

Is the Association of ADHD with Socio-Economic Disadvantage Explained by Child Comorbid Externalizing Problems or Parent ADHD?

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Abstract It has been unclear whether an associations of child ADHD with socio-economic disadvantage (SES) could be accounted for by (a) parental ADHD explaining both low SES and child ADHD, and/or (b) the joint overlap of ODD or CD with low SES and ADHD. Study 1 used a community-recruited case-control sample with detailed evaluation of SES indicators, child ADHD, child externalizing, and parent ADHD symptoms ($n = 931$ children, 521 ADHD, 577 boys, 354 girls) in a path modeling analysis with latent variables. Study 2 evaluated ADHD and externalizing behavior in a regression model using a poverty index for SES, in 70,927 children (48.2% female) aged 5–17 years from the US 2011–2012 National Survey of Children’s Health (NSCH). In Study

1, lower SES was related to the ADHD latent variable, $\beta = -.18, p < .001$; 95%CI $[-.25, -.12]$. This effect held when parent ADHD and child ODD and CD were in the model, $\beta = -.11, p < .01$, 95% CI $[-.09, -.03]$, equivalent to OR = 1.50, 95% CI $[1.12–2.04]$. In Study 2, these results replicated. Adjusting only for age and sex, children from families who were below 200% of the federal poverty line were more likely to have moderate or severe ADHD than no ADHD, versus children above that line, OR = 2.13, 95% CI $[1.79, 2.54]$, $p < .001$. The effect held after adjusting for disruptive/externalizing problems, OR = 1.61, $p < .01$, 95%CI $[1.32, 1.96]$. The effect size for comparable models was similar across both studies, lending higher confidence to the results. It is concluded that the SES association with child ADHD is not explained by artifact and requires a mechanistic explanation.

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The association between socioeconomic disadvantage (SES) and psychological and mental disorders has been established for decades (Dohrenwend and Dohrenwend 1969; Faris and Dunham 1939/1960; Hollingshead and Redlich 1958; McLaughlin et al. 2012; Reiss 2013; Silver et al. 2002). Its association with conduct disorder (CD) and oppositional defiant disorder (ODD) is likewise uncontroversial (Loeber et al. 1995; Merikangas et al. 2010; Miech et al. 1999). On the other hand, while over three dozen studies have also suggested a correlation of variously-defined measures of SES with ADHD, and a recent systematic review and meta-analysis confirmed a small but reliable association (Russell et al. 2016), the reality of this association remains in question.

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Overlap of both SES and ADHD with Comorbid Externalizing Behavior as an Artifact

As underscored by Russell et al. (2016), alternative explanations for the SES association with ADHD remain poorly studied. They reported that 23 studies of SES and ADHD have examined covariates; but we note that only six made an attempt to adjust for the conjoint association of low SES with both child ADHD and child externalizing behavior problems or ODD and CD (Bauermeister et al. 2007; Bøe et al. 2012; Counts et al. 2005; Ford et al. 2004; Larsson et al. 2014). Of those, 3 found that the SES-ADHD correlation was not independent of child externalizing problems (Counts et al. 2005; Ford et al. 2004; Szatmari et al. 1989). Citing this point, recent reviewers conclude that a primary or specific association of low SES and ADHD is unclear (Barkley 2014). This is important because if ADHD itself is partially due to low SES, then an array of potential mediating and moderating mechanisms are opened for early prevention ideas for ADHD. But if the association of SES with ADHD is accounted for statistically by the joint overlap of SES with ADHD and comorbid child externalizing problems, no new insights about etiology or possible intervention would have emerged, even though some broad risk factor (like impulsivity) might be shared by ADHD and externalizing, and influenced by low SES. ADHD emerges earlier in development than conduct disorder or delinquency, and earlier than or concurrently with oppositional defiant disorder, so in our path diagrams we do not expect externalizing to have a direct effect on ADHD. Instead, we control for the joint overlap of ADHD and ODD and CD with SES as explained below.

Parent ADHD Association with Both Low SES and Child ADHD as an Explanation

A second reason for uncertainty is that parent ADHD could statistically or mechanistically explain both social disadvantage (due to downward drift) and the child's ADHD. This potential genotype-environment correlation (Russell et al. 2015) is plausible because in addition to ADHD's heritability, adults with ADHD tend to earn and save less money and have lower educational and occupational achievement than those without ADHD (Barkley and Fischer 2010; Barkley et al. 2010; Loe and Feldman 2007; Mannuzza et al. 1997; Miech et al. 1999). If this explanation held, then a correlation of low SES with ADHD could be dismissed as relatively unimportant to understanding the specific etiology or phenomenology of ADHD. Two studies (Biederman et al. 2002; Counts et al. 2005) conducted a direct evaluation of parent ADHD; when it was controlled, the SES-child-ADHD association was statistically unreliable in both studies.

In contrast, Larsson et al. (2014) effectively controlled parent ADHD by adjusting for differences in SES indicators in

sibling pairs, and supported a causal influence of lower SES on child ADHD. They secondarily checked the result by removing children who had been diagnosed with ODD or CD; without giving details they stated that effects survived. However, this study's generalizability outside of Sweden is unclear and, like all large population surveys, it is not clear what proportion of ADHD cases were captured or how accurate were the diagnoses.

SES as Mode of Transmission?

The same covariance model that asks whether parent ADHD explains the statistical association of SES and child ADHD, can also ask the reverse: whether low SES statistically mediates the transmission of parent ADHD to child ADHD. If it does, avenues for intervention again are opened up and theories of mechanism could be expanded to account for this. For simplicity, we present only one integrated (correlational) model for the relation of parent ADHD and SES. However, for completeness, we note secondarily the regression model results in which parent ADHD effects operate statistically "through" SES and vice versa. Note that it is also possible that low SES could accentuate parent ADHD (e.g., poor health, stress, and other contributors), so the association of parent ADHD and their SES may be bidirectional.

Methodological Considerations

Before proceeding, two methodological issues bear mention. First, SES is an amalgam of multifaceted constructs including occupational status, income, wealth/poverty, and educational attainment. In Study 1 we attempt to improve the ADHD literature, in which many prior studies of ADHD looked only at one of these features, by creating a latent SES variable that incorporates all of them. In Study 2, we follow other large scale national population studies by using an accepted poverty indicator.

Second, as noted, population surveys have the advantage of generalizability, but often suffer limited measurement depth or validity (e.g., assessing SES by income alone, or ADHD by one or two global questions) due to the costs associated with large samples. Clinically recruited samples tend to be small (Russell et al. 2016) and are vulnerable to referral bias. Community-identified case-finding studies avoid these problems but remain vulnerable to volunteer bias and thus limited generalizability. To address all this, we conducted two studies using complementary sampling approaches, enabling us to comment on generalizability of our conclusions about the role of externalizing comorbidity, while Study 1 provides a strong test of a parent ADHD effect.

Summary

In all, resolving the question of whether there is a specific association of ADHD and SES is important to conceptualization of ADHD. The present report aims to determine whether a putative association of ADHD with low SES is reliable, or whether it can be explained by two obvious yet unresolved confounds: (a) an artifact in which the unexamined variance shared between SES, ODD/CD, and ADHD accounts for the apparent association of child ADHD with low SES, or (b) the association of parent ADHD with both SES (either causally or bidirectionally) and with child ADHD (e.g., a genotype-environment correlation). In Study 1, we examined these questions in a community sample with very well-characterized ADHD and SES. In Study 2, we evaluated the generalizability of the Study 1 externalizing finding in a representative national sample.

Study 1: Community-Based Case-Control Study

Study 1 Methods

Participant Recruitment and Enrollment Participants were 931 individual children ages 7–14 years (mean [M] = 9.3 years, standard deviation [SD] = 1.6 years, 62% male) and their biological parents recruited between 2009 and 2015. The 931 included 154 sibling pairs, with four families having three children in the study. Non-independence was handled statistically (below). Families were recruited via community outreach using commercial mailing lists and public advertisements to minimize referral bias. A multi-gate case-finding process was used to ascertain eligible ADHD and typically developing comparison children. All procedures were approved by the local Institutional Review Board and complied with applicable guidelines for protection of human participants.

Participant Selection and Child ADHD and ODD/CD Evaluation After an initial telephone screening to identify rule outs (listed below), an evaluation visit was scheduled. A semi-structured clinical interview (Kiddie Schedule for Affective Disorders and Schizophrenia, (K-SADS-E)(Puig-Antich and Ryan 1996) was completed with a parent by a trained master's degree level clinical interviewer. Each interviewer double coded 20 tapes with a criterion interviewer to ensure process fidelity and inter-interviewer reliability (all disorders $k > .80$ in this report). Video recordings of their interviews were viewed by supervising clinicians on a recurring basis and feedback provided as needed as a fidelity check. At least one parent and one teacher completed the ADHD Rating Scale, Version IV (ADHD-RS-IV) (DuPaul et al. 1998), the Conners ADHD Rating Scale-3rd Edition (CRS-III) (Conners 2008), and the Strengths and

Difficulties Questionnaire (SDQ)(Goodman 2001) about the child. All have satisfactory internal and test-retest reliability and validity as documented in the cited manuals and had satisfactory internal reliability in the current sample. Children completed self-report symptom ratings of mood, and an IQ and achievement screen using a reliable and valid short form of the Wechsler Intelligence Scales for Children-4th Edition (WISC-IV) (Wechsler 2003) and the Wechsler Individual Achievement Test-3rd Edition (WIAT) (Wechsler 2009). Interviewers and psychometricians wrote behavioral observations.

Exclusion Criteria Children were excluded if taking any psychiatric medication other than stimulants. At the time of participation, 34.5% of the ADHD group had a prescription (14% amphetamine salts, 20% methylphenidate or related preparations). However, a handful were enrolled inadvertently with other prescriptions (clonidine .4%, guanfacine .4%). Other exclusions were: history of non-febrile seizure, neurological impairments, a prior diagnosis of mental retardation or autism spectrum disorder, head injury with loss of consciousness, sensorimotor handicap, or other major medical conditions, as reported by the parent. This was done to minimize diagnostic errors involving ADHD. After the diagnostic evaluation, youth were ruled out if they had substance addiction, bipolar disorder, history of psychosis, medical or neurological condition discovered at the clinical screen, or estimated IQ <75, again to reduce diagnostic errors. Control children were also excluded for ADHD, learning disability, or conduct disorder but their psychiatric status was otherwise free to vary, so as to approximate typical population and avoid an artificially healthy comparison group.

Categorical Child Diagnosis For categorical diagnosis, the preceding information was presented to a “best estimate” clinical team comprising a board-certified child psychiatrist and a licensed child clinical psychologist who independently coded ADHD and other diagnoses, blind to parent ADHD or family SES-related measures. Consistent with DSM-IV ADHD criteria, the clinicians required that another disorder did not better account for symptoms, that there was evidence of impairment, and evidence of cross-situational symptoms as well as duration and age of onset. Their agreement rates for ADHD, ODD, or CD were acceptable (all $k > .80$) and disagreements were resolved by consensus. A subset of 75 children with subthreshold ADHD (5 symptoms) were included in the study but omitted when checking effects of ADHD vs non-ADHD diagnosis.

Dimensional Analysis of Child ADHD Latent Variable For the primary latent variable analysis of ADHD as a dimensional measure, teacher ratings were relied upon. This enabled an ADHD measure that was independent of parent report of their own ADHD or SES, thus avoiding source-variance

confounding. The teacher scores were the ADHD-RS-IV total score, Conners-III inattentive (cognitive problems) and hyperactive scales, and SDQ hyperactivity total score.

Dimensional Child Externalizing Measure Child ODD and CD symptoms were counted from parent semi-structured diagnostic interview (K-SADS-E) and treated as manifest variables.

Parent ADHD Parent ADHD status was evaluated with the Conners Adult ADHD rating scale (CAARS) ADHD index (Conners et al. 1999), and the Barkley Adult ADHD rating Scale (BAARS) (Barkley 2011) current and recalled childhood symptom scales. Each has adequate reliability, validity, and national norms as documented in the cited papers. Spouse ratings of current symptoms were obtained when possible and used when self-ratings were not available. These measures were used as indicators to create dimensional latent variables for mother and father ADHD.

Secondary Analysis: Familial ADHD and Liability for Psychopathology To check results using an alternative method, we adopted the familial loading score developed by Sham and colleagues for studies of schizophrenia (Andreasen et al. 1978; Van Os et al. 1997; Verdoux et al. 1996) in order to sum familial psychiatric history into a total dimensional liability score, while taking into account family size. In our adaptation, one parent completed a family psychiatric history grid in which they reported how many first- and second-degree relatives had probable or definite case of ADHD, conduct problems, antisocial behavior problems, schizophrenia, major depressive disorder, and bipolar disorder. These were converted into a weighted loading score in which first degree relatives “proportion affected” (to normalize on family size) was weighted at .5 and 2nd degree relatives by .25; these were summed. A total familial loading score was computed for (a) ADHD and for (b) all psychiatric conditions combined. These scores have had adequate reliability in past studies (Verdoux et al. 1996) but because its validity is based only on prediction of proband status, it was used as a secondary, not a primary method.

Social-Economic Disadvantage (SES) SES was operationalized with measures of parental education, family household income, and parental occupational status as follows. (a) For *parental education*, we used the highest parent education as self-reported by parents. Parent education reported with 1 = *Grade School*, 2 = *Some High School*, 3 = *High School Equivalent*, 4 = *Regular High school degree*, 5 = *Some College but no degree*, 6 = *Associates degree*, 7 = *Bachelor's degree*, 8 = *Masters or Law degree*, 9 = *Doctorate, PhD, or MD*. (b) *Total household income* was reported by the primary caregiver in bins to maximize response rate, with 1 = < \$25,000, 2 = < \$35,000, 3 = < \$50,000, 4 = < \$75,000, 5 = < \$100,000,

6 = < \$130,000, 7 = < \$150,000, 8 = *more than \$150,000* (16.6% of families chose < \$35,000 or less, suggesting they were at or below the poverty line). These were converted to dollars for descriptive purposes but analyzed as an ordinal 1–7 scale. (c) *Occupational status score*. Parental occupation was obtained by self-report during the parent interview. Then, occupation was scored by the Nam-Powers-Boyd system which provides a final occupation score of 0–100 (Nam and Boyd 2004). To establish inter-rater reliability, occupational scoring was confirmed by two independent raters who each rated a subset of the sample ($n = 50$) with acceptable agreement (Intra-class correlation >.90).¹ The highest parent occupation score was used as the analysis variable.

Data Reduction

Three latent variables were created. A latent SES variable was created using income, highest parental education, and highest parental occupational score as indicators. A child ADHD latent variable dimensional score was created using teacher ratings (to avoid source-variance confound between dependent and independent variables) listed earlier. A latent ADHD variable was created separately for mothers and for fathers, comprising all available data from among self-report ratings on CAARS, BAARS current, BAARS child, and spouse ratings.

Data Analysis

Models were developed using established path modeling logic (Kenny 1979). In path diagram logic, models are designed to be consistent with causal theory (even though, in this case, observations are cross-sectional). A hypothesized causal path gets a one-directional arrow (and computes a regression coefficient). An unexamined association that exists between two constructs gets a curved dual-headed arrow (and computes a correlation).² We developed the model based on the conceptual logic of how the variables relate as described in the introduction.

The model described in the introduction was then tested using a structural equation model (SEMs; (Shumacker and Lomax 1996) using *Mplus* 7.4 (Muthen and Muthen 1998–

¹ A potential confound in the Nam-Powers-Boyd system is that it scores an occupation score = “0” for a range of reasons. In our sample, the breakdown was as follows: Unemployed ($n = 23$), plus $n = 54$ others that might be debatable: active military ($n = 1$), retired ($n = 7$), disabled ($n = 5$), students ($n = 22$), homemaker ($n = 19$). In a robustness analysis, we recoded these $n = 54$ scores that as missing. We repeated this for all the models (with slight change in N as reported below). Results were essentially unchanged and conclusions remained the same. We therefore report the results with the code as recommended (Nam and Boyd 2004). The robustness results are available on request.

² In a path model, a hypothesized bidirectional effect gets two straight arrows, one pointing in each direction. However, models containing bidirectional effects can only be estimated given strong assumptions and other variables (or instruments) that cause each of the two variables involved in the bidirectional effect; therefore, we did not consider them viable here.

2012). Model fit was examined using multiple fit indices: the comparative fit index (CFI) (Bentler 1990), the Tucker-Lewis index (TLI) (Tucker and Lewis 1973), and the root mean squared error of approximation (RMSEA) (Browne et al. 1993). CFI and TLI values above .90 and RMSEA values below .06 indicate adequate model fit (Hu and Bentler 1999). Full information maximum likelihood (FIML) was used to handle missing data (Arbuckle 1996).

SEM models for the ADHD dimensional latent variable used the robust maximum likelihood estimator; to compare effect sizes to the literature on odds ratios, we converted beta weights to odds ratios using published formulas (Borenstein et al. 2009). When ADHD was treated as a category, the model used the robust weighted least squares estimator to produce fit statistics and robust maximum likelihood (with essentially the same results) to enable us to calculate odds ratios for comparison to other studies.³ Both estimators can identify and handle non-normally distributed data. Non-independent observations (i.e., siblings) were handled using the *Mplus cluster* command. Statistical mediation, used in secondary data checks below, was tested using the *model indirect* command.

The primary model simultaneously modeled SES, mother and father ADHD, child ODD and CD, child ADHD, and age and sex. This was repeated with ADHD as a categorical diagnosis. (In preliminary tests, non-linear (quadratic and cubic) effects were trivial, so only linear effects are reported). In secondary analysis, we computed alternative models and placed all of these in the supplemental materials (available online in Fig. S-3, and summarized in Table S3 online) including separate path models for alternative claims about the causal relation among parent ADHD and SES, for alternative ways of controlling child ODD and CD, and other secondary checks noted later. In particular, we computed effect sizes for a model without parent ADHD for direct comparison of effect size found in Study 1 with that found in Study 2. Robustness analysis was conducted on alternative definitions of child and parent ADHD, including child diagnostic group analyses, family history defined as the familial history loading score, average of parent symptom scores, and highest of parent symptoms scores.

Study 1 Results

Preliminary Data Review Demographic and descriptive statistics are presented in Table 1. Preliminary measurement models for parent ADHD, child ADHD, and SES fit well and are provided Fig. S-1 and S-2 (available online).

³ To obtain fit statistics for an SEM with a dichotomous outcome variable requires the robust weighted least squares estimator. We ran all of our models using both the robust weighted least squares and robust maximum likelihood estimators and received nearly identical results. We present the results using the robust weighted least squares estimator so that the reader has sufficient information to assess model fit.

Primary Hypothesis Test (Omnibus model) Figure 1 displays the primary model and its results. This model had satisfactory fit, $\chi^2(24, N = 931) = 87.29, p < .001, CFI = .99, TLI = .98, RMSEA = .05$. This model adjusts for the association of low SES with parent ADHD and the association of SES with child ODD and CD (i.e., it removes the overlap of SES, ODD or CD, and ADHD, which is accomplished by the singled-headed arrows from SES to child ODD and CD, and accounts for residual covariance of ODD and CD with child ADHD). It revealed an enduring small but reliable non-zero association of low SES with child ADHD in the latent variable model, $\beta = -.11, SE = .04, p < .01; 95\% CI[-.19, -.03]$. The equivalent odds ratio is $OR = 1.50, 95\% CI[1.12-2.04]$. The figure also shows that SES was related to ODD and CD symptoms independently of the SES effect on child ADHD. The complete list of all parameters (including residuals, not shown in figure to ease readability) is available in Table S-1 (online supplement).

Secondary Analysis Question One (Residual Effect After Control of Externalizing) Here, the elements of Fig. 1 relating to parent ADHD were removed to create a model that could be comparable to Study 2. The association of low SES with child ADHD in this model was moderate in size, with adjusted SES effect, $\beta = -.18, p < .001; 95\% CI[-.25, -.11]$. This model again fit well, $\chi^2(34, N = 931) = 97.03, p = .00, CFI = .99, TLI = .98, RMSEA = .05$. This adjusted effect size corresponds to an odds ratio = 1.97; 95% CI[1.52, 2.56] or a Cohen's $d = .37$, a small-to-medium effect by convention. Further details are in Fig. S-4 (online).

Secondary Analysis Question Two (Residual Effect Controlling Only Parent ADHD) Here, the ODD/CD elements in Fig. 1 were omitted to enable further scrutiny of simple effects for dissemination. Fit was excellent, $\chi^2(111, N = 931) = 211.42, p < .001, CFI = .98, TLI = .98, RMSEA = .03$, and the adjusted SES to child-ADHD effect was, as expected, still reliable (adjusted SES main effect on child ADHD: $\beta = -.09, p = .03; 95\% CI[-.17, -.01]$). This adjusted effect size corresponds to $OR = 1.42 (95\% CI[1.22, 1.82])$ or a Cohen's $d = .19$. This is a small effect by Cohen's convention. The total indirect path (obtained using the delta method, i.e., by multiplying the parent ADHD to SES path estimate by the SES to child ADHD path estimate, while also estimating the covariance between these two estimates) was reliable both for paternal ADHD, $\beta_{\text{indirect effect of paternal symptoms}} = -.054, p < .001, 95\% CI[-.08, -.02]$ and maternal ADHD, $\beta_{\text{indirect effect of maternal symptoms}} = -.047, p < .001, 95\% CI[-.07, -.02]$. Further details are in Fig. S-5 (online).

Secondary Question Three (Does Social Disadvantage Statistically Mediate ADHD Intergenerational Transmission) The third analysis is essentially a reconfiguration of the same model as just described, but with a different

Table 1 Study 1: Sample description for analysis of externalizing effects

	Control	Subthreshold	ADHD	<i>p</i>
<i>N</i>	335	75	521	–
% Male	46.9	58.7	72.2	<.001
% Caucasian	87.8	81.3	85.2	.252
% Black	3.6	9.3	7.9	.015
% Hispanic	6.3	6.7	5.0	.675
Age (SD)	9.2(1.6)	8.9(1.6)	9.4(1.5)	.025
Parent highest Education ⁺	7.2(1.3)	6.7(1.3)	6.7(1.3)	<.001
Household income (\$ thousands)	86.4(37.8)	80.0(44.6)	73.5(41.2)	<.001
Occupational Score (1–100) ⁺⁺⁺	75.3(23.1)	73.2(28.0)	65.6(29.8)	<.001
Teacher rated Conners Inattention	1.6(2.1)	5.2(2.5)	9.8(3.9)	<.001
Teacher rated Conners Hyperactivity	1.5(2.1)	4.9(3.4)	9.0(5.5)	<.001
Teacher rated ADHD-RS-IV Total Symptoms	3.2(4.3)	12.2(6.5)	26.1(11.8)	<.001
Teacher rated SDQ Hyperactivity	1.2(1.6)	4.4(2.2)	6.8(2.4)	<.001
ODD score KSAD-S-E	.2(.7)	.7(1.5)	1.7(2.1)	<.001
CD score KSAD-S-E	.04(.3)	.05(.3)	.3(.8)	<.001
Biological Mother Current BAARS Inattention	.4(1.5)	.7(1.7)	1.1(2.0)	<.001
Biological Mother Current BAARS Hyperactivity	.3(.8)	.2(.8)	1(1.7)	<.001
Biological Mother Current CAARS	5.9(4.6)	7.8(5.5)	8.7(5.7)	<.001
Biological Mother Childhood BAARS	1.1(2.4)	2.6(4.2)	3.0(4.2)	<.001
Biological Father Current BAARS Inattention	.7(1.6)	1.7(2.3)	1.7(2.4)	<.001
Biological Father Current BAARS Hyperactivity	.5(1.1)	1.1(1.8)	1.3(2.0)	<.001
Biological Father Current CAARS	6.3(5.6)	8.4(6.6)	10.0(6.9)	<.001
Biological Father Childhood BAARS	2(3.2)	1.4(1.7)	3.9(4.5)	<.001

p-values reflect results of three-group significance tests. ⁺ Parent education reported with 1 = “Grade School”, 2 = “Some High School”, 3 = “High School Equivalent”, 4 = “Regular High school degree”, 5 = “Some College but no degree”, 6 = “Associates Degree”, 7 = “Bachelors degree”, 8 = “Masters, Law, 2–3 years degree”, 9 = “Doctorate, PhD, Medical degree”. ⁺⁺ Household family income reported with 1 = “< 25,000”, 2 = “< 35,000”, 3 = “< 50,000”, 4 = “< 75,000”, 5 = “< 100,000”, 6 = “< 130,000”, 7 = “< 150,000”, 8 = “higher than 150,000”. ⁺⁺⁺ Occupational score according to Nam-Powers-Boyd (Nam and Boyd 2004). *ODD* Oppositional-Defiant Disorder, *CD* Conduct Disorder, *CAARS* Conners Adult ADHD Rating Scale, *BAARS* Barkley Adult ADHD Rating Scale

conceptual meaning. It asks whether low SES accounts for (mediates) ADHD transmission from parent to child. Thus, child ADHD was regressed on SES and on maternal and paternal symptoms, and SES was regressed on maternal and paternal ADHD. The indirect (mediational) path was reliable for paternal symptoms, $\beta_{\text{indirect effect of paternal symptoms}} = .024$, $p = .04$, 95% CI[.001,.05]; while the indirect effect of maternal symptoms was marginal, $\beta_{\text{indirect effect of maternal symptoms}} = .019$, $p = .055$, 95% CI[=.00,.04]. This model fit the data well, $\chi^2(110, N = 931) = 215.50$, $p = .00$, CFI = .98, TLI = .98, RMSEA = .03. These results are consistent with the possibility that that low SES participates in inter-generational transmission of ADHD. This model is depicted in online Fig. S-6.

Robustness Tests To evaluate whether results were attributable to our particular way of measuring family ADHD loading, we re-tested the parent model for both dimensional and

categorical outcomes using several other ways of operationalizing familial risk loading: with the family loading score for (a) all psychiatric disorders and (b) only for ADHD, and for each of these using composite parental symptom scores (average of z scores) that (a) gave equal weight to current symptoms and childhood symptoms, and (b) gave equal weight to all symptom measures, regardless of reference period, all with no change in the story—in fact all effects sizes for SES were larger than in our primary model. These secondary data are provided in Table S-2, online. Thus, how we operationalized parent ADHD or family psychiatric liability did not account for the results.

With child ADHD as a categorical diagnosis (subthreshold excluded, $n = 856$, recall Table 1 for group sizes) the results of our omnibus model (as in Fig. 1) fell shy of significance: the SES-child diagnosis main effect was $\beta = -.09$, $p = .13$, 95% CI[–.21,.03]; OR = 1.39, 95% CI[.90,2.16]. This model fit the

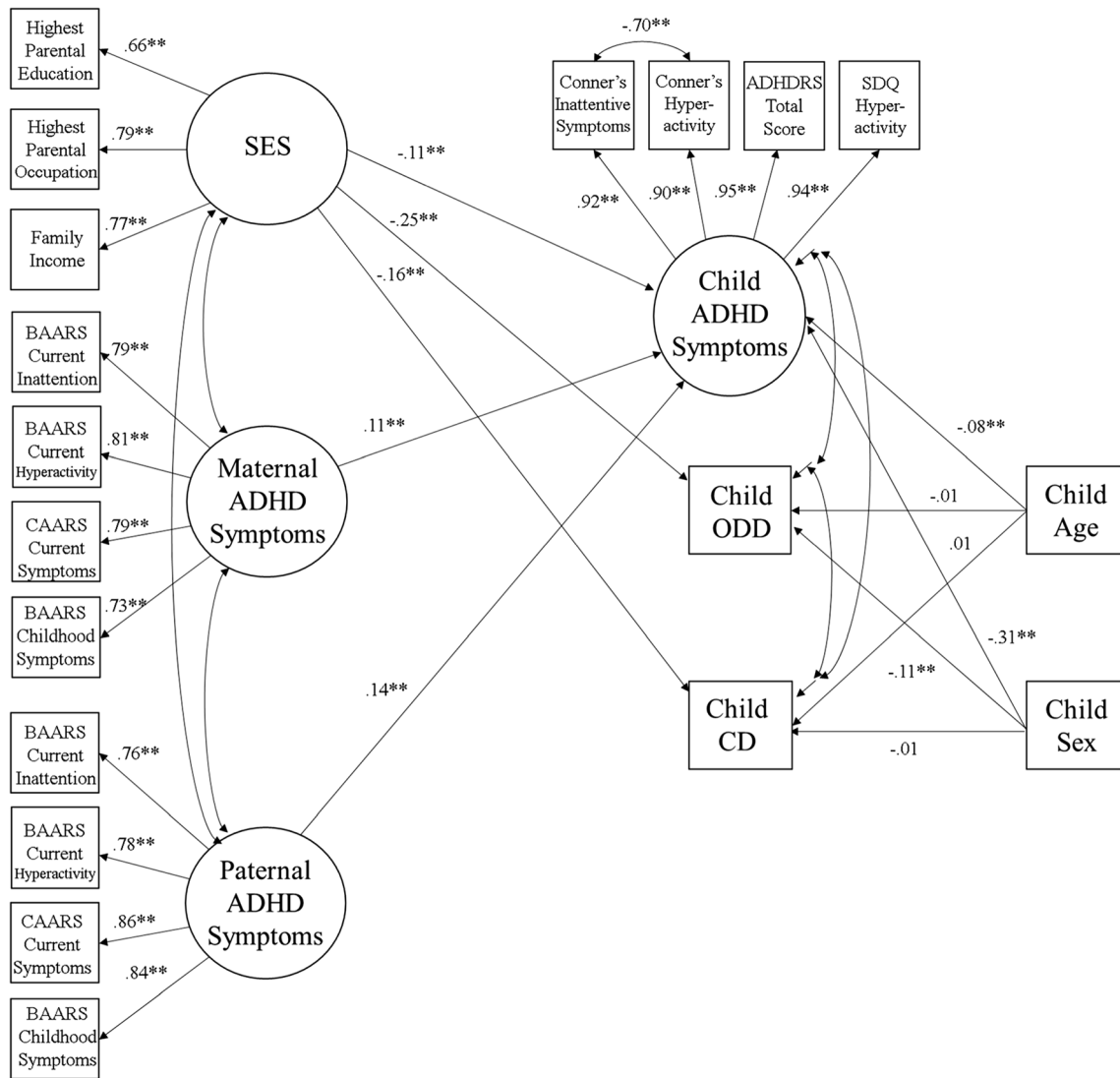


Fig. 1 Study 1: Omnibus Latent Variable Model Relating SES, Parent ADHD Symptomatology, Child ODD and CD, and Teacher-rated Child ADHD Symptomatology. Note: $\chi^2(137, N = 931) = 276.56, p = .00$, CFI = .98, TLI = .97, RMSEA = .03. * $p < .05$, ** $p < .01$. Correlations among the focal variables were estimated as depicted but parameters are not displayed to ease readability. Those correlations among the focal variables (all $p < .001$) were: SES and Maternal ADHD = $-.30$; SES and Paternal ADHD = $-.34$; Maternal ADHD and Paternal ADHD = $.23$; Child ADHD and ODD = $.26$. The correlations among

the residuals of the endogenous variables (all $p < .001$) were: Child ADHD and CD = $.13$; ODD and CD = $.44$. With parent ADHD removed from the model, the main effect of SES on Child ADHD symptoms after controlling for child ODD and CD symptoms (and age and sex) was $\beta = -.18, p < .001$ (95% CI = $-.25, -.11$). When child ODD and CD were removed from the model, the SES-ADHD association controlling for parent ADHD was $\beta = -.09, p = .03$ (95% CI = $-.17, -.01$). See text and supplemental materials for details

data well, $\chi^2(91, N = 856) = 191.80, p = .00$, CFI = .95, TLI = .93, RMSEA = .04. When parent ADHD symptoms were omitted, the results held (main effect of SES on child ADHD, $\beta = -.28, p < .001$; 95% CI [$-.37, -.19$]; OR = 2.90, 95% CI = 1.99, 4.24, $p < .00001$). This model fit the data well, $\chi^2(12, N = 856) = 36.80, p = .03$, CFI = .97, TLI = .93, RMSEA = .05. When parent ADHD was included but child ODD and CD were omitted, the main effect of SES on child ADHD was again marginal: $\beta = -.12, p = .06$; 95% CI = $-.21, .00$; OR = 1.29, 95% CI [1.00, 1.67]. This model fit the data

well, $\chi^2(71, N = 856) = 117.45, p = .03$, CFI = .96, TLI = .95, RMSEA = .03.

Study 1: Summary

Study 1 used a detailed, well supported set of measures of child ADHD, child externalizing, SES, and parent ADHD in local community sample. The results supported the conclusion that low SES is associated with ADHD and that this link is not explained by parent ADHD effects on both SES and child

ADHD, by the artifact of ADHD's overlap with both SES and ODD or CD, or the joint consideration of these effects. These results were robust in the model with dimensional measure of ADHD but fell short of significance if the lower-power categorical diagnosis of ADHD was relied upon as the outcome.

Study 2: Generalizability of ADHD-SES Effect in a National Sample

Study 2 Methods

Sample: NSCH Survey Description This study was conducted using the 2011/2012 iteration of the National Survey of Children's Health (NSCH) (*USA National Survey of Children's Health 2011-2012: Child and adolescent health measurement initiative 2014*). The NSCH is a representative, stratified, nationwide telephone survey that was conducted across the US in 2003/04, 2007/08, and 2011/12. Telephone numbers were called at random to identify households with one or more children under 18 years old. In each household, one child was randomly selected to be the subject of the parent interview. The National Center for Health Statistics of the Centers for Disease Control and Prevention conducts the survey and produces a public-use data set. Two previous studies used the 2003–2004 or 2007–2008 iterations of the NSCH survey to examine the association between poverty level and ADHD status (Kim et al. 2011; Waring and Lapane 2008). One prior study examined that association in the 2011–2012 NSCH survey (Visser et al. 2014). However, ADHD definition was not scrutinized, nor were externalizing problems covaried in those reports.

Analytic Sample A total of 95,677 children of age 0–17 years were included in 2011/2012, but only ages 5–17 were examined here. While ADHD is identified in children under age 5, including them in this analysis was judged to be problematic for three reasons. First, diagnosis of ADHD in this age range has raised concerns due to the difficult overlap of ADHD symptoms with normal behavior in very young children (Connor 2002; Ford-Jones 2015). Second, only a third of children with ADHD are identified during that period (Visser et al. 2015), so we would have many false negatives in that non-ADHD group. Third, prior to 2011 no professional diagnostic guidelines existed for this age group for ADHD, and guidelines then promulgated applied only to ages 4 and 5 (Wolraich et al. 2011), raising further questions about the meaning and accuracy of the reports in the younger age ranges in the 2011/2012 survey. We therefore followed prior convention and limited analyses to age 5–17 (Lingineni et al. 2012; Waring and Lapane 2008), yielding unweighted $N = 70,927$ (48.17% female).

Measures: A. Definition of Socioeconomic Disadvantage Using Poverty Level In study 2, we also followed prior convention by using poverty level as an index of SES disadvantage. Poverty level was determined based on Department of Health and Human Services Federal Poverty guidelines. The original variable had four categories (below 100% federal poverty level [FPL], 100%–199% FPL, 200%–299% FPL, 300%–399% FPL, 400% FPL or greater). To maximize statistical power, we used dichotomized poverty level (POVERTY; <200% vs. $\geq 200\%$). In 2012, 200% of the federal poverty guideline for a family of 4 was an annual income of \leq \$56,000.

Measures: B. ADHD The survey provided three options for describing the child's ADHD, which likely provide varying degrees of validity: (a) Ever ADHD: "For each condition, please tell me if a doctor or other health care provider ever told you that [the selected child] has the condition, even if [he/she] does not have the condition now." Attention deficit disorder (ADD) or ADHD was listed as one condition. Here, with prevalence of nearly 12%, serious questions can be raised as to the validity of all of these as true ADHD cases. Therefore, we note, (b) Current ADHD: "Does [S.C.] currently have [condition]?" Those who were ever told they had ADHD but no longer have it would include cases in which the clinician found an alternative explanation for the problem or the parent simply did not see serious enough problems to be persuaded by the health care provider's opinion, as well as true "recovered" cases. A third check is also available: (c) Severity of ADHD: "Would you describe [his/her] [condition] as mild, moderate, or severe?" A "mild" case would seem more likely to fail to meet DSM-IV diagnostic criteria in a formal evaluation than a moderate or severe case.

To maximize validity, therefore, the analysis was conducted with a three-level ADHD variable (mutually exclusive categories): (a) "not ADHD" (88.4%, $n = 62,458$); (b) "Possible ADHD" (ADHD diagnosed in the past but not current; or currently has mild ADHD; 6.3%, $n = 4469$); or (c) "probable ADHD" (currently, moderate or severe ADHD; 5.2%, $n = 3696$). The prevalence of the probable category (~5%) is congruent with estimates from epidemiological studies that used structured clinical interviews or combined parent and teacher standardized ratings to estimate prevalence of ADHD (Polanczyk et al. 2007), and thus was expected to have the best validity for defining ADHD as defined in our Study 1.

Measures: C. Externalizing Comorbidity We created a 3-level variable for externalizing: (a) "no externalizing" (never diagnosed), (b) "possible externalizing disorder" (ever diagnosed with a conduct/disruptive disorder but does not currently have behavior problems), (c) "probable externalizing disorder" (past and current conduct/disruptive disorder).

Data Analysis and Covariates

The association between ADHD and poverty level was modeled using multinomial logistic regression with the appropriate use of the weighting, ID, and state variables as required for the NSCH data set. Missing data was handled with full information maximum likelihood estimation, in *Mplus*, as in Study 1. Montecarlo integration was necessary to estimate the model given the multinomial outcome and presence of missing data. The resulting parameter estimates were based on the full sample of 70,927 except for the completely unadjusted model which was based on a sample of 70,862 (65 cases were missing on the poverty variable). We estimated an unadjusted model, an adjusted Model 1 for demographic variables (sex, age, race/ethnicity), and an adjusted Model 2 for demographic variables plus conduct/disruptive behavior disorder. We report odds ratios and 95% confidence intervals. Statistical significance was set at $p < .05$.

Study 2 Results

Table 2 provides the results of the multinomial logistic regression models for the base model and the adjusted models. It shows that in the model adjusted only for sex and age, when children were in families with income at <200% FPL, they were more likely to have “moderate/severe” or “probable” ADHD than no ADHD, OR = 2.13, 95% CI[1.79, 2.54], $p < .001$. Adjusting also for conduct/disruptive behavior disorder, the effect remained reliable, OR = 1.61, 95% CI [1.32, 1.96], $p < .001$. This corresponds to a Cohen’s $d = .26$. The effect of poverty level on “possible ADHD” was smaller and not independent of disruptive behavior problems.

Study 2: Summary

Study 2 replicated the Study 1 externalizing finding using a nationally representative data set. The result is consistent with the conclusion that the association of poverty status with ADHD exists independently of the overlap of SES with both ADHD and disruptive behavior problem diagnoses. It replicates that subset of the Study 1 finding and shows the result is generalizable.

General Discussion

The association of socioeconomic disadvantage with psychopathology and neurodevelopmental disorders has gained renewed importance in light of growing understanding of the broad impacts of social class on child development (American Psychological Association 2007), interest in the social

determinants of health and mental health, insights into neural development and its dependence on early enrichment both socially and biologically (Bowles et al. 2009; Van Praag et al. 2000), and by fresh appreciation that environmentally-responsive epigenetic mechanisms mediate life stress effects on behavior (Champagne and Curley 2005; McEwen et al. 2012; Roth 2012; Thayer and Kuzawa 2011). Low SES (or its proxies such as parental education or income) is associated with life stress and neighborhood disadvantage as well as developmental health risks such as lower cognitive ability and educational outcomes (American Psychological Association 2007; Bradley and Corwyn 2002; Evans 2004).

What has been unclear is whether ADHD is somehow an exception to all of this. The present findings are important because they move a good distance toward resolving that prior ambiguity. Study 1 confirmed using state-of-the-art research evaluation methods that ADHD is associated with carefully defined lower SES, in a community recruited sample after adjusting for the overlap of SES and ADHD with comorbid ODD or CD. Study 2 provided replication and generalizability study in a national sample, despite different methods, ascertainment, and measures. The effect size replication was notable. In study 1, the effect size for the model adjusting for the overlap of SES, child ADHD, and ODD and CD (but ignoring parent ADHD) ($\beta = -.18$) is equivalent to OR = 1.97 (or Cohen’s $d = .37$). In study 2, the analogous model had OR = 1.61 ($d = .26$). The meta-analysis (Russell et al. 2016) suggested a population effect size of OR = 1.8–2.2 across a range of indices uncorrected for any other variables. The present results are broadly in line with that figure when considering our adjustment for confounds that Russell et al. did not adjust for.

This convergence in results and effect sizes with these additional statistical controls provides compelling evidence that in the United States, low SES is associated with ADHD specifically, not merely due to the overlap of SES with both ADHD and disruptive behavior disorders, or due to the overlap of SES with a shared liability factor underlying externalizing. This overall picture supports prior conclusions and underscores the value of further study of mediators and moderators relevant to ADHD itself and not just for associated externalizing problems. The statistical association of SES and ADHD reported here is small by Cohen’s conventions. However, it is comparable to other important risk factors for ADHD and other important effects on public health. Such a statistical effect size may be of substantial public health significance due to the large number of children who are affected by social disadvantage.

The second key issue we studied was whether parent ADHD could explain the SES-ADHD link (Russell et al. 2015). We could only test this in Study 1, but there we provided the best direct test to date of whether a putative

Table 2 Study 2: Odds ratios of ADHD status among children aged 5 to 17 years from the National Survey of Children’s Health 2011–2012

Model	Unadjusted	Adjusted for demographics	Adjusted for demographics and behavioral problems
	OR (95% CI) Reference group	OR (95% CI) Reference group	OR (95% CI) Reference group
No ADHD ever			
Past only/current mild ADHD	.98 (.85, 1.13)	1.22 (1.03, 1.44)	1.08 (.92, 1.29)
Moderate/severe ADHD	1.73 (1.48, 2.03)	2.13 (1.79, 2.54)	1.61 (1.32, 1.96)

Note to Table 2. The primary ADHD outcome was considered present (“probable”) if ADHD was current, and also was coded moderate or severe. Cases coded as ADHD but not current (past only), or as ADHD but mild, were considered “possible” ADHD or “subthreshold” ADHD conceptually

low SES -ADHD association is explained by parent ADHD (a partial proxy for a genotype-environment correlation). The parent ADHD latent variables only partially explained the SES effect on child ADHD. The adjusted association of low SES and child ADHD was again small by Cohen’s convention. We can conclude that parent ADHD contributes to both child ADHD and lower SES, but that low SES has a small additional statistical association with child ADHD.

The parent ADHD result, when taken together with the sibling-based, causally-informative findings of Larsson et al. (2014) in their study in Sweden, is consistent with a causal link between low SES and ADHD. However, our cross-sectional study cannot directly inform causality. Because we could not assess parent ADHD in the national survey, we do not know the generalizability of that finding.

Despite the strength of the present findings, other limitations must be considered. Although we had multiple measures of parent ADHD, all were self-report or parental recalled childhood ADHD symptoms. A complete evaluation of parent ADHD would have included other informants. Perhaps for this reason as well, source-variance confound may explain why the secondary models using child ADHD as a categorical measure failed to detect an SES-ADHD effect. Further, potential variation across race, culture, or national context was not evaluated here (although statistical covarying of race/ethnicity did not change results).

What if this association reflects a causal influence, as implied by Larsson et al. (2014)? What might an influence of social context on ADHD mean in terms of an integrated formulation about ADHD risk and etiology? Here we venture a speculative comment to guide future work. Despite ADHD’s substantial heritability (Chang et al. 2013; Faraone and Mick 2010), effects of socio-economic context could be embedded in epigenetic (or gene by environment interaction) effects (Foulon et al. 2015), potentially even moderating heritability. To this last point, at least in some countries socioeconomic status appears to moderate the heritability of IQ (Hanscombe et al. 2012; Turkheimer et al. 2003) and of brain development (Hackman et al. 2010). This is untested for ADHD. It is possible that a negative recursive cycle occurs in which adults

with ADHD experience some degree of socio-economic setback (Barkley et al. 2010) which then amplifies the expression of ADHD in their offspring.

Integrated models of genetic and social contexts are emerging in relation to health and illness (Thayer and Kuzawa 2011). Epigenetic mediation of SES-associated stressor effects on child ADHD is an interesting but as yet speculative possibility (Nigg 2012, 2016). Social disadvantage may entail a context in which physical and social stressors contribute to physical and mental health conditions, particularly for developing children (Santiago et al. 2011). To date, the study of mediators has emphasized breakdowns in parenting and other social mediators (Russell et al. 2015). However, a widened study of mediators is warranted. Several risk factors are correlated with lower SES, such as poor diet and higher exposure to pollutants, that are associated with ADHD or altered child attentional development (Liston et al. 2009; Liston et al. 2006; Nigg 2016).

Particularly promising could be further consideration of allostatic load (Blair et al. 2011; Ganzel et al. 2010; McEwen 2015; Rogosch et al. 2011). That hypothesis suggests that the cumulative effect of exposure to stressful events creates a context in which both physical and social stressors build on one another and contribute to further stress (McEwen 2007), and to neuroendocrine and cardiovascular dysregulation (Brosschot et al. 2005). According to this theory, the elasticity and resiliency of one’s biological systems are reduced and respond to challenges less robustly and recover less efficiently (McEwen and Gianaros 2010). In particular, this idea has been extensively developed in relation to gene by environment studies that consider sensitivity to experiences “for good or for ill” as one feature of genetic variation that accounts for differential response to environments (Pluess 2017; Pluess and Belsky 2013).

Conclusion

As the association of socio-economic context with ADHD becomes established, understanding of mechanisms becomes

paramount (Froehlich et al. 2007; Russell et al. 2015). Socioeconomic disadvantage embeds a constellation of risk factors, many of which may influence ADHD. This is not to overlook the importance of patterns of resilience, as well as the historical, political, and cultural context embedded in the manifestation of poverty. Explanations and interventions ultimately will have to consider not only individual characteristics but structural landscapes that produce and sustain socioeconomic disadvantage (Mills 2015).

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Compliance with Ethical Standards

Conflict of Interest The authors declare no conflicts of interest relevant to this work.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Written informed consent was obtained from all parents participating in the study, one parent provided written informed consent for all children participating in the study, and written informed assent was obtained from all participating children.

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