is likely to involve parallel or divergent genetic mechanisms (Matos et al. 2004; Fox et al. 2009). However, a full understanding of trait evolution in this model insect must await a detailed investigation of the *C. maculatus* genome, so that candidate genes influencing a polygenic trait such as lifespan can be identified and compared among populations or experimental lines (Geiger-Thornsberry and Mackay 2004; Baldal et al. 2006; Curran and Ruvkun 2007).

Acknowledgments We thank L. Cline, L. Hitchcock and W. Wallin for help with these experiments, D. Promislow for helpful discussion regarding the evolution of mortality rates, and T. Kawecki for comments on a previous version of this manuscript. This research was funded by grants from Kentucky EPSCoR (to CWF and JDW), the University of Kentucky Agricultural Experiment Station (to CWF), and the Utah Agricultural Experiment Station (to FJM, paper number 8089).

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