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1 Mating Frequency and Inclusive Fitness in *Drosophila melanogaster*

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20

21 LRH: PRIEST ET AL.

22 RRH: MATING FREQUENCY AND INCLUSIVE FITNESS

1 ABSTRACT

2

3 Increased mating frequency reduces maternal survival and reproduction. In order to  
4 understand the evolution of this trait, we need to determine whether frequent mating  
5 stimulates cross-generational fitness tradeoffs that reduce maternal fitness but enhance  
6 the fitness of offspring. We conducted an experiment in *Drosophila melanogaster* in  
7 which we manipulated the mating frequency of mothers and examined the survival and  
8 fecundity of the mothers and their daughters. We found that mothers with the highest  
9 mating frequency had accelerated mortality and more rapid reproductive senescence. On  
10 average, they had 50% shorter lives and 30% lower lifetime reproductive success (LRS)  
11 than mothers with the lowest mating frequency. However, mothers with the highest  
12 mating frequency produced daughters with 20% greater LRS. We evaluate these results  
13 using a demographic metric of inclusive fitness. We show that the costs and benefits of  
14 mating frequency depend on the growth rate of the population. When the multi-  
15 generational consequences of increased mating frequency were evaluated in an inclusive  
16 fitness context, we found no evidence that increased mating frequency results in fitness  
17 costs for mothers. These results indicate that cross-generational fitness tradeoffs have an  
18 important role in sexual selection and life history evolution.

19

20

21 KEYWORDS

22 Multiple mating; inclusive fitness; parental effects; fitness; sexual selection;  
23 sexual conflict; mutualism

1 INTRODUCTION

2 *In short, the counter-intuitive possibility exists that females with lower than*  
3 *average lifetime reproductive success may actually be fitter.*

4 – Hanna Kokko et al. (2003).

5

6 Sexual behavior is a great evolutionary paradox. In 1948, William Bateman  
7 showed that the marginal return of a mating event, measured by changes in lifetime  
8 reproductive success (LRS), is roughly constant for male fruit flies but decreases with  
9 every additional mating for females (Bateman 1948). This work suggested that the  
10 optimal mating rate is higher for males than for females. In the 1970s, work by Trivers  
11 (1972) and Parker (1979) indicated that these reproductive conflicts of interest between  
12 males and females might, paradoxically, drive the evolution of reduced female fitness.  
13 This hypothesis was supported by the finding that increased mating substantially reduces  
14 the LRS of mothers in at least 15 different species of insects (Arnqvist and Nilsson 2000).  
15 In addition, manipulative studies and selection experiments in *Drosophila melanogaster*  
16 show that mating and exposure to compounds in male seminal fluid can reduce the  
17 survival and fecundity of females (Cohet and David 1976; Fowler and Partridge 1989;  
18 Chapman et al. 1995; Rice 1996; Holland and Rice 1999; Brown et al. 2004).

19 There are two explanations for why sexual selection can reduce female LRS  
20 (Pizzari and Snook 2003). Sexual conflict theory predicts that sexual selection on traits in  
21 males might cause reduced LRS in females (Arnqvist and Rowe 2002; Chapman et al.  
22 2003). Alternatively, mutualistic theories such as sexy son, good genes, and genetic  
23 compatibility models, have primarily focused on the possibility that sexual selection

1 might allow for the evolution of reduced maternal LRS if it improves the genetic  
2 constitution of offspring (reviewed in Jennions and Petrie 2000; Zeh and Zeh 2003). Still,  
3 despite over two decades of empirical investigation that have established that multiple  
4 mating behaviors and costs of mating are widespread in the animal kingdom, there is no  
5 clear consensus about how mating behaviors that reduce maternal LRS can evolve  
6 (Arnqvist and Nilsson 2000; Zeh and Zeh 2003; Kokko et al. 2003).

7         It is possible that LRS may not be the most appropriate fitness measure for  
8 evaluating mating success (McGraw and Caswell 1996; Brommer 2000; Brommer *et al.*  
9 2002). Though the parental LRS approach combines survival and fecundity components  
10 of fitness into a single useful measure, it assumes that the population has a growth rate of  
11 zero. When the population growth rate (often referred to as  $m$ , the *Malthusian* parameter)  
12 is non-zero, the age-specific patterns of survival and fecundity can affect how many  
13 descendents a cohort will have in subsequent generations (Cole 1954; Mertz 1971;  
14 Charlesworth 1996; Caswell 2001). As such,  $m$  can be used as a metric of fitness under  
15 deterministic, density-independent conditions (Lotka 1939). The LRS and age-specific  $m$   
16 approaches often yield remarkably different estimates of the relative success of different  
17 cohorts or genotypes (Brommer *et al.* 2002). For example, in fruit flies increased male  
18 exposure decreased fitness by 20% when  $m$  was assumed to zero (the LRS assumption),  
19 but only by 4% when  $m$  was assumed to be large and positive (Tatar and Promislow  
20 1997).

21         However, using LRS and  $m$  as estimates of fitness can be misleading when there  
22 are parental effects, which occur when traits expressed in parents influence traits  
23 expressed in offspring, independent from offspring genetic effects (Mousseau and Fox

1 1998). If maternal LRS is correlated with offspring LRS then a cross-generational  
2 approach must be used to accurately estimate maternal fitness (Grafen 1988). Hamilton  
3 (1964) developed the theory of kin-selection to explain the evolution of behaviors that are  
4 detrimental to individual fitness. Hamilton's Rule states that an altruistic behavior can  
5 evolve when the fitness costs ( $c$ ) associated with the behavior are less than the fitness  
6 benefits ( $b$ ) to relatives times the degree of relatedness ( $r$ ) between the actor and the  
7 benefiting relatives, " $c < rb$ ". Hamilton's Rule could apply to the evolution of costly  
8 mating behaviors in fruit flies if maternal mating improves offspring fitness. Recent  
9 studies have found that maternal mating frequency influences the adult fitness traits of  
10 offspring (Konior et al. 2001; Fedorka and Mousseau 2004; Priest and Promislow, in  
11 press). Maternal mating frequency has fecundity benefits for daughters (Priest et al.  
12 submitted), though it does not appear to benefit sons (Orteiza et al. 2005; Priest et al.,  
13 submitted).

14       There are two questions that must be answered in order to evaluate whether cross-  
15 generational fitness tradeoffs influence the evolution of sexual behavior. First, how does  
16 maternal mating frequency affect the survival and fecundity of mothers and their  
17 daughters? And second, are the fitness costs of increased mating frequency for females  
18 balanced by fitness benefits to daughters in an inclusive fitness context? In this study we  
19 addressed these questions through manipulative experiments using *Drosophila*  
20 *melanogaster*. We manipulated maternal mating frequency and examined the effect of  
21 that manipulation on the age-specific survival and fecundity of mothers and their  
22 daughters. We then combined these demographic estimates using an inclusive fitness

1 approach that accounts for population growth rate to evaluate the cross-generational  
2 effect of mating on female fitness.

3

#### 4 MATERIALS AND METHODS

5 Maternal mating frequency was manipulated and the influence on the survival and  
6 fecundity of mothers and the survival and fecundity of their daughters was examined. We  
7 used the Dahomey strain of *Drosophila melanogaster*, which was caught in Dahomey,  
8 West Africa in 1970, and since then has been maintained as a large (> 1000 flies) outbred  
9 strain in population cages (Partridge and Andrews 1985). We cultivated flies at 50  
10 eggs/vial for three generations before start of the experiment to equalize differences in  
11 maternal environmental effects and to account for egg density effects on fitness traits  
12 (Clare and Luckinbill 1985).

13

14

##### *Maternal generation*

15 The experiment was conducted in three replicate trials that were initiated in  
16 consecutive two-week intervals. For each trial, we collected 204 virgin females (the  
17 mothers) from strain culture vials over a 5-hour collection interval (a total of 612 mothers  
18 across all trials). Each mother was placed in her own cage. The cages, which are roughly  
19 the size of a fly media vial, consisted of a small plastic tube with rubber gussets for  
20 aspirating flies and a sleeve for removing and adding fly media vials. When the mothers  
21 were 2 days-old, three 2-4 day-old virgin males (chosen from a large pool) were added to  
22 each cage. For each trial, cages were distributed in a randomized block design in 8 trays,  
23 which were treated as blocks in the statistical analyses. The 24 mothers that comprised a

1 block were randomly assigned to one of three mating treatments: High, Medium, or Low.  
2 The basic mating design was similar to previous studies (Fowler and Partridge 1989;  
3 Brown et al. 2004; Priest et al., submitted). The Low-mating mothers were exposed to  
4 three virgin males for one day every six days. The Medium-mating mothers were exposed  
5 to three virgin males for one day every three days. The High-mating mothers were  
6 exposed to three virgin males every day. For each 3-day interval, High-mating mothers  
7 received new males on days 1 and 2 and the day 2 males were kept in the vial with the  
8 female for day 3. The males were discarded after each mother exposure. Providing  
9 mothers with virgin males helps ensure uniformity among males (Torres-Vila and  
10 Jennions 2005), and providing mothers with multiple males allows females to choose  
11 mates.

12 The mothers were transferred to new vials every other day until death. The vials  
13 in which mothers laid eggs were retained for fecundity analyses. From the age at death  
14 values we calculated longevity and age-specific mortality rate ( $\mu$ ) (Elandt-Johnson and  
15 Johnson 1980). To estimate age-specific fecundity, we counted the number of hatched  
16 pupal cases from collection vials in alternate two-day intervals. The total number of  
17 offspring produced by each mother, maternal LRS, was estimated by multiplying the  
18 number of offspring counted, in alternate two-day intervals, by 2 and summing these  
19 estimates over the life of the mother. To determine whether the alternate-day sampling  
20 procedure for fecundity provided a good representation of the total fecundity, we counted  
21 all of the hatched pupal cases for all of the mothers in Trial 3. The correlation between  
22 the alternate-day and full sampling regimes was  $r = 0.80$  ( $n=165$ ). In order to estimate  
23 egg-to-adult viability, we counted the number of eggs that each mother laid every fourth



1 two-day interval (i.e. 1/4 of all collection vials) and compared that to the number of  
2 hatched offspring these vials produced. During the course of the experiment 3.8%, of all  
3 of the maternal flies (23/612) escaped or were lost due to handling errors. These flies  
4 were censored in the survival analyses and eliminated from the lifetime reproductive  
5 success analyses, but were included in the egg-viability and age-specific fecundity  
6 analyses for the intervals before they were lost.

7

### 8 *Daughter generation*

9 Mating and exposure to compounds in male seminal fluid causes a brief and  
10 temporary increase in egg laying (Kalb et al. 1993). We expected that effects of mating  
11 on offspring quality might also be brief and temporary; therefore, we collected a single  
12 vial with eggs from each mother on day 8 when mothers had been restricted from mating  
13 for either 5 days (Low mating), 2 days (Medium mating) or 0 days (High mating) to  
14 examine how a mother's mating affected daughter survival and fecundity. In a previous  
15 study we manipulated mating frequency in a similar way, but collected offspring at an  
16 interval after all of the mothers had had recent male exposure; we found similar patterns  
17 to those reported here (Priest et al., submitted). We cleared the vials on the morning of  
18 the third day of emergence and 5 hours later collected one newly emerged female from  
19 each vial (the daughters). Daughters from all of the maternal mating treatments were  
20 placed singly in vial-sized cages (as described before) with four virgin males chosen from  
21 a large pool of males. Daughters were held with males and were provided fresh virgin  
22 males every two weeks. The daughters were transferred to new vials with their males  
23 every other day until death. The three trials of daughters differed in the timing of initial

1 male exposure. For Trial 1 the daughters were held as virgins for seven days after  
2 eclosion before they were first mated; for Trial 2 the daughters were held as virgins for  
3 four days; and for Trial 3 the daughters were provided males the first day. The number of  
4 days until females first mated contributes to differences among the three offspring trials.

5 Daughter age-specific fecundity and LRS was determined by counting all of the  
6 hatched pupal cases for each of the three daughter trials. Egg-to-adult viability of  
7 daughters was measured by counting the number of eggs that each daughter laid every  
8 fourth two-day interval. We recorded the age at death for each daughter. During the  
9 course of the experiment, 4.3% of all daughter flies (23/539) escaped or were lost due to  
10 handling errors. As with mothers, these flies were censored in the survival analyses,  
11 eliminated from the lifetime reproductive success analyses, but were used in the egg-  
12 viability and age-specific fecundity analyses for the intervals before they were lost.

13

#### 14 *Calculation of Fitness and Inclusive Fitness*

15 Charlesworth (1970, 1996) showed that for a population with overlapping  
16 generations, the fitness ( $w$ ) of a particular cohort  $i$  is described by

$$17 \quad w_i = \sum_{x=0}^{\omega} e^{-mx} k_i(x), \quad (1)$$

18 where  $x$  is age of cohort  $i$  from adult emergence 0 through last reproduction  $\omega$ ,  $m$  is the  
19 population growth rate, and  $k_i(x)$  is the age-specific product of survival and fecundity. We  
20 used this equation to estimate the fitness of High-, Medium-, and Low-mating mothers  
21 and their daughters for Trial 3, the cohort which had the full age-specific fecundity  
22 trajectories for both mothers and daughters. We treated the 8 individuals of each mating  
23 treatment within each block as a cohort. Two values of  $m$  are most appropriate for

1 estimates of fitness: 0, for LRS, and  $\lambda$ , the value of  $m$  that can be found implicitly by  
2 setting  $w_i = 1$ , using the Euler-Lotka equation (Lotka 1939). However, to understand  
3 how population growth rate assumptions influence fitness estimates, we evaluated the  
4 fitness of each block, which were treated as separate cohorts, over the range of population  
5 growth rates commonly found in fruit fly population cage culture ( $m = -0.4$  to  $0.4$ ; Prout  
6 and McChesney 1985; see Tatar and Promislow 1997 for a similar approach).

7 LRS and  $\lambda$  are inadequate measures of parental fitness when the fecundity and  
8 survival of parents influence the survival and fecundity of offspring (Hamilton 1964;  
9 Grafen 1984, 1988). When this is true we need to measure inclusive fitness, the  
10 combination of an individual's total fitness with the fitness the individual accrues by  
11 helping relatives. However, how to measure inclusive fitness is not always clear. For  
12 example, Barash (1980, from Grafen 1982) defined inclusive fitness as,

13 the sum of individual fitness (reproductive success) and the reproductive success  
14 of an individual's relatives, with each relative devalued in proportion as it is more  
15 distantly related

16 This definition incorporates the salient aspects of Hamilton's concept, but it has  
17 difficulties for the estimation of inclusive fitness. First, typically only some, not all,  
18 relatives receive help from the donor. Second, inclusive fitness should only relate to the  
19 proportion of fitness of relatives that directly results from the donor individual's actions.  
20 Finally, it equates fitness with LRS (as others have done, e.g. Creel and Wasser 1994;  
21 Griffin and West 2002).

22 Charlesworth and Charnov (1981) and Oli (2003) developed methods of  
23 measuring inclusive fitness which account for many of these technical difficulties. Both

1 models focus on the relatives whose fitness changes because of the altruistic act.  
2 Specifically, the proportion of fitness of relatives that directly results from altruistic  
3 actions is determined by the fitness differences between recipients and non-recipients of  
4 the donor's aid, scaled by the degree of relatedness between the donor and the recipient.  
5 Thus, the non-recipients are treated as "controls" and the benefits of altruism are  
6 estimated from fitness differences between the recipients and the non-recipients of the  
7 altruistic act. In addition, both models use the  $\lambda$  of the donor generation and the  $\lambda$  of the  
8 recipient generation to estimate inclusive fitness. [Oli (2003) explicitly uses  $\lambda$  and  
9 Charlesworth and Charnov (1981) use a related concept, reproductive value].

10         However, the manner that age is structured remains an issue that may bias  
11 measures of inclusive fitness. The models estimate inclusive fitness by combining fitness  
12 costs with respect to the age of the recipient generation and fitness benefits with respect  
13 to the age of the donor generation (Charlesworth and Charnov 1981; Oli 2003). In an  
14 expanding population early-age fecundity has a disproportionate effect on the value of  $\lambda$ .  
15 Thus, the age at which the fitness costs and benefits of mating appear may have an  
16 impact on how altruism genes evolve. If the population is expanding and there is a time-  
17 lag between the expression of fitness costs by the donor generation and the expression of  
18 fitness benefit by recipient generation, then both models will underestimate the fitness  
19 costs and overestimate the fitness benefit of the altruistic act.

20         A different way of handling age structure across multiple generations is to  
21 estimate the fitness benefits to the recipient with respect to the age of the donor, not the  
22 age of the recipient. This approach places cross-generational fitness tradeoffs in an age-  
23 specific context that is appropriate when there is a time lag between the altruistic act and

1 benefits to recipients. The time-lag issue is relevant for this study because in fruit flies  
 2 parental effects can only occur before egg laying and enhanced offspring fecundity can  
 3 only be expressed at a later age (because daughters take several days to eclose).

4 Therefore, we define the inclusive fitness of a maternal cohort  $i$  by

$$5 \quad w_{inclusive} = \sum_{x=0}^{\omega_i} e^{-mx} k_i(x) + \sum_{x=0}^{\omega_j} r_{ij} (e^{-mx} k_j(x) - e^{-mx} k_{control}(x)), \quad (2)$$

6 which combines the fitness of mothers with the proportion of daughter fitness that results  
 7 from maternal mating (modified from Charlesworth and Charnov 1981; Oli 2003). For all  
 8 terms,  $x$  is age of the maternal cohort  $i$  from adult emergence 0 through age of last  
 9 reproduction of the mother  $\omega_i$  and daughter cohorts  $\omega_j$ .  $m$  is the population growth rate  
 10 of the maternal generation.  $k_i(x)$ ,  $k_j(x)$  and  $k_{control}(x)$  are the products of age-specific  
 11 survival and fecundity of the mother, daughter and control cohorts, respectively. Here,  
 12 the  $k_{control}(x)$  term is the product of survival and fecundity for daughters of Low-mating  
 13 mothers.  $r_{ij}$  is the degree of relatedness between mothers and their daughters,  $r_{ij} = 0.5$ .

14 This metric calculates maternal fitness and combines that quantity with the  
 15 amount of daughter fitness that results from maternal mating frequency. Clearly not all of  
 16 the fitness of daughters should be attributed to the mothers (Grafen 1984, 1988). This  
 17 metric estimates the proportion of daughter fitness that directly results from maternal  
 18 mating frequency by subtracting the fitness of daughters from High-mating mothers from  
 19 the fitness of daughters of Low-mating mothers (“control”). Similar calculations can be  
 20 made for the Medium-mating mothers by comparing fitness difference between daughters  
 21 of Medium- and Low-mating mothers. This quantity is then multiplied by 0.5 to account  
 22 for the fact that only half of a daughter’s genes come from her mother. The quantity is

1 then scaled by the population growth rate ( $m$ ) of the maternal generation to account for  
2 the effect of exponential growth.

3 This inclusive fitness model generates inclusive fitness estimates for High- and  
4 Medium-mating mothers. Because Low-mating mothers by definition do not contribute  
5 any benefits of increased mating frequency to their daughters, the inclusive fitness of  
6 Low-mating mothers is only the maternal fitness of Low-mating mothers. We calculated  
7 the inclusive fitness of High-, and Medium-mating mothers and the fitness of Low-  
8 mating mothers for each of the 8 blocks in Trial 3. To examine how population growth  
9 rate assumptions influence estimates of fitness, we evaluated inclusive fitness over a  
10 range of  $m$  that is common to normal fruit fly culture from ( $m = -0.4$  to  $0.4$ ).

11

### 12 *Statistical Analysis*

13 The survival analyses were conducted with Proc PHreg (SAS 9.1 2003), which  
14 uses a maximum likelihood approach. The mortality rate data fit a logistic curve,  $\mu_x =$   
15  $Ae^{Bx} / [1 + (AS/B)(e^{Bx}-1)]$ , where A, B, and S are terms which describe age-independent  
16 mortality (intercept or background mortality), age-specific mortality (slope or rate of  
17 aging), and leveling-off (the deceleration of mortality at late ages), respectively. We  
18 estimated the mortality intercept, rate and leveling-off for each mating treatment and  
19 tested for differences between pairs of mating treatments using WinModest (Pletcher  
20 1999), a maximum likelihood model that accounts for censored data.

21 We tested for the effects of the maternal mating treatment on LRS and age-  
22 specific fecundity using treatment means for each of the 24 blocks that comprise Trials 1,  
23 2 and 3. Block means were used in the analysis to reduce variation in the data. For the

1 LRS data of both the mothers and daughters we performed an analysis of variance with  
2 maternal mating treatment and block as fixed effects, and used the Tukey multiple  
3 comparison test to compare differences between the mating treatments. Egg density did  
4 not differ between the maternal mating treatments ( $F_{2,536} = 2.23$ ,  $P > 0.11$ ). Egg density  
5 was tested as a covariate and was dropped from all analyses because it was not a  
6 significant source of variation. To examine differences in age-specific fecundity for both  
7 the mothers and daughters, we performed repeated measures analysis of variance on age-  
8 specific fecundity, with maternal mating treatment and block as fixed effects. To  
9 statistically examine differences in mother, daughter, and inclusive fitness, we performed  
10 analysis of variance on the values of  $w_i$  and  $w_{inclusive}$  obtained from equations 1 and 2 at  
11 each value of population growth rate, with maternal mating treatment as a fixed effect.  
12 All analyses were done using PROC MIXED (SAS 9.1 2003) on untransformed data;  
13 both the data values and the residuals approximately fit model assumptions.

14

## 15 RESULTS

16 The maternal mating treatment influenced the survival of mothers, but not  
17 daughters. Mothers with the highest mating frequency had the lowest survival ( $\chi^2 =$   
18  $194.57$ , 2 d.f.,  $P < 0.0001$ ; Figure 1a). The mortality analysis showed that mating  
19 frequency significantly influenced background mortality (mortality intercept) and the rate  
20 of aging (mortality rate) of mothers (Table 1; Figure 2a). The mortality intercepts of each  
21 mating treatment were significantly different from one another, with High-mating  
22 mothers having the highest mortality intercept and Low-mating mothers having the  
23 lowest mortality intercept. The mortality rates of High- and Low-mating mothers were

1 significantly different; but there were no significant differences in the leveling-off term  
2 (Table 1). There were no effects of maternal mating on daughter survival ( $\chi^2 = 0.41$ , 2  
3 d.f.,  $P = 0.815$ ; Figure 1b), mortality intercept, mortality rate, or mortality leveling-off  
4 (Table 1; Figure 2b).

5         Maternal mating frequency influenced both maternal fecundity and daughter  
6 fecundity, though in opposite ways for each generation. Increased maternal mating  
7 frequency reduced maternal lifetime reproductive success (LRS), but increased daughter  
8 LRS (mothers,  $F_{2,67} = 57.12$ ,  $P < 0.0001$ ; daughters,  $F_{2,67} = 5.12$ ,  $P = 0.008$ ; Figure 3).  
9 High-mating females had the lowest LRS, but their daughters had the highest LRS;  
10 Medium-mating females had intermediate LRS and their daughters had intermediate  
11 LRS; and Low-mating females had the highest LRS, but their daughters had the lowest  
12 LRS (Figure 3). Similar statistically significant patterns were observed with respect to  
13 mother and daughter age-specific fecundity (Table 2, Appendix Figure 1). The effects of  
14 maternal mating frequency on age-specific fecundity were consistent across maternal and  
15 daughter ages (no mating  $\times$  age interactions). Maternal mating frequency did not  
16 influence daughter egg-to-adult viability (Table 2). However, age-specific fecundity and  
17 egg-to-adult viability decreased with female age (Table 2; Appendix Figure 1).

18         Both mother and daughter fitness estimates were strongly influenced by the value  
19 of population growth rate ( $m$ ) that was assigned in the calculation of fitness. For the  
20 maternal generation, at all values of  $m$  less than +0.10, increased maternal mating  
21 frequency reduced maternal fitness (Appendix Table 1; Figure 4a). For the daughter  
22 generation, at all values of  $m$  greater than -0.10, increased maternal mating frequency  
23 significantly improved daughter fitness (Appendix Table 1; Figure 4b). Note that at



1 values of  $m$  less than -0.10 increased maternal mating frequency substantially increased  
2 daughter fitness, but the effect was non-significant because of variation in late-age  
3 daughter fecundity, which in a declining population has a disproportionate contribution to  
4 fitness (Figure 4b). When the fitness cost of multiple mating to females and fitness  
5 benefits to daughters were placed in an inclusive fitness context, there was no significant  
6 effect of multiple mating on fitness at any value of  $m$  (Appendix Table 1; Figure 4c).

7

## 8 DISCUSSION

9         The results presented here reveal two patterns that are important to our  
10 understanding of how the mating frequency of females may evolve in the face of  
11 substantial costs. First, increased maternal mating frequency improved the LRS and  
12 fitness of daughters. Second, multiple mating may not be as costly as is typically  
13 presumed. When the effects of maternal mating on mother and daughter fitness were  
14 integrated in a cross-generational fitness approach, multiple mating was not costly to  
15 mothers, at any assigned population growth rate.

16         With respect to the maternal generation, we found that multiple mating reduces  
17 female survival and LRS, which is similar to studies with comparable designs (Fowler  
18 and Partridge 1989; Brown et al. 2004; Priest et al., submitted). This study also revealed  
19 novel findings in the maternal generation. Multiple mating increased the age-independent  
20 mortality (intercept or background mortality), the age-specific mortality (slope or rate of  
21 aging), and decreased the age-specific fecundity of females. These results suggest that  
22 multiple mating accelerates the aging process by increasing the risk of death with age and  
23 by stimulating more rapid reproductive senescence. Previous work found that increased

1 mating frequency decreases survival and longevity (Partridge and Andrews 1985; Fowler  
2 and Partridge 1989; Chapman et al. 1995; Brown et al. 2004), but this is the first study to  
3 confirm that mating frequency directly influences the ageing process. Also, the effect of  
4 males on the background risk of death (mortality intercept) and rate of aging (mortality  
5 slope) have been shown to be genetically correlated with male sperm competitive ability  
6 and male reproductive success (Civetta and Clark 2000; Sawby and Hughes 2001).  
7 Changes in patterns of mother mortality might, therefore, occur as a consequence of  
8 sperm competition. But, it is also possible that changes in mother mortality could result  
9 from cross-generational fitness trade-offs between mothers and daughters. This  
10 hypothesis has been difficult to assess because, until now, the influence of maternal  
11 mating frequency on offspring fitness in fruit flies was unknown.

12         With respect to the daughter generation, we found that maternal multiple mating  
13 increases the LRS of daughters. Increased mating frequency improves the total fecundity  
14 of daughters by 20%. Many studies have found that multiple mating increases some  
15 components of offspring fitness, particularly in guppies, crickets, dung flies, and  
16 pseudoscorpions, (e.g. Reynolds and Gross 1992; Tregenza and Wedell 1998; Newcomer  
17 et al. 1999; Evans and Magurran 2000; Tregenza et al. 2003; Schmoll et al. 2005), but  
18 only a few studies have examined the effects of multiple mating on both parental and  
19 offspring fitness. For example, in bulb mites (Konior 2001; Kozielska et al. 2004) and  
20 crickets (Fedorka and Mousseau 2002; Fedorka and Mousseau 2004; Head et al. 2005)  
21 multiple mating increases maternal fitness and it also increases offspring fitness. In these  
22 species increased mating is beneficial for both mothers and offspring; however, in at least  
23 15 different species of insects, distributed over six insect orders, increased mating has

1 been shown to substantially reduce the LRS of mothers (Arnqvist and Nilsson 2000;  
2 Kokko *et al.* 2003). Here we show that multiple mating can increase the LRS of offspring  
3 in a species where there are substantial costs of multiple mating to mothers.

4 Previous work has focused on the genetic consequences of multiple mating  
5 (Jennions and Petrie 2000, though see Qvarnstrom and Price 2001; Priest and Promislow,  
6 in press). However this study implicates parental effects, rather than genetic benefits, for  
7 the mechanism of how multiple mating affects offspring quality. The principal factors  
8 driving costs of mating to females are accessory gland proteins (Acp), which are  
9 produced by males and delivered to females in male seminal fluid (Chapman *et al.* 1995).  
10 Acp target tissues throughout the reproductive tract, some even enter the hemolymph of  
11 females and change female physiology (Wolfer 2002; Ram *et al.* 2005). Maternal  
12 exposure to Acp also increases the early fecundity and fitness of daughters (Priest 2006).  
13 Thus, the effects of maternal multiple mating on daughter fecundity and fitness reported  
14 here may occur through Acp-induced parental effects.

15 There has been extensive debate about how to measure fitness in studies of sexual  
16 selection. Behavioral ecologists have primarily advocated LRS as the best estimate of  
17 fitness, which is equivalent to evaluating fitness ( $w_i$ ) at  $m = 0$  (Charlesworth 1996).  
18 However, life history theorists have endorsed calculating  $\lambda$ , the value of  $m$  which yields a  
19 fitness of 1 in the Euler-Lotka equation to estimate fitness (Charlesworth 1996). Because  
20 we evaluated our demographic data across a range of population growth rates ( $m$ ) our  
21 approach provides insights into the importance of growth rate assumptions in estimates of  
22 fitness.

1           The results of our study indicate that the consequences of female reproductive  
2 strategies greatly depend on the value of population growth rate ( $m$ ) that is assumed in  
3 the fitness estimate (see also Tatar and Promislow 1997). If we only focus on the  
4 maternal generation, then at  $m = 0$  multiple mating has significant fitness costs for  
5 females, but at highly positive values of  $m$  (which is closer to the value of  $\lambda$  in ideal  
6 culture conditions) multiple mating is not costly for females. Multiple mating does not  
7 reduce fitness at positive values of  $m$  because early-life survival and fecundity of mothers  
8 from the different mating treatments is equivalent (see Figure 1a and Appendix Figure  
9 1a) and in rapidly expanding populations it is early-life reproduction that makes the  
10 greatest compounded contribution to fitness. In previous studies, the survival costs of  
11 multiple mating in the Dahomey strain had only been observed when the flies were  
12 cultured in ideal (positive  $m$ ) culture conditions, not when food was restricted (zero or  
13 negative  $m$ ) (Chapman and Partridge 1996), which is similar to findings in *D.*  
14 *pseudoobscura* (Turner and Anderson 1983). These costs are ameliorated when a cross-  
15 generational approach is used. The implication of this finding is that there is no evidence  
16 for costs of mating to mothers in the Dahomey line of fruit flies when  $\lambda$  is used as the  
17 fitness estimate.

18           In an inclusive fitness approach an individual's fitness is modified to account for  
19 the influence of the individual on the fitness of relatives. This concept is important  
20 because it provides a hypothesis for how traits that are harmful to individuals can evolve.  
21 However, this has been difficult to empirically test because it has not been clear how to  
22 calculate the influence of individuals on the fitness of relatives (Grafen 1984; Creel and  
23 Wasser 1994; Griffin and West 2002). It is also not clear how to measure inclusive fitness

1 in between-generation studies (Grafen 1984, 1988; Charlesworth and Charnow 1981; Oli  
2 2003). Life history and quantitative genetic theory indicate that methods that account for  
3 population growth rate should be used when population growth is non-zero and when  
4 there are cross-generational fitness tradeoffs between parents and offspring (Brommer *et*  
5 *al.* 2002; Grafen 1988). In this study, we used an age-specific cross-generational  
6 approach to determine whether the costs of increased mating frequency are accounted for  
7 by fitness benefits to daughters. We found that when the mother and daughter fitness  
8 were combined in an age-specific cross-generational model, at each assumed value of  $m$   
9 the costs of increased mating frequency to mothers were balanced by the benefits to  
10 daughters. These results indicate that there is no evidence that increased mating  
11 frequency has fitness costs to mothers.

12         These results have implications for the evolution of multiple mating behaviors. It  
13 is thought that multiple mating behaviors either evolve because of sexual conflict, where  
14 traits evolve that benefit males but harm females, or mutualism, where traits evolve that  
15 benefit both sexes (Pizzari and Snook 2003). In one model, multiple mating would be  
16 expected to decrease the inclusive fitness of females, in the other multiple mating would  
17 increase the inclusive fitness of females. This study does not support either idea, as the  
18 inclusive fitness of females was neither significantly improved nor diminished by  
19 increased maternal mating frequency.

20         Instead, the results require a different model of mating behavior evolution. We  
21 found that when females are restricted from free access to males they respond by living  
22 longer and producing offspring with lower fecundity. Frequent mating increases the risk  
23 of death and accelerates the aging process of mothers, but it might be selected for if it

1 changes resource allocation to offspring and increases the probability of producing highly  
2 fertile offspring. There is genetic variation in the effect of multiple mating on offspring  
3 fitness (Priest and Promislow, in press). If there are genetic correlations between costs of  
4 multiple mating to mothers and fitness benefits to offspring, then mating frequency could  
5 evolve as an indirect consequence of natural selection on inclusive fitness. Thus, mating  
6 frequency might evolve as a result of fitness tradeoffs between parents and offspring, not  
7 just mutualism or sexual conflict. One finding that supports this perspective is that mating  
8 frequency can change as a correlated response to selection on longevity (Pletcher et al.  
9 1997; Sgro *et al.* 2000).

10 Our results also have general implications for life history evolution. We found  
11 that mothers with the highest rate of aging (age-specific mortality) produce offspring with  
12 the highest fitness. If there is a genetic basis to this physiological correlation, then it is  
13 possible that selection to optimize inclusive fitness can drive the evolution of shorter life  
14 and more rapid aging. This result might also help explain why we often find positive  
15 genetic correlations between survival and fecundity in natural and laboratory populations,  
16 a pattern which runs contrary to theoretical predictions (Reznick et al. 2000). Though  
17 certain lineages might produce parents that appear to be short-lived and of low fecundity,  
18 the lineages could have high fitness and persist over time because they produce offspring  
19 that are well suited for the prevailing conditions in the population. Thus, an inclusive  
20 fitness theory of aging that accounts for population growth rate might help explain the  
21 persistence of genetic variation in aging.

22

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17  
18

1 Table 1. Ln Likelihood analysis of the effects of maternal mating frequency on mortality  
 2 intercept (A), rate (B) and leveling-off (S) parameters of the logistic mortality  
 3 decomposition for mothers and daughters. The analysis examines mortality differences  
 4 between pairs of treatments.  $X^2$  statistics are reported with 1 d.f. and statistical  
 5 significance, \*\*\*,  $P < 0.001$ ; \*\*,  $P < 0.01$ .

6

7	Generation	Comparison	Intercept (A)	Rate (B)	Leveling-off (S)
8	Mother	High vs Low:	159.90***	6.912 **	0.781
9		Med vs. Low:	39.24***	3.033	3.235
10		High vs. Med:	71.35***	0.540	1.148
11					
12	Daughter	High vs Low:	0.716	0.751	0.059
13		Med vs. Low:	0.027	0.089	1.350
14		High vs. Med:	0.661	0.964	2.314

15

16

17

18

1 Table 2. Repeated measures analysis of variance on effects of maternal mating frequency  
 2 on age-specific changes in % egg viability and fecundity (# eclosed offspring) of mothers  
 3 and daughters. The F-statistics are reported with numerator degrees of freedom in  
 4 parentheses, and with statistical significance, \*\*\*, P <0.001; \*\*, P < 0.01; \*, P <0.05. The  
 5 error d.f. for the egg-to-adult viability and age-specific fecundity analyses were 268 and  
 6 435, respectively.

7

8

9	Generation	Variable	Age	Mating	Mating x Age	Block
10	Mother	Egg Viability:	34.72 (4)***	0.47(2)	1.46 (7)	14.37(2)***
11		Fecundity:	20.82 (6)***	17.65(2)***	1.57(12)	2.63(2)
12						
13	Daughter	Egg Viability:	38.19 (4)***	1.80(2)	0.34 (8)	1.66(2)
14		Fecundity:	111.98 (6)***	21.05(2)***	0.64(12)	45.39(2)***

15

16

17

Figure 1. a) Survival curves for mothers from High-, Medium- and Low-mating treatments. b) Survival curves for the daughters derived from the mothers from High-, Medium- and Low-mating treatments.

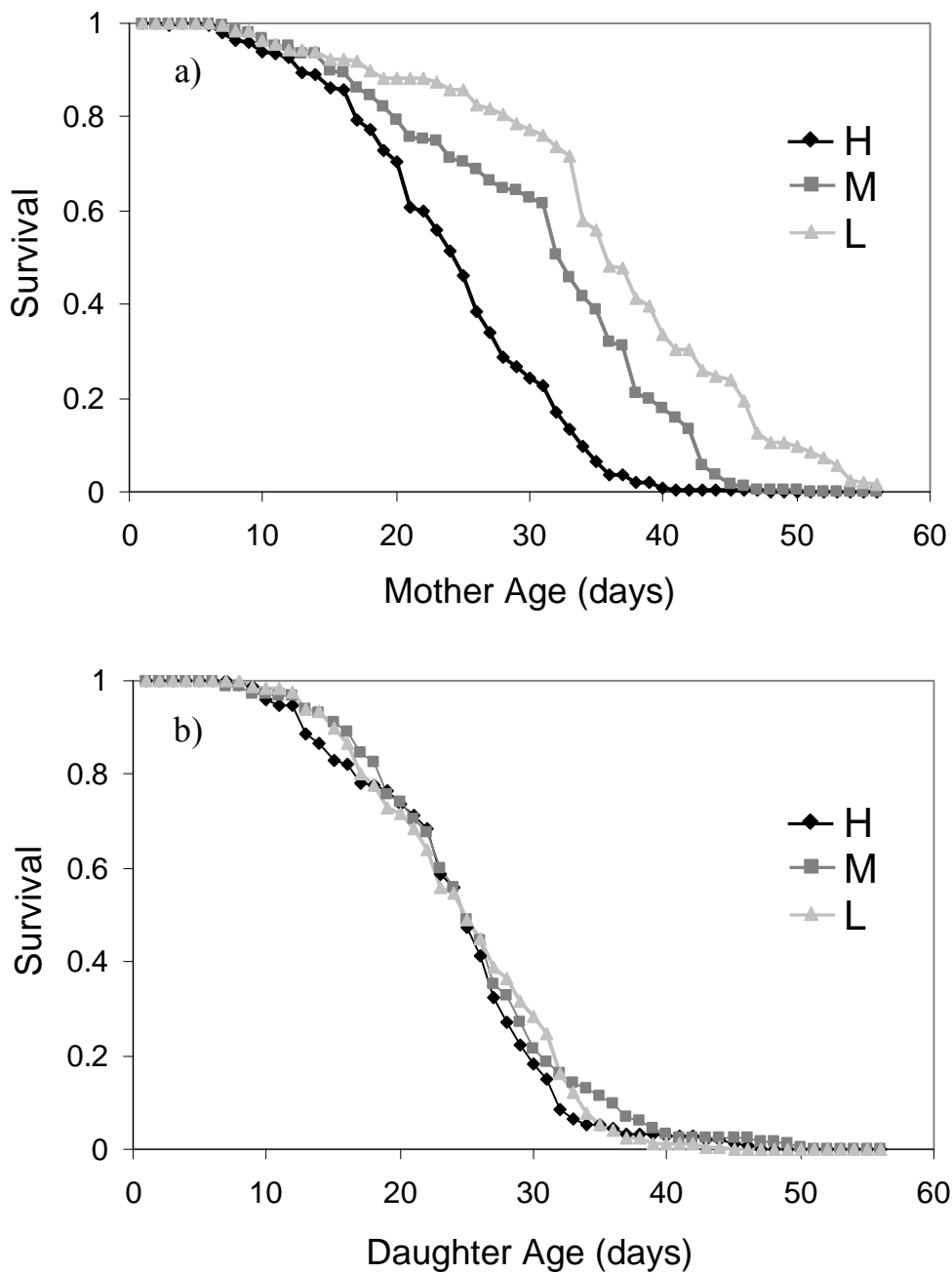


Figure 2. a) The age-specific mortality curves for mothers from High-, Medium- and Low-mating treatments fit to a logistic model. High-maternal mating frequency shows significantly higher background mortality (intercept) and rate of aging (slope). b) The age-specific mortality curves of daughters derived from the mothers from High-, Medium- and Low-mating treatments.

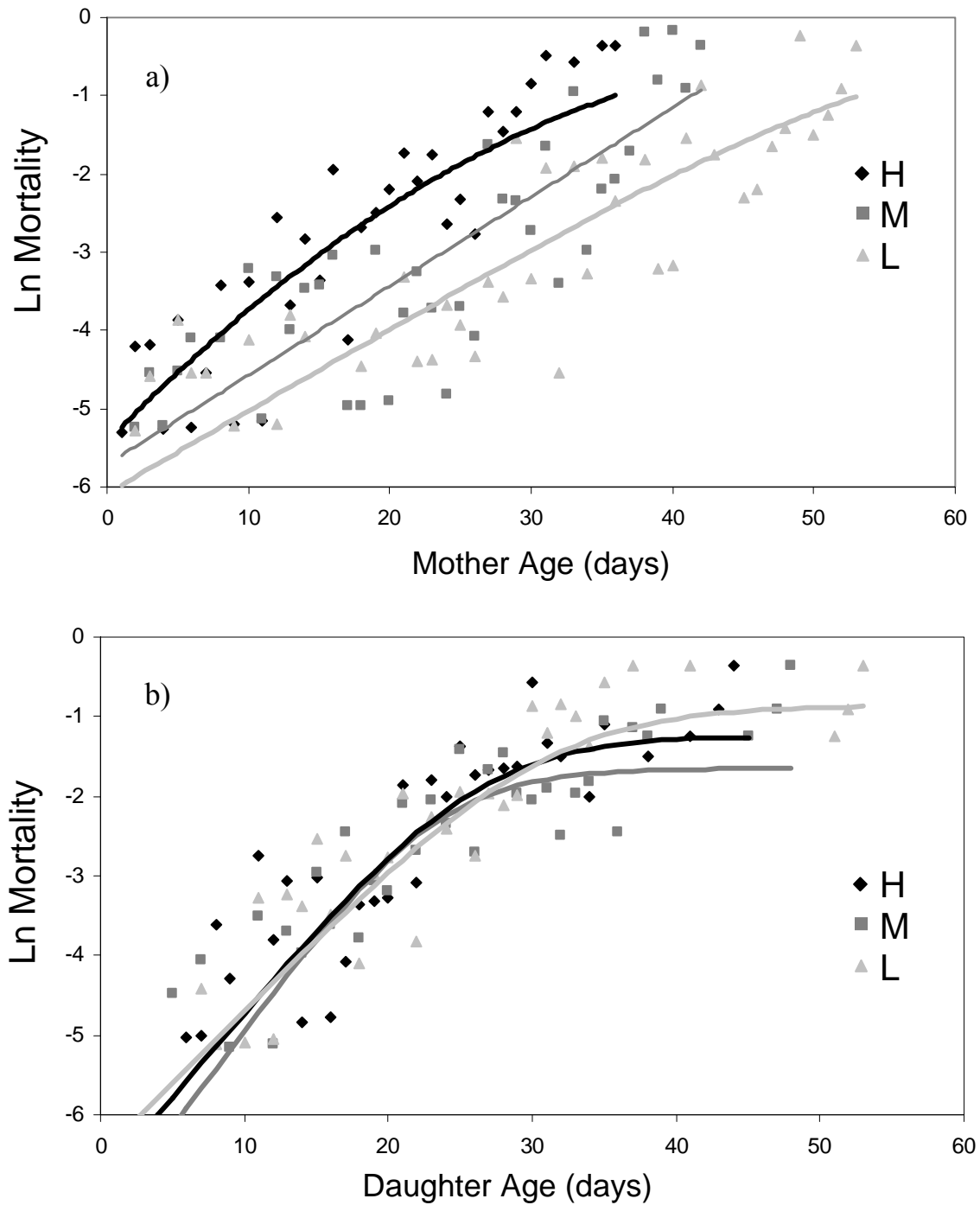


Figure 3. The lifetime reproductive success (LRS) of High-, Medium- or Low-mating a) mothers (mean  $\pm$  SE) and b) their daughters (mean  $\pm$  SE). The different letters indicate that the means differ at  $\alpha = 0.05$ .

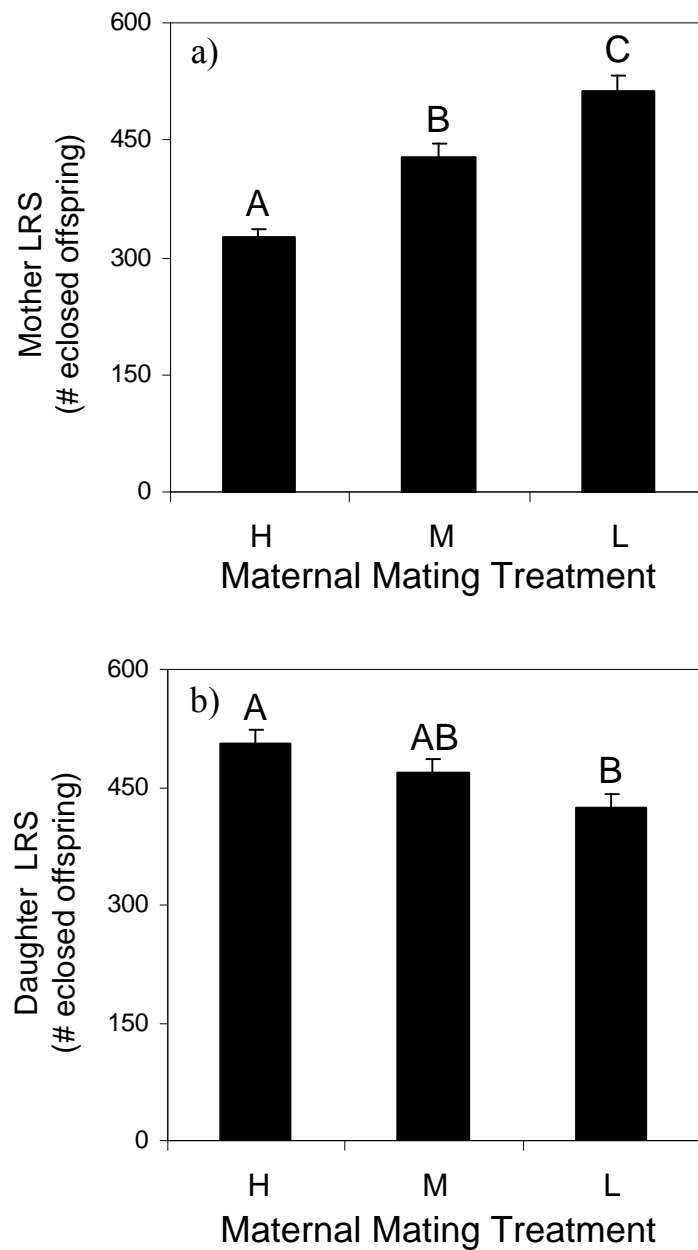


Figure 4. The effect of maternal mating on a) the fitness of mothers, b) the fitness of daughters, and c) inclusive fitness. The fitness of High- and Medium-mating treatments are depicted relative to Low-mating. The effect of mating on fitness is reported for each value of  $m$ : \*\*\*,  $P < 0.001$ ; \*\*,  $P < 0.01$ ; \*,  $P < 0.05$ ; NS,  $P > 0.05$ .

