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Attention-Deficit/Hyperactivity Disorder (ADHD) and Overweight/Obesity: New Data and Meta-Analysis

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ABBREVIATIONS: ADHD=Attention-deficit/hyperactivity disorder; BMI=body mass index;
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Introduction

In the past decade, an important claim has been made asserting an association between ADHD and overweight or obesity (Chen, Kim, Houtrow, & Newacheck, 2010; Cortese & Angriman, 2008; Cortese & Morcillo Penalver, 2010; Fuemmeler, Ostbye, Yang, McClernon, & Kollins, 2011; Pagoto et al., 2009; Taurines et al., 2010; Waring & Lapane, 2008). Exploring this claim, the literature offers a number of hypotheses as to the putative association between ADHD and obesity. Two of the symptoms central to ADHD, impulsivity and inattention, may increase the risk of obesity through separate, but related, means (Cortese, Faraone, Bernardi, Wang, & Blanco, 2013). Relatedly, the reward deficiency hypothesis, which suggests that the etiology of ADHD is due, in part, to the insufficiency of brain dopamine (Blum et al., 2000), may play an interrelated role as well. Impulsivity is thought to contribute to obesity through disordered eating patterns (Davis, Levitan, Smith, Tweed, & Curtis, 2006), both in quantity, such as binge eating (van Egmond-Fröhlich, Widhalm, & de Zwaan, 2012) and type of foods consumed (e.g. highly calorie, nutrient poor) (van Egmond-Fröhlich, Weghuber, & de Zwaan, 2012). Dysregulation of dopamine may mediate consumption of high caloric, low nutrient foods (Tanofsky-Kraff & Yanovski, 2004) as well as impulsivity, in turn contributing to the consumption of high caloric foods, low in nutrient value (i.e. processed or fast foods) and contributing to overweight or obesity (Davis et al., 2006). Binge eating behaviors, unmoderated by the impulsivity associated with ADHD, may compensate for the dopamine deficiency thought to be present in individuals with ADHD (Wang et al., 2001). These hypotheses are supported by the efficacy of stimulant medication, which reduces impulsivity, suppresses appetite (Faraone, Biederman, Morley, & Spencer, 2008) and increases synaptic dopamine availability by blocking the dopamine transporter (Schulz, Fan, Bédard, & et al., 2012; Spencer et al., 2005). Stimulant medication has been associated with lower BMI in early puberty (Poulton et al., 2013), reflecting its appetite suppressing qualities (Faraone et al., 2008).

Inattention, in turn, may play a role in obesity by diminishing an individual's receptivity to internal satiation cues (Cortese & Vincenzi, 2012) or contributing to difficulty in maintaining regular patterns of eating, thus leading to disordered consumption habits (Davis et al., 2006). Further, poor planning and self-regulation deficits may interfere with follow through on activities requiring effortful control and planning, such as exercise and dietary changes, which promote healthy weight (Cortese & Vincenzi, 2012). Finally, individuals with ADHD tend to have more mood lability than their non-disordered counterparts (Oliver, Nigg, Cassavaugh, & Backs, 2012). This fluctuating negative affect may contribute to a propensity to choose pleasurable, high caloric foods to "medicate" the low mood (Davis, Strachan, & Berkson, 2004; Wang et al., 2004).

Despite the plausibility of an association between ADHD and overweight/obesity, recent reviews (Cortese & Vincenzi, 2012; Korczak, Lipman, Morrison, Duku, & Szatmari, 2014) have been rather mixed on its robustness. The literature suggests that the clinical importance of this association may depend on moderators. A number of putative, but rarely tested factors have been noted for their association between ADHD and overweight/obesity. Age effects have rarely been tested directly, but when they were, differential age effects were reported. For example, two studies (Fliers et al., 2013; A. W. van Egmond-Fröhlich et al., 2012) found that effects were smaller in younger than older children, although Fliers et al found a curvilinear effect with age. These findings suggest that effects may be most apparent in adulthood, rather than in childhood. However, we know of only three studies that reported results for samples limited to children under age 10; all controlled for medication effects and still found that ADHD or ADHD symptoms were associated with lower, not higher BMI (Ebenegger et al., 2012; Fliers et al., 2013; A. W. van Egmond-Fröhlich et al., 2012). It may be ADHD's effect on obesity is gradual and does not emerge until the natural weight and fat gain of adolescence and adulthood relative to childhood amplifies a subtle ADHD effect. This would be relevant to the developmental timing of any secondary prevention proposals for obesity in youth with ADHD.

Second, only a handful of studies have tested differential effects by gender of the participant. In the two most methodologically rigorous studies, which directly compared effects in adolescents (Cortese, Faraone, et al., 2013; A. W. van Egmond-Fröhlich et al., 2012), both concluded that effects were confined to females. These findings need support from additional data and then clarification in a pooled analysis with other studies. It may be that, because girls normatively gain more body fat than boys after puberty due to differential hormonal development, girls with ADHD are more vulnerable to excess weight gain in adolescence and adulthood.

The putative confounders of age, gender, or their combined effects are of importance for several reasons. ADHD is most commonly diagnosed in boys, tends to modulate in adolescence, with some worsening, and others recovering amidst normative decreases in hyperactivity. Also in adolescence, girls' and boys' outcomes begin to diverge, congruent with wider gender differences in risk for psychopathology related to depression, mood disorders, substance use onset, risky sexual behavior, and delinquency (Nigg, 2013). If there are also gender differences in risk for overweight or obese status, the intersection of these outcomes would become clinically important.

Third, ADHD's many comorbidities are of obvious importance, but effects are difficult to isolate and are often overlooked in studies of the ADHD-obesity association. Two main comorbidities have been considered in the literature: depression and disruptive behavior problems such as conduct disorder. We note that depression is the most important of these, as it is an established risk factor for excess BMI and obesity, although that effect may be larger in females than in males (Berkowitz & Fabricatore, 2011; de Wit et al., 2010; Korczak et al., 2014). Once again, clarification of this effect could have important implications for targeted treatments, for identifying youth at risk, and for consideration of mechanisms. Conduct problems often co-occur with ADHD and are confounded by increased ADHD severity. Nonetheless, it is important to consider them as well.

In examining the literature for this review, it was noted that few studies were focused on children; most included teenagers through high school. Only a minority provide data enabling examination of the effects gender, age, or comorbidity, much less considering all three. Medication status was not considered as consistently as one might expect given its obvious relevance. We therefore precede our meta-analysis by supplying new data addressing these shortages in the literature, in order to increase the total power and sample size available for the meta-analysis and in particular for the moderator analyses.

Based on the cited literature and the new data presented, this paper sought to clarify, (a) the population effect size of the association of ADHD and overweight/obesity/BMI; and (b) the relative moderation of this effect by medication status, gender, age (child versus adult), and psychiatric comorbidity.

Part I. New Data to Support Meta-Analysis

Preliminary Study 1: Short term study of children

Methods

Participants. The first study focused only on children under the age of 14, and was conducted to provide effect size data for the longitudinal or prospective effects of ADHD on subsequent BMI change, controlling for gender and depression. Three hundred and thirteen families with children aged 7-11 years old at baseline (54% with ADHD, 62% male) were followed for two years to create an accelerated longitudinal design spanning ages 7-13 years (See Table 1 for demographic and diagnostic information for the full sample and by ADHD diagnostic group status). Families were recruited from the local community through mass mailings using commercial mailing lists to the entire population of parents of eligible-aged children in the greater metropolitan area of Portland. The local Institutional Review Board approved the study. Parents provided written informed consent and children provided written assent.

Diagnostic group and clinical characterization. Common rule outs for ADHD studies

were applied including neurological impairments, history of seizures, traumatic brain injury, major medical conditions, intellectual disabilities or estimated IQ<75, or pervasive developmental disorders or autism, lifetime mania or psychosis, or learning disability. Diagnosis was by standardized, research-valid procedures and included parent and teacher clinical rating scales (Conners, 2008; DuPaul, Power, Anastopoulos, & Reid, 1998; Goodman, 2001) and a clinician administered semi-structured interview of a parent using the KSADS-E (Puig-Antich & Ryan, 1996)which evaluated ADHD, depression, and conduct disorder (other evaluated disorders are ignored in this report). Depression data were also evaluated by child self-report using the Child Depression Inventory (CDI) (Kovacs, 1985). Interviews were monitored for inter-interviewer reliability (k>0.80 for ADHD for all interviewers) and fidelity to interview procedures by spot-checking of interview videotapes by a senior clinician. Children completed an IQ and academic functioning screening, and a clinician briefly interviewed and observed the child and wrote detailed notes. All data were then submitted to a best-estimate clinical diagnostic team that included a neuropsychologist and psychiatrist) who assigned final ADHD, control, or exclusion assignments. Inter-clinician agreement rates were acceptable (k>0.70 for all disorders studied here). Disagreements were resolved by discussion. For full details relating to diagnostic procedure see Musser et al., 2010.

Psychiatric Comorbidity. A binary indicator was created to represent the presence of either oppositional defiant or conduct disorder at each assessment. Similarly, a binary indicator was created to represent the presence of any mood disorder (major depression or dysthymia; there were no cases of bipolar) at each assessment. Diagnoses were made by the diagnostic team decision, based on all available information (including the CDI for depression and the KSADS, parent and teacher ratings, and staff observation notes for all other disorder).D. In Table 1, Wave 1 for the depression variable refers to whether the child had been diagnosed ever in his or her lifetime, while Waves 2 & 3 refer to the time since the previous wave or assessment.

Follow up. After baseline evaluation, children were followed annually for two years for a total of three assessments. Because children were 7-11 years old at enrollment, the design covers development from ages 7-13 years to target the child age range of interest.

Anthropometric Measurements and Body Mass Index. Children stood wearing a single layer of clothing and in stocking feet to have height measured with a stadiometer (Wall-Mounted Height Rod, HR-200, Tanita Corp., Arlington Heights, IL) and weight measured with a digital scale (Body Composition Analyzer, TBF-410GS, Tanita Corp., Arlington Heights, IL). In some cases this was not available and height and weight were measured with a permanently fixed and calibrated wall chart and analog scale, respectively, ("Big Foot" Model 150, Health-o-Meter Professional Scale, Jarden Consumer Solutions, Boca Raton, FL). BMI was calculated by dividing weight in kilograms by height in meters-squared. BMI by the two methods was nearly identical (Time 1, $r = 0.976$, $n = 38$; Time 2, $r = 0.983$, $n = 87$) so these were combined in all analyses reported here, with the analog data filling in when the Tanita was not obtained.

Medication History and Status. Current (but not lifetime) use stimulant or other psychoactive medications was exclusionary at Time 1 and free to vary at Time 2 and Time 3, where its effect was modeled statistically. Current and past medications were evaluated by a semi-structured interview using a modified version of the SCAPI (Jensen et al., 2004). This was repeated at 6-month intervals to ensure that the Time 2 and Time 3 assessments had full medication information. Two variables were retained as covariates: lifetime use of any psychoactive medication prior to Time 1, and any use of any psychoactive medication between Time 1 and Time 3.

Analytic Strategy. The main research question was whether children with ADHD differed from their non-ADHD peers with respect to BMI or BMI change. Hierarchical linear models were estimated to test whether ADHD was associated with a differential baseline or trajectory of change in BMI from ages 7-13. Full information maximum likelihood estimation accommodated different numbers of BMI values across individuals. Conditional models tested

whether ADHD status was associated with initial BMI or the rate of change in BMI across time. Two indicators of socioeconomic status (parental education, household income—both standardized to have a mean equal to 0) and child gender (male) were used as time-invariant covariates. Child use of stimulant medication, presence of oppositional defiant or conduct disorder, presence of any mood disorder, and pubertal status were included as time-varying covariates.

Results of Preliminary Study 1

Table 1 provides a descriptive summary of the sample. Participants were primarily male (62%), Caucasian (82%), and of slightly above average intelligence (Full scale IQ *M* = 112). At the three annual assessments the average age in years was 9 (range 7-12), 10 (range 8-13), and 11 (range 9-13). Reflecting population rates, youth with ADHD were disproportionately male and had lower overall IQ, although both groups had average to high-average IQ. About half of the ADHD youth were treated with stimulants at some point during the study. Covarying for age, there was no evidence for group differences in pubertal level at any wave. Among the N=313 children who participated in the first wave of the study, 83% and 72% provided BMI scores at the 2nd and 3rd assessment waves (this was partly by design, as the third wave was still ongoing). Thus, the N = 313 study participants generated J = 797 person observations, with the preponderance of those observations occurring between the age of 7-11 years. At no age point between 7 and 13 did the association of ADHD and BMI become reliably different than zero (*p*s = .07-.74).

Growth Models. The initial results thus suggested that ADHD status was unrelated to BMI across the study period. However, those comparisons did not adjust for covariates nor test for differential rates of change in BMI across time.

Unconditional Models. Descriptively, a linear functional form best characterized changes in BMI across time. Formally, an unconditional growth curve model was estimated in which BMI was regressed on child chronological age (linear slope) and age^2 (quadratic slope) terms. Age

was centered such that the intercept referred to the average BMI at age 8. There was evidence of significant linear, but not nonlinear (quadratic) change in BMI across time; moreover, there was evidence for inter-individual differences in both level and rate of change in BMI across time (*p*s < .0001). This model therefore was re-estimated omitting the age^2 term (**Table 2**), which served as a baseline model for characterizing change.

Conditional Models. The initial conditional model considered ADHD as a predictor of initial level and rate of change in BMI (model 3 in Table 2) without covariates. Neither the main effect of ADHD (*b* [se] = -.21 [.26], *p* = .42) nor the ADHD x age interaction (*b* [se] = -.17 [.09], *p* = .07) terms were statistically significant in relation to BMI, although the latter effect approached the significance cutoff. A subsequent model (model 4 in Table 2) introduced household income, parental education, and child gender as time invariant covariates and mood and oppositional defiant/conduct disorders, as well as stimulant use, as time varying covariates (in preliminary models we determined that none of these covariates interacted with age). Household income (*b* [se] = -0.29 [.13], *p* = .02) exerted a main effect, such that children from more privileged homes had lower overall levels of BMI. Moreover, stimulant use was associated with time-specific reductions in BMI (*b* [se] = -.65 [.19], *p* = .0005). However, the inclusion of these covariates did not change the substantive interpretation with respect to ADHD; ADHD was still not associated with the initial level or rate of change in BMI across time. A final model (model 5 in Table 2) introduced child pubertal status as an additional time varying covariate. This model was based on fewer observations than the previous models (because pubertal status data were not collected on the youngest children). The results with respect to ADHD were unchanged.

Preliminary Study 2: National survey data of age and gender effects in ADHD-BMI

Methods

Participants, survey description, sample weighting and generalizability. The second study considered the need for an additional large, population-based dataset to consider gender and age-specific associations. This study was conducted using the 2011/2012 iteration

of the National Survey of Children's Health (NSCH) (USA National Survey of Children's Health, 2014). The NSCH is a nationwide telephone survey that was conducted across the US, in English and Spanish, for the first time in 2003-2004. Data from that survey were used to examine ADHD and BMI/obesity in two studies (Kim, Mutyala, Agiovlasitis, & Fernhall, 2011; Waring & Lapane, 2008). A second survey was fielded in 2007-2008; those data were examined for ADHD and BMI/obesity effects in two further reports (Halfon, Larson, & Slusser, 2013; Lingineni et al., 2012). All four prior analyses of NSCH data controlled for many confounders, but none of them considered age or gender as effect modifiers. Data from the most recent NSCH survey, conducted in 2011-2012, (USA National Survey of Children's Health, 2014) are analyzed here. To our knowledge they have not been previously examined for ADHD-obesity/overweight effects.

In the NSCH, telephone numbers are called at random to identify households with one or more children under 18 years old. In each household, one child was randomly selected to be the subject of the interview. The survey results are weighted to represent the national population of non-institutionalized children 0-17 years and in each of the 50 states plus the District of Columbia. The U.S. Department of Health and Human Services, Health Resources and Services Administration, Maternal and Child Health Bureau provided the primary funding for the surveys. The National Center for Health Statistics of the Centers for Disease Control and Prevention conducts the survey and produces a public-use data set.

Age stratification. The 2011-2012 NSCH provides a BMI calculation for children ages 10-17 years (n=45,309). Thus results are reported only for children 10-17 years, and the sample was stratified into a "child" sample aged 10-13 years (n=21,497) and an "adolescent" sample aged 14-17 years (n=23,812) for purposes of examining age effects using a stratified design.

Definition of BMI and obesity status. BMI (kg/m^2) was calculated within NSCH using heights and weights reported by parents in the survey. Children were divided into four weight groups based on national standards, with those < 5^{th} percentile as underweight, 5^{th}-84^{th}

percentile as normal weight, 85th-94th percentile as overweight, and >95th percentile as obese. This was consistent with how several other population studies classified weight categories. In the primary analysis herein a two group design was used comparing (a) pooled overweight and obese children with (b) all other children.

Definition of ADHD. The survey provides four options for defining ADHD, which likely provide varying degrees of validity: (a) Ever ADHD: "have you ever been told by a health care professional this child has ADHD?" In the 2011-2012 survey, this question yielded a prevalence of 14%. While this definition was used in prior reports using earlier editions of the NSCH, it is likely to be overly inclusive. (b) Enduring ADHD: has been told in the past the child has ADHD and currently has ADHD, 10.8% prevalence. (c) Severity: currently has ADHD and is rated by parent as mild (5.0%) versus moderate or severe (5.7%). (d) ADHD-medication: 7.5% of the population was designated as currently having ADHD and taking medication for it. Analyses were conducted on each of these four definitions of ADHD to take into account the possibility that both narrow and broad definitions of ADHD may detect risk for overweight or obese status in the population survey setting.

Definition of depression. Depression was defined in a similar fashion as ADHD, by questions asking whether the parent had ever been told the child was depressed by a health care professional, was still depressed, or was treated for depression. Here, to maximize power in terms of size of groups for stratified analyses, we defined depression as "ever told by a health care professional that the child had depression."

Definition of conduct/disruptive behavior disorder. Conduct/disruptive disorder was defined similarly to depression, asking whether the parent had ever been told the child has a behavioral or conduct problems, such as oppositional defiant disorder or conduct disorder and whether the child currently has the condition. Again to maximize power we created a dichotomous variable with conduct/disruptive disorder defined as those ever or currently diagnosed with a conduct/disruptive disorder as opposed to those never diagnosed.

Data analysis and covariates. The association between ADHD and BMI-group (underweight/normal, overweight/obese) was modeled using logistic regression with the appropriate use of the weighting, ID, and state variables as required for the NSCH data set, using the SAS Complex Models procedure PROC SURVEYLOGISTIC in SAS version 9.4. When main effects were non-significant at $p<0.05$, we did not pursue covariates, but proceeded with our planned stratified analyses. When effects were identified, we examined as covariates effects that were not yet stratified (e.g., gender in age-stratified groups). To address the potential modifying effect of age and gender, data were stratified first by age group, child (10-13 years old) versus adolescent (14-17 years old) and further stratified by gender within age group.

As a proxy for SES, poverty level and subsidized school lunch eligibility were covaried in secondary analysis with no change in results or conclusions, thus SES was not maintained as a covariate in our final model.

Results of Preliminary Study 2.

Table 3 provides a simple cross tabulation of the association between ADHD and BMI group category stratified by age group and gender. It provides the raw frequencies from which population parameters were estimated in logistic regression models.

Table 4 provides the results of the logistic regression models. In the first model, the newest national survey data shows a cross sectional association between the parent ever being told the child had ADHD, and child BMI group, although the effect is small (OR = 1.17, 95% confidence interval [CI] = 1.03 – 1.34). Subsequent rows of the table show results by age, then age by gender, then by adjustments for depression alone or together with conduct disorder. When stratified by age, the effect is trivial in size and not significant for children (OR 95% CIs contain 1.0), but remains moderate (and reliably different than null) in adolescents (point estimates of ORs = 1.32 – 1.50, depending on definition of ADHD). Second, when we stratify by gender and age, effects for adolescent boys are small and not statistically reliable (95% CIs contain 1.0), but for adolescent girls, effects are moderate in size and reliably different from null

(point estimates of ORs range from 1.73 – 2.51 depending on definition of ADHD). When depression is covaried, the effect size for adolescent girls decreases (OR ranges = 1.48-2.01), but remains reliably different than null. However, when depression and conduct are covaried together, the association between ADHD and BMI is no longer reliably different than zero. **Table 4** also shows that, qualitatively, as the definition of ADHD is rendered more stringent, possibly constituting a more severe subgroup, its association with BMI tends to increase.

Part II. Meta-analysis

Methods

Study selection

Literature Search. Relevant studies were identified through literature searches using the PubMed and PsycINFO electronic databases. Search terms used were "obesity" or "overweight" or "body mass index" or "bmi," and “ADHD” or “attention-deficit/hyperactivity disorder” or “attention-deficit hyperactivity disorder” or “attention problems” or “hyperactivity-impulsivity.” To avoid publication bias, doctoral dissertations were identified and considered as well, and non-English language publications were evaluated. Reference sections from relevant articles were examined for additional resources and included if the studies met criteria. Studies in all languages through June 2014 were considered. Identification of appropriate studies was conducted by both the first and second authors (JN and JJ), independently, and any discrepancy discussed and resolved by consensus. These authors read full abstracts of relevant studies (selected if either rater judged the study by title to be potentially relevant). Next, full text versions of relevant studies were further assessed for eligibility. Agreement between the raters was 95%. The specifics regarding the number of studies reviewed and included/excluded at each review step is found in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) Flow Chart, displayed in **Figure 1**.

Data extraction and moderators. Data from the included studies were double checked (by JN and JJ) to maximize accuracy. Coded information, which served as potential moderators

based on a review of the literature, included participant gender; age at baseline, and for longitudinal studies, age at final analyses; whether or not conduct disorder or depression (CD/Dep) were covaried, and whether or not the study controlled for the use of stimulant medication. Quality of ADHD evaluation was evaluated as a moderator. The ADHD quality was scored on a 1-5 scale where 1 was best (structured clinical interview, multi-informant ratings, and reliability and validity checks with explicit data combining procedure), and 5 was worst (single reporter using non-standard rating scale). Whether a study controlled for moderators such as SES or family income was recorded and also used as a moderator.

***Inclusion Criteria*.** To be included in the meta-analysis, the sample had to be either a population survey or a sample selected as an ADHD case-control design in humans. A control group had to be included, and sufficient data had to be available for computing an effect size. Studies could include child, adolescent, or adult populations, and could be cross sectional, retrospective, or prospective. They had to provide an operational measure of the ADHD construct, defined as either 1) diagnosis of ADHD based on DSM criteria, including all editions from DSM-III to 5, or based on ICD criteria; or, 2) a measure of ADHD symptoms using a validated ADHD symptom rating scale (either the Conners scales, the ADHD Rating Scale, or an equivalent); or, 3) in the case of epidemiological studies, identification of ADHD using a diagnostic-specific question, typically, "Have you/has your child ever been told by your doctor/health care practitioner that you have ADHD?", or, by a chart review indicating a diagnosis or ADHD was made by the aforementioned methods. Study participants with psychiatric comorbidities (e.g. depression, anxiety, conduct disorder) were included. As this meta-analysis focuses on overweight and obesity status as the primary outcomes, studies had to include an operational measure of these constructs. Overweight and obesity are defined on the basis of cut-off scores for body mass index (BMI), a derived variable calculated on weight (kg)/height^2 (m^2). In adults, overweight is typically defined as a BMI > 25 < 30 kg/m^2 , and obesity is typically defined as a BMI ≥ 30 kg/m^2 (Must & Anderson, 2006). In children and

adolescents, criteria for overweight and obesity differ among studies. As such, definitions from 1) the International Obesity Task Force, and 2) the Centers for Disease Control and Prevention BMI-for-age reference (Ogden et al., 2002) were used. The most typical definition was gender-specific BMI-for-age percentiles with overweight defined as ≥ 85th percentile and obese as > 95th percentile (Must & Anderson, 2006). We also included studies in which BMIs are reported and an association between a BMI dimensional score with an ADHD measure is provided.

Exclusion Criteria. The following types of studies were excluded: 1) studies without a specific control group, such as those that compared a clinical or convenience sample to a population or reference norm; 2) studies that selected an overweight or obese sample based on either a metabolic disorder impacting weight (diabetes) or a diagnosis of disordered eating (e.g. bulimia, binge eating); 3) studies of pregnancy weight and offspring outcome, 4) studies that examined the severity of ADHD within an ADHD-only sample.

Effect Size Computation

Parameters used to compute effect sizes included means and standard deviation (SD) or standard error (SE); correlation coefficients; odds ratios (OR) or a regression coefficient with an N or 95% CI; P-value with an N. All were converted to either an OR or "g," depending on the analyses reported, using formulas supplied by Borenstein et al(2009) and implemented in the Comprehensive Meta-Analysis software (CMA). For reporting, all values are converted to the OR, the most common metric in this literature.

The identified studies offered varying numbers of analyses and effect sizes. These effect sizes varied in their outcome definition (BMI, overweight, obese, or combinations of these), covariate models, ages examined at baseline or outcome, and definitions of ADHD (self-reported diagnosis, research-verified diagnosis, single rating scale, multiple rating scales of "impulsivity" or "inattention), and whether they examined males and females together or separately or both. We handled this as in next paragraph.

Data Analysis

Because of the widely varied numbers of effect sizes provided in the studies, we employed a multi-step decomposition of the meta-analytic data. In the first step, we pooled all analyses within-study using the modified fixed-effect summarization available in the CMA software, to create a best-estimate aggregate effect without presupposition about the correct relevance of confounders or moderators. Next, we computed aggregate effects that removed potential confounders, notably stimulant medication use. For each effect size reported, a "yes," or "no" dichotomized variable was created denoting whether or not participants' use of stimulant medication was controlled (either by excluding or covarying). After that, we proceeded to examine effect modifiers (moderators) examining in turn: gender, age, and for two psychiatric comorbidities: conduct/disruptive behavior disorder (CD) or depression (DEP). Due to the small number of studies of children only or adolescents only, a dichotomized age variable was created as: child (<18 years) or adult (≥ 18 years). Considering the two psychiatric comorbidities, CD or DEP, if either were covaried or excluded, the variable for that effect size was coded "yes".

Moderator effects were tested in mixed models in which the between study type variation was quantified by the Q statistic (interpreted like chi-square in this context); ADHD quality assessment was also examined with meta-regression. Publication bias analyses were conducted in two ways, using Duval and Tweedie's Trim and Fill procedure (Duval & Tweedie, 2000a, 2000b), and Orwin's Fail-safe N (Orwin, 1983).

Meta-analysis: Results

The meta-analysis included 43 studies with 225 (non-orthogonal) extracted effect sizes, studying 703,937 total participants. **Table 5** shows the demographic and statistical details for the 43 studies included in the meta-analysis. The pooled effect suggests a composite effect size of OR=1.22 (95% CI [1.11-1.34]). Less than one-third of the studies found a statistically significant association between ADHD and overweight/obesity when effect sizes from studies were pooled within study. When one extreme outlier (Gungor, Celiloglu, Raif, Ozcan, & Selimoglu, 2013), OR=18.09) was excluded, the composite effect size was 1.20 (95% CI [1.08-

1.30]; k=42). **Figure 2** displays these results in a forest plot, with the outlier removed. However, this effect size is still very crude, as it pools all ages, genders, and effects, with and without various covariates. Thus, we proceeded to parse these results more carefully.

Table 6 summarizes the results of our moderator analyses in four areas: medication status, gender within the same studies, age without covariates, and age with comorbidities, namely conduct disorder and depression (CD/DEP). When the analyses are restricted to studies that controlled for stimulant medication use, the aggregate composite effect is OR=1.30 (95% CI [1.12-1.50]; k=22), providing perhaps the best estimate of risk in untreated individuals. In the eight studies that controlled for medication and covaried CD and/or DEP, the association between ADHD and obesity/overweight remains reliably non-zero with a similar effect size.

For gender, the comparison was restricted to studies that reported data for both males and females so that variation between study populations would not account for results. Pooling across age, the effect was qualitatively larger and statistically reliable in females, but not statistically reliable in males. However, the effect size for males and females was not statistically different from one another, Q-value = 0.82, p=0.37. In studies that controlled for medication and CD/DEP, the results were similar, as summarized in Table 6.

Next, studies are compared based on age stratification (pooling across gender). This comparison revealed that the association of ADHD with obesity or its proxies was larger in adults than non-adults (Q= 4.17, df=1, p=0.04). In studies that controlled for stimulant medication and covaried CD/DEP, the overall pattern held: the effect for adults was quantitatively larger for than children, although not statistically different because of the smaller number of studies. Nevertheless, the effects are qualitatively twice as large in adults as for children even with these controls as shown in Table 6. In adults there was no hint of a gender effect: Effects in adults were similar in males and females. For illustrative purposes, **Figure 3** depicts the qualitative age–related change in effect size across the three-category age variable: child, teen, and adult, showing a monotonic increase in effect size with each age group. Of note,

in pre-pubertal children, there is no reliable effect of ADHD on obesity/overweight.

Other data checks. After removing one extreme outlier (Gungor et al., 2013), quality of ADHD evaluation was rated by the senior author (JN) on a 1-5 scale and analyzed both in meta-regression and at two different cut points for "good" and "poor" ADHD evaluation; none of these comparisons identified a reliable effect of ADHD evaluation (all $p>.20$). SES is a critical confounder; 12 studies ignored SES (mostly non-US and non-European studies) with a pooled OR of 1.18; 30 controlled some SES proxy (income, parent education, or occupation), but in all cases SES was covaried along with various other covariates, never in isolation; they had a pooled OR of 1.23. While it is difficult to draw a conclusion about SES it is unlikely that SES explains the other findings reported.

Publication Bias. Using the trim and fill procedure, when studies were presumed missing to the left of the mean (small effects unpublished), 8 studies were trimmed, yielding a modified point estimate for all studies of 1.11 [95% CI=1.01-1.22], suggesting this literature may slightly over-estimate the population effect size. When studies were presumed missing to the right of the mean, 0 studies were trimmed. Using Orwin's Fail-safe N, 29 studies with effect of zero (OR=1.0) would be needed to drop the effect to a trivial OR=1.05.

General Discussion

Excess weight, overweight, and obesity are multi-determined, relating to socioeconomic status, mood, family structure, and other factors (Puder & Munsch, 2010). The idea that childhood ADHD might be a contributor to excess body weight would place a new importance on the need for ADHD intervention, as well as on understanding obesity. As societal activity and eating patterns continue to evolve, and rates of obesity rise, both among children and adults in the United States, and other Western countries, it remains crucial to determine whether individuals with ADHD are among those more vulnerable to the dynamics that cause obesity. If so, many mechanisms might require study, including mood regulation, impulsivity, lack of energy, shared biology vis-à-vis altered metabolism, or medication effects (Cortese & Morcillo

Penalver, 2010; Cortese & Vincenzi, 2012). Because such examination would be costly, it is important to evaluate the scope and generalizability of the putative ADHD-obesity association.

The literature has been mixed overall with regard to this association. Our child study (preliminary 1) suggests, in congruence with a very small number of other studies restricted to pre-adolescent children, that there is no discernible association of ADHD with obesity in the pre-adolescent years. The national survey data and the meta-analysis both tend to confirm this, but there remain very few studies confined to pre-adolescent children. Our new national survey study (preliminary 2) further suggested that part of the reason for this mixed picture is that the association of ADHD with overweight and obese status is larger in adolescence than in childhood, taking gender into account, is more reliable in girls than in boys during adolescence. However, when depression and conduct disorder were both covaried, the association between ADHD and obesity/overweight includes zero, suggesting that in adolescents, the association is accounted for by those with comorbid conditions. On the other hand, these may be the adolescents with more severe ADHD to start with, before comorbidity occurs, something the survey data do not enable to be examined.

The meta-analytic data clarify the picture further. Overall, the effect of ADHD with overweight and obese status, while reliably non-zero, is quite small. However, the size of the effect is moderated by age, with the association becoming more robust as one focuses on adult populations rather than children. Within the meta-analytic data, a qualitative monotonic association appeared, with the effect size increasing with age across childhood, adolescence, and adulthood. Future studies should continue to report data separately for males and females (in addition to pooling data) to enable future, more powerful meta-analytic comparison of males to females in terms of differential risk particularly during the adolescent period.

From the viewpoint of clinical implications, these results suggest the possibility of moving toward a more nuanced understanding of the association between ADHD and overweight or obese status, one in which risk is associated with particular sub-populations of ADHD, rather

than with ADHD globally. As the results indicate, for adults, and perhaps adolescent girls with comorbid disorders, the association of ADHD with BMI is larger than one would gather from overall averages, and may warrant greater clinical attention. On the other hand, in pre-adolescent boys, who represent the primary group diagnosed with ADHD, the risks are not likely to be clinically relevant, or to warrant aggressive intervention.

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FIGURE 1: PRISMA FLOW DIAGRAM OF STUDIES INCLUDED IN THE META-ANALYSIS

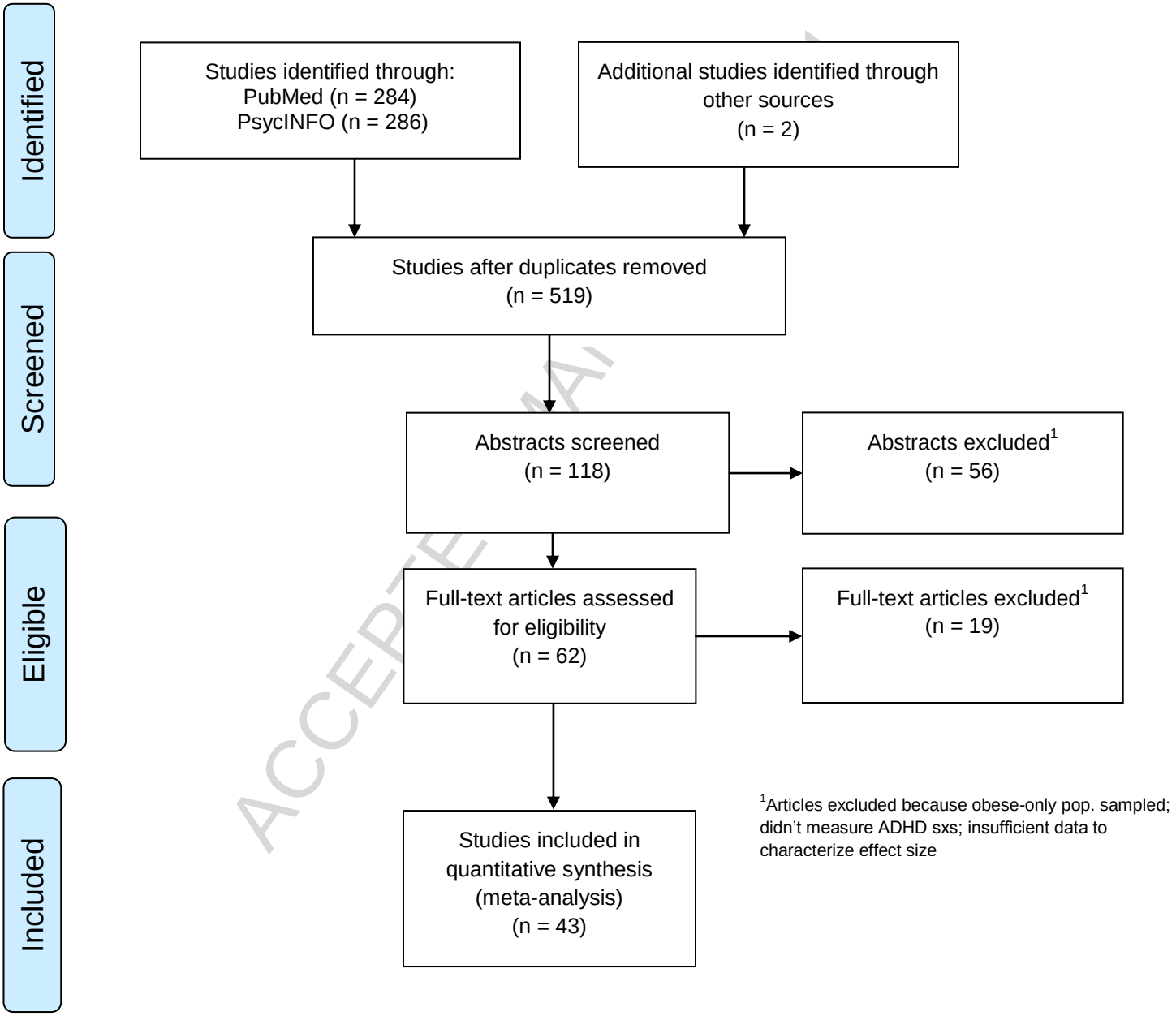
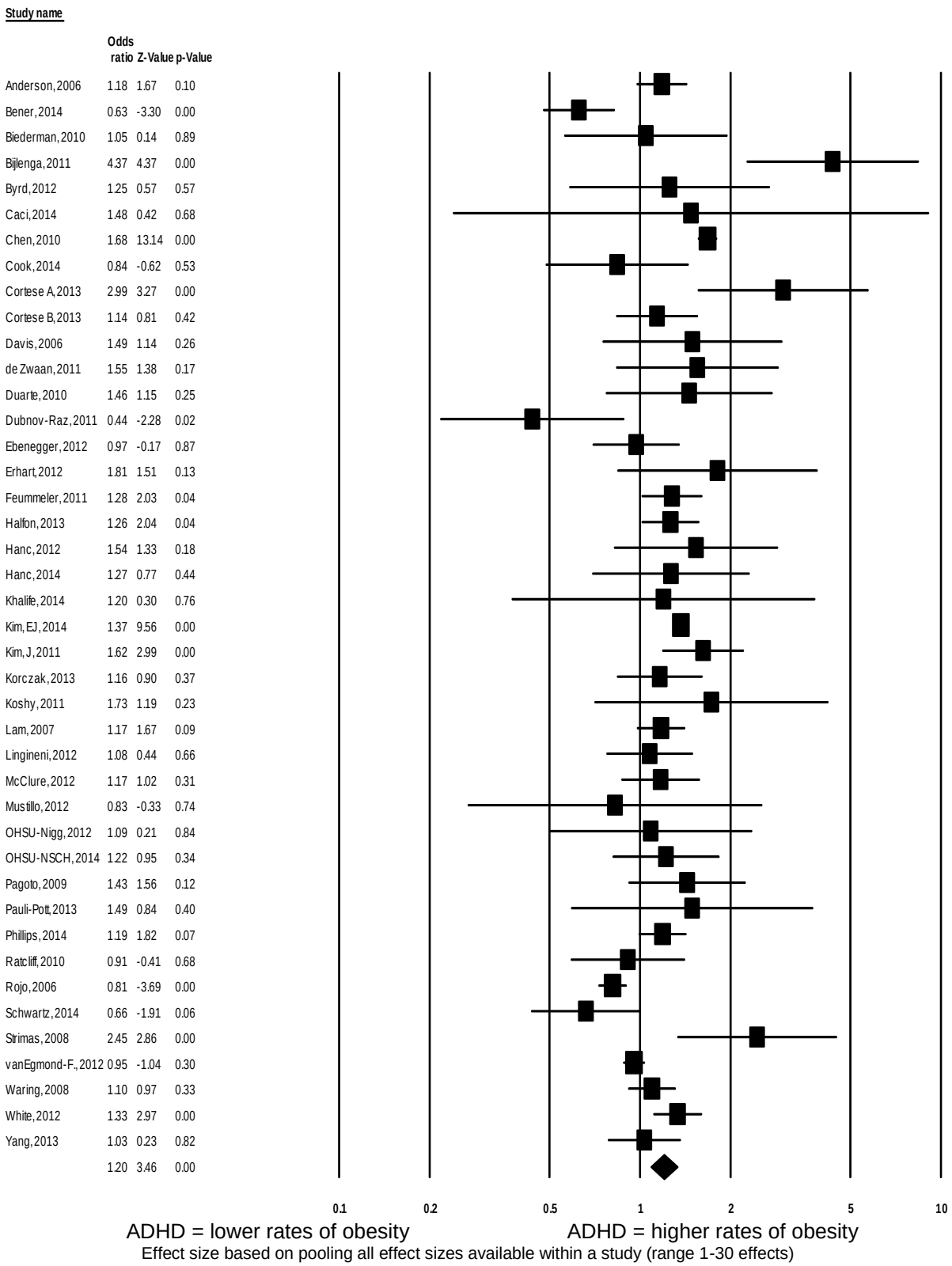
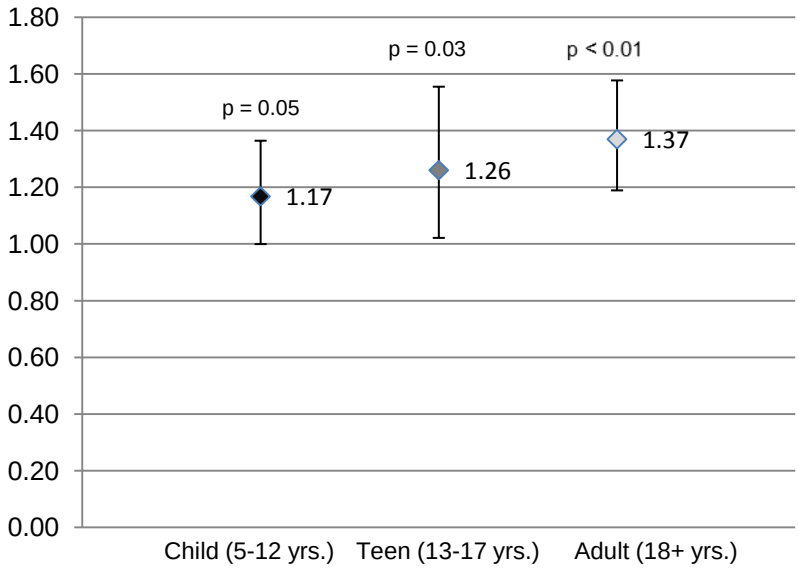


FIGURE 2: FOREST PLOT POOLED UNCORRECTED EFFECT SIZES EXCLUDING ONE OUTLIER



Effect size based on pooling all effect sizes available within a study (range 1-30 effects)

FIGURE 3: ODDS RATIOS FOR THE ASSOCIATION BETWEEN ADHD AND OBESITY STRATIFIED BY THREE AGE GROUPS



Note to Figure: Y Axis=Odds ratio. 1.0=no association; positive values indicate that ADHD is associated with more obesity or higher body mass index; negative values indicate that ADHD is associated with less obesity or lower body mass index.

Table 1: Sample description and group comparisons by wave for preliminary study 1

	Total			ADHD		Non-ADHD		Comparison
	N	Mean	SD	N	Mean	N	Mean	*t* (*df*)
Parental Education (Z score)	313	0.0	1.0	170	0.1	143	-0.2	-2.6 (311)**
Household Income (Z score)	313	0.0	1.0	170	-0.1	143	0.2	2.8 (311)**
Full Scale IQ	313	112	13.7	170	109.7	143	115.4	3.7 (311)***
ADHD-RS[a] (T-Scores)								
Inattention	309	59.6	16.8	166	72.2	143	45.0	-24.3 (307)***
Hyperactive/Impulsive	309	57.2	15.6	166	67.6	143	45.0	-18.4 (307)***
Total	309	59.0	16.6	166	71.5	143	44.6	-24.2 (307)***
Age (years)								
Wave 1	313	8.7	1.2	170	8.9	143	8.5	-3.0 (311)**
Wave 2	271	9.9	1.2	148	10.1	123	9.7	-2.9 (269)**
Wave 3	300	11.0	1.2	163	11.1	137	10.8	-2.4 (298)*
Body Mass Index								
Wave 1	313	16.2	2.8	170	16.2	143	16.1	-0.4 (311)
Wave 2	261	17.1	3.0	141	17.1	120	17.2	0.7 (259)
Wave 3	226	18.0	3.5	111	17.8	115	18.2	0.9 (224)
Pubertal Status								
Wave 1	62	1.6	0.4	48	1.7	14	1.5	-1.5 (60)
Wave 2	164	1.7	0.5	93	1.8	71	1.6	-2.4 (162)*
Wave 3	186	1.7	0.5	89	1.8	97	1.7	-1.4 (184)
	N	%		N	%	N	%	χ^2 (*df*)
Male	313	62	--	170	73	143	48	20.0 (1)***
Non-White	304	18	--	166	16	138	20	0.6 (1)
Stimulant Use[+]								
Wave 1	313	18	--	170	34	143	0	58.6 (1)***
Wave 2	268	25	--	145	46	123	0	74.3 (1)***
Wave 3	204	25	--	94	54	110	1	75.9 (1)***
Depression								
Wave 1	313	4	--	170	6	143	1	7.0 (1)**
Wave 2	271	4	--	148	5	123	2	1.0 (1)
Wave 3	228	4	--	113	6	115	1	4.8 (1)*

Note: * $p < .05$, ** $p < .01$, *** $p < .001$; Wave 1 – Wave 3 = 1st – 3rd wave of data collection; [a] Attention-Deficit/Hyperactivity Disorder Rating Scale, [+] use of stimulant medication ever for Wave 1 or since previous wave or assessment for Waves 2 & 3

Table 2: Synopsis of growth models for body mass index of sample in preliminary study 1

	Model				
	1	2	3	4	5
Random Effects	Variance (SE)	Variance (SE)	Variance(SE)	Variance(SE)	Variance (SE)
Intercept	3.91 (.41)***	4.06 (.44)***	4.07 (.44)***	3.86 (.41)***	2.88 (.59)***
Age (Linear)	0.50 (.15)***	0.33 (.06)***	0.33 (.06)***	0.32 (.06)***	0.28 (.11)**
Age^2 (Quadratic)	0.02 (.01)+	--	--	--	--
Residual	0.61 (.06)***	0.69 (.07)***	0.68 (.07)***	0.63 (.06)***	0.44 (.07)***
Fixed Effects	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)
Intercept	15.52 (.13)***	15.53 (.13)***	15.65 (.19)***	15.76 (.23)***	16.07 (.39)***
Age	0.90 (.08)***	0.86 (.05)***	0.96 (.07)***	0.97 (.07)***	0.84 (.11)***
Age^2	-0.01 (.02)	--	--	--	--
ADHD	**--**	**--**	**-0.21 (.26)**	**-0.11 (.27)**	**0.17 (.38)**
ADHD * Age	**--**	**--**	**-0.17 (.09)+**	**-0.11 (.09)**	**-0.23 (.15)**
Male	--	--	--	-0.13 (.27)	-0.71 (.34)*
Household Income	--	--	--	-0.29 (.13)*	-0.39 (.17)*
Parent Education	--	--	--	0.11 (.14)	0.06 (.18)
Depression	--	--	--	-0.05 (.23)	-0.01 (.25)
ODD/CD	--	--	--	0.01 (.17)	-0.09 (.23)
Stimulant Use[#]	--	--	--	-0.65 (.19)***	-0.94 (.29)**
Puberty	--	--	--	--	0.35 (.18)+
Model Fit					
N Observations	797	797	797	770	394
-2LL	3073.2	3085.6	3081.6	2945.4	1645.6
BIC	3130.7	3120.0	3127.5	3025.8	1728.8

Note: + $p < .10$,* $p < .05$, ** $p < .01$, *** $p < .001$; BIC = Bayesian information criterion (smaller is better). [#] use of stimulant ever for Wave 1 or since previous wave or assessment for Waves 2 & 3

Table 3. Preliminary study 2: Stratified Unadjusted Frequencies of ADHD and BMI status*

		BMI STATUS			
	Frequency	**Under**	**Normal**	**Over**	**Obese**
			Children (10-13)		
BOYS					
ADHD-NO	8,698	6.7%	54.5%	17.9%	20.9%
ADHD-YES (ever)	1,904	8.1%	52.3%	18.0%	21.5%
ADHD-Current	1,581	8.5%	54.0%	17.6%	19.9%
Mild	644	7.5%	56.3%	17.9%	18.2%
Moderate	688	8.4%	52.9%	18.2%	20.5%
Severe	225	12.0%	50.2%	15.1%	22.7%
Meds: No	371	7.5%	53.1%	19.1%	20.2%
Meds: Yes	1206	8.9%	54.3%	17.2%	19.7%
GIRLS					
ADHD-NO	9,099	6.6%	63.0%	16.0%	14.3%
ADHD-YES (ever)	888	6.5%	59.6%	17.3%	16.6%
ADHD-Current	738	6.9%	60.4%	16.7%	16.0%
Mild	347	6.3%	61.4%	15.3%	17.0%
Moderate	307	6.5%	60.3%	17.3%	16.0%
Severe	79	11.4%	57.0%	20.3%	11.4%
Meds: No	190	4.7%	60.5%	18.4%	16.3%
Meds: Yes	545	7.7%	60.4%	16.1%	15.8%
			Adolescents (14-17)		
BOYS					
ADHD-NO	9,852	5.1%	67.7%	13.8%	13.4%
ADHD-YES	2,416	5.7%	63.7%	15.0%	15.6%
ADHD-Current	1,743	6.0%	65.2%	14.5%	14.3%
Mild	860	5.8%	66.4%	14.5%	13.3%
Moderate	704	6.4%	65.2%	13.6%	14.8%
Severe	169	5.3%	59.8%	18.9%	16.0%
Meds: No	601	5.7%	63.9%	15.1%	15.3%
Meds: Yes	1,140	6.1%	66.0%	14.1%	13.8%
GIRLS					
ADHD-NO	9,938	4.3%	77.1%	11.0%	7.6%
ADHD-YES	1,001	3.5%	66.9%	15.4%	14.2%
ADHD-Current	727	4.0%	68.8%	14.0%	13.2%
Mild	361	4.2%	72.9%	12.2%	10.8%
Moderate	315	4.4%	66.0%	16.5%	13.0%
Severe	50	0.0%	56.0%	12.0%	32.0%
Meds: No	259	5.4%	66.0%	13.9%	14.7%
Meds: Yes	487	3.2%	70.4%	14.1%	12.2%

Notes to Table 3. *N's do not always add up exactly due to "don't know" or "refused".

Table 4: Preliminary Study 2. Stratified, weighted results of logistic regression models of risk of being overweight or obese as compared to normal weight in youth diagnosed with ADHD as defined by severity or currency

	ADHD Ever				Moderate and Severe ADHD				Current ADHD Without Medication			
	OR	95% CI		p	OR	95% CI		p	OR	95% CI		p
All Children (N= 43,796*)												
age and sex adj	1.17	1.03	- 1.34	0.02	1.21	0.98	- 1.49	0.07	1.29	0.99	- 1.69	0.06
10-13 years old (N= 20,589)												
sex adj	1.04	0.87	- 1.25	0.66	1.03	0.78	- 1.36	0.84	1.15	0.76	- 1.72	0.51
14-17 years old (N= 23,207)												
sex adj	1.32	1.09	- 1.60	0.004	1.50	1.11	- 2.03	0.01	1.45	1.02	- 2.06	0.04
BOYS ONLY												
10-13 years old (N=10,602)												
unadj	1.00	0.80	- 1.26	1.00	0.96	0.69	- 1.35	0.83	1.10	0.65	- 1.84	0.73
depression adj	0.99	0.78	- 1.25	0.94	0.96	0.67	- 1.38	0.83	1.08	0.64	- 1.83	0.78
depress + conduct	1.07	0.83	- 1.38	0.60	1.06	0.72	- 1.57	0.77	1.22	0.72	- 2.09	0.46
14-17 years old (N= 12,268)												
unadj	1.16	0.94	- 1.43	0.17	1.13	0.83	- 1.55	0.44	1.13	0.76	- 1.68	0.56
depression adj	1.14	0.92	- 1.41	0.25	1.08	0.79	- 1.49	0.62	1.13	0.77	- 1.67	0.53
depress + conduct	1.09	0.87	- 1.36	0.46	1.03	0.72	- 1.47	0.88	1.11	0.72	- 1.74	0.64
GIRLS ONLY												
10-13 years old (N=9,987)												
unadj	1.14	0.85	- 1.53	0.37	1.23	0.78	- 1.95	0.38	1.30	0.76	- 2.22	0.35
depression adj	0.95	0.71	- 1.28	0.75	0.89	0.56	- 1.41	0.62	1.00	0.54	- 1.84	1.00
depress + conduct	0.91	0.67	- 1.23	0.53	0.85	0.54	- 1.35	0.50	1.03	0.56	- 1.88	0.93
14-17 years old (N= 10,939)												
unadj	1.73	1.21	- 2.47	0.003	2.51	1.42	- 4.43	0.002	2.28	1.25	- 4.15	0.007
depression adj	1.48	1.01	2.19	0.05	2.03	1.11	3.74	0.02	2.01	1.05	3.84	0.04
depress + conduct	1.36	0.89	2.06	0.16	1.75	0.87	3.50	0.12	1.72	0.86	3.44	0.13

* All children 10-17 years N= 45,309; BMI data missing for N=1.445; ADHD data missing N=68

Table 5: Studies included in the meta-analysis of ADHD and overweight/obesity alphabetized by last name of first author

	1st author[@]	Year	Nation	Ages	Sample	Design	N ADHD	N ctrl	N total	OR[e]	LL	UL	Z-value	P
1	Anderson	2006	USA	9-38	com	LGT	94	561	655	1.18	1.03	1.36	2.36	0.02
2	Bener	2014	QAT	5-18	com	CRX	1,331	1,331	2,662	0.63	0.47	0.83	-3.30	0.00
3	Biederman	2010	USA	6-27	com	LGT	124	137	261	1.02	0.66	1.59	0.10	0.92
4	Bijlenga	2011	NRL	18-65	clin	CRX	202	189	391	4.37	2.25	8.47	4.37	0.00
5	Byrd	2012	USA	8-15	com	CRX	412	2,638	3,050	1.32	0.78	2.24	1.04	0.30
6	Caci	2012	FRA	20-65	com	CRX	132	1,039	1,137	1.55	0.56	4.24	0.85	0.40
7	Chen	2010	USA	10-17	com	CRX	4,848	41,859	46,707	1.68	1.55	1.81	13.14	0.00
8	Cook	2014	USA	10-17	com	CRX	1,606	44,291	45,897	0.84	0.48	1.46	-0.62	0.53
9	CortesеA	2013	USA	8-41	com	LGT	111	111	222	2.99	1.55	5.77	3.27	0.00
10	CortesеB	2013	USA	18-65+	com	CRX	616	34,037	34,653	1.15	0.97	1.37	1.63	0.10
11	Davis	2006	CAN	25-46	com	CRX	na	na	110	1.49	0.75	2.98	1.14	0.26
12	de Zwaan	2011	GER	18-64	com	CRX	77	1,556	1,633	1.55	0.83	2.90	1.38	0.17
13	Duarte	2010	FIN	8-23	com	LGT	156	2,053	2,209	1.46	0.77	2.77	1.15	0.25
14	Dubnov-Raz	2011	ISR	6-16	clin	CRX	275	51	326	0.44	0.22	0.89	-2.28	0.02
15	Ebenegger	2012	SWI	4-6	com	CRX	na	na	450	0.97	0.69	1.36	-0.17	0.87
16	Erhart	2012	GER	7-17	com	CRX	102	2,303	2,405	1.98	1.35	2.92	3.46	0.00
17	Fuemmeler	2011	USA	13-29	pop	LGT	901	10,753	11,654	1.28	1.01	1.62	2.03	0.04
18	Güngor	2013	TUR	5-15	clin	CRX	362	390	752	18.09	3.94	83.05	3.72	0.00
19	Halfon	2012	USA	10-17	pop	CRX	3,880	39,226	43,106	1.26	1.01	1.58	2.04	0.04
20	Hanc	2012	POL	6-18	clin	CRX	135	0	135	1.54	0.82	2.89	1.33	0.18
21	Hanc	2015	POL	6-18	clin	CRX	219	396	615	1.27	0.69	2.32	0.77	0.44
22	Khalife	2014	FIN	7-16	pop	LGT	987	5,947	6,934	1.20	0.37	3.84	0.30	0.76
23	Kim	2014	KOR	5-13	com	CRX	939	11,411	12,350	1.37	1.28	1.46	9.56	0.00
24	Kim	2011	USA	6-17	pop	CRX	6,070	60,573	66,643	1.52	1.25	1.84	4.21	0.00

Table 5: Studies included in the meta-analysis (continued)

	1st author	Year	Nation	Ages	Sample	Design	N ADHD	N ctrl	N total	OR[Θ]	LL	UL	Z-value	P
25	Korczak	2013	CAN	4-32	com	LGT	1,565	na	1,565	1.17	0.97	1.40	1.66	0.10
26	Koshy	2011	UK	5-11	com	CRX	32	913	945	1.73	0.70	4.24	1.19	0.23
27	Lam	2007	CHI	13-17	pop	CRX	na	na	1,429	1.17	0.97	1.42	1.67	0.09
28	Lingineni	2012	USA	5-17	pop	CRX	7,137	61,378	68,515	1.08	0.77	1.51	0.44	0.66
29	McClure	2012	USA	6-26	com	LGT	na	na	655	1.17	0.86	1.59	1.02	0.31
30	Mustillo	2012	USA	9-16	com	LGT	44	947	991	0.83	0.27	2.56	-0.33	0.74
31	Nigg*-OHSU	2012	USA	7-13	com	LGT	170	143	313	1.07	0.71	1.59	0.32	0.75
32	Nigg*-NSCH	2014	USA	10-17	pop	CRX	6,209	37,587	43,796	1.21	0.99	1.48	1.84	0.07
33	Pagoto	2009	USA	18-44	pop	CRX	492	6,245	6,737	1.43	0.91	2.25	1.56	0.12
34	Pauli-Pott	2013	GER	6-12	clin	CRX	207	153	360	1.49	0.59	3.78	0.84	0.40
35	Phillips	2014	USA	12-17	pop	CRX	845	8,141	8,986	1.19	0.99	1.43	1.82	0.07
36	Ratcliff	2010	USA	18-22	com	CRX	16	248	264	0.91	0.59	1.42	-0.41	0.68
37	Rojo	2006	SPN	13-15	com	CRX	7,571	27,832	35,403	0.81	0.76	0.86	-6.57	0.00
38	Schwartz	2014	USA	3-18	pop	LGT	13,789	150,031	163,820	0.66	0.43	1.01	-1.91	0.06
39	Strimas	2008	USA	25-50	com	CRX	na	na	145	2.45	1.33	4.53	2.86	0.00
40	van Egmond	2012	GER	6-17	com	CRX	na	na	10,218	1.00	0.97	1.03	0.03	0.98
41	Waring	2008	USA	5-17	pop	CRX	5,680	57,204	62,884	1.10	0.91	1.32	0.97	0.33
42	White	2012	UK	5-34	com	LGT	2,175	9,621	11,796	1.33	1.10	1.61	2.97	0.00
43	Yang	2013	CHI	6-16	clin	CRX	158	0	158	1.03	0.78	1.37	0.23	0.82
	Pooled Effect						**69,669**	**621,295**	**703,937**	**1.22**	**1.11**	**1.34**	**4.06**	**0.00**

[Θ]Each study effect size in the table reflects the pooling of all effect sizes reported in that paper (number of effect sizes per study ranged from 1 to 30, not shown), which were pooled within study using a modified fixed effects model. The pooled effect across studies shown in the last row of the table is based on a random effects model. Nation = research location: USA=United States, QAT=Qatar, NRL=Netherlands, FRA=France, CAN=Canada, GER=Germany, FIN=Finland,

ISR=Israel, SWI=Switzerland, TUR=Turkey, POL=Poland, KOR=Korea, UK=United Kingdom, CHI=China; com= community, clin=clinical, pop=population, LGT=longitudinal, CRX=cross sectional, "Ages" represents age range within study and for longitudinal designs, "Ages" represents age at entry and last analyses.

*Nigg studies (#31 and #32) refer to new data presented herein; OHSU = Oregon Health & Science University; NSCH = National Survey of Children's Health

(continued on next page)

@ Studies are listed here to correspond to first author names and year: (Anderson, Cohen, Naumova, & Must, 2006); (Bener & Kamal, 2014) ; (Biederman, Spencer, Monuteaux, & Faraone, 2010) ; (Bijlenga et al., 2013) ; (Byrd, Curtin, & Anderson, 2013) ; (Caci, Morin, & Tran, 2014) ; (Chen et al., 2010) ; (Cook, Li, & Heinrich, 2014) ; (Cortese, Ramos Olazagasti, et al., 2013) ; (Cortese, Faraone, et al., 2013) ; (Davis et al., 2006) ; (de Zwaan et al., 2011) ; (Duarte et al., 2010) ; (Dubnov-Raz, Perry, & Berger, 2011) ; (Ebenegger et al., 2012) ; (Erhart et al., 2012) ; (Fuemmeler et al., 2011) ; (Gungor et al., 2013) ; (Halfon et al., 2013) ; (Hanc, Cieslik, Wolanczyk, & Gajdzik, 2012) ; (Hanc et al., 2015) ; (Khalife et al., 2014) ; (Kim et al., 2014) ; (Kim et al., 2011) ; (Korczak et al., 2013) ; (Koshy, Delpisheh, & Brabin, 2011) ; (Lam & Yang, 2007) ; (Lingineni et al., 2012) ; (McClure, Eddy, Kjellstrand, Snodgrass, & Martinez, 2012) ; (Mustillo et al., 2003) ; (Pagoto et al., 2009) ; (Pauli-Pott, Neidhard, Heinzel-Gutenbrunner, & Becker, 2014) ; (Phillips et al., 2014) ; (Ratcliff, 2010) ; (Rojo, Ruiz, Dominguez, Calaf, & Livianos, 2006) ; (Schwartz et al., 2014) ; (Strimas et al., 2008) ; (A. W. van Egmond-Fröhlich et al., 2012) ; (Waring & Lapane, 2008) ; (White, Nicholls, Christie, Cole, & Viner, 2012) ; (Yang, Mao, Zhang, Li, & Zhao, 2013)

Table 6: Summary of meta-analytic moderator comparisons

Overall	k	OR	LL	UL	Z	P
Overall effect from Table 5	43	1.22	1.11	1.34	4.06	<0.001
Medication[a]						
Meds not controlled	29	1.10	0.97	1.23	1.52	0.127
Meds controlled	22	1.30	1.12	1.50	3.41	0.001
Meds controlled, CD/DEP covaried	8	1.40	1.12	1.74	2.99	0.003
Gender within the same studies						
Females, all ages, no covariates[1]	12	1.19	1.01	1.41	2.03	0.044
Females, meds controlled[2]	9	1.24	1.03	1.48	2.26	0.024
Females, CD/Dep covaried[3]	5	1.14	1.01	1.29	2.08	0.037
Females, meds controlled, CD/Dep covaried[4]	4	1.14	1.00	1.31	1.93	0.054
Males, all ages, no covariates[1]	12	1.10	0.95	1.23	1.20	0.219
Males, meds controlled[2]	9	1.11	0.98	1.27	1.62	0.105
Males, CD/Dep covaried[3]	5	1.13	1.00	1.28	1.94	0.052
Males, meds controlled, CD/Dep covaried[4]	4	1.13	0.99	1.29	1.85	0.065
Age without covariates[b]						
Children (<18yo), no covariates or controls	31	1.13	1.00	1.27	1.94	0.052
Adults (≥18yo), no covariates or controls	17	1.37	1.19	1.58	4.36	<0.001
Age with covariates						
Children (<18yo), meds controlled[5]	15	1.20	1.00	1.44	1.99	0.047
Children, CD/Dep covaried[6]	10	1.18	1.03	1.35	2.38	0.017
Children, meds controlled, CD/Dep covaried[7]	3	1.28	1.07	1.54	2.64	0.008
Adults (≥18yo), meds controlled[5]	9	1.48	1.15	1.90	3.03	0.002
Adults, CD/Dep covaried[6]	11	1.35	1.11	1.64	3.06	0.002
Adults, meds controlled, CD/Dep covaried[7]	6	1.52	1.11	2.10	2.59	0.010
Adults within the same studies by gender with covariates						
Adult females, meds controlled[8]	4	1.19	1.02	1.39	2.27	0.023
Adult females, CD/Dep covaried[9]	3	1.13	0.99	1.29	1.87	0.062
Adult females, meds controlled, CD/Dep covaried[10]	2	1.13	0.98	1.30	1.69	0.091
Adult males, meds controlled[8]	4	1.15	0.98	1.34	1.74	0.081
Adult males, CD/Dep covaried[9]	3	1.14	1.00	1.31	1.96	0.050
Adult males, meds controlled, CD/Dep covaried[10]	2	1.14	0.99	1.32	1.72	0.086

NOTES TO TABLE

All moderator effects are non-significant at p>.10 unless noted by superscript

[a]=comparison of medication to non-medication controlled, p<.10

[b]=comparison of children to adults, no covariates, p=.05

[1-4]= females compared to males in same studies with stated covariates

[5-7]=children compared to adults with stated covariates

[8-10]= adult females compared to males in same studies with covariates

For a list of studies used in each comparison, contact authors.

Highlights

- ADHD and obesity may be associated but effect moderators are unclear.
- A meta-analysis of 43 studies was conducted.
- A reliable overall ADHD-to-obesity association was found with a small effect size.
- The effect was larger in adults over 18 years old than in children.
- This association may be of minimal clinical impact in children but more in adults.