Chapter 20
Quantitative Genetic Perspectives on Female Macaque Life Histories

Heritability, Plasticity, and Trade-Offs

Gregory E. Blomquist

20.1 Introduction

All life histories are a complex of trade-offs that depend on individual conditions—including size, age, and sex—and adaptively mold suites of traits through the biased intergenerational transmission of successful physiological strategies. I explore intrapopulation variation in how female rhesus macaques on Cayo Santiago have negotiated a central life history trade-off: when to start reproducing. I emphasize how evolutionary quantitative genetic models require explicit links between hypothesized or measured patterns of selection and the genetic substrates that influence phenotypes and change intergenerationally. Perhaps counterintuitively, I also show how genetic models offer valuable insights on how environments, including those provided by mothers and other kin, affect offspring development and later female life histories. The emerging picture of female macaque maturation is one of the great flexibility and environmental responsiveness coupled with an important genetic component that is significantly entangled with later life history events.

Studies of mothers and infants have been central to understanding primate life histories. Investigators from a diverse set of disciplines and theoretical backgrounds have used a variety of approaches and scales of analysis to explore the behavioral and physiological details of this intense relationship, its life-long and intergenerational consequences, and ultimately how such patterns could be honed into adaptations by selection on genetic variation. Historically, the field has been dominated by laboratory-based experimental approaches, interspecific comparative analysis, and ethological studies in the wild (Altmann 1980; Hrdy 1999).

G.E. Blomquist (✉)
Department of Anthropology, University of Missouri,
107 Swallow Hall, Columbia, MO 65211, USA
e-mail: blomquistg@missouri.edu

K.B.H. Clancy et al. (eds.), *Building Babies: Primate Development in Proximate and Ultimate Perspective*, Developments in Primatology: Progress and Prospects 37,
DOI 10.1007/978-1-4614-4060-4_20,

In this chapter, I try to make the case for quantitative genetics, which is an underutilized method for studying primate mothers and infants. Quantitative genetics is a set of statistical techniques (described briefly below), but it is also a body of theory that links very directly with rich evolutionary population genetic models that incorporate and formally arrange information on variation in interesting maternal or infant traits and their fitness consequences (Arnold 1994; Lande 1982). Laboratory and wild studies of mothers and infants provide information on the styles of interactions, their social and ecological correlates, and sometimes how this variation translates into predictable fitness differences that result in selection in action. Interspecific comparative studies suggest adaptive patterns that may have resulted from many generations of past selection. However, none of these techniques provide the requisite information for a realistic evolutionary model about genetic variation or covariation in traits (Cheverud and Moore 1994). Quantitative genetic methods are thus complementary to the other more common approaches for studying the ecology and evolution of mother–infant interactions. In what follows, I provide a brief introduction to quantitative genetics tailored to thinking about primate life histories and then offer a handful of examples from research on female rhesus macaques (*Macaca mulatta*).

20.2 Quantitative Genetics

The core tenet of quantitative genetics is that kin should resemble one another phenotypically because they have copies of the same alleles. These alleles in common are said to be identical by descent because the copies are made by DNA replication during gamete production and transmitted across generations in fertilization. The number of genes involved and where they are located in the genome are usually unknown, although if molecular data are available they can be used to map genes that affect a given phenotype (Visscher et al. 2008). Instead, the standard assumption is that the trait is polygenic—there is a very large number of genes whose summed action results in a continuous distribution of genotypes (Fig. 20.1). The statistical match between phenotypic resemblance and predictions from rules of Mendelian inheritance (e.g., mother and infant have 1/2 of their alleles identical by descent, half siblings on average have 1/4 of their alleles identical by descent) is then used to partition phenotypic variance into genetic and nongenetic sources.

Quantitative genetics is intimately tied into the origin of statistics in the late nineteenth and early twentieth centuries (Roff 1997; Kevles 1985; Provine 1971). Galton, Pearson, and Fisher invented regression, correlation, and analysis of variance in part to measure resemblance among kin and perform this partitioning of variance. A hypothetical example of this process is shown in Fig. 20.2 where birth weights of daughters are regressed on maternal birth weights and total phenotypic variance (V_P) is broken down into additive genetic (V_A) and residual variance (V_R). The sum of the genetic and residual components is the phenotypic variance ($V_P = V_A + V_R$).

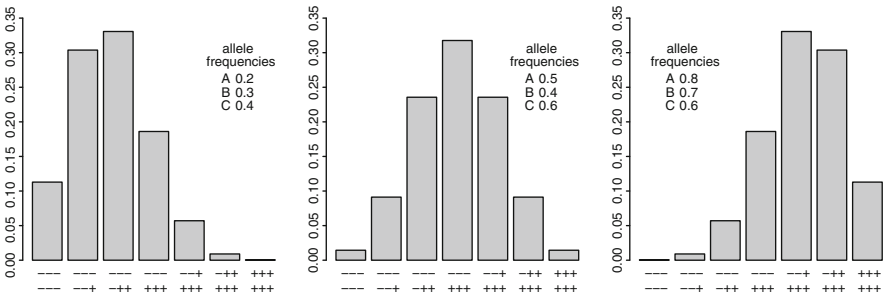

Fig. 20.1 Frequency distributions of genotypes for a polygenic trait, such as birth weight, with two additively acting alleles at each of three loci (A, B, and C). Alleles are indicated by a + (representing A, B, or C alleles) that causes slight increase in birth weight, and an alternative - (representing a, b, or c alleles) that causes reduction in birth weight. Larger infants are to the *right* in each panel. Changes in the frequencies of the alleles shift the average birth weight in the population

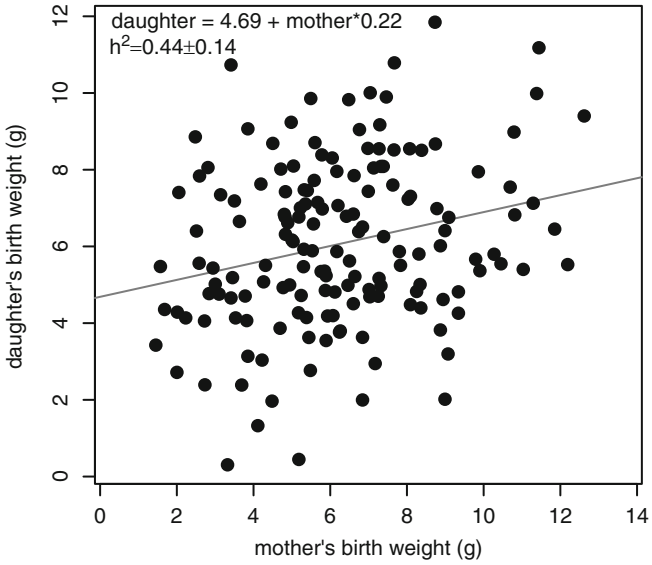

Fig. 20.2 Mother–daughter regression to estimate the heritability of gryphon birth weight in the simulated data of Wilson et al. (2010)

The key part of the regression equation in Fig. 20.2 is the slope, which by definition is the ratio of the covariance between mother and daughter phenotypes to the variance in the maternal phenotype, or Cov(mother, daughter)/Var(mothers). Because mothers and offspring only have 1/2 of their alleles identical by descent, Cov(mother, daughter) is 1/2 of the additive genetic variance. This allows the slope to be rewritten in terms of variance components as $V_A/2V_P$. To make communication easier, variance components are scaled to sum to one by dividing each component

by the phenotypic variance. Multiplying the slope from the regression by two brings the additive genetic variance onto this scale and yields a familiar term in quantitative genetics—the narrow-sense heritability $h^2 = V_A / V_P$. In Fig. 20.2 the heritability is 0.44 ± 0.14 which means that 44% of the variation in birth weight in the population is due to additive gene action, which is significantly greater than zero ($t = 0.44 / 0.14 = 3.2$, $P < 0.01$).

Heritability is an important concept in quantitative genetics, but it is crucial to recognize that its meaning is circumscribed by the statistical and biological models used in its estimation (Kruuk et al. 2008; Kruuk and Hadfield 2007; Templeton 2006; Vitzthum 2003). From the description above, it may be apparent that heritability does not identify some phenotypes as "genetic" and others as environmental. Instead, it is a within population partitioning of *variation* that depends on the allele frequencies of the population and the environments population members encounter. If either of these change (allele frequencies or environmental circumstances), so will the heritability. The importance of variation to heritability cannot be overstated. Many traits that certainly have large networks of interacting genes that make them possible, such as rhesus macaque mothers producing a hemochorial placenta during gestation, do not vary within macaque populations and thus have undefined heritabilities because their phenotypic variance is zero. Moreover, heritability estimates say little to nothing about between-group differences.

For traits that do vary, the quality of the variance components or resulting heritability estimates is very much dependent on how well the statistical model reflects the factors that could cause population members to resemble each other. In Fig. 20.2, a very simple model is used in which a phenotype is the sum of additive genetic and residual effects. Residual effects or residual variance refers to any variation that cannot be ascribed to additive effects or any other modeled factor (e.g., dominance, environmental factors, maternal environment). More elaborate models will be discussed below, but in this simple case the only reason for mothers and daughters to resemble one another in the population is the additive effect of alleles they have in common. Additivity means that alleles do not interact with one another at a locus (dominance) or across loci (epistasis). This a simplistic view of the genetics of most quantitative phenotypes but it may be defensible in many situations. For example, dominance cannot affect this mother–daughter similarity because only a single allele per locus is transmitted between generations meaning that mothers and daughters cannot share a pair of alleles at a locus (i.e., a genotype) that are both identical by descent.

More importantly, for the heritability estimate to be strictly valid, we must assume that relatives do not experience environments that might cause them to resemble one another. There are two ways to ensure that heritability estimates or variance components are not affected by this problem of shared environment. In a controlled breeding setting offspring can be randomized across environments, including cross-fostering which is the transferring of infants or even embryos between mothers (Maestripieri 2003). For unmanaged populations, this is rarely possible and the only recourse is to attempt to measure as much as possible about

the environments individual's experience and account for them in more elaborate statistical models that reflect this biological complexity.

In recent decades, the quantitative genetic techniques used by many animal breeders, ecologists, and medical geneticists have expanded to accommodate this complex reality. Animal breeders and ecologists call the most common of these approaches the "animal model" (Kruuk 2004). The techniques offer two main advantages for the problem of shared environments just described. First, they leverage all of the genealogical information in a given pedigree rather than picking a single type of relationship like parent–offspring pairs for regression or sets of siblings or cousins for analysis of variance. This increases statistical power and helps reduce the problem of relatives sharing environments because pedigrees can span large numbers of individuals and there are more likely to be some kin living in different environments. Second, an individual's phenotype is modeled in a more flexible manner as the sum of fixed and random effects allowing for a much more complex partitioning of phenotypic variance. The importance of the animal model can be understood by revisiting the genetics of birth weight. The simplest animal model for birth weight is:

$$\text{Birth weight} = \overline{x} + \text{ Genotype} + \text{Residual},$$

where $\overline{x}$ is the population mean. The model can be augmented to account for known environmental differences among individuals. An important kind of shared environment for mammalian offspring is that provided by mothers (Cheverud and Wolf 2009; Cheverud and Moore 1994). Several different kinds of "maternal effects" have been studied by primatologists that differ from the way they are typically conceptualized in quantitative genetics (Wolf and Wade 2009; Räsänen and Kruuk 2007). For quantitative geneticists, maternal effects are additional causes for resemblance among kin, particularly the offspring of the same mother. This can be modeled as another random effect in the animal model for maternal identity, which will have a variance component estimated (V_M), which is the maternal effect $m = V_M / V_P$ when scaled by the phenotypic variance. The characteristics of mothers that cause differences among their offspring are not identified by this approach, but it has the benefit of providing a general index of maternal performance.

$$\text{Birth weight} = \overline{x} + \text{Genotype} + \text{Maternal identity} + \text{Residual}.$$

In contrast, primatologists have typically studied maternal effects by measuring maternal characteristics and looking for statistical associations in populations with offspring phenotypes (e.g. dominance rank, birth order, maternal age Altmann and Alberts 2005; Maestripieri 2009). Depending on how permanent maternal characters are, these could either cause offspring of the same mother to resemble each other or be causes for dissimilarity. For example, if dominance rank is stable through adulthood, it should be a source of similarity among offspring of the same mother. On the other hand, primate mothers typically only give birth at a given age once, and being born to the same mother when she is in her prime is likely better for offspring

than when she is very young or very old. These sorts of maternal characters can be entered into the linear model as fixed effects. Additional factors unrelated to maternal effects can also be used such as neonate sex and population density.

$$\text{Birth weight} = \overline{x} + \text{Sex} + \text{Density} + \text{Maternal rank} + \text{Maternal age} + \text{Maternal age}^2 + \text{Genotype} + \text{Maternal identity} + \text{Residual}.$$

The importance of fixed effects is assessed through their regression coefficients. One would expect a negative slope for density (smaller infants due to increased feeding competition or elevated psychosocial stress under crowded conditions). Maternal age has linear and quadratic terms which would be expected to have positive and negative coefficients, respectively (older mothers have larger infants, but the effect attenuates as mothers age).

Fixed effects are generally those that are included because they have some clear influence on the phenotype that needs to be estimated, and the categories of the fixed effect are all present in the current data. Sex is a good example, having only two categories that would always be present for any comparison to be made. Random effects are different because the categories are typically only a sampling of a larger population of categories that are the focus of analysis. The additive genotype or maternal identity effects are both good examples. There is either one or just a few records in the dataset for each category and were we to repeat the analysis at a later time there would be more individuals to include (Wilson et al. 2010). Generally, regression coefficients are the result of interest for fixed effects while variance components are for random effects.

In contrast to the pattern in Fig. 20.2, most studies of birth weight in humans and livestock identify small heritabilities and relatively large maternal effects (Robson 1978; Wilson and Réale 2006). Shared uterine environment rather than sharing alleles identical by descent drives much of the similarity among a mother's offspring. Because the uterine environment provided by mothers can be influenced by her own genotype, this environment she provides has a partly genetic basis and evolves in response to selection on her or her offspring (Cheverud and Moore 1994; Wolf et al. 1998).

Ultimately, the meaning of heritability or other variance component estimates like maternal effects is limited by our knowledge of the biology of the organisms we study. The variance components identify statistical patterns that can be explained by more detailed analysis of proximate mechanisms including anatomy, energetics, endocrinology, and socioecology of primate reproductive biology and life histories. Rather than a static description of genetic variation, heritability can be viewed as the dynamic outcome of interactions within individuals, between individuals, and between the organisms and their environments over the course of their lifetimes. Continuing with the example of birth weight, this phenotypic outcome plays out as a negotiation of physiological mechanisms manipulated partly by fetal genes and partly by mothers. The theater of their effects is the molecular cross talk between mother and uterine resident situated within the larger socioecological context of the mother. Heritabilities and other quantitative genetic statistics are simply summary measurements of these complex biological processes.

20.2.1 Linking Up with Life History Evolution

These summary measurements are very useful in modeling the evolution of quantitative traits such as those that make up primate life histories. As in any model, simplifications are required. However, knowing there is a large or small amount of polygenic variation for traits in a population for selection to work on is better than knowing nothing at all about their genetics.

Much of life history theory has focused on the evolution of traits that impact components of the Euler–Lotka equation (Roff 2002), one form of which is given in Eq. (20.1). This equation relates the probability of a newborn surviving to age x (l_x) as well as the number of like-sexed offspring produced by an adult at age x (m_x) to fitness (λ). The summation covers only reproductive ages from first reproduction, α, to final reproduction, ω. Although the equation was developed by demographers to describe population growth (the λ term) it can also be interpreted as the rate of increase of a genotype specifying a particular life history pattern within a population (Stearns 1992; Charlesworth 1994).

$$1 = \sum_{x=\alpha}^{\omega} \lambda^{-x} l_x m_x. \tag{20.1}$$

Selection acts to increase the average value of λ in a population (Fisher 1930; Caswell 2001; Charlesworth 1994). Increases in λ can be achieved by earlier reproduction (smaller α), lengthening the reproductive lifespan (larger ω), and increases in the age-specific survival and fertility rates (larger l_x and m_x). Whether any of these changes to the life history can actually happen depends on the heritability of the individual life history traits and whether they are affected by phenotypic and genetic correlations with each other.

In the simplest (and least accurate) model, a single trait evolves in response to direct selection upon it. For example, initial fertility (m_α) is predicted to evolve as

$$\Delta \overline{m}_\alpha = \frac{1}{\overline{\lambda}} V_A \frac{\partial \overline{\lambda}}{\partial \overline{m}_\alpha}, \tag{20.2}$$

where $\Delta \overline{m}_\alpha$ is the change in the mean between generations, V_A is its additive genetic variance, and $\frac{\partial \overline{\lambda}}{\partial \overline{m}_\alpha}$ measures the strength and direction of selection, or how λ responds to a minute increase in initial fertility. Because variances are never less than zero, initial fertility will respond in the same direction it is selected (i.e., $\frac{\partial \overline{\lambda}}{\partial \overline{m}_\alpha}$ and $\Delta \overline{m}_\alpha$ have the same mathematical sign). Thinking about Eq. (20.1) indicates that m_α will always be selected for larger values. Taking the same univariate approach to each life history trait will lead us to expect an ideal life history of an animal born reproductively mature ($\alpha \approx 0$), which produces large numbers of healthy offspring at every age (all $m_x \gg 0$ and $\omega \approx \infty$) and lives forever (all $l_x \approx 1$). That this life history is so unfamiliar says there is something very wrong with this simplistic model (Law 1979).

Thinking about real life histories highlights the underlying problem—traits cannot change without affecting one another through their phenotypic and genetic correlations. Finite resources limit the ability of organisms to maintain or grow their own bodies while attempting to reproduce at the same time (Stearns 1989). Putting more energy into current reproduction often curtails future reproductive opportunities. In other words, the simple univariate model ignores *trade-offs*. The physiological decisions about the allocation of finite resources likely reflect evolved strategies for coping with the trade-offs faced by a given organism (Ricklefs and Wikelski 2002; Stearns and Magwene 2003). The familiar female primate way is slow growth and late maturation, with few offspring produced over a long reproductive career (Harvey et al. 1987).

Phenotypic and genetic correlations among traits, which allow for important trade-offs, do appear in multivariate extensions of Eq. (20.2) (Lande 1982; Lande and Arnold 1983; Caswell 2001). A two-trait example of this is given in Eq. (20.3) where the simultaneous evolution of survival from the beginning of reproduction to 1 year later ($p_\alpha = l_{\alpha+1} / l_\alpha$) and initial fertility is modeled. Like the simpler equation for a single trait, Eq. (20.3) can be read as "response = genetics × selection."

$$\begin{bmatrix} \Delta \bar{m}_\alpha \\ \Delta \bar{p}_\alpha \end{bmatrix} = \frac{1}{\bar{\lambda}} \begin{bmatrix} V_A(m_\alpha) & \mathrm{Cov}_A(m_\alpha, p_\alpha) \\ \mathrm{Cov}_A(m_\alpha, p_\alpha) & V_A(p_\alpha) \end{bmatrix} \begin{bmatrix} \dfrac{\partial \bar{\lambda}}{\partial \bar{m}_\alpha} \\ \dfrac{\partial \bar{\lambda}}{\partial \bar{p}_\alpha} \end{bmatrix}. \tag{20.3}$$

$\Delta \bar{m}_\alpha$ and $\Delta \bar{p}_\alpha$ are evolutionary response or changes in the mean between generations. The V_As are the respective additive genetic variances, while $\mathrm{Cov}_A(m_\alpha, p_\alpha)$ is the additive genetic covariance between the two life history traits. They summarize the influence of genetics on both traits and are often the results of pleiotropy—alleles for a single locus affecting multiple phenotypes. They are measured by the same techniques as heritabilities and all the same caveats discussed above still apply. Selection on the life history traits is described by $\dfrac{\partial \bar{\lambda}}{\partial \bar{m}_\alpha}$ and $\dfrac{\partial \bar{\lambda}}{\partial \bar{p}_\alpha}$, which indicate how λ would respond to a minute increase in m_α or p_α while the rest of the life history is held constant.

In Eq. (20.3), response in each life history trait is the result of direct selection on it and indirect selection on the other trait. For example, the response in initial fertility is $\Delta \bar{m}_\alpha = \dfrac{1}{\bar{\lambda}} V_A(m_\alpha) \dfrac{\partial \bar{\lambda}}{\partial \bar{m}_\alpha} + \dfrac{1}{\bar{\lambda}} \mathrm{Cov}_A(m_\alpha, p_\alpha) \dfrac{\partial \bar{\lambda}}{\partial \bar{p}_\alpha}$. Although selection on both traits is positive, response is likely to be ≈ 0 because of strong *negative* genetic covariance which reflects the trade-off between the traits. Any alleles in populations that increase both traits are likely to be rapidly fixed; those with opposite effects are likely to be quickly purged. Remaining variation is that which allows for roughly equivalent levels of fitness along an allocation continuum between the two traits (Fig. 20.3). While this two-trait approach is simplistic, it is a useful heuristic for

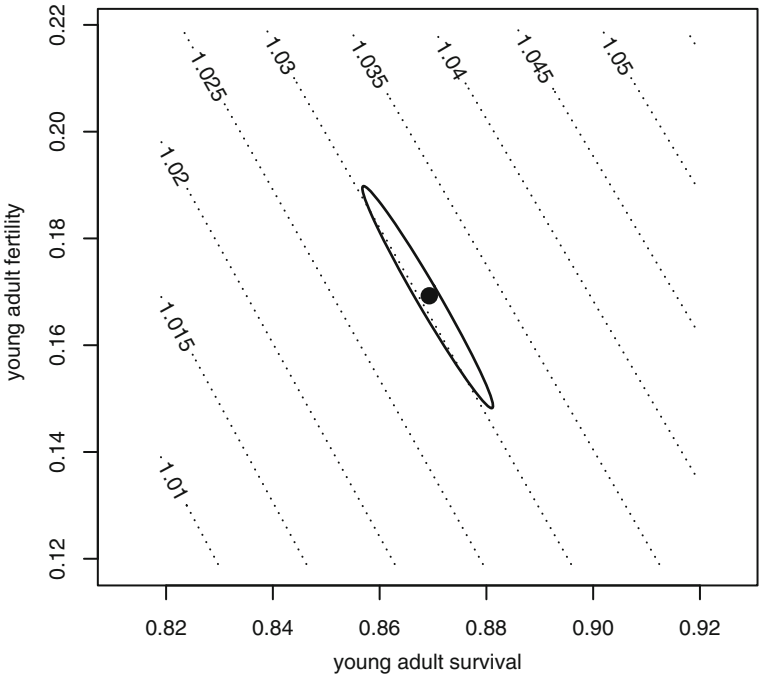

Fig. 20.3 Hypothetical negative genetic covariance between two life history traits on a landscape of fitness (λ) values using a life history model for female macaques described in Blomquist et al. (2011). The negative genetic covariance places most genetic variation along a fitness isocline at $\lambda \approx 1.03$. Selection is for increased survival and fertility (movement to the *upper right* of the figure) but the lack of genetic variation in this direction will limit response

thinking about trade-offs in life history evolution and why they ought to be reflected in genetic correlations (cf. Houle 1991; de Jong and van Noordwijk 1992; Roff 1997). Building on Eq. (20.3), more traits can be modeled but visualizing and describing the multi-way trade-offs among them becomes much more difficult.

20.3 Three Vignettes on Macaque Mothers and Infants

The remainder of this chapter demonstrates applications of these ideas to understanding the life histories of female macaques using long-term demographic data from Cayo Santiago. I will omit many of the details provided in original publications and focus on the important results and implications.

Cayo Santiago is a small island (15.2 ha) located just off the coast of Puerto Rico. Rhesus macaques have lived there since 1938, when about 400 individuals were released on the island after a long ship voyage from India (Rawlins and Kessler 1986b; Sade et al. 1985). Continuous demographic records have been kept by

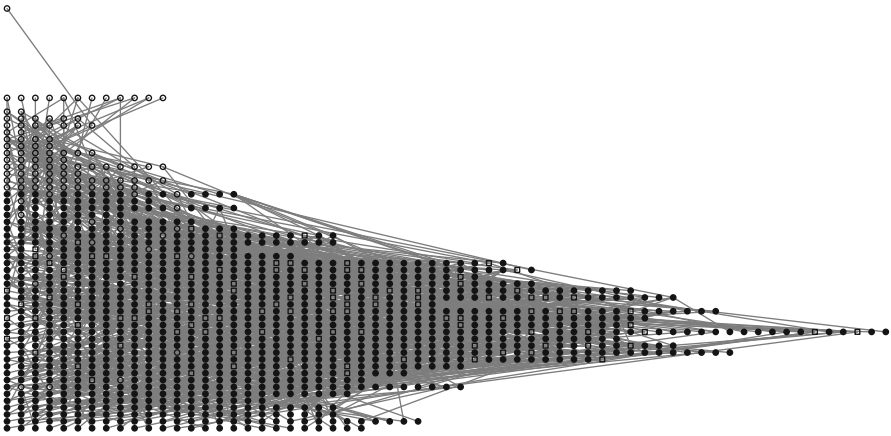

Fig. 20.4 Example pedigree for Cayo Santiago females. *Filled symbols* indicate females with reliably known age of first reproduction ($N = 975$). *Open symbols* are unphenotyped individuals that provide additional pedigree links. Each row is a single birth cohort with the oldest at the *top*

diligent observation since the mid-1950s. Monkeys are fed commercial monkey chow and provided water *ad libitum* but forage on natural vegetation and live in naturally formed social groups (Rawlins and Kessler 1986a). At the time this research began in 2005, there were nearly 8,000 known individuals who had lived on the island. The population has been managed through the removal of social groups and a random cull of juveniles such that only 34% of the individuals who had exited the population did so because of death with the other 66% taken to other facilities or sacrificed. The population was inoculated against tetanus in the mid-1980s (Kessler et al. 2006), and some animals had medical experiments conducted on them prior to the early 1970s (Sade et al. 1985). Otherwise, there has been a veterinary policy of nonintervention. Extensive maternal genealogies spanning up to nine generations in some cases are (Fig. 20.4) linked by paternities known from microsatellite markers in recent decades (Nurnberg et al. 1998). Rhesus macaques are seasonal breeders which arranges the population into well-defined birth cohorts (Rawlins and Kessler 1985). Clearly then, Cayo Santiago is not a natural/wild population, but the rich demographic and pedigree information make it a valuable group for exploring primate life history evolution.

20.3.1 Female Age of First Reproduction Is Weakly Heritable

The timing of female sexual maturation or age of first reproduction (AFR) (α in Eq. (20.1)) is one of the life history traits most frequently studied by primatologists (Kappeler and Pereira 2003; Charnov and Berrigan 1993). Within population variation in AFR has been a particular focus of research on the fitness benefits and costs

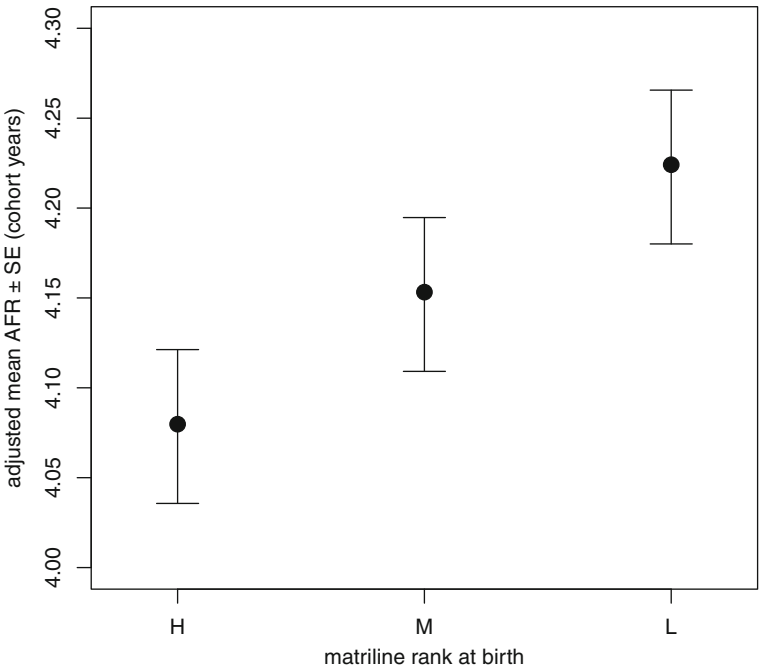

Fig. 20.5 Relationship between matriline dominance rank and female age of first reproduction (AFR). On average, high (H) ranking females mature at earlier ages than middle (M) or low (L) ranking females. Data are from the 975 individuals illustrated in Fig. 20.4

of social status in cercopithecine primates like macaques that have strong, stable dominance hierarchies and female philopatry (reviewed in Ellis 1995). Female philopatry is important because the formation of cooperative coalitions within matrilines (sets of females descended from a single founding female ancestor) is instrumental in maintaining and acquiring rank (Chapais 2004). Rank affects health by providing priority of access to limited resources (Koenig 2002) and shelter from stress induced by aggressive interactions (Sapolsky 2005). Females of higher rank tend to mature earlier which, as discussed above, yields higher fitness if no other aspects of the life history are affected by this physiological decision.

Rank-related trends in AFR are well-established among females at Cayo Santiago (Fig. 20.5, cf. Bercovitch and Berard 1993; Stucki et al. 1991). Additional environmental factors also have effects, such as being born later or earlier in a birth cohort and birth cohort membership, which is a proxy for population density and temporal changes in management or local climate. Together, these account for a little over 20% of the variance in female AFR (Table 20.1).

Using the "animal model" described above, it is also possible to estimate variance components and the heritability of female AFR with this dataset (Blomquist 2009b,a). There is very little comparative primate data to gauge our expectations for

Table 20.1 Linear model analysis of variance table predicting female age of first at Cayo Santiago ($R^2 = 0.242$)

	SS	df	*F*	*P*
Age in cohort	9.56	1	35.32	<0.001
Mother's age at birth	0.41	1	1.52	0.218
Matriline rank at birth	3.09	2	5.71	0.003
Birth cohort	69.24	34	7.52	<0.001
Residuals	253.35	936		

These are the fixed effects for the "animal model." Data are from the 975 individuals illustratedin Fig. 20.4

Table 20.2 Variance components and ratios for female AFR at Cayo Santiago

Variance	Component±SE	Ratio±SE
V_A	0.040±0.018	$h^2_{anim} = 0.149 \pm 0.065$
V_M	0.000±0.010	$m = 0.000 \pm 0.036$
V_R	0.230±0.019	$r = 0.851 \pm 0.065$
$V_{P\text{anim}}$[a]	0.270±0.013	
$V_{P\text{total}}$[b]	0.343	$h^2_{total} = 0.117$
		Fixed effects = 0.211

[a]Phenotypic variance after fixed effects are removed
[b]Total phenotypic variance including fixed effects

the results. The only primate taxa with maturation heritability estimates are captive baboons and humans (Towne et al. 2005; Williams-Blangero and Blangero 1995). For both species the heritability is usually moderate to high ($h^2 > 0.5$). This is somewhat unexpected given theoretical expectations that traits under strong selection should have little additive genetic variance, and low heritabilities, because selection quickly fixes or purges new mutations affecting the trait (Merilä and Sheldon 1999; Price and Schluter 1991; Blomquist 2009c). The heritability for female AFR at Cayo Santiago is more in line with theoretical expectations—12% of total phenotypic variance is due to additive genetic variation in the population, about half of what is attributable to fixed effects such as rank or birth cohort. The remaining two-third of the phenotypic variance is unexplained residual (Table 20.2). The heritability of AFR is small but significantly greater than zero, which suggests exploring genetic relationships between other life history traits searching for trade-offs is statistically justified (see below).

The main point of this vignette is to show that an interesting primate life history trait can be analyzed from extensive demographic data that provide both phenotypes and pedigrees. The quantitative genetic approach requires thinking clearly about environmental effects but it goes beyond this to provide variance components that appear in the equations for modeling the trait's evolution. Even from this univariate

perspective we would expect female AFR to evolve very slowly in macaques because of its low heritability. Furthermore, this low value itself may reflect a history of intense past selection because selection is expected to erode additive genetic variance by fixing beneficial and purging deleterious alleles (Roff 1997).

20.3.2 Environments Provided by Mothers Affect Infant Survival

In the previous vignette, maternal effects were not responsible for any variation in female AFR. This may be because the pedigree and data set do not allow separating maternal from additive genetic variance (Wilson et al. 2010), or because AFR is late enough in offspring life that the environment provided by mothers has been diluted by other factors.

However, the environment provided by mothers should surely have an effect on the mortality of infants in the first month of life. Infant mortality has been examined in many primates in either wild or captive settings. Most studies have focused on the social and other ecological factors affecting infant death rates, with a handful of genetic studies on inbreeding-related mortality risk (Cheney et al. 2006; Schino and Troisi 2005; Silk et al. 2003; van Schaik and Janson 2000; Takahata et al. 1998; Smith 1986; Busse 1982; Ralls and Ballou 1982; Wilson et al. 1978; Altmann et al. 1977; Hird et al. 1975). Two trends in infant mortality have already been described in good detail at Cayo Santiago. Hoffman et al. (2010) showed that infant death rates increased with maternal age, and this increase was more dramatic in their late teens and twenties. It is also known that tetanus inoculation in the early 1980s reduced death rates (Kessler et al. 2006). Infant death is a binary phenotype (1 = death, 0 = survival) which requires some additional statistical machinations. These are readily handled in generalized linear mixed models (GLMM) that allow variance component estimation for non-normally distributed phenotypes (Bolker et al. 2009; Gilmour et al. 2009).

Analysis of death in the first month of life at Cayo Santiago identifies strong maternal effects of both kinds discussed above, and consistent with previous studies of Cayo Santiago mortality. For the primatologist, older mothers are less likely to lose infants (negative linear coefficient), but very old mothers have higher infant death rates (positive quadratic coefficient, Table 20.3). From the animal breeder's view, there is a large variance component for infant death associated with maternal identity. Adjusting for the maternal age and time period (Table 20.3), about 12% of the variance in first month mortality can be attributed to maternal identity. The heritability of infant death is essentially zero (Table 20.4).

Not surprisingly, it is the aspects of the environment provided by mothers rather than infant genes that explain variation in infant death rates. The different views of maternal effects are complementary here. The variance components identify average differences among mothers in the risk of death for their infants, while the fixed effect regression coefficients measure an age-related pattern of risk common to the population. At the population level, Hoffman et al. (2010) note that older mothers

Table 20.3 Fixed effect GLMM coefficients for infant death in the first month of life at Cayo Santiago from 1960 to 2003

	Coefficient	SE	Wald *P*
(Intercept)	−0.924	0.127	
1975–1984 cohorts	0.004	0.072	<0.001[a]
1985–2003 cohorts	−0.254	0.064	
Maternal age	−0.132	0.026	<0.001
Maternal age^2	0.007	0.001	<0.001

[a]Test for significance of all levels of time period

Table 20.4 Variance components and ratios for death in the first month of life at Cayo Santiago

Variance	Component±SE	Ratio	*P*
V_A	0.000±na[b]	$h^2_{anim} = 0.000$	na
V_M	0.131±0.029	$m = 0.116$	<0.001
V_R[a]	1.000±na		

[a]Variance of the probit link function requires this fixed at $V_R = 1$
[b]Not estimated

are less active, spend more time in contact with their infants, and have smaller infants. Whether individual mothers differ in these age-related patterns of somatic and behavioral change that translate into mortality risks for their infants is a more difficult question beyond the scope of this analysis, but it can be addressed with random regression models used by animal breeders and ecologists (e.g. Meyer 2001; Brommer et al. 2007). Because all models of the evolution of senescence predict increasing genetic variance with age, we would predict that mothers become more heterogeneous in their effect on infant survival as they age (Hughes et al. 2002).

A further question about the maternal variance component is what it is actually measuring. The average environment provided by a mother is a large combination of behavioral and physiological characteristics. Some of these certainly have a genetic basis themselves, such as milk composition and yield or even responsiveness to infant solicitation (Nickerson 1995; Maestripieri 2003). This means they can evolve through direct selection on mothers or indirectly through selection on the survival of their infants. Conversely, infant traits can evolve through direct selection on infants or indirectly through selection on traits of mothers (Cheverud and Moore 1994). Slightly more elaborate quantitative genetic models for infant traits allow separating the maternal identity variance into genetic and nongenetic components. Alternatively, simple models for particular maternal traits like milk phenotypes or style of care can demonstrate a genetic basis for maternal variation directly (Bijma 2006).

This vignette makes the counterintuitive point that quantitative genetics is a very useful way for studying environments. I mean this in two ways. The first comes from the fact that partitioning variance into genetic and environmental

sources requires a good understanding, data, and statistical model of factors in the environment that affect phenotypes (e.g., maternal age). Second, when important environments are those provided by the phenotypes of other population members (e.g. mothers), quantitative genetics is invaluable for understanding the genetics of those phenotypes and thinking through how they should evolve (Wolf et al. 1999; Cheverud and Wolf 2009).

20.3.3 AFR Is Traded Off with Adult Survival

The final vignette of this chapter returns to the problem of life history trade-offs discussed above. The example extends the quantitative genetic analysis of female AFR to explore its phenotypic and genetic relationships with later life events (Blomquist 2009d), which will be measured with phenotypic and genetic correlations (Stearns 1989; Bell and Kofoupanou 1986; Reznick 1985). This approach of looking for trade-offs between two traits is simplistic and potentially problematic when we know that life histories involve many more traits, such as infant survival described above (Roff 2002). However, this is still a good place to start; once bivariate relationships are better understood, then we can start adding in greater complexity.

The question to be answered is "What is the cost of early life reproduction?" For long-lived, slow-reproducing female mammals like primates, the greatest cost they can pay is in reduced later life survival, because the only way for females to have many offspring is to live many years (Fig. 20.6). This suggests there could be a trade-off between female AFR and later life survival. Females that mature earlier would pay a cost of reduced lifespan, while those that delay reproduction for one or more years would be rewarded by a longer lifespan. This would be reflected by *positive* phenotypic and genetic correlations between AFR and lifespan or survival rates. The genetic correlations are predicted by the antagonistic pleiotropy theory of senescence (Williams 1957). Under this model, aging is caused by alleles with positive fitness effects early in life offset by negative consequences at advanced ages.

I used individual survival rates to ages 11, 16, 21, and 26 to gauge later life survival. These are basically the ratio of lifespan and the age cutoff and offer some advantages over analyzing lifespan itself (Blomquist 2009d). Like AFR most all of these have heritabilities significantly greater than zero (range: 0.13–0.39). Their phenotypic correlations with AFR are all positive but very weak (range: 0.083–0.132). The largest of these correlations are for the younger survival rates (ages 11 and 16) and both are statistically significant or nearly so (Table 20.5). The implication of these small positive correlations is that any trade-off between early reproduction and survival is very weak and may only affect survival over the first decade after maturation.

However, the genetic correlations are all large and positive (range: 0.476–0.706), indicating a strong trade-off between AFR and later life survival (Table 20.5). None

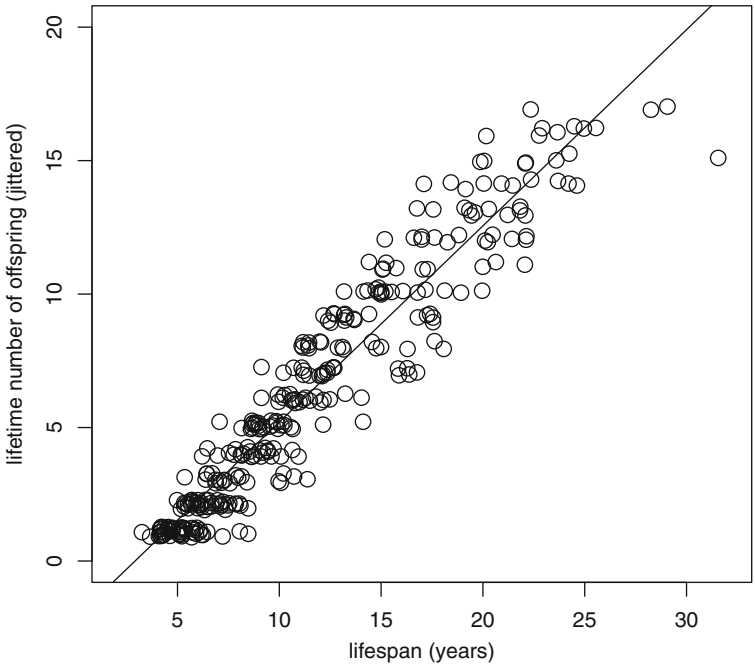

Fig. 20.6 Regression of lifetime number of offspring on adult lifespan for female rhesus macaques at Cayo Santiago ($P<0.0001$, $R^2=0.91$). Small random deviations (jittering) are added to number of offspring to expose overlapping points

Table 20.5 Phenotypic (r_P), genetic (r_A), and residual (r_R) correlations between female AFR and adult survival rates to age x (s_x)

	s_{11}	s_{16}	s_{21}	s_{26}
r_P	0.1094*	0.1323**	0.1008	0.0834
	0.065	*0.032*	*0.141*	*0.288*
r_A	0.5900*	0.5945***	0.4763***	0.7062**
	(0.320)	(0.215)	(0.170)	(0.283)
	0.067	*0.006*	*0.006*	*0.013*
r_R	0.0229	–0.0281	–0.0234	–0.0231
	(0.063)	(0.068)	(0.082)	(0.076)
	0.716	*0.680*	*0.775*	*0.761*

Standard errors are given in parentheses and *P*-values in italics above the correlation * $P<0.10$, ** $P<0.05$, *** $P<0.01$

of the residual correlations between AFR and survival rates are significant, although most of them are weakly negative, implying that environmental effects that promote early maturation will raise later life survival. It is this combination of strong positive

genetic correlations and weak negative residual correlations that results in the weak positive phenotypic values.

The strong positive genetic correlations between AFR and adult survival identify an important genetically mediated constraint on the reproductive decision-making of female macaques. Heavy investment in offspring production early in life imposes a cost of reduced lifespan for female macaques. For example, if we use the estimated genetic variances and covariances for AFR and survival to age 16, the cost of maturing 1 year earlier is a loss of about 11 months of adult life (Blomquist 2009d). Of course, there is a cost to delaying reproduction because it increases the risk of dying before ever reproducing.

The weaker phenotypic pattern probably results from environmental variation that affects each trait in similar ways and drive the negative residual correlations. For example, abundant resources or reduced psychosocial stress may allow earlier maturation and elevated survival rates. This phenomenon is sometimes called "silver spooning" as individuals living in beneficial environments often have high survival and fertility, just like the wealthy in human populations can spend lavishly on their homes, cars, clothing, and entertainment simultaneously and seemingly avoid any trade-offs in allocating their income (Grafen 1988; van Noordwijk and de Jong 1986). The opposite is also true: bad environments that delay maturation will probably also reduce survival rates. Simple models of variation in resource acquisition and allocation show that trade-offs—which are about allocation variation—will often not be apparent phenotypically when there is a great deal of variation in the resources available to population members (Roff and Fairbairn 2007). Quantitative genetic techniques are one way around this problem because the genetic correlations should be unaffected by environmental "silver spooning" and reflect trade-off-related allocation decisions.

20.4 Conclusions

These three vignettes offer a small taste of the sorts of questions mammalian and avian ecologists have addressed with quantitative genetic methods in recent decades (Kruuk et al. 2008). It may have been obvious that these techniques are "data hungry," requiring large numbers of phenotyped individuals linked up by extensive pedigrees. This is likely to prohibit their use with wild primate populations until/unless sufficient numbers of individual measurements become available from long-term research projects to achieve adequate statistical power (Jones et al. 2010; Quinn et al. 2006). It also emphasizes the extraordinary value of these projects and need for proper archiving and sharing of data (Strier et al. 2010).

The situation in captive primate colonies is quite different. Large numbers of primates in extensive pedigrees are being kept around the world (Rogers 2005), but, with some notable exceptions (e.g. Gagliardi et al. 2010; Ha et al. 2002; Martin et al. 2002; Jaquish et al. 1997, 1996; Williams-Blangero and Blangero 1995), they have rarely been used for quantitative genetic studies of phenotypes relevant to research on primate life history evolution and reproductive biology.

While estimating heritabilities or other quantitative genetic statistics has some value in itself for considering evolutionary dynamics of traits (Roff 1997), they become especially valuable when they identify new questions or can be used testing theoretical predictions about primate infants and their (allo)maternal caretakers. The examples above emphasize how life histories are a complex of genetically entangled traits (Rice 2004). Maturing early costs females reductions in later life survival. Maternal characteristics, many of which likely have a genetic component, drive variation in infant death rates and they change as mothers age. Dealing with this complexity quickly goes beyond our familiar adaptive logic of selection on individual traits. The benefit of an evolutionary quantitative genetic perspective, then, is to integrate studies of different parts of the life cycle, and potentially disparate aspects of behavior and physiology, by linking them in explicit models (Lynch and Walsh 1998).

Acknowledgements Thanks to Kate Clancy, Katie Hinde, and Julienne Rutherford for the invitation to contribute to this chapter. Cayo Santiago is part of the Caribbean Primate Research Center (CPRC) which is supported by the University of Puerto Rico, Medical Sciences Campus and the National Institutes of Health (NIH). The facility is also supported by Grant Number CM-5 P40 RR003640-20 from the National Center for Research Resources (NCRR), a component of NIH. The contents of this chapter are solely the responsibility of the author and do not necessarily represent the official views of NCRR or NIH. Additional funding for this research came from the University of Illinois Graduate College and the University of Missouri. Melissa Gerald, John Cant, Terry Kensler, Benedikt Hallgrimsson, and Jean Turnquist were all helpful resources while working with CPRC materials. Donald Sade, Richard Rawlins, John Berard, and Melissa Gerald must be credited for the upkeep of the demographic records on Cayo Santiago and Angel "Guelo" Figueroa, Edgar Davila, and Elizabeth Maldonado for their day-to-day maintenance. I also want to thank my wife, Rachel, who was busy *building a baby* and tackling the rest of life while I wrote this chapter.

References

Altmann J (1980) Baboon mothers and infants. Cambridge, Harvard University Press

Altmann J, Alberts SC (2005) Growth rates in a wild primate population: ecological influences and maternal effects. Behav Ecol Sociobiol 57:490–501

Altmann J, Altmann SA, Hausfater G, McCuskey SA (1977) Life history of yellow baboons: physical development, reproductive parameters, and infant mortality. Primates 18:315–330

Arnold SJ (1994) Multivariate inheritance and evolution: a review of concepts. In: Boake CRB (ed) Quantitative genetic studies of behavioral evolution. University of Chicago Press, Chicago, p 17–48

Bell G, Kofoupanou V (1986) The cost of reproduction. Oxf Surv Evol Biol 3:83–131

Bercovitch FB, Berard JD (1993) Life history costs and consequences of rapid reproductive maturation in female rhesus macaques. Behav Ecol Sociobiol 32:103–109

Bijma P (2006) Estimating maternal genetic effects in livestock. J Anim Sci 84:800–806

Blomquist GE (2009a) Environmental and genetic causes of maturational differences among rhesus macaque matrilines. Behav Ecol Sociobiol 63:1345–1352

Blomquist GE (2009b) Female age of first reproduction at Cayo Santiago: heritability and shared environments. Am J Phys Anthropol 138:94

Blomquist GE (2009c) Fitness-related patterns of genetic variation in rhesus macaques. Genetica 135:209–219

Blomquist GE (2009d) Trade-off between age of first reproduction and survival in a female primate. Biol Lett 5:339–342
Blomquist GE, Sade DS, Berard JD (2011) Rank-related fitness differences and their demographic pathways in semi-free ranging rhesus macaques (*Macaca mulatta*). Int J Primatol 31:193–208
Bolker BM, Brooks ME, Clark CJ, Geange SW, Poulsen JR, Stevens MHH, White J-SS (2009) Generalized linear mixed models: a practical guide for ecology and evolution. Trends Ecol Evol 24:127–135
Brommer JE, Wilson AJ, Gustafsson L (2007) Exploring the genetics of aging in a wild passerine bird. Am Nat 170:643–650
Busse C (1982) Social dominance and offspring mortality among female chacma baboons. Int J Primatol 3:267
Caswell H (2001) Matrix population models: construction, analysis, and interpretation, 2nd edn. Sinauer Associates, Sunderland
Chapais B (2004) How kinship generates dominance structures: a comparative perspective. In: Thierry B, Singh M, Kaumanns W (eds) Macaque societies: a model for the study of social organization. Cambridge University Press, New York, pp 3–10
Charlesworth B (1994) Evolution in age-structured populations. 2nd edn. Cambridge University Press, New York
Charnov EL, Berrigan D (1993) Why do female primates have such long lifespans and so few babies? or life in the slow lane. Evol Anthropol 1:191–194
Cheney DL, Seyfarth RM, Fischer J, Beehner JC, Bergman TJ, Johnson SE, Kitchen DM, Palombit RA, Rendall D, Silk JB (2006) Reproduction, mortality, and female reproductive success in chacma baboons of the Okavango Delta, Botswana. In: Swedell L, Leigh SR (eds) Reproduction and fitness in baboons: behavioral, ecological, and life history perspectives. Springer, New York, pp 147–176
Cheverud JM, Moore AJ (1994) Quantitative genetics and the role of the environment provided by relatives in behavioral evolution. In: Boake CRB (ed) Quantitative genetic studies of behavioral evolution. University of Chicago Press, Chicago, pp 67–100
Cheverud JM, Wolf JB (2009) The genetics and evolutionary consequences of maternal effects. In: Maestripieri D, Mateo JM (eds) Maternal effects in mammals. University of Chicago Press, Chicago, pp 11–37
de Jong G, van Noordwijk AJ (1992) Acquisition and allocation of resources: genetic (co)variances, selection, and life histories. Am Nat 139:749–770
Ellis L (1995) Dominance and reproductive success among nonhuman animals: a cross-species comparison. Ecol Sociobiol 16:257–333
Fisher RA (1930) The genetical theory of natural selection. Clarendon Press, Oxford
Gagliardi C, Falkenstein KP, Franke DE, Kubisch HM (2010) Estimates of heritability for reproductive traits in captive rhesus macaque females. Am J Primatol 72:811–819
Gilmour AR, Gogel BJ, Cullis BR, Thompson R (2009) ASReml User Guide, version 3.0. VSN International
Grafen A (1988) On the uses of data on lifetime reproductive success. In: Clutton-Brock TH (ed) Reproductive success: studies of individual variation in contrasting breeding systems. University of Chicago Press, Chicago, pp 454–471
Ha JC, Ha RR, Almasy L, Dyke B (2002) Genetics and caging type affect birth weight in captive pigtailed macaques (*Macaca nemestrina*). Am J Primatol 56:207–213
Harvey PH, Martin RD, Clutton-Brock TH (1987) Life histories in comparative perspective. In: Smuts B (ed) Primate societies. University of Chicago Press, Chicago, pp 181–196
Hird DW, Henrickson RV, Hendrickx AG (1975) Infant mortality in *Macaca mulatta*: neonatal and post-neonatal mortality at the California Primate Research Center, 1968–1972. A retrospective study. J Med Primatol 4:4–22
Hoffman CL, Higham JP, Mas-Rivera A, Ayala JE, Maestripieri D (2010) Terminal investment and senescence in rhesus macaques (*Macaca mulatta*) on Cayo Santiago. Behav Ecol 21:972–978
Houle D (1991) Genetic covariance of fitness correlates: what genetic correlations are made of and why it matters. Evolution 45:630–645

Hrdy SB (1999) Mother nature: a history of mothers, infants, and natural selection. Pantheon Books, New York
Hughes KA, Alipaz JA, Drnevich JM, Reynolds RM (2002) A test of evolutionary theories of aging. Proc Natl Acad Sci USA 99:14286–14291
Jaquish CE, Cheverud JM, Tardif SD (1996) Genetic and environmental impacts on litter size and early infant survival in three species of callitrichids. J Hered 87:74–77
Jaquish CE, Dyer T, Williams-Blangero S, Dyke B, Leland M, Blangero J (1997) Genetics of adult body mass and maintenance of adult body mass in captive baboons (*Papio hamadryas* subspecies). Am J Primatol 42:281–288
Jones JH, Wilson ML, Murray C, Pusey A (2010) Phenotypic quality influences fertility in gombe chimpanzees. J Anim Ecol 79:1262–1269
Kappeler PM, Pereira ME (2003) Primate life histories and socioecology. University of Chicago Press, Chicago
Kessler MJ, Berard JD, Rawlins RG, Bercovitch FB, Gerald MS, Laudenslager ML, Gonzalez-Martinez J (2006) Tetanus antibody titers and duration of immunity to clinical tetanus infections in free-ranging rhesus monkeys (*Macaca mulatta*). Am J Primatol 68:725–731
Kevles D (1985) In the name of eugenics. UCLA Press, Los Angeles
Koenig A (2002) Competition for resources and its behavioral consequences among female primates. Int J Primatol 23:759–783
Kruuk LEB (2004) Estimating genetic parameters in natural populations using the 'animal model'. Phil Trans Roy Soc Lond Ser B 359:873–890
Kruuk LEB, Hadfield JD (2007) How to separate genetic and environmental causes of similarity between relatives. J Evol Biol 20:1890–1903
Kruuk LEB, Slate J, Wilson AJ (2008) New answers for old questions: the evolutionary quantitative genetics of wild animal populations. Annu Rev Ecol Systemat 39:525–548
Lande R (1982) A quantitative genetic theory of life history evolution. Ecology 63:607–615
Lande R, Arnold SJ (1983) Measurement of selection on correlated characters. Evolution 37: 1210–1226
Law R (1979) Optimal life histories under age-specific predation. Am Nat 114:399–417
Lynch M, Walsh B (1998) Genetics and analysis of quantitative traits. Sinauer Associates, Sunderland
Maestripieri D (2003) Similarities in affiliation and aggression between cross-fostered rhesus macaque females and their biological mothers. Dev Psychobiol 43:321–327
Maestripieri D (2009) Maternal influences on offspring growth, reproduction, and behavior in primates. In: Maestripieri D, Mateo JM (eds) Maternal effects in mammals. University of Chicago Press, Chicago, pp 256–291
Martin LJ, Mahaney MC, Bronikowski AM, Dee CK, Dyke B, Comuzzie AG (2002) Lifespan in captive baboons is heritable. Mech Ageing Dev 123:1461–1467
Merilä J, Sheldon BC (1999) Genetic architechture of fitness and nonfitness traits: empirical patterns and development of ideas. Heredity 83:103–109
Meyer K (2001) Estimates of direct and maternal covariance functions for growth of australian beef calves from birth to weaning. Genet Sel Evol 33:487–514
Nickerson SC (1995) Milk production: factors affecting milk composition. In: Harding F (ed) Milk quality. Blackie Academic and Professional, London, pp 3–24
Nurnberg P, Saurmann U, Kayser M, Lanfer C, Manz E, Widdig A, Berard J, Bercovitch FB, Kessler M, Schmidtke J, Krawczak M (1998) Paternity assessment in rhesus macaques (*Macaca mulatta*): Multilocus DNA fingerprinting and PCR marker typing. Am J Primatol 44:1–18
Price T, Schluter D (1991) On the low heritability of life history traits. Evolution 45:853–861
Provine, WB (1971) The origins of theoretical population genetics. University of Chicago Press, Chicago
Quinn JL, Charmantier A, Garant D, Sheldon BC (2006) Data depth, data completeness, and their influence on quantitative genetic estimation in two contrasting bird populations. J Evol Biol 19:994–1002
Ralls K, Ballou J (1982) Effects of inbreeding on infant mortality in captive primates. Int J Primatol 3:491–505

Räsänen K, Kruuk LEB (2007) Maternal effects and evolution at ecological time scales. Funct Ecol 21:408–421
Rawlins RG, Kessler MJ (1985) Climate and seasonal reproduction in the Cayo Santiago macaques. Am J Primatol 9:87–99
Rawlins RG, Kessler MJ (eds) (1986a) The Cayo Santiago macaques: history, behavior, and biology. SUNY Press, Albany
Rawlins RG, Kessler MJ (1986b) The history of the Cayo Santiago colony. In: Rawlins RG, Kessler MJ (eds) The Cayo Santiago macaques: history, behavior, and ecology. State University of New York Press, Albany, pp 47–72
Reznick D (1985) Costs of reproduction: an evaluation of the empirical evidence. Oikos 44: 257–267
Rice SH (2004) Developmental associations between traits: covariance and beyond. Genetics 166:513–526
Ricklefs RE, Wikelski M (2002) The physiology/life-history nexus. Trends Ecol Evol 17:462–468
Robson EB (1978) The genetics of birth weight. In: Falkner F, Tanner JM (eds) Human growth: 1 principles and prenatal growth. Plenum Press, New York, pp 285–297
Roff DA (1997) Evolutionary quantitative genetics. Chapman and Hall, New York
Roff DA (2002) Life history evolution. 2nd edn. Sinauer Associates, Sunderland
Roff DA, Fairbairn DJ (2007) The evolution of trade-offs: where are we? J Evol Biol 20:433–447
Rogers J (2005) Genetics: a survey of nonhuman primate genetics, genetic management and applications to biomedical research. In: Coote SW (ed) The laboratory primate. Academic Press, New York, pp 487–501
Sade DS, Chepko-Sade BD, Schneider JM, Roberts SS, Richtsmeier JT (1985) Basic demographic observations on free-ranging rhesus monkeys. Human Relations Area Files, New Haven
Sapolsky RM (2005) The influence of social hierarchy on primate health. Science 308:648–652
Schino G, Troisi A (2005) Neonatal abandonment in Japanese macaques. Am J Phys Anthropol 126:447–452
Silk JB, Alberts SC, Altmann J (2003) Social bonds of female baboons enhance infant survival. Science 302:1231–1234
Smith DG (1986) Incidence and consequences of inbreeding in three captive groups of rhesus macacques (*Macaca mulatta*). In: Benirschke K (ed) Primates: the road to self-sustaining populations. Springer, New York, pp 857–874
Stearns SC (1989) Trade-offs in life history evolution. Funct Ecol 3:259–268
Stearns SC (1992) The evolution of life histories. Oxford University Press, Oxford
Stearns SC, Magwene P (2003) The naturalist in the world of genomics. Am Nat 161:171–180
Strier KB, Altmann J, Brockman DK, Bronikowski AM, Cords M, Fedigan LM, Lapp H, Liu X, Morris WF, Pusey AE, Stoinski TS, Alberts SC (2010) The primate life history database: a unique shared ecological data resource. Meth Ecol Evol 1:199–211
Stucki BR, Dow MM, Sade DS (1991) Variance in intrinsic rates of growth among free-ranging rhesus monkeys. Am J Phys Anthropol 84:181–191
Takahata Y, Suzuki S, Agetsuma N, Okayasu N, Sugiura H, Takahashi H, Yamagiwa J, Izawa K, Furuichi T, Hill DA, Maruhashi T, Saito C, Sato S, Sprague DS (1998) Reproduction of wild Japanese macaque females of Yakushima and Kinkazan islands: A preliminary report. Primates 39:339–349
Templeton AR (2006) Population genetics and microevolutionary theory. Wiley, Hoboken
Towne B, Czerwinski SA, Demerath EW, Blangero J, Roche AF, Siervogel RM (2005) Heritability of age at menarche in girls from the Fels longitudinal study. Am J Phys Anthropol 128: 210–219
van Noordwijk AJ, de Jong G (1986) Acquisition and allocation of resources: their influence on variation in life history tactics. Am Nat 128:137–142
van Schaik CP, Janson CH (eds) (2000) Infanticide by males and its implications. Cambridge University Press, New York
Visscher PM, Hill WG, Wray NR (2008) Heritability in the genomics era—concepts and misconceptions. Nat Rev Genet 9:255–266

Vitzthum VJ (2003) A number no greater than the sum of its parts: the use and abuse of heritability. Hum Biol 75:539–558
Williams GC (1957) Pleiotropy, natural selection, and the evolution of senescence. Evolution 11:398–411
Williams-Blangero S, Blangero J (1995) Heritability of age of first birth in captive olive baboons. Am J Primatol 37:233–239
Wilson AJ, Réale D (2006) Ontogeny of additive and maternal genetic effects: lessons from domestic mammals. Am Nat 167:E23–E38
Wilson AJ, Reale D, Clements MN, Morrissey MM, Postma E, Walling CA, Kruuk LEB, Nussey DH (2010) An ecologist's guide to the animal model. J Anim Ecol 79:13–26
Wilson ME, Gordon TP, Bernstein IS (1978) Timing of births and reproductive success in rhesus monkey social groups. J Med Primatol 7:202–212
Wolf JB, Brodie III ED, Cheverud JM, Moore AJ, Wade MJ (1998) Evolutionary consequences of indirect genetic effects. Trends Ecol Evol 13:64–69
Wolf JB, Brodie III ED, Moore AJ (1999) Interacting phenotypes and the evolutionary process, II. Selection resulting from social interactions. Am Nat 153:254–266
Wolf JB, Wade MJ (2009) What are maternal effects (and what are they not)? Phil Trans Roy Soc Lond Ser B 364:1107–1115