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RESEARCH ARTICLE

Modeling Variation in Early Life Mortality in the Western Lowland Gorilla: Genetic, Maternal and Other Effects

MONICA H. AHSAN* AND GREGORY E. BLOMQUIST
Department of Anthropology, University of Missouri, Columbia, Missouri

Uncovering sources of variation in gorilla infant mortality informs conservation and life history research efforts. The international studbook for the western lowland gorilla provides information on a sample of captive gorillas large enough for which to analyze genetic, maternal, and various other effects on early life mortality in this critically endangered species. We assess the importance of variables such as sex, maternal parity, paternal age, and hand rearing with regard to infant survival. We also quantify the proportions of variation in mortality influenced by heritable variation and maternal effects from these pedigree and survival data using variance component estimation. Markov chain Monte Carlo simulations of generalized linear mixed models produce variance component distributions in an animal model framework that employs all pedigree information. Two models, one with a maternal identity component and one with both additive genetic and maternal identity components, estimate variance components for different age classes during the first 2 years of life. This is informative of the extent to which mortality risk factors change over time during gorilla infancy. Our results indicate that gorilla mortality is moderately heritable with the strongest genetic influence just after birth. Maternal effects are most important during the first 6 months of life. Interestingly, hand-reared infants have lower mortality for the first 6 months of life. Aside from hand rearing, we found other predictors commonly used in studies of primate infant mortality to have little influence in these gorilla data. Am. J. Primatol. 77:666–678, 2015.

Key words: quantitative genetics; infant mortality; gorillas; life history; animal model

INTRODUCTION

Offspring survival is a life history trait of evolutionary significance influenced by both genetic and non-genetic variables [Cheverud, 1984; Jacquish et al., 1996; Kruuk et al., 2000]. While individuals face constant and unique challenges throughout the course of their lives, infancy is an especially vulnerable time. Quantifying maternal effects and heritable variation influencing survival has direct relevance to studies of natural selection and fitness, and the extent of additive genetic and maternal influences varies among species, locations, and individuals [Réale et al., 2003; Stoinski et al., 2013; Wilson & Réale, 2006]. This study estimates maternal effects and additive genetic variation influencing gorilla survival for age classes ranging from birth to 2 years. We also model other effects such as parity, paternal age, and rearing type on infant survival, and data for all analyses come from captive gorillas included in the international studbook for the western lowland gorilla [Wilms, 2011].

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, 2012]. 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 and maternal identity influence mortality is unknown. Furthermore, there is considerable variation in infant mortality for western lowland gorillas with studies in wild groups reporting mortality rates ranging from 8 to over 40% during the first year of infancy [Robbins et al., 2004]. Research in wild gorilla populations suggests that survival is more critical to population growth than fertility and other related traits [Robbins et al., 2011], and estimating the heritability of infant survival quantifies the impact of genes passed on from parents to offspring with regards to this crucial trait. Heritability in the narrow sense estimates the proportion of

*Correspondence to: Monica H. Ahsan, Department of Anthropology, University of Missouri, 1205 University Ave., Suite 1110 Columbia, MO 65201. E-mail: mha8tb@mail.missouri.edu

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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. Pedigree records provide information on relatedness (proportion of shared genes) among individuals, thus supplying the necessary data for estimating additive genetic variation [Walsh, 2001].

Genetic variation for traits closely related to fitness (i.e., survival) is expected to be low for several reasons [Houle, 1992]. Environmental variation and chance events or accidents influence survival, increasing the overall variation in this trait. Consequently, genetic variation for life history traits is thought to be smaller proportional to overall variation than would be the case for morphological traits [Price & Schluter, 1991]. Given the complexity of traits related to fitness and reproductive success, many loci are also likely involved in these heritabilities [Houle, 1998]. In other words, mutational target size is large for traits such as survival, and many mutations would be required to produce measurable variation in gene expression [Landry et al., 2007]. Experimental work in *Drosophila* suggests high levels of mutational input and potentially additive genetic variance for early life survival that declines at later ages [Pletcher et al., 1999]. Although we do not expect high additive genetic variation for infant survival, estimating this variable across age classes may reveal differences in genetic effects at different times throughout infancy.

The extended developmental periods and relative altriciality of gorilla offspring indicate that mothers are important for survival as well [Campbell et al., 2011]. Variation in offspring traits influenced by mothers and the care they provide for their offspring is a maternal effect, and this term is more formally defined as the observed effects of maternal performance on offspring phenotype [Cheverud, 1984; Marshall &Uller, 2007]. Like genetic variation, maternal effects play a key role in evolution for many species as they transmit traits and behaviors from one generation to the next [Maestripieri & Mateo, 2009].

Maternal effects are crucial to offspring survival in species with prolonged mother–infant interaction. Cheverud and Wolf [2009] state that maternal effects are the most important source of variation among newborn mammals. These effects generally decline in importance over time, and are most important for survival prior to weaning [Cheverud & Wolf, 2009]. Although maternal effects have not yet been quantified for gorillas, mother–infant proximity and interactions (reported from behavioral studies) decrease over time during the first 3 years of life [Nowell & Fletcher, 2007]. Maternal effects such as foraging skills, social behaviors, stress responses, and antipredator strategies are often passed from generation to generation [Mateo, 2009]. This affects natural selection, especially for primates in environments that remain stable over time. We predict that maternal effects will significantly influence infant survival and decline over time.

Both additive genetic (narrow-sense heritability) and maternal effect variance components can be quantified from sufficient pedigree information, and these estimates for gorilla infant mortality are relevant to conservation and ecological-evolutionary interests. Estimating these effects informs caretakers and researchers of the importance maternal identity and heritable variation hold for survival in these endangered apes. Maternal effects are of particular interest given the importance of maternal care and learned behaviors in social primates with long developmental periods [Maestripieri & Mateo, 2009]. Furthermore, estimating maternal effects for different time periods throughout the first 2 years of life may provide insights into the importance of maternal behaviors at various stages of infancy.

In addition to estimating maternal and genetic effects on infant survival, the effects of other variables such as parity, paternal age, sex, and hand rearing can be modeled from studbook data. Such analyses also provide information relevant to breeding programs and conservation efforts by identifying traits important to survival. Parity has been observed to influence maternal behaviors in gorillas such that primiparous mothers are characterized as more restrictive than multiparous moms [Nowell & Fletcher, 2007]. Also, parity is known to significantly influence offspring survival in mountain gorillas with primiparous mothers experiencing higher infant mortality [Robbins et al., 2006]. We expect parity to influence infant survival in these gorilla data such that primiparous mothers experience greater offspring mortality risk.

Although male gorillas do not generally care for offspring, paternal age may impact survival through germ cell mutations [Momand et al., 2013]. De novo mutations increase as males age, and preterm births, low birth weights, and impaired immune function are potential consequences of advanced paternal age [Alio et al., 2012; Eisenberg & Kuzawa, 2013]. However, mating patterns also influence sperm competition and male mutational biases such that male gorillas may exhibit less germ line mutations than other species [Venn et al., 2014]. Nevertheless, we predict that offspring sired by older fathers may exhibit higher mortality risk given the risks associated with high paternal age. There are also potential differences between male and female offspring survival. The general consensus in life history and medical literature states that females are more robust and males more vulnerable in terms of survival [Balsara et al., 2013; Kruger & Nesse, 2006]. Thus, we expect that males will generally have a higher risk of infant mortality than females.

In addition to these biological traits that may influence infant survival, there are several variables related to captivity and zoo environments that are relevant to this analysis. Birth cohort may influence variation in survival rates, as this studbook includes gorillas born from 1925–2010 [Wilms, 2011]. Following the formation of the gorilla species survival plan (SSP) in 1985, recommendations were made to change animal care practices, and reproduction became more carefully managed [Ryan et al., 2002]. Over time, research on social behaviors and other gorilla traits has also been incorporated into care in captive settings. Given the policy change in 1985, cohort may affect survival such that gorillas born after 1985 have lower mortality risk.

Zoo location is another potentially important variable due to the variation in policies and practices between zoos. This indicates that the location of an individual may also influence his/her survival [Porton & Niebruegge, 2006]. Gorillas born in captivity may be hand-reared by zoo personnel for a time. There are many reasons why zoos remove infants from their mothers to care for them such as neglect, unsuccessful nursing, and wanting to have infants in nurseries to attract visitors [Porton & Niebrugge, 2006]. Mothers may reject newborns who appear sickly or unlikely to survive as to not expend resources unnecessarily. Hand-reared gorillas often exhibit abnormal behaviors later in life, such as asocial behaviors, and lack expected social–sexual behavior. Gorillas who were hand-reared themselves also have fewer offspring than mother-reared gorillas [Ryan et al., 2002]. Thus, hand rearing is a variable with behavioral and reproductive consequences that likely influences survival as well.

There are a multitude of variables potentially impacting early life survival of gorillas in zoos, and estimates of maternal effects and heritability should account for as many relevant variables as possible. Mixed models have the capability to incorporate many variables simultaneously 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 for the use of full pedigree relationships between individuals and are referenced as "animal models" [Wilson et al., 2010].

The pedigree information required by mixed models is often unavailable for wild populations of interest to ecologists but available for animals in captive settings [Quinn et al., 2006]. Although the applicability of heritability estimates derived from captive animals has been called into question in relation to wild populations, comparisons between captive and wild estimates do not reveal significant differences [Garant, 2005; Weigensberg & Roff, 1996]. We estimated the narrow-sense heritability and maternal effects of infant survival in these studbook data with animal models, and explored the effects of other variables of interest (parity, paternal age, etc.) as well. We 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) [Wilson et al., 2010].

Gorillas are an important species for which to analyze maternal and genetic effects on infant survival because of their extended developmental periods 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 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 [Crosby & Lukas, 2004; Maestripieri et al., 2002; Stokes et al., 2003]. The international studbook for the western lowland gorilla [Wilms, 2011] provides a large dataset for analyzing variables that influence early life mortality, potentially producing valuable insights regarding survival and reproduction.

METHODS

We extracted pedigree and life event data from the 2010 international studbook for the western lowland gorilla [Wilms, 2011]. This research adhered to the American Society of Primatologists principles for the ethical treatment of primates. While there were over 2,000 total individuals in the studbook, we analyzed survival of a subset of 986 (498 female, 488 male) that satisfy several criteria. We used only western lowland gorillas born in captivity of known sex and parentage for whom maternal parity could be accurately calculated (mother was captive-born or entered captivity at age 3 or under), the mother's death did not cause the infant death, and the infant was either hand or mother-reared. We omitted any individuals that were known abortions or stillbirths, determined by name and cause of death in Dewar's unofficial gorilla studbook [Davis, 2014]. Retaining hand-reared infants in the analysis is potentially problematic, but they account for about a third of the dataset and should provide some information on inherent frailty of offspring that may have a genetic cause regardless of rearing. Moreover, comparing hand and mother-reared infants may indicate periods of early life in which hand rearing is particularly important.

We initially explored mortality in the selected subset visually, and then made decisions on age class

boundaries for later analysis. An age-specific Kaplan–Meier survivorship plot showed rapid decline in the first years of life (Fig. 1). Changing risk of death during this period was further illustrated by plotting fortnightly exponential hazard rates for the first 3 years of life [Gentleman, 2014]. This showed very high risk of mortality in the first 2 weeks of life and then a slow decline out to 2 years with death becoming very rare after that. Based on these figures, we decided to focus on the first 2 years of life (days 0–730). We did not extend the analysis to later ages where death is rarer because model results would become extraordinarily imprecise. Because of the rapid change in mortality and related behavioral and physical development of infants, we also analyzed four smaller age classes: day 0, first fortnight (days 1–14), remainder of first 6 months (days 15–182), and remainder of first year and all of second year (days

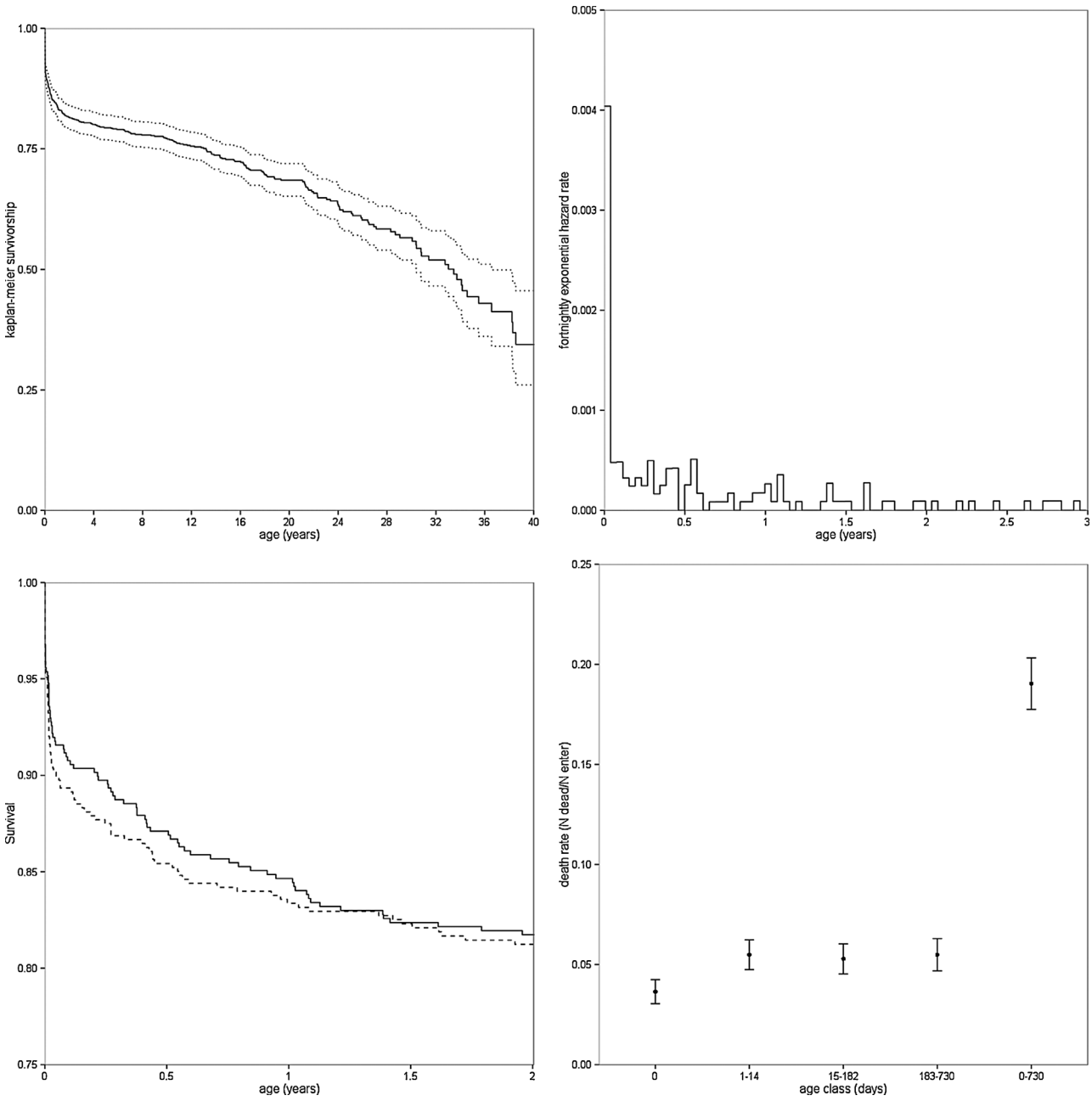

Fig. 1. Gorilla survivorship and death rates in the subset of individuals analyzed ($N = 986$). A Kaplan–Meier survivorship plot for ages 0–40 years on the top left shows the steep drop in the first years of life. The top right panel gives piecewise exponential hazard rates for fortnightly age bins from ages 1 day to 3 years. The bottom right panel gives death rates calculated for the age classes analyzed with standard error bars; dot sizes are proportional to the number of gorillas entering the age class. Sex-specific Kaplan–Meier survivorship curves are plotted for the first 2 years of life at bottom left (females solid, males dashed).

183–730). These divisions are somewhat arbitrary, but balanced between even annual or semiannual periods and shorter periods earlier to capture the changing risk of mortality (Table I). Moreover, the number of deaths in each age class would have become too small for statistical analysis if shorter age classes were used [Peduzzi et al., 1996]. The number of infants in each age class equals the number in the previous age class minus deaths in the previous class as well as infant deaths due to maternal death or other factors violating our selection criteria. The decision to use these time periods was made prior to modeling factors influencing mortality.

To model mortality, we began by screening a set of predictor variables for death in the 0–730 age class using generalized linear models implemented in the glm function of R [R Core Team, 2014]. Because mortality is a binary variable, we used a logit link. Predictors were chosen based on a priori interest in effects or knowledge of the data structure. They were mother versus hand rearing of the infant, mother versus hand rearing of the mother, region of the world (North America vs. elsewhere, primarily Europe), time period (pre- or post-1985 just after the establishment of the gorilla SSP), maternal parity (divided into three bins of primiparous, second or third birth, and fourth or higher offspring), paternal age (divided into five bins: <15 years, 15–20 years, 20–25 years, >25 years), infant inbreeding coefficient, and infant sex. We binned parity and paternal age rather than treating them as continuous variables to account for very low numbers at extreme parity or ages.

The predictor screening was approached in two ways. First, we fit a model with all of the predictors and tested the importance of each by dropping them one at a time from the model (backward selection). This yields a likelihood ratio test for each predictor as well as information criteria for each reduced model compared to the full model (Table II). As an alternative to these null hypothesis tests [Lewis

TABLE I. Descriptive Statistics of Gorilla Mortality, Rearing, and Pedigree for Each Age Class

	0	1–14	15–182	183–730	0–730
Phenotyped IDs	986	947	889	801	935
Died	36	52	47	44	178
Hand-reared	320	317	311	301	313
Pedigree members	1186	1139	1076	984	1134
Maximum generations	5	5	5	4	4
Founders	182	176	172	168	181
Maternities	985	944	883	795	935
Paternities	989	948	889	800	939
Maternal grandmothers	543	520	485	424	498
Maternal grandfathers	533	510	470	400	488
Paternal grandmothers	458	443	410	360	417
Paternal grandfathers	533	510	470	400	488
Maternal sibs	909	869	806	717	865
Paternal sibs	963	922	864	779	920
Maternal half-sibs	134	131	128	124	125
Paternal half-sibs	188	184	186	186	180
Full sibs	775	738	678	593	740
$F > 0$	27	23	23	20	25
$F \geq 1/16$	24	21	21	19	23
$F \geq 1/8$	19	16	16	14	18
$F \geq 1/4$	12	10	10	8	11
% Largest family	97.976	98.420	98.327	98.272	97.972
% Disconnected	0.675	0.615	0.651	0.610	0.617
Mean offspring per mother	3.107	3.035	2.914	2.809	3.106
Mean offspring per father	5.852	5.745	5.556	5.298	5.869
Mean maternal sibship ($n > 1$)	3.772	3.682	3.566	3.498	3.745
Mean paternal sibship ($n > 1$)	6.734	6.633	6.400	5.992	6.525
% Maternal singleton offspring	7.716	7.945	8.720	9.811	7.487
% Paternal singleton offspring	2.629	2.743	2.812	2.625	2.023
Mean A_{ij}	0.010	0.010	0.010	0.010	0.010
% $A_{ij} > 0$	7.523	7.630	7.499	6.936	7.010
% $A_{ij} \geq 1/16$	5.794	5.898	5.881	5.592	5.597
% $A_{ij} \geq 1/8$	3.800	3.869	3.908	3.824	3.763
% $A_{ij} \geq 1/4$	1.746	1.786	1.823	1.844	1.777

F is the inbreeding coefficient, A_{ij} is the kinship coefficient between individuals i and j.

TABLE II. Generalized Linear Model Test of Effects in the 0–730 Day Age Class

	Df	Deviance	Δ_{BIC}	LRT	P	β
Full model		831.943	–			
Hand-reared	1	881.047	42.263	49.104	<0.001	−1.561
Male	1	832.077	−6.707	0.134	0.715	0.064
F	1	832.715	−6.069	0.771	0.380	2.431
Mother hand/peer-reared	1	835.240	−3.544	3.297	0.069	−0.374
N. America	1	834.678	−4.105	2.735	0.098	−0.309
Pre-1985	1	837.226	−1.558	5.283	0.022	0.500
Parity	2	833.757	−11.868	1.813	0.404	
Paternal age	3	841.012	−11.453	9.069	0.028	

Each row gives fit statistics for dropping a single term from the full model. Coefficients are provided for the dummy variables and should be added to an intercept of −1.062.

et al., 2011], we also used change in the Schwartz–Bayesian information criterion (BIC) for the reduced models because it heavily penalizes model complexity with large samples. We used the dredge function of the MuMIn package [Barton, 2014] to find the best set of predictors according to the model BIC [Burnham & Anderson, 2002], examining all models with change in the BIC less than six ($\Delta_{BIC} < 6$, Table III). Both approaches indicated that hand rearing of the infant was the only strong predictor of infant mortality in the 0–730 age class (Tables II and III). We could not screen all of the predictors in the smaller age classes because there were too few deaths in each to include more than two or three predictors [Peduzzi et al., 1996], but more limited models for these age classes reinforced the importance of hand rearing and absence of infant sex effects (Table IV, Fig. 1).

Finally, we used generalized linear mixed models (GLMMs) implemented in the MCMCglmm package [Hadfield, 2010] to extend the analysis of infant mortality to include variance components for rearing environment and additive genetic effects. Based on the glm results, we used hand rearing as the only fixed effect predictor in these models. Two logistic GLMMs were run for each age class with the same fixed effects but differing random effects. Both models included rearing environment interacted with the fixed effect of infant hand rearing [Hadfield, 2010]. This rearing environment variable was coded as either maternal identity from the studbook if the infant was mother-reared, or a dummy identifier created for the zoo if the infant was hand-reared. This gave separate uncorrelated variance components for each rearing type that describe heterogeneity among the mothers (V_M) or zoos (V_{ZOO}) in infant mortality rates. The maternal variance component is of primary interest for our study. A second model run for each age class added identity of the infant linked to the full studbook pedigree to estimate additive genetic variance (V_A). Each individual's breeding value (the sum of effects of alleles at all variable loci affecting mortality) is modeled through Mendelian rules of allele sharing predicted from the studbook (see θ below) to estimate the additive genetic variance component [Kruuk, 2004; Walsh, 2001]. The genetic model can be expressed in matrix form as shown in Equation 1.

$$\text{logit}(E[y]) = X\beta + \sum_{i=1}^{3} Z_i u_i + e \quad (1)$$

With n mortality records in the vector y, the $n \times 2$ fixed effect design matrix (X) associates mortality with mother versus hand-rearing status. The vector β contains the fixed effect coefficients for the intercept and hand rearing. Random effect design

TABLE III. Model Selection Table for 0–730 Days in Generalized Linear Models

Intercept	Hand-reared	Male	F	Mother hand	North America	Pre-1985	Parity	Pat. age	df	logLik	Δ_{BIC}	Weight
−1.252	−1.570					0.693			3	−426.894	–	0.472
−1.099	−1.533				−0.441	0.760			4	−423.844	0.740	0.326
−1.139	−1.501			−0.380		0.615			4	−425.091	3.233	0.094
−1.112	−1.377								2	−432.806	4.982	0.039
−1.016	−1.474			−0.324	−0.405	0.688			5	−422.570	5.033	0.038
−0.986	−1.316			−0.493					3	−429.586	5.383	0.032

Any model with $\Delta_{BIC} < 6$ from the best model (first row) is displayed. Coefficients are displayed for the included terms.

TABLE IV. Generalized Linear Model Tests of Hand Rearing and Sex Effects in Each Age Class

Age class (days)	β	SE	z	P
0				
Intercept	−3.076	0.261	−11.780	<0.001
Hand-reared	−1.701	0.607	−2.802	0.005
Male	0.230	0.343	0.670	0.503
1–14				
Intercept	−2.611	0.217	−12.054	<0.001
Hand-reared	−1.613	0.476	−3.392	0.001
Male	0.179	0.288	0.621	0.535
15–182				
Intercept	−2.511	0.217	−11.567	<0.001
Hand-reared	−1.820	0.528	−3.449	0.001
Male	−0.021	0.302	−0.070	0.944
183–730				
Intercept	−2.565	0.228	−11.230	<0.001
Hand-reared	−0.500	0.347	−1.442	0.149
Male	−0.253	0.313	−0.807	0.420

matrices associate infants with their mothers if mother-reared ($n \times$ number of mothers, Z_1) or the zoo if hand-reared ($n \times$ number of hand-rearing zoos, Z_2) and all infants with their location in the pedigree ($n \times$ number of pedigree members, Z_3). The random effect vectors (u_i) contain the BLUPs for the effect of each mother (u_1), zoo (u_2), or infant breeding value (u_3) on infant mortality. Each is assumed to have a mean of 0, with respective variances of V_MI, V_{ZOO}I, and $2\,V_A\theta$, where θ is a matrix of kinship coefficients. The residual vector (e) is also assumed to have mean of 0 and variance of V_RI. The two models can be compared for each age class by deviance information criteria (DIC) and other descriptors of model fit, such as two recently described R^2s for GLMMs [Nakagawa & Schielzeth, 2013]. The conditional (R^2_c) is the proportion of variation explained by the model fixed effects, while the marginal (R^2_m) is that explained by both fixed and random effects.

The MCMC simulations ran for 25,900,000 iterations with a burn-in of 900,000 and thin of 5,000 in order to retain 5,000 points for the posterior distributions. We used non-informative improper priors on the random effect variance components. Fixed effect priors were restricted to prevent jumping to high values as suggested by the package author [Hadfield, 2010]. The residual variance is unidentifiable in logit GLMMs and was fixed at 10. We used the variance component posterior distributions to calculate ratios to describe maternal effects and heritabilities (i.e., $h^2 = V_A/(V_A + V_M + 10 + \pi^2/3)$ and $m = V_M/(V_A + V_M + 10 + \pi^2/3)$, where 10 is the residual and $\pi^2/3$ is the distribution-specific link variance). Autocorrelations were low (<0.054) and effective sample sizes adequately high (>4,495) for describing the posteriors.

RESULTS

Generalized linear models for mortality in the 0–730 age class showed little influence of all predictors except hand rearing of infants. In the backward selection, this was the only predictor that caused an increase in BIC when it was dropped. It also appeared in all six of the best models by BIC (Tables II and III). Coefficients indicate that hand rearing substantially reduced infant mortality ($\beta = -1.561$). The other potentially important predictors were time period (higher mortality in pre-1985 period, $\beta = 0.500$), and region (lower mortality in North America, $\beta = -0.309$). Paternal age and hand rearing of the mother were significant only in the backward selection, and showed unexpected patterns with older fathers having lower rates of infant mortality (Fig. 2) and hand-reared mothers having lower rates of infant mortality ($\beta = -0.374$). Infant sex, inbreeding coefficient, and maternal parity were not significant predictors, though in all cases their coefficients were in the expected directions for higher mortality of males, inbred infants, and offspring born to primiparous mothers (Tables II and III, Fig. 2). Exploring the effects of hand rearing and infant sex in the smaller age classes reinforced these results (Table IV). Hand rearing substantially lowered mortality up to 6 months of age after which its effect diminished. In contrast, infant sex was never a significant predictor of mortality. There was only a subtle trend for higher male mortality initially followed by greater female mortality as infants aged (Table IV, Fig. 1).

In the two sets of GLMMs, posterior mean coefficients for the fixed effects were nearly identical between the rearing and genetic models, so we present only results from the genetic models (Table V). Hand rearing reduced mortality at all ages, though the difference failed to reach significance in the 1–14 and 183–730 day age classes. Despite the consistent influence of hand rearing, fixed effects accounted for little variation in infant death in either model set (R^2_m range 0.003–0.092, Table IV). With regards to exploring variance components and their ratios, we found DIC and R^2_c to indicate that GLMMs with a genetic variance component fit the data better (Table VI), so we focus on results from this model set. Overall, we found evidence of a substantial heritability of infant mortality and a clear maternal effect. These were both well above zero in the 0–730 day age class (h^2 posterior mode: 0.598 CI: 0.298–0.808; m posterior mode: 0.212 CI: 0.036–0.403). There was notable heterogeneity among the narrower age classes in the strength of these effects, with the highest heritability at day 0 and largest maternal effects in the 1–14 and 15–182 day age classes (Table VI). Variance components were consistent with these ratios showing a decline in V_A with age, and rise and fall in V_M with

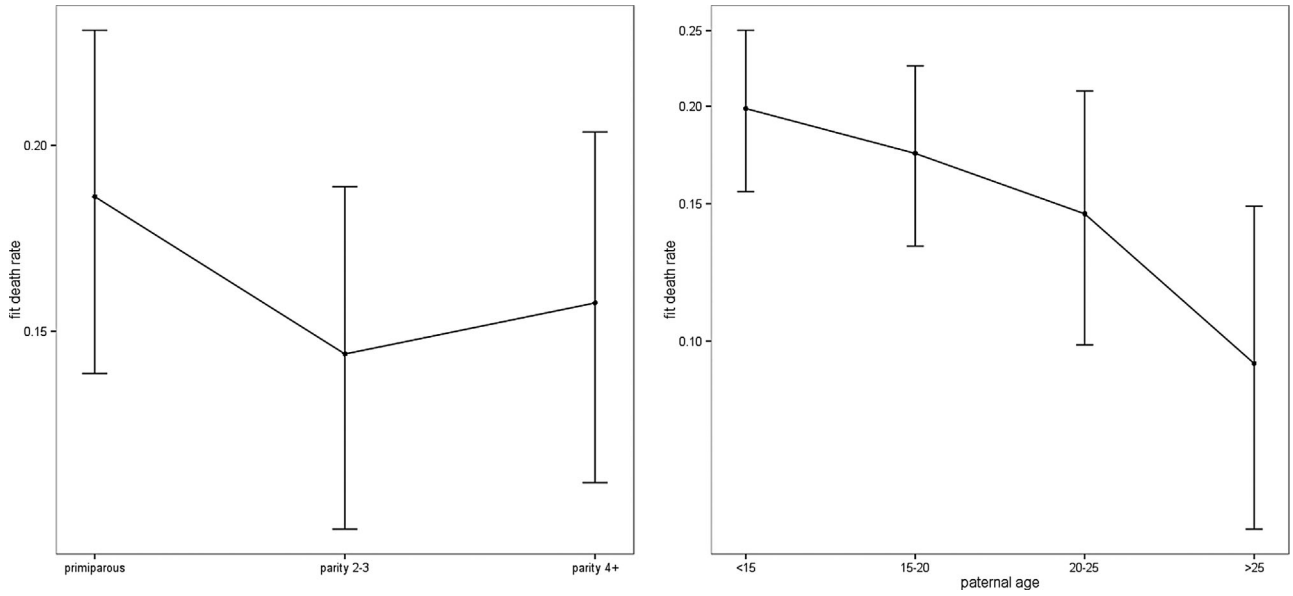

Fig. 2. Parity and paternal age effect plots from the full generalized linear model predicting death in the 0–730 day age class.

age. They also provided little evidence of consistent differences among zoos in mortality. The only age class in which the hand-rearing variance (V_{ZOO}) was potentially important was day 183–730 (Table VII).

DISCUSSION

The variance component ratios from this analysis highlight the importance of maternal effects and infant genetics to offspring survival early in life. Moreover, their influences change over time during infancy. Maternal effects influence variation in infant mortality the most during the first 6 months of life beginning the day after birth (Table VI, Fig. 3). Heritability is most important to survival on the day of birth, and heritable variation accounts for a surprisingly large component of gorilla mortality during infancy (Table VI, Fig. 3). Our results indicate that paternal age, parity, sex, time period, and zoo location are uninformative predictors of gorilla infant survival (Tables II and III). Interestingly, mortality risk is lower for hand-reared gorillas during the first 6 months of life, and infant rearing type is the only considered predictor variable with a significant impact on mortality (Tables II and III, Fig. 4). Our results detail the importance of genetics, maternal effects, and other variables of interest with regard to infant mortality, informing us of mortality risk factors across different time periods of gorilla infancy.

TABLE V. MCMCglmm Fixed Effect Posterior Means, 95% Credible Intervals and Significance Tests for Each Age Class

Age class (days)	β	CI	MCMC P
0			
Intercept	−7.860	(−10.169, −5.916)	<0.001
Hand-reared	−4.442	(−7.790, −1.357)	0.002
1–14			
Intercept	−8.482	(−10.964, −6.225)	<0.001
Hand-reared	−2.622	(−6.334, 0.546)	0.104
15–182			
Intercept	−7.882	(−10.245, −5.686)	<0.001
Hand-reared	−3.315	(−6.611, −0.315)	0.027
183–730			
Intercept	−8.736	(−11.185, −6.606)	<0.001
Hand-reared	−1.909	(−5.426, 1.083)	0.219
0–730			
Intercept	−4.110	(−6.441, −2.171)	<0.001
Hand-reared	−5.720	(−8.848, −2.783)	<0.001

Narrow-sense heritabilities provide insights regarding the evolutionary potential of the rate of infant survival [Houle, 1992]. 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 [Hansen et al., 2011; Wilson et al. 2005]. It is commonly thought that heritabilities of life history traits are low, resulting from strong selection eliminating deleterious alleles. However, our results indicate that heritable variation accounts for a large proportion of variation in gorilla mortality during the first 2 years of life. More specifically, they reveal that genetic effects are most important immediately following birth and decline over time (Table VI, Fig. 3). High mutational input of deleterious alleles affecting neonatal survival may be the cause of this heritability pattern. The rapid subsequent decline is likely caused by loss of the neonates carrying those alleles such that they do not enter later age classes. Although our results show a decline in genetic effect over time, the day 0–730 age

TABLE VI. Variance Component Ratio Posterior Modes With 95% Credible Intervals and Fit Statistics for the Two Model Sets

	Rearing model				Genetic model				
Age class (days)	m	R^2_m	R^2_c	DIC	h^2	m	R^2_m	R^2_c	DIC
0	0.042 (0.000–0.410)	0.075	0.503	213.651	0.368 (0.022–0.657)	0.030 (0.000–0.322)	0.092	0.729	182.726
1–14	0.571 (0.352–0.755)	0.003	0.691	226.638	0.109 (0.000–0.433)	0.507 (0.222–0.712)	0.006	0.770	205.543
15–182	0.473 (0.209–0.670)	0.010	0.575	222.647	0.133 (0.000–0.506)	0.363 (0.074–0.612)	0.034	0.723	201.624
183–730	0.164 (0.000–0.486)	0.004	0.570	132.092	0.063 (0.000–0.559)	0.053 (0.000–0.418)	0.004	0.687	120.937
0–730	0.393 (0.246–0.576)	0.042	0.526	605.447	0.598 (0.298–0.808)	0.212 (0.036–0.403)	0.084	0.885	402.162

TABLE VII. Variance Component Posterior Modes and 95% Credible Intervals for the Two Model Sets

	Rearing model		Genetic model		
Age class (days)	V_M	V_{ZOO}	V_A	V_M	V_{ZOO}
0	0.700 (0.000–9.221)	2.587 (0.006–29.737)	5.649 (0.002–29.963)	0.811 (0.001–11.227)	3.338 (0.000–40.908)
1–14	15.392 (4.784–35.833)	1.852 (0.002–24.274)	2.971 (0.001–25.492)	15.865 (3.965–43.716)	2.721 (0.000–33.721)
15–182	8.945 (2.229–23.359)	1.649 (0.002–22.334)	2.540 (0.005–26.460)	8.947 (0.469–28.874)	1.655 (0.003–24.303)
183–730	1.270 (0.002–12.543)	3.756 (0.003–39.666)	1.494 (0.002–20.993)	1.278 (0.000–12.988)	4.010 (0.011–45.563)
0–730	8.223 (3.758–17.048)	1.803 (0.000–11.102)	24.912 (4.170–124.524)	10.087 (0.467–49.144)	1.776 (0.002–27.248)

Residual variance is fixed at 10.

class reveals a substantial heritability (posterior mode 0.598, Table VI) higher than that of any of the smaller age classes. Partitioning these data into different age classes and running separate GLMMs shows how various effects change over time, but also treats mortality in each age model as a separate phenotype. Because of this, much of the data is ignored in smaller age class estimations as relationships between individuals in different classes are not represented. Thus, the effect estimates from the day 0–730 GLMM account for variation in mortality throughout the first 2 years of life as a whole, revealing a substantial heritability for gorilla infant mortality overall.

Our results indicate that the additive effect of genes plays a large role in gorilla infant mortality, but there are other types of genetic variation potentially influencing this life history trait. In addition to the additive genetic variance captured in narrow-sense heritability estimates, dominance

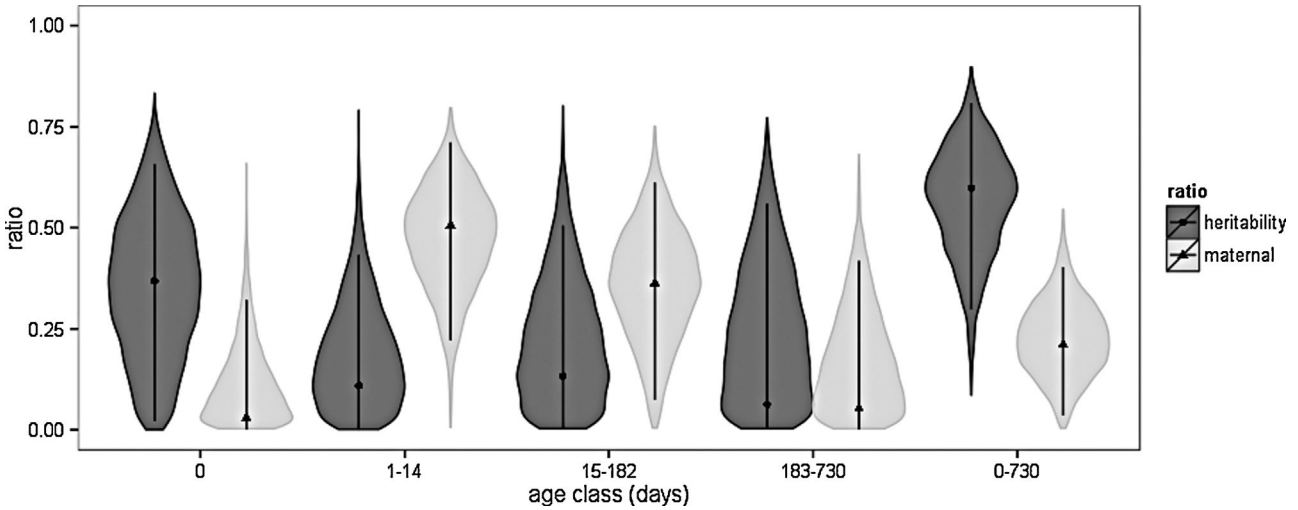

Fig. 3. Heritability and maternal identity ratios from the genetic models in each age class. Violins show the complete posterior distributions, with posterior modes and 95% credible intervals indicated by the dots and vertical lines.

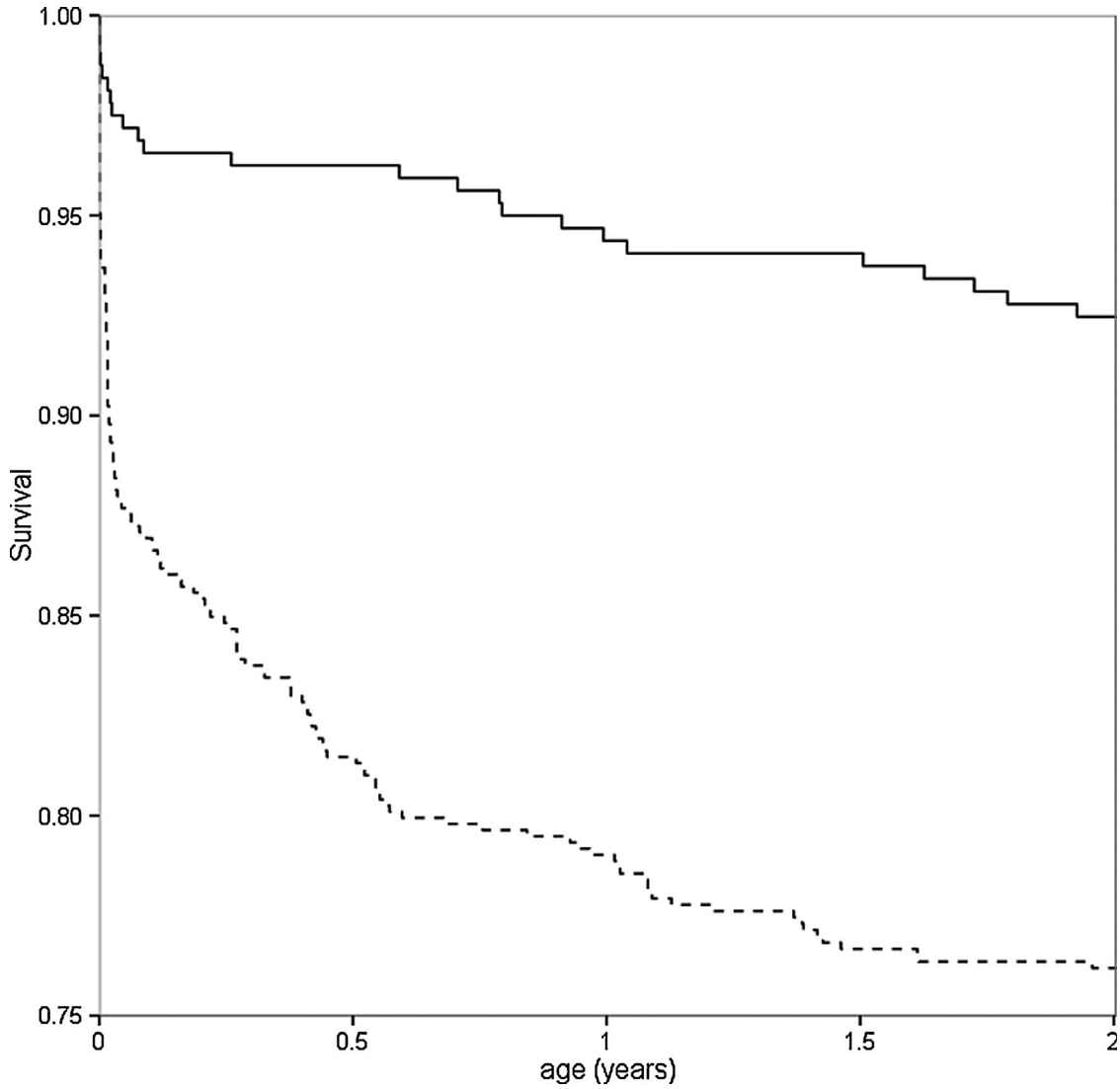

Fig. 4. Survivorship plot of hand-reared and mother-reared gorillas from 0–2 years (hand-reared solid, mother-reared dashed).

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. 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 I) 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].

Maternal effects also have evolutionary potential in species such as gorillas with large amounts of mother–offspring interactions. Our maternal effect estimates reveal that mothers are most important to offspring survival early in life, and are 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, 2004]. Thus, significant maternal effects on infant survival are unsurprising. Although we did not identify a sizeable maternal effect for survival on the day of birth, it often takes mothers 48–72 hr to display maternal behaviors such as nursing [Porton & Niebrugge, 2006]. Our results indicate that maternal effects decrease over the first year of life (Table VI, Fig. 3) just as mother–infant interactions have been recorded to steadily decrease over time and taper off [Nowell & Fletcher, 2007].

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. Difficult 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. Interestingly, gorilla mothers in captivity have reproductive parameters very similar to those observed in the wild [Knott, 2001]. 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, our results indicate that variation among mothers influences infant survival in captive settings, and observing maternal behavior toward offspring is crucial to survival during the first 6 months.

Our results indicate that infant rearing type is the only significant predictor of early life mortality in these gorilla data. Hand-reared infants have lower mortality than mother-reared gorillas for the first half year (Tables II and III, Fig. 4). This is surprising considering that much of the literature on rearing in captivity states that hand-reared animals often behave abnormally and face social and other difficulties [Ryan et al., 2002]. These studbook data suggest that hand rearing helps gorilla infants survive during the first 6 months. However, the marginal R^2 values (Table VI) indicate that this fixed effect accounts for less than 10% of the variation in gorilla infant mortality for all age classes modeled, whereas rearing type combined with additive genetic and maternal effects accounts for over 65% of variation in all models.

Model fit statistics demonstrate that many of the variables expected to influence gorilla infant mortality are insignificant in these data (Tables II–IV). The coefficients in Tables II and III indicate that time period and zoo region influence gorilla infant mortality such that infants were more likely to die before 1985 (the formation of the SSP), and gorilla infants survive better in North America. However, neither of these predictors is significant. Paternal age and mother's rearing type appear to influence mortality only in the backward selection model, and both of these variables produce unexpected results. Contrary to studies which report that hand-reared gorillas have less reproductive success [Ryan et al., 2002], the offspring of hand-reared mothers appear to survive better in these data. Advanced paternal age does not indicate higher infant mortality risk in these data as predicted by some germ cell line research [Momand et al., 2013]. This is also surprising given that gorillas age more quickly than humans. Selection for high quality sperm, aggression, and other costly reproductive benefits have tradeoffs regarding aging and longevity [Promislow, 2003], but our results do not clearly represent such paternal age trends. One potential explanation for the lack of association between advanced paternal age and infant mortality relates to viability selection and its fitness implications. Although older males have acquired more germ line mutations, they have also survived more viability selection than younger males [Hansen & Price, 1995]. It may also be that our oldest age group of gorilla males is not comparable to elderly men in which elevated levels of germ line mutations have been documented. Additionally, gorilla mating patterns and the relative lack of male competition may decrease sperm competition and mutation rates compared to those observed in other species [Venn et al., 2014].

Maternal parity, infant sex, and inbreeding did not contribute to infant mortality in a measurable way (Tables II and III). These predictors had unexpectedly small influences on this life history trait, but all show coefficients of effects in expected directions with offspring of primiparous mothers, males, and inbred individuals having higher mortality risk. The lack of sex effect is perhaps the most surprising. Hypotheses based on mate choice and sexual selection make predictions about mortality differences between sexes. The general increased longevity of female mammals is thought to be influenced by reproductive consequences, and increased male mortality is hypothesized to result from aggression and male competition for mates [Promislow, 2003]. However, our results do not reveal that being male increases mortality risk as suggested by general life history trends [Kruger & Nesse, 2006]. This indicates that sexual selection and competition have not produced sex specific differences regarding early life mortality in gorillas. This explanation is similar to that offered for the lack of paternal age effects, and it may be that relatively low competition between males in gorilla mating reduces sex differences in comparison to other species. Regardless of the underlying reasons, many predictor variables of interest lack any significant impact on gorilla infant mortality in these data.

Mortality in western lowland gorillas shows strong heritability during the first 2 years of life, substantial maternal effects that decrease over time, and survival benefits from hand rearing. Additive genetic effects are strongest just after birth, and maternal effects and hand rearing are important to survival during the first 6 months of life. Many variables expected to influence infant mortality (paternal age, sex, etc.) have no clear effect on this trait. These results have interesting implications for conservation and captive gorilla programs, indicating that infant mortality could benefit from further research focused on genetics, maternal traits and

behaviors, and hand-rearing practices. While this study identified these factors as important to gorilla survival early in life, the specific mechanisms by which they influence mortality warrant further investigation. Overall, there is still variation in gorilla mortality to be explored in hopes of improving survival in this critically endangered species, but data from the international studbook for the western lowland gorilla [Wilms, 2011] allow us to analyze the impacts of genetic, maternal, and various other effects throughout infancy.

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