Higher offspring mortality with short interbirth intervals in free-ranging rhesus macaques

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Short birth intervals have long been linked to adverse child outcomes in humans. However, it remains unclear the extent to which the birth interval has a direct influence on offspring mortality, independent of the confounding effects of modern environments and human sociocultural practices on reproductive behavior. Outside of humans, the relationship between birth intervals and offspring mortality has been rarely tested, leaving an open question of how much the findings from humans imply evolutionarily conserved mechanisms. Here, using ∼9,000 birth records from ∼1,400 free-ranging rhesus macaque mothers, we show that short birth intervals preceding or succeeding the birth of an offspring are both associated with higher offspring mortality, after controlling for heterogeneity across mothers and birth cohorts. We clarify that the mortality risk of a short birth interval to an offspring is contingent on the survival of its older or younger sibling, the condition that reduces maternal resources for investment in the offspring. This finding suggests that life-history tradeoffs between offspring quantity (a short birth interval) and quality (offspring survival) form an evolutionary force shaping variation in birth intervals. Consistent with the well-known observation made in humans, we also found a nonlinear relationship between the preceding interbirth interval and infant mortality. The overall congruence with the findings from the human literature indicates a robust relationship between birth intervals and offspring mortality.

birth interval | offspring mortality | rhesus macaques | life-history tradeoffs | maternal investment

Short birth intervals, or short lengths of time between pregnancies or births, have been linked to adverse birth outcomes in humans (1–3). Studies of the relationship between birth intervals and infant mortality have been encouraged across disciplines, in the hope of improving policy advice for family planning (4). To attain this goal, and to facilitate research in reproductive decisions at both proximate and ultimate levels, nonhuman primate models can be highly informative, due to their closeness to humans in phylogeny, physiology, neuroanatomy, and their exhibition of complex and differentiated social relationships (5). However, there are few data available outside of humans to assess the mortality risks associated with short birth intervals, leaving the question of how much the birth interval–mortality relationship observed in humans (1, 6, 7) is derived, or represents a more general pattern of primate reproduction. In this study, we present data from a population of free-ranging rhesus macaques (*Macaca mulatta*), and seek to better understand the biological link between birth interval and offspring mortality in a general evolutionary framework.

One evolutionary model for understanding variable patterns of reproduction has been the offspring quantity and quality tradeoff. The model posits that investment in offspring number utilizes finite resources that would be otherwise available for investment in offspring quality, namely a trait correlated with fitness measures such as survival (8–10). This tradeoff has been examined across taxa, mostly focusing on the resources allocated to multiple offspring born within a reproductive attempt (11–16). Relatively less is known about the quantity and quality tradeoff occurring across reproductive attempts. Such tradeoffs are likely to be salient in many large-bodied mammals, who mostly give birth to singletons at multiple reproductions spread over a long lifespan. Here, the birth interval, usually calculated as the interval between live births in the nonhuman literature (interbirth interval; IBI), is one determinant of the total number of offspring (17). The tradeoff hypothesis predicts that investment in offspring number via short IBIs creates limits on the maternal resources available for either or both of the offspring comprising that birth interval (Fig. 1).

The tradeoff between IBIs and offspring quality is likely to have been an important force shaping the life history in primates. Compared with other mammals of similar body size, primates live long and exhibit prolonged offspring care at each reproduction (18), during which maternal effects on offspring quality including survival are common and strong (19, 20). This life-history context, coupled with an absence of some derived human traits that aid maternal reproductive effort such as nutritional provisioning by nonmothers (21), may place a high pressure on nonhuman primate mothers for strategic spacing of births for optimal birth outcomes. However, outside of humans, there are few studies explicitly testing the tradeoff between IBI and offspring quality (22). The free-ranging rhesus macaques on Cayo Santiago Island, Puerto Rico, provide a unique opportunity to conduct such a study because, similar to contemporary human societies, the risks of predation and starvation are reduced, while the modern interventions that often confound the relationship

Significance

Despite the extensive body of research showing a relationship between short birth intervals and high offspring mortality, we lack studies outside of humans, especially in nonhuman primates. Filling this gap can help clarify the biological link between birth intervals and offspring mortality in a general evolutionary framework. Using longitudinal data collected from free-ranging rhesus macaques, we show that the risk of a short birth interval to an offspring is contingent on the survival of its older or younger sibling, who may constrain the maternal resources available for the offspring. This finding suggests the tradeoff in maternal resources between offspring quantity (a short birth interval) and quality (offspring survival) as an evolutionary force shaping the variation of birth intervals.

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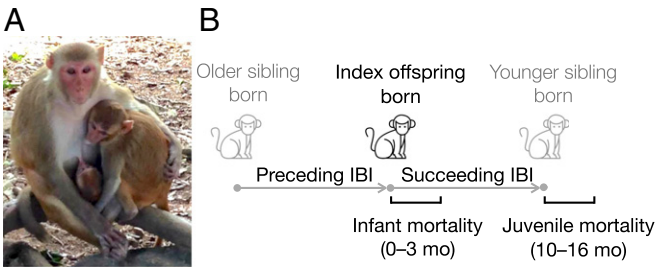

Fig. 1. (*A*) Rhesus macaque mothers may be nursing two overlapping offspring on Cayo Santiago. (*B*) The degree of tradeoffs in maternal resources that need to be divided between two offspring may be reflected in an IBI. From an offspring's perspective, there are two types of IBI: one between their birth and that of their older sibling (preceding IBI), and the other between their birth and that of their younger sibling (succeeding IBI).

among reproductive outcomes in humans, such as access to medical resources and birth control, are absent.

Macaques are among the best-studied nonhuman primates, especially as a biomedical model for human biology (5, 23–25). Although the reproductive synchrony that is often regulated at yearly cycles in rhesus macaques is different from year-round reproduction in humans (26), some key functions mediating reproductive transitions share a high degree of homology with women, allowing for laboratory research in female rhesus macaques to produce translational data crucial for understanding human reproductive diseases (23, 27). The vast majority (85%) of rhesus macaque IBIs result from reproduction across consecutive years, and they distribute in a bell shape (*SI Appendix*, Fig. S1), similar to human IBIs (28). Longer IBIs outside of this range are constrained by reproductive synchrony because birth skipping, or missing the chance of reproduction in a given year, increases an IBI by a year. Within consecutive-year IBIs, variation in IBI can either increase or decrease the chance of reproducing in the next year: Short IBIs place current reproduction in the earlier period of a birth season (*SI Appendix*, Fig. S2 and Table S2), thereby allowing enough time for a female to resume reproduction and consequently increases the chance of conception during the upcoming mating season (29, 30). As such, consecutive-year IBIs in rhesus macaques provide an ecological context to test if the chance of increasing offspring numbers is traded off with offspring quality—the reproductive decision common to all species who reproduce more than once in a lifetime.

The present study tested the offspring quantity and quality tradeoff after accounting for heterogeneities in mortality risks using mixed-effects models. The average number of lifetime offspring for females who ever reproduce is 7.1 in rhesus macaques, and our longitudinal data allow for comparison within multiple birth records of the same mothers. We predicted (*i*) higher infant mortality after short IBIs preceding the birth of an index offspring (preceding IBI; P-IBI); and (*ii*) higher juvenile mortality after short IBIs succeeding the birth of an index offspring (succeeding IBI; S-IBI). However, short IBI is no longer predicted to compromise offspring quality if the adjacent sibling (older for P-IBI and younger for S-IBI) dies prematurely (31, 32), because the death may alleviate the constraint on maternal resources. By investigating the interaction between the survival status of siblings and IBI, we also sought to addresses an alternative hypothesis that older sibling death confounds the relationship between P-IBI and infant mortality (33, 34), because the premature death of the older sibling is associated not only with a shorter time to the birth of the index offspring (i.e., its P-IBI), but also with higher mortality of the index offspring via death clustering among adjacent pairs of siblings (35–37).

Methods

Study Population. Cayo Santiago, a 15.2-ha island located 1 km off the southeastern coast of Puerto Rico, is currently inhabited by ∼1,500 rhesus macaques who descended (since 1938) from 409 monkeys of Indian origin (38). High-protein commercial monkey chow is provided daily, but the monkeys spend ∼50% of feeding time foraging on natural vegetation (39), and water collected from catching rain is distributed at water stations across the island ad libitum. The monkeys live in naturally formed social groups with evidence of dissociative mating producing genetic outbreeding (40). Rhesus macaques form multimale and multifemale groups and mate with multiple partners (41). All individuals are identified, and demographic information is recorded prospectively from birth to death through daily monitoring (Monday through Friday) by the census team at the Caribbean Primate Research Center (CPRC). Infant deaths are reliably recorded, because mothers tend to carry a dead infant for several days following death. Research permission was provided by the Institutional Animal Care and Use Committee of the University of Puerto Rico (A1500116) and New York University Animal Welfare Committee (14-1439).

Selection of Variables and Statistics. Offspring mortality was the response variable for all analyses. We first calculated age-specific mortality at monthly intervals for prereproductive ages (0 to 3 y old), using the Kaplan–Meier method that accounts for censoring of death (42). Mortality was highest in the first month postpartum and decreased until 3 mo postpartum, after which it stayed lower until 10 mo postpartum. As such, we used 3 mo postpartum as a cutoff for defining infant mortality and premature death of siblings. Mortality increases again during 10 to 16 mo postpartum, which coincides with the typical period during which the younger sibling is born and reaches its 6 mo postpartum. To test if such an increase in mortality and younger sibling birth is related, we defined juvenile mortality as death during the 6 mo since the birth of younger sibling and assessed those who survived at least until the birth of a younger sibling. We used generalized linear mixed-effects models (GLMMs) with a logit link function for a binary outcome of death and with a pair of random effects, maternal ID and birth cohort, based on previous reports that early mortalities on Cayo Santiago exhibit heterogeneities by maternal backgrounds and birth cohorts (20, 43). The random effect of maternal ID estimates the variance in offspring mortality explained by unobserved maternal characteristics that equally influence all siblings, such as maternal genetics, mothering style, or a stable environment in which a mother lives. As such, the random effect of maternal ID addresses the offspring mortality risks that are correlated within mothers.

We ran two GLMMs, for testing the relationships between P-IBI and infant mortality (prediction *i*) and between S-IBI and juvenile mortality (prediction *ii*). We defined IBI as the difference in days between the live births of offspring who were born across consecutive years. Rhesus macaques exhibit reproductive synchrony, whereby breeding and mating alternate at ∼6-mo intervals and each peaks during a concentrated 3-mo period (43). Consequently, ∼15% of mothers each year experience birth skipping, or failing to give birth during a given birth season, and their associated IBIs are longer by a year on average compared with those resulting from consecutive-year births (mean 716 vs. 367 d). In addition to causing the noncontinuous increase for longer IBIs, birth skipping also tends to be more frequent among mothers in suboptimal reproductive condition in rhesus macaques, such as first-time or older mothers (44, 45), whose offspring mortality may be higher, independent of IBI (20). We thus restricted our main analyses to consecutive-year IBIs to examine the distribution of IBIs undisturbed by birth skipping, and to reduce possible selection bias due to heterogeneity in maternal conditions that increase the risk of birth skipping. Nonetheless, for completeness, we included analyses using the entire range of IBIs, namely including IBIs resulting from birth skipping (*SI Appendix*, Table S3). For the main analyses, we divided IBIs into five equal-frequency length categories at every 20th quantile (150 to 343, 344 to 360, 361 to 377, 378 to 398, and 399 to 530 d), to align with the convention in the human literature where categorical IBIs are used to identify the range of IBIs with heightened risk of adverse birth outcomes. In addition to the main analyses, we conducted two supplemental analyses. First, we used conditional logistic regression models to make sure that the autocorrelation structure in offspring mortality is adequately controlled for (*SI Appendix*, Table S4). Second, we used the same GLMMs but replaced categorical IBI variables with IBI as a continuous variable (*SI Appendix*, Fig. S5 and Table S5). The two sets of analyses confirm that the shape and significance of the relationship between IBI and offspring mortality are overall consistent with the GLMMs using categorical IBI. For datasets used in all of these analyses, please see Lee et al. (46).

For the main analyses, we statistically controlled in each model for the following variables that may influence offspring mortality in rhesus macaques:

birth timing (number of days relative to the median birth date within a birth season); sex of offspring; maternal age (linear and quadratic); and maternal dominance rank (20, 47, 48). As in many Old World monkeys, rhesus macaques also form highly differentiated social relationships which have fitness consequences (49). One important axis of social relationships in primates is dominance rank, and females of macaque species form stable linear hierarchies of social rank and inherit rank position along matrilines which are defined as descendants of a crown female ancestor (50, 51). The current CPRC demographic database contains categorical rank data (high/middle/low) based on original data provided from Donald S. Sade, Northwestern University, Evanston, IL, and John D. Berard, Department of Veterans Affairs, Greater Los Angeles, North Hills, CA, who assessed the dominance position of a matriline yearly during 1960 to 2000 based on pairwise agonistic interaction data (51). In the Cayo Santiago population, differences in dominance rank confer fertility advantages little via birth spacing but mainly via earlier onset of reproduction and higher offspring survival rate in higher-ranking females (47, 52).

All data (total 9,088 offspring and 1,429 mothers) were obtained from the CPRC demographic database. To avoid the possibility that unmeasured factors leading to maternal death affect both IBI and offspring mortality, we excluded any offspring whose mother died during the period considered for offspring mortality. Because P-IBI by definition requires the occurrence of a previous birth, all first-born offspring were automatically excluded from the analysis of infant mortality. We further excluded the small number of offspring whose sex was not known, or with IBIs that were abnormally short, which were presumably data errors as they are shorter than the minimum viable gestation length (133 d) of rhesus macaques (53). Final sample sizes for the main analyses on consecutive-year IBI comprised 6,473 offspring from 1,314 mothers for infant mortality, and 5,452 offspring from 1,265 mothers for juvenile mortality, who were born across 57 birth cohorts between 1962 and 2017. Because dominance-rank data were only available for a subset of offspring (3,494 for infant mortality and 3,250 for juvenile mortality), we present results based on analyses conducted without the dominance-rank variable, as results with the rank variable produce consistent effect size estimates but with their SEs larger due to smaller sample size (*SI Appendix*, Fig. S6 and Table S6).

Analysis. We estimated the distributions of fixed-effect coefficients and variance components for the random effects using the Bayesian Markov chain Monte Carlo technique available via functions in the "MCMCglmm" package (54) in R version 3.4.2 (R Development Core Team, 2017) (55). We used uninformative mean-zero Gaussian priors for all fixed effects. An inverse-Wishart distribution was specified for the two variance components and residual variance. The first variance component for birth cohort was specified as a nonzero moderate estimate by setting the variance mean to 1 and its scale parameter to be 0.5, which is larger than the default 0.02, following previous research showing fluctuations in IBI according to birth cohorts (43). The second variance component for maternal identity was specified at a smaller scale parameter of 0.05, following a previous finding of low heritability of IBIs (56). Finally, the prior for the residuals was fixed at 1 with the degree of belief 1, because residual variance cannot be estimated for binary outcomes but is determined by the mean. To facilitate chain mixing, we used parameter-expanded priors for variance components, by specifying prior means as 0 and the prior covariance matrix as 1,000. The number of burn-in chains and the thinning interval were set at 3,000 and 100, respectively, and the number of iterations was set between 1 and 2 million depending on the available sample size, to ensure that effective sample sizes obtained for the posterior distributions of coefficients are higher than 5,000. Autocorrelations among sampled iterations were all below 0.01. Convergence of models was assessed by testing multiple chains with the Gelman–Rubin criterion of 1. Finally, we conducted a model comparison between models with and without the IBI variables included, using deviance information criteria (DIC), which is a Bayesian method for model comparison. For both models on infant mortality and juvenile mortality, models with IBI variables exhibited lower DIC by 29 and 14, respectively.

Since coefficients are logit-transformed in logistic regression, we transformed estimated coefficients to the metric of probability. For each model output, the intercept estimates represent the mortality of a short IBI, which is set as the shortest IBI category (150 to 343 d). Estimates of other coefficients represent how much change in mortality from the short IBI is associated with the unit change in each variable for longer IBI. We used MCMC sampling distributions to obtain the distribution of differences in the probability of death and calculated its middle 95% range as the credible interval. We determined the significance of a difference by assessing whether the credible interval includes zero.

Results

Preceding IBI and Infant Mortality. A short P-IBI was associated with high offspring mortality risk, if the older sibling did not die prematurely (Fig. 2*A* and Table 1). The probability of death for infant mortality was significantly higher in the shortest-length P-IBI category, compared with the mortality seen in longer birth intervals of the middle-length categories. Mortality did not differ significantly between the shortest and the longest P-IBI category, such that P-IBI and infant mortality form a quadratic relationship, strongly resembling that seen in humans (7, 57). The quadratic relationship was also supported in the supplemental analyses using continuous IBI (*SI Appendix*, Fig. S5). However, if the older sibling prematurely died, the relative increase in infant mortality for the shortest P-IBI category was not present (Fig. 3, gray lines). The shortest P-IBI category instead imposed lower infant mortality risk compared with the remaining longer-length categories. The high infant mortality estimates for longer IBIs may explain the significant increase in average infant mortality (0.32 [0.15 to 0.4]) for those whose older sibling died prematurely.

Succeeding IBI and Juvenile Mortality. Similar to P-IBI, a short S-IBI was associated with higher offspring mortality, in this case of juveniles, if the younger sibling did not prematurely die (Fig. 2*B*

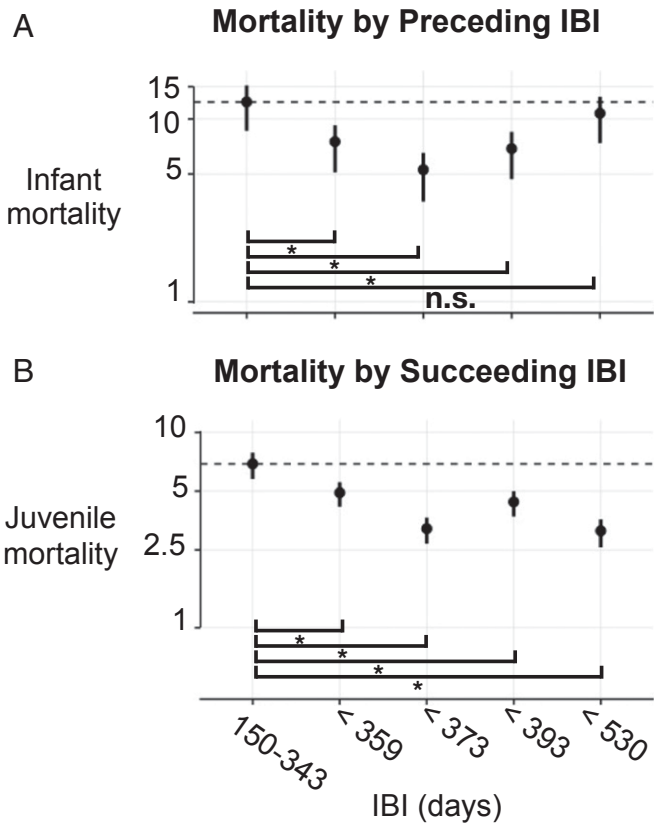

Fig. 2. Infant mortality (*A*; $n = 5{,}967$) and juvenile mortality (*B*; $n = 5{,}104$) associated with consecutive-year IBI, for offspring whose older and younger sibling, respectively, did not die prematurely. Mortality is compared with that of short IBI, which is set at the shortest IBI category (150 to 343 d; dotted horizontal lines). Each vertical line spans the interquartile range (IQR; middle 50%) of estimated mortality based on posterior distributions. An asterisk denotes nonzero difference (i.e., the 95% credible interval for difference does not include zero) in mortality compared with that of the shortest IBI category, whereas "n.s." denotes nonsignificant difference. Note that the *y* axis is drawn in log scale, which affects the appearance of the degree of overlap between vertical lines.

Table 1. Estimated difference in mortality between short and longer IBIs, among IBIs resulting from consecutive-year births

	Short IBI	Longer IBI			
Predictor	150–343 d	<359 d	<373 d	<393 d	<530 d
Preceding IBI					
Older sibling					
Survived	0.13* [0.07 to 0.18]	**−0.06 [−0.11 to −0.01]**	**−0.07 [−0.15 to −0.02]**	**−0.06 [−0.13 to −0.01]**	−0.02 [−0.08 to 0.03]
Died	0.11* [0.06 to 0.16]	0.15 [−0.03 to 0.43]	**0.43 [0.14 – 0.71]**	**0.26 [0 to 0.59]**	**0.26 [0 to 0.59]**
Succeeding IBI					
Younger sibling					
Survived	0.07† [0.04 to 0.1]	**−0.02 [−0.05 to 0]**	**−0.04 [−0.06 to −0.02]**	**−0.02 [−0.05 to 0]**	**−0.04 [−0.07 to −0.02]**
Died	0.07† [0.02 to 0.14]	−0.004 [−0.11 to 0.15]	0.03 [−0.1 to 0.23]	0.05 [−0.08 to 0.28]	−0.03 [−0.12 to 0.09]

Estimates are bolded if 95% credible intervals do not include zero. The estimates are calculated based on the MCMC GLMM outputs (*SI Appendix*, Table S7). Longer IBI mortality difference is compared with short IBI.
*Infant mortality.
†Juvenile mortality.

and Table 1). The probability of death for juvenile mortality was highest at the shortest S-IBI category and reduced for all of the remaining categories of longer S-IBI categories. Unlike in P-IBI, juvenile mortality did not differ by IBI lengths if the younger sibling died prematurely.

Discussion

Consistent with the extensive literature on the mortality risk of short birth intervals in humans, we found that short IBIs are associated with high offspring mortality in free-ranging rhesus macaques. The mortality risk of a short IBI manifests in both directions of IBI, that is, via IBIs that precede and succeed the index offspring. As such, a short P-IBI was associated with higher infant mortality, and short S-IBI with higher juvenile mortality. The finding for S-IBI augments a relatively small body of human literature indicating the additional risks of short IBI in older siblings (57–60), and thus expands the general understanding of the risks associated with short birth intervals.

Corresponding to the nonlinear relationship between the preceding birth interval and infant mortality that is well-known from human data, mortality risk was similarly higher for the longest P-IBI category as for the shortest P-IBI category (7, 61). This pattern contrasts with S-IBI, where juvenile mortality exhibits a nearly linear decrease as IBI increases (cf. Fig. 2*B*). The difference in the IBI–mortality relationship may reflect different pathways in which IBI contributes to offspring outcomes. For an interval preceding the index offspring, mothers may face tradeoffs in their physiological resources (62, 63), which are critical for optimal birth outcomes for the index offspring during conception and gestation. Too short an interval could mean limited time for a mother to restore the physiological conditions depleted from previous reproduction. However, the similarly adverse outcomes for the longest P-IBI category may not be driven by maternal depletion but rather by poor maternal condition, such as lower fertility or illness, which simultaneously delay reproduction and compromise offspring quality. For S-IBI, which is an interval after an index offspring is born, actual maternal care rather than physiological conditions of the mother may be the limiting factor. Because maternal care only becomes limited when the younger sibling is born, index offspring may benefit from a longer interval until the birth of the sibling.

The present study provides evidence in support of the offspring quantity and quality tradeoffs, by showing that survival of an older or younger sibling is the main context in which a short IBI is detrimental. Life-history tradeoff has been difficult to detect at a phenotypic level due to confounding variables (64–66). Taking advantage of a large dataset, we sought to statistically control for maternal heterogeneity and address potential environmental confounding by maternal rank. Our finding of the negative correlation between IBIs and offspring mortality, which are unlikely to be genetically correlated due to their very low and moderate heritability, respectively (20, 67), suggests instead that not all individual reproductive strategies may be optimized. For P-IBI, if the previous offspring died early, the same length of short IBI means a relatively longer time for the mother to recuperate compared with the mother whose offspring survived. This may explain why in such contexts a short P-IBI no longer confers higher infant mortality compared with longer P-IBI, similar to findings obtained from human data (37, 68–70). For S-IBI, a short interval means that the offspring is still relatively young when its younger sibling was born. Although rhesus macaques are weaned within a year, they receive significant amounts of

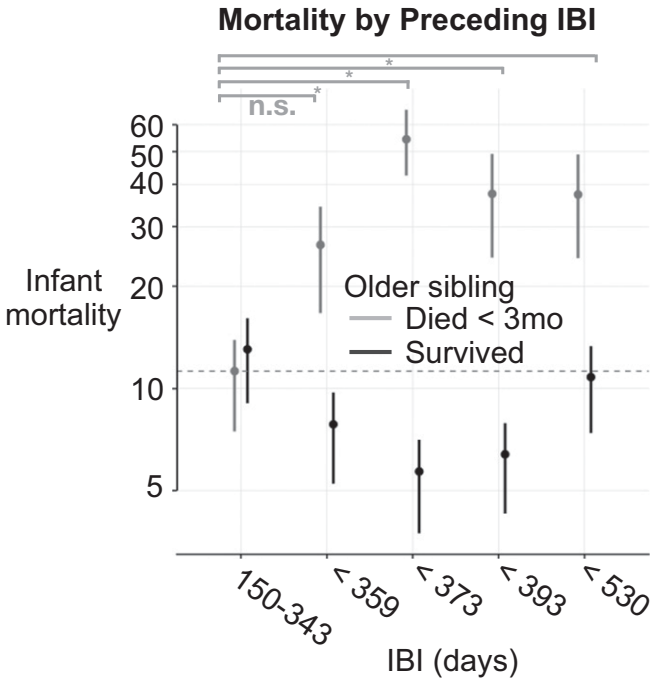

Fig. 3. Infant mortality by consecutive-year IBI, according to whether the older sibling prematurely died (gray lines; $n = 506$) or not (black lines; $n = 5{,}967$). Each vertical line spans IQR of estimated mortality based on posterior distributions. An asterisk denotes nonzero difference (i.e., the 95% credible interval for difference does not include zero) in mortality compared with that of the shortest IBI category (dotted horizontal line), whereas "n.s." denotes nonsignificant difference. Note that the *y* axis is drawn in log scale, which affects the appearance of the degree of overlap between vertical lines.

maternal care up until the birth of their younger sibling (71, 72), after which they continue to stay dependent on maternal care (73). In this context, if the index offspring is relatively young when their sibling is born, it is likely to still require maternal resources for which it must now compete with the newborn. As maternal care will prioritize the newborn, this transition is likely to be a stressful event for the index offspring, who as a consequence might experience increased mortality risk (20).

Our finding does not support the hypothesis that the relationship between short P-IBI and high infant mortality is due to the confounding effect of older sibling death (33, 74, 75). Under this hypothesis, the higher infant mortality after a short P-IBI is not attributable to the IBI per se, but to the overrepresentation of offspring whose older sibling has prematurely died, because older sibling death is associated with both shorter P-IBI and higher mortality for the current infant. However, our findings indicate that in rhesus macaques, the link between short P-IBI and higher infant mortality is present only if the older sibling has not died prematurely. In line with what is known in humans, we found that premature death of the older sibling is associated with both an overall increase in infant mortality of the index offspring, suggesting death clustering among adjacent siblings (36, 37), and a shorter P-IBI for the index offspring on average by 25 d. Importantly, however, the two observations appeared unrelated, because the higher infant mortality was evident not in the shortest IBI category but in the longer IBI categories for P-IBI. Here, the risk of death was significantly higher for longer P-IBI compared with that of the shortest P-IBI, contrary to what would be predicted by the confounding by sibling death.

The overall congruence between humans and rhesus macaques in the IBI–mortality relationship brings us a step closer to understanding the biological links between birth interval and offspring mortality. At a broader theoretical level, our work demonstrates the utility of life-history theory for understanding variation in human reproduction (76–78). For example, based on a single study, current World Health Organization guidelines advise delaying the next pregnancy after miscarriage (4). However, more recent studies conducted in humans, consistent with the present study, suggest that a delay in the birth interval may be actually more detrimental under circumstances in which a previous child has prematurely died (79, 80). These findings are consistent with the general evolutionary framework underlying the offspring quantity and quality tradeoff, that optimal birth intervals would maximize maternal reproductive success. Within this view, faster reproductive transition after the loss of recent offspring is predicted to be an adaptive strategy for increasing offspring number, as has been reported across taxa (81, 82) and various human populations (31, 32). More practically, given the well-described similarity of female rhesus macaques to humans in endocrine regulation of reproductive function (5, 23–25, 27), our findings could be readily followed up in future experimental studies on captive rhesus macaques to further clarify the physiological mechanisms by which birth interval influences offspring quality during conception and gestation. Such improved understanding of mechanisms could help resolve, at least in part, the ongoing debate in the human literature on the extent to which the birth interval has a direct influence on offspring mortality (3, 33, 83, 84).

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