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Heritability of individual fitness in female macaques

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Abstract Heritability of fitness is an important parameter for evolutionary studies, but it is controversial and difficult to estimate this quantitative genetic statistic. I compare two single-generation proxies of individual fitness estimated from demographic information (lifetime reproductive success, LRS; and individual finite rate of increase, individual λ) and lifespan for the female members of a free-ranging population of rhesus macaques (*Macaca mulatta*). All three variables have moderate heritabilities ($\lambda = 0.36$, LRS $= 0.38$, lifespan $= 0.43$) that are consistently depressed when non-reproductive individuals are censored from the analysis. This reduction suggests a large portion of the genetic variation in the fitness proxies is due to survival to reproductive age and commencement of reproduction in this population. This may be related to relatively benign, homogeneous environmental conditions. Any time gaps in modeling an animal's life cycle can introduce similar inaccuracies in heritability of fitness proxies, although the direction of error is likely to vary with environmental conditions. Genetic correlations between the three variables were all indistinguishable from $+1$ implying no independent genetic variation. The similarity of heritability estimates for λ and LRS and strong genetic correlations are attributed to the dominance of adult lifespan in determining fitness for female macaques which are slow-reproducing by mammalian standards. While the heritabilities of both proxies were similar in this study, they should both be estimated when possible because they may provide different information, particularly in taxa with larger broods.

Keywords Quantitative genetics · Lifetime reproductive success · Individual λ · Primate · Fertility · Lifespan

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Introduction

The genetic underpinnings of fitness have been subject to intense theoretical scrutiny in a wide variety of areas of evolutionary biology and animal breeding (Kruuk et al. 2008; Roff 2007; Merilä and Sheldon 1999, 2000; Barton and Turelli 1989; Fisher 1930). However, empirical studies reporting the heritability of single-generation proxies of individual fitness estimated from demographic information are rare, though recent methodological advancements are improving this situation across many taxonomic groups (Kruuk et al. 2008; Kruuk 2004). Nevertheless, studies of heritability of fitness and fitness components remain rare for humans and non-human primates (Pettay et al. 2005; Madrigal et al. 2003; Kirk et al. 2001; Martin et al. 2002; Blomquist 2009).

Two single-generation proxies for individual fitness have been used (Brommer et al. 2002), although there is no consensus on what the best way to measure individual fitness is or even if such a metric is possible despite agreement on invasability as the master theoretical criterion of fitness (Roff 2008; Brommer 2000; Link et al. 2002; Grafen 1988). Lifetime reproductive success (LRS, total number of offspring or total surviving to independence) is the most intuitive definition and has a long history (e.g. Clutton-Brock 1988). More recently, a measure sensitive to the age-schedule over which offspring are produced (λ_{ind}) has been proposed though it is less widely used. λ_{ind} is similar to the finite rate of increase of a population in demographic studies but is calculated as the dominant eigenvalue of an individual projection matrix (McGraw and Caswell 1996). For studies of phenotypic selection, including age-schedule of reproduction in the fitness measure will emphasize selection on early life traits, such as age of first reproduction (Oli et al. 2002; Kaar and Jokela 1998; Korpelainen 2003). The importance of including age-schedule, or at least the manner in which λ_{ind} discounts reproduction at later ages, has been questioned (Brommer et al. 2002, 2004; Grafen 1988).

Comparisons of heritabilities and variance components for these two proxies in the same populations are rare (Pettay et al. 2005). The discounting of reproduction at later ages reduces the phenotypic variance in λ_{ind} relative to LRS in most settings. However, it remains unclear how reduced phenotypic variance and emphasis on early life events for λ_{ind} are translated into additive genetic and remaining variance components that will determine evolutionary dynamics.

An additional problem in measuring individual fitness is defining the appropriate age to count offspring (Brown 1988; Grafen 1988; Wolf and Wade 2001; Wilson et al. 2005). While many population genetic models identify individuals as zygotes or newborns (Cheverud and Moore 1994), it is common in ecological studies to count offspring only when they have reached independence from parental care or begin reproducing themselves (Clutton-Brock 1988; Korpelainen 2000a). In either case a full life cycle is modeled—newborns to newborns or parents to potential parents. The large number of newborns that die before ever reproducing, and consequently have zero LRS or λ_{ind}, are a difficulty for the first method of counting offspring. Eliminating them from analysis introduces a gap in the life cycle which will reduce the phenotypic variance of either fitness proxy and potentially affect heritability estimates. Simulation studies in animal breeding suggest that this censoring of poorly performing individuals can cause depressed estimates of additive genetic variance and heritabilities (Burns et al. 2006; Vukasinovic et al. 1999). Theoretical work based on missing data analysis also predicts biased heritability estimates when records are not missing completely at random (Hadfield 2008; Nakagawa and Freckleton 2008; Im et al. 1989).

Here, I compare the quantitative genetics of LRS and λ_{ind} for the female members of a free-ranging population of rhesus macaques (*Macaca mulatta*). I also explore the effect of censoring females with low LRS on the heritability of these fitness proxies. Because LRS is strongly predicted by lifespan in female primates (Heppell et al. 2000; Blomquist et al. 2009) and more comparative information is available on heritability of lifespan than the fitness proxies, I also include it in the analysis. Previous work with this population showed that heritabilities of LRS and lifespan were moderate, but declined substantially when non-reproductive females were omitted, though the biological and/or methodological reasons behind this were not explored and different statistical models from those used here were employed (Blomquist 2009). Similar declines for heritability of λ_{ind} are predicted. However, because λ_{ind} is more strongly affected by early life events it is also expected that censoring non-reproductive individuals will have a larger effect on its heritability than LRS or lifespan. In addition, I calculate genetic correlations between LRS, λ_{ind}, and lifespan to clarify the extent of shared genetic variation between these traits. All genetic correlations are expected to be very high, though they may be significantly less than one-implying independent genetic variation affecting each variable. Correlations are calculated from both the censored and uncensored data sets.

Materials and methods

I used individual female life history data from the demographic database of a large population of free-ranging rhesus macaques that were transplanted to the 15.2 ha island of Cayo Santiago, Puerto Rico from India in 1938. Monkeys are fed commercial monkey chow and provided water ad libitum but forage on natural vegetation and live in naturally formed social groups where matings and other interactions are unmanaged. The population has been managed by removal of social groups and, more recently, by a random cull of juveniles (Rawlins and Kessler 1986; Sade et al. 1985).

Lifespan in years, LRS, and λ_{ind} were calculated for females that were reliably aged (born after 1959), had no evidence of pathology, and were born prior to 1990 such that the data from recent birth cohorts would not be heavily biased to individuals dying at young ages. Less than 10% of females from cohorts where all members were dead live beyond the maximum attainable for the 1989 cohort (15 years). While the demographic database contained nearly 8,000 individuals at the time of this study, only 590 individuals were females from the desired birth cohorts that died of natural causes. Forty females from these cohorts were still alive and 1,209 had been removed from the population. Finally, 106 females were dropped because they had no pedigree connection to other females with phenotypic data, and three were omitted because they lived to late ages without ever reproducing suggesting potential pathology and making them clear bivariate outliers for lifespan–LRS and lifespan–λ_{ind} correlations. Of the remaining 590 females with natural deaths only 278 reproduced (Table 1). These females provide the uncensored (LRS $\geq$ 0) and censored sets (LRS $\geq$ 1) of fitness proxies and lifespan.

LRS is simply the total number of offspring born to a female regardless of offspring sex and fate, while λ_{ind} was calculated as the dominant eigenvalue of the individual projection matrix following standard techniques (McGraw and Caswell 1996; Brommer et al. 2002). Each offspring, was counted as a 0.5 in the projection matrix. Eigenvalues were calculated in R (R Development Core Team 2007). None of the variables are normally distributed (Fig. 1). This does not seriously compromise the quantitative genetic techniques used (Kruuk 2004; Shaw 1987), but a dichotomous reproduction variable (d. rep.) was used to

Table 1 Summary statistics and heritabilities for Cayo Santiago female individual fitness proxies and lifespan

Variable	*N*	Min.	Max.	Mean	SD	$h^2 \pm$ SE	*P*
d. rep.	590	0.000	1.000	0.471	0.500	0.274 ± 0.103[a]	0.004
λ_{ind}	590	0.000	1.272	0.524	0.563	0.358 ± 0.101	<0.001
	278	0.794	1.272	1.112	0.131	0.145 ± 0.161	0.687
LRS	590	0.000	17.000	3.122	4.538	0.378 ± 0.081	<0.001
	278	1.000	17.000	6.626	4.526	0.205 ± 0.134	0.072
Lifespan	590	0.000	31.422	6.313	6.745	0.425 ± 0.080	<0.001
	278	3.118	31.422	11.893	5.932	0.269 ± 0.130	0.020

[a] On underlying liability scale, d. rep. $h^2 = 0.431 \pm 0.162$

address concerns about the normality of LRS and λ_{ind}. For this variable, reproduction was considered a threshold trait taking values of zero, for LRS < 1, and one for LRS ≥ 1. Furthermore, analysis of d. rep. provides a direct estimate for the heritability of survival to sufficient age and commencement of reproduction. This indirectly suggests how much the heritabilities for the fitness proxies may change due to censoring non-reproductive individuals. As the heritability of d. rep. increases, so should difference between uncensored and censored fitness proxy heritabilities.

Variance components and related quantitative genetic statistics were estimated using an "animal model" (Kruuk 2004) in WOMBAT (Meyer 2007), relying on the complex pedigree relationships of the study population which included paternities determined by microsatellite variation (Nurnberg et al. 1998). Of the 590 females in the analysis 95.9% were members of a single pedigree containing eight generations of monkeys, while the remaining 4.1% were members of smaller pedigrees of 3–7 females with phenotypic data. For the 278 reproductive females these respective percentages were 96.4% in the large pedigree and 3.6% in pedigrees where two females had phenotypic data. Maternal pedigree links greatly outnumber paternal links (25 times more common for the set of 590 females in the uncensored set, 12 times for the 278 in the censored set), which will hinders separation of additive genetic and maternal effects. The mean coefficient of relatedness for females with phenotypic data in the uncensored set was 0.00521; it was 0.00863 for the reproductive female set. Inbreeding was only detected for one individual used in both sets ($F = 0.25$). Coefficients of relatedness and inbreeding are underestimates because so few paternities were known.

Five random effects were present in univariate animal models used to decompose phenotypic variance (V_P) of each fitness proxy. Aside from the phenotypic mean, no fixed effects were included. Residual and additive genetic (animal) effects were included in all models. Social group at birth, birth cohort and maternal identity were included individually and in combination to control for inter-group variation, cohort effects resulting from temporal changes in the population, and maternal effects. Assuming these variance components are uncorrelated, the phenotypic variance (V_P) is the sum of the five variance components, where subscripts C, M, G, A, and R indicate birth cohort, maternal, social group, additive genetic and residual variance, respectively.

$$V_P = V_C + V_M + V_G + V_A + V_R \quad (1)$$

Heritabilities were calculated as ratios of additive genetic and total phenotypic variance ($h^2 = V_A/V_P$). Heritability of the dichotomous reproduction variable was also transformed

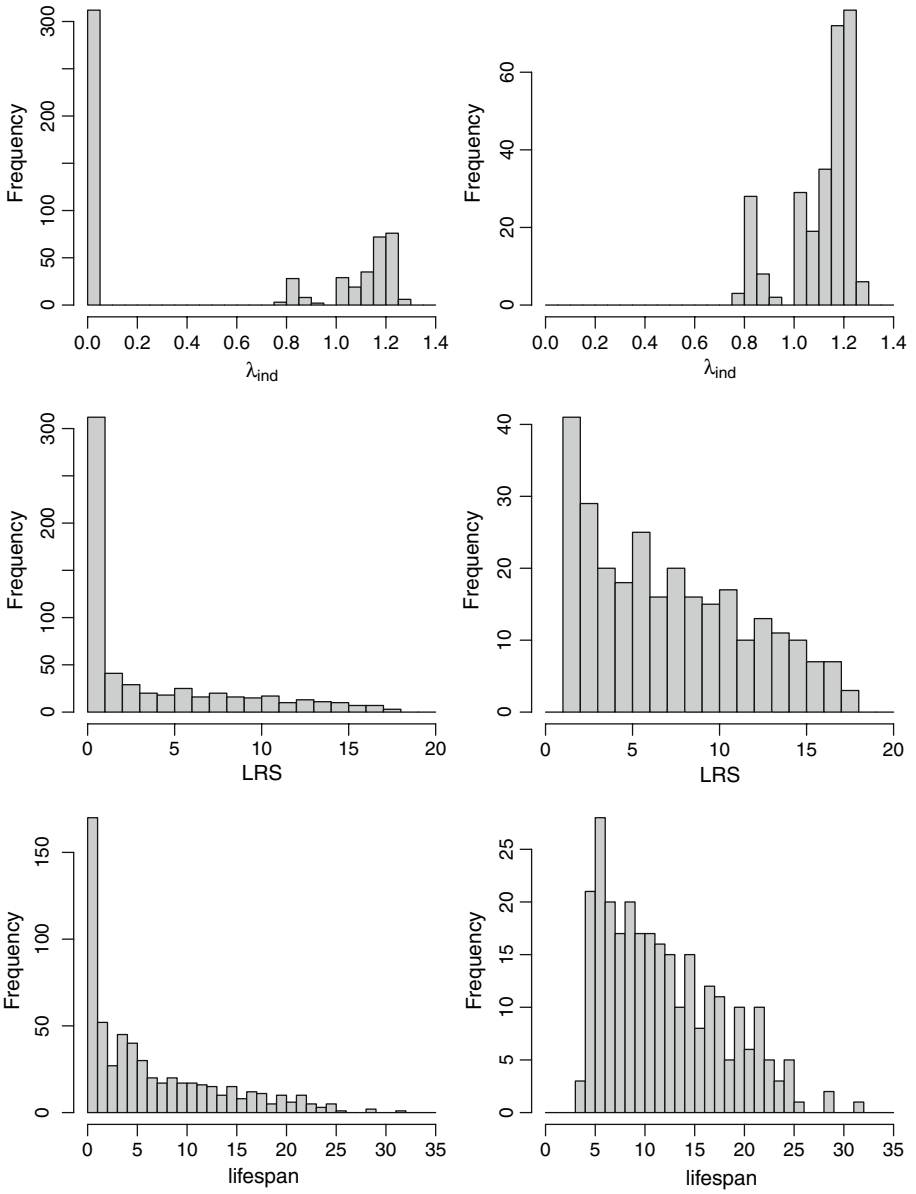

Fig. 1 Distributions of fitness proxies for Cayo Santiago female macaques. *Left column* is uncensored (LRS $\geq$ 0, $N = 590$), *right column* is only reproductive females (LRS $\geq$ 1, $N = 278$)

to the underlying liability scale following Roff (1997, p. 55). Significant difference of the heritability from zero was determined by likelihood ratio tests (Lynch and Walsh 1998) comparing the full model containing the additive genetic term and one dropping it. Twice the difference between the log-likelihoods of these models yields a χ^2-statistic with one degree of freedom. Significant difference between heritabilities estimated from the

uncensored and censored data sets was tested as $t = (h_1^2 - h_2^2)/[\mathrm{SE}(h_1^2) + \mathrm{SE}(h_2^2)]$ with degrees of freedom equal to the mean number of females in each data set (Ruxton 2006).

Results including all five parameters are used below despite little statistical support for keeping cohort and maternal effects in many models, gaged by comparing AIC among full and reduced models (Burnham and Anderson 2002). This is done to ensure heritabilities are not inflated by dropping any of these factors. In general, heritabilities were consistent between full and reduced models (h^2 from reduced models reported in Supplementary material).

Six bivariate versions of the full five-parameter model were also run to explore quantitative genetic relationships between the fitness proxies. In addition to the statistics calculated from univariate models, these provide estimates of cohort, maternal, group, additive genetic, and residual covariances or correlations between the proxies. Two *t*-tests were performed on the ratio of the additive genetic correlation (r_A) to its standard error to identify whether it was significantly greater than zero or significantly less than one. These test are only reported for the genetic correlation but summary output of the bivariate models is provided in the Supplementary material. All *t*-tests used for significance testing on heritability differences and genetic correlations may be prone to Type I errors (anti-conservative), and should be regarded with some caution.

Results

Heritabilities of the two fitness proxies and lifespan consistently declined with censoring of non-reproductive females (Table 1; Fig. 2). Each uncensored variable had a moderate heritability between 36 and 43%, all of which were within a standard error of each other (SE range 0.08–0.10). The censored variables had lower heritabilities with larger standard errors (h^2 range 0.15–0.27). While all of the uncensored variables had additive genetic

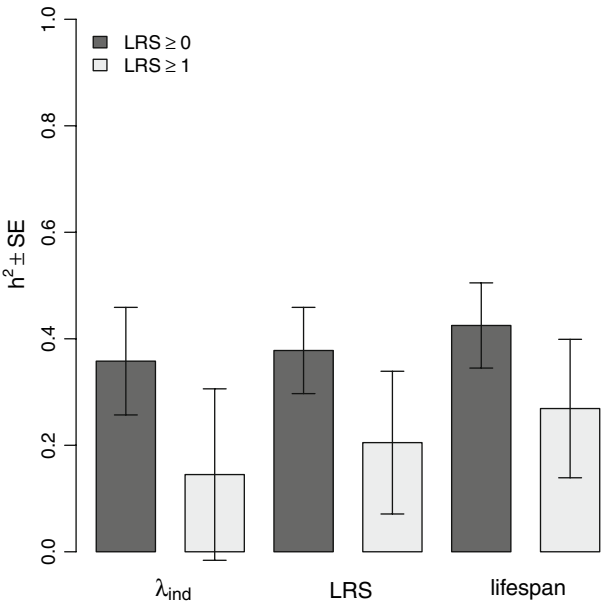

Fig. 2 Comparison of heritability estimates for individual fitness proxies and lifespan of Cayo Santiago females. *Dark grey bars* are for the uncensored set (LRS $\geq$ 0), while the *lighter bars* are for reproductive females only (LRS $\geq$ 1)

Table 2 Phenotypic (r_P) and genetic correlations (r_A) with standard errors between fitness proxies and lifespan in Cayo Santiago female macaques

Variable pair	r_P	$r_A \pm$ SE
Uncensored, $N = 590$		
λ_{ind}–LRS	0.809	1.000 ± 0.039
λ_{ind}–lifespan	0.843	1.000 ± 0.037
LRS–lifespan	0.963	1.000 ± 0.008
Censored, $N = 278$		
λ_{ind}–LRS	0.812	0.999 ± –[a]
λ_{ind}–lifespan	0.743	0.999 ± 0.605
LRS–lifespan	0.953	1.000 ± 0.028

All genetic correlations with standard errors reported were significantly greater than zero ($P < 0.05$) and not significantly less than one ($P \approx 0.5$)

[a] WOMBAT could not calculate this standard error

variance significantly greater than zero ($P < 0.001$), only the lifespan heritability was significant in the censored set ($P < 0.05$), while that of LRS was nearly so ($P = 0.07$). Heritability of the dichotomous reproduction variable was consistent with estimates for other uncensored variables (observed scale 0.27 ± 0.10, underlying scale 0.43 ± 0.16). While censoring caused declines in heritability estimates for each variable, none of these differences reaches statistical significance ($t < 0.82$, $P > 0.21$).

Maternal effects were estimated to be near zero in all the full models (range 0–0.018). However, this must be interpreted cautiously because the small number of paternal links in the pedigrees limits the separation of maternal and additive genetic variance. Cohort and particularly social group effects were larger for the uncensored data set than the censored records but always less than 0.08 and 0.15, respectively (detailed results in the Supplementary material).

Phenotypic and genetic correlations between the fitness proxies and lifespan are all very strong and close to +1 (Table 2). All three genetic correlations among uncensored variables are estimated at 1 ± 0.039. In the censored data set, correlations all remain high but standard errors increase where they could be estimated. In all cases the genetic correlations are significantly greater than zero ($P > 0.05$) by not significantly less than one ($P \approx 0.5$). These high correlations imply there is little, if any, independent genetic variation for these three variables in female macaques.

Discussion

Heritability estimates for λ_{ind}, LRS, and lifespan of female macaques were very similar in the study population. Estimates for all three variables were moderate (h^2, 0.36–0.43) and consistently depressed by censoring non-reproductive females (h^2, 0.15–0.27). Genetic correlations between the variables were indistinguishable from one, suggesting both fitness proxies and lifespan all measure essentially the same genetic trait for female macaques. The simplest explanation for the relatively high heritability of fitness is relatively homogeneous, benign ecological conditions of abundant food, no predation, and little disease for this free-ranging population of macaques (Charmantier and Garant 2005; Blomquist 2009).

Few comparable cases of censoring effects on quantitative genetic statistics can be found in the literature and all are for lifespan measures. Burns et al. (2006) found increased censoring depressed heritability estimates in a simulation study of racehorse longevity. The more data censored (10–25%) the more depressed their heritability estimate was (11–31%). Similar to the female macaque case discussed here, the individuals censored were not selected at random, but were the poorer performing individuals. Random censoring of records had no effect on the heritability.

Empirical studies also illustrate censoring effects on lifespan heritabilities but suggest its impact may be heterogeneous. In a lab study of lifespan in mice, Klebanov et al. (2001) found censoring individuals dying at young ages depressed heritability for some experimental crosses and not others. Crosses with high heritability either declined with more stringent censoring or remained stable. Similarly, when comparing heritabilities of British aristocrats and rural Finns, Korpelainen (2000b) found that excluding individuals dying under age 40 depressed heritability estimates for the Finns but not the British. Unfortunately, there is little comparative information on heritability of total lifespan, in part because studies of the genetics of lifespan are often designed to research aging, where subadult mortality is deliberately ignored (Westendorp and Kirkwood 1998; Martin et al. 2002; Wilson et al. 2008).

Taken together, these results suggest a biological interpretation of depression of heritability of fitness or lifespan due to censoring. Estimates can be depressed when there is high additive genetic variance between individuals near the left hand, or poorly performing, end of the variable distributions (Fig. 1). While environmental factors contributing to early life death are often emphasized (e.g. disease, predation, injury, maternal death), clearly genetic differences among young individuals can also reduce their chances of surviving to maturity and commencing reproduction (Ralls and Ballou 1982; Rosano et al. 2000; Charpentier et al. 2006). In circumstances of relatively benign environments (such as Cayo Santiago), genetic sources of variation may contribute more to early deaths or failure to reproduce—resulting in moderate heritabilities for uncensored fitness proxies and lifespan (Charmantier and Garant 2005; Blomquist 2009).

While it was initially expected that the heritability of λ_{ind} would decline more than LRS with censoring, because it is affected more by early life events, this was not the case. Heritability estimates of all three variables were remarkably consistent in the censored and uncensored datasets and they had genetic correlations that were never significantly different from one. Because female macaques reproduce slowly, with 1–2 year inter-birth intervals between singleton offspring starting when they are 3–5 years old, there is little difference in ranking of females by LRS and λ_{ind}, and most of the variation in both variables is explained by lifespan (phenotypic correlations in Table 2). For other species with larger litters and more rapid reproduction LRS and λ_{ind} may rank individuals differently allowing them to have independent genetic variation.

Currently, more quantitative genetic comparisons of LRS and λ_{ind} are needed in naturalistic settings. Comparisons between these fitness proxies may reveal differences in the age-specific expression of genetic variance of fitness, although models of age-specific survival and fertility—including random regression animal model techniques—are more informative for this purpose (Wilson et al. 2008; Pettay et al. 2008; McCleery et al. 2008; Brommer et al. 2007; Charmantier et al. 2006). This is an alternative or complementary interpretation of higher heritability of λ_{ind} than LRS focusing on number of loci that affect each trait (Pettay et al. 2005; Houle 1998). Regardless of whether λ_{ind} is considered an appropriate fitness proxy for a population to explore selection, it should be reported when

possible because of different phenotypic and potentially quantitative genetic information it provides.

Parental effects could inflate the heritability estimates for these variables, especially the uncensored values, such that the decline with censoring is actually the reduction of parental variance rather than additive genetic variance (Kruuk 2004; Räsänen and Kruuk 2007). However, maternal effects on fitness proxies and lifespan were estimated to be very small in this macaque population, although the small number of paternal links in the pedigrees limits interpretation of these results. Previous estimates of heritability of lifespan and LRS for this population using a smaller data set with maternal dominance rank as a fixed effect demonstrated similar declines with censoring (Blomquist 2009). Maternal effects themselves may also be reduced in good environmental conditions (Charmantier and Garant 2005), such as those that these macaques experience. Nevertheless, the potential role of maternal effects confounding the heritability declines with censoring documented here should be addressed in a population with better pedigree resolution. Additional sources of genetic variance, such as dominance and epistasis, are unlikely to influence the heritabilities compared here because of the low number of full sibships where paternities are known. Although they may be involved in the genetic architecture of these traits more generally, dominance and epistatic variance likely only contributed to the residual in this analysis (Hughes et al. 2002; Merilä and Sheldon 1999).

Counting offspring toward LRS and λ_{ind} when they become reproductive adults eliminates some of the potential problems in estimating heritabilities caused by confounding parental effects and record censoring (Wilson et al. 2005; Wolf and Wade 2001). In this context, lifespans are only meaningful for adults because sub-adult survival is interpreted as a component of parental reproduction mediating between total fertility and LRS or λ_{ind} (e.g. Brommer et al. 2004; Pettay et al. 2005). As such, heritabilities for lifespan are less likely to be influenced by parental effects but they may still influence LRS or λ_{ind} through their potential contribution to offspring survival or commencement of reproduction. Censoring can be problematic when there are individuals of reproductive age (contributing to their parent's LRS and λ_{ind}) that fail to reproduce (have zero fitness themselves). Not including these individuals may depress heritability estimates if there are genetic reasons for reproductive failure. Including these adult non-reproducers will increase phenotypic variance and exacerbate the non-normality of the fitness proxies, particularly λ_{ind}. Treating reproduction as a dichotomous threshold trait can allay some concerns about the impact of departures from normality on heritability estimates. Generalized linear models and Markov Chain Monte Carlo techniques are also attractive options for future analysis of these types of variables (Sorensen 2009; Damgaard and Korsgaard 2006a, b).

Similarly, for many field populations ecologists use a larger separation in time between offspring being counted towards parental fitness and reaching adulthood (Brommer et al. 2002, 2004). Death within this period often cannot be distinguished from migration out of or failure to return to the study area. Under this scheme, heritability of the fitness proxies and adult lifespan will not be affected by genetic variation during this gap period. While this may be desirable, it implies that part of the life cycle of the organism is not being modeled and any genetic variation expressed in this period will be ignored. In other words there are no records to censor that could potentially affect heritability of LRS or λ_{ind} when counting offspring at independence, but these variables, and their heritability estimates, may not accurately reflect heritability of LRS and λ_{ind} when counting offspring at adulthood, which are the more desirable values because they represent complete life cycles. This is analogous to the deliberate record censoring explored here with female macaques,

where newborns produce newborns in the life cycle model and censoring omits some portion of this life cycle.

While this analysis has focused on estimating heritability of fitness, censoring problems can also affect other traits that are only measured in adults such as age of first reproduction, annual fecundity, and adult morphology or behavior (Hadfield 2008). These potential adult characteristics of individuals that cannot be measured as adults are often referred to as the "invisible fraction" (Grafen 1988). Genetic or residual variation for adult traits within the invisible fraction could similarly raise or lower the heritability of many traits aside from fitness or lifespan, which alone will alter predictions of their evolutionary dynamics. Similarly, estimating the genetic covariance between a fitness proxy and an adult trait, which is a desirable way to gage selection on the adult trait and predict inter-generational response (van Tienderen and de Jong 1994; Hadfield 2008), will be affected by the necessary censoring of fitness.

Which individual fitness proxies to use and when to count the offspring that index parental fitness are difficult questions that can only be answered with knowledge of the ecology of the organisms under investigation, the data available on their individual life histories, and the costs and benefits of different methodologies (Brommer et al. 2002; Wolf and Wade 2001; Wilson et al. 2005; Wilson 2008). In this study, female macaque newborns were treated as independent individuals with lifespans and fitness proxies of their own (Cheverud and Moore 1994). Heritabilities for these variables were moderate. Censoring individuals that failed to reproduce, effectively omitting survival to reproductive age and commencement of reproduction from the modeled portions of the life cycle, lowered heritabilities of all three variables. Counting offspring at later ages can avoid this concern about censoring non-reproductive individuals, but any gaps in the life cycle create an identical problem. Omission of portions of the life cycle will have varying effects on heritability estimates that depend on the amount of genetic and environmental variation that contribute to variation during that portion of the lifetime. For female macaques in a relatively homogeneous, benign environment a significant fraction of the phenotypic variation in survival to reproductive age and commencement of reproduction appears to be genetic, but this is unlikely in harsher environments.

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