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METHODS

Skewed distributions of lifetime reproductive success: beyond mean and variance

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Abstract

Lifetime reproductive performance is quantified here by the LRS (lifetime reproductive success), the random number of offspring an individual produces over its lifetime. Many field studies find that distributions of LRS among individuals are non-normal, zero-inflated and highly skewed. These results beg the question, what is the distribution of LRS predicted by demographic models when the only source of randomness is demographic stochasticity? Here we present the first exact analysis of the probability distribution of LRS for species described by age + stage models; our analysis starts with estimated vital rates. We illustrate with three examples: the Hadza, human hunter-foragers (age-only), the evergreen tree *Tsuga canadensis* (stage-only) and Roe deer, *Capreolus capreolus* (age + stage). For each we obtain the exact distribution of LRS, but also calculate and discuss the first three moments. Our results point to important questions about how such LRS distributions affect natural selection, and life history evolution.

Keywords

Age + stage, evolution, life history, lifetime reproductive success.

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INTRODUCTION

Lifetime measures of reproductive performance describe the number of offspring an individual produces over its lifespan (Clutton-Brock 1988). Many empirical biologists consider such lifetime measures as components of individual fitness (e.g. Kruuk *et al*. 1999; Brommer *et al*. 2004), since they provide generational estimators of the opportunity for natural and sexual selection and phenotypic evolution (Trivers 1972). For instance, many empirical studies have assessed the opportunity for selection using the standardized variance (I metric, Crow 1958) of reproductive performance and revealed high variation (e.g. in females from 0.177 in lions, *Panthera leo* to 3.62 in elephant seals, *Mirounga angustirostris*, Cabana & Kramer 1991; and in males from 0.59 in Atlantic salmon *Salmo salar* to 4.52 in bighorn sheep, *Ovis canadensis*, Tatarenkov *et al*. 2008). Comparison of standardized variances of lifetime reproductive performance between sexes within a population is commonly used for assessing the strength of sexual selection (e.g. Dubuc *et al*. 2014 on Rhesus macaques, *Macaca mulatta*). These empirical studies consistently reveal that distributions of lifetime reproductive performance measures are often non-normal, zero-inflated and highly skewed (Goodwin & Vaupel 1985), and this begs the question: what are the demographic causes of these unusually shaped distributions?

Although there are many metrics utilized in the literature (depending in part on when in the life cycle offspring are counted as 'successes'), here we define lifetime reproductive success (LRS), by counting offspring at birth. Our analyses can be straightforwardly modified for different starting ages/stages that have been used to census offspring, e.g. weaning/fledging (van Noordwijk & van Schaik 1999), 1 year-old (Newton 1988), 2 years-old (Clutton-Brock 1988). In the models here, individuals are distinguished by age, stage or age + stage. Life cycles are described by probabilities of survival, stage transition and number of offspring produced at each time step. Every event in the life cycle is subject to a form of demographic stochasticity. For example, reproductive output depends on age and/or stage but an individual may have 0, 1, 2, ... offspring, with a probability distribution that changes with the individual's age and/or stage. In the same sense, individual survival and transition between stages are stochastic. The LRS for an individual, then, is a random number that is a cumulative sum over a sequence of random events that ends in death. This cumulative randomness has been called dynamic heterogeneity by Tuljapurkar *et al*. (2009b) and individual stochasticity by Caswell (2009).

The starting point here is a set of vital rates (or corresponding probability distributions) for each age and/or stage in a life cycle. We use rates that are estimated by methods such as, e.g. multistate mark-recapture models (White *et al*. 2006; Lebreton *et al*. 2009), or Aster models (Geyer *et al*. 2007), and we assume that they are known exactly. We emphasize that estimation of vital rates is different from the estimation of LRS. The former requires observations of single-period transitions and reproduction of individuals of different ages/stages, usually repeated a few times, and uses this data to estimate age + stage transition rates and fertility (examples are given by Preston *et al*. 2000; Caswell 2001a). In contrast, the empirical study of LRS requires following longitudinally each

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member of at least one large cohort of individuals over time until death. Such long-term studies are still rare. All examples used here rely only on data that allow estimation of vital rates, not data on distributions of LRS. Simulations of the LRS (Tuljapurkar *et al*. 2009b) do not substitute for exact results; they depend on sample size and can underestimate small probabilities (related points are made in Caswell 2011; van Daalen & Caswell 2015). For populations structured only by age, the variance in LRS has previously been computed by Charlesworth & Williamson (1975) and Waples *et al*. (2011). For age + stage models, Steiner & Tuljapurkar (2012) obtained equations for the moments of the LRS (such as mean, variance and skewness), but used average reproduction. In an important extension, van Daalen & Caswell (2017) (hereafter referred to as vDC) started from the probability distribution of offspring number for each age + stage, and obtained formulas for the moments of LRS (extending Caswell 2011; van Daalen & Caswell 2015). The vDC method was used by Snyder & Ellner (2018) who found that stochasticity (in the present demographic sense) matters, so in some cases 'luck' can be more important to fitness than 'pluck'.

Yet, even exact moments do not provide the same information as the exact probability distribution of the LRS. Why? One reason is that our intuitive understanding of these moments is often based on a roughly normal distribution and so can be of limited value. Another is that approximating the full LRS distribution by the first few moments by the Gram-Charlier expansion (Stuart *et al*. 1994) does not work for the LRS distributions we illustrate here, and we suspect that it may often perform poorly, especially when there is a high death rate prior to the onset of reproduction. Thus the exact probabilities for LRS are essential for describing the variability among individuals, certainly when the distribution is highly skewed or multimodal.

Here we present the first exact analysis that yields the probability distribution of LRS for species described by age + stage models (including age-only and stage-only models). This computation is not straightforward: for example, in a stage-structured population, stasis means that an individual may spend a long time in a non-reproductive stage, and this matters to the distribution of the LRS. Our method deals exactly with such possibilities. Our computation is significantly faster than a large simulation (by orders of magnitude for some stage-structured models). We show below by examples that the computed distribution of LRS contains new and useful biological insights. Our methods point to important questions: what factors determine the probability that individuals produce zero offspring during their lifetimes; how does a skewed LRS distribution affect natural selection, effective population size and life history evolution? We discuss these questions, and point towards future work.

The methods we present for age + stage structure use vital rates that may follow any specified pattern, driven for example by density or environment. We do not consider the question of whether empirical LRS samples follow the distributions we compute. The distribution we compute is exactly determined by the vital rates. Indeed, if an LRS distribution obtained by our methods (starting from vital rates) differs from an LRS distribution that is observed empirically, the mismatch would indicate that other factors are at play. In this way, the exact distributions we compute can serve as a null hypothesis, an important tool for the detection of such factors as trade-offs.

The main text explains our method, and then focuses on examples. The first example is for a human population, the Hadza, a Tanzanian group of hunter-foragers. The next example is a stage-only model for the large evergreen tree *Tsuga canadensis*. For these examples, the data sources are cited as we use them (the matrices were provided by vDC, and are also in the Appendix S1). We also provide (in the Appendix S1) a fully worked example for this case, to illustrate our methods in detail. The last example, and perhaps most general, is an age + stage model for Roe deer, *Capreolus capreolus*, based on Plard *et al*. (2015). All our examples start with vital rates by age and/or stage that have been estimated from empirical data, and our goal is to compute the distribution of LRS implied by those vital rates.

METHODS

Lifetime reproductive success is a random quantity, and we find the probability for each possible value of LRS. We model demography in discrete time; vital rates may be structured only by age, only by stage or by a combination of age and stage (age + stage). Mathematical details are in the Appendix S1; we use and explain discrete convolutions and discrete Fourier transforms, unfamiliar to some biologists, but essential here. Code [based on R, R Core Team 2019 and the R package FFTW (Fastest Fourier Transform in the West), Mersmann 2019] is available from the authors. Our results should apply to continuous stages using established methods (Ellner *et al*. 2016; Snyder & Ellner 2018). Notation is always a nuisance; where possible, we use notation similar to Caswell (2011) and Caswell *et al*. (2018).

Our logic uses three points. First, every individual has a particular age of death; second, at each age before death, there is a probability distribution for the number of offspring produced at that age; and third, convolution of these age-specific distributions (weighted appropriately by mortality) yields the distribution of LRS. We assume that the probability distribution at each age is independent of the distribution at all other ages, but is otherwise arbitrary.

General framework

Age is counted in discrete intervals $a = 1, 2, \ldots, \omega$ and newborns are always in the first age interval (parameters are defined in Tables 1 and A1). Stage is defined in discrete categories, $s = 1, 2, \ldots, S$ (indexed as i or j) for stage-only and age + stage models. Stage transition probabilities (conditional on remaining alive) over a single time step are defined in a matrix $\boldsymbol{G}$ for stage-only models, or in a set of matrices $\boldsymbol{G}_a$ for age + stage models. One-period survival probabilities (from one interval of time to the next) are p_a for age-only models, $p(j)$ for stage-only models and $p_a(j)$ for age + stage models (Table 1). Multiplying stage transitions by stage survivals, we obtain an $S \times S$ matrix of unconditional stage transition probabilities, $\boldsymbol{U}$ for stage-only models and a set $\boldsymbol{U}_a$ for age + stage models.

Age at death is a random variable (well-defined even when one-period survival depends only upon stage). The probability

Table 1 Parameters and definitions

$a = 1, \ldots, \omega$	Age, discrete intervals. Newborns are in the first interval $a = 1$. ω is maximum age.
$s = 1, \ldots, S$	Stage, discrete categories. S is the total number of stages.
$\mathbf{G}$, $0 \le \mathbf{G}(i, j) \le 1$, $i, j = 1, 2, \ldots, S$	Stage-only transition matrix, probability individual in stage j will be in stage i in the next time interval, conditional on survival. $S \times S$ matrix, column stochastic.
$\mathbf{G}_a$, $0 \le \mathbf{G}_a(i, j) \le 1$, $i, j = 1, 2, \ldots, S$	Age + stage transition matrix, individual age a in stage j will be age $a + 1$ in stage i, conditional on survival. $S \times S$ matrix, column stochastic.
p_a, $0 \le p_a \le 1$	One-period survival (age-only) from age a to age $a + 1$. $p_\omega = 0$.
$p(j)$, $0 \le p(j) \le 1$, $j = 1, 2, \ldots, S$	One-period survival (stage-only) from stage j to any other stage.
$p_a(j)$, $0 \le p_a(j) \le 1$, $j = 1, 2, \ldots, S$	One-period survival (age + stage) from age a and stage j to the next time interval. $p_\omega(j) = 0$ for all j.
l_a, $0 \le l_a \le 1$	Survivorship to age a, probability of being alive at age a. $l_1 = 1$, $l_2 = p_1\, l_1$, $l_3 = p_2\, l_2$, …, $l_{\omega+1} = 0$.
$\boldsymbol{L}_a$, $0 \le \boldsymbol{L}_a(i, j) \le 1$, $i, j = 1, 2, \ldots, S$, $a = 1, 2, \ldots, \omega$	Survivorship to age a by stage, probability of being alive in stage i at age a for those born into stage j. $S \times S$ matrix, changes with age (see text).
ϕ_a, $0 \le \phi_a \le 1$, $a = 1, 2, \ldots, \omega$, $\sum_a \phi_a = 1$	Probability of age at death, $\phi_a = l_a\,(1 - p_a)$ for age-only model (for stage-only and age + stage models see text).
$\mathbf{U}$, $0 \le \mathbf{U}(i, j) \le p(j)$, $i, j = 1, 2, \ldots, S$, $\mathbf{U}(i, j) = \mathbf{G}(i, j)\, p(j)$	Cohort projection matrix (stage-only), probability an individual in stage j is alive in stage i at the next time interval. $S \times S$ matrix, does not change with age.
$\mathbf{U}_a$, $0 \le \mathbf{U}_a(i, j) \le p_a(j)$, $i, j = 1, 2, \ldots, S$, $\mathbf{U}_a(i, j) = \mathbf{G}_a(i, j)\, p_a(j)$	Cohort projection matrix (age + stage), probability an individual age a in stage j is alive at age $a + 1$ in stage i. $S \times S$ matrix, may change with age.
$\boldsymbol{\kappa}_a$	Vector of probabilities that an individual age a produces k offspring, $\kappa_a(k)$, where $0 \le \kappa_a(k) \le 1$, $\sum_k \kappa_a(k) = 1$, and $a = 1, 2, \ldots, \omega$.
$\boldsymbol{\kappa}_j$	Vector of probabilities that an individual in stage j produces k offspring at a single time interval, $\kappa_j\,(k)$, where $0 \le \kappa_j\,(k) \le 1$, $\sum_k \kappa_j(k) = 1$, and $j = 1, 2, \ldots, S$.
$\boldsymbol{\kappa}_{a,j}$	Vector of probabilities an individual at age a and stage j produces k offspring, $\kappa_{a,j}\,(k)$, where $0 \le \kappa_{a,j}\,(k) \le 1$, $\sum_k \kappa_{a,j}(k) = 1$, and $a = 1, 2, \ldots, \omega$. For each a, an $k_m \times S$ matrix (each column is for a stage j).
$\Gamma(k), 0 \le \Gamma(k) \le 1, \sum_k \Gamma(k) = 1$	Probability an individual produces k offspring during its lifetime.
Γ	Vector of probabilities, $\Gamma(0)$, $\Gamma(1)$, $\Gamma(2)$, …, $\Gamma(k_U)$. k_U = maximum possible number of offspring.
$\gamma_a = \phi_a \boldsymbol{\kappa}_1 \bigstar \boldsymbol{\kappa}_2 \bigstar \boldsymbol{\kappa}_3 \ldots \bigstar \boldsymbol{\kappa}_a$	Vector of probabilities of lifetime reproductive success (LRS) for individuals who live to age a for $a < \beta$.
$\gamma_\beta = l(\beta) \boldsymbol{\kappa}_1 \bigstar \boldsymbol{\kappa}_2 \bigstar \boldsymbol{\kappa}_3 \ldots \bigstar \boldsymbol{\kappa}_\beta$	Vector of probabilities of LRS for individuals who live to age β and beyond.
$\Gamma = \sum_{a=1}^{a=\beta} \gamma_a$	Vector of probabilities of LRS, from summing across contributions by each age, up to and including the last reproductive age (same as Γ above).

that an individual dies at age a is the product of the probability of living to the beginning of age interval a with the conditional probability of dying during that age interval.

Reproduction for age-only and age + stage models is only possible between some specified youngest age α and an oldest age β respectively. Stage-only models may have non-reproductive stages, but such stages may occur at any age. The number of offspring produced during a single time interval is a random variable that can take on integer values $k = 0, 1, 2, \ldots, k_m$. The probability distribution of reproduction (in a single interval) depends on age ($\boldsymbol{\kappa}_a$), stage ($\boldsymbol{\kappa}_j$) or age + stage ($\boldsymbol{\kappa}_{a,j}$) respectively for the age-only, stage-only and age + stage models.

Finally, the number of offspring produced by individuals during a life-time, the LRS, is a random variable that can take on integer values $k = 0, 1, 2, \ldots, k_U$. The final goal of our analyses is always the probability distribution Γ whose elements are:

$$\Gamma(k) = \Pr[\mathrm{LRS} = k]. \tag{1}$$

Our calculations assume that, within a single time interval, reproduction precedes death. Our formulas are easily changed to suit other assumptions.

The practical flow of decisions about which of the following methods to use is shown in the Appendix S1.

Age-only model

Here, survival and reproduction during a single time interval depend only upon the age of individuals. We want the probability distribution Γ of LRS (see eqn 1).

The distribution of offspring $\boldsymbol{\kappa}_a$ at age a is taken to be binomial for age-structured human populations (so $k_m = 1$ and there are 0 or 1 offspring at each age); but our method below applies to any form of probability distribution. We break life into segments: newborns (age 1) who die before reaching age 2, those who live to age 2 but die before 3, and so on.

First, consider individuals who die before reaching age $a = 2$. The probability of such a death is $\phi_1 = l_1\,[1 - p_1]$, and reproductive success at age 1 follows the distribution $\boldsymbol{\kappa}_1$, so the LRS for such individuals has the probability distribution:

$$\gamma_1 = \phi_1\, \boldsymbol{\kappa}_1. \tag{2}$$

Next, consider individuals who reach age $a = 2$, reproduce and die before age $a = 3$. Such individuals have had two opportunities to reproduce. If these individuals have LRS = 2, they could have: (1) produced 0 offspring at age 1 and 2 offspring at age 2, or (2) produced 1 offspring at age 1 and 1 offspring at age 2, or (3) produced 2 offspring at age 1 and 0 offspring at age 2. In other words, at ages (1, 2), they could have (2, 0), (1, 1) or (0, 2) offspring. Hence the probability of LRS = 2 is $\kappa_1(2)\kappa_2(0) + \kappa_1(1)\kappa_2(1) + \kappa_1(0)\kappa_2(2)$.

Each LRS = k is analysed in this way, and the corresponding probabilities are elements of the discrete convolution:

$$\boldsymbol{\delta} = \boldsymbol{\kappa}_1 \bigstar \boldsymbol{\kappa}_2. \tag{3}$$

For more details on convolutions, see the Appendix S1. There are $(2k_m + 1) = 3$ possible outcomes resulting from this convolution. Since the proportion of a cohort that dies at age

2 is ϕ_2, the LRS for individuals in a cohort who die before reaching age $a = 3$ is distributed according to the probabilities:

$$\gamma_2 = \phi_2 \boldsymbol{\kappa}_1 \bigstar \boldsymbol{\kappa}_2. \quad (4)$$

In general then, an individual who lives to age $2 \leq a < \beta$ but dies before reaching $a + 1$, has LRS distributed as:

$$\gamma_a = \phi_a \boldsymbol{\kappa}_1 \bigstar \boldsymbol{\kappa}_2 \ldots \bigstar \boldsymbol{\kappa}_a. \quad (5)$$

Reproduction ends after age β, so survival to later ages does not change the LRS. Consequently, for an individual who lives to age $a \geq \beta$, the LRS has distribution:

$$\gamma_\beta = l_\beta \boldsymbol{\kappa}_1 \bigstar \boldsymbol{\kappa}_2 \ldots \bigstar \boldsymbol{\kappa}_\beta. \quad (6)$$

Adding, we have:

$$\Gamma = \sum_{a=1}^{a=\beta} \gamma_a. \quad (7)$$

Stage-only model

For many species, stage (such as size or developmental stage) is predictive of vital rates rather than age (Lefkovitch 1965). Survival, stage transition, and reproduction during a single time interval depend only on the stage s of individuals at the beginning of that interval.

The number of offspring (k) produced during a time interval depends only on stage j at the beginning of the time interval and is specified by probability distributions $\boldsymbol{\kappa}_j = \{\kappa_j(k)\}$ of producing $k = 0, 1, 2, \ldots$ offspring (Table 1). These distributions are often taken to be Poisson (e.g. some cases studied in vDC), but that is not a requirement of our analysis.

Stage structure requires multiple convolutions that make the age-only procedure computationally unwieldy. This is because here, in contrast to an age-structured model, individuals may visit a stage any number of times before dying. They may also move back and forth between non-reproductive and reproductive stages. So we need a fast way of (1) doing repeated convolutions, and (2) summing over repeated convolutions.

To solve (1) we use the mathematical fact that the (discrete) Fourier transform of a (discrete) convolution is the product of Fourier transforms, and the numerical fact that the FFTW package (Mersmann 2019) does a fast discrete Fourier transform. To solve (2), we exploit our earlier work (Steiner & Tuljapurkar 2012) and combine a closed-form generating function with Fourier transforms, then use an inverse FFT. For details (or if this brief description makes you curious) see the Appendix S1.

Thus we can compute the exact distribution of LRS for any stage-structured vital rates. We can separately, and quickly, compute the probability that the LRS = 0. Both analyses are given in the Appendix S1.

Age + stage model

Here survival, stage transitions and reproduction during a single time interval depend on both age and stage (e.g. Coulson 2012; Table 1 and Appendix S1). In principle, here we can proceed as with the age-structured model. But the computation depends on the 'size' of the life cycle, and determines a choice between two options. If all individuals reach a terminal age and die, and if the number of stages is modest, use the age-structured approach. If there are many ages and/or stages, and/or if all individuals reach an 'old' age with stages in which they reproduce and remain until death, the age-structured approach is too slow. In that case, use a general stage-structured approach with block matrices (see Appendix S1).

RESULTS

Age-only model

An example: the Hadza

The Hadza are a hunter-forager group thought to be representative of early humans (vDC). We consider only females with no migration. One-period survival probabilities by age, p_a, yield survivorship l_a (the probability of surviving until age a). Reproduction ceases at menopause at age $\beta \approx 50$ year, so we do not consider later ages. The commonly used fertility rate f_a is the mean number of offspring at each age. Fertility at age a can be 0 or 1 for human populations (vDC), with:

$$\begin{aligned} \kappa_a(0) &= \Pr[0 \text{ offspring} | \text{parent age } a] = (1 - f_a), \\ \kappa_a(1) &= \Pr[1 \text{ offspring} | \text{parent age } a] = f_a. \end{aligned} \quad (8)$$

For each age, these numbers constitute a distribution we denote $\boldsymbol{\kappa}_a$. We ignore twinning, which is rare in these populations [but could be included by adding the value of $\kappa_a(2)$]. For other species (e.g. if there are litters with many offspring) we need additional information to create a distribution of offspring at each age.

We used our methods to compute the exact distributions of LRS for the Hadza in Fig. 1a.

The distribution for the Hadza is dramatically bimodal, with a very high mode at 0 and another at LRS ≈ 6. These modes are very different from the mean LRS of 3.13; the spread in LRS is not described by the standard deviation (= 3.36). The third central moment (= 21.7) does imply that the distribution is skewed.

Mode at LRS = 0

In any age-structured life cycle, a female may have 0 offspring if she dies before reproduction begins, or lives on but has 0 offspring at every age. Hadza reproduction can begin at age 13 years, and the probability of dying by that age is 0.415 for Hadza. The probability of living on but childless is 0.035. So here the mode at LRS = 0 is largely determined by high infant mortality, and for any age-structured life cycle we should see a mode at LRS = 0 unless early mortality is extremely low.

Do 3 moments predict the distribution?

We can compute the LRS distribution from our methods, but can we recover a reasonable approximation from just the first 3 moments? For the Hadza, the answer is emphatically no, as shown by the Gram-Charlier approximation in Fig. 1b which was not bimodal. We tried a different method, starting with a

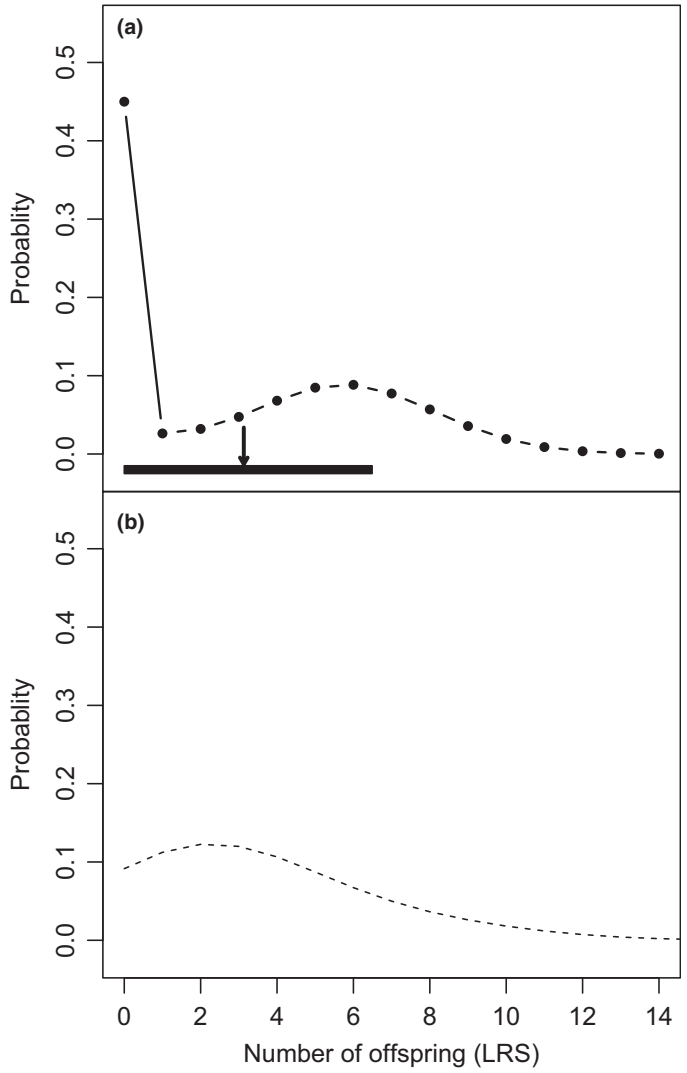

Figure 1 In (a), the exact lifetime reproductive success (LRS) distribution (dots, joined by lines to aid the eye), of the Hadza computed using only vital rates. The arrow in (a) indicates the mean LRS of Hadza at 3.13 – this is far from the mode. The horizontal bar (from 0 to 6.49) indicates an LRS range (computed as the mean LRS ± one standard deviation, with a lower bound at zero). The standard deviation does not usefully describe the variability in LRS. In (b), the dots plot a Gram-Charlier approximation to the exact curve in (a), based on exactly computed values of the first three moments.

Poisson and then using a numerical search for the 'best' modification, but the answer is still negative. We have not tried using higher moments, because they do not seem likely to change our results.

Stage-only model

An example: an evergreen tree T. canadensis

We use a stage-only model for *T. canadensis*, eastern hemlock, an evergreen tree that grows to be 40–75 feet tall in the wild (as cited in vDC). Populations of this tree, as noted by Lamar & McGraw (2005), usually include many saplings, small individuals that may remain in good condition beneath a canopy for hundreds of years. So individuals exhibit considerable stasis. The population is structured by diameter-at-breast-height (d.b.h), including six size classes (< 5.0 cm d.b.h, 5.0–10.0 cm d.b.h, 10.1–17.5 cm d.b.h, 17.6–27.5 cm d.b.h, 27.6–42.5 cm d.b.h, and > 42.5 cm d.b.h).

The LRS for *T. canadensis* has an extremely pronounced mode at 0, and the skew of the distribution is so dramatic that we had to plot the logarithms of the probabilities of LRS, as shown in Fig. 2. The average LRS is just 1.42, but the standard deviation of 37.57 is not useful as a measure of spread at either lower or higher values. The third moment is huge, $\approx 2.6 \times 10^6$, which certainly means great skew, but does not predict the actual distribution in the figure. We expect a similar skewed distribution for many species (including plants and marine organisms) that produce large numbers of offspring such as seeds or larvae, but few recruits, and even fewer adults. The skew seen in Fig. 2 is relevant to current paradigms about life history evolution and selection, as discussed in the final section.

Mode at LRS = 0

For *T. canadensis*, there is a pronounced mode at zero, with $\Gamma(0) = \Pr[\text{LRS} = 0] = 0.9883$! This number is the sum of the probability of surviving while in non-reproductive stages plus the probability of producing 0 offspring while in reproductive stages. We can always compute this total probability exactly (see Appendix S1).

For this and many other life cycles, we can also find the conditional probability:

Pr[die without reaching a stage capable of reproduction].

We always start (here) in a non-reproductive stage. Every individual that survives eventually makes an irreversible transition to one or more stages that are capable of reproducing (call these reproductive stages). For this case, it makes sense to treat the reproductive stages as 'absorbing', so we can compute the corresponding absorption probability. Then [1 − absorption probability] is just the conditional probability defined above (see Appendix S1).

The value of this conditional probability is 0.9878 for *T. canadensis*; comparing this number with 0.9883, we see that most individuals who have an LRS of zero must die without ever having entered a stage that is capable of reproduction.

Age + stage model

An example: Roe deer C. capreolus

Here an individual is classified by both age $a = 1, 2, \ldots, \omega$ and stage $s = 1, 2, \ldots, S$. We illustrate with an estimated age + stage model for Roe deer *C. capreolus* (Plard *et al.* 2015). Age is in years, and size in 200 equal body mass intervals from 1 to 44 kg. There are four age groups; in each group, there is one set of transition and survival rates and reproduction probabilities (the age groups are yearlings, adults age 2–7 year, adults age 8–11 year, adults over age 11). Age advances in single years.

The 'initial stage' is a combination of age (here, yearlings in age group 1) and size. For initial age $a = 1$ there are potentially 200 initial sizes. In practice, yearling sizes range from

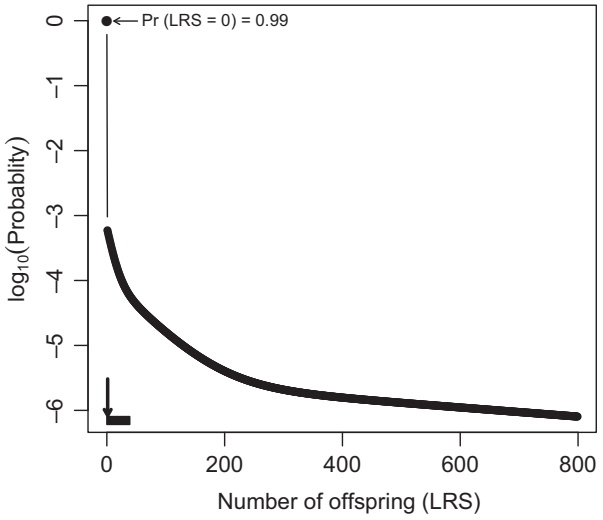

Figure 2 The exact lifetime reproductive success (LRS) distribution of *Tsuga canadensis*, eastern hemlock. Note the extreme skew and the high probability of having no LRS (indicated by the upper horizontal arrow). The lower vertical arrow indicates the mean LRS at 1.42 – even though a few trees dominate reproduction, most offspring fail to reproduce. The horizontal bar (from 0 to 38.96) indicates an LRS range (computed as the mean LRS ± one standard deviation, with a lower bound at zero). Detailed step-by-step illustration of the analysis is presented in the Appendix S1.

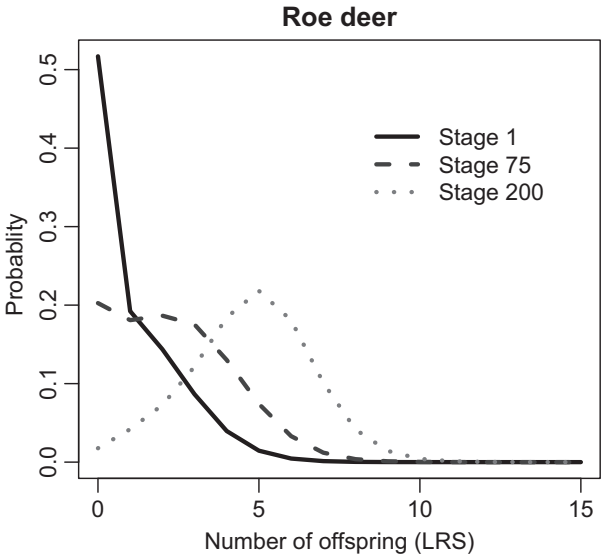

Figure 3 The lifetime reproductive success (LRS) exact distribution of yearlings of Roe deer *Capreolus capreolus* born into different initial size classes of 1 (the solid line), 75 (the dashed line) or 200 (the dotted line). For example, an individual with an initial size class of 1 will have the LRS distribution shown as the solid line. Initial size strongly influences the shape of the LRS via both juvenile survival and later fertility. Individuals born into size class 75 have an average LRS of 2.3 with a standard deviation of 1.82.

size class 40 to 100, but we analyse the full potential size range (see Appendix S1). Figure 3 plots the LRS distribution for 3 different initial size classes, 1, 75, 200. The distribution shifts from unimodal at LRS = 0 for the smallest yearlings, to unimodal at LRS $\simeq$ 5 for the largest yearlings, and can be multimodal at intermediate sizes.

Very small offspring have a high mode at 0, and thus the highest probability of leaving no offspring – no surprise. Neither the average LRS nor the standard deviation seem to be useful descriptors of the LRS distribution for the smallest yearlings (plotted in the Appendix S1). Somewhat bigger yearlings (birth size class 70–84, see Fig. 3) have a bimodal distribution of LRS. These larger juveniles have a single nonzero mode but still high odds of leaving 0 offspring. This mode shifts from an LRS $\approx$ 2 (for size class 75) to an LRS $\approx$ 5 (for size class 200) with increasing yearling size. Very large offspring have a high nonzero mode at LRS $\approx$ 5, with a normal-like LRS distribution around the mode.

Mode at LRS = 0

The height of a mode at an LRS of 0 can be computed using the age + stage method described above (see Appendix S1). This analysis reveals a potential selective advantage of birth size via both juvenile survival and later reproductive success (because the probability of nonzero fertility depends on size).

However the gain from increasing birth weight is strikingly nonlinear, so that the advantage of being born 1 kg heavier is much greater for small than for large yearlings. To dissect this effect, we analysed yearling death probability and the probability of having no offspring if you live to later ages (see Appendix S1). There is a drop in both as birth mass increases, but the gain in yearling survival reduces faster than the gain in later survival/reproduction.

DISCUSSION

We have presented an analytical method that computes the probabilities of getting every possible LRS, 0, 1, 2, 3, ... We illustrated our results for a human population for age structure, eastern hemlock for stage structure, and Roe deer for age + stage structure. We found that the LRS distribution often has one mode at LRS = 0, and a second at nonzero LRS. For several of our examples, the mean, variance and third moment are poor descriptors of the LRS distribution. The mode at zero is strongly affected in our examples by the survival probability of juveniles, as is the mode of the LRS distribution. As a result, distinct evolutionary insights (e.g. about trade-offs across the life cycle) may emerge from considering only mature individuals vs. all individuals. Inclusion of immature stages, not surprisingly, leads to an even greater variability of the LRS than expected from previous work (Tuljapurkar *et al.* 2009b; Steiner & Tuljapurkar 2012; van Daalen & Caswell 2015; Snyder & Ellner 2018) and vDC. Here we discuss extensions and implications of our results, and directions for future work.

Descriptors and sensitivity analyses

How best to describe the variability that we have found in the LRS? And given a descriptor, what are its sensitivities to vital rates? In ecology, sensitivities determine how vital rates (or underlying parameters) affect the LRS distribution, whereas

in evolution sensitivities capture selection pressures that depend on LRS (Caswell *et al.* 2018). For future analytical work, note that our stage-structured analysis applies to any age + stage combinations.

One descriptor of the inequality of LRS is a Gini index (GI; Weiner & Solbrig 1984; Shkolnikov *et al.* 2003), which corresponds to the mean of absolute differences in individual LRS relative to the average LRS, and is bounded between 0 and 1. The GI is commonly used in economics and social science to quantify inequality, but less frequently in ecology and evolution. It has been used to assess variation in the distribution of reproductive success of parasites (Dobson 1986), in the mass and fecundity distributions of a cestode infesting pikes (Shostak & Dick 1987), in the strength of forest edge effects (Matlack 1994) and in size hierarchy studies of competitive processes in plants (Weiner & Solbrig 1984). It has also been recently used to describe the shape of mortality within a population (Archer *et al.* 2018) and to quantify species differences in the distribution of reproductive ages across vertebrates (Healy *et al.* 2019). We propose that it will be valuable in analysing variation between sexes or across species in the LRS distribution. We stress that the computations here are based on knowledge of the exact distribution. In contrast, for a sample from any empirical distribution the Gini would have to be estimated, and there is a large literature relevant to that task (Atkinson 1970; Dixon *et al.* 1987; Chotikapanich & Griffiths 2002).

Here we use the exact probability distribution, define the cumulative distribution:

$$\xi(x) = \sum_{k=1}^{x} \Pr[\mathrm{LRS} = k] = \sum_{k=1}^{x} \Gamma(k),$$

and plot $\xi(x)$ vs. values (x/k_U) of the LRS (remember k_U is the largest possible LRS). This is the Lorenz plot shown for our examples in the panels of Fig. 4. Equality means that every individual has the same odds of any LRS and is indicated by the dotted lines. The actual distributions are shown by the solid lines and indicates that a few individuals do most of the reproducing.

The GI is:

$\mathrm{GI} = 2 \times$ Area between the two curves shown.

Equality means that GI = 0 while complete inequality means that GI = 1.

As we expect from our computations, the extreme inequality of LRS for *T. canadensis* is reflected in a very high GI. Surprisingly the LRS for a fairly large Roe deer yearling (see Fig. 4) is also quite unequal.

Of course, the utility of the Gini may be limited by accuracy of estimation, and the ecological implications of different Gini values need further study. A more general approach to the sensitivity of inequality in LRS should be feasible using our results in conjunction with the methods in van Raalte & Caswell (2013) and Caswell *et al.* (2018). More generally, numerical perturbations can be used to examine changes in Γ produced by changes in vital rates, and we can compare distributions using, e.g. the Kullback–Leibler distance (Cover & Thomas 2012).

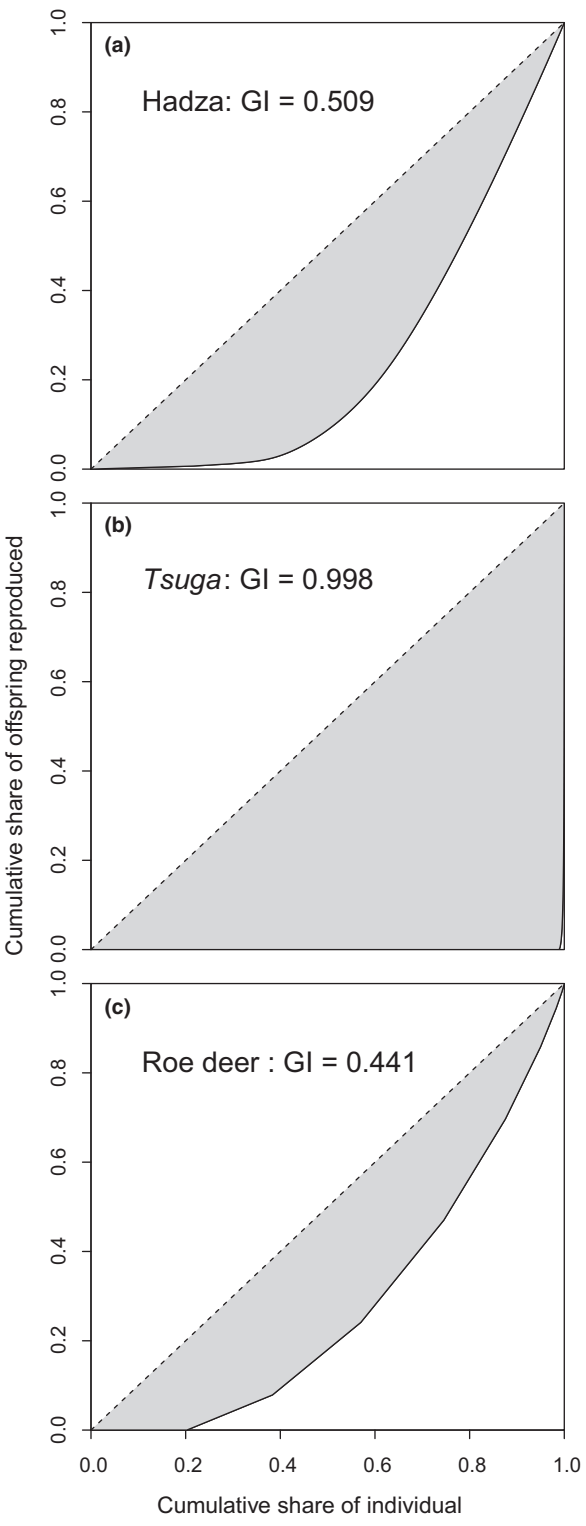

Figure 4 The vertical axis is a cumulative probability for the lifetime reproductive success (LRS), and the horizontal axis is simply the cumulative fraction of a starting cohort. The resulting Lorenz curve would lie along the diagonal if all individuals performed equally, and would lie close to the horizontal axis for high inequality (only a very few have high LRS). Panel (a) shows the Hadza. Panel (b) shows the dramatic and extreme inequality for *Tsuga canadensis*. Panel (c) for Roe deer born into initial stage 75 shows smaller inequality than the Hadza, as expected from Fig. 3. GI, Gini index.

A useful measure highlighted here is the probability of having no reproductive success:

$$\Pr[\mathrm{LRS} = 0] = \Gamma(0).$$

Derivatives of this probability with respect to these parameters should be computable using known approaches, e.g. Caswell *et al*. (2018) or Steiner *et al*. (2012).

Another useful measure is the nonzero modal value, call it k^*, of the LRS – if it exists. We found a nonzero mode in several examples, but there is no nonzero mode for the tree (*T. canadensis*). Assuming its existence, a nonzero mode means that:

$$\Gamma(k^* - 1) < \Gamma(k^*) > \Gamma(k^* + 1).$$

This modal value may be more meaningful in some populations than the commonly used average LRS. We can use our result to find k^* and the methods in Caswell *et al*. (2018) should be extendable to compute sensitivities.

Implications

In our analysis and examples, each individual is governed by the same set of vital rates (probabilities). In other words, all individuals have the same chances of surviving and reproducing at each age. Yet, the distribution of LRS reveals how much heterogeneity can be generated by chance alone. This variation caused Steiner & Tuljapurkar (2012) to conclude that entirely neutral processes could generate the multimodal distributions of LRS so frequently observed.

In stage- and age + stage-structured models individuals that start life with different phenotypic trait values can have contrasting distributions of LRS. For each phenotype there is considerable individual heterogeneity in LRS, but in addition individuals with different phenotypes have differing LRS distributions. These differences can generate opportunities for selection. Whether such selection would result in evolution will depend upon whether there is a genetic basis for individuals entering the population with different phenotypes.

Stage- and age + stage-structured models are powerful. They can be constructed for stochastic, density-dependent and frequency-dependent environments to predict population (Tuljapurkar 1990), life history (Caswell 2001b; Tuljapurkar *et al*. 2009a) and evolutionary dynamics (Coulson *et al*. 2011, 2017; Childs *et al*. 2016). A large number of ecological and evolutionarily important statistics can be calculated from these models, with analysis of these models revealing how different values of these statistics arise. By extending the toolkit of structured models to predict entire distributions of LRS we open the door for a number of powerful, new analyses. For example, we can ask how different growth trajectories will impact distributions of LRS, we can gain insight into why weak associations between birth weight and LRS are so frequently observed, and the conditions when lifetime measures of reproduction act as a good, or bad, surrogates of fitness.

Previous analyses of LRS distributions in vertebrates using the standardized variance have supported the existence of a link between mating systems and sex differences in LRS distributions, with similar distributions in males and females of monogamous species and much more widespread and skewed distributions in males than in females in highly polygynous species (Andersson 1994). In addition to sex, our approach provides a suitable way to assess how ecological factors (e.g. environmental harshness), life history tactics (e.g. slow vs. fast life cycle, capital vs. income breeding), and population structure (e.g. in terms of age, size) influence LRS distributions within and among populations. Finally, our approach should allow accurate comparisons among LRS distributions that are currently accumulating thanks to long-term individual-based monitoring of populations in a broad range of species across the tree of life.

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AUTHORSHIP

ST conceived the project and performed many of the analyses; WZ did some analysis and all computations; TC, CH and JMG contributed to the ideas, text, data and discussion.

DATA AVAILABILITY STATEMENT

Only previously published data were used in this research.

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