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Body Mass Indices of Girls with and without ADHD: Developmental Trajectories from Childhood to Adulthood

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Abstract

Objective: We examined the predictive relation between childhood-diagnosed ADHD and trajectories of body mass index (BMI) from childhood to adulthood in an all-female sample, accounting for socioeconomic status (SES), childhood comorbidities (e.g., depression/anxiety), and stimulant usage. Childhood executive functioning (i.e., planning, sustained attention, and response inhibition) was also evaluated as a possible predictor of BMI trajectories.

Method: We utilized longitudinal data from a full sample of 140 girls diagnosed with ADHD in childhood and 88 comparison girls matched on age and ethnicity. Girls were 6–12 years old at the first assessment and followed prospectively for 16 years. Data were collected on their BMI and stimulant medication usage across four evaluation waves. Using latent growth curve modeling, we evaluated the BMI trajectories of girls with ADHD and the comparison sample from childhood to adulthood.

Results: Although there was no significant difference in initial childhood BMI, girls with ADHD increased in BMI at a significantly faster rate than comparison girls across development, even when adjusting for covariates. Significant differences in BMI first emerged in adolescence; by adulthood, 40.2% of the ADHD sample met criteria for obesity versus 15.4% of the comparison sample. When covarying ADHD diagnosis, executive functioning measures were not significantly predictive of BMI increase. Adjusting for stimulant medication usage within the ADHD sample did not alter core findings.

Conclusions: We discuss health-related implications for girls with ADHD, potential underlying mechanisms, and how our findings may inform both ADHD and obesity interventions.

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Keywords

ADHD; BMI; Overweight/Obesity; Executive Functioning; Latent Growth Curve Modeling

Attention-deficit/hyperactivity disorder (ADHD) is a prevalent neurodevelopmental condition characterized by developmentally extreme and impairing levels of inattention and/or hyperactivity/impulsivity. Typically diagnosed in childhood, ADHD is associated with difficulties in numerous domains across the lifespan, including occupational, academic, and social functioning (Hechtman et al., 2016; Hinshaw et al., 2012). Over the past two decades, increasing attention has been paid to the long-term health outcomes of individuals with ADHD, with specific focus on high body mass index (BMI) and obesity. Multiple studies have found higher BMI and obesity rates among individuals with ADHD compared to the general population (Altfas, 2002; Cortese et al., 2013; Racicka et al., 2018), highlighting the need for further research on psychological factors that may contribute to this disparity.

Researchers have proposed a variety of mechanisms that may underlie the association between ADHD and BMI, including disordered eating, physical inactivity, and sleep disruption (Cortese, 2019; Vogel et al., 2015) as well as shared genetic liability (Do et al., 2019; Martins-Silva et al., 2019). Another promising factor is executive functioning (EF)—a transdiagnostic, multidimensional construct composed of several cognitive and regulatory abilities, including working memory, planning/organization, response inhibition, and attention (Gioia et al., 2000). Deficits in EF are common among youth with externalizing disorders such as ADHD and often persist into adolescence and adulthood, even when clinical symptoms have remitted (Gordon & Hinshaw, 2019). Emerging evidence suggests that deficits in various components of EF may also be significantly related to weight gain in both clinical and non-clinical populations (Gunstad et al., 2007; Yang et al., 2018). For example, poor response inhibition in childhood may lead to erratic eating behaviors and the impulsive consumption of easily accessible “junk foods” that are high in caloric content, low in nutrients, and strongly associated with weight gain (Nigg et al., 2016). Issues with sustained attention could contribute to higher rates of “mindless” eating via inattention to internal hunger and satiety cues (Davis et al., 2006), and disorganization may hinder the ability to plan healthy meals (Cortese & Vincenzi, 2011). Atypical reward processing has also been implicated as a factor related to obesity, particularly when cognitive control is insufficient to override high reward value related to eating (Rolls, 2011). In sum, existing research suggests a possible link between high BMI and several EF deficits that are prevalent among individuals with ADHD.

Another key question surrounding the potential ADHD-BMI association regards directionality. As noted above, multiple studies suggest higher-than-expected BMI among individuals with ADHD, with many researchers interpreting these findings as evidence that ADHD-associated deficits contribute causally to weight gain over time (Altfas, 2002; Cortese et al., 2013; Khalife et al., 2014). Yet other research supports the reverse temporal association, whereby elevated BMI increases risk of developing ADHD symptoms. Via genome-wide association studies, Martins-Silva et al. (2019) found a positive influence of

higher BMI on ADHD, but evidence for the genetic liability to ADHD as an influencer of BMI was less consistent. Also, greater BMI decline in childhood (ages 3–5 years) predicted a greater corresponding increase in EF (Blair et al., 2019). Importantly, this BMI-EF association was not present at baseline but emerged over the 2-year study period, underscoring the importance of examining developmental *trajectories* of BMI growth (Blair et al., 2019; see also Do et al., 2019). An informative review of salient issues is found in Cortese (2019) – in sum, more longitudinal research is needed on both possible causal directions.

Further adding to the complexity of the ADHD-BMI question is stimulant medication treatment for ADHD. Although use varies substantially by demographics, around two-thirds of U.S. youth with an ADHD diagnosis are prescribed stimulant medications (Visser et al., 2014). Stimulants are an evidence-based intervention for ADHD, with appetite reduction as a core side effect (Gillberg et al., 1997). Still, it remains unclear how the link between ADHD and BMI may be influenced by medication. Biederman et al. (2003) found that medicated girls with ADHD weighed significantly *more* than their unmedicated counterparts, whereas Waring and Lapane (2008) found the opposite pattern (Castaneda et al., 2016 found no significant association). The developmental timing of stimulant usage may partially account for these mixed findings. For example, Schwartz et al. (2014) found a “rebound” effect related to stimulant use among children with ADHD: Younger age of first stimulant use predicted slower BMI growth in childhood but more rapid BMI growth in adolescence, pointing to the need for research spanning from childhood into adulthood.

Finally, increasing evidence suggests that the strength of the ADHD-BMI link may vary significantly by gender. Several studies have found that ADHD relates to higher BMI only among females (Castaneda et al., 2016; Do et al., 2019; van Egmond-Fröhlich et al., 2012). By contrast, Racicka et al. (2018) found a higher prevalence of overweight/obesity only among boys with ADHD (yet their sample was 85% male and potentially underpowered to detect significant effects among females). Indeed, the traditional exclusion of girls and women from ADHD research has limited relevant data.

In sum, accumulating research suggests a potential association between ADHD and BMI, but important questions remain concerning the developmental timing of BMI increase in those diagnosed with ADHD in childhood, factors underlying this association, and the potential effects of stimulant treatment. These gaps highlight the need for additional *longitudinal* research on the topic, particularly among females. We investigate whether childhood diagnosis of ADHD predicts differences in the rate of BMI increase across development in girls, and if so, whether three EF deficits common in children with ADHD (i.e., poor planning, response inhibition, sustained attention) contribute to BMI growth. In line with the Research Domain Criteria (RDoC), which encourage emphasis on transdiagnostic processes, we focus on these cognitive mechanisms because of their potential relevance for weight gain across different disorders. Given research linking socioeconomic status (SES) to obesity rates (McLaren, 2007), we account for differences in SES, as well as comorbid internalizing disorders and stimulant usage. To our knowledge, the present research is the first to use structural equation modeling (SEM) to examine longitudinal trajectories of BMI in relation to childhood ADHD.

We hypothesized the following:

1. Girls diagnosed with ADHD in childhood would increase in BMI through adolescence and young adulthood at a significantly faster rate than girls without childhood ADHD, when accounting for SES, comorbid depression/anxiety, and stimulant usage.
2. Childhood deficits in EF—specifically poor planning, response inhibition, and sustained attention—would predict increased BMI growth across development, above and beyond the effect of childhood ADHD diagnostic status.

Method

Participants and Procedure

We utilized data collected from a prospective sample of girls diagnosed with ADHD in childhood (140 girls), along with a matched comparison sample without ADHD (88 girls), who participated in the Berkeley Girls with ADHD Longitudinal Study (BGALS). The racial/ethnic makeup of the sample was 53% White, 27% African American, 11% Latina, and 9% Asian American, with a wide range of socioeconomic backgrounds. ADHD and comparison samples were group-matched on age and ethnicity. Of the 140 girls with ADHD, 34% presented as predominantly inattentive and 66% combined (i.e., both hyperactive/impulsive and inattentive).

As described by Hinshaw (2002), girls were recruited from multiple sites including medical, educational, and mental health settings. Exclusionary criteria included IQ less than 70, overt neurological damage, psychosis, pervasive developmental disorder, or any medical conditions that prevented participation in the summer camp. Common psychiatric comorbidities (e.g., oppositional defiant disorder [ODD], conduct disorder [CD], learning disorders, etc.) were allowed to promote generalizability of the ADHD sample. Comparison girls with internalizing disorders and/or ODD were included to avoid creating a supernormal comparison sample.

Participants were enrolled in the study during childhood (between ages 6–12) and were then followed longitudinally approximately every five years across three additional evaluation waves. The age ranges at each wave were as follows—Wave 1: 6–12 yrs., Wave 2: 11–18 yrs., Wave 3: 17–25 yrs., and Wave 4: 22–29 yrs. Across the 16-year longitudinal study, there was excellent retention (92–95% at each wave). As described by Owens and Hinshaw (2016), the retained sample at Wave 4 was higher in SES than those who dropped out, but they showed no significant differences in race/ethnicity or age.

To assess changes in BMI across development, we excluded participants with fewer than two BMI observations from analyses (n = 9, 4% of sample). These nine participants did not significantly differ from remaining participants in income, maternal education, ethnicity, childhood ADHD diagnosis, or childhood depression and/or anxiety diagnoses. The final sample included 219 participants: 133 with ADHD in childhood and 86 comparison girls.

Measures

Body mass index.—BMI for each participant was calculated by dividing weight in kilograms by height in meters squared at each assessment period. At Waves 1 and 2, parents reported the most recent height and weight measurements for their daughters; at Waves 3 and 4, these values were measured objectively during clinic assessment whenever possible, supplemented by self-report as needed (e.g., participants unable to come to the clinic in-person for assessment). We ultimately utilized self-reported weight and/or height for 15 participants at Wave 3 and 63 at Wave 4. Enhanced reliance on self-report at Wave 4 was probably related to geographic mobility linked to this developmental stage (i.e., ages 22–29), which made in-person clinic visits more difficult.

Studies comparing parent/self-reported BMI calculations to those objectively measured suggest average reporting biases on the order of +/− 2 kg/m^2, depending on source (i.e., parent vs self-report) and child age (Weden et al., 2013, Kovalchik, 2009). Self-reporting vs. objectively measured participants did not significantly differ in childhood ADHD status, suggesting that reporting biases were evenly distributed between ADHD and comparison girls.

Childhood ADHD diagnosis.—Initial screenings included parent- and teacher-report on the Swanson, Nolan, and Pelham Rating Scale (4th ed.; SNAP– IV; Swanson, 1992). Eligibility for the ADHD group was then determined by meeting full diagnostic criteria based on parental report on the Diagnostic Interview Schedule for Children (4^{th} ed.; DISC-IV; Shaffer et al., 2000).

Planning.—In the copy condition of the Rey-Osterrieth Complex Figure Test (ROCF), children were asked to reproduce a complex figure using pencil and paper, while the original stimulus image remained in front of them. We calculated ROCF error proportion scores (i.e., the total number of reproduction errors divided by the total number of moves) for each participant as an assessment of planning skills (Sami et al., 2004), whereby higher scores indicate greater impairment (M = 0.30, SD = 0.19, Range: 0.02–0.88). Scorers of the error proportion procedure yielded intraclass correlations ranging from 0.91 to 0.94, and this scoring procedure showed a large effect size when differentiating girls with vs. without ADHD (d = 0.90). Additional evidence for the validity of this scoring procedure has been revealed in multiple investigations from our laboratory (e.g., Hinshaw et al., 2002; Miller et al., 2012).

Sustained Attention.—This variable was measured via the Conners' Continuous Performance Test (CPT; see Conners, 1995), a computerized task in which children hit a button when they see any letter except for 'X', and refrain from hitting the button when they see 'X'. Over 14 minutes, six trial blocks are shown including interstimulus intervals set at one, two, or four seconds within each block. Sustained attention was assessed via percentage of omission errors – the number of non-responses to target stimuli (i.e., letters other than 'X') divided by the total number of target stimuli presented (M = 8.50, SD = 12.32, Range: 0.01–86.70).

Response Inhibition.—This variable was also measured via the Conners' CPT described above (Conners, 1995), using the percentage of commission errors – the number of responses to nontargets (i.e., the letter "X") out of the total number of nontargets presented (M = 54.14, SD = 22.28, Range: 0.31–97.22). Past studies using this dataset indicate that girls with ADHD demonstrate significantly greater CPT omission and commission error percentages than comparisons on this task (e.g., Hinshaw et al., 2002).

Childhood comorbidities.—We determined diagnoses of depression and/or anxiety at Wave 1 (n = 38, 17.4% of sample) based on whether participants met full diagnostic criteria for either disorder, using parent-report from the DISC-IV.

Socioeconomic status (SES).—This score comprised the standardized average of maternal education (M = 4.79, SD = 0.95) and family annual income (M = 6.43, SD = 2.57). Highest level of maternal education was rated on scale from 1 (less than 8th grade) to 6 (advanced or professional degree), and income on a scale from 1 (< $10,000) to 9 ($75,000+), as in previous studies (see Hinshaw et al., 2012; Owens & Hinshaw, 2016).

Stimulant usage.—Stimulant medication usage was based on parent-report for Waves 1–2 and self-report for Waves 3–4 (supplemented with parent-report for a minority of participants). Parallel to the Multimodal Treatment Study of Children with ADHD, medication status was defined by a 50% criterion – participants were categorized as having high/consistent usage if they used stimulant medication at least 50% of the days since the previous assessment (Swanson et al., 2007). We evaluated stimulant usage on a 0 to 2 scale, where 0 indicated no stimulant usage, 1 indicated low/inconsistent usage (<50% of the time), and 2 indicated high/consistent usage (≥50% of the time). The exception was baseline stimulant usage, a binary variable based on whether the participant was using stimulant medication as of Wave 1.

Data Analytic Plan

We utilized latent growth curve modeling (LGCM) to evaluate BMI trajectories across development. A form of structural equation modeling, LGCM enables the evaluation of developmental trajectories, characterized by initial starting point (i.e., intercept) and change over time (i.e., slope), plus other polynomial terms as appropriate (e.g., a quadratic term).

Age bins.—Following the recommendations of Bollen and Curran (2006), we modeled change in BMI by participant age rather than wave of data collection. We re-organized our four waves of data into five age bins, corresponding to different developmental periods: (1) childhood (M_{age} = 8.90 years, SD_{age} = 1.24 years), (2) early adolescence (M_{age} = 12.58 years, SD_{age} = 0.95 years), (3) adolescence (M_{age} = 16.78 years, SD_{age} = 1.28 years), (4) early adulthood (M_{age} = 21.18 years, SD_{age} = 1.46 years), and (5) adulthood (M_{age} = 26.19 years, SD_{age} = 1.44 years). We assigned participant data collected at each wave to different age bins based on the participant's age at each assessment period so that each participant had missing data for at least one age bin. We estimated all missing data using full information maximum likelihood estimation procedures (as described below), affording utilization of

data from all 219 participants in LGCMs. In all models, participant age was centered around the mean age in the first age bin (M_{age} = 8.90 years).

Analyses.—Preliminary analyses included examination of normality, outliers, and missing data. Consistent with LGCM procedures, we first examined BMI trajectory shape and growth factor variability using an unconditional model (i.e., without predictors or covariates). For Hypothesis 1, we evaluated childhood ADHD as a predictor of BMI latent variables (e.g., intercept and slope), both with and without adjusting for covariates (i.e., family SES, diagnosis of depression and/or anxiety, and stimulant usage, with the latter modeled as a time-varying covariate). We also compared BMI averages and rates of obesity (i.e. BMI ≥ 30 kg/m^2) between ADHD and comparison groups at each age bin, using independent t-tests and chi-squared tests, respectively. For Hypothesis 2, we evaluated the relations between three childhood EF capacities and BMI latent variables via separate LGCMs, examining whether relations remained significant when adjusting for childhood ADHD diagnosis. All analyses were conducted using the R lavaan package, version 0.6–5. Model fit was evaluated using the model chi-square statistic ($p \geq 0.05$), comparative fix index (CFI; values ≥ 0.95), Tucker-Lewis Index (TLI; ≥ 0.95), standardized root mean square residual (SRMR; < 0.08), and root mean square error of approximation (RMSEA; < 0.06 with a 90% confidence interval containing 0) (Hu & Bentler, 1999; Kline, 2011).

Results

Preliminary Analyses

Normality.—Because LGCM assumes multivariate normality, we first analyzed univariate distributions to verify this assumption. Only CPT commissions demonstrated substantial positive skew and kurtosis, so we square-root transformed this variable [$-1/\sqrt{(1 + x)}$], resulting in a more normal distribution (Kline, 2011).

Outliers.—We examined all continuous measures (i.e., BMI, SES, ROCF error proportion scores, CPT commission and omission error percentages) for values more than three standard deviations from the mean at each age bin. There were 14 outliers in total. One outlier was high in ROCF error proportion score, and the remaining 13 outliers were high in BMI at various time points, primarily in the last three age bins. We winsorized these values to 3.1 standard deviations above the mean. This procedure is likely to provide a conservative test of hypotheses, given that 12 out of the 13 high BMI outliers emanated from the ADHD group.

Missing data.—10% of all BMI data, 5% of all stimulant-usage data, and 0–3% (M = 1%) of data for all Wave 1 predictors/covariates were missing. Due to high retention rates, subject attrition only accounted for a small percentage of this missing data (7% of missing BMI data, 8% of missing stimulant usage data). The majority of missing data was due to incomplete data collection (e.g., unanswered questions). Overall, missing stimulant-usage data were fairly evenly distributed across assessment waves, but the majority of the missing BMI data emanated from Wave 1. Specifically, 33% of all participants were missing baseline BMI data. Beyond these traditional sources of missing data, our reorganization of

four waves of repeated measures into five age bins also caused each participant to have "artificially" missing BMI and stimulant-usage data in at least one age bin (i.e., 20% of data).

All missing data were estimated using full information maximum likelihood (FILM) procedures, which have been shown to yield unbiased and efficient parameter estimation and are often recommended for LGCM (Bollen & Curran, 2006; Enders & Bandalos, 2001). FIML estimates population parameters by maximizing the probability of obtaining the observed data. This estimation method requires that data be missing at random (i.e., the probability of missingness does not depend on the missing variable itself). To verify this assumption, we approximated differences in BMI and stimulant-usage between participants with and without these data missing at a given assessment wave, using their corresponding measures from other waves. We found no significant differences between participants with and without missing data at a given wave, suggesting that BMI and stimulant-usage data were missing at random. Additionally, "artificially" missing data due to age-bin assignment were based solely on participant age—therefore, by design, random with respect to BMI values.

Unconditional Model

To evaluate BMI trajectory shape and growth factor variability, we first conducted a model of BMI development from ages 9 to 26 without predictors. A linear LGCM was a poor fit for the data, $\chi^2(10, 219) = 48.60$, $p(\chi^2) < 0.001$, CFI = 0.927, TLI = 0.927, RMSEA = 0.133, CI_{90} = [0.097, 0.171], SRMR = 0.086. Adding a quadratic term substantially improved model fit, indicating curvature in BMI trajectories, $\chi^2(6, 219) = 13.80$, $p(\chi^2) = 0.03$, CFI = 0.985, TLI = 0.975, RMSEA = 0.077, CI_{90} = [0.021, 0.131], SRMR = 0.039. All latent variables (i.e., intercept, linear, and quadratic terms) were significant. Specifically, participant BMI in childhood yielded $M = 17.3$ kg/m^2 ($SE = 0.29$, $p < 0.001$), increasing at an average rate of 1.0 kg/m^2 per year ($SE = 0.07$, $p < 0.001$), with the rate of increase in BMI across development slowly tapering over time ($M_{quad} = -0.02$, $SE = 0.004$, $p < 0.001$).

Unlike BMI intercept ($\sigma^2 = 8.05$, $SE = 2.79$, $p < 0.01$) and linear slope ($\sigma^2 = 0.28$, $SE = 0.14$, $p < 0.05$), the quadratic term did not have a significant amount of residual variance ($\sigma^2 = 0.0003$, $SE = 0.0004$, $p = 0.45$), suggesting that participants tapered in BMI growth at similar rates as they aged. Given the lack of individual variability in the quadratic term, conditional models evaluated predictors of BMI intercept and linear slope only, allowing for covariance between all latent variables.

Hypothesis 1: Effect of ADHD

Via a quadratic LGCM, we evaluated childhood ADHD diagnosis as a predictor of BMI intercept and linear slope with and without adjusting for covariates. Regarding covariates, we modeled SES and childhood diagnosis of anxiety and/or depression as predictors of BMI intercept and linear slope, and stimulant usage as a time-varying covariate across all age bins. As shown in Table 1, both models had excellent fit.

There was no significant difference in initial childhood BMI between girls diagnosed with ADHD and comparisons. Yet girls with ADHD increased in BMI at a significantly greater

rate than comparisons across development ($\beta = 0.23$, $p < 0.001$), with and without adjusting for covariates. As shown in Figure 1 and Table 2, the BMI trajectory of participants with ADHD began to diverge from that of comparison participants in adolescence (ADHD: M_{BMI} = 25.17 kg/m^2, SD_{BMI} = 6.46 kg/m^2 Comparison: M_{BMI} = 22.84 kg/m^2, SD_{BMI} = 3.99 kg/m^2; $t(169) = -2.90$, $p = 0.004$). By adulthood, two-fifths of participants with ADHD (40.2%) had BMI values in the obese range vs. under one-sixth (15.4%) of the comparison group, $\chi^2(1, 167) = 10.38$, $p = 0.001$.

Adjusting for ADHD diagnosis and examining the unique effects of each covariate, we found that higher SES predicted significantly reduced BMI increases across development ($\beta = -0.16$, $p = 0.004$), but childhood diagnosis of depression and/or anxiety was not significantly related to BMI linear slope ($\beta = 0.05$, $p = 0.44$). No predictors were significantly related to BMI intercept. Stimulant usage was not strongly correlated with BMI, although there was a slight negative association in early adulthood ($M_{age} = 21$; $r = -0.10$, $p = 0.03$); see Table 1. When adjusting for the effect of childhood ADHD diagnosis and covariates, there was no longer a significant amount of residual variance in BMI linear slope ($\sigma^2 = 0.25$, $SE = 0.14$, $p = 0.08$), such that this model adequately accounted for individual differences in BMI growth. In contrast, the residual variance of BMI intercept remained significant ($\sigma^2 = 7.97$, $SE = 2.79$, $p = 0.004$), suggesting individual variability in *baseline* childhood BMI unaccounted for by this model.

Hypothesis 2: Effects of EF Measures

To evaluate the relation between three components of EF and BMI trajectory, we used quadratic LGCMs to examine ROCF error proportion scores (planning), CPT commission errors (response inhibition), and CPT omission errors (sustained attention) as predictors of BMI intercept and linear slope, with and without adjusting for childhood ADHD and covariates.

Planning.—Higher ROCF error proportion scores (i.e., worse planning skills) predicted significantly greater BMI growth across development ($\beta = 0.15$, $p = 0.01$), even when adjusting for SES, childhood anxiety/depression, and stimulant usage. This model had excellent fit, as shown in Table 3. Yet when adjusting for ADHD, childhood planning was no longer significantly related to BMI linear slope (whereas childhood ADHD diagnosis remained a significant predictor, $\beta = 0.21$, $p = 0.002$). However, the inclusion of ADHD as a predictor degraded model fit, in that the model chi-square statistic became significant ($p = 0.04$) and the RMSEA 90% CI no longer included zero [0.012, 0.070]. Planning, and all additional predictors, were not significantly related to BMI intercept, and there was significant individual variability in baseline BMI levels unaccounted for by this model ($\sigma^2 = 8.12$, $SE = 2.81$, $p = 0.004$).

Sustained attention.—Higher percentages of CPT omission errors (i.e., worse sustained attention) also predicted significantly greater increase in BMI ($\beta = 0.14$, $p = 0.02$), even when adjusting for covariates. This model had excellent fit (see Table 4). Yet similar to the model with planning, when also adjusting for childhood ADHD diagnosis, the relation between sustained attention and BMI linear slope lost significance, whereas ADHD

remained a significant predictor ($\beta = 0.22$, $p = 0.001$). Again, the inclusion of ADHD as a predictor substantially degraded model fit, in that the model chi-square statistic became significant ($p = 0.03$) and the RMSEA 90% CI no longer included zero [0.014, 0.071]. Sustained attention, along with other predictors in the model, was not significantly related to BMI intercept, and there was significant residual variance in baseline BMI values unaccounted for by this model ($\sigma^2 = 7.99$, $SE = 2.80$, $p = 0.004$).

Response inhibition.—Percentage of CPT commission errors (i.e., response inhibition) in childhood was not a significant predictor of BMI linear slope or intercept, regardless of adjustment for childhood ADHD diagnosis and covariates.

Secondary Tests

To verify that these results remained consistent when accounting for stimulant usage as a possible confound, we also re-conducted analyses including only participants who never used stimulants. These models demonstrated poorer fit due to the constrained sample size ($n = 81$ for comparison, $n = 32$ for ADHD), but ADHD diagnosis remained a significant predictor of BMI linear slope with acceptable fit indices (after adjusting for a Heywood case; van Driel, 1978; Kolenikov & Bollen, 2012). Additionally, given the multiple sources of weight/height at Wave 4 (i.e., objective measurement and self-report), we also re-conducted all analyses adjusting for the relation between Wave 4 BMI source and BMI intercept/linear slope. Wave 4 BMI source was not significantly related to BMI linear slope; its inclusion as a covariate did not alter findings.

Discussion

We examined trajectories of BMI growth longitudinally from childhood to adulthood in girls with ADHD, hypothesizing that girls with childhood-diagnosed ADHD would increase in BMI more rapidly than comparison girls over time. Results from latent growth curve models support this hypothesis. Although participants with ADHD did not significantly differ from comparisons in childhood BMI, they increased in BMI at a significantly greater rate across development, resulting in substantially higher BMI levels in adolescence and adulthood. By adulthood, 40.2% of the ADHD sample were classified as obese, in contrast to only 15.4% of the comparison group. Additionally, we investigated three childhood executive functioning (EF) deficits (i.e., planning, response inhibition, and sustained attention) as transdiagnostic predictors of BMI trajectories. No EF predictors retained significance after accounting for ADHD diagnostic status. Findings were not altered by inclusion of key covariates (e.g., common comorbidities, SES, stimulant usage).

These findings enhance our understanding of health risks associated with ADHD among women and lend insight into an important early risk factor for later weight gain. Whereas obesity rates escalate in adolescence and adulthood (Ogden et al., 2018), ADHD is generally diagnosed in childhood (Visser et al., 2014). ADHD diagnosis may therefore help to identify individuals at risk for obesity before weight problems emerge, at a time when health behaviors may be more malleable. This finding may inform intervention research in two ways: (1) obesity-related interventions may benefit from targeting deficits associated with ADHD and (2) ADHD interventions may benefit from promoting healthy eating and

exercise behaviors, as well as body image coping skills, before BMI differences emerge. Additionally, our findings have theoretical significance, supporting the contention that some combination of mechanisms associated with ADHD may contribute to concerning weight gain.

Results indicate that three childhood EF components (i.e., planning, sustained attention, and response inhibition) were not significantly related to BMI growth beyond their associations with ADHD diagnosis. Indeed, prior research indicating relations between EF and BMI has been largely cross-sectional, and most studies have only focused on EF deficits in individuals who are already overweight or obese (e.g. Yang et al., 2018; Gunstad et al., 2007). Thus, it is possible that other mechanisms linked to ADHD (whether heritable or contextual), and even other components of EF, may be more viable predictors of BMI growth.

That said, our dataset may be underpowered to evaluate independent contributions of EF on BMI increase. As noted above, we found no significant variance in BMI linear slope remaining after accounting for ADHD diagnosis and covariates. Due to this lack of individual variability in BMI growth, our models including ADHD diagnosis and covariates would not be likely to detect the influence of any additional predictors on BMI increase, including our EF measures (Tarka, 2018). Additionally, the strong association between the components of EF explored here and ADHD (e.g., see Sami et al., 2004) may have made it especially difficult to discern any unique effects of our EF measures on BMI increase. More research is needed on these transdiagnostic predictors outside of an ADHD-based sample.

We also assessed the effects of three covariates in connection with BMI growth. Higher SES in childhood predicted lower BMI increase across development, consistent with literature indicating a negative association between SES and BMI (Ball & Crawford, 2005). Childhood anxiety and depression diagnoses were not significantly related to BMI growth beyond the effect of childhood ADHD. Prior literature indicating relations between anxiety/depression and BMI has not accounted for the potential role of ADHD-related symptoms or symptoms shared between ADHD and anxiety/depression (e.g., Pine et al., 2001; Goodman & Whitaker, 2002). Even so, our sample was ascertained with respect to ADHD, rather than internalizing conditions, so additional research on samples enriched for depression and anxiety is necessary. Regarding the potential confound of stimulant medication, several researchers have investigated how stimulants are related to BMI growth across development, but findings have been quite mixed (e.g., Biederman et al., 2003; Waring & Lapane, 2008). Although our naturalistic investigation did not manipulate medication, results suggest that stimulant use was slightly negatively related to BMI in early adulthood (i.e., ages 19–24). Still, after adjusting for the effect of stimulant usage on BMI, the relation between ADHD and BMI growth did not substantially change.

Limitations

One study limitation was our multiple BMI sources: calculations were based on parent-reported height and weight at Waves 1–2 (ages 6–17) and objective measures at Waves 3–4 (ages 17–29)—supplemented with self-reported values for a subset of participants (7% at Wave 3, 29% at Wave 4). Compared to objective measurements, parent-reported values tend

to *overestimate* childhood BMI calculations (on average by 0.8–2.2 kg/m^2 for ages 2–8; Weden et al., 2013) but to *underestimate* adolescent BMI (see also Akinbami & Ogden, 2009; O'Connor & Guggenheim, 2011). Concerning self-reported values, women slightly underestimate their actual BMI (on average by 0.1–0.8 kg/m^2; Pursey et al., 2014). Although average discrepancies between reported and measured BMI values are often small, authors caution that by adulthood, the magnitude of BMI underestimation is greater for heavier individuals (Weden et al., 2013; Kovalchik, 2009). Thus, participants with ADHD may have been disproportionately affected by BMI underestimation biases, given their significantly higher BMI values than comparisons by Wave 2. Our findings may therefore underestimate BMI growth of our participants, particularly for those with ADHD. In short, our LGCM analyses are likely to be conservatively biased.

Our measures of EF components and stimulant usage also present limitations. First, concerning EF, utilizing laboratory-based neuropsychological measures potentially constrained the ecological validity of our findings (Burgess et al., 2006), especially considering the contextual complexity of eating behaviors. For example, an individual's ability to refrain from hitting a button related to visual prompts (i.e., response inhibition) may not adequately reflect her ability to resist urges to eat due to heightened emotions or situational cues. Some researchers have raised concerns about the use of Conners' CPT subtests in accurately identifying deficits in individuals with ADHD (e.g., Perugini et al., 2000). Additionally, our CPT measures did not account for response biases (e.g., pressing the response key quickly but indiscriminately). Second, our measure of stimulant usage relied on parent- and self-report and required significant recall, approximating usage frequency for the past 4–5 years. We also lacked information about timing of medication usage. Third, our study contained a notable amount of BMI data missing at Wave 1 due to incomplete BMI data collection (33% missing). Although we estimated missing data with a robust approach in our LGCMs (i.e., full information maximum likelihood estimation; Bollen & Curran, 2006), we still consider this a study limitation.

Finally, all participants were recruited from the San Francisco Bay Area. It follows that our sample is not reflective of national U.S. and/or global demographics, which may limit generalizability. For example, the U.S. national adult obesity rate is 42.4% (Hales, 2020), whereas in the San Francisco Bay Area it is estimated at 20.1% (Wolstein et al., 2015). The 40.2% obesity rate found in our adult women with ADHD is therefore more striking in the context of regional versus national norms. Moreover, the 15.4% obesity rate in our comparison sample in adulthood is lower than the Bay Area 20.1% estimate. Further national and international research is needed with samples more reflective of the general population.

Conclusions and Future Directions

Overall, the present data reveal that childhood ADHD predicts increased BMI growth from childhood to adulthood in girls/women, resulting in higher obesity rates in adulthood relative to comparisons. The prospective, longitudinal nature of this investigation suggests that ADHD in childhood temporally precedes increased BMI growth in adolescence and

adulthood. It remains unclear, however, whether shared genetic factors between ADHD and obesity/high BMI might account for this effect (see Do et al., 2019; Cortese, 2019).

Moreover, these findings provide insight into strategies to prevent long-term challenges, such as obesity, associated with elevated BMI increase in girls with ADHD. Trajectory analyses suggest that noticeable BMI increases in girls with ADHD are likely to emerge in adolescence (ages 15–18) and continue escalating through adulthood (ages 19–30), though somewhat diminishing with time (given the significant quadratic trend). It may therefore be important to target health interventions in early adolescence before substantial weight gain is most likely to occur. This approach may also aid in the prevention of eating disorders, which are more common in girls with ADHD compared to the general population (Biederman et al., 2007).

It is important to note that BMI and health are not synonymous (e.g., Rey-Lopez et al., 2014). Instead, BMI is just one piece of our complex understanding of health and longevity. In fact, stigma related to larger body size is uniquely harmful to mental health and has been linked to a variety of negative psychological outcomes including disordered eating and suicidality (e.g., Brochu, 2020; Heras et al., 2010). Indeed, the findings here should not be used to generate further stigma related to larger body size. Rather, particularly for girls with ADHD, we need a better understanding of BMI-health connections as well as early coping skills for negative body image. More research on societal body-size stigma reduction is crucial to help these psychiatrically vulnerable girls.

Future studies should also investigate additional predictors and mechanisms of BMI increase in girls with ADHD to best inform targeted intervention strategies. Other dimensions of EF not assessed here, including “hot” EF, may be good candidates for future study. For example, individuals with ADHD often prefer smaller immediate rewards to larger delayed rewards (Mies et al., 2019), a tendency also associated with obesity (Amlung et al., 2016). Thus, reward processing may be a key predictor of interest for future researchers. “Real world” measures of EF (e.g., observational data and informant reports) will also be important given the limitations of our neuropsychological measures described above. Moreover, considering that ADHD status was a significant predictor of BMI change, ADHD symptom domains, as well as their change over time, are important candidates for future study. Further longitudinal trajectory research should also explore the potential for bidirectionality by examining how increases in BMI may affect ADHD (as well as other psychiatric) symptoms. Although we focused on neuropsychological mechanisms, additional socioenvironmental and mental health vulnerabilities may increase risk of obesity among girls with ADHD (alone or via interaction with neuropsychological factors), such as SES, psychiatric comorbidities, and peer and familial relationships. Finally, future studies should include more in-depth research on the role of stimulant medications (such as timing and duration of medication administration) to clarify their effects on BMI growth with more specificity.

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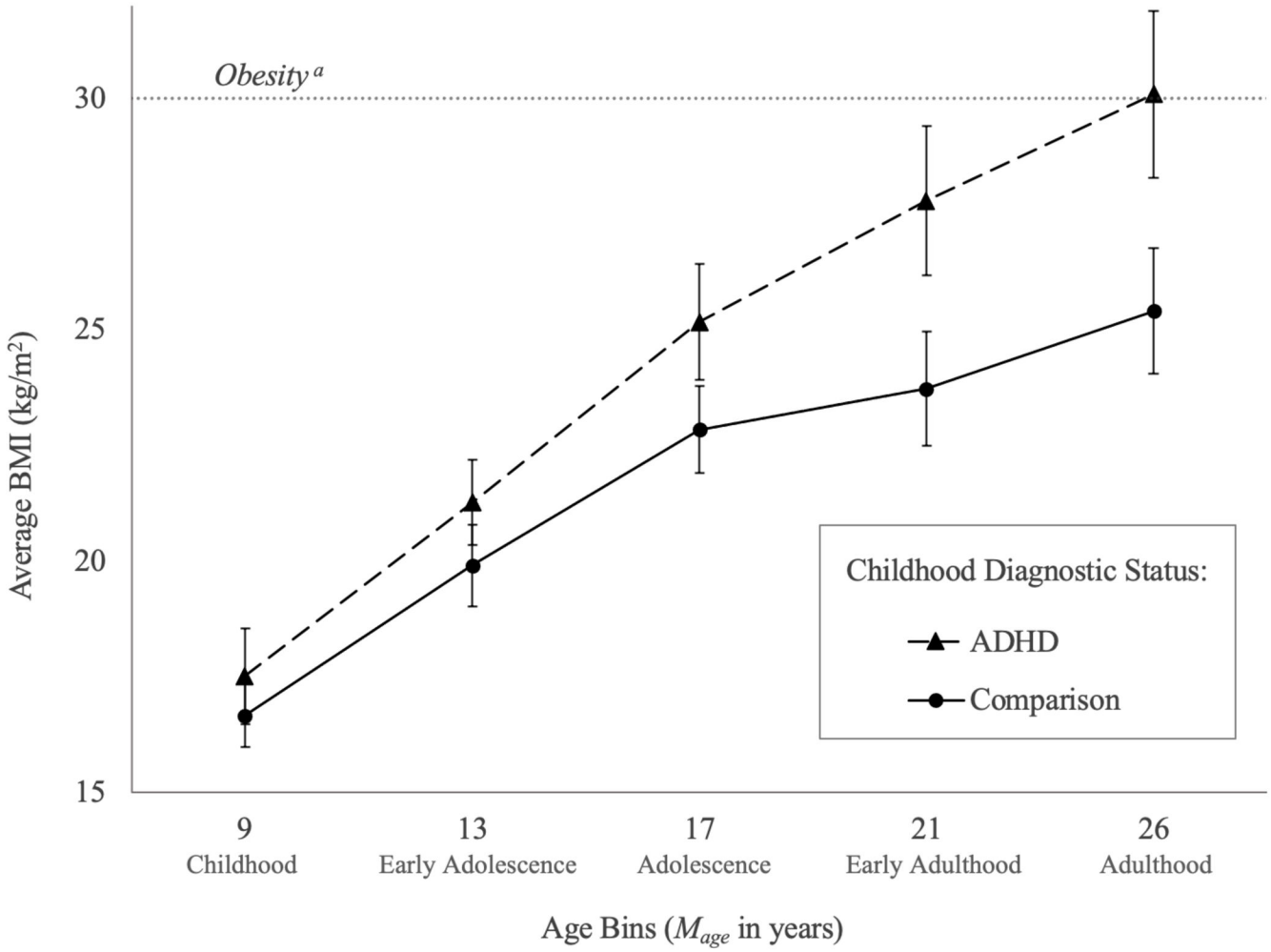

Figure 1. BMI Trajectories of Girls with and without Childhood ADHD

Note. Average body mass indices (BMI) of girls with and without childhood ADHD at each age bin, including 95% confidence intervals.

[a]BMI threshold for obesity (≥ 30 kg/m^2).

Table 1

Effect of ADHD on BMI trajectory

	ADHD		ADHD + Covariates	
	b (SE)	***β***	***b (SE)***	***β***
Estimated Effects				
BMI Linear Slope				
ADHD	0.30 (0.06)	0.27***	0.25 (0.07)	0.23***
SES			−0.10 (0.04)	−0.16**
Dep/Anx			0.06 (0.08)	0.05
BMI ~ Stimulant Use[a]				
Childhood			−0.30 (0.66)	−0.04
Early Adolescence			−0.56 (0.29)	−0.10
Adolescence			−0.45 (0.30)	−0.07
Early Adulthood			−0.95 (0.43)	−0.10*
Adulthood			0.09 (0.54)	0.007
Model Fit Indices				
χ^2 (*df, N*)	χ^2 (9, 219) = 16.28, *ns*		χ^2 (35, 219) = 46.69, *ns*	
CFI	0.987		0.980	
TLI	0.978		0.971	
RMSEA [CI_{90}]	0.061 [0.000, 0.107]		0.039 [0.000, 0.066]	
SRMR	0.036		0.037	

Note. Both models evaluate childhood ADHD diagnosis as a predictor of BMI intercept and linear slope across the lifespan, using a quadratic LGCM. The second model also adjusts for covariates (i.e., family SES, childhood diagnosis of depression and/or anxiety, and stimulant usage, with the latter modeled as a time-varying covariate). No predictors were significantly related to BMI intercept.

[a]Covariance between stimulant usage and BMI at each age bin

*$p < 0.05$;

**$p < 0.01$;

***$p < 0.001$

Table 2

BMI averages and obesity rates across development

	Childhood	Early Adolescence	Adolescence	Early Adulthood	Adulthood
Average BMI (*SD*)					
ADHD	17.51 (4.25)	21.26 (4.46)	25.17 (6.46)	27.79 (8.25)	30.08 (9.33)
Comparison	16.66 (2.39)	19.90 (3.62)	22.84 (3.99)	23.72 (5.17)	25.40 (5.58)
t	−1.34	−2.10	−2.90 *	−3.93 **	−4.06 ***
Obesity Rates					
ADHD	0.0%	4.4%	19.8%	28.7%	40.2%
Comparison	0.0%	1.5%	2.9%	7.4%	15.4%
χ^2		0.32	8.77 *	10.25 *	10.38 *

Note. We evaluated differences in body mass indices (BMI; kg/m^2) and obesity rates (i.e., proportion of girls with BMI ≥ 30 kg/m^2) between ADHD and comparison girls at each age bin, using independent samples t-tests and chi-squared tests, respectively.

* $p < 0.01$;

** $p < 0.001$;

*** $p < 0.0001$

Table 3

Effect of planning on BMI trajectory

	ROCF		ROCF + ADHD	
	b (SE)	***β***	***b (SE)***	***β***
BMI Linear Slope				
Poor Planning (ROCF)	0.42 (0.17)	0.15*	0.23 (0.18)	0.09
ADHD			0.22 (0.07)	0.21**
Model Fit Indices				
$\chi^2(df, N)$	$\chi^2(35, 219) = 48.54$, *ns*		$\chi^2(38, 219) = 55.05$*	
CFI	0.977		0.971	
TLI	0.966		0.958	
RMSEA [CI_{90}]	0.042 [0.000, 0.069]		0.045 [0.012, 0.070]	
SRMR	0.035		0.038	

Note. Using quadratic LGCMs, we evaluated poor planning (based on ROCF error proportion scores) in childhood as a predictor of BMI intercept and linear slope across the lifespan with and without accounting for childhood ADHD diagnosis. Both models adjust for covariates (i.e., SES, depression/anxiety, and stimulant usage).

ROCF = Rey-Osterrieth Complex Figure Test (copy condition)

* $p < 0.05$;

** $p < 0.01$

Table 4

Effect of sustained attention on BMI trajectory

	CPT % Omissions		CPT % Om. + ADHD	
	b (SE)	***P***	***b (SE)***	***P***
BMI Linear Slope				
Poor SA (CPT % Om.)	0.38 (0.16)	0.14 *	0.24 (0.16)	0.09
ADHD			0.23 (0.07)	0.22 **
Model Fit Indices				
χ^2 (*df, N*)	$\chi^2(35, 219) = 48.91$, *ns*		$\chi^2(38, 219) = 55.62$ *	
CFI	0.976		0.970	
TLI	0.966		0.957	
RMSEA *[CI_{90}]*	0.043 [0.000, 0.069]		0.046 [0.014, 0.071]	
SRMR	0.033		0.038	

Note. Using quadratic LGCMs, we evaluated sustained attention (based on the percentage of CPT omission errors) in childhood as a predictor of BMI intercept and linear slope across the lifespan with and without accounting for childhood ADHD diagnosis. Both models adjust for covariates (i.e., SES, depression/anxiety, and stimulant usage).

SA = sustained attention

CPT = Conners' Continuous Performance Test

* $p < 0.05$;

** $p < 0.01$