The Contributions of Maternal Age Heterogeneity to Variance in Lifetime Reproductive Output

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ABSTRACT: Variance among individuals in fitness components reflects both genuine heterogeneity between individuals and stochasticity in events experienced along the life cycle. Maternal age represents a form of heterogeneity that affects both the mean and the variance of lifetime reproductive output (LRO). Here, we quantify the relative contribution of maternal age heterogeneity to the variance in LRO using individual-level laboratory data on the rotifer *Brachionus manjavacas* to parameterize a multistate age × maternal age matrix model. In *B. manjavacas*, advanced maternal age has large negative effects on offspring survival and fertility. We used multistate Markov chains with rewards to quantify the contributions to variance in LRO of heterogeneity and of the stochasticity inherent in the outcomes of probabilistic transitions and reproductive events. Under laboratory conditions, maternal age heterogeneity contributes 26% of the variance in LRO. The contribution changes when mortality and fertility are reduced to mimic more ecologically relevant environments. Over the parameter space where populations are near stationarity, maternal age heterogeneity contributes an average of 3% of the variance. Thus, the contributions of maternal age heterogeneity and individual stochasticity can be expected to depend strongly on environmental conditions; over most of the parameter space, the variance in LRO is dominated by stochasticity.

Keywords: lifetime reproductive output, maternal age effects, heterogeneity, aging, rotifers.

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Introduction

Individuals within any population differ in fitness components such as longevity and lifetime reproductive output (LRO). Some have long lives; some have short lives. Some leave many offspring; others leave few offspring or none at all (Clutton-Brock 1988; Newton 1989).

One source of this variation is *individual heterogeneity*—differences between individuals in the same life cycle stage that generate differences in vital rates (survival, fertility, growth, etc.) among them. Heterogeneity can be a fixed property, such as genotype, early environment conditions, spatial location (for immobile organisms), sex, or maternal effects. Or it may reflect a dynamic factor, such as physiological or behavioral condition (e.g., breeding vs. nonbreeding), health status, location (for mobile organisms), or environmental state (van Daalen and Caswell 2020). In any particular situation, heterogeneity may be observed or may be estimated as an unobserved latent quality, such as frailty. Individual heterogeneity, however, is not the only source of variation in fitness components. Indeed, individuals with identical vital rates may experience dramatically different life history trajectories.

Diverging trajectories can also arise because life history events are probabilistic. By chance, individuals experiencing the same rates throughout life may differ in the pathways they take through the life cycle and in their success in producing offspring along those pathways. By the end of their lives, even such identical individuals will differ in various fitness components because some individuals were luckier than others. Differences arising from the random outcome of the same probabilities operating on identical

individuals are said to be the result of *individual stochasticity* (Caswell 2009).[1]

In laboratory and field studies, ecologists often find large variance in a given fitness component among individuals (Clutton-Brock 1988). The extent to which this variance is generated by individual heterogeneity or by individual stochasticity is unknown. Here, we describe a new method for partitioning the variance when the fitness component of concern is LRO and the heterogeneity of interest is maternal age. We then apply our method to a laboratory population of genetically identical rotifers (an organism known to exhibit substantial maternal age effects) and find that the relative contributions of heterogeneity and stochasticity depend on environmental context.

We focus on LRO—the number of offspring produced over an individual's life—because it is an important fitness component and is often used as an individual-level life history proxy for fitness (Grafen 1988; Stearns 1992; Roff 2008). The net reproductive rate, R_0, is a measure of the mean LRO when reproduction is measured as daughters per female. R_0 is also the rate of population increase per generation and is therefore indicative of population growth ($R_0 > 1$) or decline ($R_0 < 1$; Lotka 1939; Cushing and Zhou 1994; Heesterbeek 2002). Measures such as the population growth rate λ or r are more accurate measures of fitness because they take timing of reproduction into account (Metz et al. 1992; Roff 2008; Barker 2009). However, because these are population-level measures, they provide no insight into individual-level variability.

Estimates of variance in LRO provide measures of the uncertainty and risk in reproduction in conservation, epidemiological, or other applied settings. The variance in LRO is also indicative of inequality among individuals and, in particular, gives insight into the potential for selection on fitness-related traits (Crow 1958; Brown et al. 2009). Much of quantitative genetics is devoted to one particular partition of variance, between genetic and nongenetic components. We are not concerned with that partition here. Demographic analyses have shown that individual stochasticity creates a substantial amount of variance in LRO in models without heterogeneity for plants, animals, and humans (Tuljapurkar et al. 2009; Steiner et al. 2010; Caswell 2011; Steiner and Tuljapurkar 2012; van Daalen and Caswell 2015, 2017). However, with the exception of examples in van Daalen and Caswell (2020) and in Snyder and Ellner (2018), these studies have not attempted to partition the overall variance into contributions from stochasticity and heterogeneity.

One potential source of heterogeneity is maternal age. Maternal age effects occur when the age of a mother affects the phenotype of her offspring, without any change to the offspring's genotype (Bernardo 1996; Marshall and Uller 2007). We focus on maternal age as a source of heterogeneity in part because maternal age effects are widespread. They have been found in a variety of species, including *Daphnia* (Plaistow et al. 2015), seed beetles (Fox et al. 2003), birds (Bouwhuis et al. 2015), mice (Carnes et al. 2012), elephants (Reichert et al. 2020), humans (Gillespie et al. 2013), and *Drosophila* (Hercus and Hoffman 2000; Kern et al. 2001). In addition, maternal age effects can be complex. While they are often detrimental, with the survival and fertility of offspring declining with advanced maternal age (e.g., Lansing 1947; Hercus and Hoffman 2000; Kern et al. 2001), they are not necessarily so (e.g., Plaistow et al. 2007; Green 2008; Kroeger et al. 2020).

The effects of maternal age on the vigor of rotifers has been an object of study since Lansing (1947) first showed that rotifers born to older mothers tended to have a shorter life span and lower reproduction.[2] In individual-level experiments with high replication, Bock et al. (2019) found that increasing maternal age led to decreased offspring life span and fecundity in the rotifer *Brachionus manjavacas*. This rotifer reproduces clonally; there is little genetic variation among individuals, and the laboratory conditions were kept constant throughout the experiment. Individuals nonetheless differ in their fates along their life cycle (fig. 1), due to maternal age or individual stochasticity.

Maternal age effects can be short term (e.g., effects on neonatal survival sensu Moorad and Nussey 2016) or may persist through the life of the offspring, and they may be expressed as reductions in survival or fertility (e.g., Barks and Laird 2020; Hernández et al. 2020). Whether beneficial or detrimental, short term or long term, or expressed in terms of survival or fertility, heterogeneity due to maternal age is expressed as differences in the age-specific vital rates experienced by individuals over all or some part of their life.

To explore the effects of maternal age heterogeneity requires the recognition that the population is a mixture of individuals of different ages and maternal ages and inclusion of both age and maternal age in a multistate, or age × stage, model (Caswell et al. 2018). In previous work, we developed a population-level model for *B. manjavacas* and used it to calculate measures of population performance as well as selection gradients on the evolution of maternal effect senescence (Hernández et al. 2020). In this study, to analyze LRO and partition the variance therein,

1. What we call "individual stochasticity" has been referred to as "dynamic heterogeneity" by Tuljapurkar, Steiner, and collaborators (Tuljapurkar et al. 2009; Steiner et al. 2010; Steiner and Tuljapurkar 2012). More recently, Snyder and Ellner (2018) have used the term "luck" to describe such stochastic variation in fitness components.

2. This is an example of maternal effect senescence, a type of maternal age effect in which offspring quality declines with increasing age of the mother (Moorad and Nussey 2016).

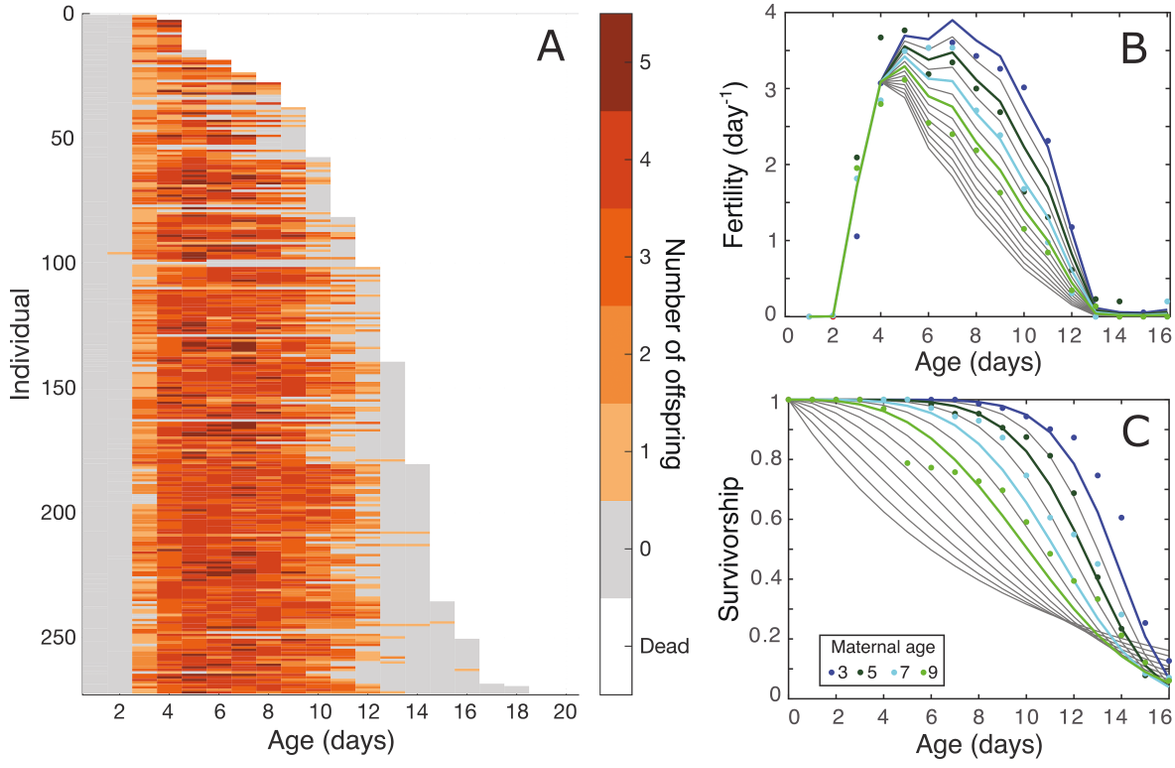

Figure 1: *A*, Event history diagram for 272 individual rotifers. *B*, Age-specific fertility schedules for each maternal age group according to the Coale-Trussell model. *C*, Survivorship curves for each maternal age group according to the Weibull model. Solid circles represent observed data. Colored lines represent model fits to the data, and gray lines represent interpolated model fits. *B* and *C* were obtained from Hernández et al. (2020).

we modify the population model to obtain an individual-level multistate Markov chain with rewards.

The relative contributions of maternal age heterogeneity and individual stochasticity to variance in LRO—and how these contributions depend on the environment—are unknown. The environment can potentially enhance or obscure the effects of heterogeneity. Studies of birds (Barbraud and Weimerskirch 2005; Jenouvrier et al. 2018) and Soay sheep (Tavecchia et al. 2005) have found that various individual differences are expressed to a lesser or greater extent depending on the environmental conditions. The rotifers in our experiment were provided abundant food, constant temperature, and protection from predators and pathogens. We use our model to define scenarios corresponding to more natural, or at least ecologically reasonable, environments and to determine the consequences of such environments for LRO and its variance.

The goals of this study are to (1) present a general, individual-level model for incorporating maternal age effects; (2) analyze the relative contributions of individual stochasticity and maternal age heterogeneity to the variance in LRO; and (3) understand how those contributions change under different environmental scenarios.

The multistate age × maternal age Markov chain with rewards is presented, step by step, in the next section. We show the calculations needed to obtain mean and variance in LRO and the variance components due to individual stochasticity and maternal age heterogeneity. The following section describes the rotifer life history and experimental system. We then present the results and conclude with a discussion.

Demographic Analysis

Our goal is to determine the contributions of an important source of heterogeneity to variance in LRO and to do so as a function of environments expressed through the vital rates. Accomplishing this requires a methodical development of demographic analyses from the individual, through the life cycle, to lifetime reproduction, and it requires a careful consideration of both deterministic and stochastic processes. In this section, we lay these steps out carefully, to provide the ground for our analyses and to provide a path that can be followed by anyone else wanting to carry out such an analysis.

A note on notation. Matrices are denoted by uppercase boldface letters (e.g., $\mathbf{P}$), and vectors are denoted by lowercase boldface letters (e.g., $\mathbf{x}$). Vectors are column vectors by default; $\mathbf{x}^\mathsf{T}$ is the transpose of $\mathbf{x}$. The vector $\mathbf{1}_n$ is an $n \times 1$ vector of ones, $\mathbf{I}_n$ is the identity matrix of order n, and $\mathbf{e}_i$ is a unit vector with a 1 in the ith entry and zeros elsewhere. The diagonal matrix with the vector $\mathbf{x}$ on the diagonal and zeros elsewhere is denoted $\mathcal{D}(\mathbf{x})$. The symbol $\circ$ denotes the Hadamard, or element-by-element product, and $\otimes$ denotes the Kronecker product. In the multistate model, vectors and matrices have a block structure that reflects the arrangement of age classes and maternal age groups. A tilde is used to indicate such vectors (e.g., $\tilde{\mathbf{n}}$) and matrices (e.g., $\tilde{\mathbf{U}}$). When it seems helpful, we will indicate the dimension of matrices and vectors when they are referenced.

The variance among individuals in LRO (or in any lifetime fitness component) depends on the life cycle pathways open to an individual and their probabilities. The pathways in turn depend on probabilities of survival and stage transition and on the probability distribution of reproduction at each stage. Incorporating estimates of those probabilities into a properly structured demographic model makes it possible to calculate the variance arising from individual stochasticity and from heterogeneity. In this section, we present the model for the study of maternal age effects, in the form of a multistate Markov chain with rewards.

In an age- or stage-classified model, every individual experiences the same rates at the same (st)age. The variance among individuals is thus due to individual stochasticity. In a multistate model combining (st)ages and "groups" (a second dimension of classification), the rates at any (st) age depend on group membership but are identical for all individuals within groups. The variance among individuals is, in such a model, due to individual stochasticity within groups and heterogeneity among groups.

Demography with Maternal Age Effects: A Multistate Matrix Model

Our demographic analysis requires a model in which individuals are jointly classified by their age and their maternal age (i.e., the age of their mother at the time of their birth). Matrix models with multiple dimensions are referred to as multistate or age × stage models (e.g., Goodman 1969; Land and Rogers 1982; Caswell 2012; for a recent synthesis, see Caswell et al. 2018). The analytical methods for matrix population models apply directly to such multistate models.

A multistate model for maternal age effects was introduced by Hernández et al. (2020). A model with similar structure was independently derived by Barks and Laird (2020). We give a brief overview of model construction here; the full details are given in Hernández et al. (2020).

The model is constructed using the vec-permutation approach (see Caswell et al. 2018), which is based on sets of matrices that contain age-specific survival probabilities and fertilities for each maternal age class and specify the assignment of offspring into maternal age groups. The result is a block-structured population vector:

$$\tilde{\mathbf{n}} = \begin{pmatrix} n_{11} \\ \vdots \\ n_{g1} \\ \hline \vdots \\ n_{1\omega} \\ \hline \vdots \\ n_{g\omega} \end{pmatrix}. \tag{1}$$

Individuals are classified by age ($x = 1, \ldots, \omega$) and maternal age groups ($\gamma = 1, \ldots, g$). The matrix $\tilde{\mathbf{A}}$ that projects this population forward in time has a block-Leslie structure:

$$\tilde{\mathbf{A}} = \left(\begin{array}{c|c|c|c|c} \mathbf{F}_1 & \mathbf{F}_2 & \cdots & \mathbf{F}_{\omega-1} & \mathbf{F}_\omega \\ \hline \mathbf{U}_1 & \mathbf{0} & \cdots & \mathbf{0} & \mathbf{0} \\ \hline \mathbf{0} & \mathbf{U}_2 & \cdots & \mathbf{0} & \mathbf{0} \\ \hline \vdots & \vdots & \ddots & \vdots & \vdots \\ \hline \mathbf{0} & \mathbf{0} & \cdots & \mathbf{U}_{\omega-1} & [\mathbf{U}_\omega] \end{array}\right) \tag{2}$$

$$= \tilde{\mathbf{U}} + \tilde{\mathbf{F}}. \tag{3}$$

If the matrix $[\mathbf{U}_\omega]$ is included, the final age class includes individuals of all ages ω or older. The matrix $\tilde{\mathbf{A}}$ is made up of a matrix $\tilde{\mathbf{U}}$ containing transition probabilities for extant individuals and a matrix $\tilde{\mathbf{F}}$ containing fertilities:

$$\tilde{\mathbf{U}} = \left(\begin{array}{c|c|c|c|c} \mathbf{0} & \mathbf{0} & \ldots & \mathbf{0} & \mathbf{0} \\ \hline \mathbf{U}_1 & \mathbf{0} & \ldots & \mathbf{0} & \mathbf{0} \\ \hline \mathbf{0} & \mathbf{U}_2 & \ldots & \mathbf{0} & \mathbf{0} \\ \hline \vdots & \vdots & \ddots & \vdots & \vdots \\ \hline \mathbf{0} & \mathbf{0} & \ldots & \mathbf{U}_{\omega-1} & [\mathbf{U}_\omega] \end{array}\right),$$

$$\tilde{\mathbf{F}} = \left(\begin{array}{c|c|c|c|c} \mathbf{F}_1 & \mathbf{F}_2 & \ldots & \mathbf{F}_{\omega-1} & \mathbf{F}_\omega \\ \hline \mathbf{0} & \mathbf{0} & \ldots & \mathbf{0} & \mathbf{0} \\ \hline \mathbf{0} & \mathbf{0} & \ldots & \mathbf{0} & \mathbf{0} \\ \hline \vdots & \vdots & \ddots & \vdots & \vdots \\ \hline \mathbf{0} & \mathbf{0} & \cdots & \mathbf{0} & \mathbf{0} \end{array}\right). \tag{4}$$

For each age class x, the $g \times g$ transition matrix $\mathbf{U}_x$ is

$$\mathbf{U}_x = \begin{pmatrix} p_{1,x} & 0 & 0 & \ldots & 0 \\ 0 & p_{2,x} & 0 & \ldots & 0 \\ 0 & 0 & p_{3,x} & \ldots & 0 \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 & \ldots & p_{g,x} \end{pmatrix} \quad x = 1, \ldots, \omega, \tag{5}$$

where $p_{i,x}$ is the probability of surviving from age x to age $x + 1$ for an individual with maternal age i. For each age class x, the $g \times g$ fertility matrix $\mathbf{F}_x$ contains fertilities specific to each age and maternal age combination and places the new offspring in the appropriate maternal age group. For example, if $g = \omega = 4$, then

$$\begin{aligned}
\mathbf{F}_1 &= \begin{pmatrix} f_{1,1} & f_{2,1} & f_{3,1} & f_{g,1} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \\
\mathbf{F}_2 &= \begin{pmatrix} 0 & 0 & 0 & 0 \\ f_{1,2} & f_{2,2} & f_{3,2} & f_{g,2} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \\
\mathbf{F}_3 &= \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ f_{1,3} & f_{2,3} & f_{3,3} & f_{g,3} \\ 0 & 0 & 0 & 0 \end{pmatrix}, \\
\mathbf{F}_4 &= \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ f_{1,4} & f_{2,4} & f_{3,4} & f_{g,4} \end{pmatrix},
\end{aligned} \tag{6}$$

where $f_{i,x}$ is the fertility of an individual of age x in maternal age group i.

The matrix $\tilde{\mathbf{A}}$ projects the age-stage structure of the population. The dominant eigenvalue λ of $\tilde{\mathbf{A}}$ gives the asymptotic population growth rate, and the derivative of λ to the entries of $\tilde{\mathbf{A}}$ is the selection gradient on the corresponding life history trait. Hernández et al. (2020) used this fact to analyze selection gradients on maternal effect senescence. They found that selection gradients on survival and fertility eventually decrease with maternal age, which shows that maternal effect senescence can evolve as a result of antagonistic pleiotropy or mutation accumulation, in much the same way as the familiar age patterns of senescence (Hamilton 1966).

The Individual Life Cycle with Maternal Age Effects: A Multistate Markov Chain

Our goal here is to determine not the growth rate of the population but rather the reproductive output over the lifetime of an individual. To that end, we use a multistate absorbing Markov chain to describe the life cycle.

The matrix $\tilde{\mathbf{U}}$ in equation (5) is the transient portion of an absorbing Markov chain. Its entries are the probabilities of transitions and survival of individuals of any maternal age group moving through their life cycle (e.g., Caswell 2001, 2009, 2014; Tuljapurkar and Horvitz 2006; Horvitz and Tuljapurkar 2008; van Daalen and Caswell 2017). Death is an absorbing state. The transition matrix for this Markov chain can be written

$$\tilde{\mathbf{P}} = \left(\begin{array}{c|c} \tilde{\mathbf{U}} & \mathbf{0} \\ \hline \tilde{\mathbf{d}}^{\mathsf{T}} & 1 \end{array}\right) \quad (g\omega + 1) \times (g\omega + 1). \tag{7}$$

Here, $\tilde{\mathbf{d}}$ is a vector of age × stage–specific probabilities of death.

The fundamental matrix of the chain plays an important role in what follows; it is given by

$$\tilde{\mathbf{N}} = (\mathbf{I}_{g\omega} - \tilde{\mathbf{U}})^{-1}. \tag{8}$$

The (i, j) entry of $\tilde{\mathbf{N}}$ gives the mean time spent in state i, prior to absorption, by an individual in state j, where i and j here range over all of the age-stage combinations in the state space.

Reproduction over the Individual Life Cycle: A Markov Chain with Rewards

The Markov chain with transition matrix $\tilde{\mathbf{P}}$ generates the possible pathways that an individual can take, starting at any age × stage combination (we start at birth) and continuing until eventual absorbtion (i.e., death; e.g., Caswell 2001, 2009; Tuljapurkar and Horvitz 2006; Horvitz and Tuljapurkar 2008). However, we are interested not only in the life trajectories but also in the reproductive output accumulated by an individual over that trajectory. Analyses of reproductive output are most realistic when individual stochasticity is represented in both survival and reproduction, as is the case in Markov chain with rewards models (Caswell 2011; van Daalen and Caswell 2017).

Individuals produce offspring, at every age and in every stage, at rates that are specific to that age-stage combination. This reproduction is treated as a "reward" that is accumulated by the individual until death. The accumulated reward is the LRO, and its mean, variance, and other statistical properties are calculated as we describe below (Caswell 2011; van Daalen and Caswell 2015, 2017, 2020, forthcoming). It is important to note that the reproductive reward at each step of the life cycle is itself a random variable, the expected value of which is given by the entries of $\tilde{\mathbf{F}}$. The calculation of the statistics of LRO using Markov chains with rewards is the only approach that accounts for both of these kinds of variation (Caswell 2011).

Let r_{ij} denote the reward associated with the transition from state j to state i, where i and j range over the entire age × stage state space. In our case, r_{ij} is the reproductive output associated with the transition from state j to state i. Because r_{ij} is a random variable, we define a set of matrices that contain the moments of the r_{ij}. The kth moments are the entries of a matrix $\tilde{\mathbf{R}}_k$,

$$\tilde{\mathbf{R}}_k = \left(E[r_{ij}^k] \right). \tag{9}$$

The reward matrices inherit the same maternal-age-within-age structure as the other vectors and matrices. The kth moment reward matrix then becomes

$$\tilde{\mathbf{R}}_k = \left(\begin{array}{c|c|c|c} \mathbf{R}_k(1) & \dots & \mathbf{R}_k(\omega) & \mathbf{0}_{g\times 1} \\ \hline \vdots & \ddots & \vdots & \vdots \\ \hline \mathbf{R}_k(1) & \dots & \mathbf{R}_k(\omega) & \mathbf{0}_{g\times 1} \\ \hline \mathbf{r}_k^{\mathsf{T}}(1) & \dots & \mathbf{r}_k^{\mathsf{T}}(\omega) & 0 \end{array}\right) \quad (g\omega + 1) \times (g\omega + 1). \tag{10}$$

The zero matrices in the last block column indicate that dead individuals cannot accumulate any more reproduction. The age-specific matrices $\mathbf{R}_k(x)$ contain the kth moments of reproductive rewards in each maternal age group at age x for individuals that remain in the transient (living) stages. The vectors $\mathbf{r}_k^{\mathsf{T}}(x)$ contain the kth moments of the rewards for individuals that move to the absorbing (dead) state during a time interval.

In demographic models, fertility typically depends on the current state of the individual, not on the state to which it moves. In our case, the rewards are given directly by the fertility rates. The $g \times g$ matrices $\mathbf{R}_k(x)$ are

$$\mathbf{R}_k(x) = E\begin{pmatrix} f_{1,x}^k & \dots & f_{g,x}^k \\ \vdots & \ddots & \vdots \\ f_{1,x}^k & \dots & f_{g,x}^k \end{pmatrix} \quad x = 1, \dots, \omega. \tag{11}$$

The $1 \times g$ vectors $\mathbf{r}_k^{\mathsf{T}}(x)$ are

$$\mathbf{r}_k^{\mathsf{T}}(x) = E(f_{1,x}^k \quad \dots \quad f_{g,x}^k) \quad x = 1, \dots, \omega. \tag{12}$$

The moment matrices $\mathbf{R}_k$ depend on the moments of age × stage–specific fertility. These moments can be calculated from published demographic models by assuming a parametric form for the distribution of fertility (Caswell 2011; van Daalen and Caswell 2017). However, in our case we are able to calculate the moments directly from the raw fertility data, thus obtaining an "empirical distribution" (see "Parameterization").

The Statistics of LRO

Given the multistate Markov chain $\tilde{\mathbf{P}}$ and the reward matrices $\tilde{\mathbf{R}}_k$, we compute the moments of LRO for individuals starting in every age-maternal age state. To calculate the mean and variance of LRO, we require its first two moments, which are given by the vectors

$$\tilde{\boldsymbol{\rho}}_1 = \tilde{\mathbf{N}}^{\mathsf{T}}\mathbf{Z}(\tilde{\mathbf{P}} \circ \tilde{\mathbf{R}}_1)^{\mathsf{T}}\mathbf{1}_{g\omega+1} \quad g\omega \times 1, \tag{13}$$

$$\tilde{\boldsymbol{\rho}}_2 = \tilde{\mathbf{N}}^{\mathsf{T}}[\mathbf{Z}(\tilde{\mathbf{P}} \circ \tilde{\mathbf{R}}_2)^{\mathsf{T}}\mathbf{1}_{g\omega+1} + 2(\tilde{\mathbf{U}} \circ \hat{\mathbf{R}}_1)^{\mathsf{T}}\tilde{\boldsymbol{\rho}}_1] \quad g\omega \times 1, \tag{14}$$

where $\hat{\mathbf{R}}_1$ is the reward matrix $\tilde{\mathbf{R}}_1$ with the absorbing state cleaved off according to

$$\hat{\mathbf{R}}_1 = \mathbf{Z}\tilde{\mathbf{R}}_1\mathbf{Z}^{\mathsf{T}} \tag{15}$$

and $\mathbf{Z} = (\mathbf{I}_{g\omega}|\mathbf{0}_{g\omega\times 1})$. See van Daalen and Caswell (2017, theorem 1) for the derivation of these formulas and extension to all moments.

The vector of the variances in LRO is

$$V(\tilde{\boldsymbol{\rho}}) = \tilde{\boldsymbol{\rho}}_2 - \tilde{\boldsymbol{\rho}}_1 \circ \tilde{\boldsymbol{\rho}}_1 \quad g\omega \times 1. \tag{16}$$

The vectors $\tilde{\boldsymbol{\rho}}_1$, $\tilde{\boldsymbol{\rho}}_2$, and $V(\tilde{\boldsymbol{\rho}})$ inherit the block structure of $\tilde{\mathbf{n}}$ in equation (1).

Cohorts, Groups, and the Mixing Distribution

The entries of $\tilde{\boldsymbol{\rho}}_1$ and $V(\tilde{\boldsymbol{\rho}})$ contain the means and variances of LRO for individuals starting in every combination of age and maternal age group. We are interested in the lifetime reproduction of a set of individuals starting at birth (i.e., the first age class) and distributed among maternal age groups according to some distribution. Thus, we extract vectors containing the means and variances of LRO for each maternal age group,

$$\mathbf{m} = (\mathbf{e}_1^{\mathsf{T}} \otimes \mathbf{I}_g)\tilde{\boldsymbol{\rho}}_1 \quad g \times 1, \tag{17}$$

$$\mathbf{v} = (\mathbf{e}_1^{\mathsf{T}} \otimes \mathbf{I}_g)V(\tilde{\boldsymbol{\rho}}) \quad g \times 1. \tag{18}$$

Here, $\mathbf{e}_1$ is an $\omega \times 1$ vector with a 1 in the first entry. The ith entry of $\mathbf{m}$ is the mean LRO for an individual in group i, similarly for $\mathbf{v}$.

The variance in LRO among a set of newborn individuals is that of a heterogeneous cohort consisting of a mixture of individuals among maternal age groups. A probability vector $\boldsymbol{\pi}$, of dimension $g \times 1$, gives the fraction of the cohort in each maternal age group. This vector is called the mixing distribution. As a biologically reasonable mixing distribution, we use the distribution among groups of newborn individuals in the stable population implied by $\tilde{\mathbf{A}}$.

The stable structure is the normalized right eigenvector $\tilde{\mathbf{w}}$ corresponding to the dominant eigenvalue of $\tilde{\mathbf{A}}$. The reproductive output of the stable population, normalized to sum to 1, provides a cohort of newborns:

$$\tilde{\boldsymbol{\pi}} = \frac{\tilde{\mathbf{F}}\tilde{\mathbf{w}}}{\mathbf{1}_{g\omega}^{\mathsf{T}}\tilde{\mathbf{F}}\tilde{\mathbf{w}}} \quad g\omega \times 1. \tag{19}$$

The vector $\tilde{\boldsymbol{\pi}}$ inherits the block structure of $\tilde{\mathbf{n}}$ in equation (1). The mixing distribution among groups at birth is then extracted as

$$\boldsymbol{\pi} = (\mathbf{e}_1 \otimes \mathbf{I}_g)\tilde{\boldsymbol{\pi}} \quad g \times 1. \tag{20}$$

(Note the lack of tilde on $\boldsymbol{\pi}$, indicating that it is not block structured.)[3]

Variance Partitioning

We are now prepared to calculate and partition the variance in LRO among the set of newborn individuals distributed among maternal age groups according to the mixing distribution $\boldsymbol{\pi}$. The mean LRO for this group is

$$\begin{aligned} E(\text{LRO}) &= E_{\boldsymbol{\pi}}[E(\text{LRO} \mid \text{maternal age group})] \\ &= \boldsymbol{\pi}^{\mathsf{T}}\mathbf{m}. \end{aligned} \tag{21}$$

The variance in LRO among the individuals in this set has two components: the variance within and the variance between maternal age groups. The within-group component of the variance is the mean of the variances within maternal age groups, weighted by $\boldsymbol{\pi}$. The between-group component is the variance of the means of maternal age groups, again weighted by $\boldsymbol{\pi}$ (e.g., Rényi 1970, p. 275, theorem 1):

$$\begin{aligned} V(\text{LRO}) &= E_{\boldsymbol{\pi}}[V(\text{LRO})] + V_{\boldsymbol{\pi}}[E(\text{LRO})] \\ &= \underbrace{V_{\text{w}}}_{\text{within}} + \underbrace{V_{\text{b}}}_{\text{between}}. \end{aligned} \tag{22}$$

The expressions for the within-group and between-group variance components are

$$V_{\text{w}} = \boldsymbol{\pi}^{\mathsf{T}}\mathbf{v}, \tag{23}$$

$$V_{\text{b}} = \boldsymbol{\pi}^{\mathsf{T}}(\mathbf{m} \circ \mathbf{m}) - (\boldsymbol{\pi}^{\mathsf{T}}\mathbf{m})^2, \tag{24}$$

where $\mathbf{m}$ and $\mathbf{v}$ are given by equations (17) and (18).

The within-group variance V_{w} is the result of the stochastic outcome of survival and reproduction among individuals experiencing identical rates. The between-group variance V_{b} is the result of the differences in the rates experienced by individuals in different maternal ages.

The fraction of the total variance due to heterogeneity can be calculated as

$$\mathcal{K} = \frac{V_{\text{b}}}{V_{\text{b}} + V_{\text{w}}}. \tag{25}$$

This fraction is known as the intraclass correlation coefficient in quantitative genetics (Falconer 1960). If there is no contribution to the variance in ρ from the maternal age groups, $\mathcal{K} = 0$. If there is no stochasticity, and the variance in LRO is a deterministic function of the maternal age groups, $\mathcal{K} = 1$, and all variance will be due to heterogeneity.

3. It is possible to calculate the *remaining* lifetime reproduction of a set of individuals starting at any age x; see sec. S2 of the supplemental PDF.

Maternal Age Heterogeneity in Rotifers

The Rotifer Model System

Rotifers of the genus *Brachionus* are microscopic, invertebrate animals with a life span of about 2 weeks and simple laboratory culture. They are cyclical parthenogens, alternating between asexual and sexual reproduction, depending on environmental conditions. Individuals in age-synchronized cohorts of *B. manjavacas* are easy to collect and monitor. These properties have made *B. manjavacas* a valuable model species for the investigation of maternal effects in laboratory environments (Gribble and Snell 2018). Asexually reproducing females produce 25–30 offspring sequentially during their 10-day reproductive period in the laboratory. Maternal investment is high; egg size is about one-third the size of the mother. There is no post-hatching parental care, which rules out parental care as a potential cause of maternal effects. The laboratory population used in this study is fully clonal and hence shows no genetic variation.

Parameterization

Our analysis is based on data from a laboratory study in which equal-sized cohorts of newborn rotifers in each of several maternal age groups (3, 5, 7, and 9 days) were individually maintained under constant food and temperature conditions (Bock et al. 2019). Figure 1*A* is an event history diagram (Carey et al. 1998) showing the survival and reproduction of each individual. Despite the uniformity of the laboratory conditions and the genetic uniformity of the organisms, considerable variation in individual fates remains. Given the lack of genetic diversity, the only known sources of this variation are individual stochasticity and maternal age. By grouping individuals by their maternal age, Bock et al. (2019) obtained age × maternal age–specific survival and fertility schedules.

In our previous study, we used these data to fit an age × maternal age matrix model; for details, see Hernández et al. (2020). We fit a Weibull model for age at death and the Coale-Trussell model for fertility (Coale and Trussell 1974) using nonlinear least squares. Both models were adapted to incorporate a maternal age parameter, and subsequently a best-fit age × maternal age fertility and survival schedule was obtained (for details, see sec. S1 of the supplemental PDF, available online, or Hernández et al. 2020). The Weibull and Coale-Trussell parameters were interpolated to obtain age-specific fertility and mortality schedules for all maternal age groups (fig. 1*B*, 1*C*).

The reward matrices in equation (11) require the first and second moments of reproductive output as a function of age and maternal age. We obtained these by fitting

a Coale-Trussell model separately to each of the moments of the raw fertility data.

Environmental Scenarios

Our data were obtained under laboratory conditions of high resource levels and no predation, resulting in levels of survival, fertility, and population growth rate that are not representative of natural ecological conditions. The laboratory conditions led to a population growth rate of $\lambda \approx 2$ per day (Hernández et al. 2020). To see how unrealistic this is, simply note that under these conditions a population of *B. manjavacas* would increase by a factor of 1 billion in just under 1 month. The results of calculations under such conditions have limited ecological relevance, applying at best only on very short, transient timescales.

In a more natural situation, survival would be reduced by predation, parasitism, and abiotic factors, and fertility would be reduced by resource limitation; over the long term, the population growth rate would be close to stationary (i.e., $\lambda = 1$). Rotifers are subject to predation by a variety of invertebrate (protozoa, cnidaria, copepods, aquatic insects) and vertebrate (fish larvae) predators (Williamson 1983; Nagata et al. 2005). Predation is common enough that rotifers have been called an important link in the freshwater food web (Williamson and Butler 1986). Food limitation reduces the amount of reproduction but leaves the shape and location of the distribution of egg production relatively unchanged (King 1967).

As in our previous analysis (Hernández et al. 2020), we modified the demographic parameters to create plausible and ecologically relevant life history scenarios. A reduction in fertility that reduces λ to 1 is obtained by multiplying the fertility matrix by $1/R_0$, leaving survival unchanged. The reward matrices for this low-fertility scenario were created by dividing $\tilde{\mathbf{R}}_1$ by the net reproductive rate R_0 and by dividing $\tilde{\mathbf{R}}_2$ by R_0^2. We reduced survival by imposing a fixed additive mortality hazard to all age and maternal age classes. We found numerically that an additive hazard that was equivalent to multiplying the $\mathbf{U}_x$ matrices by a factor $c = 0.3833$ was sufficient to reduce λ to 1. In both of these cases, we chose to modify the amount of reproduction or the level of survival, keeping the shape of the fertility or survival function fixed.

We sampled combinations of 100 values of fertility reduction between 1 and $1/R_0$ and 100 values of survival reduction between $c = 1$ and $c = 0.3833$. For each of the resulting 10,000 sets of demographic parameters, we created $\tilde{\mathbf{U}}$, $\tilde{\mathbf{F}}$, $\tilde{\mathbf{R}}_1$, and $\tilde{\mathbf{R}}_2$ and calculated the within-group and between-group variances in LRO and the fraction $\mathcal{K}$ of the variance in LRO attributable to heterogeneity. All analyses were performed in Matlab R2017B (code is available via the Dryad Digital Repository; https://doi.org/10.5061/dryad.vmcvdnctb; van Daalen 2022).

Results: Variance and Variance Partitioning

The mean and variance of LRO for each maternal age group, under laboratory conditions, are shown in figure 2. These values are the entries in the vectors $\mathbf{m}$ and $\mathbf{v}$ in equations (17) and (18). Between the earliest and the latest maternal age groups, mean LRO declines threefold, while the variance in LRO (due to individual stochasticity in each group) more than doubles. These large changes are demographic evidence, reinforcing the survival and fertility differentials in figure 1, of the magnitude of the effects of maternal age in this species.

The population mean and variance in LRO depends on the group-specific values in figure 2 and the mixing distribution. The mixing distribution is the composition of the newborns produced by a population with the stable age–by–maternal age structure (as in eq. [20]). In the

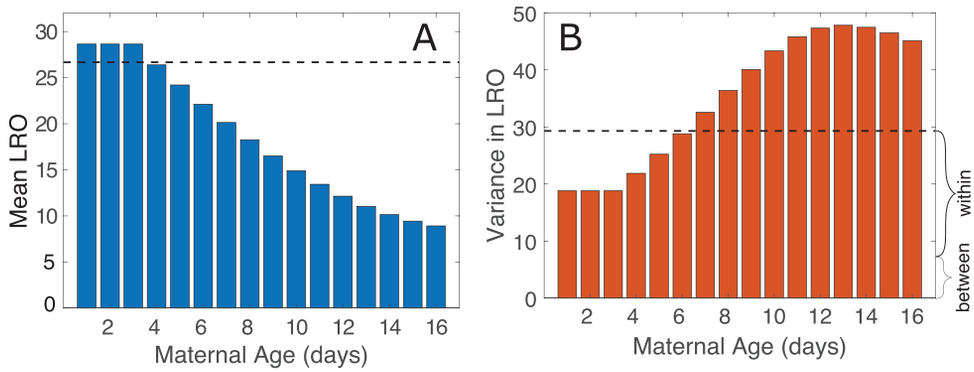

Figure 2: Mean and variance in lifetime reproductive output (LRO) for each maternal age group. The dashed black lines represent the population mean LRO in *A* and the total variance in LRO in the population in *B*. About 26% of the total variance in LRO is variance between groups due to maternal age heterogeneity; the other 74% is variance within groups due to individual stochasticity.

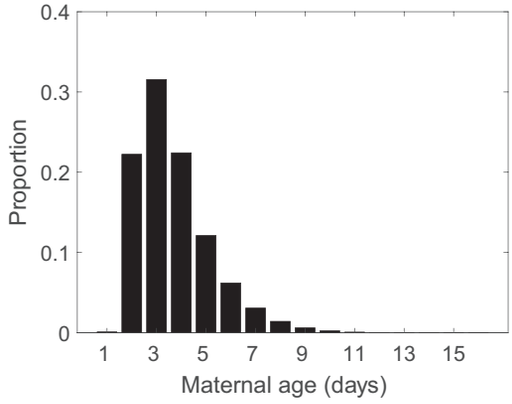

Figure 3: Mixing distribution $\boldsymbol{\pi}$ estimated under laboratory conditions. This is the maternal age group composition of a cohort of newborns in a population with the stable age × maternal age structure implied by the estimated vital rates. Although other choices for the mixing distribution could be made, it is reasonable to connect the mixing distribution to the population composition through the model.

laboratory environment, 75% of newborns were born to mothers aged 2, 3, or 4 days, with an increasingly smaller proportion born to older mothers (fig. 3). The mixing distribution, being a function of the stable stage structure and the fertility matrix, must be recalculated for each environmental scenario (see our note in sec. S3 of the supplemental PDF).

Together, the means, the variances, and the mixing distribution yield the population mean and variance of LRO, calculated following equations (21) and (22). The results, under laboratory conditions, are shown by the horizontal dashed lines in figure 2; under laboratory conditions, $E(\mathrm{LRO}) = 26.7$ and $V(\mathrm{LRO}) = 29.3$.

When the total variance in LRO is partitioned into the variance within groups (V_w) and the variance between groups (V_b), as calculated from equations (23) and (24), we find that under laboratory conditions, $\mathcal{K} = 0.26$. That is, 26% of the variance in LRO is due to heterogeneity in maternal age. The remaining 74% is due to individual stochasticity within maternal age groups.

Variance Components and Contributions under Ecological Scenarios

As discussed in "Environmental Scenarios," the laboratory conditions that produce the results in figure 2 are ecologically unrealistic. We are interested in results under more ecologically relevant scenarios. For each of the 10,000 parameter sets defined by reductions in fertility (as might be produced by food limitation) and survival (as might be produced by predation), we computed the group-specific means and variances, the mixing distribution, and within-group and between-group components of the variance in LRO.

The resulting contributions of maternal age heterogeneity and stochasticity are shown in figure 4. Reducing survival clearly reduces the contribution due to heterogeneity regardless of the value of fertility. Only at values of survival close to that in the benign laboratory environment does the contribution of heterogeneity to the variance increase with a decreasing fertility level.

The most ecologically relevant parameters are those corresponding to reasonable rates of population growth. The contours in figure 4 show parameters yielding stationary populations with $\lambda = 1$ and the region where $0.95 \leq \lambda \leq 1.05$, that is, the region corresponding to a population growing or shrinking by 5% per day. In this region of parameter space, the results differ dramatically from those under laboratory conditions. Over about 70% of this region, heterogeneity contributes less than 1% of the variance in LRO; over 85% of this range, it contributes less than 5%. Along the line representing $\lambda = 1$, heterogeneity contributes, on average, 3% of the variance. The sole exception is the bottom right corner of figure 4, corresponding to a population with extremely high survival and severely reduced fertility. There, $\mathcal{K}$ reaches values as high as 0.41 (i.e., 41% of the variance contributed by

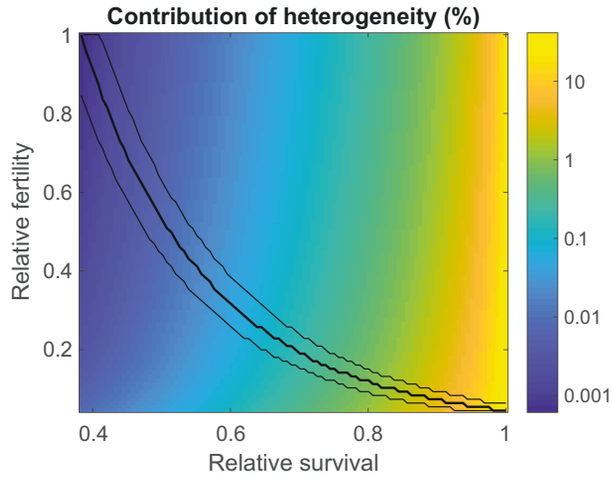

Figure 4: Contribution (%) of maternal age heterogeneity to the variance in lifetime reproductive output at birth for a range of virtual environments. The axes represent the factors with which the survival matrix $\tilde{\mathbf{U}}$ and the fertility and reward matrices have been multiplied to reduce population growth, starting at $c = 0.3833$ and $1/R_0 = 0.045$, respectively. The values on the color bar are on a log scale, with a minimum of 0.0006% and a maximum of 41.4%. The thick black line represents combinations of reduced survival and fertility where the population is stationary (i.e., $\lambda = 1$). The thin black lines below and above this line represent combinations where $\lambda = 0.95$ and $\lambda = 1.05$, respectively. The high-growth scenario is located in the upper right corner at $(1, 1)$.

heterogeneity). At the other extreme, with reduced survival but high fertility (the upper left corner of the plot), the contribution of maternal age heterogeneity is effectively zero ($\mathcal{K} = 6 \times 10^{-6}$).

We conclude that despite the pronounced effects of maternal age on survival and fertility (fig. 1) and on the mean and variance of LRO (fig. 2), over much of the ecologically reasonable parameter space, heterogeneity in maternal age contributes less than 5%—and often much less than 5%—of the variance in LRO. The remainder is due to individual stochasticity within maternal age groups.

Discussion

LRO is an important component of fitness, and under some definitions its mean is a measure of per-generation population growth (Cushing and Zhou 1994; Heesterbeek 2002; Caswell 2011). It is an integrative and empirically measurable fitness component (Clutton-Brock 1988) and provides a clear link between individual outcomes and population metrics. The variance and higher moments of LRO are indicative of inequality and uncertainty among individuals. Thus, natural selection may operate on variance in LRO. Not all of the variance in LRO will be heritable, however. In particular, variance due to individual stochasticity is not heritable and may mask heritable variation (Steiner and Tuljapurkar 2012). Disentangling the relative contributions of stochasticity and heterogeneity to interindividual variability is therefore a question of increasing interest.

Maternal age heterogeneity is expressed in rotifers as dramatically decreased survival and fertility with increasing age of a mother at the birth of an individual. The interindividual differences in vital rates generate a pronounced decrease in the mean and an increase in the variance of LRO for individuals of increasing maternal age. In the laboratory environment, this maternal age heterogeneity generates 26% of the interindividual variance; the other 74% is due to individual stochasticity.

Such laboratory environments often confront the problem of unrealistic ecological conditions. By carefully controlling the conditions (i.e., "keeping the rotifers happy"), the specific effect of a given factor, whether that is food, temperature, or maternal age, can be quantified. But optimal conditions for the experimental organisms often result in demographic models with unreasonably high growth rates, which in turn affect the age and stage structure as well as other demographic parameters of the population. In natural systems, predators or diseases will lower the survival rates of individuals, and density effects or reduced food availability will reduce fertility rates.

By exploring a range of reductions in vital rates, we show that the contribution of maternal age heterogeneity to the variance decreases when survival is lowered regardless of the level of fertility. At the same time, lowering fertility can increase the contribution of maternal age, but only when survival is high. Along the range of parameter values for which the population growth $\lambda \approx 1$, the contribution of heterogeneity tends to be low, with only about 15% of parameter combinations resulting in contributions of 5% or higher. These results demonstrate that the variance in LRO and its decomposition into components are not fixed properties of a species or a type of life cycle but should be considered as demographic outcomes that respond to environmental conditions.

The scenarios that reduce survival and/or fertility mimic the effects of a "poor" environment relative to the high-growth laboratory environment. From figure 4, it is clear that poor environments are not all equivalent. Several recent studies have reported an increase in the importance of heterogeneity when environments become poor, where poor environments result in a decrease in (some of) the vital rates (e.g., Tavecchia et al. 2005; Barbraud and Weimerskirch 2005; Jenouvrier et al. 2019). This could be due to the fact that individuals are more likely to allocate resources away from reproduction to maintain their survival when they encounter very poor environments. Studies of caloric restriction and life span extension, as well as studies of dynamic energy budgets, predict such responses (e.g., Noonburg et al. 1998; Kirkwood and Shanley 2005). Examples of this have been found in some species of rotifers (Gribble et al. 2014; Bock et al. 2019; Kirk 2001), *Daphnia* (Lynch and Ennis 1983), spiders (Austad 1989), *Drosophila* (Partridge et al. 2005), fish (Comfort 1960), and rodents (Merry 2005).

Under ecologically reasonable conditions, rotifers are likely to experience far more extrinsic mortality (given that everything eats rotifers; Williamson 1983; Nagata et al. 2005). Food limitation has furthermore been shown to suppress reproduction in rotifers (King 1967). The ephemeral nature of rotifer habitats and the fact that *Brachionus manjavacas* change their reproductive strategy from asexual to sexual depending on environmental conditions (Gilbert 2003, 2017) suggest that the interaction between rotifers and their environments is more complex than we assume here. Experiments in which food or mortality levels vary for mothers and/or offspring are necessary.

Further experimentation is also required to identify how the maternal age effect is conferred from mother to offspring. In several cases, maternal effects are due to changes in the ability of older mothers to allocate resources or provide care to their offspring, which can be beneficial if either of those processes require maternal experience (e.g., Jones et al. 2005; Robbins et al. 2006; Bogdanova et al. 2007; Plaistow et al. 2007; Green 2008; Kroeger et al. 2020). However, senescence can also reduce the provisioning by mothers with increasing age (e.g., Giron and Casas

2003; Beamonte-Barrientos et al. 2010). In *B. manjavacas*, maternal care is not observed, and although prenatal maternal provisioning decreased slightly with age of the mother, these differences were not sufficient to explain the maternal effect (Bock et al. 2019). Other potential mechanisms for the maternal age effect in this rotifer species are epigenetic effects (e.g., Markunas et al. 2016; Moore et al. 2019) and processes such as mutation accumulation and antagonistic pleiotropy. This type of maternal age heterogeneity is therefore unlikely to be heritable, although more research into the molecular mechanisms is needed to definitively make such a claim.

Maternal age generates heterogeneity in rotifer vital rates depending on environmental conditions. We expect that this conclusion is generally applicable to a species with an age-classified life history and an age-specific detrimental maternal effect and whose survival and fertility can be described by Weibull and Coale-Trussell models. Our results also reflect environmental conditions beyond those of the original laboratory experiment. Furthermore, all results are obtained from analytical formulas, suggesting that life histories with similar rates will show qualitatively similar outcomes.

Incorporating a beneficial maternal age effect rather than a detrimental one would result in a different pattern of maternal age heterogeneity. The multistate model framework we employ here, however, is easily adapted to include alternative maternal effects. It is also flexible in the life histories it can incorporate: rather than by age, one could classify individuals by size class, breeding status, or developmental stage. Combining the states of the life cycle and the maternal effect on the vital rates expressed as each of those states provides a powerful way to investigate the lifetime scope of maternal effect heterogeneity. Although we have not explored it here because the model is formulated in matrix terms, it has the potential to include stochastic environments (e.g., Caswell 2011; van Daalen and Caswell 2020), density dependence, and sensitivity analysis (van Daalen and Caswell 2017, 2020).

It has become increasingly clear that individual stochasticity nearly always dominates the variance in life history outcomes (Steiner and Tuljapurkar 2012; Hartemink et al. 2017; Snyder and Ellner 2018; Hartemink and Caswell 2018; Jenouvrier et al. 2018; van Daalen and Caswell 2020). Studies that decompose variance into contributions by stochasticity and heterogeneity have primarily been performed in the context of longevity. Most of these are attempts to quantify the contribution of latent unobserved heterogeneity to variance in longevity, for example, in humans (Caswell 2014; Hartemink et al. 2017) and in animals (Hartemink and Caswell 2018; Jenouvrier et al. 2018). These have identified contributions by heterogeneous frailty in the range of 1%–10% in human populations and a median of 35% for several populations of laboratory invertebrates.

In the few studies that partition variance in LRO, contributions have been obtained of about 39% due to heterogeneous "quality" in kittiwakes (Snyder and Ellner 2018) and of 22% from unobserved heterogeneity in the southern fulmar (Jenouvrier et al. 2018). An example of practically 0% contribution to the variance from environment at birth was found for a perennial herb in a fire-prone environment (van Daalen and Caswell 2020). In a purposely extreme comparison, treating the life history differences among 83 populations of animals and 332 populations of plants as a kind of heterogeneity, interspecies heterogeneity could account for only 11% and 1% of the variance in LRO in plants and animals, respectively (van Daalen 2020). This range of generally low numbers shows that we need to be careful in what we consider to be a substantial amount of variance due to heterogeneity; such a thing is best defined by collecting multiple measures for the same population under different conditions.

The models used in many of such variance partitioning studies, whether in longevity or in LRO, were estimated in the context of searching for latent unobserved heterogeneity in an attempt to explain variance among individuals (Cam et al. 2016; Authier et al. 2017; Gimenez et al. 2018; Hamel et al. 2018). These have therefore made certain assumptions, statistically or otherwise, about how the heterogeneity variable fits into the life cycle model. Snyder and Ellner (2018), for example, incorporate an individual quality variable x into an integral projection model of breeding status for kittiwakes and assume that survival and breeding probability are functions of x so that these probabilities are perfectly correlated. Maternal age, on the other hand, is a source of heterogeneity that can be observed, enabling direct variance partitioning rather than inferential analysis.

It is not difficult to find individual differences in physiological, behavioral, and life historical traits. Some of those traits will have large effects on individual survival, growth, and reproduction, at some or all life cycle stages. In our case, the effects of maternal age on the vital rates of *Brachionus* offspring are dramatic and long-lasting. Being born in the laboratory to a young rather than an old mother nearly triples mean LRO, and a population is a mixture of individuals of different maternal ages. In spite of this, the contribution of maternal age heterogeneity to the variance in LRO is highly labile and responds strongly to environmental differences that affect the demographic rates. In the admittedly unrealistic laboratory environment, heterogeneity accounts for 26% of the variance in LRO. In a range of more ecologically reasonable conditions, about 85% of the parameter space yields contributions of heterogeneity of less than 5%.

Our results imply that no matter how large the effects of some trait may be at the individual level, it is not safe

to assume, on this basis alone, that heterogeneity in this trait will make a major contribution to variance in LRO. Stochasticity can never be ignored, and the relative contributions of heterogeneity and stochasticity can be quantified only after incorporating the heterogeneity in a demographic model from which stochasticity can be computed.

This does not mean that maternal age effects are unimportant. Indeed, it is well documented that maternal age has important effects on the aging, survival, and reproduction of rotifers (e.g., Lansing 1947; Gribble et al. 2014; Gribble and Snell 2018; Bock et al. 2019; Hernández et al. 2020). It is known that those effects interact with dietary regimens and influence diverse physiological and molecular processes. Our conclusions are a reminder, if a reminder were needed, of the value of studies at the individual level to illuminate the processes by which maternal age has its effects.

The approach we have applied here to *B. manjavacas* is easily extended to heterogeneity in any trait in any age- or stage-classified life cycle. Accounting for the sources of variance in LRO is an important question, and our results provide, for the first time, a welcome solution to this problem.

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Statement of Authorship

S.F.v.D., C.M.H., H.C., M.G.N., and K.E.G. designed the research; K.E.G. supervised laboratory experiments; S.F.v.D. performed mathematical analyses; S.F.v.D., C.M.H., H.C., M.G.N., and K.E.G. analyzed data; S.F.v.D., C.M.H., and M.G.N. created figures; and all authors wrote and revised the manuscript.

Data and Code Availability

Data and Matlab code are accessible through the Dryad Digital Repository (https://doi.org/10.5061/dryad.vmcvdnctb; van Daalen 2022).

Literature Cited

Austad, S. N. 1989. Life extension by dietary restriction in the bowl and doily spider, *Frontinella pyramitela*. Experimental Gerontology 24:83–92.

Authier, M., L. M. Aubry, and E. Cam. 2017. Wolf in sheep's clothing: model misspecification undermines tests of the neutral theory for life histories. Ecology and Evolution 7:3348–3361.

Barbraud, C., and H. Weimerskirch. 2005. Environmental conditions and breeding experience affect costs of reproduction in blue petrels. Ecology 86:682–692.

Barker, J. S. F. S. 2009. Defining fitness in natural and domesticated populations. Pages 3–14 *in* Adaptation and fitness in animal populations. Springer, Dordrecht.

Barks, P. M., and R. A. Laird. 2020. Parental age effects and the evolution of senescence. American Naturalist 195:886–898.

Beamonte-Barrientos, R., A. Velando, H. Drummond, and R. Torres. 2010. Senescence of maternal effects: aging influences egg quality and rearing capacities of a long-lived bird. American Naturalist 175:469–480.

Bernardo, J. 1996. Maternal effects in animal ecology. American Zoologist 36:83–105.

Bock, M. J., G. C. Jarvis, E. L. Corey, E. E. Stone, and K. E. Gribble. 2019. Maternal age alters offspring lifespan, fitness, and lifespan extension under caloric restriction. Scientific Reports 9:1–16.

Bogdanova, M. I., R. G. Nager, and P. Monaghan. 2007. Age of the incubating parents affects nestling survival: an experimental study of the herring gull *Larus argentatus*. Journal of Avian Biology 38:83–93.

Bouwhuis, S., O. Vedder, and P. H. Becker. 2015. Sex-specific pathways of parental age effects on offspring lifetime reproductive success in a long-lived seabird. Evolution 69:1760–1771.

Brown, G. R., K. N. Laland, and M. Borgerhoff Mulder. 2009. Bateman's principles and human sex roles. Trends in Ecology and Evolution 24:297–304.

Cam, E., L. M. Aubry, and M. Authier. 2016. The conundrum of heterogeneities in life history studies. Trends in Ecology and Evolution 31:872–886.

Carey, J., P. Liedo, H.-G. Müller, J.-L. Wang, and J. Vaupel. 1998. A simple graphical technique for displaying individual fertility data and cohort survival: case study of 1000 Mediterranean fruit fly females. Functional Ecology 12:359–363.

Carnes, B. A., R. Riesch, and I. Schlupp. 2012. The delayed impact of parental age on offspring mortality in mice. Journals of Gerontology Series A: Biological Sciences and Medical Sciences 67A:351–357.

Caswell, H. 2001. Matrix population models: construction, analysis, and interpretation. 2nd ed. Sinauer, Sunderland.

———. 2009. Stage, age and individual stochasticity in demography. Oikos 118:1763–1782.

———. 2011. Beyond R_0: demographic models for variability of lifetime reproductive output. PLoS ONE 6:e20809.

———. 2012. Matrix models and sensitivity analysis of populations classified by age and stage: a vec-permutation matrix approach. Theoretical Ecology 5:403–417.

———. 2014. A matrix approach to the statistics of longevity in heterogeneous frailty models. Demographic Research 31:553–592.

Caswell, H., C. de Vries, N. Hartemink, G. Roth, and S. F. van Daalen. 2018. Age × stage-classified demographic analysis: a comprehensive approach. Ecological Monographs 88:560–584.

Clutton-Brock, T. H. 1988. Reproductive success. University of Chicago Press, Chicago.

Coale, A. J., and T. J. Trussell. 1974. Model fertility schedules: variations in the age structure of childbearing in human populations. Population Index 40:185–258.

Comfort, A. 1960. The effect of age on growth-resumption in fish (Lebistes) checked by food restriction. Gerontology 4:177–186.

Crow, J. F. 1958. Some possibilities for measuring selection intensities in man. Human Biology 30:1–13.

Cushing, J. M., and Y. Zhou. 1994. The net reproductive value and stability in matrix population models. Natural Resources Modeling 8:297–333.

Falconer, D. S. 1960. Introduction to quantitative genetics. Ronald, New York.

Fox, C. W., M. L. Bush, and W. G. Wallin. 2003. Maternal age affects offspring lifespan of the seed beetle, *Callosobruchus maculatus*. Functional Ecology 17:811–820.

Gilbert, J. J. 2003. Specificity of crowding response that induces sexuality in the rotifer *Brachionus*. Limnology and Oceanography 48:1297–1303.

———. 2017. Non-genetic polymorphisms in rotifers: environmental and endogenous controls, development, and features for predictable or unpredictable environments. Biological Reviews 92:964–992.

Gillespie, D. O. S., A. F. Russell, and V. Lummaa. 2013. The effect of maternal age and reproductive history on offspring survival and lifetime reproduction in preindustrial humans. Evolution 67:1964–1974.

Gimenez, O., E. Cam, and J.-M. Gaillard. 2018. Individual heterogeneity and capture-recapture models: what, why and how? Oikos 127:664–686.

Giron, D., and J. Casas. 2003. Mothers reduce egg provisioning with age. Ecology Letters 6:273–277.

Goodman, L. A. 1969. The analysis of population growth when the birth and death rates depend upon several factors. Biometrics 25:659–681.

Grafen, A. 1988. On the uses of data on lifetime reproductive success. Pages 454–471 *in* T. H. Clutton-Brock, ed. Reproductive success. University of Chicago Press, Chicago.

Green, B. S. 2008. Maternal effects in fish populations. Pages 1–105 *in* Advances in marine biology. Vol. 54, chap. 1.

Gribble, K. E., G. Jarvis, M. Bock, and D. B. M. Welch. 2014. Maternal caloric restriction partially rescues the deleterious effects of advanced maternal age on offspring. Aging Cell 13:623–630.

Gribble, K. E., and T. W. Snell. 2018. Rotifers as a model for the biology of aging. Pages 483–495 *in* J. L. Ram and P. M. Conn, eds. Conn's handbook of models for human aging. 2nd ed. Academic Press, London.

Hamel, S., J.-M. Gaillard, M. Douhard, M. Festa-Bianchet, F. Pelletier, and N. G. Yoccoz. 2018. Quantifying individual heterogeneity and its influence on life-history trajectories: different methods for different questions and contexts. Oikos 127:687–704.

Hamilton, W. D. 1966. The moulding of senescence by natural selection. Journal of Theoretical Biology 12:12–45.

Hartemink, N., and H. Caswell. 2018. Variance in animal longevity: contributions of heterogeneity and stochasticity. Population Ecology 60:89–99.

Hartemink, N., T. I. Missov, and H. Caswell. 2017. Stochasticity, heterogeneity, and variance in longevity in human populations. Theoretical Population Biology 114:107–116.

Heesterbeek, J. A. 2002. A brief history of R_0 and a recipe for its calculation. Acta Biotheoretica 50:189–204.

Hercus, M. J., and A. A. Hoffman. 2000. Maternal and grandmaternal age influence offspring fitness in *Drosophila*. Proceedings of the Royal Society B 267:2105–2110.

Hernández, C. M., S. F. van Daalen, H. Caswell, M. G. Neubert, and K. E. Gribble. 2020. A demographic and evolutionary analysis of maternal effect senescence. Proceedings of the National Academy of Sciences of the USA 117:16431–16437.

Horvitz, C. C., and S. Tuljapurkar. 2008. Stage dynamics, period survival, and mortality plateaus. American Naturalist 172:203–215.

Jenouvrier, S., L. M. Aubry, C. Barbraud, H. Weimerskirch, and H. Caswell. 2018. Interacting effects of unobserved heterogeneity and individual stochasticity in the life history of the southern fulmar. Journal of Animal Ecology 87:212–222.

Jenouvrier, S., L. M. Aubry, S. F. van Daalen, C. Barbraud, H. Weimerskirch, and H. Caswell. 2019. When the going gets tough, the tough get going: effect of extreme climate on an Antarctic seabird's life history. bioRxiv, https://doi.org/10.1101/791855.

Jones, O. R., M. J. Crawley, J. G. Pilkington, and J. M. Pemberton. 2005. Predictors of early survival in Soay sheep: cohort-, maternal- and individual-level variation. Proceedings of the Royal Society B 272:2619–2625.

Kern, S., M. Ackerman, S. C. Stearns, and T. J. Kawecki. 2001. Decline in offspring viability as a manifestation of aging in *Drosophila melanogaster*. Evolution 55:1822–1831.

King, C. E. 1967. Food, age, and the dynamics of a laboratory population of rotifers. Ecology 48:111–128.

Kirk, K. L. 2001. Dietary restriction and aging: comparative tests of evolutionary hypotheses. Journals of Gerontology Series A: Biological Sciences and Medical Sciences 56:B123–B129.

Kirkwood, T. B., and D. P. Shanley. 2005. Food restriction, evolution and ageing. Mechanisms of Ageing and Development 126:1011–1016.

Kroeger, S. B., D. T. Blumstein, K. B. Armitage, J. M. Reid, and J. G. Martin. 2020. Older mothers produce more successful daughters. Proceedings of the National Academy of Sciences of the USA 117:4809–4814.

Land, K. C., and A. Rogers. 1982. Multidimensional mathematical demography. Academic Press, New York.

Lansing, A. I. 1947. A transmissible, cumulative and reversible factor in aging. Journal of Gerontology 2:228–239.

Lotka, A. J. 1939. Théorie analytique des associations biologiques: analyse démographique avec application particulière à l'espèce humaine. Actualités Scientifiques et Industrielles. Published in translation as: Analytical theory of biological populations, translated by D. P. Smith and H. Rossert. Plenum, New York, 1998 ed. Hermann et Cie, Paris.

Lynch, M., and R. Ennis. 1983. Resource availability, maternal effects, and longevity. Experimental Gerontology 18:147–165.

Markunas, C. A., A. J. Wilcox, Z. Xu, B. R. Joubert, S. Harlid, V. Panduri, S. E. Håberg, et al. 2016. Maternal age at delivery is associated with an epigenetic signature in both newborns and adults. PLoS ONE 11:e0156361.

Marshall, D. J., and T. Uller. 2007. When is a maternal effect adaptive? Oikos 116:1957–1963.

Merry, B. J. 2005. Dietary restriction in rodents—delayed or retarded ageing? Mechanisms of Ageing and Development 126:951–959.

Metz, J. A. J., R. M. Nisbet, and S. A. H. Geritz. 1992. How should we define "fitness" for general ecological scenarios? Trends in Ecology and Evolution 7:198–202.

Moorad, J. A., and D. H. Nussey. 2016. Evolution of maternal effect senescence. Proceedings of the National Academy of Sciences of the USA 113:362–367.

Moore, A. M., Z. Xu, R. T. Kolli, A. J. White, D. P. Sandler, and J. A. Taylor. 2019. Persistent epigenetic changes in adult daughters of older mothers. Epigenetics 14:467–476.

Nagata, T., J.-Y. Ha, and T. Hanazato. 2005. The predation impact of larval *Pseudorasbora parva* (cyprinidae) on zooplankton: a mesocosm experiment. Journal of Freshwater Ecology 20:757–763.

Newton, I. 1989. Lifetime reproduction in birds. Academic Press, New York.

Noonburg, E., R. Nisbet, E. McCauley, W. Gurney, W. Murdoch, and A. De Roos. 1998. Experimental testing of dynamic energy budget models. Functional Ecology 12:211–222.

Partridge, L., M. D. W. Piper, and W. Mair. 2005. Dietary restriction in *Drosophila*. Mechanisms of Ageing and Development 126:938–950.

Plaistow, S. J., C. Shirley, H. Collin, S. J. Cornell, and E. D. Harney. 2015. Offspring provisioning explains clone-specific maternal age effects on life history and life span in the water flea, *Daphnia pulex*. American Naturalist 186:376–389.

Plaistow, S. J., J. J. H. St. Clair, J. Grant, and T. G. Benton. 2007. How to put all your eggs in one basket: empirical patterns of offspring provisioning throughout a mother's lifetime. American Naturalist 170:520–529.

Reichert, S., V. Berger, J. Jackson, S. N. Chapman, W. Htut, K. U. Mar, and V. Lummaa. 2020. Maternal age at birth shapes offspring life-history trajectory across generations in long-lived Asian elephants. Journal of Animal Ecology 89:996–1007.

Rényi, A. 1970. Probability theory. North-Holland, Amsterdam.

Robbins, A. M., M. M. Robbins, N. Gerald-Steklis, and H. D. Steklis. 2006. Age-related patterns of reproductive success among female mountain gorillas. American Journal of Physical Anthropology 131:511–521.

Roff, D. A. 2008. Defining fitness in evolutionary models. Journal of Genetics 87:339–348.

Snyder, R. E., and S. P. Ellner. 2018. Pluck or luck: does trait variation or chance drive variation in lifetime reproductive success? American Naturalist 191:E90–E107.

Stearns, S. C. 1992. The evolution of life histories. Oxford University Press, Oxford.

Steiner, U. K., and S. Tuljapurkar. 2012. Neutral theory for life histories and individual variability in fitness components. Proceedings of the National Academy of Sciences of the USA 109:4684–4689.

Steiner, U. K., S. Tuljapurkar, and S. H. Orzack. 2010. Dynamic heterogeneity and life history variability in the kittiwake. Journal of Animal Ecology 79:436–444.

Tavecchia, G., T. Coulson, B. J. Morgan, J. M. Pemberton, J. Pilkington, F. Gulland, and T. H. Clutton-Brock. 2005. Predictors of reproductive cost in female Soay sheep. Journal of Animal Ecology 74:201–213.

Tuljapurkar, S., and C. C. Horvitz. 2006. From stage to age in variable environments: life expectancy and survivorship. Ecology 87:1497–1509.

Tuljapurkar, S., U. K. Steiner, and S. H. Orzack. 2009. Dynamic heterogeneity in life histories. Ecology Letters 12:93–106.

van Daalen, S. F. 2020. Get lucky: variability in lifetime reproductive output. Doctoral diss. Universiteit van Amsterdam, Amsterdam.

———. 2022. Data from: The contributions of maternal age heterogeneity to variance in lifetime reproductive output. American Naturalist, Dryad Digital Repository, https://doi.org/10.5061/dryad.vmcvdnctb.

van Daalen, S. F., and H. Caswell. 2015. Lifetime reproduction and the second demographic transition: stochasticity and individual variation. Demographic Research 33:561–588.

———. 2017. Lifetime reproductive output: individual stochasticity, variance, and sensitivity analysis. Theoretical Ecology 10:355–374.

———. 2020. Variance as a life history outcome: sensitivity analysis of the contributions of stochasticity and heterogeneity. Ecological Modelling 417:108856.

———. Forthcoming. Demographic sources of variation in fitness. *In* R. Sear, R. Lee, and O. F. Burger, eds. Human evolutionary demography. Open Book, Cambridge.

Williamson, C. E. 1983. Invertebrate predation on planktonic rotifers. Hydrobiologia 104:385–396.

Williamson, C. E., and N. M. Butler. 1986. Predation on rotifers by the suspension-feeding calanoid copepod *Diaptomus pallidus*. Limnology and Oceanography 31:393–402.

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