HHS Public Access

Author manuscript

J Pediatr. Author manuscript; available in PMC 2020 May 01.

Published in final edited form as:

J Pediatr. 2020 May ; 220: 227–236.e1. doi:10.1016/j.jpeds.2020.01.016.

Parental Weight Status and Offspring Behavioral Problems and Psychiatric Symptoms

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Abstract

Objectives: To assess relations of pre-pregnancy maternal and paternal obesity with offspring behavioral problems and psychiatric symptoms at 7–8 years in the Upstate KIDS study, a prospective cohort study.

Study design: Maternal body mass index (BMI) was calculated from pre-pregnancy height and weight provided in vital records or self-report at 4 months post-partum. Mothers reported paternal height and weight. At 7–8 years, mothers indicated if their children had been diagnosed with ADHD or anxiety (n=1915). Additionally, children's behavior was measured with the Strengths and Difficulties Questionnaire (SDQ) at 7 years (n=1386) and the Vanderbilt ADHD Diagnostic Parent Rating Scale (VADPRS) at 8 years (n=1484). Based on SDQ scores, we identified children with borderline behavioral problems. Adjusted risk ratios (aRR) and 95% confidence intervals (CIs) were estimated with robust multivariable Poisson regression.

Results: Compared with children of mothers with BMI<25, children whose mothers had BMI 25–30, 30–35 and ≥35 kg/m^2 had higher risks of reported ADHD (aRRs, 95% CIs: 1.14, 0.78–1.69; 1.96, 1.29–2.98; and 1.82, 1.21–2.74, respectively). Risks of hyperactivity problems identified by the SDQ and a positive screen for inattentive or hyperactive/impulsive behavior with the VADPRS were also higher with increasing maternal pre-pregnancy BMI. Paternal BMI was not associated with child outcomes.

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Portions of this study were presented at the Society for Pediatric and Perinatal Epidemiologic Research Annual Meeting, << >>, 2019, << >>.

Conclusions: Our findings suggest that maternal, rather than paternal, obesity is associated with maternal report of child ADHD diagnosis and inattentive or hyperactivity problems. Further research is needed to understand how maternal obesity might influence these behavioral changes during or after pregnancy.

Keywords

pre-pregnancy; maternal obesity; paternal obesity; attention-deficit/hyperactivity disorder

In the United States (U.S.), 37% of women and 32% of men of reproductive age are obese (body mass index [BMI] ≥30 kg/m^2).[1] As the prevalence of obesity has risen in the past 15 years,[1] so has prevalence of child psychiatric disorders such as attention-deficit/hyperactivity disorder (ADHD).[2]

A meta-analysis of prospective epidemiologic studies provides evidence of an association between maternal pre-pregnancy BMI and behavioral problems and psychiatric symptoms in childhood.[3-16]. The association between maternal overweight (BMI ≥25 and <30) or overweight/obese (BMI ≥25) status and behavior is less certain.[14–18] Similarly, most longitudinal studies observed a positive association between maternal obesity and ADHD or inattention/hyperactivity problems,[12–14, 19–25] and a few show null results.[26, 27] Potential mechanisms include chronic inflammation in the intrauterine environment, increased concentrations of circulating metabolic hormones, or epigenetic modifications associated with obesity.[28]

Paternal obesity may also relate to child behavioral problems or psychiatric symptoms as evidence from animal studies has revealed mechanisms for non-mendelian epigenetic inheritance.[29] Previous studies investigated the associations of paternal obesity with child behavioral problems and psychiatric symptoms; two did not find an association between paternal obesity and child behavioral problems[12, 18], one observed a positive, but imprecise, association with ADHD symptoms,[26] and, in the largest study, a positive association between paternal obesity and offspring behavioral problems was observed.[15] An association with maternal but not paternal obesity would support the hypothesis that alterations in the intrauterine environment, rather than preconception or postnatal factors shared between parents (e.g., diet)are implicated in the development of offspring behavioral problems and psychiatric symptoms. As such, investigation of associations among fathers also serve as a "negative control" for mothers.[30] Population-based research in the U.S., where the prevalence of obesity is higher than that of European countries,[1, 31] has not examined the relation between paternal BMI and child behavioral problems and psychiatric symptoms.

In our cohort, maternal and paternal obesity have been associated with higher risk of failing a developmental screening in early childhood.[32] It is unclear if the associations between parental obesity and neurodevelopmental outcomes detected in early childhood extend into behavioral problems during middle childhood. Thus, we investigated the associations of maternal pre-pregnancy obesity and paternal obesity at 4 months post-partum with child behavioral problems and psychiatric symptoms at 7–8 years old in the Upstate KIDS cohort. We hypothesized that both maternal and paternal obesity would be positively associated with offspring behavioral problems and psychiatric symptoms.

METHODS

The Upstate KIDS study is a population-based birth cohort originally established to evaluate the effects of infertility treatment on childhood growth and development.[33] Approximately 4 months postpartum in New York State (excluding New York City), mothers of children born between 2008 and 2010 were recruited into the study with the use of birth certificates from a livebirth registry. Infants conceived by infertility treatments and multiple births were oversampled. Three singletons not conceived by infertility treatment were sampled for every child conceived using fertility treatment, frequency matched to region of birth. In total, 5,034 mothers and 6,171 children were recruited. No associations between fertility treatment status and early childhood development were observed.[34] Singletons and twins with information on behavioral problems and psychiatric symptoms in middle childhood are included in this analysis. Due to low numbers, triplets and quadruplets were excluded from follow-up through middle childhood (n=134).

The New York State Department of Health and the University of Albany Institutional Review Board (IRB) approved of the study and served as the IRB designated by the National Institutes of Health under a reliance agreement. Parents provided written informed consent prior to enrollment.

Measurements

Upon enrollment at 4 months post-partum, mothers completed a baseline questionnaire on their sociodemographic information and familial health status. We obtained information from vital records on the child's sex and plurality, as well as maternal pre-pregnancy height and weight.

Exposure information.—Maternal BMI was calculated as kg/m^2 from pre-pregnancy height and weight provided in vital records or, if missing from vital records, from self-report on the baseline questionnaire which inquired about maternal "weight before this pregnancy." Paternal BMI was calculated from height and weight reported by mothers at 4 months post-partum on the baseline questionnaire. We categorized BMI as underweight or normal weight (<25.0), overweight (25.0–29.9), obese class I (30.0–34.9) and obese classes II+ (≥35) based on the World Health Organization cutoffs. A separate category for underweight was not included due to low numbers of underweight mothers (n=45) and fathers (n=16) in our sample.

Covariate information.—Covariates were collected at 4 months post-partum either from the baseline questionnaire or vital records. Maternal age and insurance type, paternal age, and child sex were obtained from vital records. Maternal race/ethnicity was from maternal report in the baseline questionnaire, with vital records used when questionnaire data were unavailable. Maternal education, marital status, history of polycystic ovary syndrome (PCOS), and smoking and alcohol use during pregnancy, and maternal and paternal history of affective disorders were reported by mothers in the baseline questionnaire at 4 months post-partum.

Follow-up study.—When children were 7 and 8 years old, subgroups of mothers completed annual questionnaires on their children's development. Mothers reported if their child had ever been diagnosed with attention-deficit disorder (ADD)/ADHD or anxiety disorder in a clinical setting, and if the child had been prescribed medications for ADD/ADHD or anxiety disorder in the past year. In addition, mothers rated their child's behavior with the Strengths and Difficulties Questionnaire (SDQ)[35] at 7 years and a modified version of the Vanderbilt ADHD Diagnostic Parent Rating Scale (VADPRS)[36] at 8 years.

The SDQ is a validated and reliable questionnaire[35] which has been used previously in U.S. samples.[37] The use of the SDQ in this population has been described previously.[38] In brief, the SDQ is designed to measure children's behavior in the five domains of emotional symptoms, peer relationship problems, conduct problems, hyperactivity/inattention, and prosocial behaviors. The SDQ consists of 25 statements (five per domain) that mothers rate as not true (0 points), somewhat true (1 point), or certainly true (2 points). Scores for each question pertaining to one domain were summed. Although we had originally considered examining the SDQ subscales as continuous variables, the distributions of these variables were ordinal with an excess of zero values. We therefore sought to identify children with borderline behavioral problems, using cutoff points previously utilized in the U.S. (emotional symptoms ≥3, peer relationship problems ≥2, conduct problems ≥2, hyperactivity/inattention ≥5, and prosocial behaviors ≤6).[39]

When the children were approximately 8 years old, mothers completed a modified version of the VADPRS, which screens for children's ADHD and other disorders. We incorporated screening by the VADPRS in our measurement of ADHD to include children with problem behaviors who may not have received a formal diagnosis. This instrument is a validated and reliable questionnaire that has been used previously in the U.S.[36, 40, 41] The VADPRS consists of 45 statements on children's behaviors, 9 of which correspond to DSM-IV behaviors for the predominately inattentive subtype of ADHD and 9 to the hyperactive/impulsive subtype. Due to rarity and age appropriateness we dropped 3 items on the conduct disorder scale. This did not affect our assessment of ADHD and symptoms of conduct disorder were not considered in our analysis. Mothers indicated how frequently in the past six months their child has exhibited each behavior as "never," "occasionally," "often," or "very often." The questionnaire also contains eight statements on the children's school and interpersonal performance which mothers rate as "excellent," "above average," "average," "somewhat of a problem," or "problematic." The VADPRS was scored using the National Institute for Children's Health Quality's criteria.[42] To meet criteria for screening, children must have been rated as "somewhat of a problem" or "problematic" on any of the 8 performance items and "often" or "very often" on 6 items for the subtype scales. A child must screen positive for both inattentive and hyperactive/impulsive subtype scales to screen positive for ADHD. The prevalence of a positive screen for ADHD by this VADPRS scoring criteria (2.7%) was too low to include as a primary endpoint by itself and hence we combined that with maternal report. We also examined associations without limiting only to children with both inattentive and hyperactivity/impulsive subtype screens or performance items impacted (see specifics below).

Statistical analyses

Children with information on behavioral problems and psychiatric symptoms at 7 or 8 years are included in the analysis. Exposures of interest are maternal and paternal BMI. We examined BMI categorically and continuously.

At 7 years, mothers of 1386 children completed the SDQ and, at 8 years, mothers of 1484 children responded to the VADPRS. At 7 or 8 years, 1915 mothers reported on their child's diagnosis or medication status. Three methods to define child behavioral problems and psychiatric symptoms were examined as outcome measures. First, positive histories of ADHD or anxiety disorder were considered using maternal diagnostic reports. A positive history was defined as having a maternal report of clinical diagnosis or maternal report of medication use for that condition. Maternal report of child depression was not an endpoint due to low prevalence of the condition at this age (1.15%). Second, we incorporated the VADPRS to assess screening for ADHD in several ways. We considered either positive ADHD screening (with performance items impacted) or maternal report of clinical ADHD diagnosis. Positive screening for the inattentive OR hyperactive/impulsive subtype scales was considered separately. We also classified children as screening for inattentive OR hyperactive/impulsive subtype scales without including the performance items in these screenings (ie, children had to score only "often" or "very often" on 6 of the 9 subtype scale items). Lastly, parental report of child borderline behavioral problems measured with the SDQ were examined.

We first compared the distributions of mother's and father's BMI across categories of parental and child characteristics within a primary cohort of singletons and a randomly selected twin from each twin pair. We used the Wald χ^2 test to calculate *P*-values for dichotomous variables. For ordinal variables, we calculated a *P*-trend. In multivariable analysis, we estimated adjusted risk ratios (aRR) and 95% confidence intervals (CI) with the use of a modified, robust Poisson regression among all singletons and twins.[43] For all models, adjustment variables included maternal race/ethnicity, education, insurance status, smoking, alcohol intake, marital status, polycystic ovary syndrome (PCOS) diagnosis; parental history of affective disorders; and child's sex. For maternal weight status, we additionally adjusted for maternal age, parental age difference, and paternal BMI. For paternal weight status, we additionally adjusted for maternal pre-pregnancy BMI and paternal age. PCOS and parental history of affective disorders were added sequentially as there is evidence of a bidirectional association between BMI and PCOS symptoms or affective disorder. Generalized estimating equations with robust standard errors were used to account for the correlation among twins.

Among the 1915 children with outcome data at either assessment, maternal and paternal BMI was missing from 1 (0.05%) and 174 (9.09%) parent(s), respectively. The percentage of information missing on covariates ranged from 0% for child sex to 5.43% for paternal age. Thus, missing covariate and exposure information was imputed using the Markov chain Monte Carlo multiple imputation with 50 imputed datasets. In addition, we used inverse probability weighting (IPW) to account for non-response to the follow-up questionnaires. Weights were generated from a multivariable logistic regression model in which the outcome was responding to the SDQ (n=1386) or to questions pertaining to maternal report of clinical

diagnosis or screening (n=1915). To generate weights, covariates in the logistic regression model were maternal age, race, education, insurance status, marital status, smoking and drinking during pregnancy, BMI, and gynecological conditions; paternal age and BMI; parental history of affective disorder; and child sex, plurality, conception using fertility treatment, and parity. In supplemental analysis, we compared the prevalence of the diagnostic and SDQ endpoints across categories of baseline covariates among the primary cohort.

All analyses were conducted with SAS version 9.4 (SAS Institute Inc).

RESULTS

Prevalence of maternal pre-pregnancy obesity (BMI ≥30) and paternal obesity at 4 months post-partum were 24.73 and 29.97%, respectively. Maternal pre-pregnancy BMI was inversely associated with socioeconomic status markers and alcohol use during pregnancy, and positively related to maternal history of affective disorders. Paternal BMI at 4 months post-partum was positively related to maternal and paternal age, non-Hispanic white ethnicity, and being married (Table I). In general, behavioral problems were inversely associated with maternal age and socioeconomic status markers, and positively related to maternal history of affective disorders, diagnosis of PCOS, and smoking during pregnancy (Table 2 and Table 3; available at www.jpeds.com).

Maternal weight status

Maternal reported diagnosis.—Maternal pre-pregnancy obesity was associated with higher risk of maternal report of clinical diagnosis of ADHD (Table 4). After adjustment for covariates, maternal obesity class I was associated with 1.96 times the risk of maternal report of child clinical ADHD diagnosis (95% CI 1.29, 2.98) compared with children of mothers with BMI <25. Estimates were similar for obesity class II+ (aRR 1.82; 95% CI: 1.21, 2.74). Maternal obesity class I or class II were not related to maternal report of child clinical anxiety diagnosis after adjustment for covariates.

VADPRS.—Associations of pre-pregnancy maternal BMI with a positive screen for maternal reported ADHD diagnosis or ADHD screen by the VADPRS were comparable with those of maternal report of clinical diagnosis for ADHD (aRRs obesity class I, class II+: 1.77; 95% CI 1.18, 2.66 and 1.78; 95% CI 1.21, 2.62, respectively) (Table 4). The associations between maternal obesity class I and II and a positive screen on either the inattentive or hyperactive/impulsive subscales were attenuated and not precise (aRRs obesity class I, class II+: 1.42; 95% CI 0.75, 2.70 and 1.26; 95% CI 0.65, 2.42, respectively). Maternal obesity was positively related to the VADPRS inattentive or hyperactive/impulsive screen when the performance items were not included (aRRs obesity class I, class II+: 1.51; 95% CI 0.88, 2.58 and 1.68; 95% CI 1.02, 2.77).

SDQ.—Maternal obesity was additionally related to borderline hyperactivity/inattention determined by the SDQ (Table 4). After covariate adjustment, maternal obesity class I and class II+ were associated with 1.84 (95% CI 1.24, 2.73) and 1.79 (95% CI 1.21, 2.65) times the risk of child borderline hyperactivity/inattention, respectively, compared with children of

mothers with BMI <25. Maternal obesity was not related to other child psychopathology outcomes.

Paternal weight status

Paternal obesity was not related to any behavioral problems or psychiatric symptom outcome in unadjusted or adjusted analyses (Table 5).

Models without adjustment for maternal PCOS or parental history of affective disorder had similar results (data not shown).

DISCUSSSION

In this prospective birth cohort in New York State, maternal pre-pregnancy BMI was related to subsequent child behavioral problems and psychiatric symptoms at ages 7 to 8, whereas paternal BMI was not associated with these outcomes. Children of women with pre-pregnancy obesity (BMI ≥30) had approximately twice the risk of maternal report of clinical diagnosis of ADHD, compared with children of women with BMI <25. We did not find an association between maternal pre-pregnancy weight status and offspring anxiety.

Maternal obesity was related to child ADHD and hyperactivity/inattention problems assessed by maternal report of clinical diagnosis, screening with the VADPRS and maternal report of diagnosis, screening for ADHD symptoms by the VADPRS, and the SDQ. The magnitude of effect was comparable across each of these measures (aRRs, obesity class I: 1.51–1.96; aRRs, obesity class II+: 1.68–1.86). The associations were attenuated when performance indicators were included in ADHD symptom screening by the VADPRS; it is possible that children who are receiving treatment for ADHD would be misclassified in this screen. Previous longitudinal cohort studies have observed similar positive associations between maternal pre-pregnancy obesity and offspring ADHD diagnosis.[5, 19, 20, 24, 27] However, associations of maternal pre-pregnancy BMI and parental report of offspring ADHD symptoms by the SDQ or Child Behavior Checklist have been mixed.[10–15, 18, 21, 25, 26] In early childhood (aged 3–5 years), pre-pregnancy obesity class I has been associated with child ADHD symptoms in some studies,[10–12] but not in others.[14, 25, 26] Only one of these studies was able to examine obesity class II+ (BMI ≥35) as an exposure and found a positive association with child ADHD symptoms at age 3 years.[25] In middle childhood (aged 6–11 years), after children had more time in school and are likely to receive a formal diagnosis for difficulties, the results are more consistent; maternal pre-pregnancy obesity has been positively associated with parental report of child ADHD symptoms in all[13, 15, 21, 24] but one study.[5] The disparity in these results may reflect parents' difficulty in distinguishing abnormal and normal levels of hyperactivity in early childhood.

Several mechanisms have been proposed to explain the associations between maternal pre-pregnancy BMI and child behavioral problems or psychiatric symptoms. In rodent models, an obesogenic state induced by a maternal high fat diet results in structural and functional brain changes in the offspring which include altered dopaminergic response,[45] increased corticosteroid receptor expression,[46] and dendritic atrophy.[47] Rodent pups also exhibited more hyperactive and anxiety-like behavior.[45, 46] During pregnancy, women with obesity

have higher concentrations of circulating inflammatory markers, compared with women with BMI <25.[48, 49] This inflammatory state could affect placental development and alter the intrauterine environment; for example, accumulation of activated macrophages is higher in term placental cells of obese women compared with women with BMI <25.[48] These changes may in turn mediate adverse fetal development. Alternatively, pre-pregnancy obesity and other markers of inflammation relate to small variation in cord blood DNA methylation levels, which may have long-term impact on offspring.[50, 51] Non-causal explanations may also explain our findings; for example, our results may be confounded by parental ADHD status or biased due to self-report of height and weight.

In contrast to our results with pre-pregnancy maternal BMI and offspring ADHD, we did not observe an association with pre-pregnancy maternal BMI and offspring anxiety or the SDQ internalizing problem subscales of emotional symptoms and peer relationship problems. Results from previous studies have been mixed, with some finding a positive association with internalizing symptoms (eg, anxiety, depression, peer problems, somatization)[5, 11, 13–16, 24] and others finding no association with these traits.[4, 7, 9–11, 18] The associations we observed with obesity class II and maternal report of anxiety diagnosis or SDQ internalizing problem subscales were consistently positive (aRRs 1.29–1.38) but of smaller magnitude than those observed for maternal pre-pregnancy obesity and ADHD. It is possible that we did not have the power to detect associations of this magnitude.

We also found no associations between paternal BMI and offspring behavioral problems or psychiatric symptoms. These null results may indicate that contributions from the maternal intrauterine environment could influence the development of child behavioral problems and psychiatric symptoms rather than epigenetic mechanisms or shared pre- or post-conception lifestyle habits to which the father would also contribute. Results from birth cohorts with approximately 500–2500 children in England, the Netherlands, and Greece have found similar null associations between paternal BMI and offspring behavioral problems and psychiatric symptoms at 3–5 years.[12, 18] In contrast, a positive association between paternal obesity and child inattention (adjusted incidence rate ratio [aIRR]: 2.12, 95% CI 0.73–6.17) and hyperactivity symptoms (aIRR: 1.38, 95% CI 0.96–1.99) was observed at five years in the Infancia y Medio Ambiente (INMA) birth cohort in Spain (n=1827).[26] Despite a higher prevalence of paternal obesity than the INMA cohort and the ability to examine obesity class II as a separate exposure, we did not find a positive association with child inattentive or hyperactive/impulsive behavior. One potential reason for this discrepancy is that in our cohort the VADPRS was assessed by mothers whereas in INMA this assessment was done by teachers. Parent and teacher agreement on this rating scale is low.[52] In a larger study (n=38,314), the Danish National Birth Cohort (DNBC), paternal obesity was positively related to child emotional symptoms, peer relationship problems, and hyperactivity/inattention at 7 years as measured by the SDQ (adjusted odds ratios: 1.27, 1.72, and 1.37, respectively).[15] Although we also measured child behavior in middle childhood, we defined borderline problem behaviors based on a U.S. reference[39] whereas, with the larger sample size, DNBC was able to use cutoffs reflecting abnormal problems.[53] Thus, the cutoffs for DNBC were generally 1–2 points higher and sex-specific compared with the U.S. based cutoffs.

Our study has several strengths. We were able to adjust for many potential confounders of the relations between parental weight status and child behavior. Further, we examined the associations of maternal and paternal weight status with child behavioral problems and psychiatric symptoms. Previous studies which looked at paternal BMI as an exposure have been in Europe, where the prevalence of obesity is lower,[1, 31] and thus may not be generalizable to the U.S. There are limitations as well. Shared genetic risk factors may relate to both child obesity and ADHD,[54] and thus genetic contributions may be an unmeasured confounder. However, we would expect a similar magnitude of effect across mothers and fathers if the association between parental obesity and child ADHD was driven primarily by genetics. Reporting errors may be present in parental height and weight. This measurement error is likely nondifferential with respect to the outcome and could bias the results towards the null. Paternal weight status was calculated from measures of maternal report of their partner's height and weight at four months post-partum. If paternal weight status had changed from the pre-pregnancy period to four months post-partum then this measure would not reflect paternal weight status at the time period of interest (i.e., conception). Further, we do not have information on how confident mothers were in their assessment of paternal height and weight and therefore cannot assess the accuracy of this report; however, previously reports on agreement between maternal report and paternal self-report of height and weight in a European sample was high.[55] There is likely measurement error in our measurement of child behavioral problems and psychiatric symptoms as well. We are unable to quantify the extent of this measurement error in our sample and therefore cannot assess its impact on our results; however, for ADHD, parental report of the clinician diagnosis appears reliable and valid.[56–58] We may have missed cases who were taking medications prior to 7 or 8 years old whose symptoms had resolved. In addition, non-response to follow-up questionnaires were large. To address this issue, we used IPW to account for non-response to follow-up.[59] Lastly, our population is primarily Caucasian, which may limit the generalizability of our results.

In conclusion, in this examination of maternal and paternal obesity with child behavioral problems and psychiatric symptoms in the U.S., maternal obesity was related to maternal report of child ADHD diagnoses whereas paternal obesity was not associated with these outcomes. This familial information can assist in identifying high risk populations to screen for behavioral problems in childhood and provide earlier intervention. Given the consistent association of maternal obesity with child behavioral problems and psychiatric symptoms across studies in the U.S. and Europe, providers and public health practitioners should consider evidenced-based approaches for improving health literacy and lifestyle changes related to maternal prenatal weight for women considering pregnancy. Further research is needed in understanding whether lifestyle changes during pregnancy and/or any postnatal interventions can ameliorate risks.

ACKNOWLEDGEMENTS

We thank the Upstate KIDS participants and staff for their important contributions.

Supported by the Intramural Research Program of the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD; contracts #HHSN275201200005C, #HHSN267200700019C, #HHSN275201400013C, #HHSN275201300026I/27500004). The authors declare no conflicts of interest.

Abbreviations:

ADD	attention-deficit disorder
ADHD	attention-deficit/hyperactivity disorder
aRR	adjusted risk ratio
BMI	body mass index
CI	confidence intervals
DNBC	Danish National Birth Cohort
INMA	Infancia y Medio Ambiente
IPW	inverse probability weighting
IRB	Institutional Review Board
PCOS	polycystic ovary syndrome
SDQ	Strengths and Difficulties Questionnaire
U.S.	United States
VADPRS	Vanderbilt ADHD Diagnostic Parent Rating Scale

REFERENCES

[1]. Flegal KM, Kruszon-Moran D, Carroll MD, Fryar CD, Ogden CL. Trends in obesity among adults in the United States, 2005 to 2014. JAMA. 2016;315:2284–91. [PubMed: 27272580]

[2]. Visser SN, Danielson ML, Bitsko RH, Holbrook JR, Kogan MD, Ghandour RM, et al. Trends in the parent-report of health care provider-diagnosed and medicated attention-deficit/hyperactivity disorder: United States, 2003–2011. J Am Acad Child Adolesc Psychiatry. 2014;53:34–46 e2. [PubMed: 24342384]

[3]. Sanchez CE, Barry C, Sabhlok A, Russell K, Majors A, Kollins SH, et al. Maternal pre-pregnancy obesity and child neurodevelopmental outcomes: a meta-analysis. Obes Rev. 2018;19:464–84. [PubMed: 29164765]

[4]. Deardorff J, Smith LH, Petito L, Kim H, Abrams BF. Maternal prepregnancy weight and children's behavioral and emotional outcomes. Am J Prev Med. 2017;53:432–40. [PubMed: 28712831]

[5]. Jo H, Schieve LA, Sharma AJ, Hinkle SN, Li R, Lind JN. Maternal prepregnancy body mass index and child psychosocial development at 6 years of age. Pediatrics. 2015;135:e1198–209. [PubMed: 25917989]

[6]. Minatoya M, Itoh S, Araki A, Tamura N, Yamazaki K, Nishihara S, et al. Associated factors of behavioural problems in children at preschool age: the Hokkaido study on environment and children's health. Child Care Health Dev. 2017;43:385–92. [PubMed: 27868227]

[7]. Van Lieshout RJ, Schmidt LA, Robinson M, Niccols A, Boyle MH. Maternal pre-pregnancy body mass index and offspring temperament and behavior at 1 and 2 years of age. Child Psychiatry Hum Dev. 2013;44:382–90. [PubMed: 22983494]

[8]. Van Lieshout RJ, Robinson M, Boyle MH. Maternal pre-pregnancy body mass index and internalizing and externalizing problems in offspring. Can J Psychiatry. 2013;58:151–9. [PubMed: 23461886]

[9]. Tanda R, Salsberry PJ. Racial differences in teh association between maternal prepregnancy obesity and children's behavior problems. J Dev Behav Pediatr. 2014;35:118–27. [PubMed: 24509056]

[10]. Mina TH, Lahti M, Drake AJ, Raikkonen K, Minnis H, Denison FC, et al. Prenatal exposure to very severe maternal obesity is associated with adverse neuropsychiatric outcomes in children. Psychol Med. 2017;47:353–62. [PubMed: 27776561]

[11]. Menting MD, van de Beek C, de Rooij SR, Painter RC, Vrijkotte TGM, Roseboom TJ. The association between pre-pregnancy overweight/obesity and offspring's behavioral problems and executive functioning. Early Hum Dev. 2018;122:32–41. [PubMed: 29864728]

[12]. Daraki V, Roumeliotaki T, Koutra K, Georgiou V, Kampouri M, Kyriklaki A, et al. Effect of parental obesity and gestational diabetes on child neuropsychological and behavioral development at 4 years of age: the Rhea mother-child cohort, Crete, Greece. Eur Child Adolesc Psychiatry. 2017;26:703–14. [PubMed: 28050707]

[13]. Pugh SJ, Hutcheon JA, Richardson GA, Brooks MM, Himes KP, Day NL, et al. Gestational weight gain, prepregnancy body mass index and offspring attention-deficit hyperactivity disorder symptoms and behaviour at age 10. BJOG. 2016;123:2094–103. [PubMed: 26996156]

[14]. Rodriguez A Maternal pre-pregnancy obesity and risk for inattention and negative emotionality in children. J Child Psychol Psychiatry. 2010;51:134–43. [PubMed: 19674195]

[15]. Mikkelsen SH, Hohwu L, Olsen J, Bech BH, Liew Z, Obel C. Parental body mass index and behavioral problems in their offspring: A Danish national birth cohort study. Am J Epidemiol. 2017;186:593–602. [PubMed: 28535165]

[16]. Robinson M, Zubrick SR, Pennell CE, Van Lieshout RJ, Jacoby P, Beilin LJ, et al. Pre-pregnancy maternal overweight and obesity increase the risk for affective disorders in offspring. J Dev Orig Health Dis. 2013;4:42–8. [PubMed: 25080181]

[17]. Antoniou EE, Fowler T, Reed K, Southwood TR, McCleery JP, Zeegers MP. Maternal pre-pregnancy weight and externalising behaviour problems in preschool children: a UK-based twin study. BMJ Open. 2014;4:e005974.

[18]. Brion MJ, Zeegers M, Jaddoe V, Verhulst F, Tiemeier H, Lawlor DA, et al. Intrauterine effects of maternal prepregnancy overweight on child cognition and behavior in 2 cohorts. Pediatrics. 2011;127:e202–11. [PubMed: 21187310]

[19]. Andersen CH, Thomsen PH, Nohr EA, Lemcke S. Maternal body mass index before pregnancy as a risk factor for ADHD and autism in children. Eur Child Adolesc Psychiatry. 2018;27:139–48. [PubMed: 28712019]

[20]. Chen Q, Sjolander A, Langstrom N, Rodriguez A, Serlachius E, D'Onofrio BM, et al. Maternal pre-pregnancy body mass index and offspring attention deficit hyperactivity disorder: a population-based cohort study using a sibling-comparison design. Int J Epidemiol. 2014;43:83–90. [PubMed: 24058000]

[21]. Rodriguez A, Miettunen J, Henriksen TB, Olsen J, Obel C, Taanila A, et al. Maternal adiposity prior to pregnancy is associated with ADHD symptoms in offspring: evidence from three prospective pregnancy cohorts. Int J Obes (Lond). 2008;32:550–7. [PubMed: 17938639]

[22]. van der Burg JW, Jensen ET, van de Bor M, Joseph RM, O'Shea TM, Kuban K, et al. Maternal obesity and attention-related symptoms in the preterm offspring. Early Hum Dev. 2017;115:9–15. [PubMed: 28822870]

[23]. van Mil NH, Steegers-Theunissen RP, Motazedi E, Jansen PW, Jaddoe VW, Steegers EA, et al. Low and high birth weight and the risk of child attention problems. J Pediatr. 2015;166:862–9 e1–3. [PubMed: 25681197]

[24]. Kong L, Norstedt G, Schalling M, Gissler M, Lavebratt C. The risk of offspring psychiatric disorders in the setting of maternal obesity and diabetes. Pediatrics. 2018;142:e20180776. [PubMed: 30093539]

[25]. Fuemmeler BF, Zucker N, Sheng Y, Sanchez CE, Maguire R, Murphy SK, et al. Pre-Pregnancy Weight and Symptoms of Attention Deficit Hyperactivity Disorder and Executive Functioning Behaviors in Preschool Children. Int J Environ Res Public Health. 2019;16.

[26]. Casas M, Forns J, Martinez D, Guxens M, Fernandez-Somoano A, Ibarluzea J, et al. Maternal pre-pregnancy obesity and neuropsychological development in pre-school children: a prospective cohort study. Pediatr Res. 2017;82:596–606. [PubMed: 28493861]

[27]. Musser ED, Willoughby MT, Wright S, Sullivan EL, Stadler DD, Olson BF, et al. Maternal prepregnancy body mass index and offspring attention-deficit/hyperactivity disorder: a quasi-experimental sibling-comparison, population-based design. J Child Psychol Psychiatry. 2017;58:240–7. [PubMed: 27901266]

[28]. Rivera HM, Christiansen KJ, Sullivan EL. The role of maternal obesity in the risk of neuropsychiatric disorders. Front Neurosci. 2015;9:194. [PubMed: 26150767]

[29]. Zhou Y, Zhu H, Wu HY, Jin LY, Chen B, Pang HY, et al. Diet-Induced Paternal Obesity Impairs Cognitive Function in Offspring by Mediating Epigenetic Modifications in Spermatozoa. Obesity (Silver Spring). 2018;26:1749–57. [PubMed: 30358144]

[30]. Ness AR, Griffiths AE, Howe LD, Leary SD. Drawing causal inferences in epidemiologic studies of early life influences. Am J Clin Nutr. 2011;94:1959S–63S. [PubMed: 21562085]

[31]. Marques A, Peralta M, Naia A, Loureiro N, de Matos MG. Prevalence of adult overweight and obesity in 20 European countries, 2014. Eur J Public Health. 2018;28:295–300. [PubMed: 29036436]

[32]. Yeung EH, Sundaram R, Ghassabian A, Xie Y, Buck Louis G. Parental obesity and early childhood development. Pediatrics. 2017;139.

[33]. Buck Louis GM, Hediger ML, Bell EM, Kus CA, Sundaram R, McLain AC, et al. Methodology for establishing a population-based birth cohort focusing on couple fertility and children's development, the Upstate KIDS Study. Paediatr Perinat Epidemiol. 2014;28:191–202. [PubMed: 24665916]

[34]. Yeung EH, Sundaram R, Bell EM, Druschel C, Kus C, Ghassabian A, et al. Examining infertility treatment and early childhood development in the Upstate KIDS Study. JAMA Pediatr. 2016;170:251–8. [PubMed: 26746435]

[35]. Goodman R Psychometric properties of the Strengths and Difficulties Questionnaire. J Am Acad Child Adolesc Psychiatry. 2001;40:1337–45. [PubMed: 11699809]

[36]. Bard DE, Wolraich ML, Neas B, Doffing M, Laoma B. The psychometric properties of the Vanderbilt Attention-Deficit Hyperactivity Disorder Diagnostic Parent Rating Scale in a community population. J Dev Behav Pediatr. 2013;34:72–82 [PubMed: 23363972]

[37]. He JP, Burstein M, Schmitz A, Merikangas KR. The Strengths and Difficulties Questionnaire (SDQ): the factor structure and scale validation in U.S. adolescents. J Abnorm Child Psychol. 2013;41:583–95. [PubMed: 23183936]

[38]. Ghassabian A, Bell EM, Ma WL, Sundaram R, Kannan K, Buck Louis GM, et al. Concentrations of perfluoroalkyl substances and bisphenol A in newborn dried blood spots and the association with child behavior. Environ Pollut. 2018;243:1629–36. [PubMed: 30296759]

[39]. Scoring the Strengths & Difficulties Questionnaire for age 4–17 or 18+. http://www.sdqinfo.org/norms/USNorm.html:youthinmind; 2016.

[40]. Becker SP, Langberg JM, Vaughn AJ, Epstein JN. Clinical utility of the Vanderbilt ADHD diagnostic parent rating scale comorbidity screening scales. J Dev Behav Pediatr. 2012;33:221–8. [PubMed: 22343479]

[41]. Wolraich ML. Psychometric properties of the Vanderbilt ADHD Diagnostic Parent Rating Scale in a Referred Population. Journal of Pediatric Psychology. 2003;28:559–68. [PubMed: 14602846]

[42]. NICHQ Vanderbilt Assessment Scales: Used for diagnosing ADHD. National Institute for Children's Health Quality: American Academy of Pediatrics and National Initiative for Children's Healthcare Quality; 2002.

[43]. Zou G A modified poisson regression approach to prospective studies with binary data. Am J Epidemiol. 2004;159:702–6. [PubMed: 15033648]

[44]. Table 17, DSM-IV to DSM-5 Conduct Disorder Comparison Substance abuse and mental health services administration DSM-5 changes: implications for child serious emotional disturbance [Internet] Jun 2016 ed. Rockville (MD): Substance Abuse and Mental Health Services Administration (US); 2016.

[45]. Naef L, Moquin L, Dal Bo G, Giros B, Gratton A, Walker CD. Maternal high-fat intake alters presynaptic regulation of dopamine in the nucleus accumbens and increases motivation for fat rewards in the offspring. Neuroscience. 2011;176:225–36. [PubMed: 21187125]

[46]. Sasaki A, de Vega WC, St-Cyr S, Pan P, McGowan PO. Perinatal high fat diet alters glucocorticoid signaling and anxiety behavior in adulthood. Neuroscience. 2013;240:1–12. [PubMed: 23454542]

[47]. Janthakhin Y, Rincel M, Costa AM, Darnaudery M, Ferreira G. Maternal high-fat diet leads to hippocampal and amygdala dendritic remodeling in adult male offspring. Psychoneuroendocrinology. 2017;83:49–57 [PubMed: 28595087]

[48]. Challier JC, Basu S, Bintein T, Minium J, Hotmire K, Catalano PM, et al. Obesity in pregnancy stimulates macrophage accumulation and inflammation in the placenta. Placenta. 2008;29:274–81. [PubMed: 18262644]

[49]. Madan JC, Davis JM, Craig WY, Collins M, Allan W, Quinn R, et al. Maternal obesity and markers of inflammation in pregnancy. Cytokine. 2009;47:61–4. [PubMed: 19505831]

[50]. Yeung E, Guan W, Mumford SL, Silver RM, Zhang C, Tsai MY, et al. Measured maternal prepregnancy anthropometry and newborn DNA methylation. Epigenomics. 2019.

[51]. Sharp GC, Salas LA, Monnereau C, Allard C, Yousefi P, Everson TM, et al. Maternal BMI at the start of pregnancy and offspring epigenome-wide DNA methylation: findings from the pregnancy and childhood epigenetics (PACE) consortium. Hum Mol Genet. 2017;26:4067–85. [PubMed: 29016858]

[52]. Wolraich ML, Lambert EW, Bickman L, Simmons T, Doffing MA, Worley KA. Assessing the impact of parent and teacher agreement on diagnosing attention-deficit hyperactivity disorder. J Dev Behav Pediatr. 2004;25:41–7. [PubMed: 14767355]

[53]. SDQ: Normative SDQ data from Denmark. http://www.sdqinfo.org/norms/DanishNorms.html:youthinmind; 2013.

[54]. Chen Q, Kuja-Halkola R, Sjolander A, Serlachius E, Cortese S, Faraone SV, et al. Shared familial risk factors between attention-deficit/hyperactivity disorder and overweight/obesity - a population-based familial coaggregation study in Sweden. J Child Psychol Psychiatry. 2017;58:711–8. [PubMed: 28121008]

[55]. Suren P, Gunnes N, Roth C, Bresnahan M, Hornig M, Hirtz D, et al. Parental obesity and risk of autism spectrum disorder. Pediatrics. 2014;133:e1128–38. [PubMed: 24709932]

[56]. Visser SN, Danielson ML, Bitsko RH, Perou R, Blumberg SJ. Convergent validity of parent-reported attention-deficit/hyperactivity disorder diagnosis: a cross-study comparison. JAMA Pediatr. 2013;167:674–5. [PubMed: 23700143]

[57]. Bussing R, Mason DM, Leon CE, Sinha K. Agreement between CASA parent reports and provider records of children's ADHD services. J Behav Health Serv Res. 2003;30:462–9. [PubMed: 14593669]

[58]. Faraone SV, Biederman J, Milberger S. How reliable are maternal reports of their children's psychopathology? One-year recall of psychiatric diagnoses of ADHD children. J Am Acad Child Adolesc Psychiatry. 1995;34:1001–8. [PubMed: 7665438]

[59]. Howe CJ, Cole SR, Lau B, Napravnik S, Eron JJJ. Selection bias due to loss to follow up in cohort studies. Epidemiology. 2016;27:91–7. [PubMed: 26484424]

Table 1.

Maternal and paternal BMI across categories of maternal, paternal, and child characteristics among all singletons and one randomly selected twin of each pair.

	Maternal pre-pregnancy BMI, kg/m^2		Paternal BMI at 4 months post-partum, kg/m^2	
Characteristic	***n***	**Mean ± SD**	***n***	**Mean ± SD**
Total	1605	26.9 ± 7.0	1505	28.3 ± 5.5
Maternal characteristics				
Age, years				
<25	205	26.9 ± 7.1	175	26.3 ± 5.7
25–29	428	27.5 ± 7.7	398	28.6 ± 5.8
30–34	546	26.5 ± 6.7	498	28.5 ± 5.3
35–39	327	26.4 ± 6.3	304	28.6 ± 4.9
≥40	144	27.5 ± 6.9	130	28.4 ± 5.5
P-trend[1]		0.45		0.004
Race / ethnicity				
Non-Hispanic white	1411	27.0 ± 7.0	1303	28.4 ± 5.5
Other	239	26.4 ± 6.9	202	27.6 ± 5.5
p[2]		0.28		0.05
Education				
Less than high school	53	28.8 ± 10.0	35	26.6 ± 7.0
High school of GED equivalent	158	28.8 ± 8.4	138	28.2 ± 7.1
Some college	421	29.0 ± 7.9	390	29.0 ± 5.6
College	414	26.0 ± 6.2	383	28.0 ± 5.2
Advanced degree	604	25.3 ± 5.4	559	28.0 ± 4.9
P-trend		<0.0001		0.29
Private insurance				
Yes	1354	26.5 ± 6.4	1260	28.4 ± 5.3
No	296	28.4 ± 8.9	245	27.7 ± 6.1
P		<0.0001		0.06
Married or living as married				
Yes	1468	26.8 ± 6.9	1390	28.3 ± 5.4
No	139	28.4 ± 8.4	102	27.1 ± 6.5
P		0.01		0.02
History of affective disorder				
Yes	288	28.1 ± 7.5	267	28.1 ± 5.7
No	1337	26.6 ± 6.8	1238	28.3 ± 5.4
P		0.001		0.50
Diagnosis of polycystic ovary syndrome				
Yes	194	30.4 ± 8.2	183	30.4 ± 6.1
No	1427	26.4 ± 6.7	1319	28.0 ± 5.3
P		<0.0001		<0.0001
Smoking during pregnancy				

	Maternal pre-pregnancy BMI, kg/m^2		Paternal BMI at 4 months post-partum, kg/m^2	
Characteristic	***n***	**Mean ± SD**	***n***	**Mean ± SD**
Yes	162	28.1 ± 8.0	145	26.9 ± 5.8
No	1487	26.7 ± 6.8	1359	28.4 ± 5.4
P		0.02		0.002
Alcohol use during pregnancy				
Yes	209	25.1 ± 5.3	199	27.7 ± 4.9
No	1440	27.1 ± 7.2	1305	28.4 ± 5.5
P		<0.0001		0.10
Paternal characteristics				
Age, years				
<25	113	27.0 ± 7.1	104	26.4 ± 6.2
25–29	306	27.0 ± 7.2	286	28.4 ± 5.7
30–34	507	26.6 ± 6.6	474	28.3 ± 5.2
35–39	358	26.8 ± 6.8	334	29.0 ± 5.8
≥40	277	26.7 ± 6.8	257	28.3 ± 4.9
P-trend		0.60		0.02
History of an affective disorder				
Yes	127	27.5 ± 6.4	122	29.0 ± 5.8
No	1498	26.8 ± 7.0	1383	28.2 ± 5.4
P		0.32		0.11
Child characteristics				
Sex				
Male	876	27.0 ± 7.0	796	28.3 ± 5.6
Female	774	26.8 ± 6.9	709	28.2 ± 5.3
P		0.66		0.59

[1] *P*-trend from a linear regression model with BMI as the continuous outcome where a variable representing ordinal categories of the predictor was introduced as a continuous covariate.

[2] Wald chi-squared test.

Table 2,

online only. Participant characteristics according to maternal reported diagnosis of child ADHD or anxiety among all singletons and one randomly selected twin of each pair

Sociodemographic characteristics	All	Child mental health disorder	
		ADHD	Anxiety disorder
Overall, *n* (%)	1651 (100)	148 (100)	125 (100)
Maternal Characteristics			
Age, years, mean ± SD	31.27 ± 5.89	29.99 ± 6.86	30.9 ± 6.20
Non-Hispanic white, *n* (%)	1412 (85.52)	118 (79.73)	111 (88.80)
Education, *n* (%)			
Less than high school	53 (3.21)	9 (6.08)	7 (5.60)
High school of GED equivalent	158 (9.57)	28 (18.92)	13 (10.40)
Some college	421 (25.50)	48 (32.43)	35 (28.00)
College	414 (25.08)	23 (15.54)	21 (16.80)
Advanced degree	605 (36.64)	40 (27.03)	49 (39.20)
Private insurance, *n* (%)	1354 (82.01)	108 (72.97)	98 (78.40)
Married or living as married, *n* (%)	1468 (91.35)	120 (82.19)	108 (87.80)
History of affective disorder, *n* (%)	288 (17.72)	43 (29.25)	47 (37.90)
Diagnosis of polycystic ovary syndrome, *n* (%)	194(11.97)	22(15.07)	23 (18.70)
Smoking during pregnancy, *n* (%)	162 (9.82)	28 (18.92)	16 (12.80)
Alcohol use during pregnancy, *n* (%)	209 (12.67)	20 (13.51)	16 (12.80)
BMI, kg/m^2, mean ± SD	26.88 ± 6.98	29.16 ± 7.61	27.79 ± 6.71
Paternal characteristics			
Age, years, mean ± SD	33.62 ± 6.70	32.86 ± 7.57	33.65 ± 7.48
History of an affective disorder, *n* (%)	127 (7.82)	14 (9.52)	10 (8.06)
BMI, kg/m^2, mean ± SD	28.27 ± 5.46	29.07 ± 5.85	28.09 ± 5.37
Child characteristics			
Child sex, male, *n* (%)	876 (53.06)	110 (74.32)	72 (57.60)

Table 3,

online only. Participant characteristics according to maternal reported diagnosis of child Strengths and Difficulties Questionnaire (SDQ) borderline behavioral problem among all singletons and one randomly selected twin of each pair

Sociodemographic characteristics	All	Child borderline behavioral problems, SDQ				
		Emotional symptoms	Peer relationship problems	Conduct problems	Hyperactivity/ inattention	Prosocial behaviors
Overall, *n* (%)	1651 (100)	167 (100)	185 (100)	172 (100)	188 (100)	73 (100)
Maternal Characteristics						
Age, years, mean ± SD	31.27 ± 5.89	30.60 ± 6.45	30.14 ± 6.14	30.79 ± 6.60	30.58 ± 6.28	31.04 ± 6.56
Non-Hispanic white, *n* (%)	1412 (85.52)	145 (86.83)	145 (78.38)	137 (79.65)	161 (85.64)	60 (82.19)
Education, *n* (%)						
Less than high school	53 (3.21)	11 (6.59)	11 (5.95)	13 (7.56)	9 (4.79)	4 (5.48)
High school of GED equivalent	158 (9.57)	25 (14.97)	28 (15.14)	34 (19.77)	40 (21.28)	14 (19.18)
Some college	421 (25.50)	38 (22.75)	54 (29.19)	45 (26.16)	43 (22.87)	21 (28.77)
College	414 (25.08)	31 (18.56)	34 (18.38)	33 (19.19)	37 (19.68)	12 (16.44)
Advanced degree	605 (36.64)	62 (37.13)	58 (31.35)	47 (27.33)	59 (31.38)	22 (30.14)
Private insurance, *n* (%)	1354 (82.01)	122 (73.05)	131 (70.81)	116 (67.44)	138 (73.40)	49 (67.12)
Married or living as married, *n* (%)	1468 (91.35)	147 (89.09)	151 (83.89)	146 (85.88)	161 (86.56)	62 (88.57)
History of an affective disorder, *n* (%)	288 (17.72)	57 (34.34)	42 (23.08)	41 (23.84)	51 (27.27)	19 (26.03)
Diagnosis of polycystic ovary syndrome, *n* (%)	194(11.97)	31 (18.67)	25 (13.74)	21 (12.21)	29 (15.51)	12 (16.44)
Smoking during pregnancy, *n* (%)	162 (9.82)	16 (9.58)	29 (15.68)	28 (16.28)	30 (15.96)	15 (20.55)
Alcohol use during pregnancy, *n* (%)	209 (12.67)	17 (10.18)	24 (12.97)	20 (11.63)	31 (16.49)	8 (10.96)
BMI, kg/m^2, mean ± SD	26.88 ± 6.98	28.25 ± 7.48	27.55 ± 7.22	27.66 ± 7.65	28.13 ± 7.03	27.87 ± 8.11
Paternal characteristics						
Age, years, mean ± SD	33.62 ± 6.70	32.94 ± 6.93	32.99 ± 7.13	33.26 ± 7.58	32.91 ± 6.94	33.15 ± 7.59
History of an affective disorder, *n* (%)	127 (7.82)	24 (14.46)	19 (10.44)	15 (8.72)	23 (12.30)	6 (8.22)
BMI, kg/m^2, mean ± SD	28.27 ± 5.46	28.54 ± 6.23	27.75 ± 5.82	28.25 ± 5.99	28.06 ± 5.56	27.45 ± 6.26
Child characteristics						
Child sex, male, *n* (%)	876 (53.06)	77 (46.11)	115 (62.16)	106 (61.63)	135 (71.81)	52 (71.23)

Table 4.

Child behavioral problems and psychiatric symptoms according to maternal pre-pregnancy weight status, the Upstate KIDS study.

			Categories of maternal BMI[1], kg/m					
Behavioral outcome	***n***	***n*, cases**	**<25 Reference**	**≥25 and <30 (overweight) RR (95% CI)**	**≥30 and <35 (obese class I) RR (95% CI)**	**≥35 (obese class II+) RR (95% CI)**	***P*-trend[2]**	**BMI, per kg/m^2 RR (95% CI)**
Maternal reported diagnosis								
ADHD	1906	183						
Unadjusted[3]			1	1.18 (0.79, 1.76)	2.03 (1.34, 3.07)	2.50 (1.71,3.64)	<0.0001	1.04 (1.02, 1.05)
Adjusted[4]			1	1.14 (0.78, 1.69)	1.96 (1.29,2.98)	1.82 (1.21,2.74)	0.0005	1.02 (1.01, 1.04)
Anxiety	1909	150						
Unadjusted			1	1.31 (0.86,2.01)	1.80 (1.11,2.92)	1.82 (1.13,2.94)	0.0031	1.02 (1.00, 1.04)
Adjusted			1	1.28 (0.85, 1.91)	1.55 (0.95,2.54)	1.29 (0.77,2.15)	0.17	1.00 (0.98, 1.03)
VADPRS								
Maternal reported ADHD diagnosis or ADHD screen on VADPRS	1915	193						
Unadjusted			1	1.09 (0.74, 1.61)	1.81 (1.21,2.72)	2.30 (1.60,3.31)	<0.0001	1.04 (1.02, 1.05)
Adjusted			1	1.08 (0.74, 1.57)	1.77 (1.18,2.66)	1.78 (1.21,2.62)	0.0007	1.02 (1.01, 1.04)
Inattentive OR hyperactive/ impulsive screen with performance impact	1484	96						
Unadjusted			1	1.21 (0.73,2.02)	1.36 (0.71,2.60)	1.74 (0.92,3.28)	0.08	1.04 (1.01, 1.06)
Adjusted			1	1.10 (0.66, 1.84)	1.42 (0.75,2.70)	1.26 (0.65,2.42)	0.35	1.03 (1.00, 1.05)
Inattentive OR hyperactive/ impulsive screen with no performance indicators	1484	134						
Unadjusted			1	1.19 (0.76, 1.86)	1.46 (0.86,2.50)	2.18 (1.35,3.53)	0.002	1.04 (1.02, 1.06)
Adjusted			1	1.08 (0.70, 1.67)	1.51 (0.88,2.58)	1.68 (1.02,2.77)	0.03	1.03 (1.01, 1.05)
Borderline behavioral problems, SDQ	1386	193						
Emotional symptoms								
Unadjusted			1	0.94 (0.64, 1.37)	1.50 (0.97,2.32)	1.79 (1.22,2.64)	0.002	1.03 (1.01, 1.05)
Adjusted			1	0.92 (0.64, 1.32)	1.26 (0.84, 1.89)	1.30 (0.85, 1.98)	0.16	1.01 (0.99, 1.03)
Peer relationship problems	1386	211						
Unadjusted			1	1.03 (0.73, 1.45)	0.96 (0.60, 1.53)	1.69 (1.19,2.39)	0.02	1.02 (1.00, 1.04)

			Categories of maternal BMI[1], kg/m					
Behavioral outcome	***n***	***n*, cases**	**<25 Reference**	**≥25 and <30 (overweight) RR (95% CI)**	**≥30 and <35 (obese class I) RR (95% CI)**	**≥35 (obese class II+) RR (95% CI)**	***P*-trend[2]**	**BMI, per kg/m^2 RR (95% CI)**
Adjusted			1	0.94 (0.67, 1.32)	0.92 (0.58, 1.45)	1.38 (0.95,2.02)	0.20	1.01 (1.00, 1.03)
Conduct problems	1386	201						
Unadjusted				1.19 (0.85, 1.68)	1.31 (0.84,2.05)	l.74 (1.20, 2.5U)	0.004	1.02 (1.01, 1.04)
Adjusted			1	1.08 (0.78, 1.51)	1.10 (0.73, 1.66)	1.26 (0.86, 1.84)	0.25	1.01 (0.99, 1.03)
Hyperactivity/inattention	1386	214						
Unadjusted			1	1.29 (0.91, 1.82)	1.98 (1.35,2.89)	2.30 (1.63,3.26)	<0.0001	1.04 (1.02, 1.05)
Adjusted			1	1.29 (0.91, 1.83)	1.91 (1.31,2.80)	1.86 (1.28,2.70)	0.0001	1.03 (1.01, 1.05)
Prosocial behaviors	1386	86						
Unadjusted			1	0.85 (0.48, 1.51)	1.07 (0.46,2.49)	2.17 (1.25,3.75)	0.02	1.04 (1.01, 1.07)
Adjusted			1	0.68 (0.37, 1.27)	0.87 (0.41, 1.86)	1.55 (0.84,2.89)	0.24	1.03 (1.00, 1.06)

[1] ADHD, attention-deficit/hyperactivity disorder; BMI, body mass index; SDQ, Strengths and Difficulties Questionnaire; VADPRS, Vanderbilt ADHD Diagnostic Parent Rating Scale

[2] *P*-trend when a variable representing ordinal categories of the BMI was introduced as a continuous covariate.

[3] Values are risk ratios and their 95% confidence intervals from a robust Poisson regression model.

[4] Models are adjusted for mother's age, race/ethnicity, education, insurance status, smoking, alcohol intake, marital status, and PCOS diagnosis; parental age difference and BMI; parental history of affective disorders; and child's sex.

J Pediatr. Author manuscript; available in PMC 2020 May 01.

Table 5.

Child behavioral problems and psychiatric symptoms according to paternal weight status at 4 months post-partum, the Upstate KIDS study.

			Categories of paternal BMI[1], kg/m^2					
Behavioral outcome	***n***	***n*, cases**	**<25 Reference**	**≥25 and <30 (overweight) RR (95% CI)**	**≥30 and <35 (obese class I) RR (95% CI)**	**≥35 (obese class II+) RR (95% CI)**	***P*-trend[2]**	**BMI, per kg/m^2 RR (95% CI)**
Maternal reported diagnosis								
ADHD	1906	183						
Unadjusted[3]			1	0.79 (0.52, 1.19)	0.92 (0.58, 1.46)	1.34 (0.83,2.18)	0.37	1.02 (0.99, 1.05)
Adjusted[4]			1	1.05 (0.69, 1.61)	1.25 (0.76, 2.06)	1.49 (0.87, 2.55)	0.13	1.02 (0.99, 1.05)
Anxiety	1909	150						
Unadjusted			1	0.91 (0.57, 1.47)	0.99 (0.57, 1.71)	1.20 (0.65,2.24)	0.63	1.01 (0.98, 1.04)
Adjusted			1	1.13 (0.72, 1.78)	1.22 (0.70,2.1)	1.27 (0.66,2.43)	0.42	1.01 (0.98, 1.04)
VADPRS								
Maternal reported ADHD diagnosis or ADHD screen on VADPRS	1915	193						
Unadjusted			1	0.77 (0.52, 1.13)	0.86 (0.55, 1.34)	1.22 (0.76, 1.96)	0.63	l.Ol (0.98, 1.04)
Adjusted			1	0.99 (0.67, 1.48)	1.16 (0.72, 1.88)	1.34 (0.80,2.23)	0.24	l.Ol (0.99, 1.04)
Inattentive OR hyperactive/ impulsive screen with performance impact	1484	96						
Unadjusted			1	0.47 (0.27, 0.85)	0.69 (0.38, 1.25)	0.91 (0.45, 1.82)	0.63	0.99 (0.94, 1.04)
Adjusted			1	0.56 (0.33, 0.95)	0.77 (0.43, 1.38)	0.82 (0.42, 1.63)	0.65	0.99 (0.95, 1.03)
Inattentive OR hyperactive/ impulsive screen with no performance indicators	1484	134						
Unadjusted			1	0.69 (0.44, 1.09)	0.92 (0.54, 1.54)	0.81 (0.41, 1.58)	0.64	0.99 (0.96, 1.03)
Adjusted			1	0.80 (0.52, 1.25)	0.92 (0.57, 1.49)	0.66 (0.34, 1.29)	0.31	0.98 (0.95, 1.02)
Borderline behavioral problems, SDQ								
Emotional symptoms	1386	193						
Unadjusted			1	0.75 (0.50, 1.13)	0.93 (0.58, 1.48)	1.18 (0.70, 1.98)	0.60	1.02 (0.99, 1.05)
Adjusted			1	0.88 (0.61, 1.28)	0.95 (0.60, 1.50)	1.16 (0.69, 1.95)	0.68	1.01 (0.98, 1.04)
Peer relationship problems	1386	211	1					

			Categories of paternal BMI[1], kg/m^2					
Behavioral outcome	***n***	***n*, cases**	**<25 Reference**	**≥25 and <30 (overweight) RR (95% CI)**	**≥30 and <35 (obese class I) RR (95% CI)**	**≥35 (obese class II+) RR (95% CI)**	***P*-trend[2]**	**BMI, per kg/m^2 RR (95% CI)**
Unadjusted			1	0.69 (0.47, 1.00)	0.84 (0.55, 1.28)	0.82 (0.48, 1.41)	0.49	1.00 (0.97, 1.03)
Adjusted			1	0.83 (0.58, 1.18)	0.92 (0.60, 1.42)	0.85 (0.50, 1.46)	0.60	1.00 (0.97, 1.03)
Conduct problems	1386	201						
Unadjusted			1	0.66 (0.45, 0.97)	0.92 (0.61, 1.38)	0.97 (0.59, 1.59)	>0.99	1.01 (0.98, 1.04)
Adjusted			1	0.73 (0.52, 1.04)	1.02 (0.68, 1.53)	1.04 (0.64, 1.67)	0.74	1.01 (0.98, 1.04)
Hyperactivity/inattention	1386	214						
Unadjusted			1	0.70 (0.49, 1.00)	0.97 (0.65, 1.43)	1.16 (0.73, 1.85)	0.50	1.01 (0.99, 1.04)
Adjusted			1	0.79 (0.56, 1.12)	1.07 (0.72, 1.59)	1.05 (0.66, 1.66)	0.63	1.01 (0.98, 1.03)
Prosocial behaviors	1386	86						
Unadjusted			1	0.77 (0.43, 1.37)	0.78 (0.38, 1.61)	1.02 (0.42,2.48)	0.90	1.01 (0.96, 1.07)
Adjusted			1	0.79 (0.45, 1.40)	0.86 (0.42, 1.75)	0.90 (0.34, 2.38)	0.79	1.02 (0.97, 1.07)

[1] ADHD, attention-deficit/hyperactivity disorder; BMI, body mass index; SDQ, Strengths and Difficulties Questionnaire; VADPRS, Vanderbilt ADHD Diagnostic Parent Rating Scale

[2] *P*-trend when a variable representing ordinal categories of the BMI was introduced as a continuous covariate.

[3] Values are risk ratios and their 95% confidence intervals from a robust Poisson regression model.

[4] Models are adjusted for mother's race/ethnicity, education, insurance status, smoking, alcohol intake, marital status, BMI, and PCOS diagnosis; parental age; parental history of affective disorders; and child's sex.

J Pediatr. Author manuscript; available in PMC 2020 May 01.