

1 **Title:** Early life adversity and adult social relationships have independent effects on survival in a
2 wild animal model of aging

3 **Short title:** Early life, social behavior, and survival
4

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21 **Abstract**

22 Does social isolation in adulthood predict survival because socially isolated individuals
23 are already unhealthy due to adversity earlier in life (health selection)? Or do adult social
24 environments directly cause poor health and increased mortality risk (“social causation”)?
25 These alternative hypotheses are difficult to disentangle in humans because prospective
26 data on survival and the environment for both early life and adulthood are rarely
27 available. Using data from the baboon population of Amboseli, Kenya, a model for
28 human behavior and aging, we show that early adversity and adult social isolation
29 contribute independently to reduced adult survival, in support of both health selection and
30 social causation. Further, strong social bonds and high social status can buffer some
31 negative effects of early adversity on survival. These results support a growing change in
32 perspective, away from “either-or” hypotheses and towards a multi-causal perspective
33 that points to multiple opportunities to mitigate the effects of social adversity.

34

35 **Teaser**

36 Early life environments and adult social bonds have strong, but largely independent
37 effects on survival in wild baboons.

38 Introduction

39 In humans and other animals, the experience of harsh conditions in early life can have
40 profound effects on adult health and survival (1-5). For example, one recent study found that
41 American children who experience more than three sources of socioenvironmental adversity
42 before age 18 can expect a 9.5 year reduction in quality-adjusted adult life expectancy (1).
43 Importantly, adversity during early life is also linked to social adversity in adulthood, including
44 both low socioeconomic status (SES) and challenges in forming strong and supportive social
45 relationships (6-8). In turn, low SES and social isolation/low social support are linked to poor
46 health and all-cause mortality (9-12). However, while early life adversity, poor adult social
47 relationships, and low adult social status have all been linked to poor adult survival, the causal
48 relationships between these factors are not well understood.

49 Specifically, while most hypotheses acknowledge that early life environments can affect
50 both adult health and the adult social environment (Figure 1; 13, 14, competing hypotheses differ
51 in the extent to which they identify adult health (driven by early adversity) as the cause or
52 consequence of differences in adult social relationships. For instance, the health selection
53 hypothesis posits that poor health status affects the adult social environment, preventing
54 attainment of high social (or socioeconomic) status and compromising the formation of strong
55 social relationships (15; Figure 1A). Under this scenario, poor health—arising from early life
56 adversity or some other source—is the primary cause of both adverse social environments and
57 poor health/survival in adulthood. Alternatively, the social causation hypothesis posits that social
58 isolation and low social status in adulthood play a direct, causal role in the connection between
59 the social environment, health, and survival (10, 16). Under this scenario, poor social
60 relationships and/or low social status in adulthood are sufficient to trigger poor health/survival in
61 adulthood (Figure 1B). Thus, while early life adversity may also contribute to variation in health
62 and/or the social environment, enhancements to the social environment in adulthood are viable
63 paths to improving adult health and lifespan. The causal effects of adult social environments may
64 act in parallel to the effects of early adversity or function as a source of resilience against the
65 costs of early adversity (i.e., the “social buffering” hypothesis; 17-19).

66 Distinguishing between health selection and social causation is important to both
67 evolutionary biologists and social scientists. Understanding what forces drive variation in
68 survival helps to identify the traits targeted by natural selection, shedding light on the
69 evolutionary underpinnings of early life effects and sociality. At the same time, understanding
70 the causes of variation in health and mortality can inform investment in public health
71 interventions and policy. However, despite extensive research on the pathways linking early
72 experience and adult life outcomes, the relative importance of health selection versus social
73 causation in explaining social environmental effects on adult survival is widely debated (13, 20-
74 24).

75 To address this debate, the best approach is to link prospectively collected data on early
76 life adversity and prospectively collected information on the adult social environment and
77 survival in the same individuals (20, 25). Existing data typically do not permit such analyses in
78 human populations, but appropriate data are sometimes available for wild animal populations
79 that have been under continuous observation for many years (9). Further, the social determinants
80 of health in many social mammal species resemble those described in humans, making wild
81 animal models a useful tool for dissecting the relationships among early life adversity, adult

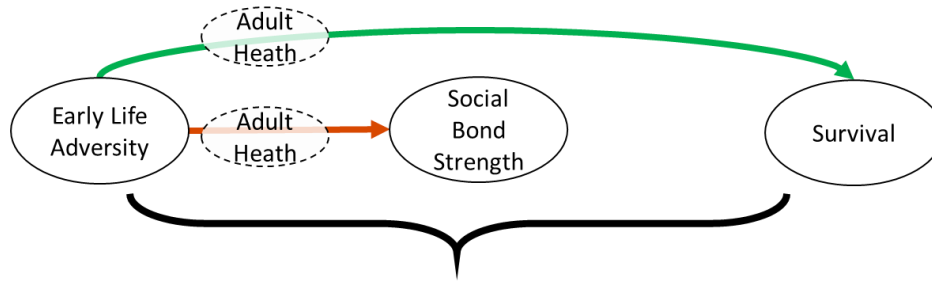
82 social behavior, and lifespan. For example, in several nonhuman mammals, early life adversity is
83 linked to low adult social status or weak adult social relationships (26-29). Similarly, low social
84 status or weak social relationships are associated with higher mortality rates in a range of social
85 mammal species (9).

86 In this study, we use a mediation analysis framework to examine the relationships among
87 early life adversity, adult social behavior, and survival in an established wild animal model of
88 aging: the baboons studied by the Amboseli Baboon Research Project in the Amboseli
89 ecosystem, Kenya (30, 31). Our goals were to determine the relative importance of health
90 selection and social causation in explaining survival patterns in adult female baboons, and to
91 determine whether adult social relationships buffer the effects of early life adversity. We focused
92 on adult females because male baboons disperse from their natal social groups when they
93 mature, making it difficult to distinguish male dispersal from death (32).

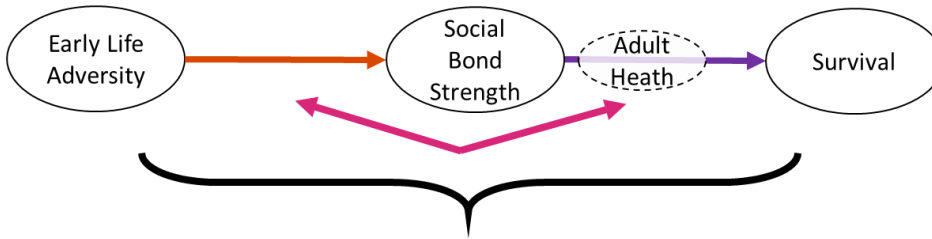
94 If health selection explains the link between adult phenotypes and survival, two
95 predictions ensue: (i) early adversity should predict weak adult social relationships, and (ii) early
96 adversity should have a direct effect on adult survival that is not mediated by adult social
97 relationships (Figure 1A). This pattern would support the idea that poor health arising from early
98 adversity leads to both social isolation and poor survival in adulthood. In contrast, social
99 causation predicts (i) that adult social bonds have strong, direct effects on survival, and (ii) that
100 these effects occur regardless of a female's early life experience (Figure 1B). Such a pattern
101 would support the idea that the effect of early life adversity on survival is at least partly mediated
102 by its effects on adult social relationships. Finally, the social buffering hypothesis, which is
103 consistent with social causation, would be supported if the effects of early life adversity on adult
104 survival are moderated by adult social behavior.

105 Previous work on female baboons in Amboseli has shown that harsh early life
106 environments predict reduced adult female lifespan (26, 33) as well as a moderate degree of
107 social isolation in adulthood (26, 34). In addition, in adult females, weak social bonds predict
108 decreased lifespan (35, 36). However, no previous study in either animals or humans has sought
109 to prospectively link early life adversity, adult social behavior, and survival in an integrated
110 analysis. Therefore, it is unknown if adult social relationships have independent effects on
111 survival or merely act as mediators that link early life to survival. We focus on adult female
112 social bonds as candidate mediators, and we exclude adult social status as a potential mediator
113 for two reasons: (i) previous studies in this population find no effects of female social status on
114 survival (35, 36), and (ii) preliminary analyses using our mediation framework ruled out social
115 status as a potential mediator of early life adversity and demonstrated that social status is not
116 influenced by cumulative early life adversity (see Materials and Methods). However, in our test
117 of the social buffering hypothesis, we consider both adult social bonds and adult social status as
118 possible moderators of the relationship between early life and survival.

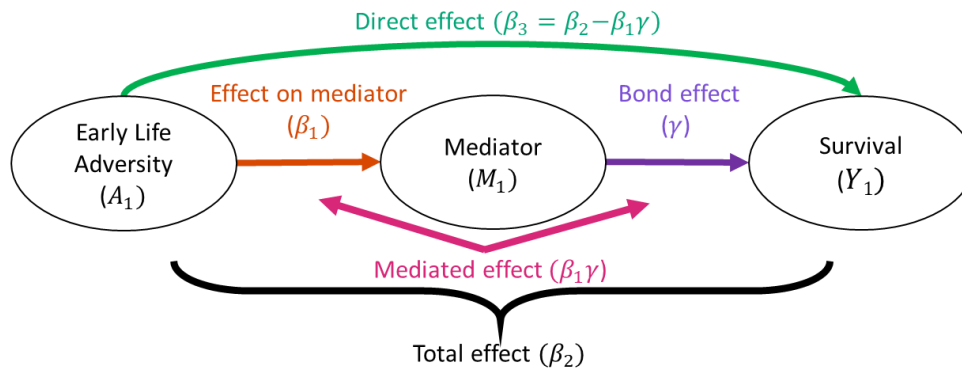
A. Health Selection Hypothesis



B. Social Causation Hypothesis



C. Mediation Analysis Framework



119

120 **Figure 1.** Hypotheses and mediation analysis framework and hypotheses linking early life
 121 adversity, adult social bond strength, and survival. The health selection hypothesis (A) posits that
 122 poor adult health arising from early life adversity prevents individuals from forming strong
 123 social relationships. Under health selection, we predict a link between early adversity and adult
 124 social bond strength (orange arrow), and a direct link between early adversity and survival (green
 125 arrow) outside of the pathway that includes social bond strength, but no mediated effect (pink
 126 arrow in B, C), and no independent effects of social bond strength on survival (purple arrow in
 127 B, C). The social causation hypothesis (B) predicts that social bond strength is a direct cause of
 128 survival differences (purple arrow). It also predicts that any effects of early life environments on
 129 survival, at least as they relate to social relationships, are due to a mediated effect (pink arrow)
 130 where early adversity affects adult social bond strength (orange arrow), which in turn affects
 131 survival (purple arrow). Under social causation, early life adversity may affect survival via other
 132 pathways (e.g., green arrow in A), but social relationships have an important causal effect. (C)
 133 The mediation analysis models the links between early life adversity (A_1), adult mediator
 134 phenotypes (M_1 , social bond strength with females or males), and survival (Y_1). Mediation
 135 models produce estimates of (i) the direct effect of early life adversity on survival outside of the

136 pathway that includes the mediator (β_3 , green arrow), (ii) the mediated effect of early life
 137 adversity on survival through the pathway that includes the mediator ($\beta_1\gamma$, pink arrow), (iii) the
 138 effect of early life adversity on the mediator (β_1 , orange arrow), (iv) the effect of the mediator on
 139 survival independent of early adversity (γ , purple arrow, the bond effect), and (v) the total effects
 140 on survival (β_2 , black bracket). Note that the expressions $\beta_1\gamma$ and ($\beta_3 = \beta_2 - \beta_1\gamma$) in panel C
 141 hold exactly only when all models (between A , M , and Y) are linear. Here we use these merely as
 142 notations (instead of mathematical equations) to label the qualitative relationship between total,
 143 mediated, direct, and bond effects.

144

145 **Mediation and moderation frameworks**

146 *Mediation models.* Our mediation analysis framework is based on structural equation
 147 models that examine the links between early life, adult social phenotypes, and survival (Figure
 148 1C, 37-39). The 199 females in this study were observed from birth and survived to at least four
 149 years old, approximately the earliest age of reproductive maturation (average age at menarche =
 150 4.73 ± 0.56 years). For each female, we evaluated her exposure to six different adverse
 151 socioenvironmental conditions in early life: 1) drought in the first year of life, 2) large group size
 152 at birth, 3) low maternal social status at birth, 4) low maternal social connectedness during the
 153 first two years of life, 5) the presence of a close-in-age younger sibling, and 6) maternal loss
 154 before four years of age (Table 1; 26, 33, 34).

155 **Table 1.** Sources of early life adversity and the number of females that experienced each source.

Source of Adversity	Description	N of females who experienced adversity	N of females who did not experience adversity
Drought	<200 mm of rainfall during the first year of life	28	171
Large group size	Group size in the top quartile (> 33 adults) at the subject's birth, indicating high social density	32	167
Close-in-age younger sibling	Younger sibling born less than 1.5 years after the subject's birth	40	159
Maternal loss	Mother dies during the first four years of the subject's life	38	161
Low maternal social status	Mother's proportional dominance rank in the lowest quartile at the subject's birth	46	153
Low maternal social connectedness	Mother's social connectedness in the lowest quartile during the subject's first two years of life	54	145

156 We constructed two sets of mediation models (see Materials and Methods), each with a
 157 different mediator variable (M), linking the treatment (early life adversity, A) to survival (Y ,
 158 measured by the hazard ratio, λ ; Figure 1C). The two mediators we examined were quantitative
 159 measures of social bond strength with other adult females and with adult males (see *Potential*
 160 *mediators*, below). Because both of these variables are known to be linked to adult survival (36),
 161 either could act as a mediator of early life adversity. We considered a female's social bonds with
 162 other adult females separately from her social bonds with adult males because same-sex and
 163 opposite sex social relationships have different relationships with early adversity and with
 164 survival and are not well-correlated (26, 34-36).

165 The mediation analysis enables us to break down the total effect of early life adversity on
 166 survival (β_2 , black arrow in Figure 1C) into direct (β_3) and mediated ($\beta_1\gamma$) effects. The direct
 167 effect (β_3) of early life adversity on survival is the pathway connecting these variables
 168 independent of the mediator (green arrow in Figure 1C). The mediated (or indirect) effect ($\beta_1\gamma$) is
 169 the pathway connecting early life adversity and survival that runs through the mediator variable;
 170 in our case, measures of social bond strength (pink arrows in Figure 1C). The mediation
 171 framework also assesses the effect of early adversity on the mediator (β_1 , orange arrow in Figure
 172 1C) and the effect of the mediator on survival independent of early adversity, hereafter the 'bond
 173 effect' (γ , purple arrow in Figure 1C).

174 For each of our mediators, we estimated the links between early life adversity (A), social
 175 bond strength (M), and survival (Y , measured by the hazard ratio λ) by fitting three equations as
 176 proposed by Zeng, Lange, Archie, Campos, Alberts and Li (40); for more details see Materials
 177 and Methods). The first equation evaluates the effect of early life adversity on observed values
 178 for the mediator, conditional on covariates, C , and random effects, r (orange arrow in Figure
 179 1C):

$$180 \quad M_{it} = M_i(t) = \beta_0(t) + A_i\beta_1(t) + \theta_1 C_{it}^M + r_{group} + \varepsilon_{it} \quad (1)$$

181 where i is individual and t is age class. Here β_1 represents the effect of early adversity on social
 182 bond strength. The second equation models the total effect of early life adversity on survival
 183 (e.g., the change in hazard rate related to early adversity; β_2 , black arrow in Figure 1C), which
 184 does not differentiate between direct and mediated effects:

$$185 \quad \lambda(t|X_i, A_i) = \lambda_0(t) \exp(\tilde{\beta}_2 A_i + \theta_2 C_{it}^S + \tilde{r}) \quad (2)$$

186 The third equation is similar to Equation 2, but incorporates estimates of the mediator based on
 187 the parameters previously fit for Equation 1. It allows us to estimate the value of the effect of the
 188 mediator on survival given the estimate of the mediator $f\{\alpha, M_i(t)\}$:

$$189 \quad \lambda(t|X_i, A_i, M_{it}) = \lambda_0(t) \exp(\tilde{\beta}_3 A_i + f\{\alpha, M_i(t)\} + \theta_3 C_{it}^S + \tilde{r}) \quad (3a)$$

190 where the mediator component $f\{\alpha, M_i(t)\}$ equals:

$$191 \quad f\{\alpha, M_i(t)\} = \int_0^{t_{max}} \alpha(u) M_i(u) du \quad (3b)$$

192 where t_{max} is the maximum lagged time (here three years) and $\alpha(u)$ is a time varying constant.
 193 Equation 3b estimates the mediator for the previous three years of life, based on values for the
 194 covariates, early life adversity, and the effect sizes estimated in Equation 1 (i.e., Equation 3b is
 195 fit based on estimated values of the mediator, not directly on observed data). We designate this

196 value the ‘three-year mediator value’, where each year corresponds to a female age class, starting
197 on her birthday and ending one day before her subsequent birthday. We also considered models
198 where the mediator was estimated based on the same year of life as survival (‘one-year mediator
199 models’) and results are consistent with three-year mediator models (Tables S1, S2).

200 Note that the effects β_2 and β_3 in Figure 1C are not numerically identical to the
201 coefficients $\tilde{\beta}_2$ and $\tilde{\beta}_3$ in Equation 2 and 3, respectively. While they are analogous to β_2 and β_3
202 in terms of the effects they represent, they differ because of the nonlinear hazard scale and
203 complex functional model adopted in the analysis (i.e., in practice, we analyze a decomposition
204 of a functional form fit to the social relationship data rather than the estimated social bond values
205 directly; see Zeng, Lange, Archie, Campos, Alberts and Li (40)). Similarly, the bond effect γ does
206 not directly correspond to a specific model parameter. Instead, β_2, β_3 , and γ are calculated from
207 functions involving all parameters in Equations 1, 2 and 3 (see the Materials and Methods and
208 derivations in Zeng, Lange, Archie, Campos, Alberts and Li (40)).

209 First, we modeled the effects of cumulative early adversity on both mediators (social
210 bond strength with females and social bond strength with males) and on survival. We measured
211 cumulative early adversity as a continuous variable representing the sum of the six individual
212 sources of adversity for each subject. No individual had a cumulative adversity score greater than
213 four (mean=1.196±0.936 SD). Second, we built multivariate models to assess the effect of each
214 individual source of adversity on each mediator and on survival, while holding the other sources
215 of adversity at zero. In these models of individual sources of adversity, each measure of
216 adversity was modeled as a categorical variable (a value of one for subjects that experience the
217 adverse event, and zero for those that did not).

218 Moderation models. To test the social buffering hypothesis, which posits that adult social
219 relationships act as a source of resilience in the face of early adversity, we next treated three
220 adult social phenotypes (social bond strength with females, social bond strength with males, and
221 social status) as potential moderators instead of mediators of early life adversity. In contrast to
222 the mediation models, the moderation models test whether the social phenotypes influence the
223 strength and direction of the effect of early life adversity on survival without making causal
224 assumptions about the pathways involved. Moderation is captured by the interaction between the
225 exposure A_i and mediator $M_i(t)$ with the interaction term $A_i g\{\eta, M_i(t)\}$ in the following model:

$$226 \quad \lambda(t|X_i, A_i, M_{it}) = \lambda_0(t) \exp(\xi A_i + f\{\alpha, M_i(t)\} + A_i g\{\eta, M_i(t)\} + \theta C_{it}^S + \tilde{r}) \quad (4)$$

227 Therefore, this approach allows us to estimate how the effects of early adversity on survival vary
228 across different levels of the social bonds or social status.

229

230 **Potential mediators and moderators**

231 Mediators. We measured each female’s social bond strength with females – i.e., the
232 strength of her social bonds with her top three female partners in each year of her life – and each
233 female’s social bond strength with males – the strength of her social bonds with her top three
234 male partners in each year of her life – as two distinct potential mediators (M) of the effects of
235 early life adversity on survival. We used grooming relationships to assess social bond strength
236 because grooming is the most prominent affiliative behavior in baboons and many other primates
237 (41-44). These mediators were represented in Equations 2 and 3 as estimates over three-year
238 periods (Equation 3b), based on the values of their covariates and the parameters fit in Equation

239 1. We also estimated mediators over shorter, one-year periods, as reported in Tables S1-S2;
240 because all analyses based on shorter periods produced qualitatively similar results, we focus on
241 the three-year estimates here.

242 *Moderators.* We considered adult social bond strength with females, adult bond strength
243 with males, and adult social status as potential moderators. We assessed social bond strength
244 using the same method described above (based on grooming relationships calculated as
245 trajectories as in Equation 1). We assessed social status using observations of wins and losses in
246 dyadic agonistic interactions between adult female study subjects. A female dominance matrix
247 was created for each month based on these win/loss outcomes, and female ordinal dominance
248 ranks were assigned by minimizing entries below the diagonal (45). We then scaled these ordinal
249 rankings by group size and assigned to each female a ‘proportional dominance rank’ (46),
250 calculated as $[1 - (\text{ordinal rank} - 1) / (\text{number adult females} - 1)]$. A female’s proportional
251 dominance rank represents the proportion of adult females that she dominates. We first
252 calculated annual mean values for social status for each subject, and then estimated their social
253 status trajectories over three-year periods, given covariates and parameter estimates for an
254 analogue of Equation 1, with M_{it} is redefined as annual mean proportional dominance rank
255 instead of annual social bond strength (see also Methods) .

256

257 **Results**

258 *Cumulative early adversity and survival: Mediated effects are weak, direct effects are strong*

259 As expected, we found a strong total effect (β_2) of cumulative early adversity on adult
260 female survival, recapitulating previous work (Tables 2-3; black bracket in Figure 2; black points
261 and lines in Figures S1-S2; 26, 33). Approximately 90% of the total effect (1.43 of 1.60 years of
262 lost life per additional exposure and 1.45 of 1.59 years, for the models considering social bond
263 strength with females and social bond strength with males, respectively) was explained by the
264 direct effect (β_3) of cumulative early adversity on survival, outside of the pathways that included
265 social bonds with either sex (Tables 2-3; green arrows in Figure 2; green points and lines in
266 Figures S1-S2). Thus, the lives of females who experience four sources of early life adversity are
267 predicted to be 6.4 years shorter than those of females that experience none, on average. Of these
268 six years, ~5.6 years would be explained by the effects of early adversity on survival,
269 independent of mediation by social bonds. Results were similar if we estimated mediation effects
270 over shorter, one-year periods instead of three-year periods (Tables S1-S2).

271 We also found substantial effects of both mediators (γ) on survival, independent of
272 effects of early life adversity. A one unit increase in social bond strength with either adult
273 females or adult males predicted a 2.2-year improvement in survival, independent of the effects
274 of early adversity, where one unit represents approximately 1.7 standard deviations for social
275 bond strength with females and 1.4 SD for social bond strength with males (Tables 2-3; purple
276 arrows in Figures 1; Figures S1-S2; see Tables S1-S2 for results with mediators estimated over
277 shorter, one-year periods). While the effects of social bonds on survival broadly recapitulate
278 previous findings in this population (35, 36), this analysis is the first to demonstrate that these
279 effects remain strong after controlling for levels of early adversity.

280 Notably, despite the fact that cumulative early adversity significantly predicted weaker

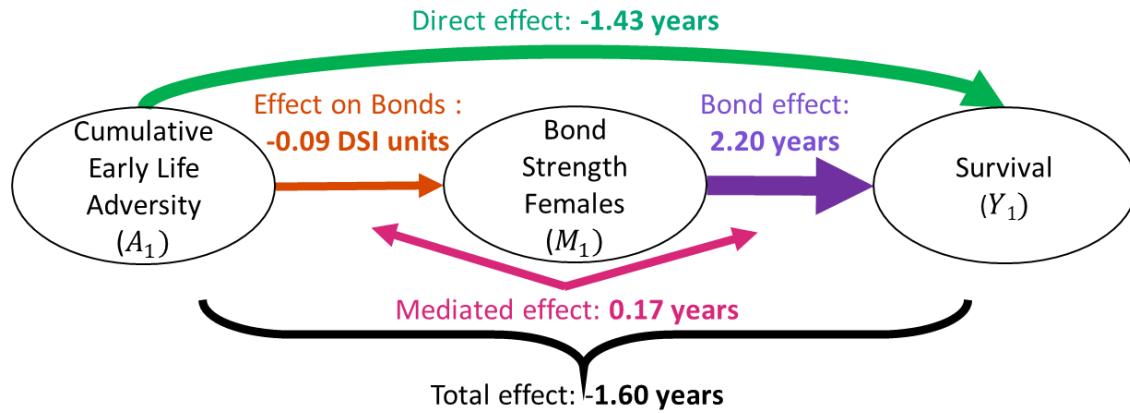
281 social bonds with females (β_1 , orange arrows in Figure 1C), and that stronger social bonds with
282 both sexes predicted higher survival, mediated effects were weak in all of our models of
283 cumulative adversity. Specifically, the pathway through social bonds with females improved
284 lifespan by only 2.04 months (10.6%), compared to the 1.60 year reduction in lifespan for each
285 additional source of adversity (the mediated effect, $\beta_{1\gamma}$, pink bracket in Figures 2A, S1; Table 2).
286 This result may stem from the fact that the effect of cumulative early adversity on social bonds,
287 while detectable, is relatively weak: early adversity is associated with a 0.09 unit decrease in
288 social bonds with females, which is small compared to the 1 unit increase in social bonds with
289 females necessary to produce a 2.2 year improvement in lifespan via the bond effect. Social
290 bonds with males did not detectably mediate the relationship between cumulative early adversity
291 and survival (Figures 2B, S2; Table 3).

292 Taken together, our results are not fully explained by either health selection or social
293 causation. Health selection would predict a direct effect of early adversity (presumed to
294 compromise adult health) on both social bond strength and survival, without a strong bond effect
295 on survival. Instead, early life adversity and social bonds both appear to have direct, independent
296 effects on survival that are of similar magnitudes. Consequently, a female baboon who
297 experienced higher than average (1 SD above the mean) cumulative early life adversity, adult
298 social bond strength with females, and adult social bond strength with males would be predicted
299 to experience a 1.35 year reduction in lifespan attributable to her early life environment, a 1.29
300 year improvement in lifespan attributable to her social bonds females in adulthood, and a 1.29
301 year improvement in lifespan attributable to her social bonds with males in adulthood. In other
302 words, both early adversity (likely via a route through poor adult health), and adult social
303 behavior are important in determining survival in adulthood.

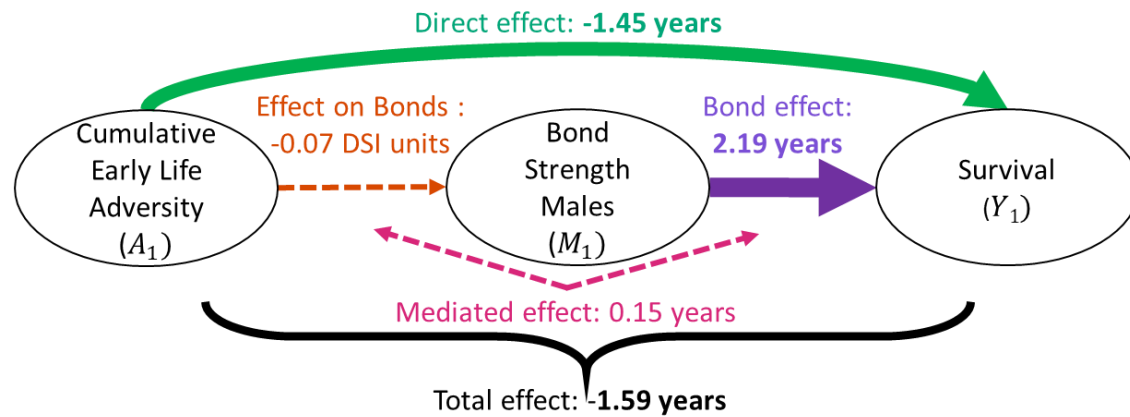
304 We next considered whether the weak mediation we observed – in spite of effects of
305 early adversity on social bonds and of social bonds on survival – might result from a mismatch in
306 the timing of these effects. To explore this possibility, we designed a simulation analysis in
307 which we defined two stages corresponding to early and late adulthood. We then assigned early
308 life effects on the mediator, and mediator effects on survival, in all possible combinations of
309 early and late timing of effects (see Supplementary Text: “Simulation to explore the small
310 mediated effect”). In our simulations, we fixed the values of both the effect of early adversity on
311 the mediator (“Effect on mediator”, orange arrow in Figure 1C) and the effect of the mediator on
312 survival (“Isolation effect”, purple arrow in Figure 1C). Even though the component parts of the
313 mediated effect were kept constant in the simulations, the estimate of the overall mediated effect
314 (pink arrows in Figure 1C) depended on the timing of these effects. Mediated effects were largest
315 when the timing of early life and mediator effects were matched; i.e., when either (i) early
316 adversity had its strongest effects on the mediator early in life *and* the mediator had its strongest
317 effects on survival early in life, or (ii) early adversity had its strongest effects on the mediator
318 late in life *and* the mediator had its strongest effects on survival late in life (see Supplementary
319 Text: “Simulation to explore the small mediated effect”, Figure S4). The results of this
320 simulation support the idea that the timing of these effects during the life course could play a role
321 in determining the strength of the mediated effect. They further suggest that, in the Amboseli
322 baboons, the timing of early life effects on adult social isolation may be mismatched with the
323 timing of social bond effects on survival. This topic merits future exploration.

324

A. Social bond strength with females as mediator



B. Social bond strength with males as mediator



325

326 **Figure 2.** Mediation analysis results. (A) Results from our mediation model using social bond
327 strength with adult females as the mediator. (B) Results from our mediation model using social
328 bond strength with adult males as the mediator. Solid lines indicate effects for which 95%
329 credible interval did not overlap zero, dashed lines indicate effects for which 95% credible
330 interval did overlap zero.

331 **Table 2.** Mediation results from models in which social bond strength with females was the mediator. Total, direct, mediated and bond
 332 effects are measured in years. The effect on the mediator is measured in social bond strength units (i.e., DSI units; 1 SD in social
 333 bonds with females=0.59 DSI units). Bolded effects are those for which the 95% credible intervals (shown in brackets below each
 334 effect size estimate) did not overlap zero. Effect names are colored as in Figure 1.

	Total effect (β_2 , years)	Direct effect (β_3 , years)	Mediated effect ($\beta_1\gamma$, years)	Effect on mediator (β_1 , DSI units)	Bond effect (γ , years)
Drought	-2.70 [-4.96, -0.44]	-2.26 [-4.04, -0.48]	0.44 [0.03, 0.85]	-0.21 [-0.38, -0.03]	2.19 [0.56, 3.82]
Large group size	-1.60 [-4.02, 0.83]	-1.38 [-2.89, 0.13]	0.22 [-0.01, 0.44]	-0.11 [-0.22, 0.01]	2.39 [0.61, 4.17]
Close-in-age younger sibling	-0.90 [-5.45, 3.65]	-0.59 [-1.99, 0.81]	0.31 [-0.04, 0.66]	-0.15 [-0.28, -0.03]	2.29 [0.75, 3.83]
Maternal loss	-3.30 [-5.79, -0.81]	-2.67 [-4.77, -0.57]	0.63 [0.06, 1.20]	-0.26 [-0.47, -0.04]	2.60 [0.79, 4.40]
Low maternal social connectedness	0.10 [-2.07, 2.27]	0.15 [-1.31, 1.62]	0.05 [-0.17, 0.27]	-0.05 [-0.15, 0.06]	2.60 [0.87, 4.34]
Low maternal social status	-1.80 [-4.37, 0.78]	-1.36 [-2.90, 0.19]	0.44 [-0.05, 0.93]	-0.14 [-0.25, -0.03]	2.49 [0.66, 4.32]
Cumulative adversity	-1.60 [-2.84, -0.36]	-1.43 [-2.52, -0.35]	0.17 [0.01, 0.32]	-0.09 [-0.16, -0.01]	2.20 [0.74, 3.65]

335

336 **Table 3.** Mediation results from models in which social bond strength with males was the mediator. Total, direct, mediated and bond
 337 effect are measured in years. The effect on the mediator is measured in social bond strength units (i.e., DSI units; 1 SD in social bond
 338 strength with males=0.70 DSI units). Bolded effects are those where the 95% credible intervals did not overlap zero. Effect names are
 339 colored as in Figure 1.

	Total effect (β_2 , years)	Direct effect (β_3 , years)	Mediated effect ($\beta_1\gamma$, years)	Effect on mediator (β_1 , DSI units)	Bond effect (γ , years)
Drought	-2.70 [-4.96, -0.44]	-2.33 [-4.17, -0.50]	0.37 [0.02, 0.71]	-0.16 [-0.29, -0.02]	2.40 [0.62, 4.17]
Large group size	-1.60 [-4.01, 0.82]	-1.53 [-3.20, 0.15]	0.07 [-1.93, 2.07]	-0.04 [-0.92, 0.84]	2.39 [0.61, 4.17]
Close-in-age younger sibling	-0.89 [-5.33, 3.55]	-0.69 [-2.08, 0.70]	0.20 [-1.20, 1.60]	-0.11 [-0.61, 0.39]	2.29 [0.75, 3.84]
Maternal loss	-3.30 [-5.78, -0.81]	-3.21 [-5.73, -0.68]	0.09 [-2.38, 2.56]	-0.06 [-1.95, 1.84]	2.20 [0.67, 3.73]
Low maternal social connectedness	0.11 [-1.90, 2.12]	0.37 [-1.26, 1.99]	0.26 [-0.15, 0.66]	-0.15 [-0.32, 0.02]	2.20 [0.73, 3.66]
Low maternal social status	-1.80 [-4.38, 0.78]	-1.16 [-2.70, 0.38]	0.64 [-0.07, 1.35]	-0.25 [-0.44, -0.06]	2.30 [0.61, 3.99]
Cumulative adversity	-1.59 [-2.82, -0.36]	-1.45 [-2.54, -0.35]	0.15 [-0.33, 0.62]	-0.07 [-0.37, 0.22]	2.19 [0.74, 3.64]

340

341 *Social bonds do not mediate the effects of individual sources of early adversity*

342 Similar to the effects of cumulative adversity, individual sources of adversity acted
343 outside of the pathway that includes social bonds, with little evidence for mediated effects in our
344 three-year mediator models (Tables 2-3; Figures S1-S2). More than 81% of the effects of
345 individual sources of adversity were attributable to direct effects (87% if only considering
346 significant direct effects). Among the six individual sources of early adversity, maternal loss and
347 drought exerted the strongest and most consistent effects on both adult female survival and social
348 bond strength with adult females (Tables 2-3; Figures S1-S2). Drought, but not maternal loss,
349 was also linked to weaker social bonds with adult males. In contrast to the effects of maternal
350 loss on social isolation from adult females, maternal loss did not predict social bond strength
351 with adult males: the estimated effect size was near zero (0.06 DSI units; Table 3). Consistent
352 with our main results, the effects of individual sources of adversity on survival were also not
353 detectably mediated by measures of social bonds with either sex based on one-year intervals
354 (Table S1-S2).

355

356 *Moderating effects: Social bonds buffer the effects of some sources of early adversity*

357 Neither social status nor social bond strength with either sex moderated the link between
358 cumulative adversity and survival (Table 4; Figure 3A,B; results were similar when we used
359 moderator trajectories estimated over a shorter, one-year period, Table S3). However, social
360 bond strength with males and social bond strength with females both moderated the link between
361 one individual source of adversity – maternal loss – and survival. Specifically, stronger social
362 bonds with either females or with males during adulthood buffered the negative effect of
363 maternal loss on survival (and conversely weaker social bonds amplified the negative effect of
364 maternal loss on survival; Table 4, Table S3: Figure 3A,B,D,E). In other words, survival was
365 disproportionately lower for females who lost their mother early in life and were more socially
366 isolated in adulthood (and conversely, survival was disproportionately higher for females who
367 lost their mother but formed strong social relationships in adulthood, with either sex; Figure
368 3A,B,D,E). Females who lost their mother early in life but maintained strong social relationships
369 with other females (1 SD above the mean) experienced a 10% reduction in hazard ratios relative
370 to females who lost their mothers and had average social bond strength to other females. In
371 contrast, females who lost their mothers and had weak social relationships with females (1 SD
372 below the mean) had 16% higher hazard ratios than females who lost their mothers and had
373 average social bond strength to other females (Figure 3D). The effect was stronger for bonds
374 with males, where females who lost their mothers in early life but maintained strong social bonds
375 with males (1 SD above the mean) had an 18% lower hazard ratio, while those who had weak
376 social bonds with males (1 SD below the mean) had a 16% higher hazard ratio, compared to the
377 effects of maternal loss for females with average social bond strength (Figure 3E). In addition,
378 another individual source of early adversity – low maternal social connectedness – was buffered
379 by strong adult social bonds with males, but not by adult social bonds with females (Figure 3B).

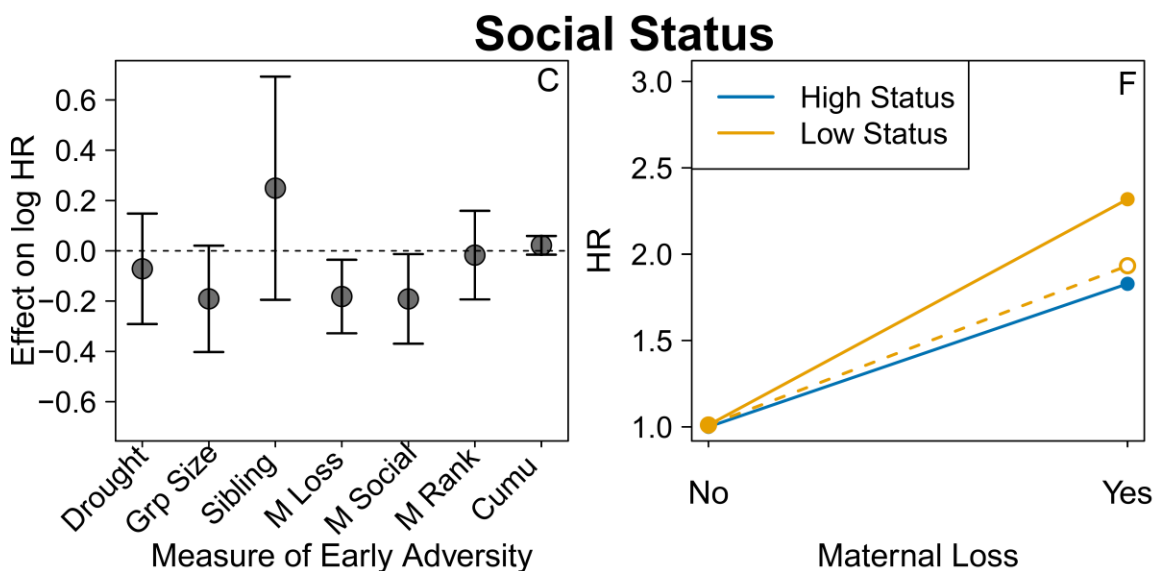
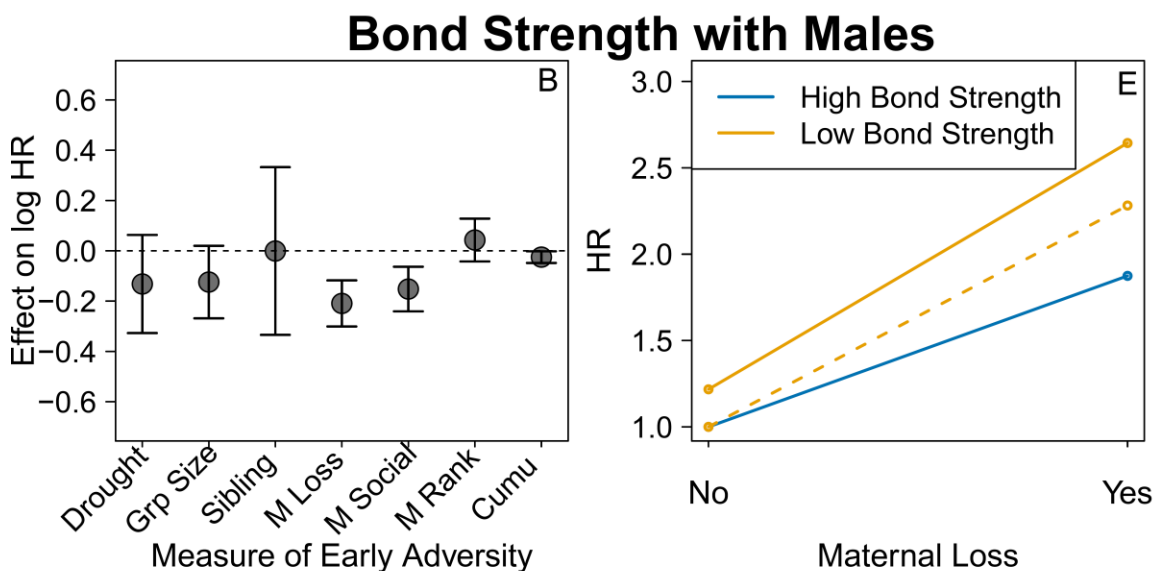
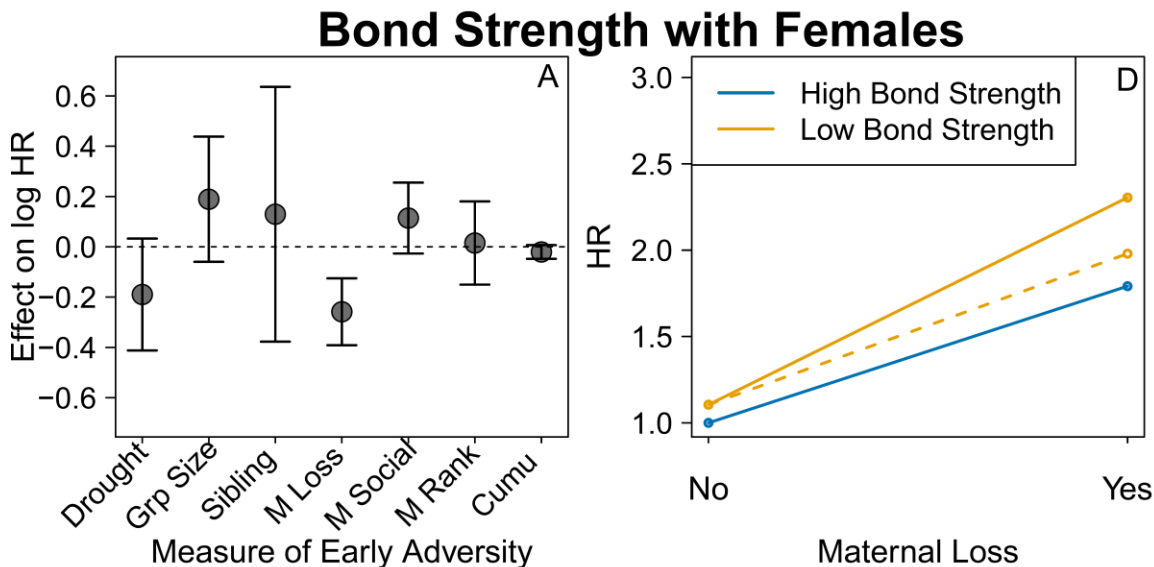
380 Female social status also moderated early life maternal loss and low maternal social
381 connectedness effects on survival (Figure 3C, Table 4; note that this effect was not detectable
382 when moderator trajectories were estimated over a shorter, one-year period, Table S3).
383 Specifically, survival was disproportionately lower for low-ranking females who lost their
384 mothers early in life or had a socially isolated mother, and disproportionately higher for high-

385 ranking females who lost their mothers early in life or had a socially isolated mother (Figure
 386 3C,F). Females who lost their mother early in life, but were high social status in adulthood (1SD
 387 above the mean) had a 5% lower hazard ratio compared to females who lost their mother but
 388 were of average social status. In contrast, females who lost their mother early in life, but were
 389 low social status in adulthood (1SD below the mean) had 20% higher hazard ratios, compared to
 390 the effects of maternal loss for females with average social status.

391
 392 **Table 4.** Moderation results from models in which social bond strength with females, social
 393 bond strength with males, and female social status were the moderators. Values represent the
 394 magnitude of the interaction effects measured in log hazard ratio (HR). Bolded effects (those for
 395 which the 95% CI did not overlap zero) show that the effects of maternal loss on survival were
 396 moderated by all three phenotypes and that the effects of low maternal social connectedness were
 397 moderated by adult social relationships with males and female social status. A negative
 398 interaction effect indicates that increased adult social bond strength or higher social status acts as
 399 a buffer to reduce the negative effects of early adversity on survival. A positive interaction effect
 400 value means that adult social bond strength or higher social status acts as an amplifier to increase
 401 the negative effects of early adversity on survival.

	Social Bonds w/ Females (log HR)	Social Bonds w/ Males (log HR)	Social Status (log HR)
Drought	-0.19 [-0.41, 0.03]	-0.13 [-0.33, 0.06]	-0.07 [-0.29, 0.15]
Large group size	0.19 [-0.06, 0.44]	-0.12 [-0.27, 0.02]	-0.19 [-0.40, 0.02]
Close-in-age younger sibling	0.13 [-0.38, 0.64]	0.00 [-0.33, 0.33]	0.25 [-0.19, 0.69]
Maternal loss	-0.26 [-0.39, -0.13]	-0.21 [-0.3, -0.12]	-0.18 [-0.33, -0.04]
Low maternal social connectedness	0.11 [-0.03, 0.26]	-0.15 [-0.24, -0.06]	-0.19 [-0.37, -0.01]
Low maternal social status	0.02 [-0.15, 0.18]	0.04 [-0.04, 0.13]	-0.02 [-0.19, 0.16]
Cumulative adversity	-0.02 [-0.05, 0.01]	-0.03 [-0.05, 0.00]	0.02 [-0.02, 0.06]

402



404 **Figure 3.** Moderation models support some forms of social buffering. (A, B, and C) Moderating
405 effects of (A) social bond strength with females, (B) social bond strength with males, and (C)
406 social status on the relationship between early adversity and survival (log of the hazard ratio,
407 HR). A positive value on the y-axis means that greater social bond strength or higher social
408 status amplify the negative effects of early adversity on survival. Grp Size indicates large social
409 group size, M Loss indicates maternal loss, M Social indicates low maternal social
410 connectedness, M Rank indicates low maternal rank, and Cumu indicates cumulative adversity.
411 Panel A shows that strong social bonds with females buffer the effects of maternal loss; Panel B
412 shows that strong social bonds with males buffer the effects of both maternal loss and low
413 maternal social connectedness; Panel C shows that high social status buffers the effects of both
414 maternal loss and low maternal social connectedness. (D, E, and, F) The effects of (D) social
415 bond strength with females, (E) social bond strength with males, and (F) social status on the
416 relationship between maternal loss and survival (measured as the hazard ratio, HR). The orange
417 dashed line in each panel represents the expected effect of maternal loss on the hazard ratio for
418 adult females in the absence of any moderating effects of social bonds or status. The blue solid
419 line shows that females with social bond strength one standard deviation (SD) above the mean
420 (i.e., females with stronger social bonds) or females with social status one SD above the mean
421 (i.e., females with high status) experience a disproportionately lower hazard ratio in the presence
422 of maternal loss. The orange solid line shows that females with social bond strength or social
423 status one SD below the mean experience a disproportionately higher hazard ratio as a function
424 of maternal loss.

425 Discussion

426 Previous work has debated the relative importance of early adversity and adult social
427 relationships in determining survival in humans (13, 20-22). Our results shed light on this debate
428 by providing an example of a wild animal model in which both early life experiences and adult
429 social relationships are important and act independently on survival, with effects of similar
430 magnitude. In addition, our moderation analysis indicates that at least for some sources of
431 adversity, social relationships in adulthood may act as sources of resilience, allowing individuals
432 to buffer the negative effects of poor early life experiences. Below, we consider several
433 implications of these results, including the puzzle of weak mediation in spite of significant links
434 between treatment, putative mediator, and outcome.

435

436 *The puzzle of weak mediation*

437 We observed strong effects of both early adversity and adult behavior on survival, and
438 effects of early adversity on at least one aspect of the adult social environment, with little or no
439 mediation. One potential explanation for this set of observations is that an assumption of the
440 mediation analysis was violated, thus producing spurious results. The most likely violated
441 assumption is that of sequential unconfoundedness: i.e., if an unmeasured confounder in our
442 system affects both the mediator and survival (47, 48). For example, individuals with better
443 phenotypic or somatic quality (resulting from either genetic or environmental differences that
444 were not included in our analysis) may experience both stronger social bonds and better survival,
445 independent of early adversity. In this case, phenotypic/somatic quality would be an unmeasured
446 confounder (see discussion of sequential unconfoundedness in 49). To examine the potential for
447 a confounding variable to affect our analyses, we conducted sensitivity analyses that assess how
448 the mediated effect estimates vary as a function of the extent of the correlation between an
449 unmeasured confounding variable and the mediator, and between that same variable and
450 survival. Our sensitivity analyses demonstrate that our results are relatively robust to the
451 assumption of sequential unconfoundedness (see Supplementary Text: “Sensitivity analysis for
452 sequential unconfoundedness”; Figures S5, S6). As a consequence, it is likely that we are
453 correctly estimating a small mediation size in this study.

454 A second, more likely, explanation is that the effects of early life adversity on social
455 bonds in adulthood, albeit detectable, are relatively weak. Rosenbaum, Zeng, Campos,
456 Gesquiere, Altmann, Alberts, Li and Archie (34) found similar results when testing for the
457 mediating effect of social bonds for the relationship between early adversity and glucocorticoid
458 levels. If early adversity does not have strong effects on social bond strength, then social bonds
459 are unlikely to strongly mediate the comparatively quite strong connection between early
460 adversity and adult survival.

461 In addition, our causal mediation pathway may be shaped by time-varying effects, as
462 suggested by our simulation model. For example, if early life adversity affects social bonds early
463 in adulthood, and survival is most strongly affected by social bond strength early in adulthood,
464 then the matched timing of these effects could give rise to a strong mediating effect of social
465 bonds. However, if early life adversity affects social bond strength earlier in adulthood, while
466 survival is most strongly affected by social bond strength later in adulthood, then the mismatched
467 timing of these effects would minimize the mediation effect. Previous work in birds and humans

468 has shown that such time-varying effects may be a general phenomenon that warrants more
469 attention (7, 50, 51). For example, in a survey of American adults, Nurius, Fleming and Brindle
470 (7) show that social relationships in young adulthood are not linked to health, but that older
471 adults with stronger social connections are in better health. Yang, Boen, Gerken, Li, Schorpp
472 and Harris (51) also identified variability in the effects of social integration on several health
473 biomarkers between American adolescents and adults. Exploring time varying effects of early
474 adversity is therefore an important future avenue of exploration.

475 Two additional explanations are consistent with our observation of independent effects of
476 social bonds and early adversity, combined with weak mediation. First, social bonds may be one
477 of a larger set of mediators that all weakly mediate the link between early life environments and
478 survival. Second, an as-yet unidentified variable could act as a strong mediator of early life
479 adversity without involving social bonds. For example, the biological embedding hypothesis
480 predicts that glucocorticoids – produced by the hypothalamic–pituitary–adrenal (HPA) axis and
481 involved in regulating multiple physiological processes – link early adversity and lifespan (52,
482 53). In our study population, early life adversity predicts elevated concentrations of
483 glucocorticoid metabolites in fecal samples in adulthood (34). Furthermore, elevated fecal
484 glucocorticoid (fGC) concentrations in adulthood are associated with a shortened lifespan (54).
485 At the same time, social bonds in adulthood are only modestly correlated with fGC
486 concentrations (34), pointing to fGCs as a possible mediator of early life adversity that bypasses
487 the pathway through social bonds. Notably, fGC concentrations, like social bonds, appear to
488 weakly mediate the effects of early life adversity on survival (40), indicating that this pathway
489 not only represents an alternative to the mediating pathway through social bonds, but also that
490 multiple mediators may be involved.

491

492 *The evolutionary significance of sources of variance in survival*

493 The independent effects of cumulative early life adversity and social bonds on female
494 baboon survival are considerable. For each additional source of early adversity, lifespan is
495 decreased by approximately 1.4 years, independent of social bond strength. Similarly, a one
496 standard deviation decrease in social bond strength with either sex predicts 2.2 years of
497 decreased lifespan, independent of early adversity. Notably, lifespan explains >80% of the
498 variation in lifetime reproductive success (26, 33, 55), and females who experience early life
499 adversity do not accelerate reproduction to compensate for the reduction in lifespan (33).
500 Consequently, the combined effects of cumulative early life adversity and adult social isolation
501 on survival have major consequences for lifetime reproductive success for female baboons.

502 These large effects on fitness indicate that phenotypes that allow individuals to survive in
503 the face of multiple sources of adversity—which include features of the physical, social, and
504 maternal environment—are likely to be favored by natural selection (56-58). Features of the
505 social and maternal environment can be under direct natural selection. For example, our results
506 suggest that selection should favor low adult mortality in part because maternal mortality directly
507 decreases offspring survival in adulthood (in addition to other effects, such as the increase in the
508 number of reproductive opportunities that comes with longer lifespans). In contrast, features of
509 the physical environment (e.g., drought) cannot be under direct natural selection. However,
510 adverse physical environments impose natural selection that acts on individual responses to
511 environmental adversity. Indeed, work in humans has identified many genetic variants that

512 influence the response to environmental stressors (e.g., pathogens, chemical stimuli), and some
513 of these variants also carry genetic signatures of selection (59, 60). Thus, we expect natural
514 selection to favor phenotypes that confer resilience to early life adversity even if the resulting
515 phenotypes have lower fitness than phenotypes produced under advantageous early life
516 conditions (56-58).

517 Adult social relationships also had strong and independent effects on adult survival,
518 indicating that adult social behavior is not merely a proxy for the early life environment but is
519 likely directly targeted by natural selection. Previous work on the links between social bonds and
520 fitness did not control for early life experience (35, 36), limiting the ability to disentangle direct
521 and indirect effects of adult behavior on fitness (9, 61). Our results suggest that adult social
522 behaviors that maintain social bonds should be under strong selection. Further, because social
523 behavior is almost always partially heritable (e.g., (62-64), these behaviors have the potential to
524 evolve via natural selection. Further, they suggest that indirect genetic effects, in which the
525 genotypes of social partners affect behavior, could play an important role in social selection and
526 evolution (65, 66). Indirect genetic effects are unique because they illustrate that the environment
527 itself can evolve and as a result create feedback loops that amplify or constrain evolutionary
528 change, even in the absence of direct selection. However, selection on sociality is also likely to
529 be limited by tradeoffs (67, 68). For example, tradeoffs may occur between the time allocated to
530 sociality versus to other activities that are important for maintenance, such as foraging. In
531 addition, sociality itself imposes costs, including potential increases in pathogen transmission,
532 intraspecific competition, and social stress. Finally, the mechanisms that link adult social
533 relationships to survival remain unclear, making it difficult to definitively identify potentially
534 important targets of selection in addition to social bonds themselves.

535

536 *Individual sources of early adversity*

537 We found strong effects of two individual sources of adversity on adult social bond
538 strength and survival: maternal loss and drought. Consistent with previous findings (26, 33, 34,
539 40, 69), females whose mothers died when they were young had weaker social bonds with other
540 females and reduced survival compared to females who did not experience early maternal loss,
541 although they exhibited no differences in social relationships with males. In nonhuman primates,
542 maternal loss during the juvenile period compromises the learning of social and foraging skills
543 (70-73). In our study system in particular, losing a mother early in life is associated with shorter
544 adult lifespans, weaker adult social bonds with females, compromised patterns of adult rank
545 acquisition (74), elevated glucocorticoid concentrations in adulthood (34, 39, 40), and relatively
546 poor survival of offspring (69, 75). Maternal loss also has negative consequences for adult
547 phenotypes and fitness in other mammal species (27, 75-79) including humans (80, 81).
548 Therefore, maternal loss during development represents a strong source of early adversity across
549 taxa, especially in species where mothers are essential for the development of crucial skills.

550 In addition to maternal loss, drought emerges as an important source of early life
551 adversity in this analysis. Females who experienced drought in their first year of life had weaker
552 social bonds with both females and males, and also experienced reduced survival relative to
553 females born in non-drought years via both mediated and direct effects (Tables 2, 3). Drought
554 threatens food availability which in turn hinders growth and development during the crucial first
555 year of life (82-86). In addition, individuals born during drought may have fewer opportunities to

556 learn foraging skills during younger years when adults are more tolerant of them during foraging
557 (72, 87). Consistent with our results, experiencing dry seasons and droughts in early life
558 negatively affects health in humans (88-93).

559 Notably, two previous analyses in our study system found that drought did not predict
560 adult survival independently of other sources of early adversity (26, 33). The difference between
561 the previous studies and this one may be attributable to using somewhat different subsets of the
562 long-term data, because of different data requirements for each analysis. For instance, the current
563 analysis includes a larger representation of females who were born during a particularly severe
564 drought in 2008-2009, a two-year consecutive period in which annual rainfall was less than 200
565 mm (94). This drought inflicted substantial mortality on wildlife and livestock throughout the
566 Amboseli ecosystem and surrounding areas (95, 96). Therefore, it represented an extreme
567 climatic event in the early lives of these individuals which may have driven the strong effects of
568 drought not detected in previous analyses (26, 33).

569

570 *Moderating effects of adult behavior*

571 Our analyses indicate that strong social bonds in adulthood may buffer the negative
572 consequences of adverse early life events—even for maternal loss, which has far-reaching effects
573 on phenotypes and fitness. Specifically, we found that female baboons who lost their mothers in
574 early life but were able to maintain strong social bonds in adulthood survived better than those
575 who lost their mother and were socially isolated. Therefore, resilient adult phenotypes may
576 buffer the negative effects of maternal loss. Social buffering has also been suggested as a
577 mechanism to counteract the negative effects of early life adversity in other mammals (18, 97)
578 and humans (81). For example, mountain gorillas who lose their mothers tend to strengthen their
579 social bonds with other group members; perhaps as a consequence, they suffer no detectable
580 survival costs from maternal loss (97). Social bonds with males may be a particularly important
581 buffer as, unlike social bonds with females, they are not weakened by maternal loss (34).

582 *Conclusions and future directions*

583 By linking prospective data on early life adversity with data on social bonds and survival
584 in adulthood, we find support for both social causation and health selection. Specifically, by
585 accounting for the complex relationships between early life, adulthood, and survival, we
586 confirmed the far-reaching effects of early life adversity – which contributes directly to both
587 compromised adult social relationships and adult survival – and we also confirmed a direct
588 influence of adult social relationships on survival. Furthermore, for at least some sources of early
589 adversity, strong adult social bonds can reduce the negative effects of early life adversity. In
590 addition to finding support for both social causation and health selection, we argue that responses
591 to early adversity, sources of early adversity, and adult social behavior are all likely targets of
592 natural selection. Future work should explore how variation in the timing of early life effects,
593 and in the timing of the effects of adult phenotypes, affect connections between early adversity,
594 mediators, and survival in other species. Future work should also examine other potential
595 mediators (e.g., phenotypic quality, immune response, glucocorticoid levels) of the relationship
596 between early adversity and lifespan.

597

598 **Materials and Methods**

599 *Study Subjects*

600 We used longitudinal data on 199 wild adult female baboons (*Papio cynocephalus*, with
601 some natural admixture from the closely related species *P. anubis* (98, 99) from the Amboseli
602 ecosystem in Kenya collected between 1983 and 2019. Subjects are habituated to and
603 individually recognized by experienced observers who collect demographic and behavioral data
604 6 days a week, year-round, following 1-2 social groups ('study groups') per day. Birth and death
605 dates for all study subjects are accurate to within a few days' error. Two original study groups
606 (studied beginning in 1971 and 1980 respectively) experienced multiple permanent fissions and
607 fusions over the years, resulting in a total of 19 different social groups that persisted for varying
608 lengths of time. Female baboons remain in their natal social group throughout their lives (except
609 for group fissions or fusions), and thus any disappearance of a female in our dataset was
610 considered a death. Of the 199 females in the study, 74 had died by the end of the study and the
611 rest were considered censored in survival analyses. To be included in the study, females had to
612 meet the following criteria: (i) they survived to at least 4 years of age (most females reach
613 menarche between 4 and 5 years of age; (100), (ii) they had available data on exposure to all six
614 sources of early adversity in the infant and juvenile period, and (iii) they were members of study
615 groups that foraged entirely on naturally occurring foods (26, 33, 34).

616

617 *Measuring Early Life Adversity*

618 We created an index of cumulative early life adversity by considering six conditions that
619 represent socioenvironmental adversity experienced during the first four years of life: drought in
620 the first year of life, large group size at birth, low maternal social status at birth, low maternal
621 social connectedness in the first two years of life, a close-in-age younger sibling, and maternal
622 loss before age four (Table 1; 26, 33). Drought years were those in which less than 200 mm of
623 rain fell. Large group sizes were considered as those in the highest quartile of the group size
624 (number of adults) distribution. Low maternal social status was assigned when the mother's
625 proportional dominance rank in the month of her offspring's birth was in the lowest quartile of
626 dominance ranks. Proportional dominance rank ranges from 0 (lowest ranking female) to 1
627 (highest ranking female) and indicates the proportion of adult females in a study subject's social
628 group that she dominated in agonistic interactions (46). Low maternal social connectedness was
629 assigned when the mother's social connectedness to other females was in the lowest quartile of
630 the distribution of social connectedness values during our study subjects' first two years of life.
631 Following previous work on early life adversity in this population (26, 33), social connectedness
632 was measured as the relative frequency of the mother's grooming interactions with other adult
633 females in her social group, adjusted for observer effort (see 'Measuring social bond strength'
634 for information about observer effort). Close-in-age younger siblings were those born within 1.5
635 years of the subject's birth, approximately the shortest quartile of observed interbirth intervals in
636 the Amboseli baboons (26). A subject was considered to experience maternal loss if her mother
637 died within her first four years of life (i.e., before the earliest age of sexual maturation for
638 females in this population).

639 Each subject's cumulative adversity index was calculated as the sum of exposures to
640 these six sources of adversity. In our dataset, 48 females experienced zero sources of adversity,

641 84 experienced one, 50 experienced two, 14 experienced three, 3 experienced four, and none
642 experienced five or six.

643

644 *Measuring Social Bond Strength*

645 We measured an adult female's social relationships by assessing the strength of social
646 bonds with her top three male or female social partners separately, in each year of her life,
647 measured relative to the social bonds of all other females in the population with males or females
648 respectively, as described in Rosenbaum, Zeng, Campos, Gesquiere, Altmann, Alberts, Li and
649 Archie (34). Briefly, grooming interactions are recorded during all hours of observation, using
650 representative interaction sampling in which observers record all the interactions they see while
651 conducting 10-minute focal follows on a randomized set of individuals. We calculated the
652 number of grooming interactions with each partner per day of co-residence in the same group
653 from these representative interaction data for each year of life for each female subject starting on
654 her birthday. Calculating interaction rates from such data is complicated by the fact that the
655 number of observers remains constant over time, while social group sizes vary, so that higher
656 numbers of grooming interactions per pair of animals (per dyad) will generally be observed in
657 smaller groups compared to larger groups. We corrected for this variation in observer effort by
658 regressing daily rates of grooming interactions per dyad against observer effort, where observer
659 effort was calculated as the number of focal samples on adult females collected during each
660 observer day, divided by the mean number of adult females in the group during those days,
661 divided by the number of days that each dyad was co-resident (34, 36). We z-scored the
662 corrected rates within years to control for temporal variation in sociality in the population.

663 Each subject's social bond strength with females and with males was taken as the average
664 of the subject's three strongest adult female grooming partners and adult male grooming
665 partners, respectively, to calculate a dyadic sociality index (DSI). A positive value for social
666 bond strength indicates a female had relatively strong social bonds with her top three partners
667 compared to the population average. A negative value for social isolation means the subject had
668 relatively weak social bonds with her top three partners.

669

670 *Random Effects and Covariates*

671 Previous work has demonstrated that several environmental and demographic variables
672 not discussed above (i.e., presence of maternal relatives, group size, social status, percent of prior
673 year with young infant, percent of prior year cycling, rainfall) explain variation in social bond
674 strength and/or survival (34-36, 42). To control for these effects, we included them as covariates
675 in our mediation and moderation analyses (for details see Supplementary Information). We also
676 included social group and hydrological year as random effects in all models to control for group-
677 to-group and interannual variation (34). Age was not included as a covariate even though social
678 bonds vary with age, because age effects are captured by our functional principal components
679 analysis (FPCA) approach to modeling the mediator (see below). Because our baboon study
680 population represents an admixed population (yellow baboon ancestry is dominant, but all
681 individuals exhibit some degree of admixture with anubis baboons), we also ran separate
682 analyses that included a covariate measure of individual admixture, a 'genetic hybrid score' that

683 represents the proportion of each individual's genome estimated to be from *P. anubis* ancestry
684 (see Supplementary Information, also (101, 102). Results that incorporated hybrid score (Tables
685 S4-S5) were similar to those of the full model (Tables 2-3).

686 In preliminary analyses we considered social status as a third potential mediator of the
687 effects of early adversity on survival. However, as previously reported (35, 36), we found no
688 effects of social status (again measured as proportional dominance rank) on female survival
689 (Table S6). In addition, we found no effect of cumulative early adversity on female social status,
690 and no mediating effects of female social status on the relationship between early life adversity
691 and survival (Tables S6). As a consequence, we focus on social bond strength as the primary
692 mediating variable in the main text, but report models for social status as a mediator in the
693 Supplementary Materials.

694 One individual source of early adversity strongly predicted proportional dominance rank:
695 low maternal dominance rank predicted low proportional rank for the study subject in adulthood
696 (Table S6), which is unsurprising as rank is matrilineally inherited in this species (103). In light
697 of this relationship, we controlled for proportional rank by including it as a covariate when
698 estimating the effect of early life adversity on the mediator.

699

700 *Mediation Analysis Implementation*

701 We fit two models in each of our mediation analyses (40). The first model captures the
702 relationship between early adversity and the mediator. The second model characterizes the
703 relationship between early adversity, the mediator, and survival. Models were implemented using
704 the R packages *survival* and *flexsurv*. The reproducible code is available at
705 [https://github.com/zengshx777/MFPCA_Codebase].

706 *The first model: the relationship between early adversity and the mediator.* Our first
707 model applies to the observed mediator trajectory M_{ij} and the measure of early adversity A_i ,
708 where i indexes individual and j indexes time. This model corresponds to the Equation 1 in the
709 main text. Because the observed mediator values are noisy and potentially measured imprecisely,
710 we consider them, after adjusting for covariates and random effects, as realizations of an
711 underlying smooth process ($M_i(t_{ij})$) with a random noise. Specifically, we modeled the
712 trajectory of the mediator M_{ij} as a combination of covariate effects $C_{ij}\beta_m$, social group random
713 effects $r_{social\ group}^m$, hydrological year random effects r_{hydro}^m , an underlying smooth process
714 $M_i(t_{ij})$, and an error term ε_{ij} ,

$$M_{ij} = C_{ij}^m \beta_m + r_{social\ group}^m + r_{hydro}^m M_i(t_{ij}) + \varepsilon_{ij}, \varepsilon_{ij} \sim N(0, \sigma_m^2) \quad (5)$$

715 Because $M_i(t_{ij})$ is of infinite dimension mathematically, we performed dimension reduction to
716 improve the statistical power of our analysis. Specifically, we used a functional principal
717 component analysis (FPCA) method to decompose the smooth process as the linear combination
718 of the fewest possible functional principal components (39, 40, 104-106). We began by
719 examining the correlation between any two time points in the mediator process (e.g., between the
720 value of the mediator at age 4 and age 8, between the value of the mediator at age 4 and age 9,
721 and so on) to produce a correlation structure between mediator values at different time points,
722 which we then expressed as principal components or eigen functions,

$$Cov(M_i(t_1), M_i(t_2)) = \sum_{k=1}^{\infty} \lambda_k \psi_k(t_1) \psi_k(t_2), \lambda_1 \geq \lambda_2 \geq \dots \geq 0 \quad (6)$$

723 where λ_k is the explained variance of the orthogonal normal principal components $\psi_k(t)$. We
 724 ordered the principal components by the amount of variance they explained to reflect the fact
 725 that principal components that explain more variance (larger λ_k) are more important in
 726 expressing the smooth process. We then used the first K principal components, where K is the
 727 number of components necessary to collectively explain at least 90% of the variance
 728 ($\sum_{k=1}^K \lambda_k / \sum_{k=1}^{\infty} \lambda_k \geq 90\%$).

729 In the next step, we represent the smooth process of each subject's mediator process as a
 730 linear combination of the K principal components,

$$M_i(t) = \sum_{k=1}^K \xi_{ik} \psi_k(t) \quad (7)$$

731 where ξ_{ik} is the principal score for individual i on the k th principal component or eigen function.
 732 The variance of ξ_{ik} corresponds to the explained variance of principal component, λ_k . We can
 733 efficiently express the smooth process and trajectory with a small number of principal
 734 components (K is never greater than 4 in our work), capturing the major variation. Therefore,
 735 coupled with the FPCA, we posit the following model of the mediator,

$$M_{ij} = C_{ij} \beta_m + r_{cluster}^m + r_{hydro}^m + \sum_{k=1}^K \xi_{ik} \psi_k(t) + \varepsilon_{ij}, \varepsilon_{ij} \sim N(0, \sigma_m^2) \quad (8)$$

736 which corresponds to Equation 1 in the main text. Furthermore, we assume that the differences in
 737 trajectories caused by early adversity are captured by the differences in the principal scores.
 738 Therefore, we use the following specification for the principal scores, with different means for
 739 each level of adversity in the cumulative model or with different means for the group that
 740 experienced each early adversity and for the group did not experience early adversity in the
 741 models of individual sources of adversity,

$$\xi_{ik} = A_i(\tau_1^k - \tau_0^k) + \tau_0^k + \eta_{ik}, \eta_{ik} \sim N(0, \lambda_k), \lambda_1 \geq \lambda_2 \geq \dots \lambda_K \geq 0 \quad (9)$$

742 where τ_1^k denotes the mean of the k^{th} principal score for the subjects in the adversity group while
 743 τ_0^k represents that for the non-adversity group (for cumulative adversity, τ_1^k denotes the k^{th}
 744 principal score for a higher level of adversity relative to τ_0^k , the score for one level lower) We fit
 745 Equation 8 simultaneously with Equation 9. Hence, instead of estimating the effect of adversity
 746 on the trajectories directly, which is a high dimensional problem, we estimate its effect on the
 747 first K principal scores $\xi_{i1}, \xi_{i2}, \dots, \xi_{iK}$.

748 The effect of early adversity parameterized with different means for the principal scores
 749 is not directly interpretable. Therefore, we estimated the effect of early adversity on the mediator
 750 as the difference in the mean of the trajectories for the adversity group versus the non-adversity
 751 groups (for the cumulative adversity measure it was the difference in means comparing two
 752 adjacent levels of adversity, e.g., for a cumulative score of 3 versus 2). Based on Equations 8 and
 753 9, we can express the conditional expectation of the mediator process M_{ij} at time point t_{ij} as

754 follows,

$$E(M_{ij}|C_{ij}, A_i) = \beta_m C_{ij} + \sum_{k=1}^K (A_i(\tau_1^k - \tau_0^k) + \tau_0^k) \psi_k(t_{ij}), \quad (10)$$

755 which corresponds to Equation 1 in the main text. Next, we express the effect of early adversity
756 on social isolation using:

$$b_1(t) = \sum_{k=1}^K (\tau_1^k - \tau_0^k) \psi_k(t) \quad (11)$$

757 The effect on the mediator is also time-indexed, because we are estimating the effect of
758 adversity on the mediator trajectory across the lifespan. Integrating $b_1(t)$ over time gives an
759 estimation of parameter β_1 (the beta coefficient associated with the effect on the mediator) in
760 Equation 1 in the main text:

$$\beta_1: \frac{1}{T} \int_0^T b_1(t) dt \quad (12)$$

761

762 *The second model: the relationship between early adversity, the mediator, and survival.* Our
763 second model estimated the survival outcomes. We adopted a Cox model for the hazard rate
764 $\lambda(t)$. Specifically, we employed the following model,

$$765 \lambda(t|C_{it}, A_i, M_i(t)) = \lambda_0(t) \exp(\widetilde{\beta}_3 A_i + f\{\alpha, M_i(t)\} + \theta_3 C_{it}^S + r_{cluster}^y + r_{hydro}^y) \quad (13)$$

766 where (i) $f\{\alpha, M_i(t)\}$ is the function of the mediator process up to time point t with parameter α
767 characterizing the effect of the mediator process on the hazard rate, and $M_i(t)$ is replaced by its
768 estimated value $\widehat{M}_i(t_{ij})$ from Equation 8, and (ii) $\lambda_0(t)$ is the baseline hazard rate, which we
769 specify as following a Gompertz distribution (107, 108),

$$770 \lambda_0(t) = a \exp(bt). \quad (14)$$

771 We consider two specifications of f in our case: (i) a model using estimated trajectories of three-
772 year mediator values that assumes the hazard rate depends on the mediator history in the
773 previous three years, $f\{\alpha, M_i(t)\} = \int_0^t \alpha(s) M_i(s) ds$, and (ii) a model using estimated
774 trajectories of one-year mediator values that assumes the hazard rate depends on the current
775 mediator value assessed in the year in which survival is assessed, $f\{\alpha, M_i(t)\} = \alpha M_i(t)$. For the
776 three-year model, we specify $\alpha(t)$ as a linear combination of the spline basis $\alpha(t) =$
777 $s(t)' \rho$, $s(t) = [1, t, (t - k_1)^2, (t - k_2)^2, \dots, (t - k_L)^2]$ (106), which allows a flexible modeling
778 of how the past mediator affects the survival.

779 Following the notation in the causal mediation analysis literature (109, 110), let $S_{z,z'}(t)$
780 denote the survival function when the subject's early adversity status is z and the mediator
781 trajectory counterfactually takes the value as if the subject has early adversity status z' . The
782 adversity status z can be ordinal (for cumulative adversities) or binary ($z=0$ for the non-adversity
783 group and $z=1$ for the adversity group mediator to estimate the total, direct, and mediated

784 effects). For example, if $z=0$ and $z'=1$, then $S_{z,z'}(t)$ is the survival function for baboons who did
 785 not experience early adversity, but whose mediator values are counterfactually calculated as if
 786 they did experience early adversity. This strategy is standard in causal mediation research; it
 787 allows us to decompose the total effect into the mediated effect and the indirect effect (40, 110-
 788 112). Based on the model for hazard rate, we can calculate the $S_{z,z'}(t)$ up to time t by
 789 integrating the hazard function. Specifically, it takes the following form,

$$790 \quad S_{z,z'}(t) = \exp(-\Lambda_{z,z'}(t)) \quad (15)$$

$$791 \quad \Lambda_{z,z'}(t) = \frac{1}{N} \sum_i^N \sum_j^T \lambda_0(t_{ij}) \exp(\alpha z + \theta_3 C'_{ij} + f\{\alpha, C'_{ij} \beta_m + \sum_{k=1}^K \tau_{z'}^k \psi_k(t_{ij})\} s)(t_{ij} - \quad (16)$$

$$792 \quad t_{ij-1})$$

793

794 where $\Lambda_{z,z'}(t)$ is the cumulative hazard function. Once we obtain $S_{z,z'}(t)$, we can calculate the
 795 total effect, direct effect, and mediated effect on the scale of years,

$$796 \quad \text{total effect} = \beta_2 = \int_0^T \{S_{1,1}(u) - S_{0,0}(u)\} du, \quad (17a)$$

$$797 \quad \text{direct effect} = \beta_3 = \int_0^T \{S_{1,0}(u) - S_{0,0}(u)\} du, \quad (17b)$$

$$798 \quad \text{mediated effect} = \beta_1 \gamma = \int_0^T \{S_{1,1}(u) - S_{1,0}(u)\} du. \quad (17c)$$

799 To estimate the effect of the mediator on survival (while controlling for the effects of
 800 early adversity on the mediator), we followed similar steps. We calculated the mediator
 801 trajectory for a one unit change in social bond strength while fixing the value of early adversity
 802 exposure to one (a value that corresponds to experiencing at least one source of adversity in the
 803 cumulative adversity model or to experiencing adversity in the models for each individual source
 804 of adversity). This approach allows us to estimate the consequences of the unit change in the
 805 mediator, irrespective of the underlying reason why it might change (i.e., whether due to the
 806 effects of early adversity or some other reason), because it controls for the effects of early
 807 adversity that act independently of the mediator. Thus, the isolation effect describes how one unit
 808 change in social bond strength affects survival in years, where a one unit change represents
 809 approximately 1.7 SD for social bond strength with females and 1.4 SD for social bond strength
 810 with males; 1 SD in social bond strength with females=0.59 social bond strength units, 1 SD in
 811 social bond strength with males=0.70 units.

812

813 Causal Assumptions

814 To interpret the above models as causal, three assumptions are required. The first is the
 815 assumption of unconfoundedness. In our case, we assume that early adversity is randomly
 816 assigned to the subjects in the study. It also assumes that no unmeasured confounding variables
 817 cause variation in both early adversity and the mediator or cause variation in both early adversity
 818 and survival time, a result that follows if exposure to early adversity is largely determined by

819 natural events that are independent of the subject's individual traits, which is most likely true in
820 our case.

821 The second is the assumption of sequential unconfoundedness, which states that no
822 unmeasured confounding variables cause variation in both the mediator and survival, besides
823 the observed covariates C and the past history of the mediator M (47, 48, 113). This assumption
824 will be violated if an unmeasured variable (for instance, phenotypic or somatic quality, resulting
825 from either genetic or environmental differences that were not included in our analysis) enhances
826 or reduces both the mediator and survival.

827 We controlled for confounders as much as possible by including covariates when
828 modeling the mediators and survival, but the sequential unconfoundedness assumption is
829 essentially untestable because it invokes the possibility of an unknown and therefore unidentified
830 covariate (49). To estimate the potential effect of one or more unidentified covariates, we
831 performed a sensitivity analysis (for details see Supplementary Text: "Sensitivity analysis for
832 sequential unconfoundedness"). Specifically, we assumed the existence of an unmeasured
833 confounder between the mediator and survival that violates the sequential unconfoundedness
834 assumption (114, 115). In our simulation, the correlation between the unmeasured confounder
835 and the mediator or outcome quantifies the degree of violation of the assumption. For a set of
836 prespecified correlation values, we repeated the mediation analysis and examined the sensitivity
837 of the results to the degree of violation of the sequential unconfoundedness assumption. We
838 found that under various degrees of violation of the assumption, the mediated effect was not
839 significant (Figures S5, S6). Therefore, our conclusions are robust to the untestable assumption.
840 Details of the sensitivity analysis can be found in the supplement and Zeng, Lange, Archie,
841 Campos, Alberts and Li (40).

842 The third assumption we impose is independent censoring, i.e., we assume that the time
843 at which a subject drops out of the study prior to death is random with respect to characteristics
844 of the subject or its experience of early adversity. This assumption is likely to hold in our study
845 because female baboons are censored in only two circumstances in our study: either they
846 survived to the end of the period of data collection, or the social group in which they lived was
847 dropped for logistical reasons.

848

849 *Moderation Analysis Implementation*

850 For the moderation analysis, we modified Equation 13 by incorporating an interaction
851 term between A and M in the hazard function for survival, as follows:

$$852 \quad \lambda(t|C_{it}^S, A_i, M_{it}) = \lambda_0(t) \exp(\xi A_i + f\{\alpha, M_i(t)\} + A_i g\{\eta, M_i(t)\} + \theta_3 C_{it}^S + r_{group}^S + r_{hyrdo}^S). \quad (18)$$

853 Adding this interaction term, $A_i g\{\eta, M_i(t)\}$, in the hazard function allows us to test for the
854 interaction between early adversity and social behavior predicted by the social buffering
855 hypothesis. Therefore, this approach allows us to estimate how the effects of early adversity on
856 survival vary across different levels of the moderator. Similar to the survival model in the
857 mediation analysis, we imposed two specifications for the interaction term $A_i g\{\eta, M_i(t)\}$: (i) a
858 three-year model $g\{\eta, M_i(t)\} = \int_0^t \eta(s) M_i(s) ds$, and (ii) a one-year model, $g\{\eta, M_i(t)\} = \eta M_{it}$.
859 For the three-year model, we use $\int_0^T \eta(s) ds$ as the summary for the moderation effect. When

860 $\eta < 0$, the model indicates that a higher value for the moderator buffers the negative effects of
861 early adversity. When $\eta > 0$, the model implies that the moderator amplifies these negative
862 effects.

863

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1139 **Acknowledgments**

1140 In Kenya, our research was approved by the Kenya Wildlife Service (KWS), the Wildlife
1141 Research & Training Institute, the National Environment Management Authority
1142 (NEMA), and the National Council for Science, Technology, and Innovation
1143 (NACOSTI). We also thank the University of Nairobi, the Institute of Primate Research,
1144 the National Museums of Kenya, the members of the Amboseli-Longido pastoralist
1145 communities, the Enduimet Wildlife Management Area, Ker & Downey Safaris, Air
1146 Kenya, and Safarilink for their cooperation and assistance in the field. Particular thanks
1147 go to the Amboseli Baboon Research Project field team (R.S. Mututua, S. Sayialel, J.K.
1148 Warutere, I.L. Siodi, G. Marinka, B. Oyath) and camp staff. We also thank T. Wango and
1149 V. Oudu for their untiring assistance in Nairobi, and Jeanne Altmann for her fundamental
1150 contributions to the Amboseli baboon research. The baboon project database, BABASE,
1151 was designed and programmed by K. Pinc and is expertly managed by N.H. Learn and
1152 J.B. Gordon. For a complete set of acknowledgments of funding sources, logistical
1153 assistance, and data collection and management, please visit
1154 <http://amboselibaboons.nd.edu/acknowledgements/>.

1155

1156 **Funding:**

1157 National Institutes of Health grant R01AG053308 (SCA)
1158 National Science Foundation Integrative Organismal Systems grant 1456832 (SCA)
1159 National Institutes of Health grant P01AG031719 (SCA)
1160 National Institutes of Health grant R01AG053330 (EAA)
1161 National Institutes of Health grant R01AG071684 (EAA)
1162 National Institutes of Health grant R01HD088558 (JT)
1163 National Institutes of Health grant R01AG075914 (JT)

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1168 Funding Acquisition: FL, JT, EAA, SCA
1169 Methodology: ECL, SZ, FAC, FL, EAA, SCA
1170 Visualization: ECL, SZ
1171 Resources FL, JT, EAA, SCA
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1174 Writing—review & editing: ECL, SZ, FAC, FL, JT, EAA, SCA

1175 **Competing interests:** Authors declare that they have no competing interests.

1176 **Data and materials availability:** The reproducible code is available at
1177 [https://github.com/zengshx777/MFPCA_Codebase]. All data used in this study will be
1178 archived on Duke University Digital Repository.