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"Fast" women? The effects of childhood environments on women's developmental timing, mating strategies, and reproductive outcomes

Tran Dinh [a,b,*], Martie G. Haselton [b,c], Steven W. Gangestad [a]

[a] Department of Psychology, University of New Mexico, Albuquerque, NM, USA
[b] Department of Psychology, University of California, Los Angeles, Los Angeles, CA, USA
[c] Department of Communication, University of California, Los Angeles, CA, USA

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ABSTRACT

The fast-slow paradigm of life history theory has been a popular approach to individual differences in the evolutionary behavioral sciences. Currently, however, the fast-slow paradigm faces several theoretical and empirical challenges. Motivated by questions regarding the validity of certain assumptions of the paradigm, the current study provides an empirical investigation of human female "fast" versus "slow" strategies. In a sample of 1867 women recruited using MTurk, we use structural equation modeling (SEM) to test whether childhood exposure to different environmental variables had unique effects on proposed life history traits, whether mediated by—or independent of—pubertal timing. Models also test whether the proposed life history traits covary with one another as expected by the paradigm. Data reveal that exposure to violence and poor health in particular, but not environmental harshness or unpredictability in general, had significant effects on pubertal timing. Pubertal timing appeared to mediate effects of childhood environments on age at sexual debut, but not any other adult outcome (e.g., sociosexual orientations, reproductive outcomes). Some associations with mating strategies were incompatible with assumptions of the prevailing fast-slow paradigm; for instance, greater short-term mating orientation was positively associated with childhood socioeconomic status and negatively associated with offspring number. These results highlight the need for a new or revised theoretical approach to understanding developmental, mating, and reproductive strategies.

1. Introduction

Life history theory is a conceptual framework for understanding how organisms adaptively allocate limited temporal and energetic resources towards survival, growth, and reproduction. Reproductive success—the primary target of selection on individuals—is a function of allocation strategies, which involve trade-offs in rates of survival and fertility. For instance, greater investment in survival or growth comes at a cost of reproducing less now. Reproducing now directly adds to total reproductive success but at increased risk of mortality and, consequently, loss of reproduction thereafter. Selection on organisms result in species-typical, age-specific survival and fertility curves that maximize total reproductive effort, integrated across the lifespan (Charnov, 1993).

A critical question addressed by life history theory is when the transition from a pre-reproductive to a reproductive phase optimally occurs. During the pre-reproductive (juvenile) period, organisms allocate energy to tissue differentiation, growth, and the acquisition of other forms of "embodied capital" (e.g., learning) that enhance capacities to acquire energy, compete for mates, and parent future offspring. These allocations delay reproduction, an opportunity cost. Longer post-juvenile survival and greater reproductive capacity later allow these costs to pay off. With body weight controlled, species that mature at later ages tend to have lower juvenile and adult mortality rates (Promislow & Harvey, 1990). Species that mature at earlier ages tend to produce more offspring at given ages; offspring are less likely to survive, in part because parents invest less in their quality. Selection gives rise to species-typical life histories, across which a "fast-versus-slow" dimension accounts for much variation in life history parameters (e.g., age of first reproduction, rate of reproduction, mean survival; Dobson & Oli, 2007).

Selection on individuals to make condition-dependent life history allocations may also result in within-species variation in developmental outcomes. Information from an individual's early life environment may

Abbreviations: SES, Socioeconomic status; STMO, Short-term mating orientation; LTMO, Long-term mating orientation; SEM, Structural equation modeling.

* Corresponding author at: Department of Psychology, MSC03 2220 1, University of New Mexico, Albuquerque, NM, 87131, USA.
E-mail address: trandinh@unm.edu (T. Dinh).

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index conditions (such as higher mortality risk) that, ancestrally, may have favored in humans a relatively fast life history or a relatively slow life history. Environmental conditions, then, may adaptively shape the development of a coordinated set of strategies that follow a fast-versus-slow dimension. This within-species fast-slow paradigm of life history strategies has been widely adopted within evolutionary psychology to explain variation in behavioral and psychological traits across humans (e.g., Ellis, Figueredo, Brumbach, & Schlomer, 2009; Belsky, Schlomer, & Ellis, 2012; Griskevicius, Delton, Robertson, & Tybur, 2011; Griskevicius et al., 2013; Del Giudice, Ellis, & Shirtcliff, 2011; see also Chisholm, 1993, 1996; see review in Del Giudice, Gangestad, & Kaplan, 2015).

Nonetheless, both theoretical and empirical issues persist. Some authors have argued that core commitments to life history approaches in evolutionary psychology may not be compatible with modeling conducted within the broader framework of life history theory in evolutionary biology (Nettle & Frankenhuis, 2020; Sear, 2020; see also Nettle & Frankenhuis, 2019; Frankenhuis & Nettle, 2020; Stearns & Rodrigues, 2020). Others have argued that the fast-slow dimension that characterizes life history variations across species may not characterize differences between individuals within a species (e.g., Zietsch & Sidari, 2020; Stearns & Rodrigues, 2020; see also Del Giudice, 2020, on the "ecological gambit" that cross-species covariation may have parallels with within-species covariation).

And within life history approaches in evolutionary psychology, there remain questions of which early-life experiences calibrate the timing of reproductive maturation; how specific early experiences influence adult mating proclivities and reproductive outcomes; whether the timing of reproductive maturation mediates childhood influences on mating and reproductive strategies; and why selection has shaped strategies contingent on specific early-life experiences. Our study focuses on these questions. Current theory has emphasized female pubertal timing as a life history outcome, as it marks a clear, important transition from a pre-reproductive phase of growth and preparation for reproduction to a phase during which effort is directly allocated to reproduction (e.g., Ellis, 2004). Therefore, the current study examines these questions in women. Prior to introducing our empirical study, we discuss theoretical underpinnings of dominant perspectives on fast-slow life histories in the evolutionary psychological literature. We provide a brief conceptual critique of these perspectives and discuss prior research that questions some empirical links expected by the perspectives.

1.1. Theoretical underpinnings of dominant perspectives on the fast-slow dimension in evolutionary psychology

1.1.1. Effects of harshness, unpredictability, and paternal investment

Leading theories on human life history variations propose that pubertal timing and parental investment strategies are driven by local *extrinsic mortality rates* (e.g., Ellis et al., 2009). Extrinsic mortality reflects threat of mortality unaffected by an organism's investment decisions (e.g., Charnov, 1993; Promislow & Harvey, 1990; Stearns, 1992).

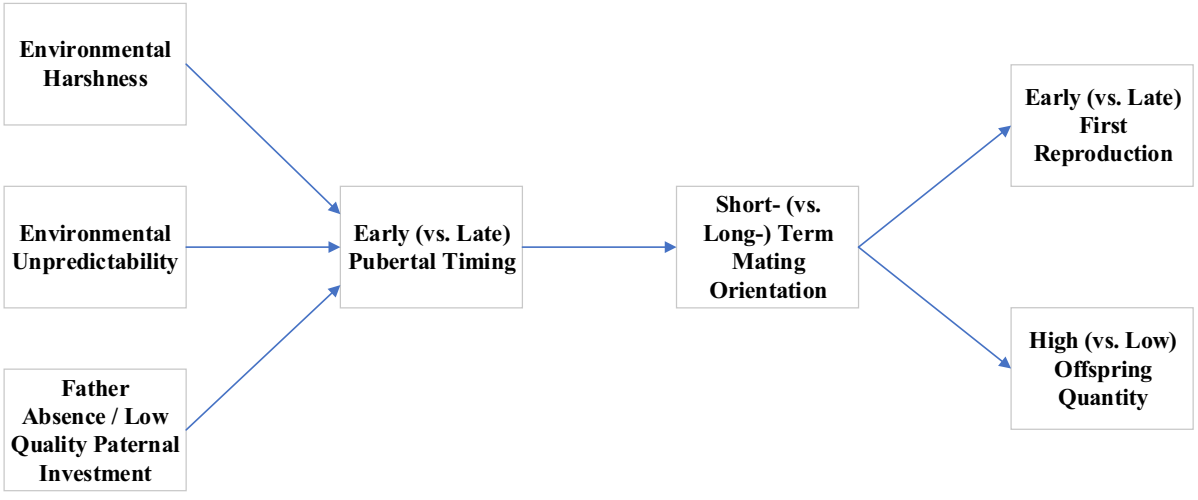
A key question concerns what conditions affect or index local extrinsic mortality risk. Ellis et al. (2009) argued that developmental trajectories are sensitive to two fundamental dimensions of environmental risk: harshness and unpredictability. Cues of harshness in modern environments include low socioeconomic status (SES), neighborhood dangers, exposure to violence, and low-quality or absent parenting. These conditions presumably affect (or index other conditions that affect) mortality rates. Cues of environmental unpredictability include frequent parental and residential changes, and they importantly influence the predictability of mortality risks. According to this perspective, childhood conditions of harshness and unpredictability should have ancestrally favored conditional adaptations leading individuals to adopt faster life history strategies (e.g., earlier puberty, earlier sexual debut, low parental investment, prioritization of offspring quantity over quality). Childhood conditions that lack harshness and unpredictability should favor slower life history strategies (e.g., later puberty, later sexual debut, high parental investment, prioritization of offspring quality over quantity).

Ellis (2004) furthermore argued that parenting quality among fathers provides daughters with unique information about the reliability of paternal investment from future male partners. Thus, father absence and poor-quality fathering purportedly also affect daughters' pubertal acceleration and development of fast life history trajectories. (For related and alternative perspectives, see Belsky et al., 2012; Boyce & Ellis, 2005; Chisholm, 1993, 1996; Del Giudice, Ellis, & Shirtcliff, 2011; Rickard, Frankenhuis, & Nettle, 2014. For evidence that father absence may not predict pubertal acceleration in non-Western settings, see Sear, Sheppard, & Coall, 2019.)

1.1.2. Mating strategies as key components of life history trajectories

Prominent versions of the fast-slow paradigm claim a critical association between reproductive timing and mating strategy. According to these ideas, early puberty should precipitate "short-term" mating strategies, leading to relationship instability and multiple partners; late puberty should precipitate "long-term" mating strategies, leading to relationship stability and few partners (Belsky, Steinberg, & Draper, 1991; Ellis, 2004; Ellis et al., 2009). The basis for this argument is that fast life history strategies include prioritizing offspring quantity and genetic diversification, whereas slow life history strategies include prioritizing offspring quality. Offspring quantity is presumably promoted by short-term mating—e.g., uncommitted sex with multiple partners—and low parental investment (Chisholm, 1993).

In sum, dominant perspectives on human life history trajectories within evolutionary psychology propose the following links:



1.2. Challenges to prevailing conceptualizations

1.2.1. Returns to mortality reduction efforts

In conceptualizations of the fast-slow paradigm, the extrinsic mortality rate is one piece of the total mortality rate—that which is completely uncontrolled by the organism (e.g., Charnov, 1993). The extrinsic (versus total) mortality rate is assumed to be a predictor of life history strategies because it is purportedly a feature exogenous to the organism. The organism's life history strategies purportedly evolve in response to this feature.

Yet organisms influence virtually all forms of mortality; to varying degrees, organisms reduce exposure to predators, avoid situations associated with morbidity risk, invest in immune systems, and repair somatic damage. Indeed, it is not clear that any form of mortality is fully extrinsic. Modeling shows that, when organisms' allocations to reduce mortality are effective, longer life spans may evolve (Chen & Maklakov, 2012). Hence, life history strategies are expected to be influenced by the rate of fitness returns per unit of mortality-reduction allocation (Del Giudice et al., 2015). All else equal, the greater the rate of returns (relative to the rate of returns from immediate reproduction), the more it pays to make mortality-reduction investments, resulting in longer lifespan, later reproductive onset, and greater benefits to investment in features that pay off once reproduction begins (see also André & Rousset, 2020; Kaplan & Gangestad, 2005). This alternative framing can yield distinct predictions. Harsh conditions may not select for fast life histories, so long as mortality reduction efforts are effective.

In addition, life history strategies are not simple outcomes of conditions completely independent of the organism; these conditions often follow from other strategic decisions of the organism. Sibly and Brown (2007) observed that mammalian species with fast strategies forage on abundant and reliable food sources, predisposing them to higher predation rates (e.g., ungulates, rabbits, voles, flesh-eating marine mammals). They rapidly convert caloric intake into offspring mass. Species with slow strategies have adaptations to reduce predation, via defense or changes in ecology (e.g., bats, burrowing rodents, primates, rhinos). These species allocate energy to mortality reduction in ways that limit immediate reproductive effort. Thus, species evolved to occupy different points on a mortality-risk continuum, which are associated with different life histories. Life history strategies co-evolve with other features, including how organisms harvest energy sources.

1.2.2. Conflicting predictions regarding resource scarcity/unpredictability

With regard to human life history variations, theorists have argued that extrinsic mortality risks increase with environmental harshness, which has often been discussed in terms of resource scarcity (Belsky, 2012). Under this framework, "the most direct prediction from life history theory is that cues of greater mortality (e.g., high levels of violent crime) *and resource scarcity (e.g., low relative income)* should lead to faster life history strategies" (Griskevicius et al., 2011, p. 243; emphasis added); and, children who perceive "resources as scarce and/or unpredictable will develop behavior patterns that function to reduce the age of biological maturation […], accelerate sexual activity, and orient them towards short-term, as opposed to long-term, pair bonds" (Belsky et al., 1991, p. 650).

But does resource scarcity or unpredictability necessarily diminish the returns to mortality reduction efforts, resulting in fast life histories? The answer appears to be no: Mammalian species with reliable and abundant food sources tend to have *faster* strategies (Sibly & Brown, 2007); resource unpredictability may select for *slower* life histories, as observed in primates (Jones, 2011). Energetic stress undoubtedly increases mortality risks. But it is not evident that low or variable energy availability diminishes investment returns to survival, as opposed to reproductive, efforts. Indeed, the opposite is true in humans. Severe energetic stress is a reliable predictor of *delayed* maturation and reproduction (Burks et al., 2000; Ellison, 2001, 2003; Evans & Anderson, 2012; Frisch, 1985; Hill, Elmquist, & Elias, 2008; see also Stearns & Rodrigues, 2020). Even in well-nourished populations, negative energy balance (energy expenditure that exceeds energy consumption) and high energy flux (variability in energy balance) are associated with reduced fecundity (Ellison, 2001, 2003). (See also Richardson et al., 2020.)

1.2.3. Infectious disease as a predictor of life history strategies

Ancestrally, infectious disease was a major cause of mortality. In foraging groups, about 40% of children die prior to age 15, mostly from infectious disease (Gurven & Kaplan, 2007). Viral, bacterial, and macroparasitic infection also accounts for a substantial proportion of adult mortality (Gurven & Kaplan, 2007). Humans invest tremendous energy in immune systems, adaptations that reduce mortality from infections. But the vastly shorter generation times, faster reproduction, and higher mutation rates of pathogens entail that immune systems cannot be perfectly designed to respond to all pathogens at all times. After a certain point, returns to pathogen resistance likely diminish.

An individual's frequent illness may be a cue that environmental pathogen load is high and/or the individual's ability to combat pathogens is relatively poor. As either condition could favor faster life history strategies via less controllable mortality risks, frequent childhood illness plausibly calibrates earlier pubertal timing. Furthermore, if childhood illness covaries with resource availability, it could generate a correlation between measures of resource availability (e.g., SES) and pubertal timing. (See Rickard et al., 2014, for a related perspective.)

1.2.4. Are unrestricted mating strategies also "fast" strategies?

Dominant psychological perspectives on human life history variations assume that offspring quantity (versus quality) is achieved via short-term mating (e.g., Belsky et al., 1991; Chisholm, 1993; Ellis et al., 2009). This assumption is questionable, in light of conditions under which human adaptations evolved. Fertility rates across foraging groups are a function of mean length of female interbirth intervals; shorter intervals between births allow for more offspring (Marlowe, 2001, 2003). A key determinant of shorter interbirth intervals in foraging societies is degree of paternal nutritional provisioning. Paternal provisioning permits earlier child age at weaning and higher maternal energy balance, leading to resumption of ovulatory cycles (Marlowe, 2001; Valeggia & Ellison, 2009). Arguably, females can more effectively increase offspring quantity by forming stable long-term partnerships (Sear, 2020), within which partner provisioning increases the rate at which offspring are produced across the reproductive lifespan. Regardless of whether male provisioning reflects mating or parenting effort, its contribution has been hypothesized to play key roles in the evolution of women's *faster* rate of reproduction (Hill & Hurtado, 2009; Kaplan, Hill, Lancaster, & Hurtado, 2000; Marlowe, 2001).

Furthermore, the assumption that female willingness to engage in uncommitted sex facilitates high offspring quantity or genetic diversity is biologically infeasible. Prior to a woman's first birth or within an interbirth interval, sex with multiple partners does not culminate in multiple offspring. Interbirth intervals among hunter-gathers average over three years (Kramer, 2010; Marlowe, 2003; Meehan, Quinlan, & Malcom, 2013), during which short-term mating strategies are ineffective—and likely counterproductive—towards offspring genetic diversification. Higher offspring number generates diversity, even when offspring share the same father (even if not to the same extent as when offspring have different fathers; Ellis et al., 2009). Female unrestricted sexuality or extra-pair sex undermines paternal certainty, leading to loss of paternal provisioning and hindering offspring number (Geary, 2000; Kramer, 2010; Marlowe, 1999, 2001; Meehan et al., 2013). Accordingly, willingness to engage in uncommitted or extra-pair sex may be distinct from a fast-slow dimension, should one exist (Dinh, Pinsof, Gangestad, & Haselton, 2017). Indeed, cross-species differences in mating strategies—e.g., social monogamy, polygynandry—appear to arise from features of a species' socioecology and phylogenetic history unrelated to extrinsic mortality risk (Díaz-Muñoz & Bales, 2016; Lukas & Clutton-Brock, 2013; Opie, Atkinson, Dunbar, & Shultz, 2013; Thompson, 2016).

1.3. Empirical links between early conditions, age at menarche, and mating orientations

Numerous studies have examined associations between childhood adversity and pubertal timing in girls. A meta-analysis of 45 studies detected no overall association with pubertal timing, though associations varied by childhood experience (Zhang, Zhang, & Sun, 2019). Low SES was associated with *later* puberty, though not significantly (see also, Richardson, La Guardia, & Klay, 2018; Sheppard & Van Winkle, 2020). No significant associations were observed with neglect or physical abuse. Small but significant associations with family dysfunction, sexual abuse, and father absence were observed (see also Black, 2016; for a meta-analysis on father absence, see Webster, Graber, Gesselman, Crosier, & Schember, 2014). Naturally, correlation does not imply causation; genetic confounds (e.g., Barbaro, Boutwell, Barnes, & Shackelford, 2017; Mendle et al., 2006; cf. Tither & Ellis, 2008) or unmeasured childhood experiences could drive associations. For instance, childhood health or exposure to violence—which may provide more plausible cues of extrinsic mortality—could drive associations of pubertal timing with other childhood experiences. Very few studies have simultaneously examined or controlled for multiple childhood predictors of pubertal timing. (A few studies have examined multiple childhood predictors, including health or exposure to violence, of purported psychosocial indicators of life history [Chua, Lukaszewski, Grant, & Sng, 2016] or of mating and reproductive behavior [Međedović, 2019, 2020].)

Links between pubertal timing and mating outcomes have received mixed support. Earlier menarche is robustly associated with age of sexual debut across Western and developing countries (e.g., Copeland et al., 2010; Helm & Lidegaard, 1989; Ibitoye, Choi, Tai, Lee, & Sommer, 2017). However, there is little support that earlier menarche leads to unrestricted mating orientations, more sexual partners, relationship instability, or low-quality parenting (reviewed in Ellis, 2004; see also Copeland et al., 2010; Ibitoye et al., 2017; Barbaro, Richardson, Nedelec, & Liu, 2020). Notably, a survey of over 10,000 women found a near-zero correlation ($r = 0.007$) between age of menarche and sociosexual attitudes (Batres & Perrett, 2016). These results suggest that earlier pubertal timing facilitates earlier onset of sexual behavior but does not necessarily precipitate a short-term (versus long-term) mating strategy.

1.4. The current study

The current study explored these issues empirically, using structural equation modeling on a sample of 1867 US-resident women. We examined effects of five retrospectively self-reported variables pertaining to childhood experiences of interest: SES, unpredictable family environments, exposure to violence, father absence/quality, and susceptibility to illness. Evidence indicates that retrospective reports of childhood adversity are moderately associated with prospective measures ($r \cong 0.5$; Reuben et al., 2016).

We tested associations of childhood experiences with the following traits: timing of puberty and sexual debut, short-term and long-term mating orientations, sociosexual behavior, age at first reproduction, and offspring number. We aimed to assess (1) which childhood experiences independently predict pubertal timing; (2) which life history outcomes are predicted by pubertal timing; (3) whether pubertal timing and long-term or short-term mating orientations mediate the impact of childhood experiences on sociosexual behavior and reproductive outcomes; and (4) which childhood experiences predict sociosexual and reproductive outcomes directly, independent of mediation.

2. Methods

2.1. Participants

The initial sample consisted of 2500 participants, recruited from Amazon Mechanical Turk as part of another study examining women's life history strategies and sexual motivation (Dinh et al., 2017). The study was approved by the University of California, Los Angeles Institutional Review Board. After excluding men, women who are not sexually attracted to men, repeat entries, and participants who failed the attention checks or admitted to not taking the survey at all seriously, the final sample for this study included 1867 women, of whom 1361 were exclusively pair-bonded. Participants were between 18 and 61 years of age (mean = 28.2, SD = 4.7) and currently lived in the United States.

2.2. Data availability

Data and Supplementary Online Materials (SOM), including analysis scripts and output, are posted on Open Science Forum (https://osf.io/mw4ny/).

2.3. Measures[1]

2.3.1. Childhood predictors

Socioeconomic status (SES). Five measures of childhood SES were obtained ($\alpha = 0.89$). Perceived resource availability was assessed with the following: "My family usually had enough money for things when I was growing up," "I felt relatively wealthy compared to the other kids in my school," and "I grew up in a relatively wealthy neighborhood" (from Griskevicius et al., 2011). Participants also indicated the socioeconomic class in which they grew up in (1 = lower, to 5 = upper) and reported their childhood family income on an 8-point scale. All measures are scored such that higher scores reflect greater SES.

Family unpredictability. Childhood environmental unpredictability was assessed using ten items measuring early-life family instability (e.g., "My family life was generally inconsistent and unpredictable from day-to-day"; $\alpha = 0.91$). This scale (or subsets of it) have been used in previous life history research as a measure of childhood unpredictability (Fennis, Gineikiene, Barauskaite, & van Koningsbruggen, 2020; Maner, Dittmann, Meltzer, & McNulty, 2017; Mittal & Sundie, 2017; Young, Griskevicius, Simpson, Waters, & Mittal, 2018).

Exposure to violence. Four items, adapted from screening questions from Crouch, Hanson, Saunders, Kilpatrick, and Resnick (2000), assessed childhood frequency of physical abuse by a caregiver, being physically hurt by a non-caregiver, witnessing someone hurt another person, and witnessing someone hurt or threatened with a weapon.

Paternal investment. Women rated their relationship quality with their father from before age sixteen, from worst (1) to best (7) possible relationship. Father absence during ages 0–5 following parental divorce was dummy-coded (father absence = 1) and had a negative factor loading.

Health. Women rated their childhood health, on a scale from poor (1) to excellent (5).

2.3.2. Life history traits

Age of pubertal onset. Women reported their age of first menses and whether their breasts developed earlier or later than female peers. Retrospective reports of relative timing have reasonable validity for large-scale studies (Mendle, Beltz, Carter, & Dorn, 2019).

Age of sexual debut. Four questions pertained to timing of sexual debut: age at initiation of sexual activities, age at first sexual intercourse, and onset of romantic dating and sexual activity relative to female peers.

Mating orientations. Women responded to a subset of the Sociosexual Orientation Inventory (Jackson & Kirkpatrick, 2007): 4 items concerned *Short-Term Mating Orientation* (*STMO*; e.g., "I could enjoy sex with someone I find highly desirable even if that person does not have long-term potential"; $\alpha = 0.91$) and 4 items concerned *Long-Term Mating*

[1] See SOM, Part 1, for complete description of measures used, frequency distributions, and data screening procedures.

Orientation (*LTMO*; e.g., "I am interested in maintaining a long-term romantic relationship with someone special"; α = 0.88).

Number of sexual partners. Women reported their lifetime number of male sexual partners, number of one-time male sexual partners, number of male sexual partners in the past year, and predicted number of male sexual partners in the next five years.

Age of first reproduction. Women reported the ages they had their first pregnancy and first birth. 866 women (46.6%) had been pregnant, and 712 women (38%) had given birth. Women who had not given birth reported the age they expected to have their first birth. Analyses reported in-text use responses for actual, but not expected, age of first birth. In SOM, we report analyses with responses from age at first birth and age at expected first birth combined as one variable (see Part 3 for results in table format; Part 7 for full output).

Offspring quantity. Women reported the number of times they had been pregnant, the number of times they had given birth, their current number of children, and their ideal number of children. Analyses reported in the main text excludes ideal number of children as an indicator. See SOM for analyses including this indicator (Part 3 for results in table format; Part 7 for full output).

2.4. Statistical analyses

Structural equation modeling (SEM) was conducted using lavaan 0.6–5 on R version 3.6.3. Observed variables described above were indicators for their respective latent factors. Diagonally weighted least squares robust estimation was used, as appropriate for ordinal data, and pairwise deletion was used for missing data.[2]

Prior to running path models, confirmatory factor analyses were conducted for individual factors to test for adequate fit (e.g., through examinations of factor loadings and correlated residuals; SOM Part 2), with the exception of paternal investment, childhood health, age at pubertal onset, and age at first reproduction, which were not identified on their own but were identified in the context of the larger mediation models. Correlated residuals were included between necessarily related indicators: witnessing someone hurt with a weapon and witnessing someone being hurt; age at first sexual activity and age at first sexual intercourse; number of one-time sexual partners and total number of sexual partners.

For primary mediation analyses, standard errors and confidence intervals were estimated using 1000 bootstrap draws. Standardized estimates are reported (see SOM Part 3 for results in table format). Full output of primary models, including factor loadings and unstandardized solutions, can be viewed in SOM Parts 4–5.

2.4.1. Control variables

Number of sex partners and one-night stands were positively related to age. Future number of sex partners was negatively related to being in an exclusive pair-bond. Thus, we included age and relationship status (effect coded, pair-bonded = 1) as covariates for the respective indicators. We also included age as a covariate for the offspring quantity factor, as all indicators were positively associated with age. SOM Part 2 includes results of invariance testing by relationship status and age group (split by tertiles) for measurement models of mating and reproductive outcomes. Metric invariance across relationship status was demonstrated for STMO, LTMO, and offspring quantity, but not for number of sexual partners. (Current relationship status may greatly affect expected future number of sexual partners, while having a weaker association with past number of sexual partners; this may thus differentially affect factor loadings across single and partnered women.) Number of sexual partners and offspring were not measurement invariant across age groups.

[2] For confirmatory factor analyses on individual factors with only continuous indicators, maximum likelihood estimation was used.

2.4.2. Model constraints

Because childhood health was a single-indicator factor, we set the factor loading to 1 and error variance to 0.36. Error variance was computed using the formula: (childhood health variance) × (1 − reliability). Observed variance in childhood health is 1.03. Reliability was specified as 0.65, based on average Goodman-Kruskal gamma estimates of women's retrospective reports of childhood health (Haas, 2007). Using inter-item reliability from a perceived vulnerability to disease scale from a different dataset (α = 0.80, error variance = 0.21; Dinh et al., 2021) produced similar results (SOM Part 6).

For Model 1, iterative procedures estimated a negative variance for the number of pregnancies indicator. Confidence intervals around the variance estimate included zero, suggesting that the negative variance is a result of sampling variability (Chen, Bollen, Paxton, Curran, & Kirby, 2001). To avoid bias in estimates that may result from a negative variance estimate, we constrained the variance to a small positive number (0.02, 1 standard error above the negative variance estimate). Models 2–4 did not estimate a negative variance. Thus, we did not constrain the parameter in those models.

3. Results

Table 1 provides a summary of models tested. In Model 1, we regressed each of the life history traits on the five childhood factors to examine associations independent of mediation by pubertal timing or mating orientations (Tables 2-4).[3] We then performed three structural equation mediation models (Models 2-4). For complete output of mediation results, with bootstrapped confidence intervals for direct and indirect effects, see SOM Part 5.

All models exhibited close approximate fit, with CFI > 0.95, and RMSEA and SRMR <0.05 (Browne & Cudeck, 1992; Hu & Bentler, 1999). Model chi-squares (exact fit tests) were significant; with very large sample sizes, even minor deviations from an idealized model often results in significant lack of fit (Kline, 2011). Notes for Table 2 and Figs. 1-3 report exact fit indices.

3.1. Which childhood experiences uniquely predict sexual and reproductive outcomes? Are these relationships mediated by pubertal timing?

3.1.1. Predictors of pubertal timing

First, we ask to what extent childhood experiences predicted pubertal timing. (We report estimates from Model 2 [Fig. 1]; results from Model 3 are similar [Fig. 2].) Of the five childhood factors, only exposure to violence and health significantly predicted pubertal timing. Girls exposed to more violence reached puberty earlier (B = -0.16, p = .017), as did girls with poorer health (B = 0.11, p = .013). We found no compelling evidence for the impact of SES (B = −0.004, p = .926) or paternal investment (B = 0.04, p = .256) on pubertal timing. Contrary to predictions, girls from unpredictable family environments tended to reach puberty later, though this effect fell short of significant (B = 0.10, p = .080).

3.1.2. Impacts of pubertal timing on outcomes

Next, we ask whether pubertal timing predicted mating and reproductive outcomes. In Model 2, earlier pubertal maturation predicted earlier sexual debut (B = 0.15, p < .001). Pubertal timing's effects on STMO and LTMO were negative and just short of significant: earlier maturation was associated with higher STMO (B = -0.06, p = .080) *and*

[3] Separate SEM regression analyses excluding women who had not had a male sexual partner (and thus could not have naturally had offspring) find that number of sex partners does not significantly predict age at first reproduction or offspring quantity. Analyses find significant effects of age at sexual debut; earlier sexual debut entails a younger age and more sexually active years during which sexual behavior and reproduction could occur (SOM, Part 15).

Table 1
Summary of structural equation models 1–4: Relationships between model factors.

Model	Childhood Predictors (IVs)	Mediator(s)	Life History Outcomes (DVs)
1	Socioeconomic Status Family Unpredictability Exposure to Violence Paternal Investment Health		All outcomes below
2		Age of Pubertal Onset	Age of Sexual Debut Short-Term Mating Orientation (STMO) Long-Term Mating Orientation (LTMO)
3		Age of Pubertal Onset	Number of Sex Partners Age of First Reproduction Offspring Quantity
4		STMO LTMO	Number of Sex Partners Age of First Reproduction Offspring Quantity

Table 2
Model 1 structural equation modeling (SEM) regression estimates, for childhood factors predicting life history outcomes.

	Life History Outcome						
Childhood Predictor	Age of Pubertal Onset	Age of Sexual Debut	Short-Term Mating Orientation	Long-Term Mating Orientation	Number of Sex Partners	Age of First Reproduction	Offspring Quantity
SES	−0.004	−0.035	**0.080***	**−0.111****	**0.085****	0.041	**−0.103****
Unpredictability	*0.100*	−0.003	**0.113***	**−0.181****	0.021	0.072	*−0.068*
Violence	**−0.165****	**−0.234*****	0.067	0.088	**0.139***	**−0.243*****	**0.166*****
Paternal Investment	0.046	**0.075***	−0.011	**0.084***	**−0.101****	0.031	−0.006
Health	**0.104****	0.003	−0.026	*0.069*	0.033	−0.042	**0.078***

$p < .10$, *$\mathbf{p<.05}$, **$\mathbf{p<.01}$, ***$\mathbf{p<.001}$.
Model fit statistics: CFI = 0.977; RMSEA = 0.042, 90% CI (0.041, 0.043); SRMR = 0.046; $\chi^2(964) = 4131.723$, $p < .001$.

Table 3
Correlations between childhood factors.

	SES	Unpredictability	Violence	Paternal Investment
Unpredictability	−0.460			
Violence	−0.423	0.710		
Paternal Investment	0.419	−0.457	−0.439	
Health	0.290	−0.429	−0.433	0.278

SEM standardized covariances from Model 1. All p's < 0.001.

LTMO (B = -0.07, p = .056). In Model 3, pubertal timing did not significantly predict number of sexual partners, age at first reproduction, or offspring quantity.

3.1.3. Mediation of childhood experiences by pubertal timing

Does pubertal timing mediate impacts of childhood experiences on outcomes? In our analyses, only with respect to age of sexual debut: Pubertal timing partially mediated the effect of exposure to violence (indirect effect = −0.02; 95% CI: −0.04, −0.003) and fully mediated the effect of childhood health (indirect effect = 0.02; 95% CI: 0.002, 0.03) (Model 2). Pubertal timing did not significantly mediate effects of childhood experiences on mating orientations (Model 2), number of sexual partners, age at first reproduction, or offspring number (Model 3).

3.1.4. Direct effects of childhood experiences on outcomes

We found evidence for several direct effects of childhood experiences on outcomes.

In Model 2, exposure to violence (B = -0.22, $p < .001$) uniquely predicted earlier sexual debut, independent of pubertal timing. Healthier girls tended to have higher adult LTMO, independent of

Table 4
Correlations between life history outcomes.

	Age of Pubertal Onset	Age of Sexual Debut	Short-Term Mating Orientation	Long-Term Mating Orientation	Number of Sex Partners	Age of First Reproduction
Age of Sexual Debut	**0.161*****					
Short-Term Mating Orientation	−0.041	**−0.236*****				
Long-Term Mating Orientation	**−0.114****	0.048	**−0.400*****			
Number of Sex Partners	−0.011	**−0.339*****	**0.533*****	**−0.200*****		
Age of First Reproduction	0.004	**0.266*****	0.015	−0.016	**−0.071****	
Offspring Quantity	0.024	**−0.241*****	**−0.139*****	0.036	**0.061***	**−0.463*****

SEM standardized covariances from Model 1. *$\mathbf{p<.05}$, **$\mathbf{p<.01}$, ***$\mathbf{p<.001}$.

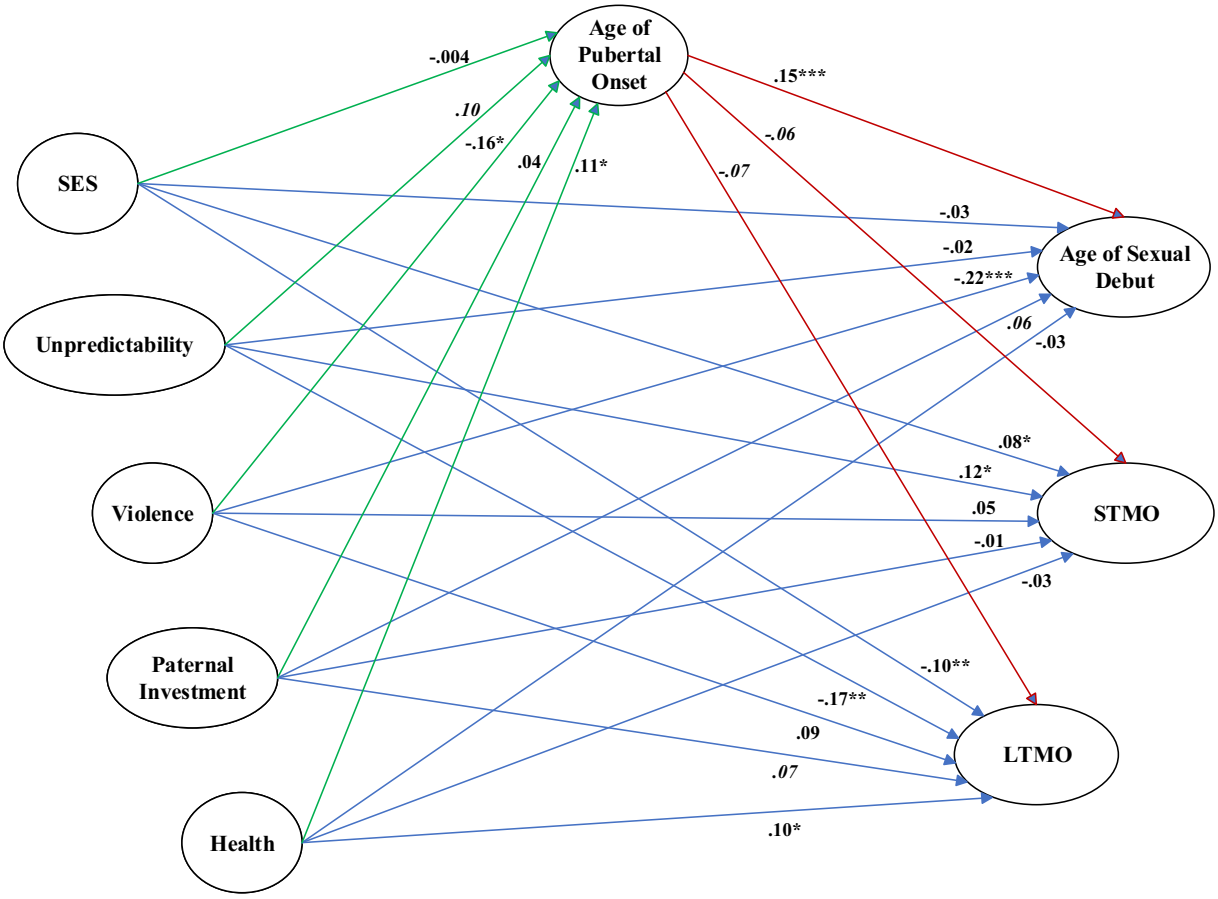


Fig. 1. Model 2 diagram.
Notes. **Childhood predictors** (ovals on the left): Socioeconomic status (SES), family unpredictability, exposure to violence, paternal investment, and health.
Mediator (oval at the top middle): Age of pubertal onset.
Life history outcomes (ovals on the right): Age of sexual debut, short-term mating orientation (STMO), and long-term mating orientation (LTMO).
For ease of viewing, only factors and a, b, and c' paths are shown. Covariances are modeled between childhood predictors and between life history outcomes but are not depicted in the diagram. Indicators and variances for each factor, as well as correlated residuals between necessarily related indicators, are not shown.
$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$. CFI = 0.985; RMSEA = 0.049, 90% CI(0.047, 0.051); SRMR = 0.043; $\chi^2(557) = 3041.979$, $p < .001$.

pubertal timing ($B = 0.10, p = .015$). Higher-quality paternal investment was associated with higher LTMO, but the direct effect fell short of significant ($B = 0.07$, $p = .068$). (Without controlling for pubertal timing, paternal investment's effects on LTMO were significant: B's = 0.08; $p = .031$, 0.042 [Model 1 and 4, respectively]). Paternal investment did not significantly predict STMO ($B = -0.01$, $p = .801$). Family unpredictability predicted STMO positively ($B = 0.12$, $p = .012$) and LTMO negatively ($B = -0.17$, $p = .002$). SES's effects on sociosexual orientations were opposite of predictions by the fast-slow paradigm: girls from higher SES households had higher STMO ($B = 0.08, p = .021$) and lower LTMO ($B = -0.10, p = .008$) as adults.

Model 3 found that women with higher childhood SES tended to have more sexual partners ($B = 0.09$, $p = .020$), as did women with lower-quality fathers ($B = -0.11, p = .013$) and women exposed to more violence ($B = 0.13$, $p = .044$). Only childhood exposure to violence predicted earlier onset of reproduction[4] ($B = -0.24$, $p < .001$). Lower childhood SES, higher exposure to violence, and better childhood health independently predicted women having more offspring (SES: $B = -0.10$, $p = .001$; violence: $B = 0.17$, $p = .001$; health: $B = 0.08$, $p = .015$).

3.1.5. Mediation by STMO and LTMO

Model 4 (Fig. 3) tests whether STMO and LTMO mediate the effects of childhood factors on number of sexual partners and reproductive outcomes. Unsurprisingly, STMO significantly predicted number of sexual partners ($B = 0.54$, $p < .001$). LTMO did not ($B = 0.01$, $p = .882$). Furthermore, STMO fully mediated the effects of SES and unpredictability on number of sexual partners (SES indirect effect = 0.04; 95% CI: 0.01, 0.08; unpredictability indirect effect = 0.06; 95% CI: 0.01, 0.11). Paternal investment remained a significant negative predictor of women's number of sexual partners, not mediated by sociosexual orientation.

STMO, but not LTMO, also significantly predicted offspring quantity, though not in the direction expected by the fast-slow paradigm: women endorsing more unrestricted mating orientations had fewer offspring ($B = -0.14, p < .001$). The negative effect of high SES on offspring number was partially mediated by STMO (indirect effect = −0.01; 95% CI: −0.02, −0.001), while family instability's negative effect was fully mediated by STMO (indirect effect = −0.02; 95% CI: −0.03, −0.001). There were no significant effects of STMO or LTMO on age at first reproduction.

3.2. Alternative model specifications and discussion of model comparisons

3.2.1. Alternative characterizations of childhood predictors

We performed several alternative model specifications with different

[4] In analyses that include expected age at first birth and ideal number of children as indicators (SOM, Part 7), higher SES emerged as a predictor of later onset of reproduction; family unpredictability's positive association with age at first reproduction and negative association with offspring number also became significant. STMO had a positive effect on age at first reproduction.

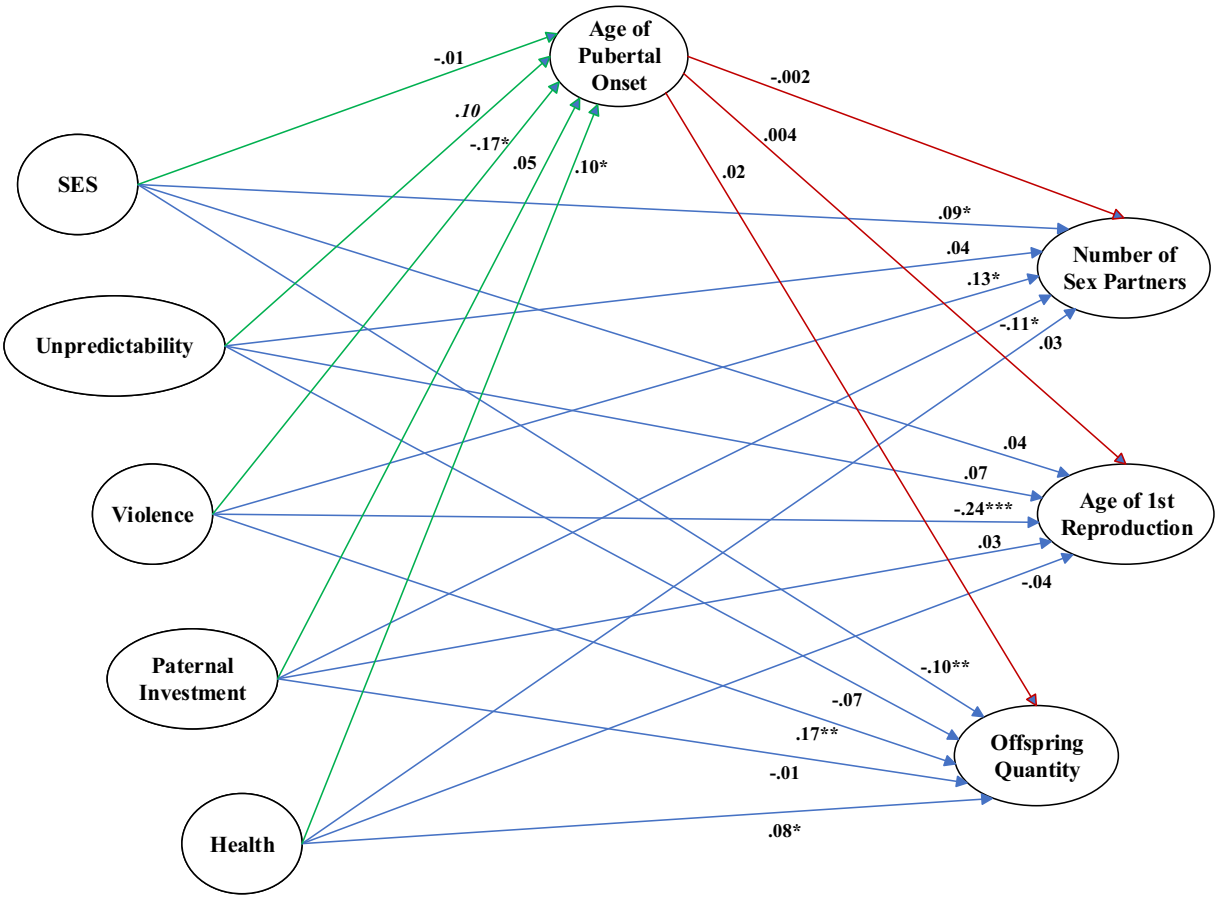


Fig. 2. Model 3 diagram.
*Notes.***Childhood predictors** (ovals on the left): Socioeconomic status (SES), family unpredictability, exposure to violence, paternal investment, and health.
Mediator (oval at the top middle): Age of pubertal onset.
Life history outcomes (ovals on the right): Number of sexual partners, age of first reproduction, and offspring quantity.
For ease of viewing, only factors and a, b, and c' paths are shown. Covariances are modeled between childhood predictors and between life history outcomes but are not depicted in the diagram. Indicators, variances, and correlated residuals are not shown.
$p < .10$, $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$. CFI = 0.981; RMSEA = 0.046, 90% CI(0.045, 0.048); SRMR = 0.049; $\chi^2(520) = 2597.083$, $p < .001$.

characterizations of childhood predictors (SOM Parts 8–11). As the childhood factors are correlated, shared effects may not be captured due to multicollinearity. To investigate this possibility, we created a second-order childhood "environmental risk" factor, encompassing the five childhood factors. We reran Models 1–4 with the childhood environmental risk factor as a predictor of life history outcomes. Models 1, 2, and 4 failed to converge, indicating that one overall risk factor is not the optimal characterization of childhood predictors of life history outcomes. One reason may be that low childhood SES has effects on mating orientations that run opposite in direction as effects of the other childhood adversity variables. Another reason may be that childhood health covaries only modestly with the other childhood factors, with SEM standardized covariances ranging from 0.27–0.43. Childhood health and SES may not belong as part of the same construct as the other childhood variables.

In contrast, because childhood exposure to violence and family unpredictability are strongly correlated (0.71 in SEM analyses), they possibly represent a similar factor whose effects are not fully represented in regression estimates. To address this possibility, we created a second-order factor with exposure to violence and family instability factors as indicators. Childhood SES, health, paternal investment, and violence-unpredictability factors were entered as predictors into Models 1–4. Violence-unpredictability did not significantly predict pubertal timing, nor did SES or paternal investment; health remained the only significant predictor of pubertal timing. The effect of a second-order factor including paternal investment, violence, and unpredictability as indicators was also not robust.

These results suggest that exposure to violence may uniquely predict pubertal timing, and that conflating its effects within a larger construct of violence/family-experiences weakens the relationship. Notably, changes to specifications of this larger construct did not lessen the impact of childhood health. Removing health as a predictor of pubertal timing led to significant effects of the second-order factor, which suggests that childhood health may partly drive relationships between pubertal timing and family instability or father absence/quality. In tests of sexual debut, mating strategies, and age at first birth, both specifications of the second-order factor had significant effects. Consistent with primary models, STMO negatively predicted offspring number and was a significant mediator of second-order childhood effects.

We next performed χ^2 difference tests, comparing fit between primary (less restricted) models and alternative (more restricted) models. All tests produced significant χ^2 differences, indicating that alternative models fit the data more poorly than primary models. (We also report modification indices for alternative models in SOM.)

Additionally, we ran Models 1–4 with each childhood factor separately. With effects of collinearity between childhood factors removed, all childhood factors significantly predicted pubertal timing, albeit with small effects (B's $< |0.16|$). Comparing these results with those of primary models (which controlled for other childhood experiences), the significant uncontrolled associations appear to be driven primarily by confounds with childhood violence and health. In models predicting mating orientations and number of sexual partners, childhood SES by

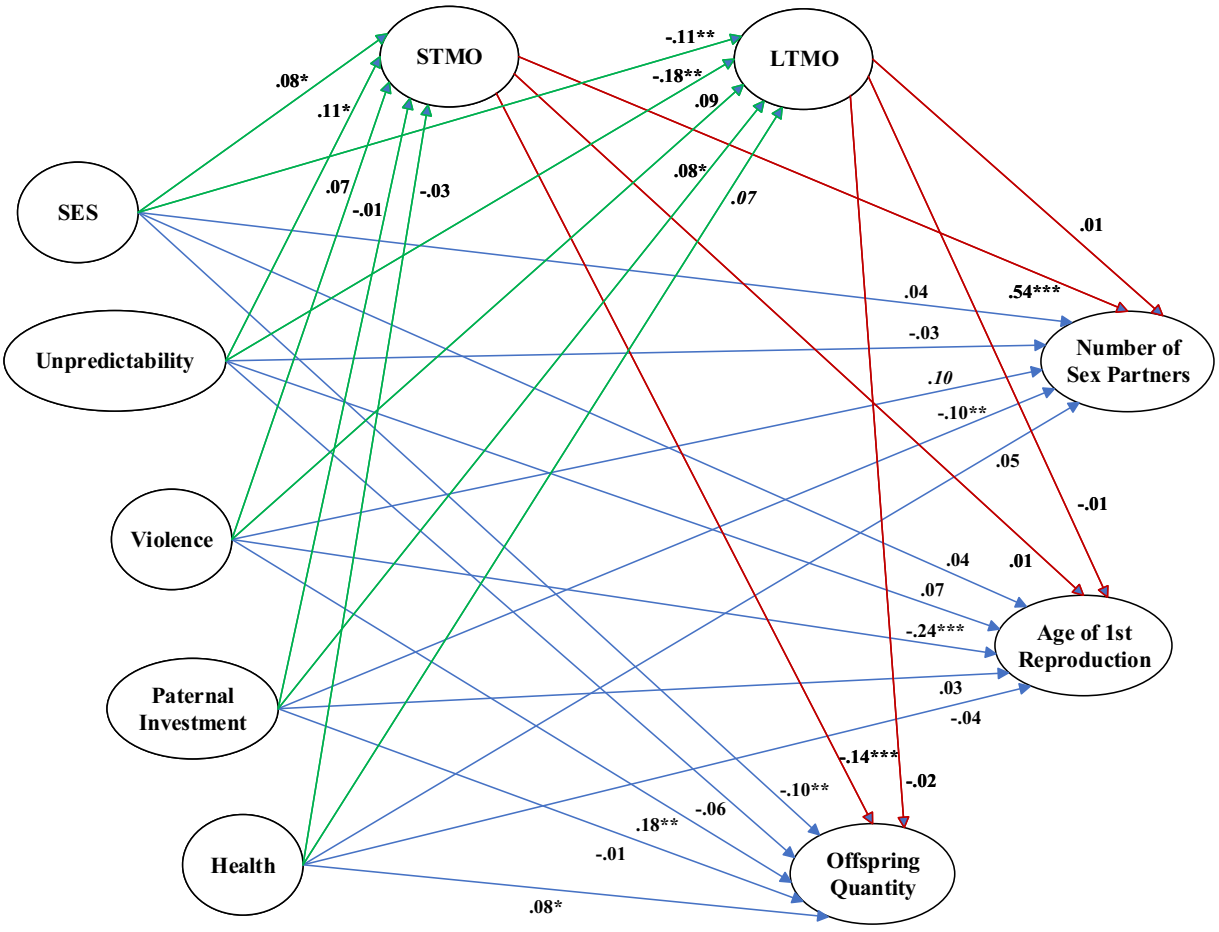


Fig. 3. Model 4 diagram.
*Notes.***Childhood predictors** (ovals on the left): Socioeconomic status (SES), family unpredictability, exposure to violence, paternal investment, and health.
Mediators (ovals at the top middle): Short-term mating orientation (STMO) and long-term mating orientation (LTMO).
Life history outcomes (ovals on the right): Number of sexual partners, age of first reproduction, and offspring quantity.
For ease of viewing, only factors and a, b, and c' paths are shown. Covariances are modeled between childhood predictors, between mediators, and between life history outcomes but are not depicted in the diagram. Indicators, variances, and correlated residuals are not shown.
$p < .10$, $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$. CFI = 0.979; RMSEA = 0.045, 90% CI(0.044, 0.047); SRMR = 0.046; $\chi^2(730) = 3534.543$, $p < .001$.

itself had nonsignificant effects. Controlling for the other childhood predictors allows the unique effects of SES to emerge.

3.2.2. *Multigroup models*

To test whether effects on mating and reproductive outcomes differ as a function of women's relationship status, we performed multigroup SEM (SOM Parts 12–13). In general, relationship status did not significantly moderate effects of childhood or mediator variables on women's number of sexual partners or age at first reproduction. The only significant interaction was that quality of paternal investment negatively predicted age at first birth among single women but not partnered women. Relationship status did, however, significantly moderate effects of several childhood factors on offspring quantity. This is perhaps unsurprising, as single women tend to have reproduced fewer (or no) times; as a result, effects tend to be stronger for partnered women.

We also conducted multigroup SEM to test whether childhood and mediator variables differentially predict women's expected age at first birth (for nulliparous women) and actual age at first birth (for parous women).[5] In general, childhood effects on age at first reproduction were stronger for parous women. Among nulliparous women, STMO positively predicted and LTMO negatively predicted expected age at first reproduction. There were no significant effects of SOI on age at first reproduction for parous women. See SOM, Part 14.

4. Discussion

4.1. *The onset of reproductive maturity*

Reproductive maturation marks an important transition from investment in juvenile growth to competing investments in reproduction. The timing of this transition is hypothesized to mediate coordinated strategies of investments in current versus future reproduction and

[5] Age of first pregnancy and expected/actual age of first birth are indicators for the age at first reproduction factor. Some nulliparous women provided responses for age of first pregnancy but, by definition, none had responses for actual age of first birth. Therefore, the main difference from primary models is that is that results for age at first reproduction in multigroup models primarily represent differences in effects for expected versus actual age of first birth.

offspring quantity versus quality (Belsky, 2012; Belsky et al., 1991; Ellis, 2004; Ellis & Essex, 2007; James & Ellis, 2013). According to leading perspectives, earlier maturity and development of faster strategies are contingent on childhood environments, which shift optimal allocation decisions. Yet there remains the question of which childhood conditions predispose early puberty. Cues emphasized by current literature covary in Western societies. Large-scale investigations simultaneously testing the effects of key childhood predictors on hypothesized life history traits, such as early puberty, are surprisingly lacking (for one exception separately examining father absence and childhood stress, see Guo, Lu, Zhu, & Chang, 2020). The current study aims to address these issues.

4.1.1. Predictors of pubertal timing

Ellis et al. (2009) proposed that less controllable mortality risk selects for earlier reproductive maturity; childhood cues of increased mortality risk therefore adaptively calibrate the timing of maturation. Using structural equation modeling, the current study examined which childhood experiences predict pubertal timing. Contrary to leading proposals, analyses failed to detect unique effects of childhood SES, family unpredictability, and father absence/quality. Instead, childhood health and exposure to violence independently predicted pubertal timing. Results may offer insight into the conditions in ancestral environments that most reliably affected uncontrolled mortality risk.

Exposure to violence may provide more reliable cues of environmental morbidity-mortality risk than do indirect cues such as SES, family instability, or paternal investment. Possibly, sampling variability in girls' experiences with violence partially accounts for the mixed results observed in the pubertal timing literature. Exposure to violence, usually not directly measured in life history research, potentially drives previously documented associations between early menarche and other childhood variables. Each childhood factor alone was significantly associated with pubertal timing, yet controlling for exposure to violence rendered the effects of all but health nonsignificant. As these results could be sample-specific, future studies should continue to examine the joint or independent effects of these variables on pubertal timing.

In contrast to other harsh experiences, childhood susceptibility to illness is likely distinct from exposure to violence or family dynamics. Though rarely considered in conceptualizations and empirical examinations of the fast-slow paradigm, pathogenic threat was likely a relatively expensive-to-control mortality risk in ancestral environments. Multiple and/or persistent bouts of infectious illness may have forecasted risk of early mortality or morbidity ancestrally, indicating earlier optimal pubertal maturation. Alternatively, poor childhood health for some may reflect underlying conditions not precipitated by exposure to pathogens. Nettle, Frankenhuis, and Rickard (2013) proposed that internal conditions may forecast mortality risks, leading to calibration of life history trajectories. For example, low birth weight is associated with congenital abnormalities and higher mortality risk extending beyond childhood and at least until early adulthood (Watkins, Kotecha, & Kotecha, 2016). Across developing and high-income countries, girls born of lower weight and after shorter gestations tend to experience earlier menarche (Aurino, Schott, Penny, & Behrman, 2018; Blell, Pollard, & Pearce, 2008; Sloboda, Hart, Doherty, Pennell, & Hickey, 2007). Individuals born of lower weight may have been predisposed to poor health and relatively high extrinsic mortality risk in ancestral environments, perhaps benefitting from accelerated reproductive maturity.

Other childhood health conditions predisposing substantial mortality threat may similarly shift adaptive strategies. Our measure of childhood health cannot distinguish between infectious illness and noncommunicable conditions (e.g., congenital, metabolic, or genetic disorders), both of which plausibly posed relatively uncontrollable mortality threats. Future research may separately examine the implications of these mortality threats.

4.1.2. Pubertal timing as a mediator of sexual outcomes

Our study replicates the unsurprising finding that early-maturing girls tend to have earlier sexual debut. Pubertal timing mediated the effects of exposure to violence and childhood health on timing of sexual debut. Yet, in our models, pubertal timing neither significantly covaried with nor predicted number of sexual partners, age at reproductive onset, or offspring number. Nominally, the effects of pubertal timing on short-term *and* long-term mating orientations were negative (such that earlier pubertal timing was associated with both), though both effects fell short of being significant at $p < .05$ (see also Batres & Perrett, 2016). While additional evidence may be needed to more fully evaluate these associations, these findings are consistent with a pattern in the literature that pubertal timing is not robustly associated with adult mating orientation, number of sexual partners, or offspring number (e.g., Ellis, 2004).

4.1.3. Is pubertal timing part of fast-versus-slow trajectories?

Given the lack of association between pubertal timing and many outcomes of interest, in both our data and the extant literature, a critical question is whether timing of reproductive maturation should be considered a component of fast-versus-slow human life history trajectories. We argue yes: Pubertal timing is likely fundamental to understanding life history variations of human females.

From a theoretical perspective, timing of reproductive maturity should be a central life history feature. Indeed, it is a central marker of the fast-slow dimension that differentiates species. The early work by Belsky et al. (1991) on psychosexual acceleration was conceptually compelling because of the central role it gave to pubertal timing—an event reflecting physiological processes. The lack of association between pubertal timing and unrestricted sociosexual orientation and behavior may reflect the possibility that these features are not appropriately construed as life history strategies, as discussed earlier (see section 1.2.4). Consideration of unique features of human evolution is important for deriving tenable hypotheses regarding human life history strategies. It is likely that mating strategies and life history strategies are developmentally sensitive to different, and potentially orthogonal, socioecological variables. The lack of robust association between pubertal timing and age at first reproduction or offspring quantity, by contrast, could possibly reflect impacts of environmental novelties and mismatch (e.g., due to the availability of effective contraception).

One threat to the idea that pubertal timing is part of fast-slow reproductive strategies is the repeated finding from pedigree studies implementing multiple family relations (not just twins) that age of menarche is roughly 50% heritable, with 50% of its variance due to environmental variance, but little to no variance due to environmental factors shared by siblings raised in the same household (e.g., Morris, Jones, Schoemaker, Ashworth, & Swerdlow, 2011; Towne et al., 2005). These findings pose challenges to claims that familial factors such as household instability or SES substantially influence pubertal timing (e.g., Barbaro et al., 2020)—and, indeed, we find little evidence that these factors (aside from exposure to violence) have much influence. However, the findings do not rule out the possibility that age of menarche is subject to adaptive calibration more generally. Childhood health status, for instance, may be partly heritable and partly due to unshared environmental variance. It may thereby partly mediate heritable and unshared environmental effects via adaptive calibration. The same may be true of a potentially related factor, low birth weight (see Sørensen et al., 2013).[6]

In sum, timing of pubertal maturation may indeed be a theoretically important life history strategy and mediator; lack of empirical coherence

[6] A GWAS study found a genome-wide SNP heritability of age of puberty of 0.25, accounting for only half of the heritability estimated from pedigree studies (Day et al., 2017). The discrepancy may be due to a variety of factors, including the impact of rare alleles not captured by SNPs, the presence of non-additive genetic effects, or overestimation of heritability in pedigree studies due to gene-environment interactions (e.g., Young, 2019; Zhu & Zhao, 2020).

between proposed life history features may stem from a focus on traits unrelated to life history trajectories. Additional theoretical work is needed.

4.2. Childhood environmental effects on sexual and reproductive outcomes

Data from the current study revealed a number of unique childhood effects on mating orientations, sexual behavior, and reproductive outcomes, independent of mediation by pubertal timing. Some effects are consistent with predictions from leading perspectives of the fast-slow paradigm. Others go in directions reverse those expected. Overall, results suggest that specific childhood conditions—rather than general harshness or unpredictability—may separately predict mating and reproductive strategies.

4.2.1. Harshness and unpredictability

Researchers have emphasized separate dimensions of environmental harshness and unpredictability, with independent but related effects (though research has at times found associations of one but not the other; e.g., Brumbach, Figueredo, & Ellis, 2009; Simpson, Griskevicius, Kuo, Sung, & Collins, 2012). The current study extends previous research by simultaneously testing childhood effects of family unpredictability, SES, and exposure to violence (as a separate harshness measure). Data revealed that SES/resource availability has associations distinct from those of violence and family instability.

Because low SES is associated with family instability and unsafe/risky local environments, controlling for the latter two isolates other effects of SES, such as resource scarcity. Doing so revealed that SES uniquely predicted certain traits in directions that are contrary to predictions from the fast-slow paradigm. Higher childhood SES (and higher female economic independence; see SOM Part 16) was a significant predictor of high short-term mating orientation, low long-term mating orientation, and more sexual partners. These results are similar to those of other studies, which find that SES associates with mating-related variables in directions opposite those expected by life history researchers in evolutionary psychology (Richardson et al., 2017, 2018; Richardson, Dariotis, & Lai, 2017). SES likely encapsulates multiple constructs, some of which may predict results in opposing directions. For example, some researchers have predicted that the "harshness" of low SES environments leads to the development of unrestricted mating. In contrast, high familial resource availability may allow women the economic freedom to pursue a more unrestricted strategy, as they may be less reliant on a partner for investment.

Independent of resource scarcity, childhood experiences with violence and family unpredictability did not uniquely predict the same outcomes. Exposure to violence may be a more reliable cue of relatively extrinsic mortality risk in the local environment, leading to earlier puberty, sexual debut, and first birth, and to more offspring. Also supporting this possibility, a study of participants from Kosovo and Serbia found that childhood exposure to violent intergroup conflict was independently associated with earlier marriage and first birth, while childhood poverty and family dysfunction were not (though childhood experiences did not predict age at sexual debut; Međedović, 2019). In our study, family instability appeared to have unique effects on women's mating orientations, which we have argued do not directly map onto a fast-slow dimension. In contrast to predictions made by the paradigm, family instability negatively predicted offspring number—an effect mediated by high short-term mating orientation.

Alternatively, exposure to violence and family instability may represent a common underlying factor. A higher-order violence-unpredictability factor predicted timing of sexual debut, mating orientations, and age at first reproduction. At the same time, the factor did not significantly predict pubertal timing or offspring number (unless controlling for mating orientations). Our data are consistent with these conditions tapping different causal factors, though additional research may further investigate their shared and distinct effects.

4.2.2. Paternal investment and family dynamics

Researchers have proposed that paternal investment is an important predictor of female mating and reproductive strategies, as the level of paternal investment a child receives may be reflective of optimal male strategies in the local ecology (Ellis, 2004). Consistent with this idea, our data suggest that father presence/quality may be specifically related to mating outcomes, including greater focus on long-term mating and having fewer sexual partners. Yet inconsistent with the theory, paternal investment was not robustly associated with the core life history trait of pubertal timing (or age at first reproduction and offspring number). Paternal investment may be less predictive of accelerated reproductive strategies and more predictive of women's mating outcomes.

As others have discussed, genetic confounds may drive associations of father absence/quality and family unpredictability with mating-related outcomes (e.g., Mendle et al., 2006). Existing studies find heritable effects on family stability and likelihood of parental divorce, yet very little evidence for shared environmental effects (e.g., effects of the rearing environment; Salvatore, Larsson Lönn, Sundquist, Sundquist, & Kendler, 2018). Some heritable effects are mediated through personality (e.g., lack of restraint [Jockin, McGue, & Lykken, 1996], which relates to unrestricted sociosexual orientation [Gangestad & Simpson, 1990; Snyder, Simpson, & Gangestad, 1986]). These heritable effects may partly explain associations between parental relationship stability and children's sociosexual attitudes. Additional genetically-informed studies are needed to assess the proportion of variance accounted for by heritable factors, compared to environmental influences.

4.3. Limitations and future directions

Many open questions in the study of human life histories remain. For instance, what are the precise circumstances in which mortality risk and other outcomes are relatively insensitive to individual and parental investment decisions? In such instances, what are the resulting strategies? Which strategies cohere, and for what specific functions? If unrestricted mating is unlikely to result in increased reproductive rate and quantity, other proximate mechanisms are needed to explain how women adaptively regulate their fertility. Indeed, a relatively unexplored area of inquiry involves examining physiological and cognitive adaptations regulating fertility outcomes and other life history strategies.

There has been some interest in the physiology facilitating fast life history strategies. Some studies investigating links between childhood adversity and accelerated pubertal timing have sought to elucidate the physiological mechanisms involved (Del Giudice et al., 2011; Ellis, 2004). Yet limited research has examined underlying adaptations regulating life history outcomes post-menarche. Some studies find that girls who experienced violence and abuse tend to have higher adult levels of follicle-stimulating hormone, a gonadotropin involved in follicular growth and maturation (Allsworth, Zierler, Krieger, & Harlow, 2001). Across and within populations, earlier menarche is associated with adult patterns of ovarian hormonal production implying heightened female fecundity (Ellison, 1996). Furthermore, endocrine mechanisms may potentiate or interact with psychological and behavioral mechanisms to regulate life history strategies (Dinh et al., 2017). More research is needed to replicate previous findings and further explore adaptations underlying life history outcomes.

As reproductive timing and output are core features that differentiate life history trajectories from an evolutionary biological perspective, the current study focused on pubertal timing and sexual and reproductive outcomes purported to reflect life history strategies in humans. Another approach has argued that more general psychosocial predilections (including personality traits) are key life history outcomes—e.g., "slow" life histories are reflected in cautiousness, valuation of familial relationships, a tendency to pair-bond, and agreeableness (e.g., Figueredo et al., 2005, 2014). These variations importantly affect social behavior

and demand evolutionary functional analysis. However, they do not appear to reflect life history "speed" (Richardson, McGee, & Copping, 2021). For instance, Black (2006) found little association between these outcomes and pubertal timing.

In addition to having a relatively targeted focus, on mating and reproductive outcomes, our study can only speak to potential strategies among human females. Future research can address these, and related, questions among males. Reproductive strategies naturally differ between the sexes. As such, developmental calibration of strategies may be expected to also differ. For instance, even when relatively high extrinsic mortality risk favors high offspring quantity, early reproductive maturity may benefit males less than females. By comparison, male reproduction is less constrained by time than it is by access to fecund sexual partners. And as the less reproductively limiting sex, males' mating and parenting strategies are predicted to be conditional on the male's characteristics (Gangestad & Simpson, 2000)—perhaps more so than on childhood conditions. For instance, males lacking certain features may be undesirable as short-term sexual partners and may benefit from a high paternal investment strategy. Attractive male mate qualities, such as certain physical features and the ability to provision, may take considerable resources to develop and may thus reduce the benefits of truncating childhood. For example, a male child with poor health—facing competing trade-offs between expensive investments in immunity and growth—who reaches puberty earlier at a cost to final body size may poorly compete for a reproductive partner in adulthood. Future research can further examine the relative effects of childhood environments on different dimensions of male and female life history strategies (see, e.g., Copping, Campbell, & Muncer, 2014).

Additionally, research on childhood environmental effects within small-scale, natural fertility populations is needed for deeper insight into ancestrally-recurrent factors affecting reproductive strategies. Given the diversity of inputs affecting life history allocations, many of which differ vastly from those in Western societies, integration of findings will potentially require modification of the existing framework. Predominant versions of the fast-slow paradigm in humans were primarily built upon evidence—and to try to explain patterns—in evolutionarily-novel, Western environments. With modern contraception, it is unclear to what extent offspring number and desire for offspring represent outcomes of selection. Integrating anthropological and psychological literatures is important for understanding the evolution of adaptive variation in human life history strategies.

Finally, a limitation of the current research is that reports of childhood experiences and pubertal timing were retrospective. Although retrospective reports may have moderate validity (Mendle et al., 2019), reports of childhood adversity may be influenced by adult personality factors (Reuben et al., 2016). Large-scale studies examining associations with prospective measures are needed.

4.4. Conclusions

Current models of the fast-slow paradigm in evolutionary psychology generally assume that childhood conditions affect pubertal timing, which precipitate mating orientations. Our data provide evidence that certain childhood conditions predict pubertal timing, but not ones highlighted by current theory. Additionally, links between pubertal timing and mating orientations were generally weak or absent. While childhood conditions may relate to mating orientations and reproductive outcomes independent of pubertal timing, associations (e.g., involving SES) did not consistently run in expected directions. Moreover, because pubertal timing did not mediate associations, it is unclear that these associations truly reflect variations in fast-slow life histories. In sum, childhood conditions, rates of maturation, sociosexual strategies, and reproductive outcomes do not covary in a way consistent with a single slow-to-fast continuum. New or revised theory is likely needed. Results offer directions in which future investigations may lead to improved understanding of the factors involved in human sexual and reproductive strategies.

Declarations of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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