Critical Examination of Life History Theory in Psychology

米谷，充史

(Degree)
博士（学術）

(Date of Degree)
2023-03-25

(Date of Publication)
2025-03-25

(Resource Type)
doctoral thesis

(Report Number)
甲第8522号

(URL)
https://hdl.handle.net/20.500.14094/0100482270

博　士　論　文

令和　４　年１２月　９　日

Critical Examination of Life History Theory in Psychology

（心理学における生活史理論の批判的検討）

神戸大学大学院人文学研究科博士課程
後期課程社会動態専攻

米　谷　充　史

Doctoral Dissertation

Critical Examination of Life History Theory in Psychology

（心理学における生活史理論の批判的検討）

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Contents

Abstract

Life history theory originated in evolutionary biology. However, it has recently become prevalent in psychology as a heuristic framework for explaining individual differences in psychological and behavioral tendencies as adaptation to certain environments. According to life history theory in psychology, childhood adversity provides cues for high morbidity/mortality during reproductive age, and individuals who experienced childhood adversity accelerate their reproductive schedule, accompanied by behavioral traits achieving some reproduction within their shortened (healthy) lifespans. Based on this framework, previous studies have reported that childhood adversity is associated with earlier reproductive timing, specifically, among women, and higher impulsivity, which are considered instances of adaptive phenotypic plasticity. Although these associations may be byproducts of environmental influence, the fundamental assumption of this explanation, that alternative phenotypes are associated with higher fitness only in matched environments, has not been subject to strict empirical examination. Therefore, this research critically tested this assumption for impulsivity and accelerated reproductive schedule in Studies 1 and 2, respectively. By targeting participants beyond reproductive age and measuring their reproductive success, we tested whether impulsivity or early reproductive initiation is associated with higher fitness among individuals who experienced childhood adversity and lower fitness among individuals who did not experience it. Both studies did not reveal this interaction, and thus, there is little evidence for the adaptive value of such phenotypic switching in response to childhood environments. Therefore, these results cast serious doubt on the fundamental idea of life history theory in psychology.

Keywords: phenotypic plasticity, childhood adversity, impulsivity, reproductive timing, life history theory

1. Introduction

1.1. Critical Issues with Life History Theory in Psychology

A growing body of research in psychology and other social science fields has been conducted under the banner of life history theory (Del Giudice et al., 2015; Nettle & Frankenhuis, 2020). Life history theory has its origin in evolutionary biology; however, in the last few decades, it has increasingly been applied to psychology (Belsky et al., 1991; Del Giudice et al., 2015) and other social sciences, such as anthropology (Sear, 2020), public health (Wells et al., 2017), and psychopathology (Del Giudice, 2014). It is expected to provide a heuristic framework for explaining individual differences in various psychological and behavioral traits (Ellis & Del Giudice, 2019). However, according to Nettle and Frankenhuis (2019, 2020), in its application to psychological research, the original core of life history theory in evolutionary biology has not been retained, and novel propositions have been added without critical arguments. Hence, it is necessary to critically examine life history theory in psychology.

Life history refers to age-specific patterns of survival and reproduction (i.e., how long an organism survives and when it reproduces during its lifetime). The principal tenet of life history theory is that there are trade-offs between different components of fitness, especially survival and reproduction: The more energy is allocated to reproduction, the less energy should be allocated to survival, and vice versa. In evolutionary biology, it is posited that selection acts on life history traits in a manner to maximize fitness (i.e., to increase gene frequency in the population). Based on this axiom, researchers have examined what kind of

life history traits evolve under the specific ecological conditions surrounding the focal species or population both theoretically and empirically (Godgil & Bossert, 1970; Stearns, 1976). Therefore, life history theory in evolutionary biology has mainly focused on species/population typical life history pattern or cross-species/population differences in life history traits.

In contrast, in psychological research, life history theory has often been used to explain individual differences. According to Nettle and Frankenhuis (2020), although life history theory in psychology has retained the most fundamental tenet of the original life history theory (i.e., the trade-off between survival and reproduction), it has added some propositions that are not inherent to the original life history theory. Specifically, it assumes that people developmentally employ adaptive reproductive strategies (life history strategies) in response to their local environments, especially childhood environments (Ellis et al., 2009). Moreover, it has extended the scope of explanation to psychological and behavioral tendencies purportedly associated with reproductive schedule (Figueredo et al., 2005; Griskevicius et al., 2013; Kruger et al., 2008). According to this theory, in adverse environments that provide cues regarding higher future morbidity/mortality, psychological and behavioral mechanisms that accelerate reproductive schedule are adaptive because the realization of reproductive potential is likely limited.

Life history strategies are considered to vary along the so-called fast–slow continuum (Ellis et al., 2009; Figueredo et al., 2005; Griskevicius et al., 2013; Nettle, 2010). Based on this framework, following predictions are typically posited based on this framework: Individuals who experienced harsh and/or unpredictable environments during childhood develop faster strategies consisting of earlier maturation, earlier reproductive timing, shorter inter-birth interval, and less parenting effort. Furthermore, faster strategies

include impulsivity and risk-taking tendency. Conversely, individuals who experienced benign environments during childhood develop slower strategies, which exhibit the opposite tendencies. Psychologists who were inspired by this framework assumed that such traits are adaptive reactions to local environments and attempted to empirically validate the associations between childhood environmental conditions and various later behavioral tendencies (see Del Giudice et al., 2015; Ellis & Del Giudice, 2019; Sear, 2020, for reviews). However, their predictions concerning individual differences were based primarily on observed variations in life history traits across species (Bielby et al., 2007; Pianka, 1970).

In evolutionary biology, the expression of different phenotypes in response to environmental conditions has been examined as phenotypic plasticity (Sommer, 2020). The expression of different morphologies or behavioral traits in response to environmental conditions (e.g., chemical cues of predators, nutrition, temperature) have been reported across various species as adaptive phenotypic plasticity (F. R. Adler & Harvell, 1990; Nijhout, 2003). However, phenotypic changes corresponding to environmental inputs are not always adaptations and can be byproducts or maladaptive consequences of the influences of environmental inputs (Gluckman et al., 2005b; Lea et al., 2017; McLaughlin et al., 2014). For example, extreme undernutrition may disrupt formal developmental processes and lead to behavioral tendencies that are unrelated to, or even detrimental to, adaptation. Therefore, if one argues that a phenotypic change is an adaptive reaction to a certain local environment, one must establish the presence of environment-specific fitness benefits of the target phenotype: In the presence of the environmental input, acquiring the specific phenotype must be associated with higher fitness (i.e., higher lifetime reproductive success) than not acquiring it, and in the absence of the environmental input, not acquiring it must be associated with higher fitness than acquiring it (Nettle & Bateson, 2015). These adaptative values of

phenotypic adjustment must be empirically demonstrated.

According to Nettle and Frankenhuis (2020) and Galipaud and Kokko (2020), in life history theory in evolutionary biology, reaction norms of life history traits have been described in formal models (e.g., Stearns & Koella, 1986; Houston & McNamara, 1992). However, in psychology, life history theory has been treated as a heuristic framework to conceive of the associations between environmental variables and behavioral tendencies as adaptive response. In other words, the conditions necessary for the evolution of phenotypic plasticity have been overlooked. In the end, the empirical associations found between environmental variables and behavioral traits have been regarded as solid evidence of adaptive plasticity without careful examination of their evolvability.

1.2. Specific Trends in Life History Theory in Psychology

Although there are criticisms regarding life history theory in psychology, as noted above, it forms a major research theme in evolutionary psychology (Del Giudice et al., 2015) and is expected to provide a useful framework for explaining individual differences in various behavioral traits and reproductive trajectory (Ellis & Del Giudice, 2019). In psychology as a whole, life history theory is becoming increasingly influential, with a growing number of articles containing the term “life history theory” (Nettle & Frankenhuis, 2019, 2020). Against this background, there is a need to critically examine the validity of the approach of life history theory in psychological research.

1.2.1. Reproductive Schedule

One prominent application of life history theory to psychological research is the individual difference in reproductive timing (Del Giudice et al., 2015; Sear, 2020). According to life history theory in psychology, in an adverse environment characterized by high morbidity/mortality, initiating reproduction earlier is advantageous because reproductive

potential is likely limited (Belsky et al., 1991; Ellis et al., 2009). Empirical studies have reported that adverse environmental conditions, especially during childhood, are associated with accelerated reproductive schedule, such as earlier first birth, earlier sexual debut, and earlier menarche, especially among females in developed countries (e.g., Chisholm et al., 2005; Ellis et al., 2003; Mell et al., 2018; Nettle et al., 2011).

However, it is unclear whether such associations between childhood environment and reproductive schedule indeed reflect adaptive properties. The condition for the evolution of such phenotypic plasticity in response to childhood environments have been discussed (Nettle & Bateson, 2015). For the evolution of phenotypic plasticity that exhibits an accelerated reproductive schedule in response to childhood adversity, it is required that earlier reproductive initiation is associated with higher lifetime reproductive success among individuals who experienced childhood adversity, and with lower lifetime reproductive success among individuals who did not experience childhood adversity. However, to the best of our knowledge, no study have empirically examined whether these conditions hold among female humans. A comparable study on female baboons showed that these conditions do not hold: Individuals who started reproduction earlier were associated with higher fitness regardless of whether they had experienced early-life stress (Weibel et al., 2020).

1.2.2. Psychological and Behavioral Tendencies

In psychological research, life history theory has been applied to various individual differences in psychological and behavioral tendencies (Figueredo et al., 2005; Griskevicius et al., 2013; Pepper & Nettle, 2017). Particularly, impulsivity or short-sighted decision making is hypothesized to be a part of fast life history strategy or a mechanism which guides adaptive behavior in adverse environmental conditions (Griskevicius et al., 2013; Kruger et al., 2008). Empirical studies have supported the associations between childhood adversity and

greater temporal discounting, more risky behaviors, higher aggressiveness, and more self-reported impulsivity and sensation seeking (Chang et al., 2019; Hill et al., 2008; Kruger et al., 2008; Lu & Chang, 2018). Moreover, Griskevicius and colleagues have maintained that individuals exhibit responses corresponding to childhood environmental conditions only when they were faced with current adversity (Griskevicius et al., 2011, 2013; Mittal & Griskevicius, 2014). Indeed, they reported that the effect of childhood adversity on impulsivity was observed only when economic recession or mortality threat was primed (but see Amir et al., 2018, for a replication failure).

However, as in the case of reproductive schedule, whether adjusting the level of impulsivity in response to the childhood environment is indeed adaptive has not been examined. Moreover, the claim by Griskevicius et al. (2013) that the expression of the strategies adjusted in response to childhood conditions is further adjusted in response to current conditions is even more problematic because theoretical models have not examined such a two-tiered behavioral adjustment mechanism.

1.3. Overview of Present Research

The psychological life history theory approach is prevalent despite its questionable theoretical bases. Therefore, the present research critically tested its uncritically accepted assumption. For the psychological life history theory account to be valid, or for life history strategies to be truly adaptive phenotypic plasticity, fast strategies must be more adaptive than slow strategies among those who experienced childhood adversity, and slow strategies must be more adaptive than fast strategies among those who did not experience childhood adversity. Therefore, we examined the interaction between early environmental conditions and later behavioral tendencies (which are supposedly manifestations of life history strategy) on biological fitness. Particularly, in this research, we focused on two oft-used putative life

history strategies: impulsivity and reproductive timing.

In Study 1, we focused on impulsivity phenotypic plasticity. We tested the assumption that impulsivity is associated with higher fitness among those who experienced childhood adversity and lower fitness among those who did not. In Study 2, we critically examined reproductive acceleration, a supposed adaptive response to environmental adversity. We tested the assumption that earlier reproductive initiation is associated with higher reproductive success among those who experienced childhood adversity and not among those who did not.

Previous studies have reported associations between childhood adversity and later behavioral tendencies. However, to the best of our knowledge, no study has yet examined the fitness consequences of phenotypic switching in response to environmental conditions. In the present research, we targeted individuals beyond reproductive age and measured their reproductive success (i.e., number of children) in addition to childhood adversity and impulsivity or reproductive timing. This allowed us to test the assumption of life history theory in psychology, that is, the adaptiveness of putative life history strategies.

2. Study 1

Phenotypic plasticity has long been recognized in evolutionary biology but has remained underappreciated until recently (Sommer, 2020). Although many instances of plasticity may be byproducts of physical and chemical development processes, certain kinds of plasticity are adaptive reactions to environmental variations (Nijhout, 2003). Recently, psychological studies on phenotypic plasticity have increased under the rubric of life history theory (Del Giudice et al., 2015; Nettle & Frankenhuis, 2020). However, phenotypic plasticity and life history theory do not necessarily go together in evolutionary biology. Consequently, less attention has been paid to the literature on biological phenotypic plasticity when evaluating purported phenotypic plasticity in humans. In this study, drawing on a biological model of phenotypic plasticity, we critically examine whether an oft-cited human phenotypic plasticity (i.e., early-life adversity causing impulsivity in adulthood) is an evolvable reaction norm.

2.1. Background and Objectives

2.1.1. Low Socioeconomic Status and Impulsivity

According to Nettle and Frankenhuis (2020), the core idea of life history theory in evolutionary biology is that natural selection acts on phenotypes associated with different components of fitness (e.g., survival and reproduction). Its goal is to understand the population-specific optimization of trade-offs between different fitness components under different environments. Hence, it is often used to explain cross-species or -population differences. In contrast, life history theory in psychology almost exclusively focuses on

individual differences along the so-called fast–slow continuum: Whether each individual prioritizes reproduction (i.e., learning toward fast strategies) or survival (i.e., learning toward slow strategies) in response to their local environment.

Such an empirical focus of psychological life history theory has its roots in abundant evidence showing that low socioeconomic status (SES) is associated with a set of apparently problematic behaviors (or behavioral constellation of deprivation [BCD]), such as saving less for the future, having teen pregnancies, investing less in one's children, having poor diets, using illicit drugs, consuming an excessive amount of alcohol, and smoking (Pepper & Nettle, 2017). Although BCD is often viewed as maladaptive, Pepper and Nettle maintain that BCD may be more accurately understood as a contextually appropriate response to environmental uncertainty experienced by low SES individuals (e.g., if you do not expect to survive until next year, there is little point in saving for the distant future). Accordingly, Pepper and Nettle consider that BCD may represent evolved responses to environmental uncertainty.

In addition, life history theory in psychology tends to emphasize the role of childhood environments (Nettle & Frankenhuis, 2020). Its tenet is that early-life adversity, such as childhood economic harshness, causes accelerated reproductive timing and other traits associated with BCD. Empirical studies have supported the effects of early-life adversity (e.g., Griskevicius et al., 2013; Mittal & Griskevicius, 2014). For example, Griskevicius et al. (2013) exposed half of the participants to a series of images indicative of economic recession (recession condition), while the other half was exposed to a series of images of nature (control condition). Participants then engaged in two tasks relevant to BCD (i.e., temporal discounting and risk taking), which Griskevicius et al. referred to as "impulsivity." Here, we follow this terminology and define *impulsivity* loosely as referring to

a group of traits that are theorized as outcomes of childhood adversity. Dividing participants into high vs. low groups by the criterion of one standard deviation above or below the mean, Griskevicius et al. found a 2 (childhood SES) × 2 (economic threat priming) interaction effect on impulsivity. In the recession condition, individuals from low SES families exhibited greater temporal discounting and risk-taking tendencies than individuals from high SES families, but no such difference was observed in the control condition. If we assume that an immediate economic threat reveals one's disposition, the result is congruent with life history theory in psychology, which predicts that individuals grow more impulsive in response to early-life adversity (but see Amir et al., 2018, for a replication failure).

2.1.2. Evolution of Reaction Norms

Although the results of Griskevicius et al. (2013) and other similar findings (e.g., Griskevicius et al., 2011; Mittal & Griskevicius, 2014; see Del Giudice et al. 2015, for a review) are congruent with life history theory in psychology, the psychology version of life history theory has been criticized in various aspects. For example, critics question, whether there are, in fact, trade-offs in reproduction and survival in humans and whether various traits necessarily cluster together along the fast–slow continuum (see articles in the special issue of *Evolution and Human Behavior* on "current debates in human life history research" edited by Frankenhuis & Nettle, 2020). Although many criticisms have been directed at its validity as a branch of life history theory, some have paid critical attention to another aspect: an evolutionary account of human plasticity (e.g., Galipaud & Kokko, 2020; Nettle et al., 2013).

For example, it may not be adaptive for any long-lived species, such as humans, to set their phenotypes based on their childhood environments because their environments may change over their long lifespans (Nettle et al., 2013). In this case, reversible lifelong plasticity may be more adaptive (Ratikainen & Kokko, 2019). However, there are reasons to expect that

childhood SES may be predictive of one's later condition (Nettle et al., 2013). For example, early-life malnutrition may have lasting effects on one's health status. Educational disadvantage during childhood may also accentuate initial disparities later in life. Although such prolonged effects of early-life adversity may make accelerated reproductive timing adaptive, a non-adaptationist account is also possible: Detrimental psychological effects of early-life adversity may be a byproduct of disruption of normal brain development (e.g., McLaughlin et al., 2014). Therefore, there are no firm answers to the question of whether apparent human phenotypic plasticity is in fact an instance of an evolved reaction norm.

As a first step to answering this question, we drew on a mathematical model of adaptive reaction norms developed in biology (Hazel et al., 1990; see also Kokko, 2007, for a simplified version). One application of this model is the predator-induced polymorphism in the acorn barnacle *Chthamalus anisopoma* (Lively, 1986a, 1986b). Although they normally develop conical shells, some develop bent shells in the presence of the predatory snail *Acanthina angelica*. This predator-induced polymorphism is adaptive because the bent morph is costly (i.e., less fecund), but more resistant to predators' attacks. This logic can be readily applied to the impulsivity reaction norm (Table 2.1). In the absence of childhood environmental harshness (high SES, conceptually corresponding to predator-absence), impulsivity is costly: High impulsivity is associated with lower fitness (α_0) than low impulsivity (β_0) is. In the presence of childhood environmental harshness (low SES, corresponding to predator-presence), impulsivity confers fitness benefits: High impulsivity is associated with higher fitness (α_1) than low impulsivity (β_1) is. In addition, it is plausible to assume that environmental harshness adversely influences fitness. Thus, the inequality $\beta_0 > \alpha_0 > \alpha_1 > \beta_1$ must hold for the reaction norm of impulsivity to be evolvable.

Table 2.1

Four Types of Individuals (Groups 1 to 4) Divided by Childhood Environment (Harsh vs. Not Harsh) and Impulsivity (High vs. Low)

		Childhood SES	
		High (0)	Low (1)
Impulsivity	High (α)	α_0 (Group 2)	**$\underline{\alpha_1}$** (Group 4)
	Low (β)	**$\underline{\beta_0}$** (Group 1)	β_1 (Group 3)

Note. Underline indicates greater fitness in each column.

2.1.3. The Present Study

To test whether the condition for the evolution of impulsivity reaction norm holds, we conducted a modified replication of Griskevicius et al. (2013). In addition to the assessment of each participant's childhood SES and their impulsivity, we had participants report five proxy indices of fitness (i.e., the number of children, marriage experience, annual household income, subjective SES, and life satisfaction). We specifically recruited participants in their early 40s because our indices of fitness included the number of children. The set of three measures (i.e., childhood SES, impulsivity, and fitness) allowed us to test the condition for the adaptive reaction norm. The first two measures (childhood SES and impulsivity) were used to divide participants into four groups: high childhood SES individuals exhibiting low impulsivity (Group 1); high childhood SES individuals exhibiting high impulsivity (Group 2); low childhood SES individuals exhibiting low impulsivity (Group 3); and low childhood SES individuals exhibiting high impulsivity (Group 4). We then tested whether the inequality held for each of the five proxy indices of fitness. In particular, we tested whether impulsivity was associated with higher fitness among individuals from low SES families ($\alpha_1 > \beta_1$) but lower fitness among individuals from high SES families ($\beta_0 > \alpha_0$).

This study has three features that merit additional explanation. First, the proxy indices of fitness included variables that are not usually considered relevant to biological fitness. The straightforward index of fitness is the number of children. However, owing to the availability of contraceptive methods in modern environments, successful individuals may not necessarily have more children (e.g., Pérusse, 1993; Vining, 1986; but see Stulp & Barrett, 2016, for counterevidence). Accordingly, we assessed participants' marriage experiences, which would have led to having children in ancestral environments. Financial

success, which is associated with high status, would also have resulted in more children and enable greater parental investment in offspring in ancestral environments. Therefore, we measured participants' annual incomes and subjective evaluations of their SES. In addition, the overall evaluation of one's quality of life may reflect some components that would have been associated with fitness in ancestral environments. In summary, to fill the gap between modern and ancestral environments, we included a wide range of variables, but recognized that some may not be relevant to fitness.

Second, unlike Griskevicius et al.'s (2013) study, we did not include economic recession priming. Our study was conducted in the midst of the COVID-19 pandemic, and people were continually exposed to real-world news about economic crises. Instead of including the priming task, we assessed whether participants' incomes had decreased from the year 2019 (one year before data collection and the COVID-19 pandemic) to 2020. Assuming that a real decrease in annual income would have a greater psychological effect than priming tasks, we also separately analyzed the data from participants whose incomes had decreased from the previous year, which did not change the general pattern of the results.

Third, as an additional measure of impulsivity, as it was loosely defined in this study, we included the Mini-K scale (Figueredo et al., 2006), which was designed to measure individual differences in fast vs. slow life history strategies. We decided to expand the measures of "impulsivity" because human life history research encompasses a wide range of traits as dependent variables, despite a lack of evidence that those traits do in fact cluster together (Sear, 2020). For example, risky behaviors may refer to at least two distinct constructs: activities that would increase the likelihood of undesirable outcomes (e.g., unprotected sex, drug use) and the willingness to accept outcome variance, the latter being standard operationalization in laboratory studies (Pepper & Nettle, 2017). Therefore, we

considered it better to expand our independent variables to cover as many purported life history traits as possible. Although some scholars question its psychometric properties (e.g., Richardson et al., 2017), the Mini-K scale is often used to measure one's position on the fast–slow continuum (e.g., Figueredo et al., 2014). Accordingly, we included it as an additional measure.

We preregistered the protocol for this study on the Open Science Framework (OSF; https://osf.io/tuav4/).

2.2. Method

2.2.1. Participants

A sample of 4,579 Japanese community-based participants was recruited in September 2020 through an online survey service (Cross Marketing Inc., Japan). Given that our primary dependent variable was the number of children, we specifically recruited participants aged 40–45 years, surpassing the Japanese average first-marriage age of 30.7 and 29.0 years for men and women, respectively (Ministry of Health, Labour and Welfare, 2016). We included a stringent attention check (i.e., asking participants to ignore an entire page of the survey; Oppenheimer et al., 2009; see also Miura & Kobayashi, for the Japanese version) along with two standard attention checks (i.e., asking participants to choose a specific option). Consequently, a large number of participants (3,446 participants) failed. We also excluded 441 participants, most of whom were not in the required age range. We retained 692 participants (352 women, 337 men, and 3 not reported; M_{age} = 42.72, SD_{age} = 1.73), which was slightly below the preregistered sample size of 700, for subsequent analyses.

2.2.2. Independent Variables

The childhood SES measure consisted of three items (e.g., "My family usually had enough money for things when I was growing up"; Griskevicius et al., 2013; see Table A1 in

Appendix A). Participants responded to each item on a nine-point scale (1 = *strongly disagree* to 9 = *strongly agree*; Cronbach's α = .79).

We operationalized impulsivity in three ways: temporal discounting, risk taking, and the Mini-K score. The temporal discounting task consisted of 20 choices between an immediate small reward and a larger delayed reward (e.g., receiving JPY 4,100 tomorrow vs. receiving JPY 5,100 after 33 days; see Table A4 in Appendix A). The risk-taking task consisted of 20 choices between definitely earning a fixed amount of money and earning a larger amount of money with a certain probability (e.g., earning JPY 5,600 for sure vs. a 54% chance of earning JPY 6,500; see Table A5 in Appendix A). The frequency of choosing the immediate reward options and gambling options was used as the index of impulsivity for the temporal discounting and risk-taking tasks, respectively. These materials, translated into Japanese by the authors, were adapted from Griskevicius et al. (2013).

Moreover, participants filled out the Japanese version of the 20-item Mini-K scale (Figueredo et al., 2006; Kawamoto, 2015; see Appendix A6), designed to measure individual differences in fast vs. slow life history strategy (e.g., "I often make plans in advance"). Participants rated each item on a seven-point scale (−3 = *strongly disagree* to 3 = *strongly agree*). We excluded two items that assess participants' childhood experiences, which have some overlap with the independent variable, childhood SES. The Mini-K score (Cronbach's α = .84) represents orientation toward the slow life history strategy, with higher scores indicating lower impulsivity.

2.2.3. Dependent Variables

The dependent variables were proxy indices of participants' fitness (i.e., the number of children, marriage experience, annual household income, subjective SES, and life satisfaction). A straightforward fitness index was reproductive success, operationalized as the

number of children that participants had throughout their lives. We explicitly asked them to include any children from previous marriages. *Marriage experience* (i.e., never married vs. married at least once) was also included; lifelong unmarried individuals are considered less reproductively successful. *Annual household income* and *subjective SES* were included because wealth enables greater investment in offspring. Participants reported their household income last year, broken down into six ranges: <1.5, [1.5, 3), [3, 5), [5, 7), [7, 10), and ≥ 10, in million JPY (integer values of 1–6 were assigned in this order). Subjective SES was measured using the MacArthur Scale (N. E. Adler et al., 2000), requiring participants to choose one of 10 SES-level rungs on a ladder. Life satisfaction was included, assuming that participants' assessment of their own quality of life may be associated with some aspects of biological fitness. It was measured using the Satisfaction With Life Scale (Diener et al., 1985; Koyasu et al., 2012) consisting of five items (e.g., "In most ways my life is close to my ideal"; see Table A7 in Appendix A), rated on a seven-point scale (1 = *strongly disagree* to 7 = *strongly agree*; Cronbach's $\alpha = .90$).

2.2.4. Analyses

We divided the participants into four groups by bivariate median splits, combining childhood SES with one of the three impulsivity indices (i.e., temporal discounting, risk taking, or life history strategy). The four groups were: low impulsivity, high childhood SES (Group 1; fitness denoted as β_0 in Table 2.1); high impulsivity, high childhood SES (Group 2; α_0); low impulsivity, low childhood SES (Group 3; β_1); and high impulsivity, low childhood SES (Group 4; α_1). Based on the hypothesized inequality, we ran a set of three planned contrasts with weights of (1, −1, 0, 0), (0, 0, 1, −1), and (0.5, 0.5, −0.5, −0.5) for (Group 1, Group 2, Group 3, Group 4). The first two contrasts were designed to test $\beta_0 > \alpha_0$ and $\alpha_1 > \beta_1$, respectively. The third contrast corresponded to the comparison between high and low

childhood SES groups, included to test the basic presumption that childhood economic adversity detrimentally influences later fitness. The data used in the reported analyses and the R code are available from OSF (https://osf/tuav4/).

2.3. Results

Tables 2.2 and 2.3 show descriptive statistics and correlation matrices of the variables of interest (Table 2.2 for the independent variables and Table 2.3 for the dependent variables). We first examined whether childhood SES was negatively associated with impulsivity. Contrary to previous studies, childhood SES was not significantly associated with temporal discounting ($r_{668} = -.067$, $p = .082$) or risk taking ($r_{673} = .014$, $p = .712$). However, it was significantly associated with a self-rated slow life history strategy (i.e., Mini-K score; $r_{663} = .26$, $p < .001$).

Figure 2.1 shows the distributions of the five fitness indices of Groups 1 to 4. The four groups were divided by childhood SES and impulsivity, which was operationalized as temporal discounting in Figures 2.1A–E, risk-taking tendency in Figures 2.1F–J, and Mini-K score in Figures 2.1K–O. Although the hypothesized inequality predicts a horizontally mirrored J-shaped pattern, none of the figures shows this (except Figure 2.1A, which exhibits a slight and nonsignificant tendency toward a horizontally mirrored J-shape).

When impulsivity was operationalized by temporal discounting (see Figures 2.1A–E; Table 2.4), Contrasts 1 and 2 yielded no significant results for any of the five fitness indices. When impulsivity was operationalized by risk-taking tendency (see Figures 2.1F–J; Table 2.4), although Contrast 2 revealed no significant results, Contrast 1 showed significant effects opposite to the hypothesis: Group 2 reported having more children ($b = -0.24$, $SE = 0.06$, $p < .001$), higher annual household income ($b = -0.17$, $SE = 0.08$, $p = .032$), higher subjective SES ($b = -0.35$, $SE = 0.10$, $p < .001$), and higher life satisfaction ($b = -0.15$, $SE = 0.07$, p

= .031) than Group 1.

When impulsivity was operationalized by Mini-K score (see Figures 2.1K–O; Table 2.3), regardless of childhood SES (i.e., in both Contrasts 1 and 2), lower impulsivity was consistently associated with higher fitness: more children (b = 0.22, SE = 0.07, $p < .001$ and b = 0.41, SE = 0.07, $p < .001$ for Contrast 1 and 2, respectively), higher likelihood of marriage (b = 0.61, SE = 0.12, $p < .001$ and b = 0.61, SE = 0.12, $p < .001$), higher annual household income (b = 0.24, SE = 0.08, p = .003 and b = 0.39, SE = 0.08, $p < .001$), higher subjective SES (b = 0.38, SE = 0.10, $p < .001$ and b = 0.50, SE = 0.09, $p < .001$), and higher life satisfaction (b = 0.39, SE = 0.07, $p < .001$ and b = 0.52, SE = 0.06, $p < .001$). This pattern suggests that impulsivity was selected against regardless of childhood economic adversity.

Contrast 3 (high vs. low childhood SES) was tested three times, though redundant, for the three grouping schemes. Therefore, as shown in Table 2.4, despite grouping schemes, Groups 1 and 2 (high childhood SES groups) were associated with higher fitness than Groups 3 and 4 (low childhood SES groups) on every measure (except for marriage experience where impulsivity was manipulated with Mini-K score), confirming the basic presumption that low childhood SES adversely influenced later fitness. Parenthetically, this result suggests the criterion validity of our childhood SES measure as well as fitness indices: Despite contemporary Japan's low fertility (1.36 in 2019; Ministry of Health, Labour and Welfare, 2020) and ever increasing lifetime unmarried rate (23.4 and 14.1 for males and females, respectively, in 2015; Ministry of Health, Labour and Welfare, 2016), the influence of the theoretically relevant variable (i.e., childhood SES) was observed for the number of children and marriage experience.

Table 2.2

Descriptive Statistics and Correlation Matrix of the Independent Variables

Variable	*N*	Mean	Median	*SD*	1	2	3
1. Temporal discounting	672	5.16	4.00	5.34	–		
2. Risk taking	676	6.61	4.00	6.74	.05	–	
3. Mini-K	666	0.35	0.38	0.84	−.02	.04	–
4. Childhood SES	687	4.61	4.67	1.57	−.06†	.01	.26***

*** $p < .001$. † $p < .100$.

Table 2.3

Descriptive Statistics and Correlation Matrix of the Dependent Variables

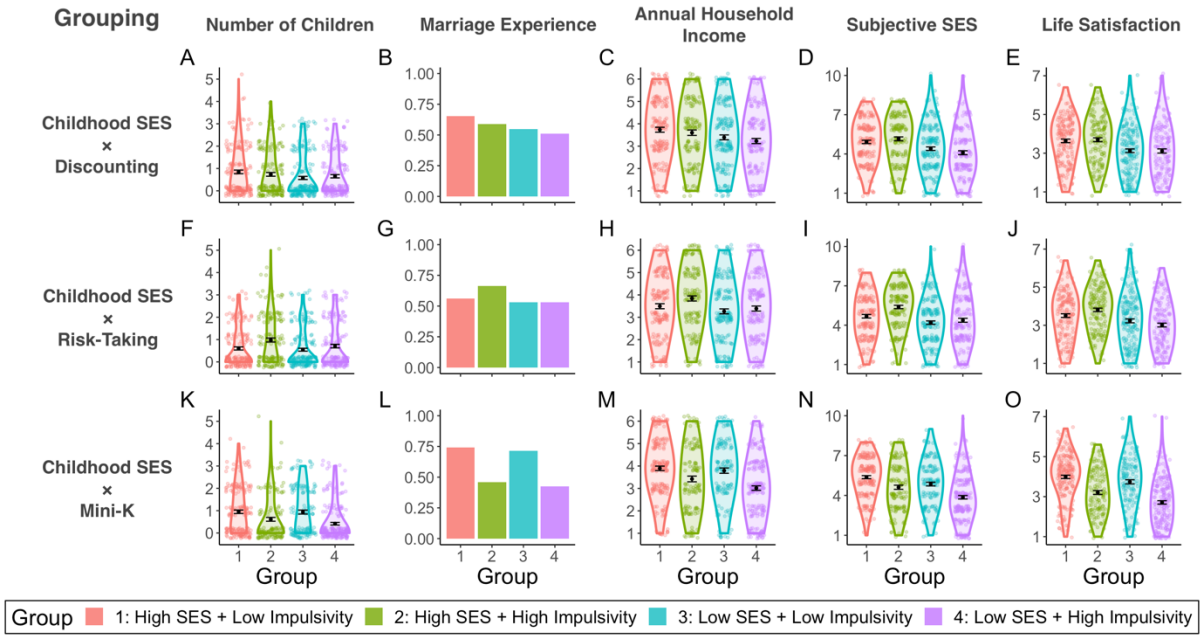
Variable	*N*	Mean	*SD*	1	2	3	4
1. Number of children	686	0.69	0.99	–			
2. Marriage experience	687	0.57	–	.59***	–		
3. Annual household income	682	3.49	1.46	.25***	.34***	–	
4. Subjective SES	688	4.63	1.83	.26***	.37***	.52***	–
5. Life satisfaction	681	3.38	1.29	.28***	.39***	.37***	.55***

Note. Marriage experience: 0 = lifetime singlehood, 1 = having at least one marriage.

*** $p < .001$.

Figure 2.1

Distribution of Fitness Indices of Four Groups Divided by Medians of Childhood Socioeconomic Status (SES) and Impulsivity



Note. The hypothesis predicts a horizontally mirrored J-shape: Highest, second highest, lowest, second lowest for Groups 1–4, respectively. Results based on childhood SES × temporal discounting grouping, childhood SES × risk taking grouping, childhood SES × Mini-K grouping are presented as Panels A–E, Panels F–J, and Panels K–O, respectively. The dependent variables were the number of children for Panels A, F, and K; marriage experience for Panels B, G, and L; annual household income for Panels C, H, and M; subjective SES for Panels D, I, and N; and life satisfaction for Panels E, J, and O.

Table 2.4

Results of a Series of Planned Contrast Analyses

	Variable Used for Grouping								
	Temporal Discounting			Risk Taking			Mini-K		
	*n*s = 173, 149, 190, and 158 for Groups 1, 2, 3, and 4, respectively			*n*s = 167, 160, 183, and 165 for Groups 1, 2, 3, and 4, respectively			*n*s = 185, 138, 145, and 197 for Groups 1, 2, 3, and 4, respectively		
	Corresponding to Panels A–E of Figure 2.1			*Corresponding to Panels F–J of Figure 2.1*			*Corresponding to Panels K–O of Figure 2.1*		
	b	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*
DV = Number of Children									
Contrast 1	0.07	0.06	.271	***−0.24***	0.06	<.001	**0.22**	0.07	<.001
Contrast 2	−0.06	0.07	.373	−0.13	0.07	.059	***0.41***	0.07	<.001
Contrast 3	**0.25**	0.09	.007	**0.21**	0.09	.023	**0.20**	0.10	.038
DV = Marriage Experience (0 = lifetime singlehood, 1 = having at least one marriage)									
Contrast 1	0.14	0.12	.218	−0.22	0.12	.055	**0.61**	0.12	<.001
Contrast 2	0.08	0.11	.482	−0.00	0.11	.994	***0.61***	0.12	<.001
Contrast 3	**0.38**	0.16	.015	**0.34**	0.16	.030	0.14	0.17	.410
DV = Annual Household Income									
Contrast 1	0.07	0.08	.420	***−0.17***	0.08	.032	**0.24**	0.08	.003
Contrast 2	0.08	0.08	.297	−0.06	0.08	.424	***0.39***	0.08	<.001
Contrast 3	**0.36**	0.11	.002	**0.34**	0.11	.003	**0.26**	0.11	.022
DV = Subjective SES									
Contrast 1	−0.12	0.10	.241	***−0.35***	0.10	<.001	**0.38**	0.10	<.001
Contrast 2	0.15	0.10	.114	−0.09	0.10	.338	***0.50***	0.09	<.001
Contrast 3	**0.79**	0.14	<.001	**0.75**	0.14	<.001	**0.64**	0.14	<.001
DV = Life Satisfaction									
Contrast 1	−0.03	0.07	.678	***−0.15***	0.07	.031	**0.39**	0.07	<.001
Contrast 2	0.01	0.07	.932	0.11	0.07	.108	***0.52***	0.06	<.001
Contrast 3	**0.55**	0.10	<.001	**0.54**	0.10	<.001	**0.37**	0.09	<.001

Note. Number of children was submitted to Poisson regression analyses; marriage experience weas submitted to logistic regression analyses. **Bold** coefficients indicate significant difference in the hypothesized direction. ***Bold italic*** coefficients indicate significant difference opposite to the hypothesized direction.

We conducted four sets of robustness checks. Figures and tables involving these analyses are provided in the Appendix B. First, we redefined the four groups using the upper and lower tertiles of childhood SES and impulsivity scores (see Figure B1 and Table B1). Second, we focused on participants experiencing real economic threats whose annual household income decreased from the previous year, possibly owing to COVID-19 (see Figure B2 and Table B2). Third, the same analyses were conducted separately by sex (see Figure B3 and Table B3 for males; Figure B4 and Table B4 for females). Fourth, we tested the hypothesis without splitting the participants into four groups. In a series of regression analyses that treat childhood SES and impulsivity measures as continuous variables, each of the five fitness measures was regressed on childhood SES, one of the three impulsivity measures (either temporal discounting, risk-taking tendency, or Mini-K score) and their interaction term (see Table B5 for the entire sample; Table B6 for the subsample of participants whose annual household income decreased from the previous year; Table B7 for the male sample; and Table B8 for the female sample). These robustness checks revealed a mostly identical pattern, with only one marginal interaction consistent with the hypothesis in the multiple regression on the number of children in the female sample (see Table B8).

In addition, because the fast strategy may result in a greater likelihood of divorce and remarriage, we conducted the three planned contrasts using the number of marriages as the dependent variable. The results were mostly similar to the planned contrasts that included the other fitness indices as the dependent variables (see Table B9).

2.4. Discussion

We examined the evolvability of the impulsivity reaction norm. Although the results confirmed the basic assumption that childhood economic harshness adversely influenced participants' later fitness, none of the other results supported the impulsivity reaction norm's

evolvability: When operationalized as risk-taking tendency, impulsivity was associated with higher fitness among individuals with high, but not low childhood SES. When operationalized by Mini-K score, it was associated with lower fitness regardless of childhood SES.

The present study failed to replicate the results of previous studies (Griskevicius et al., 2013). In particular, childhood SES was not significantly associated with either risk taking or temporal discounting. Given the robust association between childhood SES and BCD (Pepper & Nettle, 2017), the present study's operationalization of impulsivity might have provided inadequate indices of impulsivity. For example, one could argue that the temporal discounting and risk-taking tasks should have been incentivized (but see Amir et al., 2018, who reported no systematic differences between incentivized and non-incentivized risk-taking tasks). Mishra et al. (2017) recently proposed a model of risk-taking (i.e., relative state model) that distinguishes two types of risk-taking behavior: need-based and ability-based risk-taking; the former is motivated by poor environments, whereas the latter is motivated by superior abilities (i.e., the prospect of successful risk-taking). In future studies, it is worthwhile not only to incentivize risk-taking tasks but also to distinguish subtypes of risk-taking and impulsivity based on a nuanced model.

One limitation is that we assessed fitness in a modern, industrialized society, which largely differs from the environment of evolutionary adaptedness (EEA). For example, if low SES conditions in contemporary Japan are still more benign compared with harsh conditions in EEA, the present study may not be a fair test of the hypothesized phenotypic plasticity. Nonetheless, it is worth noting that childhood SES was in fact positively associated with every measure of fitness in this study. Moreover, careful analyses revealed some comparability of the modern and ancestral environments (i.e., positive association between

wealth and the number of children in both modern and ancestral environments; Nettle & Pollet, 2008). Nevertheless, this particular result does not necessarily imply that the comparability between the modern and ancestral environments extends to other aspects. For example, one could argue that impulsivity is an effective strategy for disadvantaged individuals only in EEA but not in the modern environment. Given that a wide range of differences exists between the modern environment and EEA, or the so-called evolutionary mismatch problem (Li et al., 2018), it is informative to replicate this study in populations that maintain traditional lifestyles.

We admit that this study does not disprove the evolvability of human reaction norms as a whole: It only tested the evolvability of impulsivity in response to childhood economic harshness. There are other independent and dependent variables that have attracted researchers' attention in the context of life history theory in psychology. For example, timing of puberty or reproduction and parental strategies are oft-studied life history traits (i.e., dependent variables), and childhood mortality/morbidity and unpredictability are oft-studied environmental (independent) variables (Ellis et al., 2009). Therefore, future studies need to include a wider range of measures of childhood environments and life history traits to test fully the evolvability of any form of phenotypic plasticity in response to early environments.

In summary, this study does not reveal any evidence of the evolvability of the impulsivity reaction norm in response to childhood economic harshness. Therefore, we urge researchers to critically assess the impulsivity reaction norm, especially regarding whether the adaptationist explanation is better supported by empirical data than byproduct explanations.

3. Study 2

It has been reported that exposure to early-life environmental adversity is associated with accelerated reproductive timing among women (Nettle et al., 2011). This association has often been considered evidence of an adaptive phenotypic plasticity: The accelerated reproductive timing is adaptive in harsh environments where mortality risk is high (Ellis et al., 2009; Nettle, 2011; Nettle et al., 2013). According to this explanation, early-life adversity predicts harshness of later environments and organisms to adjust their reproductive timing and maximize their lifetime reproductive success. However, for such adjustment to evolve, there must be a fitness trade-off associated with acceleration (Nettle & Bateson, 2015). In other words, acceleration must enhance organisms' fitness in harsh environments but diminish in benign environments. To best of our knowledge, no studies have directly tested whether fitness trade-off of reproductive acceleration is actually present in human life history. However, it has been reported that in wild baboons, earlier first birth was associated with a higher lifetime reproductive success regardless of early-life condition (Weibel et al., 2020). Moreover, there may be an alternative explanation that the variation in reproductive timing is explained as a mere byproduct of family economic situation. Affluent family environment may allow the daughter to be enrolled in higher education, which in turn delays her reproductive timing. This explanation has nothing to do with adaptation; however, it is consistent with a well-established phenomenon that female education decreases fertility (e.g., Jejeebhoy, 1995). In this study, we tested the validity of the adaptive phenotypic plasticity explanations and pitted them against the byproduct explanation.

3.1. Background and Objectives

3.1.1. Environmental Adversity and Accelerated Reproductive Schedule

In psychology, life history theory is often referred to as a framework that explains variations in human reproductive schedule as adaptive strategies in response to local environments (Del Giudice et al., 2015; Nettle & Frankenhuis, 2020; Sear, 2020). Life history theory in psychology assumes that individuals developmentally exhibit optimal resource allocation strategies (life history strategies) that correspond to their local environments (Ellis et al., 2009). Life history strategies are considered to vary along so-called fast–slow continuum, where accelerated reproductive timing reflects a shift toward the fast pole (i.e., prioritizing reproduction over survival). According to this theory, in adverse environments characterized by high morbidity/mortality, accelerated reproductive schedule is adaptive because realization of reproductive potential is likely to be limited, which leads to the evolution of the phenotypic plasticity of reproductive schedule.

An early version of an adaptationist explanation were proposed by Belsky et al. (1991). Since then, many studies have reported that adverse environmental conditions, particularly in childhood, are associated with accelerated reproductive schedule, such as earlier first birth, earlier sexual debut, and earlier menarche among women (Alvergne et al., 2008; Chisholm et al., 2005; Ellis et al., 2003; Farkas et al., 2022; Hartman et al., 2017; McGinnis et al., 2022; Mell et al., 2018; Nettle, 2010, 2011; Nettle et al., 2011; Quinlan, 2003; Schlomer & Sun, 2022; Sheppard et al., 2016; Sun et al., 2017, 2020; Uggla & Mace, 2016; Waynforth, 2012). Consistent patterns have also been reported at inter-community and inter-country levels, where higher mortality or shorter life expectancy are associated with lower age at first birth (Bulley & Pepper, 2017; Low et al., 2008; Wilson & Daly, 1997). These findings have been interpreted as positive evidence for the existence of the phenotypic

plasticity.

However, these findings focus on populations from developed countries with high-income economies (e.g., the UK, the USA, New Zealand, France). Findings from developing countries or preindustrial populations are relatively scarce. More importantly, the comparable data from developing countries are rather inconsistent (Sear et al., 2019, for a review on pubertal timing). For example, among Filipinos, favorable nutrition and high social class were associated with earlier menarche, whereas parental absence, another form of early-life adversity, was not (Adair, 2001; Kyweluk et al., 2018). Likewise, parental presence and higher social class (but not parental absence and lower social class) were associated with earlier first birth among pre-industrial Finns (Lahdenperä et al., 2007). Larger childhood body size, which reflected favorable nutrition, was associated with earlier first birth among women in horticultural Gambians (Allal et al., 2004). In contrast, father absence was associated with earlier first birth among Malaysians (Sheppard et al., 2014). Moreover, a study on wild baboon (Weibel et al., 2020) found no association between early adversity and age at first birth.

The aforementioned evidence from non-Western, educated, industrialized, rich, and democratic (WEIRD) samples and a non-human primate is, on average, against the notion that female humans accelerate their reproductive timing in response to childhood adversity. In other words, the universality of the phenomenon is not well founded.

3.1.2. Adaptive Phenotypic Plasticity Explanation

According to life history theory in psychology, the association between early-life adversity and accelerated reproductive schedule is explained as an adaptive phenotypic plasticity (Ellis et al., 2009). Although there is a serious doubt about the universality of this association, findings from WEIRD samples are consistent with the adaptive phenotypic

plasticity explanation. However, whether this association reflects an evolved reaction norm cannot be determined by merely observing the association. It is noteworthy that although the validity of this explanation is not directly testable, assumptions of this explanation are empirically testable. Recently, two subclasses of explanations for the adaptive phenotypic plasticity of reproductive schedule have been proposed, each of which provides slightly different assumptions (Nettle & Bateson, 2015; Nettle et al., 2013; Rickard et al., 2014).

Prediction of Environmental Condition. The first type of explanation, which is the *external predictive adaptive response* (hereafter, ePAR), posits that early-life environment provides individuals with predictive cues that allow them to expect their future environmental conditions and adjust their phenotype accordingly (Gluckman et al., 2005a, 2005b). This explanation is based on the predictive accuracy of future external condition. It presumes that one's early-life environment is an accurate predictor of their later (after maturity) environment (Ellis et al., 2009; Monaghan, 2008).

According to this explanation, environmental matches between childhood and after maturity (i.e., predictive accuracy) should result in higher fitness. However, contradictory results have been obtained in a historical Finn population (Hayward & Lummaa, 2013; Hayward et al., 2013) and other relatively long-lived mammals (Douhard et al., 2014; Lea et al., 2015; Pigeon et al., 2017). Individuals who grew up in adverse environments suffered lower fitness (i.e., lower survival and/or lower reproductive success) than those who grew up in benign environments when they found themselves in adverse environments later in life.

Furthermore, scholars generally consider that ePAR is unlikely to evolve in long-lived species because the early environment does not adequately predict their later environment (Nettle et al., 2013; Wells, 2007). Nettle et al. (2013) have shown in a mathematical model that unless the temporal autocorrelation of environmental variable is

extremely high, the conditional strategy to adjust phenotype according to early environment will not evolve in humans. In fact, archival data show that early-life conditions hardly match the environmental conditions during reproductive age among a historical Finn population (Hayward & Lummaa, 2013; Hayward et al., 2013).

Based on our review, it is implausible to assume that the adaptive plasticity of reproductive timing is accounted for by the ePAR explanation. We tested the validity of its assumption. That is, we tested whether, in harsh reproductive environment, individuals who experienced early-life adversity tend to have more offspring than those who did not, whereas in benign reproductive environments, individuals who did not experience early-life adversity tend to have more offspring than those who did.

Prediction of Physical Condition. While ePAR requires environmental consistency, there is another possibility for adaptive benefit of conditioning one's reproductive timing on the early-life environments. This explanation is called the *internal predictive adaptive response* (hereafter, iPAR) because it assumes that early-life environments have prolonged detrimental influences on somatic (or internal) conditions (Nettle et al., 2013; Rickard et al., 2014; Wells, 2012). If childhood adversity shortens healthy life span and period available for reproduction, then accelerated reproductive initiation will be adaptive. Consistent with the assumption of iPAR, there is evidence that exposure to deprived childhood environment is associated with poorer somatic health status or higher mortality risk in adulthood (Danese et al., 2009; Hartman et al., 2017; McGinnis et al., 2022; Mell et al., 2018; Nettle, 2014).

However, it is possible that childhood adversity indirectly affects later health status through unhealthy lifestyle. For example, the socioeconomic harshness may force individuals to eat unhealthy foods or not afford the time to engage in regular exercises. If the association

between early-life adversity and later low health status is not inherent but emerge owing to such cultural factors, natural selection may not act on this association and thus iPAR may not evolve. Therefore, to test the validity of the iPAR explanation, this study examined whether childhood adversity predict later poorer health status after controlling for potential confounding variables (cf. Nettle, 2014).

Adaptiveness of Phenotypic Plasticity. Although the two types of PAR have been distinguished by what childhood environment predicts later in life, they commonly assume a so-called crossover interaction between childhood environment and later phenotype. Evolutionary models of phenotypic plasticity assume the presence of fitness trade-off involved in phenotypic adjustment. According to Nettle and Bateson (2015), only when there is a crossover interaction between environmental condition and phenotype on fitness, phenotypic plasticity can evolve (see also Bateson et al., 2014). In the case of accelerated reproductive schedule, this condition predicts that earlier reproductive initiation increases fitness among those who experienced early-life adversity but decreases fitness among those who did not experience early-life adversity. To the best of our knowledge, no study has tested the presence of this crossover interaction in fitness of human females, although a life history study on female baboons has found negative evidence for the crossover interaction (Weibel et al., 2020). Among female baboons, the earlier first birth unconditionally enhanced their lifetime reproductive success regardless of their experiences of early-life adversity. The present study aims to test whether there is the crossover interaction in the lifetime reproductive success of contemporary Japanese women.

3.1.3. Byproduct Explanation

On the association between childhood adversity and earlier reproductive timing, there could be an alternative explanation that assumed no adaptive properties. Higher

education level or prolonged period of education is associated with delayed reproductive initiation in developed countries (InterLACE Study Team, 2019, for an analysis of data in 10 countries; Sakai et al., 2017, for Japanese data). Education levels may be restricted by family economic status. Women from relatively poor families may not be allowed to be enrolled in higher levels of education, such as colleges and universities. It is reasonable to expect that earlier family economic status predicts later economic status. Therefore, the association between childhood environment and age at first birth may be a spurious correlation because both family economic status and delayed reproductive schedule are possibly correlated with enrollment in higher education.

3.1.4. Preliminary Findings

To obtain preliminary insights regarding the association between early-life environment and reproductive timing, we reanalyzed Study 1 data that are available at Open Science Framework (OSF; https://osf.io/tuav4/). It was revealed that the correlation between female participants' childhood socioeconomic status (SES) and age at first birth (calculated from the current ages of participants and their first child) was significant and positive (r_{152} = .239, p = .003). However, the fitness benefit of reproductive acceleration was not observed. Among women in their early 40s with at least one child, no interaction between childhood SES and age at first birth on the number of children was found (B = −.051, SE = .055, p = .352), whereas the main effect of age at first birth was negative and significant (B = −.288, SE = .057, p = <.001). Therefore, independent of the level of childhood adversity, earlier reproductive initiation was associated with higher reproductive success. This pattern was similar to that of the results of the study on baboons, although the association between early adversity and age at first birth itself was not found among baboon females (Weibel et al., 2020).

Further analyses of data of female participants in Study 1 revealed that education level mediates the association between childhood SES and age at first birth (indirect effect: B = .069, 95% CI [.040, .106]). After controlling for education level, the direct effect of childhood SES on age at first birth was no longer significant (B = .041, SE = .043, p = .337). These results contradict with the adaptive plasticity explanation of reproductive acceleration (either ePAR or iPAR) but rather suggest that the association between childhood environment and timing of reproductive initiation observed in humans may be a mere byproduct of prolonged education among individuals from affluent families.

3.1.5. The Present Study

The results of the reanalyses of data in Study 1 identified a new research topic: The positive association between childhood SES and age at first birth is not an adaptation but a byproduct of prolonged education of high SES group. This study aimed to critically assess the validity of the adaptive phenotypic plasticity explanations that earlier reproductive initiation among individuals from low SES families is an adaptive strategy that corresponds to the environmental condition. If this association reflects such an adaptive property, there must be an interaction effect between childhood SES and age at first birth on lifetime reproductive success.

In addition, we tested the specific prediction and assumption of external/internal PAR models (Nettle et al., 2013; Nettle & Bateson, 2015). The ePAR explanation predicts that the consistency in SES between childhood and young adulthood is associated with higher fitness. The iPAR explanation predicts that lower childhood SES is associated with later detrimental health status, even after controlling for potential confounding variables (i.e., lifestyle variables). Furthermore, we assessed the alternative explanation that the variation in age at first birth is not adaptation but a mere byproduct of prolonged education. To test this

explanation, we examined a mediation effect of education level from childhood SES to age at first birth.

We preregistered the protocol for and the hypotheses of this study on the OSF (https://osf.io/3dyr5/?view_only=).

3.2. Method

3.2.1. Participants

The participants were Japanese women aged 45–50 years who were recruited in February 2022 through Cross Marketing Inc., Japan, which was used in Study 1. The targeted age range surpassed most Japanese women's reproductive age. According to the Japanese annual statistics, births by women aged 45 years or older constitute only 0.03–0.20% of all births for the last 25 years (Ministry of Health, Labour and Welfare, 2022). Therefore, it is reasonable to assume that the number of children reported by our sample well represents their lifetime reproductive success.

We conducted a priori power analyses to determine the sample size. First, we estimated the required sample size to test the correlation between childhood SES and age at first birth, using the pwr.r.test function of *pwr* package in R. Given the correlation coefficient observed in the reanalyses of data in Study 1 (r = .24), the sample size of 146 would be sufficient to attain power of .80. Second, we estimated the required sample size to test the mediation by education level of the effect from childhood SES to age at first birth. We used Fritz and McKinnon's (2007) method to determine the sample size for the mediation analysis by a bias-corrected bootstrap method. Given the path coefficients found in Study 1 data (.39 for the path from childhood SES to education level and .33 for the path from education level to age at first birth), the required sample size to attain power of .80 ranges from 71 to 116, which fell below the required sample size for the test of the childhood SES × age at first birth

correlation.

It is noteworthy that collecting data from 146 individuals is insufficient for these analyses because it minimally requires 146 women who delivered at least one child in the dataset. In Study 1 that used the same company's service but targeted a slightly younger sample (i.e., 40–45 years), 43.6% of the effective female sample reported that they had at least one child. To ensure the required number of women with at least one child, we decided to collect data from 500 women (43% of 500 = 215).

The total number of access to the survey was 636 (after excluding one access from the duplicated IP address), of which 565 participants consented with participation and matched the targeted gender and age (i.e., females aged 45–50 years). Of the 565 participants, 65 participants who failed to pass the attention check items were excluded. In addition, we observed inconsistencies (e.g., reporting "married" for current marital status while reporting "0" for the number of marriage) in 20 participants' responses. Accordingly, we analyzed data from 480 participants (M_{age} = 47.60, SD_{age} = 1.73). There were 251 participants with at least one child, which exceeded the required sample size of 146.

3.2.2. Measured Variables

Socioeconomic Status. Participants responded to the childhood SES measure, which was used in Study 1 (e.g., "My family usually had enough money for things when I was growing up"). We also assessed participants' adolescence SES by modifying the childhood SES measure (e.g., "My family usually had enough money for things when I was a high school student"; see Table A2 in Appendix A). Following Griskevicius et al.'s (2013) method, we assessed participants' young adulthood SES, which consisted of four items (e.g., "I had enough money to buy things I want in my 20s"; see Table A3 in Appendix A). Participants responded to them on a 9-point scale (1 = *strongly disagree* to 9 = *strongly*

agree). Cronbach's α coefficients for childhood, adolescence, and young adulthood SES were .82, .84, and .89, respectively.

To validate the childhood SES measure, we included questions about their experiences and family environments before and during elementary school years. Specifically, we asked (a) whether they attended private classes (*juku*) during their elementary school years; (b) the number of after-school activities (e.g., piano lesson) they were enrolled in before entering elementary school; (c) number of after-school activities during elementary school years; (d) number of times that their parents took them on domestic trips, which involved at least one night stay; (e) number of times that their parents took them on international trips; (f) whether their homes were equipped with air-conditioning; (g) whether their parents possessed a car; (h) whether they had their own room; and (i) whether they had children's room shared with siblings. In addition, we asked participants whether their parents divorced during their childhood because parental divorce was expected to be negatively associated with family financial affluence.

Education Level. Participants provided their educational background. The last school they graduated from was coded as follows: junior high school ($N = 13$), high school ($N = 133$), junior college, vocational school, or technical college ($N = 191$), 4-year university ($N = 127$), master's degree or 6-year university ($N = 14$), and doctoral degree ($N = 2$) (integer values of 1–6 were assigned in this order). The three types of schools (i.e., junior college, vocational school, and technical college) were grouped together because these schools typically require two-year enrollment after completion in high school. Two participants with doctoral degree had no children; therefore, analyses related to age at first birth did not include individuals belonging to this category.

Reproduction and Mating. For variables related to reproduction, participants

were asked the number of children they had, including any children from previous marriages or relationships, and their respective ages at first and last births. In addition, for variables related to mating, participants reported current marital status, the number of marriages, their age at first marriage, total number of romantic partners whom they had dated with, and age at first romantic relationship (age of having first romantic partner). In this study, *age at first birth* was a key variable because life history theory in psychology assumed that it would be accelerated by childhood adversity, particularly low SES.

We used the *number of children* as a straightforward fitness index. In addition, *marriage experience* (i.e., 0 = never married vs. 1 = having married at least once) and the *number of partners* were used as indirect fitness indices. Lifelong unmarried individuals are considered less reproductively successful. The number of romantic relationships may reflect individuals' attractiveness and another aspect of mating success. Participants' *current annual household income* is included as a supplementary fitness index because wealth enables greater investment in offspring. Participants reported their household income in 2021 (the last year before the survey) and 2019 (the year before the COVID-19 pandemic) from six ranges (<1.5, [1.5, 3), [3, 5), [5, 7), [7, 10), and ≥ 10, in million JPY; integer values of 1–6 were assigned in this order). The two responses were strongly correlated (r_{478} = .948, p < .001), and we used the mean of two values as the current household income.

Health Status. The following three variables were included as indices of health status: participants' experiences with disease (number of diseases), timing of first serious disease, and subjective general health.

For the *number of diseases*, participants were shown a list of diseases and were asked to check all diseases they had experienced. The list was developed by the authors based on the statistical classification of diseases used in a patient survey (Ministry of Health,

Labour and Welfare, 2015). A total of 60 diseases (e.g., hypertension, anemia, bronchitis, gastritis, migraine, arthritis) were included. In addition, if participants had experienced any serious diseases that were not included in the list, they were asked to specify them. The total number of diseases experienced was used as an indicator of poor health status.

For the *timing of first serious disease*, participants reported when they experienced their first serious diseases from 10 options (1 = *aged 10 years or younger*, 2 = *early 10s*, 3 = *late 10s*, 4 = *early 20s*, 5 = *late 20s*, 6 = *early 30s*, 7 = *late 30s*, 8 = *early 40s*, 9 = *late 40s*, 10 = *never experienced*).

For the *subjective general health*, participants were asked to rate their own general health status, ranging from 1 to 5 (1 = *very poor*, 2 = *poor*, 3 = *fair*, 4 = *good*, 5 = *very good*). The single-item measure was used in previous studies (Nettle, 2014; Mell et al., 2018).

Lifestyle. For control variables that may influence participants' health status, we assessed their health-related habits during young adulthood: alcohol consumption (referred to as "drinking"), smoking, and exercise.

For *drinking*, participants indicated the frequency of alcohol consumption in their 20s and 30s. For each period, they responded on a 6-point scale (1 = *rarely*, 2 = *1–3 days per month*, 3 = *1–2 days per week*, 4 = *3–4 days per week*, 5 = *5–6 days per week*, 6 = *every day*). They also indicated the duration of habitual drinking on a 6-point scale (1 = *never*, 2 = *less than 5 years*, 3 = *5 years or more and less than 10 years*, 4 = *10 years or more and less than 15 years*, 5 = *15 years or more and less than 20 years*, 6 = *20 years or more*). The mean of the three responses (frequency in 20s, frequency in 30s, and duration of habitual drinking) was used as the score of drinking habit (Cronbach's $\alpha = .84$).

For *smoking*, participants were asked to indicate whether they had habitually smoked cigarettes at any time in their 20s and 30s. We scored each response as 1 for "yes" and 0 for

"no" and used the sum of them as the smoking habit score (i.e., three levels from 0 to 2).

For *exercise*, participants indicated how often they exercised or played sports during their 10s, 20s, and 30s, on a 4-point scale (1 = *less than 1 day per month*, 2 = *1–3 days per month*, 3 = *1–2 days per week*, 4 = *3 or more days per week*). The mean of the three items was used as the score of exercise habit (Cronbach's $\alpha = .69$).

3.2.3. Analyses

Presence of Explanandum. We first examined the association between childhood SES and age at first birth. In addition to the bivariate analysis, to examine the unique effect of childhood SES on age at first birth after controlling for the effect of young adulthood SES, we regressed childhood SES and young adulthood SES on age at first birth.

Fitness Consequences of Conditional Acceleration. The evolution of phenotypic plasticity of reproductive schedule requires the fitness trade-off (Nettle & Bateson, 2015): Earlier reproductive initiation must be associated with higher lifetime reproductive success only among individuals who experienced childhood adversity. Therefore, we examined the consequences of reproductive acceleration on lifetime reproductive success by level of early-life adversity. Lifetime reproductive success was measured by the number of children. However, as the analysis using age at first birth involved participants with at least one child, we used the number of children added after the first birth (i.e., [participant's total number of children] − 1) as outcome variable. Multiple Poisson regression analysis was conducted, with childhood SES, age at first birth, and their interaction term as predictor variables.

In the above analysis, reproductive acceleration was operationalized as age at first birth. However, the effect of delayed reproductive initiation and its interaction with childhood SES were potentially underestimated because of the exclusion of those without children.

Therefore, we conducted similar analyses using different operationalizations: age at first marriage and age at first romantic relationship. The total number of children was the outcome variable and was submitted to Poisson regression analyses.

Fitness Consequences of Environmental Consistency. According to the ePAR model, environmental consistency (i.e., match between childhood and young adulthood environments) must be associated with higher fitness. We conducted regression analyses for the three fitness indices (i.e., number of children, marriage experience, and number of partners). We tested the effect of environmental consistency: First, we tested the childhood SES × young adulthood SES interaction on fitness. Second, we tested whether the absolute difference between childhood SES and young adulthood SES scores (both of which were standardized) would predict fitness outcomes.

Somatic Consequences of Early-Life Adversity. The assumption of the iPAR model is that childhood environmental adversity has a detrimental effect on later somatic status. We regressed each of the health status indicators (i.e., number of diseases, timing of first serious disease, and subjective general health) on childhood SES and young adulthood SES along with potential confounding variables (i.e., drinking, smoking, exercise, and number of children).

Byproduct of Prolonged Education. To test the validity of the proposed non-adaptationist explanation (i.e., delayed reproductive initiation is a mere byproduct of prolonged educational involvement among high SES individuals), we conducted the mediation analysis assuming childhood SES as predictor variable, education level as mediator variable, and age at first birth as outcome variable. In addition, we conducted a similar mediation analysis, but predictor variable was replaced with adolescence SES, which is expected to more directly influence on enrollment in higher education. Furthermore, to

examine whether adolescence SES is a better predictor of education level and age at first birth than childhood SES, (a) we compared the correlation between adolescence SES and education level with the correlation between childhood SES and education level and (b) compared the correlation between adolescence SES and age at first birth with the correlation between childhood SES and age at first birth.

In addition to the preregistered analyses described above, we conducted an exploratory analysis in which life history strategy was operationalized by education level instead of age at first birth. Although education level may be a mere byproduct of childhood environment, adaptationist explanation (i.e., life history theory in psychology or anthropology) often posits that educational investment or duration of educational involvement reflect adaptive strategies in response to childhood environment (Bulled & Sosis, 2010; Richardson et al., 2020). Therefore, we examined whether education per se would have different effects on fitness depending on their childhood environments. We regressed four fitness indices (i.e., number of children, marriage experience, number of partners, and current annual household income) on childhood SES, education level, and their interaction.

3.3. Results

Table 3.1 shows the descriptive statistics of the variables. The correlation coefficients between variables are shown in Tables 3.2 and 3.3. The three SES scores were positively correlated with each other (r_{478} = .908 for childhood and adolescence, r_{478} = .441 for childhood and young adulthood, and r_{478} = .457 for adolescence and young adulthood). As a test of the validity of childhood SES scale, we examined the correlations between childhood SES and the nine items of childhood experiences and parental divorce. Eight of the nine items (except children's room) significantly correlated with childhood SES score in the predicted

directions (rs = .126–.378, df = 478, ps < .01, for eight items; r_{443} = .03, p = .507, for children's room; Table 3.4). Parental divorce was also negatively correlated with childhood SES score (r_{474} = −.211, p < .001). Thus, the validity of this subjective scale was confirmed.

For the robustness check analyses, we created a new variable that reflects participants' childhood home environments. All variables in Table 3.4, except children's room (i.e., having a room shared with other siblings), were aggregated into the single childhood home environment score. For all analyses reported below that used childhood SES as a predictor variable, robustness checks were performed, replacing childhood SES score with childhood home environment score. Confirming the robustness of the results reported in the main text, robustness check analyses revealed largely the same results. Figures and tables involving these analyses are provided in the Appendix C.

Table 3.1

Descriptive Statistics of Measured Variables

Variable	*N*	Mean	Median	*SD*	*Min*	*Max*
Childhood SES	480	4.94	5.00	1.70	1.00	9.00
Childhood home environment	476	4.15	4.00	1.80	0.00	9.00
Adolescence SES	480	4.80	5.00	1.74	1.00	9.00
Young adulthood SES	480	5.33	5.25	1.97	1.00	9.00
Education level [a]	480	3.00	3.00	0.90	1.00	6.00
Age at first birth [b]	251	29.18	29.00	5.08	19.00	41.00
Age at first marriage [b]	349	28.03	27.00	5.38	19.00	48.00
Age at first romantic relationship [b]	448	18.95	18.00	4.07	10.00	44.00
Number of children	480	0.96	1.00	1.07	0.00	5.00
Marriage experience [c]	480	0.73	1.00	0.45	0.00	1.00
Number of partners	479	3.91	3.00	3.27	0.00	25.00
Current annual household income	480	3.56	4.00	1.42	1.00	6.00
Number of diseases	480	2.87	2.00	2.74	0.00	16.00
Timing of first serious disease [d]	480	8.21	10.00	2.87	1.00	10.00
Subjective general health	480	2.64	3.00	0.91	1.00	5.00
Drinking	480	2.74	2.67	1.53	1.00	6.00
Smoking	480	0.54	0.00	0.85	0.00	2.00
Exercise	480	1.69	1.33	0.83	1.00	4.00

[a] 1 = junior high school, 2 = high school, 3 = junior college or vocational school, 4 = 4-year university, 5 = master's degree or 6-year university, 6 = doctoral degree.

[b] Only for participants with at least one child, at least one marriage, or at least one partner.

[c] 0 = never married, 1 = married at least once.

[d] 1 = aged 10 years or younger, 2 = early 10s, 3 = late 10s, 4 = early 20s, 5 = late 20s, 6 = early 30s, 7 = late 30s, 8 = early 40s, 9 = late 40s, 10 = never experienced.

Table 3.2

Pearson's Product-Moment Correlations for Socioeconomic Status Scores, Education Level, and Proxy Fitness Indices

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Childhood SES	–										
2. Childhood home environment	**.50 (.00)**	–									
3. Adolescence SES	**.91 (.00)**	**.50 (.00)**	–								
4. Young adulthood SES	**.44 (.00)**	**.22 (.00)**	**.46 (.00)**	–							
5. Education level	**.24 (.00)**	**.21 (.00)**	**.29 (.00)**	**.29 (.00)**	–						
6. Age at first birth	.12 (.06)	.10 (.08)	.10 (.09)	**.28 (.00)**	**.30 (.00)**	–					
7. Age at first marriage	.08 (.15)	.07 (.22)	.08 (.14)	**.15 (.01)**	**.25 (.00)**	**.87 (.00)**	–				
8. Age at first romantic relationship	−.00 (.99)	**−.13 (.00)**	−.01 (.86)	.05 (.30)	.09 (.07)	**.25 (.00)**	**.27 (.00)**	–			
9. Number of children	.03 (.46)	.06 (.22)	.05 (.29)	−.03 (.48)	−.08 (.08)	**−.28 (.00)**	**−.37 (.00)**	**−.15 (.00)**	–		
10. Marriage experience	.05 (.30)	.04 (.34)	.06 (.19)	.01 (.86)	−.09 (.06)	.05 (.46)	–	**−.14 (.00)**	**.53 (.00)**	–	
11. Number of partners	.03 (.45)	**.12 (.01)**	.03 (.52)	−.02 (.64)	**−.09 (.04)**	.03 (.63)	**.15 (.00)**	**−.36 (.00)**	.07 (.11)	**.14 (.00)**	–
12. Current annual household income	**.18 (.00)**	**.21 (.00)**	**.22 (.00)**	**.27 (.00)**	**.30 (.00)**	.05 (.40)	−.02 (.73)	**−.17 (.00)**	**.16 (.00)**	**.26 (.00)**	.02 (.73)

Note. *p* values in parentheses are rounded to two decimals (i.e., .00 means < .005). **Boldface** indicates $p < .05$. Red cells indicate significant positive correlations, and blue cells indicate significant negative correlations. Correlation between each pair of variables is computed using all complete pairs of observations on those variables.

Table 3.3

Pearson's Product-Moment Correlations for Variables Related to Health and Lifestyle

	Variable	1	2	3	4	5	cSES	ENV	aSES	EDU	AFB	NCH
1.	Number of diseases	–					**−.10 (.02)**	**−.10 (.02)**	**−.16 (.00)**	**−.13 (.00)**	.11 (.08)	−.04 (.34)
2.	Timing of first serious disease	**−.38 (.00)**	–				.06 (.20)	.04 (.33)	.05 (.31)	.01 (.81)	−.05 (.06)	.05 (.27)
3.	Subjective general health	**−.42 (.00)**	**.20 (.00)**	–			**.22 (.00)**	**.16 (.00)**	**.28 (.00)**	**.15 (.00)**	−.05 (.39)	**.09 (.04)**
4.	Drinking	**.09 (.00)**	.00 (.93)	−.03 (.93)	–		−.05 (.28)	−.01 (.76)	.01 (.55)	−.03 (.55)	−.01 (.93)	.04 (.36)
5.	Smoking	.08 (.06)	.03 (.48)	**−.10 (.03)**	**.26 (.00)**	–	**−.09 (.04)**	−.04 (.35)	**−.16 (.00)**	**−.17 (.00)**	−.10 (.10)	.02 (.70)
6.	Exercise	−.03 (.50)	−.04 (.38)	**.12 (.01)**	**.13 (.00)**	−.03 (.55)	**.12 (.01)**	.08 (.08)	**.11 (.02)**	**.14 (.00)**	.04 (.54)	.02 (.67)

Note. *p* values in parentheses are rounded to two decimals (i.e., .00 means < .005). **Boldface** indicates $p < .05$. Red cells indicate significant positive correlations, and blue cells indicate significant negative correlations. Correlation between each pair of variables is computed using all complete pairs of observations on those variables. cSES = childhood socioeconomic status; ENV = childhood home environment; aSES = young adulthood socioeconomic status; EDU = education level; AFB = age at first birth; NCH = number of children.

Table 3.4

Descriptive Statistics and Correlation Coefficients with Childhood Socioeconomic Status (SES) of Variables of Childhood Home Environment and Parental Divorce

Variable	*N*	Mean	Proportion of 1	Median	*SD*	*Min*	*Max*	*r* with CSES
Private class (*Juku*) [a]	480	−	0.31	−	−	0	1	.25***
Lessons before elementary school years [b]	480	1.03	−	1	1.32	0	10	.26***
Lessons during elementary school years [b]	480	1.82	−	2	1.33	0	8	.24***
Domestic trips [b]	480	7.15	−	5	6.90	0	20	.38***
International trips [b]	480	0.26	−	0	1.59	0	20	.13**
Air conditioner [a]	480	−	0.55	−	−	0	1	.29***
Car [a]	480	−	0.82	−	−	0	1	.13**
Own room [a]	480	−	0.49	−	−	0	1	.26***
Children's room [a]	445	−	0.60	−	−	0	1	−.03
Parental divorce [a]	476	−	0.10	−	−	0	1	−.21***

Note. CSES = childhood SES.

[a] 0 = no, 1 = yes. [b] Count data.

** $p < .01$. *** $p < .001$.

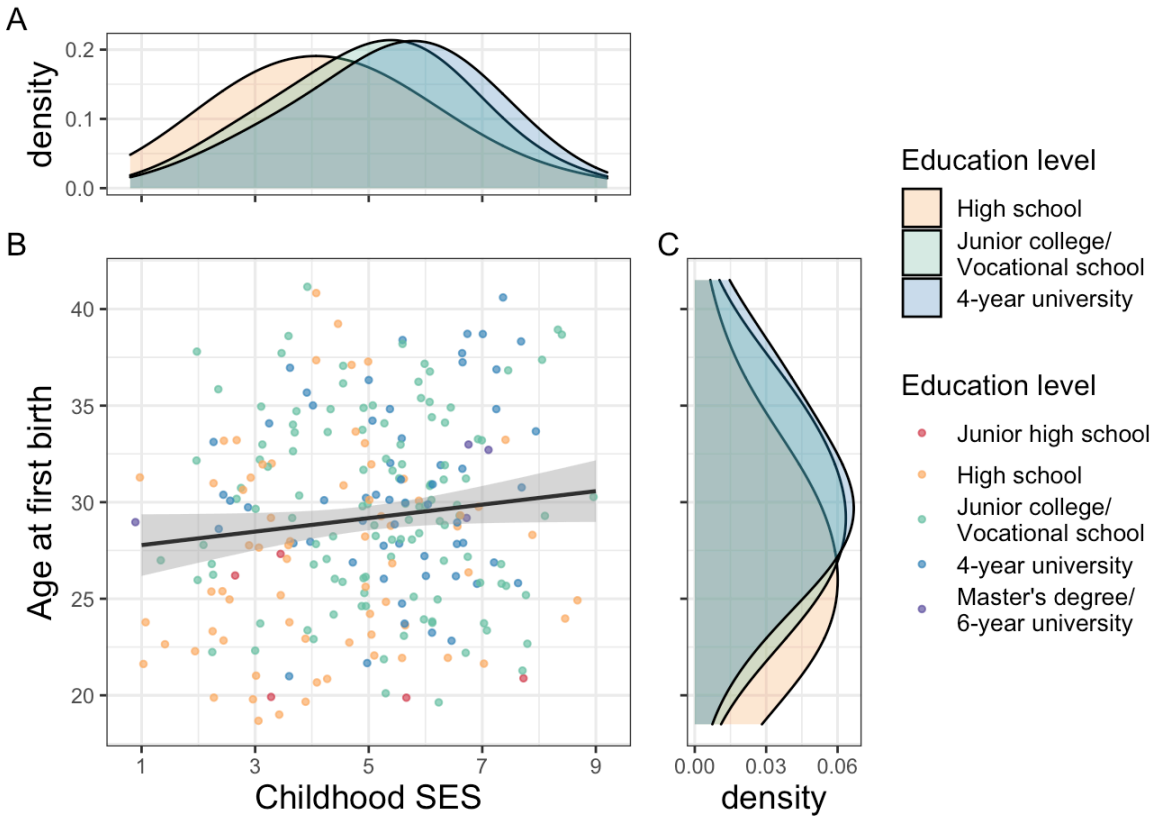
3.3.1. Presence of Explanandum

We first examined the association between childhood SES and age at first birth (see Figure 3.1). In simple regression analysis, childhood SES marginally significantly predicted age at first birth (B = .117, SE = .062, p = .061). After controlling for young adulthood SES, the association was not significant (B = .003, SE = .067, p = .965), whereas young adulthood SES was positively associated with age at first birth (B = .285, SE = .069, p < .001). Thus, childhood SES did not uniquely predict age at first birth.

We conducted similar analyses, replacing childhood SES with childhood home environment (see Figure C1). These analyses yielded similar pattern: Childhood home environment marginally significantly predicted age at first birth (B = .107, SE = .062, p = .085), but this effect disappeared when young adulthood SES was controlled for (B = .068, SE = .061, p = .267, for childhood home environment; B = .276, SE = .064, p < .001, for young adulthood SES).

Figure 3.1

Association between Childhood Socioeconomic Status (SES) and Age at First Birth and Marginal Distributions as a Function of Level of Educational Background



Note. Density plots did not include the categories of junior high school and master's degree/6-year university because of the small sample sizes (Panels A and C), although scatter plot (Panel B) contained them (i.e., five red and four violet markers for categories of junior high school and master's degree/6-year university, respectively).

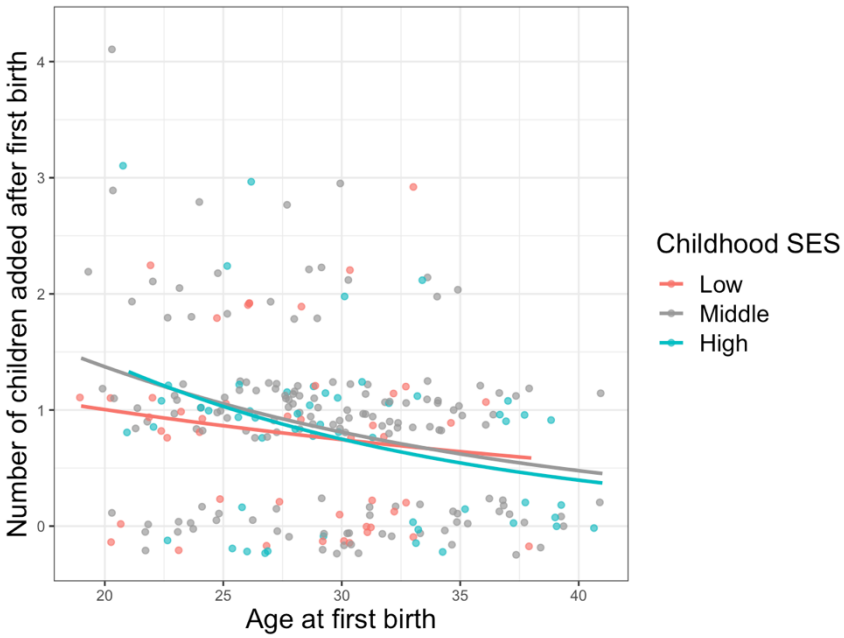
3.3.2. Fitness Consequences of Reproductive Acceleration

To test the fundamental assumption of phenotypic plasticity explanation, we conducted a multiple Poisson regression analysis with childhood SES, age at first birth, and their interaction term as predictor variables and the number of children added after first birth (M = 1.84, SD = 0.75, Mdn = 2) as the outcome variable. Childhood SES and age at first birth were standardized for this analysis prior to computing the interaction term. Results showed that the interaction term was not significant (b = −0.00, SE = 0.04, p = .925, IRR (incidence rate ratio) = 0.99, 95% CI [0.86, 1.14]), although the main effect of age at first birth was significant (b = −0.21, SE = 0.05, p < .001, IRR = 0.77, 95% CI [0.67, 0.89]): Early first birth was associated with a larger number of children irrespective of the level of childhood SES (see Figure 3.2 and Table 3.5), which was consistent with the result of a study on baboons (Weibel et al., 2020).

In addition, to examine the relative weight of the null hypothesis (i.e., absence of interaction) against the alternative hypothesis (i.e., presence of interaction), we conducted Bayes factor analyses (Kass & Raftery, 1995; Rouder et al., 2009). We calculated the Bayes factors using *brms* package ver. 2.17.0 (Bürkner, 2017) in R, with the standard Cauchy prior. The Bayes factor of the model without the interaction term to the full model is 28.07, which indicated strong evidence to favor the no interaction model over the interaction model. In other words, there is little evidence for the adaptive phenotypic plasticity explanation (i.e., accelerated reproductive timing is adaptive only among individuals from low SES families).

Figure 3.2

Association between Age at First Birth and Number of Children Added after First Birth Plotted as a Function of Level of Childhood Socioeconomic Status (SES)



Note. Sample was divided by the mean ± 1 standard deviation of childhood SES. Outcome variable was fitted to Poisson regression model. The adaptive phenotypic plasticity hypothesis predicts that the slope should be negative in the low childhood SES group and positive in the high childhood SES group.

Table 3.5

Effects of Childhood Socioeconomic Status (SES), Age at First Birth, and Childhood SES × Age at First Birth Interaction on Number of Children Added after First Birth

Variable	*b*	*SE*	*p*
DV = Number of children added after first birth			
Childhood SES	0.02	.072	.731
Age at first birth	**−0.26**	.072	<.001
Childhood SES × Age at first birth	−0.01	.070	.914

Note. Dependent variable (i.e., number of children added after first birth) is submitted to Poisson regression analysis. Childhood SES and age at first birth were standardized for this analysis before computing the interaction term. **Boldface** coefficient indicates $p < .05$.

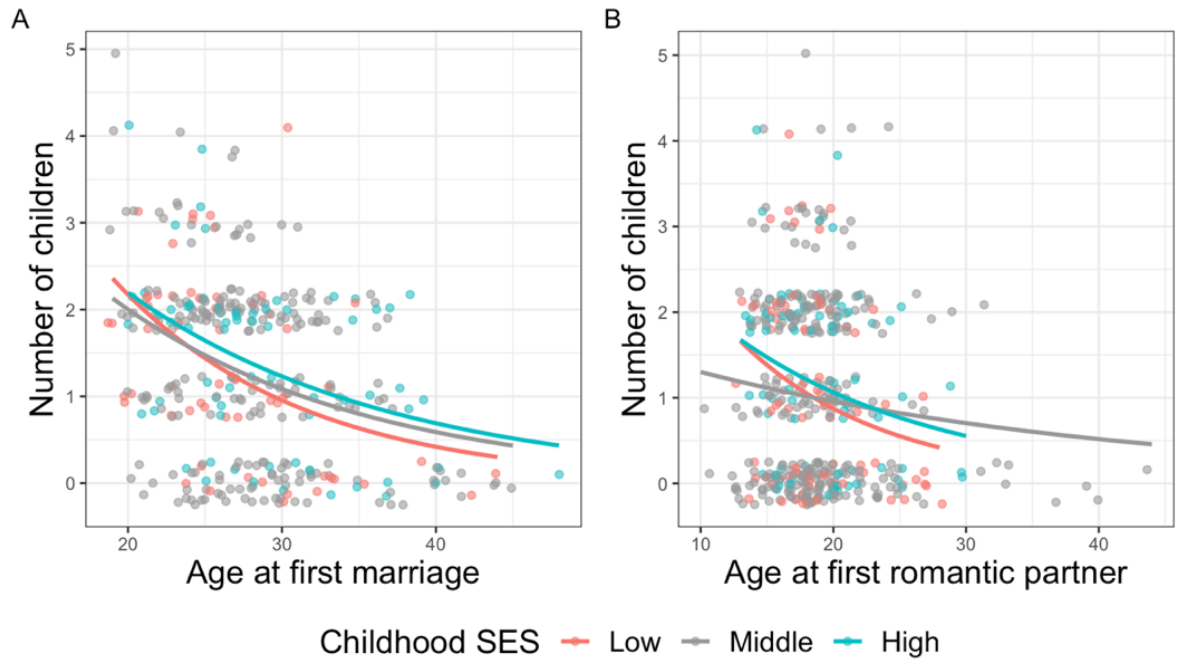
We conducted similar analyses using different operationalizations of reproductive timing: age at first marriage and age at first romantic relationship. Both variables were positively correlated with age at first birth ($r_{246} = .866$, $p < .001$, for age at first marriage; $r_{249} = .253$, $p < .001$, for age at first romantic relationship) but not with childhood SES ($r_{347} = .078$, $p = .148$, for age at first marriage; $r_{446} = -.000$, $p = .994$, for age at first romantic relationship). In addition, they may reflect the timing of reproductive initiation or shift in priority from survival or growth to reproduction. Therefore, these variables are reasonable indices of reproductive acceleration.

Even when age at first marriage or age at first romantic relationship was used as the predictor variable instead of age at first birth, the original results remained largely intact: The interaction effects on the number of children were not significant ($b = 0.04$, $SE = 0.05$, $p = .376$, IRR = 1.05, 95% CI [0.95, 1.15], for age at first marriage; $b = 0.06$, $SE = 0.05$, $p = .280$, IRR = 1.06, 95% CI [0.95, 1.17], for age at first romantic relationship; see Figure 3.3; Tables 3.6 and 3.7). In addition, the Bayes factor analyses yielded similar results for age at first marriage and age at first romantic relationship: The Bayes factors of the models without the interaction terms to the full models are 17.17 for age at first marriage and 13.46 for age at first romantic relationship, respectively, both indicating strong evidence to favor the no interaction model over the interaction model. Therefore, even if we examined the fitness consequences of reproductive timing using other indicators, we found little support for the adaptive phenotypic plasticity explanation.

For robustness checks, we conducted similar analyses, replacing childhood SES with childhood home environment for three variables of reproductive schedule (i.e., age at first birth, age at first marriage, and age at first romantic relationship). These analyses yielded the same results as reported above (see Figures C2 and C3; Tables C1, C2, and C3).

Figure 3.3

Association between (A) Age at First Marriage or (B) Age at First Romantic Relationship and Number of Children Plotted as a Function of Level of Childhood Socioeconomic Status (SES)



Note. Sample was divided by the mean ± 1 standard deviation of childhood SES. The number of children was fitted to Poisson regression model. The adaptive phenotypic plasticity hypothesis predicts that the slopes should be negative for low childhood SES group and positive for high childhood SES group.

Table 3.6

Effects of Childhood SES, Age at First Marriage, and Childhood Socioeconomic Status (SES) × Age at First Marriage Interaction on Number of Children

Variable	*b*	*SE*	*p*
DV = Number of children			
Childhood SES	0.06	0.05	.252
Age at first marriage	**−0.34**	0.05	<.001
Childhood SES × Age at first marriage	0.04	0.05	.376

Note. Dependent variable (i.e., number of children) was submitted to Poisson regression analysis. Childhood SES and age at first marriage were standardized for this analysis before computing the interaction term. **Boldface** coefficient indicates $p < .05$.

Table 3.7

Effects of Childhood Socioeconomic Status (SES), Age at First Romantic Relationship, and Childhood SES × Age at First Romantic Relationship Interaction on Number of Children

Variable	*b*	*SE*	*p*
DV = Number of children			
Childhood SES	0.05	0.05	.312
Age at first romantic relationship	**−0.19**	0.05	<.001
Childhood SES × Age at first romantic relationship	0.06	0.05	.280

Note. Dependent variable (i.e., number of children) was submitted to Poisson regression analysis. Childhood SES and age at first romantic relationship were standardized for this analysis before computing the interaction term. **Boldface** coefficient indicates $p < .05$.

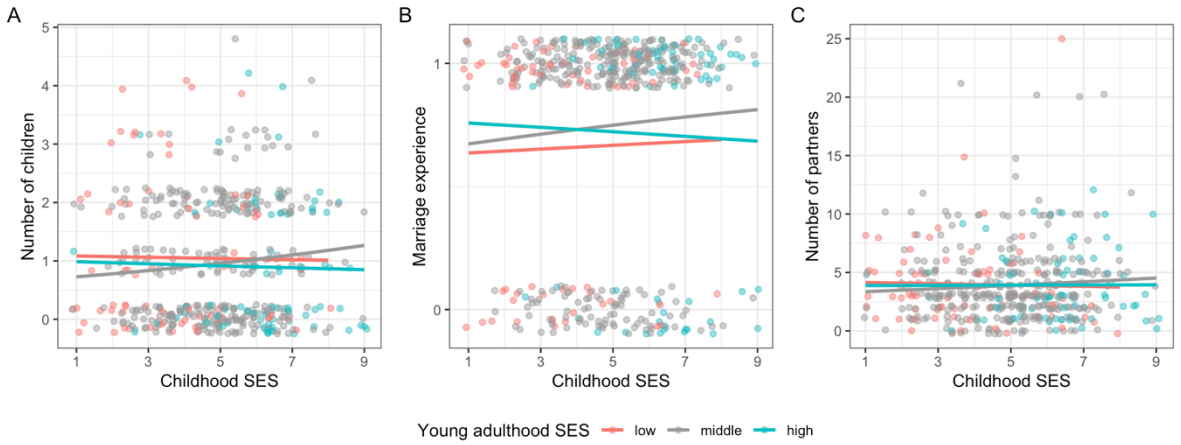
3.3.3. Fitness Consequences of Environmental Consistency

Childhood SES × Young Adulthood SES Interaction. Regarding the prediction of ePAR, we conducted multiple regression analyses, which examined the interaction of childhood SES and young adulthood SES on each proxy fitness index (i.e., number of children, marriage experience, number of partners). Both SES scores were standardized for these analyses before computing the interaction term. In the three analyses, no significant interactions between childhood SES and young adulthood SES were found (b = −0.01, SE = 0.04, p = .872, for number of children; b = −0.11, SE = 0.09, p = .221, for marriage experience; b = −0.00, SE = 0.02, p = .930, for number of partners; Figure 3.4; Table 3.8). Inspections of variance inflation factors (VIFs) indicate that multicollinearity was not an issue in the three models (all VIFs < 1.32).

For robustness checks, we conducted similar analyses, replacing childhood SES with childhood home environment (Figure C4; Table C4). The results slightly differed from the original results. First, the significant interaction between childhood home environment and young adulthood SES on marriage experience (b = −0.21, SE = 0.10, p = .040, OR (odds ratio) = 0.81, 95% CI [0.67, 0.99]). However, the observed pattern of the interaction was opposite to the hypothesized interaction: In the high young adulthood SES (1 SD above the mean) group, the coefficient of childhood home environment term was negative (b = −0.12, SE = 0.26, p = .634); in the low young adulthood SES (1 SD below the mean) group, it was positive (b = 0.31, SE = 0.26, p = .227). Second, we found a significant positive main effect of childhood home environment on the number of partners (b = 0.11, SE = 0.02, p < .001, IRR = 1.12, 95% CI [1.07, 4.07]). Individuals raised in relatively affluent family environments had the tendency to have more romantic partners later in life.

Figure 3.4

Association between Childhood Socioeconomic Status (SES) and Fitness Indices Plotted as a Function of Level of Young Adulthood SES



Note. The horizontal axis of all panels indicates childhood SES. The vertical axis of each panel indicates one of the three fitness indices: (A) number of children, (B) marriage experience (0 = never married vs. 1 = have married at least once), and (C) number of partners. The number of children and number of partners were fitted to Poisson regression model. Marriage experience was fitted to logistic regression model. The sample was divided by the mean ± 1 standard deviation of young adulthood SES. External predictive adaptive response hypothesis predicts that the slopes should be negative for low young adulthood SES group but should be positive for high young adulthood SES group.

Table 3.8

Regression Coefficients of Childhood Socioeconomic Status (SES), Young Adulthood SES, and Childhood SES × Young Adulthood SES Interaction on Fitness Indices

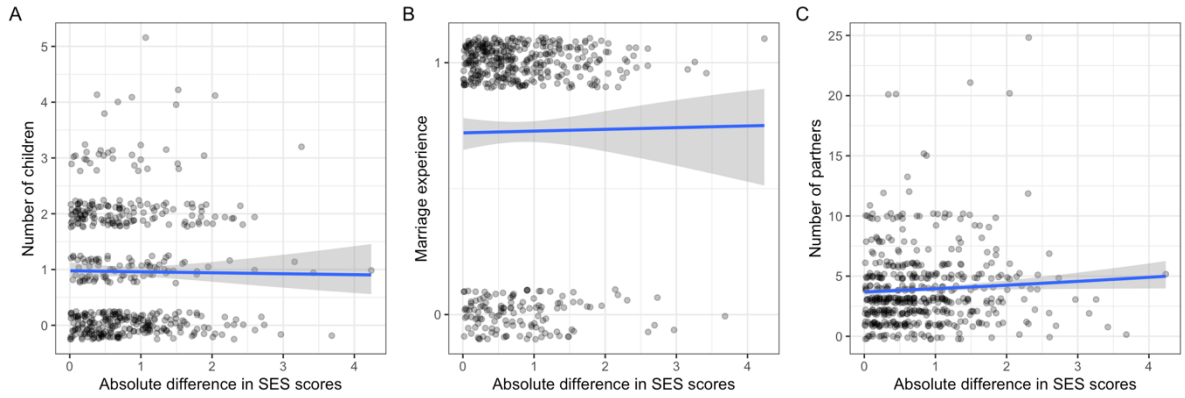
Variable	*b*	*SE*	*p*
DV = Number of children			
Childhood SES	0.07	0.05	.210
Young adulthood SES	−0.06	0.05	.218
Childhood SES × Young adulthood SES	−0.01	0.04	.872
DV = Marriage experience			
Childhood SES	0.12	0.12	.304
Young adulthood SES	−0.04	0.12	.728
Childhood SES × Young adulthood SES	−0.11	0.09	.221
DV = Number of partners			
Childhood SES	0.05	0.03	.078
Young adulthood SES	−0.04	0.03	.141
Childhood SES × Young adulthood SES	−0.00	0.02	.930

Note. The number of children and number of partners were submitted to Poisson regression analyses. Marriage experience (0 = never married vs. 1 = having married at least once) was submitted to logistic regression analysis. Childhood SES and young adulthood SES were standardized for the three analyses before computing the interaction term.

Absolute Difference between Childhood SES and Young Adulthood SES. In addition, we directly examined the effect of environmental consistency on the same three fitness indices (Figure 3.5). We conducted three regression analyses with the predictor variable of absolute difference between the standard scores of childhood SES and young adulthood SES. This variable takes 0 for no change and larger values for environmental inconsistency. None of the correlations between this variable and any of the three fitness indices were significant ($r_{478} = -.011$, $p = .808$, for number of children; $r_{478} = .011$, $p = .818$, for marriage experience; $r_{477} = .060$, $p = .192$, for number of partners). In the regression analyses, this variable had no significant effects on the number of children ($b = -0.01$, $SE = 0.05$, $p = .791$) and marriage experience ($b = 0.02$, $SE = 0.10$, $p = .817$) but had a significant effect on the number of partners, which was opposite to the hypothesized direction ($b = 0.05$, $SE = 0.02$, $p = .031$, IRR = 1.05, 95% CI [1.00, 1.10]).

Figure 3.5

Association between Absolute Difference in Socioeconomic status (SES) Scores and Fitness Indices



Note. The horizontal axis of all panels indicates absolute difference between standardized scores of childhood SES and young adulthood SES. The vertical axis of each panel indicates one of the three fitness indices: (A) number of children, (B) marriage experience (0 = never married vs. 1 = have married at least once), and (C) number of partners. External predictive adaptive response hypothesis predicted negative slope of absolute difference in SES on fitness.

3.3.4. Somatic Consequences of Early-Life Adversity

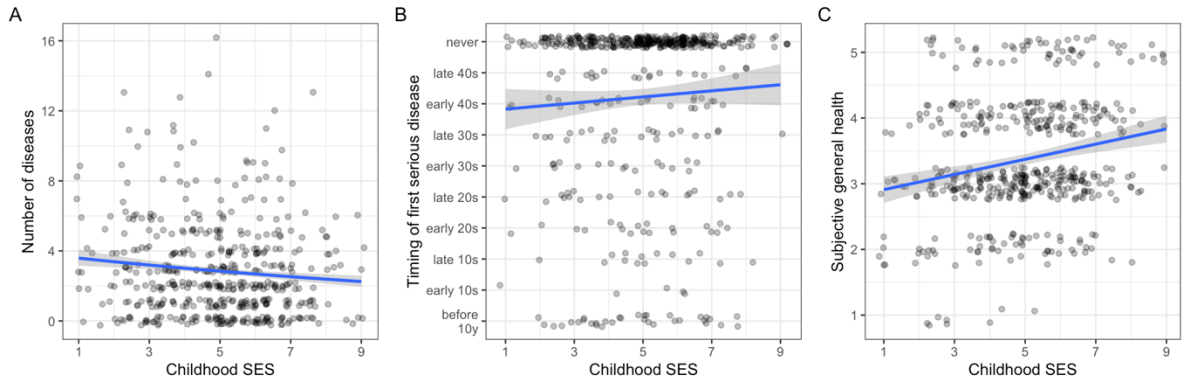
Regarding the assumption of iPAR, we examined the association between childhood SES and health indicators. All three dependent variables (i.e., health indicators) showed significant and moderate correlations with each other ($r_{478} = -.379$, $p < .001$, for number of experienced diseases and timing of first serious disease; $r_{478} = -.422$, $p < .001$, for number of experienced diseases and subjective general health; $r_{478} = .198$, $p < .001$, for timing of first serious disease and subjective general health; Table 3.3).

In bivariate analyses, childhood SES was significantly associated with the number of diseases ($r_{478} = -.104$, $p = .022$) and subjective general health ($r_{478} = .215$, $p < .001$) but not with timing of first serious disease ($r_{478} = .058$, $p = .202$): Those from low SES families had experienced more diseases and perceived poorer health in their late 40s (see Figure 3.6; Table 3.3). We conducted multiple regression analyses, which examined whether low childhood SES was associated with poor health status after controlling for young adulthood SES, lifestyles (i.e., drinking, smoking, exercise), and the number of children (Table 3.9). Results revealed that childhood SES was significantly and negatively associated with subjective general health ($B = .098$, $SE = .049$, $p = .045$). However, it was not significantly associated with objective health indices: number of diseases ($b = -0.03$, $SE = 0.03$, $p = .311$, IRR = 0.97, 95% CI [0.91, 1.03]) and timing of first serious diseases ($B = .050$, $SE = .051$, $p = .355$).

For robustness checks, we conducted similar analyses, replacing childhood SES with childhood home environment (see Figure C5 and Table C5). The multiple regression analyses showed that, even after potential confounding variables were controlled for, childhood home environment had a significant effect on the number of diseases ($b = -0.07$, $SE = 0.03$, $p = .020$, IRR = 0.93, 95% CI [0.89, 0.99]) and subjective general health ($B = .094$, $SE = .045$, $p = .037$) but not on the timing of first serious diseases ($B = .036$, $SE = .047$, $p = .453$).

Figure 3.6

Association between Childhood Socioeconomic Status (SES) and Health Indicators



Note. In each panel, horizontal axis indicates childhood SES, and vertical axis indicates one of the three health related variables: (A) number of diseases, (B) timing of first serious disease; and (C) subjective general health (1 = very poor, 2 = poor, 3 = fair, 4 = good, 5 = very good). The number of diseases was fitted to Poisson regression model. Internal predictive adaptive response hypothesis assumed that higher childhood SES is associated with better health status (i.e., negative slopes for Panels A and C, and negative slope for Panel B).

Table 3.9

Results of Multiple Regression Analyses for Health Variables

Variable	Estimate	*SE*	*p*
DV = Number of diseases			
Childhood SES	−.030	.030	.311
Young adulthood SES	**−.132**	.030	<.001
Drinking	**.078**	.028	.005
Smoking	.035	.027	.200
Exercise	−.020	.028	.477
Number of children	−.050	.027	.074
DV = Timing of first serious disease			
Childhood SES	.050	.051	.355
Young adulthood SES	.038	.052	.464
Drinking	−.000	.048	.999
Smoking	.040	.048	.401
Exercise	−.050	.047	.287
Number of children	.050	.046	.275
DV = Subjective general health			
Childhood SES	**.097**	.049	.045
Young adulthood SES	**.225**	.049	<.001
Drinking	−.030	.046	.513
Smoking	−.044	.046	.338
Exercise	.083	.044	.062
Number of children	**.098**	.044	.026

Note. All independent variables were standardized for analyses. The number of diseases was submitted to Poisson regression analysis. For the remaining dependent variables (i.e., timing of first serious disease and subjective general health), standardized coefficients are shown. **Boldface** coefficients indicate $p < .05$.

3.3.5. Byproduct of Educational Involvement

Mediation by Education Level. Although childhood SES did not significantly predict age at first birth as mentioned above, we conducted a mediation analysis, which assumed childhood SES as the predictor, education level as the mediator, and age at first birth as the outcome (Figure 3.7A). Childhood SES was significantly and positively associated with education level (B = .227, SE = .054, p < .001). The direct effect of childhood SES on age at first birth was not significant (B = .045, SE = .062, p = .470); however, education level was significantly and positively associated with age at first birth (B = .318, SE = .070, p < .001). We computed 95% confidence interval of indirect effect using a bias-corrected bootstrapping method (5,000 bootstrap samples), using the mediation function of *MBESS* package in R, and detected a significant indirect effect (B = .072, 95% CI [.031, .126]). These results did not differ when education levels of junior high school (N = 5 for those with at least one child) and master's degree/6-year university (N = 4 for those with at least one child) were excluded.

For robustness check, we conducted similar analyses, replacing childhood SES with childhood home environment and revealed the same results (see Figure C6): The total effect of childhood home environment on age at first birth did not reach statistical significance (B = .107, SE = .062, p = .085); the indirect effect via education level was significant (B = .067, 95% CI [.030, .122]); and the direct effect of childhood home environment on age at first birth was not significant (B = .040, SE = .061, p = .515).

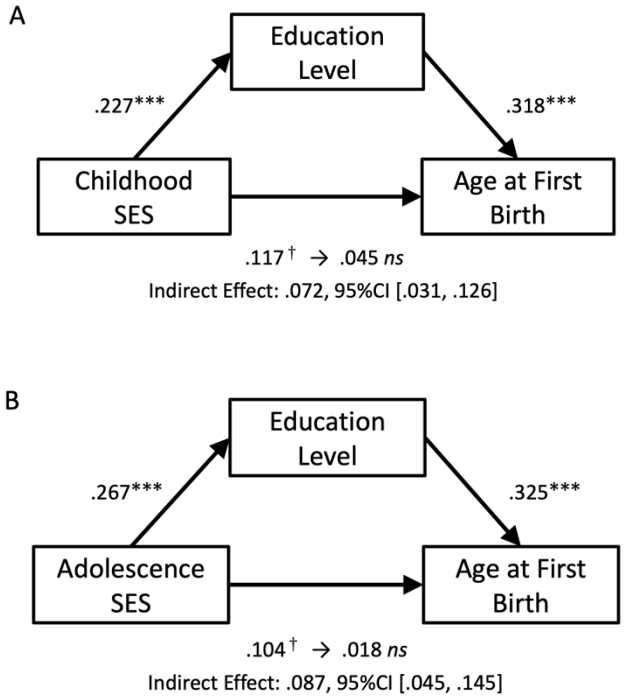
Comparing Adolescence SES with Childhood SES. We conducted a similar mediation analysis, replacing the predictor variable with adolescence SES (Figure 3.7B). Adolescence SES was expected to directly influence on the enrollment in higher education. The results were the same as that of the childhood SES: The total effect of adolescence SES

on age at first birth did not reach statistical significance (B = .104, SE = .006, p = .099); and the indirect effect of adolescence SES on age at first birth through education level was significant (B = .087, 95% CI [.045, .147]).

To examine whether adolescence SES rather than childhood SES is a better predictor of outcome variables, we compared the adolescence SES × outcome variable correlations with the childhood SES × outcome variable correlations, where education level or age at first birth was the outcome variable. The correlation coefficient between adolescence SES and education level (r_{478} = .288, p < .001) was significantly higher than that between childhood SES and education level (r_{478} = .244, p < .001) (t(248) = −2.33, p = .020, for test of difference between two dependent correlations). Thus, adolescence SES was a better predictor of education level than childhood SES. However, both the childhood SES × age at first birth correlation (r_{249} = .118, p = .006) and adolescence SES × age at first birth correlation (r_{249} = .104, p = .099) were only marginally significant and did not significantly differ from each other (t(248) = 0.50, p = .615, for test of difference between two dependent correlations).

Figure 3.7

Results of the Mediation Analyses Examining the Indirect Effect of (A) Childhood SES or (B) Adolescence SES on Age at First Birth via Education Level



† $p < .10$. *** $p < .001$.

Fitness Consequences of Prolonged Education. In addition to the preregistered analyses reported above, we conducted an exploratory analysis to examine whether education level moderates the association between childhood SES and fitness indices. In the following analyses, we operationalized life history strategies as education level and examined whether there was an interaction between childhood SES and life history strategy on fitness indices. We conducted a series of multiple regression analyses involving the interaction term of childhood SES and education level (both variables were standardized for these analyses prior to computing the interaction term), in each of the three proxy fitness indices (i.e., number of children, marriage experience, and number of romantic partners) and current annual household income (Figure 3.8; Table 3.10). The number of children and the number of partners were subjected to Poisson regressions, and marriage experience was subjected to logistic regression. Inspections of VIFs indicate that multicollinearity was not an issue in any of these four models (all VIFs < 1.10).

Education level showed negative associations with the number of children (b = −0.10, SE = 0.05, p = .211, IRR = 0.90, 95% CI [0.82, 0.99]), marriage experience (b = −0.24, SE = 0.11, p = .029, OR = 0.79, 95% CI [0.64, 0.97]), and number of partners (b = −0.09, SE = 0.02, p < .001, IRR = 0.91, 95% CI [0.87, 0.95]) and a positive association with current annual household income (B = −.372, SE = .063, p < .001). In the analyses of the former three variables, although there were slight patterns in which prolonged education was associated with lower fitness, particularly among those from low SES families, the interactions between childhood SES and education level were not significant (b = −0.01, SE = 0.05, p = .774, for number of children; b = −0.06, SE = 0.10, p = .533, for marriage experience; b = 0.03, SE = 0.02, p = .224, for number of partners). However, the analysis of the current annual household income revealed a significant interaction effect (B = .128, SE

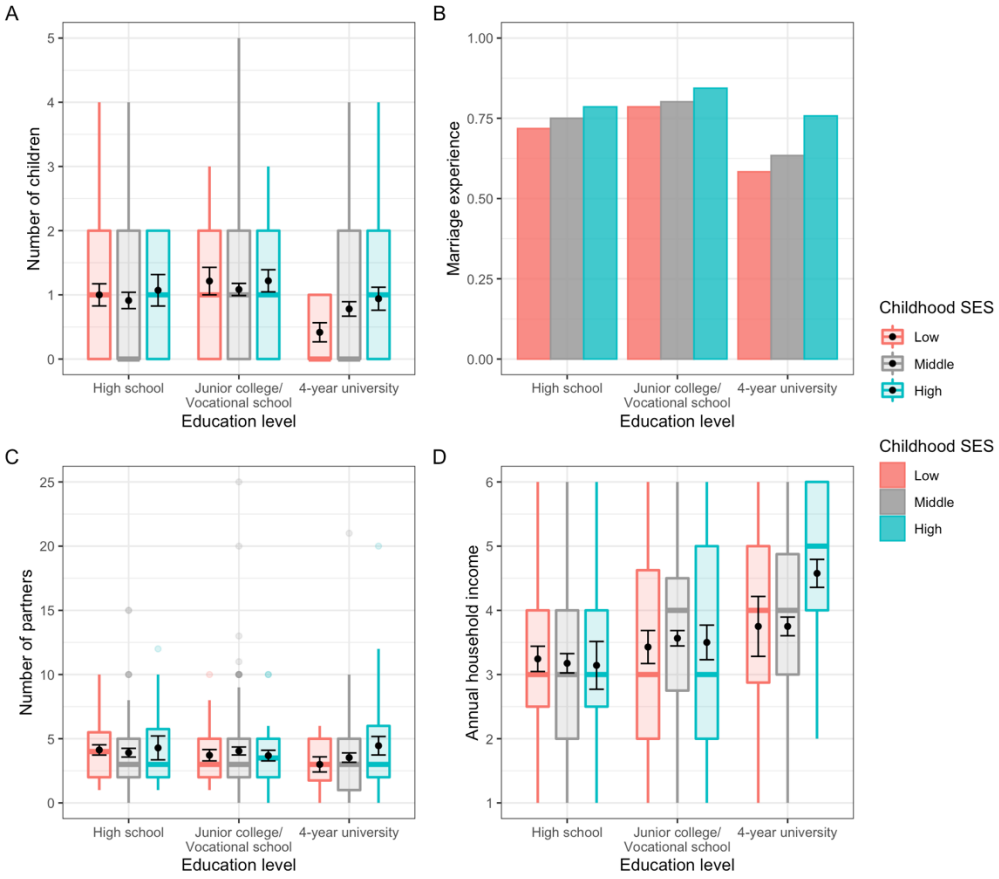
= .059, p = .030): The positive effect of a slower strategy (operationalized by prolonged education) on the current household income was greater among those from high SES families than among those from low SES families (B = .469, SE= .095, p < .001, for high childhood SES [1 *SD* above the mean] group; B = .170, SE = .110, p = .124, for low childhood SES [1 *SD* below the mean] group).

For robustness checks, we conducted similar analyses, replacing childhood SES with childhood home environment (see Figure C7 and Table C6). The similar significant interaction between childhood home environment and education level on the current household income was observed (B = .195, SE = .060, p = .001, for interaction term; B = .630, SE = .121, p < .001, for the high childhood home environment group; B = .200, SE = .131, p = .130, for the low childhood home environment group). In addition, analyses revealed a significant interaction on the number of partners (b = 0.05, SE = 0.02, p = .019, IRR = 1.05, 95% CI = [1.01, 1.10]): The negative effect of the slower strategy on the number of partners was greater among those from disadvantaged families than among those from affluent families (b = −0.04, SE = 0.04, p = .370, for the high childhood home environment group; b = −0.30, SE = 0.06, p < .001, for the low childhood home environment group).

In summary, if we assume that investment in education reflects a slower strategy, a slower strategy was generally associated with the costs in reproductive and mating success, although it was associated with the benefit in household income. The benefit in household income caused by prolonged education was boosted among those from favorable environments. However, there was little evidence that the cost of prolonged education in terms of reproductive and mating success was moderated by childhood environment, although the cost in the number of partners may be boosted among those from deprived environments.

Figure 3.8

Associations between Life History Strategy (Operationalized by Education Level) and Fitness Indices by the Level of Childhood Socioeconomic Status (SES)



Note. The horizontal axis of all panels indicates education level. The vertical axis of each panel indicates one of the four outcome variables: (A) number of children, (B) marriage experience (proportion of those who married at least once), (C) number of partners; and (D) current annual household income. The sample was divided by the mean ± 1 standard deviation of childhood SES. In all panels, boxplots and bar charts for junior high school, master's degree/6-year university, and doctoral degree were omitted because of small sample sizes (*N*s = 13, 14, and 2 for junior high school, master's degree/6-year university, and doctoral degree, respectively). In Panels A, B, and D, black dots indicate mean values, and error bars indicate standard errors.

Table 3.10

Results of a Series of Multiple Regression Analyses Examining the Interaction Effect between Childhood SES and Life History Strategy Operationalized by Education Level on Fitness Indices

Variable	Estimate	*SE*	*p*
DV = Number of children			
Childhood SES	.061	.048	.211
Education level	**−.103**	.049	.034
Childhood SES × Education level	−.013	.046	.774
DV = Marriage experience			
Childhood SES	.171	.108	.112
Education level	**−.235**	.108	.029
Childhood SES × Education level	−.061	.098	.533
DV = Number of partners			
Childhood SES	**−.055**	.024	.023
Education level	**−.094**	.024	<.001
Childhood SES × Education level	.027	.022	.224
DV = Current annual household income			
Childhood SES	**.118**	.045	.008
Education level	**.262**	.045	<.001
Childhood SES × Education level	**.090**	.041	.030

Note. Childhood SES and education level were standardized for each analysis prior to computing the interaction term. The number of children and number of partners were submitted to Poisson regression analyses. Marriage experience (0 = *never married* vs. 1 = *having married at least once*) was subjected to logistic regression. Standardized coefficients are displayed for the current annual household income. **Boldface** coefficients indicate $p < .05$.

3.4. Discussion

In life history theory in psychology, the association between childhood environmental adversity and accelerated reproductive timing has been explained as an adaptive phenotypic plasticity (Del Giudice et al., 2015; Ellis et al., 2009; Nettle, 2011). A critical assumption of this explanation is that accelerated reproductive timing increases fitness only among those who experienced childhood adversity. In this study, to test the validity of this explanation, we examined the lifetime reproductive consequence of earlier reproductive initiation by the level of childhood SES.

First, the positive correlation between childhood SES and age at first birth was not statistically significant, although such a trend was observed (r_{249} = .118, p = .061, see Figure 3.1 and Table 3.2). Thus, the findings of the previous studies (e.g., Nettle et al., 2011) and reanalyses of Study 1 data were not clearly replicated. In this study, young adulthood SES rather than childhood SES was positively associated with age at first birth, which suggested that reproductive decisions may be influenced by the immediate situation rather than by the early-life environment. More importantly, the fitness trade-offs required for the phenotypic plasticity to evolve were not observed. Specifically, earlier reproductive initiation was associated with a greater number of children at late 40s, regardless of the level of childhood SES (Figure 3.2 and Table 3.5), which was consistent with a study on baboons (Weibel et al., 2020). Moreover, when the age at first birth was replaced by other variables related to reproductive schedule (i.e., age at first marriage, age at first romantic relationship), the result remained unchanged (Figure 3.3; Tables 3.6 and 3.7). These results suggest that there is no reason to assume that early reproductive initiation confers fitness benefits exclusively to individuals who experienced adversity during childhood. Therefore, the analyses revealed no support for the adaptive phenotypic plasticity explanation, which was the fundamental idea of

life history theory in psychology.

Moreover, this study included the tests of the prediction or assumption of ePAR and iPAR models (Nettle & Bateson, 2015; Nettle et al., 2013). According to ePAR, it is predicted that the environmental consistency between early-life and after-maturity is associated with higher fitness. However, analyses revealed no associations between the consistency in SES and fitness indices (Figures 3.4 and 3.5). Thus, there was little evidence for the prediction of the ePAR model. In contrast, iPAR assumes that early-life adversity has primary detrimental influences on individual somatic condition in later life. After controlling for potential confounding variables, childhood SES was significantly associated with subjective general health but not with other health-related variables (Table 3.9). However, childhood home environment was associated with the number of diseases and subjective general health. Thus, the results were generally consistent with the assumption of the iPAR model.

The positive association between childhood SES and age at first birth did not reach statistical significance; however, education level was found to significantly mediate them (Figure 3.7A). Therefore, the association between them, if any, is accounted for by prolonged education, which is consistent with the byproduct explanation. Given that education level or period of education is sometimes used as part of a slower strategy in life history theory in psychology (Bulled & Sosis, 2010; Richardson et al., 2020), critics may argue that prolonged education per se is a manifestation of a slower strategy. In this study, education level had a positive association with household income during late 40s and negative associations with reproductive and mating success in general. Exploratory analyses revealed that the benefit of prolonged education in later household income was greater among individuals from high SES families than among individuals from low SES families, which is consistent with hypothesis of life history theory in psychology. However, such interaction was not detected for the

number of children, a direct measure of reproductive success. Whether there is an fitness advantage to delaying reproductive initiation in affluent environments needs to be examined in more detail in future studies.

This study has several potential limitations. The childhood SES measure used in this study solely relied on participants' recalls. As previously noted, this study failed to clearly replicate the association between early-life environmental adversity and age at first birth, which had been reported in many previous studies (including prospective longitudinal survey) in countries with high-income economy. In this study, some memory biases might have blurred the association between the age at first birth and childhood adversity. For example, memories of childhood adversity may be positively distorted among individuals whose socioeconomic status improved over time. However, childhood SES measure showed a modest association with more objective assessments of participants' childhood home environments. Comparable analyses using the childhood home environment score in lieu of childhood SES score yielded mostly similar results.

Another limitation is the target population. It is possible that households with low SES in the late 20th century in Japan, in which participants spent their childhood, remain a favorable side of the ancestral environment. If this is the case, the weak association between childhood adversity and age at first birth is due to the insufficient severity of their childhood condition. Therefore, to comprehensively assess the assumption of the phenotypic plasticity explanation, it is useful to expand the childhood adversity measures and research populations. However, if this interpretation of childhood environments is valid, the opposite pattern should be observed rather than the one observed in this study. As life history theory in psychology predicts that delayed reproductive timing for individuals raised in favorable environments, a negative correlation between the age at first birth and reproductive success (the later, the

more successful) should have been observed.

This study focused on females in Japan, which is an industrialized high-income country. The environment of such society differs from that in which humans evolved. Thus, it could be criticized that such a mismatch prevented the observation of the hypothesized pattern. However, it is noteworthy that most studies that supported the reproductive acceleration hypothesis have been conducted in developed countries, and data from pre-industrialized populations have provided mixed findings. In particular, some studies have shown that earlier first birth is associated with individuals who experienced relatively favorable conditions rather than adverse conditions in focal populations (Allal et al., 2004; Lahdenperä et al., 2007). Given these contradictory findings, in addition to the results of this study, the byproduct explanation is more plausible than the adaptive phenotypic plasticity explanation.

4. General Discussion

4.1. Summary and Interpretations

The present research aimed to critically examine the validity of the research trends in psychology purportedly grounded in life history theory, which has its origin in evolutionary biology. It has been pointed out that the term "life history theory" refers to different ideas in evolutionary biology and psychology (Nettle & Frankenhuis, 2020). More importantly, life history theory in psychology has been accepted without critical examination of its underlying assumptions. Possibly due to its intuitive appeal, given an observed association between the local environmental condition and individual behavioral tendencies, the adaptive phenotypic plasticity explanation tends not to be pitted against rival explanations (e.g., spurious correlation due to a third variable that has nothing to do with adaptation). Therefore, psychological life history theory needs to be critically examined.

According to life history theory in psychology, people developmentally employ different adaptive reproductive strategies (life history strategies) in response to their childhood environmental conditions (Ellis et al., 2009). Specifically, childhood adversity serves as cues to predict higher morbidity/mortality after maturity, thereby inducing fast strategies characterized by accelerated reproductive schedules and associated psychological mechanisms to achieve some reproduction in shortened healthy lifespans. According to this framework, many studies have reported that childhood environmental adversity is associated with earlier reproductive timing (e.g., Nettle et al., 2011) and higher impulsivity (e.g., Griskevicius et al., 2013), which have been regarded as positive evidence for adaptive

phenotypic plasticity.

However, the adaptive phenotypic plasticity explanation presumes that the fitness consequences of alternative phenotypes hinge on environmental conditions. In particular, characteristics of fast strategies must be associated with higher fitness among those who experienced childhood adversity and with lower fitness among those who did not. Nevertheless, little effort has been made to verify the validity of this assumption in life history theory in psychology. Therefore, in the present research, we tested the validity of this assumption by focusing on two traits typically considered a part of fast life history strategies: impulsivity and reproductive timing.

In Study 1, we focused on impulsivity. According to life history theory in psychology, impulsivity is an adaptive response to childhood environmental adversity, and those who experienced childhood adversity tend to be more impulsive (Griskevicius et al., 2013; Kruger et al., 2008). To test the evolvability of such phenotypic plasticity, we measured the participants' childhood socioeconomic status (SES) and impulsivity as well as their fitness using multiple proxy indices (i.e., number of children, marriage experience, annual household income, subjective socioeconomic status, life satisfaction). Impulsivity was operationalized in three ways (i.e., temporal discounting, risk taking, and Mini-K score). None of the results of Study 1 supported the evolvability of impulsivity phenotypic plasticity.

In Study 2, we focused on the reproductive timing among females. According to life history theory in psychology, earlier reproductive timing is an adaptive response to environmental adversity, and those who experienced childhood adversity tend to accelerate reproductive timing (e.g., Ellis et al., 2009). To test whether the acceleration indeed reflects adaptation to environmental condition, we measured childhood SES, age at first birth, and the lifetime number of children in female participants in their late 40s. Study 2 did not support

the idea that accelerated reproductive timing is an adaptive response to early-life adversity. The interaction between childhood SES and age at first birth on the number of children was not observed. Contrary to the hypothesis, early reproductive initiation was associated with higher reproductive success regardless of the level of childhood SES. Moreover, the results suggest that education level significantly mediated the association between early-life adversity and age at first birth. Therefore, a byproduct explanation is more plausible: The association between childhood family environmental condition and reproductive timing, if any, may be a byproduct of prolonged education among daughters from economically affluent families.

In summary, neither Studies 1 nor 2 supported the notion that fitness consequences of alternative phenotypes are conditioned by childhood adversity. These results cast serious doubt on life history theory in psychology, at least with regard to impulsivity and reproductive timing.

4.2. Limitations and Remained Issues

The results of the present research provided negative findings against the phenotypic plasticity explanation, at least with respect to the adjustment of impulsivity and timing of reproductive initiation in response to economic adversity. However, various other childhood environmental variables (predictor variables) and life history strategy variables (outcome variables) have been used in the literature on life history theory in psychology. For example, in previous studies in the field, unpredictability (e.g., parental transitions, household moves) and threat (e.g., family/neighborhood violence) have often been used as childhood environmental variables (Ellis et al., 2009; Ellis et al., 2022; McLaughlin et al., 2014), also age at menarche and parenting behavior as life history strategy variables (see Ellis & Del Giudice, 2019, for a review). Therefore, further research should include those variables and

critically examine human phenotypic plasticity more broadly.

However, a serious issue with life history theory in psychology is that arguments about the relevance of those variables being treated in the context of focal phenotypic plasticity are inadequate. In other words, it is unclear what cues those variables can provide to individuals and how psychological and behavioral tendencies of interest can be adaptive in response to them. Thus, researchers should avoid merely examining an association between childhood environment and later behavioral tendencies by assuming adaptive plasticity; rather, from a critical perspective, they should include a verification of the explanatory assumptions and predictions (as in the present research).

In the present research, the associations between childhood environmental adversity and putative life history strategies reported in previous studies (e.g., Griskevicius et al., 2013; Nettle et al., 2011) were not replicated—or at least not clearly. In Study 1, childhood SES was significantly correlated with Mini-K score but not with temporal discounting and risk taking. In Study 2, the association between childhood SES and age at first birth among females did not reach statistical significance. These replication failures can reflect the absence of the phenotypic plasticity. If so, this is consistent with the fact that no evidence of the adaptiveness of phenotypic plasticity emerged in Studies 1 and 2. However, another possibility cannot be completely excluded, namely that the childhood environmental variables in the present research did not capture the existing associations. If this was the case, the tests of adaptiveness of phenotypic plasticity may also be inadequate. Therefore, some caution regarding this possibility is warranted, and studies of similar designs with different variables need to be conducted in a wider range of populations.

4.3. Conclusion

Life history theory has become increasingly influential in psychology as a heuristic

framework that explains individual differences as adaptive phenotypic plasticity in response to local environments. However, its approach must be critically examined. In the present research, through two studies focusing on different traits, impulsivity in Study 1 and the timing of reproductive initiation in Study 2, we critically tested the adaptiveness of purported phenotypic plasticity. As a result, both studies did not support the idea that the fitness consequences of alternative phenotypes are conditioned by the environment.

Life history theory in psychology is now increasingly extending its explanatory scope to a wider range of behavioral individual differences and personality traits, such as prosociality and cooperation (e.g., Wu et al., 2017), "dark triad" traits (e.g., Jonason et al., 2017; McDonald et al., 2012), and mental and behavioral disorders (e.g., Hurst & Kavanagh, 2017), which do not have apparent relationships with reproductive strategies and adaptation. In these studies, the heuristic framework of life history strategy has been applied quite intuitively.

Analyses based on evolutionary theory assume that traits commonly observed in a species or population are adaptations to the surrounding ecological conditions. However, it must be verified that the observed patterns are indeed adaptations. In evolutionary biology, studies based on observational data are examined in reference to formal models of the phenomenon of interest. Fashionable applications of evolutionary theory in psychology do not incorporate such methodological practices but only the perspective of viewing observed patterns as adaptation. To correct such misapplications, theoretical studies must be coupled with empirical studies.

Acknowledgements

I would like to express my sincere gratitude to Dr. Yohsuke Ohtsubo, my former supervisor, for his generous support throughout this research. He has provided me with thoughtful guidance and encouragement over the years. I am also deeply grateful to Dr. Kuniaki Yanagisawa, my current supervisor, for his extensive support and advice. I am very indebted to Dr. Shinichi Kita, Dr. Yasuki Noguchi, and Dr. Takuya Niikawa for their insightful comments and suggestions on this dissertation.

I would like to extend my gratitude to Dr. Yuta Kawamura, Ms. Xin Jin, Ms. Fubei Lyu, Mr. Kotaro Shinoki, and Mr. Ryotaro Sakamoto for their valuable advice and help. I have had support and encouragement from all my former and current colleagues: Dr. Naoki Konishi, Dr. Mana (Yamaguchi) Ueda, Ms. Li Yueying, Ms. Erika Nishi, Ms. Honoka Shimahara, Ms. Kaede Tamura, and Ms. Yaxin Wang.

Last but not least, I would like to thank all my family and friends for their constant support.

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Appendix

Appendix A

Table A1

Childhood Socioeconomic Status Scale (Griskevicius et al., 2013; Translated by Authors into Japanese)

Item	
1.	My family usually had enough money for things when I was growing up （子どもの頃、私の家には生活に必要なお金があった）
2.	I grew up in a relatively wealthy neighborhood （子どもの頃、私は比較的裕福な家庭の多い地域で育った）
3.	I felt relatively wealthy compared to the other kids in my school （子どもの頃、私は、同じ学校に通う子ども達と比べて、裕福であると感じていた）

Table A2

Adolescence Socioeconomic Status Scale (Modified from Childhood Socioeconomic Status Scale; Griskevicius et al., 2013)

	Item
1.	My family usually had enough money for things when I was a high school student （高校生の頃、私の家には生活に必要なお金があった）
2.	I grew up in a relatively wealthy neighborhood when I was a high school student （高校生の頃、私は比較的裕福な家庭の多い地域で育った）
3.	I felt relatively wealthy compared to the other kids in my school when I was a high school student （高校生の頃、私は、同じ学校に通う子ども達と比べて、裕福であると感じていた）

Table A3

Young Adulthood Socioeconomic Status Scale (Modified from Current Socioeconomic Status Scale; Griskevicius et al., 2013)

	Item
1.	I had enough money to buy things I want in my 20s （20 代の頃、私には自分が欲しいものを買うのに十分なお金があった）
2.	I don’t need to worry too much about paying my bills in my 20s （20 代の頃、私はお金の支払いについてあまり心配しすぎる必要がなかった）
3.	I had enough money to buy things I want in my 20s （30 代の頃、私には自分が欲しいものを買うのに十分なお金があった）
4.	I don’t need to worry too much about paying my bills in my 20s （30 代の頃、私はお金の支払いについてあまり心配しすぎる必要がなかった）

Table A4

Temporal Discounting Task (Adapted from Griskevicius et al., 2013)

JPY to be received tomorrow	JPY to be received after 33 days
6400	6500
6900	7100
8600	9200
4500	4900
4200	4700
4900	5700
7200	8600
4100	5100
5800	7600
3700	5400
5300	8700
5600	9900
3500	6700
3900	8100
2800	6200
3100	7800
3500	9800
1600	5500
2400	9400
900	6000

Note. The participants were asked to choose between the two options presented in each row: The left one represented an immediate small reward, and the right one represented a delayed large reward. A total of 20 choices were presented in random order.

Table A5

Risk-Taking Task (Adapted from Griskevicius et al., 2013)

JPY to be received for sure	JPY to be received with a 54% chance
5600	6500
5300	7100
6100	9200
2900	4900
2700	4700
3100	5700
4400	8600
2400	5100
3500	7600
2400	5400
3700	8700
4000	9900
2600	6700
3000	8100
2200	6200
2600	7800
3000	9800
1500	5500
2100	9400
1000	6000

Note. The participants were asked to choose between the two options presented in each row: The left one represented definitely earning a fixed amount of money, and the right one represented earning a larger amount of money with a certain probability. A total of 20 choices were presented in random order.

Table A6

Mini-K Scale (Figueredo et al., 2006; Kawamoto, 2015, for Japanese Version)

Item	
1.	I can often tell how things will turn out （私は何かできごとが起きたときに，そのできごとが次にどうなるか分かる）
2.	I try to understand how I got into a situation to figure out how to handle it （私は今の状況に対処するため、どのように今の状況に至ったのかを理解しようとする）
3.	I felt relatively wealthy compared to the other kids in my school （私は、好ましくない状況の良い面を見つけようとする）
4.	I don’t give up until I solve my problems （私は、自分が抱えている問題を解決するまで投げ出さない）
5.	I often make plans in advance （私は何かをするときに、あらかじめ計画を立てる）
6.	I avoid taking risks （私は何かをするときに、危ない橋を渡るようなことはしない）
7.	While growing up, I had a close and warm relationship with my biological mother （子どものころ、私は自分の実の母親と、温かく親密な関係をきずいていた）
8.	While growing up, I had a close and warm relationship with my biological father （子どものころ、私は自分の実の父親と、温かく親密な関係をきずいていた）
9.	I have a close and warm relationship with my own children （私は自分の子どもと、温かく親密な関係をきずいている）
10.	I have a close and warm romantic relationship with my sexual partner （私は自分のパートナーと、温かく親密な、互いに愛し合う関係をきずいている）
11.	I would rather have one than several sexual relationships at a time （私は同時に複数のパートナーと性的な関係をもつより、一人の人と関係をもっていたい）
12.	I have to be closely attached to someone before I am comfortable having sex with them （私は人と親密な関係をきずかなければ、その人と抵抗なく性的な関係をもつことができない）
13.	I am often social contact with my blood relatives （私は自分と血のつながった親戚とよく連絡をとったりする）
14.	I often get emotional support and practical help from my blood relatives （私と血のつながった親戚は、私の気持ちをささえてくれたり、手助けしてくれたりする）

Table A6 (*continued*)

Item	
15.	I often give emotional support and practical help to my blood relatives （私は、自分と血のつながった親戚の気持ちをささえたり、何かしらの手助けをしたりする）
16.	I am often in social contact with my friends （私は友だちとよく連絡をとったりする）
17.	I often get emotional support and practical help from my friends （私の友だちは、私の気持ちをささえてくれたり、手助けをしてくれたりする）
18.	I often give emotional support and practical help to my friends （私は友だちの気持ちをささえたり、何かしらの手助けをしたりする）
19.	I am closely connected to and involved in my community （私は自分が住む地域に愛着をもっているし、積極的にかかわっている）
20.	I am closely connected to and involved in my religion （私は自分の信じる宗教を大事にしているし、積極的にかかわっている）

Table A7

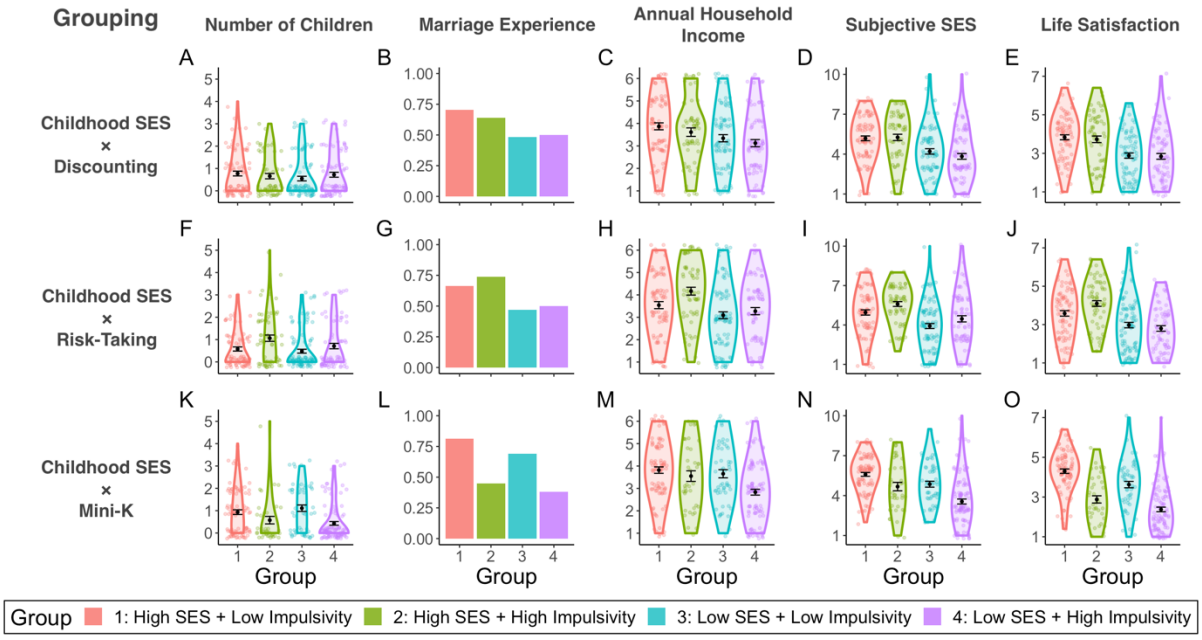
Satisfaction With Life Scale (Diener et al., 1985; Koyasu et al., 2012, for the Japanese Version)

Item	
1.	I am satisfied with my life （私は自分の人生に満足している）
2.	The conditions of my life are excellent （私の生活環境は素晴らしいものである）
3.	In most ways my life is close to my ideal （大体において、私の人生は理想に近いものである）
4.	If I could live my life over, I would change almost nothing （もう一度人生をやり直すとしても、私には変えたいと思うところはほとんどない）
5.	So far I have gotten the important things I want in life （これまで私は望んだものは手に入れてきた）

Appendix B

Figure B1

Distribution of Fitness Indices of Four Groups Divided into Upper/Lower Tertiles of Childhood Socioeconomic Status (SES) and Impulsivity



Note. The hypothesis predicts a horizontally mirrored J-shape: Highest, second highest, lowest, second lowest for Groups 1–4, respectively. Results based on childhood SES × temporal discounting grouping, childhood SES × risk taking grouping, childhood SES × Mini-K grouping are presented as Panels A–E, Panels F–J, and Panels K–O, respectively. The dependent variables were the number of children for Panels A, F, and K; marriage experience for Panels B, G, and L; annual household income for Panels C, H, and M; subjective SES for Panels D, I, and N; and life satisfaction for Panels E, J, and O.

Table B1

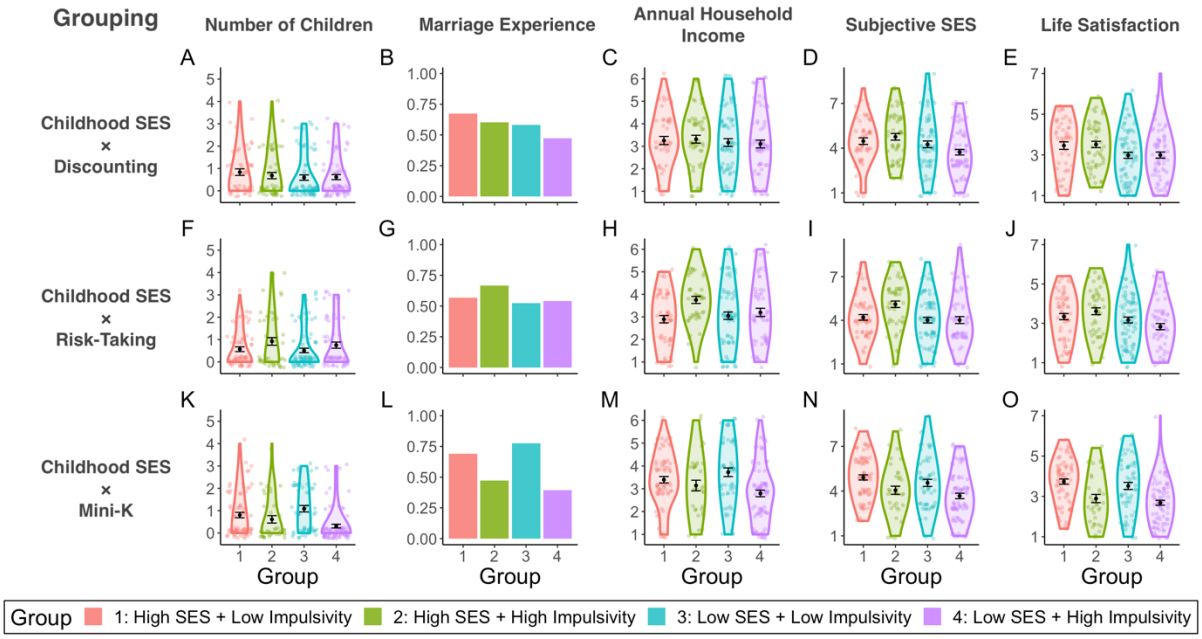
Results of a Series of Planned Contrasts Analyses for Four Groups Divided into Upper/Lower Tertiles of Childhood SES and Impulsivity

	Variable Used for Grouping								
	Temporal Discounting			Risk Taking			Mini-K		
	*n*s = 86, 62, 85, and 82 for Groups 1, 2, 3, and 4, respectively			*n*s = 84, 66, 98, and 73 for Groups 1, 2, 3, and 4, respectively			*n*s = 93, 40, 55, and 102 for Groups 1, 2, 3, and 4, respectively		
	Corresponding to Panels A–E of Figure B1			*Corresponding to Panels F–J of Figure B1*			*Corresponding to Panels K–O of Figure B1*		
	b	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*
DV = Number of Children									
Contrast 1	0.08	0.10	.410	***−0.30***	0.09	.001	**0.24**	0.12	.039
Contrast 2	−0.14	0.10	.165	−0.20	0.10	.054	***0.47***	0.10	<.001
Contrast 3	0.13	0.14	.368	**0.30**	0.14	.032	0.06	0.15	.711
DV = Marriage Experience (0 = lifetime singlehood, 1 = having at least one marriage)									
Contrast 1	0.15	0.18	.397	−0.18	0.18	.321	**0.84**	0.21	<.001
Contrast 2	−0.04	0.16	.820	−0.06	0.16	.693	***0.64***	0.18	<.001
Contrast 3	**0.76**	0.24	.001	**0.92**	0.24	<.001	0.47	0.27	.084
DV = Annual Household Income									
Contrast 1	0.13	0.12	.188	***−0.31***	0.12	.009	0.13	0.13	.303
Contrast 2	0.11	0.11	.334	−0.09	0.11	.414	***0.41***	0.12	<.001
Contrast 3	**0.51**	0.17	.002	**0.67**	0.16	<.001	**0.44**	0.17	.011
DV = Subjective SES									
Contrast 1	−0.04	0.15	.819	***−0.32***	0.15	.034	**0.46**	0.16	.005
Contrast 2	0.19	0.14	.189	−0.27	0.14	.052	***0.65***	0.14	<.001
Contrast 3	**1.21**	0.21	<.001	**1.08**	0.21	<.001	**0.93**	0.22	<.001
DV = Life Satisfaction									
Contrast 1	0.05	0.11	.621	***−0.26***	0.11	.017	**0.71**	0.11	<.001
Contrast 2	0.02	0.10	.842	0.09	0.10	.383	***0.63***	0.10	<.001
Contrast 3	**0.92**	0.15	<.001	**0.96**	0.15	<.001	**0.59**	0.14	<.001

Note. Number of children was submitted to Poisson regression analyses; marriage experience was submitted to logistic regression analyses. **Bold** coefficients indicate significant difference in the hypothesized direction. ***Bold italic*** coefficients indicate significant difference opposite to the hypothesized direction.

Figure B2

Distribution of Fitness Indices of Four Groups Divided by Medians of Childhood Socioeconomic Status (SES) and Impulsivity (Only Individuals Whose Annual Household Income Decreased from the Last Year)



Note. The hypothesis predicts a horizontally mirrored J-shape: Highest, second highest, lowest, second lowest for Groups 1–4, respectively. Results based on childhood SES × temporal discounting grouping, childhood SES × risk taking grouping, childhood SES × Mini-K grouping are presented as Panels A–E, Panels F–J, and Panels K–O, respectively. The dependent variables were the number of children for Panels A, F, and K; marriage experience for Panels B, G, and L; annual household income for Panels C, H, and M; subjective SES for Panels D, I, and N; and life satisfaction for Panels E, J, and O.

Table B2

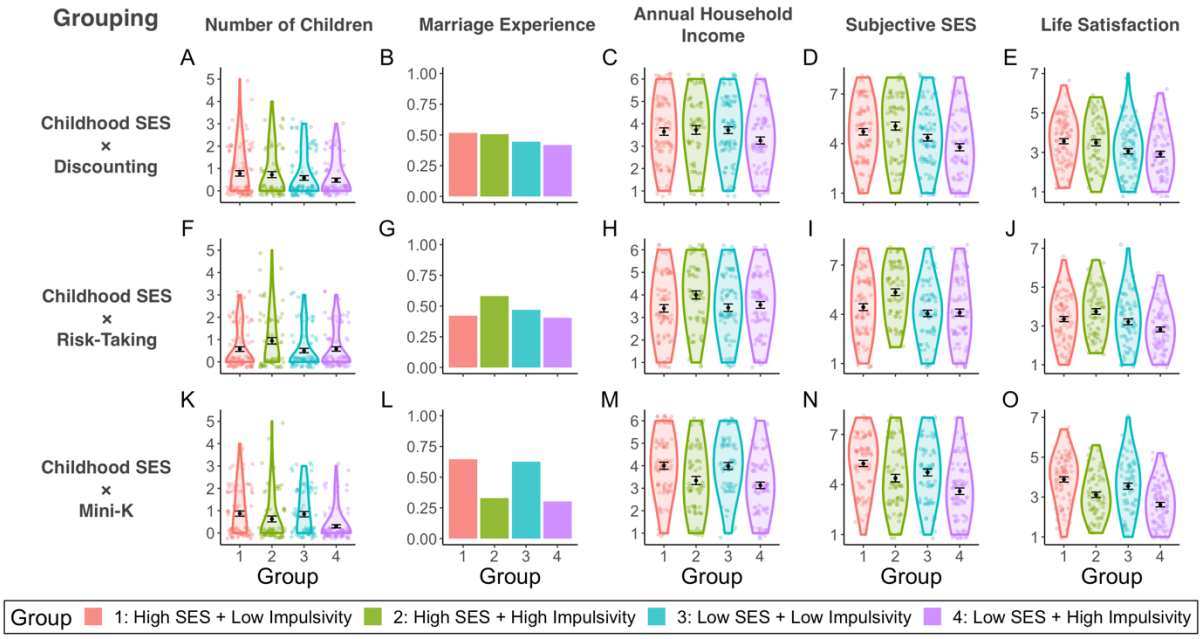
Results of a Series of Planned Contrasts Analyses (Only Participants Whose Annual Household Income Decreased from the Last Year)

	Variable Used for Grouping								
	Temporal Discounting			Risk Taking			Mini-K		
	*n*s = 49, 54, 72, and 70 for Groups 1, 2, 3, and 4, respectively			*n*s = 59, 48, 80, and 61 for Groups 1, 2, 3, and 4, respectively			*n*s = 72, 36, 58, and 84 for Groups 1, 2, 3, and 4, respectively		
	Corresponding to Panels A–E of Figure B2			*Corresponding to Panels F–J of Figure B2*			*Corresponding to Panels K–O of Figure B2*		
	b	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*
DV = Number of Children									
Contrast 1	0.10	0.11	.361	***−0.24***	0.12	.038	0.14	0.13	.277
Contrast 2	−0.02	0.11	.862	−0.19	0.11	.078	***0.63***	0.12	<.001
Contrast 3	0.22	0.16	.156	0.15	0.16	.334	0.19	0.17	.272
DV = Marriage Experience (0 = lifetime singlehood, 1 = having at least one marriage)									
Contrast 1	0.15	0.21	.465	−0.21	0.20	.305	**0.46**	0.21	.030
Contrast 2	0.23	0.17	.183	−0.03	0.17	.851	***0.84***	0.19	<.001
Contrast 3	0.46	0.27	.084	**0.35**	0.27	.182	−0.06	0.29	.838
DV = Annual Household Income									
Contrast 1	−0.03	0.14	.829	***−0.43***	0.13	.001	0.13	0.14	.358
Contrast 2	0.03	0.12	.779	−0.07	0.11	.522	***0.46***	0.11	<.001
Contrast 3	0.15	0.18	.402	0.20	0.17	.253	0.01	0.18	.960
DV = Subjective SES									
Contrast 1	−0.15	0.17	.282	***−0.45***	0.16	.006	**0.43**	0.17	.011
Contrast 2	0.26	0.14	.065	−0.00	0.14	.990	***0.44***	0.14	.002
Contrast 3	**0.62**	0.22	.005	**0.64**	0.22	.003	0.37	0.22	.089
DV = Life Satisfaction									
Contrast 1	−0.03	0.12	.813	−0.14	0.12	.265	**0.42**	0.12	<.001
Contrast 2	−0.01	0.10	.948	0.17	0.11	.113	***0.42***	0.10	<.001
Contrast 3	**0.50**	0.16	.002	**0.48**	0.16	.003	0.21	0.16	.179

Note. Number of children was submitted to Poisson regression analyses; marriage experience was submitted to logistic regression analyses. **Bold** coefficients indicate significant difference in the hypothesized direction. ***Bold italic*** coefficients indicate significant difference opposite to the hypothesized direction.

Figure B3

Distribution of Fitness Indices of Four Groups Divided by Medians of Childhood Socioeconomic Status (SES) and Impulsivity (Male Participants)



Note. The hypothesis predicts a horizontally mirrored J-shape: Highest, second highest, lowest, second lowest for Groups 1–4, respectively. Results based on childhood SES × temporal discounting grouping, childhood SES × risk taking grouping, childhood SES × Mini-K grouping are presented as Panels A–E, Panels F–J, and Panels K–O, respectively. The dependent variables were the number of children for Panels A, F, and K; marriage experience for Panels B, G, and L; annual household income for Panels C, H, and M; subjective SES for Panels D, I, and N; and life satisfaction for Panels E, J, and O.

Table B3

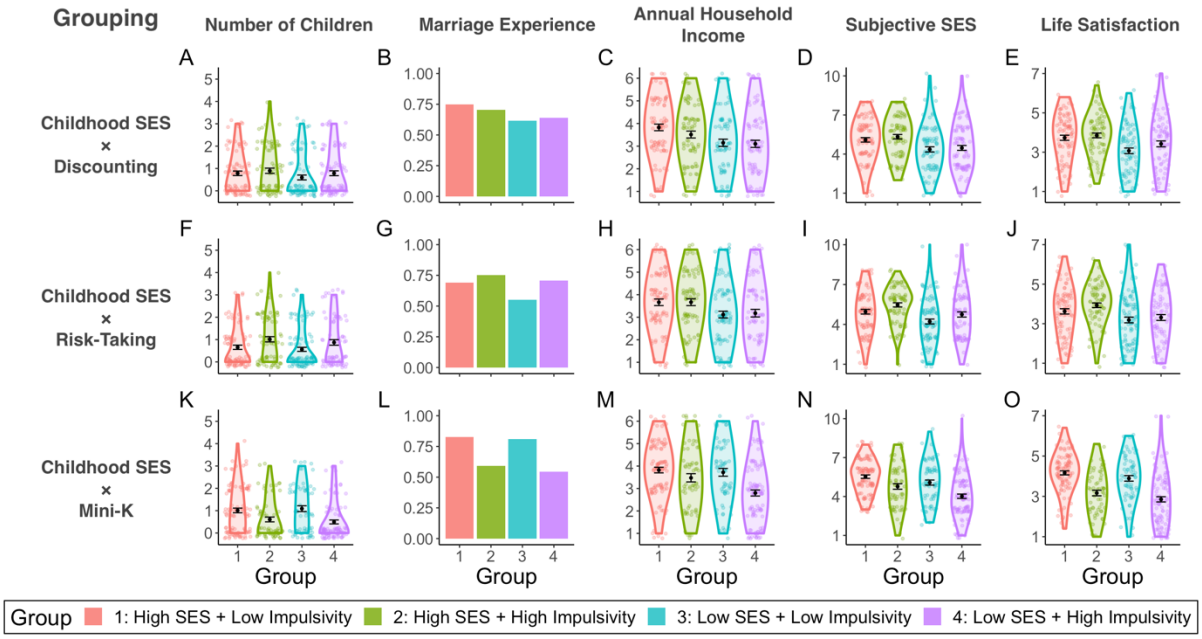
Results of a Series of Planned Contrasts Analyses (Male Participants)

	Variable Used for Grouping								
	Temporal Discounting			Risk Taking			Mini-K		
	*n*s = 88, 65, 94, and 80 for Groups 1, 2, 3, and 4, respectively			*n*s = 83, 73, 81, and 90 for Groups 1, 2, 3, and 4, respectively			*n*s = 85, 71, 76, and 93 for Groups 1, 2, 3, and 4, respectively		
	Corresponding to Panels A–E of Figures B3			*Corresponding to Panels F–J of Figure B3*			*Corresponding to Panels K–O of Figure B3*		
	b	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*
DV = Number of Children									
Contrast 1	0.04	0.10	.695	***−0.25***	0.10	.008	0.16	0.10	.090
Contrast 2	0.10	0.11	.369	−0.07	0.10	.526	***0.51***	0.11	<.001
Contrast 3	**0.36**	0.14	.011	**0.31**	0.14	.029	**0.38**	0.15	.010
DV = Marriage Experience (0 = lifetime singlehood, 1 = having at least one marriage)									
Contrast 1	0.02	0.16	.907	***−0.33***	0.16	.046	**0.66**	0.17	<.001
Contrast 2	0.06	0.15	.701	0.13	0.16	.396	***0.68***	0.16	<.001
Contrast 3	0.32	0.23	.152	0.27	0.23	.238	0.11	0.24	.649
DV = Annual Household Income									
Contrast 1	−0.04	0.12	.769	***−0.30***	0.12	.013	**0.33**	0.12	.005
Contrast 2	***0.23***	0.11	.042	−0.06	0.11	.587	***0.43***	0.11	<.001
Contrast 3	0.21	0.17	.218	0.21	0.17	.209	0.11	0.16	.477
DV = Subjective SES									
Contrast 1	−0.17	0.16	.263	***−0.46***	0.15	.003	**0.44**	0.15	.003
Contrast 2	***0.29***	0.15	.046	−0.03	0.14	.862	***0.56***	0.14	<.001
Contrast 3	**0.82**	0.21	<.001	**0.82**	0.21	<.001	**0.66**	0.21	.001
DV = Life Satisfaction									
Contrast 1	0.04	0.10	.711	−0.20	0.10	.050	**0.38**	0.10	<.001
Contrast 2	0.07	0.10	.457	***0.20***	0.10	.042	***0.46***	0.09	<.001
Contrast 3	**0.55**	0.14	<.001	**0.53**	0.14	<.001	**0.42**	0.13	.002

Note. Number of children was submitted to Poisson regression analyses; marriage experience was submitted to logistic regression analyses. **Bold** coefficients indicate significant difference in the hypothesized direction. ***Bold italic*** coefficients indicate significant difference opposite to the hypothesized direction.

Figure B4

Distribution of Fitness Indices of Four Groups Divided by Medians of Childhood Socioeconomic Status (SES) and Impulsivity (Female Participants)



Note. The hypothesis predicts a horizontally mirrored J-shape: Highest, second highest, lowest, second lowest for Groups 1–4, respectively. Results based on childhood SES × temporal discounting grouping, childhood SES × risk taking grouping, childhood SES × Mini-K grouping are presented as Panels A–E, Panels F–J, and Panels K–O, respectively. The dependent variables were the number of children for Panels A, F, and K; marriage experience for Panels B, G, and L; annual household income for Panels C, H, and M; subjective SES for Panels D, I, and N; and life satisfaction for Panels E, J, and O.

Table B4

Results of a Series of Planned Contrasts Analyses (Female Participants)

	Variable Used for Grouping								
	Temporal Discounting			Risk Taking			Mini-K		
	*n*s = 88, 65, 94, and 80 for Groups 1, 2, 3, and 4, respectively			*n*s = 83, 73, 81, and 90 for Groups 1, 2, 3, and 4, respectively			*n*s = 85, 71, 76, and 93 for Groups 1, 2, 3, and 4, respectively		
	Corresponding to Panels A–E of Figure B4			*Corresponding to Panels F–J of Figure B4*			*Corresponding to Panels K–O of Figure B4*		
	b	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*
DV = Number of Children									
Contrast 1	−0.07	0.09	.426	***−0.22***	0.09	.012	**0.26**	0.09	.006
Contrast 2	−0.14	0.09	.140	**−0.22**	0.09	.016	***0.39***	0.09	<.001
Contrast 3	0.20	0.13	.109	0.15	0.13	.229	0.06	0.13	.629
DV = Marriage Experience (0 = lifetime singlehood, 1 = having at least one marriage)									
Contrast 1	0.11	0.18	.517	−0.16	0.17	.361	**0.60**	0.18	<.001
Contrast 2	−0.06	0.16	.725	**−0.34**	0.16	.034	***0.63***	0.18	<.001
Contrast 3	**0.46**	0.24	.049	0.41	0.24	.085	0.16	0.26	.532
DV = Annual Household Income									
Contrast 1	0.16	0.11	.153	−0.00	0.11	.986	0.17	0.11	.114
Contrast 2	0.02	0.11	.833	−0.03	0.11	.773	***0.46***	0.11	<.001
Contrast 3	**0.55**	0.15	<.001	**0.52**	0.15	<.001	**0.39**	0.15	.011
DV = Subjective SES									
Contrast 1	−0.12	0.13	.350	***−0.26***	0.13	.044	**0.38**	0.13	.003
Contrast 2	−0.06	0.13	.666	**−0.26**	0.13	.036	***0.53***	0.12	<.001
Contrast 3	**0.80**	0.18	<.001	**0.73**	0.18	<.001	**0.61**	0.18	<.001
DV = Life Satisfaction									
Contrast 1	−0.06	0.10	.541	−0.15	0.10	.114	**0.50**	0.09	<.001
Contrast 2	−0.18	0.10	.066	−0.06	0.10	.515	***0.52***	0.09	<.001
Contrast 3	**0.55**	0.14	<.001	**0.53**	0.14	<.001	**0.29**	0.13	.023

Note. Number of children was submitted to Poisson regression analyses; marriage experience was submitted to logistic regression analyses. **Bold** coefficients indicate significant difference in the hypothesized direction. ***Bold italic*** coefficients indicate significant difference opposite to the hypothesized direction.

Table B5

Results of a Series of Multiple Regression Analyses

	Impulsivity Variable								
	Temporal Discounting			Risk Taking			Mini-K		
	b	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*
DV = Number of Children									
Childhood SES	.120	.047	.011	.086	.047	.068	.016	.051	.752
(Impulsivity)	.035	.046	.444	.173	.045	<.001	.406	.049	<.001
Interaction	−.046	.044	.299	.079	.044	.072	−.046	.045	.313
DV = Marriage Experience (0 = lifetime singlehood, 1 = having at least one marriage)									
Childhood SES	.276	.081	<.001	.261	.080	.001	.063	.090	.485
(Impulsivity)	−.090	.079	.259	.123	.080	.124	.898	.103	<.001
Interaction	−.062	.079	.414	.094	.079	.232	.099	.092	.284
DV = Annual Household Income									
Childhood SES	.162	.038	<.001	.167	.038	<.001	.110	.039	.005
(Impulsivity)	−.081	.038	.036	.075	.038	.049	.200	.039	<.001
Interaction	−.007	.036	.839	.061	.037	.102	−.025	.033	.449
DV = Subjective SES									
Childhood SES	.261	.038	<.001	.264	.037	<.001	.177	.037	<.001
(Impulsivity)	−.023	.037	.539	.143	.037	<.001	.328	.037	<.001
Interaction	.037	.035	.291	.018	.036	.623	−.033	.032	.293
DV = Life Satisfaction									
Childhood SES	.273	.038	<.001	.274	.037	<.001	.130	.035	<.001
(Impulsivity)	−.006	.038	.866	.027	.037	.467	.480	.035	<.001
Interaction	−.019	.036	.594	.103	.036	.005	−.000	.029	.996

Note. Childhood SES and impulsivity were standardized for the set of these multiple regression analyses. Number of children was submitted to Poisson regression analyses, and marriage experience was submitted to logistic regression analyses. For the remaining dependent variables (i.e., annual household income, subjective SES, and life satisfaction), the reported regression coefficients are standardized coefficients. For the two shaded cells (i.e., one significant and one marginally significant interaction effects), we conducted a series of simple slope tests. Neither of the interactions was consistent with the hypothesis (the slope is positive for low childhood SES group and negative for high childhood SES group).

Table B6

Results of a Series of Multiple Regression Analyses (Only Participants Whose Annual Household Income Decreased from the Last Year)

	Impulsivity Variable								
	Temporal Discounting			Risk Taking			Mini-K		
	b	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*
DV = Number of Children									
Childhood SES	.141	.080	.081	.090	.081	.268	.030	.088	.731
(Impulsivity)	.106	.078	.170	.189	.076	.013	.447	.086	<.001
Interaction	−.044	.078	.578	.084	.076	.269	−.123	.077	.112
DV = Marriage Experience (0 = lifetime singlehood, 1 = having at least one marriage)									
Childhood SES	.332	.135	.014	.298	.133	.026	.045	.147	.758
(Impulsivity)	−.038	.134	.779	.158	.133	.238	.780	.162	<.001
Interaction	−.103	.136	.451	.126	.132	.342	−.050	.143	.727
DV = Annual Household Income									
Childhood SES	.114	.064	.078	.118	.063	.063	.032	.066	.063
(Impulsivity)	−.045	.064	.483	.157	.063	.013	.202	.066	.002
Interaction	−.057	.063	.364	.094	.062	.129	−.077	.054	.151
DV = Subjective SES									
Childhood SES	.224	.063	<.001	.223	.061	<.001	.097	.061	.115
(Impulsivity)	−.060	.063	.346	.165	.061	.008	.365	.062	<.001
Interaction	.057	.061	.841	.089	.060	.140	.040	.050	.431
DV = Life Satisfaction									
Childhood SES	.219	.063	<.001	.200	.062	.001	.067	.060	.266
(Impulsivity)	.024	.063	.702	−.000	.062	.994	.418	.061	<.001
Interaction	−.011	.061	.862	.142	.061	.020	.012	.050	.803

Note. Childhood SES and impulsivity were standardized for the set of these multiple regression analyses. Number of children was submitted to Poisson regression analyses, and marriage experience was submitted to logistic regression analyses. For the remaining dependent variables (i.e., annual household income, subjective SES, and life satisfaction), the reported regression coefficients are standardized coefficients. For the shaded cell (i.e., the significant interaction effect), we conducted a simple slope test. The interaction was not consistent with the hypothesis (the slope is positive for low childhood SES group and negative for high childhood SES group).

Table B7

Results of a Series of Multiple Regression Analyses (Male Participants)

	Impulsivity Variable								
	Temporal Discounting			Risk Taking			Mini-K		
	b	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*
DV = Number of Children									
Childhood SES	.185	.073	.011	.160	.074	.031	.087	.080	.277
(Impulsivity)	.034	.070	.622	.244	.070	<.001	.439	.074	<.001
Interaction	.019	.065	.773	.026	.071	.709	−.028	.063	.657
DV = Marriage Experience (0 = lifetime singlehood, 1 = having at least one marriage)									
Childhood SES	.237	.114	.038	.208	.114	.068	.007	.126	.958
(Impulsivity)	.028	.113	.803	.163	.113	.147	.880	.149	<.001
Interaction	−.011	.103	.914	.112	.112	.317	.046	.127	.717
DV = Annual Household Income									
Childhood SES	.088	.055	.113	.111	.055	.043	.039	.055	.480
(Impulsivity)	−.066	.056	.235	.142	.055	.010	.028	.057	<.001
Interaction	.077	.050	.124	.089	.053	.096	.036	.044	.416
DV = Subjective SES									
Childhood SES	.219	.055	<.001	.238	.054	<.001	.141	.053	.008
(Impulsivity)	−.022	.055	.689	.147	.054	.006	.336	.055	<.001
Interaction	.069	.049	.164	.074	.053	.159	−.030	.043	.491
DV = Life Satisfaction									
Childhood SES	.248	.055	<.001	.263	.054	<.001	.111	.050	.027
(Impulsivity)	−.040	.055	.471	.008	.054	.884	.482	.051	<.001
Interaction	−.007	.050	.887	.140	.053	.008	.030	.040	.445

Note. Childhood SES and impulsivity were standardized for the set of these multiple regression analyses. Number of children was submitted to Poisson regression analyses, and marriage experience was submitted to logistic regression analyses. For the remaining dependent variables (i.e., annual household income, subjective SES, and life satisfaction), the reported regression coefficients are standardized coefficients. For the two shaded cells (i.e., one significant and one marginally significant interaction effects), we conducted a series of simple slope tests. Neither of the interactions was consistent with the hypothesis (the slope is positive for low childhood SES group and negative for high childhood SES group).

Table B8

Results of a Series of Multiple Regression Analyses (Female Participants)

	Impulsivity Variable								
	Temporal Discounting			Risk Taking			Mini-K		
	b	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*
Number of Children									
Childhood SES	.066	.063	.293	.027	.063	.666	−.048	.067	.476
(Impulsivity)	.049	.063	.426	.150	.060	.012	.360	.066	<.001
Interaction	−.114	.061	.063	.095	.056	.092	−.043	.063	.488
Marriage Experience (0 = lifetime singlehood, 1 = having at least one marriage)									
Childhood SES	.328	.121	.007	.325	.120	.007	.163	.137	.234
(Impulsivity)	−.129	.117	.270	.255	.125	.041	.741	.142	<.001
Interaction	−.124	.119	.301	.104	.122	.393	.116	.131	.376
Annual Household Income									
Childhood SES	.238	.053	<.001	.231	.053	<.001	.158	.055	.004
(Impulsivity)	−.089	.053	.095	−.024	.053	.655	.195	.054	<.001
Interaction	−.076	.052	.147	.058	.051	.257	−.106	.049	.033
Subjective SES									
Childhood SES	.313	.052	<.001	.302	.051	<.001	.023	.052	<.001
(Impulsivity)	.017	.052	.750	.172	.051	<.001	.295	.052	<.001
Interaction	.009	.051	.856	−.037	.049	.452	−.038	.047	.421
Life Satisfaction									
Childhood SES	.291	.052	<.001	.281	.052	<.001	.136	.049	.006
(Impulsivity)	.052	.052	.321	.073	.052	.161	.479	.049	<.001
Interaction	−.031	.051	.549	.065	.050	.197	−.044	.044	.322

Note. Childhood SES and impulsivity were standardized for the set of these multiple regression analyses. Number of children was submitted to Poisson regression analyses, and marriage experience was submitted to logistic regression analyses. For the remaining dependent variables (i.e., annual household income, subjective SES, and life satisfaction), the reported regression coefficients are standardized coefficients. For the three shaded cells (i.e., one significant and two marginally significant interaction effects), we conducted a series of simple slope tests. Of the three, the marginally significant interaction between childhood SES and temporal discounting on number of children tended to be supportive of the life history hypothesis (the slope is positive and significant for low childhood SES group and negative but non-significant for high childhood SES group), while the other two were not consistent with the hypothesis.

Table B9

Results of a Series of Planned Contrasts Using the Number of Marriages as the Dependent Variable

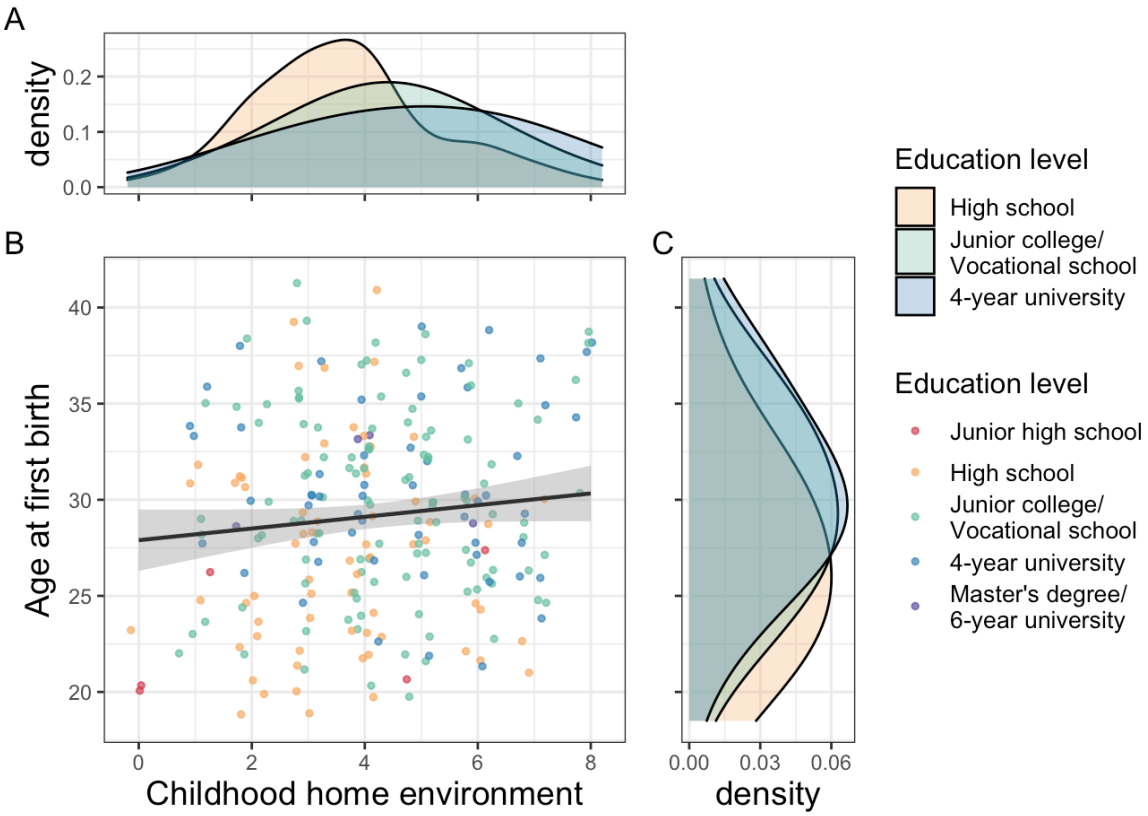
	Impulsivity Variable								
	Temporal Discounting			Risk Taking			Mini-K		
	b	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*
All participants									
DV = Number of Marriages									
	*n*s = 173, 149, 190, and 158 for Groups 1, 2, 3, and 4			*n*s = 167, 160, 183, and 165 for Groups 1, 2, 3, and 4			*n*s = 185, 138, 145, and 197 for Groups 1, 2, 3, and 4		
Contrast 1	0.07	0.07	.342	−0.11	0.07	.110	**0.21**	0.07	.004
Contrast 2	−0.01	0.07	.993	−0.04	0.07	.526	***0.26***	0.07	<.001
Contrast 3	0.13	0.10	.205	0.11	0.10	.256	0.04	0.11	.666
Four groups divided into upper/lower tertiles of childhood SES and impulsivity									
DV = Number of Marriages									
	*n*s = 86, 62, 85, and 82 for Groups 1, 2, 3, and 4			*n*s = 84, 66, 98, and 73 for Groups 1, 2, 3, and 4			*n*s = 93, 40, 55, and 102 for Groups 1, 2, 3, and 4		
Contrast 1	0.07	0.10	.468	−0.07	0.10	.489	0.24	0.12	.053
Contrast 2	−0.05	0.11	.625	−0.05	0.11	.661	***0.30***	0.11	<.001
Contrast 3	**0.33**	0.15	.025	**0.38**	0.15	.009	0.21	0.17	.207
Only participants whose annual household income decreased from the last year									
DV = Number of Marriages									
	*n*s = 49, 54, 72, and 70 for Groups 1, 2, 3, and 4			*n*s = 59, 48, 80, and 61 for Groups 1, 2, 3, and 4			*n*s = 72, 36, 58, and 84 for Groups 1, 2, 3, and 4		
Contrast 1	0.07	0.12	.570	−0.08	0.12	.497	0.11	0.13	.379
Contrast 2	0.07	0.11	.529	−0.11	0.11	.306	***0.37***	0.11	<.001
Contrast 3	0.17	0.16	.293	0.11	0.16	.485	0.05	0.17	.753
Only male participants									
DV = Number of Marriages									
	*n*s = 88, 65, 94, and 80 for Groups 1, 2, 3, and 4			*n*s = 83, 73, 81, and 90 for Groups 1, 2, 3, and 4			*n*s = 85, 71, 76, and 93 for Groups 1, 2, 3, and 4		
Contrast 1	0.02	0.11	.886	−0.18	0.11	.102	**0.26**	0.12	.028
Contrast 2	0.04	0.11	.731	0.07	0.11	.521	***0.39***	0.12	<.001
Contrast 3	0.14	0.16	.368	0.13	0.16	.430	0.08	0.17	.622
Only female participants									
DV = Number of Marriages									
	*n*s = 89, 79, 83, and 89 for Groups 1, 2, 3, and 4			*n*s = 88, 82, 96, and 79 for Groups 1, 2, 3, and 4			*n*s = 101, 66, 68, and 103 for Groups 1, 2, 3, and 4		
Contrast 1	0.03	0.09	.714	−0.07	0.09	.398	0.13	0.09	.153
Contrast 2	−0.08	0.09	.371	−0.17	0.09	.059	0.15	0.09	.095
Contrast 3	0.12	0.13	.349	0.10	0.13	.418	0.04	0.13	.777

Note. Number of marriages was submitted to Poisson regression analyses. **Bold** coefficients indicate significant difference in the hypothesized direction. ***Bold italic*** coefficients indicate significant difference opposite to the hypothesized direction.

Appendix C

Figure C1

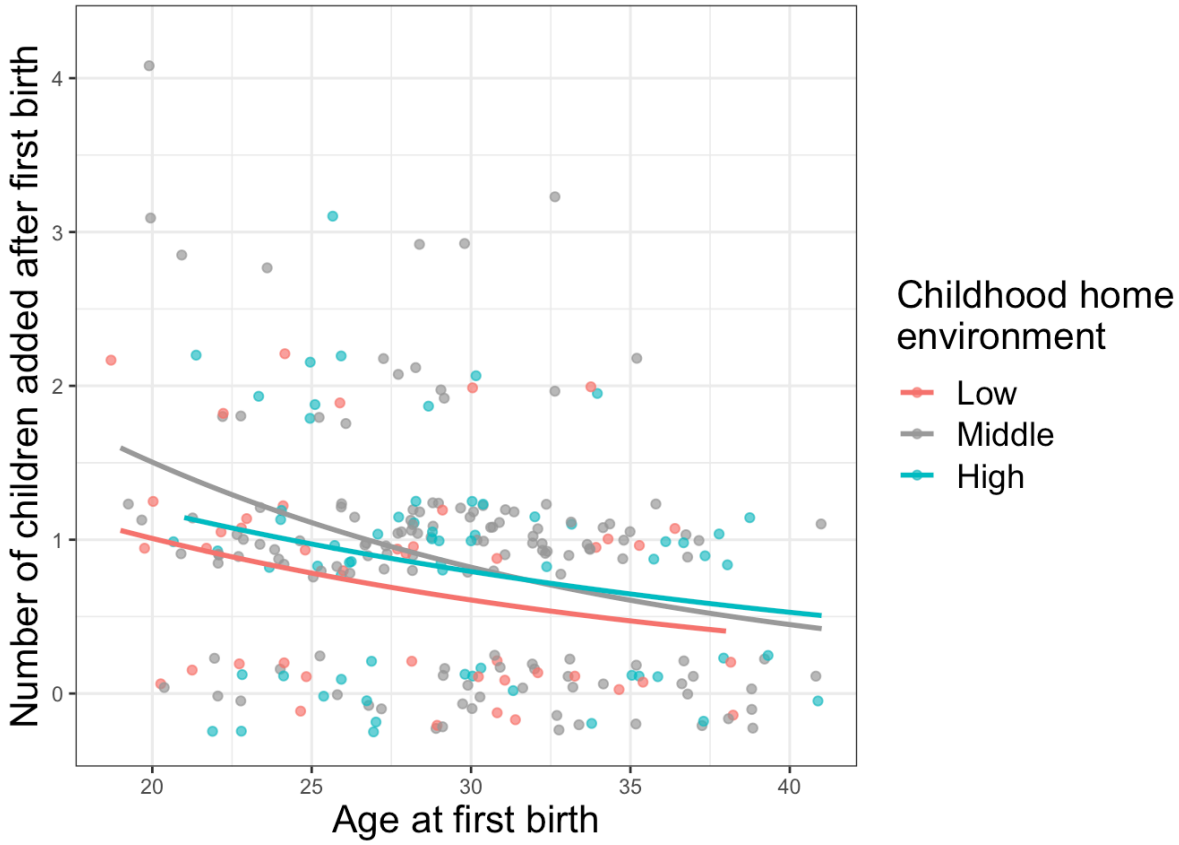
Association between Childhood Home Environment and Age at First Birth and Marginal Distributions as a Function of Level of Educational Background



Note. Density plots did not include the categories of junior high school and master's degree/6-year university because of the small sample sizes (Panels A and C), although scatter plot (Panel B) contained them (i.e., five red and four violet markers for categories of junior high school and master's degree/6-year university, respectively).

Figure C2

Association between Age at First Birth and Number of Children Added after First Birth Plotted as a Function of Level of Childhood Home Environment



Note. Sample was divided by the mean ± 1 standard deviation of childhood home environment. Outcome variable was fitted to Poisson regression model. The adaptive phenotypic plasticity hypothesis predicts that the slope should be negative for low childhood home environment group and positive for high childhood home environment group.

Table C1

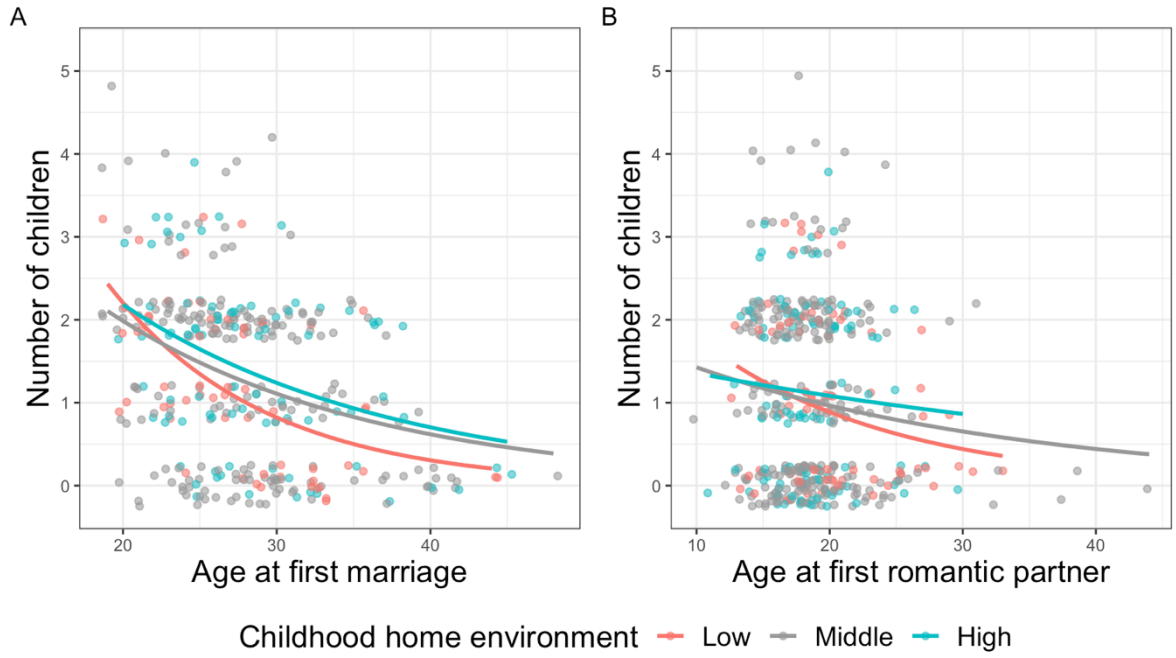
Effects of Childhood Home Environment, Age at First Birth, and Childhood SES × Age at First Birth Interaction on Number of Children Added after First Birth

Variable	*b*	*SE*	*p*
DV = Number of children added after first birth			
Childhood home environment	0.05	0.05	.302
Age at first birth	**−0.22**	0.05	<.001
Interaction	0.02	0.04	.699

Note. Dependent variable (i.e., number of children added after first birth) is submitted to Poisson regression analysis. Childhood home environment and age at first birth were standardized for this analysis before computing the interaction term. **Boldface** coefficient indicates $p < .05$.

Figure C3

Association between (A) Age at First Marriage or (B) Age at First Romantic Relationship and Number of Children Plotted as a Function of Level of Childhood Home Environment



Note. Sample was divided by the mean ± 1 standard deviation of childhood home environment. The number of children was fitted to Poisson regression model. The adaptive phenotypic plasticity hypothesis predicts that the slopes should be negative for low childhood home environment group and positive for high childhood home environment group.

Table C2

Effects of Childhood Home Environment, Age at First Marriage, and Childhood Home Environment × Age at First Marriage Interaction on Number of Children

Variable	*b*	*SE*	*p*
DV = Number of children			
Childhood home environment	0.09	0.05	.069
Age at first birth	**−0.35**	0.05	<.001
Interaction	0.06	0.05	.217

Note. Dependent variable (i.e., number of children) was submitted to Poisson regression analysis. Childhood home environment and age at first marriage were standardized for this analysis before computing the interaction term. **Boldface** coefficient indicates $p < .05$.

Table C3

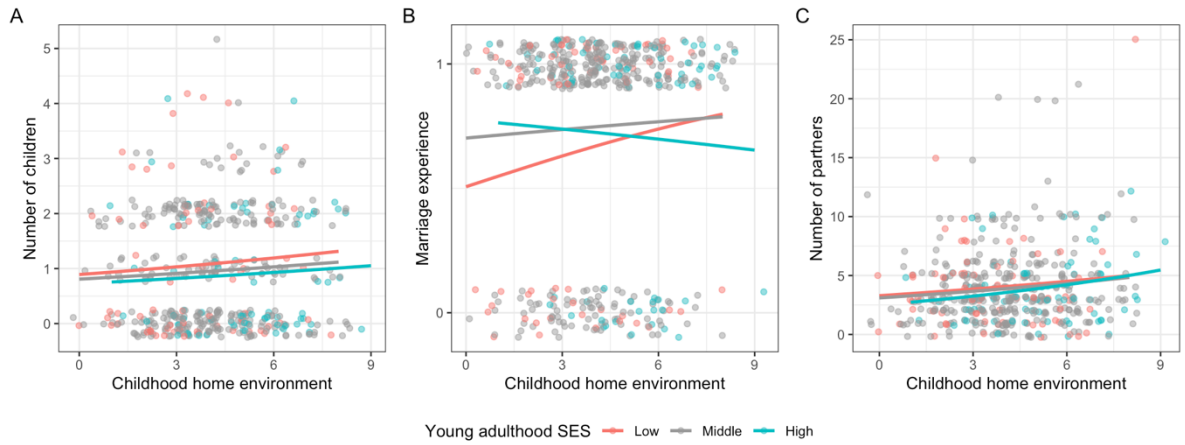
Effects of Childhood Home Environment, Age at First Romantic Relationship, and Childhood Home Environment × Age at First Romantic Relationship Interaction on Number of Children

Variable	*b*	*SE*	*p*
DV = Number of children			
Childhood home environment	0.05	0.05	.256
Age at first birth	**−0.16**	0.05	.004
Interaction	−0.04	0.05	.434

Note. Dependent variable (i.e., number of children) was submitted to Poisson regression analysis. Childhood home environment and age at first romantic relationship were standardized for this analysis before computing the interaction term. **Boldface** coefficient indicates $p < .05$.

Figure C4

Association between Childhood Home Environment and Fitness Indices Plotted as a Function of Level of Young Adulthood SES



Note. The horizontal axis of all panels indicates childhood home environment. The vertical axis of each panel indicates one of the three fitness indices: (A) number of children, (B) marriage experience (0 = never married vs. 1 = have married at least once), and (C) number of partners. The number of children and number of partners were fitted to Poisson regression model. Marriage experience was fitted to logistic regression model. The sample was divided by the mean ± 1 standard deviation of young adulthood SES. External predictive adaptive response hypothesis predicts that the slopes should be negative for low young adulthood SES group but should be positive for high young adulthood SES group.

Table C4

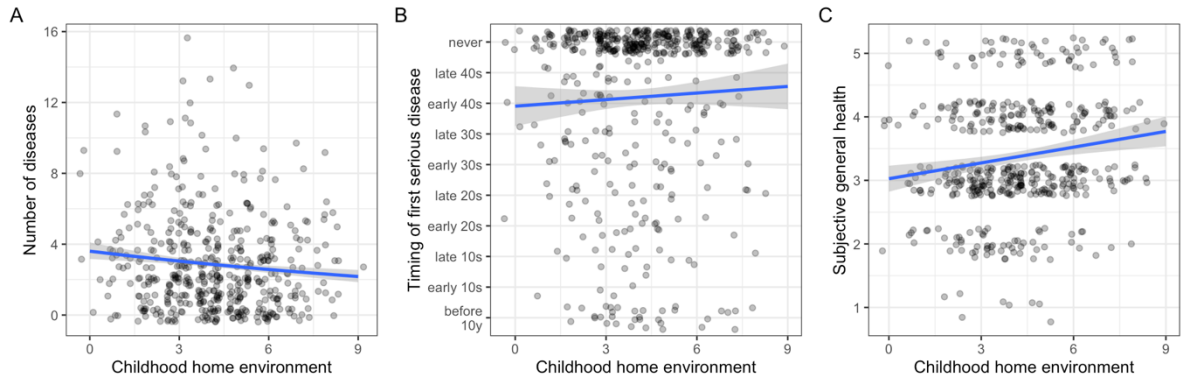
Regression Coefficients of Childhood Home Environment, Young Adulthood SES, and Childhood Home Environment × Young Adulthood SES Interaction on Fitness Indices

Variable	*b*	*SE*	*p*
DV = Number of children			
Childhood home environment	0.07	0.05	.140
Young adulthood SES	−0.05	0.05	.330
Interaction	−0.05	0.04	.267
DV = Marriage experience			
Childhood home environment	0.10	0.11	.374
Young adulthood SES	−0.00	0.11	.965
Interaction	**−0.21**	0.10	.040
DV = Number of partners			
Childhood home environment	**0.11**	0.02	<.001
Young adulthood SES	−0.05	0.02	.057
Interaction	0.01	0.02	.622

Note. The number of children and number of partners were submitted to Poisson regression analyses. Marriage experience (0 = never married vs. 1 = having married at least once) was submitted to logistic regression analysis. Childhood home environment and young adulthood SES were standardized for the three analyses before computing the interaction term. **Boldface** coefficient indicates $p < .05$.

Figure C5

Association between Childhood Home Environment and Health Indicators



Note. In each panel, horizontal axis indicates childhood home environment, and vertical axis indicates one of the three health related variables: (A) number of diseases, (B) timing of first serious disease; and (C) subjective general health (1 = very poor, 2 = poor, 3 = fair, 4 = good, 5 = very good). The number of diseases was fitted to Poisson regression model. Internal predictive adaptive response hypothesis assumed that higher childhood home environment score is associated with better health status (i.e., negative slopes for Panels A and C, and negative slope for Panel B).

Table C5

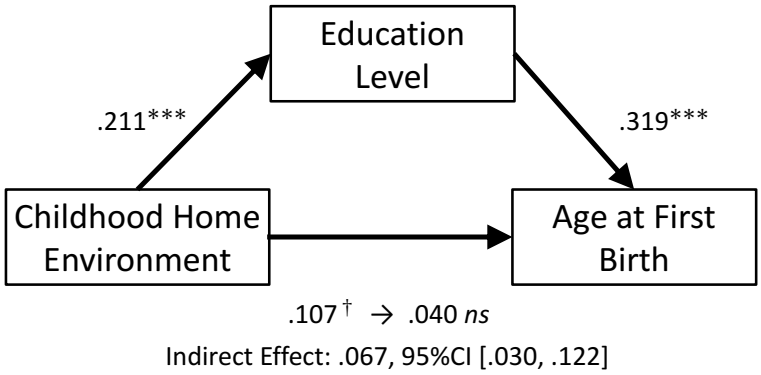
Results of Multiple Regression Analyses for Health Variables

Variable	Estimate	*SE*	*p*
DV = Number of diseases			
Childhood home environment	**−.065**	.028	.020
Young adulthood SES	**−.134**	.028	<.001
Drinking	**.080**	.028	.004
Smoking	.033	.027	.223
Exercise	−.015	.028	.583
Number of children	−.048	.028	.086
DV = Timing of first serious disease			
Childhood home environment	.036	.047	.453
Young adulthood SES	.055	.048	.253
Drinking	−.006	.048	.897
Smoking	.045	.048	.350
Exercise	−.050	.047	.288
Number of children	.054	.046	.240
DV = Subjective general health			
Childhood home environment	**.094**	.045	.037
Young adulthood SES	**.247**	.046	<.001
Drinking	−.028	.046	.534
Smoking	−.047	.046	.311
Exercise	.082	.044	.066
Number of children	**.095**	.044	.030

Note. All independent variables were standardized for analyses. The number of diseases was submitted to Poisson regression analysis. For the remaining dependent variables (i.e., timing of first serious disease and subjective general health), standardized coefficients are shown. **Boldface** coefficients indicate $p < .05$.

Figure C6

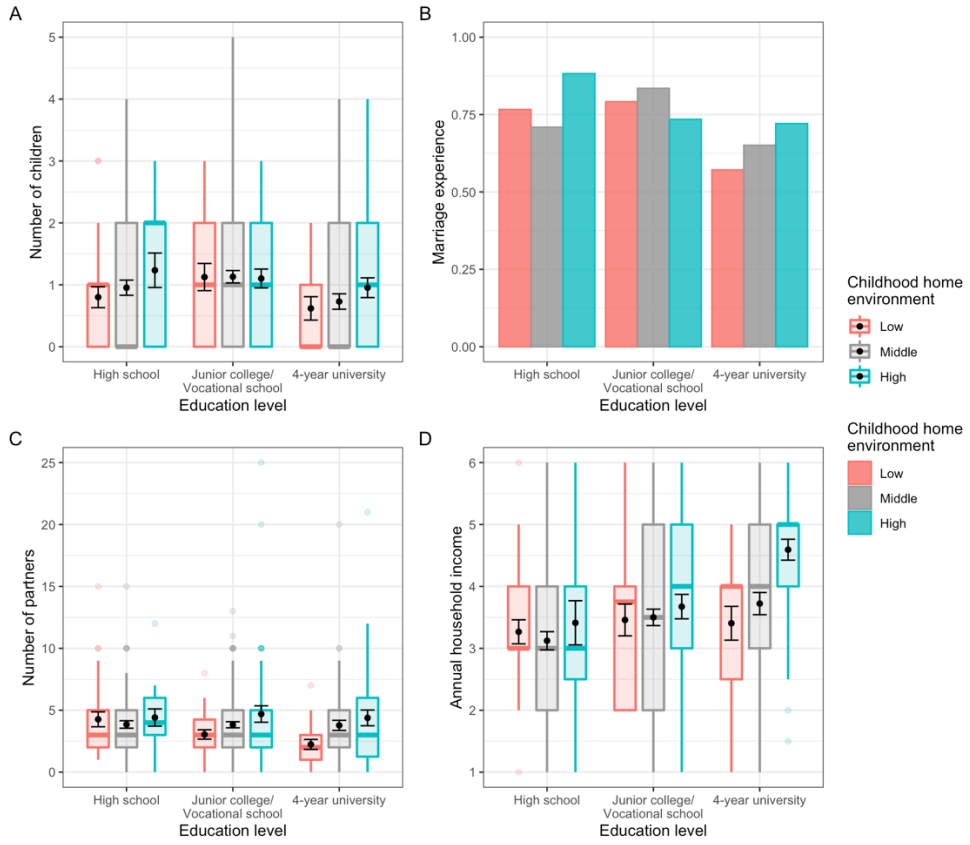
Results of the Mediation Analyses Examining the Indirect Effect of Childhood Home Environment on Age at First Birth via Education Level



† $p < .10$. *** $p < .001$.

Figure C7

Associations between Life History Strategy (Operationalized by Education Level) and Fitness Indices by the Level of Childhood Home Environment



Note. The horizontal axis of all panels indicates education level. The vertical axis of each panel indicates one of the four outcome variables: (A) number of children, (B) marriage experience (proportion of those who married at least once), (C) number of partners; and (D) current annual household income. The sample was divided by the mean ± 1 standard deviation of childhood home environment. In all panels, boxplots and bar charts for junior high school, master's degree/6-year university, and doctoral degree were omitted because of small sample sizes (*N*s = 12, 14, and 2 for junior high school, master's degree/6-year university, and doctoral degree, respectively). In Panels A, B, and D, black dots indicate mean values, and error bars indicate standard errors.

Table C6

Results of a Series of Multiple Regression Analyses Examining the Interaction Effect between Childhood SES and Life History Strategy Operationalized by Education Level on Fitness Indices

Variable	Estimate	*SE*	*p*
DV = Number of children			
Childhood home environment	.087	.048	.068
Education level	**−.120**	.049	.014
Interaction	−.039	.047	.407
DV = Marriage experience			
Childhood home environment	.171	.108	.114
Education level	**−.272**	.109	.013
Interaction	−.126	.101	.212
DV = Number of partners			
Childhood home environment	**.128**	.024	<.001
Education level	**−.109**	.024	<.001
Interaction	**.053**	.022	.019
DV = Current annual household income			
Childhood home environment	**.144**	.044	.001
Education level	**.283**	.045	<.001
Interaction	**.138**	.042	.001

Note. Childhood home environment and education level were standardized for each analysis prior to computing the interaction term. The number of children and number of partners were submitted to Poisson regression analyses. Marriage experience (0 = *never married* vs. 1 = *having married at least once*) was subjected to logistic regression. Standardized coefficients are displayed for the current annual household income. **Boldface** coefficients indicate $p < .05$.