

GENETIC AND MATERNAL EFFECTS ON NEONATAL SURVIVAL IN THE
WESTERN LOWLAND GORILLA

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Master of Arts

by

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The undersigned, appointed by the dean of the Graduate School, have examined the thesis entitled

GENETIC AND MATERNAL EFFECTS ON NEONATAL SURVIVAL IN THE
WESTERN LOWLAND GORILLA

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To my mother, Jane

Thank you for your constant support and encouragement,

and for inspiring me to do what I love.

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ABSTRACT

Quantitative genetic analyses of primate life history traits provide valuable evolutionary insights with conservation and biomedical relevance. Analyzing the proportion of variation in neonatal survival influenced by genes and maternal identity in the critically endangered Western Lowland Gorilla (*Gorilla gorilla gorilla*) explores potential variables impacting this crucial life history trait. The extended developmental periods, extreme dependence of infants on their mothers, and evolutionary relatedness of gorillas to humans indicate that analyzing neonatal survival in gorillas has applications regarding human life history evolution and biomedical concerns of neonatal mortality. I quantify the proportions of neonatal survival influenced by heritable variation and maternal effects from pedigree and survival data in The International Studbook for the Western Lowland Gorilla using variance component estimation. Markov chain Monte Carlo simulations of generalized linear mixed models provide variance component distributions in the animal model tradition. Two models, one with an additive genetic component and one with both additive genetic and maternal identity components, indicate that neonatal survival is heritable. Also, maternal effects significantly influence the observed variation in this trait. This demonstrates that mothers are essential to the survival of young gorillas, and genes have some influence over whether offspring survive the neonatal period of life.

CHAPTER 1: INTRODUCTION

Genetic and non-genetic variables influence the evolutionarily significant life history trait of offspring survival (Cheverud, 1984; Jaquish et al., 1996; Kruuk et al., 2000). While individuals face constant and unique challenges throughout the course of their lives, the neonatal period just after birth is an especially vulnerable time.

Partitioning the variation in neonatal survival influenced by different variables informs researchers how to improve survival rates. Quantifying the amount of heritable variation influencing neonatal survival has direct relevance to natural selection and fitness, and the extent of additive genetic influences vary among species, locations, and individuals (Reale et al., 2003; Wilson et al., 2006; Stoinski et al., 2013). Species with extensive pedigree records provide researchers with the tools necessary for analyzing genetic and maternal effects on survival, and addressing these variables in species closely related to humans provides biomedical applications and insights into survival in humans as well. This project explores additive genetic and maternal identity influences on neonatal survival in captive gorillas included in the International Studbook for the Western Lowland Gorilla (Wilms, 2011).

Gorilla Behavior and Life History

Primates have relatively long neonatal periods during which neonates require extensive care (Altmann, 1980). Gorillas are completely dependent on their mothers during the first few months of life and require provisioning, travel assistance, and protection in order to survive early infancy (Schaller, 1963; Nowell & Fletcher, 2006). Given their critically endangered status as of 2007, western lowland gorillas (*Gorilla*

gorilla gorilla) have become an important subject of conservation efforts and life history research (King et al., 2009; King et al., 2012; Harcourt et al., 1995). All gorilla subspecies are endangered and face threats of habitat destruction, poaching, and disease (Campbell et al., 2011), but the extent to which genetic factors influence mortality is unknown. Analyzing mortality and influences on gorilla survival is relevant to conservation efforts aimed at reestablishing gorilla populations, particularly in captive settings (King et al., 2012). Although the gorillas included in this project are located in captive settings and thus face different threats than gorillas in the wild, genetic analyses can be extended to describe heritable influences on survival in wild gorillas as well. Heritabilities derived from wild and captive populations of the same species from numerous studies yield estimates that are not significantly different (Weigensberg & Roff, 1996; Charmantier & Garant, 2005).

There are currently 2 species and 4 subspecies of gorillas recognized (*Gorilla gorilla gorilla*, *Gorilla gorilla diehli*, *Gorilla beringei beringei*, and *Gorilla beringei graueri*) that are found in 10 African countries (Campbell et al., 2011). Gorillas are long-lived, slow reproducing primates with life spans that can reach 40-50 years (King et al., 2012). Although females reach sexual maturity around age 8, they do not commonly give birth to their first offspring until age 10 and have an average of 1 offspring every 5 years (King et al., 2012). Males typically sire their first offspring around age 12 (King et al., 2012). Although many differences have been noted in the ecological settings of the various subspecies of gorillas, there do not appear to be significant differences in their reproductive parameters (Robbins et al., 2004). Partially due to their long life spans, gorillas (and great apes in general) have longer periods of juvenile development and

dependence on their mothers than other primates or mammals (Nowell & Fletcher, 2007). Regardless of its adaptive significance, this time of dependence indicates that maternal influences should have substantial impacts on survival.

Patterns of contact and mother-infant proximity in gorillas have been well-characterized (Maestriperi et al., 2002). Contact between a mother and her offspring is most common in the first year of life, and time spent in proximity with infants decreases with infant age (Hoff et al., 1981; Maestriperi et al., 2002; Crosby, 2004; Stewart, 1988). Gorilla neonates rely on their mothers for travel, food, and navigating social encounters (Nowell & Fletcher, 2007); however, gorillas are more precocial than truly altricial animals such as mice that are born blind and without fur. Gorilla neonates are more precocial than humans and more altricial than Old World monkeys (Campbell et al., 2011). Due to lactation and locomotion requirements, infants are unlikely to survive without their mothers until 2-4 years of age (Campbell et al., 2011). Given the helplessness and reliance of neonates on their mothers, one would expect mortality to be high during the neonatal (birth to 30 days) period of life, and mothers should influence survival during this time the most.

Risks facing gorilla neonates in the wild include predation, infanticide, injuries, and interactions with other gorillas or animals (Nowell & Fletcher, 2006; Stoinski et al., 2013; Schaller, 1963). Predation is fairly negligible for gorillas due to their size advantage over most species; however, infants and juveniles are vulnerable to predators. Schaller (1963) notes that leopards may pursue young gorillas persistently in the wild and cites leopards as the most frequent predator of gorillas in general. Snake bites are also observed in both juvenile and adult gorillas (resulting in mortality in some cases),

indicating reptiles as another predation risk (Schaller, 1963). Injuries from falls or collisions are other potential risks facing gorilla infants, especially since gorilla sleeping nests are often constructed in trees (Schaller, 1963). Infants sleep with their mothers and are generally not allowed beyond 10 ft of their mothers in the first 4-5 months of life, reducing their chances of incurring such injuries (Schaller, 1963).

Mothers are also essential for mediating their infants' interactions with other gorillas. Gorillas form social groups averaging 6-17 individuals which involve dominance hierarchies, and these require appropriate behaviors not yet known to infants (Schaller, 1963). Although gorillas are often characterized as aggressive, violent interactions are rarely recorded, and infanticide has not been observed in wild western lowland gorillas (Stoinski et al., 2013). Infanticide has been observed 3 times in the eastern lowland gorilla and 6 times in mountain gorillas but only suspected in western lowland gorillas (Campbell et al., 2011). Nevertheless, the constant contact between mothers and their infants in the first months of life provides protection from these potential risks.

The environment influences wild gorilla life history and survival in relation to food provisioning, travel requirements, and disease. Due to the seasonal variation and threat of food scarcity for the frugivorous lowland gorilla, wild lowland gorillas appear to delay weaning for about 1 year in comparison to mountain gorillas and captive gorillas (Stoinski et al., 2013). Captive gorillas are consistently provisioned with food, and mountain gorillas have year round access to plant foods, thus their starvation risk is minimal and survival is enhanced by faster life history development. Since infants are more vulnerable than adults to a variety of risks, especially when dependent on their

mothers prior to weaning, it is in their best interests to progress to a less dependent life history stage more quickly to minimize risks such as predation (Janson et al., 1993). Of course, this is only beneficial if food scarcity is a lesser concern. Finding food is intimately related to travel time and distance for gorillas as most of their traveling is for food procurement purposes (Stoinski et al., 2013).

Infants depend on their mothers for transport until at least 1 year of age (they do not begin to crawl until 2 ½ months), and will be left behind the group if denied assistance (Schaller, 1963; Nowell & Fletcher, 2006). As a group moves to forage, mothers must ensure that their offspring are not left behind and incapable of moving about on their own or provisioning themselves. These survival risks are pertinent in the first year of life because infants have not been observed to prepare their own food (which often requires stripping leaves or removing outer stems) before 12 months of age (Stewart, 1988). Various viruses, bacteria, and parasites similar to those found in human populations also impact health and survival in gorillas (Schaller, 1963). Thus, environmental factors such as disease, food availability, and travel requirements potentially influence survival in wild gorilla populations, particularly for dependent infants.

Gorillas in captivity may not face the environmental challenges of gorillas in the wild, but very young captive gorillas still rely on their mothers for various survival necessities. Nadler (1974) states 3 minimal conditions for the survival of gorilla neonates: initial contact with the mother following birth (perhaps to facilitate breathing), contact that provides feeding opportunities (ventral-ventral contact), and commencement of nursing. If these basic requirements are not met, infants are often removed from their

mothers for fear of neglect and may be foster or hand reared (Crosby, 2004). Captive mothers have been known to act out for attention and mistreat their infants. Bringing males into the enclosure seems to alleviate these tensions and restore mothers to more caring states, emphasizing the importance of social interactions for gorilla well-being (Nadler, 1974).

While infants and mothers are in constant contact during the first year of life for provisioning, travel, and protection, their relationships become more social than resource oriented over time (Schaller, 1963). In order to successfully integrate with other gorillas in a group and eventually reproduce themselves, offspring must learn appropriate behavioral and social skills such as grooming (Schaller, 1963). Mothers are instrumental in the acquisition of these skills, emphasizing their importance for the survival and success of their offspring. Also, mothers appearing to neglect infants or deny them contact after a period of time enable social learning that facilitates emigration characteristic of the species (Crosby, 2004; Maestriperi, et al. 2002; Robbins, et al. 2004). While not all social behaviors that are functional in the wild are applicable to captive gorillas (such as emigration), understanding mother-offspring interactions and social behaviors has implications for assessing adequate levels of care in captive settings.

Mothering gorilla infants successfully may partially depend on social learning and experience. One of the hypothesized reasons captive mothers are not always successful in providing their infants adequate care is that they have not had the opportunity to learn such skills from observing maternal behavior in others (Nadler, 1974; Schaller, 1963). While the extent to which learning through observation impacts maternal care is unknown, parity does appear to influence maternal behavior. Primiparous mothers are

characterized as more restrictive than experienced mothers (Nowell & Fletcher, 2006). Multiparous mothers tend to be more relaxed in letting their offspring explore and develop independence sooner as is common among primates in general (Altmann, 1980). Thus, experience and learning may aid mothers in effective parenting, presumably resulting in increased survival and well-being of their offspring. Additional to these benefits are those experienced by new mothers in terms of their own social status. Silverback males occupy the highest ranks in gorilla groups followed by various females and juveniles (Campbell et al., 2011). Adult females with young infants temporarily move up in rank following the birth of their offspring (Campbell et al., 2011). This may result in more grooming and attention from fellow group mates and more authority in social interactions or decisions (Campbell et al., 2011). Thus, social factors should be taken into consideration with regards to gorilla mothers and their infants as they have implications for the survival and well-being of both.

Several life history traits demonstrate the importance of maternal behaviors to fitness of both mothers and their offspring. Interbirth intervals, size of offspring, number of offspring, and age at weaning are key life history traits that have direct impacts on fitness and survival (Stewart, 1988). Weaning ages are of particular interest due to the survival implications for infants and effects on reproductive fitness of mothers.

Mothers directly impact their infants by providing milk, and the suckling frequency and weaning age of offspring influence both mother and offspring life histories. Suckling frequency is a critical factor for interbirth intervals due to its impacts on hormone levels related to ovulation and conception (Stewart, 1988). Suckling is positively correlated with prolactin concentrations and negatively correlated with gonadal

hormones, providing a potential mechanism of lactational anovulation that may explain some of the variation observed in female fertility (Stewart, 1988). The critical threshold for suckling frequency appears to be 1 bout every 2 hours above which lactation anovulation occurs and below which sexual cycling resumes (Stewart, 1988).

Suckling is difficult to observe with certainty in the wild, and too few data exist to infer causes of variation in suckling frequency at the present. However, nutrition (influenced by various environmental factors) likely influences lactation and suckling frequencies, supported by earlier weaning ages observed in gorillas with more consistent access to food sources (Stoinski et al., 2013). Average age at weaning according to Nowell & Fletcher (2006) is approximately 4.5 years in wild western lowland gorillas who do not have access to weaning fruits year round. Weaning clearly has survival and health implications for infants and juveniles, but also determines how soon mothers can energetically afford to reproduce again. In the event that an infant dies, its mother is likely to have shorter birth spacing and reproduce again sooner (Stewart, 1988). In the wild, offspring tend to remain with their mothers for approximately 3 years, and no female has been observed nursing more than 1 dependent infant at a time (Schaller, 1963). Weaning ages and interbirth intervals are estimated to vary about 25% among various gorillas in the wild and captivity, indicating that there is some plasticity in these life history traits (Stoinski et al., 2013). Resource availability likely contributes to this variation; however, further investigation is needed to address variables impacting these life history traits.

Ecological-Evolutionary Influences on Life History Traits

Partitioning the observed variation for life history traits into genetic and non-genetic variance components is essential for interpreting population dynamics and evolutionary change (Pelletier et al., 2007). Since natural selection operates on traits passed on from generation to generation, quantifying the genetic bases of phenotypes is important for describing variation in an ecological-evolutionary framework. Investigating how genes influence variation in demographic rates and fitness allows for a more detailed understanding of genetic and environmental impacts in a variety of populations (Wilson et al., 2010).

Partitioning observed variation into genetic and environmental sources can be informative on both an individual and population level (Postma, 2005). Individual variation in life history traits influences population dynamics and is directly tied to natural selection as it determines which individuals survive and reproduce. However, ecological factors limit population sizes and dynamics as well, demonstrating that quantitative genetic analyses can inform ecological-evolutionary researchers how population dynamics change when considered alongside environmental factors (Saccheri & Hanski, 2006). Although environmental factors influencing population characteristics differ between captive and wild animal populations, genetic variation can be quantified and applied to both (Weigenberg & Roff, 1996; Charmantier & Garant, 2005).

Heritability in the narrow sense estimates the proportion of variation for a phenotypic trait (such as survival) that can be explained by additive genetic variation (Falconer, 1960). This assumes that additive genetic variation results from numerous genes, each with relatively small effect, that sum together to influence a trait. As these

genes are inherited from parent to offspring each generation and expressed phenotypically, they provide natural selection something to act upon. Selection can change phenotype distributions via alteration of underlying gene frequencies. Heritability estimates attempt to partition the amount of variation in a trait influenced by additive genetic variation from other factors, but neglecting to sort out other variables can inflate heritability estimates and ignore other influences on phenotypic variation.

Variation influenced by mothers and the care they provide for their offspring is an important variable to consider for life history traits such as survival. This is called a maternal effect and can be defined as the observed effects of maternal performance on offspring phenotype (Cheverud, 1984; Marshall & Uller, 2007). These effects are additional to the genotypic effects offspring directly inherit from their mothers (Wolf & Wade, 2009). If maternal effects are not taken into account when partitioning variances in quantitative genetic analyses, heritability estimates are likely inflated due to the inclusion of influences other than additive genetic variance in their calculations (Jaquish et al., 1996). Maternal effects include behavioral characteristics of mothers, provisioning efforts, protection against predation, milk yield, etc. (Cheverud, 1984). Even when heritability estimates are low, maternal effects may represent a relatively large portion of phenotypic variance for life history traits such as birth weight, birth date (Wilson et al., 2004), number of offspring, and lactation rate (Rasanen & Kruuk, 2007). The basis for these effects may or may not be genetic, but quantifying their influence on offspring phenotypes demonstrates their importance with regards to life history traits (Blomquist, 2013).

Given the significant influence mothers have on the survival of their offspring (particularly in the neonatal period), maternal effects are thought to have the most influence on offspring phenotypes early in life (Altmann, 1980; Nowell & Fletcher, 2007; Wilson & Reale, 2006). The behaviors included in maternal effects such as food provisioning and predator protection have clear survival (and fitness) implications for offspring, but they also affect fitness of mothers (Marshall & Uller, 2007). Although maternal effects influence both maternal and offspring fitness, these are not always positively correlated as it may be more beneficial for a mother to invest more time into producing more offspring than caring for those she already has (Cheverud, 1984; Marshall & Uller, 2007). This parent-offspring conflict also influences the dynamics of maternal effects and has implications for their adaptive influences on fitness (Trivers, 1974; Cheverud, 1984).

The adaptive implications of maternal effects are context dependent, but Badyaev (2005) found that the most rapidly evolving traits have the greatest maternal effects. Given that maternal effects involve phenotypic plasticity and the ability to adapt to different environments and conditions, it is intuitive that maternal effects impact traits that are context dependent and evolve rapidly (Blomquist, 2013; Badyaev, 2005). Neonatal survival is context dependent with regards to environmental threats, should be under strong selective pressure to increase, and thus could be evolving rapidly. As demonstrated in the rhesus macaques at Cayo Santiago, maternal effects significantly affect survival in young primates and should be considered when analyzing phenotypic variation (Blomquist, 2013).

Modeling Heritability and Maternal Effects

Variance components are useful for estimating heritability and maternal effects because they partition observed (phenotypic) variation into specified components that represent proportions of that variation. In order to estimate variance components for heritability and maternal effects regarding neonatal survival in gorillas, other effects should be taken into account that also influence this trait such as sex and maternal age. Mixed models have this capability and are thus the method of choice for this project. Mixed models are mixed in the sense that they allow the inclusion of both fixed and random effects in their calculations, accounting for biases or influences from multiple variables on the trait of interest (Kruuk, 2004). These models also allow the use of full pedigree relationships between individuals. I estimated variance components for heritability and maternal effects in the gorilla studbook using mixed models developed in the animal breeding literature (Henderson, 1975). Often referenced as the “animal model”, breeding values of each animal are estimated in this tradition. However, use of mixed models has extended beyond animal breeding applications into ecological-evolutionary research, medical research, and general quantitative genetic analyses.

The animal model gives breeders a way to estimate the genetic possibilities in a species (Knott et al., 1995). Prior to the introduction of the animal model, breeders estimated genetic variances from controlled breeding experiments or by sampling a population over many generations to track a trait’s progress (Roff, 2007). The application of the best linear unbiased predictor was a breakthrough in plant/animal breeding as it allowed for better estimation of breeding values and potential genetic responses (Henderson, 1975; Belovsky & Kennedy, 1988). This predictor introduced by Henderson

is now known as the animal model and allows for prediction of random effects from explicitly modeled fixed effects (Wilson & Reale, 2006). Fixed effects affect the mean of a trait's distribution whereas random effects influence the variance (Kruuk, 2004). Including both types of effects in the predictive model produces a more accurate estimation of variance components, and effectively breeding values (Hudson & Kennedy, 1985). Also, this method uses information on all relatives in a population rather than just breeding individuals, incorporating more information than was previously used to predict trait outcomes (Belonsky & Kennedy, 1985).

Maternal effects and life history traits can also be studied via the animal model (Henderson, 1984; Quaas & Pollak, 1980). Since many of the traits animal breeders are interested in directly relate to weight and size, growth data and maternal effects are important to consider based on the impact mother's have on the growth (and survival) of their offspring (Henderson, 1984; Quaas & Pollak, 1980). Thus, the animal model has wide applications within and extending beyond animal breeding to predict the proportion of a trait's variance that can respond to selection.

Phenotypic changes have been observed on ecological timescales, demonstrating the importance of considering genetic influences on phenotypic traits in order to make inferences regarding evolution (Carroll et al., 2007; Nussey et al., 2005). The animal model provides a way of partitioning variation into genetic and non-genetic components while accounting for many of the influences that would otherwise bias quantitative analyses (Milner et al., 2000; Postma, 2005). The pedigree information required by mixed models is often unavailable for wild populations of interest to ecologists but available for animals in captive settings. Although the applicability of heritability estimates derived

from captive animals has been called into question with regards to wild populations, comparisons between captive and wild estimates do not reveal significant differences (Weigenberg & Roff, 1996; Charmantier & Garant, 2005).

I explored the narrow-sense heritability or additive genetic variance of neonatal survival in these studbook data with the animal model to partition observed variation in this trait into additive genetic and residual components. I also added maternal effects into a second model to partition phenotypic variance for neonatal survival into additive genetic variance, variance influenced by mother's identity, and residual variance. Since neonatal survival is a binary trait of either life or death, generalized linear mixed models (GLMMs) are necessary to account for the non-Gaussian nature of this characteristic. I fit these models with Bayesian Markov chain Monte Carlo simulations to produce posterior distributions of each variance component. These distributions and their posterior modes provide a more accurate reflection of uncertainty in the parameter estimates than point estimates with confidence intervals produced by restricted maximum likelihood (REML).

Conservation and Biomedical Applications

Measuring the heritable component of survival has implications for conservation in wild populations. Without knowing if a portion of variation in survival is genetic, researchers and conservationists may assume that mortality in young age classes is entirely a result of environmental disturbances. Information regarding survival heritability can help researchers assess how strongly environmental disturbances may be affecting mortality in wild populations. In other words, if there is a strong genetic component to survival, the environment cannot solely be responsible for mortality. This knowledge is relevant to policy decisions that may remove animals from their habitats

unnecessarily. Thus, mixed models allow researchers to assess numerous variables that influence the variation in demographic and life history traits.

In addition to the value heritability estimates have for researchers with ecological/evolutionary interests, maternal effects are also relevant to quantify in these contexts given the direct impact mothers have on their offspring early in life. Since these effects have implications for mothers' reproductive success as well as the survival of their offspring, they can result in immediate phenotypic change and changes in population dynamics (Rasanen & Kruuk, 2007). Population growth rates are more sensitive to adult mortality than neonatal mortality in most primates (Blomquist et al., 2008). However, adult mortality varies less in primates than in other vertebrates, and this is likely influenced by primate social and ecological adaptations resulting from cognitive abilities and flexible diets (Morris et al., 2011). Addressing variation in neonatal survival produces valuable insights regarding mothers and their offspring. Population ecologists, population geneticists, and evolutionary biologists can all benefit from this type of information given their common interests in life history traits and the related genetic and environmental influences underlying population variation (Saccheri & Hanski, 2006).

Variance components derived from mixed models are relevant to clinical research in human populations as well. Estimating heritability and partitioning variation in disease risk into genetic and environmental components has been a longstanding interest in medical research (Falconer, 1965; Olshen & Wijsman, 1996). Early attempts to understand inherited disease risk relied on twin studies or specific sets of relatives (such as full siblings) due to the limitations of regression analysis and analysis of variance methods. Heritability estimates derived from only individuals with these particular

relationships generally have less statistical power (Nance & Corey, 1975; Pawitan, 2004). Furthermore, adjustments for age, sex, parity effects, etc. are necessary in all heritability studies but not directly included in estimates generated from regression or ANOVA (Nance & Corey, 1975). Mixed models allow medical researchers to explicitly model fixed effects (sex, age, various environmental characteristics) in order to estimate heritabilities from larger, more inclusive pedigrees (Song & Lee, 2006). Heritability estimates and maternal effects have been utilized throughout medical research to better understand factors influencing disease, predict which individuals are at high risk for disease, and inform treatment/prevention efforts (Falconer, 1965; Curnow & Smith, 1975).

Investigating genetic influences on infant survival is pertinent in human populations given that malformations and genetic disorders are the leading cause of infant mortality in the U.S. (Stevenson & Corey, 2003). Infant mortality is considered a worldwide measure of health, and quantifying how it is influenced by various factors is essential to its decrease. Twin studies indicate that genetic differences influence approximately $\frac{1}{4}$ of the variation in adult lifespan, but there is considerable variation among cohorts (Christensen et al., 2006). Other estimates suggest that longevity is moderately heritable (McGue et al., 1993).

Significant heritability estimates for life history traits derived from twin studies demonstrate that there is a genetic component to survival; however, precise estimates of heritability in life history traits are problematic in human populations due to cultural transmission and common environments (Pettay et al., 2005). These factors likely inflate heritability estimates, and environmental variables are more difficult to model for human

populations due to their complexity and the inability to monitor/control environments such as in animal populations. Due to their relatively close ancestry, heritability estimates from non-human primates may be informative of the extent to which genes influence life history traits in humans.

Gorillas are an ideal species for which to analyze genetic and maternal effects on neonatal survival because of their extended developmental periods, evolutionary relatedness to humans, and endangered status. Few studies have analyzed survival in gorillas because of statistical complications (King et al., 2012). Since gorillas have long life spans and relatively few offspring, datasets with large samples sizes and high enough mortality to analyze are rare. Much of what is known about mother-offspring interactions comes from case studies of individual pairs or a few gorillas in one location (Maestriperi et al., 2002; Stokes et al., 2003; Crosby, 2004). The International Studbook for the Western Lowland Gorilla (Wilms, 2011) provides a large dataset for analyzing the heritability and maternal effects influencing neonatal survival, producing valuable insights regarding survival and reproduction.

CHAPTER 2: METHODS

Data were extracted from the publicly available 2010 Gorilla International Zoo Studbook (Wilms, 2011). Information on 2,089 individuals was stripped from a PDF of the studbook, and 3 new variables representing lifespan, mother's age, and neonatal survival were added to the dataset. Lifespan was calculated by subtracting date of birth from date of death, and mother's age was calculated by subtracting each mother's date of birth from her offspring's date of birth. In order to analyze neonatal survival, I created a binary variable from the values of lifespan with 1=neonatal death and 0 =neonatal survival. I applied the standard neonatal period from birth to 30 days used in humans to these data (Lawn et al., 2005); therefore, neonatal survival was defined as living past 30 days of age. Demographers define neonatal mortality as death following live birth during the first month of life in order to exclude stillborns or miscarriages from this calculation (Barfield, 2011). Unfortunately, information on still v. live births, miscarriages, and gestation time is unavailable for these gorilla data. Therefore, neonatal mortality in this analysis includes deaths from all recorded births through 30 days after birth. This could result in a higher neonatal mortality rate due to the potential inclusion of preterm births, stillbirths, miscarriages, etc. Heritability and maternal effect estimates quantify the proportion of all these deaths influenced by additive genetic variation and maternal identity.

Kaplan-Meier survival curves display survival percentage for these gorilla data over lifespan in years as well as months to allow a closer look at survival rates early in life (Figure 1). A steep slope in the first month of life indicates that the death rate slows substantially following the neonatal period. While males have slightly lower survival

percentages than females over much of their lifespan, the survival curves show little difference between the sexes just after birth. The survival curves include data from 2,022 individuals in the studbook for which birth dates and exit dates are recorded. This also includes individuals exiting the studbook via transfer or release. Data from these gorillas are appropriate for inclusion in survival curves based only on lifespan, but analyses requiring pedigree information and variables with more specific constraints demand additional trimming. Thus, survival rates for gorillas in the survival curves do not match precisely with rates reported for the trimmed data used in later analyses.

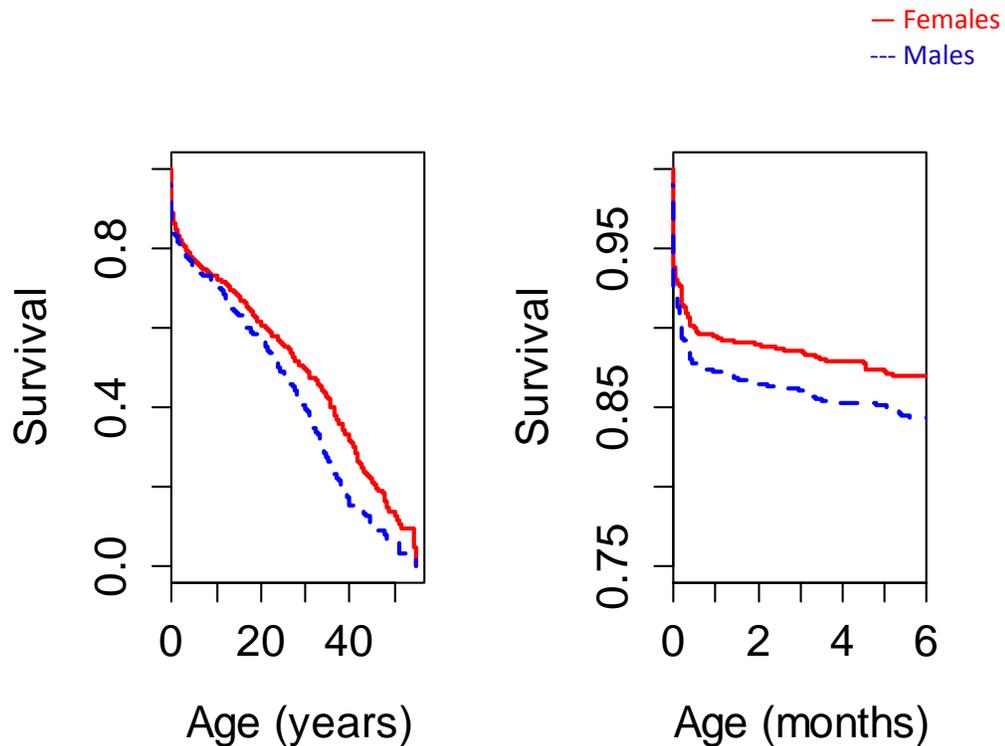


Figure 1. Kaplan-Meier Survival Curve. Survival percentage of gorillas in the studbook over lifespan in years and months.

Individuals must have been born before October 31, 2010 in order to have survived past the 30 day neonatal period, so offspring born after this date were not included in the analysis. Older age classes were not analyzed for mortality due to low

numbers of infant death in age ranges beyond the neonatal period (Figure 1). Gorillas of unknown sex were excluded from the analysis. To allow separation of maternal identity and additive genetic effects, I used only offspring from mothers that had 2 or more offspring in the dataset. The trimmed dataset includes 1,112 scored neonates with 203 cases of neonatal death, resulting in a neonatal mortality rate of 0.183.

Generalized Linear Mixed Models

Generalized linear mixed models (GLMMs) were used to analyze maternal effects and heritability of neonatal survival. GLMMs are an extension of linear mixed models (LMMs) that can be used to analyze non-Gaussian data such as these binary data. LMMs are extensions of classic linear regression models that model a mix of random and fixed effects in order to predict an outcome variable (Kruuk, 2004).

$$\mathbf{y} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}\mathbf{u} + \mathbf{e}$$

Equation 1 represents a general mixed model in matrix form (Kruuk, 2004). \mathbf{y} is the $n \times 1$ vector of the outcome variable neonatal death. \mathbf{X} is an $n \times p$ matrix of the p fixed effects and $\boldsymbol{\beta}$ is a $p \times 1$ vector of fixed effect regression coefficients. \mathbf{Z} is a $n \times q$ design matrix representing q random effects with \mathbf{u} as the vector of solutions for each random effect, and \mathbf{e} is the residual error (Kruuk, 2004; Blomquist, 2013). Fixed effects of GLMMs represent variables for which all levels are found within the data, such as sex (Wilson, 2010), and random effects quantify a portion of the variation among the individuals or units such as individual identity (Bolker et al., 2009). Although random effects presumably capture variation in the outcome variable, including fixed effect parameters is important for reducing bias. Fixed effects also influence how GLMMs can be fit and interpreted.

GLMMs are often fit by maximum likelihood (ML) or restricted maximum likelihood (REML). Both likelihoods make assumptions about fixed effects and should be avoided unless fixed effects are known with certainty and precision. Also, REML requires that models have identical fixed effect structures to produce valid likelihoods (Wilson, 2010). Other techniques for estimating parameters in GLMMs include Gauss-Hermite quadrature, penalized quasilielihood (PQL), Laplace approximation, and Markov chain Monte Carlo simulations (MCMC). Bayesian MCMC models have several advantages with regards to bias and output, and are thus the technique of choice for these analyses.

Bayesian MCMC simulations sample the model parameters many times, and the algorithm converges on the posterior probability distribution of the specified model parameters (Bolker et al., 2009). The generated samples are taken sequentially from fixed parameter values, random effect levels, and random effect parameters (Bolker et al., 2009). In order for the model to converge on the posterior probability distribution, it must first track through parameter space until the autocorrelation of a signal with itself is sufficiently low. Number of iterations, length of burn-in, and sample thinning interval for the model are specified based on the autocorrelation and how long the model takes to track through parameter space. Samples are saved at each thin interval after the model has completed the specified number of burn-in samples.

Data were analyzed in R 3.0.1 (R Development Core Team, 2013), and variance components were estimated from the MCMCglmm package (Hadfield, 2010). Two models were run with the same fixed effects (see below) but different random effect structures. Model 1 had neonatal identity as its only random effect whereas Model 2 had

neonatal identity and maternal identity as random effects. In both models, neonatal identity was linked to the pedigree such that covariance among neonates was predicted by Mendelian rules of allele sharing, providing an estimate of additive genetic variance. Neonatal sex and maternal age were the only fixed effects in both models. Maternal age was modeled as a natural cubic spline with knot points at 15 and 25 years. Additional fixed effects of neonatal birth cohort and neonatal inbreeding were explored but dropped from the final models due to lack of association with survival. Rearing type (mother v. non-mother) was also explored but not included because many neonates were uncategorized.

Pedigree information from the studbook specified individual, dam, and sire (Table 1), and kinship coefficients were derived from this information. I ran 2 simulations of 6,000,000 iterations, burn-in of 1,000,000 and a thin of 5,000 in order to estimate variance components from the posterior distributions of set parameters.

Table 1. Summary pedigree statistics for the trimmed dataset. F value denotes the number of gorillas with any amount of inbreeding greater than 0.

Pedigree Members	Maternities	Paternities	Maximum Pedigree Depth	F > 0	Mean Pairwise Relatedness
1,326	1,112	1,088	4	24	0.009

Priors were specified for fixed effects, G-structure ($V=1$, $\nu=1$, $\alpha.\mu=0$, $\alpha.V=1000$), and R-structure ($V=1$, $\text{fix}=1$). Priors for fixed effects included the mean vector and covariance matrix, G-structure refers to the random effects, and the R-structure sets the linear predictor for each residual (Hadfield, 2010). I applied parameter expanded priors to both GLMMs in which the residual variance was fixed at 1, the prior mean was set at 0, prior covariance matrix set to 1,000, and 1 degree of freedom was allowed for random effects (Hadfield, 2010). Residual variance was fixed because the

binary response variable of neonatal death/survival could only be observed once for each individual.

The MCMC simulations drew samples from the joint posterior distributions resulting in posterior distributions of each variance component. Looking at the highest posterior density (HPD) intervals gave 95% credible intervals for each simulation. Rather than providing a point estimate which may or may not contain the mean such as in frequentist confidence intervals, credible intervals are probability distributions within which 95% of the samples fall. Therefore, 95% of these data fall within the HPD intervals generated from MCMC sampling. Variance component distributions were used to calculate the genetic and maternal effects on neonatal death for the gorillas in the studbook. These mixed effects models partitioned the observed or phenotypic variance in neonatal death into additive genetic and residual variances to quantify the additive genetic component of this trait. Posterior distributions of heritability for each model were produced from the variance component distributions.

In order to account for the binary nature of these data, they were fit with the logit link function (Nakagawa & Schielzeth, 2010). This link stabilizes the variance and allows for easy interpretation on the link scale. Interpreting variance component estimates on the transformed link scale is preferable to the original scale because they are conditionally dependent on the mean (Nakagawa & Schielzeth, 2010). Since maternal effects and heritabilities are ratios of variance components, the effect of this transformation is negligible when interpreting random effects. However, interpreting fixed effect distributions requires some manipulation. The logit transformation is based on probability (Jaeger, 2008). The probability of success divided by the probability of failure for a trait,

$\frac{p}{1-p}$, is called the odds. Taking the log of the odds completes the logit transformation, and the probability can then be back calculated from $p = \frac{\exp(\log odds)}{1 + \exp(\log odds)}$.

Calculating Heritability and Maternal Effects

Heritability and maternal effect estimates were calculated from variance component ratios. The distribution specific variance for the logit link function is $\pi^2/3$ and must be accounted for in the calculations. $\frac{\sigma_a^2}{\sigma_a^2 + \sigma_e^2 + \pi^2/3}$ was used to calculate variance component ratios for Model 1 (Nakagawa & Schielzeth, 2010) where σ_a^2 is the variance distribution for individual identity (the additive genetic variance component) and σ_e^2 is the variance distribution of the residual error. For Model 2, mother's identity (σ_m^2) was added as a random effect. $\frac{\sigma_a^2}{\sigma_a^2 + \sigma_m^2 + \sigma_e^2 + \pi^2/3}$ was used to calculate heritability, and $\frac{\sigma_m^2}{\sigma_a^2 + \sigma_m^2 + \sigma_e^2 + \pi^2/3}$ produced the maternal effect for Model 2.

Heritability from Model 1 has a straightforward interpretation. The ratio of variance components in Model 1 gives the proportion of total phenotypic variance accounted for by variation among individual identities. The resulting value represents the amount of variation for neonatal survival explained by the additive effect of genes (Wilson et al., 2010). Adding maternal effects into Model 2 accounts for how much of the mother's phenotype affects her offspring. If this is not included for a population in which many of the individuals are siblings, covariance between siblings may appear as part of additive genetic variance due to sharing the same maternal environment (Wilson et al., 2010). Modeling maternal identity as a random effect does have limitations. It is

unknown whether maternal effects are of genetic or environmental origin and what aspects of the environments provided by mothers are causal (milk yield, contact time, etc.). This modeling was appropriate in these data given the known paternal identities in the pedigree and the inclusion of only mothers with more than one offspring. Without this information, maternal effects cannot be separated from additive genetic variance (Wilson et al., 2010).

1,000 samples of fixed effect log odds were retained from each MCMC simulation as well as posterior distributions of variance components and random effect solutions. The shapes and locations of these distributions were used to determine whether the parameters were greater or less than 0 by considering the range covered by the credible intervals (Blomquist, 2013). Although deviance information criterion (DIC) is often used for model comparisons with different random effects, it is not applicable to GLMMs and was not used to assess the fit of the 2 models considered (Wilson et al., 2010). However, given that Model 2 accounts for an additional factor that likely influences neonatal survival, it should more accurately quantify the relationship between additive genetic variation and neonatal survival.

CHAPTER 3: RESULTS

Both GLMMs show significant additive genetic variance for neonatal survival in gorillas. Model 1 produced a variance component for individual identity which describes the amount of variation in neonatal survival influenced by a gorilla's identity or genes. Model 2 produced variance components for animal identity and maternal identity, partitioning these influences on neonatal survival. Variance component distributions for Model 1 and Model 2 are located on the transformed logit link scale (Figure 2). The maternal variance component has an expectedly smaller distribution than that of either individual variance, and the individual variance component is larger in Model 1.

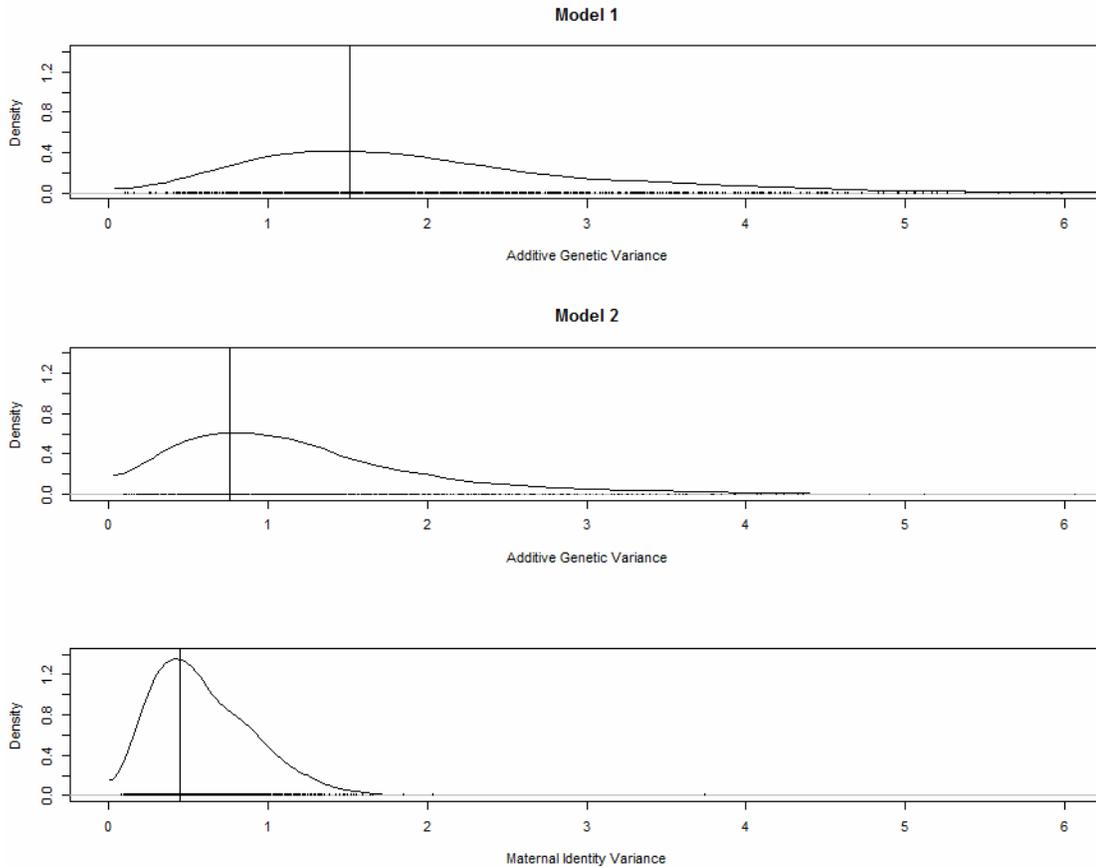


Figure 2. Variance component posterior distributions. Vertical lines denote posterior modes.

Heritability and Maternal Effect Estimates

Heritabilities and maternal effects are measured by variance component ratios and represented by posterior distributions as well. The heritability for Model 1 shows the distribution for the proportion of variance in neonatal survival explained by shared genes among the gorillas (Figure 3). The credible interval for this heritability spans 0.092 to 0.548 (Table 2), indicating that genes do have an impact on neonatal survival. The posterior mode of 0.261 (Table 2) suggests that approximately a quarter of the variation observed in gorilla neonatal survival may be attributed to additive genetic effects.

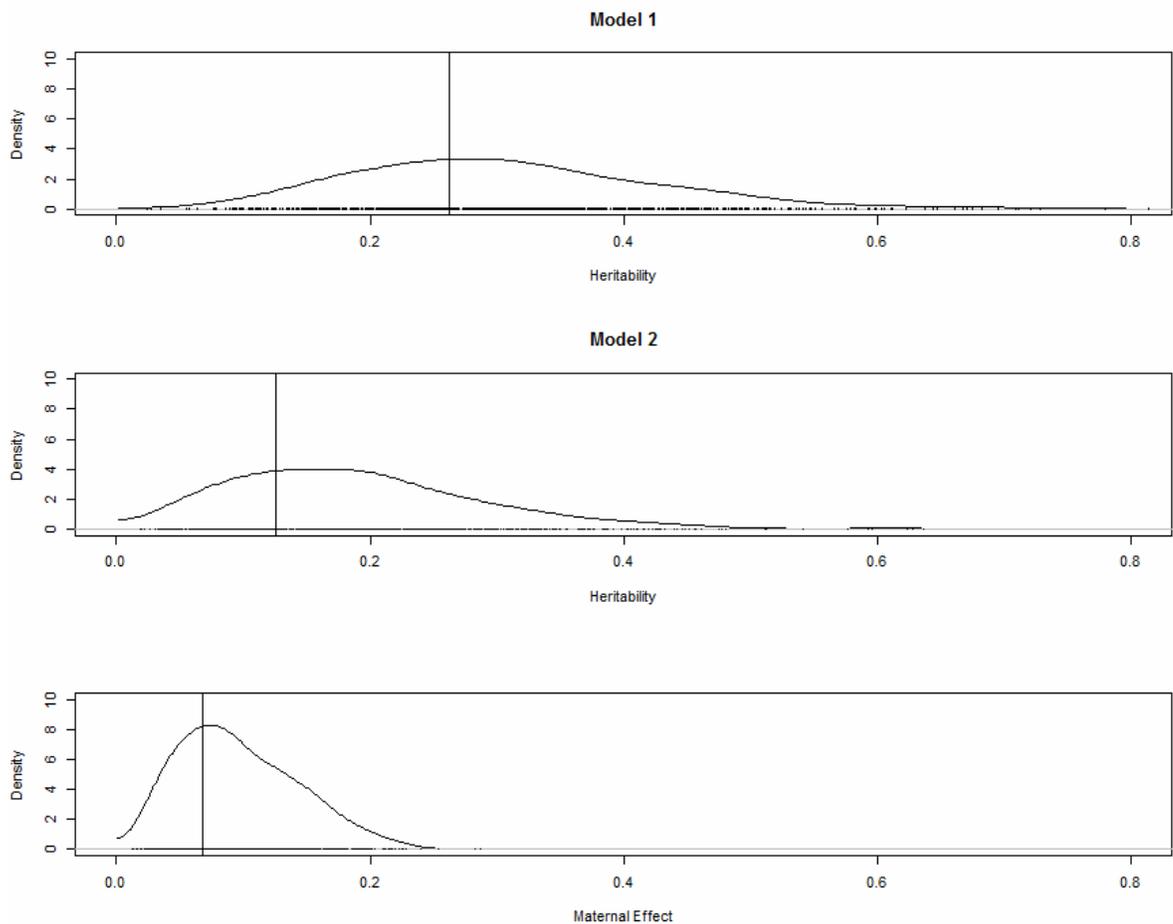


Figure 3. Heritability and maternal effect posterior distributions. Vertical lines denote posterior modes.

Model 2 has a posterior distribution for the amount of variation in neonatal survival explained by genes and the amount of variance influenced by maternal identity (Figure 2). The credible intervals span 0.030 and 0.016 to 0.387 and 0.187 respectively (Table 2). This indicates that maternal effects have a significant impact on neonatal survival. The posterior modes show 12.6% of variation in neonatal mortality is due to additive genetic effects, while 6.8% is related to maternal identity (Table 2). Whether or not maternal effects have a genetic or environmental basis is unknown. Together, the ratio of individual identity and maternal identity variances to the total explain 22.7% of the variation in neonatal survival (Table 2). This is the estimate of non-residual variation explained by the modeled random effects.

Table 2. Summary statistics for Model 1 and Model 2 posterior distributions. Credible intervals are given in parentheses.

Model		Variance Component			Variance Component Ratio	
		Mean	Posterior Mode	Effective Sample	Posterior Mode	Effective Sample
Model 1	Individual	2.160	1.51 (0.105-4.630)	895	0.262 (0.092-0.548)	1000
Model 2	Individual	1.260	0.449 (0.091-2.990)	1000	0.126 (0.030-0.387)	1000
	Maternal	0.598	0.759 (0.112-1.220)	1134	0.068(0.016-0.187)	1000
	Individual + Maternal	—	—	—	0.227 (0.123-0.470)	1211

The individual and maternal variance component ratio distributions represent 1,000 randomly chosen samples from the MCMC simulations. Effective sample sizes reflect that the simulations track through parameter space appropriately (Table 2). Both GLMMs suggest that there is a genetic component to neonatal gorilla survival. Model 2 indicates that a portion of this variation is also influenced by mother’s genes, care,

genetic influence on quality of mother’s care, the maternal genetic contribution to offspring, their covariance, or any combination of the above.

Fixed Effect Results

Both models also produced fixed effect distributions (Table 3). The posterior mean for sex in Model 1 indicates that the odds of neonatal death are 31.65% higher for males than females, and this is found by taking the exponent of the mean log odds (0.275). The logs odds mean for Model 2 shows that the odds of neonatal death are 33.46% higher for males. The baseline death rates for males and females are 0.213 and 0.168 respectively.

Table 3. Summary statistics for Model 1 and Model 2 fixed effects. Neonatal sex and mother’s age were included for both models, and mother’s age was modeled with knot points at 15 and 25 years. Credible intervals are given in parentheses.

Model	Fixed Effect	Posterior Mean	Effective Sample	p
Model 1	Intercept	-1.5354 (-2.3592- -0.7306)	1000	<0.001
	Sex	0.2750 (-0.1382-0.7130)	1000	0.214
	Mother's Age 1	-0.6593 (-1.8403-0.3561)	1000	0.254
	Mother's Age 2	-0.9893 (-3.0922-1.0942)	1000	0.370
	Mother's Age 3	0.7807 (-1.4455-2.6065)	1000	0.400
Model 2	Intercept	-1.5972 (-2.2571- -0.8208)	1000	<0.001
	Sex	0.2886 (-0.1480-0.6676)	1000	0.178
	Mother's Age 1	-0.6680 (-1.7668-0.4593)	911	0.250
	Mother's Age 2	-0.7986 (-2.9755-1.1293)	1000	0.434
	Mother's Age 3	0.9833 (-0.7951-2.7468)	1000	0.264

An effects plot displays the effect of maternal age on the probability of female and male neonatal death for Model 2 (Figure 4). Given that the fixed effect distributions for the 2 models are essentially identical, an effects plot for one model sufficiently displays the effects of maternal age on neonatal death for these data. Direct interpretation

of posterior means for mother's age is not feasible because this variable was modeled as a cubic spline. Therefore, plotting the posterior mean predictions of neonatal death from the fixed effect distribution by mother's age in years visually displays this relationship. Confidence bands were produced from joining quantiles from the generated MCMC sample solutions, and the inverse logit bands at 2.5% and 97.5% represent 95% of this sample distribution. This effects plot shows that probability of neonatal death is higher for very young and old mothers with the lowest probability around 20 years (Figure 4). Posterior mean predictions and confidence bands are similar for male and female neonates, but probability of neonatal death is slightly higher for males.

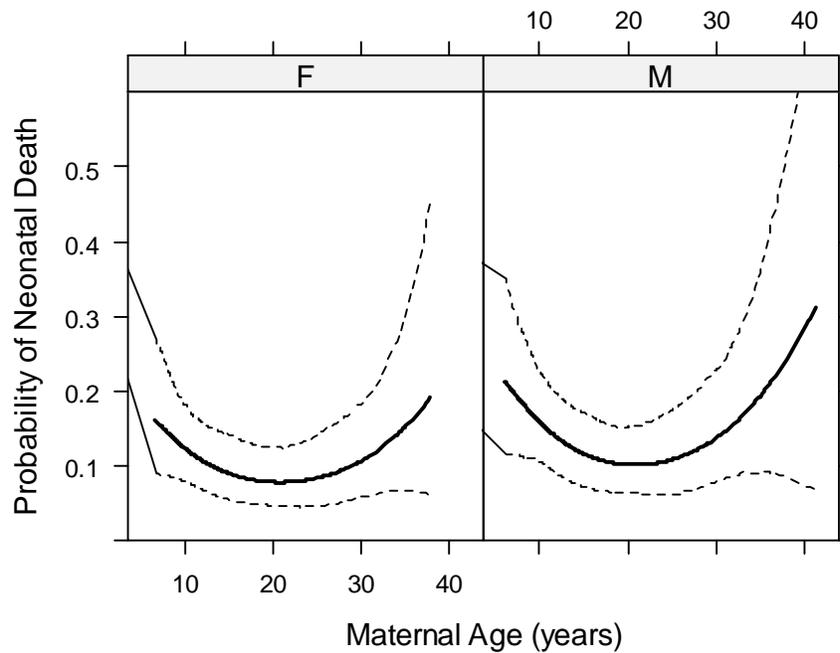


Figure 4. Effects plot of maternal age on the probability of neonatal death by sex. 95% confidence bands are plotted in dotted lines.

CHAPTER 4: DISCUSSION

The variance component ratios from this analysis reveal that genes and maternal effects contribute to neonatal survival in gorillas. Heritability estimates from both models and the maternal effects estimate from Model 2 are all significantly greater than zero, and their posterior modes suggest that up to a quarter of the variation in neonatal survival can be explained by these variables. This falls within the range of expected heritability based on analyses of life history traits in other mammals (Martin et al. 2002; Kruuk et al., 2000; Reale et al., 2007) and indicates that there is genetic and maternal variation influencing this trait.

Life history traits are predicted to have lower heritability estimates than morphological traits based on their direct relevance to fitness (Price & Schluter, 1991; Kruuk et al., 2000). Because of this relation to fitness, natural selection should play a strong role in selecting advantageous genes, thereby reducing variation by preserving only advantageous alleles. This could result in lower heritabilities if residual variation due to the environment remains high. Either very high residual variation or very low genetic variation can result in low heritabilities. Thus, the significant heritabilities and maternal effects for neonatal survival indicate that there is considerable additive genetic variation and maternal effects in this trait relative to residual variation.

The narrow-sense heritabilities reported from Model 1 and Model 2 provide insights regarding the evolutionary potential of the rate of neonatal survival (Houle, 1991). Since heritability describes the additive genetic component of a trait that can be

passed from parent to offspring, heritabilities estimate the amount of additive genetic variation upon which selection can act (Wilson et al. 2005; Hansen et al., 2011). In other words, the heritabilities of 26.2% from Model 1 and 12.6% from Model 2 indicate that these are proportions of the variation in neonatal survival that could be targeted by natural selection. This implies that survival rates have evolutionary potential.

In addition to the additive genetic variance captured in narrow-sense heritability estimates, dominance and epistasis may also contribute to genetic variation. However, variation due to the masking effects of dominant over recessive alleles and epistatic interactions between genes are difficult to disentangle for quantitative genetic studies. Very large amounts of data and many different types of relationships between individuals are needed to partition variation into these different components (Hill et al., 2008). Even so, direct estimates of dominance and epistasis are difficult to obtain due to how confounded they are with environmental factors. Inbreeding can allow for better detection of dominance variance through directional effects implied in inbreeding depression (Hill et al., 2008). However, the lack of inbreeding in these data (Table 1) prevents estimates of this sort. Furthermore, additive genetic variance is shown to account for more than 50% (and often greater than 80%) of genetic variation for complex traits when dominance and epistasis can be estimated (Hill et al., 2008). While dominance and epistasis could influence the genetic variation contributing to variation in neonatal survival in these data, their exclusion is not problematic with regards to the implications of significant additive genetic variance. Thus, dominance and epistasis are potential contributing factors to the variation observed in neonatal survival, but heritability

estimates and the partitioning of maternal identity variance provide valuable insights in their own regard.

Model Comparison

Comparing heritabilities from Model 1 and Model 2 demonstrates the importance of including maternal effects when estimating heritability. The heritability estimate from Model 1 is larger than from Model 2, suggesting that the exclusion of maternal effects inflates the estimate of the amount of variation influenced by additive genetics. The significance of the maternal effect estimate from Model 2 is consistent with the observed mortality of infants who lose their mothers in the wild. Data from wild gorilla populations show that mothers are essential to the survival of their offspring during the first year of life with very high mortality rates for those who lose their mothers (Robbins, 2011). Thus, significant maternal effects on neonatal survival are an expected result. It also confirms that maternal effects are worth including in variance component estimation.

Model 2 is preferable to Model 1 because it provides a more accurate heritability estimate. There is stronger a priori justification for Model 2 over Model 1 from current knowledge of survival requirements of gorilla neonates. Maternal effects warrant inclusion since gorillas are completely dependent on their mothers for provisioning, transportation, and protection during at least the first few months of life (Schaller, 1963; Nowell & Fletcher, 2006). Given that the definition of maternal effects refers to any maternal behaviors that influence offspring phenotypes (Cheverud, 1984; Marshall & Uller, 2007), provisioning, transportation, and protection certainly fit this description and should be considered. Furthermore, these maternal behaviors described in both wild and

captive settings have direct survival impacts. Therefore, maternal effects are a pertinent variance component with regards to the trait of interest.

Without paternity knowledge, maternal effects would be difficult to separate from additive genetic variance (Wilson et al. 2010). These data from captive gorillas are ideal for modeling maternal effects because paternities are known in most cases. If paternities were unknown, all of the measured additive genetic variance would be attributed to mothers. If all variation in neonatal survival was attributed to mothers due to limited pedigree information, it would not be possible to separate additive genetic variance from other maternal effects. Known paternities ensure that additive genetic variation accounts for relationships between offspring and their fathers as well as their mothers. Maternal effects then measure something separate from this when just mother's identity is added as a random effect. Thus, this International Studbook for the Western Lowland Gorilla (*Gorilla gorilla gorilla*) is an ideal dataset for measuring heritability and maternal effects due to extensive pedigree information available in captive settings and the large sample size.

Maternal effects may or may not have genetic sources (Marshall & Uller, 2007) and may differ between wild and captive gorillas. These can include quality of care, mother's genes that may influence her behavior towards offspring, learned maternal behaviors, or any combination thereof (Cheverud, 1984; Marshall & Uller, 2007). Captive gorillas still require care in the form of provisioning, limited transportation, social learning, and protection, thus significant maternal effects for neonatal survival are expected in these captive data. However, maternal effects may matter even more for survival in wild gorilla populations. Presumably, wild gorillas have greater exposure to

predators, other gorillas, and greater travel needs. Unfavorable environmental conditions are thought to make differences (and variation) between mothers more pronounced (Charmantier & Garant, 2005). Since wild animals likely face a wider range of threats and environmental variables than those in captivity, variation in maternal effects could be greater in the wild. This would likely result in a larger maternal effect depending on the amount of residual variation.

Due to large sample size and known paternity requirements, assessing maternal effects in wild gorillas would be a daunting task. Since it is unknown to what extent maternal effects have a genetic basis, it is difficult to say precisely how similar maternal effects may be for wild and captive gorillas facing different environmental challenges and survival needs. Since maternal effects are significant in these captive data, it is reasonable to predict that maternal effects would also be significant (likely more so) in the wild. Nevertheless, the significance indicates that variation among mothers influences neonatal survival in captive settings, and observing maternal behavior towards neonates is worthwhile.

Fixed Effect Implications

In order to account for factors that could bias heritability and maternal effect estimates, I included sex and mother's age as fixed effects for both models. The sexual dimorphism in gorillas and different growth trajectories for males and females indicate that survival could differ between sexes. In addition to physiological factors for very young or very old mothers that could influence their neonates' survival, experience and learned skills that mothers gain over time could underlie mother's age effects on survival.

Sex and mother's age variables were readily available in the studbook and required no manipulation to add into the models.

Sex and maternal age are known to influence mortality rates among various primate species, and differences between the sexes with regards to survival are attributed to sexual selection (Kruger & Nesse, 2006). The longer lifespan of females in these gorilla data are consistent with numerous animal studies (Kruger & Nesse, 2006), and higher odds of neonatal mortality for males are expected and observed among humans as well (Naeye et al., 1971; Balsara et al., 2013). While the cause(s) of greater odds for male neonatal mortality in these data are unknown, they support the general consensus in life history and medical literature which states that females are more robust and males more vulnerable in terms of survival (Kruger & Nesse, 2006; Balsara et al., 2013).

Maternal age is a recognized factor influencing neonatal health and survival as well (Vaughan et al., 2013). Offspring born to mothers of extreme maternal age (low or high) are more likely to experience health issues and mortality in several species, including humans (Vaughan et al., 2013). This trend is also found in these data with the risk of neonatal mortality decreasing with age for very young gorilla mothers and increasing with age for mothers over the age of 25. Since parity is unknown in these studbook data (mothers may have given birth before entering the studbook), maternal age is meant to capture similar effects. Parity is known to affect maternal behavior and may contribute to decreasing mortality with age for young mothers (Nowell & Fletcher, 2006), although the causes of maternal age trends vary widely among individuals (Vaughan et al., 2013). Parity and maternal age matter less with regards to maternal behaviors in taxa commonly used in studies of maternal effects such as lab mice and rats (Lehrman et al.

1972). Their relevance to maternal behaviors in primates emphasizes the importance of including maternal age as a fixed effect in this project.

I explored adding two additional fixed effects into the models by recoding cohorts and rearing types. Variables coding for rearing type and cohort were recoded to reflect type of rearing relevant to the analysis and larger cohort groupings to include more gorillas per cohort. Type of rearing was recoded to reflect either parent or non-parent rearing. I collapsed rearing type of none into parent, foster into hand rearing, and peer into unknown, leaving 3 categories of parent, hand, or unknown rearing. Cohort was recoded into semi-decades. The first cohort included all years prior to 1969 including 1969, and subsequent cohorts continued in 5 year increments through 2010.

Given the differences in mortality observed for gorillas that are reared by their mothers compared with those hand reared in captivity (Crosby, 2004), rearing type seems like a plausible influence on neonatal survival. However, neonatal survival in these data did not have a strong relationship with rearing type even when collapsed into 3 groups. Likewise, cohort did not influence survival enough to include in the models. I considered using this variable to account for potential differences in captive gorilla survival over time. It is reasonable to question whether the quality of care gorillas receive in captivity has improved or changed over time as we have more opportunities to learn about their needs. The original cohort variable provided the year in which each gorilla was born, and increasing each cohort span to five years did not show an influence on survival over time.

Out of the 1,112 gorillas included in the pedigree, only 24 have any amount of inbreeding (Table 1). This negligible variable was not included as a fixed effect due to its low prevalence. Influential amounts of inbreeding are unexpected in captive primates

since their mating partners are controlled/monitored. This monitoring allows for informative pedigrees with known maternities and paternities while also avoiding health consequences that can result from inbreeding (Rails & Ballou, 1982). Large numbers of inbred individuals are also unexpected in the wild due to the typical emigration patterns observed in which female gorillas leave their birth groups to join an unrelated silverback male (Campbell et al., 2011). Inbreeding is likely negligible for both captive and wild gorillas; however, the relatedness of gorillas is generally unknown when they enter the studbook. Genomic data would be necessary to quantify inbreeding more accurately.

Conservation, Biomedicine, and Future Directions

Genes and maternal effects influence survival in gorilla neonates, but these variables do not fully explain the causes of mortality in offspring who do not survive the neonatal period. With regards to conservation efforts, significant heritabilities indicate that neonatal survival is influenced by more than exclusively environmental factors. Efforts to increase gorilla population numbers can benefit from this information with increased attention and monitoring of neonates whose relatives have suffered neonatal mortality.

Heritability estimates from gorillas may apply to humans due to their relatively close ancestry and similarities in life history traits. Neonatal mortality rates vary among human populations, but the World Health Organization (WHO) reports that a child's risk of dying is greatest in the first month of life (WHO). Recent global estimates of combined perinatal mortality, stillbirths, and neonatal mortality in humans produce a mortality rate of 124 deaths per 1000 births (0.124) (WHO, 2006). The mortality rate of 0.183 for these gorillas is higher than that of human populations. While the causes of

neonatal death are also unknown for these gorilla data, the WHO states preterm birth, asphyxia, and infections as the most common causes of death for human neonates (WHO, 2014). These factors could certainly contribute to the neonatal mortality rate in these gorilla data, but complications during delivery, miscarriage, or still births due to various health, environmental, and genetic factors likely contribute to the rate as well. Also, humans face obstetric constraints giving birth due to large brains and pelvic modifications required by bipedalism (Rosenberg & Trevathan, 2003). While some other primate species face similar constraints, gorillas have more spacious birth canals due to their relatively large bodies (and pelves) and small neonates (Rosenberg & Trevathan, 2003). Thus, a portion of human neonatal mortality rates may reflect complications due to obstetric constraints whereas this is unlikely to be the case in gorillas.

Significant heritability for neonatal survival in gorillas indicates that survival in human neonates may be heritable. This leads to further questions regarding specific genes that contribute to neonatal mortality and whether or not these are shared between gorillas and humans. Given that there is significant additive genetic influence for this trait, it is reasonable to suspect that further research and exploration could uncover genes and biological processes to explain how additive genetic variation functionally operates to influence survival. Extensions of such research apply to biomedical interventions and potential treatments to increase neonatal survival in endangered gorilla populations and humans alike. Although the current analysis cannot address these specific questions, uncovering significant heritability for neonatal survival indicates that this is a research topic of genetic interest.

This analysis demonstrates that the additive effects of a neonate's genes contribute to his or her mortality risk, but unless genetic mutations directly cause mortality, it is the interaction of genes with other biological and environmental variables that leads to the outcome of survival or death. Survival requirements of provisioning, travel assistance, and protection apply to all gorilla neonates regardless of their genetic susceptibility to neonatal mortality. Environmental threats to these necessities may vary drastically in the wild. However, each neonate's combination of genes explains about 12% of his or her risk of dying from birth until 30 days of life.

Maternal effects indicate that mother's identity influences neonatal survival by about 6-7%, but the underlying causes of variation in maternal effects are unknown. Each mother's genes, life experiences, social learning, observations of other mothers, and environmental circumstances may influence whether or not her offspring survive. While the variables causing maternal effects are not addressed in this analysis, we can still predict that offspring born to mothers who have successfully reared neonates before are more likely to survive their own neonatal periods of infancy. Thus, gorilla conservation efforts will likely benefit from observations of maternal behavior.

The ambiguity of the causes of maternal identity variance indicates that this topic would also benefit from future research. Knowing that maternal effects significantly contribute to variation in neonatal survival provides an additional variable for conservation and biomedical researchers to consider. Further investigation into causes of maternal identity variance would allow comparisons between maternal effects in gorillas and humans, potentially improving neonatal survival in both. Gorilla neonates face a wide range of variables that challenge their likelihood of surviving the vulnerable first

month of life, but the additive effects of their individual genes and maternal effects displayed by each mother contribute to the variation observed in this evolutionarily significant life history trait.

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