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# ADHD and risk for subsequent adverse childhood experiences: understanding the cycle of adversity

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## Abstract

**Background:** Children with adverse childhood experiences (ACEs) are more likely to develop Attention-Deficit/Hyperactivity Disorder (ADHD). The reverse relationship – ADHD predicting subsequent ACEs – is vastly understudied, although it may be of great relevance to underserved populations highly exposed to ACEs.

**Methods:** Participants were 5- to 15-year-olds (48% females) with (9.9%) and without ADHD (DSM-IV criteria except age of onset) in a longitudinal population-based study of Puerto Rican youth. In each wave (3 yearly assessments, W1–3), ten ACEs (covering parental loss and maladjustment and child maltreatment) were examined, plus exposure to violence. Logistic regression models examined ADHD (including subtypes) and subsequent risk for ACEs. Also considered were interactions by age, sex, number of W1 ACEs, and recruitment site.

**Results:** Children with W1 ADHD were more likely to experience subsequent adversity (OR: 1.63; 95% CI: 1.12–2.37) accounting for child age, sex, public assistance, maternal education, site, disruptive behavior disorders, and W1 ACEs. Inattentive (OR: 2.00; 95% CI: 1.09–3.66), but not hyperactive/impulsive or combined ADHD, predicted future ACEs.

**Conclusions:** ADHD predicts subsequent risk for ACEs, and the inattentive presentation may confer the most risk. Inattentive presentations could pose a bigger risk given differences in symptom persistence, latency to access to treatment, and treatment duration. The present study suggests a pathway for the perpetuation of adversity, where bidirectional relationships between ADHD and ACEs may ensnare children in developmental pathways predictive of poor outcomes. Understanding the mechanism underlying this association can help the development of interventions that interrupt the cycle of adversity exposure and improve the lives of children with ADHD.

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**Keywords**

Attention-deficit; hyperactivity disorder; adverse childhood experiences; adversity; Boricua Youth Study

## Introduction

Sixty percent of adults report having at least one Adverse Childhood Experience (ACE) before they turn 18, and about 25% will have three or more (Merrick, Ford, Ports, & Guinn, 2018). ACEs encompass parental loss (death, divorce/separation), maltreatment (sexual, physical, emotional abuse, or neglect), and parental maladjustment (intimate partner violence, incarceration, mental illness, substance use). ACEs are more frequently experienced by individuals from disadvantaged populations, for whom witnessing or being a victim of violence, and experiences of racism/discrimination may be additional substantial sources of adversity (Cronholm et al., 2015). The detrimental long-term consequences of ACEs on physical and mental health are well documented (Hughes et al., 2017) with a substantial body of literature linking ACEs to increased likelihood of developing attention-deficit/hyperactivity disorder (ADHD). Although more than one hypothesized mechanism exists, it is believed that adversity-related toxic stress may impact brain development, including regions implicated in ADHD symptomatology. A recent national cross-sectional study supports this possibility, documenting a dose–response association where the number of accumulated ACEs predicted number of ADHD symptoms and severity (Brown et al., 2017).

However, the reverse association – ADHD leading to ACEs – has received far less attention. It is possible that children who are impulsive, lack self-regulation, and self-awareness – qualities that often characterize ADHD – would be more likely to experience ACEs. While three population-based studies have documented higher levels of ACEs in individuals with ADHD, both ADHD and ACEs were measured via retrospective self-reports in adulthood (Fuller-Thomson & Lewis, 2015; Fuller-Thomson, Mehta, & Valeo, 2014; Ouyang, Fang, Mercy, Perou, & Grosse, 2008). Retrospective ADHD self-reports are suboptimal in terms of accuracy and reliability. Most importantly, the use of retrospective ADHD and ACE self-reports precludes studies from establishing a clear temporal sequence determining whether ADHD symptoms were present before or only after the occurrence of ACEs.

Only one population-based study has explicitly tested whether childhood ADHD places children at higher risk for subsequently experiencing ACEs using a prospective, longitudinal design (Stern et al., 2018). In this study of British twins, Stern and colleagues documented that participants with childhood ADHD had increased risk of experiencing abuse and neglect by young adulthood. However, this study was not focused on children highly exposed to adversities and also did not examine a comprehensive set of ACEs, restricting analyses to maltreatment and victimization (Stern et al., 2018). Building on this initial study to examine a more complete set of ACEs is of paramount importance, as ACEs tend to co-occur, particularly in disadvantaged populations (Hughes et al., 2017; Ramos-Olazagasti, Bird, Canino, & Duarte, 2017). For example, it is possible that children's' ADHD symptoms

could contribute to household conflict and result in other ACEs like parental loss (divorce/separation) or maladjustment (intimate partner violence).

Smaller studies of selected samples also support a link between childhood ADHD and risk for subsequent ACEs, yet consist of (a) retrospective self-reports of ACEs in adult samples, (b) cross-sectional pediatric studies, and (c) samples with another presenting problem. Of note, two of such studies have failed to document elevated ACEs in participants with ADHD (Ford et al., 1999; Wozniak et al., 1999). However, both studies utilized clinical samples actively undergoing ADHD treatment and were further limited by the exclusion of females (Wozniak et al., 1999) and lack of a control group (Ford et al., 1999). Moreover, neither assessed nor controlled for pre-existing ACEs, precluding both from determining if ADHD was present before their measured ACEs.

The existing body of literature is also critically limited by its failure to include the children at highest risk for ADHD and ACEs (Merrick et al., 2018). Socioeconomically disadvantaged children have higher prevalence of ADHD and ACEs. Racial/ethnic minorities might also be at increased risk. Specifically, Latinx populations in the US have more ACEs than their non-Latinx white counterparts (Hughes et al., 2017). A national survey of 84,837 children in the US documented that only 42% of Latinx children did not have an ACE (54% in non-Latinx white children) and 31% had 2 or more (21% in non-Latinx white children; (Slopen et al., 2016)). Simultaneously, the last decade has seen a disproportionate increase in ADHD diagnoses among Latinx populations in the US, with a study documenting an 83% increase from 2003 to 2011 (Collins & Cleary, 2016). Yet, most study samples have failed to include these high-risk populations. Given the already elevated prevalence of ACEs, increasing trend in ADHD diagnoses, and comparatively low rate of ADHD service use in Latinx populations in the US (Bird et al., 2008), it is of great importance to examine if a link to subsequent ACEs exists in this population. Studies have also been limited by not consistently considering the potentially confounding role of disruptive behavior disorders (DBDs), which most frequently co-occur with childhood ADHD and could explain the association. Finally, the few studies that have examined ADHD subtype differences have reported mixed findings, one study showing stronger associations between inattentive subtype and ACEs (Ouyang et al., 2008), and two finding no differences across ADHD subtypes (Schilpzand et al., 2018; Stern et al., 2018). Given the well documented relationship between ACEs and subsequent risk for ADHD, investigating the reverse relationship is critical to ascertaining whether there is a cyclical nature to these two debilitating childhood hardships.

The present study aims to address significant gaps in the literature and inform prevention strategies by examining if children with ADHD are at an increased risk for ACEs in a prospective, longitudinal, population-based study of Puerto Rican children and adolescents in the South Bronx, New York, and Puerto Rico. By leveraging prospectively collected ADHD and ACEs assessments, this study is uniquely able to examine whether ADHD at an initial time point predicts the subsequent experience of ACEs, while adjusting for pre-existing baseline ACEs in an underprivileged population at high risk for ACEs. Further, this study aims to address existing gaps by accounting for the role of comorbid DBDs and examining ADHD subtype differences.

# Methods

## Participants

The Boricua Youth Study (BYS) is a study of two representative longitudinal probability samples of Puerto Rican children and their parents ($N$ = 2,491). Families were recruited from two sites, the South Bronx, New York (SBx) and the metropolitan areas of San Juan and Caguas, Puerto Rico (PR). Description of sampling strategies and study design are detailed elsewhere (Bird et al., 2006). Families were included if one caretaker self-identified as Puerto Rican, and there was at least one child ages 5–13 at the time of contact and excluded if they presented with severe developmental delays. Families were included in the current analysis if they participated across all three Waves, resulting in a total sample of 2,134 children (see Table 1; SBx $N$ = 932, PR $N$ = 1201). Children (male $N$ = 1,102, female $N$ = 1,031) were 5–15 years of age at Wave 1 (Mean = 9.48 years, $SD$ = 2.58).

## Procedures

Participants (children and a caretakers) provided consent/assent. The Institutional Review Boards of the New York State Psychiatric Institute and the University of Puerto Rico Medical School approved all procedures. Participants were interviewed in their homes and followed for three Waves of data collection, one year apart each between 2000 and 2004. The BYS achieved excellent retention rates (W2: 92%, W3: 88%; Bird et al., 2007). Interviews were conducted by trained interviewers in the participant's preferred language (English or Spanish).

## Measures

**Attention-deficit/hyperactivity disorder.—**The presence of ADHD was assessed at each Wave using the NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) in either English or Spanish. The DISC-IV is a structured diagnostic interview that assesses pediatric psychiatric disorders. Caretaker interviews were used as parental reports of externalizing disorders show higher test-retest reliability than child self-reports ($\kappa$ = 0.79 vs. 0.42)(Shaffer et al., 2000). To optimally investigate the study hypothesis, children were considered to have ADHD if they fulfilled DSM-IV criteria for: six symptoms or more of hyperactivity/impulsivity or inattention, clinically significant impairment, and impairment present in more than one environment. The age of onset criterion was not included because studies have shown similar impairment and comorbidity profiles in children diagnosed with ADHD before and past the age of 7 (Rohde et al., 2000) and has been recently revised in DSM-5 (Lin, Lo, Yang, & Gau, 2015).

**Adverse Childhood Experiences.—**A detailed description of the measures has been previously reported (Ramos-Olazagasti et al., 2017). ACEs were measured in each Wave with consistent instruments. In brief, 11 adversities covering four domains were examined: child maltreatment (neglect and physical, sexual, and emotional abuse), parental maladjustment (intimate partner violence, antisocial personality/ parental arrest/jail, substance use problems, and emotional problems), exposure to violence, and parental loss (death, divorce/separation). Children under the age of 10 were not asked to report about their

experiences with racism/discrimination; this ACE was thus not included in the analyses. Child maltreatment variables were derived from the Conflict Tactics Scale and the Sexual Victimization Scale. Clinical interviews and the Family Psychiatric Screening Instruments for Epidemiologic Studies were used to compile parental maladjustment variables (Lish, Weissman, Adams, Hoven, & Bird, 1995). Demographic interviews were used to create the parental loss variables. Richters' exposure to violence scale was used (Richters & Martinez, 1993). Child self-reports were used to create the exposure to violence variables, and caretaker reports were used for parental maladjustment and parental loss variables. Both parent and child reports were used to create child maltreatment variables. The presence or absence of each ACE was coded as a binary variable. Refer to Ramos-Olazagasti (Ramos-Olazagasti et al., 2017) for sample items and instrument details. Whereas Wave 1 assessments asked about having ever experienced the ACE, Wave 2, and 3 assessments inquired about the occurrence of each ACE since the last interview. Wave 2 and 3 ACEs are thus measures of new incident ACEs following Wave 1, and not a cumulative count.

**Disruptive behavior disorders.—**The presence of DBDs was also measured via the NIMH DISC-IV(Shaffer et al., 2000) and its Spanish version based on parental reports. DBDs include oppositional defiant disorder (ODD) and conduct disorder (CD). Meeting criteria for either ODD or CD was enough to be coded as having a DBD.

**Covariates.—**Caretaker reports of child sex, age, household use of public assistance, study recruitment site (South Bronx, NY or San Juan, PR), and maternal education at Wave 1 were used as covariates.

## Data analysis

To examine whether Wave 1 ADHD conferred increased risk for subsequent ACEs, logistic regressions were conducted using SURVEY procedures in SAS (Cole, 2001). Logistic regression models included site-specific sampling weights (on average subjects from two sites are weighted equally) as well as stratum and cluster variables. The outcome measure was having any new (i.e., not previously experienced) ACE at either Waves 2 or 3 (vs not having any new ACEs in either Wave). Adjusted models controlled for Wave 1 site of recruitment (SBx or PR), age, DBD, public assistance, W1 ACEs and child sex. Further models examined risk for ACEs separately for each ACE domain (e.g., child maltreatment vs. parental loss). ADHD subtype differences were also tested. The potentially moderating role of child age, sex, and recruitment site (SBx vs PR) was examined with interaction models. Models also examined interactions between number of baseline ACEs (0 vs 1, 2, or 3 or more) and risk for subsequent ADHD (examined both as presence vs. absence of study-defined ADHD, as well as presence vs. absence of Inattentive vs. Hyperactive, vs. Combined ADHD subtypes). Sensitivity analyses are detailed below and examined the effect of children without ADHD at W1 subsequently developing ADHD in Wave 2 or 3 by re-running the main model while excluding children who met the study-defined ADHD criteria at Wave 2 or 3.

## Results

### ADHD by sociodemographic characteristics and number of ACEs at Wave 1

The characteristics of children with and without W1 ADHD are reported in Table 1. At W1, 213 (~10%) children met the study-defined ADHD criteria, henceforth referred to as 'ADHD'. Prevalence of W1 ADHD was higher among males (13.1% males, 5.5% females), and children with W1 ADHD were more likely to meet diagnostic criteria for DBD (69.2% ADHD, 5.7% non-ADHD). At W1, children with ADHD were more likely to present with ACEs. Only 20.3% of all children in the sample did not report having experienced an ACE at W1, and half reported experiencing 2 or more. Common W1 ACEs included parental divorce/separation (58.0% ADHD, 49.8% non-ADHD) and having a parent with an emotional problem (38.3% ADHD, 24.5% non-ADHD). Table S1 shows prevalence of ACEs across Waves by ADHD W1 status.

### ADHD at Wave 1 predicting new ACEs at Waves 2 or 3: logistic regression

Model 1 (Table 2) provides odds ratios (ORs) and 95% confidence intervals (CIs) for the association between W1 ADHD and new ACEs at W2 and W3. Wave 1 ADHD was associated with increased odds (adjusted OR 1.62, 95% CI 1.12–2.36, C index 0.587) of experiencing new ACEs in W2 and W3. When risk for ACEs was examined separately for each ACE domain, only risk for a new occurrence of parental maladjustment at W2 and W3 was significantly predicted by W1 ADHD (adjusted OR 1.73, 95% CI 1.03–2.91; Table S2).

As a sensitivity analysis, in order to account for the fact that some children may have not yet developed or reported ADHD in W1, but subsequently meet criteria in W2 or W3, analyses were re-run excluding participants that did not meet the study-defined ADHD criteria at W1, but did so at either W2 or W3. This model showed similar results (Table S3), where having W1 ADHD was associated with increased odds of W2 and W3 ACES (adjusted OR 1.84, 95% 1.26–2.69). Further sensitivity analyses examined the association between W1 ADHD and ACES in children with no W1 ACEs (i.e., excluding children with W1 ACEs from analysis). Although this model (Table S4) was not significant (OR 1·12, 95% 0.40–3.09), the effect was in the same direction as the main model, even when these analyses were so underpowered (W1 ADHD n = 18).

ADHD subtype analyses showed that the inattentive (OR 2.00, 95% 1.09–3.66), but not the hyperactive-impulsive (OR 1.47, 95% 0.86–2.50), or combined (OR 1.53, 95% 0.77–3.03) subtypes at W1 were predictive of increased W2 and W3 ACEs (Model 2, Table 2). Analyses did not find any significant interactions between W1 ADHD and age ($p = .50$), sex (0.13), site ($p = .61$). Number of baseline ACEs did not interact with baseline ADHD (tested both as presence vs. absence of ADHD, $p = .33$ and as absence vs. inattentive vs. hyperactive-impulsive vs. combined subtypes $p = .17$) in predicting W2 and W3 ACEs.

## Discussion

This is the first study to test whether ADHD increases the likelihood of experiencing ACEs in a longitudinal, probability-based sample of children highly exposed to ACEs. Findings document that ADHD predicts increased likelihood of accruing subsequent ACEs and

that inattentive ADHD subtype predicted ACEs. Given the longitudinal prospective study design employed with three recurrent ADHD and ACEs assessments, this study was able to establish a clear temporal sequence that strongly indicates that the ACEs considered in the analyses occurred after ADHD symptoms were present. Analyses also controlled for initial ACEs and child disruptive behavior disorders. In doing so, stronger inferences about ADHD having a role in increasing risk of exposure to subsequent ACEs can be made. Given the well-documented association between ACEs and subsequent risk for ADHD, this study points to the existence of a cycle that perpetuates hardship and adversity in the most underserved populations.

The finding that inattentive ADHD subtype more strongly predicts ACEs, although at first may seem counterintuitive, is in line with a prior study (Ouyang et al., 2008). As Ouyang and colleagues note, the nature of inattentive symptoms themselves could underlie this association. Studies have found inattentive subtypes to be less likely to be detected (Sayal, Goodman, & Ford, 2006), and to be associated with later initiation and shorter duration of treatment (Barbaresi et al., 2006). Further, inattentive symptoms more frequently persist into adulthood, showing remittal rates lower than hyperactive/impulsive symptoms (Biederman, Mick, & Faraone, 2000). Longer persistence of symptoms, especially if untreated, might give more opportunities for snowballing effects. Interestingly, ADHD predicted risk for ACEs, even when accounting for co-occurring disruptive behavior disorders. Although the present study did not examine the contribution of disruptive behavior disorders independently of ADHD symptoms, findings suggests that the cluster of behavioral symptoms most classically characterized as ‘externalizing’ are not the most critical in predicting risk for future ACEs.

ADHD most strongly predicted risk for new incidences of parental maladjustment ACEs, highlighting the need for studies to include comprehensive ACE assessments. Parental maladjustment ACEs included psychopathology, intimate partner violence, arrest/jail, and substance use. Because analyses controlled for previously accrued parental maladjustment ACEs, results capture ADHD-related risk for new incidences of parental maladjustment, suggesting child ADHD may be involved in the proliferation of adversity. In line with this finding, parents of children with ADHD have documented higher rates of psychopathology, particularly depression and anxiety (Margari et al., 2013). Parental mental health might be particularly important to consider in interventions, as studies have found that among parents of children with ADHD, parents with depressive symptoms are more likely to employ negative parenting practices (Shin & Stein, 2008). Aside from being an ACE in itself, parental psychopathology may thus have cascading effects. To aid prevention and early intervention efforts, future studies should examine which specific parental mental health diagnosis has the biggest impact in the prognosis of children with ADHD, particularly as it pertains to risk for future ACEs.

Familial risk for ADHD could also explain risk for ACEs. ADHD shows strong genetic heritability, suggesting that many of the parents in the sample could have ADHD themselves. This could explain links between child ADHD and parental risk for arrest/jail and substance use, as ADHD has been found to confer long-term risk both (Lee, Humphreys, Flory, Liu, & Glass, 2011; Mannuzza, Klein, & Moulton, 2008). Parental

ADHD also predicts negative parenting practices (Park, Hudec, & Johnston, 2017), which in turn have been found to mediate parent-child ADHD transmission (Tung, Brammer, Li, & Lee, 2015). This suggests the existence of a cyclical effect in which child symptoms contribute to negative parenting, which in turn exacerbates child symptomatology. Finally, children's ADHD has been found to predict chaos in the home and marital conflict (Schermerhorn et al., 2012). Conflict may result in parents engaging in intimate partner violence, which has devastating consequences on the family unit and children's development.

The findings suggest ADHD may have a role in perpetuating the cycle of childhood adversity and highlight the dire need for treatment. Because ADHD treatment is usually followed by symptom reductions, ADHD treatment may be a viable ACE prevention strategy, yet this possibility remains an open question. However, given the well-documented disparities in Latinx's ADHD service use (Eiraldi & Diaz, 2010), and negative parental attitudes toward ADHD medication (Bird et al., 2008), finding alternative and complimentary areas for intervention is critical. Importantly, the findings strongly underscore the need for parent-centered interventions, may it be in the form of mental health and substance abuse services, or parenting support. Considering and integrating the sociocultural values that are commonly held within Puerto Rican communities can impact the effectiveness of interventions. Previous research has detailed how to integrate values such as *familism* and *personalism* into interventions, for example by providing parent psychoeducation, allowing the family greater participation in defining their problems and needs, jointly developing interventions goals that are congruent with the family's expectations, addressing family emotional involvement, and when appropriate, engaging extended family members that play important roles within the unit (Bernal et al., 2019; Parra Cardona et al., 2012; Zayas & Palleja, 1988). The generalizability of our findings to other Latinx and other racial/ethnic minority communities within the US requires future examination.

Results should be interpreted in the context of limitations. Child ethnic discrimination was not included in the ACE assessments and other indicators of socioeconomic status (e.g., occupation and income) need to be considered to more fully understand how socioeconomic class impacts the relationship between ADHD and risk for ACEs. Future studies should examine the impact of treatment on later ACEs, while carefully accounting for the factors that influence service use (e.g., access to care). Further, the role of parental perceptions and/or knowledge of their children's ADHD symptomatology, particularly inattentive symptoms, as being a part of a medical disorder should be examined. For one, parental perceptions are likely to have implications for seeking treatment. Further, it is possible that parents who either know their children have an ADHD diagnosis or can identify symptoms as a part of a medical disorder may be more protective or accommodating of the behaviors, possibly assuaging risk for future ACEs. It is also possible that unmeasured factors that increase both risk for ADHD and ACEs underlie the findings. Studies with larger ADHD samples should examine whether sex and ADHD subtype interact in predicting ACEs. Although subtype prevalence differences across sex are thought to contribute to the lower rates of referrals and treatment for females, gender biases impact referral rates, independently of ADHD subtype. Thus, females with inattentiveness might be at the highest

risk of being overlooked, and by consequence, for future ACEs. Finally, future studies could consider expanding on the conceptualization of ACEs to include other aspects such as peer victimization and bullying.

In conclusion, this study documents that a childhood diagnosis of ADHD predicts sequent occurrence of ACEs and that the inattentive subtype may be most influential in this risk. Findings have significant implications for clinical practice and prevention efforts. First, they suggest that not only do ACEs pose risk for subsequent ADHD, but that ADHD itself might be an ACE risk factor. Enough is known about the long-term sequelae of both ACEs and ADHD individually to predict that children ensnared in an ADHD-ACE cycle are at high risk for negative social, emotional, and health outcomes. To prevent further ACEs, clinicians and physicians working with children with ADHD should assess for past ACEs and attend to signs of disturbance in their patients' home and school life, directly gauging the presence of intimate partner violence and being attentive to any signs of parental mental health or substance use that may merit intervention. Whereas these practices should be commonplace among any professionals in contact with pediatric populations, this study suggests these practices may be particularly critical in improving the lives of children with ADHD. Above all, these findings echo the growing number of studies that suggest ACEs are a serious public health concern that calls for focused prevention efforts.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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**Key points**

- Children with Adverse Childhood Experiences (ACEs) are at increased risk for ADHD.
- Only one longitudinal population-based study has examined the inverse association, whether ADHD increases risk for subsequent ACEs. This study examined only maltreatment and victimization and did not focus on an at-risk population, yet found ADHD predicted risk of abuse and neglect.
- The present study is the first to document increased risk for subsequent ACEs, specifically parent maladjustment, among children with ADHD in a sample of children highly exposed to ACEs.
- Specifically, Inattentive ADHD subtype predicted ACEs.
- ACEs and ADHD may form part of a cycle that perpetuates hardship and adversity. It is critical to develop interventions that halt this cycle and steer children out of negative developmental trajectories.

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**Table 1**

ADHD by sociodemographic characteristics and number of ACEs at Wave 1

| | | | ADHD at Wave 1 Yes (*N* = 213) | | | | |
|---|---|---|---|---|---|---|---|
| **Wave 1 characteristics** | **Total N** | **Weighted %** | **n** | **Weighted row %** | **OR** | **CI** | ***p*** |
| Site | | | | | | | |
| Bronx | 932 | 43.81 | 89 | 9.37 | Ref | | |
| Puerto Rico | 1,201 | 56.19 | 124 | 9.38 | 1.00 | (0.70–1.42) | .9973 |
| Sex | | | | | | | |
| Male | 1,102 | 51.27 | 151 | 13.10 | Ref | | |
| Female | 1,031 | 48.73 | 62 | 5.45 | 0.38 | (0.27–0.54) | <.0001 |
| Age | | | | | | | |
| 5–9 | 1,040 | 52.75 | 98 | 8.86 | Ref | | |
| 10–15 | 1,093 | 47.25 | 115 | 9.95 | 1.14 | (0.82–1.58) | .4448 |
| Disruptive behavior disorders | | | | | | | |
| No | 2,009 | 94.26 | 124 | 5.73 | Ref | | |
| Yes | 124 | 5.74 | 89 | 69.20 | 36.92 | (23.06–59.11) | <.0001 |
| Public Assistance | | | | | | | |
| No | 1,260 | 59.17 | 115 | 8.44 | Ref | | |
| Yes | 863 | 40.83 | 97 | 10.70 | 1.30 | (0.91–1.85) | .1429 |
| Maternal education | | | | | | | |
| Less than HS/GED | 632 | 29.68 | 58 | 8.05 | Ref | | |
| HS/GED or more | 1,489 | 70.32 | 154 | 9.94 | 1.26 | (0.89–1.79) | .1891 |
| Number of ACEs at baseline | | | | | | | |
| 0 | 416 | 20.30 | 18 | 4.23 | Ref | | |
| 1 | 612 | 29.36 | 44 | 6.85 | 1.66 | (0.93–2.98) | .0876 |
| 2 | 470 | 21.31 | 54 | 11.14 | 2.84 | (1.54–5.22) | .0009 |
| 3 | 285 | 12.89 | 38 | 12.35 | 3.19 | (1.72–5.91) | .0003 |
| 4 | 189 | 8.95 | 24 | 11.57 | 2.96 | (1.47–5.96) | .0025 |
| 5 or more | 148 | 7.18 | 32 | 19.43 | 5.46 | (2.85–10.47) | <.0001 |

ACEs, adverse childhood experiences; ADHD, attention-deficit/hyperactivity disorder; GED, graduate equivalency degree; HS, high school.

**Table 2**

ADHD at Wave 1 predicting New ACEs at Waves 2 or 3: logistic regression

| | | | New ACEs at W2 or W3 | | | | |
|---|---|---|---|---|---|---|---|
| | | | Yes (N = 897) | | Adjusted model | | |
| **Wave 1 characteristics** | **Total *N*** | **Weighted %** | **n** | **Weighted row %** | **OR** | **CI** | ***p*** |
| Model 1: Presence/absence of ADHD | | | | | | | |
| No | 1,898 | 90.67 | 781 | 41.49 | Ref | | |
| Yes | 210 | 9.33 | 112 | 54.22 | 1.63 | (1.12–2.37) | .0110 |
| Model 2: ADHD subtype analyses | | | | | | | |
| None | 1,898 | 90.67 | 781 | 41.49 | Ref | | |
| Inattention type | 55 | 2.46 | 31 | 59.85 | 2.00 | (1.09–3.66) | .0244 |
| Hyperactive/impulsive type | 89 | 3.93 | 47 | 51.04 | 1.47 | (0.86–2.5) | .1581 |
| Combined type | 66 | 2.94 | 34 | 53.77 | 1.53 | (0.77–3.03) | .2281 |

Adjusted models controlled for Wave 1 site of recruitment (SBx or PR), age, disruptive behavior disorders, family use of public assistance, maternal education, W1 ACEs, and child sex.

ACEs, Adverse Childhood Experiences; ADHD, attention-deficit/hyperactivity disorder; W, wave of data collection.