



# Parenting

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# Being Cared for and Growing Up Slowly: Parenting Slows Human Life History

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## SYNOPSIS

**Objective.** For most animals, extrinsic mortality risks drive a fast life history (LH) strategy in which animals disregard risks and accelerate reproduction. Instead of perpetuating mortality driving fast LH, humans have reduced almost all mortality risks in living environments, resulting in a significant slowing of LH. Additionally, humans exhibit invested parenting which entails teaching their young survival or mortality reduction skills. Could parenting provide an additional pathway to the development and slowing of human LH? **Design.** Data reported here come from interviews and questionnaires administered to a community sample of 286 rural Chinese parents and their children when the children were on average 7, 8, and 11 years old. **Results.** Parental acceptance statistically mediates and moderates the longitudinal association between environmental adversities and children's LH. **Conclusions.** Parenting breaks the species-general contingency between mortality conditions and fast offspring LH strategies and provides an additional pathway to the development and slowing of human LH.

## INTRODUCTION

The final sentence of Darwin's *Origin of Species* (1859/1979, p. 460) reads: "From so simple a beginning, endless forms most beautiful and most wonderful have been, and are being, evolved." Parenting represents one of the endless forms that demonstrates astounding diversities (Klug & Bonsall, 2010), ranging from a male fish's incidental guarding of eggs spawned in his mating territories to deliberate prenatal and postnatal care of the young by mammalian mothers. Parental care in mammals also ranges widely from being exclusively maternal and obligatory to conscious socialization from both parents (Bornstein, 2015). Within species, phenotypic variations or individual differences in parenting are similarly observed, as evidenced by human parenting research that reports variations in parenting along different dimensions (e.g., warmth vs. control) to varying degrees of care (e.g., rejecting, neglecting, and supporting; Rohner, 2004). Associated with parenting variations are equally vast differences between species and individuals in life history (LH) strategies (Stearns, 1976), which are resource trade-off allocation plans prompted primarily by early childhood environments. When faced with environmental adversity that poses high mortality risks, animals tend to pursue fast (based

on between-species observations) or faster (phenotypically within species) LH strategies by allocating more resources to mating than to parenting. Conversely, in benign and safe environments, organisms tend to pursue slow or slower life history strategies, investing more in parenting than mating.

The links between parenting and LH strategies are twofold. First, parenting is shaped by and transmits offspring's childhood environments. In a safe and stable environment, parents are able and willing to invest more in their offspring (Chisholm et al., 1993), as parental investment yields higher returns in terms of offspring survival and reproductive success. However, in a harsh and unpredictable environment of high mortality risks, offspring receive less investment from parents who pursue mating over parenting because parental investment has diminishing returns. Thus, parenting acts as a mediator of the childhood environment and maintains the association between a benign or adverse childhood environment and slower or faster life history strategies. Second, parenting, mating, and their respective emphases are intrinsic components of slow and fast life history strategies, respectively. Parenting slows life (growth and reproduction) by exerting effort to reduce mortality risks and their contingency on fast LH, whereas mating accelerates the pace of life by disregarding mortality threats and perpetuating the environmental contingency on fast LH. Therefore, in addition to mediating between offspring's childhood environment and LH strategies, parenting also moderates the association between environments and LH, directing offspring's LH trajectory toward the slower end of the species' range and attenuating the causal contingency between childhood environmental adversities and fast LH strategies. In this study, we explore these two conceptions of LH in relation to parenting and test them using a longitudinal sample of rural Chinese parents and their children.

### ***LH Theory and Species-General Environmental Contingency***

The central tenet of LH theory is that early childhood environments shape LH strategies, which involve coordinated tuning and alignment of physiological (e.g., endocrine hormonal) and psychological (e.g., behavioral) systems to allocate finite energetic resources (Chang et al., 2019). Compromised allocations have to be made because there are always more life needs than the resources an animal can acquire, resulting in two LH trade-off strategies. A slow LH strategy allocates more energy and resources to growth and development and less to reproduction. The resulting slow and invested development entails and emanates from parenting, training, and socializing offspring. By contrast, a fast LH strategy allocates fewer resources to growth and development and more to reproduction, resulting in fast growth and early reproduction (Chang & Lu, 2018; Chen & Chang, 2012; Ellis, Figueredo, Brumbach, & Schlomer, 2009). Fast and slow LH strategies are shaped by

the extent to which extrinsic risks from the childhood living environment threaten the life and survival of the organism. Extrinsic risks such as predation or infectious diseases cause age-specific mortality and morbidity independent of the animal's survival ability and effort. The rate (harshness) and variance (unpredictability) at which extrinsic mortality affects an animal population determine the animal's LH strategy (Ellis, Figueredo, Brumbach, & Schlomer, 2009). In a safe and predictable environment with low mortality threats, a slow LH strategy maximizes physical and mental development by acquiring energy and resources and accumulating knowledge and skills to enhance future resource-capturing and reproductive competitiveness (Chang & Lu, 2017; Lu & Chang, 2019). Conversely, in an unsafe or unpredictable environment where casualties occur beyond an individual's survival efforts and abilities, a fast LH strategy prevails by attempting to grow rapidly and reproduce early before extrinsic mortality and morbidity could strike. Evolution tends to couple safe and stable childhood environments with slow LH strategies, and couple unsafe or unpredictable childhood environments with fast LH strategies (Chang et al., 2021). Understanding of this contingent coupling between the environmental input and the LH output is derived from observations and modeling based on between-species genotypes (Stearns & Rodrigues, 2020). However, the LH principle can also explain within-species phenotypic variations in a manner that aligns with the between-species causal contingencies (Del Giudice, 2020; Galipaud & Kokko, 2020; Kuzawa & Bragg, 2012). For example, early childhood adversities, indicated by environmental harshness or unpredictability, are associated with biobehavioral phenotypes that manifest faster LH strategies (e.g., early onset of menarche and sexual activities, Ellis, 2004; risk-taking; Lu & Chang, 2019; inert disease control effort; Lu et al., 2021).

### ***Parenting Mediates Environment and Its Contingency on LH***

However, human children initially encounter the external environment through the care provided by their parents (Bornstein, 2019). Maternal and parental care evolved to protect offspring from mortality threats such as predation (Bowlby, 1969/1982), is fundamentally sensitive to and transmits extrinsic mortality conditions (Chisholm et al., 1993), as well as the parents' ability and willingness to mitigate mortality threats (Lu, Wang, Liu, & Chang, 2022). Parenting thus acts as an intermediary that conveys external information and influences the calibration of LH strategies. According to Belsky, Steinberg, and Draper (1991), the actual environment and parenting-mediated experience should have similar effects on the child's LH calibrations. Parental care received during the first 7 years of a child's life exerts a foundational environmental influence on the development of the child's LH strategies (Belsky, Steinberg, & Draper, 1991). It is postulated in the literature that a benign and stable environment aligns with consistent and

supportive parental caregiving and with slower LH calibration in the child, including affiliative and communal sociality (Belsky, Steinberg, & Draper, 1991; Chisholm et al., 1993; Del Giudice & Belsky, 2011). Conversely, environmental harshness and unpredictability are associated with harsh, inconsistent, or dismissive parenting and with faster LH strategies and antagonistic sociality in the child (Belsky, Steinberg, Houts, Halpern-Felsher, & NICHD Early Child Care Research Network, 2010; Chen & Chang, 2012).

Other literature further suggests that unsupportive parenting, as well as parental absence, indicates and is experienced by a child as environmental harshness (Warren & Barnett, 2020). Similarly, parental behavioral inconsistency or actual parental transitions and changes signify environmental unpredictability and shape faster LH accordingly. “Rearing context shapes life history, which is itself systematically related to patterns of pair bonding and parenting” (Belsky, Steinberg, & Draper, 1991, p. 649). Thus, parenting variables representing parent-child interactions are also conceptualized as part of environmental harshness (Suor, Sturge-Apple, Davies, & Cicchetti, 2017; Warren & Barnett, 2020) or unpredictability (Brumbach, Figueredo, & Ellis, 2009; Sung et al., 2016), and in conjunction with other environmental variables, they serve as independent predictors of LH (Belsky, Houts, & Fearon, 2010; Belsky, Steinberg, Houts, Halpern-Felsher, & NICHD Early Child Care Research Network, 2010; Brumbach, Figueredo, & Ellis, 2009; Ellis & Essex, 2007). All these variables directly affect LH strategies and related outcomes. On this argument, we propose the following hypothesis:

**Hypothesis 1:** Parenting, such as parental acceptance and rejection, would statistically mediate the relation between environmental adversity (safety) and fast (slow) LH strategies.

### ***Parenting Reduces Extrinsic Mortality and Slows LH***

Parenting is also hypothesized to statistically moderate the relation between environmental adversity and fast LH strategies. Parental acceptance and supportive parenting are expected to weaken the positive association between environmental adversity and fast LH, whereas parental rejection or harsh parenting is believed to maintain or strengthen the same association. Most extrinsic risks cause fast LH in other animals by inflicting indiscriminate and uncontrollable casualties on the adult population. The human population is no longer uniformly impacted by similar risks from the external environment; instead, individual people reduce and control extrinsic mortality threats through their own abilities and efforts (Lu et al., 2021; Lu, Wang, Liu, & Chang, 2022). Simultaneously, the principal selection pressure for human

brain evolution has concentrated on intraspecific competition (Alexander, 1990), resulting in diverse individual responses to extrinsic risks (Nettle, 2006). Throughout much of human evolution, extrinsic risks from nature have become manageable and reducible, no longer uniformly affecting the entire adult population but mitigated through individual abilities and efforts. This process of addressing otherwise insurmountable challenges is integral to the evolution of human slow LH strategies, and it is closely linked to the enlargement of the human brain, which itself represents a slow LH trait and trade-off. Additionally, the exponential development of the human brain allows for extended slow LH phenotypes (Lu, Wang, Liu, & Chang, 2022) that surpass the limitations of biobehavioral adaptations in effectively reducing extrinsic risks and slowing down human LH (Smith & Tompkins, 1995).

Parenting is integral to slow LH (Kaplan, 1996). It protects offspring from mortality threats (Bowlby, 1969/1982). Parenting also serves the function of transmitting culture and knowledge from one generation to the next (Bornstein, 2012). Hence, it follows that parenting plays a pivotal role in mortality reduction and the slowing of LH, initiated by parents and instilled in and carried on by offspring. As humans provide most extensive parental investment (Geary & Flinn, 2001), parenting can foster a mind-set focused on reducing mortality and slowing LH, beginning from the early stages of a new generation's development and transmitted across generations. Through supportive parenting that prioritizes mortality reduction, each generation can contribute to the slowing of LH that differs from the trajectory shaped solely by the living environment in the absence of positive parenting. Hence, parenting has the potential to steer children toward a different developmental pathway, one of slowing LH, where the connection between extrinsic mortality and fast LH is weakened or severed through positive and supportive parenting. On this theoretical ground, we propose our second hypothesis:

**Hypothesis 2:** Parental acceptance and rejection would statistically moderate the association between environmental adversity (safety) and fast (slow) LH strategies, with parental acceptance weakening the association and with parental rejection maintaining or strengthening the association.

### ***The Present Study***

We conducted a longitudinal study with 286 Chinese rural parents and their children to examine our hypotheses related to LH and parenting. The study involved gathering data at three time points. At around 7 years old, childhood environmental adversities were assessed through reports provided by the children and their parents. A year later, we collected

information on parental acceptance and rejection, reported by both the children and their parents. Fast offspring LH behavioral profiles, including aggression, impulsivity, and risk-taking tendencies, were measured through self-reports when the children reached 11 years of age. To test our hypotheses, we conducted statistical mediation and moderation in relation to parental acceptance. The mediation and moderation hypotheses form the statistical model depicted in [Figure 1](#). The statistical moderation is expected to move in the direction of higher levels of parental acceptance weakening the association between environmental adversities and fast LH behavioral profiles, and lower levels of parental acceptance (i.e., parental rejection) maintaining or strengthening this association.

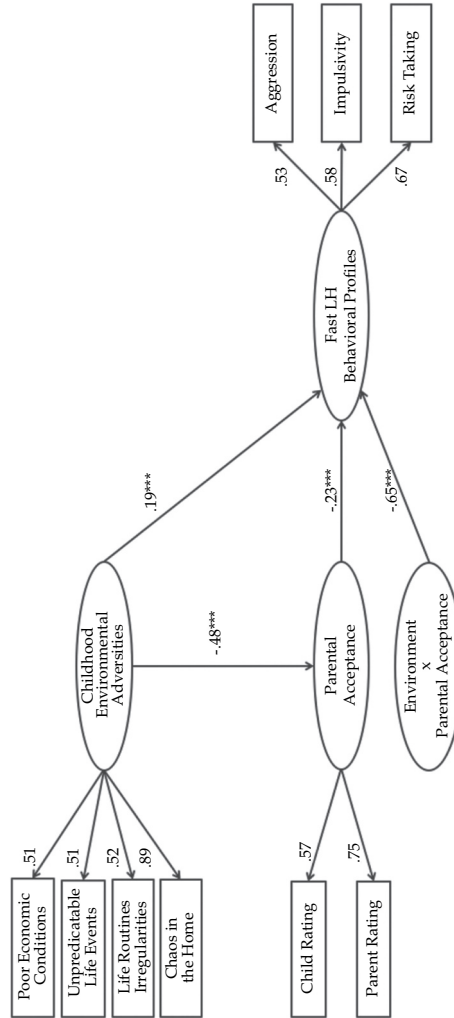
## METHOD

### *Sample*

A community sample was collected from four randomly selected rural townships in Henan Province, chosen for their high population density, rural population, and relatively low per capita income (National Bureau of Statistics, 2020). The sample consisted of 286 children (137 males;  $M_{age} = 10.97$ ,  $SD = .75$ ) and their parents (88% mothers and 12% fathers;  $M_{age}$  of mothers = 33.67,  $SD = 4.93$ ,  $M_{schooling}$  years of mothers = 9.76,  $SD = 2.62$ ;  $M_{age}$  of fathers = 35.13,  $SD = 5.42$ ,  $M_{schooling}$  of fathers = 9.49,  $SD = 2.17$ , at the first data collection). This study reports data from three observations taking place when the children's age was 7 years ( $M = 6.97$ ,  $SD = .74$ ), 8 years ( $M = 7.95$ ,  $SD = .74$ ), and 11 years ( $M = 10.97$ ,  $SD = .75$ ). The retention rate for participants across three observations was 83%. Participants who provided complete data for the entire study did not differ from the initial sample on any of the measures used.

### *Procedures*

At 7 years, data collection was carried out by two interviewers who were unaware of the study's hypotheses. Face-to-face interviews were conducted separately with the participating children and their parents at their homes to ensure privacy. Standardized questions were read aloud by the interviewers, and the participants' responses were recorded. At 8 years, the same interview procedures were followed. At 11 years, the measures used were gathered from the participating children through self-response questionnaires. These questionnaires were distributed to the children in schools, and their completed responses were collected. As a token of gratitude for their participation, younger children received small gifts, and older children and parents received modest monetary compensation.



**Figure 1.** Childhood environmental adversities, parental acceptance, and their interaction in relation to fast LH behavioral profiles.  $^{***}p < .001$ .



## Measurement

Within empirical research on human LH, assessment of extrinsic mortality and morbidity levels (harshness) and variations (unpredictability) involves sampling proxies from the current living environment. These proxies are believed to indicate harsh and unpredictable environmental conditions experienced by our ancestors (Young, Frankenhuis, & Ellis, 2020). Because harshness and unpredictability are highly correlated and both are predictive of LH (Lu, Wang, Liu, & Chang, 2022), consistent with the literature (Lu et al., 2021), we combined the two variables into a single construct, environmental adversities. The following four indicators compose the construct.

**Poor Economic Conditions.** Parents responded to seven items concerning economic hardships in the home, such as purchasing cheaper products during their child's upbringing or facing challenges in paying bills. Responses were rated on a 4-point scale (1–4: *almost never, sometimes, often, almost always*), yielding an internal consistency reliability estimate Cronbach's  $\alpha = .79$ .

**Unpredictable Life Events.** Children were asked to recall and report the number of times they experienced unpredictable and negative life events, including severe illness, accidents or injuries, and the death or injuries of significant individuals. These items were adapted from the Social Readjustment Rating Scale (Holmes & Rahe, 1967). The total count of recalled events served as the variable, which, as a count, does not yield an internal consistency reliability estimate.

**Life Routine Irregularities.** Children rated 12 items measuring irregularities in their daily routines, such as inconsistent family dinner arrangements or parents' absence at bedtime. Responses were recorded on a 4-point scale (1–4: *almost never, sometimes, often, almost always*), and Cronbach's  $\alpha = .69$ .

**Chaos in the Home.** Parents responded to ten items adapted and modified from the Confusion, Hubbub, and Order Scale (Matheny, Wachs, Ludwig, & Phillips, 1995) to assess the presence of confusion, chaos, and disorder in the home environment during the child's upbringing. Statements included descriptions like "our home was like a zoo" or "there was often commotion in our household." Participants rated these statements on a 4-point scale ranging from 1 (*not at all like our home*) to 4 (*very much like our home*). The items were worded and, if necessary, reverse coded to reflect the presence of chaos and disorder. Cronbach's  $\alpha = .74$ .

**Parental Acceptance.** The child and parent version of the 24-item Parental Acceptance-Rejection Questionnaire-Short Form (PARQ-SF; Rohner, 2005) was used to measure the frequency of perceived positive (warmth and support) and negative (neglect, indifference, and rejection) parenting behaviors. This measure of parenting is best suited for an evolutionary LH investigation of the rudimentary functioning of parenting that, consistent with the PARQ-SF, is defined as a unidimensional continuum ranging from positive parenting or parental warmth and acceptance to negative parenting or child neglect and rejection. Children rated their parent and the parent self-rated on a 4-point scale: 1 = *never or almost never*, 2 = *rarely*, 3 = *sometimes*, or 4 = *almost always*. We used the total acceptance-rejection scale, which is computed as the average of eight warmth-affection items (e.g., child version: “parent says nice things about me,” or parent version: “I say nice things about my child”), six hostility-aggression items (e.g., child version: “parent punishes me severely when angry,” or parent version: “I punish my child severely when I am angry”), four rejection items (e.g., “parent lets me know that I am not wanted”), and six neglect-indifference items (e.g., “parent is too busy to answer my questions”). With reverse coding of negative parenting items, higher scores indicate more acceptance and less rejection. Cronbach’s  $\alpha$  = .80 and .81 for the child and parent versions, respectively.

**Aggression.** In line with the fast-slow pace of life, two distinct forms of sociality emerge. One is characterized by affiliative, altruistic, and mutualistic social interactions, which prioritize future cooperation and long-term reciprocity. In contrast, the other form is marked by antagonistic, exclusive, and utilitarian sociality, serving immediate self-focused survival concerns in precarious environments (Chang et al., 2019; Figueredo et al., 2018). The present study utilized aggression as an indicator of fast life history sociality. Children completed a 32-item Problem Behavior Frequency Scale (PBFS; Farrell, Sullivan, Goncy, & Le, 2016) that measures delinquent and aggressive behaviors. They rated on a 6-point frequency rating scale from 1 (*never*) to 6 (*20 or more times*) in the last 30 days regarding behaviors such as “started a fight between other people,” “picked on someone,” and “cheated on a test.” Cronbach’s  $\alpha$  = .93.

**Impulsivity.** To capture impulsivity, children completed the 30-item Barratt Impulsiveness Scale (Patton, Stanford, & Barratt, 1995). The items have been previously used to form a single factor among 10- to 13-year-olds (Steinberg et al., 2008). Sample items include “I act on the spur of moment” and “I say things without thinking.” Participants rated each item on a 4-point scale, ranging from 1 (*almost never*) to 4 (*almost always*). Cronbach’s  $\alpha$  = .76.

**Risk Taking Tendency.** Risk taking and risk-taking tendency are behavioral traits that “may covary in predictable ways with life history traits between individuals” (Sear, 2020, p. 514). The Benthin Risk Perception Scale (Benthin, Slovic, & Severson, 1993) was adapted to measure risk taking tendency. Eight out of the original 11 risky activities were selected, considering their relevance to the rural Chinese child population. These activities included smoking cigarettes, drinking alcohol, riding with a drunk driver, vandalizing property, going to dangerous places, stealing from stores, engaging in gang fights, and using weapons to threaten someone. For each activity, children responded to four questions on a 4-point scale: “How scary are the things that could happen?” (1 = *not scary at all*; 4 = *very scary*, reverse coded); “To what extent are you at risk of something bad happening?” (1 = *very much*; 4 = *not at all*); “How would you compare the benefits of this activity with the risks?” (1 = *the risks are far greater than the benefits*; 4 = *the benefits are far greater than the risks*); “If something bad happened because of this activity, how serious would it be?” (1 = *not at all serious*; 4 = *very serious*, reverse coded). The construct was formed by averaging the ratings across the four questions for each of the eight activities, with a higher score indicating a greater inclination to take risks, independent of the actual opportunity to do so (Duell et al., 2016). Cronbach’s  $\alpha = .94$ .

## RESULTS

Table 1 presents the means, standard deviations, and correlations of the variables used in the study. The correlations were based on different informants (i.e., children and parents) and over time lags of up to 5 years. The coefficients showed good convergent and discriminant validity with variables measuring the same trait by different informants more highly correlated with each other than with variables from the same informant tapping different

**Table 1.** Means, standard deviations, and zero-order correlations of variables used in the study.

	1	2	3	4	5	6	7	8	9
Childhood Environmental Adversities									
1. Poor Economic Conditions	–								
2. Unpredictable Life Events	.32***	–							
3. Life Routine Irregularities	.12*	.13*	–						
4. Chaos in the Home	.27***	.27***	.10	–					
Parental Acceptance									
5. Child Rating	–.12*	–.16**	–.37***	–.13*	–				
6. Parent Rating	–.19**	–.18**	–.12*	–.35***	.16**	–			
Fast LH Behavioral Profiles									
7. Aggression	.004	.08	.24***	.19**	–.11 <sup>†</sup>	–.13*	–		
8. Impulsivity	.12*	.17**	.26***	.04	–.20***	–.05	.27***	–	
9. Risk Taking	.13*	.23***	.32***	.16**	–.19**	–.14*	.33***	.33***	–
<i>M</i>	1.90	2.00	1.59	2.04	3.02	3.02	1.12	1.98	1.67
<i>SD</i>	.57	2.99	.41	.51	.43	.38	.25	.32	.55

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

traits. Inter-trait correlations are also aligned with our theoretical rationale. Specifically, indicators of environmental adversities (e.g., poor economic conditions and chaos in the home, both of which were obtained from parents) longitudinally and significantly correlated with indicators of fast LH behavioral profiles reported by the children. These indicators were also correlated with parental acceptance in the expected directions. These and other variables under study showed no gender differences except that boys scored marginally higher on aggression than girls. We also computed zero-order correlations for boys and girls separately and found no sex differences in these correlations.

Because we used latent constructs rather than directly observed variables, we conducted structural equation modeling using *Mplus* 7.0 (Muthén & Muthén, 1998/2012), and we used full information maximum likelihood estimation to account for missing data (Schafer & Graham, 2002). We adopted the following recommended cutoff values to assess model fit: chi-square to degrees of freedom ratio ( $\chi^2/df < 5.0$ ; Kline, 1998), Comparative Fit Index (CFI  $\geq .90$ ; Marsh, Balla, & McDonald, 1988), Tucker-Lewis Index (TLI  $\geq .90$ ; Marsh, Balla, & McDonald, 1988), Root Mean Squared Error of Approximation (RMSEA  $\leq .08$ ; Browne & Cudeck, 1993), Standardized Root Mean Square Residual (SRMR  $\leq .08$ ; Hu & Bentler, 1999), and minimum factor loading (loading  $> .32$ ; Tabachnick & Fidell, 2013; loading  $> .50$ ; Bagozzi & Yi, 1988).

We first tested the model in Figure 1 without the interaction term representing the moderation hypothesis. The goodness of fit statistics ( $\chi^2/df = 1.89$ , CFI = .93, TLI = .90, RMSEA = .051, SRMS = .061) of the model met the recommended cutoff values for adequate model fit. We then included the interaction construct representing the statistical interaction or moderation hypothesis. The two concerning variables forming an interaction or moderation (environmental adversity and parental acceptance) are normally treated as correlated with unspecified causal directions. Because we also formulated a mediation hypothesis involving parental acceptance as the mediator, in the model presented in Figure 1, environmental adversity and parental acceptance were specified as one leading to the other rather than as two correlates. Statistical estimation of the interaction or moderation effect remains the same whether the two main effect variables, environmental adversity and parental acceptance, represent correlation or causation. We computed the interaction construct by using the default approach of *Mplus* rather than manually pairing the concerning indicators of the two constructs and multiplying them (Marsh, Wen, & Hau, 2004). The *Mplus* default approach does not provide goodness of fit statistics (Maslowsky, Jager, & Hemken, 2015; Muthén & Muthén, 1998/2012). Instead, *Mplus* provides a measure, D, of relative fitness of the interaction model compared to the main-effect-only model without the interaction term. D is the difference of the log-likelihood values of the two models ( $D = -2 \times [(\log\text{-likelihood for the main effect model})$

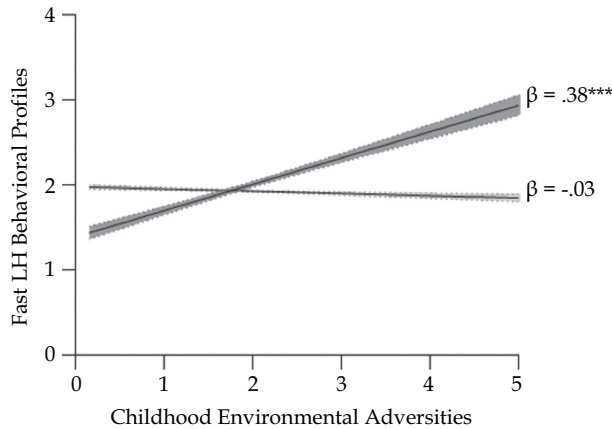
- (log-likelihood for the interaction model)]; Muthén & Muthén, 1998/2012);  $D$  follows a chi-square distribution with  $df$  being the difference in the number of estimated parameters between the two models, which, in the present case, was 1. The log-likelihood for the main-effect-only or baseline model was  $-3147.90$  and that for the interaction model was  $-3077.74$ ;  $D = 140.32$ ,  $p < .001$ . The statistically significant reduction of log-likelihood value indicates substantial improvement in data fit by the hypothesized moderation over the baseline estimation.

As shown in Figure 1, all the parameter estimates were in the expected directions and were statistically significant. First, the factor loadings were moderate in part because they were obtained from different informants and some of the proxies of environmental adversity are not expected to be highly correlated in approximating diverse environmental conditions. However, the loadings were adequate, more than meeting the minimum and stringent standard for adequate measurement (Bagozzi & Yi, 1988; Tabachnick & Fidell, 2013). Second, parameter estimates of the structural model were consistent with our hypotheses. Environmental adversity was negatively associated with parental acceptance ( $\beta = -.48$ ,  $p < .001$ ) and positively predicted fast LH behavioral profile ( $\beta = .19$ ,  $p < .001$ ), whereas parental acceptance was negatively related with fast LH profiles ( $\beta = -.23$ ,  $p < .001$ ). The mediating effect of parental acceptance between environmental adversity and fast LH profiles was significant ( $\beta = .22$ , 95% Confidence Interval = [.08, .43]) based on a bootstrapping procedure with 2000 resamples and the maximum likelihood estimation. This finding supports our first hypothesis that parental acceptance mediates the relation between environmental adversity and fast LH strategies, providing support for the species-general contingency pathway.

Finally, the interaction between environmental adversity and parental acceptance was significant ( $\beta = -.65$ ,  $p < .001$ ), supporting our second moderation hypothesis. Figure 2 displays the simple slopes of environmental adversity on fast LH behavioral profiles at  $+1 SD$  ( $\beta = -.03$ ,  $ns$ ) and  $-1 SD$  of parental acceptance ( $\beta = .38$ ,  $p < .001$ ). As predicted, parental acceptance attenuated or nullified the positive association of environmental adversity to fast LH at higher levels of parental acceptance and maintained and amplified the association at lower levels of parental acceptance (i.e., parental rejection).

## DISCUSSION

Extrinsic mortality risks indiscriminately affect a species, irrespective of individual members' abilities and efforts to survive (Ellis, Figueredo, Brumbach, & Schlomer, 2009). According to this definition, a successful or evolutionarily stable strategy for survival in an adverse environment with high mortality



**Figure 2.** Simple slopes and 95% confidence bands of the regression of fast LH behavioral profiles on childhood environmental adversities at 1 *SD* above (light) and 1 *SD* below (darkened) the mean of parental acceptance.  $^{***}p < .001$ .

threats would involve disregarding the extrinsic risks and accelerating growth and reproduction to increase the chances of surviving to reproductive age. Similarly, a winning strategy would not heavily invest in offspring quality through parenting and training, as those factors do not reduce uncontrollable mortality threats. Instead, focusing on producing a greater quantity of offspring through low parenting and high mating effort may increase the likelihood of some offspring surviving by chance. This fast LH strategy is selected for high-mortality living environments. Most animals, in the course of evolution, tend to disregard extrinsic mortality threats rather than attempting to reduce them. As a result, extrinsic mortality conditions remain unchanged, the fast LH strategy persists, and the species-general contingency between high mortality environments and fast LH continues.

However, human LH development exhibits certain exceptions or additional pathways beyond the species-general LH principle described above. Over the past two million years of evolution, humans have not simply disregarded but have significantly reduced extrinsic casualties resulting from almost all mortality factors, including predation, natural disasters, food shortages, and infectious diseases (Alexander, 1990). The effort to reduce extrinsic mortality is rooted in and reinforces the slow LH strategy observed in humans, which includes delayed reproduction, prolonged child development, and notably an enlarged brain (Chen & Maklakov, 2012; Hill & Kaplan, 1999). The exponential development of the human brain extends the biobehavioral phenotypes of slow LH to encompass culture (e.g., collectivism) and technology (e.g., medicine). These extended slow LH phenotypes (Lu, Wang, Liu, & Chang, 2022) significantly accelerate the reduction of extrinsic mortality and the slowing of human LH.

Considering these factors collectively, it can be inferred that humans have taken an additional pathway toward slow LH, and as proposed and tested in the present study, parenting plays a crucial role in this pathway. Parenting, being an integral component of slow LH (Kaplan, 1996), is further emphasized in the case of humans through other contingent slow LH events. These events include premature birth, which is an adaptation to the enlarged cranium, an extended childhood (the longest among animals), and postnatal brain development that accounts for three-fourths of adult brain size. These interconnected LH events provide human parenting with the necessary time (a long childhood) and space (brain plasticity) to socialize offspring with a slower LH strategy than what would be predicted based solely on the species-general mortality-LH contingency. The significant moderation finding supportive of our second hypothesis demonstrates that parental acceptance or positive parenting weakens or nullifies the longitudinal association between environmental adversities and fast LH behavioral profiles. Also consistent with our hypothesis, parental rejection or low levels of parental acceptance perpetuates and exacerbates the association between adverse childhood environments and fast LH behavioral profiles. Caregiving, parenting, and maternal socialization, in general, aim to reduce mortality risks (e.g., protection from predation; Bowlby, 1969/1982) and embody slow LH strategies (Kaplan, 1996). Successful maternal and parental socialization reduces environmental adversity and its impact on offspring LH, in part, by instilling the belief that, even in the face of adversity, the external environment can eventually be controlled (Lu, Liu, & Chang, 2022). Such a positive worldview aligns with cognitive components of slow LH strategies, including insight, planning, and control (Figueredo et al., 2018). When facing environmental adversity, successfully socialized human offspring should redirect their LH behavioral pathway from the externally predicted faster track (i.e., disregarding mortality and accelerating reproduction) to a slower pathway aimed at reducing mortality risk and delaying reproduction. Unsuccessful parenting, on the other hand, merely transmits the effect of environmental adversities on LH. Consistent with the mediation finding supporting our first hypothesis, parenting serves the rudimentary function of mediating environmental or mortality conditions and transmuting their effects on LH by adhering to the species-general contingency between mortality and fast LH. In harsh and unpredictable environments, parenting transmits and even intensifies faster LH, whereas in stable and benign environments, parenting promotes slower LH. This finding implies that, in addition to providing an additional slow LH pathway, parenting also serves as a conduit that merely transmits environmental effects on offspring LH calibration.

The current study is subject to several limitations. First, we did not account for potential genetic influences on LH strategies, which can exhibit heritability (Braendle, Heyland, & Flatt, 2011). Genetic confounding poses



a validity threat, particularly in evolutionary studies due to the studies' focus on distal factors, as well as in parenting research because parenting transmits both environmental and heritable effects across generations. Future investigations could consider employing twin or sibling designs to differentiate between environmental and potential genetic influences on LH, while also assessing LH in both children and parents to disentangle cross-generational heritability. Future studies could also include both parents to determine the unique contribution of maternal and paternal parenting in slowing offspring LH. Second, our results, particularly certain factor loadings, displayed moderate values, suggesting that we may not have fully or accurately captured the underlying distal evolutionary processes under investigation. However, this limitation was partially mitigated by the utilization of multidimensional, multi-informant, and longitudinal data, which likely yielded more nuanced results with reduced inflation caused by method variance. The absence of measurements pertaining to the biological aspects of LH represents another noteworthy limitation. However, psychological LH research should focus on behavior and cognition that lead to biological LH outcomes. Despite these and other limitations, our study represents one of the initial theoretical and empirical endeavors to establish a parenting-moderated LH pathway, aiming to elucidate the slowing of human LH and expand the scope of parenting research.

## **IMPLICATIONS FOR PRACTICE AND THEORY**

The present study offers a novel perspective on human LH development and research. For most animal species, because extrinsic mortality risks engender a fast LH strategy characterized by disregarding these risks, mortality risks and fast LH both persist in the animal kingdom. Human LH has slowed significantly, resulting from and reinforcing the reduction of mortality risks. Parenting entails teaching the young survival or mortality reduction skills, which, for humans, extend to culture and technology, ranging from stone tools to AI robotics, and from traditional herbal remedies to modern pharmacology. Through such extended phenotypes, parenting has significantly reduced extrinsic mortality risks and slowed human LH. Research on humans should incorporate parenting and cultural transmission into a more comprehensive investigation of LH and its human evolutionary developmental process.

## **AFFILIATIONS AND ADDRESSES**

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Each author signed a form for disclosure of potential conflicts of interest. No authors reported any financial or other conflicts of interest in relation to the work described.

### ***Ethical principles***

The authors affirm having followed professional ethical guidelines in preparing this work. These guidelines include obtaining informed consent from human participants, maintaining ethical treatment and respect for the rights of human or animal participants, and ensuring the privacy of participants and their data, such as ensuring that individual participants cannot be identified in reported results or from publicly available original or archival data.

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