QUANTITATIVE GENETICS OF LIFE HISTORY MICROEVOLUTION IN THE CAYO SANTIAGO RHESUS MACAQUES (*MACACA MULATTA*)

BY

GREGORY ERIK BLOMQUIST

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Graduate College

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We hereby recommend that the thesis by:

GREGORY ERIK BLOMQUIST

Entitled:

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Be accepted in partial fulfillment of the requirements for the degree of:

Doctor of Philosophy

Signatures:

Director of Research - Steven R. Leigh

Head of Department - Steven R. Leigh

Committee on Final Examination*

Chairperson - Steven R. Leigh

Committee Member - Rebecca Stumpf

Committee Member - Paul A. Garber

Committee Member - Charles C. Roseman

Committee Member - James Cheverud

Committee Member -

* Required for doctoral degree but not for master's degree

Abstract

This is a study of the evolutionary genetics of a large colony of free-ranging rhesus macaques on Cayo Santiago, a small island located just off the coast of Puerto Rico. It focuses on documenting genetic and environmental influences on life history variation in female primates. The results are discussed in terms of understanding primate life history and sociality through analyses targeting variation within populations rather than interspecific comparisons. Population patterns of genetic variation and covariation, because of their role in evolutionary theory, are essential parameters to estimate, but are relatively understudied in primates. There are three sets of results. First, variation in female life history and morphology are shown to have a substantial genetic component documented in trait heritabilities and coefficients of additive genetic and residual variation. The patterning of trait heritabilities and coefficients of variation does not fit the classic model predicting lower genetic variation in traits closely associated with fitness. Instead, it accords with schemes emphasizing the developmental and physiological interdependencies among traits. Second, the social rank of female matrilines—sets of females related through maternal genealogy—is shown to have pervasive effects on life history, elevating both the fertility and survival of higher ranked individuals. The most important effect of rank on female fitness is mediated through adult survival rates, though high rank also increases infant survival and young adult fertility. Additionally, predicted breeding values are used to demonstrate homogeneity among rank levels—that observed life history differences between ranked individuals are primarily due to the nutritional and stress environment provided by social rank and not the genes individuals carry. Finally, trade-offs among life history variables are explored. Little to no evidence of trade-offs is found in the phenotypic correlations among traits. However, one key trade-off was identified in the genetic correlation between early fertility and lifespan. This is an important microevolutionary trade-off constraining the evolution of these fitness components and predicted by the antagonistic pleiotropy theory of aging. Analogies between rhesus female and human patterns of resource manipulation to mitigate life history trade-off are suggested.

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List of Abbreviations

CPRC	Caribbean Primate Research Center
LPMG	Laboratory of Primate Morphology and Genetics
UIUC	University of Illinois Urbana-Champaign
ML	Maximum Likelihood
REML	Restricted Maximum Likelihood
PBV	Predicted Breeding Value
QTL	Quantitative Trait Locus

List of Symbols

F	Wright's inbreeding coefficient.
μ or $\bar{x}$	sample mean
y_k	phenotypic value for individual k
a_k	additive genetic or breeding value for individual k
e_k	residual deviation for individual k
$\mathbf{y}$	vector of phenotypic values
$\mathbf{b}$	vector of fixed effects
$\mathbf{a}$	vector of additive genetic or breeding values
$\mathbf{e}$	vector of residual deviations
$\mathbf{X}$	design matrix for fixed effects
$\mathbf{Z}$	design matrix for random effect
$\mathbf{I}$	identity matrix
$\boldsymbol{\phi}$	matrix of kinship coefficients
$\mathbf{V}$	phenotypic covariance among individuals
σ^2_A	additive genetic variance
σ^2_E	residual or environmental variance
σ^2_C	cohort variance
σ^2_P	phenotypic variance
σ^2_R	variance due to social rank
h^2	narrow sense heritability σ^2_A/σ^2_P
e	residual or environmental effect σ^2_E/σ^2_P
c	cohort effect σ^2_C/σ^2_P
CV_A	coefficient of additive genetic variation $100\sqrt{\sigma^2_A}/\bar{x}$
CV_R	coefficient of residual variation $100\sqrt{\sigma^2_E}/\bar{x}$
λ	population finite rate of increase, or individual fitness

a_{ij}	population projection matrix entry
e_{ij}	elasticity of λ to changes in a_{ij}
r_P	phenotypic correlation
r_A	additive genetic correlation
r_E	residual correlation
r_s	Spearman's rank correlation
r	Pearson's correlation, or intrinsic rate of increase ($\lambda = e^r$)

Chapter 1

Introduction

Evolution is fundamentally a genetic process. Ironically, much of what biological anthropologists know about primate evolution has no defined connection between the agency of natural selection and the genetic bases of how animals are built and behave. Often this is not a problem. Darwin himself knew no more about inheritance than that offspring and parents resembled one another for some unclear reason (Fisher, 1930). This did not stop him from carefully amassing hundreds of examples of "descent with modification"—species whose form fit their present environments, but imperfections that belied common ancestry (Darwin, 1859, 1871; Gould, 1978). The comparative method is still a powerful tool for describing variation and generating hypotheses for its causes. For testing those hypotheses, however, it is critically deficient (Harvey and Pagel, 1991). If we want to study selection, or other evolutionary processes, as they occur or project their effects over much longer spans of time we must do more. This requires explicit evolutionary models, which have clearly defined genetic substrates and processes that modify them. Though difficult to implement the theoretical edifice is available for doing just this.

This thesis uses quantitative genetic models to address several problems that limit understanding primate life history evolution, using the female members of a free-ranging population of rhesus macaques (*Macaca mulatta*). First, the patterns of selection on primate life history traits are uncertain. Moreover, the genetic bases for these traits are largely unknown. Finally, the phenotypic and particularly the genetic relationships among traits are poorly understood. All of these deficiencies limit knowledge of how primate life histories experience and respond to selection, and hinder discussion of novel features of primate biology such as complex sociality, large brains, low reproductive rates, long lives, and extended juvenile periods (Stearns et al., 2003; Kappeler et al., 2003).

To address these issues, I begin by measuring the genetic bases of traits targeted by selection. These include morphological variables of animal size and shape and life history variables on the timing, frequency, and magnitude of demographically important events in the individual life cycle. Measuring both sets of variables enables the evaluation of ties between morphology, life history, and fitness and the testing of general hypotheses on the level of standing genetic variance in these traits (Chapter 3). From this I proceed to outline the impact of

social rank, an essentially environmental source of variation, on life history and fitness. Rank has a pervasive effect on life history variables, elevating the fitness of high-ranking females (Chapter 4). Because of how female macaques acquire rank and pass this status on to their daughters, female macaques circumvent trade-offs postulated by life history theory and empirically demonstrated in this population. Similar inter-generational transfers of environmental conditions are important mechanisms for humans to mitigate trade-offs (Kaplan and Robson, 2002; Lee, 2003). Furthermore, the mismatch between observable phenotypic patterns of variation in and covariation among traits and the genetic ones that will mold their response to selection implies much more attention should be paid to actively researching the genetic bases of intrapopulation variation in primates and enumerating the genetic assumptions of adaptationist selective scenarios.

1.1 Primate Life History Evolution

From an evolutionary perspective primates do not lead ideal lives. All else being equal, selection will favor a narrow combination of traits including perinatal sexual maturation, maximum reproductive output, and infinite lifespan (Roff, 2002). Instead, primates have much longer periods of development and subadult infertility than other mammals of their size, and females give birth infrequently to few offspring at a time. Primates do, however, tend to live longer than other mammals of comparable size (Martin and MacLarnon, 1990). This pattern suggests that all things are not equal—there are physiological and evolutionary relationships among these key life history variables (Harvey et al., 1987). In the case of primates, early maturation and high reproductive rates have been sacrificed in favor of longer lives. These relationships that limit organisms from becoming "Darwinian demons" of massive, precocious reproduction and infinite lifespan are life history trade-offs (Chapter 5; Law, 1979).

Candidate explanations for the primate pattern of delayed maturation, low reproductive rates, and long life focus on particular trade-offs among these demographic factors and their additional relationships with ecologically relevant variables such as foraging skill, body size, and brain size. One popular explanation for variation among mammals suggests that longer periods of slow growth allow for the attainment of larger body size, which in turn lowers adult mortality rates and increases fertility. The down side of this is a increased chance of dying before ever getting reap these survival and fertility benefits as an adult. The prime mover in this scheme for primates might be slow growth rates (Charnov, 1993; Charnov and Berrigan, 1993; Charnov, 1991), or reduced adult mortality (Kozlowski and Weiner, 1997). Other ideas focus on explaining just a particular aspect of the primate pattern. For example, delayed maturation has been explained by the need for juvenile primates grow slowly because of ecological risks that result from foraging in groups where they must balance the dangers of predation and feeding competition (Janson and van Schaik, 1993). Juvenile

mortality is again traded off with a long prereproductive period of life, though connections with adult fertility and mortality are unspecified. Often, these ideas are very difficult to test because the required data are unavailable and may never be possible to collect. Interspecific comparative tests (see Section 1.2) of the models usually focus on secondary predictions and are often indeterminate, yielding mixed support for each (e.g. Ross and Jones, 1999; Purvis et al., 2003).

The goal of this thesis is not to overtly test any of these ideas. Instead, I generate different data which allow the use of a very general framework to approach primate life history evolution focusing on analyzing naturally occurring variation within populations. By isolating what portion of this variation is a consequence of the action of genes or specific environmental factors like social rank, one can identify not only the amount of raw material for evolutionary change within a population (genetic variation), but also make short-term predictions on how the population would respond to different patterns of selection, estimate the selection pressures it is in fact experiencing, and speculate in a more informed fashion on the long-term evolution of primate life histories.

1.2 Studying Evolution in Biological Anthropology

Biological anthropologists are broadly concerned with the diversity of ways human and non-human primates make a living today and have evolved in the geological past (e.g. Fleagle, 1998; Schutkowski, 2006). Ultimately, most biological anthropologists are interested in explaining observed patterns of similarity and difference among living and fossil species, within and between populations, or between the sexes based on evolutionary theory. For a variety of reasons biological anthropologists typically invoke the action of natural or sexual selection as causal agents in producing patterns of commonality and divergence. By implication, most biological anthropologists are in the business of describing adaptations (Lauder, 1995; Grafen, 1988; Williams, 1966a). However, there is little reason to think that selection is the only process producing the patterns seen in living and extinct primates. It is, after all, only one of four population genetic forces that can alter allele frequencies in populations. Exclusive focus on selection neglects these other evolutionary processes and reduces debate to which selective scenario is most plausible (Gould and Lewontin, 1979). Often, none of the proposed ideas has any clear connection to the mechanistic bases of changes in gene frequency between generations that is microevolution.

In spite of the existence of elegant theory on the evolution of the kinds of quantitative characters that many biological anthropologists are interested in, two other approaches have largely dominated the field for explaining their evolution—the comparative method and model building. The comparative method is not exclusive to biological anthropology, or even the sciences. The

essence of the method is to find entities that differ in some interesting property, and based on other properties of those entities held in common or not to develop an explanation for the differences. Because living organisms are all descended from some common ancestor, the comparative method for biological anthropologists uses knowledge of these relationships and the patterns of changes from what are often hypothesized common ancestors to living taxa. For example, folivorous primates tend to have smaller brains for their body weights than their non-folivorous close relatives and presumed non-folivorous common ancestors. Folivores also have smaller home ranges suggesting they do not need to store and process as much information about where foods are located and how to find them (Clutton-Brock and Harvey, 1980; Milton, 1981). The recurrence of this pattern due to parallel or convergent evolution is taken to indicate natural selection must have favored smaller brains in folivores.

This is how much of the field operates. Regressions, analyses of variance, or other statistical techniques, including those that quantitatively deal with phylogenetic relationships among taxa (Nunn and Barton, 2001; Harvey and Pagel, 1991), using interspecific or inter-population data are used to identify unusual species, groups of species, or populations. Whatever is different about them is then a potential adaptation, fashioned by selection due to differing environmental conditions in the murky past.

Model building resolves some of the inherent shortcomings of the comparative method (Harvey and Pagel, 1991; Lauder, 1995). It is often used in refining and testing ideas generated by the comparative method. One of the important deficiencies of the comparative method is a weak connection between "form" and function. Model building will more clearly indicate function than comparisons. For example, a biomechanical model identifies how an anatomical structure works to generate forces in particular magnitudes and directions allowing faster runners, stronger diggers, more dextrous hands and feet, or teeth more suited to a particular diet (Hildebrand, 2001). Different designs perform differently, and a model will clearly identify this connection between design and performance. Similarly, a life history model identifies the benefits in reproductive fitness resulting from possible changes in the life cycle, provided an animal could make them. Constraints, or trade-offs, are placed within many models, including those of life history evolution (e.g. Charnov, 1991). When multiple traits are under selection and they are connected by trade-offs, a classic optimization problem results. For example, if greater adult survival rates entail a cost of reduced fertility (a trade-off), there is an optimal combination of survival and fertility that maximizes lifetime fitness (Williams, 1966b; Charlesworth, 1994). Optimization models of this kind are better than simple comparisons, but they still invoke the action of selection in a manner that can be deficient (Roff, 1994).

1.3 Quantitative Genetics and Multivariate Selection Theory

The comparative method and model building are still widely used among biologists and are likely to yield countless more insights into evolutionary ecology. However, animal and plant biologists have actively applied another scheme for understanding evolution and adaptation, formalized under the heading *multivariate selection theory* in the early 1980s, which is firmly rooted in quantitative genetics (Lande, 1979, 1982; Lande and Arnold, 1983; Arnold, 1983; Arnold and Wade, 1984a,b; Phillips and Arnold, 1989; Arnold, 1994). Applications in biological anthropology have been limited (e.g. Cheverud and Buikstra, 1981b; Cheverud, 1982; Cheverud and Dittus, 1992; Altmann and Alberts, 2003b; Altmann, 1991; DeGusta et al., 2003; Lawler et al., 2005; Lawler, 2006) and generally under-appreciated. Its limited use can be attributed to logistical difficulties in collecting the required data and that many biological anthropologists simply are not aware of or understand it fully.

Details of multivariate selection theory are provided in Chapter 2, but a short description is in order here. Generally, evolutionary quantitative genetics attempts to answer very basic questions about how particular traits evolve by identifying the mechanisms by which populations change over time. Quantitative genetics is a discipline concerned with the inheritance of continuously varying traits (e.g. height, molar tooth area, maximum running speed, age of sexual maturation) and how they respond to evolutionary forces of selection, drift, mutation, and gene flow (Roff, 1997; Lynch and Walsh, 1998). The connection between evolutionary processes and genetic substrates they act upon are much more explicit than in an optimization model or interspecific comparison. The variation within populations is also primary in quantitative genetics, though it has provided many interspecific and macroevolutionary insights (Lande, 1979; Ackermann and Cheverud, 2004; Schluter, 1996).

Multivariate selection theory requires two important linkages of information: the connection between phenotype and genotype, and the association between fitness and phenotype. These linkages are critical to understanding evolutionary change due to selection, but are not overt in modeling or comparative studies.

One goal of quantitative genetics is the description of the genetic bases of traits. This is the connection of phenotype, the outward measurable appearance of the trait, and genotype, its genetic structuring. For example, What fraction of the variation seen in a population is due to the action of genes? Or, when two or more traits are being investigated, To what extent do the same genes influence the separate traits? These are central statistics in quantitative genetics—the heritability and genetic correlation, respectively. Usually, the genes involved or even the number of them are unknown, but it is often assumed that there are enough genes involved and that they do not interact with one another greatly, such that the observed distribution is approximately normal

(i.e. Gaussian, a "bell curve"). A mean and variance adequately describe the distribution. Estimates of heritabilities and genetic correlations usually come from studies of phenotypic resemblance among individuals of varying degrees of relatedness (e.g. parents and offspring, siblings, cousins, etc.).

Knowing how much variation in and covariation among traits is due to genes is important because it dictates how the traits respond to selection (or move in concert with drift). If there is no genetic variance in a trait, meaning that individuals within the population do not vary in their genetic values for a given trait, it cannot respond to selection. Selection on multiple traits is readily accommodated, and this can highlight aspects of trade-offs. In the case of two traits each under selection for larger values, as in the example of survival and fertility in Section 1.2, if the genes that positively impact survival negatively impact fertility there will be little change in either trait despite strong selection.

Multivariate selection theory offers a cohesive manner of describing the relationship between reproductive fitness and suites of quantitative traits (Arnold, 1994). Selection has a very clear definition in this framework as a statistical association between fitness and a trait, such as tall individuals having more offspring than short ones, faster running juveniles surviving to maturity in greater numbers than slow ones, or earlier maturing females leaving more offspring in their reproductive careers. This is the second crucial connection of information, between fitness and phenotype. With information on the inheritance of traits (genotype–phenotype) and relationship between fitness and phenotype, multivariate selection theory provides a predictive model of how the population will respond. It indicates whether or not future generations will be any different from their parents on average (e.g. taller, faster, younger maturing).

If either informational connection is unavailable (fitness–phenotype or phenotype–genotype), no prediction can be made. However, these links are still useful to analyze in isolation. Knowing the fitness–phenotype connection provides information on what traits are under selection and in what directions (e.g. Lawler et al., 2005). On the other hand, knowledge of the phenotype–genotype map lets investigators know how much a trait or set of traits would respond to a given pattern of selection, and suggests how selection may have acted in the past (e.g. Hlusko et al., 2006, 2003).

Obstacles to collecting the needed data for making either of these connections in wild or free-ranging primate populations are formidable. There is, currently, very little empirically known about either kind of population (reviewed in Chapters 3 and 5). Long-term demographic data that provide information on reproductive fitness, or some proxy component, on many individuals are difficult to gather on animals that can live several decades (Alberts and Altmann, 2003; Rhine, 1992). Connecting fitness to phenotype then requires further collection of morphological, behavioral, or other data on those same individuals to answer whatever adaptive question is being asked. Making the phenotype–genotype connection was until recently even more formidable. Though recent statisti-

cal techniques have alleviated the problem somewhat (Kruuk, 2004), one still must gather phenotypic information on many individuals of known genealogical relationship to separate genetic from environmental effects (i.e. estimate heritabilities and genetic correlations).

1.4 Microevolution in the Cayo Santiago Macaques

This thesis is a deliberate attempt to use quantitative genetic models to answer a set of related questions about primate evolution. As an order, primates share a common set of features that sets them apart from other mammals.[1] I use a large population of free-ranging rhesus macaques (*Macaca mulatta*) from Cayo Santiago, Puerto Rico to explore their evolution (Section 2.2). This population, though not wild animals, is unique for its extremely long sequence of demographic records that provide demographic fitness measures and known mother-offspring relationships. Additionally, the paternity of many offspring has been identified. This genealogical information connects most of the population in a large extended pedigree suitable for quantitative genetic analysis—linking phenotype and genotype. Linking fitness and phenotype can be done by identifying statistical associations between fitness and morphological measurements on the skeletons of deceased population members that have been carefully curated, and between fitness and life history markers distilled from the demographic records. Only selection on females will be explored because they are easier to collect the life history and fitness information on, and are the limiting sex in demographic theory (Caughley, 1977).

Two distinctive features of primates are characteristics of their life histories (Harvey et al., 1987; Leigh and Blomquist, 2007). As noted previously, primates tend to have extended periods of growth and development in which they are totally to heavily dependent on the care of their mother and/or other close associates. This implies a high level of parental investment each time a female primate reproduces and low rate of reproduction compared to other mammals (Ross and Jones, 1999; Charnov and Berrigan, 1993). Second, primates can have very long lives which makes them interesting cases for the study of the evolution of aging (Roth et al., 2004; Finch and Ruvkun, 2001; Austad, 1997). Both of these traits are likely to be tied into life history trade-offs that limit following the ideal life course of early maturation, high fertility, and long lifespan (Roff, 2002).

Intuitively, it is clear that a population in which individuals reduce fertility below replacement levels will disappear without compensatory increases in survival. However, the selection pressures that may have molded and genetic architecture that underpins these distinctive life history traits are poorly de-

[1] These are, of course, distinctions identified using the comparative method.

scribed in non-human primate species. What ecological factors favor reducing fertility or raising survival in primates are unclear. Furthermore, whether the relationship between fertility and survival is mediated by genes is unknown. If it is, then it is a powerful constraint on the evolution of these traits—a real trade-off. If it is not, then there is little reason to think that such presumed life history trade-offs are anything more than epiphenomena of models requiring demographic balance (Harvey and Purvis, 1999). While genetic architecture and selection pressures are better understood in human populations, there is the intervening problem that humans live in very different environments today than they did only a few generations or decades ago in most parts of the world. Knowledge of the selective patterns and genetic bases of life history variation in wild and free-ranging primates will clarify the uniqueness of humans within primate and mammalian diversity.

Primates are also characterized by high levels of social interaction and complexity of behavior (Aiello and Dunbar, 1993; Whiten et al., 1999). Most primates live in social groups and form long-term associations with other group members (Sterck et al., 1997; Smuts, 1985). While social living is thought to offer a number of benefits (e.g. predator detection, success encountering food sources) it comes with a set of costs (e.g. predator attraction, competition for food sources), both of which group members may experience in varying degrees. This is often the result of dominance hierarchies within groups that array individuals in unequal positions of relative social power (Flack and de Waal, 2004; Lewis, 2002). In most cases, high-ranking individuals are expected to lead more comfortable lives and end up with higher lifetime fitness, but empirical studies of the fitness benefits of high social rank have been equivocal in many cases (reviewed in Bercovitch, 1991; Ellis, 1995). Some of this lack of a clear signal is due to the statistical problem of finding significant differences between high/low ranking segments of groups when the differences are fairly small and variances large (i.e. low power or high Type II error rate). The size of the Cayo Santiago population will minimize this problem as best can be done in a primate study.

In rhesus macaques there is a further problem to identifying rank-related fitness differences. High-ranking females tend to all be close relatives. The inequality a mother experiences is likely to be the same inequality her offspring suffer or enjoy. Life history differences among rank classes, which may translate into fitness differences, could be due to two confounded causes—genes that promote these patterns, or the strictly environmental differences of being high or low ranked. Aside from causing problems of identification, this may have some interesting effects on how populations experience selection and genetic drift. Populations that are subdivided into segments experiencing very different environments will inflate the total variance in reproductive parameters. This increases the opportunity for selection and drift to change the population composition. Furthermore, most human societies since the origin of agriculture and sedentism, which allowed the permanent accumulation of wealth, have had

systems of ascribed rather than achieved status (Fried, 1967; Sahlins, 1972). Wealth, power, and prestige can be inherited from one's parents along with their genes; offspring need not demonstrate ability to maintain status. Studies of genetics and selection in primate populations with analogous extrasomatic inheritable inequality provide a simplified model for how this may affect evolution in human populations. Regardless of the mode of status acquisition in human populations, the manipulation of resource availability is a common element for the circumvention of life history trade-offs in human and macaque populations (Chapter 5).

1.5 Chapter Overviews

Chapter 2: Background

This chapter provides a basic review of macaque socioecology and discussion of the history and particularities of the Cayo Santiago population. A treatment of the quantitative genetic methods for estimating heritabilities and genetic correlations and exposition of multivariate selection theory offer further background to the technical aspects of the thesis.

Chapter 3: Genetic Bases of Primate Life History and Morphology

This chapter lays the groundwork for understanding microevolution in the Cayo Santiago females. It begins with a review of studies on the quantitative genetics of non-human primates and Western human populations. Some further review places these studies in a broader context of mammalian and avian research. I then calculate the heritability and coefficients of additive genetic and residual variation in 6 life history traits, including fitness, and 15 skeletal measurements. The strength of selection on each trait is measured crudely by its correlation with fitness.

In addition to providing basic information on the genetics of these traits, I test several ideas on how genetic variation should be patterned among them. One view stresses the strength of the relationship between fitness and other traits. In populations at an evolutionary equilibrium, those traits that are tightly correlated with fitness are predicted to have little genetic variance (Fisher, 1930; Robertson, 1966; Mouseau and Roff, 1987; Crnokrak and Roff, 1995). The primary cause of this patterning is the erosion of genetic variance by selection, which should proceed at a faster pace in traits under stronger selection. Contrasting views focus on the genetic and developmental processes that generate genetic and residual variance in traits either through differences in mutational input or interdependencies among traits (Price and Schluter, 1991; Houle, 1998).

In contrast to expectations based on the "erosion of variance" view, fitness

and life history traits closely correlated with it have modest heritabilities in the Cayo Santiago females. Additionally, the patterning of coefficients of variation offers support to models stressing mutational inputs and interdependencies among traits. While these results do not fit equilibrium expectations, they are not unusual when compared to similar investigations of genetic variation in other mammalian species.

Chapter 4: Rank as an Environmental Impact on Life History

This chapter focuses on how social rank impacts female life history in macaques. I review previous studies on rank-fitness or rank-life history associations in catarrhine primates, and make some suggestions as to where linkages are expected based on current evidence, and where they would be most important based on simple demographic models.

Using a resampling procedure, I demonstrate that high-ranking segments of the Cayo Santiago population have fitness that is greater on average than lower-ranking segments. Further investigation, using recently developed techniques for analyzing demographic data (van Tienderen, 2000; Coulson et al., 2003), identifies differences in survival rates of mature adults and their infants as the primary pathways by which these overall disparities in fitness arise. This is particularly interesting because adult survival rates are the most important demographic factor in female primate life histories. Affecting adult survival rate will have the largest impact on fitness.

Parallel investigation of patterns within some key life history variables identifies that high-ranking females also tend to mature earlier. This is well-known in this population and expected from a review of other taxa, as well. A final portion of the analysis attempts to illuminate whether high-ranking females mature earlier because of genes that favor this or simply as a result of the environmental benefits of high rank. This is done by comparing the predicted breeding values—an estimate of the genetic value of an individual for a trait (Section 2.5)—among differently ranked segments of the population. No differences are found, suggesting that the earlier maturation of high-ranking females is environmental in origin. What specific environmental differences rank creates among females are unclear, but two possible mechanisms—shelter from stress and priority of access to resources—are discussed.

I also make some methodological points on how to best use heavily censored demographic data, like those from Cayo Santiago. The main argument is that life history variables that require completed lifespans (e.g. mean interbirth interval, lifespan itself) will represent only a small fragment of the total population and may well be biased toward certain subpopulations. Furthermore, relying on them entails "throwing out" the information provided by censored life histories. The other techniques used in this chapter are superior at utilizing all

the available information and effectively identifying rank-related differences in female life history.

Chapter 5: Trade-Offs Among Components of Fitness and Life History Traits

Several sets of life history trade-offs are explored in this chapter. Trade-offs, though central to life history theory, are poorly described in primates particularly as microevolutionary constraints shown through genetic correlations among life history traits or fitness components (Roff and Fairbairn, 2007; Stearns, 1989). I review some of the theoretical arguments about trade-offs and empirical studies in human populations, non-human primates, mammals, and laboratory organisms. I speculate on what some of the more important trade-offs are likely to be in primates. Hypothesized trade-offs are then sought in the phenotypic and genetic correlations among the fitness components and life history variables explored in previous chapters.

Essentially no evidence of trade-offs is provided by the correlations among fitness components, but analysis of some life history variables provides good evidence for trade-offs between early fertility and lifespan in this population. This is a key trade-off hypothesized by one model for the evolution of aging that implicates pleiotropic genes with beneficial effects on early life fitness components and negative effects on late life ones. Furthermore, it links distinctive primate traits of low reproductive rate and long lifespan to a common genetic foundation. This is important because it reaffirms that life history models have direct relevance for explaining selective processes and do not simply reflect the necessities of a demographic balance between births and deaths in a viable population.

However, these important trade-offs are not observed phenotypically. This emphasizes the importance of genetic techniques to exploring trade-offs. Phenotypic and genetic correlations among life history traits often do match. This is particularly true in female macaques where life history traits and fitness components tend to covary *positively*. Predicting their evolution based on phenotypic patterns alone (positive to 0 correlations) can give very misleading evolutionary dynamics—such as large simultaneous increases in survival and fertility. Negative genetic correlations, implying trade-offs among traits, provide an explanation for the commonly observed inter-generational stasis in life history traits and fitness components.

Chapter 6: Conclusion

Conclusions from each of the preceding chapters are reviewed and integrated. Some suggestions for future research are offered. I emphasize that primates do not appear to be all that dissimilar from other, better characterized groups of animals in the genetic architecture of life history and morphology. I argue that

the case of life history trade-off circumvention in heterogeneous social environments in female macaques is a useful analogue for processes in human evolution.

Chapter 2

Background

This chapter contains a description the primate genus *Macaca* focusing on locating it in primate diversity, and emphasizing the position of rhesus macaques within the genus. This is followed by further information on the history and particularities of the Cayo Santiago colony the sources of data used in this thesis. Next, multivariate selection theory is reviewed providing the framework in which macaque life history and morphology will be analyzed. The chapter concludes with a technical overview of the quantitative genetic methods used in later chapters of the thesis. It is included here to avoid profuse and redundant explanation of methods there.

2.1 Macaque Natural History

Macaques are an important group of primates to study for evolutionary biology, ethology, conservation, anthropology, and medicine (Roth et al., 2004). Despite a common ancestor of only $\approx$ 5.5 million years ago, macaques have diversified into distinct body shapes, habitats, and perhaps 19 species in 3 or 4 main groups (Hoelzer and Melnick, 1996; Hayasaka et al., 1996; Tosi et al., 2003). The macaque radiation corresponds roughly with the divergence of the *Homo* and *Pan* lineages suggesting humans and macaques may have dealt with similar macro-climatological changes during their respective dispersals. Other speciose primate groups in Africa began radiations around this time, as well (*Papio*, *Cercopithecus*). Fossil macaques in Southeast Asia increase in number and diversity after 2 million years ago, corresponding to the first human immigration to the region (Delson, 1980; Huffman, 2001). Macaques often live in contact with current human populations and the swidden agriculture common among Southeast Asian peoples may have played a role in recent macaque evolution in opening and changing habitats (Wheatley et al., 1996). Old World monkeys, such as macaques, and apes, including humans, share a common ancestor around 30 million years ago (Steiper et al., 2004).

All macaque species live in multimale-multifemale groups of about 15 to 60 animals in size (Menard, 2004; Thierry, 2007). Larger groups are documented but they tend to permanently fragment into new groups when over 100 individuals. Females remain in their natal troop for life, while males migrate among

troops one or more times after reaching sexual maturity (Thierry, 2007; van Noordwijk and van Schaik, 2001, 1985). In some macaque species (e.g. Barbary macaques), males may stay and breed in their natal group first, or may never leave (Menard and Vallet, 1996). Groups are usually composed of more females than males, though there is variation in the strength of this skew among species. For example, in rhesus macaques (*M. mulatta*) at Asorori in India the adult sex ratio was 1 male to every 3 or 4 females, but among wild Barbary macaques (*M. sylvanus*) in Algeria 1 male to every 1 or 2 females is common (Menard and Vallet, 1996). Female skew can be dramatic; Singh et al. (2000) recorded 9.9 females per male in wild lion-tailed macaques (*M. silenus*).

The basic outline of social hierarchies among female macaques has been known for almost half a century (Kawai, 1958, 1965; Kawamura, 1958, 1965; Sade, 1967; Missakian, 1972). Adult females form linear dominance hierarchies as determined by agonistic encounters. The daughters of each adult female follow their mother in rank. As younger sisters reach sexual maturity they displace their older sisters and occupy the rank immediately below their mother—a rule known as "youngest ascent." This pattern is seen most strongly in macaque species described as "despotic," such as Japanese (*M. fuscata*) and rhesus macaques. However, social dominance appears to have far less importance in other macaque species described as "egalitarian," such as Tonkean macaques (*M. tonkeana*) (Hill and Okayasu, 1996; Thierry, 1980). Intervention in conflicts by close female kin is suggested to be the proximate mechanism by which young females rise in rank (Datta, 1983; Berman, 1986). When groups fission they usually do so along maternal genealogical lines (Widdig et al., 2006; Menard and Vallet, 1993; Oi, 1988; Melnick and Kidd, 1983; Cheverud and Dow, 1985), and rank patterns can change in these periods.

Provisioned macaque troops, tend to have increased levels of aggression during feeding compared to their wild counterparts. This accords with models describing such food sites as clumped, rich food sources which individuals can actively defend by threatening and chasing away subordinates (Sterck et al., 1997; van Schaik, 1989; Wrangham, 1980). More even distribution and generally inferior production of food patches in the wild draws foraging individuals apart, reducing chances for encounters and the number of kin to support aggressive acts, while also lowering the benefit from defending any patch (Matsumura, 2001; Hill and Okayasu, 1996). Not all macaque species respond in this way to provisioning, however (e.g. Barbary macaques Chapais, 2004).

Macaque species and even populations of the same species range widely in body size. Adult female rhesus macaques on Cayo Santiago weigh about 7 kg ($\bar{x} = 6.94$ $n = 93$) (Campbell and Gerald, 2004). Rhesus are average sized macaques, but among the least dimorphic with males only about 25% larger than females. Rhesus macaques on Cayo Santiago are much larger and thus heavier than their wild counterparts, though they are proportioned identically (Weinstein, 2001; Melnick et al., 1984). Other macaque species range in body

size from about 3.5 kg (*M. fascicularis*, *M. sinica*, *M. radiata*) up to around 19 kg (e.g. *M. thibetana*, *M. sylvanus*) (Smith and Jungers, 1997).

Rhesus macaques have the largest geographic range of any non-human primate stretching from Afghanistan through northern India and Nepal and southern China. No long-term studies have been done of rhesus in the wild, but they are ubiquitous around towns and temples, simultaneously considered pests and sacred in many places (Imam et al., 2002). Human–rhesus interactions are so common in India that Malik (2001) estimated about 100 people a day are bitten by them. Diets of wild rhesus macaques are flexible. One study in a mixed deciduous-conifer forest in northern India found rhesus diets to be intermediate with respect to other macaques in their focus on ripe fruits (70% Lindburg, 1975). In a more harsh environment in the Murree Hills near Dunga Gali, Pakistan rhesus fed primarily on leaves and buds (84.4% Goldstein and Richard, 1989).

Wild rhesus females have their first offspring at 5 or 6 years of age and typically breed every other year when their infant survives. Females at Cayo Santiago begin breeding about 4 years of age and often give birth every year even if infants survive. Twins are very rare among macaques but have been observed in large captive and free-ranging colonies including Cayo Santiago. Seasonal breeding is common among macaque species, though its strength varies from discrete 2–3 month periods in several species to only rough peaks in a more-or-less year-round pattern of continuous reproduction (Oi, 1996). Females of some macaque species have pronounced anogenital sexual swellings whenever they are in estrus. In rhesus macaques, however, only adolescent females exhibit a deepening of color and mild swelling along the insides of the hind legs. This color change in accompanied by complementary reddening of facial skin (Bercovitch and Harvey, 2004). Rhesus macaques can live for many years. The oldest known member of the Cayo Santiago colony was 31 when she died. Captive individuals on calorie-restricted diets can live over 40 years (Roth et al., 2004).

2.2 The Cayo Santiago Colony

The Cayo Santiago colony has been thoroughly described in two major volumes which are recommended for the reader unsatisfied with the following brief discussion (Sade et al., 1985; Rawlins and Kessler, 1986). Cayo Santiago is a small island of 15.2 ha (37.6 acres) located at 18°09′ N 65°44′ W about 1 km off the southeast coast of the main island of Puerto Rico in the Caribbean Sea. The climate is subtropical (Holdridge, 1967) with a daily temperatures ranging from 23.8° C to 27.1°. Average annual rainfall is 163 cm. Hurricanes and tropical storms have impacted the island often inflicting damage, but cause little to no mortality among monkeys (John Berard, personal communication). Cayo Santiago is divided into two main sections of land called “Big Cay” and “Small Cay” connected by a narrow rocky isthmus that is sometimes submerged in shallow

sea water (See Figure 2.1). Both cays have rocky cliff faces, areas of dense vegetation, and open clearings. The northwestern end of the Big Cay is a mangrove swamp building out into the sea.

Rhesus macaques ($n = 409$) were released on Cayo Santiago in December of 1938 from wild populations trapped over a wide area in the mountainous region around Lucknow, India near the Nepalese border. C. R. Carpenter was the motivating force behind the establishment of the colony (Haraway, 1989), which was to serve the dual purpose as breeding colony of healthy animals for biomedical experiments in the continental United States and easily accessible and controllable location for socio-behavioral investigations circumventing the distance and logistical difficulties of monitoring monkeys and apes in the wild. Prior to the lease of the island (and later titling to the University of Puerto Rico) from the Roig family who owned it, Cayo Santiago was used as pasture for goats and had little vegetation. The maintenance of livestock on the island may have contributed to later problems of tetanus among the macaques. Despite some initial work by Carpenter and other scientists with the colony soon after its founding, the intervening Second World War caused the colony to be little used by primatologists until the mid-1950s. Support by the University of Puerto Rico, local scientists, and generous townsfolk maintained the small macaque population ($n \approx 178$).

Long-term demographic records of the colony began with the census and re-tattooing of animals by Stuart Altmann who came to the island in June 1956 and left in May 1958. A short lapse in the demographic records occurred between his departure and the census of January 1959 by James Gavan's group studying macaque physical growth. The records remained sporadic and were not collected in a systematic manner, but nearly all monkeys were captured and tattooed in this period. Carl Koford arrived at the colony in December of 1958 to study the composition and dynamics of the population. He instituted a regular census that has been maintained with only minor interruptions since the end of 1959. In most cases the data used in this thesis are based only on records of animals born in or after the 1960 birth season.

Births at Cayo Santiago are strongly seasonal (Rawlins and Kessler, 1985). Births have been recorded in all months of the year except for August, but half of the births occur between January and March and 90% are between December and April. Birth seasons are referred to by the year in which this peak is found. For example, the 1990 birth season ran from 18 December 1989 to 30 June 1990. The frequency of births in December and November has increased since 1960 (Figure 2.4). Photoperiod and the onset of spring rains influence the timing of the breeding/birth season. Wild rhesus macaques are also seasonal breeders, though some other macaques species are not (Lindburg, 1971; Oi, 1996).

The population of the Cayo Santiago has been capable of rapid growth since the institution of regular diet and care (Sade et al., 1976). Figure 2.3 shows the size of the population on January first of each year since 1960. A pattern of

growth followed by large-scale removal typifies much of this history. This was accomplished primarily by excision of social groups. By the end of 1973 groups A, C, E, K, and H had all been removed. Many members of these groups had been subject to medical experimentation (Sade et al., 1985) and their removal left only a few individuals with such history. They have all been excluded from the analyses in this thesis. Care must be taken in using records prior to the early 1970s as monkeys were routinely removed for medical experimentation disturbing the age and sex structure of groups. Further group removals have occurred and are shown in Table 2.1. The only groups present in 1960 with members surviving in them or daughter groups in December 2004 when this study commenced were A, F, and G.

While minimizing social disruption, group removals may increase inbreeding in the colony although there was no evidence for inbreeding from blood protein studies in the 1970s (summarized by Duggleby et al., 1986). The frequency of male transfer among groups may effectively distribute genes among groups, but strong female philopatry and group fission along genealogical lines promotes high levels of relatedness among female group members. Current colony maintenance practice relies on the annual removal of a subset of 2 year olds selected at random.[1] Inbreeding coefficients (F) calculated from the known pedigree of individuals range from 0 to 0.25. Only about 2% of individuals had $F >0$ (151 out of 7938), but this is clearly a underestimate of inbreeding in the colony as paternities are only known since 1989. Inbreeding appears to be more of a problem when noting 20% (151 out of 751) of individuals with known sires have $F >0$. This is a much better indicator as individuals with unknown paternity cannot have $F >0$. However, it remains an underestimate as the extensive maternal pedigree networks surely interlock via paternities prior to 1989 but these links are unknown. One estimate of effective population size for the colony was 70 (Duggleby et al., 1986).

Monkeys on Cayo Santiago have been regularly provided with commercially manufactured high-protein animal food since Altmann's arrival in 1956. Prior to that the diet was quite variable and consisted of raw fruits and grains. A food shortage in July 1968, is the only interruption since that time. The effect of this unusual event that claimed the lives of 10% of individuals over 1 year old in one social group, is controlled statistically in this thesis. Exactly how much food was provided to monkeys is unclear for much of the colony's history. Between 1959 and 1975, the average amount of monkey chow regularly provided per monkey varied between 0.11 and 0.27 kg/day (Sade et al., 1985). For some of this period 0.20 kg was the target. Current practice proscribes a 0.23 kg/monkey/day provision (http://ucm.rcm.upr.edu/cayotoday.html).

Monkeys also forage on natural vegetation, insects, larvae and will consume soil from time to time. During a two month study of diet in group L, monkeys spent only 10.8% of their waking day feeding. Of this 50.2% was spent feeding

[1] A larger fraction of males is removed to maintain a sex ratio of 1 male to 2 females.

on monkey chow with the remaining 49.8% devoted to other sources (Marriott and Roemer, 1989). Wild rhesus outside of temples or human settlements have been recorded to spend about 45% of their day feeding (Goldstein and Richard, 1989). The 10.8% devoted to feeding for Cayo Santiago macaques may be an underestimate as the study was carried out during the breeding season when males are known to curtail feeding to engage in mate guarding (Berard et al., 1994). Nevertheless, Cayo Santiago macaques spend far less of their day feeding than their wild counterparts. Water, collected through a rainwater catchment system is piped to stations in many places throughout the island, but food is distributed only in enclosed corrals from metal bins. Social groups maintain preferences for particular parts of the island and their included food sites (Lauer, 1980).

Food distribution bins are large metal containers with several openings that allow simultaneous feeding of several individuals on either side of the bin (photos in Figure 2.2).[2] There are currently 3 bins on the island, each located inside a separate chain-link fence corral. Corrals are several hundred meters away from their closest neighbor and well out of sight.

Causes of death have been recorded when possible for the Cayo Santiago macaques. However, of the 2670 deaths in the database at the time of this study only 389 (14.6%) had been categorized as something other than "unknown." Several categories of infant or pre-natal death—abortion (29 cases), stillbirth (98), orphaned (17), missing with mother (11)—together accounted for 40% of categorized deaths. The largest single cause of death was tetanus infection (121/389=31%). Trauma from wounds accounted for 14% of deaths (56). Tetanus infection has been largely eliminated from the colony since an inoculation program in the early 1980s. Only two deaths after 1985 have been attributed to tetanus. Aside from tetanus inoculation, no veterinary care is provided and should not affect mortality patterns.

2.3 Sources of Information

2.3.1 Census Records

The collected census records are currently maintained in a Microsoft Access database in Punta Santiago, Puerto Rico, which includes information on the dates of birth, death, removal, current status (dead/removed/alive), group membership, and maternal relationship of all monkeys known to researchers in the history of the colony. Four tables (named "dead," "event," "primate," and "transfer") were exported from this database and used in SAS programs to develop the set of data used in this thesis. In total, records were available for 7938 monkeys. Census records provide information required for generating indi-

[2] Another black and white photograph of Cayo Santiago macaques feeding at a bin can be found on page 319 of Silk (1987).

vidual fitness measurements and life history variables, and identifying maternal genealogical links. These data are used in exploring the genetic variation of traits associated with fitness (Chapter 3), searching for social rank–life history associations (Chapter 4), and illustrating life history trade-offs by analyzing the covariation among traits (Chapter 5).

2.3.2 Paternity Data

Beginning in 1989 the Cayo Santiago Genetics Group of John Berard, Fred Bercovitch, Matt Kessler, Michael Krawczak, Peter Nürnberg, and Jorg Schmidtke used variation in DNA microsatellites from blood samples to identify paternity of individuals from Cayo Santiago and some monkeys removed from the island living at Sabana Seca, another facility on the Puerto Rican mainland near San Juan. Methods used in paternity determination are described in Nurnberg et al. (1998). The paternity data have been used in a number of studies (e.g. Bercovitch and Nurnberg, 1996; Bercovitch et al., 2003; Widdig et al., 2001), and were used with permission for this thesis. Paternity data are crucial for this study because in many cases they genetically link together extensive maternal pedigrees. There were 751 paternities, from 190 different sires, available at the time of this study, which is about one tenth of the known maternities (n=7722, 1448 different mothers). Collection of paternity data continues to expand this dataset (Melissa Gerald, personal communication). Paternity data are used in any quantitative genetic analysis in this thesis (Chapter 3, heritabilities and CVs; Chapter 4, predicted breeding values; and Chapter 5, genetic correlations).

2.3.3 Social Rank

Information on the dominance status of the different matrilines (female genealogies) composing social groups were provided by John Berard and Donald Sade. Matriline ranks are determined by the directionality of agonistic dyadic interactions—fights, displacements, and stereotypical submissive displays. Rank information was available for all but three social groups. These groups were C, E, and H. C was large and apparently broke into subgroups regularly (Sade et al., 1985). E and H were both quite small. All three were removed by 1973 (see Table 2.1 and Figure 2.3). Information on social rank was used for testing the statistical associations between rank and fitness, rank and fitness components, and rank and life history variables (Chapter 4) and as environmental controls in the quantitative genetic analyses (Chatpers 3 and 5).

The numbers of observations resulting in the assessment of linear dominance among matrilines varied. However, dominance matrices can be constructed in many cases from a fairly limited duration of observation on Cayo Santiago. For example, Sade (1967) observed group F for the summer of 1963 and accumulated 100 fights between the 7 adult females of the group. More than one encounter was recorded for all but 5 of the 21 dyads. In a much longer study, Missakian

(1972) scored 5159 dyadic encounters among the 22 matrilines of group A. The study ran from January of 1968 through December 1969 and included more than 1360 hours of observation. Between 231 and 288 monkeys formed group A during this period. Only 4 of the 231 inter-matriline dyads were ranked based on only 1 or 2 encounters. Sade (1972) states that all "sociometric technicians" working on Cayo Santiago were trained to scores agonistic interactions in the same systematic manner and there was very little difference among observers.

Many social groups in recent years contain only a single matriline. Matriline differences thus cannot be compared within these groups. Furthermore, early records of social groups contain many matrilines while later groups contain few. To accommodate this difference, a set of rankings for matrilines was constructed for each group placing the matrilines into *high*, *medium*, or *low* rank. In the early groups with many matrilines a set of high, medium, and low ranking matrilines were specified. In groups with fewer matrilines, high, medium, and low refer to single matrilines. In groups with just two matrilines they are designated high and low, with no medium ranked matriline. While categorizing the sequence of matrilines into these three categories is somewhat arbitrary, it was done blind to the results reported in Chapter 4.

2.3.4 Skeletal Collection

The skeletons of some animals once living on Cayo Santiago are kept in San Juan, on the University of Puerto Rico Medical Sciences Campus (Centro Médico) at the Laboratory for Primate Morphology and Genetics (LPMG). The earliest members of this collection are just the skull and perhaps one humerus or femur. These were obtained opportunistically when animals died. Later members are complete skeletons, although some lack particular skeletal elements that have been used destructively for research purposes or were lost prior to the recovery of the body. The collection is a mixture of animals removed from Cayo Santiago that lived for some time afterwards at other facilities, animals removed from Cayo Santiago and immediately killed, and those that died on Cayo Santiago naturally. Identification of dead bodies found on the island is made based on animal tattoos, distinctive physical features and the last observations of animals as alive. All morphometric data used in the thesis were collected by the author using an 8 inch Mitutoyo sliding calipers that output measurements directly to a laptop computer. Only adult females from Cayo Santiago in the skeletal collection who had reproduced at least once were measured ($n = 121$). All skeletons were double-measured on left and right sides of the body (4 measurements total: e.g. trial 1 left humerus, trial 1 right humerus, trial 2 left humerus, trial 2 right humerus) following the procedures of Palmer and Strobeck (2003) for gathering fluctuating asymmetry data. Measurements taken are defined in Table 2.2 and match data collection in Hallgrimsson et al. (2002). Skeletal data were collected for comparing heritabilities and coefficients of genetic and residual variation

with life history variables calculated from census records (Chapter 3).

2.4 Methods for Studying Adaptation and Selection

This section reviews multivariate selection theory—the body of theory developed and popularized in the late 1970s and early 1980s for studying the evolution of multiple quantitative traits under selection (Lande, 1976, 1979; Lande and Arnold, 1983; Arnold and Wade, 1984b; Arnold, 1983, 1994). It is intended to provide some detailed background for understanding why genetic variation is so important for understanding how populations respond to evolutionary forces. Some literature review will also position the later chapters (3–5) in a broader framework by providing some illustrative examples and highlighting gaps in current knowledge.

2.4.1 Univariate Response—The Breeder's Equation

Explanation of the framework is most straightforward for a single quantitative variable—such as body mass in rhesus macaques. The *response* of any quantitative variable to selection is a function of *inheritance* and the form of *selection*. These three terms must be clearly defined (Arnold, 1994). *Response* is perhaps the easiest to explain. It is the change in the average value of the trait *between* generations. *Selection*, on the other hand, describes the change in trait means *within* a parental generation. For example, it is a common practice in animal breeding to only allow individuals that exceed some threshold value to reproduce. Using body mass as an example, a monkey breeder might limit reproduction only to individuals greater than 9 kilograms from a population with an average mass of 7 kilograms. The mean in this pool of actual parents is greater than the mean of the total population of potential parents. In unmanaged populations, however, breeding is not binary and selection must be defined in a more continuous manner. In these situations selection is defined as the regression of fitness (number of offspring) on the the trait being investigated (e.g. body mass). Finally, *inheritance* refers to the genetic transmission of differences among parental individuals in into offspring. If the selected parents are heavy, then their offspring should also be heavy. Common methods for describing inheritance rely on the regression of parents on offspring or analysis of variance designs (Roff, 1997). The animal model methods described in Section 2.5 are more flexible, but provide the same information by relying on the phenotypic resemblance among different kinds of relatives (Kruuk, 2004).

These three terms are summarized in the "breeder's equation" with $\Delta\bar{z}$ as the response, S describing selection, and h^2 the heritability (Section 2.5).

$$\Delta\bar{z} = h^2 S \tag{2.1}$$

Note that because h^2 is always positive, the sign of S will determine the sign of $\Delta\bar{z}$. This should be intuitively clear; selection for larger body size will result in larger individuals on average in the next generation. The stronger selection is (larger S), the greater the difference. Also, because h^2 is always between 0 and 1 the intergenerational response is always less than selection ($\Delta\bar{z} < S$).

An example from the literature can illustrate this approach further. Reale et al. (2003a,b) demonstrated response to selection in wild red squirrels (*Tamiasciurus hudsonicus*) from the Yukon. Parturition date in this population was estimated to have a small heritability of h^2 =0.16±0.03. This meant that if parturition date were under selection it could respond. They also found a significant relationship between parturition date and fitness, with females that gave birth earlier weaning a larger number of offspring during her lifetime (i =-0.17±0.05, i is the standardized selection differential—the ratio of S and the phenotypic standard deviation of the trait being analyzed $i = S/\sigma_P$). Both ingredients for a response to selection were present—significant if small heritability, and clear association between the trait and fitness. Over the period of the study (1989–2001) a population shift toward earlier parturition was seen. Furthermore, the observed change in parturition date was statistically indistinguishable from the predicted response to selection using the breeders equation across four generations of squirrel mothers ($\Delta\bar{z}$=-0.60±0.17 days/generation).[3]

2.4.2 Variance in Fitness Sets the Upper Limit for Selection

Demographic data, records of birth and death on known individuals, are key to understanding evolutionary processes because they set an upper limit on how selection can affect populations. In populations where all individuals contribute equal numbers of offspring to future generations, selection cannot occur. When there is variance in fitness either selection or genetic drift may operate. This is often measured with the "opportunity for selection," which is the squared coefficient of variation in absolute fitness (Crow, 1958, 1962; Arnold and Wade, 1984b; Downhower et al., 1987)

$$I = \frac{\sigma_W^2}{\bar{W}^2} \tag{2.2}$$

where I is the opportunity for selection, σ_W^2 is the variance in absolute fitness, and $\bar{W}$ is the mean absolute fitness.

Opportunity for selection has been described in many human populations (Cavalli-Sforza and Bodmer, 1971). Howell (1982), for example, estimated total opportunity for selection in the !Kung hunter-gatherer people of the Kalahari desert in southern Africa. She found there was substantial variance in both

[3]Predicted breeding values were used to demonstrate this change was microevolutionary and not entirely a plastic response to improved environmental conditions. See Sections 2.5 and Chapter 4 for discussion of breeding values.

males and females that selection could take advantage of. The value of I for women was 1.42 ($= 6.52/2.14^2$) and that for men 2.07 ($= 9.03/2.09^2$).

Variance in fitness is a prerequisite for selection to occur, but it does not ensure that it will happen. For there to be any selection, some of this variance in fitness must overlap with the variance for a quantitative trait of interest. If body mass varies within a population, but it does not covary with fitness in any way (i.e. there is no reproductive or survival advantage to being heavy, light, or average mass) then selection will not occur—S is 0 in Equation 2.1. The variance in fitness in this case is instead opportunity for genetic drift, as sampling effects in small populations may cause some difference between the means of actual and potential parents.

2.4.3 Selection v. Response

If fitness and the trait of interest do covary, then selection is occurring. For example, if heavy parents produce more offspring on average than light ones, then there is selection for increased mass—$S > 0$ in Equation 2.1. In other words, the slope of the regression of fitness on mass is significantly positive. The average mass of parents weighted by their number of offspring is greater than the unweighted mass of all potential parents. This is *selection*, a purely phenotypic phenomenon occurring within a generation.

However, that selection is occurring does not ensure that there will be any *inter-generational* change in the distribution of the quantitative trait. Continuing with the scenario started in the previous paragraph, if all heavy parental individuals are only heavy because of better diet, then the differences among parents that caused them to have higher fitness are environmental and cannot be genetically transmitted to their offspring. The distribution of mass in the offspring generation will be the same as in the parental generation. In this case $S > 0$, but h^2 is 0—selection is ongoing but there will be no *response*. While selection can be studied without genetic information, predicting response to it cannot.

2.4.4 Multivariate Response

Because many aspects of an animal's morphology, life history, and behavior may contribute to fitness, it is important that this framework is readily extended to model the evolution of multiple quantitative traits. The equations remain similar but are extended through matrix algebra to keep track of the different terms.

$$\mathbf{\Delta}\bar{\boldsymbol{z}} = \mathbf{G}\mathbf{P}^{-1}\mathbf{S} \tag{2.3}$$

If there are n traits, the response to selection is replaced by a column vector of length n indicating changes in the trait means ($\mathbf{\Delta}\bar{\boldsymbol{z}}$), and heritability is replaced by the $n \times n$ genetic variance-covariance matrix ($\mathbf{G}$). Selection is still

measured through regression of fitness on the traits ($\mathbf{S}$), but the multivariate context requires that phenotypic correlations among the traits be know. These are modeled in the phenotypic covariance matrix ($\mathbf{P}^{-1}$, with the exponent indicating the inverse of the matrix). More succinctly, the direction and magnitude of selection on a each individual trait involved in the analysis can be identified through multiple regression to produced a vector of partial regression coefficients of fitness on the set of traits. This vector ($\boldsymbol{\beta}$) replaces $\mathbf{P}^{-1}$ and $\mathbf{S}$ in Equation 2.3. The vectors $\mathbf{S}$ and $\boldsymbol{\beta}$ describe selection in different ways and have different names. The elements of $\mathbf{S}$ are called *selection differentials* and describe the univariate relationship between fitness and each trait individually. In $\boldsymbol{\beta}$, however, are *selection gradients*, which identify the relationship between fitness and each trait while all other traits are held constant.

In the example developed above, body mass is unlikely to be the only factor that affects fitness. Perhaps the area of molar teeth also affects fitness by raising the amount or rate of food that can be ingested enhancing survival and/or fertility. Separate univariate analyses could be carried out for these two variables, but this is likely to give an inaccurate picture of selection, the genetics of the traits, and their predicted response. This is due to the correlations between the two traits genetically and phenotypically. Heavy individuals may also tend to have large teeth. This correlation, regardless of its genetic or environmental cause, will affect the multivariate response to selection even if only a single variable is under selection.

Working through the matrix algebra, the response to selection for each of the variables depends on how they are being selected, their phenotypic, and genetic covariance with the other variable. If mass is trait 1, then the equation for its response is $\Delta\bar{z}_1 = G_{11}\beta_1 + G_{12}\beta_2$ and that for tooth size is $\Delta\bar{z}_2 = G_{22}\beta_2 + G_{12}\beta_1$. Or, using the selection differentials instead, the responses are $\Delta\bar{z}_1 = G_{11}P_{11}^{-1}S_1 + G_{12}P_{12}^{-1}S_2$ for body mass and $\Delta\bar{z}_2 = G_{22}P_{22}^{-1}S_2 + G_{12}P_{12}^{-1}S_1$ for tooth size. Subscripts in these equations indicate the row $\times$ column position of the element within each vector or matrix referenced.[4]

Note that when only a single trait is modeled is this way we recover the univariate breeder's equation (Equation 2.1); G_{11} is the additive genetic variance being "divided" by the phenotypic variance P_{11} to yield heritability and there is no phenotypic (P_{12}) or genetic covariance (G_{12}) to worry about as $\mathbf{P}$ and $\mathbf{G}$ are 1×1. Furthermore, if the genetic and phenotypic covariances among traits are 0, separate univariate analyses will give identical results to those using the multivariate equations.

[4] P_{11}^{-1} is the element in the first column and first row of the inverse of $\mathbf{P}$. It is *not* equal to $1/P_{11}$.

2.4.5 The Effects of Correlations Among Traits

However, non-zero correlations among traits greatly complicate the understanding of univariate selection differentials. For example, if body mass in fact contributes nothing to fitness beyond its positive correlation with tooth size then $\beta_1 = 0$ despite $S_1 > 0$ in Equation 2.3 and $S > 0$ in Equation 2.1. The response reduces to body mass being "dragged" along by its genetic covariance, if it has any, with tooth size ($\Delta\bar{z}_1 = G_{12}\beta_2$) (Hlusko et al., 2006; Fedorka and Mousseau, 2002). Unless this covariance is very large, the amount of response in body mass will be much smaller than would be predicted from a univariate consideration of its heritability and selection differential in Equation 2.1.

Genetic correlations have important effects on the response of traits to selection. Positive correlations between traits under positive selection will increase the response of each. However, negative genetic correlations ($G_{12} = G_{21} < 0$) between traits under positive selection (β_1 and $\beta_2 > 0$) can lead to situations in which there is little to no response. If for some reason the genes increasing tooth size reduced body mass, selection for larger values of each will not result in large increases of either. If the selection gradient on one trait is much larger than the other then the trait with the larger selection gradient will increase in the next generation while that with the smaller selection gradient will actually decrease. There will be a gain in fitness but it is accomplished through the net gain from increase in one trait and reduction in another.

This is a classic example of a trade-off between traits, in which fitness is maximized at intermediate values of the two traits involved. Trade-offs are central to life history theory because life history traits are consistently under strong directional selection (more offspring per year, longer lifespan, earlier maturation) and have a heritable component, but change very little between generations in wild populations (Roff, 2002, Ch. 3). Negative genetic correlations between traits are a possible explanation for this pattern. However, there is almost no published evidence of trade-offs represented as negative genetic correlations in $\mathbf{G}$ among life history traits in humans or non-human primates (Jaquish et al., 1996; Pettay et al., 2005).

2.4.6 Summary

As evolutionary biologists, practitioners of biological anthropology often describe adaptations in various primate species, or other levels of biological organization, without any reference to a particular model of how selection may have acted to produce a hypothesized adaptation. Differences among populations or species need not be due to the action of selection (Gould and Lewontin, 1979) and would not be thought of as adaptations in the conventional sense (Williams, 1966a).

Within population variation can, however, provide direct evidence of the action of selection in progress (Grafen, 1988). The hurdles to collecting the

required data in primate populations are substantial, but an explicit model and test for selection operating, however difficult it may be to employ, is better than no test at all. Many long-term studies of wild primates are reaching a point that these methods will be feasible for testing any variety of adaptationist hypotheses (Strier et al., 2006). Furthermore, one of the problems faced in employing these methods in primate populations is lessened by new techniques for estimating heritabilities and genetic correlations (**G**). These are reviewed in the final section of this chapter.

2.5 Quantitative Genetic Methods

This section provides an overview of the quantitative genetic methods used in this thesis. Further details of these methods can be found in Lynch and Walsh (1998) and Kruuk (2004). The treatment is somewhat technical and assumes some background in statistical theory and matrix algebra. Quantitative genetics is a large field, but attempts to answer some very basic questions about the variation seen in populations. How much of this variation that is due to genes is important to know. Without this information one cannot know how a trait will respond to selection (Section 2.4), or attempt to make comparisons among groups that may have different genetic properties. For example, there may be strong selection for earlier female age of first reproduction, but without additive genetic variation in this trait there can be no response to selection. Subsequent generations will, on average, have the same mean age of first reproduction. Furthermore, when attempting to compare high and low ranking females one might incorrectly ascribe differences in a trait to rank when, in fact, rank has no effect and the differences are entirely genetic (see Section 1.5, Chapter 4).

Some caveats should be made about the statistics produced from a quantitative genetic analysis (Sarkar, 1998). While heritabilities are typically thought of as the portion of observed variation due additive gene action, this does not mean that heritabilities index how "genetic" the trait being analyzed is (Wood and Lieberman, 2001). Colloquial discussion of heritability often can be confused because there must be some phenotypic variation in a trait for heritability to have any meaning. Simply by being a ratio, heritability ($h^2 = \sigma^2_A/\sigma^2_P$) is undefined when the phenotypic variance for a trait is 0. Provided there is phenotypic variance, a trait with a heritability of 0.90 simply has a greater fraction of its phenotypic variation explained by additive genetic variation than a trait with a heritability of 0.10. The trait with the lower heritability may in fact have many more loci that contribute to its phenotypic expression, but unless there is allelic variation in the population at these loci the heritability cannot capture the importance of genes to the phenotype. Which of these traits is more "genetic" is a subjective discussion that heritability cannot resolve. Where there is allelic variation at many loci, comparisons among populations are complicated by the simple fact that heritabilities are dependent on the frequencies of alle-

les (Roff, 1997). Because allele frequencies vary among populations it should not be surprising that there is often disagreement among studies estimating the heritability or additive genetic variance of the same trait. A further restriction on the generalization of heritability estimates is that they apply to a fairly narrow range of environmental conditions—those in which the sample population lives. Large changes in the environment will alter heritability (Schlichting and Pigliucci, 1998; Crow, 1962).

2.5.1 The Animal Model

In this thesis, I adopted an *animal model* approach to estimating quantitative genetic parameters. The animal model is a statistical linear model for a phenotypic trait that includes fixed and random effects. Having both types makes it a *mixed model*. In the simplest case of a single fixed effect—the sample mean—and two random effects of animal identity and residual error, an animal model can be written as

$$y_k = \mu + a_k + e_k \tag{2.4}$$

In Equation 2.4, y_k is the phenotypic or observed value for some trait measured on k individuals, such as female age of first reproduction or body mass. On the right side of the equation are the individual effects that contribute to variation in y. They are μ the mean for the trait, a_k the additive genetic or breeding value for each of the k individuals, and e_k the residual deviation for each individual. The additive genetic value is the genetic constitution of the individual. It is the difference from the population mean that offspring of individual k would on average have if individual k were randomly mated throughout the population.

The model can be written in a condensed matrix form (Equation 2.5) with multiple fixed effects, along with the mean, included in the vector $\mathbf{b}$. Records are associated with the fixed effects by the design matrix $\mathbf{X}$. When the mean is the only fixed effect $\mathbf{X}$ is just a $k \times 1$ column of 1s. In this simple case $\mathbf{b}$ is just μ. The terms $\mathbf{y}$, $\mathbf{a}$, and $\mathbf{e}$ are $k \times 1$ vectors. The matrix $\mathbf{Z}$ is a design matrix for the random animal effect and will always be an identity matrix unless there are repeated records on some individuals.

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Za} + \mathbf{e} \tag{2.5}$$

The additive genetic values ($\mathbf{a}$) have a mean of zero and covariance matrix $2\boldsymbol{\phi}\sigma_A^2$, where $\boldsymbol{\phi}$ is a matrix of coefficients of coancestry between individuals and σ_A^2 is the additive genetic variance. The ϕ_{ij} represent the probability that an allele drawn at random from individual i is identical by descent to that in individual j according to rules of autosomal Mendelian inheritance. For example, ϕ_{ij} between a parent i and offspring j is $\frac{1}{4}$, assuming there is no inbreeding. The residual deviations ($\mathbf{e}$) have a mean of zero. Also, $\mathbf{e}$ has a covariance matrix $\mathbf{I}\sigma_E^2$ where $\mathbf{I}$ is an identity matrix and σ_E^2 is the residual error

variance. Because $\mathbf{Z}$ is an identity matrix, and it is assumed the random effects ($\mathbf{a}$ and $\mathbf{e}$) are uncorrelated, the phenotypic covariance ($\mathbf{V}$) in the pedigree is given by Equation 2.6.

$$\mathbf{V} = 2\boldsymbol{\phi}\sigma_A^2 + \mathbf{I}\sigma_E^2 \tag{2.6}$$

More generally, as in the case of multiple records for an individual, the covariance among relatives can be determined by

$$\mathbf{V} = \mathbf{ZGZ}' + \mathbf{R} \tag{2.7}$$

when $\mathbf{Z}$ is not an identity matrix, $\mathbf{G}$ is $2\boldsymbol{\phi}\sigma_A^2$, and $\mathbf{R}$ is a modified covariance matrix for the residuals (Lynch and Walsh, 1998, p. 770). Equation 2.6 is an explicit form of Equation 2.7 for the animal model.

Written most simply, the animal model yields a decomposition of phenotypic variance (σ_P^2) into additive genetic (σ_A^2), and residual (σ_E^2) components.

$$\sigma_P^2 = \sigma_A^2 + \sigma_E^2 \tag{2.8}$$

Standardizing these variance components by the phenotypic variance yields the narrow-sense heritability ($h^2 = \sigma_A^2/\sigma_P^2$) and environmental effect ($e = \sigma_E^2/\sigma_P^2$) which must sum to 1. Houle (1992) has also suggested standardizing the additive genetic variance by the trait mean to derive another index of genetic variation in a trait. He calls this "evolvability" ($I_A = \sigma_A^2/\bar{x}^2$). It is very similar to a coefficient of variation (e.g. $CV_A = 100 \times \sqrt{\sigma_A^2}/\bar{x}$).

In summary, data are collected on some trait of interest ($\mathbf{y}$) for a group of individuals who are related to one another ($\boldsymbol{\phi}$). This information is used to estimate fixed effects ($\mathbf{b}$) and breeding values ($\mathbf{a}$), and to derive variance components (e.g. σ_A^2 and σ_E^2) for the trait. The observed phenotypic value for each individual measured is broken down into additive genetic and environmental components. For the whole group of animals measured the variance components indicate what portion of observed variance is due to genetic differences and what portion is due to unknown environmental effects that animals experienced.

2.5.2 Advantages of the Animal Model

Mixed model methodology is preferable to traditional means of estimating variance components, such as parent-offspring regression and sib analysis for several reasons. They flow from the fact that all known genealogical relationships can be used instead of just a specific kind of relationship within a pedigree (Roff, 1997). This gives two major benefits. First, statistical power will be greater because more information is on hand for making estimates and significance testing. Second, environmental factors that confound specific types of estimation techniques are less of a problem. For example, in rhesus macaques mothers

and daughters not only share genes but they also share social group membership and social rank. Which aspect of this commonality is being captured in a mother-daughter regression is uncertain, but it is likely that it inflates estimates of additive genetic variance. Heritabilities estimated with an animal model are usually lower than those estimated by parent-offspring regression (Kruuk, 2004). Mixed model methodology is also quite flexible in allowing the inclusion of additional fixed and random effects (such as the social group or rank effects just noted) that may obfuscate the genetic analysis. Not only are they controlled for in this manner (i.e. they will not inflate or depress the genetic terms being estimated by altering the resemblance among relatives) but the direction of the effects can be estimated (e.g. individuals born at high population density will mature later).

2.5.3 Adding Other Effects

Fixed effects aside from the mean are easily added in a linear mixed model approach. They expand the vector $\mathbf{b}$ and design matrix $\mathbf{X}$. In the animal breeding literature fixed effects often include sex, year of birth, or some experimental treatment. They can also include the regression of some continuous variable such as population size, age, rainfall, or temperature that have been of greater interest for field biologists (e.g. Garant et al., 2005; Postma and van Noorwijk, 2005; McLeery et al., 2004; Coltman et al., 2003; Reale et al., 2003a; Kruuk et al., 2002).

Fixed effects are usually variables for which all of the levels in the analysis are all of those you would ever be interested in. Typically there are rather few levels if it is a categorical variable. With random effects one considers the levels a small sampling of an infinite universe of possible levels. Fixed effects are variables that have simple effects on expected phenotypic values (means). Random effects influence trait variances and covariance among population members. In practice the distinction is somewhat blurry, however. Often, a good case can be made for including a particular variable in either fashion.

While adding fixed effects presents no great difficulty, adding random effects requires more substantial modification of the linear mixed model. For example to add another random effect uncorrelated with the additive genetic or residual effects one would modify the mixed model as shown in Equation 2.9.

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_1\mathbf{a} + \mathbf{Z}_2\mathbf{d} + \mathbf{e} \tag{2.9}$$

A vector $\mathbf{d}$ and design matrix $\mathbf{Z}_2$ are added for the new random effect. Unless there are repeated records, both $\mathbf{Z}_i$ will be identity matrices. As before, all random effects ($\mathbf{a}$, $\mathbf{d}$, and $\mathbf{e}$) are assumed to have a mean of zero. The new random effect $\mathbf{d}$ has a covariance matrix of $\mathbf{D}\sigma^2_D$, where σ^2_D is the variance due to this additional random effect and $\mathbf{D}$ is a matrix that groups individuals into sharing or not sharing this effect. For example, if the random effect is for having

been born in the same year then all d_{ij} for individuals born in the same year would equal 1, and for those born in different years it would be 0. If the random effect is genetic dominance then the d_{ij} are coefficients of fraternity determined from pedigree relationships. The phenotypic covariance among individuals ($\mathbf{V}$) can be calculated the same way as in Equation 2.7, but with $\mathbf{R} = \mathbf{D}\sigma^2_D + \mathbf{I}\sigma^2_E$. The phenotypic variance of the trait is simply the sum of the individual variance components ($\sigma^2_P = \sigma^2_A + \sigma^2_D + \sigma^2_E$).

2.5.4 Multivariate Analysis—Correlations Among Traits

One of the main goals of this thesis is the estimation of genetic correlations among life history and morphological traits for Cayo Santiago females. This is indispensable for understanding life history trade-offs and the multivariate response of suites of traits to selection. Just as the phenotypic variance of a single trait can be decomposed into genetic and other effects using an animal model, the phenotypic correlation or covariance between two traits can be broken down into these separate factors. A two-trait animal model takes the form of Equation 2.10.

$$\begin{pmatrix} \mathbf{y}_1 \\ \mathbf{y}_2 \end{pmatrix} = \begin{pmatrix} \mathbf{X}_1 & \mathbf{0} \\ \mathbf{0} & \mathbf{X}_2 \end{pmatrix} \begin{pmatrix} \mathbf{b}_1 \\ \mathbf{b}_2 \end{pmatrix} + \begin{pmatrix} \mathbf{a}_1 \\ \mathbf{a}_2 \end{pmatrix} + \begin{pmatrix} \mathbf{e}_1 \\ \mathbf{e}_2 \end{pmatrix} \tag{2.10}$$

If t traits are measured then Equation 2.10 can be written as Equation 2.11 where the elements of each matrix or vector in the equation are themselves matrices or vectors.

$$\mathbf{y}_t = \mathbf{X}_t\mathbf{b}_t + \mathbf{a}_t + \mathbf{e}_t \tag{2.11}$$

Equation 2.7 can still be used to calculate the phenotypic covariance among population members for each of the t traits individually, and the "cross-covariance" between the values for different traits. However, the structures of $\mathbf{G}$ and $\mathbf{R}$ are more complicated than in the univariate case. The same assumptions are made for each trait as in the univariate analysis. For $\mathbf{R}$ this means the expected residual covariance in the same trait between individuals is assumed to be 0. However, the covariance between traits in the same individual ($\sigma_E(m,n)$) is not assumed to be 0. For the two-trait animal model

$$\mathbf{R} = \begin{pmatrix} \mathbf{I}\sigma^2_E(1) & \mathbf{I}\sigma_E(1,2) \\ \mathbf{I}\sigma_E(2,1) & \mathbf{I}\sigma^2_E(2) \end{pmatrix} \tag{2.12}$$

The residual error covariance matrix expressing the residual variance for the two traits and the residual covariance between them is given in Equation 2.13; the matrix is symmetric as $\sigma_E(1,2) = \sigma_E(2,1)$.

$$\mathbf{R}_0 = \begin{pmatrix} \sigma^2_E(1) & \sigma_E(1,2) \\ \sigma_E(2,1) & \sigma^2_E(2) \end{pmatrix} \tag{2.13}$$

Similarly for $\mathbf{G}$, the additive genetic covariance for a single trait between individuals is just $2\boldsymbol{\phi}\sigma_A^2$. If $\mathbf{A} = 2\boldsymbol{\phi}$ and $\sigma_A(m,n)$ is the additive genetic covariance between traits m and n in the same individual then for the two-trait animal model

$$\mathbf{G} = \begin{pmatrix} \mathbf{A}\sigma_A^2(1) & \mathbf{A}\sigma_A(1,2) \\ \mathbf{A}\sigma_A(2,1) & \mathbf{A}\sigma_A^2(2) \end{pmatrix} \tag{2.14}$$

The additive genetic covariance matrix expressing the additive genetic variance for the two traits and the additive genetic covariance between them is given in Equation 2.15;[5] the matrix is symmetric as $\sigma_A(1,2) = \sigma_A(2,1)$.

$$\mathbf{G}_0 = \begin{pmatrix} \sigma_A^2(1) & \sigma_A(1,2) \\ \sigma_A(2,1) & \sigma_A^2(2) \end{pmatrix} \tag{2.15}$$

Genetic (r_A) and residual environmental (r_E) correlations can be constructed from the elements of $\mathbf{G}_0$ and $\mathbf{R}_0$. These come from standard equations for a covariance and correlation. For example

$$r_A = \frac{\sigma_A(1,2)}{\sqrt{\sigma_A^2(1) \times \sigma_A^2(2)}} \tag{2.16}$$

Finally, the phenotypic correlation (r_P) between two traits (m and n) is given by Equation 2.17, where this phenotypic value is partitioned into separate correlations corresponding to each of the random effects in the animal model.

$$r_P = r_A\sqrt{h_m^2 h_n^2} + r_E\sqrt{(1-h_m^2)(1-h_n^2)} \tag{2.17}$$

Extension of the two-trait model presented above to more traits is straightforward, as is the inclusion of additional random effects (Lynch and Walsh, 1998, p. 775).

2.5.5 Variance Component Estimation Techniques

Two related techniques exist for estimating variance components with the pedigree data presented by an animal model. They are maximum likelihood (ML) and restricted maximum likelihood (REML). The distinction between the two is that REML removes the fixed effects prior to estimating variance components through a linear transformation based on the design matrix for fixed effects such that

$$\mathbf{KX} = \mathbf{0} \tag{2.18}$$

REML estimates are then ML estimates for these transformed variables.

$$\mathbf{Ky} = \mathbf{K}(\mathbf{Xb} + \mathbf{Za} + \mathbf{e}) \tag{2.19}$$

[5] In matrix algebra $\mathbf{G}$ is the Kronecker product of $\mathbf{G}_0$ and $\mathbf{A}$. Note that $\mathbf{G}_0$ here is equivalent to the $\mathbf{G}$-matrix discussed in Section 2.4.

REML is generally preferable to ML for several reasons. ML estimates are biased because they assume that fixed effects are known without error, which they rarely—if ever—are. This tends to reduce the estimate of residual error. The bias can become quite large when there are many fixed effects in the model and sample size is low. The transformation to remove fixed effects also reduces the parameter space to search for solutions for the mixed model equations. This can cause substantial drop in the amount of time and computational power needed.

Two free packages are used in this thesis for performing these genetic analyses. The program SOLAR (Almasy and Blangero, 1998) uses ML to estimate variance components. The other program, DFREML (Meyer, 2000), provides REML estimates.

2.5.6 Significance Testing

The decision of whether model terms are significant can be made in several ways. All software used to run these genetic analyses output standard errors on each of the estimated values. A rough manner of testing the significance of the term is to create a z or t statistic from the ratio of the estimate to its standard error. A p-value can then be read from standard tables. Alternatives to this approach are randomization or bootstrapping procedures (Manly, 1997) and likelihood ratio tests (Lynch and Walsh, 1998, p. 857). Randomization procedures must be programmed to feed altered data sets to the genetics software and collect the outputs. The real estimated value is compared to the distribution of values from randomized or bootstrapped data sets. For example, if only 5 out of 1000 randomized data sets had a heritability estimate greater than the real value then the p-value for the real estimate is 5/1000=0.005. Randomization significance tests were explored for some tasks in this thesis. However, the time and computing resources required for their use was prohibitive.

One genetics software package used in this thesis, SOLAR, outputs likelihood ratio tests for fixed and random effects. A likelihood ratio test compares the log-likelihood of a model with a term (full model) and without it (reduced model). The difference between them is χ^2 distributed with degrees of freedom equal to the number of terms absent in the reduced model. A p-value can then be taken from χ^2 tables. Similar discrimination among models with and without terms can be done by fixing the value of a variance component or correlation to some value. For example, a genetic correlation can be tested to be significantly different from 0 by comparing the likelihood of the model with the correlation estimated and with it fixed at 0. Similar comparisons can be made with the correlation set at 1 or -1 (Almasy and Blangero, 1998).

2.5.7 Model Assumptions

The animal model, and its extensions with other effects discussed here, relies on a number of assumptions. Fundamentally, it is assumed that traits being modeled are subject large numbers of independently acting genetic loci. The additive genetic values and residual deviations should be normal and independently distributed, though departures from this do not matter much (Shaw, 1987). The phenotypic values should thus be normally distributed, too. If the base population itself is unselected then the variance component estimates using ML or REML are unbiased by selection (Mrode, 1996). Furthermore, finite population size, assortative mating, and inbreeding do not affect the variance component estimates (Kruuk, 2004). When adding additional random effects they are typically assumed to be uncorrelated with any other random effect in the model (e.g. Equation 2.9).

2.6 Tables and Figures

Figure 2.1: An aerial view of Cayo Santiago in December 1999. The Puerto Rican mainland can be seen in the upper-left corner of the photo. The image is public domain, available online from NOAA at `http://ccma.nos.noaa.gov/products/biogeography/benthic/htm/data.htm`. The white scale bar in the bottom left corner is 500 meters.

Table 2.1: Alphabetical listing of social groups on Cayo Santiago 1960–2004.

group	formation		fate	fission	
	source	date		daughter	date
A	original in 1960		removed by end of 1971	J	1-Jan-1965
				K	10-Nov-1969
				L	10-Nov-1969
AA	F	1-Nov-1992	removed early 1995	none	
BB	R	1-Jan-1996	removed by early 2004[1]	none	
C	original in 1960		removed by end of 1969	none	
CC	S	1-Jan-1996	removed by early 2004[1]	none	
DD	S	1-May-1997	removed Jan.–March 2002	none	
E	original in 1960		removed by end of 1971	none	
EE	F	1-Jun-1997	removed Jan.–March 2002	none	
F	original in 1960		still on Cayo Santiago	M	1-Aug-1973
				O	1-Nov-1976
				P	1-Dec-1983
				W[2]	1-Sep-1992
				Y	1-Nov-1992
				Z[2]	1-Nov-1992
				AA	1-Nov-1992
				EE	1-Jun-1997
				HH	28-Feb-1998
				II	24-Aug-1999
				KK	7-Jun-2000
				LL	1-Jul-2000
G	original in 1960		fission 1961–G dropped	H	1-Jan-1961
				I	1-Jan-1961
GG	S	1-May-1997	removed Feb–March 2002	none	
H	G	1-Jan-1961	removed by end of 1972	none	
HH	F	28-Feb-1998	still on Cayo Santiago	none	
I	G	1-Jan-1961	removed in early 1990	Q	1-Mar-1985
				R	1-Aug-1985
				S	1-Jan-1986
				T[3]	1-Apr-1987
II	F	24-Aug-1999	removed in early 2002	none	
J	A	1-Jan-1965	removed in early 1984	N[2]	Oct–Nov-1973
JJ	R	16-Aug-1999	removed early 2002	none	
K	A	10-Nov-1969	removed by end of 1972	none	
KK	F	7-Jun-2000	still on Cayo Santiago	none	
L	A	10-Nov-1969	removed early 1992	V	Jan–Dec-1991
LL	F	1-Jul-2000	removed 2001/02[1]	none	
M	F	1-Aug-1973	removed early 1984	none	
N	J	Oct–Nov-1973	remerged with J in 1974	none	
O	F	1-Nov-1976	removed April–May 1984	none	
P	F	1-Dec-1983	removed early 1985	none	
Q	I	1-Mar-1985	removed early 1994	none	
R	I	1-Aug-1985	still on Cayo Santiago	BB	1-Jan-1996
				JJ	16-Aug-1999
				T[3]	1-Apr-1987
S	I	1-Jan-1986	still on Cayo Santiago	CC	1-Jan-1996
				DD	1-May-1997
				GG	1-May-1997
T	I+R	1-Apr-1987	removed early 1992	none	
V	L	Jan–Dec-1991	still on Cayo Santiago	none	
W	F	1-Sep-1992	remerged with F in 1993	none	
Y	F	1-Nov-1992	removed early 1995	none	
Z	F	1-Nov-1992	remerged with F Jan 1993	none	

[1] The MS-Access database was still set up to census these groups in January 2005 but returns 0 individuals.

[2] These groups remerged with their source group within a year.

[3] Group T formed initially from R and was later joined by members of T's source group—I (John Berard, pers. comm.).

Table 2.2: Measurements collected on the skeletons of Cayo Santiago mothers. Further explanation of these skeletal measurements can be found in published sources (Hallgrimsson et al., 2002; Hallgrimsson, 1999; Bass, 1995). The column n indicates the number of skeletons on which the measurement could be taken. For cranial length and bizygomatic width this is the number of individuals measured. For all other distances n indicates the number of skeletons on which at least one side was measured.

measurement	abbreviation	n
POST-CRANIUM		
humerus length	humerus	119
humerus anterior-posterior diameter at midshaft	h_ap_dim	119
radius length	radius	119
3^{rd} metacarpal length	mcarp3	118
femur length	femur	120
femoral bicondylar width	f_bcw	120
femoral anterior-posterior diameter at midshaft	f_ap_dim	120
tibia length	tibia	119
3^{rd} metatarsal length	mtars3	117
CRANIUM		
mesial canine-distal M^2 distance	ctom2	114
orbital height	orbht	121
glenoid tubercle-endomolare	glenm1	121
basion-external auditory meatus	baseam	120
lateralmost infraorbital foramen-external auditory meatus	eamiof	121
bizygomatic width	bizyg_w	120
cranial length (alveolare-most posterior point)	cranial_l	121

Figure 2.2: Cayo Santiago feeding bins. Monkeys are provisioned with commercial monkey chow diet distributed at several locations on the island in metal bins. These are archival photos taken in 1985 by Curt Busse and are available online at `http://www.curtbusse.com/` distributed under an open copyright license.

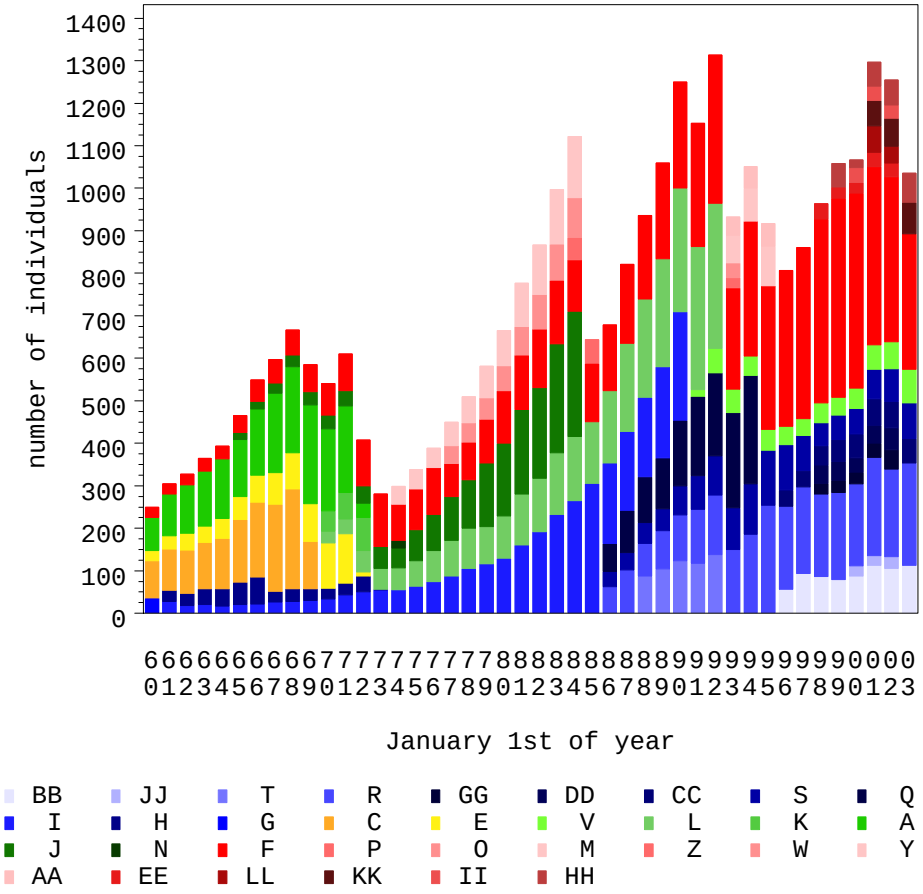


Figure 2.3: Population size of the Cayo Santiago colony on January first of each year. The few extra-group males and individuals unassigned to any group are excluded. Social groups are color-coded in the key at the base of the figure. Daughter groups are indicated in colors similar to their parent. Consult Table 2.1 for further information on the origin and fate of social groups.

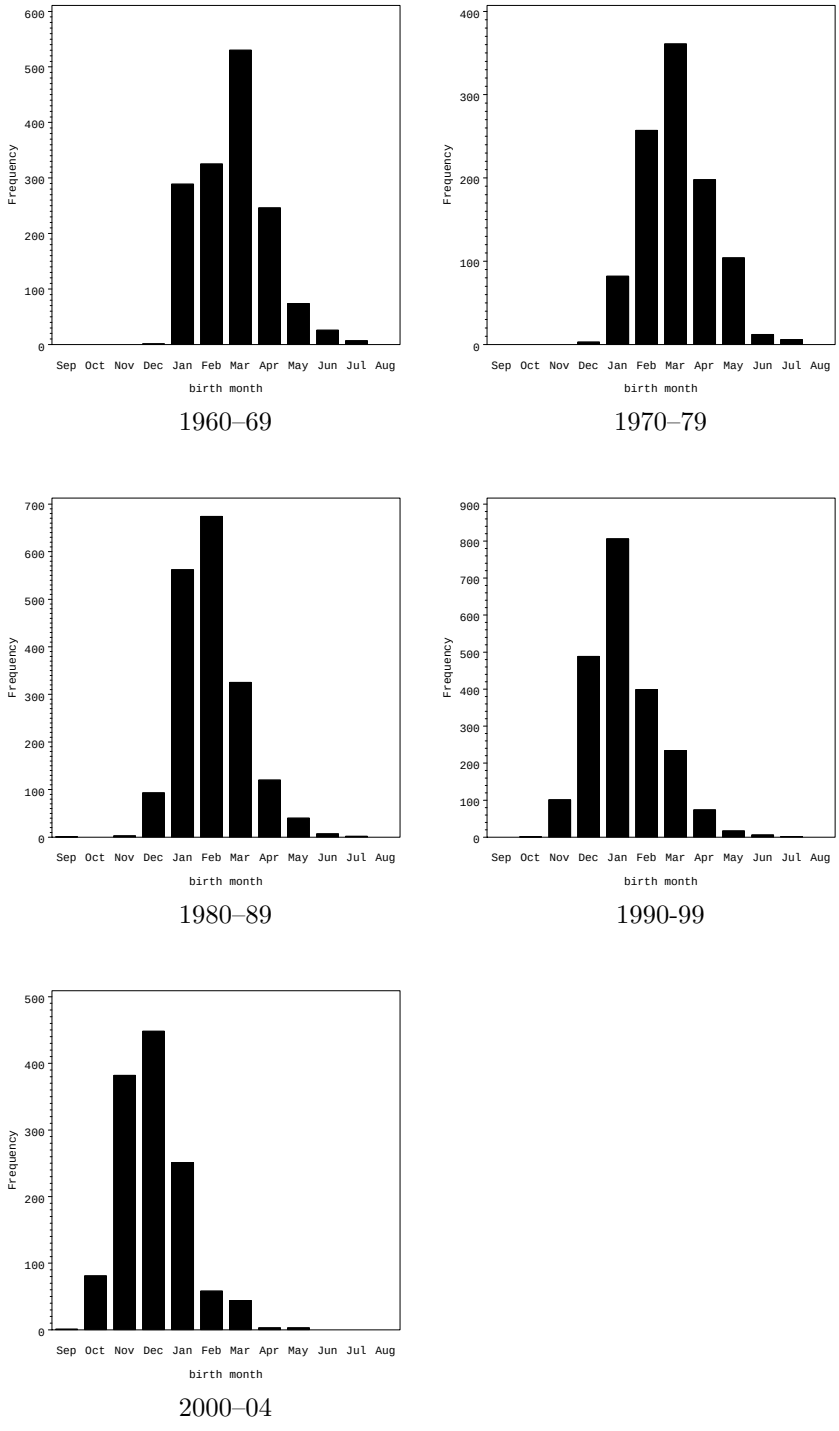


Figure 2.4: Birth seasonality by decade in the Cayo Santiago colony. Each panel displays the frequency distribution by month of births for the indicated time span. The birth season has slowly become earlier.

Chapter 3

Genetic Bases of Primate Life History and Morphology

3.1 Introduction

Understanding the level of genetic variation in animal behavior, physiology, morphology, and life history is a major goal of evolutionary biology. In part, this is because the predicted response a population will make to selection is proportional to the amount additive genetic variance of the trait (Roff, 1997). Additive genetic variance is that due to the independent action of many loci affecting a quantitative trait and is often measured as heritability—the ratio of additive genetic to total phenotypic variance in a trait ($h^2 = \sigma_A^2/\sigma_P^2$). The genetic variance in traits, and patterns of covariation among them, are also important for inferring long-term evolutionary patterns of speciation, adaptation, and drift (Schluter, 1996, 2000; Marriog and Cheverud, 2004; Ackermann and Cheverud, 2004).

Despite the importance of a knowledge of inheritance to understanding evolutionary processes, little is known about the genetic bases of traits in primate populations. In this chapter I estimate the heritability and coefficients of genetic and residual variation for a set of life history and morphological variables for the Cayo Santiago female macaques. In addition to providing basic information on the genetics of these traits, I test several ideas on how genetic variation should be patterned among them. The theoretical predictions themselves are extremely general, and are expected to apply to most animals and plants (Roff, 1997), though they have been explored primarily in mammals, birds, and insects. One set of ideas implicates the action of selection on traits in removing deleterious alleles and fixing beneficial as the primary predictor of their standing genetic variance. Alternatives emphasize physiological and developmental interdependencies among traits that inflate residual variance in traits heavily dependent on the expression of others, or the amount of mutational input traits recieve. Including primates in this type of analysis will clarify if there is anything fundamentally different about the way they experience selection or the genetic architechture of their life histories. Demonstrating a genetic basis to life history traits in the Cayo Santiago females is also an important precursor to later analysis of genetic and phenotypic covariation among these traits (Chapter 5).

3.1.1 Proposed Explanations

The standing level of additive genetic variance for a trait is due to some relationship between input of novel variants from mutation or migration and its elimination by selection and drift. In the ecological literature two hypotheses have been discussed to explain the patterning of genetic variances in traits with fitness. The first, referred to as the *erosion of variance* hypothesis, invokes Fisher's (1930) "fundamental theorem of natural selection"—that the rate of change in fitness is equal to the additive genetic variance in fitness—and Robertson's (1966) "secondary theorem of natural selection"—that the rate of change in a trait under selection is equal to its additive genetic covariance with fitness. Rephrased, these state that, all else being equal, traits with greater correlations with fitness should have less additive genetic variance in populations near an evolutionary equilibrium. Alleles that affect fitness should quickly be driven to fixation or eliminated, and this should occur more rapidly in traits more strongly correlated with fitness. Selection quickly erodes the additive genetic variance in traits closely correlated with fitness. The prevalence of low heritabilities for life history traits, which should be under strong selection, has been argued as evidence in favor of this hypothesis (Gustafsson, 1986; Roff and Mouseau, 1987; Mouseau and Roff, 1987).

A contrasting idea in the ecological literature is that traits with low heritabilities, like life history traits, do *not* have low amounts of additive genetic variance, but instead have elevated amounts of residual variance because they are functionally "downstream" from other morphological variables (Price and Schluter, 1991). For example, age of sexual maturation in many animals is dependent on reaching a critical size threshold. If there is genetic variation for this threshold size and growth rate differences are environmental then the heritability of age of maturation will be considerably lower than the size threshold it depends on (Roff, 1997). Age of maturation is downstream of the size threshold and incorporates the environmental variance in growth rate. I will refer to this as the *incorporation of residual variance* hypothesis. Because of this relationship, the phenotypic variance of traits closely associated with fitness contains the sum of residual variance of their "upstream" variables and any independent residual variance of their own. Symbolically, the heritability of an upstream variable (u) is $h^2(u) = \sigma^2_A(u)/[\sigma^2_A(u) + \sigma^2_E(u)]$, while that for the downstream variable (d) is $h^2(d) = \sigma^2_A(u)/[\sigma^2_A(u) + \sigma^2_E(u) + \sigma^2_E(d)]$. Empirical studies of wild birds and mammals demonstrate life history traits do indeed have large amounts of additive genetic variance when compared with morphological traits on a mean-standardized scale as a coefficient of variation ($CV_A = 100 \times \sqrt{\sigma^2_A}/\bar{x}$) (Kruuk et al., 2000; McLeery et al., 2004; Merilä and Sheldon, 2000, 1999).

However, both sorts of mechanisms may act to explain patterns in heritabilities among traits differently correlated with fitness. In contrast to the scheme proposed by Price and Schluter (1991), if a downstream variable, like a life his-

tory trait, has some additive genetic variance of its own, then its heritability is instead $h^2(d) = [\sigma^2_A(u) + \sigma^2_A(d)]/[\sigma^2_A(u) + \sigma^2_E(u) + \sigma^2_E(d)]$. With stronger selection on life history traits $\sigma^2_A(d)$ will be reduced faster than $\sigma^2_A(u)$ and the common pattern of low heritabilities of life history traits will still be observed. Some evidence suggests this is a more realistic model for the inheritance of life history traits. Crnokrak and Roff (1995) compared published values for the relative amounts of dominance and additive genetic variance in life history and morphological traits. Under the assumption that life history traits are under stronger selection that will erode additive variance quickly, they predicted and indeed found greater dominance variance in life history traits than morphological traits in wild animal populations. This pattern is not predicted by the incorporation of residual variance model for life history traits which have no independent source of additive genetic variance.

Laboratory experimentalists have refined these ideas, particularly in the details of the genetic architecture of traits. Houle (1998) provides a comprehensive and critical review of *Drosophila* studies on genetic variance. He advocates explaining standing genetic variance in traits through the variety of mutational inputs they may experience. Traits closely related to fitness, and total reproductive output itself, are likely to be influenced by many loci, making them a large "mutational target." Additionally, pleiotropy among fitness-related traits may also maintain genetic variation by involving the traits in trade-offs (Rose, 1982). Houle further suggests that traits expressed later in life should have greater variance than those early in life, provided there are temporally cumulative effects of alleles. Finally, traits more closely related to fitness may be more canalized by epistatic interactions among loci such that variation in locus A does not translate into phenotypic variation because it is masked by a modifier locus B (Stearns and Kawecki, 1994; Stearns et al., 1995). However, much of this research remains speculative because of difficulties in clearly defining the size of "mutational targets" and estimating the number of segregating alleles for different traits. Furthermore, some results depend heavily on the life history model used to asses the traits' correlations with fitness. Inferences from such laboratory studies can also be difficult to extrapolate to wild populations with different selective optima and distinct population histories.

3.1.2 Predictions of Proposed Explanations

There are a number of predictions of these hypotheses that can be tested in wild and free-ranging populations. The erosion of variance hypothesis predicts low heritability of fitness and decreasing heritability of traits as their association with fitness increases. At evolutionary equilibrium additive genetic variance in fitness (σ^2_A or CV_A) should be approximately 0. The incorporation of residual variance hypothesis does not require evolutionary equilibrium and does not predict 0 additive genetic variance in fitness or traits closely associated with

it. Its important prediction is that traits functionally dependent upon others have higher residual variance (CV_R). If one accepts that such traits are more closely related with fitness, then it also predicts a positive relationship between a trait's association with fitness and its residual variance (CV_R). More detailed hypotheses on the genetic architecture of traits are difficult to distill into exclusive predictions. However, one prediction of the mutational target hypothesis is that additive genetic variance (CV_A) should be greater in traits more closely associated with fitness. This requires the assumption that fitness is the ultimate mutational target—the sum total of all allelic effects, and that traits more closely associated with it are subject to greater mutational input. These predictions are summarized in Table 3.1.

3.1.3 Review of Previous Studies on Genetic Variation

Little is known about the genetic architecture of or pattern of selection acting upon quantitative traits in primates. This is particularly true of life history and behavioral variables, which are some of the most widely discussed aspects of the order within biological anthropology. The bulk of what is known comes from two sources—human populations and captive primate colonies. Neither is an ideal case for examining the genetic architecture or patterns of selection on morphological, life history, or behavioral traits.

Human populations sampled are either historical or modern groups, each with particular challenges to interpretation. Historical groups are assayed through what are often incomplete demographic registers and typically lack additional information on environmental conditions population members may have experienced (Pettay et al., 2005). Modern groups, though surveyable to gather pertinent environmental and lifestyle information on, live in conditions very different from what they were only a few generations ago including access to health care such as immunization, surgical intervention, and effective mechanisms of birth control (Kirk et al., 2001). Despite these drawbacks, matings in human populations are certainly unmanaged, except by the population members themselves, and measures of lifetime reproductive fitness can usually be calculated for most population members. However it often is not, because most of these studies have a biomedical rather than evolutionary interest. Furthermore, the abundance of humans makes for large sample sizes and good statistical power in these studies.

One objection to studies of genetics and selection in human populations is that humans, particularly in modern populations, do not seek to maximize their genetic representation in future generations (Roth, 2004; Lam, 2003). While valid in a philosophical sense, this is unimportant to evolutionary dynamics. As long as there is variance in fitness there is opportunity for selection and selection is always for higher fitness (Lande, 1982), whether this is attained with greater survival, fertility, ingenuity, altruism, nastiness, or prestige.

Colonies of captive non-human primates offer other challenges. Standardized conditions reduce the range of environments animals are exposed to, likely reducing total variance (Williams-Blangero and Blangero, 1995). Captive conditions may also be very different from the wild in which one would expect the population to be close to equilibrium values for gene frequencies and heritabilities. This may also inflate total variance in traits, but it depends on the gene×environment interactions between wild and captive conditions. This is similar to the situation of studies of modern groups of human living in conditions very different from those in the recent past. Breeding in captive primate colonies is typically tightly managed to maintain genetic diversity rendering measures of lifetime reproductive fitness uninterpretable. Consequently, correlations between traits and fitness cannot be calculated.

For clarity, these hurdles to interpretation of evolutionary dynamics in human and captive primate populations can be rephrased in the language of Section 2.4. In human populations selection gradients ($\boldsymbol{\beta}$) and differentials ($\mathbf{S}$) can be estimated though there is difficulty in knowing how similar these patterns of selection are to what they were in the past. In captive primate colonies selection usually cannot be measured because breeding is managed. Because kinship is usually well known in human populations and primate colonies, $\mathbf{G}$ can be estimated without much difficulty (see Section 2.5). However, the heritabilities and genetic correlations in $\mathbf{G}$ may be difficult to interpret because of changing environmental conditions for modern human populations or between wild and captivity in primates.

However, there is a third source of comparative information. This is long-term studies of evolutionary dynamics in wild mammal and bird populations. These studies resolve most of the potential drawbacks noted in primate and human work in the estimation and interpretation of $\boldsymbol{\beta}$ and $\mathbf{G}$, but are unfortunately conducted in taxa very different phylogenetically and ecologically from primates. In a number of elegant studies, precise estimates of selection, genetic architecture, and response have been made. Moreover, the temporal depth of these projects has provided evidence of changing patterns of selection with shifts in weather, population density, and human habitat alteration. Recent work in one wild primate population has documented patterns of selection and estimated heritabilities of quantitative morphological traits (Lawler et al., 2005; Lawler, 2006).

Humans

There have been many studies on the heritability of different aspects of human life history and somatic development. Gerontologists have investigated familial patterns of human longevity in the heritability of lifespan by analyzing data sets of twins, parent and offspring pairs, or extended pedigrees. While most of these studies have supported a small to modest heritability (h^2 range 0.10–

0.50, see: Lee et al., 2004; Mitchell et al., 2001; Gudmundsson et al., 2000), some have found none and others have documented important environmental factors confounding simple quantitative genetic analysis. Individuals in the same generatation tend to have more similar lifespans than those in different generations. This is likely due to secular trends in human lifespan over the last few centuries. Interestingly this phenomenon is not seen in the Amish, where environments are inferred to be similar across generations (Mitchell et al., 2001). Additionally, heritability of lifespan in Swedish twins reared apart was significantly lower than that of twins reared together (Ljungquist et al., 1998). This also suggests that environmental effects masquerade as genetic ones in many of these studies where there is little difference in environments among related individuals. This is particularly problematic for twin studies, but less of an issue when dealing with extended pedigrees.

A further difficulty in comparing among studies of human longevity is different criteria for inclusion of recorded lifespans in each analysis. Some authors use only individuals dying over age 15, other restrict analysis to deaths over 65, or other cut-points in between. Intuitively this is problematic as most people recognize leading causes of death for teenagers are different from those of retirees. There is also evidence of stronger familial effects on early adult life mortality and that the genetic underpinnings of mortality in the very oldest of the old are different from those in the rest of the population (McGue et al., 1993). This data censoring is also a hindrance to evolutionary analysis. While these studies are generally not conducted with evolutionary questions in mind, it is important to recognize who should be included when estimating heritability of lifespan to predict the proper dynamics of evolutionary response. The quantitative genetic answer to this question is that an individual's phenotype is the expression of its genes and the individual should thus "own" whatever lifespan it exhibits (Cheverud and Moore, 1994). Attributing sub-adult death to the mother or some other factor potentially gives misleading evolutionary dynamics (Wolf and Wade, 2001). With selection on lifespan the heritability of total lifespan for all measurable individuals, regardless of their age at death, should be used for predicting response.

Further work with human populations has identified familial patterning to age of menarche, first reproduction, menopause, and total fertility. Menarcheal age is modestly to highly heritable in nearly all studies of its inheritance in human populations (h^2 range 0.32–1.0, reviewed in Towne et al., 2005). Those studies using relatives in multiple households typically find heritabilities around 0.50. Estimates of heritability of total fertility (fitness) tend to be low in human populations(h^2 range 0.04–0.42, reviewed in Madrigal et al., 2003). In a recent study of Australian twins fitness was found to have moderate heritability (0.39) comparable to that for ages of menarche (0.50), first reproduction (0.23), and menopause (0.44) in the population. Only age of first reproduction covaried strongly with fitness in this data set (r_A=-0.21). Another recent study of rural

Finns from the 18th to 20th centuries also documented high heritability of total fitness for women (0.31 or 0.56 depending on cases included, Pettay et al., 2005). Genetic correlations involving fitness traits in trade-offs may have contributed the maintenance of high heritability of fitness in this population.

Non-human Primates

Olive baboons (*Papio hamadryas anubis*) at the Southwest Foundation for Biomedical Research have been the subject of two studies on heritability of life history traits. Both showed surprisingly large additive genetic components of variance in female age of first reproduction (h^2=0.87, Williams-Blangero and Blangero, 1995) and adult lifespan (h^2=0.23, Martin et al., 2002). Adult body mass is also known to be moderately heritable in this population (h^2=0.51, Jaquish et al., 1997). Other studies on morphometric traits in this population have yielded a range of heritabilities. For example, the heritabilities of relative mass of brain, heart, kidneys, and liver were all between 0.37 and 0.60. Additionally, the brain liver and kidneys were all related by significant positive genetic correlations (Mahaney et al., 1993). Molar tooth dimensions are highly heritable ($h^2 > 0.67$) as is molar enamel thickness ($h^2 > 0.32$) (Hlusko et al., 2002, 2003). A variety of physiological traits have also been studied in this population (references in Rogers, 2005).

Published heritability estimates for life history traits are available for several other captive primate populations. Litter size at birth and 2 weeks after parturition in three callitrichine species (*Callitrhix jacchus*, *Saguinus fuscicollis*, and *S. oedipus*) were explored in the colony from the Oak Ridge Associated Universities Marmoset Research Center (Jaquish et al., 1996). While there was substantial genetic variance in litter size at birth for each species the heritability was only significantly greater than 0 in *S. fuscicollis* (h^2=0.31). Heritability of litter size at 2 weeks was not statistically greater than 0 in any of the species, being largest in *S. fuscicollis* (h^2=0.19). For both measures this indicated residual variance was very large. Genetic correlations between litter size at birth and 2 weeks were estimated but none were significantly different from 0. A significant, negative residual correlation was found in *C. jacchus* suggesting environments that were good for litter size at birth were bad for litter size at 2 weeks.

Some other aspects of the quantitative gentics of this colony are known. Heritability of adult body mass in the *S. fuscicollis* members was 0.35 (Cheverud et al., 1994). There was also the suggestion of a paternal effect of care with an increased h^2=.54 when only using paternal pedigree links. Heritability decreased when only using maternal links, implying no maternal effect on body mass. However, both of these estimates fell within the range of the original and are not statistically significant differences. The authors also provided coefficients of additive genetic variation (litter size CV_A=10.63, body mass CV_A=8.0) for comparison with heritabilities. With only two points—litter size at birth and

body mass in *S. fuscicollis*—they were limited but found a negative relationship between CV_A and heritability. This emphasizes that the low heritability of litter size is not due to reduced additive genetic variance as would be predicted by the erosion of variance hypothesis. This is important, if selection is inferred to be stronger on litter size than body mass, because it indicates selection is not effectively eroding additive genetic variance. Incorporation of residual variance and mutational target size hypotheses accomodate this pattern. Some additional aspects of this cotton-top tamarin (*S. oedipus*) colony are known. A set of 39 cranial features had a mean heritability of 0.45. The heritability of risk of colon cancer was also 0.17, which is not significantly different from 0 (Cheverud et al., 1993).

Birth weight in pig-tailed macaques (*Macaca nemestrina*) was shown to be heritable in the Washington Regional Primate Research Center colony (Ha et al., 2002). Not surprisingly there were significant maternal effects, as well. The heritability was 0.51 when maternal effects were excluded from linear model and 0.23 when they were included. The maternal effect variance was of approximately the same size. Maternal effects are expected to be strong on early life traits such as neonatal body mass which should rather directly reflect maternal investment during gestation (Cheverud and Moore, 1994; Cheverud, 1984).

Lawler et al. have tested several hypotheses on primate development and mating behavior in wild sifakas (*Propithecus verreauxi verreauxi*) using quantitative genetic techniques. Limb element lengths in juvenile sifakas have heritabilities between 0.44 and 0.16. The lowest value was found in the foot which he suggested was due to the stronger action of selection, following the erosion of variance hypothesis. Because this is a wild population studied over many years, he was able to measure the selection gradients on the size of each limb element. The foot is, in fact, under the strongest selection (β=0.20 versus $|\beta|$s<0.07) as larger footed sub-adults have greater survivorship (Lawler, 2006). As relatively large hands and feet are common in primate juveniles this was suggested as selection operating to maintain a common allometric pattern in this population. No attempts to estimate quantitative genetic statistics for life history variables in this population have been published.

Other Mammals and Birds

Studies of genetics and selection in the wild are far more common in non-primate mammals and birds (Kingsolver et al., 2001; Grant, 1986; Endler, 1986). Data from several long-term studies of mammals and birds have been used to directly test the erosion of variance and incorporation of residual variance hypotheses. Kruuk et al. (2000) compared heritabilities and coefficients of additive genetic and residual variation in wild red deer (*Cervus elaphus*). Using a dataset of 6 life history and 3 morphological traits, they found declines in trait heritabilities with increasing correlation with fitness—a generic pattern both the ero-

sion of variance and incorporation of residual variance models were proposed to explain—along with increasing CV_R, which is only predicted by the incorporation of residual variance model. Low heritability of fitness in this population is primarily due to low heritability of adult or total lifespan. There was no association between CV_A and trait correlation with fitness. In Soay sheep (*Ovis aries*), fecal egg count—a measure of parasitism thought to be under strong selection—has a low heritability (0.11–0.14). This is much lower than most morphometric traits in the population (0.28-0.35). However, there is a large amount of additive genetic variance for fecal egg count, suggesting the incorporation of residual variance rather than the erosion of additive gentic variance accounts for this (Wilson et al., 2004; Coltman et al., 2001).

Similar patterns were found in great tits (*Parus major*, McLeery et al., 2004) and collared flycatchers (*Fideculla albicollis*, Gustafsson, 1986; Merilä and Sheldon, 2000). Merilä and Sheldon (2000) found the suggestion of a positive relationship between CV_A and correlation with fitness. This would accord with the mutational target hypothesis. While some of these studies have estimated maternal effects, none have estimated dominance variance for these traits. This is the needed information to test Crnokrak and Roff (1995) idea that combines erosion of variance by selection and incorporation of environmental variance in fitness traits.

Other studies of wild mammals have found high heritabilities of fitness or traits closely correlated with it. Reale and Festa-Bianchet (2000) estimated number of offspring to have a heritability of 0.66 and 0.19 in two neighboring populations of bighorn sheep (*Ovis canadensis*). Kelley (2001) also found high heritability of fitness ($h^2 \approx 0.90$) in Serengeti cheetahs (*Acinonyx jubatus*).

Summary

The currently available evidence on the quantitative genetics of primate traits associated with fitness suggests they are little different from other well-studied populations of mammals and birds. Furthermore, the limited evidence from non-human primates addressing the predictions of different hypotheses currently can only be said to accomodate either the erosion of variance or incorporation of residual variance models. Using the current evidence for testing between them requires assumptions about how strong selection is on each trait. Cheverud et al. (1994) offer a small attempt to do this, which does not match the erosion of variance hypothesis.

3.1.4 Conditions of the Cayo Santiago Sample

Cayo Santiago presents some difficulties in assessing what factors contribute to standing genetic variance, given its history and management. Source animals were trapped from a variety of different places in India, meaning that the colony may have been genetically diverse to begin with and experienced

outbreeding depression through the breakdown of favorable epistatic interactions when released onto Cayo Santiago. This would mean a loss of canalization of fitness-related traits. Transplantation into a novel environment which may have different optimal phenotypes may also have displaced the population from evolutionary equilibrium. Management practices have also kept the population from stable age structure at times. Stable age structure is a requirement for true evolutionary equilibrium, though small departures do not matter much (Charlesworth, 1994). The population bottleneck of the late 1940s and early 1950s reduced numbers to around 180 individuals, making the effective size of the population considerably smaller. Duggleby et al. (1986) state that 88% of the population in 1972 was derived from only 15 females alive in 1956. Their relationships are unknown but have been presumed, perhaps inaccurately, to have been unrelated.

Bottlenecks have been experimentally shown to convert epistatic to additive genetic variance in some laboratory studies (Bryant et al., 1986; Cheverud et al., 1999). This occurs through the elimination of alleles at some modifier locus B which affects expression of alleles at locus A (Templeton, 1980; Goodnight, 1987, 1988). This is unlikely to have played a role in Cayo Santiago's history, as the estimated effective population size in the mid-1950s is around 70. Most experimental and theoretical work has relied on very small bottlenecks (e.g. 2 individuals), and experimental work often shows reduction of additive genetic variance during bottlenecks of this size. Cayo Santiago's population has never been small enough for this to have occurred to any detectable degree. More generally, additive genetic variance is expected to decrease during bottlenecking, due to inbreeding causing the expression of deleterious recessive traits and the random loss of alleles through sampling effects in small populations (Roff, 1997).

The role of these processes in the history of Cayo Santiago is difficult to assess. Blood protein studies in the 1970s found little evidence of inbreeding (Duggleby et al., 1986). Furthermore, the heterogeneous sources of the Indian macaques founding the colony may have perturbed the population from evolutionary equilibrium. A few generations of random mating among the subpopulations should have eliminated any linkage disequilibrium resulting from this mixture. Population processes *after* the beginning of accurate demographic records will not substantially affect the estimation of quantitative genetic statistics in this study. They are robust to selection and inbreeding, provided paternity assignment is available such that it is known to have occurred. The estimates apply to the population alive in the mid-1950s (Kruuk, 2004; Mrode, 1996).

3.1.5 Hypotheses for the Cayo Santiago Females

Considering the history of the Cayo Santiago population, and the results of previous studies on humans, non-human primates, and other mammals and

birds, the following predictions can be made on the patterning of additive genetic and residual variance in female life history and morphology. The predictions of each available explanation are given in Table 3.1 and Section 3.1.2. Generally, one might expect any of these criteria to be satisfied. However, the history of Cayo Santiago suggests it may depart from equilibrium and thus predictions based on the erosion of variance view can only be weakly tested. If trait h^2 and CV_A decline as their correlation with fitness increases then this explanation is supported, but not observing this pattern does not refute it. The other explanations do not require equilibrium populations for testing. If trait CV_R increases as trait correlation with fitness increases then the incorporation of residual variance view is supported; if not is should be rejected. Furthermore, if trait CV_A increases with increasing trait correlation with fitness then the mutational target size view is supported; if not it should also be rejected. Note that the critical tests for each of these later two explanations leave the possibility of both being equally supported. They can operate simultaneously.

Additionally, correlations between heritabilities and coefficients of variation clarify whether changes in additive genetic variance or residual variance are responsible for change in heritability. If h^2 and CV_A are *positively* correlated then differences in heritabilities can be attributed to changes in levels of additive genetic variance. If h^2 and CV_R are *negatively* correlated then differences in heritabilities can be attributed to changes in levels of residual variance. The erosion of variance view predicts the positive relationship between h^2 and CV_A. The incorporation of residual variance view predicts the negative relationship between h^2 and CV_R.

3.2 Methods

A set of 15 morphological measurements were used in this portion of the study (Tables 2.2 and 3.2). Measurements were selected for comparisons with previous heritability estimates by Hallgrimsson et al. (2002) and Lawler (2006). Additionally, these measurements are spread throughout the cranial and post-cranial skeleton and should capture developmentally and functionally separate aspects of skeletal variation. Data were collected from the skeletons of individual females born between 1957 and 1990 (see Chapter 2). All individuals in this data set were sexually mature adult females who reproduced at least once and died naturally on Cayo Santiago or were removed and immediately sacrificed. No significant differences were found between removed and naturally dying individuals for any of these morphological traits. Measurements were taken on the left and right side of each individual if possible and these values were averaged for this analysis. All morphological measurements were normally distributed.

Life history variables were used from females born between 1960 and 1990 for all variables except age of first birth for which birth cohorts up to 1999 were accepted. I defined the total number of offspring born to a female as a fitness

indicator (# offspring). This was calculated in two ways. First, only females that reproduced were included in the measure, censoring those that died prior to maturity or reached reproductive age but never reproduced. Second, all females who died on Cayo Santiago were included, assigning a 0 for females who did not reproduce. A similar approach was taken to lifespan which was measured once for females that reproduced, and once for all females with recorded deaths. Two other life history variables were used. Age of first reproduction is the cohort age of the female when she gave birth to her first offspring. Birth seasonality at Cayo Santiago ensures that cohorts are all roughly of the same age. Females with first births after their sixth year were excluded because of potential pathology. With these restrictions, age of first birth is an ordinal variable taking only integer values between 3 and 6. The final life history variable analyzed is mean interbirth interval (mean IBI). This is the average number of integer years between successive births by a female. Only females reproducing three or more times had this variable calculated (minimum of two intervals to average). Age of first reproduction and mean IBI are roughly normally distributed. Lifespan and number of offspring are not, but this does not substantially affect the estimates of quantitative genetic statistics (Section 2.5). Lifespan and number of offspring have left-truncated distributions and are strongly positively skewed. Their variances are also larger than their means. This affects their CVs, making them larger than then would be if they had a truly normal distribution (Kruuk et al., 2000).

For the life history variables only a single pedigree of interlocked individuals was used for the quantitative genetic analysis. This pedigree involves 6543 known individuals, in 17 matrilines connected by paternities. This is 82.43% of the entire demographic database. Morphological data were only used on individuals belonging to this same pedigree and one other containing 55 individuals. Pedigree membership was identified with PEDSYS (Dyke, 1996).

A linear mixed model was used for quantitative genetic analysis of the morphological and life history traits in the program DFREML 3.1 (Section 2.5 Meyer, 2000). Fixed effects to be included in the model were first tested in general linear models in SAS. For the morphological traits four fixed effects were tested: matriline social rank, natural death/removal, age at death, and contemporary group. Contemporary group was used to control for temporal changes in colony population size, management, and weather. Contemporary groups were defined as 5 year intervals of birth cohorts beginning in 1960. Animals born prior to 1960 were assigned to a separate contemporary group. Rank and removal were not significant in for any traits. Age at death and contemporary group were used for traits which their p-values were less than 0.10. The only fixed effects for the life history variables were matriline social rank and contemporary group. These were significant or nearly significant for all variables except adult lifespan. Analyses with and without the non-significant predictor rank for this variable were nearly equivalent and only the results including rank

are presented here. The only random effects in the model were animal identity and the residual. A maternal effect was explored by fitting the additional random effect of maternal identity, but it was not significant for any of the variables. It was estimated to be 0 for all the morphological traits, but small values were estimated for some of the life history variables. Dropping the maternal effect had little effect on the heritabilities of any traits. Heritabilities and coefficients of additive and residual variation were calculated by formulae given in Section 2.5. Significance tests for the heritabilities are derived from z-scores computed by dividing the heritability by its standard error and examining z-tables for p-values. Dominance and epistatic variance cannot be calculated in this population. The low frequency of full-sibships when paternities are known argues against dominance playing much of a role in phenotypic resemblance in these traits among siblings and should not pollute estimates of additive genetic variance. Any dominance and epistatic variance should be included in residual variance.

Trait correlations with fitness were assessed with a Pearson's correlation (r) between the trait and the uncensored value for number of offspring. This is the most basic method of measuring this relationship, but the difficulty of relating morphological measurements in any other ways to fitness precludes use of more rigorous techniques such as sensitivity analysis (Houle, 1998). Two traits negatively correlated with number of offspring, age of first reproduction and mean IBI, were "positivized" by taking their absolute value. The association between these correlations and trait heritabilities and coefficients of variation was measured with the Spearman rank correlation (r_s).

3.3 Results

3.3.1 Heritabilities

Heritabilities of morphological traits ranged from 0.017 to 0.731, and 0.072 to 0.474 in life history traits (Table 3.2, Figure 3.1). When trait correlations with fitness were low (age of first reproduction and mean IBI), life history traits had substantially lower heritabilities than morphological traits. As would be expected from the erosion of variance hypothesis, there was a generally weak decline in heritability with increasing correlation with fitness (Figure 3.1). This was strongest in the morphological traits. However, the opposite pattern was seen in the life history variables. In contrast to predictions of the erosion of variance view, increasing correlation with fitness increased the heritability of life history traits. However, none of these patterns reach statistical significance (Table 3.3). Including females that died before reproducing more than doubles the heritability of lifespan and number of offspring, suggesting there is a great deal more additive genetic variance or less residual variance when including sub-adult survival in the measures. These two uncensored measures are both

statistically significant as is the heritability for age of first reproduction. The heritability of the majority of the morphological traits are statistically significant (9 of 15), and the bulk of those are postcranial (7 of 9).

3.3.2 Coefficients of Variation

The patterns in heritabilities are explained, in part, by the coefficients of variation (Figure 3.2). As predicted by the incorporation of residual variance hypothesis, the coefficient of residual variation increases significantly as the trait correlation with fitness increases ($r_s = 0.577$, $p = 0.006$). This pattern holds in both the morphological and life history traits, though it is much stronger in the life history traits.

Coefficients of additive genetic variation show no overall trend ($r_s = 0.281$, $p = 0.217$), though there is a nearly significant increase with fitness in the life history traits ($r_s = 0.754$, $p = 0.084$). This is because CV_A for age of first reproduction and mean IBI are similar to morphological traits, but those for lifespan and number of offspring are much higher. A positive relationship between CV_A and correlation with fitness was predicted by the mutational target size hypothesis. The nearly significant relationship for the life history traits is weak support for this model.

CV_R for life history traits are always higher than morphological traits, suggesting that they are more responsive to environmental inputs. For lifespan and number of offspring, both CV_A and CV_R are higher when including females that died before reproducing. Although both CVs increase, the increase is much greater in CV_A implying there is relatively more additive genetic variance in these traits when including sub-adult survival. Note that this also causes the elevation in heritability for these traits when all cases are included.

3.3.3 Correlations between h^2 and CVs

The quantitative genetic statistics themselves are highly correlated in some respects (Table 3.4). Importantly, CV_R and heritability are strongly negatively correlated ($r_s = -0.757$, $p < 0.001$), but CV_A is uncorrelated with heritability ($r_s = -0.001$, $p = 0.996$). This agrees with the prediction that heritabilities decline with increasing correlation with fitness because of increased residual variance (incorporation of residual variance), not reduced additive genetic variance (erosion of variance). The correlation between CV_A and CV_R is also significantly positive, indicating that traits with greater genetic variance also have larger residual variance. However, this must be interpreted cautiously as the CVs must have some correlation because of division by the same mean.

3.4 Discussion

3.4.1 Match with Theoretical Predictions

This chapter addressed genetic inheritance as a cause of variation in life history and morphology for primate females because of the centrality of inheritance to evolutionary processes. A wide range of heritabilities and coefficients of additive genetic and residual variation was identified that reflects differences in genetic structure for traits that are physiologically or developmentally dependent on other traits. These results offer support to the incorporation of residual variance model (Price and Schluter, 1991). Traits closely correlated with fitness in this population have lower heritabilities because of increased residual variance, not reduced additive genetic variance as suggested by the erosion of variance model which overly prioritizes the action of selection (Fisher, 1930; Roff and Mouseau, 1987; Mouseau and Roff, 1987). The incorporation of residual variance model is the only hypothesis which predicted any of the important patterns observed.

Little support was found for the mutational target size hypothesis (Houle, 1998). The predicted increase in coefficients of additive genetic variance with increasing correlation with fitness was not found overall, though there was a nearly significant trend in this direction in the life history variables. Life history traits more directly tied to fitness may be larger mutational targets, but this is not the case for the morphological variables.

Furthermore, the negative relationship between coefficients of additive genetic variation and fitness, predicted by the erosion of variance view, was not found nor was the predicted strong decline in heritability with increasing correlation with fitness observed. However, because the Cayo Santiago population is unlikely to be in equilibrium these are weak tests of the erosion of variance hypothesis.

3.4.2 Comparison with Previous Studies

Heritabilities of morphological traits from the Cayo Santiago population in previous studies are comparable to values reported here. Animal model estimates of heritabilities are often lower than those from parent-offspring regression, but this does not appear to be the case in with this study. Cheverud (1982) found a range of values from -.040 to 0.866 with a mean of 0.327 in a set of 56 cranial linear distances. In other analyses, non-metric cranial characters had somewhat higher average heritabilities (Cheverud and Buikstra, 1981a) as did cranial capacity and surface features of the brain (Cheverud et al., 1990).

Hallgrimsson et al. (2002) calculated heritabilities on many of the same measurements reported here. Surprisingly, their measurements are essentially uncorrelated with those in Table 3.2 ($r_s = -0.063$, $p = 0.845$). One pattern they noted was decreasing heritability as one moved distally down the limb. The opposite pattern is reported here. Additionally, the average heritability

reported here is higher than their study (0.44 versus 0.34). Several factors may contribute to these differences. Disagreement between animal model and regression heritabilities are well documented, but they tend to be ordered similarly. Inclusion of fixed effects to eliminate temporal variation may also contribute differences. Somewhat different data sets were used in each analysis, with only females who reproduced included here and a mixed-sex set of individuals over 5 years analyzed by Hallgrimsson et al. (2002). Furthermore, left and right side measurements were averaged in this study and it is unclear whether this was done by Hallgrimsson et al. (2002).

Based only on the heritabilities reported in their study and the heritabilities and correlations of the traits with fitness shown here, their study does not support the erosion of variance hypothesis while those reported here do. Like Hallgrimsson et al. (2002), Lawler (2006) found descending heritability of limb segment length as one moves down the limb in young sifakas. However, his analysis included selection gradients on these limb elements. This showed that traits under stronger selection also had lower heritabilities. According to both Lawler (2006) and the results reported here, selection may sufficiently erode genetic variance in limb elements to reduce their heritabilities, but which elements are under stronger selection and thus have reduced heritability can vary among taxa or populations. Caution should be exercised in accepting this conclusion, as the erosion of variance hypothesis recieved no general support in this study, though it is difficult to deploy in the case of the Cayo Santiago population. Furthermore, selection was indexed rather crudely as the bivariate correlation between each trait and lifetime fitness.

Comparison of the heritabilities of age of first reproduction and censored lifespan with the Southwest Foundation baboons illustrate some differences. Age of first reproduction is quite high in the Southwest baboons but low in the Cayo Santiago females (Williams-Blangero and Blangero, 1995). In constrast, adult lifespan has a lower heritability in the baboons than in macaques (Martin et al., 2002).

As noted in Section 3.1.3, studies of wild mammal and bird populations have provided support for the incorporation of residual variance hypothesis (Merilä and Sheldon, 1999, 2000), and some limited support for the mutational targer hypothesis. The results of this study are quite similar. However, the modest heritability of fitness in the Cayo Santiago females requires further explanation.

First, it should be noted that although fitness is predicted to have very low heritability, the results of this study are not unusual. Studies of human populations and wild mammals have calculated large heritability of fitness (e.g. Pettay et al., 2005; Reale and Festa-Bianchet, 2000; Kelley, 2001). Second, there are a number of processes that could result in modest-high heritability of fitness. One factor that can maintain additive genetic variance in traits closely related to fitness is antagonistic pleiotropy. This cannot suffice as an explanation for fitness itself, but may function for lifespan. If lifespan is negatively genetically

correlated with other traits under strong selection, such as early fertility (see Chapter 5), additive genetic variance may be preserved (Rose, 1982). Additionally, there may be a role for phenotypic plasticity in explaining the modest heritability of lifespan and number of offspring. If the environments rhesus macaques in the wild generally encounter are quite different from Cayo Santiago it is possible that genotypes express different phenotypes in this novel environment. Perhaps there is little additive genetic variance relative to environmental variance in the wild, but at Cayo Santiago non-parallel reaction norms cause an increase in additive genetic variance (see p. 206 in Roff, 1997). Finally, fluctuating selection pressures based on cycles of rapid evolution of parasite resistance has been suggested as a source of true heritability of fitness (Eshel and Hamilton, 1984). Any of these might operate in the Cayo Santiago females.

However, the simplest explanation for modest heritability of fitness is the reduction of residual variance for fitness in the homogenous, mild environment of Cayo Santiago. While CV_Rs for lifespan and number of offspring are high, they would likely be much higher without provisioning or with greater spatial variation in environments. The difference in heritability between the censored and uncensored h^2 for number of offspring (0.088 versus 0.336) suggests this effect is largely due to females ever reproducing. This depends heavily on subadult mortality. Cayo Santiago is a generally benign environment for subadults with little disease and no predation, which—assuming their random action—would drive down heritability for total lifespan and thus number of offspring.

3.4.3 Data Censoring for Lifespan and Fitness

These considerations suggest that estimates of heritabilities and other quantitative genetic statistics are sensitive to the censoring of cases, particularly for measures of lifespan and total fitness. There is ample *a priori* justification for including females that never reproduce in lifespan or number of offspring when calculating their heritability. A large amount of information on reproductive success is lost by excluding these females. This can be quantified according to the following expression from Grafen (see Brown, 1988)

$$p(\sigma^2_{Pb}) + p(1-p)\bar{x}^2_{Pb} \tag{3.1}$$

where p is the proportion of females that breed [$p = n_b/(n_b + n_f)$ with n_b as the number that breed and n_f as the number that fail to breed], σ^2_{Pb} is the phenotypic variance in fitness for reproductive females, and $\bar{x}_{Pb}$ is the mean fitness of reproductive females. The terms on the left of the addition are the fraction of the total variance due to reproductive females; those on the right are variance due to non-reproducers. Using the values in Table 3.2 only 51.3% of the variance in lifetime fitness is due to females that reproduced. The remaining half of the variance is due to non-reproducers. As the opportunity for selection

is the total mean-standardized variance in fitness, the best estimate for the variance in total fitness should be used, rather than half of it (Section 2.4.2).

Furthermore, simulation studies in animal breeding have demonstrated that censoring observations in this fashion tends to downwardly bias estimates of additive genetic variance (Burns et al., 2006; Vukasinovic et al., 1998). Removing individuals from the analysis who die before a cut-point age, or who never reproduce will yield lower estimates of additive genetic variance, and, depending on the magnitude of residual variance, lower heritability estimates. For example, in the study of Burns et al. (2006), the more data censored (10–25%) the more depressed the heritability estimate was (11–31%). As in the case under discussion here, the data censored were not selected at random, but were the poorer performing individuals.

While this effect can be seen in the results for the Cayo Santiago females, several studies of wild bird and wild mammal populations and one preliminary study of a human population have found ≈ 0 heritability of female fitness regardless of censoring (Merilä and Sheldon, 2000; Kruuk et al., 2000; McLeery et al., 2004; Gustafsson, 1986; Esparza et al., 2006). In these populations which are unmanaged, and probably closer to evolutionary equilibrium, censoring has little effect on the heritability estimates.

3.4.4 Incomplete Data in Managed Populations

One concerning possibility for the appearance of familial patterning to measures of uncensored lifespan and number of offspring is the removal of subadults. In attempting to analyze the survival rate of females to reproductive age (4 years) fully 32.5% (1034/3186) were removed through colony management. This was a greater fraction than those that die 22.8% (727/3186). Furthermore, there is a bias to these removals. Because higher ranking matrilines produce more offspring that survive infancy, they have more of them removed by colony managers (see Table 4.6). The subadult deaths that are observed, and were analyzed in this chapter, over-represent lower ranking matrilines. In this situation, no response to selection toward higher mean fitness or longer average lifespan would be observed, because the biased removal of higher ranking matrilines offsets the deaths of lower ranking matrilines. The lifespan or fitness of this invisible segment of the population that removal creates cannot be analyzed.

This is an intractable problem of working with a managed population that lies somewhere between the extremes of a laboratory population, in which breeding and survival can be purposefully manipulated, and the wild, where there are no concerns of "unnatural" removal biases. Primate populations unregulated by human intervention would be better opportunities to test evolutionary predictions on the standing level of additive genetic variance in fitness and fitness-related traits (e.g. Gombe chimpanzees, Amboseli baboons, see Strier et al., 2006). The quantitative genetic methods now available for dealing with

unbalanced pedigree information that arises in studies of natural populations, eliminate previous obstacles to genetic analysis in the wild (Moore and Kukuk, 2002; Kruuk, 2004).

3.4.5 Future Directions

Despite these difficulties, some predictions on the genetic architechture of traits related to fitness in the Cayo Santiago females can be tested. The incorporation of residual variance hypothesis (Price and Schluter, 1991) appears to be widely applicable to mammalian and avian populations, whether they are in evolutionary equilibrium or not. Traits closely related to fitness can have large additive gentic variances, that may get translated into sizable heritabilities when environmental conditions change such that the residual variance is reduced. If fitness-related traits have large amounts of additive genetic variance, they may well "share" variance and be genetically correlated with one another. Genetic correlations of this kind are likely to be important in mediating trade-offs among fitness-related traits. Some analysis of these types of correlations is reported in Chapter 5.

Another focus for future studies is to refine our understanding of what is contained within the residual variance (σ^2_R or CV_R). Traits closely associated with fitness are widely thought to have large amounts of dominance and epistatic variance (Merilä and Sheldon, 1999; Crnokrak and Roff, 1995). This implies that they will maintain genetic variation and respond asymmetrically in the face of strong selection. Furthermore, residual variance includes the effects of environmental inputs—such as diet, climate, microhabitat, disease, and injury. The ability of humans, and to some extent other animals including primates, to control their environments suggests the potential for behavioral mechanisms that strongly impact fitness, but may not be under direct gentic control. In the following chapter one such mechanism, social rank in female macaques, is analyzed from this perspective.

3.5 Summary

The heritability and coefficients of additive genetic and residual variation were estimated for a set of morphological and life history traits for the Cayo Santiago females with the aim of testing predictions about the standing level of additive genetic variance in these traits based on their relationship with fitness. Morphological traits have higher heritabilities and lower additive genetic variance than life history traits in this population. Fitness itself appears to be modestly heritable (0.336), though the management practices of Cayo Santiago seriously complicate the interpretation of this result for testing predictions of the erosion of variance hypothesis (Fisher, 1930; Robertson, 1966; Mouseau and Roff, 1987). Future study in primate populations free of the complications of

systematic culling will help resolve the utility of this hypothesis.

However, other evidence is consistent with the incorporation of residual variance explanation (Price and Schluter, 1991) for the low heritabilities of life history traits. Even in this less than ideal dataset coefficients of residual variation are higher in traits strongly correlated with fitness. This is true for morphological and life history traits. There was little trend in the coefficients of additive genetic variation implying mutational target size was a poor explanation for the overall pattern, but may function for the life history traits. In summary, the results implicate the developmental and physiological relationships among traits as determinants of their heritabililties, as opposed to the mutational addition or selective removal of genetic variation they experience.

3.6 Tables and Figures

Table 3.1: Hypotheses on heritability (h^2) and coefficients of additive genetic (CV_A) and residual variation (CV_R) of traits based on their association with fitness (r_{fit}), and hypothesized relationships between heritabilities and coeffcients of variation. Positive and negative relationships are indicated where strong predictions are made.

hypothesis	h^2-r_{fit}	CV_A-r_{fit}	CV_R-r_{fit}	h^2-CV_A	h^2-CV_R
erosion of additive genetic variance	–	–	?	+	?
incorporation of residual variance	?[1]	?	+	?	–
mutational target size[2]	?	+	?	?	?

[1] A negative relationship would be expected in this case, but it is not an essential prediction. If CV_A happens to increase with r_{fit} along with CV_A the drop in heritabilities would not be observed.

[2] Further refinements and predictions of this hypothesis require more detailed information on mutational and epistatic variance.

Table 3.2: Variance components, heritabilities, and CVs for life history and morphological traits in the Cayo Santiago females. Morphological measurements are defined in Table 2.2. Age of first reproduction (age of first rep.) and lifespan are given in cohort years. The mean number of years between births for a female is "mean IBI." Total fecundity, a fitness indicator, is given by "# offspring." The phenotypic Pearson's correlation between the trait and fitness is given in the column r_{fit}. Fixed effects used for the individual traits are noted in Section 3.2.

	n	$\bar{x}$	σ^2_P	σ^2_A	σ^2_R	$h^2 \pm SE$		CV_A	CV_R	r_{fit}
morphological variables										
baseam	104	32.209	1.6798	0.8677	0.8121	0.51656±0.33039		2.8921	2.7979	0.61033
bizyg_w	102	82.921	11.4253	5.9982	5.4271	0.52499±0.35906		2.9536	2.8094	0.66901
cranial_l	105	117.249	17.8170	3.9615	13.8554	0.22235±0.31445		1.6975	3.1747	0.61802
ctom2	98	31.553	1.0433	0.7780	0.2653	0.74567±0.26020	**	2.7954	1.6326	0.05460
eamiof	105	53.932	4.8024	1.4015	3.4009	0.29183±0.27617		2.1951	3.4194	0.45646
femur	104	166.601	41.8044	21.6718	20.1327	0.51841±0.31547	*	2.7943	2.6932	0.25227
glenm1	105	58.076	7.8989	0.1356	7.7633	0.01717±0.32070		0.6341	4.7976	0.60200
h_ap_dim	103	10.812	0.4808	0.3461	0.1347	0.71976±0.28267	**	5.4410	3.3951	0.54377
humerus	103	142.061	30.6069	9.2577	21.3492	0.30247±0.37483		2.1418	3.2525	0.21159
innom_l	102	146.174	34.7148	23.9331	10.7816	0.68942±0.38988	*	3.3468	2.2463	0.42403
mcarp3	102	35.995	2.9341	1.9999	0.9342	0.68162±0.40616	*	3.9288	2.6852	0.01915
mtars3	101	48.964	4.7093	3.4420	1.2673	0.73090±0.35816	*	3.7890	2.2991	0.03830
orbht	105	29.434	2.8057	1.4836	1.3221	0.52879±0.25473	*	4.1382	3.9064	0.43222
radius	103	139.440	30.7092	14.8039	15.9054	0.48207±0.27565	*	2.7593	2.8601	0.17429
tibia	103	155.030	29.1571	15.8957	13.2614	0.54517±0.27418	*	2.5717	2.3490	0.29762
life history variables										
age of first rep.	883	4.224	0.2882	0.0340	0.2542	0.11792±0.06292	*	4.3640	11.9359	-0.04410
mean IBI	148	1.238	0.0605	0.0044	0.0562	0.07219±0.18760		5.3381	19.1375	-0.14411
lifespan	208	10.798	26.8717	5.6507	21.2211	0.21028±0.17506		22.0142	42.6615	0.95032
lifespan[1]	377	7.040	29.8902	14.1770	15.7132	0.47430±0.11293	***	53.4851	56.3083	0.95243
# offspring	208	5.861	16.2294	1.4325	14.7969	0.08826±0.17538		20.4222	65.6365	1.00000
# offspring[1]	377	3.233	15.7151	5.2788	10.4364	0.33590±0.11704	**	71.0567	99.9108	1.00000

* $p < .05$, ** $p < .01$, *** $p < .001$ for the heritability
[1] This is for all available females, *not* just those that reproduced.

Table 3.3: Spearman correlations (r_s) between quantitative genetic statistics and the trait's correlation with fitness (see the values in Table 3.2). These correlations correspond to the scatterplots in Figures 3.1 and 3.2.

	all n=21		morphological n=15		life history n=6	
	r_s	p	r_s	p	r_s	p
h^2	-0.36116	0.1077	-0.48571	0.0664	0.34786	0.4993
CV_A	0.28126	0.2168	-0.20357	0.4668	0.75370	0.0835
CV_R	0.57746	0.0061	0.51429	0.0498	0.98561	0.0003

Table 3.4: Spearman correlations (r_s) among quantitative genetic statistics for complete set of traits (n=21), with p-values immediately below the correlation.

	CV_R	h^2
CV_A	0.52468	-0.00130
	0.0146	0.9955
CV_R		-0.75714
		<.0001

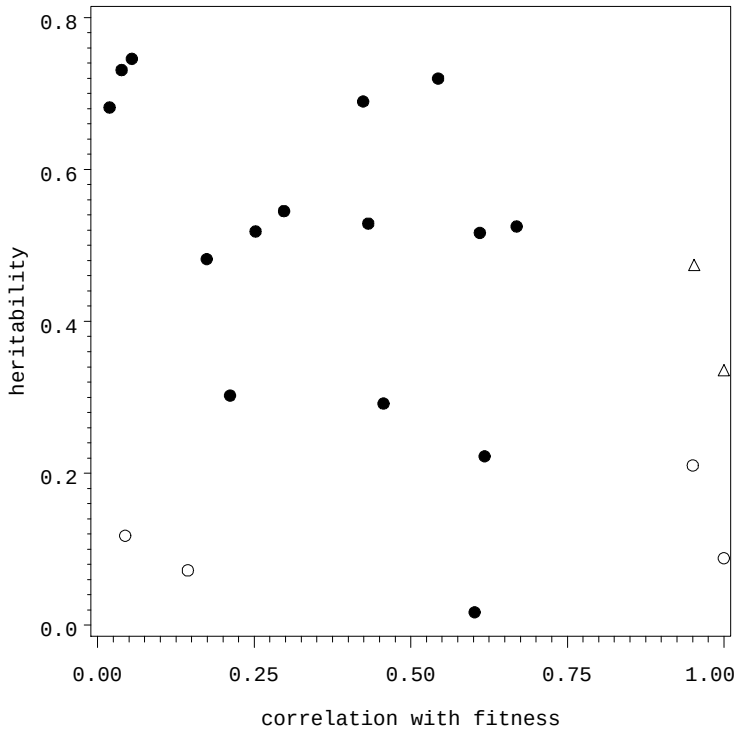


Figure 3.1: The relationship of heritabilities and fitness. Morphological traits are the filled dots. Life history traits are the open symbols. Uncensored lifespan and # of offspring are shown with open triangles. Censored values for these variables are the open circles.

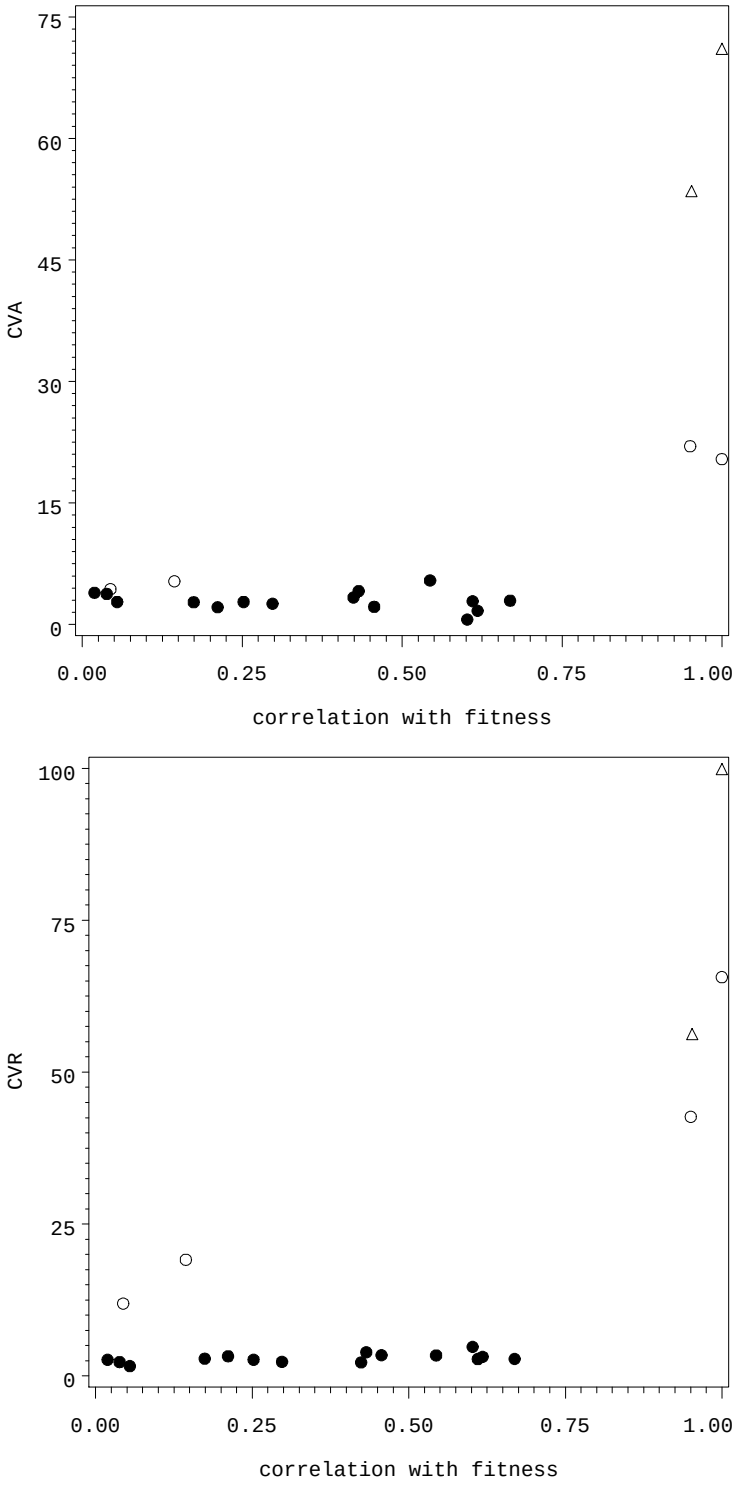


Figure 3.2: The relationship of additive (CV_A) and residual (CV_R) coefficients of variation with fitness. Morphological traits are the filled dots. Life history traits are the open symbols. Uncensored lifespan and # of offspring are shown with open triangles. Censored values for these variables are the open circles.

Chapter 4

Rank as an Environmental Impact on Life History

4.1 Introduction

Hierarchies of social rank are important features of many primate groups (Ellis, 1995; Harcourt, 1987; Fedigan, 1983). Evolutionary perspectives on social rank focus on the relative costs and benefits of high rank to individuals or kin networks with the basic prediction that high rank confers fitness benefits, and therefore high rank is worth competing for (Silk, 1987; Sterck et al., 1997). Testing this prediction has fueled many investigations of primate behavior, demography, and morphology. Furthermore, a great deal of primatological research has focused on explaining why social groups of some species have strong dominance hierarchies and others do not (e.g. Kappeler and van Schaik, 2002). Macaques have figured prominently in both of these discussions.

Among macaque species there is considerable variation in the strength of dominance hierarchies, kin bias, and patterns of affiliation and aggression. Thierry (2000; 2004) classified macaques into 4 "grades" according to their dominance style. Grade 1, described by others as "despotic" and "nepotistic" (*M. mulatta, M. fuscata, M. cyclopis*), is characterized by unidirectional conflicts in which targets of aggression flee and may be severely injured. Reconciliation after conflicts is rare, particularly across kin boundaries. Kinship networks and dominance hierarchies are a strong determinant of who interacts and in what ways. Grade 4, elsewhere referred to as "tolerant" or "egalitarian," is composed mostly of Sulawesi macaque species (e.g. *M. tonkeana, M. maura*), have low rates of aggression, and frequently reconcile after conflicts including stereotypic reconciliation gestures even across kin boundaries. Kinship and dominance hierarchies are far less important in structuring social relationships in these species. Grades 2 and 3 fall in intermediate locations.

In this chapter I examine some genetic aspects of how dominance rank may affect evolutionary processes in macaque populations and test the general prediction that high rank confers fitness benefits. I provide a short review of the socioecological model for female primate sociality and offer some contrasts with recently developed social selection models in the behavioral genetics literature. I find that rank exerts pervasive effects throughout the life history of female rhesus macaques on Cayo Santiago. The benefits of high rank accrue through

both increased fertility and especially increased survivorship.

4.1.1 Rank and Variance in Female Fitness

Social rank is important for its effect on microevolutionary processes in primate populations. If hierarchies of social rank impact fitness as predicted, they will inflate the total variance in lifetime reproductive success. This is because they will raise the fitness of high-ranking and depress the fitness of low-ranking females. Variance in fitness is critically important to microevolutionary processes affecting the composition of populations. Variance in fitness is typically measured as the "opportunity for selection" (see discussion in Section 2.4.2) and thought of as the upper boundary for selection to affect the distribution of any trait that is associated with fitness (Crow, 1958, 1962). Furthermore if this increased variance in fitness is unrelated to phenotypic traits being investigated it is simply "opportunity for drift." Thus rank can affect rates of adaptive and neutral evolution (Lynch and Hill, 1986; Lynch, 1990).

Effect on Selection

If the additional variance in fitness due to rank is unassociated with the breeding value of a given trait in the population, then strong hierarchies of social rank can either speed up or slow down the rate of adaptive evolution of quantitative characters. This can be seen from a facile exploration of the breeder's equation (Equation 4.1 and discussion in Section 2.4).

$$\Delta\bar{z} = h^2 S \tag{4.1}$$

Expanding the terms into their constituent variances or covariances, for the case of no variance caused by social rank, this becomes Equation 4.2, with σ_A^2 the additive genetic variance of a trait being studied, σ_E^2 the residual environmental variance in the trait, σ_{WA} the additive genetic covariance between fitness and trait, and σ_{WE} the covariance of residual environmental effects and fitness.

$$\Delta\bar{z}_{norank} = \left(\frac{\sigma_A^2}{\sigma_A^2 + \sigma_E^2}\right)\left(\frac{\sigma_{WA} + \sigma_{WE}}{\sigma_A^2 + \sigma_E^2}\right) \tag{4.2}$$

In the case of social rank causing increased variance in both fitness and a target trait of interest this is augmented to Equation 4.3, with the additional terms σ_R^2 as the variance in the trait caused by social rank, and σ_{WR} the covariance between social rank and fitness.

$$\Delta\bar{z}_{withrank} = \left(\frac{\sigma_A^2}{\sigma_A^2 + \sigma_E^2 + \sigma_R^2}\right)\left(\frac{\sigma_{WA} + \sigma_{WE} + \sigma_{WR}}{\sigma_A^2 + \sigma_E^2 + \sigma_R^2}\right) \tag{4.3}$$

All variances in Equations 4.2 and 4.3 must be positive. Covariances however may be of positive or negative sign. If the trait of interest is a fitness compo-

nent, such as survival or fertility, then S should ultimately be positive. The covariances contained within the numerator of S may vary in sign. For fitness components under circumstances of no effect of social rank, the sum of σ_{WA} and σ_{WE} must be positive ($S > 0$). Introducing σ_{WR} and σ_R^2 in Equation 4.3 allows for rank to either act as a break on adaptive evolution ($\Delta\bar{z}_{norank} > \Delta\bar{z}_{withrank}$) or accelerate it ($\Delta\bar{z}_{norank} < \Delta\bar{z}_{withrank}$).[1] This depends in part on the sign of σ_{WR}, but more specifically on the inequality in Equation 4.4.

$$\left(\sigma_A^2 + \sigma_R^2 + \sigma_E^2\right)^2 (\sigma_{WA} + \sigma_{WE}) > \left(\sigma_A^2 + \sigma_E^2\right)^2 (\sigma_{WA} + \sigma_{WR} + \sigma_{WE}) \quad (4.4)$$

If the inequality is satisfied then the rate of adaptive evolution will be *slowed* by social rank ($\Delta\bar{z}_{norank} > \Delta\bar{z}_{withrank}$). If we continue to assume that the sum of σ_{WA} and σ_{WE} is positive then there are two main ways the inequality can be satisfied. As already noted, if σ_{WR} is negative, the right side of the inequality will be reduced. However, this corresponds to a case where high rank yields low fitness, which is the opposite of pattern predicted for female macaques (Stucki et al., 1991; Silk, 1987). The more likely scenario is with σ_{WR} positive. In this case, rank can still reduce the rate of evolution if the phenotypic variance in the trait of interest caused by rank (σ_R^2) is large. This is because σ_R^2 appears in the denominator of both the quantities that yield h^2 and S. Conversely, if σ_R^2 is small and σ_{WR} is positive then rank will act to *increase* the evolutionary response in the trait under investigation. It should be clear that the left side of the inequality is simply the squared phenotypic variance including rank multiplied by the phenotypic covariance between trait and fitness without rank. The right side mirrors this; it is the squared phenotypic variance without rank multiplied by the phenotypic covariance between fitness and trait including rank. These conditions for when rank will accelerate or slow the evolution of traits under directional selection are summarized graphically in Figure 4.1.

A standard statistical analysis looking for relationships between traits and fitness can only identify S and not its sub-components—the variances and covariances related in Equation 4.4 that will specify whether rank will slow or accelerate the directional response of a given trait. Nevertheless, measuring S is a first approximation of how rank, fitness, and attributes of female life history are related and should evolve under selection. Regression analyses reported in this chapter are analogous to identifying S.

One assumption of the preceding discussion is that the variance due to rank, residual, and additive genetic effects are all uncorrelated ($\sigma_{RA} = \sigma_{RE} = \sigma_{AE} = 0$). If there are correlations between any of these the selection dynamics will be altered (Roff, 1997). Furthermore, any attempt to compare categories of differently ranked females will confound these factors. For example, if high rank is associated with earlier maturation but high ranking matrilines also carry

[1] In this discussion an increase in rank is movement to the right on a number line (e.g. 1 to 2). This convention is abandoned in the empirical exploration of rank and fitness components below.

genes for this, then the phenotypic differences in rank categories will mix these effects. Assigning agency to either rank or genes would be inaccurate. This generally unrecognized assumption has complicated previous investigations of rank-related life history differences (Silk, 1984, p. 557), but it can be tested by calculating breeding values for particular traits using quantitative genetic methods and comparing them among ranked population segments (Postma, 2006). PBVs are calculated for female life history variables in this chapter as a test of both of these assumptions.

Effect on Drift

Even if traits are unassociated with fitness their evolution will still be affected by the inflated variance in fitness due to social rank. This will be more important in small populations where sampling errors from random variation in reproductive success, will lead to unpredictable changes in allele frequencies and trait distributions. For this reason variance in fitness is alternatively construed as "opportunity for drift." This point is of particular interest for attempting to estimate effective population size (N_e) of animal populations. If reproduction is concentrated in a few highly successful individuals the census population size (N) will be much larger than the effective population size (Nunney, 1993). Census population sizes will misrepresent population risk of extinction and inbreeding—points of considerable concern for effective conservation programs and management of captive animal colonies (Strier, 2007; Lynch, 1996; Ralls and Ballou, 1982; Ralls et al., 1988).

In sum, inference about the evolutionary history of primate clades, whether adaptive or neutral, is strongly influenced by how concentrated reproduction is in small sets of individuals. Predicted patterns for rhesus macaque females, and other primate species in which rank is a strong predictor of lifetime fitness implies a mismatch between effective and census population sizes and altered evolutionary dynamics whether or not trait breeding values and social rank are correlated.

4.1.2 Why are some macaques "nicer" than others?

In addition to these population genetic properties, identifying patterns of rank-related fitness variation helps to clarify the fitness costs and benefits of social rank. Why some macaque species are nepotistic and nasty and others are tolerant or egalitarian has not been adequately explained (Menard, 2004; Chapais, 2004). Some of this deficiency stems from limited appreciation of the diversity of evolutionary mechanisms that can affect social behavior. These include byproduct mutualism, reciprocal altruism, kin selection, and group selection (Silk, 1987; Dugatkin, 2002).

The current socioecological models focus primarily on byproduct mutualism (Wrangham, 1980; van Schaik, 1989; Sterck et al., 1997; Kappeler and van

Schaik, 2002). Social behaviors are promoted in byproduct mutualism simply through the selfish interests of the actors. Typical examples involve cooperation in the face of "harsh environments." Broadly interpreted, this may involve huddling in cold temperatures, cooperative hunting of large prey items, or giving out vocal signals for discovery of large food items that attract group-mates. The costs of cooperating are so low that the benefits provided outweigh them (Dugatkin, 2002). Individuals simply act to raise their individual fitness. In socioecological models of primate sociality, female primates are thought to respond to predation risk by forming social groups (van Schaik, 1989; Janson, 2003). Group living provides benefits in predator detection, dilution of risk of capture, and perhaps communal defense. Females need not be related to gain these benefits from group living. Feeding competition is thought to set upper boundaries on group size in these models. Above a certain threshold, group members can no longer gather adequate food and social relationships breakdown. Differences in foraging ecology may then result in variation in dominance hierarchies between populations or species. Primates that forage on clumped resources that can be defended by single individuals or coalitions (regardless of their relationship) are expected to form dominance hierarchies. This is often described as within group contest competition (van Schaik, 1989). Another benefit of group living that socioecological models posit is group defense of resources such as preferred territories containing water, shelter, and feeding sites. It is the relative strength of within and between group contest competition that is suggested to predict the strength of dominance hierarchies. When between group competition is high, fairly relaxed hierarchies are expected; when it is low relative to within group competition, much stronger hierarchies are expected. With low levels of between group competition, high-ranking females can risk losing group members and not suffer from reduced group competitive ability. When between group competition is stronger they pay more severely in reduced fitness from diminished between group competitive ability (Wrangham, 1980).

This model is currently popular despite difficulty in testing its core predictions such as levels of predation and relative strength of within and between group contest competition (Kappeler and van Schaik, 2002; Sussman et al., 2005). Generally, we would predict that nepotistic/despotic/Grade 1 macaques (*M. mulatta, M. fuscata, M. cyclopis*) should feed on more clumped resources that can be readily defended, have relaxed relationships with neighboring groups, and be at some risk of predation to encourage group formation. Macaques with egalitarian/Grade 4 social relationships should feed on foods that are less easily monopolized and have more "xenophobic" responses to other groups, but still face predation pressure (Sterck et al., 1997). Defining and measuring these variables is complex and relies on a variety of assumptions about the relationship between food categories and their spatial distribution.

Whatever the merits of socioecological models, attempts to relate macaque social styles to feeding ecology have generally failed. Menard (2004) surveyed

published geographic distributions, diets, group composition, and between-group interactions of macaque species and found little relationship with predicted patterns of despotism/nepotism. For example, rhesus macaques are typical despotic/nepotistic macaques, but they do not feed on clumped monopolizable resources. In wild settings, the majority of their diet is low quality, widely distributed leaves and grasses. Other macaques that feed primarily on more patchy, high quality foods like fruits do not have rigid nepotistic dominance like rhesus macaques. Furthermore, patterns of between-group interactions did not fit expectations. Egalitarian/tolerant/Grade 4 moor (*M. maura*) and Barbary (*M. sylvanus*) macaques have very low levels of between group competition.

Thierry (2000; 2004) has noted there is a phylogenetic pattern to the distribution of social styles among macaques, with the most despotic/nepotistic concentrated within a single recent radiation of macaques (*M. mulatta, M. fuscata, M. cyclopis*). Phylogeny may explain some of the taxonomic patterning of macaque social styles, but it is a non-explanation for mechanisms of how dominance styles emerge and what the ultimate causal factors are. Furthermore, Thierry assigned many macaques to his "grades" by the social style of their close relatives, not indicative behavioral data. However, Thierry also emphasizes contingency in the fit of macaque species to their current ranges and habitats. Historical contraction and expansion of macaque habitats in the Plio-Pleistocene likely induced speciation among fragmented macaque populations (Abegg and Thierry, 2002) and possibly encouraged change in dominance styles. History may explain much of the mis-match between macaque dominance style and current ecology.

Recent theoretical work has summarized the many proposed evolutionary mechanisms for social behavior under the umbrella term "social selection" (Moore et al., 1997; Wolf et al., 1998, 1999; Moore et al., 2002). Models of social selection account for the selective influence provided by social partners. This includes the byproduct mutualisms emphasized by the socioecological model. However, it also applies to much more diverse evolutionary models of kin and group selection, reciprocal altruism, indirect genetic effects (e.g. maternal effects), and sexual selection. Their application to macaque sociality and dominance relationships emphasizes a much wider array of processes and complexity of relationships of social behavior with ecology, demography, distribution, phylogeny.

In social selection models for a single trait, individual relative fitness (w) is an additive function of the trait in a focal individual (z_i) and the same trait or another trait expressed in a social partner ($z_j\prime$), weighted by their average contributions to fitness (β).

$$w = \alpha + \beta_N z_i + \beta_S z_j\prime + \varepsilon \tag{4.5}$$

The remaining terms are a constant (α) and residual deviation (ε). The two βs are selection gradients that measure the strength and direction of natural

selection (β_N) and social selection (β_S). Extensions of this model to multiple traits in the focal individual and multiple traits in multiple social partners are given by Wolf et al. (1999).

The total selection differential on the focal trait (s_i) can also be partitioned into components of natural and social selection.

$$s_i = P_{ii}\beta_N + C^{ij\prime}\beta_S \tag{4.6}$$

Here, P_{ii} is the phenotypic variance of the focal trait ($P_{ii}\beta_N$ is the natural selection differential) and $C^{ij\prime}$ is the phenotypic covariance between trait z_i in the focal individual and $z_{j\prime}$ in the social partner ($C^{ij\prime}\beta_S$ is the social selection differential). $C^{ij\prime}$ relates the agent of social selection (z_j) to its target (z_i). This is a key feature of social selection models—traits are both agents and targets of selection. Note that if $C^{ij\prime} = 0$ there is no social selection and a standard selection differential results (Lande and Arnold, 1983). However, the myriad behavioral, ecological, and demographic factors that cause $C^{ij\prime} \neq 0$ explain how models of social selection incorporate so many diverse evolutionary processes, and offer a broad framework for speculating on the evolution of macaque social relationships.

Wolf et al. (1999) identify four factors that can influence $C^{ij\prime}$. First, *non-random interactions* among individuals can increase or decrease $C^{ij\prime}$. If individuals preferentially associate with other that are similar to them then $C^{ij\prime}$ will increase. If they avoid those that are like themselves it will decrease $C^{ij\prime}$. This includes the byproduct mutualisms emphasized by the socioecological model—female primates seek out others with similar requirements for food, predator avoidance and mates. This also likely the benefit of polyspecific associations reported for small-bodied platyrrhines and guenons (Buchanan-Smith, 1990; Garber and Bicca-Marques, 2002; Enstam and Isbell, 2007). Non-random interactions can also include reciprocal altruism if there are phenotypic signals of altruistic behavior.

Second, *behavioral modification* can influence $C^{ij\prime}$. If individuals change their behaviors based on the phenotypes of other individuals they interact with $C^{ij\prime}$ will be changed. When individuals of like phenotypes treat each other more favorably than those of divergent phenotypes $C^{ij\prime}$ will be positive. In the special case of reciprocal altruism the tit-for-tat strategy would create a large positive $C^{ij\prime}$. In the case of rhesus macaque social dominance, females adopt different behaviors depending on the status of individuals they interact with. Dominant females make alliances with other dominant females but rarely do so with subordinate females. Grooming is typically restricted within rank categories or matrilines. Violent aggression can be common across rank categories but is rare within them. All of these factors will create positive $C^{ij\prime}$ within rank levels or matrilines and negative $C^{ij\prime}$ between matrilines. Behavioral modification also applies to the between group relationships stressed by the socioecological model.

Aggressive encounters between groups depress $C^{ij\prime}$ for individuals in different groups, but raise it for individuals within groups.

Third, *relatedness and inbreeding* alter $C^{ij\prime}$. Kin selection is the most obvious form of this, where individuals who resemble one another phenotypically do so because they share genes. Altruistic behaviors are widely recognized to evolve when they raise the inclusive fitness of donor and recipient. In the social selection framework this means that s_i in Equation 4.6 is positive (altruism evolves) but it occurs through negative natural selection and positive social selection ($\beta_N < 0$ and $\beta_S > 0$). For rhesus macaque social behavior the implications of kin selection have long been recognized. Aiding relatives in agonistic conflicts may reduce your individual fitness, but will elevate that of shared genes (Silk, 1987, 1984).

Less commonly recognized is that inbreeding also raises resemblance among individuals by increasing the probability that they share alleles and genotypes that are identical by descent from genealogical ancestors (Lynch and Walsh, 1998). This emphasizes the historical role population size and structure of macaque social groups and metapopulations may have played in the evolution of social behaviors. When lineages within groups are inbred it will provide a relative increase $C^{ij\prime}$ between members of the same lineage and a reduction in $C^{ij\prime}$ between members of different lineages. When social groups are inbred it will increase $C^{ij\prime}$ among all group members and decrease it between individuals of different groups (Agrawal et al., 2001). Strong female philopatry among macaque groups and group fission along matriline boundaries promotes genetic subdivision among social groups (Ober et al., 1984; Cheverud and Dow, 1985; Harpending and Cowan, 1986; Melnick and Hoelzer, 1996; Tosi et al., 2003).

Finally, *indirect genetic effects* can alter the covariation in phenotypes among interacting individuals. These occur when the genes coding for a phenotype in a social partner affect the phenotype of another individual. The most widely recognized examples of indirect genetics effects are maternal effects in mammals, where the quality of milk and other care provided by the mother affects the growth rate and size of its offspring. Genes carried by the mother modifying her phenotype (e.g. milk quality) will alter the offspring phenotype (e.g. neonate mass or growth rate).

Similar processes may apply to female macaque social behaviors. Moore et al. (2002) provide a theoretical context for behaviors of social dominance, and performed a selection experiment on male cockroaches (*Nauphoeta cinerea*) to alter the expression of a dominant (z_d) and subordinate phenotypes (z_s). Male cockroaches have clear phenotypic differences in the expression of dominant (butting, kicking, lunging, biting) and subordinate behaviors (flee, crouch). With only 7 generations of selection they were able to alter the probabilities of expressing dominance and subordination behaviors between the differently selected lines.

In this scenario the covariance between an unrelated dominant males' phe-

notype and subordinate males' phenotype (equivalent to $C^{ij\prime}$ above[2]) is

$$C^{d,s\prime} = \frac{G_{d,s} + \psi_{d,s}\psi_{s,d}G_{d,s}}{(1 - \psi_{d,s}\psi_{s,d})^2} \tag{4.7}$$

In this equation $G_{d,s}$ is the genetic covariance between dominant and subordinant phenotypes in the population, $\psi_{d,s}$ describes the effect of the subordinant individual's subordinant phenotype on the dominant individual's dominant phenotype, and $\psi_{s,d}$ describes the effect of the dominant individual's phenotype on the expression of the subordinant individual's subordinant phenotype. Both ψ terms are path coefficients ranging from -1 to 1, provided z are standardized variables. For example, if an individual's performance of dominant behaviors induces the expression of subordinate behaviors in a social partner then $\psi_{s,d} > 0$. This would be the expected behavioral description of conflicts between dominants and subordinates. If an individual's performance of subordinate behaviors reduces the expression of dominant behaviors in a social partner then $\psi_{d,s} < 0$. This would be the expected description of "appeasement" by subordinates to avoid an outright conflict. If appeasement behaviors are not effective at reducing dominant behaviors then $\psi_{d,s} = 0$ and if they instead encourage dominant behaviors $\psi_{d,s} > 0$.

However, indirect genetic effects contained in the genetic covariance between these the expression of dominant and subordinant phenotypes ($G_{d,s}$) largely dictates how $C^{d,s\prime}$ contributes to social selection. Whatever the signs of the ψ coefficients, the sign of $C^{d,s\prime}$ will be the same as $G_{d,s}$. Initially this may seem to be a constraint on the evolution of dominance hierarchies—if the genes carried by individuals that promote aggressive, dominant behaviors also inhibit subordinate behaviors, either by pleiotropy or linkage disequilibrium, then $G_{d,s}$ and $C^{d,s\prime}$ would be negative. However, such direct relationships between genetic structures and behaviors are unlikely in primates. Instead, one can envision positive genetic covariances between dominant and subordinate behaviors, due again to either pleiotropy or disequilibrium, resulting from shared genetic perturbations in the level of psychological and metabolic sensitivity to social environments. Thus if individuals are genetically and physiologically "primed" to strongly express the behavioral phenotype of whatever rank category they find themselves in then $G_{d,s}$ and $C^{d,s\prime}$ will be positive.

Recent laboratory work on captive macaques has demonstrated presumably genetic differences among species for social behaviors. Despotic/nepotistic/Grade 1 rhesus macaques are polymorphic for genes in the serotonergic and dopaminergic pathways, while more tolerant macaques are monomorphic (Wendland et al., 2005). The functional importance of these differences are currently unclear, but they may signal differences among macaque species with different social styles that may operate through these neuroendocrine path-

[2]The only change is that two traits are necessarily being tracked in the two social partners whereas before they could be the same trait (Moore et al., 1997).

ways. Serotonin and dopamine are neurotransmitters widely implicated in impulsivity, depression, and aggression in studies of laboratory animals (Lesch and Meschdorf, 2000) and are known to affect the hormonal reproductive axis through the stimulation of gonadotropin-releasing hormone (Cameron, 2003). Psychologically induced stress from the threat of violence or lack of social partners may be an important mechanism in causing rank-related fitness differences in female rhesus macaques (Section 4.1.3).

This overview of social selection models emphasizes there are many evolutionary processes that could have played a role in the molding macaque sociality. Whatever its merits or flaws as currently discussed in the primatological literature, the socioecological model is particularly narrow in what factors it identifies as important in the evolution of dominance styles. If history is primarily to blame for why some macaques are "nicer" than others (Thierry, 2000; Abegg and Thierry, 2002), then a theoretical framework that makes predictions about events or factors in historical macaque evolution is required. History is a complication for the socioecological model—a source of "noise" in what should be a clear signal of fit between social style and feeding ecology. Did the common ancestor of rhesus, Japanese, and Formosan macaques forage on clumped high, quality resources? We can never know because paleoenvironmental reconstructions are never this precise.

Social selection models, however, incorporate a variety of evolutionary mechanisms, some of which may be recorded in the the genetic structure of organisms. Patterns of speciation, range expansion, introgression, bottlenecks, and subdivision are becoming much better understood in macaques (e.g. Tosi et al., 2003; Evans et al., 2003). The evolutionary effects of relatedness and particularly population structure on social evolution have not been fully appreciated or explored in studies of primate sociality (Agrawal et al., 2001). Furthermore, social selection models make predictions about the genetic architecture of traits related to dominance and subordination behaviors. Though difficult to study these predictions can be addressed minimally in the form of interspecific comparisons and eventually within populations of known genotyped individuals matched to behavioral records.

4.1.3 Mechanisms and Evidence for Rank-Related Differences in Fitness

Two mechanisms by which females achieve higher reproductive fitness have been offered. Both identify energy budgets as a the primary means by which rank can influence fitness. First, high ranking females may have greater access to scarce resources such as food, water and shelter. By harvesting more resources from the environment they are able to produce more offspring and rear them successfully to independence. They may also spend less time and energy in feeding to satiation (Bercovitch and Strum, 1993). While logically clear, no

concrete tests of this proposition have been made. Detailed studies of caloric intake of wild or free-ranging primates are exceptionally difficult. Most studies of foraging behavior record foods consumed and proportion of waking day spent feeding (Altmann, 1998). Many observers have suggested that total resource scarcity should mimic the effect of low rank. For example, Strum and Western (1982) noted that female olive baboons (*Papio anubis*) with a low index of food availability matured later and had lowered fertility. Some evidence for the role of rank-mediated feeding competition in this population comes from the greater response of low ranking females to periods of scarcity (Bercovitch and Strum, 1993). Low-ranking females always matured later than the high-ranking members of their cohort. While suggestive, the mechanism that causes these rank-related differences is unknown.

Second, high rank may reduce the amount of aggression directed toward a female and/or the stress caused by such aggression. The short term consequences of hormonal stress responses are well documented (Cameron, 1997; Abbott et al., 2003; Sapolsky, 2005). Stressors cause a change in allocation of resources away from reproductive activity and can impair immune function if at chronic levels for extended periods of time. Aggressive acts themselves can also negatively impact females fitness. Bites and scratches, require the mobilization of immunological response and repair to ward of infection. If low ranking individuals suffer more injury during conflicts, then they could also function to produce rank related fitness differences. Direct tests of this idea are lacking, but evidence is suggestive for psychosocial stress mechanisms that suppress reproduction. For example, women with functional hypothalamic amenorrhea do not suffer undernutrition, or any other physiological cause that would suppress menstruation, but are infertile. Women with this condition do not have high numbers of stressful life events, but they respond much more strongly to them, including elevated hypothalamic-pituitary-adrenal axis activity (Cameron, 2003). Furthermore, populations of captive macaques which all individuals were known to have similar nutrition still exhibit rank-related differences in life history (Deutsch and Lee, 1991).

These two mechanisms, priority-of-access and stress, can function simultaneously. Altered hormone profiles are one physiological means of suppressing reproduction in periods of resource scarcity (Cameron, 2003). Bercovitch (1991) explains the mixture of studies on baboons and macaques finding and not finding rank-related differences in fitness as a function of scarcity and patchiness of resources, population density, stability of social relationships, and reduced predator pressure. The greater any of these the more likely it should be one finds fitness differences among rank levels (Harcourt, 1987).

Whatever the cause, high rank is expected to confer some net benefit in fitness for female primates. There is a growing body of evidence on this topic summarized in Table 4.1. Despite the diversity of catarrhine primates, research has focused almost exclusively on macaques and baboons, which have similar social

structures and female life histories. Several general patterns emerge from this table. First, researchers have used a variety of different fitness indicators and life history variables. Thus comparisons among studies are difficult. However, the only sources able to test for rank-related differences in fitness surrogates that found *no* association come from a free-ranging group of Japanese macaques in which dominance relationships are reported to be quite flexible (Fedigan, 1991; Gouzoules et al., 1982) and a wild chacma baboon group in which predation is the most important source of mortality (Cheney et al., 2006). Some caution must be exercised in interpreting this table, as with any literature review, given reporting biases among authors and publishers. Despite some conflicting results, several generalizations can be made based on these variables. Higher-ranking females frequently give birth to their first offspring at younger ages. However, interbirth intervals are *not* consistently shorter in higher-ranking females, particularly among the macaques. Where information is available, it appears adult body size is not related to rank, but high-ranking females consistently have higher offspring growth rates. This may help to explain the earlier maturation of high-ranking females who reach a threshold mass prior to their age-mates. Finally, adult survival rates (lifespan) do not appear to be closely tied to rank, though there is a tendency for high ranked mothers to have improved offspring survival rates.

Closer examination of some of these studies demonstrate some of the difficulties and unexpected patterns in research on associations between female primate rank and fitness. One problem often encountered, particularly in wild settings, is low power and lack of statistically significant differences among rank groups for different life history variables or fitness proxies. Van Noordwijk and van Schaik (1999) report on a 12 year study of long-tailed macaques (*M. fascicularis*) in Sumatra. Their efforts yielded complete reproductive careers on 65 adult females. For each of three variables (birth rate, offspring survival rate, age of first reproduction, and adult female survival rate) differences among the ranked groups of females were in the predicted direction but almost never statistically significant.

Curiously, they argued that these minor differences that could be due to chance are important and interpretable indicators of female rank–fitness associations in this macaque population. They even provide demographic projections for the different rank categories based on their average values for the life history variables and use them to argue "small (and not always statistically significant) differences between females in their [life history variables] become biologically quite significant." This echoes the earlier statement of Silk (1987) that "[e]ven if the magnitude of the differences in fitness among high- and low-ranking individuals is very small (and even if it does not reach statistical significance), the adaptive consequences of dominance rank may be important in shaping behavior." Demographic projections for rank categories based on average values are horridly misleading because they do not account for the variance around

these averages. While the differently ranked population segments may diverge in relative size because they are projected to grow at different rates, the variance around the predicted size of a ranked population segment *will grow at a much greater rate* than the difference between the ranked segments does.

Much research can be done outside of an explicit hypothesis testing framework (Peters, 1991). However, testing for rank-related fitness differences is not one of these cases. Significance tests indicate when differences can be attributed to a causal or associated factor (social rank) or, more parsimoniously, could arise by chance alone. Ignoring the results of significance tests insulates this research from an empirical grounding—both significant or non-significant results are interpreted in precisely the same way. Demonstrating rank-related fitness differences should be done with statistical tests. Assuming the variance in life history variables or fitness components are roughly constant, the only way this can be done in observational studies is to increase sample sizes (Ott and Longnecker, 2001). Currently, this is a powerful argument for the utility of studying primates in free-ranging conditions such as Cayo Santiago.

Unexpected fitness *costs* have also been documented for high-ranking female Old World Monkeys. Packer et al. (1995) noted that high-ranking female olive baboons have a higher proportion of miscarriages. This cost was accompanied by several benefits to high rank including shorter post-partum amenorrhea, earlier age at first birth, and higher infant survival rate. There are few studies of cercopithecoids documenting costs to high rank. Pathological infertility of high-ranking females in this baboon population was suggested to serve as a constraint on female aggression through hormonal mechanisms. Elevated androgens may have promoted aggressive behaviors and acquisition of high rank but damaged reproductive ability. While there are methodological concerns about the results of this study (Altmann et al., 1995; Packer, 1995), hormonally mediated trade-off between aggressive rank acquisition and impaired fertility remains a provocative hypothesis for why primate groups vary in their dominance styles. Identifying costs to high rank is a relatively under-explored area of research on primate sociality but would fit with patterns seen in other mammals (Creel, 2001).

4.1.4 Previous Research on Cayo Santiago Females

Cayo Santiago females have been the subject of several analyses of social rank–fitness associations. Drickamer (1974) demonstrated that high and middle ranking females living at La Parguera, another set of Puerto Rican islets[3], gave birth to their first offspring at earlier ages than low ranking females. These monkeys were a mixture of transplants from Cayo Santiago and direct imports from India. He suggested that the increased attention higher ranking females receive from adult males might induce this difference. He reported several other rank-

[3]The La Parguera colony was moved to Morgan Island in coastal South Carolina beginning in 1979 (Taub and Mehlman, 1989).

related life history differences but their interpretation is complicated by the age-structure of the colony (Fedigan, 1983).

Sade et al. (1976) calculated population growth rates (λ) of different social groups and dominance categories on Cayo Santiago for 1973–74. They noted that high, middle, and low ranking segments of the population were growing at different rates ($\lambda_{high} = 1.098$, $\lambda_{middle} = 1.021$, $\lambda_{low} = 0.962$), but did not test for statistically significant differences in these rates. Citing Drickamer (1974), they suggested that differences in age of maturity contributed to this pattern, but admitted they had no clear answer. They emphasized that the abundance of food on the island should eliminate priority of access as an explanation for rank-related fitness differences.

Stucki et al. (1991) extended this analysis in an attempt to identify differences in growth rate among high, middle, and low ranked matrilines of 5 different social groups (F, M, J, I, and L) for the years 1973–74 and 1975–76. They used two different kinds of bootstrap tests to calculate confidence intervals on the population growth rates of the ranked population segments. Both of these indicated that there was great uncertainty in the estimated growth rates of the population segments, but they reported significant differences between high and middle ranked population segments, and high and low ranking segments when excluding one social group. While this study was pioneering in its application of resampling methods to primate demographic data and approaching rank-related fitness questions, this form of hypothesis testing through overlapping confidence intervals is no longer favored (see Section 4.2.3), nor would the number of samples generated (200) be deemed acceptable given the computing power now available (Manly, 1997).

Bercovitch and Berard (1993) analyzed a number of life history variables of Cayo Santiago females comparing females ranked in high, middle, and low matrilines. This was partly in response to the analysis of Sade (1990) which did not directly address rank-related differences. They assigned the highest ranking matriline from each social group to "high," the lowest to "low," and all remaining ones to "middle." They were conservative in their analysis and only used females who died on the island and had all of their offspring avoid removal prior to reaching maturity. They demonstrated that variation in population density contributed to the observed changes in ages of first birth in the colony. At high density early maturity (3 years) was rare in lower ranked females, while at low density it was common. This increase in frequency of early maturity was also seen in the high ranked females at low density. They also report that rank had no relationship with infant survival or adult survival. In comparing the number of offspring high rank, early maturing females had with low rank, late maturers (5 years) they found no significant difference, though the samples were extremely small (7 and 2, respectively) and the means for these categories were 4.4 and 1.5. The power on these tests is obviously very low and limits any hope of detecting statistically significant differences. In contrast to Sade et al. (1976),

they invoked differential access to food as a possible explanation for rank-related life history differences. This study was important in demonstrating that lifespan explained much of the variation in lifetime reproductive success among Cayo Santiago females, shifting concern away from age of maturity. Indeed, this is a common pattern for long-lived mammals which are limited in their reproductive output to one or only a few offspring per reproductive event (Clutton-Brock, 1988). Any effect that rank has on adult survival rates will be crucial to fitness.

Some of the disagreement between these research groups (Sade et al., 1976; Sade, 1990; Stucki et al., 1991; Bercovitch and Berard, 1993) can be attributed to using different fitness surrogates. The individual λs used by Sade et al. will always weight early reproduction more heavily and increase the importance of age of first birth. Furthermore, they use different sets of life history data, and define rank categories is distinct ways. Nevertheless, the analyses presented here offer some resolution to this disagreement by employing new techniques to analyze variation across the lifespan in the largest number of individuals possible. Other disagreement on the mechanisms causing rank-related differences are matters of perspective that can only be resolved with further research on the dietary intake and stress environment of females.

4.1.5 Analyzing the Cayo Santiago Demographic Records: New Tools

A reanalysis of Cayo Santiago female life history data is best justified when something new is to be offered. In this chapter I use two novel approaches to the question of rank-related fitness and life history differences.

The first of these is a recently developed method for analyzing demographic data, that incorporates demographic matrix modeling (Caswell, 2001) to directly relate traits such as rank, or any other independent variable, through a set of fitness components to the population growth rate—λ. This is called elasticity path analysis, or the *hierarchical decomposition of selection* (van Tienderen, 2000). To date, I know of only one other empirical application of this method, despite its demonstrable utility (Coulson et al., 2003). The great benefit of this method is that it offers a cohesive framework in which to analyze population demography, fitness components, and variables like rank that affect them. With sufficient information on morphological or behavioral traits it can be used to calculate the direction and strength of selection on these traits, as well, though this is not done here because of limited samples of morphological data and no behavioral data (Section 2.3). Additionally, this method allows the maximum utilization of information from the female life histories. In particular, the removal of animals from the colony for management purposes does not complicate the analysis. Information on removed females, and females who had offspring removed, is maintained and sample sizes remain much larger than they would otherwise be (Section 3.4.4).

The second tool used in the analysis of these rank-related life history differences is the calculation of predicted breeding values (PBV). Though initially developed in animal breeding applications, these have been successfully used in a growing number of ecological studies of mammal and bird populations (e.g. Garant et al., 2005; Postma and van Noorwijk, 2005; Kruuk et al., 2002). Predicted breeding values are an estimate of the additive genetic value of an individual for a trait. They are based on the trait heritability, an individual's own phenotypic value for the trait, and those of related individuals (adjusting for any other fixed or random factors needed). They are calculated with the mixed model methodology described in Section 2.5 as solutions for the vector **a**. Postma (2006) provides an extensive discussion of their calculation and use in ecological studies (see also Mrode, 1996). Knowing the PBVs for a fitness component or life history trait allows one to see if these differ on average among rank levels within the population. For example, PBVs will indicate whether high ranking females mature earlier because they have genes that favor this, or if this is simply the result of the beneficial environment provided by high rank. Untangling these potentially confounding factors is an important advance in the study of rank-related life history differences (Section 1.5; Silk, 1984). It also directly tests one of the assumptions of Equation 4.4 on how rank can speed or slow the evolution of traits.

4.1.6 Hypotheses

In this chapter I test the general hypothesis that social rank affects the life histories of female rhesus macaques at Cayo Santiago. More specifically, differences among rank levels are sought in overall measures of lifetime fitness. High ranking females are predicted to have higher lifetime fitness than medium, and low ranked females. Medium ranked females should have higher fitness than low ranked. Furthermore, the fitness differences are expected to be the result of disparities in survival and fertility rates throughout the lifespan. High ranking females should mature earlier, have shorter interbirth intervals, and live longer than medium and low ranked females. Finally, I predict that rank-related differences in life history are environmental and not due to genetic differences in breeding values among rank categories.

4.2 Methods

4.2.1 Matriline Social Ranks

Sources for matriline social ranks were noted in Section 2.3.3. Rank was treated as an ordinal category of high, medium, or low. Groups in recent years typically only have one matriline in each of these categories and assignment is thus simple because only two or three matrilines are found in a group. When there were only two matrilines they were assigned to high and low. In earlier years there

are many matrilines in groups and assignment is more difficult. Groups A, K, and F are the most problematic in this regard. In general, the top third of the matrilines were assigned as high, middle third as medium, and bottom third as low. In intermediate cases, where there were four or five matrilines in a group, assignment to one of the three categories was made such that only a single matriline was high ranked, the following one or two medium ranked, and the remaining matrilines low ranked. All assignments were made blind to the demographic and life history measurements reported below. Females in social groups composed of a single matriline were excluded from the analysis. High, medium, and low rank were coded as 0, 1, and 2, respectively. For the analyses reported below, this means that an "increase" in rank is movement from higher to lower rank.

Coding rank in this way allows for the pooling of records over social groups. This requires the assumption that rank affects life history and fitness in the same way regardless of group membership, but greatly increases the number of observations in the individual rank levels. If there is heterogeneity in the effect of rank among groups it is likely to make estimates when pooling across them more conservative.

Analyses of fitness components, life history variables, and fitness measures below requires the averaging of rank over the time period in which the variables is measured or represents (e.g. the average rank over all years an individual was alive is used for the relationship between rank and lifespan). This averaging can result in non-integer values. Including the birth cohort an individual belongs to in regression models for life history variables and fitness surrogates accommodates temporal differences in the effect of rank on these variables (Section 4.2.5). Analyzing separate blocks of time in the analysis of fitness components and fitness for different ranked segments of the population also accommodates these potential changes (Sections 4.2.3 and 4.2.4).

4.2.2 Life Cycle Model

The hierarchical decomposition of selection developed by van Tienderen (2000) begins with a simple model of the life cycle of the organism being studied (see also Coulson et al., 2003). I used a demographic matrix model based on the division of the female macaque life cycle into three stages (See Figure 4.2 and Tables 4.2 and 4.4). These are: *juvenile* 1–2 years old, *young adult* 3–5 years old, and *mature adult* 6 or more years old. These divisions were used because of interest in the differences among these life stages noted previously (Table 4.1). Survival and fertility probabilities were calculated for these stages as the mathematical product of fitness components (Table 4.2). Because a pre-breeding census model was used, fertility probabilities incorporate the survival of infants to their first census (Caswell, 2001). Midnight on September 1 was used as the time of census as this has always preceded the beginning of the birth season

(Figure 2.4).

Fitness components in the hierarchical decomposition of selection must be scored in two ways. Once is for the *years* being analyzed to create the matrix entries for predicting population growth rate (λ) and computing elasticities, which are defined below (right side of Figure 4.3). The other scoring is done to derive fitness component values for *individuals* while they were in each age class. These values are used as dependent variables in regressions with some other independent predictor, such as matriline social rank (left side of Figure 4.3), or to perform regressions/correlations between the values to examine trade-offs (Chapter 5). In these regressions the fitness components are standardized by dividing by their respective means.

The same set of life histories is used for generating both sets of scores, but information is pooled differently in each scoring to come up with counts of individuals in different states. In the calculation of the matrix entries the grouping is done for age class and year, while for the construction of fitness components the counts are made grouping by age class and individual identity. Once the proper counts are made, fitness components are calculated from the same formulae for both sets of scoring. All of the fitness components are probabilities defined such that an increase in any one of them will result in an increase in fitness (λ). This is a common definition of fitness components (Hughes and Burleson, 2000).

Removal of animals from Cayo Santiago has been common practice (Section 2.2). This can be accommodated in the hierarchical decomposition of selection as a separate "fitness component" which represents the probability of avoiding removal. If there are correlated effects on other fitness components to the removal of individuals, this should be detectable in the correlation between fitness components. For example, if the removal of a mother's infants increases her birth rate this should result in a negative correlation between these components.

Two fitness components are used to define the survival and graduation probabilities (σ_i) in the transition matrix in Figure 4.4 (See also Table 4.2 and Figure 4.2). The annual survival or graduation probability is the product of these two components. The probability of a juvenile or adult avoiding removal (*probnonremoval*) is the difference of 1 and the ratio of the number of females that were removed (*nremmoved*) and the total number that entered the age class (*totalenter*).

$$probnonremoval = 1 - \frac{nremoved}{totalenter} \tag{4.8}$$

The annual probability of a juvenile or adult surviving *given that she was not removed* (*probsurvival*) is the difference of 1 and the ratio of the number of females in the age class that died during the year (*ndie*) and the number that

escaped removal ($totalenter - nremoved$).

$$probsurvival = 1 - \frac{ndie}{totalenter - nremoved} \tag{4.9}$$

Five fitness components are used to define the fertility probabilities (f_i) in the transition matrix in Table 4.4 (See also Table 4.2 and Figure 4.2). The matrix fertility probability is the product of these five components. The annual birth rate ($birthrate$) is the ratio of the number of offspring of any sex born to females in the desired age class in the year ($nborn$) and the number of adult females alive for any portion of the age class ($totalenter$).

$$birthrate = \frac{nborn}{totalenter} \tag{4.10}$$

The probability of knowing the sex of the infant is used to accommodate the production of infants that were never sexed by colony observers, usually due to the young death of the infant. This fitness component ($knowrate$) is largely an infant survival measure, but cannot be interpreted because of variation from year to year in the number of unsexed infants. It is given by the ratio of the number of infants of known sex ($nknowsex$) to the total number born ($nborn$).

$$knowrate = \frac{nknowsex}{nborn} \tag{4.11}$$

The female sex ratio ($fsexratio$) of the infants is the ratio of female infants ($nfemale$) to the number of infants of known sex ($nknowsex$).

$$fsexratio = \frac{nfemale}{nknowsex} \tag{4.12}$$

Because a pre-breeding census is used the final two fitness components that affect the transition matrix entries for fertility (f_i) document infant survival or escape of removal prior to census. These work similarly to the juvenile and adult survival and graduation probabilities (σ_i) described above. The probability of infants escaping removal ($proboffnonrem$) is the difference of 1 and the ratio of number of infants removed ($nremoved$) and the number of female infants ($nfemale$).

$$proboffnonrem = 1 - \frac{nremoved}{nfemale} \tag{4.13}$$

Finally, the probability of female infants surviving to census given that they were not removed ($proboffsurv$) is the difference of 1 and the ratio of the number of female infants that died ($ndie$) and the number of female infants that were not removed ($nfemale - nremoved$).

$$proboffsurv = 1 - \frac{ndie}{nfemale - nremoved} \tag{4.14}$$

The hierarchical decomposition of selection requires the calculation of several

parameters from the transition matrix in Figure 4.4. Population growth rate (λ) is calculated from the transition matrix as its dominant eigenvalue. This is the finite rate of increase, and is related to the intrinsic rate of increase (r) as $\lambda = e^r$. Elasticities of the transition matrix entries document the proportional response of λ to a minute proportional increase in a matrix entry (a_{ij}) while all others are held constant. Thus, they document how much fitness responds to changes in each of the matrix entries. Formally, an elasticity the scaled partial derivative of λ with respect to the matrix entry.

$$e_{ij} = \frac{a_{ij}}{\lambda} \frac{\partial \lambda}{\partial a_{ij}} \tag{4.15}$$

Elasticities were calculated by perturbing each of the matrix entries individually and recalculating the change in λ. Because the fitness components that make up each of the matrix entries are multiplicative, the elasticities of the matrix entries are also the elasticities of each of their constituent fitness components (Caswell, 2001, p. 232).

4.2.3 Differences in Growth Rate (λ) among Rank Levels

A randomization procedure was used to test for significant differences in λ among population segments of differing social rank. Randomization methods are ideal for this application, as there is no way to construct standard errors for λ without knowing the standard errors of its constituent components (Caswell, 2001, Ch. 12). As these are unknown for the differently ranked population segments, resampling or randomization methods are the only recourse.

The growth rate of high, medium, and low ranked segments of the population were calculated for the entire span of records available (1960–2000) and three shorter timespans (1960–1973, 1974–1984, 1985–2000). As it was known that removals have impacted the rank levels differently, the probability of escaping removal was set to 1 for time spans in which removals occurred. The middle timespan (1974–1984) had less than 5 removals. Calculated λs for this period are thus very close to the realized rate of increase, which was approximately exponential population increase. For the other periods the rates will be greater than that actually observed. The first time period also corresponds to a period in which matrilines contain fewer individuals and relate more to single females. The construction of matriline rank levels for this period is more subjective and problematic. For most of the final time period the population was at high density (Figure 2.3) and a regular cull was instituted in the later years.

The randomization procedure worked as follows. The number of yearly observations of individual females in each age class and rank level was found for a chosen time span. Then, the number of records on females in each rank level was recreated by randomly dividing up the pool of records for each age class. For example, if there were 30 high rank young adult females, 40 middle ranked,

and 20 low ranked this structure was maintained in the randomly constructed groupings. This was done in each age class. Females in single matriline groups for the year were excluded. Finally, λ was calculated for each of these randomly created population segments, with all non-removal probabilities set to 1. This process was repeated 3000 times to generate a distribution of randomized λs for each population segment.

Hypothesis testing was 1-tailed as the question asked is whether high ranked females have higher fitness than medium or low ranked ones, or medium greater than low, etc. Three null hypotheses were tested with the alternative in each case that the higher ranking segment had greater λ. A significant difference was declared if the difference between the actual values for λ was exceeded in less than 0.05 (=150/3000) of cases in the difference of randomized rank levels. For example, if the observed difference between λ_{high} and λ_{low} was $1.15-1.12 = 0.03$ and only 30 out of the 3000 randomly created differences exceeded this value, then the p-value for this test is $30/3000 = 0.01$ (Manly, 1997). This difference would be declared statistically significant.

4.2.4 Differences in Fitness Components among Rank Levels

The randomization procedure just described in Section 4.2.3 identifies if there are significant differences in fitness or growth rate among the ranked segments of the population. Examination of the relationship of fitness components with rank identifies how rank alters the life history of females. In other words, it shows how these fitness differences are accomplished. For example, higher ranked females may have greater adult survival probabilities or greater birth rates.

To address this question, the 16 fitness components described in Section 4.2.2 were used in linear regression on the ordinally coded matriline rank variable (see also Figures 4.2 and 4.3, and Table 4.2). Only components calculated from the entire 1960–2000 timespan were used for these regressions because of small sample sizes in the shorter time periods. All of the fitness components are constructed such that an increase in them will produce an increase in fitness. The matriline rank variable, however, identifies high as 0, medium as 1, and low as 2 such that an "increase" in rank is movement to a lower rank level. This means that positive slopes on the regressions indicate that the fitness component increases with a drop to lower rank. Conversely, a negative slope indicates the fitness component decreases with a drop in rank. Naturally, it is expected that many of the fitness components will decrease with a drop in rank and should have negative slopes in these regressions. Matriline rank was averaged over the period in which the fitness components was measured and can result in non-integer values.

4.2.5 Differences in Other Life History Variables among Rank Levels

While the hierarchical decomposition of selection is powerful, there are two drawbacks to using it. Some of the components required are difficult to interpret or many include some more easily interpreted variable. For example, the birth rate of young adults is going to be heavily influenced by the age at which a female first gives birth. Furthermore, there are few examples in the literature of applications of this method to animal populations, though there is abundant research on simple life history variables like age of first reproduction or interbirth intervals (Table 4.1). To interface with these studies and aid in interpretation of fitness components in the hierarchical decomposition of selection, a set of commonly used life history variables were analyzed. These were age of first reproduction, lifespan, mean interbirth interval, number of offspring produced, and a transformation of the number of offspring produced that is sensitive to the age-schedule by which they were born—individual λ (McGraw and Caswell, 1996; Sade, 1990).

However, there are also two major drawbacks to using life history variables in this fashion. First, a great deal of information is lost because only females who suffered natural deaths can be used for many of the variables, and there is no way of knowing whether they are unusual given the patterning of removals (Section 3.4.4). Second, rank must be averaged over a very long period of time for most of them. The average rank over the entire lifetime must be used for all of them but age of first reproduction, for which the first 4 years of life suffice. This is the age most females have their first birth. The relationship between rank and these life history variables was analyzed in a general linear model including rank and the birth cohort to which females belonged to control for temporal differences in density, weather, and management practices. Birth cohort was treated as a categorical variable.

4.2.6 Testing for Genetic Contribution to Rank Level Differences

All of the analyses detailed above rely on the assumption that phenotypic differences among rank levels for fitness components and life history variables are solely due to the beneficial environment provided by high rank. This is equivalent to stating that rank groups are a random sampling of the genetic variation known to exist for these variables in the population (Chapter 3). This can be tested by the calculation of predicted breeding values (PBV) for the traits in question and regressing these PBVs on rank. Breeding value calculations were done in DFREML using a linear mixed model with the trait mean, birth cohort, and the linear regression of rank as fixed effects. Animal identity and the residual were the only random effects (see Section 2.5). Breeding values are the

solutions for **a** in the mixed model and can be thought of as the additive genetic value of an individual for the trait being analyzed. Low connectedness among rank levels limits the power of these analyses (Postma, 2006). By this it is meant that few individuals live in rank levels different from their close relatives. Thus, major genetic differences among rank levels will be required to identify any trend, and there is risk of Type II error. To minimize this risk as best as possible, only individuals from a large interlocking pedigree were included in the analysis. This pedigree involves 6543 known individuals, in 17 matrilines connected by numerous paternities. This is 82.43% of the entire demographic database.

4.3 Results

4.3.1 Differences in Growth Rate (λ) among Rank Levels

The rank levels of females on Cayo Santiago were found to be growing at different rates for much of the period of study (Tables 4.4 and 4.5). Using the entire span of 1960–2000 the high ranking segment of the population would have grown at a rate of about 14.7% per year (λ =1.14716). Medium and low ranking fractions would have grown at 13.7% and 12.9%, respectively. Randomization tests indicate the difference between high and low ranking segments is greater than that expected by chance (p=0.0110). The difference between high and medium, and medium and low are no greater than expected by chance.

Examining shorter spans of time, however, demonstrates important temporal differences in this pattern. For the earliest years of records 1960–1973 the expected pattern of high>medium>low ranking was not found. In fact, the low ranking segment of the population had the highest expected λ for this period. In contrast, the two later time spans (1974–1984 and 1985–2000) do have the expected high>medium>low pattern in λ. The difference in λ between high and low is significant for both of these time periods. The difference for 1974–1984 is important because these are realized rates of increase rather than ones predicted by setting the probabilities of escaping removal to one.

4.3.2 Differences in Fitness Components among Rank Levels

A number of relationships between fitness components and rank were also found. These demonstrate the life history pathways by which rank influences fitness (Table 4.6). Inclusion of data from 1960–1973, when rank and fitness were unassociated, will make these regressions more conservative. High rank provides both survival and fertility benefits for female macaques. Higher ranked mature females have greater survival probabilities (p=0.00413) as do their infants (p=0.00020) and juvenile offspring (p=0.01727). There is also a non-significant

trend for higher ranked young females to higher survival rates (p=0.19471).

Higher ranked young females have higher birth rates when only females that reproduced are analyzed (p=0.00296), and there is the suggestion of this trend, though it is not significant for mature females (p=0.06510). Including females that did not reproduce obscures these relationships. This is particularly true for the young adults; almost a quarter of the females that entered the age class died or were removed prior to reproducing. Finally, high rank seems to put females and their infants at greater risk for removal by colony managers in all phases of life. Management practices thus have balanced the rank-related propensities of the colony by differentially excising high ranked females.

Elasticities for fitness components in which there are significant rank differences can be quite large (Table 4.3). Components with large elasticities are the survival and removal probabilities. Any change in these components, while holding all others constant, will have a large effect on fitness (λ). Mature adult survival has the largest elasticity (0.41490). The fact that it is significantly related to rank means that rank differences in adult survival will greatly affect fitness. The birth rate and infant survival or removal rates have much smaller elasticities implying that rank-related differences in them will not have a substantial impact on fitness.

4.3.3 Differences in Other Life History Variables among Rank Levels

Of the five life history variables analyzed, only one has a significant relationships with rank (Table 4.7). As is well known for the Cayo Santiago colony, higher ranked individuals mature earlier (Sade et al., 1976; Sade, 1990; Bercovitch and Berard, 1993). This is also found here (p <.0001). Mean interbirth interval may also be shorter in higher ranked individuals, but this does not reach statistical significance (p=0.0602). Lifespan appears to be unrelated to rank, though its estimated regression coefficient implies higher rank may yield longer lifespan (p=.5129). Neither of the individual fitness surrogates are significantly related to rank, though their coefficients are also in the predicted direction (number of offspring p=0.2438; individual λ p=0.2475).

4.3.4 Testing for Genetic Contribution of Rank Level Differences

Regressions with predicted breeding values for the traits reported in Section 4.3.3 suggest there is no genetic basis for differences in life history traits among rank levels (Table 4.8). All of the regressions have coefficients that cannot be distinguished from zero. This implies that the observed phenotypic relationship between age of first reproduction and rank is solely the result of environmental differences, and the lack of association in the other variables is not complicated

by genetic differences. The rarity of rank reversals and few paternities available for the analysis (relative to the number of maternal links in the pedigrees) means there is low connectedness among the different rank levels *within* pedigrees. Thus it is difficult to detect any rank-related trend in the breeding values unless it is very strong (Postma, 2006). This provides suggestive evidence that life history differences among rank levels are due to environment alone. Accordingly, the common inference that rank is a proxy measure of environment quality (e.g. diet, shelter from stress) is justified, and comparisons among rank levels are not confounded by genetic differences (Silk, 1984). Furthermore, speculations on how rank can speed or slow the evolution of quantitative traits are not complicated by rank-gene covariance (Section 4.1.1).

4.4 Discussion

4.4.1 Fitness and Social Rank

The analyses presented here demonstrate fitness differences among rank levels in the Cayo Santiago females. This is true for the entire period of study (1960–2000), and for years in which there were nearly no removals and the population was allowed to grow unmanaged (1974–1984). Sade et al. (1976) and Stucki et al. (1991) identified this pattern many years ago from their analysis of several years in the 1970s. In my analysis, rank-related fitness differences were not found in the earliest years of the study period (1960–1973). This could be due to regular removal of animals for experimental purposes which disrupted social relationships, the relatively low density of the population during this period, or the subjective nature of assigning the many matrilines to high, medium, and low categories. I know of no way to untangle these factors or weight their importance. Over the entire study period, the decline in λ with a drop in rank appears to be roughly linear, provided one accepts that matrilines have been properly placed in high, medium, and low groups. By this I mean that the differences of $\lambda_{high} - \lambda_{medium}$ and $\lambda_{medium} - \lambda_{low}$ are approximately equal. This is a departure from Stucki et al. (1991), who suggested that high ranked females were much better off than medium and low ranked females, with no difference between medium and low rank. Little can be made of this difference, because assignment of matrilines to rank categories was not identical between studies.

4.4.2 Life History Differences Among Rank Levels

Fitness component differences among rank levels identify how this disparity in λ arises. Three processes are primarily responsible. First, the most important effect rank has on female life histories is to elevate the survival rate of mature adults. According to the fitness component regressions, high ranking females have mature adult survival rates of 0.971 compared to 0.942 for medium and

0.914 for low ranked females.[4] Life expectancies for each of the rank categories are all unrealistically high, but illustrate how large these differences in survival rates are. A 6-year old high ranking female can expect to live another 17.2 years on average compared to 9.4 for a medium, and 5.8 for a low ranking female.[5]

These are key differences because λ will respond the most to changes in mature adult survival (elasticity=0.415 ,Table 4.3). Many previous researchers have implicated lifespan or adult survival as the critical variable to explaining variation in reproductive success in long-lived mammals like primates (Heppell et al., 2000; Bercovitch and Berard, 1993; Altmann et al., 1988; Cheney et al., 1988), but variation in lifespan and adult survival seemed be unrelated with rank (e.g. Table 4.1). These results for the Cayo Santiago females are important, because they are the first clear demonstration that social rank affects adult survival in female primates.

Second, rank strongly affects the infant survival rate of mature adult females. Fitness component regressions predict female offspring survival rates for mature females of 0.953 for high rank, 0.907 for medium, and 0.861 for low rank. The small elasticity on this fitness component (0.079) implies these differences among rank levels will not have strong effects on lifetime fitness; it is only about one fifth of the size of the elasticity on mature adult survival. However, results with infant survival may be important for methodological reasons. Attempts to identify rank-related differences in numbers of offspring born to a female (a common fitness surrogate), or short-term reproductive success within a breeding cycle will not pick up on this effect of rank.

In contrast, infant survival of young adults is *not* related to rank. This is likely due to the inexperience or inadequate body condition, of all young females in rearing offspring that elevates infant death rates (Wilson et al., 1978; Altmann et al., 1988; Smuts and Nicolson, 1989; Koyama et al., 1992; Paul and Kuester, 1996; Bercovitch et al., 1998). This age difference may contribute to the mixed results on offspring survival reported in Table 4.1.

Finally, and more specific to the Cayo Santiago data, the management practices of the colony have clearly worked in opposition to the rank-related differences in survival and fertility. The signs of the regression coefficients of the survival and non-removal probabilities in each age class are different, implying that at all ages the "selection" operating through management is differentially removing high ranked individuals from the population. For example, the probabilities of escaping removal for mature adults are 0.883 for high, 0.921 for medium, and 0.958 for low ranking females. Multiplying them by the survival probabilities in the first paragraph of this section demonstrates that these forces approximately

[4]These have been converted from the mean-standardized scale for the regressions reported in Table 4.6 by multiplying the predicted value by the original mean (0.9449) for this fitness component from Table 4.3.

[5]Using the equation $e_x = \sum_{x=6}^{\infty} l_x$ where x is age, l_x is the probability of survival from initial age (6) to age x, and e_x is the life expectancy at age x. For the sake of this projection l_6 is assumed to be 0.5 for all rank categories.

balance one another. The transition matrix probabilities (σ_i) are all approximately equal—0.857, 0.868, and 0.876. It should also be noted that the current practice of randomly culling individuals will not eliminate this pattern. Because of the rank-related differences already noted in survival and fertility, this will occur even if removals are done at random, as high-ranked individuals are consistently tending towards greater representation in the population through both survival and fertility.

The life history variables provide far less in the way of insights into rank-related differences among female macaques. Indeed, the analysis of the life history variables takes one no further than the existing body of literature on the life histories of the Cayo Santiago females. As has already been identified, higher ranking females mature earlier (Drickamer, 1974; Sade et al., 1976; Bercovitch and Berard, 1993). This is consistent with the significant regression of young adult birth rate on rank. The only other suggestion of a relationship between rank and these life history variables is in the average interbirth intervals which may be longer in low ranked females. As in other analyses of the Cayo Santiago females and other primate populations, *no* connection was found between lifespan and rank. This may seem odd given the striking relationship between survival rates and rank just discussed. However, the sample available for assessing the lifespan-rank relationship is less than half of that in each of the age classes in the survival rate-rank regression. This lost information is effectively utilized in the hierarchical decomposition of selection (van Tienderen, 2000; Coulson et al., 2003). The great utility of this method, particularly in situations with data censored for various reasons, argues for its future application in primate demography.

4.4.3 Genetic Differences Do Not Confound Rank Level Comparisons

Predicted breeding values identified no differences among rank levels for the life history variables analyzed. The pedigree data for this portion of the study were not ideal. The lack of connectedness among rank levels limits the power to detect genetic trends or differences among the rank levels (Postma, 2006). Future work with in the Cayo Santiago colony with a pedigree interlocked by much greater numbers of paternities (or rank reversals) will be able to address these questions more thoroughly. For now, the results presented here provide weak evidence that the rank-related acceleration/delay of age of first birth is due to the beneficial environment provided by rank, and not to any genetic difference among rank levels. This is important also because it implies the selection occurring on these traits via rank is random with respect to genotype and should not alter evolutionary response beyond what is explored in Equation 4.4.

4.4.4 Mechanisms

The mechanisms rank might act through to cause these life history differences on Cayo Santiago have received mixed support from other investigators (Sade et al., 1976; Bercovitch and Berard, 1993). I agree with Sade et al. (1976) that the abundance of food on Cayo Santiago argues against nutritional deprivation as a cause of these differences. Indeed, 10% of the adult population was classified as *obese* in 1988 (Schwartz, 1989; Schwartz and Kemnitz, 1992; Schwartz et al., 1993). Some of these over-nourished females came from high ranking matrilines, but no comprehensive study of the differences in food intake or their effect on body condition among rank levels has been conducted. The only study of feeding behavior on Cayo Santiago suggests the monkeys spend considerably less time feeding (only 10.8% of their waking day) compared to rhesus in the wild or at temples (≈45%, Marriott and Roemer, 1989; Goldstein and Richard, 1989). About $\frac{1}{2}$ of this is spent feeding on the provided monkey chow with the remainder devoted to natural vegetation, insects, larvae soil (Marriott and Roemer, 1989). Nevertheless, if low ranking females are unable to meet their nutritional needs even in this abundant environment there is some potential for a role of nutrition in explaining rank related fitness differences. Limited evidence from captive rhesus macaque groups suggests that dominance has little influence on amount of food consumed, but lower ranking females spend greater times feeding to achieve this similar intake (Deutsch and Lee, 1991). Male macaques at many captive facilities are known to lose weight through the breeding season, presumably because of increased time spent copulating and guarding mates at the expense of feeding accompanied by hormonal shifts in fat metabolism (Bercovitch, 1992, 1997; Bernstein et al., 1988; Muhlenbein et al., 2002). This may be a common pattern for male cercopithecoids (Alberts et al., 1996).

The role of stress in the Cayo Santiago colony in producing rank-related fitness differences is somewhat clearer. Injuries from bites and scratches are often observed and many individuals have scars of past conflicts. Osteologically this is evident in infections of the periosteum and sometimes extreme skeletal degradation. Deep infections may occur because of a high frequency of bites on bony joint elements on the hind limb and tail as an individual flees during a conflict (Jean Turnquist, personal communication). Psychologically induced stress from conflicts or lack of social partners to interact with affiliatively may also differentially impact low-ranking females (Sapolsky, 2005; Tamashiro et al., 2005). If stress causes immuno-incompetence, lower-ranking females may have greater numbers or duration of infections and parasite loads. However, surveys of intestinal parasites in the Cayo Santiago macaques have failed to demonstrate any rank-related differences (Kessler et al., 1984). Further studies of viral antibody concentrations have not explored rank differences (Sariol et al., 2006; Kessler et al., 1989; Kessler and Hilliard, 1990). Additional evidence on the levels of circulating stress hormones is currently unavailable in this popu-

lation. However, there is abundant evidence that lower ranking individuals in multimale-multifemale social systems like rhesus macaques have higher cortisol levels both in the wild and in captivity (Sapolsky, 1983; Abbott et al., 2003).

There is no evidence for *costs* of high rank in female macaques. While hormonal mechanisms that might function to increase aggressive behavior for the attainment of rank but decrease fertility or survival have been documented in other studies (Packer et al., 1995; Creel, 2001), there is no evidence for this in the Cayo Santiago females. The effect of rank is substantially positive. This does not imply that there are no costs to high rank, simply that any costs of high rank that exist are more than offset in the demographic variables tracked in this analysis. The cooperative nature of rhesus female aggression may dilute costs onto many individuals (Silk, 2002; Datta, 1983). However, no special explanation is required, as costs of high rank are rarely documented (Table 4.1).

4.4.5 Genes "for Rank"

Rank itself cannot have a genetic basis because it is a property of the interactions among individuals forming a group (Moore et al., 2002; Moore, 1993; Barrette, 1993; Capitanio, 1993, 1991; Dewsbury, 1993). Nevertheless, a question worth addressing is whether these results in any way clarify the genetic basis of behaviors related to the acquisition and maintenance of rank in rhesus females. The traits explored in this analysis are life history variables. Some of them differ markedly among rank categories and some of display genetic variation (Chapter 3). However, these results are mute on the genetic basis of rank-related behaviors themselves. Investigation of the genetic basis of these behavioral traits in rhesus females could be accomplished at Cayo Santiago (see Dingemanse and Réale, 2005; Reale et al., 2000). However, this will require detailed behavioral observations and much more highly connected pedigrees with more paternities to break apart matriline gene-rank environment associations.

As suggested in Section 4.1.2, the rank-related behaviors such as severity of violence, frequency of aggressive acts, and associated submissive responses likely depend on life experiences interacting with genetically primed neuroendocrine systems (Suomi, 2006; Newman et al., 2005). It is not that individual females attain high or low rank because of genetically-determined behaviors but rather their social environments during development affect these neuroendocrine pathways such that particular sets of behaviors come to typify rank categories. Known genetic differences in serotonergic and dopaminergic pathways among macaque species may be related to thresholds of responsiveness during development to psychosocial stress inputs (Wendland et al., 2005). Alternatively, polymorphism noted in despotic rhesus macaques may indicate a variety of selection pressures at this locus including heterozygote advantage, density or frequency dependence, or spatial and/or temporal heterogeneity in fitness. Regardless, the results reported here indicate there are strong fitness

costs and benefits to the social positions females acquire within groups. This variation in fitness is elevated opportunity for selection and drift at these loci.

4.5 Summary

The identification of fitness differences due to social rank is of critical interest to primatologists because of their potential effects on adaptive and neutral evolution. Four major results of this chapter address this topic. First, there are clear differences in fitness among high, medium, and low ranking population segments of females at Cayo Santiago. This establishes the generality of a pattern in this population first noted by Sade et al. (1976) for a short period of time. Second, these fitness differences are accomplished through differences in survival and fertility, though it is the differences in survival—particularly of mature adults—that have the greatest effect on fitness. Third, the hierarchical decomposition of selection is an excellent methodological tool for assessing the relationship between variables such as social rank and fitness, particularly in the context of incomplete records as on removed Cayo Santiago females and their infants. Finally, analysis of breeding values provides some weak evidence that the well known rank-related differences among females in age of first birth are entirely due to the beneficial environment provided by rank and are not due to genetic differences among ranks. While there is genetic variation in life history traits and fitness components it appears to be randomly distributed among rank levels and will not complicate predicting response to selection.

4.6 Tables and Figures

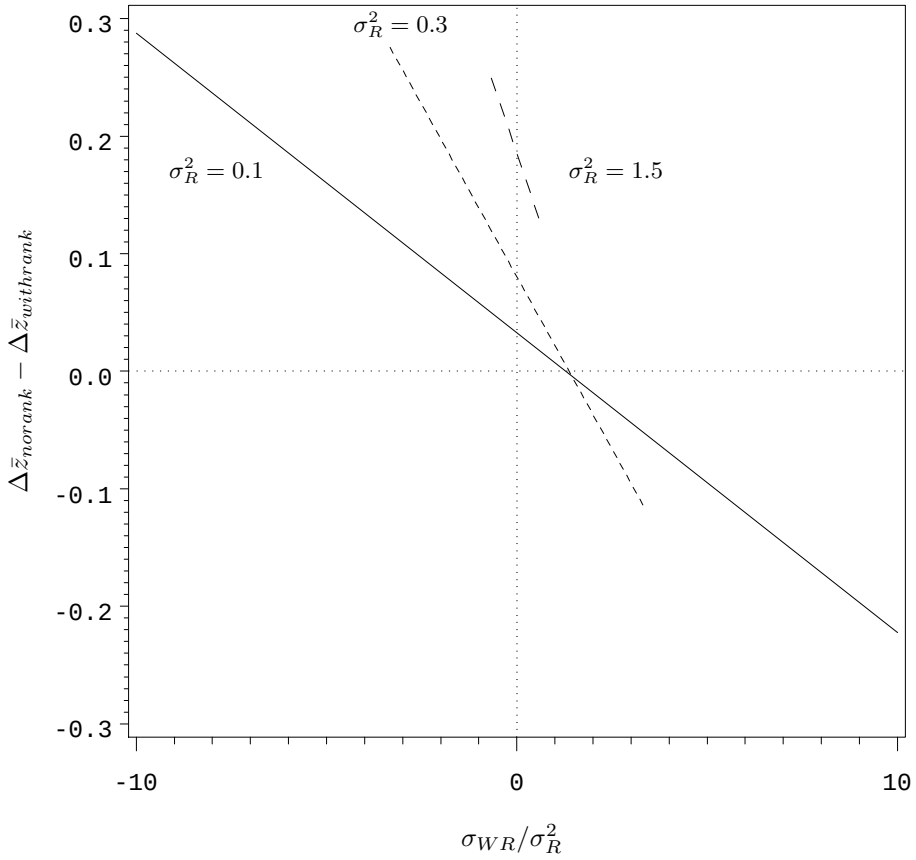


Figure 4.1: Graphical representation of the potential effects of social rank on the evolution of a quantitative trait under directional selection, such as a component of fitness (Equation 4.4). Three values for σ_R^2 are shown. The domain of each line is limited by exploring only values between -1 and 1 for σ_{WR}. Other variances and covariances are held constant: $\sigma_{WA} + \sigma_{WE} = 1$, $\sigma_A^2 = 0.5$, and $\sigma_E^2 = 0.8$. In the upper half of the figure ($\Delta\bar{z}_{norank} - \Delta\bar{z}_{withrank} > 0$) rank acts to *slow* the evolution of the trait either because $\sigma_{WR} < 0$ (upper left quadrant) or σ_R^2 is relatively large (upper right quadrant). In the lower half of the figure rank acts to *accelerate* the evolutionary response because $\sigma_{WR} > 0$ and σ_R^2 is relatively small. Generally, $\sigma_{WR} > 0$ for fitness components because high rank is thought to elevate fitness. By this logic, how rank will affect the evolutionary response of fitness components depends on the sizes of σ_{WR} and σ_R^2.

Table 4.1: Old World monkey and ape species in which the relationship between social rank and reproductive success has been explored. This is a selection of higher-quality literature sources excluding captive and short-term studies. Cells in the table answer the question "Do higher ranking females have x?" for each column with "Y"=yes and "N"=no. Statistically significant and non-significant trends are pooled such that a non-significant but noted trend in the predicted direction is labelled as "Y". The x are defined in the table footnotes. Their numbering is unrelated to that in Figure 4.2 and Table 4.2.

Species	site[a]	years of study	fitness or measured component												
			1	2	3	4	5	6	7	8	9	10	11	12	13
Macaca[b]															
fascicularis	W	12					Y	N	Y			Y		Y	
fuscata	F	11-30+	N		Y		N	N	?			N		N	
fuscata	P	11						Y	Y	N					Y
fuscata	W	34					Y	Y	Y		Y	N			
fuscata	P	28		Y				N							
mulatta	F	20-30					N		Y		Y	N		Y	Y
sinica	W	16					N	Y		Y		Y		Y	Y
sylvanus	F	11					N	Y	Y						
Papio[c]															
anubis	W	25		N	N	N	Y	Y	Y					Y	
cynocephalus	W	30					Y	Y	Y		Y				
cynocephalus	W	22			N	Y	Y	Y	Y			Y		Y	
ursinus	W	10+					Y	N	N		Y	N		N	
Cercopithecus[d]															
aethiops	W	10			N		N	N	N			N	Y		
Pan[e]															
troglodytes	W	22	N				Y	Y	Y	N	Y	Y		Y	
column sum	Y		0	1	1	1	7	8	9	1	5	4	1	6	3
	N		2	1	3	1	5	5	2	2	0	5	0	2	0

[a]W=wild, P=provisioned, F=free-ranging Column numbers indicate the following variables: 1=higher frequency of copulation, 2=fewer infertile cycles, 3=earlier conception in breeding season, 4=lower rate of abortion or miscarriage, 5=lower interbirth interval after surviving infant, 6=higher offspring survival rate, 7=earlier age of first parturition, 8=larger adult body size, 9=higher offspring growth rate, 10=longer life span, 11=lower rate of disease mortality, 12=higher # offspring, 13=higher λ.

[b]Ketambe, Indonesia (van Noordwijk and van Schaik, 1999); Arashiyama, Japan and "West" group in Texas Dominance hierarchies are quite flexible in these groups. "?" indicates a significant "Y" the authors declare to be "N" (Takahata et al., 1999; Takahata, 1980; Koyama et al., 1992; Fedigan et al., 1986; Fedigan, 1991; Gouzoules et al., 1982; Wolfe, 1984); Mt. Ryozen, Japan (Sugiyama and Ohsawa, 1982); Koshima, Japan animals were provisioned for part of the study period (Watanabe et al., 1992; Mori, 1979); Katsuyama, Japan (Itoigawa et al., 1992); Cayo Santiago, Puerto Rico (Stucki et al., 1991; Sade, 1990; Sade et al., 1976; Bercovitch and Berard, 1993; Berman, 1988; Bercovitch and Goy, 1990); Pollonaruwa, Sri Lanka (Dittus, 1986, 1979, 1998); Salem, Germany (Paul and Kuester, 1990; Paul and Thommen, 1984)

[c]Gombe, Tanzania (Packer et al., 1995); Amboseli, Kenya (Altmann and Alberts, 2003b,a; Silk et al., 2003; Altmann and Alberts, 1987; Altmann et al., 1988); Mikumi, Tanzania (Wasser et al., 2004; Rhine et al., 2000); Moremi, Botswana (Cheney et al., 2006; Johnson, 2006)

[d]Amboseli, Kenya (Cheney et al., 1988, 1981)

[e]Gombe, Tanzania; animals were provisioned for part of the study period (Pusey et al., 1997; McGrew, 1996)

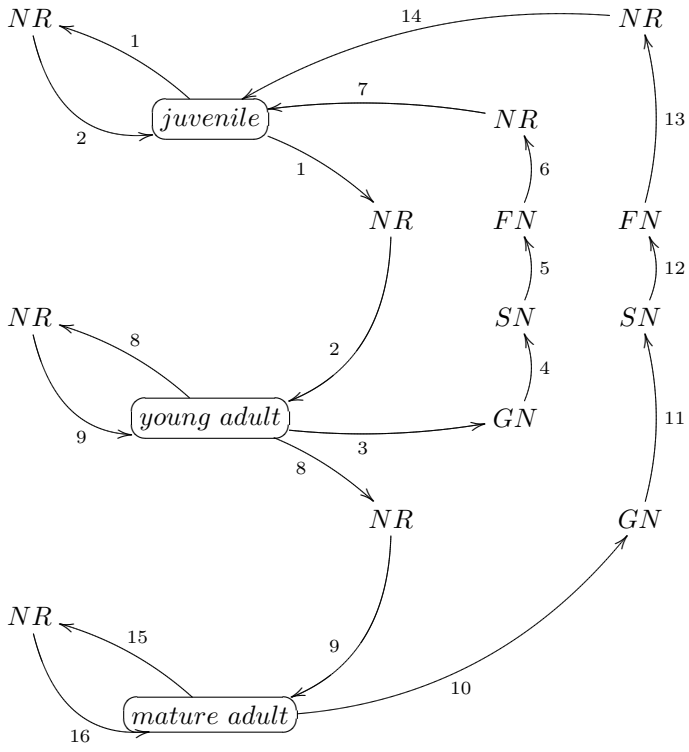


Figure 4.2: A life cycle diagram illustrating fitness components in female rhesus macaques from Cayo Santiago. Juveniles are 1–2 cohort years old, young adults 3–5, mature adults ≥6. Symbols used are as follows: NR=not removed, GN=generic (all) newborns, SN=sexed newborns, FN=female newborns. Transitions between stages are computed by the multiplication of the numbered paths, which are probabilities, between the stages. A key to the numbered fitness components and further explanation of how the total probability of each stage transition is computed are given with Table 4.2. Note that this numbering in unrelated to that in Table 4.1.

Table 4.2: Fitness components illustrated in Figure 4.2. The top table contains number references for survival/graduation probabilities. The values in the cells of these tables can be thought of as subscripts of w for consistency with van Tienderen (2000). The survival/graduation probabilities (σ_i) are the product of the non-removal and survival given non-removal probability for each age class. The bottom table contains number references for reproduction probabilities. The product of all elements in a row is the pre-breeding projection matrix entry for fertility (f_i). Juvenile probabilities for reproduction, which do not appear in Figure 4.2, are 0 or undefined by definition and are left unnumbered.

stage	non-removal	survival given non-removal	σ_i
juvenile	1	2	1×2
young adult	8	9	8×9
mature adult	15	16	15×16

stage	birth rate	known sex	female sex ratio	offspring non-removal	offspring survival given non-removal	f_i
juvenile	–	–	–	–	–	–
young adult	3	4	5	6	7	$3 \times 4 \times 5 \times 6 \times 7$
mature adult	10	11	12	13	14	$10 \times 11 \times 12 \times 13 \times 14$

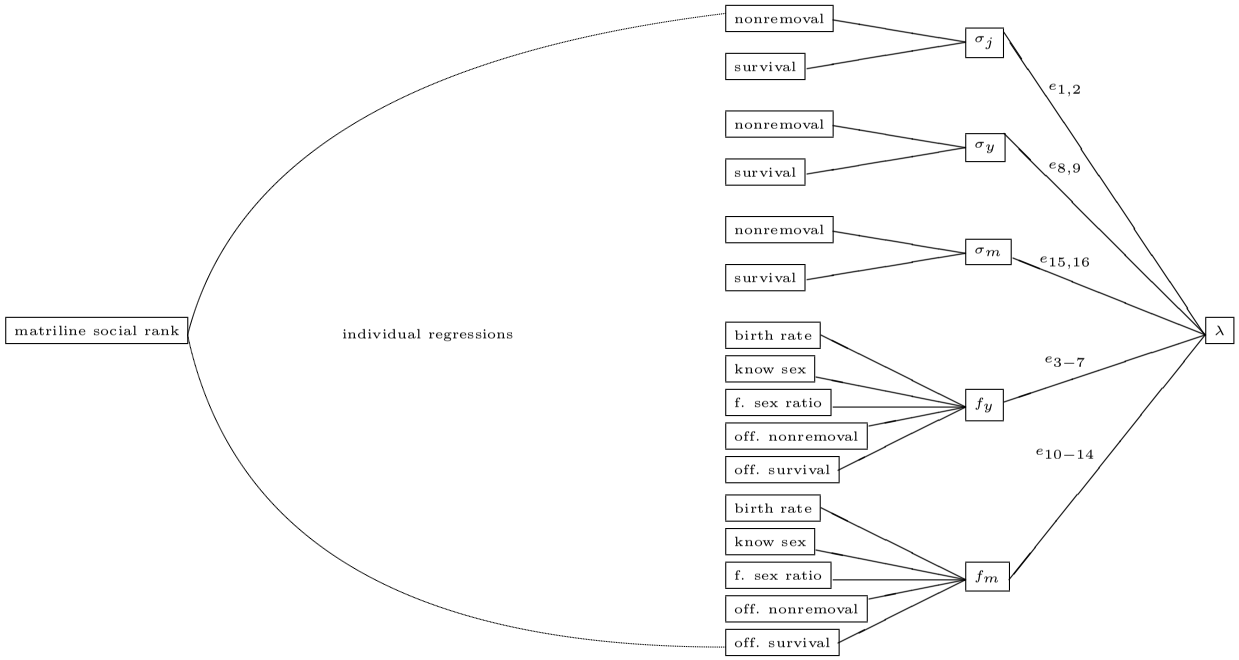


Figure 4.3: Elastogram illustrating the locations of the calculated elasticities in determining λ. Paths on the left side of the figure connect matriline social rank with the fitness components and represent the bivariate regression of the component on social rank. Correlational paths can also be drawn on the left hand side. These are the regressions of each fitness component on every other, and can be used in searching for trade-offs among components and calculating integrated elasticities (see Chapter 5; van Tienderen, 2000, 1995).

$$\mathbf{A} = \begin{pmatrix} 0 & 0 & f_y & f_y & f_y & f_m \\ \sigma_j & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_j & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma_y & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma_y & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma_y & \sigma_m \end{pmatrix}$$

Figure 4.4: The resulting transition matrix containing the survival (σ_i) and fertility (f_i) probabilities calculated in Tables 4.2. Each stage, i, probability occurs the number of times as the duration of the stage. Stage durations are given with Figure 4.2. The subscripts j, y, and m refer to juveniles, young adults, and mature adults, respectively.

Table 4.3: Elasticities of λ to fitness components for Cayo Santiago females 1960–2000.

fitness component	number in Table 4.2	elasticity	component value
juveniles: 1–2 years old			
probnonremoval	1	0.21550	0.88366
probsurvival	2	0.21550	0.93576
young adults: 3–5 years old			
birthrate	3	0.02905	0.40888
knowrate	4	0.02905	0.98037
fsexratio	5	0.02905	0.49706
proboffnonrem	6	0.02905	0.95379
proboffsurv	7	0.02905	0.88944
probnonremoval	8	0.26185	0.90722
probsurvival	9	0.26185	0.95759
mature adults: ≥6 years old			
birthrate	10	0.07870	0.76312
knowrate	11	0.07870	0.98543
fsexratio	12	0.07870	0.47978
proboffnonrem	13	0.07870	0.94563
proboffsurv	14	0.07870	0.91231
probnonremoval	15	0.41490	0.91679
probsurvival	16	0.41490	0.94490

Table 4.4: λ for categories of matriline social rank at Cayo Santiago over different time intervals.

rank category	λ			
	1960–2000	1960–1973	1974–1984	1985–2000
high	1.14716	1.13498	1.14756	1.14983
medium	1.13683	1.12219	1.13958	1.13898
low	1.12865	1.14947	1.11101	1.12784

Table 4.5: Randomization p-values for hypothesis tests on λs in Table 4.4. p is the probability the null hypothesis H_o: $\lambda_x = \lambda_y$ is true. Tests are 1-tailed. See Section 4.2.3 for details of randomization procedure.

alternative hypothesis H_a	p					
	1960–2000		1974–1984		1985–2000	
$\lambda_{high} > \lambda_{medium}$	0.1047		0.3353		0.1337	
$\lambda_{medium} > \lambda_{low}$	0.1790		0.0657		0.1527	
$\lambda_{high} > \lambda_{low}$	0.0110	*	0.0083	*	0.0247	*

* $p < .05$

Table 4.6: Regressions of fitness components from hierarchical decomposition of selection (1960–2000) on matriline social rank categories for Cayo Santiago females.

fitness component	n	$R^2 \times 100$	intercept	β	p	
juveniles: 1–2 years old						
probnonremoval	1970	1.01	0.96392	0.03783	0.00001	***
probsurvival	1851	0.31	1.01990	-0.02043	0.01727	*
young adults: 3–5 years old						
birthrate	1240	0.01	1.00693	-0.00753	0.75176	
birthrate[1]	940	0.94	1.37249	-0.05651	0.00298	**
knowrate	940	<0.01	1.00015	-0.00016	0.97643	
fsexratio	925	0.29	0.94478	0.05856	0.09929	
proboffnonrem	557	1.10	0.97505	0.02591	0.01334	*
proboffsurv	535	0.02	0.99427	0.00586	0.73254	
probnonremoval	1240	4.65	0.92717	0.07920	<0.00001	***
probsurvival	1160	0.15	1.00970	-0.01009	0.19471	
mature adults: $\geq$ 6 years old						
birthrate	754	0.06	1.00992	-0.01102	0.51051	
birthrate[1]	698	0.49	1.10037	-0.02220	0.06514	
knowrate	698	0.05	1.00256	-0.00283	0.55139	
fsexratio	693	0.35	1.04173	-0.04611	0.11722	
proboffnonrem	575	0.60	0.98131	0.02117	0.06443	
proboffsurv	556	2.46	1.04482	-0.05038	0.00020	***
probnonremoval	754	1.24	0.96290	0.04121	0.00221	**
probsurvival	707	1.16	1.02710	-0.02971	0.00413	**

* $p < .05$, ** $p < .01$, *** $p < .001$

[1] Regression only using values for females that gave birth one or more times in this age class.

Table 4.7: Differences in life history variables among rank categories. Regression models include the continuous effect of rank level and categorical effect of birth cohort. Data from 1960 to 2000 are used. Birth cohort was not significant in models for lifespan, number of offspring, or individual λ. Dropping it did not change the significance of rank. For age of first reproduction rank is the average rank over the first four years of a female's life. For the other variables it is the average over her entire lifespan.

variable	n	model p	$R^2 \times 100$	rank β	rank p	
age of first rep.	1033	<0.0001	21.63	0.0950	<0.0001	*
lifespan	248	0.7277	10.25	-0.3213	0.5129	
mean IBI	210	0.0038	24.74	0.0466	0.0602	*
# offspring	248	0.8618	9.01	-0.4476	0.2438	
individual λ	248	0.8354	9.29	-0.0131	0.2475	

* $p < .10$ for rank

Table 4.8: Breeding value regressions for life history variables and fitness surrogates on social rank. Number of individuals for each regression is reduced (see Table 4.7) because data were limited to a single interlocking pedigree.

	n	intercept	β	p
age of first rep.	847	-0.0058	0.0019	0.5205
lifespan	187	0.4468	-0.0215	0.8682
mean IBI	132	0.0008	-0.0001	0.9209
# offspring	187	0.1063	-0.0011	0.9678
individual λ	187	0.0074	-0.0012	0.6442

Chapter 5

Trade-Offs Among Components of Fitness and Life History Traits

5.1 Introduction

5.1.1 Trade-Offs

Trade-offs are to understanding adaptation. Trade-offs occur when organisms pay a fitness cost to gain a fitness benefit. They can be biomechanic (Svanbäck and Eklöv, 2003), behavioral (Marler and Moore, 1988), or ingrained in the life history of an organism (Reznick, 1985; Bell and Kofoupanou, 1986; Stearns, 1989). In life history studies trade-offs are generally defined as negative relationships between traits that contribute positively to lifetime fitness. These traits are called fitness components (Hughes and Burleson, 2000), and are typically age-specific rates of survival and fertility.

Trade-offs are important because they represent limits on adaptation. To illustrate this in life history evolution, one can contrast some hypothetical organisms (Law, 1979). The "Darwinian Demon" is an organism that is born sexually mature, reproduces in enormous quantities, and lives forever. It follows the ideal life course to maximize fitness. That there are no organisms capable of doing so suggests there are trade-offs among different life history variables, or components of fitness. The "Darwinian Demon" operates without the constraints of finite resources, metabolic relationships among life history variables, or the shackles of phylogenetic heritage.

While there is only one "Darwinian Demon" imaginable, there are many possible "Darwinian Dolts." These would be organisms who maximize some component of fitness, but fail to achieve high lifetime fitness. They do not achieve the proper balance among components that maximizes fitness. For example, one possible "Darwinian Dolt" lives its entire long life without reproducing and then dies. It has maximized survival but at the total expense of reproduction. The role of trade-offs in life history theory then is to identify why and in what proportions organisms are willing to pay costs in one fitness component for the benefits rendered through another component.

Life history trade-offs are of great interest for students of primate and human biology. Primate life histories are distinguished from other mammals of similar body size by long sub-adult periods, low reproductive rates, and potentially very long lives (Harvey et al., 1987; Kappeler and Pereira, 2003). Identifying

ecological and social correlates of these life history characteristics has been a long-term goal of primate biology because they elucidate potential adaptive scenarios for why the entire Order would trade-off reproduction for survival at their earliest paleontological emergence (Williams, 1966a; Prothero and Jurgens, 1987; Wilkinson and South, 2002; Hartwig, 2007). Emerging extinction threats to many living primate populations have refocused investigation of primate life histories on how they can best be managed in spite of their limited capacity for population increase and often narrow range of habitat requirements (Strier, 2007; Blomquist et al., 2007).

Human life histories share a great deal with other primates, but offer unique twists on this common theme (Smith and Tompkins, 1995). Humans have even longer sub-adult periods than close primate relatives of similar body size and can have much longer lifespans, but humans typically have higher reproductive rates (Hawkes et al., 1998). The long human period prior to sexual maturity has also been suggested to have novel aspects. Bogin (1999) has argued that childhood—the period from ages 3–7 "following infancy, when the youngster is weaned from nursing but still depends on older people for feeding and protection" (Bogin, 1997)—is a novel insertion into the human developmental trajectory, not found elsewhere among primates. He also notes that humans have a growth spurt in body mass and long bone length during adolescence. Body mass growth spurts have been documented in a number of primate species (Leigh, 1994), and there is some evidence for long bone growth spurts for chimpanzees (Buchanan, 2002; Hamada and Udono, 2002). When in human prehistory these distinctive life history features emerged is an active area of paleoanthropological research. Current consensus is that elements of the human growth pattern, and possibly other life history traits, emerged at the earliest with *Homo erectus* (broad sense), but possibly no earlier than in anatomically modern *H. sapiens* (Zihlman et al., 2004; Caspari and Lee, 2004; Coqueugniot et al., 2004; Leigh, 2004; Dean et al., 2001; Smith, 1994; Trinkaus and Tompkins, 1990).

Culture affects human life histories. The ability of humans to provide one another with resources, regardless of whether they are kin, complicates the simple assumptions about limiting resources of most animal populations. The "grandmother hypothesis," initially an explanation for the occurrence of menopause, is the most famous example of this (Hawkes et al., 2003, 1998; Williams, 1966a), whereby elderly females curtail their own reproduction to increase the amount of aid they can provide to their daughters in raising infants. However, recent theoretical work on human demography has suggested this may be part of a more general phenomenon—that long human lifespans may be as much a way of producing commodities that are transfered to social partners as it is about producing offspring themselves (Lee, 2003; Kaplan and Robson, 2002). These economic insights into human life history identify "production" more generally than "reproduction" and complicate a naïve biological analysis of human life history evolution.

Culture also offers non-biological reasons to consciously change life history. The widely discussed demographic transition—reduced fertility and increased survival, particularly at young ages—in Western countries is unlikely to have a biological basis, though reproduction and death are clearly biological events (Korpelainen, 2003; Mace, 2000; Kaplan and Lancaster, 1999; Borgerhoff-Mulder, 1998). Instead cultural change has altered the utility of offspring. Parents no longer desire children for agrarian labor, nor must they conceive more offspring to offset high infant child mortality due to medical technologies. Instead they can have few offspring in whom they invest heavily. Fitness maximization is not the driving force behind this decision; it can be done purely to increase the happiness of parents (Roth, 2004; Lam, 2003; Becker, 1991).

5.1.2 When Should We Find Trade-Offs?

Despite the importance of trade-offs to life history theory they are notoriously difficult to document (Roff and Fairbairn, 2007; Stearns, 1989; Bell and Koufopanou, 1986). At least four factors may contribute to the empirical scarcity of trade-offs. Recognizing their effects should temper over-emphasis on negative results and aid in designing effective research programs.

First, environmental variation can mask real trade-offs. Many organisms, including primates, do not encounter environments with equal resources or threats to their livelihood. Good environments for one fitness component are often good environments for others. For example, both reproduction and survivorship are positively affected by increased food availability (Altmann and Alberts, 2003b; Strum and Western, 1982). Where a negative relationship is expected between these fitness components when their correlation is explored among population members, a positive one is often found. Grafen (1988) calls this phenomenon the "silver spoon effect." Some individuals are born into good environments that they will enjoy throughout their lives; others suffer from the bad environments they encounter. As a economic analogy, one can consider the relationship between housing and automobile costs in normative U. S. culture (van Noordwijk and de Jong, 1986). We might predict that people who spend more money on their house have less money to spend on a car—the price of houses and cars people buy should be negatively related in the population. However, this is usually *not* the case because there is much greater variation in populations for the resources they acquire (income) than in the allocation decisions they make (car v. house). More often the relationship is positive because rich people can afford to spend lavishly on both housing and transportation. If we were able to statistically control the income of individuals (e.g. look just at the allocation decisions of people in similar income categories) we might observe the predicted negative relationship (Borgerhoff-Mulder, 2000).

For animals, which are assumed to make allocation decisions to maximize fitness, one productive avenue for controlling environmental variation that in-

hibits documenting trade-offs is the examination of genetic instead of phenotypic correlations among fitness components (Reznick, 1985). Genetic correlations (off-diagonal elements in **G**, see Section 2.4) result from physiological and developmental linkages among fitness components (Stearns, 1989, 1992). When properly estimated they reflect the additive action of genes on different measured traits—pleiotropy—and should not be complicated by environmental variation such as "silver spooning." Through their role in multivariate response equations, negative genetic correlations are effective constraints on the simultaneous evolution of increased values for fitness components. For this reason genetic correlations are sometimes discussed as evidence of "microevolutionary trade-offs" (Stearns, 1992).

However, a second factor looms in exploring genetic correlations. This is because even at the genetic level, acquisition-allocation hierarchies can eliminate trade-offs. Quantitative genetic models demonstrate that when there is greater genetic variation for acquiring resources from the environment than there is for allocating them to different metabolic process or fitness components, trade-offs will not be observed (Houle, 1991; de Laguerie et al., 1991; de Jong and van Noordwijk, 1992; de Jong, 1993; Worley et al., 2003). Most observers consider this to be a common situation. This is because how effective animals are at gathering resources from their environment is often dependent on many traits which may all be genetically variable to some degree (i.e. have many segregating loci). Metabolic allocation decisions are probably more finely controlled by a limited number of loci and physiological "switches," such as hormones (Finch and Rose, 1995).

Third, it is naïve to expect all correlations among fitness components to be negative. Verbal models for negative genetic correlations due to pleiotropy emphasize that: mutations with favorable effects on two traits should be quickly fixed, those with mutually detrimental effects will be quickly eliminated, so only those that have positive effects on one trait and negative effects on another will remain in populations (Roff, 1997). The simplicity of this verbal model is appealing but it ignores much of the complex genetic architecture of fitness components (Merilä and Sheldon, 1999) and denies that there may often be more than two fitness components involved in trade-offs. Charlesworth (1990) explored genetic correlations among fitness components in simple model life cycle in which he specified a limited number of functional constraints. Genetic correlations between functionally constrained traits are -1 in the resulting life cycle, but those between other traits can be weakly negative or even positive. In sum, expectations of strong negative genetic correlations among *all* fitness components are unwarranted (Roff, 1994, 2002, p. 85). If the life cycle of an organism can be divided into two meaningful variables that capture relevant trade-offs, then their genetic correlation should be strongly negative. With the life cycle more elaborately modeled with larger numbers of fitness components there is no *a priori* reason to expect negative correlations between any pair of

them. For genetic correlations to be complete constraints on adaptive evolution of life history traits, the determinant of the **G** matrix must be 0 (Charlesworth, 1993). This implies a mixture of mathematical signs in **G** is still a feasible constraint.

Finally, gene×environment interactions complicate our understanding of trade-offs. Gene×environment interactions, or reaction norms, are situations in which a single genotype produces different phenotypes in different environments; genetic expression is environment-dependent. Stearns (1991; 1992) provides examples of crossing reaction norms such that in one extreme environment a positive genetic correlation is observed, in the other extreme a negative correlation is found, and in the middle there is no correlation at all. Relating these changes to food availability he suggests that no general conclusion can be made—which correlation is found and under what conditions depends on animal physiology, should vary among taxa, and can only be determined experimentally. While the expression of any trait is environmentally dependent to some degree, the empirical difficulties of exploring this in any primate population preclude its further pursuit with the Cayo Santiago females. However, the recognition that genetic correlations, and the trade-offs they entail, depend on the environment of measurement implies they must be assessed in a naturalistic setting or, at the very least, in standardized conditions. Whether negative genetic correlations emerge in this environmental range in the reaction norms of the population is an open question. Nevertheless focused analysis of trade-offs, even in a single environment, will be an advance from the diffuse discussion of plasticity that pervades the primatological literature (Lee and Kappeler, 2003; Silver and Marsh, 2003; Fuentes, 1999).

5.1.3 Important Trade-Offs for Female Primates

Considerations outlined in the previous section imply there is often little hope of identifying trade-offs by an uninformed search through phenotypic or genetic correlation matrices of fitness components. Ideally a trade-off function would be estimated from phenotypic data, physiological and developmental links among fitness components would be known, and the details of the genetic architecture of traits would be fully explored (Stearns, 1989). In the absence of this wealth of data, one can search for trade-offs with a few key fitness components in mind. Isolating which trade-offs ought to be important for a particular species or sex may be crudely done through comparisons with other taxa. While there are many potential trade-offs in the life histories of female animals only a handful have received much attention, and few of these are likely to be critical for primates.

One classic trade-off is between offspring quality and quantity. In this case an allocation decision is made between increased numbers of low quality offspring, or decreased numbers of high quality offspring. Lack (1947; 1948; 1954b) was

the first biologist to investigate this trade-off, focusing on clutch size in birds and litter size in mammals. Lack's insight, novel for the time, was that selection favors intermediate sized clutches or litters. If they were too small, then genes that promoted small clutches would be numerically overwhelmed in future generations, but if they were too large then parents could not adequately feed offspring—reducing their chances of survival to adulthood and perhaps diminishing their reproductive ability as adults.

While there has been interest in offspring quality/quantity trade-offs in primates (Hagen et al., 2006; Borgerhoff-Mulder, 2000; Hill and Hurtado, 1996), aspects of primate biology suggest it is relatively unimportant. Unlike the birds and mammals Lack discussed, primates typically rear a single offspring at a time (Eisenberg, 1981; Starck and Ricklefs, 1998). There can be no direct competition for resources within a litter of one. Longer term depletion of resources must be operating for there to be trade-offs between offspring quality and quantity with singleton births (Jönsson, 1997). This is not to say that the span of time between reproductive events is unaffected by trade-offs in primates. It is conceivable that female primate do face trade-offs in when they wean their offspring and conceive a subsequent one. However, this trade-off is entangled with allocation decisions made about the current and future reproduction of the mother—not just the quality and quantity of present offspring.

The spacing of births can have interesting ameliorative effects on offspring quality/quantity, as well. Human children and adolescents are often important caregivers to their younger siblings (Draper and Hames, 2000; Cicirelli, 1994; Weisner and Gallimore, 1977). Increasing the number of offspring can actually make life easier for a parent. Primates may face offspring quality/quantity trade-offs, but they are likely to be far less important in structuring primate life histories than other mammals and birds where there is synchronic competition for resources among siblings. In extreme cases where resources are limited, or can be diluted among siblings of different ages, offspring quality/quantity trade-offs may emerge in humans or other primates. For example, Strassmann and Gillespie (2002) demonstrated in a West African farming community, the optimal completed family size (4.1 surviving offspring) was not achieved by giving birth to the highest number of offspring. Instead, childhood mortality risk was elevated by increasing fertility beyond the ideal number of offspring born. However, this levelling off in fitness returns from increased reproductive output is relatively minor compared to the differences in clutch/litter survival discussed in other animals (Lack, 1947, 1948, 1954b,a). In graphical terms, the fitness landscape for human or primate offspring quality/quantity trade-offs in fairly shallow—penalties for deviating from the optimum are small. In other animals there are sharper drop-offs; mothers pay dearly for having too few or too many offspring (compare figures in Strassmann and Gillespie versus those in Ch. 7 of Stearns, 1992). Nevertheless, trade-offs of this kind can be explored in the Cayo Santiago females by relating birth rates and offspring survival rates.

Another classic trade-off of interest in life history studies is between growth and reproduction. This trade-off is most obvious in animals that can do both at the same time, including plants, invertebrates, fish, amphibians, and reptiles (Heino and Kaitala, 1999). The theoretically interesting aspect of this trade-off is that increased size usually leads to increased fertility (benefit), but abstaining from reproduction to grow is time in which an animal might die and conspecifics are reproducing (costs). Primates do not face this trade-off to the degree of fish and reptiles, although there is a widely known theory of mammalian life history evolution in which this is a central trade-off (Charnov, 1993, 1991), and there are elegant models of optimal reaction norms for age and size at maturity (Kawecki and Stearns, 1993; Stearns and Koella, 1986; Kozlowski and Uchmanski, 1987). This is because primates have a narrow period of life in which these processes can occur simultaneously (Leigh and Bernstein, 2006); most primate life is devoted to either infant and juvenile growth and development or reproductive adulthood. Models of age and size at maturity for primates and other mammals focus on this as a switch that balances the costs of mortality and not reproducing as a sub-adult and the benefits of reproducing, and perhaps reduced mortality risk, at larger size. Because these are diachronic processes, they are not directly models of growth and reproduction trade-offs but incorporate one through their connections with sub-adult mortality and adult mortality and fertility (Charnov and Berrigan, 1993; Charnov, 1991).

There is, however, growing evidence that pre- and postnatal conditions affecting growth rates can impact adult fertility and survival (Lummaa and Clutton-Brock, 2002; Bateson et al., 2004; Metcalfe and Monaghan, 2001). Catch-up growth, in particular, appears to have pervasive negative effects on the health of adults. Opportunities for studying these kinds of trade-offs in primates other than humans is not feasible at this time because of the dual requirements of matched longitudinal morphological data on growing individuals and long-term records of their demographic performance.

The clearest set of trade-offs for female primates are between current reproduction and survival or, more generally, current and future reproduction. The primate emphasis on extended juvenility, long lifespan, and reduced reproductive rate implies it takes primate females a long time to achieve reproductive success. Much of their ability to attain high fitness depends on their continued survival from year to year. Simple demographic models of primate life histories demonstrate that lifetime fitness (λ) is most responsive to adult survival rates. This is in contrast to other animals who can live short lives but produce many offspring in only a few reproductive bouts (Blomquist et al., 2007; Lawler and Caswell, 2006; Heppell et al., 2000).

Trade-offs between current reproduction and survival or future reproduction may act over a variety of time scales. In the most narrow range, birth rates and survival rates of individuals in the same age class may be negatively correlated. More expansive time scales may also be required to identify trade-

offs, such as birth rates in non-adjacent age classes, or birth rates in young age classes and much later survival rates (Rose and Charlesworth, 1981). If costs of reproduction have long-term additive or multiplicative consequences, it is these comparisons of distant life history events that will reveal trade-offs. Exploring relationships between life history variables can supplement results on fitness components. For example, average interbirth intervals may be positively correlated with lifespan. This would indicate a trade-off between lifespan and reproductive rate—for a long life a female must space her births out more. Similarly, a positive relationship between age of first reproduction and lifespan would also indicate a trade-off. However, this relationship mixes at least two trade-offs: reproduction and survival, and growth and reproduction.

While the discussion above has focused on some particular bivariate trade-offs widely recognized to be important in life history theory, there are clear cases in which more than two variables or fitness components can become involved in trade-offs. Multivariate trade-offs are certainly possible. When all of the involved variables have been measured, principal components of the genetic correlation matrix may identify main axes of evolutionary change that are prohibited (e.g. simultaneous increases in all fitness components) (Coltman et al., 2005). However, unmeasured variables seriously complicate any analysis of multivariate selection and response (Lande and Arnold, 1983; Roff, 2002, p. 94–97). Despite these concerns, bivariate trade-offs remain a productive initial level at which to explore relationships among fitness components or life history variables.

5.1.4 Trade-Offs and Senescence

Senescence, the decline in organism function often called aging, represents a classic problem for the evolutionary theory of life histories (Charlesworth, 2000). Senescence is particularly intriguing in long-lived taxa, such as primates, and it may be the result of trade-offs between early reproduction and survival. Mid-twentieth century biologists addressed the general problem in relation to adaptation at the level of individual fitness, in seeking to explain this apparently maladaptive, but ubiquitous, phenomenon. Two compatible but very different explanations for senescence have been offered. Both rely on the declining force of selection with age, but identify differing patterns of gene action responsible for senescence (Hamilton, 1966; Baudisch, 2005). The first of these is *mutation accumulation*, which proposes there are mutant genes with detrimental effects expressed only late in life (Medawar, 1952). In contrast, *antagonistic pleiotropy* posits there are genes with differing effects at different ages (Williams, 1957). These pleiotropic genes should have beneficial effects early in life but detrimental effects in old age. This trade-off between early and late life function is fundamental to antagonistic pleiotropy. There need not be any trade-offs among fitness components at different ages for mutation accumulation to be an effective

source of senescence in old age. Experimental evidence from laboratory studies offers some support for both theories (Snoke and Promislow, 2003; Hughes et al., 2002).

Primates are a particularly interesting group in which to study senescence and life history trade-offs because of their potentially long lives. While this makes collecting the required data difficult, consideration discussed in the previous section suggests important trade-offs may be limited or of different kinds in primates (Charnov, 2004; Leigh and Blomquist, 2007). However, trade-offs between reproduction and survival or current and future reproduction may be some of the clearest constraints on primate life histories. Several studies of human populations have identified trade-offs between reproduction and survival, though none seem to be unequivocal. Furthermore, many of the populations examined were undergoing dramatic demographic changes, and only a handful of these studies have dealt with genetic rather than phenotypic patterns (Westendorp and Kirkwood, 1998; Thomas et al., 2000; Lycett et al., 2000; Kirk et al., 2001; Muller et al., 2002; Korpelainen, 2000, 2003). Pettay et al. (2005), for example, identified positive genetic correlations (trade-off indicating) between female age of first reproduction and lifespan and between average interbirth interval and lifespan in rural Finns from the 18th and 19th centuries. Often statistical limitations or the *ad hoc* nature of these historical comparisons limits separating genes from common environment as the cause of familial resemblance. For example, in the study of Westendorp and Kirkwood (1998) the heritability of lifespan based on parent and offspring regression was similar to the correlation between lifespans of parents (who should share nothing but environment). These few studies incorporating quantitative genetic techniques contrast with many others relying on phenotypic patterns alone. In these studies any variety of relationships from those indicating trade-offs (e.g. Korpelainen, 2003, 2000) to those strongly indicating simultaneous improvement of traits can be found (e.g. Muller et al., 2002).

5.1.5 Hypotheses for Cayo Santiago Females

The focus of this chapter is on life history trade-offs in the Cayo Santiago female macaques. Based on the preceding discussion and collecting variables explored in previous chapters, several trade-offs can be explored. These are expected to be found in the phenotypic, and more likely, the genetic correlations between variables.

Fitness Components The fitness components calculated for individual females using van Tienderen's (2000) hierarchical decomposition of selection can be correlated to look for bivariate trade-offs. Specifically, offspring quantity/quality trade-offs would be shown by negative correlations between birth rate and offspring survival rate in the same age class. A reproduction and survival trade-off would manifest itself as a negative

correlation between birth rates of young adults and survival rates of mature adults. Finally, a negative correlation between birth rates of young adults and birth rates of mature adults would indicate a trade-off between current and future reproduction.

Life History Variables Similarly, several other life history variables can be calculated to examine variation not captured in the life cycle model used in Chapter 4. A negative correlation between early and late reproductive output would indicate a trade-off between current and future reproduction. In this case reproductive output is measured for all females who reach maturity, regardless of whether they survive into the defined age classes. Second, the relationship between early fertility and lifespan is predicted to be negative. This identifies one pathway by which reproductive output in early and late life may be traded off—mortality. It would be a trade-off between current reproduction and survival.

5.2 Methods

5.2.1 Fitness Components

Six fitness components were selected from the hierarchical decomposition of selection presented in Section 4.2.2. They were chosen because they represent meaningful aspects of female life history that are subject to selection in wild populations (Altmann and Alberts, 2003b). The six components were the birth rate, offspring survival rate and adult survival rate of the two adult age classes (young: 3–5 years, mature: $\geq$6 years). Birth rates were limited to individuals that reproduced in the age class. This eliminates trivial correlations between birth rates and adult survival that can result from birth rates scored on females who died early in age classes.

A total of 15 bivariate trade-offs ($[6 \times (6-1)]/2$) are possible in this correlation matrix. However, three of these must be excluded because they involve correlations between fitness components across age classes that require young adult survival to be 1 (e.g. young adult survival and mature adult birth rate). The other six cross-age class correlations can be calculated and represent potential trade-offs (e.g. young adult birth rate and mature adult birth rate). The remaining six correlations are within age class (e.g. young adult birth rate and infant survival of young adults).

Bivariate animal models were used to calculate heritabilities and phenotypic, genetic, and residual correlations. Models included two fixed effects for all variables. The fixed effect of social rank category was averaged over the years the female spent in the age class (see Section 4.2.1). Only females with integer values for this fixed effect were used. Rank was included in models because it was clearly demonstrated to affect these fitness components (Ch. 4). Second, birth cohorts were grouped into 7 successive categories called contemporary groups

to control for temporal variation in colony population size, management, and weather. Contemporary groups were defined as 5 year intervals of birth cohorts beginning in 1960. Only animals born between 1960 and 1998 were used in the analysis of the young adult fitness components while birth cohorts between 1960 and 1990 were used for the mature adults. This ensures results are not biased to females who died young. The only random effects in the model were the additive genetic effect of animal identity, and the residual (see Section 2.5). The analysis was limited to a single large interlocking pedigree involving 6543 known individuals, in 17 matrilines connected by paternities. This is 82.43% of the entire demographic database.

Heritabilities, and phenotypic, genetic, and residual correlations were collected from DFREML 3.1 (Meyer, 2000). Two runs were required to obtain variance component estimates in DXMUX, the multivariate animal model program within DFREML. The first uses a simplex search algorithm to obtain starting values for a second run using restricted maximum likelihood (McLeery et al., 2004).[1] The significance of heritabilities and genetic and residual correlations was tested by creating a z-score from their estimated value and standard error. For the correlations these are unique. However, heritabilities from the different bivariate animals differ slightly, and between five and two estimates were available for each fitness component. The mean z-score was used for significance testing in this case. As heritabilities cannot be less than 0, the hypothesis test is 1-sided. Phenotypic correlations were tested by the standard t-statistic for a correlation to obtain 2-sided p-values (Ott and Longnecker, 2001, p. 596).

5.2.2 Life History Variables

To examine reproductive output across the lifespan outside of variation captured in the life cycle model of Chapter 4, I used a set of 273 females from the demographic database who had died on the island, reproduced at least once, and were born in or after the 1960 birth season and could thus be reliably aged for their entire lifespan. Females born after the 1990 birth season were excluded to not bias the results heavily to individuals dying young. All females were members of the same large interlocking pedigree described in Section 5.2.1.

To generate the life history variables I divided the reproductive lifespan into three parts of unequal duration: early (3–5 years old), middle (6–10), and late (11-25). Age was treated as the age of the birth cohort to which the belonged. The number of offspring produced by an individual female while in each interval was tallied from the demographic database. This measure combines fertility and survival. Any female that produced an offspring at any point in her life will be scored for all of these age intervals, whether she was alive in it or not. Dead individuals were scored a 0 for intervals in which they were never alive

[1] This is done to save time. The REML search can be extremely slow when poor starting values are used, but is the best procedure for finding the desired solutions.

(Table 5.4, Figure 5.1). Phenotypic correlations in reproductive output among the intervals were measured with the parametric Pearson's correlation in SAS 9.1 (SAS Institute, 2003).

The relationship between early fertility and lifespan was assessed in a subset of the females described previously in this subsection, all dying at ages greater than or equal to 8 years old (n=198). A slightly larger early fertility age interval (3–7 years), and lifespan—rounded to the nearest year—were used. Limiting the data in this way removes any expectation of a correlation from considerations of survival into each period of life. Exploring the relationship between early fertility and lifespan identifies one of the contributing factors to the correlations among the offspring counts at different ages. The parametric phenotypic correlation was calculated in SAS.

Bivariate animal models were used to calculate genetic correlations between pairs of variables (Section 2.5). No fixed effects other than the population mean were fit. The birth cohort an individual female belonged to was fitted as an additional random effect to account for changes over time in population size, colony management, weather and any other temporally varying factors. The analysis was performed in SOLAR 2.1.4 (Almasy et al., 1997). Likelihood ratio tests within SOLAR indicated the cohort effect should be included in each bivariate model. Likelihood ratio tests were also used for testing the significance of residual (r_E) and genetic (r_A) correlations. No test was performed for the cohort correlations r_C.

5.2.3 Selection Gradients

To estimate the strength of selection on early fertility and lifespan selection gradients were calculated from the multiple regression of fitness, measured as a female's individual λ, on these two variables (Lande and Arnold, 1983). Individual λ was calculated as the dominant eigenvalue of the individual projection matrix following McGraw and Caswell (1996). Individual λ summarizes not only the total number of offspring a female gives birth to, but also the age-schedule by which she does so—applying greater weight to earlier births. SAS was used to calculated individual λs and perform the multiple regression analysis. All three variables were transformed into z-scores prior to running the regression, by subtracting their mean and dividing by their standard deviation. The multivariate response equation of Lande and Arnold (1983) is used to predict the intergenerational change in these variables (Section 2.4). Calculating selection gradients is unnecessary for the fitness components because their elasticities provide an indicator of strength of selection (Table 4.3, see van Tienderen (2000)).

5.3 Results

5.3.1 Fitness Components

All six fitness components have modest heritabilities ranging from 0.262 to 0.487 (Table 5.2). Only the heritability for infant survival of early adults ($proboffsurv2\ h^2 = 0.262$) is outside of the tight range of mean estimates for other components between 0.41 and 0.49. Low heritability of infant survival rate for early adults is not surprising given the ineptitude of many primiparous female primates (Wilson et al., 1978; Altmann et al., 1988; Smuts and Nicolson, 1989; Koyama et al., 1992; Paul and Kuester, 1996; Bercovitch et al., 1998). The only other component that does not have significant heritability is early adult survival ($probsurvival2\ h^2 = 0.487$). This is due to large standard errors on both heritability estimates (SE=0.398 and 0.533). These heritabilities are similar to or larger than estimates reported in Chapter 3 for lifespan and number of offspring, and larger than those for mean interbirth interval and age of first reproduction.

Phenotypic correlations offer little evidence of trade-offs and, moreover, suggest a lack of strong correlations among fitness components (Table 5.3). This is equally true of the phenotypic relationships before and after the effects of social rank and contemporary group are statistically removed. Four of the 12 correlations are negative, but only one reaches statistical significance. Young adult birth rate and young adult infant survival rate are negatively correlated phenotypically ($r_P = -0.103$, $p = 0.023$). This is the only evidence for trade-offs in the phenotypic correlation matrix. The only other significant phenotypic correlations are positive. The strongest correlation in the matrix is between young adult survival and infant survival of young adults ($r_P = 0.295$, $p < 0.001$). The same correlation for mature adults is also significant ($r_P = 0.163$, $p < 0.001$). These are easily understood; if a a mother dies her infant is likely to die with her. None of the trade-offs hypothesized to be important for female macaques are significant. The birth rates are positively correlated with one another ($r_P = 0.049$), as are early birth rate and mature adult survival ($r_P = 0.028$). Both correlations between birth rates and their matched adult survival rates are negative, but not significant (early: $r_P = -0.030$, mature: $r_P = -0.068$).

None of the genetic or residual correlations are significant, either. All have absolute values less than 0.344. Standard errors are larger than the estimated correlation in all but one case—survival of young adults and their infants ($r_E = 0.344$, $SE = 0.243$). This would indicate environments favoring maternal survival also favor infant survival. All of the genetic and residual correlations relevant to trade-offs hypothesized to be important for female macaques match the sign of their phenotypic counterparts reviewed above. In summary, there is no evidence for trade-offs in the genetic correlation matrix.

5.3.2 Life History Variables

Correlations in Reproductive Output among Age Intervals

Phenotypic correlations between reproductive output in the early, middle, and late age intervals provide *no* evidence for trade-offs. All correlations are either significantly positive or cannot be declared significantly different from 0 (Table 5.6). This implies that high reproductive output early in life is compatible with high output later in life. Correlations between adjacent ages are significantly positive. The middle–late output correlation is strongest and highly significant (r_P=0.54215, p <0.001) while that between early and middle is weak but significant (r_P=0.12745, p <0.05). The distant age intervals, early and late, have a very weak correlation that cannot be distinguished from 0 (r_P=0.05575, p >0.05).

Genetic correlations, however, provide a very different set of relationships because of one important case (Table 5.6). The correlation between early and late output is strongly negative (r_A=-1.0000, p <0.05), unlike its phenotypic counterpart which was essentially 0. This provides evidence of a trade-off between reproductive output in early and late phases of life. Any increase in one will be counteracted by decrease in the other. In contrast to this one negative correlation, both correlations between adjacent age intervals are either strongly positive, as in middle and late output (r_A=0.89330, p <0.05), or positive but not significantly different from 0 for early and middle output (r_A=0.08387, p >0.05). These genetic correlations approximate their phenotypic counterparts and indicate there are no trade-offs in reproductive output between these phases of life—increased output in one phase will be accompanied by increases in another. Estimated heritabilities and cohort effects are all small (h^2 0.097–0.329, c^2 <0.001–0.055), and heritabilities are greater than cohort effects in all cases (Table 5.5).

The phenotypic correlation between reproductive output in each phase of adult life is further decomposed into a common effect of birth cohort and residual environmental effects (Table 5.6). The birth cohort correlations (r_C) have a mixture of signs making them difficult to interpret, particularly without significance tests. Early and late output appear to be unrelated, while early and middle are positively related, and middle and late are negatively related. This suggests that temporal variation among birth cohorts affects output in different phases of adult life in differing ways. However, because the cohort effects are small for all variables these correlations will be of little consequence.

The residual environmental correlations are consistently positive and small. The residual correlation between early and late output is significant (r_E=0.32738, p <0.01) as is that between middle and late output (r_E=0.43888, p <0.01). Early and middle output have the weakest residual correlation (r_E=0.13013, p >0.05). Uniformly, these residual environmental correlations indicate environmental effects that increase or reduce reproductive output in one phase of

life will have an effect similar in sign on reproductive output other phases of life. Because the heritabilities and cohort effects are small for these variables the phenotypic relationships among them are dominated by these environmental correlations (Roff, 1997).

Early Fertility & Lifespan Correlation

The phenotypic correlation between early fertility and lifespan fails to reach significance and is very close to 0 (r_P =0.03535, p >0.05, Table 5.6). This implies there is no penalty, in terms of reduced lifespan, from high fertility early in adult life. However, the genetic correlation is strongly negative and statistically significant (r_A=-1.0000, p <0.05). This indicates a potent trade-off between how long individuals live and the number of offspring they have as young adults, even when controlling for mortality differences early in life. The residual correlation between these traits is positive and significantly different from zero (r_E=0.34756, p <0.05). The common environmental effect of birth cohort is weakly negative for these traits also (r_C=-0.15405). The residual correlation indicates remaining covariance between the early fertility and lifespan is positive; good environments for early fertility are the same environments for later lifespan. Though difficult to interpret without a test of significance, the cohort correlation indicates the opposite; a good birth year for early fertility may be a bad year for later lifespan.

Selection Gradients

Early fertility and lifespan are strong predictors of total lifetime fitness (λ). The selection gradient on early fertility is 0.639, while that for later lifespan is 0.531 (Table 5.7). Both of these indicate that a single standard deviation increase or decrease in one of these traits, while the other is held constant, will result in about a $\frac{1}{2}$ to $\frac{2}{3}$ standard deviation change in fitness in the same direction.

The inter-generational response for each of these variables is strongly constrained by their genetic correlation, estimated to be -1. Using the response equation (Equation 2.3) for the two traits predicts a 0.108 increase in early fertility and a 0.108 decrease in later lifespan. These results are expressed in phenotypic standard deviations from the phenotypic mean. Converting them back to the proper units they predict a 0.090 offspring increase in the mean of early fertility from 3.157 to 3.247, and a 0.542 year decrease in later lifespan from 14.540 to 13.998. Considerations of statistical power imply changes of this magnitude are impossible to detect without extraordinarily large samples. For example, to detect either of these differences between two generations, with a Type I error rate of $\alpha = 0.05$ and Type II error rate of $\beta \leq 0.10$, requires about 1800 females in each generation (Ott and Longnecker, 2001, p. 315).

5.4 Discussion

5.4.1 Difficulties in Identifying Trade-offs

This study documented relatively few trade-offs in female macaques from Cayo Santiago. Life history trade-offs are notoriously difficult to identify (Roff and Fairbairn, 2007; Reznick, 1985). This can result from a number of factors including positive environmental covariation between traits thought to be traded off, greater variance in acquisition than allocation, poor identification of critical trade-offs, trade-offs involving more than two traits, and gene×environment interactions (Roff, 2002; Stearns, 1989; van Noordwijk and de Jong, 1986).

Some of these difficulties were circumvented in this study by examining genetic correlations, and removing measurable environmental influences such as social rank. However, even with genetic correlations under controlled laboratory conditions trade-offs remain elusive. Roff (2002, p. 144–147) presents several genetic correlation matrices from large laboratory insect studies in which there were no genetic correlations indicating trade-offs. As in this analysis, genetic correlations generally had very large standard errors that prohibit the identification of trade-offs. Although genetic analysis clearly has advantages over reliance phenotypic correlations, it has limitations of its own in the expansive standard errors on the estimated correlations (Roff, 1997).

In this study, results with the fitness components generally did not identify any important trade-offs. Two of the four trade-offs hypothesized to be important for female macaques have negative correlations in all three correlation matrices. These are the pairs of birth rate and adult survival rate in the same age classes. These are extremely weak and non-significant correlations, and offer only suggestive evidence that female macaques face allocation decisions between concurrent reproduction and survival that result in negative relationships between these fitness components. The lack of a negative correlation between early adult birth rate and mature adult survival is likely due to the short time difference between these periods. In studies of *Drosophila*, fertility in adjacent age classes are often positively correlated, but more distant age classes and early fertility and lifespan often have negative correlations (Rose and Charlesworth, 1981).

Within the fitness components, the only significant phenotypic evidence for a trade-off is between early adult birthrate and infant survival rate of early adults. This is matched by negative genetic and residual correlations. However it is dominated by the residual contribution. Environmental factors, beyond matriline social rank, must mediate this apparent trade-off. Although they do not represent microevolutionary constraints, phenotypic correlations still affect response to selection if both traits are under selection, as these fitness components surely are (Roff, 2002, p. 98). Primiparous mothers often suffer higher infant mortality than multiparae either due to inexperience or neuroendocrine

mechanisms (Bercovitch et al., 1998). Some aspects of mothering style are also rank-related in female macaques at Cayo Santiago (Berman, 1983, 1988). However, the phenotypic relationship identified here between early adult birth rate and infant survival is primarily due to environmental effects independent of social rank.

5.4.2 Implication of Detected Life History Trade-offs

The scarcity of trade-offs among fitness components is contrasted by an important trade-off between current and future reproduction documented in the life history variables. This trade-off has far-reaching implications for the study of primate life histories and the evolution of senescence.

First, the presence of a negative genetic correlation between early and late life reproductive output, in part explained by the negative genetic correlation between early fertility and lifespan, emphasizes that there are important genetically mediated constraints on the reproductive decision-making of female primates. Heavy investment in offspring production early in life bears a cost of reduced lifespan for female macaques. This cost can be quantified using the estimated variances and covariances of early fertility and lifespan (Section 2.5.4). After rearranging the following equation for the phenotypic correlation where the subscripts x and y represent two traits such as early fertility and lifespan

$$r_P = r_A\sqrt{h_x^2 h_y^2} + r_C\sqrt{c_x c_y} + r_E\sqrt{(1-h_x^2-c_x)(1-h_y^2-c_y)} \tag{5.1}$$

the phenotypic regression (β_P) of trait y on x is given by

$$\beta_P = \frac{\sigma_P(x,y)}{\sigma_P^2(x)} = \frac{\sigma_A(x,y)+\sigma_C(x,y)+\sigma_E(x,y)}{\sigma_P^2(x)} = \beta_A + \beta_C + \beta_E \tag{5.2}$$

The βs represent the slope of the phenotypic regression (β_P), and its constituent additive genetic (β_A), cohort (β_C) and residual (β_E) regressions. Treating early fertility as x and later lifespan as y these βs are 0.259, -1.283, -0.020, and 1.563, respectively. There is a very weak positive relationship between these variables phenotypically—for one offspring added between ages 3 and 7 a female should gain 0.259 years of later lifespan. However, the genetic regression predicts a 1.283 year *loss* in later lifespan for each offspring added; and the residual regression predicts a 1.563 year *gain*. These two underlying regressions largely negate one another and cause there to be little discernible phenotypic relationship (Figure 5.2).

This strong negative genetic regression emphasizes the presence of genetic structure for a trade-off. However, environmental effects counteract this trade-off because females who produce more offspring while young tend to be in beneficial environments that also promote longer lifespan. Despite there being combinations of these fitness components–early fertility and later lifespan–that yield high fitness, there will be very little selective response between generations for a

change in the population averages for these traits. This is because the combinations yielding highest fitness (high early fertility and long lifespan) and lowest fitness (low early fertility and short lifespan) are environmentally induced and cannot be transmitted genetically from parent to offspring. Social rank was not included in models for these life history variables, because it could not be measured on enough females and had little relationship with the variables when it could be. Rank may work to elevate values for both traits in higher ranking females, causing positive environmental covariation.

Predicted responses to selection for early fertility and lifespan further emphasize the strength of the trade-off between these variables. Despite both traits being under strong selection little inter-generational response is predicted. This is due to their strongly negative genetic correlation and approximately equal selection gradients. Because early fertility is estimated to be under slightly stronger selection it is predicted to increase, and, despite being under strong positive directional selection, later lifespan should decrease. However, neither trait's predicted response would be detectable in the Cayo Santiago population. This is important because many life history traits show very little change across generations despite well-documented variation within populations (see Ch. 3 and Bercovitch and Strum, 1993; Bercovitch and Berard, 1993; Pusey et al., 1997). It is quite possible that these traits are constrained by trade-offs such that under normal circumstances they do not evolve (i.e. change between generations).

The second important feature of the documented trade-offs is that they support the antagonistic pleiotropy model for the evolution of senescence (Williams, 1957). However, they also point to other kinds of gene action in the patterning of age-specific reproductive output. Negative genetic correlations between early and late life function are the essential prediction of antagonistic pleiotropy and this is confirmed in the present study. These results are similar to those of Rose and Charlesworth (1981) studying *Drosophila melanogaster*. They found negative genetic correlations in fertility between age intervals, and between early fertility and lifespan. In contrast, the positive genetic correlations between adjacent ages reported here cannot be accommodated by antagonistic pleiotropy. The pattern of decreasing correlation with increasing distance between age intervals may indicate age-specific gene action that overlaps adjacent but not more distant phases of life. The strongly positive genetic correlation between middle and late output suggests commonality between these two phases. This is paralleled in the fitness components by positive correlations between young adult birth rate and mature adult survival. If there are many late-acting deleterious mutations, as predicted under mutation accumulation, this pattern could arise. Hughes et al. (2002) and Engstrom et al. (1992) also found decreasing genetic correlations with increasing distance between ages in *Drosophila* age-specific reproductive success, but all correlations remained positive in both studies. Other unique predictions of mutation accumulation, such as increasing

ratio of dominance to additive genetic variance with age, were not dealt with here (Charlesworth and Hughes, 1996). The similarity between these studies of *Drosophila*, which live only a few months, and rhesus macaques, which can live over 30 years, is remarkable, and underscores the breadth of the evolutionary theory of senescence and life history theory in general (Vaupel et al., 2004).

The frequency of antagonistic pleiotropy has been challenged on strictly theoretical grounds (Curtsinger et al., 1994; Hendrick, 1999), though much of these arguments focuses on antagonistic pleiotropy as a mechanism for maintaining genetic polymorphism, and not on the presence of trade-offs or involvement of negative genetic correlations in senescence. Spatial and temporal heterogeneity and mutation-selection balance can also maintain genetic variation in trade-offs (Roff and Fairbairn, 2007; Hendrick, 1999; Roff, 2002, p. 107). However, the strong negative correlations reported here are both -1, which is consistent with low levels of polymorphism.

5.4.3 Contrasts between Data Sets

The results of this study with fitness components were generally negative—few trade-offs of any strength were documented. Clear trade-offs were identified in the genetic correlations among the life history variables. There are several factors that may contribute these differences. First, the life cycle model used for calculating fitness components may not capture the trade-offs documented in the life history variables between early fertility and later lifespan or early and late reproductive output. The mature adult age class in the life cycle model includes individuals 6 years and older. While it is conceivable that there would be trade-offs between young and mature adult birth rates or birth rates and survival rates between the adult age classes, there would have to be many females dying or suffering reduced fertility early on in this mature adult age class to detect a trade-off. The reproductive output intervals and lifespan begin counting females at later ages. Late reproductive output begins at age 11, and lifespan begins at age 8. It may well be that the costs of early reproduction only manifest themselves at these more advanced ages. The large mature adult age class pools younger ages, where current and future reproduction trade-offs may not yet be strong, and later ages, where they are, resulting in no detectable relationship. The weak positive genetic correlation between early and middle reproductive output is consistent with this interpretation. While the life cycle model used in Chapter 4 was suitable for analysis of social rank-life history relationships, it may not be appropriate for examining life history trade-offs in female macaques. Divisions of the life cycle in constructing the life history variables are valid for inference to natural macaque populations. The Cayo Santiago females are not unusual for having many females who reach advanced ages (≥ 8 or ≥ 11). Information on lifespans of provisioned and non-provisioned Japanese macaques suggests that enough females reach these ages that the identified trade-offs can

operate in wild macaque populations (Takahata et al., 1995, 1998).

Other contributing factors to the difference between these data sets are less easily interpreted. The role of social rank and its statistical control were different. With the fitness components rank was known to affect all of them and using it as a fixed effect to remove its variation was justified. No such procedure was undertaken with the life history variables because rank did not have a significant predictive relationship with any of them. Rank was also only available for a subset of females with completed life histories. It is possible that in removing the effect of rank prior to the genetic analysis of the fitness components collinear genetic effects were also removed. Additionally, these are different samples of females from the Cayo Santiago population. Sample sizes for the fitness components are much larger because they can include females who were removed by colony management. The life history variables can only be calculated for naturally dying females. Some differences in these analyses are to be expected from this alone. Finally, the fitness components are not well distributed for a genetic analysis or calculating linear correlations. How this might affect the results is unclear, but the life history variables are not ideally distributed either (Figure 5.1). This seems to be a problem common to both data sets and is unlikely to explain the differences in results.

5.4.4 Merits of Phenotypic and Genetic Analyses

The contrast between genetic and phenotypic patterns of covariation in the life history variables is an important result of this study. Phenotypic measurements are often used as surrogates for genetic values in searching for life history trade-offs or modeling the short and long-term traits to selection (e.g. Hill and Hurtado, 1996; Strassmann and Gillespie, 2002; Muller et al., 2002; Borgerhoff-Mulder, 2000; Dobson et al., 1999; McElligott and Hayden, 2000; Oli et al., 2002). The practical reasoning behind this is understandable—phenotypic correlations are much easier to estimate with much greater power (Cheverud, 1988; Roff, 1997). Behavioral field data are also interpreted as representative of genetic covariance patterns or traits are treated as having 0 genetic covariance and are thus freely malleable for selection, whether this assumption is stated or not (Lawler et al., 2005; Bercovitch and Berard, 1993). This can be problematic.

Roff (1996) reviewed about 1800 published phenotypic and genetic correlations in plant and animal species. He concluded that the phenotypic correlation could not be taken as a reliable indicator of the genetic correlation for life history and behavioral traits, though they may serve adequately for morphological traits (see also Cheverud, 1988). The results presented here corroborate Roff's interpretation. The trade-offs identified with genetic correlations are not apparent in a strictly phenotypic analysis. Relying only on phenotypic relationships leads to the erroneous conclusion that female macaques face no penalty for having more offspring early in life.

While genetic analyses provide a clear interface between the processes of inheritance and selection, and observable patterns in primate lives (Bernstein, 2004), they come at a price. Very few populations can currently be examined in this way because long-term demographic records are required on individuals of known genealogical relationship (Strier et al., 2006; Kruuk, 2004). Furthermore, the large standard errors that typically accompany genetic correlations make their interpretation difficult (Roff, 1997). Phenotypic analyses can be productive in identifying trade-offs when both traits are known to be under selection. However, they only represent microevolutionary constraints to the extent that phenotypic correlations approximate genetic patterns ($\mathbf{P} \approx \mathbf{G}$). When manipulative experiments can be performed on animals of homogeneous genetic background, the phenotypic relationships among traits can be particularly useful ways to index trade-offs (Bell and Kofoupanou, 1986). The theoretical considerations of environmental covariation and acquisition-allocation hierarchies, along with the empirical contrasts between genetic and phenotypic correlations, argue against a completely phenotypic analysis of life history variation, especially when attempting to document trade-offs. Opportunities for manipulative experiments with primates are extremely rare. Methodological plurality may be the best route to a detailed understanding of life history trade-offs in primates (Stearns, 1989). Currently, genetic correlations are a major gap in our knowledge of primate trade-offs but offer a clear method for their identification.

5.5 Summary

Recognizing the critical trade-offs in an animal's life history and constructing the proper indices among its component parts is a major obstacle to detecting life history trade-offs. In this study, trade-offs between current and future reproduction were hypothesized to be important for female macaques from comparative considerations of primate versus other vertebrate life histories. These speculations were supported, but only in the specific context of two related trade-offs between early fertility and lifespan and early and late reproductive output. These trade-offs were apparent only in the genetic correlations between these life history variables because environmental covariation masked the trade-offs, making their phenotypic relationships positive or ≈ 0. The combination of nearly equivalent selection on early fertility and lifespan and a strong negative genetic correlation between them explains why these traits do not change between generations. Increase in one will be offset by decrease in the other. Highest fitness is achieved at intermediate values of both traits. Because of this, aging in female macaques can be explained, at least in part, by antagonistic pleiotropy between early fertility and longevity.

5.6 Tables and Figures

Table 5.1: Trade-offs in female macaque life history. Fitness components are defined in Chapter 4. Life history variables are defined in Section 5.2 and Chapter 3. Not all of the hypothesized trade-offs among life history variables are explored. More complex relationships between variables are possible (Ch. 3 Roff, 2002), though the focus of this analysis is on bivariate trade-offs.

general trade-off	variable pair	correlation sign for trade-off
Fitness components		
offspring quality v. quantity	birth rate–offspring survival rate[1]	–
reproduction & survival	young birth rate–mature survival	–
current & future reproduction	young birth rate–mature birth rate	–
Life history variables		
current & future reproduction[2]	early–late reproductive output	–
reproduction & survival	early fertility–lifespan	–
reproduction & survival	age first rep.–lifespan	+
reproduction & survival	mean IBI–lifespan	+

[1] For fitness components in the same age class (young-young and mature-mature).
[2] Because the reproductive output measure is blind to death this is the most appropriate categorization. However, much of the relationship could be due to death at younger ages by individuals investing heavily in early reproduction.

Table 5.2: Heritabilities of selected fitness components in the hierarchical decomposition of selection. The terminal digit on the fitness component names indicate the age class they apply to. Young adulthood is age class 2, and mature adulthood is age class 3. Multiple heritability estimates are provided from each of the bivariate models; the number of these is indicated in column n. The number of individual females in each bivariate model are given in Table 5.3. The mean, minimum, and maximum heritability are shown along with the mean z-score from the ratio of h^2 to its standard error for significance testing.

fitness component	n	h^2			mean z	
		mean	min.	max.		
birthrate2r	5	0.41400	0.4047	0.4333	2.39165	**
proboffsurv2	5	0.26234	0.2395	0.2857	1.51229	
probsurvival2	2	0.48735	0.4741	0.5006	1.07325	
birthrate3r	4	0.42720	0.4064	0.4638	2.05567	*
proboffsurv3	4	0.43540	0.3819	0.4664	2.11610	*
probsurvival3	4	0.46625	0.4486	0.4900	2.00704	*

* $p < .05$, ** $p < .01$, *** $p < .001$

Figure 5.1: Frequency distribution of reproductive output, early fertility, and lifespan for the specified age intervals. In the top row (reading from left to right) are reproductive outputs (offspring counts) for early (3–5 years old), middle (6–10), and late ($\geq$11) life phases. The bottom row shows the distribution of fertility for females surviving 8 or more years while they were 3–7 years old, and their corresponding lifespans.

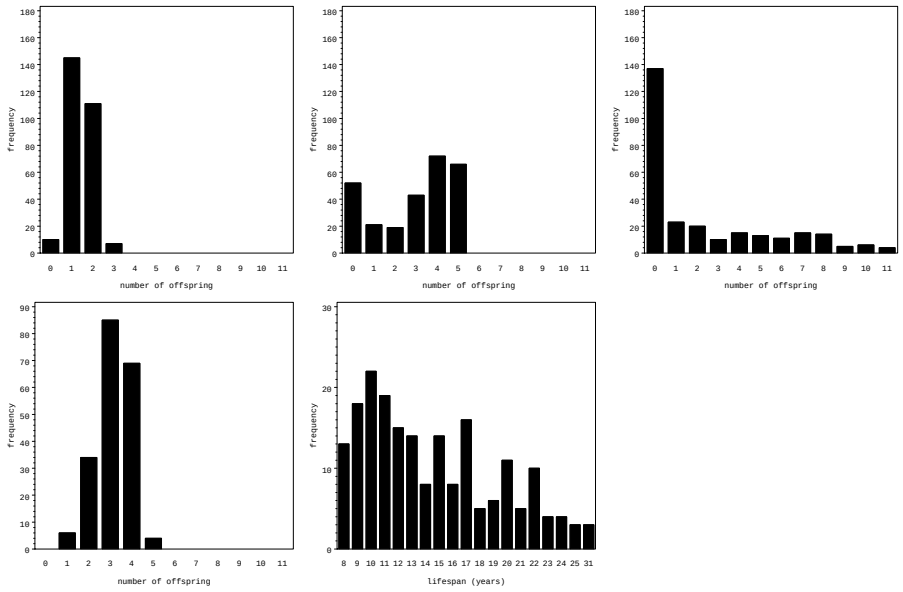


Table 5.3: Phenotypic (r_P), genetic (r_A) and residual (r_E) correlations among fitness components in the hierarchical decomposition of selection. The terminal digit on the fitness component names indicate the age class they apply to. Young adulthood is age class 2, and mature adulthood is age class 3. Correlations are given is the upper triangle of each matrix. The lower triangle of top matrix provides the number of females used in computing each correlation. In the last two matrices standard errors of the correlations are provided in the lower triangle. Correlations omitted with a "." cannot be computed because a female must survive through young adulthood to record a value in mature adulthood. All values for these females on probsurvival2 are 1, and no correlation can be calculated for fitness components in age class 3. DFREML also failed to return a standard error on the correlation between probsurvival2 and birthrate2r. Standard errors are larger than the estimated correlations in all but one case. None of the genetic or residual correlations are significantly different from 0.

Raw phenotypic correlations (r_P) and sample sizes

	birthrate2r	proboffsurv2	probsurvival2	birthrate3r	proboffsurv3	probsurvival3
birthrate2r		**-0.1020**	-0.0607	0.0229	0.0249	0.0031
proboffsurv2	484		**0.3910**	0.0703	-0.0135	0.0690
probsurvival2	860	484		.	.	.
birthrate3r	490	293	.		0.0794	-0.0625
proboffsurv3	395	240	.	425		**0.2201**
probsurvival3	478	288	.	499	425	

Phenotypic correlations (r_P) controlled by social rank and contemporary group

	birthrate2r	proboffsurv2	probsurvival2	birthrate3r	proboffsurv3	probsurvival3
birthrate2r		**-0.1034**	-0.0301	0.0493	0.0451	0.0275
proboffsurv2			**0.2953**	0.1054	-0.0129	0.0417
probsurvival2				.	.	.
birthrate3r					0.0731	-0.0677
proboffsurv3						**0.1630**
probsurvival3						

Genetic correlations (r_A) and standard errors

	birthrate2r	proboffsurv2	probsurvival2	birthrate3r	proboffsurv3	probsurvival3
birthrate2r		-0.0417	-0.0064	0.0991	-0.0329	0.0304
proboffsurv2	0.4099		0.2302	-0.0252	-0.0971	0.0130
probsurvival2	.	0.5626		.	.	.
birthrate3r	0.3714	0.4839	.		-0.0118	-0.0569
proboffsurv3	0.3835	0.5899	.	0.3647		0.1949
probsurvival3	0.3266	0.4800	.	0.3689	0.4374	

Residual correlations (r_E) and standard errors

	birthrate2r	proboffsurv2	probsurvival2	birthrate3r	proboffsurv3	probsurvival3
birthrate2r		-0.1346	-0.0499	0.0147	0.1040	0.0252
proboffsurv2	0.1429		0.3443	0.1834	0.0274	0.0591
probsurvival2	.	0.2434		.	.	.
birthrate3r	0.1867	0.2160	.		0.1414	-0.0764
proboffsurv3	0.2186	0.2192	.	0.2211		0.1339
probsurvival3	0.1898	0.2154	.	0.2167	0.3061	

correlation $p < .05$ in **bold**

Table 5.4: Descriptive statistics of reproductive output, early fertility, and lifespan.

variable	n	mean	std	min	max
3–5	273	1.4212	0.6080	0	3
6–10	273	2.9524	1.8255	0	5
≥11	273	2.3810	3.1660	0	11
3–7	198	3.1566	0.8374	1	5
life ≥8	198	14.5404	5.0153	8	31

Table 5.5: Heritabilities (h^2) and cohort effects (c) for reproductive output, early fertility, and lifespan from bivariate models. Two estimates are available of each for the reproductive output intervals; only a single estimate is given for early fertility and lifespan.

variable pair	variable	$h^2 \pm SE$	$c \pm SE$
3–5 & 6–10	3–5	0.09741±0.10488	0.05497±0.04192
	6–10	0.19584±0.17739	0.00216±0.00885
3–5 & ≥11	3–5	0.10509±0.07489	0.03612±0.03659
	≥11	0.30743±0.11220	0.02991±0.03501
6–10 & ≥11	6–10	0.15864±0.15814	0.00073±0.00549
	≥11	0.32910±0.12282	0.02781±0.03114
3–7 & life ≥8	3–7	0.17414±0.11883	0.05018±0.04555
	life ≥8	0.26355±0.13330	0.00982±0.04237

Table 5.6: Correlations among reproductive output intervals, and early fertility and lifespan. Phenotypic (r_P), additive genetic (r_A), cohort (r_C) and residual (r_E) correlations are given. Significant correlations are indicated with stars. No significance tests are provided for the cohort correlations.

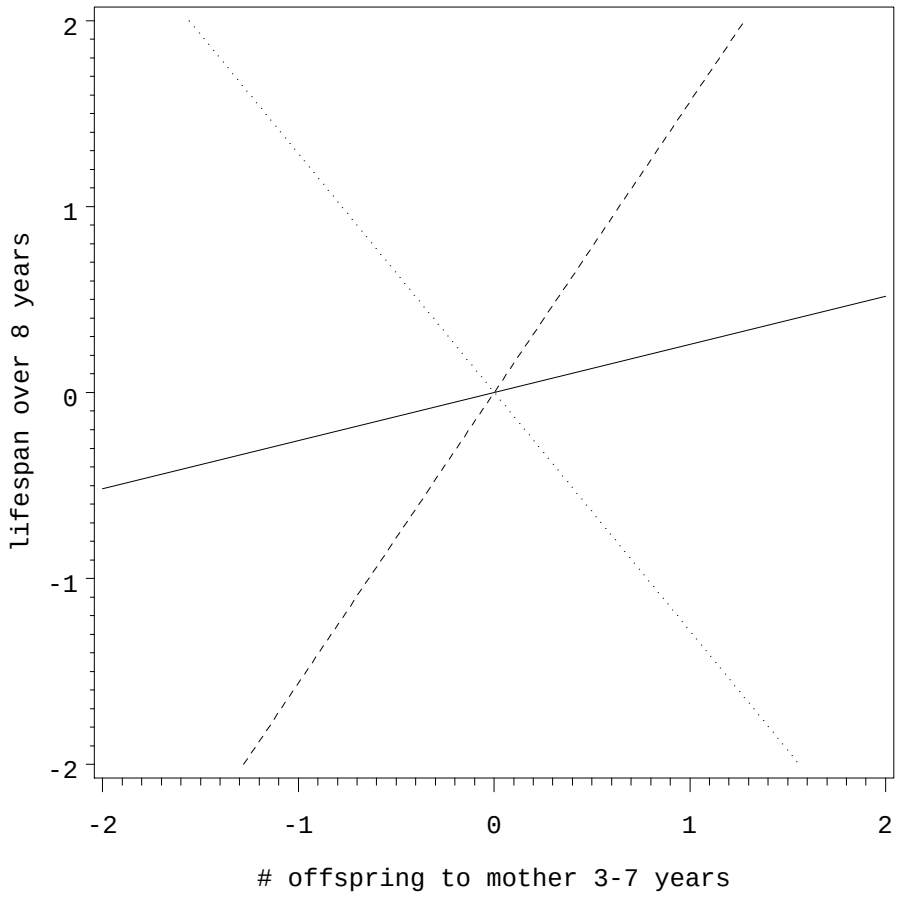
variable pair	r_P		r_A		r_C	r_E	
3–5 & 6–10	0.12745	*	0.08387		1.00000	0.13013	
3–5 & ≥11	0.05575		-1.00000	*	-0.05221	0.32738	**
6–10 & ≥11	0.52415	***	0.89330	*	-1.00000	0.43888	**
3–7 & life ≥8	0.03535		-1.00000	*	-0.15405	0.34756	*

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 5.7: Selection gradients (β) from the multiple regression of fitness, measured as individual λ, on early fertility (3–7 years) and lifespan past 8 years. The full model was highly significant ($F_{2,196} = 244.41$, $p < .0001$; $R^2 = 0.714$).

independent variable	$\beta \pm SE$	t	p
3–7	0.63870±0.03833	16.66	<.0001
life ≥8	0.53092±0.03833	13.85	<.0001

Figure 5.2: Regression lines predicted for early fertility (the number of offspring a female has between ages 3 and 7) and later lifespan (age at death of females living 8 years or more). Axes are centered at the mean for each variable and indicate the addition or subtraction of one offspring or year. The solid line is the phenotypic regression ($\beta = 0.259$), the dotted line is the genetic regression ($\beta = -1.283$), and the dashed line is the residual environmental regression ($\beta = 1.5628$).



Chapter 6

Conclusion

6.1 Insights on Primate Life Histories

There are many levels at which evolution can be studied (Williams, 1992). Investigations of primate life history and sociality have been largely interspecific and comparative. They are typically targeted at identifying broad presumably adaptive trends among radiations of living taxa, by examining interspecific differences in species average values (e.g. Ross and Jones, 1999; Harvey et al., 1987). Details of the evolutionary processes and genetic substrates they are acting upon in populations are unspecified. While the comparative method is of great utility in identifying patterns of association, it is notoriously deficient for testing and refining hypotheses about causal mechanisms (Harvey and Pagel, 1991).

In Chapter 1 I argued that gaps in current understanding of primate life histories and sociality had not been and could not be addressed through the comparative methods typically employed by most biological anthropologists (Altmann and Alberts, 2003a,b). The approach in this thesis has been to focus on the population as the level of analysis. The concept of the Mendelian population, as the site of diachronic evolutionary transformations or "unit of evolution," was a critical development in the formulation of the modern evolutionary synthesis (Dobzhansky, 1950, 1955). By shifting the focus from variation between species average values to variation within populations, insights are available on primate life history and sociality.

First, this study demonstrates a genetic basis for a number of life history and morphological traits in a single primate population. A significant portion of the phenotypic variation seen in primate life histories can be attributed to the additive action of many genes. Having estimates for multiple traits allows for a number of interesting comparisons. The amount of genetic variation varies substantially among traits. This makes a blanket assumption of sizable heritability for any given trait unwarranted. Life history traits tend to have small heritabilities compared to the morphological traits. This patterning of genetic and residual variation among traits conforms to a set of theoretical predictions that emphasizes the developmental and physiological interdependencies among traits—the incorporation of residual variance model of Price and Schluter

(1991). The classical argument that traits more closely association with fitness should have lower genetic variance, because of depletion by stronger selection, was unsupported (Fisher, 1930; Robertson, 1966). This is weak evidence against this model because data for Cayo Santiago females are heavily censored and an equilibrium population is required for the model to apply. In general, heritabilities and the patterning of genetic and residual variation in the Cayo Santiago females and the few other primates for which estimates of any life history variables are available are no different from other mammals and birds (Merilä and Sheldon, 1999, 2000; Kruuk et al., 2000; McLeery et al., 2004). This is promising because it suggests the theoretical work and empirical investigations carried out regarding these taxa are also applicable to humans and non-human primates.

Second, matriline social rank is an environmental variable with pervasive beneficial effects for high ranking females in their life histories at Cayo Santiago (see Section 1.5 and Chapter 4). This study confirms earlier work noting that high ranking females mature earlier (Sade et al., 1976; Sade, 1990; Bercovitch and Berard, 1993), thereby achieving higher birth rates as young adults. However, unique results of this study point to much broader effects throughout females' lives. These include higher ranking females having greater infant and juvenile survival rates. Most importantly, though, they also have higher survival rates as mature adults. This is the fitness component with the greatest impact on fitness according to the life cycle model employed. Rank affects female fitness most by altering female adult survival rates. The strong sensitivity of fitness to adult survival is a general feature of long-lived animals with low reproductive rates including primates (Blomquist et al., 2007; Heppell et al., 2000; Bercovitch and Berard, 1993; Clutton-Brock, 1988). These life history differences mean that the differently ranked segments of the Cayo Santiago population have grown at different rates, with higher ranked matrilines growing at the highest rates. This is true for the entire span of demographic records and during a decade-long period when the population was undisturbed by removals. Rank has dramatic effects on female life history at Cayo Santiago.

The environmental nature of the life history differences among rank levels is confirmed by the analysis of breeding values for several female life history traits. This emphasizes that these rank-related differences are *not* due to genetic differences in life history traits among rank categories. Rank and genes do not appear to covary in any detectable way for these traits. This implies that traditional analyses of rank simply as an environmental effect causing phenotypic variation among individuals in fitness or other life history traits are valid (e.g. van Noordwijk and van Schaik, 1999; Packer et al., 1995). However, caution must be exercised in accepting this result because of limited pedigree connectedness among rank categories (Postma, 2006). If there were any real genetic differences in life history among rank categories, they would have to have been very large to be detected.

Some theoretical considerations on how rank contributes to variance in fit-

ness and traits associated with it suggests that geneticists ought to pay close attention to the social behaviors of primates. Strong hierarchies that concentrate reproduction in a few successful individuals can alter selection dynamics by either slowing or speeding up the rate of evolution of traits affected by the environment provided by rank (Chapter 4). Furthermore, strong hierarchies can depress effective population sizes, a major concern for conservation of primate populations and understanding patterns of drift and inbreeding (Ralls and Ballou, 1982; Nunney, 1993; Strier, 2007). Hierarchies of rank can affect adaptive and neutral evolution.

Finally, life history trade-offs do exist for female primates (Chapter 5). They can be found empirically in the genetic structures of female primate life histories. They are not simply epiphenomena of models requiring demographic viability (Harvey and Purvis, 1999). A major trade-off was found for Cayo Santiago females in the number of offspring they have as young adults and the length of their subsequent life. They face an early fertility–lifespan trade-off that is mediated by a strong negative genetic correlation between these two variables. This is a unique demonstration of antagonistic pleiotropy that contributes to aging in non-human primates (Williams, 1957). Genes that favor early fertility in female macaques also tend to reduce their lifespan, or, conversely, genes that reduce early fertility should extend lifespan. This trade-off is a microevolutionary constraint on the evolution of these fitness components (Stearns, 1989, 1992). Despite both being under strong selection to for increased values, neither will show much inter-generational change because of the strong negative relationship between them.

Life history trade-offs, despite their theoretical importance, have been empirically elusive (Roff, 2002). Two factors contributed to the paucity of trade-offs identified in this study. First, environmental variation can mask the genetic relationship between variables (see Section 6.2). Second, the related variables must capture important trade-offs for the animals being studied. Fitness components from the life cycle model explored in Chapters 4 and 5 did not capture any relevant trade-offs. If the key life history trade-offs for female macaques are between fertility and lifespan, it is likely that the adult lifespans measured in the life cycle model did not identify the "extreme" individuals for which trade-offs would have been noticeable. Regardless of whether this explanation is correct, careful consideration of the life history of the organism being studied must be undertaken to properly identify and measure potential trade-offs. A naïve exploration of correlations among fitness components should not be expected to uncover trade-offs.

6.2 Social "Inheritance" Circumvents Life History Trade-Offs

As discussed in Chapters 2 and 4, the social rank of a female rhesus macaque is predictable by the ranks of close maternal kin, particularly her mother. The genealogical character to social dominance creates a system of inheritance that parallels the genetic (Thierry, 2007; Missakian, 1972). Rhesus macaque females have social mechanisms by which they transmit status from one generation to the next. This social "inheritance" makes it possible for high ranking females to circumvent life history trade-offs by controlling environmental variation in resource scarcity or stress that they and their offspring experience. It is likely that high ranking females attain high fertility and long lifespan despite the strong negative genetic correlation between these variables. This is accomplished through strong positive environmental covariance. High ranking females do not experience resource scarcity or stress to the same degree as lower ranking females (Sapolsky, 2005; Bercovitch and Berard, 1993).

This likely makes the demography of high ranking rhesus macaques less tightly linked to local ecological conditions. Periods of resource scarcity will differentially affect group members. High ranking females will suffer less than low-ranked. They are able to circumvent trade-offs because they do not encounter the physiological limitations set by poor environments. It is possible that strong hierarchies of social rank in macaque groups allow them to persist in more marginal habitats with ephemeral resources. In periods or regions of resource scarcity high ranking females might maintain adequate reproductive output such that this segment of the population may grow or remain stable while lower-ranked segments declined (Dittus, 1977, 1979, 1987).

Climatological cycles over the last 2.5 million years have caused dramatic fluctuations in terrestrial habitats available for macaques in East Asia (Abegg and Thierry, 2002). This period corresponds to the divergence of rhesus macaques and their closest relatives (*M. fuscata* and *M. cyclopis*) from other macaque species. The population history for proto-rhesus macaques then was likely one of repeated geologically rapid interglacial range expansions into forest and forest margin environments uninhabited by other macaque species and shrinkage into more localized glacial refugia (Jablonski et al., 2000; Jablonski, 1993; Han et al., 1997). Population structure during periods of expansion may have been particularly important in this process in promoting relative isolation of related and/or inbred sets of females (Section 4.1.2).

The circumvention of trade-offs by high-ranking female macaques may have some loose parallels in human populations with or without the complications of ascribed status. Living groups of human foragers are relatively egalitarian. They lack systems of ascribed status or caches of wealth, power, and prestige that can be transfered along genealogical lines or any other manner. The personal achievements of individuals are far more important in determining relative

status and resources available to an individual are those they harvest from the environment themselves or which are distributed from other group members. Kin ties are important in distributing food and other resources in human foragers, but there are also cultural norms that ensure equality among family or other units within groups (Gurven, 2004; Hill and Hurtado, 1996; Howell, 1982). With limited mechanisms of food storage and often placing a premium on mobility, human foragers cannot amass material wealth. The redistribution of surplus food is, for the distributor, an exchange of food into social "insurance," which may be required to get them through a critical life period in the future (Hill et al., 2007; Hewlett et al., 2000; Weissner, 1996; Cashdan, 1985).

Human foragers are capable of rapid demographic increase, but over the long run their populations are thought to remain relatively stable due to crashes on localized or regional scales (Hill and Hurtado, 1996; Boone, 2002). Resource redistribution in human foragers suggest ways that life history trade-offs can be mitigated. For example, cultural norms that specify the provisioning of the disabled or infirm represent the "funneling" of resources to individuals in critical periods of life through which they might otherwise not survive (Gurven, 2004; Lee, 1979). This non-random shuffling of resources through human groups places resources where they could have been limiting. This novel way of dealing with problems of limiting resources may be an ancient aspect of human adaptation (O'Connell et al., 1999; Aiello and Key, 2002). The partitioning of finite resources is thought to underlie many life history trade-offs. If human groups are capable of placing resources where their "finiteness" is being felt it mitigates trade-offs. This integration ties group members into the locally available resource base on a roughly equal footing (Gurven et al., 2000; Cadelina, 1982). Plenitude will allow for rapid increase, and scarcity will be felt by all. However, getting resources where they are most needed may be a key element of hominid social systems that encourages demographic expansion.

The ecological and demographic situation of human populations that have systems of ascribed status is different but offers other parallels with female rhesus macaques. Sedentism and food production through agriculture are common characteristics of human groups with ascribed status. Transitions to this form of subsistence in many archaeological populations roughly coincide with major population increases.[1] Often this transition is also marked by reduction in indicators of average health of populations (Larsen, 1995). However, population segments likely experienced health insults to differing degrees. Instead of resources moving to locations where they are limiting as is suggested for foragers, they can be amassed by, and in some cases flow to, higher status population segments. This population segment would be expected not to face life history trade-offs to the same degree and account for larger portions of population growth particularly in times of relative resource scarcity. All population

[1] Whether food production causes population increase or is a solution to it is unimportant to the discussion here.

members may experience benefits of cooperative food production or collective integration that increases the amount of food available, but the relative benefits are much greater for the higher status segments of the population (Pauketat, 2000).

Analogies are dangerous intellectual devices (Marks, 2002). Often this is because they suspiciously rely on connotations of chosen words or literary imagery rather than exposition of specific mechanisms by which the similarities being explored arise (e.g. "nepotism" in macaques or "warfare" in chimpanzees). I have tried to argue that demographic consequences of differences in status in female rhesus macaques may have parallels in human populations. The core mechanism of this analogy is access to limited resources. Strong hierarchies in rhesus females may allow high-ranking females to experience resource abundance, perhaps in times of relative environmental scarcity. This alleviates the effect of life history trade-offs and likely allows them to be have higher population growth rates in marginal habitats (Richard et al., 1989). Egalitarian human foragers have a different solution to the distribution of resources which may also mitigate life history trade-offs. This is the allocation of resources to locations of greatest need. Sedentary human populations that produce their own food rather than gather it from naturally occurring sources have stratified social hierarchies. Resource abundance is likely common at all times among those with greater social power. They should not experience life history trade-offs to any great degree and may experience substantial population growth.

The greatest problem with making an analogy between human and non-human primates at this level is that humans are not always interested in increasing their biological fitness, or at least do not through the same mechanisms that non-human primates do (Roth, 2004; Lam, 2003). They can make conscious decisions about when to reproduce and often have technologies, of varying degrees of effectiveness, to prevent it (Wood, 1998). While high status macaques may simply live longer and produce more offspring, high status humans live in a cultural nexus that offers opportunities for converting calories and other forms of wealth into commodities other than offspring. How and why particular conversions become more popular or widespread may be due to imitation of "successful" individuals (Boyd and Richerson, 1985) or result from the maximization of offspring fitness through heavy investments in education and other social training (Kaplan and Lancaster, 1999). The clearest example, or problem for application of this body of theory, is the "demographic transition." In wealthier countries around the world, and wealthier segments within them, people tend to have fewer offspring (Borgerhoff-Mulder, 1998; Mace, 2000; Hill and Kaplan, 1999). Nevertheless, the social behaviors of rhesus macaque females provide a valuable empirical system demonstrating how fitness should change in response to some of the elements of social inheritance without the opportunities of storage or conversion of "wealth" to anything other than more offspring. When human environments, broadly interpreted to include cultural

norms, prioritize offspring production, the demographic effect of the trade-off circumventing mechanisms in macaque females and human groups are comparable because of the manner in which they manipulate resource availability.

6.3 Unanswered Questions

The conclusions reported above by no means exhaust the potential range of questions to be answered. By and large, they represent an "initial stab" at applying quantitative genetic techniques for understanding the evolution primate life histories and sociality. Much remains to be learned about primate adaptation and evolution by continuing detailed studies of populations (Altmann and Alberts, 2003b).

More detailed study of the genetic architecture of life history, behavioral, and morphological traits should be carried out. The initial investigation here isolated additive genetic and residual variance components for analysis. There are good theoretical reasons to think that life history traits have more complex genetic architectures that include larger amounts of dominance and epistasis than morphological traits (Crnokrak and Roff, 1995; Merilä and Sheldon, 1999, 2000). Dominance and particularly epistasis are departures from the simple additive genetic model for quantitative traits and have interesting effects on the evolution of traits (Roff, 1997). For example, dominance can create asymmetric responses to selection (Hill and Caballero, 1992), and epistatic variance can be converted to additive as populations pass through bottlenecks (Goodnight, 1987, 1988). Large pedigrees with many paternities are necessary for estimating dominance or epistatic variance. These requirements will not be satisfied in most primate populations other than humans, and some laboratory colonies.

Quantitative genetic studies are now often a starting point for identifying not just phenomenological genetic effects, but actual regions of the genome through molecular markers that flank candidate genes that effect the traits being analyzed (Lynch and Walsh, 1998; Rogers, 2005). The data required for such studies that identify quantitative trait loci (QTL) are even more difficult to obtain—molecular data on large numbers of individuals for adequate power. However, one study of wild red deer has already identified a QTL for a fitness component (Slate et al., 2002, 1999), suggesting it may also be possible in wild or free-ranging primate populations. This is also an active area of research with captive primates (Rogers, 2005).

Trade-offs, such as the one identified in this study between early fertility and lifespan, will be further clarified by examining the genetic loci and physiological pathways they act through. Studies of life history trade-offs in laboratory model organisms are being revolutionized as researchers unpack the molecular basis of pleiotropic relationships among fitness components (Bochdanovits and de Jong, 2004) and age-specific patterns of gene expression (Stearns and Magwene, 2003). A much richer picture of the physiological connections among life history traits

and their genetic underpinnings will also force reconsideration of the "many genes of small effect" phenomenological models quantitative genetics (Ricklefs and Wikelski, 2002; Roff and Fairbairn, 2007).

The proximate mechanisms, relative costs and benefits, and historical evolution of hierarchies of social rank should continue to be explored within primate populations, preferably using genetic models or information. The two mechanisms social rank is thought to affect female life history through are stress and priority of access to limiting resources (Sapolsky, 2005; Cameron, 2003; Abbott et al., 2003; Bercovitch and Strum, 1993; Bercovitch, 1991). At Cayo Santiago, the available information allows one to make a legitimate argument for either mechanism, though it is easier to argue for stress in this provisioned population. Detailed studies of food intake and stress-related hormones and behavior in matched individuals would be of great utility in clarifying their relative roles. Furthermore, the potential role of high rank in circumventing life history tradeoffs could be clarified by comparable study of macaque species with more relaxed social styles (e.g. Barbary or Sulawesi macaques).

High social rank in the Cayo Santiago females provides major benefits in fitness. However, it is unclear whether these are net benefits incurred at some small costs. That high ranking females have elevated fitness components does not preclude costs at some point in the reproductive or life cycle. Outside of the free-ranging setting of this island population it may be that costs of high rank increase dramatically to negate some of its benefits. While counterintuitive costs of high rank are well documented in other mammalian taxa (Creel, 2001; Packer et al., 1995) and may be more important in the evolution of hierarchies of social rank than previously thought.

The socioecological model has been of limited use in explaining the evolution of social rank among macaques (Menard, 2004). New directions in research on macaque sociality should incorporate a social selection framework that allows for many factors to mold social behaviors (Wolf et al., 1999). Blending these models with attention to the climatological and biogeographic history of macaques rather than their current ecology will be a productive advance (Abegg and Thierry, 2002). Additionally, molecular genetic research on the genetic and neuroendocrine mechanisms that mediate social dominance and stress responses in macaques will illuminate interspecific differences that may correlate with dominance styles (Wendland et al., 2005).

The prospects for greater applications of molecular and quantitative genetic techniques in wild and free-ranging primates are positive. New statistical and laboratory methods, falling costs of once prohibitively expensive procedures, and increasing cooperation among field researchers with long-term records bode well for future studies of primate life history and sociality (Kruuk, 2004; Strier et al., 2006). Getting the most out of these new resources will require greater emphasis and training in biological anthropology on fields that are less than canonical (e.g. evolutionary genetics, experimental design). The promise of

an evolutionary anthropology of human and non-human primates integrating documented patterns of selection and known genetic variation awaits (Moore and Kukuk, 2002; Marks and Lyles, 1994).

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Author's Biography

Gregory Blomquist was born May 25, 1978 in Bloomington, Illinois. He grew up in Lexington, Kentucky and attended Ohio Wesleyan University, where he received a B.A. in biology and history, prior to coming the the University of Illinois in 2000. He was awarded an American Association of Anthropological Genetics student prize in 2006 for portions of research presented in Chapter 5. Greg spent the 2006–07 academic year as a visiting instructor in anthropology at the University of Notre Dame in South Bend, Indiana. He will move to an assistant professor position in the department of anthropology at the University of Missouri in Fall 2007.