UNIVERSITY OF CALIFORNIA,
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Early-Life Socioeconomic Disadvantage, Domain-Specific Self-Regulation, and Pediatric Obesity Risk: Evidence from a Longitudinal Birth Cohort

DISSERTATION

submitted in partial satisfaction of the requirements
for the degree of

DOCTOR OF PHILOSOPHY

in Epidemiology

by

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Dissertation Committee:
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DEDICATION

TO

My family and friends from near and afar

in recognition of their irreplaceable place in my life

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All three chapters of this dissertation used data from the NICHD Study of Early Child Care and Youth Development (SECCYD). The authors thank the participants and investigators involved in the study.

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This degree was not earned in a conventional way, nor on a conventional timeline. I came to epidemiology after earning both my Bachelor's and Master's degrees in social sciences, a decade building a career in strategic communications in the luxury brands industry, and seven years teaching in Hong Kong — a path that taught me to accept imperfections in studying human behavior and to translate complexity into something people can understand, skills I now realize I have used in every chapter of this dissertation.

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VITA

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ABSTRACT OF THE DISSERTATION

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Doctor of Philosophy in Epidemiology

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Background: Childhood obesity disproportionately affects children from lower-income households, yet the early developmental pathways linking socioeconomic disadvantage to later weight outcomes remain incompletely understood. Self-regulation — a child's ability to control attention, behavior, and emotion — has been proposed as a potential mechanism, given its sensitivity to socioeconomic stress and its links to energy-balance behaviors. Additionally, adiposity rebound, the second rise in BMI typically occurring between ages 5 and 7, is an established early-life marker of obesity risk whose socioeconomic determinants remain unclear. This dissertation uses data from the NICHD Study of Early Child Care and Youth Development (SECCYD), a prospective cohort of 1,364 children recruited at birth from 10 US hospitals, to address three interrelated questions about how early socioeconomic conditions and self-regulatory capacities shape weight trajectories across childhood and adolescence.

Methods: In Chapter 1, I examined whether early childhood household income is associated with adiposity rebound timing using propensity score matching in 818 children, followed by modified Poisson regression in approximately 288 matched children. In Chapter 2, I examined whether three domain-specific self-regulation measures — cool self-regulation, food-related hot self-regulation, and nonfood hot self-regulation, assessed between 36 and 54 months — were differentially associated with overweight/obesity at age 15 in 698 children, using modified Poisson regression with sex-stratified analyses. In Chapter 3, I applied counterfactual-based causal mediation analysis to decompose the total effect of early childhood household income on overweight/obesity at age 9 into natural direct and natural indirect effects operating through cool and food-related hot self-regulation in 782 children.

Results: In Chapter 1, low household income was not associated with early adiposity rebound in the matched sample (RR=0.91; 95% CI: 0.71–1.17), with consistent null findings across sensitivity analyses. In Chapter 2, cool self-regulation and food-related hot self-regulation were each independently associated with lower overweight/obesity risk at age 15 (cool SR: aRR=0.96 per 10-point increase, 95% CI: 0.93–0.99; food hot SR: aRR=0.78, 95% CI: 0.64–0.96), while nonfood hot self-regulation showed no significant association. The protective association for food-related hot self-regulation was concentrated among boys (aRR=0.64; 95% CI: 0.52–0.78) but not girls. In Chapter 3, although low household income was associated with both higher odds of overweight/obesity at age 9 (adjusted OR=1.54; 95% CI: 1.02–2.33) and poorer self-regulation across both domains, neither domain mediated the income–weight status association; all natural indirect effects were close to null and non-significant.

Conclusions: Early childhood household income does not appear to independently shape adiposity rebound timing in this cohort, and socioeconomic disparities in childhood obesity

appear to operate through pathways beyond early self-regulatory capacity. These findings are based on a single U.S. birth cohort and warrant replication in other populations. At the same time, domain-specific self-regulation — particularly cool and food-related hot self-regulation — independently predicts adolescent obesity risk, underscoring the value of disaggregating self-regulation constructs in research and intervention design. These findings collectively highlight the importance of structural and environmental mechanisms in linking socioeconomic disadvantage to pediatric obesity and suggest that interventions targeting self-regulation alone may be insufficient to reduce income-related disparities in childhood weight outcomes.

INTRODUCTION

Over the past decade, the percentage of births with a normal birth weight (2,500-3,999 grams) in the US ranged between 80% and 85%.[1] Incongruously, the prevalence of obesity among children and adolescents in the US reached 20.9% in 2020, climbing from 16.9% in 2016.[2] Why do children with the same starting point (i.e., normal birth weight) have differential growth trajectories?

Body mass index (BMI), calculated from an individual's height and weight, is most commonly used to define one's status of having overweight and obesity (OWO).[3] Being at the higher tail of the BMI curve during childhood is alarming not only because there is a 70% chance the excess weight will be carried into adulthood,[4] but it also predicts increased risks for negative physical and mental health outcomes in later life.[5]

There are significant benefits to studying the trajectory and dynamics of changes in a child's BMI longitudinally from early childhood, such as effectively predicting future risks of overweight or obesity (OWO).[6,7] The timing of adiposity rebound (AR) is one such indicator. AR is the second rise of BMI in children after reaching its nadir, typically around age 5-7.[8] Rolland first reported the concept in 1984 that children with an early rebound (before age 5.5) had a higher adiposity level at age 16 than their counterparts who experienced the rebound after age 7.[9] Studies that followed found congruent results that early AR predicts higher weight in later life.[10] Some have further identified other negative health impacts associated with early AR, such as risks of metabolic syndrome during adolescence[11] and Type 2 diabetes in adulthood.[12]

Socioeconomic status (SES) is negatively associated with childhood obesity prevalence in high-income countries, including the US.[13–15] It is often considered the upstream factor leading to other disadvantages related to unhealthful weight gain in children, such as the lack of

green space and parks in the neighborhood[16] and overconsumption of ultra-process foods.[17] Compared to cross-sectional studies using BMI as a weight indicator, research on the relationship between SES and timing of AR is scant, and findings are inconclusive.[18–21]

Self-regulation (SR), a neurocognitive development that takes crucial form during earlier years in life,[22,23] has been linked to both SES[24–27] and pediatric weight status.[28–30] There has been sufficient evidence illustrating a negative association between SR and pediatric weight gain that encouraged interventions targeting to improve children's SR ability as primary or secondary prevention to OWO — such as parenting programs that coach caregivers on scaffolding children's emotion and behavior regulation, and preschool-based curricula teaching strategies like delay of gratification and impulse control; however, results from these interventions have not been consistent.[31–33] One conjecture is the nuance between hot vs. cool SR, that these two types of SR might not operate the same regarding weight-related behaviors[34] but were not typically studied singly in the literature. Another gap in the literature is that despite the consistent evidence between SR and SES, SR and pediatric weight outcomes, and SES and weight outcomes, no study has examined the mediating role of SR in the relationship between SES and weight outcomes in children.

While SES and SR have been vastly studied in their associations with pediatric OWO, research on the interplay between the three constructs is limited. In this dissertation, SES is operationalized through its resource-based component — specifically household income — as distinct from prestige-based indicators such as parental education or occupation. Findings from our studies will provide insights into intervention strategies that consider SES-related attributes in the social-ecological framework[35,36] and components of SR building. I propose to use data

from The National Institute of Child Health and Development (NICHD) Study of Early Child Care and Youth Development (SECCYD) to achieve the following aims:

Aim 1: Examine whether resource-based SES during infancy predicts the timing of AR.

Aim 2: Investigate whether hot and cool SR in early childhood differentially predict obesity at 15 years old. Based on preliminary evidence from the literature, I will also examine whether there are heterogeneous effects by sex at birth.

Aim 3: Test whether hot and cool SR in middle childhood mediate the relationship between SES at infancy and OWO status at 9 years old. I will stratify the analysis by sex to test whether there are heterogeneous effects by sex at birth.

Impact

Based on national data from 2011-2016, estimates associated pediatric obesity with $1.32 billion in medical spending, with $116 and $310 excess costs for each child with obesity and severe obesity, respectively.[37] Despite efforts to tackle the issue, the prevalence of pediatric obesity in the US has increased steadily over the past decade,[38] to an extent that triggered debates among medical professionals to declare pediatric obesity a public health emergency.[39] My research aims to connect the dots between resource-based SES, SR, and pediatric weight trajectory that need to be clarified, given the wealth of evidence on each front. Each aim is expected to add impactfully to the literature regarding the nuances in the interplay between SES, SR development, and pediatric weight trajectories, informing intervention efforts in tackling this urgent public health issue.

Significance

A BMI between the 85th and 95th percentile of their peers of the same sex and age

indicates overweight, whereas a BMI at or over the 95th percentile is categorized as having obesity.[3] The same cutoffs apply to children, and the measurements are based on weight-for-length for children under two years of age. What is worrying is not the extra weight some children carry but the substantial association between childhood OWO and comorbidities in later life, including metabolic syndrome,[40] cardiovascular diseases,[41] type-2 diabetes,[42] and even increased risks for cancers.[43,44] Besides physical health, having OWO during childhood is also associated with negative mental health outcomes as children grow.[45]

What sets apart children who manage to stay below the 85th percentile line through childhood? One quick answer is genetics, illustrated by evidence supporting a genetic predisposition to obesity risk.[46–48] Yet, the resounding evidence on environmental factors (i.e., any influences outside of genetics) that burgeoned over the past two decades has shifted part of the narrative away from heritability. As knowledge on the etiology of childhood obesity continues to accumulate, there is a general consensus that the condition is a result of an interplay between polygenic predisposition and obesogenic environmental factors,[49] or in layman's terms, "the genetic background loads the gun, but the environment pulls the trigger."[50]

An early AR is one of the earliest indicators of risks for high adiposity in later life[51,52] although most early rebounders do not have OWO at the point of rebound,[9] making it an optimal tool in early obesity prevention strategies targeting at-risk groups. However, evidence of the association between SES and the timing of AR is lacking. The literature contains substantial evidence that childhood OWO, as measured by BMI, tends to carry into adulthood — a pattern that has motivated using cross-sectional childhood BMI as an early warning sign of adult obesity. Less discussed, however, is that the reverse does not

necessarily hold: a substantial proportion of adults with OWO did not have the condition as children.[53] Adult obesity prevalence was 41.9% in 2017-2020, over double the childhood obesity prevalence of 19.7% during the same period.[38] Besides identifying children with OWO as a predictor of adult OWO, it is also conducive to bring it a step further to identify those at higher risk of developing pediatric OWO before it happens for proper intervention, and observing children's timing of AR is one such tool.

Contrary to the initial belief that AR was due to an increase in fat mass when the term was first coined,[9] recent studies found that when AR occurs, the decline in fat mass ceases, and lean mass starts rising. This phenomenon is more accurately described as a "BMI rebound" since the increase in weight does not necessitate an increase in adiposity. There is a two to three-year lag after AR occurs for adiposity to start rising.[54] Nonetheless, an early AR has been consistently associated with obesity in later childhood and adolescence,[10,55] and is further linked to other health complications, including type 2 diabetes,[56,57] dyslipidemia,[58] and insulin resistance.[59]

Only a handful of studies have examined the relationship between SES and early AR. Two of the earliest studies found no association between parental education attainment and early AR.[18,19] A more recent study using an aggregated measure of SES with parental education attainment, occupation, and household income found that SES partially explained the disparities of early AR observed in ethnic minority groups.[60] Two additional studies found significant associations between maternal education attainment and early AR.[21,61] One of the two studies that had a Latino farmworkers sample living in the US further identified that early rebounders were more likely to be living in undocumented families.[21] Although documentation status is not a common SES index, being undocumented often means earning

lower wages and having less favorable living conditions.[62] With the current conflicting evidence and as childhood OWO disproportionately impacts children living in low SES households in the US, further research on how SES is related to the timing of AR is warranted.

Evidence on the mechanisms involving the timing of AR is also lacking. One modifiable risk factor related to parenting is breastfeeding, but findings remain dubious.[20,63–65] Screen time has also been positively associated with early AR.[66] Identifying modifiable risk factors of AR will contribute to interventions starting in early childhood, which has recently gained consensus as essential in tackling pediatric obesity.[3]

The literature points strongly to a negative association between children's self-regulation (SR) ability and risks of childhood obesity,[28,67–69] intuitively positing that SR is related to energy-balance-related behaviors (EBRB), such as eating in the absence of hunger[70,71] and getting oneself to exercise.[72,73] SR is generally considered the ability to control one's behavior, attention, and emotions in order to fulfill personally relevant goals despite conflicting external cues.[74] It is often referred to as self-control, effortful control, or inhibitory control. There are overlapping elements with executive functioning, another cognitive function involving elements of impulse inhibition.[61] While these cognitive abilities have a genetic basis, SR is malleable based on life experiences or targeted training.[75]

Children living in low SES households are at risk of having lower SR,[76–79] as chronic stress can affect SR development, a mechanism particularly crucial during the first years of life.[80] The constellation of evidence has motivated intervention programs to increase SR ability for weight management in children, with equivocal findings that do not seem to fare better than pure chance. There have been interventions that significantly improved both

children's SR and weight status post-intervention.[81,82] Contrasting these successes, some interventions significantly improved children's SR post-intervention but did not significantly improve weight.[31,33,83] Most peculiarly, one study that did not see an improvement in children's SR post-intervention observed a significantly lower proportion of children with OWO in the intervention group three years post-treatment.[32] Nonetheless, what is clear from the current literature is that SR is malleable, and children with better SR fare better with weight management. Then, why were interventions targeted to improve children's SR not effective in improving children's weight status? The gap in this research remains on three fronts: 1) the lack of distinction between hot and cool SR, 2) the convolution of food-related and dispositional self-regulation ability in relation to weight trajectory, and 3) insufficient integration of pathways through which SR operates.

SR dominantly relies on the prefrontal cortex (PFC).[84] When faced with problems that involve little emotion or affection, the dorsolateral regions of PFC (DL-PFC) are activated to exercise cool SR. When the situation on hand triggers emotions, such as appetitive desires or fear-inducing stimuli, the ventral or medial regions of the PFC (VM-PFC) are activated to exercise hot SR.[85] Scholars across disciplines recently called for a distinction to guard against oversimplifying PFC-mediated processes.[85,86] Evidence of different correlates of the two SRs provide further support for such call: Cool SR has been found to correlate with intelligence,[87] academic performance,[88] and aspects of temperaments, while hot SR correlates with inattentive-overactive behaviors[86] and smoking in adults.[89] EBRE behaviors in children might relate more to one domain of SR than the other, but there is little evidence of this. Sifting them apart will merit interventions that more accurately target the type of SR essential for children's weight management.

Because of the rich data structure and wide range of variables, a handful of studies with the proposed population examined the relationship between SR and subsequent weight outcomes. However, all existing studies operationalized SR with either the delay of gratification (DoG) task or as a composite measure that included the DoG task,[28,29,90,91] as this variation of the Marshmallow test seems to be a classic choice to test children's battery of self-control. The current study will be the first to examine this relationship, operationalizing SR with a cool SR measure in this population. Findings of studies in other populations that operationalized SR with a cool SR instrument remain inconclusive, as they tend to be fewer in number and mostly cross-sectional.[71,92–94] A longitudinal study, as currently proposed, will add insight into whether cool SR is a domain of SR that significantly predicts subsequent weight status in children.

Second, most previous studies that examined the hot domain of self-regulation in children measured it through the delay of gratification task using food as prompts.[28,29,69,90,91] While these studies termed such a general measure of self-regulation, studies from other domains suggest nuances in the constructs of dispositional (i.e., nonfood related) self-regulation and self-regulation that taps into one's food responsiveness, which is referred to as reward sensitivity with food consumption, external food cue responsiveness, and external eating, etc..[95–98] Furthermore, the extent to which global SR measures predict eating-specific SR is unclear.[99–101] Testing the distinctive associations between food versus nonfood-related self-regulation with subsequent weight outcomes can further inform the specific domain of self-regulation battery that needs to be targeted in interventions in hopes of weight management in children.

Lastly, mapping out the pathways between SES, SR, and weight status is crucial in intervention design and targeting the most at-risk populations. The pathways yet to be identified include whether SR acts as a mediator [95–98] between SES and weight status and the mechanisms of how SR leads to different weight outcomes, such as through food consumption habits.

The proposed study, which aims to fill the literature gaps mentioned above, is informed by the psychosocial and developmental frameworks that have garnered much attention recently in the discussion of childhood obesity.[102] There has been a seismic shift in recognizing the psychosocial factors that contribute to children's OWO, as opposed to viewing the condition purely as a result of overeating and/or the lack of physical activity.[103] A strong base of evidence has also accumulated, linking childhood obesity to a cascade of events taking place through a series of developmental stages starting from the perinatal period.[104]

Innovation

The approach of distinguishing hot and cool SR concerning weight status is novel. To my best knowledge, no prior research treating children's weight status as the main outcome distinguished between hot vs. cool SR. One study that differentiated hot and cool SR in association with eating behaviors found differing associations between the two domains of SR, but how these behaviors are associated with weight status remained speculative.[105]

The current study will be the first to disaggregate hot SR using two measures from different tasks that prompt food versus nonfood-related hot SR. The delivery of behavioral interventions to children that could result in substantial effect sizes is an existing tall order.[106] It is crucial to deliver evidence-based interventions that target the most influential component of SR contributing to weight management, and this empirical inquiry will help fill this gap.

To the best of my knowledge, there is no existing longitudinal study that examines the role of SR in the pathway of resource-based SES and children's weight status at this time. One mediation study collected cross-sectional data on SR and nine proxies of cumulative risks, including poverty, and found that SR mediated the linkage between cumulative risks and concurrent and subsequent BMI.[107] Another study that collected cross-sectional data on all three constructs did not find a mediating but a moderating role of SR combined with knowledge of healthy eating.[108] While these studies are relevant, they do not shed light on how SES contributes to the development of SR in subsequent childhood stages and sequentially influences weight status in later years.

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CHAPTER 1: Household Income and Timing of Adiposity Rebound in Early Childhood

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Abstract

Background/Objectives: Adiposity rebound (AR) has been proposed as an early-life marker of later obesity risk, yet evidence on its socioeconomic determinants remains limited and inconsistent. This study examined whether early childhood household income, representing resource-based socioeconomic status (SES), is associated with the timing of AR using a quasi-experimental approach.

Subjects/Methods: We used data from 1,364 children enrolled in the US-based NICHD Study of Early Child Care and Youth Development. Children with ≥4 BMI measurements from birth to age 15 and at least one income measure between 1–24 months were eligible (n=927), with 818 included in propensity score (PS) estimation after exclusions. Household income was defined as income-to-needs ratio <1.85 vs. ≥1.85. AR age was estimated from mixed-effects cubic growth models and classified as early (<5 years) vs. later. PS matching (1:2 nearest neighbor with caliper) was conducted across 10 multiply imputed datasets, yielding ~288 matched participants. Modified Poisson regression with robust standard errors estimated risk ratios (RRs) of early AR.

Results: In the matched sample, low household income was not associated with early AR (RR=0.91; 95% CI: 0.71–1.17; p=0.47). There was no evidence of effect modification by sex (p interaction=0.71). Findings were consistent in regression models using the full analytic sample (RR=1.04; 95% CI: 0.86–1.25; p=0.69) and complete-case analyses (RR=0.97; 95% CI: 0.78–1.20; p=0.77). Sensitivity analyses excluding potential mediators and alternative SES indicators from the PS model yielded similar results.

Conclusions: Early childhood household income was not associated with the timing of adiposity rebound. These findings suggest that resource-based SES alone may not be a primary

determinant of early growth dynamics reflected by AR, highlighting the potential importance of more proximal behavioral or biological factors in shaping early-life obesity risk trajectories.

Introduction

There are significant benefits to studying longitudinal changes in a child's Body Mass Index (BMI) from infancy, including the ability to predict future risks of overweight or obesity more effectively.[1,2] One such indicator is the timing of adiposity rebound (AR).[3,4] AR refers to the second rise in BMI following its nadir around six months of age, typically occurring between ages 5 and 7.[5] Rolland first reported in 1984 that children who experienced AR before age 5.5 had higher adiposity at age 16 compared with peers whose rebound occurred after age 7.[6] Subsequent studies have consistently shown that early AR is associated with obesity in later childhood and adolescence,[7,8] as well as with other adverse health outcomes, including metabolic syndrome during adolescence,[9] Type 2 diabetes in adulthood,[10] dyslipidemia,[11] and insulin resistance.[12]

Although recently a study has shown that a child's BMI at age 3 predicted later adiposity more strongly than AR age alone,[13] this comparison may obscure an important distinction: early BMI captures children already trending toward overweight, whereas AR age identifies risk in children whose weight appears normal at the time of AR.[14] As such, monitoring AR timing may therefore help identify children at elevated risk during early childhood, potentially before excess weight becomes clinically evident.

Socioeconomic status (SES) is multidimensional, and its components may not relate equally to early growth processes. Weight trajectories are shaped by different constellations of influences across infancy, early childhood, and later childhood,[15,16] highlighting the importance of identifying which components of SES are most relevant during specific developmental windows. Parental education and occupation may reflect knowledge, social position, or longer-term opportunities, whereas household income more directly captures material resources available

during infancy and early childhood. These resources may shape early weight trajectories through pathways such as food security, diet quality, housing stability, and access to health-promoting environments.[17,18] Because adiposity rebound reflects growth dynamics that emerge before school age, household income may be a particularly relevant SES indicator for studying AR timing.

Evidence linking SES or proxies of SES, such as parental education level, to the timing of AR remains limited and inconsistent. Two of the earliest studies found no association between parental education attainment and early AR.[19,20] One study using a composite SES measure found that SES partially explained disparities in early AR observed among ethnic minority groups.[21] Two additional studies found significant associations between maternal education attainment and early AR.[22,23] Although children living in lower-income households in the United States experience a disproportionate burden of overweight and obesity,[24–26] applying this pattern uniformly across all stages of childhood growth may obscure important developmental nuances. Examining household income specifically may help clarify whether material disadvantage is associated with the timing of AR, rather than assuming all SES indicators operate similarly across childhood growth processes.

Much of the existing literature examining SES and AR relies on conventional regression-based approaches, which may be limited in their ability to address confounding in observational data adequately. SES is strongly correlated with a host of early-life social, behavioral, and environmental factors that influence childhood growth trajectories.[27,28] Failure to account for differences in these factors may contribute to biased or inconsistent findings across studies. Quasi-experimental methods, such as propensity score matching (PSM), offer an alternative framework by improving covariate balance between exposure groups and approximating the

conditions of a randomized comparison,[29,30] yet have not been applied to the study of SES and AR.

Using data from a longitudinal U.S. birth cohort, the current study examined the association between household income in early childhood, representing the resource-based dimension of SES, and the risk of early adiposity rebound. By applying PSM to balance socioeconomic and early-life characteristics across income groups, this study aimed to reduce confounding commonly present in observational analyses of SES and child growth.

Methods

Study Population

We used data from the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD), a longitudinal study of children's development from birth through age 26. Families were recruited at childbirth between 1991 and 1992 from 10 hospitals in the US. The starting cohort consisted of 1364 children. Details on recruitment and protocols are publicly available.[31] The current study included children with at least four valid BMI measurements from birth to age 15, calculated from measured height and weight (weight in kilograms divided by height in meters squared), as the minimal requirement to estimate AR timing, and at least one measurement of household income between 1 and 24 months of age (n=927).

The NICHD SECCYD was approved by the Institutional Review Board of the Eunice Kennedy Shriver National Institute of Child Health and Human Development and the institutional review boards at each participating study site. Written informed consent was obtained from parents or legal guardians of all participating children.

Exposure: Household Income

Household income was the exposure reflecting early-life resource-based SES context. Data on household income were recorded at 1, 6, 12, and 24 months of age. Non-missing data from these time points were averaged to create the exposure variable of early childhood household income. An income-to-need ratio, provided in the SECCYD dataset and calculated as the ratio of total household income to the federal poverty threshold for a family of that size and composition, was calculated based on the average household income. The exposure variable of this study was binary: income-to-need ratio <1.85 or ≥1.85, reflecting income eligibility thresholds (approximately 185% of the federal poverty level) commonly used in federal nutrition and health assistance programs during the study period.

Outcome: Early Adiposity Rebound

We estimated each child's AR age using longitudinal BMI trajectories modeled with mixed-effects cubic polynomials. Boys and girls were modeled separately, with log(BMI) specified as a function of age (centered at 5 years) and including random intercepts and random slopes up to the cubic term. This yielded individual-level predicted BMI trajectories of the form where A is the centered age:

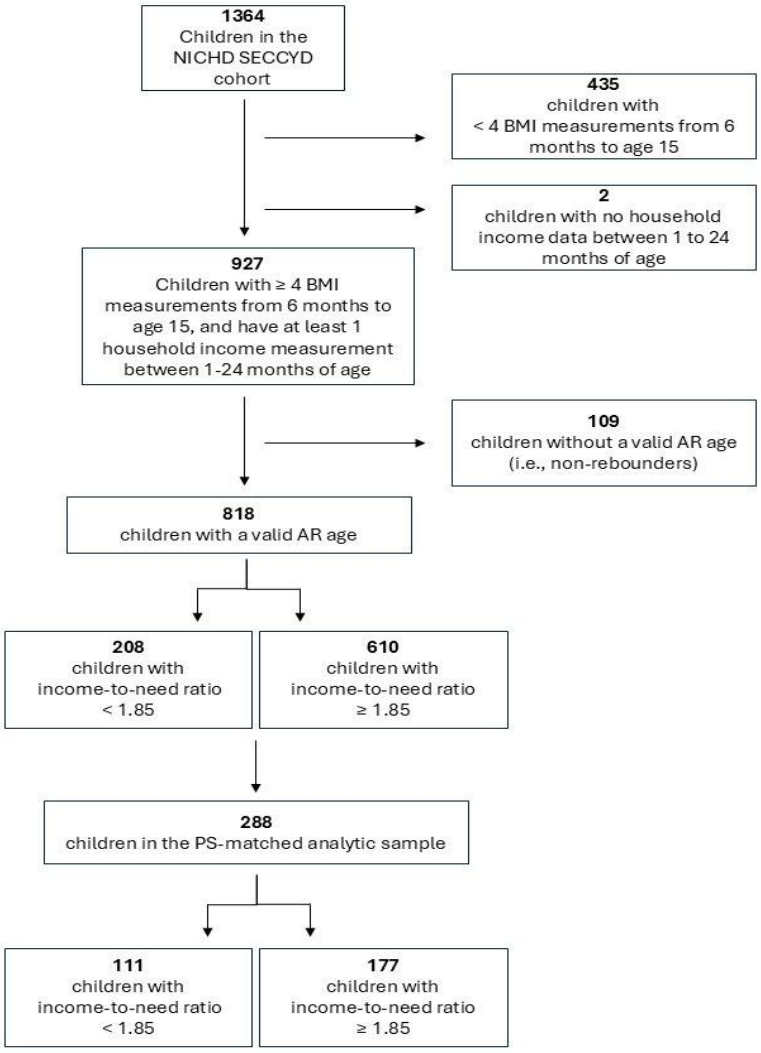
$$\widehat{logBMI}_i(A) = \beta_{0i} + \beta_{1i}A + \beta_{2i}A^2 + \beta_{3i}A^3$$

AR is defined as the age at which BMI reaches its nadir before increasing again and is conventionally interpreted as the minimum point of a child's BMI curve. To obtain consistent and visually verifiable AR estimates, we adopted a grid-search method. For each child, model-predicted log(BMI) was evaluated on a dense age grid from 2 to 9 years in 0.05-year increments, and AR was defined as the grid point yielding the minimum predicted BMI. This method yields minima that align with visual inspection of growth curves.

The biological definition of AR requires the presence of a BMI nadir, that is, a period where

BMI decreases during early childhood and subsequently rises again. A child whose BMI curve monotonically increases (or, rarely, decreases) over the entire age range does not exhibit a measurable AR. To identify such cases, we evaluated each child's predicted BMI curve over the 2–9-year grid and flagged trajectories that showed no decrease at any point. Consistent with prior literature, children with monotonic BMI trajectories were classified as non-rebounders and excluded from analyses (n=109, Figure 1).

Figure 1 Flow diagram of participant selection



After excluding non-rebounders, the distribution of AR ages matched prior epidemiologic studies (typically around 5–7 years).[4,32] For quality assurance, we produced individual-level plots overlaying observed BMI, model-predicted curves, and the identified AR point. In all cases, the observed AR age corresponded to the lowest point of the smooth model-predicted trajectory. Figure 2 shows six sample graphs. Early AR is treated as a binary variable, <age 5 or ≥age 5.

Covariates

Covariates were selected a priori based on literature and conceptual relevance to both household income and children's weight trajectory. These included parents' early-life demographic and socioeconomic factors (parental education, occupation, race/ethnicity, and mother's marital status and age), children's early-life characteristics (birthweight, gestational age, temperament), and other contextual factors including parents' general health status, mother's perceived social support, parenting stress, beliefs in raising children, and scale of depression, and lastly, study site. All covariates were measured before age 2 to avoid adjustment for post-exposure variables. Multiple imputation addressed missing covariate data under the missing-at-random assumption (Paternal education = 6.8% missing; maternal and paternal occupation = 16.1% and 13.1% missing, respectively). We generated 10 imputed datasets using a model that incorporated the exposure, outcome, and all covariates.

Propensity Score Estimation and Matching

We estimated propensity scores (PS) for exposure to low household income using logistic regression, with low income (income-to-needs ratio < 1.85 or ≥ 1.85) as the dependent variable. Baseline sociodemographic, children's early-life characteristics, and other contextual factors discussed under covariates were used as predictors. PS were estimated separately within each of the 10 imputed datasets.

We trimmed observations with extreme PS outside [0.01, 0.99] to improve common support. We then conducted 1:2 nearest-neighbor matching without replacement using a caliper defined as 0.2 times the standard deviation of the PS. Each matched set consisted of one child living in a low-income household and up to two children from higher-income households with closely matched PSs.

We evaluated covariate balance between exposure groups before and after matching using standardized mean differences (SMDs) for both continuous and categorical variables. For each imputed dataset, we considered $|SMD| < 0.1$ to indicate good balance and $|SMD| < 0.2$ to be acceptable, consistent with recommendations in the PS literature.[33]

Statistical Analysis

To estimate the association between low household income and early AR, we fit a modified Poisson regression with robust standard errors in the PS–matched sample. Since the outcome, having an early AR is common in the sample 49.5%), the estimated odds ratios from logistic regression might be inflated and misleading. The Modified Poisson regression with robust error variance allows the estimation of relative risk while ensuring that the standard errors are correctly estimated.[34] Early AR was modeled as a function of low household income, including matched set fixed effects via a set-specific indicator. To assess effect modification, we fit the matched-set model including an interaction term with children's sex and household income. Risk ratios (RRs) and 95% confidence intervals are reported. Because the analysis was performed on multiple imputed datasets, we fit the outcome model separately in each of the 10 imputed and matched datasets and then combined effect estimates.

We performed two secondary analyses to evaluate the robustness of the primary findings. First, we fit a regression model in the unmatched full analytic sample, adjusting for parental education,

child's race/ethnicity, sex, gestational age, birthweight, and study site. Second, we conducted a complete-case analysis, restricting to children with non-missing data on Early AR, early-life household income, and the covariates listed above, and fitting the same regression model. All secondary analyses used modified Poisson regression with robust standard errors for comparability to the primary matched analysis.

Sensitivity Analyses

Sensitivity analyses were conducted to assess the robustness of the findings to alternative model specifications, particularly with respect to covariate selection in the PSM. First, to evaluate the potential for overadjustment, we re-estimated the PSM after excluding early childhood characteristics that may plausibly lie on the causal pathway between household income and AR. These included maternal depression, parenting stress, parenting beliefs, perceived social support, parental general health status, and child temperament. Although these factors are often treated as confounders, their temporal positioning and potential role as mediators remain uncertain; thus, excluding them allowed us to assess whether results were sensitive to possible overadjustment bias. Second, we conducted an additional sensitivity analysis excluding parental education and occupation from the PS model. These variables are closely correlated with household income and may represent alternative indicators of SES rather than independent confounders. Removing them from the matching procedure allowed us to evaluate whether the observed associations were robust to different operationalizations of socioeconomic conditions.

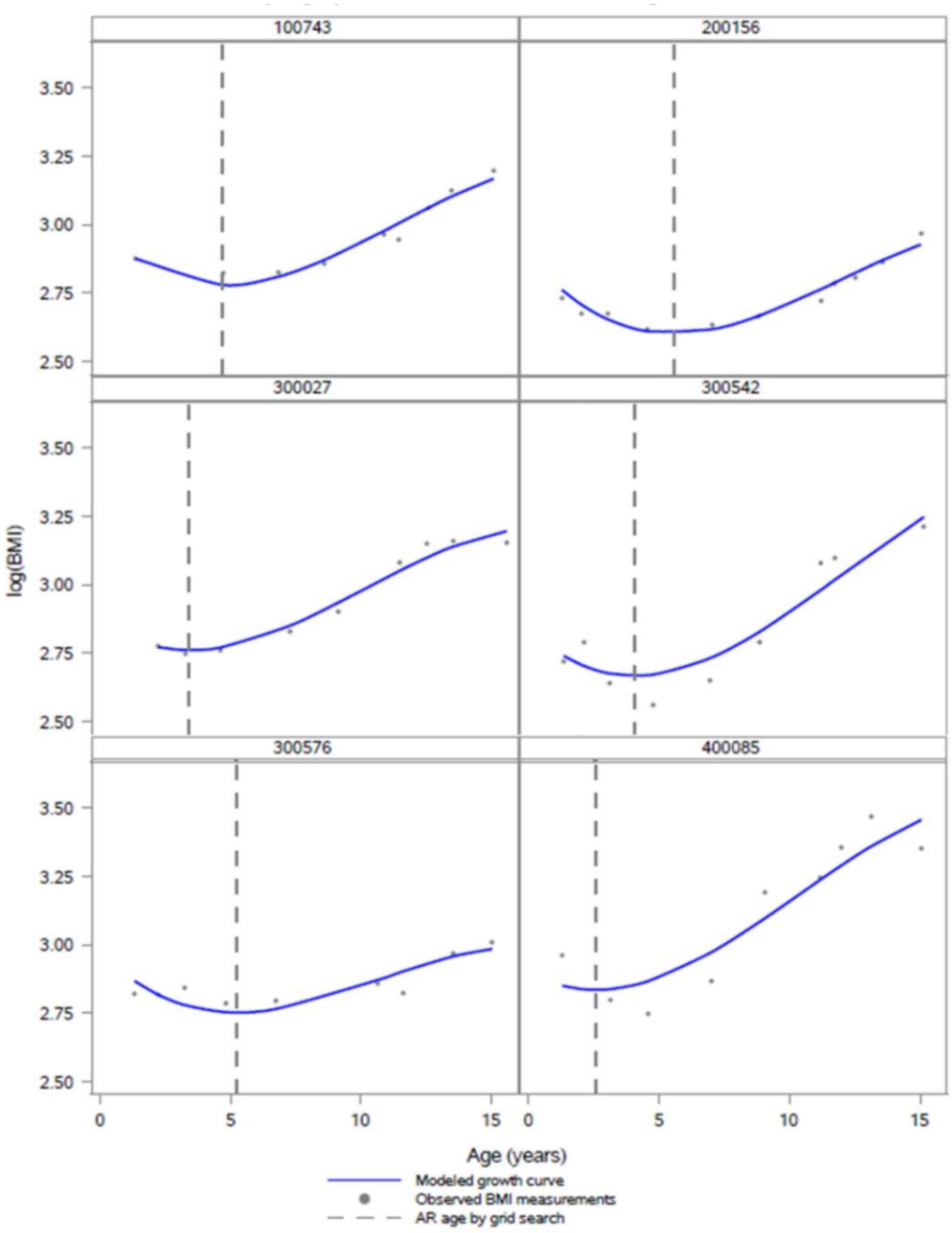
All statistical tests were 2-sided with $\alpha = .05$. Analyses were conducted with SAS, version 9.4 (SAS Institute Inc.).

Results

A total of 818 children with a valid AR age were included in the PS calculation.

PS matching was conducted within each imputed dataset. Across imputations, with an intended 1:2 matching ratio, a total of 111 children living in low-income households were matched with an average of 1.6 children from higher-income households, resulting in a final matched sample of approximately 288 children, with slight differences across imputations. Figure 1.2 provides a flow chart of the study sample.

Figure 2 Sample graphs of modeled growth curve, observed BMI measurements, and AR age



AR age is indicated by the dashed vertical line. Dots represent observed BMI measurements; the solid blue line represents the modeled growth curve. Six representative children are shown.

Table 1 shows baseline characteristics of children eligible for PS matching. Of the 818 children included in the PS estimation, 208 (25.4%) lived in households with an income-to-need ratio

<1.85, 635 (77.6%) were non-Hispanic Whites, and slightly more than half were female (50.7%). The average AR age was 4.8 ± 1.1 (boys: 5.0 ± 1.1; girls: 4.7 ± 1.1). With < age 5 defined as early AR in the current study, 405 (49.5%) children had an early AR.

Table 1 Baseline characteristics of children included in the propensity score estimation sample, by early adiposity rebound status

Characteristic	No. (%)	Early AR (n [%])	Normal AR (n [%])
Overall (n = 818)		405	413
Child's Race/Ethnicity			
Non-Hispanic White	635 (77.6)	304 (75.1)	331 (80.2)
Black or Afro-American	93 (11.4)	53 (13.1)	40 (9.7)
Hispanic	50 (6.1)	27 (6.7)	23 (5.6)
Other	40 (4.9)	21 (5.2)	19 (4.6)
Child's Sex			
Male	403 (49.3)	169 (41.7)	234 (56.7)
Female	415 (50.7)	236 (58.3)	179 (43.3)
Household Income-to-Need Ratio			
<1.85	208 (25.4)	112 (27.7)	96 (23.2)
≥1.85	610 (74.6)	293 (72.4)	317 (76.8)
Gestational age, mean (SD)	39.3 ± 1.4	39.4 ± 1.5	39.2 ± 1.5
Child's birthweight (g), mean (SD)	3479.3 ± 505.4	3525.7 ± 511.0	3433.9 ± 496.4
Maternal Marital Status			
Living with spouse or partner	720 (88.0)	347 (85.7)	373 (90.3)
Others	98 (12.0)	58 (14.3)	40 (9.7)
Maternal Education Level			
No college degree	492 (60.1)	260 (64.2)	232 (56.2)
College degree or higher	326 (39.9)	145 (35.8)	181 (43.8)
Paternal Education Level			
No college degree	440 (53.8)	227 (56.1)	213 (51.6)
College degree or higher	322 (39.4)	148 (36.5)	174 (42.1)
Missing	56 (6.8)	30 (7.4)	26 (6.3)

AR = adiposity rebound; early AR defined as AR occurring before age 5.

Covariate balance of the analytical sample before and after PS matching is presented in Table 2.

Table 2 Baseline characteristics among children living in low-income and non low-income households before and after PSM

	Before PSM		Std-diff.	After PSM		Std-diff.
	Low-income (n=207)	Non low-income (n=541)		Low-income (n=111)	Non low-income (n=177)	
Maternal age, mean (SD)	24.4 ± 5.2	29.5 ± 4.6	1.04	26.2 ± 5.3	27.0 ± 4.7	0.15
Maternal education — college degree+	15 (7.3%)	233 (44.2%)	0.96	15 (13.4%)	28 (15.6%)	0.06
Maternal marital status — with spouse/partner	133 (64.3%)	502 (95.5%)	0.84	93 (83.2%)	158 (88.4%)	0.15
Maternal race — Non-Hispanic White	133 (64.3%)	452 (85.9%)	0.58	80 (71.2%)	135 (75.4%)	0.20
Maternal general health — Excellent	64 (30.9%)	289 (54.9%)	0.59	38 (33.9%)	65 (36.2%)	0.08
Paternal education — college degree+	22 (10.8%)	232 (44.2%)	0.81	19 (17.3%)	35 (19.8%)	0.06
Child's birthweight (g), mean (SD)	3462.8 ± 518.1	3486.4 ± 506.1	0.05	3481.4 ± 532.3	3510.7 ± 506.6	0.06

PSM = propensity score matching; Std-diff. = standardized mean difference. |SMD| < 0.1 indicates good balance; |SMD| < 0.2 is acceptable.

Before PSM, mothers' mean age was 24.4 ± 5.2 in the low-income group and 29.5 ± 4.6 in the higher-income group. The higher-income group had higher proportions of the following characteristics compared to the low-income group: mothers and fathers with a college degree or above (mother: 44.2% vs 7.3%; father: 44.2% vs 10.8%), mothers living with spouse or partner (94.5% vs 64.3%), and mothers being non-Hispanic Whites (85.9% vs 64.3%). After matching and trimming, all covariates had an average |SMD| < 0.2 across imputations (Table 2).

Based on the matched sample, having a household income-to-need ratio <1.85 is not associated with early AR (RR=0.91; CI, 0.71–1.17) (Table 3). The interaction between household income and sex was not significant ($P = 0.71$), indicating no statistically meaningful sex differences. The conventional regression model fitted with the full analytical sample (n = 818) yielded similar

results, indicating no significant association between household income and early adiposity rebound (RR = 1.04; CI, 0.86–1.25). The complete case analysis (n = 609) also gave similar results (RR = 0.97; CI, 0.78–1.20).

Table 3 Association of early childhood low household income and early adiposity rebound

Analysis	Sample	N	RR/aRR (95% CI)	p-value
Propensity Score Matching	Matched sample	~288	0.91 (0.71–1.17)	0.47
p for sex × exposure interaction			0.71	
Regression — full analytic sample	Full sample	818	1.04 (0.86–1.25)	0.69
Complete-case analysis	Complete cases	609	0.97 (0.78–1.20)	0.77

[a]Early AR defined as before age 5. [b]RR = risk ratio estimated from modified Poisson regression with robust standard errors. Matched analysis includes matched-set fixed effects. N across imputations varies slightly.

Findings from the sensitivity analyses were consistent with the primary results. Excluding early childhood characteristics that may plausibly lie on the causal pathway between household income and adiposity rebound did not significantly change the estimated associations (Table 1.4). Similarly, further removing parental education and occupation from the PS model yielded comparable results (Table 4).

Table 4 Association of early childhood low household income and early adiposity rebound[a]

Sample	N	Model 1[b] RR (95% CI)	Model 2[c] RR (95% CI)
Matched analytical sample	288[d]	0.84 (0.63-1.11)	0.87 (0.62-1.12)

[a]Early adiposity rebound defined as before age 5.

[b]Propensity score matching performed removing the following early-life characteristics from the main analysis: maternal depression, maternal parenting stress, maternal parenting beliefs, maternal perceived social support, parental general health status, and child's temperament.

[c]Propensity score matching performed further removing the following variables from model 1: parental education level and parental occupation.

[d]N across imputations vary slight.

Discussion

Using a quasi-experimental approach based on PS matching, the present study found no association between low household income in early childhood and early AR. By improving covariate balance across a broad set of early-life social, parental, and contextual factors, this analysis more directly isolates the association between resource-based SES and AR timing than prior regression-based studies.

The null association observed here is largely consistent with evidence linking AR timing to SES measures that more closely reflect material resources. Specifically, SES indicators such as parental occupation[19,20] and household food security[23] have shown no association with AR age. In contrast, studies reporting significant associations between SES and AR often utilized composite SES indices[35] or SES proxies that less closely capture resource-based dimensions, such as maternal educational attainment.[23] Together, these patterns suggest that associations between SES and AR may be driven less by household income per se and more by correlated social or behavioral factors.

The null association between household income and early AR after accounting for these characteristics lends further support to the hypothesis that proximal caregiving practices, such as regulating screen time and discouraging sedentary behaviors as discussed earlier, may exert a stronger influence on early weight trajectories than income-based resources during this developmental period. Parental control over feeding has been linked to earlier AR,[36] whereas breastfeeding and regular bedtime routines have been associated with later AR.[4,37] In the present study, the PS model explicitly balanced groups on parenting-related factors, including parenting beliefs and parenting stress.

The developmental timing of AR may also help explain the lack of association observed. AR typically occurs between ages 5 and 7, a period during which the potential benefits of higher household income for structured recreation may be limited. While increased income could facilitate access to healthier foods, opportunities for income-related investments in physical activity emerge later in childhood. Conversely, higher household income may be a double-edged sword to sedentary-promoting resources. A study found there is a higher chance for children from higher SES households to have a television in their bedroom, which leads to more screen time;[38] on the other hand, evidence also speaks to a mixed pattern of how parents from different SES households regulate screen time for their children, such as more time spent on electronic tablets in children from higher SES households. These countervailing pathways related to parenting practices may attenuate any net association between income and early AR.

There has been a longstanding interest in identifying critical periods for obesity development, as these windows offer opportunities for early prevention and insight into underlying mechanisms. Continued efforts to identify modifiable determinants of early AR are essential for addressing pediatric obesity, a key upstream risk factor for chronic conditions, including metabolic syndrome,[39] cardiovascular disease,[40] and type 2 diabetes.[1]

Strengths and limitations

This study has several notable strengths. To our knowledge, it is the first investigation of SES and AR timing to employ a quasi-experimental design to address confounding in observational data. By incorporating parenting-related factors into the PS model, the analysis provides an assessment of the independent association between resource-based SES and AR age, separating

income from correlated caregiving characteristics. In addition, AR age was estimated using growth curve modeling, a gold-standard approach for identifying the nadir of BMI trajectories.[32] Several limitations should be acknowledged. The study sample was predominantly non-Hispanic White (77%), which may limit generalizability to more racially and ethnically diverse populations. In addition, although longitudinal BMI data were available from birth through age 15, measurements in early childhood were collected at annual intervals beyond the 6-month measure. More frequent assessments during the early years could yield more precise estimates of AR timing. The analytic sample size may also have limited statistical power to detect modest associations. Lastly, data collection for this cohort began in 1990. Given substantial changes in childhood contexts since that time, the generalizability of these findings to present-day settings may be limited, though the study's focus on early developmental processes may still provide insight into pathways linking socioeconomic conditions to later health outcomes.

Conclusion

Using propensity scores to better account for confounding, the present study found no association between resource-based SES, as measured by household income, and early AR. These findings suggest that household income alone may not be a primary driver of early AR and highlight the importance of considering developmental timing and specific social and behavioral pathways when designing early-life obesity prevention strategies.

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Author contributions:

J.W.Y.K. conceived the study, developed the analytic strategy, conducted the statistical analyses, and drafted the manuscript. L.J., D.D.P., and A.O.O. contributed to study design and conceptual development, provided guidance on analytic approach and interpretation of findings, and critically revised the manuscript for important intellectual content. All authors contributed to the interpretation of results, reviewed the manuscript, and approved the final version.

Competing interests:

The authors declare no competing financial or non-financial interests.

Data availability statement:

The data that support the findings of this study are available from the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD). These data are publicly available to qualified researchers upon request through the NICHD Data and Specimen Hub (DASH) repository (https://dash.nichd.nih.gov/), subject to relevant data use agreements and ethical approvals. The analytic code used in this study is available from the corresponding author upon reasonable request.

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CHAPTER 2: Beyond a Single Construct: Domain-Specific Early Childhood Self-Regulation and Weight Outcomes at Age 15

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Abstract

Background: Self-regulation (SR) has been associated with pediatric obesity risk, but studies frequently aggregate across SR domains, potentially obscuring domain-specific relationships. This study examined whether three early childhood SR domains — cool SR, food-related hot SR, and nonfood hot SR — are differentially associated with overweight/obesity (OWO) at age 15, and whether associations vary by sex.

Methods: Data from 698 children in the NICHD SECCYD with non-missing SR measures and BMI at age 15 were analyzed. Modified Poisson regression with robust standard errors and site-clustered SEs estimated adjusted relative risks (aRRs) for each SR domain. Sex interaction tests and stratified analyses were conducted.

Results: Cool SR (per 10-point increase) was associated with a 4% reduction in OWO risk at age 15 (aRR=0.96; 95% CI: 0.93–0.99). Passing the food hot SR task was associated with a 22% lower OWO risk (aRR=0.78; 95% CI: 0.64–0.96), with the association concentrated among boys (aRR=0.64; 95% CI: 0.52–0.78) but not girls. Nonfood hot SR showed no significant association (aRR=0.94; 95% CI: 0.81–1.09).

Conclusions: Early childhood SR domains are differentially associated with adolescent OWO risk. The divergence between food-related and nonfood SR underscores the importance of contextual reward sensitivity. These findings offer novel insights for domain-specific pediatric obesity interventions.

Introduction

Despite ongoing public health efforts, pediatric obesity in the US has climbed from 17.2% in 2013 to 21.1% in 2023,[1] sparking calls to declare it a public health emergency.[2] A disparity in obesity prevalence has also emerged among boys. In 2013, rates were nearly identical for boys and girls, but by 2023, prevalence in boys was 3.9 percentage points higher at 23.0% compared to girls at 19.1%.[1] About 70% of children with obesity carry the excess weight into adulthood,[3] setting the stage for metabolic syndrome,[4] cardiovascular disease,[5] type 2 diabetes,[6] certain cancers,[7,8] and mental health issues.[9]

Self-regulation (SR), a child's ability to control behavior, attention, and emotions to attain long-term goals,[10–12] has gathered solid evidence in understanding pediatric obesity and informing interventions. Lower SR in children has been associated with greater risk of overweight or obesity (OWO)[13–16] and may influence energy-balance behaviors such as eating in the absence of hunger.[17,18] Prior studies using observational and behavioral measures of SR in early childhood have also found that poorer regulatory capacity is associated with higher weight status and obesogenic eating behaviors.[19] More recent work further suggests that SR may shape obesity risk through its influence on multiple behavioral pathways, particularly among youth exposed to psychosocial stressors or other adversities.[20] Although SR has a genetic basis,[21] it can be shaped through experience and intervention.[22]

Within the context of eating behavior, researchers have increasingly distinguished appetite self-regulation (ASR), the ability to regulate food intake in response to internal physiological cues (e.g., hunger and satiety) and external food-related stimuli, and is increasingly conceptualized as involving interactions between bottom-up reactivity to food cues and top-down regulatory control processes.[23] Conceptual frameworks now differentiate food-related SR from more

general self-regulatory processes, suggesting that self-regulation in childhood may include both general self-regulation (GSR) and ASR domains.[24]

Most prior studies combined SR measures into a composite score. But are all domains of SR equally relevant to obesity risk? Interventions leveraging SR for weight management have seen mixed results,[25–27] which may stem from treating SR as a single, uniform skill rather than distinct domains. Hot SR involves managing behavior in emotionally charged or reward-based situations and is commonly assessed with delay-of-gratification (DOG) tasks.[28] Hot SR can be further divided into food-related and nonfood-related domains. Measures such as the marshmallow test (food rewards) and the forbidden toy task (nonfood rewards) are often combined into a single construct, potentially obscuring contextual differences.[13,29] Cool SR, by contrast, governs behavior in emotionally neutral situations and is typically evaluated through executive function tasks such as the Continuous Performance Test (CPT), which measures attention and impulse control.[30]

One study differentiating hot and cool SR found distinct associations with eating behaviors.[31] Research on cool SR shows mixed results and often relies on cross-sectional data.[32–34] Hot SR assessments frequently blur food and nonfood tasks,[11,16,35] which can obscure differences in food-specific versus general SR responsiveness.[36–39] Reliance on composite measures limits understanding of the distinct roles of various domains of SR in pediatric OWO risk.[40–42] Sex differences in eating behavior and self-regulation have also been documented, with boys generally exhibiting stronger food approach behaviors and responsiveness to food cues, whereas girls often show earlier body image awareness and dietary restraint, reflecting interacting biological, psychological, and sociocultural influences.[43]

To address these gaps, we leveraged longitudinal data to 1) to examine whether three early childhood SR domains, cool SR, food hot SR, and nonfood hot SR, show distinct associations with adolescent OWO risk and 2) explore sex-specific patterns in SR domains and OWO outcomes.

Methods

Study Population

The National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD) was a longitudinal study of children's development from birth through age 26. It was originally designed to examine the effects of childcare modes on children's development and has been widely used for child health and human development research.

In 1991, families were recruited at childbirth from 10 hospitals in the US. Families were excluded if: 1) the mother was under 18 years old or could not communicate in English, 2) the newborn had visible disabilities at birth or was not discharged within 7 days, or 3) the family planned to move out of the catchment area within 3 years. The starting cohort consisted of 1364 families. Details on recruitment and protocols are available.[44]

The current study sample includes SECCYD children with non-missing measures for the three SR measures and BMI at 15 years of age (n=698).

Ethics Statement

The NICHD SECCYD was approved by the Institutional Review Board of xxx (redated institution information as instructed by editorial team) and the institutional review boards at each

participating study site. Written informed consent was obtained from parents or legal guardians of all participating children.

Exposure Variables

Primary exposures were cool SR, food hot SR, and nonfood hot SR assessed between 36 and 54 months of age. We selected SECCYD measures and mapped them to each SR domain based on their domain-specific nature a priori. eSupplement 1 provides measurement details.

Cool SR was assessed at 54 months using the Continuous Performance Test (CPT), which measures sustained attention and impulsivity in emotionally neutral contexts. The two CPT scores were combined and reverse-coded so that higher values indicate better cool SR (possible range 0–195). Cool SR was analyzed as a continuous variable in 10-point increments in all regression models to preserve statistical efficiency and avoid bias introduced by categorizing continuous measures. Food hot SR was assessed at 54 months of age using the marshmallow test. Children chose a preferred food from three options and were shown a large and a small quantity of the food. They were given a bell and told they could receive the larger quantity if they waited until the researcher returned. Children were grouped as “passed” or “failed” based on whether they waited the full 7 minutes.

Nonfood hot SR was measured at 36 months of age using the forbidden toy task. Children were asked not to touch an age-appropriate toy placed within arm's reach while the researcher left the room for 2.5 minutes. Children were grouped as “passed” or “failed” based on whether they touched the toy while the researcher was away. In SECCYD, both hot SR tasks were administered and coded live by trained assessors during the visit, with videotapes used only for reliability checks. The food and nonfood hot SR tasks are validated NICHD SECCYD measures of delay of gratification (DOG) and hot inhibitory control, with established construct and

predictive validity in early childhood.[44–46] Both tasks are conventionally scored as binary pass/fail indicators to reflect successful inhibition across the full delay interval, consistent with classic DOG paradigms.[47,48]

Outcome Variable

At age 15, height and weight were collected during a laboratory visit. Height was recorded in inches to the nearest one-eighth inch and weight in pounds and ounces. We classified children with BMI z-scores greater than 1.036 and less than 1.645 (>85th to <95th percentile, age and sex-adjusted) as having overweight, and children with BMI z-scores at or greater than 1.645 (≥95th percentile, age and sex-adjusted) as having obesity, based on the CDC 2000 BMI-for-age growth chart definitions.[49] Weight status at age 15 was treated as a binary variable: normal weight and OWO.

Covariates

Covariates were selected a priori based on prior literature on children's SR and weight trajectory.[13,19,25,50,51] Sociodemographic information was reported by mothers when children were 1 month old: maternal education (no college degree vs. college degree and above), total household income-to-need ratio (continuous, log-transformed), and child's ethnicity (Non-Hispanic White, Non-Hispanic Black or African-American, Hispanic, American Indian, Eskimo, Aleutian, Asian, Pacific Islander, or other).

Maternal and paternal OWO statuses were classified based on weight and height reported by mothers at the age 15, using BMI greater than 25 to define OWO. Children's pubertal status was determined by a nurse practitioner or doctor during the 15-year-old physical development assessment using Tanner stage criteria. Girls were assessed in terms of breast development and boys in terms of genital development. Because biologically, pubertal status does not reverse, if a

child had a score of 5 at a previous visit, this score was carried forward to age 15 if data was missing. A binary variable indicated fully developed (stage 5) vs. not fully developed (stages 1–4). Age in months at assessment and sex at birth were included as covariates.

Statistical Analysis

Our analytical sample included 698 children with non-missing data on all three SR domains and BMI at age 15. We fit separate modified Poisson regression models with robust standard errors to examine associations between each SR domain (cool SR, food hot SR, and nonfood hot SR) and OWO status at age 15. Standard errors were clustered by site to account for potential unmeasured site-level confounding. All covariates identified a priori based on prior SECCYD studies were included in each model.

Cool SR was analyzed as a continuous variable. We evaluated potential nonlinearity by adding a quadratic term. We also tested interaction terms between sex at birth and each SR measure to assess sex-specific associations. Because interaction terms may not fully reflect sex-specific effect sizes, we additionally conducted sex-stratified analyses and report results for boys and girls separately. Results are presented as adjusted relative risks (aRRs) with 95% confidence intervals (CIs).

To assess robustness, we conducted sensitivity analyses using waist-to-height ratio to represent OWO status. Waist-to-hip ratio was calculated as waist circumference divided by hip circumference, with waist measured at the level of the natural waist and hip circumference measured at the widest portion of the hips. We also removed paternal OWO status as a covariate, given uncertain accuracy, as fathers' heights and weights were reported by mothers. Finally, we included a complete-case analysis.

Multiple imputation addressed missing covariate data under the missing-at-random assumption. We generated 10 imputed datasets using a model that incorporated the three exposures, outcome, all covariates, as well as auxiliary variables (pubertal status from ages 9–14 and income-to-need ratio at 15 and 36 months) to improve estimation. All statistical tests were 2-sided with $\alpha = .05$. Analyses were conducted with SAS, version 9.4 (SAS Institute Inc.).

Results

The analytic sample included 698 children. Of the 698 participants, 208 (29.8%) had OWO at age 15. Prevalence was 33.8% among boys and 26.2% among girls. 47.4% were male; 107(15.3%) of families had an income-to-need ratio <1 at 1 month postpartum (Table 5).

Table 5 Sociodemographic Characteristics of Analytical Sample by Age 15 OWOB Status[a]

Characteristic	No. (%)	Have OWO (n [%])	Normal weight (n [%])
N (all)	698 (100.0)	208 (29.8)	490 (70.2)
Race/Ethnicity			
Non-Hispanic White	554 (79.4)	153 (73.6)	401 (81.8)
Black or Afro-American	81 (11.6)	36 (17.3)	45 (9.2)
Hispanic	35 (5.0)	9 (4.3)	26 (5.3)
Other	28 (4.0)	10 (4.8)	18 (3.7)
Sex			
Male	331 (47.4)	112 (53.9)	219 (44.7)
Female	367 (52.6)	96 (46.1)	271 (55.3)
Income-to-Need Ratio			
<1.0	107 (15.3)	46 (22.1)	61 (12.5)
1.0–2.0	142 (20.3)	58 (27.9)	84 (17.1)
>2.0	409 (58.6)	88 (42.3)	321 (65.5)
Missing	40 (5.7)	16 (7.7)	24 (4.9)
Maternal Education			
No college degree	420 (60.2)	150 (72.1)	270 (55.1)
College degree or higher	278 (39.8)	58 (27.9)	220 (44.9)
Maternal OWO			
Yes	369 (52.9)	144 (69.2)	225 (45.9)
No	297 (42.6)	47 (22.6)	250 (51.0)
Missing	32 (4.6)	17 (8.2)	15 (3.1)
Paternal OWO			
Yes	464 (66.5)	157 (75.5)	307 (62.7)
No	182 (26.1)	31 (14.9)	151 (30.8)
Missing	52 (7.4)	20 (9.6)	32 (6.5)
Pubertal Status			
Tanner stage 1–4	90 (12.9)	20 (9.6)	70 (14.3)
Tanner stage 5	429 (61.5)	141 (67.8)	288 (58.8)
Missing	179 (25.6)	47 (22.6)	132 (26.9)

OWO = overweight or obesity. Overweight or obesity defined by BMI z scores >1.036 to <1.645 (>85th to <95th percentile) as overweight, and ≥1.645 (≥95th percentile) as obesity, age and sex-adjusted.

Distributions of the three SR types are shown in Table 6. Girls consistently performed better on SR tasks than boys.

Table 6 Early Childhood Self-Regulation Measures in the Analytic Sample (n = 698)

Exposure	Metric	Overall	Boys	Girls
Cool SR	Mean (SD)	137.0 (22.0)	131.9 (25.2)	141.5 (17.5)
	Median (IQR)	144.4 (130.4–152.4)	139.4 (123.4–149.4)	147.4 (135.4–152.4)
	Range	21.7–158.4	21.7–158.4	35.4–158.4
Food Hot SR	Passed, n (%)	377 (54.0)	172 (52.0)	205 (55.9)
	Failed, n (%)	321 (46.0)	159 (48.0)	162 (44.1)
Nonfood Hot SR	Passed, n (%)	299 (42.8)	121 (36.6)	178 (48.5)
	Failed, n (%)	399 (57.2)	210 (63.4)	189 (51.5)

Cool SR is modeled continuously (per 10-point increase). Hot SR tasks are binary pass/fail indicators.

Of the 698 children with BMI measured at age 15, 49.9% had at least one missing covariate value. Complete-case models included 410 cases. Compared to the analytic sample, children with missing outcome data were more likely to be male, have low income-to-need ratios, both parents having overweight and obesity, and mothers without a college degree (Table 7).

Table 7 Sociodemographic Characteristics of SECCYD Cohort participants included in the analytic sample compared to those not included in the analytic sample

	Analytic sample No. = 698	Not in Analytic Sample No. = 666	*P* value	Missingness (%)
Characteristic	No. (%)			
Race/Ethnicity			0.050	0.0
Non-Hispanic White	554 (79.4)	488 (73.3)		
Black or Afro-American	81 (11.6)	92 (13.8)		
Hispanic	35 (5.0)	48 (7.2)		
Other	28 (4.0)	38 (5.7)		
Missing	0 (0.0)	0 (0.0)		
Sex			0.001	0.0
Male	331 (47.4)	374 (56.2)		
Female	367 (52.6)	292 (43.8)		
Missing	0 (0.0)	0 (0.0)		
Income-to-Need Ratio			<0.001	6.7
<1.0	107 (15.3)	166 (24.9)		
1.0–2.0	142 (20.3)	150 (22.5)		
>2.0	409 (58.6)	299 (44.9)		
Missing	40 (5.7)	51 (7.7)		
Maternal Education			0.001	0.1
No college degree	420 (60.2)	461 (69.2)		
College degree or higher	278 (39.8)	204 (30.6)		
Missing	0 (0.0)	1 (0.2)		
Maternal Overweight/Obese			<0.001	31.2
Yes	369 (52.9)	124 (18.6)		
No	297 (42.6)	148 (22.2)		
Missing	32 (4.6)	392 (59.2)		
Paternal Overweight/Obese			<0.001	33.7
Yes	464 (66.5)	193 (29.0)		
No	182 (26.2)	65 (9.8)		
Missing	52 (11.3)	408 (61.2)		
Pubertal Status			<0.001	49.9
Tanner staging 1-4	90 (13.0)	24 (3.6)		
Tanner staging 5	429 (61.5)	140 (21.0)		
Missing	224 (26.5)	502 (75.4)		

In the adjusted models, early childhood cool SR and food hot SR were significantly associated with OWO risk at age 15; nonfood hot SR was not (Table 8).

Table 8 Association of Self-Regulation Types in Early Childhood to Overweight or Obesity Status at Age 15

Exposure	Sample	N	Unadjusted OWO %	aRR (95% CI)	*p* interaction
Cool SR (per 10-pt increase)	Full sample	698	29.8	0.96 (0.93–0.99)	
	Boys	331	33.8	0.96 (0.93–0.99)	0.14
	Girls	367	26.2	0.97 (0.92–1.02)	
Food Hot SR (pass vs fail)	Full sample	698	Failed: 37.4 / Passed: 23.3	0.78 (0.64–0.96)	
	Boys	331	Failed: 44.7 / Passed: 23.8	0.64 (0.52–0.78)	0.24
	Girls	367	Failed: 30.3 / Passed: 22.9	1.00 (0.64–1.55)	
Nonfood Hot SR (pass vs fail)	Full sample	698	Failed: 32.6 / Passed: 26.1	0.94 (0.81–1.09)	
	Boys	331	Failed: 36.7 / Passed: 28.9	0.85 (0.67–1.08)	0.63
	Girls	367	Failed: 28.0 / Passed: 24.2	0.96 (0.76–1.22)	

aRR = adjusted relative risk estimated from modified Poisson regression with robust standard errors, clustered by site. Adjusted for child sex, race/ethnicity, household income-to-need ratio (log-transformed), maternal education, maternal and paternal OWO, pubertal status, and age at BMI assessment. [a]Age at SR task: cool SR and food hot SR assessed at 54 months; nonfood hot SR assessed at 36 months.

Each 10-point increase in cool SR at 54 months was associated with a 4% reduction in OWO risk at age 15 (aRR=0.96; CI, 0.93–0.99). The interaction between cool SR and sex at birth was not significant, indicating no statistically meaningful sex differences. In sex-stratified models, the direction of association was similar for boys but was only statistically significant for boys. Children who passed the food hot SR task had a 22% lower risk of OWO (aRR=0.78; 95% CI, 0.64–0.96). The sex interaction term was not statistically significant, indicating no statistically detectable difference in the association by sex in formal interaction testing. In sex-stratified analyses, the protective association was evident only among boys (aRR=0.64; 95% CI, 0.52–

0.78) and was not observed among girls (aRR=1.00; 95% CI, 0.64–1.55). Nonfood hot SR at 36 months showed no significant association with OWO risks at age 15 (aRR=0.94; 95% CI, 0.81–1.09).

Sensitivity analyses removing paternal OWO status showed similar magnitudes and significance across all SR domains (Table 9).

Table 9 Association of Self-Regulation Types in Early Childhood[a] to Overweight or Obesity Status[b] at Age 15: sensitivity analyses excluding paternal weight status

Exposure	Sample	Age	N[d]	Exposure metric	aRR (95% CI)[e]
Cool SR[c]	Full sample	54 mo	698	Per 10-point increase	0.96 (0.93-0.99)
	Boys[f]		331		0.96 (0.93-0.99)
	Girls[f]		367		0.97 (0.92-1.03)
Food Hot SR	Full sample	54 mo	698	Passed vs failed food delay task, ref: fail	0.78 (0.63-0.97)
	Boys[f]		331		0.62 (0.49-0.77)
	Girls[f]		367		1.01 (0.64-1.60)
Non-Food Hot SR	Full sample	36 mo	698	Passed vs failed wrapped-gift task, ref: fail	0.92 (0.80-1.06)
	Boys[f]		331		0.85 (0.68-1.05)
	Girls[f]		367		0.96 (0.75-1.22)

[a]The three self-regulation tasks were administered when children were between 36 months and 54 months of age.

[b]Overweight or obesity defined by BMI z scores > 1.036 and < 1.645 (>85th to < 95th percentile, age and sex-adjusted) as having overweight, and children with BMI z scores ≥ 1.645 (≥ 95th percentile, age and sex-adjusted) as having obesity.

[c]Associations for Cool SR reflect a linear specification (per 10-point increase); nonlinear terms were not retained.

[d]Number of participants in the analytical sample with non-missing data on all three SR measures and BMI measure at age 15.

[e]Model adjusted for child sex at birth (male or female), race/ethnicity (Non-Hispanic White, Black or Afro-American, Hispanic, or American Indian, Eskimo, Aleutian, Asian, Pacific Islander, and others), household income-to-need ratio at 1 month (continuous, log-transformed), maternal education level at 1 month postpartum (no college degree or college degree and above), maternal and paternal overweight or obesity status at child age 15 (yes or no overweight or obesity), pubertal status (Tanner stage 1-4, or Tanner stage 5), and child's age to the month at age 15 BMI assessment.

[f]Models stratified by sex at birth.

Using waist-to-height ratio of 0.5, a commonly used cut-off as a healthy ratio, as an alternative indicator of OWO, associations for all SR types remained similar in both magnitude and

significance (Table 10). These findings suggest that SR–weight associations were robust across adiposity measures.

Table 10 Association of Self Regulation Types in Early Childhood[a] to Overweight or Obesity Status[b] at Age 15: sensitivity analyses using waist-to-height ratio

Exposure	Sample	Age	N[c]	Exposure metric	aRR[d] (95% CI) of waist-to-height ratio >= 70 percentile	aRR[d] (95% CI) of waist-to-height ratio >= 0.5
Cool SR[e]	Full sample	54 mo	698	Per 10-point increase	0.98 (0.94-1.02)	0.94 (0.89-0.99)
	Boys[f]		331		0.98 (0.94-1.02)	0.93 (0.88-0.98)
	Girls[f]		367		0.97 (0.89-1.05)	0.97 (0.88-1.07)
Food Hot SR	Full sample	54 mo	698	Passed vs failed food delay task, ref: fail	0.69 (0.52-0.90)	0.58 (0.50-0.69)
	Boys[f]		331		0.63 (0.50-0.81)	0.49 (0.36-0.67)
	Girls[f]		367		0.72 (0.48-1.08)	0.65 (0.43-0.96)
Non-Food Hot SR	Full sample	36 mo	698	Passed vs failed wrapped-gift task, ref: fail	1.09 (0.82-1.15)	0.85 (0.56-1.30)
	Boys[f]		331		0.99 (0.70-1.39)	0.82 (0.42-1.61)
	Girls[f]		367		0.92 (0.69-1.22)	0.79 (0.48-1.28)

[a]The three self-regulation tasks were administered when children were between 36 months and 54 months of age.

[b]Overweight or obesity defined by BMI z scores > 1.036 and < 1.645 (>85th to < 95th percentile, age and sex-adjusted) as having overweight, and children with BMI z scores ≥ 1.645 (≥ 95th percentile, age and sex-adjusted) as having obesity.

[c]Number of participants in the analytical sample with non-missing data on outcome.

[d]Model adjusted for child sex at birth (male or female), race/ethnicity (Non-Hispanic White, Black or Afro-American, Hispanic, or American Indian, Eskimo, Aleutian, Asian, Pacific Islander, and others), household income-to-need ratio at 1 month (continuous, log-transformed), maternal education level at 1 month postpartum (no college degree or college degree and above), maternal and paternal overweight or obesity status at child age 15 (yes or no overweight or obesity), pubertal status (Tanner stage 1-4, or Tanner stage 5), and child's age to the month at age 15 BMI assessment.

[e]Associations for Cool SR reflect a linear specification (per 10-point increase); nonlinear terms were not retained.

[f]Models stratified by sex at birth.

In complete-case analysis, the association between cool SR is no longer statistically significant, while food hot SR remained statistically significant (Table 11).

Table 11 Association of Self Regulation Types in Early Childhood[a] to Overweight or Obesity Status[b] at Age 15: sensitivity analyses with complete cases

Exposure	Sample	Age	N[d]	Exposure metric	aRR (95% CI)[e]
Cool SR[c]	Full sample	54 mo	410	Per 10-point increase	1.00 (0.94-1.08)
	Boys[f]		193		1.05 (0.98-1.12)
	Girls[f]		217		0.96 (0.85-1.07)
Food Hot SR	Full sample	54 mo	410	Passed vs failed food delay task, ref: fail	0.70 (0.56-0.88)
	Boys[f]		193		0.55 (0.40-0.75)
	Girls[f]		217		0.83 (0.56-1.22)
Non-Food Hot SR	Full sample	36 mo	410	Passed vs failed wrapped-gift task, ref: fail	0.91 (0.74-1.12)
	Boys[f]		193		0.83 (0.59-1.17)
	Girls[f]		217		1.03 (0.75-1.40)

[a]The three self-regulation tasks were administered when children were between 36 months and 54 months of age.

[b]Overweight or obesity defined by BMI z scores > 1.036 and < 1.645 (>85th to < 95th percentile, age and sex-adjusted) as having overweight, and children with BMI z scores ≥ 1.645 (≥ 95th percentile, age and sex-adjusted) as having obesity.

[c]Associations for Cool SR reflect a linear specification (per 10-point increase); nonlinear terms were not retained.

[d]Number of complete cases with no missing values for exposures, outcome, and covariates.

[e]Model adjusted for child sex at birth (male or female), race/ethnicity (Non-Hispanic White, Black or Afro-American, Hispanic, or American Indian, Eskimo, Aleutian, Asian, Pacific Islander, and others), household income-to-need ratio at 1 month (continuous, log-transformed), maternal education level at 1 month postpartum (no college degree or college degree and above), maternal and paternal overweight or obesity status at child age 15 (yes or no overweight or obesity), pubertal status (Tanner stage 1-4, or Tanner stage 5), and child's age to the month at age 15 BMI assessment.

[f]Models stratified by sex at birth.

Discussion

Using longitudinal data, we found that early childhood SR domains were differentially associated with adolescent OWO at age 15. Cool SR and food hot SR were associated with lower OWO risk, while nonfood hot SR showed no association. This domain-specific pattern, particularly the divergence between food-related and nonfood SR tasks, underscores the

importance of contextual reward sensitivity in shaping eating behavior and weight outcomes. These findings suggest distinct SR domains influence children's weight trajectories, offering novel insights for childhood development and obesity interventions.

On a scale of 0–194 points, each 10-point increase in cool SR in early childhood was associated with a 4% reduction in OWO risk at 15 years of age, supporting a temporal association between cool SR and later weight status. Over a 50-point difference, roughly the span between children with very low vs. moderate cool SR, this corresponds to an approximate 20% lower risk. Prior studies on cool SR (ages 5–15) consistently show that higher inhibitory control and cognitive flexibility are related to lower BMI,[18,32,34] but most were cross-sectional studies. This is the first study to use a longitudinal cohort sample to examine these associations in cool SR.

The interaction between sex was not statistically significant with either cool SR or food hot SR, suggesting no definitive evidence of sex differences in the association. However, the stratified estimates showed a noteworthy pattern: passing the food hot SR task was associated with a substantially lower risk of adolescent OWO among boys, whereas the association was not statistically significant among girls. These patterns should be interpreted with caution, but they may reflect emerging evidence that boys and girls engage with appetitive cues differently across development. For example, adolescent girls may experience stronger sociocultural pressures around eating and body image that could attenuate earlier SR-related vulnerabilities, whereas boys may face fewer external constraints, allowing early SR deficits to translate into more persistent patterns of overeating.[52] Prior work also suggests that sex differences in children's eating behaviors may arise through a combination of biological, psychological, and social influences—including differences in parental feeding practices, body image concerns, and responsiveness to food cues—which may shape how appetitive regulation develops across

childhood. Emerging conceptual models further emphasize that appetite self-regulation reflects interactions between biologically driven food motivation and top-down regulatory processes that are shaped by developmental and socialization influences.[43,53] With evidence that SR may operate differently across sexes in the literature,[51,54–56] further research is needed to clarify whether these sex-specific patterns reflect true developmental differences or arise from measurement, contextual, or sociocultural factors.

Earlier studies distinguishing food from nonfood SR have suggested that DOG deficits are more pronounced for food rewards than for nonfood incentives when it comes to weight outcomes in children.[57,58] Our findings align with this pattern: food hot SR was significantly associated with weight outcomes, whereas nonfood hot SR was not. This distinction underscores the importance of identifying which SR domains are most relevant to obesity risk to better inform early intervention targets. However, the null association for nonfood hot SR should be interpreted with caution. The forbidden toy task was administered at an earlier developmental stage than the food delay task, when SR capacities are still rapidly emerging and may be less stable predictors of later outcomes. This may have limited the task's ability to detect associations with later weight outcomes.

These findings may help explain why SR-enhancing interventions have had mixed results. Lack of domain-specific granularity in intervention design and reporting makes it difficult to identify what works and for whom. Distinct SR domains may influence weight through different behavioral pathways; future research should investigate these mechanisms.

Taken together, our findings underscore the importance of moving beyond a unitary view of SR. A more nuanced understanding could inform tailored pediatric obesity interventions. For example, an intervention targeting food-related hot SR in children with severe obesity led to

significantly greater weight loss than controls over 8 weeks,[59] while another program that trained cool SR through game-based inhibition and working memory exercises also produced reductions in weight.[27] These interventions illustrate the promising value of focusing on specific SR domains. Future research should also examine potential sex-specific associations across SR domains. Such evidence could help design interventions focused on the most relevant SR domains for each group, maximizing impact and efficiently directing resources.

Another key research need is to clarify the mechanisms that link specific SR domains to weight trajectories. For food hot SR, resisting early appetitive temptations may promote healthier weight over time, though this is rarely tested. For cool SR, redirecting focus from temptations may support better eating habits and activity adherence. Mechanisms for nonfood hot SR are less clear, as difficulty resisting a toy may not translate to resisting palatable food.

Strengths and Limitations

This study has several strengths. We accounted for paternal weight status, a factor rarely controlled for in pediatric obesity research and included sensitivity analyses using waist-to-height ratio, conducted in recognition of debates about whether BMI adequately reflects body fat distribution and composition. These analyses yielded similar results, reinforcing the robustness of the observed associations across alternative adiposity indicators.

Study limitations include collapsing three race/ethnicity groups to avoid small cell sizes, which may have masked subgroup differences. Nonfood hot SR was assessed 18 months earlier than other domains, potentially affecting comparisons. In the absence of established guidelines, SR tasks were selected by authors to best represent each domain. Self-regulation can be operationalized using a variety of behavioral tasks and questionnaire-based measures, and

different operationalizations may capture distinct aspects of the construct, which may influence comparability across studies. Finally, the sample was predominantly non-Hispanic White (77%), which may limit generalizability to more diverse populations.

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Author Contributions:

J.W.Y.K. conceived the study, developed the analytic strategy, conducted the statistical analyses, and drafted the manuscript. L.J., D.D.P., and A.O.O. contributed to study design and conceptual development, provided guidance on analytic approach and interpretation of findings, and critically revised the manuscript for important intellectual content. All authors contributed to the interpretation of results, reviewed the manuscript, and approved the final version.

Competing Interests:

The authors declare no competing financial or non-financial interests.

Data Availability Statement:

The data that support the findings of this study are available from the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD). These data are publicly available to qualified researchers upon request through the NICHD Data and Specimen Hub (DASH) repository (https://dash.nichd.nih.gov/), subject to

relevant data use agreements and ethical approvals. The analytic code used in this study is available from the corresponding author upon reasonable request.

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CHAPTER 3: Socioeconomic Disparities and Self-Regulation in Childhood Weight Trajectory: Evidence from a Longitudinal Mediation Analysis

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Abstract

Objective: To examine whether domain-specific self-regulation (SR) in early childhood mediates the association between household income in infancy and overweight/obesity (OWO) in later childhood.

Design: Prospective cohort study using data from the NICHD Study of Early Child Care and Youth Development.

Participants: A total of 782 children with complete data on exposure, mediators, and outcome.

Exposure: Household income measured as the mean income-to-needs ratio (ITNR) from birth to 24 months, dichotomized at <1.85 versus ≥1.85.

Mediators: SR at 54 months, including cool SR (Continuous Performance Task; continuous) and food-related hot SR (delay of gratification; pass/fail).

Outcome: OWO at age 9 years, defined using CDC BMI-for-age criteria.

Methods: Causal mediation analysis was conducted to estimate total, natural direct, and natural indirect effects for each SR domain, adjusting for child and maternal characteristics.

Results: Children from low-income households had higher odds of OWO at age 9 (OR = 1.54, 95% CI: 1.02–2.33). Low income was associated with poorer SR across both domains. However, neither cool SR nor food-related hot SR was associated with OWO, and no evidence of mediation was observed, as all natural indirect effects were close to null.

Conclusions: We did not observe evidence that early childhood SR mediates the association between household income and later OWO. These findings suggest that socioeconomic disparities in childhood obesity may operate through pathways beyond early regulatory capacity, highlighting the role of broader structural and environmental influences.

Introduction

Childhood obesity remains a major public health concern in the United States, with prevalence reaching 21.1% in 2023.[1] Excess weight during childhood is associated with persistence into adulthood and increased risks of cardiometabolic and psychosocial complications.[2,3] Even among children with similar starting birthweights, children exhibit substantial heterogeneity in weight trajectories across development, underscoring the importance of early-life determinants of obesity risk.[4]

Socioeconomic status (SES) is consistently associated with childhood obesity, with children from lower-income households experiencing higher risk of overweight and obesity.[5,6] As an upstream determinant, SES shapes access to material and environmental resources that influence diet, physical activity, and overall health.[7,8] However, the mechanisms linking early-life socioeconomic disadvantage to later weight outcomes have not been explored. Conducting this work presents several challenges, including the need for longitudinal data with temporally ordered measures across development, the complexity of operationalizing self-regulation as a multidimensional construct, and the methodological demands of mediation analyses in developmental research. These challenges may partly explain why relatively few studies have examined self-regulation as a potential pathway linking early socioeconomic disadvantage to later obesity risk.

Self-regulation (SR), encompassing the ability to control attention, behavior, and emotions, has been proposed as one potential pathway.[9,10] SR develops rapidly in early childhood through age 3-7[9] and is sensitive to environmental influences, including chronic stress associated with low SES.[11,12] It has also been linked to energy balance–related behaviors, such as eating in the absence of hunger and physical activity, suggesting a role in shaping weight trajectories.[13–15]

Evidence regarding SR and pediatric obesity remains mixed, with inconsistent associations and equivocal findings from intervention studies.[16–18] One potential explanation is that SR is not a unitary construct and can refer to multiple related but distinct domains/processes. Distinctions between "cool" SR, reflecting executive control in emotionally neutral contexts, and "hot" SR, engaged in emotionally salient or reward-driven situations, have been increasingly emphasized.[19,20] Aggregating across these domains may obscure domain-specific relationships with weight-related behaviors, particularly as cool SR captures general regulatory capacities while food-related hot SR reflects the ability to delay gratification in the presence of appetitive cues directly relevant to eating. Yet, these domains have rarely been examined simultaneously in relation to weight outcomes.

At the same time, existing studies have largely examined relationships between SES, SR, and weight outcomes in isolation, linking SES to SR, SR to weight outcomes, or SES directly to weight outcomes, without evaluating the full pathway connecting these constructs. Although this body of work suggests that SR may plausibly link early-life socioeconomic conditions to later obesity risk, few studies have formally tested this mediating role, particularly using longitudinal data with appropriate temporal ordering of exposure, mediator, and outcome.[21,22] One study using cumulative risk exposure measured at age 9, which included indicators such as household income, maternal education, housing problems, and family turmoil, found that food-related SR significantly mediated the association between cumulative risk and BMI at age 13.[21] In contrast, another study of children aged 9 to 14 years did not identify a significant mediating role of SR-related healthy eating behaviors in the relationship between household income and BMI.[22] However, evidence from these studies remains limited, as both relied on cross-sectional

assessments that restrict conclusions regarding temporality, and both examined only a single dimension of SR.

To address these gaps, this study uses data from the NICHD Study of Early Child Care and Youth Development to examine whether domain-specific SR in early childhood mediates the association between household income in infancy and overweight and obesity in later childhood. By distinguishing between cool and food-related hot SR within a causal mediation framework, this study aims to clarify whether early regulatory processes represent a significant pathway linking socioeconomic disadvantage to pediatric obesity.

Methods

Exposure

Household income during early childhood was operationalized using the income-to-needs ratio (ITNR) assessed between birth and 24 months. For each participant, available ITNR measures during this period were averaged to capture overall economic conditions in the first two years of life. Participants were included if at least one ITNR measure was available during the 0–24-month window.

Children were categorized as living in low-income households if their mean ITNR was <1.85, a commonly used threshold approximating eligibility for federal assistance programs, and known as the poverty-income ratio, and as higher-income if their mean ITNR was ≥1.85.[23] Although ITNR was available as a continuous measure, it was dichotomized to enhance interpretability and align with policy-relevant definitions of low-income status.

Mediators

Cool self-regulation

Cool SR was assessed at 54 months of age using the Continuous Performance Task (CPT). The CPT measures sustained attention and impulsivity in emotionally neutral contexts.[24] The two CPT scores measuring impulsivity and inattention were combined and reverse-coded so that higher values indicate better cool SR (possible range = 0 to 195). Participants' scores exhibited a non-normal distribution; therefore, a rank-based inverse normal transformation (RINT) was applied to reduce skewness and the influence of extreme values. This nonparametric transformation converts ranked values to corresponding quantiles of the standard normal distribution, yielding an approximately normally distributed variable without imposing parametric assumptions on the original scale. The transformed cool self-regulation score was modeled as a continuous variable.

Food-related hot self-regulation

Food-related hot SR was assessed at 54 months of age using a delay of gratification task. Children chose a preferred food from three options and were shown a large and a small quantity of the food. They were given a bell and told they could receive the larger quantity if they waited until the researcher returned. Children were grouped as "passed" or "failed" based on whether they waited the full 7 minutes. The distribution exhibited substantial ceiling effects, with approximately half of the children waiting the full 7 minutes (420 seconds). Given this clustering at the upper bound, modeling the measure as continuous or ordinal would impose strong distributional assumptions. Therefore, the variable was operationalized as a binary indicator reflecting whether the child successfully waited the full duration (pass) or terminated the task

before completion (fail). This approach aligns with the conceptualization of delay of gratification as the ability to sustain waiting under temptation and provides an interpretable mediator.[25,26]

Outcome

The primary outcome was overweight/obesity status at age 9 years. This time point was selected to provide adequate temporal separation from SR measured at 54 months, allowing for the potential emergence of behavioral pathways linking early regulatory capacity to weight status. Age 9 also represents a preadolescent developmental period during which weight patterns may be less influenced by the broader physiological and behavioral changes that emerge during adolescence. At age 9, height and weight were collected during a laboratory visit. Height was recorded in inches to the nearest one-eighth inch, and weight in pounds and ounces. We classified children with BMI z-scores greater than 1.036 and less than 1.645 (>85th to <95th percentile, age and sex-adjusted) as having overweight, and children with BMI z-scores at or greater than 1.645 (≥95th percentile, age and sex-adjusted) as having obesity, based on the CDC 2000 BMI-for-age growth chart definitions.[27] Weight status at age 9 was treated as a binary variable: normal weight versus having overweight or obesity.

Covariates

Covariates were selected a priori based on prior literature and conceptual considerations to address potential confounding of the exposure–mediator and mediator–outcome relationships. These included child sex at birth, race/ethnicity, maternal education, age, and marital status at birth. Child age at assessment was included for both the 54-month SR measures and the age 9 BMI outcome to account for minor variation in measurement timing. The number of ITNR assessments between 0 and 24 months was additionally included to account for variability in

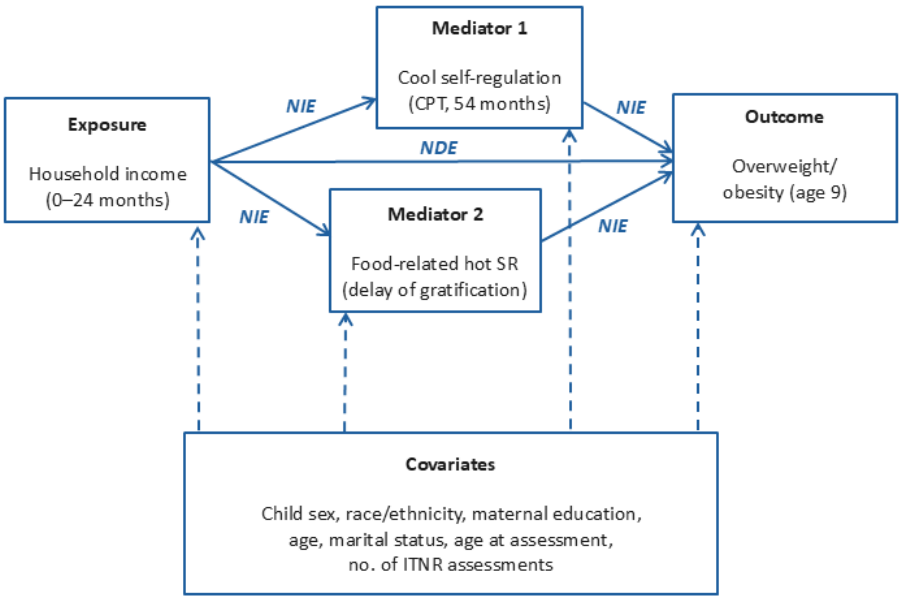
exposure measurement precision. All covariates were measured prior to the mediator or outcome as appropriate to preserve temporal ordering.

Statistical Analysis

The analytical sample included 782 children with non-missing data on exposure, mediators, and outcome. Missingness in covariates was minimal (one observation); therefore, analyses were conducted using complete-case data.

Causal mediation analysis was conducted within the counterfactual framework to decompose the total effect of early childhood household income on overweight/obesity at age 9 into natural direct and natural indirect effects.[28] The natural indirect effect represents the portion of the association operating through the mediator (i.e., the two SR domains), whereas the natural direct effect represents the effect not operating through the mediator (Figure 3). Separate models were estimated for each SR domain (cool SR and food-related hot SR) using PROC CAUSALMED in SAS to quantify domain-specific mediation effects. To assess the presence of exposure-mediator interaction, the outcome model in the PROC CAUSALMED analysis for each mediator was specified to allow for exposure-mediator interaction.

Figure 3 Proposed mediation pathways linking early childhood household income to overweight/obesity through cool and food-related hot self-regulation



NIE = natural indirect effect; NDE = natural direct effect. Solid arrows represent hypothesized indirect pathways through each self-regulation domain. The dashed arrow represents the natural direct effect. Covariate arrows are dashed.

In each model, the alternate SR domain was included as a covariate in both the mediator and outcome models to account for potential overlap between regulatory processes. This approach allows estimation of domain-specific natural indirect effects while partially adjusting for shared variance between SR domains. Accordingly, the estimated indirect effects should be interpreted as effects operating through each domain conditional on the other, rather than as the combined mediation effect of SR overall.

Associations between household income and weight status, household income and SR, and SR and weight status were estimated using multivariable regression models adjusted for the same set of covariates. Logistic regression was used for binary outcomes to estimate odds ratios, and linear regression was used for continuous SR measures.

Sensitivity Analyses

Several sensitivity analyses were conducted to evaluate the robustness of findings. First, weight status at age 15 years was examined as an alternative outcome to assess whether associations differed at a later developmental stage. Second, household income was alternatively modeled as a log-transformed continuous ITNR and using an alternate low-income threshold (<2.0) to assess sensitivity to exposure specification. Finally, birthweight was added to models to evaluate potential confounding by perinatal factors.

Results

The analytic sample included 782 children, of whom 186 (23.8%) were from households with an income-to-need ratio <1.85. Table 12 provides baseline characteristics of the analytic sample. Compared with children from higher-income households, children from lower-income households were more likely to be non-Hispanic Black or Hispanic. Mothers of children in lower-income households were younger, less likely to have a college degree, and less likely to be living with a spouse or partner. Children from lower-income households also had lower mean cool SR scores and were less likely to pass the food-related SR task. The prevalence of overweight or obesity at age 9 was higher among children from lower-income households compared with those from higher-income households.

Table 12 Baseline Characteristics Overall and by Household Income from 0–24 Months of Age

Characteristic	No. (%)	ITNR <1.85 (n [%])	ITNR ≥1.85 (n [%])
Overall (n = 782)		186	594
Child's Race/Ethnicity			
Non-Hispanic White	625 (79.9)	110 (58.5)	515 (86.7)
Black or Afro-American	89 (11.4)	56 (29.8)	33 (5.6)
Hispanic	38 (4.9)	15 (8.0)	23 (8.9)
Other	30 (3.8)	7 (3.7)	23 (3.9)
Child's Sex			
Male	363 (46.4)	97 (51.6)	266 (44.8)
Female	419 (53.6)	91 (48.4)	328 (55.2)
Child's birthweight (g), mean (SD)	3500.6 ± 525.0	3478.9 ± 555.1	3509.7 ± 515.3
Maternal Age at Childbirth (year), mean (SD)	28.9 ± 5.5	25.0 ± 5.6	30.1 ± 4.8
Maternal Marital Status			
Living with spouse or partner	700 (89.5)	124 (66.0)	576 (97.0)
Others	81 (10.5)	63 (34.0)	18 (3.0)
Missing	1 (0.0)	1 (0.0)	0 (0.0)
Maternal Education Level			
No college degree	472 (60.4)	173 (92.0)	299 (50.3)
College degree or higher	310 (39.6)	15 (8.0)	295 (49.7)
Cool SR, mean (SD)	114.7 ± 22.3	107.2 ± 26.3	117.1 ± 20.3
Food-Related Hot SR			
Pass	420 (53.7)	67 (35.6)	353 (59.4)
Fail	362 (46.3)	121 (64.4)	241 (40.6)
Age 9 Overweight or Obesity			
Yes	242 (31.0)	78 (41.5)	164 (27.6)
No	540 (69.0)	110 (58.5)	430 (72.4)

ITNR = income-to-needs ratio.

Association between household income and age 9 weight status

In the full sample, children from low-income households had 86% higher odds of overweight/obesity at age 9 compared with those from higher-income households (OR = 1.52, 95% CI: 1.02–2.33). In sex-stratified analyses, the association was statistically significant among girls (OR = 1.94, 95% CI: 1.06–3.55) but not among boys (OR = 1.29, 95% CI: 0.72–2.32) (Table 13).

Table 13 Association of Early Childhood Low Household Income and Age 9 Weight Status

Sample	n	Crude OR (95% CI)	Adjusted OR (95% CI)
Full (n=782)			
ITNR ≥1.85 (ref)	540	Ref	Ref
ITNR <1.85	242	1.86 (1.32–2.61)	1.54 (1.02–2.33)
Boys (n=363)			
ITNR ≥1.85 (ref)	238	Ref	Ref
ITNR <1.85	125	1.59 (0.98–2.56)	1.29 (0.72–2.32)
Girls (n=419)			
ITNR ≥1.85 (ref)	302	Ref	Ref
ITNR <1.85	117	2.13 (1.30–3.46)	1.94 (1.06–3.55)

[a]Low household income defined by ITNR below 1.85. [b]OWO defined by BMI z scores >1.036 to <1.645 (overweight) and ≥1.645 (obesity). [c]Adjusted for child sex, race/ethnicity, age at BMI measurement, maternal education, maternal age and marital status at childbirth, and number of ITNR assessments.

Associations between household income and SR domains

Table 14 shows that early childhood low household income was associated with poorer SR across both domains. Children from lower-income households had lower cool SR scores in the full sample (β = 0.26, 95% CI: 0.10, 0.42), with similar directions of association observed among both boys and girls.

Similarly, low household income was associated with lower odds of passing the food-related SR task (OR = 0.57, 95% CI: 0.39, 0.83). While the direction of association was consistent across sex, the association was statistically significant among girls but not among boys.

Table 14 Association of early childhood household income[a] and early childhood self-regulation domains

Cool Self-regulation

Sample	n	Model 1[b] β (95% CI)[c]
Full sample	782	0.26 (0.10-0.42)
Boys	363	0.29 (0.06-0.52)
Girls	419	0.23 (0.00-0.47)

Food Hot Self-Regulation

Sample	n	Model 1[b] OR (95% CI)[d]
Full sample	782	0.57 (0.39-0.83)
Boys	363	0.72 (0.43-1.22)
Girls	419	0.44 (0.25-0.76)

[a]Low household income defined by income-to-need ratio below 1.85.

[b]Model adjusted for child sex at birth (male or female), child race/ethnicity (Non-Hispanic White, Black or Afro-American, Hispanic, or American Indian, Eskimo, Aleutian, Asian, Pacific Islander, and others), child's exact age at SR tasks measurement day, maternal education (no college degree, or college degree or above), mother's age at childbirth, and mother's marital status at childbirth (living with spouse/partner or not living with spouse/partner), and number of available household income measures.

[c]β coefficients represent adjusted mean differences in cool self-regulation for children with income-to-need ratio <1.85 compared with ≥1.85 (reference: ≥1.85).

[d]Odds ratios represent the odds of passing the food-related delay of gratification task among children with income-to-need ratio <1.85 compared with ≥1.85 (reference: ≥1.85).

Associations between SR domains and age 9 weight status

Table 15 shows the associations between early childhood SR domains and overweight/obesity at age 9. Neither cool self-regulation nor food-related hot self-regulation was significantly associated with weight status in the full sample. Similarly, no statistically significant associations were observed in sex-stratified analyses.

Table 15 Association of early childhood self-regulation domains and age 9 weight status[a]

Self-Regulation Domain	Full sample (n=782) OR (95% CI)	Boys (n=363) OR (95% CI)	Girls (n=419) OR (95% CI)
Cool Self-regulation[b] [d]	0.86 (0.73-1.02)	0.92 (0.73-1.61)	0.82 (0.65-1.04)
Food Hot Self-Regulation[c] [d]	1.22 (0.88-1.67)	1.66 (1.05-2.61)	0.87 (0.55-1.39)

[a]Overweight or obesity defined by BMI z scores > 1.036 and < 1.645 (>85th to < 95th percentile, age and sex-adjusted) as having overweight, and children with BMI z scores ≥ 1.645 (≥ 95th percentile, age and sex-adjusted) as having obesity.

[b]Odds ratios represent the odds of overweight/obesity associated with a one SD-unit increase in Cool SR scores.

[c]Odds ratios compare children who passed versus failed the food-related delay of gratification task (reference: failed).

[d]Model adjusted for child sex at birth (male or female), child race/ethnicity (Non-Hispanic White, Black or Afro-American, Hispanic, or American Indian, Eskimo, Aleutian, Asian, Pacific Islander, and others), child's exact age at BMI measurement day, maternal education (no college degree, or college degree or above), mother's age at childbirth, and mother's marital status at childbirth (living with spouse/partner or not living with spouse/partner).

Mediation role of domains of self-regulation

Table 16 shows that early childhood low household income was associated with a higher risk of overweight/obesity at age 9, with statistically significant total and natural direct effects. In contrast, there was no evidence that this association was mediated through either domain of SR, as the natural indirect effects for both cool SR and food-related hot SR were not statistically significant. These findings suggest that the relationship between early-life income and later weight status operates primarily through pathways other than these measured domains of SR. The exposure-mediator interaction was non-significant in both models of SR, and the controlled

direct effects and natural direct effects were equivalent, indicating no meaningful interaction. Results are therefore interpreted without a four-way decomposition.

Table 16 Causal Mediation Analysis of Early Childhood Household Income and Age 9 Overweight/Obesity Through Self-Regulation Domains

	Full sample (n=782)	**Boys (n=363)**	**Girls (n=419)**
Model 1: Mediated by Cool SR	Estimate (95% CI)	Estimate (95% CI)	Estimate (95% CI)
TE	1.53 (1.08–2.62)	1.30 (0.82–3.17)	1.97 (1.21–5.18)
NDE	1.52 (1.07–2.60)	1.31 (0.82–3.16)	1.97 (1.22–5.09)
NIE	1.01 (0.99–1.03)	0.99 (0.96–1.03)	1.01 (0.95–1.07)
Proportion mediated (%)	1.28 (−2.83–5.40)	−1.88 (−14.66–10.91)	1.02 (−5.01–7.06)
Model 2: Mediated by Food-Related Hot SR			
TE	1.52 (1.08–2.61)	1.29 (0.82–3.17)	1.88 (1.17–4.88)
NDE	1.52 (1.08–2.60)	1.31 (0.82–3.16)	1.97 (1.22–5.09)
NIE	1.00 (0.99–1.02)	0.99 (0.93–1.06)	0.96 (0.89–1.04)
Proportion mediated (%)	0.48 (−2.77–3.73)	−2.84 (−27.39–21.71)	−4.71 (−15.86–6.44)

TE = total effect; NDE = natural direct effect; NIE = natural indirect effect. Estimates are odds ratios comparing ITNR <1.85 vs. ≥1.85. [a]Same covariate adjustment as Table 3.2; alternate SR domain included as covariate in each model.

Sensitivity analyses

In sensitivity analyses using overweight/obesity at age 15 as the outcome (Table 17), early childhood low household income remained associated with higher risk of overweight/obesity, with statistically significant total effects and natural direct effects observed in the full sample and among boys, and for the natural direct effect among girls. In contrast, there was no evidence of mediation through either domain of SR, as all natural indirect effects remained non-significant. Overall, these findings were consistent with the primary analyses at age 9. Findings were also robust to alternative definitions of low household income; results were unchanged when using an income-to-need ratio cutoff of <2.0 instead of <1.85.

Table 17 Causal mediation analysis of early childhood household income[a] and age 15 overweight/obesity[b] through self-regulation domains, overall and by sex

	Full sample (n=782)	Boys (n=363)	Girls (n=419)
	Estimate (95% CI)	Estimate (95% CI)	Estimate (95% CI)
Model 1: Mediated by Cool Self-Regulation[c]			
TE	1.53 (1.04–2.92)	1.41 (0.84–4.49)	1.62 (1.02–5.57)
NDE	1.52 (1.03–2.89)	1.41 (0.84–4.47)	1.57 (0.92–5.34)
NIE	1.01 (0.98–1.04)	1.00 (0.97–1.04)	1.03 (0.96–1.12)
Proportion mediated (95% CI)	1.74 (-4.93–8.41)	0.83 (-7.04–8.70)	5.36 (-10.45–21.17)
Model 2: Mediated by Food Hot Self-Regulation[c]			
TE	1.51 (1.03–2.89)	1.33 (0.78–4.35)	1.55 (0.91–5.36)
NDE	1.52 (1.03–2.89)	1.41 (0.84–4.47)	1.57 (0.92–5.34)
NIE	1.00 (0.96–1.03)	0.94 (0.83–1.09)	0.99 (0.93–1.05)
Proportion mediated (95% CI)	-0.95 (-7.72–5.82)	-18.12 (-87.46–51.21)	-2.08 (-13.56–9.41)

[a]Low household income defined by income-to-need ratio below 1.85.

[b]Overweight or obesity defined by BMI z scores > 1.036 and < 1.645 (>85th to < 95th percentile, age and sex-adjusted) as having overweight, and children with BMI z scores ≥ 1.645 (≥ 95th percentile, age and sex-adjusted) as having obesity.

[c]Model adjusted for child sex at birth (male or female), child race/ethnicity (Non-Hispanic White, Black or Afro-American, Hispanic, or American Indian, Eskimo, Aleutian, Asian, Pacific Islander, and others), child's exact age at BMI measurement day, maternal education (no college degree, or college degree or above), mother's age at childbirth, and mother's marital status at childbirth (living with spouse/partner or not living with spouse/partner), and number of available household income measures.

Discussion

In this longitudinal cohort, low household income in early childhood was associated with a higher risk of overweight/obesity in later childhood. However, this association was not mediated by early childhood SR, whether assessed as cool executive function or food-related delay of gratification. Although children from lower-income households demonstrated poorer SR across both domains, these measures were not associated with subsequent weight status. Together, these findings suggest that pathways linking early-life socioeconomic disadvantage to later obesity risk may operate independently of these aspects of SR.

These results are broadly consistent with the existing literature. Prior work has documented associations between socioeconomic disadvantage and lower SR, as well as modest and heterogeneous associations between SR and pediatric weight outcomes.[29] However, relatively few studies have formally tested mediation. In this context, the present study adds longitudinal evidence suggesting that early SR, as measured here, may not represent a key mechanism linking socioeconomic conditions to later obesity risk.

The absence of a mediating role for SR in this study likely reflects both the lack of association between early SR and later weight outcomes and the broader context in which these processes operate. Although children from lower-income households exhibited poorer SR, these differences did not translate into differences in weight status. This pattern aligns with emerging evidence suggesting that associations between early SR and later outcomes may attenuate substantially after accounting for family background characteristics.[30–32] It is also possible that SR capacities assessed in early childhood do not capture the forms of regulation most relevant to weight-related behaviors later in development, as these capacities continue to evolve across childhood.[33,34] For example, regulation in adolescence may involve navigating more autonomous, socially embedded, and emotionally complex decision-making that differs substantially from the structured settings used to assess SR in early childhood. More broadly, the influence of SR on behaviors such as diet may be diluted over time, as body weight reflects the cumulative result of multiple interacting biological, behavioral, and environmental processes.[35] In environments characterized by constrained access to healthy foods, limited opportunities for physical activity, or higher exposure to stress, individual-level regulatory capacity may be insufficient to offset structural disadvantages.[36] These considerations may help explain why

differences in early SR did not translate into differences in weight outcomes and why mediation through SR was not observed.

The persistent association between early childhood low household income and later overweight/obesity, independent of SR, points to the importance of alternative mechanisms. Material and environmental factors linked to socioeconomic disadvantage – including food insecurity, diet quality, neighborhood environments, and access to health-promoting resources – may play a more direct role in shaping weight trajectories. [7,8] Chronic stress and related physiological pathways may also contribute, influencing metabolic processes and health behaviors over time.[37] These pathways are likely to operate cumulatively across childhood, reinforcing disparities in obesity risk.

Strengths and limitations

This study has several strengths. The use of a well-characterized longitudinal cohort allowed for prospective assessment of early-life socioeconomic conditions, SR, and later weight outcomes. The availability of domain-specific measures of SR enabled a more nuanced examination of potential mechanisms. Additionally, the application of causal mediation analysis allowed for formal evaluation of indirect effects, advancing understanding beyond composite-based approaches.

Several limitations should be considered. SR was assessed at a single time point in early childhood, which may not capture changes in regulatory capacity over development. The measures used may not fully reflect all relevant domains, particularly those more proximal to eating behaviors. Residual confounding by unmeasured factors, such as parental health behaviors or genetic influences, cannot be ruled out. Finally, the cohort was predominantly non-Hispanic White, which may limit generalizability to more diverse populations.

Conclusion

In conclusion, early childhood low household income was associated with increased risk of overweight/obesity, but this relationship was not mediated by early SR. These findings suggest that interventions targeting SR alone may be insufficient to reduce socioeconomic disparities in childhood obesity. Future research should more directly examine structural and environmental mechanisms, such as food environments, chronic stress, and access to health-promoting resources, which may play a more significant role in linking early-life socioeconomic disadvantage to later obesity risk.

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Author Contributions:

J.W.Y.K. conceived the study, developed the analytic strategy, conducted the statistical analyses, and drafted the manuscript. L.J., D.D.P., and A.O.O. contributed to study design and conceptual development, provided guidance on analytic approach and interpretation of findings, and critically revised the manuscript for important intellectual content. All authors contributed to the interpretation of results, reviewed the manuscript, and approved the final version.

Competing Interests:

The authors declare no competing financial or non-financial interests.

Data Availability Statement:

The data that support the findings of this study are available from the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD). These data are publicly available to qualified researchers upon request through the NICHD Data and Specimen Hub (DASH) repository (https://dash.nichd.nih.gov/), subject to relevant data use agreements and ethical approvals. The analytic code used in this study is available from the corresponding author upon reasonable request.

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SUMMARY AND CONCLUSIONS

This dissertation examined early-life socioeconomic conditions, self-regulatory capacities, and their relationships to weight trajectories across childhood and adolescence using data from the NICHD SECCYD. Three interrelated studies were conducted, each addressing a distinct question while contributing to a cohesive understanding of the developmental pathways linking socioeconomic disadvantage to pediatric obesity.

Chapter 1 found no association between early childhood household income and adiposity rebound timing, even after employing propensity score matching to address confounding by a broad set of parenting-related and sociodemographic characteristics. This null finding is informative: it suggests that resource-based socioeconomic disadvantage, per se, may not independently shape the AR growth dynamic, and points toward the potential primacy of proximal behavioral and caregiving influences on early weight trajectories. The quasi-experimental design employed here represents a methodological advance over prior regression-based studies in this area.

Chapter 2 found that early childhood SR domains were differentially associated with adolescent OWO at age 15. Cool SR and food-related hot SR were each independently protective, while nonfood hot SR showed no significant association. The concentration of the food hot SR association among boys, but not girls, adds a sex-specific dimension to the SR–obesity relationship that warrants further investigation. These findings challenge the prevailing treatment of SR as a unitary construct in pediatric obesity research and intervention, suggesting that domain specificity matters.

Chapter 3, building on the preceding two aims, formally tested whether domain-specific SR mediates the income–OWO association using causal mediation analysis. Despite significant

associations between low income and both SR domains, and between low income and OWO, neither SR domain mediated the income–weight status relationship. These null mediation findings do not diminish the relevance of SR to obesity risk demonstrated in Chapter 2; rather, they suggest that the pathways through which SR influences obesity risk are largely independent of the socioeconomic pathways driving income-related disparities.

Taken together, these three studies suggest several important conclusions. First, household income operates on childhood obesity risk primarily through mechanisms other than AR timing or early self-regulatory capacity. Material and environmental factors — including food insecurity, diet quality, neighborhood conditions, and chronic physiological stress — likely represent more direct pathways. Second, domain-specific self-regulation in early childhood does predict later obesity risk, but the relevant mechanisms are largely independent of income-mediated pathways. Third, interventions designed solely to improve early SR may be insufficient to address socioeconomic disparities in childhood obesity, though they may still reduce obesity risk more broadly.

Future research should examine structural and environmental mechanisms more directly, including food environments, built environments, and chronic stress exposures, as potential mediators of the income–obesity association. Longitudinal studies with repeated SR assessments across development would help clarify whether later regulatory capacities — more proximal to adolescent eating behavior — serve as stronger mediators. Finally, intervention studies that distinguish SR domains and attend to sex differences may yield more targeted and effective strategies for pediatric obesity prevention.