ORIGINAL RESEARCH

Infant effortful control predicts BMI trajectories from infancy to adolescence

LillyBelle K. Deer¹ | Jenalee R. Doom¹ | Kylie K. Harrall² | Deborah H. Glueck^{2,3} | Laura M. Glynn⁴ | Curt A. Sandman⁵ | Elysia Poggi Davis^{1,5}

1 Department of Psychology, University of Denver, Denver, Colorado, USA

² Lifecourse Epidemiology of Adiposity and Diabetes (LEAD) Center, University of Colorado, Anschutz Medical Campus, Aurora, Colorado, USA

3 Department of Pediatrics, University of Colorado, Anschutz Medical Campus, Aurora, Colorado, USA

4 Department of Psychology, Chapman University, Orange, California, USA

5 Department of Psychiatry & Human Behavior, University of California, Irvine, California, USA

Correspondence

LillyBelle K. Deer, Psychology Department, University of Denver, 2155 S. Race Street, Denver, CO 80210, USA. Email: lillybelle.deer@du.edu

Funding information

National Heart, Lung, and Blood Institute, Grant/Award Numbers: F32HL165844, K01HL143159, R01HL155744; National Institute of General Medical Sciences, Grant/Award Number: 5R01GM121081-08; National Institutes of Health, Grant/Award Numbers: HD-28413, HD-40967, HD-51852, HL-155744, MH-96889, NS-41298; National Institute of Mental Health, Grant/Award Number: R01MH109662

Summary

Background: Effortful control, or the regulation of thoughts and behaviour, is a potential target for preventing childhood obesity.

Objectives: To assess effortful control in infancy through late childhood as a predictor of repeated measures of body mass index (BMI) from infancy through adolescence, and to examine whether sex moderates the associations.

Methods: Maternal report of offspring effortful control and measurements of child BMI were obtained at 7 and 8 time points respectively from 191 gestational parent/ child dyads from infancy through adolescence. General linear mixed models were used.

Results: Effortful control at 6 months predicted BMI trajectories from infancy through adolescence, $F(5,338) = 2.75$, $p = 0.03$. Further, when effortful control at other timepoints were included in the model, they added no additional explanatory value. Sex moderated the association between 6-month effortful control and BMI, $F(4, 338) = 2.59$, $p = 0.03$, with poorer infant effortful control predicting higher BMI in early childhood for girls, and more rapid increases in BMI in early adolescence for boys. Conclusions: Effortful control in infancy was associated with BMI over time. Specifically, poor effortful control during infancy was associated with higher BMI in childhood and adolescence. These findings support the argument that infancy may be a sensitive window for the development of later obesity.

KEYWORDS

BMI, effortful control, obesity, sex differences

1 | INTRODUCTION

Obesity in childhood and adolescence is increasingly prevalent worldwide, from 4% of children in 1975 to 18% in 2016. 1,2 1,2 1,2 Paediatric obesity is a robust predictor of poor physical and mental health across the lifespan.^{[3,4](#page-7-0)} The Developmental Origins of Health and Disease

Abbreviations: BMI, body mass index; CDC, Centers for Disease Control and Prevention; DOHaD, developmental origins of health and disease; HPA Axis, hypothalamic–pituitary– adrenal axis; INR, income-to-needs ratio; PDS, pubertal development scale.

(DOHaD) hypothesis posits that early life factors shape physiological systems (e.g., HPA axis, metabolic system) and behaviour to impact health throughout the lifespan, $5-7$ $5-7$ and that these early factors may have sex-specific effects. $8,9$ The current study examines the longitudinal association between effortful control and body mass index (BMI) from infancy through adolescence, aiming to identify sensitive periods when effortful control most robustly predicts BMI.

Infancy is a sensitive period during which adult obesity may be programmed through biological and behavioural mechanisms, such that early experience is embedded to impact health. 10 To date, most 2 of 10 WILEY Pediatric DEER ET AL.

of the obesity literature has focused on contributors to BMI that emerge in childhood and adolescence, and has shown that childhood and adolescent BMI predicts BMI through adulthood.^{11,12} However, it is likely that vulnerability to obesity is developed even earlier in life .^{7,13,14} There are several lines of evidence that support this hypothesis. First, the fetal programming epidemiology literature has documented that prenatal factors, including fetal growth and birthweight, are associated with obesity throughout the lifespan.^{[15,16](#page-8-0)} Second, BMI among children who later develop obesity begins to differ from children with normal weight in early childhood, indicating that processes influencing BMI likely originate even earlier. 17 Third, rapid increases in BMI during infancy (birth to 1-year), toddlerhood (1–3 years) and early childhood (3–5 years) are predictive of obesity in adolescence and adulthood. $18,19$ Finally, obesity may become largely intractable by childhood, with small-to-null effects of interventions on changing BMI in childhood and adolescence. $20,21$ As meta-analytic analyses of interventions conducted between 2 and 18 years of age suggest that these later interventions are minimally effective, $20,21$ and because infancy may be a sensitive period for the development of obesity, 10 identifying early-life factors associated with obesity may provide more promising targets for prevention and intervention.

Poor effortful control has been identified as a risk factor for the development of obesity. Effortful control is the effortful regulation of thoughts and behaviour, including attention, planning future actions and inhibiting impulsive behaviours. 22 The precursors of effortful control, such as orienting and regulation of attention, emerge in infancy. $22,23$ Effortful control continues to develop rapidly through childhood and adolescence into adulthood, with the skills involved becoming more complex as children age and the focus of effortful control shifting from external sources (i.e., caregivers regulating child behaviour) to internal. $22-24$ Effortful control is influenced by early experiences, including parenting and contextual factors. 24 Effortful control has been theorized to impact obesity through a multitude of pathways, including shaping neural systems (e.g., prefrontal cortex, nucleus accumbuns) and other physiological systems (e.g., inflammation, HPA axis), $25-31$ that regulate eating behaviours that impact subsequent BMI such as satiety and sensitivity to food cues.^{28,32,33} Meta-analyses of cross-sectional studies of children ages 2–21 years document that poorer effortful control is associated with obesity and overweight.^{[34,35](#page-8-0)} These cross-sectional studies provide important evidence for the link between effortful control and BMI, but longitudinal strategies are needed to understand the developmental course of this relation and whether effortful control may be a predictor of later obesity.

Only a few studies have examined the longitudinal relation between effortful control in childhood and BMI through early adolescence. These studies show that poor effortful control in preschool, the earliest childhood timepoint investigated, predicts higher BMI in adolescence. $36-38$ $36-38$ Only one study evaluating the role of effortful control and BMI probed effortful control during infancy. This study found that poorer effortful control at 1 year of age predicted higher odds of obesity at 6-years in boys but not girls.³⁹ This study included only one outcome assessment at 6-years, so the longer-term effects of poor infant effortful control are unknown. Additionally, the existing literature focuses on the difference in BMI over 2 timepoints, rather than

examining trajectories, which may preclude the examination of patterns over development. Thus, the cross-sectional evidence, combined with the few longitudinal studies, suggest that early life effortful control may be an important risk factor for higher BMI over time.

An additional gap in the literature is that despite strong evidence for sex differences in BMI over time, 40 and in sex-specific responses to early experiences, 8.9 sex differences in the association between effortful control and BMI only have been examined in a few studies, with mixed findings. $36-39$ $36-39$ This small literature provides a signal that there might be sex differences in the relation between early effortful control and later BMI, but the evidence is not consistent. More studies are needed to better understand potential sex differences.

The current longitudinal study examined the association between effortful control in infancy through early adolescence and BMI trajectories (repeated measures of BMI over time) from infancy to adolescence. Study aims were to (1) assess whether effortful control was associated with BMI measures from infancy through adolescence, and specifically, whether effortful control in infancy predicted BMI, and (2) to evaluate sex differences in these associations.

2 | METHODS

2.1 | Study overview

Figure [1](#page-2-0) demonstrates the assessment timepoints and primary constructs included in the current study. All research assessments were conducted in a family friendly research laboratory.

2.2 | Participants

The current study utilized a dataset where pregnant individuals were enrolled. Participants included 191 gestational parents and their children (44.5% female). Participants were recruited by a research nurse during the prenatal period from a large medical centre in Orange, California to participate in an observational longitudinal study assessing early life influences on development.

Demographic information was obtained via parental report. For race/ethnicity, parents identified 44.7% of children as non-Latinx White, 1.8% as non-Latinx Black, 6.1% non-Latinx Asian, 17.5% Multiracial/Multiethnic and 29.8% Latinx. Other demographic information can be found in Table [1](#page-2-0). Recruitment occurred between 2002 and 2007 during the first trimester of pregnancy. Pregnant individuals were approached and invited to participate in the study if they were English-speaking, non-smokers, over 18 years of age, with a singleton pregnancy and had no evidence of drug or alcohol use during pregnancy. A total of 227 participants were initially recruited during pregnancy. Participants in the current analyses were the 191 dyads who were born after 34 gestational weeks, participated in the postnatal assessments, and had effortful control and anthropometric measurements when the child was 6 months of age.

Ethical approval for the study was obtained from the Institutional Review Board for Protection of Human Subjects at the University of

 $\overline{\text{DeER et al.}}$ FW II FV 3 of 10

FIGURE 1 Timeline of the study. In the figure, BMI, body mass index; M and SD are age in years at measurement.

TABLE 1 Demographic characteristics.

Abbreviations: INR, income-to-needs ratio; PDS, pubertal developmental scale.

TABLE 2 Effortful control measures.

California, Irvine. Each gestational parent provided written and informed consent for herself and her child. Children provided written assent starting at the 9.5-year visit. Further recruitment procedures for the cohort are described elsewhere. 41 In order to keep families engaged in the study, consistent contact was maintained with families including sending birthday cards and newsletters. Further, long-term contact information was collected in case researchers could not reach the participant.

2.3 | Measures

Effortful control. Effortful control was assessed via maternal report on the effortful control scale from the Rothbart Temperament Questionnaires at the 6-month, 1-year, 2-year, 5-year, 6.5-year, 9.5-year and 11.5-year visits (see Table 2). The effortful control scale of this measure assesses individual differences in reactivity and selfregulation across development 42 42 42 by assessing assesses a child's typical response pattern to a variety of scenarios assessing components of effortful control. The age appropriate version of this measure was used to characterize effortful control at each time point.⁴³⁻⁴⁵ The questionnaires at each time point assess or characterize aspects of effortful control such as attentional focusing and inhibitory control through asking about specific, age-appropriate behaviours in concrete situations (see Table 2).

The underlying constructs of effortful control in the Rothbart assessments are consistent across assessment ages, and is operationalized so that items assessment are developmentally appropriate. In the

4 of 10 WILEY Pediatric DEER ET AL.

Infant Behaviour Questionnaire, which is used at the 6-month and 1-year timepoints, effortful control is measured using the duration of orienting, soothability, low-intensity pleasure and cuddliness constructs. In the Early Childhood Behaviour Questionnaire, which is used at the 2-year assessment, inhibitory control, attention shifting, attention focusing, low-intensity pleasure and cuddliness are used. In the Temperament in Middle Childhood Questionnaire, which is used at the 5-year, 6.5-year and 9.5 year assessments, effortful control is measured using the inhibitory control, attention focusing, activation control, low-intensity pleasure and perceptual sensitivity constructs. Lastly, in the Early Adolescent Temperament Questionnaire, which is used at the 11.5-year assessment, effortful control is measured using the inhibitory control, attention focusing and activation control constructs.

This measure has strong internal reliability (consistency across items), external reliability (stability over time and across raters) and external validity (consistency with observational data).⁴⁶ These measures were completed by gestational parents at the time of the visit. Gestational parents rated their infant/child using a Likert scale (higher scores indicate better effortful control) on each scenario. The effortful control score was calculated as an average of the subscales scores, consistent with the standardized scoring of this construct, 42 and then standardized to be on the same scale across ages. The Cronbach's alpha in the current study ranged from 0.88 to 0.91 across timepoints.

Body mass index (BMI). Weight and length or height were measured by trained research assistants at each of the eight visits to calculate BMI. Length was measured with children in a supine position at the 6-month, 1-year and 2-year visit while standing height was measured at the subsequent visits. Weight was measured on a digital scale. Measurements were taken once without heavy clothing or shoes. BMI was calculated using the standard formula at each time point (kg/m²). Raw BMI was used in the analyses in concordance with previous research.^{[47](#page-8-0)}

Sex at birth. Sex was collected from obstetric and neonatal medical records.

Covariates. Child birthweight, income-to-needs ratio (INR) at the 6-month visit, breastfeeding status at 6 months of age and pubertal status were assessed as covariates based on the existing literature (see supplement). These covariates were included in the final model if they were correlated $p < 0.10$ with the primary predictor: effortful control at 6 months of age. Birthweight was assessed via birth records. Gestational parents reported their income and family size when their child was 6 months of age. INR was calculated by dividing their total family income by the federal poverty threshold for a family of their size in the year the data were collected. At 6 months, gestational parents reported whether or not their child was still being breastfed. Lastly, gestational parents and children completed the pubertal developmental scale (PDS) at the 9.5-year, 11.5-year and 13-year visits, which is a well-validated measure of pubertal status.^{48,49} Gestational parent report was used on the PDS for children younger than 12 years of age while child selfreport was used for children older than 12 years of age. This was done as prior work documents that, compared with clinical measurements such as Tanner staging, parents are more accurate at reporting pubertal

development before age 12, while children become more accurate at rating their own development following age $12^{50,51}$ Birthweight, pubertal stage and breastfeeding status at 6 months were not included in the final model because they did not meet the aforementioned correlation criteria ($ps = 0.18 - 0.87$; Table S1). INR ($r = 0.13$, $p = 0.07$) met covariate criteria and therefore was included in the final model.

2.4 | Data analysis plan

Descriptive analyses. Descriptive analyses were performed using means and standard deviations for continuous variables and frequencies and percentages for categorical variables (see Table [1](#page-2-0) and Table S1). Correlations among the major variables of interest are reported in Table S2. The percentage of participants with present (i.e., non-missing data) at each measurement was assessed. The correlation between missingness and effortful control at 6 months, sex, and the covariate INR was also assessed.

The majority of children (58%) completed at least five visits (23.4% completed all eight visits, and 85% completed at least two visits). Some missingness was due to the structure of the study resulting in not all participants being eligible for all visits. Timing of grant funding meant that some participants missed age windows for assessments, resulting in them missing visits. All data that participants provided was used in the current analyses. The general linear mixed model used in the analyses allows estimation and inference for participants with complete as well as missing data.⁵² Missing data were more common at the end of the study than the beginning. Missingness was not associated with effortful control at 6 months ($p = 0.08$) or sex ($p = 0.67$). Missingness was significantly associated with INR at 6 months ($p = 0.02$), such that participants who had a lower INR were more likely to be missing data.

2.5 | Hypothesis testing

Effortful control and BMI. To examine the role of effortful control in infancy and whether effortful control at additional time points increased explanatory power, a planned sequence of hypothesis tests was conducted (see Table [3](#page-4-0)). General linear mixed models assessed the associations between repeated (6 months, 1 year, 2 years, 5 years, 6.5 years, 9.5 years and 11.5 years of age) or time-period-specific (6 months) measures of effortful control and BMI trajectories. This approach was used as it is optimal for applying a hypothesis-driven approach to determine the most parsimonious, best-fitting model. A random intercept for each participant and a random slope for age with an unstructured covariance between the random effects were fit to account for within-participant correlation between repeated measurements and the increase in variance of BMI across time.

It is known that BMI has a roughly quadratic pattern across the age range specified. 53 The typical BMI trajectory consists of an increase from birth to about 6 months to the 'infancy peak', followed by a gradual decrease until about 5 years of age, where the 'adiposity

TABLE 3 Summary of models tested.

rebound' occurs and BMI again increases through adolescence.^{[53](#page-9-0)} The linear and quadratic terms in age were added to the model as precision covariates, to allow better discernment of the association between effortful control and BMI. Hypotheses were tested using the mixed model F statistic (Wald statistic) using Kenward–Roger degrees of freedom.⁵⁴ Significance was assessed at a 0.05 Type I error rate for each of a series of planned hypothesis tests.

Three models were compared using planned hypothesis tests. These models differed in the effortful control measures included as predictors. The sequence of models used the multiple design matrix approach advocated by Srivastava, 55 which allows a different set of predictors for the outcome at each time period. The sequence of hypothesis tests compared three nested models which are referred to as: the 'early-life model', 'all-times-before' model and the 'immediately-before' model. The sequence of hypothesis tests identified which of the three hypothesized models best fit the data. Equations used to estimate the three models are provided in the supplement (see Supplement Analytic Strategy Sections [1](#page-0-0) and [2\)](#page-1-0).

The 'early-life' model contained, as a predictor of each measure of BMI, only the effortful control measure taken at 6 months of age. The 'immediately-before' model contained, as a predictor of the BMI measure at each age (t), only the measurement of effortful control taken immediately before $(t-1)$. For example, to predict BMI at 6.5 years of age, only the effortful control measure at 5 years of age was used. The 'all-times-before' model included all measures of effortful control measured before the outcome measure of BMI. For example, to predict BMI at 6.5 years of age, the effortful control measures at 6 months, 1 year, 2 years and 5 years of age were used. Table 3 details the approach and hypotheses for each of the three models.

Early-life model. If the first sequence of hypothesis testing arrived at an 'early-life' model as the best fitting model, one more hypothesis test was planned. As a comparator to the 'early-life' model, which allowed different magnitude and direction of association at each time period, a nested model which assumed the same direction and magnitude of association between the early life measure and the outcome (BMI) at each time point was evaluated.

Finally, if the model which assumes the same direction and magnitude of association was the better fitting model, an additional step was planned such that the model was collapsed into a more parsimonious model with fewer parameters, increasing power. In this model, 2-way interactions between age or age² and the effortful control predictor(s) were added. Starting with the interactions of age² first, interactions were tested and removed using an alpha level of 0.05. In the final model, model assumptions were tested using Jackknifed residuals. Global hypothesis testing was then used to determine if there was a significant association between effortful control and BMI trajectories.

Sex-specific trajectories. Additionally, in the final model, moderation by sex was tested by adding 2- and 3- way interactions of sex with age polynomials and effortful control. To do this, all terms in the model were stratified by sex and a contrast test was used to determine if there was an interaction using the Wald F-test and an alpha level of 0.05. INR at 6 months was included as a covariate in the final model. Analyses were conducted in SAS version 9.4 (SAS Institute.). In order to illustrate the relation between effortful control and BMI trajectories in the final model, percentiles of effortful control at the 90th, 50th and 10th percentile were computed.

3 | RESULTS

Effortful control and BMI. Hypothesis tests were conducted comparing the three models to assess whether the 'early-life' model or the two alternative models were the best fit to the data. A global hypothesis test was first conducted to examine the overall effect of effortful control in the 'early-life' model. This test showed a significant association between 6-month effortful control and BMI trajectories from infancy through adolescence, $F(5, 338) = 2.75$, $p = 0.03$, indicating that lower effortful control predicted higher BMI over time.

The first hypothesis of the current study was that effortful control at 6 months is a robust predictor of BMI over time, and that adding effortful control at other timepoints would not increase explanatory ability. This hypothesis was assessed by conducting two

TABLE 4 Model estimates for the early-life model.

Abbreviation: INR, income-to-needs ratio.

*implies interaction terms.

	BMI Males M (SE)						BMI Females M (SE)					
Effortful Control	2.5	5	7.5	10	12.5	15	2.5	5	7.5	10	12.5	15
	Years	Years	Years	Years	Years	Years	Years	Years	Years	Years	Years	Years
10th percentile (Low)	16.50	15.92	16.60	18.31	21.27	25.41	16.56	16.40	17.12	18.71	21.18	24.53
	(0.23)	(0.36)	(0.49)	(0.62)	(0.79)	(1.01)	(0.28)	(0.43)	(0.60)	(0.77)	(0.98)	(1.26)
Mean (Moderate)	16.82	16.25	16.76	18.12	20.56	24.00	16.48	16.17	16.82	18.43	21.02	24.56
	(0.15)	(0.23)	(0.32)	(0.40)	(0.51)	(0.64)	(0.18)	(0.26)	(0.36)	(0.47)	(0.60)	(0.76)
90 th percentile (High)	17.17	16.60	16.86	17.93	19.82	22.53	16.39	15.92	16.50	18.15	20.84	24.60
	(0.26)	(0.41)	(0.56)	(0.71)	(0.90)	(1.14)	(0.25)	(0.39)	(0.55)	(0.71)	(0.91)	(1.16)

FIGURE 2 Model predicted BMI over time by sex with point estimates of BMI from 6 months.

tests to compare the early-life model with two more complex models described in Table [3](#page-4-0). The sequence of hypothesis tests conducted indicated that the additional measures of effortful control in the 'immediately-before' ($F(21, 257) = 1.21$, $p = 0.25$) and the 'all-timesbefore' (F(21, 254) = 1.18, $p = 0.27$) models did not explain more variance than the early life model. Output from the 'immediately-before' and 'all-times-before' models are provided in the supplement (see Analytic Strategy Section [3](#page-4-0)). Removal of predictors that add little explanatory value results in a model with less overfitting and higher generalisability.^{[56](#page-9-0)} As a result, the more parsimonious, 'early-life' model where effortful control at 6 months predicted BMI trajectories was the best fitting model. Because the 'early-life' model was the best fitting model, the additional test to evaluate the nature of the relation between effortful control at 6-months and BMI at all later ages was conducted. This model indicated that the magnitude and direction of the association between effortful control at 6 months and BMI at each time point did not differ ($F(6, 651) = 0.80$, $p = 0.57$). Beta coefficients and confidence intervals for the individual terms predicting BMI trajectories are shown in Table [4](#page-5-0).

Sex-specific trajectories. A global hypothesis test was conducted to examine whether the BMI trajectories predicted by effortful control at 6 months differed by sex. This test indicated that BMI trajectories differed by sex, $F(4, 338) = 2.59$ $F(4, 338) = 2.59$ $F(4, 338) = 2.59$, $p = 0.03$. Figure 2 shows BMI trajectories estimated from the model for those at the 10th sample percentile, 50th sample percentile and 90th sample percentiles of effortful control, as well as model predicted BMI at different time points. Effortful control was used as a continuous variable in all analyses. The model produces predicted trajectories for people at every percentile of effortful control in the range of the study sample.

For females, as shown in Figure [2,](#page-5-0) low effortful control was associated with higher BMI from early to middle childhood (F(5, 338) $= 2.59$, $p = 0.03$ for overall trajectory difference). The model estimates in Figure [2](#page-5-0) also demonstrate this association. For males, as shown in Figure [2](#page-5-0), low effortful control was associated with higher BMI as males entered adolescence, and the difference in BMI for those with high and low effortful control continued to widen into adolescence (F(3, 296) = 3.84, $p = 0.01$ for overall trajectory difference). This is also illustrated well in the estimates from the model that are shown in Figure [2.](#page-5-0)

4 | DISCUSSION

The current study provides novel evidence that effortful control during infancy is a predictor of BMI trajectories from infancy, childhood and adolescence. Effortful control at 6 months of age predicted BMI trajectories, and the inclusion of effortful control at later time (i.e., childhood) did not provide any additional explanatory value. This finding adds to the current literature by leveraging a longitudinal design with multiple repeated measures. Findings from the current study are consistent with other evidence that infancy may be a sensitive period for potential biobehavioural programming of later BMI.^{10,57}

There are a number of plausible pathways by which early selfregulatory abilities might be associated with later BMI. First,

precursors of effortful control that are present in infancy may shape neural systems including the prefrontal cortex and nucleus accumbens, as well as other physiological systems (e.g., HPA axis) that regulate satiety and sensitivity to food cues, which then predict BMI through subsequent intake of calories.^{[28,32,33](#page-8-0)} Second, early selfregulatory abilities may shape processes that operate independently of effortful control, such as food-related parenting. Infants' regulatory abilities may influence how they interact with their early feeding environment, influencing parental feeding practices, which may set up feeding habits that persist into childhood and adolescence. Lastly, it is possible that parallel prenatal factors (e.g., biological, environmental) shape both infant effortful control and systems that contribute to appetite and satiety processes.^{[5](#page-7-0)-7}

The current study identified associations between effortful control and BMI that were sex dependent. Specifically, analyses presented here indicated that poorer infant effortful control in females was associated with higher BMI in early and middle childhood, while poorer infant effortful control in males was associated with more rapid increases in BMI starting in early adolescence. Although the prior literature evaluating sex-differences is fairly limited, the finding that effortful control is a stronger predictor of BMI in early adolescence for boys is consistent with at least one prior study.⁵⁸ The mechanisms underlying these sex differences are unknown. Given that there are known differences in body composition over development, 40 it is possible that physiological alterations in systems related to appetite, eating behaviour and body composition that occur early in life are not apparent in BMI for females until late adolescence or adulthood.

The differences for females in this sample in early and middle childhood by infant effortful control were, while statistically significant, quite small. For males in this sample, the difference in BMI associated with differences in effortful control was small in early childhood and relatively large during adolescence. By 15 years of age, males in this sample with effortful control at the 10th percentile (low) had a predicted BMI that was almost 3 kg/m^2 higher than those with effortful control at the 90th percentile (high) of effortful control. According to CDC guidelines for 15-year-old males, this difference in BMI at 15 years of age means that males with effortful control at the 10th percentile would fall into the 'overweight' range of BMI (23.4-26.8 kg/m²), while males with effortful control at the 90th percentile would be considered what the CDC calls a 'healthy weight' range (16.6-23.4 kg/m²).⁵⁹ If the sample results reported in this manuscript hold in the general population, this difference in BMI would be important, as paediatric overweight and obesity are documented predictors of poor physical and mental health across the lifespan. $3,4$ Additionally, having high body weight puts youth at risk for experiencing weight stigma, which also places youth at risk for worse social and academic outcomes and poorer mental and health.^{[60](#page-9-0)}

4.1 | Strengths and limitations

Strengths of this study include multiple assessments of BMI and effortful control from infancy through adolescence and the modelling of BMI. The repeated, longitudinal measures of BMI and effortful

8 of 10 WILEY Pediatric DEER ET AL.

control allowed for a novel modelling strategy to identify sensitive periods during which effortful control shapes BMI over development. The new analyses described here identified that effortful control in infancy predicted BMI trajectories, and that the addition of other time points in the model did not improve explanatory ability. Additionally, the diverse sample is a strength, and increases generalizability of the findings. Further consideration of potential confounding factors such as INR, birthweight, breastfeeding and pubertal timing is a strength.

The present study has several limitations. First, effortful control was assessed by parental report. Although a strength of parental report is that parents observe their children over a wide range of contexts, there is a possibility of parental bias. However, this bias is somewhat mitigated by the effortful control measures, which ask about specific behaviours in concrete situations, rather than subjective judgements about global behaviours. Additionally, these scales exhibit good internal reliability and validity as well as high consistency between parent-report and behavioural measures of effortful control. Second, BMI is just one measure of cardiometabolic health, and while it is correlated with other cardiometabolic health measures, 61 it is not a perfect measure of adiposity. An additional limitation of the current study is that height and weight measurements were taken once, rather than in duplicate. Lastly, missing data in the study was predicted by family INR, and not missing completely at random.^{[62](#page-9-0)}

4.2 | Future directions

In order to bolster the current literature, there are additional questions that need to be explored. First, future work should incorporate additional early-life predictors of BMI trajectories such as eating behaviours and parenting practices. Inspection of these alternate predictors would allow for a fuller understanding of early influences on BMI. Second, future work should examine other longitudinal measures of cardiometabolic health, and potential cardiometabolic mechanisms underlying the association between infant effortful control and later BMI. Because BMI is just one measure of cardiometabolic health, future research would benefit from using additional assessments of cardiometabolic health repeatedly measured from infancy.

5 | CONCLUSION

The current study used effortful control and BMI measured at multiple time points with rigorous statistical methods to examine whether effortful control in infancy or later in development predicts BMI trajectories. These analyses support the hypothesis that effortful control measured in infancy is associated with BMI trajectories into adolescence, and that the strength and pattern of the associations differed by sex.

AUTHOR CONTRIBUTIONS

Laura M. Glynn, Curt A. Sandman, and Elysia Poggi Davis conceptualized the study. Laura M. Glynn, Curt A. Sandman, and Elysia Poggi

Davis contributed to data collection. LillyBelle K. Deer, Jenalee R. Doom, Kylie K. Harrall, Deborah H. Glueck, and Elysia Poggi Davis analysed and interpreted the data. LillyBelle K. Deer, Jenalee R. Doom, Kylie K. Harrall, Deborah H. Glueck, and Elysia Poggi Davis drafted the manuscript. All authors contributed to editing the manuscript and approved the final manuscript.

ACKNOWLEDGEMENTS

The authors thank the families who participated in these projects. We also thank the dedicated staff at the Early Human and Lifespan Development Program. This research was supported by grants from the National Institutes of Health (HD-28413 Curt A. Sandman), HD-40967 (Laura M. Glynn), HD-51852 (Curt A. Sandman), NS-41298 (Curt A. Sandman), and MH-96889 (Elysia Poggi Davis, Laura M. Glynn, Curt A. Sandman). This manuscript was prepared with support from the National Heart, Lung, and Blood Institute (NHLBI) grant F32HL165844 to LillyBelle K. Deer, NHLBI grant K01HL143159 to Jenalee R. Doom, NHLBI grant R01HL155744 to Elysia Poggi Davis and Jenalee R. Doom, the National Institute of Mental Health grant R01MH109662 to Elysia Poggi Davis, as well as from the National Institute of General Medical Sciences grant 5R01GM121081-08 to Kylie K. Harrall and Deborah H. Glueck.

CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest relevant to this article to disclose.

ORCID

LillyBelle K. Deer **b** <https://orcid.org/0000-0001-9934-9381>

REFERENCES

- 1. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. Lancet Lond Engl. 2017;390(10113):2627-2642. doi[:10.1016/S0140-6736\(17\)32129-3](info:doi/10.1016/S0140-6736(17)32129-3)
- 2. World Health Organization. Obesity and overweight. Accessed May 18, 2022. [https://www.who.int/news-room/fact-sheets/detail/](https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight) [obesity-and-overweight](https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight)
- 3. Jacobs DR, Woo JG, Sinaiko AR, et al. Childhood cardiovascular risk factors and adult cardiovascular events. N Engl J Med. 2022;386: 1877-1888. doi:[10.1056/NEJMoa2109191](info:doi/10.1056/NEJMoa2109191)
- 4. Sahoo K, Sahoo B, Choudhury AK, Sofi NY, Kumar R, Bhadoria AS. Childhood obesity: causes and consequences. J Fam Med Prim Care. 2015;4(2):187-192. doi:[10.4103/2249-4863.154628](info:doi/10.4103/2249-4863.154628)
- 5. Barker DJ. In utero programming of chronic disease. Clin Sci Lond Engl. 1979;95(2):115-128.
- 6. Gluckman PD, Hanson MA. Developmental origins of disease paradigm: a mechanistic and evolutionary perspective. Pediatr Res. 2004; 56(3):311-317. doi[:10.1203/01.PDR.0000135998.08025.FB](info:doi/10.1203/01.PDR.0000135998.08025.FB)
- 7. Stout SA, Espel EV, Sandman CA, Glynn LM, Davis EP. Fetal programming of children's obesity risk. Psychoneuroendocrinology. 2015;53:29- 39. doi[:10.1016/j.psyneuen.2014.12.009](info:doi/10.1016/j.psyneuen.2014.12.009)
- 8. Davis EP, Pfaff D. Sexually dimorphic responses to early adversity: implications for affective problems and autism spectrum disorder. Psychoneuroendocrinology. 2014;49:11-25. doi[:10.1016/j.psyneuen.](info:doi/10.1016/j.psyneuen.2014.06.014) [2014.06.014](info:doi/10.1016/j.psyneuen.2014.06.014)
- 9. Sandman CA, Glynn LM, Davis EP. Is there a viability-vulnerability tradeoff? Sex differences in fetal programming. J Psychosom Res. 2013; 75(4):327-335. doi:[10.1016/j.jpsychores.2013.07.009](info:doi/10.1016/j.jpsychores.2013.07.009)
- 10. Doom JR, Rivera KM, Blanco E, et al. Sensitive periods for psychosocial risk in childhood and adolescence and cardiometabolic outcomes in young adulthood. Dev Psychopathol. 2020;32(5):1864-1875. doi: [10.1017/S0954579420001248](info:doi/10.1017/S0954579420001248)
- 11. Rundle AG, Factor-Litvak P, Suglia SF, et al. Tracking of obesity in childhood into adulthood: effects on body mass index and fat mass index at age 50. Child Obes. 2020;16(3):226-233. doi[:10.1089/chi.](info:doi/10.1089/chi.2019.0185) [2019.0185](info:doi/10.1089/chi.2019.0185)
- 12. Ryder JR, Jacobs DR, Sinaiko AR, Kornblum AP, Steinberger J. Longitudinal changes in weight status from childhood and adolescence to adulthood. J Pediatr. 2019;214:187-192.e2. doi[:10.1016/j.jpeds.](info:doi/10.1016/j.jpeds.2019.07.035) [2019.07.035](info:doi/10.1016/j.jpeds.2019.07.035)
- 13. Brisbois TD, Farmer AP, McCargar LJ. Early markers of adult obesity: a review. Obes Rev Off J Int Assoc Study Obes. 2012;13(4):347-367. doi:[10.1111/j.1467-789X.2011.00965.x](info:doi/10.1111/j.1467-789X.2011.00965.x)
- 14. Geserick M, Vogel M, Gausche R, et al. Acceleration of BMI in early childhood and risk of sustained obesity. N Engl J Med. 2018;379(14): 1303-1312. doi[:10.1056/NEJMoa1803527](info:doi/10.1056/NEJMoa1803527)
- 15. Barker DJP. Adult consequences of fetal growth restriction. Clin Obstet Gynecol. 2006;49(2):270-283.
- 16. Oken E, Gillman MW. Fetal origins of obesity. Obes Res. 2003;11(4): 496-506. doi:[10.1038/oby.2003.69](info:doi/10.1038/oby.2003.69)
- 17. Stuart B, Panico L. Early-childhood BMI trajectories: evidence from a prospective, nationally representative British cohort study. Nutr Diabetes. 2016;6(3):e198. doi:[10.1038/nutd.2016.6](info:doi/10.1038/nutd.2016.6)
- 18. Niclasen BVL, Petzold MG, Schnohr C. Overweight and obesity at school entry as predictor of overweight in adolescence in an Arctic child population. Eur J Public Health. 2007;17(1):17-20. doi[:10.1093/](info:doi/10.1093/eurpub/ckl246) [eurpub/ckl246](info:doi/10.1093/eurpub/ckl246)
- 19. Sutharsan R, O'Callaghan MJ, Williams G, Najman JM, Mamun AA. Rapid growth in early childhood associated with young adult overweight and obesity—evidence from a community based cohort study. J Health Popul Nutr. 2015;33:13. doi:[10.1186/s41043-015-0012-2](info:doi/10.1186/s41043-015-0012-2)
- 20. Cerrato-Carretero P, Roncero-Martín R, Pedrera-Zamorano JD, et al. Long-term dietary and physical activity interventions in the school setting and their effects on BMI in children aged 6-12 years: metaanalysis of randomized controlled clinical trials. Healthc Basel Switz. 2021;9(4):396. doi:[10.3390/healthcare9040396](info:doi/10.3390/healthcare9040396)
- 21. Kamath CC, Vickers KS, Ehrlich A, et al. Behavioral interventions to prevent childhood obesity: a systematic review and Metaanalyses of randomized trials. J Clin Endocrinol Metab. 2008;93(12):4606-4615. doi:[10.1210/jc.2006-2411](info:doi/10.1210/jc.2006-2411)
- 22. Rothbart MK, Bates JE. Temperament. Handbook of Child Psychology: Social, Emotional, and Personality Development. Vol 3. 6th ed. John Wiley & Sons, Inc.; 2006:99-166.
- 23. Tominey S, Mcclelland M. The development of self-regulation and executive function in young children. Zero Three J. 2014;35:2-8.
- 24. Stifter CA, Moding KJ. Temperament in obesity-related research: concepts, challenges, and considerations for future research. Appetite. 2019;141:104308. doi:[10.1016/j.appet.2019.05.039](info:doi/10.1016/j.appet.2019.05.039)
- 25. Isasi CR, Wills TA. Behavioral self-regulation and weight-related behaviors in Inner-City adolescents: a model of direct and indirect effects. Child Obes. 2011;7(4):306-315. doi:[10.1089/chi.2011.0011](info:doi/10.1089/chi.2011.0011)
- 26. Steinsbekk S, Bjørklund O, Llewellyn C, Wichstrøm L. Temperament as a predictor of eating behavior in middle childhood—a fixed effects approach. Appetite. 2020;150:104640. doi:[10.1016/j.appet.2020.104640](info:doi/10.1016/j.appet.2020.104640)
- 27. Zhou Z, SooHoo M, Zhou Q, Perez M, Liew J. Temperament as risk and protective factors in obesogenic eating: relations among parent temperament, child temperament, and child food preference and eating. J Genet Psychol. 2019;180(1):75-79. doi[:10.1080/00221325.2019.1575180](info:doi/10.1080/00221325.2019.1575180)
- 28. Birch L, Savage JS, Ventura A. Influences on the development of children's eating Behaviours: from infancy to adolescence. Can J Diet Pract Res. 2007;68(1):s1-s56.
- 29. Brown A, Lee MD. Early influences on child satiety-responsiveness: the role of weaning style. Pediatr Obes. 2015;10(1):57-66. doi[:10.](info:doi/10.1111/j.2047-6310.2013.00207.x) [1111/j.2047-6310.2013.00207.x](info:doi/10.1111/j.2047-6310.2013.00207.x)
- 30. Reynolds R. Programming effects of glucocorticoids. Clin Obstet Gynecol. 2013;56(3):602-609. doi[:10.1097/GRF.0b013e31829939f7](info:doi/10.1097/GRF.0b013e31829939f7)
- 31. Nusslock R, Miller GE. Early-life adversity and physical and emotional health across the lifespan: a neuroimmune network hypothesis. Biol Psychiatry. 2016;80(1):23-32. doi[:10.1016/j.biopsych.2015.05.017](info:doi/10.1016/j.biopsych.2015.05.017)
- 32. Russell CG, Russell A. "Food" and "non-food" self-regulation in childhood: a review and reciprocal analysis. Int J Behav Nutr Phys Act. 2020;17(1):33. doi:[10.1186/s12966-020-00928-5](info:doi/10.1186/s12966-020-00928-5)
- 33. Stoeckel LE, Birch LL, Heatherton T, et al. Psychological and neural contributions to appetite self-regulation. Obes Silver Spring Md. 2017; 25(suppl 1):S17-S25. doi[:10.1002/oby.21789](info:doi/10.1002/oby.21789)
- 34. Anzman-Frasca S, Stifter CA, Birch LL. Temperament and childhood obesity risk: a review of the literature. J Dev Behav Pediatr. 2012; 33(9):732-745. doi:[10.1097/DBP.0b013e31826a119f](info:doi/10.1097/DBP.0b013e31826a119f)
- 35. Thamotharan S, Lange K, Zale EL, Huffhines L, Fields S. The role of impulsivity in pediatric obesity and weight status: a meta-analytic review. Clin Psychol Rev. 2013;33(2):253-262. doi[:10.1016/j.cpr.](info:doi/10.1016/j.cpr.2012.12.001) [2012.12.001](info:doi/10.1016/j.cpr.2012.12.001)
- 36. Francis LA, Susman EJ. Self-regulation and rapid weight gain in children from age 3 to 12 years. Arch Pediatr Adolesc Med. 2009;163(4): 297-302. doi:[10.1001/archpediatrics.2008.579](info:doi/10.1001/archpediatrics.2008.579)
- 37. Graziano PA, Kelleher R, Calkins SD, Keane SP, Brien MO. Predicting weight outcomes in preadolescence: the role of toddlers' selfregulation skills and the temperament dimension of pleasure. Int J Obes (Lond). 2013;37(7):937-942. doi[:10.1038/ijo.2012.165](info:doi/10.1038/ijo.2012.165)
- 38. Seeyave DM, Coleman S, Appugliese D, et al. Ability to delay gratification at age 4 years and risk of overweight at age 11 years. Arch Pediatr Adolesc Med. 2009;163(4):303-308. doi:[10.1001/](info:doi/10.1001/archpediatrics.2009.12) [archpediatrics.2009.12](info:doi/10.1001/archpediatrics.2009.12)
- 39. Faith MS, Hittner JB. Infant temperament and eating style predict change in standardized weight status and obesity risk at 6 years of age. Int J Obes (Lond). 2010;34(10):1515-1523. doi[:10.1038/ijo.](info:doi/10.1038/ijo.2010.156) [2010.156](info:doi/10.1038/ijo.2010.156)
- 40. Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, et al. CDC growth charts: United States. Adv Data. 2000;314:1-27.
- 41. Glynn LM, Howland MA, Sandman CA, et al. Prenatal maternal mood patterns predict child temperament and adolescent mental health. J Affect Disord. 2018;228:83-90. doi:[10.1016/j.jad.2017.11.065](info:doi/10.1016/j.jad.2017.11.065)
- 42. Gartstein MA, Rothbart MK. Studying infant temperament via the revised infant behavior questionnaire. Infant Behav Dev. 2003;26(1): 64-86. doi[:10.1016/S0163-6383\(02\)00169-8](info:doi/10.1016/S0163-6383(02)00169-8)
- 43. Lin B, Liew J, Perez M. Measurement of self-regulation in early childhood: relations between laboratory and performance-based measures of effortful control and executive functioning. Early Child Res Q. 2019;47:1-8. doi[:10.1016/j.ecresq.2018.10.004](info:doi/10.1016/j.ecresq.2018.10.004)
- 44. Nigg JT. Annual research review: on the relations among selfregulation, self-control, executive functioning, effortful control, cognitive control, impulsivity, risk-taking, and inhibition for developmental psychopathology. J Child Psychol Psychiatry. 2017;58(4):361-383. doi: [10.1111/jcpp.12675](info:doi/10.1111/jcpp.12675)
- 45. Schmidt H, Daseking M, Gawrilow C, Karbach J, Kerner auch Koerner J. Self-regulation in preschool: are executive function and effortful control overlapping constructs? Dev Sci. 2022;25(6):e13272. doi:[10.1111/desc.13272](info:doi/10.1111/desc.13272)
- 46. Rothbart MK, Ahadi SA, Evans DE. Temperament and personality: origins and outcomes. J Pers Soc Psychol. 2000;78(1):122-135. doi[:10.](info:doi/10.1037//0022-3514.78.1.122) [1037//0022-3514.78.1.122](info:doi/10.1037//0022-3514.78.1.122)
- 47. East P, Delker E, Blanco E, et al. BMI trajectories from birth to 23 years by cardiometabolic risks in young adulthood. Obes Silver Spring Md. 2020;28(4):813-821. doi[:10.1002/oby.22754](info:doi/10.1002/oby.22754)
- 48. Petersen AC, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: reliability, validity, and initial norms. J Youth Adolesc. 1988;17(2):117-133. doi:[10.1007/BF01537962](info:doi/10.1007/BF01537962)

10 of 10 WII FY Pediatric DEER ET AL.

- 49. Koopman-Verhoeff ME, Gredvig-Ardito C, Barker DH, Saletin JM, Carskadon MA. Classifying pubertal development using child and parent report: comparing the pubertal development scales to Tanner staging. J Adolesc Health Off Publ Soc Adolesc Med. 2020;66(5):597- 602. doi[:10.1016/j.jadohealth.2019.11.308](info:doi/10.1016/j.jadohealth.2019.11.308)
- 50. Terry MB, Goldberg M, Schechter S, et al. Comparison of clinical, maternal, and self pubertal assessments: implications for health studies. Pediatrics. 2016;138(1):e20154571. doi[:10.1542/peds.2015-](info:doi/10.1542/peds.2015-4571) [4571](info:doi/10.1542/peds.2015-4571)
- 51. Lum S, Bountziouka V, Harding S, Wade A, Lee S, Stocks J. Assessing pubertal status in multi-ethnic primary schoolchildren. Acta Paediatr Oslo Nor. 1992;104(1):e45-e48. doi[:10.1111/apa.12850](info:doi/10.1111/apa.12850)
- 52. Laird NM, Ware JH. Random-effects models for longitudinal data. Biometrics. 1982;38(4):963-974.
- 53. Wen X, Kleinman K, Gillman MW, Rifas-Shiman SL, Taveras EM. Childhood body mass index trajectories: modeling, characterizing, pairwise correlations and socio-demographic predictors of trajectory characteristics. BMC Med Res Methodol. 2012;12(1):38. doi:[10.1186/](info:doi/10.1186/1471-2288-12-38) [1471-2288-12-38](info:doi/10.1186/1471-2288-12-38)
- 54. Kenward MG, Roger JH. An improved approximation to the precision of fixed effects from restricted maximum likelihood. Comput Stat Data Anal. 2009;53(7):2583-2595. doi:[10.1016/j.csda.2008.12.013](info:doi/10.1016/j.csda.2008.12.013)
- 55. Srivastava VK, Giles DEA. Seemingly unrelated regression equations models: estimation and inference. CRC Press; 1987.
- 56. Harrell FE, Lee KL, Mark DB. Multivariable prognostic models: issues in developing models, evaluating assumptions and adequacy, and measuring and reducing errors. Stat Med. 1996;15(4):361-387. doi: [10.1002/\(SICI\)1097-0258\(19960229\)15:43.0.CO;2-4](info:doi/10.1002/(SICI)1097-0258(19960229)15:4<361::AID-SIM168>3.0.CO;2-4)
- 57. Ziyab AH, Karmaus W, Kurukulaaratchy RJ, Zhang H, Arshad SH. Developmental trajectories of body mass index from infancy to 18 years of age: prenatal determinants and health consequences. J Epidemiol Community Health. 2014;68(10):934-941. doi[:10.1136/](info:doi/10.1136/jech-2014-203808) [jech-2014-203808](info:doi/10.1136/jech-2014-203808)
- 58. Connell LE, Francis LA. Positive parenting mitigates the effects of poor self-regulation on BMI trajectories from age 4 to 15 years. Health Psychol Off J Div Health Psychol Am Psychol Assoc. 2014;33(8): 757-764. doi:[10.1037/hea0000014](info:doi/10.1037/hea0000014)
- 59. CDC. About Child and Teen BMI. Centers for Disease Control and Prevention. Published September 24, 2022 Accessed January 27, 2023. [https://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/](https://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html) [about_childrens_bmi.html](https://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html)
- 60. Puhl RM, Lessard LM. Weight stigma in youth: prevalence, consequences, and considerations for clinical practice. Curr Obes Rep. 2020; 9(4):402-411. doi[:10.1007/s13679-020-00408-8](info:doi/10.1007/s13679-020-00408-8)
- 61. Kahn HS, Cheng YJ. Comparison of adiposity indicators associated with fasting-state insulinemia, triglyceridemia, and related risk biomarkers in a nationally representative, adult population. Diabetes Res Clin Pract. 2018;136:7-15. doi:[10.1016/j.diabres.2017.11.019](info:doi/10.1016/j.diabres.2017.11.019)
- 62. Little RJA, Rubin DB. Statistical Analysis with Missing Data. 2nd ed. John Wiley & Sons, Inc; 2002. Accessed December 16, 2022. doi[:10.](info:doi/10.1002/9781119013563) [1002/9781119013563](info:doi/10.1002/9781119013563)
- 63. Putnam SP, Gartstein MA, Rothbart MK. Measurement of finegrained aspects of toddler temperament: The Early Childhood Behavior Questionnaire. Infant Behav Dev. 2006;29(3):386-401. doi[:10.](info:doi/10.1016/j.infbeh.2006.01.004) [1016/j.infbeh.2006.01.004](info:doi/10.1016/j.infbeh.2006.01.004)
- 64. Simonds J. The role of reward sensitivity and response: Execution in childhood extraversion. Published online 2006.
- 65. Ellis LK, Rothbart MK. Early Adolescent Temperament Questionnaire-Revised. APA PsycTests. Published online 2001. doi:[10.1037/](info:doi/10.1037/t07624-000) [t07624-000](info:doi/10.1037/t07624-000).

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Deer LK, Doom JR, Harrall KK, et al. Infant effortful control predicts BMI trajectories from infancy to adolescence. Pediatric Obesity. 2023;e13059. doi[:10.1111/](info:doi/10.1111/ijpo.13059) [ijpo.13059](info:doi/10.1111/ijpo.13059)