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Offspring Provisioning Explains Clone-Specific Maternal Age Effects on Life History and Life Span in the Water Flea, *Daphnia pulex*

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abstract: Genetic inheritance underpins evolutionary theories of aging, but the role that nongenetic inheritance plays is unclear. Parental age reduces the life span of offspring in a diverse array of taxa but has not been explained from an evolutionary perspective. We quantified the effect that maternal age had on the growth and maturation decisions, life history, rates of senescence, and life span of offspring from three *Daphnia pulex* clones collected from different populations. We then used those data to test general hypotheses proposed to explain maternal age effects on offspring life span. Three generations of breeding from young or old mothers produced dramatic differences in the life histories of fourth-generation offspring, including significant reductions in life span. The magnitude of the effect differed between clones, which suggests that genetic and nongenetic factors ultimately underpin trait inheritance and shape patterns of aging. Older parents did not transmit a senescent state to their offspring. Instead, offspring from older ancestors had increased early-life reproductive effort, which resulted in an earlier onset of reproductive senescence, and an increased rate of actuarial senescence, which shortened their life span. Our results provide a clear example of the need to consider multiple inheritance mechanisms when studying trait evolution.

Keywords: transgenerational plasticity, rates of senescence, Lansing effect, probabilistic maturation reaction norms, nongenetic inheritance, *Daphnia pulex*.

Introduction

Aging or senescence is the intrinsic deterioration of an organism with age, leading to decreased physiological functioning and, ultimately, death (Medawar 1952; Williams 1957; Rose 1991; Partridge and Gems 2006; Flatt and Schmidt 2009; Partridge 2009). Evolutionary theories of aging predict that between-individual differences in rates of senescence should have a heritable genetic basis (reviewed in Rose 1991); this hypothesis is supported by the results of numerous laboratory-based experiments (reviewed in Flatt and Schmidt 2009; Partridge 2009) and evidence from wild populations (Charmantier et al. 2006; Wilson et al. 2007). However, there is an increasing realization that parents may also alter the phenotypes of their offspring via nongenetic inheritance mechanisms, such as the transmission of epigenetic variation, the transmission of plastic phenotypes (acquired traits), and the effects of parental environment and state on offspring phenotype (Uller 2008; Bonduriansky and Day 2009; Danchin and Wagner 2010; Danchin et al. 2011; Bonduriansky 2012; Hallsson et al. 2012; Danchin 2013). However, we are only just beginning to investigate how genetic and nongenetic inheritance mechanisms combine to shape offspring traits (Badyaev and Uller 2009; Bonduriansky and Day 2009; Hallsson et al. 2012; Danchin 2013; Townley and Ezard 2013; Uller 2013).

Parental age effects on offspring life histories offer an example of nongenetic inheritance that is especially interesting with respect to the evolution of aging and life span (Priest et al. 2002). Evolutionary theories do not typically assume that the age of the parent has any proximate influence on rates of senescence of offspring (but see Kong et al. 2012), yet offspring from older parents have been found to have a shorter life span in taxa as diverse as rotifers, duckweed, houseflies, stink bugs, fruit flies, flour beetles, mealworms, nematodes, yeast, and humans for over a hundred years (reviewed in Priest et al. 2002; Fox et al.

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2003). This pattern is termed the Lansing effect, after Albert Lansing (1947, 1948, 1954), who demonstrated that rearing successive generations of clonal rotifers, *Philodina citrina* and *Euclanis triquetra*, from old mothers dramatically reduced offspring longevity. There is still no accepted explanation for this effect (Priest et al. 2002). Moreover, the validity and general importance of Lansing's findings have repeatedly been questioned (Comfort 1953; King 1983; Rose 1991), despite the fact that supportive evidence continues to accumulate (Jennings and Lynch 1928; Lansing 1947, 1948, 1954; Murphy and Davidoff 1972; Lints and Hoste 1974, 1977; King 1983; Beardmore and Shami 1985; Hercus and Hoffmann 2000; Priest et al. 2002; Fox et al. 2003; Tarín et al. 2005; Gillespie et al. 2013). Reservations about the Lansing effect derive from Lansing's (1947, 1948, 1954) failure to identify the mechanism underpinning the effect and from subsequent studies having focused on testing for a Lansing effect, rather than explaining it (but see King 1983 for an exception). Previous studies have linked parental age with alterations in reproductive schedules (King 1983; Bouwhuis et al. 2010), mortality rates (Priest et al. 2002), investment in offspring (reviewed in Marshall et al. 2010), and offspring viability (Hercus and Hoffmann 2000; Kern et al. 2001; Priest et al. 2002; Tarín et al. 2005; Benton et al. 2008; Reid et al. 2010). However, no study has ever simultaneously linked parental age, patterns of offspring development, offspring life histories, and rates of aging. Here we collect these data to test two general hypotheses that could explain how parental age effects (including the Lansing effect) have evolved as a form of nongenetic inheritance.

The senescent parent hypothesis proposes that offspring from older parents die sooner because they transmit their physiological deterioration to their offspring, via a direct reduction in the provisioning of offspring, a deterioration of the offspring's developmental environment, or the transmission of a higher mutation load (Kong et al. 2012). The hypothesis is supported by studies that have demonstrated that older, senescent parents produce offspring with reduced fitness attributes (Kern et al. 2001; Diaz and Esponda 2004; Tarín et al. 2005; Gillespie et al. 2013) and predicts that offspring from older mothers are born with an elevated "biological age," meaning that they have higher age-independent mortality rates (frailty), lower age-independent reproductive potential, and lower growth rates.

In addition, the nonmutually exclusive offspring response hypothesis proposes that offspring from older parents die sooner because the plastic responses that they adopt to counteract parental age-related changes in offspring provisioning are costly. If older parents provision offspring less, offspring might employ compensatory strategies, such as catch-up growth (Boersma and Wit 1997), that lead to an earlier onset or accelerated rate of senescence and a shortened life span (Metcalfe and Monaghan 2001; Monaghan 2008). Alternatively, they might adopt a terminal-investment-like strategy (Williams 1966; Cluttonbrock 1984) that increases early-life reproductive effort but also induces costs that shorten the life span (Gustafsson and Pärt 1990; Reid et al. 2003; Nussey et al. 2006; Descamps et al. 2008; Reed et al. 2008; Massot et al. 2011). A reduction in offspring life spans could also derive from mothers that invest more in their offspring as they age (see Marshall et al. 2010 for a review of causes and examples of positive relationships between maternal age and offspring size) if a head start in life facilitates faster growth rates and increased early-life reproductive effort (Ebert 1994; Fox and Czesak 2000).

Irrespective of whether parental reproductive investment increases or decreases with age, the offspring response hypothesis predicts that the offspring from older mothers will have increased growth rates and/or increased early-life reproductive effort and an earlier onset or accelerated rate of actuarial and/or reproductive senescence that shortens offspring life span. The hypothesis is supported by studies linking increased investment in early growth and reproduction with late-life costs (Metcalfe and Monaghan 2001; Monaghan 2008) and proximate mechanisms linked with increased rates of senescence, such as oxidative stress (Monaghan et al. 2009), telomere loss (Hall et al. 2004; Monaghan and Haussmann 2006; Houben et al. 2008), and stress responses (McEwen 2007). Exactly how parental age translates into offspring with increased early-life reproductive effort is unclear, but the size and age at which individuals mature may play a key role, because body size often constrains fecundity (Roff 1992). Our recent finding that the decision to mature (modeled as a probabilistic maturation reaction norm [PMRN]) is itself plastic (Harney et al. 2013) raises the possibility that parental age may alter the maturation decisions of their offspring as well as influence offspring growth rates, although this hypothesis has never been tested.

The four objectives of this study were therefore to (1) quantify the effect that nongenetic inheritance, here manifested through maternal age effects, has on the life history, life span, and rates of actuarial and reproductive senescence of *Daphnia pulex*; (2) distinguish between the two hypotheses that we have proposed to explain the Lansing effect; (3) determine whether the pattern and the magnitude of nongenetic inheritance effects vary between clones; and (4) determine whether a plastic response of offspring to maternal age–related changes in offspring provisioning, predicted by the offspring response hypothesis, includes a plastic adjustment in the age and/or size at which offspring decide to mature. The first three objectives were tested in an experiment using three *D. pulex* clones isolated from three different populations. We repeatedly bred from young

or old mothers for three generations to create maternal age lines (see fig. 1*A*). We then reared offspring from the fourth generation and quantified the effect that genotype and three generations of maternal age inheritance had on offspring size, growth rate, age and size at maturity, age-specific reproductive effort, age-specific mortality rate (hazard rate), and survival. The fourth objective was tested using a separate experiment (see fig. 1*B*), because maturation decisions are modeled as a process or reaction norm rather than as an individual trait (Heino et al. 2002; Van Dooren et al. 2005) and require a range of growth trajectories for their quantification (Harney et al. 2013). The second experiment provides an independent test of the hypothesis that maternal age influences offspring life histories, but over a single generation, rather than over accumulated generations.

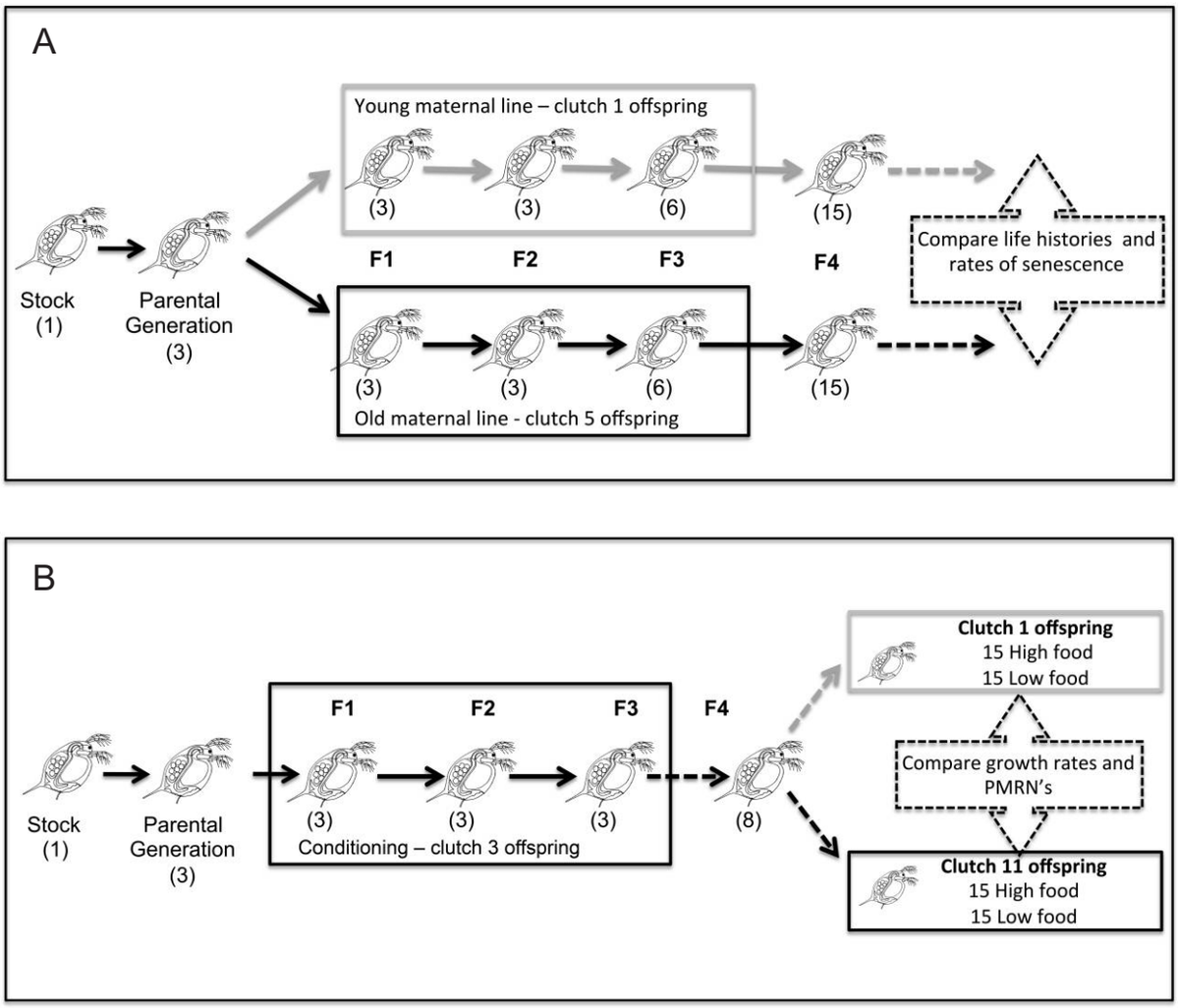


Figure 1: *A*, Experimental design used for experiment 1. For each of the three clones used in this experiment (Boris, D8.7A, NBG70), young and old maternal age lines were created by randomly selecting offspring from young mothers (clutch 1) or old mothers (clutch 5) to set up each new generation. The effect of maternal age line on offspring life histories, life span, and rates of senescence was then compared in the fourth generation by randomly selecting 15 offspring from the first clutch of females in the young maternal age line and 15 offspring from the fifth clutches of females in the old maternal age line (see "Methods" for details). *B*, Experimental design used for experiment 2. For each of the three clones used (Boris, D8.7A, NBG70), offspring from middle-aged mothers (clutch 3) were conditioned with a diet of high food for three generations. The numbers in brackets refer to the number of mothers set up in each generation. In the fourth generation, the effect that maternal age had on offspring development was tested by comparing the growth rates and maturation decisions (probabilistic maturation reaction norm [PMRN]) of 30 randomly selected offspring from mothers' first clutches and 30 randomly selected offspring from the same mothers' eleventh clutches (see "Methods" for details).

Methods

Experimental Animals

Daphnia pulex clones used in this study were isolated from various sites in the United Kingdom. Clone NBG70 came from a pond in Ness Botanical Gardens, Ness, Merseyside, United Kingdom (53°16′16″N, 03°02′47″W); clone Boris came from a pond in Sheffield, South Yorkshire, United Kingdom (53°24′18″N, 01°27′27″W); and clone D8.7A came from a pond near Corfe Castle in Dorset, United Kingdom (50°38′33″N, 02°05′58″W). Since being isolated, the clones have been maintained in incubators at a mean (±SD) temperature of 21° ± 1°C on a 14L:10D cycle and kept in hard artificial pond water media (ASTM International; OECD 1998), enriched with a standard organic extract (Baird et al. 1989).

Experiment 1: Genotype and Maternal Age Line Effects on Offspring Life History, Life Span, and Rates of Senescence

The experimental design is outlined in figure 1*A*. All animals in the experiment were reared in individual 200-mL glass jars containing 150 mL of ASTM enriched with a standard organic extract (Baird et al. 1989), replaced every other day, and were fed high food (200,000 cells mL^{-1} day^{-1} of batch-cultured *Chlorella vulgaris*, quantified with a hemocytometer). For each clone, a single female was isolated from stocks and reared with high food until she produced at least three offspring in a clutch. From that clutch, three offspring were randomly selected and reared individually to become the parental generation (see fig. 1*A*). Young and old maternal lines were set up from the parental generation using offspring from mothers' first clutches as the young maternal line and offspring from their fifth clutches as the old maternal line. To minimize the possibility that within-clone lineage effects (Sakwinska 2004) or clonal selection of de novo mutations or epimutations could explain any of our treatment effects, we set up each new generation of the experiment with randomly selected offspring from multiple mothers that produced offspring within a 12-h period (see fig. 1). On occasion, offspring may have come from fewer than three mothers (not recorded) if mothers did not produce offspring at the same time. However, systematic bias due to female mortality was unlikely, because almost all mothers survived to fifth clutch during the setup of the experiment (see table S1, available online, also available in the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.3k8c0; Plaistow et al. 2015). By the end of the experiment, fourth-generation offspring had mothers, grandmothers, and great-grandmothers that had all come from first or fifth clutches (see fig. 1*A*). The life history, life span, and rates of senescence of these fourth-generation offspring were then compared.

For each clone, 15 first-clutch offspring from the young maternal line and 15 fifth-clutch offspring from the old maternal line were randomly selected from the pooled offspring of six mothers that had dropped their clutches within a 12-h window. All individuals were photographed as neonates and then every time they molted throughout their life using a Canon EOS 350D digital camera connected to a Leica MZ6 dissecting microscope. Body size was measured as the distance from the top of the head to the base of the tail spine using the image analysis software ImageJ, version 1.45s (Rasband 1997). Prematuration growth rates for each individual were estimated as the best limited unbiased predictors from a linear mixed-effects model with size as the response variable, age as a covariate, and individual fitted as a random term for intercept and slope (Crawley 2002). Individuals were recorded as being mature once eggs were observed in the brood pouch. We counted the number of offspring each individual produced in each clutch for the rest of their lives until the day of death.

Experiment 1: Statistics

The effects that clone and maternal age line had on the life history and life span of fourth-generation offspring were compared using a MANOVA with neonate size, growth rate (intercept), growth rate (slope), size at maturity, age at maturity, lifetime fecundity, and life span as response variables and clone (Boris, D87A, NBG70) and maternal age line (young, old) fitted as fixed factors. Differences were then visualized and interpreted using a principal component analysis (PCA) to summarize how maternal age line altered the life histories of fourth-generation offspring. Because PCAs cannot be used for hypothesis testing, pairwise differences in clone responses to maternal age line treatment were tested by comparing the length and angle of phenotypic change vectors following the methodology of Collyer and Adams (2007).

The data collected in experiment 1 were also used to compare the age-specific reproduction and mortality rate of offspring from young and old maternal age lines. Data for all three clones were combined to maximize sample size and statistical power, but clone-by-treatment interactions were also tested in a subsequent analysis. Age-specific reproduction was modeled using generalized additive mixed models (GAMMs) implemented using the gam function from the mgcv library, version 1.7-23 (Wood 2011), in R, version 3.0.1, but with random effects incorporated as penalized regression terms (Wood 2011). In all models, the default settings of the gam package were used, with the number of knots (k) being estimated as part of the fitting

procedure (Wood 2011). After fitting a simplistic model with the same smoothing function for age for all individuals, and with individual included as a random intercept and smoothing function, the effects of maternal age line and clone and the interactions between them were assessed using Akaike information criterion (AIC) and by statistically comparing the residual variance explained by models of increasing complexity using *F* statistics (Crawley 2002; Wood 2011). Following Jones et al. (2008), the age at the onset of senescence was estimated for each group as the point of peak fecundity predicted by the GAMM. Differences in the rate of reproductive senescence were then tested using a linear mixed-effects model with clutch size following the onset of senescence fitted as a response variable, age after the onset of senescence fitted as a covariate, maternal age line fitted as a fixed factor, and individual fitted as a random term.

Age-specific mortality rates were compared by fitting parametric survival models implemented using the flexsurvreg function within the flexsurv package, version 0.3, in R, version 3.1.0. Note that mortality rates cannot be unambiguously measured directly from the data, because each death could be interpreted as an instantaneous burst of infinite mortality rate. We are only able to infer mortality rates by fitting a survival model to the data or estimate them by binning the survival events (as in Bronikowski and Flatt 2010). Because we have only 10–15 individuals per clone/treatment combination, binning the data does not provide enough mortality rate:age combinations to make any robust inferences. Nevertheless, there are enough points to fit survival models directly to the (unbinned) data and to test hypotheses about how these depend on inferred mortality rates, clone, and treatment.

A suite of survival distributions (exponential, Weibull, Gompertz, and piecewise-linear hazard) were fitted, under the assumption that the parameters of the distribution depended on the treatment (maternal age line) and on clone, and the AIC corrected for small sample sizes (AICc) was used to choose the distribution to use in the analysis. Likelihood ratio tests (LRTs) were used to investigate how the survival curves depended on the treatment and/or on the clone, assuming that the likelihood ratio followed a χ^2 distribution. Survival was assumed to depend on the maternal age line when testing for the effect of clone and vice versa. A *P* value is not given for the interaction between these factors, because there is no meaningful way to have direct effects but no interaction in this type of model. This is because the absence of an "interaction" would mean that the parameters could be expressed as the sum of separate effects from the different explanatory variables, but this has no particular biological significance, because the hazard depends on these parameters in a nonlinear way.

Experiment 2: Genotype and Maternal Age Effects on Growth and Maturation Decisions

We ran a separate experiment alongside experiment 1 to test the hypothesis that maternal age affects the maturation decisions of offspring in *D. pulex* (see fig. 1*B*). Three replicate offspring from each clone (parental generation females in fig. 1*B*) were individually reared to maturity on high food (200,000 cells mL^{-1} day^{-1} of *C. vulgaris*) as above. To remove any unwanted maternal effects and to condition animals to their current environment, clones were conditioned for three generations using third-clutch offspring to set up each new generation (see fig. 1*B*). In the F_4 generation, the number of mothers used to set up the experiment was increased to eight to ensure that enough offspring would be available (see table A1 for the data on the mortality of mothers used to set up the experiment; tables A1–A3 available online). Thirty randomly selected offspring from young mothers (clutch 1) and 30 randomly selected offspring from old mothers (clutch 11) were isolated for each clone; for each group of 30 offspring, 15 were reared individually with high food (200,000 cells mL^{-1} day^{-1} of *C. vulgaris*), and the other 15 were reared individually with low food (40,000 cells mL^{-1} day^{-1} of *C. vulgaris*). Offspring were photographed as neonates and after every molt and were measured as in experiment 1. Size and age data were collected until offspring had dropped their first clutch.

Experiment 2: Statistics

The prematuration growth rates of offspring in experiment 2 were compared using a linear mixed-effects model with size as the response variable, age as a covariate, maternal age (young, old) and clone (Boris, D8.7A, NBG70) fitted as fixed factors, and individual fitted as a random term for intercept and slope. The models were implemented using the lmer function within the LME4 package, version 1.7-23 (Bates et al. 2014), in R, version 3.0.1. The process of maturation is stochastic rather than deterministic, such that genetically similar individuals reared under similar environmental conditions may still undergo maturation at different ages and sizes (Bernardo 1993; Morita and Morita 2002). We have previously shown that the decision to mature in *Daphnia* is best modeled using age and size intervals that precede ovary formation (stage IM-1) using a logit-link generalized linear model (GLM), potentially with an offset (Harney et al. 2013). Consequently, we used the same approach here. Initially, models were fitted that contained clone (Boris, D8.7A, NBG70) and maternal age line (old, young) as fixed factors, age and size as covariates, and the interactions between these variables. Food was not included as a factor, because to effectively fit PMRNs, individuals must mature at a range

of ages and sizes (Heino et al 2002), and rather than being a treatment, different food levels therefore serve to generate a range of growth trajectories. Maturation rates are then integrated over size or age or both (see Harney et al. 2013). To find the model that best fitted the data, a large number of different GLMs (57 in total) were fitted simultaneously, and the model with the lowest AIC was selected. In different models, age and size were fitted as covariates singly (age or size) and in combination (age and size) using interval start points, midpoints, or end points and with either untransformed or log-transformed values, and GLMs were fitted with and without offsets for intervals, with both age offsets and size offsets considered (see Harney et al. 2013 for more details). Once the model with the lowest AIC had been chosen from among these 57 possible models, the importance of clone, maternal age, and their interactions with each other and with age and size was determined using likelihood ratio tests. PMRNs were visualized by simulating growth curves for the best-fitting model and calculating maturation probabilities per curve, then approximating and plotting the twenty-fifth, fiftieth, and seventy-fifth percentiles (see Van Dooren et al. 2005 for details). All statistical analyses and generation of PMRNs were conducted in R, version 2.13.2, using packages Hmisc (Harrell 2012*a*), gplots (Warnes 2012), lme4 (Bates et al. 2014), MASS (Venables and Ripley 2002), survival (Therneau 2013), rms (Harrell 2012*b*), and arm (Gelman and Yu-sung 2014). All data from this article are available in the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.3k8c0 (Plaistow et al. 2015).

Results

Experiment 1

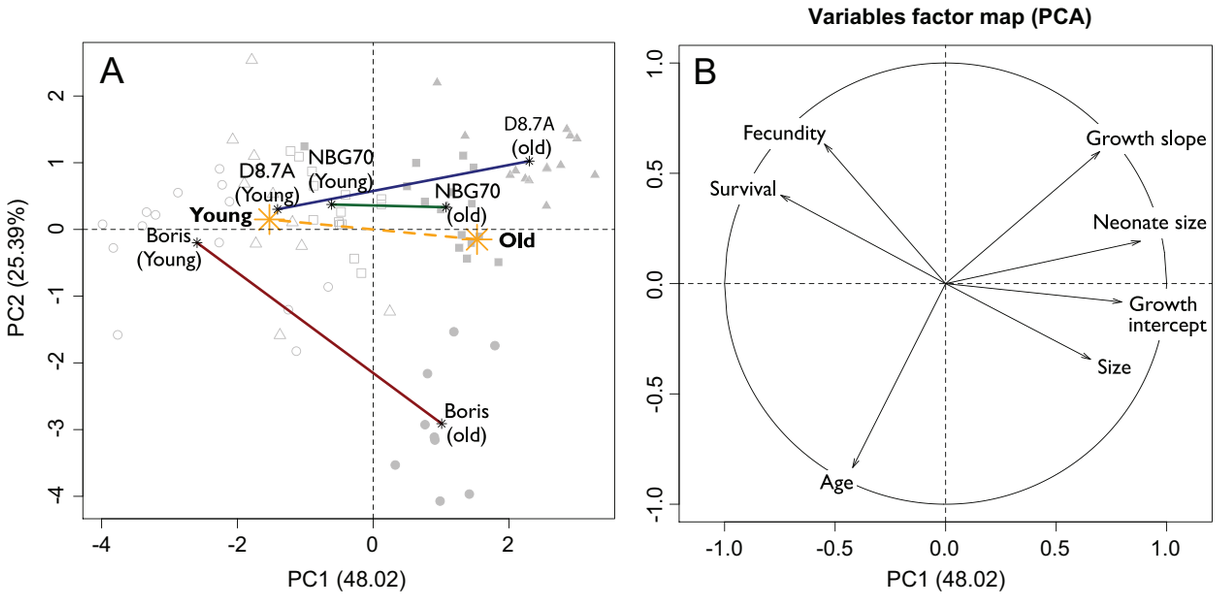
Effect of Clone and Maternal Age Line on Offspring Life History and Life Span. The life histories of fourth-generation offspring differed between clones (Pillai's trace = 1.051, $F_{2,74} = 10.938$, $P < .0001$, MANOVA), but the nongenetic inheritance effect associated with maternal age line was even stronger (Pillai's trace = 0.825, $F_{2,74} = 45.728$, $P < .0001$, MANOVA) and varied between clones (Pillai's trace = 1.432, $F_{2,72} = 24.149$, $P < .0001$; table A2). In the PCA, 48% of the total offspring phenotypic variation was explained by principal component 1, which was closely aligned with maternal age line ($R^2 = 0.7$, $P < .0001$; see fig. 2*A*–2*C*). Offspring from the old maternal line were larger neonates than those from the young maternal line, with higher growth rates and larger size at maturation, but they also had reduced life span and reduced lifetime reproductive success (fig. 2*B*, 2*C*). Principal component 2, which explained 25.4% of the total life-history variation, was predominantly associated with offspring from the old maternal age taking longer to mature in clone Boris. The pairwise comparisons of the phenotypic change vectors revealed that maternal age line effects were of the same magnitude in clones Boris and D8.7A but significantly reduced for clone NBG70. In contrast, the angle of the phenotypic change vector differed for all three clones, demonstrating that responses to maternal age line were all clone specific (see fig. 2*D*).

Effect of Clone and Maternal Age Line on Offspring Age-Specific Reproduction. The GAMM analysis revealed considerable age-dependent variation in clutch sizes across lifetimes (table 1; model 3 vs. model 2, ΔAIC = 180.4). For all clones together, clutch sizes increased until around day 20–30, after which there was clear evidence of reproductive senescence (see fig. 3*A*). Offspring from the old maternal age line had significantly increased clutch sizes earlier in life (table 1; model 5 vs. model 4, ΔAIC = 167.2) and subsequently demonstrated a much earlier onset of senescence (age, 23 days) compared with offspring from the young maternal age line (age, 34 days; see fig. 3*A*). Once senescence had begun, there was no difference in the rate of reproductive decline in offspring from young and old maternal lines ($\chi^2 = 1.80$, df = 1, $P = .18$, LRT). Adding clone-specific smoothing functions improved the fit of the model (table 1, model 9). All clones showed a similar pattern, with offspring from the old maternal line demonstrating an earlier onset of senescence (see fig. 3*B*–3*D*).

Effect of Genotype and Maternal Age Ancestry on Offspring Age-Specific Mortality Rates. Based on AIC scores, the Gompertz survival distribution in which survival depended on clone and maternal line gave the best fit to the data, although there was some support for the Weibull distribution (ΔAICc = 2.27). The data did not support an exponential distribution (ΔAICc = 165.78), showing that the mortality rates did increase significantly with age in this study ($\chi^2 = 181.6$, df = 3, $P < .001$, LRT). The remaining results assumed a Gompertz survival distribution, although we found the same qualitative conclusions when using a Weibull distribution.

Survival depended significantly on clone ($\chi^2 = 66.67$, df = 8, $P < .001$, LRT) and maternal age line ($\chi^2 = 81.26$, df = 6, $P < .001$, LRT), but the effect of maternal age differed between clones (fig. 4). Age-specific mortality accelerated more quickly in the old maternal lines of clones Boris ($\chi^2 = 37.06$, df = 2, $P < .001$, LRT) and D8.7A ($\chi^2 = 38.84$, df = 2, $P < .001$, LRT), demonstrating a strong Lansing effect. However, age-specific mortality did not differ between maternal lines in NBG70 ($\chi^2 = 5.36$, df = 2, $P > .05$, LRT).

We have included in figure 4*D*–4*F* the estimates of age-specific mortality rates obtained by binning the survival



C

	PC1	PC2
Variance	3.361	1.777
% of variation	48.018	25.393
Cumulative % of variation	48.018	73.411
Variable	*PCA loadings*	
Size	0.656	-0.343
Age	-0.421	-0.834
Neonate size	0.881	0.192
Growth (intercept)	0.797	-0.083
Growth (slope)	0.697	0.599
Fecundity	-0.548	0.633
Survival	-0.746	0.401

D

Contrast	Diff in length	p-value	Diff in angle	p-value
Boris-D8.7A	0.628	0.199	58.472	<0.001
D8.7A-NBG	1.634	<0.001	60.192	<0.001
Boris-NBG	2.262	<0.001	50.444	<0.001

Figure 2: *A*, Scores plot for the principal component analysis (PCA). Individuals from the old maternal line are plotted as filled circles for Boris, filled squares for NBG70, and filled triangles for D87A, whereas individuals from the young maternal lines are plotted as open circles, open squares, and open triangles, respectively. The mean multivariate phenotypes of each clone after three generations of maternal age selection are plotted as stars and linked by solid line vectors, labeled at each end. The average effects of the maternal age selection are plotted as large asterisks, linked by a dashed line vector. *B*, Vector plot of the loadings from the PCA. Vectors that are close in space indicate positive correlations between traits. Vectors that point in opposite directions are negatively correlated, and vectors that are perpendicular are uncorrelated. The length of the vector indicates the amount of variation associated with it. *C*, Details of the variation explained by each component and the loadings. *D*, Pairwise comparisons of differences in the length and direction of phenotypic change vectors, summarizing the differences in the multivariate phenotypes of the three clones when reared from young and old maternal age lines. Significant differences are shown in bold. PC = principal component.

events in 3-day bins (as in Bronikowski and Flatt 2010). These estimates are included only as a guide to the eye, as there are insufficient data points to draw any inferences from the binned data. Note that our models (illustrated by the lines in fig. 4*D*–4*F*) were fitted directly to the survival event data and that there are indeed enough data to test our hypotheses (as illustrated by the statistics quoted in the previous two paragraphs).

Experiment 2

Effect of Genotype and Maternal Age on Offspring Growth Rates and Maturation Decisions. As in experiment 1, offspring from older mothers produced significantly larger neonates. However the effect of maternal age varied between clones (maternal age × clone, $\chi^2 = 555.18$, df = 2, $P < .0001$, LRT; fig. 5*A*–5*C*). These offspring also grew

Table 1: Statistical comparison of additive mixed models in which nonparametric smoothing functions for age were fitted to age-specific clutch sizes for all individuals and subsets of individuals on the basis of maternal age treatment and clone identity

Model	Terms	No. functions	AIC	Model comparison	*F*	*P*
1	R1	1	4,139.3			
2	R1 + R2	2	3,875.7	2 vs. 1	10.66	<.0001
3	A + R1 + R2	3	3,695.3	3 vs. 1	19.24	<.0001
4	T + A + R1 + R2	3	3,690.6	4 vs. 3	1.31	.229
5	T + (T × A) + A + R1 + R2	5	3,523.4	5 vs. 4	33.48	<.0001
6	**T + (T × A) + R1 + R2**	**4**	**3,523.4**	**6 vs. 5**	**2.51**	**.008**
7	C + T + (T × A) + A + R1 + R2	4	3,517.0	7 vs. 5	.90	.502
8	C + (C × A) + T + (T × A) + A + R1 + R2	8	3,329.5	8 vs. 5	14.79	<.0001
9	**C + (C × A) + T + (T × A) + R1 + R2**	**7**	**3,329.5**	**9 vs. 8**	**.59**	**.008**

Note: Maternal age line effects are shown in models 4–6; clone effects are shown in models 7–9. The models that best fitted the data with and without clone effects are shown in boldface type. A = age term for all individuals; AIC = Akaike information criterion; C = clone effect (Boris, D87A, NBG70); R1 = random intercept term for individual; R2 = random age term for individuals; T = maternal age treatment (old line, young line).

faster than the offspring produced from young mothers (maternal age × age, $\chi^2 = 384.26$, df = 1, $P < .0001$, LRT) to the extent that, in all three clones, offspring from old mothers who received low food grew as fast as offspring from young mothers who received high food (fig. 5*A*–5*C*). This result clearly demonstrates that young mothers constrain the growth rates of their offspring. Interestingly, there was no difference in the magnitude of this effect across the different clones (maternal age × clone × age, $\chi^2 = 4.644$, df = 2, $P = .098$, LRT; fig. 5*A*–5*C*). However, there was a significant difference in the way the growth rate of the different clones responded to the food treatment (food × clone × age, $\chi^2 = 549.22$, df = 2, $P < .0001$, LRT; fig. 5*A*–5*C*).

For the analysis of maturation decisions, the GLM with the lowest AIC included a size offset and featured age and size covariates based on interval end points (a table of all models is included in table S1, available online). The minimum adequate model did not feature interactions between maternal age and offspring age ($\chi^2 = 2.206$, df = 1, $P = .1373$, LRT) or size ($\chi^2 = 1.812$, df = 1, $P = .1781$, LRT), suggesting that maternal age does not alter the rate at which incremental increases in size or age influence the probability of maturing. However, there was a significant maternal age-by-clone effect ($\chi^2 = 7.5023$, df = 2, $P = .0234$, LRT) caused by the fact that offspring from older mothers demonstrated a significant upward shift in the size at which maturation was initiated in clones Boris and D8.7A but not in clone NBG70 (fig. 5*D*–5*F*).

Discussion

Understanding how genetic and nongenetic inheritance mechanisms interact to shape trait variation may be crucial for understanding how traits evolve (Day and Bonduriansky 2011; Hallsson et al. 2012; Danchin 2013; Townley and Ezard 2013). With respect to the evolution of senescence, the role that nongenetic inheritance plays has been understudied (Priest et al. 2002). We show here that, in *Daphnia pulex*, maternal age line is the main factor contributing to a principal component that explained 48% of the total offspring phenotypic variation, including substantial reductions in the life span of two of the three clones that we studied. Our results support previous studies of the Lansing effect (Jennings and Lynch 1928; Lansing 1947, 1948, 1954; Davidoff 1972; Lints and Hoste 1974, 1977; King 1983; Beardmore and Shami 1985; Hercus and Hoffmann 2000; Murphy and Priest et al. 2002; Fox et al. 2003; Tarín et al. 2005; Gillespie et al. 2013) and studies linking parental age effects to offspring viability (Hercus and Hoffmann 2000; McIntyre and Gooding 2000; Kern et al. 2001; Priest et al. 2002; Fox et al. 2003; Groothuis et al. 2005; Tarín et al. 2005; Benton et al. 2008; Reid et al. 2010). Moreover, they support the hypothesis that nongenetic inheritance is an integral part of offspring trait evolution (Priest et al. 2002; Badyaev and Uller 2009; Danchin 2013).

The senescent parent hypothesis predicted that older mothers would produce offspring with an elevated "biological age," meaning that they would have lower age-independent reproductive potential, lower growth rates, and intrinsically higher age-independent mortality rates (frailty). Yet, in experiment 1, there was no suggestion that offspring from older mothers had higher age-independent mortality rates (frailty), as might have been expected if older senescent mothers simply transmitted their senescent state to their offspring (Diaz and Esponda 2004). Instead, our results suggest that, in *D. pulex* at least, the Lansing effect is best explained by the offspring response hypothesis. Offspring from older ancestors were larger neonates that grew faster; initiated maturation at the same, or larger, body sizes (fig. 5*D*–5*F*); and demonstrated increased fecundity over the first few clutches laid (fig. 3). This resulted in advanced reproductive senescence (fig. 3)

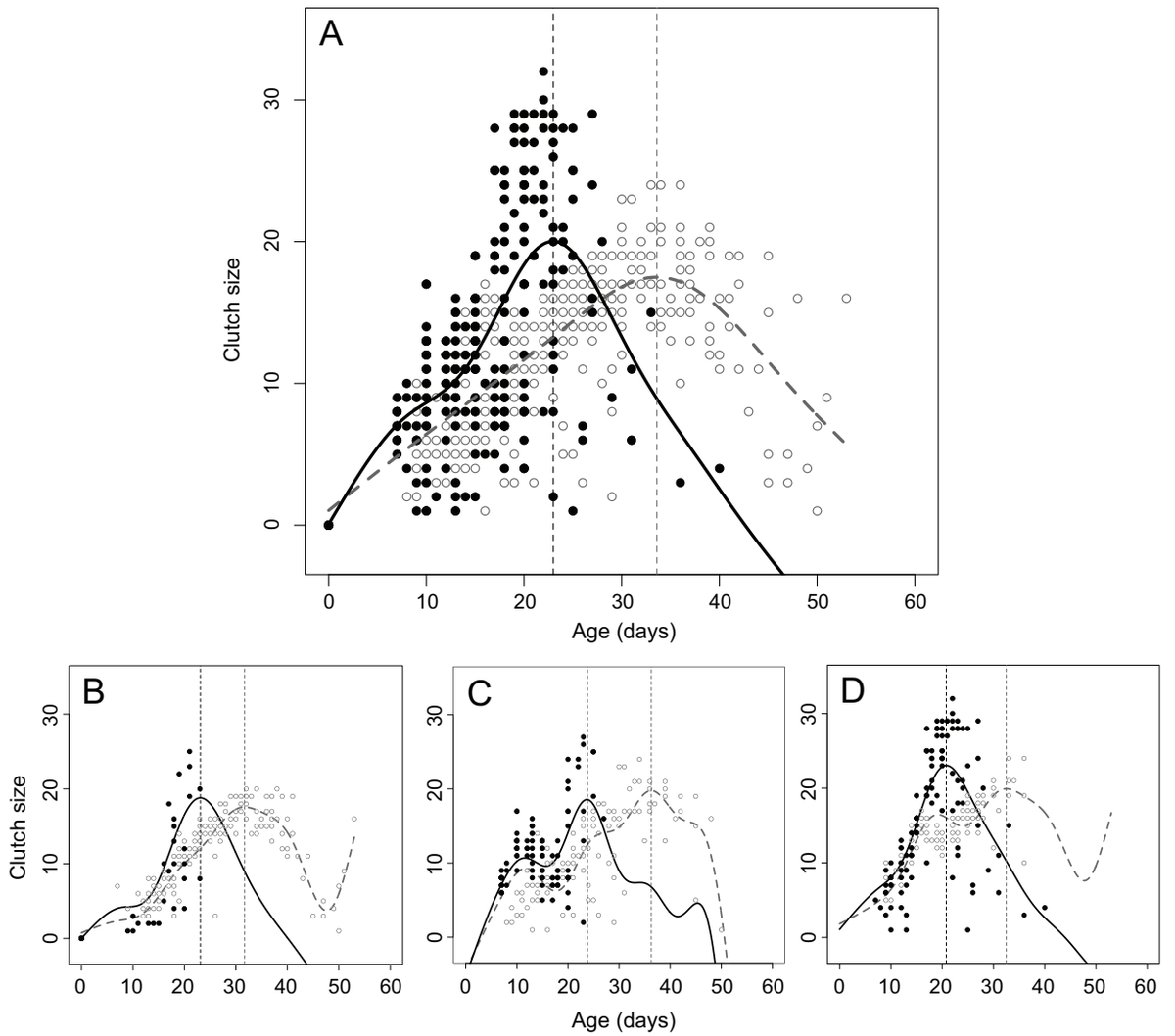


Figure 3: Age-specific reproductive effort of offspring from young (gray dots, dashed lines) and old (black dots, solid lines, and light gray shaded area) maternal lines for all clones combined (*A*), clone Boris (*B*), clone D8.7A (*C*), and clone NBG70 (*D*). The lines represent the reproductive effort predicted by best-fitting generalized additive mixed models (GAMMs; see table 1) for offspring from the old maternal age line (solid lines) and the young maternal age line (dashed lines). The age at the onset of reproductive senescence was predicted as the peak of each fitted GAMM and is marked as a thin dashed line. In all cases, early clutch sizes were larger and the age at the onset of reproductive senescence was earlier for offspring from the old maternal age line. However, there was no difference in the rate of reproductive senescence between maternal age line treatments.

and an increased mortality rate (fig. 4), explaining why offspring from older ancestors typically died sooner and had lower lifetime reproductive success. Such effects could conceivably also have come from clonal selection of de novo mutations. However, we think this is unlikely given that all experiments started from a single individual and the rates of trait divergence across the four generations of maternal age selection (see table A3) were much higher than those expected for divergence on the basis of mutations alone (Lynch et al. 1998). Clonal selection of epimutations or somatic variants is another possibility. But again, this seems unlikely, because there was little differential mortality between treatments during the setup of the experiment (see table A1) and the response to maternal age line selection was broadly comparable in all three of the clones, despite setting up each generation with randomly selected offspring from multiple mothers.

Our findings concur with Lansing's original studies (Lansing 1947, 1948, 1954), which also demonstrated earlier and increased reproduction in offspring from old mater-

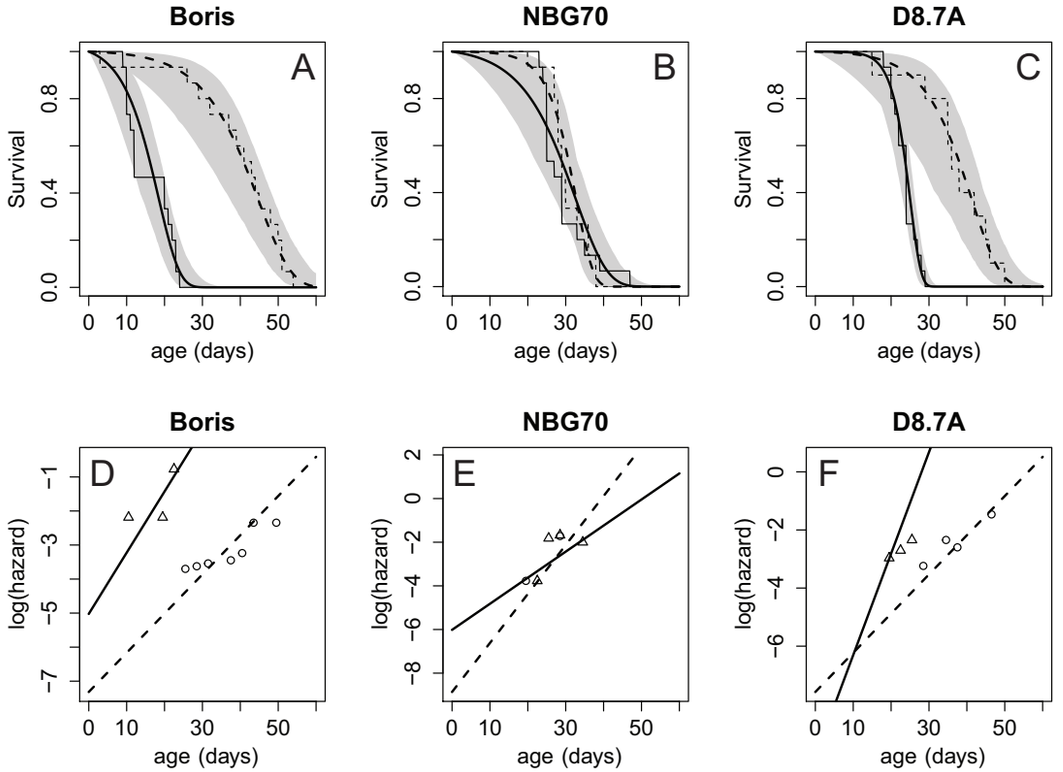


Figure 4: The survival probability (*A*–*C*) and log mortality rate (*D*–*F*) of offspring from the old maternal age line (solid lines) and the young maternal age line (dashed lines) for the three different clones, Boris (*A*, *D*), D8.7A (*B*, *E*), and NBG70 (*C*, *F*). The thick lines are the predictions from a best-fitting parametric survival model with a Gompertz error distribution, with 95% confidence intervals in the predicted survival as gray shaded regions. For comparison purposes, thin lines show Kaplan-Meijer estimates of the survival rate, and triangles (old maternal line) and circles (young maternal line) show the estimated hazard obtained by binning the events in 3-day bins.

nal lines (King 1983). Moreover, Bouwhuis et al. (2010) recently found that, in great tits, offspring hatched from older mothers initially recruited more offspring but then suffered from advanced and increased rates of reproductive senescence. Finally, in *Drosophila*, offspring born from older mothers showed high fertility in early life but reduced fertility in late life (Priest et al. 2008). However, in these previous studies, the reason that offspring from older parents increased early-life reproductive effort or senesced at a faster rate was unclear. The results presented in this study make the link between parental age, offspring development, life history, and rates of aging explicit. In *D. pulex*, the Lansing effect is the result of offspring responses to increased egg provisioning by older mothers. The increased provisioning leads to higher offspring growth rates. In fact, in experiment 2, offspring from old mothers (clutch 11) that were fed low food were able to grow as fast as offspring from young mothers (clutch 1) that were fed high food (fig. 5*A*–5*C*). Higher growth rates have been linked to senescence-related processes such as antioxidant defenses (Blount et al. 2003), telomere dynamics (Hall et al. 2004; Houben et al. 2008; Monaghan and Haussmann 2006), and stress responses (McEwen 2007). We also found that the probabilistic maturation reaction norms (PMRNs) of offspring from the old maternal lines were shifted upward in two of the three clones we studied, meaning that those offspring initiated maturation at larger body sizes (fig. 5*D*, 5*E*). Although it is known that PMRNs in *Daphnia* are plastic and clonally variable (Harney et al. 2013), this is the first study demonstrating that the position of a PMRN can be altered by a parental effect. Maturing at a larger size might increase mortality rates of offspring by facilitating increased early-life reproductive effort and a higher cost of reproduction (reviewed in Roff 1992, 2007; Stearns 1992) or by increasing maintenance costs that scale with body size, such as the cost of molting (Hessen and Alstad Rukke 2000). Interestingly, the clone that showed no maternal age–related upshift in the PMRN of its offspring (see fig. 5*F*) also showed no increase in the age-specific mortality rate of offspring from older mothers (see fig. 4*E*) and no Lansing effect. The variable maternal age effects on offspring in different clones (see fig. 2*A*) mirror genetically variable

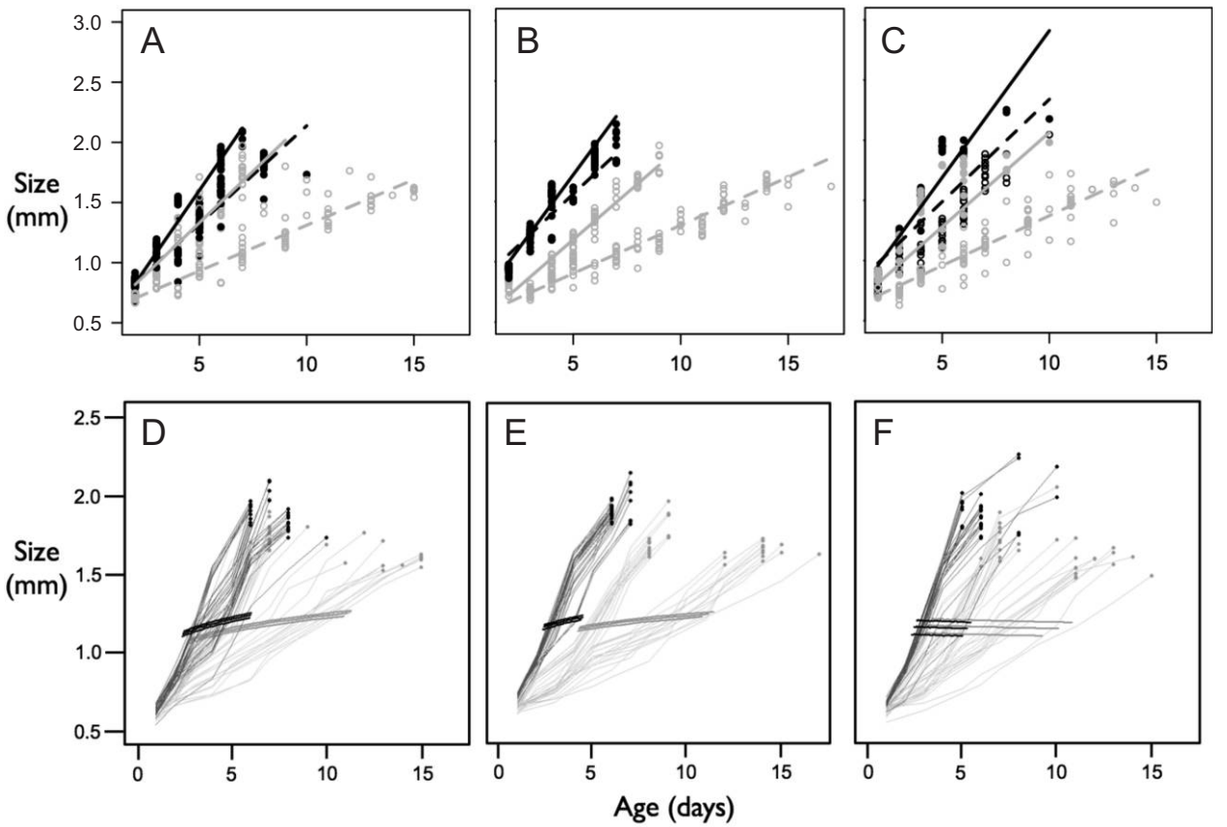


Figure 5: Top panels show the effect of maternal age and food on the growth rates of offspring from clones Boris (*A*), D8.7A (*B*), and NBG70 (*C*). Solid lines and filled points represent the growth rates of offspring fed with high food, whereas dashed lines and open points represent offspring fed with low food. Offspring from the old maternal age line are shown as black lines and points, whereas offspring from the young maternal age lines are show as gray lines and points. The bottom panels show the effect of maternal age on the probabilistic maturation reaction norms of offspring from clones Boris (*D*), D8.7A (*E*), and NBG70 (*F*). Dark gray lines culminating in black circles represent growth and maturity of old (clutch 11) mothers and are intersected by black 25%, 50%, and 75% probabilistic maturation reaction norm (PMRNs) from the best-fitting model. Light gray lines represent growth trajectories of offspring born to young (clutch 1) mothers, culminating in light gray circles at maturity. These growth trajectories are intersected by gray 25%, 50%, and 75% PMRNs.

parental age effects previously observed in *Drosophila* juvenile survival (Kern et al. 2001) and *Drosophila* life span and age-specific mortality rates (Priest et al. 2002). Our results suggest that such variation might arise from genetic differences among mothers in their age-dependent reproductive investment and provisioning of individual offspring (see Plaistow et al. 2007). Alternatively, they could reflect differences in the way that different genotypes respond to the maternal developmental environment. Irrespective of the mechanism, our results suggest that genetic and nongenetic effects determine the life histories of offspring.

The nature of inheritance underpinning trait variation in any population is important, because it may greatly alter the response of the trait to selection (Bonduriansky et al. 2011; Danchin et al. 2011; Hallsson et al. 2012). Nongenetic inheritance is especially likely to influence the evolution of environmentally sensitive traits, because nongenetic inheritance can be considered an environmental component from the perspective of the genotype. Our results support studies of life span extension, genetic intervention, and dietary restriction in other model organisms that have also revealed that senescence is a plastic trait (Flatt and Schmidt 2009; Flatt and Heyland 2011). However, they also suggest that the significance that nongenetic inheritance has on trait evolution is itself genetically variable. Given the potentially significant role that nongenetic inheritance may play in facilitating rapid evolution (Bonduriansky and Day 2009; Bonduriansky et al. 2011; Danchin 2013), this is an important result. Quantifying the extent that nongenetic inheritance differs within and between populations is an important goal for future studies.

Strong parental-age effects on offspring life histories also have important ecological implications. Our results suggest that the offspring from young and old mothers are

demographically very different. Parental-age effects on demography and population dynamics have rarely been studied. However, in the soil mite, *Sancassania berlesei*, experimentally induced maternal age effects altered population dynamics for at least three generations (Benton et al. 2008). In harvested populations, such as in fisheries, in which age structures are often massively truncated (Conover and Munch 2002), maternal-age effects could help to explain why population dynamics are often unstable compared with those of nonharvested populations (Anderson et al. 2008) and why some stocks are slow to recover even when fishing pressure is released (Hutchings 2000; Walsh et al. 2006).

In conclusion, we have demonstrated that, in *D. pulex*, genetic and nongenetic factors shape patterns of offspring aging. Older parents do not just transmit a senescent state to their offspring, as is sometimes assumed. Instead, they produce larger offspring that shift their development in a manner that increases early-life reproductive performance, resulting in advanced and increased rates of senescence that shorten offspring life span. Such an effect may have evolved because *D. pulex* are strongly selected to breed early (they are indeterminate growers), resulting in small young mothers that can produce large clutches only by sacrificing offspring size (Glazier 1992). This constrains the early-life reproductive effort of individual offspring from young mothers, but it maximizes maternal fitness, because the mother produces more offspring (Einum and Fleming 2000; Marshall and Uller 2007). The constraint that young mothers place upon their offspring may then extend offspring life span in a manner similar to that by which dietary restriction has been shown to extend life span in a vast array of different taxa (Masoro 2005; Partridge et al. 2005).

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