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A Meta-Analysis of the Association Between Birth Weight and Attention Deficit Hyperactivity Disorder

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Attention-Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by a constellation of symptoms that includes developmentally inappropriate inattention, impulsivity, and hyperactivity, with symptom onset during childhood (American Psychiatric Association, 2013). ADHD is one of the most common neurodevelopmental disorders, with the prevalence estimated to be between 2.5 and 11 percent in adults and children (American Psychiatric Association, 2013; Fayyad et al., 2007; Visser et al., 2014; Willcutt, 2012). Individuals with ADHD typically experience impairments that affect work, school, and relationships (Able, Johnston, Adler, & Swindle, 2007; de Graaf et al., 2008; Hinshaw, 2002; Wehmeier, Schacht, & Barkley, 2010). Additionally, ADHD can be a stressor to families because children with ADHD tend to be more difficult to parent and are more likely to be injured (Hinshaw, 2002). Finally, ADHD is a societal burden -- the annual societal cost of ADHD was estimated to be $143 - $266 billion (Doshi et al., 2012). The large burden to individuals, families, and society has led to a burgeoning field of research regarding risk factors for ADHD.

Potential Mechanisms Linking Birth Weight and ADHD

Given the developmental nature of ADHD, much research has focused on early life risk factors that may result in adverse neurodevelopmental sequela and the manifestation of symptoms of ADHD, with birth weight emerging as one of the most robust early life risk factors for ADHD. The Developmental Origins of Health and Disease (DOHaD), an important theory related to such research, proposed that a developing fetus may undergo “fetal programming” (prenatal modifications to help the developing fetus adapt to perturbations in the prenatal environment), but such modifications may ultimately leave the fetus ill-equipped for the perinatal environment, resulting in chronic health conditions (Barker, 1998). Originally, DOHaD was advanced to link early development adversity with cardiovascular conditions, but research predicated on DOHaD has expanded to examine developmental origins of psychological symptoms and disorders.

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Genetic variation is one of the first factors affecting a developing fetus's ability to survive, both in-utero and after birth. Therefore, it has been proposed that a common genetic liability may account for the association between birth weight and ADHD. Additionally, twin studies investigating the genetic and environmental contributions to ADHD and birth weight have consistently demonstrated that both traits have a high heritability (70% and 50%, respectively; Derks, Hudziak, & Boomsma, 2009; Faraone et al., 2005; Lunde, Melve, Gjessing, Skjaerven, & Irgens, 2007; Nikolas & Burt, 2010). A study utilizing longitudinal data from the Netherlands Twin Registry examined the extent to which birth weight exerts a causal influence on attention problems by testing the association between birth weight and attention problems in monozygotic twin pairs, dizygotic twin pairs, and unrelated pairs (randomly selected unrelated individuals) discordant for birth weight (Groen-Blokhuis, Middeldorp, van Beijsterveldt, & Boomsma, 2011). Interestingly, the negative association between birth weight and attention problems was stronger in the twin with lower birth weight, and this finding was consistent across monozygotic, dizygotic, and unrelated pairs, indicating genetic factors are likely not driving the association between birth weight and ADHD. Similarly, a twin study utilizing population-level data from Sweden came to the same conclusion, as the associations between birth weight and ADHD symptom ratings were similar for monozygotic and dizygotic twins (Pettersson et al., 2015). Thus, a common genetic liability does not appear to explain the association between ADHD and birth weight, although future work should continue to examine potential mediation or moderation of the association by genetic variations.

Another potential mechanism linking birth weight and ADHD is catch-up growth, or the weight gained in the first years of life. Individuals born low birth weight often receive special nutritional formulas to help them reach normal weight within the first one to two years of life. Despite the positive effects conferred by the nutritional formulas, empirical work has found that rapid catch-up growth is associated with increased risk for diabetes and hypertension and lower IQ (Estourgie-van Burk et al., 2009; Huxley, Shiell, & Law, 2000; Ong, 2007). To investigate this further, Groen-Blokhuis et al. (2011) examined the effect of catch-up growth in twin pairs, as well as unrelated pairs, and found that catch-up growth was not associated with attention problems. However, additional research is required to replicate this finding.

Additionally, a recent review (Smith, Schmidt-Kastner, McGeary, Kaczorowski, & Knopik, 2016) postulated an etiological pathway linking birth weight and ADHD via prenatal ischemia-hypoxia (IH), an insufficient supply of blood and oxygen in utero. As reviewed in the article, prenatal IH is associated with lower birth weight (Henriksen & Clausen, 2002; Kinzler & Vintzileos, 2008) and an increased risk for ADHD (Getahun et al., 2013; Owens & Hinshaw, 2013). Further, prenatal IH is thought to result in epigenetic changes and altered gene expression in the brain as a means of promoting tissue survival (Watson, Watson, McCann, & Baugh, 2010; Wu, Sun, & Li, 2013), but these epigenetic changes may also confer risk for neurodevelopmental problems later in life (Mueller & Bale, 2008; Schmidt-Kastner, van Os, Esquivel, Steinbusch, & Rutten, 2012).

Although the etiological pathways described above may prove fruitful, it is also important to consider confounding factors associated with birth weight and ADHD, and to determine the

extent to which the association between birth weight and ADHD may vary by such factors. Clarifying the degree to which confounds may contribute to their association will lead to a better understanding of the relationship between birth weight and ADHD, and indicate which pathways are more likely.

Factors Associated with Birth Weight and ADHD

Several other prenatal risk factors have been identified as risk factors for both low birth weight and ADHD (Silva, Colvin, Hagemann, & Bower, 2014; Sucksdorff et al., 2015; Wiggs, Elmore, Nigg, & Nikolas, 2016). Unfortunately, many of these correlated risk factors are not controlled for in a number of studies, and questions remain regarding specificity of the effect of birth weight, as opposed to other correlated risk factors, on the development of ADHD symptoms.

Gestational age at birth is highly correlated with birth weight, with earlier gestational age correlated with lower birth weight (Kitchen, 1968; Olsen, Groveman, Lawson, Clark, & Zemel, 2010). Several studies have focused on gestational age at birth instead of birth weight as a risk factor for ADHD, and meta-analyses indicate there is indeed a negative association between gestational age and ADHD symptoms (Aarnoudse-Moens, Weisglas-Kuperus, van Goudoever, & Oosterlaan, 2009; Bhutta, Cleves, Casey, Cradock, & Anand, 2002). In light of these findings, some have suggested that birth weight is merely a proxy for gestational age and does not exert an independent effect on ADHD symptoms.

Similar to gestational age, prenatal exposure to tobacco also has been associated with both birth weight and the development of ADHD (Linnet et al., 2005; Mick, Biederman, Faraone, Sayer, & Kleinman, 2002; Milberger, Biederman, Faraone, Chen, & Jones, 1996; Milberger, Biederman, Faraone, & Jones, 1998), resulting in contentious debate about the harm prenatal smoking may cause not only during the newborn period, but also throughout childhood and adulthood. Thus, given the possibility that prenatal smoking is a causal risk factor for both low birth weight and ADHD, it is possible that the association between birth weight and ADHD symptoms is partially, if not fully, accounted for by exposure to prenatal smoking.

Finally, two related factors, the geographic region of the study and the race of study participants, are often correlated with socio-economic status (Costello, Keeler, & Angold, 2001; National Center for Education Statistics, 2007; World Health Organization, 2009), which is in turn associated with access to prenatal care, birth weight, and risk for development of ADHD (Fiscella, Franks, M.R., & Clancy, 2008; Gray, Edwards, Schultz, & Miranda, 2014; Larsson, Sariaslan, Langstrom, D'Onofrio, & Lichtenstein, 2014; Silal, Penn-Kekana, Harris, Birch, & McIntyre, 2012). Individuals of lower SES typically have reduced access to adequate prenatal care and prenatal nutrition, which increases the likelihood that they will have a child born low birth weight (Gortmaker, 1979; Moore, Origel, Key, & Resnik, 1986; Quick, Greenlick, & Roghmann, 1981; Showstack, Budetti, & Minkler, 1984). Further, inadequate prenatal nutrition has been linked to neurodevelopmental disorders (Susser, Hoek, & Brown, 1998).

In addition to considering factors associated with birth weight and ADHD, it is also important to consider the extent to which the correlation between ADHD and birth weight may vary based on sex, given the large sex difference in the prevalence of ADHD (Akinbami, Xiang, Pastor, & Reuben, 2011; Visser et al., 2014). Previous studies have rarely examined sex as a moderator of the association between birth weight and ADHD symptoms, and the few studies that have examined sex as a moderator have produced mixed results (Momany et al., 2016; Murray et al., 2015). A recent review by Martel (2013) proposed that Sexual Selection Theory could explain the sex difference observed in ADHD. Sexual Selection Theory argues that males have an increased risk of developing ADHD after exposure to early life risk factors such as low birth weight (please see Martel (2013) for a review of sexual selection theory).

Methodological Considerations in Studies of Birth Weight and ADHD

In addition to the factors that may conceptually account (or partially account) for the association between birth weight and ADHD, there are several methodological factors worth considering as well. First, a variety of measures have been used to assess ADHD symptoms (e.g., questionnaire, diagnostic interview), and these measures vary by both method of administration and diagnostic validity (Gordon et al., 2006; Lahey & Willcutt, 2002; Pelham, Fabiano, & Massetti, 2005). Similarly, previous work has indicated the validity of ADHD symptom ratings varies by the informant used (e.g., parent, teacher, self; Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999; Martel, Schimmack, Nikolas, & Nigg, 2015; Powe et al., 1998; Van Voorhees, Hardy, & Kollins, 2011). Methodological variation also exists in the ascertainment of birth weight (i.e. hospital record, parental report, self-report). Retrospective parental reports have been demonstrated to be reliable, even up to 30 years after birth (Catov et al., 2006; Lumey, Stein, & Ravelli, 1994), but retrospective self-reports have not been found to be as reliable (Troy et al., 1996). Age of study participants may also affect the estimate of the association between birth weight and ADHD symptoms since previous work has suggested that individuals manifesting primarily hyperactive-impulsive symptoms or less severe symptoms of ADHD may no longer experience impairment in adulthood (American Psychiatric Association, 2013; Hart, Lahey, Loeber, Applegate, & Frick, 1995; Lara et al., 2009; Leopold et al., 2016; Willcutt, 2012).

Finally, several different study designs have been employed to investigate birth weight and ADHD symptoms. Some studies dichotomize individuals based on birth weight, other studies dichotomize individuals based on ADHD status, and still other studies examine one or both variables as continuous measures. Furthermore, studies that dichotomize individuals based on birth weight or ADHD symptoms often use stringent cut-offs, such as birth weight less than 1500g. Given the influence of sampling from extreme ends of the birth weight and ADHD symptom spectrums may have on the association, it is important to consider the participant recruitment method (e.g., based on birth weight, based on ADHD diagnosis, etc.) when quantifying the association across studies. Similarly, the mean birth weight of the sample provides information about the composition of the sample with regard to birth weight (i.e. primarily participants born low birth weight), and allows for the examination of the association between birth weight and ADHD symptoms across the distribution of birth weight.

The Current Study

As discussed, numerous studies have suggested that there is an association between birth weight and ADHD symptoms. However, a systematic quantification of the association between birth weight and ADHD symptoms is warranted given the variability in effect sizes and study designs, the lack of studies controlling for risk factors correlated with birth weight and ADHD, and the paucity of studies examining sex as a moderator of the association. It should be noted that a meta-analysis examining the association between birth weight and ADHD has been conducted previously (Aarnoudse-Moens et al., 2009), and findings of this study indicated infants born very low birth weight and/or very preterm were more likely to experience attention problems compared to infants born normal birth weight or at term. However, the previous meta-analysis focused solely on the association among very low birth weight and/or very preterm child. Thus, the purpose of the current meta-analysis is to quantify the magnitude of the overall association between birth weight and ADHD across the birth weight and ADHD symptomology spectrums, and to determine whether there are moderators of this association. It is hypothesized that there will be a small, but significant, association between birth weight and ADHD symptoms, such that lower birth weight is associated with greater ADHD symptoms. Further, because previous literature has indicated there is a dose-response relationship between birth weight and ADHD symptoms, it is hypothesized that the association will be moderated by the mean birth weight of the sample, such that samples with a lower mean birth weight have a larger (more negative) effect size. Additionally, given the recent theoretical argument that males may be particularly susceptible to early life risk factors that confer risk for ADHD (Martel, 2013), it is hypothesized that the association will be moderated by sex, with samples that have a higher percentage of males exhibiting larger effect sizes. Finally, given previous empirical evidence that birth weight is a robust risk factor for ADHD, it is hypothesized that the association will not differ when including variables correlated with birth weight and ADHD as moderators (i.e., gestational age and prenatal tobacco exposure).

METHOD

Sample of Studies

Studies were identified for the current meta-analyses by conducting searches through May 2016 in the Medline (PubMed), PsychInfo, and ProQuest Dissertation and Theses databases. The search terms “birth weight” and “birthweight” were used in pairwise combination with the terms “attention deficit hyperactivity disorder”, “attention deficit disorder”, “hyperkinetic disorder”, “hyperkinesis”, “attention problems”, “inattention”, “impulsivity”, and “hyperactivity.” The terms could appear anywhere in the text for the searches conducted in the PubMed and Psychinfo databases, but the terms were required to be keywords for the ProQuest Disseration and Theses database. To further identify eligible studies, a backward search was conducted from the reference list of a previous meta-analysis that examined neurobehavioral outcomes in extremely low birth weight infants (Aarnoudse-Moens et al., 2009).

Inclusion criteria—Studies had to meet the following criteria to be included in the meta-analysis: (1) studies were empirical and published in the English language in a peer-reviewed journal or were a component of a dissertation or thesis (literature reviews, meta-analyses, and case studies were not included), (2) studies included a measure of birth weight, (3) studies included a measure of ADHD symptoms or diagnosis (parent report of previous diagnosis, diagnosis from a medical record, diagnostic interview, or questionnaire assessing ADHD symptoms) and (4) studies examined the relationship between birth weight and ADHD in human subjects.

The literature search produced 977 unique studies. Article titles and abstracts were initially examined for inclusion in the meta-analysis by the main coder (AMM). Manuscripts were reviewed if it was unclear whether the study met inclusion criteria based on the article title and abstract. Of the 977 unique studies identified, 701 studies did not meet inclusion criteria per the main coder (see Figure 1). An independent rater (JMK) examined 10 percent of the identified studies to determine study inclusion with adequate reliability (κ = .65, percentage agreement = 87%). Any disagreements were identified by both coders and discussed; in all cases, agreement was reached among discussion.

Additionally, studies were excluded if the participants in the study were part of a specific medical population (e.g., fetal alcohol syndrome, velocardiofacial syndrome), if the study population had already been reported on in another study (the study with the largest sample size was retained), or if the study did not provide sufficient data to calculate an effect size. Several study population characteristics were used to ensure study populations did not overlap, and these were: the dates of recruitment, cohort name if applicable (e.g., TRAILS study), and the city and country from which participants were recruited. If a study did not provide sufficient data to calculate an effect size, the corresponding author was contacted to obtain this information (21% response rate). This resulted in 94 unique effect sizes *(N* = 4,645,482) in the current meta-analysis. Of the studies included, five studies (n = 1,580) were unpublished dissertations or theses and, therefore, have not undergone formal peer-review (Adger-Antonikowski, 2009; Duris, 2002; Fazio, 2008; Johnson-Cramer, 1999; Williams, 2010).

Management of non-independent samples

Studies had non-independent data for several reasons. First, studies often had more than one measure of ADHD symptoms (e.g., self-report of symptoms and clinician diagnosis). Second, studies had non-independent data if multiple informants were included in the study. Finally, some studies were longitudinal and therefore included data on ADHD symptoms at multiple time points, resulting in non-independent data. For several of the studies with non-independent samples, one of the measures or one of the time points in the longitudinal studies had a larger sample size. In these cases, these data were used to calculate the effect size. If the sample size was the same for multiple time points and the same type of measure (e.g. two questionnaires) was used at each time point, an average effect size was calculated. However, if the sample size was the same at multiple time points, but different types of measures (e.g. parental questionnaire and interview) were used at each time point, the effect size from the time point that used the measure with the highest diagnostic validity was used.

Hence, DSM-based diagnostic interviews or clinical diagnoses were utilized over informant and self-report questionnaires and informant report measures were utilized over self-report measures (Erhardt et al., 1999; Powe et al., 1998; Van Voorhees et al., 2011). Finally, some studies included data on two informant-report measures (i.e., teacher and parent or mother and father) with the same sample size. In these cases, an average effect size was calculated (for procedures see Borenstein, 2009).

Coding Procedures

Variables coded included the following (see moderator variables section below for additional information): mean gestational age of the sample, percent exposed to prenatal tobacco, country, race (i.e. percent Caucasian, African American, Asian, and Latino), sex (percentage of sample that is male), ADHD measurement method, informant who provided ratings of ADHD symptoms, mean age at assessment of ADHD symptoms, birth weight source, sample type, mean birth weight of the sample, and test statistics. For each study, an effect size *r* (Pearson's correlation) was calculated for the association between birth weight and ADHD symptoms. Effect sizes calculated from unadjusted statistics were used when possible; however statistics adjusted for various covariates were included if unadjusted statistics were not reported.

All studies were initially coded by the main coder (AMM), who also developed the coding procedure. This rater trained the independent rater (JMK) on coding procedures, and the independent rater coded 10 percent of the included studies (effect size estimates, sample sizes, and the twelve moderator variables) with adequate reliability (κ = 0.6 1, percentage agreement = 70 100%). The raters reviewed coding discrepancies and were able to reach consensus among discussion.

Moderator Variables

Several variables were examined as moderators of the association between ADHD and birth weight. The following moderators were analyzed as continuous variables: percent of participants in a study exposed to any prenatal tobacco, percent of the sample that was male, and the percent of the sample that identified as Caucasian, African American, Asian, and Latino. Importantly, race was analyzed within countries, as opposed to across all countries, because of the different meanings race may have in different countries.

Several variables were examined as categorical moderators: geographic region, ADHD measurement method, informant of ADHD symptoms, sample type, and method of birth weight ascertainment. Geographic region was coded as the continent from which the participants were recruited, as the number of studies from any one country was too small for analysis. ADHD measurement method was classified as "medical record" if the study used ADHD diagnostic status as indicated in a medical or school record, " interview" if the study employed structured or semi-structured diagnostic interviews with the participant or with a parent, "questionnaire" if the study used questionnaire measures filled out by a parent, a teacher, or the participant, and "multiple methods" if the study utilized more than one measurement method (e.g., questionnaire and interview) to assess ADHD symptoms (see Supplemental Table 1 for specific measures used in the included studies). Informant of

ADHD symptoms was classified as “clinician rated” if a clinician (e.g., psychologist, psychiatrist, etc.) determined ADHD status or the study utilized diagnostic status from a medical or school record, whereas studies utilizing parental reports, teacher reports, or self-reports of ADHD symptoms were classified as “parent rated,” “teacher rated,” and “self rated,” respectively. Studies that used ratings from multiple informants (e.g., parent and teacher) to determine ADHD status were classified as “multi-person rated.” Sample type was coded as “birth weight based” if studies compared individuals born low birth weight to individuals born normal birth weight, “ADHD based” if studies dichotomized individuals based on ADHD diagnostic status, “community based” if participants were recruited from the community, and “population based” if the study utilized large databases containing medical information on all individuals born in a particular country, county, or city during a specified time period. Finally, studies that extracted birth weight data from medical records were categorized as “medical record,” studies relying on retrospective parental report or self-report were categorized as “parental report” or “self-report,” respectively, and studies that used more than one method to ascertain birth weight (i.e., medical record and parental report) were categorized as “multiple methods.”

Mean gestational age of the sample, the mean birth weight of the sample, and mean participant age at ADHD assessment were analyzed as continuous and categorical variables. Studies were categorized as term (>=37 weeks), moderate preterm (28 weeks <gestational age <37 weeks), and very preterm (<=28 weeks; World Health Organization, 1992). Similarly, the mean birth weight of the sample was categorized into normal birth weight (>=2500g), low normal birth weight (1500g>birth weight>2500g), and very low birth weight (<=1500g; World Health Organization, 1992). Finally, age at ADHD assessment was binned into four groups for categorical analysis: 3–5.9 years, 6–8.9 years, 9–12.9 years, and 13–20 years.

Analytic Strategy

Analyses were conducted using the *metafor* package in R (Viechtbauer, 2010). First, analyses were conducted to examine potential publication bias. Then, analyses were employed to identify studies that were potential outliers. Following these analyses, average effect sizes were calculated for all 94 studies (including unpublished studies) included in the current meta-analysis and for the 89 studies that were published. Additionally, average effect sizes were calculated for the following subsets: studies that reported effect sizes adjusted for various covariates, all studies that reported unadjusted effect sizes (unpublished dissertations and theses included), and studies that reported unadjusted effect sizes and had undergone formal peer-review (unpublished dissertations and theses excluded). None of the adjusted studies were unpublished, and thus separate analyses with and without unpublished studies were not necessary for the adjusted studies. Finally, the twelve moderators detailed above were examined.

Publication Bias—The trim-and-fill method (Duval & Tweedie, 2000) was used to evaluate the impact of publication bias and to address the “file drawer problem” (Rosenthal, 1979). This method provides an estimate of the number of missing studies, as well as an adjusted effect size that includes the filled studies. Additionally, a test of funnel plot

asymmetry was conducted using Egger's Test of the Intercept (Egger, Davey Smith, Schneider, & Minder, 1997) which provided a statistical test of the relation between sample size and effect size.

Outlier Analyses—Several analyses were employed in an attempt to detect outliers that may be affecting the results, but a study was only excluded from analysis if it was an outlier in all three of the outlier analyses. First, standardized residuals for each study were derived and any study with a standardized residual with an absolute value greater than three was considered an outlier (Viechtbauer & Cheung, 2010). The second method was a visual examination of a forest plot from a leave-one-out analysis, with outliers indicated by the effect size deviating a significant amount from the other effect sizes when a particular study is omitted (Viechtbauer, 2010; Viechtbauer & Cheung, 2010). The third method was to generate a forest plot of increasing effect sizes. Any study that had a confidence interval that did not overlap with other study confidence intervals was considered an outlier.

Meta-analytic strategy—Average effect sizes were calculated using the 'rma' function, which fit meta-analytic random and mixed effects models, and the method was specified to be DerSimonian-Laird (DerSimonian & Laird, 1986). A test for residual heterogeneity was also conducted by the 'rma' function, resulting in a Q-statistic (Cochran, 1954), which tested whether the variability in the observed effect size is larger than expected based on the sampling variability. A significant test suggested that the true outcomes were heterogeneous, warranting the examination of moderators. Additionally, the I^2 statistic, which quantified the percentage of variation representing true heterogeneity as opposed to sampling error, was calculated and included, as I^2 is less sensitive to the number of studies included in the analysis (Higgins & Thompson, 2002; Higgins, Thompson, Deeks, & Altman, 2003).

Moderator Analyses—Studies that only provided statistics adjusted for covariates were not included in moderator analyses. To examine the effect of moderators, mixed-effect models were employed, with continuous and categorical moderators specified using the 'mods' argument (categorical moderators used the 'factor' function within the 'mods' argument). Continuous moderators were tested using meta-regression techniques and categorical moderators were tested one at a time, similar to an ANOVA. Once moderators were included in the model, a Q_m-test was utilized to test whether variability in the observed outcomes was significantly accounted for by variability in the moderators. The amount of variance in effect sizes accounted for by each moderator (R^2) was estimated. Finally, moderator analyses were conducted with and without the unpublished studies included to determine the extent to which the unpublished studies may be influencing the results.

RESULTS

Study information, including sample size and data on moderator variables for studies with unadjusted effect sizes is presented in Appendix 1. Sample size and covariates included in analysis for studies with adjusted effect sizes are presented in Appendix 2.

Publication Bias and Outlier Analyses

Results of the trim-and-fill analysis examining whether effect sizes were missing from the left side of the mean effect size estimated two effect sizes to be missing. However, the trim-and-fill analysis examining whether effect sizes were missing from the right side of the mean effect size did not indicate any studies to be missing on the right. Egger's Test of the Intercept did not show evidence of publication bias in the current analyses ($z = -1.19$, $p = .23$), and the estimate of the effect size after adjusting for studies missing on the left resulted in a small change in the effect size (change in $r = 0.0036$). Thus, the degree of publication bias detected in the trim-and-fill analysis of the left side is likely not influencing the estimate of the effect size substantially. The only study found to be an outlier in all three outlier analyses and excluded from subsequent analyses was the study by Astbury and colleagues (1985).

Birth Weight and ADHD

A summary of the effect size estimates and 95% confidence intervals for the overall effects for all studies, adjusted studies only, and unadjusted studies only are presented in Table 1.

Adjusted and unadjusted studies—The analysis examining the association between birth weight and ADHD symptoms in 93 studies (including unpublished studies) revealed a small, albeit significant, overall meta-analytic effect of $r = -.15$ (95% CI: [−.16, −.13]), indicating lower birth weight was associated with increased ADHD symptoms. Significant heterogeneity was detected ($Q(92) = 8673.9.0$, $p < .0001$ and $I^2 = 98.9$) indicating examination of moderators of the association was warranted. Exclusion of unpublished studies did not significantly alter the overall meta-analytic effect ($r = -.15$ (95% CI: [−.17, −.13]), and significant heterogeneity was detected ($Q(87) = 8646.8$, $p < .0001$ and $I^2 = 99.0$).

Unadjusted studies—The analysis examining the association between birth weight and ADHD symptoms in studies that reported statistics unadjusted for covariates also found a small, but significant, overall meta-analytic effect. This analysis included 85 studies and revealed a meta-analytic effect of $r = -.14$ (95% CI: [−.16, −.12]), again indicating lower birth weight was associated with increased ADHD symptoms. Significant heterogeneity was still detected when examining only unadjusted studies ($Q(84) = 6598.0$, $p<.0001$ and $I^2 = 98.7$). Results remained the same after excluding the five unpublished studies ($r = -.14$ (95% CI: [−.16, −.12]) and significant heterogeneity remained ($Q(79) = 6571.2$, $p < .0001$ and $I^2 = 98.8$).

Adjusted studies—Similar to the analyses for all studies and unadjusted studies only, the analysis examining the association between birth weight and ADHD symptoms in studies that reported statistics adjusted for covariates resulted a significant overall effect of $r = -.18$ (95% CI: [−.35, −.01]). Eight studies were included in the analysis and significant heterogeneity was detected in the analysis ($Q(7) = 1286.4$, $p < .0001$ and $I^2 = 99.5$).

Moderators of the Association Between Birth Weight and ADHD

Each class within a categorical moderator was required to have at least three studies to be included in analyses. Moderator analyses used effect sizes from studies that reported unadjusted statistics. Thus a total of 85 studies were potentially included in analyses of moderators, although the number of studies for any given moderator varied based on availability of relevant data. The results from the categorical and continuous moderator analyses with all unadjusted studies (unpublished included) are presented in Tables 2 and 3, respectively. The results from the categorical and continuous moderator analyses with unpublished studies excluded are presented in Supplementary Tables 1 and 2, respectively. Given that the overall meta-analytic effect size was the same with and without the unpublished studies, and that the moderator analyses resulted in similar results regardless of inclusion of unpublished studies, the moderator analyses discussed below included $k = 94$ studies (studies from all effect sizes were unadjusted for covariates, $k = 89$ published and 5 unpublished).

Gestational age—Raw data on gestational age at birth was provided for 25 of the studies and ranged between 27 and 40 weeks. Gestational age was not found to significantly moderate the association between birth weight and ADHD symptoms when it was examined as a continuous ($Q_M(1) < .1$, p = .91, R^2=.00) or categorical variable ($Q_M(2) = .2$, $p = .91$, $R^2 = .00$).

Prenatal tobacco exposure—The percentage of participants exposed to any maternal prenatal tobacco use was calculated for 20 studies and ranged from 5 to 55 percent. Effect sizes were not found to differ based on exposure to maternal prenatal tobacco use ($Q_M(1) = 0.6$, $p = .43$, R^2=.04).

Geographic region—Two studies were excluded from analysis as these studies reported results for samples recruited from two different continents. The remaining 83 unadjusted studies included three studies from South America, five studies from Asia, seven studies from Australia & New Zealand, 31 studies from Europe, and 37 studies from North America. Results indicated significant differences in effect sizes by geographic region ($Q_M(4) = 34.7$, $p < .0001$, $R^2 = .22$). Effect size estimates for each continent were as follows: $r = -.04$ (−.07, −.01) for South America, $r = -.28$ (−.49, −.06) for Asia, $r = -.13$ (−.21, −.05) for Australia & New Zealand, $r = -.13$ (−.16, −.11) for Europe, and $r = -.15$ (−.18, −.11) for North America.

Race—The United States was the only country with enough studies to examine race within country as previously discussed. Thus, race was examined within the 20 studies conducted in the United States. Effect sizes varied systematically by the percentage of the sample that was Caucasian ($Q_M(1) = 4.7$, $p = .03$, $R^2 = .28$) and African American ($Q_M(1) = 25.7$, $p < .001$, $R^2 = .67$). Notably, the magnitude of the association between birth weight and ADHD symptoms was found to be smaller (less negative) as the percentage of Caucasian participants in a sample increased. Additionally, the magnitude of the association between birth weight and ADHD symptoms was found to be larger (more negative) as the percentage of African American participants in a sample increased. Finally, the percentages of the

samples that were Asian ($Q_M(1) = 3.7$, $p = .05$, $R^2 = .14$) and Latino ($Q_M(1) = 1.3$, $p = .26$, $R^2 = .00$) were not found to be significant moderators.

Sex—The majority of studies (k=77) reported raw data on the percentage of participants who were males and ranged from 0 to 100 percent. Significant heterogeneity was not explained by the percentage of the participants who were male ($Q_M(1) = 1.3$, $p = .25$, $R^2 = .00$).

ADHD measurement method—The type of ADHD measure used to assess ADHD symptoms was classified into four categories: interview (k=14), multiple methods (k=6), medical record (k=15), and questionnaire (k=50). Analyses revealed effect sizes differed according to the type of ADHD measure ($Q_M(3) = 35.5$, $p < .0001$) and approximately 11% of the heterogeneity was accounted for by the variability associated with the type of ADHD measure used ($R^2 = .11$). The effect size estimates for samples utilizing medical records, interviews, questionnaires, and multiple methods were $r = -.14$ (−.17, −.10), $r = -.21$ (−.29, −.13), $r = -.11$ (−.13, −.08), and $r = -.25$ (−.43, −.07) respectively.

Informant—Five categories were used to classify the individual rating the ADHD symptoms or participants: parent ($k = 47$), clinician ($k = 14$), teacher ($k = 6$), self ($k = 3$) or multiple informants ($k = 15$). Of the studies including ratings from multiple people, 14 of the studies included ratings from a parent and a teacher and the remaining study had ratings from a parent, teacher, and clinician. The individual who rated participant ADHD symptoms accounted for significant heterogeneity in effect sizes ($Q_M(4) = 9.9$, $p = .04$, $R^2 = .24$). Effect sizes were $r = -.13$ (−.15, −.10) for parental ratings, $r = -.15$ (−.18, −.11) for clinician ratings, $r = -.13$ (−.19, −.08) for teacher ratings, $r = -.13$ (−.44, −.17) for self-ratings, and $r = -.18$ (−.29, −.07) for ratings from multiple people.

Age at assessment—Seventy-two studies provided raw data on the age of participants at the time of assessment of ADHD symptoms. Participant age was not found to explain significant heterogeneity in the current analysis when examined as a continuous ($Q_M(1) = 0.3$, $p = .86$, $R^2 = .00$) or categorical moderator ($Q_M(3) = 0.2$, $p = .98$, $R^2 = .00$).

Birth weight ascertainment—Four studies did not provide information on the method used to ascertain birth weight of participants. Additionally, studies that used multiple methods to ascertain birth weight were excluded, as these studies often used one method (i.e., medical record extraction) for some participants and a different method (i.e., parental report) for other participants. Finally, studies utilizing self-reported birth weight were excluded because only two studies used this method. In the end, 74 studies were included in the analysis; 26 studies that utilized retrospective parental report and 48 studies that extracted birth weight from medical records. Effect sizes did not vary systematically by the method used to ascertain birth weight ($Q_M(1) = 0.5$, $p = .49$, $R^2 = .19$).

Sample type—As detailed in the methods section, studies were categorized into one of four sample types: ADHD based studies (k=16), birth weight based studies (k=40), community studies (k=17), or population studies (k=12). Results indicated that the association between birth weight and ADHD symptoms did vary significantly by sample

type (Q_M(3) = 43.4, p < .0001, R^2 = .05). Estimates of effect sizes by sample type were as follows: r = −.18 (−.27, −.08) for ADHD based samples, r = −.20 (−.24, −.15) for birth weight based samples, r = −.06 (−.09, −.04) for community based samples, and r = −.09 (−.12, −.06) for population based samples.

Birth weight—Forty-six studies provided data on participant's birth weight, allowing for calculation of the mean birth weight of the sample. Again, we examined mean birth weight of the sample as a moderator to determine if effect sizes systematically across the spectrum of reported birth weights. The mean birth weight of the sample did not account for a significant amount of heterogeneity when examined as a continuous (Q_M(1) = 2.9, p = .09, R^2 = .23 or categorical (Q_M(2) = 5.6, p = .06, R^2 = .20) variable.

DISCUSSION

The current study aimed to quantitatively investigate the association between birth weight and ADHD symptoms, and to test whether the magnitude of this association varied depending on a variety of factors. Results indicate that there is a small, but significant, negative association between birth weight and ADHD, such that lower birth weight is associated with increased symptoms of ADHD. Further, sample type, geographic region of the sample, Caucasian and African American race (in the United States), informant ratings of ADHD symptoms, and measurement method used to assess ADHD symptoms all accounted for significant heterogeneity in the association between birth weight and ADHD symptoms.

Previous research investigating the association between birth weight and ADHD symptoms has resulted in mixed findings, with some studies reporting a positive association such that lower birth weight was associated with fewer symptoms of ADHD (e.g., Duris, 2002; Johnson-Cramer, 1999), and other studies reporting a negative association (e.g., Indredavik et al., 2010; Sasaluxnanon & Kaewpornsawan, 2005). Further, the effect sizes in previous studies varied greatly given the diverse populations from which participants were recruited (e.g., Anderson, Doyle, & Victorian Infant Collaborative Study, 2003; Asbury, Dunn, & Plomin, 2006; Beverly, McGuinness, & Blanton, 2008; Class, Rickert, Larsson, Lichtenstein, & D'Onofrio, 2014; Groen-Blokhuis et al., 2011) and the variety of study designs that were utilized to examine the association between birth weight and ADHD (e.g., Frederick, 2012; Hack et al., 2004; Hultman et al., 2007; Kreppner, O'Connor, & Rutter, 2001; Linnet et al., 2005). The current findings therefore extend the literature by combining effect sizes from a variety of samples to provide an overall effect size regardless of recruitment method or sample population. The largest effect sizes were found for studies that recruited participants based on birth weight or on ADHD diagnostic status. These studies typically recruited individuals from the extreme ends of the ADHD and birth weight spectrums, such that individuals with sub-threshold ADHD symptoms were not included in the studies, or individuals were required to be born very low birth weight (<1500g) or extremely low birth weight (<1000g) as opposed to just low birth weight (<2500g). Thus, this finding suggests that the effect size may increase as the extremity of low birth weight or ADHD symptoms increases. Interestingly, previous research has indicated there is indeed a dose-response relationship between birth weight and ADHD symptoms, with individuals

born at increasingly lower birth weights at increasingly higher risks of developing ADHD (D'Onofrio et al., 2013). However, mean birth weight of the sample was not a significant moderator of the association. This may be due to insufficient power since many studies did not provide mean birth weight of the sample. Additionally, it may be that studies recruiting from the extreme end of the birth weight studies vary along a number of confounding factors (e.g., neonatal morbidities), which in turn may affect the estimate of the effect size between birth weight and ADHD. As is discussed later, continued investigation regarding the effects of such confounding factors is important for an enhanced understanding of the relationship between birth weight and ADHD.

The geographic region from which participants were recruited and Caucasian and African American race (among United States samples) were found to significantly contribute to the heterogeneity in the overall effect size. Notably, socio-economic status and access to adequate prenatal care, which are associated with birth weight and ADHD (Gray et al., 2014; Larsson et al., 2014; Silal et al., 2012), vary greatly by geographic region and ethnicity, and could potentially explain the different effect sizes observed for different geographic regions and ethnicity. Still, the result of the geographic region analysis is likely limited to North American and European samples because very few published papers included samples that were recruited from the other geographic regions (Asia, South America, Australia/New Zealand). Furthermore, there may have been insufficient power to detect moderation by Asian and Latino race/ethnicity given the small number of studies reporting the percentage of the sample that identified as Asian or Latino.

Studies that used multiple informants (i.e. parent and teacher) of ADHD symptoms or that used multiple measurement methods had significantly larger effect sizes than those that used other informants (i.e. parent, teacher, self, or clinician) or single measurement methods (i.e. questionnaire or medical record). Notably, the use of multiple informants and multiple measurement methods has been demonstrated to have the highest validity in ADHD assessment (Erhardt et al., 1999; Gualtieri & Johnson, 2005; Powe et al., 1998; Van Voorhees et al., 2011). Thus, studies that used methods with higher diagnostic validity may have been better able to differentiate between individuals with ADHD and controls. Lastly, gestational age at birth, birth weight, prenatal tobacco exposure, sex, age at assessment, and birth weight ascertainment were not found to account for heterogeneity in the association between birth weight and ADHD symptoms.

Limitations

There are some limitations to the current research. First, some of the categorical variables had a small number of studies included in certain classes (e.g., geographic region, rater of ADHD symptoms), which may have resulted in less accurate estimates of effect sizes for the categorical classes with fewer studies. Similarly, the continuous variables included as moderators varied in the number of studies reporting data, with some variables having few studies reporting usable data, and this may have resulted in decreased power to detect significant effects. Additionally, given the different meaning and implications race may have depending on country, future investigations would benefit from examining the effect of race in countries other than the United States.

Although the current analyses did examine moderation of effect sizes by variables associated with birth weight and ADHD symptoms (i.e., prenatal tobacco exposure, gestational age), there are several other factors that have been shown to be associated with birth weight and/or ADHD symptoms that could not be included in the current analyses due to lack of reported data. For instance, maternal medical status (e.g., maternal age at birth, gestational diabetes, maternal BMI, maternal depression) and prenatal exposure to other substances (e.g., recreational drugs, alcohol) are potential moderators of interest, but studies did not report sufficient data on these variables to include them in the analyses.

A final limitation of the current meta-analysis is that the majority of included studies examined the association between birth weight and ADHD symptoms in children and adolescents. Although age at assessment was not found to be a significant moderator of effect sizes, only three studies examined the association in adult participants, limiting the ability to conclude whether the effect of birth weight on symptoms of ADHD remains in adults. Importantly, the decision-making that was used to handle non-independent data from longitudinal studies may have biased the results, as studies with larger sample sizes (and therefore typically younger participant ages) were included. For instance, five studies conducted when participants were 18 years or older were excluded because the participants were part of a longitudinal study that examined birth weight and ADHD symptoms at an earlier age. Excluding such studies may have resulted in a decreased ability to detect variation in effect sizes across mean participant age.

Implications

The findings of the current study have important considerations for theory, methodology, and clinical practice. First, in line with the Developmental Origin of Health and Disease hypothesis, present findings confirm a negative association between an early life risk factor, low birth weight, and the development of ADHD symptoms such that being born at a lower birth weight is associated with risk for adverse neurodevelopmental sequela. Additionally, the findings of the present study indicate the association does not vary depending on factors correlated with birth weight and symptoms of ADHD, such as gestational age at birth and prenatal tobacco exposure. Although the current analyses identified differences in effect sizes based on several variables, substantial heterogeneity remained after accounting for these variables, indicating other factors not examined in the current analyses may be contributing to variation in effect sizes. Several early life risk factors (e.g. maternal medical status, prenatal alcohol exposure) could not be examined in the current study, as data on these variables was largely unreported. From a methodological standpoint, future research examining early life risk factors of ADHD should examine the numerous other variables that are associated with low birth weight and ADHD, as these other variables may account for some of the heterogeneity that is unexplained in current analyses.

Finally, clinicians monitoring the growth and development of children born low birth weight should also monitor for the development of ADHD symptoms. Although individuals born low birth weight are not guaranteed to develop symptoms of ADHD, they are at a higher risk. By monitoring individuals at higher risk of developing ADHD, detection of symptoms may occur earlier in development, allowing for clinical intervention to be implemented

before impairment due to symptoms becomes severe. Ultimately, it is in the best interest of the individual to identify ADHD symptoms and provide intervention as soon as possible in order to reduce impairments in school, work, and relationships.

Future Directions

As discussed previously, future work should focus on disentangling the multiple correlated risk factors shown to be associated with ADHD. Birth weight has emerged as one of the most robust risk factors, but it is also one of the most researched risk factors due to the ease with which researchers can ascertain this information from medical records or parents. However, individuals who are born low birth weight, particularly those born very or extremely low birth weight, are at risk for a number of prenatal and neonatal comorbidities, and they are often exposed to a variety of neonatal interventions. Indeed, a better understanding of the biological mechanisms underlying the association between birth weight and ADHD may be garnered by investigating the interaction among various prenatal and neonatal risk factors. Moreover, a better understanding of the biological mechanisms contributing to low birth weight and ADHD could potentially lead to the identification of protective factors that may buffer against the development of ADHD symptoms in individuals born low birth weight. Additionally, prior work (Morgan, Loo, & Lee, 2016; Wiggs et al., 2016) has found neuropsychological mechanisms to be significant mediators of the association between birth weight and ADHD symptoms. As such, future research may benefit from examining the extent to which prenatal and neonatal risk factors commonly suffered by those born low birth weight are associated with the neuropsychological mediators of the association between birth weight and ADHD symptoms.

Additionally, although gestational age and prenatal tobacco exposure may not have an effect on the magnitude of the association between birth weight and ADHD, they may still be important risk factors for the development of ADHD. Research efforts examining early life risk factors for ADHD should continue to consider the contribution of these risk factors to the development of ADHD, and future studies investigating the association between birth weight and ADHD symptoms should consider including these factors as covariates in analyses. Furthermore, future research should examine other variables previously identified as predictors of birth weight (e.g., prenatal exposure to other substances, gestational diabetes; Knopik et al., 2005; Linnet et al., 2003; Ornoy, 2005; Ornoy, Ratzon, Greenbaum, Wolf, & Dulitzky, 2001) as potential moderators of the association between birth weight and ADHD.

Finally, the results of the current moderation analyses indicate that sampling from extreme samples results in a larger effect size, suggesting there is a nonlinear relationship between birth weight and ADHD symptoms. However, a nonlinear association would be more conclusively demonstrated via the use of continuous measures of birth weight and ADHD symptoms. A recent study by Momany and colleagues (2016) tested such a nonlinear association using continuous measurements, and found the nonlinear association to be significant in predicting hyperactivity-impulsivity. However, few, if any, other studies have examined the nonlinear association between birth weight and ADHD using continuous

measures. Therefore, future research should attempt to replicate this finding utilizing continuous measures of birth weight and ADHD symptoms.

Conclusion

The current study provided a meta-analytic estimate of the association between birth weight and symptoms of ADHD. Findings indicate birth weight appears to be a robustly associated with ADHD symptoms, such that lower birth weight is associated with increased ADHD symptoms. Interestingly, variation in the association is partially explained by sample type, geographic region, African American and Caucasian race, the informant of ADHD symptoms, and ADHD measurement method. However, substantial unexplained variation in the association between birth weight and ADHD symptoms remains, indicating future work should focus on the correlates of birth weight that may also be contributing to the development of ADHD symptoms.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Asterisks (*) indicate studies included in the meta-analysis.

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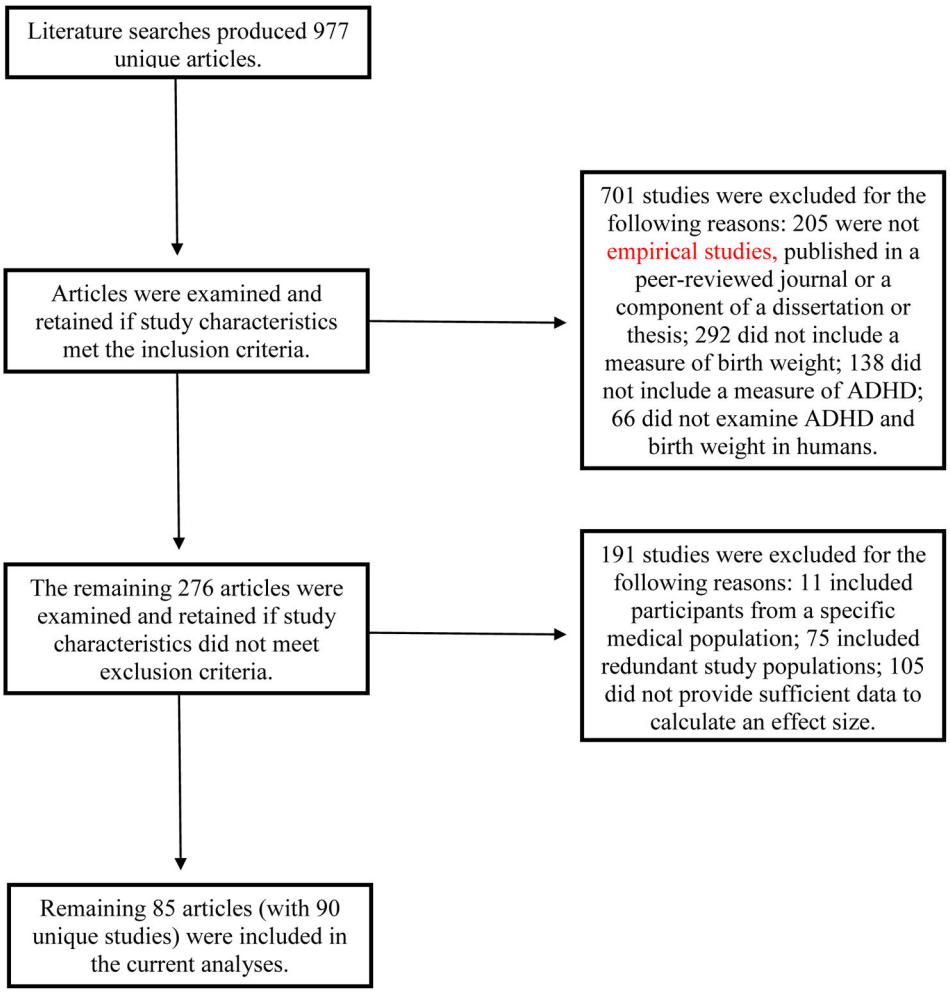

Figure 1.
Flow chart of studies excluded from current analyses.

Table 1

Predicted overall effects with 95% confidence intervals from random effects analyses of birth weight and ADHD symptoms

	k	N	r	95% CI	I^2
All Studies	93	4,645,482	−.15***	−.16, −.13	98.9
All Published Studies	88	4,643,902	−.15***	−.17, −.13	99.0
Unadjusted Studies	85	4,609,947	−.14***	−.16, −.12	98.7
Unadjusted Published Studies	80	4,608,367	−.14***	−.16, −.12	98.8
Adjusted Studies	8	35,535	−.18*	−.35, −.01	99.5

Note.

* $p < .05$,

** $p < .01$,

*** $p < .001$. CI=confidence interval. Adjusted studies include studies that only reported statistics that had been adjusted for covariates. Unadjusted studies include studies that reported statistics without any adjustment for covariates.

Table 2

Predicted effects with 95% confidence intervals from mixed effects analyses for categorical moderators including unpublished studies

	k	N	r̄	95% CI	Q_M	p	R^2
Gestational Age					0.2	.91	.00
Very preterm	4	326	−.15	−.43, .13			
Moderate preterm	15	3,516	−.16	−.24, −.08			
Term	6	3,299,845	−.13	−.23, −.03			
Geographic Region					34.7	<.0001	.22
South America	3	4,112	−.04	−.07, −.01			
Asia	5	873	−.27	−.49, −.06			
Australia & New Zealand	7	5,881	−.13	−.21, −.05			
Europe	31	4,477,464	−.13	−.16, −.11			
North America	37	120,440	−.15	−.18, −.11			
ADHD measure					35.5	<.0001	.11
Medical record	15	4,542,862	−.14	−.17, −.10			
Interview	14	9,262	−.21	−.29, −.13			
Questionnaire	50	55,665	−.11	−.13, −.08			
Multiple methods	6	2,158	−.25	−.43, −.07			
Informant					9.9	.04	.24
Parent	47	146,703	−.13	−.15, −.10			
Clinician	14	4,455,284	−.15	−.18, −.11			
Teacher	6	3,396	−.13	−.19, −.08			
Multiple	15	3,912	−.18	−.29, −.07			
Self	3	652	−.13	−.44, −.17			
Age					0.2	.98	.00
3–5.9 years	13	20,071	−.14	−.19, −.08			
6–8.9 years	30	21,436	−.16	−.21, −.11			
9–12.9 years	17	4,406	−.15	−.23, −.07			
13–20 years	12	8,113	−.16	−.24 −.08			
Birth Weight							
Ascertainment					0.5	.49	.19

J Abnorm Child Psychol. Author manuscript; available in PMC 2019 October 01.

	k	N	r̄	95% CI	Q_M	p	R^2
Parental report	26	128,760	−.15	−.18, −.12			
Medical or birth record	48	4,474,495	−.13	−.15, −.10			
Sample Type					43.4	<.0001	.05
ADHD based	16	6,376	−.18	−.27, −.08			
Birth Weight based	40	8,546	−.20	−.24, −.15			
Community based	17	18,678	−.06	−.09, −.04			
Population based	12	4,576,347	−.09	−.12, −.06			
Birth Weight					5.6	.06	.20
Very low birth weight	4	310	−.09	−.30, .11			
Low birth weight	21	5,009	−.20	−.28, −.13			
Normal birth weight	21	11,803	−.11	−.16, −.06			

Note. CI=confidence interval. P-values derived from tests of heterogeneity examining the amount of variability explained by each moderator variable.

Table 3

Predicted effects with 95% confidence intervals from mixed effects analyses for continuous moderators with unpublished studies included

	k	N	Intercept	b	SE	Q_m	p	R^2
Birth weight (g)	46	17,122	−.28	<.0001	<.0001	2.9	.09	.23
Caucasian	20	104,812	−.30	.0024	.0011	4.7	.03	.28
African American	13	91,199	−.006	−.0055	.0010	25.7	<.001	.67
Asian	9	2,704	−.188	.0130	.0067	3.7	.05	.14
Latino	8	2,445	−.145	.0071	.0063	1.3	.26	.00
Gestational age (wks)	25	3,298,161	−.12	−.0010	.0089	<.1	.91	.00
Prenatal tobacco	20	22,749	−.05	−.0017	.0021	0.6	.43	.04
Sex	77	4,588,590	−.12	−.0004	.0003	1.3	.25	.00
Age at assessment (yrs)	72	54,026	−.15	−.0006	.0033	<.1	.86	.00

Note: p-values derived from tests of heterogeneity examining the amount of variability explained by each moderator variable. Caucasian and non-Caucasian refers to the percentage of the sample that is Caucasian and non-Caucasian, respectively. Prenatal tobacco is the percentage of the sample that was exposed to prenatal tobacco. Sex is the percentage of the sample that is male.

J Abnorm Child Psychol. Author manuscript; available in PMC 2019 October 01.

Appendix 1

Unadjusted studies included in the meta-analysis

Study	N Birth Weight (g)	Sample Type	Geographic Region	Measure Type	Informant	Birth Weight Source	Age (years)	Birth Weight (g)	Male (%)	Caucasian (%)	Non-Caucasian (%)	Gestational Age (weeks)	Tobacco (%)
Adger-Antonikowski, 2009	68	BW	N. America	Questionnaire	Parent	Parent report	4.3	1497	41	83	10	34	---
Anderson et al., 2011	362	BW	Australia/NZ	Questionnaire	Parent	Medical record	8.1	2111	53	---	---	33	---
Anderson et al., 2003	498	BW	Australia/NZ	Questionnaire	Parent	Medical record	8.8	2014	47	---	---	32	---
Andreias et al., 2010	359	BW	N. America	Questionnaire	Parent	Medical record	8	2031	38	---	---	---	---
Asbury et al., 2006	938	Community	Europe	Questionnaire	Teacher	Parent report	7	---	100	---	---	---	---
Asbury et al., 2006	1104	Community	Europe	Questionnaire	Teacher	Parent report	7	---	0	---	---	---	---
Beverly et al., 2008	45	Community	N. America	Questionnaire	Parent	Medical record	11	---	44	---	---	---	---
Bora et al., 2011	212	BW	Australia/NZ	---	Multiple	Medical record	6	2343	53	---	---	34	---
Botting et al., 1997	284	BW	Europe	Interview	Parent	Medical record	12	---	65	---	---	---	---
Boulet et al., 2011	87,578	Population	N. America	Medical record	Parent	Parent report	---	---	51	78	15	---	---
Breeman et al., 2016	488	BW	Europe	Interview	Parent	Parent report	7	2275	50	---	---	---	---
Breslau et al., 1996	414	BW	N. America	---	Multiple	Medical record	6	---	44	23	77	---	35
Breslau et al., 1996	409	BW	N. America	---	Multiple	Medical record	6	---	53	92	8	---	27
Chu et al., 2012	407	ADHD	Asia	Medical record	Clinician	Medical record	9	3186	79	---	---	38	---
Class et al., 2014	3,292,773	Population	Europe	Medical record	Clinical	Medical record	---	---	52	---	---	40	---
Conrad, Richman, Lindgren, & Nopoulos, 2010	104	BW	N. America	---	Multiple	Medical record	11.5	2365	51	88	---	---	---
Crea et al., 2014	449	Community	N. America	Questionnaire	Parent	Parent report	16.7	3161	51	55	32	---	---
Davis, Burns, Snyder, & Robinson, 2007	94	BW	N. America	Questionnaire	Parent	Medical record	5.3	1010	44	70	20	28	---
de Zeeuw et al., 2012	88	ADHD	Europe	Interview	Parent	Parent report	10	3493	86	---	---	40	17
Duris, 2002	117	BW	N. America	Medical record	Clinician	Parent report	8	2316	51	52	48	35	---
Elgen et al., 2013	268	BW	Europe	Interview	---	---	19	---	---	---	---	---	---
Emond et al., 2008	164	BW	S. America	---	Multiple	Medical record	8.2	2774	---	---	---	39	---
Fazio, 2012	588	ADHD	N. America	Interview	Parent	---	8.5	---	70	90	---	---	26
Frederick, 2012	50	Community	N. America	Questionnaire	---	---	---	3500	100	---	---	---	---
Galera et al., 2011	1665	Community	N. America	Questionnaire	Parent	Medical record	---	---	51	---	---	---	---
Groen-Blokhuis et al., 2011	6631	Population	Europe	Questionnaire	Parent	Parent report	3	---	0	---	---	---	---
Groen-Blokhuis et al., 2011	6661	Population	Europe	Questionnaire	Parent	Parent report	3	---	100	---	---	---	---
Grunau et al., 2004	84	BW	N. America	Questionnaire	Parent	---	17.5	1748	60	---	---	31	---
Hack et al., 2004	207	BW	N. America	Questionnaire	Parent	Medical record	20	---	100	---	---	---	---
Hack et al., 2004	235	BW	N. America	Questionnaire	Parent	Medical record	20	---	0	---	---	---	---

Study	N Birth Weight (g)	Sample Type	Geographic Region	Measure Type	Informant	Birth Weight Source	Age (years)	Birth Weight (g)	Male (%)	Caucasian (%)	Non-Caucasian (%)	Gestational Age (weeks)	Tobacco (%)
Halmoy, Klungsoyr, Skjaerven, & Haavik, 2012	1,150,705	ADHD	Europe	Medical record	Clinician	Medical record	---	---	51	---	---	---	---
Hanc et al., 2015	615	ADHD	Europe	Medical record	Clinician	Medical record	10.5	3383	100	---	---	---	---
Hanke et al., 2003	136	BW	Europe	Questionnaire	Parent	Medical Record	6.2		45	---	---	---	---
Hatch, Healey, & Halperin, 2014	197	Community	N. America	---	Multiple	Parent report	4.3	3288	74	40	42	---	---
Huang et al., 2012	84	BW	Asia	Questionnaire	Parent	---	---	---	59	---	---	---	---
Hultman et al., 2007	1055	Population	Europe	Questionnaire	Parent	Medical record	8.5	---	100	---	---	---	---
Hultman et al., 2007	1066	Population	Europe	Questionnaire	Parent	Medical record	8.5	---	0	---	---	---	---
Indredavik et al., 2010	146	BW	Europe	Interview	Parent	Medical record	14	2753	48	---	---	35	---
Jaspers et al., 2013	1664	ADHD	Europe	Questionnaire	Parent	Medical record	---	---	48	---	---	---	31
Johnson-Kramer, 1999	36	BW	N. America	Medical record	Clinician	Medical record	10.4	1787	100	---	---	---	---
Kadziela-Olech & Piotrowska-Jastrzebska, 2005	100	ADHD	Europe	Questionnaire	Parent	Parent report	7.4	---	85	---	---	---	---
Knopik et al., 2005	5,872	Population	N. America	Interview	Parent	Parent report	14.4	---	0	87	---	---	37
Kreppner, O'Connor, & Rutter, 2001	214	Community	Europe	---	Multiple	Medical record	6	---	50	---	---	---	---
Lahti et al., 2006	267	Community	Europe	Questionnaire	Parent	Medical record	---	3573	46	---	---	---	9
Langley et al., 2007	356	ADHD	Europe	Interview	Parent	Parent report	9.2	3286	90	---	---	---	46
Linnet et al., 2005	4,105	Population	Europe	Medical record	Clinician	---	---	---	---	---	---	---	36
McCormick, Gortmaker, & Sobol, 1990	10,522	Population	N. America	Questionnaire	Parent	Parent report	---	---	---	---	---	---	---
McGrath et al., 2005	184	BW	N. America	Questionnaire	Parent	Medical record	4	1742	50	88	12	32	---
Melchior et al., 2015	1,113	Community	Europe	Questionnaire	Parent	Medical record	5	---	53	---	---	---	21
Mick et al., 2002	483	ADHD	N. America	Interview	Parent	Parent report	11.7	---	53	96	---	---	14
Minde, Webb, & Sykes, 1968	112	ADHD	N. America	Medical record	Clinician	Medical record	9.8	3250	89	---	---	---	---
Mitchell, Aman, Turbott, & Manku, 1987	97	ADHD	Australia/NZ	---	Multiple	Parent report	8.9	3236	85	---	---	---	---
Momany et al., 2016	773	ADHD	N. America	---	Multiple	Parent report	12.3	3398	56	74	16	---	10
Morales, Polizzi, Sulliotti, Mascolino, & Perricone, 2013	120	BW	Europe	Questionnaire	Parent	Parent report	5.2	2463	46	---	---	36	---
Murray et al., 2015	3,748	Population	S. America	Questionnaire	Parent	Medical record	4	---	52	---	---	---	27
Nosarti, Allin, Frangou, Rifkin, & Murray, 2005	116	BW	Europe	Questionnaire	Parent	Medical record	15	2300	55	---	---	34	---
O'Callaghan & Harvey, 1997	87	BW	Australia/NZ	---	Multiple	Medical record	10.7	860	31	---	---	27	---
Offord, Sullivan, Allen, & Abrams, 1979	63	ADHD	N. America	Interview	Parent	---	12.5	3315	100	---	---	---	---
Parker, Collett, Speltz, & Werler, 2016	489	Community	N. America	Questionnaire	Multiple	Parent report	7	3429	49	75	24	39	16
Perkinson-Gloor et al., 2015	105	BW	Europe	Questionnaire	Parent	---	8.3	2235	66	---	---	34	7

Study	N Birth Weight (g)	Sample Type	Geographic Region	Measure Type	Informant	Birth Weight Source	Age (years)	Birth Weight (g)	Male (%)	Caucasian (%)	Non-Caucasian (%)	Gestational Age (weeks)	Tobacco (%)
Razza et al., 2016	751	Community	N. America	Questionnaire	Teacher	Medical record	6	---	48	---	---	---	---
Rice et al., 2010	779	Community	---	Questionnaire	Parent	---	6.7	3074	---	---	---	---	5
Rickards, Kelly, Doyle, & Callanan, 2001	161	BW	Australia/NZ	Questionnaire	Teacher	Medical record	14	1740	56	---	---	32	---
Ross, Lipper, & Auld, 1992	168	BW	N. America	Medical record	Clinician	---	7.5	---	52	51	45	---	---
Sasaluxnanon & Kaewpornsawan, 2005	241	ADHD	Asia	---	Multiple	Parent report	8.8	---	88	---	---	---	---
Sato et al., 2004	80	BW	Asia	Medical record	Clinician	Medical record	5	1812	---	---	---	---	---
Schmitz et al., 2006	200	ADHD	S. America	Interview	Parent	Parent report	11.8	3305	68	---	---	---	24
Sciberras, Ukoumunne, & Efron, 2011	4,464	Community	Australia/NZ	Questionnaire	Parent	Parent report	6.8	---	51	---	---	---	---
Scott et al., 2012	259	BW	N. America	Interview	Parent	Parent report	6	1917	46	40	60	---	---
Sengupta et al., 2006	191	ADHD	N. America	Medical record	Clinician	Parent report	9	---	87	---	---	---	51
Simonds & Aston, 1980	106	BW	N. America	Interview	Parent	Medical record	---	---	---	---	---	---	---
Smith et al., 2014	398	ADHD	---	Questionnaire	Parent	---	10.7	3389	83	---	---	40	---
St. Sauver et al., 2004	5,631	Population	N. America	Medical record	Clinician	Medical record	---	---	52	98	---	---	---
Strang-Karlsson et al., 2008	334	BW	Europe	Questionnaire	---	Medical record	22.5	2396	41	100	0	35	18
Sullivan, Msall, & Miller, 2012	180	BW	N. America	Medical record	Clinician	Medical record	17.1	1805	46	---	---	33	---
Sykes et al., 1997	260	BW	Europe	Questionnaire	Teacher	Medical record	7.4	---	0	---	---	---	---
Sykes et al., 1997	182	BW	Europe	Questionnaire	Teacher	Medical record	7.4	---	100	---	---	---	---
Szatmari, Saigal, Rosenbaum, & Campbell, 1993	274	BW	N. America	---	Multiple	Parent report	8	2163	46	---	---	---	---
Taylor, Klein, Minich, & Hack, 2000	164	BW	N. America	Medical record	Clinician	Medical record	11.1	---	32	55	---	---	---
Van den Bergh & Marcoen, 2004	72	Community	Europe	---	Multiple	Medical record	8.5	3301	53	---	---	---	---
Van Hus, Potharst, Jeukens-Visser, Kok, & Van Wassenear-Leemhuis, 2014	165	BW	Europe	---	Multiple	Medical record	5.2	2285	45	---	---	35	---
Wagner, Schmidt, Lemery-Chalfant, Leavitt, & Goldsmith, 2009	748	Community	N. America	---	Parent	Medical record	8.1	2503	51	---	---	36	---
Wiles et al., 2006	5,333	Community	Europe	Questionnaire	Parent	Medical record	6.8	3470	---	---	---	---	---
Williams, 2010	771	BW	N. America	Questionnaire	Parent	Medical record	---	2292	48	72	---	---	29
Yang, Chen, Yen, & Chen, 2015	61	BW	Asia	Interview	Parent	Medical record	13.4	1208	52	---	---	30	---

Appendix 2

Adjusted studies included in the meta-analysis

Study	N	Covariates included in estimate of effect size
Gatzke-Kopp & Beauchaine, 2007	117	Household income, maternal and paternal antisocial symptoms, gestational age, maternal substance abuse & maternal smoke exposure
Heinonen et al., 2011	893	Sex
Jackson & Beaver, 2015	1,266	Age, sex, race, maternal disengagement, & genetic markers
Kelly, Nazroo, McMunn, Boreham, & Marmot, 2001	5,181	Age, sex, birth weight, social class, single parenthood, & smoke exposure
Langley et al., 2010	1261	Age & medication use
Nadeau, Tessier, Boivin, Lefebvre, & Robaey, 2003	162	Family adversity
Pettersson et al., 2015	21,775	Sex & gestational age
van Mil et al., 2015	6,015	Sex & age

J Abnorm Child Psychol. Author manuscript; available in PMC 2019 October 01.