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The impacts of early environmental adversity on cognitive functioning, body mass, and life-history behavioral profiles

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ABSTRACT

Early adverse experiences or exposures have a profound impact on neurophysiological, cognitive, and somatic development. Evidence across disciplines uncovers adversity-induced alternations in cortical structures, cognitive functions, and related behavioral manifestations, as well as an energetic trade-off between the brain and body. Based on the life history (LH) framework, the present research aims to explore the adversity-adapted cognitive-behavioral mechanism and investigate the relation between cognitive functioning and somatic energy reserve (i.e., body mass index; BMI). A structural equation modeling (SEM) analysis was performed with longitudinal self-reported, anthropometric, and task-based data drawn from a cohort of 2,607 8- to 11-year-old youths and their primary caregivers recruited by the Adolescent Brain Cognitive Development (ABCDSM) study. The results showed that early environmental adversity was positively associated with fast LH behavioral profiles and negatively with cognitive functioning. Moreover, cognitive functioning mediated the relationship between adversity and fast LH behavioral profiles. Additionally, we found that early environmental adversity positively predicted BMI, which was inversely correlated with cognitive functioning. These results revealed an adversityadapted cognitive-behavioral mechanism and energy-allocation pathways, and add to the existing knowledge of LH trade-off and developmental plasticity.

1. Introduction

Early environmental adversities, encompassing experience of and exposure to violence, abuse, and neglect, may result in substantial neurobiological and cognitive consequences. The literature documents that individuals who experienced early adversities tend to have decreased volumes in the prefrontal cortex, amygdala, and hippocampus, which actively engage in executive, emotional, and memory processes [\(McEwen et al., 2016; McLaughlin et al., 2019; Tottenham](#page-7-0) & [Sheridan, 2010](#page-7-0)). From an evolutionary-developmental perspective, this may raise questions of how energy is exactly allocated among brain and somatic functions under adverse conditions and how such energyallocation strategy could be adaptive for one's fitness. One of the answers may lie in research exhibiting that reductions in cortical regions, as well as deficits in cognitive functions, are associated with increased fat deposition (registered using the body mass index [BMI]; [Blair et al.,](#page-6-0) [2020; Laurent et al., 2020; Ronan et al., 2020](#page-6-0)), suggesting a trade-off between the brain and body (Kuzawa & [Blair, 2019](#page-7-0)).

Trade-off is a key notion in the life history (LH) theory – an

evolutionary biological framework to conceptualize living organisms' energy-allocation decisions among components of fitness (e.g., growth, maintenance, and reproduction; [Del Giudice et al., 2015; Ellis et al.,](#page-6-0) [2009; Stearns, 1992\)](#page-6-0). The LH theory has two tenets: (1) all living organisms have only a finite energy budget that cannot maximize all their life functions, and (2) energy allocated to one function cannot be used for another. Such biological constraints render organisms subject to making energy-allocation trade-offs among components of fitness ([Del](#page-6-0) [Giudice et al., 2015\)](#page-6-0). Although LH theories have long focused on tradeoffs *between* fitness components (e.g., somatic vs. reproductive effort, growth vs. maintenance), mounting evidence of the brain-body energetic trade-off (for a review see Kuzawa $\&$ [Blair, 2019](#page-7-0)) implies that trade-offs could be further specified *within* particular components, proposing an alternative way to understanding the adaptive plasticity of energy allocation. Based on this body of evidence and extant LH research on the environmental calibration of cognitive and behavioral development, the present research proposed to apply an LH approach to investigate the relations between early environmental adversity, body mass status, cognitive functioning, and behavioral manifestations in

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children.

1.1. The Life-History framework of adaptive plasticity

Energy-allocation decisions are considered as optimal when they are coordinated in the same direction to jointly maximize one's inclusive fitness [\(Ellis et al., 2009](#page-6-0)). This gives rise to divergent *combinations* of LH traits, known as LH strategies. LH traits can be conceptualized as varying on a fast-slow continuum (Promislow & [Harvey, 1990\)](#page-8-0), characterizing different paces of life. Faster LH traits involve early ages at maturity, early and frequent mating activities, giving more births, and less investment in each offspring. Whereas, slower LH traits encompass delayed maturation, less mating activities, fewer birth outputs, and relatively extended somatic growth and brain development [\(Del Giudice](#page-6-0) [et al., 2015; Geary, 2002; Yang et al., 2022](#page-6-0)). Such prolonged brain development permits intensive learning, including acquisition of language and social skills ([Del Giudice et al., 2015\)](#page-6-0).

Energy-allocation decisions are contingent on the extent of environmental adversity (e.g., predation, pathogens). Environmental adversity can be indexed by *harshness*, the rate of age-specific morbiditymortality that is insensitive to individuals' effort (i.e., extrinsic morbidity-mortality), and *unpredictability*, the variance of morbiditymortality rates [\(Ellis et al., 2009\)](#page-6-0). In general, faster LH traits would be favored in harsh and unpredictable environments because they directly increase fitness by yielding a higher quantity of reproductive outputs. By contrast, slower LH traits would be favored in safe and benign environments as they equip agents with increased or specialized abilities to cope with the increasingly tense intraspecific competition by population growth ([Ellis et al., 2009\)](#page-6-0). In LH psychological research, environmental adversity was indexed using socioeconomic and demographic proxies, such as socioeconomic status (SES), income-to-need ratio, parental transition (or absence), family conflict, and neighborhood violence ([Brumbach et al., 2009; Chang et al., 2019a; Chang](#page-6-0) & Lu, 2018; Gris[kevicius et al., 2011; Li et al., 2018; Szepsenwol et al., 2017\)](#page-6-0).

The LH framework was originally proposed to account for betweenspecies variations in resource allocation, yet has been increasingly applied to explain within-species observations. Humans, for instance, are seen as a slow LH species, characterized by extended maturation, delayed reproduction, fewer births, and substantial parental care [\(Hill,](#page-7-0) [1993; Kaplan et al., 2000\)](#page-7-0). Despite the species-general decelerated LH, existing research has pinpointed substantial individual differences in LH-related traits and their relations with environmental adversities. For instance, individuals raised in harsh or unpredictable environments were found to have earlier romantic relationships, more sexual partners, and more engagements in sexual activities [\(Belsky et al., 2012; Brum](#page-6-0)bach et al., 2009; Maranges & [Strickhouser, 2022; Simpson et al., 2012](#page-6-0)). In addition, aggression, risk-taking, impulsivity, and social deviance have also been found positively related to adverse childhood experiences ([Chang et al., 2019a; Griskevicius et al., 2013; Lu](#page-6-0) & Chang, 2019; [Mishra et al., 2017; Salas-Rodríguez et al., 2021](#page-6-0)). It has been discussed in the LH literature that harsh and unpredictable environments with too few or no cues of future opportunities would foster preferences for shortterm benefits and means to leverage social dominance (e.g., bullying; [Ellis et al., 2012; Frankenhuis et al., 2016\)](#page-6-0).

In addition to behavioral traits, LH researchers have paid increased attention to environmental effects on cognitive development. There is evidence showing that environmental adversity inversely predicted effortful control (e.g., inhibitory control; Warren & [Barnett, 2020](#page-8-0)) and the ability to delay gratification [\(Griskevicius et al., 2011; Martinez](#page-7-0) [et al., 2022\)](#page-7-0). [Wenner et al. \(2013\)](#page-8-0) found a positive relation of slow LH strategy with executive functioning, and that executive functioning mediated the relations between slow LH strategy with socially antagonistic attitudes and behaviors. A landmark work by Mittal and colleagues (2015) revealed that individuals with unpredictable childhoods exhibited poorer inhibitory control but enhanced attention shifting, suggesting a trade-off between cognitive functions. [Young et al. \(2018\)](#page-8-0)

uncovered similar trade-offs between aspects of working memory: environmental unpredictability is negatively related to memory retrieval and capacity, yet positively related to memory updating under conditions of uncertainty. The researchers reasonedthat these increased cognitive functions may aid in rapidly shifting among miscellaneous tasks, which would be adaptive in highly unpredictable environments. By reviewing these findings, [Ellis et al. \(2022\)](#page-6-0) posited that adversityadapted increases in certain abilities are concomitant with decreases in others. Given that decreased abilities may not adequately contribute to success in normative contexts, adapted abilities may instead be leveraged as "hidden talents" to promote success in these contexts.

Despite existing evidence and theorizing on the environmental calibration of cognitive and behavioral development, few LH studies have investigated the associations between cognitive functions and LH behavioral manifestations under adverse environmental conditions. We believe that understanding the links between environmentally calibrated cognitive and behavioral profiles is of great importance for obtaining a full picture of the adaptive cognitive-behavioral mechanism. This thus leads to our goal of exploring the role of cognitive functioning in the relation between early environmental adversity and LH behavioral manifestations.

1.2. Developmental plasticity and the Brain-Body Trade-Off

Resources allocated to growth and development are embodied in the enlargement of structural tissues (e.g., the brain, muscles, and other organic tissues) and improvement in their functioning (e.g., metabolic rate, immune function; [Del Giudice et al., 2015\)](#page-6-0). For humans, having a large brain may result in higher overall somatic energy expenditure, accelerated basal metabolic rates, and the evolution of higher body fat percentages for conserving energy ([Pontzer et al., 2016; Raichle](#page-7-0) & [Gusnard, 2002\)](#page-7-0). The human brain consumes roughly 20 % of the total energy while it only accounts for about 2 % of the total body weight (Attwell & [Laughlin, 2001\)](#page-6-0). The high energy demand of the brain could render it particularly susceptible to energy-allocation decisions [\(Raichle](#page-8-0) & [Gusnard, 2002; Watts et al., 2018](#page-8-0)), which are, in turn, sensitive to environmental cues, especially at early developmental stages.

Neuroplasticity, the ability of the neural systems to modify their structures and functions according to environmental inputs, is heightened during certain windows of time (also known as the *critical periods*; Hensch, 2004; Kolb & [Gibb, 2014; Reh et al., 2020](#page-7-0)). Within this developmental window, specific synaptic connections are strengthened and some others are pruned, encoding environmental-experiential stimuli and resulting in adapted neural organizations ([Nelson](#page-7-0) & [Gabard-Durnam, 2020](#page-7-0)). Given such experience-driven nature of cortical specialization, expectant early experiences that did not occur or occurred in atypical ways should lead to abnormal brain development (Nelson & [Gabard-Durnam, 2020\)](#page-7-0). Evidence shows that early environmental adversities (e.g., low SES, negative or stressful life events, family psychiatric history) are associated with lower grey matter volume in regions such as the medial temporal lobe, medial prefrontal cortex, anterior cingulate cortex, amygdala, hippocampus ([Ansell et al., 2012;](#page-6-0) [Pollok et al., 2022; Tyborowska et al., 2018; Walsh et al., 2014](#page-6-0)), as well as reduced white matter integrity ([Hanson et al., 2013](#page-7-0)). [Hodel et al.](#page-7-0) [\(2015\)](#page-7-0) found lower prefrontal and hippocampal volumes in children who were adopted from institutional care compared to their nonadopted counterparts. In terms of cognitive functioning, a body of research has unveiled robust positive associations between early adversities (e.g., institutionalized experiences, low SES, neglect, abuse) and deficits in executive functions such as inhibitory control, working memory, and cognitive flexibility in children ([Bos et al., 2009; Johnson](#page-6-0) [et al., 2021; Loman et al., 2013; Lund et al., 2020; McDermott et al.,](#page-6-0) [2012; Pollak et al., 2010\)](#page-6-0).

The *selfish* brain theory ([Peters et al., 2004\)](#page-7-0) posits that, owing to its high energetic consumption yet low energy storage capacity, the brain has evolved to prioritize its own energetic demand while competing for energy with other organs. This was supported by [Kuzawa et al.](#page-7-0)'s (2014) evidence that the ratios of the brain's glucose update to the body's resting metabolic rate (maintenance) and daily energy requirements (including daily maintenance, activity, and growth) are negatively associated with the growth rate of body weight, and such inverse relationship peaks around the age of 5 years. [Vandekar et al. \(2019\)](#page-8-0) replicated this finding and added that this brain-body trade-off may persist through early adolescence. In addition, some brain regions are particularly susceptible to this brain-body energetic trade-off. Evidence reveals that decreased glucose metabolism, blood flows, and cortical volumes in the prefrontal cortex are related to higher BMI ([Laurent et al.,](#page-7-0) 2020; Marqués-Iturria et al., 2013; Volkow et al., 2009; Willeumier [et al., 2011\)](#page-7-0). Given the coupling between the prefrontal regions and executive functions (Friedman & [Robbins, 2022; Menon](#page-7-0) & D'Esposito, [2022\)](#page-7-0), a growing body of research exhibited that increased BMI is associated with poorer inhibitory control, attention shifting, cognitive flexibility, and working memory (Blair et al., 2020; Favieri et al., 2019; [Laurent et al., 2020; Ronan et al., 2020](#page-6-0)).

One explanation for the inverse relationship between body weight and cognitive functioning is that excess fat disposition may result in mild inflammation in the hypothalamus and thus irregular hormone production, which may, in turn, alter neural organizations and functions (Kuzawa & [Blair, 2019\)](#page-7-0). The hypothalamus has been known for its sensitivity to stress as a key constituent of the hypothalamic–pituitary–adrenal (HPA) axis. Extended and repeated activation of the HPA axis and releasing of glucocorticoids and catecholamines (epinephrine, norepinephrine) in response to chronic stress may alter the structures of certain regions (e.g., the prefrontal cortex, hippocampus, amygdala) and related cognitive functions ([Chiang et al., 2015;](#page-6-0) [Girotti et al., 2018; Raymond et al., 2018](#page-6-0)). In addition to increasing the visceral accumulation of adipose tissue, chronically heightened glucocorticoids decrease insulin receptors (IRs) and hinder the expression and translocation of glucose transporters (e.g., GLUT-1, GLUT-3, GLUT-4), resulting in diminished insulin-induced glucose uptake, known as insulin resistance ([Batra et al., 2021](#page-6-0)). Such dysregulated insulin signaling alters neurogenesis and synaptogenesis, which leads to an altered cell population in the brain to form synaptic connections, particularly in main regions with IRs such as the prefrontal cortex, hypothalamus, amygdala, and hippocampus (Alberry & [Silveira, 2023](#page-6-0)). Understanding these neuroendocrine mechanisms should lead to hypotheses on the tripartite relations of early adversity, cognitive functioning, and body mass. A recent latent growth study on children found that deprivation is associated with higher BMI and poorer working memory, with working memory inversely associated with BMI (Farkas & [Jacquet, 2023\)](#page-7-0).

Although unexpectedly altered cortical structures and neuroendocrine circuitries could lead to unfavorable cognitive development and health consequences (e.g., metabolic and cardiovascular diseases), they could be construed as adaptive responses to environmental adversities. Enhanced cognitive functioning would be selected for in benign and predictable environments as it could enable individuals to excel in intraspecific competition. Nonetheless, excess investment in this capital is less likely to pay off in harsh and unpredictable environments if the developing individuals can even hardly make their reproductive stage. In this case, survival-sustaining functions would be prioritized instead. The "thrifty genotype" hypothesis proposes that human genetic predisposition for excess fat deposition was evolved to maintain survival through periods of resource scarcity. This was refined by the "drifty genotype" hypothesis that as predatory risk declined over time, humans were emancipated from the selection for leanness and agility, rendering the genetic predisposition for fat deposition less constrained; it was not eliminated but allowed to drift over the history of human evolution ([Higginson et al., 2016; Sellayah et al., 2014; Speakman, 2013](#page-7-0)).

Taken together, it should be reasonable to conceptually concatenate adversity-induced phenotypic variations with neural sensitivity to environmental cues. The neural systems encode cues of environmental adversities with an adaptively developed neural organization, which

allows basic cognitive functions to operate in an energy-sparing fashion. This organization should make the brain less energy-costly and sufficiently energetically compatible with increases in somatic energyconserving functions for survival. This experience-adapted neural organization may help explain why somatic energy reserve, embodied as fat deposition, could escalate to become a prioritized destination of energy allocation, given the evolved "selfish" nature of the brain. Certainly, this speculation calls for more in-depth considerations and thorough investigations.

1.3. The present study

The present research aims to explore the underlying cognitive processes of LH behavioral manifestations and the relations between early environmental adversity, cognitive functioning, and body mass status (i. e., BMI). To test these relationships, we conducted a longitudinal study using a cohort sample of 2,607 children and their caregivers from the Adolescent Brain Cognitive Development study (ABCDSM release 5.0; htt [ps://abcdstudy.org\)](https://abcdstudy.org). Based on extant evidence and theorizing, we hypothesized that environmental adversities experienced at an earlier time positively predict fast LH behavioral profiles (e.g., aggression, impulsivity, risk-taking) and negatively predict cognitive functions (e.g., attention and inhibitory control, working memory, cognitive processing speed) at a later time. Decreased cognitive functioning was expected to, at least, partially mediate the effect of environmental adversity on fast LH behavioral manifestations. In addition, we also expected early environmental adversities to predict higher BMI, and that BMI would be inversely correlated with cognitive functioning.

2. Method

2.1. Sample

The present research adopted tabulated assessment data from the ABCDSM study (ABCDSM release 5.0; [https://abcdstudy.org\)](https://abcdstudy.org) released by the National Institute of Mental Health Data Archive (NDA). The ethical review and approval of research protocol were issued by the central Institutional Review Board (IRB) at the University of California, San Diego, and local IRBs [\(Auchter et al., 2018](#page-6-0)). Data from three waves of data collection were used for the present study: the baseline collection, year-1 follow-up, and year-4 follow-up of the ABCDSM study. 2,925 subjects completed target measures over the selected timepoints. Our exclusion criteria involve: having had brain injury ($n = 56$); weighted BMI z scores are beyond \pm 3 (n = 184; WHO Multicentre Growth [Reference StudyGroup, 2006](#page-8-0)); median reaction times fall out of the allowable range (100 ms \sim 10,000 ms) or are greater than 3 SDs away from the mean by completing the Flanker Task ($n = 78$; Slotkin et al., [2012\)](#page-8-0). The final sample consists of 2,607 8- to 11-year-old children (female = 46.64 %; *M*age = 9.52 years, *SD* = 0.50; Non-Hispanic White $= 60.68$ %, Non-Hispanic Black = 8.63 %, Hispanic = 19.14 %, Asian = 2.42 %, and Others = 9.13 %) and their caregivers (female = 89.67 %; *M*age = 40.65 years, *SD* = 6.44; Non-Hispanic White = 67.51 %, Non-Hispanic Black = 8.68 %, Hispanic = 16.03 %, Asian = 3.37 %, and Others $= 4.41$ %) at the baseline. 86.27 % of the caregivers are the biological mothers of their youth, and 9.67 % are the biological fathers. 74.96 % of the caregivers are married, 12.15 % are separated or divorced, and 4.19 % are living with a partner. 4.06 % of the families make a total income less than \$12,000, 5.78 % make a total income between \$12,000 ~ 24,999, 13.58 % between \$25,000 ~ 49,999, 29.74 % between \$50,000 ~ 99,999, and 46.86 % greater than \$100,000.

2.2. Measures

Neighborhood Unsafety was reported by both children and their caregivers using a 5-point Likert questionnaire inquiring about their subjective experience of their living neighborhood (variable name noted as "Neighborhood Safety & Crime" in the ABCD data dictionary: [https](https://data-dict.abcdstudy.org/) [://data-dict.abcdstudy.org/?](https://data-dict.abcdstudy.org/)). Caregivers responded to "I feel safe walking in my neighborhood, day or night," "Violence is not a problem in my neighborhood," and "My neighborhood is safe from crime" with 1 $=$ "Strongly Disagree" through $5 =$ "Strongly Agree." Children responded to "My neighborhood is safe from crime" on the same scale. Items were reverse-coded, and child- and caregiver-reported scores were averaged to derive a total score. The data used was collected in Time 1 of the present study (the baseline collection of the $\mathrm{ABCD}^\mathrm{SM}$ study). The Cronbach's α of this measure is 0.85.

Adverse Life Events were measured using the Adverse Life Event Scale ([Tiet et al., 1998\)](#page-8-0), a 25-item checklist inquiring about children's past adverse experiences that they felt having few or no control ("Life Events"). The data used was collected at Time 2 of the present study (1 year follow-up of the ABCDSM study). Both children and caregivers were presented a list of 25 events (e.g., "Saw crime or incident," "One parent was away from home more often," "Negative change in parent's financial situation"). For each, they were asked to respond whether the child had experienced the event $(1 = "Yes," 0 = "No")$, whether the experience was generally positive or negative $(1 = "Mostly good," 2 = "Mostly"$ bad"), and how much this experience had affected the child $(0 = "Not at$ all," $1 = "A little," 2 = "Some," 3 = "A lot".$ Only experienced events that were rated "Mostly bad" and affecting the child "A little", "Some," or "A lot" were identified as adverse events and summed up to generate a total score, ranging from 0 to 25. Because the total score was derived based on the number of the adverse events, this measure was not examined for internal consistency reliability. Child- and caregiverreported scores were averaged for analysis.

Socioeconomic Disadvantage was employed as a household-level material deprivation based on household combined income reported by caregivers at Time 1 of the present study ("Demographics"). The sources of income involve wages, rent from properties, social security and benefits, compensations, and endowment from relatives and other relations. Caregivers were asked to respond "What is your total combined family income for the past 12 months" with $1 =$ "Less than \$12,000," $2 =$ " $$12,000 \sim 24,999$," $3 =$ " $$25,000 \sim 49,999$," $4 =$ " $$50,000 \sim 99,999$ ", and 5 = "Equal to or greater than \$100,000." Reported scores were reversed, such that higher scores indicate higher levels of economic disadvantage.

Area Poverty was involved as a proxy of neighborhood-level material deprivation ("Area Deprivation Index"). It was extracted from the poverty dimension of the area deprivation index, a census tract measure derived from the American Community Survey ([Fan et al., 2021\)](#page-6-0). As provided by the ABCDSM study, the scores used in the present study were derived from the percentage of households below the state poverty level reported at Time 1 of the present study. Higher scores reflect higher neighborhood socioeconomic disadvantage. Reported scores were standardized prior to formal statistical analyses.

Body Mass Index (BMI) was acquired from the anthropometric measures of children's height (in centimeters) and weight (in kilograms) at Time 3 of the present study (4-year follow-up of the ABCDSM study; "Anthropometrics"). Calculated BMI were corrected by age and gender according to the World Health Organization (WHO) growth reference data for 5–19 years [\(https://www.who.int/tools/growth-reference-dat](https://www.who.int/tools/growth-reference-data-for-5to19-years) [a-for-5to19-years](https://www.who.int/tools/growth-reference-data-for-5to19-years)) and converted into z scores following the recommended algorithms for z score computation and cutoffs ([WHO Multi](#page-8-0)[centre Growth Reference StudyGroup, 2006\)](#page-8-0).

Cognitive Functioning was indexed by children's performances on the Flanker Inhibitory Control and Attention Test ("Flanker Task"), List Sorting Working Memory Test, and Pattern Comparison Processing Speed Test ("NIH Toolbox") drawn from the National Institutes of Health (NIH) Toolbox Cognitive Battery ([Weintraub et al., 2013\)](#page-8-0). The data used was collected at Time 3 of the present study. The total score of the List Sorting test is the number of visual stimuli (e.g., foods or animals) correctly recalled and sequenced in both 1-List (one type of stimuli) and 2-List (two types of stimuli), ranging from 0 to 26. The total score of the

Pattern Comparison test reflects the total number of pictures correctly discerned within 90 s, ranging from 0 to 130. The scoring of the Flanker test was based on a two-vector method – accuracy and reaction time. The accuracy vector denotes the number of trials that participants answered correctly (20 trials in total, 12 congruent and 8 incongruent). Whereas, the reaction time vector reflects the participant's median reaction time over the incongruent trials. Score calculations for both vectors followed the NIH Toolbox scoring and interpretation guide ([Slotkin et al., 2012](#page-8-0)).

Aggression was registered using the Aggression subscale of the Child Behavior Checklist (CBCL, 6–18 version; Achenbach & [Rescorla, 2001](#page-6-0); "Child Behavior Checklist"). The CBCL Aggression subscale consists of twenty items (e.g., "Physically attacks people," "Threatens other people, "Screams a lot," "Stubborn, sullen, or irritable"), of which each is rated on a 3-point scale ($0 =$ "Not true," $1 =$ "Sometimes true," $2 =$ "Often true"). Item scores were averaged to generate a total score. Higher total scores indicate higher aggression. The data selected was collected at Time 3 of the present study. The internal consistency reliability estimate of this measure is 0.90.

Impulsivity was measured using the Short UPPS-P Impulsive Behavior Scale (SUPPS-P; [Cyders et al., 2014](#page-6-0)), a 20-item self-report questionnaire that covers five subscales: positive urgency, negative urgency, lack of perseverance, lack of premeditation, and sensation seeking ("UPPS-P Impulsive Behavior Scale"). Children responded to items (e.g., "When I am upset I often act without thinking," "I tend to lose control when I am in a great mood") with $1 =$ "Very much like me" through $4 =$ "Not at all like me." Item scores were reverse-coded and averaged to generate a total score, such that higher total scores indicate higher impulsivity. The data used was collected at Time 3 of the present study. The internal consistency estimates of the five subscales range from 0.69 to 0.88. The internal consistency estimate of the complete measure is 0.87.

Risk-taking was indexed based on children's performance on the Game of Dice Task (GDT; "Game of Dice"). The GDT is a computerized gambling task designed to assess participants' risk-taking propensity. Before rolling the virtual die, participants were asked to guess the number that would appear on the die (1 to 6) and to place a bet on either a single die face or a combination of two, three, or four die faces. The fewer the dice faces selected, the higher the reward for winning. Bets on 3- or 4-face combinations were coded as safe bets, whereas bets on a single face or 2-face combinations were coded as risky bets, aligned with their probabilities of winning. Children in the present study completed a total of 18 game trials (18 bets in total). Their risk-taking propensity was derived through dividing the number of times they made risky bets by the total number of bets placed. A higher total score indicates higher risk-taking. The data collected at Time 3 of the present study was used for analysis.

2.3. Statistical Analysis

IBM SPSS 27 and Mplus 8.0 were employed for descriptive statistics and structural equation modeling (SEM). A zero-order bivariate correlation analysis was performed to examine the associations between all variables. SEM was used to create latent variables for early environmental adversity, cognitive functioning, and fast LH behavioral profile. An exploratory factor analysis was performed to screen indicator variables with a standardized loading less than 0.30 or being loaded on more than one factor ([Hair et al., 2006\)](#page-7-0). Indicator variables selected were entered into a measurement model with full information maximum likelihood estimation applied to account for missing data. Model fit was assessed using recommended cut-off values, including the ratio of chisquare to degrees of freedom $(\chi^2/df < 10;$ [Kline, 1998](#page-7-0)), comparative fit index (CFI \geq 0.90), Tucker-Lewis index (TLI \geq 0.90; Marsh et al., [1988\)](#page-7-0), root mean squared error of approximation (RMSEA \leq 0.08; Browne & [Cudeck, 1993\)](#page-6-0), and standardized root mean square residual (SRMR \leq 0.08; Hu & [Bentler, 1999](#page-7-0)). Modification indices were consulted for model fit improvement if the prospective model did not fulfill the recommended cutoffs. BMI was entered when establishing the final

mediation structural model. This model involves early environmental adversity as the independent variable, cognitive functioning and BMI as the mediator variables, and fast LH behavioral profile as the dependent variable. Early environmental adversity consists of four indicator variables: neighborhood unsafety, adverse life events, socioeconomic disadvantage, and area poverty; cognitive functioning is comprised of three indicator variables: attention and inhibitory control, working memory, and cognitive processing speed; fast LH behavioral profile includes three indicator variables: aggression, impulsivity, and risktaking. The mediating effects of cognitive functioning and BMI on the relationship between early environmental adversity and fast LH behavioral profile were examined using a bias-corrected bootstrap analysis with 5,000 resamples, with 95 % confidence intervals (CI) estimated to evaluate the statistical significances of the total, direct, and indirect effects.

3. Results

Table 1 displays the means, standard deviations, and zero-order Pearson bivariate correlations of the indicator variables involved in the present study. In brief, the correlations are mostly statistically significant and consistent with the LH hypotheses and our expectations. The observed indicators of each latent variable are positively correlated with one another. Specifically, indicators of early environmental adversity, such as neighborhood unsafety, adverse life events, socioeconomic disadvantage, and area poverty are negatively correlated with inhibitory control, working memory, and cognitive processing speed and mostly positively correlated with aggression, impulsivity, risktaking, and BMI. BMI is negatively correlated with all cognitive functions and positively correlated with aggression, impulsivity, and risktaking.

The measurement for early environmental adversity, cognitive functioning, and fast LH behavioral profile indicates an acceptable model fit: $\chi^2/df = 4.52$, CFI = 0.92, TLI = 0.89, RMSEA = 0.047, SRMR $= 0.041$. All indicator variables have a standardized factor loading greater than 0.30: $0.31 \sim 0.68$ for early environmental adversity, 0.36 \sim 0.63 for cognitive functioning, and 0.31 \sim 0.47 for fast LH behavioral profile. The mediation structural model is shown in [Fig. 1](#page-5-0). The goodness of fit statistics suggest an acceptable model fit: $\chi^2/df = 6.16$, CFI = 0.91, $TLI = 0.88$, RMSEA = 0.044, SRMR = 0.037. All latent variables fulfill the standardized threshold of 0.30: $0.34 \sim 0.69$ for early environmental adversity, $0.38 \sim 0.61$ for cognitive functioning, and $0.32 \sim 0.42$ for fast LH behavioral profile. All parameter estimates are statistically significant and aligned with our predictions. Specifically, early environmental adversity negatively predicts cognitive functioning (β = -0.46, *p* < 001) and positively predicts fast LH behavioral profile ($β = 0.17, p < 0.01$) and

Table 1

Means, Standard Deviations, and Zero-Order Bivariate Correlations of Indicators.

BMI ($β = 0.24$, $p < .001$). Cognitive functioning negatively predicts fast LH behavioral profile ($β = -0.14$, $p < .05$) and BMI positively predicts fast LH behavioral profile ($β = 0.16, p < .001$). Meanwhile, cognitive functioning covaries with BMI at $β = -0.08$, $p < 0.05$. A bias-corrected bootstrap analysis with 5,000 resamples was performed to investigate the mediating effects. The total indirect effect is statistically significant ($\beta =$ 0.10, 95 % CI = $[0.041, 0.178]$, accounting for 37.27 % of the total effect $[\beta_{indirect} = 0.10, \beta_{direct} = 0.17;$ Percentage = $\beta_{indirect}$ / ($\beta_{indirect}$ + β_{direct}) × 100 %]. The total indirect effect consists of the indirect effects of cognitive functioning and BMI. The indirect effect of cognitive functioning is significant ($\beta_{\text{cognitive}} = 0.063$, CI = [0.005, 0.138]), accounting for 23.25 % of the total effect [Percentage = $β_{cognitive} / (β_{indirect} + β_{direct})$ \times 100 %]. The indirect effect of BMI is also significant ($\beta_{\text{BMI}} = 0.038$, CI $=$ [0.019, 0.059]), accounting for 14.02 % of the total effect [Percent $age = \beta_{BMI} / (\beta_{indirect} + \beta_{direct}) \times 100 \%$.

4. Discussion

Extant human LH research has extensively investigated individual variations in cognitive and behavioral development as a function of environmental harshness and unpredictability. However, the association between environmentally calibrated cognitive and behavioral profiles remains underexplored. The first aim of the present study is to explore this association in an attempt to understand the experience-adapted cognitive-behavioral mechanism. In addition, existing evidence of the relations between brain metabolic rate, cortical structures, and related cognitive functions with body mass inspired us to investigate the tripartite relations of early environmental adversity, cognitive functioning, and body mass. We tested a series of hypotheses and obtained several major findings. Each is discussed as follows.

Aligned with our hypothesis, the SEM analysis with cognitive functioning as a mediator suggests that early environmental adversity positively predicted fast LH behavioral profile, partially through the mediation of cognitive functioning. That environmental adversity had distinct effects on fast LH behavioral versus cognitive profiles is also consistent with preceding evidence (e.g., Chang et al., 2019; [Lu](#page-7-0) & [Chang, 2019; Mittal et al., 2015; Warren](#page-7-0) & Barnett, 2020). The finding that cognitive functioning mediated the relation of environmental adversity with fast LH behavioral profile adds to the literature by empirically demonstrating the experience-adapted cognitive-behavioral mechanism. This cognitive-behavioral mechanism could be construed as adaptive. As predicted by the LH framework, the merits of fast LH behavioral traits are most pronounced in harsh and/or unpredictable environments. When individuals have little control over environmental contingencies, it would be adaptive to focus on opportunistic, immediate rewards rather than long-term benefits. As aforementioned, such

Note. * p *<*.05, ** p *<*.01, *** p *<*.001.

Fig. 1. The influence of early environmental adversity on fast LH behavioral profile through the mediation of cognitive functioning and BMI.

reward preference has its root in present-orientation, which has been theorized as an adaptation to unpredictable environmental conditions ([Frankenhuis et al., 2016\)](#page-7-0). With present-orientation being fostered in environments with immediate survival challenges, such abilities as effortful control, deliberation, delaying gratification, or planning that focus on future opportunities would be compromised, accordingly. Simply put, in an environment where accelerated paces of life and traits are favored, cognitive "brakes" would become less essential for fitness and thus be deprioritized for energy allocation. This leads to our reasoning that the net energy budget may be relaxed as the brain becomes less energy-consuming by perhaps only minimally developing certain structures and functions. With less burden on the energy budget, energy-allocation pathways could be finely tuned to the demands of functions that more directly address immediate survival challenges.

This reasoning is underlaid by an assumption that cognitive deficits, along with altered cortical structures, might be the consequences of the adversity-induced calibration of energy-allocation pathways. Following the LH principle that energy invested in one function at a time cannot be used for another, we conjectured that holding the net energy budget constant, if energy was not allocated to the expected destination, it must have been allocated elsewhere. We speculated that budget energy that was reserved for the brain was actually diverted to organs for energy conservation (i.e., fat deposition) in response to immediate survival challenges. This speculation is, first, supported by the finding that early environmental adversity positively predicted BMI, which, in turn, mediated the relation between early environmental adversity and fast LH behavioral profiles, suggesting that increased body mass may be a somatic signature of accelerated LH [\(Maner et al., 2017\)](#page-7-0). There is also evidence unveiling that higher BMI is associated with earlier onset of puberty ([Crocker et al., 2014; Kaplowitz et al., 2001; Styne, 2004\)](#page-6-0), more directly aligning increased body mass to fast LH traits. Second, we found that early environmental adversity predicted decreased cognitive functioning and higher BMI, while cognitive functioning and BMI were negatively correlated. This inverse association implies a potential tradeoff between cognitive development and somatic energy reserve, suggesting an adversity-adapted energy-allocation pathways. There is reason to believe that such evolved plasticity of energy-allocation pathway may aid in equilibrating fitness across various environmental conditions. For example, in benign and predictable environments, it would be favorable for juveniles to exploit their energy budget by expending it all on somatic growth and brain development, as thus maximizing their competitiveness over current or anticipated contests for resources. By contrast, in adverse environments where survival becomes a prioritized task of fitness, it would be favorable to conserve

energy (i.e., putting the unused budget in their own pocket), as thus maximizing their chance of making their future reproductive cycles.

The present research is subject to several limitations. First, the present research did not account for the distinct effects of different types of environmental adversities. Developmental scientists have increasingly focused on dimensional approaches to differentiate the influences of different adversities. The dimensional model of adversity and psychopathology ([McLaughlin et al., 2014; Sheridan](#page-7-0) & McLaughlin, 2014), for instance, identifies threat and deprivation as two axes of adversity, denoting the *presence* of unexpectant environmental inputs and the *absence* of expectant inputs, respectively. Empirical evidence exhibits a robust association between deprivation (e.g., neglect, parental absence, low SES) and executive functions (e.g., inhibitory control, working memory), but rather mixed relations between threat (e.g., domestic violence, abuse) and these functions (Johnson et al., 2021; Schäfer et al., [2023; Sheridan et al., 2017\)](#page-7-0). Given the debate regarding the problem of statistical multicollinearity upon the co-occurrence of threat and deprivation, [McLaughlin et al. \(2021\)](#page-7-0) suggested multiple approaches to address this issue (e.g., latent class analysis, network models). Owing to limited methodology and data availability, however, the present research treated environmental adversity as a single construct. Nevertheless, we recognize the merits of dimensional models and advocate for their application in future research. Second, the present study did not account for potential genetic effects due to limited available genetic data in current sample. However, genetic components could be particularly relevant to this study as they are estimated to account for roughly 40–70 % of the variation in body weight status [\(Bouchard, 2021; Loos](#page-6-0) & Yeo, [2022\)](#page-6-0). We suggest that future research may employ samples of twins and siblings to discern between environmental versus genetic effects and explore their potential interactions. Third, it is important to note that our use of BMI as a proxy measure of somatic energy reserve must be interpreted with caution. It should not be treated as a direct measure of fatness because it does not differentiate between fat mass versus lean mass (e.g., bone, muscle), although BMI has been found moderately to highly correlated with direct measures of fatness (e.g., body fat percentage, fat mass index, visceral and subcutaneous adipose tissue; [Camhi et al., 2011; Flegal et al., 2009; Jeong et al., 2023; Ranasinghe](#page-6-0) [et al., 2013](#page-6-0)), with higher BMI more strongly associated with these measures ([Chung, 2015\)](#page-6-0). Future research may consider adopting measures that directly reflect body fatness, such as body fat percentage, fat mass index (in relation to lean/fat-free mass index), adipose tissue area, or incorporating other anthropometric measures such as the waist-toheight ratio in addition to BMI.

Despite these limitations, the present research empirically

demonstrated the experience-adapted cognitive-behavioral mechanism and the trade-off between cognitive and somatic development under the influence of early environmental adversity. These findings unveiled the possibility that energy-allocation trade-offs may not only exist between the well-theorized major components of fitness but could be further specified between aspects of a particular component, thereby adding to the existing knowledge of LH trade-off and developmental plasticity.

Ethics approval

The ABCD research sites are subject to the ethical review and approval by the central Institutional Review Board (IRB) at the University of California, San Diego, and IRBs at local institutions.

Consent to publish

Informed consent to publish was not necessary because all data used for the present study came from the National Institute of Mental Health Data Archive.

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CRediT authorship contribution statement

Anting Yang: Writing – original draft, Methodology, Investigation, Data curation, Conceptualization. **Hui Jing Lu:** Writing – review & editing, Visualization, Validation, Methodology, Investigation, Formal analysis. **Lei Chang:** Writing – review & editing, Supervision, Methodology, Investigation, Conceptualization.

Declaration of competing interest

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

Data availability

The authors do not have permission to share data.

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